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Letter to the Editor

Letter to the Editor: Cystatin C-derived measures of renal function as risk factors for mortality and renal replacement therapy in the critically ill - an analysis of the SWECRIT cohort

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The recent article by Linné et al., entitled "Cystatin C-derived measures of renal function as risk factors for mortality and renal replacement therapy in the critically ill – An analysis of the SWECRIT cohort," presents a rigorous and commendable analysis of a large ICU cohort. While the authors should be applauded for the quality of their work, the following commentary is offered.

In their discussion, the authors note: "Interestingly, we did not find the same association between shrunken pore syndrome (SPS) and mortality in adjusted analyses in non-sepsis patients. The reasons for this result are not readily apparent," and they speculate that the observed difference is most likely due to variations in pathophysiology between sepsis and non-sepsis patients [1]. As they mention, SPS has been associated with the accumulation of proteins of similar molecular size to cystatin C, which may contribute to mortality in inflammatory conditions such as sepsis. This mechanism might not play an equally significant role in non-septic patients [1–3].

However, the conclusion that SPS-related mortality is limited to septic states is disputed. In a recent cohort study, Dardashti et al. demonstrated a significant association between SPS and increased mortality in patients undergoing elective coronary artery bypass grafting (CABG), despite the absence of sepsis-level inflammatory responses. This suggests that the pathophysiological mechanisms underlying SPS may not be restricted to inflammatory conditions. They proposed an alternative hypothesis: SPS may share pathophysiological similarities with preeclampsia, particularly involving endothelial dysfunction [4]. Since preeclampsia is known to be reversible and amenable to treatment, this raises the possibility that SPS might also be modifiable if the underlying endothelial mechanisms are better understood [5].

In conclusion, contrary to the assertion by Linné et al., SPS may indeed occur in non-septic patients and may be associated with adverse outcomes independent of a pronounced inflammatory response. Further investigation into the underlying mechanisms, including causes of death and endothelial dysfunction, is warranted.

CRediT authorship contribution statement

Thibault Gennart: Conceptualization, Writing – original draft, Writing – review & editing. **Julien Moury:** Conceptualization, Writing – original draft, Writing – review & editing. **Sydney Blackman:** Writing – original draft, Writing – review & editing. **Nathan De Lissnyder:** Writing – original draft, Writing – review & editing. **Ilann Oueslati:** Writing – original draft, Writing – review & editing. **Patrick M. Honore:** Conceptualization, Supervision, Writing – original draft, Writing – review & editing.

Consent for publication

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Ethics approval and consent to participate

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Declaration of competing interest

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