

Table S1. miRNAs that target ATG genes

Target gene	microRNA	ceRNA ¹	Type of Cell line (Tissue)	Function	Ref.
<i>ULK1</i>	<i>MIR106A</i>	-	HCT116 and SW480 cells (colorectal cancer)	Combination of metformin, doxorubicin, and sodium oxamate treatment suppress <i>MIR106A</i> expression and induce autophagy and subsequent apoptosis in colorectal cancer cells.	(Salgado -García et al., 2021)
		-	Macrophages	<i>MIR106A</i> promotes <i>M. tuberculosis</i> survival through inhibiting autophagy, and <i>MIR106A</i> expression is downregulated in tuberculosis patients.	(Liu et al., 2020b)
	<i>MIR1262</i>	-	MGC80-3 and HGC-27 (GCA)	<i>MIR1262</i> inhibits gastric cardia adenocarcinoma cell proliferation, migration and invasion through downregulating the <i>ULK1</i> level.	(Zheng et al., 2021b)
	<i>MIR128</i>	-	Primary spinal cord neurons	Overexpression of <i>MIR128</i> downregulates <i>ULK1</i> and thereby inhibits autophagy, which will inhibit neuronal cell apoptosis and inflammation in rats with spinal cord injury.	(Liu et al., 2021)
	<i>MIR132-5p</i>	-	SH-SY5Y (neuroblastoma)	<i>MIR132-5p</i> is upregulated in MPTP-treated cells and inhibition of <i>MIR132-5p</i> reduces the <i>ULK1</i> level, autophagy, and MPTP-induced apoptosis.	(Zhao et al., 2020a)
	<i>MIR142-5p</i>	<i>TMEM87A</i> (circRNA)	BGC823 cells (gastric cancer)	<i>TMEM87A</i> is upregulated in gastric cancer cells and promotes cell proliferation through sponging <i>MIR142-5p</i> and upregulating <i>ULK1</i> .	(Wang et al., 2021)
	<i>MiR192-5p</i>	<i>CIRC_0005774</i> (circRNA)	HL-60 and NB4 cells (AML)	Knocking down <i>CIRC_0005774</i> inhibits AML cell proliferation and induces apoptosis through the <i>MIR192-5p</i> - <i>ULK1</i> axis.	(Li et al., 2021c)
	<i>Mir214</i>	-	Hepatocytes and in vivo	<i>Mir214</i> suppresses <i>Ulk1</i> expression and inhibition of <i>Mir214</i> expression ameliorates fatty liver disease in high-fat diet mice through upregulation of <i>Ulk1</i> .	(Lee et al., 2021)

	-	Rat renal proximal tubule cells	<i>Mir214</i> is upregulated in diabetic kidneys and high-glucose treated tubular cells, which leads to reduced autophagy, tubular hypertrophy and decline of renal function.	(Ma et al., 2020b)
<i>MIR26A-5p</i>	<i>SNHG6</i>	RKO, HT29 and HCT116 cells (colorectal cancer)	<i>SNHG6</i> enhances 5-fluorouracil resistance and suppresses apoptosis of colorectal cancer cells by upregulating autophagy through sponging <i>MIR26A-5p</i> , which binds to <i>ULK1</i> mRNA and suppresses its expression.	(Wang et al., 2019a)
	-	Primary cardiac fibroblasts	<i>MIR26A-5p</i> suppresses autophagy in cardiac fibroblasts through binding <i>ULK1</i> mRNA and inhibiting its expression.	(Zheng et al., 2018)
<i>Mir30B-3p</i>	<i>Gm15834</i>	HL-1 cells (cardio myocytes)	<i>Gm15834</i> promotes myocardial hypertrophy by sponging <i>Ulk1</i> -targeting <i>Mir30B-3p</i> and upregulating autophagy.	(Song et al., 2021)
<i>MIR34A-5p</i>	<i>CIRC_0009910</i> (circRNA)	K562 cells (CML)	<i>CIRC_0009910</i> mediates imatinib resistance via upregulating autophagy by binding <i>MIR34A-5p</i> which targets <i>ULK1</i> .	(Cao et al., 2020a)
<i>MIR373</i>	-	HCCC9810 and RBE cells (cholangiocarcinoma)	<i>MIR373</i> upregulation inhibits autophagy by targeting <i>ULK1</i> and promotes cell apoptosis of cholangiocarcinoma cells.	(Lv et al., 2020)
<i>MIR4463</i>	-	HUVEC	<i>MIR4463</i> promotes cell apoptosis under hypoxia conditions and suppressing <i>MIR4463</i> attenuates apoptosis through promoting autophagy.	(He et al., 2022)
<i>MIR514A-3p</i>	<i>LUCAT1</i>	A549 (NSCLC)	<i>LUCAT1</i> has a high level in cisplatin-resistant NSCLC cells and mediates promoted autophagy, migration, and invasion through sponging <i>MIR514A-3p</i> and thereby upregulating <i>ULK1</i> expression.	(Shen et al., 2020b)

	<i>Mir558</i>	<i>MALAT1</i>	H9C2 (rat myocardial cell)	<i>Malat1</i> protects cardiomyocytes against isoproterenol-induced apoptosis, through sponging <i>Ulk1</i> -targeting <i>Mir558</i> and inducing autophagy.	(Guo et al., 2019b)
	<i>MIR665</i>	<i>MIAT</i>	THP-1 cells (monocytic cells)	<i>MIAT</i> promotes autophagy in human macrophages after BCG infection through sponging <i>MIR665</i> and upregulating <i>ULK1</i> expression, which suppresses BCG survival.	(Jiang et al., 2021)
	<i>MIR93</i>	-	MEF and HE LA cells	<i>MIR93</i> suppresses hypoxia-induced autophagy through binding <i>ULK1</i> mRNA and functions differently regarding cell viability under hypoxia in cancer and non-cancer cells.	(Li et al., 2017)
<i>ATG2B</i>	<i>MIR130A</i>	-	Primary vascular smooth muscle cells	<i>MIR130A</i> inhibits cell proliferation by suppressing autophagy via targeting <i>ATG2B</i> .	(Zheng et al., 2021a)
		<i>HOTAIR</i>	GIST-882 and GIST-T1 (GIST)	<i>HOTAIR</i> regulates autophagy through sponging <i>MIR130A</i> , which binds to <i>ATG2B</i> mRNA and mediates imatinib resistance of GIST cells.	(Zhang et al., 2021a)
	<i>MIR143</i>	-	HL60 cells (AML)	<i>MIR143</i> promoted cytarabine toxicity in AML cells through inhibiting the expression of <i>ATG2B</i> and <i>ATG7</i> and thus suppressing autophagy.	(Zhang et al., 2020)
		<i>DUXAP9/LINC01296</i>	NSCLC cell lines	<i>DUXAP9/LINC01296</i> regulates <i>ATG2B</i> expression through <i>MIR143</i> and knockdown of <i>DUXAP9</i> leads to slower tumor growth and metastasis <i>in vivo</i> .	(Li et al., 2021f)
	<i>MIR17-5p</i>	<i>HOTAIR</i>	A549 and BEAS-2B cells (lung tissue)	<i>HOTAIR</i> inhibition suppresses autophagy and cell apoptosis through releasing <i>MIR17-5p</i> , which targets multiple <i>ATG</i> genes and alleviates lipopolysaccharide-induced lung injury <i>in vivo</i> .	(Li et al., 2021d)
<i>ATG3</i>	<i>MIR1</i>	-	A549 and H1299 (NSCLC)	<i>MIR1</i> overexpression enhances cisplatin sensitivity of NSCLC cells through suppressing <i>ATG3</i> expression and autophagy.	(Hua et al., 2018)

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<i>MIR204</i>	<i>NEAT1</i>	HepG2 and Huh7 cells (HCC)	<i>NEAT1</i> promotes autophagy through sponging <i>MIR204</i> and upregulating <i>ATG3</i> expression and HCC cell resistance to sorafenib.	(Li et al., 2020b)
	<i>KCNQ1OT1</i>	A549 and H460 cells (NSCLC)	<i>KCNQ1OT1</i> depletion induces NSCLC cell apoptosis through releasing <i>MIR204</i> , which inhibits <i>ATG3</i> expression and autophagy.	(Kang et al., 2019)
<i>MIR365</i>	<i>PVT1</i>	HepG2 and Huh7 cells (HCC)	Downregulating <i>PVT1</i> suppresses autophagy and HCC cell proliferation through releasing <i>MIR365</i> and thus reducing <i>ATG3</i> expression.	(Yang et al., 2019)
<i>MIR378B</i>	<i>CDKN2B-AS1/ANRIL</i>	PC12 cells and primary neurons	<i>CDKN2B-AS1/ANRIL</i> protects neurons from hypoxia-ischemia injury through sponging <i>MIR378B</i> and upregulating <i>ATG3</i> expression.	(Li et al., 2021a)
<i>MIR431</i>	-	Cardiom yocytes	Overexpression of <i>MIR431</i> protects cardiomyocytes from hypoxia/reoxygenation-induced apoptosis through inhibiting <i>ATG3</i> expression and autophagy.	(Zhou et al., 2021a)
	-	SW620 and HCT116 cells (colorectal cancer)	Overexpressing <i>MIR431-5p</i> suppresses colon cancer progression through inhibiting <i>ATG3</i> expression and autophagy.	(Huang et al., 2019b)
<i>MIR495</i>	-	CAL-62 and SW579 cells (thyroid carcinoma)	<i>MIR495</i> is modulated by pseudogene <i>LGMDN</i> . <i>LGMDN</i> promotes the progression of thyroid carcinoma through inhibiting <i>MIR495</i> and thus inducing <i>ATG3</i> expression and autophagy.	(Sun et al., 2021)

	<i>MIR622</i>	<i>SP100-AS1</i>	SW480 and HCT116 cells (colorectal cancer)	<i>SP100-AS1</i> mediates radioresistance of colorectal cancer cells through binding to <i>MIR622</i> and upregulating <i>ATG3</i> expression and autophagy.	(Zhou et al., 2022)
<i>ATG4B</i>	<i>MIR34A</i>	-	HeLa and SKOV3 cells	Rapamycin treatment reduces the expression of <i>MIR34A</i> and <i>MIR34C-5p</i> and the overexpression of either miRNA inhibits autophagy activity.	(Wu et al., 2017)
	<i>MIR34C-5p</i>	-			
	<i>MIR34A</i>	-	<i>In vivo</i>	<i>MIR34A</i> suppresses autophagy through binding <i>ATG4B</i> mRNA and <i>MIR34A</i> inhibition ameliorates lung injury in septic mice.	(Chen et al., 2020b)
		<i>NEAT1</i>	HCT8 and SW480 (colorectal cancer)	<i>NEAT1</i> knockdown downregulates cell proliferation and induces its sensitivity to 5-FU through suppressing autophagy via <i>MIR34A</i> , which binds and regulates <i>ATG4B</i> and <i>ATG9A</i> expression.	
	<i>MIR93</i>	-	Patient-derived glioma stem-like cells	Ectopic <i>MIR93</i> expression inhibits treatment-induced autophagy through targeting multiple <i>ATG</i> genes and sensitizes the GSCs to the treatment.	(Huang et al., 2019a)
<i>ATG4C</i>	<i>MIR142-3p</i>	-	RAW26 4.7(macrophages)	Overexpression of <i>MIR142-3p</i> inhibits autophagy in macrophages through downregulating autophagy through binding <i>ATG4C</i> and <i>ATG16L1</i> and promotes the survival of <i>M. tuberculosis</i> .	(Qu et al., 2021)
<i>ATG5</i>	<i>MIR130A</i>	-	Huh7.5.1 cells (hepatic cells)	<i>MIR130A</i> inhibits hepatitis C virus replication through downregulating <i>ATG5</i> , which is important for the expression of interferon-stimulated genes.	(Duan et al., 2019)
	<i>MIR137</i>	-	PANC-1 (pancreatic cancer)	<i>MIR137</i> sensitizes pancreatic cancer cells to doxorubicin through inhibiting <i>ATG5</i> and autophagy <i>in vitro</i> and <i>in vivo</i> .	(Wang et al., 2019b)

<i>MIR140-3p</i>	<i>CCAT1</i>	AGS and MKN-45 cells (gastric cancer)	<i>CCAT1</i> promotes gastric cancer cell proliferation, migration, and invasiveness through sponging <i>MIR140-3p</i> and upregulating <i>ATG5</i> expression and autophagy.	(Yang et al., 2022)
	<i>PVT1</i>	A549 and SK-MES-1 cells (lung cancer)	<i>PVT1</i> mediates hypoxia-induced chemoresistance through sponging <i>MIR140-3p</i> and upregulating <i>ATG5</i> and autophagy.	(Wang et al., 2022a)
<i>MIR142-3p</i>	-	HepG2 and SMMC-7721 cells (HCC)	Ectopic expression of <i>MIR142-3p</i> sensitizes HCC cells to sorafenib treatment via targeting <i>ATG5</i> and <i>ATG16L1</i> and inhibiting autophagy.	(Zhang et al., 2018b)
<i>MIR149-5p</i>	<i>FOXM1</i> (cirRNA)	H1581 and A549 cells (NSCLC)	<i>FOXM1</i> knockdown or <i>MIR149-5p</i> overexpression suppresses NSCLC cell growth through inhibiting <i>ATG5</i> expression.	(Wei et al., 2021)
<i>MIR153</i>	<i>OIP5-AS1</i>	HOS cells (osteosarcoma)	<i>OIP5-AS1</i> promotes osteosarcoma cell migration and invasion through downregulating the <i>ATG5</i> -targeting <i>MIR153</i> level.	(Li et al., 2021e)
<i>MIR153-3p</i>	-	PC-9/GR and HCC827/GR cells (NSCLC)	Overexpression of <i>MIR153-3p</i> can enhance gefitinib-sensitivity of NSCLC cells through suppressing <i>ATG5</i> expression and autophagy.	(Zhang et al., 2019c)
<i>MIR181A</i>	-	HepG2 (HCC)	<i>MIR181A</i> inhibition promotes autophagy and apoptosis in HCC cells and inhibits tumor growth.	(Yang et al., 2018)
<i>MIR181C-5p</i>	<i>GAS5</i>	RAW264.7 (mouse macrophage)	<i>GAS5</i> preserves the expression of <i>Atg5</i> through sponging <i>MIR181C-5p</i> and promotes autophagy in macrophages.	(Xu et al., 2021b)

<i>MIR187-3p</i>	<i>GAS8-AS1</i>	TPC1 and BCPAP cells (PTC)	<i>GAS8-AS1</i> promotes autophagy and suppresses thyroid cancer cell proliferation partially through sponging <i>MIR187-3p</i> and upregulating <i>ATG5</i> expression.	(Qin et al., 2020)
<i>MIR20A</i>	-	LoVo and SW48 cells (colorectal cancer)	<i>MIR20A</i> is downregulated under hypoxia conditions, which inhibits hypoxia-induced autophagy via downregulating <i>ATG5</i> and <i>RB1CC1</i> expression.	(Che et al., 2019)
<i>MIR214</i>	<i>BACE1-AS</i>	SH-SY5Y cells (neuroblastoma)	Knocking down <i>BACE1-AS</i> downregulates autophagy through releasing <i>MIR214</i> , which suppresses <i>ATG5</i> expression and protects neurons from A β_{1-42} -induced injury.	(Zhou et al., 2021b)
<i>MIR216B-5p</i>	<i>IDH1-AS1</i>	VCaP, and LNCaP (prostate cancer)	<i>IDH1-AS1</i> expression is upregulated in prostate cancer cells and promotes tumor growth by upregulating <i>ATG5</i> -mediate autophagy via sponging <i>MIR216B-5p</i> .	(Zhang et al., 2019b)
<i>MIR2355-5p</i>	<i>ARRDC1-AS1</i>	RCK-8 and OCI-LY-3 cells (DLBC L)	<i>ARRDC1-AS1</i> promotes cell proliferation and migration through upregulating autophagy via sponging <i>ATG5</i> targeting <i>MIR2355-5p</i> .	(Xu et al., 2021a)
<i>MIR30A</i>	-	BEAS-2B (lung epithelia 1 cell) and in vivo	<i>MIR30A</i> suppresses airway inflammation and fibrosis in asthma through suppressing autophagy by targeting <i>ATG5</i> .	(Li et al., 2020a)
<i>MIR30A-5p</i>	-	SW900 and NH91 cells (LUSC)	<i>MIR30A-5p</i> attenuates LUSC progression through downregulating <i>ATG5</i> expression and autophagy.	(Yang et al., 2021)
<i>MIR30B</i>	<i>DICER1-AS1</i>	MG-63 and U2OS cells	Knocking down <i>DICER1-AS1</i> reduces osteosarcoma cell proliferation, migration, and invasiveness, upregulates <i>MIR30B</i> expression and downregulates	(Gu et al., 2018)

		(osteosarcoma)	<i>ATG5</i> expression and autophagy.		
<i>MIR372-3p</i>	<i>KCNQ1OT1</i>	A549 and H1975 cells (LUAD)	Knocking down <i>KCNQ1OT1</i> improves LUAD cell radiosensitivity through releasing <i>MIR372-3p</i> , which inhibits autophagy through binding <i>ATG5</i> and <i>ATG12</i> .	(He et al., 2020)	
<i>MIR93</i>	-	Patient-derived glioma stem-like cells	Ectopic <i>MIR93</i> expression inhibits treatment-induced autophagy through targeting multiple <i>ATG</i> genes and sensitizes the GSCs to the treatment.	(Huang et al., 2019a)	
<i>BECN1</i>	<i>MIR326</i>	<i>CIRC_0020850</i> (circRNA)	A549 and HCC827 cells (LUAD)	Knocking down <i>CIRC_0020850</i> and overexpression of <i>MIR326</i> inhibit proliferation, migration, and invasion of LUAD cells through inhibiting <i>BECN1</i> expression.	(Li et al., 2022b)
	<i>MIR93</i>	-	Patient-derived glioma stem-like cells	Ectopic <i>MIR93</i> expression inhibits treatment-induced autophagy through targeting multiple <i>ATG</i> genes and sensitizes the GSCs to the treatment.	(Huang et al., 2019a)
	<i>MIR142-5p</i>	-	SH-SY5Y cells (neuroblastoma)	<i>MIR142-5p</i> protects 6-hydroxydopamine-induced cell damage through downregulating <i>BECN1</i> expression and thus inhibiting autophagy.	(Chen et al., 2020a)
	<i>MIR506-3p</i>	-	HUVEC	<i>MIR506-3p</i> inhibits HUVEC proliferation and migration and promotes apoptosis through inhibiting <i>BECN1</i> expression.	(Yi et al., 2018)
<i>ATG7</i>	<i>MIR106A</i>	-	Macrophages	<i>MIR106A</i> promotes <i>M. tuberculosis</i> survival through inhibiting autophagy and <i>MIR106A</i> expression is downregulated in the tuberculosis patients.	(Liu et al., 2020b)
	<i>MIR129</i>	<i>CircRAB11FIP1</i> (circRNA)	SKOV3 and A2780 cells (ovarian cancer)	<i>CircRAB11FIP1</i> induces autophagy through sponging <i>MIR129</i> and promoting <i>ATG14</i> and <i>ATG7</i> expression, which accelerates ovarian cancer cell proliferation and migration.	(Zhang et al., 2021b)
	<i>MIR1343-3p</i>	<i>GAS8-AS1</i>	TPC1	<i>GAS8-AS1</i> promotes autophagy and	(Qin et

		and BCPAP cells (PTC)	suppresses thyroid cancer cell proliferation partially through sponging <i>MIR1343-3p</i> and upregulating <i>ATG7</i> expression.	al., 2020)
<i>Mir143</i>	-	Mouse cardiac progenitor cells	<i>Mir143</i> mediates oxidative stress-induced cardiac progenitor cell apoptosis by inhibiting <i>Atg7</i> expression and autophagy.	(Ma et al., 2018)
	-	HL60 cells (AML)	<i>MIR143</i> promotes cytarabine toxicity in AML cells through inhibiting the expression of <i>ATG2B</i> and <i>ATG7</i> and thus suppressing autophagy.	(Zhang et al., 2020)
	<i>H19</i>	HL-1 cells (cardio myocyte)	Upregulation of <i>H19</i> relieves cardiomyocyte injury caused by hypoxia-reoxygenation via targeting <i>MIR143</i> and promoting <i>ATG7</i> expression and autophagy.	(Lv et al., 2021)
<i>MIR17-5p</i>	<i>HOTAIR</i>	A549 and BEAS-2B cells (lung tissue)	<i>HOTAIR</i> inhibition suppresses autophagy and cell apoptosis through binding <i>MIR17-5p</i> , which targets multiple <i>ATG</i> genes and alleviates lipopolysaccharide-induced lung injury <i>in vivo</i> .	(Li et al., 2021d)
<i>MIR181</i>	<i>CCAT1</i>	HepG2 and Huh7 cells (HCC)	<i>CCAT1</i> promotes HCC cells proliferation and upregulates autophagy through sponging <i>MIR181</i> and upregulating <i>ATG7</i> .	(Guo et al., 2019a)
<i>MIR192-5p</i>	-	Primary airway smooth muscle cells and <i>in vivo</i>	<i>MIR192-5p</i> suppresses airway smooth muscle cell proliferation partially through downregulating <i>ATG7</i> expression and attenuates inflammation and airway remodeling in asthmatic mice.	(Lou et al., 2020)
<i>MIR20B-5p</i>	-	U251 and A172 cells (glioma)	<i>MIR20B-5p</i> is downregulated by PSPD3R, which induces <i>ATG7</i> expression, autophagy and apoptosis in glioma cells.	(Wang et al., 2022b)
	<i>HOTAIR</i>	Primary hepatocytes	<i>HOTAIR</i> promotes autophagy in hepatocytes through targeting <i>Mir20B-5p</i> and upregulating <i>Atg7</i> expression.	(Tang et al., 2019)
<i>MIR3657</i>	<i>circRACGA</i>	BGC-	Knocking down <i>circRACGAP1</i> inhibits	(Ma et

	<i>P1</i> (circRNA)	823 and HGC-27 cells (gastric cancer)	autophagy and sensitizes gastric cancer cells to apatinib treatment through targeting <i>MIR3657</i> and <i>ATG7</i> .	al., 2020a)
<i>Mir375</i>	-	AR42J cells (rat pancreat ic acinar cells)	<i>Mir375</i> promotes inflammation and apoptosis of pancreatic acinar cells through inhibiting <i>Atg7</i> expression and thus suppressing autophagy.	(Zhao et al., 2020b)
	<i>TINCR</i>	SMMC- 7721 and Hep G2 cells (HCC)	<i>TINCR</i> mediates HCC proliferation and invasion through downregulating <i>MIR375</i> , which binds and inhibits <i>ATG7</i> expression.	(Tang et al., 2022)
<i>Mir485</i>	<i>SNHG3</i>	N2a cells (mouse neurobla stoma)	Overexpressing <i>SNHG3</i> accelerates oxygen and glucose deprivation and reperfusion-induced cell apoptosis through binding <i>Mir485</i> and upregulating <i>Atg7</i> expression and autophagy.	(Cao et al., 2020b)
<i>MIR582-5p</i>	<i>UCA1</i>	T24 and 5637 cells (bladder cancer)	<i>UCA1</i> knockdown suppresses bladder cancer cell proliferation, migration and invasion through downregulating autophagy via the <i>MIR582-5p-ATG7</i> axis.	(Wu et al., 2019)
<i>MIR588</i>	<i>SNHG8</i>	HCT116 and SW480 cells (colorect al cancer)	<i>SNHG8</i> promotes colorectal cancer cell proliferation and autophagy through sponging <i>MIR588</i> and upregulating <i>ATG7</i> expression.	(He et al., 2021)
<i>MIR615-3p</i>	<i>H19</i>	HCC827 and A549 (NSCLC)	<i>H19</i> mediates the erlotinib resistance of NSCLC cells through binding to <i>MIR615-3p</i> and upregulating <i>ATG7</i> expression.	(Pan and Zhou, 2020)
<i>Mir93</i>	<i>H19</i>	MMQ and GH3 cells (rat prolactin oma)	Overexpression of <i>H19</i> or knocking down <i>Mir93</i> promotes cabergoline treatment efficiency in pituitary tumor through upregulating <i>Atg7</i> expression.	(Wu et al., 2018, Wu et al., 2020)

	<i>MIR154</i>	-	T24 and UM-UC-3 cells (bladder cancer)	<i>MIR154</i> inhibits proliferation, migration, and invasion of bladder cancer cells through downregulating <i>ATG7</i> expression.	(Zhang et al., 2019a)
	<i>MIR138-5p</i>	-	A549 cells (NSCLC)	Upregulation of <i>MIR138-5p</i> through TRIM65 knockdown suppresses cisplatin resistance and autophagy activity via downregulating <i>ATG7</i> .	(Pan et al., 2019)
<i>ATG9A</i>	<i>Mir29b</i>	<i>Neat1</i>	JS1 cells (mouse hepatic stellate cell)	<i>Neat1</i> is important for hepatic stellate cell activation through sponging <i>Mir29b</i> and upregulating <i>Atg9a</i> expression and autophagy.	(Kong et al., 2019)
	<i>MIR34A</i>	<i>NEAT1</i>	HCT8 and SW480 (colorectal cancer)	<i>NEAT1</i> knockdown downregulates cell proliferation and induces its sensitivity to 5-FU through suppressing autophagy via <i>MIR34A</i> , which binds and regulates <i>ATG4B</i> and <i>ATG9A</i> expression.	(Liu et al., 2020a)
<i>ATG10</i>	<i>MIR221</i>	-	K1 cells (PTC)	<i>MIR221</i> and <i>MIR222</i> promote migration and invasiveness of PTC cells through inhibiting <i>ATG10</i> expression and thus autophagy. <i>MIR221</i> and <i>MIR222</i> can be used as a diagnostic biomarker of aggressive PTC.	(Shen et al., 2020a)
	<i>MIR222</i>				
	<i>MIR27B-3p</i>	-	SW480 and HCT116 cells (colorectal cancer)	<i>MIR27B-3p</i> enhances colorectal cancer cell sensitivity to oxaliplatin by inhibiting <i>ATG10</i> and autophagy.	(Sun et al., 2020)
	<i>MIR369-3p</i>	-	HEC-1-A cells (EEC)	Overexpression of <i>MIR369-3p</i> inhibits EEC cell proliferation and migration through suppressing <i>ATG10</i> expression and thus inhibiting autophagy.	(Liu et al., 2019)
	<i>MIR519A-3p</i>	<i>SNHG14</i>	SK-N-SH cells (neuroblastoma)	<i>SNHG14</i> knockdown suppresses MPP ⁺ neurotoxicity through downregulating <i>ATG10</i> via releasing <i>MIR519A-3p</i> .	(Zhuang et al., 2022)
<i>ATG12</i>	<i>Mir128a</i>	-	Chondrocyte and	<i>Mir128a</i> inhibits autophagy through targeting <i>Atg12</i> , and knocking down	(Lian et al., 2018)

		<i>in vivo</i>	<i>Mir128a</i> alleviates cartilage damage in osteoarthritis mouse models.	
<i>MIR214</i>	-	SW480 and HCT116 cells (colorectal cancer)	<i>MIR214</i> increases radiosensitivity of CRC cells through downregulating <i>ATG12</i> and radiation-induced autophagy.	(Hu et al., 2018)
<i>MIR26A-5p</i>	<i>HCG11</i>	MHCC97-H and Hep3B cells (HCC)	<i>HCG11</i> promotes the proliferation and metastasis of HCC cells through sponging <i>MIR26A-5p</i> and upregulating <i>ATG12</i> and autophagy.	(Li et al., 2019b)
	<i>KCNQ1OT1</i>	Human cardiomyocyte (HCM)	Knocking down <i>KCNQ1OT1</i> inhibits autophagy and cardiomyocyte apoptosis through the <i>MIR26A-5p-ATG12</i> axis.	(Li et al., 2021b)
<i>MIR372-3p</i>	<i>KCNQ1OT1</i>	A549 and H1975 cells (LUAD)	Knocking down <i>KCNQ1OT1</i> improves LUAD cell radiosensitivity through releasing <i>MIR372-3p</i> , which inhibits autophagy through binding <i>ATG5</i> and <i>ATG12</i> .	(He et al., 2020)
<i>MIR5095</i>	<i>HAGLROS</i>	Huh7 and HepG2. 2.15 cells (HCC)	<i>HAGLROS</i> protects HCC cells from apoptosis through targeting <i>MIR5095</i> and promoting autophagy.	(Wei et al., 2019)
<i>MIR520D-3p</i>	-	Human cardiomyocytes	Overexpressing <i>MIR520D-3p</i> attenuates the cardiomyocyte apoptosis induced by hypoxia-reoxygenation through downregulating <i>ATG12</i> expression.	(Wu et al., 2021)
<i>MIR570-3p</i>		143B and U2OS cells (osteosarcoma)	Attenuated metastasis of osteosarcoma cells by metformin is mediated by <i>MIR570-3p</i> upregulation and <i>ATG12</i> downregulation.	(Bao et al., 2018)
<i>MIR93</i>	<i>HOTAIR</i>	SW480 and HCT116 cells (colorectal)	Knocking down <i>HOTAIR</i> improves radiosensitivity of colorectal cancer cells through binding <i>MIR93</i> and regulating <i>ATG12</i> expression and autophagy.	(Liu et al., 2020c)

		al cancer)			
	<i>Mir1192</i>	<i>Gas5</i>	RAW26 4.7 (mouse macroph age)	<i>Gas5</i> preserves the expression of <i>Atg12</i> through sponging <i>Mir1192</i> and promotes autophagy in macrophages.	(Xu et al., 2021b)
	<i>MIR30E-5p</i>	<i>OIP5-AS1</i>	K562 cells (CML)	<i>OIP5-AS1</i> knockdown induces CML cell sensitivity to imatinib through the <i>MIR30E-5p-ATG12-autophagy</i> axis.	(Dai et al., 2021)
<i>ATG13</i>	<i>MIR206</i>	-	H9c2 cells (cardio myocyte)	Inhibition of hypoxia-induced cardiomyocyte autophagy and apoptosis by histamine is mediated by increased <i>MIR206</i> and <i>MIR216B</i> expression, which target and inhibit <i>ATG13</i> .	(Ding et al., 2018)
	<i>MIR216B</i>	-			
	<i>MIR646</i>	<i>PKD2</i> (circRNA)	SCC-15 and CAL-27 cells (OSCC)	<i>PKD2</i> promotes OSCC cell autophagy and cisplatin sensitivity through sponging <i>MIR646</i> and upregulating <i>ATG13</i> expression.	(Gao et al., 2022b)
<i>ATG14</i>	<i>MIR129</i>	<i>RAB11FIP</i> 1 (circRNA)	SKOV3 and A2780 cells (ovarian cancer)	<i>RAB11FIP1</i> induces autophagy through sponging <i>MIR129</i> and promoting <i>ATG14</i> and <i>ATG7</i> expression, which accelerates ovarian cancer cell proliferation and migration.	(Zhang et al., 2021b)
	<i>Mir129-5p</i>	-	H9c2 cells (heart tissue)	<i>Mir129-5p</i> inhibits autophagy and apoptosis induced by H ₂ O ₂ through binding to <i>Atg14</i> mRNA.	(Zhang et al., 2018a)
	<i>Mir152-5p</i>	-	Rat mesench ymal stem cells	<i>Mir152-5p</i> downregulates <i>Atg14</i> expression and thus inhibits autophagy, which is necessary for mesenchymal stem cell differentiation.	(Li et al., 2022a)
		<i>Pvt1</i>	Primary mouse hepatic stellate cells	<i>Pvt1</i> mediates hypoxia-induced autophagy, which is important for the activation of hepatic stellate cells via regulating <i>Mir152</i> and <i>Atg14</i> .	(Yu et al., 2020)
	<i>MIR186</i>	<i>SNHG14</i>	SW620 and	<i>SNHG14</i> promotes colorectal cancer cell proliferation and migration through	(Han et al., 2020)

		SW480 cells (colorectal cancer)	sponging <i>MIR186</i> and upregulating <i>ATG14</i> expression.	
<i>MIR188-3p</i>	<i>EIF3J-DT</i>	MGC803 and MKN45 cells (gastric cancer)	<i>EIF3J-DT</i> is highly expressed in the drug-resistant gastric tumor cells and promotes chemotherapy resistance through upregulating <i>ATG14</i> and autophagy via sponging <i>ATG14</i> -targeting <i>MIR188-3p</i> and directly binding and stabilizing <i>ATG14</i> .	(Luo et al., 2021)
<i>MIR199A-5p</i>	-	HepG2 cells (human hepatoma) and <i>in vivo</i>	<i>MIR199A-5p</i> inhibits insulin sensitivity through downregulating <i>ATG14</i> expression and autophagy in HepG2 cells and <i>in vivo</i> .	(Li et al., 2018)
<i>MIR375</i>	-	Huh7 and HepG2 (HCC)	Increased expression of <i>MIR375</i> sensitizes HCC cells to sorafenib through inhibiting <i>ATG14</i> expression and autophagy.	(Yang et al., 2020)
<i>MIR424-5p</i>	<i>CircCBFB</i> (circRNA)	Huh-7 and HCCLM3 (HCC)	<i>CircCBFB</i> promotes HCC cell development through binding <i>MIR424-5p</i> and upregulating <i>ATG14</i> expression and autophagy.	(Zhao et al., 2022)
<i>MIR493</i>	<i>SNHG1</i>	T24 and RT4 cells (bladder cancer)	<i>SNHG1</i> promotes bladder cancer cell proliferation and invasion through sponging <i>ATG14</i> -targeting <i>MIR493</i> and upregulating autophagy.	(Guo et al., 2021)
<i>MIR619-5p</i>	<i>PVT1</i>	PANC-1 and SW1990 cells (pancreatic cancer)	<i>PVT1</i> mediates gemcitabine resistance and promotes autophagy activity through sponging <i>MIR619-5p</i> and upregulating <i>ATG14</i> .	(Zhou et al., 2020)
<i>ATG16L1</i>	<i>MIR106A</i>	-	Macrophages	<i>MIR106A</i> promotes <i>M. tuberculosis</i> survival through inhibiting autophagy and <i>MIR106A</i> expression is downregulated in tuberculosis patients.
	<i>MIR142-3p</i>	-	HepG2 and	Ectopic expression of <i>MIR142-3p</i> sensitizes HCC cells to sorafenib

		SMMC-7721 cells (HCC)	treatment via targeting <i>ATG5</i> and <i>ATG16L1</i> and inhibiting autophagy.	2018b)	
<i>MIR17-5p</i>	<i>HOTAIR</i>	A549 and BEAS-2B cells (lung tissue)	<i>HOTAIR</i> inhibition suppresses autophagy and cell apoptosis through releasing <i>MIR17-5p</i> , which targets multiple <i>ATG</i> genes and alleviates lipopolysaccharide-induced lung injury <i>in vivo</i> .	(Li et al., 2021d)	
<i>MIR874</i>	-	SGC7901 and BGC823 cells (gastric cancer)	Overexpressing <i>MIR874</i> promotes sensitivity to chemotherapy through downregulating <i>ATG16L1</i> and autophagy.	(Huang et al., 2018)	
<i>RB1CC1/IP200</i>	<i>Mir124-3p</i>	-	BV2 cells (microglia) and HT22 cells (mouse hippocampal cell)	<i>Mir124-3p</i> in microglial exosomes can protect neurons from trauma-induced injury through inhibiting <i>Rb1cc1</i> and autophagy in neurons.	(Li et al., 2019a)
	<i>MIR20A</i>	-	LoVo and SW48 cells (colorectal cancer)	<i>MIR20A</i> is downregulated under hypoxia conditions, which inhibits hypoxia induced autophagy via downregulating <i>ATG5</i> and <i>RB1CC1</i> expression.	(Che et al., 2019)
	<i>Mir224-3p</i>	-	N2a cells (mouse neuroblastoma) and primary cultured neurons	Overexpression of <i>Mir224-3p</i> alleviates apoptosis and ROS production and protects cells from ischemic/reperfusion injury through downregulating <i>Rb1cc1</i> expression.	(Deng et al., 2019)

<i>WIPI2</i>	<i>MIR195-5p</i>	<i>CERS6-AS1</i>	BxPC-3 and PANC-1 cells (pancreatic cancer)	<i>CERS6-AS1</i> facilitates pancreatic cancer cell proliferation and suppresses apoptosis by sponging <i>MIR195-5p</i> and upregulating <i>WIPI2</i> expression.	(Gao et al., 2022a)
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Abbreviations: AML: acute myeloid leukemia; BCG: Bacillus Calmette-Guerin; CML: chronic myeloid leukemia; DLBCL: diffuse large B-cell lymphoma; EEC: endometrioid adenocarcinoma; GCA: gastric cardia adenocarcinoma; GIST: gastrointestinal stromal tumors; HCC: hepatocellular carcinoma; HUVEC: human umbilical vein endothelial cell; LUAD: lung adenocarcinoma; LUSC: lung squamous cell carcinoma; MPP⁺: 1-methyl-4-phenylpyridinium; MPTP: 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine; NSCLC: non-small cell lung cancer; OSCC: oral squamous cell carcinoma; PDA: pancreatic ductal adenocarcinoma; PTC: papillary thyroid carcinoma.

¹Most of the competing endogenous RNA (ceRNA) listed in the table are lncRNA; if it is a circRNA, it is indicated in parentheses.

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