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Review

A review on the role of PCGEM1 lncRNA in cancer

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ABSTRACT

PCGEM1 Prostate-Specific Transcript is an lncRNA participating in the carcinogenesis process in different tissues. The gene coding this lncRNA is located on chr2:192,749,008–192,783,952 (GRCh38/hg38), plus strand and has 34,945 bases. In addition to prostate cancer, it has been shown to influence progression of cervical, endometrial, gastric, ovarian, hepatocellular and renal cancers. Functionally, PCGEM1 regulates activity of a number of molecular axes, namely miR-642a-5p/LGMN, miR-182/FBXW11, miR-129–5p/STAT3, miR-129–5p/P4HA2, miR-433–3p/FGF2, miR-539–5p/CDK6, miR-129–5p/ETV1, miR-433–3p/WTAP, miR-590–3p/SOX11 and miR-506–3p/TRIAP1. Moreover, it has a regulatory effect on RhoA, NF-κB and β-catenin/TCF signaling pathways. We have designed this literature search to collect all relevant data about the function of PCGEM1 in the carcinogenesis.

1. Introduction

Long non-coding RNAs (lncRNAs) are a newly appreciated of regulators of epigenetic marks. They regulate epigenetic modifications principally in the nucleus [6,7]. Their impacts on transcription of genes are exerted through modulation of histone or DNA modifications [31]. These transcripts can also regulate methylation or acetylation histones [13]. Their functions as scaffolds facilitate establishment of protein complexes that affect histone marks [23]. In addition to their regulatory functions at the chromatin level, lncRNAs located in the cytoplasm modulate mRNAs stability, affect translation of mRNAs, serve as competing endogenous RNA (ceRNA), function as precursor for production of microRNAs (miRNAs), and mediate modification of proteins [5,21]. Such diverse functions of lncRNAs endow them the potential for regulation of several cellular processes and participation in the pathoetiology of human disorders, particularly cancers.

PCGEM1 Prostate-Specific Transcript is an lncRNA with important functions in the carcinogenesis [19,22]. The gene coding this lncRNA is located on chr2:192,749,008–192,783,952 (GRCh38/hg38), plus strand and has 34,945 bases. Functionally, it is a negative regulator of apoptotic pathways, and might activate androgen receptor and Myc. A handful of studies have reported over-expression of PCGEM1 in cancer cell lines clinical samples obtained from patients with a variety of malignancies such as those originated from including those originated from cervix [18], endometrium [15], stomach [30], ovary [3], liver [2], kidney [1] and brain [17]. Furthermore, the impact of over-expression of PCGEM1 in cancer-derived cell lines has been evaluated in xenograft models of cancers. We have searched literature to collect all relevant data about the function of PCGEM1 in the carcinogenesis.

2. Cell line studies

PCGEM1 has been shown to be up-regulated in cervical cancer cells. PCGEM1 silencing has suppressed proliferation, migratory aptitude, and invasive features, while inducing G1 arrest in these cells. Besides, PCGEM1 could target miR-642a-5p. Suppression of miR-642a-5p partially eliminated the impact of PCGEM1 silencing on proliferation, cell cycle progression, migratory potential and invasiveness. miR-642a-5p

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has an inhibitory effect on LGMN, an up-regulated gene in the cervical cancer cells. Taken together, miR-642a-5p/LGMN has been identified as the main molecular mechanism of participation of PCGEM1 in cervical carcinogenesis [18]. Another study in this type of cancer has shown that PCGEM1 promotes proliferation of cervical cancer cells and enhances cell cycle progression, migration and invasiveness through acting as a ceRNA for miR-182 to increase expression of FBXW11. Moreover, PCGEM1 has the capacity for activation of the NF-κB and β-catenin/TCF signals, which has been reversed by suppression of FBXW11 [28].

Similarly, over-expression of PCGEM1 in endometrial cancer cells has been associated with enhancement of proliferation, migration, and invasive properties of these cells. PCGEM1 could up-regulate expression of STAT3, thus increasing levels of BCL-2, survivin, VEGF-A, and MMP-2. These effects have been shown to be mediated through sponging miR-129–5p [15].

Experiments in gastric cancer cells have shown high levels of expression of PCGEM1 in the cytoplasm. PCGEM1 knock down has decreased metastatic and invasive abilities of these cells. Functionally, PCGEM1 acts as a ceRNA of P4HA2 through sequestering miR-129–5p [30]. Another study has shown specific expression of PCGEM1 in exosomes of hypoxia-cultured gastric cancer cells. PCGEM1-containing exosomes have the potential to increase invasiveness and migratory potential of gastric cancer cells. Mechanistically, PCGEM1 could increase the stability and reduce destruction of SNAI1. These effects enhance epithelial-mesenchymal transition (EMT) of gastric cells [20].

PCGEM1 has also been shown to be upregulated in glioma cell lines. PCGEM1 silencing has suppressed proliferation, colony construction, migratory aptitude and invasive properties. Mechanistically, PCGEM1 plays a competing endogenous RNA (ceRNA) role for miR-539–5p to increase expression of CDK6 expression. Over-expression of miR-539–5p could suppress progression of this cancer while up-regulation of CDK6 reverses the impacts of PCGEM1 silencing [17].

Another study has shown up-regulation of PCGEM1 and ETV1 in oxaliplatin-resistant Hep3B cells. Besides, PCGEM1 silencing has reduced migratory potential, invasiveness and expression levels of LRPA, MDR1 and MDR3. Moreover, knock down of this lncRNA has decreased viability of oxaliplatin-resistant Hep3B cells. miR-129–5p has predicted to target PCGEM1 and ETV1, indicating that PCGEM1 enhances expression of ETV1 through sequestering miR-129–5p. Taken together, PCGEM1 modulates resistance to oxaliplatin through influencing miR-129–5p/ETV1 axis in hepatocellular carcinoma cells [2].

An experiment in lung cancer cells has revealed up-regulation of PCGEM1, while down-regulation of miR-433–3p. PCGEM1 knock-down or miR-433–3p up-regulation has suppressed proliferation, migratory potential and invasion but enhanced apoptosis of these cells. Functional studies have indicated that miR-433–3p is sequestered by PCGEM1. Furthermore, WTAP has been proven to be miR-433–3p target. Up-regulation of WTAP or miR-433–3p silencing has reversed the suppressive impacts of PCGEM1 knock down on malignant behaviors of lung cancer cells [25]. Another study in this kind of cancer has revealed the effect of PCGEM1 on activity of miR-590–3p/SOX11 axis [24]. Furthermore, PCGEM1 has been found to contribute to the proliferation, migration and invasive features of lung cancer cells through sponging miR-152–3p [12]. Fig. 1 shows the impact of PCGEM1 in the progression of different cancers.

Over-expression of PCGEM1 in ovarian cancer cells has promoted proliferation, migration, and invasive propertied of cancer cells, yet decreasing their apoptosis via enhancing expression of RhoA, YAP, MMP2, Bcl-xL, and P70S6K. Besides, RhoA knock down has reversed the impact of PCGEM1. Thus, PCGEM1has been suggested to induce epithelial ovarian carcinogenesis through increasing expression of RhoA [3].

PCGEM1 silencing in prostate cancer cells has suppressed proliferation, invasiveness and migration of these cells. Functionally, miR-506–3p has interaction with PCGEM1 and TRIAP1. The inhibitory impact of PCGEM1 silencing on prostate cancer cells has reversed by co-transfection of TRIAP1 or a miR-506–3p inhibitor. Taken together, PCGEM1 enhances progression of prostate cancer through sequestering miR-506 and subsequent upregulation of TRIAP1 [16]. Table 1 demonstrates function and expression of PCGEM1 in cancer cell lines.

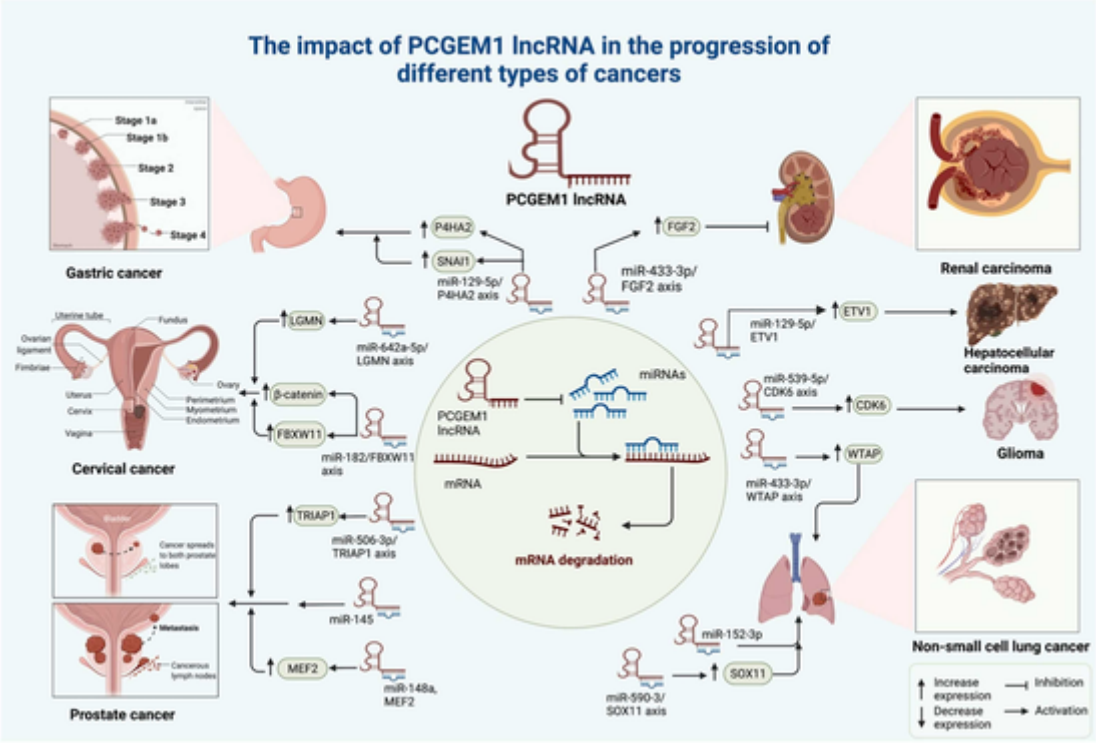

Fig. 1. The impact of PCGEM1 in the progression of different cancers.

Table 1
Function and expression of PCGEM1 in cancer cell lines (Δ: knock-down or deletion, DIM: 3,3′-Diindolylmethane, DOX: doxorubicin).

Tumor type	Targets/ Regulators and Signaling Pathways	Cell line	Function	Reference
Cervical cancer	miR-642a-5p/ LGMN axis	Hela, SiHa, Caski, and H8	Δ PCGEM1: ↓ proliferation, migration, and invasion and ↑ G1 phase arrest	[18]
	miR-182/ FBXW11 axis and NF-κB and β-catenin/TCF signaling pathways	Ect1/E6E7, C33A, HeLa, SiHa, and CaSki	Δ PCGEM1: ↓ proliferation, colony formation, migration, invasion, ↑ cell cycle arrest and apoptosis	[28]
Endometrial carcinoma	miR-129–5p/ STAT3 axis	Ishikawa and HEC-1B	↑↑ PCGEM1: ↑ proliferation, migration and invasion and ↓ apoptosis	[15]
Gastric cancer	miR-129–5p/ P4HA2 axis	BGC-823, SGC-7901 and GES-1	Δ PCGEM1: ↓ metastasis and invasion	[30]
	SNAI1	AGS and MKN45	↑↑ PCGEM1: ↑ viability, migration and invasion, EMT process by maintaining stability and reducing the degradation of SNAI1	[20]
	SNAI1	GSE-1, SGC-7901 and BGC-823	Δ PCGEM1: ↓ invasion and metastasis ↑↑ PCGEM1: ↑ metastasis, invasion, EMT process by regulating SNAI1	[27]
Glioma	miR-539–5p/ CDK6 axis	U251, LN229, NHA	Δ PCGEM1: ↓ cell proliferation, ↓ colony formation, ↓ migration and ↓ invasion	[17]
Hepatocellular carcinoma	miR-129–5p/ ETV1 axis	Hep3B/ OXA	Δ PCGEM1: ↓ cell viability, viability, migration and invasion	[2]
Non-small cell lung cancer	miR-433–3p/ WTAP axis	BEAS-2B, A549, NCI-H1299, NCI-H1650	Δ PCGEM1: ↓ proliferation, migration, invasion and ↑ apoptosis	[25]
	miR-590–3p/ SOX11 axis	A549, H1299, H460, H1975, BEAS-2B	Δ PCGEM1: ↓ proliferation, viability, migration and invasion	[24]
	miR-152–3p	NSCLC cells	Δ PCGEM1: ↓ proliferation, migration and invasion	[12]
Ovarian cancer	RhoA pathway	A2780 and OVCAR3	↑↑ PCGEM1: ↑ proliferation, migration and invasion and ↓ apoptosis via upregulating RhoA pathway Δ PCGEM1: ↓ proliferation, migration, and invasion and ↑ apoptosis	[3]
Prostate cancer	miR-506–3p/ TRIAP1 axis	PC-3, LNPCa, Du-145, C4–2B, RWPE1	Δ PCGEM1: ↓ cell proliferation, ↓ migration and ↓ invasion	[16]
	–	LNCaP and CWR22Rv1	Δ p54/nrb: ↓ PCGEM1 expression ↑ p54/nrb: ↑ PCGEM1 expression : ↑ androgen receptor splice variant AR3: ↑ castration resistance DIM preventes the interaction of p54/nrb with the PCGEM1 promoter, so reduced PCGEM1 levels.	[11]
	miR-148a, MEF2	LNCaP, DU145, and PC-3	MEF2 was found to regulate expression of PCGEM1 by affecting PCGEM1 promoter activity	[29]
	–	LNCaP	↑↑ PCGEM1: ↓ apoptosis induced by DOX	[4]
	miR-145	RWPE-1, HEK293T and LNCaP	Δ PCGEM1: ↓ cell proliferation, migration, invasion and ↑ apoptosis A reciprocal regulation has been found between miR-145 and PCGEM1. miR-145 could regulate PCGEM1 expression by binding to target sites within the PCGEM1 sequence.	[9]
	–	LNCaP and in NIH3T3	↑↑ PCGEM1: ↑ cell proliferation, ↑ colony formation	[19]
	–	LNCaP, DU145 and PC3	γ-oryzanol: ↓ caveolin-1 and PCGEM1 expression: ↑ apoptosis and/or necrosis death and ↓ cell cycle progression	[10]
	–	LNCaP	Δ PCGEM1: ↓ colony formation, ↑ sensitivity of LNCaP cells to baicalein, ↑ autophagy and ↑ cell cycle arrest	[8]
	hnRNP A1 and U2AF65	LNCaP and CWR22Rv1	AD-induced PCGEM1 interacts with both hnRNP A1 and U2AF65 with different consequences. The interaction of PCGEM1 with hnRNP A1 inhibits AR3 by exon skipping; its interaction with U2AF65 induces AR3 by exonization.	[32]
Renal carcinoma	miR-433–3p/ FGF2 axis	HK-2, OSRC-2, ACHN, A498 and 786 O	Δ PCGEM1: ↓ cell proliferation, migration and ↑ apoptosis	[1]

3. Animal studies

Consistent findings from experiments in animal models of endometrial carcinoma, glioma, lung cancer, ovarian cancer, prostate cancer and renal carcinoma have proved the impact of PCGEM1 up-regulation in enhancement of tumor growth and suppressive effects of PCGEM1 silencing in this process (Table 2). Thus, in vivo studies has confirmed the findings obtained from in vitro investigations.

Table 2
Function of PCGEM1 in animal models (Δ: knock-down or deletion).

Tumor Type	Animal models	Results	Reference
Endometrial carcinoma	4-week-old female BALB/c mice	↑↑ PCGEM1: ↑ tumor volumes	[15]
Glioma	6-week old female Nude mice	Δ PCGEM1: ↓ tumor growth	[17]
Non-small cell lung cancer	15 4–5 week-old BALB/c nude mice	Δ PCGEM1: ↓ tumor growth and tumor weights	[24]
Ovarian cancer	4-week-old female BALB/c nude mice	↑↑ PCGEM1: ↑ tumor growth	[3]
Prostate cancer	4-week-old female BALB/c nude mice	Δ PCGEM1: ↓ tumor volumes and tumor weights	[16]
	BalB/c (nu/nu) mice	Δ PCGEM1: ↓ tumor growth	[9]
Renal carcinoma	Male nude mice	Δ PCGEM1: ↓ tumor size, tumor volumes and tumor weights	[1]

4. Clinical studies

In cervical cancer samples, up-regulation of PCGEM1 has been correlated with advanced FIGO stage, lymph node involvement, distant metastases and poor clinical outcome [28]. PCGEM1 has also been shown to be upregulated in glioma tissues in correlation with unsatisfactory prognosis [17]. Expression of PCGEM1 has been found to be elevated in epithelial ovarian cancer samples compared with normal ovarian samples and has been correlated with level of differentiation cancer cells [3]. In prostate cancer cells, over-expression of PCGEM1 has been correlated with high Gleason score, distant metastases and extracapsular invasion [16]. In gastric cancer, circulatory levels of PCGEM1 have been found to be elevated. Besides, PCGEM1 expression levels have been associated with level of differentiation of tumor cells and clinical stage. The diagnostic power of PCGEM1 levels has been found to be superior to conventional tumor biomarkers, yet integrative assessment of all biomarkers has exhibited the best predictive value. Taken together, plasma levels of PCGEM1 can be putative circulating marker for diagnosis of gastric cancer and prediction of its prognosis [14]. Survival analyses in cervical cancer, glioma and renal carcinoma have proved the impact of up-regulation of PCGEM1 on deterioration of clinical outcomes. Table 3 shows dysregulation of PCGEM1 in clinical samples and their relationship with clinical features.

5. Discussion

PCGEM1 is an oncogenic lncRNAs, being identified firstly in prostate cancer. However, additional studies have revealed its effects in a variety of cancers, including those originated from cervix, endometrium, stomach, ovary, liver, kidney and brain. Functionally, PCGEM1 acts as a ceRNA for several miRNAs such as miR-642a-5p, miR-182, miR-129–5p, miR-129–5p, miR-539–5p, miR-433–3p, miR-590–3p and miR-506–3p. Moreover, it has a regulatory effect on RhoA, NF-κB and β-catenin/TCF signaling pathways.

Up-regulation of PCGEM1 in clinical samples has been correlated with advanced tumor stage or grade, potentiating this lncRNA as a predictor of cancer behavior. Independent studies in cervical and renal cancer as well as glioma have shown significant correlations between up-regulation of PCGEM1 and short survival of patients.

Notably, PCGEM1 has a number of polymorphisms, two of its intronic ones being associated with risk of prostate cancer. However, associations between these polymorphisms and other types of cancers have not been assessed. Since PCGEM1 has been shown to contribute in the pathogenesis of a variety of human cancers, it is recommended to assess such associations in different ethnic groups to find genetic basis for increased risk of cancers.

PCGEM1 has a potential impact in determination of chemoresistance. This feature has been well recognized for resistance to oxaliplatin. The impact of this lncRNA on induction of resistance to other chemotherapeutic agents should be assessed in future studies.

Diagnostic impact of PCGEM1 has only been investigated in gastric cancer. Since it can be detected in the circulation of patients, it might

Table 3
Dysregulation of PCGEM1 in clinical samples (ANCTs: adjacent non-cancerous tissues, OS: Overall survival, PCa: Prostate cancer, FIGO: International Federation of Gynecology and Obstetrics, EC: Endometrial carcinoma).

Tumor/disease type	samples	Expression (Tumor vs. Normal)	Kaplan-Meier analysis (impact of PCGEM1 up-regulation)	Association of low expression PCGEM1 with Clinicopathologic characteristics	Association studies	Reference
Cervical cancer	68 pairs of tumor tissues and ANCTs	increased	Shorter OS	advanced FIGO stage, lymph node, distant metastasis and poor prognosis	_	[28]
Endometrial carcinoma	95 EC tissues and 27 normal endometrial tissues	increased	_	stage III/IV		[15]
Gastric cancer	317 GC patients and 100 healthy controls	increased	_	tumor differentiation and TNM stage	_	[14]
Glioma	43 pairs of tumor tissues and ANCTs	increased	Shorter OS	grades III/IV	_	[17]
Non-small cell lung cancer	50 pairs of tumor tissues and ANCTs	increased	_	_	_	[25]
	40 pairs of tumor tissues and ANCTs	increased	_	_	_	[24]
Ovarian cancer	50 epithelial ovarian cancer tissues and 14 normal tissues	increased	_	differentiation	_	[3]
Prostate cancer	TCGA (GEPIA) database 50 pairs of tumor tissues and ANCTs	increased	_	high Gleason score, distant metastasis and extracapsular extension	_	[16]
	60 pairs of tumor tissues and ANCTs	increased	_	_	_	[11]
	670 PCa patients and 715 healthy controls	_	_	_	AC and AC/AA genotypes of rs6434568 and AC and AC/CC genotypes of rs16834898 showed a significantly decreased risk of PCa.	[26]
Renal carcinoma	47 pairs of tumor tissues and ANCTs	increased	Shorter OS	TNM stage, tumor size and distant metastasis	_	[1]

be a diagnostic marker. Thus, its levels should be assessed in the plasma samples of patients with different types of cancers to unravel its diagnostic impact.

6. Conclusion

Finally, future studies should detect the functional relationship between PCGEM1 and other non-coding RNAs, particularly miRNAs. Since miRNAs can also be targeted by circular RNAs (circRNAs), miRNAs can provide links between lncRNAs and circRNAs. Identification of this complex network between diverse classes of transcripts would facilitate recognition of mechanisms involved in the carcinogenesis.

CRediT authorship contribution statement

SGF and AB wrote the manuscript and revised it. MT and MM supervised and designed the study. TK and BMH collected the data and designed the figures and tables. All authors read and approved the submitted version.

Declaration of Competing Interest

The authors declare they have no conflict of interest.

Availability of data and materials

The analyzed data sets generated during the study are available from the corresponding author on reasonable request.

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Ethics approval and consent to Participant

Not applicable.

Consent of publication

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