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normal group (K-) and treated group (K+). Both groups were fed ad libitum for 4 weeks. K+ group was fed with a high cholesterol diet for the induction of hyperlipidemia using quail egg yolk with a dose of 5 ml/200 grams of body weight each day for 4 weeks. The cardiac organ were examined for malondialdehyde level. All data were statistically analyzed using statistic software.
Results: Mean of MDA levels (mg/dl) were 1.285±0.088 (K-) and 10.300±0.107 (K+). The Mann Whitney test revealed significant difference of malondialdehyde level in cardiac tissue between two groups with the p value=0.000 ($p<0.05$).
Conclusions: Quail egg yolk induction results increased levels of ROS and oxidative stress lead to increase malondialdehyde level in cardiac tissue.

P234 / #895, E-POSTERS TOPIC: *2. LIPIDS AND LIPOPROTEINS / 2.08 CELLULAR LIPID METABOLISM AND LIPID DROPLETS.*
CHOLESTEROL AND COVID-19: FINDINGS FROM LIPID PROFILE OF PATIENTS WITH SARS-COV-2 INFECTION.

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Background and Aims: At the moment COVID-19 is the most relevant global health problem. It seems that during SARS-CoV-2 infection total cholesterol (TC), LDL-C, and HDL-C values decrease. In our clinical practice we frequently observed alterations of the lipid profile in patients with COVID-19. This study aims to evaluate whether SARS-CoV-2 infection could be actually involved in the determining of lipid profile alterations, and to study the possible correlation of TC, LDL-C, HDL-C lowering and disease severity and/or clinical outcome.

Methods: We performed a retrospective analysis of the 118 patients who required hospitalization to Internal Medicine Unit of Chieti University Hospital (Italy) for COVID-19 between March and May 2020. We compared pre-infection lipid values collected from our laboratory exams software (53 of the 118 patients enrolled) to those measured on admission.
Results: Preliminary Median values showed on admission for COVID-19 were: TC 136.89±42.73 mg/dl, LDL-C 81.53±30.35 mg/dl , HDL-C 32,36±15.13 mg/dl and triglycerides 115±40.45 mg/dl ($p=0.001$, $p<0.001$ respectively). Median values of pre-infection total cholesterol and HDL-C were significantly higher than those measured on admission. C-reactive protein negatively correlated with LDL-C and HDL-C ($p=0.036$). No significant influence of lipid alterations on clinical outcome was highlighted.
Conclusions: Such results pointed out the impact of SARS-CoV-2 infection on TC and HDL-C, with the lowest values of LDL-C and HDL-C that are more likely to be detected at the highest inflammatory state in COVID-19 patients. It remains to better define a possible role for lipid metabolism and eventually for statins in the clinical and therapeutic approach to COVID-19.

P235 / #1083, E-POSTERS TOPIC: *2. LIPIDS AND LIPOPROTEINS / 2.08 CELLULAR LIPID METABOLISM AND LIPID DROPLETS.*
SPRING IS A NOVEL DETERMINANT IN SREBP SIGNALLING AND CHOLESTEROL METABOLISM

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Background and Aims: Disturbed lipid metabolism is a key contributor to development of cardiovascular diseases. We recently identified SPRING as a new determinant of sterol regulator element- binding protein (SREBP)-1 and -2 mediated transcriptional activation of cholesterol and fatty acid synthesis, and of low-density lipoprotein uptake. SPRING encodes a Golgi-resident, glycosylated membrane protein that is ubiquitously expressed. In this study we aim to further investigate and characterize this previously unknown regulator of lipid metabolism.
Methods: To elucidate the role of SPRING *in vivo* we are using transgenic mouse models to study the consequences of hepatic gain or loss of SPRING expression coupled with various cellular assays to increase our understanding of the mechanistic workings of SPRING *in vitro.*
Results: SPRING KO cells fail to robustly activate SREPB target genes and therefore cholesterol biosynthesis and lipoprotein uptake in response to sterol depletion. Whole body knockout of SPRING is embryonically lethal. However, adenoviral knockdown of SPRING in mouse livers showed impaired SREBP 1 and 2 signalling after fasting and subsequent refeeding. Mechanistically we showed that SPRING is likely influencing S1P mediated proteolysis of SREBP, as well as proper retrograde transport of SCAP.
Conclusions: Our data further establish the role of SPRING as a novel regulator of the SREBP pathway and thereby may help to develop mechanism-based strategies to treat dysregulated lipid metabolism.

P236 / #1160, E-POSTERS TOPIC: *2. LIPIDS AND LIPOPROTEINS / 2.08 CELLULAR LIPID METABOLISM AND LIPID DROPLETS.*
PACAP ANTAGONIZES OXLDL-INDUCED LIPID-/TRIGLYCERIDE ACCUMULATION AND VPAC1-EXPRESSION IN HUMAN M2-MΦ.

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Background and Aims: PACAP (pituitary adenylate cyclase-activating polypeptide) deficiency is proatherogenic in hypercholesterolemic mice and inhibits oxidized-low density lipoprotein (oxLDL)-induced TNF-α release, as well as foam cell formation in macrophages (MΦ). PACAP receptors, i.e. VIP receptor type 1 and 2 (VPAC1; VPAC2) and PACAP selective receptor PAC1 have been reported in MΦ of different tissues. The aim of this study is to examine the effects of PACAP38 on oxLDL-mediated lipid accumulation with respect to PACAP-receptor VPAC1 expression in human M1/M2-MΦ.

Methods: PMA-differentiated THP-1 MΦ were polarized into M1/M2-MΦ and treated with 50μg/ml oxLDL and/or 0.1nM PACAP38 for 16h. The phenotypes were proved by ELISA and qRT-PCR. The lipid- and triglyceride accumulation were determined by using OilRedO-staining and triglyceride-assay. Additionally, we determined the PACAP-receptor VPAC1 expression using western-blot
Results: The inflammatory M1-MΦ showed an enhanced TNF-α-release and CCR7 mRNA-expression, whereas immunosuppressive M2-MΦ revealed an increased IL-10-release and CCL17 mRNA-expression. By using OilRedO-staining and triglyceride-assay, we observed in M1/M2-MΦ an oxLDL-dependent increased intracellular lipid and triglyceride accumulation. In M2-MΦ, PACAP38 antagonized the oxLDL-dependent increase of the lipid accumulation and counteracts oxLDL-mediated increased triglyceride content. Thus, we determined the PACAP-receptor VPAC1 expression. OxLDL-treated M1-MΦ showed a decreased VPAC1-protein level, which can be antagonized by PACAP38. Additionally, treatment with PACAP38 without or with oxLDL increased the VPAC1-protein level in M2-MΦ.
Conclusions: Our present study demonstrates that PACAP38 counteracts the oxLDL-induced intracellular lipid accumulation and VPAC1-expression in human MΦ subtypes. Therefore, PACAP38 or PACAP agonists may be suggested as a novel class of atheroprotective therapeutics that have to be critically viewed.

P237 / #44, E-POSTERS TOPIC: *2. LIPIDS AND LIPOPROTEINS / 2.09 LIPID AND LIPOPROTEIN METABOLISM: MISCELLANEOUS.*
PREGNANCY IN HOMOZYGOUS FAMILIAL HYPERCHOLESTEROLEMIA – A CASE SERIES

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