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Volume 106, Issue 6, 1 December 2017, Pages 811-819**Imbalanced expression of polycistronic miRNA in acute myeloid leukemia** (Article)Kotaki, R.^a, Higuchi, H.^a, Ogiya, D.^a, Katahira, Y.^a, Kurosaki, N.^a, Yukihira, N.^a, Ogata, J.^a, Yamamoto, H.^a, Mohamad Alba, S.^c, Azhim, A.^d, Kitajima, T.^b, Inoue, S.^b, Morishita, K.^b, Ono, K.^b, Koyama-Nasu, R.^a,  Kotani, A.^{a,b} ^aDivision of Hematological Malignancy, Institute of Medical Sciences, Tokai University, 143 Shimokasuya, Isehara, Kanagawa, Japan^bDepartment of Hematology and Oncology, Tokai University School of Medicine, Isehara, Kanagawa, Japan^cDepartment of Electronic Systems Engineering, Malaysia-Japan International Institute of Technology, University of Technology Malaysia, Kuala Lumpur, Malaysia[View additional affiliations >](#)

Abstract

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miR-1 and miR-133 are clustered on the same chromosomal loci and are transcribed together as a single transcript that is positively regulated by ecotropic virus integration site-1 (EV1). Previously, we described how miR-133 has anti-tumorigenic potential through repression of EV1 expression. It has also been reported that miR-1 is oncogenic in the case of acute myeloid leukemia (AML). Here, we show that expression of miR-1 and miR-133, which have distinct functions, is differentially regulated between AML cell lines. Interestingly, the expression of miR-1 and EV1, which binds to the promoter of the miR-1/miR-133 cluster, is correlative. The expression levels of TDP-43, an RNA-binding protein that has been reported to increase the expression, but inhibits the activity, of miR-1, were not correlated with expression levels of miR-1 in AML cells. Taken together, our observations raise the possibility that the balance of polycistronic miRNAs is regulated post-transcriptionally in a hierarchical manner possibly involving EV1, suggesting that the deregulation of this balance may play some role in AML cells with high EV1 expression. © 2017, The Japanese Society of Hematology.

Author keywords

AML EV1 miR-1 miR-133

Indexed keywords

EMTREE drug terms:

doxorubicin microRNA microRNA 1 microRNA 133 TAR DNA binding protein unclassified drug
DNA binding protein MDS1 and EV1 complex locus protein MECOM protein, human microRNA
MIRN1 microRNA, human MIRN133 microRNA, human RNA TDP-43 protein, human tumor protein

EMTREE medical terms:

acute myeloid leukemia acute myeloid leukemia cell line Article cell survival cell viability cistron controlled study
drug sensitivity ecotropic virus integration site 1 gene gene gene overexpression human human cell
promoter region protein expression acute myeloid leukemia biosynthesis gene expression regulation genetics
HL-60 cell line metabolism multigene family THP-1 cell line U-937 cell line

MeSH:

DNA-Binding Proteins Gene Expression Regulation, Leukemic HL-60 Cells Humans Leukemia, Myeloid, Acute
MDS1 and EV1 Complex Locus Protein MicroRNAs Multigene Family Neoplasm Proteins RNA, Neoplasm THP-1 Cells
U937 Cells

Chemicals and CAS Registry Numbers:

doxorubicin, 23214-92-8, 25316-40-9; RNA, 63231-63-0;

DNA-Binding Proteins; MDS1 and EV1 Complex Locus Protein; MECOM protein, human; MicroRNAs; MIRN1 microRNA, human; MIRN133 microRNA, human; Neoplasm Proteins; RNA, Neoplasm; TDP-43 protein, human

Drug tradename:

adriamycin

Funding details

Funding number	Funding sponsor	Acronym
	Japan Agency for Medical Research and Development	AMED
	Japan Society for the Promotion of Science	JSPS
	Japan Agency for Medical Research and Development	AMED

Funding text

Acknowledgements We thank all our lab members for helpful discussions and continuous encouragement, and the entire staff of the Education and Research Support Center at Tokai University for technical assistance. This work was supported by the Japan Society for the Promotion of Science, Japan Leukemia Research Fund and the Research Program on Hepatitis from Japan Agency for Medical Research and Development, AMED.

ISSN: 09255710
CODEN: IJHEE
Source Type: Journal
Original language: EnglishDOI: 10.1007/s12185-017-2314-1
PubMed ID: 28831750
Document Type: Article
Publisher: Springer Tokyo

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