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To the editor:

Acidic and neutral sialidase in the erythrocytes of patients with type 2 diabetes: an answer to comments by Richard et al

In a recent letter to the editor by Richard et al,[1] the letter authors made some comments about our work published early this year.[2] In our study we observed a sharp decrease of neutral sialidase activity on the surface of erythrocytes of diabetic patients, which accounts at the same time for the significant increase (40%) of sialic acid content. At the end of our discussion, we hypothesized that the higher negative charge at the erythrocyte surface due to this increase results in a premature sequestration of diabetic red cells by macrophages, in accordance with the data reported by Mazzanti et al[3] and Jain et al.[4] Our hypothesis has been criticized by Richard and coworkers because it conflicts with the notion that a reduction of total sialic acid content is responsible for phagocytosis of senescent red cells.

Our thoughts on this matter are as follows: (1) The hypothesis reported by Richard et al has been extensively debated over the years,[5] and contrary to what the authors hint, it is not the only one known nor the most accepted, nonetheless it was not in our intentions to discredit it. (2) We believe in the importance of sialic acid in the process of recognition of senescent red cells, but as part of a more complex process, where other molecules are involved, as suggested in other hypotheses.[6] Indeed, according to Beppu et al[7] and Kannan et al,[8] the molecular consequences of the oxidative damage occurring in senescent erythrocytes are likely responsible for their clearance. We think that the reduction of sialic acid content in specific domains of the surface, and not its overall decrease, may trigger the macrophage recognition.[9,10] (3) The theory reported by Richard et al is eventually unsuitable to explain our experimental results. In fact, if the overall sialic acid decrease was responsible for senescent erythrocytes recognition, we should have observed an increase in life span of erythrocytes in diabetes mellitus, yet we observed the opposite phenomenon.

In conclusion we would like to emphasize once again that it was not in our intentions to invalidate the role of sialic acid decrease in erythrocyte removal, even though we do believe in a different hypothesis on erythrocytes senescence, at least in diabetic patients.

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To the editor:

Increased CMV infection following nonmyeloablative allogeneic stem cell transplantation: a search for the guilty

I read with interest the brief report by Bainton et al on cytomegalovirus (CMV) reactivation following the use of Campath-based nonmyeloablative conditioning regimens.[1] The authors found a high incidence of CMV infection, similar to that reported by us.[2] However, they suggest that fludarabine rather than Campath was responsible for this, but the existing literature on nonmyeloablative transplants does not seem to support the idea. A recent study reported CMV reactivation in 87% with and only 25% without the addition of alemtuzumab (Campath-1H) ($P < .001$) to fludarabine-melphalan regimen.[3] The Seattle group did not find a difference in the incidence of CMV infection or disease with the addition of fludarabine to the low-dose radiation regimen.[4] Similar low