

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.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active. **Conclusions:** EVs enriched in PCSK9 appear to favor a pro-atherogenic inflammatory phenotype.

EP141 / #1197, TOPIC: ASA01 - PATHOGENESIS OF ATHEROSCLEROSIS / ASA01-12 OTHER, POSTER VIEWING SESSION. COVID-19 AND CARDIOVASCULAR SYSTEM: NOT ONLY HEART BUT ALSO VASCULAR. THE EFFECTS OF THE INFECTION ON ARTERIAL STIFFNESS

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Background and Aims : SARS-CoV-2 determines a framework of multiorgan dysfunction that can involve the cardiovascular system creating damages of different nature. Among these, endothelial damage could play a key role in increasing arterial stiffness and thus the cardiovascular risk of infected patients. The aim of this study is to evaluate the Pulse Wave Velocity (PWV) of a population of patients after recovery from infection and to compare them with those of a group affected by arterial hypertension.

Methods: This prospective observational monocentric study involved 143 patients with previous diagnosis of Covid-19 who undergone PWV measurement during the follow-up at a median time of 3.8 months after the infection. These patients were compared to a population of 143 patients with hypertension matched by age, sex, Systolic Blood Pressure values and Body Mass Index.

Results: PWV values were higher in Covid-19 group comparing to hypertension group ($10.5 \pm 3.0 \text{ m/s}$ VS $8.9 \pm 2.5 \text{ m/s}$). Furthermore, there is a correlation between higher PWV values and lower values of SpO2% at time of admission at the Emergency Department. (R= -0.302; p<0.001).

Conclusions: SARS-CoV-2 infection seems related to increased PWV values. Moreover, higher arterial stiffness seems correlated to a worse oxygen saturation in Emergency Department. More studies with longer follow-up time are necessary to establish whether the vascular damage is reversible and whether it correlates with an increase of long-term cardiovascular risk.

EP142 / #1377, TOPIC: ASA01 - PATHOGENESIS OF ATHEROSCLEROSIS / ASA01-12 OTHER, POSTER VIEWING SESSION. FT-IR SPECTROSCOPIC STUDY OF ATHEROMATIC PLAQUE FORMATION

AND THE RISK OF COVID-19 MORTALITY OF PATIENTS WITH CARDIOVASCULAR DISEASES. A MATHEMATICAL SIMULATION APPROACH

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Background and Aims: COVID-19 various, a pandemic disease since 2019, attacks the patients with cardiovascular disease. However, the mechanism of this sensitivity has not yet been clear. The aim is focused to describe the relationship between the clinical history and atherosclerosis development with the survival.

Materials and method: To carry out we used the nationwide date for three categories of patients: diabetic with coronary events, diabetic and hypertensive. Fourier-Transform-Infrared spectra were used for patients of 54-74 years. The simulation was performed with ODE-(SIR) using the Λ fractional derivative. The arteries were received from patients who underwent to endarterectomy during Coronary Artery Bypass Grafting (CABG).

Results: FT-IR spectra showed that in diabetic cardiovascular patients the oxidative stress produces amyloid protein and AGEs (Advanced Glycation End products). The AGEs contain increasing number of -OH and -NH groups, which could bind to spike glycoptrotein of COVID-19 as donor-

acceptor by hydrogen bonds (Scheme). Moreover, from the patients' damaged ATP the PO_4^3 and bases are used from viral RNA for its replication.



Conclusions: Mathematical simulation of an association between the risk of COVID-19 infection in combination with FT-IR data showed amyloid proteins and AGEs of atheromatic plaques increased the risk of the influence in a higher order in diabetic patients with cardiovascular disease, diabetic and hypertensive. The onset time of the disease is significantly reduced from 8.5 days for hypertensive patients to 4.5 for diabetic and 2.5 for diabetic heart patients.

EP143 / #863, TOPIC: ASA01 - PATHOGENESIS OF ATHEROSCLEROSIS / ASA01-12 OTHER, POSTER VIEWING SESSION.

MIR-210-3P MEDIATES THE UP-REGULATION OF NADPH OXIDASE EXPRESSION IN THE ATHEROSCLEROTIC AORTA OF HYPERCHOLESTEROLEMIC APOLIPOPROTEIN E-DEFICIENT MICE; POTENTIAL IMPLICATIONS FOR HUMAN ATHEROSCLEROSIS

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Background and Aims : Reactive oxygen species overproduction driven by up-regulated NADPH oxidase (Nox) plays a major role in atherosclerosis. Hypoxia-induced miR-210-3p has been largely implicated in human malignancies as key regulator of oncogenic mechanisms. The role of miR-210-3p in atherogenesis is yet to be discovered. This study aimed at investigating the potential role of miR-210-3p in mediating Nox up-regulation in atherosclerosis.

Methods: Human non-/atherosclerotic arterial specimens, ApoE-/- mice, and human THP-1 monocytes (Mon) were studied. Male ApoE-/- mice fed a normal or atherogenic diet were randomized into four experimental groups to receive (i.p.) PBS or 10 mg/kg negative control/miR-210-3p LNA inhibitor, once per week, for 6 weeks. Resting macrophages (M0-Mac) were transfected with negative control/miR-210-3p LNA inhibitor and subjected to polarization into pro-inflammatory (M1) or anti-inflammatory (M2) phenotype. Real-time PCR, Western blot, and high-resolution fluorescence imaging were employed.

Results: Significant increases in miR-210-3p levels were detected in human carotid atherosclerotic lesions, mice atherosclerotic aorta and in M1-Mac. Bioinformatics analysis predicted that miR-210-3p targets negative regulators of NF- κ B signaling (TNIP1, SOCS1). Biodistribution studies confirmed the efficient uptake of FAM-labeled miRNA control inhibitor in the atherosclerotic aorta/liver of mice. Inhibition of miR-210-3p suppressed the up-regulation of Nox1-4 expression in atherosclerotic aorta of mice and Nox1-5 subtypes in cultured human M1-Mac.

Conclusions: In experimental atherosclerosis, miR-210-3p controls the up-regulation of Nox subtypes potentially by targeting negative regulators of NF-kB-related signaling pathways. These data point to miR-210-3p as