

Health Recommendations and Selection in Health Behaviors*

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Abstract

Consider a case in which a new research finding links a health behavior with good health outcomes. A possible consequence is take-up of this behavior among individuals who engage in other positive health behaviors. If this occurs, later analyses of observational data may be biased by the change in selection. This paper evaluates these dynamic biases in empirical settings. Using data from vitamin supplementation and diet I show that selection responds endogenously to health recommendations. These results highlight how spurious findings on health behaviors can be self-reinforcing.

The starting point for this paper is two facts about health behavior.

First, adherence to health recommendations varies systematically across people. Positive health behaviors tend to cluster – people who exercise are also less likely to smoke, for example – and they also correlate with education and income (e.g. Berrigan et al, 2003; Friel, Newell and Kelleher, 2005; Finke and Huston, 2003; Kirkpatrick et al, 2012; Cutler and Lleras-Muney, 2010; Cutler, Lleras-Muney and Vogl, 2008; Goldman and Smith, 2002). These adherence differences may generate bias in estimated links between behaviors and health outcomes in observational data (e.g. Greenland et al, 1999; Vandenbroucke et al, 2007).

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Second, health recommendations – about the best diet, the optimal amount of exercise, vitamin supplements – often change over time in response to new information or changes in expert opinion.

This paper connects these two facts and argues that in the presence of differential *response* to new health advice, bias in estimates of the impact of health behaviors on health outcomes may be dynamic.

To be concrete, consider a hypothetical case in which researchers are evaluating the relationship between pineapple and cardiovascular health. Imagine that although the true effect is zero, sampling variability leads to a study showing that pineapples significantly reduce heart attacks. One result may be positive pineapple-related news coverage or even a change in official guidelines about pineapple consumption. In response, some people will increase their consumption of pineapple. These may be the people who are most concerned about their health. But this group is also likely to be engaged in other heart-healthy behaviors (exercise, not smoking, etc). As a result of this differential adoption of the recommendation, later observational studies of the pineapple-heart health relationship may see a more substantial link between pineapple and health, since a bias has now been created by changes in selection.

If dynamics like this are present and quantitatively important, it bodes especially poorly for our ability to learn about null effects. False positive or negative results are an inevitable part of statistical analysis, even with randomized trials. If such results are self-reinforcing, we cannot rely on later observational evidence to correct these statistical accidents.

That these dynamics *could* arise follows directly from the two motivating facts. It is less obvious that they do arise or will be quantitatively important. The goal of this paper, therefore, is to explore these dynamics empirically.

I present data from a number of settings. I first describe detailed evidence on a single case – vitamin E – where there have been sharp changes in recommendations over time and I observe evidence on behavior, selection patterns and health outcomes. I then show corroborating, suggestive evidence from other examples, including Vitamin D and several dietary patterns.

Section I describes an empirical model of the research process which highlights the circumstances under which these dynamics may be important. Of note in this discussion is that a key assumption underlying my interpretation of the results is that the treatment effects considered are homogeneous. In the absence of this assumption, changes in the relationship between the behavior and health outcomes over time could reflect heterogeneous treatment effects, not changes in selection. I discuss this issue in more detail below.

Section II describes the data used in the paper. I make use of three survey datasets. First, the National Health and Nutrition Examination Survey (“NHANES”), which is a repeated cross-sectional survey of a nationally representative sample of individuals. It includes data on health behaviors, objectively measured health outcomes and demographics. Second, the Nurses’ Health Study (“NHS”) which is a panel survey of female nurses which began in the 1970s. Data is collected by mail survey every two years, and information is available on health behaviors and some health outcomes, notably mortality. Third, the Nielsen HomeScan scanner panel which is a panel dataset of grocery, drugstore and other purchases among a (non-representative) set of households.

Section III analyzes Vitamin E. There are significant changes in recommendations over time. In the early 1990s several studies suggested vitamin E supplements could prevent heart disease and cancer; this led to an uptick in health advice around vitamin E supplementation. In 2004 new evidence - this time from a meta-analysis of randomized trials - suggested vitamin E supplements not only didn’t help but might actually *increase* mortality. Overall consumption of vitamin E responds strongly to these changes in information.

The selection patterns in vitamin E consumption also change over time. In the period when vitamin E is more recommended, there is a much stronger positive relationship between vitamin E consumption and education, income, exercise, not smoking and diet quality. For example, in the period before 1993, those who take vitamin E are 0.7 percentage points less likely to smoke. During the 1993 to 2004 period, those who take vitamin E are 4 percentage points less likely to smoke. After 2004, this falls again to 1.6 percentage points.

These changes in selection are reflected in changes in the relationship between supplementation and health outcomes. In the NHANES, I estimate a relationship between vitamin E and heart health, and show some (large but statistically imprecise) evidence that heart health is more positively related to vitamin E consumption in the 1993 to 2004 period than in the period before or after. In the Nurses’ Health Study, I estimate the relationship between vitamin E consumption and short-term mortality. In the period before 1993 taking vitamin E is associated with a 10 percent reduction in the risk of death over the next two years. After the positive health recommendation, this jumps to a significant 25 percent reduction in the mortality risk. After 2004, the coefficient on supplementation in the mortality regression is again around 10 percent.

In both cases - heart health in the NHANES and mortality in the Nurse Health Study - I show the results in raw correlations (adjusting only for age and gender) and with comprehensive controls. These include all of the selection variables discussed, plus additional ones (race, marital status in

the case of NHANES and a full set of disease controls for the Nurse Health Study). Including these controls does lower the variance across time, but it leaves the overall message virtually unchanged.

In Section IV I analyze several other settings: Vitamin D, sugar consumption, fat consumption and the Mediterranean diet. In these cases the movements in recommendations are more gradual, and the data is less complete. Nevertheless, I am able to look at many of the same dynamics.

Vitamin D becomes more favored in the mid-2000s with release of evidence suggesting a wide range of benefits, but in the early 2010s some additional findings suggested this might have been overblown. Consumption of the supplement in the data increases and then decreases corresponding to this timing. Selection in the behavior also follows this pattern, with initial increases in the link between Vitamin D and other positive health behaviors, income and education and then later decreases.

Diet recommendations have changed over time: sugar and fat have become less recommended, and the Mediterranean diet more so. There are corresponding changes in the selection of behavior. Those who decrease their sugar consumption over time are more likely to exercise, less likely to smoke, have higher income and more education. Similar patterns show up for fat and the Mediterranean diet (opposite in the latter case as it becomes more recommended over time).

When diet behaviors are more recommended, they are more strongly associated with lower BMI and better heart health. In the case of sugar, in the earliest period of the data, my sugar measure and BMI are uncorrelated. By the latest period of the data it is strongly associated with a higher BMI.

Overall, the evidence in these empirical sections suggests that these dynamics are quantitatively important for estimated correlations. As noted above, this may be especially problematic for null results, given that false positive and negative results are statistically inevitable and this suggests that they may be self-reinforcing.

The paper contributes to a large literature in economics on the relationship between socioeconomic status and adherence to health recommendations (e.g. Berrigan et al, 2003; Friel, Newell and Kelleher, 2005; Finke and Huston, 2003; Kirkpatrick et al, 2012; Cutler and Lleras-Muney, 2010; Cutler, Lleras-Muney and Vogl, 2008; Goldman and Smith, 2002; Kowalski, 2018) and on consumer response to health information (e.g. Cutler, 2004; Chern et al, 1995; Brown and Schrader, 1990; Chang and Just, 2007; Roosen et al, 2009; Kinnucan et al, 1997; Ippolito and Mathios, 1995; Einav et al, 2019).

I Empirical Framework

In this section I formalize the statistical model for the dynamics described in the introductory example. I focus here on the statistical framework. In Appendix B, I outline one (by no means the only) utility model which would deliver these implications.

Consider the empirical problem of evaluating the effect of some health behavior Λ_j on outcome Y . There is a vector of other behaviors and characteristics (i.e. demographics), denoted $\mathring{\Lambda}$, indexed by $k = 1 \dots n$.

I assume that Y is fully determined by behavior Λ_j and $\mathring{\Lambda}$. If both were fully observed, I assume we could estimate the causal effect of Λ_j on Y through Equation (1).

$$Y_i = \eta + \beta \Lambda_{j,i} + \vartheta \mathring{\Lambda}_i + \epsilon_i \quad (1)$$

I assume a homogeneous treatment effect. Below, I discuss the possible role of treatment effect heterogeneity.

Researchers may not observe all of the elements of $\mathring{\Lambda}$. If the unobserved elements are correlated with Λ_j then the feasible estimates of β will be biased. Imagine researchers draw a sample of individuals and collect data on behavior Λ_j , outcome Y and a set of other variables Θ , which are a subset of $\mathring{\Lambda}$. The researchers estimate feasible equation (2) below.

$$Y_i = \alpha_t + \hat{\beta} \Lambda_{j,i} + \varsigma \Theta_i + \epsilon_i \quad (2)$$

It is well understood that if some elements of $\mathring{\Lambda}$ are omitted from Θ and correlated with Λ_j then $\beta \neq \hat{\beta}$.

The bias in the estimate of $\hat{\beta}$ will be controlled by the degree of correlation between Λ_j and the unobserved elements of $\mathring{\Lambda}$. In this paper, I consider a situation in which between periods t and $t + 1$, the perceived health benefits of behavior Λ_j increase. If there is selection in the new adoption of behavior Λ_j , this may increase the covariance between Λ_j and the unobserved set. In turn, this increase in covariance will increase the bias in the estimated impact of Λ_j on outcome Y . The key argument in the paper is that observing the change in $\hat{\beta}$ over time around changes in recommendations is a way to understand how large the selection problem is.

The empirical work below focuses on analyzing what happens when there is a change in recommendation about some behavior Λ_j . I look for several signatures in the data. First, a change in the

incidence of behavior Λ_j after the recommendation changes. Second, a change in the correlation between Λ_j and other health behavior or demographic correlates of health behaviors. Finally, a change in the estimated relationship between behavior Λ_j and outcome Y .

Instability of Null Effects Under this model of the research process a true treatment effect of zero will be fragile and difficult to sustain.

To see why, consider the case where $\beta = 0$, and imagine that initially selection is minimal so in the population the estimated treatment effect would also be zero. Due to sampling variability, with repeated sampling, 5% of the time the data will yield a significant positive or negative effect. When this happens, the endogenous behavioral reaction will reinforce that finding. In later periods the estimated treatment effect in the population will be biased by selection.

A true treatment effect that is positive (or, conversely, negative) will be self-reinforcing. We expect a move away from a positive effect (for example) *only if* the sampling variability in the data generates a significant negative effect despite the true significant positive effect. This will happen strictly less than 2.5% of the time. As a result, the null effects will be less stable than non-null.

Treatment Effect Heterogeneity The empirical framework focuses on the case with homogeneous treatment effects and in which selection drives changes in estimated coefficients over time. The empirical component of the paper will focus on estimating variation in selection and in outcome-treatment relationships over time, and interpreting them as connected. However, observing changes in outcome correlations over time could alternatively reflect treatment effect heterogeneity, if that heterogeneity is correlated with selection variables.

Imagine that the true treatment effect varies across groups and is larger for individuals who select into the behavior after the recommendation.¹ In this case, a larger estimated treatment effect in the “more recommended” period could reflect a larger causal effect for this group. Put differently, in this case we could observe movement in coefficients over time even if the regressions all yielded unbiased causal effects for the relevant treated population.

It is impossible to fully rule out this type of heterogeneity as an explanation for the patterns I observe. However, the data may be informative about the degree to which this is more or less plausible. More specifically, I can ask whether estimated heterogeneity on *observable* dimensions

¹In other selection-in-health behaviors settings, this may be fundamental. For example, Einav et al (2019) show mammography recommendations are disproportionately adopted by otherwise healthy individuals, and the resulting mammography results are more likely to be false positives or non-invasive tumors.

can explain some of the results. To do so, consider the underlying assumption that the treatment effects estimated in each period represent the causal effect for the treated population. Under this assumption, it is possible to estimate variation in treatment effects along *observable* dimensions in the baseline period. I can then re-weight along observable dimensions to match the distribution of observables in later periods. If treatment effect heterogeneity is important in explaining these movements over time, I would expect the re-weighting to explain some of the coefficient movements between periods. If this is not the case, it would be less consistent with this explanation for the results.

II Data

A NHANES

The National Health and Nutrition Examination Survey (NHANES) is a nationally representative survey which has been run, in some form, since the 1960s. In this project, I use data from the NHANES III (1988 through 1994) and from the continuous NHANES (beginning in 1999/2000 through 2014/2015). Summary statistics for all NHANES variables appear in Panel A of Appendix Table A2.

Target Behavior Data Information on vitamin supplementation is obtained from the vitamin supplement modules. I focus on individual vitamin supplements - that is, if someone is taking a single-ingredient Vitamin D or E supplement. Information on diet is generated from the daily dietary recalls in the study. I generate a Mediterranean diet score as described in Trichopoulou et al (2003). For sugar I use the share of carbohydrates that are sugar; for fat, the share of total fat that is saturated fat. I do this rather than total calorie shares since the recommendations for total fat and carbohydrates are also moving over time, and this approach better isolates “sugar” and “saturated fat” as the key target components.

Selection Variables: Other Health Behaviors and Demographics The NHANES measures exercise and smoking behavior. In the case of exercise, I focus on measures of vigorous exercise which is reported in episodes per month or week. Due to some variations in question structure, I standardize relative to the mean for each year. Smoking is a measure of current smoking behavior. I create a simple measure of diet quality: specifically, whether the individual consumes above or

below the median number of vegetable calories.

Finally, I extract data on education (in five categories), income (in eight bins), age and gender from the demographic survey portion of the NHANES.

Health Outcomes I create an index of heart health based on the first principal component of blood pressure, total cholesterol and good cholesterol. Body mass index is defined (as is usual) as weight in kilograms divided by height in meters-squared. All variables are measured by the surveyor, not self-reported.

B Nurses' Health Study

The Nurses' Health Study (NHS) is a panel dataset of female nurses. The NHS recruited a cohort of approximately 120,000 female nurses in 1976 and conducted mail surveys of the cohort every two years. The study is ongoing; as of 2012 the average per-wave response rate was 86.2% (Bao et al, 2016). The cohort is described in more detail in Colditz et al (1997). I use data from 1984 through 2010. Summary statistics for the variables used in from the NHS appear in Panel B of Appendix Table A2.

Target Behavior Data The NHS asks participants about vitamin E supplementation.

Selection Variables: Other Health Behaviors The NHS asks nurses about their smoking behavior.

Health Outcome The health outcome is mortality, measured as the chance of death before the next survey (in two years).

C HomeScan

The Nielsen HomeScan panel tracks consumer purchases using at-home scanner technology. Households that are part of the panel are asked to scan purchases, and the Nielsen data records the UPC of items purchased. I use Nielsen data for 2004 through 2016, available through the Kilts Center at the University of Chicago Booth School of Business. There is no information on health outcomes in these data. Summary statistics for HomeScan appear in Panel C of Appendix Table A2.

Target Behavior Data The target behavior is household purchases of Vitamin D supplements by year. I use product descriptions to identify the set of products which are Vitamin D supplements.

Selection Variables: Other Health Behaviors and Demographics Household smoking is identified based on household purchases of cigarettes. I construct a diet quality score based on Hut and Oster (2019); this approach uses information from a survey of doctors to code food quality and aggregate to a score.

Household head education and income (both in bins) are drawn from yearly panelist surveys.

III Results: Evidence from Vitamin E

Recommendations about vitamin E consumption have changed over time. In the early 1990s, a number of studies pointed to significant health benefits to vitamin E. Most notable was a pair of studies in the New England Journal of Medicine in 1993 which cited large benefits of vitamin E in preventing heart disease in men and women (Rimm et al, 1993; Stampfer et al, 1993). There was significant media attention (i.e. Brody, 1993; Miller, 1993).

In 2004, however, new evidence suggested that excess vitamin E supplementation could actually *increase* mortality (Miller et al, 2005).² This evidence consisted of a meta-analysis of small randomized controlled trials, and was similarly widely covered by media organizations (Kolata, 2004).

These changes are reflected in trends in vitamin E consumption over time, which are illustrated in Figure 1. There is clear evidence that purchase and consumption of these products vary around the events identified above: vitamin E consumption increases after 1993, and then decreases, with a significant drop after 2004.

A Selection in Vitamin E Consumption

The first suggestion in the statistical model above is that the group who consumes vitamins *after* they are more strongly recommended will be differently selected than those who consume before. To explore this, I define three time periods around the changes in recommendations: before 1994, between 1994 and 2004 and after 2004.³ For each selection variable X_i I estimate Equation (3) in

²This study was released in 2004 but the publication was in print in January, 2005.

³Due to the timing of the NHANES-III, which covers 1988 through 1994, the first time period in the NHANES analysis will include some observations from 1994. It is not possible to pull out the 1994 observations.

each time period:

$$VitE_i = \alpha + \beta^T X_i + \Pi \mathbf{A}_i + \varepsilon_i \quad (3)$$

The dependent variable, $VitE_i$, is an indicator for whether the respondent reports taking a vitamin E supplement. Each time period produces a coefficient β^T ; these are the results of interest. \mathbf{A}_i is a vector including only age, age squared and gender (in the NHANES; the NHS has only women). I estimate this using a standard linear probability model, with homoskedastic standard errors.

The results are shown in Figure 2. Panel A uses the NHANES data, where I observe both socioeconomic status measures and other health behaviors. Panel B uses the NHS data to look at smoking (yearly estimates appear in the Appendix). For the NHS, the coefficients are reported relative to the average level of smoking in these data so they are interpreted as percent changes.

There is clear positive selection in vitamin E as consumption increases in the middle period relative to the other two. These differences are statistically significant and the coefficients are large. The relationship between education and vitamin E consumption, for example, is twice as large after the positive recommendation as before. In the case of the NHS, the data is a panel, so I can directly interpret this to say (for example) that the new adopters of vitamin E in the period after 1993 are less likely to be smokers than those who do not adopt.

The patterns in this table are consistent with the selection posited in the statistical model above. In the periods in which vitamin E is more recommended the consumers are less likely to smoke, more likely to exercise, eat a better quality diet and are richer and better educated.

B Vitamin E and Health Outcomes

I now look at the relationship between Vitamin E consumption and health outcomes. I estimate Equation (4) for each time period:

$$Y_i = \gamma + \Psi^T (VitE_i) + \Pi \mathbf{A}_i + \varepsilon_i \quad (4)$$

As above, the coefficients of interest are the Ψ^T —the by period correlations. I estimate this regression first including only age, age-squared and gender (where appropriate) in the \mathbf{A}_i vector. I then estimate the regression including some additional demographic controls - the selection variables used above, and some additional health variables for the Nurse Health Study. Full lists of controls are included in figure notes. The outcome Y_i is the index of heart health in the NHANES and two-year mortality in the NHS. In the latter case, the coefficients are scaled by the mortality rate

so are interpretable as percentage changes.

Panel A of Figure 3 shows the NHANES results. Qualitatively, both with and without controls, the patterns echo the patterns in selection, with vitamin E supplementation being more strongly associated with better heart health in the middle period (when it is more recommended) than in the surrounding periods. The differences are statistically noisy, although in the more precise controlled regression we can reject equality between the latter two bars at the 10% level.

Despite the lack of statistical precision, it is worth noting that the size of the differences are large. For example, between the middle and the latter period in the controlled regression, the estimated coefficient falls by 0.080. Because this is an index, this has no direct interpretation but I can compare to other variables. This decline is equivalent to moving a full education category in the data and just a bit less than the difference between white and black participants.

Panel B of Figure 3 shows the NHS results.⁴ The evidence shows the size of the correlation between supplementation and mortality moving significantly with the recommendations. In the early period, taking vitamin E is associated with approximately a 10 percent reduction in death risk. In the period from 1993 through 2003 this jumps to a 25 percent reduction in mortality risk (the mean is approximately 2 percentage points over this period). In the time period after the 2004 release of evidence undermining the value of vitamin E, the relationship is again smaller at around 10 percent. The data reject equality of coefficients across periods with a high level of significance. The dynamics are similar with and without the controls included.

The variation across time periods is large. The estimated coefficient on vitamin E in the period from 1993 through 2004 is about a third of the size of the impact of smoking on mortality. Given the fairly simple nature of the health intervention “take vitamin E supplements”, if the mortality effect were actually a third of the size of quitting smoking, this would be quite notable. Put simply, depending on which time period you draw observational data from, the conclusions about the impacts of vitamin E on health could be very different.

The variation in coefficients over time seems consistent with variation in selection, but as I note in Section 2 these change could also be driven by treatment effect heterogeneity. As I suggest there, it may be possible to get a sense of the scope of this possibility by using the baseline period to estimate treatment effect heterogeneity in the observed data and then calculate the implied treatment effects in later periods by re-weighting to match the observables. I do that in these examples; in general, the reweighting does not suggest a treatment effect heterogeneity explanation.

⁴Results by year are shown in the Appendix.

In the case of the NHANES, if we take the cross sectional results as causal, the baseline effect of Vitamin E on heart health is 0.060. Using the estimated heterogeneity in this effect in the baseline period, applied to the observable distribution in the intermediate period, I would predict a second-period coefficient of 0.064. In fact, the coefficient is 0.094. A similar result obtains in the NHS. The predicted intermediate period effect would actually be smaller in magnitude than the baseline effect in this analysis, whereas the actual coefficient is much larger in magnitude. While this does not rule out treatment effect heterogeneity driving the effects, it does not support that theory either.

IV Auxiliary Evidence from Vitamin D, Diet

Vitamin E is an effective case study for several reasons, including the availability of rich data and the fact that it becomes both more and less recommended over the period of the data. However, it is only a single example. I therefore present suggestive, corroborating evidence in another vitamin (Vitamin D) and in three diet measures (sugar, fat and the Mediterranean diet).

A Vitamin D

Background and Setup In 2007 there were a number of pieces of positive news about vitamin D supplementation. These included a widely cited *New England Journal of Medicine* article on the benefits of vitamin D (Holick, 2007), coverage in the *New York Times* and other outlets (e.g. Nagourney, 2007) and a corresponding spike in Google searches. On the other side, around 2012 there was a push back. An Institute of Medicine report (Rosen et al, 2012) suggested the purported benefits of vitamin D were overblown, and media coverage in the same period reinforced this (e.g. Bakalar, 2012a; Kolata, 2012; Bakalar, 2012b).

Using the NHANES and HomeScan data I will be able to explore trends in purchases over time and changes in selection of these behaviors. There is no linked outcome data to use here so I will focus only on the changes in selection. I will estimate equations of the form in Equation (3) but with Vitamin D rather than Vitamin E as the outcome.

Results

Panel A of Figure 4 shows the time trends in consumption (NHANES) or purchases (HomeScan) of Vitamin D around these events. There is evidence of increasing purchases of Vitamin D in the

period where the news is more positive. The HomeScan evidence clearly points to a decrease in purchases after 2012.

Panels B and C of Figure 4 show changes in selection in the NHANES (Panel B) and the HomeScan (Panel C). In both datasets, across all the metrics, the evidence points to changes in selection consistent with the theory and the patterns in vitamin E. When Vitamin D is more recommended it is more strongly associated with high education and income, a good diet, less smoking and more exercise. These associations are significantly different over time.

The difference across education groups in Vitamin D consumption doubles from the late 1990s to the late 2000s; by the late 2000s, increasing one education category increases the chance of consuming vitamin D supplements by about 25%.

B Dietary Patterns

Background and Setup I analyze three dietary patterns: sugar intake, saturated fat intake and adoption of a Mediterranean diet. I focus in presenting the results on the case of sugar, for illustrative purposes, but include all three in the Appendix.

Recommended levels of sugar and fat intake have fallen over time. In the case of sugar, the revision of the US Dietary guidelines in 2000 marks the first mention of avoiding sugar for health reasons (Krauss et al, 2000). Following this, in 2011/2012 there was a surge of popular media interest in the dangers of sugar (e.g. Taubes, 2011; Chang, 2012; 60 Minutes “Sugar”).

Saturated fat was first restricted in the US dietary guidelines in 1990, with a suggested limit of 10% of calories (Peterkin, 1990). In 2005 this limit was lowered to 7% (Thompson and Veneman, 2005).

The Mediterranean diet has been, conversely, increasingly recommended over time, largely due to positive findings from major research studies. The first of these was in 2004, when two articles in the Journal of the American Medical Association (JAMA) showed positive health benefits from the diet (Knoops et al, 2004; Esposito et al, 2004). In the 2009 period additional studies argued for cognitive benefits (Feart et al, 2009). A large randomized trial released in 2013 showed significant cardiovascular benefits (Estruch et al, 2013), although this largely post-dates my data.

In analyzing diet I focus on the NHANES data. I am able to use these data to look at trends over time, changes in selection and changes in links with health outcomes, specifically BMI and heart health. A downside of the diet analysis relative to vitamins is that in all three cases the trends are secular. As a result, it is not possible to look for the reversals of selection that are a

feature of the vitamin analysis.

For each dietary measure, I will estimate Equation (3) and Equation (4) replacing the Vitamin E measures in each case with diet measures.

Results I present the results for sugar in the main text, focusing on the BMI outcome. The full set of diet results, including both health outcomes and all three dietary measures, are shown in Appendix Table A3. The overall patterns are similar across dietary choices and outcomes.

The sugar results are shown in Figure 5. Panel A shows changes in sugar intake over time; it declines with the changes in recommendations.

Panel B documents corresponding changes in selection. Consuming more sugar (measured as a share of carbohydrates) becomes, over time, more positively associated with smoking and more negatively associated with exercise, education and income. It is notable that in the earliest periods of the data those with higher income and education actually consume on average *more* sugar. In the latest periods of the data, they consume much less.

Panel C of Table Figure 5 shows links with BMI. These patterns echo the selection results. Over time sugar becomes *much* more positively associated with having a high BMI. Indeed, this link is roughly zero in the earliest periods of the data, and only becomes large and positive over time. The second set of columns in Panel C shows the regressions including controls, and demonstrates that the patterns persist.

V Discussion and Conclusion

In this paper I analyze the role of behavior change in driving biases in estimates of the impact of health behaviors on outcomes. I describe a simple data generating process in which new health recommendations are more likely to be adopted by individuals who invest more in their health in other ways. Under this process, the bias in observational data-based estimates of the relationship between health behaviors and health outcomes will be endogenous to these recommendations.

That this is a possibility follows directly from the theory; using data on vitamin supplements and diet I demonstrate that this is not just a possibility but is reflected by dynamics in the data. The degree of selection in behaviors varies over time, and the relationship between behavior and health also varies with these changes in selection. The changes over time – both in the patterns of selection and the behavior-outcome relationships - are large.

These results have several implications that may be worth highlighting.

First, I will reiterate the possible implications for null effects. The nature of statistical precision is that – with a 5% significance cutoff – 5% of the time treatment effect analysis will yield a significant result even if the true effect is zero. This is true even when the analysis is done through a randomized controlled trial. The dynamics here can reinforce such false positive results. This issue is exacerbated by publication bias (i.e. Ioannidis, 2005). In the presence of a publication process which favors significant outcomes, we may be even more likely to observe false positive results published and, subsequently, reinforced by these patterns.

Second, I note that the results on selection in uptake of recommended behaviors may be independently interesting. In particular, to the extent that the recommended behaviors *are* beneficial, this provides insight into how improvements in knowledge about health behaviors may exacerbate health inequality.

Finally, although this paper focuses on diet and vitamin supplementation, it seems possible that similar dynamics could show up in other behaviors where health recommendations vary over time. Linking back to the first point above, this seems most likely in cases where the true treatment effects are likely to be (at best) small.

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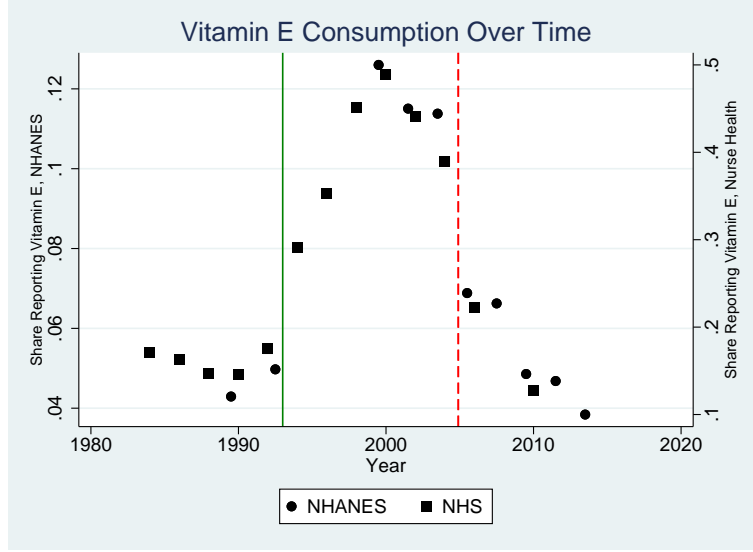
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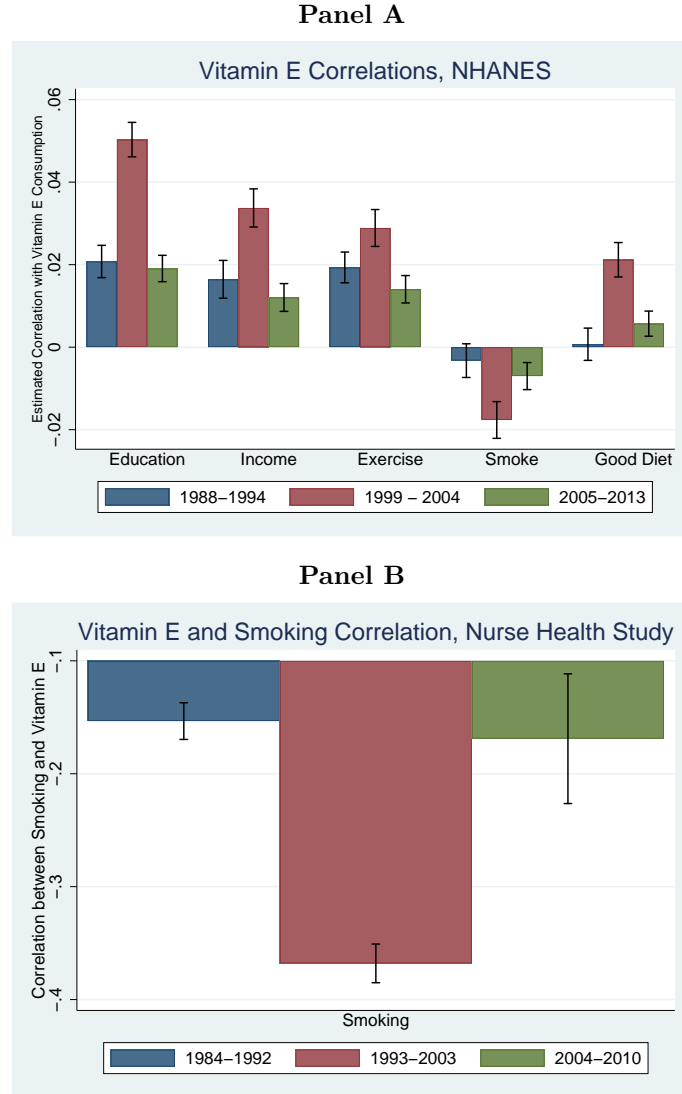
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Figure 1: Vitamin E Consumption Levels over Time



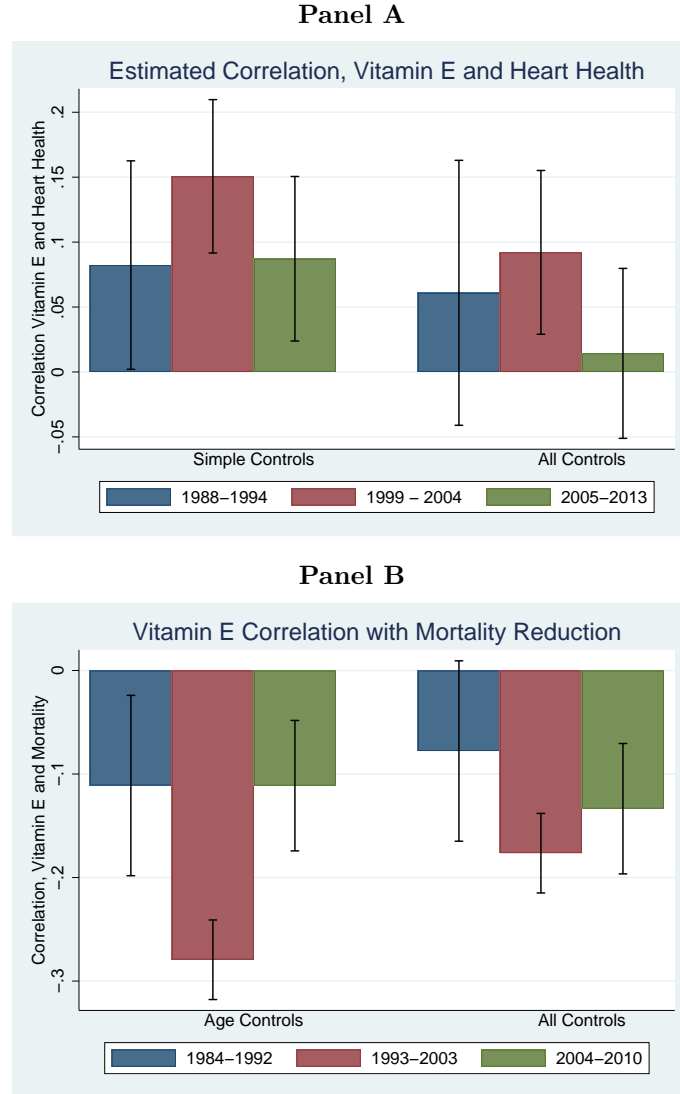
Notes: These graphs show evolution of reported vitamin E consumption in the NHANES and Nurses' Health Study. In both cases, the variable measures the share of survey respondents who report consuming any vitamin E supplements. In the Nurse Health Study respondents are asked about Vitamin E directly. For the NHANES I code this based on the reported list of vitamin supplements, including only individual Vitamin E supplements, not multivitamins. Events are marked with vertical lines; details of the events appear in Appendix Table A1. The solid green line indicates the release of positive news about vitamin E; dotted red lines indicate the release of negative news. Number of observations per year: NHANES (4,252 to 7,921); Nurse Health Study (76,343 to 99,382).

Figure 2: **Vitamin Consumption, Socioeconomic Status and Health Behaviors**



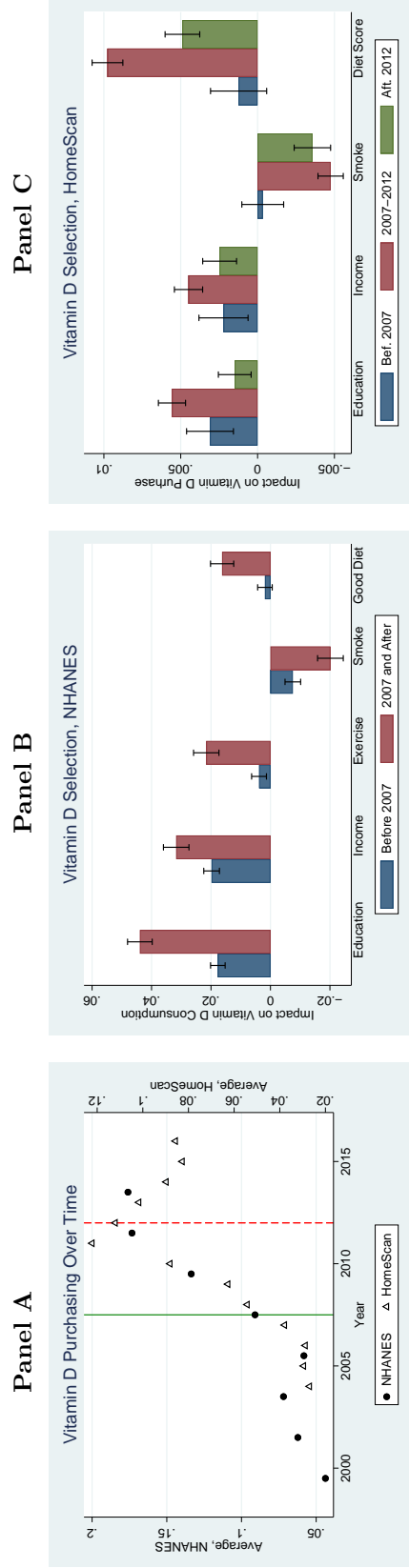
Notes: These figures show changes in the relationship between Vitamin E consumption and health behavior or socioeconomic status over time; they are the results from estimating Equation (3) in the text. The regression outcome is an indicator for Vitamin E consumption and I create these figures by extracting coefficients on each selection variable in each time period. Panel A uses data from the NHANES. In Panel A all effects can be interpreted as the estimated relationship between a one standard deviation change in the selection variable on the probability of reporting Vitamin E supplement consumption. A good diet is defined as having above median calories from vegetables. Panel B uses data from the Nurse Health Study. In this case I divide the coefficients from the regression by the mean level of smoking, which addresses the overall reduction in smoking rates over time. The bars can be interpreted as the percent decrease in smoking among those who take Vitamin E supplements. Error bars show 95% confidence intervals. Number of observations by time period: NHANES ($N_1=15,748$; $N_2=13,421$; $N_3=24,524$); Nurse Health Study, in person-years ($N_1 = 446,700$; $N_2 = 513,518$; $N_3 = 140,614$).

Figure 3: Vitamin E and Health Outcomes



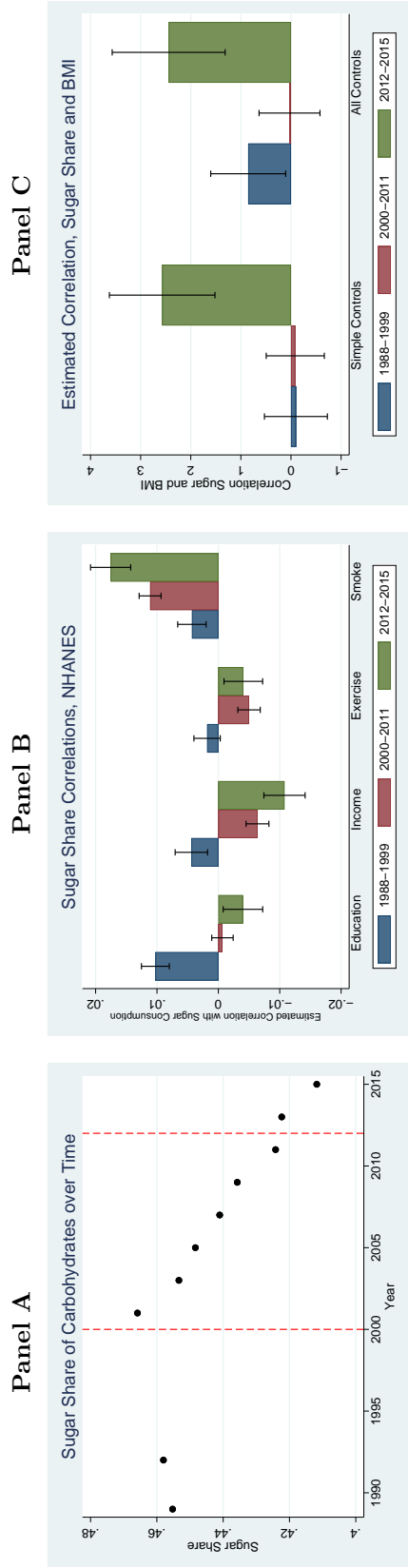
Notes: This figures shows changes in the Vitamin E-health outcome relationship over time; these result from estimation of Equation (4) in the text. In Panel A (NHANES) the outcome is an index of heart health and key independent variable is reporting consumption of a vitamin E supplement. The first set of bars control for only age, age squared and gender; the second set include controls for education, income, marital status, race, smoking behavior, exercise and vegetable consumption relative to the mean. In Panel B (Nurse Health Study) the outcome is two-year mortality and the key independent variable is reported consumption of Vitamin E. The coefficients are scaled by the death rate so can be interpreted as a percent change. The first set of bars control only for age dummies. The second set include controls for smoking, BMI, and indicators for hypertension, high cholesterol, diabetes. cancer diagnosis post-menopausal hormone use, angina, myocardial infarction, and menopausal status (this is a set similar to what is used in Stampfer et al, 1993). Error bars show 95% confidence intervals. Observations: NHANES, simple controls ($N_1 = 14,770$; $N_2 = 11,986$; $N_3 = 20,152$); NHANES, all controls ($N_1 = 10,199$; $N_2 = 10,666$; $N_3 = 18,554$); Nurse Health Study, person-years, age controls ($N_1 = 470,264$; $N_2 = 521,313$; $N_3 = 144,715$); Nurse Health Study, person-years, all controls: ($N_1 = 390,355$; $N_2 = 470,587$; $N_3 = 123,802$)).

Figure 4: Evidence on Vitamin D



Notes: This figure shows evidence on Vitamin D. Panel A shows data on an indicator for vitamin D consumption in NHANES or purchasing in HomeScan. Events are marked with vertical lines; details of the events appear in Appendix Table A1. The NHANES data ranges from 1998 through 2013; the 2014-2015 NHANES does not have dietary supplement data available. HomeScan data ranges from 2004 through 2016. Vitamin D consumption in the NHANES is based on reported supplements taken; in the HomeScan this is a measure of whether the household records a purchase of any single Vitamin D supplement during the calendar year. Panels B and C show the relationship between Vitamin D consumption and selection variables in both the NHANES and HomeScan data. These coefficients come from regressions of the form of Equation (3), with Vitamin D rather than Vitamin E as the outcome. The NHANES regression control for age, age squared and gender. HomeScan regressions include controls for household composition and household size. In both cases the coefficients can be interpreted as the effect of a one standard deviation change in the selection variable on vitamin D consumption or purchase. In the NHANES a good diet is defined as having above median calories from vegetables; in the HomeScan diet score is constructed based on Hut and Oster (2019). Smoking in the HomeScan data is defined based on whether the household purchases cigarettes. Error bars show 95% confidence intervals. Number of observations in Panel A: NHANES (4252 to 7827), HomeScan (38,863 to 69,247). Panel B: $N_1 = 38,706$, $N_2 = 131,536$, $N_3 = 399,313$. Panel C: $N_1 = 14,841$, $N_2 = 14,841$, $N_3 = 264,228$.

Figure 5: Evidence on Sugar



Notes: This figure shows evidence on sugar. Panel A shows data on the sugar share of carbohydrates over time in the NHANES. Events are marked with vertical lines; details of the events appear in Appendix Table A1. The NHANES data ranges from 1998 through 2015. Sugar share is measured based on dietary recall data. Panel B shows the relationship between the sugar share variable and selection variables in the NHANES. These coefficients come from regressions of the form of Equation (3), with sugar share rather than Vitamin E as the outcome. The NHANES regression control for age, age squared and gender. The coefficients can be interpreted as the effect of a one standard deviation change in the selection variable on sugar share. Panel C shows changes in the sugar-BMI relationship over time; these result from estimation of Equation (4) in the text. The outcome is BMI (weight in kg divided by height in meters-squared) and the key independent variable is sugar share of carbohydrates. The first set of bars control for only age, age squared and gender; the second set includes controls for education, income, marital status, race, smoking behavior and exercise. Error bars show 95% confidence intervals. Number of observations in Panel A: (4064 to 7777). Panel B: $N_1 = 12,004 - 15,682$, $N_2 = 24,807 - 26,954$, $N_3 = 7,872 - 8,683$. Panel C: Simple controls ($N_1 = 15,651$; $N_2 = 26,423$; $N_3 = 8,606$); All controls ($N_1 = 10,800$; $N_2 = 24,308$; $N_3 = 7,800$).