

# Population Health Significance of Gestational Diabetes

N. WAH CHEUNG, PHD<sup>1</sup>  
KAREN BYTH, PHD<sup>2</sup>

**OBJECTIVE** — Women who have had gestational diabetes mellitus (GDM) have a high risk of subsequently developing diabetes. However, the contribution of GDM toward the total population of people with diabetes, or its population health impact, has not been examined. Therefore, the aim of this study is to determine the population health significance of GDM by estimating the proportion of cases of diabetes in women that would have been preceded by a pregnancy complicated by GDM.

**RESEARCH DESIGN AND METHODS** — A MEDLINE search was conducted to identify controlled follow-up studies of women with GDM. Meta-analysis of these studies, using the Mantel-Haenszel method for pooling relative risks (RRs), provided an overall RR for the development of diabetes in women with GDM versus control women who had been pregnant without GDM. Recent large studies examining the prevalence of GDM were also reviewed. This enabled the calculation of the population-attributable risk (PAR) for these populations. In this case, the PAR represents the proportion of cases of diabetes among parous women that were associated with previous GDM.

**RESULTS** — From six controlled follow-up studies, the overall RR for developing diabetes after GDM was calculated to be 6.0 (95% CI 4.1–8.8). Applying this to the studies of GDM prevalence, the PAR for GDM ranged from 0.10 to 0.31 (i.e., 10–31% of parous women with diabetes would have experienced a GDM pregnancy earlier).

**CONCLUSIONS** — In some populations, women who have had GDM comprise a substantial proportion of subjects who ultimately develop diabetes. Effective measures to prevent women with GDM from progressing to frank diabetes could therefore have a significant population health impact.

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With the rapidly increasing prevalence of diabetes around the world (1), there is an urgent need to develop affordable and effective preventative strategies and identify high-risk populations in whom such strategies can be implemented. People with impaired glucose tolerance or impaired fasting glucose are one such group, and the remarkable results of the Diabetes Prevention Program highlight that lifestyle or pharmacological intervention may be ef-

fective in preventing or delaying the onset of diabetes (2). Women with gestational diabetes mellitus (GDM) may represent another such population. GDM is a common disorder of pregnancy, and women who have had GDM are at high risk for the development of diabetes. For many, GDM can be considered a transient unmasking of an underlying predisposition to diabetes, induced by the metabolic changes of pregnancy (3). Recognition of this predisposition to diabetes may provide patients

the opportunity and incentive to undertake lifestyle measures to reduce their risk for diabetes and encourage regular screening for diabetes so that intervention can be instituted before the establishment of harmful end-organ effects of diabetes.

Whereas it is clear that women who have had GDM are at high risk of subsequent diabetes, how significant is this group in population health terms? For interventions directed at women with GDM to have a meaningful population health impact, women with GDM would need to comprise a significant proportion of the people ultimately destined to develop diabetes. The aim of the current study is to review earlier studies examining the prevalence of GDM and risk of subsequent diabetes and, from these reports, estimate the potential population health impact of GDM. To do so, we have used an epidemiological tool—the population-attributable risk (PAR).

PAR (also known as population etiological fraction) is an established method of estimating the proportion of all events of interest that may be attributable to a given exposure (4). In this case, we can use it to estimate the proportion of cases of diabetes in the female population that might be related to the earlier development of GDM given reasonable assumptions about the prevalence of GDM and the risk of developing diabetes. Even in the absence of any obvious etiological role of GDM, the PAR provides an estimate of the potential impact of an effective intervention on overall diabetes prevalence.

## RESEARCH DESIGN AND METHODS

### Literature search

A MEDLINE literature search was conducted for English articles containing the subject heading “gestational diabetes.” This was expanded to include all articles with the subheadings of “blood,” “classification,” “complications,” “diagnosis,” “economics,” “epidemiology,” “ethnology,” “genetics,” and “prevention and control.” This search yielded 609 articles.

From the <sup>1</sup>Department of Diabetes and Endocrinology, Westmead Hospital, University of Sydney, Sydney, Australia; and the <sup>2</sup>Westmead Millennium Institute, University of Sydney, Sydney, Australia.

Address correspondence and reprint requests to N. Wah Cheung, Department of Diabetes and Endocrinology, Westmead Hospital, Westmead, NSW 2145, Australia. E-mail: wah@westgate.wh.usyd.edu.au.

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**Abbreviations:** ADIPS, Australasian Diabetes in Pregnancy; GDM, gestational diabetes mellitus; PAR, population-attributable risk; USPHS, U.S. Public Health Service; WHO, World Health Organization.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

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Table 1—Diagnostic criteria for GDM

Criteria	Glucose load (g)	Duration (h)	Abnormal values for diagnosis	Blood glucose thresholds*
NDDG (6)	100	3	≥2	0 h, 105 mg/dl; 1 h, 190 mg/dl; 2 h, 165 mg/dl; 3 h, 145 mg/dl
Carpenter and Coustan (7)	100	3	≥2	0 h, 95 mg/dl; 1 h, 180 mg/dl; 2 h, 155 mg/dl; 3 h, 140 mg/dl
American Diabetes Association (8)†	75	2	≥2	0 h, 95 mg/dl; 1 h, 180 mg/dl; 2 h, 155 mg/dl
Damm et al. (9)	50	3	≥2	0 h, 115 mg/dl; 0.5 h, 182 mg/dl; 1 h, 182 mg/dl; 1.5 h, 157 mg/dl; 2 h, 137 mg/dl; 2.5 h, 137 mg/dl; 3 h, 119 mg/dl
O'Sullivan and Mahan (10)	100	3	≥2	0 h, 90 mg/dl; 1 h, 165 mg/dl; 2 h, 145 mg/dl; 3 h, 125 mg/dl
Gillmer et al. (11)	50	3		Area under curve of 0, 0.5, 1, 1.5, 2, 2.5, and 3 h ≥792 units
EASD (12)	75	2	1	2 h ≥162 mg/dl
WHO (13)	75	2	≥1	0 h, 126 mg/dl; 2 h, 140 mg/dl
ADIPS (14)	75	2	≥1	0 h, 99 mg/dl; 2 h, 144 mg/dl
Mercy (15)	50	3	2	1 h ≥162 mg/dl and 2 h ≥126 mg/dl

\*All the criteria use venous plasma except for reference 10 (whole blood) and 15 (capillary plasma). †Current American Diabetes Association position statement recommends use of either the 100-g glucose tolerance test with Carpenter and Coustan criteria or the 75-g glucose tolerance test. EASD, European Association for the Study of Diabetes; NDDG, National Diabetes Data Group.

Abstracts of these articles were reviewed to:

1. Estimate the relative risk (RR) of developing diabetes after GDM. These articles needed to have followed women after GDM and included a control pregnancy population who did not have GDM.
2. Assess the current prevalence of GDM. These articles needed to apply universal screening to an unselected population of >1,000 pregnant women, have screened at least 80% of the population, and have been published within the last 10 years, between 1992 and 2002. The purpose of this was not to conduct a comprehensive review of the prevalence of GDM, but to gain an estimate of its current prevalence.

Articles that appeared to be suitable were obtained and reviewed in more detail. Further articles were also identified from reference lists of the articles obtained. In total, six distinct articles meeting the selection criteria were found for number 1 above, and 14 articles were found for number 2. A range of different criteria for the diagnosis of GDM had been applied in these studies (Table 1).

### Meta-analysis of RR

The Mantel-Haenszel method (5) was used to combine the information across all the follow-up studies about the number of women who subsequently developed diabetes in the GDM and control groups. The RRs were computed for each study and combined in a weighted mean or overall RR. An RR of 1 indicated no

effect of GDM on subsequent development of diabetes. If a study reported no cases of diabetes among either GDM or control subjects, 0.5 was added to each cell entry to allow estimation of the RR (5). Mantel-Haenszel's  $\chi^2$  test of homogeneity (5) of the RRs across the studies was performed to assess the effect of heterogeneity among the studies.

### PAR

The PAR is the excess number of cases of a disease resulting from an exposure divided by the total number of cases in a defined population. This can be calculated by the following:

$$PAR = \frac{P_e(RR - 1)}{P_e(RR - 1) + 1}$$

where  $P_e$  is the population prevalence of the exposure and RR is the relative risk of the disease occurring in people subjected to the exposure (4).

RR can be determined from controlled follow-up studies of women with GDM. The control subjects provide an indication of the prevalence of diabetes within the unexposed female population who have had a pregnancy. For a specific population,  $P_e$  is the local prevalence of GDM, but a better estimate of the prevalence in the wider population can be obtained from large-scale studies of GDM prevalence.

**RESULTS**— The RRs for developing diabetes after GDM were calculated for the individual controlled studies of GDM

follow-up (Table 2). These ranged from 1.8 to 20.4. O'Sullivan (20) has reported data using both World Health Organization (WHO) and U.S. Public Health Service (USPHS) criteria for the diagnosis of subsequent diabetes. The results using the WHO criteria (RR 6.6) have been used for the calculation of overall RR below, even though the application of the USPHS criteria (RR 7.1) would have given slightly more impressive findings. Two controlled studies were excluded from the analysis because they were earlier reports of studies already included. These studies included one from O'Sullivan and Mahan (10) (RR 5.2 at up to 8 years follow-up by USPHS criteria) and one of the same population as in the study by Persson et al. (21) (RR 3.2 at 3–4 years follow-up).

The studies were combined to give an overall RR of 6.0 (95% CI 4.1–8.8). The large long-term study by O'Sullivan contributed most to the overall RR estimate, with a weighting of 67%. The  $\chi^2$  test of homogeneity was not significant ( $P = 0.4$ ), indicating no statistically significant evidence of heterogeneity among the RRs for the individual studies, despite differences in diagnostic criteria for GDM and different durations of follow-up.

The large studies obtained in the literature search (Table 3) were then used to provide estimates of GDM prevalence. PARs for this range of GDM prevalences were calculated using the overall estimated RR of 6.0. The 95% CIs for the PARs were obtained by combining the 95% CI for the overall RR of 4.1–8.8, with each tabulated GDM prevalence. The calculated PARs ranged from 0.10 to

Table 2—Controlled studies of women with and without GDM who were followed up and tested for the development of diabetes

Study	Proportion of subjects with diabetes at follow-up		Study weight (%)	RR for type 2 diabetes	95% CI	Years follow-up	GDM criterion	Diabetes criterion	Study population
	GDM subjects	Control subjects							
Lee et al. (16)	18/193 (9.3%)	3/58 (5.2%)	13.1	1.8	0.55–5.9	6	WHO	WHO*	Hong Kong
Hanson et al. (17)	8/145 (5.5%)	0/23 (0%)	2.4	2.8	0.17–46.9	6–7	Gillmer et al.	WHO†	Sweden
Aberg et al. (18)	21/229 (9.2%)	1/61 (1.6%)‡	4.5	5.6	0.77–40.8	1	EASD	WHO†	Sweden
Benjamin et al. (19)	14/47 (30%)	3/47 (6%)	8.5	4.7	1.4–15.2	3–9	O'Sullivan and Mahan	WHO†	Zuni Indian, U.S.
O'Sullivan et al. (20)	224/615 (36.4%)	18/328 (5.5%)	66.9	6.6	4.2–10.5	22–28	O'Sullivan and Mahan	WHO†	U.S.
Damm et al. (9)	42/241 (17.4%)	0/57 (0%)	2.3	20.4	1.3–326	2–11	Damm et al.	WHO†	Denmark
Overall	332/1,615	25/615	100.0	6.0	4.0–8.7				

$\chi^2$  homogeneity = 5.33, df = 5,  $P$  = 0.4. \*Modified WHO criteria: fasting plasma glucose  $\geq 144$  or 2-h plasma glucose  $\geq 200$  mg/dl on a 75-g glucose tolerance test. †WHO criteria (1980) for the diagnosis of diabetes: fasting plasma glucose  $\geq 140$  or 2-h plasma glucose  $\geq 200$  mg/dl on a 75-g glucose tolerance test (13). ‡One control subject known to have developed diabetes who declined to participate in the follow-up study has been included in our calculations. EASD, European Association for the Study of Diabetes.

0.31 (95% CI 0.06–0.41). That is, 10–31% of cases of diabetes in parous women are associated with previous GDM. Because the PAR is a function of the prevalence of GDM, the population effect of GDM very much depends on its prevalence, and this in turn depends in part on the population and diagnostic criteria used. Therefore, in Australia, where the less stringent Australasian Diabetes in Pregnancy (ADIPS) and Mercy criteria were used, the prevalence of GDM was

relatively high (5.2–8.8%) and the population impact of GDM considerable, with an estimated PAR of 0.21–0.31 (95% CI 0.14–0.41). In studies where the more stringent National Diabetes Data Group criteria were used, the estimated PAR was 0.10–0.23 (95% CI 0.06–0.32) and, with the Carpenter and Coustan criteria, the PAR was 0.19–0.25 (95% CI 0.12–0.34). Where the WHO criteria were used, the estimated PAR range was 0.10–0.27 (95% CI 0.07–0.37).

**CONCLUSIONS**— This study demonstrates that a significant proportion of the female population with diabetes might have been detected earlier through the diagnosis of GDM. Although the potential range is wide, it would be reasonable to surmise that up to one-third of parous women with diabetes would have gone through a GDM pregnancy.

In estimating the PAR of GDM for diabetes, we have combined studies with differing durations of follow-up,

Table 3—Studies of GDM prevalence and calculated PAR published from 1992 to 2002

Author	GDM criteria	Study type	Country	Subjects (n)	GDM (%)	PAR	95% CI
Jang et al. (22)	NDDG	Prospective cohort	Korea	3,581	2.2	0.10	0.06–0.15
Jimenez-Moleon et al. (23)	NDDG	Retrospective cohort	Spain	2,574	2.5	0.11	0.07–0.16
Xiong et al. (24)	NDDG	Retrospective cohort	Canada	111,563	2.5	0.11	0.07–0.16
Danilenko-Dixon et al. (25)	NDDG	Retrospective cohort	U.S.	18,504	3.0	0.13	0.09–0.19
Ferrara et al. (26)	NDDG	Retrospective cohort	U.S.	28,330	3.2	0.14	0.09–0.20
Corrado et al. (27)	NDDG	Prospective cohort	Italy	1,000	3.4	0.15	0.10–0.21
Bartha et al. (28)	NDDG	Prospective cohort	Spain	3,986	5.9	0.23	0.15–0.32
Corrado et al. (27)	Carpenter and Coustan	Prospective cohort	Italy	1,000	4.6	0.19	0.12–0.26
Ferrara et al. (26)	Carpenter and Coustan	Retrospective cohort	U.S.	28,330	4.8	0.19	0.13–0.27
Yalcin and Zordu (29)	Carpenter and Coustan	Prospective cohort	Turkey	1,000	6.6	0.25	0.17–0.34
Schmidt et al. (30)	ADA 2000 75-g GTT	Prospective cohort	Brazil	4,977	2.4	0.11	0.07–0.16
Yang et al. (31)	WHO	Prospective cohort	China	9,471	2.3	0.10	0.07–0.15
Schmidt et al. (30)	WHO	Prospective cohort	Brazil	4,977	7.2	0.26	0.18–0.36
Lee et al. (16)	WHO	Retrospective cohort	Hong Kong	11,300	7.4	0.27	0.19–0.37
Davey and Hamblin (32)	ADIPS	Retrospective cohort	Australia	6,032	5.2	0.21	0.14–0.29
Martin et al. (33)	ADIPS	Prospective cohort	Australia	1,371	5.5	0.22	0.15–0.30
Moses et al. (34)	ADIPS	Retrospective cohort	Australia	1,829	7.2	0.26	0.18–0.36
Beischer et al.* (15)	Mercy	Retrospective cohort	Australia	16,820	8.8	0.31	0.21–0.41

References 26, 27, and 30 examined prevalence of GDM by two different sets of diagnostic criteria and hence are listed twice. \*Data for the 1991–1994 cohort in Beischer's study. ADA, American Diabetes Association; GTT, glucose tolerance test; NDDG, National Diabetes Data Group.

with differing diagnostic criteria for GDM, and in different populations. It would be expected that the more stringent the diagnostic criteria for GDM and the longer the follow-up, the greater the likelihood that these subjects will develop future diabetes. However, by examining RR, the same conditions applied to both the control pregnant women and GDM groups. Therefore, longer follow-up, or more stringent criteria, results in higher diabetes rates in both the GDM and the control groups. Hence, the RR of developing diabetes subsequent to GDM showed no statistically significant variation across the studies. This is also illustrated by the reports of O'Sullivan (10,20), which in the one cohort, showed little change in the RR of developing diabetes over time (RR 5.2 after 8 years of follow-up, RR 7.1 after 22–28 years by USPHS criteria), as did the studies of Persson et al. (21) and Hanson et al. (17) (RR 3.2 after 3–4 years, RR 2.8 after 6–7 years).

The generalizability of the calculated overall RR also has implications for the estimation of PAR in the prevalence studies. A potential limitation of the current study is that the calculation of overall RR has been heavily weighted to the study of O'Sullivan (which used relatively stringent criteria for the diagnosis of GDM), and this overall RR has then been used to estimate PAR in prevalence studies using a range of different diagnostic criteria. Using the overall RR of 6.0, the highest PARs were calculated in populations where the less stringent WHO, ADIPS, or Mercy criteria were applied. Although there was no statistical evidence of heterogeneity between the follow-up studies, there was only one follow-up study using the WHO criteria for GDM, which contributed to the calculation of overall RR for the development of diabetes after GDM. If the RR of 1.8 seen in the study of Lee et al. (16) was to be representative of the effect of using the WHO diagnostic criteria for GDM, then the true PAR may well be lower in these populations. With the increasing adoption of the WHO criteria, especially in the developing world, further controlled follow-up studies using these criteria are needed to enable a better estimation of the PAR of GDM for diabetes in these populations.

Another potential obstacle to a representative estimation of PAR in the current study is that the prevalence of GDM has

increased over time, even without the spurious effect of criteria changes (15). We have attempted to limit this effect by restricting the review of GDM prevalence to studies conducted within the last 10 years. Nonetheless, there was a wide range of GDM prevalence, even between studies using the same diagnostic criteria in similar populations, which in turn has produced a wide range of PARs in the studies reviewed.

This study has analyzed the proportion of cases of diabetes associated with prior GDM among women with diabetes who have had a pregnancy. The actual contribution of GDM for the development of diabetes in women is in fact lower because not all women will go through a pregnancy. However, this is unlikely to dramatically alter the findings of this study because the vast majority of women will experience pregnancy. Data from the 1995 U.S. National Survey of Family Growth (35) indicate that 88% of women aged 40–44 years will have had a pregnancy, and 82.5% will have at least one child. In the 1996 Australian census (36), only 11% of women aged 45 years were childless. Another issue is that some women diagnosed with GDM would have had unrecognized preexisting diabetes. This has not been factored into our calculations, but in the majority of populations in which the studies in Table 2 were conducted, women with preexisting diabetes would only comprise a small percentage of the cases of GDM, so again the effect on the results would be minimal.

The above issues notwithstanding, our study suggests that a significant proportion of women with diabetes may have been identified earlier via the diagnosis of GDM. Therefore, effective preventative strategies directed at women who have had GDM may have the potential to produce a significant population health impact. The effect is likely to be greatest in regions such as Australasia, where studies have consistently demonstrated a high incidence of GDM and about one-third of women with diabetes might have been identified earlier via a GDM pregnancy.

There are also other factors that reinforce the applicability of population health prevention strategies directed at women with GDM. The adoption of universal screening in many countries (and even selective screening) would identify to health care providers the vast majority of women at risk. This would enable

health providers to provide affected women with education regarding diabetes risk and lifestyle advice. Undergoing a GDM pregnancy also lets the woman experience life as a diabetic individual, and this warning may facilitate a long-term positive change in lifestyle. The systematic identification of women by the health system may also provide opportunities to deliver structured public health intervention programs.

The results of both the Diabetes Prevention Program (2) and the Finnish Diabetes Prevention Study (37), where intensive lifestyle intervention reduced diabetes rates in subjects with impaired glucose tolerance by 58% over 3–4 years, indicate that diabetes can be prevented or delayed in high-risk individuals. These results give optimism that similar effects might be achievable in the GDM population, and indeed, ~15% of subjects in the Diabetes Prevention Program and the Finnish study had a history of GDM. In economic terms, this would have profound benefits, with a study in 1993 estimating that a reduction of type 2 diabetes among women who have had GDM in the U.S. by 50% over 10 years would save the country \$331 million (38).

In conclusion, in some populations, women with GDM may account for a large proportion of women with diabetes in the future. Therefore, effective preventative strategies directed at this group may have the capacity to exert a significant population health impact.

## References

1. King H, Aubert RE, Herman WH: Global burden of diabetes, 1995–2025: prevalence, numerical estimates, and projections. *Diabetes Care* 21:1414–1431, 1998
2. Diabetes Prevention Program Research Group: Reduction in the incidence of type 2 diabetes with intensive intervention or metformin. *N Engl J Med* 346:393–403, 2002
3. Coustan D: Gestational diabetes. *Diabetes Care* 16 (Suppl. 3):8–15, 1993
4. Woodward M: *Epidemiology: Study Design, Data Analysis*. New York, Chapman & Hall, 1999, p. 132–137
5. Deeks JJ, Altman DG, Bradburn MJ: Statistical methods for examining heterogeneity and combining results from several studies in meta-analysis. In *Systematic Reviews in Health Care: Meta-Analysis in Context*. 2nd ed. London, BMJ Publishing Group, 2001, p. 285–312
6. National Diabetes Data Group: Classifica-

- tion and diagnosis of diabetes mellitus and other categories of glucose intolerance. *Diabetes* 28:1039–1057, 1979
7. Carpenter MW, Coustan DR: Criteria for screening tests for gestational diabetes. *Am J Obstet Gynecol* 144:768–773, 1982
  8. American Diabetes Association: Gestational diabetes mellitus (Position Statement). *Diabetes Care* 23 (Suppl. 1):S77–S79, 2000
  9. Damm P, Kuhl C, Berelsen A, Molsted-Pedersen L: Predictive factors for the development of diabetes in women with previous gestational diabetes mellitus. *Am J Obstet Gynecol* 167:607–616, 1992
  10. O'Sullivan JB, Mahan CM: Criteria for the oral glucose tolerance test in pregnancy. *Diabetes* 13:278–285, 1964
  11. Gillmer MDG, Beard RW, Broke FM, Oakley NW: Carbohydrate metabolism in pregnancy. I. Diurnal plasma glucose profile in normal and diabetic women. *Br Med J* 3:339–402, 1975
  12. Lind T, Phillips PR: Influence of pregnancy on the 75g OGTT: a prospective multicenter study: The Diabetic Pregnancy Study Group of the European Association for the Study of Diabetes. *Diabetes* 40:8–13, 1991
  13. World Health Organization: *WHO Expert Committee on Diabetes Mellitus Second Report*. Geneva, World Health Org., 1980 (Tech. Rep. Ser., no. 646)
  14. Martin FIR: The diagnosis of gestational diabetes. *Med J Aust* 155:122, 1991
  15. Beischer NA, Wein P, Sheedy MT, Steffen B: Identification and treatment of women with hyperglycaemia diagnosed during pregnancy can significantly reduce perinatal mortality rates. *Aust N Z J Obstet Gynaecol* 36:239–247, 1996
  16. Lee CP, Wong HS, Chan FY, Pun TC, To WK, Lam YH, Baldwin S, Wong VCW: Long-term prognosis of women with abnormal glucose tolerance in pregnancy. *Aust N Z J Obstet Gynaecol* 34:507–510, 1994
  17. Hanson U, Hartling SG, Persson B, Binder C: Increased molar proinsulin-to-insulin ratio in women with previous gestational diabetes does not predict later impairment of glucose tolerance. *Diabetes Care* 19:17–20, 1996
  18. Aberg AEB, Jonsson EK, Eskilsson I, Landin-Olsson M, Frid AH: Predictive factors of developing diabetes mellitus in women with gestational diabetes. *Acta Obstet Gynecol Scand* 81:11–16, 2002
  19. Benjamin E, Mayfield J, Winters D, Gohdes D: Diabetes in pregnancy in Zuni Indian women. *Diabetes Care* 16:1231–1235, 1993
  20. O'Sullivan JB: Diabetes mellitus after GDM. *Diabetes* 29 (Suppl. 2):131–135, 1991
  21. Persson B, Hanson U, Hartling SG, Binder C: Follow-up of women with previous GDM: insulin, C-peptide, and proinsulin responses to oral glucose load. *Diabetes* 40 (Suppl. 2):136–141, 1991
  22. Jang HC, Cho NH, Jung KB, Oh KS, Dooley SL, Metzger BE: Screening for gestational diabetes in Korea. *Int J Gynaecol Obstet* 51:115–122, 1995
  23. Jimenez-Moleon JJ, Bueno-Cavanillas A, Luna-Del-Castillo JD, Garcia-Martin M, Lardelli-Claret P, Galvez-Vargas R: Prevalence of gestational diabetes: variations related to screening strategy used. *Eur J Endocrinol* 146:831–837, 2002
  24. Xiong X, Saunders LD, Wang FL, Demanczuk NN: Gestational diabetes: prevalence, risk factors, maternal and infant outcomes. *Int J Gynaecol Obstet* 75:221–228, 2001
  25. Danilenko-Dixon DR, Van Winter JT, Nelson RL, Ogburn PL: Universal versus selective screening: application of 1997 American Diabetes Association recommendations. *Am J Obstet Gynecol* 181:798–802, 1999
  26. Ferrara A, Hedderston MM, Quesenberry CP, Selby JV: Prevalence of gestational diabetes mellitus detected by the National Diabetes Data Group or the Carpenter and Coustan plasma glucose thresholds. *Diabetes Care* 25:1625–1630, 2002
  27. Corrado F, Stella NC, Mancuso A, Triolo O, Bruno L, Artesio AC: Screening for gestational diabetes in Sicily. *J Reprod Med* 44:875–878, 1999
  28. Bartha J, Martinez-Del-Fresno P, Comino-Delgado R: Gestational diabetes mellitus diagnosed during early pregnancy. *Am J Obstet Gynecol* 183:346–350, 2000
  29. Yalcin HR, Zordu CG: Threshold value of glucose screening tests in pregnancy. Could it be standardised for every population? *Am J Perinat* 13:317–320, 1996
  30. Schmidt MI, Spichler ER, Duncan BB, Pousada JMDC, Reichelt AJ, Teixeira MM, Branchtein L, Yamashita T, Matos MC, Reichelt AJ, Forti AC: Gestational diabetes mellitus diagnosed with a 2-h 75-g oral glucose tolerance test and adverse pregnancy outcomes. *Diabetes Care* 24:1151–1155, 2001
  31. Yang X, Hsu-Hage B, Zhang H, Yu L, Dong L, Li J, Shao P, Zhang C: Gestational diabetes mellitus in women of single gravidity in Tianjin City, China. *Diabetes Care* 25:847–851, 2002
  32. Davey RX, Hamblin PS: Selective vs universal screening for gestational diabetes mellitus: an evaluation of predictive risk factors. *Med J Aust* 174:113–114, 2001
  33. Martin FIR, Ratnaik S, Wootton A, Condos P, Suter PEN: The 75g oral glucose tolerance in pregnancy. *Diabetes Res Clin Pract* 27:147–151, 1995
  34. Moses RG, Griffith RD, McPherson S: The incidence of gestational diabetes in the Illawarra area of New South Wales. *Aust N Z J Obstet Gynaecol* 34:425–427, 1994
  35. Centers for Disease Control and Prevention, National Center for Health Statistics: *Fertility, Family Planning and Women's Health: New Data From the 1995 National Survey of Family Growth*. Hyattsville, MD, 1997 (ser. 23, no. 19)
  36. Australian Demographic Statistics: *Lifetime Childlessness*. Canberra, Australia, Australian Bureau of Statistics, September 1999 (3101.0)
  37. Tuomilehto J, Lindstrom J, Eriksson JG, Valle TT, Hamalainen H, Ilanne-Parikka P, Keinonen-Kiukaanniemi S, Laakso M, Louheranta A, Rastas M, Salminen V, Uusitupa M, Finnish Diabetes Prevention Study Group: Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med* 344:1343–1350, 2001
  38. Gregory KD, Kjos SL, Peters RK: Cost of non-insulin-dependent diabetes in a woman with a history of gestational diabetes: implications for prevention. *Obstet Gynaecol* 81:782–786, 1993