

Accession: SL23-000310

Received: 04/20/23

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Phone Number:
Account Number:



Pet: [Redacted] Owner: [Redacted] Species: Canine Breed: Mixed Breed Sex: M/N Age: 11 y

Site: Pulmonary mass Diagnosis: Pulmonary carcinoma

SearchLight DNA Overview

8 Biomarker(s) identified in the following genes:


ATM	CDKN2B
EGFR	ERRF1
FANCA	FANCG
FLCN	TP53


Sample QC Metrics


Specimen Type: FNACyto
Tumor Content (>20%): 90%
Mean Target Coverage (>200x): 416x

Number of Clinical Trials:

- This Cancer Type: 2
- General Cancer: 25

 8 Diagnostic Biomarkers

 2 Prognostic Biomarkers

 4 Matching Drugs: mobocertinib, olaparib, palbociclib, platinum-based chemotherapy

SearchLight DNA Summary

An integrated review of the genomic data, as well as clinical history and pathology reports, supports the diagnosis of lung carcinoma. Specifically, EGFR activating mutations (Ala701_Val703dup) have been commonly found in human lung carcinoma.

Notably, we identified mutations with therapeutic and prognostic associations, as described on page 2. To the best of our knowledge, mobocertinib is currently not available in veterinary compounding pharmacies. In addition, EGFR activating mutations, such as Ala701_Val703dup as found in this case, may be associated with treatment sensitivity to other EGFR inhibitors, such as lapatinib, based on pathway biology and pre-clinical studies in human cancers. (PMID: 34771085, 18588508) Although the therapeutic association of EGFR Ala701_Val703dup with lapatinib has not been fully explored in dogs, we are providing this information in light of the absence of other available EGFR-targeting agents.

The most compelling evidence supports the initial consideration of the EGFR:lapatinib association, followed by the ATM:olaparib or ATM:carboplatin, and CDKN2B:palbociclib associations, based on mutation-level evidence of pathogenicity, tumor-type-specificity, as well as drug availability/safety/toxicity and evidence levels for the therapeutic biomarker associations in the report. Monographs describing published data on the use of these agents in dogs are available upon request, or you can find them on our website (<https://vidiumah.com/monographs/>).

This test evaluated 120 cancer genes in the submitted sample. The ABCB1-1delta (MDR1-1delta) mutation was not detected, indicating that the patient is unlikely to experience the ABCB1-1delta-related adverse effects of chemotherapy.

SearchLight DNA Clinician Report

Pet Name:



Therapeutic Biomarkers

Treatment Options Based on Mutations

Drug	Mutation	Available for dogs	Used in humans
mobocertinib	EGFR p.Ala701_Val703dup	No	Yes
olaparib	ATM Copy Number Loss	Yes	Yes
palbociclib	CDKN2B Copy Number Loss	Yes	Yes
platinum-based chemotherapy	ATM Copy Number Loss	Yes	Yes

Drug Resistance-Associated Biomarkers

Drug	Mutation
-	-

Pharmacogenomic Biomarkers

Gene	Mutation
ABCB1	ABCB1-1Δ Mutation Present



Diagnostic Biomarkers

Described in:

Gene	Mutation	Canine cancer	Human cancer
ATM	Copy Number Loss	Mammary Cancer , Prostate Carcinoma	Yes
CDKN2B	Copy Number Loss	Fibrosarcoma , Glioma , Head and Neck Squamous Cell Carcinoma , Hemangiosarcoma , Histiocytic Sarcoma , Lung Carcinoma , Mammary Cancer , Mast Cell Tumor , Melanoma , Osteosarcoma	Yes
EGFR	p.Ala701_Val703dup	-	Yes
ERRF1	Copy Number Loss	-	Yes
FANCA	Copy Number Loss	-	Yes
FANCG	Copy Number Loss	-	Yes
FLCN	Copy Number Loss	Osteosarcoma	-
TP53	Copy Number Loss	Colorectal Adenocarcinoma , Hemangiosarcoma , Histiocytic Sarcoma , Mammary Cancer , Mast Cell Tumor , Osteosarcoma , Prostate Carcinoma	Yes



Prognostic Biomarkers

Negative Prognostic Factor in:

Gene	Mutation	Canine cancer	Human cancer
CDKN2B	Copy Number Loss	T-Cell Lymphoma	-
TP53	Copy Number Loss	Mast Cell Tumor	Yes

Evidence Level Key

- a Validated biomarker - Proven biomarker with wide consensus and whose use is included in professional guidelines
- b Clinical evidence - Biomarker with consensus from experts in the field with data obtained from large, well-powered studies
- c Case studies - Biomarker suggested by data from one or more individual case reports from clinical journals
- d Preclinical evidence - Biomarker suggested by data from *in vivo* or *in vitro* models

Mutation Summaries

Pet Name:

Gene	ABCB1	Mutation	ABCB1-1Δ Mutation Present
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Variant Summary:

A polymorphism (referred to as ABCB1-1delta) occurs in a subset of dog breeds, including many herding breeds. The ABCB1-1delta polymorphism is a 4-base pair deletion that causes a shift in the reading frame that results in premature truncation of P-glycoprotein and loss of P-glycoprotein function. Dogs that are homozygous or heterozygous for this polymorphism can experience increased toxicity for chemotherapeutic agents that are substrates for ABCB1, such as doxorubicin, vincristine, and vinblastine. Dogs without this polymorphism (non-mutant) show standard susceptibility to chemotherapy-associated adverse effects, and a dosing adjustment based on ABCB1 status is not needed. (Mealey et al. J Vet Intern Med 2008; Mealey et al. Vet Clin North Am Small Anim Pract 2013)

Detailed Summary:

Please see [Link](#) for a detailed summary of this gene as well as information regarding this variant and its associated canine and human data.

Gene	ATM	Mutation	Copy Number Loss
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Roles in this case:  Diagnostic  Therapeutic

Variant Summary:

The gene ATM encodes for the "ATM Serine/Threonine Kinase", a member of the PI3/PI4-kinase family of proteins. The encoded protein and related kinase ATR are key controllers of cell cycle checkpoint signaling pathways that are required for cell response to DNA damage and for genome stability. It is a tumor suppressor gene and is inactivated either via deletions (copy number loss) or loss-of-function mutations in cancer. ATM has been mutated in canine prostate carcinoma (20%), hemangiosarcoma (7.7%), and mammary cancer (0.5-6%). ATM copy number losses have been observed in canine prostate carcinoma (20%). ATM copy number losses have been seen in 0.7% of human cancers, including cervical, uveal cancers, and melanoma, among others. (COSMIC, TCGA Pan-Cancer Atlas accessed via cBioPortal). ATM copy number losses are associated with treatment sensitivity to olaparib (a PARP inhibitor) in human BRCA1-mutant and/or BRCA2-mutant ovarian, breast, and pancreatic cancers, as well as in homologous recombination repair (HRR) gene-mutant prostate cancers. It has also been shown to confer sensitivity to platinum-based chemotherapy in human homologous recombination repair (HRR) gene-mutant ovarian carcinomas.

Detailed Summary:

Please see [Link](#) for a detailed summary of this gene as well as information regarding this variant and its associated canine and human data.

Gene	CDKN2B	Mutation	Copy Number Loss
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Roles in this case:  Diagnostic  Prognostic  Therapeutic


Variant Summary:

The gene CDKN2B encodes for the "Cyclin Dependent Kinase Inhibitor 2B" protein. The encoded protein functions as a cell growth regulator that controls cell cycle G1 progression. The gene interacts strongly with CDK4 and CDK6 cyclins, and acts a potent inhibitor. It is a tumor suppressor gene and is inactivated either via deletions (copy number loss) or loss-of-function mutations in cancer. CDKN2B has been mutated in canine osteosarcoma (37.5-69.7%), melanoma (2.7-68%), histiocytic sarcoma (62.79-63%), lymphoma (40-55.6%), glioma (9.88-50%), mammary cancer (50%), lung carcinoma (40%), hemangiosarcoma (3.7-28%), urothelial carcinoma (26%), squamous cell carcinoma (25%), and mast cell tumor (21.2%). CDKN2B copy number losses have been observed in canine osteosarcoma (37.5-69.7%), melanoma (2.7-68%), histiocytic sarcoma (62.79-63%), T-cell lymphoma (40-55.6%), glioma (9.88-50%), mammary cancer (50%), lung carcinoma (40%), hemangiosarcoma (21.74-28%), urothelial carcinoma (26%), squamous cell carcinoma (25%), mast cell tumor (21.2%). CDKN2B copy number losses have been seen in 24.5% of human cancers, including glioblastoma, mesothelioma, and esophageal cancers, among others. (COSMIC, TCGA Pan-Cancer Atlas accessed via cBioPortal). CDKN2B copy number loss is a negative prognostic biomarker in canine T-cell lymphoma. CDKN2B copy number losses are associated with treatment sensitivity to palbociclib (a CDK4/6 inhibitor) in human prostate cancer.

Detailed Summary:

Please see [Link](#) for a detailed summary of this gene as well as information regarding this variant and its associated canine and human data.

Gene	EGFR	Mutation	p.Ala701_Val703dup
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Roles in this case:  Diagnostic  Therapeutic


Variant Summary:

The gene EGFR encodes for the "Epidermal Growth Factor Receptor" protein, a transmembrane glycoprotein that is a member of the protein kinase superfamily. EGFR binds to epidermal growth factor, thus inducing receptor dimerization and tyrosine autophosphorylation leading to cell proliferation. It is an oncogene that is frequently activated via oncogenic mutations or copy number gains in cancer. EGFR has been mutated in canine osteosarcoma (85%), mammary cancer (0.5-25%), hemangiosarcoma (3.7%), and lung carcinoma (1%). This specific mutation, EGFR A701_V703dup, the human equivalent of EGFR A767_V769dup has been observed in human cancers. (COSMIC; TCGA Pan-Cancer Atlas accessed via cBioPortal). EGFR has been found to be mutated in 4.3% of human cancers, including glioblastoma, lung, melanoma, and other cancers. (COSMIC; TCGA Pan-Cancer Atlas accessed via cBioPortal). EGFR A701_V703dup mutation is associated with treatment sensitivity to mobocertinib (a kinase inhibitor) in human non-small cell lung cancer.

Detailed Summary:

Please see [Link](#) for a detailed summary of this gene as well as information regarding this variant and its associated canine and human data.

Gene	ERRF11	Mutation	Copy Number Loss
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Roles in this case:  Diagnostic


Variant Summary:

The gene ERF11 encodes for the "ERBB Receptor Feedback Inhibitor 1" protein, a cytoplasmic scaffold protein involved in negative regulation of ERBB receptors and is upregulated with cell growth. It is a tumor suppressor gene and is inactivated either via deletions (copy number loss) or loss-of-function mutations in cancer. ERF11 copy number losses have been seen in 0.7% of human cancers, including cholangiocarcinoma, adrenocortical carcinoma, and lymphoma, among others. (COSMIC, TCGA Pan-Cancer Atlas accessed via cBioPortal).

Detailed Summary:

Please see [Link](#) for a detailed summary of this gene as well as information regarding this variant and its associated canine and human data.

Gene	FANCA	Mutation	Copy Number Loss
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Roles in this case:  Diagnostic


Variant Summary:

The gene FANCA encodes for the "Fanconi Anemia Complementation Group A" protein. Fanconi anemia is a genetically heterogeneous recessive disorder characterized by cytogenetic instability, hypersensitivity to DNA crosslinking agents, increased chromosomal breakage, and defective DNA repair. The members of the Fanconi anemia complementation group are related by their assembly into a common nuclear protein complex. It is a tumor suppressor gene and is inactivated either via deletions (copy number loss) or loss-of-function mutations in cancer. FANCA copy number losses have been seen in 1.2% of human cancers, including prostate, breast cancer, and ovarian cancers, among others. (COSMIC, TCGA Pan-Cancer Atlas accessed via cBioPortal).

Detailed Summary:

Please see [Link](#) for a detailed summary of this gene as well as information regarding this variant and its associated canine and human data.

Gene	FANCG	Mutation	Copy Number Loss
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Roles in this case:  Diagnostic


Variant Summary:

The gene FANCG encodes for the "Fanconi Anemia Complementation Group G" protein. Fanconi anemia is a genetically heterogeneous recessive disorder characterized by cytogenetic instability, hypersensitivity to DNA crosslinking agents, increased chromosomal breakage, and defective DNA repair. The members of the Fanconi anemia complementation group are related by their assembly into a common nuclear protein complex. It is a tumor suppressor gene and is inactivated either via deletions (copy number loss) or loss-of-function mutations in cancer. FANCG copy number losses have been seen in 0.1% of human cancers, including esophageal, lung, and head and neck cancers, among others. (COSMIC, TCGA Pan-Cancer Atlas accessed via cBioPortal).

Detailed Summary:

Please see [Link](#) for a detailed summary of this gene as well as information regarding this variant and its associated canine and human data.

Gene	FLCN	Mutation	Copy Number Loss
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Roles in this case:  Diagnostic



Variant Summary:

The gene FLCN encodes for the "Folliculin" protein, a GTPase activating protein (GAP) for RagC/D GTPase proteins involved in amino acid sensing and signaling to mTORC1, and is involved in regulation of oxidative metabolism at the mitochondria, mitochondrial biogenesis, and autophagy. It is a tumor suppressor gene and is inactivated either via deletions (copy number loss) or loss-of-function mutations in cancer. FLCN has been mutated in canine osteosarcoma (4-62%), and hemangiosarcoma (7.7%). FLCN copy number losses have been observed in canine osteosarcoma (62%). FLCN copy number losses have been seen in 0.5% of human cancers, including lymphoma, liver, and ovarian cancers, among others. (COSMIC, TCGA Pan-Cancer Atlas accessed via cBioPortal).

Detailed Summary:

Please see [Link](#) for a detailed summary of this gene as well as information regarding this variant and its associated canine and human data.

Gene	TP53	Mutation	Copy Number Loss
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Roles in this case:  Diagnostic  Prognostic

Variant Summary:

The gene TP53 encodes for the "Tumor Protein P53" protein. The encoded protein, known as the "guardian of the genome", responds to varied cellular stresses to regulate expression of target genes, thereby inducing cell cycle arrest, apoptosis, senescence, DNA repair, or changes in metabolism. In most cancers, it is a tumor suppressor gene that is inactivated either via deletions (copy number loss) or loss-of-function mutations. It has also been reported to be an oncogene in certain tumors. TP53 has been mutated in canine hemangiosarcoma (2-93.3%), osteosarcoma (3-83.3%), mammary cancer (0.55-50%), histiocytic sarcoma (3.85-46.15%), peripheral nerve sheath tumor (37.5%), mast cell tumor (3.57-30.8%), leiomyosarcoma (28.57-28.6%), gastrointestinal stromal tumor (25%), prostate carcinoma (20%), melanoma (1.54-18.92%), lymphoma (1-16.7%), lung carcinoma (1-12.5%), glioma (5.4%), and squamous cell carcinoma (4%). TP53 copy number losses have been observed in canine osteosarcoma (53-65%), mammary cancer (3.5-50%), mast cell tumor (14.7-30.8%), prostate carcinoma (20%), histiocytic sarcoma (9.3%), hemangiosarcoma (7.2%). TP53 copy number losses have been seen in 1.1% of human cancers, including sarcoma, prostate, and liver cancers, among others. (COSMIC, TCGA Pan-Cancer Atlas accessed via cBioPortal). TP53 copy number loss is a negative prognostic biomarker in canine mast cell tumor.

Detailed Summary:

Please see [Link](#) for a detailed summary of this gene as well as information regarding this variant and its associated canine and human data.

Clinical Trials Summary

Pet Name:

Clinical Trial for this tumor type	Location	Website
AAHSD005258 - Phase II Study of Neratinib in Dogs with Pulmonary Adenocarcinoma	Blue Buffalo Veterinary Clinical Trials Office, Ohio State University Columbus, OH	Link
WSU-VCS Lung Cancer - Capecitabine in Dogs with Carcinomas, Part 2	Washington State University - Veterinary Clinical Sciences Pullman, WA	Link

Other Clinical Trials That May Be Applicable

25 identified	See link for details
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Variants of Uncertain Significance

The following variants were detected in Ollie Crotzer's tumor sample. These variants are considered variants of uncertain significance, meaning the functional impact of the alteration on gene function is unknown or the role of the mutation in tumor diagnosis, prognosis, or treatment is unknown. Future research may reveal a role for the mutations in cancer.

Variants of Uncertain Significance

References

Pet Name:

1. Alteration frequencies in human cancers are derived from COSMIC (<https://cancer.sanger.ac.uk/>) and the curated set of non-redundant studies, as accessed through cBioPortal (<https://www.cbioportal.org/>).
2. Alteration frequencies in human cancers are derived from COSMIC (<https://cancer.sanger.ac.uk/>) and the TCGA pan-cancer cohort or the curated set of non-redundant studies, as accessed through cBioPortal (<https://www.cbioportal.org/>).
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Additional Supporting Information

1. Alteration frequencies in human cancers are derived from COSMIC <https://cancer.sanger.ac.uk/> and the TCGA pan-cancer cohort, as accessed through cBioPortal <https://www.cbioportal.org/>
2. Gene summaries are based on gene descriptions provided by the National Library of Medicine and National Center for Biotechnology Information <https://www.ncbi.nlm.nih.gov/gene>
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Genes Evaluated by SearchLight DNA

Pet Name:

SearchLight DNA detects multiple types of gene mutations:

- Single nucleotide variants, small nucleotide insertions and deletions (SNVs) occurring in selected commonly mutated regions in oncogenes ("Selected Exons") or in any coding region of a tumor suppressor gene ("All Coding Exons").
- Copy number variants (CNVs) including copy number gains encompassing oncogenes and copy number losses encompassing tumor suppressor genes.
- Internal tandem duplications (ITDs) occurring in oncogenes.
- Pharmacogenomic variants in genes that regulate drug processing.

	ARCB1	AKT1	AKT3	ALK	APC	ARAF	ARID1A	ASXL1	ATM	ATR	ATRX	BAP1	BRAF	BRCA1	BRCA2	BTX	CALR	CBL	CCND1	CCND2	CCND3	CCNE1	CDK12	CDK4
SNV Selected Exons		•	•	•		•							•				•	•	•					
SNV All Coding Exons					•		•	•	•		•		•	•									•	
CNV	•				•		•	•	•	•	•	•	•						•	•	•	•	•	•
ITD																								
Pharmacogenomic	•																							

	CDK6	CDKN2A	CDKN2B	CHIE2	CRKL	CSF3R	CTNNB1	DDR2	DNMT3A	EGFR	ERBB2	ERBB3	ESR1	EZH2	FANCA	FANCC	FANCG	FANCL	FBXW7	FGF3	FGFR1	FGFR2	FGFR3	FLCN
SNV Selected Exons							•	•	•		•		•								•	•	•	
SNV All Coding Exons				•					•		•									•				•
CNV	•	•	•	•	•					•	•	•			•	•	•	•	•	•	•	•	•	•
ITD																								
Pharmacogenomic																								

	FLT3	GNAS	GNAS	GNB1	H3F3A	HRAS	IDH1	IDH2	IKZF1	JAK1	JAK2	KDR	KIT	KMT2D	KRAS	MAP2K1	MAP2K2	MAPK1	MDM2	MDM4	MIEN1	MET	MILH1	MSH2
SNV Selected Exons	•	•	•	•	•	•	•	•	•	•	•	•	•		•	•	•	•				•		
SNV All Coding Exons															•						•		•	•
CNV								•				•	•		•	•	•	•	•	•	•	•	•	•
ITD	•																							
Pharmacogenomic																								

	MSH3	MSH6	MTOR	MYC	MYCN	MYD88	NF1	NF2	NF2L2	NOTCH1	NPM1	NRAS	NTSC2	PALB2	PDGFRA	PIK3CA	PIK3R1	PLCG1	PMS2	POLD1	POLE	PITCH1	PTEN	PTPN11
SNV Selected Exons			•	•		•		•		•	•	•		•	•	•	•	•				•		•
SNV All Coding Exons	•	•					•	•		•				•						•	•	•	•	•
CNV	•	•		•	•		•	•						•	•	•	•	•	•	•	•	•	•	•
ITD																								
Pharmacogenomic																								

	RAC1	RAF1	RBI	REL	RET	RICTOR	RUNX1	SDHB	SDHD	SETD2	SESN1	SMAD4	SMARCA4	SMARCB1	SMO	STAT3	STK11	TP53	TRAF3	TSC1	TSC2	UZAF1	VEGFA	VHL
SNV Selected Exons	•	•			•	•		•		•					•	•					•			
SNV All Coding Exons			•				•	•		•		•	•	•	•			•	•	•	•	•		•
CNV		•	•	•			•	•		•		•	•	•	•			•	•	•	•	•	•	•
ITD																								
Pharmacogenomic																								

Assay Description

Pet Name: 

SearchLight DNA[®] detects multiple types of mutations in cancer genes:

SearchLight DNA is a Next Generation Sequencing targeted tumor-only assay that provides for the detection of single nucleotide variants (SNVs), small nucleotide insertions and deletions (indels), copy number variants (CNVs), internal tandem duplications (ITDs), and polymorphisms in tumor tissue. Genomic DNA is extracted from the patient's tumor sample. Isolated DNA is then prepared for sequencing using a custom, hybrid capture panel to enrich for target genomic regions with high actionability (Agilent). Sequencing library preparation includes shearing, purification, adaptor ligation and PCR amplification. Libraries are then clustered on a flow cell and sequenced using Illumina instruments. Sequence data are analyzed using validated bioinformatics tools (SearchLight DNA Pipeline) and canine polymorphism databases. The reference genome assembly used for alignment is CanFam 3.1. Each tumor's candidate cancer-specific mutations are queried against Vidium's proprietary knowledgebase, Vidium Insight™, which contains thousands of canine cancer biomarker associations derived from primary peer-reviewed literature to identify potential pharmacogenomic, diagnostic, prognostic, and therapeutic associations. Additionally, Vidium Insight contains human cancer biomarker associations inferred via genomic and proteomic alignments and conservation scores from the Catalogue of Somatic Mutations in Cancer (COSMIC) database. ABCB1 germline genotype is determined based on tumor-only sequencing. SNVs are reported when present at $\geq 3\%$ allele fraction. Allele fractions are dependent on tumor purity. Tumor purity is not taken into account when calculating allele fractions. Reported CNVs (gains/losses) are identified based on comparison to a copy number baseline generated from normal tissues across major breed clades and tissue types. Reported CNVs may be focal, arm-level, or chromosome-level. ITDs are reported only for KIT and FLT3 in selected exons. Pharmacogenomic polymorphisms are reported only for ABCB 1 (also known as MDR1). Indeterminate results may occur due to poor sample quality or sequencing coverage, but a "qualified report" may be delivered in these below-threshold instances after a manual data review. Mean target coverage for tumor sample DNA is $\geq 200x$ (unique reads) and $\geq 89\%$ of target bases bearing $\geq 100x$ coverage.

Limitations

Samples with tumor content less than 30% may have reduced sensitivity and lead to false negative results. It is also possible that the sample contains a mutation below our established limit of detection or in a genetic region not included in our assay. Mutations present in repetitive or high GC content region or non-coding areas may not be detected. Indels larger than 40bp may not be detected. Copy number signal relative to background noise inherent in DNA from FFPE samples may affect sensitivity of reporting CNV gains/losses. The lack of a variant call does not necessarily indicate the absence of a mutation since technical limitations in some genomic regions may limit assay detection. ABCB1 germline genotype is inferred from tumor-only sequencing and it remains possible, though unlikely, that either ABCB1 loss of heterozygosity in the tumor or somatic acquisition of an ABCB1 mutation could interfere with accurate genotyping.

Disclaimers

This test was developed, and performance characteristics determined, by Vidium Animal Health. This test has not been approved by the U.S. FDA. The FDA has determined that such clearance or approval for veterinary diagnostics is not necessary. This test is used for clinical purposes for veterinary patients. It should also be noted that the data interpretations are based on our current understanding of genes and mutations and are current as of the report date. Mutations may not be listed in order of strength of evidence or appropriateness for the patient's disease. When the report does identify mutations with therapeutic implications, this does not promise or guarantee that a particular drug or treatment regimen will be effective or helpful in the treatment of disease in any patient, and the selection of any drug for patient treatment is done at the discretion of the treating veterinarian. These treatment options are based solely on published biomarker associations and do not include dosing, safety, or combinatorial guidelines. Please refer to drug labeling, published literature, and safety data for warnings, precautions, and dosing guidelines. Use caution when combining multiple drugs and be aware of potential drug interactions. Drug availability in dogs is broadly referring to their availability at a reasonable price from any of the major veterinary compounding pharmacies within the United States. Genomic mutations should be considered in the context of the patient's history, risk factors and any previous genomic testing. Variants of Uncertain Significance (VUS) may be associated with potential therapies in the future. Vidium does not update reports or send notification regarding reclassification of these mutations. Vidium Animal Health's services, including but not limited to the results contained in this report, are governed by Vidium's Terms & Conditions, which are available at vidiumah.com/terms-conditions.

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Electronically Signed By:



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