Store at -20C	Flightless-I Antibody	C T	Cell Signaling		
		Orders:	877-616-CELL (2355) orders@cellsignal.com		
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418		Web:	info@cellsignal.com cellsignal.com		
1#1		3 Trask Lane Danvers Ma	ssachusetts 01923 USA		

For Research Use Only. Not for Use in Diagnostic Procedures.

Applications: W, IP	Reactivity: H M R Mk	Sensitivity: Endogenous	MW (kDa): 145	Source/Isotype: Rabbit	UniProt ID: #Q13045	Entrez-Gene Id: 2314		
Product Usage Information	e	Application Western Blotting Immunoprecipitation	tium HEPES (nH 7	5) 150 mM NaCl 100 ug	Dilution 1:1000 1:200	vcerol Store at -		
Storage		20°C. Do not aliquot the antibody.						
Specificity/Sensitivity		Flightless-1 Antibody recognizes endogenous levels of total Flightless-I protein.						
Source / Purification		Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to residues near the amino terminus of human Flightless-I protein. Antibodies are purified by protein A and peptide affinity chromatography.						
Background		The <i>flightless-I</i> (<i>flil</i>) gene was first identified in <i>Drosophila</i> mutant screens for genes involved in flight behavior. Homozygous mutant alleles at the <i>flil</i> locus are embryonic lethal, whereas heterozygous mutations yield a "flightless" phenotype resulting from defects in flight muscle fiber development (1). The encoded protein (flightless-I, FLII) is a highly conserved member of the gelsolin superfamily, defined by the presence of C-terminal gelsolin motifs that function as actin-binding domains (2). Genetic knock-out studies in mice and worms confirmed that Flightless-I plays a critical and highly conserved role in embryonic development, likely through its effects on actin remodeling of the cytoskeleton (3,4). Postnatally, Flightless-I is recognized to play an important role in wound repair (5). Flightless-I protein levels are increased in many wound types, and depletion of Flightless-I protein levels has been shown to accelerate wound repair by promoting fibroblast proliferation and epithelial migration (6-8). Studies in animal models suggest that Flightless-I may inhibit the wound repair process by modulating TGF-β signaling dynamics in the wound environment (9).						
Background R	eferences	1. Miklos, G.L. and De 2. Campbell, H.D. et al 3. Campbell, H.D. et al 4. Deng, H. et al. (2007 5. Kopecki, Z. and Cow 6. Cowin, A.J. et al. (200 7. Ruzehaji, N. et al. (200 8. Ruzehaji, N. et al. (200 9. Adams, D.H. et al. (200)	Couet, H.G. (1990) . (1993) <i>Proc Natl /</i> . (2002) <i>Mol Cell Bi</i> () <i>Genetics</i> 177, 84 (in, A.J. (2008) <i>Int J</i> (07) <i>J Pathol</i> 211, 57 (012) <i>Eur J Dermato</i> (013) <i>Biomed Res I</i> (009) <i>Br J Dermato</i>	/ Neurogenet 6, 133-51. (cad Sci U S A 90, 11386-9 ol 22, 3518-26. 7-60. Biochem Cell Biol 40, 141 2-81. / 22, 740-50. nt 2013, 389792. 161, 326-36.	90. 15-9.			
Species Reactivity		Species reactivity is de	termined by testin	g in at least one approve	ed application (e.g.,	western blot).		
Western Blot Buffer		IMPORTANT: For west dry milk, 1X TBS, 0.1%	ern blots, incubate Tween® 20 at 4°C	lots, incubate membrane with diluted primary antibody in 5% w/v nonfat en® 20 at 4°C with gentle shaking, overnight.				
Applications Key		W: Western Blotting IP: Immunoprecipitation						
Cross-Reactivity Key		H: Human M: Mouse R: Rat Mk: Monkey						
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