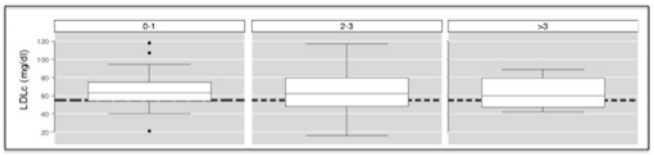
Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

(adjusted by age, sex and intensity of lipid treatment). -The relationship between lipid levels and the time elapsed since the infarction was assessed using Pearson's correlation coefficient.

Results: -The level of control was 31% considering 55 mg/dL and an additional 29% if 70 mg/dL was the desired LDLc threshold. Although the percentage of controlled patients increased according to the number of antihypertensive medications, these differences were not statistically significant.

-The same occurs for the LDLc levels, which were similar among groups: 0-1; 2-3 or more than 3 antihypertensive drugs (figure 1). The only significant differences were found in those having diuretics who have higher LDL levels in the unadjusted but not after adjustment and without any differences in the percentage of control.

-A positive correlation was found between the LDLc and the elapsed time since the AMI.



Conclusions: The percentage of lipid control after an AMI is low and it seems to get worse along time.

-We did not find an association between the complexity of antihypertensive treatment and the LDL levels.

P253 / #1066, E-POSTERS TOPIC: *2. LIPIDS AND LIPOPROTEINS / 2.09 LIPID AND LIPOPROTEIN METABOLISM: MISCELLANEOUS.*

CAN PLASMA LIPID LEVELS BE CORRELATED TO SEVERE COVID-19 INFECTION?

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Background and Aims: Alterations in plasma lipid levels are correlated with the severity of infections due to various pathogens such as bacteria, viruses, tuberculosis. While total cholesterol,LDL-C,and HDL-C levels tend to decrease, plasma triglyceride levels can vary(1).

Methods: We set up a COVID-19 follow-up outpatient clinic to address and manage the clinical needs of non-critical COVID-19 patients after discharge. Non-critical COVID-19 patients who were hospitalized in COVID-19 wards were evaluated after 14-30 days of discharge in terms of plasma lipid levels and these levels were compared to those at the time of admission to COVID-19 wards.Patients on statin treatment continued to take their drug while those who were not on statins were not initiated statin treatment during the study period.

Results: The admission and follow-up plasma lipid profiles were present for 95 patients. The mean(S.D) age was 48.49(16.4), and 49(51.6%) were male(Table 1). The mean(S.D) day between the admission and the first visit in the COVID-19 follow-up outpatient clinic was 27.8(12.8). Plasma total cholesterol(p=0.018), LDL-C(p=0.007), and HDL-C(p=0.003) levels were significantly lower in the severely ill group than the mild/moderate group at the admission. Plasma total cholesterol,LDL-C,HDL-C, and triglyceride levels on follow-up were significantly higher than those levels on the admission day(p<0.001). Delta(Follow-up - Admission) levels LDL, total cholesterol, and triglyceride levels were significantly high in patients who have received steroid therapy. Only delta LDL was significantly high in patients who require Intensive Care Unit(Table 2).

Table 1: Characteristic of patients

	Total n=95
Age, years, mean (SD)	48.5 (16.4)
Gender, n (%)	
Female	46 (48.4)
Male	49 (51.6)
Smoking, n (%)	
Never smokers	68 (71.6)
Active smoker	15 (15.8)
Ex-smoker	11 (11.6)
Alcohol consumption	8 (8.4)
Chronic diseases	
Type 2 diabetes mellitus, n (%)	29 (30.5)
Hypertension, n (%)	35 (36.8)
Coronary artery disease, n (%)	13 (13.7)
Chronic obstructive pulmonary disease, n (%)	8 (8.4)
Medications	
Angiotensin-converting enzyme (ACE) inhibitors / Angiotensin receptor blockers (ARBs) , n (%)	26 (27.4)
Metformin, n (%)	18(18.9)
Statin, n (%)	8 (8.4)
Acetylsalicylic acid, n (%)	13 (13.4)
Beta-blocker, n (%)	17 (17.9)
Calcium channel blockers, n (%)	6 (6.3)
Disease severity on admission, n (%)	
Mild	55 (57.9)
Moderate	26 (27.4)
Severe	10 (10.5)
Treatment, n (%)	
No treatment	5 (5.3)
Hydroxychloroquine	11 (11.6)
Hydroxychloroquine + Azithromycin	11 (11.6)
Hydroxychloroquine + Azithromycin + Favipiravir	10 (10.5)
Hydroxychloroquine + Favipiravir	4 (4.2)
Favipiravir	54 (56.8)
Corticosteroids add-on	8 (8.4)
Intensive Care Unit requirement, n (%)	10 (10.5)

Table 2: Plasma lipid levels of the COVID-19 patients at the admission and in the follow-up

	Admission level Median (IQR)	Follow-up level Median (IQR)	Delta Mean (SD)	P
LDL-C, mg/dl, n=95	106.8 (53)	125 (46)	16.36 (24.89)	<0.001*
LDL-C of patients who have received steroid therapy, n=8	86 (70.8)	134 (75.4)	42.4 (16.94)	0.004**
LDL-C of patients who required ICU admission, n=10	95.6 (71.4)	136.3 (48.6)	29.633 (26.11)	0.045**
HDL-C, mg/dl, n=95	38.1 (15.1)	45.5 (13.5)	5.9 (9.58)	<0.001*
HDL-C of patients who have received steroid therapy, n=8	28 (28)	36.8 (7.4)	6.24 (8.14)	0.912**
HDL-C of patients who required ICU admission, n=10	29.4 (13.4)	42.5 (11.2)	12.1 (14.27)	0.242**
Total cholesterol, mg/dl, n=95	168.2 (62.17)	207 (167.5)	35.18 (48.74)	<0.001*
Total cholesterol of patients who have received steroid therapy, n=8	127.4 (100.10)	222.9 (92)	83.32 (37.231)	0.004**
Total cholesterol of patients who required ICU admission, n=10	155.2 (89.2)	249.2 (65)	79.93 (78.991)	0.096**
Triglyceride, mg/dl, n=95	119.5 (87)	148 (153)	59.37 (68.25)	<0.001*
Triglyceride of patients who have received steroid therapy, n=8	83 (97)	292 (235)	173.428 (127)	0.042**
Triglyceride of patients who required ICU admission, n=10	126 (65)	249 (141)	191 (435.77)	0.341**

ICU: intensive care unit, *Wilcoxon **T-test

Conclusions: These findings support the evidence demonstrating that low LDL-C and/or HDL-C levels can increase the risk of developing severe infections(2).

P254 / #1097, E-POSTERS TOPIC: *2. LIPIDS AND LIPOPROTEINS / 2.09 LIPID AND LIPOPROTEIN METABOLISM: MISCELLANEOUS.*
AN EVALUATION OF DIFFERENT METHODS TO STUDY THE ASSOCIATION OF PROPROTEIN CONVERTASE SUBTILISIN-KEXIN TYPE 9 TO LIPOPROTEINS.

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Background and Aims: Proprotein convertase subtilisin-kexin type 9 (PCSK9) enhances the degradation of the hepatic low-density lipoprotein (LDL) receptor, increasing LDL-cholesterol levels in plasma. PCSK9 has been reported to associate with lipoproteins (LPs) in plasma, but data in literature are discordant. We compared different methods for LPs isolation, aiming at finding the best one for studying this association.

Methods: Fresh serum was collected from healthy volunteers. LPs were isolated with different methods, including precipitation with phosphotungstic acid, fast protein liquid chromatography (FPLC), ultracentrifugation using both KBr and iodixanol gradient (IGr). The PCSK9 content of the LP fractions obtained was quantified with ELISA. Cholesterol, APOB and APOA1 were measured using clinical-grade reactives.
Results: In the precipitation-mediated assay, more than 80% of PCSK9 was found in the APOB precipitate. Negligible amount of PCSK9 was detected in the LPs isolated with the KBr ultracentrifugation. PCSK9 was found in the LDL fraction obtained with both IGr ultracentrifugation and FPLC. The percentage of association showed inter and intra individual variability, ranging from 1% to 30% of total recovered PCSK9.
Conclusions: Based on our observations, the PCSK9-LDL association exists and is sensitive to high salt concentrations. IGr ultracentrifugation and FPLC appear to be both suitable for further studies.

P255 / #1103, E-POSTERS TOPIC: *2. LIPIDS AND LIPOPROTEINS / 2.09 LIPID AND LIPOPROTEIN METABOLISM: MISCELLANEOUS.*
PROPROTEIN CONVERTASE SUBTILISIN/KEXIN TYPE 9 (PCSK9) ASSOCIATION TO LDL IN PATIENTS TREATED WITH ANTI-PCSK9 MONOCLONAL ANTIBODIES.

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Background and Aims: Proprotein convertase subtilisin-kexin type 9 (PCSK9) increases low-density lipoprotein cholesterol (LDL-C) levels in plasma by enhancing the degradation of the hepatic LDL receptor (LDLR). Monoclonal antibodies (mAbs) targeting PCSK9 have shown efficacy in LDL-C reduction. PCSK9 associates to LDL in plasma. The aim of our study was to clarify whether mAbs administration could interfere with the PCSK9-LDL association.

Methods: Fresh plasma was collected from subjects enrolled for anti-PCSK9 mAbs therapy, before and after mAbs administration. Lipoproteins (LPs) were isolated with Iodixanol gradient (IGr) ultracentrifugation. The PCSK9 content of the LP fractions obtained was quantified with a commercially available ELISA kit; cholesterol was measured using a clinical-grade colorimetric assay.
Results: PCSK9 was detected in the LDL fraction both before and after mAbs administration, without significant intra-individual changes in terms of percentage of association. The PCSK9/cholesterol ratio in the LDL fraction seems to increase after mAbs administration.
Conclusions: According to our preliminary results, the PCSK9 - LDL association remains, even if the LDL level are dramatically reduced due to the mAbs therapy.

P256 / #1181, E-POSTERS TOPIC: *2. LIPIDS AND LIPOPROTEINS / 2.09 LIPID AND LIPOPROTEIN METABOLISM: MISCELLANEOUS.*
MAJORITY OF CLINICALLY DIAGNOSED FAMILIAL HYPERCHOLESTEROLAEMIA IN AN ASIAN COMMUNITY INHERIT VARIANTS OF FH CANDIDATE AND/OR ASSOCIATED GENES.

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Background and Aims: There were many reports on the identification of LDL receptor gene (*LDLR*) variants among Malaysian FH patients. However, the identification of multiple causative genes on FH individuals were yet to be reported. Therefore, we aimed to investigate the prevalence of monogenic and non-monogenic FH in Malaysia.

Methods: Sixteen Potential and 152 Possible FH patients by Dutch Lipid Clinic Network Criteria were clinically screened. DNA libraries were prepared using Illumina Ampliseq kit. Targeted next-generation sequencing were performed using Iseq sequencer. Basespace Variant Interpreter (Illumina) was used to annotate the variants and generated variants were filtered based on minor allele frequency of $\leq$0.05. The pathogenicity of the variants were checked using ClinVar while *in silico* prediction of pathogenicity were interpreted using PolyPhen and SIFT
Results: A total of 130/168 (77.4%) individuals inherited 36 major *(LDLR: 10, APOB: 18, PCSK9: 8)* and 18 minor FH-associated gene variants (*LDLRAP1:* 1, *APOE:* 2, *ABCG5*:7, *ABCG8*: 8). Of these, 39/130 (30%) of them were with compound or double heterozygous variants. Among the identified major gene variants, 10/36 (27.7%) of them were pathogenic (*LDLR*: 6, *APOB*: 2, *PCSK9*: 2), while among the identified minor gene variants, 8/18 (44.4%) of them were pathogenic (*LDLRAP1:* 1, *ABCG5*: 3, *ABCG8*: 4). Overall, 23/130 (17.7%) individuals carried pathogenic gene variants.
Conclusions: More than three quarters of the clinically diagnosed FH in the community are with FH-associated gene variants. A substantial number these variant-positive patients carry more than one variant, of whom <20% of them are with pathogenic variants.

P257 / #1218, E-POSTERS TOPIC: *2. LIPIDS AND LIPOPROTEINS / 2.09 LIPID AND LIPOPROTEIN METABOLISM: MISCELLANEOUS.*
CHOLESTEROL ESTER TRANSFER PROTEIN (CETP) EXPRESSION IN MICE PROMOTES CONTROL OF EXPERIMENTAL CUTANEOUS LEISHMANIASIS

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Background and Aims: Leishmaniases are diseases caused by protozoan of the genera *Leishmania* where lipoproteins may participate in the development of infection. The cholesteryl ester transfer protein (CETP) has an extensive impact on lipoprotein metabolism, and recent findings show that CETP may play a role in inflammation. Thus, we aimed to study the role of CETP in experimental cutaneous leishmaniasis.