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Full Length Research Paper

Unfavorable apoAI-containing lipoproteins profile in Tunisian obese women group

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ApoAI-containing lipoproteins, which have been reported in relation with the development of ischemic diseases, have never been studied in obese subjects that have any other factor affecting the lipoproteins metabolism. Control and obese women groups were constituted on the basis of the body weight: less than 110% and more than 125% of the ideal body weight, respectively. Different lipid and lipoprotein parameters, including the two apoAI-containing lipoproteins species, were quantified. Compared to control group, obese group exhibited a higher levels of plasma triglycerides, high density lipoprotein-triglycerides (HDL-TG) and lipoproteins with both apo AI and apo AII (LpAI:AII) but a lower values of plasma-apoAI percentage associated with LpAI particles (P < 0.05). The other studied parameters, including the distribution of apoAI and LpAI between HDL and non-HDL fractions, were similar in the two subject groups. In our obese subjects, plasma TG appear to be the more determinant for the atherosclerosis risk as suggested by their strong positive and negative correlation with LpAI:AII (P < 0.001) and with LpAI/LpAI:AII, respectively. The profile of apoAI-containing lipoproteins appears to be more sensitive to obesity effect than traditional lipid and lipoprotein parameters. The nature of its alteration could explain, at least in part, the association of obesity with high atherosclerosis risk.

Key words: Obesity, atherosclerosis, apoAI-containing lipoproteins.

INTRODUCTION

The role of lipids in the pathogenesis of heart disease has now been firmly established. In particular, the positive relation of plasma cholesterol (Chol), triglycerides (TG), apo B, low density lipoprotein (LDL)-Chol and the negative relation of high density lipoprotein (HDL)-Chol and apoAI with the development of this disease have been known for a long time (Barter and Rye, 1996; Brewer et al., 2004; Vinik, 2005; Paul and Baudin, 2009). More recently, other lipoprotein parameters have been proposed as atherosclerosis markers. Among these parameters, apoAI-containing lipoproteins were particularly studied. These lipoproteins are often distinguished in two particle populations: particles with apo AI and apoAII (LpAI:AII) and particles with apoAI but without apo AII (LpAI) (Cheung et al., 1986; Koren et al., 1987; Cheung and Wolf, 1989; Barkia, 1990; Barkia et al., 1991). These lipoprotein populations were shown to be metabolically and functionally different (Fielding and Fielding, 1981; Cheung et al., 1986; Barkia et al., 1991). While the rise of atherosclerosis has being observed to be positively associated with the LpAI:AII concentration, it was negatively linked to the LpAI level (Puchois et al., 1987; Asztalos et al., 2000; Nowicka and Jarosz, 2001). Moreover, experimental studies reported that, among the two apoAI-containing lipoproteins, only LpAI was able to trigger the cellular cholesterol efflux (Fielding and Fielding, 1981; Barkia et al., 1991). The magnitude of the last one was diminished by the co-presence of LpAI: AII (Barkia et al., 1991). Consequently, LpAI was considered to be an anti-atherogenic particle, while an antagonist LpAI-role was suggested for LpAI: AII. These lipoproteins

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Abbreviations: HDL, High density lipoprotein; **TG,** triglycerides; **CVD,** cardiovascular disease; **LDL,** low density lipoprotein; **Chol,** cholesterol; **PL,** phospholipids; **NEFA,** non-esterified fatty acid**; LCAT,** lecithin:cholesterol acyltransferase; **CETP,** cholesteryl ester transfer protein; **CHD,** coronary heart disease.

appear to be decisive factors in the development of the atherosclerosis process. So, their quantification, particularly in physiopathology associated with an elevated atherosclerosis risk, could re-inforce the prediction of the risk of cardiovascular disease (CVD).

Obesity, which concerns a very large population (Kosti and Panagiotakos, 2006; Lazarou et al., 2008), is a physiopathology condition known for its association with increased atherosclerosis risk (Nguyen et al., 2010) which could be explained in terms of the co-presence of several risk factors (Marinou et al., 2010). In particular, it is well established that obesity is strongly associated with quantitative and qualitative alterations in plasma lipids (Bertière et al.,1988; Kissebah et al., 1989; Pascot et al., 2001; Ho et al., 2002; Bamba and Rader, 2007; Franssen et al., 2008; Korsten-Reck et al., 2008; Chan et al., 2009 ; Mori et al., 2009; Taha et al., 2009). These alterations classically included an elevation of triglycerides, a low HDL-Chol and an excess of small dense LDL fractions.

We have no information concerning the profile of apoAI-containing lipoproteins. The investigation of the impact of obesity on this profile is the principal objective of our study.

MATERIALS AND METHODS

Subjects

On the basis of the body weight excess, two volunteer groups were constituted. One control group was formed by 36 women having a body weight less than 110% of ideal body weight and one obese group was formed by 33 women having a body weight higher than 125% of ideal body weight. The ideal weights of persons were determined by LORENTZ formula and the weight excess was calculated as follows [(real weight - ideal weight) / real weight] x100.

In the order to avoid the sex and hormonal influence, all subjects were premenopausal women. Also, all recruited subjects have the same ethnic group and socioeconomic origins. They were aged between 24 and 40 years. They were not suffering from any known pathologies (diabetes, hepatic and kidney disease...), not using oral contraceptives or any other medications and they were not on a special diet. Except for three subjects (two obese subjects and one control) who smoked fifteen to twenty cigarettes daily, the tobacco use in others subjects was inexistent or very low (less than of 4 cigarettes/ day). For all subjects, the declared alcohol intake was inexistent.

Treatment of specimen samples

After an overnight fast, blood was collected on sodium azide (2 µg/ml), gentamycin (5 µg/ml) and disodium ethylene diaminetetraacetic acid (0.01 mM). Plasma was rapidly prepared by blood centrifugation at 6 - 8°C for 15 min at 3000 rpm. One ml of plasma was used for preparation of the HDL fraction.

The glucose assay was immediate, while other parameters were quantified in the two following days. During the 48 h, samples were kept at 4°C. Chol, TG, phospholipids (PL), LpAI, apoAI, apoAII and apoB were quantified in the plasma (p-Chol, p-TG and p-PL, p-LpAI, p-apoAI, p-apoAII and p-apoB) whereas only Chol, TG, LpAI and apoAI were assayed in the HDL fraction (HDL-Chol, HDL-TG HDL-LpAI and HDL-apoAI).

Analytical procedures

The level of glucose in the plasma was measured by the glucose oxidase method (glucose Analyser2, Beckman, USA). The HDL fraction was represented by the supernatant liquid obtained after precipitation of the plasma apoB-containing lipoproteins with phosphotungstic acid (Biomerieux, France), followed by centrifugation at 2500 rpm for 15 min. The non-existence of apoB in the supernatant was confirmed by electro-immunoassay using plates ready containing antibodies anti-apoB. PL (in the plasma) TG and Chol (in the plasma and in the HDL fraction) were quantified by enzymatic assays employing commercial kits (SERA-PAK for cholesterol and triglycerides; BIO-Merieux for phospholipids). ApoAII (in the plasma), apoAI, apoB and LpAI (in the plasma and in the "HDL" fraction), on the other hand, were quantified by differential electroimmunoassay using ready-to-use plates (SEBIA, France). The LpAI: AII concentrations were represented by the difference between the LpAI and apoAI levels. The amounts of apoAI and LpAI in non-HDL lipoproteins were considered as the differences between their respective values in the plasma and in the HDL.

Statistical analysis

The data were expressed as means (Standard deviation). The average concentrations obtained in the two subject groups were compared using Student-t test, while percentage were compared using the χ^2-test (chi-square test). Differences were considered as significant at $P < 0.05$. The linear correlation of TG and of apoAI-containing lipoproteins with other parameters was examined.

RESULTS AND DISCUSSION

The mean values (standard deviation) of analysed parameters obtained in two studied populations are shown in Table 1. Compared to the control subjects group, the obese subject group had a similar average age [30.7 (7.3) years vs. 32.4 (5.4) years] and fasting plasma glucose [4.97 (0.68) mmol/l vs. 4.76 (0.55 mmol/l)] but a higher weight excess [38.2 (7.8) vs 5.8 (4.1)%]. In addition, average concentrations of p-Chol, HDL-Chol, p-apoAI, p-apoAII and p-apoB were comparable in the two subject groups. However, obese subjects were differentiated through a significant increase of p-TG [1.31 (0.63) g/l vs. 0.83 (0.18) g/l], HDL-TG (0.23 (0.12) g/l vs 0.17 (0.09) g/l) and p-LpAI:AII [0.72 (0.16) g/l vs. 0.61 (0.11 g/l)] and a significant decrease of plasma-apoAI percentage associated with LpAI particles [(p-LpAI/ p-apoAI) x100] [40.77 (8.27) vs. 47.53 (8.65)%] ($P < 0.05$). Besides, a similar distribution of both apoAI and LpAI between the HDL and the non-HDL fraction was observed in the two subject groups; approximately 90% of apoAI and LpAI were associated to the HDL fraction.

The relation of TG, which was the unique changed lipid parameter, to other studied parameters was examined. A significant correlation was obtained with HDL-Chol ($r = 0.332$, $P < 0.05$), HDL-TG ($r = 0.416$, $P < 0.05$), LpAI:AII ($r = 0.532$, $P < 0.001$) and LpAI/ LpAI:AII ($r = 0.381$, $P < 0.05$).

The correlation coefficients of the LpAI, LpAI:AII and LpAI/LpAI:AII with studied traditional lipid and lipoprotein

Table 1. Comparison of control (n-Ob) and obese (Ob) subjects groups.

Parameter	n-Ob	Ob	n-Ob vs Ob
Age (years)	32.4 (5.4)	30.7 (7.3)	ns
Weight excess (%)	5.8 (4.1)	38.2 (7.8)	P < 0.001
p-Glucose (mmol/ll)	4.76 (0.55)	4.97 (0.68)	ns
p-Chol (g/l)	1.69 (0.39)	1.65 (0.24)	ns
p-TG (g/l)	0.83 (0.18)	1.31 (0.63)	P < 0.05
p-PL (g/l)	1.91 (0.25)	1.79 (0.24)	ns
HDL-Chol (g/l)	0.39 (0.14)	0.36 (0.09)	ns
HDL-TG (g/l)	0.17 (0.09)	0.23 (0.12)	
p-apoAI (g/l)	1.17 (0.12)	1.20 (0.17)	ns
p-apo AII (g/l)	0.25 (0.05)	0.23 (0.04)	ns
p-apoB (g/l)	0.76 (0.18)	0.77 (0.18)	ns
p-LpAI (g/l)	0.56 (0.12)	0.49 (0.10)	ns
p-LpAI:AII (g/l)	0.61 (0.11)	0.72 (0.16)	P < 0.05
p-LpAI/p-apoAI (%)	47.53 (8.65)	40.77 (8.27)	P < 0.05
HDL-apoAI/p-apoAI (%)	88.97 (4.73)	90.35 (2.10)	ns
HDL-LpAI/p-LpAI (%)	89.54 (4.66)	88.70 (2.01)	ns
HDL-TG	0.17	0.23	p< 0.05

Values are expressed as means (standard error). Concentrations and percentages were compared by the t-test and the $\chi 2$-test, respectively.

Table 2. Linear correlation coefficients of LpAI and LpAI:AII levels and of LpAI/LpAI:AII ratio with lipids and proteins parameters having known value of atherosclerosis marker.

Parameter	LpAI	LpAI:AII	LpAI/LpAI:AII
TC	-0.234 (ns)	+0.231 (ns)	-0.257 (ns)
TG	-0.176 (ns)	+0.532 (***)	-0.381 (*)
HDL-Chol	+0.108 (ns)	+0.032 (ns)	-0.042 (ns)
apo AI	+0.440 (*)	+0.635 (***)	-0.144 (ns)
apo B	+0.010 (ns)	+0.033 (ns)	-0.017 (ns)

*,*** Significant at P < 0.05 and 0.001, respectively; ns, not significant.

parameters was examined. Their values and their significations are summarized in Table 2.

The high prevalence of coronary heart disease associated with obesity has been attributed, at least partially, to the alteration of lipids and lipoprotein profiles. Notably, a decrease of HDL-Chol and an increase of both TG and LDL were commonly observed in obese populations (Bertière et al., 1988; Kissebah et al., 1989; Pascot et al., 2001; Ho et al., 2002; Franssen et al., 2008; Korsten-Reck et al., 2008; Chan et al., 2009; Ghandehari et al., 2009; Taha et al., 2009; Marinou et al., 2010). Among the assayed traditional lipid and lipoprotein parameters, only the TG concentration was affected (P < 0.05) in our obese group. In accordance with our observation, high TG levels were often observed in both humans and animals. This abnormality could be the results of different metabolic dysregulations resulting, notably, from the insulin resistance accompanying obesity (Hsiao et al., 2007; Marinou et al., 2010). The insulin resistance state leads to high levels of circulating non-esterified fatty acid (NEFA) (due to an excessive adipocytes lipolysis) and an increase of hepatic lipogenesis followed by an overwhelming hepatic secre-tory capacity (Marinou et al., 2010). Consequently, the liver could produce an abnormal HDL characterized by high TG levels which contribute to the observed HDL-TG rise.

Contrarily to what had been observed in soee studies, the high TG levels of our obese subjects group was not accompanied by the reduction of HDL levels. Also, TG and HDL-Chol showed a positive correlation in our obese subjects, while they have been reported to be associated negatively in other subject populations (Gou et al., 2005).

Our study investigates for the first time the profile of apoAI-containing lipoproteins which was proposed as a determinant factor in the development of the atherosclerosis process (Barkia, 1990). LpAI showed anti-atherogenic properties, while an opposite effect was proposed for LpAI:AII. Our results shows an increase of LpAI:AII (P

< 0.05), and no change of LpAI.

Previous studies reported that LpAI:AII are characterized by more TG content than LpAI (Barkia et al., 1991). This difference could be due, at least in part, to the presence of apoAII which could have a potential inhibiting effect on the catabolism of TG as suggested by the positive relationship between TG and apoAII observed in mice (Castellani et al., 2008). In our obese subjects, plasma apoAII concentration was unchanged. So, other causes than the reduction of TG catabolism must be considered to explain the enhancement of the TG levels in our obese group.

Also, the enhancement of LpAI:AII particles could justify the non rise of apoB concomitantly to the increase of TG. However, the possible existence of abnormal lipoproteins characterized by a high ratio of TG to apoB (TG/apoB), similar to those reported in homozygous stout rat (cp/cp) (Dolphin et al., 1987), must be considered.

The obesity state was associated with an alteration of the enzyme activities involved in the lipid and lipoprotein metabolism (lipoprotein lipase, cholesteryl ester transfer protein, hepatic lipase etc) (Marinou et al., 2010) which could lead to the alteration of the lipoprotein profile and composition. In agreement with this eventuality, our obese subjects seemed to have an abnormal distribution of apoAI between the two examined apoAI-containing particles. However, no alteration was observed concerning the distribution of both apoAI and apoAI-containing lipoproteins between HDL and non-HDL fractions (Table 1). Also, the correlation of HDL-TG with TG ($p < 0.05$) and with LpAI:AII ($p < 0.001$) suggests that obese LpAI:AII particles are principally of the HDL type.

Considering the impact of obesity on the apoAI-containing lipoproteins profile, our results cannot be compared with others. However, they appear to be concordant with the negative correlation existing between TG and HDL2/HDL3 ratio (Takahisa et al., 2005). Indeed, because LpAI:AII and LpAI are the major constituents of HDL3 et HDL2, respectively (Koren et al., 1987; Barkia, 1990), the observed variation of LpAI/ LpAI:AII indicates the reduction of HDL2/HDL3. Also, our result confirms the positive association of obesity with atherosclerosis development. Indeed, the observed increase of relative concentration of LpAI:AII which are smaller than LpAI, suggests the reduction of HDL size and, then, the increase of atherosclerosis risk. Effectively, large HDL particles have been associated with a reduction of atherosclerosis risk (Pascot et al., 2001).

Since the LpAI:AII have been shown to be able to reduce the cholesterol efflux from cell membrane promoted by the LpAI, an antagonist role to the one of LpAI (Barkia et al., 1991), the observed increase levels of LpAI:AII may represent a alteration of reverse cholesterol transport leading to an accumulation of cholesterol in non hepatic tissues.

High LpAI:AII levels would act at different manners. Firstly, to promote cellular cholesterol efflux, LpAI must interact with a specific receptors of cellular membrane (apoAI/ apoAII) which link LpAI:II as well (Barkia, 1991). The increase of the relative concentration of LpAI:AII decreases the LpAI chance to interact with the receptors leading to a reduction of cholesterol efflux, and thus to an accumulation of cholesterol in non-hepatic tissues.

Secondly, cholesterol reverse transport depends on the lecithin: cholesterol acyltransferase (LCAT) and cholesteryl ester transfer protein (CETP) which is important in the production of cholesterol esters and in their transfer from apoAI-containing lipoproteins to apoB-containing lipoproteins. Hence, the presence of adequate levels of these two activities is necessary for an adequate flow of cholesterol from cells to lipoprotein particles. The establishment of this condition requires the presence of the two apoAI-containing lipoproteins at adequate concentrations. Indeed, LpAI and LpAI:AII have different contents of LCAT and CETP (Cheung et al., 1986). Consequently, because they have an abnormal LpAI/LpAI:AII ratio, our obese subjects could have inadequate levels of LCAT and CETP activities. In accordance with this suggestion, it has been reported that the increase of triglycerides is associated with the loss of the normal positive LCAT-CETP correlation (Tato et al., 1997). Also, the activity of LCAT was reported low in individuals with high triglyceride values (Lee et al., 2001). While they have a normal level of traditional lipids and lipoproteins parameters, our obese group has clearly an altered apoAI-containing lipoproteins profile. On the basis of literature data, the observed change of this profile is favourable to the atherogenic processes.

Our results show that the interest of traditional lipid and lipoprotein parameters in the evaluation of the atherosclerosis risk could be limited, and they reinforce the validity of LpAI and LpAI:AII concentrations and/or the LpAI/ LpAI:AII ratio as markers of atherosclerosis risk which seem to have a more effective discrimination value. Future studies are necessary to quantify the independent contribution of this new parameter to the evaluation of coronary heart disease (CHD) risk and the identification of the exact anti-atherogenic apoAI-containing lipoproteins.

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