haematologica
Journal of the Ferrata Storti Foundation

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Received: November 5, 2026.
Accepted: November 6, 2026.

Citation: Jiahuan Li and Wenya Li. Functional DNase assessment in posttransplant lung injury. Comment on: "Neutrophil extracellular traps and DNase I levels in the bronchoalveolar lavage fluid of children with pulmonary injury after allogeneic hematopoietic stem cell transplantation: a TRANSPIRE study". Haematologica. 2026 Nov 12. doi: 10.3324/haematol.2026.302282 [Epub ahead of print]

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Functional DNase assessment in posttransplant lung injury. Comment on: "Neutrophil extracellular traps and DNase I levels in the bronchoalveolar lavage fluid of children with pulmonary injury after allogeneic hematopoietic stem cell transplantation: a TRANSPIRE study"

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Running head: Functional DNase testing after transplantation

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Author contributions

Jiahuan Li reviewed the literature, analyzed the published evidence, and drafted the manuscript. Wenya Li supervised the work, interpreted the findings, and critically revised the manuscript.

Funding and conflicts of interest

No funding was received for this work. The authors declare no conflicts of interest.

Data availability

No new data were generated or analyzed for this correspondence.

To the Editor:

We commend Strecker and colleagues for characterizing extracellular DNA and DNase I in bronchoalveolar lavage fluid after hematopoietic stem cell transplantation.[1] Their analysis brings attention to the local pulmonary environment and identifies a clinically relevant direction for exploring targeted treatment after transplant-associated lung injury.

Building on this work, we respectfully ask: how might the proposed high-DNA, low-DNase I subgroup be complemented by a functional assessment of whether adding DNase restores DNA degradation? Connecting protein abundance to the response to enzyme supplementation could help refine the biological basis for selecting patients for subsequent intervention studies.

Among 48 posttransplant samples with DNA concentrations above the median, 18 had DNase I concentrations below the median.[1] These observations provide a rationale for investigating local enzyme supplementation. Protein concentration and DNA-degrading capacity, however, describe different properties. The same group previously measured plasma DNase activity using plasmid degradation after transplantation and discussed the potential role of inhibitory factors.[2] Actin is a well-established inhibitor of DNase I,[3] making the activity of added enzyme within the airway sample itself a particularly informative question.

Could the authors consider pairing DNase I concentration with a standardized DNA-degradation assay and an ex vivo supplementation experiment? Matched aliquots could be assessed under the same incubation conditions with and without a defined amount of recombinant DNase I, alongside a buffer control for enzyme activity. Measuring the remaining DNA over a specified interval would distinguish baseline degradation from the additional degradation achieved after supplementation. Consistent sample processing, dilution, and storage conditions would be important for comparing these responses across specimens.

This approach could distinguish several biologically informative patterns. A low-protein sample with restored degradation after supplementation would provide direct support for a correctable local enzyme deficit. A sample with substantial protein but limited baseline activity could still show a response to added enzyme. Conversely, little improvement after supplementation would direct attention to the sample environment and potential inhibition. Relating these patterns to the reported pulmonary-event categories could help define which patients should be prioritized for a subsequent treatment study and which local biological measurements would be most useful alongside clinical outcomes.

We thank the authors for establishing this pulmonary biomarker framework and would welcome their views on integrating concentration, activity, and response to supplementation when developing DNase-based interventions after transplantation.

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