Abstract: Although ignored in the past, with the recent deepening of research, significant progress has been made in the field of non-coding RNAs (ncRNAs). Accumulating evidence has revealed that microRNA (miRNA) response elements regulate RNA. Long ncRNAs, circular RNAs, pseudogenes, miRNAs, and messenger RNAs (mRNAs) form a competitive endogenous RNA (ceRNA) network that plays an essential role in cancer and cardiovascular, neurodegenerative, and autoimmune diseases. Gastric cancer (GC) is one of the most common cancers, with a high degree of malignancy. Considerable progress has been made in understanding the molecular mechanism and treatment of GC, but GC’s mortality rate is still high. Studies have shown a complex ceRNA crosstalk mechanism in GC. lncRNAs, circRNAs, and pseudogenes can interact with miRNAs to affect mRNA transcription. The study of the involvement of ceRNA in GC could improve our understanding of GC and lead to the identification of potential effective therapeutic targets. The research strategy for ceRNA is mainly to screen the different miRNAs, lncRNAs, circRNAs, pseudogenes, and mRNAs in each sample through microarray or sequencing technology, predict the ceRNA regulatory network, and, finally, conduct functional research on ceRNA. In this review, we briefly discuss the proposal and development of the ceRNA hypothesis and the biological function and principle of ceRNAs in GC, and briefly introduce the role of ncRNAs in the GC’s ceRNA network.

Keywords: non-coding RNAs; competitive endogenous RNA; gastric cancer

1. Introduction

A non-coding RNA (ncRNA) is a type of RNA that does not have the function of a coding protein [1]. NcRNAs, which account for 98% of the human genome, include ribosomal RNAs (rRNAs), short ncRNAs, circRNAs, pseudogenes, and many IncRNAs [2]. For a long time, IncRNAs, circRNAs, and pseudogenes were regarded as useless components in the genome. In 1976, scholars discovered the existence of circRNA (pathogenic single-stranded circular virus) in higher plants [3]. In 1977, the first pseudogene was discovered in the Xenopus genome [4]. In the 1990s, researchers discovered an imprinted gene, lncRNA H19, which forms the H19/IGF-2 imprinted gene group with the similarly-located insulin-like growth factor 2 [5]. At the same time, other studies showed that the lncRNA XIST can participate in the transcriptional regulation of genes on sex chromosomes [6,7]. Thus, ncRNA began receiving attention. HOTAIR, another IncRNA, was discovered in 2007. Studies have shown that it can enhance the PRC2 activity of the HOXD locus and participate in PRC2-mediated chromatin silencing [8]. In 2013, a study revealed, for the first time, that circRNA could be used as a miRNA sponge to adsorb miRNA, thereby affecting gene expression [9]. With the deepening of research, it was found that IncRNAs, circRNAs, and pseudogenes can play biological functions in immune response [10], nerve conduction [11], growth and development [11], and stress response [12]. With the help of microarray and RNA sequencing technology, people have ascertained that IncRNAs, circRNAs, and pseudogenes are involved in regulating various tumor cell biological activities [13].
It was discovered that ncRNAs contain miRNA response elements (MREs) and act as a miRNA sponge, and an increasing number of studies have shown that they participate in the formation of a complex regulatory network. The ceRNA hypothesis proposes that certain transcripts, such as IncRNAs, circRNAs, pseudogenes, and mRNAs, have MREs in common, regulating the transcription of gene expression through competitive binding of miRNAs [14]. Thus, they are each other’s ceRNA. It has been 10 years since the ceRNA hypothesis was put forward, and research on ceRNA has been steadily increasing yearly. Researchers found that the ceRNA network plays an important role in cardiovascular diseases such as myocardial hypertrophy [15,16], myocardial infarction [17,18], atherosclerosis [19–22], neurodegenerative diseases such as Alzheimer’s disease [23,24], Parkinson’s disease [25,26], Huntington’s disease [27,28], and neuroimmune diseases such as progressive muscular dystrophy and cocaine syndrome [29–31]. Therefore, studying the ceRNA regulatory network is of great significance in understanding the diseases’ occurrence and development, and improving clinical diagnosis, treatment methods, and prognosis.

Cancer became the main cause of death and the single most important obstacle to increasing people’s life expectancy in the 21st century. Cancer is mainly related to genetic factors [32], immune factors [33], endocrine factors and other endogenous factors, as well as living habits [34,35], environmental pollution [36], biological factors [37], and other exogenous factors. ceRNAs play an important role in cancer progression, including gastric cancer (GC), colon cancer, liver cancer, breast cancer, and lung cancer [38,39]. GC is a common cancer worldwide. Studies have found that IncRNAs, circRNAs, and pseudogenes such as ceRNAs can participate in biological behaviors such as GC proliferation, differentiation, and cell resistance. Therefore, an increasing number of studies on the ceRNA network in GC are expected to provide new ideas for understanding the mechanism of GC occurrence and development, and simultaneously provide direction for finding new targets for treating GC.

2. Gastric Cancer

As the fifth-most-common cancer and the third-leading cause of cancer death worldwide, GC is a deadly digestive system disease afflicting many people. GC was responsible for over 1,000,000 new cases in 2018 and an estimated 783,000 deaths (equating to one in every 12 deaths globally) [38,40].

Global cancer statistics 2018 show that GC incidence and mortality in Asia rank first by world region. Factors that cause this disease include Helicobacter pylori infection, age, high salt intake, and low fruit and vegetable diets. Alcohol consumption and active tobacco smoking are also established risk factors [38].

However, the gold standard for GC diagnosis is endoscopic biopsy plus enhanced computed tomography. Many patients resist examination due to the insidious onset, unobtrusive symptoms, and invasive examination methods. Furthermore, since early GC has nonspecific symptoms, most GC patients are diagnosed at advanced stages, and the 5-year survival rates range between 20% and 30% [41,42].

Surgical treatment plus chemotherapy remains the first-line approach to provide a cure for GC. Despite advances in surgical techniques, radiotherapy, chemotherapy, and neoadjuvant therapy, chemotherapy resistance or drug resistance is still an important issue that needs to be faced because cancer cells will form a mechanism to counteract the effects of chemotherapy drugs, leading to more clones and aggressiveness, and eventually a poor prognosis. Chemoresistance can be inherent and acquired, and it is a multi-factor event, including dysregulation of key signaling pathways, acquired mutations, and DNA damage responses [43].

Therefore, exploring the pathogenesis and looking for key factors to guide diagnosis and treatment has always been a research focus.

The occurrence and development of GC is a multi-stage and multi-factor process, and its pathogenesis is complex. The current research shows that its occurrence is often related to abnormal transcription. This abnormality is not limited to abnormal protein-coding
RNA (mRNA) levels and includes abnormalities in the regulatory ability of ncRNA in the genome. Studies have shown that the cancer stem cell (CSC) is one of the main reasons for the failure of cancer treatment. The expression of miRNAs plays an important role in the maintenance of stem/progenitor cells. The dysregulation of miRNAs in gastric cancer stem cells (GCSCs) is closely related to the occurrence and development of gastric cancer [44].

3. ceRNAs

In 2007, Ebert et al. artificially synthesized miRNA inhibitors called miRNA sponges. With an increasing number of experimental verifications and the discovery of endogenous miRNA sponges, in 2011, Salmena et al. proposed the ceRNA hypothesis for the first time. It was expounded that in addition to the traditional miRNA→RNA mode of action, there is also an RNA–miRNA–mRNA regulation mode [14,45–47].

Here, “ceRNA” does not refer to a specific RNA but to a brand-new mode of gene expression regulation, describing a mode of action of RNA. The mechanism of ceRNA is that when the ceRNA expression is silenced, mRNAs are transcribed and exported to the cytoplasm, where they are targeted by the miRNA-mediated silencing complex (miRNA–RISC), resulting in accelerated degradation, blocking of translation, and reduction of gene expression; Second, when the ceRNA expression is activated, there will be competition for miRNA targeting and binding to the RISC complex, reducing miRNA inhibition; the miRNA–RISC complex is isolated from the gene, resulting in increased gene expression.

ceRNAs use similar MREs to bind miRNAs, thereby indirectly regulating genes’ expression competitively. This competitive miRNA binding effect is also called miRNA sponge action. According to this theory, any RNA that contains MREs may be a ceRNA, its core is miRNAs, and its members include IncRNAs, circRNAs, mRNAs, and pseudogenes. Among the RNAs that can be used as ceRNAs, those that regulate tumor progression play an important role [48,49].

Besides, there are multiple MREs on each mRNA so that each mRNA can have multiple miRNA pathways. Each miRNA has multiple ceRNAs, thus forming the last “many-to-many” ceRNA networks (ceRNETs). Compared with the miRNA regulation network, ceRNETs are more sophisticated and complex, involving more RNA molecules. When ceRNAs are abnormally expressed, they affect the expression of multiple target genes in the body and further influence cancer progression.

Research shows that ceRNAs play critical roles in the development and progression of cancers. Considering the complexity of the network of ceRNAs, this research is still in its infancy. At present, the most effective way to reveal the ceRNA function in cancer is to build ceRNETs first. A common research method is to obtain samples from different tissues, screen different miRNAs, IncRNAs, and mRNAs through microarray or sequencing technologies or the use of databases to collect information, screen differentially expressed RNAs, construct ceRNETs, extract key networks, and finally perform functional enrichment analysis and survival analysis to discover genes related to cancer development and prognosis [50–52].

The most commonly used databases are the Cancer Genome Atlas (TCGA) database and Gene Expression Omnibus (GEO) microarray datasets. Furthermore, researchers have also established some dedicated tools to facilitate the identification of ceRNA networks, including ceRDB, Line2GO, starBase v2.0, InCeDB, and Cupid. Details and resources are summarized in chronological order in Table 1. The functions of these tools are different. Researchers should choose according to their needs.
| Tool Name | Functions | Website | Reference |
|-----------|-----------|---------|-----------|
| ceRDB | Predict ceRNAs for specific mRNAs targeted by miRNAs by examining the co-occurrence of miRNA response elements in the mRNAs on a genome-wide basis. MicroRNA–mRNA and microRNA–lincRNA interaction data were integrated to generate lincRNA functional annotations based on the ‘competing endogenous RNA hypothesis’. | [http://www.oncomir.umn.edu/cefinder/](http://www.oncomir.umn.edu/cefinder/) (accessed on 20 May 2021) | [53] |
| Linc2GO | A database of human lncRNAs (from GENCODE 19 version) that can potentially act as ceRNAs. | [http://www.bioinfo.tsinghua.edu.cn/~liuke/Linc2GO/index.html](http://www.bioinfo.tsinghua.edu.cn/~liuke/Linc2GO/index.html) (not available on 20 May 2021) | [54] |
| StarBase v2.0 | A database of human mRNAs (from GENCODE 19 version) that can potentially act as ceRNAs. | [http://starbase.sysu.edu.cn/](http://starbase.sysu.edu.cn/) (accessed on 20 May 2021) | [55] |
| Lnc2GO | MicroRNA–mRNA and microRNA–lncRNA interaction data were integrated to generate lincRNA functional annotations based on the ‘competing endogenous RNA hypothesis’. | [http://www.bioinfo.tsinghua.edu.cn/~liuke/Linc2GO/index.html](http://www.bioinfo.tsinghua.edu.cn/~liuke/Linc2GO/index.html) (not available on 20 May 2021) | [54] |
| StarBase v2.0 | A database of human mRNAs (from GENCODE 19 version) that can potentially act as ceRNAs. | [http://starbase.sysu.edu.cn/](http://starbase.sysu.edu.cn/) (accessed on 20 May 2021) | [55] |
| HumanViCe | Provide the potential ceRNA networks in virus-infected human cells. | [http://gyanxet-beta.com/humanvice](http://gyanxet-beta.com/humanvice) (not available on 20 May 2021) | [57] |
| Cupid | A method for simultaneous prediction of microRNA-target interactions and their mediated competitive endogenous RNA (ceRNA) interactions. | [http://cupidtool.sourceforge.net/](http://cupidtool.sourceforge.net/) (accessed on 20 May 2021) | [58] |
| miR Sponge | Provide an experimentally supported resource for miRNA–sponge interactions and ceRNA relationships. | [http://www.bio-bigdata.net/miRSpome/](http://www.bio-bigdata.net/miRSpome/) (not available on 20 May 2021) | [59] |
| SomamiR 2.0 | A database of cancer somatic mutations in miRNA and their target sites that potentially alter the interactions between miRNAs and ceRNA including mRNAs, circRNA, and lncRNA. | [http://compbio.uthsc.edu/SomamiR](http://compbio.uthsc.edu/SomamiR) (accessed on 20 May 2021) | [60] |
| dreamBase | Provide insights into the transcriptional regulation, expression, functions, and mechanisms of pseudogenes as well as their roles in biological processes and diseases. | [http://rna.sysu.edu.cn/dreamBase](http://rna.sysu.edu.cn/dreamBase) (accessed on 20 May 2021) | [61] |
| LncCeRBase | Encompass 432 lncRNA–miRNA–mRNA interactions. | [http://www.insect-genome.com/LncCeRBase](http://www.insect-genome.com/LncCeRBase) (accessed on 20 May 2021) | [62] |
| LncACTdb 2.0 | Provide comprehensive information of competing endogenous RNAs (ceRNAs) in different species and diseases. | [http://www.bio-bigdata.net/LncACTdb/](http://www.bio-bigdata.net/LncACTdb/) (not available on 20 May 2021) | [63] |
| DIANA-LncBase v3.0 | Provide correlations of miRNA–lncRNA pairs, as well as lncRNA expression profiles in a wide range of cell types and tissues. Provide genomic variations that disturb lncRNA-associated ceRNA network regulation curated from the published literature and high-throughput data sets. | [http://www.microrna.gr/LncBase](http://www.microrna.gr/LncBase) (accessed on 20 May 2021) | [64] |
| LnCeVar | A repository of ceRNAs in blood exosomes. | [http://www.bio-bigdata.net/LnCeVar/](http://www.bio-bigdata.net/LnCeVar/) (not available on 20 May 2021) | [65] |
| ExoceRNA atlas | A repository of ceRNAs in blood exosomes. | [https://www.exocerna-atlas.com/exoceRNA#/](https://www.exocerna-atlas.com/exoceRNA#/)(accessed on 20 May 2021) | [66] |
| Cerina | Predict biological functions of circRNAs based on the ceRNA model. Document cellular-specific lncRNA-associated ceRNA networks for personalised characterisation of diseases based on the ‘One Cell, One World’ theory. | [https://www.bswhealth.med/research/Pages/biostat-software.aspx](https://www.bswhealth.med/research/Pages/biostat-software.aspx) (accessed on 20 May 2021) | [67] |
| LnCeCell | Document cellular-specific lncRNA-associated ceRNA networks for personalised characterisation of diseases based on the ‘One Cell, One World’ theory. | [http://www.bio-bigdata.hrbmu.edu.cn/LnCeCell/](http://www.bio-bigdata.hrbmu.edu.cn/LnCeCell/) (accessed on 20 May 2021) | [68] |
4. IncRNAs as ceRNAs in GC

IncRNAs are greater than 200 nucleotides in length molecules lacking obvious open reading frames, not translated into proteins, and widely transcribed in the genome of eukaryotic cells [69].

Recently, IncRNAs have become a research focus in the field of oncology. There are diverse mechanisms for IncRNAs to regulate miRNA. This article focuses on their actions as ceRNAs, where IncRNAs can play the role of endogenous “miRNA sponges” competing with mRNAs to bind the MREs of miRNAs, thereby inhibiting miRNA expression and its negative regulation of target genes, and participating in the occurrence and development of tumors, providing a new perspective for the study of tumor formation mechanisms and tumor detection methods [70–74].

4.1. HOTAIR

Using high-resolution chip analysis technology, scholars discovered a IncRNA transcribed from the HOXC locus in the study of 11 human fibroblasts and named it HOTAIR in 2007. HOTAIR was the first antisense transcription IncRNA to be discovered. It contains 2158 nucleotides, and its expression level in cancer tissues is higher than in normal tissues [8]. Studies have found that it functions as a ceRNA in the occurrence and development of GC, breast cancer [75], lung cancer [76], liver cancer [77], and other tumors [78–80], and it is also related to drug resistance [81].

In 2016, a study showed that, in GC, HOTAIR directly binds to miR-126 and inhibits its expression, thus enhancing the expression of VEGFA and PIK3R2 and activating the PI3K/AKT/MPR1 pathway. HOTAIR acts as a ceRNA to promote cisplatin resistance [82]. In 2017, scholars found that the expression of HOTAIR was negatively correlated with the expression of miR-34a. The up-regulation of miR-34a caused by the down-regulation of HOTAIR can reduce cisplatin resistance in GC. The effect of the HOTAIR/miR−34a axis on GC cells may be related to PI3K/Akt and Wnt/β-catenin signaling pathway [83]. In 2018, it was found that the expression of HOTAIR was negatively correlated with the expression of miR-217. HOTAIR inhibits the expression of miR-217 and promotes the expression of GPC5 and PTPN14 as a ceRNA. Overexpression of HOTAIR inhibited the expression of miR-217 and enhanced the resistance of GC cells to paclitaxel and adriamycin [84]. In the same year, scholars discovered that HOTAIR directly targets miR-17-5p, and PTEN is modified by HOTAIR and miR-17-5p, which affects the proliferation and apoptosis of GC cells [85]. That year a study also found that the expression of HOTAIR was negatively correlated with the expression of miR-454-3p. By inhibiting the activity of STAT3/cyclin D1, down-regulating HOTAIR to stimulate the expression of miR-454-3p could inhibit the cell growth of GC [86]. Researchers then found that HOTAIR and miR-126 negatively regulate each other, which can increase or decrease the expression of CXCR4. Highly expressed HOTAIR promotes the proliferation and metastasis of GC through the miR-126/CXCR4 axis and downstream signaling pathways [87]. In addition, miR-618 is also a direct target of HOTAIR. The silence of HOTAIR makes miR-618 spongy, thereby blocking the development of GC and inhibiting the growth of xenograft tumors in vivo [88]. In 2020, researchers discovered a negative regulatory relationship between HOTAIR and miR-1277-5p. HOTAIR regulates the growth of GC by stimulating miR-1277-5p and up-regulating COL5A1 [89]. In the same year, a study found that HOTAIR can promote the carcinogenesis of GC by regulating the levels of miRNA in cells and exosomes. Over-expressed HOTAIR induced the degradation of miR-30a or -b, thus acting as a ceRNA [90]. The latest research shows that HOTAIR and miR-148b can induce the methylation of the tumor suppressor gene PCKG10 and promote GC [91]. These data indicate that HOTAIR can promote the occurrence and development of GC in various ways and enhance the drug resistance of GC cells as a ceRNA.

4.2. XIST

XIST is located in the X chromosome’s inactive central region, affecting the activation of X-chromosome-related genes [6,7]. Studies have found that XIST is abnormally
expressed in various tumors and acts as a ceRNA to mediate tumor cell proliferation, migration, invasion, and drug resistance [92,93].

lncRNA XIST is significantly up-regulated in GC tissues and cell lines, and there is a negative correlation between its expression level and that of miR-101. Down-regulating the expression of XIST can inhibit the occurrence, development, and metastasis of GC by regulating the expression of EZH2 through miR-101 [94]. Studies have found that XIST promotes cell development from the G1 phase to the S phase and protects cells from apoptosis. XIST participates in the miR-497/MACC1 axis to regulate the proliferation and invasion of GC cells [95]. In addition, the researchers found that the expression of XIST and miR-185 are negatively correlated. miR-185 can negatively regulate the expression of TGF-β1 in vitro, and XIST can be used as a ceRNA to participate in the development of GC through the miR-185/TGF-β1 axis [96]. In 2020, studies found that XIST acts as a ceRNA in GC to regulate JAK2 by competing with miR-337. Up-regulation of miR-337 can reduce the expression of JAK2, thereby inhibiting the proliferation and migration of GC cells [97]. In addition to competing with miR-337, XIST can up-regulate the expression of PXN by competitively binding miR-132, which can enhance the ability to form GC cell proliferation, and migration. In studying the relationship between XIST and cisplatin resistance in GC, researchers found that XIST and miR-let-7b levels are negatively correlated, and the interaction between the two promotes cisplatin resistance [98].

4.3. H19

As the first imprinted gene to be discovered, IncRNA H19 is located on the H19/IGF2 gene cluster of human chromosome 11p15 [5]. With the deepening of research, it was found that IncRNA H19 plays an important role in the occurrence and development of cancer. It acts as an oncogene in some tumors to mediate the tumor process, while in others it plays a role as a tumor suppressor gene [99–101].

Studies have found that the expression of H19 is positively correlated with the expression of miR-675. The up-regulated expression of H19 and miR-675 can promote cell proliferation and inhibit cell apoptosis. The H19/miR-675 axis promotes GC’s occurrence and development through the FADD/caspase 8/caspase 3 signaling pathway [102]. In 2018, researchers found that the expression of H19 was negatively correlated with the expression of miR-let-7c. miR-let-7c belongs to the let-7 family and functions as a tumor suppressor gene. Silencing H19 resulted in a significant increase in let-7c expression, while HER2 protein expression decreased, indicating that H19 competes with miR-let-7c as a ceRNA in GC and regulates HER2 expression [103]. In the analysis of the GC ceRNA network, scholars found that the differentially regulated miR-21 and miR-148a play an important role in coordinating the sponge activity of H19, and the overexpression of H19 may be a landmark event in gastric tumorigenesis [104]. In 2019, studies showed that H19 expression is inversely proportional to miR-22-3p expression in GC tissues, and the inhibition of Snail1 can partially reverse the cell growth and metastasis induced by miR-22-3p down-regulation. H19 promotes tumor growth and metastasis through the miR-22-3p/Snail1 signaling pathway [105]. In 2020, when analyzing the IncRNA–miRNA–mRNA network of GC, scholars found that H19, miR-29a-3p, COL3A1, COL5A2, COL1A2, and COL4A1 can form a ceRNA network. H19 stimulates miR-29a-3p to promote GC [106]. The latest research shows that knocking down the expression of H19 can promote the up-regulation of miR-138, and E2F2 can be negatively regulated by miR-138, thereby inhibiting the proliferation and invasion of GC, increasing the rate of apoptosis [107].

4.4. MALAT1

In 2003, researchers discovered a differentially expressed gene in tumor cells of patients with early-stage non-small-cell lung cancer [108]. After screening and comparison, they found that it was an alpha transcript that had been described in 1997 and is known as MALAT1 [109]. Studies have shown that MALAT1 is involved in tumor proliferation, metastasis, apoptosis, epigenetic regulation, cell signal transduction, and other
Recently, MALAT1 has attracted more researchers’ attention due to its role as a ceRNA in GC [113].

In 2016, scholars found that MALAT1 is up-regulated in GC tissues. Knockdown of MALAT1 can negatively regulate miR-202 and significantly reduce the expression of Gli2, thereby inhibiting the proliferation of GC cells and inducing apoptosis [114]. The expression of MALAT1 is relatively high in the cancer tissues of patients with short survival and poor prognosis. MALAT1 can sponge miR-1297, and they are negatively correlated. The up-regulation of MALAT1 leads to miR-1297, thus reducing the ability to inhibit the expression of HMGB2 [115]. A 2017 study showed that the expression of MALAT1 is related to the chemoresistance of GC cells. As a ceRNA of miR-23b-3p, MALAT1 can weaken the inhibitory effect of miR-23b-3p on ATG12, leading to the chemical induction of GC cell autophagy and chemical resistance [116]. The ceRNA network shows that the differentially regulated miR-21 and miR-148a play an important role in coordinating the sponging activity of MALAT1 in GC [104]. In 2019, scholars found that MALAT1 inhibits miR-30b expression as a ceRNA in the study of chemical resistance to GC. MALAT1 enhanced autophagy-related chemical resistance of GC by inhibiting the miR-30b/ATG5 axis [117]. Research in the same year showed that MALAT1 acts as a sponge of miR-125a, and the dysregulation of the MALAT1/miR-125a axis causes IL-21R to play a carcinogenic role in GC [118]. MALAT1 can also competitively bind to miR-181a-5p, which prevents miR-181a-5p from binding to AKT3 mRNA, thereby up-regulating the level of AKT3 protein and ultimately promoting tumor growth in GC [119]. In 2020, when investigating the autophagy activity of GC tissues, researchers found that MALAT1 can inhibit the expression of miR-204 in GC cells and prevent miR-204 from down-regulating LC3B and transient receptor potential melastatin 3 (transient receptor potential melastatin 3), which activates autophagy and promotes cell proliferation [120]. MALAT1 is also negatively correlated with the expression of miR-22-3p. MiR-22-3p can negatively regulate ErbB3. The high expression of MALAT1 promotes proliferation and prevents apoptosis of GC cells by down-regulating miR-22-3p and up-regulating ErbB3. In the study of MALAT1 and miR-22-3p, it was also found that MALAT1 regulates ZFP91 through sponge miR-22-3p to enhance GC cells’ resistance to oxaliplatin (OXA) [121]. The latest research shows that hydrogen gas can inhibit the proliferation of GC cells and the expression of MALAT1 and EZH2, up-regulating the expression of miR-124-3p at the same time. It shows that the expression of MALAT1 and miR-124-3p is negatively correlated. Overexpression of MALAT1 can eliminate the effect of hydrogen [122].

In summary, some regulatory axes have been identified in the representative lncRNA-mediated ceRNETs that affect multiple hallmarks of GC progression, including proliferation, invasion, apoptosis, and migration (Figure 1 and Table 2). Studies have found that during the epithelial to mesenchymal transition (EMT) of gastric cancer, LncRNAs can act as ceRNAs to directly regulate the expression of E-cadherin and also to participate in the regulation of the expression of EMT-inducing transcription factors (EMT-TF) [123]. Further, many other lncRNAs also play the role of ceRNAs in GC. We have summarized studies on the role of lncRNAs as ceRNAs in GC during the past five years in Table 2.
Figure 1. Representative lncRNA-mediated ceRNets in GC.

| LncRNA       | The Mechanism of ceRNA | Biological Functions | Reference |
|--------------|------------------------|----------------------|-----------|
| BC032469     | miR-1207-5p/hTERT      | Proliferation        | [124]     |
| COL1A1-014   | miR-1273h-5p/CXCL12/CXCR4 | Proliferation      | [125]     |
| CRAL         | miR-505/CYLD/AKT       | Resistance           | [126]     |
| CTC-497E21.4 | miR-22/NET1            | Proliferation, invasion | [127] |
| DLX6-AS1     | miR-204-5p/OCT1        | Proliferation, migration, invasion | [128] |
| FLVCR1-AS1   | miR-155/c-Myc          | Proliferation, invasion | [129] |
| GAS5         | miR-23a/MT2A           | Apoptosis            | [130]     |
| H19          | miR-675/FADD/caspase 8/caspase 3 | Proliferation | [102] |
| H19          | miR-let-7c/HER2        | Proliferation        | [103]     |
| H19          | miR-22-3p/Snail1       | Proliferation, migration | [105] |
| H19          | miR-138/E2F2           | Proliferation, invasion | [107] |
| HNF1A-AS1    | miR-661/CDC34          | Proliferation        | [131]     |
| HOTAIR       | miR-126/VEGFA/PIK3R2   | Resistance           | [82]      |
| HOTAIR       | miR-34a/PI3K/Akt       | Resistance           | [83]      |
| HOTAIR       | miR-34a/Wnt/β-catenin  | Resistance           | [83]      |
| HOTAIR       | miR-217/GPC5 and PTPN14| Resistance           | [84]      |
| HOTAIR       | miR-17-5p/PTEN         | Proliferation        | [85]      |
| HOTAIR       | miR-454-3p/STAT3/cyclin D1 | Proliferation | [86] |
| HOTAIR       | miR-126/CXCR4          | Proliferation, migration | [87] |
| LncRNA     | The Mechanism of ceRNA                      | Biological Functions                | Reference |
|------------|---------------------------------------------|-------------------------------------|-----------|
| HOTAIR    | miR-618/KLF12                              | Proliferation                       | [88]      |
| HOTAIR    | miR-1277-5p/COL5A1                         | Proliferation                       | [89]      |
| HOTAIr    | miR-148b/PCDH10                            | Proliferation                       | [91]      |
| IGFL2-AS1 | miR-802/ARPP19                             | Proliferation, migration            | [133]     |
| KCNQ1OT1  | microRNA-9-LMX1A                           | Proliferation, migration, invasion  | [134]     |
| KCNQ1OT1  | miR-4319/DRAM2                             | Proliferation                       | [135]     |
| LINC00565 | miR-665/AKT3                               | Proliferation                       | [136]     |
| LINC01234 | miR-204-3p/CFBFB                           | Proliferation                       | [137]     |
| LINC01606 | miR-423-5p/Wnt/β-catenin                   | Migration, invasion                 | [138]     |
| LINC01939 | miR-17-3p/ERG2                             | Migration                           | [139]     |
| LINC02163 | miR-593-3p/FOXK1                           | Proliferation                       | [140]     |
| LINC02532 | miR-129-5p and miR-490-5p                  | Proliferation, migration, invasion  | [141]     |
| Lnc-ATB   | MiR-141-3p/TGFβ2                           | Proliferation                       | [142]     |
| lncR-D63785| miR-422a/MEF2D                             | Chemotherapy sensitivity            | [143]     |
| LOXL1-AS1 | miR-708-5p/USF1                            | Proliferation, migration            | [144]     |
| LOXL1-AS1 | miR-142-5p/PIK3CA                          | Proliferation, migration            | [145]     |
| MALAT1    | miR-202/Gli2                               | Proliferation                       | [114]     |
| MALAT1    | miR-1297/HMG12                             | Proliferation, invasion             | [115]     |
| MALAT1    | miR-23b-3/ATG12                            | Resistance                          | [116]     |
| MALAT1    | miR-30b/ATG5                               | Resistance                          | [117]     |
| MALAT1    | miR-125a/IL-21R                            | Proliferation, invasion             | [118]     |
| MALAT1    | miR-181a-5p/AKT3                           | Proliferation                       | [119]     |
| MALAT1    | miR-204/LC3B                               | Proliferation                       | [120]     |
| MALAT1    | miR-204/transient receptor potential       | Proliferation                       | [120]     |
| MALAT1    | miR-22-3p/ErbB3                            | Proliferation                       | [121]     |
| MALAT1    | miR-22-3p/ZFP9                               | Resistance                          | [121]     |
| MALAT1    | miR-124-3p/EZH2                            | Proliferation                       | [122]     |
| MYO5LID   | miR-29c-3p/MCL-1                           | Proliferation, inhibits apoptosis   | [146]     |
| NORAD     | miR-608/FOXO6                              | Proliferation                       | [147]     |
| NORAD     | miR-214/Akt/mTOR                           | Proliferation, inhibits apoptosis   | [148]     |
| NORAD     | miR-433-3p/ATG5,ATG12                      | Resistance                          | [149]     |
| PWRN1     | miR-425-3p/PTEN                            | Proliferation                       | [150]     |
| SLC25A5-AS1| miR-19a-3p/PTEN/PI3K/AKT                    | Proliferation                       | [151]     |
| SNHG5     | miR-32/KLF4                                | Migration                           | [152]     |
| SPRY4-IT1 | miR-101-3p/AMPK                            | Proliferation, migration            | [153]     |
| TINCR     | miR-375/PDK1                               | Proliferation                       | [154]     |
| TP73-AS1  | miR-194-5p/SDAD1                           | Proliferation, migration            | [155]     |
| TUBA4B    | miR-214 and miR-216a/b/PTEN                | Proliferation, invasion             | [156]     |
| UCA1      | miR-590-3p/CREB1                           | Proliferation, invasion             | [157]     |
| UCA1      | miR-7-5p/EGFR                              | Migratation                         | [158]     |
| UCA1      | miR-495-3p/SATB1                           | proliferation and invasion          | [159]     |
| UCA1      | miR-203/ZEB2                               | Metastasis                          | [160]     |
| UCA1      | miR-26a/b, miR-193a, miR-214/PDL1           | Proliferation, migration, immune    | [161]     |
| UCA1      | miR-495/PRL-3                              | Proliferation, migration            | [162]     |
| UCA1      | miR-513-3p/CYP1B1                          | Resistance                          | [163]     |
| XIST      | miR-101/EZH2                               | Proliferation, migration            | [94]      |
| XIST      | miR-497/MACC1                              | Proliferation, invasion             | [95]      |
| XIST      | miR-185/TGF-β1                             | Growth, migration and invasion      | [96]      |
| XIST      | miR-337/JAK2                               | Proliferation, migration            | [97]      |
| XIST      | miR-132/PXN                                | Proliferation, migration            | [164]     |
| XIST      | XIST/miR-let-7b                            | Resistance                          | [98]      |
5. circRNAs as ceRNAs in GC

circRNAs are closed loops in the cytoplasm, with neither a 5′ cap structure nor a 3′ polyadenylic acid tail structure. They were found in viroids for the first time [3]. With the development of RNA sequencing technology and in-depth research, it was found that circRNAs are widely transcribed in eukaryotes [165–168]. Compared with other linear ncRNAs, they have a high degree of conservation and stability. According to its components, they can be divided into three categories: exon circular RNAs (ecircRNAs) [169], intron circular RNAs (ciRNAs) [170], and exon–intron circular RNAs (EIciRNAs) [171], each of which has different molecular structures but have similar binding sites and regulatory functions, and provides a template for biosynthesis.

In recent years, there have been more studies on the function of circular RNAs as ceRNAs in GC. In 2017, researchers found that the expression of circNRIP1 can up-regulate the AKT1 levels in GC cells and promote cell proliferation, migration, and invasion. Up-regulation of miR-149-5p can prevent the malignant behavior caused by circNRIP1. The circNRIP1/miR-149-5p/AKT1/mTOR axis is responsible for changes in GC cells’ metabolism and promotes the development of GC [172]. In 2019, researchers discovered a new type of circRNA, has_circ_0001368. The low expression of has_circ_0001368 can promote tumor growth, and it plays a tumor suppressor effect in GC through the miR-6506-5p/FOXO3 axis [173]. In the same year, it was found that the expression of circCOL6A3 and miR-3064-5p are inversely proportional. Overexpression of circCOL6A3 promotes GC cell proliferation, migration, and apoptosis by eliminating the inhibitory effect on COL6A3 induced by miR-3064-5p [174]. Studies have found that circRNA0047905 can bind miR4516 and miR1227-5p, thereby reducing the inhibition of SERPINB5 and MMP11, activating the Akt/CREB signaling pathway, and promoting the progression of GC. Circular RNA 0047905 may act as a tumor promoter in the pathogenesis of GC [175]. TGFBR1 is the receptor of the TGF-β ligand. Studies have found that circCACTIN promotes the progression of GC by sponging miRNA-331-3p and regulating the expression of TGFBR1 mRNA [176]. In studies to confirm the function of circGRAMD1B, it was found that circGRAMD1B inhibited the proliferation, migration, and invasion of GC cells by regulating miR-130a-3p-PTEN/p21 [177]. Through bioinformatics methods, it was found that miRNA-145-5p is the target gene of circ-ZNF609. Down-regulating the expression of miRNA-145-5p can partially reverse the effect of circ-ZNF609 on the growth and migration of GC cells [178]. In 2020, researchers found that the expression of circRHOBTB3 is low in GC tissues and cell lines. circRHOBTB3 acts as a ceRNA for miR-654-3p and activates the p21 signaling pathway to inhibit GC’s growth. circRHOBTB3 is promising as a new diagnostic marker, and therapeutic target for GC [179]. circ_0006282 is a newly identified human circular RNA. Studies have found that its high expression can down-regulate miR-155, thereby activating the expression of FBXO22 and promoting the proliferation and migration of GC cells [180]. Similar to the expression of circRHOBTB3, circCCDC9 was significantly down-regulated in GC tissues and cell lines. circCCDC9 can inhibit tumor progression through the miR-6792-3p/CAV1 axis [181]. circ-MAT2B is mainly located in the cytoplasm and can act as a ceRNA to compete with miR-515-5p and increase the expression of HIF-1α [182]. circCYFIP2 is significantly up-regulated in GC tissues. Research suggests that circCYFIP2 may act as a carcinogenic circRNA to promote GC progression through the miR-1205/E2F1 axis [183]. circ_0081143 modulates the abundance of miR-497-5p by making the miR-497-5p sponge. miR-497-5p directly targets EGFR and down-regulates circ_0081143 to affect hypoxia-induced migration, invasion, and EMT of GC cells [184]. circHIPK3 is derived from the homology domain-interacting protein kinase 3 (HIPK3) gene. In GC tissues and cell lines, circHIPK3 is up-regulated. It regulates the miR-876-5p/PIK3R1 axis through the mechanism of ceRNA and mediates the proliferation, migration, and invasion of GC cells [185]. circRNA_100782 is lowly expressed in GC. Studies have found that it can be used as a molecular sponge. It can bind to miR-574-3p to regulate the expression of the tumor suppressor gene Rb. This mechanism is closely related to the proliferation and invasion of GC [186]. In the study of hsa_circ_0005556, it was found that down-regulating
the expression of hsa_circ_0005556 can inhibit the growth of GC. The hsa_circ_0005556/miR-4270/MMP19 axis participates in the proliferation, migration, and invasion of GC cells through the ceRNA mechanism [187]. When circPDZD8 is highly expressed, the survival rate of GC patients is poor. circPDZD8 can up-regulate the expression of miR-197-5p to promote the proliferation and metastasis of GC [188]. The latest research shows that the expression level of circ-ITCH and miR-199-5p are negatively correlated in GC tissues. circ-ITCH can inhibit GC metastasis by acting as a sponge of miR-199a-5p and increasing Klotho expression [189]. So far, there are 18 miRNAs that have been identified as ceRNAs in the circRNA-mediated ceRNETs that affect multiple hallmarks of gastric progression, including proliferation, migration, invasion, and apoptosis (Figure 2 and Table 3).

Figure 2. CircRNA-mediated ceRNETs in GC.

| CircRNA       | The Mechanism of ceRNA                  | Biological Functions                        | Reference |
|---------------|----------------------------------------|---------------------------------------------|-----------|
| circNRIP1     | miR-149-5p/AKT1/mTOR                  | Proliferation, migration, invasion           | [172]     |
| circRNA has_circ_0001368 | miR-6506-5p/FOXO3                     | Proliferation                               | [173]     |
| circCOL6A3    | miR-3064-5p/COL6A3                    | Proliferation, migration, apoptosis          | [174]     |
| circRNA0047905 | miR-4516/miR-1227-5p/SERPINEB5/MMP11 | Proliferation                               | [175]     |
| circCACTIN    | miRNA-331-3p/TGFBR1                   | Proliferation                               | [176]     |
| circGRAMD1B   | miR-130a-3p/PTEN/p21                  | Proliferation, migration, invasion          | [177]     |
| circZNF609    | miRNA-145-5p                          | Proliferation                               | [178]     |
| circRHOBTB3   | miR-654-3p/p21                        | Proliferation                               | [179]     |
| circ_0006282  | miR-155/FBXO22                        | Proliferation, migration                     | [180]     |
| circCCDC9     | miR-6792-3p/CAV1                      | Proliferation                               | [181]     |
| circ-MAT2B    | miR-515-5p/HIF-1α                    | Proliferation                               | [182]     |
| circCYFIP2    | miR-1205/E2F1                         | Proliferation, invasion                      | [183]     |
| circ_0081143  | miR-497-5p/EGFR                       | migration, invasion, EMT                    | [184]     |
| CircHIPK3     | miR-876-5p/PIK3R1                     | Proliferation, migration, invasion           | [185]     |
| circRNA_10078 | miR-574-3p/Rb                         | Proliferation, invasion                      | [186]     |
| hsa_circ_0005556 | miR-4270/MMP19                      | Proliferation, migration                     | [187]     |
| circPDZD8     | miR-197-5p/CHD9                       | Proliferation, migration                     | [188]     |
| circ-ITCH     | miR-199a-5p/Klotho                    | Migration                                    | [189]     |
6. Pseudogenes as ceRNAs in GC

Pseudogenes were once considered to be genomic fossils without bodily functions resulting from the accumulation of natural mutations of genes during biological evolution. Later, it was discovered that pseudogenes play a crucial role in gene transcription [190]. They can be used as ceRNAs to regulate gene transcription. In addition, pseudogenes can also regulate gene expression by interacting with RNA-binding proteins [191–193].

There are few studies on pseudogenes as ceRNAs in GC. In 2015, researchers reported for the first time that the pseudogene \textit{FER1L4} acts as a ceRNA in the proliferation of GC. Down-regulation of \textit{FER1L4} increased the abundance of miR-106a-5p, decreased PTEN mRNA and protein quantity, and promoted GC proliferation [194]. In 2017, a study found that the pseudogene \textit{PTENP1} of PTEN can be used as a ceRNA to regulate the expression of PTEN together with miR-106b/miR-93 [195]. The up-regulated expression of \textit{PTENP1} can inhibit the proliferation, metastasis, and invasion of GC cells. In the latest study, it was found that \textit{GBAP1} can competitively bind to miR-212-3p, promote GBA expression, and participate in GC development [196].

7. Conclusions

In summary, GC is a common gastrointestinal cancer with an insidious onset, and patients are often in the middle or late stage when they are diagnosed. It is important to understand the molecular mechanism of GC and to explore effective detection and treatment strategies.

The role of ncRNAs in tumors has been a hot spot in oncology research recently. The miRNA mechanism in tumors is now relatively clear, and IncRNAs, circRNAs, and pseudogenes have entered people’s fields of vision. Evidence shows that ceRNAs play an important regulatory role in GC. So far, researchers have established some RNA–miRNA–mRNA regulatory axes [197–200]. With the effective use of advanced bioinformatics tools, researchers can systematically construct more regulatory networks, and the identification of GC-related ceRNA networks should become more efficient and accurate. Some IncRNAs, circRNAs, and pseudogenes are found to act as ceRNAs. Studies showed that IncRNAs, circRNAs, and pseudogenes could promote the occurrence and development of tumors, inhibit tumor progression and metastasis, and regulate the sensitivity of tumor cells to chemotherapeutic drugs. However, the database of IncRNAs, circRNAs, and pseudogenes is not yet perfect.

Because studies usually use transfected oligonucleotides or expression vectors, there is a risk that the transfected oligonucleotide inhibitors (antagomir and miRNA sponge) may be collected by lysosomes and cannot cause miRNA activity. It is difficult to directly measure the potential activity of the introduced miRNAs. The current verification experiments are usually untested at the physiological level, artificially providing high quantification after the whole cell is lysed. Thus, the technologies to verify the effect of ceRNAs on target genes at the protein and RNA levels require a rigorous evaluation and should be complemented by studies in animal models to discover additional genes involved in cancer.

Moreover, the map of the complex IncRNA, circRNA, and pseudogene regulatory networks needs to be further improved and supplemented. However, researchers have mainly focused on a single axis or a single binding partner, and there is no uniform naming principle for IncRNAs, circRNAs, and pseudogenes. The secondary and indirect interactions may also affect the occurrence and development of GC and drug resistance. Therefore, further research should also pay attention to the complex IncRNA, circRNA, pseudogene, miRNA, and mRNA networks. Analyzing the IncRNA-specific molecular mechanisms underlying their biological function and transforming basic research into clinical application is still an enormous challenge.

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Abbreviations

AKT  AKT serine/threonine kinase 1
AKT3  AKT serine/threonine kinase 3
AMPK  Protein kinase AMP-activated catalytic subunit alpha 1
ARPP19 CAMP-regulated phosphoprotein 19
ATG5  Autophagy-related 5
ATG12  Autophagy-related 12
CAV1  Caveolin 1
CBFB  Core-binding factor subunit beta
CDC34  Cell division cycle 34, ubiquitin-conjugating enzyme
ceRNAs  Competitive endogenous RNAs
ceRNETs  ceRNA networks
CHD9  Chromodomain helicase DNA-binding protein 9
ciRNAs  Intron circular RNAs
COL1A2  Collagen type I alpha 2 chain
COL3A1  Collagen type III alpha 1 chain
COL4A1  Collagen type IV alpha 1 chain
COL5A1  Collagen type V alpha 1 chain
COL5A2  Collagen type V alpha 2 chain
CREB1  CAMP-responsive element-binding protein 1
CXCL12  C-X-C motif chemokine ligand 12
CXCR4  C-X-C motif chemokine receptor 4
CYLD  CYLD lysine 63 deubiquitinase
CYP1B1  Cytochrome P450 family 1 subfamily B member 1
DRAM2  DNA-damage-regulated autophagy modulator 2
E2F1  E2F transcription factor 1
E2F2  E2F transcription factor 2
eircRNAs  Exon circular RNAs
EGFR  epidermal growth factor receptor
EGR2  Early growth response 2
ElciRNAs  Exon–intron circular RNAs
EMT  Epithelial to mesenchymal transition
EMT-TF  EMT-inducing transcription factor
ErbB3  Erb-B2-receptor tyrosine kinase 3
EZH2  Enhancer of zeste 2 polycomb-repressive complex 2 subunit
FADD  Fas-associated via death domain
FBXO22  F-box protein 22
FOXK1  Forkhead box K1
FOXO3  Forkhead box O3
FOXO6  Forkhead box O6
GBA  Glucosylceramidase beta
GC  Gastric cancer
GEO  Gene Expression Omnibus microarray datasets
| Gene Name   | Description                                             |
|-------------|---------------------------------------------------------|
| Gli2        | GLI family zinc finger 2                               |
| GPC5        | Glypican-5                                              |
| H19         | H19-imprinted maternally-expressed transcript           |
| HER2        | Erb-B2-receptor tyrosine kinase 2                       |
| HIF-1α      | Hypoxia-inducible factor 1 subunit alpha                |
| HIPK3       | Homeodomain-interacting protein kinase 3               |
| HMGB2       | High-mobility group box 2                               |
| HOTAIR      | HOX transcript antisense RNA                            |
| hTERT       | Human telomerase reverse transcriptase                  |
| IGF2        | Insulin-like growth factor 2                            |
| IL-21R      | Interleukin 21 Receptor                                 |
| JAK2        | Janus kinase 2                                          |
| KLF4        | Kruppel-like Factor 4                                   |
| LC3B        | Microtubule-associated protein 1 light chain 3 beta    |
| LMX1A       | LIM homeobox transcription factor 1 alpha               |
| IncRNAs     | Long non-coding RNAs                                    |
| MACC1       | MET transcriptional regulator MACC1                     |
| MALAT1      | Metastasis-associated lung adenocarcinoma transcript 1 |
| MCL-1       | MCL1 apoptosis regulator, BCL2 family member            |
| MEF2D       | Myocyte enhancer factor 2D                             |
| miRNA–RISC  | miRNA-mediated silencing complex                        |
| miRNAs      | MicroRNAs                                               |
| MMP11       | Matrix metalloprotease 11                               |
| MMP19       | Matrix metalloprotease 19                               |
| MRE         | miRNA response element                                  |
| mRNAs       | Messenger RNAs                                          |
| MT2A        | Metallothionein 2A                                      |
| mTOR        | Mechanistic target of rapamycin kinase                  |
| NET1        | Neuroepithelial cell transforming 1                     |
| OCT1        | POU class 2 homeobox 1                                  |
| PIK3CA      | Phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit alpha |
| PIK3R1      | Phosphoinositide-3-kinase regulatory subunit 1          |
| PIK3R2      | Phosphoinositide-3-kinase regulatory subunit 2          |
| PCDH        | Protocadherin 10                                        |
| PDK1        | Pyruvate dehydrogenase kinase 1                         |
| PDL1        | CD274 molecule                                          |
| PRC2        | Polycomb repressive complex 2                           |
| PRL-3       | Protein tyrosine phosphatase 4A3                        |
| PTEN        | Phosphatase and tensin homolog                          |
| PTPN14      | Protein tyrosine phosphatase non-receptor type 14       |
| PXN         | Paxillin                                                |
| SATB1       | SATB homeobox 1                                          |
| SDAD1       | SDA1 domain-containing 1                                |
| SERPINC5    | Serpin family B member 5                                |
| SHOX2       | Short stature homeobox 2                                |
| Snail1      | Snail family transcriptional repressor 1                |
| STAT3       | Signal transducer and activator of transcription 3     |
| TCGA        | Cancer Genome Atlas database                            |
| TGF-β1      | Transforming growth factor beta 1                       |
| TGF-β2      | Transforming growth factor beta 2                       |
| TGFBR1      | Transforming growth factor beta receptor 1              |
| USF1        | Upstream transcription factor 1                         |
| VEGFA       | Vascular endothelial growth factor A                    |
| XIST        | X inactive specific transcript                          |
| ZEB2        | Zinc finger E-box-binding homeobox 2                    |
| ZFP91       | ZFP91 zinc finger protein, atypical E3 ubiquitin ligase |
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