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THE EFFECT OF DENTAL CARE ON CARDIOVASCULAR DISEASE OUTCOMES: AN APPLICATION OF INSTRUMENTAL VARIABLES IN THE PRESENCE OF HETEROGENEITY AND SELF-SELECTION

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SUMMARY

Studies show a relationship between oral inflammatory processes and cardiovascular risk factors, suggesting that dental care may reduce the risk of cardiovascular disease (CVD) events. However, due to the differences between men and women in the development and presentation of CVD, such effects may vary by sex. We use a valid set of instrumental variables to evaluate these issues and include a test of essential heterogeneity. CVD events include new occurrences of heart attack (including death from heart attack), stroke (including death from stroke), angina, and congestive heart failure. Controls include age, race, education, marital status, foreign birthplace, and cardiovascular risk factors (health status, body mass index, alcohol use, smoking status, diabetes status, high-blood-pressure status, physical activity, and depression). Our analysis finds no evidence of essential heterogeneity. We find the minimum average treatment effect for women to be −0.01, but find no treatment effect for men. This suggests that women who receive dental care may reduce their risk of future CVD events by at least one-third. The findings may only apply to married middle-aged and older individuals as the data set is only representative for this group.

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KEY WORDS: dental care; instrumental variables; essential heterogeneity; cardiovascular disease

1. INTRODUCTION

Research is growing on the connection between dental care and cardiovascular disease (CVD). Studies suggest that dental care may affect CVD via inflammatory conditions such as periodontitis and related conditions (Friedewald *et al.*, 2009). The effect of dental care on CVD may differ by sex, given recent findings on the different ways in which CVD develops and presents itself in women versus men (Grady *et al.*, 2000; Milner *et al.*, 2004; Mosca *et al.*, 2007; Mendelsohn and Karas, 2005).

To estimate the average treatment effect (ATE) of dental care on CVD outcomes, we follow Heckman *et al.* (2006) and use the method of instrumental variables (IV) including tests for essential heterogeneity (Basu *et al.*, 2007; Brooks and Chrischilles, 2007). If no essential heterogeneity is found, this approach yields estimates of the ATE which are not subjected to attenuation bias, omitted variable bias, or endogeneity bias (Wooldridge, 2002). We apply this approach to panel data on women and men aged 44 and older in order to accurately estimate the ATE of dental care on future CVD events. This group of individuals was chosen as they are more likely to experience CVD events. We hypothesize that

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dental care will reduce the likelihood of suffering a CVD event in this cohort and that this reduction will be larger for women. Knowledge of the ATEs for men and women in this age cohort will allow policy makers to estimate the effects of expanding dental insurance to these groups.

1.2. Background

Studies have found a relationship between periodontal health and CVD and its risk factors. Findings include a positive association between tooth loss and heart disease and between severe periodontitis and inflammatory and metabolic risk factors for CVD (Okoro *et al*., 2005; Nibali *et al*., 2007).

Periodontal destruction is positively associated with coronary artery disease, and periodontal infection is positively associated with acute coronary syndrome (Accarini and de Godoy, 2006; Gotsman *et al*., 2007; Pussinen *et al*., 2007). Long-term use of antibiotics reduces CVD events in individuals without periodontitis, but is ineffective in patients with periodontitis (Paju *et al*., 2007). Desvarieux *et al*. (2005) found a positive relationship between the level of bacteria in the mouth associated with periodontal disease and the beginnings of atherosclerosis. Söder *et al*. (2007) found that periodontal disease was positively associated with death from all causes including CVD.

A number of studies have examined whether dental interventions may be effective in reducing CVD risk. These pilot studies find that intensive treatment of periodontal disease is associated with a reduction in CVD risk factors (D'Aiuto *et al*., 2006; Elter *et al*. 2006; Ellis *et al*., 2007; Söder and Yakob, 2007; Tonetti *et al*., 2007). In response, multi-center trials have begun.

One multi-center trial is the Periodontitis and Vascular Events study, which randomly assigned patients to periodontal therapy or community dental care, but found no difference in outcomes in their pilot study (Couper *et al*., 2008; Beck *et al*., 2008). Although this pilot was not designed to be long enough nor large enough for an adequate estimate of treatment effects, another potential reason for this finding is that periodontal therapy was received by at least 11% of the control group. Nonetheless, high sensitivity C-reactive protein was lower in non-obese individuals in the treatment group relative to those who received no care (Offenbacher *et al*., 2009).

A randomized controlled trial (RCT) has also found a connection between periodontal treatment (compared with no care) and low-density lipoprotein (LDL) with LDL levels falling approximately 25% in 90 days (Oz *et al*., 2007). Another RCT found reduced levels of fibrinogen and reduced levels of serum IL-6 and C-reactive protein in response to periodontal treatment (compared with no care) (Vidal *et al*., 2009).

These findings are encouraging, but there may be sex differences that have not yet been adequately explored. Studies show differences by sex with respect to the way CVD develops and presents itself (Milner *et al*., 2004; Wenger *et al*., 2007). These results suggest that the effect of dental care on CVD events may vary by sex. The differing ways in which CVD develops in women relative to men suggest that middle-aged and older women may receive more protection from dental care relative to men in this age group. Owing to the protective effect of estrogen and other factors, women tend not to develop CVD until after the onset of menopause, which occurs, on average, at age 51 (Mendelsohn and Karas 2005; Xing *et al*., 2009; Bromberger *et al*., 1997). Women who receive dental care at the time they are beginning to lose the protective effect of estrogen may receive greater effects than men of the same age who will tend to have more advanced CVD. For example, the reduction of oral inflammation via dental procedures may be more important for women than men in this age group as it appears that inflammation may be a more important risk factor in women than in men (Cushman *et al*., 2005; Kip *et al*., 2005; Khera *et al*., 2005).

There is a clear need in the literature to examine the causal effect of dental care on CVD events. Differentiating by sex appears to be essential for accurate findings.

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2. METHODS

2.1. Theoretical model

Our theoretical model contains a cardiovascular-health production function and a demand-for-dental-care function. We specify a Grossman-type model (Grossman, 1972; Zweifel and Breyer, 1997). Health production functions include factors that influence health outcomes, but do not include factors related to the purchasing of these factors (*e.g.* prices, income/wealth, insurance). Including these latter items in the production function changes it into a hybrid demand-production function resulting in biases to the parameter estimates of the production function that cannot be statistically corrected for (Rosenzweig and Schultz, 1983).

Cardiovascular health varies by risk factors such age, sex, race, smoking, cholesterol, blood pressure, physical activity, body mass index (BMI), diabetes, alcohol use, and depression (Davidson *et al.*, 2005; American Heart Association, 2009). Education levels are inversely related to CVD (Lenfant, 1996). In addition, cardiovascular health will vary with the use of cardiovascular-relevant medical care. Finally, CVD events may vary with dental care. A cardiovascular-health production function may be written as follows:

$$CVDE = CVDE(D, R, MD, DDS) \tag{1}$$

where $CVDE$ is CVD events, D is demographics (age, sex, race, education), R is non-demographic risk factors (including health status, BMI, alcohol use, smoking status, hyperlipidemia, diabetes status, high-blood-pressure status, exercise, stress, poor diet, family history, and depression), MD is cardiovascular-relevant medical care (non-dental pharmaceutical interventions, non-dental surgical interventions), and DDS is dental care.

The demand for dental care is a function of insurance, price, income/wealth, and taste. Taste is made up of all other factors that may influence an individual's preferences regarding dental care. A demand-for-dental-care function may be written as follows:

$$DDS = DDS(INS, P, T, Y) \tag{2}$$

where INS is dental insurance, P is price, T is taste, and Y is income/wealth. Note that non-demographic cardiovascular risk factors do not enter the demand-for-dental-care function. This is because individuals in our data would not be likely to have information that dental care affects CVD. A public consensus statement about dental care and prevention of CVD events did not occur until 2009 (Friedewald *et al.*, 2009).

2.2. Econometric model

The above theoretical model suggests an IV strategy where dental care in the cardiovascular-health production function is identified by exogenous variables in the demand-for-dental-care equation (Winship and Morgan, 1999). Using this strategy, it is possible to estimate the ATE of dental care on CVD events even in the presence of heterogeneity in treatment effects or self-section into treatment. The ATE estimates the average gain of undergoing treatment as compared with no treatment.

Following the application of Heckman *et al.* (2006) by Basu *et al.* (2007), a model of heterogeneous outcomes, selection, and the ATE can be summarized. We compare the case of receiving dental care ($j = 1$), the case of not receiving dental care ($j = 0$), and their corresponding cardiovascular outcomes,

$$CVDE_1 = \mu_1(X) + U_1 \tag{3}$$

$$CVDE_0 = \mu_0(X) + U_0 \tag{4}$$

where $\mu_j(X)$ is a function of characteristics (X) that both the analysts (ourselves) and patients observe and U_j are characteristics that are observed by the patients, but not by the analysts. Thus, U_j refers to *unobserved heterogeneity*. We can interpret $\mu_j(X)$ as the conditional expectation of the outcome or

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$E(\text{CVD}_j|X = x) = \mu_j(X)$ under the assumption $E(U_j|X = x) = 0$, $j = 0, 1$. The individual gain from dental care, $\Delta = CVDE_1 – CVDE_0$, has two parts: (1) the ATE ($\text{ATE} = \mu_1(X) – \mu_0(X)$), or the average cardiovascular benefit for a person with characteristics X, and (2) $(U_1 – U_0)$, the idiosyncratic cardiovascular benefit to a particular person receiving dental care.

The treatment effect on the treated (TT), which is the average gain to those selecting treatment, can be compared with the ATE to clarify when self-selection and heterogeneity of treatment effects matter. The TT is defined as follows:

$$\text{TT}(x) = E(\Delta|X = x, D = 1) = (\mu_1(x) - \mu_0(x)) + E(U_1 - U_0|X = x, D = 1) \tag{5}$$

where D takes a value of 1 if an individual selects dental care and 0 otherwise. If the term $E(U_1 – U_0|X = x, D = 1)$ in Equation (5) is constant across the population, then ATE = TT, conditional on X. In other words, only in the case where unobserved cardiovascular benefits vary with selection into dental care does the distinction between TT and ATE matter.

2.2.1. Using ordinary least squares with selection on unobserved and heterogeneous gains. To illustrate the potential bias that occurs when conventional methods are used in the presence of selection on unobserved and heterogeneous gains, still following Basu *et al.* (2007), consider the following. As each individual either receives dental care, D, or does not, the outcome variable, $CVDE$, can be represented as

$$CVDE = (D)CVDE_1 + (1 - D)CVDE_0 \tag{6}$$

Substituting Equations (3) and (4) into Equation (6) we obtain

$$\begin{aligned} CVDE &= \mu_0(X) + D(\mu_1(X) - \mu_0(X)) + \{D(U_1 - U_0) + U_0\} \\ &= \mu_0(X) + D[E(\Delta|X)] + \{D(U_1 - U_0) + U_0\} \end{aligned} \tag{7}$$

which can be interpreted as a regression where $CVDE$ is regressed on X and D (interacted with the covariates) with $D(U_1 – U_0) + U_0$ being the error term. The error term in Equation (7) depends on the treatment variable which makes this estimator biased when estimated using ordinary least squares (OLS). The OLS estimator of the dental care effect is the difference in the adjusted mean cardiovascular outcomes for treated and untreated individuals, which can be written as follows:

$$\begin{aligned} &E(CVDE|X = x, D = 1) - E(CVDE|X = x, D = 0) \\ &\quad = [(\mu_1(X) + E(U_1|X = x, D = 1)] - [(\mu_0(X) + E(U_0|X = x, D = 1)] \\ &\quad = E(\Delta|X) + [E(U_1 - U_0)|X = x, D = 1) + \{E(U_0|X = x, D = 1) - E(U_0|X = x, D = 0)\}] \end{aligned} \tag{8}$$

The term in square brackets in Equation (8) is the bias due to selection into treatment based on the idiosyncratic outcomes and contains two components: the idiosyncratic gain for those treated and the difference between those treated and those untreated.

Using this framework, it can be shown that using the method of IV, TT = ATE except for the case of selection into dental care based on the unobserved heterogeneous cardiovascular benefits. Formally, an instrument, Z, must meet the following assumptions:

A1. The probability of choosing dental care is a function of the instrument Z conditional on X, i.e. $\Pr(DDS|X = x, Z = z) \neq \Pr(DDS|X = x)$,
A2. The instrument Z is mean independent of the error terms in Equations (3) and (4) conditional on X, i.e. $E(U_1|X = x, Z = z) = 0$ and $E(U_0|X = x, Z = z) = 0$.

According to Basu *et al.* (2007), under these assumptions, three possible situations arise with respect to selection effects and heterogeneity in treatment effects.

(a) No unobserved heterogeneity. This refers to the situation where $U_1 = U_0$. In other words, all individuals receive the same protection from CVD events from dental treatment (conditional on X).

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Therefore, TT = ATE. Because the error term reduces to U_0, selection bias affects only the estimates of D through a dependence of D with U_0. Thus, instruments that meet the conditions of assumptions 1 and 2 would remove this dependence and allow the ATE to be identified.

(b) Unobserved heterogeneity is present, but selection into treatment does not depend on unobserved heterogeneity. This is non-essential heterogeneity. Formally, $U_1 \neq U_0$, but D is statistically independent of $(U_1 - U_0)$ given X. In other words, individuals with the same X respond differently to treatment, but do not choose treatment based on these idiosyncratic differences in treatment outcomes. For example, individuals may receive different levels of cardiovascular benefit in response to receiving dental care, but may not know this at the time they choose whether or not to obtain dental care. Because the distribution of $(U_1 - U_0)$ is not dependent on the treatment status of individuals, the expected idiosyncratic gain in Equation (8) is zero: $E(U_1 - U_0)|X = x,\ D = 1) = E(U_1 - U_0)|X = x) = 0$. Thus, TT = ATE. Just as above, the error term reduces to U_0, and hence selection affects only the estimates of D through a dependence of D with U_0. As above, instruments that meet the conditions of assumptions 1 and 2 would remove this dependence and allow the ATE to be identified. This situation would continue across time if selection into treatment is not based on the expected idiosyncratic differences in treatment outcomes. This is true even when idiosyncratic differences in treatment outcomes are subsequently observed by patients if such differences are not attributed to dental care.

(c) Essential heterogeneity: unobserved heterogeneity where selection into treatment is based on the unobserved benefits. This is the case where $U_1 \neq U_0$ and D is statistically dependent on $(U_1 - U_0)$ given X. In other words, individuals receive different cardiovascular benefits from receiving dental care, and their knowledge of these benefits influences their choice to receive dental care. In this situation, the method of IV does not, in general, estimate the ATE or the TT (Heckman *et al.*, 2006).

Heckman *et al.* (2006) have developed a test for essential heterogeneity which consists of estimating the marginal treatment effect (MTE) and determining whether this differs across individuals whose margins differ. In our application, the MTE is the average cardiovascular benefit to individuals who are indifferent between receiving and not receiving dental care. As the name implies, these are individuals at the margin as defined by X and Z. Since values of Z vary across individuals, the MTE will also vary. The test involves using a probit model to estimate the propensity of individuals to receive dental care conditional on X and Z, estimating the MTE where X is included along with the propensity score interacted with X, and then adding the square, cubic, and quartic of the propensity score, sequentially, to the MTE estimation (estimated using OLS). If any polynomial terms are statistically significant, then essential heterogeneity is considered to be present (for details, see Basu *et al.*, 2007).

Note that this approach may yield estimates different from the local average treatment effect (LATE) approach, where LATE is defined as the mean outcome for persons induced to participate in treatment by a change in the value of the instrument (Imbens and Angrist, 1994). Different instruments determine different treatment effects. Although this is extremely useful in many contexts, Basu *et al.* (2007) recommend using all available valid instruments when the goal is to identify all margins of choice.

2.2.2. Model specification. Based on Equation (1), we empirically specify a cardiovascular-health production function as follows:

$$\Pr(CVDE_t) = \beta_0 + \beta_1 D_t + \beta_2 R_{t-1} + \beta_3 DDS_{t-1} + \beta_4 TIME + \varepsilon \tag{9}$$

where CVD is the occurrence of any CVD event listed above (heart attack, stroke, angina, congestive heart failure, death from heart attack or stroke). The vector D includes demographic characteristics including age (less than 55, 55–59, 60–64, 65–69, 70–74, 75–79, 80 or older), race-ethnicity (White, African-American, Hispanic, other race), birth place (foreign born, US born), and education (less than high school, high school/GED, some college, college and above). The vector R contains non-demographic risk factors for CVD. Included are self-reported health status (excellent, very good, good, fair, poor), (underweight/normal (<25), overweight (25–30), obese (>30)), alcohol use (does not drink,

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1 drink or less per day, 1–2 drinks per day, 3 or more drinks per day), smoking status (never smoked, past smoker, current smoker), diabetes status (not told by doctor, told by doctor), high blood pressure (not told by doctor, told by doctor), participation in vigorous physical activity 3 or more times per week (no, yes), and whether an individual had three or more depressive symptoms using the abridged 8-point Center for Epidemiologic Studies Depression Scale (CES-D) (Turvey *et al.*, 1999; Rice *et al.*, 2009). The variable *DDS* is a dummy that indicates whether an individual received dental care. The vector T contains biennial time dummies.

The error term, ε, contains unobserved factors that may affect the likelihood of CVD events such as poor diet, stress, hyperlipidemia, family history, non-dental pharmaceutical interventions, and non-dental surgical interventions. The extent to which these factors also correlate with the choice to receive dental care, after conditioning on covariates, is the extent to which the parameter estimates for DDS may be biased. Family history is unlikely to have any correlation with *DDS*. Poor diet, stress, and hyperlipidemia are risk factors positively correlated with *CVDE* and are likely to be negatively correlated with DDS (those with poor health habits are less likely to seek preventative dental care) inducing a negative bias in β_3. However, any correlation of poor diet and hyperlipidemia with *CVDE* is likely to be reduced or eliminated by the presence of measured items such as health status, BMI, and exercise, whereas the correlation of stress with *CVDE* is likely to be reduced or eliminated by a person's measured CES-D score. This would likely reduce or eliminate any negative bias in β_3.

On the contrary, factors such as cardiovascular-relevant pharmaceutical interventions and cardiovascular-relevant surgical interventions are likely to be negatively correlated with *CVDE* and negatively correlated with *DDS* as such factors indicate CVD problems and patients with less stable CVD may avoid dental procedures known to increase the risk of CVD events (Niwa *et al.*, 2000; Hupp, 2006; Joshipura *et al.*, 2006; Wilson *et al.*, 2007). This would induce a positive bias in β_3. Thus, although we are unable to definitively sign any potential bias to β_3, it is likely that the uncorrected OLS estimates of β_3 will be positively biased, obscuring the negative effect of *DDS* on *CVDE*.

In order to determine appropriate instruments for *DDS* in the cardiovascular-health production function, we also empirically specify a demand-for-dental-care equation based on Equation (2):

$$\Pr(DDS_t) = \alpha_0 + \alpha_1 D_t + \alpha_2 SH_t + \alpha_2 SD_{t-1} + \alpha_3 \ln Y_t + \alpha_4 TIME + \eta \tag{10}$$

Compared with Equation (2), Equation (10) does not contain dental insurance (*INS*) or price (P) due to data limitations. In addition, for the taste vector (T in Equation (2)), we include the following influences on individuals' preferences, D, defined above, spousal health status (excellent, very good, good, fair, poor), *SH*, and spousal use of dental care in the previous period, *SD*. It is reasonable that individuals whose spouse is in poor health may choose to receive dental care less often due to the burden of caring for their spouse. It is also reasonable that individuals may be influenced to obtain dental care if their spouse receives it. The vector Y contains a measure of overall wealth: the value of a family's financial instruments. The error term, η, contains dental insurance, price, knowledge about the value of dental care, and other factors not observed that may affect an individual's decision to obtain dental care.

The first-stage equation to estimate the predicted value of *DDS* for inclusion in Equation (9) would include all of the exogenous variables from Equations (9) and (10):

$$\Pr(DDS_t) = \gamma_0 + \gamma_1 D_t + \gamma_2 SH_t + \gamma_3 SD_{t-1} + \gamma_4 R_{t-1} + \gamma_5 Y_t + \gamma_6 TIME + \eta \tag{11}$$

Note that the instruments, SH_t, SD_{t-1}, in Equation (11) are assumed not to be related to the types of things that are picked up by the error term in Equation (9) after conditioning on the covariates in Equation (9). Although spouses tend to have similar lifestyles, with poor health status being related to poor health habits that both spouses share, any potential correlation between SH_t or SD_{t-1} and poor health habits in the error term in Equation (9) is likely to have been conditioned out by the inclusion of health status, BMI, smoking status, and alcohol use. Similarly, the instrument Y may be correlated to poor diet, stress, and hyperlipidemia in the error term in Equation (9) because wealthier individuals are

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more likely to be able to afford better diets and to have less stressful lives. Yet, again, any such potential correlations are likely to have been conditioned out due to terms included in Equation (9). We perform overidentification tests to evaluate this.

We estimate Equations (9) and (11) simultaneously using two approaches. Because tests of the strength of IVs (Stock and Yogo, 2005), the exogeneity of IVs (Hayashi, 2000), and essential heterogeneity using IVs (Heckman *et al*., 2006; Basu *et al*., 2007) are all well developed for two-stage least squares (2SLS), we use 2SLS to conduct these tests (Baum *et al*., 2007). In addition, we test for first-order autocorrelation in the 2SLS estimation using the procedure suggested by Wooldridge (2009).

However, since the linearity assumption in linear probability models may result in predictions greater than one or less than zero, we avoid this problem by also estimating identically specified bivariate probit models which estimate recursive simultaneous probit models that are structurally similar to the 2SLS model (Brown *et al*., 2005; Nguyen *et al*., 2007; Greene, 2003; Maddala, 1983). In addition, in order to obtain appropriate confidence intervals for the ATE using bivariate probit, we use *biprobittreat*, developed by Andrabi *et al*. (2007) and available from Chiburis (2010). See also Babalola and Kincaid (2009).

Our specification follows the recommendation that when attempting to control for the correlation of observations within time periods and the correlation of observations within clusters that one control for the former parametrically and for the latter using clustered standard errors (Petersen, 2009). We control for time parametrically and for probability sampling units using clustered standard errors. Each 2SLS estimation accounts for probability weighting, probability sampling units (clusters), and heteroscedasticity. Each bivariate probit estimation accounts for probability sampling units, stratification, and uses bootstrapping (500 replications) to obtain appropriate standard errors for each parameter and for the ATE.

2.3. Data

We use the Health and Retirement Study (HRS), a biennial longitudinal survey of individuals aged 50 and over (and their spouses) (Juster and Suzman, 1995). Spouses may be younger than 50. The HRS has been used to study the relationship between CVD and various demographic and economic characteristics, health conditions, and health habits (Oldridge *et al*., 2001; Adams *et al*., 2003; Gallo *et al*., 2004; Gallo *et al*., 2006).

We choose the HRS because it surveys individuals within an age range during which CVD events are more likely, has a panel structure, and contains information on dental visits, cardiovascular outcomes, and risk factors (Bromberger *et al*., 1997; American Heart Association, 2009). We use data from the 1996, 1998, 2000, 2002, and 2004 waves.

Our dependent variable contains information on whether an individual has experienced any of the CVD events listed above since the previous wave. Our independent variable of interest is whether an individual has visited the dentist since the previous wave. We have no information on the nature of these visits. Based on the literature above, visits that include procedures that reduce inflammation may reduce the risk of CVD events. However, as noted above, other literature suggests that some procedures may increase the risk of CVD events. Thus, a dental visit may contain elements that both increase and decrease the risk of subsequent cardiovascular events.

About 79% of adults (18 years and older) and 75% of older adults (65 years and older) who visited the dentist over a 1-year period received preventive services (cleaning, fluoride, or sealant) (Manski and Brown, 2007). It is therefore likely that most dental visits involve some type of procedure that seeks to improve or maintain the condition of the periodontium. These are the procedures expected to be associated with an improved cardiovascular risk profile. Owing to the use of spousal information for two of the three instruments, we restrict the sample to married individuals.

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3. RESULTS

Descriptive statistics by sex and the receipt of dental care one period prior are summarized in Table I. Comparing those who received dental care with those who did not, we find that those who received dental care are more educated and have fewer risk factors, underlining the importance of correcting for omitted variable bias.

First-stage estimates are listed in Table II. Weak instrument tests of the identifying instruments yield a partial *F*-statistic of 67.69 for women and 84.88 for men each relative to the critical value of 13.91 for

Table I. Descriptive statistics by use of dental care in previous period (means)

Variables	Women		Men	
Dental Care (*previous period*)	0.735		0.687	
	Dental	*No dental*	*Dental*	*No dental*
CVD event	0.016	0.030	0.030	0.045
Demographics				
Age (years)				
<55	0.068	0.063	0.026	0.024
55–59	0.323	0.281	0.230	0.197
60–64	0.287	0.284	0.286	0.279
65–69	0.232	0.254	0.249	0.258
70–74	0.083	0.098	0.153	0.179
75–79	0.006	0.015	0.048	0.049
≥ 80	0.001	0.005	0.008	0.014
Race/ethnicity				
White	0.912	0.792	0.897	0.807
Black	0.041	0.097	0.039	0.081
Hispanic	0.046	0.085	0.043	0.086
Other	0.016	0.026	0.021	0.026
Education				
Less than high school	0.337	0.335	0.096	0.290
High school/GED	0.394	0.451	0.302	0.392
Some college	0.270	0.159	0.216	0.175
College or above	0.238	0.056	0.386	0.143
US born	0.908	0.911	0.926	0.919
Risk factors				
Self-reported health status (1–5)	2.363	2.947	2.418	2.858
BMI				
Under/normal weight (<25 BMI)	0.398	0.301	0.234	0.245
Overweight (25–30 BMI)	0.352	0.339	0.497	0.443
Obese (>30 BMI)	0.238	0.350	0.267	0.310
Alcohol use				
Does not drink	0.664	0.830	0.513	0.653
One drink or less per day	0.191	0.083	0.175	0.112
1–2 drinks per day	0.111	0.055	0.185	0.107
3 or more drinks per day	0.145	0.032	0.127	0.128
Smoking status				
Never smoked	0.532	0.450	0.330	0.250
Past smoker	0.350	0.314	0.550	0.523
Current smoker	0.118	0.236	0.120	0.227
Diabetes	0.088	0.171	0.137	0.184
High blood pressure	0.385	0.483	0.458	0.483
Vigorous activity 3+ times/week	0.490	0.406	0.577	0.489
Abridged CES-D score (≥ 3)	0.156	0.283	0.111	0.178
Instruments				
Financial instruments ($100 000s)	2.955	0.700	3.032	1.086
Spouse health > poor	0.965	0.899	0.976	0.925
Spousal dental visit (-2 periods)	0.782	0.452	0.850	0.565
Observations	6035	2453	5273	2726

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Table II. First-stage estimates – dental care one period prior (all exogenous variables from demand for dental care and health production functions)

	Women		Men	
Variables	Coefficients	*t*-statistics	Coefficients	*t*-Statistics
Demographics				
Age (years) (reference: < 55)				
55–59	0.024	0.87	−0.014	−0.25
60–64	0.021	0.81	−0.022	−0.40
65–69	−0.005	−0.18	−0.034	−0.62
70–74	−0.017	−0.54	−0.049	−0.90
75–79	−0.113	−1.49	−0.020	−0.35
≥80	−0.191	−1.25	−0.096	−1.16
Race/ethnicity (reference: white)				
Black	−0.085	−4.87**	−0.066	−2.15*
Hispanic	−0.039	−1.79	−0.057	−2.03*
Other	−0.119	−2.55**	−0.063	−1.69
US born (reference: foreign born)	0.093	4.57**	0.040	1.73
Education (reference: less than high school)				
High school/GED	0.165	7.01**	0.113	5.07**
Some college	0.242	8.42**	0.187	6.76**
College or above	0.276	9.91**	0.255	10.21**
Risk Factors (one period prior)				
Health status (1–5: 1 is excellent)	−0.027	−4.45**	−0.026	−4.00**
Overweight (25–30 BMI) (reference: < 25 BMI)	0.000	0.02	0.040	2.29*
Obese (> 30 BMI)	−0.033	−1.97*	0.015	0.77
Does not drink (reference: 3+ drinks/day)	−0.020	−0.62	−0.018	−0.85
One drink or less per day	0.021	0.63	0.008	0.31
1–2 drinks per day	0.026	0.83	0.042	1.77
Past smoker (reference: never smoked)	−0.032	−2.06*	−0.014	−0.99
Current smoker	−0.128	−5.27**	−0.099	−4.56**
Diabetes (reference: none)	−0.038	−1.52	−0.006	−0.30
High blood pressure (reference: none)	−0.004	−0.37	0.006	0.46
Vigorous activity 3+/week (reference: none)	−0.005	−0.54	0.032	2.59**
Abridged CES-D score (≥3)	−0.031	−1.80	−0.005	−0.28
Time period (reference: 2000)				
2002	−0.001	−0.11	−0.010	−0.83
2004	−0.013	−1.00	−0.029	−2.64**
Instruments				
Financial instruments ($100 000s)	0.004	4.10**	0.003	3.65**
Spousal dental visit (−2 periods)	0.203	13.68**	0.227	14.43**
Spouse health > poor	0.075	2.62**	0.118	3.91**
Constant	0.462	7.65**	0.336	5.42**
R^2	0.21		0.18	
F-statistic	74.42**		36.78**	
Observations	8488		7999	

First-stage of two-stage least squares. *Statistically significant at the 5% level (two-tailed *t*-test); **statistically significant at the 1% level (two-tailed *t*-test); BMI, Body Mass Index; CES-D, Center for Epidemiologic Studies Depression Scale (8-point scale); GED, General Educational Development test.

5% maximal IV relative bias, rejecting the null hypothesis that the instruments are weak (Stock and Yogo, 2005).

Second-stage 2SLS estimates are listed in Tables III and IV. Tests of the exogeneity of the overidentifying instruments fail to reject exogeneity, yielding a Hansen *J*-statistic of 0.29 ($p = 0.86$) for women and a Hansen *J*-statistic of 1.53 ($p = 0.47$) for men. We find no evidence of first-order autocorrelation in the equations for men or women.

Table V summarizes the results of tests for essential heterogeneity. The relevant test statistic is the partial *F*-statistic for the higher order propensity scores. For both women and men, no set of higher order propensity scores were singly or jointly statistically significant. For the ATE to be fully identified,

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Table III. Cardiovascular disease events among married women

	OLS		Second-Stage 2SLS	
Variables	Coefficients	*t*-Statistics	Coefficients	*t*-Statistics
Dental use				
Dental care one period prior	−0.006	−1.44	−0.044	−2.57**
Demographics				
Age (years) (reference: < 55)				
55–59	0.006	1.27	0.007	1.51
60–64	0.006	1.31	0.007	1.57
65–69	0.013	2.46*	0.013	2.60**
70–74	0.027	3.15**	0.026	3.08**
75–79	0.002	0.13	−0.003	−0.17
≥80	−0.020	−1.88	−0.027	−2.53**
Race/ethnicity (reference: white)				
Black	0.006	0.63	0.002	0.21
Hispanic	0.002	0.42	0.000	−0.08
Other	0.013	0.68	0.008	0.43
US born (reference: foreign born)	−0.003	−0.56	0.000	0.06
Education (reference: less than high school)				
High school/GED	0.003	0.58	0.010	1.96*
Some college	0.007	1.05	0.018	2.39*
College or above	0.001	0.12	0.014	1.87
Risk Factors (one period prior)				
Health status (1–5: 1 is excellent)	0.006	3.46**	0.005	2.66**
Overweight (25–30 BMI) (reference: < 25 BMI)	0.002	0.48	0.001	0.41
Obese (> 30 BMI)	−0.001	−0.31	−0.003	−0.67
Does not drink (reference: 3+drinks/day)	0.002	0.20	0.0005	0.06
One drink or less per day	−0.002	−0.26	−0.001	−0.14
1–2 drinks per day	−0.005	−0.52	−0.003	−0.38
Past smoker (reference: never smoked)	0.007	1.96*	0.006	1.60
Current smoker	0.011	1.91	0.005	0.76
Diabetes (reference: none)	0.011	1.71	0.009	1.46
High blood pressure (reference: none)	0.012	2.67**	0.012	2.72**
Vigorous activity 3+/week (reference: none)	−0.001	−0.24	−0.001	−0.27
Abridged CES-D score (≥3)	0.006	1.20	0.005	0.92
Time period (reference: 2000)				
2002	−0.005	−1.17	−0.005	−1.18
2004	−0.011	−2.80**	−0.012	−2.94**
Constant	−0.009	−0.82	0.017	0.97
F-statistic	4.85**		3.66**	
Observations	8488		8488	

Second-stage of 2SLS. *Note*: R^2 has no meaning in second-stage estimates and is therefore not reported. *Statistically significant at the 5% level (two-tailed *t*-test); **statistically significant at the 1% level (two-tailed *t*-test); BMI, Body Mass Index; CES-D, Center for Epidemiologic Studies Depression Scale (8-point scale); GED, General Educational Development test.

it must be the case that the propensity score gives adequate support over the [0, 1] interval. Using probit models, our propensity score covers [0.056, 1.0] for women and [0.065, 1.0] for men, showing that we have virtually full support. We thus infer that estimates using our instrument set represent the ATE of dental care on CVD events. For women, this effect is −0.04 [95% confidence interval: −0.08, −0.01]; we find no evidence of a treatment effect for men.

We also estimate the ATE using bivariate probit models, following Angrist and Pischke (2009), and bootstrap our estimate (500 repetitions) in order to obtain appropriate confidence intervals (Chiburis, 2010). Table VI shows that for women, the ATE is −0.03 [95% confidence interval: −0.06, −0.01]. As the lower end of the confidence interval using either method is −0.01, the evidence suggests that, at minimum, the probability of experiencing a CVD event for women who currently do not receive regular dental care is reduced by at least one-third when these individuals receive dental care and possibly more. As with the 2SLS estimates, there is no detectable treatment effect for men (data not shown).

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Table IV. Cardiovascular disease events among married men

Variables	OLS		Second-Stage 2SLS	
	Coefficients	*t*-Statistics	Coefficients	*t*-Statistics
Dental use				
Dental care one period prior	−0.012	−1.84	−0.012	−0.48
Demographics				
Age (years) (reference: < 55)				
55–59	−0.017	−0.65	−0.017	−0.65
60–64	−0.018	−0.70	−0.018	−0.70
65–69	−0.010	−0.38	−0.010	−0.38
70–74	−0.020	−0.77	−0.020	−0.78
75–79	−0.003	−0.09	−0.003	−0.09
≥80	0.040	0.94	0.040	0.97
Race/ethnicity (reference: white)				
Black	−0.026	−3.52**	−0.026	−3.59**
Hispanic	−0.013	−1.94	−0.013	−1.87
Other	0.015	1.00	0.015	1.01
US born (reference: foreign born)	0.002	0.25	0.002	0.24
Education (reference: less than high school)				
High school/GED	0.004	0.51	0.004	0.44
Some college	0.011	1.09	0.011	0.92
College or above	0.011	1.22	0.011	0.87
Risk Factors (one period prior)				
Health status (1–5: 1 is excellent)	0.008	2.36*	0.008	2.27*
Overweight (25–30 BMI) (reference: < 25 BMI)	0.007	1.32	0.007	1.35
Obese (> 30 BMI)	0.002	0.33	0.002	0.33
Does not drink (reference: 3+drinks/day)	0.016	1.93	0.016	1.94
One drink or less per day	0.007	0.74	0.007	0.74
1–2 drinks per day	0.001	0.12	0.001	0.12
Past smoker (reference: never smoked)	0.007	1.66	0.007	1.66
Current smoker	0.025	2.89**	0.025	2.72**
Diabetes (reference: none)	0.010	1.34	0.010	1.34
High blood pressure (reference: none)	0.002	0.35	0.002	0.36
Vigorous activity 3+/week (reference: none)	−0.001	−0.14	−0.001	−0.15
Abridged CES-D score (≥3)	−0.006	−0.84	−0.006	−0.85
Time period (reference: 2000)				
2002	−0.005	−0.88	−0.005	−0.88
2004	−0.007	−1.03	−0.007	−1.00
Constant	0.013	0.42	0.013	0.42
F-statistic	4.73**		4.67**	
Observations	7999		7999	

Second-stage of 2SLS. *Note*: R^2 has no meaning in second-stage estimates and is therefore not reported. *Statistically significant at the 5% level (two-tailed *t*-test); **statistically significant at the 1% level (two-tailed *t*-test); BMI, Body Mass Index; CES-D, Center for Epidemiologic Studies Depression Scale (8-point scale); GED, General Educational Development test.

Table V. Heterogeneity tests using all three instrumental variables

Propensity Score	Alternative specifications of propensity scores					
	Women			Men		
	Quadratic	*Cubic*	*Quartic*	*Quadratic*	*Cubic*	*Quartic*
p^2	0.075	0.305	1.150	0.004	−0.520	0.708
	(1.05)	(0.85)	(1.09)	(0.04)	(−1.69)	(0.48)
p^3		−0.121	−1.566		0.312	−1.255
		(−0.66)	(−1.04)		(1.69)	(−0.65)
p^4			0.609			0.696
			(1.03)			(0.81)
X^{2*} (*p*-value)	1.10 (0.29)	1.51 (0.47)	2.15 (0.54)	0.00 (0.97)	2.93 (0.23)	3.22 (0.34)

t-Statistics in parentheses except where noted. Standard errors computed using bootstrapping with 500 replications. *Joint test of higher order polynomials of propensity score *p*. *Note*: this test incorporated propensity scores from a probit model.

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Table VI. Cardiovascular disease events among married women – bivariate probit

	Dental Care		Cardiovascular Events	
Variables	Coefficients	*z*-Statistics	Coefficients	*z*-Statistics
Dental use				
Dental care one period prior	—	—	−0.541	−3.65**
Demographics				
Age (years) (reference: < 55)				
55–59	−0.030	−0.48	0.177	1.26
60–64	−0.038	−0.60	0.165	1.16
65–69	−0.163	−2.35*	0.313	2.23*
70–74	−0.211	−2.40*	0.519	3.21**
75–79	−0.320	−1.94	−0.011	0.00
≥80	−0.762	−1.16	−5.217	-20.41**
Race/ethnicity (reference: white)				
Black	−0.114	−2.11*	0.046	0.63
Hispanic	−0.076	−1.57	−0.001	−0.03
Other	−0.308	−3.12**	0.115	0.69
US born (reference: foreign born)	0.252	4.77**	0.027	0.34
Education (reference: less than high school)				
High school/GED	0.397	10.35**	0.166	2.47**
Some college	0.666	12.68**	0.283	3.13**
College or above	0.880	16.28**	0.170	1.75
Risk Factors (one period prior)				
Health status (1–5: 1 is excellent)	−0.081	−5.47**	0.130	4.18**
Overweight (25–30 BMI) (reference: < 25 BMI)	0.027	0.73	0.068	1.41
Obese (> 30 BMI)	−0.087	−2.26*	0.025	0.37
Does not drink (reference: 3+drinks/day)	−0.058	−0.67	0.013	0.09
One drink or less per day	0.122	1.36	−0.035	−0.21
1–2 drinks per day	0.121	1.43	−0.134	−0.68
Past smoker (reference: never smoked)	−0.110	−2.40*	0.165	3.41**
Current smoker	−0.380	−8.84**	0.187	2.98**
Diabetes (reference: none)	−0.048	−1.10	0.149	2.59**
High blood pressure (reference: none)	−0.029	−1.00	0.210	3.68**
Vigorous activity 3+/week (reference: none)	0.043	1.65	−0.053	−1.10
Abridged CES-D score (≥3)	−0.048	−1.32	0.153	2.78**
Time period (reference: 2000)				
2002	−0.028	−1.23	−0.080	−1.46
2004	−0.062	−2.39*	−0.206	−3.79**
Instruments				
Financial instruments ($100 000 s)	0.107	12.78**	—	—
Spousal dental visit (−2 periods)	0.570	19.22**	—	—
Spouse health > poor	0.184	3.44**	—	—
Constant	−0.044	−0.32	−2.598	−9.10**
X^2	23 502.02**			
Observations	8488			
ATE (95% confidence interval)	−0.034 [−0.059, −0.009]			

Standard errors computed using bootstrapping with 500 replications. *Statistically significant at the 5% level (two-tailed *z*-test); **statistically significant at the 1% level (two-tailed *z*-test); BMI, Body Mass Index; CES-D, Center for Epidemiologic Studies Depression Scale (8-point scale); GED, General Educational Development test.

Both the 2SLS model (Table III) and the bivariate probit model (Table VI) show an inverted-U effect with respect to age and CVD events among women as well as showing no differences across race/ethnicity or birthplace. Both models also show similar effects for education and time period. Differences between the models do occur among risk factors with the bivariate probit model, showing positive associations with CVD events for worse health status, smoking (past and present), diabetes, high blood pressure, and depression, whereas the 2SLS model picks up only worse health status and high blood pressure. This suggests that the bivariate probit model fits the data better than the 2SLS model.

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4. DISCUSSION

To determine the effect of dental care on CVD events, we obtained panel data on individuals aged 44 years and older and estimated a cardiovascular-health production function using IV to control for potential omitted variable bias and attenuation bias with respect to dental care. We also performed the Heckman *et al*. (2006) test for essential heterogeneity in order to determine if our results represent the ATE.

We find that, for women, the ATE on CVD events (heart attack, stroke, angina, congestive heart failure, and death from heart attack or stroke) from receiving dental care is at least -0.01. In other words, dental care is causally associated with a reduction in CVD events of at least one-third. We find no treatment effect for men. We attribute this difference to the differing ways in which CVD develops and is treated in women relative to men.

The primary difference in the development of CVD between the sexes is that women tend to develop CVD approximately 10 years later than men due to the hormonal protection women receive from estrogen until the time of menopause, and other biological reasons (Mendelsohn and Karas, 2005). We suggest that this may allow preventative dental procedures to have a greater effect on the development of CVD in women than men in this cohort since preventive measures may be more effective when the disease is just beginning. If the effect observed in women is due to intervening in the development of CVD at an earlier stage, then presumably, studying the effect of increased dental care in younger men might also find an association between dental care and the reduction of CVD events.

Limitations of this study include the lack of information on some variables in both the cardiovascular-production function and the demand-for-dental-care function as well as a lack of information on the dental procedures performed during each visit. Although theoretically the results should not be different if these variables were included, it is possible empirically that their inclusion could affect the magnitude of the effect measured. Owing to the size of this effect, research using other data sets should be performed to confirm these findings.

Research on dental care and its relationship to non-oral health conditions is yielding important results. Economists can contribute by applying their skills to the many observational data sets available. In addition, partnerships with dental and medical researchers can focus economists on the most productive research areas.

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Ethics: A description of this research has been submitted to the Office of the Protection of Human Subjects at the University of California at Berkeley and has been granted exempt status.

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