



Title	Functional analysis of miR-199a-3p in tumor endothelial cells
Author(s)	洪, 諭瑩
Description	この博士論文の全文は利用可能日以降に公開されます。利用可能日以前の閲覧方法は https://www.lib.hokudai.ac.jp/dissertations/copy-guides/ をご参照ください。
Degree Grantor	北海道大学
Degree Name	博士(歯学)
Dissertation Number	甲第16940号
Issue Date	2026-03-25
DOI	https://doi.org/10.14943/doctoral.k16940
Doc URL	https://hdl.handle.net/2115/99785
Type	doctoral thesis
File Information	Yuying_Hong_abstract.pdf, 論文内容の要旨 https://creativecommons.org/licenses/by/4.0/



学位論文内容の要旨

Dissertation Abstract

博士の専攻分野の名称：博士（歯学）

氏名:Hong Yuying

学 位 論 文 題 名

Functional analysis of miR-199a-3p in tumor endothelial cells

（ 腫瘍内皮細胞における miR-199a-3p の機能解析 ）

Keywords (5) miR-199a-3p , Tumor endothelial cells , CD151 , angiogenesis , Cell migration and invasion

Tumor endothelial cells (TECs) are known to play a key role in tumor growth and metastasis, exhibiting distinct phenotypic and functional features compared to normal endothelial cells (NECs). Although microRNAs (miRNAs) have been reported to play diverse roles in tumor progression, the differences in miRNA expression profiles between TECs and NECs and their roles in TEC phenotype remain unclear.

In this study, we isolated NECs and TECs from non-tumor and tumor regions of human renal cancer tissues and conducted miRNA array analysis. Among 13 differentially expressed miRNAs, miR-199a-3p was the most highly expressed in TECs. Overexpression of miR-199a-3p in NECs significantly enhanced their proliferation, migration, and invasion.

Target gene analysis using four databases identified CD151 as a potential target of miR-199a-3p. CD151 expression level was downregulated in TECs compared to NECs, and miR-199a-3p-transfection decrease CD151 expression level in NECs. CD151 knockdown by siRNA promoted cell proliferation, migration, invasion, and reduced cell adhesion to extracellular matrix. In addition, both miR-199a-3p overexpression and CD151 silencing upregulated MMP2 expression in TECs.

These results suggest that miR-199a-3p is involved in proangiogenic phenotype in TECs by suppressing CD151 expression.