serum cytokine levels. Moreover, patients with a low caffeine intake seemed to have a more severe disease phenotype⁶.

**Objectives:** The aim of this study was to evaluate the role of caffeine intake on endothelial function in SLE patients, by assessing its effect on number and function of EPCs both ex vivo in SLE patients and in vitro in healthy donors (HD) treated with SLE sera.

**Methods:** We performed a cross-sectional study enrolling consecutive SLE patients (revised 1997 ACR criteria), referring to the Sapienza Lupus Clinic. Patients with history of traditional CV risks factors were excluded. Caffeine intake was evaluated using a 7-day food frequency questionnaire. At the end of questionnaire filling circulating EPCs were detected by using a flow cytometry analysis defined as CD34+KDR+ cells. Subsequently, EPCs pooled from 6 HD were co-cultured with caffeine at 0.5 mM and 1 mM with and without SLE sera. After 7 days, we evaluated the cells morphology and the ability to form colonies. Moreover, we analyzed for the percentage of annexin V-positive (AV) apoptotic cells by flow cytometry analysis and for levels of autophagy and apoptotic markers LC3-II, p62 and Bcl2 by western blot.

**Results:** We enrolled 31 SLE patients (F:M 30:1, median age 43 years, IQR 18; median disease duration 144 months, IQR 180). The median intake of caffeine was 166 mg/day (IQR 194). We found a EPCs median percentage of 0.03% (IQR 0.04) observing a positive correlation between caffeine intake and EPCs percentage (p=0.03, r=0.4). Moving on in vitro experiments, after 7 days of cell cultures, HD EPCs treated with SLE sera and caffeine showed an improvement in morphology and in number of EPCs-CFU in comparison with those incubated with SLE sera without caffeine (p=0.0003). Moreover, the colonies treated with SLE sera were poorly organized in comparison with HD; the addition of caffeine restored the colony structure. After treated HD-EPCs with SLE sera we observed an increase in AV positive cells and p62 and LC3-II values and a reduction of Bcl2 values; the addition of caffeine was able to significantly reduce AV positive cells and p62 and LC3-II values and to significantly increased Bcl2 values, without any significant differences between caffeine 0.5 mM and 1 mM treatment (Figure 1A-D).

**Conclusion:** Our data demonstrated the ability of caffeine in increasing the number of circulating EPCs in SLE patients. Moreover, in vitro experiments seem to suggest a protective role of caffeine on EPCs survival and vitality through the promotion of autophagy and the inhibition of apoptosis.

**REFERENCES:**


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**Disclosure of Interests:** None declared

**DOI:** 10.1136/annrheumdis-2022-eular.2695