**INTRODUCTION**

Serum amyloid A (SAA) is a sensitive inflammatory marker rapidly increased during the acute phase, followed by a steady decline to physiological levels during resolution. While resolution and SAA reduction have been documented, the exact mechanism remains elusive. Although antibodies against SAA (anti-SAA) have been previously identified in healthy blood donors (HBDs) in smaller, preliminary studies, their potential function is still unclear.1,2

**OBJECTIVES**

To detect anti-SAA and anti-SAA1 in the sera of 300 HBDs using ELISA, characterise their subclasses and avidity. Additionally, we aimed to evaluate their presence in intravenous immunoglobulin (IVIG) and potential effects on released IL-6 from SAA-treated peripheral blood mononuclear cells (PBMCs).

**METHODS**

An in-house ELISA was adapted from Rosenau and Schur1 and developed2 for detection of anti-SAA and anti-SAA1. Both antibody fractions were isolated from IVIG using MicroLink Protein Coupling Kit (Thermo Scientific). PBMCs were purified from 5 HBDs by density gradient centrifugation and stimulated with SAA or SAA1 (1.5 μg/ml) in the presence/absence of anti-SAA and anti-SAA1 for 5 hours, 37°C. IL-6 concentration was measured in supernatants by ELISA (Invitrogen). The median (IQR) absorbance in HBDs was 0.655 (0.262–1.293) for anti-SAA and 0.493 (0.284–0.713) for anti-SAA1. Both anti-SAA and anti-SAA1 reached peak levels between 41–50 years and diminished with age, with women exhibiting significantly higher levels than men. Good positive correlation was observed between anti-SAA and anti-SAA1. Both antibodies were prevalently of the IgG subclass, with heterogeneous to high avidity and were detected also in IVIG. Stimulation of PBMCs with SAA significantly induced IL-6 release (mean ±SD) (385.9±184.4 pg/ml) with levels decreasing significantly upon addition of 4.5 (131.4±44.4 pg/ml) or 9.0 μg/ml (118.1±57.4 pg/ml) anti-SAA. A similar trend was also found for SAA1 and anti-SAA1.

**CONCLUSIONS**

Anti-SAA could play a physiological role in down-regulating proinflammatory activity of SAA and could represent an attractive, novel therapeutic option for patients with chronic inflammatory diseases.

**REFERENCES**


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