

Table S1 Information of the screened drugs

Drug	Biological activity	Target (IC ₅₀)	Molecular genetics
Cyclophosphamide	Immunosuppressant	AChE (511 μM)	Cyclophosphamide promotes the proliferation inhibition of mouse ovarian granulosa cells and premature ovarian failure by activating the lncRNA-Meg3-p53-p66Shc pathway
Zoledronate	Bisphosphonate		Zoledronic acid inhibits osteoclast differentiation and function through the regulation of NF-κB and JNK signaling pathways
Fulvestrant	Antiestrogen	Estrogen receptor (9.4 nM)	Fulvestrant-treated breast cancer cells increased the expression of lncRNA H19 and regulated drug resistance
JAK1	TYK2/JAK1 inhibitor	Tyk2 (6 nM)	JAK1 shows anti-inflammatory effect by regulating the expression of related TYK2/JAK1-regulated genes, as well as the formation of Th1, Th2, and Th17 cells
Sinularin	Natural products	SK-HEP-1 cells (10 μM)	Sinularin, an Anti-Cancer Agent-Causing Mitochondria-Modulated Apoptosis and Cytoskeleton Disruption in Human Hepatocellular Carcinoma
Leflunomide	Pyrimidine synthesis inhibitor	Protein tyrosine kinases (30–100 nM)	Leflunomide prevents the expansion of activated and autoimmune lymphocytes by interfering with cell cycle progression due to insufficient rUMP production and utilizing mechanisms involving p53
XAV939	Tankyrase inhibitor	TNKS1 (5 nM); TNKS2 (2 nM); ARTD2 (479 nM); ARTD1 (5,500 nM)	XAV-939 inhibited Wnt/β-catenin signaling and promoted the expression of SFRP3 and SFRP4
BMS-345541	Selective inhibitor of the catalytic subunits of IKK	IKK-1 (4 μM); IKK-2 (0.3 μM)	BMS-345541 inhibited LPS-stimulated tumor necrosis factor-α, interleukin-1β, interleukin-8 and interleukin-6 in THP-1 cells
Entinostat	Selective class I HDAC inhibitor	HDAC1 (243 nM); HDAC2 (453 nM); HDAC3 (248 nM)	Entinostat can induce autophagy and apoptosis
BMS-754807	IGF-1R/IR inhibitor	IR (1.7 nM); IGF-1R (1.8 nM)	BMS-754807 inhibits the activities of IGF-1R and IR by inhibiting the catalytic domain of IGF-1R, and induces apoptosis of tumor cells
Cytochalasin-b	Cell-permeable mycotoxin	F-actin, with Mg ²⁺ (2.2 nM); F-actin, with Mg ²⁺ /K ⁺ (1.4 nM)	Cytochalasin B binds to the barbed ends of actin filaments, disrupting actin polymer formation and preventing cell migration
Linifanib	Multi-target inhibitor of VEGFR and PDGFR family	PDGFRβ (66 nM); FLT3 (4 nM)	Linifanib is a specific miR-10b inhibitor that blocks miR-10b biogenesis

Table S2 The primers for qRT-PCR

Gene	Primer sequence (5'-3')	Fragment length (bp)	Annealing temperature (°C)
Nova1			
M-Nova1-S	CACAGCAGGTCTGATAATAGGGAA	257	60
M-Nova1-A	GATTGGAATTTGCCACTGGAC		60
Trib3			
M-Trib3-S	CTCTCCGGCAGATGGCTAG	111	60
M-Trib3-A	CAGCTTCGTCCTCTCACAGTTG		60
UQCRH			
M-UQCRH-S	AGGACGAACGAAAGATGCTCAC	165	60
M-UQCRH-A	ACCGGGAAGACACGCGATT		60
Psmb5			
M-Psmb5-S	CAATAAGGAACGCATCTCGGT	291	60
M-Psmb5-A	CTGTAGGTGGCTTGGTAGATGG		60
dio3os			
M-dio3os-S	GTACAGGGGAGCCCACTTTC	103	60
M-dio3os-A	ATGCATCTGCTGAACAGGCT		60
Aox1			
M-Aox1-S	ACAGCATAAACCCAGCCCTTG	143	60
M-Aox1-A	GGCAGGAATCTTGATTGGTTTG		60
Mfap4			
M-Mfap4-S	TGGCTATACCCTCTACGTGGCT	187	60
M-Mfap4-A	GTAGAAACCGTTGAGATTGGCG		60
Abca9			
M-Abca9-S	TGTGGTGGATCTGGGACGTGT	287	60
M-Abca9-A	CTGAATGGTCTTTGTGCTCTTTTGT		60
Ackr1			
M-Ackr1-S	GGGAACTGTCTGTATCCGGTG	261	60
M-Ackr1-A	CCAGTGGAAGAAAGGTCTGAGAAT		60

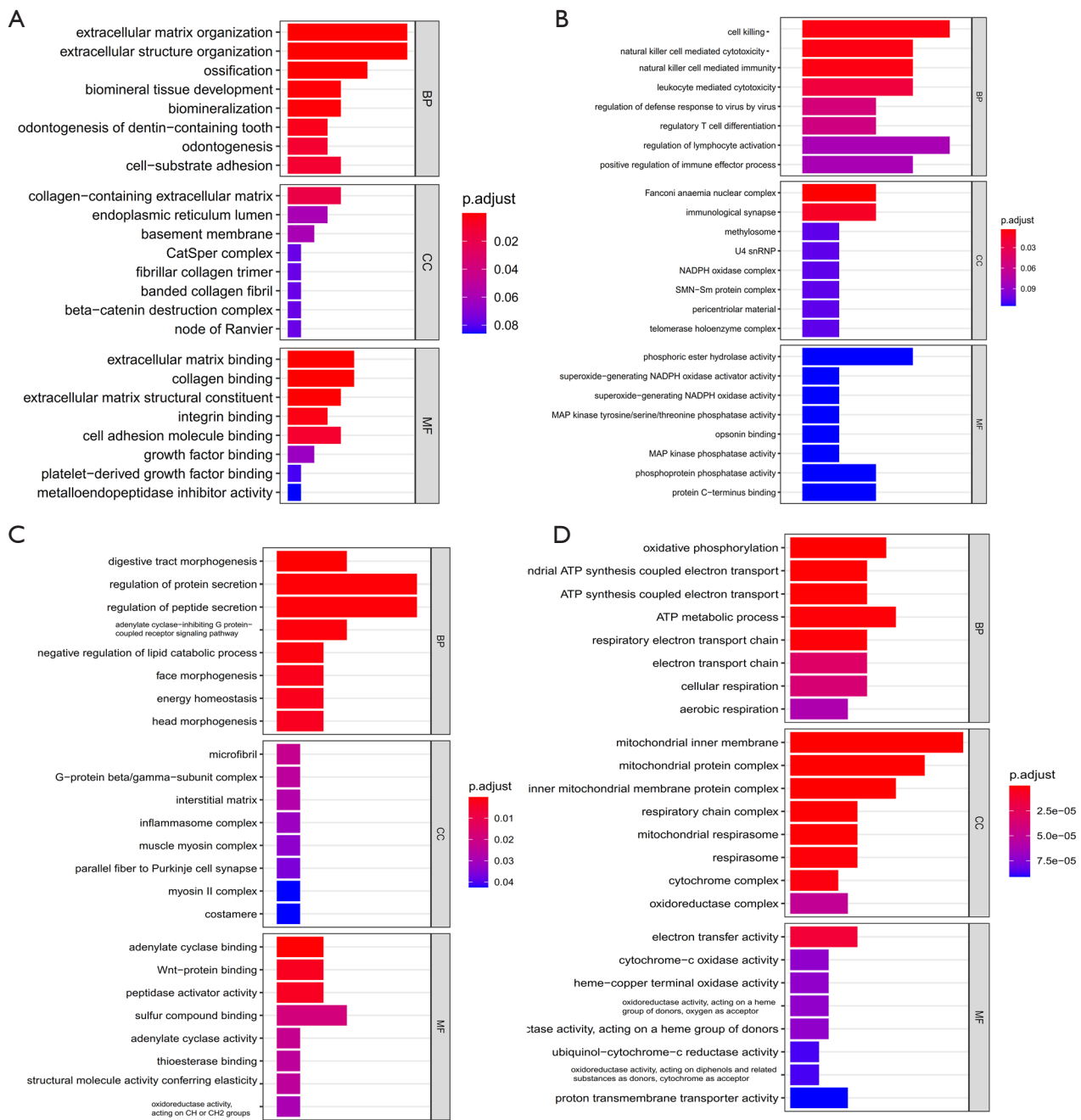


Figure S1 Gene Ontology enrichment analysis. (A) GO analysis was used to determine the significant enrichment pathways of mRNAs (upregulated lncRNA: $r > 0.5$ and $P < 0.01$); (B) GO analysis was used to determine the significant enrichment pathways of mRNAs (downregulated lncRNA: $r < -0.4$ and $P < 0.01$); (C) GO analysis was used to determine the significant enrichment pathways of mRNAs (upregulated lncRNA: $r > 0.5$ and $P < 0.01$); (D) GO analysis was used to determine the significant enrichment pathways of mRNAs (downregulated lncRNA: $r < -0.4$ and $P < 0.01$). mRNA, messenger RNA; GO, Gene Ontology.