

编号: YY001-20190923001

**标题: Maternal obesity and gestational diabetes: Impact on arterial wall layer thickness and stiffness in early childhood - RADIEL study six-year follow-up**

简介: Background and aims

Gestational diabetes (GDM) and maternal obesity are linked to weight gain in childhood and an increased risk of cardiovascular disease later in life. We assessed the effects of GDM and maternal obesity on arterial function and morphology in relation to body anthropometrics and composition in early childhood.

Methods

We assessed body size and composition, blood pressure (BP), arterial morphology and stiffness in 201 pairs of obese mothers (pre-pregnancy BMI  $30.7 \pm 5.6$  kg/m<sup>2</sup>, 96 with GDM) and their children at 6.1 years (SD 0.5).

Results

Child BMI (z-score  $0.45 \pm 0.92$ ;  $p < 0.001$ ) and common carotid intima-media thickness (IMT, z-score  $0.15 \pm 0.75$ ,  $p = 0.003$ ) were increased compared with a healthy Finnish reference population. No associations with maternal GDM was found. Carotid IMT and pulse wave velocity were unrelated to child sex, anthropometrics, body composition, BP, as well as maternal anthropometrics and body composition. Carotid stiffness was independently predicted by second trimester fasting glucose. Child lean body mass was the strongest independent predictor for radial (RA), and brachial artery (BA) lumen diameter (LD) and BA IMT (LD: RA:  $r^2 = 0.068$ ,  $p < 0.001$ ; BA:  $r^2 = 0.108$ ,  $p < 0.001$ ; IMT: BA:  $r^2 = 0.161$ ,  $p < 0.001$ ) and carotid LD ( $r^2 = 0.066$ ,  $p < 0.001$ ).

Conclusions

Children of obese mothers have increased BMI, blood pressure and carotid IMT suggesting a transgenerational effect of maternal obesity and clustering of cardiovascular risk factors in the population. Arterial dimensions were mainly predicted by child LBM, and not associated with maternal or child adiposity, or GDM. There was a weak association with maternal gestational fasting glucose and increased carotid artery stiffness.

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编号: YY001-20190923002

**标题: Effect of lipid-lowering treatment in cardiovascular disease prevalence in familial hypercholesterolemia**

简介: Background and aims

The impact on heterozygous familial hypercholesterolemia (HeFH) health led by high-intensity lipid-lowering therapy (HILLT) is unknown, and the question remains if there is still an unacceptably high residual risk to justify treatment with new lipid-lowering drugs.

Methods

This observational, retrospective, multicenter, national study in Spain, whose information was obtained from a national dyslipidemia registry, was designed to establish the current prevalence of cardiovascular disease (CVD) in HeFH and to define the impact of HILLT

on CVD in this population. Odds were estimated using several logistic regression models with progressive adjustment.

### Results

1958 HeFH, mean age  $49.3 \pm 14.3$  years, were included in the analysis. At inclusion in the registry, 295 patients (15.1%) had suffered CVD and 164 (55.6%) had suffered the first event before the onset lipid-lowering treatment. Exposition to treatment associated more than ten times lower odds for CVD than in subjects naïve to treatment (OR 0.085, 95% CI 0.063–0.114,  $p < 0.001$ ). A first CVD event after a mean treatment period of  $9.1 \pm 7.2$  years occurred in 131 out of 1615 (8.1%) HeFH subjects, and 115 (87.8%) of them were on HILLT.

### Conclusions

Current prevalence of CVD among HeFH is one third of that reported before the statins era. Early initiation and prolonged lipid-lowering treatment was associated with a reduction in CVD. New cases of CVD, in spite of HILLT, appeared mostly among patients accumulating risk factors and probably they may be considered for further lipid-lowering drugs.

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编号: **YY001-20190923003**

**标题: Clinical, behavioral and biomarker predictors of PCSK9 levels in HIV-infected patients naïve of statin therapy: A cross-sectional analysis from the Swiss HIV cohort**

简介: Background and aims

Better characterization of Proprotein Convertase Subtilisin/Kexin 9 (PCSK9) profile is currently needed to tailor appropriate lipid-lowering strategies in HIV patients.

### Methods

HIV-infected individuals aged  $\geq 40$  years and naïve of statin therapy included in the Swiss HIV cohort study were screened for PCSK9 levels with a routine blood sample collection in 2014 at the Geneva University Hospitals. An exploratory linear regression model was built including clinical (age, sex, ethnicity, cardiovascular risk factors, body mass index, low CD4 defined as  $\leq 200$  cells/ $\mu\text{l}$ , leucocytes, lymphocytes, platelet, antiretroviral therapy), behavioral (tobacco and marijuana smoking, alcohol use and physical activity) and biomarker (CRP, TNF- $\alpha$ , IL-8, IL-10 and MCP-1) to investigate association with continuous PCSK9 levels.

### Results

We studied 239 HIV-infected individuals who met inclusion criteria and available PCSK9 levels with a mean age of 49 years. 35 subjects (14.6%) reported marijuana consumption, of whom 20 (57.1%) reported daily consumption and 15 (6.3%) occasional use. PCSK9 levels were correlated with low-density lipoprotein-cholesterol (LDL-C). Our exploratory model identified marijuana consumption ( $p=0.023$ ) and low CD4 values ( $p=0.020$ ) as significantly associated factors with higher PCSK9 levels. No association was found with Framingham risk score. Patients with marijuana consumption had

significantly higher levels of PCSK9 with a dose-response effect ( $p < 0.001$ ); the association persisted after adjustment for the calculated Framingham risk score ( $p=0.003$ ) and additional adjustment for clinical variables ( $p=0.027$ ).

#### Conclusions

In HIV-infected individuals naïve of statin treatment, marijuana consumption and low CD4 values are associated with higher PCSK9 levels independently of clinically relevant confounding factors.

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编号: **YY001-20190923004**

**标题: Iron and Atherosclerosis: The Link Revisited**

简介: Iron represents the second most abundant metal of the earth's crust [1]. Iron has a Janus-faced role (essential versus toxic) in health and disease. It is essential as an important micronutrient, implicated in the development of normal red blood cells by being part of hemoglobin. It can also be toxic if present in excess amount (iron overload), due to free radical generation that leads to deleterious effects on various tissues and organs [1]. In the circulation, transferrin (Tf)-bound iron represents the major form of iron [2]. Tf transports and delivers iron to the cells via Tf receptor 1-mediated endocytosis and to bone marrow, where iron facilitates the production of new erythrocytes [1]. Non-transferrin bound iron (NTBI) represents the form of iron that is not bound to Tf and other iron-binding proteins [2]. NTBI is a heterogeneous form of iron that can be detected in the circulation of patients with iron overload [such as hereditary haemochromatosis (HH), a common autosomal recessive disease] [3]. Increased free iron augments the production of reactive oxygen species (ROS) via the Haber-Weiss reaction, thereby increasing lipid peroxidation [1]. See a recent review on source, fate, and cellular interactions of Tf-bound iron and NTBI

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编号: **YY001-20190923005**

**标题: Extracellular vesicles in atherosclerosis**

简介: Extracellular vesicles (EVs), which exist in human blood, are increased in some inflammation-related cardiovascular diseases. EVs are involved in inflammation, immunity, signal transduction, cell survival and apoptosis, angiogenesis, thrombosis, and autophagy, all of which are highly significant for maintaining homeostasis and disease progression. Therefore, EVs are also associated with key steps in atherosclerosis, including cellular lipid metabolism, endothelial dysfunction and vascular wall inflammation, ultimately resulting in vascular remodelling. In this review, we summarize recent studies on EV contents and biological function, focusing on their potential effect in atherosclerosis, including cholesterol metabolism, vascular inflammation, angiogenesis, coagulation and the development of atherosclerotic lesions. EVs may represent potential biomarkers and pharmacological targets for atherosclerotic diseases.

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