

# Anti-CRMP2 (C-terminal region) Antibody

Catalog # AN1731

## Product Information

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<b>Application</b>	WB, ICC, IP
<b>Primary Accession</b>	<a href="#">Q16555</a>
<b>Host</b>	Rabbit
<b>Clonality</b>	Rabbit Polyclonal
<b>Isotype</b>	IgG
<b>Calculated MW</b>	62294

## Additional Information

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<b>Gene ID</b>	1808
<b>Other Names</b>	DRP-2, Toad-64, CRMP-62

<b>Target/Specificity</b>	CRMP2 (CRMP-62, TOAD-64, DRP-2) is a microtubule associated protein involved in neuron development and axon pathfinding. CRMP2 binds to tubulin heterodimers and promotes microtubule assembly. The overexpression of CRMP2 facilitates the rate of axonal growth, whereas the mutated form that lacks activity toward the microtubule assembly inhibits axonal growth in a dominant negative manner. Phosphorylation of CRMP2 regulates its activity and this type of regulation has been implicated in axon growth cone collapse induced by several repulsive cues. Cdk5 and GSK3 phosphorylation occurs downstream of the repulsive cue, Sema-3A. Several residues in CRMP2 are phosphorylated by GSK3 (Ser-518, Thr-514, and Thr-509), and a priming site (Ser-522). These sites are conserved in human CRMP1 and CRMP4, but not in CRMP3 or CRMP5. The priming site is also phosphorylated by Cdk5. In contrast, ROCK phosphorylates Thr-555 leading to LPA, MAG, or Ephrin-A5 mediated growth cone collapse. Thus, CRMP2 phosphorylation status may be a critical element of pathways that control axon pathfinding.
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<b>Dilution</b>	WB~~1:1000 ICC~~N/A IP~~N/A
<b>Format</b>	Antigen Affinity Purified
<b>Storage</b>	Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
<b>Precautions</b>	Anti-CRMP2 (C-terminal region) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.
<b>Shipping</b>	Blue Ice

## Background

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CRMP2 (CRMP-62, TOAD-64, DRP-2) is a microtubule associated protein involved in neuron development and

axon pathfinding. CRMP2 binds to tubulin heterodimers and promotes microtubule assembly. The overexpression of CRMP2 facilitates the rate of axonal growth, whereas the mutated form that lacks activity toward the microtubule assembly inhibits axonal growth in a dominant negative manner. Phosphorylation of CRMP2 regulates its activity and this type of regulation has been implicated in axon growth cone collapse induced by several repulsive cues. Cdk5 and GSK3 phosphorylation occurs downstream of the repulsive cue, Sema-3A. Several residues in CRMP2 are phosphorylated by GSK3 (Ser-518, Thr-514, and Thr-509), and a priming site (Ser-522). These sites are conserved in human CRMP1 and CRMP4, but not in CRMP3 or CRMP5. The priming site is also phosphorylated by Cdk5. In contrast, ROCK phosphorylates Thr-555 leading to LPA, MAG, or Ephrin-A5 mediated growth cone collapse. Thus, CRMP2 phosphorylation status may be a critical element of pathways that control axon pathfinding.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.