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Noncoding RNAs as biomarkers of gestational diabetes mellitus

Jovana Stevanović, Zorana Dobrijević
University of Belgrade – Institute for the Application of Nuclear Energy, Belgrade, Serbia

Correspondence: zorana.dobrijevic@inep.co.rs

Abstract

Non-coding RNAs (ncRNAs), including microRNAs (miRNAs), long non-coding RNAs (lncRNAs), and circular RNAs (circRNAs), show potential to serve as biomarkers of metabolic disorders, oxidative stress and inflammatory status, and thus may have diagnostic and prognostic significance in gestational diabetes mellitus (GDM). Aberrant expression of specific ncRNAs has been identified in placental tissue, plasma, serum, extracellular vesicles, peripheral blood mononuclear cells (PBMCs), as well as in other maternal and fetal sources. These ncRNAs exhibit high stability in circulation and can reflect underlying molecular alterations, making them promising non-invasive biomarkers for diagnosis, prognosis, and disease monitoring. Therefore, this article will review the major findings of GDM-related ncRNAs and highlight their potential role in improving clinical outcomes through biomarker-based approaches.

Keywords: gestational diabetes mellitus, miRNA, lncRNA, circRNA, liquid biopsy

Nekodirajuće RNK kao biomarkeri gestacijskog dijabetesa

Jovana Stevanović, Zorana Dobrijević
Univerzitet u Beogradu – Institut za primenu nuklearne energije INEP, Beograd, Srbija

Kontakt: zorana.dobrijevic@inep.co.rs

Apstrakt

Nekodirajuće RNK (nkRNK), uključujući mikroRNK, duge nekodirajuće RNK i kružne RNK, pokazuju potencijal da služe kao biomarkeri metaboličkih poremećaja, oksidativnog stresa i inflamatornog statusa, i stoga mogu imati dijagnostički i prognostički značaj kod gestacijskog dijabetesa melitusa (GDM). Aberantna ekspresija specifičnih nkRNK je identifikovana u tkivu placente, plazmi, serumu, ekstracelularnim vezikulama, mononuklearnim ćelijama periferne krvi, kao i u drugim izvorima poreklom od majke ili fetusa. Ove nkRNK pokazuju visoku stabilnost u cirkulaciji i mogu odražavati osnovne molekularne promene, što ih čini obećavajućim neinvazivnim biomarkerima za dijagnozu, prognozu i praćenje bolesti. Stoga će ovaj članak pregledati glavna saznanja o nkRNK povezanih sa GDM i istaći njihovu potencijalnu ulogu u poboljšanju kliničkih ishoda kroz pristupe zasnovane na biomarkerima.

Ključne reči: gestacijski dijabetes melitus, mikroRNK, duge nekodirajuće RNK, cirkularne RNK, tečna biopsija

Introduction

Gestational diabetes mellitus (GDM) is recognized as one of the three most common types of diabetes by the International Diabetes Federation. It is defined as a pathological condition of glucose intolerance, first recognized during pregnancy, and represents one of the most frequent metabolic complications affecting pregnant women worldwide, with an average prevalence of approximately 14% of pregnancies [1].

If left undiagnosed or poorly managed, GDM can lead to serious health risks for both the mother and the offspring. It is associated with a wide range of both short- and long-term complications, including preeclampsia, fetal macrosomia, neonatal hypoglycemia, shoulder dystocia, preterm labor, cesarean delivery, and fetal mortality. Children of mothers with GDM are at significantly increased risk of developing type 2 diabetes mellitus (T2DM), obesity, and cardiovascular diseases later in life. Furthermore, women diagnosed with GDM have a significantly elevated risk of developing T2DM postpartum, and female infants are also at increased risk of developing GDM during their own pregnancies [2].

Despite the well-established consequences of GDM, its pathogenesis remains incompletely understood. It is thought to arise from a combination of genetic, epigenetic, environmental, and neurohormonal factors that contribute to insulin resistance, hyperglycemia, and β-cell dysfunction during pregnancy. There is also evidence that (glyco)oxidative stress, low-level chronic inflammation, adipose tissue expandability, and placental factors contribute to the pathology of GDM [3].

Currently, GDM is most commonly diagnosed using the oral glucose tolerance test (OGTT), performed between 24 and 28 weeks of gestation, although there is a lack of consensus on the use of diagnostic criteria or protocols. Moreover, the OGTT is time-consuming and may cause significant discomfort, vomiting, or even more serious consequences in women with pre-existing glucose intolerance or undiagnosed diabetes [4, 5]. Since this test has limited predictive value and currently there are no GDM biomarkers that are in clinical use, there is a growing need for novel biomarkers which could aid in diagnosing, predicting, or monitoring GDM, resulting in a reduced risk of maternal and fetal complications.

In recent years, the focus of biomarker research has moved towards molecules that possess these three types of clinically relevant properties and are also accessible through non-invasive sampling. Circulating biomarkers, detectable in many biological fluids, including plasma, serum, urine, extracellular vesicles, or in blood cells, have gained increasing attention due to their ability to be monitored throughout pregnancy. Among them, non-coding RNAs (ncRNAs), particularly microRNAs (miRNAs), long noncoding RNAs (lncRNAs), and circular RNAs (circRNAs), have emerged as promising candidates [6].

Non-coding RNAs represent a diverse class of functional RNA molecules that, generally, do not encode proteins but instead regulate gene expression at epigenetic, transcriptional and post-transcriptional levels. These ncRNA species have been implicated in key physiological and pathological processes such as glucose metabolism, insulin signalling, inflammation, oxidative stress, and placental development, all of which are closely linked to the pathophysiology of GDM [6, 7].

NcRNAs possess several features that make them attractive as potential biomarkers, including a high level of stability in many body fluids, within extracellular vesicles, associated with RNA-binding proteins, or, in the case of circRNAs, due to circular structure, which protects them from RNase degradation [8]. Additionally, they have tissue- and disease-specific expression patterns and act through direct involvement in regulatory pathways. Furthermore, the desirable features of ncRNAs as biomarker candidates are non-invasive sample acquisition through liquid biopsy, detectability early in the disease, which is extremely im-

portant for timely diagnosis, and the availability of sensitive molecular techniques, such as next generation sequencing (NGS), microarray analysis, digital droplet PCR (ddPCR) and quantitative real-time PCR (qRT-PCR), which allows accurate quantification [9].

In the context of GDM, numerous studies have reported altered expression of ncRNAs in maternal plasma, serum, placental tissue, peripheral blood mononuclear cells (PBMCs), as well as in other maternal and fetal biological sources [7]. NcRNAs, especially in combination with clinical, metabolic, and redox-status parameters, may serve as valuable components of novel advanced algorithms constructed for risk prediction, early diagnosis, disease monitoring or prediction of adverse outcomes and treatment strategy guidance. Furthermore, understanding the regulatory networks mediated by ncRNAs may reveal novel therapeutic targets and provide insight into mechanisms through which dysregulated ncRNAs participate in the development of metabolic diseases [7].

In this review, we summarize the current evidence regarding the role of circulating ncRNAs in the pathophysiology of GDM and evaluate their potential utility as biomarkers for detection, prediction, and disease monitoring. By highlighting recent findings, we aim to contribute to the growing body of knowledge that supports the integration of ncRNA profiling into clinical practice.

MiRNAs

MiRNAs are highly conserved non-coding RNA sequences that are expressed in plants, animals, and some viruses [10]. They play an essential role in the post-transcriptional regulation of gene expression, which is achieved through endonucleolytic degradation of target RNAs, destabilization of target mRNAs, translational repression, or, in some cases, translational activation [11]. This regulation is mediated by a phenomenon known as RNA interference (RNAi), a natural mechanism that also protects cells from the harmful propagation of viruses and transposons [12].

The interaction between miRNA and the target mRNA is achieved by base pairing. The most miRNA-binding sites (MREs) are located in the 3 -untranslated region (3'-UTR) of mRNA. Target recognition is generally determined by the complementarity between the bases 2 to 8 of the miRNA, called the seed sequence, and the 3'-UTR of mRNA. One mRNA contains MREs for a large number of the same and different miRNAs, and one miRNA can regulate hundreds of target mRNAs, which indicates a complex post-transcriptional regulation by miRNAs that occur within regulatory networks [13].

MiRNAs play a pivotal role in normal development and are involved in a variety of biological processes. Apart from their role in cells in which their biogenesis occurs, they also serve as signaling molecules, mediating cell-to-cell communication [14]. Aberrant expression of miRNAs has been associated with numerous human diseases, including cancer, immunological disorders, and developmental abnormalities. Since miRNAs are secreted into extracellular fluids, they are also recognized as potential biomarkers for a wide range of diseases [15].

One of the most remarkable features of miRNAs is their stability in body fluids, such as blood, urine, saliva, and amniotic fluid. Unlike most RNA molecules, which are rapidly degraded by RNases, circulating miRNAs are resistant to enzymatic degradation due to their association with RNA-binding proteins (miRISC/AGO2 complexes) and incorporation in lipoprotein complexes, including high-density lipoproteins (HDL) or low-density lipoproteins (LDL), or extracellular vesicles (EVs) [16]. In the context of biomarker research and mechanistic studies on between-cell communication, EVs are particularly interesting biological entity, since they carry bioactive molecules such as proteins, lipids, and RNAs, including miRNAs, and me-

diate intercellular communication by carrying their cargo to recipient cells. Additionally, their cargo reflects the biological processes active in the cells which produce them [17]. Therefore, miRNAs enveloped in EVs, which are selectively incorporated and are among the most extensively analyzed RNA species enriched in EVs, may represent promising biomarker candidates. In GDM, EVs miRNA content reflects the physiological state of the affected maternal tissues and the placenta, while EV-associated miRNAs also modulate gene expression in distant cells, influencing insulin sensitivity, immune responses, and endothelial function [18, 19]. Importantly, placenta-derived EVs are released in increasing amounts throughout pregnancy and can be detected in maternal circulation, making their miRNA content a promising source of non-invasive biomarkers for early detection and monitoring of GDM [20].

Multiple studies have reported tissue-specific dysregulation of miRNAs in GDM, including placenta-specific changes [21, 22]. Dysregulation of a variety of miRNAs is linked to altered trophoblast proliferation, migration, invasion, and mitochondrial function [23, 24]. Specific miRNAs in pancreatic islets are involved in insulin gene expression and β-cell mass regulation [25]. These changes contribute to inadequate insulin secretion in response to increased insulin demand during pregnancy. Furthermore, altered levels of various miRNAs in adipose tissue are affecting adipocyte differentiation, lipid metabolism, and inflammatory cytokine production [26]. Taken together, these findings supported the concept that miRNAs are not just byproducts but active regulators in the pathophysiology of GDM and that miRNAs released by GDM-relevant tissues may change circulating miRNA profiles which would reflect the changes in the cells of origin.

Despite their promise as biomarkers, results from circulating miRNA studies in GDM are heterogeneous and difficult to compare across different investigations. Several factors contribute to this variability. First, miRNA expression is influenced by maternal age, BMI, ethnicity, lifestyle, diet, and pre-existing metabolic conditions [27], which can confound the results if not properly matched or adjusted. Additionally, miRNA profiles change dynamically throughout pregnancy, reflecting ongoing physiological adaptations in both maternal and placental tissues [28]. Namely, pregnancy is a highly dynamic physiological state, and miRNA expression changes across trimesters to accommodate maternal-fetal adaptation. Studies have shown that some miRNAs, especially placenta-specific miRNAs, like those from the C19MC cluster, are progressively upregulated during pregnancy and hyperexpressed in the third trimester [16]. Therefore, the timing of sample collection is critical when evaluating miRNAs as biomarkers, and results obtained at different gestational stages may not be directly comparable. Differences in sample preference (serum, plasma, whole blood, or extracellular vesicle-enriched fractions) can significantly affect miRNA content and stability. Furthermore, variability in sample collection procedure, processing time, and storage temperature can alter miRNA integrity [29]. Finally, a lack of universal endogenous controls for circulating miRNA quantification leads to inconsistent normalization methods, further contributing to inter-study variation [30].

Considering these obstructions, interpreting and comparing findings across studies remains difficult. Nevertheless, several investigations have identified matching candidate circulating miRNAs associated with GDM, or their main findings referred to members of the same miRNA families. Table 1 provides an overview of miRNA-oriented studies on GDM biomarkers [31-75], illustrating both their findings and methodological differences.

One of the commonly reported dysregulated miRNA in plasma or serum samples of GDM patients was miR-16-5p. However, these results need to be taken with caution, since miR-16-5p is highly expressed in erythrocytes and the quantity in blood fractions is severely affected by hemolysis, for which reason it is generally avoided as a potentially usable biomarker [76]. Other miRNAs were rarely reported as upregulated or downregulated in more than a single study. For instance, miR-146a-5p was reported as upregulated in two

studies on GDM in similar pregnancy stage, but with different sample types: plasma, serum-derived EVs and PBMCs [55, 67]. Similarly, miR-423-5p was among the upregulated miRNAs in plasma and plasma-derived EVs from GDM patients [55, 65], while an upregulation was also detected for miR-210-3p, miR-195-5p, miR-520h, miR-23a-3p and miR-342-3p in serum and plasma [39, 46, 49, 53, 55, 57, 63]. When it comes to miR-17-5p, higher levels of expression in plasma samples of GDM patients were reported in two studies [50, 51], while for other two members of the same family, miR-19a-3p and miR-19b-3p, two studies supported an upregulation in GDM [35, 50]. As for miR-330-3p and miR-223-3p, there were three or more reports on their upregulation in serum or plasma samples [34, 37, 40, 52, 53, 57, 77].

Besides inconsistencies regarding the panels of dysregulated miRNA hits, the data presented in the table indicate that certain miRNAs exhibited increased expression in pregnant women with GDM compared to normoglycemic controls in some studies, while in others they show decreased expression. For instance, miR-132-3p, miR-20a-5p, miR-122-5p, miR-29a-3p and miR-222-3p were reported as both downregulated and upregulated miRNA in GDM [31, 33, 34, 36, 45, 55, 50, 51, 63, 65]. In the case of miR-20a-5p, which belong to the same miRNA family as miR-17-5p and originate from a miRNA gene cluster miR-17~92, the observed differences may be caused by sample selection, since matching results were found for two studies that utilized plasma as a source of analyzed miRNAs [50, 51]. The direction of the change in the expression of specific miRNAs varied even between subgroups of samples within the same study, such was the case with miR-125b-5p and miR-183-5p in different pregnancy trimesters [32]. Several of the reported dysregulated miRNAs belong to the same family, which may be explained by similarities in their target mRNA pools and the common regulation of gene expression for those originating from clustered genes. Most prominent are the reports on the dysregulation of the members of miR-17~92 family: miR-17-5p, miR-18a-5p, miR-19a-3p, miR-19b-3p, miR-20a-5p, miR-92a-3p, which are mostly reported as upregulated in GDM (Table 1).

The interpretation of circulating miRNA profiles in GDM is highly dependent on the type of biological sample analyzed, since miRNA content and stability vary between these sources. Despite the increasing number of studies, blood cells are still rarely exploited as a source of circulating miRNAs. However, there have been studies GDM biomarkers focusing on miRNAs from whole blood or PBMCs, which demonstrated the potential diagnostic and/or prognostic significance of several candidate miRNAs, as well as their role as indicators of metabolic and redox status in hyperglycemic pregnancy [67-75, 78]. Although the selection of the most adequate type of biological sample for biomarker analysis is an important issue, studies that compare different sources of potentially relevant miRNAs in the same cohorts are rare [67, 79]. One such study is ours, in which we analyzed matched samples of EVs and PBMCs obtained from the same individuals [67]. This approach allowed us to directly compare miRNA expression patterns across two biological sources within the same physiological context. Notably, we observed consistent trends in the expression of selected miRNAs, including miR-146a-5p and miR-21-5p, in both PBMC- and EV-derived fractions [67]. The agreement in direction of change between these paired sources further reinforces the potential of these miRNAs as biomarkers of GDM.

Beside the necessity of validating the diagnostic utility of potential GDM biomarker miRNAs in large prospective studies, another important issue is determining the major factors which may affect the expression of candidate miRNAs, such as age of study participants, their life habits and previously mentioned pregnancy and sample characteristics. For certain miRNAs reported as dysregulated in GDM, previous studies have reported a substantial influence of common genetic variants on their biogenesis, or even on the sequence of mature miRNA molecules [80-83], which may interfere with quantification and results interpretation. Therefore, integration of miRNA quantification results with genetic data, clinical characteristics and other potentially relevant sources of bias should be considered during GDM biomarker evaluation.

Table 1. Summary of dysregulated miRNAs in GDM

Authors	Year	No. GDM/ Controls	Gestation week	Method	qPCR normalization	Up-regulated in GDM	Down-regulated in GDM	Reference
Serum								
Zhao et al.	2011	24/24	16–19	TLDA, qRT-PCR	cel-miR-39-3p		hsa-miR-132-3p, hsa-miR-29a-3p, hsa-miR-222-3p	[31]
Lamadrid-Romero et al.	2017	14/27	first trimester	qRT-PCR	cel-miR-39-3p	hsa-miR-125b-5p		[32]
Lamadrid-Romero et al.	2017	26/26	second trimester	qRT-PCR	cel-miR-39-3p		hsa-miR-125b-5p	[32]
Lamadrid-Romero et al.	2017	27/21	third trimester	qRT-PCR	cel-miR-39-3p		hsa-miR-125b-5p	[32]
Lamadrid-Romero et al.	2017	13/12	first trimester	qRT-PCR	cel-miR-39-3p	hsa-miR-183-5p, hsa-miR-200b-3p, hsa-miR-1290		[32]
Lamadrid-Romero et al.	2017	24/24	second trimester	qRT-PCR	cel-miR-39-3p	hsa-miR-183-5p,	hsa-miR-128-5p	[32]
Lamadrid-Romero et al.	2017	20/16	third trimester	qRT-PCR	cel-miR-39-3p		hsa-miR-183-5p, hsa-miR-200b-3p	[32]
Pheiffer et al.	2018	28/53	13-31	qRT-PCR	cel-miR-39-3p		hsa-miR-20a-5p, hsa-miR-222-3p	[33]
Martínez-Ibarra et al.	2019	18/22	second trimester	qRT-PCR	hsa-miR-454	hsa-miR-9-5p, hsa-miR-29a-3p, hsa-miR-330-3p		[34]
Wang et al.	2019	100/100	24-28	qRT-PCR	RNU6	hsa-miR-19a-3p, hsa-miR-19b-3p		[35]
Zhou et al.	2019	108/50	24-28	qRT-PCR	RNU6	hsa-miR-132-3p		[36]
Abdeltawab et al.	2020	109/103	third trimester	qRT-PCR	cel-miR-39-3p	hsa-miR-223-3p		[37]
Feng et al.	2020	12/12	NA	qRT-PCR	RNU6	hsa-miR-33a-5p		[38]
Wang et al.	2020	102/102	25	qRT-PCR	RNU6	hsa-miR-195-5p		[39]
Xiao et al.	2020	30/10	16-28	qRT-PCR	RNU6	hsa-miR-330-3p		[40]
Hua et al.	2021	30/38	third trimester	qRT-PCR	RNU6	hsa-miR-377-3p		[41]
Li et al.	2021	93/93	24–28	qRT-PCR	RNU6		hsa-miR-497-5p	[42]
Liu et al.	2021	110/78	24-28	qRT-PCR	RNU6	hsa-miR-1323-5p		[43]
Shen et al.	2021	25/30	NA	qRT-PCR	RNU6	hsa-miR-181d-5p		[44]
Sørensen et al.	2021	82/41	<20	qRT-PCR	RNU6, ath-miR-159, cel-miR-39-3p	hsa-miR-16-5p, hsa-miR-29a-3p, hsa-miR-134-5p		[45]
Wen et al.	2021	32/48	second/third trimester	qRT-PCR	RNU6	hsa-miR-520h		[46]
Juchnicka et al.	2022	24/24	9-12	qRT-PCR	miR-103a-3p	hsa-miR-16-5p, hsa-miR-142-3p, hsa-miR-144-3p		[47]
Li et al.	2022	118/65	23-28	qRT-PCR	RNU6	hsa-miR-518		[48]
Jamalpour et al.	2023	24/24	first trimester	PCR array	SNORD61, SNORD68, SNORD72, SNORD95, and SNORD96A	hsa-miR-193a, hsa-miR-21-5p, hsa-miR-23a-3p, hsa-miR-361	hsa-miR-130a-3p	[49]
Jamalpour et al.	2023	24/24	second trimester	PCR array	SNORD61, SNORD68, SNORD72, SNORD95, and SNORD96A	hsa-let-7i-5p, hsa-miR-126, hsa-miR-129	hsa-miR-125, hsa-miR-129–2, hsa-miR-130a, hsa-miR-34, hsa-miR-375	[49]
Jamalpour et al.	2023	24/24	third trimester	PCR array	SNORD61, SNORD68, SNORD72, SNORD95, and SNORD96A	hsa-let-7e, hsa-miR-107, hsa-miR-361, hsa-miR-370	hsa-miR-125, hsa-miR-129, hsa-miR-130a	[49]
Plasma								
Zhu et al.	2015	10/10	16–19	NGS, qRT-PCR	miR-221	hsa-miR-16-5p, hsa-miR-17-5p, hsa-miR-19a-3p, hsa-miR-19b-3p, hsa-miR-20a-5p		[50]
Cao et al.	2017	85/72	24–28	qRT-PCR	cel-miR-39, cel-miR-54, cel-miR-238	hsa-miR-16-5p, hsa-miR-17-5p, hsa-miR-20a-5p		[51]
Sebastiani et al.	2017	21/10	24-33	TAC, qRT-PCR	miR-320, miR-374a	hsa-miR-330-3p, hsa-miR-483-5p	hsa-miR-548c-3p , hsa-miR-532-3p	[52]
Wander et al.	2017	36/80	7-23	qRT-PCR	cel-miR-39-3p	hsa-miR-155-5p, hsa-miR-21-3p, hsa-miR-146b-5p, hsa-miR-210-3p, hsa-miR-223-3p, hsa-miR-517-5p		[53]
Peng et al.	2018	11/12	24-28	qRT-PCR	RNU6	hsa-miR-137-3p		[54]
Tagoma et al.	2018	13/9	23-31	qRT-PCR, qRT-PCR arrays	cel-miR-39-3p	Let-7e-5p, let-7g-5p, hsa-miR-100-5p, hsa-miR-101-3p, hsa-miR-146a-5p, hsa-miR-18a-5p, hsa-miR-195-5p, hsa-miR-222-3p, hsa-miR-23b-3p, hsa-miR-30b-5p, hsa-miR-30c-5p, hsa-miR-30d-5p, hsa-miR-342-3p, hsa-miR-423-5p, hsa-miR-92a-3p		[55]

Balci et al.	2020	30/30	24–28	qRT-PCR using 96.96 Dynamic Array IFCs	global mean	hsa-miR-7-5p		[56]
Yoffe et al.	2020	23/20	9-12	qRT-PCR		hsa-miR-23a-3p, hsa-miR-223-3p		[57]
Wang et al.	2021	53/46	37-40	microarray, qRT-PCR	cel-miR-39-3p		hsa-miR-574-5p, hsa-miR-3135b	[58]
Yu et al.	2021	123/123	24-28	qRT-PCR	RNU6		hsa-miR-96-5p	[59]
Filardi et al.	2022	12/12	third trimester	qRT-PCR	RNU6, ath-miR-159a	hsa-miR-222-3p, hsa-miR-409-3p		[6]
Liu et al.	2022	30/30	39	qRT-PCR	RNU6		hsa-miR-143-3p	[60]
Leng et al.	2023	60/60	NA	qRT-PCR	RNU6		hsa-miR-138	[61]
EVs								
Nair et al.	2018	12/12	>37	miRNA sequencing, qRT-PCR	RNU6-2	hsa-miR-125a-3p, hsa-miR-224-5p, hsa-miR-584-5p, hsa-miR-186-5p, hsa-miR-22-3p, hsa-miR-99b-5p, hsa-miR-433-3p, hsa-miR-197-3p, hsa-miR423-3p	hsa-miR-208a-3p, hsa-miR-335-5p, hsa-miR-451a, hsa-miR-145-3p, hsa-miR-369-3p, hsa-miR-483-3p, hsa-miR-203a-3b, hsa-miR-574-3p, hsa-miR-144-3p, hsa-miR-6795-5p, hsa-miR-550a-3-3p, hsa-miR-411-5p, hsa-miR-140-3p	[62]
Gillet et al.	2019	23/46	6-15	qRT-PCR	cel-miR-39-3p	hsa-miR–122-5p, hsa-miR–132-3p, hsa-miR–1323-5p, hsa-miR–136-5p, hsa-miR–182-3p, hsa-miR–210-3p, hsa-miR–29a-3p, hsa-miR–29b-3p, hsa-miR–342-3p, hsa-miR-520h		[63]
Zhang et al.	2021	57/61	26-40	qRT-PCR	RNU6	hsa-miR-144-3p	hsa-miR-125b-5p	[64]
Ye et al.	2022	102/101	24-28	qRT-PCR	cel-miR-39–3p	hsa-miR-423–5p	hsa-miR-122–5p, hsa-miR-148a-3p, hsa-miR-192–5p, hsa-miR-99a-5p	[65]
Zhang et al.	2023	30/30	24-28	qRT-PCR	RNU6	hsa-miR-135a-5p		[66]
Stevanović et al.	2025	50/50	24-28	qRT-PCR	miR-191-5p	hsa-miR-146a-5p, hsa-miR-21-5p		[67]
Blood/PBMC								
He et al.	2017	20/20	NA	qRT-PCR	RNU6	hsa-miR-494-3p		[68]
Xu et al.	2017	25/25	NA	qRT-PCR	NA	hsa-miR-503		[69]
Stirm et al.	2018	15/15	24-32	qRT-PCR	RNU6	hsa-miRNA-340		[70]
Bian et al.	2022	30/30	23-27	qRT-PCR	RNU6	hsa-miR-296-3p		[71]
Hocaoglu et al.	2019	19/28	third trimester	qRT-PCR	RNU6		hsa-miR-21-3p	[72]
Hu et al.	2020	35/35	24-28	qRT-PCR	RNU6		hsa-miR-4646-5p hsa-miR-5196-5p	[73]
Zhang et al.	2020	30/30	NA	qRT-PCR	RNU6	hsa-miR-770-5p		[74]
Radojičić et al.	2022	42/34	24-30	qRT-PCR	RNU6-1	hsa-miR-27a-3p		[75]
Stevanović et al.	2025	50/50	24-28	qRT-PCR	miR-191-5p	hsa-miR-146a-5p, hsa-miR-21-5p		[67]

Abbreviations: TLDA - TaqMan Low-Density Array; qRT-PCR - quantitative Reverse Transcription Polymerase Chain Reaction; NGS - Next-Generation Sequencing; TAC - TaqMan Array Card; miRNA seq - miRNA sequencing; NA – not avalilable

LncRNAs

Long non-coding RNAs (lncRNAs) are a diverse class of transcripts longer than 200 nucleotides, that generally lack the protein-coding potential, but have critical regulatory roles in gene expression at multiple levels. They can act as molecular scaffolds, signals, guides, decoys, or sponges for miRNAs, thereby modulating various signaling pathways and cellular processes [84]. LncRNAs exhibit highly specific spatial and temporal expression patterns, often characteristic of certain cell types or developmental stages, suggesting their involvement in finely tuned physiological and pathological processes, due to their exceptionally regulated expression [85].

Table 2. Summary of dysregulated lncRNAs in GDM

Authors	Year	No. GDM/ Controls	Gestation week	Method	qPCR normalization	Up-regulated in GDM	Down-regulated in GDM	References
Serum								
Zhang et al.	2017	50/47	24-28	qRT-PCR	β-actin	*MALAT1*		[94]
Li et al.	2021	93/93	24-28	qRT-PCR	*GAPDH*	*XIST*		[42]
Su et al.	2021	99/98	25-29	qRT-PCR	*GAPDH*	*HOTAIR*		[95]
Huang	2024	70/50	24-28	qRT-PCR	*GAPDH*	*DLX6-AS1*		[96]
Chen et al.	2025	118/112	24-28	qRT-PCR	*GAPDH*	*HCG18*		[97]
Ma et al.	2025	128/125	24-28	qRT-PCR	*GAPDH*	*SNHG14*		[98]
Plasma								
Li et al.	2021	60/60	24–28	qRT-PCR	*GAPDH*		*SNHG17*	[99]
Li et al.	2021	2/3	24–40	qRT-PCR		*ERMP1*, *TSPAN32*, *MRPL38*, *RPL13P5*		[90]
Ran et al.	2021	52/164	0.9-2.8 months	qRT-PCR	18S rRNA	*SOX2OT*		[100]
Jiang et al.	2023	56/58	24–28	LncRNA microarray, qRT-PCR	*GAPDH*	*NONHSAT054669.2*, ENST00000525337		[101]
Jiang et al.	2023	27/45	12–14	LncRNA microarray, qRT-PCR	*GAPDH*	*NONHSAT054669.2*, ENST00000525337		[101]
Leng et al.	2023	60/60	NA	qRT-PCR	*GAPDH*	*UCA1*		[61]
Zhao et al.	2023	34/186	resampling every month (from 1M)	qRT-PCR	18S rRNA	*HCP5*		[102]
Blood/PBMC								
Zhang	2019		28	qRT-PCR	*GAPDH*	*MEG3*		[103]
Li et al.	2021	25/19	24-28	qRT-PCR		*RPL13P5*		[104]
Bian et al.	2022	30/30	23-27	qRT-PCR	*GAPDH*	*MEG8*		[105]
Stevanović et al.	2024	50/50	24-30	qRT-PCR	*GAPDH*		*MALAT1*, *H19*	[106]

Abbreviations: TLDA - TaqMan Low-Density Array; qRT-PCR - quantitative Reverse Transcription Polymerase Chain Reaction; NGS -Next-Generation Sequencing; TAC - TaqMan Array Card; miRNA seq-miRNA sequencing; NA – not available.

In the field of metabolic disorders such as GDM, lncRNAs are of particular interest due to their regulatory roles in glucose and lipid metabolism, insulin signaling, and inflammatory pathways [6]. Their dysregulation has been reported in various tissues relevant to diabetes pathophysiology, including pancreatic islets, adipose tissue, muscles, liver, and placenta, emphasizing their involvement in both maternal and fetal physiology [86-89]. In addition, lncRNAs are important regulators of inflammation and oxidative stress, both of which are hallmarks of GDM pathophysiology. Aberrant lncRNA expression can increase proinflammatory cytokine production and disrupt redox balance, thereby exacerbating metabolic dysfunction [7, 90].

The placenta, a key organ in pregnancy, is a critical site where lncRNAs exert their effects. Placenta-derived lncRNAs regulate trophoblast proliferation, invasion, and apoptosis, processes essential for proper placental development and nutrient transfer [3]. Dysregulated placental lncRNAs have been linked to abnormal angiogenesis and impaired fetal growth, reflecting their dual impact on maternal and fetal outcomes [91]. Furthermore, lncRNAs have been implicated in β-cell dysfunction, a contributing factor to glucose intolerance in GDM. Certain lncRNAs negatively affect insulin biosynthesis and secretion, while others interfere with β-cell survival under conditions of glycotoxicity and lipotoxicity [6]. Collectively, the dysregulation of lncRNAs in GDM points to their involvement in a network of interrelated processes: insulin resistance, inflammation, oxidative stress, placental dysfunction, and β-cell function impairment [3]. Due to a fact that changes in the expression of specific lncRNAs in placental tissue were followed by their up- or downregulation in body fluids, lncRNAs are not only recognized as mechanistic players in disease pathogenesis, but also as potential biomarkers for early diagnosis and prognosis, as well as possible therapeutic targets.

In contrast to miRNAs, for which numerous studies have reported aberrant expression in GDM [31-75], research on lncRNAs remains relatively rare or non-existing, especially for studies investigating the expression of lncRNAs derived from EVs. One of the main reasons for this scarcity is technical complexity associated with the isolation and analysis of lncRNAs from EVs, serum and plasma. These RNAs are typically expressed at low levels, may be fragmented, and require highly sensitive and standardized methods for reliable detection and quantification [92, 93]. Even when such studies are conducted, there are inconsistencies in the reported expression patterns across different investigations [42, 61, 90, 94-106]. For instance, *RPL13P5* is a rarely reported upregulated lncRNA within more than a one study on this topic. A notable example is also our study on the expression of *MALAT1*, for which we showed a downregulation in PBMCs obtained from GDM patients [106], while a previous study reported upregulation [94]. However, these two studies used different biological sources of lncRNAs for the analysis, while the sampling period during pregnancy also differed [94, 106]. Despite the contradictory results obtained in these two studies, both demonstrate a correlation between *MALAT1* and *H19*, which may indicate the presence of common stimuli inhibiting the expression of these lncRNAs. Another example of inconsistencies is our result which did not replicate previously observed upregulation of *MEG3* in blood samples of GDM patients [103]. However, this previous study [103] included merely 20 participants and analyzed whole blood instead of PBMCs, which were used in our recent study [106].

Apart from their diagnostic role, many of the reported dysregulated lncRNAs displayed a correlation between the level of expression and the values of glycemic or lipid status parameters [42, 61, 95-98, 101, 104, 106]. Additionally, a correlation was detected with the parameters of redox status and zinc concentration, all reported as altered in GDM and indicative of pregnancy outcome [106, 107]. Furthermore, several lncRNA hits from GDM studies demonstrated association with the adverse pregnancy outcomes [61, 98, 100].

CircRNA

CircRNAs are a class of endogenous non-coding RNA molecules characterized by a covalently closed loop structure without 5'-3' polarity and a poly-A tail. Typically, circRNAs originate from pre-mRNA transcripts through a "back-splicing" process and contain circularized exonic sequences [108]. Their high chemical stability, caused by specific structure, as well as the tissue specificity, represent desirable features of potentially reliable disease biomarkers. However, these ncRNAs are often expressed at low level, which associate with low abundance in biological samples, while their accurate and sensitive quantification has proved challenging due to their circular structure, lack of poly-A tail, homology with parental mRNA and technical limitations of quantification methods [109].

The most extensively investigated mode of action of circRNAs is their sponging activity toward miRNAs. However, regarding biomarker significance, this type of non-coding RNA molecules is much less analyzed, compared to circulatory miRNAs. Therefore, there is a limited data on the functional significance and biomarker properties of circRNA molecules in GDM [110]. In a relatively recent study, which was multicentric and included a relatively large group of pregnant women in different stages of pregnancy, hsa_circ_0031560 and hsa_circ_0000793 were identified and validated as potentially reliable early biomarkers for GDM. Serum-derived circRNAs from this study also proved predictive for the adverse pregnancy outcome [111]. In an earlier study, plasma samples of GDM patients and matched controls were used for circRNA profiling by NGS, which demonstrated an upregulation of hsa_circ_0008285 and downregulation of hsa_circ_0001173 in

GDM [112]. Furthermore, the expression of these two circRNAs correlated with the values of various lipid and glycemic status parameters, while the knockout of hsa_circ_0008285 counteracted the increase in proliferation, migration and invasion induced by hyperglycemia in trophoblast cell line HTR-8/SVneo [112]. Previously, this circRNA (also known as circCDYL) was reported as dysregulated in different malignant diseases and cardiovascular disorders [113]. Another study, focused on plasma EV-derived circRNAs, identified hsa_circRNA_0039480 and hsa_circRNA_0026497 as potential GDM biomarkers through microarray analysis [114]. Hsa_circRNA_0039480 demonstrated significant diagnostic significance even in the first trimester of pregnancy, which was enhanced by combining with hsa_circRNA_0026497 in a novel potentially valuable early diagnostic panel [114]. A microarray profiling also identified hsa_circRNA_102893 as a downregulated circRNA in plasma samples of GDM patients, with a good diagnostic significance in early pregnancy [115].

Patients of non-Asian origin were rarely included in circRNA-oriented GDM biomarker studies. A relatively recent study in Polish population identified over 50 transcripts (both linear and circular) corresponding to a large panel of circRNA-producing genes which are dysregulated in GDM plasma samples. The relative expression of hsa_circ_0002268 (circPHACTR1) was elevated in GDM patients, compared to controls [116].

Apart from studies that relied on circRNA profiling by high-throughput methods, several candidate circRNA-based GDM biomarker studies were conducted to date. CircVEGFC-focused analysis revealed an upregulation of this circRNA in GDM, with higher values in plasma corresponding to increased incidence rates of fetal malformation and hypertension [117]. Similarly, an upregulation of circACTR2 in GDM plasma samples was determined in a candidate-based study, associated with higher rates of GDM-related adverse events [118]. One of the promising candidates for GDM biomarkers among circRNAs, hsa_circRNA_0054633, was initially determined as dysregulated in type 2 diabetes (T2DM). The supposed upregulation of this circRNA in serum samples was confirmed in a GDM study, while the expression level correlated with the values of glycemic status parameters [119] An increased expression in GDM vs. controls was determined for second and third trimester samples, as well as for placental tissue [119]. Diagnostic significance as early biomarker of GDM was not evaluated for this circRNA. However, potential significance for diabetic disorders is supported by the findings suggesting that hsa_circRNA_0054633 may serve as an indicator of the clinical characteristics and the effect of insulin therapy in T2DM [120, 121].

Conclusion

Although a multitude of evidence has suggested a role of different species of circulatory ncRNAs as novel potentially valuable biomarkers of GDM, results are still inconsistent and require carefully planned validation. The main objective is to identify clinically useful early biomarkers of GDM, enabling more precise risk stratification. The key strengths of ncRNA as potential biomarkers for GDM are their chemical stability in body fluids, minimal invasiveness of sampling procedure and the reliability of quantification procedures.

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