DOI: 10.38095/2181-466X-20261221-185-190 УДК 616.379-008.64:618.3:615.252.349

METFORMIN IN GESTATIONAL DIABETES MELLITUS: A REVIEW OF CURRENT EVIDENCE ON BENEFITS AND POTENTIAL RISKS

N. N. Shavazi, M. R. Ilkhomova
Samarkand state medical university, Samarkand, Uzbekistan

Key words: gestational diabetes mellitus, metformin, insulin therapy, glycemic control, perinatal outcomes, long-term effects, pregnancy, safety profile.
Tayanch so‘zlar: gestatsion qandli diabet, metformin, insulin terapiyasi, glikemik nazorat, perinatal natijalar, uzoq muddatli ta’sirlar, homiladorlik, xavfsizlik profili.
Ключевые слова: гестационный сахарный диабет, метформин, инсулинотерапия, гликемический контроль, перинатальные исходы, долгосрочные эффекты, беременность, профиль безопасности.

Gestational diabetes mellitus is associated with an increased risk of adverse maternal and perinatal outcomes. The use of metformin in GDM remains under discussion because the drug crosses the placental barrier and evidence regarding potential long-term effects of intrauterine exposure is limited. Analysis of randomized trials, meta-analyses, and clinical guidelines indicates that metformin provides glycemic control comparable to insulin and is associated with less gestational weight gain, although some patients require additional insulin therapy to achieve target glycemic levels. Short-term perinatal outcomes in most studies do not differ significantly. However, the issue of long-term metabolic effects in offspring remains unresolved and warrants further prospective research.

GESTATSION QANDLI DIABETDA METFORMINNING O‘RNI: FOYDA VA POTENSIAL XAVFLAR BO‘YICHA ZAMONAVIY MA’LUMOTLAR SHARHI

N. N. Shavazi, M. R. Ilhomova
Samarqand davlat tibbiyot universiteti, Samarqand, O‘zbekiston

Gestatsion qandli diabet onalarda va perinatal davrda noxush natijalar xavfining oshishi bilan bog‘liq. GQDda metformin qo‘llanilishi hali ham muhokama qilinmoqda, chunki preparat platsentar to‘siqdan o‘tadi va uning homilaga ta’siriga doir uzoq muddatli oqibatlar haqidagi ma’lumotlar cheklanganligicha qolmoqda. Randomizatsiyalangan tadqiqotlar, meta-tahlillar hamda klinik tavsiyalar tahlili shuni ko‘rsatadiki, metformin insulin bilan taqqoslanadigan darajada glikemik nazoratni ta’minlaydi va homilador ayollarda tana vazni ortishining kamroq bo‘lishi bilan kechadi, biroq ayrim bemorlarda qo‘shimcha insulinoterapiya zarurati yuzaga keladi. Shu bilan birga, qisqa muddatli perinatal natijalar aksariyat tadqiqotlarda sezilarli farq ko‘rsatmaydi. Avlodda kuzatilishi mumkin bo‘lgan uzoq muddatli metabolik ta’sirlar masalasi ochiq qolmoqda va kelgusida prospektiv tadqiqotlar o‘tkazilishini talab etadi.

МЕТФОРМИН ПРИ ГЕСТАЦИОННОМ САХАРНОМ ДИАБЕТЕ: ОБЗОР СОВРЕМЕННЫХ ДАННЫХ О ПОЛЬЗЕ И ПОТЕНЦИАЛЬНЫХ РИСКАХ

Н. Н. Шавази, М. Р. Ильхомова
Самаркандский государственный медицинский университет, Самарканд, Узбекистан

Гестационный сахарный диабет связан с повышенным риском неблагоприятных материнских и перинатальных исходов. Применение метформина при ГСД продолжает обсуждаться, поскольку препарат проходит через плацентарный барьер, а данные о возможных отдалённых эффектах внутриутробного воздействия остаются ограниченными. Анализ рандомизированных исследований, мета-анализов и клинических рекомендаций показывает, что метформин обеспечивает гликемический контроль, сопоставимый с инсулином, и сопровождается меньшей прибавкой массы тела у беременных, хотя у части пациенток возникает необходимость дополнительной инсулинотерапии. При этом краткосрочные перинатальные исходы в большинстве работ существенно не отличаются. Вопрос долгосрочных метаболических эффектов у потомства остаётся открытым и требует дальнейших проспективных исследований.

Relevance. Gestational diabetes mellitus (GDM) is one of the most common metabolic complications of pregnancy and an expanding global public health concern. According to the International Diabetes Federation, approximately one in six live births is affected by hyperglycemia in pregnancy, predominantly GDM, with prevalence estimates ranging from 10% to 25% depending on diagnostic criteria, population characteristics, and screening strategies [7]. The rising incidence parallels increasing rates of obesity, insulin resistance, and type 2 diabetes among women of reproductive age [7,2]. Pathophysiologically, GDM reflects inadequate pancreatic β-cell compensation for progressive placental hormone–mediated insulin resistance in late pregnancy. Resulting maternal hyperglycemia promotes transplacental glucose transfer, fetal hyperinsulinemia, and enhanced growth signaling, contributing to risks of macrosomia, shoulder dystocia, operative delivery, neonatal hypoglycemia, and respiratory morbidity. Beyond the perinatal period, up to 50% of affected women develop type 2 diabetes within 5–10 years, while exposed offspring demonstrate increased susceptibility to overweight and metabolic dysregulation, supporting the concept of developmental programming [2].

Insulin has traditionally served as the standard pharmacologic therapy when lifestyle measures are insufficient, owing to its established efficacy and lack of placental transfer, although its injectable administration and monitoring requirements may affect adherence and accessibility. Metformin has emerged as an alternative or adjunct due to oral administration, insulin-sensitizing effects, lower hypoglycemia risk, and reduced gestational weight gain, with randomized trials demonstrating comparable short-term perinatal outcomes in selected populations. However, placental transfer with fetal concentrations approaching maternal levels sustains debate regarding potential long-term offspring effects. Clinical practice varies internationally; in several countries, including Uzbekistan, metformin is not formally approved during pregnancy, reflecting precautionary regulation, limited local longitudinal data, and preference for non–placenta-crossing therapies. Conversely, contemporary reviews and updated guidance, including ADA Standards of Care (2023–2024) [1,11], recognize metformin as an option under defined conditions. Accordingly, its role in GDM management remains nuanced, with established short-term benefits but unresolved long-term questions. In this review, current evidence is summarized, its strengths and limitations are critically evaluated, and a balanced perspective on the benefits and remaining uncertainties of metformin in contemporary GDM management is presented.

Materials and Methods. A focused narrative literature review was conducted using the PubMed/MEDLINE database, with additional searches performed in the Cochrane Library. The analysis primarily included key randomized controlled trials investigating metformin use during pregnancy, such as the MiG study and its long-term offspring follow-up (MiG-TOFU), as well as the MiTy trial and the MiTy Kids extension study involving women with type 2 diabetes in pregnancy. The 2017 Cochrane systematic review and selected recent analytical publications (2023–2025) were also considered. Epidemiological context was informed by reports from the International Diabetes Federation and major meta-analyses evaluating maternal and neonatal risks associated with gestational diabetes mellitus.

Results and Discussion. Over the past two decades, the role of metformin in the management of gestational diabetes mellitus (GDM) has evolved from a controversial alternative to insulin into a widely investigated therapeutic option supported by an expanding body of randomized clinical evidence. Initial concerns regarding transplacental transfer and potential fetal exposure prompted rigorous evaluation of both short-term perinatal safety and longer-term offspring outcomes. As a result, a series of large randomized controlled trials and follow-up cohort studies have progressively clarified the maternal, neonatal, and early childhood effects of metformin therapy during pregnancy. In the randomized controlled trial by Rowan et al. [12] involving 751 women with gestational diabetes mellitus, the incidence of the primary composite neonatal outcome was similar between the metformin and insulin groups (32.0% vs 32.2%), confirming non-inferiority in short-term perinatal safety. Severe neonatal hypoglycemia occurred less frequently with metformin, whereas preterm birth was more common, mainly due to spontaneous deliveries. Neonatal anthropometry, cord blood insulin levels, and maternal hypertensive complications did not differ significantly between groups. Overall glycemic control was comparable, although metformin-treated women achieved target postprandial glucose levels more rapidly. At 6–8 weeks postpartum, impaired glucose tolerance was observed in 23.0% of the metformin group and 20.6% of the insulin group, indicating no clinically meaningful difference in short-term metabolic outcomes. Metformin therapy was associated with lower gestational weight gain by 36 weeks and greater postpartum weight reduction, as well as higher treatment acceptability (76.6% vs 27.2%). Serious adverse events were infrequent and similar between groups. Notably, 46.3% of women randomized to metformin required supplemental insulin; however, treatment intensification did not increase adverse neonatal outcomes compared with metformin monotherapy, supporting preserved perinatal safety.

The multicenter randomized MiG trial (Metformin in Gestational Diabetes trial) represented a pivotal study evaluating metformin for gestational diabetes mellitus, providing the first large-scale direct comparison with insulin in a clinically representative population. Participants were women at 20–33 weeks' gestation requiring pharmacologic glycemic control, with a mean age of 33.3 ± 5.3 years and body mass index of 32.1 ± 7.8 kg/m², indicating substantial insulin resistance. The cohort was ethnically diverse, and baseline metabolic characteristics (fasting plasma glucose 5.3 ± 1.1 mmol/L; HbA1c 5.7 ± 0.8%) were typical for GDM. Interim safety analysis confirmed acceptable tolerability, with infrequent serious adverse events not related to treatment. As pregnan-

cy progressed, 30–50% of women receiving metformin required supplemental insulin, reflecting physiological increases in insulin resistance during late gestation. The study demonstrated comparable perinatal outcomes between metformin and insulin, supporting non-inferiority of metformin as a first-line pharmacologic option for GDM. With similar glycemic control, metformin also showed advantages in treatment convenience and acceptability, consistent with patient-reported preferences [9]. The original MiG trial was followed by the prospective MiG-TOFU (Metformin in Gestational diabetes – The Offspring Follow-Up) study [13], undertaken to assess potential long-term effects of intrauterine metformin exposure given its transplacental transfer and fetal concentrations comparable to maternal levels. At two years of age, 318 children were evaluated (154 metformin-exposed, 164 insulin-exposed), with comparable maternal characteristics, pregnancy glycemic control, and neonatal outcomes across groups. Metformin-exposed children demonstrated modestly greater measures of subcutaneous adiposity, including larger mid-upper arm circumference and increased subscapular and biceps skinfold thickness, differences that remained statistically significant after adjustment for key confounders. However, no differences were observed in total fat mass, body fat percentage, central adiposity, or body composition assessed by Dual-Energy X-ray Absorptiometry (DEXA), indicating that variations were limited to subcutaneous fat depots without evidence of increased visceral adiposity. These findings suggest a potential shift toward a more metabolically favorable fat distribution pattern following metformin exposure, although the clinical significance remains uncertain. The authors highlight the need for continued longitudinal follow-up into later childhood and adolescence with detailed evaluation of insulin sensitivity, growth patterns, and cardiometabolic risk to clarify whether these differences represent adaptive metabolic programming or a transient early-life phenomenon. Subsequent follow-up of the MiG-TOFU cohort [14] assessed offspring at 7 and 9 years following intrauterine exposure to metformin or insulin. Overall analyses demonstrated no significant differences in total body fat percentage, visceral adiposity, or metabolic biomarkers, indicating that early variations in subcutaneous fat distribution were not associated with adverse metabolic effects in mid-childhood. At 7 years, groups remained comparable despite higher maternal glycemia and a greater proportion of infants born above the 90th percentile in the metformin group. By 9 years, metformin-exposed children showed higher body weight, larger waist and mid-upper arm circumferences, and an increased waist-to-height ratio, with borderline differences in body mass index and triceps skinfold thickness and a trend toward greater abdominal adipose tissue volume on MRI. Nevertheless, total body fat percentage measured by Dual-Energy X-ray Absorptiometry (DXA) and BIA, regional fat distribution on MRI, and metabolic markers—including fasting glucose, insulin, HOMA-IR, HbA1c, lipid profile, leptin, and adiponectin—did not differ significantly, supporting preserved metabolic health despite modest anthropometric variation. The 2017 Cochrane systematic review [3] synthesized evidence from 11 randomized controlled trials (n = 1,487) evaluating oral glucose-lowering agents in gestational diabetes mellitus. Insulin was not included, having been assessed in separate analyses. Substantial heterogeneity, small sample sizes, and low certainty of evidence precluded identification of superiority of any oral agent. Critical outcomes, including perinatal mortality, severe neonatal morbidity, and long-term offspring effects, were inadequately reported. The review emphasized the need for larger randomized trials with clinically meaningful endpoints and extended follow-up, highlighting persistent gaps in long-term safety data.

The review by Vera Tocci et al. [17] integrates evidence from clinical trials, cohort studies, experimental models, and guidelines on metformin use in gestational diabetes mellitus. Short-term data show that metformin improves glycemic control, reduces gestational weight gain, lowers preeclampsia risk, and—compared with glyburide—reduces macrosomia and neonatal hypoglycemia. Metformin crosses the placenta, and some studies report slightly lower birth weight and smaller neonatal anthropometric measures, while experimental findings suggest potential effects on mitochondrial and growth signaling pathways. Long-term evidence remains inconsistent: some data indicate accelerated postnatal growth and increased adiposity, whereas prospective human studies demonstrate modest BMI increases without metabolic abnormalities. Overall, the review concludes that metformin is an effective short-term alternative to insulin in GDM, but uncertainty regarding long-term offspring outcomes supports cautious, individualized use with appropriate counseling. The updated American Diabetes Association (ADA) Standards of Care (2023–2024) [1] recognize metformin as a therapeutic option for gestational diabetes mellitus, contingent upon informed consent and readiness to initiate or intensify insulin therapy if glycemic targets are not

achieved. While short-term perinatal outcomes are comparable to insulin, the guidelines underscore persistent uncertainty regarding long-term effects of intrauterine exposure. Accordingly, treatment decisions should be individualized, integrating discussion of benefits, uncertainties, and patient preferences.

In light of the findings from MiG and MiG-TOFU—where metformin demonstrated perinatal outcomes comparable to insulin but was associated with modest shifts in anthropometric parameters during later childhood—subsequent investigation in women with established type 2 diabetes in pregnancy represented a logical extension of the evidence base. This objective was addressed in the large international randomized controlled trial MiTy, which evaluated metformin as an adjunct to background insulin therapy. In the MiTy (Metformin in Women with Type 2 Diabetes in Pregnancy) trial [4], 502 women were randomized to receive metformin 1000 mg twice daily or placebo in addition to insulin. The primary composite neonatal outcome was identical between groups (40% vs 40%), indicating no increase in early neonatal risk with metformin. Maternal benefits were evident, including improved glycemic control (HbA1c 5.90% vs 6.10%), reduced insulin requirements, less gestational weight gain (7.2 vs 9.0 kg), and a lower cesarean delivery rate. Metformin was also associated with a shift in birth weight distribution, with lower mean birth weight and reduced rates of large-for-gestational-age and macrosomia, accompanied by a modest increase in small-for-gestational-age infants, suggesting attenuation of excessive fetal growth. The MiTy Kids follow-up study [5] evaluating offspring up to 24 months (n = 283) showed that these differences largely resolved by two years, with comparable body mass index z-scores, skinfold thickness, and growth trajectories, aside from a minor body mass index increase among boys that was not clinically meaningful. Overall, MiTy and MiTy Kids findings indicate that adjunctive metformin improves maternal glycemic control and reduces macrosomia risk without sustained differences in early childhood growth or adiposity. Consistent with recent meta-analyses and reviews published between 2023 and 2025 [3], current evidence supports favorable short-term safety of metformin in pregnancy, while long-term offspring outcomes remain insufficiently defined. Metformin provides glycemic control comparable to insulin in most women with gestational diabetes mellitus, although 10–46% require supplemental insulin, particularly those with higher baseline body mass index, elevated fasting glucose, or prior GDM. Randomized trials and meta-analyses demonstrate similar rates of neonatal morbidity, cesarean delivery, macrosomia, and perinatal mortality, while metformin is consistently associated with reduced gestational weight gain, lower maternal hypoglycemia, and in some studies decreased neonatal hypoglycemia. Reports of increased preterm birth are inconsistent. Despite placental transfer with fetal concentrations approximating maternal levels, current evidence shows no increased risk of congenital malformations, including with first-trimester exposure. Follow-up studies reveal no adverse differences in overall adiposity or metabolic parameters in early and mid-childhood, although modest variations in fat distribution and limited long-term data persist. In women with polycystic ovary syndrome, metformin use during pregnancy has been linked to reduced early pregnancy loss, lower progression to GDM, and decreased preterm birth without increased congenital anomalies. Overall, metformin demonstrates short-term equivalence to insulin with advantages in maternal weight and hypoglycemia risk; however, frequent need for adjunctive insulin and ongoing uncertainty regarding long-term offspring outcomes support its selective use following informed counseling [15].

A single-center observational cohort study of 1,269 women with gestational diabetes managed in routine practice (2007–2009) compared outcomes across diet alone (n = 371), insulin (n = 399), and metformin (n = 465; 249 monotherapy, 216 combined with insulin). Women requiring pharmacotherapy had significantly higher baseline body mass index and fasting glucose than those treated with diet alone ($p < 0.001$). Insulin therapy was associated with higher rates of cesarean delivery, preterm birth, customized large-for-gestational-age infants, neonatal admission, and need for intravenous dextrose compared with metformin or diet, while neonatal outcomes were similar between diet and metformin groups. Overall, metformin use was linked to fewer adverse outcomes than insulin, although baseline differences between groups may have contributed to these findings [6]. A prospective randomized controlled trial from Finland compared metformin with insulin in women with gestational diabetes requiring pharmacologic therapy after dietary measures failed. No significant differences were observed in rates of large-for-gestational-age infants, mean birth weight, cord arterial pH, or overall neonatal morbidity, indicating comparable efficacy in prevent-

ing fetal overgrowth. Approximately one third of women initially treated with metformin required supplemental insulin, particularly those with higher baseline body mass index, elevated fasting glucose, and earlier need for pharmacologic treatment. These findings support metformin as an effective alternative in selected patients—especially lean or moderately overweight women with later-onset gestational diabetes—whereas women with marked obesity and pronounced fasting hyperglycemia may benefit from primary insulin therapy [8].

A systematic review and meta-analysis of 28 randomized controlled trials (n = 3,976) examined long-term growth outcomes following intrauterine exposure to metformin versus insulin in pregnancies complicated by gestational diabetes mellitus. At birth, metformin exposure was associated with lower birth weight and ponderal index, along with reduced risks of macrosomia and large-for-gestational-age birth, while neonatal length and rates of small-for-gestational-age infants were similar between groups. Postnatal growth trajectories differed, with metformin-exposed children demonstrating greater weight in infancy (18–24 months) and higher body mass index in mid-childhood (5–9 years), although absolute weight differences were inconsistent. Limited evidence also indicated increased abdominal and visceral adiposity among metformin-exposed offspring. Overall, these findings suggest that metformin reduces neonatal overgrowth but may be associated with accelerated postnatal growth and higher body mass index later in childhood, underscoring the need for longitudinal studies to clarify potential cardiometabolic consequences of intrauterine metformin exposure [16]. Growing interest has focused on potential long-term offspring effects of metformin use in pregnancy. While short-term maternal and neonatal safety is well established, outcomes beyond infancy remain insufficiently characterized. Metformin crosses the placenta and, through mitochondrial complex I inhibition and AMP-activated protein kinase activation, may theoretically influence fetal energy regulation and developmental programming. Follow-up studies suggest modest alterations in early growth patterns, including slightly higher body mass index or increased subcutaneous adiposity, although findings are inconsistent. Most data demonstrate no meaningful differences in glucose metabolism, lipid profile, blood pressure, or neurodevelopment in mid-childhood, and meta-analyses indicate a small body mass index increase at 1–3 years that does not persist at older ages. The clinical significance of these early anthropometric differences remains uncertain. Interpretation is limited by study heterogeneity, attrition, and incomplete adjustment for confounders, with little evidence extending into adolescence or adulthood. Overall, current data do not indicate clinically significant long-term harm following intrauterine metformin exposure, but signals of altered growth trajectories underscore the need for well-powered prospective studies and harmonized individual patient data meta-analyses [10].

Conclusions. Available evidence from randomized and observational studies indicates that metformin used for gestational diabetes mellitus provides short-term perinatal outcomes comparable to insulin in women requiring pharmacologic therapy, without a consistent increase in serious neonatal morbidity or mortality. Metformin is associated with reduced gestational weight gain and lower maternal hypoglycemia risk, although many women require supplemental insulin, limiting its universal effectiveness as monotherapy. Because metformin crosses the placenta and reaches fetal concentrations similar to maternal levels, concerns persist regarding long-term metabolic programming. Follow-up data into early and mid-childhood generally show no consistent differences in overall adiposity, metabolic markers, or neurodevelopment, although modest variations in growth patterns and fat distribution have been reported with uncertain clinical relevance. Interpretation remains constrained by study heterogeneity, attrition, and limited long-term follow-up beyond childhood, preventing definitive conclusions on long-term safety. This uncertainty—rather than evidence of harm—underpins the cautious stance of several clinical communities, including Uzbekistan, where insulin remains the preferred pharmacologic therapy due to extensive clinical experience and absence of placental transfer, and where metformin lacks a registered pregnancy indication. Overall, metformin demonstrates clear short-term maternal and perinatal benefits, yet clarification of long-term offspring outcomes requires prospective research and structured longitudinal registries. Until such data are available, a conservative preference for insulin remains justified, while metformin use may be reasonable in settings supporting individualized decision-making, informed consent, and reliable long-term monitoring.

References:

1. American Diabetes Association. Standards of Medical Care in Diabetes — Section “Diabetes in Pregnancy”. Diabetes Care. 2023–2024.
2. Bellamy L., Casas J.P., Hingorani A.D., Williams D. Type 2 diabetes after gestational diabetes: a systematic review and meta-analysis. Lancet. PMID: 19465232 DOI: 10.1016/S0140-6736(09)60731-5
3. Cochrane Pregnancy and Childbirth Group. Oral anti-diabetic pharmacological therapies for treating women with gestational diabetes mellitus. Cochrane Database of Systematic Reviews. 2017.
4. Denice S Feig, Lois E Donovan, Bernard Zinman, J Johanna Sanchez, Elizabeth Asztalos. Metformin in women with type 2 diabetes in pregnancy (MiTy): a multicentre, international, randomised, placebo-controlled trial. PMID: 32946820 DOI: 10.1016/S2213-8587(20)30310-7
5. Denice S Feig, J Johanna Sanchez, Kellie E Murphy, Elizabeth Asztalos, Bernard Zinman. Outcomes in children of women with type 2 diabetes exposed to metformin versus placebo during pregnancy (MiTy Kids): a 24-month follow-up of the MiTy randomised controlled trial. PMID: 36746160 DOI: 10.1016/S2213-8587(23)00004-9
6. Goh JE, Sadler L, Rowan J. Metformin for gestational diabetes in routine clinical practice. Diabet Med. 2011 Sep;28(9):1082-7. doi: 10.1111/j.1464-5491.2011.03361.x. PMID: 21679232.
7. International Diabetes Federation. IDF Diabetes Atlas. 10th ed. 2021. PMID: 35914061
8. Ijäs H, Vääräsmäki M, Morin-Papunen L, Keravuo R, Ebeling T, Saarela T, Raudaskoski T. Metformin should be considered in the treatment of gestational diabetes: a prospective randomised study. BJOG. 2011 Jun;118(7):880-5. doi: 10.1111/j.1471-0528.2010.02763.x. Epub 2010 Nov 18. PMID: 21083860.
9. JANET A. ROWAN, FRACP A Trial in Progress: Gestational Diabetes. Treatment with metformin compared with insulin (the Metformin in Gestational Diabetes [MiG] trial) PMID: 17596475. DOI: 10.2337/dc07-s219
10. Mayadunne, T., Saadati, S., Asmelash, D., Mason, T., Vanky, E., Teede, H., & Mousa, A. (2025). Long-term effects of metformin on offspring health: A review of current evidence and future directions. Diabetes, obesity & metabolism, 27 Suppl 3(Suppl 3), 48–63. https://doi.org/10.1111/dom.16418
11. Recent systematic and narrative reviews on metformin in pregnancy (2023–2025). Wiley Online Library (selected publications).
12. Rowan J.A., Hague W.M., Gao W., et al. Metformin versus Insulin for the Treatment of Gestational Diabetes. PMID: 18463376 DOI: 10.1056/NEJMoa0707193
13. Janet A Rowan 1, Elaine C Rush, Victor Obolonkin, Malcolm Battin, Trecia Wouldes, William M Hague. Metformin in gestational diabetes: the offspring follow-up (MiG TOFU): body composition at 2 years of age. PMID: 21949222 PMCID: PMC3177748 DOI: 10.2337/dc11-0660
14. Janet A Rowan, Elaine C Rush, Lindsay D Plank, Jun Lu, Victor Obolonkin, Suzette Coat, William M Hague Metformin in gestational diabetes: the offspring follow-up (MiG TOFU): body composition and metabolic outcomes at 7-9 years of age. PMID: 29682291 PMCID: PMC5905785 DOI: 10.1136/bmjdrc-2017-000456
15. Singh, A. K., & Singh, R. (2015). Metformin in gestational diabetes: An emerging contender. Indian journal of endocrinology and metabolism, 19(2), 236–244. https://doi.org/10.4103/2230-8210.149317
16. Tarry-Adkins JL, Aiken CE, Ozanne SE. Neonatal, infant, and childhood growth following metformin versus insulin treatment for gestational diabetes: A systematic review and meta-analysis. PLoS Med. 2019 Aug 6;16 (8):e1002848. doi: 10.1371/journal.pmed.1002848. PMID: 31386659; PMCID: PMC6684046.
17. Vera Tocci, Maria Mirabelli, Alessandro Salatino, Luciana Sicilia, Stefania Giuliano, Francesco S Brunetti, Eusebio Chiefari, Giovambattista De Sarro, Daniela P Foti, Antonio Brunetti. Metformin in Gestational Diabetes Mellitus: To Use or Not to Use, That Is the Question. PMID: 37765126 PMCID: PMC10537239 DOI: 10.3390/ph16091318