The spillover effects of diabetes diagnosis on own and partner's behaviour: Using HbA1c and fuzzy regression kink design

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Abstract

Diabetes is a unique condition, in that the first line of treatment of the condition is the same as the suggested prevention of the disease, both being positive lifestyle behaviours. It is theoretically possible that by jointly partaking in diabetes treatment, partners of people with diabetes would substantially benefit from their partners' diabetes diagnosis. Using blood data from the Health Survey for England, and a fuzzy regression kink design, we causally estimate the effect of a diabetes diagnosis on health-related behaviours of the individual with diabetes, as well as their partners. We find that a diagnosis of diabetes results in a significant increase in the probability of exercising, as well as a decrease in the probability of currently being a smoker, but find no effect on diet and alcohol consumption. Partners' also tend to have an increased probability of exercising although there is limited evidence of any other behavioural changes. From a public health perspective, our results are especially important for the evaluation of diabetes related policies, while positive spillovers, particularly within households, should also be taken into account in the evaluation process.

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1 Introduction

There is substantial literature documenting and analysing the positive correlation between spouses' behaviours. Much of the work thus far has focused on the correlation between spouses in terms of smoking behaviour (Barrett-Connor et al.; 1982; Venters et al.; 1984; Graham and Braun; 1999; Franks et al.; 2002; Bloch et al.; 2003; Clark and Etil; 2006; Stimpson et al.; 2006; Christakis and Fowler; 2008; Falba and Sindelar; 2008; Cobb et al.; 2014) and alcohol consumption (Kolonel and Lee; 1981; Graham and Braun; 1999; Leadley et al.; 2000; Leonard and Mudar; 2003; Stimpson et al.; 2006; Falba and Sindelar; 2008). However Farrell and Shields (2002) and Falba and Sindelar (2008) also analyses physical activity, and find that there is a strong positive correlation of physical activity between household constituents. Kolonel and Lee (1981), Barrett-Connor et al. (1982), Macario and Sorensen (1998), Bove et al. (2003) and Lyu et al. (2004) all estimate the correlation between spouses' diets, and consistent with the other studies on concordance of behaviour, find that spouses' diets show a strong correlation. However, these correlations extend beyond behaviours alone. There has been work documenting spousal correlation in mental health and physical health[1]. There has been some work attempting to understand the causes of these strong correlations, and a number of theories have been presented that attempt to explain this finding. Broadly, the empirical correlations have been attributed to three root causes: assortative matching, shared environment, and joint household decision making (Wilson; 2002; Clark and Etil; 2006; Meyler et al.; 2007).

The basic idea behind assortative matching, which has also referred to as the marriage market hypothesis, is that individuals choose to marry people whom have similar preferences and characteristics. This theory was first introduced by Becker (1973), and claims that positive assortative matching, that is, partners match based on similar characteristics, occurs if characteristics and preferences of each partner are compliments. In other words, if partners enjoy partaking in activities together, then they choose to match with a partner who has shared their characteristics and preferences.

Shared environment is the second explanation of these correlations. Partners individually make decisions based on their preferences, but are constrained by shared resources and are exposed to common shocks. This implies that partners' behaviours will be similar precisely because they share these resources and are exposed to similar shocks. An alternative ex-

[1]See systemmatic review by Meyler et al. (2007) and references therin for full discussion

planation of this phenomena, relying on more epidemiological terminology, is that partners whom share a common environment, are exposed to the same health risks as a result. Another component of this, which is important in our setting, is shared information. Partners not only share resources, but also share information sets, by transferring information between each other, Clark and Etil (2006) call this social learning. Common information sets mean that they also have similar expectations of future uncertainty and risk, and as a result make similar behavioural choices.

Finally, joint household production is also proposed as a source of correlation between partners. This source of correlation leans on the theory of New Home Economics, originally established by Lancaster (1966) and Becker (1981). The basic idea behind this theory is that households jointly produce goods which enter individuals' utility functions. Individuals within the household bargain and as a result produce and consume some shared output. This implies a correlation both in behaviour and health, because the output is shared between partners. Payoffs from producing and subsequently consuming a particular good is a function of own private payoffs, and an externality from their partner consuming the same good. As with assortative matching, if behaviours or specific consumption goods are complements, then partners may choose to jointly produce and consume them, which would explain the correlations in consumption and behaviour.

The latter two of these theories suggest that if one partner was received an update in their health knowledge, then this would lead to a change in behaviour of the other partner, and potentialy impose a beneficial spillover onto them. Although theoretically justified, Clark and Etil (2006) found that social learning and household decision making do not play an important role in smoking behaviour. They claim that it is matching in the marriage market which explains the majority of the raw correlations found between partners. In this paper we investigate whether spillover effects on partners do indeed exist after a diabetes diagnosis. By exploiting blood data from the Health Survey for England (HSE), as well as a seemingly arbitrary cut-off of diabetes risk, we are able to causally estimate the impact of own diabetes diagnosis on own behaviour, as well as the effects of own diagnosis on partners' behaviour. We find that there is an effect of diabetes diagnosis on own physical activity. We also find that partners' of people with diabetes also change their behaviour, by increasing their physical activity. Our identification strategy allows us to reasonably exclude the possibility that assortative matching is the cause of this result and in doing so, we are able to claim that spillovers do indeed exist for physical activity and that assortative

matching does not explain the entire raw correlation between spouses, at least in terms of physical activity.

We contribute to the literature in a number of ways. Firstly, we contribute to the field of household economics within the context of health by providing evidence that the observed correlation in partners' behaviours is not solely limited to assortative matching, but that social learning and joint household decision making are important components of the observed correlation. We also contribute to the literature on diabetes, and sit besides work done by Hut and Oster (2018), Oster (2018) and Kim et al. (2019) in estimating the behavioural responses of a diabetes diagnosis. Our results suggest that individuals with diabetes are compliant to some treatment, we also estimate that this behavioural change is persistent over time. Our results are also particularly interesting for policy makers in health, as our findings suggest that there are substantial spillover effects from a diabetes diagnosis, which may otherwise be ignored in the evaluation of policies in this area.

This paper is organised as follows: firstly we will discuss the background of this paper, specifically, we will discuss diabetes in detail, and noting the institutional setting as well as previous literature in this area, then we will discuss the theory and literature on spousal correlation and how the theories attempting to understand this correlation corresponds to our setting. Then we will outline the data we use before moving onto our identification strategy. Following this, we discuss our results and validate the identifying assumptions. Finally we end with a conclusion of our results, where we attempt to put them into wider context.

2 Background

We begin this section by providing background on diabetes, and the setting which allows us to exploit the RKD and provide us with a causal interpretation. Then, we discuss our theoretical justification of a spillover effect onto partners of those with diabetes.

2.1 Diabetes

The World Health Organization (WHO) state that diabetes "is a chronic, metabolic disease characterized by elevated levels of blood glucose (or blood sugar), which leads over time

to serious damage to the heart, blood vessels, eyes, kidneys, and nerves" (WHO; n.d.). Diabetes is categorised into two types, the most prevalent type being type 2 diabetes. Of the 4.7 million people in the UK that have diabetes, approximately 90% of them have type 2 diabetes (Diabetes UK; 2019). Type 2 diabetes occurs when the body becomes resistant to insulin. Approximately 8% of people with diabetes in the UK have Type 1 diabetes, which occurs when insulin production in the body is limited. Although there is limited understanding as to what causes type 1 diabetes, diet or lifestyle are known not to have any impact on probability of being having type 1 diabetes. Type 2 diabetes on the other hand is usually a result of poor diet and lifestyle (Helmrich et al.; 2010; Pan et al.; 1997; Hu et al.; 2001; Liu et al.; 2000).

Glycated haemoglobin refers to the amount of haemoglobin (the protein within red blood cells) which have been 'glycated'. This occurs when the body processes sugar, and glucose in the blood then attaches to haemoglobin proteins. The red blood cells which contain the haemoglobin proteins usually survive for between 8 and 12 weeks, and therefore HbA1c is considered to be an average blood sugar level over the previous three months. HbA1c is considered a useful measure in the diagnosis of diabetes, in that it provides an indication of blood sugar level over a longer duration. The alternative measure, blood glucose level, is the concentration of sugar in the blood at a single point in time and is highly variable over time, and more dependent on very recent consumption and behaviours than persistent behaviours.

The World Health Organisation (WHO; 2011) state that "An HbA1c of 6.5% is recommended as the cut point for diagnosing diabetes". They also state, however that a value below 6.5% does not exclude a diabetes diagnosis. A level below 6% is considered to be normal blood sugar level and therefore low-risk, while levels between 6% and 6.5% are considered to be at high risk, also called pre-diabetes. Although pre-diabetes is not a clinically term, and usually does not involve any symptoms, NICE recommendations state that "For people at high risk (a high risk score and fasting plasma glucose of 5.5 - 6.9 mmol/l, or HbA1c of 42 - 47 mmol/mol [6.0 - 6.4%]), offer a blood test at least once a year (preferably using the same type of test). Also offer to assess their weight or BMI." NICE (2012).

On the topic of prediabetes Yudkin and Montori state that "glycaemia are continuous, with no inflections to provide obvious cut-off points. Cut-offs for the diagnosis of diabetes are based on thresholds for risk of retinopathy. Lesser degrees of hyperglycaemia increase

the risk of developing diabetes and maybe arterial disease. But in both cases the risk is graded, making any choice of cut-off point purely arbitrary."

Individuals who have been tested to be at high risk of type 2 diabetes therefore have a high probability of being diagnosed with diabetes simply as a result of being subject to annual assessment of their HbA1c level. Individuals just below the threshold of 6.0% have a similar probability of actually having diabetes as those juts above the threshold but have a lower probability of being diagnosed as a result of them not being annually tested.

2.2 Spousal Correlation

As mentioned above, we believe that there is theoretical justification that a spillover effect may be present from one of the partners being diagnosed as having diabetes. Firstly, a diabetes diagnosis transfers health information to the patient either in an updated knowledge of own health state (i.e. diagnostic), or knowledge of the disease itself (i.e. information regarding the causes and consequences of diabetes). Updated knowledge of own health state can be thought of as the direct impact of a diagnosis. Prior to the diagnosis people with diabetes would not be aware that they have the disease, and therefore a diagnosis updates this information, making them aware that they have the disease. Whereas knowledge of the disease is the information a physician provides alongside the diagnosis regarding the treatment and causes of the disease. Social learning implies that individuals with diabetes will transfer the newly received information to their partners. By transferring this information to their partners they are both in possession of the same information set with which they can make new expectations of future uncertainties and risks. It may or may not be the case that individuals do indeed change their behaviour as a result of the new information, this is dependent on their idiosyncratic preferences, structural determinants of health and their information set pre-diagnosis. However, if an individual has a preference for health, and they are not fully informed of the risks of diabetes, then we expect that the new information would result in them partaking in healthy behaviours as a result of the newly acquired information. If this is the case, then we expect that through a diagnosis, partners' benefit from receiving new health information.

For the health information causal channel, the effect on partners' is independent of the observed behaviours of the diagnosed individual themselves post-diagnosis. The partner privately re-evaluates and makes new utility maximising decisions based on their new in-

formation set which was transferred to them by their partners, but based on their own idiosyncratic preferences. Although the information set would be shared between partners, their preferences are not identical, and therefore realised behaviours are not perfectly correlated. The magnitude of this effect is also dependent on the information set pre-diagnosis. The expectation is that partners' in possession of realistic expectations of the risks of diabetes would make less adjustments to their behaviour, than partners' in possession of a information set which leads them to make unrealistic expectations of risks and consequences of the disease. We would expect that for partners' in possession of realistic expectations pre-diagnosis, the new information they receive does not substantially change their expectations, whereas unrealistic expectations would be updated to more realistic ones because of the receipt of new information. The claim here is that individuals preferences remain stable, but the expectation of uncertain events is updated.

Secondly, we expect that a diabetes diagnosis changes their partner's behaviour through joint household decision making. Using the New Household Economics theory (Becker; 1973, 1981), if a diabetes diagnosis changes the optimal consumption of health-related activities of the individual, through the updated information discussed above, then we can expect that this would also impact the production and consumption decisions of the other productive household members. A simplified example that illustrates this point is the following: post-diagnosis it may be the case that, physical activity has a higher expected payoff for the partner with diabetes but physical activity requires an allocation of time which could otherwise be spent doing other martial production. If it were the case that the partner without diabetes had a strong preference for spending joint time with their partner, they may choose to participate in physical activity because they gain utility from consuming joint time, but would not gain utility from engaging in physical activity *per se*. This would clearly result in an increase in physical activity of the partner without diabetes. Although, it is not necessarily the case that there is a spillover. A diagnosed individual could be non-compliant to treatment, and therefore we wouldn't expect a change in their partners behaviour through joint household decision making. Alteratively, the individual with diabetes could be compliant with treatment but if the partner without diabetes has a preference for joint time, but a strong preference against physical activity, then they may choose to forgo joint time in an attempt to avoid the negative payoff of physical activity. Also, if the partner had a strong preference for a different good which required joint allocation of time, the individual with diabetes may participate in less physical activity

than their private optimal, so that they are avoid a negative payoff from a "conflict" with their partner. The effect of a diabetes diagnosis through this causal channel is somewhat more ambiguous, however we do beleive there is theoretical justification of a spillover effect through this channel.

We note that, although it is possible that individuals may assortatively match based on diabetes diagnosis, this would require diagnosis to happen pre-match. We believe that our analysis excludes the possibility of assortative matching explaining the phenomena we observe. What is more reasonable however, is that individuals match based on behaviours which may impact the cause of diabetes. What we mean by this is that two individuals who share a preference for not partaking in physical activity match in the marriage market, these individuals are more likely to be diagnosed as having diabetes precisely because they do not regularly partake in exercise. This is not the effect we seek to estimate, and therefore partners' diabetes status would be endogenous in this case. We believe that our identification strategy excludes the possibility that our estimates are the result of assortative matching and we discuss it in detail below.

The reminder of this paper seeks to estimate whether the theory proposed here does materialise in the data, and whether a spillover from a diabetes diagnosis does exist.

3 Data

This paper uses the Health Survey for England (HSE), years 2003 to 2017, which is an annual survey aiming to monitor trends in national health. Over 9,000 addresses are sampled over the course of the calendar year. Within each household, all individuals are eligible for survey inclusion, however children under 15 years old are asked to complete a different survey. Each year over 9,000 individuals respond to the survey, which includes both adults and children. In addition to the individual questionnaire, all respondents are eligible for a nurse visit. The nurse visit involves both physical measurements of individuals and a blood sample being taken for further analysis. The physical measurements include height, weight, waist and hip circumference, and blood pressure. The blood sample once taken, is sent to a specialist laboratory to measure glycated haemoglobin (HbA1c) and cholesterol. Cholesterol measurements included total cholesterol and HDL cholesterol. Although 82% of individuals agreed to be contacted by the nurse regarding a visit across

all years, only 44% of the final sample had blood taken by the nurse for analysis. Of the 61,540 individuals who had blood taken in the survey, 58,694 individuals had valid HbA1c measurments.

Table 1 provides descriptive statistics of the data that we use in our analysis. The first column provides the means and standard deviations of a number of observable characteristics and stated health-related behaviours for the entire HSE sample, which includes those that did not have blood measurements taken. In the following columns we present the descriptive statistics of the sub-sample of individuals who did have blood taken for analysis, and this is the sample we use to estimate the effects of individuals. We break these descriptive statistics into those with measured HbA1c levels above 6.0% and those with levels below 6.0%. The right most columns in the table are descriptive statistics of the sub-sample of individuals who have valid HbA1c levels and additionally have partners living in their household. This is also separately broken down into HbA1c levels above 6.0% and below 6.0%.

The Blood and Partners sample is substantially smaller than the Blood Sample for two reasons. Clearly, not all individuals included in the blood sample have partners, and therefore we would expect lower numbers, however we are also unable to identify partners for some years of the survey. Although we are able to match partners in most years of the survey, the public access version of the HSE for years 2015-2017 does not include a household identifier, and therefore we are unable to match partners in these years. There are also some variables, denoted with a † in table 1, which were not asked in every year of the survey, and therefore the number of observations for these variables are smaller than the total number of observations given at the bottom of the table. One example is physical activity, which was surveyed in the years 2003, 2004, 2006, 2008, 2012, 2016 only, and therefore we do not observe physical activity for all years. This is also true for household size and equivalized income, but for different years.

4 Identification Strategy

The main aim of this paper is to estimate the causal impact of own or partner's diabetes diagnosis on a variety of health related lifestyle behaviours, specifically, tobacco and alcohol consumption, physical activity and diet. This relationship can be described by the following

equation:

$$y_i = \theta_0 + \theta_1 EverDi_i + \theta_2 EverDi_j + e_i \tag{1}$$

where y_i denotes the health related lifestyle behaviour of interest and $EverDi_i$ denotes whether individual i has ever been diagnosed with diabetes, and $EverDi_j$ denotes whether the partner of individual i, person j, has ever been diagnosed with diabetes. A naive OLS of this form using survey data would most likely provide a biased estimate of both θ_1 and θ_2.

The first and possibly most salient source of bias is simultaneity. On average, when observed, individual with diabetes will behave in a way which is more damaging to their health than those without diabetes. This ignores the critical importance of knowing that these individuals would have been diagnosed as having diabetes precisely because they behaved in this damaging way. Indeed, the causes of type 2 diabetes are poor lifestyle factors, (Helmrich et al.; 2010; Pan et al.; 1997; Hu et al.; 2001; Liu et al.; 2000). To estimate the impact of diagnosis itself, we require an exogenous variation.

In addition, we suspect that if we were to estimate equation (1), θ_2 would also be biased. As discussed above, there has been some literature emphasising the importance of matching in the marriage market, indeed Dupuy and Galichon (2014) found there to be considerable matching in personality traits and attitude towards risk. This work, suggests that individuals selectively marry along similar traits and therefore if we naively ignore this channel our estimate of the spillover effect will be biased. Given that our specific aim is to estimate the spillovers of a diabetes diagnosis, we require an alternative means of identifying the causal effect of own and partner's diabetes diagnosis on behavioural outcomes.

4.1 Regression Kink Design

To identify the causal effect of diabetes diagnosis on health-related behaviours, we utilise a regression kink design, where the kink is a slope change in the treatment probability of a binary treatment variable. Figure (1) motivates the use of the RKD within this setting. As is shown, there is an increasing but consistently low probability of ever being diagnosed with diabetes when plotted against HbA1c, until the kink point of 6%, at which point there is a dramatic increase in the slop of the probability of being diagnosed. As discussed in the

background section of this paper, the NHS recommends that individuals who are tested and have a glycated hemoglobin (HbA1c) level above 6% are offered annual blood tests to monitor their blood sugar levels, and to diagnose diabetes as early as possible. The initial test could be for a variety of reasons, sometimes as part of a regular check up offered by the NHS, or if an individual shows symptoms that warrants a blood test. Our estimating strategy exploits this change in slope to causally estimate the impact of own and partner's diabetes diagnosis. Dong (2011) provides the theoretical framework for identification in this setting: the RKD identifies the causal effect of a binary treatment where there is not a discontinuity in the probability of treatment but rather a kink. It is worth emphasising the point we make earlier in the paper, that such a kink in the probability of a diabetes diagnosis is not supported in the medical sense. Yudkin and Montori state clearly that an inflection point of diabetes risk does not indeed exist, meaning that the assignment of diabetes risk is purely arbitrary.

To estimate the causal effect of diabetes diagnosis on health-related outcomes we use the RKD combined with an two-stage least squares (2SLS) specification. The first stage identifies the effect of the kink on probability of the treatment:

$$EverDi_i = \gamma_0 + \gamma_1(x_i - k)D_i + \left[\sum_{p=1}^{p^*} \nu_p^-(x_i - k)^p\right] + \left[\sum_{p=2}^{p^*} \nu_p^+(x_i - k)^p D_i\right] + \xi_i \qquad (2)$$

where $EverDi_i$ is a binary variable taking value of one for individual i if they have ever been diagnosed with diabetes, and zero otherwise. x_i denotes the running variable, which is HbA1c level in this case, and k is the kink point of 6%. $D_i = \mathbb{1}(x_i \geq k)$, is an indicator variable, taking the value of one if the individual's level of HbA1c is above the kink point, and where $(x_i - k)D_i$ is the excluded instrument for the fuzzy RKD. p^* denotes the highest order of polynomial used in the regressions, ν_p^- and ν_p^+ are the estimates of the polynomial function below and above the kink point respectively.

We then estimate the following second stage regression where the the kink is used as an instrument for the binary treatment, whether ever diagnosed with diabetes:

$$y_i = \beta_0 + \beta_1 \widehat{EverDi_i} + \left[\sum_{p=1}^{p^*} \alpha_p^-(x_i - k)^p\right] + \left[\sum_{p=2}^{p^*} \alpha_p^+(x_i - k)^p D_i\right] + \epsilon_i \qquad (3)$$

where y_i denotes the health related behavioural outcome of interest. $\widehat{EverDi}_i$ is the predicted probability, from the first stage, of ever being diagnosed with diabetes. Now, if the assumptions outlined by Dong (2011) and Card et al. (2015) hold, the coefficient β_1 can be interpreted as the unbiased local average treatment effect (LATE) of ever having been diagnosed with diabetes.

Our selection of outcomes focus on behaviours that both contribute to causing diabetes, and have been outlined as means of managing and treating diabetes (WHO; 2016). Low amounts of physical activity, poor diet, tobacco and alcohol consumption have all been shown to both cause diabetes and are first line treatments for managing the condition. The impact on exercise will be estimated using "any exercise done in the last four weeks", as the dependent variable. We will estimate the effects on diet by using two related variables, "whether consumed any vegetables yesterday" and "whether consumed any fruit yesterday". Estimated impact on smoking and drinking behaviour will be done by using "whether currently a smoker" and "whether currently a drinker".

This setup allows us to identify the causal effect of diabetes diagnosis on a number of health-related behaviours, because the kink provides us with an exogenous variation in diabetes probability. However, the identification of this effect relies on the intuition that those just to the left of the kink are almost identical to those just to the right of the kink and it was a random variation that resulted in them falling either side of the kink-point. This intuition is the same as is common for the RDD. As with the RDD identification strategy there is a bias-variance trade-off to be made when selecting the estimation sample. Larger samples are more likely to bias the estimates because other factors apart from the kink are likely to influence diabetes diagnosis, however small samples will not have sufficient power to reject a false null hypothesis. Because the RKD is more demanding on data than the RDD we require wider bandwidths than the RDD would require. To convince the reader that our results are not sensitive to alternative bandwidths, alongside our main results we present figures which show a number of alternative bandwidths. Given that we have very few observations of individuals whom have been diagnosed as having diabetes on the right hand side of the kink-point, we increase that bandwidth and keep the left-hand side bandwidth much narrower where low sample size is less of a problem.

Another consideration when using RDD or RKD as an identification strategy is the polynomial order used in the regressions. In line with Gelman and Imbens (2019)'s suggestions,

for our main results we use a quadratic polynomial specification to estimate our effects, however we also report cubic polynomial specifications.

4.2 Partner's Diabetes Status

To handle the endogeneity of partners' diabetes diagnosis on own behaviour, we extend the technique used for own diagnosis to partners' diagnosis. We do so by using own kink as an instrument for own probability of being diagnosed with having diabetes, and use partners' kink as an instrument for partners' probability of being diagnosed with diabetes.

In the first stage, we estimate the following first stage for both i and j:

$$EverDi_i = \lambda_0 + \lambda_1(x_i - k)D_i + \lambda_2(x_j - k)D_j + \left[\sum_{p=1}^{p^*} \theta_p^-(x_i - k)^p\right] + \left[\sum_{p=2}^{p^*} \theta_p^+(x_i - k)^p D_i\right] + \left[\sum_{p=1}^{p^*} \rho_p^-(x_j - k)^p\right] + \left[\sum_{p=2}^{p^*} \rho_p^+(x_j - k)^p D_j\right] + u_i \quad (4)$$

Where the subscript j denotes the partners', and the subscript i denotes the own, we replace i with j, and vice versa for partners' first stage equation.

We then estimate the following second stage to estimate the causal relationship:

$$y_i = \delta_0 + \delta_1 \widehat{EverDi}_i + \delta_2 \widehat{EverDi}_j + \left[\sum_{p=1}^{p^*} \pi_p^-(x_i - k)^p\right] + \left[\sum_{p=2}^{p^*} \pi_p^+(x_i - k)^p D_i\right] + \left[\sum_{p=1}^{p^*} \tau_p^-(x_j - k)^p\right] + \left[\sum_{p=2}^{p^*} \tau_p^+(x_j - k)^p D_j\right] + \varepsilon_i \quad (5)$$

The main difference between these estimations and those presented above are that $EverDi_j$ is whether partner has ever been diagnosed with diabetes, and x_j denotes the partners HbA1c level.

As discussed above, for us to identify a causal effect, we require reasonable bandwidths either side of the kink-point. We use the same bandwidths for partners' as we do for own, and therefore the final estimation sample are those whom have HbA1c levels within

the bandwidths, have partners, and those partners' also have HbA1c levels within the bandwidths.

5 Results

5.1 Effect of own diagnosis

We present estimates of the effect of own diabetes diagnosis on own behaviour in table 2. First of all we note the relevance of the kink as an instrument for ever being diagnosed with diabetes. In the third row we present the first stage coefficients of the effect of the kink on the probability of ever being diagnosed. As may have been clear by figure 1, we find a positive and highly statistically significant effect of the kink on probability of being diagnosed with diabetes. As is typical for instrument variable applications we use the p-value of the instrument in the first stage to assess the relevance of our instrument, and using this approach we can be confident that our instrument is relevant across all our estimated models.

The second row in table 2 shows the coefficient β_1 from equation (3). We find that being diagnosed as with diabetes has a significantly positive impact on probability of having done physical activity in the last four weeks. In terms of diet, we find no evidence to suggest an impact on consuming fruit consumption today, and no evidence that there is a change in vegetable consumption. There does also appear to be evidence that diabetes diagnosis reduced the probability of currently being a smoker. The final result of the effect of being diagnosed with diabetes on currently being a drinker is somewhat perplexing as we find that there is in fact a small but positive effect.

In addition to the estimates we present in table 2, we also show graphical results. Figure 2 shows the reduced form [2] quadratic prediction graphically imposed over the mean outcomes per bin for HbAqc levels. The graphs show a similar conclusion to table 2. We see that physical activity shows the clearest slope change around the kink point, whereas fruit, smoking and alcohol consumption show a far more subtle change in slope.

[2] where the reduced form is estimated using the following equation: $y_i = \chi_0 + \chi_1(x_i - k)D_i + \left[\sum_{p=1}^{p^*} \psi_p^-(x_i - k)^p\right] + \left[\sum_{p=2}^{p^*} \psi_p^+(x_i - k)^p D_i\right] + \mu_i$.

5.2 Sensitivity to alternative bandwidths and polynomials

To ensure that our results are not sensitive to alternative specifications and bandwidths we present a series of robustness graphs in figures 7 to 11. These graphs show the point estimate β_1, and the corresponding confidence intervals, from equation 3, estimated using a two-stage least squares, for each y_i that we present in the main text. Our specification varies by polynomial order, either quadratic or cubic, and the selected estimation sample, which we vary by selecting different upper and lower bounds. The lower bound describes the relative distance below the kink point, and upper bound being the relative distance above the kink point.

If we inspect the figure 7, where the dependent variable is physical activity, we see that across all specifications the point estimate is above zero and in most cases the confidence intervals do not include zero. The specifications at the right most side of the figure have much larger point estimates and confidence intervals, we believe that these specifications are over-fitting our data because they have both a narrow estimating sample window (in terms of the upper and lower bound) but also the most flexible polynomial. Indeed, when we inspect these specifications further we find that they are the ones with the highest out-of-sample mean squared error too. This is also true more generally for the specifications with wide confidence intervals. Overall, this figure gives us confidence in our estimate and proves that our physical activity estimate is not overly sensitive to specification.

Vegetable consumption and fruit consumption estimates in figures 8 and 9 do not warrant such a detailed analysis across specifications. In most specifications the point estimate is close to zero, and the confidence interval includes zero in the majority of cases. Once again, we are satisfied that our estimates are not sensitive to specification.

In terms of smoking behaviour, in figure 10, the story is much the same as for physical activity, where the point estimate does not vary much across specifications (until we reach the right hand tail), and the vast majority of specifications have tight confidence intervals which do not include zero. Therefore, as with physical activity we are not concerned that the results are sensitive to alternative specifications.

Finally, we present the alternative specifications of the effect of diabetes diagnosis on alcohol consumption in figure 11. The result of alcohol consumption which we present in table 2 was the most perplexing of all our results, as we would not expect diabetes diagnosis to

have an increasing effect on probability of alcohol consumption, however as is shown in figure 11, this is the effect that we find is most sensitive to specification choice. Not only does the point estimate lie both below and above zero depending on specification, we also see that in most specifications the point estimate is in fact insignificant. Given that this effect is clearly sensitive to specification, we proceed very cautiously when interpreting the estimates in table 2. The results with regards to alcohol consumption are the least conclusive of all the results we present in this paper, and although we do not conclude a finding of no effect, we acknowledge that this effect is sensitive to specification and therefore we will avoid making conclusive claims regarding this effect.

5.3 Effect of partner's diagnosis

The estimates of the impact of partners' diabetes diagnosis on own behaviour is presented in table 3. As for own diabetes diagnosis, we find that the instrument is once again relevant in this case too. Although, this is not particularly noteworthy, as the instrument and excluded variable are the same as for own diabetes diagnosis.

The second row of table 3 presents the two-stage least squares estimates of the effect. We find that, as for own diagnosis, partners' of those with diabetes have a higher probability of exercising in the past four weeks. In terms of diet, the effect diverges somewhat from the effect on own, and we find that there is a significantly negative impact on consuming both fruit and vegetables. Finally, there appears to be no significant impact on either currently being smoker, or currently being a drinker.

It is worth noting that individuals whom have ever been diagnosed as diabetes in our sample had, on average, been diagnosed 10 years ago (standard deviation of 10.5). Therefore, our results should be interpreted as the causal effect of being diagnosed at a random time post diagnosis. Given that the average person wit diabetes in our sample was diagnosed in the distant past, it is reassuring to see that individuals' maintain a higher probability of physical activity. This seems to suggest that the change in behaviour is persistent from the time of diagnosis.

As we did above, we also present graphical results of a simplified reduced form RKD [3]

[3] where the simplified reduced form is estimated using the following equation: $y_i = \chi_0 + \chi_1(x_i - k)D_i + \left[\sum_{p=1}^{p^*} \psi_p^-(x_i - k)^p\right] + \left[\sum_{p=2}^{p^*} \psi_p^+(x_i - k)^p D_i\right] + \mu_i$

in figure 3. The physical activity graph is once again the most prominent slope change, whereas there is only a modest slope change for fruit consumption and very little evidence of a slope change elsewhere.

5.4 Validity of Identifying Assumptions

For our presented estimates to be considered the LATE of diabetes diagnosis, it is required that the two testable implications outlined by Card et al. hold. The first of these observable implications is smooth density of the assignment variable, which tests the assumption that there is no deterministic sorting. The second observable implication is that there is no kink in the pre-determined covariates. In this section we will analyse these observable implications of Card et al. assumptions to ensure that they hold in our setting.

5.4.1 Smooth Density of the Assignment Variable

The first testable implication of the RKD is that of smooth density of the assignment variable, this implication tests the assumption that there is no deterministic sorting. We require that there is no discontinuity in the density of the assignment variable, which is the same assumption required for the RDD. However, the RKD assumption is stronger than the equivalent assumption for the RDD, as it additionally requires that there is not a kink in the density of the assignment variable. McCrary (2008) provides a test for assessing whether there is deterministic sorting by analysing whether a discountinuity exists at the cutoff, however this does not test the stronger version of the assumption requiring no kink. We therefore also follow Landais (2015) by extending the McCrary test to also test discontinuity in the first derivative.

Figure 4 presents graphically the density of the assignment variable by HbA1c. As can be seen in the graph, the density is neither uniform nor entirely smooth across the entire range of HbA1c levels, however it is clear that there is no graphical evidence of either a jump or a kink in the density at the kink point of 6% (red vertical line). The graph also shows the McCrary statistic and the statistic of the first derivative discontinuity test used by Landais, both of which are insignificant. These results are not particularly surprising, given that HbA1c is extremely difficult to exactly manipulate.

5.4.2 Predetermined Variables

The second testable implication of the RKD is that there should be no discountinuity or kink in the pre-determined variables, which tests the assumption that the marginal effect of the assignment variable on the outcome is smooth. As stated by Card et al. (2015), this is similar to the "test of random assignment" usually required in a randomized control trial. As above, this observable implication is more restrictive than the equivalent RDD observable implication. We require that there is neither a discontinuity *or* a kink in the pre-determined variables. In order to validate that this assumption holds in our setting we graphically present the mean values per bin by the assignment variable for a number of predetermined variables. Our strategy to assess whether the observable assumption holds in our setting relies on the visual inspection of these variables.

As Card et al. makes clear this observable implication relies on the existence of a set of variables which, by definition, are not determined by the treatment. We are somewhat limited in terms of the variables available at our disposal. As the HSE is a cross-sectional study, most survey questions ask about that given point in time, and do not substantively ask the respondents about past, and even in cases where they do so we can not necesserily determine whether this was prior to treatment or not. However, we do our best in this regards and present a number of variables in figure 5, these being: gender, ethnicity, whether individual has degree level education, whether the individual has any educational qualifications [4], whether a partner lives in the household, whether *ever* a smoker, whether *ever* a drinker, and age.

As can be seen in figure 5 there does not appear to be any clear discontinuities or kinks at the kink point of any of the variables that we present here. This validates that the assumptions hold in our analysis, and indeed that we can interpret the results of the RKD as LATEs.

[4] Any qualification corresponds to a long list of education qualifications surveyed in the HSE, which include (but is not limited to) degree education, high school and professional qualifications (i.e. teaching, nursing, vocational).

6 Heterogeneity

The next step in our analysis is to assess whether effects of a diabetes diagnosis are heterogenous across observables. In this section we explore whether heterogeneous effects do indeed exist for both own and partners'. Firstly, we explore whether those that live with a spouse behave differently to those that do not. We then analyse whether there is a differential impact by time since diagnosis, in an attempt to estimate whether the effects of a diagnosis varies over time, specifically assessing whether individual eventually converge back to pre-diagnosis behaviours. Finally, we estimate whether there is a differential effect by education levels for both own and partners'.

To estimate these effects, we derive the Hetergoenous Local Average Treatment Effect (HLATE) in a similar vein as Becker et al. (2013), by replacing the LATE of $EverDi_i$ in equation 3 and manipulating it to allow for heterogeneous effects along the variable z_i. We do so in the usual way by replacing the coefficients with an interaction, so in the general case $\gamma = \widehat{\gamma} + \widetilde{\gamma} z_i$ where z_i denotes the trait which we suspect there is heterogeneity across. The first stage equation is then re-written as:

$$EverDi_i = \widehat{\gamma_0} + \widetilde{\gamma_0} z_i + \widehat{\gamma_1}(x_i - k)D_i + \widetilde{\gamma_0}(x_i - k)D_i z_i + \sum_{p=1}^{p^*} \left[\widehat{\nu_p^-}(x_i - k)^p + \widetilde{\nu_p^-}(x_i - k)^p z_i \right] + \sum_{p=2}^{p^*} \left[\widehat{\nu_p^+}(x_i - k)^p D_i + \widetilde{\nu_p^+}(x_i - k)^p D_i z_i \right] + \upsilon \quad (6)$$

The second stage of the the 2SLS is then described by:

$$y_i = \widehat{\beta_0} + \widetilde{\beta_0} z_i + \widehat{\beta_1} \widehat{EverDi_i} + \widetilde{\beta_1} \widehat{EverDi_i} z_i + \sum_{p=1}^{p^*} \left[\widehat{\alpha_p^-}(x_i - k)^p + \widetilde{\alpha_p^-}(x_i - k)^p z_i \right] + \sum_{p=2}^{p^*} \left[\widehat{\alpha_p^+}(x_i - k)^p D_i + \widetilde{\alpha_p^+}(x_i - k)^p D_i z_i \right] + \psi_i \quad (7)$$

The inclusion of an additional term to estimate $\widetilde{\beta_1}$ which is dependent on the endogenous variable $EverDi_i$ requires an additional instrument for the 2SLS estimates to be correctly

identified. I therefore also estimate the following regression in the first stage:

$$EverDi_i z_i = \widehat{\vartheta_0} + \widetilde{\vartheta_0} z_i + \widehat{\vartheta_1}(x_i - k)D_i + \widetilde{\vartheta_1}(x_i - k)D_i z_i + \sum_{p=1}^{p^*} \left[\widehat{\sigma_p^-}(x_i - k)^p + \widetilde{\sigma_p^-}(x_i - k)^p z_i\right] + \sum_{p=2}^{p^*} \left[\widehat{\sigma_p^+}(x_i - k)^p D_i + \widetilde{\sigma_p^+}(x_i - k)^p D_i z_i\right] + \zeta_i \quad (8)$$

We additionally extend this approach to partners', to estimate whether there is heterogeneity in their behaviour, by their own educational level and the time since diagnosis of the person with diabetes.

This estimation strategy is very similar to that of Becker et al. (2013)'s strategy to estimate the HLATE, and is one we follow closely, the key difference is of course that our estimation uses an RKD unlike an RDD in Becker et al.'s case.

For us to indeed estimate a HLATE we require that two additional assumptions hold in addition to those discussed in the previous section, these are outlined by Becker et al.. The first of these is that there is continuity of the interaction variables at the threshold vector. In our setting we require a strong version of this assumption, namely we require that there is neither a jump nor a kink in the interaction variables at the threshold. To check whether this assumptions holds, we plot the average per bin of the interaction variables against Glycated Hemoglobin (HbA1c) to visually check as to whether this assumption holds. Figure 5 shows, amongst other variables, whether individual has degree level education. As can be seen, and as is discussed above, these variables do not clearly show either a jump or a kink at the threshold HbA1c level of 6%, and therefore we are confident that this assumption holds.

Time since diagnosis is dealt with in a slightly different way. Clearly, for those that have never been diagnosed they will not have an observed time since diagnosis. To handle this problem, we employ the method used by Kleven et al. (2019) and assign placebo time since diagnosis for those individuals whom were never diagnosed with diabetes. We assign placebo values of time since diagnosis by drawing observations from a log-normal in the same way Kleven et al. does. This allows us to have time since diagnosis observations for each individual and we can therefore estimate the HLATE effect by time since diagnosis. For the analysis, we additionally demean the variable so that $\widehat{\beta_1}$ represents the effect for

the average time since diagnosis.

The second required assumption is the random assignment of the interaction variables conditional on the running variable. In this setting, we require that z_i is not correlated with the error term in the estimating equation. To ensure that this is indeed the case, we include a number of observable individual level characteristics in the estimating equations, these being: a binary of whether individual is male, a binary indicator of whether individual is white, a continuous age variable, we also include a binary indicator of whether individual has degree level education in the estimating equations where we are not directly estimating the heterogeneity along this dimension.

6.1 Partner in Household

Firstly, table 4 shows the estimates of the heterogeneity of the effect of own diabetes diagnosis, by whether an individual lives with a partner or not. Across most of the health-related outcomes there appears to be little heterogeneity, apart from vegetable consumption. It appears to be the case that those with diabetes who do not live with partners are more likely to consume vegetables, and indeed increase their probability of vegetable consumption by around 22%. However, people with diabetes and do live with a partner have a lower probability of consuming vegetables, and this reduction is of a similar magnitude as those without partners' increase their consumption in response to a diagnosis. The inference here is that those living with partners' do not change their vegetable consumption as a result of a diabetes diagnosis.

6.2 Time Since Diagnosis

Tables 5 and 6 show the heterogenous impact of time since diagnosis on own and partners' respectively. Across all interactions for both own and partners', there appears to be no significant heterogeneity by time since diagnosis. Which supports a hypothesis of habit formation. Individuals significantly increase their physical activity and decrease their smoking behaviour, and consistently maintain this new state. This result is somewhat different to the findings of Kim et al. (2019) who find that for their outcomes measures (outpatient visits, medicated days, basic exercise) there was no significant effect in the long-run. However, our results suggest that the effect of a diagnosis is persistent, rather than limited to

the short-run.

It is also reassuring to note that time since diagnosis is insignificant in all models, apart from one. This is precisely what we would expect, given that time since diagnosis for individuals who have not had a diabetes diagnosis is a placebo time since diagnosis, or placebo time since partners' diagnosis.

6.3 Education

Finally, heterogeneity in terms of educational attainment is presented in tables 7 and 8. Once again, here we find that there is limited evidence of a heterogeneous effect. Fruit consumption does appear to show some heterogeneity. On average, those with degree-level education are more likely to consume fruit than those with lower educational attainment. In response to a diabetes diagnosis, highly educated individuals appear to decrease their fruit consumption, whereas those with lower educational attainment appear to increase their fruit consumption. It is plausibly the case that higher education individuals are able to better absorb information regarding the high sugar levels of fruit, and decrease their consumption of it accordingly. Whereas in an attempt to improve diet less educated individuals choose to increase their fruit consumption. On average, those individuals may be less able to absorb or seek new health information, and do not realise that higher fruit consumption may actually negatively effect their health, due to to the high sugar content of those foods. We find a similar partner for partners' of those with diabetes, where partners' with degree level education decrease their fruit consumption, whereas we find no effect for individuals who do not have degree level education.

7 Conclusion

In this paper, we find that individuals whom have ever been diagnosed with diabetes do significantly increase their physical activity and have a lower probability of currently being a smoker, which suggests that there is compliance to first line treatment, at least in terms of physical activity. We additionally claim that our results suggest that there is a persistence in the effect, given that we observe individuals, on average 10 years post their initial diabetes diagnosis, and find no evidence of a change in behaviours over time.

We also find that there is substantial spillover effects from diabetes diagnosis in the form of an increase in physical activity. Our results are consitent with Clark and Etil (2006), in that we find no evidence that there is a spillover in smoking behaviour. Our identification strategy allows us to make the claim that this effect is a combination of joint household decision making and health-related information transfer between partners.

From a public health perspective, it is reassuring to note that there is compliance to diabetes treatment in terms of physical activity. Our findings should also be of interest to policy makers as we show that there are substantial additional benefits, in terms of increased physical activity of partners', from a diabetes diagnosis, beyond what might be usually considered in economic evaluations. However, our results also suggest that more work should be done in promoting healthy diets, and reduced alcohol consumption in individuals diagnosed with diabetes and their partners.

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Tables and Figures

Table 1: Descriptive Statistics

	HSE Adult Sample	Blood Sample			Blood and Partner Sample		
		All	Below Kink	Above Kink	All	Below Kink	Above Kink
Observable Characteristics							
Age†	50.32	52.14	49.69	64.34	52.38	50.46	62.51
	(18.67)	*(17.57)*	*(17.25)*	*(13.59)*	*(15.21)*	*(14.89)*	*(12.70)*
Males	0.46	0.47	0.47	0.51	0.51	0.50	0.58
	(0.50)	*(0.50)*	*(0.50)*	*(0.50)*	*(0.50)*	*(0.50)*	*(0.49)*
White	0.86	0.90	0.91	0.88	0.91	0.92	0.87
	(0.34)	*(0.29)*	*(0.29)*	*(0.32)*	*(0.29)*	*(0.28)*	*(0.33)*
Any Qualifications	0.74	0.76	0.80	0.59	0.77	0.80	0.62
	(0.44)	*(0.43)*	*(0.40)*	*(0.49)*	*(0.42)*	*(0.40)*	*(0.49)*
Degree level education	0.21	0.23	0.24	0.13	0.23	0.25	0.14
	(0.41)	*(0.42)*	*(0.43)*	*(0.34)*	*(0.42)*	*(0.43)*	*(0.35)*
Partner living in household †	0.64	0.67	0.68	0.64	–	–	–
	(0.48)	*(0.47)*	*(0.47)*	*(0.48)*			
Household Size†	2.66	2.56	2.65	2.14	2.88	2.95	2.57
	(1.39)	*(1.31)*	*(1.33)*	*(1.14)*	*(1.17)*	*(1.18)*	*(1.05)*
Employed	0.59	0.60	0.65	0.36	0.66	0.70	0.43
	(0.49)	*(0.49)*	*(0.48)*	*(0.48)*	*(0.47)*	*(0.46)*	*(0.49)*
Equivalised Income†	29,920.77	31,176.52	32,276.71	25,478.14	33,001.36	34,184.01	26,439.02
	(27,403.78)	*(27,695.47)*	*(28,047.43)*	*(25,035.71)*	*(26,122.98)*	*(26,404.12)*	*(23,447.45)*
Subjective Health either good or excellent (%)	0.73	0.76	0.80	0.56	0.78	0.82	0.59
	(0.44)	*(0.43)*	*(0.40)*	*(0.50)*	*(0.41)*	*(0.39)*	*(0.49)*
Glycated Hemoglobin (HbA1c)	–	5.61	5.39	6.74	5.60	5.39	6.73
		(0.75)	*(0.33)*	*(1.17)*	*(0.74)*	*(0.32)*	*(1.16)*
Stated Behaviours							
Physical Activity (%)†	0.43	0.46	0.49	0.26	0.45	0.48	0.27
	(0.50)	*(0.50)*	*(0.50)*	*(0.44)*	*(0.50)*	*(0.50)*	*(0.44)*
Vegetable Consumption (%)	0.53	0.54	0.54	0.54	0.53	0.53	0.55
	(0.50)	*(0.50)*	*(0.50)*	*(0.50)*	*(0.50)*	*(0.50)*	*(0.50)*
Fruit Consumption (%)	0.62	0.63	0.62	0.67	0.63	0.62	0.67
	(0.49)	*(0.48)*	*(0.48)*	*(0.47)*	*(0.48)*	*(0.48)*	*(0.47)*
Currently a drinker (%)	0.77	0.82	0.84	0.72	0.84	0.86	0.76
	(0.42)	*(0.39)*	*(0.37)*	*(0.45)*	*(0.36)*	*(0.35)*	*(0.43)*
Currently a smoker (%)	0.20	0.18	0.18	0.17	0.16	0.16	0.16
	(0.40)	*(0.38)*	*(0.39)*	*(0.38)*	*(0.37)*	*(0.37)*	*(0.37)*
Number of Observations	124,260	57,964	48,373	9,591	31,215	26,238	4,977

Table shows the mean and, in parentheses, the standard deviation of observable characteristics and stated behaviours. The HSE adult sample column shows the descriptive statistics for the entire Health Survey for England sample, including those that did not have valid HbA1c measurements. The blood sample column shows only the sub-sample of individuals whom we have valid HbA1c measurements for. Blood and Partner sample represents the sub-sample of individuals who had both valid HbA1c measurements and that we were able to identify partners in the Health Survey for England. Below kink columns represent the sub-sample of individuals with HbA1c levels below 6.0%, and above kink columns represent the sun-sample of individuals with HbA1c levels above 6.0%.
† denotes variables which were not available to us for all years of the survey, and therefore the true number of observations used to calculate them are less than the number of observations denoted at the bottom of the table.

Table 2: Fuzzy Regression kink estimates of change in own Behaviour as a result own Diabetes Status

	Exercise	Vegetable Consumption	Fruit Consumption	Currently a Smoker	Currently a drinker
Reduced Form					
Effect of Own Diabetes	0.124***	0.0436	0.0380	-0.187***	0.0328*
	(0.0354)	(0.0310)	(0.0293)	(0.0218)	(0.0195)
Two-stage Least Squares					
Effect of Own Diabetes	0.303***	0.0825	0.0721	-0.345***	0.0606*
	(0.0881)	(0.0589)	(0.0558)	(0.0412)	(0.0362)
First Stage Coefficient	0.410***	0.528***	0.527***	0.541***	0.541***
	(0.0233)	(0.0181)	(0.0181)	(0.0167)	(0.0167)
Observations	28725	44065	44096	49096	49103

Coefficients are estimated using a quadratic specification each side of the kink point, equally weighted observations, and with bounds of 5.0 on the right hand tail, and 2.0 on the left hand side. Parentheses includes cluster-robust standard errors, clustered at the household level. Each specification includes the following controls: Age and dummies for whether individual i is male, white and has degree level education. First stage coefficient corresponds to γ_2 in equation (2). The reduced form is estimate is the coefficient χ_1 in the following model: $y_i = \chi_0 + \chi_1(x_i - k)D_i + \left[\sum_{p=1}^{p^*} \psi_p(x_i - k)^p\right] + \left[\sum_{p=2}^{p^*} \psi_p^*(x_i - k)^p D_i\right] + \mu_i$.

*** denotes P-value of 0.01 or less , ** denotes P-value of 0.05 or less, * denotes P-value of 0.10 or less

Table 3: Fuzzy Regression kink estimates of change in own Behaviour as a result Partners' Diabetes Status

	Exercise	Vegetable Consumption	Fruit Consumption	Currently a Smoker	Currently a drinker
Reduced Form					
Effect of Partner's Diabetes	0.243***	-0.0843*	-0.0960**	0.00317	0.00615
	(0.0536)	(0.0441)	(0.0426)	(0.0285)	(0.0285)
Two-stage Least Squares					
Effect of Partner's Diabetes	0.589***	-0.182*	-0.215**	0.0152	0.00866
	(0.166)	(0.0957)	(0.0926)	(0.0612)	(0.0608)
First Stage Coefficient	0.346***	0.457***	0.457***	0.466***	0.465***
	(0.0334)	(0.0270)	(0.0270)	(0.0251)	(0.0250)
Observations	11224	16804	16808	18773	18773

Coefficients are estimated using a quadratic specification each side of the kink point, equally weighted observations, and with bounds of 6.0 on the right hand tail, and 3.0 on the left hand side. Parentheses includes cluster-robust standard errors, clustered at the household level. First stage coefficient corresponds to λ_1 in equation (4). Effect of Partner's Diabetes in the two stage least squares section of the table corresponds to δ_2 in equation(4)). The reduced form is estimate is the coefficient γ_2 in the following model: $y_i = \delta_0 + \gamma_1 \widehat{Ever Di}_i + \gamma_2 \widehat{Ever Di}_j + \left[\sum_{p=1}^{p^*} \varrho_p (x_i - k)^p\right] + \left[\sum_{p=2}^{p^*} \varrho_p^* (x_i - k)^p D_i\right] + \left[\sum_{p=1}^{p^*} \varrho_p (x_j - k)^p\right] + \left[\sum_{p=2}^{p^*} \varrho_p^* (x_j - k)^p D_j\right] + \upsilon_i$
*** denotes P-value of 0.01 or less , ** denotes P-value of 0.05 or less, * denotes P-value of 0.10 or less

Table 4: Heterogeneous Fuzzy Regression kink estimates of change in own Behaviour as a result own Diabetes Status by whether individual has a partner

	Exercise	Vegetable Consumption	Fruit Consumption	Currently a Smoker	Currently a drinker
Two-stage Least Squares					
Effect of Own Diabetes	0.324**	0.224**	-0.0325	-0.322***	0.107
	(0.158)	(0.101)	(0.0966)	(0.0752)	(0.0669)
Partner - Diabetes Interaction	-0.0312	-0.216*	0.152	-0.0340	-0.0653
	(0.189)	(0.124)	(0.118)	(0.0892)	(0.0788)
Partner in household	-0.00892	0.0575***	0.0245*	-0.0724***	0.0358***
	(0.0191)	(0.0153)	(0.0144)	(0.0111)	(0.00978)
Observations	28725	44065	44096	49096	49103

Coefficients are estimated using a quadratic specification each side of the kink point, equally weighted observations, and with bounds of 5.0 on the right hand tail, and 2.0 on the left hand side. Parentheses includes cluster-robust standard errors, clustered at the household level. *** denotes P-value of 0.01 or less , ** denotes P-value of 0.05 or less, * denotes P-value of 0.10 or less

Table 5: Heterogeneous Fuzzy Regression kink estimates of change in own Behaviour as a result own Diabetes Status by Time Since Diagnosis

	Exercise	Vegetable Consumption	Fruit Consumption	Currently a Smoker	Currently a drinker
Two-stage Least Squares					
Effect of Own Diabetes	0.298***	0.0754	0.0763	-0.350***	0.0622*
	(0.0883)	(0.0596)	(0.0565)	(0.0421)	(0.0368)
Time Since Diagnosis - Diabetes Diagnosis Interaction	0.000791	0.00536	-0.00183	0.00345	-0.000582
	(0.00820)	(0.00489)	(0.00477)	(0.00324)	(0.00293)
Time Since Diagnosis	-0.000388	-0.000423	0.000196	-0.000794*	0.0000779
	(0.000827)	(0.000637)	(0.000602)	(0.000434)	(0.000373)
Observations	28725	44065	44096	49096	49103

Coefficients are estimated using a quadratic specification each side of the kink point, equally weighted observations, and with bounds of 5.0 on the right hand tail, and 2.0 on the left hand side. Parentheses includes cluster-robust standard errors, clustered at the household level. Estimates include the additional covariates: a dummy for whether individual is a male, age, a dummy for whether individual is white, and a dummy for whether individual has degree level education. *** denotes P-value of 0.01 or less , ** denotes P-value of 0.05 or less, * denotes P-value of 0.10 or less

Table 6: Heterogeneous Fuzzy Regression kink estimates of change in own Behaviour as a result Partner's Diabetes Status by their time since diagnosis

	Exercise	Vegetable Consumption	Fruit Consumption	Currently a Smoker	Currently a drinker
Two-stage Least Squares					
Effect of Partner's Diabetes	0.305*	-0.130	-0.0862	-0.109*	-0.0292
	(0.158)	(0.0983)	(0.0937)	(0.0627)	(0.0553)
Time Since Diagnosis - Diabetes Interaction	0.00199	0.0258**	-0.0106	-0.00468	-0.00685
	(0.0174)	(0.0116)	(0.0100)	(0.00627)	(0.00559)
Time Since Partner's Diagnosis	0.000334	-0.00278**	0.00171	0.000746	0.00108*
	(0.00162)	(0.00129)	(0.00115)	(0.000699)	(0.000623)
Observations	11204	16769	16773	18734	18734

Coefficients are estimated using a quadratic specification each side of the kink point, equally weighted observations, and with bounds of 6.0 on the right hand tail, and 3.0 on the left hand side. Parentheses includes cluster-robust standard errors, clustered at the household level. *** denotes P-value of 0.01 or less , ** denotes P-value of 0.05 or less, * denotes P-value of 0.10 or less

Table 7: Heterogeneous Fuzzy Regression kink estimates of change in own Behaviour as a result own Diabetes Status by Educational level

	Exercise	Vegetable Consumption	Fruit Consumption	Currently a Smoker	Currently a drinker
Two-stage Least Squares					
Effect of Own Diabetes	0.348***	0.119*	0.124**	-0.341***	0.0332
	(0.0985)	(0.0663)	(0.0632)	(0.0478)	(0.0411)
Education - Diabetes Interaction	-0.299	-0.132	-0.264**	0.0433	0.163*
	(0.220)	(0.140)	(0.134)	(0.0883)	(0.0867)
Degree level Education	0.230***	0.0980***	0.125***	-0.123***	0.0372***
	(0.0238)	(0.0184)	(0.0169)	(0.0111)	(0.0105)
Observations	28725	44065	44096	49096	49103

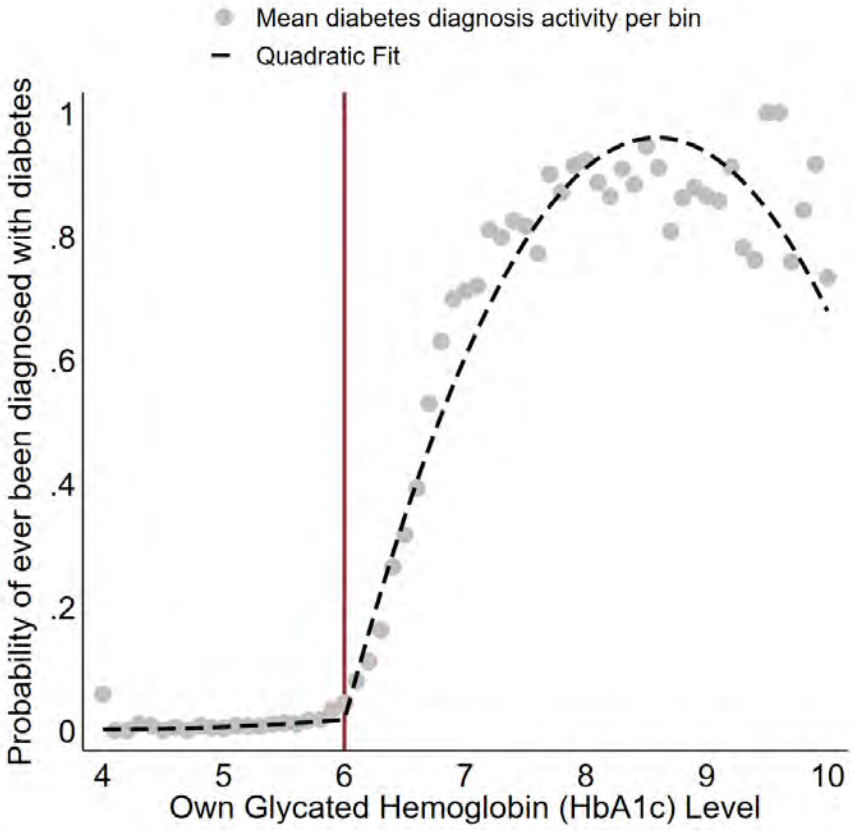
Coefficients are estimated using a quadratic specification each side of the kink point, equally weighted observations, and with bounds of 5.0 on the right hand tail, and 2.0 on the left hand side. Parentheses includes cluster-robust standard errors, clustered at the household level. Estimates include the additional covariates: a dummy for whether individual is a male, age, a dummy for whether individual is white. *** denotes P-value of 0.01 or less , ** denotes P-value of 0.05 or less, * denotes P-value of 0.10 or less

Table 8: Heterogeneous Fuzzy Regression kink estimates of change in own Behaviour as a result Partner's Diabetes Status by Education

	Exercise	Vegetable Consumption	Fruit Consumption	Currently a Smoker	Currently a drinker
Two-stage Least Squares					
Effect of Partner's Diabetes	0.323*	-0.0749	-0.00373	-0.125*	-0.0170
	(0.165)	(0.106)	(0.103)	(0.0709)	(0.0600)
Education - Diabetes Interaction	-0.227	-0.154	-0.446*	0.129	-0.157
	(0.449)	(0.253)	(0.232)	(0.120)	(0.133)
Degree level Education	0.171***	0.0743**	0.137***	-0.0861***	0.0398**
	(0.0436)	(0.0301)	(0.0277)	(0.0142)	(0.0158)
Observations	11218	16791	16795	18756	18756

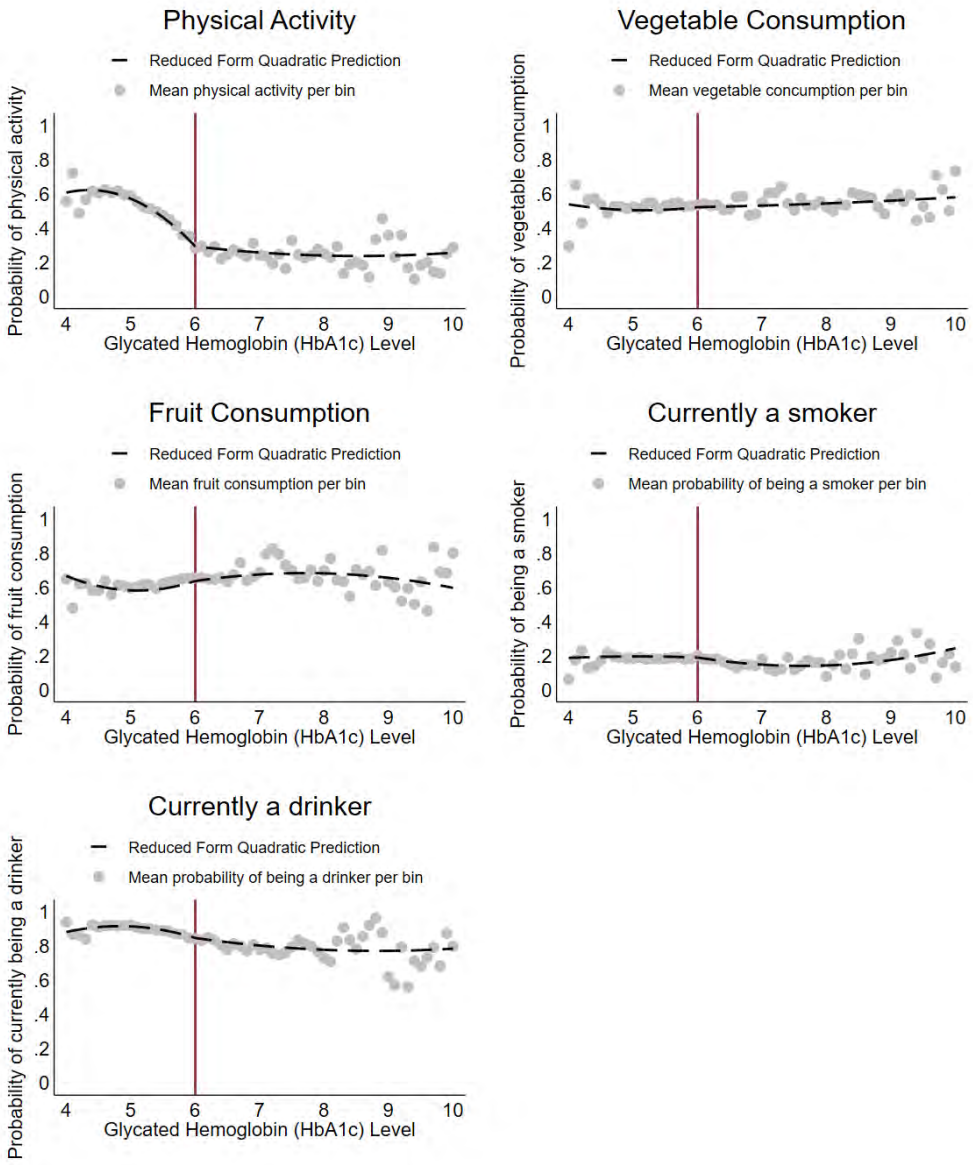
Coefficients are estimated using a quadratic specification each side of the kink point, equally weighted observations, and with bounds of 6.0 on the right hand tail, and 3.0 on the left hand side. Parentheses includes cluster-robust standard errors, clustered at the household level. *** denotes P-value of 0.01 or less , ** denotes P-value of 0.05 or less, * denotes P-value of 0.10 or less

Figure 1: Probability of Diabetes Diagnosis by HbA1c Level



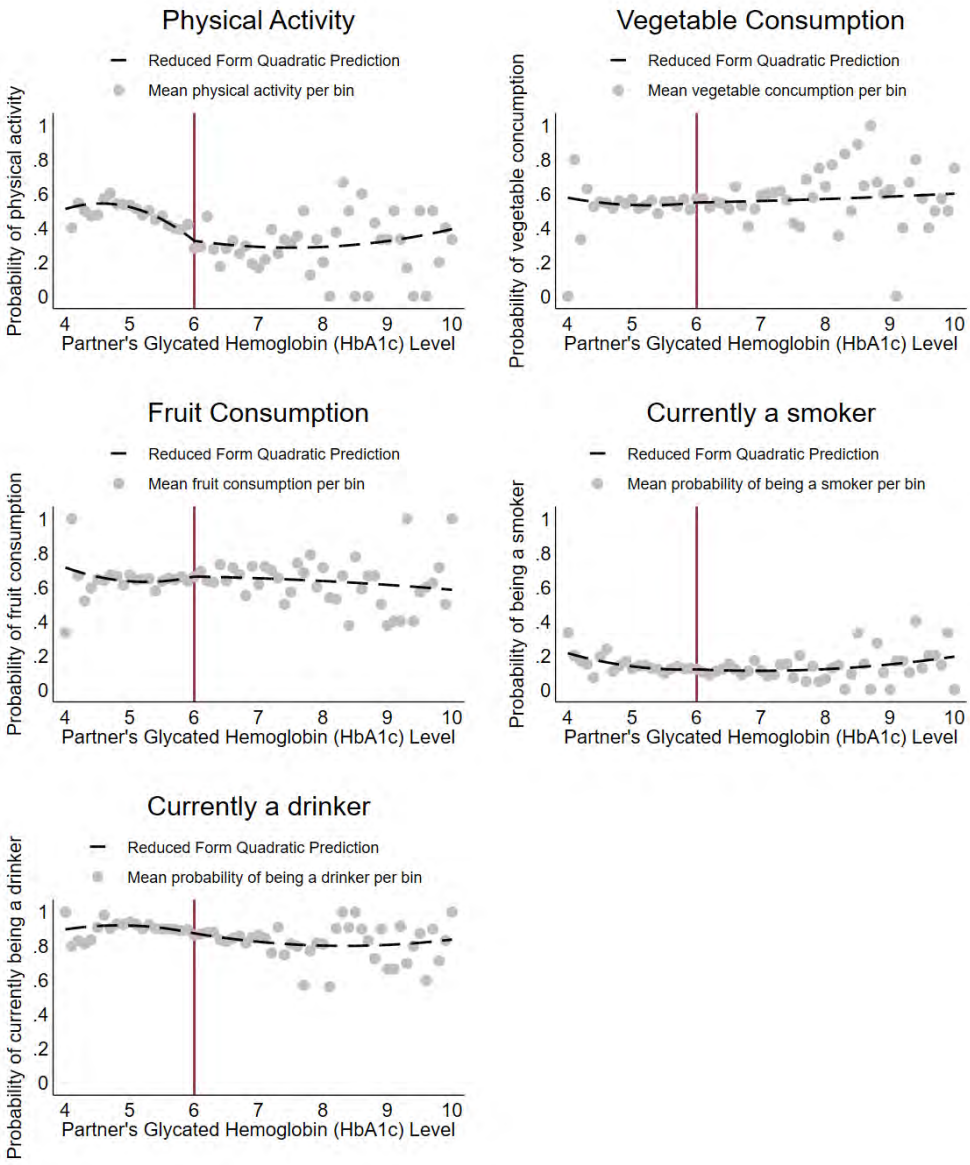
NOTE: Mean of the probability of ever being diagnosed with diabetes per bin. Bin width of 0.1 for glycated hemoglobin levels between 4 and 10. Quadratic fit is separately estimated for the left and right hand sides of the kink. Red line represents the kink point, where glycated hemoglobin is a value of 6.0.

Figure 2: Graphical Representation of Reduced Form RKD Results - Own Glycated Hemoglobin



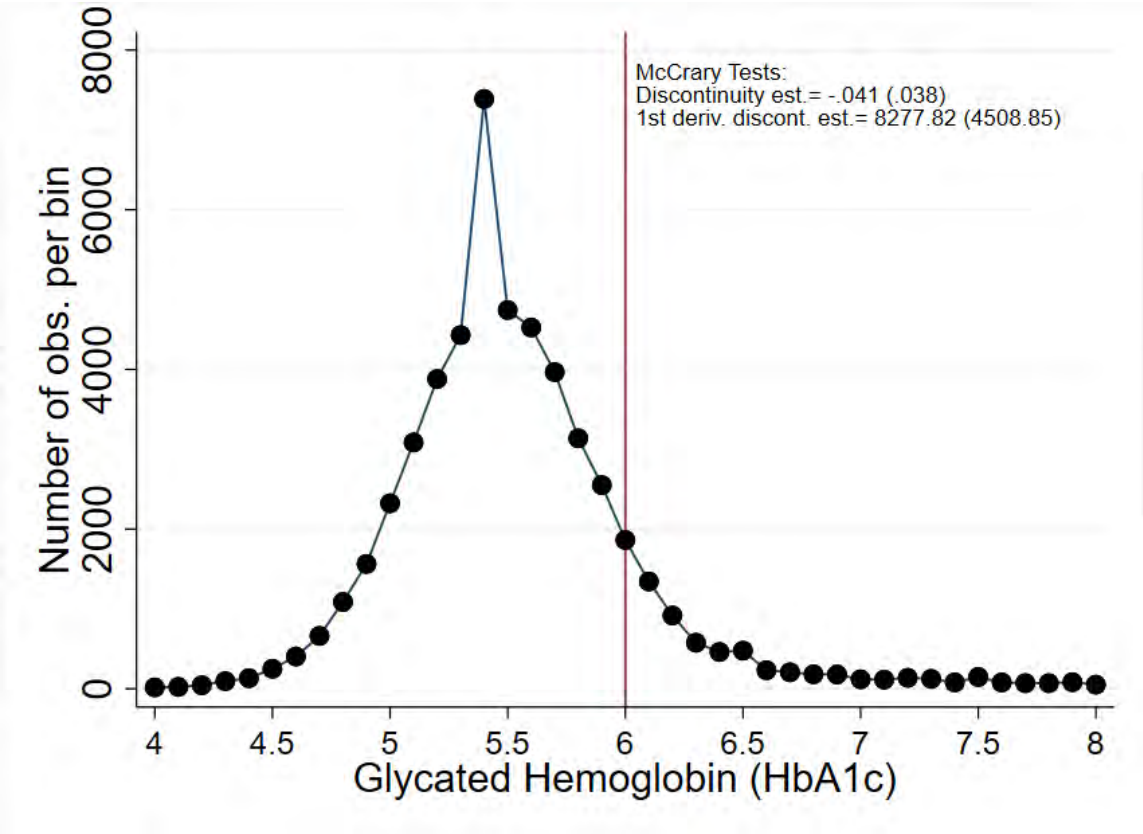
NOTE: These figures are a graphical representation of the RKD. Figures show the mean outcomes per bin (grey points), where bin width is 0.1, between HbA1c levels between 4.0 and 10.0. Black dashed line represents the quadratic prediction from the reduced form a regression of the form: $y_i = \chi_0 + \chi_1(x_i - k)D_i + \left[\sum_{p=1}^{p^*} \psi_p^-(x_i - k)^p\right] + \left[\sum_{p=2}^{p^*} \psi_p^+(x_i - k)^p D_i\right] + \mu_i$. The red lines represents the kink point where HbA1c is 6.0%. Precise estimates of the fuzzy RKD using equations (2) and (3) are available in table (2).

Figure 3: Graphical Representation of Reduced Form RKD Results - Partner's Glycated Hemoglobin



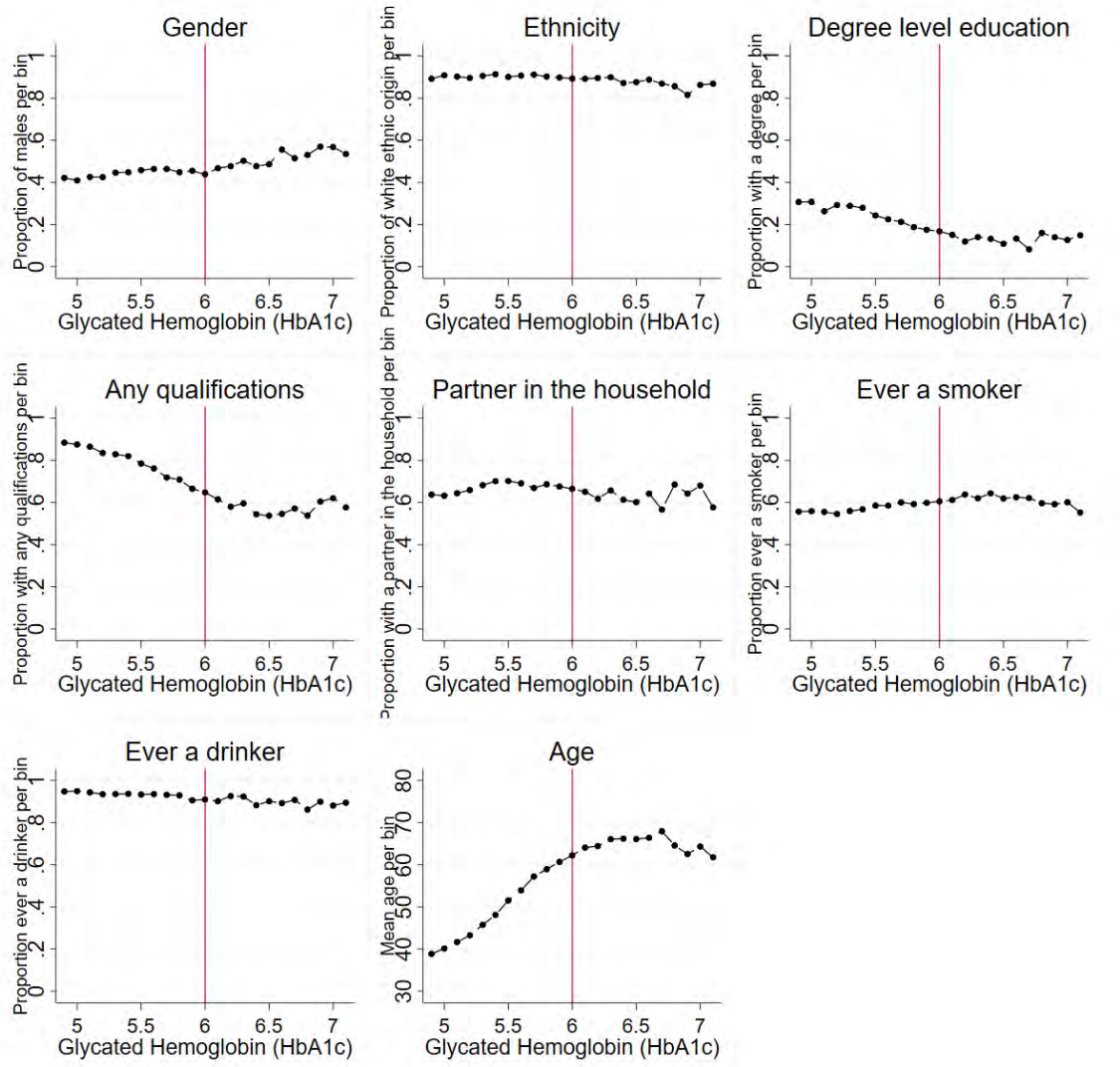
NOTE: These figures are a graphical representation of the partner RKD. Figures show the mean outcomes per bin, where bin width is 0.1 (grey points), between HbA1c levels between 4.0 and 10.0. Black dashed line represents the quadratic prediction from the reduced form a regression of the form: $y_i = \sigma_0 + \sigma_1(x_j - k)D_j + \left[\sum_{p=1}^{p^*} \phi_p^-(x_j - k)^p\right] + \left[\sum_{p=2}^{p^*} \phi_p^+(x_j - k)^p D_j\right] + \zeta_i$. The red lines represents the kink point where HbA1c is 6.0%. Precise estimates of the fuzzy RKD using the first stage and second stage in equations (4) and (5) respectively are available in table (3).

Figure 4: Smooth Density of the Assignment Variable



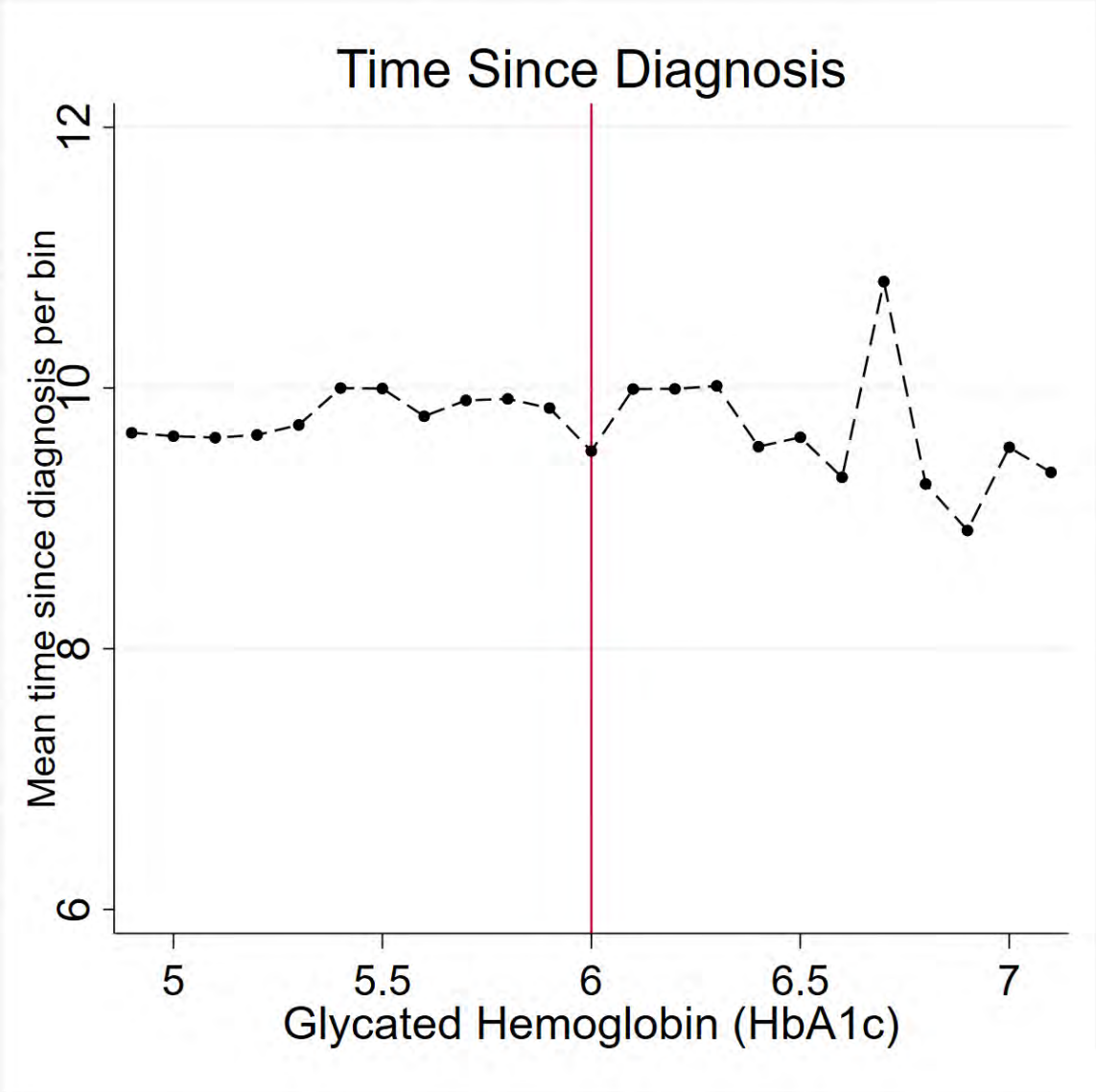
NOTE: Number of observations per bin. Bin width of 0.1 for glycated hemoglobin levels between 4 and 8. Graph also shows McCrary discontinuity statistic and the first derivative discontinuity statistic, both of which are insignificant.

Figure 5: Predetermined variables



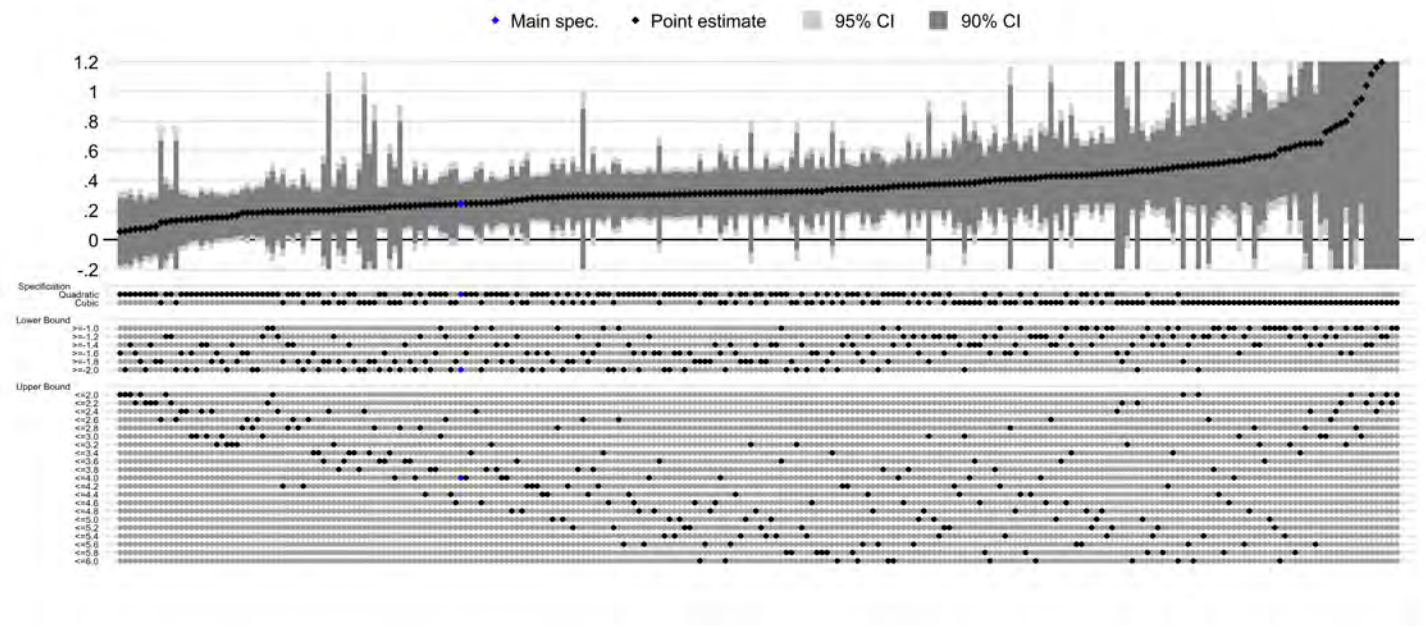
NOTE: Graphical representation of the mean of each predetermined variable by glycated hemoglobin (HbA1c) level. Each graph shows the mean of the predetermined variable per bin, with a bin width of 0.1. Predetermined variables included are gender, ethnicity, degree level education, any qualifications, whether a partner lives in the household, whether ever a smoker, whether ever a drinker and age. Red line represents the kink point of 6.0 %.

Figure 6: Time Since Diagnosis



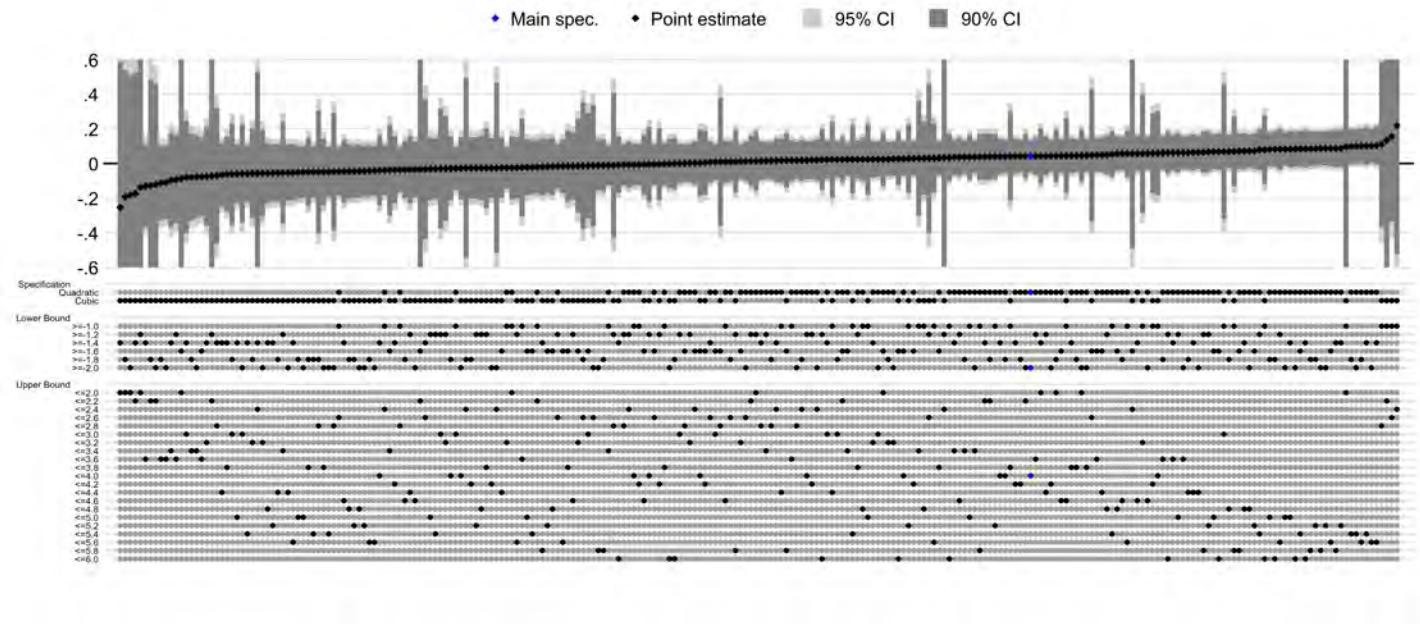
NOTE: Graphical representation of the mean of time since diabetes diagnosis by glycated hemoglobin (HbA1c) level. Graph shows the mean of the time since diagnosis per bin, with a bin width of 0.1. For individuals whom have never been diagnosed as having diabetes, they are assigned a placebo time since diagnosis, and are also included in this graph. Red line represents the kink point of 6.0 %.

Figure 7: Sensitivity to alternative bandwidths and polynomials - Physical Activity



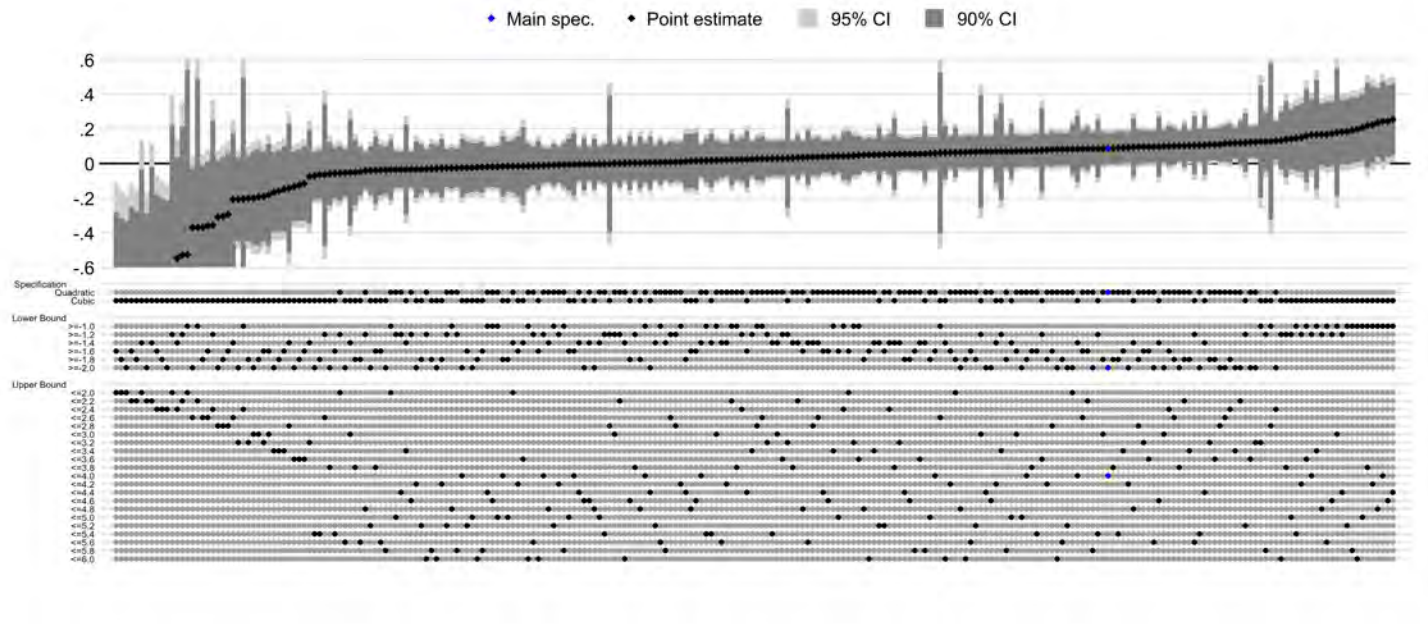
NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents that it was estimated using that polynomial, lower bound and upper bound. We estimate alternative specifications by, the order of polynomial, the upper bound of the estimation sample, and the lower bound of the estimation sample, and present all possible combinations, none of which are excluded from this figure. The blue dot represents the main specification which we present in our tables in the main text. We thank Peter Eibich, Uri Simonsohn and Hans H. Sievertsen for developing the idea and the code for this figure. Stata code is available at *https://github.com/hhsievertsen/speccurve*.

Figure 8: Sensitivity to alternative bandwidths and polynomials - Vegetable Consumption



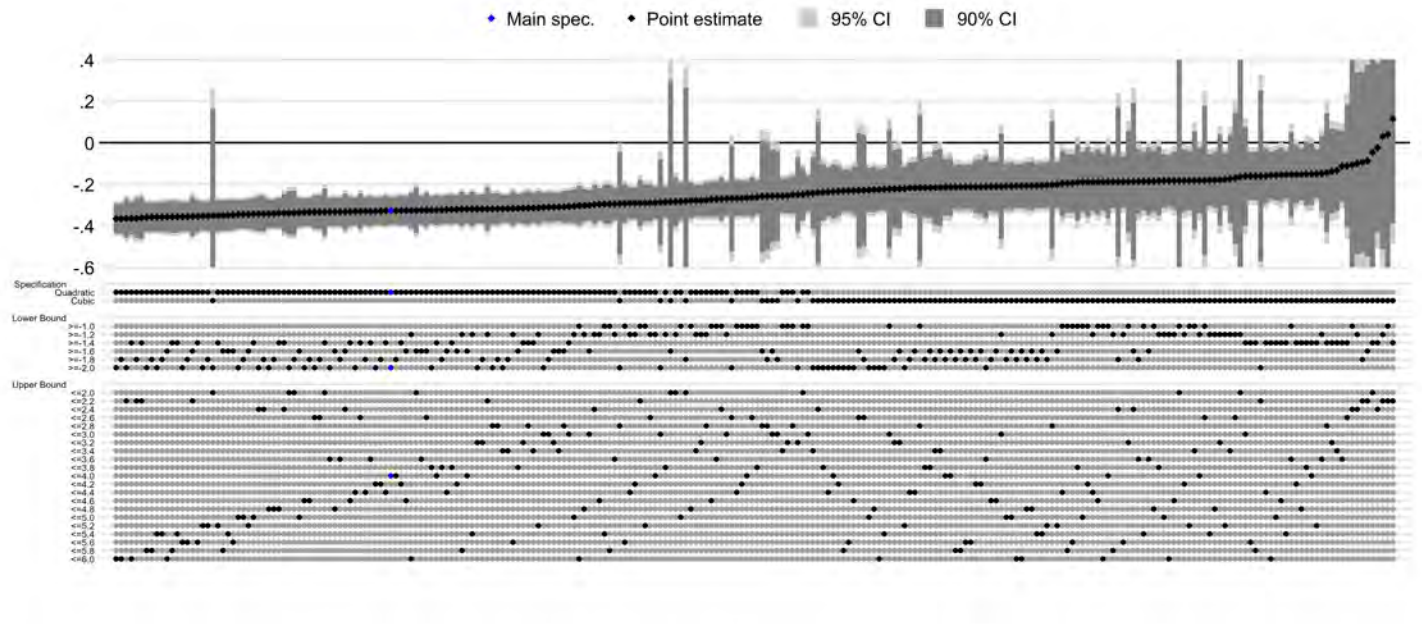
NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents that it was estimated using that polynomial, lower bound and upper bound. We estimate alternative specifications by, the order of polynomial, the upper bound of the estimation sample, and the lower bound of the estimation sample, and present all possible combinations, none of which are excluded from this figure. The blue dot represents the main specification which we present in our tables in the main text. We thank Peter Eibich, Uri Simonsohn and Hans H. Sievertsen for developing the idea and the code for this figure. Stata code is available at *https://github.com/hhsievertsen/speccurve*.

Figure 9: Sensitivity to alternative bandwidths and polynomials - Fruit Consumption



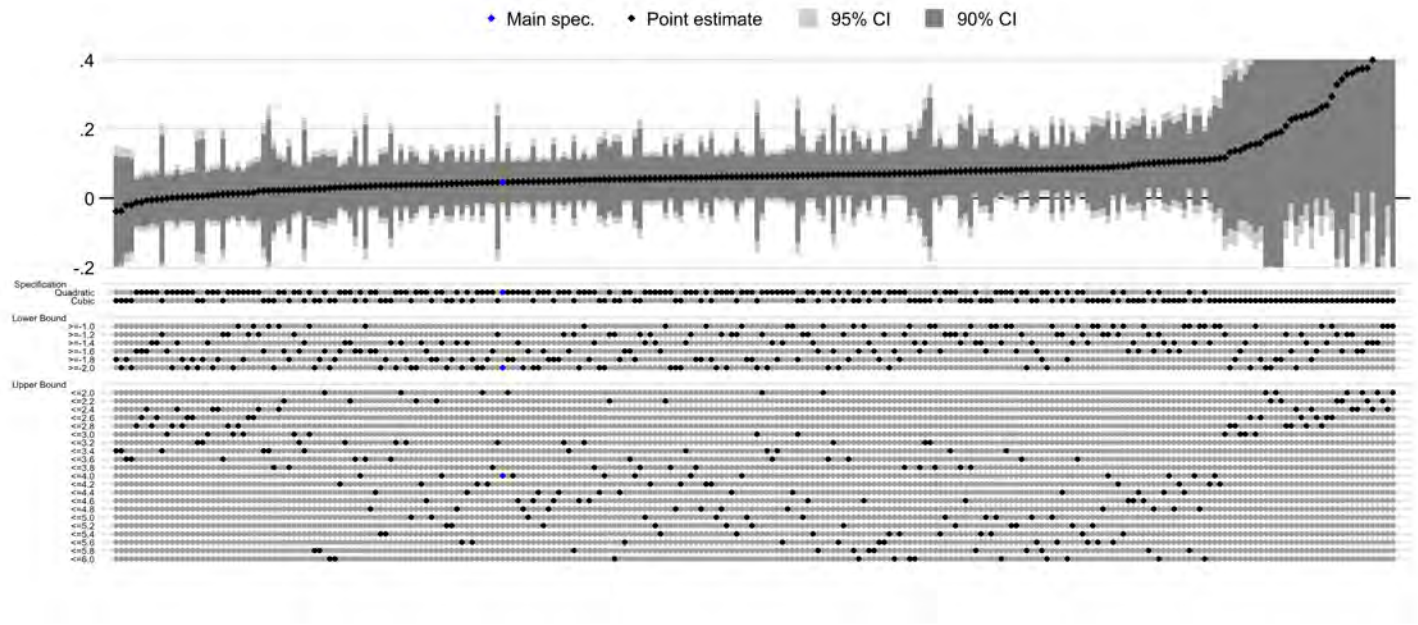
NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents that it was estimated using that polynomial, lower bound and upper bound. We estimate alternative specifications by, the order of polynomial, the upper bound of the estimation sample, and the lower bound of the estimation sample, and present all possible combinations, none of which are excluded from this figure. The blue dot represents the main specification which we present in our tables in the main text. We thank Peter Eibich, Uri Simonsohn and Hans H. Sievertsen for developing the idea and the code for this figure. Stata code is available at *https://github.com/hhsievertsen/speccurve*.

Figure 10: Sensitivity to alternative bandwidths and polynomials - Smoking Behaviour



NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents that it was estimated using that polynomial, lower bound and upper bound. We estimate alternative specifications by, the order of polynomial, the upper bound of the estimation sample, and the lower bound of the estimation sample, and present all possible combinations, none of which are excluded from this figure. The blue dot represents the main specification which we present in our tables in the main text. We thank Peter Eibich, Uri Simonsohn and Hans H. Sievertsen for developing the idea and the code for this figure. Stata code is available at *https://github.com/hhsievertsen/speccurve*.

Figure 11: Sensitivity to alternative bandwidths and polynomials - Alcohol Consumption



NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents that it was estimated using that polynomial, lower bound and upper bound. We estimate alternative specifications by, the order of polynomial, the upper bound of the estimation sample, and the lower bound of the estimation sample, and present all possible combinations, none of which are excluded from this figure. The blue dot represents the main specification which we present in our tables in the main text. We thank Peter Eibich, Uri Simonsohn and Hans H. Sievertsen for developing the idea and the code for this figure. Stata code is available at *https://github.com/hhsievertsen/speccurve*.