**Supplementary table 2** -Cardiovascular and metabolic parameters explained.

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| **Marker** | **Function** |
| ADMA (µM) | ADMA interferes with L-arginine in the production of [nitric oxide](https://en.wikipedia.org/wiki/Nitric_oxide) (NO), a key chemical involved in normal [endothelial](https://en.wikipedia.org/wiki/Endothelium) function and, by extension, [cardiovascular](https://en.wikipedia.org/wiki/Circulatory_system) health. |
| E-selectin (ng/mL) | E-selectin is an adhesion receptor involved in slowing down [leukocyte rolling](https://www.sciencedirect.com/topics/biochemistry-genetics-and-molecular-biology/leukocyte-rolling) and its expression is restricted to endothelial cells. Its expression on [endothelial cells](https://www.sciencedirect.com/topics/medicine-and-dentistry/endothelial-cell) is transcriptionally upregulated by various proinflammatory substances such as IL-1, TNFα and [lipopolysaccharide](https://www.sciencedirect.com/topics/medicine-and-dentistry/lipopolysaccharide) (LPS). E-selectin may also be transcriptionally regulated by [oxidative stress](https://www.sciencedirect.com/topics/immunology-and-microbiology/oxidative-stress): for example, in [atherosclerosis](https://www.sciencedirect.com/topics/immunology-and-microbiology/atherosclerosis). |
| P-selectin (ng/mL) | P-Selectin is expressed in platelets and [endothelial cells](https://www.sciencedirect.com/topics/medicine-and-dentistry/endothelial-cell), and plays an essential role in the initial recruitment of [leukocytes](https://en.wikipedia.org/wiki/Leukocytes) to the site of injury during [inflammation](https://en.wikipedia.org/wiki/Inflammation). When endothelial cells are [activated](https://en.wikipedia.org/wiki/Endothelial_activation) by molecules such as histamine or thrombin during inflammation, P-selectin moves from an internal cell location to the endothelial cell surface. |
| ICAM-1 (pg/mL) | ICAM-1 is an [endothelial](https://en.wikipedia.org/wiki/Endothelium)- and [leukocyte](https://en.wikipedia.org/wiki/Leukocyte)-associated transmembrane protein facilitating leukocyte endothelial transmigration. ICAM-1 facilitates a variety of immune responses. |
| ICAM-3 (ng/mL) | ICAM-3 is expressed on all resting leukocytes and, and is important for [leukocyte aggregation and activation](https://www.sciencedirect.com/topics/medicine-and-dentistry/leukocyte-activation). |
| VCAM-1 (pg/mL) | VCAM-1 is expressed in endothelial cells in response to cytokines (e.g., TNFα and IL-1β) and mediates adhesion of leukocytes. Increased ICAM-3 and VCAM-1 blood levels reflects their expression on the blood vessel walls and should be considered a clinical sign of developing inflammation-dependent disorders, including atherosclerosis. |
| MCP-1 (pg/mL) | MCP-1 is the most important [chemokine](https://www.sciencedirect.com/topics/medicine-and-dentistry/chemokine) that regulates migration and infiltration of monocytes/macrophages. MCP‐1 functions in the development of atherosclerosis by recruiting monocytes into the subendothelial cell layer. |
| Il-6 (pg/mL) | [IL-6](https://www.sciencedirect.com/topics/medicine-and-dentistry/interleukin-6) is a [proinflammatory cytokine](https://www.sciencedirect.com/topics/biochemistry-genetics-and-molecular-biology/proinflammatory-cytokine). It is also a stimulator of [acute-phase proteins](https://www.sciencedirect.com/topics/medicine-and-dentistry/acute-phase-protein) such as CRP. IL-6 is elevated in the [adipose tissue](https://www.sciencedirect.com/topics/medicine-and-dentistry/adipose-tissue) and plasma of obese subjects. IL-6 decreased after [weight loss](https://www.sciencedirect.com/topics/biochemistry-genetics-and-molecular-biology/body-weight-loss) in the adipose tissue and the plasma. Plasma IL-6 levels are associated with insulin resistance and risk of type 2 diabetes. |
| Il-8 (pg/mL) | [Interleukin-8](https://www.sciencedirect.com/topics/medicine-and-dentistry/interleukin-8) (IL-8) is one of the major mediators of the inflammatory response.  IL-8 is associated with [obesity](https://en.wikipedia.org/wiki/Obesity). |
| TNF-α (pg/mL) | TNF-α is synthesized in adipose tissues by adipocytes and other cells in the tissue matrix. Blood levels and adipocyte production of TNF-α correlate with BMI and hyperinsulinemia, and TNF-α impairs insulin action by inhibiting insulin signaling. |
| TNFR-1 (pg/mL) | The receptors of TNF-a (TNFR-1 and TNFR-2) are involved in the regulation of insulin signaling and insulin resistance in adipocytes, TNFR-2 has also been observed to increase the differentiation of pre-adipocytes, and the concentration of soluble TNFR-2 is found to be more elevated in obese patients than the concentration of TNFR-1. |
| TNFR-2 (pg/mL) |
| SAA (pg/mL) | SAA refers to a family of [apolipoproteins](https://www.sciencedirect.com/topics/medicine-and-dentistry/apolipoprotein) synthesized in the liver and is associated with high-density [lipoproteins](https://www.sciencedirect.com/topics/agricultural-and-biological-sciences/lipoprotein) (HDL) in plasma. SAA has been related to cholesterol transport and recycling. |
| CRP (fg/mL) | CRP is a general marker for inflammation and infection and is strongly related to total and central abdominal obesity, blood pressure, and lipid levels. |
| TM (ng/mL) | Thrombomodulin (TM) is an endothelial cell-associated cofactor for thrombin-mediated activator of protein C. Plasma TM level is regarded as a primary molecular marker reflecting injury of [endothelial cells](https://www.sciencedirect.com/topics/medicine-and-dentistry/endothelial-cell). |
| Apo A-1 (g/L) | Apo A-1 ensures the transport of the HDL (high-density lipoprotein). It absorbs excess cholesterol and transports it to the liver. The amount of Apo A-1 increases as the amount of HDL in the blood increases. |
| Apo B-100 (g/L) | Apo B-100 is part of the fat transporter particles VLDL (very low density lipoprotein) and LDL (low density lipoprotein) that transport cholesterol and triglycerides to the tissues. The amount of Apo B increases as the amount of LDL in the blood increases. It is made in the liver. |
| Apo C-2 (mg/mL) | Apo C-2 is a component of [very low density lipoproteins](https://en.wikipedia.org/wiki/Very_low_density_lipoprotein) and [chylomicrons](https://en.wikipedia.org/wiki/Chylomicron). This protein activates the enzyme [lipoprotein lipase](https://en.wikipedia.org/wiki/Lipoprotein_lipase) in capillaries, which hydrolyzes triglycerides and transfers the fatty acids to tissues. |
| Apo C-3 (mg/mL) | Apolipoprotein C-III, secreted from the liver and to a lesser extent by the intestines, is a component of both HDL and Apo B containing lipoprotein particles, impairs catabolism and hepatic uptake of Apo B-containing lipoproteins, appears to enhance the catabolism of HDL particles, enhances monocyte adhesion to vascular endothelial cells, and activates inflammatory signaling pathways. An increase in Apo C-3 levels induces the development of [hypertriglyceridemia](https://en.wikipedia.org/wiki/Hypertriglyceridemia). |
| Apo B-48 (ng/mL) | Apo B-48 is part of the fat transporter particles VLDL (very low density lipoprotein) and LDL (low density lipoprotein) that transport cholesterol and triglycerides to the tissues. The amount of Apo B increases as the amount of LDL in the blood increases. It is made in the small intestine. |

**Abbreviations:** ADMA, plasma amino acid and dimethylarginine; ICAM, intercellular adhesion molecule; VCAM, vascular cell adhesion molecule; MCP, Monocyte Chemo-attractant Protein; IL, interleukin; TNF, tumor necrosis factor; TNFR, TNF-receptor; SAA, serum amyloid A protein; CRP, C-reactive protein; TM, thrombomodulin; Apo, Apolipoprotein.