Antioxidant, enzyme inhibitory and antiproliferative activity of polyphenolic-rich fraction of commercial dry ginger powder

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Summary Polyphenolic-rich fraction obtained from locally produced dry ginger powder in Brahmaputra valley, India, and commercially available dry ginger (Zingiber officinale) rhizome powder consisted of [6]-gingerol (41.9%), [6]-shogaol (24.3%), 1-dehydro-6-gingerdione (8.6%), [8]-gingerol (7.2%), [10]-gingerol (5.1%), [6]-paradol (5.9%) and [4]-gingerol (3.6%). Traces of methyl-[6]-gingerol and methyl-[8]-gingerol (both at 1.8%) were also detected. The fraction exhibited high antioxidant capacity [total phenolics (TP), ferric reducing antioxidant power (FRAP), oxygen radical absorbance capacity (ORAC) and cellular antioxidant activity (CAA assay)], effectively inhibited isolated digestive enzymes (α-glucosidase, pancreatic lipase and angiotensin converting enzyme) and inhibited the proliferation of colon (HT29; IC50 of 1.06/C6 0.02 mg mL/C0 1) and gastric (AGS IC50 of 1.29/C6 0.03 mg mL/C0 1) adenocarcinoma cells, without affecting the proliferation of their nontransformed counterparts (IC50 > 2.0 mg mL/C0 1). This case study demonstrates that locally produced and commercially available dry ginger powder from Brahmaputra valley, India, retains numerous food components that may enhance human health.

Keywords Antioxidant activity, enzyme inhibition, functional properties, polyphenols.

Introduction

Ginger (Zingiber officinale Roscoe, Zingiberaceae) is an important culinary spice and a medicinal plant used by the traditional Chinese, Ayurvedic and Unani herbalists since ancient times.

At present, ginger is utilised as herbal remedy against various conditions, including arthritis, rheumatism, sprains, muscular aches, pains, sore throats, cramps, constipation, indigestion, vomiting, hypertension, dementia, fever and infectious diseases (Ali et al., 2008). Research over the last decade has shown that ginger has the potential to be used in the prevention and treatment of a myriad of diseases through modulation of biological activities (Rahmani et al., 2014). The main pharmacological actions of ginger phytochemicals include immuno-modulatory, antitumorigenic, anti-inflammatory, anti-apoptotic, antihyperglycaemic, antilipidemic and antifungal activities (Kim et al., 2005; Wu, 2007; Ali et al., 2008; Hsiang et al., 2013; Ojaghian et al., 2014). Ginger rhizome contains approximately 1.0–2.5% pungent constituents (nonvolatile homologous polyphenols) that give this spice its pungent or hot flavour (Zick et al., 2008). The main classes of these pungent compounds are gingerols, paradols, zingerones and shogaols (Chrubasik et al., 2005). Antioxidant activity of ginger extract can be equal to that of synthetic antioxidants (Brewer, 2011), while the gingerol-related compounds and diarylheptanoids from ginger rhizomes exhibit higher antioxidant activity than α-tocopherol (Kikuzaki & Nakatani, 2006). Fresh and dried ginger contains also relatively large amounts of volatile oils, including camphene, p-cineole, α-terpineol, zingiberene and pentadecanoic acid, which contribute towards the flavour and therapeutic properties (Bartley & Jacobs, 2000).

The yellow rhizome (underground stem) of ginger is consumed worldwide in three forms: as a fresh vegetable, preserved and dry ginger spice. The most accessible and commercially available form of ginger is dry ginger powder, and the demand for this spice at the international market increases. Bartley & Jacobs (2000) reported that drying of mature ginger rhizomes may alter the composition and/or reduce the levels of pungent constituents in the final product; therefore, it is important that composition of commercially available dried ginger powder is examined.
The major world producer of commercially available ginger is India, followed by China and Nepal (FAO, 2014). One of the ginger producing centres in India is the Brahmaputra valley region. This region, situated between hill ranges of the eastern and north-eastern Himalayan range, is characterised by rainforest-like climate and has some of the most productive soils in the world, regularly enriched by deposits of fresh alluvium brought by the Brahmaputra River. To facilitate continuous production of a high-quality dry ginger powder for the local and international markets in the Brahmaputra valley region, the Tribal Development Foundation (TDF; nongovernmental organization (NGO) in Arunachal Pradesh, India) assisted the locals in setting up a ginger processing plant where ginger rhizome is mechanically dried and ground under strict quality control. The objective of the present study was to characterise phytochemical composition and retention of health-enhancing properties of this commercially produced spice. The sample was evaluated with regard to antioxidant capacity, composition of phenolic compounds and inhibitory activity towards selected digestive enzymes linked to obesity and hypertension. Finally, the antiproliferative properties against colon adenocarcinoma (HT-29) and gastric adenocarcinoma (AGS) cells and their nontransformed counterparts were evaluated.

Materials and methods

Plant material

The dried ginger powder, contributed by Dr G. Srzednicki, was obtained from the Tribal Development Foundation (TDF), Arunachal Pradesh, India. The rhizomes were produced in 2011 in the area near Paisinghat in the Brahmaputra valley. The authenticity of plant material was confirmed by the TDF. The dry ginger powder was produced in a commercial ginger processing plant equipped with electric dryers allowing close control over the drying process and a consistent product quality. The dried ginger rhizomes were ground in the same processing plant to produce a dry ginger powder spice for commercial purposes. A randomly selected sample (500 g) of dry ginger powder obtained from TDF was delivered to the New South Wales University, Sydney, Australia, for evaluation. On arrival to the laboratory, the sample was stored at −20 °C for maximum 2 months before analysis.

Chemicals

AAPH [2,2-azobis (2-methylpropionamide) dihydrochloride], chlorogenic acid, fluorescein, gallic acid, (+-) catechin hydrate, myricetin, rutin, quercetin dihydrate, trolox, Amberlite XAD-16 polymeric resins, acetonitrile, ethanol, methanol, trifluoroacetic acid (TFA), aluminium chloride hexahydrate (AlCl3•6H2O), NaOH, porcine pancreatic lipase, dimethyl sulfoxide (DMSO) and 4-methylumbelliferyl oleate were purchased from Sigma-Aldrich, Inc. (Sydney, NSW, Australia). Folin–Ciocalteu reagent and TPTZ (2,4,6-Tri(2-pyridyl)-s-triazine) were obtained from Merck (Sydney, NSW, Australia). Orlistat and captopril were purchased from Sigma–Aldrich, and acarbose was purchased as ‘glucobay’ from Bayer (Bayer Australia Ltd., Pymble, NSW, Australia). 3-(4,5-Dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT), penicillin and streptomycin were purchased from Invitrogen (Melbourne, Vic., Australia).

Cell lines

All cell lines were purchased from the American Type Culture Collection (ATCC, Rockville, MD, USA) and cultured at 37 °C in a humidified 5% carbon dioxide (CO2) atmosphere in media containing 10% foetal bovine serum (FBS; Invitrogen), 100 μg mL−1 streptomycin and 100 units mL−1 penicillin (Invitrogen) unless otherwise stated. AGS (gastric adenocarcinoma) was cultured in F12-K Ham’s medium (Invitrogen). CCD-18Co (colon nontransformed) and HepG2 (hepatocellular carcinoma) were cultured in Eagle’s minimum essential medium (EMEM; Sigma-Aldrich); Hs 738.St/Int (mixed stomach and intestine, nontransformed) was cultured in Dulbecco’s modified Eagle’s medium (DMEM; Invitrogen); HT-29 (colorectal adenocarcinoma) was cultured in McCoy’s 5a medium (Invitrogen). Experiments were conducted in cell lines with <40 passages.

Preparation of hydrophilic fraction from commercial sample of dry ginger powder

One hundred grams of ginger powder was steeped in a fivefold volume of acidified methanol (80% methanol, 19% H2O and 1% acetic acid, v/v/v/v), stirred for 2 h at low temperature (4 °C) and centrifuged for 20 min at 15320 × g at 4 °C (Sorvall RC-5B; DuPont, Wilmington, DE, USA). The supernatant was filtered, and the residue was extracted twice. The third extraction was carried out overnight (16 h). The supernatants from the consecutive extractions were combined, and the solvent evaporated under reduced pressure at 40 °C using a rotary evaporator (Rotavapor R-205; Buchi, Switzerland). The concentrated alcoholic extract was chromatographed using an XAD-16 open column (300 × 60 mm i.d.). The extracts were dissolved in acidified water (99% H2O, 1% acetic acid; v/v), applied to the column, washed with acidified water and eluted with 80% ethanol (80% ethanol, 19.9% H2O, 0.1% trifluoroacetic acid, v/v/v). The eluted material was collected and evaporated under reduced pressure at 37 °C using a rotary evaporator. The purification was repeated, and
the resulting fraction was dissolved in purified water and freeze-dried under vacuum to obtain a fine hydrophilic fraction. The extraction yield was calculated as a percentage of the original raw plant material.

**Antioxidant testing**

The antioxidant capacity was evaluated using the total phenolic (TP; Folin-Ciocalteu method) and the ferric reducing antioxidant power (FRAP) assays to estimate the total reducing capacities as well as the oxygen radical absorbance capacity (ORAC) assay to estimate the oxygen radicals scavenging capacities, as previously described (Konczak et al., 2010).

**Cellular antioxidant activity (CAA)**

Cellular antioxidant activity was conducted according to Sakulnarmrat et al. (2013). The results were expressed as quercetin equivalent per gram of dry weight (mg QE/g DW) of purified polyphenolic-rich lyophilized fractions and IC50 (the amount of lyophilized fraction necessary to reduce the oxidative stress in 50%).

**Analysis of phenolic compound by liquid chromatography–photo-diode array–mass spectrometry (LC-PDA-MS/MS)**

LC-PDA-MS/MS analysis was conducted on a Quantum triple stage quadrupole (TSQ) mass spectrometer (Thermo Fisher Scientific), equipped with a quaternary solvent delivery system, a column oven, a photo-diode array detector, and an autosampler. An aliquot (3 μL) of ethanol extract was chromatographed on a 150 × 2.1 mm i.d. μm Luna Synergy Hydro column (Phenomenex) which was heated to 30 °C. Analytes were separated using 0.5% formic acid in purified water (A) and 0.5% formic acid in acetonitrile (B) at a flow rate of 200 μL min⁻¹. The gradient employed was 0% B for 2 min, followed by an increase to 100% B over 18 min and hold for 1 min. The photodiode array detector was used to acquire data from 190 to 520 nm. Ions for mass spectrometry were generated using an electrospray source in the positive mode with an electrospray negative control, and where A/S and A/C were the absorbance of sample and control blanks.

**Lipase inhibition assay**

The lipase inhibitory activity was assayed as described by Shimura et al. (1992) using 4-methylumbelliferyl oleate as substrate, except the porcine pancreatic lipase (Sigma type II) was prepared using a concentration of 0.085 g mL⁻¹. The relative lipase inhibition activity was calculated using the following formula:

\[
%\text{Inhibition} = \frac{(A_{SB} - A_{C}) - (A_{SB} - A_{S})}{(A_{CB} - A_{C})} \times 100
\]

where \(A_{S}\) and \(A_{C}\) were the absorbance of sample and negative control, and where \(A_{SB}\) and \(A_{CB}\) were the absorbance of sample and control blanks.

**Angiotensin converting enzyme (ACE) inhibition assay**

The angiotensin converting enzyme (ACE) inhibition assay was carried out as described by Shalaby et al. (2006) using furanacroloyl-Phe-Glu-Glu (FA-PGG) as a substrate. The ACE enzyme solution from rabbit lung (Sigma-Aldrich, Castle Hill, NSW Australia) (0.25 unit mL⁻¹) was freshly prepared everyday by adding 1 mL of 50 mM tris-HCl buffer containing 0.3 mM sodium chloride (pH 7.5) into a vial containing 0.25 unit of ACE enzyme. A sample (10 μL) of lyophi-
lised extract dissolved in tris-HCl buffer (1.0 mg mL$^{-1}$) or buffer (control) and ACE solution (10 μL) were added to the wells of 96-well microplate at room temperature. ACE inhibition was evaluated using extracts at concentration of 1.0 mg mL$^{-1}$. The microplate was transferred to the incubator of a spectrophotometer (POLARStar Omega; BMG Labtech, Ortenberg, Germany), and 150 μL of the FA-PGG solution in tris-HCl buffer (0.1 m) was injected into the wells through the liquid handling system. The mixture was mixed thoroughly for 5 s at 500 rpm, and the absorbance was recorded every 30 s for 30 min at 340 nm. The slope of curve obtained from the absorbance vs. time was the indicator of enzyme activity. The ACE activity was determined using the following formula:

$$\%\text{ACE inhibition} =\frac{1 - (rA_{\text{inhibitor}}/rA_{\text{control}}) \times 100}{1}$$

where $rA_{\text{inhibitor}}$ was the slope of decrease in absorbance at 340 nm in the presence of inhibitor and $rA_{\text{control}}$ was the slope of decrease in absorbance at 340 nm in the absence of inhibitor. A captopril (Sigma Chemicals) standard curve (final concentration in tris-HCl buffer of 0.04–0.40 μM) was prepared, and the results were expressed as μg of captopril equivalents per gram dry weight of extract.

### Antiproliferative activities

The colorimetric MTT (3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (Invitrogen Pty Ltd, Mulgrave, Australia) assay was used to assess the sensitivity of AGS, HT-29, CCD-18Co, Hs 738.St/Int, BL-13 and HepG2 cells to ginger extract as previously described (Sakulnarmrat et al., 2013).

### Statistical analysis

The mean of results was calculated based on at least three independent evaluations ($n = 3$), and the standard deviations (SD) were also calculated. All IC$_{50}$ values were calculated from the corresponding dose inhibition curve according to their best fit shapes based on at least four reaction points using Microsoft Excel. Statistical correlation analyses were performed using GraphPad Prism 5 (GraphPad Software, La Jolla, CA, USA), which were considered statistically significant when the $P < 0.05$.

### Results and discussion

#### Yield and antioxidant capacity of hydrophilic fraction obtained from commercial sample of dry ginger powder

Purification of an alcoholic extract obtained from the commercially available dry ginger rhizome powder, using an open column chromatography (XAD-16 resins), allowed to produce a fine lyophilised fraction comprising hydrophilic compounds. The same hydrophilic compounds would be released from foods during the digestion process. The recovery rate accounted for 2.72% of the original sample dry mass (Table 1) and was higher than the recovery yield of a similar ethanol-based fraction obtained from dry Chinese ginger (1.7%) (Hsiang et al., 2013), but lower than that reported by Da Silva et al. (2012) (5.3%).

The antioxidant capacities of the isolated ginger fraction were characterised using reagent-based and cellular assays (Table 1). Cellular antioxidant activity (CAA) of the lyophilised polyphenolic ginger fraction was assayed at concentrations from 0.2 to 0.6 mg mL$^{-1}$, which did not affect cell proliferation (data not presented). This assay allows evaluating the activity of an antioxidant within a living cell, and the efficacy of antioxidant depends on the successful uptake, distribution and metabolism of the compounds within a cellular system (Wolfe & Liu, 2007). The CAA of ginger fraction (IC$_{50}$ of 1.02 of mg mL$^{-1}$, or 29.1 ± 1.7 μmol QE/g DW; Table 1) was significantly higher than the CAA of crude common vegetable extracts, including beetroot (19.3 ± 4.1 mg mL$^{-1}$), red pepper (19.2 ± 0.9 mg mL$^{-1}$), eggplant (21.0 ± 1.3 mg mL$^{-1}$), Brussels sprout (22.7 ± 2.3 mg mL$^{-1}$) and broccoli (26.3 ± 2.7 mg mL$^{-1}$) (Song et al., 2010), but was inferior to a similar fraction obtained from bay leaf (Sakulnarmrat et al., 2013). This assay shows that phytochemicals present in commercially produced ginger spice have entered a life cell and exerted antioxidant activity within a living cell.

#### Phytochemical composition of ginger fraction

The liquid chromatography mass spectrometry analysis of the hydrophilic fraction of dry ginger powder evaluated in this study revealed the presence of nine major phenolic compounds detected at 280 nm wavelength (Supporting information, Fig. S1). Among them are seven gingerols, which comprised approximately 70% of the mixture, with

| Table 1 Total phenolic content and antioxidant capacity of polyphenolic-rich fraction obtained from commercially produced dry ginger powder |
|---|---|---|---|---|---|
| Yield (%) | TP *GAE/g DW | FRAP μmol Fe$^{2+}$/g DW | ORAC μmol TE/g DW | CAA μmol IC$_{50}$/g DW; μmol QE/g DW |
| 2.72 | 308 ± 25 | 5474 ± 230 | 6994 ± 130 | 1.02 ± 0.2 | 29.1 ± 1.7 |

*GA E/gDW: gallic acid equivalent per gram dry weight lyophilised fraction; μmol Fe$^{2+}$/g DW: micromole ferric ion per gram of dry weight lyophilised fraction; μmol T E/g DW: micromole trolox equivalent per gram of dry weight lyophilised fraction; μmol IC$_{50}$/g DW: half maximal effective concentration; μmol QE/g DW: micromole quercetin equivalent per gram of dry weight lyophilised fraction.
[6]-gingerol accounting for 41.9%, followed by 1-dehydro-6-gingerdione (8.6%), [8]-gingerol (7.2%), [10]-gingerol (5.1%), [4]-gingerol (3.6%), methyl [6]-gingerol and methyl [8]-gingerol, both present at the level of 1.8% (Table 2). Shogaols are the dehydrated form of gingerols, present at very low levels in fresh rhizome and mainly found in dried and thermally treated rhizomes, with 6-shogaol being the most abundant (Jolad et al., 2004). In agreement, 6-shogaol was identified in the evaluated fraction, and constituted 24.3% of the polyphenolics mixture. Paradols are generated by hydrogenation of shogaol (Du et al., 2006); [6]-paradol constituted 5.9% of the polyphenolic mixture evaluated in this study. In agreement with our findings, Wu (2007) identified eight nonvolatile pungent phenolic compounds in a crude fresh ginger extract, including 6-gingerol (5.37%), 8-gingerol, 10-gingerol (1.84%), 6-shogaol (6.73%), 8-shogaol, 10-shogaol (1.05%), 6-paradol and 1-dehydro-6-gingerdione. The same author reported a high DPPH radical scavenging ability of isolated 6-shogaol and 6-gingerol.

\[ \text{\textbf{Table 2}} \text{ Major compounds of polyphenolic-rich fraction obtained from commercially produced dry ginger powder} \]

<table>
<thead>
<tr>
<th>Peak</th>
<th>( t_R ) (min)</th>
<th>([M+H]^+)</th>
<th>Major fragments ( \text{m/z} )</th>
<th>Relative concentration (%)</th>
<th>Tentative identification</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>11.20</td>
<td>249</td>
<td>177</td>
<td>3.6 ± 0.1</td>
<td>[4]-gingerol</td>
</tr>
<tr>
<td>2</td>
<td>13.52</td>
<td>249</td>
<td>177</td>
<td>5.9 ± 0.2</td>
<td>[6]-paradol</td>
</tr>
<tr>
<td>3</td>
<td>13.97</td>
<td>277</td>
<td>177</td>
<td>41.9 ± 0.4</td>
<td>[6]-gingerol</td>
</tr>
<tr>
<td>4</td>
<td>15.29</td>
<td>291</td>
<td>191</td>
<td>1.8 ± 0.1</td>
<td>Methyl[6]-gingerol</td>
</tr>
<tr>
<td>5</td>
<td>16.39</td>
<td>305</td>
<td>177</td>
<td>7.2 ± 0.1</td>
<td>[8]-gingerol</td>
</tr>
<tr>
<td>6</td>
<td>17.02</td>
<td>277</td>
<td>137</td>
<td>24.3 ± 0.2</td>
<td>[8]-shogaol</td>
</tr>
<tr>
<td>7</td>
<td>17.74</td>
<td>319</td>
<td>191</td>
<td>1.81 ± 0.0</td>
<td>Methyl[8]-gingerol</td>
</tr>
<tr>
<td>8</td>
<td>18.10</td>
<td>291</td>
<td>177</td>
<td>8.6 ± 0.1</td>
<td>1-dehydro-6-gingerdione</td>
</tr>
<tr>
<td>9</td>
<td>19.34</td>
<td>333</td>
<td>177, 137</td>
<td>5.1 ± 0.2</td>
<td>[10]-gingerol</td>
</tr>
</tbody>
</table>

Relative concentration calculated based on peak area at 280 nm (%).

\( \text{\textbf{Table 3}} \text{ Inhibitory activity of the polyphenolic-rich fraction obtained from bay leaf and comparable inhibitory activity of a polyphenolic-rich fraction obtained from native Australian herb, Tasmannia pepper leaf} \)

\( \text{IC}_{50} = 0.83 \pm 0.36 \text{ mg mL}^{-1} \) evaluated in the same experimental setting (Sakulnarmrat et al., 2013). A different potency was observed with regard to pancreatic lipase, with a high IC\(_{50}\) value of 6.16 ± 0.1 mg mL\(^{-1}\). In agreement, in vivo studies by Mahmoud & Elnour (2013) showed that incorporation of ginger powder (5%) to a high-fat diet of rats over 4 weeks did not alter the activity of pancreatic lipase, although it reduced body weight and lipid profile and had a greater effect in increasing HDL cholesterol than orlistat (tetrahydrolipstatin), a drug used to treat obesity.

The inhibitory activity of the polyphenolic-rich ginger fraction obtained in this study towards ACE compared favourably to that of a bay leaf extract (13.9 ba.8%; 1 mg mL\(^{-1}\) concentration) (Sakulnarmrat & Konczak, 2012). In a similar in vitro study, Akinyemi et al. (2013) demonstrated inhibition of ACE by aqueous extracts (0.05 mg mL\(^{-1}\)) of red ginger (Zingiber officinalis L.) and white ginger (Zingiber officinale Roscoe). The red ginger extract had a significantly higher inhibitory effect on ACE (IC\(_{50} = 27.5; \ P < 0.05\)) than the white ginger extract (IC\(_{50} = 87.0 \text{ mg mL}^{-1}\)). However, subsequent supplementation of rat’s diet with fresh red and white ginger (either 2% or 4%) over a period of 3 days showed a higher ACE-inhibitory activity of the white ginger. The ACE-inhibitory activity occurred concomitantly with antihypercholesterolemic action visible in significant decrease of plasma total cholesterol, triglyceride, very low-density lipoprotein cholesterol and low-density lipoprotein cholesterol levels (Akinyemi et al., 2014). The results of the present study are in agreement with those of Akinyemi and collaborators and suggest that commer-
commercially produced dry ginger powder retained its ACE-inhibitory properties.

**Antiproliferative activities**

During the digestion process compounds released from foods interact with the surrounding tissue. Therefore, within this study the effect of the hydrophilic ginger fraction on the proliferation of two cancer cell lines relevant to the digestive tract: colon adenocarcinoma (HT-29) and stomach adenocarcinoma (AGS), as well as their equivalent nontransformed cell lines: colon (CCD-18Co) and stomach/intestine mix (HS738.St/Int), was evaluated. The cells were exposed to polyphenolic-rich fraction applied at the concentrations of 0.0–2.0 mg mL\(^{-1}\). and their proliferation rates were monitored. The addition of ginger fraction to culture media suppressed the proliferation of cancer cells in a dose-dependent manner (Supporting information, Fig. S2). No significant differences were observed between the HT-29 (IC\(_{50}\) of 1.06 ± 0.02 mg mL\(^{-1}\)) and AGS (IC\(_{50}\) of 1.29 ± 0.03 mg mL\(^{-1}\)) cell lines. The ginger fraction did not affect the proliferation of nontransformed cell lines (CCD-18Co and HS 738.St/Int; IC\(_{50}\) > 2.0 mg mL\(^{-1}\)), which suggest potential chemopreventive properties of hydrophilic compounds present in commercially produced dry ginger powder. A number of cell-cultural-based studies support this observation. Da Silva *et al.* (2012) reported suppression of the proliferation of human breast tumour (MDA-MB-231) cell line by [6]-, [8]- and [10]-gingerol, with the highest efficiency exhibited [10]-gingerol (12.1 ± 0.3 μM; MW 350.49), followed by [8]-gingerol and [6]-gingerol (IC\(_{50}\) of 666.2 ± 134.6 μM; MW 294.39). These compounds applied at concentrations higher than 500 μM also inhibited the proliferation of human fibroblasts (HF) (Da Silva *et al.*, 2012), while [6]-gingerol and [6]-paradol inhibited the proliferation of human endothelial cells and mouse skin carcinogenesis and exhibited anti-inflammatory activities (Keum *et al.*, 2002; Kim *et al.*, 2005). Shieh *et al.* (2010) have established that isolated [8]-shogaol suppressed the proliferation of human promyelocytic leukaemia cells (HL-60), induced apoptosis through activation of procaspase-9 and -3, rapid loss of mitochondrial transmembrane potential, stimulation of ROS production and release of mitochondrial cytochrome c into cytosol. Aqueous ginger extract induced apoptosis of adenocarcinomic human alveolar basal epithelial (A549) cells by upregulation of the tumour suppressor gene p53, altered Bax/Bcl-2 ratio and downregulation of pro-caspase-3 (Choudhury *et al.*, 2010), while isolated shogaol induced apoptosis of human colorectal carcinoma cells via reactive oxygen species (Pan *et al.*, 2008). This study has shown that commercially produced and available on market dry ginger powder retains its chemopreventive properties and suppresses the proliferation of cancers associated with the digestive system.

**Conclusions**

This case study aimed at evaluation of health-enhancing properties of a commercially produced dry ginger powder from north-eastern India available on local market. Hydrophilic polyphenolic-rich fraction obtained from the dry ginger powder comprised of nine gingerols, with 6-gingerol and 6-shogaol as the dominating compounds contributing over 65% of the mixture. The fraction exhibited pronounced antioxidant capacities in vitro, and retained and manifested antioxidant activity within a living cell. The polyphenolic-rich ginger fraction inhibited the activities of isolated α-glucosidase and angiotensin converting enzyme and was less effective against isolated lipase. The fraction suppressed the proliferation of colon and gastric adenocarcinomas, without inhibition of proliferation of their nontransformed counterparts.

**References**


