mutations in the SHR that do not impact on the molecular weight of the native protein, but may affect its post-translational modification (PTM). This notion is supported by additional data, which suggest an alteration in *O*-linked -N-acetylglucosamination.

Conclusion: Our results emphasize the role of the SHR Cd36 gene defect as the factor underlying its cardiac restricted LCFA utilization. The functional impact of this gene defect includes alterations in CD36 protein level and susceptibility to PTM.

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In vivo detection of non-occlusive thrombi in drug-eluting stents by scintigraphy and radio-labelled annexin V in a rabbit model

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Introduction: Thrombi in contact with non re-endothelialized stent struts associated with drug-eluting stent (DES) thrombosis. Hence, detection of thrombi in DES could help to evaluate the risk of DES thrombosis. Annexin V radio-labelled with 99mTechnetium (99mTc) is a radio-tracer with a high affinity for activated platelets.

Objectives: Our objectives were: 1) to develop an animal model of non-occlusive thrombosis of stents, 2) to evaluate the ability of annexin V 99mTc for the detection of in-stent thrombi using scintigraphy.

Methods: Right carotid arteries of NZW rabbits (n = 14) fed a high cholesterol diet were implanted with overlapping DES (n = 7) or bare-metal stents (BMS; n = 7). Four weeks after stent implantation, rabbits underwent a first scintigraphy 3 hours after injection of 200 MBq of radio-labelled annexin V 99mTc. At the end of the first scintigraphy, a suture was placed surgically proximal to the stented carotid arteries in order to induce a thrombus-prone flow limiting stenosis. Four days later, a second scintigraphy was performed. After the second scintigraphy, stents were excised, imaged ex vivo and then fixed for histological examination and scanning electron microscopy (SEM).

Results: Activities measured in vivo in the stented carotid arteries after injection of annexin V 99mTc were higher on the second scintigraphy after creation of a surgical stenosis as compared to the first scintigraphy (0.24 vs. 0.15 counts/pixel/ MBq, respectively; $p<0.05$). On the second scintigraphy, activities were higher in DES vs. BMS (0.26 vs. 0.19 counts/pixel/ MBq, respectively; $p < 0.005$). High activities measured in stents in vivo were associated with the detection of thrombi on corresponding histological sections and SEM.

Conclusions: In this work, we developed a rabbit model of non-occlusive thrombosis of stents in carotid arteries. In this model, in-stent thrombi could be detected using annexin V 99mTc scintigraphy.

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The metabolic effects of glutamine in the heart beyond anaplerosis : role of the hexosamine biosynthetic pathway

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Aim: Glutamine is the most abundant amino acid in the plasma, and has been shown to exert cardioprotective effects. However, the underlying mechanisms remained unclear, but may include an anaplerotic effect via its metabolic conversion to citric acid cycle (CAC) intermediates or activation of the hexosamine biosynthetic pathway (HBP).

Methods: To assess the potential roles of these mechanisms, we evaluated the metabolic effects of a physiologically relevant concentration of glutamine (0.5mM) in isolated working rat hearts perfused with ^{13}C-labeled substrates with or without 20 M azaserine (an HBP inhibitor) and under restricted supply of carbohydrates (CHO, i.e. without pyruvate/insulin).

Results: When perfused with a mixture of CHOs, a fatty acid oleate and insulin (controls), the addition of glutamine had no effect on functional parameters except for a 17% ($p<0.05$) decrease in relaxation. However it resulted in an increase in the percent contribution of ^{13}C-oleate to acetyl-CoA production (51%) and triglyceride formation (2.8 folds). This was accompanied by a significant reduction ($p>0.05$) tissue levels of the CAC intermediates (in nmol/gww): citrate: 260±10 *vs*. 222±6 and malate 128±5 *vs*. 103±3 . Inhibition of HBP with azaserine restored oleate oxidation and tissue CAC levels, but not triglyceride formation. When perfused under restricted supply of CHOs, hearts displayed significantly decreased cardiac output (65%), a greater percent contribution of glucose to pyruvate formation (60%), and lower tissue citrate and malate levels (45%). Addition of glutamine restored cardiac output and glucose contribution to pyruvate formation but not tissue CAC levels.

Conclusion: Collectively, these results demonstrate the capacity of glutamine to modulate energy substrate selection and function in the perfused heart. Furthermore, the potential mechanisms underlying these effects appear to be mediated via the HBP rather than anaplerosis.

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Prevalence of aspirin resistance in stable coronary heart diseased patients and correlation with platelet turn-over.

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Background: Aspirin resistance has been widely reported but the underlying mechanisms remain unclear. Previous studies have suggested a relationship between accelerated platelet turn-over and aspirin resistance in patients with coronary artery disease. The purpose of this study was to determine whether aspirin resistance could be linked to accelerated platetet turn-over.

Methods: We performed a prospective monocentric study including 50 consecutive patients with stable coronary artery disease treated by aspirin (75 to 250 mg/day) without any other antiaggregant treatment. Aspirin resistance was characterized 24 hours after aspirin intake by light transmission aggregometry using 0.5 mg/mL arachidonic acid. Aspirin resistance was defined as >20% residual agregation. Platelet turn-over was estimated at the same time by measurement of mean platelet volume, % of reticulated platelets, serum P-selectin, platelet P-selectin and serum thrombopoietin.

Results: Among 50 patients (70±11 y.o. mean±1,5, 76% male, 52% type 2 diabetes mellitus, 16% active smokers), 18 (36%) were identified as aspirin resistants. Table 1 shows the mean value of markers currently linked to platelet turn-over depending on the presence of aspirin resistance. Serum thrombopoietin was significantly increased in patients with aspirin resistance compared to patients with no aspirin resistance. No statistical difference was demonstrated for mean platelet volume, reticulated platelets, platelet P-selectin and serum P-selectin. Serum thrombopoietin values were not correlated with other platelet turn-over parameters. There was no significant correlation between serum thrombopoietin and inflammatory markers.

Conclusion: Serum thrombopoietin is associated with aspirin resistance, but no other parameters currently linked to platelet turn-over. Further studies are needed to determine whether serum thrombopoietin can predict aspirin resistance in a larger cohort.

	Aspirin sensitive	Aspirin resistant	p
Platelet volume (fl)	8.78±0.26	8.82±0.30	0.92
Reticulated platelet (%)	8.4±0.52	8.6±0.76	0.82
Serup P selectin (ng/ml)	42.6±4.29	42.9±4.75	0.97
Platelet P selectin (%)	11.1±1.0	9.5±1.5	0.35
Serum thrombopoietin (pg/ml)	130.6±11.3	319.9±97.8	0.01

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C(-260)T polymorphism in the promoter of CD 14 gene is not associated with myocardial infarction in the tunisian population

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Introduction: Recent finding suggest that inflammation plays a role in atherosclerosis and its acute complications. Several known mechanisms may play at least a partial role in this process. One of the most likely mechanisms involves lipopolysaccharide (LPS) and its receptor, CD14. The C(-260)T single nucleotide polymorphism in the promoter region of the CD14 receptor gene has been reported to be associated with a higher risk of MI. Others studies, however, have not corroborated these findings. Considering the contradictory results, the aim of the present study was to investigate the possible association between the CD14 C(-260)T polymorphism and the risk of MI in the Tunisian population.
Material and Methods: A total of 333 Tunisian patients with MI and 345 healthy controls were included in the study. Genotyping was performed by polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) analysis.

Results: The frequency of TT homozygous genotype for the CD14 C(-260)T polymorphism was 24.9% in MI patients and 24.0% in the control group. However, the genotype distribution and allele frequencies were not significantly different between MI and controls subjects. Moreover, the odds ratio for MI associated with the T allele failed to reach statistical significance (OR=1.08; 95% CI: 0.86 - 1.34; P=NS).
Conclusion: These results do not support the hypothesis that the C-260T polymorphism of CD14 gene contributes to the genetic susceptibility to MI in the Tunisian population studied.

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Association of rs 2781666 G/T polymorphism of arginase 1 gene with myocardial infarction in the Tunisian population

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Introduction: Arginase I (ARG I) is the final enzyme of the urea cycle that converts L-arginine to urea and ornithine. Emerging evidence have identified ARG I as a critical regulator for nitric oxide (NO) production via nitric oxide synthase (NOS).
Therefore upregulation of ARG I inhibit NOS-mediated NO and may contribute to endothelial dysfunction. In addition, pathophysiological role of ARG I on vascular disease have been extensively documented, and some recent studies support a role for ARG I in the development and complications of coronary artery disease (CAD). The aim of the present study is to investigate the possible association between rs 2781666 G/T polymorphism of ARG I gene and myocardial infarction (MI) in the Tunisian population.

Methods: In a case-control study, a total of 321 patients with MI and 436 controls were included. The rs 2781666 G/T polymorphism of ARG I was determined by PCR-RFLP analysis.
Results: Patients with MI had significantly higher frequency of TT genotype compared to controls (10.3 % vs 6.7 %; OR (95 % CI), 2.05 (1.19 - 3.52), p=0.009). The MI patients showed higher frequency of T allele compared to the controls (0.32 vs 0.23, OR (95 % CI), 1.58(1.25 - 2.00), p<0.001). The association between rs 2781666 G/T polymorphism of ARG I gene and MI was no longer significant after adjustment for other well established risk factors.

Conclusion: Our results revealed a significant but not independent association between rs2781666 G/T polymorphism of ARG I gene and (MI) in the Tunisian population.

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Lack of association between the -420C>G genetic variant in the resistin locus and myocardial infarction in the Tunisian population

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Objective: Resistin is a polypeptide (hormone) that is specifically secreted from adipocytes. It plays an important role in communication between adiposity and insulin resistance and it has been linked to the pathogenesis of atherosclerosis. Recently, -420C>G, a variant located in the promoter region of the resistin gene (RETN) (rs1862513) was identified. The aim of this study was to investigate the association between this polymorphism and the risk of myocardial infarction (MI) in the Tunisian population.

Design and methods: A total of 787 unrelated male Tunisian subjects including 455 healthy controls and 332 patients who had survived a first MI were prospectively recruited. Standard definitions and criteria for MI diagnosis were employed. Blood samples were obtained after an overnight fast. Serum glucose, TC and TG were measured by standardized enzymatic methods using commercial kits (Roche Diagnostics, Mannheim, Germany) on a Hitachi 912 analyzer. Genomic DNA was extracted from peripheral blood leukocytes according to standard methods. -420C>G polymorphism genotypes were determined by polymerase chain reaction followed by restriction analysis with 5 units of BbsI. Digested products were separated on 2.5% agarose gel with ethidium bromide staining.

Results: The frequencies of RETN -420G allele in MI group, and control group were 0.45 and 0.41, which are met with the Hardy-Weinberg equilibrium. Compared with controls, there was no significant differences in distribution of genotypes and allele frequencies of -420 C>G polymorphic site in MI patients and controls. In addition, there was no significant difference between the genotypes with lipid profiles.

Conclusion: Our data suggest that the RETN -420C>G polymorphism is not associated with an increased risk of MI in a Tunisian population.

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Study of arterial rigidity QAS
Evaluation by radio frequency of the quality of carotide rigidity on level in real-time, in patients with cardiovascular risk factors

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