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Green tea extract (AR25®) inhibits lipolysis of triglycerides in gastric and duodenal medium in vitro

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In this study, we aimed to evaluate in vitro the inhibitory activity of a green tea extract (AR25® standardized at 25% catechins) on gastric and pancreatic lipase activities. We first used tributyrin as a substrate to evaluate the capability of AR25 to induce digestive lipase inhibition. Gastric lipase was totally inhibited by 40 mg AR25/g tributyrin whereas pancreatic lipase inhibition was maximum (78.8 ± 0.7%) with 80 mg AR25/g tributyrin. We then used triolein, a long-chain triglyceride, to check whether AR25 could alter lipase activities on a physiologic substrate. AR25 60 mg/g triolein induced a dramatic inhibition of gastric lipase (96.8 ± 0.4%) whereas pancreatic lipase activity was partially reduced (66.50 ± 0.92%). Finally, the concerted action of gastric and pancreatic lipases was studied with an excess of enzymes to mimic the physiologic conditions observed in vivo. Incubation of AR25 with an excess of digestive lipases resulted in a drastic decrease in gastric lipolysis but the inhibitory effect on pancreatic lipase was less marked. On the whole, as compared to the control, lipolysis of triolein under the successive action of the two digestive lipases was reduced by 37 ± 0.6% in the presence of AR25. Because a lipid/water interface is necessary for lipolysis to occur, lipid emulsification and emulsion droplet size were measured in gastric and duodenal media in the presence of AR25. In gastric and duodenal conditions, AR25 inhibited the lipid emulsification process. From these data we conclude that (1) in vitro, fat digestion is significantly inhibited by 60 mg AR25/g triolein, and (2) gastric as well as pancreatic lipase inhibition could be related to altered lipid emulsification in gastric or duodenal media. The green tea extract AR25 exhibiting marked inhibition of digestive lipases in vitro is likely to reduce fat digestion in humans. (J. Nutr. Biochem. 11:45–51, 2000)

Keywords: green tea extract; gastric lipase; pancreatic lipase; triglycerides; emulsion

Introduction

It is well established that digestion of dietary triglycerides in the gut is governed by two key enzymes in humans and most mammals.[1,2] The first step of fat digestion starts in the stomach where gastric lipase, secreted in the gastric juice, hydrolyzes 10 to 30% of dietary triglycerides under acidic conditions.[3–5] Dietary triglyceride lipolysis is completed by pancreatic lipase acting with colipase as cofactor, in the small intestine where absorption of lipolytic products occurs.[1] In healthy humans, intragastric lipolysis, even limited, appears to initiate fat digestion and emulsification and thereby facilitate lipolysis catalyzed by pancreatic lipase in the duodenum.[5,6] Because triglycerides are insoluble substrates, the characteristic feature of gastric and pancreatic lipases is that both enzymes act at the lipid/water interface.[2] Studies from our laboratory, performed in healthy humans, showed that dietary fats are basically emulsified in the stomach and duodenum contents, with most droplet sizes in the range 10 to 50 μm.[5,6] Under these conditions of heterogenous catalysis, the extent of enzyme binding to the interface as well as lipolysis depend on the concentration and the properties of the interface.[2,7,8] Moreover, emulsified droplet size and composition alter the binding and the activity of gastric and pancreatic lipases.[6–10]

From epidemiologic studies in humans and from in vitro experiments, it has been suggested that chronic ingestion of

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green teas (*Camellia sinensis*) have a protective effect on the development of several cancers.[11] Some hypocholesterolemic effects[12] as well as the ability of green tea components to prevent low density lipoprotein oxidation also were reported.[13,14] Mainly catechins and others polyphenols are implicated in these processes. Although the effect of some polyphenols on digestive enzymes was investigated,[15] the specific effect of green tea catechins on digestive lipases is poorly documented.

In this study, we aimed to evaluate whether gastric and/or pancreatic lipase activities could be inhibited by the well-defined green tea extract AR25®, standardized at 25% catechins. The results obtained showed that in vitro AR25 markedly inhibited gastric and pancreatic lipase activities measured on short-chain or long-chain triglycerides as substrates. Such effects make the natural green tea extract AR25 a candidate for reducing fat digestion and assimilation in vivo and a potential complement to the pharmacologic and dietary treatments of obesity.

Materials and methods

Lipids

Tributyrin, a short-chain triglyceride, and triolein, a long-chain triglyceride, were purchased from ICN (ICN Biomedicals Inc., Eschwege, OH USA). L-α-phosphatidylcholine type XVI-E from egg yolk, free cholesterol, and taurodeoxycholic acid (TDC) were from Sigma (La Verpillière, France). Radiolabeled triolein ([9,10-3H(N)]-1.0mCi/2 mL) was purchased from Du Pont de Nemours Gmbh (NEN Life Science Products Division, Dreiech, Germany). Bile was collected from pig gallbladder (Abattoirs de l'Estaque, Marseille, France), analyzed for bile salt concentration, sampled, and stored at −70°C before use.

Enzymes

Human gastric lipase was obtained from gastric aspirates collected for diagnostic purposes in adult patients under pentagastrin stimulation in hospital facilities (gifts from Dr. Peyrot, Gastroenterology Department, University Hospital, Marseille, France). Samples were neutralized to pH between 4 and 5 and those with comparable high activities were pooled and stored at −20°C. All experiments were done using the same enzyme source. The lipase activity of the pooled gastric juices was 150 units/mL on tributyrin as substrate. Pure gastric lipase was obtained following the procedure of Tiruppathi and Balasubramanian[16] with a specific activity of 1,000 units/mg on tributyrin.

Pancreatin (Sigma) was used as a source of total pancreatic enzymes. Pure porcine pancreatic lipase and colipase were purchased from Boehringer Mannheim (Mannheim, Germany) and their specific activities were 1,370 units/mg and 1,590 units/mg, respectively.

Gastric and pancreatic lipase activities were measured at pH 5.4 (optimal pH for human gastric lipase) and 7.5, respectively, which are in the range of gastric and intestinal pH values reached in the postprandial state.[5,6]

AR25 (green tea extract standardized at 25% catechins)

AR25 (active compound of Exolise® capsules) was obtained from Arkopharma Laboratories (Carros, France) and was used as a powder. AR25 contains 53% total polyphenols, including 25% catechins and 5% caffeine. The catechins contained in AR25 are mainly represented by the epigallocatechin-3-gallate. The quantities to be tested in vitro were calculated taking into account a reasonable daily AR25 intake of 1,500 mg/day in humans and the usual range of fat ingested (50–150 g/day) in western countries.[17]

Measurements of gastric and pancreatic lipase activities on tributyrin

To evaluate the potential inhibitory effect of AR25, gastric and pancreatic lipases activities were measured on tributyrin with or without (control) AR25 using a pH-stat Titrator (Metrohm, Herisau, Switzerland) at pH 5.4 and 37°C.[18] To determine the inhibition dose response, several quantities of AR25 extract were used (range 0–80 mg/g of tributyrin). The reaction medium was a 15 mL system containing 5 mM sodium acetate, 150 mM NaCl, 6 mM $CaCl_2$, 1.5 μM bovine serum albumin (BSA; fatty acid free from Sigma), 2 mM TDC, and 0.5 g tributyrin as already described.[8–10] Prior to the addition of pure gastric lipase (10 IU), the reaction medium was mechanically stirred at 37°C for 5 minutes. At the end of the reaction time (1 min at pH 5.4), the butyric acid released was fully titrated at pH 9.4.[8–10] Parallel controls were run without enzyme and the values obtained were subtracted from the corresponding values of lipase activity measurement.

Activity of pure pancreatic lipase was measured at pH 7.5 and at 37°C in a 15 mL reaction medium containing 150 mM NaCl, 2 mM Tris, 10 mM $CaCl_2$, 8 mM bile salts from pig bile, and 0.5 g tributyrin as previously described.[19] After being mechanically stirred for 5 minutes, colipase and then lipase (12 IU) were introduced in the reaction medium containing or not containing (control) 10 to 80 mg AR25/g tributyrin. Pancreatic lipase activity was recorded until the maximal velocity was reached for 3 minutes. One lipase unit (IU) was defined as 1 μmole of fatty acid titrated per minute.

Preparation of triolein emulsion and droplet size measurements

The relative proportions of lipids used were chosen in the range of human daily dietary intake (i.e., 100 g triglycerides, 2.5 g phospholipids, 0.5 g free cholesterol).[17] Thus, a lipid mixture was prepared with 97% (w/w) triolein, 2.5% (w/w) L-α-phosphatidylcholine, and 0.5% (w/w) free cholesterol plus [^{3}H]-triolein to reach a specific activity of 30,000 dpm/μmol. The lipid mixture was dissolved in chloroform/methanol 2:1 (v:v). Solvent was evaporated to dryness under nitrogen and the lipid mixture obtained was stored at −20°C.

The standard emulsion was prepared by sonication of the lipid mixture (100 mg in 3 mL distilled water) for 5 minutes at 20°C and on ice (Sonifer Branson 250W, Osi, France). These operating conditions resulted in an emulsion measuring 2 μm in median diameter. The droplet size of the emulsion was determined using a particle-size analyzer (Capa 700, Horiba, Kyoto, Japan) as previously described.[5,8,9] Measurements were carried out using gradient mode analysis at a constant centrifuge acceleration rate (1 g/min) that allowed an accurate measurement of large (100 μm) to small (0.09 μm) particles. Results are given as droplet size distributions calculated by the particle-sizer software as a fraction (%) of the total volume occupied by lipid droplets in a given sample. The median size values were calculated by the particle-sizer software from the droplet size distribution.

Measurements of gastric and pancreatic lipase activities on a long-chain triglyceride (triolein)

Lipase activities were assayed in the absence (control) or presence (60 mg/g triolein) of AR25 using the triolein radiolabeled emulsion and with an excess of substrate.

Pure gastric lipase activity was assayed at pH 5.4 in a 0.5 mL reaction medium containing 100 mM sodium acetate, 150 mM NaCl, 6 mM $CaCl_2$, 1.5 μM BSA, and 5 μmol (4.41 mg) triolein. Gastric lipase (75 mIU) was added 5 minutes after stirring the reaction medium. Gastric lipase reactions were carried out for 15 minutes at 37°C while stirring.

Pure pancreatic lipase activity was measured at pH 7.5 in a 0.5 mL system composed of 150 mM NaCl, 200 mM Tris, 6 mM $CaCl_2$, 8 mM bile salts from pig bile, and 5 μmol (4.41 mg) triolein. Reaction medium was stirred for 5 minutes prior to the additions of colipase and lipase (20 mIU). Lipolysis was performed for 5 minutes at 37°C while stirring.

In both cases, lipolyses were stopped with 1 mL chloroform/methanol (2:1, v:v). Lipids were extracted using the method of Folch et al.[20] To ensure complete protonation of fatty acids, the organic solvent phases were partitioned with 20% (vol/vol) 150 mM NaCl solution containing 2% glacial acetic acid (vol/vol, pH 3.0).[5] The radiolabeled neutral lipid classes (i.e., triglycerides, diglycerides, monoglycerides, fatty acids) were separated by thin layer chromatography (TLC) on silica gel (ready plastic sheet F 1500, Schlecher and Schuell, Dassel, Germany) using a chloroform/methanol/acetic acid (98:2:0.1, v:v:v) system. The plates were exposed to iodine to allow identification of the different spots. TLC silica corresponding to the fatty acid spots were scrapped and [^{3}H] oleic acid was measured by scintillation counting with a Packard 1600TR equipment (Packard, Meriden, CT USA).

Determination of the extent of gastric and pancreatic lipolysis under physiologic conditions

In this set of experiments, excess amounts of enzymes were sequentially added to emulsified long-chain triglycerides. The emulsions were first submitted to hydrolysis by gastric lipase using whole gastric juice at pH 5.4 for 30 minutes. The initial reaction medium was 1 mL mixture of 100 mM sodium acetate, 150 mM NaCl, 6 mM $CaCl_2$, 1.5 μM BSA at pH 5.4, and 10 μmol triolein from the radiolabeled emulsion. The kinetic study started just after the addition of gastric juice (25 IU gastric lipase) and took place at 37°C while stirring. Aliquots (100–200 μL) were collected at 5, 15, and 30 minutes to determine the amounts of generated free fatty acids. At the end of the incubation period in gastric conditions, the volume of the reaction medium was completed to 1 mL with a buffer solution containing 150 mM NaCl, 200 mM Tris, 6 mM $CaCl_2$, and 8 mM bile salts from pig bile (pH 7.5). Pancreatic colipase and lipase (75 IU) provided by pancreatin were added and the reaction lasted up to 60 minutes. Free fatty acids that were released were measured in aliquots (200 μL) collected 15, 30, and 60 minutes after pancreatin addition.

In gastric and duodenal conditions, free fatty acids were extracted, separated, and quantified as described above. The total amount of free fatty acid generated was calculated taking into account the successive samplings.

Emulsification measurements

The percentage of emulsified lipids as well as the droplet size of the formed emulsion was determined in gastric or duodenal media, in presence or absence (control) of AR25 (6 mg) following a procedure already described.[9,10] Herein 3 mL of either gastric buffer (50 mM sodium acetate, 150 mM NaCl, pH 5.4) or duodenal buffer (150 mM NaCl, 50 mM Tris, 8 mM bile salts from pig bile, pH 7.5) were mixed with 100 mg of the [^{3}H] radiolabeled lipid mixture in 7 mL glass tubes. The tubes were stoppered, attached horizontally, and shaken at 200 strokes/min for 2 hours at 37°C. These operating conditions generated lipid emulsions with droplet size in the range of those found in the human digestive tract during fat digestion.[5,7–10]

At the end of the emulsification process, the tubes were left to stand in a vertical position to allow the building of a floating oily layer made of unemulsified lipids (oily materials plus lipid droplets $\geq$ 100 μm) above the infranatant aqueous solution. The upper limit for emulsion droplet size was set at 100 μm given the instability and negligible interfacial area of lipid droplets above this value.[5] The time needed (approximately 10 min) was calculated from Stoke's sedimentation equation for droplets measuring 100 μm or more as previously reported.[5,9] The relation between sedimentation time and particle diameter is expressed in the following equation for natural gravity sedimentation: $D = [18\ n_o\ H / (\rho - \rho_o)\ gt]^{1/2}$, where *D* is the particle diameter (m), n_o is the viscosity coefficient of the solvent (N.s/m^2), ρ is the density of the sample (kg/m^3), ρ_o is the density of the solvent (kg/m^3), *g* is the acceleration due to gravity (m.s^{-2}), *t* is the sedimentation time (s), and *H* is the distance of sedimentation (m). Thus two fractions were collected; the first one was the upper layer composed of unemulsified lipids and the second one the infranatant containing emulsified lipid droplets.

Radioactivity was measured by liquid scintillation counting from 100 μL of the floating oily layer fraction. The distribution of the emulsion droplet size in the infranatant solution was determined using a particle-size analyzer as noted above. The percentage of emulsified triglycerides was calculated from the initial amount of radiolabeled triglyceride in the lipid mixture and the quantity of triglycerides recovered in the floating oil fraction.

Statistics

All experiments were performed in triplicate and the results are expressed as means ± SEM. The statistical significance of the differences observed was assessed by one-way analysis of variance and Fisher's test ($P < 0.05$) using the Stat-View II micro-computer program (Abacus, Berkeley, CA USA).[21]

Results

Dose-response inhibitory effect of AR25 on short-chain triglycerides

As shown in *Figure 1*, the lowest dose of AR25 tested induced a drastic inhibition (79.06 ± 0.85%) of gastric lipase. When 80 mg AR25/g tributyrin was added, we observed a total inhibition of gastric lipase activity. In contrast, under the same conditions, inhibition of pancreatic lipase was slightly less marked and reached 78.76 ± 0.68%. From these data, we concluded that 60 mg AR25/g triglyceride could be used to evaluate the AR25 inhibitory effect on hydrolysis of long-chain fatty acid triglycerides by the two lipases.

Inhibitory effect of AR25 on emulsified long-chain triglycerides

The figures for hydrolysis of triolein by the two digestive lipases with or without AR25 (60 mg/g triolein) are given in *Table 1*. Most gastric lipase activity was abolished by AR25 (inhibition: 96.79 ± 0.39%). AR25 reduced pancreatic lipase activity to a lesser extent (inhibition: 66.50 ± 0.92%).

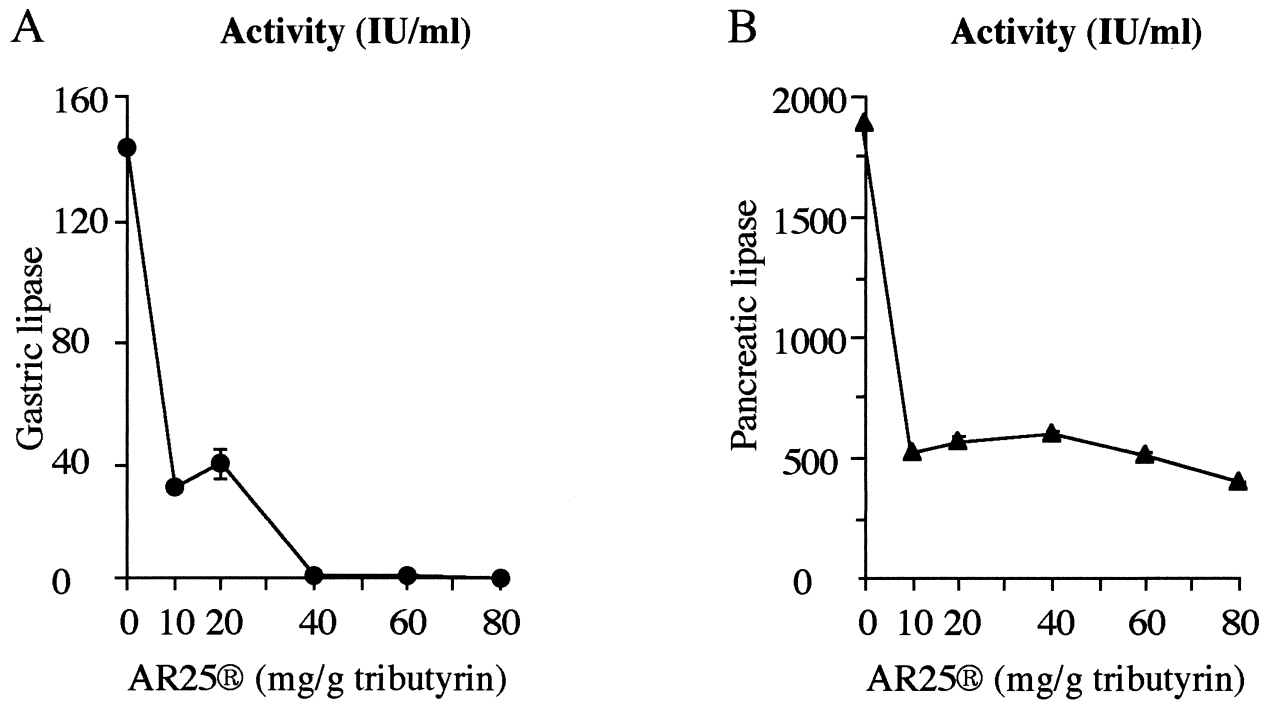

Figure 1 Effect of increasing amounts of AR25® (mg/g tributyrin) on (*A*) gastric lipase and (*B*) pancreatic lipase activities. Tributyrin hydrolysis in gastric or duodenal operating conditions was measured using a pH-stat Titrator (see text for experimental details). Values are means ± SEM.

Inhibitory effect of AR25 measured in physiologic conditions

To mimic the in vivo conditions, triolein emulsion was successively incubated with an excess of gastric lipase in acidic medium and an excess of pancreatic lipase and colipase in duodenal conditions. As shown in *Figure 2*, we compared the fatty acid released in absence (control) or presence of AR25 (60 mg/g triolein). Compared with the control, the amount of fatty acids released was markedly and significantly lower (11.7 ± 5.2% of control) during gastric lipolysis. The inhibitory effect of AR25 on pancreatic lipolysis appeared to be delayed compared with the result obtained in excess of substrate and was observed at 60 minutes (i.e., 30 minutes after the pancreatic lipase and colipase additions). On the whole, lipolysis catalyzed by the action of both gastric and pancreatic lipases on triolein was significantly reduced by 37.1 ± 0.6%.

Table 1 Effect of AR25® (60 mg/g triolein) on gastric and pancreatic lipases activities

	Lipase activities (IU)	
	Gastric lipase	Pancreatic lipase
Control	11.37 ± 0.27	484.44 ± 9.07
AR25	0.36 ± 0.04*	162.27 ± 4.43*

Lipase activities are expressed as IU (μmol free fatty acid released/min) and were measured using excess triolein emulsion (median diameter 2 μm) as described in Materials and methods. Values are means ± SEM.
*Significantly different from control (one-way analysis of variance and Fisher's test at $P < 0.0001$).

Effect of AR25 on triolein emulsification

AR25 (60 mg/g triolein) dramatically altered lipid emulsification in gastric medium (*Table 2*). In duodenal medium, the presence of AR25 completely prevented lipid emulsification.

Discussion

The aim of this study was to evaluate the possible inhibitory effect of the green tea extract AR25, containing 25% catechins, on digestive lipases in vitro.

We first demonstrated an inhibitory effect of AR25 on digestive lipases employing widely-used tributyrin as a short-chain triglyceride substrate.[18] Results obtained from this experiment in the presence of excess tributyrin showed that AR25 inhibited gastric lipase and pancreatic lipase activities. The highest inhibitory effect for gastric and pancreatic lipases was induced by 40 mg and 80 mg AR25 per gram of fat, respectively. In further experiments we investigated the inhibition of digestive lipases by AR25 using another classical substrate, triolein, which is more representative of dietary long-chain triglycerides. Using triolein, gastric lipase was drastically inhibited whereas pancreatic lipase was inhibited to a lower extent.

These two sets of experiments clearly showed that activities of both gastric and pancreatic lipases are markedly reduced in the presence of green tea extract AR25. AR25 is a green tea extract containing 25% catechins, mainly in the form of epigallocatechin-3-gallate. This compound is identified as a nonhydrolyzable polyphenol also called condensed tannin. It has already been shown in vitro that some condensed tannins have an inhibitory action on certain

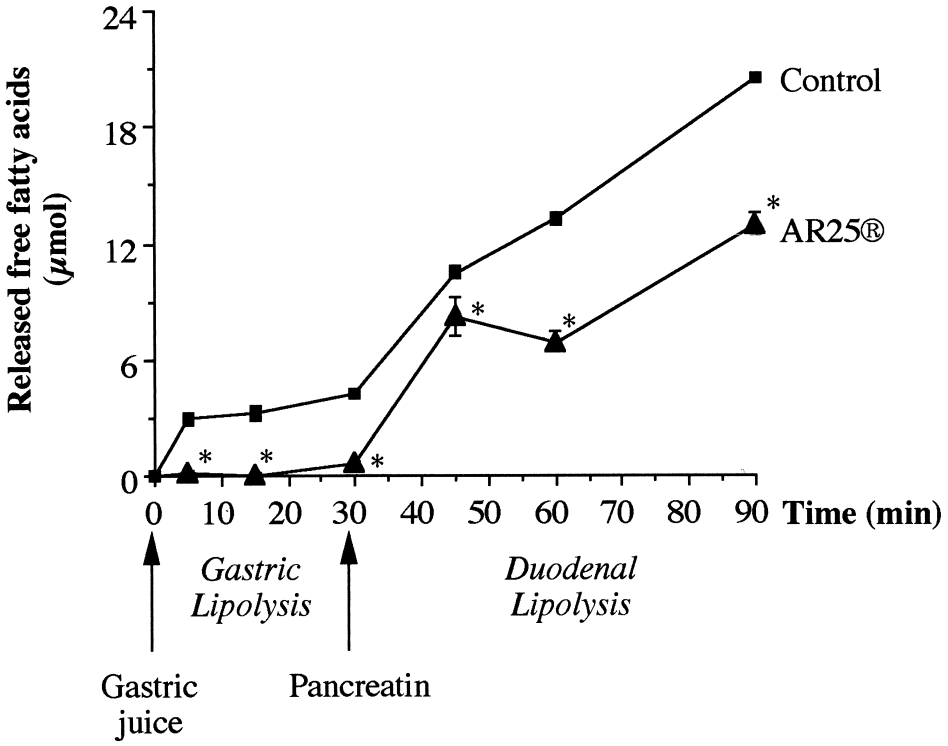

Figure 2 Fatty acid released (μmol) in absence (control, ■) or presence of AR25® (60 mg/g triolein, ▲). Triolein emulsion (median diameter 2 μm) was incubated for 30 minutes in acidic conditions with gastric lipase. At 30 minutes, and up to 90 minutes, gastric medium was replaced by neutral medium providing pancreatic lipase and colipase and bile. In these conditions, gastric lipolysis lasted 30 minutes and duodenal lipolysis lasted 60 minutes. Values are means ± SEM. *Significant difference from control at a given time point (one-way analysis of variance and Fisher's test at $P < 0.001$).

digestive enzymes such as proteases, α-amylase, and pancreatic lipase.[15] Nevertheless, the inhibition of digestive enzymes, especially lipases, by green tea catechins is poorly documented compared with that of other vegetable condensed tannins.[15] A recent report has described the inhibition of salivary amylase, measured intraorally, in the presence of green tea.[22] More recently, Han et al.[23] failed to demonstrate any inhibitory effect of tea catechins on pancreatic lipase activity whereas oolong tea extract induced pancreatic lipase inhibition. Nevertheless, the lack of a defined catechins/lipase ratio used in this experiment does not allow comparison with data from the present study. This does not rule out that other components of tea extract might be implicated in some inhibitory effect.

In the two first sets of experiments, the inhibition of the two digestive lipases by AR25 was measured separately. In fact, in vivo, triglyceride hydrolysis is first initiated by an excess amount of gastric lipase under acidic conditions[1,3] and is then completed by an excess amount of pancreatic lipase in neutral conditions.[1] Thus, to mimic normal in vivo conditions, we measured the inhibitory effect of AR25 on triolein lipolysis by incubating gastric lipase, from gastric juice, and then pancreatic lipase and colipase, provided by pancreatin. Compared with the data obtained for activity with excess amount of triglycerides, gastric lipolysis was still markedly inhibited whereas duodenal lipolysis was moderately inhibited 30 minutes after pancreatin addition. Total reduction of fat digestion reached 37% under these in vitro mimicking physiologic conditions.

It has been suggested that high molecular weight polyphenols form insoluble complexes with proteins or other macromolecules. The process is established by the interaction of at least two hydroxyl groups of polyphenols with proteins.[15] Alteration on protein digestion by condensed tannins is supposed to be achieved in part following this process.[15] The fact that catechins, especially epigallocatechin-3-gallate, possess more than two hydroxyl groups does not implicate their participation in such interactions.

Several studies have already demonstrated that dietary fibers inhibit gastric and pancreatic lipases in vitro and in

Tsble 2 Effect pf AR25® (60 mg/g triolein) on lipid emulsification and droplet size of formed emulsions in gastric and duodenal media

	Emulsified lipids (%)*		Emulsion droplet size (μm)†	
	Gastric medium	Duodenal medium	Gastric medium	Duodenal medium
Control	36.61 ± 4.24	64.93 ± 1.37	4.49 ± 1.61	30.69 ± 0.61
AR25	2.15 ± 0.56	<1[a]	—	—

*The percentage of emulsified lipids was calculated using the measurement of radiolabeled triolein in the floating oily layer, composed of unemulsified lipids (see text for experimental details.
†Median diameters were calculated from droplet size distribution by the particle size software. Values are means ± SEM.
[a]Significantly different from control (one-way analysis of variance and Fisher's test at $P < 0.001$).

vivo.[24] In some cases (i.e., wheat germ) inhibition of pancreatic lipase activity was related to the ability of wheat germ proteins to interact with the emulsified substrate.[25] In vitro experiments, mimicking the prevailing in vivo situation, showed that soluble dietary fibers displaying sufficient viscosity in solution reduced activity of digestive lipases.[9,10] The authors concluded that soluble viscous fibers alter lipid assimilation by reducing emulsification of dietary lipids in stomach and duodenum and subsequent lipolysis. To determine one possible mechanism involved in lipase inhibition by AR25, lipid emulsification was measured in vitro. Herein we observed that AR25 fully abolished lipid emulsification. Indeed, we do not know by which physicochemical mechanism the green tea extract, or specifically catechins, could exert such a marked effect on fat emulsification. This indicates that the overall inhibitory effect of AR25 could be the sum of a reduction of triglyceride emulsification and a direct effect on lipolysis reaction (−37% as measured herein).

Results from this in vitro study clearly demonstrate the inhibitory effect of a green tea extract containing 25% catechin on digestive lipases. Although experiments were made in vitro, this data prompts us to speculate that the inhibitory effect of AR25 could be observed in vivo. This hypothesis is supported by the reduced fat digestion (−37%) obtained under a protocol design carefully mimicking the physiologic conditions of the digestive tract. The extent of inhibition could even be amplified if reduction of fat emulsification occurred in vivo. Moreover, the inhibitory effect of AR25 on lipolysis catalyzed by the two lipases was measured using 60 mg AR25/g triolein, which corresponds with a reasonable daily dose (1,500 mg) and the amount of dietary fat consumed by food restricted subjects (30–50 g/day).

Considering that dietary fats, due to their high caloric value, play a role in the development of obesity,[26] reduction of fat digestion is considered as a novel approach in obesity treatment.[27] Recently, a gastrointestinal lipase inhibitor (Xenical®, containing the microbial derivative tetrahydrolipstatin) (Produits Roche, Neuilly sur Seine, France) was proposed to induce weight loss in obese subjects. In a placebo-controlled trial, it was demonstrated that Xenical reduced dietary fat digestion by approximatively 30% (apparent maximum percentage of ingested fat excreted in the feces due to tetrahydrolipstatin was 27%), thus reducing energy intake.[28] Xenical promotes weight loss and lesser weight regain and limits some obesity-related disease risk factor.

Specific compounds from dietary origin are poorly used in obesity treatment. It has been proposed that to prevent obesity, especially in childhood,[29] the recommended fiber intake should be doubled. Dietary fibers are supposed to reduce caloric density and decrease lipid absorption rate, and are known to have beneficial effects in several chronic metabolic diseases.[24] In addition, this study now clearly shows that a specific green tea extract, AR25, has fat digestion lowering properties. Given that the in vitro inhibitory effect of AR25 on digestive lipases is confirmed in humans, this natural product rich in catechins could be of interest to limit fat digestion and reduce body weight in obese patients and to improve metabolic status.

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