The emerging roles and mechanisms of FAM83H-AS1 in cancer: Pathophysiology and therapeutic implications (Review)

JIN-LONG SHI[1], CHEN-SHI LIN[2], MING-HUI GONG[3] and ZHI-QIANG CAI[2]

[1]Department of Cardio-Thoracic Surgery, The First Affiliated Hospital of Yangtze University, Jingzhou, Hubei 434000, P.R. China;
[2]Department of Oncology, The First Affiliated Hospital of Yangtze University, Jingzhou, Hubei 434000, P.R. China;
[3]Intensive Care Unit, The First Affiliated Hospital of Yangtze University, Jingzhou, Hubei 434000, P.R. China

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Abstract. Long non-coding RNAs (lncRNAs) are key regulators of gene expression at transcriptional and post-transcriptional levels and serve roles in tumour progression, cancer diagnosis and prognosis. Among these, family with sequence similarity 83 member H-antisense RNA 1 (FAM83H-AS1) is an oncogenic lncRNA with elevated expression in several malignancies. FAM83H-AS1 promotes cancer cell proliferation, inhibits apoptosis, enhances migration and contributes to chemoresistance through interactions with microRNA (miR)-136-5p, miR-545-3p, miR-15a miR-10a-5p and signalling pathways such as Wnt/β-catenin and Notch receptor. FAM83H-AS1 may be a promising biomarker for cancer diagnosis and prognosis. The present review summarises the expression, mechanism and potential clinical application of FAM83H-AS1 in cancer diagnosis and prognosis.

Contents

Correspondence to: Professor Zhi-Qiang Cai, Department of Oncology, The First Affiliated Hospital of Yangtze University, 55 Jianghan North Road, Jingzhou, Hubei 434000, P.R. China
E-mail: helloasha@163.com

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1. Introduction

Advancements in medical technology and increased health awareness have improved prognoses for patients with cancer. However, the overall prognosis of cancer remains poor, necessitating the exploration of innovative approaches to enhance patient outcomes. Long non-coding RNAs (lncRNAs) have been identified as key regulators of cancer cell proliferation, apoptosis and metastasis, as well as valuable diagnostic and prognostic biomarkers (1-5). For example, Chen *et al* (1) reported marked upregulation of zinc finger homeobox 4-antisense RNA 1 (ZFHX4-AS1) in adrenocortical carcinoma, with elevated expression being associated with poor prognosis and serving as an independent prognostic risk factor. Suppression of ZFHX4-AS1 expression has been reported to inhibit cancer cell proliferation and migration in adrenocortical carcinoma (1). Similarly, Wu *et al* (2), observed a marked downregulation of long intergenic non-protein coding RNA 1550 (LINC01550) in colorectal cancer (CRC) tissue, which was associated with advanced cancer stages, increased metastasis and reduced overall survival (OS) rate. Overexpression of LINC01550 suppressed cell proliferation, migration, invasion and epithelial-mesenchymal transition (EMT) and promoted apoptosis in CRC cells. These findings highlight the potential of lncRNAs as biomarkers and therapeutic targets to improve cancer prognosis.

Family with sequence similarity 83 member H-antisense RNA 1 (FAM83H-AS1) is an oncogenic lncRNA overexpressed in multiple cancers, including gastric, bladder and liver cancer. Its overexpression has been associated with unfavourable diagnostic and prognostic indicators, promoting cancer cell proliferation, apoptosis and metastasis (6-35). Liu *et al* (6) reported that FAM83H-AS1 expression is markedly elevated in gastric cancer and associated with poor prognosis. Increased FAM83H-AS1 expression enhances proliferation, migration and invasion of gastric cancer cells. To the best of our knowledge, however, comprehensive reviews of the functional mechanisms of FAM83H-AS1 in cancers remain limited. The present review aims to summarise its expression patterns and elucidate its roles in cancer progression, diagnosis and

prognosis. Consequently, the present review may provide a theoretical foundation for the development of cancer therapies.

2. FAM83H-AS1 overexpression in tumours

FAM83H-AS1 is overexpressed across several cancers (Table I). Compared with normal tissue, it exhibits marked upregulation in gastric, bladder, liver, endometrial, CRC, pancreatic, breast, non-small cell lung (NSCLC), ovarian, prostate, glioma and oesophageal cancer tissue. Similarly, elevated expression of FAM83H-AS1 is observed in multiple cancer cell lines (6-34).

3. Roles of FAM83H-AS1 in cancer

FAM83H-AS1 serves a key role in regulating malignant traits in cancer cells, including uncontrolled proliferation, invasion, metastasis and drug resistance, as well as cell cycle progression and apoptosis (6-14,16,18,19,23,25-28,31-34).

FAM83H-AS1 serves as a carcinogenic factor in several cancers by promoting cell proliferation, facilitating cell cycle progression and suppressing apoptosis (Table II). High FAM83H-AS1 expression is associated with enhanced proliferation in gastric cancer (AGS, NCI-N87, MKN74, MKN45), bladder cancer (T24, BK10, J82), liver cancer (SMCC-7721, MHCC97H), endometrial cancer (HEC-1A, Ishikawa), cervical cancer (CaSki), CRC (DLD1, RKO, SW480, HT29), pancreatic cancer (PANC-1, SW1990), breast cancer (MCF7, MDA-MB-231, MDA-MB-468), NSCLC (HCC827, H1650, A549, SPC-A1, PC9), ovarian cancer (CAR-3), prostate cancer (LNCaP, DU145), glioma (U251, U87, 1800) and oesophageal cancer (KYSE30, KYSE510, TE1) cells (6,7,9-14,16,18,19,23,25-28,31-34). FAM83H-AS1 overexpression facilitates cell cycle progression in bladder (T24, BK10, J82) and endometrial cancer (HEC-1A, Ishikawa), lung adenocarcinoma (PC9, H1650), prostate cancer (LNCaP, DU145) and glioma (U251, U87) cells (9,10,12,27,31,32). Furthermore, its overexpression inhibits apoptosis in bladder (T24, BK10) and cervical cancer (CaSki), CRC (SW480, HT29), breast cancer (MCF7), lung adenocarcinoma (A549, SPC-A1) and glioma (U251, U87) cells (9,13,16,19,26,32). *In vivo* studies corroborate these findings, demonstrating that FAM83H-AS1 enhances tumour formation in bladder and endometrial cancer, CRC, pancreatic and breast cancer, NSCLC and lung adenocarcinoma models in nude mice (9,12,14,18,23,25,26).

Roles of FAM83H-AS1 in tumour metastasis. FAM83H-AS1 contributes to cancer metastasis, influencing recurrence and progression (Table III). Overexpression of FAM83H-AS1 promotes cancer cell invasion and metastasis in gastric (AGS, NCI-N87), bladder (T24, BK10, J82), liver (SMCC-7721, MHCC97H), CRC (SW480, HT29), pancreatic (PANC-1, SW1990) and breast cancer (MDA-MB-231, MDA-MB-468), lung adenocarcinoma (A549, SPC-A1, PC9, H1650) and ovarian (ES-2, SKOV-3) and oesophageal cancer (KYSE150, TE1) cells (6,9-11,16,18,23,26,27,29,34). Additionally, elevated FAM83H-AS1 levels promote cell migration in cervical (CaSki), breast (MCF7), ovarian (CAR-3), prostate (PC3, DU145) and oesophageal cancer (KYSE30) (13,19,28,31,33). It also facilitates cell invasion in NSCLC HCC827 cells (25).

Role of FAM83H-AS1 in chemotherapy and radiotherapy resistance. FAM83H-AS1 contributes to cancer progression by enhancing resistance to chemotherapy and radiotherapy (Table II). Elevated FAM83H-AS1 expression has been observed in chemotherapy-resistant gastric cancer (SGC7901/R) compared with parental SGC7901 cells. Silencing FAM83H-AS1 sensitises drug-resistant SGC7901/R cells to cisplatin and 5-fluorouracil, indicating its role in mediating chemoresistance (8). Combining FAM83H-AS1 with oxaliplatin/cisplatin markedly suppresses tumour growth in CRC (14). Moreover, the overexpression of FAM83H-AS1 can trigger metastasis and confer radiation resistance in ovarian cancer cells (30).

4. Signalling mechanism of FAM83H-AS1 in tumours

lncRNAs serve critical roles in cellular signalling by regulating gene expression, the cell cycle and cell differentiation (1-4). They also function as signalling molecules, regulators or mediators in signal transduction. And these interactions influence several physiological and pathological processes, including cancer progression (23,25,31,34). Studies have revealed that FAM83H-AS1 contributes to cancer cell proliferation, apoptosis, migration and chemoresistance by modulating signalling pathways such as microRNA (miR)-136-5p, miR-545-3p, miR-15a, miR-10a-5p, Wnt/β-catenin and Notch receptor (Table IV). Understanding these signalling mechanisms offers insight into cellular signal transduction regulation and presents potential therapeutic targets for cancer treatment.

FAM83H-AS1 serves as a competing endogenous RNA. FAM83H-AS1 promotes cancer progression by competitively binding miR-136-5p, miR-545-3p, miR-15a and miR-10a-5p (23,25,31,34). Han *et al* (23) reported decreased expression of miR-136-5p in breast cancer tissue. Overexpression of miR-136-5p inhibits the proliferation, migration and invasion of breast cancer cells. Silencing FAM83H-AS1 reverses these effects via the miR-136-5p/metadherin axis, thereby promoting tumour growth and metastasis. Zhang *et al* (25) observed that miR-545-3p expression is markedly decreased in lung cancer tissues and inhibition of miR-545-3p increases heparan sulphate 6-O-sulfotransferase 2 (HS6ST2) protein levels, enhancing lung cancer cell invasion. By targeting the miR-545-3p/HS6ST2 axis, FAM83H-AS1 facilitates NSCLC progression. Liu *et al* (31) reported the overexpression of FAM83H-AS1 in prostate cancer cells. FAM83H-AS1 promotes cyclin E2 expression by sequestering miR-15a, thus regulating cell proliferation, cell cycle progression and migration. Additionally, FAM83H-AS1 is markedly upregulated in oesophageal cancer tissue and sequesters miR-10a-5p, promoting Girdin expression, thereby enhancing proliferation, migration and invasion of oesophageal cancer cells (34).

Wnt/β-catenin signalling pathway. The Wnt/β-catenin signalling pathway, also known as the canonical Wnt signalling pathway, is a highly conserved and complex cascade that serves a key role in regulating cell proliferation, differentiation, embryonic development and tissue homeostasis (36). Understanding the mechanisms of this pathway is essential for developing therapeutic interventions targeting this pathway in

Table I. Expression of family with sequence similarity 83 member H antisense RNA 1 in cancer.

Cancer	Expression in tissue or serum	N	Expression in cancer cells	Cancer cell lines	Relative normal cell lines	(Refs.)
Gastric	High	315	High	SNU-1, NCI-N87, AGS, SGC7901, MKN45, MKN74, SNU216, BGC823	GES-1	(6-8)
Bladder	High	122	High	T24, SW780, HT-1197, BK10, HTB-9, RT4, J82	SV-HUC-1	(9,10)
Liver	High	66	High	HepG2, Huh-7, SMCC-7721, MHCC97H	THLE-3	(11)
Endometrial	High	35	-	-	-	(12)
Cervical	-	-	High	CASKI, W12/20863, W12/201402, C-33A	HCK	(13)
Colorectal	High	336	High	SW480, LoVo, HCT116, HT29	HCoEpiC	(14-17)
Pancreatic	High	89	High	MIA PaCa-2, PANC-1, SW 1990, AsPC-1, BxPC-3, Capan-2	HPDE	(18)
Breast	High	187	High	MCF-7, MDA-MB-361, MDA-MB-468, Hs-578, ZR75, BT20, MDA-MB-436, MDA-MB-231, T47D, ZR-75-1	MCF10A	(19-24)
Non-small cell lung	High	201	High	H1299, H1650, HCC827, A549, SPC-A1, H1975, H358, PC9	BEAS-2B, HBE	(25-27)
Ovarian	High	186	High	ES-2, SKOV-3, A2780, SW626	IOSE386	(28-30)
Prostate	High	20	High	LNCaP, 22Rv1, C4-2B, PC-3, DU 145	WPMY-1	(31)
Glioma	High	10	High	U251, U87	1800	(32)
Oesophageal	High	201	High	KYSE410, KYSE510, KYSE520, KYSE30, KYSE170, TE1, KYSE150, Eca109	NE1, Pools	(33,34)

Table II. *In vitro* functional characterisation of family with sequence similarity 83 member H antisense RNA 1 in cancer.

Cancer	Effect on proliferation	Effect on cell cycle	Effect on apoptosis	Effect on metastasis	Effect on chemotherapy or radiotherapy resistance	(Refs.)
Gastric	Promotion	-	-	Promotion	Promotion	(6-8)
Bladder	Promotion	Promotion	Inhibition	Promotion	-	(9,10)
Liver	Promotion	-	-	Promotion	-	(11)
Endometrial	Promotion	Promotion	-	-	-	(12)
Cervical	Promotion	-	Inhibition	Promotion	-	(13)
Colorectal	Promotion	-	Inhibition	Promotion	Promotion	(14,16)
Pancreatic	Promotion	-	-	Promotion	-	(18)
Breast	Promotion	-	-	Promotion	-	(19,23)
Non-small cell lung	Promotion	Promotion	-	Promotion	-	(25-27)
Ovarian	Promotion	-	-	Promotion	Promotion	(28,30)
Prostate	Promotion	Promotion	-	Promotion	-	(31)
Glioma	Promotion	Promotion	-	-	-	(32
Oesophageal	Promotion	-	-	Promotion	-	(33,34)

various diseases. Wang *et al* (8) demonstrated that suppressing FAM83H-AS1 expression inhibits gastric cancer progression via the Wnt/β-catenin pathway. Notably, activation of this pathway counteracts the enhanced chemosensitivity observed in gastric cancer cells following FAM83H-AS1 knockdown. Similarly, Ma *et al* (11) demonstrated that silencing FAM83H-AS1 decreases the expression of β-catenin and WNT1, thereby suppressing liver cancer cell proliferation and migration. In pancreatic ductal adenocarcinoma, Zhou *et al* (18) revealed that FAM83H-AS1 promotes proliferation, invasion and metastasis by stabilising FAM83H mRNA. This stabilisation decreases β-catenin ubiquitination, thereby enhancing pathway activation and tumour progression. Furthermore, dysregulated Wnt/β-catenin signalling influences downstream target genes such as transcriptional regulator c-Myc, cyclin D1 and axin 2. In bladder cancer, FAM83H-AS1 activates unc-51 like kinase

Table III. Family with sequence similarity 83 member H antisense one promotes tumor growth in BALM/c nude mice.

Cancer	Cell line	(Refs.)
Bladder	BK10, T24	(9)
Endometrial	Ishikawa	(12)
Colorectal	RKO, DLD1	(14)
Pancreatic	PANC-1	(18)
Breast	MDA-MB-231, MDA-MB-468	(23)
Non-small cell lung	HCC827	(25)
Lung adenocarcinoma	A549	(26)

3 (ULK3) expression by binding transcription factor c-Myc, contributing to tumour progression (9).

EMT signalling pathway. The EMT signalling pathway serves a key role in cellular transformation into mesenchymal cell types. For example, Liu *et al* (9) reported that FAM83H-AS1 promotes bladder cancer cell proliferation by upregulating N-cadherin, Snai1 and Slug proteins while downregulating E-cadherin expression, thereby facilitating EMT-mediated tumour progression. Similarly, Feng *et al* (34) demonstrated that FAM83H-AS1 enhances oesophageal cancer cell proliferation, migration and invasion by increasing N-cadherin levels and decreasing E-cadherin expression, underscoring its role in EMT activation.

N6 methyladenine (m6A) modification. m6A modification is a prevalent RNA modification that regulates gene expression, stem cell fate determination and the development of numerous diseases, and m6A-modified genes are involved in cancer growth and metastasis (37,38). Liu *et al* (6) reported that the m6A-modified gene WT1 associated protein (WTAP) mediates FAM83H-AS1 expression in an m6A-dependent manner. WTAP inhibition reverses the effects of FAM83H-AS1 overexpression, thereby decreasing gastric cancer cell proliferation, migration and invasion. Additionally, Luo *et al* (14) observed that m6A modification, via polypyrimidine tract binding protein 1 phosphorylation, decreases FAM83H-AS1 expression, thereby inhibiting gastric cancer growth and metastasis. This suggests that m6A can regulate the expression levels of FAM83H-AS1 to affect cancer progression.

Other signalling pathways. In bladder cancer, FAM83H-AS1 suppresses the Hedgehog signalling pathway, whereas ULK3 overexpression activates this pathway. Targeting the Hedgehog signalling pathway effectively counteracts the oncogenic effects of FAM83H-AS1 in bladder cancer cells (9). In CRC, FAM83H-AS1 downregulates SMAD1 gene expression within the TGF-β signalling pathway and promotes CRC cell proliferation via Notch1 in the Notch signalling pathway (15,16). In lung adenocarcinoma cells, FAM83H-AS1 enhances cell proliferation, migration and invasion by modulating the MET proto-oncogene, receptor tyrosine kinase/EGFR signalling pathway (27). Additionally, it interacts with heterogeneous nuclear ribonucleoprotein K to upregulate the anti-apoptotic oncogenes RAB8B and RAB14, thereby inhibiting apoptosis in lung adenocarcinoma cells (26). In endometrial cancer, FAM83H-AS1 promotes tumour progression by increasing the methylation of the CDO1 promoter via DNMT1 recruitment, resulting in decreased CDO1 expression. Decreased CDO1 levels inhibit iron-induced cell death and support cancer growth (12). FAM83H-AS1 stabilises the human antigen R (HuR) protein through cycloheximide, as demonstrated by RNA immunoprecipitation and western blot assays (30). Enhanced HuR expression reverses the effects of FAM83H-AS1 silencing, restoring radiotherapy resistance and metastasis in ovarian cancer cells (30). Finally, in glioma, FAM83H-AS1 regulates the cell cycle progression and proliferation by recruiting enhancer of zeste homolog 2 to the CDK inhibitor 1 (CDKN1A) promoter, leading to increased CDKN1A expression (32).

5. FAM83H-AS1 serves as a potential prognostic biomarker in patients with cancer

FAM83H-AS1 is overexpressed in numerous types of cancer tissue and its elevated expression is associated with poor prognosis in patients with cancer (Table V). Specifically, high levels of FAM83H-AS1 are inversely associated with OS in patients with gastric, bladder and liver cancer, CRC, pancreatic cancer, lung adenocarcinoma, ovarian cancer, glioma and oesophageal squamous cell carcinoma (7-11,15,16,18,20,26-28,30,32-34). Additionally, in gastric cancer and oesophageal squamous cell carcinoma, FAM83H-AS1 overexpression is associated with worse disease-specific survival (7,8,32,34). Elevated FAM83H-AS1 expression is associated with differentiation, depth of invasion and chemotherapy response in gastric cancer. In bladder cancer, its expression is associated with Ki-67 levels, lymph node metastasis, pathological stage, differentiation, invasion pattern and muscular invasion. Similarly, in liver cancer, FAM83H-AS1 expression is associated with tumour size and vascular invasion (7-11,15,16,20,28,30,32-34). These findings highlight FAM83H-AS1 as a promising biomarker for cancer diagnosis and prognosis. Furthermore, targeting FAM83H-AS1 to suppress its expression may slow cancer progression and improve patient prognosis.

6. Conclusions

Studies on FAM83H-AS1 in the context of cancer have consistently demonstrated its notable role as an oncogene (6-34). FAM83H-AS1 does not exhibit dual functionality as both an oncogene and a tumour suppressor gene across different cancers. FAM83H-AS1 serves a key role in pathological processes, substantially contributing to cancer progression (6-34). This unique characteristic warrants further investigation. Mechanistically, FAM83H-AS1 functions as a molecular sponge, binding miRNAs to regulate downstream target genes, thereby exerting oncogenic effects (Fig. 1). Its overexpression is associated with enhanced cell proliferation, migration, invasion and drug resistance, underscoring its potential as both a prognostic marker and therapeutic target in cancer treatment. Moreover, FAM83H-AS1 is associated with adverse prognostic factors such as advanced pathological

Table IV. FAM83H-AS1-miR-mRNA signalling pathways in cancer.

FAM83H-AS1 target miRs	Methods	Target genes	Cancer	(Refs.)
miR-136-5p	Luciferase reporter assay, RT-PCR	MTDH	Breast	(23)
miR-545-3p	Luciferase reporter assay, RIP, RT-PCR	HS6ST2	NSCLC	(25)
miR-15a	Luciferase reporter assay, RT-PCR	CCNE2	Prostate	(31)
miR-10a-5p	Luciferase reporter assay, RIP, RT-PCR	Girdin	ESCA	(34)

miR, microRNA; RT, reverse transcription; FAM83H-AS1, family with sequence similarity 83 member H-antisense RNA 1; NSCLC, non-small cell lung cancer; ESCA, oesophageal cancer; RIP, RNA immunoprecipitation; MTDH, metadherin; HS6ST2, heparan sulfate 6-o-sulfotransferase 2; CCNE2, cyclin E2.

Table V. Family with sequence similarity 83 member H-antisense RNA 1 overexpression is associated with prognosis in patients with cancer.

Cancer	Prognostic indicator	Associated clinical features	(Refs.)
Gastric	OS, DFS	Diagnostic value, differentiation, invasion depth, chemotherapy	(7,8)
Bladder	OS	Ki-67, lymph node metastasis, pathological stage, differentiation, invasion depth, muscularis invasion	(9,10)
Liver	OS	Tumor size, vascular invasion	(11)
Colorectal	OS	TNM stage, tumor size	(15,16)
Pancreatic	OS	-	(18)
Breast	-	Diagnostic value, T stage, clinical stage, lymph node metastasis, distant metastasis, ER status	(20)
Lung adenocarcinoma	OS	-	(26,27)
Ovarian	OS	Tumor grade, distant metastasis, TNM stage, tumor size, FIGO stage, lymph node metastasis	(28,30)
Glioma	OS	Tumor grade	(32)
Oesophageal	OS, DFS	TNM stage, lymph node metastasis, tumor grade	(33,34)

OS, overall survival; DFS, disease-free survival; TNM, tumour, lymph node, metastasis stage; ER, oestrogen receptor; FIGO, International Federation of Gynecology and Obstetrics.

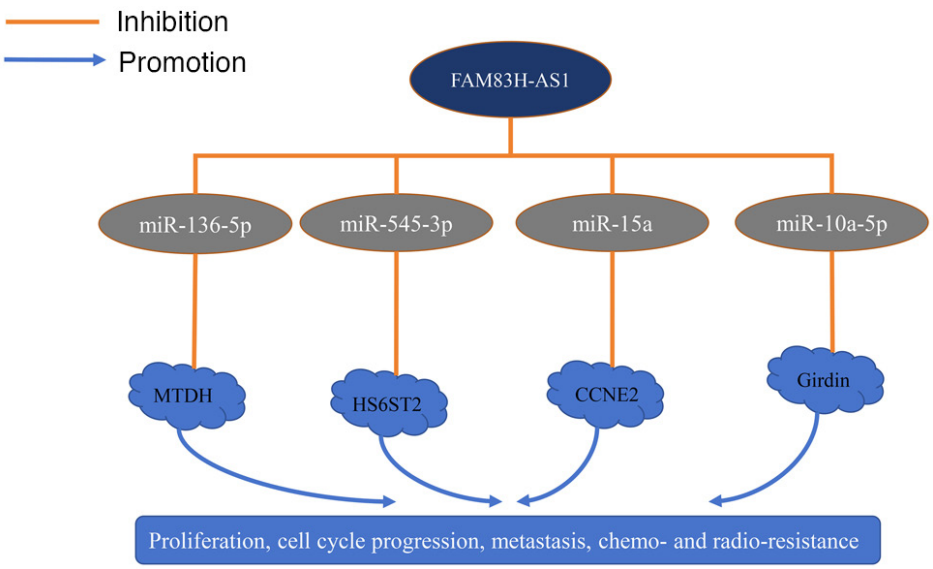

Figure 1. FAM83H-AS1-miR-mRNA signalling mechanism in cancer. FAM83H-AS1, family with sequence similarity 83 member H antisense RNA 1; miR, microRNA; MTDH, metadherin; HS6ST2, heparan sulfate 6-o-sulfotransferase 2; CCNE2, cyclin E2.

staging, increased lymph node metastasis and decreased OS, establishing it as a novel biomarker with notable clinical value for targeted therapy and prognosis assessment.

Despite these findings, several challenges remain. Firstly, the function of FAM83H-AS1 in normal cells is unknown, and the molecular mechanisms (such as Notch, Hedgehog and TGFβ-MET/EGFR signalling) of FAM83H-AS1 vary across different cell types in patients with cancer, complicating the development of universal therapeutic strategies targeting FAM83H-AS1. In the future, the association between FAM83H-AS1 and these mechanisms needs to be further explored. Secondly, most data on FAM83H-AS1 stem from basic research, highlighting the necessity of integrating preclinical findings with clinical studies (6-34). Furthermore, effective treatment options targeting FAM83H-AS1 are limited, emphasising the need for further research into its regulatory mechanisms and drug resistances.

In conclusion, advancing the understanding of the biological roles and regulatory networks of FAM83H-AS1 in cancer is key for its application in diagnostics and therapeutics.

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ZQC conceived the study. JLS reviewed the literature and drafted the manuscript. JLS, CSL and MHG reviewed the manuscript. All authors have read and approved the final manuscript. Data authentication is not applicable.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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