Resource Summary Report

Generated by RRID on Aug 18, 2026

hE-cadherin-pcDNA3

RRID:Addgene_45769
Type: Plasmid

Proper Citation

RRID:Addgene_45769

Plasmid Information

URL: http://www.addgene.org/45769

Proper Citation: RRID:Addgene_45769

Insert Name: hE-cadherin

Organism: Homo sapiens

Bacterial Resistance: Ampicillin

Defining Citation: PMID:11381089

Vector Backbone Description: Backbone Marker:Invitrogen; Backbone Size:5446; Vector Backbone:pcDNA3; Vector Types:Mammalian Expression; Bacterial Resistance:Ampicillin

Comments: Partial cDNA for human E-cadherin was provided by D. Rimm (Yale University, New Haven, CT) and subcloned into the pcDNA3 mammalian expression vector (Invitrogen), to generate Addgene plasmid 45772. Sequence analysis revealed that the 3? end of the gene was missing after nucleotide 2644 (according to EMBL/GenBank/DDBJ under accession number L08599). This resulted in a truncation of the last 35 amino acids of the E-cadherin cytoplasmic domain and, as a result, did not contain the ?-catenin binding region, as defined by Stappert and Kemler 1994. The COOH terminus of this truncation mutant (E-cadherin ? ?-catenin) ends at amino acid 844 (NH3-ASLSSD); the frameshift added a single histidine residue before a stop codon is introduced. The full-length human E-cadherin was reengineered using RNA from human A431 cells and the RT-PCR method (Primer A, 5?-TGACACCCGGGACAACGTTTATTA-3 ?, and Primer C, 5?-CTAGTCTAGACCCCTAGTGGTCCTCG-3?) to generate a 425-bp fragment encoding the missing COOH-terminal residues. This fragment was sub-cloned into the truncated hEcad-? 35/pcDNA3 vector (Addgene plasmid 45772) to generate full-length hEcad/pcDNA3 (Addgene plasmid 45769).

Plasmid Name: hE-cadherin-pcDNA3

Record Creation Time: 20220422T222236+0000

Record Last Update: 20260815T081526+0000

Ratings and Alerts

No rating or validation information has been found for hE-cadherin-pcDNA3.

No alerts have been found for hE-cadherin-pcDNA3.

Data and Source Information

Source: Addgene

Usage and Citation Metrics

We found 18 mentions in open access literature.

Listed below are recent publications. The full list is available at RRID .

Kwak JH, et al. (2026) Gapmer Antisense Oligonucleotide Targeting E-Cadherin Rescues Abnormal Keratinization in X-Linked Ichthyosis Models. Biomolecules & therapeutics, 34(1), 213.

Urushima H, et al. (2021) Activation of Hepatic Stellate Cells Requires Dissociation of E-Cadherin-Containing Adherens Junctions with Hepatocytes. The American journal of pathology, 191(3), 438.

Li JN, et al. (2021) E-cadherin Interacts With Posttranslationally-Modified AGO2 to Enhance miRISC Activity. Frontiers in cell and developmental biology, 9, 671244.

Kitchen P, et al. (2020) A Runaway PRH/HHEX-Notch3-Positive Feedback Loop Drives Cholangiocarcinoma and Determines Response to CDK4/6 Inhibition. Cancer research, 80(4), 757.

Tasdemir N, et al. (2020) Proteomic and transcriptomic profiling identifies mediators of anchorage-independent growth and roles of inhibitor of differentiation proteins in invasive lobular carcinoma. Scientific reports, 10(1), 11487.

Wang SC, et al. (2020) Hispanic/Latino Patients with Gastric Adenocarcinoma Have Distinct Molecular Profiles Including a High Rate of Germline CDH1 Variants. Cancer research, 80(11), 2114.

Shields BD, et al. (2019) Loss of E-Cadherin Inhibits CD103 Antitumor Activity and Reduces Checkpoint Blockade Responsiveness in Melanoma. Cancer research, 79(6), 1113.

Yi Y, et al. (2019) Activin A promotes ovarian cancer cell migration by suppressing E-cadherin expression. Experimental cell research, 382(2), 111471.

Martyn I, et al. (2019) A wave of WNT signaling balanced by secreted inhibitors controls primitive streak formation in micropattern colonies of human embryonic stem cells. Development (Cambridge, England), 146(6).

Jolly MK, et al. (2019) E-Cadherin Represses Anchorage-Independent Growth in Sarcomas through Both Signaling and Mechanical Mechanisms. Molecular cancer research : MCR, 17(6), 1391.

Boone PG, et al. (2019) A cancer rainbow mouse for visualizing the functional genomics of oncogenic clonal expansion. Nature communications, 10(1), 5490.

Gaber A, et al. (2018) EpCAM homo-oligomerization is not the basis for its role in cell-cell adhesion. Scientific reports, 8(1), 13269.

Daugaard I, et al. (2017) miR-151a induces partial EMT by regulating E-cadherin in NSCLC cells. Oncogenesis, 6(7), e366.

Xiong S, et al. (2016) Activin B promotes endometrial cancer cell migration by down-regulating E-cadherin via SMAD-independent MEK-ERK1/2-SNAIL signaling. Oncotarget, 7(26), 40060.

Song H, et al. (2016) miR-92a-3p Exerts Various Effects in Glioma and Glioma Stem-Like Cells Specifically Targeting CDH1/?-Catenin and Notch-1/Akt Signaling Pathways. International journal of molecular sciences, 17(11).

Wang S, et al. (2016) PRKAR1A is a functional tumor suppressor inhibiting ERK/Snail/E-cadherin pathway in lung adenocarcinoma. Scientific reports, 6, 39630.

Cui T, et al. (2015) XPC inhibits NSCLC cell proliferation and migration by enhancing E-Cadherin expression. Oncotarget, 6(12), 10060.

Huang TS, et al. (2015) A Regulatory Network Involving ?-Catenin, e-Cadherin, PI3k/Akt, and Slug Balances Self-Renewal and Differentiation of Human Pluripotent Stem Cells In Response to Wnt Signaling. Stem cells (Dayton, Ohio), 33(5), 1419.