Biological and Clinical Sciences Research Journal
eISSN: 2708-2261; pISSN: 2958-4728
www.bcsrj.com
DOI: https://doi.org/10.54112/bcsrj.v6i4.1535
*Biol. Clin. Sci. Res. J., Volume 6(4), **2025**: 1535*
Review Article

Mapping Gestational Diabetes Mellitus – A Narrative Review of Key Determinants and Epidemiology

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(Received, 24th March 2025, Accepted 18thApril 2025, Published 30st April 2025)

Abstract: *Gestational diabetes mellitus (GDM) is a glucose intolerance disorder that arises during pregnancy, posing significant risks to both maternal and neonatal health. The global burden of GDM is increasing, reflecting changes in demographic, genetic, and lifestyle factors.* ***Objective:*** *This narrative review aims to explore the epidemiology, risk factors, diagnostic criteria, and current preventive and management strategies associated with GDM, with an emphasis on its public health implications.* ***Methods:*** *This review utilized a comprehensive literature search of international databases including PubMed, Scopus, and Google Scholar. Relevant articles published in English over the last ten years were included. Keywords such as "gestational diabetes," "GDM risk factors," "insulin resistance in pregnancy," and "maternal outcomes" were used. Epidemiological trends, diagnostic approaches, and evidence-based prevention and treatment methods were synthesized from cohort studies, meta-analyses, and clinical guidelines.* ***Results:*** *The global prevalence of GDM varies significantly, ranging from 1% to 28%, largely influenced by screening protocols, lifestyle, and ethnic composition. Risk factors for GDM include maternal obesity, advanced maternal age, family history of diabetes, polycystic ovarian syndrome (PCOS), and ethnicity. The pathophysiology involves insulin resistance, exacerbated by placental hormones. Lifestyle interventions—particularly dietary modifications and physical activity—remain the cornerstone of management. Continuous glucose monitoring (CGM) and early screening contribute to improved glycemic control and better maternal-fetal outcomes. The link between GDM and future development of type 2 diabetes mellitus highlights the importance of postpartum follow-up.* ***Conclusion:*** *GDM is a growing public health concern due to rising obesity and type 2 diabetes rates. Early detection, individualized care plans, lifestyle interventions, and postpartum monitoring are critical to minimizing complications and improving long-term outcomes for both mothers and offspring.*

Keywords: Gestational diabetes mellitus, Glucose intolerance, Insulin resistance, Obesity, Polycystic Ovarian Syndrome

[***How to Cite:*** Sarfaraz S, Rizwan N, Sharif A, Altaf R, Nasim MB, Syed SK, Mushtaq A, Asim F, Mushtaq MN, Sarfraz M, Ahmad W, Ullah BB, Qader A. Mapping Gestational Diabetes Mellitus - A Narrative Review Of Key Determinants And Epidemiology. *Biol. Clin. Sci. Res. J.*, **2025;** 6(4): 223-226. doi: https://doi.org/10.54112/bcsrj.v6i4.1535

Introduction

Diabetes, a chronic metabolic disorder, is characterized by high blood glucose levels that may lead to various organ damage if left uncontrolled over the period. The organs affected by diabetes or high blood glucose levels include the eyes, kidneys, blood vessels, heart, and nerves (1). Diabetes has been categorized into different types according to the underlying mechanism of hyperglycemia. Gestational diabetes mellitus (GDM) was described by O'Sullivan as "carbohydrate intolerance of varying severity with onset or first recognition during pregnancy" (2). The initial criteria, which relied on the OGTT, were developed to detect those women who are at higher risk of developing subsequent type 2 diabetes mellitus (3). Since then, many diagnostic approaches to GDM have been developed globally. GDM is associated with both short and long-term maternal complications such as shoulder dystocia, pre-eclampsia, Cesarean delivery, type 2 diabetes mellitus, metabolic syndrome, and cardiovascular diseases (4, 5). The child would have complications such as macrosomia, birth trauma, neonatal hypoglycemia, impaired glucose tolerance, metabolic syndrome, and cardiovascular disease (6). Identifying women with GDM early is important because the majority of the described short-term complications may be prevented by proper management involving lifestyle and diet changes, metformin or insulin treatment (7, 8).

Prevalence

GDM occurs in nearly 1-28% of pregnant women worldwide. Thus, when comparing the prevalence of GDM even at similar diagnostic criteria and techniques, variations could be observed because of the differences in the population – mean age, ethnic distribution, prevalence of overweight/obesity, physical activity, diet, and the background incidence of type 2 diabetes mellitus (9-11). In 2010, a new diagnostic criteria of GDM were set by the International Association of Diabetes and Pregnancy Study Groups (IADPSG) for the first time that criterion depended on adverse pregnancy outcomes (12). In 2013, the WHO used these IADPSG criteria─ GDM is diagnosed when the 75-g OGTT is positive, the thresholds are adjusted to 1.75 times higher odds ratio of adverse pregnancy outcomes. The fasting glucose concentration was

defined as ≥ 5.1 mmol/L, 1-hour postprandial glucose as ≥ 10.0 mmol/L, and/or 2-hour postprandial glucose as ≥ 8.5 mmol/L. With the adoption of these criteria, which were based on consensus and have shifted to lower fasting plasma glucose thresholds, inclusion of one-hour value, requirement for a single elevated value to make the diagnosis, and using a one-step approach (as opposed to screening), there was a 2-11 fold as reported earlier, increase in the prevalence of GDM compared to the previous criteria (9, 12-14). This rise is in line with the increase in global obesity and the consequent rise in diabetes, especially in young people (15, 16). These hikes in GDM cases have therefore provoked a number of concerns on the possible effects on the system, health, quality, and cost.

Risk factors of gestational diabetes mellitus

Continuous glucose monitoring

Continuous glucose monitoring (CGM) has become one of the important tools in the maintenance of glucose levels in women diagnosed with GDM. The findings of a trial by Yu, Lv (17), comparing 340 pregnant Chinese women with GDM, indicated that CGM, when coupled with standard care, led to better glycemic control and fewer adverse pregnancy outcomes, such as preeclampsia, an increase in cesarean deliveries compared to routine care. For instance, the mean amplitude of glycemic excursions (MAGEs) and mean of daily differences were statistically lower in the group undergoing CGMs, which correlated better with maternal as well as neonatal outcomes.

Dietary patterns

There is scientific evidence that diet influences the probability of developing GDM; moreover, there are preventive or aggravating diets. An observational prospective cohort analysis examining the relation between low-carbohydrate diets and the incidence of GDM was conducted on participants of the Nurses' Health Study II. The results validated that animal protein and fat were positively associated with GDM, while vegetable protein and fat were not significantly linked to GDM (18). These outcomes suggest that women of childbearing age could lower their risk for GDM by consuming more plant-based sources of protein and fat than animal sources. A high saturated fat consumption and processed foods, and low fiber content make subjects susceptible to GDM due to insulin resistance. On the other hand, a diet rich in fiber and low saturated fat has been advised in order to reduce this risk (19).

Lifestyle

The Finnish Gestational Diabetes Prevention Study, known as RADIEL, was conducted to assess whether moderate lifestyle control of diet and exercise could prevent GDM in high-risk women. The study provided evidence that counseling on these aspects individually reduced the risk of GDM by 39 percent for the experimental group compared to the control group, which received conventional antenatal care (20). The intervention group also demonstrated significantly less gestational weight gain and increased physical activity levels, which are related to a better quality diet. From the study, it emerged that the best approach to managing GDM is through lifestyle interventions because overweight women and those with a history of obesity or previous GDM are considered a high-risk population. The lack of exercise is even, strongly linked with the GDM danger level. Engaging in moderate intensity physical activity may help increase insulin responsiveness, decrease body fat, and perhaps reduce the chances of getting GDM (21). However, the meta-analysis mentioned in the study suggested that the analysis produced conflicting evidence about the effects of prenatal exercise on the prevalence of GDM. Since observational studies have shown that GDM risk is inversely proportional to the levels of exercise, randomized controlled trials failed to offer a significant reduction in the risk of the disease.

Other preventive factors of GDM include diet, exercise, and sleep, which are also associated with the development of the disease. Unhealthy physical inactivity and irregular diets are thought to promote obesity and insulin resistances that triggers GDM. On the other hand, physical exercise and an appropriate diet before and during pregnancy can prevent GDM, particularly among women with greater risk factors such as maternal age and BMI (22).

Obesity and weight

One of the prevalent causes of GDM is obesity before pregnancy. That is why women with high pre-pregnancy BMI are characterized by impaired insulin sensitivity and, therefore, at a higher risk of GDM. Several papers, including the randomized clinical trials highlighted in the documents, draw attention to the fact that women with a BMI of 25 or more are at higher risk of GDM (21, 23). In the Hawkins et al. (2015) trial, a lifestyle modification intervention focused on overweight Hispanic women, as they have a higher risk of GDM due to high rates of obesity in this population group. The intervention appeared to be feasible for lowering gestational weight gain and increasing physical activity, but did not decrease GDM risk (23).

Obesity, as indexed by higher BMI, is one of the most potent risk factors associated with GDM. Research evidence has indicated time and again that obesity before conception is a risk factor for GDM. For example, pre-pregnant women with a BMI of 30 kg/m2 and above are five times more likely to develop GDM than women with a BMI of 25 kg/m2 and below (24). This association is particularly alarming given that obesity is rapidly becoming a global health concern. In the United States, for example, an estimated 60% of women of reproductive age are either overweight or obese (24).

Obesity causes insulin resistance.

Insulin resistance (IR) is perhaps the most significant mechanistic component of GDM, and obesity only worsens the IR. The deposition of an excessive amount of adipose tissue triggers the secretion of pro-inflammatory cytokines and free fatty acids (FFAs) that antagonize insulin signaling pathways. This leads to a decline in the sensitivity of insulin, which may become a risk factor during pregnancy since insulin demand rises due to the action of hormones secreted by the placenta (25). Obese women who have a pre-pregnancy weight of more than 25 and women who put on a lot of weight after delivery are likely to develop T2DM after GDM. For instance, an increase in weight of 5 kg to any subsequent value after a diagnosis of GDM triples the risk of the development of T2DM (25). The connection between obesity and GDM is not limited to the women's BMI status before pregnancy. GDM risk increases with weight gain during pregnancy, particularly if the woman was already overweight or obese when she conceived. Skipping meals or gaining excessive weight before pregnancy or during pregnancy, therefore, increases this risk (25). In GDM, insulin resistance is therefore a critical factor as it is in the rest of the gestational diabetes spectrum. During pregnancy, the degree of insulin resistance is elevated, primarily as a result of the actions of the hormones produced by the placenta, including hPL – human placental lactogen. However, in women with insulin resistance or a reduced capacity of insulin secretion, these physiological changes put stress on the regulation of blood glucose levels.

Insulin resistance and beta cells

The two categories of women most at risk include those having a history of GDM or those whose insulin resistance level was high before pregnancy. It has been postulated that GDM identifies a subcategory of patients who have an element of metabolic disturbance that may have been present before pregnancy. This dysfunction therefore manifests as a lack of beta-cell compensation for the resistance to insulin, and if not well managed, GDM will develop into T2DM (25). GDM should be viewed as

an early predictor of future metabolic disorders, since GDM may progress to T2DM in as much as 70% of the affected women in the future (22). Therefore, GDM is an opportunity for intervention because modifications in lifestyle following pregnancy can greatly help in preventing the development of diabetes.

Ethnic factors

Some ethnic groups, such as Hispanic, African American, and Asian, are at higher risk of developing GDM. For instance, Hispanic women possess significantly higher prevalence and complications of GDM than their counterparts, equally owing to other aspects like higher obesity levels and lower physical activity. Some of these trials concerned this population, whose interventions sought to prevent GDM by promoting modifiable factors such as diet and exercise (21, 23). In addition to both age and weight, other factors that can cause GDM include genetics and ethnicity. There is evidence that South Asian women are more at risk than white women for developing GDM. This increased risk may be attributed to genetic components as well as diet and exercise patterns (22). Besides genetic predisposition, other risk factors include the mother's previous history of diabetes, the child's birth weight, and ethnicity. Although genetic screening for GDM has not been the norm, knowledge of these hereditary and ethnic factors could assist physicians in proactively identifying groups at risk and, subsequently, introduce early interventions.

History of GDM or diabetes

A history of GDM carries a high risk of repetition in subsequent pregnancies especially the subsequent pregnancy. In addition, one investigation also indicated that having a family history of type 2 diabetes increases the risk of developing GDM. The review highlights that interventions regarding weight management and healthy behaviors after GDM could decrease the risk of the subsequent development of diabetes (19). There is an increased tendency for women who have a family history of type 2 diabetes to develop GDM (26, 27). In addition to this, there is evidence that genetic factors play a role in the development of insulin resistance, together with the ability of the body to respond to hormones during pregnancy.

Maternal age

It should be noted that one of the main established risk factors for the development of GDM is an increase in maternal age. Studies show that high age of the mother is another factor that influences the chances of high GDM risk. A study involving primiparous women recommended that the prevalence of GDM rises with the mother's age. The incidence of GDM was 2.67 times higher in women aged ≥35 years than the women aged <25 years (24). It is proposed that the risk results from a normal ageing process which causes insulin resistance and or a decline in beta-cell activity – risks that make older women particularly susceptible to developing GDM during their pregnancy.

Moreover, there is evidence that maternal age increases the risk across various categories of BMI. Normal weight women at forty years had a 3.2 times the risk of developing GDM compared to normal weight women who were twenty-eight years old; therefore, maternal age is an independent risk factor for GDM (24). Since there is an increase in child childbearing age of mothers, especially in developed countries, greatly caused by education or career pursuits, the occurrence of GDM will continue to rise.

Polycystic ovarian syndrome

GDM has been predicted that polycystic ovarian syndrome (PCOS) is one of the leading risk factors for the development of GDM. In PCOS, women have elevated levels of insulin that increase the probability of glucose intolerance. According to available research, PCOS and GDM have similar mechanisms of metabolic dysfunction, mainly insulin resistance. PCOS is a genetic endocrine disorder that is challenging to diagnose and often presents diversified clinical manifestations of clinical and/or biochemical androgen excess, ovulation disturbance, and polycystic ovaries (28). Impaired insulin action and β-cell dysfunction are involved in the pathogenesis of many aspects of PCOS and other forms of infertility, as well as in several young patients with impaired glucose tolerance and non-insulin-dependent diabetes (29). Several cross-sectional and case-control studies noted that women with PCOS had a significantly increased prevalence of glucose intolerance and type 2 diabetes (30-32), but other research demonstrated a higher proportion of GDM in women with PCOS than in those without (33, 34). A Taiwanese population-based study has shown that women with PCOS were significantly more likely to develop GDM compared to women without PCOS (20.46% vs 10.54%) (35). Furthermore, thyroids were found to be independently associated with GDM with an OR of 2.15, adjusting for confounding factors and plausible effects; thus, reinforcing the positive association between PCOS and GDM (35).

Conclusion

In conclusion, in the current state of scientific knowledge regarding GDM, this literature review reveals its polyfactorial character, including the prevalence of the phenomenon, the factors influencing GDM development, and the adverse effects of this pathology on the mother and child health. Different diagnostic criteria and strategies used to diagnose GDM and manage this condition have been developed in an effort to minimize the risk of complications. CGM and diet compliance, obesity, ethnic differences, past medical history of GDM, and maternal age are found to be contributory in the course/management of GDM. Knowledge of these factors, combined with early preventive measures, could reduce risk factors, protect maternal and neonatal health, and prevent the development of type 2 diabetes, according to research studies. Therefore, personalised care and prevention measures in GDM, whilst aiming at improving the health of the former and her offspring, are highlighted from the study.

Declarations

Data Availability statement
All data generated or analysed during the study are included in the manuscript.
Ethics approval and consent to participate
Approved by the department concerned.
Consent for publication
Approved
Funding
Not applicable

Conflict of interest

The authors declared the absence of a conflict of interest.

Author Contribution

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All participants contributed equally.

All authors reviewed the results and approved the final version of the manuscript. They are also accountable for the integrity of the study.

References

1. WHO. Diabetes 2024 [Available from: https://www.who.int/health-topics/diabetes?gad_source=1&gclid=Cj0KCQjwgrO4BhC2ARIsAKQ7zUnHpkDoMEb7dqkRd0c1rIngDb2NgWyLM0FQq8uQIUWNRvQGm5EBIMaAlOWEALw_wcB#tab=tab_1.
2. O'Sullivan JB. Gestational diabetes: unsuspected, asymptomatic diabetes in pregnancy. New England Journal of Medicine. 1961;264(21):1082-5.
3. Metzger BE. Summary and recommendations of the third international workshop-conference on gestational diabetes mellitus. Diabetes. 1991;40(Supplement_2):197-201.
4. Bellamy L, Casas J-P, Hingorani AD, Williams D. Type 2 diabetes mellitus after gestational diabetes: a systematic review and meta-analysis. The Lancet. 2009;373(9677):1773-9.
5. Fadl H, Magnuson A, Östlund I, Montgomery S, Hanson U, Schwarcz E. Gestational diabetes mellitus and later cardiovascular disease: a S wedish population based case–control study. BJOG: An International Journal of Obstetrics & Gynaecology. 2014;121(12):1530-6.
6. Pettitt DJ, Bennett PH, Knowler WC, Robert Baird H, Aleck KA. Gestational diabetes mellitus and impaired glucose tolerance during pregnancy: long-term effects on obesity and glucose tolerance in the offspring. Diabetes. 1985;34(Supplement_2):119-22.
7. Landon MB, Spong CY, Thom E, Carpenter MW, Ramin SM, Casey B, et al. A multicenter, randomized trial of treatment for mild gestational diabetes. New England Journal of Medicine. 2009;361(14):1339-48.
8. Crowther CA, Hiller JE, Moss JR, McPhee AJ, Jeffries WS, Robinson JS. Effect of treatment of gestational diabetes mellitus on pregnancy outcomes. New England journal of medicine. 2005;352(24):2477-86.
9. Behboudi-Gandevani S, Amiri M, Bidhendi Yarandi R, Ramezani Tehrani F. The impact of diagnostic criteria for gestational diabetes on its prevalence: a systematic review and meta-analysis. Diabetology & metabolic syndrome. 2019;11:1-18.
10. McIntyre HD, Catalano P, Zhang C, Desoye G, Mathiesen ER, Damm P. Gestational diabetes mellitus. Nature reviews Disease primers. 2019;5(1):47.
11. Fadl HE, Gärdefors S, Hjertberg R, Nord E, Persson B, Schwarcz E, et al. Randomized controlled study in pregnancy on treatment of marked hyperglycemia that is short of overt diabetes. Acta Obstetricia et Gynecologica Scandinavica. 2015;94(11):1181-7.
12. Metzger BE, Gabbe SG, Persson B, Lowe LP, Dyer AR, Oats JJ, et al. International association of diabetes and pregnancy study groups recommendations on the diagnosis and classification of hyperglycemia in pregnancy: response to Weinert. Diabetes care. 2010;33(7):e98-e.
13. Association AD. Standards of medical care in diabetes-2016. Diabetes care. 2016;39(1):S1.
14. Jiwani A, Marseille E, Lohse N, Damm P, Hod M, Kahn JG. Gestational diabetes mellitus: results from a survey of country prevalence and practices. The Journal of Maternal-Fetal & Neonatal Medicine. 2012;25(6):600-10.
15. Ng M, Fleming T, Robinson M, Thomson B, Graetz N, Margono C, et al. Global, regional, and national prevalence of overweight and obesity in children and adults during 1980–2013: a systematic analysis for the Global Burden of Disease Study 2013. The lancet. 2014;384(9945):766-81.
16. Chooi YC, Ding C, Magkos F. The epidemiology of obesity. Metabolism. 2019;92:6-10.
17. Yu F, Lv L, Liang Z, Wang Y, Wen J, Lin X, et al. Continuous glucose monitoring effects on maternal glycemic control and pregnancy outcomes in patients with gestational diabetes mellitus: a prospective cohort study. The Journal of Clinical Endocrinology & Metabolism. 2014;99(12):4674-82.
18. Bao W, Bowers K, Tobias DK, Olsen SF, Chavarro J, Vaag A, et al. Prepregnancy low-carbohydrate dietary pattern and risk of gestational diabetes mellitus: a prospective cohort study. The American journal of clinical nutrition. 2014;99(6):1378-84.
19. Nicklas JM, Zera CA, England LJ, Rosner BA, Horton E, Levkoff SE, et al. A web-based lifestyle intervention for women with recent gestational diabetes mellitus: a randomized controlled trial. Obstetrics & Gynecology. 2014;124(3):563-70.
20. Koivusalo SB, Rönö K, Klemetti MM, Roine RP, Lindström J, Erkkola M, et al. Gestational diabetes mellitus can be prevented by lifestyle intervention: the Finnish Gestational Diabetes Prevention Study (RADIEL) a randomized controlled trial. Diabetes care. 2016;39(1):24-30.
21. Nobles C, Marcus BH, Stanek III EJ, Braun B, Whitcomb BW, Solomon CG, et al. Effect of an exercise intervention on gestational diabetes mellitus: a randomized controlled trial. Obstetrics & Gynecology. 2015;125(5):1195-204.
22. Daly B, Toulis KA, Thomas N, Gokhale K, Martin J, Webber J, et al. Increased risk of ischemic heart disease, hypertension, and type 2 diabetes in women with previous gestational diabetes mellitus, a target group in general practice for preventive interventions: a population-based cohort study. PLoS medicine. 2018;15(1):e1002488.
23. Hawkins M, Hosker M, Marcus B, Rosal MC, Braun B, Stanek III EJ, et al. A pregnancy lifestyle intervention to prevent gestational diabetes risk factors in overweight Hispanic women: a feasibility randomized controlled trial. Diabetic Medicine. 2015;32(1):108-15.
24. Laine MK, Kautiainen H, Gissler M, Raina M, Aahos I, Järvinen K, et al. Gestational diabetes in primiparous women–impact of age and adiposity: a register-based cohort study. Acta obstetricia et gynecologica Scandinavica. 2018;97(2):187-94.
25. Bao W, Yeung E, Tobias DK, Hu FB, Vaag AA, Chavarro JE, et al. Long-term risk of type 2 diabetes mellitus in relation to BMI and weight change among women with a history of gestational diabetes mellitus: a prospective cohort study. Diabetologia. 2015;58:1212-9.
26. De Wilde M, Veltman-Verhulst S, Goverde A, Lambalk C, Laven J, Franx A, et al. Preconception predictors of gestational diabetes: a multicentre prospective cohort study on the predominant complication of pregnancy in polycystic ovary syndrome. Human reproduction. 2014;29(6):1327-36.
27. Bond R, Pace R, Rahme E, Dasgupta K. Diabetes risk in women with gestational diabetes mellitus and a history of polycystic ovary syndrome: a retrospective cohort study. Diabetic Medicine. 2017;34(12):1684-95.
28. Goodarzi MO, Dumesic DA, Chazenbalk G, Azziz R. Polycystic ovary syndrome: etiology, pathogenesis and diagnosis. Nature reviews endocrinology. 2011;7(4):219-31.
29. Holte J, Gennarelli G, Wide L, Lithell H, Berne C. High prevalence of polycystic ovaries and associated clinical, endocrine, and metabolic features in women with previous gestational diabetes mellitus. The Journal of Clinical Endocrinology & Metabolism. 1998;83(4):1143-50.
30. Lo JC, Feigenbaum SL, Escobar GJ, Yang J, Crites YM, Ferrara A. Increased prevalence of gestational diabetes mellitus among women with diagnosed polycystic ovary syndrome: a population-based study. Diabetes care. 2006;29(8):1915-7.
31. Legro RS, Gnatuk CL, Kunselman AR, Dunaif A. Changes in glucose tolerance over time in women with polycystic ovary syndrome: a controlled study. The Journal of Clinical Endocrinology & Metabolism. 2005;90(6):3236-42.
32. Conn JJ, Jacobs HS, Conway GS. The prevalence of polycystic ovaries in women with type 2 diabetes mellitus. Clinical endocrinology. 2000;52(1):81-6.
33. Weerakiet S, Srisombut C, Rojanasakul A, Panburana P, Thakkinstian A, Herabutya Y. Prevalence of gestational diabetes mellitus and pregnancy outcomes in Asian women with polycystic ovary syndrome. Gynecological endocrinology. 2004;19(3):134-40.
34. Reyes-Muñoz E, Castellanos-Barroso G, Ramírez-Eugenio BY, Ortega-González C, Parra A, Castillo-Mora A, et al. The risk of gestational diabetes mellitus among Mexican women with a history of infertility and polycystic ovary syndrome. Fertility and sterility. 2012;97(6):1467-71.
35. Pan M-L, Chen L-R, Tsao H-M, Chen K-H. Relationship between polycystic ovarian syndrome and subsequent gestational diabetes mellitus: a nationwide population-based study. PloS one. 2015;10(10):e0140544.