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PATIENT: XXXXXXXXXXXXX			TEST REF: GNL-NL-XXXXX
TEST NUMBER: G-NL-XXXXX	COLLECTED: 00-XXX-2023		PRACTITIONER: XXXXXXXXXXXX
GENDER: XXXXXX	RECEIVED: 00-XXX-2023		
AGE: XX	TESTED: 00-XXX-2023		XXXXXXXXXXXXXXXXXXXX

TEST NAME: Cancertrack™

54 Clinical trials available: Please refer to page no. 13 - 22

Report Highlights

Indications	USFDA Approved*/ NCCN Recommended*	Off Label Therapy*
NRAS p.A59T (MAF 0.36% at 129371X)	☒ Cetuximab ☒ Panitumumab	☒ Necitumumab
NRAS p.A59T (MAF 0.36% at 129371X) **MAP2K1 p.F53S** (MAF 0.27% at 79852X)	⊟ None	☑ Trametinib ☑ Binimetinib ☑ Cobimetinib ☑ Selumetinib

☑ SOC Drugs with Benefit ☑ Off Label Drugs with Benefit ☒ Drugs without Clinical Benefit / with Potential Resistance

MAF: Mutant Allele Frequency; SOC: Standard of Care; NCCN: National Comprehensive Cancer Network - Colon cancer and Rectal cancer

* The USFDA approval or NCCN recommendation may not be for the detected biomarker or alteration. The association of the detected biomarker or alteration and the drug may be based only on the literature evidence.

Longitudinal Monitoring Biomarkers

Biomarkers	Result	Biomarkers	Result
Highest mutant allele frequency (HMAF)	0.73%	Number of CTCs detected	3 CTCs/ ml

Disease Relevant Findings

Biomarkers	Result	Biomarkers	Result
NRAS p.A59T	Mutation detected	KRAS	No mutations detected
BRAF	No mutations detected	ERBB2/HER2	No alterations detected
RET	No fusions detected	NTRK1/3	No fusions detected

Summary of other Genomic Alterations

Gene	Alteration Type (SNAs / Indels / CNAs/ Fusion)	Variant Classification	Therapeutic / Clinical Significance
APC	p.R1450* (MAF 0.62% at 58228X)	Pathogenic	Refer to page no. 3
TP53	p.L330R (MAF 0.27% at 104366X) p.V172A (MAF 0.11% at 166267X)	Pathogenic	Refer to page no. 4
TP53	p.D184G (MAF 0.15% at 166785X) p.Q104H (MAF 0.73% at 66335X)	Likely Pathogenic	Refer to page no. 4
TP53	p.P177P (MAF 0.11% at 166280X)	VUS	---

SNA: Single Nucleotide Alteration; CNA: Copy Number Alteration; INDELS: Insertion / Deletion; CTC: Circulating Tumor Cells; VUS: Variant of Unknown/Uncertain Significance

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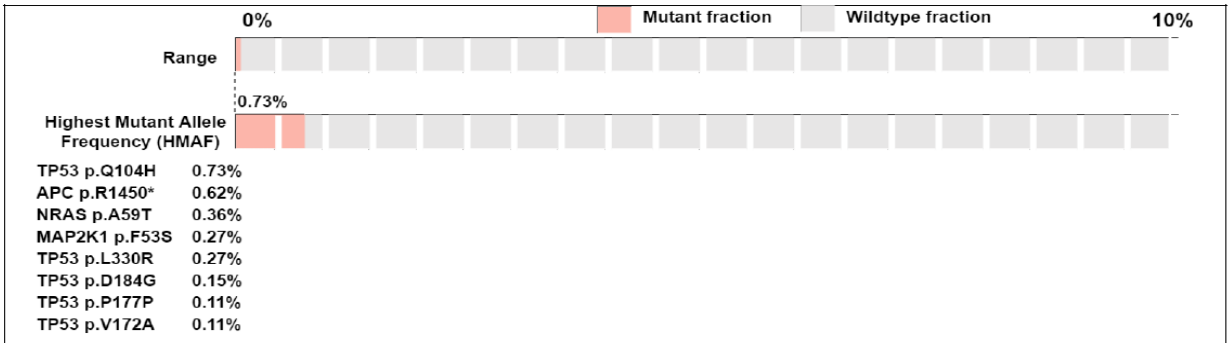
Cell Free Nucleic Acids : Somatic Genome Alterations

Genomic Findings

Highest Mutant Allele Frequency



HMAF of 0.73% was detected in the cell free nucleic acids isolated from patient's plasma.

Genomic Findings
Single Nucleotide Alterations / Indels / Copy Number Alterations / Fusion

Markers (Transcript ID)	Variant	Result	Category
NRAS (NM_002524.5)	c.175G>A p.A59T; [p.(Ala59Thr)]	Detected	Tier I (Level A)

Interpretation: Mutations in NRAS gene are commonly reported in colorectal cancer. NRAS mutations in exons 2 and 3 are associated with distant metastasis and shorter overall survival in these patients (Schirripa et al., 2015; Guo et al., 2017; Kuhn et al., 2021).

Multiple studies have shown that patients with tumors harboring mutations in KRAS or NRAS exons 2, 3, or 4 predicts resistance to anti-EGFR antibody therapies, Cetuximab, Panitumumab and Necitumumab, as well as anti-EGFR tyrosine kinase inhibitors (Glynne-Jones et al., 2010; Douillard et al., 2013; Tejpar et al., 2014; Stintzing et al., 2014; Ciardiello et al., 2014; Peeters et al., 2014; Chan et al., 2017; Lovly et al., 2017). Also, mutations in NRAS gene are suggestive of less responsiveness to HER2-directed monotherapies Trastuzumab, Pertuzumab, Lapatinib, Neratinib and Tucatinib in HER2 positive tumors (Lubner et al., 2017; Fakih et al., 2018; Chen et al., 2020; Shimozaki et al., 2022).

Activating NRAS mutations are found to increase PI3K/AKT as well as RAS/RAF/MAPK signalling pathways and therefore is suggestive of potential therapeutic benefit from MEK inhibitors, Trametinib, Binimetinib, Cobimetinib and Selumetinib (Ascierto et al., 2013; Johnson and Puzanov, 2015; Kiessling et al., 2016; Vu and Aplin, 2016; Boespflug et al., 2017; Dummer et al., 2017; Han et al., 2018; Sarkisian and Davar, 2018; Ogino et al., 2018; Grisham et al., 2019).

Trametinib, used alone or with Dabrafenib is USFDA approved for the treatment of BRAF V600E positive anaplastic thyroid and non-small cell lung cancer as well as BRAF V600E or V600K positive unresectable or metastatic melanoma.
In a phase I dose-escalation trial, Trametinib showed 4 partial response in 39 BRAF wild-type melanoma patients. In BRAF wild-type cohort, 2 out of 7 patients with NRAS mutation showed stable disease, one of whom received treatment for 48 weeks (Falchook et al., 2012).
In a study of Cetuximab in combination with Trametinib in patients with KRAS mutant tumors (n=9) including colorectal cancer, the combination showed significantly longer progression-free survival as compared to previous lines of treatments in KRAS exon 2 mutation positive patients (Ledys et al., 2019).

Binimetinib in combination with Encorafenib is USFDA approved for the treatment of BRAF V600E or V600K positive unresectable or metastatic melanoma.
In the multicenter NCI-MATCH trial, Binimetinib in NRAS-mutated cancers (n=47) showed objective response rate of 2.1% (1 of 47

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patients), and the median progression free survival of 3.5 months. 2 patients with NRAS codon 61 mutated colorectal cancer had stable disease for at least 12 months (Cleary et al., 2021).
In a case study, treatment of Binimetinib, Hydroxychloroquine and Bevacizumab demonstrated 17% reduction in the size of tumor lumps in a patient with KRAS p.G12D-mutated colorectal cancer (Orlov et al., 2020).

Cobimetinib is a USFDA approved kinase inhibitor indicated for the treatment of patients with unresectable or metastatic melanoma with a BRAF V600E or V600K mutation, in combination with Vemurafenib.
In a phase Ib trial, Belvarafenib in combination with Cobimetinib showed partial response in 5 of 13 NRAS mutated melanoma patients with response rate of 38.5% (Shin et al., 2021).
In a study, treatment of Cobimetinib and Atezolizumab in 22 KRAS mutant and 1 KRAS wild type colorectal cancer patients was well tolerated and showed overall response rate of 17% (Bendell et al., 2016)

Selumetinib is USFDA approved for the treatment of pediatric patients with neurofibromatosis type 1.
In a clinical study, treatment with Selumetinib resulted in increase in iodine uptake and retention in 5 of 5 NRAS mutated and 4 of 9 BRAF mutated thyroid cancer (Ho et al., 2013).
In a phase II study, Selumetinib plus Irinotecan as second-line therapy in patients with exon 2 KRAS mutated colorectal cancer, demonstrated partial response in 3 and stable disease for ≥4 weeks, (including three >1 year) in 16 of 31 evaluable patients (Hochster et al., 2015).

However, the efficacy of these drugs in NRAS mutated colorectal cancer is not well evaluated.

NRAS mutations in advanced melanoma correlate with increased benefit from immune-based therapies compared to other genetic subtypes (Johnson et al., 2015).

NRAS p.A59T lies within the GTP-binding domain of the NRAS protein. It has been predicted to lead to a gain of NRAS function based on the effects of HRAS A59T, which results in increased nucleotide exchange rate and transformation of cultured cells (Lacal et al., 1986; Castellano and Santos, 2011). In silico analysis also predicts this variant to be a gain-of- function mutation. It is reported in tumors of pancreas, autonomic ganglia, skin, haematopoietic and lymphoid system.

This is an N-ras oncogene encoding a membrane protein that shuttles between the Golgi apparatus and the plasma membrane. This shuttling is regulated through palmitoylation and depalmitoylation by the ZDHHC9-GOLGA7 complex. The encoded protein, which has intrinsic GTPase activity, is activated by a guanine nucleotide-exchange factor and inactivated by a GTPase activating protein. Mutations in this gene have been associated with somatic rectal cancer.

Markers (Transcript ID)	Variant	Result	Category
APC (NM_000038.6)	c.4348C>T p.R1450*; [p.(Arg1450Ter)]	Detected	Tier I (Level B)

Interpretation: Inactivating APC mutations are reported frequently in colorectal cancer and are associated with an adverse prognosis (Aoki and Taketo, 2007; Kwong and Dove, 2009; Van den Broek et al., 2016; Zhang and Shay, 2017; Liu et al., 2018; Aghabozorgi et al., 2019). APC loss stabilizes beta-catenin and constitutively activates the WNT pathway even in the absence of a WNT signal and therefore it is suggestive of potential benefit from non-conventional drugs, Quercetin and Celecoxib (Rubinfeld et al, 1997; Hankey et al, 2018; Neamtu et al., 2022).

Pre-clinical studies reported that Quercetin inhibits cancer growth through inhibition of Wnt/beta-catenin signaling pathway (Shan et al., 2009; Amado et al., 2011).

Celecoxib is one of the most commonly used non-steroidal anti-inflammatory drugs (NSAIDs), which has chemo-preventive activity against cancers. It acts by down-regulating the Wnt pathway activity (Gong et al., 2012; Egashira et al., 2017; Huang et al., 2017).

APC p.R1450* results in a premature truncation of the APC protein at amino acid 1450 of 2843. It results in reduced suppression of beta-catenin activity in culture (Azzopardi et al., 2008), and therefore, is predicted to lead to a loss of APC protein function. In silico analysis also predicts this variant to be a loo-of-function mutation. It is reported in tumors of large intestine, stomach, small intestine, endometrium and pancreas.

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APC gene encodes a tumor suppressor protein that acts as an antagonist of the WNT signaling pathway. It is also involved in other processes including cell migration and adhesion, transcriptional activation, and apoptosis. This protein also helps ensure that the number of chromosomes in a cell is correct following cell division.

Markers (Transcript ID)	Variant	Result	Category
MAP2K1 (NM_002755.4)	c.158T>C p.F53S; [p.(Phe53Ser)]	Detected	Tier II (Level D)

Interpretation: Mutations in MAP2K1 gene are reported in colorectal cancer (Kim et al., 2017; Chuang et al, 2021). Activating mutations in MAP2K1/MEK1 gene are suggestive of potential benefit from MEK inhibitors, Trametinib, Binimetinib, Cobimetinib and Selumetinib (Brown et al., 2014; Papapanagiotou et al., 2017; Homicsko et al., 2019; Drenner et al., 2021).

Kindly refer to USFDA labels and/or studies of these drugs mentioned earlier.

In a case study, Trametinib showed modest response after 6 months of treatment in MAP2K1 mutated hairy cell leukemia patient (Andritsos et al., 2018).
However, the efficacy of these drugs in MAP2K1 p.F53S mutated colorectal cancer is not well evaluated.

MAP2K1 mutations are known to be potential mechanism of acquired resistance to single agent EGFR targeted monoclonal antibodies, Cetuximab, Panitumumab and Necitumumab in metastatic colorectal cancer (CRC) and has also been reported to promote acquired resistance to BRAF and MEK inhibitors in KRAS/ BRAF mutated cancers (Russo et al., 2016; Kim et al., 2017; Knebel et al., 2020; Drenner et al., 2021).

MAP2K1 p.F53S lies within the negative regulatory region of the MAP2K1 protein (Waterfall et al., 2014). It demonstrates phosphorylation profile similar to wild-type MAP2K1 (Kinoshita-Kikuta et al., 2019), and results in increased ERK phosphorylation in cell culture (Delaney et al., 2002; Rodriguez-Viciana et al., 2006). In silico analysis predicts this variant to be a gain-of-function mutation. It is reported in tumors of haematopoietic and lymphoid system.

The MAP2K1 gene encodes for MEK1 protein kinase. It is a member of the dual specificity protein kinase family, which acts as a mitogen-activated protein (MAP) kinase kinase. It lies upstream of MAP kinases and stimulates the enzymatic activity of MAP kinases upon wide variety of extra- and intracellular signals. As an essential component of MAP kinase signal transduction pathway, this kinase is involved in many cellular processes such as proliferation, differentiation, transcription regulation and development.

Markers (Transcript ID)	Variant	Result	Category
TP53 (NM_000546.5)	c.515T>C p.V172A; [p.(Val172Ala)] c.551A>G p.D184G; [p.(Asp184Gly)] c.312G>T p.Q104H; [p.(Gln104His)] c.989T>G p.L330R; [p.(Leu330Arg)]	Detected	Tier I (Level B)

Interpretation: Approximately half of all colon cancers show TP53 gene mutations, with higher frequencies observed in distal colon and rectal tumors and lower frequencies in proximal tumors and those with the microsatellite instability or methylator phenotypes (Iacopetta, 2003; Sakai et al., 2016; Michel et al., 2021). Dysregulation of TP53 tumor suppressor gene is one of the most frequent events contributing to the transformation of colorectal cancer (CRC), as well as the aggressive and metastatic features of CRC. Also, CRC patients with mutant TP53 appear to be more chemo resistant than those with wild type TP53 (Li et al., 2015; Chow et al., 2016).

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TP53 p.V172A, p.D184G and p.Q104H lie within DNA binding domain of the TP53 protein (Freed-Pastor and Prives, 2012). In silico analysis predicts these variants to be loss-of-function mutations. TP53 p.L330R lies within the tetramerization domain of the TP53 protein (Kamada et al., 2011). It results in decreased TP53 tetramerization and loss of transcriptional activity in cell culture (Imagawa et al., 2009; Lang et al., 2014). TP53 p.V172A is reported in tumors of prostate, large intestine, skin, haematopoietic and lymphoid systems. TP53 p.D184G is reported in tumors of large intestine. TP53 p.Q104H is reported in tumors of adrenal gland. TP53 p.L330R is reported in tumors of pleura, liver, pancreas, upper aerodigestive tract and breast.

The TP53 gene provides instructions for making a protein called tumor protein p53 (or p53). This protein acts as a tumor suppressor, which means that it regulates cell division by keeping cells from growing and dividing too fast or in an uncontrolled way. Because p53 is essential for regulating cell division and preventing tumor formation, it has been nicknamed the "guardian of the genome".

Circulating Tumor Cells Enumeration

CTCs

Circulating tumor cells (CTCs): **DETECTED**

No. of CTCs: **3 CTCs/ ml peripheral blood**

CTCs are defined as CK+, EPCAM+, CD45- cells.

Interpretation

3 CTCs/ ml peripheral blood detected in the submitted sample.

Recommendation

Circulating tumor cell enumeration may be performed every 8 to 12 weeks to monitor disease status in consultation with the treating physician.

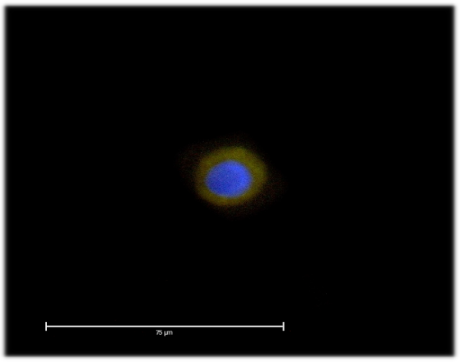

Fig 1: Fluorescent microscopic image of CTC

Cell Free Nucleic Acids Analysis

Variant Allele Fraction And Coverage

Variant (Transcript ID)	Genomic co-ordinates	Allele fraction	Coverage (X)
NRAS (NM_002524.5) c.175G>A, p.A59T	chr1:115256536C>T	0.36	129371
APC (NM_000038.6) c.4348C>T, p.R1450*	chr5:112175639C>T	0.62	58228
MAP2K1 (NM_002755.4) c.158T>C, p.F53S	chr15:66727442T>C	0.27	79852
TP53 (NM_000546.5) c.989T>G, p.L330R	chr17:7576857A>C	0.27	104366
TP53 (NM_000546.5) c.515T>C, p.V172A	chr17:7578415A>G	0.11	166267
TP53 (NM_000546.5) c.551A>G, p.D184G	chr17:7578379T>C	0.15	166785
TP53 (NM_000546.5) c.312G>T, p.Q104H	chr17:7579375C>A	0.73	66335
TP53 (NM_000546.5) c.531C>T, p.P177P	chr17:7578399G>A	0.11	166280

Due to suboptimal coverage or no sequence, the presence or absence of variants contained within certain target regions of the gene listed below could not be meaningfully assessed.
MET

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Criteria For Classification of Somatic Variants

Analysis Criteria

The criteria/guidance used in this report is in accordance with the guidelines provided by the American College of Medical Genetics and Genomics (ACMG) for the interpretation and reporting of sequence variants in cancer. Somatic sequence variations are categorized into four tiers based on their clinical significance (Li et al, 2017).

- **Tier I:** Variants/biomarkers with strong clinical significance (therapeutic, prognostic and/or diagnostic)
  - **Level A evidence:** FDA approved therapies or standard guidelines for a specific tumor type.
  - **Level B evidence:** Statistically significant studies with consensus for specific tumor type.
- **Tier II:** Biomarkers with potential clinical significance (therapeutic, prognostic and/or diagnostic)
  - **Level C evidence:** FDA approved therapies or standard guidelines for a different tumor type (off-label use of the drug). An inclusion criteria for clinical trials.
  - **Level D evidence:** No consensus among different studies.
- **Tier III:** Biomarker whose association with cancer is not evident from available literature and is not frequently present in general population.
- **Tier IV:** Biomarker whose association with cancer has not been reported till date and is frequently present in general population. This category of variants is not included in this report as per guidelines.

Genes Analyzed

Gene List

SNV Genes:

AKT1	ALK	APC	AR	ARAF	BRAF	CHEK2	CTNNB1	DDR2	EGFR
ERBB2	ERBB3	ESR1	FBXW7	FGFR1	FGFR2	FGFR3	FGFR4	FLT3	GNA11
GNAQ	GNAS	HRAS	IDH1	IDH2	KIT	KRAS	MAP2K1	MAP2K2	MET
MTOR	NRAS	NTRK1	NTRK3	PDGFRA	PIK3CA	PTEN	RAF1	RET	ROS1
SF3B1	SMAD4	SMO	TP53						

Fusion Genes:

ALK	BRAF	ERG	ETV1	FGFR1	FGFR2	FGFR3	MET	NTRK1	NTRK3
RET	ROS1								

CNV Genes:

CCND1	CCND2	CCND3	CDK4	CDK6	EGFR	ERBB2	FGFR1	FGFR2	FGFR3
MET	MYC								

Methods and Limitations

Methods

Cell free nucleic acids analysis:
Cell free nucleic acids were analyzed for mutation and fusion detection using semiconductor based Next Generation Sequencing technology. Cell free nucleic acids extracted from the plasma of submitted specimen was subjected to target enrichment by multiplex PCR amplification using panel of genes (see gene list in the 'Genes analysed section'). Enriched DNA sequences were ligated with platform specific adaptor molecules and were sequenced on using semiconductor P1 chip. The minimum average depth was 17000x for gene panel analyzed. High quality sequencing data (proportion Q20 bases ≥ 75%) was analyzed using a customized in-house pipeline DCGL NGS Bioinformatics Pipeline vS11.11 designed to accurately detect the rare somatic variants.

Lower limit of detection of the mutations targeted is 0.1% and variants present below 0.1% may not be detectable with this assay, whereas analytical sensitivity is 97.06% and specificity is 100% for SNV, CNV and Fusion.

A negative test result does not exclude the possibility of mutations being present in the test sample probably due to the reads representing minor allele fraction is below the detectable limit of the assay or other limiting technical / analytical factors.

The clinical sensitivity of most assays for detection of mutant cell free nucleic acids is limited as compared with tumor tissue-based

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testing. This may result from a high ratio of normal to tumor DNA or excess degradation of cell free nucleic acids or may simply reflect the biologic heterogeneity of solid tumors, some of which may shed abundant nucleic acid into the circulation and others that may not. Tumor type, size, disease stage, sites of metastasis, histologic grade, or other features may also affect levels, however, much remains to be elucidated.

This test does not detect variants in gene other than tested. Cancertrack is limited in detecting the epigenetic factors, mutations in repetitive or high GC rich regions. Rare and novel mutations may be clinically uncharacterized.

CTCs enumeration:
Enriched CTCs from the submitted peripheral blood were labelled with EPCAM, Cytokeratin and CD45 antibodies and analyzed by High content imaging platform.
Analytical Validation of this assay shown sensitivity of 99.99% and specificity 99.99%

About CTCs
CTC detection is a promising prognostic tool in both primary and metastatic setting.

CTCs are rare cells in a background of 10^6 -10^7 nucleated blood cells.

Evaluation of CTCs at any time during the course of therapy allows assessment of patient prognosis and is predictive of progression-free survival and overall survival. Circulating tumor cells (CTCs) in the blood stream play a critical role in establishing metastasis.

As an adjunct to standard monitoring methods, monitoring patients with the circulating tumor cell test can help to assess patient's status based on real-time prediction. Enumeration of the number of circulating tumor cells (CTCs) before and during treatment helps predicting response to chemotherapy. Throughout therapy, CTC testing can be used to monitor a patient's status to understand response to the given therapy is favorable or unfavorable at any given time.

Circulating tumor cell test results should be used in conjunction with a clinical information derived from other diagnostic tests, physical examination and complete medical history, in consultation with treating oncologist.

Information to Patients
This is a Laboratory developed test, and its performance characteristics were determined by Datar Cancer Genetics UK Private Limited, United Kingdom. It has not been cleared or approved by the U.S. Food and Drug Administration. This Laboratory is registered under the Clinical Laboratory Improvement Amendments (CLIA)-USA to perform high complexity clinical laboratory testing.

The processing of samples for Molecular Genetics and Cell Culture analysis is carried out at our Laboratory - Datar Cancer Genetics UK Private Limited, United Kingdom.

The analysis of the generated data as well as the preparation of Reports is carried out by our partner laboratory - Datar Cancer Genetics Private Limited, Nasik, India.

This facility is certified by the College of American Pathologists (CAP) and under the Clinical Laboratory Improvement Amendments (CLIA)-USA as qualified to perform high complexity clinical laboratory testing. It is accredited under ISO 15189:2012 and ISO 27001:2013 for Information Security Management Systems.

Disclaimer

The aberrant / absent/ downregulated expression of cell surface or intracellular markers used for CTCs detection can give rise to ambiguous test results. Cells with EPCAM/Cytokeratin down regulation or absent expression will not be detected with this test.

This report documents the genetic alterations detected in the submitted sample material. Information in this report is provided for information purpose only and should only be considered in conjunction with all other relevant information regarding a particular patient before the patient's treating physician recommends a course of treatment.

Decisions on patient care and treatment must be based on the independent medical judgment of the treating physicians, taking into consideration all applicable information concerning the patient's condition, such as patients and family history, physician's

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examination, information from other diagnostic test and patient references, in accordance with the standard of care in a given community. A treating physician's decisions should not be based on a single test or on the information contained in this report.

The information in this report does not constitute a treatment recommendation by Datar Cancer Genetics, either to use or not to use any specific therapeutic agent and should not be interpreted as treatment advice. Decisions on patient care and treatment rest solely within the discretion of the patient's treating physician.

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End of Report

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TEST NAME: Cancertrack™

Clinical Trials

TP53 mutation

NCT number: NCT03188965 **Phase:** I/II **Treatment:** Elimusertib **Cancer Type:** Colorectal Cancer	**Study Title:** An Open-label, First-in-human, Dose-escalation Study to Evaluate the Safety, Tolerability, Pharmacokinetics, Pharmacodynamics, and Maximum Tolerated Dose and / or Recommended Phase II Dose of the ATR Inhibitor BAY1895344 in Patients With Advanced Solid Tumors and Lymphomas **Variant Classification:** DNA repair pathway **Locations:** Canada, Japan, Singapore, Switzerland
NCT number: NCT05002868 **Phase:** I **Treatment:** RP12146 **Cancer Type:** Colorectal Cancer	**Study Title:** A Multi-center, Open-label, Phase I/Ib Study to Assess the Safety, Pharmacokinetics and Anti-tumor Activity of RP12146, a Poly (ADP-ribose) Polymerase (PARP) Inhibitor, in Patients With Locally Advanced or Metastatic Solid Tumors. **Variant Classification:** DNA repair pathway **Locations:** Czech Republic, Poland
NCT number: NCT02029001 **Phase:** II **Treatment:** Olaparib **Cancer Type:** Unspecified Solid Tumor	**Study Title:** A Two-period, Multicenter, Randomized, Open-label, Phase II Study Evaluating the Clinical Benefit of a Maintenance Treatment Targeting Tumor Molecular Alterations in Patients with Progressive Locally-advanced or Metastatic Solid Tumors. **Variant Classification:** HRR mutation **Locations:** France
NCT number: NCT03415659 **Phase:** I **Treatment:** HWH-340 **Cancer Type:** Unspecified Solid Tumor	**Study Title:** A Phase I, Open-label, Single-center, Single/Multiple-dose, Dose-escalation/Dose-expansion Clinical Study on Tolerance and Pharmacokinetics of HWH340 Tablet in Patients With Advanced Solid Tumors **Variant Classification:** HRR mutation **Locations:** China
NCT number: NCT03767075 **Phase:** II **Treatment:** Atezolizumab **Cancer Type:** Unspecified Solid Tumor	**Study Title:** Basket of Baskets: A Modular, Open-label, Phase II, Multicentre Study To Evaluate Targeted Agents in Molecularly Selected Populations With Advanced Solid Tumours **Variant Classification:** DNA repair mutation **Locations:** France, Germany, Netherlands, Spain, Sweden, United Kingdom
NCT number: NCT04905914 **Phase:** I/II **Treatment:** ATRN-119 **Cancer Type:** Unspecified Solid Tumor	**Study Title:** A Phase I/IIa, Open-Label, Safety, Pharmacokinetic, And Preliminary Efficacy Study Of Oral ATRN-119 In Patients With Advanced Solid Tumors **Variant Classification:** DNA repair mutation **Locations:** United States **Contacts:** Crystal Miller [617-463-9385; crystal.miller@aprea.com]

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TEST NAME: Cancertrack™

NCT number: NCT04901702 **Phase:** I/II **Treatment:** Talazoparib, Chemotherapy **Cancer Type:** Unspecified Solid Tumor	**Study Title:** A Randomized Phase I/II Study of Talazoparib or Temozolomide in Combination With Onivyde in Children With Recurrent Solid Malignancies and Ewing Sarcoma **Variant Classification:** HRR pathway **Locations:** Canada, United States **Contacts:** Dr. Sara Federico [866-278-5833; referralinfo@stjude.org]
NCT number: NCT04992013 **Phase:** II **Treatment:** Niraparib **Cancer Type:** Unspecified Solid Tumor	**Study Title:** Genomically Guided Phase II Study to Evaluate the Clinical Benefit of Niraparib in Tumors Metastatic to the CNS **Variant Classification:** DNA repair pathway **Locations:** United States **Contacts:** Dr. Priscilla Brastianos [617-643-1938; pbrastianos@mgh.harvard.edu]
NCT number: NCT04267939 **Phase:** I **Treatment:** Elimusertib, Niraparib **Cancer Type:** Unspecified Solid Tumor	**Study Title:** An Open-label Phase Ib Study to Determine the Maximum Tolerated and/or Recommended Phase 2 Dose of the ATR Inhibitor Elimusertib (BAY 1895344) in Combination With PARP Inhibitor Niraparib, in Participants With Recurrent Advanced Solid Tumors and Ovarian Cancer **Variant Classification:** DNA repair pathway **Locations:** United States **Contacts:** Bayer Clinical Trials Contact [888-842-2937; clinical-trials-contact@bayer.com]
NCT number: NCT04693468 **Phase:** I **Treatment:** Talazoparib, Palbociclib, Axitinib, Crizotinib **Cancer Type:** Unspecified Solid Tumor	**Study Title:** Modular Phase IB Hypothesis-Testing, Biomarker-Driven, Talazoparib Combination Trial (TalaCom) **Variant Classification:** DNA repair pathway **Locations:** United States **Contacts:** Timothy A. Yap [713-563-1784; tyap@mdanderson.org]
NCT number: NCT04853043 **Phase:** II **Treatment:** Cetuximab **Cancer Type:** Colorectal Cancer	**Study Title:** APK Mutant: A Single Arm Phase II Study of Cetuximab in Third Line for Mutant APC, TP53 and RAS Patients With Refractory Metastatic Colorectal Cancer **Variant Classification:** APC mutation, NRAS mutation, TP53 mutation **Locations:** United States **Contacts:** Susan Sharry [801-585-3453; susan.sharry@hci.utah.edu]

NRAS p.(A59T) c.175G>A

NCT number: NCT02885753 **Phase:** III **Treatment:** Bevacizumab, Chemotherapy **Cancer Type:** Colorectal Cancer	**Study Title:** Systemic Oxaliplatin or Intra-arterial Chemotherapy Combined With LV5FU2 and an Target Therapy in First Line Treatment of Metastatic Colorectal Cancer Restricted to the Liver **Variant Classification:** NRAS exon 3 mutation **Locations:** France, Switzerland

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TEST NAME: Cancertrack™

NCT number: NCT05593328 **Phase:** II **Treatment:** Onvansertib, Bevacizumab, Chemotherapy **Cancer Type:** Colorectal Cancer	**Study Title:** A Phase II, Randomized, Open-label Study of Onvansertib in Combination With FOLFIRI and Bevacizumab Versus FOLFIRI and Bevacizumab for Second Line Treatment of Metastatic Colorectal Cancer in Patients With a KRAS or NRAS Mutation **Variant Classification:** NRAS exon 3 mutation **Locations:** United States **Contacts:** Vicki Kelemen [858-952-7570; info@cardiffoncology.com]
NCT number: NCT03087071 **Phase:** II **Treatment:** Panitumumab, Trametinib **Cancer Type:** Colorectal Cancer	**Study Title:** A Phase II Enrichment Study of Panitumumab as a Single Agent or in Combination With Trametinib in Anti-EGFR-Refractory Stage IV Colorectal Cancer Patients **Variant Classification:** NRAS exon 3 mutation **Locations:** United States **Contacts:** Christine Parseghian [713-795-9280; cparseghian@mdanderson.org]
NCT number: NCT04189055 **Phase:** II **Treatment:** Cetuximab, Chemotherapy **Cancer Type:** Colorectal Cancer	**Study Title:** Cetuximab as Salvage Therapy in Patients with Neo Wild-type RAS/RAF Metastatic Colorectal Cancer with Liver Metastases. A Proof-of-concept Study **Variant Classification:** NRAS mutation **Locations:** France
NCT number: NCT03519412 **Phase:** II **Treatment:** Pembrolizumab **Cancer Type:** Colorectal Cancer	**Study Title:** Pembrolizumab in MMR-Proficient Metastatic Colorectal Cancer Pharmacologically Primed to Trigger Dynamic Hypermutation Status **Variant Classification:** NRAS mutation **Locations:** Italy
NCT number: NCT05221320 **Phase:** II **Treatment:** Ulixertinib, Antimalarial **Cancer Type:** Colorectal Cancer	**Study Title:** A Phase II Basket Trial of Ulixertinib (BVD-523) in Combination With Hydroxychloroquine in Patients With Advanced GI Malignancies Harboring Mitogen-activated Protein Kinase (MAPK) Pathway Mutations (BVD-523-HCQ) **Variant Classification:** NRAS mutation **Locations:** United States **Contacts:** Biomed Valley Discoveries [816-960-6600; ERK@biomed-valley.com]
NCT number: NCT05039177 **Phase:** I/II **Treatment:** ASN007, Palbociclib **Cancer Type:** Colorectal Cancer	**Study Title:** A Phase Ib/II Study of Agents Targeting the Mitogen-Activated Protein Kinase Pathway in Patients With Advanced Gastrointestinal Malignancies (HERKULES-3) **Variant Classification:** NRAS mutation **Locations:** United States **Contacts:** Erasca Clinical Team [858-465-6511; clinicaltrials@erasca.com]

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TEST NAME: Cancertrack™

NCT number: NCT04892017 **Phase:** I/II **Treatment:** DCC-3116, Trametinib **Cancer Type:** Colorectal Cancer	**Study Title:** A Phase I/II, First-in-human Study of Dcc-3116 as Monotherapy and in Combination with Ras/Mapk Pathway Inhibitors in Patients with Advanced or Metastatic Solid Tumors with Ras/Mapk Pathway Mutations **Variant Classification:** NRAS mutation **Locations:** United States **Contacts:** Clinical Team [833-432-2237; clinicaltrials@deciphera.com]
NCT number: NCT02613650 **Phase:** I **Treatment:** Chemotherapy, Binimetinib **Cancer Type:** Colorectal Cancer	**Study Title:** A Phase Ib Trial of a Combination of mFOLFIRI With MEK162 in Patients With Advanced RAS (HRAS, NRAS, or KRAS) Positive Metastatic Colorectal Cancers **Variant Classification:** NRAS mutation **Locations:** United States **Contacts:** Arun Athithan [801-587-4624; arun.athithan@hci.utah.edu]
NCT number: NCT05069935 **Phase:** I **Treatment:** FT538, Cetuximab **Cancer Type:** Colorectal Cancer	**Study Title:** A Phase I, Open-Label, Multicenter Study of FT538 in Combination With Monoclonal Antibodies in Subjects With Advanced Solid Tumors **Variant Classification:** NRAS mutation **Locations:** United States **Contacts:** DeShaun Noakes [858-875-1800; clinical@fatetherapeutics.com]
NCT number: NCT05004441 **Phase:** II **Treatment:** Fruquintinib, Chemotherapy **Cancer Type:** Colorectal Cancer	**Study Title:** Fruquintinib Combined With mFOLFOX6/FOLFIRI in First-line Treatment for Metastatic Colorectal Cancer:HCCSC C02 Trial **Variant Classification:** RAS activating mutation **Locations:** China
NCT number: NCT04781270 **Phase:** III **Treatment:** Bevacizumab, Chemotherapy **Cancer Type:** Colorectal Cancer	**Study Title:** mFOLFOXIRI Plus Bevacizumab Versus mFOLFOX6 Plus Bevacizumab for the First Line Treatment of RAS Mutant Unresectable Colorectal Liver-limited Metastases **Variant Classification:** RAS mutation **Locations:** China
NCT number: NCT05727163 **Phase:** II **Treatment:** Bevacizumab, Chemotherapy **Cancer Type:** Colorectal Cancer	**Study Title:** FOLFOX Via Hepatic Artery Infusion Chemotherapy (HAI) Plus Systemic Irinotecan With or Without Bevacizumab Versus Systemic FOLFOXIRI With or Without Bevacizumab in Patients With Initially Unresectable RAS-mutated Colorectal Cancer With Liver Metastases: A Prospective, Randomized, Controlled Clinical Study **Variant Classification:** RAS mutation **Locations:** China

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TEST NAME: Cancertrack™

NCT number: NCT03905148 **Phase:** I/II **Treatment:** Mirdametinib, Lifirafenib **Cancer Type:** Colorectal Cancer	**Study Title:** A Phase Ib, Open-Label, Dose-escalation and Expansion Study to Investigate the Safety, Pharmacokinetics and Antitumor Activities of a RAF Dimer Inhibitor BGB-283 in Combination With MEK Inhibitor PD-0325901 in Patients With Advanced or Refractory Solid Tumors **Variant Classification:** RAS mutation **Locations:** Australia, United States **Contacts:** BeiGene [877-828-5568; clinicaltrials@beigene.com]
NCT number: NCT03284502 **Phase:** I **Treatment:** Cobimetinib, Belvarafenib **Cancer Type:** Colorectal Cancer	**Study Title:** A Phase Ib, Open-label, Multicenter, Dose Escalation Study of the Safety, Tolerability, and Pharmacokinetics of HM95573 in Combination With Either Cobimetinib or Cetuximab in Patients With Locally Advanced or Metastatic Solid Tumors **Variant Classification:** RAS mutation **Locations:** Republic of Korea
NCT number: NCT03900442 **Phase:** I **Treatment:** GGTI-2418, Bortezomib **Cancer Type:** Colorectal Cancer	**Study Title:** Phase Ib Pharmacodynamic and Pharmacokinetic Study of the Geranylgeranyltransferase I Inhibitor PTX-100 (GGTI-2418) in Patients With Advanced Malignancies **Variant Classification:** RAS mutation **Locations:** Australia
NCT number: NCT04303403 **Phase:** I **Treatment:** Trametinib, Ruxolitinib **Cancer Type:** Colorectal Cancer	**Study Title:** Phase Ib Study Evaluating Safety and Tolerability of Combination Trametinib and Ruxolitinib in Patients with Advanced RAS Mutant Colorectal Cancer and Pancreatic Adenocarcinoma **Variant Classification:** RAS mutation **Locations:** Singapore
NCT number: NCT05678257 **Phase:** II **Treatment:** Bevacizumab, Leucovorin, Chemotherapy **Cancer Type:** Colorectal Cancer	**Study Title:** A Randomised, Open-label, Phase II, Dose/Schedule Optimisation Study of NUC-3373/Leucovorin/Irinotecan Plus Bevacizumab (NUFIRI-bev) Versus 5-FU/Leucovorin/Irinotecan Plus Bevacizumab (FOLFIRI-bev) for the Treatment of Patients With Previously Treated Unresectable Metastatic Colorectal Cancer **Variant Classification:** RAS mutation status **Locations:** United States **Contacts:** Bryn Dixon [bryndixon@nucana.com]
NCT number: NCT04963283 **Phase:** II **Treatment:** Cabozantinib, Nivolumab **Cancer Type:** Colorectal Cancer	**Study Title:** A Phase II Study of Cabozantinib and Nivolumab in Refractory Metastatic Microsatellite Stable (MSS) Colorectal Cancer **Variant Classification:** RAS mutation status **Locations:** United States **Contacts:** Matt Lee [720-848-0630; Matthew.R.Lee@cuanschutz.edu]

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TEST NAME: Cancertrack™

NCT number: NCT04116541 **Phase:** II **Treatment:** Trametinib **Cancer Type:** Unspecified Solid Tumor	**Study Title:** MegaMOST - A Multicenter, Open-label, Biology Driven, Phase II Study Evaluating the Activity of Anti-cancer Treatments Targeting Tumor Molecular Alterations /Characteristics in Advanced / Metastatic Tumors. **Variant Classification:** NRAS activating mutation **Locations:** France
NCT number: NCT05585320 **Phase:** I/II **Treatment:** IMM-1-104 **Cancer Type:** Unspecified Solid Tumor	**Study Title:** A Phase I/II a, Open-Label, Multicenter, Nonrandomized, Safety and Anti-tumor Activity Study of IMM-1-104, a Novel Oral Dual MEK1/2 Inhibitor in Participants With Previously Treated RAS-Mutated Advanced or Metastatic Solid Tumors **Variant Classification:** NRAS mutation **Locations:** United States **Contacts:** IMM1104-101 Study Team [860-321-1302; clinicaltrials@immuneering.com]
NCT number: NCT03919292 **Phase:** I/II **Treatment:** Neratinib, Valproic Acid **Cancer Type:** Unspecified Solid Tumor	**Study Title:** Phase 1/2 Study of Neratinib and Divalproex Sodium (Valproate) in Advanced Solid Tumors, With an Expansion Cohort in Ras-Mutated Cancers **Variant Classification:** NRAS mutation **Locations:** United States **Contacts:** massey SIIT Team [804-628-9238; masseysiit@vcu.edu]
NCT number: NCT04599140 **Phase:** I/II **Treatment:** SX-682, Nivolumab **Cancer Type:** Colon Cancer, Rectal Cancer	**Study Title:** Phase Ib/II Trial of SX-682 in Combination With Nivolumab for Refractory RAS Mutated (RAS) Microsatellite Stable (MSS) Metastatic Colorectal Cancer (mCRC) (STOPTRAFFIC-1) **Variant Classification:** NRAS mutation **Locations:** United States **Contacts:** Dr. Benny Johnson [713-792-2330; bjohnson6@mdanderson.org]
NCT number: NCT04249843 **Phase:** I **Treatment:** BGB-3245 **Cancer Type:** Unspecified Solid Tumor	**Study Title:** A First-in-Human, Phase Ia/Ib, Open Label, Dose-Escalation and Expansion Study to Investigate the Safety, Pharmacokinetics, and Antitumor Activity of the RAF Dimer Inhibitor BGB-3245 in Patients With Advanced or Refractory Tumor **Variant Classification:** NRAS mutation **Locations:** Australia, United States **Contacts:** MapKure [877-828-5568; clinicaltrials@mapkure.com]
NCT number: NCT04418167 **Phase:** I **Treatment:** JSI-1187 **Cancer Type:** Unspecified Solid Tumor	**Study Title:** A Phase I Study of ERK1/2 Inhibitor JSI-1187 Administered as Monotherapy and in Combination With Dabrafenib for the Treatment of Advanced Solid Tumors With MAPK Pathway Mutations **Variant Classification:** NRAS mutation **Locations:** United States **Contacts:** Georgine N. Price [301-610-4990; georgineprice@westat.com]

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TEST NAME: Cancertrack™

NCT number: NCT05340621 **Phase:** I/II **Treatment:** OKI-179, Binimetinib **Cancer Type:** Unspecified Solid Tumor	**Study Title:** NAUTILUS: A Phase I b/II Study of OKI-179 Plus Binimetinib in Patients With Advanced Solid Tumors and Activating Mutations in the RAS Pathway (Phase 1b) and in Patients With Advanced NRAS-Mutated Melanoma (Phase 2) **Variant Classification:** RAS activating mutation **Locations:** United States **Contacts:** Onkure [720-307-2892; info@onkuretherapeutics.com]
NCT number: NCT05425940 **Phase:** III **Treatment:** XL-092, Atezolizumab, Regorafenib **Cancer Type:** Colon Cancer, Rectal Cancer	**Study Title:** A Randomized Open-Label Phase III Study of XL092 + Atezolizumab vs Regorafenib in Subjects With Metastatic Colorectal Cancer **Variant Classification:** RAS mutation **Locations:** United States **Contacts:** Exelixis Clinical Trials [888-393-5494; druginfo@exelixis.com]
NCT number: NCT04800822 **Phase:** I **Treatment:** PF-07284892, Binimetinib **Cancer Type:** Unspecified Solid Tumor	**Study Title:** A Phase I Open-Label, Multi-Center, Dose Escalation and Dose Expansion Study to Evaluate the Safety, Tolerability, Pharmacokinetics, and Preliminary Evidence of Antitumor Activity of PF-07284892 (ARRY-558) as a Single Agent and in Combination Therapy in Participants With Advanced Solid Tumors **Variant Classification:** RAS mutation **Locations:** United States **Contacts:** Pfizer CT.gov Call Center [800-718-1021; ClinicalTrials.gov_Inquiries@pfizer.com]
NCT number: No NCT ID **Phase:** II **Treatment:** Bevacizumab, Ramucirumab, Aflibercept, Chemotherapy **Cancer Type:** Colon Cancer, Rectal Cancer	**Other identifiers:** Brave Ace study, JCOG2004, jRCTs031220058 **Study Title:** JCOG2004: Randomized Phase II Study of Bevacizumab plus FOLFIRI versus Ramucirumab plus FOLFIRI versus Aflibercept plus FOLFIRI for Metastatic Colorectal Cancer after failure of First-line chemotherapy with Fluoropyrimidine and Oxaliplatin to Explore Predictive Biomarker (Brave Ace study) **Variant Classification:** RAS mutation status **Locations:** Japan
NCT number: No NCT ID **Phase:** I/II **Treatment:** GH-55 **Cancer Type:** Unspecified Solid Tumor	**Other identifiers:** CTR20222761, GH55-CRS001 **Study Title:** A Phase I/II Clinical Study to Evaluate the Safety, Tolerability, Pharmacokinetics, Pharmacodynamics Characteristics and Efficacy of Oral Administration of GH55 in Patients with Advanced Solid Tumors with Mutations in MAPK Signaling Pathway **Variant Classification:** RAS/RAF/MEK/ERK mutation **Locations:** China

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TEST NAME: Cancertrack™

NCT number: NCT04198818 **Phase:** I/II **Treatment:** HH-2710 **Cancer Type:** Unspecified Solid Tumor	**Study Title:** A First-in-Human, Open Label, Phase I/II Study to Evaluate the Safety, Tolerability and Pharmacokinetics of HH2710 in Patients With Advanced Tumors **Variant Classification:** RAS/RAF/MEK/ERK mutation **Locations:** China, United States **Contacts:** Dr. Harb Wael Abou [765-446-5111; Wharb@horizonbioadvance.com]
NCT number: NCT05557045 **Phase:** I **Treatment:** JZP-815 **Cancer Type:** Unspecified Solid Tumor	**Study Title:** Phase I, FIH, Open-label, Nonrandomized, Multicenter Study of JZP815 in Participants With Advanced or Metastatic Solid Tumors Harboring Alterations in the MAPK Pathway **Variant Classification:** RAS/RAF/MEK/ERK mutation **Locations:** United States **Contacts:** Clinical Trial Disclosure & Transparency [215-832-3750; ClinicalTrialDisclosure@JazzPharma.com]
NCT number: NCT04551521 **Phase:** II **Treatment:** Atezolizumab + Cobimetinib **Cancer Type:** Unspecified Solid Tumor	**Study Title:** Continuous ReAssessment With Flexible ExTension in Rare Malignancies - CRAFT: The NCT-PMO-1602 Phase II Trial **Variant Classification:** RAS/RAF/MEK/ERK pathway **Locations:** Germany
NCT number: NCT03520075 **Phase:** I/II **Treatment:** ASTX029 **Cancer Type:** Unspecified Solid Tumor	**Study Title:** A Phase I/II Study of the Safety, Pharmacokinetics, and Activity of ASTX029 in Subjects With Advanced Solid Tumors **Variant Classification:** RAS/RAF/MEK/ERK pathway **Locations:** France, Spain, United Kingdom, United States **Contacts:** General Inquiries [925-560-0100; clinicaltrials@astx.com]
NCT number: NCT05580770 **Phase:** I/II **Treatment:** Mirdametinib, BGB-3245 **Cancer Type:** Unspecified Solid Tumor	**Study Title:** A Phase I/IIa Open-Label, Dose Escalation and Expansion Study to Investigate the Safety, Pharmacokinetics, Pharmacodynamics and Efficacy of Mirdametinib in Combination With BGB-3245 in Patients With Advanced Solid Tumors **Variant Classification:** RAS/RAF/MEK/ERK pathway **Locations:** United States **Contacts:** SpringWorks Clinical [919-790-1002; clinical@springworkstx.com]
NCT number: NCT04305249 **Phase:** I **Treatment:** AZD-0364, Nivolumab **Cancer Type:** Unspecified Solid Tumor	**Study Title:** A Phase I, Open-Label, Multi-Center Dose Finding Study to Investigate the Safety, Pharmacokinetics, and Preliminary Efficacy of ATG-017 Monotherapy or Combination Therapy With Nivolumab in Patients With Advanced Solid Tumors and Hematological Malignancies **Variant Classification:** RAS/RAF/MEK/ERK pathway **Locations:** Australia

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NCT number: NCT04528836 **Phase:** I **Treatment:** BBP-398 **Cancer Type:** Unspecified Solid Tumor	**Study Title:** A Phase I/IB First-in-Human Study of the SHP2 Inhibitor BBP-398 (Formerly Known as IACS-15509) in Patients With Advanced Solid Tumors **Variant Classification:** RAS/RAF/MEK/ERK pathway **Locations:** United States **Contacts:** Navire Clinical Operations [650-391-9740; nav1001ct.gov@bridgebio.com]
NCT number: NCT05354843 **Phase:** I **Treatment:** ET0038 **Cancer Type:** Unspecified Solid Tumor	**Study Title:** A Phase I, Open-Label, Multi-Center Dose Finding Study to Investigate the Safety, Pharmacokinetics, and Preliminary Efficacy of SHP2 Inhibitor ET0038 Monotherapy in Patients With Advanced Solid Tumors **Variant Classification:** RAS/RAF/MEK/ERK pathway **Locations:** China
NCT number: NCT05488821 **Phase:** I **Treatment:** QLH11906 **Cancer Type:** Unspecified Solid Tumor	**Study Title:** A Phase I Clinical Study to Evaluate the Safety, Tolerability and Pharmacokinetics of the Oral Pan-RAF Inhibitor QLH11906 in Subjects With Advanced Solid Tumors Harboring MAPK Pathway Alterations. **Variant Classification:** RAS/RAF/MEK/ERK pathway **Locations:** China

MAP2K1 p.(F53S) c.158T>C

NCT number: NCT04534283 **Phase:** II **Treatment:** Abemaciclib + Temuterkib **Cancer Type:** Unspecified Solid Tumor	**Study Title:** A Phase II Basket Trial of an ERK1/2 Inhibitor (LY3214996) in Combination With Abemaciclib for Patients Whose Tumors Harbor Pathogenic Alterations in BRAF, RAF1, MEK1/2, ERK1/2, and NF1 **Variant Classification:** MAP2K1 mutation **Locations:** United States **Contacts:** Anne Younger [317-274-0951; anefoste@iupui.edu]
NCT number: NCT04185831 **Phase:** II **Treatment:** Cobimetinib **Cancer Type:** Unspecified Solid Tumor	**Study Title:** MEGALiT - a Multicenter, Basket and Umbrella Explorative Trial on the Efficacy and Safety of Molecular Profile Selected Commercially Available Targeted Anti-cancer Drugs in Patients With Advanced Cancers Progressive on Standard Therapy **Variant Classification:** MAP2K1 mutation **Locations:** Sweden
NCT number: No NCT ID **Phase:** I/II **Treatment:** GH-55 **Cancer Type:** Unspecified Solid Tumor	**Other identifiers:** CTR20222761, GH55-CRS001 **Study Title:** A Phase I/II Clinical Study to Evaluate the Safety, Tolerability, Pharmacokinetics, Pharmacodynamics Characteristics and Efficacy of Oral Administration of GH55 in Patients with Advanced Solid Tumors with Mutations in MAPK Signaling Pathway **Variant Classification:** RAS/RAF/MEK/ERK mutation **Locations:** China

APC p.(R1450*) c.4348C>T

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TEST NAME: Cancertrack™

NCT number: NCT04851119 **Phase:** I/II **Treatment:** Tegatrabetan **Cancer Type:** Unspecified Solid Tumor	**Study Title:** A Phase I/II Study of Tegavivint (NSC#826393) in Children, Adolescents, and Young Adults With Recurrent or Refractory Solid Tumors, Including Lymphomas and Desmoid Tumors **Variant Classification:** APC mutation **Locations:** United States **Contacts:** Multiple contacts: See www.clinicaltrials.gov for complete list of contacts.

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