Figure S1. Sequence read distribution across 67 amplicons generated from the 40 FFPE specimens, normalized to 300,000 reads per sample. (A) Distribution of the average coverage of each amplicon. Values are expressed as the mean ± standard deviation. (B) The number of amplicons with a given read depth that is sorted in bins of 1,000 reads. The grey bars represent the number of target amplicons within a particular read depth and the black line represents the percentage of target amplicons greater than or equal to the read depth.

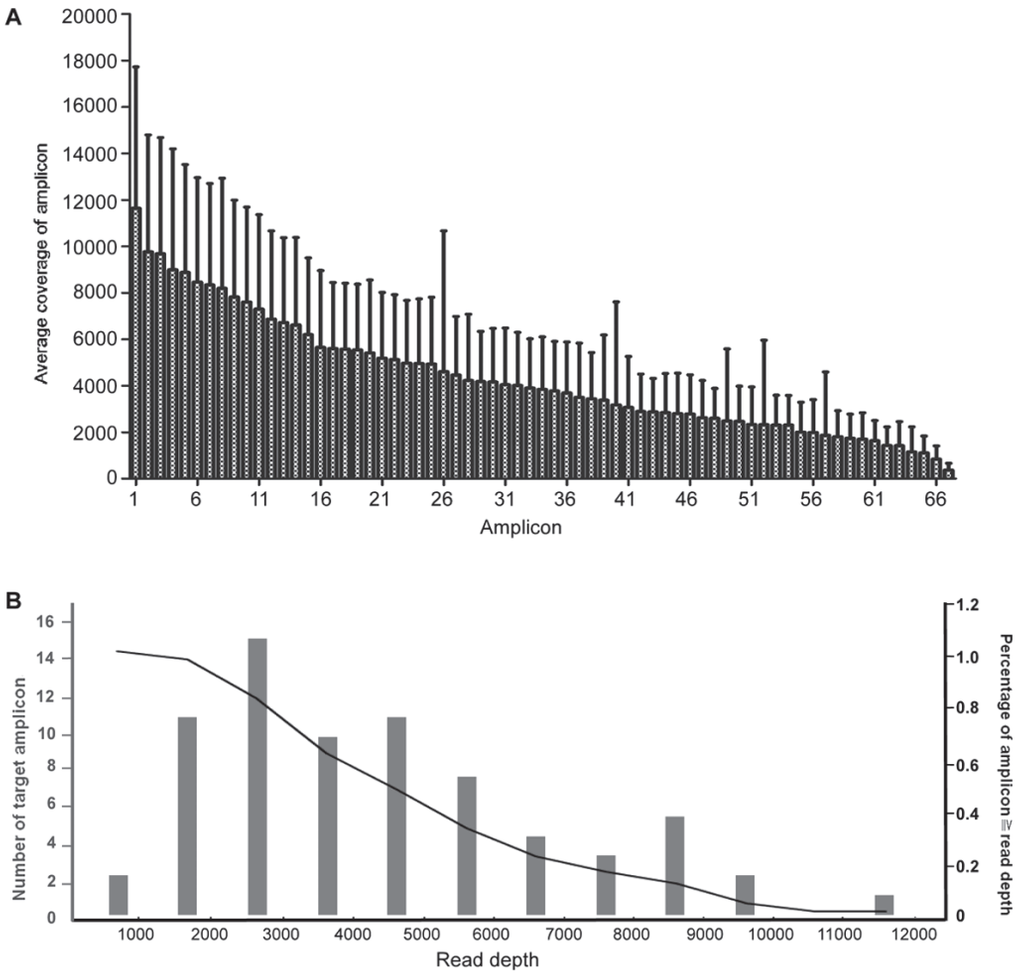

Table SI. Summary of the tested genes' functions.

| Gene name | Description[a]: Cancer type (mutation frequency, %), significance of the gene mutation. [other types of gene changes] |
|---|---|
| KRAS | CRC (36-40%), mutated tumors are resistant to EGFR antibodies cetuximab[b] and panitumumab[b]. KRAS inhibitor AMG-510 may emerge as a potential therapeutic for KRAS-mutated tumors in clinical trials. |
| NRAS | CRC (1-6%), mutated tumors are resistant to EGFR antibodies cetuximab[b] and panitumumab[b]. |
| BRAF | CRC (5-15%), poor prognostic marker and mutated tumors may be resistant to EGFR antibody therapy. |
| PIK3CA | CRC (10-30%), poor prognostic marker and mutated tumors may be resistant to EGFR antibody therapy. [sensitive to the PI3K inhibitor Alpelisib[b] in BC.] |
| KIT | GIST (~85%), prognostic marker and variable sensitivity to KIT inhibitor imatinib[b] based on different mutations. |
| PDGFRA | GIST (~5%), variable sensitivity to KIT inhibitor imatinib[b] based on different mutations. |
| EGFR | NSCLC (10-35%), associated with EGFR TKIs (crizotimab[b]) resistance or sensitivity based on different mutations. |
| ERBB2 | NSCLC (2-8%), response to anti-HER2 TKI or antibodies is unknown; EBRR2 activing mutation may mediate resistance to EGFR antibodies in CRC-PDX. [ERBB2 amplified GC and BC response to inhibitor herceptin[b].] |
| DDR2 | SCC of lung (2.5-3.8%), TKI dasatinib emerged as a novel therapeutic option for DDR2-mutated tumors in clinical trials. |
| ALK | Neuroblastoma (8-9%), as a potential therapeutic target. [ALK fusions are identified at different frequencies in NSCLC, ALCL etc., and patients with ALK fusions showed good response to ALK inhibitor therapy.] |
| RET | MTC (sporadic 50%), response to non-specific or specific RET kinase inhibitor. [10-20% papillary thyroid cancers harbor RET fusion.] |
| SMO | BCC (12%), mutated tumor resistant to the SMO inhibitor vismodegib[b]. |
| TSC1 | Bladder cancers (7-12%), sensitive to the mTOR inhibitor everolimus[b]. |
| FLT3 | AML (24%), poor prognosis marker and responsive to kinase inhibitor midostaurin[b]. |
| NPM1 | AML (8%), FLT3/NPM1 double mutations is a favorable prognostic marker; NPM1 may be considered as a potential therapeutic target. |
| DNMT3A | AML (20%), poor prognostic marker and may be considered as a potential therapeutic target. |
| ABL1 | CML, ~95% CML has BCR-ABL1 fusion, 30-40% patients acquire resistance to ABL TKI imatinib[b] as point mutation occurs in ABL1. Mutation-specific treatment decision recommendations were adopted by the NCCN. |

[a]List of the common cancers harboring the genetic mutation; the data are referenced from MyCancerGenome database (https://www.mycancergenome.org/). [b]Approved by the Food and Drug Administration of the US. CRC, colorectal cancer; GIST, gastrointestinal stromal tumor; NSCLC, non-small cell lung cancer; TKI, tyrosine kinase inhibitors; PDX, patient-derived xenograft; GC, gastric cancer; BC, breast cancer; SCC, squamous cell carcinoma; ALCL, anaplastic large cell lymphoma; MTC, medullary thyroid cancer; PCT, papillary thyroid cancer; BCC, basal cell carcinoma; AML, acute myeloid leukemia; CML, chronic myeloid leukemia; NCCN, National Comprehensive Cancer Network. KRAS, KRAS proto-oncogene, GTPase; NRAS, NRAS proto-oncogene; BRAF, B-Raf proto-oncogene, serine/threonine kinase; PIK3CA, phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit alpha; KIT, KIT proto-oncogene, receptor tyrosine kinase; PDGFRA, platelet derived growth factor receptor alpha; EGFR, epidermal growth factor receptor; ERBB2, erb-b2 receptor tyrosine kinase 2; DDR2, discoidin domain receptor tyrosine kinase 2; GTPase; ALK, ALK receptor tyrosine kinase; RET, ret proto-oncogene; SMO, smoothened, frizzled class receptor; TSC1, TSC complex subunit 1; FLT3, fms related receptor tyrosine kinase 3; NPM1, nucleophosmin 1; DNMT3A, DNA methyltransferase 3 alpha; ABL1, ABL proto-oncogene 1.