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Letter to the Editor

Evaluation of Serum Amyloid A as a Marker of Persistent Inflammation in Patients with Rheumatoid Arthritis

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We read with great interest the paper by Targońska-Stępniak and Majdan titled "Serum Amyloid A as a Marker of Persistent Inflammation and an Indicator of Cardiovascular and Renal Involvement in Patients with Rheumatoid Arthritis" in which the investigators reported that high serum amyloid A (SAA) concentration was strongly associated with activity of the disease and risk of cardiovascular and renal involvement in patients with rheumatoid arthritis [1]. We thank the authors for their detailed report. However, we wish to make some comments on SAA.

SAA is produced in the liver in response to proinflammatory cytokines such as tumor necrosis factor alpha (TNF-α), interleukin-1 (IL-1), and IL-6 [2]. Previous studies suggested that several diseases such as ankylosing spondylitis, autoinflammatory diseases (Hashimoto's thyroiditis, familial Mediterranean fever), major depression, systemic lupus erythematosus, diabetes mellitus, inflammatory bowel diseases, acute pancreatitis, several types of vasculitis, psoriasis, and epilepsy could affect SAA levels [3, 4]. In addition to these diseases, glucocorticoids, statins, disease-modifying antirheumatic drugs, corticosteroids, and nonsteroidal anti-inflammatory drugs could alter SAA levels [5, 6]. Also, dietary food supplements such as antioxidants (ascorbic acid, taurine, and phytic acid), vitamin E, vitamin A, α linoleic acid, omega-3 fatty acids, and polyunsaturated fatty acids can influence SAA levels [7, 8]. In this respect, without defining these contributing factors, interpreting the results is problematic.

Alcohol use and smoking status of participants are other confounding factors for SAA measurement [9, 10]. Therefore, these factors have to be expressed and a multivariate regression analysis should be applied to show whether these variables have an impact on SAA levels.

In conclusion, clarifying these concerns will certainly provide a clearer picture when interpreting SAA levels among participants.

Conflict of Interests

The authors declare no conflict of interests.

References

[1] B. Targońska-Stępniak and M. Majdan, "Serum amyloid A as a marker of persistent inflammation and an indicator of cardiovascular and renal involvement in patients with rheumatoid arthritis," *Mediators of Inflammation*, vol. 2014, Article ID 793628, 7 pages, 2014.

[2] J. C. H. van der Hilst, "Recent insights into the pathogenesis of type AA amyloidosis," *TheScientificWorldJournal*, vol. 11, pp. 641–650, 2011.

[3] L. Obici, S. Raimondi, F. Lavatelli, V. Bellotti, and G. Merlini, "Susceptibility to AA amyloidosis in rheumatic diseases: a critical overview," *Arthritis Care & Research*, vol. 61, no. 10, pp. 1435–1440, 2009.

[4] G. Li, F. Ren, J. Yao, M. Wang, X. Feng, and D. Liu, "Human serum amyloid A (SAA) protein changes in acute epilepsy

patients," *International Journal of Neuroscience*, vol. 123, no. 4, pp. 265–268, 2013.

[5] T. Fushimi, Y. Takahashi, Y. Kashima et al., "Severe protein losing enteropathy with intractable diarrhea due to systemic AA amyloidosis, successfully treated with corticosteroid and octreotide," *Amyloid*, vol. 12, no. 1, pp. 48–53, 2005.

[6] Y. Horiuchi, S. Hirayama, S. Soda et al., "Statin therapy reduces inflammatory markers in hypercholesterolemic patients with high baseline levels," *Journal of Atherosclerosis and Thrombosis*, vol. 17, no. 7, pp. 722–729, 2010.

[7] D. Giugliano, A. Ceriello, and K. Esposito, "The effects of diet on inflammation: emphasis on the metabolic syndrome," *Journal of the American College of Cardiology*, vol. 48, no. 4, pp. 677–685, 2006.

[8] A. Spreafico, L. Millucci, L. Ghezzi et al., "Antioxidants inhibit SAA formation and pro-inflammatory cytokine release in a human cell model of alkaptonuria," *Rheumatology (United Kingdom)*, vol. 52, no. 9, pp. 1667–1673, 2013.

[9] B. S. Pruett and S. B. Pruett, "An explanation for the paradoxical induction and suppression of an acute phase response by ethanol," *Alcohol*, vol. 39, no. 2, pp. 105–110, 2006.

[10] A. I. Al-Sieni, A. I. Al-Alawy, Z. S. Al-Shehri, and F. A. Al-Abbasi, "Serum amyloid-a protein and serum rheumatoid Factor as serological surrogate markers for smoking risk factor in Saudi population," *Pakistan Journal of Pharmaceutical Sciences*, vol. 26, no. 2, pp. 239–243, 2013.