SureSeq myPanel



A Sysmex Group Company

Custom AML NGS panels

Features

Unparalleled coverage uniformity across all content including CEBPA

• Confidently detect AML variants and remove the requirement for supplementary fill-in approaches

Bespoke panels with pre-optimised content

• Create your ideal AML panel and sequence only what's relevant for your AML research

Robust detection of *FLT*3-ITDs and *KMT*2A-PTDs

• Streamline your laboratory workflow with a single NGS assay for comprehensive aberration detection in AML

Complimentary Interpret NGS data analysis software

• Easy-to-use analysis solution for accurate detection of all variants

Introduction

Acute myeloid leukaemia (AML) is the most common type of acute leukaemia in adults. Our understanding of AML has been transformed in recent years to a disease classified largely based on genetic, genomic and molecular characteristics. Key genes implicated in AML progression include *CEBPA*, *NPM*1, *FLT*3 and *KMT*2A (*MLL*) with mutations in multiple additional genes identified in recent research¹.

Choose your perfect AML NGS panel from our range of fully tested and optimised panel content. Simply mix and match the genes or individual exons you require for your research and get the most out of your sequencing runs.

Excellent Coverage Uniformity of the CEBPA Gene

Mutations in the *CEBPA* gene are among the most common molecular alterations in AML. Sequencing of *CEBPA* is challenging due to the presence of repeat regions and the high GC-content of the gene, leading to poor coverage across these regions and potentially missed variants. OGT's expert bait design overcomes these issues and provides exceptional coverage uniformity, even in difficult to sequence genes, enabling reliable detection of variants and eliminating the requirement for supplementary fill-in with Sanger sequencing (Figure 1).

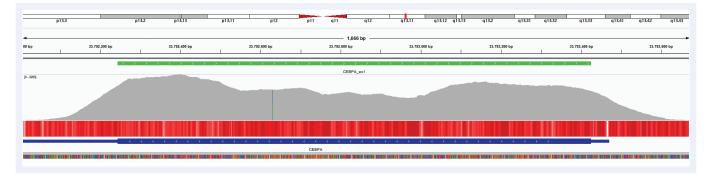


Figure 1: Illustration of the excellent coverage uniformity of the CEBPA gene. Depth of coverage per base (grey). Targeted region (green). Gene coding region as defined by RefSeq (blue). GC percentage (red).



Sophisticated Bait Design Strategies allowing reliable *FLT3*-ITD and *KMT2A*-PTD detection

The most prevalent type of *FLT3* mutations in AML are internal tandem duplications (ITDs). *FLT3*-ITDs are challenging to target because they are by nature repetitive and can be very long. As a result, *FLT3*-ITDs are generally masked in most panel designs, necessitating additional techniques to generate a complete picture of the genetic makeup of AML. To provide optimal results OGT employs sophisticated bait design strategies to generate uniform coverage across, as well as upstream and downstream of the repetitive region, allowing easy detection of *FLT3*-ITDs ranging from a handful of base pairs to >200 bp (Figure 2).



Figure 2: Facilitated by OGT's expert bait design, FLT3-ITDs of various sizes and even regions containing multiple ITDs can be confidently detected. ITD sizes are A 174 bp, B 225 bp, C 195 bp with additional 6 bp, D 120 bp and E 168 bp with additional 69 bp.

Other tandem duplications frequently observed in AML are partial tandem duplications (PTDs) in *KMT2A (MLL)*. Similar to ITDs, *KMT2A*-PTDs are notoriously difficult to detect due to their size, with duplications spanning exons 3 to 9, exons 3 to 10 and exons 3 to 11 being the most commonly found in AML². With OGT's expertise in hybridisation-based panel design, your SureSeq myPanel[™] offers robust detection of all sizes of *KMT2A*-PTDs, alleviating the burden of running multiple assays (Figure 3).



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Figure 3: PTD detected spanning exons 2-8 of KMT2A by OGT's Interpret NGS analysis software.

Complimentary Interpret NGS analysis software

Interpret is OGT's powerful and easy-to-use data analysis solution, facilitating analysis and visualisation of a wide range of somatic variants and structural aberrations. Designed to work seamlessly with all SureSeq[™] panels, you'll be able to accurately detect all SNVs, indels, ITDs and PTDs covered by your SureSeq myPanel Custom AML Panel. Additionally, if you choose to expand your panel further, our Interpret software can reliably detect copy-number variations (CNVs), loss-of-heterozygosity (LOH) and translocations, for example *BCR-ABL*. Following detection, all variants can be readily visualised in the user-friendly variant browser, for an effortless translation of all your AML data into meaningful results.

ASXL1	BCOR	BCORL1	CBLB	CBLC	CEBPA
CUX1	DDX41	DNMT3A	ETV6	FLT3	GATA1
IDH1	IDH2	IKZF1	IRF1	JAK3	KIT
KMT2A	KRAS	NPM1	NRAS	PHF6	RUNX1
SMC1A	TET2	TP53	U2AF1	WT1	

Select from any of the following SureSeq myPanel AML gene or exonic content

Offering comprehensive content covering a wide range of aberrations, you can now design your perfect SureSeq myPanel Custom AML Panel and generate a comprehensive genetic picture of all your AML samples using a single streamlined workflow.



Getting started with your next SureSeq myPanel NGS Custom Cancer panel could not be simpler



If you are looking for an extended myeloid or pan-haem panel, talk to us and let our expertise help you advance your cancer research.

For more information about SureSeq myPanel Custom AML Panels or customisation queries, visit ogt.com/CustomAML or contact us at contact@ogt.com.



Ordering information

UK +44 (0) 1865 856800 US +1 914 467 5285 contact@ogt.com ogt.com

Product	Contents	Cat. No.
SureSeq myPanel NGS Custom AML Panels	Enrichment baits; Interpret Software	Various
SureSeq NGS Library Preparation Complete Solution (16)	Bundle of 1x SureSeq NGS Library Preparation Kit (16), containing adaptors, PCR primers and enzymes, 1x SureSeq NGS Index Kit – Collection A, 1x SureSeq NGS Hyb & Wash Kit (16), 1x Dynabeads M270 Streptavidin (2ml) and 1x AMPure XP beads (10ml). Sufficient for 16 samples	500084
SureSeq NGS Library Preparation Complete Solution (48)	Bundle of 1x SureSeq NGS Library Preparation Kit (48), containing adaptors, PCR primers and enzymes, 1x SureSeq NGS Index Kit – Collection B, 3x SureSeq NGS Hyb & Wash Kit (16), 3x Dynabeads M270 Streptavidin (2ml) and 3x AMPure XP beads (10ml). Sufficient for 48 samples	500085

References

- 1. Döhner et al., Blood 2017; 129(4):424-447
- 2. Steudel et al., Genes Chromosomes Cancer 2003; 37(3):237-51



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What binds us, makes us.

Oxford Gene Technology Ltd., Begbroke Science Park, Woodstock Road, Begbroke, Oxfordshire, OX5 1PF, UK

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