INTRODUCTION

The risk of kidney disease, stroke and coronary heart disease is increased in individuals with either type 1 or type 2 diabetes. In addition, a higher incidence of cancer has been found in people with type 2 diabetes. Different types and locations of cancer have been associated with diabetes such as cancer of the pancreas, stomach, colon, testes...
and the endometrium. Insulin and IGF1 as growth factors but also inflammation and hyperglycaemia per se have been implicated to be links between diabetes and cancer.2–4 The great majority of epidemiological studies on the association between cancer and diabetes suffer methodological drawbacks e.g. do not separate type 1 and type 2 diabetes.5 The greater part of data involves persons with type 2 diabetes. In contrast to individuals with autoimmune diabetes, persons with type 2 are older at onset, have experienced many years of relative hyperinsulinemia before clinical diagnosis and often suffer from overweight, a known risk factor for certain cancers. It is therefore of interest to separately study the risk of cancer in persons with type 1 diabetes.

For type 1 diabetes, a few population-based studies3,5 exist and reviews6,7 have found diverging results, probably due to differences in study design, type 1 diabetes definition, outcome measures and study region.

Considering the continued uncertainties seen in previous studies regarding the risk of cancer for persons with type 1 diabetes, we wanted to explore the association between type 1 diabetes and cancer in a proportionally large, nation-wide cohort of prospectively recorded cases of type 1 diabetes in Sweden, all diagnosed before 15 years of age.

2 | STUDY POPULATION AND METHODS

The Swedish Childhood Diabetes Register (SCDR), a prospective research register has recorded incident cases of type 1 diabetes in children below 15 years of age in Sweden since July 1st, 1977. The coverage has been estimated to be 96%–99%.8 All children with newly diagnosed type 1 diabetes in Sweden are initially treated at paediatric clinics in a hospital setting. Until 2010, the clinics reported their persons with type 1 diabetes to the SCDR with the date of diagnosis, birth date and each person’s unique identification number. Since 2010, the SCDR instead recruits cases from the Prescribed Drug Register, an official, nationwide register kept by the Swedish National Board of Health and Welfare.9 Any child below 15 years of age with at least two prescriptions of insulin will be registered in the SCDR. The Prescribed Drug Register started in 2005 and includes all drugs in Sweden retrieved on prescription at a pharmacy. During a 3-year period, 2007, 2008 and 2009, the concordance between the data sampling methods were found to be 97%. Over the course of this period 3% more people with type 1 diabetes were identified through filled prescriptions of insulin (n = 2011) than through reports from paediatric clinics (n = 1950). In this study, we included 18,724 cases with childhood-onset type 1 diabetes recorded in the SCDR from 1 July 1977 to 31 December 2013.

What is already known about this subject?
• Persons with type 2 diabetes have an increased risk of cancer. However, for type 1 diabetes, existing studies have found diverging results.

What is the key question?
• What is the risk of cancer in individuals with childhood-onset type 1 diabetes in Sweden?

What are the new findings?
• Women with type 1 diabetes have a small but significantly elevated risk of cancer. Only women who were diagnosed with type 1 diabetes at an age of 0–4 years had a significantly elevated cancer incidence. No risk elevation was seen in men.

How might this impact on clinical practice in the foreseeable future?
• Clinicians need to be aware of the increased risk of cancer in women with childhood-onset type 1 diabetes and ensure that these persons adhere to cancer screening programs.

For every type 1 diabetes patient, four referents without type 1 diabetes were randomly selected from the general population using the total population register at Statistics Sweden. The referents were individuals not recorded in the SCDR, matched on month of birth and place of residence. If a referent before the age of 15 developed type 1 diabetes, they were detected as a case and were subsequently removed as a referent. Statistics Sweden organized the individual-level linking of the SCDR and the referents to the Swedish Cancer Register and the Cause of Death Register, using personal identification numbers. Pseudonymized data was returned to the researchers.

The Swedish Cancer Register started in 1958 with the purpose to chart all malignant tumours. The register allows the computation of yearly incidence of various types of cancer, controlling for sex and age. The overall completeness of the cancer register is 96%. This is comparable to other high-quality registers in Northern Europe.10,11 From the cancer register we also retrieved date of cancer diagnosis and type of cancer according to the World Health Organization ICD classification system.12 All individuals in the cancer register are registered with an ICD 7 code and from 1997 they all have an ICD 10 code. In our material, we only included cancers considered to be malignant in the Cancer register.
The Cause of Death Register started in 1961 and provides information on time, date and cause of death. The date of death is considered to be very reliable and was the variable used in this study.13 The study was approved by the Regional Ethics Review Board in Umeå (Dnr 2014-331-32M) and by the Ethics committees at the National Board of Health and Welfare and Statistics Sweden, respectively and was performed according to the Helsinki declaration.

2.1 Statistical analysis

Descriptive data are presented as medians and quartiles. To compare the risk of cancer overall among persons with type 1 diabetes to that of the Swedish general population, we calculated standardized incidence ratios (SIR) with 95% confidence intervals between the observed incidence (cases with cancer/person years at risk) of cancer in the type 1 diabetes cohort and the expected incidence in the Swedish population, based on data from the Cancer Register, controlling for calendar year, sex and age. All participants were followed until incident cancer, death or end of study. Migration was not controlled for due to missing data. To further evaluate the risk of cancer in individuals with type 1 diabetes, we analysed the interaction between type 1 diabetes and sex. Since this interaction was significant, the following Cox proportional hazards models, providing hazard ratios and 95% confidence intervals, was stratified by sex. The interaction between type 1 diabetes and age of type 1 diabetes onset was significant at the 0.1 level but based on our previous studies we wanted to further explore the effect of age of type 1 diabetes onset14 on the subsequent risk of cancer. Thus, we performed stratified analysis according to age at onset groups. The models were adjusted for birth year and the model including all ages was adjusted for age at start of follow up. To account for death as a competing risk, proportional subdistribution hazard models15 were also used and also adjusted for birth year or age at start of follow up. For the persons with diabetes, follow up started with diabetes diagnosis and ended with cancer, death or end of study. For the referents, follow up started at the corresponding index date of onset of diabetes and ended with cancer, death or end of study. The date for the end of study was 31 December 2013. Age at onset of diabetes (start of follow up) was categorized into 0–4 years of age, 5–9 years of age and 10–14 years of age. Those who had a cancer diagnosis before the index date were excluded. The relative risk of specific cancers was calculated by dividing the number of cases per 100,000 among individuals with childhood-onset diabetes and referents. Cells with no cases, for example, bladder cancer among referent women, was addressed with the Haldane-Anscombe correction.16,17 Statistical analyses were performed using SPSS 27.0 (IBM SPSS Statistics for Windows) and R.18

3 RESULTS

3.1 Descriptive data

We included 18,724 prospectively recorded individuals with type 1 diabetes between 1 July 1977 and 31 December 2013. The study group included 8835 women (47%) and 9889 men (53%). The median age at diabetes onset was 9 years (interquartile range 6–12). The median age at the end of follow up (death or end of study) was 23 years (interquartile range 16–33). In this cohort, we found 125 individuals with type 1 diabetes with 135 cancer diagnoses. We excluded 37 persons with cancer that were diagnosed before the individual’s diabetes diagnosis (Figure 1). Ten individuals in the cohort had two cancer diagnoses. Of the 125 individuals with cancer, 76 were women (61%) and 49 were men (39%). The median age at first cancer diagnosis was 28 years (interquartile range 20–35) and median calendar year at first cancer diagnosis was 2008 with a range from 1985 to 2013. The median duration of follow up from diabetes diagnosis to incident cancer, death or end of study was 14 years (interquartile range 7–24) with a minimum and maximum follow up time of 0 and 37 years respectively. The median duration from diabetes diagnosis to the first cancer diagnosis was 19 years (interquartile range 10–26) (Table 1). Among the 125 individuals with both type 1 diabetes and cancer, 15 (7.5%) had died before the end of follow up, of whom 11 individuals died within 3 years after a cancer diagnosis.

For every person with type 1 diabetes, we included four referents without diabetes. The referent group included
74,706 individuals, 36,455 women and 38,251 men respectively. In the reference group, we found 440 individuals with 476 cancer diagnoses. If cancer had occurred in a person with type 1 diabetes or in a referent before the onset of diabetes in the index case they were excluded from the analyses.

### 3.2 Analysis

With 135 cases of cancer among 125 individuals with type 1 diabetes and an expected number of cases with cancer in the Swedish population during the same time at 118, the overall SIR (95% CI) for cancer, adjusted for age, sex and calendar year of cancer incidence, was 1.14 (0.96, 1.35). Stratification by sex showed that women with childhood-onset type 1 diabetes have an increased risk of cancer compared to the female Swedish population, with SIR of 1.28 (1.02, 1.58) (Table 2). In the subpopulation of men, no elevated risk could be established with SIR of 0.98 (0.73, 1.28) (Table 2). Of the 440 individuals in the reference group with cancer, 230 were women (52%) and 210 were men (48%) (Table 1).
Using Cox regression, calculating time to cancer, we compared cancer risk between persons with type 1 diabetes and their referents without type 1 diabetes. In the estimation of the risk of cancer, there was significant interaction between type 1 diabetes and sex, thus we performed stratified analysis by sex. We found an increased rate of cancer by 42% for women with type 1 diabetes (Table 3). We performed a sensitivity analysis where we excluded those with cancer diagnosis up to 12 months after diabetes onset yielding a hazard ratio (95% CI) of 1.35 (1.03, 1.76). No significant elevated cancer rate was seen for men (Table 3). There was no statistical difference in the number of cancer cases between men and women overall or among the referents. However, in the group of interest in this paper, i.e., individuals with onset of type 1 diabetes before 15 years of age, there was a significant difference in the cumulative incidence of cancer among individuals with type 1 diabetes (365 per 100,000, 95% CI 127 per 100,000, 602 per 100,000, p = 0.002). The cumulative incidence rates of cancer over time for persons with type 1 diabetes and their referents are illustrated in Figure 2.

The mortality rate was higher among individuals with type 1 diabetes, both among women (1.2% among those with type 1 diabetes vs. 0.2% among referents, p < 0.001) and men (2.0% among those with diabetes type 1 and 0.6% among referents, p < 0.001). Mortality also differed across the age at onset groups, where the proportions among 0–4, 5–9 and 10–14 years were 1.2%, 1.3% and 2.1% (p < 0.001), respectively, for type 1 diabetes individuals and 0.3%, 0.2% and 0.2%, respectively, among referents. To account for death as a competing risk, proportional subdistribution hazard models were estimated, showing only minor differences in the hazard ratios to the Cox regression (Table 3). The subdistribution hazard ratios show that type 1 diabetes is associated with an increase in the incidence of cancer among women. When exploring the differences in cancer incidence among different ages at the start of follow up groups, we found that women who were diagnosed with type 1 diabetes during age 0–4 years had an elevated cancer incidence compared to referents (Table 3). For those who developed diabetes later in childhood no such effect was found. Among women, cancer of the breast was most common (Table S1). In women with onset of type 1 diabetes before 5 years of age, there were no predominant types of cancer (Table S1). Among men, cancer of the testis was most common (Table S2).

### Table 2

| Sex     | Observed cancers | Expected cancers | SIR (95% CI)       |
|---------|------------------|------------------|--------------------|
| Total   | 135              | 118.18           | 1.14 (0.96, 1.35)  |
| Women   | 83               | 65.06            | 1.28 (1.02, 1.58)  |
| Men     | 52               | 53.13            | 0.98 (0.73, 1.28)  |

### Discussion

In this comparably large, prospective and population-based study, we found a small but statistically significant excess risk of cancer incidence among women with type 1 diabetes with a maximum follow up until the age of 51. The adjusted effect was mainly seen in individuals with diabetes onset at age 0–4 years. Among men, we found no increased risk. As expected in this young population, breast and testicular cancer were the most common types of cancer among women and men respectively. The number of cases for each cancer type were small and differences between type 1 diabetes persons and reference individuals should be interpreted with caution. A possible explanation to the increased risk in the youngest age-group could be longer exposure to the metabolic effects of type 1 diabetes or a greater vulnerability to these effects. These findings need to be confirmed in future studies.

Our results agree with the findings in some previous studies. A retrospective Taiwanese population-based study has shown a 13% increase in all-cause cancer and the effects were mainly seen in women. In a recent multi-centre study from five different European countries, the authors found a higher overall cancer risk among women (hazard ratio 1.07) and significantly higher risk for certain types of cancer. No increase in all-cause cancer risk for men was observed. In that study, diabetes diagnosis was based on hospital discharge diagnose and all individuals diagnosed below 40 years of age were considered to have type 1, which may have introduced a misclassification regarding type of diabetes. A review by Gordon-Dseagu et al. showed mixed results regarding both the association between type 1 diabetes and cancer incidence and the cause-specific cancer mortality. Results differed by method of defining type 1 diabetes, by study region and by measure of outcome data. A Swedish register study showed a small risk increase (relative risk 1.2) for cancer in hospitalized persons with type 1 diabetes and the types of cancers differed from those reported for type 2 diabetes. Again, the diagnosis of type of diabetes was uncertain and based on hospital discharge diagnosis using different coding systems. Finally, an Australian study looking at time trends in mortality among persons with type 1 diabetes reported that while all-cause, cardiovascular disease and diabetes mortality rate had decreased over time, no such decline was seen for cancer.

It has been suggested that hyperinsulinemia promotes carcinogenesis directly through insulin action or indirectly...
through the effect of insulin-like growth factor (IGF)-1.²,⁴ Hyperinsulinemia leads to an increased bioactivity of IGF-1 by inhibiting IGF binding protein-1. IGF-1 is described to have more potent mitogenic and anti-apoptotic activities than insulin. Hyperinsulinemia has thus been considered as a primary factor for cancer growth whereas hyperglycaemia has been considered a surrogate marker. High glucose levels may exert direct and indirect effect upon cancer cells to promote proliferation. High glucose levels in the presence of insulin deficiency, however, does not seem to contribute to pathologic cell growth.² Diabetes with poor metabolic control causes a permanent pro-inflammatory condition and inflammatory cytokines such as interleukin 6 and plasminogen activator inhibitor-1 may play important roles in cancer progression.² In the present study, we have no information on metabolic control and thus cannot link clinical data to the risk of developing cancer in individuals with childhood-onset type 1 diabetes.

It may be assumed that people with chronic disease, e.g., type 1 diabetes, who have regular contact with medical care may be detected differently with other diseases including cancer compared with people without such contact. The association between chronic diseases and cancer detection is complex, where the characteristics of the underlying chronic illness as well as patient characteristics

| Onset age of type 1 diabetes | Number of cancers in people with type 1 diabetes | Number of cancers in referents | Hazard ratio (95% CI) | Subdistribution hazard ratio (95% CI) |
|------------------------------|--------------------------------------------------|-------------------------------|-----------------------|--------------------------------------|
|                              | Women | Men | Women | Men | Women | Men | Women | Men | Women | Men |
| 0–14 years                   | 76    | 49  | 230   | 210 | 1.42 (1.10, 1.85)³ | 0.91 (0.66, 1.24)³ | 1.40 (1.08, 1.82)³ | 0.89 (0.66, 1.22)³ |
| 0–4 years                    | 13    | 5   | 20    | 26  | 2.91 (1.45, 5.86)ᵇ | 0.73 (0.28, 1.90)ᵇ | 2.89 (1.45, 5.76)ᵇ | 0.72 (0.28, 1.88)ᵇ |
| 5–9 years                    | 24    | 19  | 80    | 65  | 1.18 (0.75, 1.87)ᵇ | 1.20 (0.72, 2.00)ᵇ | 1.18 (0.75, 1.87)ᵇ | 1.19 (0.71, 1.97)ᵇ |
| 10–14 years                  | 39    | 25  | 130   | 119 | 1.34 (0.94, 1.92)ᵇ | 0.81 (0.53, 1.25)ᵇ | 1.30 (0.91, 1.85)ᵇ | 0.79 (0.52, 1.22)ᵇ |

ᵃAdjusted for age at start of follow up.
ᵇAdjusted for birth year.

FIGURE 2 Cumulative incidence of cancer among individuals with type 1 diabetes and referents by sex (women blue line, men red line, type 1 diabetes dashed line, referents solid line) over years from start of follow-up. Accounting for death as a competing risk, the p-values for comparing the subdistribution for the cause cancer across groups (type 1 diabetes/referents) are 0.014 and 0.511 for women and men respectively [Colour figure can be viewed at wileyonlinelibrary.com]
and health care factors all contribute. Type 1 diabetes is a complex disease with a lifetime of contact with specialized medical care. Unlike type 2 diabetes, no particular cancer type has yet been associated with type 1 diabetes. We have found no support for the notion that cancer among women with type 1 diabetes is explained by earlier detection. Thus, the explanation of the higher cancer risk must be sought elsewhere. One possible explanation is that the complexity of the care of type 1 diabetes may interfere with the attention to other health issues. Cancer screening is one group of preventive health services where women with diabetes seem to differ from non-diabetic women as reported in several studies. Two Spanish studies indicated that women with diabetes underuse breast and cervical cancer screening. In addition, a large study from 12 states in the US found that American women with diabetes did not attend cervical cancer screening as often as women without diabetes. A Canadian study showed that women with diabetes, regardless of socioeconomic status, had lower rates of screening mammograms and a US study from the National Cancer institute, observed that women with diabetes were at increased risk of late-stage diagnosis of breast cancer. From Sweden, we have no indication of such screening neglect among individuals with diabetes, but this needs further investigation.

Our study has some limitations. With the present study population, we cannot say anything about the risk of cancer if the type 1 diabetes diagnosis occurs after the age of 15 years. The maximum follow-up time was 37 years, which means a maximum age at the end of follow up of 51 years. With age being an important risk factor for cancer, it is necessary to further follow up the cohort. Also, we did not have access to information on other potential risk factors for cancer such as weight, smoking habits, number of pregnancies, hormone treatment or average glucose levels. The present results are population-based but only applicable to Sweden.

The strength of our study is the accurate definition of type 1 diabetes. All individuals received their diagnosis in a paediatric clinic before the age of 15 and they were all insulin-treated from the start. We are not aware of any other study that has used the same rigorous method in defining type 1 diabetes as exposure when assessing cancer risk. In the large, European multicentre study, diabetes diagnosis was based on hospital discharge diagnosis and all persons diagnosed below 40 years of age were considered as type 1 diabetes, which may have introduced misclassification regarding type 2 diabetes.

In conclusion, in a large and well-defined population-based cohort of middle-aged individuals with childhood-onset type 1 diabetes, we found a slightly increased risk of cancer incidence among women, especially among those with young age at onset of diabetes. No increased risk was found among men. The cohort should be further followed up.

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AUTHOR CONTRIBUTIONS
Marie Fredriksson contributed to design of the study, compiled and analysed the data, wrote the first draft of the manuscript, reviewed and edited the manuscript. Emma Persson contributed to design of the study, assisted with the statistical analysis and reviewed the manuscript. Gisela Dahlquist contributed to design of the study, contributed to the discussion, and reviewed and edited the manuscript. Anna Mållsten contributed to the discussion and reviewed and edited the manuscript. Torbjörn Lind contributed to design of the study, assisted with the statistical analysis, contributed to the discussion and reviewed and edited the manuscript.

DATA AVAILABILITY STATEMENT
The data that support the findings of this study are available from the National Board of Health and Welfare, but restrictions apply to the availability of these data, which were used under license for the current study, and so are not publicly available. Data are, however, available from the authors upon reasonable request and with permission of the National Board of Health and Welfare.

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SUPPORTING INFORMATION
Additional supporting information may be found in the online version of the article at the publisher’s website.

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