

SUPPLEMENTS

Supplemental Table 1. 54 gene panel (N = 1 patient (1 sample)): identifies potential tumor-related genomic alterations within 54 cancer-related genes including amplifications in *ERBB2*, *EGFR*, and *MET*. Only non-synonymous alterations were analyzed.

GENES WITH COMPLETE EXON COVERAGE							
<i>ALK</i>	<i>APC</i>	<i>AR</i>	<i>BRAF</i>	18 GENES 3 Copy Number Variations in BOLD			
<i>CDKN2A</i>	<i>EGFR</i>	<i>ERBB2</i>	<i>FBXW7</i>				
<i>KRAS</i>	<i>MET</i>	<i>MYC</i>	<i>NOTCH1</i>				
<i>NRAS</i>	<i>PIK3CA</i>	<i>PTEN</i>	<i>PROC</i>				
<i>RB1</i>	<i>TP53</i>						
GENES WITH CRITICAL EXON COVERAGE			36 GENES				
<i>ABL1</i>	<i>AKT1</i>	<i>ATM</i>	<i>CDH1</i>	<i>CSF1R</i>	<i>CTNNB1</i>		
<i>ERBB4</i>	<i>EZH2</i>	<i>FGFR1</i>	<i>FGFR2</i>	<i>FGFR3</i>	<i>FLT3</i>		
<i>GNA11</i>	<i>GNAQ</i>	<i>GNAS</i>	<i>HNF1A</i>	<i>HRAS</i>	<i>IDH1</i>		
<i>IDH2</i>	<i>JAK2</i>	<i>JAK3</i>	<i>KDR</i>	<i>KIT</i>	<i>MLH1</i>		
<i>MPL</i>	<i>NPM1</i>	<i>PDGFRA</i>	<i>PTPN11</i>	<i>RET</i>	<i>SMAD4</i>		
<i>SMARCB1</i>	<i>SMO</i>	<i>SRC</i>	<i>STK11</i>	<i>TERT</i>	<i>VHL</i>		

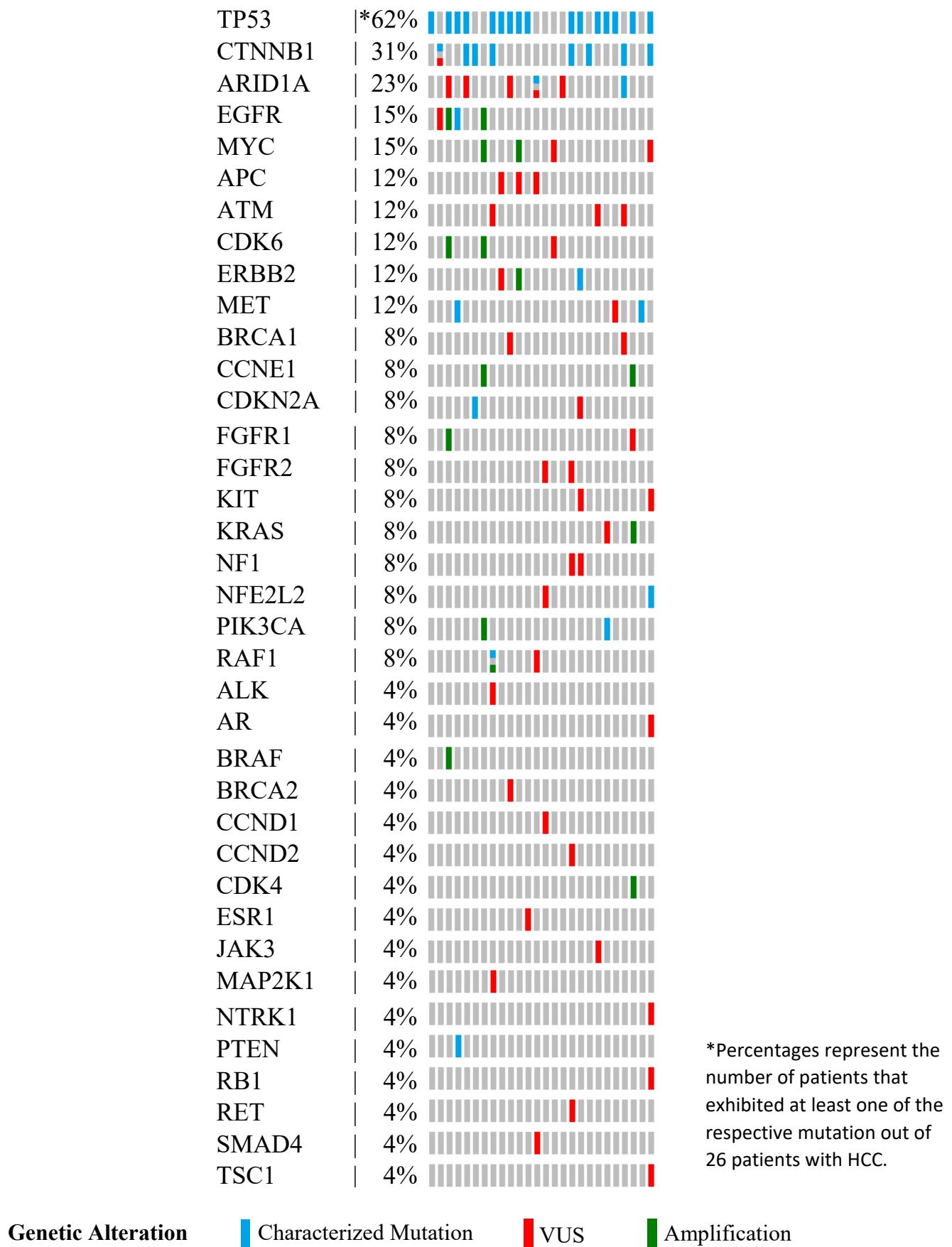
Supplemental Table 2. 68 gene panel (N = 23 patients (28 samples)), comprising amplifications in 16 genes as well as some fusions and indels. Only non-synonymous alterations were analyzed.

POINT MUTATIONS			AMPLIFICATIONS		FUSIONS	INDELS	
<i>AKT1</i>	<i>ALK</i>	<i>APC</i>	<i>AR</i>	<i>AR</i>	<i>ALK</i>	<i>EGFR</i> exon 19 deletions	
<i>AFAR</i>	<i>ARID1A</i>	<i>ATM</i>	<i>BRAF</i>	<i>BRAF</i>	<i>RET</i>	<i>EGFR</i> exon 20 insertions	
<i>BRCA1</i>	<i>BRCA2</i>	<i>CCND1</i>	<i>CCDN2</i>	<i>CCNE1</i>	<i>ROS1</i>		
<i>CCNE1</i>	<i>CDH1</i>	<i>CDK4</i>	<i>CDK6</i>	<i>CDK4</i>	<i>NTRK1</i>		
<i>CDKN2A</i>	<i>CDKN2B</i>	<i>CTNNB1</i>	<i>EGFR</i>	<i>CDK6</i>			
<i>ERBB2</i>	<i>ESR1</i>	<i>EZH2</i>	<i>FBXW7</i>	<i>EGFR</i>			
<i>FGFR1</i>	<i>FGFR2</i>	<i>FGFR3</i>	<i>GATA3</i>	<i>ERBB2</i>			
<i>GNA11</i>	<i>GNAQ</i>	<i>GNAS</i>	<i>HNF1A</i>	<i>FGFR1</i>			
<i>HRAS</i>	<i>IDH1</i>	<i>IDH2</i>	<i>JAK2</i>	<i>FGFR2</i>			
<i>JAK3</i>	<i>KIT</i>	<i>KRAS</i>	<i>MAP2K1</i>	<i>KIT</i>			
<i>MAP2K2</i>	<i>MET</i>	<i>MLH1</i>	<i>MPL</i>	<i>KRAS</i>			
<i>MYC</i>	<i>NF1</i>	<i>NFE2L2</i>	<i>NOTCH1</i>	<i>MET</i>			
<i>NPM1</i>	<i>NRAS</i>	<i>NTRK1</i>	<i>PDGFRA</i>	<i>MYC</i>			
<i>PIK3CA</i>	<i>PTEN</i>	<i>PTPN11</i>	<i>RAF1</i>	<i>PDGFRA</i>			
<i>RET</i>	<i>RHEB</i>	<i>RHOA</i>	<i>RIT1</i>	<i>PIK3CA</i>			
<i>ROS1</i>	<i>SMAD4</i>	<i>SMO</i>	<i>SRC</i>	<i>RAF1</i>			
<i>STK11</i>	<i>TERT</i>	<i>TP53</i>	<i>VHL</i>	*Complete exon coverage for genes in bold			

Supplemental Table 3: 70 gene panel (N = 3 patients (3 samples))

Complete Exon Sequencing									
Point Mutations (SNVs) (70 Genes)						Amplifications (CNVs) (18 Genes)		Fusions (6 Genes)	Indels (3 Genes)
<i>AKT1</i>	<i>ALK</i>	<i>APC</i>	<i>AR</i>	<i>ARAF</i>	<i>ARID1A</i>	<i>AR</i>	<i>BRAF</i>	<i>ALK</i>	<i>EGFR*</i>
<i>ATM</i>	<i>BRAF</i>	<i>BRCA1</i>	<i>BRCA2</i>	<i>CCND1</i>	<i>CCND2</i>	<i>CCND1</i>	<i>CCND2</i>	<i>FGFR2</i>	<i>ERBB2*</i>
<i>CCNE1</i>	<i>CDH1</i>	<i>CDK4</i>	<i>CDK6</i>	<i>CDKN2A</i>	<i>CDKN2B</i>	<i>CCNE1</i>	<i>CDK4</i>	<i>FGFR3</i>	<i>MET**</i>
<i>CTNNB1</i>	<i>EGFR</i>	<i>ERBB2</i>	<i>ESR1</i>	<i>EZH2</i>	<i>FBXW7</i>	<i>CDK6</i>	<i>EGFR</i>	<i>NTRK1</i>	
<i>FGFR1</i>	<i>FGFR2</i>	<i>FGFR3</i>	<i>GATA3</i>	<i>GNA11</i>	<i>GNAQ</i>	<i>ERBB2</i>	<i>FGFR1</i>	<i>RET</i>	
<i>GNAS</i>	<i>HNF1A</i>	<i>HRAS</i>	<i>IDH1</i>	<i>IDH2</i>	<i>JAK2</i>	<i>FGFR2</i>	<i>KIT</i>	<i>ROS1</i>	
<i>JAK3</i>	<i>KIT</i>	<i>KRAS</i>	<i>MAP2K1</i>	<i>MAP2K2</i>	<i>MET</i>	<i>KRAS</i>	<i>MET</i>		
<i>MLH1</i>	<i>MPL</i>	<i>MYC</i>	<i>NF1</i>	<i>NFE2L2</i>	<i>NOTCH1</i>	<i>MYC</i>	<i>PDGFRA</i>		
<i>NPM1</i>	<i>NRAS</i>	<i>NTRK1</i>	<i>PDGFRA</i>	<i>PIK3CA</i>	<i>PTEN</i>	<i>PIK3CA</i>	<i>RAF1</i>		
<i>PTPN11</i>	<i>RAF1</i>	<i>RB1</i>	<i>RET</i>	<i>RHEB</i>	<i>RHOA</i>				<i>*exons 19 & 20 **exon 14 skipping</i>
<i>RIT1</i>	<i>ROS1</i>	<i>SMAD4</i>	<i>SMO</i>	<i>SRC</i>	<i>STK11</i>				
<i>TERT</i>	<i>TP53</i>	<i>TSC1</i>	<i>VHL</i>						

Supplemental Figure 1: Oncoprint of genomic alterations at the molecular level in 26 HCC patients



Genetic Alteration

Characterized Mutation

VUS

Amplification