Results: The mean age of patients was 57.11 ± 7.51 years, and 57 patients (76%) were males. The correlation between MS components and SYNTAX score using Spearman correlation showed a strong correlation with correlation coefficient r = 0.837, p-value<0.001*.

Table 2
Distribution of MS components according to SYNTAX score

SYNTAX score	MS components		
	2 (n= 68)	3 (n=54)	4 (n=28)
Low (1-22)	54	14	8
Intermediate (23-32)	8	23	14
High (≥33)	6	17	6

Conclusions: In non-diabetic patients with MS, components of MS had a strong positive correlation with CAD severity assessed by SYNTAX score.

EP662 / #129, TOPIC: *ASA04 - CLINICAL VASCULAR DISEASE / ASA04-12 PREVENTION AND TREATMENT OF CARDIOVASCULARDISEASE; MISCELLANEOUS*, POSTER VIEWING SESSION.
CORONAVIRUS AT THE HEART CENTER OF PUERTO RICO INCIDENCE-DEATH: THE ROLE OF GENETICS

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Background and Aims : Coronavirus disease mostly affects the respiratory system. Since it is a novel disease, little is known about the connection between heart involvement and COVID-19.

Methods: In total, 50 patient records with positive PCR for SARS-CoV-2 were analyzed from the Heart Center Hospital. Within the medication section, our prime focus was to find whether these P. were currently taking or took Losartan in the past, because this drug reduces the penetration intracellularly of the virus.
Results: All of the 50 P. were from Puerto Rico (P.R.), a Hispanic population. None of the P. was taking Losartan. According to the records 96% had severe health problems previously to being contaminated by the virus. Ten percent of these P. died; cause of death was not a result of a clear correlation between COVID-19 and other comorbidities. These P. were chronically ill. The CFR was .005, while the total CFR of the Puerto Rican population with the virus was .1. Probably, this increase is due an aged population (age >65 years) and comorbidities.
Conclusions: In P.R., and possibly other Hispanic countries, there are genes which we call "protective genes" (P.G.) (*CYP₂C9, VXORC1 and VKORC1-1639>A allele in sector 1*) that control the incidence and degree of heart disease. In addition, since none of the 50 P. took Losartan, we think this is a factor which will increase the incidence of the disease.

EP663 / #130, TOPIC: *ASA04 - CLINICAL VASCULAR DISEASE / ASA04-12 PREVENTION AND TREATMENT OF CARDIOVASCULARDISEASE; MISCELLANEOUS*, POSTER VIEWING SESSION.
PROTECTIVE GENES AND EVOLUTION REDUCES THE ATHEROSCLEROTIC PROCESS IN HISPANICS (PUERTO RICO) WHEN COMPARED WITH THE U.S.A. MAINLAND

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Background and Aims : Atherosclerosis (A.) is a complicated process produced by many factors, including genetic factors, diabetes mellitus, hypertension and others. In Puerto Rico (P.R.) and possibly other Hispanic countries, especially genes, which we call "protective genes" (P.G.) controls the degree of this A. process. It is the purpose to describe these mechanisms.

Methods: These genes, whose origins are Europeans, African and Amerindians, described by Duconge and colleagues at the University of Puerto Rico. The most important genes reducing this inflammatory process are an admixture of: *CYP₂C9, VXORC1 and VKORC1-1639>A allele in sector 1. These genes are homogeneous in the full Puerto Rican culture. Another factor is a reduction of monocytes transformation to macrophage producing sub endothelial accumulation in the union of the A pub by blocking Angiotensin II and cytokines-mechanisms, especially by Losartan. The most frequent antihypertensive drug used in P.R. This will reduce the endothelial damage which will produce plaques, foam cells and organ ischemia.*
Results: *We think the P.G. are crucial in these mechanisms reducing the origin and progression of the atherosclerotic process, more in P.R. (30%), U.S.A. Island, than in the U.S.A., because they don't have these anti-inflammatory P.G.*
Conclusions: *The reduction of damage to the endothelial lining, reducing plaque formation and foam cells, the prelude of severe damage to the endothelium and myocardial damage. Probably the main factor is gene induced by the P.G. Evolution has a big role.*

EP664 / #333, TOPIC: *ASA04 - CLINICAL VASCULAR DISEASE / ASA04-12 PREVENTION AND TREATMENT OF CARDIOVASCULARDISEASE; MISCELLANEOUS*, POSTER VIEWING SESSION.
EARLY CARDIORENAL RISK IS MOLECULARLY EVIDENCED IN HYPERTENSIVE SUBJECTS WITHIN THE NORMOALBUMINURIA CONDITION. NOVEL GLYCOTARGETS FOR CARDIOVASCULAR RISK STRATIFICATION

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Background and Aims : Clinical evidences show early cardiorenal risk in normoalbuminuric subjects within the high-normal range (ACR=10-30). But anticipating who will progress is not possible and subjacent mechanisms are unknown. Thus, normoalbuminuric subjects are out of therapeutic management. We aimed to identify molecular targets of early cardiorenal risk which may aid in individual risk stratification

Methods: Urine samples were collected from hypertensives subjects under RAS suppression classified in control if ACR<10mg/g and high-normal (HN) if ACR=10-30mg/g. The urinary glycoproteome was analyzed by omics (isobaric labeling) (n=16), systems biology analysis was performed and proteins showing differential abundance in HN (Mann-Whitney, p-value <0.05) were confirmed by target-MS (n=37). Pathogenicity score was calculated on glycopeptides and immunohistochemistry was performed on human kidney and aortic tissue to evaluate the renal and vascular components of the observed changes.
Results: 482 N-glycoproteins were identified; 29 show the most significant alteration in HN, mainly A1AT, HPTR, CERU, ATL2, DBF4A and TOM6. Main altered pathways are complement/coagulation cascades (p-value =0.0002), ferroptosis (p-value =0.0015) and platelet degranulation (p-value =0.0009). Increased levels of urinary A1AT reflect significant diminishment in microalbuminuric kidney and in atherosclerotic aorta. A panel of 23 N-glycopeptides reveal the biological significance of N-glycosylation in CVD, showing pathogenicity score ≥0.8 and significant abundance variation in HN even though they protein of origin do not vary.
Conclusions: Urinary protein glycosylation reveal molecular targets for early cardiorenal risk stratification in normoalbuminuric subjects. Those may aid in revising current therapeutic strategies for prevention in hypertensives which may be extended to general population.