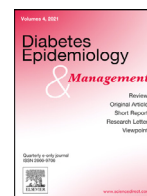




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Original article

The risk of developing type 2 diabetes after gestational diabetes: A registry study from Finland



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ABSTRACT

Aims: Women with a history of gestational diabetes (GDM) have an increased risk of developing type 2 diabetes (T2DM). We studied the risk for T2DM in women with and without GDM in relation to body mass index (BMI) and examined whether insulin treatment for GDM associates with the risk of developing T2DM. In addition, we investigated whether the risk of developing T2DM after GDM had changed in 15 years.

Methods: We used data by linking four registers; Medical Birth Register, Hospital Discharge Register and Primary Care Register run by THL Finnish Institute for Health and Welfare, and Medical Reimbursement Statistics run by the Social Insurance Institution of Finland (Kela). Registry data were collected from 2005 to 2020. The follow-up started from woman's delivery in 2006–2020 and ended to the diagnosis of T2DM or December 2020. Cox proportional hazard modelling was used to estimate the effect of GDM exposure to T2DM. To assess whether the risk of developing T2DM after GDM had changed in 15 years, we compared the HR between years 2006–2008 and 2018–2020.

Results: In total, 462 401 women were included in the study: 96 353 (21%) women had previous GDM. There were 5370 (1.2%) women who developed T2DM after childbirth during the follow-up. Among women with prior GDM, 3995 (4.1%) developed T2DM, while 1375 (0.4%) women without prior GDM developed T2DM during follow-up. The mean follow-up was 6.86 years (SD 4.21) for women with GDM and 9.07 years (SD 4.35) for women without GDM. The hazard ratio (HR) for developing T2DM after GDM was 18.49 (95% CI 17.39–19.67). The incidence of T2DM in women with a history of GDM began to rise almost steadily from the first year of follow-up. As BMI increased, T2DM incidence increased in both women with and without prior GDM but more in women with prior GDM. Insulin treatment had an independent association with increased risk of T2DM (HR 3.81, 95% CI 3.57–4.07). We did not observe any difference in HR between years 2006–2008 and 2018–2020.

Conclusions: The relative risk for T2DM was 11-fold for women with previous GDM compared to women without previous GDM. A higher BMI and insulin treatment increased the risk of future diabetes. All measures to prevent the conversion of GDM to T2DM should be taken especially among women with overweight or obesity.

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Introduction

Gestational diabetes mellitus (GDM) is an established risk factor for type 2 diabetes (T2DM). In 2009 Bellamy et al. showed that women with GDM have 7.4-fold (95% CI 4.8–11.5) risk of developing T2DM compared with women who had a normoglycaemic pregnancy [1]. A recently published meta-analysis with a longer follow-up period (mean follow-up of 9.1 years, range from 1 to 25 years) revealed that the relative risk for T2DM is 9.5 times higher in women with previous GDM than in women with a normoglycaemic pregnancy. The risk is 17 times higher in the first five years after delivery [2]. Recent studies have shown that the cumulative incidence of T2DM increases steadily over time after GDM [2,3].

Overweight (BMI ≥ 25 kg/m²), family history of diabetes, non-white ethnicity, multiparity and older maternal age are significant risk factors for progression to diabetes after GDM [4]. Also, abnormal glucose levels in oral glucose tolerance test (OGTT) and high glycated hemoglobin (HbA1c) during pregnancy are associated with an increased risk of future diabetes. Women with GDM who require insulin treatment or have pregnancy complications such as hypertensive disease and preterm delivery (<37 weeks) are more likely to develop T2DM compared with other GDM women [4,5].

In 2008, after an international multicenter cohort Hyperglycemia and Adverse Pregnancy Outcome (HAPO) study demonstrated an association between plasma glucose levels and adverse pregnancy outcomes with no obvious thresholds at which risks increased, Finland started using Finnish Current Care Guidelines and began comprehensive testing with 75 g OGTT at gestational weeks 24–28, and at gestational weeks 12–16 for those at high risk for developing GDM [6]. The Finnish diagnostic criteria for GDM were plasma glucose levels ≥ 5.3 mmol/l (fasting), ≥ 10.0 mmol/l (1 h) and ≥ 8.6 mmol/l (2 h). As in the criteria of The International Association of the Diabetes and Pregnancy Study Groups and later WHO, one abnormal value was considered diagnostic for GDM. This led to the diagnoses of greater number but milder GDM cases [7].

A higher pre-pregnancy BMI has been reported to increase the risk of T2DM after GDM [8,9,10]. However, to our knowledge, there have been no studies comparing the incidence of T2DM in relation to pre-pregnancy BMI in women with and without a history of GDM.

In this study, we explored the risk for T2DM for Finnish women with and without prior GDM. We also examined their risk for T2DM in relation to pre-pregnancy BMI and the need for insulin treatment in GDM pregnancy. We also investigated whether the risk of developing T2DM after GDM had changed in 15 years.

Participants and methods

Data sources

In Finland, every citizen and permanent resident has a personal identification number, which enables the combination of different register data. We used data from four different registers:

- Medical Birth Register run by THL Finnish Institute for Health and Welfare. All live births and stillbirths with a gestational age of at least 22+0 weeks or with a birth weight of ≥ 500 grams are included in the register. The register includes mother's and child's identification numbers and information on maternal background, health care and interventions during pregnancy and delivery and newborn's outcome until age of 7 days. After linkage to Central Population Register and the Cause-of-Death Register, the register

is considered complete in terms of numbers of births and newborns. Previous data quality studies have found that the majority of register content corresponded well or satisfactorily with the hospital record data [11]. We collected data from the Medical Birth Registry from 1 January 2006 to 31 December 2020.

- Care Register for Health Care run by THL Finnish Institute for Health and Welfare. This register collects information on all episodes of inpatient care in health care institutions and outpatient visits in public hospitals. The register contains information on each patient's background, hospitalization period, procedures in NCSP codes (NOMESCO Classification of Surgical Procedures), and main and secondary diagnosis in International Statistical Classification of Diseases and Related Health Problems (ICD-10) codes. According to a review of data quality studies more than 95% of discharges could be identified from the register [12]. We collected data from the Care Register for Health Care from 1 January 2005 to 31 December 2020.
- Register of Primary Health Care visits run by THL Finnish Institute for Health and Welfare. This register collects information on all primary health care visits in health care centers. The register contains information on each patient's background, primary care visit, and main and secondary diagnosis in ICD-10 or ICPC2 codes (International Classification of Primary Care, 2nd edition) since 2011. We collected data from the Register of Primary Health Care visits from 1 January 2011 to 31 December 2020.
- Reimbursement Entitlements for Medicines run by Kela – The Social Insurance Institution of Finland. The register contains data on the special and limited reimbursement entitlements for medication from the databases maintained by Kela and by workplace sickness funds. We collected data from the Reimbursement Entitlements for Medicines from 1 January 2005 to 31 December 2020.

Definition of cases and controls

Women who had given birth between 2006 and 2020 and had gestational diabetes and who did not have gestational diabetes in any pregnancy between 2006 and 2020 were identified from the Medical Birth Register, where healthcare professionals enter the mother's diagnoses during pregnancy. In 2006–2020, the Medical Birth Register had information on 800 570 singleton births by 467 838 women. Of these, 96 144 (21 %) women were diagnosed with GDM. Gestational diabetes was defined as ICD-10 code O24.4. A GDM diagnosis required at least one abnormal value in the 75 g OGTT at 24–28 weeks of pregnancy (or at 12–16 weeks of pregnancy). The diagnostic limit values for GDM were 5.3 mmol/l (fasting), ≥ 10.0 mmol/l (1 h) and ≥ 8.6 mmol/l (2 h).

Women with pre-existing T1DM in pregnancy, childbirth and the puerperium (O24.0) or pre-existing T2DM (O24.1) and women with T2DM (E11), other specified diabetes mellitus (E13) or unspecified diabetes mellitus (E14) registered in the Hospital Register or Primary Care Register during pregnancy were excluded from the material. Also, women who got special reimbursement granted by Kela before or during pregnancy with diagnosis of E11 were excluded from the material. The composition of the material can be seen in Figure 1.

Outcome variable

The main outcome was the development of type 2 diabetes. This was defined as occurrence of type 2 diabetes ICD-10 code E11 or ICPC2 code T90.

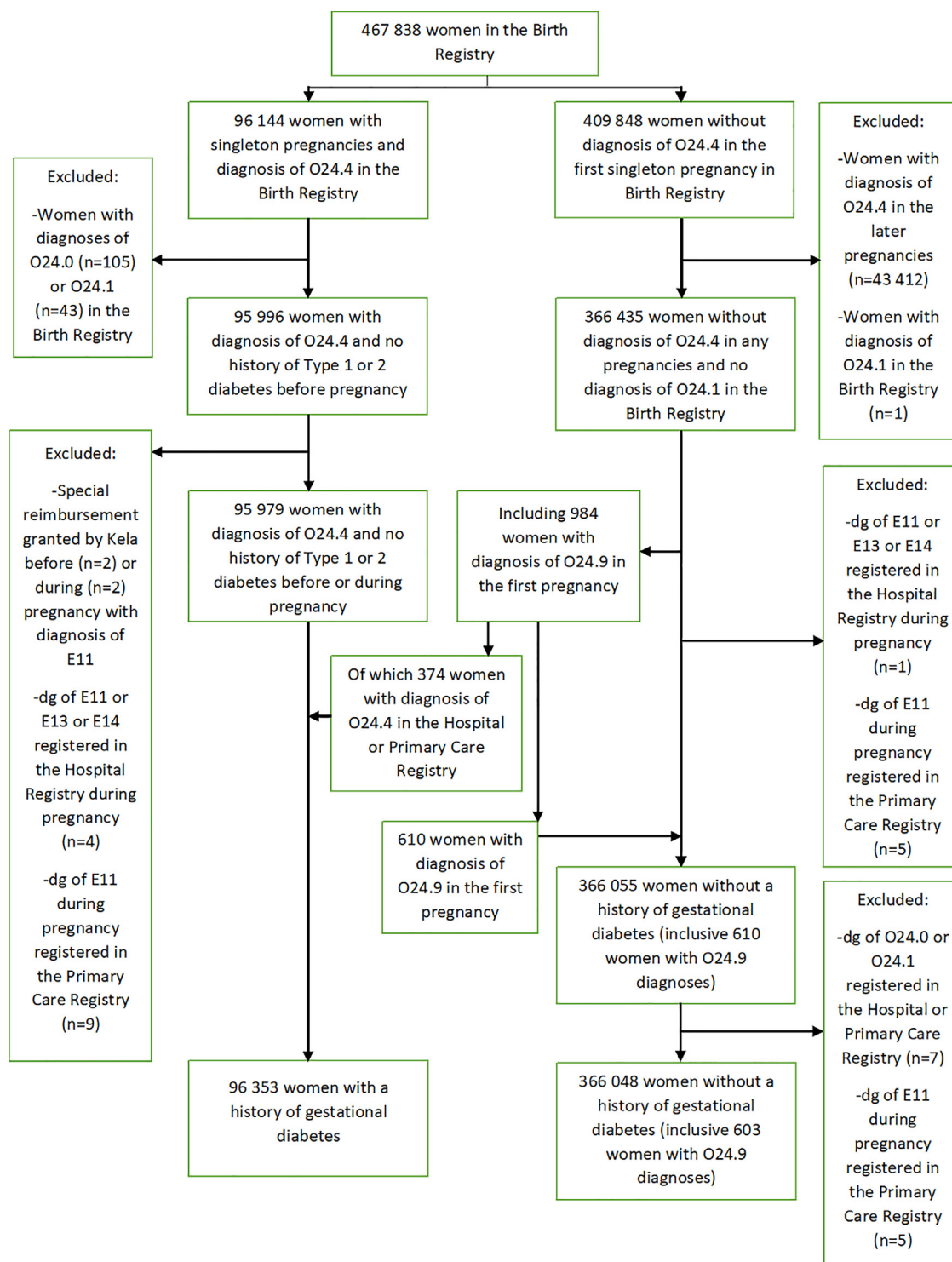


Fig. 1. The data was created based on the Birth Registry and the information was combined with data from the Hospital Registry, Primary Care Registry and The Social Insurance Institution of Finland (Kela). Dg = diagnosis. O24.0: Pre-existing type 1 diabetes mellitus, in pregnancy, childbirth and the puerperium. O24.1: Pre-existing Type 2 Diabetes Mellitus, in Pregnancy, Childbirth and the Puerperium. O24.4: Gestational diabetes mellitus. O24.9: Unspecified diabetes mellitus in pregnancy, childbirth and the puerperium. E11: Type 2 Diabetes Mellitus. E13: Other specified diabetes mellitus. E14: Unspecified diabetes mellitus.

The Medical Birth Register data were linked to all subsequent hospitalizations (since 2006), primary health care visits (since 2011) and reimbursement entitlements for medicines to find all women with deliveries who received T2DM diagnoses after delivery between 1 January 2006 and 31 December 2020.

Covariates

Women's age, parity, pre-pregnancy BMI and information on gestational diabetes and initiation of insulin therapy during pregnancy were obtained from the Medical Birth Register. The pre-pregnancy

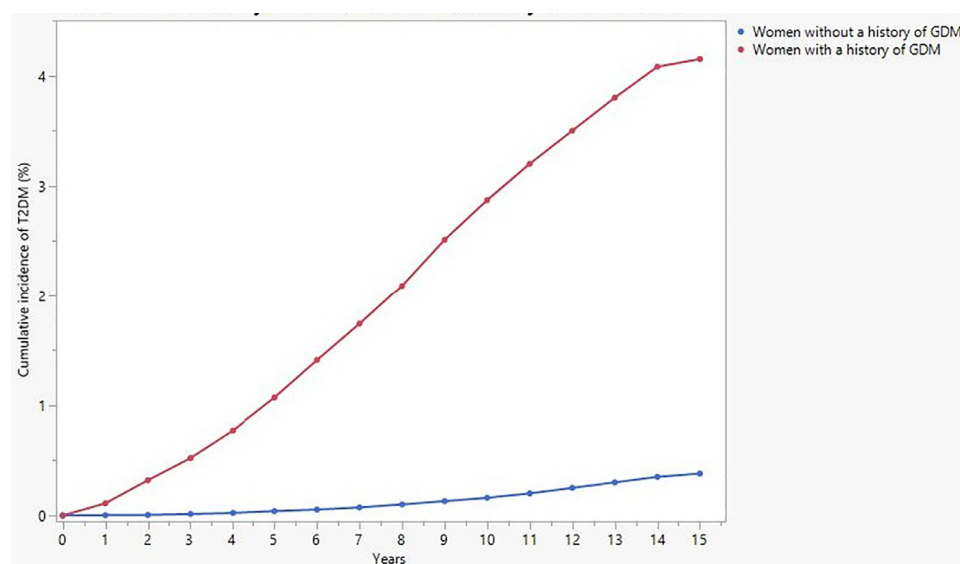


Fig. 2. The cumulative incidence of type 2 diabetes (T2DM) by time (years) in women with and without a history of gestational diabetes (GDM).

BMI had been calculated and entered into the register based on the women's self-reported weight and height.

Statistical methods

Cox proportional hazard modelling, taking account of the follow-up time, was used to estimate the effect of GDM exposure to T2DM. The follow-up started from woman's delivery in 2006 at the earliest and ended to the diagnosis of type 2 diabetes (E11 or T90) or 31 December 2020. If a woman had given birth several times in 2006–2020, the follow-up started from the first delivery during the period. Crude and adjusted hazard risk ratios (HRs) with 95% confidence intervals (CIs) were reported. The three adjusted models were I: age and parity, II: age, parity and BMI, and III: age, parity and insulin treatment started during pregnancy. All statistical analyses were performed using SAS version 9.4 (SAS Institute Cary, USA) in Kapseli which is a secure operating environment for the processing of anonymized data on individuals. Findata, the Finnish data permit authority for the social and health care sector, gave their data permit to use sensitive health register data in this study (permission THL/2703/14.02.00/2021).

Results

After combining the registries, we included 462 401 women in this study. There were 96 353 (21%) women with a history of GDM and 366 048 (79%) women without a history of GDM. The mean age of the women was 30 years (median 29 years, IQR 26–33 years). There were 14 091 women, meaning 15% of women with GDM, who started insulin treatment during pregnancy. The mean follow-up was 6.86 years (SD 4.21) for women with GDM and 9.07 years (SD 4.35) for women without GDM.

During 2006–2020, 5370 (1.2%) women received diagnosis of type 2 diabetes (E11 or T90). Among women with GDM, 3995 (4.1%) developed T2DM, while 1375 (0.4%) women without GDM diagnoses received T2DM diagnoses after childbirth (Figure 2). Hence, the risk for T2DM was 11 times higher in women with than without a history of GDM. Of the women with GDM who developed T2DM during the follow-up time, 1286 (32%) required insulin therapy during pregnancy.

The hazard ratio for developing T2DM after GDM was 18.49 (95% CI 17.39–19.67). 74% of women diagnosed with T2DM had previous

GDM. After adjusting for age and parity, the HR was still 17.01 (95% CI 15.98–18.11). Adjustment for age, parity, and BMI slightly lowered the hazard ratio, but the risk of T2DM was still 10-fold compared with women without GDM (HR 10.06, 95% CI 9.41–10.75) (Table 1). Need for insulin therapy during GDM independently increased the risk of T2DM compared with women with GDM and no insulin therapy (HR 3.81, 95% CI 3.57–4.07).

Table 1 shows that each year of age increased the risk of T2DM by 2–5%, each delivery by 2–8%, and each BMI unit by 6–12%, depending on the adjusted model. Only GDM, initiation of insulin therapy during pregnancy, and pre-pregnancy BMI were statistically significant risk factors for T2DM in each model.

In 2006–2008 the HR for T2DM was 15.01 (95% CI 13.83–16.30) while in 2018–2020 the HR was 17.50 (95% CI 10.89–28.12).

As BMI increased, the incidence of T2DM increased in both women with and without prior GDM but more in women with prior GDM (Figure 3). As BMI increased in overweight women with a history of GDM, the HR for developing T2DM decreased but absolute risk increased. When comparing women with prior GDM and those without prior GDM, the risk of developing T2DM in women with prior GDM was nine-fold for overweight women (BMI 25–29), six-fold for women with class 1 obesity (BMI 30–34), five-fold for women with class 2 obesity (BMI 35–39) and three-fold for women with class 3 obesity (BMI ≥ 40). The hazard ratio was highest in underweight women (Table 2).

Discussion

Our results demonstrate that gestational diabetes is the major risk factor for type 2 diabetes (HR 18.36) as shown in previous studies [1,2]. We showed that obesity and overweight as well as the need for insulin treatment to control glucose levels during pregnancy were the most important risk factors for future T2DM.

The risk for T2DM was 11 times higher in women with a history of GDM than in women without a history of GDM. This is in line with earlier reports [13,14].

This study provides new information on the significance of pre-pregnancy BMI for T2DM risk in women with and without gestational diabetes. As BMI increased, the hazard ratio for T2DM decreased and the absolute risk increased (Table 2). As BMI increased, the incidence of T2DM was significantly higher in women with than without a history of GDM (Figure 3).

Table 1

Hazard ratios (HR) for type 2 diabetes with 95% confidence intervals for women with prior gestational diabetes compared with women without prior gestational diabetes. Adjusted 1: Age and parity. Adjusted 2: Age, parity and body mass index (BMI). Adjusted 3: Age, parity and insulin treatment started during pregnancy. *p<0.0001.

	Maternal Age	Parity	Gestational diabetes	Insulin therapy	BMI
	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)
2006-2020					
Crude	1.08* (1.08-1.09)	1.19* (1.18-1.21)	18.49* (17.39-19.67)	18.66* (17.54-19.86)	1.149* (1.146-1.152)
Adjusted 1	1.04* (1.03-1.05)	1.04* (1.02-1.06)	17.01* (15.98-18.11)	-	-
Adjusted 2	1.04* (1.04-1.05)	1.02 (1.00-1.04)	10.06* (9.41-10.75)	-	1.101* (1.097-1.105)
Adjusted 3	1.03* (1.03-1.04)	1.04* (1.02-1.06)	13.08* (12.25-13.97)	3.81* (3.57-4.07)	-
2006-2008					
Crude	1.08* (1.07-1.09)	1.19* (1.16-1.21)	15.01* (13.83-16.30)	17.20* (15.64-18.92)	1.173* (1.068-1.178)
Adjusted 1	1.04* (1.03-1.05)	1.08* (1.05-1.11)	13.79* (12.69-14.99)	-	-
Adjusted 2	1.05* (1.04-1.05)	1.06* (1.03-1.08)	7.28* (6.63-7.98)	-	1.115* (1.109-1.121)
Adjusted 3	1.04* (1.03-1.05)	1.08* (1.06-1.11)	10.61* (9.69-11.61)	3.34* (3.01-3.70)	-
2018-2020					
Crude	1.08* (1.05-1.10)	1.31* (1.21-1.41)	17.50* (10.89-28.12)	21.19* (15.64-28.73)	1.089* (1.078-1.100)
Adjusted 1	1.03 (0.99-1.06)	1.03 (0.92-1.15)	16.04* (9.89-26.02)	-	-
Adjusted 2	1.03 (1.00-1.06)	1.02 (0.91-1.14)	11.80* (7.13-19.52)	-	1.064* (1.050-1.079)
Adjusted 3	1.02 (0.99-1.05)	1.03 (0.92-1.15)	11.12* (6.77-18.26)	8.19* (5.99-11.19)	-

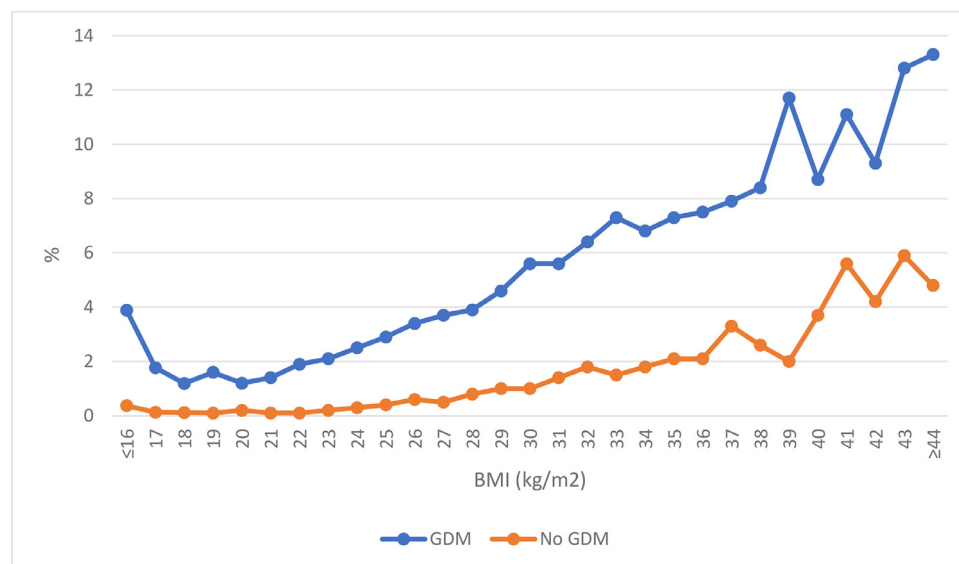


Fig. 3. The incidence of type 2 diabetes by pre-pregnancy body mass index (BMI) for women with and without a history of gestational diabetes (GDM).

This study focuses especially on the risk of T2DM caused by overweight and obesity, in addition to GDM. However, the results also show a relatively high incidence of T2DM in underweight women with prior GDM. A few studies have reported on type 2 diabetes in underweight people, especially in Asia [15]. Normal-weight women with a history of GDM have been found to display the most β -cell

dysfunction [16]. More research is needed on the ethnic and genetic background of these women, as well as weight changes during and after pregnancy.

Our observation that the cumulative incidence of T2DM begin to increase soon after delivery was remarkable (Figure 2). While Vounzoulaki et al. reported in their meta-analysis that the cumulative inci-

Table 2

Adjusted hazard ratios for developing type 2 diabetes (T2DM) according to body mass index (BMI) before pregnancy with 95 % confidence intervals for women with a history of gestational diabetes (GDM). Adjusted for maternal age and parity. *p<0.0001.

	Maternal age	Parity	Gestational diabetes	GDM – T2DM	No GDM – T2DM
BMI before pregnancy (kg/m ²)	HR (95% CI)	HR (95% CI)	HR (95% CI)	%	%
< 20 (n=62 197)	1.00 (0.97-1.03)	1.08 (0.94-1.25)	26.50* (18.87-37.21)	2.3	0.2
20–24.9 (n=233 168)	1.02 (1.01-1.04)	1.00 (0.95-1.05)	18.67* (16.32-21.35)	1.9	0.2
25–29.9 (n=98 721)	1.04* (1.03-1.05)	1.03 (0.99-1.06)	9.07* (8.07-10.20)	3.6	0.6
30–34.9 (n=37 440)	1.05* (1.04-1.06)	1.02 (0.99-1.06)	6.13* (5.37-6.98)	6.2	1.4
35–39.9 (n=13 175)	1.06* (1.04-1.07)	1.01 (0.96-1.06)	4.87* (4.02-5.91)	8.2	2.4
40–44.9 (n=4269)	1.05* (1.03-1.07)	0.95 (0.88-1.04)	3.49* (2.64-4.62)	11.2	4.7
≥ 45 (n=1477)	1.05 (1.02-1.09)	0.98 (0.88-1.08)	3.24* (2.08-5.05)	13.3	5.8
Unknown (n=11 954)	1.04 (1.00-1.07)	1.15 (1.03-1.28)	14.16* (9.72-20.63)	4.0	0.4

dence of T2DM seemed to increase steadily over time, the exact onset of T2DM remained open [2]. In a study performed shortly after delivery (3–16 months), compared with control subjects, women after GDM had a reduced disposition index, higher levels of plasma fetuin-A, and a lower insulin sensitivity index. A low insulin sensitivity index was also the major determinant of pathological glucose tolerance after GDM. The factors most strongly predictive of low insulin sensitivity were high plasma leptin, body mass index, triglycerides, and waist circumference. β -cell function is already impaired in women with recent GDM [17].

We showed that insulin treatment during pregnancy had an independent effect of increasing the risk of T2DM compared to women with diet-treated GDM (HR 3.81). The result is consistent with previous studies that have shown that the need for insulin increases the risk of future T2DM [4]. The need to start insulin treatment during pregnancy may indicate a more severe state of disease involving, in addition to excess weight and insulin resistance, pancreatic beta cell dysfunction and a more likely conversion of GDM to T2DM.

The strength of this study is the extensive anonymized database, which consisted of data from four different registers that used individuals' information. Based on the individual data, it was possible to determine the analyzes we performed retrospectively.

Our analyses account for different follow-up time between cases and controls. However, the mean follow-up was relatively short: seven years for the women with GDM and nine years for women without GDM. It has been shown that the prevalence of T2DM increases steadily over the years after GDM [2,3]. Further follow-up of these women would be needed to determine the long-term conversion of GDM to T2DM. It should be noted that even with such a short follow-up interval, the incidence of T2DM after GDM was significant. It is therefore important to target prevention at an early stage immediately after childbirth without forgetting the importance of finding ways to prevent GDM.

Another weakness of this study is the variable use of ICD10 codes in different hospitals. According to our understanding, the use of ICD-10 diagnoses is not completely uniform in Finland, and it is possible that the control population also includes women with a history of diet-treated GDM. Although 603 women who may have had diet-treated GDM were included in the group of women who did not have GDM, the total number of women in that group was so large ($n=366,084$) that it had no significant effect on the results. However, it may have slightly increased the incidence of T2DM among women without a history of GDM.

This study supports the Finnish Current Care guidelines, which recommend a 75 g OGTT 6–12 weeks after delivery for those who have had medical treated GDM and after one year for those who have had diet-treated GDM. After that, a 75 g OGTT is recommended every 1–3 years, depending on the T2DM risk factors [18]. According to our study, women who need insulin therapy for GDM have a three-fold risk of developing T2DM when compared to diet-treated GDM women, so OGTT 6–12 weeks after delivery and once a year after that could be recommended.

In studies with long follow-up, T2DM incidence after GDM of up to 70% has been reported [13]. There is also no clear evidence for any beneficial interventions to prevent diabetes in patients with prior history of gestational diabetes. Intensive lifestyle intervention or metformin treatment has been reported to reduce the incidence of T2DM by 35 and 40 percent over 10 years of follow-up [19]. However, commitment to the lifestyle intervention is weak among postpartum women [20]. Liraglutide and bariatric surgery are possible options but based on inadequate data. There remains a need for randomized, placebo-controlled studies to evaluate various pharmacologic treatments, with and without lifestyle interventions, to prevent type 2

diabetes mellitus in women with a history of gestational diabetes [21].

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Declaration of Competing Interest

We have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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