



Document details

< Back to results | 1 of 1

Export Download Print E-mail Save to PDF Add to List More... >

International Journal of Oncology
Volume 58, Issue 4, April 2021, Pages 1-12

Distinct and overlapping roles of ARID3A and ARID3B in regulating E2F-dependent transcription via direct binding to E2F target genes (Article)

(Open Access)

Saadat, K.A.S.M.^{a,b}, Lestari, W.^{a,c}, Pratama, E.^a, Ma, T.^a, Iseki, S.^a, Tatsumi, M.^a, Ikeda, M.-A.^a ✉

^aDepartment of Molecular Craniofacial Embryology, Graduate School of Medical and Dental Sciences, Tokyo Medical and Dental University, Tokyo, 113-8549, Japan

^bDepartment of Medical Biology, Faculty of Medicine, Institute of Health Sciences, Gaziantep University, Gaziantep, 27310, Turkey

^cFaculty of Dentistry, International Islamic University Malaysia, Kuantan, 25200, Malaysia

Abstract

View references (43)

The AT-rich interacting domain (ARID) family of DNA-binding proteins is involved in various biological processes, including the regulation of gene expression during cell proliferation, differentiation and development. ARID3A and ARID3B are involved in chromatin remodeling and can bind to E2F1 and retinoblastoma tumor suppressor protein (RB), respectively. However, their role in regulating E2F target gene expression remains poorly understood. E2F transcription factors are critical regulators of cell cycle progression and are modulated by RB. Herein, putative ARID3-binding sites (BSs) in E2F target genes were identified, including Cdc2, cyclin E1 and p107, and it was found that ARID3A and ARID3B bound to these BSs in living cells. The mutation of ARID3 BSs reduced Cdc2 promoter activity, while ARID3A and ARID3B overexpression increased the promoter activity, depending on both ARID3 and E2F BSs. ARID3B knockdown blocked the transcription of Cdc2, cyclin E1 and p107 in normal human dermal fibroblasts (NHDFs), whereas the effects of ARID3A knockdown varied depending on the target genes. ARID3B overexpression, but not that of ARID3A, upregulated the transcription of E2F target genes, and activated cyclin E1 transcription and induced cell death with E2F1 assistance. Finally, ARID3A and ARID3B knockdown attenuated the cell cycle progression of NHDFs and T98G cells, and suppressed tumor cell growth. On the whole, these results indicate that ARID3A and ARID3B play distinct and overlapping roles in E2F-dependent transcription by directly binding to the E2F target genes. The present study provides novel insight into the mechanisms underlying the E2F dysregulation caused by ARID3A and ARID3B overexpression, which may have a significant influence on the progression of tumorigenesis. © 2021 Spandidos Publications. All rights reserved.

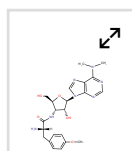
SciVal Topic Prominence ⓘ

Topic: Single Stranded DNA Binding Protein | B-Lymphocyte Subset | Trophoblasts

Prominence percentile: 44.736 ⓘ

Chemistry database information ⓘ

Substances



Author keywords

Metrics ⓘ View all metrics >



PlumX Metrics

Usage, Captures, Mentions, Social Media and Citations beyond Scopus.

Cited by 0 documents

Inform me when this document is cited in Scopus:

Set citation alert >

Related documents

Critical role of ARID3B in the expression of pro-apoptotic p53-target genes and apoptosis

Pratama, E., Tian, X., Lestari, W. (2015) *Biochemical and Biophysical Research Communications*

ARID3A and ARID3B induce stem promoting pathways in ovarian cancer cells

Dausinas, P., Pulakanti, K., Rao, S. (2020) *Gene*

ARID3B directly regulates ovarian cancer promoting genes

Bobbs, A., Gellerman, K., Hallas, W.M. (2015) *PLoS ONE*

View all related documents based on references

Find more related documents in Scopus based on:

Authors > Keywords >

Indexed keywords

EMTREE drug terms:

ARID3A protein ARID3B protein caspase 3 caspase 7 complementary DNA
cyclin dependent kinase 1 DNA binding protein protein p107 protein p27 protein p53
short hairpin RNA small interfering RNA transcription factor E2F unclassified drug

EMTREE medical terms:

amino acid sequence apoptosis Article binding site carcinogenesis Cdc2 gene
cell aging cell cycle progression cell proliferation controlled study cyclin E1 gene
disease association DNA cross linking DNA sequencing DNA synthesis enzyme activity
gene gene expression genetic transcription glioblastoma human human cell
in vitro study mRNA expression level newborn p107 gene priority journal
promoter region protein DNA binding protein expression protein function
skin fibroblast T98G cell line transactivation transcription initiation
transcription initiation site transcription regulation upregulation

Chemicals and CAS Registry Numbers:

caspase 3, 169592-56-7; caspase 7, 189258-14-8; protein p107, 150549-31-8

Funding details

Funding sponsor	Funding number	Acronym
Japan Society for the Promotion of Science See opportunities by KAKEN	JP19K10259,JP21390502,JP24659870	KAKEN

Funding text

The present study was supported by JSPS KAKENHI (grant nos. JP21390502, JP24659870 and JP19K10259).

ISSN: 10196439

CODEN: IJONE

Source Type: Journal

Original language: English

DOI: 10.3892/ijo.2021.5192

PubMed ID: 33649863

Document Type: Article

Publisher: Spandidos Publications

References (43)

[View in search results format >](#)

All Export Print E-mail Save to PDF Create bibliography

- 1 Dimova, D.K., Dyson, N.J.
The E2F transcriptional network: Old acquaintances with new faces ([Open Access](#))
(2005) *Oncogene*, 24 (17), pp. 2810-2826. Cited 533 times.
doi: 10.1038/sj.onc.1208612
[View at Publisher](#)

- 2 DeGregori, J., Johnson, D.G.
Distinct and overlapping roles for E2F family members in transcription, proliferation and apoptosis
(2006) *Current Molecular Medicine*, 6 (7), pp. 739-748. Cited 372 times.
<http://www.ingentaconnect.com/content/ben/cmm/2006/00000006/00000007/art00006>
doi: 10.2174/1566524010606070739
[View at Publisher](#)