



## Correction to: *Dgcr8* knockout approaches to understand microRNA functions in vitro and in vivo

Wen-Ting Guo<sup>1</sup> · Yangming Wang<sup>2</sup>

Published online: 29 April 2019  
© Springer Nature Switzerland AG 2019

**Correction to: Cellular and Molecular Life Sciences**  
<https://doi.org/10.1007/s00018-019-03020-9>

The section: “miRNA-independent functions of DICER” was missed between the section “miRNA-independent functions of DROSHA and DGCR8” and the section “The *Dgcr8* knockout strategy to study miRNA functions” in the original publications.

### miRNA-independent functions of DICER

DICER plays critical roles in miRNA processing by cleaving pre-miRNAs into mature miRNAs [14–16]. In addition, DICER is well known for its function in the generation of endogenous small-interfering RNAs [82–84]. This activity is essential for repressing the expression or activity of repeat sequences including L1 retrotransposons, Alu repeats as well short interspersed nuclear elements [85, 86]. In certain cases, small RNAs processed from repeat sequences may have specific biological functions. Human DR2 Alu repeat derived small RNAs promote the differentiation of human pluripotent stem cells by down-regulating important stem cell mRNAs, including NANOG [87]. Other than repeat associated double-strand RNAs, DICER also processes tRNAs and RNAs with hairpin-like structures [51, 88, 89]. Although DICER is considered as mainly located in cytoplasm [90], recent studies show that DICER may also

have nuclear functions. Immunoprecipitation assay shows that nuclear DICER interacts with RNA polymerase II at actively transcribed gene loci [91]. Nuclear DICER has transcriptional regulation potential and restricts the production of endogenous double-strand RNAs that are toxic to most mammalian cells [91]. In addition, nuclear DICER is found to promote the formation of shorter alternative polyadenylation (APA) isoform of ETNK1 by binding DNA sequences close to its proximal polyadenylation site [92]. A plausible mechanism is proposed that DICER promotes heterochromatin formation at associated sites by enhancing EHMT2 binding, in turn slowing down the progression of RNA polymerase II, which favors the usage of proximal polyadenylation site.

**Publisher's Note** Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

The original article can be found online at <https://doi.org/10.1007/s00018-019-03020-9>.

✉ Yangming Wang  
yangming.wang@pku.edu.cn

<sup>1</sup> Central Laboratory, Peking Union Medical College Hospital, Peking Union Medical College and Chinese Academy of Medical Sciences, No. 1 Shuaifuyuan, Beijing 100730, People's Republic of China

<sup>2</sup> Beijing Key Laboratory of Cardiometabolic Molecular Medicine, Institute of Molecular Medicine, Peking University, Beijing 100871, People's Republic of China