 DOI: https://doi.org/10.5114/ceji.2024.145408

How BATF silencing protects neonatal lungs in sepsis

ELIZA GLODKOWSKA-MROWKA[1], MARLENA TYNECKA[2], ANDRZEJ ELJASZEWICZ[2]

[1]Department of Hematological and Transfusion Immunology, Institute of Hematology and Transfusion Medicine, Warsaw, Poland
[2]Centre of Regenerative Medicine, Medical University of Bialystok, Bialystok, Poland

(Cent Eur J Immunol 2024; 49 (3): 215)

Neonatal sepsis (NS) continues to pose a critical challenge in neonatology, characterized by high morbidity and mortality rates despite significant advancements in medical care. This severe condition frequently triggers systemic inflammation, multi-organ dysfunction, and life-threatening complications, including acute lung injury [1, 2]. A recent study by Jihui Zhang and Huimin Jiang provides valuable insights into the molecular mechanisms underlying NS-associated lung damage, emphasizing the pivotal role of the BATF-COTL1 signaling axis [3].

Sepsis is characterized by a dysregulated immune response, often involving key transcription factors and signaling pathways contributing to the immune response and organ failure [4, 5]. The study presents coactosin-like protein 1 (COTL1), a key regulator of actin cytoskeleton dynamics and inflammatory pathways, as a central mediator in NS pathogenesis. Using a cecal slurry (CS)-induced model of neonatal sepsis and *in vitro* experiments with lipopolysaccharide (LPS)-stimulated pulmonary endothelial cells, the authors demonstrate that silencing COTL1 significantly mitigates lung injury [3]. This protective effect is attributed to reduced inflammation, apoptosis, and oxidative stress, hallmark processes driving NS-induced tissue damage.

Beyond providing mechanistic insights, the study underscores the translational potential of targeting the BATF-COTL1 axis for therapeutic intervention. These findings advocate for precision medicine approaches to address the complex molecular drivers of NS-associated complications [3]. The identification of BATF and COTL1 as potential therapeutic targets highlights the need for further preclinical and clinical research to validate their efficacy in modulating inflammation and oxidative stress in neonates.

In conclusion, the work of Jihui Zhang and Huimin Jiang marks an advancement in neonatal critical care and respiratory research.

References

1. Kim F, Polin RA, Hooven TA (2020): Neonatal sepsis. BMJ 371: m3672.
2. Shane AL, Sánchez PJ, Stoll BJ (2017): Neonatal sepsis. Lancet 390: 1770-1780.
3. Zhang J, Jiang H (2024): Knockdown of BATF alleviates lung injury in septic neonates through transcriptional regulation of COTL1. Cent Eur J Immunol 49: 238-251.
4. McGovern M, Giannoni E, Kuester H, et al. (2020): Challenges in developing a consensus definition of neonatal sepsis. Pediatr Res 88: 14-26.
5. Molloy EJ, Bearer CF (2022): Paediatric and neonatal sepsis and inflammation. Pediatr Res 91: 267-269.

Correspondence: Andrzej Eljaszewicz, PhD, Centre of Regenerative Medicine, Medical University of Bialystok, Waszyngtona 15 B, 15-269 Białystok, Poland, e-mail: ceji@umb.edu.pl