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Are diabetes and blood sugar control associated with the diagnosis of eye diseases? An English prospective observational study of glaucoma, diabetic eye disease, macular degeneration and cataract diagnosis trajectories in older age

Caitlin Lin [1], Stephen Jivraj [2]

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[1]UCL Global Business School for Health, London, UK
[2]UCL Institute of Epidemiology and Health Care, London, UK

Correspondence to
Dr Stephen Jivraj;
stephen.jivraj@ucl.ac.uk

ABSTRACT

Background The growing global burden of diabetes suggests a currently unrealised growth in prevalence of eye disease. This prospective observational study addresses gaps in evidence of blood sugar control as a risk factor for the diagnosis of glaucoma, diabetic eye disease, macular degeneration and cataract using waves 2–9 (2004–2019) of the English Longitudinal Study of Ageing.

Methods Logistic regression modelling is used to predict the probability of self-reported diagnosis of four eye conditions separately over a 14-year period in a community-dwelling sample in England. Analysis of approximately 29 000 person observations over eight study waves from around 5600 participants for each eye disease is conducted with an average of 5.7 waves per participant. Participants' baseline blood sugar control is categorised as non-diabetic (diabetes not previously diagnosed and glycated haemoglobin (HbA1c)<6.5), controlled (diabetes previously diagnosed and HbA1c<6.5), uncontrolled (diabetes previously diagnosed and HbA1c≥6.5) and undiagnosed (diabetes not previously diagnosed and HbA1c≥6.5). Controls at baseline for age, sex, physical activity level, body mass index and smoking status are included in the regression analysis.

Results The mean age of the sample is 66 and 53% are female. The main finding from this study is that older adults in England who are controlling a diabetes diagnosis have a lower probability of developing glaucoma, diabetic eye disease or macular degeneration compared with those either without a diabetes diagnosis or with uncontrolled diabetes. Compared with those with controlled diabetes, the adjusted odds of developing glaucoma was 1.29 times higher (95% CI 1.01 to 1.65) among those not diabetic; the adjusted odds of developing diabetic eye disease was 1.20 times higher (95% CI 1.00 to 1.45) among those with uncontrolled diabetes; and the adjusted odds of developing macular degeneration was 1.38 times higher (95% CI 1.04 to 1.82) among those with undiagnosed diabetes. There was no statistically significant difference in the probability of developing cataracts by category of blood sugar control.

Conclusion This study illustrates the importance of blood sugar control in the development of eye diseases and therefore supports more regular screening measures for eye disease in older age among groups at risk of diabetes.

STRENGTHS AND LIMITATIONS OF THIS STUDY

⇒ This study uses a large nationally representative sample of older adults followed up over a 14-year period.
⇒ The inclusion of blood analytes and covariate data sets the current study apart from most evidence in the research area.
⇒ Longitudinal attrition reduces the sample size by more than half over the period of study.
⇒ The analysis is limited by a subjective measurement of physical activity that does not stipulate a time duration for activities.

INTRODUCTION

Diabetes is substantially contributing to the global burden of disease with more than half a billion people estimated to be living with the disease in 2021, and projected to increase to almost 800 million by 2045.[1] In England, the prevalence of diabetes diagnosis more than doubled among working age people during the 2000s from 2.8% to 6.8%.[2] This trend is concerning for clinical ophthalmology given the relationship between diabetes and vision loss due to a number of eye diseases that become more common in older age.[3–7] The direction of

causality between diabetes and eye conditions appears complicated, possibly bi-directional and dependent on the eye condition.

The most established pathway from diabetes to vision loss is as a consequence of diabetic retinopathy and diabetic macular oedema, which are thought to result from excessive blood glucose (ie, poor glycaemic control) on the vessels that produces microvascular damage.[3 4 8–10] People with diabetes also have an increased risk of glaucoma,[11–18] macular degeneration[12 19–26] and cataract,[11 12 24 27–29] each of which can cause vision loss. There are theorised pathways between diabetes and glaucoma, macular degeneration and cataract; however, the epidemiological evidence is mixed. There are suggestions that greater monitoring of the eye among people with diabetes is responsible for the association between diabetes and some eye diseases.[13] Diabetic retinopathy is also known to lead to the development of cataract and glaucoma, which may explain their association with diabetes.[18 30–32]

Glycaemic control among diabetic populations has been shown to reduce the likelihood of diabetic retinopathy. Results from the UK Prospective Diabetes Study and the Diabetic Control and Complications Trial indicate that better monitoring and glycaemic control significantly reduce the advancement of retinopathy in diabetic patients.[33–36] Aside from its effects on retinopathy, long-term glycaemic control has been associated with a decrease in macular oedema incidence in diabetic patients.[37–39] The evidence is mixed for glaucoma, macular degeneration and cataract. Linked Danish population-based databases and medical registries show that the probability of receiving glaucoma treatment was the same among those diagnosed with diabetes, irrespective of glycaemic control.[15] Analysis of IQVIA Medical Research Data for patients aged 40 and over with a newly reported diagnosis of type 2 diabetes who were followed for a mean of 6 years between 1995 and 2019 shows that development of age-related macular degeneration is more likely if patients had poor glycaemic control at baseline.[20] In a recent review of risk factors for cataract in those with type 2 diabetes, glycaemic control was consistently reported.[24]

The current evidence lacks analysis using nationally representative samples, often relies on short periods of follow-up and rarely compares to non-diabetic populations. The current study aims to address these gaps in the existing knowledge of glycaemic control in the development of eye disease using a representative sample of older adults in England. The paper intends to answer the following research question:

> To what extent is diabetes (absence, controlled, uncontrolled or undiagnosed) associated with the development of glaucoma, diabetic eye disease (including diabetic retinopathy), macular degeneration (including age-related maculopathy) and cataracts among the older adult population over time?

METHODS

Study data

To address the current study's research question, the English Longitudinal Study of Ageing (ELSA), an ongoing panel study focused on a nationally representative cohort of adults aged 50 years and older in England, is used. ELSA, which commenced in 2002 (wave 1), was designed to capture a holistic view on the ageing experience of those residing in private households in England using computer-assisted in-person interviews (main interview) and self-completion questionnaires every 2 years, as well as nurse visits to collect biomarkers every 4 years.[40] This study uses data collected from the main interview at ELSA waves 2–9 and the first nurse visit at ELSA wave 2. ELSA aims to explore the ways in which the different aspects of health and health behaviours, socioeconomic position, and social and civic participation evolve over time as people approach and enter retirement.[40]

The sample for the first wave of ELSA in 2002 was recruited from the 1998, 1999 and 2001 Health Survey for England (HSE) using a four-stage sampling strategy, whereby participants were required to meet criteria set in order to be issued an interview at wave 1: (1) responded to HSE, (2) was age-eligible, (3) remained alive and (4) gave permission to be re-contacted in future waves.[40 41]

The participants meeting the ELSA selection criteria interviewed at wave 1 are referred to as *core members*. There were 11 391 productive interviews with core members at wave 1, a response rate of 67% of the issued sample. By wave 2, 2612 core members were lost to follow-up (ie, did not complete an interview or a nurse visit at wave 2). A further 2551 core members who were interviewed at wave 2 did not provide blood samples and are therefore excluded from this study. This study included 5672 core members (hereafter referred to as participants) who provided a blood sample at wave 2 and who had not reported an eye disease at wave 1 or who reported no longer having an eye disease at wave 2.

Measures

Outcome

Eye disease is reported in ELSA at the main interview. In separate questions, participants are asked whether a doctor or optician has ever told them they currently have or have had glaucoma or suspected glaucoma, diabetic eye disease (including diabetic retinopathy), macular degeneration (including age-related maculopathy) and cataracts. These four eye diseases serve as separate binary outcome variables at ELSA waves 2–9 for the current study. Participants are asked in each subsequent wave whether they still have a previously reported eye disease and those who reply with no are treated as disease-free at each wave. This accounts for less than 0.5% of the sample at each wave and is almost exclusively a result of the participant reporting they never had the disease (ie it was incorrectly reported at a previous wave).

Exposure

ELSA participants were asked at the main interview at wave 2 whether a doctor had ever told them they had diabetes or high blood sugar. Participants who consented to a nurse visit and had a blood sample taken at wave 2 had their blood analysed for glycated haemoglobin (HbA1c). HbA1c is the blood glucose level in the body. An HbA1c value at or above 6.5% indicates a participant in the diabetic range according to clinical diagnosis thresholds.[42] Participants are categorised in the exposure variable as non-diabetic (diabetes not previously diagnosed and HbA1c<6.5), controlled (ie, diabetes previously diagnosed and HbA1c<6.5), uncontrolled (diabetes previously diagnosed and HbA1c≥6.5), undiagnosed (diabetes not previously diagnosed and HbA1c≥6.5).

Covariates

Control variables included in the regression analysis are physical activity, body mass index (BMI), smoking status, age and sex. These measures were included as known determinants of both eye disease and diabetes. Physical activity is categorised in this paper as light, moderate or intense using questions in the main interview at wave 2 that ask ELSA participants how frequently they do moderate and vigorous sports or activities and the level of physical activity in their main job. Those who hardly ever or never partake in moderate or vigorous activity and have a sedentary occupation or do not have a main job are categorised as lightly physically active. Participants who partake in moderate or vigorous activity at least a couple of times per month or spend most of the time in their main job standing are categorised as moderately physically active. Partaking in moderate or vigorous activities more than once a week or having a job involving physical work is categorised as intensely physically active. The physical activity questions were taken from a validated physical activity instrument and correlate with biomarkers.[43 44] BMI was measured from height and weight measurements taken at the wave 2 nurse visit. Smoking status is measured by whether the participant mentioned that they currently smoke cigarettes at wave 2. Age at wave 2 was recorded in a single year and sex was recorded as male or female and verified at ELSA wave 1 from the HSE data collection.

Binary measures for injection of insulin, taking of diabetes medication and training for diabetes management as well as length of time in years since diabetes diagnosis, were used in descriptive analysis for those with a diabetes diagnosis. These variables are not used in the regression analysis because of the small sample of participants with controlled or uncontrolled diabetes.

Statistical analysis

A logistic regression model is fitted for each eye disease outcome, with between one and eight observations for each participant between ELSA waves 2–9. Robust SEs are clustered at the participant level. Each model contains explanatory dummy variables for diabetes controls with controlled diabetes diagnosis as the reference group and parameters added for each control variable, including a linear term for wave of observation and quadratic terms for age and wave to take account of the non-linear relationship between eye disease and age and eye disease and time. There is no attempt made to isolate cohort effects from age or wave and therefore the age and wave estimates should not be considered as separated from the cohort. An interaction term is added between diabetes control and wave to test whether the development of eye disease varies over time for those without diabetes, with controlled diabetes, with uncontrolled diabetes and with undiagnosed diabetes. The models are adjusted for sample weights that reduce bias from the ELSA baseline to selection into the blood sample and aim to ensure representativeness.

Item non-response at wave 2 (ie, participants at wave 2 who provided information for some but not all measures used in the study) reduces the complete case study sample by almost 12%. Table 1 shows the extent to which variables contribute to item non-response. Diabetes control is missing for 7% of respondents, BMI is missing for 4%, glaucoma, diabetic eye disease and macular degeneration are missing for 1% and cataract is missing for less than 1%. There is no missing data at wave 2 for age, sex and physical activity level. Multiple imputation by chained equations is used to impute missing values (20 imputed datasets) for the regression analysis, though the results were not substantively different from a complete case analysis.

The complete case participants used in the regression analysis are 5603 for diabetic eye disease, 5607 for macular degeneration, 5612 for glaucoma and 5646 for cataracts. Participants contributed, on average, 5.7 waves with around 29 000 unbalanced participant-waves in each of the four models. There was no attempt made to adjust for longitudinal attrition because of the uncertainty on the reason for non-response at a subsequent wave by participants. It is expected that some participants would have died between ELSA waves 2 and 9. There is no mortality information currently provided to researchers on ELSA participants after wave 5. By wave 9, more than half of the wave 2 samples were missing. Longitudinal balanced sample weights are available from the ELSA team for participants who have provided data in each wave up to wave 9. Application of these weights using a balanced sample of around 2000 participants reduced the complete sample at wave 2 by more than 50%. Results (not reported here) using these balanced sample weights are substantively similar to those using the cross-sectional blood sample weights from wave 2 reported in the analysis in the current paper.

Model results are presented using predicted probabilities of each eye disease outcome for each category of blood sugar control (ie, not diabetic, controlled, uncontrolled and undiagnosed). The predictions are adjusted at the means of all covariates described above. The models are fitted in Stata V.18 using the *logit* command and predicted values derived using the *margins* command.

Table 1 Summary statistics for English Longitudinal Study of Ageing participants with no eye disease at or before wave 2

Variable	Wave	Mean (SD) or %	% Missing
Outcomes			
Glaucoma	2	1.67%	1.06
	3	2.83%	14.35
	4	3.91%	23.38
	5	5.17%	27.80
	6	5.53%	32.97
	7	6.52%	40.81
	8	7.31%	47.39
	9	8.58%	53.72
Retinopathy	2	0.57%	1.22
	3	1.12%	14.56
	4	1.43%	23.57
	5	1.74%	28.03
	6	2.30%	33.20
	7	2.68%	40.99
	8	3.12%	47.46
	9	3.03%	53.79
Diabetic macular oedema	2	0.93%	1.15
	3	2.43%	14.44
	4	3.45%	23.47
	5	4.50%	27.96
	6	5.84%	33.13
	7	6.31%	40.96
	8	7.82%	47.44
	9	8.71%	53.74
Cataracts	2	6.47%	0.46
	3	15.24%	13.79
	4	16.42%	22.92
	5	21.37%	27.33
	6	25.56%	32.60
	7	30.74%	40.51
	8	35.75%	47.06
	9	41.85%	53.42
Diabetes control			6.66
No diabetes	2	90.85%	
Controlled diabetes	2	2.84%	
Uncontrolled diabetes	2	3.65%	
Undiagnosed diabetes	2	2.65%	
Sex			0.00
Female	2	52.87%	
Age	2	65.87 (9.94)	0.00
Physical activity level			0.00
Sedentary	2	13.61%	
Moderate activity	2	28.27%	
Intense activity	2	58.12%	
Body mass index	2	27.83 (4.81)	4.27
Smoker	2	16.34%	0.18
Unweighted N		5672	

Patient and public involvement

Patients and the public were not involved in the design or implementation of the study, or in the interpretation or writing up of results. Dissemination of findings to study participants will be conducted as part of the broader dissemination activity of the ELSA study (https://www.ELSA-project.ac.uk).

RESULTS

Table 1 shows weighted summary statistics for the analytical sample across each of the four eye diseases between waves 2 and 9, diabetes control at wave 2 and the control variables at wave 2 for ELSA participants who did not report an eye disease at wave 1 or reported no longer having the disease at wave 2. Less than 1% of participants reported a new diagnosis of either diabetic eye disease or macular degeneration at wave 2, 1.7% reported a new diagnosis of glaucoma and 6.5% reported a new diagnosis of cataract. The eye diseases each progressively increase in prevalence across ELSA waves 2–9, with an increase of around six times the percentage of participants reporting a diagnosis from wave 2 to wave 9.

Diabetes was absent for more than 90% of participants at wave 2. Almost 3% reported a diagnosis of diabetes but had blood sugar in the normal range, a further 3.7% reported a diagnosis but had blood sugar in the clinically diabetic range and 2.7% did not report a diagnosis but had blood glucose in the clinically diabetic range. Most participants (58.1%) at wave 2 engaged in moderate or vigorous physical activity more than once per week or had a main job requiring intense physical activity. The mean BMI value of participants at wave 2 was 27.8. A minority of participants were currently smoking (16.3%) at wave 2. A little over half (52.9%) of participants were female and the mean age at wave 2 was 65.9 years.

Online supplemental table S1 shows that those who were not diabetic were more likely to be female, younger, taking physical activity and have lower BMI compared with those with controlled, uncontrolled or undiagnosed diabetes. There was little difference in these covariates across those with diabetes, whether controlled, uncontrolled or undiagnosed. Those controlling a diabetes diagnosis were likely to smoke compared with those who were not diabetic or had uncontrolled diabetes. Those

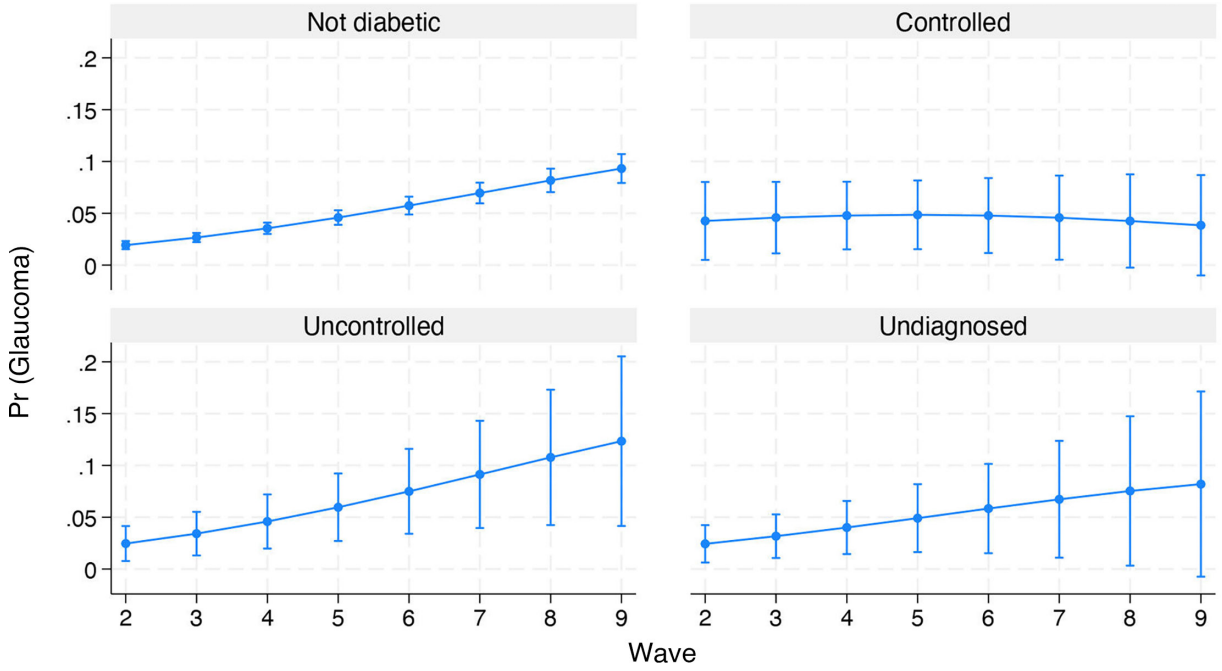

Figure 1 Adjusted predicted probability of glaucoma by diabetes control and wave of study (n=5612). See online supplemental table S3 for full model estimates.

with undiagnosed diabetes were most likely to be smokers. There was a considerable difference in the proportion of participants with controlled diabetes compared with uncontrolled diabetes who are injecting insulin and taking medication for diabetes (see online supplemental table S2). There was little difference in the percentage who had received training in diabetes management or the length of time since diabetes diagnosis between those with controlled and uncontrolled diabetes (see online supplemental table S2).

Figures 1–4 show the adjusted predicted probability of each eye disease over ELSA waves 2–9 by category of diabetes control using logistic regression models, controlling for physical activity level, BMI, smoking status, age and sex. The error bars on each plot show 95% CIs for each predicted value. The full model results are shown in online supplemental table S3.

The predicted probability of glaucoma became progressively greater for those who were not diabetic or who were not controlling their diagnosed diabetes (see figure 1).

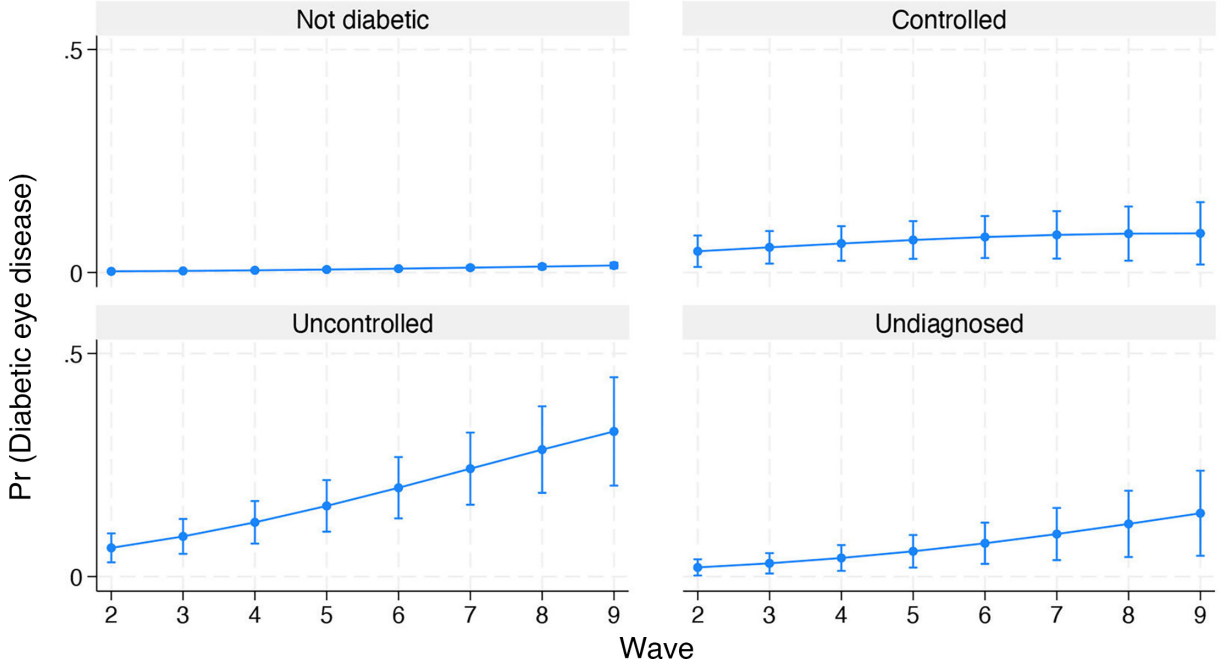

Figure 2 Adjusted predicted probability of diabetic eye disease by diabetes control and wave of study (n=5603). See online supplemental table S3 for full model estimates.

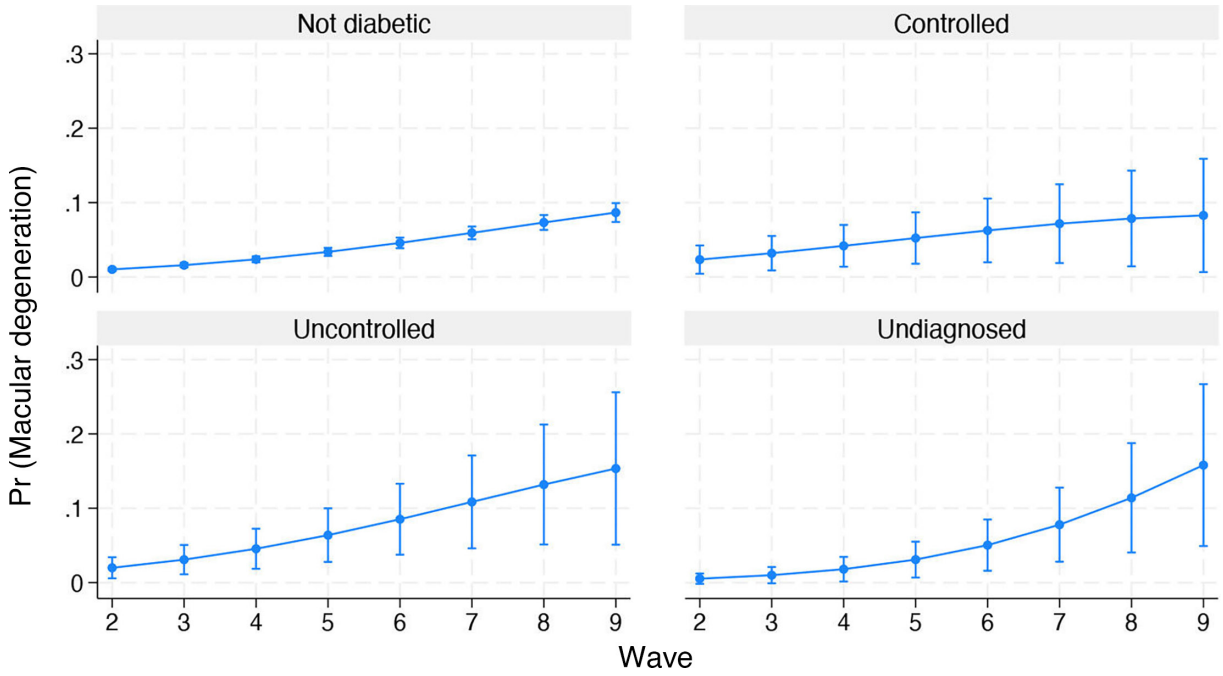

Figure 3 Adjusted predicted probability of age-related macular degeneration by diabetes control and study wave (n=5607). See online supplemental table S3 for full model estimates.

At wave 2, 1.7% (95% CI 1.3% to 2.1%) of participants without diabetes were predicted to report glaucoma compared with 9.0% (95% CI 7.6% to 10.5%) by wave 9. For those with uncontrolled diabetes, the predicted probability of glaucoma rose from 2.2% (95% CI 1.0% to 3.9%) at wave 2 to 13.1% (95% CI 4.1% to 22.2%) by wave 9. There were no significant differences over ELSA waves between the predicted probability of glaucoma for those with controlled diagnosed diabetes or undiagnosed diabetes.

The predicted probability of diabetic eye disease increased over time for all categories of diabetes control, although there was no significant difference for the controlled diabetes group (see figure 2). The increase was greatest in absolute terms for the uncontrolled diabetes group, which was 5.4% (95% CI 2.4% to 8.3%) at wave 2

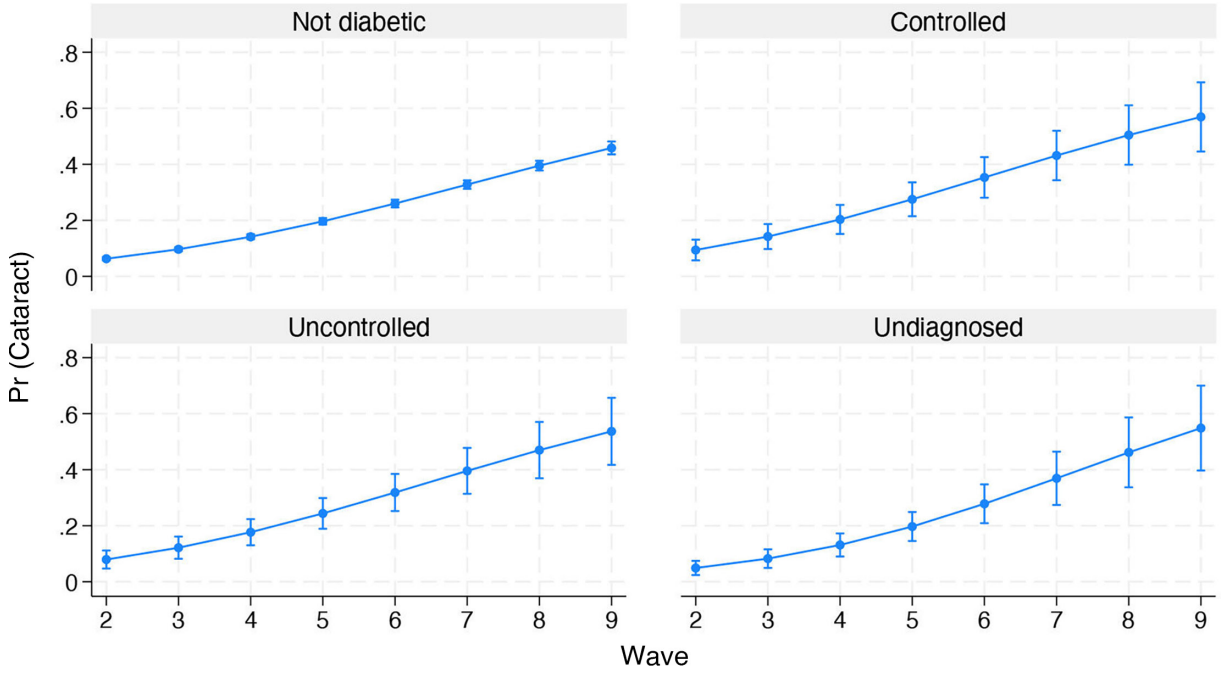

Figure 4 Adjusted predicted probability of cataract by diabetes control and wave of study (n=5646). See online supplemental table S3 for full model estimates.

compared with 30.8% (95% CI 18.4% to 43.2%) by wave 9. The relative increase was greater for the undiagnosed diabetes group, which increased its predicted probability of diabetic eye disease from 1.4% (95% CI 0.2% to 2.6%) at wave 2 compared with 15.0% (95% CI 5.0% to 25.0%) at wave 9. The increase for the non-diabetic group was marginal from 0.7% (95% CI 0.6% to 1.0%) at wave 2 to 1.3% (95% CI 0.8% to 1.9%) by wave 9.

All categories of diabetes control are shown to have increased predicted probabilities of macular degeneration over time, although the difference was not significant for the controlled diabetes group (see figure 3). The uncontrolled diabetic group increased its probability of macular degeneration from 1.7% (95% CI 0.3% to 3.1%) at wave 2 to 15.4% (95% CI 4.8% to 26.0%) by wave 9. The not diabetic group increased its predicted probability of macular degeneration from 0.8% (95% CI 0.1% to 1.0%) at wave 2 to 8.2% (95% CI 6.9% to 9.5%). The undiagnosed diabetic group had a non-linear increase in its predicted probability of macular degeneration, which increased after wave 3 from 1.0% (95% CI 0.0% to 2.1%) to 12.0% (95% CI 2.7% to 21.3%) at wave 9.

Figure 4 shows the predicted probability of a cataract diagnosis over time by category of diabetes control. All groups of diabetes diagnosis are predicted to have a higher probability of a cataract diagnosis at later waves of ELSA. The trajectory is similar across each group of diabetes control, with less than 10% at wave 2 and increasing to between 45% and 55% at wave 9.

DISCUSSION

The main finding from this study is that older adults (aged 52 and over) in England who are controlling a diabetes diagnosis do not increase their probability of developing glaucoma, diabetic eye disease or macular degeneration over a 14-year period (2004–2018). In contrast, older adults who have been told by a doctor that they have diabetes and have blood glucose levels above a clinical diagnosis threshold (ie, uncontrolled diabetics) are progressively more likely to develop glaucoma and diabetic eye disease over the same period. This finding demonstrates the importance of blood sugar control in those with a diabetes diagnosis.

The same reason may explain why those who have not been diagnosed with diabetes but have elevated blood glucose levels (HbA1c>6.5%) have heightened probabilities of developing diabetic eye disease and macular degeneration over time. A somewhat surprising finding is that older adults who have no diagnosis of diabetes and whose blood sugar is not above the clinical diabetic threshold are more likely to develop glaucoma over time relative to those controlling a diabetes diagnosis. This may reflect the small sample size of participants who are controlling their diabetes. The analysis points to no differences by diabetes control in the development of cataracts, which becomes substantially more likely for older adults, irrespective of diabetes diagnosis and control of the disease.

These findings reflect the ageing of the ELSA cohort used in the analysis and the fact that the eye diseases analysed become more common in older age for adults with and without diabetes.[45] A question that remains is what is protecting those who are controlling their diabetes from the development of certain eye diseases. The analysis in this paper controlled for baseline age, sex, physical activity level, BMI and smoking status. At face value, this suggests physical activity, obesity and smoking status are not explanations for the unexplained finding in this paper (ie, those controlling their diabetes are protected from some age-related eye diseases). Further research should explore this in more detail. Descriptive analysis of the sample in this paper shows the controlled diabetic group has lower levels of physical activity, higher BMI values but lower levels of smoking compared with the non-diabetic group.

There could be other explanations, including medical and other interventions to control and reduce blood glucose which are used by those who have their diabetes under control. Descriptive analysis shows that the uncontrolled diabetic group is more likely to inject insulin or take medicine to control diabetes compared with the controlled diabetic group. This could reflect that diabetes treatment is a surrogate for a poor history of blood glucose control.[24] There could also be an effect of the length of time since diagnosis, and therefore people are at a different stage of managing their symptoms, which is different for those who are controlling and not controlling their diabetes that explains the potential inhibiting effect of diabetes control on the development of age-related eye disease. There were no differences found between length of time since diagnosis or percentage who had received training to manage diabetes between those with controlled and uncontrolled diabetes in the current study. The severity of diabetes at diagnosis might have been mild for those in ELSA who are currently controlling their diabetes and, irrespective of their blood sugar level at diagnosis, they might have had less severe symptoms at the point of diagnosis and since.

These factors and others, such as alcohol consumption and blood pressure level, could potentially be explored using ELSA. The main limitation of such analysis, which applies to some extent to the analysis reported here, is the small sample size of those with a diabetes diagnosis in ELSA. Analysis of these participants by the suggested explanations above would not have the statistical power to test differences between groups. The current analysis is at a threshold of such concerns with only 150 participants in the group with a diabetes diagnosis who have a controlled blood sugar level at baseline. This could be addressed in further research using alternative data or by building up the waves of ELSA for people with diabetes before measuring the development of eye diseases. Further analysis of ELSA could also explore whether later-born cohorts at the same ages are likely to develop eye diseases using refreshment cohorts who have entered the study at later time points.

There are several limitations that the current study should be set against. The starkest is the longitudinal attrition that reduces the sample size by more than half over the 14-year period of analysis. This not only reduces the ability to draw statistically significant differences between the diabetes control categories, but it could potentially bias estimates of the development of eye disease. In the unlikely circumstance that the longitudinal attrition is random across the categories of diabetes control, there would be unbiased estimates. The current analysis was not able to adjust for the longitudinal attrition because of the unknown reason for withdrawal from the study and the problem of imputing values for those who have died, which becomes an increasingly more common reason for non-response at later waves in older age samples.

There is some suggestion that the potential longitudinal attrition bias is not affecting the results because estimates of the regression models using longitudinal weights for a balanced sample (ie, participants who responded to every ELSA wave between 2 and 9) were not substantively different to those reported in the paper. The robustness of this sensitivity test relies on the reduction in the bias by using the longitudinal weights. The same limitation applies to the use of blood sample weights from ELSA wave 2 used for the analysis reported in the current paper. These weights intend to reduce the non-response bias from ELSA wave 1 and selection into those who have blood samples taken at wave 2. Details on the derivation of the ELSA survey weights are available elsewhere.[27]

The analysis reported here is also limited by the imperfect operationalisation of the measures used in the analysis. Perhaps the most concerning measurement error is the physical activity variable that relies on participant interpretation of moderate and vigorous activity and does not stipulate a time duration of said activities. Adjustment for physical activity level (and BMI and smoking) does not alter the main substantive findings reported here. Similar concerns could be levied at the understanding by participants in how the diagnosis of both diabetes and eye diseases were recorded in the ELSA main interview. For example, the definition of diabetic eye disease includes both diabetic retinopathy and diabetic macular oedema, which may have been misunderstood by participants, as well as the distinction between type 1 or type 2 diabetes. Furthermore, the analysis reported here assumes that diabetes diagnosis and blood sugar are constant over the period with which eye disease is estimated. It is likely that some participants develop diabetes during the 14-year period.

Notwithstanding these limitations, there are several strengths of the current analysis. It uses a large nationally representative sample (at study baseline) of older adults that enables one of the first analyses of trajectories of eye disease development in a community-dwelling sample. The 14-year period of follow-up is much greater compared with many randomised control trials or observational site-specific studies. The inclusion of important blood analytes and covariate data is uncommon and sets the current study apart from most evidence in the research area.

The evidence reported here supports stricter screening measures for eye disease as early as possible, particularly for those in older age, those at risk of developing diabetes and those who already have diabetes. In England, this could be realised by reducing the age at which free tests are available for older adults (currently at age 60) and the frequency with which these take place (currently biennially). The difference in the propensity to develop an eye disease between groups who are controlling and not controlling their diabetes highlights the importance of the diagnosis of diabetes as early in life as possible to ensure successful management of its risk on eye health. There is currently no routine test for diabetes in England.

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Competing interests None declared.

Patient and public involvement Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

Patient consent for publication Not applicable.

Ethics approval This study involves human participants. ELSA Wave 9 received ethical approval from the South Central – Berkshire Research Ethics Committee on 10 May 2018 (17/SC/0588). ELSA Wave 8 received ethical approval from the South Central – Berkshire Research Ethics Committee on 23 September 2015 (15/SC/0526). ELSA Wave 7 received ethical approval from the NRES Committee South Central – Berkshire on 28 November 2013 (13/SC/0532). ELSA Wave 6 received ethical approval from the NRES Committee South Central – Berkshire on 28 November 2012 (11/SC/0374). ELSA Wave 5 received ethical approval from the Berkshire Research Ethics Committee on 21 December 2009 (09/H0505/124). ELSA Wave 4 received ethical approval from the National Hospital for Neurology and Neurosurgery & Institute of Neurology Joint Research Ethics Committee on 12 October 2007 (07/H0716/48). ELSA Wave 3 received ethical approval from the London Multi-Centre Research Ethics Committee on 27 October 2005 (05/MRE02/63). ELSA Wave 2 received ethical approval from the London Multi-Centre Research Ethics Committee on 12 August 2004 (MREC/04/2/006). ELSA Wave 1 received ethical approval from the London Multi-Centre Research Ethics Committee on 7 February 2002 (MREC/01/2/91). All participants of ELSA gave informed consent to participate in the study before taking part.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data may be obtained from a third party and are not publicly available. ELSA data are available from the UK Data Services. Researchers, students and teachers from any discipline, organisation or country may register with the UK Data Service and obtain these data. Other uses should check access restrictions via the UK Data Service.

ORCID iDs
Caitlin Lin http://orcid.org/0009-0004-0270-499X
Stephen Jivraj http://orcid.org/0000-0001-5751-983X

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