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# Age of Type 1 Diabetes Onset and Dementia Risk: A Swedish Nationwide, Register-Based Cohort Study

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## ABSTRACT

**Aims:** Individuals with Type 1 diabetes are at a higher risk of dementia compared to individuals without diabetes. Younger age at Type 1 diabetes onset has been linked to an increased risk of cardiovascular disease and higher mortality. This study aimed to determine whether younger age at Type 1 diabetes onset was also associated with all-cause dementia.

**Materials and Methods:** This nationwide prospective cohort study used data from individuals with Type 1 diabetes from the Swedish National Diabetes Register. All-cause dementia, including Alzheimer's disease, vascular dementia and non-Alzheimer's-non-vascular dementia, was prospectively ascertained as ICD-10 codes. Age at onset was stratified into < 10 years, 10–17 years and 18–30 years. The 18–30 years group was used as the reference group for all analyses. Cox regression modelling with age as the time-axis, from age at diabetes onset, was deployed to investigate dementia risk.

**Results:** Of 43 440 included individuals (mean age 33.0 (SD 14.0) years; 44% female), Type 1 diabetes developed before age 10 in 11 776 individuals (27%), between age 10 and 17 in 15 846 (36%) and between 18 and 30 (reference group) in 15 818 (36%). During a median 29.3 [17.6–43.7] years follow-up, 530 (1.2%) developed all-cause dementia. Compared to the reference group, all-cause dementia risk was increased in individuals aged < 10 years at onset (HR 1.37 [95% CI 1.08–1.73]), but not in individuals aged 10–17 years at onset (HR 1.04 [95% CI 0.85–1.26]) (fully adjusted model).

**Conclusions:** Type 1 diabetes onset before age 10 is associated with an increased risk of all-cause dementia compared to onset between ages 18 and 30.

**Twitter Summary**

Type 1 diabetes onset before age 10 is associated with an increased risk of all-cause dementia compared to onset between ages 18 and 30.

## 1 | Introduction

As survival improves amongst individuals with Type 1 diabetes, the prevalence of Type 1 diabetes amongst individuals aged ≥65 years increased by 180% between 1990 and 2019, rising from 1.3 to 3.7 million cases globally [1, 2]. With this trend, the burden of age-related comorbidities in these individuals, particularly dementia, will increase. Previous research has shown that individuals with Type 1 diabetes are at risk of cognitive decline and have a 1.5–2.0-fold increased risk of all-cause dementia compared to individuals without diabetes [3–8]. Several factors associated with this higher dementia risk have been identified, including recurrent diabetic ketoacidosis, [9] recurrent severe hypoglycaemia [10], single (versus married) status, low education and higher HbA1c levels [11, 12].

Younger age at Type 1 diabetes onset, particularly in the youngest age group (<10 years), has been linked to a higher risk of diabetes complications later in life, notably cardiovascular disease (CVD) and higher all-cause mortality [13]. However, whether this is also related to a higher dementia risk is unknown. Previous research indicated that childhood-onset Type 1 diabetes is associated with white matter hyperintensities, [14] greater loss of functional brain connectivity, [15] and a higher risk of cognitive impairment later in life [16]. Individuals who develop Type 1 diabetes at a younger age may exhibit a more aggressive disease phenotype, characterised by a strong autoimmune response, rapid beta-cell destruction, earlier initiation of insulin therapy and a shortened 'honeymoon phase' [17]. Moreover, a younger age at onset has been associated with a low-grade inflammation [18, 19] and increased inflammation in the pancreas [16]. Because younger brains are still developing, they may be particularly susceptible to harmful effects such as hyperglycaemia and inflammation [16, 20], which may affect neurodevelopment and accelerated brain ageing [16], increasing dementia risk later in life. This possibility was explored using nationwide, register-based data from the Swedish National Diabetes Register (NDR).

## 2 | Materials and Methods

### 2.1 | Data Source and Study Population

Data were used from the Swedish NDR, which contains prospectively collected information on diabetes status, a range of risk factors, treatments and complications amongst adults with diabetes mellitus since 1996 [21]. For the present pre-specified analysis, individuals with Type 1 diabetes were selected with at least one entry in the Swedish NDR between 1 January 1996 and 31 December 2019, with information on age at diabetes onset and no history of dementia recorded (Figure S1). This was in line with our previous research [12]. Type 1 diabetes was defined as treatment with insulin and an age at diagnosis ≤30 years. This definition has been validated against physician-assigned diabetes classification in the Swedish NDR and found to be accurate in 97% of individuals [22]. All participants provided written informed consent before enrolment in the NDR. The design of the study is shown in Figure S2. The analysis was done in a dynamic cohort, with follow-up starting at the age of Type 1 diabetes diagnosis. Participants therefore had different index dates. Most clinical characteristics were derived at cohort entry. Age at onset of diabetes and age at cohort entry were not necessarily the same, with age at diabetes onset often occurring at an earlier age (Figure S2). For example, an individual diagnosed with Type 1 diabetes at age 10 years and followed until age 60 years contributed risk time between ages 10 and 60 years.

### 2.2 | Baseline Characteristics

Baseline characteristics were derived from the first year following the first entry in the Swedish NDR. Social economic variables were derived from the Swedish Total Population register by Statistics Sweden. Income was stratified into annual quartiles. Education was defined as low (<9 years of education), intermediate (9–12 years of education) and high (≥12 years of education). Baseline information on history of CVD and history of diabetic ketoacidosis came from the Swedish National Patient Register [23]. History of CVD comprised coronary artery disease (CAD) (ICD-10 codes I20-I25), stroke (I60-I69), transient ischemic attack (TIA) (G45) and peripheral artery disease (PAD) (I70-I71, excluding I70.9, I71.0, I71.1 and I71.2). History of diabetic ketoacidosis included ICD-10 codes E.10.1, E.10.10 and E.10.11. Other characteristics were derived from the Swedish NDR. Albuminuria was defined as an albumin-creatinine ratio of ≥3 mg/mmol (~30 mg/g) or a urinary albumin clearance of ≥20 μg/min (~20 mg/L), in at least two out of three positive urine samples within 1 year. Retinopathy was defined as absent or present and included simplex, non-proliferative or proliferative retinopathy.

### 2.3 | Exposures and Outcomes

Individuals with Type 1 diabetes were categorised into three groups, according to age at diagnosis: <10 years, 10–17 years and 18–30 years. The youngest group was in accordance with previous research [13, 24], reflecting early-onset Type 1 diabetes; the oldest group represents an onset from adulthood until the cut-off for the epidemiological definition of Type 1 diabetes and we used an intermediate onset group. The primary outcome was all-cause dementia and included Alzheimer's disease (ICD-10 codes F00, G30), vascular dementia (F01) and other dementia (F02, F03), derived from the Swedish National Patient Register. Exploratory outcomes included the dementia subtypes described above. ICD-10 codes may have limited ability to reliably distinguish between dementia subtypes because, in clinical practise, these subtypes often co-exist. Therefore, we considered these outcomes exploratory. The Swedish National Patient Register contains data on hospital admissions and specialist outpatient care since 1987 [23]. Inpatient data was fully covered since 1987. Outpatient data had become complete during the present millennium.

Previous research has validated all-cause dementia diagnosis as accurate in 81%–96% of cases, whilst this was lower for dementia subtypes [25, 26]. Dementia diagnoses derived from primary care were not included in this analysis because these data were not available. For individuals with multiple recorded dementia subtypes, the first entry was used in the analyses. Follow-up data on dementia outcomes was available from 1997 to 31 December 2020. All-cause mortality was ascertained from the Swedish Cause of Death Register [27], but dementia-related deaths were not recorded.

### 2.4 | Statistical Analysis

Descriptive statistics were presented as means and SD for continuous variables and as counts and percentage for categorical variables. First, incidence rates for the primary and secondary outcomes were estimated as the number of events per 1000 person-years with 95% exact Poisson confidence intervals (CI). Second, incidence rates were adjusted for age and sex at cohort entry and using the total population as the reference. In addition, cumulative incidence of all-cause dementia was estimated from Kaplan–Meier survival functions at ages 65, 70 and 75 years by age at Type 1 diabetes onset group. Next, Cox regression was used to estimate dementia risk according to categories of age at onset (i.e., < 10 years, 10–17 years and 18–30 years) with the 18–30 year age at onset group as the reference. The first model was crude, the second model was adjusted for sex and the third model (main model) was adjusted for sex, region of birth, education and marital status at cohort entry. Chronological age was used as the time axis to account for the increasing baseline hazard associated with age (Figure S2). A delayed-entry (left-truncation) approach was used, with each participant entering the risk set at their age at Type 1 diabetes diagnosis. Follow-up was until dementia, death or censoring (31 December 2020), whichever came first. The analyses estimated age-specific dementia risks conditional on having survived and remained event-free up to a given age.

Additional analyses were performed. First, we explored dementia risk additionally stratified by sex, as dementia incidence rates might differ by sex in the general population [28]. Second, we adjusted for diabetes duration at cohort entry. Third, to account for complication status and temporal treatment effects, we adjusted for sex, education, marital status, region of birth, history of CVD, history of stroke or TIA, history of diabetic ketoacidosis and calendar year of cohort inclusion. Fourth, the main analysis was repeated with Type 1 diabetes being defined as either ≤ 30 years diabetes onset age and insulin treatment or as determined through clinical assessment by the treating physician (irrespective of age) [29]. Fifth, the main analysis was repeated with follow-up time from cohort entry instead of age at onset.

Schoenfeld residual plots were visually inspected and met the proportional hazard assumption. Multiple imputation using the multivariate imputation by chained equations (MICE) package was deployed for missing baseline data using 60 datasets to ensure convergence, with final results pooled according to Rubin's rules. Variables used for the imputation procedure are provided in Table S1. All analyses were performed using R software, version 4.4.2 (R Foundation for Statistical Computing, Vienna, Austria), using RStudio (Gothenburg, Sweden).

## 3 | Results

Forty-three thousand four hundred forty individuals with Type 1 diabetes were included (Figure S1). At cohort entry, mean age was 33.0 (SD 14.0) years (median age was 28.0 [21.0–42.0] years) and 19 114 (44%) were female. Eleven thousand seven hundred seventy-six individuals (27%) had an age at onset < 10 years, 15 846 (36%) of 10–17 years and 15 818 (36%) of 18–30 years. In the < 10 years age at onset group, median follow-up was longest with 34.2 [22.7–48.4] years, the mean age at cohort entry was 29.6 (SD 12.6) years (median age was 23.0 [20.0–36.0] years) and 49% were female. In the 10–17 years of age at onset group the median follow-up was 28.6 [16.1–43.7] years, mean age at cohort entry was 31.1 (SD 13.8) years (median age was 24.0 [20.0–39.0] years) and 44% were female and in the 18–30 years of age at onset the median follow-up was 26.8 [13.9–40.2] years, the mean age at cohort entry was 37.5 (SD 14.2) years (median age was 33.0 [26.0–47.0] years) and 41% were female. Other characteristics at cohort entry are presented in Table 1. Baseline characteristics for individuals before multiple imputation are presented in Table S2. The percentage of missing data for variables age, sex and region of birth was 0%, for education 7.6% and for marital status 7.1% (Table S2).

Over an overall median follow-up time of 29.3 [17.6–43.7] years, 100 (0.9%), 158 (1.0%) and 272 (1.7%) all-cause dementia events were recorded in the < 10 years, 10–17 years and 18–30 years age at onset groups, respectively. Median age of dementia detection was 66.3 years, 70.9 and 72.8 years in the < 10, 10–17 and 18–30 years age at onset group. Age- and sex-adjusted incidence rates were in the < 10 years age at onset group 0.21 [95% CI 0.09–0.39] per 1000 person-years, in the 10–17 years age at onset group 0.22 [95% CI 0.10–0.40] per 1000 person-years and 0.28 [95% CI 0.14–0.48] in the 18–30 years age at onset group per 1000 person-years (Table S3). Figure 1 presents the dementia-free survival curve. Cumulative incidence of all-cause dementia at age 75 ranged from 4.85% in those with onset at 18–30 years to 6.75% in those with onset before age 10 years, with a consistent gradient across onset groups (Table S4). Based on the crude model, dementia risk was significantly higher in the < 10 years age at onset group (HR 1.37 [95% CI 1.08–1.72]) compared to 18–30 years age at onset. In model 3 (fully adjusted), individuals aged < 10 years at diabetes onset had a 37% higher risk (HR 1.37 [95% CI 1.08–1.73]) for all-cause dementia compared to individuals aged 18–30 years at diabetes onset (Figure 2). The hazard ratio for dementia was higher in the youngest age-at-onset group despite the lower crude incidence rate because dementia was diagnosed at a younger age in these individuals. The risk for individuals aged 10–17 years at diabetes onset did not differ from those aged 18–30 years at diabetes onset (HR 1.04 [95% CI 0.85–1.26]).

Figure 3 presents the risk of dementia subtypes. In total, 23 (0.2%), 27 (0.2%) and 64 (0.4%) Alzheimer's disease events, 36 (0.4%), 48 (0.3%) and 79 (0.5%) vascular dementia events and 41 (0.3%), 83 (0.5%) and 129 (0.8%) other dementia events were

**TABLE 1** | Baseline characteristics.

| Characteristics | Overall | Age at Type 1 diabetes onset | | |
|---|---|---|---|---|
| | | < 10 years | 10–17 years | 18–30 years |
| *N* | 43 440 | 11 776 | 15 846 | 15 818 |
| Sex (female), *n* (%) | 19 240 (44) | 5745 (49) | 7052 (44) | 6443 (41) |
| Age (years) | 33.0 (14) | 29.6 (13) | 31.1 (14) | 37.5 (14) |
| Marital status, *n* (%) | | | | |
| Married | 11 223 (26) | 2180 (18) | 3465 (22) | 5578 (35) |
| Separated or widowed | 2840 (6.5) | 562 (4.8) | 927 (5.9) | 1351 (8.5) |
| Single | 29 377 (68) | 9034 (77) | 11 454 (72) | 8889 (56) |
| Education, *n* (%) | | | | |
| < 9 years | 7919 (18) | 1997 (17) | 2774 (18) | 3148 (20) |
| 9–12 years | 24 628 (57) | 7128 (60) | 9406 (59) | 8094 (51) |
| > 12 years | 10 893 (25) | 2651 (22) | 3666 (23) | 4576 (29) |
| Income quartiles, *n* (%) | | | | |
| First quartile | 17 366 (40) | 5378 (46) | 6877 (43) | 5111 (32) |
| Second quartile | 11 645 (27) | 3173 (27) | 4120 (26) | 4352 (28) |
| Third quartile | 8274 (19) | 1972 (17) | 2792 (18) | 3510 (22) |
| Fourth quartile | 6155 (14) | 1253 (11) | 2057 (13) | 2845 (18) |
| Region of birth, *n* (%) | | | | |
| Sweden | 39 945 (92) | 11 196 (95) | 14 786 (93) | 13 963 (88) |
| Europe other than Sweden | 1903 (4.4) | 376 (3.2) | 628 (4.0) | 899 (5.7) |
| Rest of the world | 1592 (3.7) | 204 (1.7) | 432 (2.7) | 956 (6.0) |
| Diabetes duration (years) | 18.1 (14) | 23.9 (13) | 17.8 (14) | 14.2 (14) |
| History of diabetic ketoacidosis, *n* (%) | 4495 (10) | 1567 (13) | 1887 (12) | 1041 (6.6) |
| History of severe hypoglycaemia, *n* (%) | 838 (1.9) | 335 (2.8) | 312 (2.0) | 191 (1.2) |
| Diabetes treatment | | | | |
| Insulin pump use, *n* (%) | 7804 (18) | 3735 (32) | 3169 (20) | 900 (5.7) |
| Multiple daily insulin injections, *n* (%) | 35 636 (82) | 8041 (68) | 12 677 (80) | 14 918 (94) |
| Use of oral blood glucose lowering medication, *n* (%) | 189 (0.4) | 13 (0.1) | 23 (0.1) | 153 (1.0) |
| History of cardiovascular disease, *n* (%) | 1741 (4.0) | 432 (3.7) | 566 (3.6) | 743 (4.7) |
| History of stroke/TIA, *n* (%) | 524 (1.2) | 134 (1.1) | 170 (1.1) | 220 (1.4) |
| History of retinopathy, *n* (%) | 19 025 (44) | 6429 (55) | 6727 (42) | 5869 (37) |
| eGFR (mL/min/1.73 $m^2$) | 101 (29) | 101 (29) | 102 (30) | 98 (28) |
| Albuminuria, *n* (%) | 8000 (19) | 2456 (21) | 2831 (18) | 2713 (17) |
| Current smoking, *n* (%) | 6283 (14) | 1628 (14) | 2172 (14) | 2483 (16) |
| Body mass index (kg/$m^2$) | 24.8 (4.1) | 24.7 (3.7) | 24.7 (3.9) | 25.1 (4.4) |
| Systolic blood pressure (mmHg) | 125 (16) | 124 (16) | 124 (16) | 126 (17) |
| Diastolic blood pressure (mmHg) | 73 (9.1) | 73 (9) | 73 (9) | 74 (9) |

(Continues)

**TABLE 1** | (Continued)

| Characteristics | Overall | Age at Type 1 diabetes onset | | |
|---|---|---|---|---|
| | | <10 years | 10–17 years | 18–30 years |
| HbA1c (mmol/mol) | 65 (16) | 69 (16) | 66 (16) | 62 (17) |
| LDL cholesterol (mmol/L) | 2.6 (0.8) | 2.6 (0.8) | 2.6 (0.8) | 2.6 (0.8) |
| Use of blood pressure-lowering medication, *n* (%) | 7402 (17) | 2108 (18) | 2559 (16) | 2735 (17) |
| Use of lipid lowering-medication, *n* (%) | 6369 (15) | 1486 (13) | 2057 (13) | 2826 (18) |
| Follow-up (years) | 29.3 [17.6–43.7] | 34.2 [22.7–48.4] | 28.6 [16.1–43.7] | 26.8 [13.9–40.2] |

*Note:* Continuous data presented as mean (SD) or median (interquartile range), and categorical data presented as *n* (%).
Abbreviations: eGFR: estimated glomerular filtration rate. LDL cholesterol: low-density-lipoprotein cholesterol. Severe hypoglycaemia: Hypoglycaemic event with an insult or requiring others to recover in the past 12 months. TIA: Transient ischemic attack.

recorded in the <10, 10–17 and 18–30 years age at diabetes onset groups, respectively (Figure 3 and Table S3). Compared to individuals aged 18–30 years at diabetes onset, individuals aged <10 years at diabetes onset and those aged 10–17 years did not have a different risk of Alzheimer's disease (HR 1.28 [95% CI 0.78–2.09] and HR 0.74 [95% CI 0.47–1.17], respectively). Conversely, individuals aged <10 years at diabetes onset had a significantly higher risk of vascular dementia than those 18–30 years at diabetes onset (HR 1.65 [95% CI 1.10–2.47]). No other significant differences were identified (Figure 3).

The relationship between age at onset and dementia did not differ by sex with *p*-values for interaction >0.30 (Figure S3). When additionally adjusted for diabetes duration, the risk for all-cause dementia was higher in the <10 years at onset group (HR 2.14 [95% CI 1.60–2.87]) and in the 10–17 years age at onset group (HR 1.33 [95% CI 1.07–1.66]) compared to the 18–30 years at onset group (Figure S4). Results remained consistent with the main analysis when additionally adjusted for history of CVD, history of stroke/TIA, history of DKA and calendar year at cohort entry (Figure S4). When using a broader Type 1 diabetes definition (i.e., age at onset ≤30 years and insulin use, or according to the assessment of the clinician (irrespective of age)), dementia risk in the youngest age at onset group (<10 years) was highest compared to the oldest age at onset group (>30 years) as the reference group (Tables S5 and S6 and Figure S5). The results of the analyses with follow-up starting from cohort entry were consistent with the main analyses (Figure S6). Table S7 shows the age-specific incidence rates of all-cause dementia across different age bands (>50 to >75 years) stratified by age at diabetes onset, with highest incidence rates for the youngest age at onset group (Table S7).

## 4 | Discussion

In this large, nationwide cohort study of over 43 000 individuals with Type 1 diabetes, a younger age at diabetes onset (<10 years) was associated with a higher risk of all-cause dementia compared to those with onset between 18 and 30 years. Specifically, individuals diagnosed with Type 1 diabetes before the age of 10 years had a 37% higher dementia risk compared with those diagnosed between 18 and 30 years, after adjustment for sociodemographic factors.

Previous studies have consistently shown that individuals with Type 1 diabetes have a higher dementia risk compared with individuals without diabetes [3–7], but these studies did not consider heterogeneity by age at onset. The present study findings are in agreement with previous data showing that younger age at onset of Type 1 diabetes is also associated with higher risks of CVD, including heart failure hospitalizations, chronic kidney disease, as well as increased mortality [13, 30]. Our findings extend this evidence to cognitive outcomes, suggesting that individuals who develop Type 1 diabetes early in life may also experience an increased risk of dementia, particularly vascular dementia, later in life.

The explanations for our findings are likely to be multifaceted. Early-onset Type 1 diabetes is related to a disease phenotype with a more severe and rapid loss of β-cells, which contributes to increased glycemia [16, 17] and a heightened systemic inflammation response [18, 19], all of which may promote (micro)vascular damage and neurodegeneration, key mechanisms in cognitive impairment [20]. Given that the developing brain may be particularly susceptible to metabolic and inflammatory insults [16, 20], early-onset Type 1 diabetes may predispose individuals to either impairment of neurodevelopmental processes, possibly already impacting cerebral reserve or accelerated brain ageing processes and a higher risk of dementia in late life. Furthermore, diabetes duration is likely to have a role. Individuals with a younger age at onset inherently have a longer lifetime exposure to diabetes. Prolonged diabetes duration implies greater cumulative exposure of the brain to hyperglycaemia and related metabolic insults. The third potential is that the greater incidence of CVD and chronic kidney disease in younger onset Type 1 diabetes [13, 30] may accelerate cognitive decline [31, 32], particularly vascular dementia. Although adjustment for diabetes duration did not change the direction of effect, residual confounding by duration cannot be excluded. Cardiovascular events preceding vascular dementia diagnosis may additionally mediate this association, and the extent to which this explains the subtype-specific findings warrants further investigation.

Strengths of this study include its large sample size, nationwide coverage and long follow-up, allowing for robust estimation of dementia incidence across onset age groups. The linkage of multiple Swedish national registers enabled comprehensive adjustment for sociodemographic factors, and the use of validated

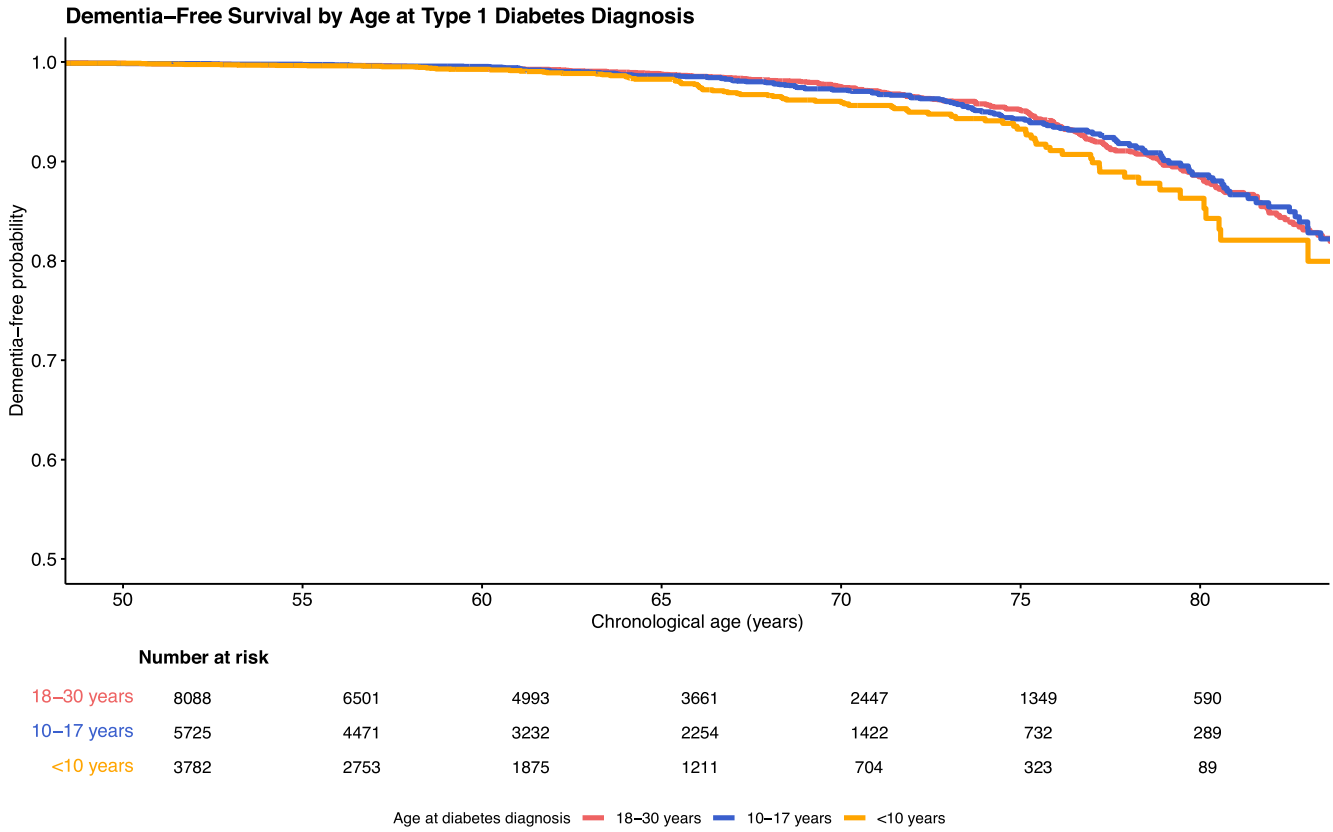


**FIGURE 1** | Incidence of all-cause dementia in individuals with Type 1 diabetes stratified by age at diabetes onset. Yellow line indicated < 10 years, blue line 10–17 years and red line 18–30 years age at diabetes onset, respectively. Age is used as the time axis.

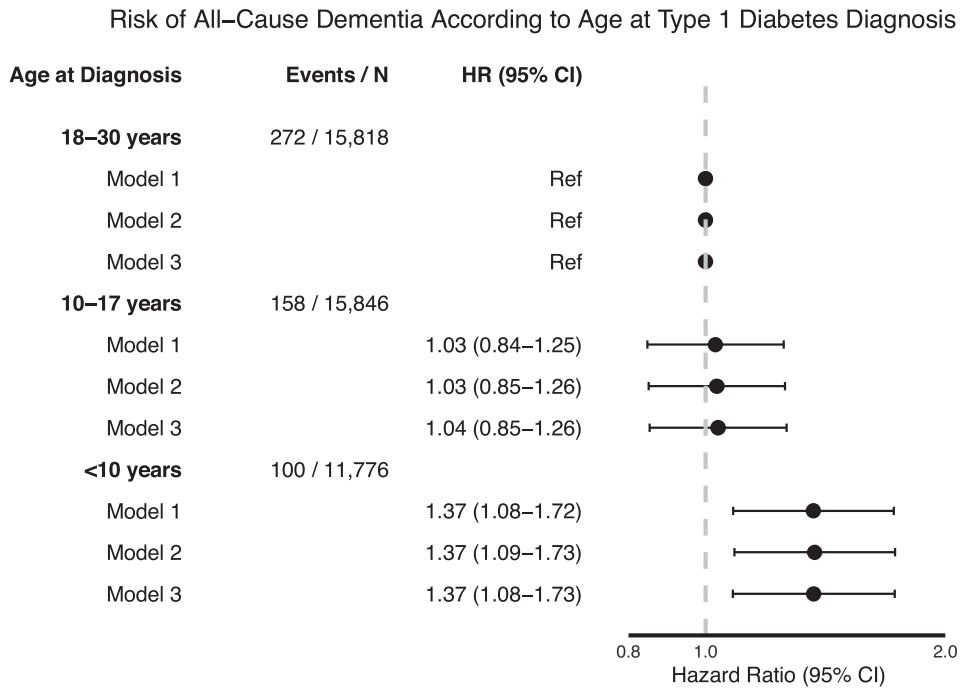


**FIGURE 2** | Risk of all-cause dementia in individuals with Type 1 diabetes stratified by age at diabetes onset. Model 1: Crude (unadjusted). Model 2: Adjusted for sex. Model 3: Adjusted for sex, region of birth, education and marital status at cohort entry. All Cox models have chronological age as the time axis. The group with 18–30 years at Type 1 diabetes onset was the reference category.

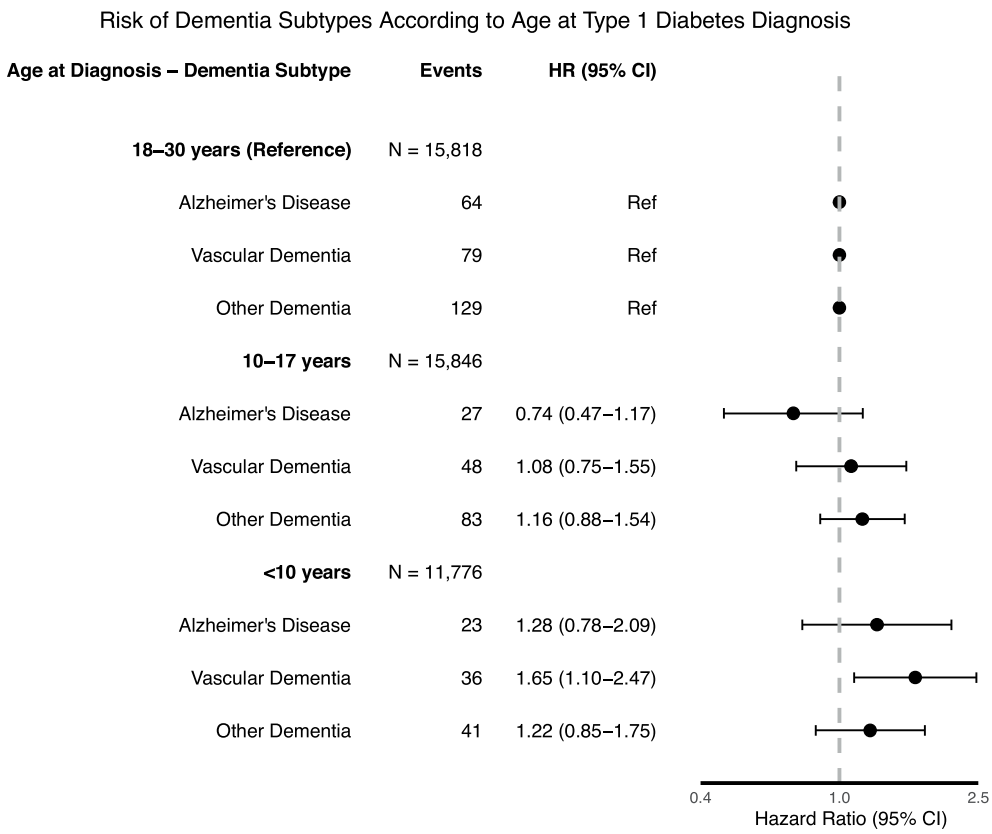


**FIGURE 3** | Risk of dementia subtypes, including Alzheimer's disease, vascular dementia and non-Alzheimer's-non-vascular dementia in individuals with Type 1 diabetes stratified by age at diabetes onset. All models adjusted for sex, region of birth, education and marital status at cohort entry. All Cox models have chronological age as the time axis. The group with 18–30 years at Type 1 diabetes onset was the reference category.

algorithms for both Type 1 diabetes and dementia ensured a high level of diagnostic accuracy. Nevertheless, several limitations merit consideration. First, despite adjustments, residual confounding cannot be ruled out. The study lacked detailed data on lifestyle factors (e.g., smoking, physical activity, diet) and glycemic variability, which influence dementia risk. Second, ascertainment of dementia relied on hospital and specialist outpatient records; thus, cases diagnosed in primary care may have been missed, leading to underestimation of incidence rates. Third, ICD-10 codes might offer limited capability to reliably differentiate between dementia subtypes, because, in clinical practise, subtypes often coexist. Therefore, these analyses should be considered exploratory. Also, there were no data on biomarkers such as β-amyloid and tau protein levels to support an Alzheimer disease diagnosis. Fourth, individuals with earlier-onset diabetes are less likely to have reached the typical dementia age range at the end of follow-up, potentially leading to conservative estimates due to limited event accumulation. Fifth, clinical characteristics were derived at cohort entry, a median of 14–24 years after diabetes onset (Figure S2) and may therefore not accurately reflect status at diabetes onset. Sixth, although validation of Type 1 diabetes diagnosis was high (97%), it cannot be excluded that some individuals included had another diabetes type, notably (early-onset) Type 2 diabetes. Seventh, we cannot exclude that individuals with younger age at onset received lower quality of care in earlier treatment eras, which may partially explain the observed associations. Last, the study population consisted predominantly of individuals born in Sweden (>90%), potentially limiting generalizability of our findings to other geographic regions and ethnic backgrounds.

These findings highlight the need for a life-course approach to brain health in Type 1 diabetes. As survival improves and more individuals live into older ages with long-standing disease, dementia prevention and early cognitive monitoring may become increasingly important components of care. Further research is needed to clarify whether incorporating brief validated cognitive screening instruments into routine diabetes follow-up is a feasible approach to identify cognitive decline at an early stage. Targeted preventive strategies or more aggressive targeting of other vascular risk factors for example, smoking, LDL-cholesterol, reported to be linked to dementia risks, are warranted [12, 28]. Future studies integrating neuroimaging and biomarker assessments could help disentangle the relative contributions of vascular, metabolic and neurodegenerative pathways in this population.

In conclusion, a younger age at Type 1 diabetes onset (<10 years) was associated with a higher risk of all-cause dementia compared with later onset (18–30 years).

## Author Contributions

M.J., F.L.J.V., J.H.D.V., B.E. and Thomas T. van Sloten designed the study. M.J. and Thomas T. van Sloten conducted the statistical analyses. All authors contributed to data interpretation. M.J. and Thomas T. van Sloten wrote the initial manuscript draught. All authors provided

critical feedback and approved submission. M.J. and Thomas T. van Sloten are the guarantors of this work and, as such, had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.


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### Conflicts of Interest

B.E. reports honoraria for speaking from AstraZeneca, Boehringer Ingelheim, Eli Lilly, MSD, Novo Nordisk and Sanofi, and reports consulting fees from Amgen, AstraZeneca, Boehringer Ingelheim, Eli Lilly, MSD, Novo Nordisk and Sanofi. H.C.G. holds the McMaster-Sanofi Population Health Institute Chair in Diabetes Research and Care. He reports research grants from Novo Nordisk; continuing education grants from Eli Lilly, Abbott, Sanofi, Novo Nordisk and Boehringer Ingelheim; honoraria for speaking from Jiangsu Hanson; and consulting fees from Abbott, Eli Lilly, Novo Nordisk and Zealand, and holds a patent for the use of GDF15 as a biomarker for metformin intake. N.S. reports research grants from AstraZeneca, Boehringer Ingelheim, Novartis and Roche Diagnostics, honoraria for speaking from Abbott, AbbVie, AstraZeneca, Boehringer Ingelheim, Eli Lilly, Janssen, Novo Nordisk and Sanofi and reports consulting fees from Abbott, AbbVie, Amgen, AstraZeneca, Boehringer Ingelheim, Eli Lilly, Hanmi, Janssen, Menarini-Ricerche, Novartis, Novo Nordisk, Pfizer, Roche and Sanofi. Tali Cukierman-Yaffe reports speaking honoraria from Sanofi, Novo Nordisk, AstraZeneca, Boehringer Ingelheim, Geffen medical, Medtronic, consulting fees from Amgen, Gefen medical and research grants from Medtronic, Regeneron, Sanofi and Novo Nordisk. The other authors declare no conflicts of interest.

### Data Availability Statement

Data are located in the Swedish National Diabetes Register, Swedish Total Population Register and the Swedish National Patient Register. Researchers can receive access to the registries analysed in this article after ethical approval by the Swedish Review Authority.

### Peer Review

The peer review history for this article is available at https://www.webofscience.com/api/gateway/wos/peer-review/10.1111/dom.71123.

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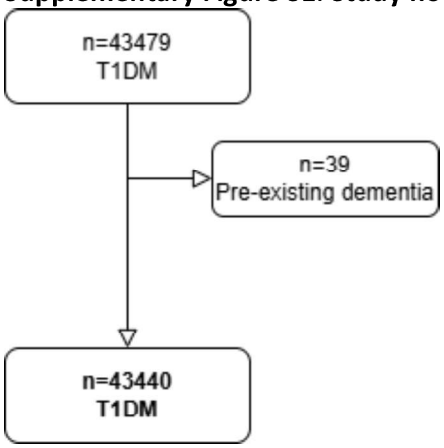
## Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Figure S1:** Study flowchart. **Figure S2:** Study design. **Table S1:** Variables used in the imputation algorithm. **Table S2:** Baseline characteristics and percentage of missing values of individuals with Type 1 diabetes before imputation. **Table S3:** Incidence of all-cause dementia and dementia subtypes in individuals with Type 1 diabetes stratified on age at diabetes onset. **Table S4:** Cumulative incidence estimates of all-cause dementia at ages 65, 70 and 75 years age stratified by age at Type 1 diabetes onset. **Figure S3:** All-cause dementia risk in Type 1 diabetes according to age at diabetes onset stratified on sex. **Figure S4:** Incident all-cause dementia risk in individuals with Type 1 diabetes using additionally adjusted models. **Table S5:** Baseline characteristics of individuals with Type 1 diabetes defined as insulin use and age at onset ≤ 30 years or based on clinical judgement (irrespective of age). **Table S6:** Incidence of all-cause dementia and dementia subtypes in individuals with Type 1 diabetes defined as insulin use and age at onset ≤ 30 years, or based on clinical judgement (irrespective of age). **Figure S5:** Risk of all-cause dementia in individuals with Type 1 diabetes defined as insulin use and age at onset ≤ 30 years, or based on clinical judgement (irrespective of age). **Figure S6:** Risk of all-cause dementia in individuals with Type 1 diabetes starting follow-up from cohort entry instead of follow-up from age at diabetes onset. **Table S7:** Age-specific incidence rates of all-cause dementia in age bands, stratified by age at Type 1 diabetes onset.

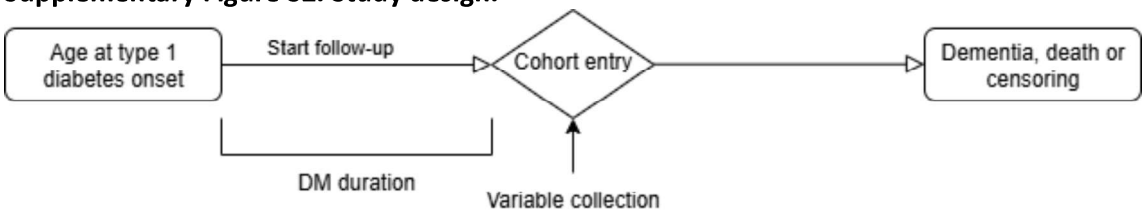
**Supplementary Appendix**

**Supplementary Figure S1. Study flowchart.**




All individuals with a diagnosis of type 1 diabetes (i.e., treatment with insulin and an age at diagnosis ≤30 years) were included from the Swedish National Diabetes Register. All individuals had information on age at diabetes onset. Individuals with a history of dementia before type 1 diabetes diagnosis were excluded. T1DM: Type 1 diabetes.

**Supplementary Figure S2. Study design.**




Variables were collected at cohort entry. Most individuals had a type 1 diabetes diagnosis before cohort entry. N=35 had an age at onset older than at cohort entry, but did not have any dementia events.

**Supplementary Table S1. Variables used in the imputation algorithm.**

| Variables used in the imputation algorithm |
| --- |
| Age, sex, history of cardiovascular disease, history of stroke, history of transient ischemic attack, history of cardiovascular disease, history of peripheral artery disease, education, marital status, income, Nelson-Aalen estimator* including time to outcome for all-cause dementia, diabetes duration, HbA1c, systolic blood pressure, diastolic blood pressure, current smoking, body mass index, low-density-lipoprotein cholesterol, estimated glomerular filtration rate, albuminuria, retinopathy, insulin pump use, use of blood pressure lowering medication, use of acetylsalicylic acid, and use of lipid lowering medication. |

*Nelson-Aalen estimator is a nonparametric estimator of the cumulative hazard function.

**Supplementary Table S2. Baseline characteristics and percentage of missing values of individuals with type 1 diabetes before imputation.**

| Characteristics | Type 1 diabetes | Missing (%) |
|---|---|---|
| N | 43,440 | |
| Sex (female), n (%) | 19,240 (44) | 0 |
| Age (years) | 33.0 (14) | 0 |
| Marital status, n (%) | | 7.1 |
| Married | 10,716 (25) | |
| Separated or widowed | 2,691 (6.2) | |
| Single | 26,932 (62) | |
| Education, n (%) | | 7.6 |
| <9 years | 7,306 (17) | |
| 9-12 years | 22,794 (52) | |
| >12 years | 10,101 (23) | |
| Income quartiles, n (%) | | 29 |
| First quartile | 9,032 (21) | |
| Second quartile | 8,742 (20) | |
| Third quartile | 7,227 (17) | |
| Fourth quartile | 5,709 (13) | |
| Region of birth, n (%) | | 0 |
| Sweden | 39,945 (92) | |
| Europe other than Sweden | 1,903 (4.4) | |
| Rest of the world | 1,592 (3.7) | |
| Diabetes duration (years) | 18.1 (14) | 0.1 |
| History of diabetic ketoacidosis, n (%) | 4,495 (10) | 0 |
| History of severe hypoglycemia, n (%) | 838 (1.9) | 0 |
| Diabetes treatment | | |
| Insulin pump use, n (%) | 4,898 (11) | 45 |
| Multiple daily insulin injections, n (%) | 18,803 (43) | 45 |
| Use of oral blood glucose lowering medication, n (%) | 196 (0.5) | 0 |
| History of cardiovascular disease, n (%) | 1,741 (4.0) | 0 |
| History of stroke/TIA, n (%) | 524 (1.2) | 0 |
| History of retinopathy, n (%) | 6,420 (14) | 0 |
| eGFR (mL/min/1.73m$^2$) | 105 (29) | 47 |
| Albuminuria, n (%) | 5,634 (13) | 26 |
| Current smoking, n (%) | 5,921 (14) | 5.0 |
| Body mass index (kg/m$^2$) | 24.9 (4.1) | 11 |
| Systolic blood pressure (mmHg) | 125 (16) | 3.9 |
| Diastolic blood pressure (mmHg) | 73 (9) | 4.2 |
| HbA1c (mmol/mol) | 65 (16) | 2.1 |
| LDL cholesterol (mmol/L) | 2.6 (0.8) | 53 |
| Use of blood pressure-lowering medication, n (%) | 7,219 (17) | 3.9 |
| Use of lipid lowering-medication, n (%) | 4,933 (11) | 17 |

Continuous data presented as mean (SD) and categorical data presented as n (%). CVD: Cardiovascular disease. TIA: Transient ischemic attack. Severe hypoglycemia: Hypoglycemic event with an insult or requiring others to recover in the past 12 months. eGFR: estimated glomerular filtration rate. LDL cholesterol: low-density-lipoprotein cholesterol.

**Supplementary Table S3. Incidence of all-cause dementia and dementia subtypes in individuals with type 1 diabetes stratified on age at diabetes onset.**

| Age at onset | Events (n/Ntot) | Incidence rate per 1,000 person years (95% CI) | Age- and sex-standardized incidence rate per 1000 person years (95% CI) |
|---|---|---|---|
| *All-cause dementia* | | | |
| <10 years | 100/11,776 | 0.23 (0.19-0.28) | 0.21 (0.09-0.39) |
| 10-17 years | 158/15,846 | 0.32 (0.28-0.38) | 0.22 (0.10-0.40) |
| 18-30 years | 272/15,818 | 0.62 (0.55-0.70) | 0.28 (0.14-0.48) |
| *Alzheimer's disease* | | | |
| <10 years | 23/11,776 | 0.05 (0.03-0.08) | 0.05 (0.01-0.17) |
| 10-17 years | 27/15,846 | 0.06 (0.04-0.08) | 0.03 (0.0-0.15) |
| 18-30 years | 64/15,818 | 0.15 (0.11-0.19) | 0.07 (0.01-0.20) |
| *Vascular dementia* | | | |
| <10 years | 36/11,776 | 0.08 (0.06-0.11) | 0.07 (0.02-0.21) |
| 10-17 years | 48/15,846 | 0.10 (0.07-0.13) | 0.06 (0.01-0.19) |
| 18-30 years | 79/15,818 | 0.18 (0.14-0.22) | 0.08 (0.02-0.23) |
| *Other dementia* | | | |
| <10 years | 41/11,776 | 0.10 (0.07-0.13) | 0.09 (0.02-0.23) |
| 10-17 years | 83/15,846 | 0.17 (0.14-0.21) | 0.12 (0.04-0.27) |
| 18-30 years | 129/15,818 | 0.29 (0.25-0.35) | 0.13 (0.04-0.29) |

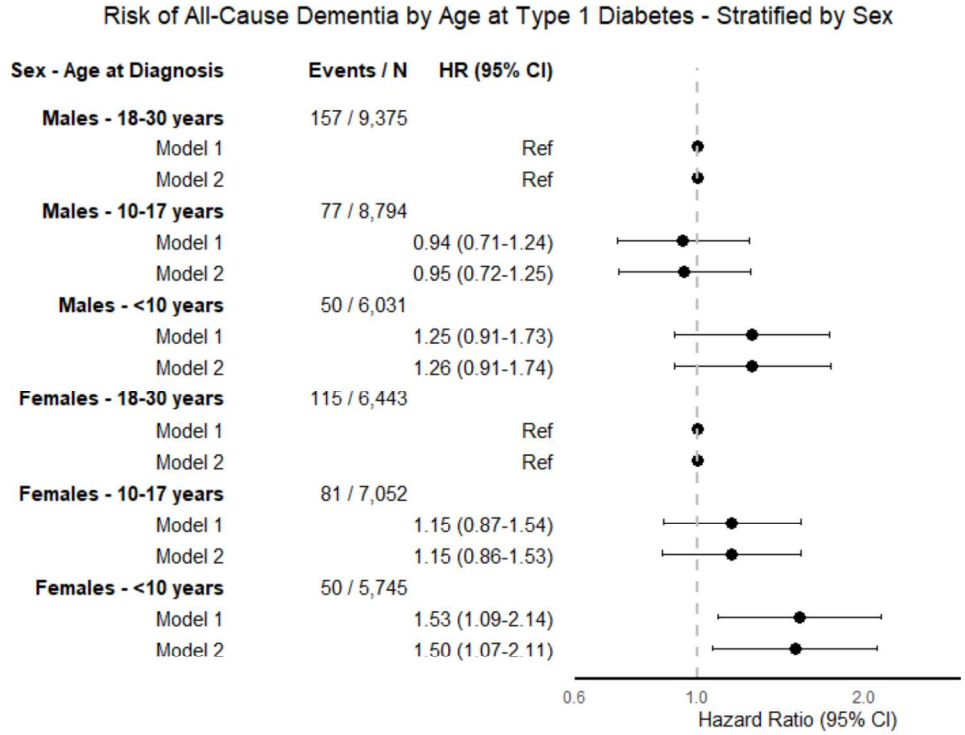
Incidence rates standardized for age and sex. Total follow-up included 1,354,556 person years. CI: confidence interval.

**Supplementary Table S4. Cumulative incidence estimates of all-cause dementia at ages 65, 70 and 75 years age stratified by age at type 1 diabetes onset.**

| Age | Number at risk | Dementia events | Cumulative incidence (95%CI) |
|---|---|---|---|
| *65 years* | | | |
| <10 years | 1,211 | 38 | 1.74% (1.14–2.33) |
| 10-17 years | 2,254 | 50 | 1.38% (0.98–1.79) |
| 18-30 years | 3,661 | 68 | 1.21% (0.91–1.51) |
| *70 years* | | | |
| <10 years | 704 | 23 | 3.94% (2.86–5.00) |
| 10-17 years | 1,422 | 26 | 2.82% (2.14–3.50) |
| 18-30 years | 2,447 | 38 | 2.48% (1.98–2.98) |
| *75 years* | | | |
| <10 years | 323 | 14 | 6.75% (4.91–8.55) |
| 10-17 years | 732 | 30 | 5.72% (4.48–6.94) |
| 18-30 years | 1,349 | 46 | 4.85% (4.00–5.69) |

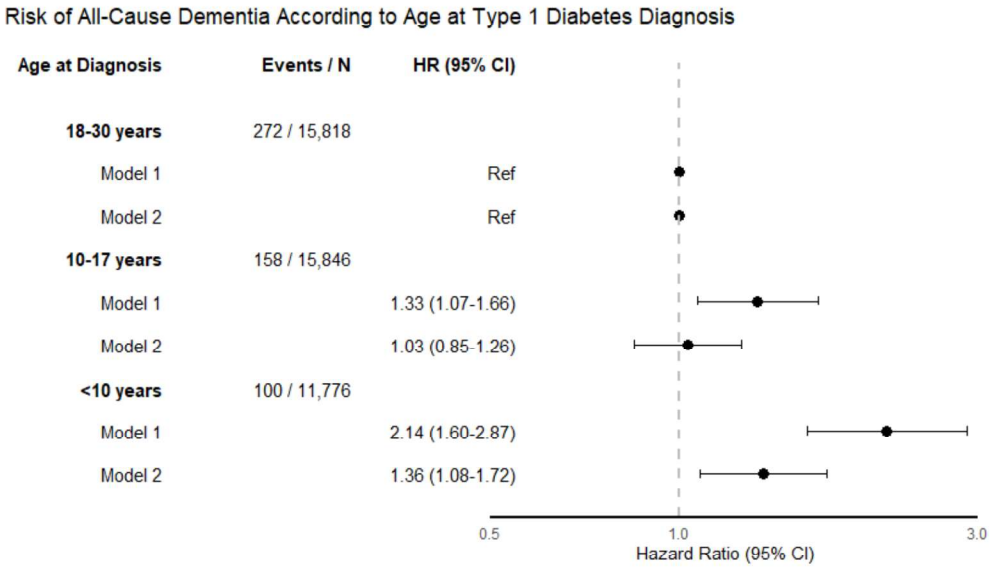
Cumulative incidence was estimated from Kaplan-Meier survival functions and expressed as percentage with 95% confidence intervals. Numbers at risk and incident dementia cases at each age are shown stratified by age at type 1 diabetes onset group. CI=Confidence interval.

**Supplementary Figure S3. All-cause dementia risk in type 1 diabetes according to age at diabetes onset stratified on sex.**




Model 1: crude. Model 2: adjusted for education, marital status and region of birth. All Cox models have age as the time axis. 18-30 years at diabetes onset group was the reference category.

**Supplementary Figure S4. Incident all-cause dementia risk in individuals with type 1 diabetes using additionally adjusted models.**




Model 1: adjusted for sex, education, marital status, region of birth, and diabetes duration at cohort entry. Model 2: adjusted for sex, education, marital status, region of birth, year of cohort entry, history of cardiovascular disease, history of stroke/TIA and history of ketoacidosis at cohort entry. All Cox models have age as the time axis. 18-30 years at diabetes onset group was the reference category.

**Supplementary Table S5. Baseline characteristics of individuals with type 1 diabetes defined as insulin use and age at onset ≤30 years, or based on clinical judgement (irrespective of age).**

| | Age at onset <10 years | Age at onset 10-17 years | Age at onset 18-30 years | Age at onset ≥30 years |
|---|---|---|---|---|
| n | 11,931 | 15,998 | 16,871 | 9,923 |
| Sex = female (%) | 5,816 (49) | 7,128 (45) | 6,854 (41) | 4,238 (43) |
| Age (years) | 29.5 (13) | 31.0 (14) | 37.6 (14) | 50.2 (11) |
| *Marital status, n (%)* | | | | |
| *Married* | 2,193 (18) | 3,488 (22) | 6,018 (36) | 5,604 (56) |
| *Separated or widowed* | 568 (4.8) | 929 (5.8) | 1,435 (8.5) | 1,490 (15) |
| *Single* | 9,170 (77) | 11,581 (72) | 9,418 (56) | 2,829 (28) |
| Education, n (%) | | | | |
| *<9 years* | 2,020 (17) | 2,810 (18) | 3,322 (20) | 2,040 (21) |
| *9-12 years* | 7,230 (61) | 9,498 (59) | 8,620 (51) | 4,701 (47) |
| *>12 years* | 2,681 (22) | 3,690 (23) | 4,929 (29) | 3,182 (32) |
| Income quartiles, n (%) | | | | |
| *First quartile* | 5,457 (46) | 6,947 (43) | 5,397 (32) | 1,450 (15) |
| *Second quartile* | 3,216 (27) | 4,162 (26) | 4,631 (27) | 2,530 (26) |
| *Third quartile* | 1,993 (17) | 2,821 (18) | 3,767 (22) | 2,825 (28) |
| *Fourth quartile* | 1,265 (11) | 2,068 (13) | 3,076 (18) | 3,118 (31) |
| Region of birth, n (%) | | | | |
| Sweden | 11,344 (95) | 14,931 (93) | 14,893 (88) | 8,861 (89) |
| Europe other than Sweden | 379 (3.2) | 631 (3.9) | 962 (5.7) | 630 (6.3) |
| Rest of the world | 208 (1.7) | 436 (2.7) | 1,016 (6.0) | 432 (4.4) |
| Diabetes duration (years) | 23.8 (13) | 17.8 (14) | 14.0 (14) | 7.8 (8.7) |
| History of diabetic ketoacidosis, n (%) | 1,594 (13) | 1,915 (12) | 1,094 (6.5) | 491 (4.9) |
| History of severe hypoglycemia, n (%) | 338 (2.8) | 313 (2.0) | 203 (1.2) | 93 (0.9) |
| Diabetes treatment | | | | |
| Insulin pump use, n (%) | 3,808 (32) | 3,221 (20) | 969 (5.7) | 368 (3.7) |
| Multiple daily insulin injections, n (%) | 8,123 (68) | 12,777 (80) | 15,902 (94) | 9,555 (96) |
| Use of oral blood glucose lowering medication, n (%) | 53 (0.4) | 60 (0.4) | 277 (1.6) | 443 (4.5) |
| History of cardiovascular disease, n (%) | 434 (3.6) | 569 (3.6) | 786 (4.7) | 490 (4.9) |
| History of stroke/TIA, n (%) | 135 (1.1) | 170 (1.1) | 230 (1.4) | 174 (1.8) |
| History of retinopathy, n (%) | 6,505 (54) | 6,785 (42) | 6,193 (37) | 2,578 (26) |
| eGFR (mL/min/1.73m$^2$) | 101 (29) | 103 (30) | 98 (28) | 91 (23) |
| Albuminuria, n (%) | 2,476 (21) | 2,861 (18) | 2,835 (17) | 1,087 (11) |
| Current smoking, n (%) | 1,642 (14) | 2,189 (14) | 2,647 (16) | 1,800 (18) |
| Body mass index (kg/m$^2$) | 24.7 (3.7) | 24.7 (3.9) | 25.1 (4.4) | 25 (3.7) |
| Systolic blood pressure (mmHg) | 123 (16) | 124 (16) | 126 (17) | 129 (17) |
| Diastolic blood pressure (mmHg) | 73 (8.9) | 73 (9.0) | 74 (9.1) | 76 (9.0) |
| HbA1c (mmol/mol) | 69 (16) | 66 (16) | 62 (17) | 59 (15) |
| LDL cholesterol (mmol/L) | 2.6 (0.8) | 2.6 (0.8) | 2.7 (0.8) | 2.8 (0.9) |
| Use of blood pressure-lowering medication, n (%) | 2,139 (18) | 2,588 (16) | 2,900 (17) | 2,155 (22) |
| Use of lipid lowering-medication, n (%) | 1,499 (13) | 2,073 (13) | 3,006 (18) | 2,515 (25) |

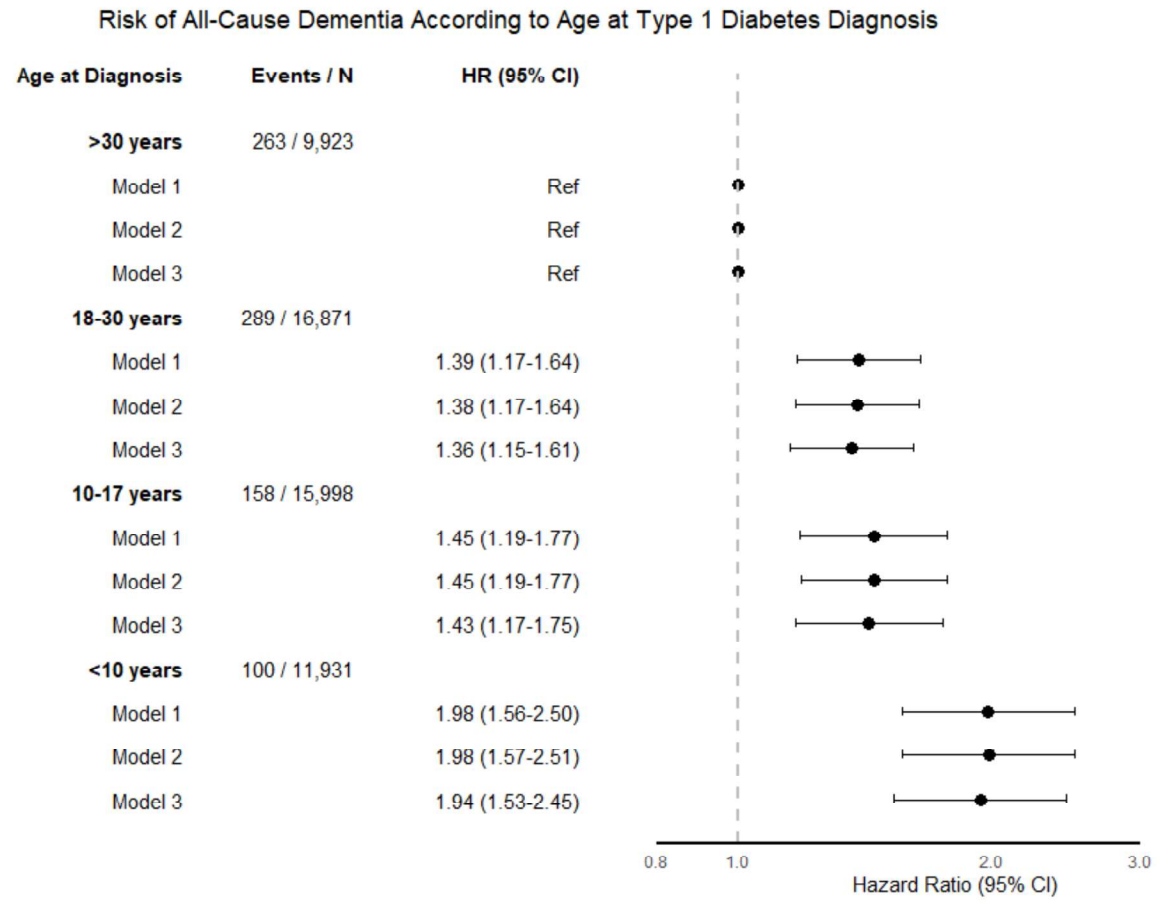
Type 1 diabetes was defined as insulin use and age at onset ≤30 years, or based on clinical judgement (irrespective of age). Continuous data presented as mean (SD) and categorical data presented as n (%). CVD: Cardiovascular disease. TIA: Transient ischemic attack. Severe hypoglycemia: Hypoglycemic event with an insult or requiring others to recover in the past 12 months. eGFR: estimated glomerular filtration rate. LDL cholesterol: low-density-lipoprotein cholesterol.

**Supplementary Table S6. Incidence of all-cause dementia and dementia subtypes in individuals with type 1 diabetes defined as insulin use and age at onset ≤30 years, or based on clinical judgement (irrespective of age).**

| Age at onset | Events (n/Ntot) | Incidence rate per 1000 person years (95% CI) | Incidence rate per 1000 person years[1] (95% CI) |
|---|---|---|---|
| *All-cause dementia* | | | |
| <10 years | 100/11,931 | 0.23 (0.19-0.28) | 0.28 (0.15-0.45) |
| 10-17 years | 158/15,998 | 0.32 (0.27-0.38) | 0.28 (0.16-0.46) |
| 18-30 years | 289/16,871 | 0.62 (0.55-0.70) | 0.37 (0.23-0.57) |
| >30 years | 263/9,923 | 1.30 (1.15-1.47) | 0.57 (0.38-0.80) |
| *Alzheimer's disease* | | | |
| <10 years | 23/11,931 | 0.05 (0.03-0.08) | 0.06 (0.02-0.17) |
| 10-17 years | 27/15,998 | 0.05 (0.04-0.08) | 0.05 (0.01-0.15) |
| 18-30 years | 72/16,871 | 0.16 (0.12-0.19) | 0.09 (0.03-0.22) |
| >30 years | 68/9,923 | 0.34 (0.26-0.42) | 0.13 (0.05-0.26) |
| *Vascular dementia* | | | |
| <10 years | 36/11,931 | 0.08 (0.06-0.11) | 0.10 (0.03-0.22) |
| 10-17 years | 48/15,998 | 0.10 (0.07-0.13) | 0.08 (0.03-0.20) |
| 18-30 years | 81/16,871 | 0.17 (0.14-0.22) | 0.11 (0.04-0.24) |
| >30 years | 61/9,923 | 0.30 (0.23-0.38) | 0.16 (0.08-0.31) |
| *Non-Alzheimer's-non-vascular dementia* | | | |
| <10 years | 41/11,931 | 0.09 (0.07-0.13) | 0.12 (0.04-0.25) |
| 10-17 years | 83/15,998 | 0.17 (0.14-0.21) | 0.15 (0.07-0.29) |
| 18-30 years | 136/16,871 | 0.29 (0.25-0.35) | 0.17 (0.08-0.32) |
| >30 years | 134/9,923 | 0.66 (0.56-0.78) | 0.27 (0.15-0.45) |

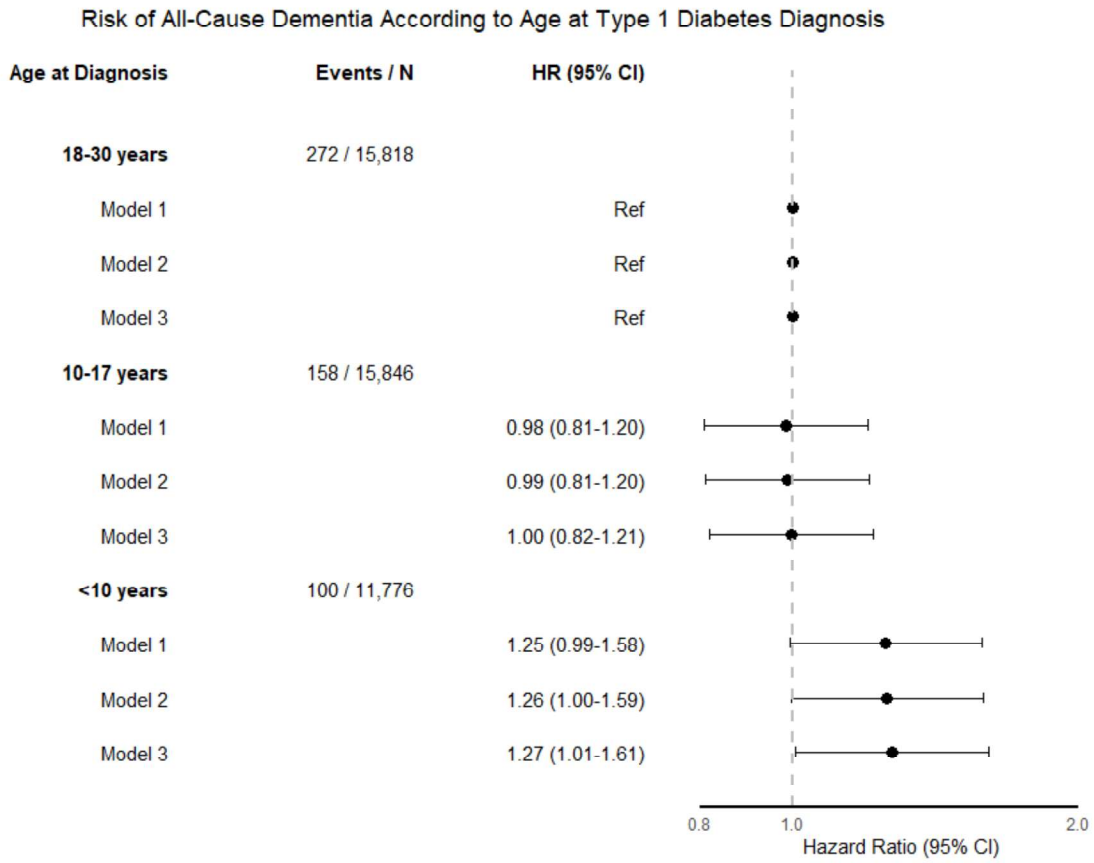
Type 1 diabetes was defined as insulin use and age at onset ≤30 years, or based on clinical judgement (irrespective of age). [1]Incidence rates standardized for age and sex. Total follow-up included 1,590,722 person years. CI: confidence interval.

**Supplementary Figure S5. Risk of all-cause dementia in individuals with type 1 diabetes defined as insulin use and age at onset ≤30 years, or based on clinical judgement (irrespective of age).**




*Type 1 diabetes was defined as insulin use and age at onset ≤30 years, or based on clinical judgement (irrespective of age). Median follow-up was 13.3 [7.3-19.0] years. Model 1: Crude. Model 2: adjusted for sex. Model 3: adjusted for sex, education, marital status and region of birth at cohort entry. All Cox models have age as the time axis. CI: Confidence Interval. HR: Hazard Ratio. Ref: Reference. >30 years at diabetes onset group was the reference category.

**Supplementary Figure S6. Risk of all-cause dementia in individuals with type 1 diabetes starting follow-up from cohort entry instead of follow-up from age at diabetes onset.**




Median follow-up was 13·4 [7·2-19·1] years. Model 1: Crude. Model 2: adjusted for sex. Model 3: adjusted for sex, education, marital status and region of birth at cohort entry. All Cox models have age as the time axis, follow-up started from cohort entry. CI: Confidence Interval. HR: Hazard Ratio. Ref: Reference. 18-30 years at diabetes onset group was the reference category.

**Supplementary Table S7. Age-specific incidence rates of all-cause dementia in age bands, stratified by age at type 1 diabetes onset.**

| Age band | Incidence rate per 1,000 person years (95% CI) | | |
|---|---|---|---|
| | Age at type 1 diabetes onset | | |
| | <10 years | 10-17 years | 18-30 years |
| 50-60 years | 0.58 (0.33-0.93) | 0.29 (0.16-0.50) | 0.52 (0.36-0.73) |
| >60-65 years | 1.96 (1.09-3.22) | 1.90 (1.24-2.79) | 1.01 (0.63-1.53) |
| >65-75 years | 5.13 (3.61-7.07) | 3.87 (2.93-5.03) | 3.41 (2.72-4.22) |
| >75 years | 19.71 (12.76-29.1) | 14.82 (11.07-19.44) | 17.17 (14.23-20.53) |

Incidence rates are presented as number of events per 1,000 person-years with 95% exact Poisson confidence intervals. Person-years were calculated within each age band using a delayed-entry approach, with follow-up time split at age boundaries 50, 60, 65, and 75 years. CI=confidence interval.