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LETTER TO THE EDITOR

Vox Sanguinis ISBT International Society of Blood Transfusion

Response to 'Unlikely influence of ABO blood group polymorphism on antibody response to COVID-19 mRNA vaccine against SARS-CoV-2 spike protein'

We, the authors, want to thank the working group of Yamamoto et al., which provided interesting insights into the interpretation of our results [1, 2]; in our study, we failed to identify a significant difference in antibody titres among patients of different blood groups who underwent a full mRNA vaccination course (ComiRNAty PFIZER-BioNTech).

We did not assess the influence of ABO type on natural SARS-CoV-2 infection. Our work was based on the hypothesis that there could be differences in the antibody response elicited by the vaccine, regardless of the A/B glycosylation of the spike protein.

Other authors have also hypothesized a difference in the behaviour of the immune system in response to COVID-19 vaccines in patients with different blood types. In a letter from Sgherza et al., the authors start from the same hypothesis and describe how, even in their sample, there are no differences in response to the ComiRNAty vaccine based on the patient's ABO group [3].

These data are consistent with the assertion by Yamamoto et al. that the absence of pre-existing glycosylation in mRNA-induced spike proteins avoids an ABO-based difference in immune response following vaccination.

Yamamoto et al. also provide an overview of the effect that the glycosylation of the spike protein of SARS-CoV-2, induced by the blood group of the host in which the virus is replicating, can have both on infectivity and clinical outcome.

These statements add to a growing body of literature, which, similarly to SARS-CoV, suggests that patients with blood group A are at greater risk of infection than group O patients. Amoroso et al. found, in a study conducted on transplant patients or on transplant wait-listed individuals, that blood group A patients had a higher incidence of COVID-19, possibly due to a lack of protection induced by the absence of anti-A agglutinins.

If the fact that group O patients are at lower risk of infection fits well with the protection afforded by agglutinins against viruses generated in ABO individuals not identical to the host, protection from disease progression once the individual is infected (and therefore produces viruses with ABO identical glycosylation) could instead be explained by a lower propensity to thrombosis in group O patients.

In a recent paper, Sardu et al. compared the incidence of cardiac injury, pro-thrombotic index levels and death among group O versus non-O hypertensive and COVID-19 patients; in the non-O group, the incidence of these pathologies was significantly increased [4]. As this study is focalized on O versus non-O patients, applying the considerations discussed by Yamamoto et al., the results could also be attributed to the reduced levels of von Willebrand factor and factor VIII in group O patients [5].

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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