



To help you understand how to find information on your Pathology Report page, below are descriptions and corresponding annotations of the different sections that may appear.

CBL (2) CDH1 (3) CDKN2A1 (1) CSF1R (2) CSF3R (4) CTNNB1 (1) DNMT DPYD (2) EGFR (9) ERBB2 (3) ERBB4 (8) EZH2 (19) FBXW7 (6) FGFR FGFR2 (9) FGFR3 (8) FGFR4 (8) FLT3 (5) GNA11 (4) GNAQ (4) GNAS HNF1A (2) HRAS (2) IDH1 (1) IDH2 (1) IGF1R (6) JAK2 (5) JAK3 (KDR (12) KIT (10) KMT2A (5) KRAS (3) MAP3K9 (4) MLH1 (1) MPL (1) MYD88 (1) NOTCH1 (2) NPM1 (1) NRAS (2) PAX5 (9) PDGFRA (5) PHF6 PIK3CA (13) PTEN (6) PTEN 18 (8) RB1 (8) RET (9) RUNX1 (8) SF3B1	Patient: Provider:					Date of Bir Receive	
Actionable Applicable	Results:						
TP53_Q165*_TPMT_A154T, KDR_Q472H, PIKSCA_J891M TPMT_Y240C			cer			on Specimen	Source
Biomarkers Sequenced (targeted regions) ABLI (8) ACT (1) ALK (10) APC (11) ASXLI (1) ATM (15) BRAF CBL (2) CDH (3) CDKN2AI (1) CSFIR (2) CSF3R (4) CTNNBI (1) DNMT DPYD (2) EOFR (9) ERBB2 (3) ERBB4 (8) EZH2 (19) FBXW7 (5) FGFR FGFR2 (9) FOFR3 (8) FBB2 (3) ERBB4 (8) EZH2 (19) FBXW7 (5) FGFR FGFR2 (9) FOFR3 (8) FGFR4 (7) FLT3 (5) GNAI1 (4) GNA2 (4) GNA2 HNF1A (2) HRAS (2) IDHI (1) (1) HIZ (1) IGFIR (6) JAV2 (5) JAV3 (KDR (12) HT2 (10) KMT2A (5) KRAS (3) MAPS(4) (4) MLH (1) MPL MYDB8 (1) NOTCH1 (2) NPM1 (1) KMT2A (5) KRAS (3) MAPS(4) (4) MLH (1) MPL MYDB8 (1) NOTCH1 (2) NPM1 (8) RB1 (8) RET (9) FDGFRA (5) PHF6 FIRSCA (1) TFBN (6) PTPN1 (8) RB1 (8) RET (9) RNMX (10) SPB3 SMAD4 (8) SNARCBI (3) SMO (5) SRC (1) STK11 (5) TET2 (9) TF53: TPMT (3) TYMS (1) UGT1AI (1) VHL (2) WTI (3) TPS3 The tumor suppressor gene TP53 encodes a transcription factor, p53, which is activated in response to sever forms of cellular stress and exerts multiple, antiproliferative functions. The biological consequences of p53 as include cell-cycle regulation, induction of appoiss, development, differentiation, gene amplification, but a circums of the composition of the	Actionable		TP53_Q16	55*, TPMT_A15			
CBL (2) CDH (3) CDKN2A1 (1) CSFIR (2) CSF3R (4) CTNNB1 (1) DNMT DPYD (2) EGR (9) ERBB2 (3) ERBB4 (8) EZH2 (19) EFXW7 (5) FGFR FGFR (8) FGFR3 (8) FGFR4 (8) FLT3 (5) GNA11 (4) GNA0 (4) GNA9 (4) GNA9 (4) HAS (2) HAS (2) HAS (2) HAN (11) IDM (11) IGF1R (6) JAA2 (5) JAA3 (MDR (12) KT (10) KMT2A (5) KRAS (3) MAP3/8 (4) MLH (1) MPL (7) MVD8 (1) NOTCH1 (2) NPM1 (1) NRAS (2) PAX5 (9) POFRA (5) PHOFRA (Biomarkers Se	equenced (targ		400	1		
TPS3 The tumor suppressor gene TP53 encodes a transcription factor, p53, which is activated in response to sever forms of cellular stress and exerts multiple, antiproliferative functions. The biological consequences of p53 a include cell-cycle regulation, induction of apoptosis, development, differentiation, gene amplification, DNA recombination, chromosomal segregation, and cellular senescence (PMID: 10066147, PMID: 11099028, PMID: 17401424). Somatic TP53 gene alterations are frequent in most human cancers (~50 %). TP53 Q165* (NM 000546.5:c.493C-T) The effect of this aberration on protein function is not known. However, this aberration is predicted to in a truncation of the protein, and may lead to reduction or loss of TP53 function. Therapeutic Therapeutic There are no approved drugs for the treatment of cancers with TP53 aberrations or directly target p53. There are several pre-clinical compounds in development that to p53 and work to either stimulate wild-type p53 protein function or induce p53 must p53 and work to either stimulate wild-type p53 protein function or induce p53 must p53 and work to either stimulate wild-type p53 protein function or induce p53 must p53 and work to either stimulate wild-type p53 protein function or induce p53 must p53 and work to either stimulate wild-type p53 protein function or induce p53 must p53 and work to either stimulate wild-type p53 protein function or induce p53 must p53 and work to either stimulate wild-type p53 protein function or induce p53 must p53 and work to either stimulate wild-type p53 protein function or induce p53 must p53 and work to either stimulate wild-type p53 protein function or induce p53 must p53 and work to either stimulate wild-type p53 protein function or induce p53 must p53 and p53 protein p53 and p53 protein p53 protei	ABL1 (8) CBL (2) DPYD (2) FGFR2 (9) HNF1A (2) KDR (12) MYD88 (1) PIK3CA (13)	AKT1 (3) CDH1 (3) EGFR (9) FGFR3 (8) HRAS (2) KIT (10) NOTCH1 (2) PTEN (6)	ALK (10) CDKN2A1 (1) ERBB2 (3) FGFR4 (8) IDH1 (1) KMT2A (5) NPM1 (1) PTPN11 (8)	CSF1R (2) ERBB4 (8) FLT3 (5) IDH2 (1) KRAS (3) NRAS (2) RB1 (8)	CSF3R (4) EZH2 (19) GNA11 (4) IGF1R (6) MAP3K9 (4) PAX5 (9) RET (9)	CTNNB1 (1) FBXW7 (5) GNAQ (4) JAK2 (6) MLH1 (1) PDGFRA (5) RUNX1 (8)	BRAF (2) DNMT3A FGFR1 (GNAS (2) JAK3 (2) MPL (1) PHF6 (8) SF3B1 (1)
The tumor suppressor gene TPS3 encodes a transcription factor, p53, which is activated in response to serv forms of cellular stress and exerts multiple, antiproliferative functions. The biological consequences of p53 a include cell-cycle regulation, induction of apoptosis, development, differentiation, gene amplification, DNA recombination, chromo somal segregation, and cellular senescence (PMID:10065147, PMID:11099028, PMID:11099028). TPS3, Q165* (NM 000546.5:c.493C>T) The effect of this aberration on protein function is not known. However, this aberration is predicted to in a truncation of the protein, and may lead to reduction or loss of TPS3 function. Therapeutic There are no approved drugs for the treatment of cancers with TPS3 aberrations or directly target p53. There are several pre-clinical compounds in development that the p53 and work to either stimulate wild-type p53 protein function or induce p53 muttan proteins to resume wild-type functions (PMID:15603511, PMID:24768524). Several have reported that various forms of p53 gene therapy (such as Ad. p53) are general and have demonstrated clinical efficacy in patients with lung cancer, including non-scell lung cancer (NSCLC) (PMID:1258456, PMID:13038106). In one phase 2 trial, evaluable patients with locally advanced breast cancer achieved an objective clinical response but did not achieve a pathologic complete response when treated with the harboring nonreplicating adenoviral vector, AdCMV-p53, and chemotherapy (PMID:15874816). Potential treatment approaches to consider include using Weel inhibitors, P53 vaccines, p53 activators, Avastin, or Votinent. These treatment approaches are further supported by data in other cancers. NCT01748825: MK-1775 for Advanced Solid Tumors NCT01748825: MK-1775 for Advanced Solid Tu						(-,	
include cell-cycle regulation, induction of apoptosis, development, differentiation, gene amplification, DNA recombination, chromosomal segregation, and cellular sene scence (PMID: 10065147, PMID: 11099028, PMID: 17401424). Somatic TP53 gene alterations are frequent in most human cancers (~50%). TP53_Q165^* (NM_000546.5:c.493C>T) The effect of this aberration on protein function is not known. However, this aberration is predicted to in a truncation of the protein, and may lead to reduction or loss of TP53 function. Therapeutic Relevance There are no approved drugs for the treatment of cancers with TP53 aberrations or directly target p53. There are several pre-clinical compounds in development that tap 153 and work to either simulate wild-type p53 protein function or induce p53 mutans proteins to resume wild-type functions (PMID: 15603511, PMID: 24768524). Several have reported that various forms of p53 gene therapy (such as Api32) are generall and have demonstrated clinical efficacy in patients with lung cancer, including non-cell lung cancer (NSCLC) (PMID: 12534656, PMID: 10329105). In open base 2 trial, evaluable patients with locally advanced breast cancer achieved an objective clinical response but did not achieve a pathologic complete response when treated with the harboring nonreplicating adenoviral vector, AdCMV-553, and chemotherapy. (PMID: 16874916). Potential treatment approaches to consider include using Weel inhibitors, TP59 vaccines, p53 activators, Avastin, or Votrient. These treatment approaches are further supported by data in other cancers. NCT01399871: Phase I Study of Pazopanib and Vorinostat METHODOLOGY Formalin-fixed, paraffin-embedded (FFPE) tissue sections are reviewed by a pathologist and manually microdissected to improve the troad. DNA extracted from the selected tissue area is subjected to target enrichment and massive parallel sequencing analysis. NCT01399871: Phase I Study of Pazopanib and Vorinostat METHODOLOGY Formalin-fixed, paraffin-embedded (FFPE) tissue sections are	The tumor supp						
TP53_Q165* (NM_000546.5:c.493C>T) The effect of this aberration on protein function is not known. However, this aberration is predicted to in a funcation of the protein, and may lead to reduction or loss of TP53 function. Therapeutic There are no approved drugs for the treatment of cancers with TP53 aberrations or Relevance directly target p53. There are several pre-clinical compounds in development that ta p53 and work to either stimulate wild-type p53 protein function or induce p53 mutan proteins to resume wild-type functions (PMID.15603511, PMID.24768524). Several have reported that various forms of p53 gene therapy (such as Ad, p53) are general and have demonstrated clinical efficacy in patients with lung cancer, including non-cell lung cancer (NSCLC) (PMID.1263456, PMID.10329108). In passage 2 rais, a valuable patients with locally advanced breast cancer achieved an objective clinical response but did not achieve a pathologic complete response when treated with the harboring nonreplicating adenoviral vector, AdC MV-p53, and chemotherapy. (PMID.16874816). Potential treatment approaches to consider include using Weel inhibitors; Pp53 vaccines, p53 activators, Avastin, or Votrient. These treatment approaches are further supported by data in other cancers. NCT01748825: MK-1775 for Advanced Solid Tumors METHODOLOGY Formalin-fixed, paraffin-embedded (FFPE) tissue sections are reweved by a pethologist and manually microdissected to improve the triangle of the parafficial sensitivity of this assay is approximately 5% mutant alleles within a wild-type background following turn or enrichment by microdissection. COMMENTS Results of this test cannot be used as sole mean for diagnosis and should be interpreted in context with other clinical and laboratory fin result of not detected in other regions/genes of this panel does not the out the presence of a mutation belowfhe analytical sensitivity of this assays is approximately 5% mutant alleles within a wild-type background following turn or enrichment by microdis	include cell-cycl	le regulation, in	duction of apoptos	is, developmen	it, differentiation,	, gene amplification	n, DÑA
The effect of this aberration on protein function is not known. However, this aberration is predicted to in a truncation of the protein, and may lead to reduction or loss of TPS3 function. Therapeutic Relevance There are no approved drugs for the treatment of cancers with TPS3 aberrations or directly target p53. There are several pre-clinical compounds in development that ta p53 and work to either stimulate wild-type p53 protein function or induce p53 mutan proteins to resume wild-type functions (PMID:15603511, PMID:24768524). Several have reported that various forms of p53 gene therapy (such as Ad. p53) are general and have demonstrated clinical efficacy in patients with lung cancer, including non-scell lung cancer (NSCLC) (PMID:12538456, PMID:10328105). In one phase 2 trial, evaluable patients with locally advanced breast cancer achieved an objective clinical response but did not achieve a pathologic complete response when treated with the harboring nonreplicating adenoviral vector, AdCMV-p53, and chemotherapy. (PMID:16874916). Potential treatment approaches to consider include using Weel inhibitors, TP53 vaccines, p53 activators, Avastin, or Votrient. These treatment approaches are further supported by data in other cancers. **CinicalTrial** **NCT01748825: MK-1775 for Advanced Solid Tumors** **NCT01339871: Phase I Study of Pazopanib and Vorinostat** **METHODOLOGY** Formalin-fixed, parafin-embedded (FPFE) tissue sections are reviewed by a pathologist and manually microdissected to improve the toled. DNA extracted from the selected tissue area is subjected to target enrichment and massive parallel sequencing analysis: **ANALYTICAL SENSITIVITY** The analytical sensitivity of this assay is approximately 5% mutant alleles within a wild-type background following tum or enrichment by microdissection. **COMMENTS** Results of this test cannot be used as sole mean for diagnosis and should be interpreted in context with other clinical and laboratory fined to tested and in other regions/genes of this panel wi							020,
in a truncation of the protein, and may lead to reduction or loss of TP53 function. There are no approved drugs for the treatment of cancers with TP53 aberrations or directly target p53. There are several pre-clinical compounds in development that ta p53 and work to either stimulate wild-type p53 protein function or induce p53 mutan proteins to resume wild-type p63 protein function or induce p53 mutan have reported that various forms of p53 gene therapy (such as Ad p53) are general and have demonstrated clinical efficacy in patients with lung cancer including non-scell lung cancer (NSCLC) (PMID-12538456, PMID-1328105). In one phase 2 trial, evaluable patients with locally advanced breast cancer achieved an objective clinical response but did not achieve a pathologic complete response when treated with the harboring nonreplicating adenoviral vector, AdCMV-p53, and chemotherapy (PMID-18874815). Potential treatment approaches to consider include using Wee1 inhibitors, TP53 vaccines, p53 activators, Avastin, or Votrient. These treatment approaches are further supported by data in other cancers. **NCT01748825: MK-1775 for Advanced Solid Tumors** **NCT01748825: MK-1775 for Advanced Solid Tumors** **METHODOLOGY** Formalin-fixed, paratini-embedded (FFPE) tissue sections are reviewed by a pathologic stand manually microdissected to improve the tribod. DNA extracted from the selected tissue area is subjected to target enrichment and massive parallel sequencing analysis. **ANALYTICAL SENSITIVITY** The analytical sensitivity of this assay is approximately 5% mutant alleles within a wild-type background following tun or enrichment by microdissection. **COMMENTS** Results of this test cannot be used as sole mean for diagnosis and should be interpreted in context with other clinical and laboratory fines the formation of the detection other regions genes of this parel does not rule out the pressibility of the presence of germline mutations in the mixed cell production. To rule out the genetic contribution, it testing							_
Relevance directly target p53. There are several pre-clinical compounds in development that ta p53 and work to either stimulate wild-type p53 protein function or induce p53 mutan proteins to resume wild-type functions (PMID:15603611, PMID:24768524). Several have reported that various forms of p53 gene therapy (such as Ad. p53) are general and have demonstrated clinical efficacy in patients with lung cancer, including non-scell lung cancer (NSCLC) (PMID:12538456, PMID:10328105). In one phase 2 trial, evaluable patients with locally advanced breast cancer achieved an objective clinical response but did not achieve a pathologic complete response when treated with the harboring nonreplicating adenoviral vector, AdCMV-p53, and chemotherapy (PMID:16874816). Potential treatment approaches to consider include using Wee1 inhibitors, TP53 vaccines, p53 activators, Avastin, or Votrient. These treatment approaches are further supported by data in other cancers. NCT02042989: MLN9708 and Vorinostat in Patients With Advanced p53 Mutant Malignancies NCT01748825: MK-1775 for Advanced Solid Tumors NCT01339871; Phase I Study of Pazopanib and Vorinostat METHODOLOGY Formalin-fixed, parafin-embedded (FFPE) tissue sections are reviewed by a pathologist and manually microdissected to improve the troad. DNA extracted from the selected tissue area is subjected to target enrichment and massive parallel sequencing analysis. ANALYTICAL SENSITIVITY The analytical sensitivity of this assay is approximately 5% mutant alleles within a wild-type background following tun or enrichment by microdissection. COMMENTS Results of this test cannot be used as sole mean for diagnosis and should be interpreted in context with other clinical and laboratory fin result of not detected in other regions/genes of this parel does not rule out the pressibility of the presence of the protein mutations in the mixed cell population. To rule out the germline contribution, it testing by using peripheral blood sample is recommended. Powered by Collabra. This				notion is not kn	ouis Houseup t		radiated to r
p53 and work to either stimulate wild-type p53 protein function or induce p53 mutan proteins to resume wild-type functions (PMID:15603511, PMID:24768524). Several have reported that various forms of p53 gene therapy (such as Ad.p53) are general and have demonstrated clinical efficacy in patients with lung cancer, including nonscell lung cancer, (NSCCL) (PMID:1538466, PMID:10328105). In one phase 2 trial, evaluable patients with locally advanced breast cancer achieved an objective clinical response but did not achieve a pathologic complete response when treated with the harboring nonreplicating adenoviral vector, AdCMV-p53, and chemotherapy. (PMID:16874816). Potential treatment approaches to consider include using Weet inhibitors, TP53 vaccines, p53 activators, Avastin, or Votrient. These treatment approaches are further supported by data in other cancers. NCT02042989: MLN9708 and Vorinostat in Patients With Advanced p53 Mutant Malignancies NCT01748825: MK-1775 for Advanced Solid Tumors NCT01748825: MK-1775 for Advanced Solid Tumors NCT01339871: Phase I Study of Pazopanib and Vorinostat METHOD OLOGY Formalin-fixed, paraffin-embedded (FFPE) tissue sections are reviewed by a pathologist and manually microdissected to improve the troad. DNA extracted from the selected tissue area is subjected to target enrichment and massive parallel sequencing analysis. ANALYTICAL SENSITIVITY The analytical sensitivity of this assay is approximately 5% mutant alleles within a wild-type background following tumor enrichment by microdissection. COMMENTS Results of this test cannot be used as sole mean for diagnosis and should be interpreted in context with other clinical and laboratory fin result of not detected in other regions/genes of this panel does not rule out the presence of germline mutations in the mixed cell population. To rule out the gene microorithoution, it testing by using peripheral blood sample is recommended. Powered by Collabra. This test was developed and its performance characteristics determined by	The effe	ect of this aberr ncation of the pr	ation on protein fu otein, and may le:	ad to reduction	or loss of TP53 t	this aberration is pr function.	
ANALYTICAL SENSITIVITY The analytical sensitivity of this assay is approximately 5% mutant alleles within a wild-type background following turn or enrichment by microdissection. COMMENTS Results of this test cannot be used as sole mean for diagnosis and should be interpreted in context with other clinical and laboratory fine result of not detected in other regions (or genes not covered by this panel will not be detected in the regions or genes not covered by this panel will not be detected. This assay is dependently of the presence of a nut attain below the analytical sensitivity of this panel does not rule out the presence of an utation below the analytical sensitivity. Mutations in the regions or genes not covered by this panel will not be detected. This assay is designed for detection of som altic mutation cannot rule out the presence of germ line mutations in the mixed cell population. To rule out the germline contribution, it testing by using peripheral blood sample is recommended. Powered by CollabRx. This test was developed and its performance characteristics determ ined by CelllNetix P athology & Laboratories. It has not been approved us for the performance characteristics determined by CelllNetix P athology & Laboratories. It has not been approved by Chincip and the performance characteristics determined by CelllNetix P athology & Laboratories. It has not been approved us for the performance characteristics determined by CelllNetix P athology & Laboratories. It has not been approved by Celllothon to be regarded as investigational or for research. This laboratory is regulated under the 1988 CLIA amendments a qualitied to perform high-complexity clinical testing.	The effe in a trur Therape	ect of this aberr ncation of the pr eutic There nce directl p53 ar proteir	ation on protein fur rotein, and may lea are no approved o y target p53. There nd work to either s ns to resume wild-t	ad to reduction drugs for the tre e are several pr timulate wild-typ type functions (or loss of TP53 t atment of cance e-clinical compo pe p53 protein fu PMID:15603511	this aberration is pr function. rrs with TP53 aberr runds in developme unction or induce p , PMID:24768524)	rations or the ent that targe 53 mutant . Several st
METHODOLOGY Formalin-fixed, paraffin-embedded (FFPE) tissue sections are reviewed by a pathologist and manually microdissected to improve the tribod. DNA extracted from the selected tissue area is subjected to target enrichment and massive parallel sequencing analysis. ANALYTICAL SENSITIVITY The analytical sensitivity of this assay is approximately 5% mutant alleles within a wild-type background following turn or enrichment by microdissection. COMMENTS Results of this test cannot be used as sole mean for diagnosis and should be interpreted in context with other clinical and laboratory fin result of not detected in other regions/genes of this panel will not be detected. This assay is designed for detection of some tic mutatic cannot rice out the possibility of the presence of germline mutations in the regions or genes not covered by this panel will not be detected. This assay is designed for detection of som atic mutatic cannot rice out the possibility of the presence of germline mutations in the mixed cell proputation. To rule out the germline contribution, it testing by using peripheral blood sample is recommended. Powered by CollabRx. This test was developed and its performance characteristics determined by CelliNetix P athology 8 Laboratories. It has not been approved using the properties of the propulation of the presence of the propulation of the propulation of the presence of the propulation of the presence of a propulation of the propulation of the propulation of the propulation of the presence of the propulation of the propulation of the presence of the propulation of the presence of the propulation of the presence of this section. This laboratory is regulated under the 1988 CLIA amendments a qualified to perform high-complexity dinical testing. Therapeutic Confidence Level	The effe in a trur Therape	ect of this aberr neation of the pre- eutic There need direct! p53 ar proteir have r and he cell lur evalua respor	ation on protein function and may less are no approved of ytarget p53. There is to resume wild-leported that various demonstrated gramer (NSCLC bible patients with less but did not aching nonreplicating 15874816). Pote	ad to reduction frugs for the tre are several pritinulate wild-typtype functions (see functions) (PMID:12538. cally advances) (PMID:12538. adenoviral vectotial treatment	or loss of TP53 i amment of cance et-clinical compo- pe p53 protein fu PMID:15603511 gene therapy (s in patients with 456, PMID:1032 I breast cancer s juc complete res tor, AdCMV-p53 approaches to complete to c	this aberration is profunction. Tra with TP53 abern unds in development of the control of the control pMID:24768524), such as Ad, p53) and lung cancer, inclue 8106). In one phatic ponse when treate one control of the control of	rations or the ent that targe 53 mutant . Several stee generally sting non-sm. see 2 trial, all twee dinical d with the Tipy.
METHODOLOGY Formalin-fixed, parafin-embedded (FFPE) tissue sections are reviewed by a pathologist and manually microdissected to improve the tribod. DNA extracted from the selected tissue area is subjected to target enrichment and massive parallel sequencing analysis. ANALYTICAL SENSITIVITY The analytical sensitivity of this assay is approximately 5% mutant alleles within a wild-type background following turn or enrichment by microdissection. COMMENTS Results of this test cannot be used as sole mean for diagnosis and should be interpreted in context with other clinical and laboratory fin result of not detected in other regions/genes of this panel does not rule out the presence of a mutation below the analytical sensitivity. Mutations in the regions or genes not covered by this panel will not be detected. This assay is designed for detection of som acid in utation to be out the possibility of the presence of germline mutations in the mixed cell proputation. To rule out the germline contribution, it testing by using peripheral blood sample is recommended. Powered by CollabRx. This set was developed and its performance characteristics determined by Cellillettix Pathology & Laboratories. It has not been approved u.S. Food and Drug Administration. The FDA has determined that such clearance or approval is not necessary. This test is for clinical purposes. It should not be regarded as investigational or for research. This laboratory is regulated under the 1988 CLIA amendments a qualified to perform high-complexity dinical testing.	The effi	ect of this abern ncation of the pr eutic There nce directly p53 ar protein have r and h cell lur ev alua respor harbor (PMID in hibit	ation on protein Tu ordein, and may lea are no approved of y target p53. There ind work to either sins to resume wild- eported that various we demonstrated ng cancer (NSCLC ble patients with lease but did not ach ing nonreplicating 15874816). Pote ors, TP55 vaccines aches are further s	ad to reduction drugs for the tre a are several primulate wild-tyr type functions () type functions () (PMID: 1258 a) (PMID: 1258 a) (PMID: 1258 a) adenoviral vectorial attention of the desired of the several part of the several properties of the	or loss of TP53 i atment of cance e-clinical compo e p53 protein fu PMID:15603511 gene therapy (s in patients with 456, PMID:1032 b breast cancer a jic complete res tor, AdCMV-p53 approaches to c s, Avastin, or Vo ta in other cancer	this aberration is pro- function. To with TF53 abern unds in development unction or induce pro- policy 24768524) such as Ad, p53) an lung cancer, inclue 8106). In one pha achieved an object ponse when treate and chemotherar onsider include usi trient. These treat ers.	rations or the ent that targe 53 mutant. Several stee generally stilling non-sm. se 2 trial, all ive clinical dwith the Tipy time the second of the second o
METHODOLOGY Formalin-fixed, paraffin-embedded (FFPE) tissue sections are reviewed by a pathologist and manually microdissected to improve the triload, DNA extracted from the selected tissue area is subjected to target enrichment and massive parallel sequencing analysis. ANALYTICAL SENSITIVITY The analytical sensitivity of this assay is approximately 5% mutant alleles within a wild-type background following turn or enrichment by microdissection. COMMENTS Results of this test cannot be used as sole mean for diagnosis and should be interpreted in context with other clinical and laboratory fin result of not detected in other regions/genes of this panel does not rule out the presence of a mutation below the analytical sensitivity. Mutations in the regions or genes not covered by this panel will not be detected. This assay is designed for detection of som ation utation and rule out the possibility of the presence of germline mutations in the mixed cell population. To rule out the germline contribution, testing by using peripheral blood sample is recommended. Powered by CollabRx. This test was developed and its performance characteristics determined by CelliNettix Pathology & Laboratories. It has not been approve U.S. Food and Drug Administration. The FDA has determined that such clearance or approval is not necessary. This test is for clinical purposes. It should not be regarded as investigational or for research. This laboratory is regulated under the 1988 CLIA amendments a qualified to perform high-complexity dinical testing.	The effi	ect of this abern ncation of the pr eutic There nce directl p53 ar proteir have r and ha cell lur evalua respor harbor (PMID inhibit a pproteir	ation on protein furotein, and may leare no approved of the protein and may leave the protein and work to either situations to resume wild-teported that variouse demonstrateding cancer (NSCLC bible patients with It is se but did not aching nonreplicating 116874816). Pote carbon are further second the protein and the	ad to reduction drugs for the tre a are several primulate wild-tyr type functions () type functions () (PMID: 1258 a) (PMID: 1258 a) (PMID: 1258 a) adenoviral vectorial attention of the desired of the several part of the several properties of the	or loss of TP53 i atment of cance e-clinical compo e p53 protein fu PMID:15603511 gene therapy (s in patients with 456, PMID:1032 b breast cancer a jic complete res tor, AdCMV-p53 approaches to c s, Avastin, or Vo ta in other cancer	this aberration is pro- function. To with TF53 abern unds in developm unction or induce pro- post as Ad, p53) and ung cancer, inclue 8106). In one pha achieved an object ponse when treate, and chemotheray onsider include usitnent. These treaters.	rations or the ent that targe 53 mutant. Several stee generally stilling non-sm. se 2 trial, all ive clinical dwith the Tipy time the second of the second o
Formalin-fixed, paraffin-embedded (FPE) tissue sections are reviewed by a pathologist and manually microdissected to improve the troad. DNA extracted from the selected tissue area is subjected to target enrichment and massive parallel sequencing analysis. ANALYTICAL SENSITIVITY The analytical sensitivity of this assay is approximately 5% mutant alleles within a wild-type background following tumor enrichment by microdissection. COMMENTS Results of this test cannot be used as sole mean for diagnosis and should be interpreted in context with other clinical and laboratory fin result of not detected in other regions/genes of this panel does not rule out the presence of a mutation belowthe analytical sensitivity. Mutations in the regions or genes not covered by this panel does not rule out the presence of a mutation belowthe analytical sensitivity. Mutations in the regions or genes not covered by this panel value for the following the properties of the presence of germ line mutations in the mixed cell population. To rule out the germline contribution, it esting by using peripheral blood sample is recommended. Powered by CollabRx. This test was developed and its performance characteristics determined by CellNettix Pathology & Laboratories. It has not been approv U.S. Food and Drug Administration. The FDA has determined that such clearance or approval is not necessary. This test is for clinical purposes. It should not be regarded as investigational or for research. This laboratory is regulated under the 1988 CLIA amendments a qualified to perform high-complexity clinical testing. Therapeutic Confidence Level	The effi in a truu Therapi Relevar	ect of this aberr neation of the pre eutic The pre ence directly p53 ar proteir have r and ha cell lur evalua respor (PMID nhibit approa	ation on protein furotein, and may lei- are no approved of y target p53. There id work to either is sto resume wild- eported that various demonstrated g cancer (NSCLC bile patients with I isse but did not accoming nonreplicating i16874816). Pote ors, TP53 vaccines aches are further is 2042989: MLN970 lancies	ad to reduction drugs for the tre are several primulate wild-try type functions (type functions) (PMID: 12538) (PMID: 12538) (PMID: 13538) (P	or loss of TP53 i atment of cance e-clinical compo ep 53 protein fu PMID:15603511 gene therapy (s in patients with 456, PMID:1032 b breast cancer a jic complete res tor, AdCMV-p53 approaches to c s, Avastin, or Vo ta in other cancer at in Patients Wi	this aberration is pro- function. To with TF53 abern unds in developm unction or induce pro- post as Ad, p53) and ung cancer, inclue 8106). In one pha achieved an object ponse when treate, and chemotheray onsider include usitnent. These treaters.	rations or the ent that targe 53 mutant. Several stee generally stilling non-sm. se 2 trial, all ive clinical dwith the Tipy time the second of the second o
ANALYTICAL SENSITIVITY The analytical sensitivity of this assay is approximately 5% mutant alleles within a wild-type background following turn or enrichment by microdissection. COMMENTS Results of this test cannot be used as sole mean for diagnosis and should be interpreted in context with other clinical and laboratory fin result of not detected in other regions/genes of this panel does not rule out the presence of a mutation below the analytical sensitivity. Mutations in the regions or genes not covered by this panel will not be detected. This assays is designed for detection of somatic mutatic cannot rule out the possibility of the presence of germ line mutations in the mixed cell population. To rule out the germline contribution, it testing by using peripheral blood sample is recommended. Powered by Collabarx. This test was developed and its performance characteristics determlined by CellNetix Pathology & Laboratories. It has not been approve U.S. Food and Drug Administration. The FDA has determined that such clearance or approval is not necessary. This test is for clinical purposes. It should not be regarded as investigational or for research. This laboratory is regulated under the 1988 CLIA amendments a qualified to perform high-complexity clinical testing. Therapeutic Confidence Level	The effi	ect of this abern coation of the prevail of the pre	ation on protein furotein, and may leare no approved a are no approved a remove for the second work to either sits to resume wild-eported that various demonstrated no cancer (NSCLC bile patients with learning nonreplicating (16874816). POS avaccines are further size42899; MLN970 lancies.	ad to reduction drugs for the tre are several primulate wild-try type functions (type functions (type functions () (PMID: 12538 collect a patholog adenoviral vectorial treatment s, p53 activator supported by da 8 and Vorinost: 5 for Advanced	or loss of TP53 i atment of cance e-clinical compo e p53 protein fu PMID:15603511 gene therapy (s in patients with 456, PMID:1032 b breast cancer a jic complete res jic complete res jor complete res tor, AdCMV-p53 approaches to c s, Avastin, or Vo ta in other cancer at in Patients Wi	this aberration is profunction. rs with TF53 abern unds in developm unction or induce profunction or induce profunction or induce profunction or induce profunction in the profunction of the profunction	rations or the ent that targe 53 mutant. Several stee generally stilling non-sm. se 2 trial, all ive clinical dwith the Tipy time the second of the second o
The analytical sensitivity of this assay is approximately 5% mutant alleles within a wild-type background following turn or enrichment by microdissection. COMMENTS Results of this test cannot be used as sole mean for diagnosis and should be interpreted in context with other clinical and laboratory fin result of not detected in other regions/genes of this panel does not rule out the presence of a mutation belowfhe analytical sensitivity. Mutations in the regions or genes not covered by this panel will not be detected. This assays is designed for detection of som atto mutatic cannot rule out the possibility of the presence of germline mutations in the mixed cell population. To rule out the germline contribution, it esting by using peripheral blood sample is recommended. Powered by CollabRx. This test was developed and its performance characteristics determlined by CellNetix Pathology & Laboratories. It has not been approve U.S. Food and Drug Administration. The FDA has determined that such clearance or approval is not necessary. This test is for clinical purposes. It should not be regarded as investigational or for research. This laboratory is regulated under the 1988 CLIA amendments a qualified to perform high-complexity dirical testing. Therapeutic Confidence Level	The effination in a truth Therapy Relevan	ect of this abern coation of the prevalence of the coation of the prevalence of the coation of the prevalence of the coation o	ation on protein fur orderin, and may lear are no approved of ytarget p53. There id work to either sits to resume wild- eported that variously demonstrated ng cancer (NSCLC bile patients with see but did not ach ing nonreplicating 16874816). POS et aches are further sits 2042989: MLN970 lancies.	ad to reduction drugs for the tre are several primulate wild-try type functions (us forms of p53 clinical efficacy) (PMID: 12538 cally advanced nieve a patholog adenoviral vectorial treatment s, p53 activator supported by da 8 and Vorinost: 5 for Advanced Study of Pazop	or loss of TP53 i atment of cance e-clinical compo ep 53 protein fu PMID:15603511 gene therapy (s in patients with 456, PMID:1032 b breast cancer a jic complete res jic complete res jic complete res jic complete res or, AdCMV-p53 approaches to complete res s, Avastin, or Vo ta in other cancer at in Patients Wi Solid Tumors anib and Vorino:	this aberration is profunction. To with TP53 aberration or winds in development of the second of th	rations or the not that targ 53 mutant . Several state e generally sing non-sm se 2 trial, all alive clinical d with the T DY. You would be seen that the s
COMMENTS Results of this test cannot be used as sole mean for diagnosis and should be interpreted in context with other clinical and laboratory fin result of not detected in other regions/genes of this panel does not rule out the presence of a mutation belowfhe analytical sensitivity. Mutations in the regions or genes not covered by this panel will not be detected. This assays is designed for detection of somation fusion to the control of the possibility of the presence of germline mutations in the mixed cell population. To rule out the germline contribution, it esting by using peripheral blood sample is recommended. Powered by CollabRx. This test was developed and its performance characteristics determined by CellNetix Pathology & Laboratories. It has not been approve U.S. Food and Drug Administration. The FDA has determined that such clearance or approval is not necessary. This test is for clinical purposes. It should not be regarded as investigational or for research. This laboratory is regulated under the 1988 CLIA amendments a qualified to perform high-complexity dinical testing. Therapeutic Confidence Level	The effination at truth Therapy Relevant Relevan	ect of this aberr ncation of the pr tentral trial tria	ation on protein full oterin, and may leave are no approved of the protein full of the	ad to reduction drugs for the tre are several primulate wild-type functions (s) forms of p53 clinical efficacy). (PMID: 12538 ocally advancedieve a pathology advancedieve a pathology advancedieve a pathology and point of the pathology of the pa	or loss of TP53 i atment of cance e-clinical compose p53 protein fu PMID:16603511 i gene therapy (s in patients with 456, PMID:1646, PMID:1610 i gic complete resi tor, AdC MV-553 approaches to cis, Awastin, or Vo tata in other cancer at in Patients With Solid Tumors anib and Vorino:	this aberration is production. To with TP53 abern unds in development on or induce produced as Ad. p53) and lung cancer, inclue 8106). In one pha achieved an object ponse when treate, and chemotheras onsider include usithent. These treaters. The Advanced p53 Notes and the phase when the same the streaters.	rations or the not that targ 53 mutant . Several state e generally state e generally state e generally state e see 2 trial, all wire clinical d with the T by ing Wee1 ment
result of not detected in other regions/genes of this panel does not rule out the presence of an utation belowthe analytical sensitivity. Mutations in the regions or genes not covered by this panel will not be detected. This assays is designed detection of some action utation action utation and the mutations in the mixed cell population. To rule out the germline contribution, it esting by using peripheral blood sample is recommended. Powered by CollabRx. This test was developed and its performance characteristics determined by CellNetix Pathology & Laboratories. It has not been approv U.S. Food and Drug Administration. The FDA has determined that such clearance or approval is not necessary. This test is for clinical purposes. It should not be regarded as investigational or for research. This laboratory is regulated under the 1988 CLIA amendments a qualified to perform high-complexity clinical testing. Therapeutic Confidence Level	The effination at truth Therapy Relevant Relevan	ect of this aberrincation of the princation of the protein have reported in the princation of the prin	ation on protein furotein, and may lear on approved of the protein and may lear on approved of the protein and work to either sits to resume wild-reported that various demonstrated granger (NSCLC bille patients with It use but did not aching nonreplicating 156874816). Potential of the protein and protein	ad to reduction drugs for the tre are several primulate wild-type functions (type functions (type functions) (clinical efficacy) (PMID: 12538 ocally advancedieve a pathologo adenoviral vectitial treatment s, p53 activator supported by da 8 and Vorinost:	or loss of TP53 i atment of cance e-clinical compo ep 53 protein fu PMID:15603511 gene therapy (s in patients with 466, PMID:1646, PMID:30 il breast cancer a jic complete res tor, AdCMV-23 approaches to s, Avastin, or Vo ta in other cancer at in Patients With Solid Tumors anib and Vorino:	this aberration is production. To with TP53 abern unds in development in the control of the co	rations or the not that targ 53 mutant. Several steepersely sing non-sm see 2 rivia, all vive clinical d with the TUY ing Wee1 ment.
cannot rule out the possibility of the presence of germline mutations in the mixed cell population. To rule out the germline contribution, it esting by using peripheral blood sample is recommended. Powered by CollabRx. This steat was developed and its performance characteristics determined by CellNetix Plathology & Laboratories. It has not been approve U.S. Food and Drug Administration. The FDA has determined that such clearance or approval is not necessary. This test is for clinical purposes, it should not be regarded as investigational or for research. This laboratory is regulated under the 1988 CLIA amendments a qualified to perform high-complexity clinical testing. Therapeutic Confidence Level	The effination and the interest of the efficient of the e	ect of this abermanication of the prevented in the selection of the select	ation on protein Tu otein, and may lea are no approved o ytarget p53. There ind work to either si is to resume wild- eported that variou ble patients with It is but did not ach ing nonreplicating 15874816). Pote ors, TP55 vaccine aches are further si 2042989: MLN970 lancies 1748625: MK-1775 1339871: Phase I PE) tissue sections are issue area is subjected.	ad to reduction drugs for the tre are several primulate wild-type functions (type functions (type functions)	or loss of TP53 i atment of cance at inner to fance be clinical components of the co	this aberration is production. The with TP53 abern unds in development of the work of the	rations or the not that targe 53 mutant . Several stage 53 mutant . Several stage generally sing non-sm se 2 trial, all give clinical d with the Tipy ing Wee1 ment
U.S. Food and Drug Administration. The FDA has determined that such clearance or approval is not necessary. This test is for clinical purposes. It should not be regarded as investigational or for research. This laboratory is regulated under the 1988 CLIA amendments a qualified to perform high-complexity clinical testing. Therapeutic Confidence Level	The effination and the interest of the efficient of the e	ect of this aberm cation of the prevail of the prev	ation on protein Tu otein, and may lea are no approved o ytarget p53. There d work to either si is to resume wild- eported that variou ble patients with le se but did not ach ing nonreplicating 15874816). Pote ors, TP55 vaccine aches are further si 2042989: MLN970 lancies 1748625: MK-1775 1339871: Phase I PE) tissue sections are issue area is subjected s approximately 5% mu sole mean for diagnosis enes of this panel doe	ad to reduction drugs for the tre are several primulate wild-type functions (ype functions (ype functions) (yp	or loss of TP53 i atment of cance at ment of cance be clinical compose p53 protein fur PMID:15603511 i gene therapy (s in patients with 456, PMID:15603513 i protein fur patients with 456, PMID:1503 approaches to c s, Avastin, or Votta in other cancer at in Patients With Solid Tumors anib and Vorino: anib and Vorino: a wild-type background a wild-type background are repreted in context weresence of a mutatic endication of contexts.	this aberration is production. The with TP53 abern unds in development of the work of the	rations or the not that targe 53 mutant . Several state e generally sing non-sm see 2 trial, all give clinical d with the Tipy ing Wee1 ment
Therapeutic Confidence Level	METHODOLOG Formalin-fixed, parational, DNA extracted ANALYTICAL S The analytical sensit microdissection. COMMENTS Results of this test or result of not detected Mutations in the out the present of the output from the out	ect of this abern cation of the prevail of the protein have rand resportant of the prevail of th	ation on protein furotein, and may lear on approved of the protein and may lear on approved of the protein and work to either sits to resume wild-reported that various demonstrated grancer (NSCLC ble patients with lease but did not aching nonreplicating 15874816). Potential 15874816 are further sized and sare sare sare six subjected as approximately 5% microsoft sare sare further sized and sare sare sare six subjected as approximately 5% microsoft sare sare soft sare sare sare sare sare sare sare sare	ad to reduction drugs for the tre are several primulate wild-type functions (ype functions (or loss of TP53 i atment of cance at timent of cance pedinical compone p53 protein fur PMID:15603511 i gene therapy (s in patients with 456, PMID:15603511 def, PMID:1503 approaches to c s, Avastin, or Vota in other cancer at in Patients With 1500 and 1500 at a in other cancer at in Patients With 1500 and 1500 at a in other cancer at in Patients With 1500 at a in other cancer at in Patients With 1500 at a in other cancer at in Patients With 1500 at a in other cancer at in Patients With 1500 at a in other cancer at in Patients With 1500 at a in other cancer at in Patients With 1500 at a in other cancer at in Patients With 1500 at a in other cancer at in Patients With 1500 at a in other cancer at in Patients With 1500 at a in other cancer at in Patients With 1500 at a in other cancer at a in other c	this aberration is production. Transwith TP53 aberration is provided by the production or induce produced as Ad. p53) and lung cancer, inclus 8106). In one phase achieved an object set of the production of the	rations or the ant that targe 53 mutant. Several state e generally see 2 trial, all give clinical d with the Ti
	METHOD OLOG Formalin-fixed, paraiload, DNA extracted ANALYTICAL S The analytical sensit microdissection. COMMENTS Results of this test or result of not detected fullations in the region cannot rule out the petaling by using peri	ect of this aberrication of the princation of th	ation on protein furotein, and may be are no approved of the protein function	ad to reduction drugs for the tre are several primulate wild-type functions (ype functions (ype functions) (yp	or loss of TP53 i atment of cance at timent of cance pedinical compone p53 protein fur PMID:15603511 i gene therapy (s in patients with 456, PMID:15603511 i gene therapy (s in patients with 456, PMID:15003) in patients with 456, PMID:15003 in patients with 456, PM	this aberration is production. The system of the system o	rations or the not that targe 53 mutant. Several state 9 generally 1 ling non-sm se 2 trial, all live clinical d with the Tip 1 ling Wee1 ment. Wutuant work of the tumor sis. I characteristics and solve the tumor sis. I characteristics with the several proved the tumor sis.
High Low L	METHOD OLOG Formalin-fixed, parait load, DNA extracted ANALYTICAL S The analytical sensit microdissection. COMMENTS Results of this test or eastly to find teleder Mutations in the region cannot rule out the present of the sensit of the of	ect of this aberrication of the prication of the pricatio	ation on protein furotein, and may leare no approved of are no approved of the protein for the	ad to reduction drugs for the tre are several primulate wild-type functions (ype functions (ype functions) (yp	or loss of TP53 i atment of cance at timent of cance pedinical compone p53 protein fur PMID:15603511 i gene therapy (s in patients with 456, PMID:15603511 i gene therapy (s in patients with 456, PMID:15003) in patients with 456, PMID:15003 in patients with 456, PM	this aberration is production. The system of the system o	rations or the not that targe 53 mutant. Several state 9 generally 1 ling non-sm se 2 trial, all live clinical d with the Tip 1 ling Wee1 ment. Wutuant work of the tumor sis. I characteristics and solve the tumor sis. I characteristics with the several proved the tumor sis.
	METHOD OLOG Formalin-fixed, parari load, DNA extracted ANALYTICAL S The analytical sensit microdissection. COMMENTS Results of this test or result of not detected for the testing by using perigonator rule out the petaling by using perigonator rule of the petaling by using perigonator rule of the petaling by using perigonator rule out the petaling by using perigonator rule of the petaling by using perigonator rule out the petaling by using perigonator rule of the petaling by using perigonator rule out the petaling by using perigonator r	ect of this aberrication of the prication of the pricatio	ation on protein function on protein function and mark proved of are no approved of the protein function of the protein functi	ad to reduction drugs for the tre are several primulate wild-type functions (ype functions (ype functions) (yp	or loss of TP53 i atment of cance atment of cance e-clinical compose p53 protein file gene therapy (s in patients with 466, PMID-16032) breast cancer a jic complete resistor, AdCMV-p53 approaches to c s, Avastin, or Vo. Solid Tumors and band Vorino: AdCMV-p53 approaches to c s, Avastin, or Vo. Solid Tumors and band Vorino: AdCMV-p53 approaches to c s, Avastin, or Vo. Solid Tumors and band Vorino: AdCMV-p53 approaches to c s, Avastin, or Vo. Solid Tumors and band Vorino: AdCMV-p54 approaches to c s, Avastin, or Vo. Solid Tumors and band Vorino: AdCMV-p54 and Vorino: AdCMV-p55 and Vorino: AdCMV-p55 approaches to control to the control of the con	this aberration is production. Transwith TP53 abern unds in development on or induce production or induce production or induce production or induce produced as Ad. p53) and lung cancer, include 8106). In one phalachieved an object 8106, in one phalachieved an object ponse when treate, and chemotheras, onsider include usitation. These treaters. The Advanced p53 has been under the Advanced p53 has been under the decident of some particular and leading the production of some under the decident of some under the decident of some under the 1988 CLIA and aboretories. It has not beneessary. This test is other the 1988 CLIA and the source of the decident of some under the decident of some under the 1988 CLIA and the source of the decident of some under the 1988 CLIA and the source of the source	rations or the not that targe 53 mutant . Several size e generally ling non-sm see 2 trial, all vive clinical divide the clinical see a see a sporce deen approved to crimical size and a control of the clinical size and control of
	METHOD OLOG Formalin-fixed, parari load, DNA extracted ANALYTICAL S The analytical sensit microdissection. COMMENTS Results of this test or result of not detected for the testing by using perigonator rule out the petaling by using perigonator rule of the petaling by using perigonator rule of the petaling by using perigonator rule out the petaling by using perigonator rule of the petaling by using perigonator rule out the petaling by using perigonator rule of the petaling by using perigonator rule out the petaling by using perigonator r	ect of this aberrication of the prication of the pricatio	ation on protein function on protein function and mark proved of are no approved of the protein function of the protein functi	ad to reduction drugs for the tre are several primulate wild-type functions (ype functions (ype functions) (yp	or loss of TP53 i atment of cance atment of cance e-clinical compose p53 protein file gene therapy (s in patients with 466, PMID-16032) breast cancer a jic complete resistor, AdCMV-p53 approaches to c s, Avastin, or Vo. Solid Tumors and band Vorino: AdCMV-p53 approaches to c s, Avastin, or Vo. Solid Tumors and band Vorino: AdCMV-p53 approaches to c s, Avastin, or Vo. Solid Tumors and band Vorino: AdCMV-p53 approaches to c s, Avastin, or Vo. Solid Tumors and band Vorino: AdCMV-p54 approaches to c s, Avastin, or Vo. Solid Tumors and band Vorino: AdCMV-p54 and Vorino: AdCMV-p55 and Vorino: AdCMV-p55 approaches to control to the control of the con	this aberration is production. Transwith TP53 abern unds in development on or induce production or induce production or induce production or induce produced as Ad. p53) and lung cancer, include 8106). In one phalachieved an object 8106, in one phalachieved an object ponse when treate, and chemotheras, onsider include usitation. These treaters. The Advanced p53 has been under the Advanced p53 has been under the decident of some particular and leading the production of some under the decident of some under the decident of some under the 1988 CLIA and aboretories. It has not beneessary. This test is other the 1988 CLIA and the source of the decident of some under the decident of some under the 1988 CLIA and the source of the decident of some under the 1988 CLIA and the source of the source	rations or the not that targe 53 mutant. Several six e generally: ling non-sm see 2 trial, all vive clinical d with the Tuy ing Wee1 ment. Several six e generally: which is the target of the target in the target

- Find patients and provider information here, as well as when we received the specimen for testing.
- Overview of results from the diagnosis, to the biomarker's used during the analysis that have significance in the results.

Actionable = An approved therapy is available
Applicable = An off-label drug or a clinical trial may be available.
Unknown Significance = No clear

Here you will find information regarding the applicable genes specific to this case.

therapy at this time.

- There are hundreds of clinical trials available to which a patient may apply. Our systems cross reference a clinical trial database to find relevant clinical trials specific to the patients genes. Links in the report take you directly to www.clinicaltrials.gov.
- This methodology section provides a brief transparent snapshot of the way we prepare each sample.
- The therapeutic confidence level is our summation of how effective we believe that the clinical trials will be for the targeted gene therapy.
- We always include the direct contact information of the pathologists who diagnosed the case if you have any questions or need further explanation on the diagnosis.