Table SII. lincRNAs promote the expression and stability of IGF1R by regulating miRNAs.

lincRNAs	miRNAs	Functions in cancer progression
MLETA1	miR-186-5p and miR-497-5p	Exosomal lncRNA MLETA1 drives non-small cell lung cancer metastasis by dual sponging of miR-186-5p and miR-497-5p to elevate EGFR and IGF1R expression respectively. This exosome-mediated intercellular communication represents a promising diagnostic biomarker and therapeutic target for metastatic NSCLC (62).
LINC02381	/	LINC02381 functions as a tumor suppressor in breast cancer by downregulating IGF1R expression and attenuating PI3K/AKT signaling, ultimately impairing cell proliferation while promoting apoptosis. This lncRNA-mediated regulation of the IGF1R axis presents a novel mechanism for controlling breast cancer progression (63).
HULC	/	lncRNA HULC epigenetically activates IGF1R transcription through direct binding to intragenic regulatory elements and promoting H3K9 acetylation, thereby stimulating the PI3K/AKT pathway. This HULC-IGF1R axis promotes breast cancer metastasis and cisplatin resistance by enhancing cancer stemness, representing a novel therapeutic target for combination therapy approaches (64).
OIP5-AS1	miR-147a	lncRNA OIP5-AS1 promotes cervical cancer metastasis and EMT progression through a competitive endogenous RNA mechanism whereby it sequesters miR-147a, resulting in subsequent upregulation of IGF1R expression. This OIP5-AS1/miR-147a/IGF1R regulatory axis represents a potential therapeutic target for inhibiting cervical cancer invasion and metastasis (65).
MIAT	miR-488-3p	lncRNA MIAT promotes colorectal cancer progression by functioning as a molecular sponge for miR-488-3p, leading to subsequent upregulation of IGF1R expression and enhanced tumor proliferation, migration, invasion and glycolytic metabolism. This MIAT/miR-488-3p/IGF1R regulatory axis represents a promising therapeutic target for colorectal cancer intervention (66).
LINC01287	miR-98	LINC01287 promotes breast cancer progression by functioning as a competitive endogenous RNA that sequesters miR-98, leading to upregulated IGF1R expression and subsequent activation of the MEK/ERK signaling pathway. This lncRNA-mediated regulatory mechanism enhances tumor cell proliferation, migration and invasion, suggesting LINC01287 as a potential therapeutic target for IGF1R-driven breast cancer (11).
SNHG11	/	lncRNA SNHG11 promotes prostate cancer progression by functioning as a competitive endogenous RNA that sequesters miR-184, leading to subsequent upregulation of IGF1R expression and enhanced tumor cell proliferation

		and metastasis. Rescue experiments confirm that IGF1R overexpression reverses the tumor-suppressive effects of SNHG11 knockdown, establishing this lncRNA-miRNA-IGF1R axis as a critical regulatory mechanism in prostate cancer pathogenesis (67).
AFAP1-AS1	miR-15b	lncRNA AFAP1-AS1 promotes castration-resistant prostate cancer progression by functioning as a ceRNA that sequesters miR-15b, leading to IGF1R upregulation and enhanced tumor cell proliferation and invasion. Paradoxically, enzalutamide treatment increases AFAP1-AS1 expression, suggesting this lncRNA-mediated pathway may contribute to therapeutic resistance in advanced prostate cancer (68).
CASC11	miR-145	ncRNA CASC11 drives prostate cancer aggressiveness by functioning as a ceRNA that sequesters miR-145, resulting in IGF1R upregulation and subsequent activation of the PI3K/AKT/mTOR signaling pathway. This CASC11/miR-145/IGF1R regulatory axis represents a promising therapeutic target for suppressing tumor progression and overcoming treatment resistance in advanced prostate cancer (69).
LINC00324	miR-139-5p	LINC00324 promotes NSCLC progression by functioning as a molecular sponge for miR-139-5p, leading to subsequent upregulation of IGF1R expression and enhanced tumor cell proliferation and invasion. This lncRNA-mediated regulatory axis represents both a potential diagnostic biomarker and therapeutic target for NSCLC intervention strategies (70).
LINC00319	miR-147a	LINC00319 drives cervical cancer progression by functioning as a molecular sponge for miR-147a, leading to subsequent upregulation of IGF1R expression and activation of its downstream oncogenic signaling pathways. This lncRNA-mediated regulatory axis represents a potential therapeutic target for cervical cancer intervention, particularly for inhibiting tumor proliferation and metastasis (71).
Linc00210	miR-195-5p	Linc00210 drives thyroid cancer progression by functioning as a ceRNA that sequesters miR-195-5p, leading to IGF1R upregulation and subsequent activation of the PI3K/Akt signaling pathway. This lncRNA-mediated regulatory axis represents a potential therapeutic target for suppressing tumor growth and metastasis in thyroid cancer (72).
LINC00958	miR-378a-3p	LINC00958 promotes bladder cancer progression by functioning as a ceRNA that sequesters miR-378a-3p, leading to IGF1R upregulation and enhanced tumor cell proliferation, migration and invasion. This lncRNA-mediated regulatory axis represents a potential therapeutic target for bladder cancer intervention strategies (73).
LINC00339	miR-497-5p	LINC00339 promotes pancreatic cancer

	progression by functioning as a ceRNA that sequesters miR-497-5p, leading to IGF1R upregulation and enhanced tumor cell proliferation and metastasis. This lncRNA-mediated regulatory axis represents a potential diagnostic biomarker and therapeutic target for pancreatic cancer intervention strategies (74).

IGF1R, insulin-like growth factor 1 receptor; lincRNA, long intergenic noncoding RNA; miR/miRNA, microRNA; NSCLC, non-small cell lung cancer; lncRNA, long non-coding RNA; ceRNA, competing endogenous RNA.