Chapter

The Role of miR-107 in Prostate Cancer: A Review and Experimental Evidence

Maria Elizbeth Alvarez-Sanchez, Oscar Rojas Espinosa, Julio César Torres-Romero, Ereth Ameyatzin Robles Chávez, Edgar Estrella-Parra, María Dolores Ponce Regalado, Raúl Aragón Franco, Jose Gadú Campos Saucedo, Cesar López-Camarillo, Minerva Camacho Nuez and Verónica Fernández Sánchez

Abstract

Over the past two decades, several research groups have focused on the functioning of microRNAs (miRNAs), because many of them function as positive or negative endogenous regulators of processes that alter during the development of cancer. Prostate cancer is the second most commonly occurring cancer in men. New biomarkers are needed to support the diagnosis of prostate cancer. Although it is necessary to deepen the research on this molecule to explore its potential utility in the diagnosis, follow-up, and prognosis of cancer, our results support a role of miR-107 in the signaling cascades that allow cancer progression, and as shown here, in the progression of Prostate Cancer (PCa). These findings strongly suggest that miR-107 may be a potential circulating biomarker for the diagnosis and prognosis of prostate cancer.

Keywords: microRNAs, miR-107, biomarker, cancer hallmarks, therapeutic target

1. Introduction

Cancer can be defined as a state of uncontrolled cell-growth and dissemination that alters several cellular processes and functions [1]. Hanahan and Weinburg described, in 2000, six characteristics of cancer called "cancer hallmarks" that increased to ten in 2011 due to the complexity of the disease and the number of biological mechanisms that become altered. These cancer hallmarks include sustained proliferative signals, evading growth suppressors, avoidance of immune destruction, replicative immortality, tumor-promoting inflammation, sustained invasion and metastasis, induction of angiogenesis, genetic instability and mutation, resistance to cell death, and cell-metabolism deregulation [2].

Currently, more than 100 different types of cancer have been identified. According to the International Agency for Research in Cancer (IARC) in 2020, the

most common types of cancer were those of lung, breast, prostate, colon, and stomach [3]. In the 2014 World Cancer Report, the World Health Organization (WHO) reported 14 million new cases and 8.2 million deaths in the year 2012 [4]. Last year, the Pan American Health Organization (PAHO) reported that in the Americas, the most frequently diagnosed types of cancer in men are prostate (21.7%), lung (9.5%), and colorectal (8.0%) cancer [5].

In 2018, the estimated number of new cases increased to 18 million, and it was expected that by 2030, more than 20 million cases will be registered annually [4]. Under this scenario, different methods of early diagnosis and treatment have been sought, which can effectively defeat the disease, leading to a significant reduction in the number of cases.

2. MicroRNAs as a potencial cancer biomarkers

MicroRNAs (miRNAs) are endogenous regulators of different biological processes, including those that are altered in cancer development such as cell growth and proliferation, differentiation, apoptosis, angiogenesis, and others [6]. miRNAs are a family of small non-coding RNAs, 18 to 22 nucleotides long whose function is the post-transcriptional regulation of gene expression [7]. They have a specific region composed of around 6 nucleotides called the seed region that binds the 3'UTR region of the target messenger RNA (mRNA). This union alters the stability of the transcript leading to its degradation or storage in intracellular structures called p-bodies, thus leading to effective repression of translation [8].

Different authors have reported that miRNAs have a tissue-specific expression pattern and that this pattern is altered in cancer tissues [9, 10]. Therefore, it has been suggested that these changes in the expression of miRNAs can be used as possible biomarkers of the disease [11]. However, the standard determination of the relative expression of miRNA requires a tissue sample from the cancer patient, which is considered a highly invasive process [12, 13]. Consequently, other diagnostic methods are required that are safe, effective, and accessible.

In 2008, Mitchell et al. described a specific type of miRNAs that were present in their stable form in different biological fluids, which they called circulating miRNAs (c-miRNAs) [14]. In their research, they analyzed the expression of different miRNAs in plasma, serum, and epithelial tissue samples. They observed that the miRNAs remained stable in serum and plasma, and in the tissue samples. They concluded that blood was a reliable source for the extraction and quantification of miRNAs. These findings have allowed the analysis of the expression of circulating miRNAs as a possible method for cancer diagnosis [14, 15].

There are more than 1000 miRNAs registered in the miRNA database (miRDB) that have been found in the human body; one of them is miR-107, which, according to miRDB, can have more than 800 possible targets [16].

3. Is miR-107, a circulating miRNA?

MicroRNA-107 (miR-107) is a molecule composed of 23 nucleotides; it is considered a c-miRNA because it can be found in a stable form in plasma and urine [17]. This miRNA is highly conserved in humans and other species. In humans, it is transcribed from the introns of the pantothenate kinase 1 gene, located on the long arm of chromosome 10 [16]. It belongs to the miR-103/107 family, which participates in the regulation of proteins involved in cell proliferation, cell cycle arrest, and programmed cell death or apoptosis [18].

Different research groups have reported that miR-107 is altered in different types of cancer in both men and women, including cervical [19], breast [20], ovary [21], colorectal [22], gastric [23], oral [24], penile [25], and prostate [13] cancers. Because of this, it has been proposed as a possible biomarker of cancer and a potential target for treatment.

So far, information on the expression of miR-107 in plasma has been published only on gastrointestinal cancer by Parvaee et al. [15], and in the urine of patients with prostate cancer by Lekchnov et al. [17]. Therefore, there is a great opportunity in this field to contribute to the design of new diagnostic methods for the detection of cancer from the early stages of the disease.

4. miR-107 and its participation in prostate cancer

PCa is the second leading cause of death in men over 45 years of age; the American Cancer Society estimates that 1 in 41 men will die this year from this cause. Nevertheless, prostate cancer has not been studied to the same extent as breast cancer, and hence, there is a lack of information on the development, evolution, and the treatment of this disease. Although the evaluation of prostatic specific antigen (PSA) has contributed to the early detection of PCa, its use may also lead to non-conclusive results because of false positive and negative results. Also, its low specificity can lead to misdiagnosis and incorrect treatment of indolent PCa patients [25, 26]. Patients with prostate cancer who receive radical treatment for presumably locally confined cancer can experience clinical relapses, indicating that the extent of these cancers was greater than the one previously diagnosed [27]. The median survival of metastatic PCa patients is 5 to 8 months, and their 5-year survival rate is less than 30%. This poor prognosis is the result of many factors including the lack of initial symptoms when PCa develops, local invasiveness, or metastases to distant organs in the early stage of the disease, and misdiagnosis [28, 29].

A majority of PCa patients who initially respond to androgen suppression treatment (AST) develop metastatic castration-resistant prostate cancer (CRPCa) within 2 years of treatment [29]. This condition still cannot be predicted, although several biomarkers have been tested (proteomic and metabolomic approaches) [30, 31]. This is mainly due to the wide variability of proteins being tested, the masking effect of abundant serum proteins, the high salt concentration in the samples, and the variability between individuals that drastically reduce the reproducibility of the biomarker's determinations. miRNAs have been found to participate in the alteration of many cellular processes that lead to the development of cancer including proliferation [32, 33], differentiation [34, 35], angiogenesis, and evasion of apoptosis [36]. miRNAs can serve as biomarkers because they are resistant to degradation by RNases either in tumors or serum. miRNAs are small (18-24 nt), endogenous, non-coding RNAs, encoded either on intergenic regions of DNA or on introns and exons of genes that act as post-transcriptional control genes, triggering degradation or blocking translation of mRNAs by complementary base pairing [37]. When discovered, miRNAs were shown to control fundamental cellular processes, such as cell differentiation and timing of the organism development [38, 39], suggesting that aberrations of miRNAs could be involved in various human diseases, including cancer.

In recent years, several detailed studies have described the mechanisms through which miRNAs are stabilized and how they are detected in plasma and serum [10, 14]. Plasma/serum miRNAs are resistant to endogenous ribonuclease activity by binding to specific plasma proteins or by packing into various serum secretory vesicles, including

apoptotic bodies and exosomes [14, 40–42]. Various blood-based miRNAs have been identified, including those in this study, and can be used for cancer detection, monitoring tumor dynamics, and predicting prognosis and chemoresistance [43–47].

Several reports have shown that changes in the level of circulating miRNAs associate with prostate cancer [48]. One of these miRNAs is miR-107, which is overexpressed by more than 11-fold in PCa [48] but whose role has not been studied in the context of cancer progression [49].

In 2019, Zhang et al. studied the role of miR-107 in prostate cancer in both the tissue and cancer cells (DU145 and PC3); they found that its levels decreased compared to the levels in healthy cells and tissue. Then they induced the overexpression of miR-107 and performed functional tests to evaluate its effects. In the colony formation test, they found that the increase in the expression of miR-107 significantly inhibited cell proliferation. They complemented this finding with the flow cytometric cell cycle test and concluded that the inhibition resulted from an arrest in the G1/S phase of the cycle, due to the binding of miR-107 to cyclin E1. In the migration and invasion tests, they did not find any influence of miR-107 on these cellular processes [13].

Previous studies show the tumorigenicity of the miR-107 family [50–53]; thus, the inhibition of the expression of miR-107 might be a target for the treatment. miR-107 may promote the progression of prostate cancer to CRPCa, the end-stage of a multifactorial and heterogeneous disease process [54, 55]. Significant progress in understanding the molecular basis of CRPCa has been achieved in recent years [56] but despite this, CRPCa remains a lethal disease [57].

Recently, Liang et al. [58] reported that miR-107 induces chemoresistance in colorectal cancer (CRC) through the CAB39-AMPK-mTOR pathway, promoting metastasis. In this context, miR-107 levels could potentially be determined at the time of diagnosis to identify patients with aggressive disease/micro metastases and/or to predict recurrence following primary treatment.

Studies of Bryant et al. [48] strongly suggest that the presence of miR-107 in plasma can be used as a biomarker for cancer detection, monitoring, and prognosis predictor in PCa patients. They reported 12 miRNAs differentially expressed in the plasma of PCa patients compared to controls. Among these 12 miRNAs, 11 were significantly correlated with metastases. The association of two miRNAs, miR-141 and miR-375 with metastatic PCa was confirmed in a separate cohort. Furthermore, an analysis of urine revealed that miR-107 and miR-574-3p were also notably associated with PCa risk [48].

The role of miR-107 in other hallmarks of prostate cancer is not yet known. However, it would be relevant for the medical and scientific community to determine the participation of this miRNA in the development, growth, and metastasis of this disease; this will encourage further research tending to mitigate this pathology. The pleiotropic functions of miR-107 in diverse types of cancer indicate that it targets a variety of genes involved in tumor proliferation, invasiveness, metastasis, angiogenesis, and response to chemotherapy. Because of their carcinogenic or cancer-suppressor effects, miR107 can be used as a potential diagnostic and prognostic biomarker, or as a target for therapeutic intervention [48].

5. miR-107 and its participation in other urologic cancers

Both men and women can develop urological cancers, such as urethral, bladder, and kidney cancers. Other urologic cancers are gender specific. Males, besides the prostate, can experience testicular and penile cancer. The role of miR-107 in some of these cancers has not been reported previously.

Investigation of bladder cancer-related miRNAs shows a specific downregulation of miR-107 in the *in-situ* carcinoma lesions in comparison to a normal bladder [49]. Several studies have shown that miR-107 sponge effects could be involved in processes that upregulate circular RNAs (circRNA) in bladder cancer-related pathways. For example, overexpression of the circRNAs of the transcription factor (TCF) 25 (circ-TCF25) and transferrin receptor (TFRC) (circ-TFRC) negatively correlated with miR-107 promotes progression of bladder cancer through the circ-TCF25-miR-103a-3p/miR-107-CDK6 and circ-TFRC-miR-107-TFRC pathways, respectively [59, 60].

So far, only one study mentioning a relationship between miR-107 and kidney cancer has been published. Song et al. found that cell proliferation and invasiveness of renal cell carcinoma, which is the most common type of kidney cancer in adults, can be suppressed by high expression of miR-107 through an apparent cell cycle arrest at the G2/M phase [61]. In contrast, it has been found that high expression of miR-107 is frequently associated with a bad prognosis in patients with penile cancer [25]. Overexpression of miR-107 in penile cancer tissues was also reported by Zhang et al. [62].

6. Strategy and methodology of study of miR-107

While several techniques and methodologies have been used for the study of miR-107, the most used techniques are qRT-PCR, proliferation assays, Western blotting, luciferase reporter, and immunofluorescent and immunohistochemical assays (**Figure 1**, **Table 1**). Yet, other authors have used less conventional methodologies. Thus, the optimized electrochemical sensor technic enabled the PCR-free

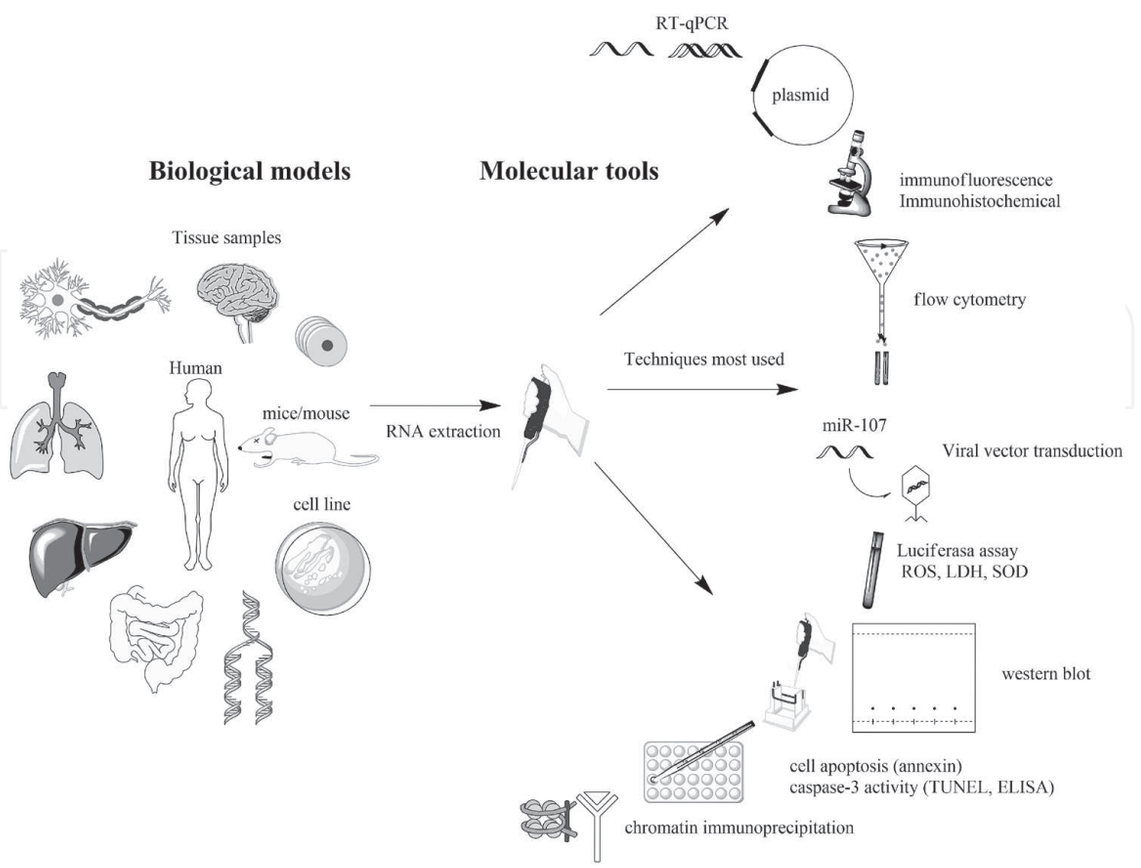

Figure 1.
Methodology most frequently used in the study of miR-107.

Pre-analysis	Techniques/molecular tools	Post-analysis		Authors
Organism/ pre-treatment	**Methodology**	**Statistical analysis**	**Conclusions**	
Mice (LDLr $^{-/-}$): fed a high-fat (liver tissue). *Hibiscus sabdarifa*: polyphenols. quercetin3-O-β-D-glucuronide	chromatography column, LC-MS, RT-PCR, Western blot, GC-MS, liver histology to measure the lipid droplet content.	ANOVA,U Mann–Whitney, t-Student's, Fisher's test P< 0.05	Polyphenols reduced the expression of miR-107 in the liver	[63]
Human glioma tissue. U251 and MO59K cell line. BALB/C-nu athymic nude mice.	qRT-PCR, transfection (miR-107), proliferation assay (MTT), Anchorage-independent growth assay, Cell apoptosis (annexing), luciferase reporter, Lentiviral construct transduction, Western blot and immunofluorescence analysis, computer-aided algorithms from PicTar.	Student's t-tests, Spearman's rank correlation. P< 0.05.	upregulation of miR-107 suppressed glioma cell growth	[64]
HepG2 cells and HEK 293.	cell transfection (miR-107), western blot, RT-PCR, DNA constructs, luciferase assay, Triglyceride assay,	student's t-test. P<0.05	miR-107 induced lipid accumulation in hepatocytes.	[65]
52 tumor of Kidney cancer. HKC cell line. Male nude mice	RT-PCR, Western blot, plasmid construction, transfection/ infection, luciferase reporter assay, flow cytometric	Wilcoxon rank sum test, ANOVA, Student t test.	miR-107 inhibit cell proliferation in renal cell carcinoma.	[61]
Primary cortical neurons, HEK293 and SHSY5Y cells. Osthole (7-methoxy-8-isopentenoxy coumarin)	Viral vector transduction, activity of LDH, cellular apoptosis (TUNEL), Immunofluorescence, RT-PCR, transfection (miR-107), western blot.	ANOVA, Bonferroni's, P< 0.05.	Osthole is neuroprotective via up-regulate miR-107 in AD.	[66]
SGC-7901, MKN-45, KATO III, BGC-823, AGS, MKN-28 and MKN1 cells lines. male nude mice: implanted miRNA-107 (tumor)	Transfection (miRNA-107), si-RNA, plasmid, RT-PCR, wound healing assay, luciferase activity, Western blot.	ANOVA, dunnett's multiple. P< 0.05	miR-107 acts as tumor inhibitor for gastric cancer	[67]
primary preadipocyte (mice)	Plasmid vector, transfection, cDNA synthesis, qPCR, luciferase reporter assays, caspase-3 activity, flow cytometry (apoptosis), TUNEL, western bloth, immunofluorescent.	Student's t-test. P< 0.05 and < 0.01.	miR-107 induced apoptosis pathway.	[68]
SGBS cells:	qPCR, western blotting, Transfected SGBS cells (miR-107, [^{3}H]palmitic acid).	not reported	miR-107 reduces adipogenesis	[69]

Pre-analysis	Techniques/molecular tools	Post-analysis		Authors
Organism/ pre-treatment	**Methodology**	**Statistical analysis**	**Conclusions**	
HCC patients: 1.FFPE cohort (tissue) 2. serum cohort (serum) Patient receiving before cisplatin, lipiodol, doxorubicin.	qPCR. Total RNA	Chi-square test, Mann-Whitney U. $P \leq 0.05$	miR-107 is biomarkers for predication of TACE treatment outcomes in HCC patients.	[70]
42 OA patients (cartilage samples): chondrocytes 48 rats: establish OA model	Transfection (chondrocytes/miR-107), flow cytometry, apoptosis (annexin/TUNEL assay), luciferase reporter, western blot, RT-q PCR,	Student's t-test. $P \leq 0.05$	miR-107 induced apoptosis and autophagy of OA chondrocytes	[71]
PC-3, DU145, LNCaP, 267B1, X/267B1, and Ki/267B1 cells	RNA isolation, qRT-PCR, electrochemical measurements (CHI 660D electrochemical workstation): frequency range of 0.1 Hz to 100 kHz with an alternating current amplitude of 10 mV.	Student's t-test. $P < 0.05$	Viability of the electrochemical evaluation method in clinical environment.	[72]
75 ADHD patients: stimulation in rDLPFC. for 6 weeks: 18 healthy children. Venous blood in both case.	RNA extraction, serum miRNA extraction, miRNA reverse transcription, fluorescence qPCR.	Mann-Whitney U test or Student's t-test. $P < 0.05$	serum miRNA-let-7d in ADHD patients is higher as compared to healthy children.	[73]
SKOV3 and 293T cells; transfected with lentiviruses	qRT-PCR, luciferase report assay, western blot, xenograft model and immunohistochemistry assay.	ANOVA, Student's t test, $P<0.05$	miR-107 as a tumor suppressor in ovarian cancer.	[74]
Patients with AD. SH-SY5Y and PC12 cells Mouse model: introducing 6-OHDA into the right ventral tegmental area	Cell transfection, qPCR, caspase-3 activity (ELISA), ROS, LDH, SOD, luciferase reporter assay, western blot. Rota-rod test. patients with AD: motor imagery test (fMRI)	ANOVA, Tukey's test, $P < 0.05$.	miR-107 may be a therapeutic target for the treatment of AD-related impairments.	[75]
HT-22 cell line	coimmunoprecipitation, chromatin immunoprecipitation, Luciferase reporter, qRT-PCR, apoptosis detection (annexin), western blot.	Student's t-test, ANOVA, Tukey's test $P < 0.05$	RMST activates p53/miR-107 signaling pathway	[76]
Serum samples (NSCLC). cell line BESA-2B, NSCLC, A549, H1299, HCC827, PC-9, 95-D, H1975, HEK-293T	luciferase reporter gene, qRT-PCR, western blot, Immunohistochemistry.	t-tests for two-group comparisons$P < 0.05$.	miR-107 could inhibit the progression of NSCLC	[77]

Pre-analysis	Techniques/molecular tools	Post-analysis		Authors
Organism/ pre-treatment	**Methodology**	**Statistical analysis**	**Conclusions**	
40 Sprague Dawley (SD) rats. MC3T3-E1 cells *Agrimonia pilos* (isolated polysaccharide) Rats induced SANFH (dex)	Femoral head tissue: apoptosis cellular (TUNEL), RT-PCR, cell proliferation (annexin), MC3T3-E1 cells transfected with anti-miR-107, flow cytometry, ALP activity, western blot.	ANOVA. P< 0.05	polysaccharides promote cell proliferation/ osteogenic differentiation by enhancing miR-107	[78]
HeLa and HEK	Reporter gene assay (FuGENE 6), RT-PCR, immunoblot/ Immunofluorescence assay, chromatin immunoprecipitation, ChIP-seq analysis: microRNA103–3p/107 target,. motif discovery (RSAT), Gene ontology analysis, genomic distribution (CEAS), siRNA transfections.	ANOVA, Bonferroni, KruskalWallis, Dunn's, t-test, Mann-Whitney U. P< 0.05.	miR-107 is potent regulators of GR function	[79]
30 HIBD rats Neonatal male Sprague-Dawley: HIBD injected with adenovirus: primary neuron cells	Neuronal cells (infected with adenovirus): RT-qPCR, Protein extraction, western blot, luciferase reporter, immunofluorescence, fluorescent hybridization, RNA immunoprecipitation. hippocampal neurons (NisslStaining).	unpaired t-test, ANOVA, Tukey test. P < 0.05.	miR-107 network has the potential to provide novel insights into treatment targets for HIBD.	[80]
70 tumor human tissue (NPC) Fresh frozen cell lines: HNE1, HONE1, 5–8F, 6–10B, and C666-1 and NP69:	qRT-PCR, western blot, cell apoptosis (anexin), flow cytometry, DIG-UTP-labeled miR-107, FISH kit, Immunofluorescence/ Immunohistochemical analysis BALB/c mice: (transfected HNE1 cells) lung	Student's t-test/Chi-square test, Pearson correlation. P< 0.05	circTGFBR2 suppresses the proliferation, migration, and invasiveness of NPC cells by sponging miR-107	[81]

HIBD: hypoxic-ischemic brain damage. NPC: nasopharyngeal carcinoma. NP69: immortalized nasopharyngeal epithelial cell line. RT-qPCR: quantitative polymerase chain reaction. HeLa: Human cervical carcinoma cells. HEK: human embryonic kidney cells. GR: glucocorticoids. SANFH: steroid-induced osteonecrosis of the femoral head. Dex: dexamethasone. ALP: Alkaline phosphatase. BESA-2B, NSCLC, A549, H1299, HCC827, PC-9, 95-D, H1975: Human normal bronchial epithelial cell line: HEK-293T: human embryonic kidney cell line. NSCLC: Non-small cell lung cancer. TGFβR2: Transforming growth factor β receptor 2. HT-22: Immortalized mouse hippocampal neuron cell line. RMST: rhabdomyosarcoma 2-associated transcript. hnRNPK: Heterogeneous nuclear ribonucleoprotein K. SH-SY5Y: human neuroblastoma cell line. PC12: cells rat pheochromocytoma. LDH lactate dehydrogenase release. SOD: superoxide dismutase. ROS: reactive oxygen species. 6-OHDA: 6-hydroxydopamine. fMRI: magnetic resonance imaging. AD: Alzheimer's disease. ADHD: attention deficit hyperactivity disorder. rDLPFC: right dorsolateral prefrontal cortex. A549: human lung carcinoma cells. Human_Br07: human-origin seasonal influenza A virus H3N2. PC-3, DU145 and LNCaP: human transformed prostate epithelial cell lines. 267B1, X/267B1: cell line nontumorigenic in nude mice. Ki/267B1: cell line moderately tumorigenic nontumorigenic in nude mice. OA: Osteoarthritis. TRAF3: TNF Receptor Associated Factor 3. HCC: hepatocellular carcinoma. FFPE: Formalin-Fixed Paraffin-Embedded. TACE: transcatheter arterial chemoembolization (cancerous tumor therapy). SGBS: Simpson-Golabi-Behmel syndrome preadipocytes. LDLr -/-: deficient in LDL receptor. LC-MS: Liquid chromatography–mass spectrometry. GC-MS: Gas chromatography–mass spectrometry. U251 and MO59K: Human glioma cell lines. MTT: 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide. SALL4: Sal-like 4. HKC: Human renal proximal tubular epithelial cell line. SHSY5Y: neuron cell line. SGC-7901, MKN-45, KATO III, BGC-823, AGS, MKN-28 and MKN1: gastric cancer cell lines. TGFBR2: transforming growth factor-β receptor II.

Table 1.
Methodology and techniques used in the study of miR-107.

quantification of miR-375 in CaP cells with acceptable specificity, confirming its potential applicability for point care (POC) purposes [72]. Detection at attomole levels of miRNA in samples is possible by electrocatalytic detection using gold-loaded nanoporous superparamagnetic iron oxide nanocubes, that has proved successful in the detection of miR-107 from cancer cell lines [82]. Other authors mentioned the use of chromatographic techniques such as Liquid chromatography-mass spectrometry (LC–MS) to extract and identify polyphenols from the plant *Hibiscus sabdarifa*, which reduced the expression of miR-107 in the liver [63] as well as of osthole (a coumarin compound) which is neuroprotective [66], and polysaccharides from *Agrimonia pilosa*, which promote cell proliferation enhanced by miR-107 [78, 83].

A variety of statistical tests have been used to validate the results, including parametric (ANOVA, student-*t*, Tukey) and non-parametric (Mann–Whitney U, Kruskal Wallis, Bonferroni, Dunn's multiple comparison, Chi-square, and Pearson correlation) tests. The probability level in all cases have been $P < 0.05$. In conclusion, the new technology and the use of diverse statistical tools validate the study and significance of mirR-107 in diverse biological situations, including CaP.

7. miR-107 as a possible blood biomarker

In 2019, Parvaee et al., evaluated the expression of three miRNAs, including miR-107, in blood samples from 50 patients with gastrointestinal cancer. After extracting and analyzing the plasma, they observed that the expression of miR-107 was significantly lower in patients with this type of cancer, compared to healthy volunteers (93.8% sensitivity and 78.8% specificity). From the ROC curve evaluation, they found that the patients could be distinguished from healthy people at cutoff levels of 0.504 (miR-107), 0.266 (miR-194), and 0.394 (miR-210). Finally, they concluded that miR-107 could serve as a possible plasma biomarker, assuring the minimum degree of invasion for the patient and an adequate level of reliability in the diagnosis [15].

8. The future for the miR-107

Given its role in cell cycle arrest and its direct involvement in cancer, the therapeutic potential of miR-107 is anticipated [13]. miR-107 may be a key target for the treatment of prostate cancer, arresting tumor growth, and cell survival.

9. Conclusion

The findings condensed in this review enable us to envisage miR-107 as a biomarker and possible therapeutic target for diverse types of prostate cancer. Although it is necessary to deepen the research on this molecule to explore its potential utility in the diagnosis, follow-up, and prognosis of cancer, our results support a role of miR-107 in the signaling cascades that allow cancer progression, and as shown here, in the progression of PCa to CRPCa. Our research suggests a potential utility of miR-107 as an accurate tool for diagnoses and follow-up of PCa.

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Conflict of interest

The authors declare that they have no competing interests.

Author's contribution

MEAS, EARC, and EEP conceived, designed, and wrote the review. ORE, CLC, MDPR, JCTR, MCN and RAF reviewed and edited the manuscript. All authors read and approved the manuscript and agreed to be accountable for all aspects of the research ensuring that the accuracy or integrity of any part of the work is appropriately investigated and resolved.

Author details

Maria Elizbeth Alvarez-Sanchez[1*], Oscar Rojas Espinosa[2], Julio César Torres-Romero[3], Ereth Ameyatzin Robles Chávez[1], Edgar Estrella-Parra[4], María Dolores Ponce Regalado[5], Raúl Aragón Franco[6], Jose Gadú Campos Saucedo[7], Cesar López-Camarillo[1], Minerva Camacho Nuez[1] and Verónica Fernández Sánchez[4]

1 Posgrado en Ciencias Genómicas, Universidad Autónoma de la Ciudad de México, Ciudad de México, México

2 Departamento de Inmunología, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional (IPN), Ciudad de México, México

3 Laboratorio de Bioquímica y Genética Molecular, Facultad de Química, Universidad Autónoma de Yucatán, Mérida, Yucatán, México

4 Facultad de Estudios Superiores Iztacala, Universidad Nacional Autónoma de México (UNAM), Ciudad de México, México

5 Departamento de Ciencias de la Salud, Centro Universitario de los Altos, Universidad de Guadalajara, Tepatitlán de Morelos, Jalisco, México

6 Universidad de la Salud "Vasco de Quiroga", Ciudad de México, México

7 Hospital Central Militar, Dirección General de Sanidad SEDENA, Ciudad de México, México

*Address all correspondence to: maria.alvarez@uacm.edu.mx

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