The direct and spillover effects of diabetes diagnosis on lifestyle behaviours

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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.

Keywords: Diabetes, Health, Spillover, Household Behaviour, Regression Kink Design
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1 Introduction

There is substantial literature documenting a positive correlation in spousal behaviours with much of the work focusing on smoking behaviour and alcohol consumption (Christakis and Fowler; 2008; Falba and Sindelar; 2008). Similar strong positive correlation of behaviour between spouses has also been reported for physical activity Farrell and Shields (2002); Falba and Sindelar (2008) and diet (Macario and Sorensen; 1998; Bove et al.; 2003). However, such correlations extend beyond behaviours alone with previous work reporting spousal correlation in mental and physical health (Meyler et al.; 2007; Di Castelnuovo et al.; 2009). Three theories have been put forward to understand the causal pathways of these strong empirical correlations, namely: assortative matching, shared environment, and joint household decision making (Clark and Etil; 2006; Cutler and Glaeser; 2010; Chiappori et al.; 2012).

Assortative matching views partners' characteristics and preferences as complements which drive individuals to match with partners they share preferences and characteristics with (Becker; 1973). In a shared environment partners make decisions individually based on their preferences, but are constrained by shared resources and exposed to common shocks, which give rise to observed correlated behaviours. An epidemiological dimension is implicit whereby partners who share a common environment are also exposed to common health risks factors. An additional channel under this pathway relates to 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 partners also have similar expectations of future uncertainty and risk, and as a result make similar behavioural choices (Khwaja et al.; 2006). Finally, joint household production leans on the theory of New Home Economics where households jointly produce goods which enter individuals' utility functions (Lancaster; 1966; Becker; 1981). 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 results in empirical 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. Only a handful of studies have explored such externalities in the context of health. Fadlon and Nielsen (2019) analyse the spillover effects on an extended network of individuals as a result of fatal and non-fatal heart attacks. They find significant and persistent increases in statin consumption of spouses, children and co-workers of individuals who had a non-fatal heart attack, and offer evidence in support of both learning new health information, and salience explaining the estimated effect. Fletcher and Marksteiner (2017) use experimental data to estimate spillover effects of smoking cessation therapy program and alcoholism treatments. They find significant impact in both behaviours and their experimental design can reasonably preclude a matching in the marriage market explanation. However, their results are at odds with the conclusions by Clark and Etil (2006) who show that social learning and household decision making play a minor role in explaining raw correlations between partners. Once controlling for individual random effects smoking behaviours are statistically independent between partners, suggesting that all spousal correlation in smoking behaviour is the result of correlations in the individuals' effects, which Clark and Etil interpret as evidence of assortative matching. Finally, Janssen and Parslow (2021) examine the presence of spillover effects within a household when looking at the impact of pregnancy on alcohol consumption. Pregnancy persistently reduces household alcohol consumption with reductions in purchasing of both beer and wine. Given that males are the prominent beer drinkers in the United States, the authors interpret this as evidence in favour of a spillover effect from females onto males in the household.

In this paper we investigate the effect of diabetes on individual and partner' lifestyle behaviours, namely physical activity, diet, alcohol and smoking consumption. These lifestyle behaviours are well established risk factors of non-communicable diseases (Willi et al.; 2007; Ezzati and Riboli; 2012, 2013) and constitute the first line of treatment of diabetes (WHO; 2016). Using 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 on own behaviour, as well as, the effects of own diabetes status on partners' behaviour. The identification strategy allows us to exclude assortative matching as a causal pathway, while through a recursive model we decompose the spillover effect into its shared environment and joint household production contributions. Further, we explore three sources of heterogeneity over observables. First, we test whether own behaviour changes as a function of living with a spouse or not. Second, we use time since diabetes diagnosis to examine differential impact on own and partner lifestyle outcomes, which, in the absence of panel data, approximates long-term effects or recidivism to pre-diagnosis behaviours. Third, we assess whether there are observable heterogeneities by individual education. Finally, we present falsification tests over multiple health outcomes that would not be expected to be impacted by diabetes status.

Briefly, we find significant effects of diabetes diagnosis on own physical activity and smoking, while partners' of individuals with a diabetes diagnosis also increase their physical activity and decrease their probability of currently being a smoker. Spillover effects are mostly driven by partner's behaviour and less so by the partner's diabetic status. We find little evidence of heterogeneity of the effect of own or partner diabetes on behaviour by presence of partner in the household, time since diagnosis or education. All falsification tests support our identification strategy and provide evidence towards the robustness of the results.

We contribute to the literature in a number of ways. First, we provide evidence within the household economics literature that observed correlated partners' health behaviours are not limited to assortative matching, but that social learning and joint household decision making are important components of the observed correlation. Second, we 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). This is related to how these behaviours are determined and influenced, as well as to individuals' compliance with first line treatments for diabetes. Our results suggest that individuals with diabetes 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 not accounted for in the evaluation of health care policies in this area. Finally, we contribute to a new and growing literature on health-related spillover effects by analysing the effects of a health shock on lifestyle behaviours commonly acknowledged as important risk factors of non-communicable diseases.

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) defines diabetes as "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, which occurs when insulin production in the body is limited (Diabetes UK; 2019). Although there is limited understanding on its causes, diet or lifestyle are not known 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 and is usually found to be a result of poor diet and lifestyle (Helmrich et al.; 1991; Hu et al.; 2001).

Glycated haemoglobin (HbA1c) refers to the amount of haemoglobin (i.e. protein within red blood cells) which has 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.[1]

The World Health Organisation 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 (WHO; 2011). Levels below 6% are considered normal blood sugar levels and

[1] An alternative measure, blood glucose level, is the concentration of sugar in the blood at a single point in time and is highly variable within individuals, and more dependent on very recent consumption than persistent behaviour.

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.[2] Nevertheless, although pre-diabetes usually has no symptoms, NICE[3] 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 pre-diabetic and 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 probability 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 associated with non-communicable diseases. Clinical recommendations regarding such risk-factors are clear and well-known to the general population, rendering a priori expectation of the effects straightforward. Namely increasing physical activity and vegetables consumption and decreasing tobacco and alcohol consumption mitigate the risk of developing diabetes and are important first-line treatments of the disease (WHO; n.d.). On the contrary, while the health benefits of fruit are well established, recommendations on fruit consumption for diabetic patients is somewhat ambiguous and possibly misunderstood by the general population [4] making a priori expectations unclear.

[2]Yudkin and Montori (2014) state that "glycaemia are continuous, with no inflections to provide obvious cut-off points. Cut-offs for the diagnosis of diabetes are based on thresholds for risk of retinopathy. Lesser degrees of hyperglycaemia increase the risk of developing diabetes and maybe arterial disease. But in both cases the risk is graded, making any choice of cut-off point purely arbitrary." This claim is also supported by NICE (2011, 2012)

[3]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.

[4]On one hand, experts encourage fruit consumption due to their low energy density, and high content of vitamins, minerals, phytochemicals and dietary fibre. On the other hand, 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). 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), which can potentially cause confusion due to the high sugar content 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' behaviours 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,

Indeed, there are a number of ongoing campaigns to resolve understanding of the guidelines (Diabetes UK; n.d.). However, confusion is present both among healthcare professionals and patients with 25% and 57%, respectively, stating that "fresh fruit can be eaten freely with little effect on blood glucose levels" (Speight and Bradley; 2001). Forouhi et al. (2018) 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".

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[5] 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 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 in the present paper and our identification strategy minimizes the possibility that our estimates are the result of assortative matching.

3 Data

The paper uses data from the Health Survey for England (HSE) for years 2003 to 2015. 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

[5] 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.

to measure among others, glycated haemoglobin (HbA1c). Although 82.4% of individuals (across all years) agreed to be contacted for a nurse visit, only 34.7% of the full sample had blood samples taken for analysis. Of the 56,245 individuals who had blood taken in the survey, 53,450 individuals had valid HbA1c measurements [6].

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". Information relating to diet in the HSE is limited, however we use two relevant 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 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 subsequent columns we give summary statistics of the sub-sample of individuals who did have blood taken for analysis and whose data is used in our estimations. We break 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

[6] 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.

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 10.06 years ago (standard deviation of 10.46). 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 Marginal Treatment Effect (MTE) 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. We note, however that our identification strategy is not invalidated by such data structure and we present it in detail in the following section.

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:

$$y_i = \theta_0 + \theta_1 EverD_i + \theta_2 EverD_j + e_i \tag{1}$$

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 θ_1 and θ_2.

The first and possibly most salient source of bias is simultaneity. It is possible that individuals with diabetes may display behaviour damaging to their health compared to those without diabetes. Such correlation, however, ignores 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; Hu et al.; 2001). A second source of endogeneity that would bias least squares estimation of θ_2 in equation (1) is matching in the marriage market (Dupuy and Galichon; 2014). Individuals selectively marry along similar traits and therefore ignoring this channel through a naive estimation will again bias estimates of the spillover effect.

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 precise 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) provides the theoretical framework for identification in our setting, whereby the RKD identifies the causal effect of a binary treatment when there is no 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). A fuzzy RKD 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%. $D_i = \mathbb{1}(x_i \geq k)$, is an indicator variable, taking the value of one if the individual's level of HbA1c is above the kink point, and where $(x_i - k)D_i$ is the excluded instrument for the fuzzy RKD. p^* denotes the highest order of polynomial used in the regressions, ν_p^- and ν_p^+ are the estimates of the polynomial function below and above the kink point, respectively.

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

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

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 β_1 can be interpreted as the unbiased Marginal Treatment Effect (MTE) 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. Given that in the dataset diabetes diagnosis is predetermined (i.e. past diagnosis), yet HbA1c is contemporaneous, this potentially creates confusion over identification but does not invalidate it.

The fact that individuals to the left of the 6% cut-off may have received a past diabetes diagnosis and others to the right of 6% may not have had a diagnosis [7] suggests that we

[7]There are individuals to the right of the kink-point that are not diagnosed with diabetes, and indeed being to the right does not strictly increase their probability of being diagnosed with diabetes. These individuals can be thought of as never-takers. Being to the right of the kink-point does not increase their probability of being diagnosed. Various explanations could be offered for this. Correspondingly, there are individuals who are to the left of the kink-point and yet have been diagnosed with diabetes. Firstly, being to the left of

do not have a strictly deterministic function of diabetes diagnosis by HbA1c level but a kinked function (i.e. the change in the probability of diagnosis around the cut-off) driven by a policy rule. There is no medical reason for this kink in the diabetes probability, and most individuals are not even aware of their HbA1c level.

It is this exogeneous kink that identification rests upon, and not HbA1c per se, or an individuals place in the HbA1c distribution. Past and present lifestyle behaviours can both be correlated and impacting HbA1c (this would certainly be expected as a result of diabetes treatment) but are all unable to precisely affect HbA1c location around the kink-point (Dong; 2011).

Hence, exogeneity would require that kinks around the cut-off would not be expected for lifestyle behaviours and, by implication, any kink in behaviours would be driven by the kinked probability of diabetes diagnosis. Such assumption (i.e. kinks not being present in the structural outcome equation) is, by and large, innocuous as there is no reason why the kink in HbA1c should directly impact behaviour. On the contrary, the running variable HbA1c may be reasonably included in the structural outcome equation, however inclusion of the kink itself is hard to justify intuitively. Rather, the kink has a predictive effect on diabetes diagnosis, hence its relevant as an instrument. The kink can only plausibly impact behaviours through its effect on probability of diabetes diagnosis.

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

the cut-off does not strictly eliminate the probability that an individual is diagnosed with diabetes, these individuals also face a small probability that they were diagnosed with diabetes. These individuals can be thought of as always takers and not defiers. Although these individuals have a diabetes diagnosis, being to the left does not make them defiers, and they do not per se violate the monotonicity assumption. It is implausible that we have defiers in our setting. A defier in our setting would need to have a decreasing probability of ever being diagnosed with diabetes due to being to the right of the cut-off, which does not seem reasonable. Alternatively, being to the left of the kink-point leads to a higher probability of ever being diagnosed, than those to the right, which seems implausible. An individual may have a positive diabetes diagnosis, however, being to the right of the kink would always increase this probability.

null hypothesis.

However, to ensure that our results are robust, we transparently present a number of alternative specifications and 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 small sample size is less of a problem (i.e. asymmetric bandwidths). Our main set of results, uses a bandwidth of 4.0% on the right hand side of the cut-off and 2.0% on the left hand side (i.e. HbA1c values of 4% to 10% are included in the estimation sample).

To improve precision and reduce 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, and a binary indicator denoting whether a partner lives in the household.

4.2 Partner's Diabetes Status

To handle the endogeneity in the effect of partner's diabetes diagnosis on own behaviour, we adapt the previous setup by using the partner's kink as an instrument for partner's 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 estimating the causal relationship is

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

Once again, y_i denotes the health related behavioural outcome of interest. $\widehat{EverD}_j$ is the predicted probability, from the first stage, of partner ever being diagnosed with diabetes, while again the terms in the square brackets denote the polynomial function below and above the kink point. 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 is reduced as it is restricted to those who have partners, and those partners have HbA1c levels within the bandwidths. As previously, the same set of covariates for both i and j (excluding whether partner lives in the household) are included in the regression.

We interpret these results to be a spillover effect, and exclude the possibility that our estimates are the result of assortative matching. To exclude assortative matching, we require that matching does not happen based on being either side of the kink-point. It is certainly possible to assume that individuals match based on their relative position in the HbA1c distribution, or some unobservable variable correlated with HbA1c, and indeed, doing so does not violate the identifying assumption, however it seems less plausible that individuals would specifically match based on being just either side of the kink-point. For matching to explain our estimates, it would require individuals to be aware enough of their own HbA1c level at the time of matching, and to selectively match based on being either side of the arbitrary kink-point. Given that most individuals are not aware of their own HbA1c for this to be possible, and there appears to be no underlying incentive to match based on this arbitrary threshold, it seems implausible that assortative matching would be affecting our estimates.

5 Main estimation results

5.1 Effect of own diagnosis

Panel (a) in Table 8 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 8 gives the coefficient β_1 from equation (3). We find that being diagnosed with diabetes significantly increases the probability of having done 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. [8]

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, β_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 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, across all specifications point estimates are above zero and in almost all cases confidence intervals exclude zero. Overall, results 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 includes 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 either outcome, given that our main specification, a quadratic polynomial, supports a null effect, and that significance of these estimates are clearly specification dependent. We therefore conservatively claim lack of evidence of an effect of diabetes on vegetable or fruit consumption.

Findings for smoking behaviour, Figure 8, are similar to those of physical activity with

[8] 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 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.

point estimates varying little across specifications and all specifications featuring tight confidence intervals excluding zero. Estimates from a quadratic specification appear to be very robust and all sitting within a small interval around -0.3 also with 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 for our preferred specifications with a quadratic polynomial.

5.2 Spillover effect

The spillover estimates as a result of partners' diabetes diagnosis, i.e. parameter δ_1 in eq. 5, are presented in Panel (b) of Table 8. 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[9]. Specifically, we find significant positive effects for exercising in the 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.

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 similar patterns to those for the effect of own diabetes diagnosis. One point of difference is that confidence intervals for spillover effects are substantially larger than those for own behaviour. This is to be expected given differences in the estimation sample sizes between spillover and own effects. Indeed, we find that large confidence intervals are especially present in specifications with narrow bandwidths or higher order

[9] 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.

polynomials, and therefore power might be of concern 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 MTE of diabetes diagnosis, two observable implications must hold (Card et al.; 2015). 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 discountinuity 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 does hold 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 is that the tests 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 $k = 0$ implies a null hypothesis of linearity and an alternative hypothesis of non-linearity around the threshold (i.e. jump or kink), which would mean that our assumption of smooth density fails. 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, which 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

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 the lack of any discontinuity it 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 [10], 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 assumption and suggesting that interpretation of the results of the RKD as MTEs is valid.

6 Robustness checks

6.1 Simultaneous Own and Partner's diabetes status

Having obtained evidence for the consistency of the RKD estimations in our setting we pursue sensitivity issues 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, $z = j$.

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

[10] 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).

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 8. 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 substantially. This is 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 (b) and (c) of Table 8 suggest that for the vast majority of models, coefficients magnitudes are comparable, and indeed are almost identical for physical activity and smoking behaviour, but effects in Panel (c) are estimated with less precision and hence much higher standard errors.

6.2 Falsification Tests

As additional robustness checks, we present falsification tests, where we use the identification strategy presented in section 4.1 to estimate the effect on several outcomes which we *a priori* expect to be zero. Estimating a null effect in outcomes which we do not expect to be effected by a diabetes diagnosis provides further evidence that our identification strategy is valid and our estimated effects are not spurious.

We analyse the effect on a set of three other medical outcomes, namely whether individuals take: antibiotics, anti-depressants or statins. We, further, include one other pre-determined variable, whether ever been in paid employment, to extend the falsification checks beyond only medical outcomes. In addition, to check the robustness of identification for disentangling own and spillover effects, we examine the direct and spillover effect of diabetes diagnosis on whether currently taking anti-diabetic medication. In this case, we would expect to find a strong direct effect, but no evidence of a spillover effect onto partners as partner's diabetes diagnosis should not *per se* increase probability of own receiving anti-diabetic medication.

Estimates of the effects on these outcomes are presented in Table 9, and, we additionally present the same robustness graphs for our main estimates in Figures 15 - 24. Firstly, as expected, we find clear evidence of an increase in probability of taking anti-diabetic medication for own diabetes diagnosis but no evidence of a spillover effect. In terms of our other estimates, reassuringly we find no evidence of an effect on any of the outcomes used in the falsification tests. The robustness graphs also support the results presented in Table 9, in almost all specifications we estimate we find null effects, aside from the direct effect on antidiabetes medication, where there is clear significant effects across all specifications. All in all, testing strongly supports our identification strategy.

7 Causal Pathways

As discussed in detail in section 2.2, the correlation between spouses can theoretically be attributed to assortative matching, shared environment and joint household decision making. Our identification strategy allows us to plausibly exclude attributing spillover effects to assortative matching, which leaves us with two possible channels. We decompose spillover effects, and assess the contribution of shared enviroment and joint household production to the overall spillover effect. To do so we use a recursive model where we separately identify changes in behaviour that are the result of partner's diagnosis, and changes in behaviour that are the result of the induced change in partner's behaviours. The first and second stage of the recurisive model are the same as those used for the estimation of the direct effects (i.e. equations 2 and 3 from Section 4.1), namely, the first stage:

$$EverD_i \;=\; \gamma_0 + \gamma_1(x_i - k)D_i + \left[\sum_{p=1}^{p^*}\nu_p^-(x_i-k)^p\right] + \left[\sum_{p=2}^{p^*}\nu_p^+(x_i-k)^pD_i\right] + \xi_i \quad (8)$$

and second stage:

$$y_i \;=\; \beta_0 + \beta_1\widehat{EverD}_i + \left[\sum_{p=1}^{p^*}\alpha_p^-(x_i-k)^p\right] + \left[\sum_{p=2}^{p^*}\alpha_p^+(x_i-k)^pD_i\right] + \epsilon_i \quad (9)$$

To assess the contribution of each of these pathways to the overall spillover effect we estimate a third stage, which takes the form:

$$y_j \;=\; \vartheta_0 + \vartheta_1\widehat{y_i} + \vartheta_2\widehat{EverD}_i + \left[\sum_{p=1}^{p^*}\varrho_p^-(x_i-k)^p\right] + \left[\sum_{p=2}^{p^*}\varrho_p^+(x_i-k)^pD_i\right] + \varsigma_j \quad (10)$$

Following the same notational convention as above, $EverD_i$ is whether individual i has ever been diagnosed with diabetes, and $\widehat{EverD}_i$ is the predicted probability from equation 8. y_i denotes the health related behavioural outcome of interest, and $\widehat{y_i}$ is the predicted equivalent from 9. x_i denotes the running variable (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. p^* denotes the highest order of polynomial used in the regressions, terms in square brackets are the estimates of the polynomial function below and above the kink point, respectively.

ϑ_2 is an estimate of the effect of change in partner j's behaviour that is a result of partner i's diagnosis itself. We attribute this pathway to the health information causal channel. In this case, the diabetes diagnosis of partner i has a "direct" effect on partner j's behaviours. As a result of the diagnosis, partner i receives new health information, possibly from a physician, about their diagnosed condition which they then share with the non-diagnosed partner j. The transfer of information from parter i to j therefore provides j with a new information set which they use to privately re-evaluate their optimal behaviour. This informational transfer may induce a change in behaviour in partner j if the new health information changes expected future payoffs. However, the magnitude of the effect is dependent on the pre-diagnosis information set, as well as idiosyncratic preferences.

Estimate ϑ_1 captures the change in own behaviour that is caused by the induced change in partner's behaviours. This effect is attributed to the joint household decision making causal pathway. If jointly participating in these activities are complements, that is behaviours co-move independent of diabetes status or new health information, because individuals' gain utility from jointly participating in these behaviours, then it is reasonable to attribute the spillover to joint household decision making. The complementarity of these behaviours induce a change in partner j's behaviours as a result of i's diagnosis-induced behavioural change. This is clear in the case of smoking behaviour, as we would expect that quitting tobacco would be more difficult if another household member continued consuming tobacco. In terms of physical activity, individual j may get a utility or dis-utility from taking exercise, however joint time with their partner may provide significant utility, sufficient enough to make it a utility increasing choice.

We do not estimate the recursive model for vegetable consumption, fruit consumption and currently being a drinker because we find no evidence of a spillover effect for these outcomes, and there is no evidence of a direct effect. Therefore such outcomes do not meet the relevance restriction required. Estimates from equations 8 and 9 are the same as the direct effect presented in table 8.

Table 10 provides estimates of ϑ_1 and ϑ_2 from equation 10. For physical activity and tobacco consumption we find that the spillover effect is driven by partner's behaviour y_i, and we find limited evidence that the diagnosis itself is causing a change in behaviours of j. Results suggest that the estimated spillover effect we find is the result of joint household production rather than information sharing.

8 Observed heterogeneity in RKD estimates

Following the main analysis and identification properties, we assess whether effects of a diabetes diagnosis are heterogenous 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 Hetergoenous Marginal Treatment Effect (HMTE) in a similar vein to Becker et al. (2013), by replacing the MTE 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 $\gamma = \widehat{\gamma} + \widetilde{\gamma} z_i$ where z_i denotes the trait across which heterogeneity is examined. The first stage equation is re-written as:

$$\begin{aligned} EverD_i = \mu_0 + \mu_1 z_i + \mu_2 (x_i - k) D_i + \mu_3 (x_i - k) D_i z_i + \sum_{p=1}^{p^*} \left[\nu_p^- (x_i - k)^p + \nu_p^- (x_i - k)^p z_i \right] \\ + \sum_{p=2}^{p^*} \left[\nu_p^+ (x_i - k)^p D_i + \nu_p^+ (x_i - k)^p D_i z_i \right] + w \quad (11) \end{aligned}$$

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

$$\begin{aligned} y_i = \psi_0 + \psi_1 z_i + \psi_2 \widehat{EverD}_i + \psi_3 \widehat{EverD_i} z_i + \sum_{p=1}^{p^*} \left[\upsilon_p^- (x_i - k)^p + \upsilon_p^- (x_i - k)^p z_i \right] \\ + \sum_{p=2}^{p^*} \left[\upsilon_p^+ (x_i - k)^p D_i + \upsilon_p^+ (x_i - k)^p D_i z_i \right] + m_i \quad (12) \end{aligned}$$

The parameters of interest here are ψ_2 and ψ_3, where the estimate of ψ_3 describes the heterogeneity in the treatment effect over the trait under inspection z_i. The rest of the notation is as previously. The inclusion of an additional term to estimate, ψ_3, which is dependent on the endogenous variable $EverD_i$ requires an additional instrument for the 2SLS estimates to be correctly identified. We, therefore, estimate an auxiliary first stage regression:

$$EverD_iz_i = \omega_0 + \omega_1 z_i + \omega_2(x_i - k)D_i + \omega_3(x_i - k)D_iz_i + \sum_{p=1}^{p^*}\left[\sigma_p^-(x_i - k)^p + \sigma_p^-(x_i - k)^p z_i\right]$$
$$+ \sum_{p=2}^{p^*}\left[\sigma_p^+(x_i - k)^p D_i + \sigma_p^+(x_i - k)^p D_i z_i\right] + z_i \quad (13)$$

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 HMTE estimation strategy closely follows that of Becker et al. (2013) with the key difference being that ours is implemented within an RKD instead of an RDD setting. For HMTE estimation we require that two additional assumptions hold in addition to those discussed in the previous section. First, 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 HMTE effects estimation possible, for those with missing observations, we follow Kleven et al. (2019) and assign placebo time-since-diagnosis values by randomly drawing values with replacement, from observed individuals who have a time-since diagnosis values. For the analysis, we demean the variable so that ψ_2 represents the effect for the average time since diagnosis. To ensure smooth density of time since diagnosis we present a similar graphic to those in figure 3 but for time since diagnosis in figure 4. There is no clear evidence of a jump or a kink in time since diagnosis at the threshold, however it is worth keeping in mind that for those not diagnosed with diabetes, the time since diagnosis values are placebo values.

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, 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.

8.1 Partner in Household

Table 11 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, increases the probability of consuming vegetables, reduces the probability of smoking, while also increases the probability of drinking. Yet, there is little heterogeneity on the effect of own diabetes diagnosis on any of the own outcomes.

8.2 Time Since Diagnosis

Heterogeneity estimates across time-since-diagnosis are given in Table 12 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. 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, which is precisely what we would expect, given that time since diagnosis for individuals who have not had a diabetes diagnosis is a placebo time since diagnosis, or placebo time since partner's diagnosis.

8.3 Education

Finally, heterogeneity in terms of educational attainment is presented in Table 13. 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 discussed in Section 2.1 and its footnotes, clinical 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.

9 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 status on own lifestyle behaviours, namely exercising, eating habits, smoking and drinking. Exploiting national guidelines around the levels of sugar in the blood and recommendation for annual testing for those above of 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 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 those of 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 seem do not directly confirm these studies we once again note the difference in time-since-diagnosis between studies and suggest that our findings largely 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 reduce 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.

Unfortunately, there are no studies to directly compare our estimated spillover effects onto partners to, albeit 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, comparisons with Fletcher and Marksteiner are harder, given that they investigate the direct and spillover effects of alcoholism treatment, rather than a diabetes diagnosis. Unlike Janssen and Parslow (2021), we find no evidence in favour of a change in alcohol consumption as a result of the diabetes diagnosis, however our results concur with theirs in that both studies find evidence of persistent effects and evidence of a spillover effect in behaviours, albeit for different behaviours. Finally, although again we cannot directly compare our results to Fadlon and Nielsen (2019), both studies 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 on how to induce behavioural changes in terms of diet and alcohol consumption in diabetic patients. From a policy perspective, our findings suggest that benefit evaluation of diabetes interventions needs to be revisited in the presence of substantial spill-over effects, as their current benefit-cost ratio is likely to be substantially underestimated, especially in relation to physical activity and smoking of partners, from a diabetes diagnosis.

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Tables and Figures

Table 1: Descriptive Statistics

	HSE Adult Sample	Blood Sample			Blood and Partner Sample		
		All	Below Kink	Above Kink	All	Below Kink	Above Kink
Observable Characteristics							
Age†	49.52	51.53	49.11	63.91	51.95	50.04	62.19
	(18.72)	*(17.63)*	*(17.29)*	*(13.66)*	*(15.19)*	*(14.84)*	*(12.75)*
Males	0.45	0.46	0.45	0.49	0.48	0.47	0.56
	(0.50)	*(0.50)*	*(0.50)*	*(0.50)*	*(0.50)*	*(0.50)*	*(0.50)*
Any Qualifications	.74	0.76	0.8	0.58	0.78	0.81	0.62
	(0.44)	*(0.43)*	*(0.40)*	*(0.49)*	*(0.42)*	*(0.39)*	*(0.48)*
Degree level education	0.20	0.22	0.23	0.13	0.24	0.25	0.15
	(0.40)	*(0.41)*	*(0.42)*	*(0.34)*	*(0.42)*	*(0.43)*	*(0.35)*
Partner living in household	0.64	0.67	0.67	0.64	–	–	–
	(0.48)	*(0.47)*	*(0.47)*	*(0.48)*			
Household Size†	2.69	2.59	2.68	2.16	2.90	2.96	2.57
	(1.39)	*(1.32)*	*(1.34)*	*(1.15)*	*(1.17)*	*(1.18)*	*(1.05)*
Employed	0.60	0.61	0.65	0.37	0.67	0.71	0.43
	(0.49)	*(0.49)*	*(0.48)*	*(0.48)*	*(0.47)*	*(0.45)*	*(0.50)*
Equivalised Income†	30,457.38	31,732.89	32,834.61	25,894.29	33,227.54	34,392.52	26,659.59
	(27,527.94)	*(27,879.07)*	*(28,212.34)*	*(25,253.62)*	*(26,157.03)*	*(26,439.76)*	*(23,445.57)*
Self-assessed general health (1 = Very Good, 5 = Very Poor)	2.04	1.98	1.89	2.43	1.93	1.85	2.36
	(0.95)	*(0.91)*	*(0.87)*	*(1.00)*	*(0.87)*	*(0.83)*	*(0.97*
Glycated Hemoglobin (HbA1c)	–	5.61	5.39	6.73	5.60	5.39	6.72
		(0.75)	*(0.33)*	*(1.17)*	*(0.73)*	*(0.32)*	*(1.16)*
Stated Behaviours							
Physical Activity †	0.44	0.46	0.5	0.26	0.46	0.48	0.27
	(0.50)	*(0.50)*	*(0.50)*	*(0.44)*	*(0.50)*	*(0.50)*	*(0.44)*
Vegetable Consumption	0.53	0.53	0.53	0.54	0.54	0.54	0.55
	(0.50)	*(0.50)*	*(0.50)*	*(0.50)*	*(0.50)*	*(0.50)*	*(0.50)*
Fruit Consumption	0.61	0.62	0.61	0.67	0.63	0.63	0.67
	(0.49)	*(0.48)*	*(0.49)*	*(0.47)*	*(0.48)*	*(0.48)*	*(0.47)*
Currently a drinker	0.85	0.89	0.90	0.82	0.90	0.91	0.84
	(0.36)	*(0.32)*	*(0.30)*	*(0.38)*	*(0.30)*	*(0.29)*	*(0.37)*
Currently a smoker	0.21	0.19	0.19	0.18	0.16	0.16	0.16
	(0.40)	*(0.39)*	*(0.39)*	*(0.38)*	*(0.37)*	*(0.36)*	*(0.37)*
Ever a drinker	0.90	0.93	0.94	0.90	0.94	0.94	0.91
	(0.30)	*(0.25)*	*(0.24)*	*(0.29)*	*(0.24)*	*(0.23)*	*(0.29)*
Ever a smoker	0.58	0.59	0.58	0.63	0.58	0.57	0.62
	(0.49)	*(0.49)*	*(0.49)*	*(0.48)*	*(0.49)*	*(0.50)*	*(0.49)*
Number of Observations	121,849	53,146	44,448	8,698	32,910	27,740	5,170

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 whom have a full set of non-missing observations for our control variables, 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
(a)					
1^{st} Stage	0.675***	0.738***	0.738***	0.729***	0.730***
	(0.0285)	(0.0190)	(0.0190)	(0.0177)	(0.0186)
Effect of Own Diabetes	0.203***	0.0376	0.0650	-0.248***	0.00843
	(0.0688)	(0.0480)	(0.0454)	(0.0339)	(0.0248)
Obs.	20641	39666	39690	44828	41686
(b)					
1^{st} Stage	0.678***	0.743***	0.743***	0.730***	0.738***
	(0.0404)	(0.0270)	(0.0270)	(0.0254)	(0.0266)
Partner's Diabetes	0.235**	0.0166	-0.0907	-0.105**	0.0372
	(0.0967)	(0.0666)	(0.0626)	(0.0421)	(0.0315)
Obs.	10581	20013	20015	22610	20941
(c)					
Own 1^{st} Stage	0.525***	0.600***	0.600***	0.592***	0.595***
	(0.0402)	(0.0274)	(0.0274)	(0.0250)	(0.0263)
Own Diabetes	0.214*	0.103	0.187**	-0.237***	0.0753*
	(0.116)	(0.0783)	(0.0753)	(0.0517)	(0.0430)
Partner's 1^{st} Stage	0.525***	0.600***	0.600***	0.592***	0.605***
	(0.0402)	(0.0274)	(0.0274)	(0.0250)	(0.0261)
Partner's Diabetes	0.244**	0.0508	-0.121	-0.0526	0.0453
	(0.121)	(0.0782)	(0.0745)	(0.0503)	(0.0379)
Obs.	8064	15055	15055	17019	15871

Notes: Coefficients are estimated using a quadratic specification each side of the kink point, equally weighted observations, and with bounds of 4.0 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, a partner lives in the household, and has degree level education. Panels (b) and (c) additionally include the same set of controls for individual j, but excluding whether partner lives in the household. *** denotes P-value of 0.01 or less , ** denotes P-value of 0.05 or less, * denotes P-value of 0.10 or less

Table 3: Fuzzy RKD estimates of change in own behaviour as a result own and partner's

	Whether taking				
	Anti-diabetic medication	Antibiotic medication	Anti-depressant medication	Statins	Whether even been in paid employment
(a)					
1^{st} Stage	0.776***	0.776***	0.776***	0.753***	0.747***
	(0.0325)	(0.0325)	(0.0325)	(0.0199)	(0.0240)
Effect of Own Diabetes	0.883***	0.000726	-0.00786	-0.0207*	0.0374
	(0.0298)	(0.0271)	(0.0553)	(0.0109)	(0.0310)
Obs.	12138	12138	12138	34638	19546
(b)					
1^{st} Stage	0.826***	0.826***	0.826***	0.774***	0.749***
	(0.0483)	(0.0483)	(0.0483)	(0.0291)	(0.0346)
Partner's Diabetes	0.00881	0.0281	-0.0265	-0.000439	0.0694
	(0.0717)	(0.0292)	(0.0691)	(0.0138)	(0.0431)
Obs.	5604	5604	5604	16528	9833

Notes: Coefficients are estimated using a quadratic specification each side of the kink point, equally weighted observations, and with bounds of 4.0 on the right hand tail, and 2.0 on the left hand side for panels (a) and (b). 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, a partner lives in the household, and has degree level education. Panel (b) additionally include the same set of controls for individual j, but excluding whether partner lives in the household. *** 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: Third Stage Estimates of the Recursive Model estimating the causal pathways

	Exercise	Currently a Smoker
Partner's Behaviour (y_i)	0.411***	0.379***
	(0.0146)	(0.0136)
Partner's Diagnosis ($EverD_i$)	0.149*	-0.0108
	(0.0821)	(0.0389)
Observations	10,581	22,607

Notes: Coefficients are estimated using a quadratic specification each side of the kink point, equally weighted observations, and with bounds of 4.0 on the right hand tail, and 2.0 on the left hand side. Parentheses includes cluster-robust standard errors, clustered at the household level. Each specification includes the following controls: Age, and dummies for whether individual i is male, a partner lives in the household, and has degree level education, as well as the same set of controls for individual j.
*** 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 diabetes diagnosis by whether individual has a partner

	Exercise	Vegetable Consumption	Fruit Consumption	Currently a Smoker	Currently a drinker
Own Diabetes	0.141	0.0548	-0.0221	-0.227***	0.00394
	(0.120)	(0.0833)	(0.0793)	(0.0624)	(0.0464)
Partner in HH	-0.00825	0.0403**	0.0195	-0.0742***	0.0293***
	(0.0241)	(0.0161)	(0.0152)	(0.0115)	(0.00805)
Own Diabetes x Partner in HH	0.101	-0.0261	0.122	-0.0298	0.0123
	(0.146)	(0.102)	(0.0971)	(0.0740)	(0.0547)
Obs.	20641	39666	39690	44828	41686

Coefficients are estimated using a quadratic specification each side of the kink point, equally weighted observations, and with bounds of 4.0 on the right hand tail, and 2.0 on the left hand side. Parentheses includes cluster-robust standard errors, clustered at the household level. Each specification includes the following controls: Age, and dummies for whether individual i is male, a partner lives in the household, 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 6: 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
(a)					
Own Diabetes	0.201***	0.0322	0.0694	-0.252***	0.0137
	(0.0693)	(0.0482)	(0.0456)	(0.0342)	(0.0250)
Time Since Own Diagnosis (TSoD)	0.000114	-0.000173	0.000523	-0.000126	0.000829***
	(0.00112)	(0.000727)	(0.000698)	(0.000489)	(0.000308)
Own Diabetes x TSoD	-0.00337	0.00285	-0.00363	0.00104	-0.00544**
	(0.00726)	(0.00466)	(0.00454)	(0.00325)	(0.00239)
Obs.	20641	39666	39690	44828	41686
(b)					
Partner Diabetes	0.237**	0.0108	-0.0761	-0.104**	0.0410
	(0.0988)	(0.0672)	(0.0631)	(0.0420)	(0.0312)
Time Since Partner Diagnosis (TSpD)	-0.0000979	-0.000407	0.000876	0.000486	-0.000129
	(0.00166)	(0.00106)	(0.00106)	(0.000621)	(0.000539)
Partner Diabetes x TSpD	0.00428	0.00510	-0.0119*	-0.00285	-0.00257
	(0.00960)	(0.00697)	(0.00714)	(0.00468)	(0.00401)
Obs.	10563	19983	19985	22580	20924

Coefficients are estimated using a quadratic specification each side of the kink point, equally weighted observations, and with bounds of 4.0 on the right hand tail, and 2.0 on the left hand side. Parentheses includes cluster-robust standard errors, clustered at the household level. Each specification includes the following controls: Age, and dummies for whether individual i is male, a partner lives in the household, and has degree level education. Panel (b) additionally include the same set of controls for individual j, but excluding whether partner lives in the household. *** denotes P-value of 0.01 or less , ** denotes P-value of 0.05 or less, * denotes P-value of 0.10 or less

Table 7: Heterogeneous fuzzy 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
(a)					
Own Diabetes	0.266***	0.0507	0.109**	-0.242***	0.00176
	(0.0757)	(0.0535)	(0.0509)	(0.0392)	(0.0286)
Own College Degree (OCD)	0.249***	0.108***	0.136***	-0.124***	0.0176**
	(0.0303)	(0.0191)	(0.0176)	(0.0115)	(0.00782)
Own Diabetes x OCD	-0.368*	-0.0301	-0.234**	0.0276	0.0562
	(0.192)	(0.124)	(0.114)	(0.0754)	(0.0498)
Observations	20641	39666	39690	44828	41686
(b)					
Partner Diabetes	0.256**	0.0402	-0.0606	-0.0921*	0.0427
	(0.104)	(0.0717)	(0.0677)	(0.0477)	(0.0339)
Own College Degree (OCD)	0.173***	0.0768***	0.108***	-0.0804***	0.0239**
	(0.0432)	(0.0275)	(0.0256)	(0.0128)	(0.0111)
Partner Diabetes x OCD	0.0114	-0.158	-0.134	-0.0703	0.0222
	(0.284)	(0.219)	(0.202)	(0.104)	(0.0945)
Observations	10581	20013	20015	22610	20941

Coefficients are estimated using a quadratic specification each side of the kink point, equally weighted observations, and with bounds of 4.0 on the right hand tail, and 2.0 on the left hand side. Parentheses includes cluster-robust standard errors, clustered at the household level. Each specification includes the following controls: Age, and dummies for whether individual i is male, a partner lives in the household, and has degree level education. Panel (b) additionally include the same set of controls for individual j, but excluding whether partner lives in the household. *** denotes P-value of 0.01 or less , ** denotes P-value of 0.05 or less, * denotes P-value of 0.10 or less

Table 8: 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
(a)					
1^{st} Stage	0.675***	0.738***	0.738***	0.729***	0.730***
	(0.0285)	(0.0190)	(0.0190)	(0.0177)	(0.0186)
Effect of Own Diabetes	0.203***	0.0376	0.0650	-0.248***	0.00843
	(0.0688)	(0.0480)	(0.0454)	(0.0339)	(0.0248)
Obs.	20641	39666	39690	44828	41686
(b)					
1^{st} Stage	0.678***	0.743***	0.743***	0.730***	0.738***
	(0.0404)	(0.0270)	(0.0270)	(0.0254)	(0.0266)
Partner's Diabetes	0.235**	0.0166	-0.0907	-0.105**	0.0372
	(0.0967)	(0.0666)	(0.0626)	(0.0421)	(0.0315)
Obs.	10581	20013	20015	22610	20941
(c)					
Own 1^{st} Stage	0.525***	0.600***	0.600***	0.592***	0.595***
	(0.0402)	(0.0274)	(0.0274)	(0.0250)	(0.0263)
Own Diabetes	0.214*	0.103	0.187**	-0.237***	0.0753*
	(0.116)	(0.0783)	(0.0753)	(0.0517)	(0.0430)
Partner's 1^{st} Stage	0.525***	0.600***	0.600***	0.592***	0.605***
	(0.0402)	(0.0274)	(0.0274)	(0.0250)	(0.0261)
Partner's Diabetes	0.244**	0.0508	-0.121	-0.0526	0.0453
	(0.121)	(0.0782)	(0.0745)	(0.0503)	(0.0379)
Obs.	8064	15055	15055	17019	15871

Notes: Coefficients are estimated using a quadratic specification each side of the kink point, equally weighted observations, and with bounds of 4.0 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, a partner lives in the household, and has degree level education. Panels (b) and (c) additionally include the same set of controls for individual j, but excluding whether partner lives in the household. *** 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 9: Fuzzy RKD estimates of change in own behaviour as a result own and partner's

	Whether taking				Whether even been in paid employment
	Anti-diabetic medication	Antibiotic medication	Anti-depressant medication	Statins	
(a)					
1^{st} Stage	0.776***	0.776***	0.776***	0.753***	0.747***
	(0.0325)	(0.0325)	(0.0325)	(0.0199)	(0.0240)
Effect of Own Diabetes	0.883***	0.000726	-0.00786	-0.0207*	0.0374
	(0.0298)	(0.0271)	(0.0553)	(0.0109)	(0.0310)
Obs.	12138	12138	12138	34638	19546
(b)					
1^{st} Stage	0.826***	0.826***	0.826***	0.774***	0.749***
	(0.0483)	(0.0483)	(0.0483)	(0.0291)	(0.0346)
Partner's Diabetes	0.00881	0.0281	-0.0265	-0.000439	0.0694
	(0.0717)	(0.0292)	(0.0691)	(0.0138)	(0.0431)
Obs.	5604	5604	5604	16528	9833

Notes: Coefficients are estimated using a quadratic specification each side of the kink point, equally weighted observations, and with bounds of 4.0 on the right hand tail, and 2.0 on the left hand side for panels (a) and (b). 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, a partner lives in the household, and has degree level education. Panel (b) additionally include the same set of controls for individual j, but excluding whether partner lives in the household. *** 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 10: Third Stage Estimates of the Recursive Model estimating the causal pathways

	Exercise	Currently a Smoker
Partner's Behaviour (y_i)	0.411***	0.379***
	(0.0146)	(0.0136)
Partner's Diagnosis ($EverD_i$)	0.149*	-0.0108
	(0.0821)	(0.0389)
Observations	10,581	22,607

Notes: Coefficients are estimated using a quadratic specification each side of the kink point, equally weighted observations, and with bounds of 4.0 on the right hand tail, and 2.0 on the left hand side. Parentheses includes cluster-robust standard errors, clustered at the household level. Each specification includes the following controls: Age, and dummies for whether individual i is male, a partner lives in the household, and has degree level education, as well as the same set of controls for individual j.
*** 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 11: 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	0.141	0.0548	-0.0221	-0.227***	0.00394
	(0.120)	(0.0833)	(0.0793)	(0.0624)	(0.0464)
Partner in HH	-0.00825	0.0403**	0.0195	-0.0742***	0.0293***
	(0.0241)	(0.0161)	(0.0152)	(0.0115)	(0.00805)
Own Diabetes x Partner in HH	0.101	-0.0261	0.122	-0.0298	0.0123
	(0.146)	(0.102)	(0.0971)	(0.0740)	(0.0547)
Obs.	20641	39666	39690	44828	41686

Coefficients are estimated using a quadratic specification each side of the kink point, equally weighted observations, and with bounds of 4.0 on the right hand tail, and 2.0 on the left hand side. Parentheses includes cluster-robust standard errors, clustered at the household level. Each specification includes the following controls: Age, and dummies for whether individual i is male, a partner lives in the household, 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 12: 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
(a)					
Own Diabetes	0.201***	0.0322	0.0694	-0.252***	0.0137
	(0.0693)	(0.0482)	(0.0456)	(0.0342)	(0.0250)
Time Since Own Diagnosis (TSoD)	0.000114	-0.000173	0.000523	-0.000126	0.000829***
	(0.00112)	(0.000727)	(0.000698)	(0.000489)	(0.000308)
Own Diabetes x TSoD	-0.00337	0.00285	-0.00363	0.00104	-0.00544**
	(0.00726)	(0.00466)	(0.00454)	(0.00325)	(0.00239)
Obs.	20641	39666	39690	44828	41686
(b)					
Partner Diabetes	0.237**	0.0108	-0.0761	-0.104**	0.0410
	(0.0988)	(0.0672)	(0.0631)	(0.0420)	(0.0312)
Time Since Partner Diagnosis (TSpD)	-0.0000979	-0.000407	0.000876	0.000486	-0.000129
	(0.00166)	(0.00106)	(0.00106)	(0.000621)	(0.000539)
Partner Diabetes x TSpD	0.00428	0.00510	-0.0119*	-0.00285	-0.00257
	(0.00960)	(0.00697)	(0.00714)	(0.00468)	(0.00401)
Obs.	10563	19983	19985	22580	20924

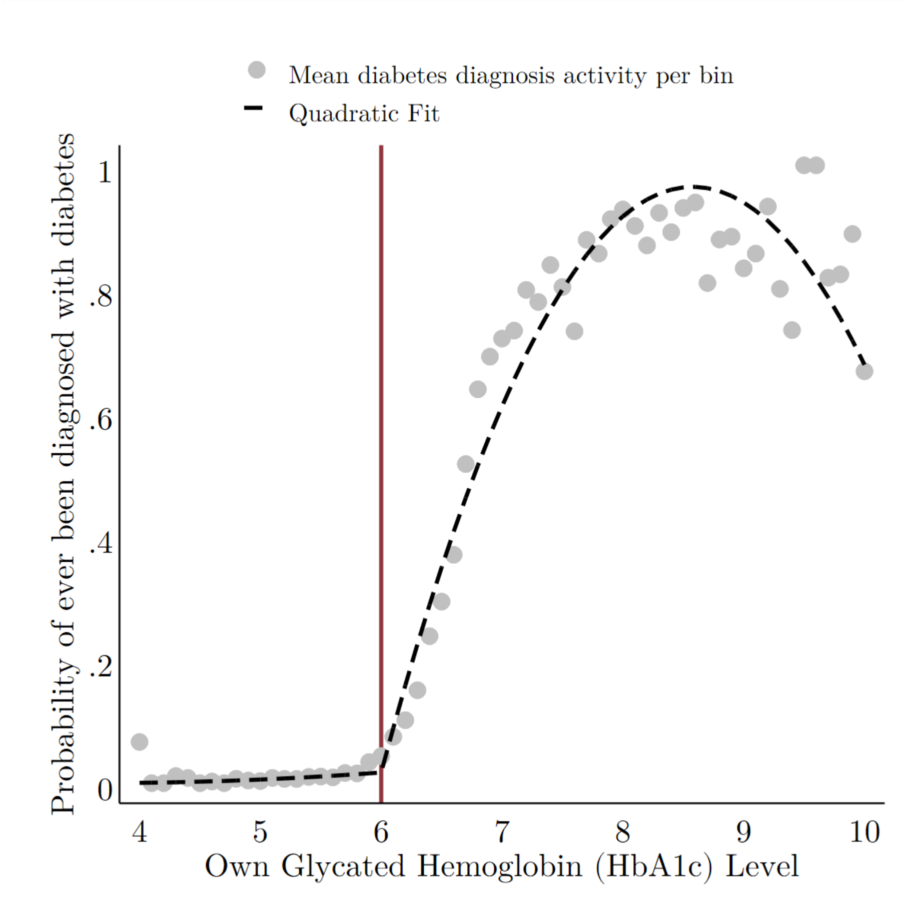
Coefficients are estimated using a quadratic specification each side of the kink point, equally weighted observations, and with bounds of 4.0 on the right hand tail, and 2.0 on the left hand side. Parentheses includes cluster-robust standard errors, clustered at the household level. Each specification includes the following controls: Age, and dummies for whether individual i is male, a partner lives in the household, and has degree level education. Panel (b) additionally include the same set of controls for individual j, but excluding whether partner lives in the household. *** 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 13: 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
(a)					
Own Diabetes	0.266***	0.0507	0.109**	-0.242***	0.00176
	(0.0757)	(0.0535)	(0.0509)	(0.0392)	(0.0286)
Own College Degree (OCD)	0.249***	0.108***	0.136***	-0.124***	0.0176**
	(0.0303)	(0.0191)	(0.0176)	(0.0115)	(0.00782)
Own Diabetes x OCD	-0.368*	-0.0301	-0.234**	0.0276	0.0562
	(0.192)	(0.124)	(0.114)	(0.0754)	(0.0498)
Observations	20641	39666	39690	44828	41686
(b)					
Partner Diabetes	0.256**	0.0402	-0.0606	-0.0921*	0.0427
	(0.104)	(0.0717)	(0.0677)	(0.0477)	(0.0339)
Own College Degree (OCD)	0.173***	0.0768***	0.108***	-0.0804***	0.0239**
	(0.0432)	(0.0275)	(0.0256)	(0.0128)	(0.0111)
Partner Diabetes x OCD	0.0114	-0.158	-0.134	-0.0703	0.0222
	(0.284)	(0.219)	(0.202)	(0.104)	(0.0945)
Observations	10581	20013	20015	22610	20941

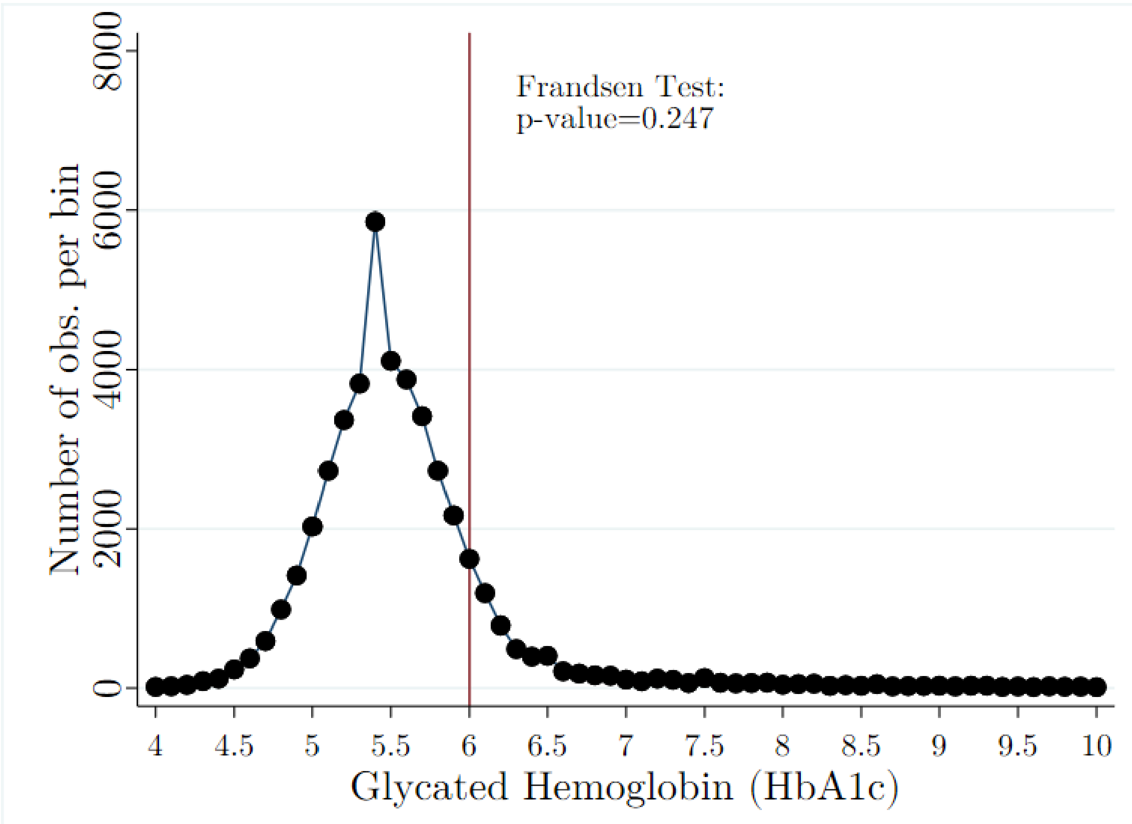
Coefficients are estimated using a quadratic specification each side of the kink point, equally weighted observations, and with bounds of 4.0 on the right hand tail, and 2.0 on the left hand side. Parentheses includes cluster-robust standard errors, clustered at the household level. Each specification includes the following controls: Age, and dummies for whether individual i is male, a partner lives in the household, and has degree level education. Panel (b) additionally include the same set of controls for individual j, but excluding whether partner lives in the household. *** 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



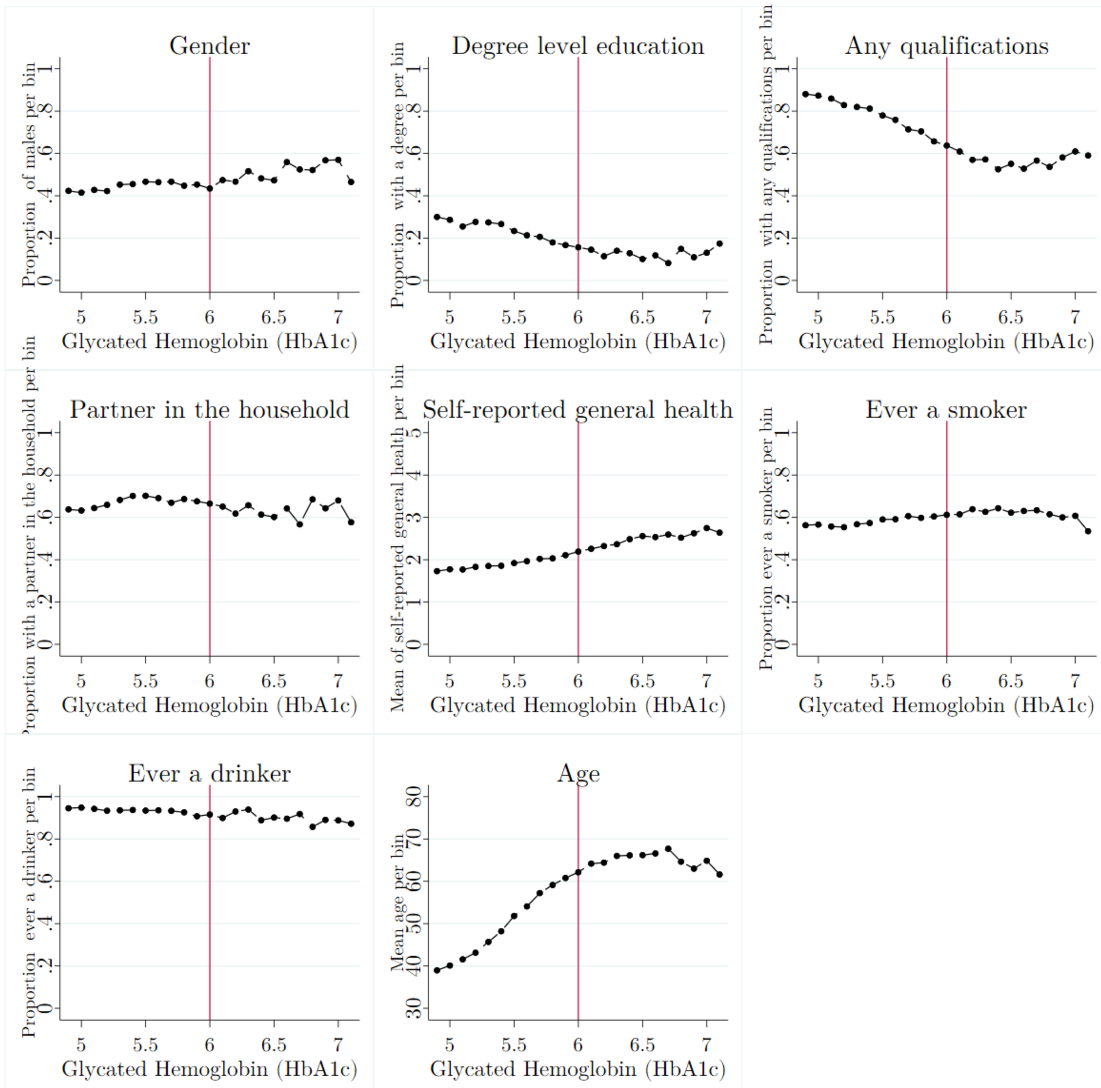
NOTE: Mean of the probability of ever being diagnosed with diabetes per bin. Bin width of 0.1 for glycated hemoglobin levels between 4 and 10. Quadratic fit is separately estimated for the left and right hand sides of the kink. Red line represents the kink point, where glycated hemoglobin is a value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

Figure 2: Smooth Density of the Assignment Variable



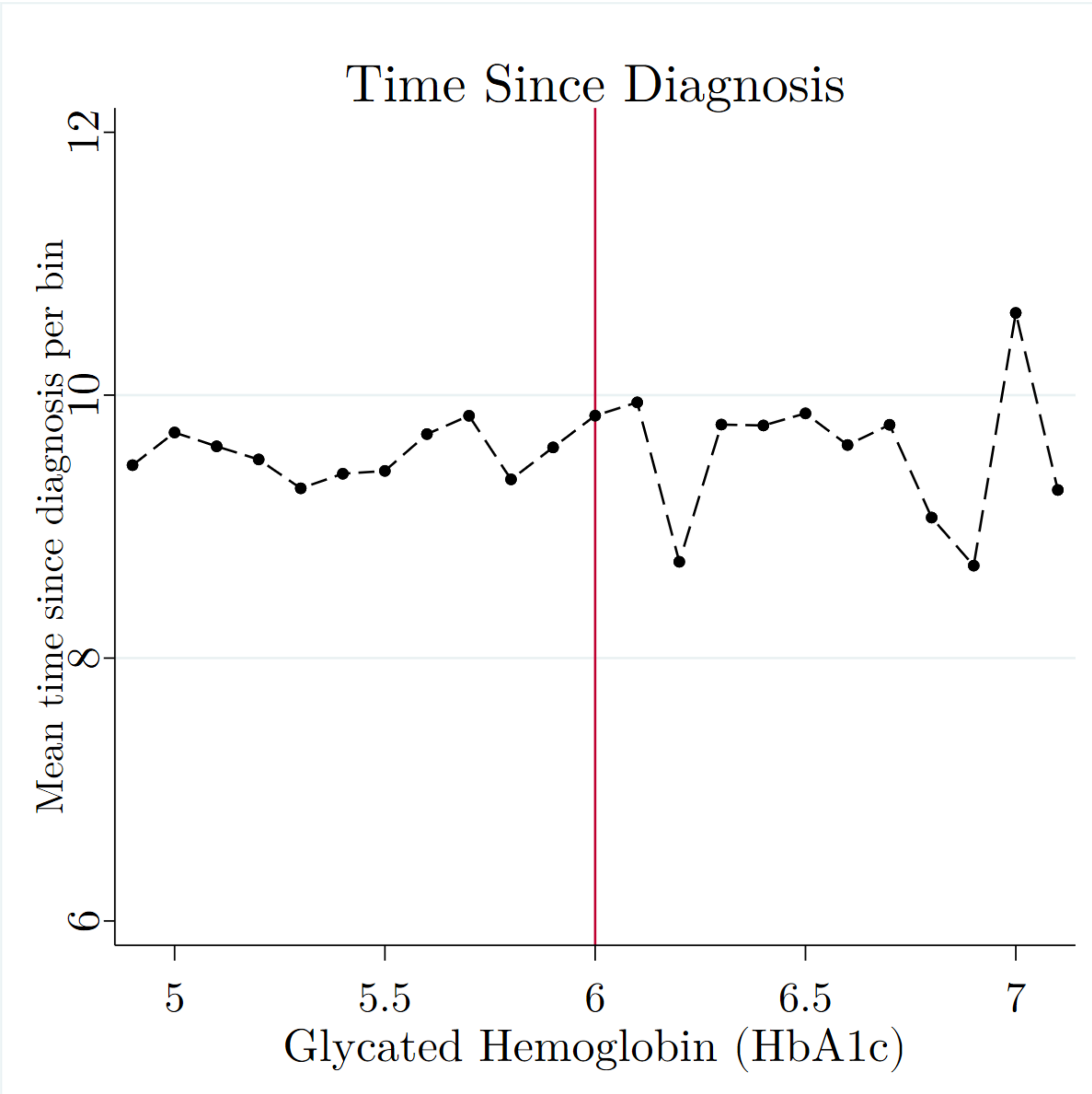
NOTE: Number of observations per bin. Bin width of 0.1 for glycated hemoglobin levels between 4 and 10. Graph also shows Frandsen (2017) discontinuity statistic.

Figure 3: Predetermined variables



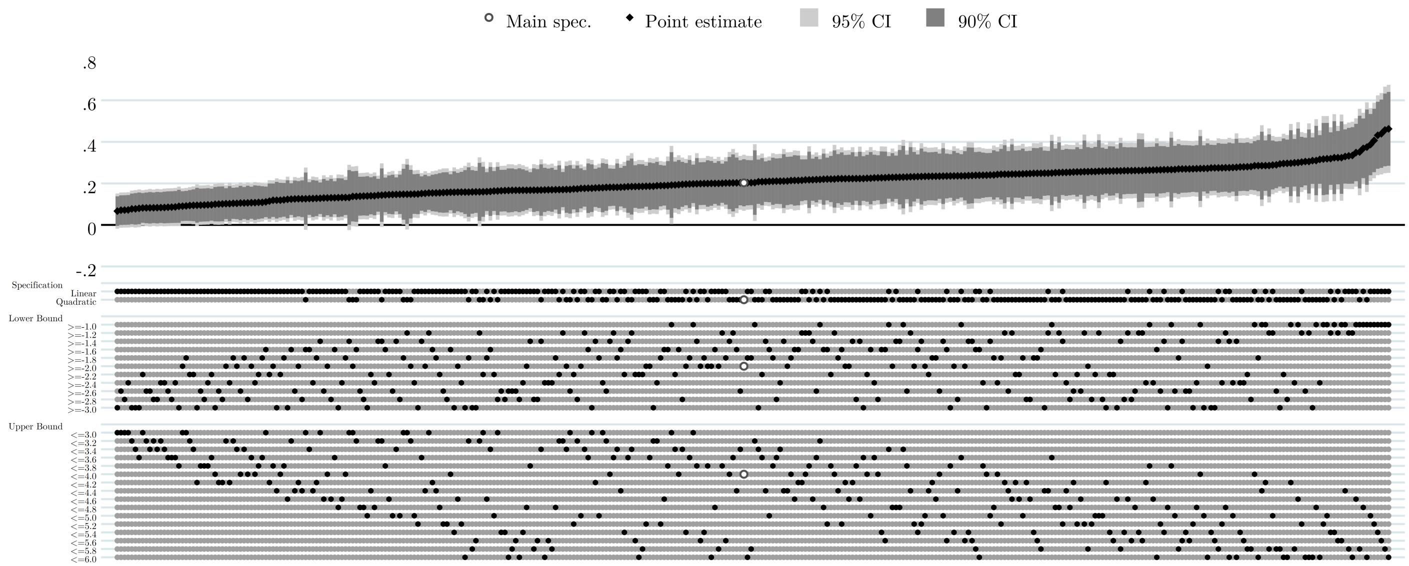
NOTE: Graphical representation of the mean of each predetermined variable by glycated hemoglobin (HbA1c) level. Each graph shows the mean of the predetermined variable per bin, with a bin width of 0.1. Predetermined variables included are gender, ethnicity, degree level education, any qualifications,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



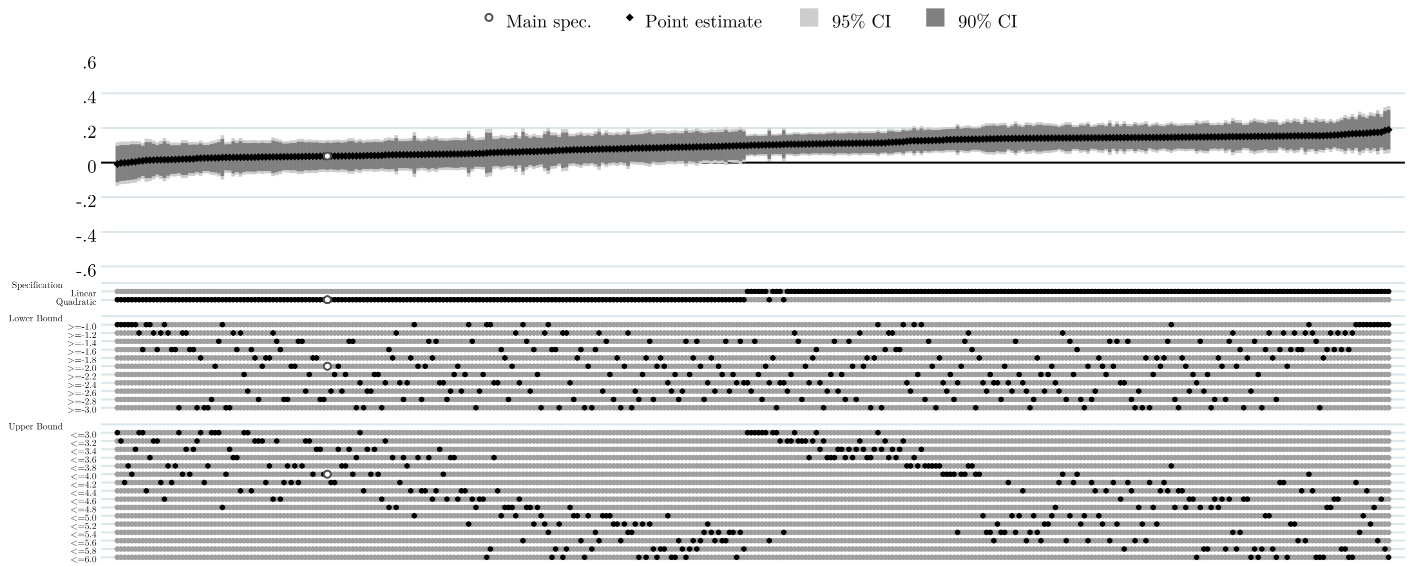
NOTE: Graphical representation of the mean of time since diabetes diagnosis by glycated hemoglobin (HbA1c) level. Graph shows the mean of the time since diagnosis per bin, with a bin width of 0.1. For individuals whom have never been diagnosed as having diabetes, they are assigned a placebo time since 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



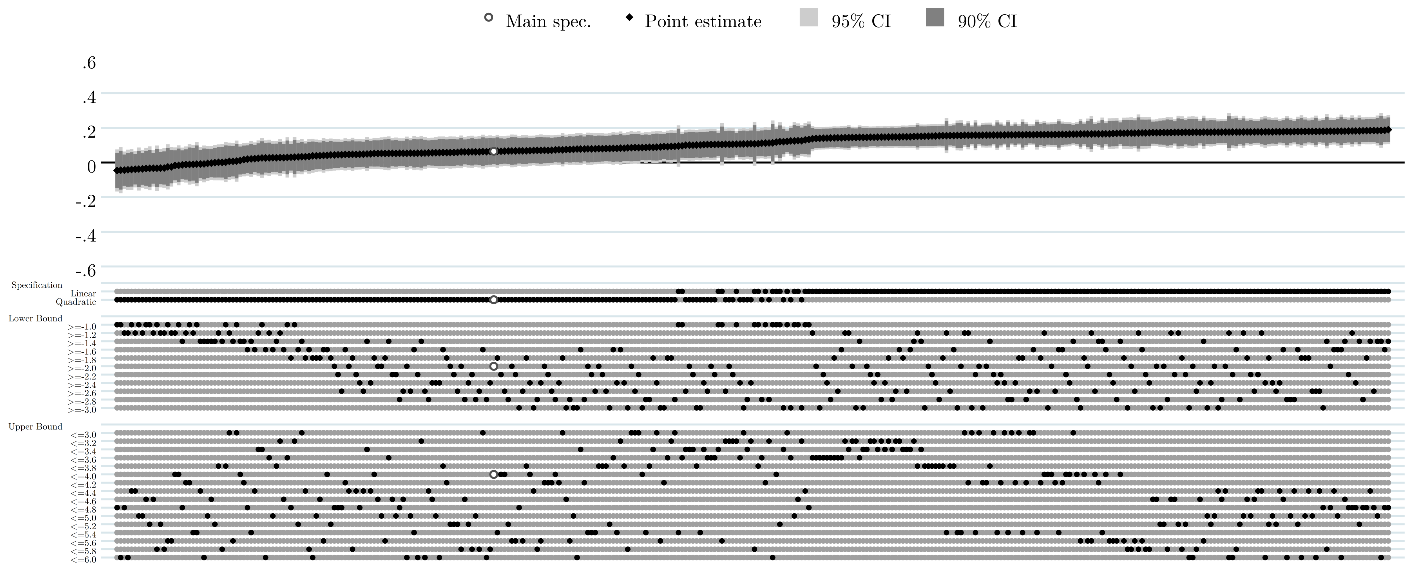
NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents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



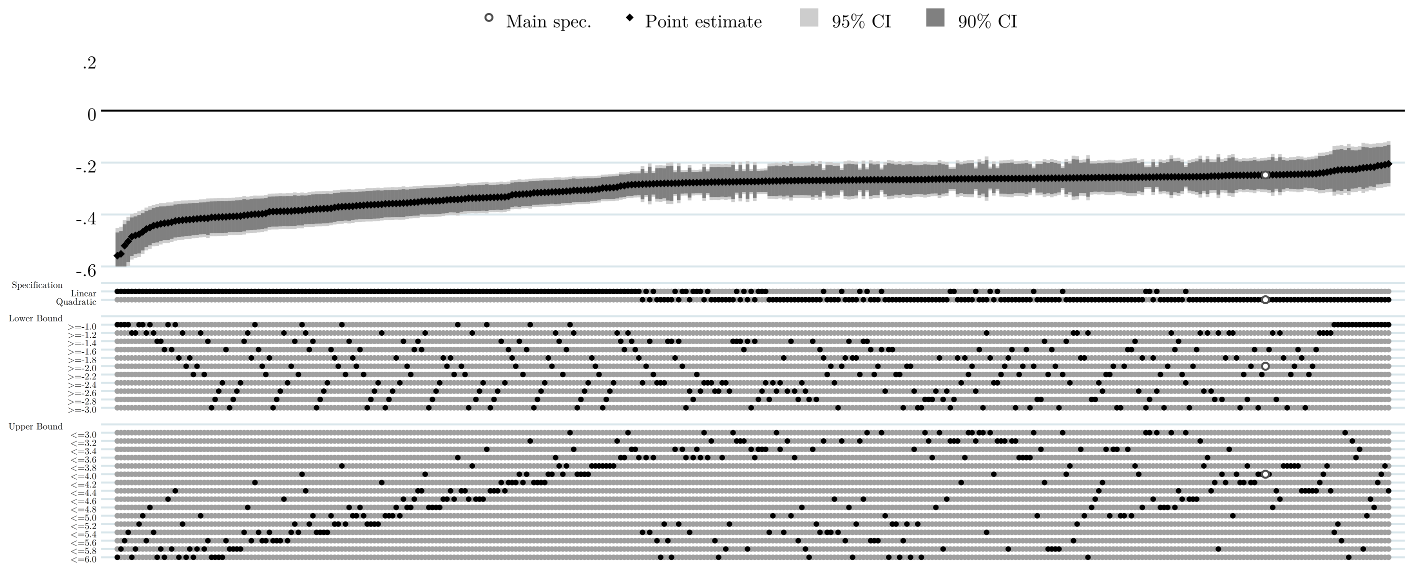
NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents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



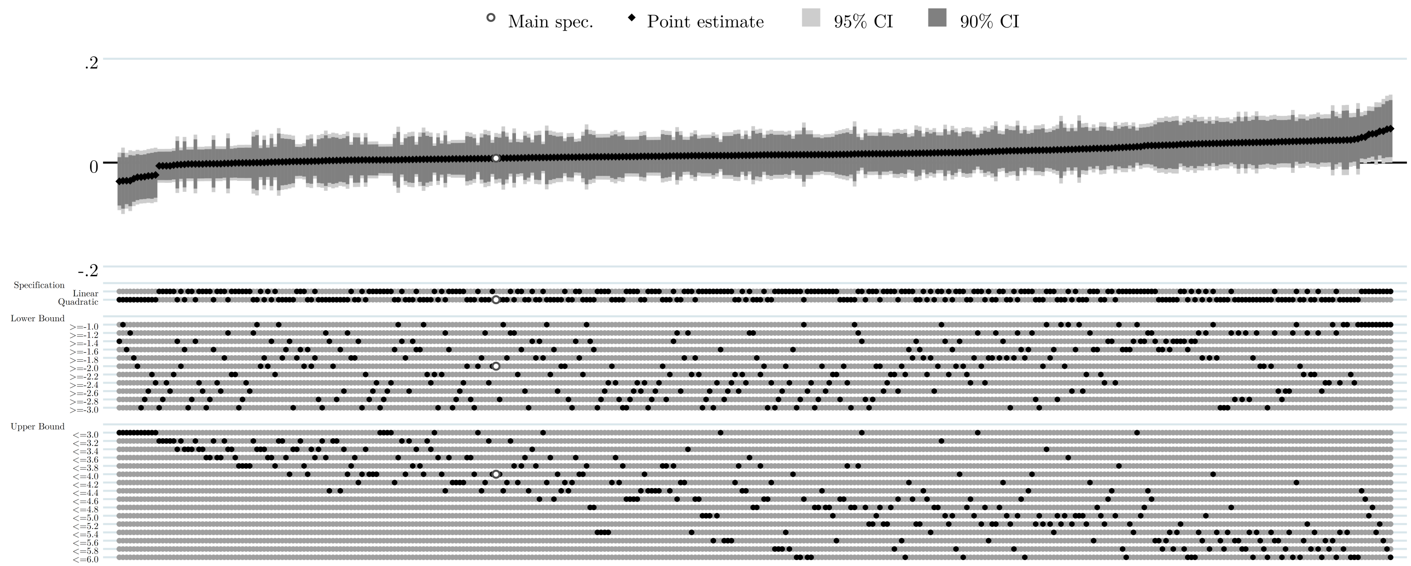
NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents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



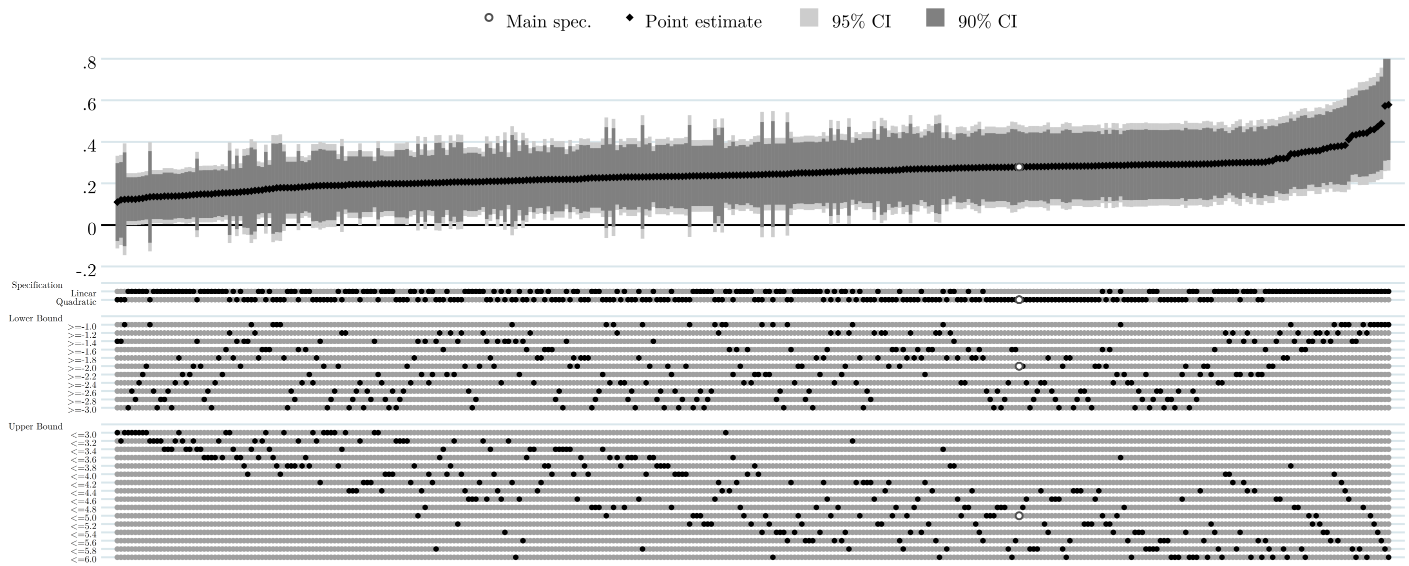
NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents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



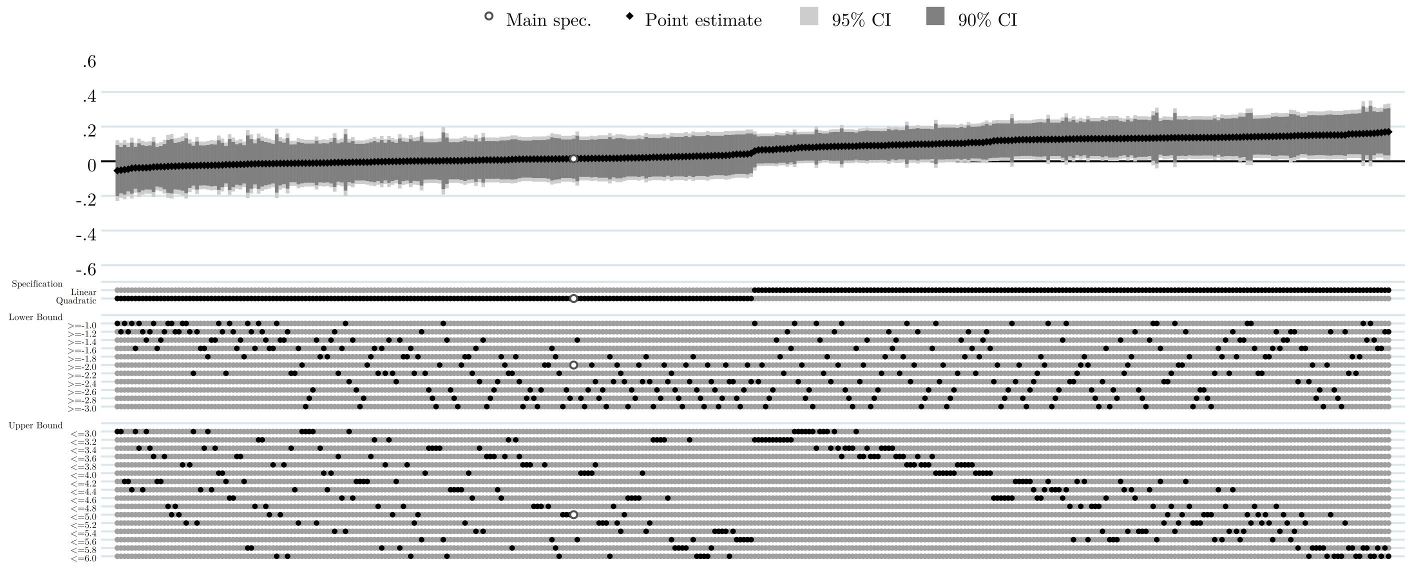
NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents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



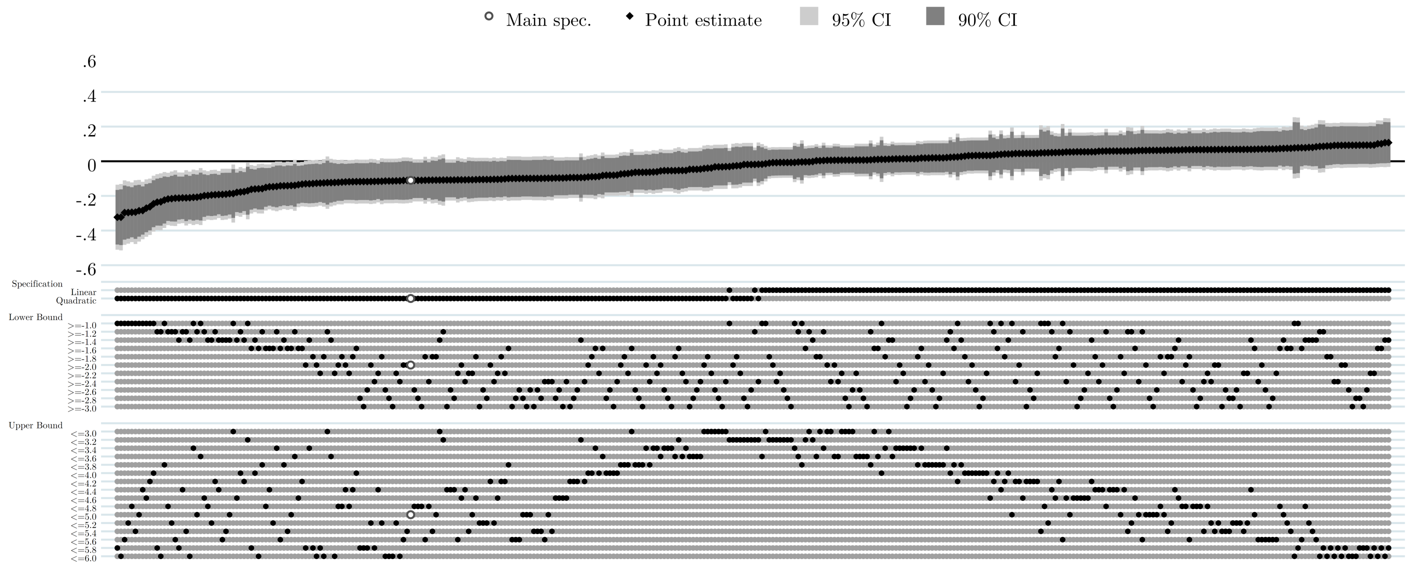
NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents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



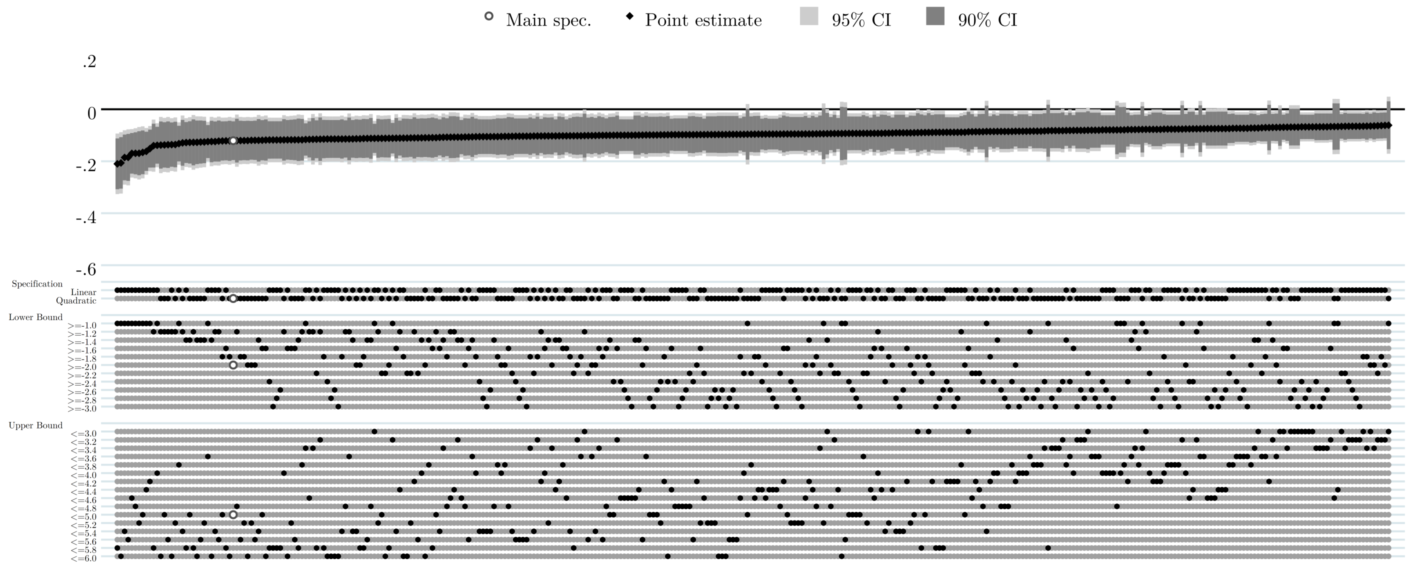
NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents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



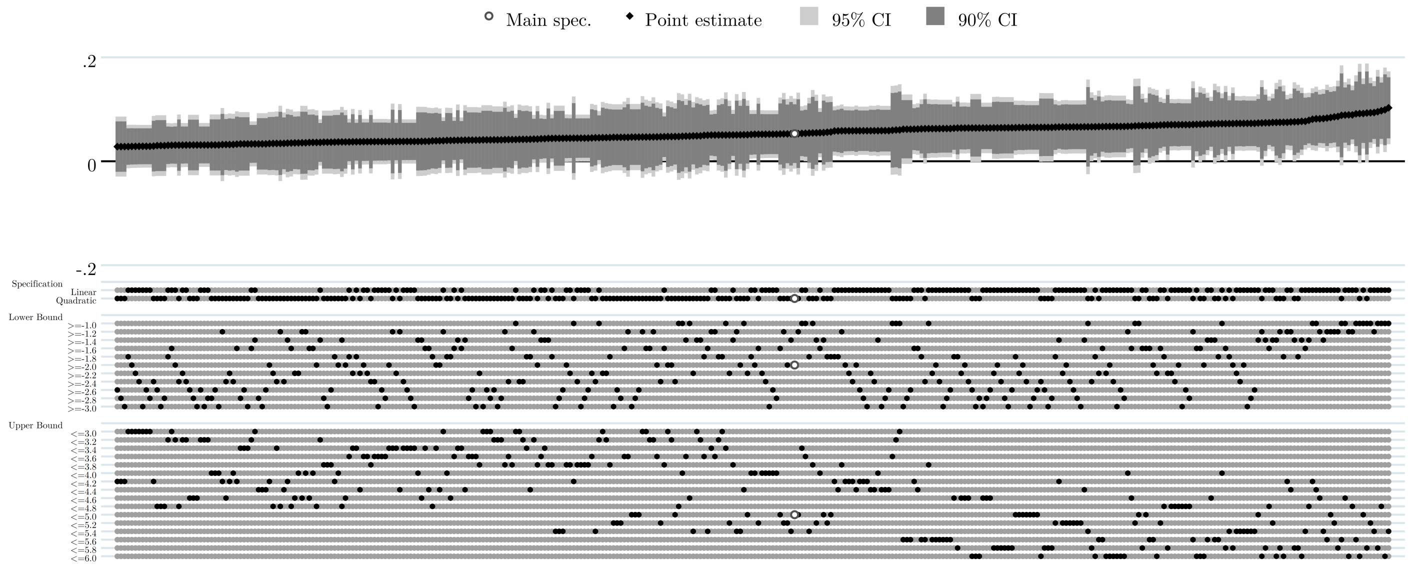
NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents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



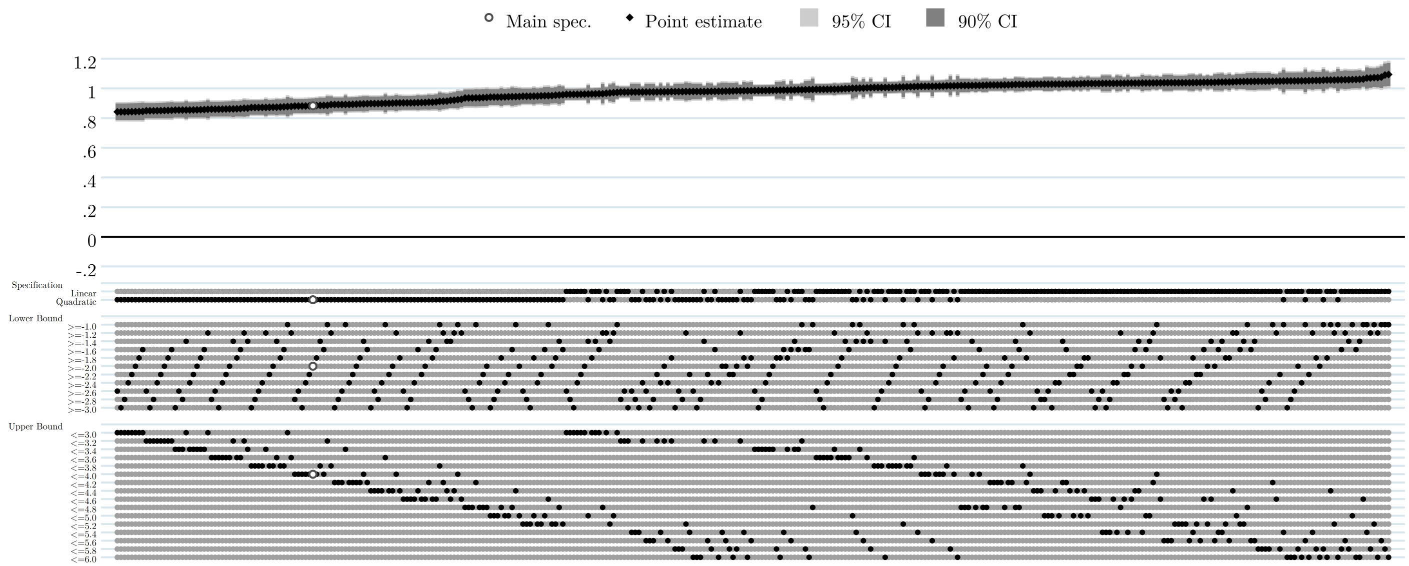
NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents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



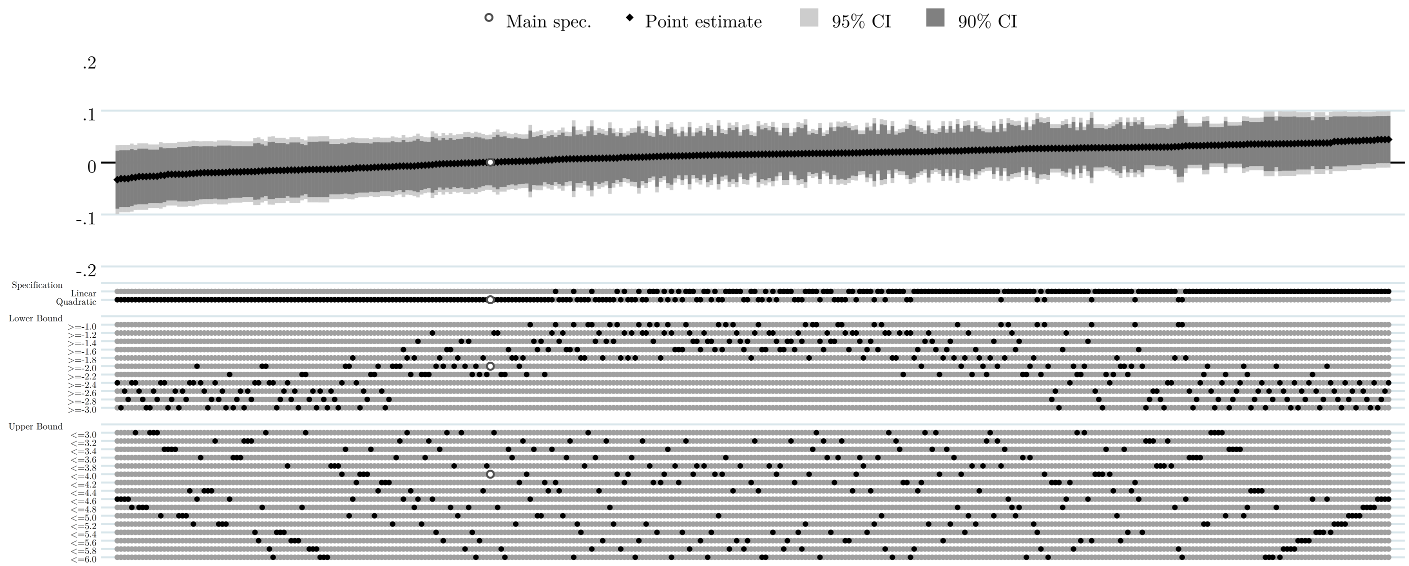
NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents 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 15: Sensitivity to alternative bandwidths and polynomials - Whether taking Anti-diabetic medication



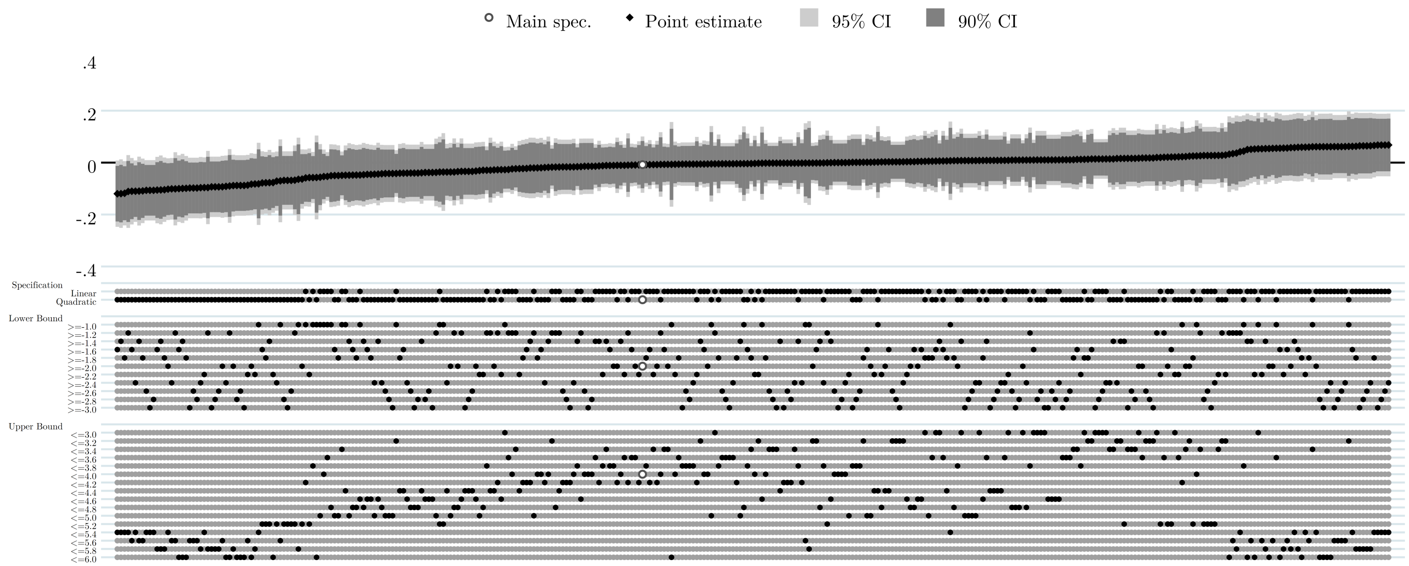
NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents 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 16: Sensitivity to alternative bandwidths and polynomials - Whether taking Antibiotic medication



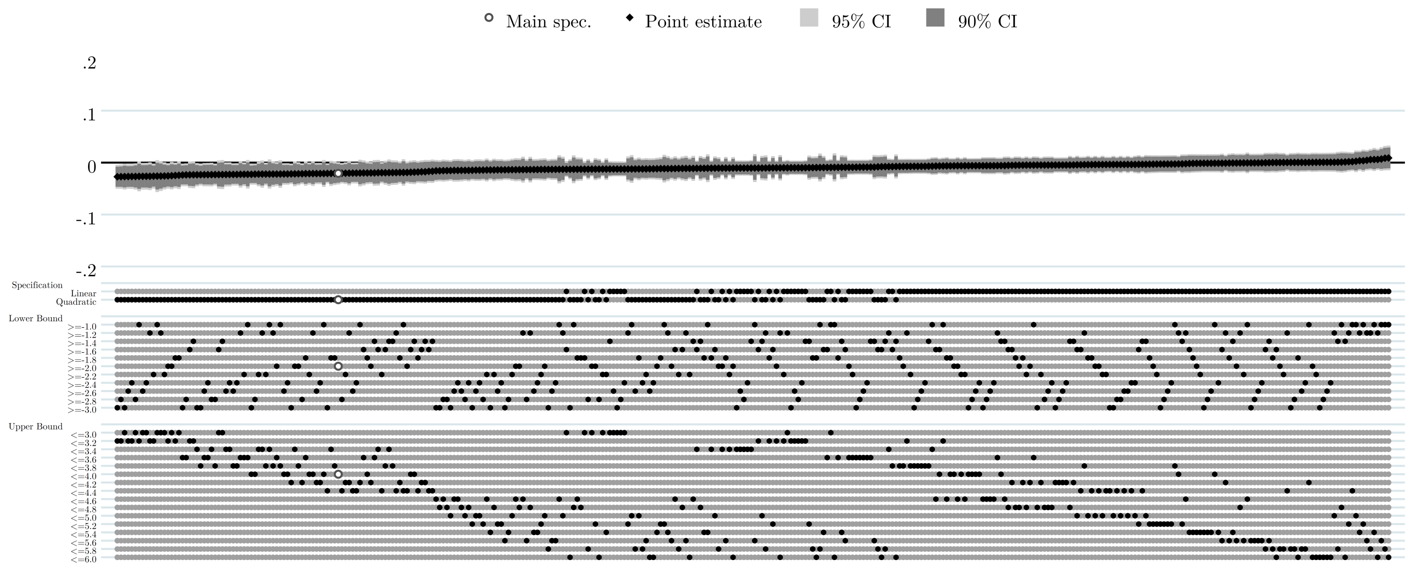
NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents 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 17: Sensitivity to alternative bandwidths and polynomials - Whether taking Anti-depressant medication



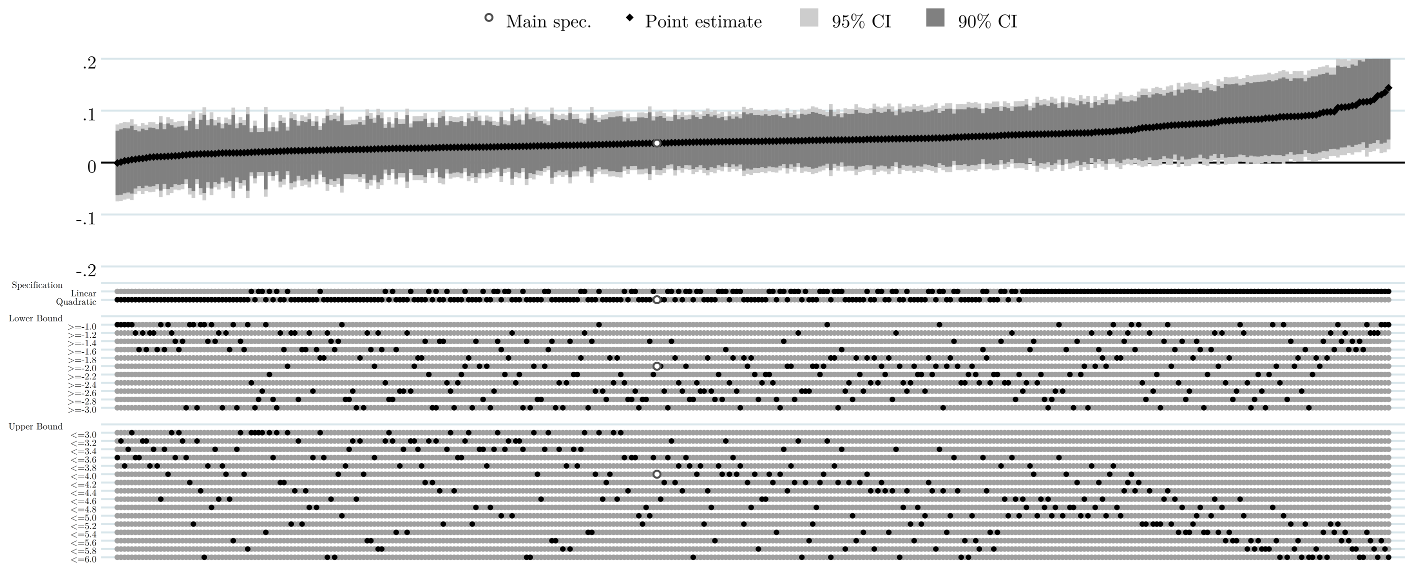
NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents 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 18: Sensitivity to alternative bandwidths and polynomials - Whether taking Statins



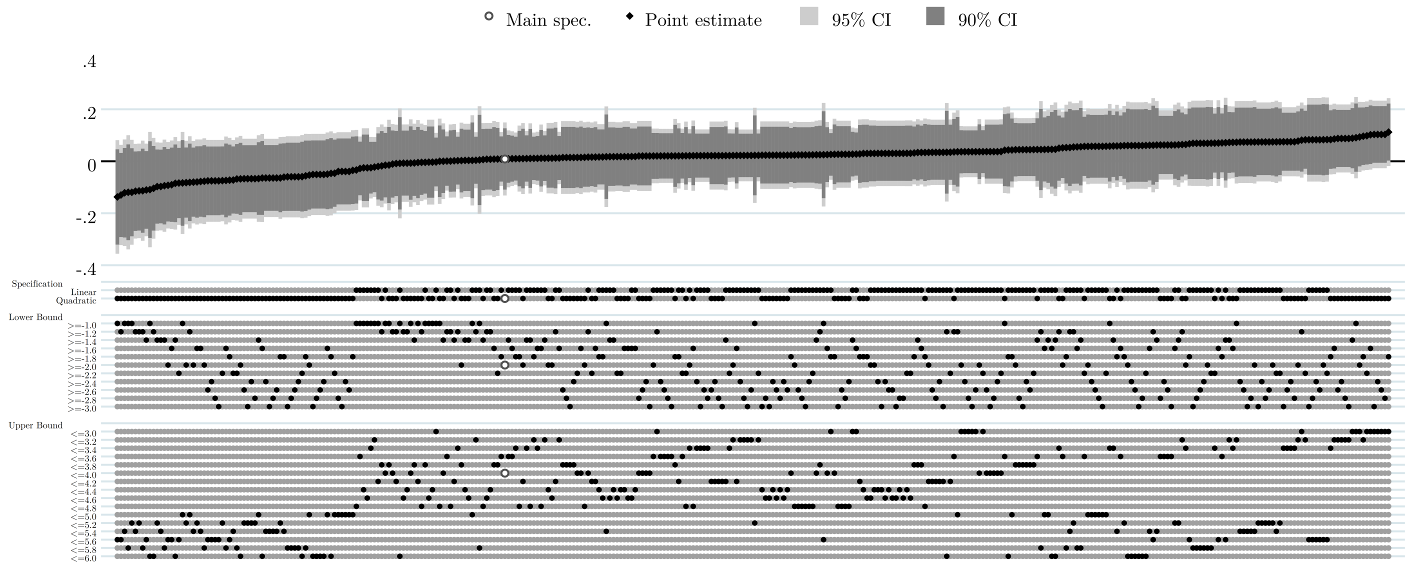
NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents 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 19: Sensitivity to alternative bandwidths and polynomials - Whether ever had a job



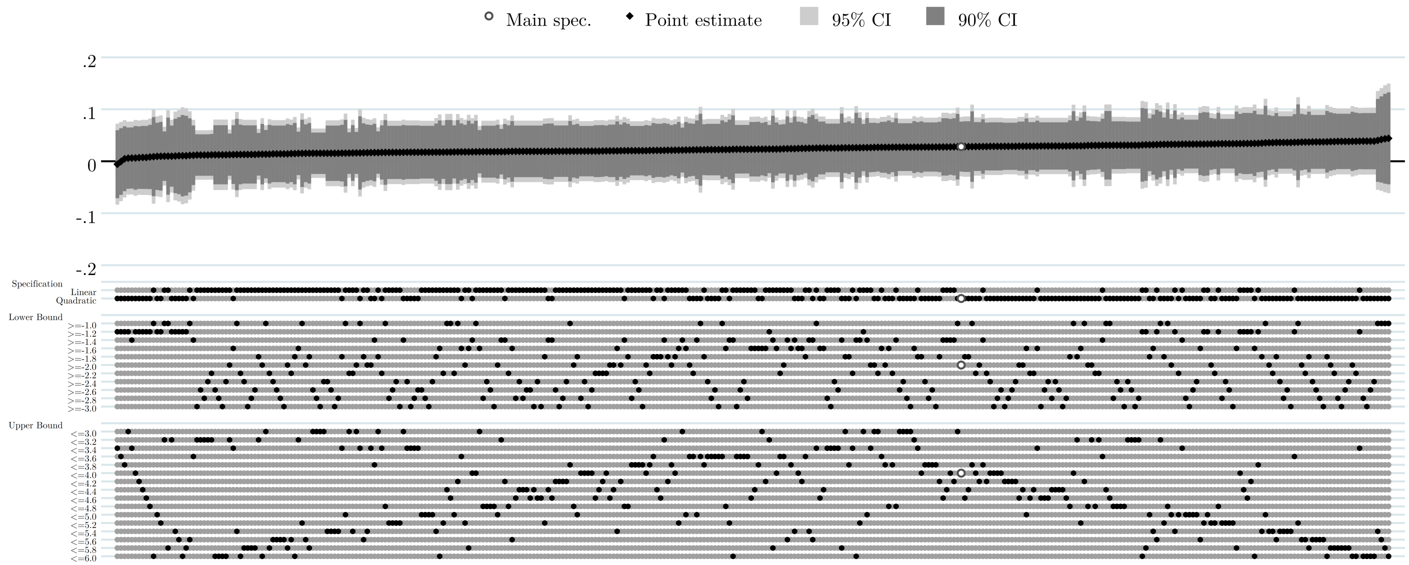
NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents 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 20: Sensitivity to alternative bandwidths and polynomials - Spillover effect of whether taking Anti-diabetic medication



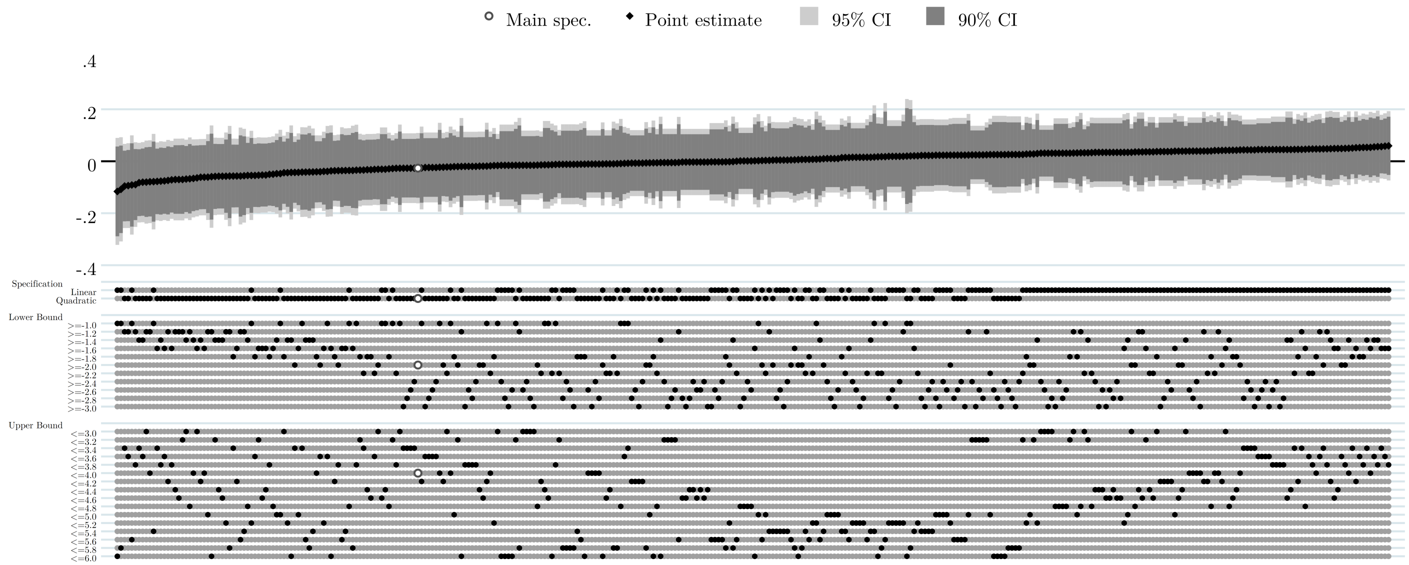
NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents 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 21: Sensitivity to alternative bandwidths and polynomials - Spillover effect of whether taking Antibiotic medication



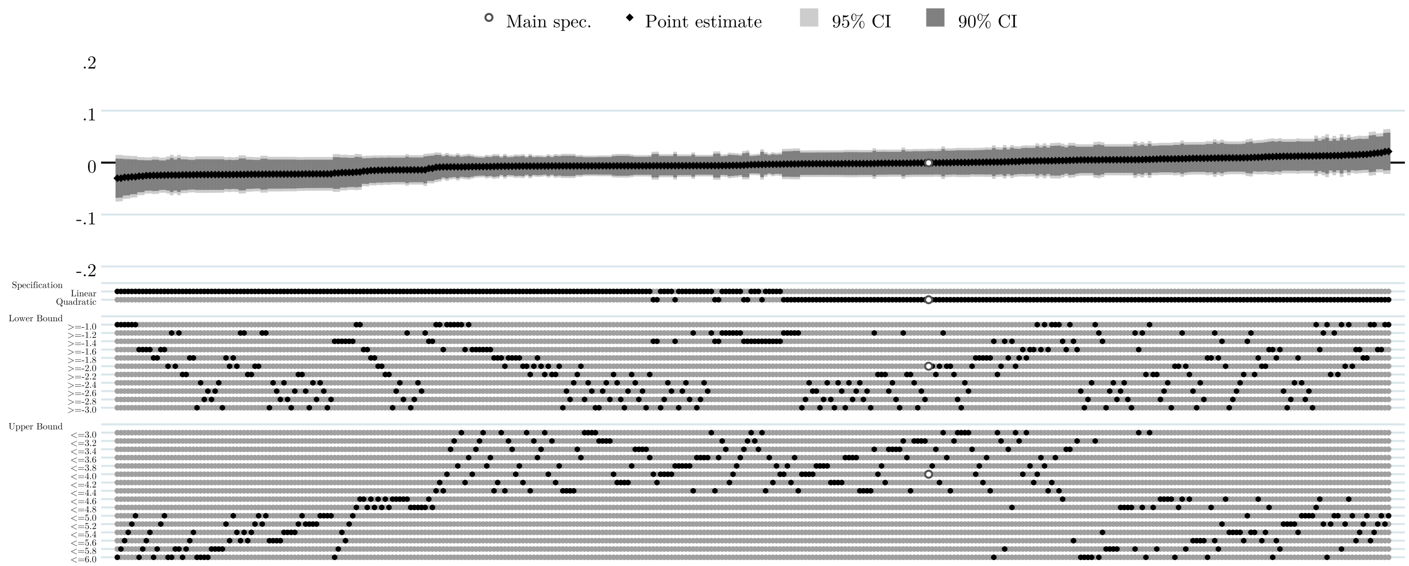
NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents 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 22: Sensitivity to alternative bandwidths and polynomials - Spillover effect of whether taking Anti-depressant medication



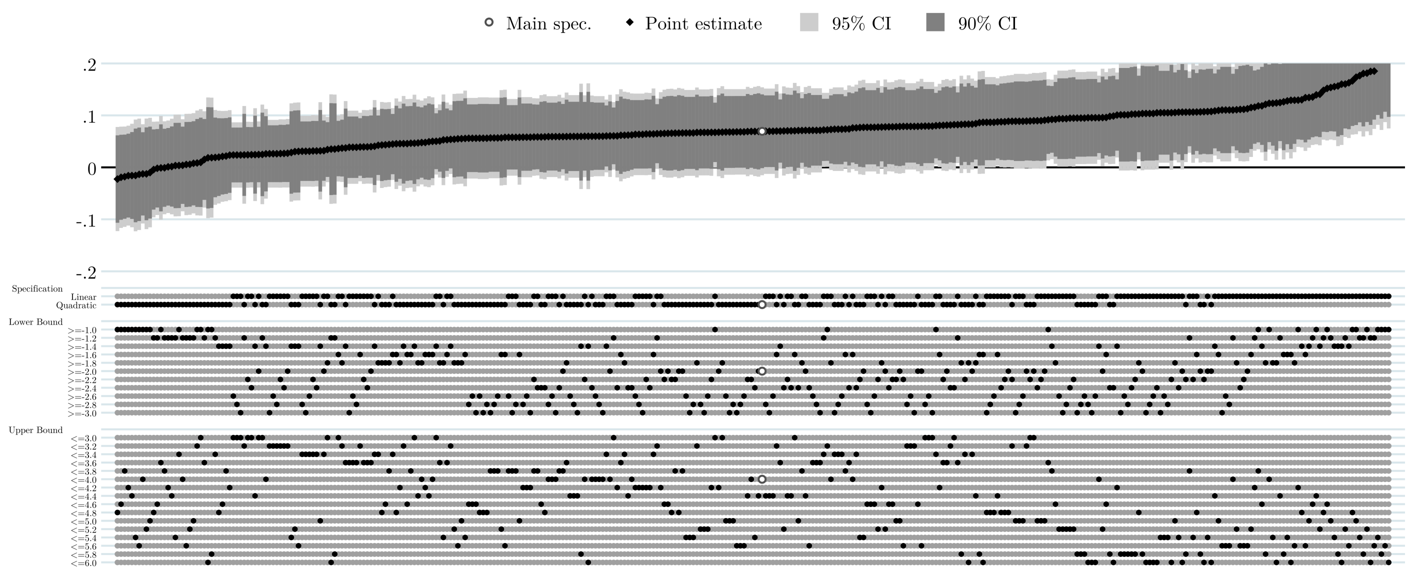
NOTE: The figure shows point estimates, 90% and 95% confidence intervals across a variety of specifications all using the two-stage least squares estimation procedure outlined in equations (2) and (3). Each point estimates shows the corresponding specification underneath, where a black dot represents 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 23: Sensitivity to alternative bandwidths and polynomials - Spillover effect of whether taking Statins



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*.

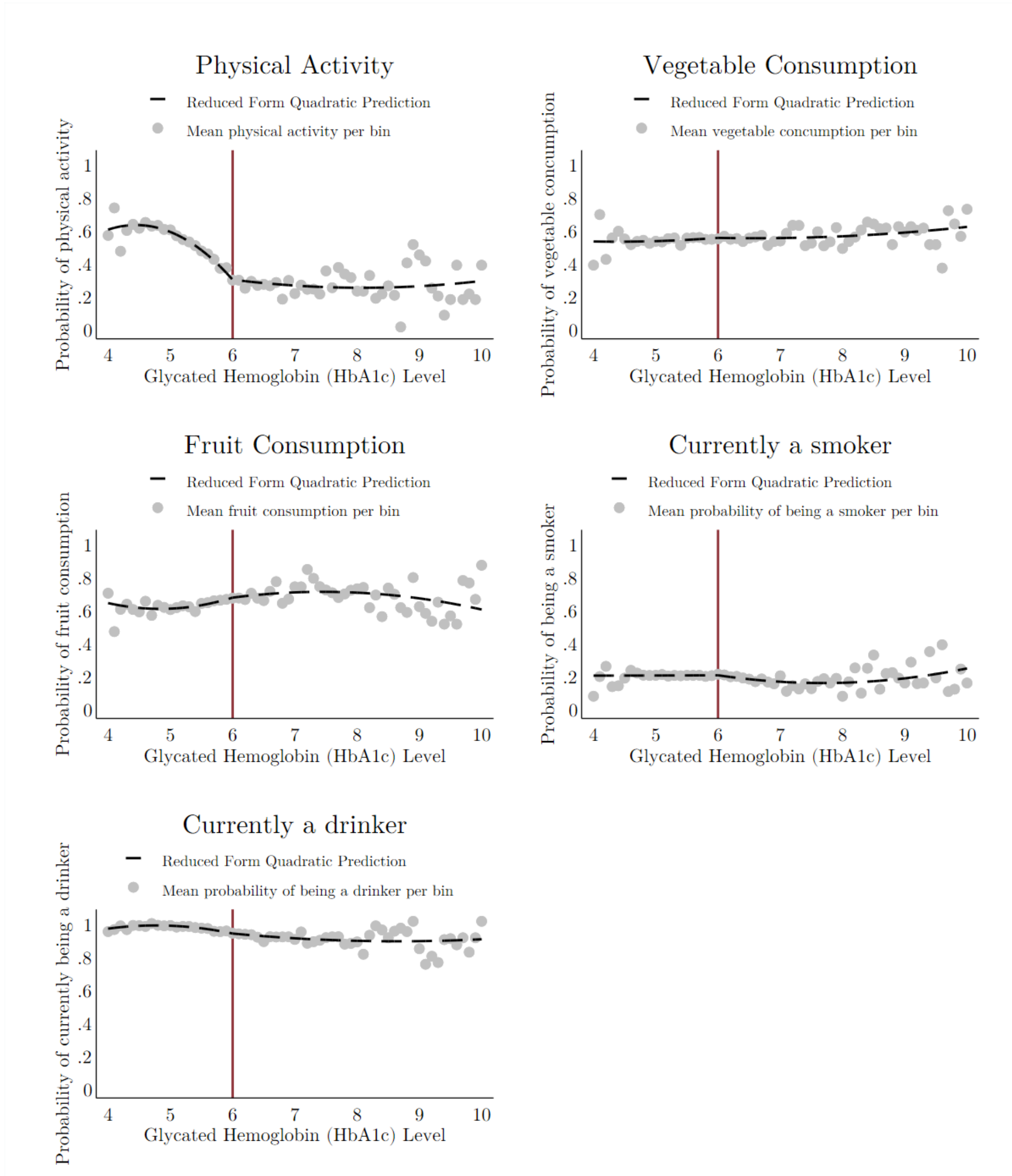
Figure 24: Sensitivity to alternative bandwidths and polynomials - Spillover effect of ever having a job



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*.

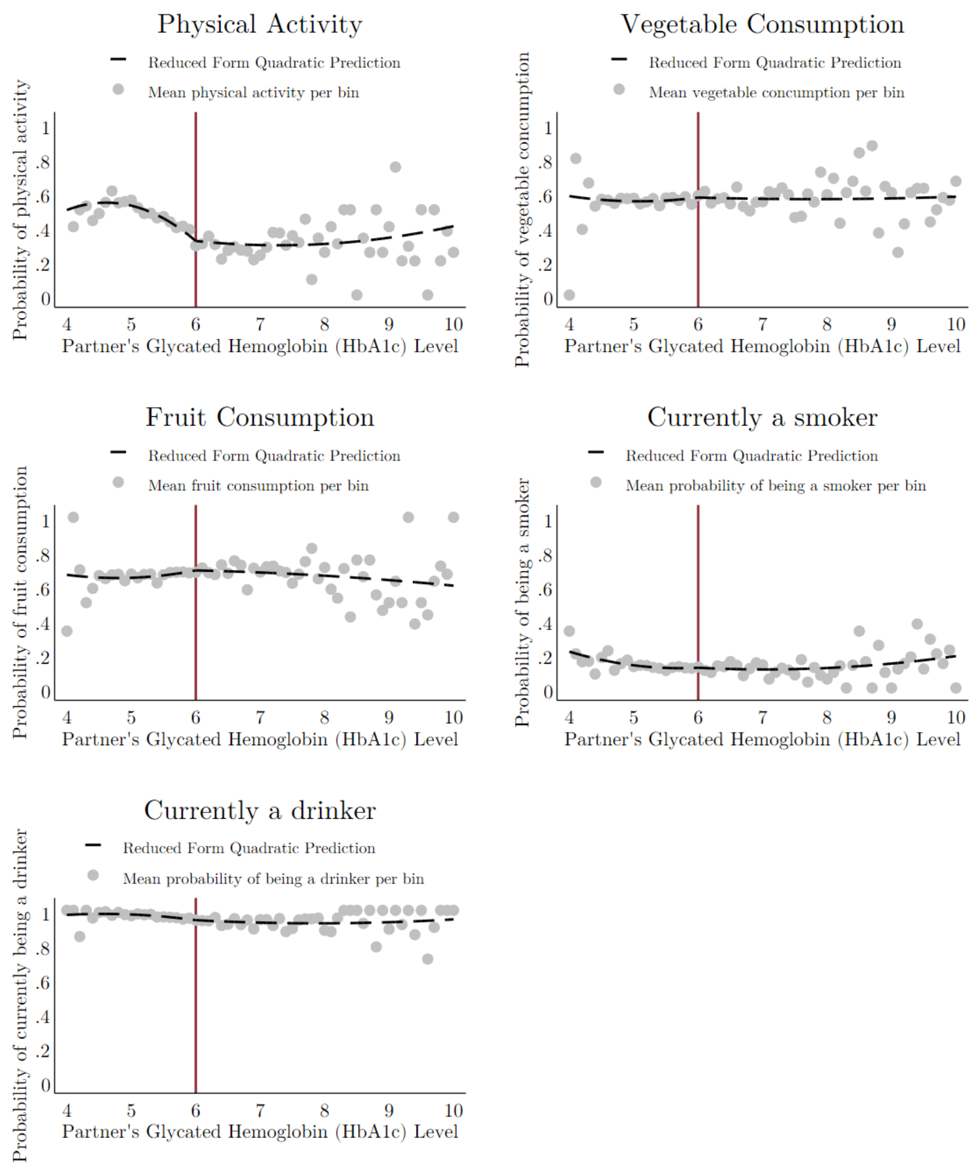
10 Appendix

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 (8).

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 (8).