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Abstract

Type 2 diabetes mellitus (T2DM) is a major public health challenge in low- and middle-income countries, yet little is known about how treatment, behavior, adiposity, and glycemic control evolve after diagnosis. Using repeated cross-sectional data from ENSANUT (2006, 2012, 2018), we analyzed 9,277 Mexican adults aged 20 and older with prior physician-diagnosed T2DM, examining post-diagnosis trajectories in treatment use, household expenditure on non-recommended foods, body mass index (BMI), overweight, obesity, glycosylated hemoglobin (HbA1c), and fasting blood glucose, using pooled models with fixed effects for age-at-diagnosis-by-sex and municipality, and testing heterogeneity by sex and socioeconomic status (SES). Treatment uptake rose and remained elevated with time since diagnosis, reaching +24.4 percentage points at 12+ years ($p<0.01$), while household expenditure on non-recommended foods showed little sustained change. BMI declined gradually, remaining 2.54 kg/m^2 lower at 12+ years ($p<0.01$); obesity was 16.9 percentage points lower ($p<0.01$); overweight followed a less consistent trend. Glycemic control did not improve: HbA1c remained elevated at longer durations, reaching +1.01 percentage points at 12+ years ($p<0.01$), with fasting glucose showing a similar pattern. Diagnosis is associated with rapid increases in clinical engagement, but glycemic control remains persistently poor despite rising treatment use, suggesting that improving outcomes may require integrated care combining pharmacological management with sustained behavioral support, closer metabolic monitoring, and attention to the social conditions shaping long-term self-management.

Keywords: type 2 diabetes mellitus; post-diagnosis trajectories; glycemic control; obesity; treatment uptake; Mexico.
JEL Classification: I12, I14, I18, I15

1 Background

Type 2 diabetes mellitus (T2DM) is a major global health challenge. It affects more than 462 million people worldwide, and both prevalence and mortality have increased steadily since 1990 [1, 2]. The burden is increasingly concentrated in low- and middle-income countries (LMICs), which now account for most cases and have experienced increases in overweight and obesity [3]. Although a large literature has examined the determinants, prevalence, and economic costs of diabetes, less is known about what happens after diagnosis: how treatment, health behaviors, and metabolic risk evolve once individuals learn they are living with T2DM [4, 5].

This matters because diabetes management unfolds over time. A diagnosis may prompt clinical follow-up, as well as medication use, dietary adjustment, and other forms of self-management, but these responses may be incomplete, delayed, or difficult to sustain [4, 6]. Some individuals may translate diagnosis into improved disease management, whereas others may face financial constraints, time scarcity, competing care responsibilities, or barriers within the health system [7, 8]. The period following diagnosis is therefore relevant for understanding whether clinical recognition is followed by meaningful risk reduction or by continued metabolic deterioration despite contact with care [9–11].

The literature on post-diagnosis patterns in T2DM remains limited in several respects. Much of it describes average trends or cross-sectional differences in body weight, glycemic control, diet, or treatment use after diagnosis [12–14]. These studies are informative but not well suited to characterizing how outcomes evolve after diagnosis and separating post-diagnosis responses from the underlying progression of disease [4, 15]. This distinction matters because treatment uptake, behavioral adjustment, self-management burden, and metabolic change often do not move together and often unfold at different rates [4, 16].

In particular, increased treatment use does not imply improved metabolic control. Body weight may decline modestly without corresponding reductions in glycated hemoglobin (HbA1c), and greater clinical engagement may coexist with persistently poor glycemic control [15, 16]. This potential decoupling is clinically relevant because poor glycemic control is associated with long-term diabetes complications and with greater vulnerability to severe viral and bacterial infections, including through impaired immune function, heightened inflammation, and higher risks of hospitalization and mortality [2, 17–19]. Evidence from Mexico and Latin America suggests that glycemic control remains poor even among adults who are diagnosed and receiving treatment: Population-based studies document that a substantial proportion of diagnosed adults in Mexico have HbA1c levels above recommended targets and that treatment uptake does not reliably translate into adequate metabolic control [20–22]. Similarly, cross-sectional evidence from the region indicates that obesity and overweight are highly prevalent among adults with T2DM at the time of diagnosis and persist over time, and dietary quality tends to remain poor despite clinical contact [23, 24]. By contrast, improvements in weight status do not automatically translate into sustained glycemic improvement [25, 26]. Examining post-diagnosis trajectories across multiple domains may therefore help clarify whether diagnosis is followed by broad-based metabolic improvement or by more selective changes [4, 5].

Post-diagnosis trajectories can be shaped by social and structural conditions. A diabetes diagnosis provides clinical information, but the ability to act on that information depends on resources and constraints that are unequally distributed. Diabetes self-management may be facilitated by access to regular care, medication continuity, timely treatment adjustment, recommended food, time available for self-care, household support, and sustained guidance from the health system [7, 8, 23]. These conditions may vary by gender, socioeconomic status (SES), ethnicity, and household context [27]. For example, caregiving responsibilities and time available for self-care may differ by gender; lower-SES populations may face more constrained food environments, less stable access to services, and greater difficulty sustaining follow-up; and Indigenous adults in Mexico receive lower-quality care than non-Indigenous adults [22, 28, 29]. These differences may shape whether diagnosis is followed by treatment uptake, behavioral adjustment, weight change, or improved glycemic control. These considerations motivate us to include

individual, household, and geographic covariates in the empirical models and examine heterogeneity by gender, age at diagnosis, and SES.

Only a few studies have used quasi-experimental approaches to examine the effects of diabetes diagnosis, and these have largely focused on short- to medium-term changes in body weight [5, 30]. Such analyses often rely on specialized administrative data, focus on individuals close to diagnostic thresholds, and consider a relatively narrow set of outcomes. Less is known about how diagnosis is associated with longer-run trajectories in treatment use, adiposity, glycemic control, and behavioral adjustment across broader populations [4, 5, 15]. This gap is especially relevant in LMICs, where the diabetes care cascade remains incomplete. Across 28 LMICs, many people living with diabetes remain untested, undiagnosed, untreated, or poorly controlled [11, 31]. These patterns suggest that diagnosis alone is not sufficient to secure effective metabolic management.

Evidence from Mexico and Latin America suggests that diagnosis and treatment contact do not necessarily translate into adequate metabolic control. Population-based studies in Mexico have documented a high prevalence of T2DM and poor glycemic control among diagnosed adults, while evidence on effective coverage indicates persistent gaps in access to quality diabetes care [20–22]. These findings support the expectation that treatment uptake may increase after diagnosis without corresponding improvements in glycemic control. Evidence also indicates that overweight and obesity are common among adults with T2DM and that diabetes self-management in Latin America is constrained by barriers related to diet, access to care, affordability, continuity of services, and household context [23, 24]. This literature motivates us to examine whether adiposity, household food-purchase patterns, and glycemic biomarkers move together or evolve differently after diagnosis.

Mexico provides a useful setting for studying these dynamics. National evidence has documented both a high prevalence of T2DM and poor glycemic control among diagnosed adults [20, 21]. More broadly, diabetes management in Mexico and Latin America is shaped by uneven access to care, resource constraints, and persistent social inequalities [22, 23]. Among individuals without social security who rely on public primary care, effective coverage has been estimated to be low, with marked geographic disparities [22].

We examine how clinical, metabolic, and behavioral outcomes evolve after a physician diagnosis of T2DM in Mexico. We consider trajectories in treatment use, glycemic control, adiposity, and dietary behavior and examine whether these outcomes differ by gender, age at diagnosis, and SES. The study therefore provides evidence on how diabetes management evolves after diagnosis in a middle-income-country setting marked by a high disease burden and persistent gaps in effective chronic disease care.

2 Methods

2.1 Conceptual framework

Our analysis is guided by an adapted conceptual framework of post-diagnosis adaptation in T2DM, informed by the literature on chronic disease management, behavioral responses to diagnosis, and the diabetes care cascade [7, 11, 32]. Within this framework, a physician diagnosis may influence subsequent disease management through two broad pathways: a care pathway, reflected in treatment initiation and clinical engagement, and a behavioral pathway, reflected in dietary adjustment and other forms of self-management [4, 33–35]. These responses may, in turn, shape metabolic and clinical outcomes, including adiposity and glycemic control [15, 36].

The framework allows for uneven adjustment across domains. Increased treatment use may coexist with limited behavioral change, and modest improvements in body weight might not be accompanied by improved glycemic control [15, 36]. Post-diagnosis patterns therefore reflect the joint influence of treatment uptake, self-management, and the underlying progression of disease, rather than a single, uniform response to diagnosis [15, 37].

The framework also recognizes that post-diagnosis adaptation is shaped by the social and clinical context. Financial constraints, time scarcity, caregiving responsibilities, multimorbidity, and uneven access to effective care may limit the extent to which individuals are able to translate diagnosis into sustained behavioral or metabolic improvement [7, 23, 38]. For this reason, post-diagnosis trajectories may differ by gender, age at diagnosis, SES, and hypertension status [7, 11, 39].

2.2 Design, data, and study population

This is a pooled repeated cross-sectional study based on three waves (2006, 2012, and 2018) of the Mexican National Health and Nutrition Survey (ENSANUT). ENSANUT is a nationally representative survey that collects detailed information on health, nutrition, health care access, and socioeconomic conditions at the individual and household levels. Its design permits comparisons across survey rounds using harmonized measures and broadly comparable sampling procedures. The full survey documentation is available elsewhere [40].

ENSANUT does not consistently distinguish gestational from non-gestational diabetes diagnosis across the survey waves used in this analysis. We therefore could not exclude women whose first diabetes diagnosis may have occurred during pregnancy. The main analytical sample was restricted to adults who reported a prior physician diagnosis of diabetes. Time since diagnosis was constructed from self-reported duration since that physician diagnosis.

The biomarker information was not used to construct elapsed time since diagnosis. HbA1c and fasting blood glucose were used only as outcome measures in the biomarker subsample (n = 2,092 across the three waves). Respondents without a prior physician diagnosis but with biomarker values consistent with diabetes were not included in the main event-time analysis because they had not necessarily received a prior clinical diagnosis.

2.3 Exposure measure

The main exposure was time since T2DM diagnosis. ENSANUT asks respondents whether they have ever been diagnosed with diabetes by a physician and how long ago the diagnosis occurred. Self-reported time since diagnosis is widely used in population-based surveys to characterize duration-related patterns in chronic disease management, although it may be affected by recall error [15, 37]. In the main analysis, time since diagnosis was defined in discrete yearly intervals (years 0–11 and a terminal category grouping respondents with 12 or more years since diagnosis). The yearly analysis was complemented by a monthly analysis based on self-reported elapsed months since diagnosis for respondents diagnosed within the 12 months preceding the interview.

2.4 Outcome measures

Metabolic outcomes. We used body mass index (BMI, kg/m^2) as a continuous measure and defined overweight and obesity using standard thresholds: overweight as BMI 25.0–29.9 kg/m^2 and obesity as BMI $\geq$ 30.0 kg/m^2 [41, 42].

Clinical outcomes. We used glycated hemoglobin (HbA1c), measured as a continuous percentage [43], for the biomarker subsample. We also examined fasting blood glucose, available for a subsample of respondents, as a complementary biomarker of glycemic control.

Behavioral and care-related outcomes. We examined whether respondents reported current treatment for T2DM, defined as use of insulin or oral hypoglycemic medication. We also analyzed the household share of expenditure on non-recommended foods relative to total food expenditure as a proxy for food-purchase patterns relevant to diabetes management. Household expenditure on non-recommended foods was constructed from ENSANUT household food-expenditure categories. This

measure was not derived from a clinical dietary guideline or a validated individual dietary-intake instrument. We classified as non-recommended foods those expenditure categories corresponding to ultra-processed products under the NOVA classification, which groups foods according to the extent and purpose of industrial processing [44]. We use the term 'non-recommended' descriptively to refer to ultra-processed food and beverage categories that are generally discouraged in dietary guidance for diabetes management; however, the variable itself is based on NOVA processing categories rather than an individual dietary-intake instrument. These categories include sugar-sweetened beverages, packaged snacks, confectionery, processed meats, and other industrially formulated products. This classification has been applied in prior ENSANUT-based studies [45]. The measure therefore captures the household food-purchasing context rather than individual dietary intake. Although household expenditure does not measure individual dietary intake directly, it captures the food purchasing environment in which diabetes self-management takes place and provides a feasible behavioral indicator in large household surveys lacking detailed individual dietary follow-up [38, 46].

2.5 Covariates

Following previous studies [7, 11, 24, 47] and our conceptual framework, all models adjusted for individual, household, and geographic characteristics. These included age-at-diagnosis-by-sex fixed effects, and municipality fixed effects. Additional covariates captured socioeconomic position and household composition. SES was measured using an asset-based index estimated through factor analysis of household assets and housing materials, with higher values indicating greater assets and better housing conditions [48]. In heterogeneity analyses, we further stratified by gender, age at diagnosis, and SES. We also used physician-diagnosed hypertension as an additional clinical characteristic in selected mechanism analyses. Hypertension was identified using the standard ENSANUT question on prior physician diagnosis of high blood pressure.

Municipality fixed effects absorb broad geographic and contextual variation — including differences in health care access, infrastructure, and socioeconomic conditions — but do not replace individual-level ethnicity controls. Indigenous status was therefore included as an additional covariate in robustness specifications; the coefficient was not statistically significant, likely reflecting the limited sample of Indigenous adults with diagnosed T2DM in our sample (n = 863).

3 Empirical strategy

3.1 Event-time design

Our empirical strategy characterizes post-diagnosis trajectories in clinical, metabolic, and behavioral outcomes as a function of elapsed time since diagnosis among adults with a prior physician diagnosis of T2DM. The aim is to examine how outcomes differ across respondents observed at different durations since diagnosis after adjusting for observed covariates and fixed effects.

This approach has two main advantages. First, it allows post-diagnosis patterns to be examined over a relatively long window. Second, by comparing respondents diagnosed at similar ages and observed under common geographic conditions, it reduces the extent to which estimated gradients reflect simple compositional differences across respondents. The resulting coefficients can therefore be interpreted as adjusted differences in outcomes by time since diagnosis.

Each respondent was observed once only. Because the number of observations declined at longer durations since diagnosis, the main trajectory analysis included event-time indicators for years 0–11 and a terminal category grouping respondents with 12 or more years since diagnosis. For each outcome, we estimated models of the following form:

$$y_i = \sum_{t=1}^{T} \beta_t \, 1\{\tau_i = t\} + X_i'\gamma + \alpha_{a(i),s(i)} + \mu_{mun(i)} + \epsilon_i \tag{1}$$

y_i denotes the outcome of interest (metabolic, clinical, and behavioral and care-related outcomes), τ_i is the time since diagnosis when the interview for individual i occurred ($\tau_i = 0$ is the omitted category), X_i is a vector of individual and household covariates, $\alpha_{a(i),s(i)}$ are age-at-diagnosis-by-sex fixed effects, and $\mu_{mun(i)}$ are municipality fixed effects.

This specification compares respondents interviewed at different durations since diagnosis while holding constant age at diagnosis and accounting for common geographic factors. Age-at-diagnosis-by-sex fixed effects ensure that comparisons are made among respondents who entered diagnosed status at similar points in the life course within sex groups, while municipality fixed effects absorb geographic differences that could otherwise confound the estimated gradients.

The event-time coefficients summarize how outcomes vary with duration since diagnosis relative to the reference category. These estimates should therefore be interpreted as adjusted differences in outcomes by time since diagnosis rather than simple cross-sectional contrasts across pooled respondents.

For binary outcomes, we estimated linear probability models to retain a common interpretation across specifications and to facilitate inclusion of multiple fixed effects [49]. Continuous outcomes were estimated using linear regression.

3.2 Design checks and sensitivity analyses

Because the study is observational and based on repeated cross sections, the estimated trajectories may still be affected by residual selection, measurement error, or survivor bias. We therefore conducted a set of design checks and sensitivity analyses to assess whether the observed gradients were consistent with the proposed empirical strategy.

First, we examined balance in predetermined characteristics across time since diagnosis. Specifically, we regressed respondent and household characteristics that should not be altered by post-diagnosis adaptation on the event-time measure to assess whether respondents observed at different durations since diagnosis differed systematically in their demographic and socioeconomic characteristics. This analysis helps assess whether respondents observed at different durations since diagnosis are broadly comparable in their predetermined characteristics, providing a check on whether the estimated gradients are driven by systematic compositional differences rather than post-diagnosis change.

Second, we estimated placebo specifications using variables that should not plausibly respond to time since diagnosis. Flat placebo gradients would be consistent with the view that the event-time structure is not simply capturing unrelated socioeconomic or contextual differences across respondents.

3.3 Heterogeneity and mechanism analyses

To assess whether post-diagnosis trajectories differed across population groups, we estimated stratified models by gender, age at diagnosis, and SES. These analyses were intended to characterize heterogeneity in post-diagnosis responses rather than estimate fully specified models of effect modification. To explore potential mechanisms, we examined trajectories in treatment use and household food-expenditure patterns. We also assessed whether trajectories differed by hypertension status as a way of exploring the role of multimorbidity in post-diagnosis management. These analyses were interpreted as suggestive evidence on pathways rather than definitive tests of mechanisms.

4 Results

4.1 Average post-diagnosis trajectories

We pooled the 2006, 2012, and 2018 ENSANUT waves to construct a repeated cross-sectional dataset of adults aged 20 years and older who reported a previous physician diagnosis of T2DM. The analytical sample comprised 9,277 individuals: 2,505 in 2006, 3,593 in 2012, and 3,179 in 2018. Of the pooled sample, 59.8% were women and 40.2% were men. The mean age at interview was 55.7 years (SD 13.1), the mean age at T2DM diagnosis was 49.9 years (SD 12.7), and the mean elapsed time since diagnosis was 5.59 years (SD 4.08). Table 1 reports descriptive statistics for the pooled sample.

Table 2 reports adjusted differences in behavioral, metabolic, and clinical outcomes by time since diagnosis, relative to respondents interviewed in the year of diagnosis. Three patterns are apparent. Longer time since diagnosis was associated with higher treatment use. Adiposity declined over time, with larger and more regular changes for obesity than for overweight. Glycemic biomarkers did not show sustained improvement over longer durations since diagnosis. Figures 1 and 2 show similar overall patterns.

Treatment uptake rose early and remained above the year-0 reference throughout the observation window. The probability of currently receiving treatment was 3.8 percentage points higher in year 1 and 11.0 percentage points higher in year 2, increasing to 24.4 percentage points in the category of 12 or more years. By contrast, the household share of expenditure on non-recommended foods showed little evidence of systematic change. Coefficients were small and imprecise and did not follow a monotonic pattern.

Two of the three measures of adiposity declined gradually. BMI was significantly lower from year 4 onwards and reached its largest estimated reduction in year 11 ($-2.85\ kg/m^2$, $p < 0.01$), remaining below the reference category in the 12-or-more-years group ($-2.55\ kg/m^2$). These continuous changes translated more clearly into obesity than into overweight. The probability of obesity decreased significantly from year 4 onwards, by -16.9 percentage points in the 12-or-more-years category. Overweight started to decline, but the pattern was irregular and eventually started to increase, with significant increments appearing from year 7 onwards and increasing by 7 percentage points in the 12-or-more-years category.

Clinical outcomes followed a different pattern. Fasting blood glucose was higher from year 3 and remained elevated thereafter, reaching its highest estimate in year 9 (+66.4 mg/dL). HbA1c did not show sustained early improvement. After two small negative coefficients during the first 2 years and little difference through years 3–5, estimates became positive and statistically significant from year 6 onwards, including in the 12-or-more-years category (+1.01%). The main pattern in Table 2 is therefore one of increasing treatment uptake without corresponding improvement in glycemic biomarkers over longer durations since diagnosis.

Appendix A1 reports month-by-month estimates for the first 12 months after diagnosis. These short-run estimates complement the yearly results but do not alter the overall interpretation from Table 2.

4.2 Heterogeneity in post-diagnosis trajectories

Table 3 and Table 4 show heterogeneity by sex and SES. For adiposity, reductions in BMI were more apparent among women than among men, particularly in the earlier years after diagnosis. Among women, BMI was decreasing in year 1 and remained below the reference category thereafter; among men, reductions were smaller and less precisely estimated in the early years and became clearer only at longer durations. In the 12-or-more-years category, BMI was 3.40 kg/m^2 lower among women and 0.80 kg/m^2 lower among men.

A similar pattern was observed for obesity. Among women, the probability of obesity was already lower by year 2 and remained negative thereafter. Among men, early coefficients were positive or close to zero before turning negative at longer durations. At 12 or more years, the estimated reduction in

obesity was 22 percentage points among women and 7 percentage points among men. Overweight did not significantly change among men, but among women, it tended to increase the probability of overweight approximately 13 percentage points in the 12-or-more-years group.

Differences by SES were also evident. BMI declined in both SES groups, with larger and more consistent reductions among higher-SES respondents at several durations and in the 12-or-more-years category ($-2.86\ kg/m^2$ versus $-2.46\ kg/m^2$ in lower-SES respondents). Obesity also declined in both groups. At longer durations, the magnitudes were similar, although reductions appeared earlier and more consistently among higher-SES respondents. For overweight, increments were irregular in both SES groups.

In Table 4 treatment increased in all subgroups, but the increase was larger among men than among women and larger among higher-SES than lower-SES respondents. At 12 or more years, the treatment-use coefficient was 27 percentage points among men and 23 percentage points among women, and 25 percentage points among higher-SES respondents compared with 20 percentage points among lower-SES respondents. Household non-recommended-food expenditure again showed little evidence of sustained change, both by sex and by SES.

Table 4 also shows that HbA1c increased over time across subgroups, but the pattern differed in magnitude. By sex, increases were more consistently estimated among women than among men. By SES, coefficients were generally nonsignificant. Among the biomarker subsample, fasting blood glucose showed a qualitatively similar pattern of persistent elevation at longer durations since diagnosis; however, because of the reduced subsample size, sex- and SES-stratified estimates for fasting blood glucose are not separately tabulated.

4.3 Dynamic patterns in treatment, behavior, and health outcomes

Figure 3 presents predicted post-diagnosis trajectories stratified by household food-expenditure context, defined by whether the share of spending on recommended foods (100 minus the household share of non-recommended-food expenditure) was above or below the national median (88%). In descriptive terms, the trajectories were more favorable among respondents in households with a higher share of spending on recommended foods. In this group, glycemic trajectories were less adverse, while treatment uptake increased in both groups. BMI and obesity declined in both groups, although the declines appeared more favorable in the group with a higher share of recommended-food expenditure. Overweight tended to increase more consistently among the group spending less in recommended foods.

Because these comparisons are based on a household expenditure proxy rather than direct measures of individual dietary intake, they are best read as descriptive differences by household context rather than as estimates of dietary effects.

4.4 Design checks and sensitivity analyses

The appendix reports several additional checks. First, A.1 reports that month-by-month estimates in the first year after diagnosis were qualitatively consistent with the annual results. Second, supplementary analyses based on subgroup specifications by sex (B1), median age of T2DM diagnosis (49.6 years) (B2), and SES (B3) did not materially alter the pattern of rising treatment uptake, declining adiposity, and limited improvement in glycemic control. Trajectories stratified by hypertension (B4) status were broadly similar across respondents with and without a prior hypertension diagnosis, consistent with the view that the main patterns are not driven by differential multimorbidity burden.

A potential concern is selective mortality, which can lead to compositional changes in the sample. To address this concern, we reestimated our main specifications restricting the sample to younger individuals at the time of diagnosis,[1] a cohort for whom short- to medium-run mortality risk is substantially lower. If our baseline results were driven primarily by selective survival, we would expect attenuated effects in

[1] Mean age of diagnosis minus 1 standard deviation = 37 years.

this younger subsample. Instead, we observed qualitatively and quantitatively similar patterns (Panel B in Figure B5) to those in the full sample (Panel A in Figure B5), suggesting that selective mortality is unlikely to account for the main findings.

In Figure B6 the placebo specifications—using variables that should not plausibly respond to time since diabetes diagnosis—showed flat gradients, consistent with the idea that event-time structure does not simply capture unrelated socioeconomic trends

Taken together, the supplementary analyses were consistent with the main empirical patterns and did not materially change their interpretation.

5 Discussion and conclusions

This study examines post-diagnosis trajectories in treatment, behavioral, metabolic, and clinical outcomes among adults living with T2DM in Mexico. Using variation in time since diagnosis within a repeated cross-sectional framework, we document that longer durations since diagnosis are associated with higher treatment uptake, gradual reductions in adiposity, and persistently elevated glycemic biomarkers. Dietary behavior, proxied by household expenditure on non-recommended foods, showed little evidence of sustained adjustment These patterns were not uniform across groups: Treatment uptake was more strongly associated with longer post-diagnosis duration among men and higher-SES respondents, and less favorable glycemic trajectories were more consistently observed among women. Taken together, the findings are consistent with a post-diagnosis process in which clinical engagement increases more readily than durable metabolic control is achieved, though the observational design precludes causal inference.

A central pattern is the asymmetry between treatment and dietary behavior. Longer durations since diagnosis were associated with higher pharmacological treatment use but not with meaningful reductions in household spending on non-recommended foods. This is consistent with prior evidence that clinical engagement tends to intensify after diagnosis, while behavioral adjustment depends more heavily on household resources, food environments, and time constraints—factors less directly shaped by contact with the health care system [4, 8, 50–52].

A second pattern is the absence of improved glycemic control despite higher treatment uptake at longer durations. Elevated HbA1c persisted across groups with increasing treatment use, which is consistent with evidence that pharmacological management alone is often insufficient to achieve sustained glycemic control [53–57]. This pattern may also reflect undertreatment or suboptimal intensification, as documented in other settings in which many adults with T2DM remain above recommended HbA1c targets despite being on treatment [55, 58, 59]. However, given the observational design, these interpretations remain speculative.

The observed decline in obesity alongside persistently poor glycemic control warrants cautious interpretation. Several non-mutually-exclusive explanations are plausible: Pharmacological treatment may influence body weight partly independently of dietary change [60]; diagnosis may trigger short-term behavioral responses that reduce caloric intake without improving broader diet quality [5, 61]; and in the context of poorly controlled T2DM, weight loss may reflect catabolic processes associated with severe hyperglycemia rather than genuine metabolic improvement [62–64]. The latter interpretation is especially relevant in Mexico, where gaps in effective coverage and quality of care have been documented [22, 29, 65]. Reductions in obesity should therefore not be interpreted automatically as evidence of broad metabolic recovery.

Subgroup differences suggest that post-diagnosis trajectories are socially patterned, though formal tests of subgroup differences were not conducted and these comparisons should be interpreted cautiously. Higher treatment uptake was observed at longer durations among men and higher-SES respondents, less favorable glycemic trajectories were more consistently observed among women, and SES differences in

HbA1c were less clear. These patterns are broadly consistent with evidence that structural barriers—including income constraints, caregiving responsibilities, and uneven access to quality care—shape the capacity to translate diagnosis into sustained disease management [7, 8, 66–68].

Although our heterogeneity analyses focused on sex and SES, ethnic inequalities represent an important additional dimension of diabetes care in Mexico. Recent evidence shows that Indigenous adults with T2DM receive lower-quality care than non-Indigenous adults, suggesting that post-diagnosis trajectories may also differ by ethnicity. Future research should examine whether treatment uptake, behavioral adjustment, adiposity, and glycemic control after diagnosis evolve differently among Indigenous and non-Indigenous populations and whether these differences reflect unequal access to high-quality primary care, continuity of care, language and cultural barriers, or broader structural disadvantages.

Although the models include municipality fixed effects, these do not fully account for ethnic inequalities. Municipality fixed effects absorb shared geographic context, including time-invariant differences in local health care access, socioeconomic resources, food environments, health-care-system capacity, and broad ethnic composition. This is relevant in Mexico, where Indigenous populations are unevenly distributed across municipalities and where ethnic density often overlaps with differences in local resources and health care access. However, municipality fixed effects do not capture within-municipality differences between Indigenous and non-Indigenous respondents, nor do they directly measure language barriers, discrimination, continuity of care, or quality of diabetes management. Future research should examine post-diagnosis trajectories explicitly by ethnicity and assess whether treatment uptake, behavioral adjustment, adiposity, and glycemic control evolve differently among Indigenous and non-Indigenous adults with T2DM in Mexico.

The policy implications of these findings are suggestive rather than definitive. The results are consistent with the view that expanding pharmacological treatment, while necessary, may not be sufficient if not accompanied by metabolic monitoring, treatment intensification, and behavioral support [55, 57, 69, 70]. The period immediately following diagnosis may represent a window of increased participation in care, but maintaining that participation and translating it into metabolic improvement likely requires integrated care models that address both the clinical and structural dimensions of self-management [22, 32, 71–73].

The study has several strengths, including nationally representative data across three survey waves, a relatively long post-diagnosis horizon, and a common analytical framework across multiple outcome domains. Key limitations include the repeated cross-sectional design, which does not permit within-person inference; self-reported time since diagnosis and treatment use; biomarker data available only for a subsample; and the inability to fully rule out residual confounding or survivor bias. The sample size for Indigenous adults was insufficient to estimate ethnicity-specific trajectories with adequate precision, and subgroup findings should be interpreted as a basis for generating hypotheses. ENSANUT does not consistently distinguish gestational from non-gestational diabetes diagnosis across the survey waves used in this analysis. As a result, we could not exclude women whose first diabetes diagnosis may have occurred during pregnancy. This should be considered when interpreting trajectories among women, as women first diagnosed during pregnancy may differ from other women with diabetes in age at diagnosis, treatment history, and metabolic trajectories.

In sum, longer durations since T2DM diagnosis are associated with higher treatment uptake and lower obesity but not with improved glycemic control. These associations suggest that clinical engagement and effective metabolic control are not equivalent outcomes of diagnosis and that post-diagnosis care should be assessed on both dimensions. For Mexico and similar LMIC settings, improving outcomes after diagnosis is likely to require integrated approaches combining pharmacological management, sustained behavioral support, and attention to the structural conditions that shape long-term self-management.

Abbreviations

12+: 12 or more years
BMI: Body mass index
Dx: Standard medical abbreviation for diagnosis
ENSANUT: Encuesta Nacional de Salud y Nutrición
FE: Fixed effects related to age-at-diagnosis-by-sex and municipality
FEM: Females
HbA1c: Percentage of glycosylated hemoglobin
HH: Household
HH NRF: Household percentage of non-recommended food expenditure over total food expenditure
HH SES: Index of Household Socioeconomic Status, based on assets and housing
HSES: Higher socioeconomic status (quintiles 4–5).
HH Size: Household number of equivalent adults
INEGI: Instituto Nacional de Estadística y Geografía
LMIC: Low- and middle-income countries
LSES: Lower socioeconomic status (quintiles 1–3).
SES: Socioeconomic status
T2DM: Type 2 diabetes mellitus

Declarations

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Availability of data and material

The data used in this study come from ENSANUT and are publicly available through the survey repositories. The analytic code and derived analytic files are available from the corresponding author upon reasonable request.

Authors' contributions

RGD: conception, development of methodology, data analysis, drafting the initial manuscript, revising drafts, and manuscript editing.

MTM: methodology, identification strategy, empirical model, supervision, writing, review, and editing.

ESM: methodology, supervision, writing, review, and editing.

MM: supervision, writing, review, and editing.

EDC: methodology, data analysis, writing, review, and editing.

All authors read, reviewed, and approved the final manuscript.

Ethics approval and consent to participate

This study used de-identified secondary survey data from ENSANUT. The authors did not collect new data from human participants; therefore, additional ethics approval was not required.

Conflicts of interest/Competing interests

The authors declare no conflict of interest.

Consent for publication

Not applicable.

Memorial quote

We dedicate this manuscript to our colleague, professor and friend, Sandra Sosa-Rubí, PhD, who passed away in March 2021; Sandra consistently inspired us in our analysis of equity and financial protection in health during her fruitful lifetime.

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Table 1: Descriptive Statistics

	2006		2012		2018		Full
	2505 [27%]		3593 [38.7%]		3179 [34.3%]		Sample
	Female	Male	Female	Male	Female	Male	
Observations	1508	997	2134	1459	1904	1275	9277
Positive Response (%)							
Is married or coupled	62.14	83.25	60.73	81.56	57.56	74.04	67.83
Currently Working	19.79	67.20	23.43	62.92	39.55	71.06	43.61
Has overweight	36.60	45.96	35.37	41.72	33.18	41.55	38.21
Has obesity	45.86	33.70	50.15	40.64	55.99	41.00	45.71
Has a HTA dx.	42.48	32.70	48.46	39.89	48.89	39.87	43.34
Receiving T2DM treatment	85.43	82.46	88.19	83.55	86.19	81.25	85.04
Age when surveyed, yrs.							
Mean	55.02	53.82	56.44	56.77	55.64	55.85	55.73
SD	13.48	13.58	12.60	12.93	12.80	13.51	13.10
Age at dx T2DM, yrs.							
Mean	49.36	48.48	50.61	50.78	49.59	50.03	49.91
SD	12.95	13.12	12.16	12.77	12.44	13.30	12.72
Years since T2DM dx.							
Mean	5.36	5.08	5.54	5.68	5.92	5.74	5.59
SD	4.11	3.97	4.12	4.14	4.11	3.92	4.08
BMI (kg/m^2)							
Mean	30.11	28.70	30.94	29.57	31.48	29.23	30.19
SD	5.97	5.26	6.52	5.75	6.15	5.01	6.03
Glucose (mg/dL)							
Mean	157.06	162.51	178.68	179.78	171.36	175.04	172.66
SD	84.99	80.96	86.23	87.87	85.23	90.76	86.67
HbA1c (%)							
Mean	10.79	10.59	9.52	9.10	7.94	8.07	8.83
SD	2.89	2.74	2.65	2.53	2.37	2.27	2.70
HH SES (0-100)							
Mean	69.27	71.89	69.66	71.79	71.14	72.24	70.83
SD	14.93	14.31	13.88	13.24	11.75	11.98	13.39
HH Size							
Mean	2.57	2.69	2.33	2.43	2.23	2.35	2.41
SD	1.16	1.09	1.06	1.03	1.03	1.05	1.08
HH (%) NR food exp.							
Mean			9.90	11.21	10.48	12.58	10.85
SD			11.25	11.40	11.51	13.17	11.77

Source: Authors' elaboration on ENSANUT.

Notes: BMI: Body Mass Index (kg/m^2). Overweight is defined as BMI 25.0–29.9, obesity as BMI $\geq$ 30.0 kg/m^2. HTA: High blood pressure (hypertension).

Glucose: Fasting blood glucose (mg/dL). HbA1c: Percentage of glycosylated hemoglobin.

HH means Household. HH Size: Number of equivalent adults.

HH SES: Index of Household Socioeconomic Status, based on assets and housing.

HH (%) NR food exp: Household percentage of non-recommended food expenditure over total food expenditure.

Table 2: Main Results

	Behavioral and care-related outcomes [1-2]		Metabolic outcomes [3-5]			Clinical outcomes [6-7]	
	1	2	3	4	5	6	7
	Treatment	HH NRF	BMI	Obesity	Overweight	Glucose	HbA1c
Intercept	0.699***	10.896***	31.547***	0.536***	0.349***	146.225***	8.321***
	(0.015)	(0.423)	(0.249)	(0.018)	(0.018)	(5.668)	(0.238)
Year							
1	0.038*	1.123*	−0.485	0.010	−0.035	4.478	−0.595*
	(0.020)	(0.664)	(0.350)	(0.029)	(0.028)	(8.515)	(0.319)
2	0.110***	0.455	−0.430	0.007	−0.024	15.166	−0.094
	(0.020)	(0.640)	(0.377)	(0.029)	(0.029)	(9.697)	(0.344)
3	0.140***	−0.418	−0.691*	−0.021	−0.017	26.166***	0.411
	(0.021)	(0.726)	(0.387)	(0.031)	(0.031)	(9.348)	(0.349)
4	0.159***	−0.729	−1.508***	−0.082**	0.023	25.570**	0.316
	(0.022)	(0.678)	(0.381)	(0.033)	(0.034)	(10.132)	(0.378)
5	0.198***	0.360	−1.457***	−0.100***	0.072**	18.739*	0.355
	(0.021)	(0.695)	(0.379)	(0.031)	(0.031)	(11.020)	(0.343)
6	0.167***	0.360	−1.137***	−0.039	0.010	34.410***	0.985***
	(0.023)	(0.715)	(0.428)	(0.033)	(0.032)	(10.285)	(0.371)
7	0.206***	−0.218	−1.566***	−0.139***	0.127***	28.216**	0.262
	(0.023)	(0.830)	(0.441)	(0.037)	(0.038)	(11.281)	(0.422)
8	0.187***	0.187	−2.288***	−0.159***	0.065*	37.593***	1.049***
	(0.021)	(0.997)	(0.453)	(0.036)	(0.035)	(9.858)	(0.385)
9	0.218***	0.044	−2.691***	−0.238***	0.206***	66.493***	1.609***
	(0.026)	(0.952)	(0.472)	(0.043)	(0.043)	(13.223)	(0.464)
10	0.207***	−0.952	−2.239***	−0.131***	0.058**	39.506***	1.015***
	(0.019)	(0.682)	(0.340)	(0.027)	(0.028)	(9.097)	(0.310)
11	0.223***	0.451	−2.855***	−0.191***	0.097**	39.049***	0.227
	(0.027)	(1.076)	(0.713)	(0.047)	(0.046)	(13.319)	(0.531)
12+	0.244***	−0.520	−2.545***	−0.169***	0.070***	39.336***	1.016***
	(0.019)	(0.653)	(0.367)	(0.027)	(0.026)	(7.803)	(0.303)
Obs	9038	6469	5805	5947	5947	2020	1735
Adj. R^2	.073	.046	.084	.061	.023	.11	.14
FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Source: Authors' elaboration.

Notes: Municipal clustered standard errors in parentheses.

Year 12+ (12 or more years) collapses long-term diagnosis effects.

Treatment: Currently receiving T2DM treatment.

HH NRF: Household percentage of non-recommended food expenditure over total food expenditure.

BMI: Body Mass Index (kg/m^2). Overweight is defined as BMI 25.0–29.9. Obesity as BMI $\geq$ 30.0 kg/m^2.

Glucose: Fasting blood glucose (mg/dL). HbA1c: Percentage of glycosylated hemoglobin.

FE: Fixed effects related to age-at-diagnosis-by-sex and municipality.

* p<0.1, ** p<.05, *** p<.01.

Table 3: Heterogeneity. Metabolic Outcomes

	BMI [1-4]				Obesity [5-8]				Overweight [9-12]			
	1	2	3	4	5	6	7	8	9	10	11	12
	FEM	MALE	LSES	HSES	FEM	MALE	LSES	HSES	FEM	MALE	LSES	HSES
Intercept	32.78***	29.45***	30.96***	32.32***	0.63***	0.39***	0.53***	0.56***	0.26***	0.48***	0.34***	0.36***
	(0.34)	(0.39)	(0.37)	(0.36)	(0.02)	(0.03)	(0.03)	(0.03)	(0.02)	(0.04)	(0.03)	(0.03)
Year												
1	−1.23**	0.83	−0.40	−1.10**	−0.04	0.10*	−0.02	−0.01	0.04	−0.17***	−0.03	−0.04
	(0.50)	(0.58)	(0.60)	(0.50)	(0.04)	(0.05)	(0.05)	(0.04)	(0.04)	(0.06)	(0.05)	(0.04)
2	−0.97*	0.82	−0.45	−0.65	−0.06	0.09*	−0.06	0.03	0.05	−0.12**	0.01	−0.05
	(0.52)	(0.62)	(0.62)	(0.51)	(0.04)	(0.05)	(0.05)	(0.04)	(0.04)	(0.05)	(0.05)	(0.04)
3	−1.27**	0.66	−1.00*	−0.99*	−0.10**	0.12**	−0.10*	0.01	0.08*	−0.17***	0.02	−0.07
	(0.58)	(0.54)	(0.60)	(0.52)	(0.04)	(0.05)	(0.05)	(0.04)	(0.04)	(0.05)	(0.05)	(0.04)
4	−1.95***	−0.84	−1.92***	−1.56***	−0.11**	−0.02	−0.13**	−0.07	0.08*	−0.10	0.06	0.01
	(0.53)	(0.57)	(0.55)	(0.57)	(0.04)	(0.06)	(0.05)	(0.05)	(0.04)	(0.06)	(0.05)	(0.05)
5	−1.81***	−0.82	−0.97	−1.98***	−0.13***	−0.06	−0.09	−0.12***	0.12***	0.00	0.04	0.08**
	(0.51)	(0.60)	(0.67)	(0.51)	(0.04)	(0.05)	(0.06)	(0.04)	(0.04)	(0.06)	(0.06)	(0.04)
6	−1.86***	−0.00	−1.32*	−1.58***	−0.08*	−0.00	−0.04	−0.08*	0.04	−0.02	0.02	0.01
	(0.57)	(0.68)	(0.73)	(0.58)	(0.04)	(0.06)	(0.06)	(0.04)	(0.04)	(0.06)	(0.06)	(0.04)
7	−2.12***	−0.36	−2.10***	−1.81***	−0.20***	−0.05	−0.14**	−0.14***	0.22***	0.01	0.13**	0.12**
	(0.61)	(0.73)	(0.75)	(0.56)	(0.05)	(0.07)	(0.07)	(0.04)	(0.05)	(0.07)	(0.07)	(0.05)
8	−3.13***	−0.51	−2.69***	−2.55***	−0.24***	−0.02	−0.20***	−0.15***	0.16***	−0.08	0.04	0.07
	(0.61)	(0.67)	(0.83)	(0.58)	(0.05)	(0.07)	(0.06)	(0.05)	(0.05)	(0.06)	(0.07)	(0.05)
9	−3.44***	−1.44*	−2.22***	−3.45***	−0.32***	−0.12*	−0.40***	−0.22***	0.34***	0.05	0.43***	0.12**
	(0.63)	(0.75)	(0.82)	(0.61)	(0.06)	(0.07)	(0.08)	(0.06)	(0.06)	(0.08)	(0.08)	(0.05)
10	−3.21***	−0.21	−2.38***	−2.31***	−0.18***	−0.05	−0.15***	−0.12***	0.12***	0.01	0.04	0.06
	(0.49)	(0.66)	(0.60)	(0.51)	(0.04)	(0.05)	(0.06)	(0.04)	(0.04)	(0.06)	(0.05)	(0.04)
11	−3.98***	−0.11	−2.92***	−3.60***	−0.23***	−0.07	−0.17**	−0.22***	0.13**	0.05	0.08	0.09
	(0.95)	(1.16)	(0.99)	(1.02)	(0.06)	(0.08)	(0.08)	(0.07)	(0.06)	(0.08)	(0.08)	(0.07)
12+	−3.40***	−0.80	−2.46***	−2.86***	−0.22***	−0.07	−0.22***	−0.16***	0.13***	−0.01	0.14***	0.04
	(0.48)	(0.61)	(0.55)	(0.51)	(0.04)	(0.05)	(0.05)	(0.04)	(0.03)	(0.05)	(0.05)	(0.04)
Obs	3478	1982	2020	3445	3547	2053	2085	3526	3547	2053	2085	3526
Adj. R^2	.092	.023	.094	.074	.061	.024	.065	.044	.021	.034	.022	.025
FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Source: Authors' elaboration.

Notes: Municipal clustered standard errors in parentheses. Year 12+ (12 or more years) collapses long-term diagnosis effects.

BMI: Body Mass Index (kg/m^2). Overweight is defined as BMI 25.0–29.9, obesity as BMI $\geq$ 30.0 kg/m^2.

FEM: Females. LSES: Lower socioeconomic status (quintiles 1–3). HSES: Higher socioeconomic Status.

FE: Fixed effects related to age-at-diagnosis-by-sex and municipality.

* p<0.1, ** p<.05, *** p<.01.

Table 4: Heterogeneity. Behavioral and Clinical Outcomes

	Behavioral and care-related outcomes						Clinical outcome			
	HH NRF [1-2]		Receiving T2DM treatment [3-6]				HbA1c [7-10]			
	1	2	3	4	5	6	7	8	9	10
	LSES	HSES	FEM	MALE	LSES	HSES	FEM	MALE	LSES	HSES
Intercept	9.36***	11.74***	0.73***	0.65***	0.72***	0.69***	8.26***	8.69***	8.28***	8.15***
	(0.87)	(0.55)	(0.02)	(0.03)	(0.03)	(0.02)	(0.31)	(0.54)	(0.44)	(0.36)
Year										
1	0.43	1.57*	0.03	0.05	0.00	0.04	−0.87*	−0.47	−0.02	−0.49
	(1.21)	(0.87)	(0.03)	(0.04)	(0.04)	(0.03)	(0.45)	(0.64)	(0.75)	(0.49)
2	1.20	0.23	0.08***	0.15***	0.07*	0.12***	−0.19	−0.33	0.20	−0.27
	(1.40)	(0.76)	(0.03)	(0.03)	(0.04)	(0.03)	(0.55)	(0.70)	(0.61)	(0.46)
3	−0.05	−0.57	0.11***	0.17***	0.12***	0.14***	0.16	0.59	0.78	0.38
	(1.54)	(0.90)	(0.03)	(0.04)	(0.04)	(0.03)	(0.46)	(0.78)	(0.68)	(0.53)
4	0.09	−1.45	0.13***	0.19***	0.13***	0.16***	0.30	0.18	0.91	0.36
	(1.34)	(0.93)	(0.03)	(0.04)	(0.04)	(0.03)	(0.53)	(0.74)	(0.62)	(0.50)
5	1.53	0.38	0.20***	0.20***	0.19***	0.20***	0.41	−0.12	0.51	0.54
	(1.45)	(0.89)	(0.02)	(0.04)	(0.04)	(0.03)	(0.46)	(0.77)	(0.61)	(0.52)
6	2.22	−0.36	0.16***	0.19***	0.08*	0.20***	1.20**	0.20	1.89***	1.00**
	(1.58)	(0.91)	(0.03)	(0.04)	(0.05)	(0.03)	(0.49)	(0.90)	(0.71)	(0.51)
7	0.67	−0.81	0.21***	0.20***	0.16***	0.22***	0.47	−1.44	0.39	0.43
	(1.78)	(1.00)	(0.02)	(0.04)	(0.04)	(0.03)	(0.55)	(0.94)	(0.93)	(0.56)
8	0.97	−0.07	0.17***	0.23***	0.14***	0.20***	1.58***	0.20	0.57	1.29**
	(1.79)	(1.10)	(0.02)	(0.04)	(0.04)	(0.03)	(0.56)	(0.76)	(0.68)	(0.60)
9	−0.19	−0.04	0.24***	0.22***	0.14***	0.26***	2.31***	0.77	1.46	1.83***
	(1.95)	(1.30)	(0.03)	(0.05)	(0.06)	(0.03)	(0.64)	(0.86)	(0.93)	(0.61)
10	−0.66	−0.83	0.20***	0.22***	0.17***	0.21***	1.13***	0.40	0.72	1.35***
	(1.18)	(0.89)	(0.02)	(0.04)	(0.04)	(0.03)	(0.39)	(0.69)	(0.69)	(0.45)
11	1.45	−0.01	0.20***	0.28***	0.15***	0.25***	0.26	0.30	0.93	0.73
	(1.86)	(1.46)	(0.03)	(0.05)	(0.05)	(0.03)	(0.69)	(1.04)	(0.87)	(0.82)
12+	−0.15	−0.74	0.23***	0.27***	0.20***	0.25***	1.14***	0.85	1.00*	1.06**
	(1.26)	(0.84)	(0.02)	(0.03)	(0.03)	(0.03)	(0.41)	(0.63)	(0.59)	(0.44)
Obs	2096	4042	5262	3427	3048	5652	1013	512	524	994
Adj. R^2	.062	.033	.084	.059	.072	.072	.11	.15	.16	.13
FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Source: Authors' elaboration.

Notes: Municipal clustered standard errors in parentheses. Year 12+ (12 or more years) collapses long-term diagnosis effects.

FEM: Females. LSES: Lower socioeconomic status (quintiles 1-3). HbA1c: Percentage of glycosylated hemoglobin.

HH NRF: Household percentage of non-recommended food expenditure over total food expenditure.

FE: Fixed effects related to age-at-diagnosis-by-sex and municipality.

* p<0.1, ** p<.05, *** p<.01.

Figures Legends

Figure 1: To present the trajectories, points represent predictions (not coefficients) from the pooled cross-sectional event-study specification. Vertical bars indicate 95% confidence intervals. The omitted category corresponds to individuals interviewed in the year of diagnosis (Year 0). The red horizontal line indicates the intercept. Year 12+ (12 or more years) collapses long-term diagnosis effects. All specifications include fixed effects related to age-at-diagnosis-by-sex and municipality.

Figure 2: To present the trajectories, points represent predictions (not coefficients) from the pooled cross-sectional event-study specification. Vertical bars indicate 95% confidence intervals. The omitted category corresponds to individuals interviewed in the year of diagnosis (Year 0). The red horizontal line indicates the intercept. Year 12+ (12 or more years) collapses long-term diagnosis effects. All specifications include fixed effects related to age-at-diagnosis-by-sex and municipality.

Figure 3: To present the trajectories, points represent predictions (not coefficients) from the pooled cross-sectional event-study specification. Vertical bars indicate 95% confidence intervals. The omitted category corresponds to individuals interviewed in the year of diagnosis (Year 0). The red horizontal line indicates the intercept. Year 12+ (12 or more years) collapses long-term diagnosis effects. All specifications include fixed effects related to age-at-diagnosis-by-sex and municipality. The criteria for restricting the subsamples were based on the national median expenditure on recommended food per household.

Figure B1: To present the trajectories, points represent predictions (not coefficients) from the pooled cross-sectional event-study specification. Vertical bars indicate 95% confidence intervals. The omitted category corresponds to individuals interviewed in the year of diagnosis (Year 0). The red horizontal line indicates the intercept. Year 12+ (12 or more years) collapses long-term diagnosis effects. All specifications include fixed effects related to age-at-diagnosis-by-sex and municipality.

Figure B2: To present the trajectories, points represent predictions (not coefficients) from the pooled cross-sectional event-study specification. Vertical bars indicate 95% confidence intervals. The omitted category corresponds to individuals interviewed in the year of diagnosis (Year 0). The red horizontal line indicates the intercept. Year 12+ (12 or more years) collapses long-term diagnosis effects. All specifications include fixed effects related to age-at-diagnosis-by-sex and municipality. The criteria for restricting the subsamples were the median age of T2DM diagnosis.

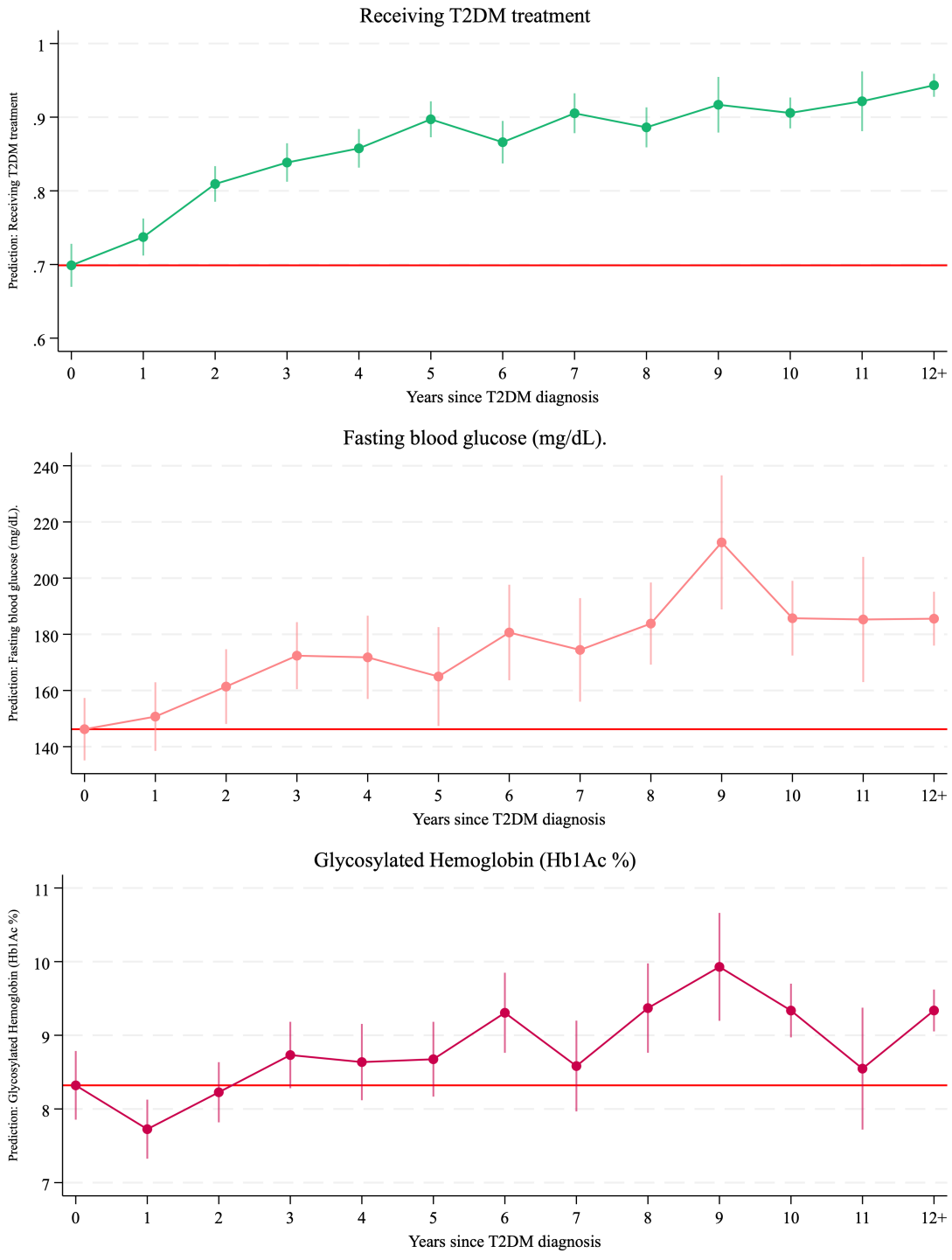
Figure B3: To present the trajectories, points represent predictions (not coefficients) from the pooled cross-sectional event-study specification. Vertical bars indicate 95% confidence intervals. The omitted category corresponds to individuals interviewed in the year of diagnosis (Year 0). The red horizontal line indicates the intercept. Year 12+ (12 or more years) collapses long-term diagnosis effects. All specifications include fixed effects related to age-at-diagnosis-by-sex and municipality. The criteria for stratifying the subsamples were SES quintiles 1–3 for lower SES and quintiles 4–5 for higher SES.

Figure B4: To present the trajectories, points represent predictions (not coefficients) from the pooled cross-sectional event-study specification. Vertical bars indicate 95% confidence intervals. The omitted category corresponds to individuals interviewed in the year of diagnosis (Year 0). The red horizontal line indicates the intercept. Year 12+ (12 or more years) collapses long-term diagnosis effects. All specifications include fixed effects related to age-at-diagnosis-by-sex and municipality. Trajectories are stratified by hypertension status.

Figure B5: To present the trajectories, points represent predictions (not coefficients) from the pooled cross-sectional event-study specification. Vertical bars indicate 95% confidence intervals. The omitted category corresponds to individuals interviewed in the year of diagnosis (Year 0). The red horizontal line indicates the intercept. Year 12+ (12 or more years) collapses long-term diagnosis effects. All specifications include fixed effects related to age-at-diagnosis-by-sex and municipality. The criteria for restricting

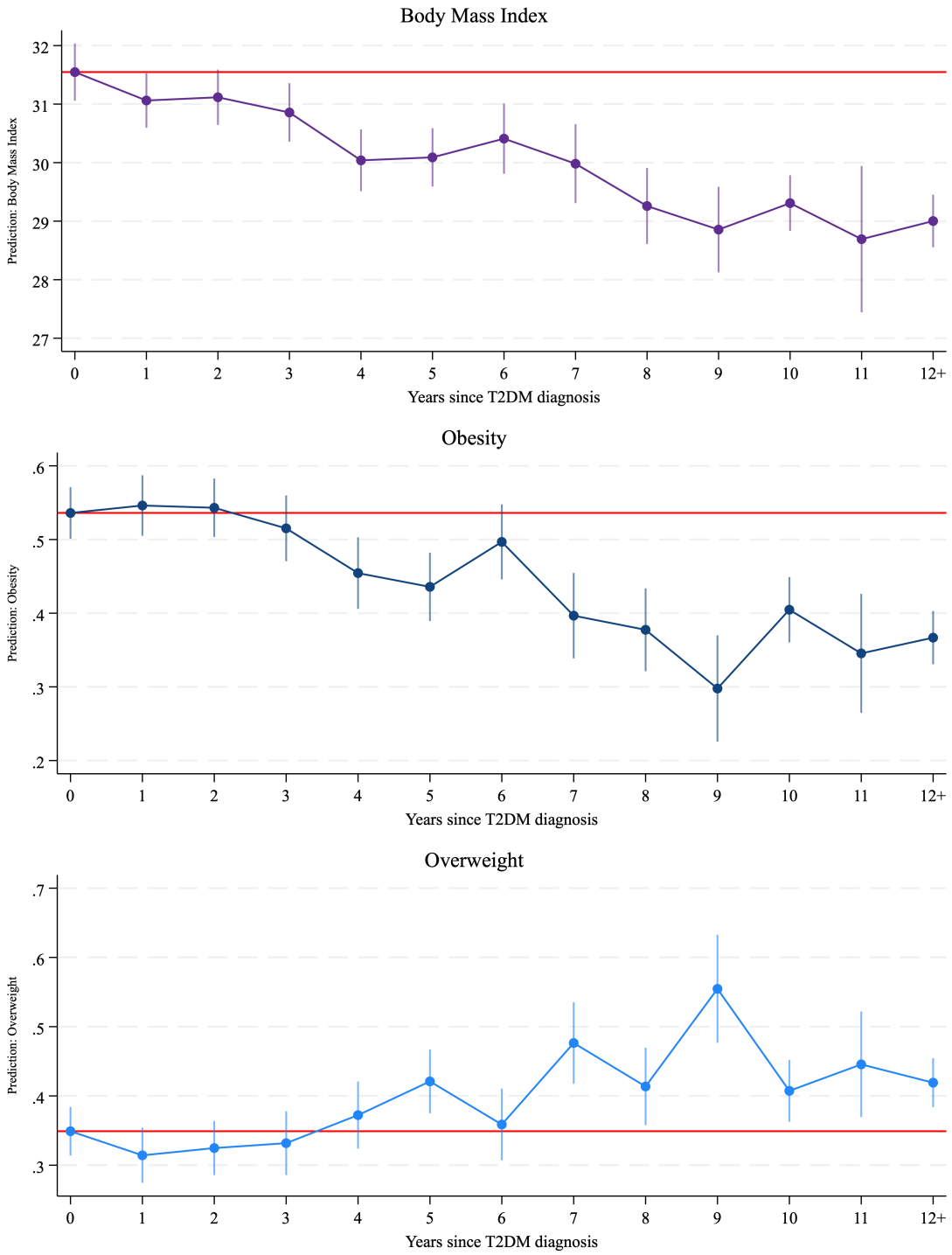
the younger subsample in Panel B were based on the mean age at diagnosis (49.9) minus one standard deviation (12.72) = 37 years.

Figure B6: To present the trajectories, points represent predictions (not coefficients) from the pooled cross-sectional event-study specification. Vertical bars indicate 95% confidence intervals. The omitted category corresponds to individuals interviewed in the year of diagnosis (Year 0). The red horizontal line indicates the intercept. Year 12+ (12 or more years) collapses long-term diagnosis effects. All specifications include fixed effects related to age-at-diagnosis-by-sex and municipality. Placebo specifications should not plausibly respond to time since diabetes diagnosis.



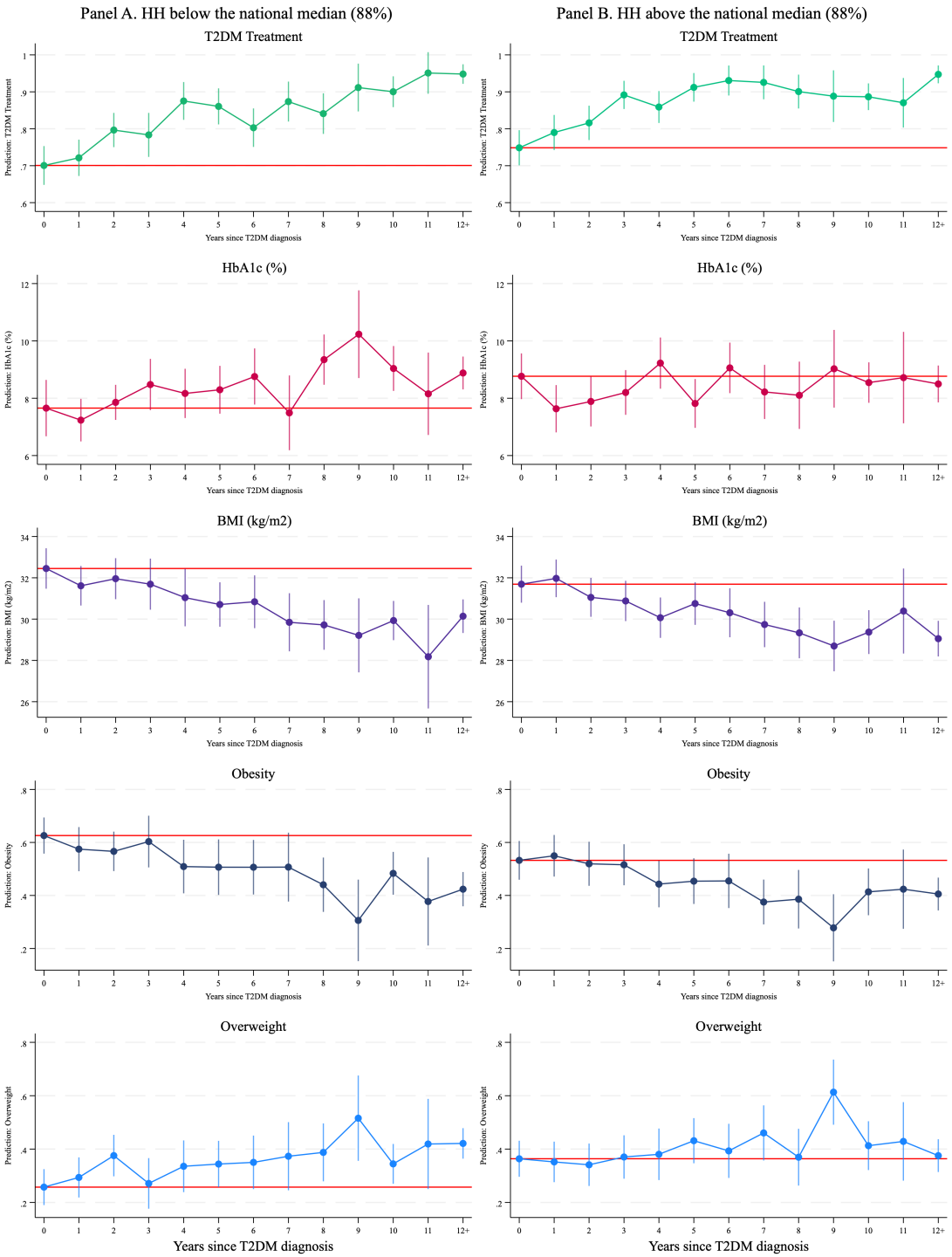
Note: 12+ (12 or more years) collapses long-term diagnosis effects.

Figure 1: Post-Diagnosis Dynamics of Treatment Use and Glycemic Biomarkers



Note: 12+ (12 or more years) collapses long-term diagnosis effects.

Figure 2: Post-Diagnosis Dynamics of Adiposity



Note: 12+ (12 or more years) collapses long-term diagnosis effects.

Figure 3: Post-Diagnosis Dynamics by Household Recommended-Food Expenditure Share Below or Above the National median

Appendices

Appendix A Tables

A.1 Evolution of monthly outcomes after the diagnosis

Table A1: First 12 Months After T2DM diagnosis

	Behavioral and care-related outcomes [1-2]		Metabolic outcomes [3-5]			Clinical outcomes [6-7]	
	1	2	3	4	5	6	7
	Treatment	HH NRF	BMI	Obesity	Overweight	Glucose	HbA1c
Intercept	0.578***	10.579***	31.148***	0.836***	0.520***	162.955***	10.754***
	(0.052)	(2.025)	(1.208)	(0.055)	(0.073)	(20.380)	(1.218)
Month							
1	0.062	1.489	0.666	−0.003	−0.016	−6.863	−0.045
	(0.075)	(2.687)	(1.455)	(0.067)	(0.103)	(31.901)	(1.558)
2	0.118*	1.543	1.015	0.065	0.069	2.069	−1.573
	(0.067)	(2.308)	(1.495)	(0.077)	(0.105)	(35.044)	(1.742)
3	0.093	−0.457	0.589	0.063	−0.062	−40.029	−3.353**
	(0.073)	(2.501)	(1.699)	(0.069)	(0.112)	(30.127)	(1.544)
4	0.124	2.601	1.624	0.098	0.077	−7.861	−2.799
	(0.079)	(2.740)	(1.589)	(0.064)	(0.108)	(43.339)	(2.029)
5	0.217***	1.931	2.978*	0.093	0.142	16.431	−0.153
	(0.078)	(3.398)	(1.697)	(0.085)	(0.122)	(36.149)	(1.719)
6	0.103	−0.632	0.361	0.058	0.072	−15.937	−2.188
	(0.067)	(2.382)	(1.337)	(0.070)	(0.089)	(36.323)	(2.207)
7	0.244***	−1.632	0.999	0.153**	0.232*	−75.193**	−3.771*
	(0.081)	(3.098)	(1.604)	(0.072)	(0.118)	(33.267)	(2.128)
8	0.206***	−1.851	0.815	0.033	0.089	−56.298*	−4.646***
	(0.078)	(2.971)	(1.601)	(0.080)	(0.113)	(31.036)	(1.726)
9	0.187**	−3.374	−0.376	0.164*	0.039	−24.957	4.562**
	(0.081)	(3.018)	(1.806)	(0.096)	(0.138)	(51.674)	(1.868)
10	0.294***	1.753	0.249	0.076	0.101	−55.940*	−4.401***
	(0.076)	(2.842)	(1.976)	(0.073)	(0.120)	(33.291)	(1.372)
11	0.152	−1.539	0.085	−0.032	−0.251*	34.326	−1.497
	(0.112)	(3.696)	(1.887)	(0.089)	(0.133)	(41.447)	(2.011)
12	0.291***	0.317	−1.310	−0.011	−0.087	13.523	−1.849
	(0.056)	(2.141)	(1.297)	(0.060)	(0.078)	(22.013)	(1.263)
Obs	2207	1477	1450	1483	1483	363	268
Adj. R^2	.11	.028	.13	.057	.079	.12	.13
FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Source: Authors' elaboration.

Notes: Municipal clustered standard errors in parentheses.

Treatment: Currently receiving T2DM treatment. HH NRF: Household percentage of non-recommended food expenditure over total food expenditure.

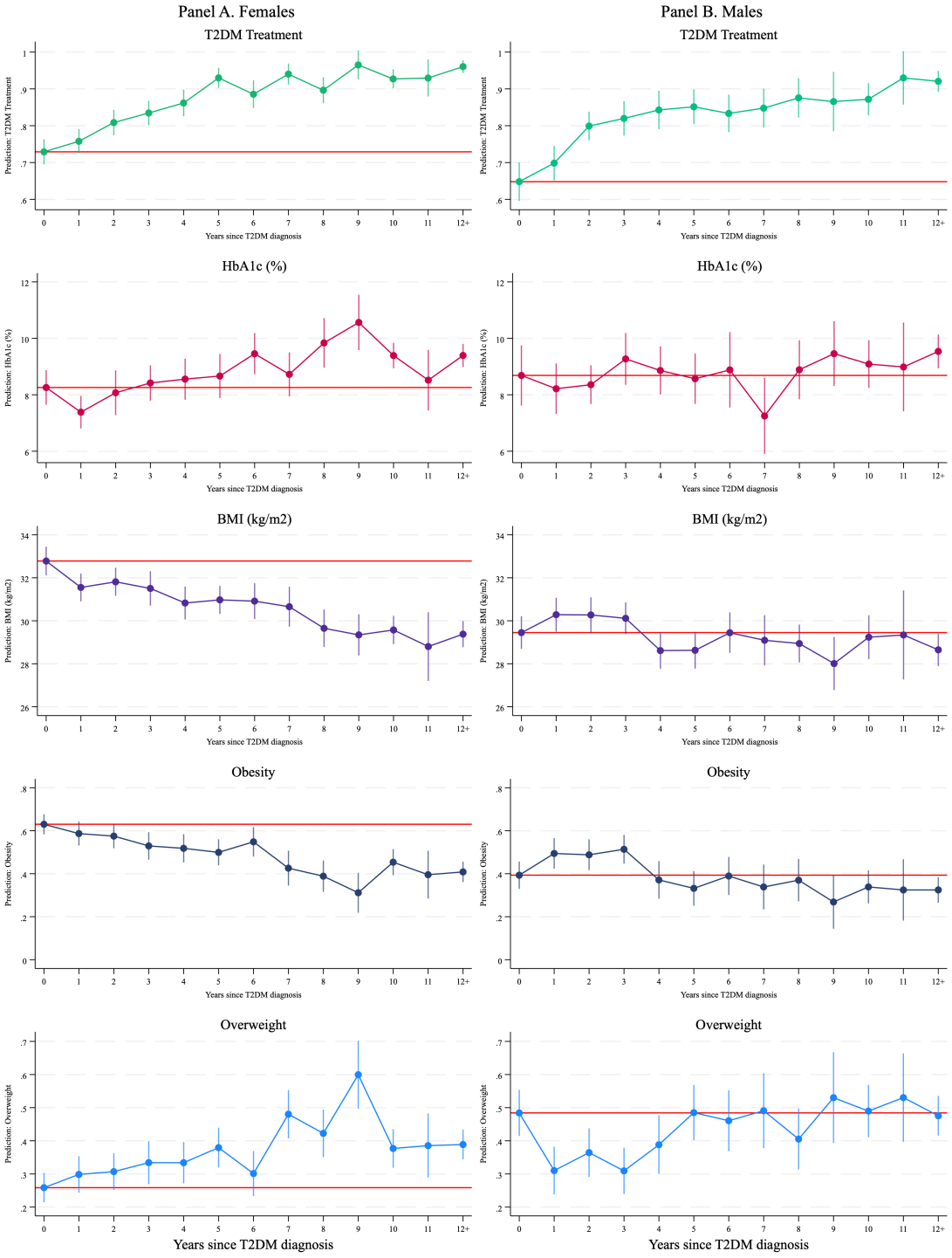
BMI: Body Mass Index (kg/m^2). Overweight is defined as BMI 25.0–29.9. Obesity as BMI $\geq$ 30.0 kg/m^2.

Glucose: Fasting blood glucose (mg/dL). HbA1c: Percentage of glycosylated hemoglobin.

FE: Fixed effects related to Age-at-diagnosis-by-sex and municipality.

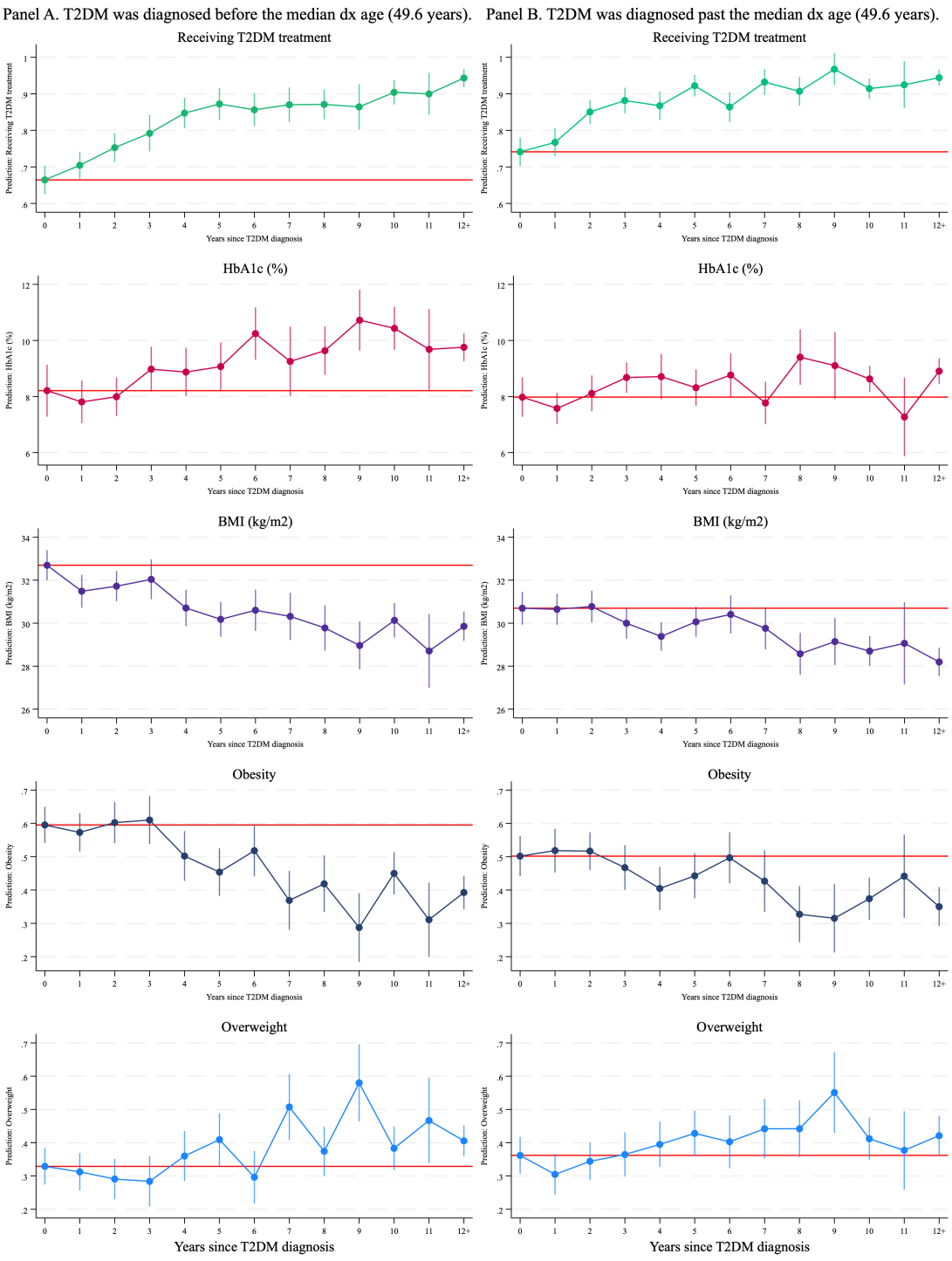
* p<0.1, ** p<.05, *** p<.01.

Appendix B Figures



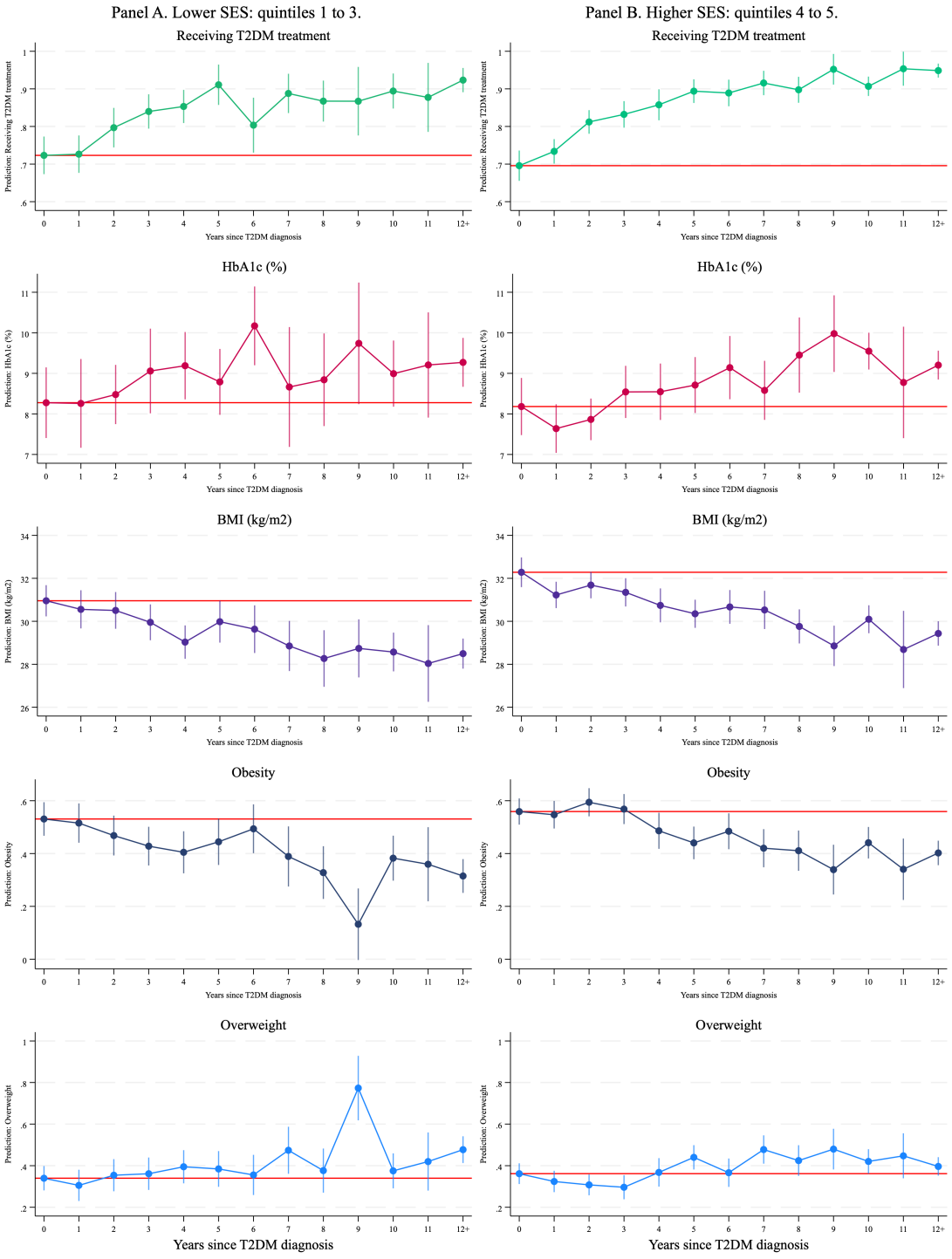
Note: 12+ (12 or more years) collapses long-term diagnosis effects.

Figure B1: Sex Heterogeneity.



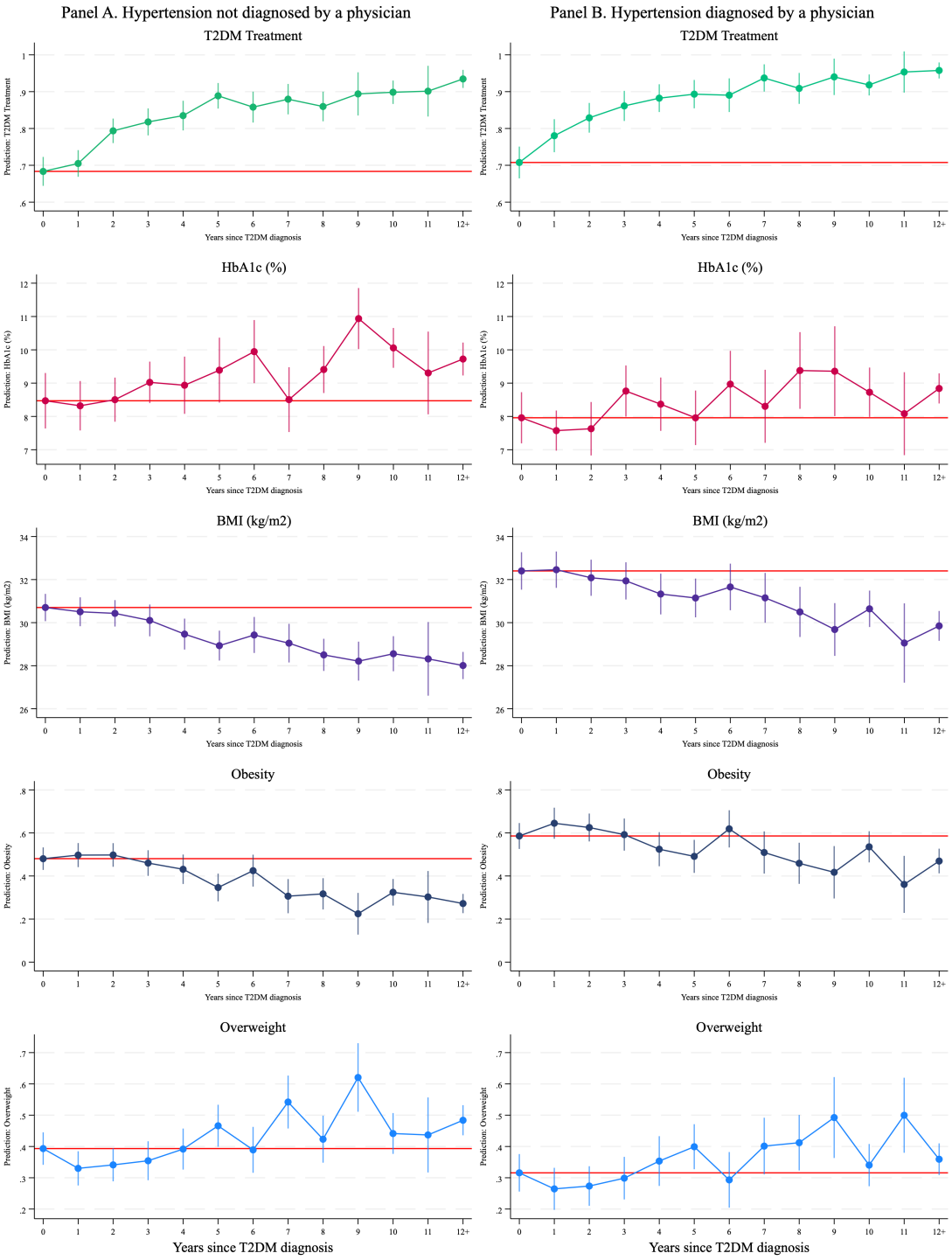
Note: 12+ (12 or more years) collapses long-term diagnosis effects.

Figure B2: Age of diagnosis Heterogeneity



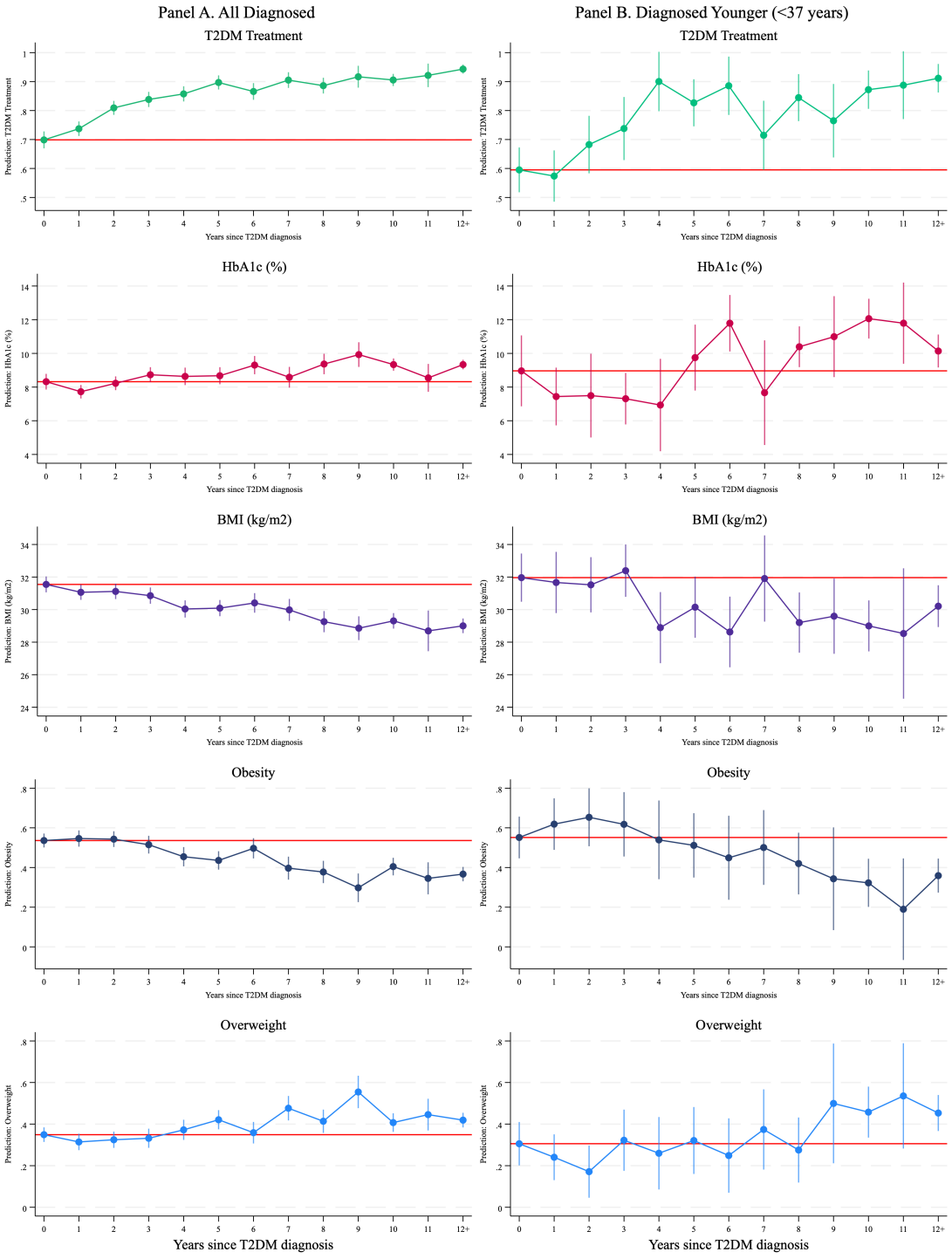
Note: 12+ (12 or more years) collapses long-term diagnosis effects.

Figure B3: SES Heterogeneity



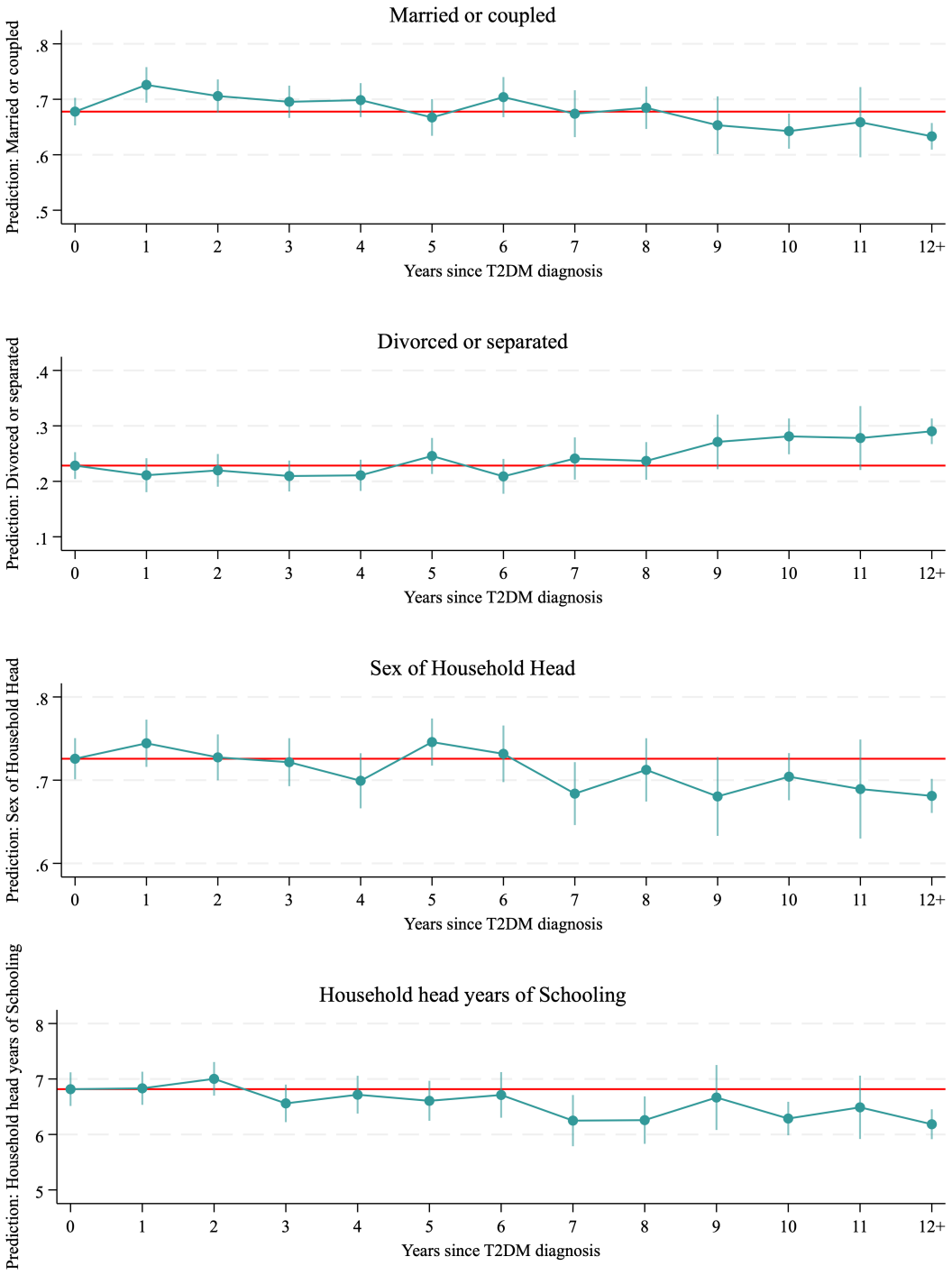
Note: 12+ (12 or more years) collapses long-term diagnosis effects.

Figure B4: Hypertension Status Heterogeneity



Note: 12+ (12 or more years) collapses long-term diagnosis effects.

Figure B5: Selective mortality check



Note: 12+ (12 or more years) collapses long-term diagnosis effects.

Figure B6: Placebo specifications