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Supplemental Information

lncRNA lnc-POP1-1 upregulated by VN1R5 promotes cisplatin resistance in head and neck squamous cell carcinoma through interaction with MCM5

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Supplementary Figures

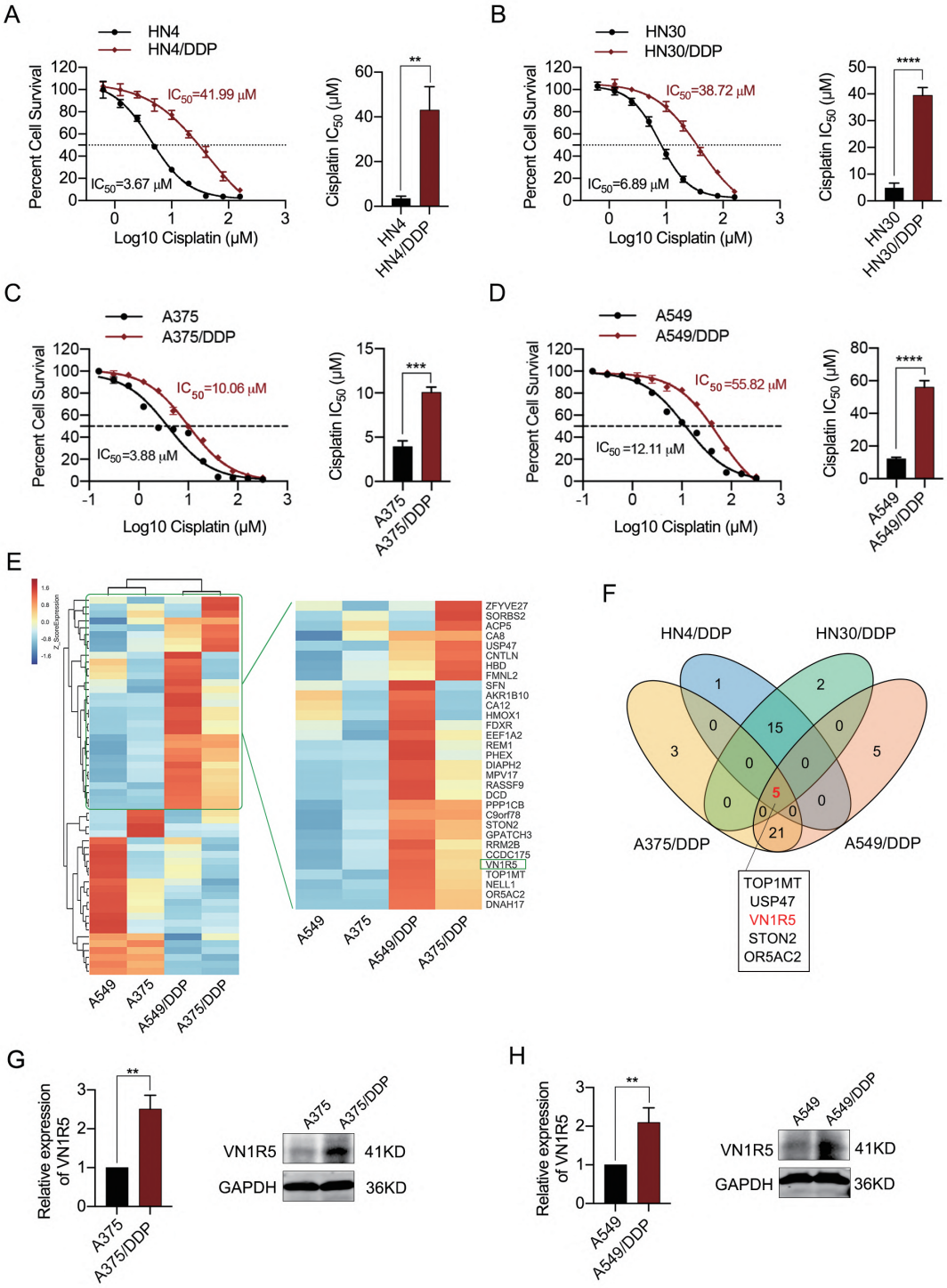

Figure S1. Cisplatin-induced resistance in HNSCC and A375/A549 cells. VN1R5 expression and cell survival were measured.

A. IC_{50} values of cisplatin in cisplatin-sensitive and -resistant HN4 cells established for microarray analysis and subsequent experiments.

B. IC_{50} values of cisplatin in cisplatin-sensitive and -resistant HN30 cells established for microarray analysis and subsequent experiments.

C. IC_{50} values of cisplatin in cisplatin-sensitive and -resistant A375 cells established for microarray analysis.

D. IC_{50} values of cisplatin in cisplatin-sensitive and -resistant A549 cells established for microarray analysis.

E. Microarray analysis of cisplatin-sensitive/-resistant A375 and A549 cells. VN1R5 was upregulated in cisplatin-resistant HNSCC cells.

F. VN1R5 was one of the upregulated proteins in the four cisplatin-resistant cell lines.

G. VN1R5 expression was measured by qPCR and Western blotting in cisplatin-sensitive and -resistant A375 cells.

H. VN1R5 expression was measured by qPCR and Western blotting in cisplatin-sensitive and -resistant A549 cells.

** $p<0.01$, *** $p<0.001$, **** $p<0.0001$. Error bars, means ± SDs.

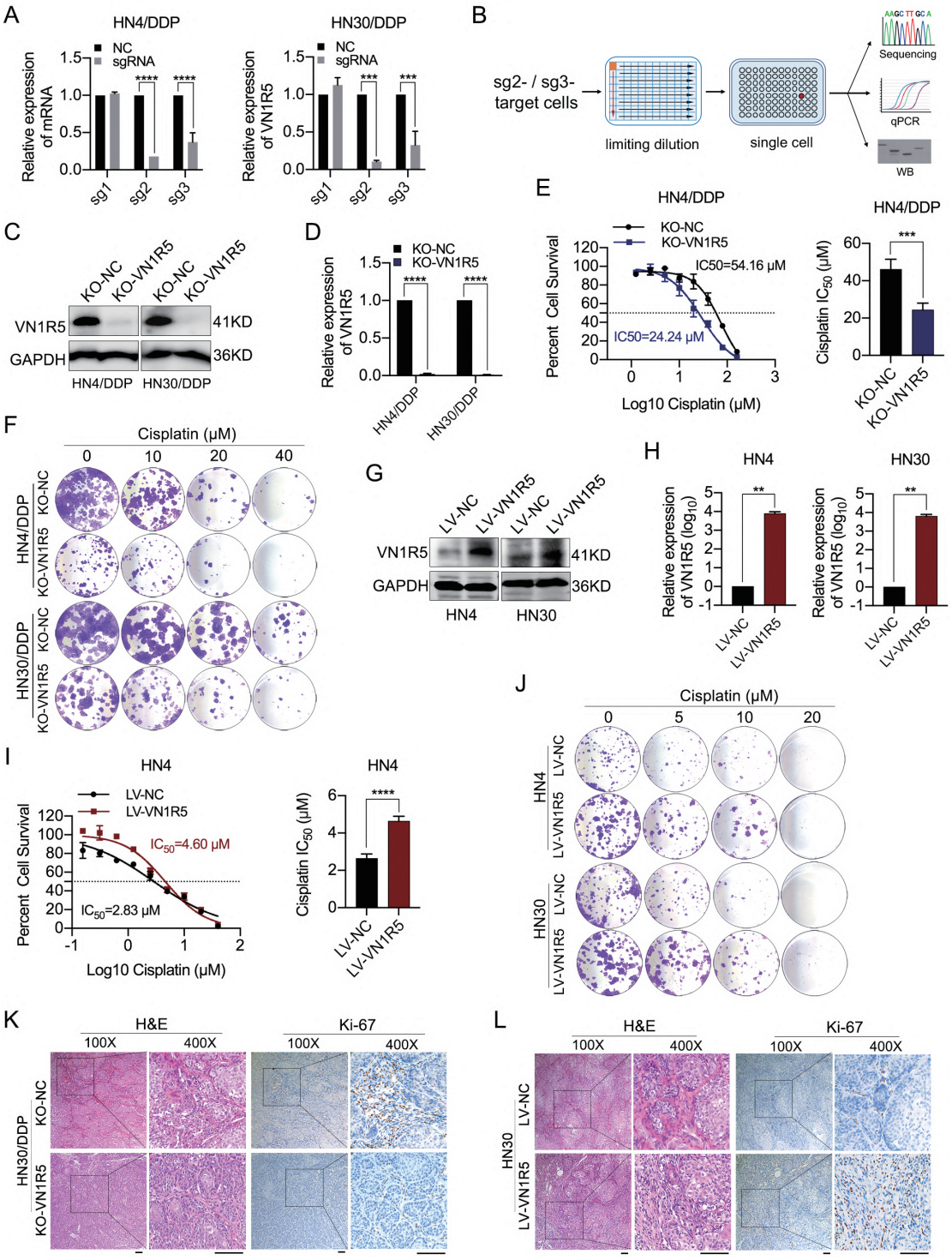

Figure S2. Effect of VN1R5 on cisplatin resistance in HNSCC cells.

A. The knockdown efficiency was analyzed by qPCR in HN4/DDP and HN30/DDP cells stably transfected with the targeted sgRNA CRISPR/Cas9 vector.

B. Procedure of VN1R5 KO in HNSCC cells. HN4/DDP and HN30/DDP stably transfected with sg2- and sg3-targeted vectors were used for further screening and verification. The limited dilution method was used to obtain single cells, after which VN1R5 expression analysis and DNA sequencing were performed.

C. After selection of the cell clones grown from single cells, the KO efficiency of VN1R5 was verified by Western blotting.

D. After selection of the cell clones grown from single cells, the KO efficiency of VN1R5 was verified by qPCR.

E. Compared with NC-transfected cells (black line), HN4/DDP cells with VN1R5 downregulation were sensitized to cisplatin (blue line). The IC_{50} values are shown on the right.

F. The colony formation ability of KO-VN1R5 HN4/DDP and HN30/DDP cells treated with a specific cisplatin concentration gradient is shown.

G. The relative expression levels of VN1R5 in HN4 and HN30 cells stably transduced with LV-VN1R5 were measured by Western blotting.

H. The relative expression levels of VN1R5 in HN4 and HN30 cells stably transduced with the VN1R5 lentiviral vector (LV-VN1R5) were measured by qPCR.

I. Compared with NC-transfected cells (black line), HN4 cells with VN1R5 upregulation (red line) exhibited cisplatin resistance. The IC_{50} values are shown on the right.

J. The colony formation ability of VN1R5-overexpressing HN4 and HN30 cells treated with a specific cisplatin concentration gradient is shown.

K. H&E and Ki-67 staining of xenograft tissues from the KO-VN1R5 and KO-NC groups. Scale bar: 100 μm.

L. H&E and Ki-67 staining of xenograft tissues from the LV-VN1R5 and LV-NC groups. Scale bar: 100 μm.

** $p<0.01$, *** $p<0.001$, **** $p<0.0001$. Error bars, means ± SDs.

(NC, negative control; KO, knockout; LV, lentiviral vector)

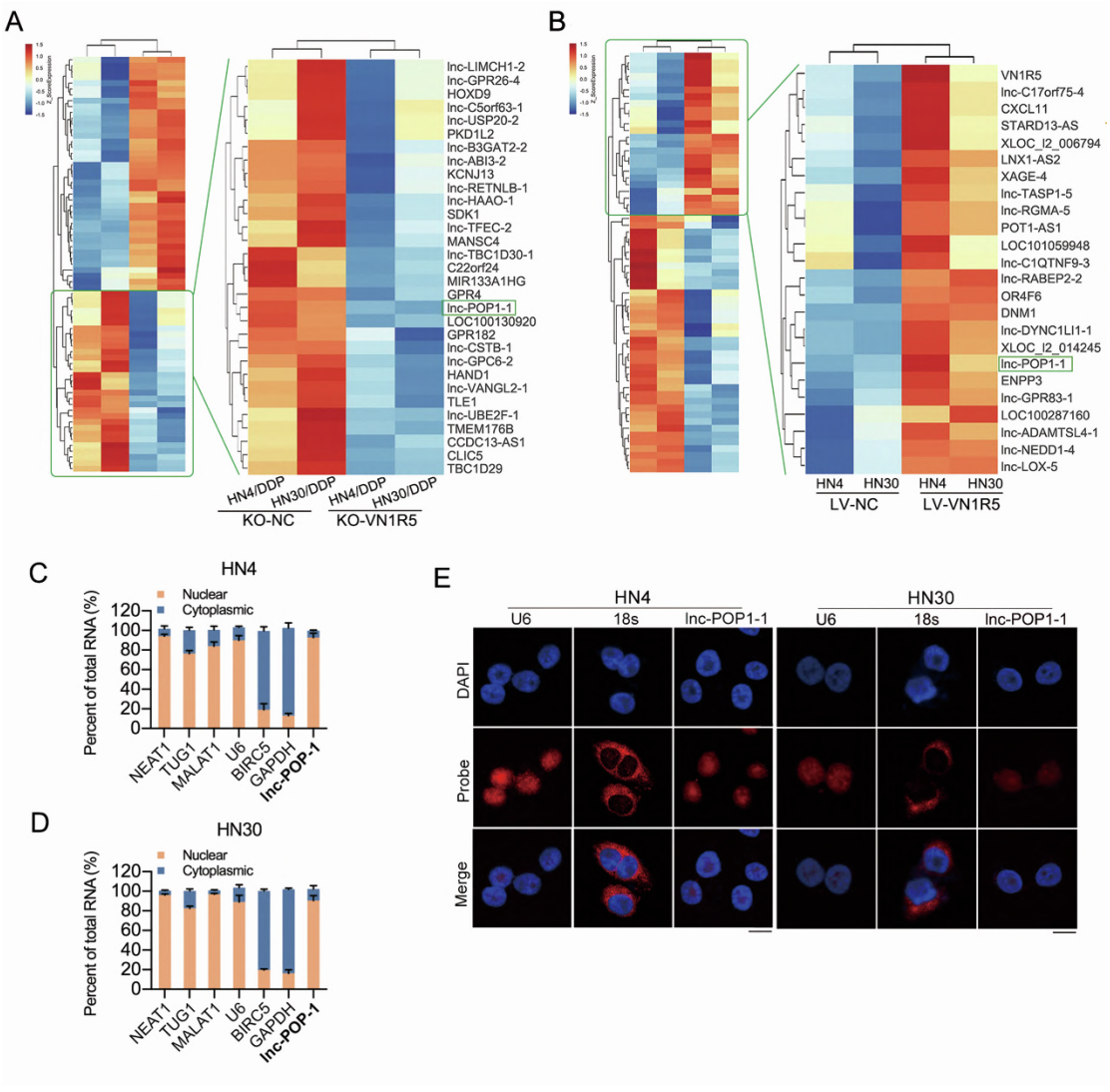

Figure S3. lnc-POP1-1 expression and localization in HNSCC cells.

A. Microarray analysis to assess the gene expression profiles of KO-VN1R5 HN4/DDP and HN30/DDP cells.

B. Microarray analysis to assess the gene expression profiles of VN1R5-overexpressing (LV-VN1R5) HN4 and HN30 cells.

C. Cell nuclear/cytoplasmic fractionation and qPCR showed the cellular distribution of lnc-POP1-1 in HN4 cells. NEAT1, TUG1, MALAT1, U6, BIRC5 and GAPDH were used as separation quality standards and endogenous controls, as appropriate.

D. Cell nuclear/cytoplasmic fractionation and qPCR showed the cellular distribution of lnc-POP1-1 in HN30 cells. NEAT1, TUG1, MALAT1, U6, BIRC5 and GAPDH were used as separation quality standards and endogenous controls, as appropriate. Error bars, means ± SDs.

E. FISH analysis of lnc-POP1-1 in HN4 and HN30 cells. (The nuclei were stained with DAPI. U6 and 18S rRNA were used as nuclear and cytoplasmic markers, respectively. Scale bar: 10 μm.)

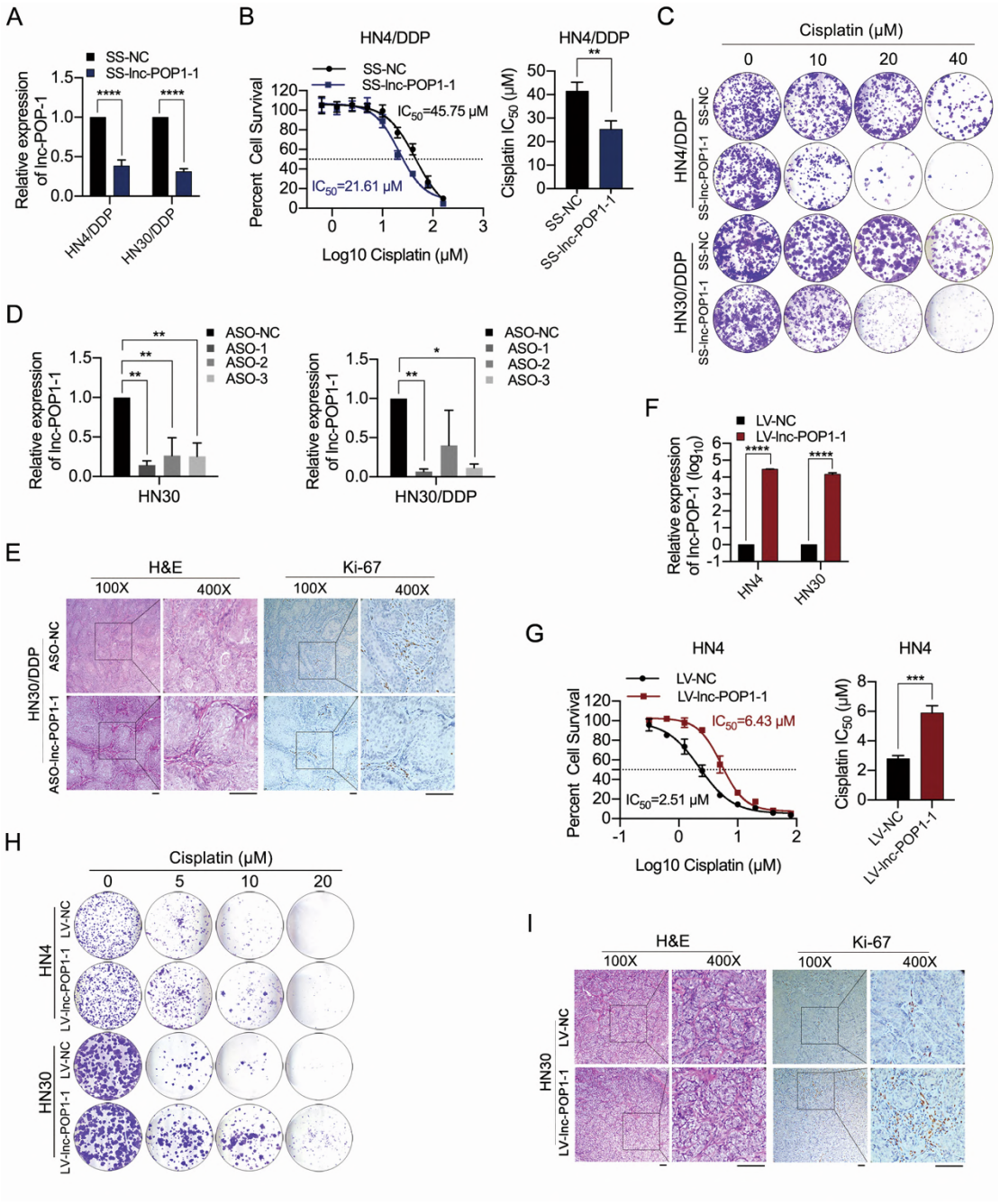

Figure S4. Effect of lnc-POP1-1 on cisplatin resistance in HNSCC cells.

A. The relative expression levels of lnc-POP1-1 in HN4/DDP and HN30/DDP cells transfected with SS-lnc-POP1-1 were determined by qPCR.

B. Compared with NC-transfected cells (black line), HN4/DDP cells with lnc-POP1-1 downregulation were sensitized to cisplatin (blue line). The IC_{50} values are shown on the right.

C. The colony formation ability of lnc-POP1-1-silenced HN4/DDP and HN30/DDP cells treated with a specific cisplatin concentration gradient is shown.

D. The silencing efficiency of ASO-lnc-POP1-1 (ASO-1, ASO-2 and ASO-3) in HN30 and HN30/DDP cells was determined by qPCR.

E. H&E and Ki-67 staining of xenograft tissues from the ASO-lnc-POP1-1 and ASO-NC groups. Scale bar: 100 μm.

F. The relative expression levels of lnc-POP1-1 in HN4 and HN30 cells stably transduced with the LV-lnc-POP1-1 vector were determined by qPCR.

G. Compared with NC-transfected cells (black line), HN4 cells with lnc-POP1-1 upregulation were resistant to cisplatin (red line). The IC_{50} values are shown on the right.

H. The colony formation ability of lnc-POP1-1-overexpressing HN4 and HN30 cells treated with a specific cisplatin concentration gradient is shown.

I. H&E and Ki-67 staining of xenograft tissues from the LV-lnc-POP1-1 and LV-NC groups. Scale bar: 100 μm.

* $p<0.05$, ** $p<0.01$, *** $p<0.001$, **** $p<0.0001$. Error bars, means ± SDs.

(NC, negative control; SS, Smart Silencer; LV, lentiviral vector; ASO, antisense oligonucleotide)

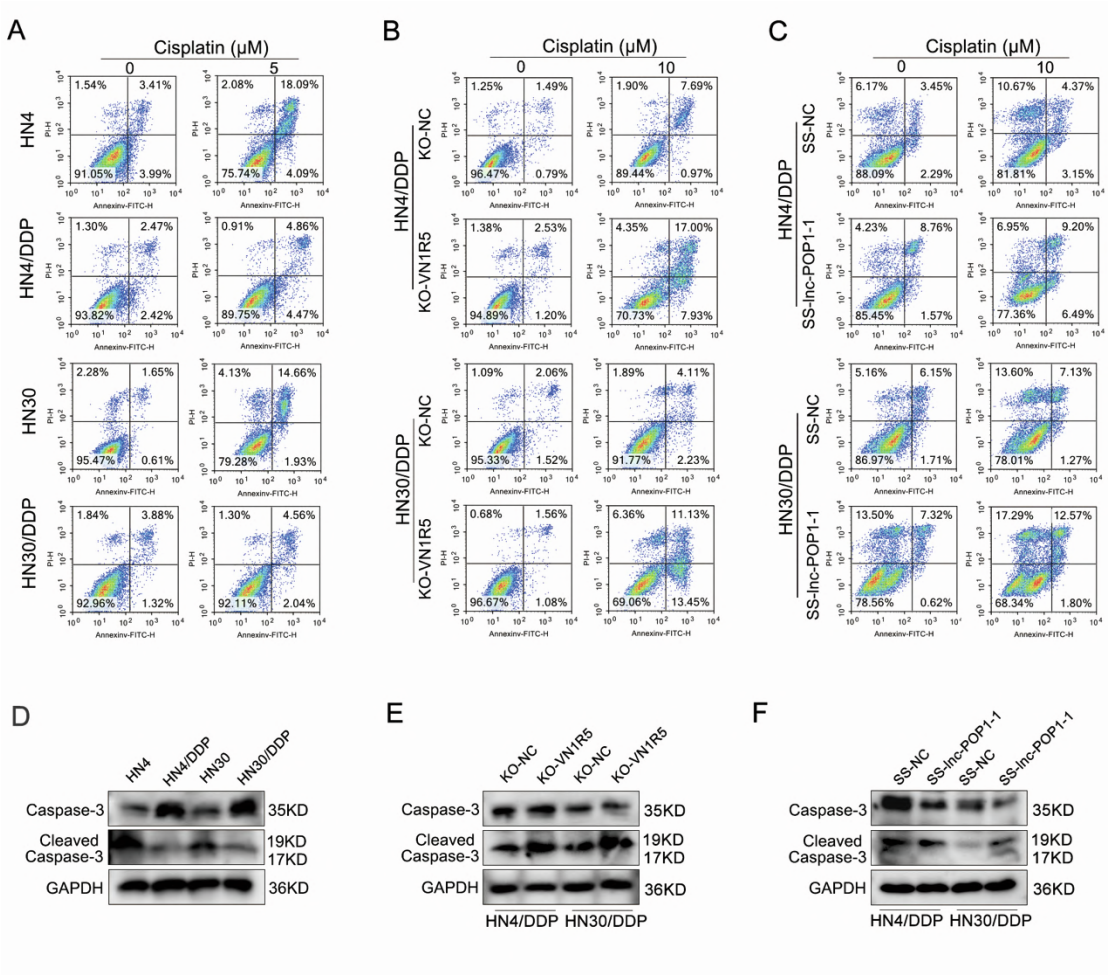

Figure S5. VN1R5 and lnc-POP1-1 affect HNSCC cell apoptosis.

A. Apoptosis was detected using flow cytometry in cisplatin-sensitive/-resistant HN4 and HN30 cells treated with 0 or 5 μM cisplatin for 48 h.

B. Apoptosis was detected using flow cytometry in HN4/DDP and HN30/DDP cells treated with 0 or 10 μM cisplatin after KO of VN1R5.

C. Apoptosis was detected using flow cytometry in HN4/DDP and HN30/DDP cells treated with 0 or 10 μM cisplatin after knockdown of lnc-POP1-1.

D. The expression level of Cleaved Caspase-3 was analyzed by Western blotting in cisplatin-sensitive/-resistant HN4 and HN30 cells treated with 5 μM cisplatin for 24 h.

E. The expression level of Cleaved Caspase-3 was analyzed by Western blotting in KO-VN1R5 HN4/DDP and HN30/DDP cells treated with 10 μM cisplatin.

F. The expression level of Cleaved Caspase-3 was analyzed by Western blotting in HN4/DDP and HN30/DDP cells treated with 10 μM cisplatin after knockdown of lnc-POP1-1.

(NC, negative control; SS, Smart Silencer; LV, lentiviral vector; KO, knockout)

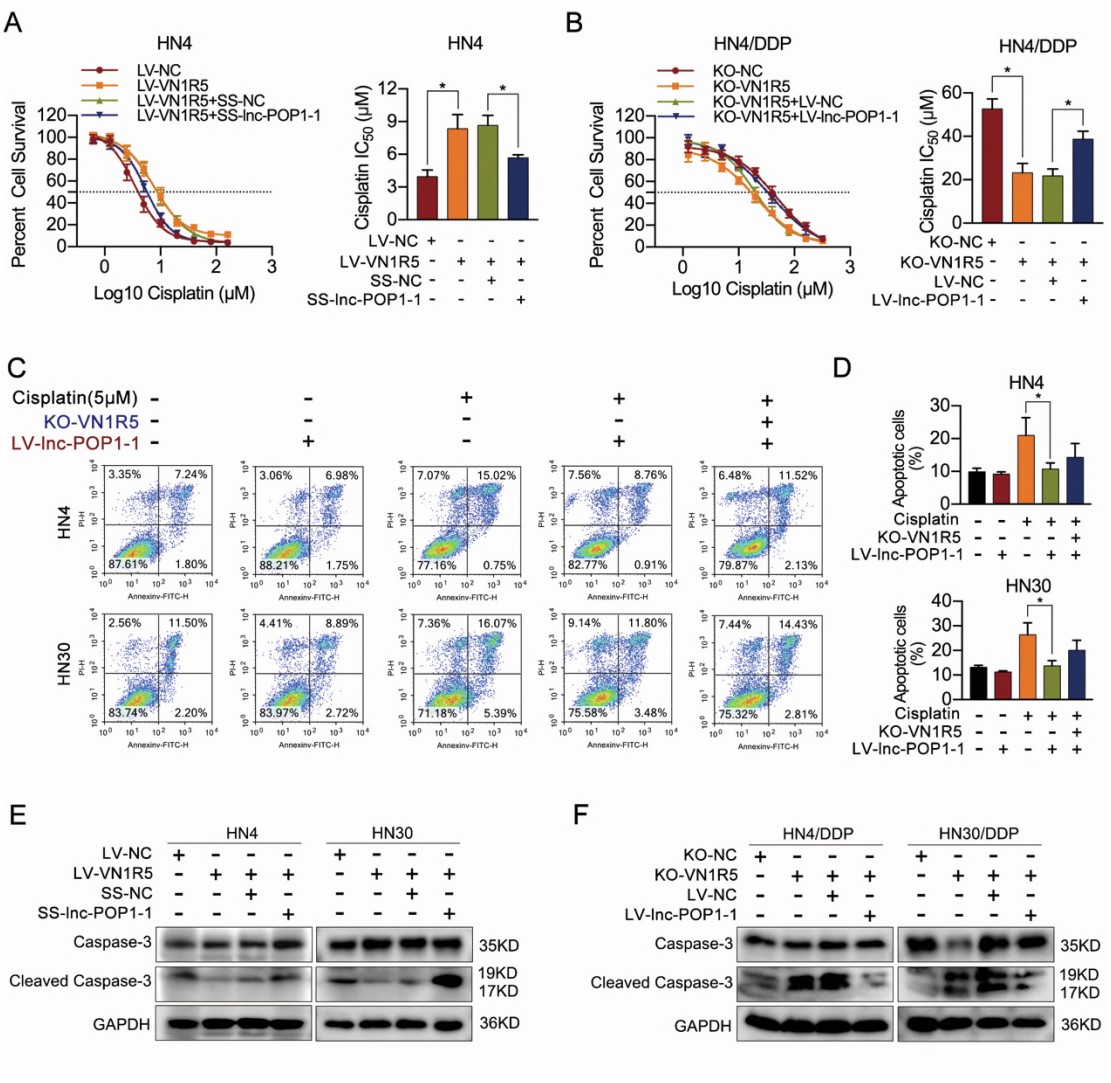

Figure S6. lnc-POP1-1 was regulated by VN1R5 to affect cisplatin resistance in HNSCC cells.

A. Cell viability was detected by CCK-8 assays when lnc-POP1-1 was knocked down in HN4 cells stably transfected with LV-VN1R5. The IC_{50} values are shown on the right.

B. Cell viability was detected by CCK-8 assay when lnc-POP1-1 was overexpressed in KO-VN1R5 HN4/DDP cells. The IC_{50} values are shown on the right.

C, D. Apoptosis was detected using flow cytometry in lnc-POP1-1-overexpressing HN4 and HN30 cells treated with 0 or 5 μM cisplatin after KO of VN1R5.

E. The expression level of Cleaved Caspase-3 was analyzed by Western blotting in VN1R5-overexpressing HN4 and HN30 cells treated with 5 μM cisplatin after knockdown of lnc-POP1-1.

F. The expression level of Cleaved Caspase-3 was analyzed by Western blotting in KO-VN1R5 HN4/DDP and HN30/DDP cells treated with 10 μM cisplatin after overexpressing lnc-POP1-1.

* $p<0.05$. Error bars, means ± SDs.

(NC, negative control; SS, Smart Silencer; LV, lentiviral vector; KO, knockout)

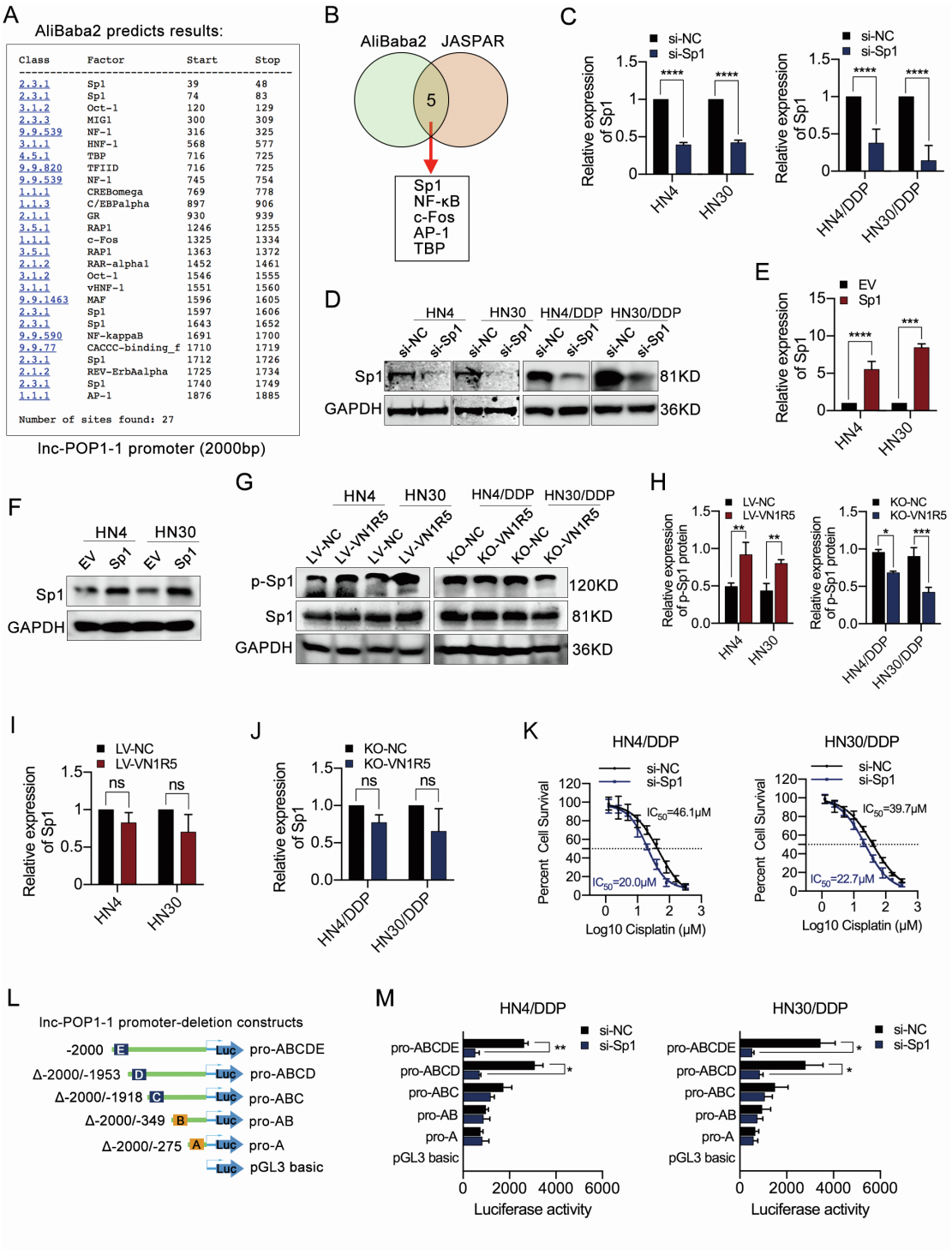

Figure S7. Sp1 promoter activity of Sp1 and lnc-POP1-1 expression regulation by VN1R5.

A. Prediction of the TFs of lnc-POP1-1 with AliBaba2.1 (http://gene-regulation.com/pub/programs/alibaba2/index.html).

B. The common predicted TFs were analyzed with AliBaba2.1 and JASPAR (http://jaspar.genereg.net).

C, D. The relative expression levels of Sp1 in cisplatin-sensitive and -resistant HNSCC cells transfected with si-Sp1 were determined by qPCR (C) and Western blotting (D).

E, F. The relative expression levels of Sp1 in HN4 and HN30 cells transfected with the Sp1 vector were determined by qPCR (E) and Western blotting (F).

G. Sp1 expression were analyzed by Western blotting in HNSCC cells transfected with LV-VN1R5 or KO-VN1R5.

H. The relative intensity of p-Sp1 expression in HNSCC cells with VN1R5 overexpression or knockout was analyzed.

I. Sp1 expression was analyzed by qPCR in HNSCC cells transfected with LV-VN1R5.

J. Sp1 expression was analyzed by qPCR in HNSCC cells transfected with KO-VN1R5.

K. Compared with NC-transfected cells (black line), HN4/DDP and HN30/DDP cells with Sp1 downregulation were sensitized to cisplatin (blue line).

L. 5’ serial deletion constructs for the promoter region of the lnc-POP1-1 gene were constructed.

M. Relative luciferase activity of different lnc-POP1-1 promoter constructs (5′ deletions) in HN4/DDP and HN30/DDP cells cotransfected with si-Sp1 or si-NC.

* $p<0.05$, ** $p<0.01$, *** $p<0.001$, **** $p<0.0001$. Error bars, means ± SDs.

(NC, negative control; si, siRNA; EV, empty vector; LV, lentiviral vector; KO, knockout; p-Sp1, phosphorylated Sp1)

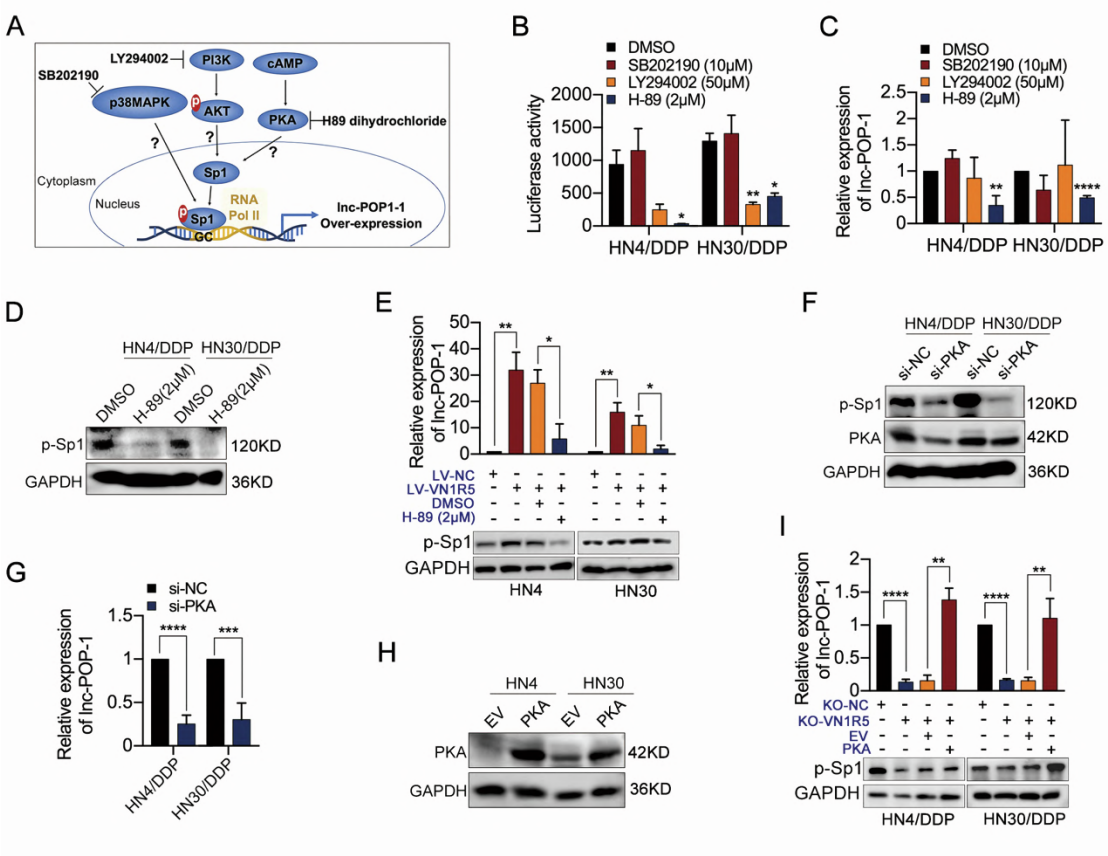

Figure S8. The promoter activity of Sp1 and the expression of lnc-POP1-1 regulated by VN1R5 were affected by the cAMP/PKA pathway.

A. Main pathways and their inhibitors that may affect the promoter activity of Sp1.

B. The luciferase activity of the lnc-POP1-1 promoter was measured in cisplatin-resistant HNSCC cells treated with inhibitors (LY294002, SB202190 and H-89 dihydrochloride).

C. lnc-POP1-1 expression was analyzed by qPCR in cisplatin-resistant HNSCC cells treated with inhibitors (LY294002, SB202190 and H-89 dihydrochloride).

D. The expression levels of phosphorylated Sp1 were analyzed by Western blotting in cisplatin-resistant HNSCC cells treated with a cAMP/PKA pathway inhibitor (H-89 dihydrochloride, 2 μM).

E. The levels of lnc-POP1-1 and phosphorylated Sp1 were measured in VN1R5-overexpressing HNSCC cells treated with H-89 dihydrochloride (2 μM).

F. The levels of phosphorylated Sp1 were measured in HN4/DDP and HN30/DDP cells treated with si-PKA by Western blotting.

G. The levels of lnc-POP1-1 were measured in HN4/DDP and HN30/DDP cells treated with si-PKA by qPCR.

H. The relative expression levels of PKA in HN4 and HN30 cells transfected with PKA-Cα vector (PKA) were determined by Western blotting.

I. The levels of lnc-POP1-1 and phosphorylated Sp1 were measured in KO-VN1R5 HNSCC cells treated with the PKA vector.

* $p<0.05$, ** $p<0.01$, *** $p<0.001$, **** $p<0.0001$. Error bars, means ± SDs.

(NC, negative control; si, siRNA; EV, empty vector; LV, lentiviral vector; KO, knockout; p-Sp1, phosphorylated Sp1; H-89, H-89 dihydrochloride; PKA, PKA-Cα)

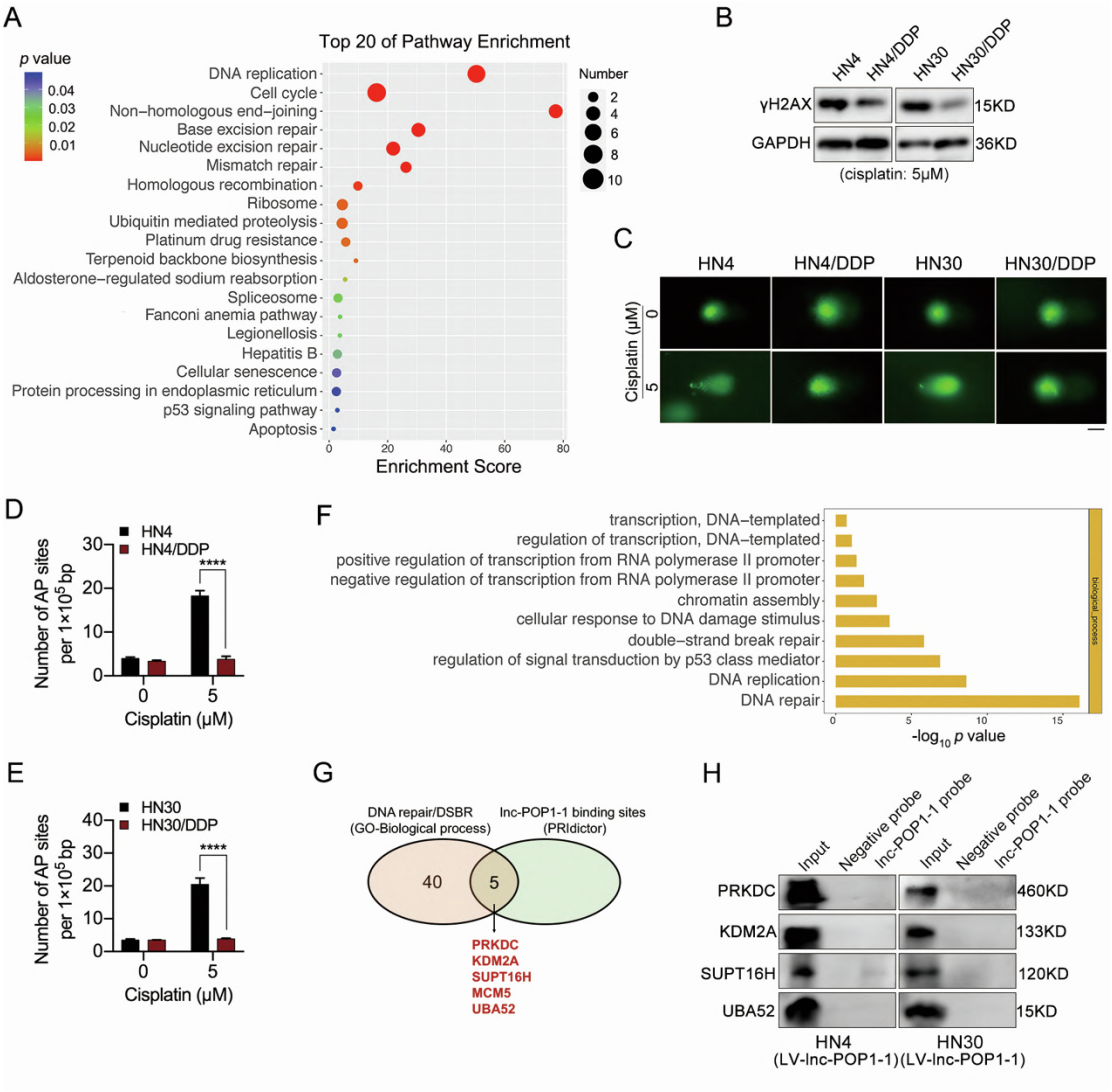

Figure S9. lnc-POP1-1 participated in DNA repair pathways.

A. Pathway analysis demonstrated that RBPs of lnc-POP1-1 were involved in several DNA repair pathways in humans.

B. γH2AX expression was measured by Western blotting in cisplatin-resistant/cisplatin-sensitive HN4 and HN30 cells.

C. Representative images of the alkaline comet assays to analyze DNA damage in cisplatin-resistant/cisplatin-sensitive HN4 and HN30 cells. Scale bar: 20 μm.

D, E. Following exposure to 0 μM or 5 μM cisplatin for 24 h, the AP sites of cisplatin-resistant/cisplatin-sensitive HN4 (D) and HN30 (E) cells were quantified using an AP site counting kit to analyze DNA damage.

F. GO analysis data including the predicted biological processes of RBPs of lnc-POP1-1.

G. According to the protein characteristics and predicted binding sites for lnc-POP1-1, 5 predicted proteins (PRKDC, MCM5, SUPT16H, KDM2A and UBA52) were selected for subsequent experimental validation.

H. RNA pull-down plus Western blot analyses showed that PRKDC, SUPT16H, KDM2A and UBA52 could not be pulled down by lnc-POP1-1 probes in HN4 and HN30 cells transfected with LV-lnc-POP1-1.

**** $p<0.0001$. Error bars, means ± SDs.

(LV, lentiviral vector)

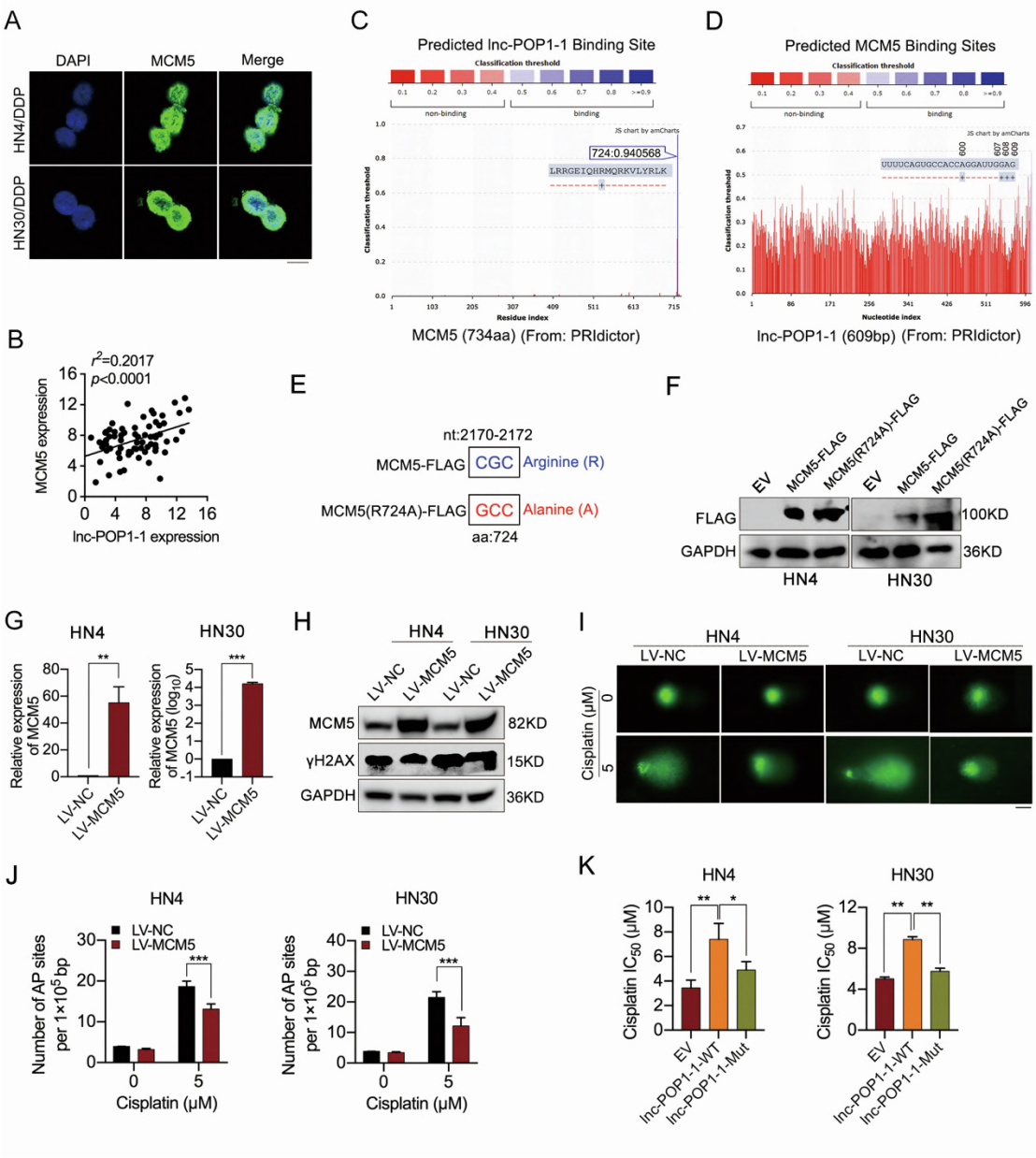

Figure S10. lnc-POP1-1 physically interacted with MCM5 and participated in DNA repair pathways.

A. IF analysis of MCM5 in HN4/DDP and HN30/DDP cells. Scale bar: 10 μm.

B. Pearson correlation analysis of the expression of lnc-POP1-1 and MCM5 in HNSCC tissue. r^2 = 0.2017, n =70.

C. The predicted lnc-POP1-1 binding site of MCM5 was obtained from the PRIdictor database (http://bclab.inha.ac.kr/pridictor).

D. The predicted MCM5-binding sites of lnc-POP1-1 were obtained from the PRIdictor database (http://bclab.inha.ac.kr/pridictor).

E. Construction of the MCM5-FLAG-Mut vector MCM5(R724)-FLAG, which mutated CGC to GCC at base pairs 2170 to 2172.

F. The relative expression of MCM5 was detected by Western blotting in HN4 and HN30 cells transfected with the MCM5-FLAG-WT and MCM5-Mut vectors.

G. The relative expression of MCM5 was detected by qPCR in HN4 and HN30 cells stably transduced with LV-MCM5.

H. The expression of MCM5 and γH2AX was detected by Western blotting in HN4 and HN30 cells stably transduced with LV-MCM5.

I. Representative images of the alkaline comet assays used to analyze DNA damage in HN4 and HN30 cells overexpressing MCM5. Scale bar: 20 μm.

J. Following exposure to 0 μM or 5 μM cisplatin for 24 h, the AP sites of HN4 and HN30 cells overexpressing MCM5 were quantified using an AP site counting kit to analyze DNA damage.

K. Compared with the WT plasmid, the lnc-POP1-1 mutant plasmid endowed HN4 and HN30 cells with sensitivity to cisplatin. The IC_{50} values are shown.

* $p<0.05$, ** $p<0.01$, *** $p<0.001$. Error bars, means ± SDs.

(LV, lentiviral vector; NC, negative control; SS, Smart Silencer; EV, empty vector; WT, wild-type; Mut, mutation)

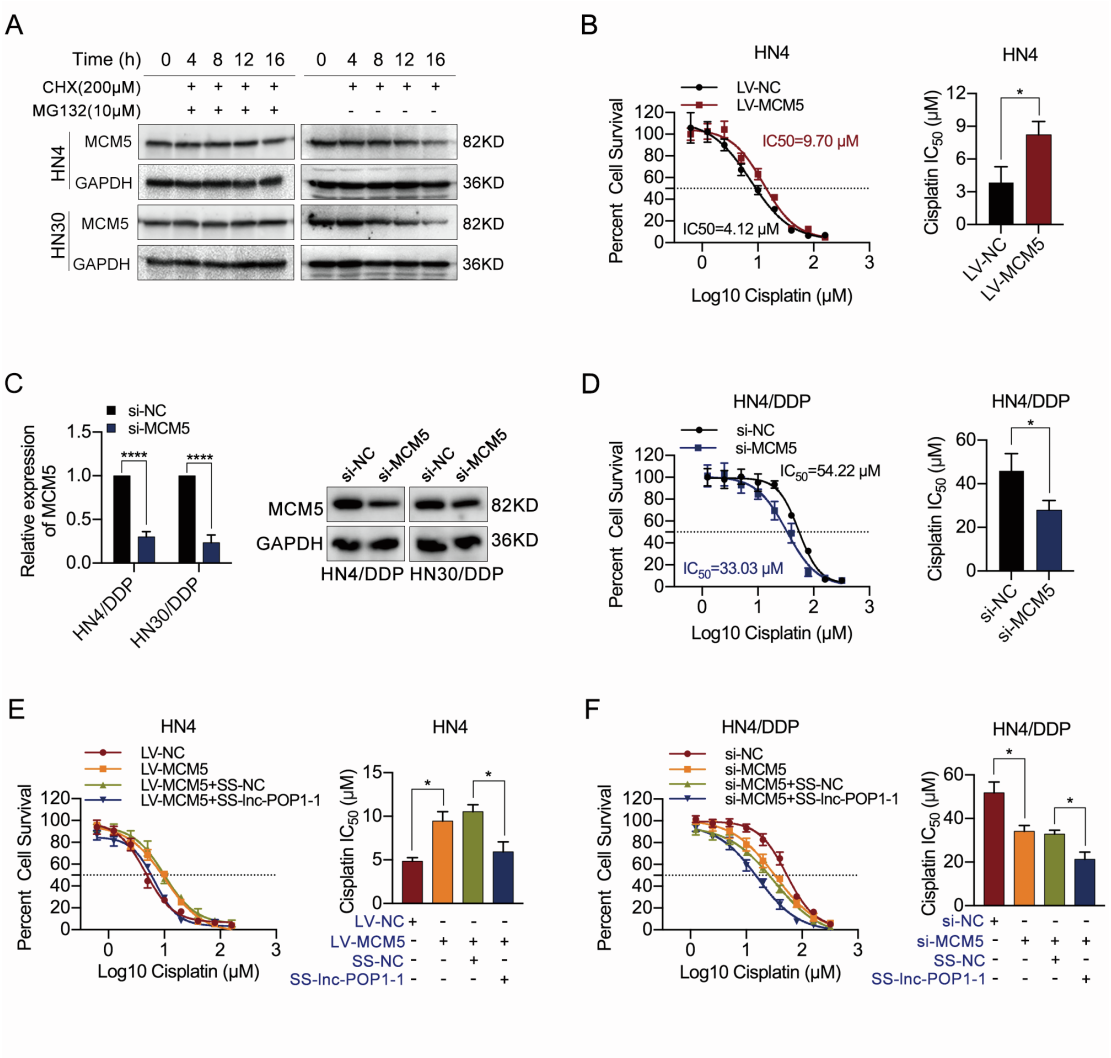

Figure S11. MCM5 affected the DNA repair and cisplatin resistance of HNSCC cells by interacting with lnc-POP1-1.

A. Proteins were isolated every 4 h from HN4 and HN30 cells treated with 10 μM or/and 200 μM CHX for 0-16 h and analyzed by Western blotting.

B. Compared with NC-transfected cells (black line), HN4 cells with MCM5 upregulation exhibited resistance to cisplatin (red line). The IC_{50} values are shown on the right.

C. The relative expression of MCM5 was detected by qPCR and Western blotting in HN4/DDP and HN30/DDP cells transfected with si-MCM5.

D. Compared with NC-transfected cells (black line), HN4/DDP cells with MCM5 downregulation were sensitized to cisplatin (blue line). The IC_{50} values are shown on the right.

E. Cell viability was detected by CCK-8 assays when lnc-POP1-1 was knocked down in HN4 cells stably transfected with LV-MCM5. The IC_{50} values are shown on the right.

F. Cell viability was detected by CCK-8 assays when lnc-POP1-1 and MCM5 were both knocked down in HN4/DDP cells. The IC_{50} values are shown on the right.

* $p<0.05$, **** $p<0.0001$. Error bars, means ± SDs.

(NC, negative control; si, siRNA; LV, lentiviral vector; SS, Smart Silencer)

Supplementary Tables

Table S1. Sequences of qPCR primers.

Primers		Sequences (5’-3’)
VN1R5	Forward	TTCAGTCACAGGTCTAAGTCCA
	Reverse	ACCAAAGAAGTCTAAGGACACCA
lnc-POP1-1	Forward	CAGAAATCATGAGAGATCATCAGTG
	Reverse	AAGGAGGTAGATTGGAATCCAGG
GAPDH	Forward	GAACGGGAAGCTCACTGG
	Reverse	GCCTGCTTCACCACCTTCT
U6	Forward	CTCGCTTCGGCAGCACATATACT
	Reverse	ATTTGCGTGTCATCCTTGCGCA
TUG1	Forward	TCCTTGTTTAGTGCATCTTTGCC
	Reverse	TGAGTGGTTATTCTGATAGCCTGC
NEAT1	Forward	GCATACGCAGCAGATCAGCAT
	Reverse	CCCACAATATAGGCATTTACAAGG
MALAT1	Forward	CCTAACCAGGCATAACACAGAAT
	Reverse	CGAATGGCTTTGTCTCCGAA
BIRC5	Forward	GCAATGTCTTAGGAAAGGAGATCA
	Reverse	AGAGAAGCAGCCACTGTTACCA
Sp1	Forward	GGCAATAACCAGTCCACACCAC
	Reverse	GCATTTACCCACACAGCCC
MCM5	Forward	TGGACTGACAGCCTCGGTGATG
	Reverse	GGATTGCCACACGGTCATCTTCTC

Table S2. Relationship between VN1R5 level and clinicopathologic features (N=83)

Characteristics	No. of Patients		VN1R5 ΔCt[a] Mean± SD	Non-parametric test value	*P* value
	No.	%			
Age (years)					
≥60	38	45.78	8.960 ± 1.557	Z=1.549	0.121
<60	45	54.22	8.411 ± 1.413		
Gender					
Male	47	56.63	8.718 ± 1.479	Z=0.505	0.613
Female	36	43.37	8.590 ± 1.538		
Smoking History					
Nonsmoker	59	71.08	8.509 ± 1.497	Z=1.527	0.127
Smoker	24	28.92	9.040 ± 1.457		
Alcohol History					
Nondrinker	53	63.86	8.670 ± 1.534	Z=0.038	0.970
Drinker	30	36.14	8.649 ± 1.454		
Tumor Size(cm)					
≤4	55	66.27	8.844 ± 1.495	Z=1.560	0.119
>4	28	33.73	8.304 ± 1.460		
Lymph Node Metastasis					
pN0	51	61.45	8.592 ± 1.472	Z=0.379	0.705
pN1 to pN3	32	38.55	8.774 ± 1.553		
TNM Stage					
I-II	41	49.40	8.500 ± 1.455	Z=0.765	0.444
III-IV	42	50.60	8.821 ± 1.537		
Pathological Differentiation					
Well	37	44.58	8.387 ± 1.465	Z=1.379	0.168
Moderatedly/poorly	46	55.42	8.885 ± 1.500		
Tumor Type					
Primary	67	80.72	8.553 ± 1.436	Z=1.160	0.246
Recurrent	16	19.28	9.119 ± 1.704		
Efficacy of TPF regimen					
Sensitive	39	46.99	9.946 ± 0.898	Z=7.309	**0.000****
Resistant	44	53.01	7.524 ± 0.860		

** $p<0.01$

Abbreviations: SD, standard deviation; pN, pathological lymph node status; TNM stage, tumor-lymph node-metastasis stage.

a ΔCt indicates the difference in the cycle number at which a sample's fluorescent signal passes a given threshold above baseline (Ct) derived from a specific gene compared with that of GAPDH in tumor tissues.

Table S3. Relationship between lnc-POP1-1 level and clinicopathologic features (N=83)

Characteristics	No. of Patients		Lnc-POP1-1 ΔCt[a] Mean± SD	Non-parametric test value	*P* value
	No.	%			
Age (years)					
≥60	38	45.78	10.586 ± 1.811	Z=0.361	0.718
<60	45	54.22	10.176 ± 1.665		
Gender					
Male	47	56.63	10.273 ± 1.655	Z=0.671	0.502
Female	36	43.37	10.482 ± 1.852		
Smoking History					
Nonsmoker	59	71.08	10.364 ± 1.729	Z=0.332	0.740
Smoker	24	28.92	10.361 ± 1.787		
Alcohol History					
Nondrinker	53	63.86	10.254 ± 1.755	Z=0.711	0.477
Drinker	30	36.14	10.558 ± 1.712		
Tumor Size(cm)					
≤4	55	66.27	10.491 ± 1.799	Z=0.732	0.464
>4	28	33.73	10.113 ± 1.604		
Lymph Node Metastasis					
pN0	51	61.45	10.313 ± 1.846	Z=0.281	0.779
pN1 to pN3	32	38.55	10.444 ± 1.568		
TNM Stage					
I-II	41	49.40	10.175 ± 1.877	Z=1.070	0.284
III-IV	42	50.60	10.548 ± 1.585		
Pathological Differentiation					
Well	37	44.58	10.217 ± 1.904	Z=0.912	0.362
Moderatedly/poorly	46	55.42	10.481 ± 1.598		
Tumor Type					
Primary	67	80.72	10.407 ± 1.779	Z=0.600	0.548
Recurrent	16	19.28	10.183 ± 1.579		
Efficacy of TPF regimen					
Sensitive	39	46.99	11.915 ± 0.860	Z=7.287	**0.000****
Resistant	44	53.01	8.988 ± 0.983		

** $p<0.01$

Abbreviations: SD, standard deviation; pN, pathological lymph node status; TNM stage, tumor-lymph node-metastasis stage.

a ΔCt indicates the difference in the cycle number at which a sample's fluorescent signal passes a given threshold above baseline (Ct) derived from a specific gene compared with that of GAPDH in tumor tissues.

Table S4. The sequences of lnc-POP1-1 Smart Silencer.

Product Name	Sequences (5’-3’)
lnc-POP1-1 Smart Silencer	GCCATGAGTGAGCCACCTT
	CTACAGAAATCATGAGAGA
	AGTTCCCTCAAGTGTGAAA
	CAACTGACATCCAACTACAA
	AGCCAAGCTGTCCCTGAATT
	AATCTACCTCCTTCACTGAC

Table S5. The sequences of ASO and siRNA.

Product Name	Target Sequences (5’-3’)
ASO-lnc-POP1-1 (1)*	CAACTGACATCCAACTACAA
ASO-lnc-POP1-1 (2)	AGCCAAGCTGTCCCTGAATT
ASO-lnc-POP1-1 (3)	AATCTACCTCCTTCACTGAC
si-Sp1	CTCCCAACTTACAGAACCA
si-NF-κB	AAAAAAAAGGGACTTTCATTGTACTGGT
si-Fos	GGGATAGCCTCTCTTACTA
si-JUN	CCAACATGCTCAGGGAACA
si-TBP	CCTAAAGACCATTGCACTT
si-PKA	ATGTTTGAAAGGATAGTCAAAGC
si-MCM5	GCATCTACTCCATCAAGAA

* The sequence of ASO-lnc-POP1-1 cholesterol-conjugated for *in vivo* ASO delivery.

Table S6. VN1R5-CRISPR/cas9-sgRNA target sequences.

NO.	TargetSeq
sgRNA 1	TCTAAGATGATCAAACTTCC
sgRNA 2	GTGACTAATTATCATGTCAA
sgRNA 3	GCAGTATGTGGATGAGAGAC

Table S7. Sequences of lnc-POP1-1 promoter ChIP primers.

No.		Sequences (5'-3')
1	Forward	TCTCCTCTTTGATTTCCTCTGCTGC
	Reverse	CACAGTTGGGGTGCAAGGG
2	Forward	TATGCCATACAGAATCAATTTTGGGTG
	Reverse	GTGAAATGATATAGCAGTGAGAGTGAG
3	Forward	CCAGGCTGGAGTGAAGTGG
	Reverse	GATCACCTGAAGTCAGGAGCTCA

Table S8. Sequences of RIP primers for lnc-POP1-1.

No.		Sequences (5'-3')
P1	Forward	CTGGCCTGTGTGATGAATAGAATATGATGG
	Reverse	GCTGGCTTCCTGCAGAGC
P2	Forward	GGAAGCCAGCTGCCATCC
	Reverse	CTCCCAAGGTGGCTCACTCAT
P3	Forward	ACCTTGGGAGGGGAACCTC
	Reverse	CAGGGACAGCTTGGCTGG
P4	Forward	CTGTCCCTGAATTCCTGACCTACAGA
	Reverse	CCATGAGCTCCCACCTGCG
P5	Forward	AGCTCATGGCGTGGGAAG
	Reverse	GGAGACTAATCAGCTTTGCCTTGG
P6	Forward	CTGATTAGTCTCCCTGAGCCTCAG
	Reverse	CTCCAATCCTGGTGGCACTGA