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More common, less harmful? Gestational diabetes and obesity before and after the introduction of universal screening in Germany: a before-after comparison of two population-based birth cohorts

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Research Article

Keywords: Gestational diabetes, obesity, pregnancy outcome, screening, socioeconomic factors, cohort studies, body mass index

Posted Date: November 25th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11627509/v1

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Additional Declarations: No competing interests reported.

Abstract

Background.

In 2012 revised German maternity guidelines replaced selective, urine-based case finding for gestational diabetes mellitus (GDM) with universal two-step screening, changing both case detection and the diagnosed population. We compared the prevalence, determinants and perinatal outcomes of GDM and maternal obesity across this change, asking whether a changed clinical impact of GDM reflects what the diagnosis denotes rather than outcomes.

Methods.

Before-after comparison of two population-based birth cohorts of the Survey of Neonates in Pomerania (SNiP), Germany: SNiP-I (2003–2008; n = 4542) and SNiP-II (2013–2017; n = 2726). Determinants of GDM and its associations with perinatal outcomes were estimated by confounder-adjusted regression. The primary estimand was the GDM-by-cohort interaction in the pooled cohorts (ratio of odds ratios, ROR, for dichotomous outcomes). Absolute event rates were reported by GDM and obesity status.

Results.

GDM prevalence rose from 4.9% to 7.3% and pre-pregnancy obesity from 9.6% to 13.1%. Obesity was the strongest determinant of GDM and strengthened over time (odds ratio 3.91 to 4.99); higher education and income were protective. GDM-associated odds ratios were lower in SNiP-II (neonatal hypoglycaemia ROR 0.41, 95% CI 0.22–0.76; inpatient admission ROR 0.52, 0.34–0.81), and macrosomia, large-for-gestational-age (LGA) and vaginal infection were no longer significant. Absolute rates showed two patterns: for hypoglycaemia, respiratory distress and inpatient admission, rates rose in pregnancies without GDM (hypoglycaemia 2.0% to 4.5%) while remaining unchanged or higher with GDM; for macrosomia and LGA, rates without GDM were stable while rates with GDM fell (macrosomia 8.0% to 4.0%). Among women with GDM, additional obesity was accompanied by higher rates of macrosomia, LGA and increased head circumference in both cohorts, and in SNiP-II also of caesarean delivery, respiratory distress and inpatient admission.

Conclusions.

Under universal screening GDM became more common and denoted a less selected group. The attenuation of GDM-associated odds ratios for metabolic and care-utilisation outcomes reflected rising event rates in pregnancies without GDM rather than better glycaemic management; only the decline in fetal overgrowth was confined to pregnancies with GDM. GDM remains a useful marker of hypoglycaemia risk, and GDM with obesity the main target for intensified care.

Background

Gestational diabetes mellitus (GDM) is one of the most common disorders of pregnancy and is defined as glucose intolerance first diagnosed during pregnancy [1]. It is associated with adverse maternal outcomes such as pre-eclampsia, preterm labour and caesarean delivery, and with adverse neonatal outcomes including respiratory distress, hypoglycaemia and admission to neonatal intensive care [2, 3, 4]. Maternal obesity increases the risk of GDM and is independently associated with pre-eclampsia, preterm birth and large-for-gestational-age (LGA) birth [5, 6, 7], and more than one third of women of reproductive age in Germany and worldwide are now overweight or obese [8, 9]. High pre-pregnancy body mass index (ppBMI) and excessive gestational weight gain (GWG) show partly overlapping effects on fetal growth and neonatal morbidity [10, 11].

Reported GDM prevalence in Germany rose more than fivefold between 2002 and 2021, from 1.5% to 8.5% [12, 13]. This increase is not attributable to metabolic change alone. Until 2011 the German maternity guidelines required only repeated urine glucose testing, with a 75-g oral glucose tolerance test (oGTT) reserved for women with glucosuria or clinical risk factors. From 2012 a two-step procedure became mandatory for all pregnant women, a 50-g glucose challenge followed by a confirmatory 75-g oGTT [14]. The revision therefore changed two things at once: how many women are tested, and which women end up carrying the diagnosis. Under selective, glucosuria-triggered testing the diagnosed group is enriched for women with pronounced hyperglycaemia. Under universal screening the same label is applied across the whole distribution of glucose tolerance. A revision of diagnostic criteria therefore alters the composition of the diagnosed group itself, a mechanism described for stage migration in oncology [15].

Using data from the first Survey of Neonates in Pomerania birth cohort (SNiP-I), we previously described the maternal and neonatal morbidity associated with GDM under the pre-2012 screening regime, and identified pre-pregnancy overweight and obesity, maternal age and socioeconomic position as its principal determinants [16]. Since then, maternal obesity and age at delivery have increased, and universal two-step screening has been in place since 2012 [12]. The second SNiP birth cohort (SNiP-II; 2013–2017), recruited in the same region, allows the two cohorts to be compared across this guideline change. We aimed to compare the prevalence and sociodemographic determinants of GDM and maternal obesity between the cohorts, to compare the maternal and neonatal outcomes associated with GDM, and to quantify the change in these associations in a pooled analysis.

Methods

Design, setting and participants

This is a before-after comparison of two independent, population-based birth cohorts. SNiP is a prospective birth cohort study conducted in the rural region of West Pomerania, north-eastern Germany (1962 km2; population density 70 inhabitants per km2), where obstetric and neonatal care is

concentrated at one university hospital and two community hospitals. SNiP-I was enrolled from 2003 to 2008 (n = 5801 mother-child dyads; 75% active participation) and SNiP-II from 2013 to 2017 (n = 3502 dyads; 54% active participation). Each participant contributed medical record data, personal interview data and socioeconomic variables; SNiP-II additionally collected fetal growth parameters, urine samples and maternal DNA. All women giving birth in one of the three hospitals and registered as permanent residents of the study area were eligible. Minimal data were collected for non-responders, women with insufficient German language skills, minors, and cases in which the newborn was given up for adoption. Full details of both cohorts are given in the design papers [17, 18]. Results for SNiP-I under the pre-2012 screening regime have been reported previously [16]. The present analysis population (4542 mother-child pairs, 225 of them with GDM) corresponds to the participants with complete covariate data in that report. Derivation of the analysis cohorts, with the number of and reasons for exclusions at each step, is shown in Fig. 1.

Definition of gestational diabetes and the 2012 guideline revision

GDM was diagnosed according to the German maternity policy guidelines in force at the time of each cohort [12, 14]. During SNiP-I recruitment (2003–2008) midstream urine samples of all pregnant women were tested for glucose, first after the 10th week of gestation (median) and then every four weeks; women with an abnormal result underwent a 75-g oGTT. During SNiP-II recruitment (2013–2017) all women were offered a 50-g glucose challenge test without prior fasting, and those with a one-hour value of 7.5 mmol/l (135 mg/dl) or above underwent a confirmatory 75-g oGTT. In both cohorts GDM was diagnosed if at least one 75-g oGTT threshold was exceeded: fasting 5.1 mmol/l (92 mg/dl), one hour 10.0 mmol/l (180 mg/dl), two hours 8.5 mmol/l (153 mg/dl). Overweight was defined as a ppBMI of 25.0-29.9 kg/m2 and obesity as a ppBMI of 30.0 kg/m2 or above. Definitions of macrosomia, neonatal hypoglycaemia, respiratory distress, LGA, small-for-gestational-age and the remaining outcomes are unchanged from our previous analysis [16]. Individual 75-g oGTT values and urine glucose results were not recorded in either cohort.

Statistical analysis

Continuous data are reported as medians with the 25th and 75th percentiles, categorical data as absolute numbers and percentages. Associations between potential maternal risk factors and GDM were analysed using logistic regression models adjusted for confounders. Associations of GDM and/or obesity with maternal and neonatal outcomes were analysed using linear, logistic and multinomial logistic regression models adjusted for confounders. The confounders used in each model are given in the table legends and were identical in both cohorts.

To quantify the change in these associations between cohorts, the ratio of the two cohort-specific effect estimates (ratio of odds ratios, ROR) was calculated, with its confidence interval derived from the standard errors of the cohort-specific estimates. Because the cohorts are independent samples, this is equivalent to the GDM-by-cohort interaction term of a pooled model, which was fitted for neonatal

hypoglycaemia as a check. An ROR below 1 indicates a weaker association in SNiP-II. Absolute event rates are reported alongside the odds ratios for pregnancies with and without GDM in each cohort, so that a change in the odds ratio can be attributed to the exposed group, the unexposed group, or both. Given the number of comparisons, inference is confined to these cohort-interaction analyses; comparisons within the descriptive and stratified tables are interpreted on the basis of effect estimates rather than statistical significance. Because the proportion of preterm births differed between cohorts, the rate of neonatal hypoglycaemia in pregnancies without GDM was additionally compared between cohorts within strata of gestational age (term, 37 weeks or more; preterm, below 37 weeks), and the SNiP-II rate was directly standardised to the gestational age distribution of SNiP-I. All analyses were performed using Stata 19.5 (StataCorp, College Station, TX, USA).

Declaration of Use of AI

During the preparation of this work the authors used Claude (Anthropic) in order to improve language and readability. After using this tool, the author(s) reviewed and edited the content as needed and take full responsibility for the content of the publication.

Results

Cohorts and prevalence

Of 5801 dyads enrolled in SNiP-I, complete data were available for 4542, of whom 225 had GDM (4.9%); ppBMI was available for 4010, among whom 196 had GDM (4.9%) and 386 had obesity (9.6%). Of 3502 dyads enrolled in SNiP-II, complete data including ppBMI were available for 2726, of whom 199 had GDM (7.3%) and 357 had obesity (13.1%) (Fig. 1). Median maternal age at delivery rose from 27 to 30 years in pregnancies without GDM and was 29 and 30 years in pregnancies with GDM. The proportion of preterm births below 37 weeks of gestation rose from 8.8% to 12.1% of the whole cohort, and from 8.7% to 12.1% among pregnancies without GDM. Caesarean delivery increased in both exposure groups, from 25.9% to 31.7% without GDM and from 40.2% to 43.7% with GDM (Table 1).

Table 1

Characteristics of the SNiP-I and SNiP-II cohorts, stratified by gestational diabetes mellitus. Continuous data are expressed as median (25th; 75th percentile) and categorical data as absolute numbers and percentages. Percentages for categorical variables are column percentages within the respective cohort and GDM stratum. BMI - body mass index; GDM - gestational diabetes mellitus; SNiP - Survey of Neonates in Pomerania.

Characteristic	SNiP-I		SNiP-II	
	No GDM	GDM	No GDM	GDM
N	4317 (95.1%)	225 (4.9%)	2527 (92.7%)	199 (7.3%)
Age of the mother (years)	27 (23; 31)	29 (25; 34)	30 (27; 33)	30 (27; 34)
Parity				
1st child	2023 (46.8%)	83 (37.1%)	1093 (43.6%)	81 (40.9%)
2nd child	1287 (29.8%)	73 (32.6%)	748 (29.8%)	53 (26.8%)
3rd child	565 (13.1%)	37 (16.5%)	381 (15.2%)	35 (17.7%)
4th child or more	444 (10.3%)	31 (13.8%)	284 (11.3%)	29 (14.6%)
Educational level				
< 10 years	569 (15.1%)	31 (15.8%)	211 (8.5%)	20 (10.3%)
10 years	1947 (51.8%)	105 (53.6%)	949 (38.3%)	84 (43.1%)
> 10 years	699 (18.6%)	36 (18.4%)	546 (22.0%)	40 (20.5%)
Higher degree	544 (14.5%)	24 (12.2%)	772 (31.2%)	51 (26.2%)
Equivalent income (EUR)	1060 (619; 1588)	1060 (505; 1375)	1371 (866; 2012)	1227 (650; 1750)
Smoking status				
Never	1446 (39.5%)	73 (39.5%)	1011 (41.3%)	62 (31.5%)
Former	452 (12.4%)	31 (16.8%)	1207 (49.3%)	108 (54.8%)
Quit during pregnancy	1058 (28.9%)	51 (27.6%)	232 (9.5%)	27 (13.7%)
Alcohol during pregnancy	935 (24.5%)	33 (17.0%)	151 (6.1%)	10 (5.2%)
Pre-pregnancy BMI (kg/m2)	22.5 (20.3; 25.3)	24.9 (22.5; 29.7)	22.9 (20.7; 26.3)	26.6 (23.2; 31.2)
< 19	419 (11.0%)	9 (4.6%)	202 (8.0%)	3 (1.5%)
19-24.99	2386 (62.6%)	91 (46.4%)	1521 (60.2%)	75 (37.7%)
25-29.99	671 (17.6%)	48 (24.5%)	513 (20.3%)	55 (27.6%)

Characteristic	SNiP-I		SNiP-II	
	No GDM	GDM	No GDM	GDM
30 or above	338 (8.9%)	48 (24.5%)	291 (11.5%)	66 (33.2%)
Gestational weight gain (kg)	15.0 (11.0; 19.0)	13.0 (8.0; 18.0)	16.0 (12.0; 20.0)	13.0 (9.2; 19.0)
Number of antenatal examinations				
9 or fewer	576 (13.8%)	24 (11.2%)	421 (17.6%)	32 (17.0%)
10–12	1424 (34.2%)	57 (26.5%)	1024 (42.9%)	76 (40.4%)
More than 12	2159 (51.9%)	134 (62.3%)	943 (39.5%)	80 (42.6%)
Positive vaginal smear	543 (14.8%)	51 (24.8%)	421 (17.5%)	35 (18.4%)
Gestational age (weeks)	39 (38; 40)	39 (37; 39)	39 (38; 40)	39 (38; 40)
< 32 weeks	73 (1.7%)	2 (0.9%)	52 (2.1%)	8 (4.0%)
32–36 weeks	301 (7.0%)	25 (11.1%)	251 (10.0%)	20 (10.1%)
37–41 weeks	3895 (90.1%)	198 (88.0%)	2191 (87.3%)	171 (85.9%)
> 41 weeks	54 (1.2%)	0 (0.0%)	16 (0.6%)	0 (0.0%)
Spontaneous delivery (incl. operative spontaneous)	3194 (74.1%)	134 (59.8%)	1724 (68.3%)	112 (56.3%)
Male sex	2276 (52.7%)	105 (46.7%)	1242 (49.3%)	116 (58.6%)
Birth weight (g)	3410 (3056; 3740)	3480 (3100; 3770)	3390 (2980; 3720)	3360 (3040; 3730)
Appropriate for gestational age	3457 (80.0%)	169 (75.1%)	1978 (78.5%)	157 (78.9%)
Small for gestational age	409 (9.5%)	11 (4.9%)	272 (10.8%)	13 (6.5%)
Large for gestational age	457 (10.6%)	45 (20.0%)	270 (10.7%)	29 (14.6%)
Macrosomia	147 (3.4%)	18 (8.0%)	81 (3.2%)	8 (4.0%)
Birth length (cm)	51 (49; 53)	51 (49; 52)	51 (49; 52)	51 (49; 52)
Normal	3543 (82.0%)	192 (85.3%)	2014 (80.1%)	168 (84.4%)
Small	526 (12.2%)	22 (9.8%)	347 (13.8%)	21 (10.6%)
Large	252 (5.8%)	11 (4.9%)	154 (6.1%)	10 (5.0%)
Head circumference (cm)	35 (34; 36)	35 (34; 36)	35 (34; 36)	35 (34; 36)
Normal	3423 (79.5%)	177 (79.0%)	2041 (81.4%)	159 (80.3%)

Characteristic	SNiP-I		SNiP-II	
	No GDM	GDM	No GDM	GDM
Small	436 (10.1%)	15 (6.7%)	279 (11.1%)	20 (10.1%)
Large	444 (10.3%)	32 (14.3%)	186 (7.4%)	19 (9.6%)
Inpatient admission	805 (18.7%)	104 (46.4%)	596 (23.8%)	85 (42.7%)
Respiratory distress	213 (4.9%)	15 (6.7%)	227 (9.0%)	25 (12.6%)
Neonatal hypoglycaemia	86 (2.0%)	47 (20.9%)	114 (4.5%)	36 (18.1%)

Maternal determinants of GDM

The determinants of GDM were similar in direction and magnitude in both cohorts (Table 2). Higher maternal age (odds ratio [OR] 1.06 per year in SNiP-I, 1.05 in SNiP-II) and higher ppBMI (OR 1.10 and 1.12 per kg/m2) were associated with increased odds of GDM. The highest educational level (OR 0.53 and 0.57) and higher equivalent household income (OR 0.62 and 0.71 per 1000 EUR) were associated with reduced odds. The association with obesity was the strongest of all determinants and was more pronounced in the later cohort (OR 3.91 in SNiP-I versus 4.99 in SNiP-II, relative to normal weight). The ppBMI difference between women with and without GDM widened accordingly, from 24.9 versus 22.5 kg/m2 in SNiP-I to 26.6 versus 22.9 kg/m2 in SNiP-II (Table 1).

Table 2
Associations between potential risk factors and gestational diabetes using logistic regression.

Potential risk factor	SNiP-I	SNiP-II
	OR (95% CI)	OR (95% CI)
Maternal age	1.06 (1.03; 1.08)	1.05 (1.02; 1.08)
Educational level	Reference	Reference
- Low	0.78 (0.51; 1.20)	0.85 (0.51; 1.42)
- Middle	0.73 (0.44; 1.21)	0.72 (0.41; 1.26)
- Mid-high	0.53 (0.29; 0.94)	0.57 (0.33; 0.99)
- High		
Income (per 1000€)	0.62 (0.46; 0.83)	0.71 (0.56; 0.89)
Smoking during pregnancy	0.80 (0.51; 1.24)	1.36 (0.85; 2.17)
Alcohol use during pregnancy	0.61 (0.41; 0.90)	0.83 (0.43; 1.61)
Pre-pregnancy BMI (in kg/m^2)	1.10 (1.08; 1.13)	1.12 (1.10; 1.15)
- Underweight (< 19)	0.71 (0.35; 1.44)	0.32 (0.10; 1.01)
- Normal weight (19–24.99)	Reference	Reference
- Overweight (25–29.99)	1.87 (1.28; 2.74)	2.17 (1.49; 3.15)
- Obese (> = 30)	3.91 (2.64; 5.79)	4.99 (3.45; 7.21)
Gestational weight gain	0.99 (0.97; 1.01)	0.98 (0.95; 1.01)
Parity		
- 1st child	Reference	Reference
- 2nd child	1.11 (0.78; 1.58)	0.80 (0.55; 1.16)
- 3rd child	1.27 (0.81; 1.99)	1.00 (0.65; 1.55)
- 4th+ child	0.90 (0.52; 1.58)	0.91 (0.55; 1.50)

CI – confidence interval; BMI – body mass index; OR – odds ratio; SNiP – Survey of Neonates in Pomerania; results are derived from logistic regression models adjusted for confounding: Maternal age not adjusted. Educational level and equivalent income adjusted for maternal age. Smoking, alcohol consumption, and parity adjusted for maternal age and educational level. Pre-pregnancy BMI adjusted for maternal age, educational level and smoking. Gestational weight gain adjusted for maternal age, educational level, smoking and pre-pregnancy BMI.

Association of GDM with perinatal outcomes and its change between cohorts

In both cohorts GDM was associated with neonatal hypoglycaemia, inpatient neonatal admission, caesarean section and shorter gestation (Table 3). The odds ratios were consistently lower in SNiP-II: neonatal hypoglycaemia fell from 11.8 to 4.81, inpatient admission from 4.14 to 2.16 and caesarean section from 1.82 to 1.43, while the associations with macrosomia, LGA and vaginal infection were no longer statistically significant. In the pooled interaction model the attenuation was statistically significant for neonatal hypoglycaemia (ROR 0.41, 95% CI 0.22–0.76, p = 0.005).

Table 3
Association of gestational diabetes with perinatal outcomes in both cohorts: absolute event rates, adjusted effect estimates and the change in association between cohorts.

Outcome	SNiP-I			SNiP-II			
	No GDM (%)	GDM (%)	Effect estimate (95% CI)	No GDM (%)	GDM (%)	Effect estimate (95% CI)	Change between cohorts (95% CI)
Vaginal infections	14.8	24.8	2.18 (1.52; 3.13)	17.5	18.4	1.09 (0.73; 1.63)	0.50 (0.29; 0.86)
Macrosomia	3.4	8.0	2.30 (1.32; 4.01)	3.2	4.0	1.10 (0.51; 2.35)	0.48 (0.19; 1.23)
Neonatal hypoglycaemia	2.0	20.9	11.8 (7.56; 18.5)	4.5	18.1	4.81 (3.11; 7.44)	0.41 (0.22; 0.76)
Inpatient admission	18.7	46.4	4.14 (3.05; 5.62)	23.8	42.7	2.16 (1.58; 2.95)	0.52 (0.34; 0.81)
Respiratory distress	4.9	6.7	1.61 (0.91; 2.81)	9.0	12.6	1.38 (0.87; 2.18)	0.86 (0.41; 1.77)
Caesarean section	25.9	40.2	1.82 (1.34; 2.46)	31.7	43.7	1.43 (1.05; 1.95)	0.79 (0.51; 1.21)
Large for gestational age	10.6	20.0	1.67 (1.14; 2.45)	10.7	14.6	1.07 (0.69; 1.67)	0.64 (0.36; 1.15)
Small for gestational age	9.5	4.9	0.53 (0.25; 1.09)	10.8	6.5	0.52 (0.27; 0.98)	0.98 (0.37; 2.61)
Gestational age (weeks)	-	-	-0.79 (-1.10; -0.48)	-	-	-0.44 (-0.80; -0.07)	+ 0.35 (− 0.13; +0.83)†

Percentages are crude event rates. Effect estimates are adjusted estimates from the cohort-specific models. For dichotomous outcomes, the comparison measure is the ratio of effect estimates (SNiP-II/SNiP-I); for large- and small-for-gestational-age, the underlying estimates are relative risk ratios. †For gestational age, the interaction measure is the difference in linear regression coefficients (SNiP-II minus SNiP-I), not a ratio. The final column compares the cohorts: for all outcomes except gestational age it is the ratio of the two cohort-specific estimates (SNiP-II/SNiP-I), a value below 1 indicating a weaker association in SNiP-II. Confidence intervals for the interaction were reconstructed from the cohort-specific 95% confidence intervals assuming independent cohorts; CI - confidence interval; GDM - gestational diabetes mellitus; SNiP - Survey of Neonates in Pomerania.

The absolute event rates in Table 3 show that these outcomes fall into two groups. For neonatal hypoglycaemia, inpatient admission and respiratory distress, the rate among infants of mothers without GDM rose markedly between cohorts: hypoglycaemia from 2.0% to 4.5%, respiratory distress from 4.9% to 9.0% and inpatient admission from 18.7% to 23.8%. Among infants of mothers with GDM the same rates were essentially unchanged for hypoglycaemia (20.9% to 18.1%) and inpatient admission (46.4% to 42.7%), and higher for respiratory distress (6.7% to 12.6%). The fall in the odds ratios for these outcomes therefore arose predominantly in the comparison group.

Because the proportion of preterm births rose between the cohorts, the increase in neonatal hypoglycaemia among pregnancies without GDM was examined within strata of gestational age. The rate increased in both strata: from 1.70% (67/3942) to 3.35% (74/2207) among term infants (risk ratio 1.97, 95% CI 1.42 to 2.73) and from 5.08% (19/374) to 13.20% (40/303) among preterm infants (risk ratio 2.60, 95% CI 1.54 to 4.39). Standardising the SNiP-II rate to the gestational age distribution of SNiP-I gave 4.21% against the observed 4.54%, so the change in gestational age composition accounted for 0.34 of the 2.55 percentage-point increase, or 13%.

For macrosomia and LGA the pattern was the reverse. Rates among infants of mothers without GDM were stable (macrosomia 3.4% to 3.2%; LGA 10.6% to 10.7%), whereas rates among infants of mothers with GDM fell substantially (macrosomia 8.0% to 4.0%; LGA 20.0% to 14.6%). At the level of the whole cohort, hypoglycaemia (2.9% to 5.5%), respiratory distress (5.0% to 9.2%) and inpatient admission (20.0% to 25.0%) were more frequent in SNiP-II, whereas macrosomia (3.6% to 3.3%) and LGA (11.0% to 11.0%) were unchanged.

GDM and obesity: four-group stratification

Stratification by GDM and obesity defined four groups in each cohort (Table 4). Women with neither condition comprised 86.7% of SNiP-I and 82.0% of SNiP-II. Lower socioeconomic position clustered with obesity rather than with GDM in SNiP-I, whereas in SNiP-II it extended to GDM as well. Median equivalent income illustrates this: in SNiP-I it was almost identical in women with GDM alone and in women with neither condition (1062 versus 1061 EUR) but lower in both groups with obesity (795 and 750 EUR), whereas in SNiP-II all three risk groups fell below the reference group (1188, 1227 and 1207 versus 1371 EUR).

Table 4
Characteristics of subgroups stratified by gestational diabetes and obesity (BMI > 30 kg/m2)

SNiP-I	GDM - BMI -	GDM - BMI +	GDM + BMI -	GDM + BMI +
N	3476 (86.7%)	338 (8.4%)	148 (3.7%)	48 (1.2%)
Age of the mother	27 (24; 31)	27 (23; 31)	29 (25; 33)	27 (24; 32)
Parity				
- 1st child	1655 (47.7%)	132 (39.2%)	55 (37.4%)	19 (39.6%)
- 2nd child	1052 (30.3%)	113 (33.5%)	54 (36.7%)	11 (22.9%)
- 3rd child	448 (12.9%)	44 (13.1%)	22 (15.0%)	13 (27.1%)
- 4th+ child	318 (9.2%)	48 (14.2%)	16 (10.9%)	5 (10.4%)
Educational level				
- < 10 years	472 (14.0%)	77 (23.5%)	17 (11.6%)	12 (25.5%)
- = 10 years	1722 (51.1%)	194 (59.3%)	77 (52.7%)	27 (57.4%)
- > 10 years	659 (19.5%)	34 (10.4%)	32 (21.9%)	4 (8.5%)
- Higher degree	518 (15.4%)	22 (6.7%)	20 (13.7%)	4 (8.5%)
Income (€)	1061 (619; 1588)	795 (505; 1326)	1062 (619; 1503)	750 (361; 1061)
Smoking status				
- Never	1312 (40.0%)	109 (34.5%)	55 (40.1%)	17 (37.0%)
- Former	410 (12.5%)	39 (12.3%)	22 (16.1%)	9 (19.6%)
- quit during pregnancy	952 (29.0%)	93 (29.4%)	38 (27.7%)	13 (28.3%)
- Current	610 (18.6%)	75 (23.7%)	22 (16.1%)	7 (15.2%)
Alcohol during pregnancy	865 (25.3%)	55 (16.5%)	26 (17.9%)	7 (14.9%)
BMI before pregnancy (kg/m2)	22.0 (20.1; 24.3)	33.2 (31.2; 35.9)	23.5 (21.4; 25.9)	35.4 (32.1; 39.1)
BMI before pregnancy (kg/m2)				
- < 19	419 (12.1%)	0 (0.0%)	9 (6.1%)	0 (0.0%)
- 19–24.99	2386 (68.6%)	0 (0.0%)	91 (61.5%)	0 (0.0%)
- 25–29.99	671 (19.3%)	0 (0.0%)	48 (32.4%)	0 (0.0%)

Data are expressed as median, 25th, and 75th percentile for continuous data or as absolute numbers and percentages for categorical data

SNiP-I	GDM - BMI -	GDM - BMI +	GDM + BMI -	GDM + BMI +
- >= 30	0 (0.0%)	338 (100.0%)	0 (0.0%)	48 (100.0%)
Weight change in pregnancy (kg)	15.0 (12.0; 19.0)	10.0 (5.0; 15.0)	14.0 (10.0; 18.0)	7.5 (0.0; 16.0)
Number of gyn. examinations				
- <= 9	448 (13.3%)	31 (9.7%)	17 (11.9%)	6 (13.0%)
- 10–12	1158 (34.5%)	102 (32.1%)	38 (26.6%)	10 (21.7%)
- > 12	1754 (52.2%)	185 (58.2%)	88 (61.5%)	30 (65.2%)
Positive vaginal smear	444 (15.1%)	32 (11.4%)	34 (24.8%)	11 (25.0%)
Gestational age (weeks)	39 (38; 40)	40 (38; 40)	38 (37; 39)	39 (38; 40)
Gestational age (full weeks)				
- < 32	53 (1.5%)	5 (1.5%)	2 (1.4%)	0 (0.0%)
- 32–36	247 (7.1%)	14 (4.1%)	21 (14.2%)	1 (2.1%)
- 37–41	3138 (90.3%)	310 (91.7%)	125 (84.5%)	47 (97.9%)
- > 41	38 (1.1%)	9 (2.7%)	0 (0.0%)	0 (0.0%)
Birth mode				
(incl. operative spontaneous)	2597 (74.9%)	230 (68.0%)	84 (57.1%)	30 (62.5%)
Sex				
- male	1839 (53.0%)	173 (51.2%)	70 (47.3%)	22 (45.8%)
- female	1632 (47.0%)	165 (48.8%)	78 (52.7%)	26 (54.2%)
Birth weight (g)	3400 (3060; 3730)	3520 (3210; 3840)	3420 (3035; 3725)	3650 (3355; 3960)
Birth weight				
- Normal	2792 (80.3%)	255 (75.4%)	116 (78.4%)	29 (60.4%)
- Small	325 (9.3%)	32 (9.5%)	6 (4.1%)	4 (8.3%)
- Large	359 (10.3%)	51 (15.1%)	26 (17.6%)	15 (31.2%)
Birth height (cm)	51 (49; 53)	51 (50; 53)	51 (49; 52)	51 (50; 52)

Data are expressed as median, 25th, and 75th percentile for continuous data or as absolute numbers and percentages for categorical data

SNiP-I	GDM - BMI -	GDM - BMI +	GDM + BMI -	GDM + BMI +
Birth height				
- Normal	2865 (82.4%)	274 (81.1%)	124 (83.8%)	45 (93.8%)
- Small	416 (12.0%)	37 (10.9%)	17 (11.5%)	2 (4.2%)
- Large	195 (5.6%)	27 (8.0%)	7 (4.7%)	1 (2.1%)
Macrosomia	114 (3.3%)	16 (4.7%)	12 (8.1%)	5 (10.4%)
Head circumference (cm)	35 (34; 36)	35 (34; 36)	35 (34; 36)	36 (35; 37)
Head circumference				
- Normal	2773 (80.1%)	248 (73.6%)	119 (80.4%)	34 (72.3%)
- Small	344 (9.9%)	33 (9.8%)	11 (7.4%)	1 (2.1%)
- Large	345 (10.0%)	56 (16.6%)	18 (12.2%)	12 (25.5%)
Inpatient admission	611 (17.6%)	62 (18.3%)	66 (44.9%)	24 (50.0%)
Respiratory distress	169 (4.9%)	14 (4.1%)	9 (6.1%)	5 (10.4%)
Hypoglycaemia	67 (1.9%)	5 (1.5%)	27 (18.2%)	11 (22.9%)
Data are expressed as median, 25th, and 75th percentile for continuous data or as absolute numbers and percentages for categorical data				

Pre-pregnancy obesity in the absence of GDM was accompanied by higher birth weight and more frequent LGA and caesarean delivery in both cohorts. In SNiP-I median birth weight was 3520 g versus 3400 g, LGA 15.1% versus 10.3% and caesarean delivery 32.0% versus 25.1%; in SNiP-II, LGA was 15.2% versus 10.1% and caesarean delivery 39.5% versus 30.7%. Obesity alone was not accompanied by higher rates of neonatal hypoglycaemia, preterm birth or inpatient admission. Among women with GDM, additional obesity was accompanied by higher rates of macrosomia, LGA and increased head circumference in both cohorts, and in SNiP-II also by higher rates of caesarean delivery (57.6% versus 36.8%), respiratory distress (21.2% versus 8.3%) and inpatient admission (51.5% versus 38.3%). The combination of GDM and obesity thus carried the highest event rates in both cohorts.

Exploratory analyses of effect modification

In exploratory analyses of effect modification (Table 5), GWG modified the association between GDM and inpatient neonatal admission in both cohorts, more strongly in SNiP-II (Fig. 2A, B). In SNiP-I ppBMI modified the association between GDM and neonatal hypoglycaemia and, less clearly, respiratory distress (Fig. 2C, D); neither was apparent in SNiP-II. Among women without GDM, higher GWG was associated with macrosomia within each ppBMI stratum in both cohorts (Fig. 3A, B).

Table 5
Interactions of pre-pregnancy BMI (ppBMI) / gestational weight gain (GWG) during pregnancy with gestational diabetes on pregnancy outcomes

	SNiP-I		SNiP-II	
	ppBMI	GWG	ppBMI	GWG
	p	p	p	p
Vaginal infections	0.213	0.327	0.207	0.404
Macrosomia	0.496	0.597	0.846	0.571
Hypoglycaemia	0.045	0.358	0.777	0.202
Inpatient admission	0.804	0.092	0.667	0.033
Respiratory distress	0.074	0.903	0.136	0.900
Sectio vs. spontaneous birth	0.152	0.700	0.776	0.077
Gestational age	0.346	0.052	0.856	0.682

Results are expressed as p for interaction derived from logistic and linear regression models adjusted for maternal age, alcohol consumption, and maternal education. GWG further adjusted for ppBMI

Discussion

In two population-based birth cohorts spanning the 2012 introduction of universal GDM screening in Germany, GDM prevalence rose from 4.9% to 7.3%, pre-pregnancy obesity from 9.6% to 13.1%, and the odds ratios linking GDM to classical perinatal outcomes fell. Separating absolute event rates by exposure group shows that the attenuation has two different origins. For neonatal hypoglycaemia, respiratory distress and inpatient admission it arose in the comparison group, whose event rates rose substantially. Rates among infants of mothers with GDM were unchanged for hypoglycaemia and inpatient admission and rose in parallel for respiratory distress. For macrosomia and LGA the comparison group was stable and the decline occurred among pregnancies with GDM. The conclusion is therefore not that GDM became less harmful. It is that universal screening changed what the diagnosis denotes, with fetal overgrowth as the one domain in which an improvement is confined to the exposed group.

Rising prevalence: secular trend and case detection

The magnitude of the prevalence increase matches German, Danish and Swedish national data reporting annual increases of approximately 7% over comparable periods [13, 19, 20, 21], and the parallel rise in pre-pregnancy obesity mirrors European registry trends [22, 23]. Obesity was the strongest determinant of GDM in both cohorts and strengthened over time, in line with population-based data from Finland and Spain [24, 25]. Both a true increase in metabolic risk and the change from selective to universal case

finding contribute, and with these data the two cannot be separated: SNiP-II contains no measure of glycaemic severity from which a pre-2012-detectable subgroup could be reconstructed. We therefore make no claim about their relative size and do not treat the prevalence increase itself as evidence about screening performance.

What the attenuation of the odds ratios does and does not show

The GDM-associated odds ratio for neonatal hypoglycaemia fell by more than half, and the pooled model confirms that this change is unlikely to be due to chance (ROR 0.41, 95% CI 0.22–0.76). The absolute rates locate the change in the comparison group: hypoglycaemia among infants of mothers without GDM more than doubled, from 2.0% to 4.5%, whereas the rate among infants of mothers with GDM fell only from 20.9% to 18.1%. Two co-occurring changes contribute. Preterm birth became more frequent in the comparison group, rising from 8.7% to 12.1%, and preterm infants are at higher risk of hypoglycaemia. The gestational age-stratified analysis shows that this accounts for little of the increase. Hypoglycaemia almost doubled among term infants alone, from 1.70% to 3.35%, and standardising the later rate to the earlier gestational age distribution left 87% of the rise unexplained by composition. The increase is therefore not a consequence of more preterm births and is most plausibly explained by more frequent glucose monitoring of infants without a maternal GDM diagnosis. The diagnostic threshold, a plasma glucose below 2.5 mmol/l within the first 24 h, was unchanged between the periods, and the standard operating procedures defining it were already established during SNiP-I. What differed was the reach of screening: during SNiP-I it was routine policy to screen all infants of mothers with GDM [16], while infants of mothers without that diagnosis were tested on clinical indication. A broader testing indication lowers the threshold for testing, not for diagnosis. Respiratory distress almost doubled in the comparison group, from 4.9% to 9.0%, and rose in parallel among infants of mothers with GDM, from 6.7% to 12.6%. Its ascertainment likewise depends on clinical attention. Together with the rise in caesarean delivery in both exposure groups, this indicates that obstetric and neonatal practice changed for the whole population, not only for pregnancies complicated by GDM.

What the data show is narrower and clinically useful. At population level the absolute burden of hypoglycaemia, respiratory distress and inpatient admission did not fall. Under universal screening GDM also remains a strong marker of hypoglycaemia risk, with an absolute rate of 18.1% against a background of 4.5%. The demonstrable benefit of universal screening is thus the identification of a larger group of newborns warranting structured glucose monitoring, not better outcomes in pregnancies already known to be affected.

Fetal overgrowth: the one domain with a confined improvement

Macrosomia and LGA behaved differently. Rates in the comparison group were stable across cohorts. The decline among pregnancies with GDM - macrosomia from 8.0% to 4.0%, LGA from 20.0% to 14.6% - therefore cannot be attributed to a shift in background risk or in ascertainment, since both would also

have affected the comparison group. Two explanations remain and our data cannot distinguish them: the inclusion of women with milder glucose intolerance under universal screening and improved glycaemic management of fetal growth. Both are compatible with the Swedish national registry, which reported falling LGA rates and birth-weight z-scores in GDM-affected pregnancies between 1998 and 2012 [26], and with French analyses showing a narrowing gap between treated GDM and unaffected pregnancies while residual risks persist [27, 28]. That the pattern is restricted to fetal growth is itself informative, since growth is the outcome most directly responsive to glycaemic control whereas hypoglycaemia additionally depends on detection. The persistence of elevated hypoglycaemia and inpatient admission in SNiP-II indicates that intrauterine metabolic programming, as described in HAPO and PEACHES, is not fully reversed by glycaemic control alone [5, 29].

GDM with obesity as the residual high-risk phenotype

In both cohorts the combination of GDM and obesity carried the highest rates of caesarean delivery, macrosomia, inpatient admission, hypoglycaemia and respiratory adaptation disorders, consistent with the HAPO subanalysis [30] and with Swedish, Finnish and Portuguese data and a recent systematic review reporting independent and additive effects [31, 24, 32, 33]. Isolated maternal obesity was associated with larger infants and more caesarean deliveries but not with a substantial excess of metabolic or respiratory morbidity, unlike the PEACHES finding that obese women without a GDM diagnosis frequently show subclinical dysglycaemia conveying risk to the offspring [11, 29]. One explanation is that universal screening reclassifies part of that group as GDM-positive, concentrating risk in the GDM-plus-obesity stratum - again a consequence of the changed case definition rather than a biological difference. For practice, women with both conditions are those in whom intensified glycaemic management, fetal surveillance and delivery planning are most clearly warranted.

Socioeconomic gradient and gestational weight gain

Higher education and income were protective against GDM in both cohorts, in line with the Generation R Study, Finnish register data and a Mendelian randomisation analysis indicating that much of the effect of maternal education is mediated by modifiable factors such as obesity and smoking [34, 35, 36]. In SNiP-I the gradient was confined largely to obesity, whereas in SNiP-II lower income was present across all three risk groups, suggesting a broadening rather than a narrowing of socioeconomic differentiation. That it persisted through the introduction of universal screening is the clearest preventive message of the analysis: better case detection does not reach the structural determinants of metabolic risk in pregnancy, and pre-conception weight management needs to be directed at socioeconomically disadvantaged women [16]. In exploratory analyses, excessive GWG remained associated with inpatient neonatal admission among women with GDM, and with macrosomia but not metabolic morbidity among women without GDM, broadly consistent with existing literature [37, 38]. Given the number of interaction terms examined and the absence of multiplicity correction, these results are hypotheses rather than findings.

Strengths and limitations

The strengths are two large population-based cohorts from the same region with consistent data collection, more than 7000 mother-child pairs in total, and detailed sociodemographic, clinical and neonatal data allowing multivariable adjustment and stratification by both GDM and obesity. Reporting absolute event rates in both exposure groups alongside the relative measures, and testing the change in association formally, allows the location of a change to be identified rather than inferred.

The principal limitation is structural. GDM in SNiP-I and SNiP-II are not the same clinical entity, because the route to diagnosis differed while the confirmatory thresholds did not. Two shifts follow. The exposed group in SNiP-II includes women with milder glucose intolerance who would not have been tested before 2012. The unexposed group in SNiP-I contains an unknown number of women with undetected GDM, who were reclassified as exposed in SNiP-II. Both shifts attenuate relative measures of association in SNiP-II, independently of any change in care. We can therefore characterise the direction of this bias but not its magnitude. No severity-matched subgroup can be constructed, because individual oGTT values and urine glucose results were not recorded in either cohort, and model-based partitioning of the prevalence rise into a secular and a detection component would rest on untestable assumptions about the counterfactual detection rate. Reporting absolute event rates in both exposure groups mitigates this limitation, since those rates are unaffected by relabelling of the exposure, but does not remove it.

A second limitation concerns recruitment. Active participation fell from 75% in SNiP-I to 54% in SNiP-II, and the cohorts differ markedly in composition: among women without GDM the proportion holding a higher degree rose from 14.5% to 31.2%. Part of this shift is expected, because educational attainment rose generally over the decade and the women in SNiP-II were on average three years older and therefore more likely to have completed tertiary education. Differential participation may nevertheless contribute, and since socioeconomic position is associated with both GDM and perinatal outcome, a change in who was recruited cannot be separated from a change in the underlying population. ppBMI was available for only 4010 of 4542 SNiP-I participants.

Further limitations apply. Treatment of GDM was not recorded, so the analysis cannot address whether management changed. Ascertainment of hypoglycaemia and respiratory distress depends on testing and admission thresholds that were not held constant by design, and the contributions of the higher preterm proportion and of more frequent testing to the rise in the comparison group cannot be separated here. Residual confounding by unmeasured factors such as physical activity and diet cannot be excluded. Finally, the rural, predominantly white population of West Pomerania limits generalisability to urban and ethnically diverse populations, in which ethnicity is a well-documented determinant of GDM risk [39, 40].

Conclusions

Between 2003 and 2017 the population-based SNiP study documents a substantial rise in GDM prevalence and in maternal pre-pregnancy obesity, together with a marked attenuation of the odds ratios linking GDM to perinatal outcomes. Separating the exposed from the unexposed group shows what this

attenuation reflects. It arises mainly from a rising event rate in pregnancies without GDM, together with the inclusion of less severely affected women under universal screening, rather than from improved outcomes among pregnancies with GDM. Only for fetal overgrowth is the improvement confined to the exposed group. For clinical practice, GDM under universal screening remains a strong marker of neonatal hypoglycaemia risk and therefore an indication for structured neonatal glucose monitoring. The persistent socioeconomic gradient and the combination of GDM and obesity remain the principal targets for prevention and for intensified antenatal care. More generally, the analysis illustrates that a change in diagnostic criteria alters the risk associated with a diagnosis for reasons that have nothing to do with the quality of care, and that before-after evaluations of screening policy should report absolute rates in both exposure groups.

Abbreviations

BMI - body mass index

CI - confidence interval

GDM - gestational diabetes mellitus

GWG - gestational weight gain

LGA - large for gestational age

oGTT - oral glucose tolerance test

OR - odds ratio

ppBMI - pre-pregnancy body mass index

ROR - ratio of odds ratios

RRR - relative risk ratio

SES - socioeconomic status

SGA - small for gestational age

SNiP - Survey of Neonates in Pomerania

Declarations

Ethics approval and consent to participate

The design and content of the SNiP-I and SNiP-II baseline and follow-up studies were reviewed and approved by the Ethics Committee of the University Medicine Greifswald (SNiP-I: III UV 20/00 and BB

020/16; SNiP-II: BB 133/12 and BB 133/12a). All participating mothers gave written informed consent before enrolment, as described in both design papers [17, 18]. The study was conducted in accordance with the Declaration of Helsinki.

Consent for publication

Not applicable. The manuscript contains no data relating to an identifiable individual.

Availability of data and materials

The datasets generated and analysed during the current study are not publicly available because they contain individual-level perinatal and socioeconomic data from a defined geographical region, which precludes public deposition. The data are held in the SNiP research data repository of the Research Cooperation Community Medicine, University Medicine Greifswald, where internal and external researchers can register and apply for access free of charge. Data and biomaterials are additionally available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

Funding

This work is part of the Research Cooperation Community Medicine (RCC) of the University of Greifswald, Germany, funded by the Federal Ministry of Education and Research (grant no. ZZ 96030). SNiP was supported by (a) the German Federal Ministry for Education and Research (NBL3 programme, grant 01 ZZ 0103), (b) the Excellence Support Programmes of the Federal State of Mecklenburg-Vorpommern (UG 07 034), and (c) the Epidemiological Study on Childhood Cancer and Malformations in the Vicinity of Nuclear Power Plants (StSch 4493). None of the funders had any role in the design of the study, in the collection, analysis or interpretation of the data, in the writing of the manuscript, or in the decision to submit it for publication.

Authors’ contributions

JB and JG conceived the analysis together with AL and MH and contributed equally to this work. HA, CK and AL carried out the investigation and data acquisition. JG, HA, MG and DES curated the data. TI performed the formal statistical analysis. JB, JG and MH developed the methodology. JB and TI produced the visualisations. AL and MH administered the project and MH supervised it. JB wrote the

original draft. JB, JG, DES and MH reviewed and edited the manuscript. All authors read and approved the final manuscript.

Acknowledgements

Not applicable.

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Figures

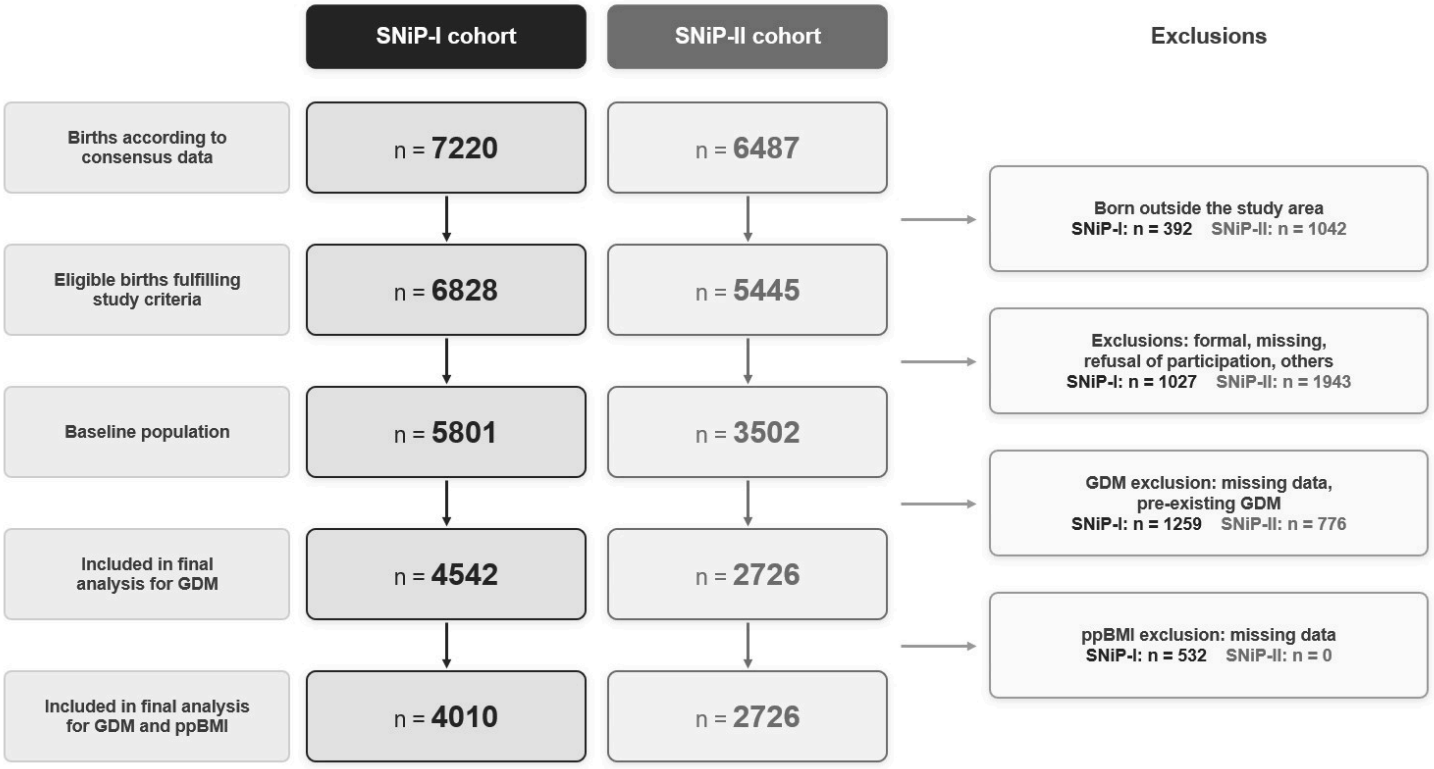

Figure 1

Derivation of the SNiP-I and SNiP-II analysis cohorts. Flow of participants from all births recorded in the consensus data to the cohorts included in the final analyses for gestational diabetes (GDM) and for GDM and pre-pregnancy body mass index (ppBMI), with the number of and reasons for exclusions at each step.

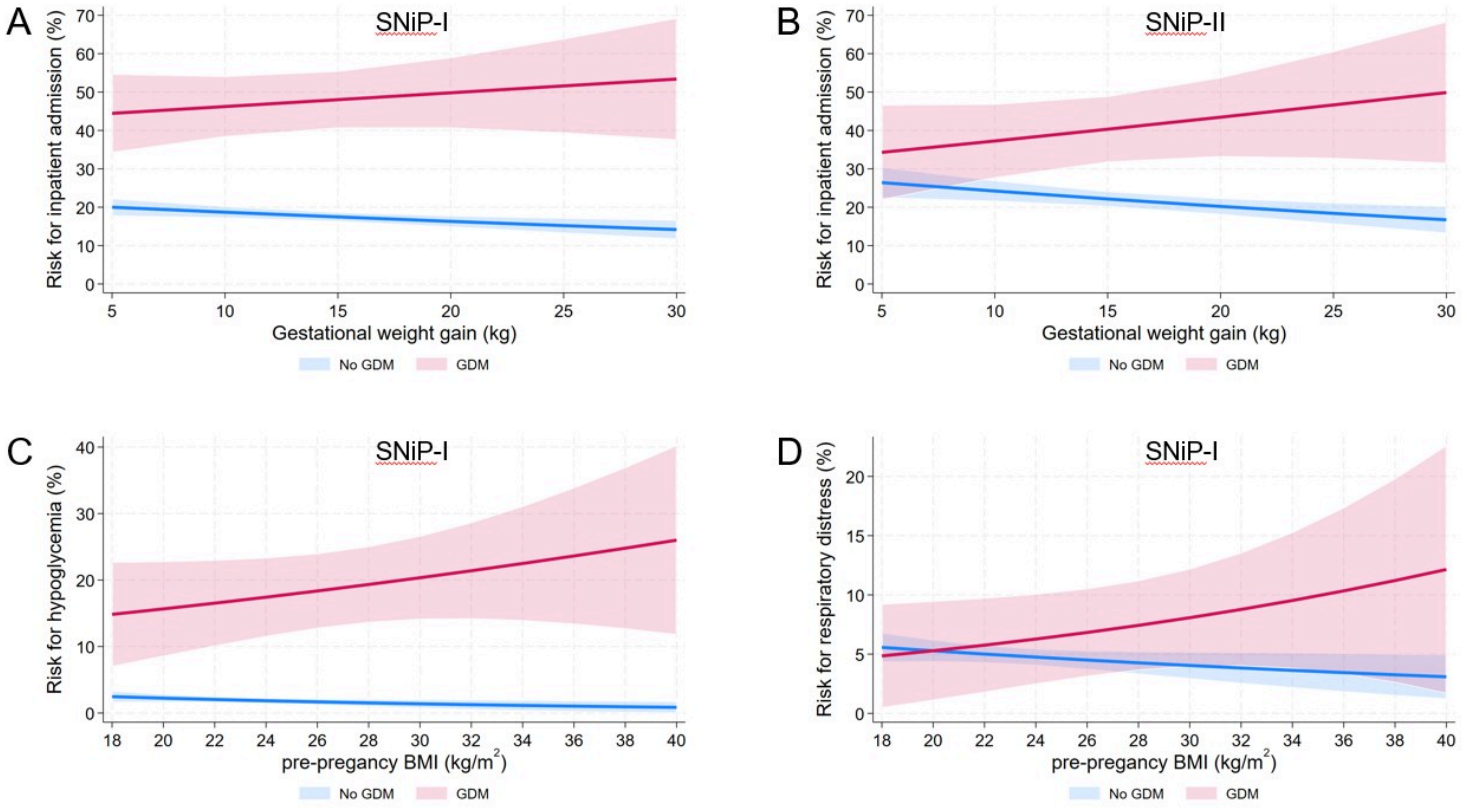

Figure 2

Effect modification of GDM-associated risk in SNiP-I and SNiP-II, derived from logistic and linear regression models adjusted for maternal age, alcohol consumption and maternal education. A) SNiP-I: gestational weight gain (GWG) and inpatient admission. B) SNiP-II: GWG and inpatient admission. C) SNiP-I: pre-pregnancy body mass index (ppBMI) and neonatal hypoglycaemia. D) SNiP-I: ppBMI and respiratory distress. These analyses are exploratory.

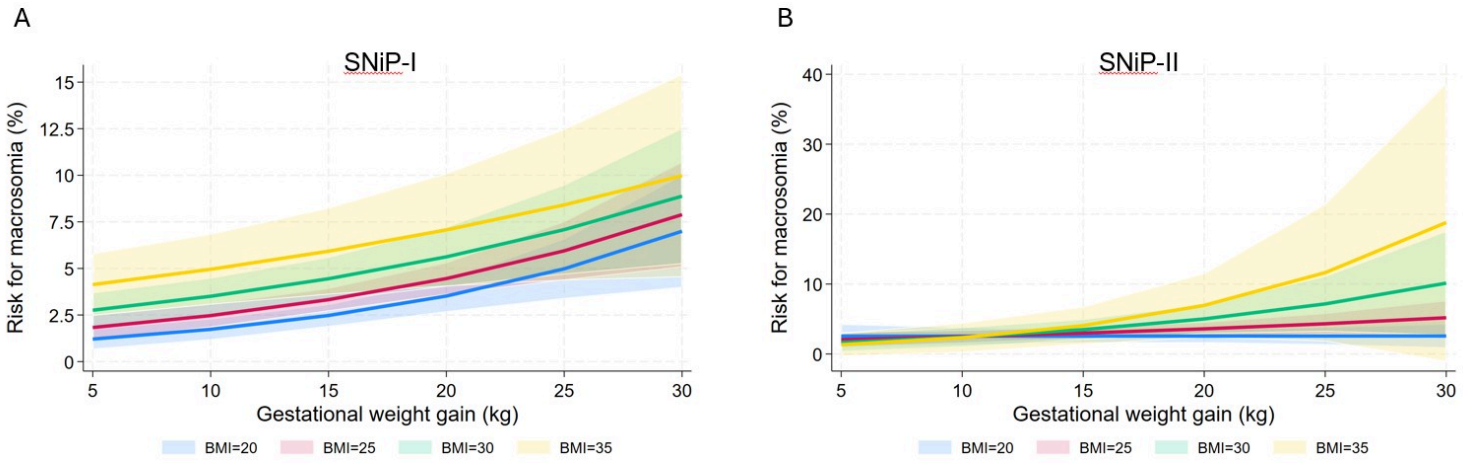

Figure 3

Interactions of pre-pregnancy body mass index (ppBMI) and gestational weight gain (GWG) during pregnancy on the risk of macrosomia in mothers without GDM in A) the SNiP-I cohort and B) the SNiP-II cohort. These analyses are exploratory.

Supplementary Files

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