Iridoid enriched fraction from *Ajuga iva* reduce cholesterolemia, triacylglycerolemia and increase the lecithin:cholesterol acyltransferase activity of rats fed a cholesterol-rich diet

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**Summary**

**Objective:** In this study, we examined the effect of iridoid (I) derived from lyophilized aqueous extract of *Ajuga iva* on serum HDL2 and HDL3 compositions and lecithin:cholesterol acyltransferase (LCAT) activity, enzyme responsible for reverse cholesterol transport.

**Methods:** Male Wistar rats (n=24) weighing 120±5 g were fed a diet containing 1% cholesterol-rich diet for 15 days. After this phase, the hypercholesterolemic (HC) rats were divided into groups fed the same diet and received or not doses (5, 10 or 15 mg/kg b.w by intraperitoneal injection) of iridoid for 15 days.

**Results:** Compared to HC group, serum total cholesterol value was 1.4- and 1.2-fold lower in the I5-HC and I10-HC groups. C-HDL2 and C-HDL3 values were increased in the I5-HC, I5-HC and I10-HC groups (3.2- and 4.2-, 2.2- and 4.2-, and 3.2- and 8.7-fold, respectively). HDL2 amounts were 4-, 6- and 2.5-fold higher in the I5-HC, I10-HC and I15-HC groups. In HDL3, phospholipids contents were similar, whereas, unesterified cholesterol values were 3.3-, 2.8- and 3-fold higher in the I5-HC, I10-HC and I15-HC groups. In HDL3, cholesteryl esters contents were significantly higher in the groups treated with iridoid (p<0.05). LCAT activity was increased in the I5-HC and I10-HC groups.

**Conclusion:** Treatment with iridoid at doses 5 or 10mg/kg b.w reduce cholesterolemia. These molecules act efficiently on the efflux of cholesterol from peripheral tissues to the liver by increasing LCAT activity.

**Key words:**

Ajuga iva; HDL2; HDL3; Hypercholesterolemic rat; Iridoids; LCAT activity

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**Introduction**

Coronary heart disease (CHD) is the most common cardiovascular disease and atherosclerosis is considered as the most frequent cause of CHD [1] varying with time and population but more prominently with increased prosperity and longevity [2]. Lipoproteins are closely related to the risk of cardiovascular diseases, as follows: low-density lipoproteins (LDL) indicate an increased risk, and high-density lipoproteins (HDL) are considered a protective factor [3], because of its central role in reverse cholesterol transport [4]. Reverse cholesterol transport (RCT) is a complex process ensuring the efflux of cholesterol from peripheral cells and its transport back in the liver for its metabolism and biliary excretion [5]. The plasma enzyme lecithin:cholesterol acyltransferase (LCAT, EC 2.3.1.43) is of central importance to the lipoproteins metabolism. It is a key enzyme involved in the maturation of lipoproteins [6] and in the generation of cholesteryl esters in HDL, and is fundamental to the processes of reverse cholesterol transport [7], a mechanism generally held to be atheroprotective [8].

Plants have the potential to reduce lipid and cholesterol in body and encourage safety profile [9]. The presence of bioactive compounds in some plants involved reduction in lipids and cholesterol [10]. *Ajuga iva* (L.) Schreber (Labiateae) is used as an antihelmintic, against intestinal disorders [11], and as a diuretic agent [12]. According to ethnobotanical data collected in oriental Morocco by Ziyyat et al [13], *Ajuga iva*, locally known as ‘Chendgoura’, is also alleged to possess hypoglycemic activity and it is believed by many
Moroccan diabetics that the decoction of this plant consumed over a long time removes the cause of diabetes. Iridoids are found in many medicinal plants and may be responsible for some of their pharmaceutical activities. Isolated and purified, iridoids exhibit a wide range of bioactivities including cardiovascular [14] and hypolipidemic activities [15].

We hypothesized that consuming iridoid from Ajuga iva may involve changes in serum LCAT activity. First we tested the effect on serum lipids. Second we determined the effect on serum HDL₂ and HDL₃ amounts and composition. Third we evaluated if iridoid had an effect on LCAT activity in hypercholesterolemic rats.

Materials and methods

Preparation of the aqueous extract of Ajuga iva

Mature whole Ajuga iva (L) Schreber (Lamiaceae) (No. 2345) plants were collected in November 2004 from Bechar, southwest of Algeria, and stored at room temperature in a dry place before use. The aerial parts of Ajuga iva plant were dried at ambient temperature. Afterward, 500 mL of distilled water was added to 50 g of aerial plant finely powered, the mixture was heated under reflux for 60 min, and then the decoction was filtered. The filtrate was frozen at -20°C and lyophilized. The crude yield of the lyophilized material was approximately 18% w/w; it was stored at ambient temperature until further use [16].

Extraction and isolation of iridoid from Ajuga iva

Aqueous extract of Ajuga iva was added to H₂O-saturated n-butanol. After mixing, the supernatant was recuperated and evaporated to dryness in vacuum. The extract was submitted to a vacuum liquid chromatography on RP-C18 silica gel to eliminate the free sugar. Samples were subjected to flash chromatography to give different fractions. After evaporation of the solvent in vacuum, the fractions were diluted with H₂O; and the crude yield of the lyophilized material was approximately 2% w/w. The compound in the aqueous extract of Ajuga iva was identified as iridoid, and especially 8-O-acetylharpagide by comparison with literature data [17].

Animals and diets

Male Wistar rats (n=24) (Iffa Credo, l’Arbresle, Lyon, France) weighing 120±5 g were used in this study. Experimental hypercholesterolemia was induced by feeding normcholesterolemic rats (with total cholesterol value of 2.40±0.62 mmol/L) 1% cholesterol-enriched diet for 15 days (Table 1).

| Table 1. Ingredient composition of the cholesterol-enriched dieta |
|-----------------|--------|
| Ingredient      | g/kg   |
| Caseinb         | 200    |
| Corn starchc    | 542.5  |
| Sucrosec        | 40     |
| Sunflower oild  | 50     |
| Celluloseb      | 50     |
| Vitamin mixd    | 20     |
| Mineral mixe    | 20     |
| Cholesterolf    | 10     |
| Cholic acidg    | 5      |
| Methionineh     | 3      |

The diets contained 17.780 J/kg and was given in powdered form. aProlabo, Paris, France; bCommercial products, Oran, Algeria; cUAR 200, Villemeisson, Epinay, S/Orge, France (vitamin mix provides: thiamin 40, riboflavin 30, nicotinic acid 140, pyridoxine 20, pyridoxal 300, cyanocobalamin 0.1, ascorbic acid 1600, tocopherol 340, calcium panthenate 200, choline 2720, pteroylmonoglutamic acid 10, p-aminobenzoic acid 100, biotin 0.6, retinol 12 and cholecalciferol 0.125 mg/kg diet); dUAR 205, Villemeisson, Epinay, S/Orge, France (mineral mix provides: Ca 4, K 2.4, Na 1.6, Mg 0.4, Fe 0.12, Mn 0.032, Cu 0.005, Zn 0.018, Co 0.00004, I 0.00002 g/kg diet); eMerck, Darmstadt, Germany.

After this phase, serum total cholesterol (TC) concentration was measured and the value was 2.7-fold higher than that in the beginning of the study (6.5±0.6 mmol/L).

Hypercholesterolemic (HC) rats were divided into four groups fed for 15 days with the same diet; 3 groups were treated by different doses (5, 10 or 15 mg/kg b.w, intraperitoneal) of iridoid and one group was untreated. Diets and tap water were freely available. Animals were kept in wire bottom cages at temperature of 24°C, relative humidity of 60% and light were automatically turned on from 07:00 h to 19:00 h. We followed the ‘General Guidelines for the Use of Living Animals in Scientific Investigations’ [18] and protocol and use of rats were approved by the institutional committee on animal care and use.

Blood samples

At day 15, rats were food deprived for 12 hours, anaesthetized with sodium pentobarbital (60 mg/kg b.w) and euthanized with overdose. Blood was collected from abdominal aorta into dried tubes and centrifuged at 4°C, 1000g for 15 min. Serum aliquots were preserved in tubes containing 0.1% Na₂-EDTA and 0.02% sodium azide for different assays.

Isolation and characterisation of serum HDL₂ and HDL₃

Serum high density lipoproteins, i.e. HDL₂ and HDL₃, were determined by differential dextran sulphate magnesium chloride precipitation.
according to Burstein et al [19]. To estimate the validity of this method, ultracentrifugation according to Havel et al [20] was performed to quantitate lipoproteins.

Total protein content of each lipoprotein fraction was measured by the method of Lowry et al [21] using bovine serum albumin as standard. Total cholesterol and triacylglycerols (TG) concentrations were determined by enzymatic colorimetric methods (kits purchased from Human GmbH, Wiesbaden, Germany) and unesterified cholesterol (UC) was estimated by the enzymatic method (kit from Boehringer, Meylan, France). Esterified cholesterol (EC) was calculated from the difference between total and unesterified cholesterol. EC value was multiplied by 1.67 times to obtain cholesterol esters amount. Analysis of phospholipids (PL) was assessed by enzymatic determination (BioMerieux, Lyon, France).

**Assay for LCAT activity**

LCAT activity was determined by the conversion of [3H] UC to [3H] EC according to the method of Glomset and Wright [22] as modified by Knipping [23]. Cholesterol and egg phosphatidylcholines (PC) used for preparation of liposomes were purchased from Merck (Darmstadt, Germany). Radiolabelled 7(n)-[3H] cholesterol was obtained from Amersham (Les Ullis, France). Specifically, 2 mg cholesterol and 16 mg egg PC in chloroform-methanol (2/1, v/v) were evaporated to dryness under a nitrogen stream. After adding 1 mL of (10 mM Tris-HCl, 150 mM NaCl and 1 mM EDTA, pH 7.4), the solution was sonicated for 30 min at 100 W and 25°C with a sonifier (Bioblock Scientific, Strasbourg, Illkirch, France). The mixture was used within two days. The final cholesterol and PC contents were estimated according to the method of Assous & Girard [24] and with the CHOP-PAP test (kit from Boehringer, Mannheim, Germany), respectively. The final PC to cholesterol molar ratio was 4/1.

LCAT activity was determined using liposomes prepared by the procedure described above. For each assay, 88 nM of liposomes-cholesterol, 200 nCi [3H] cholesterol and 40 mg of fatty acid-free albumin (Merck, Darmstadt, Germany) were mixed. This mixture was preincubated for 1 h at 37°C, then 100 mL serum (enzymatic source) were added, together with 10 mL of 1 mmol mercaptoethanol per L (Merck). The amount of apoA1 in 100 mL of serum/mL assay mixture was sufficient for maximal activity when compared with other investigation by Knipping [23] The reaction was stopped by adding 20 volumes of chloroform-methanol (2/1, v/v). After 2 h, the mixture was filtered and the precipitate extracted three times with chloroform-methanol (2/1, v/v). The combined filtrate was concentrated in a vacuum and the water-free residue was extracted three times with 3 mL absolute ethanol (Prolabo, Paris, France). The lipid phase was evaporated to dryness and redissolved into 200 mL CHCl3, and 50 mL was applied on Kieselgel G 60 plates (Merck). Lipids were separated with petroleum ether-diethyl ether-acetic acid (60/30/1, v/v/v) and located by exposure to iodine vapour. After evaporation of iodine, the areas containing UC and EC were scrapped into a liquid scintillant in counting vials (Ready Solve HP/b, Beckman) and counted in a Beckman 7500 LS scintillation counter (Palo Alto, CA, USA). The cholesterol esterifying activity was expressed as µmol/h/L of serum.

**Statistical analysis**

Results were expressed as means ± SEM for 6 rats per group. Statistical evaluation of the data was carried out by the parametric Student t test. The calculations were performed using STATISTICA 6.0 for Windows software (StatSoft Inc., Tulsa, OK, USA). The limit of statistical significance was set at p<0.05 between the different groups treated or not with iridooids from Ajuga iva aqueous extract.

**Results**

**Serum TC, TG, C-HDL2 and C-HDL3 contents**

At day 15, serum TC concentration was 1.4- and 1.2-fold lower in the I5-HC and I10-HC groups compared with the HC group, respectively.

Serum TG values were significantly lower, 3-fold in the I5-HC group, 3.4-fold in the I10-HC group and 3.5-fold in the I15-HC group than the untreated group.

C-HDL2 and C-HDL3 values were increased 3.2- and 4-fold in the I5-HC group; 2.2- and 4.2-fold in the I10-HC group and 3.2- and 8.7-fold in the I15-HC group than the HC group, respectively (Table 2).

**Serum LCAT activity**

Compared to untreated HC rats, different doses of iridoids treatment for 15 days showed that LCAT activity was 1.5- and 1.6-fold higher in the I5-HC and I10-HC groups than the HC group, respectively (Table 2).

**Serum HDL2 amounts and composition**

Compared to HC group, the HDL2 amount, which was the sum of apolipoproteins (apo), TG, cholesteryl esters (CE), UC and PL contents (expressed in g/ L) were 4-, 4- and 2.5-fold higher in the I5-HC, I10-HC and I15-HC groups, respectively. Apo-HDL2 concentrations were
similar in all groups. The TG-HDL₂ value was higher, 3.8-fold in the I₃-HC group, 4.2-fold in the I₁₀-HC group and 2.4-fold in the I₁₅-HC group, respectively. The PL-HDL₂ concentrations were 1.5-, 2.2- and 2-fold increased in the I₃-HC, I₁₀-HC and I₁₅-HC groups. UC-HDL₂ contents were similar in all groups. CE-HDL₂ contents were increased, 4.3-fold in the I₃-HC group, 3.8-fold in the I₁₀-HC group and 2-fold in the I₁₅-HC group (Table 3).

**Serum HDL₃ amounts and composition**

The HDL₃ amount was 1.2-fold higher in the I₃-HC group than the HC group. Apo contents were similar in all the groups. TG-HDL₃ values were 2.3-fold higher in the I₃-HC group compared to the untreated HC group. PL contents were unchanged between groups. UC-HDL₃ values were higher, 3.3-fold in the I₃-HC group, 2.8-fold in the I₁₀-HC group and 3-fold in the I₁₅-HC group than the HC group. CE-HDL₃ contents were 3.5-fold increased in the I₁₀-HC group compared to the HC group (Table 4).

**Discussion**

In the present study, we have demonstrated that iridoids from the lyophilized aqueous extract of Ajuga iva reduces cholesterolemia and triglyceridemia, affects serum HDL₂ and HDL₃ amounts and composition and increases LCAT activity in rat fed cholesterol-enriched diet.

Our previous study has shown that Ajuga iva had no effect on TC in HC rats. This result has been explained, in part, by the similar values of lipid digestibility and fecal cholesterol excretion. In addition, Ajuga iva treated group ate more cholesterol (20 mg/d) compared to untreated group, which could be explained by enhanced C-HDL or and in cholesterol used [25]. However, in the present study, the iridoids treatment at doses 5 or 10 mg/kg b.w decreased significantly TC, but there was no sustained hypocholesterolemia in rat receiving 15 mg/kg b.w dose. The possible underlying mechanism by which iridoids could exert its cholesterol lowering activities was not elucidated.

At this stage of the study, several fundamental mechanisms can be proposed to explain our results. Iridoids may act by decreasing the cholesterol biosynthesis especially by decreasing the 3-hydroxy-3-methyl-glutaryl-coenzyme A reductase activity and/or by reducing the NADPH required for fatty acids and cholesterol synthesis.

**Table 2. Serum TC, TG, C-HDL₂ and C-HDL₃ values and LCAT activity of rats given the different treatments**

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th>I₃-HC</th>
<th>I₁₀-HC</th>
<th>I₁₅-HC</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC (mmol/L)</td>
<td>3.7±0.9</td>
<td>2.68±0.09</td>
<td>2.98±0.33</td>
<td>4.18±1.31</td>
</tr>
<tr>
<td>TG (mmol/L)</td>
<td>2±0.11</td>
<td>0.47±0.05</td>
<td>0.41±0.33</td>
<td>0.40±0.05</td>
</tr>
<tr>
<td>C-HDL₂ (mmol/L)</td>
<td>0.3±0.07</td>
<td>0.96±0.1</td>
<td>0.67±0.07</td>
<td>0.97±0.20</td>
</tr>
<tr>
<td>C-HDL₃ (mmol/L)</td>
<td>0.24±0.06</td>
<td>0.8±0.07</td>
<td>0.85±0.01</td>
<td>1.75±0.07</td>
</tr>
<tr>
<td>LCAT (nmol/h/mL serum)</td>
<td>9.01±1.41</td>
<td>14±0.91</td>
<td>15±1.2</td>
<td>10.1±2.6</td>
</tr>
</tbody>
</table>

Values are means ± SEM of 6 rats per group. *p < 0.05, I-HC treated vs untreated HC group.

**Table 3. Serum HDL₂ amounts and compositions of rats given the different treatments**

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th>I₃-HC</th>
<th>I₁₀-HC</th>
<th>I₁₅-HC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amount (g/L)</td>
<td>0.33±0.06</td>
<td>1.28±0.4</td>
<td>1.27±0.18</td>
<td>0.82±0.11</td>
</tr>
<tr>
<td>Apolipoproteins (g/L)</td>
<td>0.11±0.03</td>
<td>0.11±0.02</td>
<td>0.18±0.05</td>
<td>0.18±0.11</td>
</tr>
<tr>
<td>TG (mmol/L)</td>
<td>0.05±0.01</td>
<td>0.19±0.02</td>
<td>0.21±0.07</td>
<td>0.12±0.02</td>
</tr>
<tr>
<td>PL (mmol/L)</td>
<td>0.67±0.01</td>
<td>1±0.09</td>
<td>1.5±0.3</td>
<td>1.32±0.5</td>
</tr>
<tr>
<td>UC (mmol/L)</td>
<td>0.1±0.02</td>
<td>0.1±0.01</td>
<td>0.12±0.02</td>
<td>0.17±0.05</td>
</tr>
<tr>
<td>CE (mmol/L)</td>
<td>0.33±0.08</td>
<td>1.43±0.5</td>
<td>1.25±0.11</td>
<td>0.67±0.07</td>
</tr>
</tbody>
</table>

Values are means ± SEM of 6 rats per group. *p < 0.05, I-HC treated vs untreated HC group.

**Table 4. Serum HDL₃ amounts and compositions of rats given the different treatments**

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th>I₃-HC</th>
<th>I₁₀-HC</th>
<th>I₁₅-HC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amount (g/L)</td>
<td>4.46±0.35</td>
<td>5.24±0.16</td>
<td>6.19±1.28</td>
<td>5.46±0.95</td>
</tr>
<tr>
<td>Apolipoproteins (g/L)</td>
<td>4.2±0.3</td>
<td>4.6±0.1</td>
<td>5.4±1.2</td>
<td>4.9±0.85</td>
</tr>
<tr>
<td>TG (mmol/L)</td>
<td>0.07±0.01</td>
<td>0.16±0.01</td>
<td>0.05±0.01</td>
<td>0.07±0.02</td>
</tr>
<tr>
<td>PL (mmol/L)</td>
<td>1.5±0.2</td>
<td>1.78±0.35</td>
<td>1.80±0.49</td>
<td>1.9±0.3</td>
</tr>
<tr>
<td>UC (mmol/L)</td>
<td>0.18±0.02</td>
<td>0.59±0.07</td>
<td>0.51±0.06</td>
<td>0.53±0.09</td>
</tr>
<tr>
<td>CE (mmol/L)</td>
<td>0.4±0.01</td>
<td>0.37±0.03</td>
<td>1.4±0.09</td>
<td>0.37±0.08</td>
</tr>
</tbody>
</table>

Values are means ± SEM of 6 rats per group. *p<0.05, I-HC treated vs untreated HC group.
On the other hand, it can be suggested that iridoids led to a decrease in cholesterol absorption from the intestine and may have the ability to inhibit the intestinal absorption of bile acids and neutral steroids and to enhance hepatic cholesterol 7α-hydroxylase activity. Treatment with this iridoids could have a beneficial effect on dyslipidemia, particularly with the doses 5 and 10 mg/kg b.w.

The TGs are carried in plasma lipoprotein to provide energy for peripheral tissues and are independently related to coronary heart disease [26]. In this study, the decrease of serum TG level in the iridoids treated rats compared to the untreated rats was an important finding. Indeed, in the HC rats treated with Ajuga iva aqueous extract, a decrease in serum TG was observed compared to the untreated rats [16]. A reasonable explanation was the assumption that elevated post-heparin lipolytic activity, observed in Ajuga iva treated group (data not published) was sufficient to abolish the hypertriglyceridemia anticipated with cholesterol feeding.

Dyslipidemia is a primary risk factor for cardiovascular disease, peripheral vascular disease, and stroke. Current guidelines recommend diet as first-line therapy for patients with elevated plasma cholesterol concentrations [27]. In addition, high-cholesterol diet is regarded as an important factor in the development of cardiac diseases, since it leads to development of hyperlipidemia, atherosclerosis, and ischemic heart disease [28]. In our previous study, C-HDL$_2$ and C-HDL$_3$ values were increased in the Ajuga iva-HC than the untreated HC group [25]. The increased levels of C-HDL in the HC rats treated with aqueous extract from Ajuga iva may be due to the presence of bioactive molecules like iridoids. Effectively, in this study, iridoids treatments increased C-HDL$_2$ and C-HDL$_3$ compared to the untreated group.

In our study, the higher levels of cholesterol associated with HDL and the increase in the activity of serum LCAT in the I$_3$-HC and I$_{10}$-HC groups compared to the HC group may result in a higher amount of cholesterol being removed from extra hepatic tissues which may contribute to the hypocholesterolemia observed in these animals. Although the levels of total apo-HDL$_3$ values, including apoA1, the cofactor-activator of LCAT and PL-HDL$_3$ (substrate of LCAT) contents, are similar, the EC of HDL$_2$, a product of the reaction of LCAT are increased in all iridoids treated groups compared to the untreated group.

In addition, LCAT activity has been traditionally regarded as anti-atherogenic. The increase in LCAT activity observed in this study could, therefore, be a protective mechanism that counteracts iridoids-induced hyperlipidemia. This study showed that treatment with iridoids (5 and 10 mg/kg b.w) increases the activity of LCAT causing high levels of EC-HDL$_2$. By increasing the activity of LCAT with iridoids in these groups, cholesterol previously picked up from peripheral cells by nascent HDL would be esterified at a faster rate. The rate at which newly formed CE contained in HDL could be transferred to apoB-containing lipoproteins for subsequent elimination would also be increased. Taken together, the effects of iridoids on the activity of LCAT would seem to suggest that the iridoids-treated rats were attempting to clear the serum cholesterol burden as rapidly as possible.

To conclude, consuming iridoids from the lyophilized aqueous extract of Ajuga iva reduced triglyceridemia and hypercholesterolemia in hypercholesterolemic rats and specially at doses 5 or 10 mg/kg b.w. Iridoids appeared to ameliorate hypercholesterolemia probably by decreasing the exogenous cholesterol absorption and increasing the endogenous cholesterol conversion to bile acid, though to know the exact mechanism further studies are needed.

Acknowledgements
This research was funded by the Algerian Ministry of Higher Education and Scientific Research and by the French Foreign Office with International Research Extension Grant 04 MDU 629. The authors declared no conflict of interest.

Abbreviations
Apo (apo); apolipoprotein
b.w; body weight
CE; cholesteryl esters
EC; esterified cholesterol
HC; hypercholesterolemic
HDL; high density lipoproteins
I; iridoids
LCAT; lecithin:cholesterol acyltransferase
PL; phospholipids
TC; total cholesterol
TG; triacylglycerols
UC; unesterified cholesterol
References


