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# *Letter to the Editor*

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## Microvascular leakage of albumin in inflammatory states: in reply to Dr Frisbie

First of all, I would like to thank Dr Frisbie for his interest in our work.

It should be noted that the principle aim of our work was to demonstrate the close relationship between anemia and hypoalbuminemia and the presence of pressure sores, because one of the criticisms moved to our previous article on this issue[1] was the possibility that the alterations we found could not be related to the presence of pressure sores, SCI being a multifaceted disease, which could imply several metabolic changes. The second item we wanted to clarify was that these two metabolic alterations are due to the chronic inflammatory state induced by the pressure sore and not by a leakage of blood and serum from the ulcer.

With regard to the first problem, we believe we managed to demonstrate our hypothesis by showing the fast release of hypoalbuminemia and anemia after the surgical intervention for pressure sore healing. With regard to the second issue, we are glad that Dr Frisbie agrees with our thesis of the relationship between albumin decrease and the inflammatory state due to pressure sores. We partially disagree with Dr Frisbie with regard to the nature of this relationship: a decreased synthesis of albumin has been considered the cause of hypoalbuminemia in several inflammatory conditions such as bowel disease[2] and renal disease that requires hemodialysis,[3] and in patients with acute conditions such as sepsis, trauma, burns or after an extensive operative procedure.[4] Being inflammatory states a very complex condition, other pathogenic factors should be considered and we agree that a redistribution of albumin with leakage to extravascular space could be a cause of hypoalbuminemia too.

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## References

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