

Accepted Manuscript

miR-3065-5p regulates mouse odontoblastic differentiation partially through bone morphogenetic protein receptor type II

Chujiao Li, Qian Zhang, Shuaitong Yu, Yuxiu Lin, Shuchen Li, Huan Liu, Zhi Chen



PII: S0006-291X(17)32203-9

DOI: [10.1016/j.bbrc.2017.11.026](https://doi.org/10.1016/j.bbrc.2017.11.026)

Reference: YBBRC 38819

To appear in: *Biochemical and Biophysical Research Communications*

Received Date: 23 October 2017

Accepted Date: 3 November 2017

Please cite this article as: C. Li, Q. Zhang, S. Yu, Y. Lin, S. Li, H. Liu, Z. Chen, miR-3065-5p regulates mouse odontoblastic differentiation partially through bone morphogenetic protein receptor type II, *Biochemical and Biophysical Research Communications* (2017), doi: 10.1016/j.bbrc.2017.11.026.

This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

miR-3065-5p regulates mouse odontoblastic differentiation partially through bone morphogenetic protein receptor type II

Chujiao Li ^a, Qian Zhang ^a, Shuaitong Yu ^a, Yuxiu Lin ^a, Shuchen Li ^a,

Huan Liu ^{a,*}, Zhi Chen ^{a,*}

a): State Key Laboratory Breeding Base of Basic Science of Stomatology (Hubei-MOST) and Key Laboratory for Oral Biomedicine of Ministry of Education (KLOBM), School and Hospital of Stomatology, Wuhan University, Wuhan, China, 430079.

* Corresponding authors: Huan Liu and Zhi Chen

State Key Laboratory Breeding Base of Basic Science of Stomatology (Hubei-MOST) and Key Laboratory for Oral Biomedicine of Ministry of Education (KLOBM), School and Hospital of Stomatology, Wuhan University, Wuhan, China, 430079.

E-mail: liu.huan@whu.edu.cn (Huan Liu), zhichen@whu.edu.cn (Zhi Chen)

Phone: 86-27-87686198

Fax: 86-27-87873849

Abstract:

Illumination of the molecular mechanisms regulating odontoblastic differentiation of dental papilla cells is of great significance for proper dentinogenesis and dental pulp regeneration. In this study, we discovered that microRNA (miR)-3065-5p is up-regulated during odontoblastic differentiation. Overexpression of miR-3065-5p promoted odontoblastic differentiation *in vitro*. Dual luciferase report assay verified that miR-3065-5p could bind to the 3'UTR of bone morphogenetic protein receptor type II (BMPR2), which dramatically increased in the beginning of odontoblastic differentiation but decreased in the terminal differentiation stage. Inhibition of *Bmpr2* in the early stage retarded odontoblastic differentiation while knockdown of *Bmpr2* in the terminal stage enhanced odontoblastic differentiation, resembling the effect of miR-3065-5p. Taken together, our present study suggests that miR-3065-5p positively regulates odontoblastic differentiation by directly binding to *Bmpr2* in the terminal differentiation stage.

Key words: miR-3065-5p, Odontoblast, *Bmpr2*, Differentiation

1. Introduction:

Odontogenesis is regulated by an intricate network of cell-to-cell signaling during all developmental steps. The specific functions of the key signaling pathways during tooth development have been widely investigated[1]. Signaling by members of the bone morphogenetic protein (BMP) family has been shown to be critical for many aspects of tooth development. In addition, BMPs fulfill many diverse functions during tooth development through crosstalk with other signaling pathways, such as Shh signaling, Wnt/ β -catenin signaling, FGF and Notch signaling[2]. BMPs transduce their signals by combining different types of receptors. Three type I BMP receptors (BMPR1A, BMPR1B,

Download English Version:

<https://daneshyari.com/en/article/8295671>

Download Persian Version:

<https://daneshyari.com/article/8295671>

[Daneshyari.com](https://daneshyari.com)