ABclonal Technology®

Bio Genomics Asia
LOCAL EXPERT, GLOBAL SOLUTIONS

ABCLONAL NGS PRODUCTS AND SELECTED LITERATURE

Selected References of ABclonal NGS-Related Products

No.	Title	Journal	Year	Product
1	Epstein-Barr virus hijacks histone demethylase machinery to drive epithelial malignancy progression through KDM5B upregulation	Signal transduction and targeted therapy (IF 52.7)	2025	• Sonication ChIP Kit (RK20258)
2	Family with sequence similarity 114 member A1 orchestrates immune evasion in triple-negative breast cancer	Signal transduction and targeted therapy (IF 52.7)	2025	• Sonication ChIP Kit (RK20258)
3	Composite transposons with bivalent histone marks function as RNA-dependent enhancers in cell fate regulation	Cell (IF 42.5)	2025	• Rapid Plus DNA Lib Prep Kit for Illumina (RK20208)**(replaced by RK20271)** • Poly(A) mRNA Capture Module (RK20340)
4	Phase 1 multicenter, dose-expansion study of ARX788 as monotherapy in HER2-positive advanced gastric and gastroesophageal junction adenocarcinoma	Cell Rep Med (IF 10.6)	2022	• Rapid Plus DNA Lib Prep Kit for Illumina (RK20208)**(replaced by RK20271)**
5	Transcriptional repression of estrogen receptor alpha by YAP reveals the Hippo pathway as therapeutic target for ER + breast cancer	Nat Commun (IF 15.7)	2022	• Rapid DNA Lib Prep Kit for Illumina (RK20200)**(replaced by RK20271)**
6	ONE-seq: epitranscriptome and gene-specific profiling of NAD-capped RNA	Nucleic acids research (IF 16.7)	2023	• mRNA-seq Lib Prep Module for Illumina(RK20350) • Poly(A) mRNA Capture Module (RK20340)
7	WDR36 Regulates Trophectoderm Differentiation During Human Preimplantation Embryonic Development Through Glycolytic Metabolism	Advanced science (IF 14.1)	2025	• Single Cell/Low Input cDNA Synthesis & Amplification Module (RK20310)
8	Nucleolar MED19 regulates 2 -O-methylation of rRNA in supporting cancer cell growth	Nucleic acids research (IF 13.1)	2025	• Small RNA Lib Prep Kit for Illumina V3 (RK20312)
9	ZAR1 and ZAR2 orchestrate the dynamics of maternal mRNA polyadenylation during mouse oocyte development	Genome biology (IF 14.8)	2025	• rRNA Depletion module (H/M/R) (RK20348) • AFTMag NGS DNA Clean Beads (RK20257)
10	Comparative transcriptomics reveals a mixed basal, club, and hillock epithelial cell identity in castration-resistant prostate cancer	Proc Natl Acad Sci U S A (IF 9.1)	2025	• mRNA-seq Lib Prep Module for Illumina (RK20350) • Poly(A) mRNA Capture Module (RK20340)
11	Whole-Genome Sequencing of 54 Dengchuan Cattle (Bos taurus) from Southwest China	Scientific data (IF 6.9)	2025	• FS Pro DNA Lib Prep Kit V2 (RK20275)**(replaced by RK29832)**
12	Long-range interaction within the chromatin domain determines regulatory patterns in porcine skeletal muscle	Genomics (IF 3)	2022	• FS Pro DNA Lib Prep Kit for Illumina (RK20261)**(replaced by RK29832)** • T4 DNA Ligase (RK21501)

ABclonal NGS-Related Product Catalogue

DNA Lib Prep Kit

Categories	Product No.	Product Name	Specifications
Mechanical Fragmentation -based DNA Library Prep	RK20271	Rapid Plus DNA Lib Prep Kit V2 (No DDREs)	24 RXN/96 RXN
	RK20255	Rapid Plus DNA Lib Prep Kit for Illumina V2	24 RXN/96 RXN
Enzymatic Fragmentation -based DNA Library Prep	RK29832 (replaces RK20275)	FS Pro DNA Lib Prep Kit V2	24 RXN/96 RXN

RNA Lib Prep Kit

Categories	Product No.	Product Name	Specifications
RNA enrichment Module	RK20340	Poly(A) mRNA Capture Module	24 RXN/96 RXN
	RK20348	rRNA Depletion module (H/M/R)	24 RXN/96 RXN
	RK30209	rRNA Depletion Kits (Human rRNA & Globin)	24 RXN/96 RXN
	RK30207	rRNA Depletion Kits (Pan-Prokaryote)	24 RXN/96 RXN
	RK30208	rRNA Depletion Kits (Plant)	24 RXN/96 RXN
mRNA-seq Lib Prep	RK20350	mRNA-seq Lib Prep Module for Illumina	24 RXN/96 RXN
Small RNA Lib Prep	RK20312	Small RNA Lib Prep Kit for Illumina V3	24 RXN/96 RXN
Single Cell/Low Input RNA Lib Prep	RK20310	Single Cell/Low Input cDNA Synthesis & Amplification Module	24 RXN/96 RXN

Epigenetic-Related products

Categories	Product No.	Product Name	Specifications
ChIP Kit	RK20258	Sonication ChIP Kit	8 RXN/24 RXN
DNA Methylation	RK20723	Gloria U 2X Mix for NGS	1 mL/5 mL
	RK20722	Gloria U 2X HS Master Mix	1 mL/5 mL

NGS-Related Enzymes

Categories	Product No.	Product Name	Specifications
End Repair & A-Tailing	RK20530	DNA Polymerase I (E. coli)	500 U/2,500 U
	RK20525	DNA Polymerase I, Large (Klenow) Fragment	200 U/1,000 U/5,000 U
	RK20526	Klenow Fragment 3’→5’ exo-	200 U/1,000 U/5,000 U
	RK20539	T4 DNA Polymerase (5,000 U/mL)	200 U/1,000 U
	RK20524	T4 Polynucleotide Kinase	250 U/2,500 U
DNA/RNA Ligases	RK21500	T4 DNA Ligase (High Conc.) 2000 U/μL	80,000 U/400,000 U
	RK21501	E. coli DNA Ligase	200 U/1,000 U
	RK21502	Taq DNA Ligase	2,000 U/10,000 U
	RK21503	T4 RNA Ligase 1	1,000 U/5,000 U
	RK20516	T4 RNA Ligase 2	150 U/750 U
	RK20505	T4 RNA Ligase 2, truncated	1,000 U/10,000 U
	RK20504	T4 RNA Ligase 2, truncated KQ	2,000 U/10,000 U
	RK20515	Mth RNA Ligase	10 RXN/50 RXN
	RK20508	Circle Ligase	1,000 U/5,000 U
Reverse Transcriptase	RK20414	ABScript Full Length Reverse Transcriptase for Single Cell	5,000 U/20,000 U
	RK20408	ABScript III Reverse Transcriptase	4,000 U/10,000 U
	RK21400	ABScript II Reverse Transcriptase	4,000 U/10,000 U
	RK21401	RNase Inhibitor, Mammalian	2,000 U/10,000 U
Nuclease	RK20549	DNase I , RNase-free (5,000 U/mL)	1,000 U/5,000 U
	RK20535	RNase H	250 U/1,250 U
	RK20527	Uracil-DNA Glycosylase (UDG)	1,000 U/5,000 U
	RK20531	Exonuclease I (E. coli)	1,500 U/15,000 U
	RK20533	Exonuclease III (E. coli)	5,000 U/25,000 U
	RK20563	Exonuclease VII	200 U/1,000 U
	RK20529	Lambda Exonuclease	1,000 U/5,000 U
	RK20534	Endonuclease VIII	1,000 U/5,000 U
	RK20580	5' Deadenylase	2,500 U

NGS Adapter

Categories	Product No.	Product Name	Specifications
UDI Truncated Adapters	RK21624 ~ RK21627	Unique Dual Index for Illumina Set_A/B/C/ D (48 indices)	5 μL_each
UDI (with UMI) Truncated Adapters	RK21702/RK21703	Unique Dual Index (with UMI) for Illumina Set_A /B (48 indices)	5 μL_each

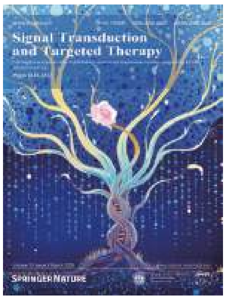
Other NGS-Related Products

Categories	Product No.	Product Name	Specifications
NGS Cleanup & Size Selection Beads	RK20257	AFTMag NGS DNA Clean Beads	5 mL/60 mL
Library Amplification	RK20726	TruePol 2X PCR Mix for NGS	24 RXN/96 RXN

Article | Open access | Published: 10 March 2025

Epstein-Barr virus hijacks histone demethylase machinery to drive epithelial malignancy progression through KDM5B upregulation

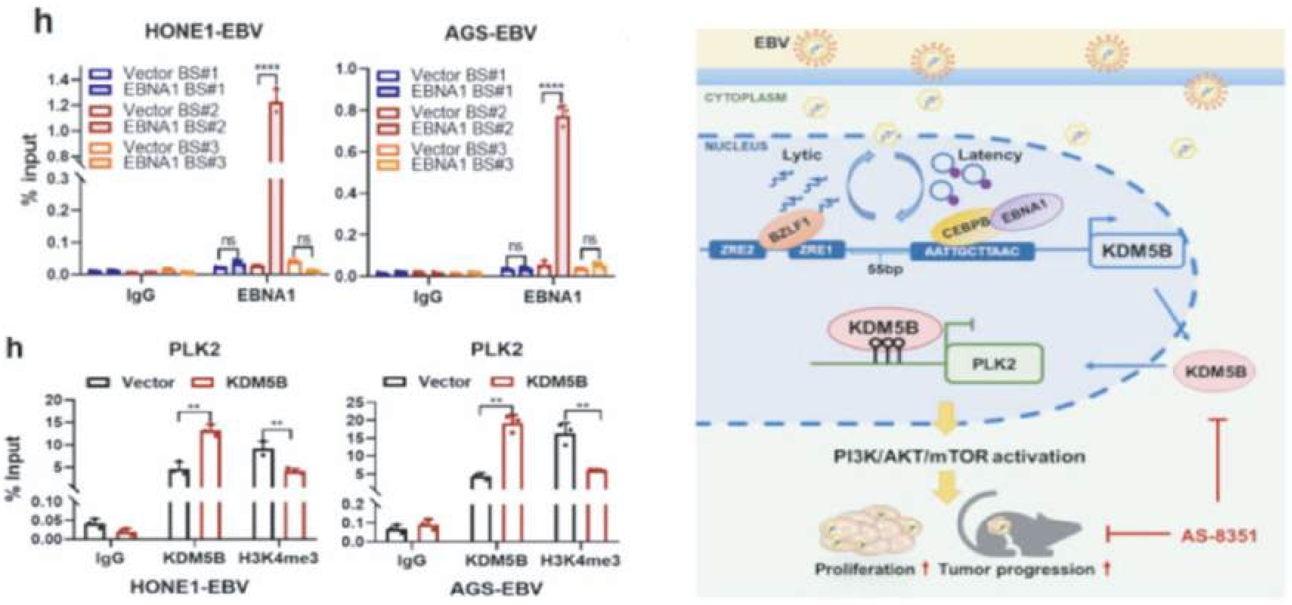
Ya-Qing Zhou, Jia-Xin Jiang, Shuai He, Yi-Qi Li, Xi-Xi Cheng, Shu-Qiang Liu, Pan-Pan Wei, Xin-Yuan Guan, Choon Kiat Ong, Vivien Ya-Fan Wang, Chun-Ling Luo & Jin-Xin Bei



Journal	Signal transduction and targeted therapy
IF	52.7
Year	2025
Relative Products	**Sonication ChIP Kit (RK20258)**
Application	**ChIP-qPCR**
Affiliations	Sun Yat-sen University Cancer Center

Abstract: Epstein-Barr virus (EBV) is a significant epigenetic driver in the development of epithelial-origin nasopharyngeal carcinoma (NPC) and gastric cancer (GC), which together represent 80% of EBV-associated malignancies. Despite its known association, the specific mechanisms, particularly those involving EBV-induced histone modifications, remain poorly understood. Through integrative analyses of single-cell and bulk transcriptome data from epithelial tumor tissues and EBV-infected cells, we identified KDM5B as a critical histone-modifying factor consistently upregulated following EBV infection. We demonstrated that EBV stimulates KDM5B expression via interactions of its latent gene EBNA1 with transcription factor CEBPB and through direct binding of its lytic gene BZLF1 to Zta-response elements on the KDM5B promoter. Functional assays revealed that KDM5B acts as an oncogene, correlating with poor survival outcomes in EBV-associated epithelial cancers. Mechanistically, KDM5B inhibited the tumor suppressor gene PLK2 through histone demethylation, thereby activating the PI3K/AKT/mTOR signaling pathway and promoting malignant progression. Furthermore, treatment with the KDM5B inhibitor AS-8351 markedly attenuated this signaling activity and exhibited strong anti-tumor effect in both in vitro and in vivo patient-derived xenograft models from EBV-associated tumors. Together, these findings provide novel insights into how EBV hijacks KDM5B to mediate histone demethylation of PLK2, facilitating tumor progression through the PI3K/AKT/mTOR pathway in epithelial cancers, highlighting promising therapeutic strategies targeting epigenetic alterations in EBV-associated cancers.

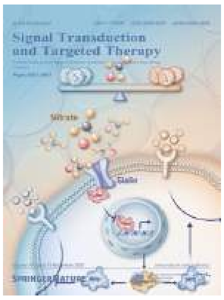
Related Figure:



The ChIP-related experiments in the article were performed using the ChIP kit (RK20258) from ABclonal.

Article | Open access | Published: 18 November 2025

Family with sequence similarity 114 member A1 orchestrates immune evasion in triple-negative breast cancer

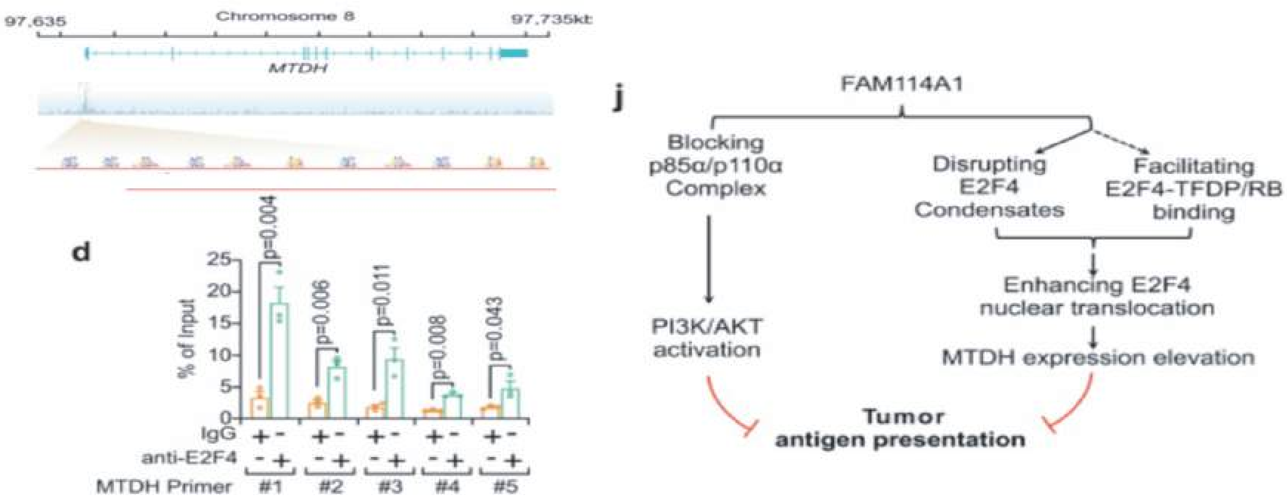


Wenhao Zhang, Yanzhi Gai, Mengxue Qiao, Michelle Rowicki, Yong Wei, Xiang Hang, Zhengkai Wei, He Yang, Xifu Ye, Hang Ju, Yi Lu ✉, Yibin Kang ✉ & Minhong Shen ✉

Journal	Signal transduction and targeted therapy
IF	52.7
Year	2025
Relative Products	**Sonication ChIP Kit (RK20258)**
Application	**ChIP-qPCR**
Affiliations	Fudan University； Tongji University； Princeton University

Abstract: Immune checkpoint blockade (ICB) therapy, which has revolutionized cancer treatment, has been approved for the treatment of triple-negative breast cancer (TNBC). Unfortunately, most patients with TNBC are either not eligible for treatment or exhibit resistance, resulting in limited overall survival benefits. There is an urgent need to elucidate the mechanisms of resistance and enhance therapeutic efficacy. Here, via CRISPR activation (CRISPRa) screening, we identified family with sequence similarity 114 member A1 (FAM114A1) as a key mediator of immune evasion and ICB resistance in TNBC. Mechanistically, FAM114A1 binds p85α to disrupt the p85α/p110α protein complex, thus activating the PI3K/AKT pathway and simultaneously preventing condensate formation of E2F Transcription Factor 4 (E2F4) to promote E2F4-driven Metadherin (MTDH) transcription. Upregulation of these FAM114A1-mediated pathways suppresses tumor antigen presentation and consequently attenuates antitumor immunity in TNBC. Moreover, targeting FAM114A1 improves the therapeutic effectiveness of anti-PD-1 therapy in mouse models, and a FAM114A1-based signature shows strong predictive performance for identifying patients with TNBC who may benefit from ICB. Collectively, our findings not only reveal that FAM114A1 is an immune evasion driver but also highlight it as a promising biomarker and therapeutic target. Our study provides new insights into TNBC immune evasion and outlines a potential avenue to improve the effectiveness of ICB.

Related Figure：



The ChIP-related experiments in the article were performed using the ChIP kit (RK20258) from ABclonal.

> Cell. 2025 Oct 16;188(21):5878-5894.e18. doi: 10.1016/j.cell.2025.07.014. Epub 2025 Aug 6.

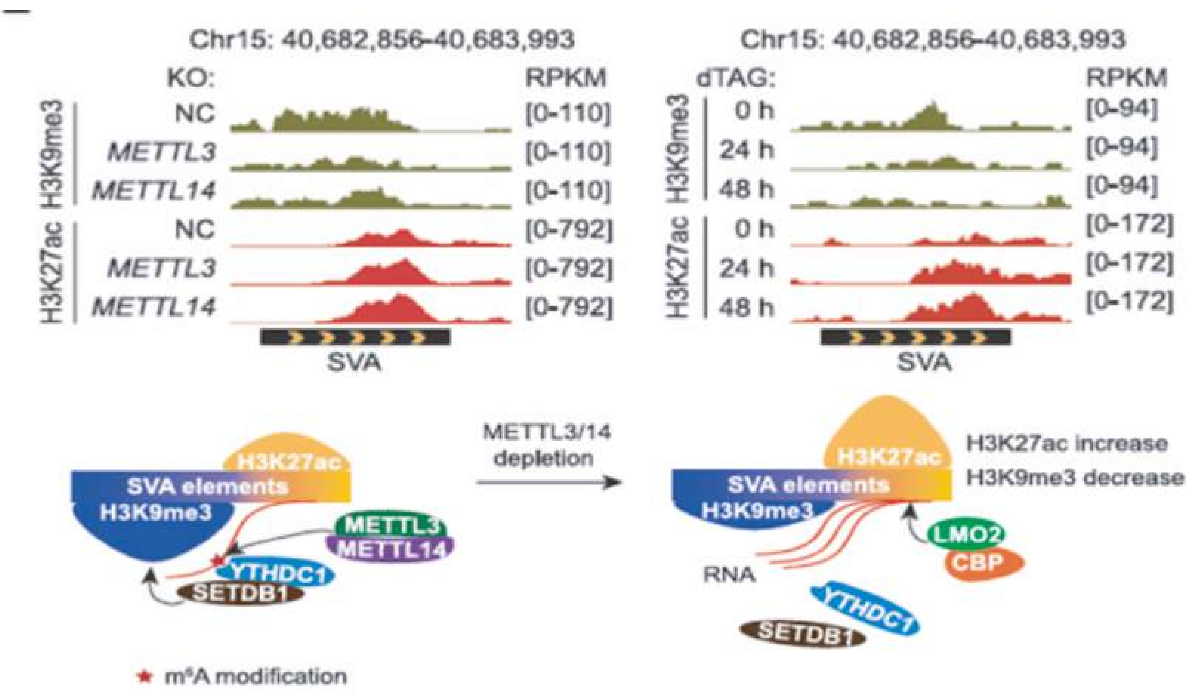
Composite transposons with bivalent histone marks function as RNA-dependent enhancers in cell fate regulation

Ziqiang Zhou [1], Shicong Zhu [2], Yaqiang Hong [1], Guangpu Jin [1], Rui Ma [3], Fan Lin [3], Yiyang Zhang [2], Hsiang-Ying Lee [4], Nian Liu [5]

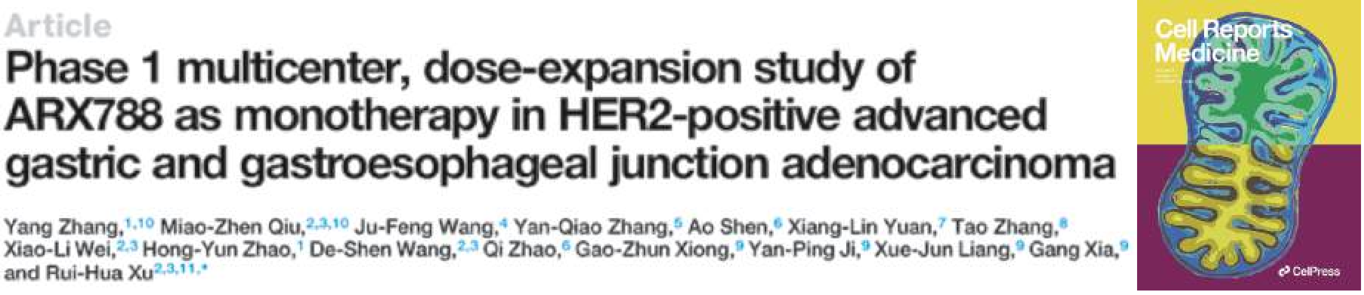
Journal	Cell
IF	42.5
Year	2025
Relative Products	**Rapid Plus DNA Lib Prep Kit for Illumina (RK20208)(replaced by RK20271); Poly(A) mRNA Capture Module (RK20340)**
Application	**ChIP-seq**，**RNA-seq**
Affiliations	FTsinghua University

Abstract: Discrete genomic units can recombine into composite transposons that transcribe and transpose as single units, but their regulation and function are not fully understood. We report that composite transposons harbor bivalent histone marks, with activating and repressive marks in distinct regions. Genome-wide CRISPR-Cas9 screening, using a reporter driven by the hominid-specific composite transposon SVA (SINE [short interspersed nuclear element]-VNTR [variable number of tandem repeats]-Alu) in human cells, identified diverse genes that modify bivalent histone marks to regulate SVA transcription. SVA transcripts are critical for SVA's cis-regulatory function in selectively contacting and activating long-range gene expression. Remarkably, a subset of bivalent SVAs is activated during erythropoiesis to boost multiple erythroid gene expression, and knocking down these SVAs leads to deficient erythropoiesis. The RNA-dependent cis-regulatory function of SVA activates genes for myelopoiesis and can contribute to aging-associated myeloid-biased hematopoiesis. These results reveal that the cis-regulatory functions of composite transposons are bivalently regulated to control cell fate transitions in development and aging.

Related Figure:



The DNA library construction kit used in the ChIP-seq in this article is from ABclonal's RK20208.



Article

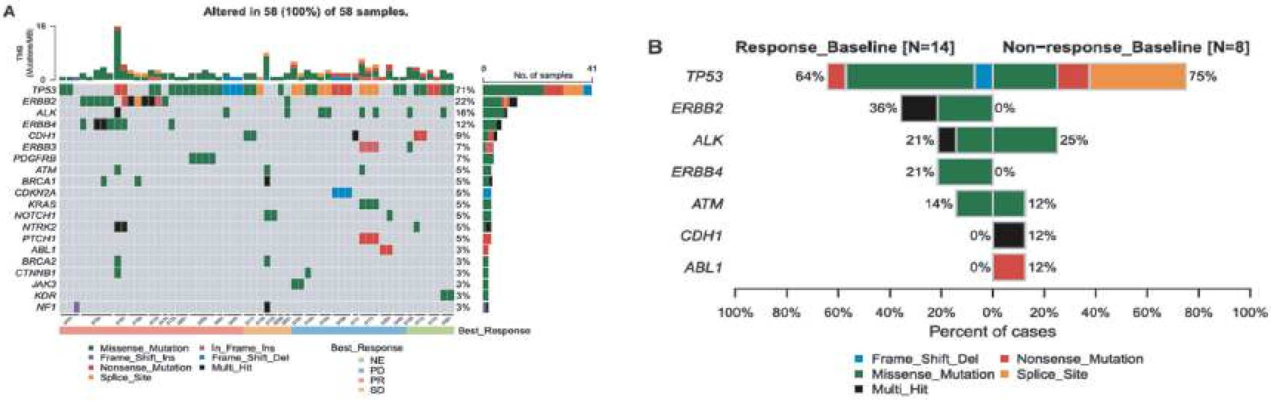
Phase 1 multicenter, dose-expansion study of ARX788 as monotherapy in HER2-positive advanced gastric and gastroesophageal junction adenocarcinoma

Yang Zhang,[1,10] Miao-Zhen Qiu,[2,3,10] Ju-Feng Wang,[4] Yan-Qiao Zhang,[5] Ao Shen,[6] Xiang-Lin Yuan,[7] Tao Zhang,[8] Xiao-Li Wei,[2,3] Hong-Yun Zhao,[1] De-Shen Wang,[2,3] Qi Zhao,[6] Gao-Zhun Xiong,[9] Yan-Ping Ji,[9] Xue-Jun Liang,[9] Gang Xia,[9] and Rui-Hua Xu[2,3,11,*]

Journal	Cell Rep Med
IF	10.6
Year	2022
Relative Products	**Rapid Plus DNA Lib Prep Kit for Illumina (RK20208)(replaced by RK20271)**
Application	**Hybridization capture sequencing**
Affiliations	Sun Yat-sen University

Abstract: ARX788 is an anti-human epidermal growth factor receptor 2 (HER2) antibody-drug conjugate with AS269 as cytotoxic payload. In this phase 1 multicenter dose-expansion clinical trial, patients with HER2-positive advanced gastric/gastroesophageal junction adenocarcinoma failing to respond to prior trastuzumab-based standard treatment were enrolled. Between July 15th, 2019, and March 14th, 2022, 30 participants were enrolled. Twenty-eight (93.3%) patients experienced at least one drug-related adverse event (AE) and 13.3% experienced grade 3 ARX788-related AEs. The confirmed objective response rate is 37.9% (95% confidence interval [CI]: 20.7%-57.7%) and the disease control rate is 55.2% (95% CI: 35.7%-73.6%). With a median follow up of 10 months, the median progression-free survival and overall survival are 4.1 (95% CI: 1.4-6.4) and 10.7 months (95% CI: 4.8-not reached), respectively. The median duration of response is 8.4 (95% CI: 2.1-18.9) months. ARX788 is well tolerated and has promising anti-tumor activity in patients with HER2-positive advanced gastric adenocarcinoma (ChinaDrugTrials.org.cn: CTR20190639).

Related Figure:



The DNA library construction kit used in the targeted sequencing in this article is from ABclonal's RK20208.

ARTICLE

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https://doi.org/10.1038/s41467-022-28691-0 OPEN

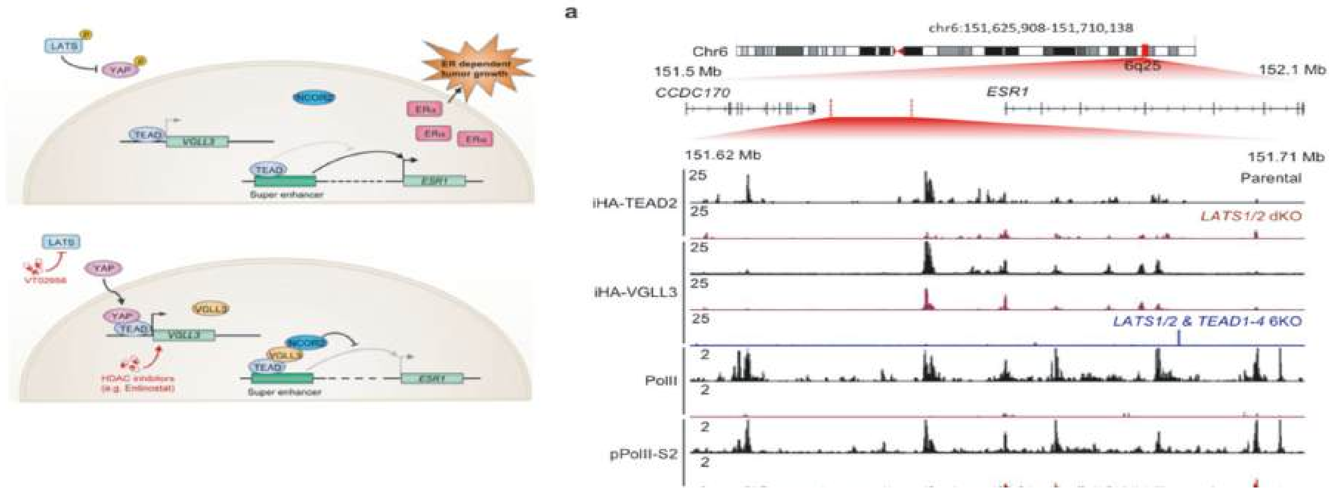
Transcriptional repression of estrogen receptor alpha by YAP reveals the Hippo pathway as therapeutic target for ER$^+$ breast cancer

Shenghong Ma[1], Tracy Tang[2], Gary Probst[2], Andrei Konradi[2], Chunyu Jin[3], Fulong Li[1], J. Silvio Gutkind[1],
Xiang-Dong Fu[4] & Kun-Liang Guan[1]✉

Journal	Nat Commun
IF	15.7
Year	2022
Relative Products	**Rapid DNA Lib Prep Kit for Illumina (RK20200) (replaced by RK20271)**
Application	**ChIP-seq**
Affiliations	University of California San Diego

Abstract: Extensive knowledge has been gained on the transcription network controlled by ERα, however, the mechanism underlying ESR1 (encoding ERα) expression is less understood. We recently discovered that the Hippo pathway is required for the proper expression of ESR1. YAP/TAZ are transcription coactivators that are phosphorylated and inhibited by the Hippo pathway kinase LATS. Here we delineated the molecular mechanisms underlying ESR1 transcription repression by the Hippo pathway. Mechanistically, YAP binds to TEAD to increase local chromatin accessibility to stimulate transcription of nearby genes. Among the YAP target genes, Vestigial-Like Protein 3 (VGLL3) competes with YAP/TAZ for binding to TEAD transcription factor and recruits the NCOR2/SMRT repressor to the super-enhancer of ESR1 gene, leading to epigenetic alteration and transcriptional silencing. We developed a potent LATS inhibitor VT02956. Targeting the Hippo pathway by VT02956 represses ESR1 expression and inhibits the growth of ER+ breast cancer cells as well as patient-derived tumour organoids. Moreover, histone deacetylase inhibitors, such as Entinostat, induce VGLL3 expression to inhibit ER+ breast cancer cells. Our study suggests LATS as unexpected cancer therapeutic targets, especially for endocrine-resistant breast cancers.

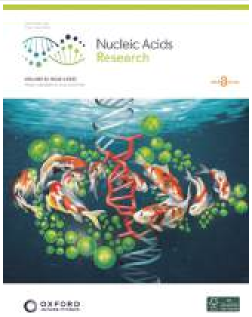
Related Figure:



The DNA library construction kit used in the ChIP-seq in this article is from ABclonal's RK20208.

ONE-seq: epitranscriptome and gene-specific profiling of NAD-capped RNA

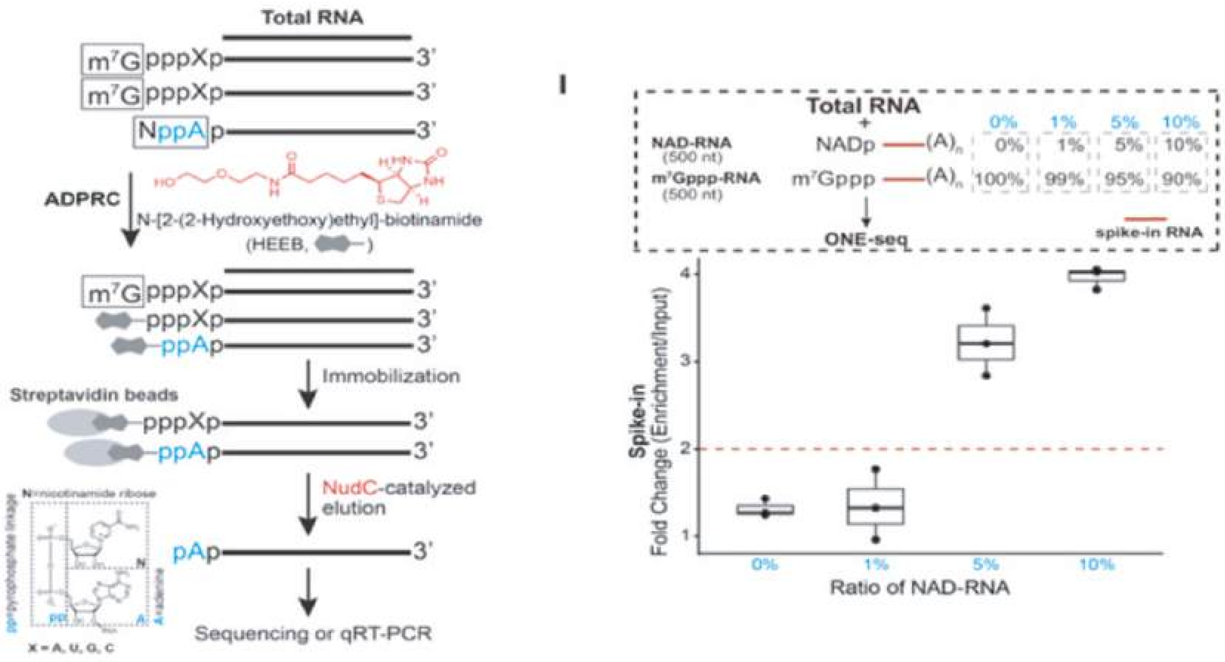
Kongyan Niu[1,2,†], Jinyang Zhang[1,2,†], Shuwen Ge[1,2,†], Dean Li[1,2,†], Kunfeng Sun[1,2], Yingnan You[1,2], Jiaqian Qiu[1,2], Kun Wang[1,2], Xueting Wang[1,2], Rui Liu[3], Yandong Liu[4], Bing Li[5], Zheng-Jiang Zhu[1,6], Lefeng Qu[4,*], Hong Jiang[1,*] and Nan Liu[1,6,*]



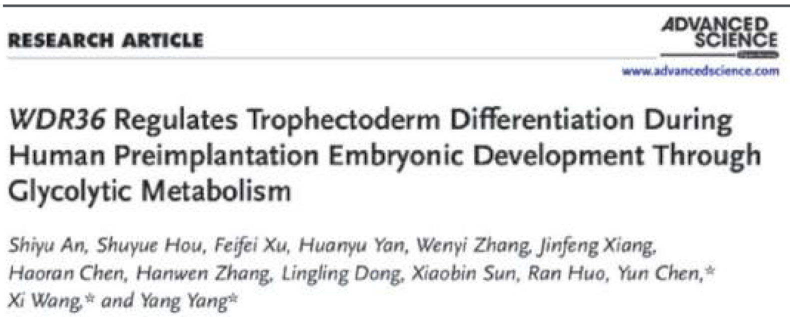
Journal	Nucleic Acids Research
IF	13.1
Year	2023
Relative Products	**mRNA-seq Lib Prep Module for Illumina (RK20350); Poly(A) mRNA Capture Module (RK20340)**
Application	**One-seq (RNA-seq)**
Affiliations	Shanghai Institute of Organic Chemistry, Chinese Academy of Sciences

Abstract: The hub metabolite, nicotinamide adenine dinucleotide (NAD), can be used as an initiating nucleotide in RNA synthesis to result in NAD-capped RNAs (NAD-RNA). Since NAD has been heightened as one of the most essential modulators in aging and various age-related diseases, its attachment to RNA might indicate a yet-to-be discovered mechanism that impacts adult life-course. However, the unknown identity of NAD-linked RNAs in adult and aging tissues has hindered functional studies. Here, we introduce ONE-seq method to identify the RNA transcripts that contain NAD cap. ONE-seq has been optimized to use only one-step chemo-enzymatic biotinylation, followed by streptavidin capture and the nudix phosphohydrolase NudC-catalyzed elution, to specifically recover NAD-capped RNAs for epitranscriptome and gene-specific analyses. Using ONE-seq, we discover more than a thousand of previously unknown NAD-RNAs in the mouse liver and reveal epitranscriptome-wide dynamics of NAD-RNAs with age. ONE-seq empowers the identification of NAD-capped RNAs that are responsive to distinct physiological states, facilitating functional investigation into this modification.

Related Figure:



The RNA library construction part of One-seq in this article uses the RNA library construction kit RK20350 from ABclonal.


RESEARCH ARTICLE

ADVANCED SCIENCE

www.advancedscience.com

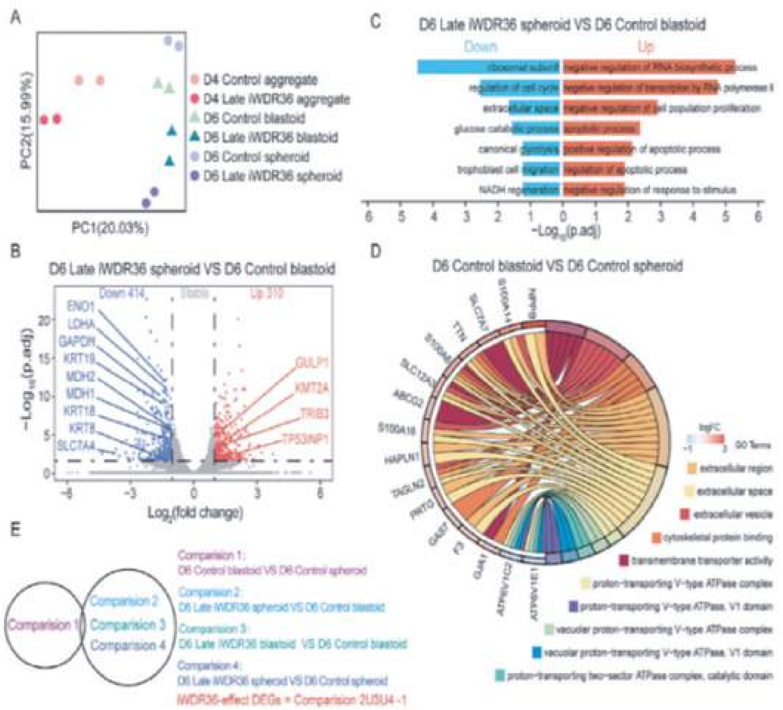
WDR36 Regulates Trophectoderm Differentiation During Human Preimplantation Embryonic Development Through Glycolytic Metabolism

Shiyu An, Shuyue Hou, Feifei Xu, Huanyu Yan, Wenyi Zhang, Jinfeng Xiang, Haoran Chen, Hanwen Zhang, Lingling Dong, Xiaobin Sun, Ran Huo, Yun Chen, Xi Wang,* and Yang Yang**

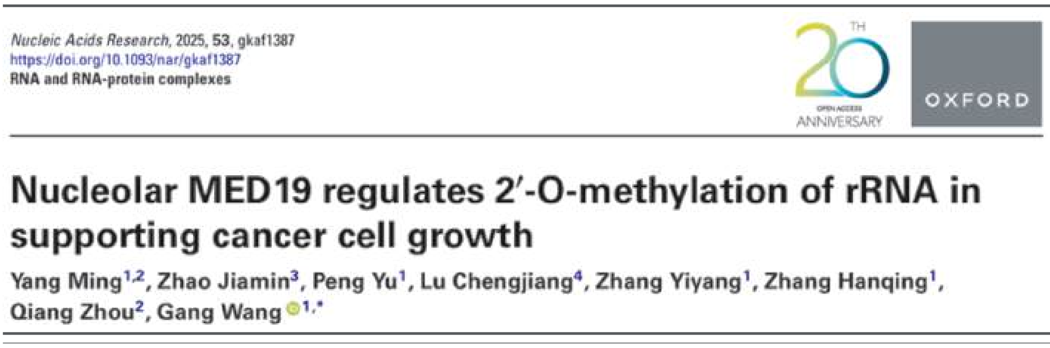
Journal	Adv Sci (Weinh)
IF	14.1
Year	2025
Relative Products	**Single Cell/Low Input cDNA Synthesis & Amplification Module (RK20310)**
Application	**RNA-seq (low input)**
Affiliations	Nanjing Medical University

Abstract: Mammalian pre-implantation development is a complex process involving sophisticated regulatory dynamics. WD repeat domain 36 (WDR36) is known to play a critical role in mouse early embryonic development, but its regulatory function in human embryogenesis is still elusive due to limited access to human embryos. The human pluripotent stem cell-derived blastocyst-like structure, termed a blastoid, offers an alternative means to study human development in a dish. In this study, after verifying that WDR36 inhibition disrupted polarization in mouse early embryos, it is further demonstrated that WDR36 interference can block human blastoid formation, dominantly hindering the trophectoderm lineage commitment. Both transcriptomics and targeted metabolomics analyses revealed that WDR36 interference downregulated glucose metabolism. WDR36 can interact with glycolytic metabolic protein lactate dehydrogenase A (LDHA), thereby positively regulating glycolysis during the late stage of human blastoid formation. Taken together, the study has established a mechanistic connection between WDR36, glucose metabolism, and cell fate determination during early embryonic lineage commitment, which may provide potential insights into novel therapeutic targets for early adverse pregnancy interventions.

Related Figure:



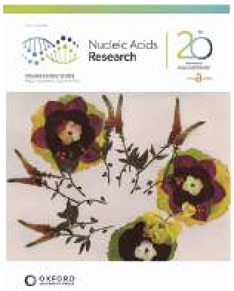
The RNA library construction kit used for the transcriptome sequencing of mouse early embryos (low input sample) in this article is RK20312 from ABclonal.



Nucleic Acids Research, 2025, **53**, gkaf1387
https://doi.org/10.1093/nar/gkaf1387
RNA and RNA-protein complexes

Nucleolar MED19 regulates 2′-O-methylation of rRNA in supporting cancer cell growth

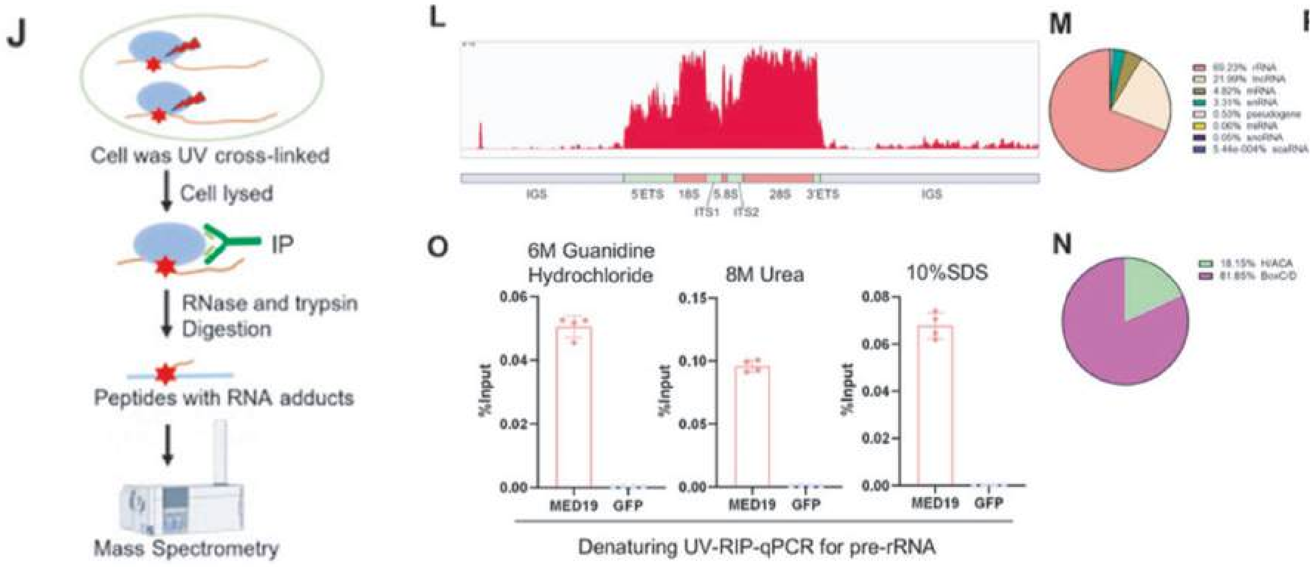
Yang Ming[1,2], Zhao Jiamin[3], Peng Yu[1], Lu Chengjiang[4], Zhang Yiyang[1], Zhang Hanqing[1], Qiang Zhou[2], Gang Wang[1,*]



Journal	Nucleic Acids Research
IF	13.1
Year	2025
Relative Products	**Small RNA Lib Prep Kit for Illumina V3 (RK20312)**
Application	**CLIP-seq, RiboMeth-seq**
Affiliations	Fudan University

Abstract: The Mediator complex is a transcriptional co-factor for RNA polymerase II (Pol II)-dependent gene expression, with MED19 serving as an integral subunit. While MED19 overexpression has been documented in diverse cancer types to promote tumor progression, the underlying molecular mechanisms remain poorly understood. Here, we uncover a previously unrecognized function whereby MED19 localizes to the nucleolus independently of the Mediator complex. This nucleolar targeting is mediated by a conserved poly-lysine motif at the MED19 C-terminus, which enables binding to ribosomal RNA (rRNA) and fibrillarin (FBL), a catalytic component of the 2'-O-methyltransferase complex and pre-rRNA processing factor. Mechanistically, MED19 facilitates rRNA processing and 2'-O-methylation that promotes the efficiency of internal ribosome entry site-dependent translation for a number of onco-promoting genes including c-Myc. Collectively, these findings reveal a novel Mediator-independent function of MED19 in regulating ribosome-mediated translational control, thus providing mechanistic insights into its onco-promoting role.

Related Figure:



The library construction kits used in the related studies of ClIP-seq and RiboMeth-seq in this article are RK20312 from ABclonal.

RESEARCH **Open Access**

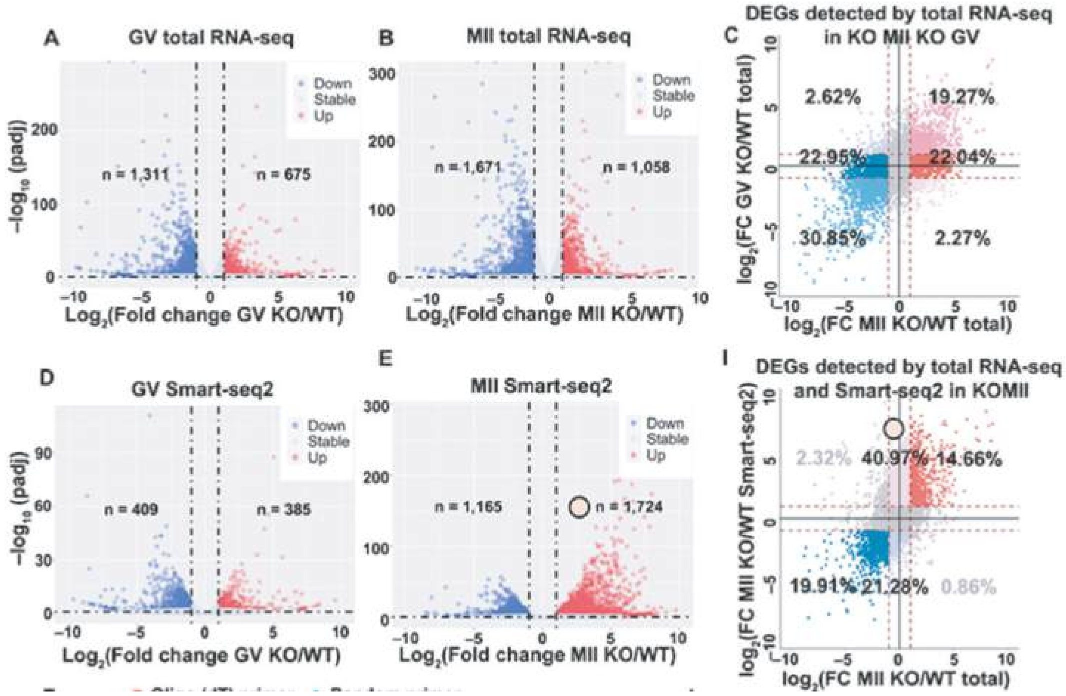
ZAR1 and ZAR2 orchestrate the dynamics of maternal mRNA polyadenylation during mouse oocyte development

Yu-Ke Wu[1], Ruibao Su[2], Zhi-Yan Jiang[1], Yun-Wen Wu[1], Yan Rong[1,3], Shu-Yan Ji[4], Jingwen Liu[5,6], Zhuoyue Niu[5,6], Zhiyi Li[7], Yuanchao Xue[2], Falong Lu[5,6] and Heng-Yu Fan[1,3,8*]

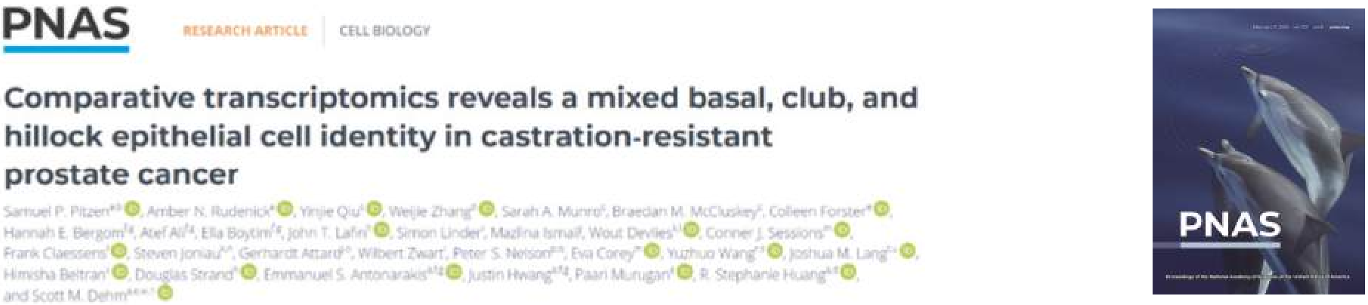
Journal	Genome Biol
IF	9.4
Year	2025
Relative Products	**rRNA Depletion module (H/M/R) (RK20348), AFTMag NGS DNA Clean Beads (RK20257)**
Application	**Total RNA-seq**
Affiliations	Zhejiang University

Abstract: BACKGROUND During meiosis, the oocyte genome keeps dormant for a long time until zygotic genome activation. The dynamics and homeostasis of the maternal transcriptome are essential for maternal-to-zygotic transition. Zygotic arrest 1 (ZAR1) and its homolog, ZAR2, are RNA-binding proteins that are important for the regulation of maternal mRNA stability.RESULTS Smart-seq2 analysis reveals drastically downregulated maternal transcripts. However, the detection of transcript levels by Smart-seq2 may be biased by the polyadenylated tail length of the mRNAs. Similarly, differential expression of maternal transcripts in oocytes with or without Zar1/2 differs when analyzed using total RNA-seq and Smart-seq2, suggesting an influence of polyadenylation. Combined analyses using total RNA-seq, LACE-seq, PAIso-seq2, and immunoprecipitation-mass spectrometry reveals that ZAR1 may target the 3'-untranslated regions of maternal transcripts, regulates their stability in germinal vesicle stage oocytes, and interacts with other proteins to control the polyadenylation of mRNAs.CONCLUSIONS The jointly analyzed multi-omics data highlight the limitations of Smart-seq2 in oocytes, clarify the dynamics of the maternal transcriptome, and uncover new roles of ZAR1 in regulating the maternal transcriptome.

Related Figure:



The rRNA depletion module used for Total-seq in this article is RK20348 from ABclonal.



PNAS

RESEARCH ARTICLE | CELL BIOLOGY

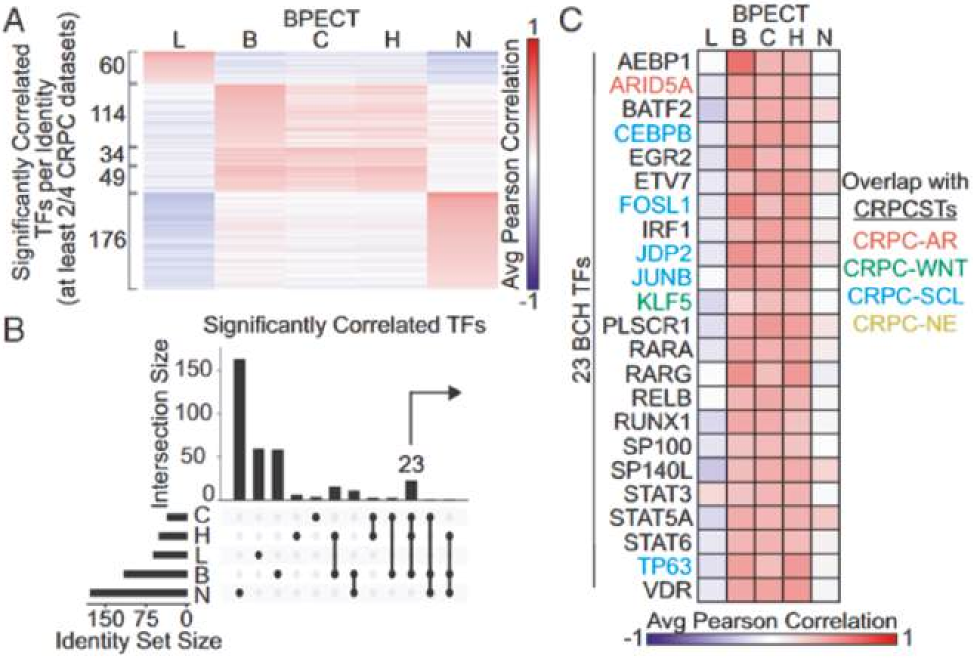
Comparative transcriptomics reveals a mixed basal, club, and hillock epithelial cell identity in castration-resistant prostate cancer

Samuel P. Pitzen, Amber N. Rudenick, Yinjie Qiu, Weijie Zhang, Sarah A. Munro, Braedan M. McCluskey, Colleen Forster, Hannah E. Bergom, Atef Ali, Ella Boytim, John T. Lafin, Simon Linder, Mazlina Ismail, Wout Devlies, Conner J. Sessions, Frank Claessens, Steven Joniau, Gerhardt Attard, Wilbert Zwart, Peter S. Nelson, Eva Corey, Yuzhuo Wang, Joshua M. Lang, Himisha Beltran, Douglas Strand, Emmanuel S. Antonarakis, Justin Hwang, Paari Murugan, R. Stephanie Huang, and Scott M. Dehm

Journal	Proc Natl Acad Sci U S A
IF	9.1
Year	2025
Relative Products	**mRNA-seq Lib Prep Module for Illumina (RK20350)；Poly(A) mRNA Capture Module (RK20340)**
Application	**RNA-seq**
Affiliations	University of Minnesota

Abstract: Inhibiting the androgen receptor (AR) is effective for treatment of advanced prostate cancers because of their AR-dependent luminal epithelial cell identity. Tumors progress during therapy to castration-resistant prostate cancer (CRPC) by restoring AR signaling and maintaining luminal identity or by converting through lineage plasticity to a neuroendocrine (NE) identity or double-negative CRPC (DNPC) lacking luminal or NE identities. Here, we show that DNPC cells express genes defining basal, club, and hillock epithelial cells from benign prostate. We identified KLF5 as a regulator of genes defining this mixed basal, club, and hillock cell identity in DNPC models. KLF5-mediated upregulation of RARG uncovered a DNPC sensitivity to growth inhibition by retinoic acid receptor agonists, which down-regulated KLF5 and up-regulated AR. These findings offer CRPC classifications based on prostate epithelial cell identities and nominate KLF5 and RARG as therapeutic targets for CRPC displaying a mixed basal, club, and hillock identity.

Related Figure:



The library construction kit used for RNA-seq in this article is RK20350 from ABclonal.

nature > scientific data > data descriptors > article

Data Descriptor | Open access | Published: 01 July 2025

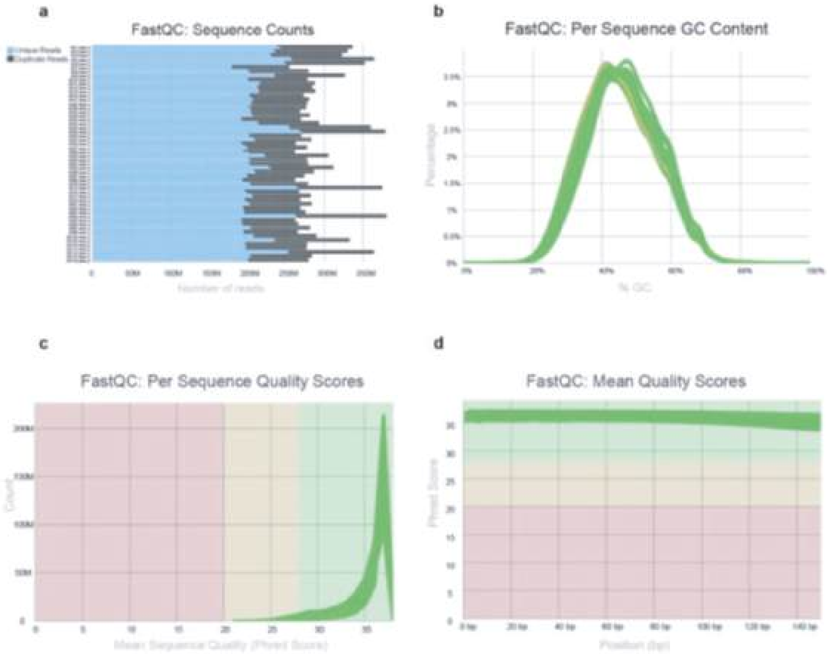
Whole-Genome Sequencing of 54 Dengchuan Cattle (*Bos taurus*) from Southwest China

Kun-Jiang Feng, Mei Yang, Yong-Yan Liu, Zong-You Huang, Ya-Man Li, Ling-Yue Wen, Heng Xiao & Shanyuan Chen

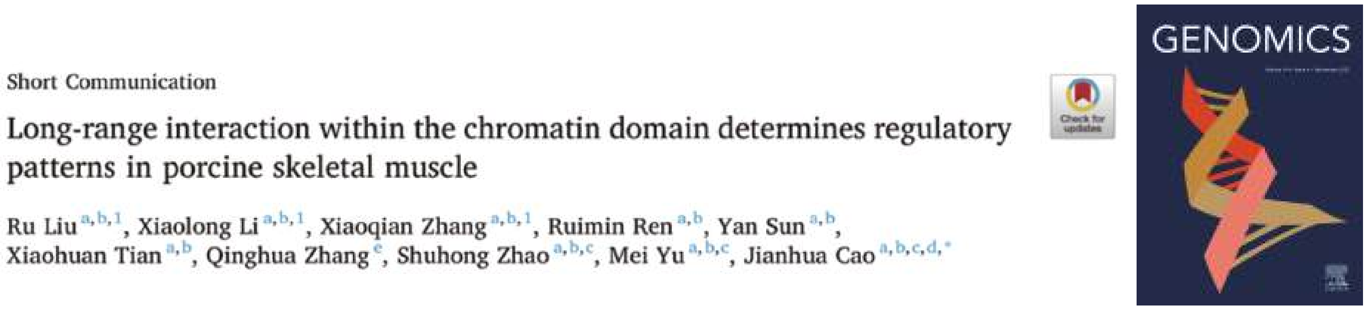
Journal	Sci Data
IF	6.9
Year	2025
Relative Products	**FS Pro DNA Lib Prep Kit V2 (RK20275) (replaced by RK29832)**
Application	**DNA-seq**
Affiliations	Yunnan University

Abstract: Domestic cattle (Bos taurus) play a significant role in human society as they provide abundant food resources and contribute to the development of agriculture and traditional culture. Dengchuan cattle, a local breed from Yunnan, Southwest China, are known for their high-quality milk and are at risk of extinction due to crossbreeding. To preserve the superior genetic resources of Dengchuan cattle, this study conducted whole-genome sequencing of 54 Dengchuan cattle using blood DNA samples, generating approximately 3.56 TB of clean data with an average sequencing depth of 32.78X. The sequencing data were aligned to the bovine reference genome (ARS-UCD2.0), achieving an average alignment rate of 99.85%. A total of 9,950,420 SNPs and 2,476,207 indels were detected using variant calling workflow. These data were utilized to characterize genomic profile of this unique cattle breed. The data generated in this study can be incorporated into the global cattle genomic diversity database, providing valuable information for comparative studies on cattle.

Related Figure:



The library preparation kit used for DNA resequencing in this article is RK20275 from ABclonal.



Short Communication

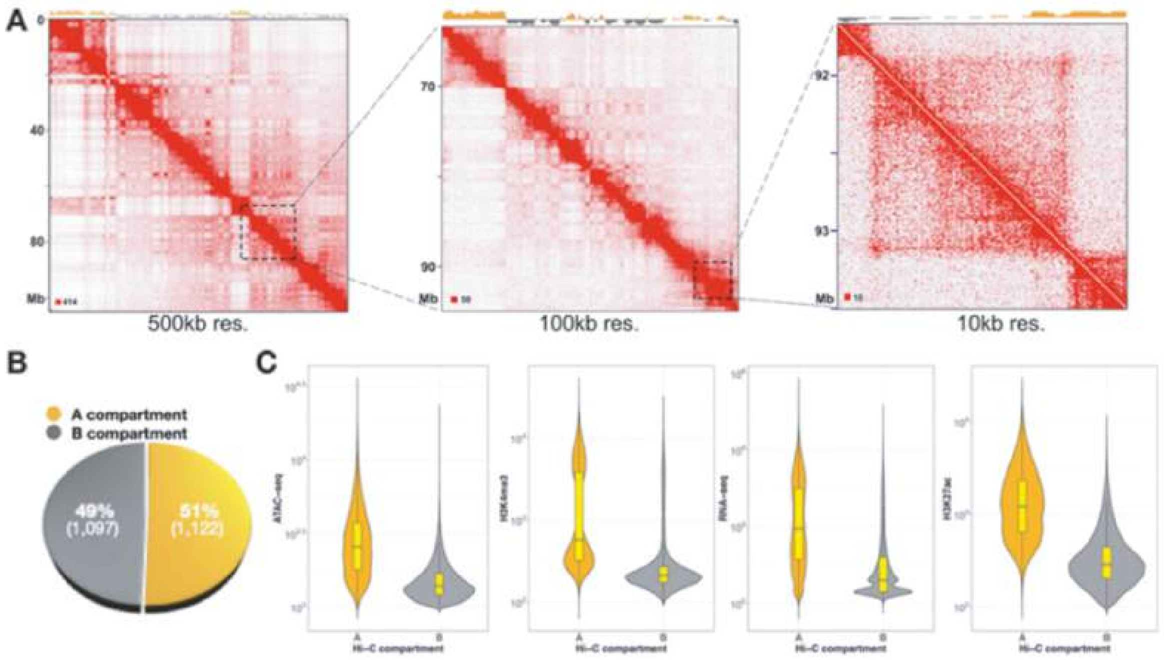
Long-range interaction within the chromatin domain determines regulatory patterns in porcine skeletal muscle

Ru Liu[a,b,1], Xiaolong Li[a,b,1], Xiaoqian Zhang[a,b,1], Ruimin Ren[a,b], Yan Sun[a,b],
Xiaohuan Tian[a,b], Qinghua Zhang[e], Shuhong Zhao[a,b,c], Mei Yu[a,b,c], Jianhua Cao[a,b,c,d,*]

Journal	Genomics
IF	3
Year	2022
Relative Products	**FS Pro DNA Lib Prep Kit for Illumina (RK20261) (replaced by RK29832); T4 DNA Ligase (RK21501)**
Application	**Hi-C**
Affiliations	Huazhong Agricultural University

Abstract: Spatial chromatin structure is crucial for understanding the early growth and development of porcine skeletal muscle. However, its characteristic of 3D architecture and elaborate regulation of gene transcription remains unclear. In this study, ChIA-PET method is used to study the changes of early chromatin three-dimensional structure in skeletal muscle of lean type Yorkshire pig and fat type Meishan pig. Integrating the in situ Hi-C data revealed the 3D architecture and long-range interaction of the porcine muscle. The results showed the CTCF/RNAPII mediated long-range interaction shapes the different chromatin architecture and dominates the unique regulation of enhancers. In addition, the results revealed that key myogenic genes like ssc-mir-1 had a unique enhancer regulation function in myogenesis. Interestingly, the FGF6 gene is of breed-specific regulation, implying the difference between two breeds in skeletal muscle development. Our research thus may provide a clue for the porcine genetic improvement of skeletal muscle.

Related Figure:



The library construction kit and T4 DNA ligase used in the HiC experiment in the article are RK20261 and RK20250 from ABclonal, respectively.

Common application scenarios and product portfolio of NGS & epigenetic biology

Application	ABclonal Related Products
ChIP-seq	• Sonication ChIP Kit (RK20258) • Rapid Plus DNA Lib Prep Kit V2(No DDREs) (RK20271) • AFTSpin Multifunction DNA Purification Kit (RK30100)
CUT&RUN	• pAG-MNase (RK20560) • Rapid Plus DNA Lib Prep Kit V2(No DDREs) (RK20271)
HiC-seq	• Dpn II (RK21110), Hind III (RK21108) • Klenow Fragment 3'→5' exo- (RK20526) • T4 DNA Polymerase (5,000 U/mL)(RK20539) • T4 Polynucleotide Kinase (RK20524) • T4 DNA Ligase (High Conc.) (RK21500) • FS Pro DNA Lib Prep Kit V2 (RK29832)(replaces RK20275)
cfDNA Sequencing	• AFTMag Quick Cell-Free DNA Extraction Maxi Kit (RK30184) • Rapid Plus DNA Lib Prep Kit V2(No DDREs) (RK20271)
GUIDE-seq	• AFTSpin Blood/Tissue/Cell Fast DNA Extraction Kit (RK30110) • Rapid Plus DNA Lib Prep Kit V2(No DDREs) (RK20271) • TruePol 2X PCR Mix for NGS (RK20726)
mNGS	• AFTMag Stool/Soil DNA Extraction Kit (RK30157) • FS Pro DNA Lib Prep Kit V2(RK29832)(replaces RK20275)
16S rRNA Sequencing	• AFTMag Stool/Soil DNA Extraction Kit (RK30157) • TruePol 2X PCR Mix for Microbiome (RK20270) • Rapid Plus DNA Lib Prep Kit V2(No DDREs) (RK20271)
WGA/MDA	• Pilot phi29 DNA Polymerase (RK21006) • FS Pro DNA Lib Prep Kit V2(RK29832)(replaces RK20275)

Application	ABclonal Related Products
RNA-seq	• Poly(A) mRNA Capture Module (RK20340) • rRNA Depletion module (H/M/R) (RK20348) • rRNA Depletion Kits (Human rRNA & Globin)(RK30209) • rRNA Depletion Kits (Pan-Prokaryote)(RK30207) • rRNA Depletion Kits (Plant)(RK30208) • mRNA-seq Lib Prep Module for Illumina (RK20350)
Ribo-seq	• Small RNA Lib Prep Kit for Illumina V3 (RK20312) • T4 Polynucleotide Kinase (RK20524) • T4 RNA Ligase 2, truncated KQ (RK20504) • T4 RNA Ligase 1 (RK20503) • rRNA Depletion module (H/M/R)(RK20348) • rRNA Depletion Kits (Pan-Prokaryote)(RK30207) • rRNA Depletion Kits (Plant)(RK30208)
Pro-seq	• Small RNA Lib Prep Kit for Illumina V3 (RK20312) • T4 Polynucleotide Kinase (RK20524) • T4 RNA Ligase 2, truncated KQ (RK20504) • T4 RNA Ligase 1 (RK20503)
Clip-seq	• RNase Inhibitor, Mammalian (RK21401) • T4 Polynucleotide Kinase (RK20524) • T4 RNA Ligase 2, truncated KQ (RK20504) • T4 RNA Ligase 1 (RK20503) • Small RNA Lib Prep Kit for Illumina V3 (RK20312)
SMART-seq	• Single Cell/Low Input cDNA Synthesis & Amplification Module (RK20310) • ABScript Full Length Reverse Transcriptase for Single Cell (RK20414) • RNase Inhibitor, Mammalian (RK21401)

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