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Do menu-labelling laws translate into results? The disparate impacts on population obesity and diabetes

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**ABSTRACT**

Despite their joint importance to health care costs, the nature of the relationship between obesity and diabetes is contested within the medical literature. We leverage California’s 2008 law mandating menu-labelling at restaurants to confirm that the law reduced obesity compared to the experience of counties not subject to such regulation. Despite this reduction in obesity, we find no California-specific reduction in the prevalence of diabetes and we find a significantly positive impact on the likelihood of new diabetes diagnoses. We evaluate a range of potential hypotheses that rationalize the divergent findings on obesity and diabetes.

**KEYWORDS**

Obesity; diabetes; health; calories; menu

**JEL CLASSIFICATION**

I18; I12

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**I. Introduction**

Obesity and diabetes are the twin health scourges of the modern world. The two conditions, often aggregated along with elevated blood pressure and cholesterol under the umbrella of metabolic syndrome, represent sizable and growing components of health care expenses in both the U.S. and abroad. Recent estimates place the annual U.S. direct medical costs of diabetes in 2017 at $237 billion as compared to $188 billion (CPI adjusted) in 2012, an increase of 26% in only five years.\textsuperscript{1} Furthermore, the medical profession’s consensus is that the significant positive correlation between obesity and Type 2 diabetes is causal, and researchers have proposed widely accepted biological mechanisms of how obesity leads to the insulin resistance that characterizes the more common form of diabetes. In all, the primary advice to reduce the prevalence of diabetes is to reduce the prevalence of obesity.

Given that diabetes is presumed to be a central way that obesity inflicts its costs, we examine the impact on both obesity and diabetes of laws mandating that restaurants include calorie information. Prior research has considered the impact of such laws on purchasing behaviour, body mass index (BMI, the underlying continuous measure on which obesity is based), and obesity prevalence. Much of this research has found significant impacts consistent with individuals using the additional information provided on menus to make more healthful (i.e. lower calorie) decisions, but the matter is still considered open.

We use population (county-level) data rather than individual data and consider only the 2008 passage of California’s menu-labelling law, which made calorie-labelling mandatory. Because restaurants in both treatment and control counties may have voluntarily chosen to add calorie-labelling to their menus prior to the mandate, our estimates capture the reduced form impact of the law on health outcomes, not the treatment effect of calorie-labelling per se.\textsuperscript{2} Simple difference-in-differences (DID) estimates and those with full sets of controls indicate that the menu-labelling law significantly lowered obesity rates. We continue with a piecewise-linear method that is distinct from the literature, focusing on rates of change rather than levels.

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\textsuperscript{2}Under the Patient Protection and Affordable Care Act passed in March 2010, national mandatory menu labelling became the law for chains of 20 restaurants or more. The law was scheduled to take effect in December 2015, though this deadline was eventually extended to 7 May 2018. To the extent that restaurants in the control states responded to the federal legislation prior to the end of our sample period (2013) despite its uncertain prospects of enforcement, our estimates of the law’s impact would be biased towards zero.

Supplemental data for this article can be accessed here.

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Despite these differences, our results are also consistent with the introduction of such menu-labelling mandates causing significant reductions in the growth rate of obesity. While counties outside of California without such menu-labelling laws saw substantially slower but still positive growth in obesity after 2008, obesity rates in California counties effectively stabilized (i.e. showed no growth).

DID applications also suggest a significant decline in diabetes prevalence but no significant difference in diabetes incidence. These results, however, are tenuous because the DID approach depends crucially on the assumption of parallel trends in the absence of treatment. The piecewise-linear method can both identify the violation of parallel trends and allow for hypothesis testing in the face of such violations. When we apply this method to diabetes, we find no negative impact of mandatory menu labelling on diabetes prevalence and a higher diabetes incidence rate for California counties relative to the control counties. As with obesity, growth in diabetes prevalence slowed after 2008 across our sample, but the reduced growth in California counties was not significantly different from the control counties. Growth of diabetes incidence, on the other hand, dropped after 2008 across the sample but significantly less so for California.

Section 2 details the existing economics literature on menu-labelling. We document the medical consensus consistent with menu-labelling laws reducing diabetes and the semi-heterodox theories that may explain our results in Section 3. Compared to the literature’s emphasis on capturing the average post-treatment effect, our piecewise-linear model offers a more intuitive measurement of policy impact when effects are likely to be gradual and cumulative. We outline its motivation, details, and relevant hypothesis tests in Section 4. Section 5 introduces and summarizes the data, and we explore the results of our various estimations in Section 6. Section 7 concludes with recommendations on menu-labelling to make the provided information more effective with respect to diabetes prevention.

II. Background on menu-labelling

The interplay of information and consumer behaviour has drawn economists to analyse the impact of mandated calorie labels on restaurant menus. The large percentage of the U.S. population classified as obese (39.8% in 2015–16) implies that even modest reforms might have substantial impacts on obesity prevalence.

Bollinger, Leslie, and Sorensen (2011) provide the most compelling evidence of how calorie-labelling on menus affects purchase behaviour. Using the mandated posting of calorie information on chain restaurants implemented in New York City in April 2008, that study considers transaction data from January 2008 through February 2009 at Starbucks from New York City, Boston, and Philadelphia. In all, the sample covers 316 Starbucks locations and includes over 100 million transactions. Consumers on average decreased calories per transaction by 6% in response to the menu-labelling, an impact that did not attenuate over their time period. This impact was almost entirely related to food purchases (which fell by 14%), and the impact on calories from drink purchases was indistinct from zero.

Such first-stage evidence is a necessary precondition for calorie-labelling to have an impact, but there still remains the possibility that consumers merely shift calorie consumption to different times or locations (e.g. home). Deb and Vargas (2016) and Restrepo (2017) use individual-level data from the Behavioural Risk Factor Surveillance System to address this issue by considering the impacts of mandated calorie-labelling by local jurisdictions on body mass index (BMI) and obesity prevalence. Deb and Vargas (2016) use a wide set of state and county laws and find an average 0.3 point decrease in BMI for all men and a larger

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1Prevalence reflects the percentage of people in a population with a condition, while incidence reflects the number of new diagnoses of diabetes per 1,000 individuals in a county.


3In contrast, the other studies cited in the survey by Van Epps et al. (2016) suffer from relatively small samples and use primarily on-site consumer surveys and very limited transaction data.

4Linking local demographics with Starbucks locations suggested a 26% decline for consumers whose pre-mandate transactions were greater than 250 calories.

5An individual’s Body Mass Index is a ratio of weight to height and is calculated as BMI = kg/m² = 703 x lb/in². The BMI thresholds for overweight and obese are 25 and 30.
Type 2 diabetes is characterized by insulin resistance in which the hormone loses its effectiveness within the body are critical. Type 1 diabetes is a chronic condition in which the body’s immune system attacks the pancreas and leads to reduced production of insulin. Current research suggests causal roles for genetic predisposition and viral triggers. In 2015, approximately 1.25 million individuals in the U.S. had Type 1 diabetes.

A much larger population (about 22 million persons in the U.S.) have been diagnosed with Type 2 diabetes. Type 2 diabetes is characterized by insulin resistance in which the hormone loses its effectiveness in instructing cells to accept blood sugar, and blood sugar levels remain elevated for extended periods. These elevated blood sugar levels damage kidneys, eyes, and nerves through microvascular complications (i.e. damaged capillaries). Elevated blood sugar levels also create macrovascular complications (e.g. heart attack, stroke) and retard wound healing, e.g. causing foot ulcers that can force amputation.

The growth in the prevalence of diabetes (1% in 1958, 2.5% in 1978, 4% in 1998, 7% in 2015 in the U.S.) is overwhelmingly caused by an increase in Type 2 diabetes. These growth rates have largely paralleled the growth in obesity prevalence both over time and across demographic groups. While the exact causes of diabetes are not yet understood, the prevailing theory is that body fat, especially abdominal fat, causes the release of fat cells into the bloodstream, and these fat cells then interfere with the function of insulin. This mechanism therefore suggests that any reduction in obesity will lead to a reduction in diabetes.

The economics literature finds a number of demographic and economic factors that are associated with obesity and diabetes rates. While both conditions are prevalent across all demographic groups, disparities exist. Individuals with lower education, with lower incomes, and who are older or Black or Hispanic are more likely to be obese or suffer from diabetes (Smith 2007; Baum and Ruhm 2009). Moreover, the awareness that one suffers from diabetes seems to follow a racial disparity. Comparing biomarkers for diabetes to self-reports, Chatterji, Joo, and Lahiri (2012) find that African-Americans and Latinos are more likely to be unaware of having diabetes compared to non-Latino Whites. The results from these papers suggest that we should use the aforementioned simple determinants of obesity and diabetes as controls in our analysis.

The existing medical consensus is that obesity increases the likelihood of developing diabetes and that controlling blood sugar levels through diet is a critical part of diabetes management after diagnosis. There is, however, another school of thought that posits a more direct avenue by which blood sugar can cause Type 2 diabetes. Specifically, high levels of sugar (especially fructose) may prompt accumulation of fat in the liver and degrade liver function. The weakened liver then sets in motion the insulin resistance. Basu et al. (2013) provide a meta-analysis documenting links of sugar consumption to diabetes. A similar mechanism unfolds with artificial sweeteners and responses to the increased insulin that they prompt (Suez et al. 2014). Additionally, Lustig, Schmidt, and Brindis (2012) point out that 40% of normal-weight people develop diseases including diabetes, hypertension, lipid problems, and cardiovascular disease, whereas only 20% of obese people develop these diseases.

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9Type 1 Diabetes Facts. http://www.jdrf.org/about/what-is-t1d/facts/.
This suggests that obesity does not cause diabetes and instead points to sugar consumption as the cause. If this hypothesis is correct, it is possible that an individual could substitute away from calories derived from protein and fat and towards those derived from carbohydrates and sugar, losing weight without the attendant decline in likelihood of becoming diabetic.

There is no cure for Type 2 diabetes, but a diabetic can manage the disease through dietary changes. The mechanism from menu-labelling laws to reductions in diabetes incidence is through reducing the likelihood of diagnosis for the marginal non-diabetic. Individuals with obesity and diabetes, though, are often reluctant to change their diet (Broadbent, Donkin, and Stroh 2011). For example, diabetics often receive warnings about the necessary dietary changes to prevent the disease long before receiving the diagnosis (Ogden et al. 2007), but they do not undertake the changes necessary to avoid the diagnosis. Using household scanner data, Oster (2018) finds only small changes in diet following a diabetes diagnosis, despite the large medical incentives of doing so. Thus, we would expect the impacts of menu-labelling laws on diabetes incidence to be gradual and increasing as the small changes in diet add up over time. An abrupt and immediate change in incidence should give the analyst pause.

To the extent that menu-labelling laws prompt disclosure that emphasizes calories irrespective of their type, these laws provide a policy-relevant way to consider the mechanism by which sugar consumption affects health. Specifically, the medical consensus that obesity causes diabetes would be supported by a finding that such menu laws significantly reduced both obesity and diabetes. Finding that these laws cause a reduction in obesity without affecting diabetes would support the semi-heterodox view that sugar causes diabetes directly.

**IV. Methods**

The now well-developed literature on the difference-in-differences (DID) method is admirably precise. The sample is divided into four groups: not-yet-treated and control observations before the treatment and now-treated and control observations after the treatment. If and only if not-yet-treated and control observations exhibit parallel trends in the periods leading up to treatment, the econometrician can plausibly assert that post-treatment trends will continue on their pre-treatment path. The estimated coefficient on the interaction of the treated group with the post-treatment indicator then yields the average impact of the treatment. If the assumption of pre-treatment parallel trends is violated, the interaction’s estimated coefficient conflates the treatment’s causal impact with the trend differences.

While the above has been extended to allow for the construction of better control groups (e.g. synthetic control in Abadie, Diamond, and Hainmueller 2010; entropy-balancing in Hainmueller 2012) and to accommodate the likely correlation of disturbances through clustered standard errors (Bertrand, Duflo, and Mullainathan 2004), it accurately characterizes the identification strategy. Its average treatment effect is exactly what the econometrician desires if the treatment realizes its full impact immediately. If, however, the treatment’s impact is realized gradually, then this average measure becomes less robust. It will, for example, depend on the length of time that post-treatment observations are made. The literature recognizes this and generally performs robustness checks with various leads and lags. We propose to use an established technique to model this gradual change directly.

Most work on piecewise (segmented) regression now focuses on estimating the unknown ‘change’ points at which segmentation occurs, but the features of piecewise regression with known change points are well established. The piecewise linear (PWL) model that we consider asserts that observations exhibit a linear time-trend before the treatment and a (not necessarily same-sloped) linear time-trend after the treatment. Its most general form allows for the treatment to create a discontinuity at the treatment point. Such a discontinuity would be consistent with the treatment having an instantaneous impact. Even with annual data, we do not expect to see such an immediate impact on health conditions such as obesity and diabetes. While we estimate the most general unrestricted model and then impose various restrictions, the following continuous piecewise linear function model is our preferred specification and facilitates discussion:
\[ y_{c,t} = \beta_1(t[t \leq t_0] + t_0[t > t_0]) + \beta_2(t[t \leq t_0] + t_0[t > t_0]) \times CA_c + \beta_3(t - t_0)[t > t_0] + \beta_4(t - t_0)[t > t_0] \times CA_c + x_{c,t}y + \varepsilon_{c,t}, \]  

in which \( y_{c,t} \) is the obesity or diabetes rate in county \( c \) at year \( t \), \( t_0 \) is the labelling law’s passage year of 2008, CA is an indicator for California counties, and \( x_{c,t} \) includes county fixed effects and other variables. Inequalities in brackets denote indicator functions that equal 1 if the inequality is a true statement for that observation. The hypothesis that \( \beta_1 = \beta_3 \) then addresses whether there was a change to the dependent variable’s trend in the control states at the treatment period.

If \( \beta_2 = 0 \) is zero, the assumption of pre-treatment parallel trends holds. The coefficient on the interaction of the post-passage time trend with the California indicator (\( \beta_4 \)) captures the differential trend in the post-passage dependent variables between California and the control group and is then the primary parameter of interest. If, however, \( \beta_2 \neq 0 \), the relevant hypothesis test is whether \( \beta_4 = \beta_2 \). That is, the PWL in our application allows for California’s marginal trend rate to differ systematically both before and after the treatment from those of the control states, and the question becomes whether those pre- and post-differences are statistically distinct.

The specification’s emphasis on California’s marginal differences somewhat masks the change on California trends that coincided with the law. To more clearly see the gross impact of menu-labelling laws on California, we also highlight the parameters \( \beta_1 + \beta_2 \) (California’s trend rate before 2008) and \( \beta_3 + \beta_4 \) (California’s trend rate 2008 and after). In all, the specification permits a wide range of hypotheses to be tested.

The primary alternative to the above piecewise linear specification is the inclusion of period fixed effects (year fixed effects in our application). The tradeoff between the two specifications is the standard one of additional inference gained from a functional form assumption (outlined above) against the concern that imposing an incorrect specification will bias estimates elsewhere. In our results, we highlight the benign nature of our linear trend assumption by superimposing our piecewise-linear estimates’ implications upon the estimates of the maximally flexible year fixed effects. We note the folly of extrapolating out too far along these trends but with this caveat believe that this remains a valuable tool for the econometrician.

V. Data

We use age-adjusted county-level estimates for obesity and diabetes rates from the Centres for Disease Control and Prevention (CDC) between 2004 and 2013. Given that the stated intent of menu-labelling legislation was to lower aggregate obesity rates, we prefer to observe the ultimate outcome of interest. The county level obesity estimates additionally provide more accurate coverage, as the Behaviour Risk Factor Surveillance System (BRFSS) individual level survey does not necessarily interview a sufficient number of individuals in every county in the United States every year. Lastly, the usage of coarser data provides further assurance and confirmation of the robustness of the results of Deb and Vargas (2016) and Restrepo (2017) that consider the impact of such laws on body mass and obesity using the BRFSS survey directly.

To construct county-level estimates, the CDC uses Bayesian multilevel modelling techniques. Models predict the probability of an individual being obese or diabetic based on age, sex, and race characteristics from three-year samples (over 1.2 million individuals) of the BRFSS survey. That is, the BRFSS is a repeated cross-section rather than a panel. Estimates are calculated using annual county population data from the census and are validated using direct estimates from 298 large counties.\(^{13}\) A BMI greater than 30 classifies an individual as obese, and a medical diagnosis of diabetes classifies an individual as diabetic. Given that the CDC measures diabetes prevalence as the proportion of individuals who have ever been diagnosed with diabetes, we also consider diabetes incidence rates, or the number of newly diagnosed cases per 1,000 individuals in a county.

\(^{13}\)The CDC’s county-level estimation calculation method is online at http://www.cdc.gov/diabetes/pdfs/data/calculating-methods-references-county-level-estimates-ranks.pdf.
The prospect that obesity prevalence could be reduced with more complete nutritional information has made menu-labelling at restaurants a common policy target. Under the Patient Protection and Affordable Care Act (PPACA) passed in March 2010, national mandatory menu labelling became the law for chains of 20 restaurants or more. Specifically, menu boards must declare calorie counts. This provision, however, faced considerable uncertainty with respect to implementation. The law was scheduled to take effect in December 2015, though this deadline was extended to December 2016 (in July 2015), then to May 2017 (in May 2016), and to May 2018 (in May 2017). The FDA finally required that restaurants abide by rules dictating calorie labelling after 7 May 2018. Enforcement tools are the same as for other misbranding actions under the Federal Food, Drug, and Cosmetic Act (Title 21, Ch. 9 of United States Code). This code specifies penalties of not more than one year in prison and fines of not more than $1,000 for first violations and not more than three years in prison and fines of not more than $10,000 for second violations or violations ‘with the intent to defraud or mislead’ (21.9.III.333.a).

Before the PPACA’s 2010 passage, five states (including California) and nine smaller jurisdictions had already passed and implemented menu labelling laws. Given this reality, we choose to exclusively study the impact of California’s state-level legislation. Details of menu-labelling laws (especially regarding enforcement) may differ across jurisdictions, an issue that focusing on a single law change sidesteps. Furthermore, California offers the largest jurisdiction among the early adopters.

California became the first state to pass menu labelling legislation in September 2008, following New York City, NY, and King County, WA. Under the requirements of this law, restaurants in California with at least 19 other franchised facilities under the same name must post calorie count information directly next to items on menus or menu boards. Brochures accessible upon request are required to show carbohydrates, saturated fat, and sodium. The law became effective and enforced with fines up to $500 in January 2011.

We focus on identifying changes following the passage date of the California law in 2008 instead of the effective date in 2011. Unlike with the federal law, there was little political uncertainty regarding implementation and California restauranteurs knew they would shortly need to comply. We therefore expect to see menu board changes and consumer responses shortly after passage. Our choice of passage rather than enforcement is especially appropriate given the relatively long lag (28 months) between adoption and enforcement; most laws had only a 6 month lag. As a check of treatment definition, we also estimated our model using the effective date (2011) as the relevant threshold. Those estimates, reported in supplemental appendix Table A.1, indicated a significant instantaneous impact on obesity prevalence as well as a further gradual impact, results that are consistent with misspecification when the passage date is the relevant threshold.

Our control group consists of all counties outside of California that neither passed menu-labelling legislation nor were part of states that did so. All counties in Massachusetts, Maine, Oregon, and New Jersey are excluded because state-level menu-labelling legislation was passed during the sample period. For a list of counties that passed menu-labelling legislation, see footnote 16. We further exclude counties in Alaska, Hawaii, and Washington, D.C. To the extent that restaurateurs in the control states responded to the federal legislation prior to the end of our sample period

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16 States: California (September 2008), Massachusetts (May 2009), Maine (June 2009), Oregon (June 2009), and New Jersey (January 2010). Counties: New York, NY (January 2008), King, WA (April 2008), Philadelphia, PA (November 2008), Westchester, NY (November 2008), Suffolk, NY (February 2009), Ulster, NY (April 2009), Albany, NY (September 2009), Montgomery, MD (November 2009) and Davidson, TN (February 2010).
17 New York City’s five boroughs each coincide with a county, though they do not have county governments.
19 Deb and Vargas (2016) use enforcement of any laws relevant to the respondent’s county of residence as its preferred date of treatment. That paper also includes an indicator for whether the survey respondent resided in a county that had calorie-labelling mandates at any point in the sampled period (2003–12); the estimated coefficient of which was large and significantly negative for both men and women. Restrepo (2017) simultaneously considers both passage and enforcement dates for its sample period (2004–12). While only enforcement is statistically significant, the estimated coefficient on passage is negative and is a magnitude consistent with instantaneous implementation over the limited time period (six months) between passage and enforcement.
(2013) despite its uncertain prospects of enforcement, our estimates of the law’s impact should be biased towards zero.

As discussed above, the economic literature finds that a number of demographic variables are associated with obesity and diabetes prevalence and incidence rates (Smith 2007; Baum, and Ruhm 2009; Chatterji, Joo, and Lahiri 2012; Oster 2018). These demographic variables include: race and ethnicity, the age distribution, education, income, and urban-rural disparities. Differences between treatment and control counties in the level of these characteristics do not bias our methods. The county fixed effect accounts for these. Bias, however, could enter our estimates to the extent differential changes over time in these characteristics are correlated with treatment status. For example, diabetes prevalence increases with age. If control counties age more rapidly than treated counties during our sample period, our estimates of the impact of menu-labelling mandates would be overstated.

Table 1 presents results from a number of balance tests on county-level demographic and economic characteristics. Observations are at the annual county level. Columns (1) through (4) present the mean values in the pre-treatment period for control and treatment counties, and the difference with its associated t-statistic. Clearly, California counties are different, on average, then the rest of the United States. The difference between treatment and control counties is statistically significant at the 10% level in 20 of the 21 variables, and in 15 of the 21 variables at the 1% level. Compared to the rest of the nation, California counties are less White and Black, and more Asian and Hispanic; have higher levels of education; are younger and more urban; and have higher incomes, lower poverty rates, and higher unemployment. Columns (6) and (7) report means for the post-treatment period.

The main concern for identification in our setting is differential changes over time in unobservables that are correlated with changes in menu-labelling legislation and the outcome variables. We directly test for this using the observable county demographic and economic characteristics at hand. In Columns (7) and (8), we report the coefficient on the \((POST_t \times TREAT_t)\) interaction from a difference-in-differences framework and its associated t-statistic. Economically large and statistically significant coefficients would suggest that treated and control counties faced differential underlying trends in demographic or economic characteristics with known associations with obesity or diabetes. Overall, we find small, if any, differential trends between California and the control counties. Both treated and control counties became more Asian, but California increasingly so by 0.68 percentage points. Control counties became more educated relative to counties in California. We find no differences in relative changes in the age distribution, median incomes, or population density. California counties experienced increases in the poverty rate and unemployment rate relative to control counties. As people tend to live more healthfully during recessions and excessive weight gain is consequently procyclical (Ruhm 2005), this change works against us finding an effect of the mandate.

Given the potential for differential trends in unobservables to bias our estimates, we prefer specifications that control for all demographic and economic characteristics included in Table 1. The remaining sources of bias in our estimates come from any unobservable trends that are correlated with both treatment and the dependent variables and that are not captured by the demographic and economic characteristics that we include. Note that these characteristics need not capture a causal effect on the outcomes, only that they capture correlations between unobservables that do have a causal effect on the outcomes and that are correlated with the control characteristics.

VI. Results

We begin by displaying summary statistics for the outcome variables in Table 2. In the ‘Post-Pre’ rows, we show the general trend of obesity prevalence, diabetes prevalence, and diabetes incidence for California vs. control counties. A general increase in obesity and diabetes prevalence and decrease in diabetes incidence can be observed overall. We, however, see clearly that obesity prevalence increases much less

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20Sources of control variables can be found in the Data Sources section on page 23.
for California vs. control counties. Diabetes incidence does not appear to be different for California vs. control counties, and diabetes incidence appears to decrease less for counties in California than it does in the control counties.

We next turn to our DID estimates in Table 3. The dependent variables of obesity and diabetes are identified atop each column. Columns (1), (3), and (5) show simple DID estimates with no controls, and Columns (2), (4), and (6) show DID estimates with a full set of controls including county fixed effects and year fixed effects. All standard errors are clustered at the state level.

Obesity’s simple DID with and without additional controls are consistent with casual observation and the literature on menu-labelling laws that finds significant impacts. While counties in California were less obese than counties in the control states and obesity was higher in 2008 and after than before, California’s counties after the menu-labelling law were less obese than one would have otherwise expected. Even after including all controls, the DID coefficient indicates that the prevalence of obesity was 0.7 percentage points lower than would have been expected.
Table 3. Difference-in-differences results.

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</tr>
</thead>
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<td>CA</td>
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<td>−</td>
<td>−1.438***</td>
<td>−</td>
<td>−2.206***</td>
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<tr>
<td></td>
<td>(0.392)</td>
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<td>(0.244)</td>
<td>(0.324)</td>
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<tr>
<td>Post</td>
<td>3.656***</td>
<td>−</td>
<td>1.240***</td>
<td>−</td>
<td>−0.729***</td>
<td>−</td>
</tr>
<tr>
<td></td>
<td>(0.137)</td>
<td></td>
<td>(0.070)</td>
<td>(0.095)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post*CA</td>
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<td>−0.685***</td>
<td>−0.494***</td>
<td>−0.273***</td>
<td>0.131</td>
<td>0.253</td>
</tr>
<tr>
<td></td>
<td>(0.137)</td>
<td>(0.176)</td>
<td>(0.070)</td>
<td>(0.095)</td>
<td>(0.095)</td>
<td>(0.144)</td>
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<td>0.101</td>
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</tr>
<tr>
<td>County FE</td>
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<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Year FE</td>
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<td>Yes</td>
<td>No</td>
<td>Yes</td>
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</tr>
<tr>
<td>Other Controls</td>
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<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

Notes: N = 30,090 county-year observations. *p < 0.05, **p < 0.01, ***p < 0.001. Clustered standard errors at the state level in parentheses under point estimates. CA is an indicator for a county being in California, and Post references passage of California’s menu-labelling law in 2008 (i.e. 2008 or later). Specifications (2) and (4) include county and year fixed effects and control for race, age, income, education, population density, unemployment, and poverty. Years of observation are 2004–2013.

Sources: County level obesity and diabetes rates are taken from CDC. Race, age, income, population density, and education are taken from 2000 and 2010 Census. Poverty is from SAIPE and unemployment is from BLS. Included states are California and all states in the US except any state or county that passed menu labelling legislation or Alaska, Hawaii, and DC (44 states).

expected \((t \approx 3.9)\).21 The results on diabetes prevalence are qualitatively similar. California faces a lower prevalence of diabetes, diabetes prevalence is higher in the control states 2008 and after than before, and California’s diabetes prevalence rates after the menu-labelling law are lower than would be expected. Results for diabetes incidence show no significant effect post-menu-law with and without controls.

All DID results are subject to the criticism that counties in the control group may differ systematically in their pre-treatment rate of change when compared to California counties. We turn to our piecewise-linear specification to consider and address this concern in Table 4. Relevant hypotheses are articulated (with inference) in the lower panel. Columns (1), (3), and (5) display piecewise linear results that allow for discontinuities at 2008, while Columns (2), (4), and (6) enforce that such discontinuities are restricted to zero.

We first consider our estimated impacts of the menu-labelling law on the prevalence of obesity. Because the estimated coefficients on the discontinuity variables are insignificantly distinct from zero, we emphasize the results of Column (2). Parallel trends in the pre-treatment period are (relatively weakly) rejected in favour of the hypothesis that California’s obesity rate increased at a somewhat slower rate than that of the control states \((\beta_2 = -0.116, p = 0.02)\). While less than other states, the obesity rate in California counties was still increasing at a statistically significant rate \((\beta_1 + \beta_2 = 0.887)\). This was no longer the case in 2008 and after. During that period, the control counties experienced a significantly lower growth rate in obesity than before 2008 \((\beta_3 - \beta_1 = -0.690)\), but the gap between California counties and those controls significantly increased in magnitude: \((\beta_4 - \beta_2 = -0.18, p < 0.001)\). In fact, our estimates cannot reject that the change in California counties’ obesity rate fell to zero (i.e. obesity prevalence stabilized) after 2008 \((\beta_3 + \beta_4 = 0.020, s.e. = 0.058)\). The implications of these estimates appear in Figure 1. Estimated coefficients of year fixed effects are also plotted with confidence bands, highlighting the mildness of our assumed functional form compared to the completely unrestricted form.

In all, our preferred results qualitatively conform to the literature that finds that menu-labelling laws appear to reduce obesity. Our estimated magnitudes (0.2 percentage points lower with each year after passage) and their implications are statistically significant but substantially less than those found in the literature.22 Given the relatively weak rejection of

21We note that this is substantially smaller than the estimate of a 3 percentage point decrease found by Restrepo (2017). This difference could be driven by the different population (New York City vs. California) and dining habits, the different policy treatment (New York City’s law vs. California’s law), or by the differing aggregation of data (individual vs. county-level).

22Deb and Vargas (2016) use a wide set of such laws and find a 0.3 point decrease in BMI for all men and a larger decrease for men with the highest BMI. Relatedly, Restrepo (2017) uses the laws from New York City, finding a 0.4 point decrease in BMI irrespective of gender and a 3 percentage point decrease in obesity prevalence.
Table 4. Obesity and diabetes impacts after California passage date (2008).

<table>
<thead>
<tr>
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<tr>
<td>$y_{i,t} = \beta_1(t &lt; t_0) + \beta_2(t &gt; t_0)$</td>
<td>$1.051^{***}$</td>
<td>$1.003^{***}$</td>
<td>$0.458^{***}$</td>
<td>$0.412^{***}$</td>
<td>$0.369^{***}$</td>
<td>$0.227^{***}$</td>
</tr>
<tr>
<td></td>
<td>(0.046)</td>
<td>(0.052)</td>
<td>(0.044)</td>
<td>(0.035)</td>
<td>(0.078)</td>
<td>(0.058)</td>
</tr>
<tr>
<td>PreTrend*CA ($\beta_2$)</td>
<td>$-0.149^{**}$</td>
<td>$-0.116^{*}$</td>
<td>$-0.051$</td>
<td>$-0.097^{***}$</td>
<td>$-0.175^{*}$</td>
<td>$-0.112^{*}$</td>
</tr>
<tr>
<td></td>
<td>(0.049)</td>
<td>(0.046)</td>
<td>(0.037)</td>
<td>(0.025)</td>
<td>(0.063)</td>
<td>(0.044)</td>
</tr>
<tr>
<td>Post</td>
<td>$-0.224$</td>
<td>$-0.217^{**}$</td>
<td>$-0.674^{***}$</td>
<td>$0.160$</td>
<td>$0.160$</td>
<td>$0.160$</td>
</tr>
<tr>
<td></td>
<td>(0.132)</td>
<td>(0.074)</td>
<td>(0.144)</td>
<td></td>
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</tr>
<tr>
<td>Post*CA</td>
<td>$0.098$</td>
<td>$-0.220^{*}$</td>
<td>$-0.674^{***}$</td>
<td>$0.160$</td>
<td>$0.160$</td>
<td>$0.160$</td>
</tr>
<tr>
<td></td>
<td>(0.156)</td>
<td>(0.098)</td>
<td>(0.174)</td>
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<tr>
<td>PostTrend ($\beta_3$)</td>
<td>$0.329^{***}$</td>
<td>$0.317^{***}$</td>
<td>$0.202^{***}$</td>
<td>$0.191^{***}$</td>
<td>$-0.418^{***}$</td>
<td>$-0.454^{***}$</td>
</tr>
<tr>
<td></td>
<td>(0.053)</td>
<td>(0.057)</td>
<td>(0.039)</td>
<td>(0.031)</td>
<td>(0.040)</td>
<td>(0.042)</td>
</tr>
<tr>
<td>PostTrend*CA ($\beta_4$)</td>
<td>$-0.306^{***}$</td>
<td>$-0.297^{***}$</td>
<td>$0.021$</td>
<td>$0.008$</td>
<td>$0.183^{***}$</td>
<td>$0.202^{***}$</td>
</tr>
<tr>
<td></td>
<td>(0.039)</td>
<td>(0.039)</td>
<td>(0.023)</td>
<td>(0.027)</td>
<td>(0.036)</td>
<td>(0.044)</td>
</tr>
<tr>
<td>$R^2$</td>
<td>$0.885$</td>
<td>$0.884$</td>
<td>$0.913$</td>
<td>$0.912$</td>
<td>$0.877$</td>
<td>$0.875$</td>
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<tr>
<td>$H_0$ $\beta_1 + \beta_2 = 0$</td>
<td>$0.903^{***}$</td>
<td>$0.887^{***}$</td>
<td>$0.407^{***}$</td>
<td>$0.315^{***}$</td>
<td>$0.194^{***}$</td>
<td>$0.115^{*}$</td>
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<td></td>
<td>(0.058)</td>
<td>(0.061)</td>
<td>(0.029)</td>
<td>(0.032)</td>
<td>(0.044)</td>
<td>(0.053)</td>
</tr>
<tr>
<td>$H_0$ $\beta_3 + \beta_4 = 0$</td>
<td>$0.023$</td>
<td>$0.020$</td>
<td>$0.223^{***}$</td>
<td>$0.199^{***}$</td>
<td>$-0.236^{***}$</td>
<td>$-0.251^{***}$</td>
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<tr>
<td></td>
<td>(0.056)</td>
<td>(0.058)</td>
<td>(0.026)</td>
<td>(0.027)</td>
<td>(0.037)</td>
<td>(0.040)</td>
</tr>
<tr>
<td>$H_0$ $\beta_3 - \beta_4 = 0$</td>
<td>$-0.722^{***}$</td>
<td>$-0.690^{***}$</td>
<td>$-0.256^{***}$</td>
<td>$-0.221^{***}$</td>
<td>$-0.788^{***}$</td>
<td>$-0.680^{***}$</td>
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<tr>
<td></td>
<td>(0.047)</td>
<td>(0.043)</td>
<td>(0.048)</td>
<td>(0.040)</td>
<td>(0.091)</td>
<td>(0.076)</td>
</tr>
<tr>
<td>$H_0$ $\beta_4 - \beta_2 = 0$</td>
<td>$-0.158^{***}$</td>
<td>$-0.181^{***}$</td>
<td>$0.072$</td>
<td>$0.105^{*}$</td>
<td>$0.358^{***}$</td>
<td>$0.314^{***}$</td>
</tr>
<tr>
<td></td>
<td>(0.046)</td>
<td>(0.045)</td>
<td>(0.051)</td>
<td>(0.040)</td>
<td>(0.089)</td>
<td>(0.071)</td>
</tr>
</tbody>
</table>

Notes: N = 30,090 county-year observations. *p < 0.05, **p < 0.01, ***p < 0.001. Pre and Post reference passage of California’s menu-labelling law in 2008. Clustered standard errors at the state level in parentheses under point estimates. Delta-method standard errors in parentheses under hypothesis test point estimates. All regressions include county fixed effects and control for race, age, income, education, population density, unemployment, and poverty. Years of observation are 2004–2013.

Sources: County level obesity and diabetes rates are taken from CDC. Race, age, income, population density, and education are taken from 2000 and 2010 Census. Poverty is from SAIPE and unemployment is from BLS. Included states are California and all states in the US except any state or county that passed menu labelling legislation or Alaska, Hawaii, and DC (44 states).

parallel trends, it is unsurprising that they are, however, consistent with our estimates from the DID with control regressors.

To get a sense of the plausibility of these estimates, we return to the finding of Bollinger, Leslie, and Sorensen (2011) that food-calories purchased at Starbucks fell by an average 6% after calorie labels were mandated. As consumers probably consume some of these forgone calories in other settings, this figure is presumably an upper bound. Block et al. (2012) estimates that an adult’s fast food meal averages 836 calories, suggesting a 50 calorie decline per meal. Since Wishnfsky (1958), it is widely accepted that a 3500 calorie deficit will lead to a one-pound loss of body weight, so no change in physical activity and no added calories elsewhere would require 70 fast-food visits to lose one pound. This relatively modest magnitude is potentially magnified by the estimate that 36.6% of Americans eat at least one fast-food meal per day.23 Spreading this fast-food consumption uniformly across the population yields 2.7 weekly visits or 142 over a year, suggesting a potential annual loss of two pounds.

What impact would a yearly uniform two-pound loss (or lack of two-pound gain) have on the prevalence of obesity? Following Ng et al. (2016), we construct the beta distribution of BMI that matches the 2007 estimates of California’s mean BMI, overweight prevalence (BMI>25), and obesity prevalence (BMI>30).24 The match to these 2007 California statistics is good. Estimated average BMI is 26.8, and our beta distribution’s counterpart is 26.6. Estimated overweight and obesity prevalences are 57.3% and 23.1%, and our beta distribution implies prevalences of 57.8% and 23.5%. The resulting parameters generate the skewed-right distribution also found by Ng et al. (2016). About 6.4% of the population in our beta distribution has a BMI that exceeds 35 and 1% has BMI that exceeds 40.25

Using average adult height for American males and females to back out body-weights from this BMI


distribution, a nationwide two-pound loss would generate a nearly two percentage point decrease in obesity prevalence in 2007 California. Generating our predicted yearly obesity ‘decline’ of 0.2 percentage points could likewise be achieved by a uniform loss of 0.2 pounds each year. Returning to BMI, a yearly uniform loss of 0.2 pounds would correspond to a yearly decline of 0.03 in a population’s average BMI. That is, if consumers at fast-food restaurants respond as predicted by Bollinger, Leslie, and Sorensen (2011) but they consume up to 90% of the forgone calories in other settings, one would still expect to see the magnitudes of our estimates.

Our estimates of the piecewise-linear specification using diabetes prevalence as the dependent variable are quite different in their implications. First, the estimated coefficients on the discontinuity variables are no longer statistically indistinct from zero. Column (3) indicates a statistically significant instantaneous decline in 2008 across all states ($p = 0.003$) as the piecewise-linear specification contorts itself to match the data. This would be consistent with the pre-treatment linear trend being overly restrictive. California counties exhibit an even larger instantaneous decline, though this is of less statistical significance ($p = 0.025$). Because of our belief that any effects on diabetes will be gradual and because of our focus on the California regime change, we restrict these instantaneous impacts to be zero in Column (4). Pre-treatment and post-treatment parameters are qualitatively similar regardless of this restriction, and the precision of pre-treatment parameters increases markedly.

The marginal California trend impact post-treatment ($\beta_4 - \beta_2$) is positive in both columns but negligible in the more general case of Column (3). Unlike its counterpart with obesity, California counties’ diabetes prevalence rate of change when instantaneous impacts are ruled out in Column (4) is worse ($\beta_4 - \beta_2 = 0.105, p = 0.009$). That is, California’s pre-treatment trend advantage not only does not grow larger as would be expected if the menu-labelling law improved diabetes prevalence but the pre-treatment advantage is eliminated. Furthermore, Column (4) shows that the violation of the parallel trends assumption is much starker for diabetes prevalence than for obesity prevalence. This latter finding rationalizes the piecewise-linear results with those of the DID analysis. The implications of these estimates appear in Figure 2. Again, estimated coefficients of year fixed effects are also plotted with confidence bands, highlighting the harmlessness of our functional form compared to the completely unrestricted form. This is the case despite the statistical significance of the discontinuity parameters for diabetes.

Lastly, the diabetes incidence results of Columns (5) and (6) show more potential evidence supporting the hypothesis under which sugar directly causes diabetes. This outcome captures a measure of the new

\[ \beta_4 - \beta_2 = 0.105, p = 0.009 \]

For context, the average American heights for males and females are 5’ 9” and 5’ 4”. A BMI of 35 for males and females of average height corresponds to weights of 237 lbs and 204 lbs. A BMI of 40 would correspond to weights of 271 lbs and 233 lbs.
diagnoses of diabetes over time. As results are qualitatively similar regardless of our restriction on discontinuous impacts, we consider Column (6) for rhetorical simplicity. Our piecewise linear results find that the rate of new diabetes diagnoses is fairly different for California vs. control counties pre-passage ($\beta_2 = -0.112, p = 0.011$) and that the rate of new diagnoses declines significantly post-passage for the control states ($\beta_3 = -0.454, p < 0.001$). California incidence, though, declines much less than the control counties ($\beta_4 = 0.202, p < 0.001$).

When combined with California’s pre-treatment advantage in incidence, the implied causal impact of the menu-labelling law on new diagnoses of diabetes is even greater ($\beta_4 - \beta_2 = 0.314, p < 0.001$). This marked contrast with diabetes prevalence is illustrated in Figure 3, in that the rate of new diabetes diagnoses by 2013 is lower outside of California than in it.

VII. Discussion

These results indicate that the aggregate level of data and methods that we employ can detect impacts of menu-labelling laws on obesity. We nevertheless find no evidence that California’s law had the impact on its diabetes prevalence that the prevailing medical conventional wisdom predicts. In fact, our results strongly suggest that the menu-labelling law coincided with a worsening of new diabetes diagnoses in California. We now discuss a number of theories and their potential to rationalize our findings, cognizant of the fact that we do not formally model the endogenous relationship between diabetes and obesity. Note that, as always, our estimates are subject to potential bias under violations of the identification assumption: that there are no remaining unobservable shocks to trends in obesity and diabetes correlated with the timing of menu-labelling legislation, after including controls for demographic and economic characteristics.

First, our findings are not consistent with obesity directly causing diabetes, which would suggest the significant reduction in obesity following the menu-labelling mandate be accompanied by reductions in diabetes. We find the opposite, an increase in diabetes. A second theory suggests causation moving in the opposite direction, from diabetes to obesity. A new diagnosis of diabetes could potentially cause a person to manage their disease through weight reduction. In this scenario, the increase in diabetes diagnoses following the menu-labelling legislation caused the reduction in obesity prevalence. However, evidence from the public health and economics literatures suggests that new diabetics struggle to manage their disease through a reduction in calories (Ogden et al. 2007; Broadbent, Donkin, and Stroh 2011; Oster 2018). Moreover, the magnitude of the change in diabetes incidence is too small to explain the entire estimated change in obesity. Thus, we find the reverse causation hypothesis unconvincing.

Taken together, the estimated decline in obesity and increase in diabetes incidence are consistent with some combination of diabetes being caused by a mechanism outside of obesity (perhaps directly through the semi-heterodox theory of direct liver damage from sugar consumption) and consumers adjusting consumption in a way that simultaneously reduces overall calories but shifts calories towards a more damaging mix. Our work is not conclusive, and more research is required on the underlying determinants of diabetes.

To conclude, menu-labelling legislation that requires only the posting of calories does not appear to be a useful policy lever to reduce diabetes. A potential policy is to extend the calorie information to include composition with respect to sugar, but this would presumably run into the same criticisms faced by the California legislation. Restaurants fought the new calorie-labelling requirements, and a recurring
argument by industry was the visual clutter and overload caused by including additional information. Additional sugar information would worsen visual clutter even further. Our study indicates that more research is warranted to determine if benefits outweigh these costs.

Disclosure statement

No potential conflict of interest was reported by the authors.

Data Sources


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Ogden, C. L., M. D. Carroll, M. A. McDowell, and K. M. Flegel. 2007. Obesity among Adults in the United States – No


