White and green tea polyphenols inhibit pancreatic lipase in vitro

Anais Gondoin, Dominic Grussu, Derek Stewart, Gordon J. McDougall

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Obesity
Health
Tannins

1. Introduction

Tea is often quoted as being the most commonly consumed beverage in the world other than water. The majority of commercial teas arise from dried leaf material from Camellia sinensis L. and a wide range of different teas can be produced. The main consumed types are black and green tea but recently white tea has become more available to consumers in the West. These teas mainly differ in their degree of processing but white tea is generally composed only of the unopened bud and/ or first leaves (Hilal & Engelhardt, 2007). After picking, white tea is dried and retains the white leaf hairs from which it derives its name. Green tea is heat-treated (steaming and/or pan-frying) to inactivate endogenous polyphenol oxidase (PPO), rolled and dried. At the other extreme, during black tea production there is no heat-inactivation of PPO and a “fermentation” or oxidation phase after rolling allows large-scale PPO-catalyzed conversion of simple phenolics to more complex forms and forms the dark coloration. As a result, white, green and black teas differ in their sensorial properties and have markedly different chemical compositions (e.g. Del Rio et al., 2004; Hilal & Engelhardt, 2007; Mizukami, Sawai, & Yamaguchi, 2007; Wang & Ho, 2009).

The major phenolics present in teas are the flavan-3-ols and the flavonols. The flavan-3-ols are characterized by (-)epicatechin and its galloylated derivatives, especially in green tea, whereas black tea has lower amount of these derivatives due to their oxidative conversion into theaflavins and thearubigins (Balentine, 1992; Del Rio et al., 2004; Finger, Kuhr, & Engelhardt, 1992; Mizukami et al., 2007). The flavonols are mainly derivatives of quercetin and kaempferol (Del Rio et al., 2004; Finger, Kuhr, & Engelhardt, 1992, 1998) but there are smaller amounts of tannins and hydroxycinnamate derivatives. Of course, teas also contain substantial and physiologically relevant levels of caffeine and theobromine (e.g. Roberts & Barone, 1983).

Epidemiological studies have suggested correlations of tea intake with favorable outcomes with regard to cardiovascular disease (Grassi et al., 2008), cancer incidence (Yang, Maliakal, & Meng, 2002), inflammation (Gonzalez de Mejia, Vinicio Ramirez-Mares, and Puangpraphant, 2009), obesity (Hsu & Yen, 2007; Wolfram, Wang, & Thielecke, 2006) and type 2 diabetes risk (Venables, Hulston, Cox, and Jeukendrup, 2008). Indeed, a range of mechanisms have been proposed for the beneficial effects of tea and health (e.g. Higdon and Frei, 2003), which largely focus on the polyphenol components, especially the flavan-3-ols.

Considering the epidemic of obesity now forecasted for the Western World (Anon, 2003), the possibility that tea intake could influence obesity through lipid metabolism and digestion is intriguing. Tea, and phenolic components of tea, have been suggested to have anti-obesity and anti-diabetic effects in humans (Kao, Chang, Lee, & Chen, 2006), to reduce adipose mass in rodent models (e.g. Kim et al., 2009) and to influence lipid digestion in vitro (Juhel et al., 2000). In this study we compare examples of black, green and white teas for their ability to inhibit pancreatic lipase and attempt to identify the active polyphenolic components.

White and green tea polyphenols inhibit pancreatic lipase in vitro

Green, white and black teas were assayed for inhibition of pancreatic lipase activity in vitro. White tea proved to be more effective than green tea with black tea showing little inhibition even at 200 μg GAE/ml. The EC50 values for inhibition were 22 μg/ml for white tea and 35 μg/ml for green tea; both easily achievable from normal infusions of tea. Liquid chromatography-mass spectrometry analysis showed that white and green teas had essentially equal amounts of flavan-3-ols but green tea had higher levels of flavonols. White tea had higher levels of 5-galloyl quinic acid, digalloyl glucose, trigalloyl glucose and the tannin, strictinin.

After chromatography on Sephadex LH-20, the main inhibitory fraction was enriched in strictinin and fractions enriched in other components were ineffective. This suggests that strictinin content may be crucial for inhibition of pancreatic lipase. However, the possibility of synergies between the polyphenols cannot be disregarded.

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2. Materials and methods

2.1. Tea extraction

Black tea (Tetley) and green tea (Clipper, Green China Tea) were purchased at a local supermarket and hand picked, loose leaf white tea was a gift [from Mr. White, Director of Honeybush & Butterbum Ltd, St Peter’s Gate, Charles St, Sunderland SR6 0AN (www.t-please.com)]. The black and green teas were removed from their bags and weighed. An amount of loose white tea (~2 g) equivalent to the green tea was weighed into flasks. Triplicate tea samples were extracted with 200 ml of boiled water and incubated for 15 min at room temperature stirred with rotary shaking at 100 rpm. After cooling, the tea infusions were filtered through Whatman No. 1 filter paper and the recovery noted. The loose white tea had a larger particle size than the other teas.

Phenol content was measured using a modified Folin–Ciocalteu method (Deighton, Brennan, Finn, & Davies, 2000) and quantified as gallic acid equivalents (GAE). Samples were dried in aliquots of suitable phenol content in a Speed-Vac (Thermo Scientific, Basingstoke, UK).

A portion of each infusion was subjected to solid phase extraction (SPE) procedure used to gather phenol-rich fractions. Briefly, the infusion (50 ml) were applied to C18 solid phase extraction (SPE) units (Strata C18-E, GIGA units, 10 g capacity; Phenomenex Ltd., Macclesfield, UK) pre-washed in 0.1% (v/v) formic acid in acetonitrile then pre-equilibrated in 0.1% (v/v) formic acid in water. The unbound material was discarded. The SPE units were washed with a unit volume of 0.1% (v/v) aqueous formic acid then with two volumes of ultra-pure water. The polyphenol-enriched bound

![Fig. 1. Inhibition of lipase by tea infusions. Assays contained 25 µg GAE for white tea (WT) and green tea (GT) but contained 100 µg GAE for black tea (BT). EC is enzyme control, no inhibitors added. Values are expressed as % control activity and were triplicates ± standard errors.](image1)

A

![A](image2)

B

![B](image3)

![Fig. 2. Inhibition of lipase by green and white tea infusions. The dose response for green tea (A) and white tea (B) on lipase inhibition was assessed. Values are expressed as % control activity and were triplicates ± standard errors. Equations for the slopes assume straight lines relationships.](image4)
extracts eluted with acetonitrile. After measurement of phenol content, aliquots of the C18-bound extracts were evaporated to dryness in a Speed-Vac.

2.2. Chromatography on Sephadex LH-20

This procedure essentially followed previous work (McDougall et al., 2005). White tea infusion (50 ml) was diluted with an equal volume of ethanol and mixed by inversion. A small precipitate was removed by centrifugation (5000 g, 10 min, 5 °C) and a sample of the supernatant was removed for phenol content assessment and LC-MS analysis. The remainder was applied to a Sephadex LH-20 column which had been washed in 50% acetone/water then re-equilibrated in 50% ethanol/water. The unbound material that eluted during the application of the sample was collected then a wash fraction of 20 ml of 50% ethanol/water was applied and collected. The bound material was eluted in 3 × 20 ml fractions of 50% acetone. Each fraction was assayed for phenol content and aliquots of equivalent phenol content were dried for LC-MS and lipase inhibitor assay.

2.3. Lipase assay

This assay was as reported previously (McDougall, Kulkarni, & Stewart, 2009). Lipase from porcine pancreas Type II (Sigma product L3126) was dissolved in ultra-pure water at 10 mg/ml; then the supernatant was used after centrifugation at 16,000 rpm for 5 min. The assay buffer was 100 mM Tris buffer (pH 8.2) and p-nitrophenyl laurate (pNP laurate) was used as the substrate. The substrate stock was 0.08% w/v pNP laurate dissolved in 5 mM sodium acetate (pH 5.0) containing 1% Triton X-100 and was heated in boiling water for 1 min to aid dissolution, mixed well, then cooled to room temperature.

The control assay contained 400 μl assay buffer, 450 μl substrate solution and 150 μl lipase. Tea samples were dissolved in ultra-pure water and added in 50 μl total volume. The buffer, enzyme and tea extracts were added and then substrate was added to start the reaction. The samples were incubated at 37 °C for 2 h. Then samples were centrifuged at 16,000 rpm for 1 min and read at 400 nm in a UV spectrophotometer. All samples were assayed in triplicate and an inhibitor blank was prepared for each sample. The results are expressed as % control activity (McDougall et al., 2009).

2.4. Liquid chromatography–mass spectrometry (LC-MS) analysis

Triplicate samples containing 20 μg phenols (gallic acid equivalents; GAE) were analyzed on an LCQ-Deca system, comprising Surveyor autosampler, pump and photodiode array detector (PDA) and a ThermoFinnigan ion-trap mass spectrometer. The PDA

Fig. 3. LC-MS analysis of white and green tea infusions. Each trace (A280) is from 20 μg GAE. The figures in the top right corner represent the full scale deflection of the detector. Fig 3A is the white tea and (B) is the green tea extract. Peaks annotations match those in Table 1.
scanned discrete channels at 280 nm, 365 nm and 520 nm and recorded spectra from 200–600 nm. The samples were applied to a C18 column (Synergi Hydro C18 with polar endcapping, 4.6 mm × 150 mm, Phenomenex Ltd.) and eluted using a gradient of 5% acetonitrile (0.5% formic acid) to 40% acetonitrile (0.5% formic acid) over 60 min at a rate of 400 µl/min. The LCQ-Deca LC-MS was fitted with an ESI (electrospray ionization) interface and analyzed. The samples were applied to a C18 column (Synergi Hydro C18 with polar endcapping, 150 mm, Phenomenex Ltd.) and eluted using a gradient of 5% acetonitrile (0.5% formic acid) to 40% acetonitrile (0.5% formic acid) over 60 min at a rate of 400 µl/min. The LCQ-Deca LC-MS was fitted with an ESI (electrospray ionization) interface and analyzed.

The relative amounts of polyphenols in white and green tea was estimated by searching for the major m/z value of the compound in question and calculating the peak areas using Xcalibur software. All estimations were carried out on LC-MS runs from three separate infusions. Peak areas were checked against PDA peak areas where possible.

3. Results

The black tea infusions yielded the highest phenol content (217 ± 0.9 mg GAE total) whereas white tea and green tea provided lower amounts (194 ± 1.4 and 176 ± 1.1 mg GAE total, respectively). However, because the infusions were made from the tea bags as supplied, the order was different when expressed as mg/g dry weight with white tea yielding the highest phenol content (97 ± 0.3 mg GAE/g) compared to 89.1 ± 0.5 (green tea) and 67.9 ± 0.3 (black tea). These values are in the same range as previously reported. White tea also contained detectable amounts of the flavan-3-ol derivatives (peak 15) and contained a range of quercetin and kaempferol tetraglucoside (Dou, Lee, Tzen, & Lee, 2008) derivatives (peaks 23–28) whereas white tea had higher levels of one kaempferol hexose rhamnose hexose derivative (peak 16). However, the main components which were higher in the white tea and could explain the higher lipase inhibition were identified as 5-galloyl quinic acid (3.2-fold), digalloyl glucose (7.1-fold), trigalloyl glucose (3.9-fold) and the tannin, strictinin (8.3-fold).

Chromatography on Sephadex LH-20 produced a range of unbound, wash and bound samples (Fig. 6). However, it should be noted that the main flavonol glycosides were not detectable in any sample. The main flavonols were present in green tea in greater concentrations than in white and black tea. The flavonol quercetin was detected in white and black tea with the highest levels in black tea (5.3-fold).

Peak annotations are shown on Figs. 3–6.

### Table 1

<table>
<thead>
<tr>
<th>Peak No.</th>
<th>T&lt;sub&gt;r&lt;/sub&gt;</th>
<th>PDA</th>
<th>M/Z (M–H)</th>
<th>MS&lt;sup&gt;2&lt;/sup&gt;</th>
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<td>1</td>
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<td>343, 191</td>
<td>191, 169</td>
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<td>2</td>
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<tr>
<td>3</td>
<td>15.08</td>
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<td>305</td>
<td>423, 331, 271</td>
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<td>455, 289</td>
<td>903, 885, 301</td>
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<tr>
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<td>563</td>
<td>Theafalvin derivative</td>
<td></td>
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</tbody>
</table>

Peak annotations are shown on Figs. 3–6.

Figures in bold represent the main signal and the one chosen for MS<sup>2</sup> analysis. + denotes that the component ionized most readily in positive mode. *, ** and *** = three peaks containing a [M – H] signal at 483 could be discerned at RT 16.02, 16.94 and 17.57 suggesting the presence of isomers of digalloyl glucose (see Fig. 6). These are discussed in the results concerning fractionation on Sephadex LH-20.

White tea also contained detectable amounts of [M – H] signal at 785 (RT 22.59) which can be assigned to galloylated strictinin MS<sup>3</sup> = 633 [M = 152 = galloyl]. Identifications are supported by references (Del Rio et al., 2004; Dou et al., 2008; Mammea, Savolainen, Lindroos, Kanga, & Vartiainen, 2000).

White and green tea showed substantial inhibition of pancreatic lipase activity when screened at 25 µg GAE/ml. However, black tea showed no inhibition at 100 µg/ml (Fig. 1) and was ineffective at 200 µg/ml (results not shown). White tea was more effective than green tea with an EC<sub>50</sub> value of 22 µg GAE/ml compared to 35 µg GAE/ml for green tea (Fig. 2a and b). Samples enriched in polyphenols obtained by solid phase extraction gave similar EC<sub>50</sub> values of inhibition (results not shown). This strongly suggests that the inhibition was caused by the phenolic components. However, the order in which the components was added did not affect inhibition; i.e. preincubation of the enzymes with the tea samples was not essential for inhibition.
noted that diluting the white tea sample to 50% ethanol caused a marked change in the polyphenol composition (compare Fig. 6A and B); most obvious of which was the reduction in strictinin content (peak 6). We can only assume that some strictinin precipitated along with other components. The unbound fraction contained mainly caffeine (peak 7), theobromine (peak 2) and 5-galloyl quinic acid (peak 1) with smaller amounts of peaks (marked in Fig. 6C), the first of which eluted where strictinin eluted, identified as p-coumaroyl quinic acid derivatives [PDA max = 310; m/z = 337, MS2 191 and 163], which were concentrated in this fraction.

Due to carry-over, the wash fraction (Fig. 6 D) also contained the peaks found in the unbound fraction but was also enriched in flavonol hexose rhamnose hexose derivatives (e.g. peaks 12, 14 and 16).

Bound fraction 1 (Fig. 6E) was enriched in (−)epigallocatechin, (+)catechin and epicatechin (peaks 5, 7a and 8) but also contained considerable amounts of three peaks that appear to be digalloyl glucose isomers (peaks 4 and 4'). The peak marked ** was gallic acid, which was not apparent in the original extract. This fraction also contained some strictinin (peak 6) and a range of flavonols derivatives (peaks 12, 14–20). Myricetin hexose, presumably myricetin-3-O-glucoside (Del Rio et al., 2004) was also detected in this fraction.

Bound fraction 2 was enriched in strictinin (peak 6) with smaller amounts of epicatechin and (−)epigallocatechin (peaks 5 and 8), digalloyl glucose (peak 4) and EGCG (peak 10). This fraction also contained trigalloyl glucose (peak 9, not shown on Fig. 6).

Bound fraction 3 was dominated by the catechin gallates (EGCG, peak 10; ECG, peak 21) with small amounts of methyl ECG (peak 22) but also contained strictinin (peak 6). Bound fraction 3 also contained another flavan-3-ol derivative not detected at first pass in the original white tea samples [at RT 22.95 with PDA max = 275; m/z = 425, 273 and MS2 = 273, 169] which can be putatively identified as (epi)afzelechin gallate (Zeeb, Nelson, Albert, & Dalluge, 2000). This component was also present in the green tea infusions at roughly 3-fold lower levels than white tea but was overshadowed by flavonol derivatives (peaks 23 and 24).

The new white tea sample required 100 µg GAE/ml to give consistent lipase inhibition (Fig. 7) which may have been related to its apparently lower amounts of strictinin. However, the original white tea extract was also not so effective (EC50 48 µg/ml) at this point despite having an effectively identical polyphenol composition by LC-MS (results not shown). This reduction in effectiveness may have been the result of a new batch of lipase, which had higher protein content per unit of lipase activity. Therefore, although the lipase activity per mg weight was not affected, it had 1.5-fold higher protein content.

In any case, if 100 µg GAE/ml was used, it was clear that the bulk of the lipase inhibitory activity was recovered in bound fraction 2 with some activity in bound fraction 3 (Fig. 7).
4. Discussion

The main difference between white and green tea was in the amounts of strictinin and digalloyl glucose derivatives. Fractionation on Sephadex LH-20 suggested that the strictinin content was most important for lipase inhibition. The variation in EC50 values noted between lipase batches was caused by variation in protein content which re-emphasizes the difficulty in comparing EC50 between studies or laboratories. The amelioration of inhibition by higher levels of non-enzymatic protein suggests that strictinin causes inhibition by binding to the lipase directly (Frazier et al., 2010). It is clear that more work to compare inhibition by purified compounds identified in this study or synergistic effects of mixtures of flavan-3-ols and tannins would be useful. However, this was not possible in this study but one could also argue that the value of using artificial mixtures of purified polyphenols when the commercially-available teas with different polyphenolic compositions are effective is debatable.

Previous work on lipase inhibition has focused on flavan-3-ol content and EGCG (Juhel et al., 2000). The lack of inhibition by black tea with its lower flavan-3-ol content generally supports this idea. However, it has been previously noted that strictinin and similar derivatives could inhibit lipase at low levels (Nakai et al., 2005).

Strictinin content and the general polyphenol composition of green tea (and probably white teas, although there is less data) varies with variety of tea, processing and location of growth (Engelhardt, Lakenbrink, & Pokorny, 2004; Komes, Horžič, Belščak, Kovačević, & Vulić, 2010; Lin, Tsai, Tsay, and Lin, 2003). Also different polyphenols are differently influenced by the means of preparing tea beverages (Mizukami et al., 2007; Rusak et al., 2008; Yang, Hwang, & Lin, 2007) and subsequent storage (Friedman, Levein, Choi, Kozukue, & Kozukue, 2006; Mizukami et al., 2007). Indeed, tea flavan-3-ols can undergo epimerization reactions on heating (Sato, Nakamura, Nanjo, and Hara, 1997) which may influence their stability and bioactivity (Ikeda et al., 2003). In this study, we noted that strictinin in white tea preparations was substantially less stable in frozen aqueous solution than when reconstituted from freeze-dried powder or from frozen speed-vac dried samples (results not shown).

It has also become clear that tea polyphenols may have different stabilities under simulated gut conditions (e.g. Record and Lane, 2001) and may form other products during digestion. However, although it is clear that they are present in the gut in sufficient amounts to inhibit lipase (Stalmach et al., 2009), it is not known if they are active in vivo.

Ingestion of green tea has been shown to have blood lipid and cholesterol lowering effects in animal models fed high fat diets (e.g. Ikeda et al., 1992, 2003). Systemic effects on lipid metabolism have been identified (e.g. Hsu & Yen, 2007; Kim et al., 2009) and tea ingestion increases serum antioxidant levels (Serafini, Ghiselli, & Ferro-Luzzi, 1996). However, due to the low serum bioavailability of tea polyphenols (e.g. Henning et al., 2005), it has been sug...
gested that effects on lipid levels in vivo must be “mediated largely via their influence on the intestinal processes involved in digestion and absorption of lipids” (Koo & Noh, 2007). Inhibition of lipase and subsequent slowing of fat digestion could be one of a range of mechanisms whereby tea polyphenols could influence obesity. Work has largely focused on EGCG and related compounds and has suggested that as well as inhibition of lipases, the main effect of these compounds may be to interfere with the emulsification and micellar solubilization of lipids and thereby reduce their overall uptake (Koo & Noh, 2007). However, it seems entirely possible that teas with elevated strictinin levels could increase inhibition of lipase and act synergistically with the flavan-3-ols...
to increase the overall lipid-lowering effect. This synergy may explain the greater lipid-lowering effectiveness of green tea extracts over EGCG alone (e.g. Koo & Noh, 2008). Such beverages could find plen the greater lipid-lowering effectiveness of green tea extracts to increase the overall lipid-lowering effect. This synergy may ex-

**Fig. 7.** Inhibitory activity of LH-20 fractions. Lipase inhibition assays were carried out at 100 μg GAE. Values are expressed as % control activity and were triplicates ±standard errors.

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terbum Ltd, via Dr Ed Okello, University of Newcastle. We also thank the École Nationale Supérieure d’Agronomie et des Indus
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**References**


lipolytic effect of green tea extract in C57BL/6j lept ob/mice. Phytotherapy Research, 23, 467–471.


Koo, S. I., & Noh, S. K. (2008). Green tea extract is more effective than epigallocatechin gallate (EGGC) and epicatechin (EC) in inhibiting the lymphatic absorption of cholesterol (CH) and other lipids. FASEB Journal, 22, 7026–7038.


