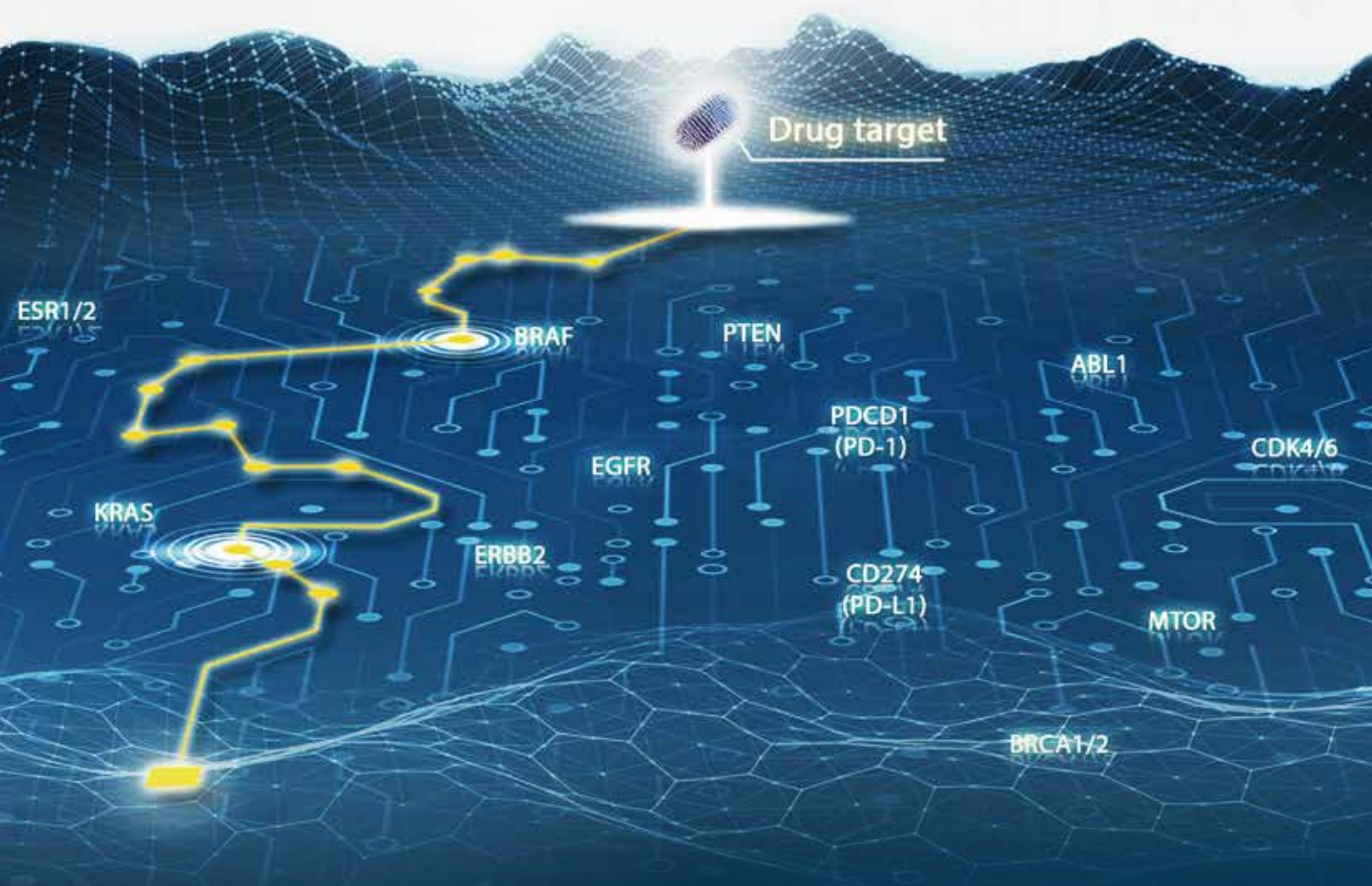


Pathway-driven tumor profiling connects insights to unlock new opportunities

ACT Onco[®] +

Planning treatment strategies through comprehensive genomic profiling for more than 400 cancer-related genes.



This material is intended for healthcare professionals only.

ACT Onco[®] + Technical Specifications

Sample Types

FFPE (formalin-fixed paraffin-embedded) sample:
5–20 unstained sections (5 µm/slide)
1 H&E stained slide (5 µm)

* Cell block from pleural effusion, ascites, or pericardial hydrops ($\geq 1 \times 10^6$ cells)

* The test may return with limited or no result. Tumor purity for ACT Onco[®] + should be more than 30%.

Sequencing of all exons and selected introns of cancer-related genes
> 400 cancer-related genes

Integrated ACTFusion[™] test

Comprehensive tumor RNA fusion tests for 13 genes with known and novel fusion transcripts.

Prediction of Immunotherapy Response

Tumor Mutational Burden (TMB)
Microsatellite Instability (MSI) Testing

Target Region

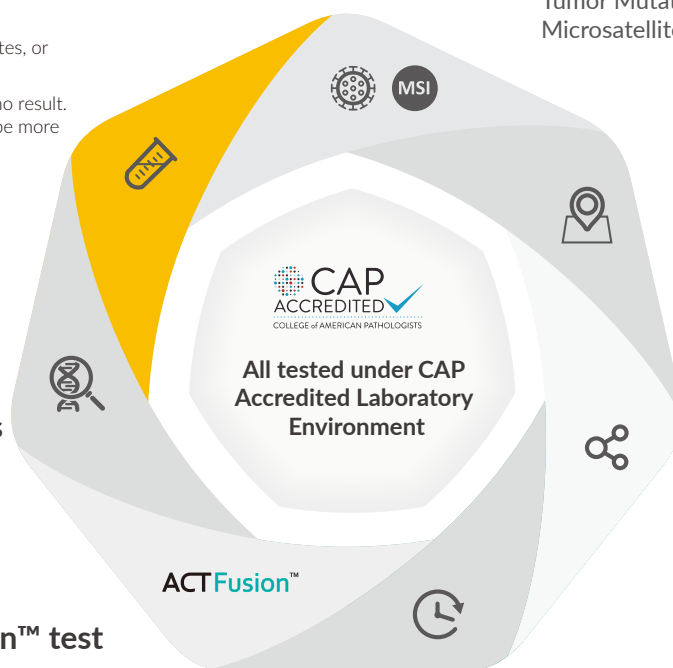
> 1.8 Mbps

NGS Sequencing Mean Depth

$\geq 500\times$

Turnaround Time

8 working days



For Solid Tumor

Locally advanced, metastatic, or recurrent

Comprehensively understanding genetic mutations to select suitable drugs

Ineffective standard treatment

Leverage genetic insights to identify alternative drug options beyond standard treatment.

Newly diagnosed

Assess genetic mutations early to choose the best treatment

ACT Onco[®] +
A pathway-based approach to genomic insights

ACT Onco[®] +



A Pathway-Based Approach To Genomic Insights

Sequencing of >400 cancer-related genes covering most key cancer signaling pathways for targeted therapy, analyzing single nucleotide variants (SNVs), insertions/deletions (InDels), copy number variations (CNVs), and fusion gene alterations.



Predicting Immunotherapy Response

Incorporating TMB (Tumor Mutational Burden), immune-related gene information, and MSI (Microsatellite Instability) status for a more accurate immunotherapy recommendation.



Short Turnaround Time

Providing professional solutions within 8 working days (starting from the day qualified specimen is received at the lab).



Quality Guaranteed

All tests carried out by ACT Genomics are conducted in a CAP Accredited Laboratory. Also garnered the LDTs (Laboratory Developed Tests and Services) certification by the Taiwan FDA (LDT0001).



Providing Report Consultation

Every report is complemented with a one-to-one consultation with the medical team.



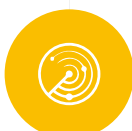
Integrated Medical Information

Integrating cancer biology, medical biology, molecular biology, cell biology, immunology, bioinformatics, data mining, and pharmaceutical biology to provide insightful therapeutic interpretation.



High Sensitivity

Next Generation Sequencing (NGS) offers high sensitivity, ensuring accurate detection of genetic mutations addressing challenges associated with tumor heterogeneity.



For Solid Tumor

Locally advanced, metastatic, or recurrent

Comprehensively understanding genetic mutations to select suitable drugs.

Ineffective standard treatment

Use genetic insights to find additional drug options than standard treatment.

Newly diagnosed

Identify genetic mutations early to optimize treatment selection.



Comprehensive Information Provided

Targeted Therapy

Chemotherapy

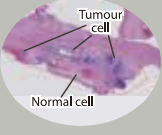
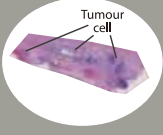
Immunotherapy

Hormonal Therapy

Advantages For Clinical Application

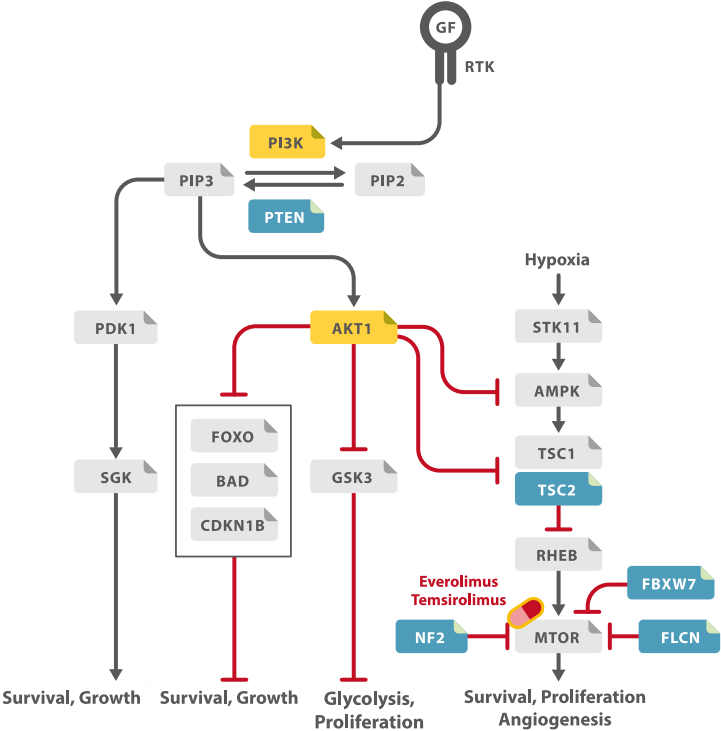
Macrodissection for Accurate Copy Number Variation (CNV) Analysis

Using an example of ERBB2 gene amplification in cancer cells at 8 ploidy, when the tumor DNA proportion extracted increases from 20% to 70%, the calculated CNV rises from 3.2 to 6.2. This allows for a more accurate prediction of Herceptin treatment efficacy.

Macrodissection	Before Processing	After Processing
Viable tumor cells		
Tumor DNA Proportion	20% 	70% 
Calculated ERBB2 CNV	3.2 $= 8 \times 0.20 + 2 \times 0.80$	6.2 $= 8 \times 0.70 + 2 \times 0.30$



Integrating Tumor Signaling pathway Analysis to Provide Personalized Treatment Strategies



Genetic mutations detected in the test are analyzed within the signaling pathway. By identifying the correct mutation source from upstream and downstream gene networks, bioinformatics can perform systematic drug analysis, assisting physicians in developing more accurate treatment plans.



Citation:

1. He Y et al., Targeting PI3K/Akt signal transduction for cancer therapy. Signal Transduct Target Ther 6: 425, 2021.
2. Shen S et al., Sequential use of PI3K/AKT/mTOR pathway inhibitors alpelisib and everolimus in patients with hormone receptor-positive (HR+) metastatic breast cancer. J Clin Oncol 42: 10, 2024.
3. Bozec A et al., The mTOR-targeting drug temsirolimus enhances the growth-inhibiting effects of the cetuximab-bevacizumab-irradiation combination on head and neck cancer xenografts. Oral Oncol 47: 340-344, 2011.

Bioinformatics: Transforming Data into Practical Treatment Strategies

Gene testing goes beyond DNA sequencing; more importantly, it involves deciphering clinically meaningful information from complex genetic data. The bioinformatics and medical informatics teams integrate extensive domestic and international databases to continuously update and maintain an in-house database for accurate identification of cancer-related mutations. By comparing genetic and drug-related information, a comprehensive and valuable medical report is provided.

The main process of Bioinformatics and Medical informatics:

- **Variant Calling:** Accurately identifying genetic mutations from sequencing data.
- **Data Annotation:** Annotating mutations and identifying cancer-related genetic variations.
- **Clinical Interpretation:** Utilizing literature to find clinically significant genetic mutations and providing guidance for physicians on treatment selection.

ACT Onco[®] + Report

Name
Project ID:
Report No.:
Date Reported:

PATIENT		
Name:		Patient ID:
Date of Birth:		Gender:
Diagnosis: Lung cancer		
ORDERING PHYSICIAN		
Name:		Tel:
Facility:		
Address:		
SPECIMEN		
Specimen ID:	Collection site:	Type: FFPE tissue
Date received:	Lab ID:	D/ID:

ABOUT ACT Onco[®] +

The test is a next-generation sequencing (NGS)-based assay developed for efficient and comprehensive genomic profiling of cancers. This test interrogates coding regions of 440 genes associated with cancer treatment, prognosis and diagnosis. Genetic mutations detected by this test include small-scale mutations like single nucleotide variants (SNVs), small insertions and deletions (InDels) (≤ 15 nucleotides) and large-scale genomic alterations like copy number alterations (CNAs). The test also includes an RNA test, detecting fusion transcripts of 13 genes.

SUMMARY FOR ACTIONABLE VARIANTS

VARIANTS/BIOMARKERS WITH EVIDENCE OF CLINICAL SIGNIFICANCE

Genomic Alterations/Biomarkers	Probable Effects in Patient's Cancer Type		Probable Sensitive in Other Cancer Types
	Sensitive	Resistant	
EGFR L858R	Afatinib, Dacomitinib, Erlotinib, Gefitinib, Osimertinib	-	-
EGFR T790M	Osimertinib	Afatinib, Dacomitinib, Erlotinib, Gefitinib	-

VARIANTS/BIOMARKERS WITH POTENTIAL CLINICAL SIGNIFICANCE

Genomic Alterations/Biomarkers	Possibly Sensitive	Possibly Resistant
TSC1 S457fs	Everolimus, Temsirolimus	-

Note:

- The above summary tables present genomic variants and biomarkers based on the three-tiered approach proposed by US FDA for reporting tumor profiling NGS testing. "Variants/biomarkers with evidence of clinical significance" refers to mutations that are widely recognized as standard-of-care biomarkers (FDA level 2/AMP tier 1). "Variants/biomarkers with potential clinical significance" refers to mutations that are not included in the standard of care but are informational for clinicians, which are commonly biomarkers used as inclusion criteria for clinical trials (FDA level 3/AMP tier 2).
- The therapeutic agents and possible effects to a given drug are based on mapping the variants/biomarkers with ACT Genomics clinical knowledge database. The mapping results only provide information for reference, but not medical recommendation.
- Please refer to corresponding sections for more detailed information about genomic alteration and clinical relevance listed above.



ACT Genomics only provides a technical report of the test; please consult a specialist physician to determine the appropriate clinical solution and follow the instruction of the physician. The results are only valid for the tested samples.
ACT Genomics Laboratory (HQ): 3F, No. 345, Kinde 3rd Rd, Taipei, Taiwan 11454
Email: service@actgenomics.com | T: +886-2-2755-0680 | F: +886-2-2755-5016

CNV

A DNA segment of 1Kb or larger that is present at a variable copy number in comparison with a reference genome¹ including amplification, heterozygous deletion or homozygous deletion.

¹ Zack TI et al., Pan-cancer patterns of somatic copy number alterations. Nature Genetics 45: 1134-1140, 2013

ACT Fusion[™]

FUSION GENE

Fusion gene is the product formed from the joining of two separate genes when there is a translocation, interstitial deletion or chromosomal inversion occurring in the genome.

TMB

Tumor mutational burden (TMB) represents the number of nonsynonymous mutations per megabase in the coding regions of sequenced DNA. TMB is a quantitative biomarker used to predict sensitivity to checkpoint inhibitor therapy.

Note: All tests carried out by ACT Genomics are conducted in a CAP-Accredited laboratory. Please consult your physician for any enquiries related to clinical interpretation of the test results. The images used are for illustrative purposes only. The actual report may differ.

provides a comprehensive & concise report to aid in clinical decision making

SNV

SNV represents a difference in a single DNA building block, called a nucleotide (e.g. activating hotspot mutation in an oncogene or truncating mutation in a tumor suppressor gene).

LEVEL OF MUTATION FREQUENCY

Allele frequency provides information about the allele that may be responsible for the tumorigenesis.

ACTOnco® + Report

Name
Project ID:
Report No.:
Date Reported:

TESTING RESULTS

VARIANT(S) WITH CLINICAL RELEVANCE

- Single Nucleotide and Small InDel Variants

Gene	Amino Acid Change	Allele Frequency
EGFR	L858R	17.5%
EGFR	T790M	10.9%
TSC1	S457fs	21.5%

- Copy Number Alterations

Chromosome	Gene	Variation	Copy Number
Chr7	IL6	Amplification	7*
Chr5	RICTOR	Amplification	15

* Increased gene copy number was observed.

- Fusions

Fusion Gene & Exon	Transcript ID
No fusion gene detected in this sample.	

- Immune Checkpoint Inhibitor (ICI) Related Biomarkers

Biomarker	Results
Tumor Mutational Burden (TMB)	3.2 muts/Mb
Microsatellite Instability (MSI)	Microsatellite stable (MSS)

Note:

- Loss of heterozygosity (LOH) information was used to infer tumor cellularity. Copy number alteration in the tumor was determined based on 30% tumor purity.
- TMB was calculated by using the sequenced regions of ACTOnco®+ to estimate the number of somatic nonsynonymous mutations per megabase of all protein-coding genes (whole exome). The threshold for high mutation load is set at ≥ 7.5 mutations per megabase. TMB, microsatellite status and gene copy number deletion cannot be determined if calculated tumor purity is $< 30\%$.

THERAPEUTIC IMPLICATIONS

TARGETED THERAPIES

Genomic Alterations	Therapies	Effect
Level 1		
EGFR L858R	Afatinib, Dacomitinib, Erlotinib, Gefitinib, Osimertinib	sensitive
EGFR T790M	Osimertinib	sensitive
Level 2		
EGFR T790M	Afatinib, Dacomitinib, Erlotinib, Gefitinib	resistant
Level 3B		
TSC1 S457fs	Everolimus, Temsirolimus	sensitive

Therapies associated with benefit or lack of benefit are based on biomarkers detected in this tumor and published evidence in professional guidelines or peer-reviewed journals.

Level	Description
1	FDA-approved biomarkers predictive of response or resistance to FDA-approved drugs in this indication
2	Standard care biomarkers (recommended by the NCCN guideline) predictive of response or resistance to FDA-approved drugs in this indication
3A	Biomarkers predictive of response or resistance to therapies approved by the FDA or NCCN guideline in a different cancer type
3B	Biomarkers that serve as inclusion criteria for clinical trials (minimal supportive data required)
4	Biomarkers that show plausible therapeutic significance based on small studies, low case reports, or preclinical studies

MSI

Microsatellite instability (MSI) is a form of genomic instability due to the insertion or deletion of repeating units during replication, and failure of the mismatch repair system. Checkpoint inhibitors have been approved for MSI high or mismatch repair deficient solid tumors.

THERAPEUTIC IMPLICATIONS

Providing several treatment options for patient's and physician's reference.



ACT Genomics only provides a technical report of the test, please consult a specialist physician to determine the appropriate clinical solution and follow the instruction of the physician. The results are only valid for the tested samples.
ACT Genomics Laboratory (HQ) 3F, No. 345, Kailu, 1st Rd, Taipei, Taiwan 11416
Email: sales@actgenomics.com T: +886-2-2765-0666 F: +886-2-2765-9176

Genes List

Genes in ACTOnco[®] + (◆ SNV / InDel ■ CNV)

ABCB1 ◆	CBFB ◆◆	DPYD ◆◆	GNAQ ◆◆	MAX ◆◆	PIK3CG ◆◆	SF3B1 ◆◆
ABCC2 ◆	CBL ◆◆	DTX1 ◆◆	GNAS ◆◆	MCL1 ◆◆	PIK3R1 ◆◆	SGK1 ◆◆
ABCG2 ◆	CCNA1 ◆◆	E2F3 ◆◆	GREM1 ◆◆	MDM2 ◆◆	PIK3R2 ◆◆	SH2D1A ◆
ABL1 ◆◆	CCNA2 ◆◆	EGFR ◆◆	GRIN2A ◆◆	MDM4 ◆◆	PIK3R3 ◆◆	SLC19A1 ◆
ABL2 ◆◆	CCNB1 ◆◆	EP300 ◆◆	GSK3B ◆◆	MED12 ◆◆	PIM1 ◆◆	SLC22A2 ◆
ADAMTS1 ◆◆	CCNB2 ◆◆	EPCAM ◆◆	GSTP1 ◆	MEF2B ◆◆	PMS1 ◆◆	SLCO1B1 ◆
ADAMTS13 ◆◆	CCNB3 ◆◆	EPHA2 ◆◆	GSTT1 ◆	MEN1 ◆◆	PMS2 ◆◆	SLCO1B3 ◆
ADAMTS15 ◆◆	CCND1 ◆◆	EPHA3 ◆◆	HGF ◆◆	MET ◆◆	POLB ◆◆	SMAD2 ◆◆
ADAMTS16 ◆◆	CCND2 ◆◆	EPHA5 ◆◆	HIF1A ◆◆	MITF ◆◆	POLD1 ◆◆	SMAD3 ◆◆
ADAMTS18 ◆◆	CCND3 ◆◆	EPHA7 ◆◆	HIST1H1C ◆	MLH1 ◆◆	POLE ◆◆	SMAD4 ◆◆
ADAMTS6 ◆◆	CCNE1 ◆◆	EPHB1 ◆◆	HIST1H1E ◆	MPL ◆◆	PPARG ◆◆	SMARCA4 ◆◆
ADAMTS9 ◆◆	CCNE2 ◆◆	ERBB2 ◆◆	HNF1A ◆◆	MRE11 ◆◆	PPP2R1A ◆◆	SMARCB1 ◆◆
ADAMTSL1 ◆◆	CCNH ◆◆	ERBB3 ◆◆	HR ◆◆	MSH2 ◆◆	PRDM1 ◆◆	SMO ◆◆
ADGRA2 ◆◆	CD19 ◆◆	ERBB4 ◆◆	HRAS ◆◆	MSH6 ◆◆	PRKAR1A ◆◆	SOCS1 ◆
ADH1C ◆	CD274 ◆◆	ERCC1 ◆◆	HSP90AA1 ◆◆	MTHFR ◆	PRKCA ◆◆	SOX2 ◆
AKT1 ◆◆	CD58 ◆◆	ERCC2 ◆◆	HSP90AB1 ◆◆	MTOR ◆◆	PRKCB ◆◆	SOX9 ◆◆
AKT2 ◆◆	CD70 ◆	ERCC3 ◆◆	HSPA4 ◆◆	MUC16 ◆◆	PRKCG ◆◆	SPEN ◆◆
AKT3 ◆◆	CD79A ◆◆	ERCC4 ◆◆	HSPA5 ◆◆	MUC4 ◆◆	PRKCI ◆◆	SPOP ◆◆
ALDH1A1 ◆	CD79B ◆◆	ERCC5 ◆◆	IDH1 ◆◆	MUC6 ◆◆	PRKCQ ◆◆	SRC ◆◆
ALK ◆◆	CDC73 ◆◆	ERG ◆◆	IDH2 ◆◆	MUTYH ◆◆	PRKDC ◆◆	STAG2 ◆◆
AMER1 ◆◆	CDH1 ◆◆	ESR1 ◆◆	IFNL3 ◆	MYC ◆◆	PRKN ◆◆	STAT3 ◆◆
APC ◆◆	CDK1 ◆◆	ESR2 ◆◆	IGF1 ◆◆	MYCL ◆◆	PSMB8 ◆◆	STK11 ◆◆
AR ◆◆	CDK12 ◆◆	ETV1 ◆◆	IGF1R ◆◆	MYCN ◆◆	PSMB9 ◆◆	SUFU ◆◆
ARAF ◆◆	CDK2 ◆◆	ETV4 ◆◆	IGF2 ◆◆	MYD88 ◆◆	PSME1 ◆◆	SYK ◆◆
ARID1A ◆◆	CDK4 ◆◆	EZH2 ◆◆	IKBKB ◆◆	NAT2 ◆	PSME2 ◆◆	SYNE1 ◆◆
ARID1B ◆◆	CDK5 ◆◆	FAM46C ◆◆	IKBKE ◆◆	NBN ◆◆	PSME3 ◆◆	TAF1 ◆◆
ARID2 ◆◆	CDK6 ◆◆	FANCA ◆◆	IKZF1 ◆◆	NEFH ◆◆	PTCH1 ◆◆	TAP1 ◆◆
ASXL1 ◆◆	CDK7 ◆◆	FANCC ◆◆	IL6 ◆◆	NF1 ◆◆	PTEN ◆◆	TAP2 ◆◆
ATM ◆◆	CDK8 ◆◆	FANCD2 ◆◆	IL7R ◆◆	NF2 ◆◆	PTGS2 ◆◆	TAPBP ◆◆
ATR ◆◆	CDK9 ◆◆	FANCE ◆◆	INPP4B ◆◆	NFE2L2 ◆◆	PTPN11 ◆◆	TBX3 ◆◆
ATRX ◆◆	CDKN1A ◆◆	FANCF ◆◆	INSR ◆◆	NFKB1 ◆◆	PTPRD ◆◆	TEK ◆◆
AURKA ◆◆	CDKN1B ◆◆	FANCG ◆◆	IRF4 ◆◆	NFKBIA ◆◆	PTPRT ◆◆	TERT ◆◆
AURKB ◆◆	CDKN2A ◆◆	FANCL ◆◆	IRS1 ◆◆	NKX2-1 ◆	RAC1 ◆◆	TET1 ◆◆
AXIN1 ◆◆	CDKN2B ◆◆	FAS ◆◆	IRS2 ◆	NOTCH1 ◆◆	RAD50 ◆◆	TET2 ◆◆
AXIN2 ◆◆	CDKN2C ◆◆	FAT1 ◆◆	JAK1 ◆◆	NOTCH2 ◆◆	RAD51 ◆◆	TGFBR2 ◆◆
AXL ◆◆	CEBPA ◆	FBXW7 ◆◆	JAK2 ◆◆	NOTCH3 ◆◆	RAD51B ◆◆	TMSB4X ◆
B2M ◆◆	CHEK1 ◆◆	FCGR2B ◆◆	JAK3 ◆◆	NOTCH4 ◆◆	RAD51C ◆◆	TNF ◆◆
BAP1 ◆◆	CHEK2 ◆◆	FGF1 ◆	JUN ◆	NPM1 ◆◆	RAD51D ◆◆	TNFAIP3 ◆◆
BARD1 ◆◆	CIC ◆◆	FGF10 ◆◆	KAT6A ◆◆	NQO1 ◆	RAD52 ◆◆	TNFRSF14 ◆◆
BCL10 ◆◆	CREBBP ◆◆	FGF14 ◆◆	KDM5A ◆◆	NRAS ◆◆	RAD54L ◆◆	TNFSF11 ◆◆
BCL2 ◆	CRKL ◆◆	FGF19 ◆	KDM5C ◆◆	NSD1 ◆◆	RAF1 ◆◆	TOP1 ◆◆
BCL2L1 ◆◆	CRLF2 ◆◆	FGF23 ◆◆	KDM6A ◆◆	NTRK1 ◆◆	RARA ◆◆	TP53 ◆◆
BCL2L2 ◆	CSF1R ◆◆	FGF3 ◆◆	KDR ◆◆	NTRK2 ◆◆	RB1 ◆◆	TPMT ◆
BCL6 ◆◆	CTCF ◆◆	FGF4 ◆	KEAP1 ◆◆	NTRK3 ◆◆	RBM10 ◆◆	TSC1 ◆◆
BCL9 ◆◆	CTLA4 ◆◆	FGF6 ◆◆	KIT ◆◆	PAK3 ◆◆	RECQL4 ◆◆	TSC2 ◆◆
BCOR ◆◆	CTNNA1 ◆◆	FGFR1 ◆◆	KMT2A ◆◆	PALB2 ◆◆	REL ◆◆	TSHR ◆◆
BIRC2 ◆◆	CTNNB1 ◆◆	FGFR2 ◆◆	KMT2C ◆◆	PARP1 ◆◆	RET ◆◆	TYMS ◆◆
BIRC3 ◆◆	CUL3 ◆◆	FGFR3 ◆◆	KMT2D ◆◆	PAX5 ◆◆	RHOA ◆◆	U2AF1 ◆◆
BLM ◆◆	CYLD ◆◆	FGFR4 ◆◆	KRAS ◆◆	PAX8 ◆◆	RICTOR ◆◆	UBE2A ◆
BMPR1A ◆◆	CYP1A1 ◆	FH ◆◆	LCK ◆◆	PBRM1 ◆◆	RNF43 ◆◆	UBE2K ◆◆
BRAF ◆◆	CYP2B6 ◆	FLCN ◆◆	LIG1 ◆◆	PDCD1 ◆◆	ROS1 ◆◆	UBR5 ◆◆
BRCA1 ◆◆	CYP2C19 ◆	FLT1 ◆◆	LIG3 ◆◆	PDCD1LG2 ◆◆	RPPH1 ◆◆	UGT1A1 ◆
BRCA2 ◆◆	CYP2C8 ◆	FLT3 ◆◆	LMO1 ◆◆	PDGFRA ◆◆	RPTOR ◆◆	USH2A ◆◆
BRD4 ◆◆	CYP2D6 ◆◆	FLT4 ◆◆	LRP1B ◆◆	PDGFRB ◆◆	RUNX1 ◆◆	VDR ◆
BRIP1 ◆◆	CYP2E1 ◆	FOXL2 ◆	LYN ◆◆	PDIA3 ◆◆	RUNX1T1 ◆◆	VEGFA ◆◆
BTG1 ◆	CYP3A4 ◆	FOXP1 ◆◆	MALT1 ◆◆	PGF ◆◆	RXRA ◆◆	VEGFB ◆◆
BTG2 ◆	CYP3A5 ◆	FRG1 ◆◆	MAP2K1 ◆◆	PHOX2B ◆◆	SDHA ◆◆	VHL ◆◆
BTX ◆◆	DAXX ◆◆	FUBP1 ◆◆	MAP2K2 ◆◆	PIK3C2B ◆◆	SDHB ◆◆	WT1 ◆◆
BUB1B ◆◆	DCUN1D1 ◆◆	GATA1 ◆◆	MAP2K4 ◆◆	PIK3C2G ◆◆	SDHC ◆◆	XIAP ◆◆
CALR ◆◆	DDR2 ◆◆	GATA2 ◆◆	MAP3K1 ◆◆	PIK3C3 ◆◆	SDHD ◆◆	XPO1 ◆◆
CANX ◆◆	DICER1 ◆◆	GATA3 ◆◆	MAP3K7 ◆◆	PIK3CA ◆◆	SERPINB3 ◆◆	XRCC2 ◆◆
CARD11 ◆◆	DNMT3A ◆◆	GNA11 ◆◆	MAPK1 ◆◆	PIK3CB ◆◆	SERPINB4 ◆◆	ZNF217 ◆◆
CASP8 ◆◆	DOT1L ◆◆	GNA13 ◆◆	MAPK3 ◆◆	PIK3CD ◆◆	SETD2 ◆◆	

Fusion Genes in ACTOnco[®] +

ALK	EGFR	FGFR2	MET	NTRK1	NTRK3	ROS1
BRAF	FGFR1	FGFR3	NRG1	NTRK2	RET	



ACT Onco® +



ACT Genomics (Thailand) Limited



+66 02 105 4670



+66 2103 2261



san.service.thai@actgenomics.com

Taipei | Singapore | Hong Kong | Tokyo | Wales | Bangkok

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