



Comment to “Molecular approach to the classification of chronic fibrosing lung disease—there and back again”

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Dear Editors,

As a compulsive, picky, and avid reader, I have been pleased to read in the journal, over the years, novelties in all fields of pathology, a sign of a robust health.

Among these, I just read a well-written and comprehensive review of the chronic fibrosing lung diseases [1]. This field has been revolutionized by single-cell biology techniques [2, 3], hardly a surgical pathologist-friendly topic, and by a lightning-fast preprint flood, rather than manuscripts on peer-reviewed journals.

What struck me, however, was the use in the review of the term “Krebs von den Lungen-6 (KL-6)” for a well-studied biomarker.

KL-6 is an insular term for an internationally defined and codified protein, MUC1 (Mucin 1) [4]. Other names for MUC1 are epithelial membrane antigen (EMA), Ca15-3, CD227, and etc.

An international effort to define a common nomenclature for leukocyte antigens (human leukocyte differentiation antigens; HLDA), to which I did participate, began in 1982 [5], followed by a similar effort to adopt a common nomenclature for mucins [4] and other molecules. The aim of these initiatives was to implement a standard nomenclature for unique proteins over multiple independent assays and research fields. Nobody these days uses Leu2 for CD8; analogously, one should not use KL-6 for MUC1 or, at least, reference in parenthesis the designed standard name.

I believe that pathology journals should join the efforts, spearheaded by more generalistic journals, to enforce the use of such standard nomenclature, in order to favor cross-field research, comparison, and immediate recognition of unique entities. The future of pathology in a rapidly evolving

“multi-omic” scientific field, as shown by the forceful entrance of single-cell transcriptomics in lung fibrosis, is at stake.

Author contribution Solely the author.

Declarations

Conflict of interest The author declares no competing interests.

References

1. Verleden SE, Braubach P, Kuehnel M, Dickgreber N, Brouwer E, Tittmann P, et al (2020) Molecular approach to the classification of chronic fibrosing lung disease—there and back again. *Virchows Arch*. <https://doi.org/10.1007/s00428-020-02964-9>
2. Adams TS, Schupp JC, Poli S, Ayaub EA, Neumark N, Ahangari F et al (2020) Single-cell RNA-seq reveals ectopic and aberrant lung-resident cell populations in idiopathic pulmonary fibrosis. *Sci Adv* 6(28):eaba1983. <https://doi.org/10.1126/sciadv.aba1983>
3. Habermann AC, Gutierrez AJ, Bui LT, Yahn SL, Winters NI, Calvi CL et al (2020) Single-cell RNA sequencing reveals profibrotic roles of distinct epithelial and mesenchymal lineages in pulmonary fibrosis. *Sci Adv* 6(28):eaba1972. <https://doi.org/10.1126/sciadv.aba1972>
4. Apostolopoulos V, Stojanovska L, Gargosky SE (2015) MUC1 (CD227): a multi-tasked molecule. *Cell Mol Life Sci* 72(23):4475–4500. <https://doi.org/10.1007/s00018-015-2014-z>
5. Bernard A, Boumsell L (1984) Human leukocyte differentiation antigens. *Presse Med* 13(38):2311–2316

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