ZETA Corporation

TARGET-VALIDATED AND CHARACTERIZED IVD ANTIBODIES
FOR PATHOLOGY AND IMMUNOTHERAPY

Product Focus

Zeta is very excited and proud to share IVD antibodies researched and developed for Anatomic Pathology market for Immunohistochemistry. Zeta is incorporating highly sensitive technology to develop many of these Monospecific primary antibodies that are Target-Validated and Characterized for IHC on FFPE tissue sections.

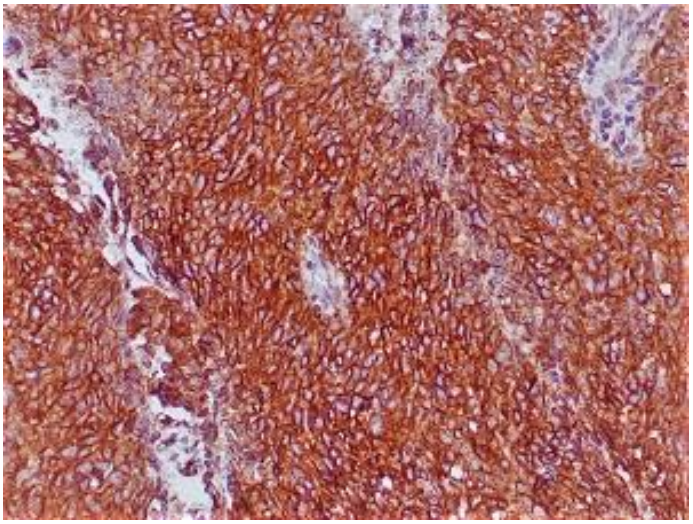
Zeta provides over 300 IVD antibodies for cancer targeted therapy and immunotherapy due to gene mutations, chromosomal translocations or gene amplifications.

CD117 (c-Kit) Rabbit Monoclonal Antibody

ASR (US)/IVD

Anti-rabbit: clone YR145, Cat # Z2030

The c-Kit proto-oncogene is a member of the receptor tyrosine kinase family and, more specifically, is closely related to the platelet derived growth factor receptor (PDGFR). Mutations in Kit are integral for tumor growth and progression in various cancers, such as gastrointestinal stromal tumor (GIST). YR145R a protein of 145kDa. It precipitates both the unoccupied as well as the occupied form of c-kit. C-kit plays an important role in hematopoiesis, melanogenesis, and gametogenesis. Over 98% of GISTs are positive for CD117.



GIST stained with CD117

*Reference:

1. van Oosterom AT, et al. Lancet 2001; 358: 1421-3.
2. Hornick JL, et al. Am J Clin Pathol 2002; 117:188-93
3. Smithey BE, et al. Am J Surg Pathol 2002; 26:486-92

All antibodies are offered in different format and size with the Suffix after the Catalog #s; “L”, “S” & “T” for Concentrated antibodies in 1.0 ml, 0.5 ml & 0.1 ml sizes and Suffix “P” for Ready To Use (RTU) in 7 ml.

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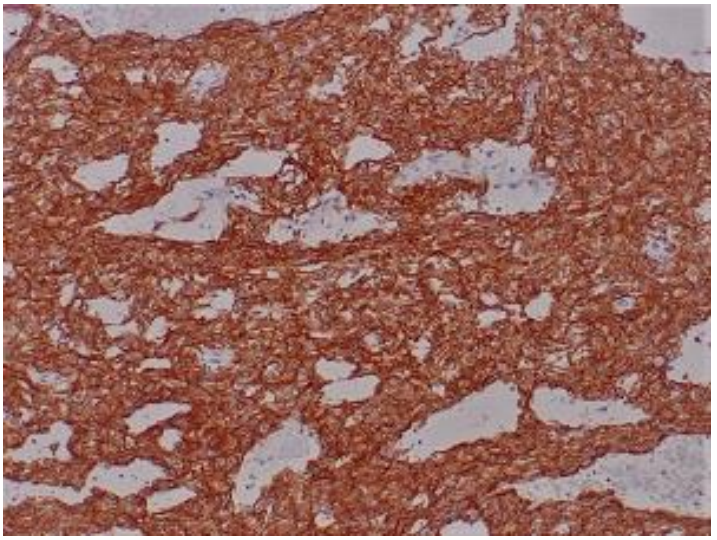
DOG1 Mouse Monoclonal Antibody

IVD

Anti-mouse: clone DOG 1.1, Cat # Z2013

DOG1 gene, a gastrointestinal stromal tumor (GIST) specific gene, encoding for the hypothetical protein FLJ10261, which was named "Discovered on GIST 1" (DOG1). DOG1 protein is expressed ubiquitously in gastrointestinal stromal tumors irrespective of KIT or PDGFRa mutation status. DOG1-1 monoclonal antibody yielded positive staining in 95% GIST. For special GISTs, DOG1 immunoreactivity was detected in 79%, while only 9% stained for CD117; in 36% KIT-negative GISTs; in 100% NF1-associated GISTs; and in 82% pediatric GISTs. In addition, DOG1.1 immunoreactivity was seen in fewer cases of carcinoma, melanoma, and seminoma as compared with KIT mutation. Therefore, DOG1.1 is a sensitive and specific immunohistochemical marker for GIST, comparable with KIT, with the additional benefit of detecting KIT-negative GISTs. DOG1.1 is also a sensitive marker for unusual GIST subgroups lacking KIT or PDGFRA mutations. In tumors that are negative for both KIT and DOG1, mutational screening may be required to confirm the diagnosis of GIST.



GIST stained with DOG.1

*Reference:

1. Kang HG, et al. Mod Pathol. 2011; 24:866-77.
2. Rizzo FM, et al. BMC Cancer. 2016; 16:87.
3. Katoh M, et al. Int J Oncol. 2003; 22:1375-81.

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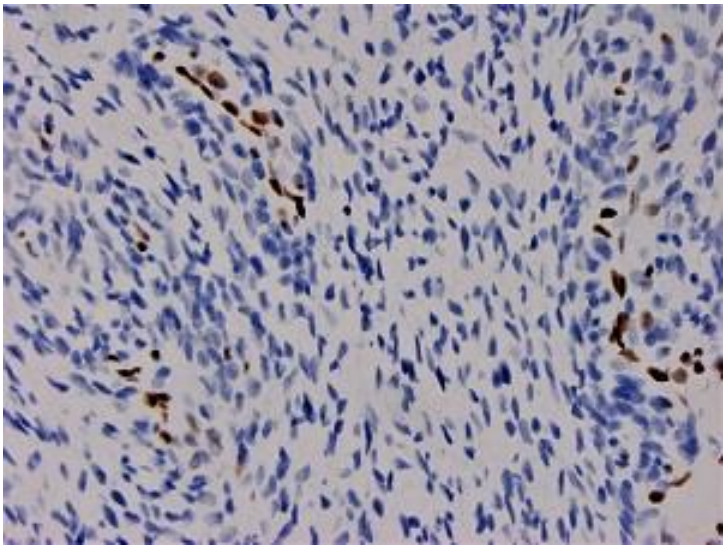
H3K27me3 Rabbit polyclonal Antibody

IVD

Anti-rabbit: Poly, Cat # Z2319

Histone H3 is one of the five main histone proteins involved in the structure of chromatin in eukaryotic cells. The N-terminal tail of histone H3 can undergo several different types of epigenetic modifications that influence cellular processes. These modifications include the covalent attachment of methyl or acetyl groups to lysine and arginine amino acids and the phosphorylation of serine or threonine. Loss-of-function somatic alterations in different components of the polycomb repressive complex 2 (PRC2) occur in the majority of malignant peripheral nerve sheath tumors (MPNSTs). These highly recurrent and specific inactivation of PRC2 components co-occurred with somatic alterations of CDKN2A and NF1. MPNSTs with PCR2 inactivation through EED or SUZ12 alterations showed consistent complete loss of trimethylation at lysine 27 of histone H3 (H3K27me3) by IHC analysis. Approximately 90% of sporadic and radiation associated MPNSTs and 50% NF1-associated MPNSTs show loss of H3K27me3 expression.



MPNST stained with HeK27me3. Note tumor cells negative whereas internal control (endothelial cells positive)

*Reference:

1. Makise N, et al. Am J Surg Pathol. 2017; 41:1523-1531.
2. Busam KJ, et al. Am J Surg Pathol. 2017; 41:396-404.
3. Huang SC, et al. Am J Surg Pathol. 2016; 40:876-85.

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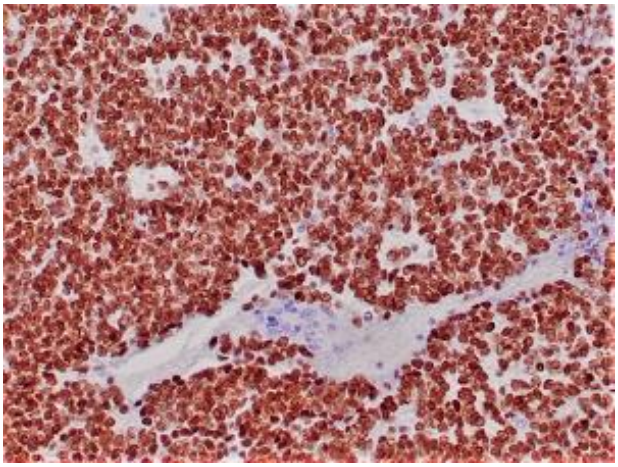
NKX2.2 MonoSpecific Mouse Monoclonal Antibody

IVD

Anti-mouse: clone ZM14, Cat # Z2348

Expression of NKX2.2 has been found in neuroendocrine tumors of the gut, making it a potential marker for the study of gastrointestinal neuroendocrine tumors. More recently, NKX2.2 protein was identified as a target of EWS-FLI-1, the fusion protein specific to Ewing sarcoma, and was shown to be differentially upregulated in Ewing sarcoma on the basis of array-based gene expression analysis. It acts as a valuable marker for Ewing sarcoma, with a sensitivity of 93% and a specificity of 89%, and aids in the differential diagnosis of small round cell tumors.



Ewing's sarcoma stained with NKX2.2

*Reference:

1. Yoshida A, et al. Am J Surg Pathol. 2012; 36:993-9.
2. Smith R, et al. Cancer Cell. 2006; 9:405-16.
3. Hung Y, et al. Mod Pathol. 2016; 29:370-80.

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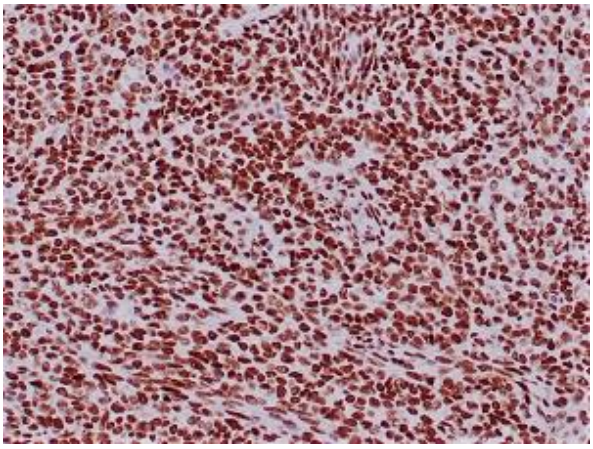
TLE1 MonoSpecific Mouse Monoclonal Antibody

IVD

Anti-mouse: clone ZM93, Cat # Z2403

Key players in the Notch pathway are the TLE genes, which are human homologs of the Drosophila Groucho gene. Groucho is a transcriptional repressor that plays a key role in neurogenesis, segmentation and sex determination. Transducin-like enhancer protein 1 (TLE1) is a protein that is encoded by the TLE1 gene and is involved in control of hematopoiesis, neuronal, and terminal epithelial differentiation. Positive immunohistochemical nuclear staining with anti-TLE-1 has been shown to be a useful addition to an IHC panel when differentiating synovial sarcoma from other soft tissue malignancies.



Synovial sarcoma stained with TLE1

*Reference:

1. Terry J, et al. Am J Surg Pathol. 2007; 31:240-6.
2. Schoolmeester JK, et al. Am J Surg Pathol. 2014; 38:60-5.
3. Jagdis A, et al. Am J Surg Pathol. 2009; 33:1743-51.