

Empire Genomics' **Multiple Myeloma FISH Panel** is made up of probes for detecting 6 major genetic lesions found in the disease:

1p/1q

On the p arm of chr. 1, loss of tumor suppressor CDKN2C triggers abnormal cell proliferation; on the q arm, extra copies of CKS1B cause genomic breakdown.¹

IGH

Disease-initiating IGH translocations, found in about 40% of MM cases, result in fusions with partner genes that are upregulated by IGH enhancers.²

RB1/ LAMP1

Chromosome 13q deletions are one of the most frequent alterations in MM, and often include loss of RB1 and LAMP1 at 13q14.2 and 13q34, respectively.³

MYC







MYC rearrangements are complex in MM, occurring as translocations, inversions and insertions. Studies show they could be found in up to 50% of patients.⁴

CCND1/ IGH

CCND1 is found fused to IGH in approximately 20% of MM cases. As with other IGH fusions, CCND1/IGH results in overexpression of the IGH fusion partner.⁵

TP53

Found in just under 10% of newly diagnosed MM patients, TP53 deletions result in haploinsufficiency of this important tumor suppressor.⁶

GENE PROBE	LOCATION / STS	DYE COLOR	SKU
1p1q	1p32.3/1q21.3		1p1q-20-GROR
IGH break apart	14q32.33		IGHBA-20-ORGR
RB1/LAMP1	13q14.2/13q34		RB1-LAMP1-20-GR
MYC break apart	8q24.21		MYCBA-20-ORGR
CCND1/IGH fusion	11q13.3/14q32.33		CCND1-IGH-Split-20-ORGR
TP53	17p13.1		TP53-20-OR

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1. Chang H et al. (2010) Bone marrow transplantation 45.1: 117-121.
2. Kim Gna et al. (2014) Genes, Chromosomes and Cancer 53.6: 467-474.
3. Binder M et al. (2017) Blood cancer journal 7.9: e600-e600.
4. Affer M et al. (2014) Leukemia 28.8: 1725-1735.
5. Kuroda J et al. Acta haematologica 120.3: 177-181.
6. Teoh PJ et al. (2014) Leukemia 28.10: 2066-2074.