

E2F1 Antibody (S337) Blocking Peptide
Synthetic peptide
Catalog # BP7593a**Specification**

E2F1 Antibody (S337) Blocking Peptide - Product InformationPrimary Accession [Q01094](#)**E2F1 Antibody (S337) Blocking Peptide - Additional Information****Gene ID** 1869**Other Names**

Transcription factor E2F1, E2F-1, PBR3, Retinoblastoma-associated protein 1, RBAP-1, Retinoblastoma-binding protein 3, RBBP-3, pRB-binding protein E2F-1, E2F1, RBBP3

Target/Specificity

The synthetic peptide sequence used to generate the antibody [AP7593a](/products/AP7593a) was selected from the S337 region of human E2F1. 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.

E2F1 Antibody (S337) Blocking Peptide - Protein Information**Name** E2F1 {ECO:0000303|PubMed:8964493, ECO:0000312|HGNC:HGNC:3113}**Function**

Transcription activator that binds DNA cooperatively with DP proteins through the E2 recognition site, 5'-TTTC[CG]CGC-3' found in the promoter region of a number of genes whose products are involved in cell cycle regulation or in DNA replication (PubMed:[10675335](http://www.uniprot.org/citations/10675335), PubMed:[12717439](http://www.uniprot.org/citations/12717439), PubMed:[17704056](http://www.uniprot.org/citations/17704056), PubMed:[17050006](http://www.uniprot.org/citations/17050006), PubMed:[18625225](http://www.uniprot.org/citations/18625225), PubMed:[28992046](http://www.uniprot.org/citations/28992046)). The DRTF1/E2F complex functions in the control of cell-cycle progression from G1 to S phase (PubMed:[10675335](http://www.uniprot.org/citations/10675335)).

PubMed:12717439, PubMed:17704056). E2F1 binds preferentially RB1 in a cell-cycle dependent manner (PubMed:10675335, PubMed:12717439, PubMed:17704056). It can mediate both cell proliferation and TP53/p53- dependent apoptosis (PubMed:8170954). Blocks adipocyte differentiation by binding to specific promoters repressing CEBPA binding to its target gene promoters (PubMed:20176812). Directly activates transcription of PEG10 (PubMed:17050006, PubMed:18625225, PubMed:28992046). Positively regulates transcription of RRP1B (PubMed:20040599).

Cellular Location

Nucleus

E2F1 Antibody (S337) Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

E2F1 Antibody (S337) Blocking Peptide - Images

E2F1 Antibody (S337) Blocking Peptide - Background

E2F1 is a member of the E2F family of transcription factors. The E2F family plays a crucial role in the control of cell cycle and action of tumor suppressor proteins and is also a target of the transforming proteins of small DNA tumor viruses. The E2F proteins contain several evolutionally conserved domains found in most members of the family. These domains include a DNA binding domain, a dimerization domain which determines interaction with the differentiation regulated transcription factor proteins (DP), a transactivation domain enriched in acidic amino acids, and a tumor suppressor protein association domain which is embedded within the transactivation domain. This protein and another two members, E2F2 and E2F3, have an additional cyclin binding domain. This protein binds preferentially to retinoblastoma protein pRB in a cell-cycle dependent manner. It can mediate both cell proliferation and p53-dependent/independent apoptosis.

E2F1 Antibody (S337) Blocking Peptide - References

O'Donnell, K.A., et al., Nature 435(7043):839-843 (2005). Wang, C., et al., J. Biol. Chem. 280(13):12339-12343 (2005). Joshi, B., et al., Oncogene 24(13):2204-2217 (2005). Saberwal, G., et al., Int. J. Hematol. 80(2):146-154 (2004). Chaussepied, M., et al., Mol. Cell 16(5):831-837 (2004).