MHA MEDIHEALTH ACADEMY
Surgery on Children
Letter to the Editor
doi: 10.51271/SOC-0073

"microRNAs (miRNAs) targeting oxidant-antioxidant mechanisms in primary ciliary dyskinesia and cystic fibrosis: epigenetic insights into therapeutic biomarkers"

Yasemin Kartal

Department of Physiology, Faculty of Medicine, Kırklareli University, Kırklareli, Turkiye

Received: 17/02/2026 ◆ **Accepted**: 11/03/2026 ◆ **Published**: 27/04/2026

Cite this article: Kartal Y. "microRNAs (miRNAs) targeting oxidant-antioxidant mechanisms in primary ciliary dyskinesia and cystic fibrosis: epigenetic insights into therapeutic biomarkers". *Surg Child*. 2026;3(2):69-70. doi:10.51271/SOC-0073

Corresponding Author: Yasemin Kartal, yagmuryaseminkartal@gmail.com

Keywords: Cystic fibrosis, miRNA, oxidative stress, primary ciliary dyskinesia

Dear Editor,

Primary ciliary dyskinesia (PCD) is a rare disorder marked by abnormalities in ciliary ultrastructure and/or function which are associated with disrupted mucociliary clearance and give rise to recurrent or persistent respiratory tract infections, bronchiectasis, bronchitis as well as a broad spectrum of clinical manifestations including sinonasal disease, neonatal respiratory distress, infertility, laterality defects and heterotaxy.[1]

Cystic fibrosis (CF) is also classified as a rare disease and is characterized by a defective CF transmembrane conductance regulator (CFTR) gene, whereas more than 40 disease-causing genes have been implicated in PCD to date.[2] CFTR dysfunction is associated with altered homeostasis in various organs and biological systems, including the pancreas, salivary glands, gastrointestinal tract, reproductive organs, and the respiratory system.[2,3] Within the framework of pulmonary homeostasis, impairment of mucociliary clearance constitutes a crucial pathogenic mechanism in CF which is linked to a sustained hyperinflammatory milieu and progressive pulmonary injury. Additionally, a close interconnection between inflammatory responses and the oxidant–antioxidant equilibrium was evident, as demonstrated by the significant correlations observed recently in our study.[4] Accordingly, levels of the intracellular antioxidant enzyme glutathione peroxidase (GPx) were significantly associated with the pro-inflammatory cytokines interleukin-1β (IL-1β), IL-6, and IL-8 in patients with PCD and CF.[4] From this perspective, the pro-inflammatory/-oxidant milieu and cellular antioxidant mechanisms represent pivotal targets for potential therapeutic strategies in patients with PCD and CF.

CF treatment is guided by symptomatic/supportive therapeutic modalities and novel therapeutic strategies including ion channel-targeted therapies, small-molecule approaches, and gene-based therapies.[2] Meanwhile, PCD exhibits a comparatively lower prevalence in relation to CF, notably among Caucasian populations and its clinical management continues to be predominantly guided by CF-derived therapeutic frameworks.[1,2]

It is noteworthy that microRNAs (miRNAs), a class of small non-coding RNAs, have garnered increasing attention in recent years as a potential therapeutic strategy in CF, due to their roles in modulating physiological functions and mediating disease-related pathophysiological mechanisms.[3] Among these miRNAs, miR-155 is regarded as a pivotal pro-inflammatory miRNA in patients with CF, with its increased expression being related with elevated IL-8 expression. In this context, miR-155-based therapeutic approaches can represent a promising strategy to restrict inflammatory responses by attenuating IL-8 mediated inflammatory processes.[5] Given the functional crosstalk between oxidative stress and inflammation, miRNAs associated with oxidant–antioxidant homeostasis have the potential to provide a relevant conceptual framework for research in both PCD and CF. Mitochondrial dynamics are well recognized as being indispensable for the maintenance of oxidant-antioxidant balance. In this context, miRNAs targeting genes associated with mitochondrial dynamics were assessed using bioinformatic approaches based on miRNA-target prediction tools, including TargetScan, Diana-microT and miRDB in this current study. Consequently, hsa-miR-369-3p, hsa-miR-381-3p and hsa-miR-382-3p were identified as candidate

miRNAs with the potential to regulate key mitochondrial dynamics related genes, including mitofusin 1 (MFN1), MFN2, and optic atrophy 1 (OPA1).

In conclusion, a comprehensive understanding of miRNAs as epigenetic regulators of genes related to mitochondrial dynamics reflecting oxidant-antioxidant equilibrium, particularly hsa-miR-369-3p, hsa-miR-381-3p and hsa-miR-382-3p, may provide critical insights to improve therapeutic outcomes and offer substantial translational relevance in these rare diseases.

ETHICAL DECLARATIONS

Peer Review Process

This letter was externally peer-reviewed.

Conflict of Interest

The author declare no conflicts of interest.

Financial Disclosure

No financial support was received for the preparation or publication of this letter.

Author Contributions

The author is solely responsible for the entirety of conception, execution, analysis, and writing of the manuscript.

REFERENCES

1. Despotes KA, Zariwala MA, Davis SD, Ferkol TW. Primary ciliary dyskinesia: a clinical review. *Cells*. 2024;13(11):974. doi:10.3390/cells13110974
2. Pereira R, Barbosa T, Cardoso AL, Sá R, Sousa M. Cystic fibrosis and primary ciliary dyskinesia: similarities and differences. *Respir Med*. 2023;209:107169. doi:10.1016/j.rmed.2023.107169
3. Finotti A, Gambari R. Perspectives in microRNA therapeutics for cystic fibrosis. *Noncoding RNA*. 2025;11(1):3. doi:10.3390/ncrna11010003
4. Kartal Y, Bozdemir Özel C, Çakmak A, et al. The relationship between lung function, exercise capacity, oxidant and antioxidant response in primary ciliary dyskinesia and cystic fibrosis. *Turk J Pediatr*. 2024;66(3):309-322. doi:10.24953/turkjpediatr.2024.4581
5. Bhattacharyya S, Balakathiresan NS, Dalgard C, et al. Elevated miR-155 promotes inflammation in cystic fibrosis by driving hyperexpression of interleukin-8. *J Biol Chem*. 2011;286(13):11604-11615. doi:10.1074/jbc.M110.198390