Table – Prevalence of dyslipidemia depends on guidelines

	Baseline N=186 (%)	Year 1 N=153 (%)	Year 2 N=141 (%)	Year 3 N=137 (%)	Year 4 N=128 (%)
SCORE	25 (13.4)	20 (13.1)	23 (16.3)	25 (18.2)	28 (21.9)
SCORE × 1,5 (2 conditions)	26 (14)	20 (13.1)	23 (16.3)	25 (18.2)	28 (21.9)
SCORE × 1,5 (1 condition)	49 (26.3)	54 (35.3)	49 (34.7)	60 (43.8)	59 (46.1)
NCEP (FRS)	51 (27.4)	41 (26.8)	30 (21.3)	32 (23.4)	44 (34.4)
NCEP × 1,5 (2 conditions)	51 (27.4)	42 (27.4)	30 (21.3)	32 (23.4)	44 (34.4)
NCEP × 1,5 (1 condition)	54 (29)	44 (28.7)	31 (22)	32 (23.4)	45 (35.2)
AFSSAPS (FRS global)	21 (11.3)	14 (9.2)	11 (7.8)	14 (10.2)	22 (17.2)
AFSSAPS (FRS global) × 1,5 (2 conditions)	22 (11.8)	15 (9.8)	11 (7.8)	14 (10.2)	22 (17.2)
AFSSAPS (FRS global) × 1,5 (1 condition)	33 (17.7)	27 (17.6)	22 (15.6)	23 (16.8)	30 (23.4)

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Heightened risk of coronary atheroma conferred by a decrease in the plasma concentrations of lithocholic acid

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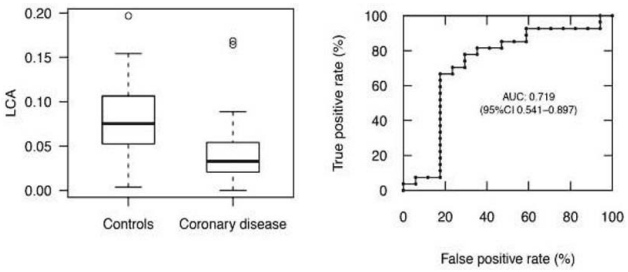
Context: The bile acids receptors Farsenoid X and TGR5 protect against the formation of atheroma in mice, though no evidence have linked coronary atheroma and bile acid in human. Bile acids links these receptors with more or less efficient activation, depending on the species.

Objective: To test the hypothesis that changes in concentrations of circulating bile acid species influence the risk of developing coronary atheromas in humans.

Methods: Pilot, prospective, observational study conducted between June and September 2010. The serum concentrations of cholic, chenodeoxycholic, deoxycholic, and lithocholic acids were measured in a fasting blood sample. Consecutive hospitalized or ambulatory patients undergoing emergency or elective coronary angiograms were eligible for inclusion. Post-cardiac arrest and non-fasting states, hepatic disease, and treatment with antimicrobials, corticosteroids, statins or fibrates were exclusion criteria. Of 393 screened patients, 44 met the study entry criteria, and were divided between 27 patients with (Group A) and 17 without (Group B) angiographically visible coronary atheromas. The pool of circulating bile acids was analyzed to measure the plasmatic concentrations of 28 different bile acid species. The variables associated with the presence of angiographically visible coronary atheromas were examined by single and multiple variable logistic regression analysis.

Results: The serum lithocholic acid concentration was significantly lower in group A than in group B. By multiple variable analysis, lithocholic acid was the only predictor of coronary atheroma independently of patient gender (odds ratio 2.41 per 0.05 decrease; 95% confidence interval 1.11 to 5.25, *P*=0.027

Conclusion: A low serum concentration of lithocholic acid was an independent predictor of coronary atheroma in human.



Figures

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At early phase of endotoxemic shock the increased β-adrenergic contractility is dependent of the endothelial β1-adrenoceptor

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Cardiovascular alterations in the septic shock include an hypotension associated with a cardiomyopathy. The sympathetic regulation of the cardiovascular system is impaired during the shock and associated with an altered endothelial function. However, involved cellular mechanisms are not clear. The aims of this project were to determine the role of the three β-adrenoceptor subtypes, β_1, β_2 and β_3-AR in the cardiac dysfunction in endotoxemic rats.

Methods: Rats (12w) received either endotoxin (LPS, 5mg. kg^{-1}) or saline *i.v.* (C). 3h later, cardiac parameters were studied *in vivo* by echocardiography. Selective β-AR responses were studied on papillary muscle contractility with or without a functional endothelium. Endothelium damage was realized with 3s Triton X-100 at 0.5%.

Results: *In vivo*, LPS rats presented altered systolic (shortening fraction –21±4% vs C p<0.05) and diastolic (E wave –47±4% vs C p<0.05) functions. In papillary muscle, isoproterenol (non selectif βAR agonist) induced contractility was increased in LPS (+105±21% vs C; p<0.05). This increase did not result from β_3-AR and β_2-AR because there expressions were respectively decreased by 20±4%; (p<0.05 vs C) and 47±7% (p<0.05 vs C) in LPS and correlated to a maintained β_3-AR-induced contractility and a decreased β_2-AR (–38±8% vs C; p<0.05). The β_2-AR-induced contractility was not modified in LPS muscle without endothelium whereas it was reduced in C muscle without endothelium (79±6% vs C; p<0.05). Conversely, albeit β_1-AR expression was decreased (–66±5% vs C; p<0.05), β_1-AR response was increased in papillary muscles (+94±16% vs C; p<0.05) from LPS rats. Surprisingly, the disrupted endothelium abolished this increase.

Conclusion: Our results demonstrate, for the first time, an increased β_1-AR contractility, on papillary muscle form LPS rats, dependent of the functional endothelium. This suggests that β_1-AR could be involved in the persistent tachycardia observed in the shock leading to propose β_1-AR blockers in this disease.

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Mobilization of CD34+KDR+ cells among circulating progenitors predicts target lesion revascularization

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