

# The direct and spillover effects of diabetes diagnosis on behaviour

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## **Abstract**

Diabetes is a unique condition, in that a positive change in lifestyle and behaviour, is both the first line treatment and the recommended method of preventing the disease. 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 and a decrease in the probability of currently being a smoker both for the diabetic individual and their partner. However, we find limited evidence of other lifestyle 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 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 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). Farrell and Shields (2002), Falba and Sindelar (2008) and Harada et al. (2018) analyse physical activity, and also confirm the strong positive correlation of behaviour between household constituents. Others (Kolonel and Lee; 1981; Barrett-Connor et al.; 1982; Macario and Sorensen; 1998; Bove et al.; 2003; Lyu et al.; 2004) explore correlations between spousal diets, and consistent with studies on concordance of tobacco and alcohol consumption between couples, also find that spouses' diets indeed show a strong association. However, such correlations extend beyond behaviours alone with previous work documenting spousal correlation in mental and physical health<sup>1</sup>. Three theories have been put forward in understanding the causes of these strong empirical correlations, namely assortative matching, shared environment, and joint household decision making (Wilson; 2002; Clark and Etil; 2006; Meyler et al.; 2007; Cutler and Glaeser; 2010; Chiappori et al.; 2012; Davillas and Pudney; 2017).

Assortative matching (also referred to as the marriage market hypothesis) characterizes a behaviour whereby individuals choose to marry those with whom they have similar preferences and characteristics. This 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.

Under the shared environment theory, partners make decisions individually based on their preferences, but are constrained by shared resources and exposed to common shocks. Hence, correlated behaviours are precisely the result of these shared resources and shocks.

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<sup>1</sup>See systematic reviews by Meyler et al. (2007), Di Castelnuovo et al. (2009) and references therein for full discussion.

An alternative explanation relies on more epidemiological terminology where partners who share a common environment, are exposed to the same health risks as a result. Another component of this, potentially 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. Indeed, this conclusion is supported by Khwaja et al. (2006)

Finally, joint household production leans on the theory of New Home Economics, originally established by Lancaster (1966) and Becker (1981), where households jointly produce goods which enter individuals' utility functions. Individuals within the household bargain and as a result produce and consume some shared output, implying a correlation both in behaviour and health. 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 an individual was to have health knowledge that would lead to curative or require preventative changes in behaviour, then such changes would likely have a beneficial spillover onto their partner. However, analysing smoking behaviour, Clark and Etil (2006) found that social learning and household decision making play a minor role, whereas they claim that it is matching in the marriage market that explains the majority of the raw correlations found between partners. Clark and Etil find that once individual random effects are controlled for "smoking behaviours are statistically independent" between partners' in their smoking participation model. Their estimates suggest that all spousal correlation in smoking behaviour is the result of correlations in the individuals effects. They claim that this result implies that spousal correlation is the result of assortative matching on smoking behaviours.

The focus of our analysis is on the impact of diabetes diagnosis on common risk factors of non-communicable diseases: physical activity, diet, alcohol and smoking consumption. The motivation behind analysing these behaviours is twofold. Firstly, these behaviours are well established to determine health, in particular non-communicable diseases (Ezzati

et al.; 2002; Willi et al.; 2007; Prospective Studies Collaboration; 2009; Danaei et al.; 2009; Rehm et al.; 2010; Lim et al.; 2012; Ezzati and Riboli; 2012, 2013). Substantial work has already been done in understanding how these behaviours are determined and can be influenced, and our work adds to that discussion. Secondly, the initial treatment of diabetes specifically aims to change these behaviours to decrease blood sugar and treat the disease (WHO; 2016). In addition, to understand the determinants of these behaviours further, we investigate as to whether those diagnosed with diabetes are compliant to this treatment. We hypothesis that if those diagnosed as diabetic are compliant to treatment, we will observe increases in physical activity, improvements in diet, and a decrease in alcohol and smoking. We also expect to observe significant changes in partners' behaviour in the same direction if transfer of health knowledge and joint household production are significant contributors to health-related behaviour.

In this paper we investigate whether individuals change lifestyle behaviours after a diabetes diagnoses and whether spillover effects on partners do indeed exist. With blood sample data from the Health Survey for England (HSE) dataset we exploit a seemingly arbitrary cut-off of diabetes risk and through a fuzzy regression kink design we 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 a significant effect of diabetes diagnosis on own physical activity and smoking. We also find that partners' of people with diabetes also change their behaviour, by increasing their physical activity and decreased probability of currently being a smoker. 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 and smoking.

We contribute to the literature in a number of ways. Firstly, we contribute to household economics within the context of health by providing further 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 existing literature on diabetes, by causally estimating the behavioural responses of a diabetes diagnosis (Hut and Oster; 2018; Oster; 2018; Kim et al.; 2019). Our results suggest that diabetic individuals comply with some treatments and that this behavioural change is persistent over time. Our results are

of particular importance to health policy makers, as the evidence for substantial positive spillover effects from diabetes diagnoses potentially suggests additional health benefits that are currently ignored in the health care evaluation of policies in this area.

Finally, we also contribute to a new and growing literature on health-related spillover effects. Three papers by Clark and Etil, Fletcher and Marksteiner and Fadlon and Nielsen are closely related to ours. Fadlon and Nielsen (2019), analyse the spillover effects on an extended network of individuals as a result of fatal and nonfatal heart attacks. They find significant and persistent increases in statin consumption of spouses, children and co-workers of individuals who had a nonfatal heart attack, and also analyse the causal mechanisms of these effects, finding evidence in support of both learning new health information, and salience explaining the estimated effect. Our paper complements this work by analysing the effects of a health shock on lifestyle behaviours commonly known to be risk factors of non-communicable diseases.

Fletcher and Marksteiner (2017) use an experimental data to analyse the spillover effects of smoking cessation therapy program and alcoholism treatments. They find that the smoking cessation therapy program resulted in a significant spillover effects in the form of reduced probability of partners currently being a smoker, and alcoholism therapy had a significant negative impact on spouses drinking behaviour. Attributing these effects to matching in the marriage market can be reasonably discounted given their experimental design. However, their results are somewhat at odds with the conclusions made by Clark and Etil, who find that spouses smoking behaviour are statistically independent once individual random effects are included in their model, which suggests that spousal correlation is driven by matching in the marriage market and not the result of social learning.

This paper is organised as follows, first we offer background for the context and premise of the paper, specifically, we discuss diabetes in detail, noting the institutional setting as well as previous literature in this area. Second, we present the theory and literature on spousal correlation and how such theories fit in our setting. Third, we present the data and move onto our identification and estimation strategy. Then, we present our results and validate the identifying assumptions. Finally, we discuss our findings, and place them within a wider context.

## 2 Background

### 2.1 Diabetes

The World Health Organization (WHO) states 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 classified into two types, Type 1 and Type 2. Of the 4.7 million people with diabetes in the UK, approximately 8% have type 1 diabetes, which occurs when insulin production in the body is limited (Diabetes UK; 2019). Although there is limited understanding as to what causes type 1 diabetes, diet or lifestyle are known not to have any impact on the probability of having or developing type 1 diabetes. Type 2 diabetes affects approximately 90% of those with diabetes, and occurs when the body becomes resistant to insulin (Diabetes UK; 2019) and is usually found to be a result of poor diet and lifestyle (Helmrich et al.; 1991; Pan et al.; 1997; Hu et al.; 2001; Liu et al.; 2000).

Glycated haemoglobin (HbA1c) refers to the amount of haemoglobin (i.e. 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 than persistent behaviour.

The World Health Organisation (WHO; 2011) recommends an HbA1c of 6.5% as the cut-off point for diagnosing diabetes, while stating that values below 6.5% do not exclude a diabetes diagnosis. Levels below 6% are considered normal blood sugar levels and therefore low-risk, while levels between 6% and 6.5% are considered at high risk of becoming diabetic, also called pre-diabetes. However, while the link between HbA1c and the probability to develop diabetes is well-established, the choice of specific cut-off for diabetes and pre-diabetes are relatively arbitrary<sup>2</sup>. Nevertheless, although pre-diabetes usually has no

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<sup>2</sup>Yudkin and Montori (2014) state that “glycaemia are continuous, with no inflections to provide obvious

symptoms, NICE<sup>3</sup> recommends 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).

Therefore, individuals who have been found to be at high risk of type 2 diabetes have a significantly higher probability of being diagnosed with diabetes simply as a result of being subject to annual assessment of their HbA1c level. On the other hand, individuals just below the threshold of 6.0%, while having similar probabilities of actually having diabetes as those just above the threshold, have a much lower probability of being diagnosed as a result of them not being annually tested, as per the NICE guidelines.

Our analysis focuses on the impact of a diabetes diagnosis on risk-factors commonly considered to be associated with increased risk of non-communicable diseases. When analysing the impacts of these risk factors on health, the recommendations are clear, namely increasing physical activity and decreasing tobacco and alcohol consumption have beneficial impacts on the risk of developing diabetes and are important in the treatment of the disease (World Health Organization; n.d.a), and this is reasonably well known in the general population. The importance of diet in treating diabetes cannot be understated, and although there are a number of dietary recommendations for individuals with diabetes, due to data limitations, this paper will focus specifically on vegetable and fruit consumption. As with physical activity, alcohol and smoking consumption, the literature and general knowledge of vegetable consumption is clear and consistent: an increase in the proportion of calories consumed from vegetable has significant positive health consequences (World Health Organization; n.d.b) and plays an important role in the treatment of diabetes. However, although health benefits of fruit consumption are well established, fruit consumption for diabetes patients is somewhat more ambiguous and there is misunderstanding in the general population with regards to the optimal consumption of fruit for those with diabetes.

In the medical literature there is an ongoing debate around the consumption of fruit (Forouhi et al.; 2018). On one hand, experts believe that fruit should be encouraged

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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.”

<sup>3</sup>The National Institute for Health and Care Excellence (NICE) is an executive non-departmental public body of the Department of Health which publishes guidelines for clinical practice and the use of healthcare technologies in the National Health Service.

due to their low energy density, and the high content of vitamins, minerals, phytochemicals and dietary fibre. However, others argue that fruit should be limited due to the high carbohydrate content which raises blood sugar, which is problematic in those with diabetes (Forouhi et al.; 2018). The NHS advice states that those with diabetes should “eat a wide range of foods - including fruit”, the advice also states that individuals should “keep sugar, fat and salt to a minimum” (NHS; 2018). This has the potential to cause confusion among the population with diabetes with regards to fruit consumption, due to the high sugar content of fruit. Indeed, there is preventable misunderstanding among the diabetic population with regards to the NHS guidelines, and there are a number of ongoing campaigns to clarify this understanding of the guidelines (Diabetes UK; n.d.). However, there is also confusion in the opposing direction: Speight and Bradley found that, in their sample, 25% of Healthcare professionals and 57% of patients incorrectly stated that fruit “fresh fruit can be eaten freely with little effect on blood glucose levels”. Forouhi et al. state that “consumption of fruits should be guided within the overall dietary pattern of an individual, their taste and other preferences and by their glycaemic control and need for antidiabetic medication, supported by healthcare professionals”. Although we would expect physical activity to increase, and a decrease in tobacco and alcohol consumption due as a result of a diabetes diagnosis, given the misunderstanding of fruit consumption in the UK it is not clear *a priori* what impact a diabetes diagnosis will have on consumption of fruit.

## 2.2 Spousal Correlation

As discussed in Section 1, there is theoretical justification for the presence of a spillover effect from one of the partners being diagnosed with diabetes. Firstly, a diabetes diagnosis transfers health information to the patient both in relation to their own health state (i.e. diagnosis of the disease) and to the disease itself (i.e. causes and consequences of diabetes). Social learning implies that this knowledge would be passed on from patient to partner and having the same information set each partner updates their expectations of future risk and uncertainties. Whether this new information promotes behavioural changes is dependent on idiosyncratic preferences, structural determinants of health and their information set pre-diagnosis Orphanides and Zervos (1995). However, if an individual has a preference for health but they were not, previously, fully informed of the risks of diabetes, we would

expect the newly acquired information to result in a reduction in the probability or level of engaging in risky health behaviours.

For the health information causal channel, the effect on partners' is independent of the observed behaviours of the diabetic individual post-diagnosis. The partner privately re-evaluates and makes new utility maximising decisions based on their new information set that was transferred to them by their partners (Cutler and Glaeser; 2010), 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 moderated by the information set pre-diagnosis. Partners in possession of realistic expectations of the risks of diabetes pre-diagnosis would not substantially change their expectations and would require smaller adjustments to their behaviour as a result of the new information. The claim here being that individuals' preferences remain stable, but the expectation of uncertain events is updated.

Secondly, if a diabetes diagnosis changes the optimal consumption of health-related activities of the diabetic individual, through the updated information channel discussed above, we can also expect it to impact the production and consumption decisions of the other productive household members (i.e. partners) through joint household decision making (Becker; 1973, 1981). For instance, post-diagnosis, physical activity may have higher expected payoff for the diabetic partner. A non-diabetic partner with strong preference for joint time consumption (Jenkins and Osberg; 2004) may choose to participate in physical activity even if they gain relatively less utility from physical activity *per se* compared to other household production activities (Cutler and Glaeser; 2010). However, a positive spillover is not necessarily always the case<sup>4</sup> making the effect of a diabetes diagnosis through this causal channel, while still possible, somewhat more ambiguous.

Finally, assortative matching on diabetes diagnosis would imply that individuals actively seek partners with diabetes (even if they themselves are not diabetic) and would also require diagnosis to happen pre-match. Hence, it is less likely that assortative matching is the driving force behind our findings. What is possible, however, is that individuals match based on behaviours which may impact the cause of diabetes. For instance, individuals

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<sup>4</sup>Presence of a non-compliant to treatment diabetic partner or a stronger dislike for physical activity than preference for joint time consumption for the non-diabetic parter could also explain explain minimal behavioural change for the non-diabetic partner.

sharing a dislike for physical activity or preference for smoking match in the marriage market, these individuals are more likely to be diagnosed with diabetes precisely as a result of the shared preferences. In such case, partners' diabetes status would be endogenous. However, this is not the causal effect we estimate and our identification strategy minimizes the possibility that our estimates are the result of assortative matching.

### 3 Data

This paper uses data from the Health Survey for England (HSE) for years 2003 to 2014. HSE is an annual cross sectional dataset aiming to monitor trends in national health. More than 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. In addition to the individual questionnaire, all respondents are eligible for a nurse visit, in which individuals' physical measurements and a blood sample are taken. Once taken, the blood sample is sent to a specialist laboratory to measure among others, glycated haemoglobin (HbA1c). Although 72% of individuals (across all years) agreed to be contacted for a nurse visit, only 32% of the full sample had blood samples taken for analysis. Of the 52,263 individuals who had blood taken in the survey, 49,568 individuals had valid HbA1c measurements <sup>5</sup>.

Our selection of outcomes analysed (i.e. physical activity, diet, tobacco and alcohol) focus on behaviours that have all been shown to cause diabetes, and have been outlined as a first line treatment for managing and treating diabetes (WHO; 2016). Physical exercise is taken as the response to "any exercise done in the last four weeks". Variables specifically relating to diet in the HSE are limited, however we use two related variables, "whether consumed any vegetables yesterday" and "whether consumed any fruit yesterday", while smoking and drinking behaviour are captured by "whether currently a smoker" and "whether currently a drinker" excluding those that are never drinkers, respectively.

Table 1 provides descriptive statistics of the data used in the analysis. The first column

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<sup>5</sup>A change in calibration of the equipment used for analysis HbA1c was made in 19th of September 2013, which resulted in a slight change in result for equivalent blood samples. Throughout the analysis we use "valid HbA1c result", as recommend in the Health Survey for England documentation, which adjusts the results post-2013 to be equivalent to pre-2013 results for the same blood samples.

provides means and standard deviations of a number of observable characteristics and stated health-related behaviours for the entire HSE sample, including those that did not have blood measurements taken. In the following columns we give summary statistics of the sub-sample of individuals who did have blood taken for analysis and whose info is used in our estimations. We break these descriptive statistics into those with measured HbA1c levels below and above the 6.0% cut-off. The right-most columns in the table are descriptive statistics of the sub-sample of individuals who have HbA1c results in the data and additionally have partners living in their household with HbA1c results in the data. These are also separately broken down into HbA1c levels below and above 6.0%.

The Blood and Partners sample is substantially smaller than the Blood Sample. Not all individuals included in the blood sample have partners, and not all partners that responded had valid HbA1c measurements, therefore we would expect and indeed observe fewer observations for this sample. Variables marked with a † in Table 1, denote variables that they 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 not surveyed in all years but only in 2003, 2004, 2006, 2008, 2012. This is also true for household size and equivalized income, but for different years.

It is worth noting that in our sample, individuals who have ever been diagnosed as diabetic were, on average, diagnosed 9.86 years ago (standard deviation of 10.4). Therefore our results are not interpreted as the immediate effect of a diabetes diagnosis, unlike previous studies that observe behavioural responses in a short-time frame post-diagnosis (Hut and Oster; 2018; Oster; 2018; Kim et al.; 2019). These studies use a panel data structure and observe the pre-diagnosis period, and a short time frame post diagnosis, up to four years in Kim et al.’s setting. Because on average we observe individuals who were diagnosed in the distant past, our LATE is more akin to the long-term effect of a diabetes diagnosis. This additionally allows us to investigate the temporal effects over a longer time-frame than previous studies, and indeed we do analyse these temporal effects.

## 4 Identification Strategy

The 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:

$$y_i = \theta_0 + \theta_1 EverD_i + \theta_2 EverD_j + e_i \quad (1)$$

where  $y_i$  denotes the health related lifestyle behaviour of interest and  $EverD_i$  denotes whether individual  $i$  has ever been diagnosed with diabetes, and  $EverD_j$  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 biased estimates of both  $\theta_1$  and  $\theta_2$ .

The first and possibly most salient source of bias is simultaneity. It is possible that, on average, when observed, individual with diabetes may behave in a way which is more damaging to their health than those without diabetes. This ignores, however, 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.; 1991; Pan et al.; 1997; Hu et al.; 2001; Liu et al.; 2000).

In addition, if we were to estimate equation (1) through least squares,  $\theta_2$  would also be biased. Past literature has emphasised the importance of matching in marriage markets with Dupuy and Galichon (2014) observing considerable matching in personality traits and attitude towards risk. This suggests that individuals selectively marry along similar traits and therefore if a naive estimation ignores this channel the estimate of the spillover effect will be biased.

### 4.1 Regression Kink Design

To identify the causal effect of diabetes diagnosis on health-related behaviours, we utilise a regression kink design (RKD), 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 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 slope of the probability of being diagnosed. As discussed in Section 2.1, NHS recommends that individuals with 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 warrant a blood test. It is worth emphasising that such kink in the probability of a diabetes diagnosis is not supported in the medical sense as Yudkin and Montori (2014) explicitly explain that an inflection point of diabetes risk does not indeed exist, meaning that the assignment of diabetes risk is arbitrary.

Dong (2011) provide the theoretical framework for identification in our setting, whereby the RKD identifies the causal effect of a binary treatment when there is not a discontinuity in the probability of treatment but rather a kink. When the policy rule is implemented with some error (i.e. the kink is not deterministic) a fuzzy RKD design can be implemented (Card et al.; 2015). Causal effect of diabetes diagnosis on health-related outcomes combines the RKD with a two-stage least squares (2SLS) specification. The first stage identifies the effect of the kink on the probability of treatment:

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

where  $EverD_i$  is a binary variable taking the 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%.  $D_i = \mathbf{1}(x_i \geq k)$ , 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,  $\nu_p^-$  and  $\nu_p^+$  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:

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

where  $y_i$  denotes the health related behavioural outcome of interest.  $\widehat{EverD}_i$  is the predicted probability, from the first stage, of ever being diagnosed with diabetes, while again the terms in the square brackets denote the polynomial function below and above the kink point. In line with Gelman and Imbens (2019), the main analysis uses quadratic polynomial specifications to estimate effects, while linear specifications are also reported in sensitivity tests. Under the assumptions outlined by Dong (2011) and Card et al. (2015) the coefficient  $\beta_1$  can be interpreted as the unbiased local average treatment effect (LATE) of ever having been diagnosed with diabetes.

Identification comes from the exogenous variation that the kink provides in the probability of diabetes diagnosis. This relies on the assumption that those just to the left of the kink are almost identical to those just to the right of the kink and it was random variation that resulted in them falling either side of the kink-point. As with regression discontinuity designs (RDD) there is a bias-variance trade-off to be made when selecting the estimation sample. Larger samples are more likely to bias estimates because other factors apart from the kink are likely to influence diabetes diagnosis, whereas smaller samples will not have sufficient power to reject a false null hypothesis. In our data we observe HbA1c measurements to one decimal place, and therefore we have data which looks more discrete in nature around the cut-off. For this reason, we limit our polynomial specification to a quadratic, to ensure we are not over-fitting to our data. In addition, we choose a bandwidth that is relatively large so that we have sufficient power to reject a false null hypothesis. However, to ensure that our results are robust, we transparently present a number of alternative specifications and bandwidths in sensitivity tests.

To examine sensitivity of results to bandwidth choice, we consider a large number of alternative bandwidths in sensitivity tests. Given the few observations of individuals who 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. We use asymmetric bandwidths because we observe fewer individuals on the right-hand side of the cut-off, than on the left-hand side, and without extending the right-hand side we would not have sufficient power. In our main set of results, we use a bandwidth of 5.0% on the right hand side of the cut-off and 2.0% on the left hand side (i.e. HbA1c values of 4% to 11% are included in the estimation sample).

To improve precision and reduce the bias of our estimates (Imbens and Lemieux; 2008) we

additionally include the following covariates in our estimating equation: a gender dummy, a continuous age variable, we also include a binary indicator of whether individual has degree level education, self-assessed general health (1-5 point scale), and a binary indicator denoting whether a partner lives in the household.

## 4.2 Partner's Diabetes Status

To handle the endogeneity of partners' diabetes diagnosis own behaviour, we adapt the previous set up and instead look at partners' diagnosis. This estimates the effect of partner's diagnosis on own behaviour by using the partners' kink as an instrument for partners' probability of being diagnosed with diabetes. The first stage of the 2SLS is specified as

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

where  $j$  denotes the partner,  $EverD_j$  is whether partner has ever been diagnosed with diabetes, and  $x_j$  denotes the partners HbA1c level. The second stage defining the causal relationship is

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

As discussed previously, causal identification requires reasonable bandwidths either side of the kink-point. Using the same bandwidths for partners' as for own, the estimation sample are those who have partners, and those partners have HbA1c levels within the bandwidths.

We also include the same set of covariates as for the effect of own diagnosis, excluding whether partner lives in the household.

## 5 Main estimation results

### 5.1 Effect of own diagnosis

Panel (a) in Table 2 presents estimates of the effect of own diabetes diagnosis on own behaviour. The relevance of the kink as an instrument for ever being diagnosed with diabetes is given in the first stage coefficients with results suggesting a highly statistically positive significant effect of the kink on probability of being diagnosed with diabetes. The second row of Table 2 gives the coefficient  $\beta_1$  from equation (3). We find that being diagnosed with diabetes has a significantly increases the probability of having some physical activity in the last four weeks and significantly reduces the probability of currently being a smoker. We find no evidence to suggest an impact on consumption of fruit or vegetable, and there is no evidence to suggest that diabetes diagnosis changes drinking behaviour.

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#### 5.1.1 Sensitivity to alternative bandwidths and polynomials

To assess the sensitivity of results to alternative specifications and bandwidths we explore a series of robustness graphs in Figures 5 to 9. Graphs show the point estimate,  $\beta_1$ , and the corresponding 90% and 95% confidence interval, from equation 3, estimated using 2SLS for each  $y_i$  outcome of the main analysis. Specifications vary by polynomial order (i.e. linear or quadratic) and the selected estimation sample, which we vary by selecting different bandwidths for above and below the cutoff (bounds of the estimation sample). The upper bound describes the relative bandwidth above the kink point with the lower bound being the corresponding bandwidth below the kink point, (i.e. a lower bound of 2 corresponds to a HbA1c value of 4%, a bandwidth of 2% below the kink-point of 6%. An upper bound of 3 corresponds to a HbA1c value of 9%, a bandwidth of 3% above the kink-point).

Inspecting Figure 5, for physical activity, point estimates across all specifications are above zero and in almost all cases confidence intervals do not include zero. Overall, results

<sup>6</sup>In addition to the estimates presented, Figure A1 in the Appendix shows the reduced form quadratic prediction graphically imposed over the mean outcomes per bin for HbAqc levels, where the reduced form estimates are from  $y_i = \chi_0 + \chi_1(x_i - k)D_i + \left[ \sum_{p=1}^{p^*} \psi_p^-(x_i - k)^p \right] + \left[ \sum_{p=2}^{p^*} \psi_p^+(x_i - k)^p D_i \right] + \mu_i$ . The graphs show a similar results to the 2SLS estimated with physical activity having the clearest slope change around the kink point, whereas fruit, smoking and alcohol consumption show a far more subtle changes in slope.

seem robust with physical activity estimates not being overly sensitive to specification chosen.

Vegetable consumption and fruit consumption estimates in Figures 6 and 7, respectively, follow a similar pattern to one another. For quadratic specifications the estimates are both close to zero in magnitude, and have a relatively tight confidence interval which include zero in almost every case. However, for both fruit and vegetable the linear specifications seem to have a positive and significant effect. We are cautious in claiming that an effect exists for each of these, given that our main specification, using a quadratic polynomial supports a null effect, and that significance of these estimates are clearly specification dependent. We will therefore conservatively state that we find no evidence of an effect on vegetable or fruit consumption, rather than claiming that there is no effect.

Findings for smoking behaviour, Figure 8, are similar to those of physical activity with point estimates varying little across specifications and the all specifications featuring tight confidence intervals excluding zero. Estimates from a quadratic specification appear to be very robust and all sit within a very small interval around -0.3 and also have tight confidence intervals.

Finally, alternative specifications for the effect of diabetes diagnosis on alcohol consumption are presented in Figure 9. Almost all specifications have confidence intervals which include zero and are also tightly bounded around zero, especially our preferred specifications with a quadratic polynomial.

## 5.2 Spillover effect

The spillover estimates as a result of partners' diabetes diagnosis, i.e. parameter  $\delta_1$  in eq. 5, are presented in Panel (b) of Table 2. In this case, partner's kink is used as an instrument for partner diabetes diagnosis and its relevance is given in the first stage estimates implying very good identification properties. 2SLS estimates are presented in the second row of Panel (b), with findings suggesting very similar patterns to those of own diabetes diagnosis.<sup>7</sup> Specifically, we find significant positive effects for exercising in the

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<sup>7</sup>Reduced form RKD estimates from  $y_i = \chi_0 + \chi_1(x_i - k)D_i + \left[ \sum_{p=1}^{p^*} \psi_p^- (x_i - k)^p \right] + \left[ \sum_{p=2}^{p^*} \psi_p^+ (x_i - k)^p D_i \right] + \mu_i$  are plotted in Table A2 in the Appendix. Physical activity once again exhibits the most prominent slope change, with little evidence of a slope change elsewhere.

past four weeks and significant negative effects for currently being smoker, in the former the magnitude is similar to that of the effect of own diagnosis and about half as large for the latter. There is some suggestive evidence of a change in fruit consumption, however these results are not robust when we look at the sensitivity to alternative specifications, and therefore we will not claim any such effect on fruit consumption. Little evidence of an effect is present for the remaining two outcomes.

### 5.2.1 Sensitivity to alternative bandwidths and polynomials

We additionally assess the sensitivity of our spillover estimates in figures 10 to 14. Broadly these figures follow a similar pattern to those for the effect of own diabetes diagnosis. One significant point of difference is that the confidence intervals for the spillover effect are substantially larger than those for the effect on own. This is to be expected given that the estimation sample for the spillover effect is smaller than the estimation sample for own and therefore power is more of a problem. Indeed, we find that large confidence intervals are especially a problem in specifications with narrow bandwidths or higher order polynomials, and therefore power is a problem in these cases. Nevertheless, the pattern for figures 10 to 14 follow a similar pattern to the effect on own, and indeed the results for physical activity, and smoking do not appear to be sensitive to specification and the majority of specifications are significantly different from zero.

## 5.3 Validity of identifying assumptions

For RKD estimates to be considered the LATE of diabetes diagnosis two observable implications, as outlined in Card et al. (2015), must hold. The first relates to the smooth density of the assignment variable and empirically tests the assumption of no deterministic sorting. The second relates to the lack of discontinuity or kinks in the pre-determined covariates and tests the assumption that the marginal effect of the assignment variable on the outcome is smooth.

### 5.3.1 Smooth density of the assignment variable

The smooth density of the assignment variable implies no discontinuity in its density (an assumption similar to that required for RDD settings) but additionally for the RKD case, requires the lack of a kink in its density. While one's position in the distribution can be coarsely influenced by changes in diet and other health behaviours, the value of HbA1c is not able to be manipulated precisely as would be required for it to exhibit a kink or discontinuity at the threshold given Yudkin and Montori (2014). However, this observable implication of the RKD assumptions is testable, and therefore we do so to ensure that this assumption holds in our context.

McCrary (2008) provides a test for deterministic sorting for continuous assignment variables but ignores the stronger version of the assumption requiring no kink. There are two important considerations for testing this assumption in our setting. The first issue that we face is that the McCrary test is designed with continuous assignment variables in mind, however in our data HbA1c levels are rounded to the nearest 0.1. The discrete nature of our assignment variable can lead to both size and power issues if we were to use the McCrary test. Therefore, instead we use the Frandsen (2017) test for manipulation when the assignment variable is discrete.

The second consideration we need to make is that the test proposed by both McCrary (2008) and Frandsen (2017) do not claim to explicitly test the stronger assumption of no jump *or* kink in the density of the assignment variable, required for the RKD. However, the Frandsen (2017) test allows the user to choose a degree of departure from linearity which is tolerated, by choosing the value of the bound coefficient  $k$ . A choice of  $k = 0$  specifies that the distribution must be linear for no manipulation to be concluded. If one were to set the bound coefficient to zero the null hypothesis is of linearity across the threshold, and the alternative hypothesis is that the distribution is non-linear around the threshold. Non-linearity around the threshold would either imply a jump or a kink, which would mean that our assumption of smooth density does not hold. As a result, we set the bound coefficient to equal zero and report the p-value of this test.

Figure 2 presents graphically the density of the assignment variable by HbA1c. 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 p-value from the Frandsen (2017) test. The tests is unable to reject the null of linearity across the threshold suggesting that the first identifying principle for our RKD holds. Such findings are not particularly surprising, given that by nature HbA1c is extremely difficult to exactly manipulate and influence around the threshold.

### 5.3.2 Predetermined Variables

As stated by Card et al. (2015), this assumption is similar to the “test of random assignment” commonly required in randomized control trials. As above, this observable implication is more restrictive than the equivalent RDD implication as in addition to any discontinuity also requires the lack of any kink in the pre-determined variables. We assess whether the observable assumption holds in our setting by visual inspection and graphically present the mean values per bin by the assignment variable for a number of predetermined variables.

Card et al. (2015) make clear this observable implication relies on the existence of a set of variables which, by definition, are not determined by the treatment. As such, we are somewhat limited in terms of the variables available at our disposal for testing. HSE is a cross-sectional study and most survey questions refer to specific points in time without eliciting information about the past, and in the cases where they do, it is unknown if such information relates to periods prior or post treatment. However, we examine a number of relevant variables, namely age, gender, self-reported health, whether individual has degree level education, whether the individual has any educational qualifications <sup>8</sup>, whether a partner lives in the household, whether ever a smoker and whether ever a drinker.

Graphical results are given in Figure 3. There is no evidence of clear discontinuities or kinks at the kink point for any of the variables presented here, validating our second necessary assumptions and suggesting that interpretation of the results of the RKD as LATEs is valid.

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<sup>8</sup> Any qualification corresponds to a long list of education qualifications surveyed in the HSE, which include (but not limited to) degree education, high school and professional qualifications (i.e. teaching, nursing, vocational).

## 6 Robustness check for simultaneous Own and Partner's diabetes status

Having evidence for the consistency of the RKD estimations in our setting we further pursue sensitivity issue and examine the robustness of the effect of own and partner diabetes diagnoses on own behaviour when both effects are simultaneously identified and estimated. In this specification own and partners' kinks are used as instruments for own and partners' probability of being diagnosed diabetic. Two separate first stage estimations are required, one equation for own,  $z = i$ , and one for partner,  $z = j$ .

$$EverD_z = \eta_0 + \eta_1(x_i - k)D_i + \eta_2(x_j - k)D_j + \left[ \sum_{p=1}^{p^*} \chi_p^-(x_i - k)^p \right] + \left[ \sum_{p=2}^{p^*} \chi_p^+(x_i - k)^p D_i \right] \\ + \left[ \sum_{p=1}^{p^*} \zeta_p^-(x_j - k)^p \right] + \left[ \sum_{p=2}^{p^*} \zeta_p^+(x_j - k)^p D_j \right] + q_z \quad (6)$$

Obtaining predicted probabilities for both equations, the second stage is correspondingly defined as

$$y_i = \kappa_0 + \kappa_1 \widehat{EverD}_i + \kappa_2 \widehat{EverD}_j + \left[ \sum_{p=1}^{p^*} \pi_p^-(x_i - k)^p \right] + \left[ \sum_{p=2}^{p^*} \pi_p^+(x_i - k)^p D_i \right] \\ + \left[ \sum_{p=1}^{p^*} \phi_p^-(x_j - k)^p \right] + \left[ \sum_{p=2}^{p^*} \phi_p^+(x_j - k)^p D_j \right] + r_i \quad (7)$$

Results are given in Panel (c) of Table 2. Own diabetic diagnosis, increases the probability of exercise, increases the probability of fruit consumption and decreases the probability of currently smoking. Partner's diagnosis also increases the probability of exercise, however the effect for smoking behaviour is lost in these specifications.

Overall, findings confirm the main analysis albeit for some specifications significance is reduced. This is likely the result of smaller sample sizes and reduced estimation power. We note that given the set-up, the relevant estimation sample only includes those who have HbA1c levels within the bandwidths, have partners, and those partners also have HbA1c

levels within the bandwidths. Further, in support of power issues as the reason behind lower significance levels, we note that comparisons of the corresponding 2SLS estimates between Panels (a) and (c) and Panels (a) and (b) of Table 2 suggest that for the vast majority of models, coefficients magnitudes are comparable but effects in Panel (c) are estimated with less precision and hence higher standard errors.

## 7 Observed heterogeneity in RKD estimates

Following the main analysis and identification properties, we assess whether effects of a diabetes diagnosis are heterogeneous across observables both for own and partners' diagnoses. We explore three sources of heterogeneity. First, we test whether those that live with a spouse behave differently to those that do not. Second, in an attempt to estimate whether the impact of diagnosis on behavioural change varies over time we analyse whether those being diagnosed for longer behave differently to those recently diagnosed. In the absence of panel data, differential impact by time since diagnosis approximates long-term effects or recidivism to pre-diagnosis behaviours. Finally, we estimate whether there are observable heterogeneities by education.

For estimation, we derive the Heterogeneous Local Average Treatment Effect (HLATE) in a similar vein to Becker et al. (2013), by replacing the LATE of  $EverD_i$  in equation 3 and manipulating it to allow for heterogeneous effects along the variable  $z_i$ . This is implemented by replacing coefficients with an interaction, the general case being  $\gamma = \hat{\gamma} + \tilde{\gamma}z_i$  where  $z_i$  denotes the trait across which heterogeneity is examined. The first stage equation is re-written as:

$$EverD_i = \mu_0 + \mu_1 z_i + \mu_2(x_i - k)D_i + \mu_3(x_i - k)D_i z_i + \sum_{p=1}^{p^*} [\nu_p^-(x_i - k)^p + \nu_p^-(x_i - k)^p z_i] + \sum_{p=2}^{p^*} [\nu_p^+(x_i - k)^p D_i + \nu_p^+(x_i - k)^p D_i z_i] + w \quad (8)$$

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

$$y_i = \psi_0 + \psi_1 z_i + \psi_2 \widehat{EverD}_i + \psi_3 \widehat{EverD}_i z_i + \sum_{p=1}^{p^*} [\nu_p^- (x_i - k)^p + \nu_p^- (x_i - k)^p z_i] \\ + \sum_{p=2}^{p^*} [\nu_p^+ (x_i - k)^p D_i + \nu_p^+ (x_i - k)^p D_i z_i] + m_i \quad (9)$$

The parameters of interest here are  $\psi_2$  and  $\psi_3$ , where the estimate of  $\psi_3$  describes the heterogeneity in the treatment effect over the trait under inspection  $z_i$ . The notation is the same as above, where  $y_i$  are the same behaviours of individual  $i$  analysed above,  $EverD_i$  is a binary variable taking the 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,  $\nu_p^-$  and  $\nu_p^+$  are the estimates of the polynomial function below and above the kink point, respectively.

However, the inclusion of an additional term to estimate,  $\psi_3$ , which is dependent on the endogenous variable  $EverD_i$  requires an additional instrument for the 2SLS estimates to be correctly identified. We therefore, additionally, estimate an auxiliary first stage regression:

$$EverD_i z_i = \omega_0 + \omega_1 z_i + \omega_2 (x_i - k) D_i + \omega_3 (x_i - k) D_i z_i + \sum_{p=1}^{p^*} [\sigma_p^- (x_i - k)^p + \sigma_p^- (x_i - k)^p z_i] \\ + \sum_{p=2}^{p^*} [\sigma_p^+ (x_i - k)^p D_i + \sigma_p^+ (x_i - k)^p D_i z_i] + z_i \quad (10)$$

The above framework refers to own behaviour in response to own diabetes diagnosis. We extend this approach to partners and estimate whether there is heterogeneity in own behaviour as a result of partner diagnosis and heterogeneity according to time since partner's diagnosis and their educational level.

Our HLATE estimation strategy is very similar to that of Becker et al. (2013) which we

follow closely with the key difference being that ours is implemented within an RKD instead of an RDD setting. For HLATE estimation we require that two additional assumptions hold in addition to those discussed in the previous section (Becker et al.; 2013). The first is that there is continuity of the interaction variables at the threshold vector. In our setting we require a stronger version of this, namely 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). Figure 3 shows, amongst other variables, whether individual has degree level education. As discussed previously there is little evidence of either a jump or kink at the threshold HbA1c level of 6%.

Time since diagnosis cannot be handled in a similar fashion as is not observed (i.e. it does not exist) for those that have never been diagnosed. To make HLATE effects estimation possible, for those with missing observations, we assign placebo time-since-diagnosis values by randomly drawing values, with replacement, from observed observations (individuals whom have a time-since diagnosis value) (Kleven et al.; 2019). For the analysis, we demean the variable so that  $\psi_2$  represents the effect for the average time since diagnosis.

The second required assumption is the random assignment of the interaction variable conditional on covariates. 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, which we also include in our main estimates, namely a gender dummy, a continuous age variable, self-assessed general health, 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.

## 7.1 Partner in Household

Table 3 presents the effect of own diabetes diagnosis by whether an individual lives with a partner or not. Having a partner in the household on its own, also increases the probability of consuming vegetables reduces the probability of smoking, while also increases the probability of drinking. However, there is little heterogeneity on the effect of own diabetes diagnosis on own behaviour. Therefore we infer that the effect of a diabetes diagnosis on own is not influenced by whether the diagnosed individual has a partner living in the same

household.

## 7.2 Time Since Diagnosis

Heterogeneity estimates across time-since-diagnosis are given in Table 4 with Panel (a) showing the effect of own diabetes and Panel (b) the effect of partner’s diabetes diagnosis. For both own and partner’s diabetes main effects we find that diagnosis increases exercise and reduces smoking with no variation in any of the estimates by time since diagnosis, apart from for exercise for own, however the effect size is very small in magnitude. Such finding, supports a hypothesis of habit formation, whereby individuals make positive lifestyle changes that they consistently maintain going forward. This is somewhat contrary to Kim et al. (2019) who find that for their specific outcomes measures (i.e. outpatient visits, medicated days, basic exercise) there were no significant long-run effects.

It is also reassuring to note that time since diagnosis, as a main effect, is insignificant in almost all models, 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.

## 7.3 Education

Finally, heterogeneity in terms of educational attainment is presented in Table 5. On average, those with degree level education tend to make better lifestyle choices than those without degree level education. Those with degree level education are more likely to exercise, eat vegetables and fruit, and are less likely to smoke. However, they are also more likely to currently be a drinker, which is somewhat at odds with what we would expect. In terms of the interaction between diabetes diagnosis and education, we find limited evidence of a heterogeneous effect by education for most of our outcomes, the only exception being fruit consumption.

We find that degree educated individuals decrease their fruit consumption in response to a diagnosis, whereas those without a degree increase their consumption of fruit. At first glance this may be somewhat perplexing, however as outlined in the introduction, the guidelines state that fruit should not be eaten freely, and although its consumption is

encouraged, the amount should be limited. Considering that degree educated eat more fruit than those without degree level education, a higher proportion of them are at the upper bound, or exceed the recommended fruit consumption prior to a diagnosis, and therefore the diagnosis induces them to reduce their fruit consumption. However, there is potential concern in that we find no evidence to suggest that these individuals offset their decrease in fruit consumption with an increase in vegetable consumption, which would be medically recommended.

## 8 Conclusion

Diabetes is a unique condition, in that a positive change in lifestyle and behaviour, is both the first line treatment and the recommended method of preventing the disease. By jointly partaking in diabetes treatment, partners of people with diabetes could substantially benefit from their partners' diabetes diagnosis. In this paper we estimate the causal effect of own or partner's diabetes diagnosis on own lifestyle behaviours, namely exercising, eating habits, smoking and drinking. Exploiting national guidelines around the levels of sugar in the blood and their recommendation for annual testing after a specific threshold, a fuzzy kink regression design is implemented using data on blood samples and individual behaviours from the Health Survey for England (HSE) dataset.

Findings show that individuals who have ever been diagnosed with diabetes do significantly increase their physical activity and reduce probability of currently being a smoker, suggesting compliance with first line treatment guidelines for diabetes. We additionally find evidence of persistence over time 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. Most importantly, we uncover substantial spillover effects from diabetes diagnosis in the form of an increase in physical activity and reduction in the probability of smoking for the partners of those diagnosed with diabetes. Through our identification strategy such effects are likely to be is a combination of joint household decision making and health-related information transfer between partners.

Comparing our results of the direct effect to previous studies, our estimated impacts on diet differ to those of Hut and Oster (2018) and Oster (2018), and are somewhat at odds with the impact on physical activity estimated by Kim et al. (2019). Hut and Oster estimated

there to be significant and positive changes in diet post-diagnosis, and found that increased fruit purchases was the fourth largest contributor to these dietary changes. However, their results somewhat suggest that the improvements in diet begin to fade over time. They also find that single-person households do not significantly change their diet as a result of a diabetes diagnosis. Finally, they find that individuals with college education or higher improve their diet marginally more than the average as a result of a diagnosis. The findings of Oster (2018) follow a similar pattern to the results of Hut and Oster, in that calories purchased of fruit and vegetables both increase in the month post-diagnosis, however once again, the effect appears to decrease over time, and between months 2-12 post-diagnosis there is no significant increase in calories purchased of fruit and vegetables. Although our results might immediately seem somewhat at odds with the findings of Hut and Oster (2018) and Oster (2018), we once again note the difference in time since diagnosis that we study compared to their work. We believe, that our findings do seem to follow the temporal pattern of these studies. Given that the average time since diagnosis in our sample is over 10 years, and that Hut and Oster and Oster both find decreasing effects over time, it might be expected that the effects decrease to zero in the long-run. However, when we analyse the temporal effects for diet, we again find no evidence that there are changes over time. Kim et al. finds there to be no significant increase in physical activity as a result of a diabetes diagnosis in either the short-run (1 or 2 years) or the long-run (3 or 4 years), whereas we find there to be both a significant and persistent change in physical activity as a result of a diabetes diagnosis.

When we compare our results to those studies that estimate spillovers onto partners there are no papers we are able to compare to directly, however our broader conclusions do concur with previous studies, with the exception of Clark and Etil (2006). Clark and Etil found that the correlation between partners smoking behaviour was driven mainly by matching in the marriage market, whereas our findings, as well as those of Fletcher and Marksteiner (2017), find there to be significant spillover effects in terms of smoking behaviour. In terms of alcohol consumption, it is somewhat harder to compare our results with those of Fletcher and Marksteiner, given that they investigate the direct and spillover effects of alcoholism treatment, rather than a diabetes diagnosis. Finally, although we are unable to directly compare our results to Fadlon and Nielsen (2019), our broad conclusions are in agreement with theirs, in that both their study and ours find significant health-related behavioural spillovers.

From a public health perspective, confirmation of long-term compliance of diabetics to first line treatments and necessary lifestyle changes is reassuring, at least in relation to physical activity and smoking. However, further work is required to induce behavioural changes in terms of diet and alcohol consumption in diabetes patients. Our findings should also be of interest to policy makers seeking to evaluate the benefits of diabetes interventions as we offer evidence of substantial additional benefits, also in terms of increased physical activity and reduced smoking of partners', from a diabetes diagnosis, beyond what might be usually considered in economic evaluations. However, our results also potentially suggest that more work should be done in promoting healthy diets, and reduced alcohol consumption in individuals diagnosed with diabetes and their partners as there is little evidence of behavioural changes therein.

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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	
<b>Observable Characteristics</b>							
Age†	50.32 (18.67)	52.14 (17.57)	49.69 (17.24)	64.34 (13.59)	52.38 (15.21)	50.46 (14.89)	62.51 (12.69)
Males	0.47 (0.50)	0.48 (0.50)	0.47 (0.50)	0.51 (0.50)	0.50 (0.50)	0.50 (0.50)	0.58 (0.49)
Any Qualifications	0.73 (0.44)	0.75 (0.43)	0.79 (0.41)	0.57 (0.49)	0.77 (0.38)	0.80 (0.40)	0.62 (0.49)
Degree level education	0.20 (0.40)	0.21 (0.41)	0.23 (0.42)	0.13 (0.33)	0.23 (0.42)	0.25 (0.43)	0.14 (0.35)
Partner living in household	0.64 (0.48)	0.67 (0.47)	0.68 (0.42)	0.64 (0.48)	— —	— —	— —
Household Size†	2.66 (1.39)	2.56 (1.31)	2.65 (1.33)	2.15 (1.14)	2.88 (1.17)	2.95 (1.18)	2.57 (1.05)
Employed	0.59 (0.49)	0.60 (0.49)	0.65 (0.48)	0.36 (0.48)	0.66 (0.47)	0.70 (0.46)	0.43 (0.49)
Equivalised Income†	29,920.77 (27,403.78)	31,176.52 (27,695.47)	32,376.71 (28,047.43)	25,478.14 (25,095.71)	33,001.36 (26,122.98)	34,184.01 (26,404.12)	26,439.02 (23,447.45)
Self-assessed general health (1 = Very Good, 5 = Very Poor)	2.06 (0.96)	1.99 (0.91)	1.90 (0.87)	2.44 (1.00)	1.93 (0.88)	1.85 (0.89)	2.37 (0.98)
Glycated Hemoglobin (HbA1c)	— ,	5.61 (0.76)	5.38 (0.33)	6.74 (1.18)	5.60 (0.74)	5.38 (0.32)	6.73 (1.16)
Number of Observations	100,693	46,528	38,748	7,780	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 RKD estimates of change in own behaviour as a result own, partner's, own and partner's diabetes diagnosis

	Exercise	Vegetable Consumption	Fruit Consumption	Currently a Smoker	Currently a drinker
<b>(a)</b>					
1 <sup>st</sup> Stage	0.593*** (0.0248)	0.647*** (0.0178)	0.646*** (0.0178)	0.642*** (0.0162)	0.648*** (0.0171)
Own Diabetes	0.237*** (0.0710)	0.0677 (0.0531)	0.0590 (0.0505)	-0.255*** (0.0369)	0.0351 (0.0266)
Obs.	20692	35897	35918	41080	38181
<b>(b)</b>					
1 <sup>st</sup> Stage	0.592*** (0.0360)	0.639*** (0.0256)	0.639*** (0.0256)	0.631*** (0.0234)	0.637*** (0.0241)
Partner's Diabetes	0.214** (0.101)	-0.0442 (0.0752)	-0.126* (0.0710)	-0.108** (0.0480)	0.0284 (0.0357)
Obs.	10604	18186	18187	20792	19255
<b>(c)</b>					
Own 1 <sup>st</sup> Stage	0.523*** (0.0402)	0.585*** (0.0287)	0.585*** (0.0287)	0.577*** (0.0259)	0.580*** (0.0273)
Own Diabetes	0.240** (0.116)	0.0545 (0.0832)	0.234*** (0.0805)	-0.253*** (0.0550)	0.0867* (0.0456)
Partner's 1 <sup>st</sup> Stage	0.523*** (0.0402)	0.585*** (0.0287)	0.585*** (0.0287)	0.578*** (0.0259)	0.590*** (0.0269)
Partner's Diabetes	0.244** (0.119)	0.00196 (0.0829)	-0.113 (0.0798)	-0.0560 (0.0534)	0.0201 (0.0387)
Obs.	8064	13635	13635	15600	14556

Notes: 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 for panels (a) and (b), and bounds of 6.0 on the right hand tail, and 3.0 on the left hand side for panel (c). 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.  
\*\*\* 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: Heterogeneous fuzzy RKD estimates of change in own behaviour as a result own diabetes diagnosis by whether individual has a partner

	Exercise	Vegetable Consumption	Fruit Consumption	Currently a Smoker	Currently a drinker
Own Diabetes	0.184 (0.121)	0.132 (0.0881)	-0.00113 (0.0849)	-0.238*** (0.0664)	0.0614 (0.0480)
Partner in HH	-0.00179 (0.0248)	0.0469*** (0.0174)	0.0224 (0.0165)	-0.0690*** (0.0123)	0.0321*** (0.00858)
Own Diabetes x Partner in HH	0.00363 (0.149)	-0.0874 (0.110)	0.114 (0.106)	-0.0531 (0.0795)	-0.0536 (0.0573)
Obs.	20680	35877	35899	41054	38156

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 4: Heterogeneous fuzzy RKD estimates of change in own behaviour as a result own and partner's diabetes diagnosis by time-since-diagnosis

	Exercise	Vegetable Consumption	Fruit Consumption	Currently a Smoker	Currently a drinker
<b>(a)</b>					
Own Diabetes	0.183*** (0.0716)	0.0724 (0.0532)	0.0749 (0.0507)	-0.276*** (0.0376)	0.0272 (0.0272)
Time Since Own Diagnosis (TSoD)	-0.00285** (0.00113)	-0.0000563 (0.000811)	-0.000826 (0.000780)	-0.000706 (0.000523)	-0.000000826 (0.000376)
Own Diabetes x TSoD	0.0141* (0.00743)	0.00379 (0.00537)	0.00641 (0.00535)	0.00287 (0.00346)	-0.00135 (0.00265)
Obs.	20680	35877	35899	41054	38156
<b>(b)</b>					
Partner Diabetes	0.212** (0.101)	-0.0272 (0.0761)	-0.0919 (0.0723)	-0.147*** (0.0495)	0.0411 (0.0365)
Time Since Partner Diagnosis (TSpD)	0.00121 (0.00173)	0.000924 (0.00116)	0.0000667 (0.00113)	-0.000827 (0.000646)	-0.000510 (0.000516)
Partner Diabetes x TSpD	0.00647 (0.0122)	-0.000978 (0.00813)	-0.00312 (0.00797)	0.000893 (0.00483)	0.0000568 (0.00411)
Obs.	10587	18156	18156	20768	19231

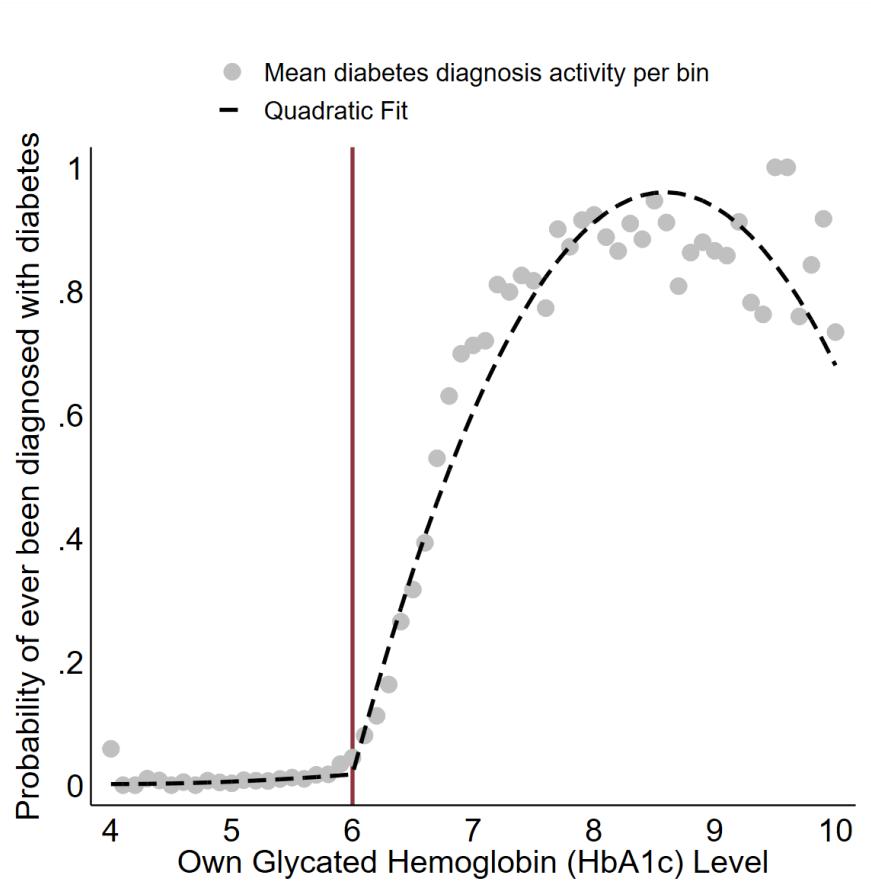
Coefficients are estimated using a quadratic specification each side of the kink point, equally weighted observations, and with bounds of 5.0 on the right hand tail, and 2.0 on the left hand side. Parentheses includes cluster-robust standard errors, clustered at the household level. Estimates include 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 5: Heterogeneous fuzzy RKD estimates of change in own behaviour as a result own and partner's diabetes diagnosis by educational level

	Exercise	Vegetable Consumption	Fruit Consumption	Currently a Smoker	Currently a drinker
<b>(a)</b>					
Own Diabetes	0.243*** (0.0796)	0.114* (0.0591)	0.126** (0.0566)	-0.264*** (0.0429)	0.00714 (0.0298)
Own College Degree (OCD)	0.257*** (0.0310)	0.103*** (0.0210)	0.128*** (0.0195)	-0.118*** (0.0123)	0.0173** (0.00863)
Own Diabetes x OCD	-0.334* (0.193)	-0.173 (0.133)	-0.264** (0.128)	0.0118 (0.0824)	0.121* (0.0657)
Observations	20680	35877	35899	41054	38156
<b>(b)</b>					
Partner Diabetes	0.257** (0.110)	-0.00217 (0.0819)	-0.0453 (0.0783)	-0.135** (0.0560)	0.0492 (0.0408)
Own College Degree (OCD)	0.199*** (0.0440)	0.0715** (0.0300)	0.118*** (0.0283)	-0.0937*** (0.0134)	0.0270** (0.0112)
Partner Diabetes x OCD	-0.216 (0.298)	-0.137 (0.223)	-0.286 (0.213)	0.0465 (0.0987)	-0.0223 (0.0812)
Observations	10590	18160	18160	20772	19234

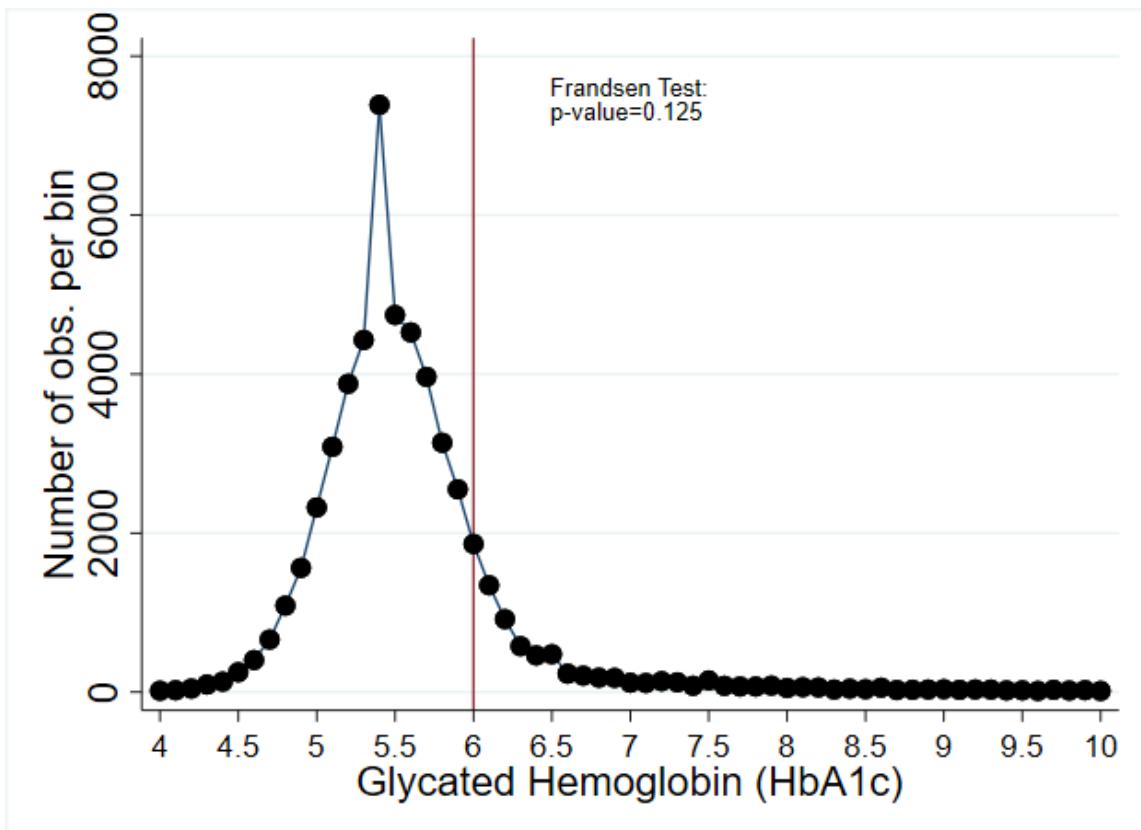
Coefficients are estimated using a quadratic specification each side of the kink point, equally weighted observations, and with bounds of 5.0 on the right hand tail, and 2.0 on the left hand side. Parentheses includes cluster-robust standard errors, clustered at the household level. Estimates include 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

Figure 1: Probability of Diabetes Diagnosis by HbA1c Level



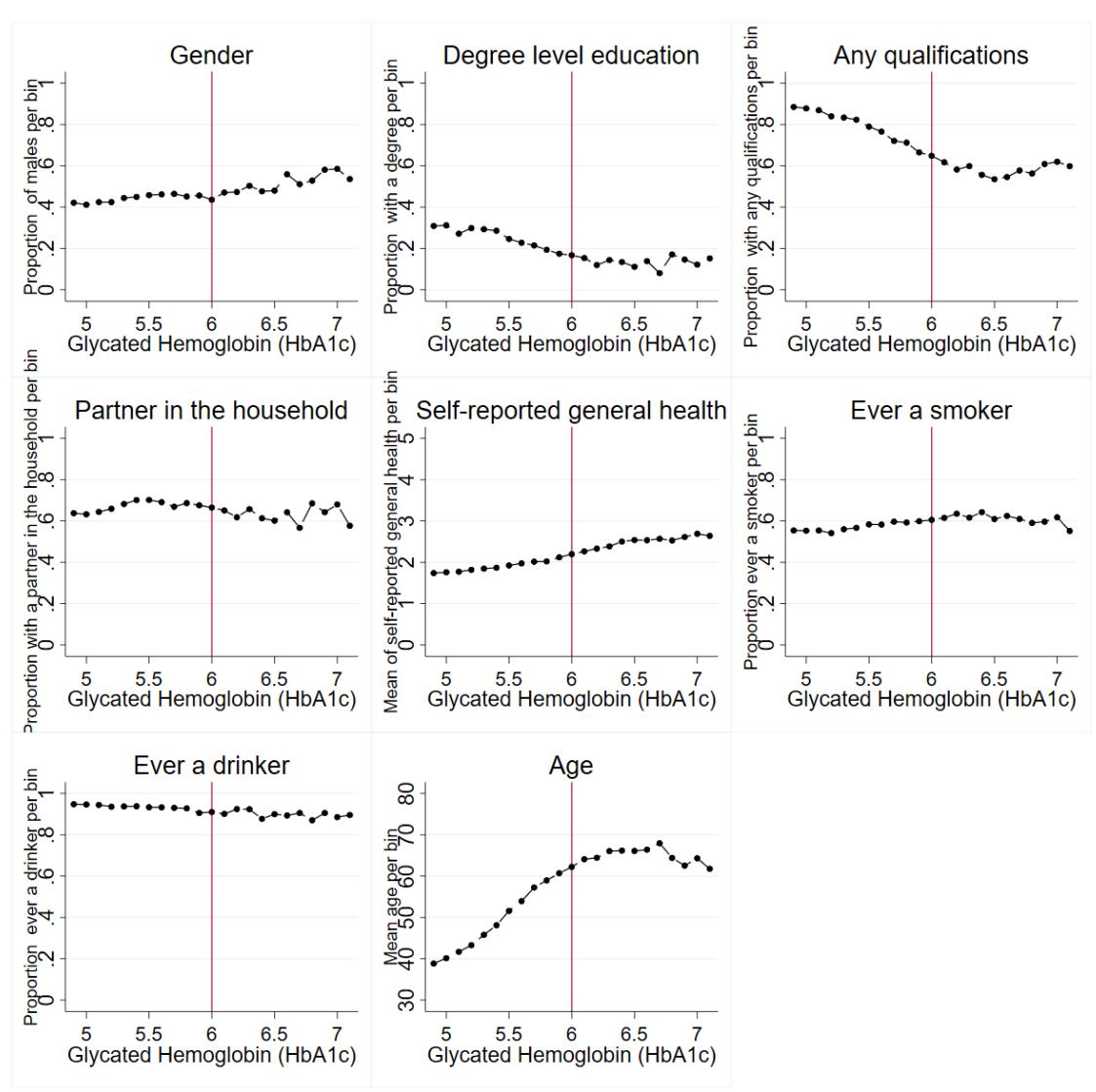
*NOTE:* Mean of the probability of ever being diagnosed with diabetes per bin. Bin width of 0.1 for glycated hemoglobin levels between 4 and 10. Quadratic fit is separately estimated for the left and right hand sides of the kink. Red line represents the kink point, where glycated hemoglobin is a value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

Figure 2: Smooth Density of the Assignment Variable



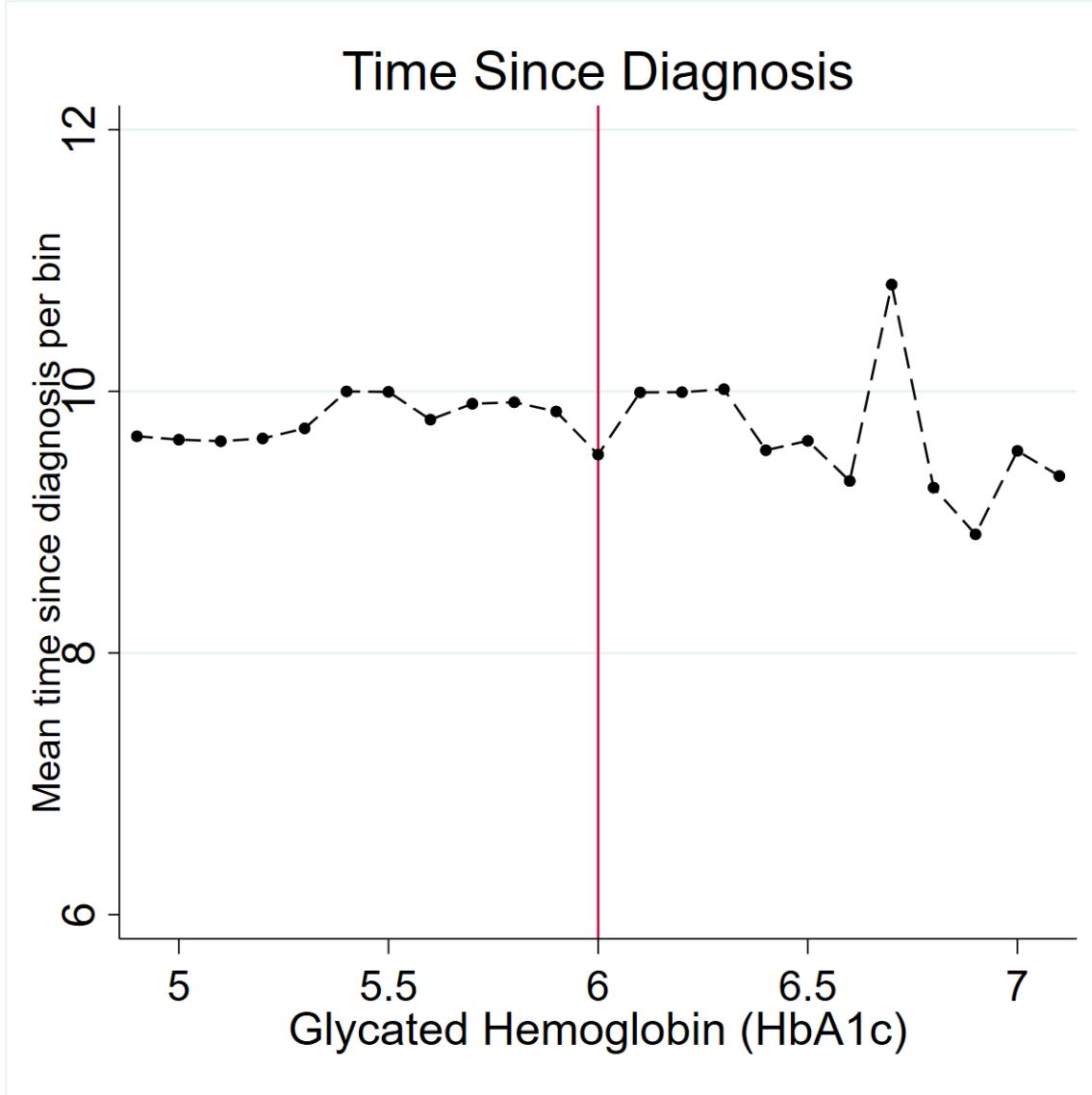
*NOTE:* Number of observations per bin. Bin width of 0.1 for glycated hemoglobin levels between 4 and 8. Graph also shows McCrary discontinuity statistic and the first derivative discontinuity statistic, both of which are insignificant.

Figure 3: Predetermined variables



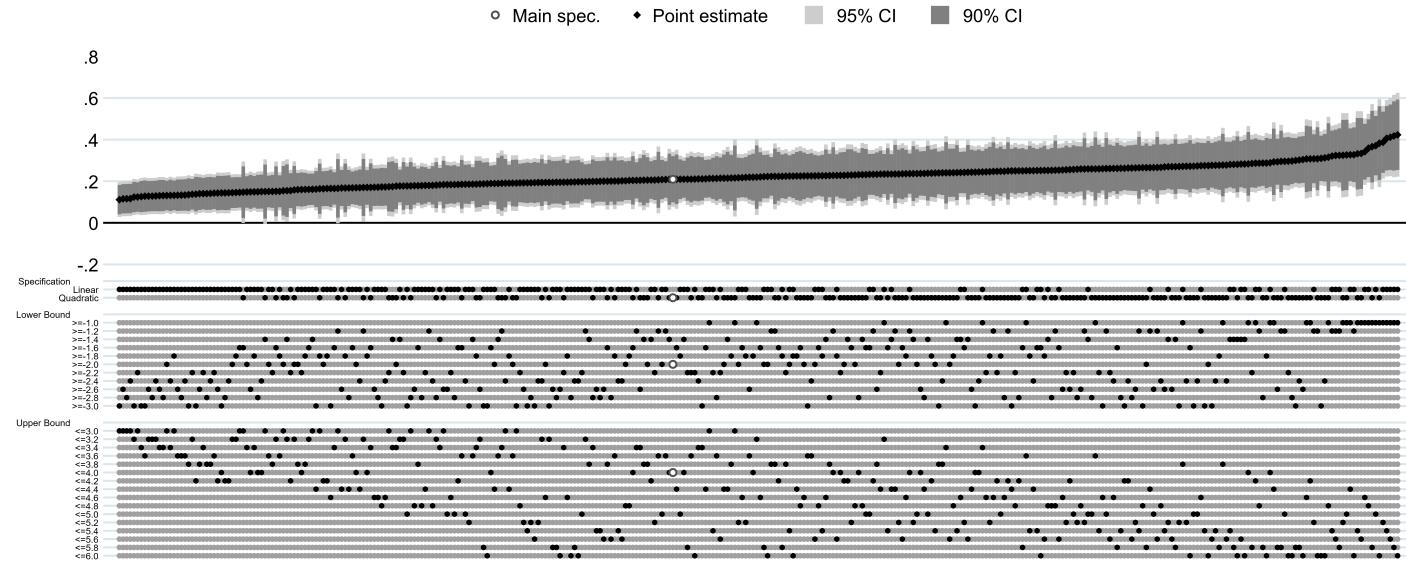
*NOTE:* Graphical representation of the mean of each predetermined variable by glycated hemoglobin (HbA1c) level. Each graph shows the mean of the predetermined variable per bin, with a bin width of 0.1. Predetermined variables included are gender, ethnicity, degree level education, any qualifications, 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 4: Time Since Diagnosis



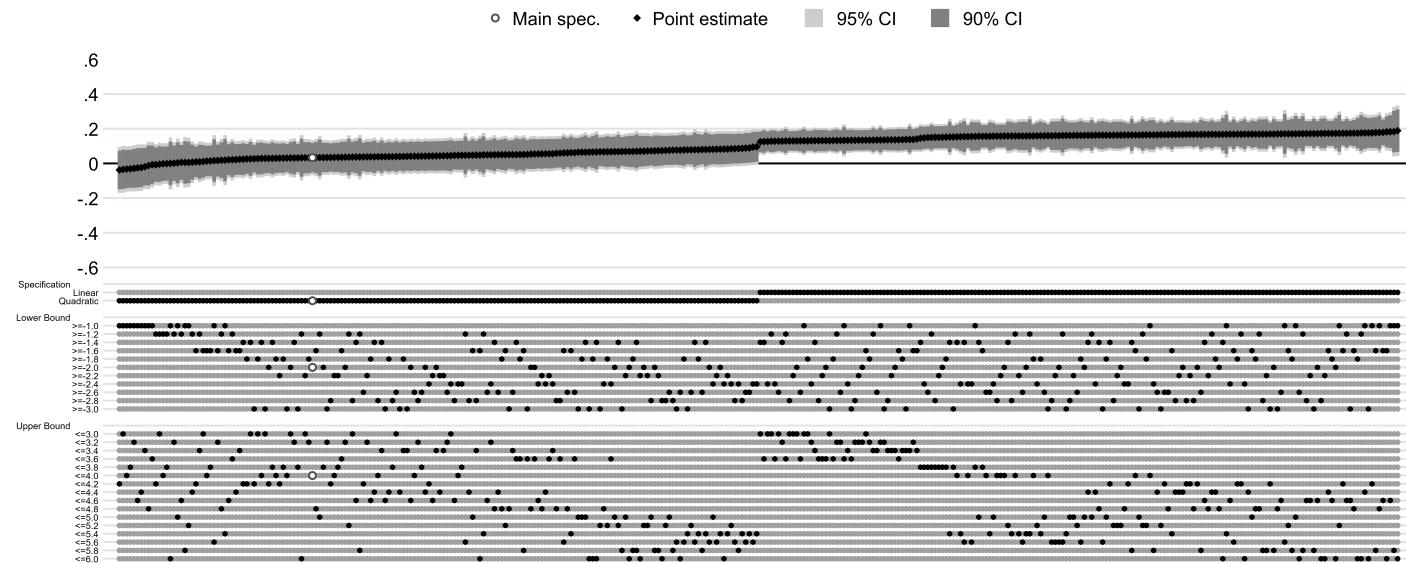
NOTE: Graphical representation of the mean of time since diabetes diagnosis by glycated hemoglobin (HbA1c) level. Graph shows the mean of the time since diagnosis per bin, with a bin width of 0.1. For individuals whom have never been diagnosed as having diabetes, they are assigned a placebo time since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

Figure 5: Sensitivity to alternative bandwidths and polynomials - Physical Activity



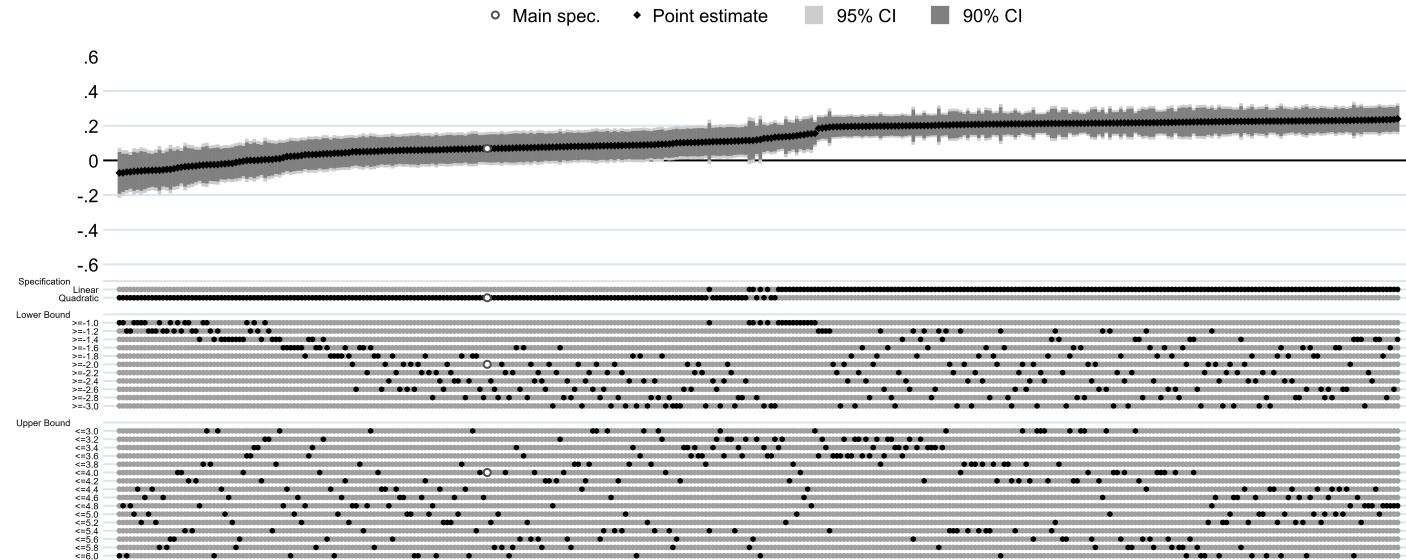
*NOTE:* The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents 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 white 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 6: Sensitivity to alternative bandwidths and polynomials - Vegetable Consumption



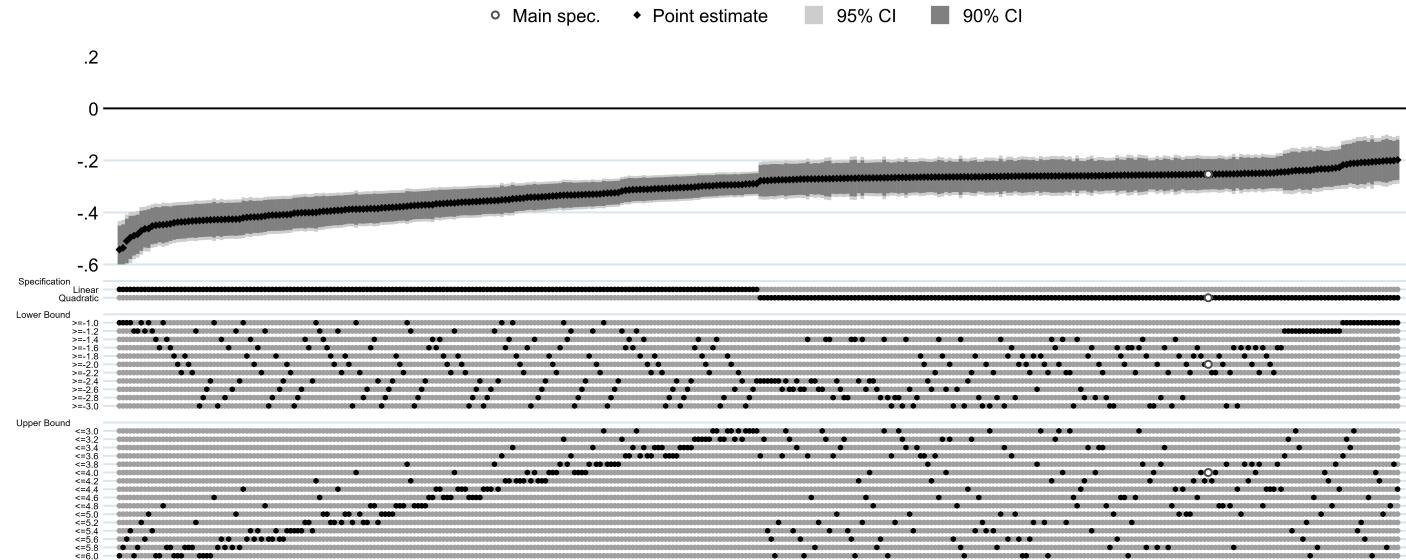
*NOTE:* The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents 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 white 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 7: Sensitivity to alternative bandwidths and polynomials - Fruit Consumption



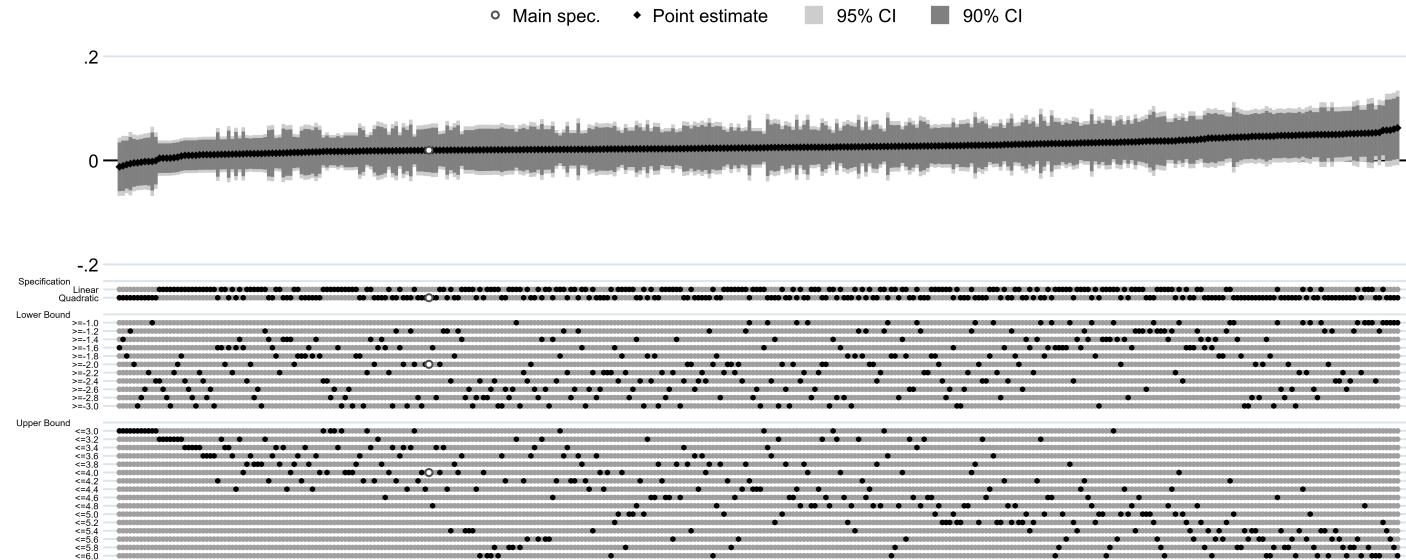
*NOTE:* The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents 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 white 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 - 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 white 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>.

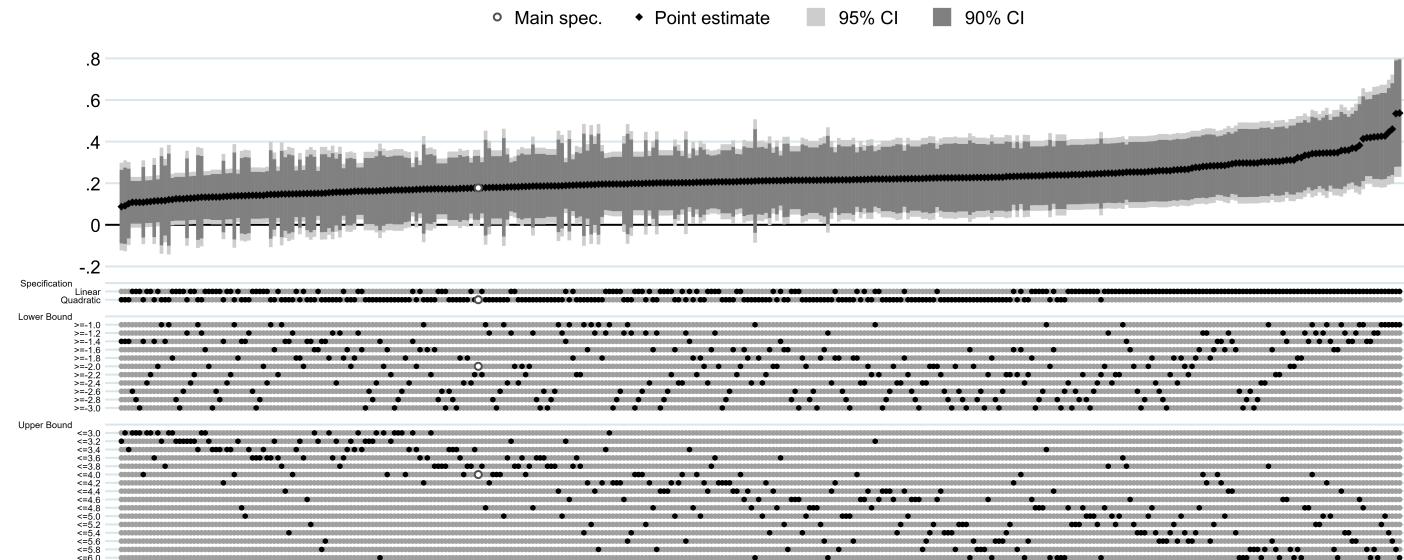
Figure 9: Sensitivity to alternative bandwidths and polynomials - Alcohol Consumption



*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 white 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 - Partner Spillover Estimates of Physical Activity

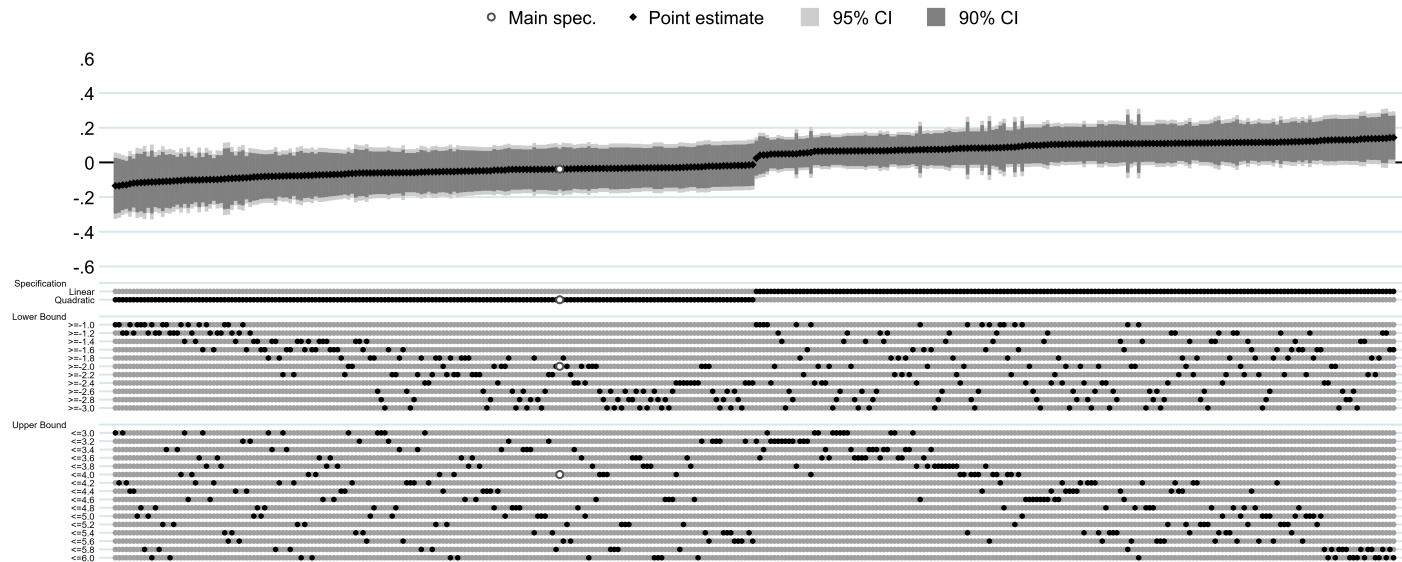
52



*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 white 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 - Partner Spillover Estimates of Vegetable Consumption

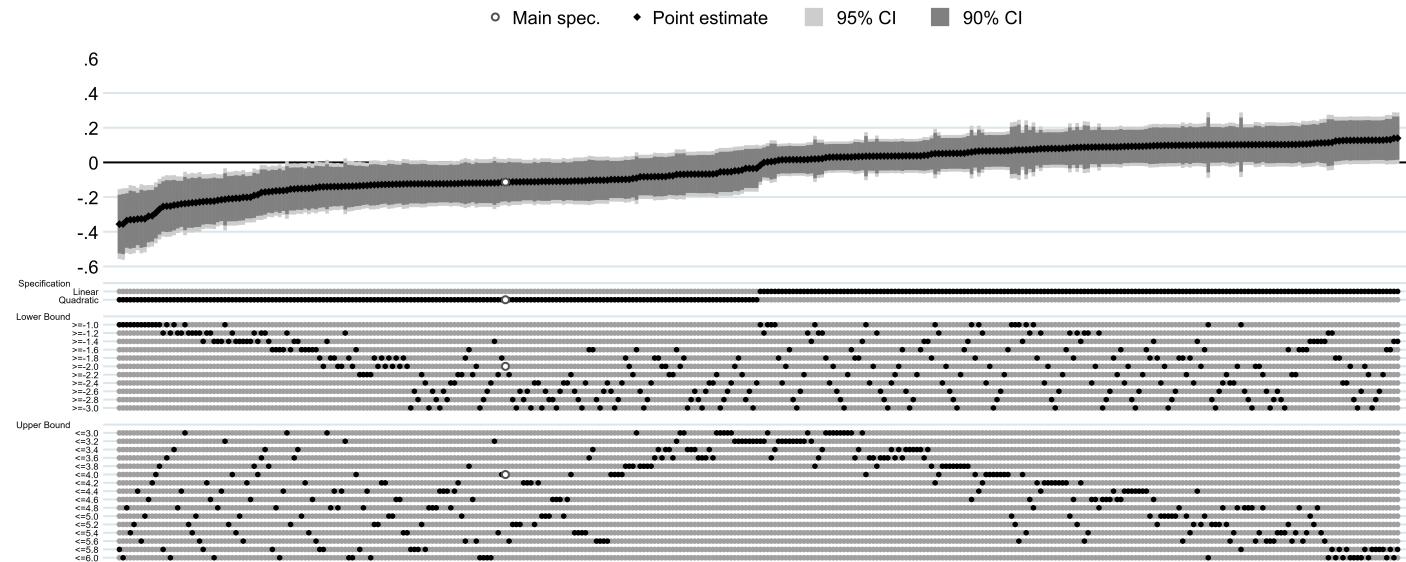
53



*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 white 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>.

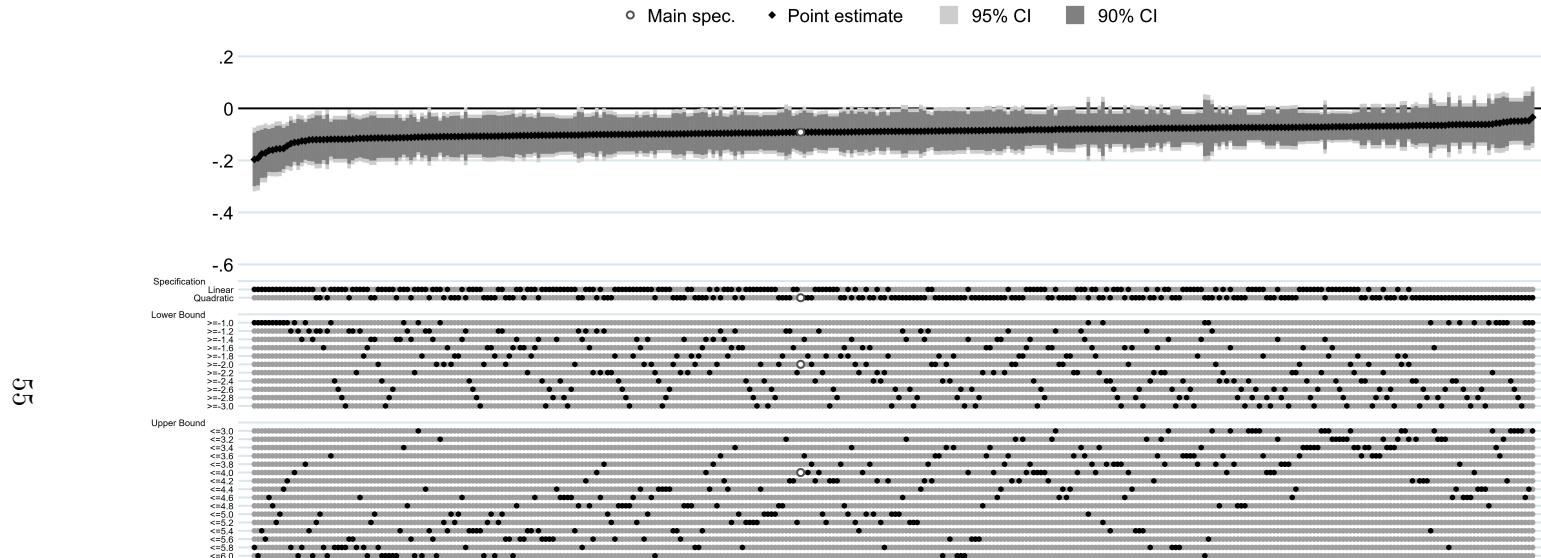
Figure 12: Sensitivity to alternative bandwidths and polynomials - Partner Spillover Estimates of Fruit Consumption

54



*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 white 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>.

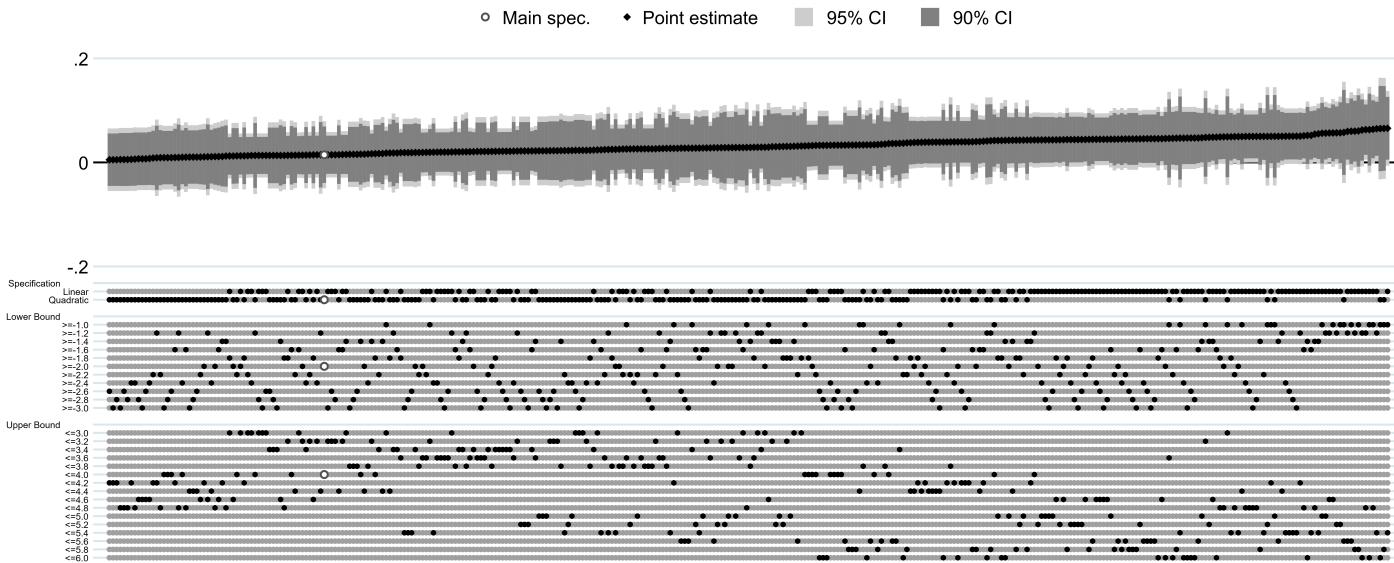
Figure 13: Sensitivity to alternative bandwidths and polynomials - Partner Spillover Estimates of Smoking Behaviour



*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 white 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 14: Sensitivity to alternative bandwidths and polynomials - Partner Spillover Estimates of Alcohol Consumption

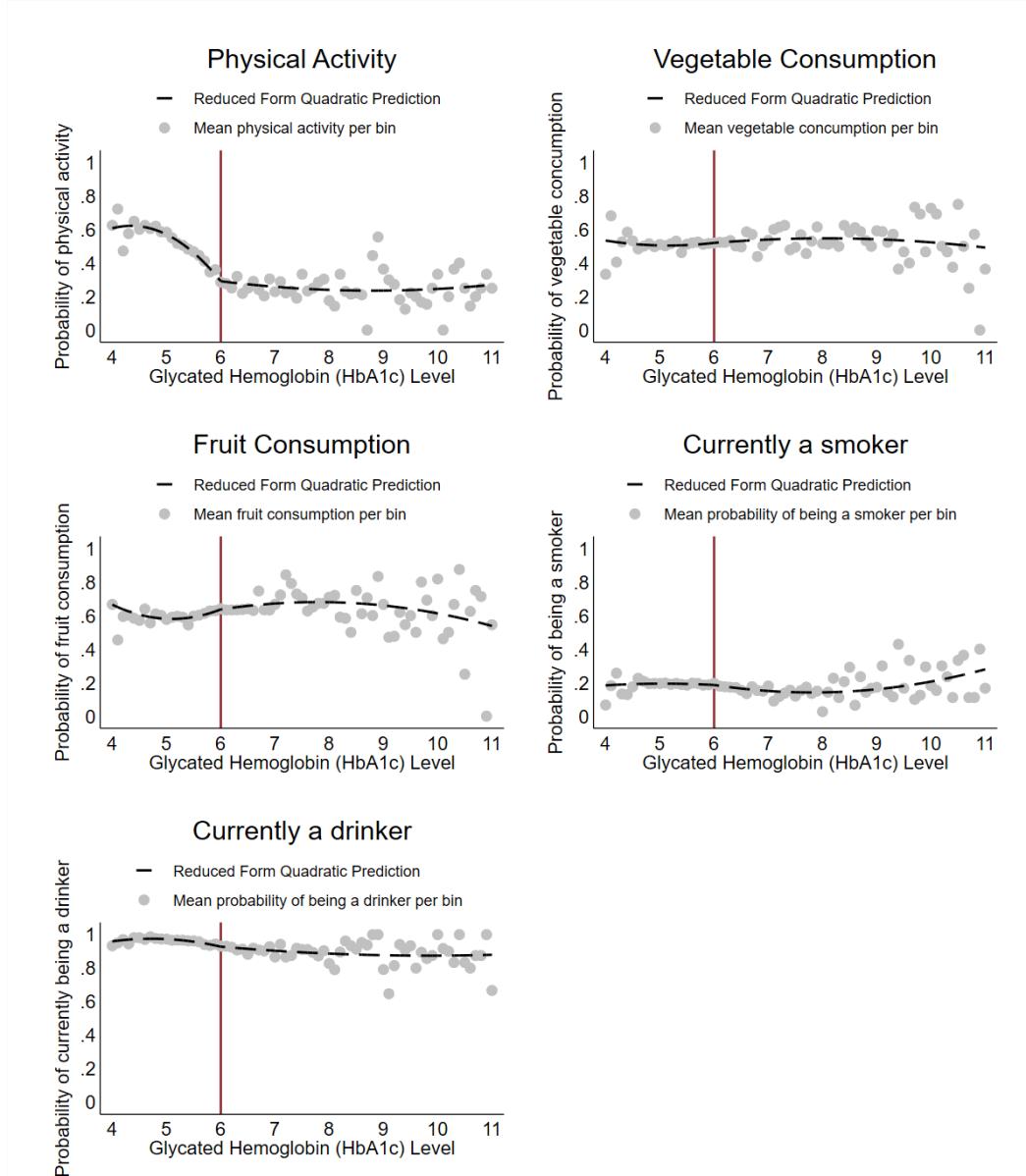
56



*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 white 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>.

## **9 Appendix**

Figure A1: Graphical Representation of Reduced Form RKD Results - Own Glycated Hemoglobin

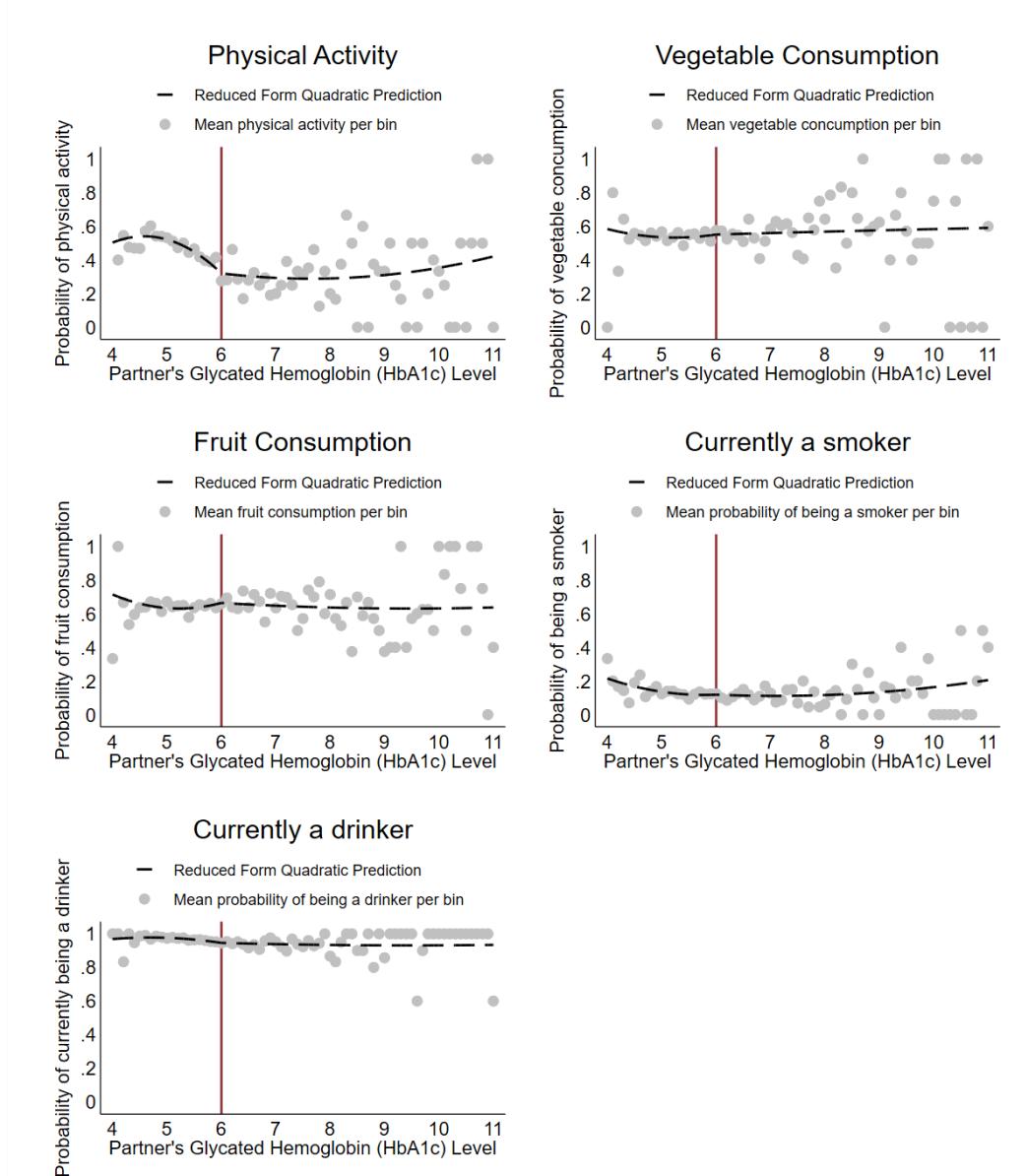


*NOTE:* These figures are a graphical representation of the RKD. Figures show the mean outcomes per bin (grey points), where bin width is 0.1, between HbA1c levels between 4.0 and 10.0. Black dashed line represents the quadratic prediction from the reduced form a regression of the form:

$$y_i = \chi_0 + \chi_1(x_i - k)D_i + \left[ \sum_{p=1}^{p^*} \psi_p^- (x_i - k)^p \right] + \left[ \sum_{p=2}^{p^*} \psi_p^+ (x_i - k)^p D_i \right] + \mu_i$$

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 A2: 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 (6) and (7) respectively are available in table (2).