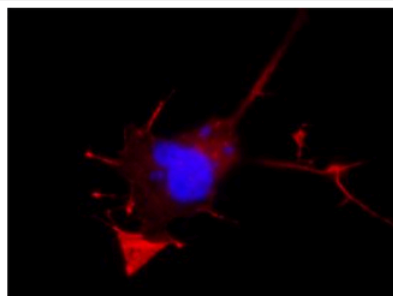


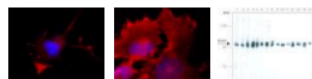
E8655 **Sigma-Aldrich**

Anti-E6AP antibody, Mouse monoclonal

clone E6AP-330, purified from hybridoma cell culture

MDL number [MFCD04034520](#) | NACRES NA.41[SDS](#) [Certificate of Analysis \(COA\)](#) [Specification Sheet](#) [Datasheet \(PDF\)](#)

SKU-Pack Size	Availability	Pack Size	Price (USD)	Quantity
E8655-200UL	Available to ship on 01/05/21 - FROM	200 µL	498.00	<input type="text" value="0"/>

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Properties

Related Categories	Alphabetical Index , Antibodies , Antibodies for Cell Biology , Antibodies for Epigenetics and Nuclear Signaling , Antibodies to E1,E2,E3 Ligase , More...
conjugate	unconjugated
clone	E6AP-330, monoclonal
biological source	mouse
application(s)	immunocytochemistry: suitable immunoprecipitation (IP): suitable indirect ELISA: suitable microarray: suitable western blot: 1-2 µg/mL using total cell extract from 293T cells
species reactivity	human, rat, mouse, monkey
mol wt	antigen ~100 kDa
form	buffered aqueous solution
shipped in	dry ice
storage temp.	−20°C
isotype	IgG1
Quality Level	200
antibody product type	primary antibodies
UniProt accession no.	O75461
Gene Information	human ... E2f6(1876) mouse ... E2f6(50496) rat ... E2f6(313978)

Description

General description
E6AP is an E3 ubiquitin ligase that is expressed by the UB3A gene. Inhibiton or alterations of the UB3A gene may cause a neurological disorder called the Angelman Syndrome. E6AP interacts with E1 and E2 enzymes to mediate ubiquitination of proteins marked for degradation. E6AP also binds with the E6 viral protein present in HPV-infected cells.^{[1][4][2][3]}
Monoclonal Anti-E6AP antibody is a useful tool for the study of E6AP and its function in protein degradation. This antibody is specific for E6AP protein in rat, mouse, human and monkey.

Immunogen
human full-length recombinant E6AP.

Application
Monoclonal Anti-E6AP antibody is suitable for use in western blot (1-2 µg/mL using total cell extract from 293T cells), immunocytochemistry, immunoblotting, immunoprecipitation, indirect ELISA and microarray.

Physical form
Solution in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide.

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Properties

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clone	E6AP-330, monoclonal
biological source	mouse
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storage temp.	-20°C
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Western Blotting refers to the electrophoretic transfer of proteins from sodium dodecyl sulfate polyacrylamide gels to sheets of PVDF or nitrocellulose membrane, followed by immunodetection of prote...
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The HPV-16 E6 and E6-AP complex functions as a ubiquitin-protein ligase in the ubiquitination of p53.

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Cell, 75(3), 495-505 (1993-11-5)

The ubiquitin-dependent proteolytic pathway plays a major role in selective protein degradation. Ubiquitination of proteins requires the sequential action of the ubiquitin-activating enzyme (E1), ubiquitin-conjugating enzymes (E2), and in some cases ...[Read More](#)

Angelman syndrome reviewed from a neurophysiological perspective. The UBE3A-GABRB3 hypothesis.

Read Abstract

B Dan and S G Boyd

Neuropediatrics, 34(4), 169-176 (2003-9-16)

Angelman syndrome is characterised by neurodevelopmental impairment (with or without epileptic seizures) associated with functional deficit of the UBE3A gene. Different mechanisms of UBE3A inactivation correlate with clinical phenotypes of varying se...[Read More](#)

[Targeting E3 ubiquitin ligases for cancer therapy.](#)[Read Abstract](#)

Yi Sun

Cancer biology & therapy, 2(6), 623-629 (2003-12-23)

E3 ubiquitin ligases are a large family of proteins that can be classified into three major structurally distinct types: N-end rule E3s, E3s containing the HECT (Homology to E6AP C-Terminus) domain, and E3s with the RING (Really Interesting New Gene)...[Read More](#)

The role of TP53 in Cervical carcinogenesis. Tommasino, M., et al. Human Mutation 21, 307-312, (2003)[Tissue-specific variation of Ube3a protein expression in rodents and in a mouse model of Angelman syndrome.](#)[Read Abstract](#)

Richard M Gustin et. al

Neurobiology of disease, 39(3), 283-291 (2010-4-29)

Angelman syndrome (AS) is a neurogenetic disorder caused by loss of maternal UBE3A expression or mutation-induced dysfunction of its protein product, the E3 ubiquitin-protein ligase, UBE3A. In humans and rodents, UBE3A/Ube3a transcript is maternally ...[Read More](#)

[Ube3a reinstatement identifies distinct developmental windows in a murine Angelman syndrome model.](#)[Read Abstract](#)

Sara Silva-Santos et. al

The Journal of clinical investigation, 125(5), 2069-2076 (2015-4-14)

Angelman syndrome (AS) is a severe neurodevelopmental disorder that results from loss of function of the maternal ubiquitin protein ligase E3A (UBE3A) allele. Due to neuron-specific imprinting, the paternal UBE3A copy is silenced. Previous studies in...[Read More](#)

[Intracellular Analysis of the Interaction between the Human Papillomavirus Type 16 E6 Oncoprotein and Inhibitory Peptides.](#)[Read Abstract](#)

Christina Stutz et. al

PloS one, 10(7), e0132339 (2015-7-8)

Oncogenic types of human papillomaviruses (HPVs) cause cervical cancer and other malignancies in humans. The HPV E6 oncoprotein is considered to be an attractive therapeutic target since its inhibition can lead to the apoptotic cell death of HPV-posi...[Read More](#)

[Imbalanced mechanistic target of rapamycin C1 and C2 activity in the cerebellum of Angelman syndrome mice impairs motor function.](#)[Read Abstract](#)

Jiandong Sun et. al

The Journal of neuroscience : the official journal of the Society for Neuroscience, 35(11), 4706-4718 (2015-3-20)

Angelman syndrome (AS) is a neurogenetic disorder caused by deficiency of maternally expressed ubiquitin-protein ligase E3A (UBE3A), an E3 ligase that targets specific proteins for proteasomal degradation. Although motor function impairment occurs in...[Read More](#)

[Protein Delivery of an Artificial Transcription Factor Restores Widespread Ube3a Expression in an Angelman Syndrome Mouse Brain.](#)[Read Abstract](#)

Barbara J Bailus et. al

Molecular therapy : the journal of the American Society of Gene Therapy, 24(3), 548-555 (2016-1-5)

Angelman syndrome (AS) is a neurological genetic disorder caused by loss of expression of the maternal copy of UBE3A in the brain. Due to brain-specific genetic imprinting at this locus, the paternal UBE3A is silenced by a long antisense transcript. ...[Read More](#)

[Proteomic discovery of MNT as a novel interacting partner of E3 ubiquitin ligase E6AP and a key mediator of myeloid differentiation.](#)

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Isha Kapoor et. al

Oncotarget, 7(7), 7640-7656 (2015-10-28)

Perturbed stability of regulatory proteins is a major cause of transformations leading to cancer, including several leukemia subtypes. Here, for the first time we demonstrate that E6-associated protein (E6AP), an E3 ubiquitin ligase negatively target...[Read More](#)

[Enhanced Transmission at the Calyx of Held Synapse in a Mouse Model for Angelman Syndrome.](#)

[Read Abstract](#)

Tiantian Wang et. al

Frontiers in cellular neuroscience, 11, 418 (2018-1-23)

The neurodevelopmental disorder Angelman syndrome (AS) is characterized by intellectual disability, motor dysfunction, distinct behavioral aspects, and epilepsy. AS is caused by a loss of the maternally expressed UBE3A gene, and many of the symptoms ...[Read More](#)

[UBE3A-mediated p18/LAMTOR1 ubiquitination and degradation regulate mTORC1 activity and synaptic plasticity.](#)

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Jiandong Sun et. al

eLife, 7, undefined (2018-7-19)

Accumulating evidence indicates that the lysosomal Ragulator complex is essential for full activation of the mechanistic target of rapamycin complex 1 (mTORC1). Abnormal mTORC1 activation has been implicated in several developmental neurological diso...[Read More](#)

[The Angelman syndrome protein Ube3a is required for polarized dendrite morphogenesis in pyramidal neurons.](#)

[Read Abstract](#)

Sheng Miao et. al

The Journal of neuroscience : the official journal of the Society for Neuroscience, 33(1), 327-333 (2013-1-4)

Pyramidal neurons have a highly polarized dendritic morphology, characterized by one long apical dendrite and multiple short basal dendrites. They function as the primary excitatory cells of the mammalian prefrontal cortex and the corticospinal tract...[Read More](#)

[Enhanced Operant Extinction and Prefrontal Excitability in a Mouse Model of Angelman Syndrome.](#)

[Read Abstract](#)

Michael S Sidorov et. al

The Journal of neuroscience : the official journal of the Society for Neuroscience, 38(11), 2671-2682 (2018-2-13)

Angelman syndrome (AS), a neurodevelopmental disorder associated with intellectual disability, is caused by loss of maternal allele expression of UBE3A in neurons. Mouse models of AS faithfully recapitulate disease phenotypes across multiple domains,...[Read More](#)

[E6AP inhibits G-CSFR turnover and functions by promoting its ubiquitin-dependent proteasome degradation.](#)

[Read Abstract](#)

Stuti Chhabra et. al

Biochimica et biophysica acta, 1864(10), 1545-1553 (2017-6-6)

Granulocyte colony-stimulating factor receptor (G-CSFR) plays a crucial role in regulating myeloid cell survival, proliferation, and neutrophilic granulocyte precursor cells maturation. Previously, we demonstrated that Fbw7 α negatively regulates G-CS...[Read More](#)

Adult Ube3a Gene Reinstatement Restores the Electrophysiological Deficits of Prefrontal Cortex Layer 5 Neurons in a Mouse Model of Angelman Syndrome.

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Diana C Rotaru et. al

The Journal of neuroscience : the official journal of the Society for Neuroscience, 38(37), 8011-8030 (2018-8-8)

E3 ubiquitin ligase (UBE3A) levels in the brain need to be tightly regulated, as loss of functional UBE3A protein is responsible for the severe neurodevelopmental disorder Angelman syndrome (AS), whereas increased activity of UBE3A is associated with...[Read More](#)

Felis catus papillomavirus type-2 E6 binds to E6AP, promotes E6AP/p53 binding and enhances p53 proteasomal degradation.

[Read Abstract](#)

Gennaro Altamura et. al

Scientific reports, 8(1), 17529 (2018-12-5)

E6 from high risk human papillomaviruses (HR HPVs) promotes ubiquitination and degradation of p53 tumour suppressor by mediating its binding to ubiquitin ligase E6AP in a ternary complex, contributing to cell transformation in cervical cancer. We hav...[Read More](#)

Novel Insights into the Role of UBE3A in Regulating Apoptosis and Proliferation.

[Read Abstract](#)

Lilach Simchi et. al

Journal of clinical medicine, 9(5), undefined (2020-5-28)

The UBE3A gene codes for a protein with two known functions, a ubiquitin E3-ligase which catalyzes ubiquitin binding to substrate proteins and a steroid hormone receptor coactivator. UBE3A is most famous for its critical role in neuronal functioning....[Read More](#)

Assessing the requirements of prenatal UBE3A expression for rescue of behavioral phenotypes in a mouse model for Angelman syndrome.

[Read Abstract](#)

Monica Sonzogni et. al

Molecular autism, 11(1), 70 (2020-9-20)

Angelman syndrome (AS) is a rare neurodevelopmental disorder caused by the loss of functional ubiquitin protein ligase E3A (UBE3A). In neurons, UBE3A expression is tightly regulated by a mechanism of imprinting which suppresses the expression of the ...[Read More](#)

Quantitative proteomics reveals neuronal ubiquitination of Rngo/Ddi1 and several proteasomal subunits by Ube3a, accounting for the complexity of Angelman syndrome.

[Read Abstract](#)

Juanma Ramirez et. al

Human molecular genetics, 27(11), 1955-1971 (2018-5-23)

Angelman syndrome is a complex neurodevelopmental disorder caused by the lack of function in the brain of a single gene, UBE3A. The E3 ligase coded by this gene is known to build K48-linked ubiquitin chains, a modification historically considered to ...[Read More](#)

Detailed Dissection of UBE3A-Mediated DDI1 Ubiquitination.

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Nagore Elu et. al

Frontiers in physiology, 10, 534 (2019-5-28)

The ubiquitin E3 ligase UBE3A has been widely reported to interact with the proteasome, but it is still unclear how this enzyme regulates by ubiquitination the different proteasomal subunits. The proteasome receptor DDI1 has been identified both in D...[Read More](#)

Structure of E3 ligase E6AP with a proteasome-binding site provided by substrate receptor hRpn10.

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Gwen R Buel et. al

Nature communications, 11(1), 1291 (2020-3-12)

Regulated proteolysis by proteasomes involves ~800 enzymes for substrate modification with ubiquitin, including ~600 E3 ligases. We report here that E6AP/UBE3A is distinguished from other E3 ligases by having a 12 nM binding site at the proteasome co...[Read More](#)

Proteome Instability Is a Therapeutic Vulnerability in Mismatch Repair-Deficient Cancer.

[Read Abstract](#)

Daniel J McGrail et. al

Cancer cell, 37(3), 371-386 (2020-2-29)

Deficient DNA mismatch repair (dMMR) induces a hypermutator phenotype that can lead to tumorigenesis; however, the functional impact of the high mutation burden resulting from this phenotype remains poorly explored. Here, we demonstrate that dMMR-ind...[Read More](#)

UBE3A regulates the transcription of IRF, an antiviral immunity.

[Read Abstract](#)

Ryohei Furumai et. al

Human molecular genetics, 28(12), 1947-1958 (2019-1-29)

UBE3A is a gene responsible for the pathogenesis of Angelman syndrome (AS), a neurodevelopmental disorder characterized by symptoms such as intellectual disability, delayed development and severe speech impairment. UBE3A encodes an E3 ubiquitin ligas...[Read More](#)

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Product Information

Anti-E6AP antibody, Mouse monoclonal

clone E6AP-330, purified from hybridoma cell culture

Product Number **E8655**

Product Description

Anti-E6AP antibody, Mouse monoclonal (mouse IgG1 isotype) is derived from the E6AP-330 hybridoma produced by the fusion of mouse myeloma cells (NS1 cells) and splenocytes from BALB/c mice immunized with the human full-length recombinant E6AP. The isotype is determined by a double diffusion immunoassay using Mouse Monoclonal Antibody Isotyping Reagents, Product Number ISO2.

Monoclonal Anti-E6AP recognizes human, monkey, rat and mouse (approx. 100 kDa). The antibody may be used in ELISA, immunoblotting, immunoprecipitation, and immunocytochemistry.

E6AP belongs to the E3 ubiquitin ligase family and is encoded by the UB3A gene located in chromosome region 15q11-q13. Mutations in this gene or loss of its expression may lead to the Angelman syndrome (AS) that is characterized by neuro developmental impairment.¹⁻³ E6AP protein together with ubiquitin activating enzyme E1 and ubiquitin conjugating enzyme E2, catalyzes the ubiquitination of different protein substrates for targeted degradation via the 26S proteasome. E3 ubiquitin ligases are enzymes that determine the specificity of protein substrates to be marked for degradation. E6AP was originally identified as the ubiquitin-protein ligase involved in human papillomavirus (HPV) E6-mediated p53 degradation and has since been shown to act as an E3 ubiquitin-protein ligase in the ubiquitination of several other protein substrates. E6AP complexes with the E6 viral protein in cells infected with papilloma virus (HPVs). This complex binds to the central region of p53 and as a consequence p53 is ubiquitinated and targeted to the proteasome. Low expression of p53 may lead to several pathological events such as cervical tumor.¹⁻⁴

Monoclonal antibodies to E6AP are an important tool for studying E6AP and its role in protein degradation.

Reagent

Supplied as a solution in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide.

Antibody Concentration: Approx. 2 mg/ml.

Precautions and Disclaimer

This product is for R&D use only, not for drug, household, or other uses. Please consult the Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

For continuous use, store at 2-8 °C for up to one month. For prolonged storage, freeze in working aliquots. Repeated freezing and thawing, or storage in frost-free freezers, is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilution samples should be discarded if not used within 12 hours.

Product Profile

Immunoblotting: a working antibody concentration of 1-2 µg/ml is recommended using total cell extract from 293T cells.

Note: In order to obtain the best results using various techniques and preparations, we recommend determining optimal working dilutions by titration.

References

1. Scheffner, M., et al., *Cell*, **75**, 495-505 (1993).
2. Tommasino, M., et al., *Human Mut.*, **21**, 307-312 (2003).
3. Dan, B., and Boyd, S.G., *Neuroped.*, **34**, 169-176 (2003).
4. Sun, Y., *Cancer Biol.*, **2**, 623-629 (2003).

DS,PHC 01/17-1