

**BCR Antibody (N-term) Blocking Peptide**  
**Synthetic peptide**  
**Catalog # BP7113a****Specification**

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**BCR Antibody (N-term) Blocking Peptide - Product Information**Primary Accession [P11274](#)**BCR Antibody (N-term) Blocking Peptide - Additional Information****Gene ID** 613**Other Names**

Breakpoint cluster region protein, Renal carcinoma antigen NY-REN-26, BCR, BCR1, D22S11

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP7113a](/product/products/AP7113a) was selected from the N-term region of human BCR. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

**Format**

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

**Precautions**

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

**BCR Antibody (N-term) Blocking Peptide - Protein Information****Name** BCR ([HGNC:1014](#))**Synonyms** BCR1, D22S11**Function**

Protein with a unique structure having two opposing regulatory activities toward small GTP-binding proteins. The C-terminus is a GTPase-activating protein (GAP) domain which stimulates GTP hydrolysis by RAC1, RAC2 and CDC42. Accelerates the intrinsic rate of GTP hydrolysis of RAC1 or CDC42, leading to down-regulation of the active GTP-bound form (PubMed:[7479768](http://www.uniprot.org/citations/7479768), PubMed:[1903516](http://www.uniprot.org/citations/1903516), PubMed:[17116687](http://www.uniprot.org/citations/17116687)). The central Dbl homology (DH) domain functions as guanine nucleotide exchange factor (GEF) that modulates the GTPases CDC42, RHOA and RAC1. Promotes the conversion of CDC42, RHOA and RAC1 from the GDP-bound to the GTP-bound form (PubMed:[7479768](#)).

href="http://www.uniprot.org/citations/7479768" target="\_blank">7479768</a>, PubMed:<a href="http://www.uniprot.org/citations/23940119" target="\_blank">23940119</a>). The amino terminus contains an intrinsic kinase activity (PubMed:<a href="http://www.uniprot.org/citations/1657398" target="\_blank">1657398</a>). Functions as an important negative regulator of neuronal RAC1 activity (By similarity). Regulates macrophage functions such as CSF1-directed motility and phagocytosis through the modulation of RAC1 activity (PubMed:<a href="http://www.uniprot.org/citations/17116687" target="\_blank">17116687</a>). Plays a major role as a RHOA GEF in keratinocytes being involved in focal adhesion formation and keratinocyte differentiation (PubMed:<a href="http://www.uniprot.org/citations/23940119" target="\_blank">23940119</a>).

#### **Cellular Location**

Postsynaptic density {ECO:0000250|UniProtKB:Q6PAJ1}. Cell projection, dendritic spine {ECO:0000250|UniProtKB:Q6PAJ1}. Cell projection, axon {ECO:0000250|UniProtKB:Q6PAJ1}. Synapse {ECO:0000250|UniProtKB:F1LXF1}

#### **BCR Antibody (N-term) Blocking Peptide - Protocols**

Provided below are standard protocols that you may find useful for product applications.

- [Blocking Peptides](#)

#### **BCR Antibody (N-term) Blocking Peptide - Images**

#### **BCR Antibody (N-term) Blocking Peptide - Background**

A reciprocal translocation between chromosomes 22 and 9 produces the Philadelphia chromosome, which is often found in patients with chronic myelogenous leukemia. The chromosome 22 breakpoint for this translocation is located within the BCR gene. The translocation produces a fusion protein which is encoded by sequence from both BCR and ABL, the gene at the chromosome 9 breakpoint. Although the BCR-ABL fusion protein has been extensively studied, the function of the normal BCR gene product is not clear. The protein has serine/threonine kinase activity and is a GTPase-activating protein for p21rac.

#### **BCR Antibody (N-term) Blocking Peptide - References**

Burchert, A., et al., Blood 103(9):3480-3489 (2004).H, et al., Exp. Hematol. 32(5):476-482 (2004).Sallese, S., et al., Leukemia 18(4):727-733 (2004).Klein, F., et al., J. Exp. Med. 199(5):673-685 (2004).Hsu, H.C., et al., J. Lab. Clin. Med. 143(2):125-129 (2004).