Managing women with gestational diabetes mellitus in the postnatal period

Catherine Kim

Departments of Medicine and Obstetrics & Gynecology, University of Michigan, Ann Arbor, MI, USA

Guidelines for management of women with a history of gestational diabetes mellitus (GDM) in the postpregnancy period have lagged behind the recognition that this is an important time for medical intervention. However, in the past decade, the evidence-base for screening algorithms, contraceptive management, diabetes prevention strategies and implications for offspring has expanded. In this review, we discuss current recommendations for managing women with GDM in the postnatal period, with particular attention to postpartum diabetes screening, prevention of future glucose intolerance and family planning.

Keywords: gestational diabetes, postpartum, women

Received 10 July 2009; returned for revision 6 August 2009; revised version accepted 6 August 2009

Introduction

For half a century, the strong association between gestational diabetes mellitus (GDM), or glucose intolerance first recognized during pregnancy and postpartum maternal glucose intolerance has been acknowledged [1]. In a 1991 review, John B. O'Sullivan observed: 'Although the variability in diabetes incidence rates is wide, there is broad general agreement on the predictive nature of gestational blood glucose levels' [2], a statement that still holds. In a recent meta-analysis, GDM conferred a sevenfold risk for future maternal diabetes [3], and up to one-third of women with diabetes may have been affected by prior GDM [4]. GDM women's greater risk for postpartum glucose intolerance also includes risk for another episode of GDM [5].

Guidelines for management of this risk have lagged behind its recognition. Several factors may have interfered with studies to guide management. These factors include: the long length of time elapsed between GDM and incident future diabetes, the management of GDM and postpartum diabetes by different medical providers, and the traditional focus on fetal as opposed to maternal outcomes. However, in the past decade, the evidence-base for screening algorithms, contraceptive management, diabetes prevention strategies and implications for offspring has expanded. In this review, I discuss current recommendations for managing women with GDM in the postnatal period, with particular attention to postpartum diabetes screening, prevention of future glucose intolerance and family planning.

Postpartum Screening

According to cohort studies from Latina (Hispanic) populations in the USA, approximately 10% of women diagnosed with GDM had unrecognized preconception diabetes [6]. Postpartum glucose screening in the early postnatal period will detect these women. Later screening will detect women who eventually do develop elevated fasting and/or postchallenge glucose levels [7], despite initial normal fasting and postchallenge glucose levels. Although the time of initial postpartum screening is usually recommended at 6 weeks to coincide with the first postpartum visit [8–14], glucose may normalize much earlier after the delivery of the placenta, and screening before 6 weeks but after delivery might increase diabetes screening rates in recent gravidas [8].

Recommendations for postpartum diabetes screening for GDM women vary between medical organizations. The primary debate revolves around whether screening should consist of performance of a postpartum fasting glucose alone vs. a 75-g oral glucose tolerance test (OGTT). As of the time of the writing of this review, no organizations endorse the haemoglobin A_{1C} (HbA_{1C}) for diabetes screening, although this may change with the recent emphasis by the American Diabetes Association (ADA) upon the value of the HbA_{1C} for screening. As of 2008, the UK-based National Institute for Health and Clinical Excellence (NICE) recommends a postpartum fasting glucose only, specifically without the OGTT [8]. The 2007 Fifth-International Workshop Conference on GDM recommended that a 75-g OGTT be performed at ≥ 6 weeks postpartum [9]. In 2009, the American College of Obstetricians and Gynecologists stated that screening should be performed and notes that the OGTT demonstrates greater sensitivity than the fasting glucose, but that fasting glucose is acceptable [10], a contrast with its previous agnostic recommendations regarding screening. The diabetes screening guidelines of other medical organizations

adopt the guidelines for general at-risk populations. The 1999/2006 World Health Organization (WHO) guidelines recommend a 75-g OGTT [11,12]. The 1997/2003 ADA guidelines recommend a fasting glucose in general practice, although the guidelines recognize the OGTT as a valid diagnostic method [13,14].

The differences between medical organizations are because of disagreement regarding the importance of the greater sensitivity of the OGTT vs. its lower reliability, greater inconvenience and cost. Women may have defects in fasting glucose, postchallenge glucose, or both [15]; therefore, the OGTT will detect more glucose intolerant persons than the fasting value alone. In the general US population in the National Health and Examination Survey III, 44% of adults ≥ 40 years with either abnormal fasting or 2-h glucose values met both the fasting and 2-h glucose criteria [16]. Fourteen per cent met the fasting criteria but not the 2-h criteria, and fourty-one per cent met the 2-h criteria alone [16]. In the Diabetes Epidemiology: Collaborative analysis of Diagnostic criteria in Europe (DECODE) study, also a general population, only 28% of participants with impaired fasting glucose (IFG) or elevated 2-h values met both criteria and 31% met the 2-h criteria only [17]. However, the greater sensitivity of the OGTT is offset by the greater variability in the 2-h glucose level compared with the fasting glucose [18]. The day-to-day intraindividual coefficients of variation range from 6.4 to 11.4% for fasting glucose and 14.3-16.7% for the 2-h glucose among the general population [18,19]. In addition, the OGTT has greater initial cost and inconvenience, drawbacks cited by the ADA and NICE. In one study, almost 20% of women declined a postnatal GTT presumably because of its relatively lower acceptability [20].

Although organizations differ as to whether or not to obtain the 2-h glucose, the cut-offs for diabetes are similar across groups: fasting glucose of 7.0 mmol/l or 126 mg/dl and, if obtained, a 2-h glucose of 11.1 mmol/l or 200 mg/dl after a 75g challenge. The fasting glucose value of 126 mg/dl was chosen because of its threshold association with retinopathy [21]. The 2-h criterion of 200 mg/dl corresponded with both allcause and cardiovascular disease mortality, as well as providing roughly the same risk as a fasting glucose of 126 mg/dl [21]. Impaired glucose regulation (IGR) consists of IFG and/or impaired glucose tolerance (IGT). The ADA defines IFG as a fasting glucose level ≥ 100 mg/dl or 5.6 mmol/l [14] and the WHO defines IFG as a fasting glucose level >110 mg/dl or 6.1 mmol/l [12]. The ADA defines IGT as a 2-h glucose 140-199 mg/dl or 7.8-11.0 mmol/l, as does the WHO. The IFG cut-off was chosen based on review of receiver operator curves for diabetes prediction and the disagreement between ADA and WHO was based on whether receiver operator curves should be the basis for cut-offs [12,14]. The IGT cut-off was initially chosen more arbitrarily, although the 2-h glucose does correspond with cardiovascular mortality and future diabetes [12,14].

Several issues unique to women with recent GDM could potentially influence choice of screening test in this population. However, the influence of these issues is speculative and their impact has not been formally examined. First, women with recent GDM might benefit from more

sensitive screening strategies, because diabetes poses risk to future pregnancies. Specifically, this risk includes congenital anomalies of cardiac malformations, neural tube defects and limb dysgenesis. All are strongly associated with glucose levels before conception [22]. Second, women with GDM are, on average, approximately 10-20 years younger than other populations diagnosed with glucose intolerance [23,24]. Women with GDM who develop diabetes face relatively prolonged dysglycaemia, which could potentially place them at higher risk for diabetes complications than the general population. Earlier identification with more sensitive screening could lead to reduction of complications. In the Diabetes Control and Complications Trial and its follow-up, intensive treatment reduced microvascular complications, with effects persisting after discontinuation of the trial [25]. In statistical models, the postpartum OGTT is generally more advantageous among women with recent GDM if diabetes identification is the endpoint [26]. Finally, because the glucose test results may also be used to determine the presence of IGR or 'prediabetes' [11], earlier identification of IGR using the OGTT may lead to earlier prevention efforts.

Prevention of Maternal Glucose Intolerance

Two influential studies have sparked interest in diabetes prevention in the GDM population by examining diabetes prevention intervention among glucose intolerant adults. The Diabetes Prevention Program (DPP) [27], a multicentre randomized controlled trial, and the Finnish Diabetes Prevention Study [28], another large randomized controlled trial, allocated participants with IGT to intensive lifestyle interventions. The DPP also randomized participants to daily metformin. Both studies demonstrated that these interventions successfully delayed or prevented diabetes [27,28].

One of the recruitment criteria for the DPP was a history of GDM, and in a subanalysis, intervention effectiveness was compared between women with and without histories of GDM [29,30]. Women with histories of GDM enrolled in the placebo or control arm had a higher cumulative incidence of diabetes than parous women without histories of GDM, 38% vs. 26% [29]. Although metformin and lifestyle changes both reduced diabetes risk among GDM and non-GDM women, metformin and lifestyle had similar effectiveness in GDM women (53% vs. 50%), whereas lifestyle changes were more effective in non-GDM women and the overall cohort (58% vs. 31%) [30]. The reason for the reduced effectiveness of lifestyle among GDM women was their difficulty with maintaining weight losses over the 3-year study period. Although reasons are speculative, GDM women may have had more difficulty adhering to the demanding lifestyle changes; goals in the DPP were a 7% weight reduction through diet and physical activity, with physical activity goals set at 150 min per week of moderate physical activity [27].

These subanalyses suggest that women with a GDM history from over a decade ago would benefit from metformin or lifestyle changes. Some factors may limit implementation in the general GDM population. GDM women in the DPP were on average, over 40 years old, greater than the average age of

the majority of GDM women in the immediate postpartum period [31]. Moreover, the randomized trial probably selected for a highly motivated group of participants that did not face or faced reduced barriers to implementation of lifestyle changes. Although other studies have demonstrated that lifestyle changes are possible in the several years postpartum [32–34], these studies have been small, focused on weight reduction, and did not examine glucose tolerance.

Other studies have explored the use of pharmacologic agents to prevent diabetes in GDM women. In the Troglitazone in Prevention of Diabetes (TRIPOD) study, women with GDM in the past 4 years were randomized to receive troglitazone, a thiazolidinedione, or placebo [35]. Troglitazone reduced the cumulative incidence of diabetes compared to placebo (5.4% vs. 12.1%), but troglitazone has since been discontinued because of reports of hepatotoxicity [35]. Although pioglitazone may also have similar effects [36], the safety profile of the thiazolidinediones regarding future cardiovascular and osteoporotic disease may limit their use for diabetes prevention.

Although women with GDM are at risk for future episodes of GDM, few studies have examined whether interventions can successfully modify this risk. In one study of overweight women, a low-intensity walking program during pregnancy led to decreased glucose values during a prenatal OGTT, but this study was limited by lack of a control group and small sample size (n=23) [37]. One ongoing study conducted in Western Massachusetts by Lisa Chasan-Taber and colleagues is randomizing pregnant high-risk women to a physical activity intervention with the aim of reducing risk for GDM (Chasan-Taber, personal communication); the intervention aims to increase exercise and also aims to identify serum biomarkers that may assess in future risk stratification.

Postpartum Contraception, Including Breastfeeding

As GDM women are by definition of child-bearing age, family planning is a key issue in postpartum period. Pregnancy itself may be diabetogenic for GDM women through weight gain or other hormonal factors [38], with an increase in relative risk for diabetes between 2 and 3 [39]. Therefore, if family planning is desired by the GDM woman, prevention of further pregnancy will prevent both another GDM pregnancy as well as reduce diabetes risk [38]. Several contraceptive strategies may influence diabetes risk, particularly in GDM women. The following section reviews these strategies, which include the lactation amenorrhoea method (LAM) and hormonal contraceptive methods. Other methods, such as barrier contraception and intrauterine devices (IUDs), have similar effectiveness in postpartum GDM women as in other populations and do not appear to influence diabetes risk.

LAM has effectiveness rates comparable with birth control pills and other common and effective methods of contraception [40]. Moreover, breastfeeding has the added benefit of decreasing weight [41], itself a risk factor for future diabetes. Although breastfeeding may also affect glucose levels independent of weight [42], the impact upon maternal diabetes incidence has not yet been proven. Among GDM women, two

studies have examined the association between breastfeeding in the immediate postpartum and future diabetes and did not find associations, although these results may have been limited by lack of diabetes screening in all participants [43,44]; similarly, the lack of association between breastfeeding and diabetes in the Nurses Health cohort may have also been because of underascertainment [45]. Interestingly, more studies support that breastfeeding may reduce diabetes incidence among offspring, as opposed to diabetes incidence in mothers [46–48].

However, to practice LAM effectively, women must begin breastfeeding immediately after delivery, avoid any supplementation and breastfeed at least every 4 h during the daytime and every 6 h during the night [49]. If women begin menstruating or supplementing earlier, the effectiveness of LAM decreases dramatically [49]; theoretically, women might conceive after ovulation but before their first menstrual period and might now know they were more susceptible to pregnancy. In addition, the effectiveness of LAM is limited after the 6 months after delivery, at which time another method must be initiated [49]. Because of these potential difficulties with implementation, the use of condoms in conjunction with LAM may decrease contraceptive failure rates.

Hormonal methods, including combination oral contraceptives, have comparable effects on glucose tolerance in women affected and not affected by GDM [50,51], although these comparisons were limited by small numbers of participants. Impact of any hormonal contraceptive method upon glucose tolerance appeared to be minimal in several small randomized controlled trials [52] and in one larger prospective study compared to no hormonal contraception [53]. Of the hormonal contraceptives, combination oral contraceptives or oestrogen—progestin birth control pills are the most popular [54]. The choice of oestrogen—progestin pill affects glucose metabolism in glucose intolerant women in individual studies, although studies conflict regarding the optimal progestin [52].

Because of the birth control pill requirement for daily ingestion, and the associated reduction in effectiveness because of missing pills, other oestrogen-progestin hormonal methods have been developed that do not require daily ingestion. The contraceptive vaginal ring is a flexible ethinyl vinyl acetate ring which releases ethinyl estradiol and the progestin etononorgestrel continuously [55]. Absorption is dependent upon contact with vaginal mucosae, and the ring does not need to be placed in a particular location, like the diaphragm. Impact on glucose appears to be minimal [55]. The contraceptive patch relies on a transdermal delivery system, which releases ethinyl estradiol and the progestin norelgestromin continuously [56]. The patch releases higher doses of ethinyl estradiol than lowdose birth control pills, and for this reason may be less desirable than other effective delivery methods with lower oestrogen release; however, the patch does not appear to adversely affect glucose tolerance [57].

Progestin-only pills may have reduced effectiveness compared to their combination oestrogen-progestin counterparts [58], but these pills are still popular in the early postpartum period because they are not thought to alter milk supply [58]. In addition, progestin-only pills are thought to have less effects on blood pressure and coagulation risk [58].

However, the use of a progestin-only pill in combination with breastfeeding increased risk of conversion to type 2 diabetes among Latinas with histories of GDM [53]. Although explanations are speculative, lactation may be a relatively progestogenic state [53], and women at high risk for glucose intolerance may be particularly vulnerable to the insulin resistance in this state.

Long-acting progestin methods may have even larger effects on carbohydrate metabolism. Depot medroxyprogesterone acetate (Depo Provera, Pfizer; New York, NY, USA), or medroxyprogesterone delivered intramuscularly every 13 weeks, is a highly effective method of contraception, in part because it does not require daily ingestion of a pill [56]. However, Depo Provera may increase diabetes risk in populations at high risk, such as Navajo women [59] and Latinas [60] with histories of GDM who had abnormal triglycerides or who were breastfeeding. These effects could potentially be mediated by increases in adipose tissue associated with Depo Provera use and possibly through insulin secretion [61]. Newer progestin-only methods include Implanon (Schering; Kenilworth, NJ, USA), an etonogestrel implant. Although the doses of progestin are relatively low, no carbohydrate metabolism studies examine the implant during breastfeeding or by triglyceride status. Therefore, in the absence of other contraindications to oestrogen, progestinonly methods are not first-line choices for women with histories of GDM.

If the GDM woman does not anticipate conceiving in the few years after the postpartum, IUDs are highly effective methods that do not appear to adversely affect glucose metabolism, although no studies have been conducted among GDM women [62]. The copper IUD (TCu380A) and the levonorgestrel-releasing IUD are the most commonly used [62]. The different side effect profiles may cause selection of one over the other; copper IUDs can extend the menstrual period and associated menstrual cramping, whereas levonorgestrel IUDs may lead to amenorrhoea and a delay in return to fertility. If women desire permanent sterilization, vasectomy is easy to perform, has little morbidity, and obviously does not adversely affect women's metabolic profile. In contrast, the microinsert coil requires a hysteroscopic or laparoscopic procedure for insertion in the fallopian tubes. Like vasectomy or bilateral tubal ligation, these procedures are not believed to affect glucose metabolism.

Conclusion

GDM presents a unique opportunity to modify future disease risk for women and their offspring. Women receive closer medical attention than at other times in their adult lives, they are highly motivated to improve the health of their children, and women's health and their children's health are tightly linked. In order to successfully reduce the risk of future disease, postpartum women with histories of GDM need: (i) postpartum screening to identify previously undiagnosed diabetes, (ii) postpartum screening to identify women potentially eligible for lifestyle and metformin intervention, (iii) effective family planning to minimize the diabetes risk associated with an additional pregnancy as well as risk of

another GDM pregnancy, (iv) lifestyle modification to reduce chronic disease risk and (v) lifestyle modification to reduce risk of another GDM pregnancy. Regarding the last two points, the effectiveness of particular lifestyle modification programs in newly postpartum has not yet been demonstrated. Further investigation of which types of programs benefit which women and identification of women at particularly high risk for diabetes conversion are needed to increase the effectiveness of any prevention efforts.

Acknowledgements

Dr Kim was supported by grant K23DK071552 from the National Institutes of Health.

References

- 1. O'Sullivan J, Mahan C. Criteria for the oral glucose tolerance test in pregnancy. Diabetes 1964; **13**: 278–285.
- O'Sullivan J. Diabetes mellitus after GDM. Diabetes 1991; 29(Suppl. 2): 131–135.
- Bellamy L, Casas J, Hingorani A, Williams D. Type 2 diabetes mellitus after gestational diabetes: a systematic review and meta-analysis. Lancet 2009; 373: 1773–1779.
- Cheung N, Byth K. Population health significance of gestational diabetes. Diabetes Care 2003; 26: 2005–2009.
- Kim C, Berger D, Chamany S. Recurrence of gestational diabetes mellitus: a systematic review. Diabetes Care 2007; 30: 1314–1319.
- Buchanan T, Xiang A, Kjos S et al. Gestational diabetes: antepartum characteristics that predict postpartum glucose intolerance and type 2 diabetes in Latino women. Diabetes 1998; 47: 1302–1310.
- 7. Buchanan T, Catalano P. The pathogenesis of GDM: implication for diabetes after pregnancy. Diabetes Rev 1995; **3**: 584–601.
- 8. National Institute for Health and Clinical Excellence (NICE). Diabetes in Pregnancy: Management of Diabetes and its Complications from Preconception to the Postnatal Period. London: National Collaborating Centre for Women's and Children's Health; 2008.
- Metzger B, Buchanan T, Coustan D et al. Summary and recommendations of the Fifth International Workshop-Conference on Gestational Diabetes Mellitus. Diabetes Care 2007; 30: S251–S260.
- ACOG Committee on Obstetric Practice. Postpartum screening for abnormal glucose tolerance in women who had gestational diabetes mellitus. Obstet Gynecol 2009; 113: 1419–1421.
- World Health Organization. Definition, Diagnosis, and Classification of Diabetes Mellitus and its Complications: Report of a WHO Consultation. Part 1. Diagnosis and Classification of Diabetes Mellitus. Geneva: World Health Organization; 1999.
- 12. World Health Organization. Definition and diagnosis of diabetes mellitus and intermediate hyperglycemia: A report of WHO/IDF consultation. Geneva: World Health Organization; 2006.
- American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care 2008; 31(Suppl. 1): S55–S60.
- The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Follow-up report on the diagnosis of diabetes mellitus. Diabetes Care 2003; 26: 3160–3167.
- Nathan D, Davidson M, DeFronzo R et al. Impaired fasting glucose and impaired glucose tolerance: implications for care. Diabetes Care 2007; 30: 753-759.

review article

- Harris M, Eastman R, Cowie C, Flegal K, Eberhardt M. Comparison of diabetes diagnostic categories in the U.S. population according to 1997 American Diabetes Association and 1980–1985 World Health Organization diagnostic criteria. Diabetes Care 1997; 20: 1859–1862.
- 17. DECODE Study Group. Will new diagnostic criteria for diabetes mellitus change phenotype of patients with diabetes? BMJ 1998; **317**: 371–375.
- Mooy J, Grootenhuis P, de Vries H et al. Intra-individual variation of glucose, specific insulin and proinsulin concentrations measured by two oral glucose tolerance tests in a general Caucasian population: the Hoorn Study. Diabetologia 1996; 39: 298–305.
- Feskens E, Bowles C, Kromhout D. Intra- and interindividual variability of glucose tolerance in an elderly population. J Clin Epidemiol 1991; 44: 947–953.
- Holt R, Goddard J, Clark P, Coleman M. A postnatal fasting plasma glucose is useful in determining which women with gestational diabetes should undergo a postnatal oral glucose tolerance test. Diabet Med 2003; 20: 594–598.
- 21. The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Report of the expert committee on the diagnosis and classification of diabetes mellitus. Diabetes Care 1997; 20: 1183–1197.
- 22. Ray J, O'Brien T, Chan W. Preconception care and the risk of congenital anomalies in the offspring of women with diabetes mellitus: a meta-analysis. Q J Med 2001; **94**: 435–444.
- Harris M, Flegal K, Cowie C et al. Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in U.S. adults: the Third National Health and Nutrition Examination Survey. Diabetes Care 1998; 21: 518.
- Lawrence J, Contreras R, Sacks D. Trends in the Prevalence of Pre-Existing Diabetes and Gestational Diabetes Mellitus among a Racially/Ethnically Diverse Population of Pregnant Women, 1999–2005. Diabetes Care 2008; 31: 899–904.
- 25. The Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications Research Group. Retinopathy and nephropathy in patients with type 1 diabetes four years after a trial of intensive therapy. N Engl J Med 2000; 342: 381–389.
- Kim C, Herman W, Vijan S. Efficacy and cost of postpartum screening strategies for diabetes among women with histories of gestational diabetes mellitus. Diabetes Care 2007; 30: 1102–1106.
- Knowler W, Hamman R, Edelstein S et al. Prevention of type 2 diabetes with troglitazone in the Diabetes Prevention Program. Diabetes 2005; 54: 1150–1156.
- Tuomilehto J, Lindstrom J, Eriksson J et al. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. N Engl J Med 2001; 344: 1343–1350.
- Ratner R. Women with a history of gestational diabetes in the Diabetes Prevention Program. Fourth International Workshop-Conference on Gestational Diabetes Mellitus, 11/13/2005 Chicago, IL; 2005.
- 30. Ratner R. Prevention of type 2 diabetes in women with previous qestational diabetes. Diabetes Care 2007; **30**(Suppl. 2): S242–S245.
- Kim C, Sinco B, Kieffer E. Racial and ethnic variation in access to healthcare, provision of healthcare services, and rating of health among women with a history of gestational diabetes mellitus. Diabetes Care 2007; 30: 1459–1465
- O'Toole M, Sawicki M, Artal R. Structured diet and physical activity prevent postpartum weight retention. J Womens Health 2003; 12: 991–998.
- 33. Leermarkers E, Anglin K, Wing R. Reducing postpartum weight retention through a correspondence intervention. Int J Obes 1998; 22: 1103–1109.
- Lovelady C, Garner K, Moreno K, Williams J. The effect of weight loss in overweight, lactating women on the growth of their infants. N Engl J Med 2000; 342: 449–453.

- Buchanan T, Xiang A, Peters R et al. Preservation of pancreatic beta-cell function and prevention of type 2 diabetes by pharmacological treatment of insulin resistance in high-risk Hispanic women. Diabetes 2002; 51: 2796–2803.
- Xiang A, Peters R, Kjos S et al. Effect of pioglitazone on pancreatic betacell function and diabetes risk in Hispanic women with prior gestational diabetes. Diabetes 2006; 55: 517–522.
- 37. Mottola M. The role of exercise in the prevention and treatment of gestational diabetes mellitus. Curr Diab Rep 2008; 8: 299–304.
- Gunderson E, Lewis C, Tsai A et al. A 20-year prospective study of childbearing and incidence of diabetes in young women, controlling for glycemia before conception: the Coronary Artery Risk Development in Young Adults (CARDIA) Study. Diabetes 2007; 56: 2990–2996.
- Baptiste-Roberts K, Barone B, Gary T et al. Risk factors for type 2 diabetes among women with gestational diabetes: a systematic review. Am J Med 2009; 122: 207–214.
- Kennedy K, Visness C. Contraceptive efficacy of lactational amenorrhea. Lancet 1992; 339: 227–230.
- Olson C, Strawderman M, Hinton P, Pearson T. Gestational weight gain and postpartum behaviors associated with weight change from early pregnancy to 1 year postpartum. Int J Obes Relat Metab Disord 2003; 27: 117–127.
- McManus R, Cunningham I, Watson A, Harker L, Finegood D. Beta-cell function and visceral fat in lactating women with a history of gestational diabetes. Metabolism 2001; 50: 715–719.
- Kjos S, Peters R, Xiang A, Henry O, Montoro M, Buchanan T. Predicting future diabetes in Latino women with gestational diabetes. Diabetes 1995; 44: 586–591.
- 44. Buchanan T, Xiang A, Kjos S, Trigo E, Lee W, Peters R. Antepartum predictors of the development of type 2 diabetes in Latino women 11–26 months after pregnancies complicated by gestational diabetes. Diabetes 1999; **48**: 2430–2436.
- Stuebe A, Rich-Edwards J, Willett W, Manson J, Michels K. Duration of lactation and incidence of type 2 diabetes. JAMA 2005; 294: 2601–2610.
- Pettitt D, Knowler W. Long-term effects of the intrauterine environment, birth weight, and breast-feeding in Pima Indians. Diabetes Care 1998; 21(Suppl. 2): B138–B141.
- Mayer-Davis E, Dabelea D, Lamichhane A et al. Breast-feeding and type 2 diabetes in the youth of three ethnic groups: the SEARCH for diabetes in youth case-control study. Diabetes Care 2008; 31: 470–475.
- Young T, Martens P, Taback S et al. Type 2 diabetes mellitus in children: prenatal and early infancy risk factors among Native Canadians. Arch Pediatr Adolesc Med 2002; 156: 651–655.
- Labbok M, Hight-Laukaran V, Peterson A, Fletcher V, von Hertzen H, Van Look P. Multicenter study of the Lactational Amenorrhea Method (LAM): I. Efficacy, duration, and implications for clinical application. Contraception 1997; 55: 327–336.
- Skouby S, Kuhl C, Molsted-Pedersen L, Petersen K, Christensen M. Triphasic oral contraception: metabolic effects in normal women and those with previous gestational diabetes. Am J Obstet Gynecol 1985; 153: 495–500.
- Skouby S, Molsted-Pedersen L, Kuhl C. Low dosage oral contraception in women with previous gestational diabetes. Obstet Gynecol 1981; 59: 325–328.
- Lopez L, Grimes D, Schulz K. Steroidal contraceptives: effect on carbohydrate metabolism in women without diabetes mellitus. Cochrane Database Syst Rev 2007; CD006133.
- Kjos S, Peters R, Xiang A, Thomas D, Schaefer U, Buchanan T. Contraception and the risk of type 2 diabetes mellitus in Latina women with prior gestational diabetes mellitus. JAMA 1998; 280: 533–538.

DIABETES, OBESITY AND METABOLISM

review article

- Petitti D. Clinical practice: combination estrogen-progestin oral contraceptives. N Engl J Med 2003; 349: 1443–1450.
- Madden T, Blumenthal P. Contraceptive vaginal ring. Clin Obstet Gynecol 2007; 50: 878–885.
- Hatcher R, Trussell J, Nelson A, Cates Jr W, Stewart F, Kowal D. Contraceptive Technology. New York City, NY: Ardent Media, Inc.; 2007.
- Goa K, Warner G, Easthope S. Transdermal ethinylestradiol/norelgestromin: a review of its use in hormonal contraception. Treat Endocrinol 2003; 2: 191–206.
- 58. Chi I. The safety and efficacy issues of progestin-only oral contraceptives—an epidemiologic perspective. Contraception 1993; **47**: 1–21.
- Kim C, Seidel K, Begier E, Kwok Y. Diabetes and depot medroxyprogesterone contraception in Navajo women. Arch Intern Med 2001; 161: 1766–1771.
- Xiang A, Kawakubo M, Kjos S, Buchanan T. Long-acting injectable progestin contraception and risk of type 2 diabetes in Latino women with prior gestational diabetes mellitus. Diabetes Care 2006; 29: 613–617.
- 61. Kahn H, Curtis K, Marchbanks P. Effects of injectable or implantable progestin-only contraceptives on insulin-glucose metabolism and diabetes risk. Diabetes Care 2003; **26**: 216–225.
- 62. Thonneau P, Almont T. Contraceptive efficacy of intrauterine devices. Am J Obstet Gynecol 2008; **198**: 248–253.