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Article · January 2021

DOI: 10.1016/j.genrep.2021.101392

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MiR-1307: A comprehensive review of its role in various cancer

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ARTICLE INFO

Keywords:
miRNAs
miR-1307
Cancer

ABSTRACT

Various investigations have indicated that miRNAs play a critical role in a wide range of biological functions, like cell differentiation, metabolism of energy, proliferation, and apoptosis. MiRNAs are significantly dysregulated in cancerous cells and malignancies. It has been demonstrated miRNA has two functions in cancer progression: it can stimulate carcinogenesis by blocking tumor suppressors or it functions as a tumor suppressor by helping down-regulation of oncogenes. MiR-1307, which has been recently discovered as a cancer-associated miRNA, is considered a risk factor for the development of metastatic types of cancers. MiR-1307 levels in serum are increased in breast cancer and can be used to diagnose the disease early. Data show that miR-1307-3p is an oncogenic miRNA with a significant contributory role in the development and progression of the thorough targeting SMYD4. Furthermore, this miRNA helps to the proliferation of cancer cells include of breast, ovarian, colorectal, lung, and so on. MiR-1307 overexpress in gastric cancer cell lines and its interaction with DAB2 protein lead to promote the behavior of tumoral cells such as proliferation and migratory and metastasis. In hepatocellular carcinoma (HCC) miR-1307-3p promotes HCC proliferation, invasion, and advancement by reducing DAB2 protein. In ovarian cancer, miR-1307 influence the cell cycle of tumoral cells and decline the response to chemotherapy drugs in ovarian cancer by regulation of the CIC (capicua transcriptional repressor) gene. Another mechanism for miR-1307-3p was seen in colon adenocarcinoma miR-1307-3p can be bind to the 3′-untranslated area of TUSC5 and control progression and metastasis of colon adenocarcinoma cells. MiR-1307-5p can binds to TRAF3 and stimulates the NF-B/MAPK pathway to enhance the proliferation of lung tumor. In this review study, we try to recapitulate the role of miR-1307 in the process of pathogenesis and treatment of various cancers according to recently published studies.

1. Introduction

MicroRNAs (miRNAs), as a group of small non-coding RNAs are involved in regulating a variety of biological events like differentiation, proliferation, apoptosis, physiology, metabolism, and diseases (Abkhooie et al., 2021; O'Brien et al., 2018; He et al., 2019; Wang et al., 2018). It has been shown that miRNAs are significantly dysregulated in cancerous cells and malignancies (Chen et al., 2019a; Peng and Croce, 2016; Si et al., 2019; Daoud et al., 2019). Abnormalities of chromosome changes in the regulation of transcription, epigenetic modifications, and deficiencies in the biogenesis machinery of miRNAs are considered the underlying mechanisms (Assis et al., 2021; Abkhooie et al., 2021; Morales et al., 2017; Tomasetti et al., 2019). Comparing malignant cells to the normal ones revealed that abnormal expression of miRNA is frequently associated with changes in genomic copy numbers of miRNAs and location of genes (translocation amplification or deletion). Lin-4 was the first miRNA that was identified in *Caenorhabditis elegans* (*C. elegans*) by Ambros et al. (Maatouk and Harfe, 2006).

According to evidence collected, microRNAs are very influential on formation of a tumor, progress and its treatment. About 50% of miRNAs are placed in friable spots concerned with tumor or genomic areas, which shows their great importance (Yang et al., 2017). MicroRNAs

Abbreviations: miRNA, microRNA; B-CLL, B-cell chronic lymphocytic leukemia; TGF signaling, transforming growth factor signaling; RCC, renal cell carcinoma; TKI, tyrosine kinase inhibitors; SMYD4, SET and MYND domain contained 4; DAB2IP, DAB2 protein; HCC, hepatocellular carcinoma; SEC14L2, SEC14 Like Lipid Binding 2; ENG, Endoglin; MEIS2, Meis Homeobox 2; LATS1, Large Tumor Suppressor Kinase 1; CIC, capicua transcriptional repressor; Colon adenocarcinoma, COAD; TUSC5, Tumor suppressor candidate 5; PRRX1, Paired Related Homeobox 1; ISM1, Isthmin 1; TYMS, Thymidylate Synthetase; RFS, recurrence/relapse-free survival; NSCLC, Non-Small Cell Lung Cancer; TRAF3, TNF Receptor Associated Factor 3; NF-κB, nuclear factor-κB; MAPK, mitogen-activated protein kinase.

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https://doi.org/10.1016/j.genrep.2021.101392
Received 31 August 2021; Received in revised form 28 September 2021; Accepted 5 October 2021
Available online 12 October 2021

which have abnormal expression, are concerned with most tumors, and directly or indirectly interact with oncogenes and tumor suppressive genes to adjust their state. In addition, microRNAs act as oncogenes or tumor-inhibiting genes in various cellular functions, including angiogenesis, proliferative apoptosis, infestation and tumor metastasis (Liu, 2012). MicroRNAs are symptoms of possible tumor and are predicted to be new targets for treatment. Several research groups have identified a number of uncontrolled microRNAs in cancers, one of which is MicroRNA 1307 (Eun et al., 2020). According to recent research, microRNA 1307 can cause more multiplication of prostate cancer cells by targeting FOXO3A that microRNA-1307-3p through dropping SMYD4 (SET and MYND Domain containing protein 4) causes breast cancer cells to be created and increased, and that microRNA 1307 was effective in the development and metastasis of liver cancer or HCC (hepatocellular carcinoma) (Qiu and Dou, 2017; Eun et al., 2020). It was clear that, in liver cancer or HCC tissues, in large tumors at advanced stage of the tumor, microRNA 1307 was increased and a less favorable overall clinical outcome was achieved (Eun et al., 2020). An analysis within and outside of a living organism showed that the destruction of miR-1307-3p through adjusting Disabled Homolog 2 interacting protein to a specific level or condition, Stopped the development of the tumor (Yue et al., 2020). In this review study, we try to summarize the role of miR-1307 in the process of pathogenesis and treatment of various cancers according to recent published studies.

2. MiRNA biology and the miR-1307

MicroRNAs, which were identified in 1993, are small non-coding RNAs, modulating the expression of genes by directly interacting with the 3′ untranslated regions of target mRNAs (Stefani and Slack, 2008). Various investigations have indicated that miRNAs have an key character in a wide range of biological functions, like cell differentiation, metabolism of energy, proliferation, and apoptosis (Kabekkodu et al., 2018). Notably, it has been demonstrated miRNA has two functions in cancer progression: it can stimulate carcinogenesis by blocking tumor suppressors or it functions as a tumor suppressor by helping down-regulation of oncogenes (Mott, 2009). Investigating the association of miRNA with ovarian cancer has been recently received attention. It has been found that miRNA blocks pro-apoptotic signaling pathways in ovarian cancer, leading to chemoresistance (Li and Mahato, 2014). Moreover, losing Let-7 is thought to result in chemoresistance in ovarian cancerous cells. Additionally, miR-300 up-regulation through TGF signaling may prevent cellular apoptosis, resulting in promoting chemoresistance in ovarian cancerous cells (Ali et al., 2011).

miRNA dysregulation may play a significant role in determining ovarian cancer cell susceptibility to chemotherapy (Mehrgou and Akouchekian, 2017; Fu et al., 2012). Moreover, miR-1307-3p, which has been recently discovered as a cancer-associated miRNA, is considered a risk factor for the development of metastatic renal cell carcinoma (RCC) when treated with tyrosine kinase inhibitors (TKI) (Zhou et al., 2015; Kovacova et al., 2018). MiR-1307-3p levels in serum are increased in breast cancer and can be used to diagnose the disease early. Furthermore, this miRNA helps to the proliferation of breast cancerous cells in vitro and tumor development in vivo (Chen et al., 2017b; Martinez-Gutierrez et al., 2020). Furthermore, miR-1307-3p has been linked to capecitabine-based treatment in colon cancer (Chen et al., 2017a). Nevertheless, miR-1307-3p expression and its biological role in HCC remained to be clear (Eun et al., 2020). MiRNA action was explained in detailed with a Fig. 1.

2.1. MiR-1307 and breast cancer

Different researchers have identified a variety of dysregulated miRNAs in breast cancer, including miR-1307-3p. Shimomura et al. discovered an increase in miR-1307-3p in the serum of individuals with breast cancer (Eun et al., 2020). Moreover, Sanghak Han's research has revealed a novel mechanism for the development and progression of breast cancer (Han et al., 2019). Data show that miR-1307-3p is an oncogenic miRNA with a significant contributory role in the development and progression of the thorough aiming SMYD4. Furthermore, miR-1307-3p might be a promising therapeutic candidate for breast tumor treatment (Han et al., 2019; Humphries et al., 2019).

Remarkably, miR-1307-3p has been indicated higher expression in specimens with breast cancer rather than the normal ones (Han et al., 2019; Sathipati and Ho, 2018; Satomi-Tsushita et al., 2019). Moreover, there was a significant association between the increased levels of expression and decreased overall survival rate amongst patients suffering from breast cancer (AiErken et al., 2017).

Furthermore, researcher showed that miR-1307 brings about cancer formation in non-tumorigenic human mammary epithelial cells of nude

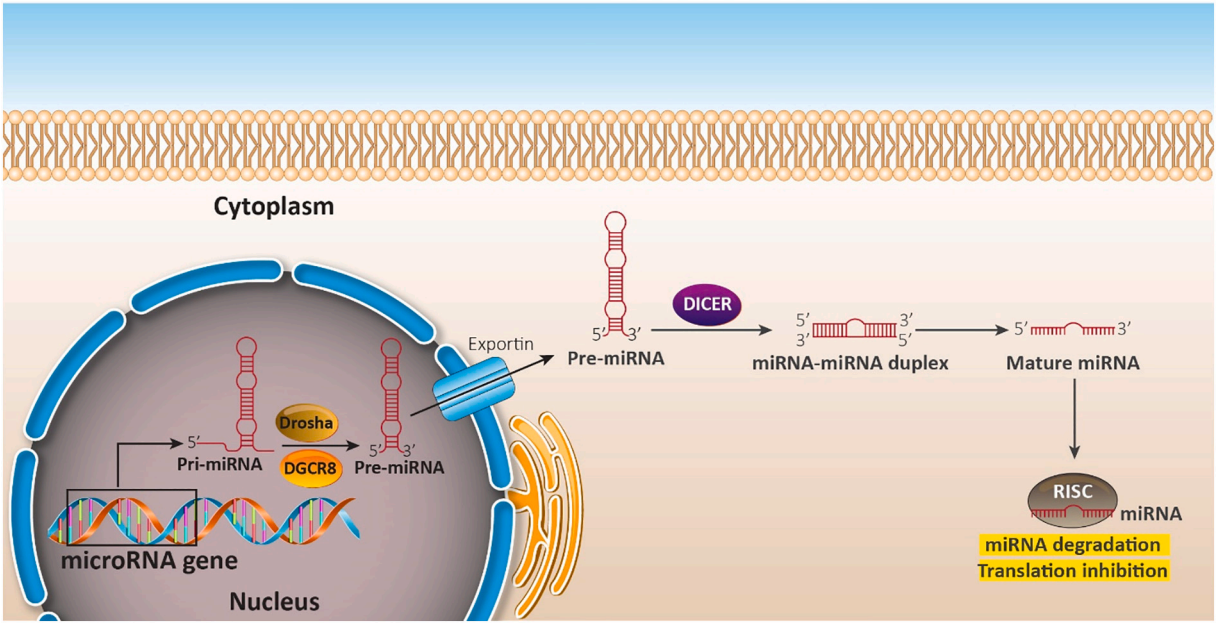

Fig. 1. MiRNA action.

mice and stimulates cell proliferation through affecting tumor suppressor genes SET and MYND domain contained 4 (SMYD4) (Ozawa et al., 2020; Han et al., 2019). Akihiko Shimomura et al, revealed that levels of miRNA were examined between breast tumor sufferers and non-affected ones, in which five different miRNAs were combined including miR-1246, miR-1307-3p, miR-4634, miR-6861-5p, and miR-6875-5p (Shimomura et al., 2016). This miRNAs combination was discovered to be practical in breast cancer diagnosis (Frères et al., 2016).

Wang et al. have recently shown that inhibition of miR-1307 reduces the cytotoxicity of cisplatin in breast cancer cells and increases resistance to chemotherapy. The miR-1307 induces hypersensitivity to cisplatin in tumoral cells by decreasing the MDM4 expression. Therefore, miR-1307 expression is inversely related to MDM4 (Yan and Yan, 2014; Wang and Zhu, 2018). The sensitivity, specificity, and accuracy of the miRNAs combination were 97.3%, 82.9%, and 89.7%, respectively for breast cancer detection in the analysis cohort (Shimomura et al., 2016). Moreover, this combination was capable of breast cancer detection at an early stage Table 1. (Shimomura et al., 2016; Jang et al., 2021).

2.2. MiR-1307 and gastric cancer

MiR-1307-3p upregulation in gastric cancer has been shown. Moreover, abnormal expression and function of miR-1307 in hepatocellular carcinoma, breast tumor, as well as colon cancer are demonstrated Fig. 2 (Zheng et al., 2019; Vaghari-Tabari et al., 2020). Some researchers have found a link between miR-3117 and stomach cancer (Sarabandi et al., 2021). Furthermore, according to research performed by Sahel Sarabandi et al., the miR-3117 rs7512692 and miR-1269 rs73239138 variants are associated with tumor grade, and progesterone receptor status, respectively (Sarabandi et al., 2021). Their research indicated that miR-1307, miR-1269, and miR-3117 polymorphisms may have a critical role in the susceptibility of the Iranian population (Sarabandi et al., 2021). Yanhui Ma and colleagues discovered a substantial increase the level of miR-1307 in gastric tumor tissues as compared to normal ones (Ma et al., 2021). MiR-1307-3p expression was also shown to be strongly linked to the TNM stage and weak prognosis in patients (Huang et al., 2021). As a result, miR-1307-3p may act as a tumor inducer in gastric tumor (Yang et al., 2020). Moreover, its overexpression considerably induces the proliferation, migratory, and invasive capacities of gastric cancerous cells. These findings showed that inhibiting miR-1307-3p might be a possible therapeutic approach for the treatment of gastric cancer (Ma et al., 2021).

Also, miR-1307 overexpress in gastric cancer cell lines and its interaction with DAB2 protein lead to promote the behavior of cancerous cells such as proliferation and migratory and metastasis (Ma et al., 2021).

Many genes considered as purposes for miR-1307-3p and involve multiple biological signaling pathways, such as cellular proliferation, differentiation, metastasis, and apoptosis. Accordingly, abnormal expression and function of miR-1307-3p can be lead to hepatocellular carcinoma, ovarian cancer, lung cancer, as well as colon cancer.

2.3. MiR-1307 and hepatocellular carcinoma (HCC)

Numerous studies have revealed that microRNAs (miRNAs) show a crucial role in regulating the onset and development of hepatocellular carcinoma (HCC) (Chen et al., 2019b; Eun et al., 2020; Jing-Rui et al., 2020). The expression of miR-1307-3p in HCC and the effect of its expression on malignant manners of cancer cells have been studied (Chen et al., 2019b). The studies show that miR-1307-3p expression was significantly higher in HCC related to adjacent noncancerous tissues (Chen et al., 2019b; Eun et al., 2020). Also, the patients with intravenous infusion showed much higher miR-1307-3p in HCC tissues. While deletion of miR-1307-3p expression inhibits the proliferation and invasion of MHCC97H and HCCLM3 cells and suppresses the growth and metastasis of HCCLM3 cells (Chen et al., 2019b). Also, the analysis of nine lncRNAs by Zhan et al. discovered that high expression of miR-183-5p, miR-1307-3p, and their corresponding lncRNAs occurred in HCC patients (Zhan et al., 2021).

DAB2 protein (DAB2IP) is a straight target of miR-1307 that its expression is inversely related to the miR-1307 in HCC tissues. MiR-1307 promotes HCC proliferation, invasion, and advancement by reducing DAB2IP (Chen et al., 2019b). Investigation using Target Scan display down-regulation of SEC14L2 and ENG occurs in metastasis in HCC by downstream pathways of miR-1307 (Eun et al., 2020). Wang 2019 demonstrated that decreased expression of hsa_circ_0091570 promotes tumor growth in cases with HCC. The results show that inhibition of miR-1307 expression can be associated with decreased expression of hsa_circ_0091570 and this is due to the effect of miR-1307 on ISM1 expression (Wang et al., 2019). Guan et al., 2019 indicate that miR-1307-3p can be induced by MEIS2 and downregulates LATS1 in HCC. This role of MEIS2 is expert by the miR-1307-3p/LATS1 axis and helping YAP nuclear translocation along with its association with Wnt/β-catenin signaling (Guan et al., 2019).

3. MiR-1307 and ovarian cancer

Ovarian tumor is the prevalent female malignant tumor universal and the 5-year survival rate for patients with progressive ovarian tumor is 30%, despite the application of chemotherapy (Ferreira et al., 2020; Bast et al., 2020). Lack of early detection of ovarian cancer cause that the mortality rate is very high and the disease is usually diagnosed in the progressive stages (Chen et al., 2017b). Examination of ovarian tumor cell line A2780/Taxol shows that miR-1307 expression is increased. Chen et al. reported that increased expression of miR-1307 increased resistance to chemotherapy in ovarian cancer (Chen et al., 2017b). The expression of miR-1307 is inversely related to the expression of ING5,

Table 1
Effect of miR-1307, on genes and pathways in various cancer.

Organ	Sample	Targets	Signaling pathway	MiR-1307 expression	Reference
Breast	Tissue/Cell lines	SEC14L2, ENG		Increase	(Eun et al., 2020)
Breast	Cell lines	SMYD4		Increase	(Han et al., 2019)
Colon	Tissue/Cell lines	ISM1	Wnt3a/β-catenin		(Zheng et al., 2019)
Colon	Cell lines	TUSC5		Increase	(Yue et al., 2020)
Colon	Cell lines	PRRX1		Increase	(Yang et al., 2020)
Gastric	Tissue/Cell lines	DAB2IP		Increase	(Ma et al., 2021)
Liver	Tissue/Cell lines	DAB2IP		Increase	(Chen et al., 2019b)
Liver	Tissue/Cell lines	SNHG3, LINC00205	lncRNA–miRNA–mRNA	Increase	(Zhan et al., 2021)
Liver	Tissue/Cell lines	ISM1		Increase	(Wang et al., 2019)
Liver	Tissue/Cell lines	MEIS2C/D	Wnt/β-catenin	Increase	(Guan et al., 2019)
Ovarian	Cell lines	ING5		Increase	(Chen et al., 2017b)
Ovarian	Tissue/Cell lines	DAPK3		Increase	(Wang et al., 2021)
Ovarian	Tissue/Cell lines	CIC, ETV4, ETV5	miR-1307/CIC axis	Increase	(Zhou et al., 2019)
Lung	Cell lines	TRAF3	NF-κB/MAPK	Increase	(Du et al., 2020)

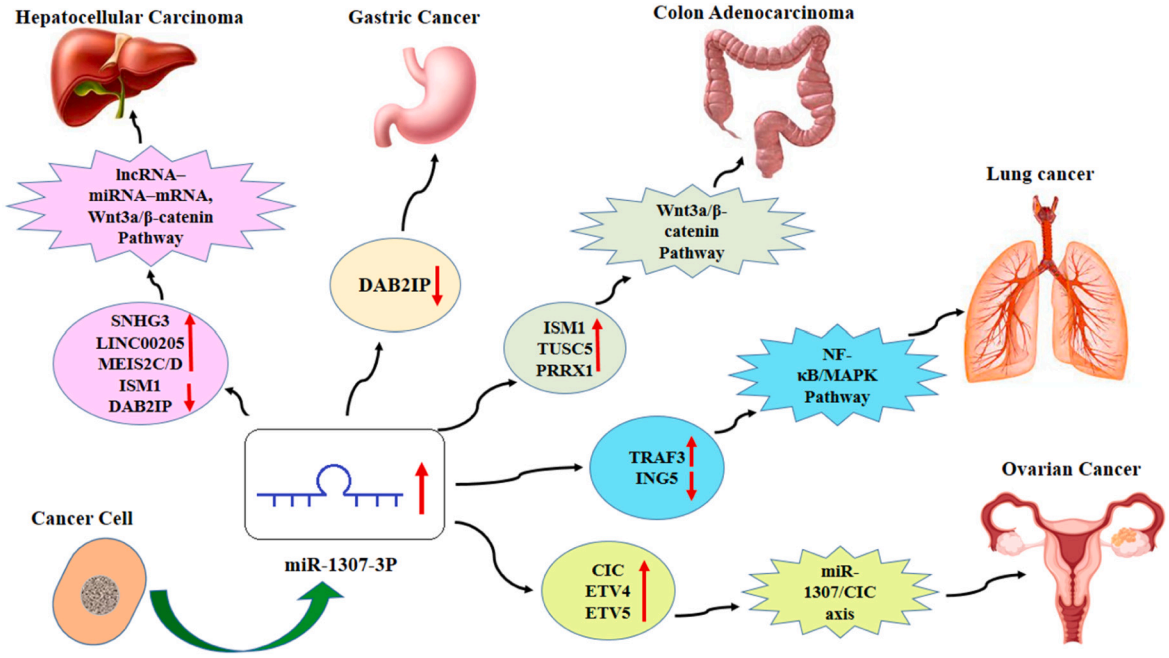

Fig. 2. Overview of some target genes of MiR-1307-3p.

whereas ING5 reduces the proliferation of cancer cells and also increases apoptosis (Chen et al., 2017b; Li et al., 2015; Gou et al., 2015; Cengiz et al., 2010).

Zhou et al. reveal that DAPK3 is a nuclear protein with serine/threonine kinase activity (Zhou et al., 2015). Upregulation of DAPK3 are associated with increased survival and metastasis of tumor cells and led to resistance to chemotherapy. Studies show that miRNA-1307 can target DAPK3 and could be considered a candidate in the treatment of gynecological cancers such as uterus, ovarian, and placenta (Zhou et al., 2015; Wang et al., 2021). Also, the other studies proposed that miR-1307 may influence the cell cycle of tumoral cells and increase resistance to chemotherapy drugs in ovarian cancer by regulation of the CIC (capicua transcriptional repressor) gene (Zhou et al., 2019).

4. MiR-1307 and colon adenocarcinoma (COAD)

Colon adenocarcinoma (COAD) is the prevalent malicious tumor of the gastrointestinal tract and the research reported that abnormal expression of genes happens as an outcome of abnormal expression of microRNA and is complicated in the rise of colon adenocarcinomas (Yue et al., 2020; Ghafouri-Fard et al., 2021; Lizarbe et al., 2017).

The level of miR-1307 expression increase in CRC cells. MiR-1307 can be bind to the 3′-untranslated area of TUSC5 and control progression and metastasis of CRC cells by targeting TUSC5 (Yue et al., 2020). In the presence of the rs7911488-T allele, the level of miR-1307 increases, and the expression of PRRX1 decreases, followed by the progression of CRC (Yang et al., 2020). Zheng and colleagues discovered that overexpression of ISM1 can be stop the start of the Wnt3a/-catenin signaling path (Zheng et al., 2019). This suggested that miR-1307–3p might influence Wnt3a/-catenin signaling pathway activity by selectively regulating ISM1 expression, therefore influencing cell apoptosis and proliferation. Finally, miR-1307–3p is expressed at a low level in COAD tissues (Hao et al., 2017). Chen et al. recognized that rs7911488 C-allelic pre-miR-1307 can decline the response to capecitabine chemotherapy by decreasing miR-1307-3p expression and increasing of TYMS in colon tumor (Chen et al., 2017a).

5. MiR-1307 and lung cancer

According to a study, the expression patterns were examined for their relationships with subtypes of tumor, lung cancer, brain metastases, and recurrence/relapse-free survival (RFS) (Du et al., 2017). The expression profiles of 171 miRNAs, consisting of Let-7 family members and miR-205, differed considerably between squamous cell carcinoma and lung adenocarcinoma. There are ten miRNAs related to brain metastasis, such as miR-145, which inhibits cell metastasis and invasion (Jiang et al., 2014). There are two miRNA signatures, greatly prognostic of RFS. The primary group comprised 34 miRNAs obtained from 357 patients with stage I NSCLC regardless of subtypes of tumor, whereas the another included 27 miRNAs specific to adenocarcinoma (Lu et al., 2012). The two signatures were confirmed in separate datasets with 170 patients with stage I NSCLC by the use of fresh frozen and/or formalin-fixed paraffin-embedded tissues (Lu et al., 2012).

In another study focused on lung cancers, it was designated that miR-1307-5p is considerably involved in lung adenocarcinoma progression (cellular growth, proliferation, and invasion) (Du et al., 2020). It was found that miR-1307-5p binds to TRAF3 and stimulates the NF-B/MAPK pathway to enhance the proliferation of lung tumor. As a result, the miR-1307-5p/TRAF3/NF-kB/MAPK axis might be a novel aim for lung adenocarcinoma therapy and a possible prognostic factor, in the future (Du et al., 2020).

6. Prospective direction of miR-1307–based treatment

MiRNAs are recognized as different analytical, predictive, and therapeutic devices, and various studies have been devoted to the identifying effect of miRNAs in tumor (Ma et al., 2021; Kalinowski et al., 2014; Yadav et al., 2017). By the advance of molecular biology, several dysregulated miRNAs are recognized that can be play role in the advancement of various cancer (Ma et al., 2021).

Also, According to recent reports, the miR-1307-3p shows both oncogenic (Han et al., 2019) and anti-oncogenic activity (Hashimoto et al., 2021; Zheng et al., 2019). MiR-1307 has been known as an upregulated miRNA that helps to the proliferation of cancer cells include of breast (Frères et al., 2016), ovarian (Zhou et al., 2015), colorectal, lung (Du et al., 2020), and so on. Studies by Nguyen et al. display that

the level of miR-1307-3p and miR-1246 can be helpful in the primary identification of breast cancer (Nguyen et al., 2020). Also, the combination of miR-1307 with four other miR, including miR-1246, miR-4634, miR-6861, and miR-6875 can be utilized to differentiate breast cancer from other cancers (Nguyen et al., 2020; Shimomura et al., 2016). Ma et al. showed that the proliferation and invasion of tumoral cells are directly related to the expression of miR-1307 and that gastric cancer progresses with the increase of miR-1307. As a result, inhibition of miR-1307 can be used to control and treat cancer disease (Ma et al., 2021). Hashimoto et al. reported that the first diagnosis of thirteen types of solid tumors was possible by checking the expression of miR-1307 in human serum (Hashimoto et al., 2021).

So, miR-1307-3p can be regarded as a biomarker that can help in the development and detection of multiple types of tumors.

7. Conclusion

Tumors comprise six characterizations including maintaining proliferative signaling, escaping growth suppressors, opposing cell death, letting replicative permanence, initiating aggression, metastasis and, provoking angiogenesis. Abnormal expression of miRNAs such as miR-1307-3p can affect the symbol markers of these cancerous cells and cause the development, progression and, antitumor drug resistance of cancer (Paul, 2020; Fares et al., 2020). Also, miR-1307-3p can be used as a biomarker for diagnostic, prognostic, and cure of cancer.

8. Consent for publication

This is the review work of the authors. The work characterized has not been presented to another journal, and all authors enumerated cooperated in the writing of document.

Declaration of competing interest

None.

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