Review

Cancer preventive properties of ginger: A brief review

Yogeshwer Shukla *, Madhulika Singh

Environmental Carcinogenesis Division, Industrial Toxicology Research Centre, P.O. Box 80, M.G. Marg, Lucknow 226 001, Uttar Pradesh, India

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Abstract

Ginger, the rhizome of Zingiber officinalis, one of the most widely used species of the ginger family, is a common condiment for various foods and beverages. Ginger has a long history of medicinal use dating back 2500 years. Ginger has been traditionally used from time immemorial for varied human ailments in different parts of the globe, to aid digestion and treat stomach upset, diarrhoea, and nausea. Some pungent constituents present in ginger and other zingiberaceous plants have potent antioxidant and anti-inflammatory activities, and some of them exhibit cancer preventive activity in experimental carcinogenesis. The anticancer properties of ginger are attributed to the presence of certain pungent valloinoids, viz. [6]-gingerol and [6]-paradol, as well as some other constituents like shogaols, zingerone etc. A number of mechanisms that may be involved in the chemopreventive effects of ginger and its components have been reported from the laboratory studies in a wide range of experimental models.

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1. Introduction

A number of biologically active phytochemicals have been identified in plant foods (Lampe, 1999). Consuming a diet rich in plant foods can provide a milieu of phytochemicals and non-nutritive plant substances that possess health-protective effects. Natural dietary agents including fruits, vegetables, and spices have drawn a great deal of attention from both the scientific community and the general public due to their various health promoting effects including suppression of cancers. The questions that remain to be answered are which components of these dietary agents are responsible for the chemopreventive and chemotherapeutic effects and what is the mechanism by which they suppress cancer? Dietary agents consist of a wide variety of biologically active compounds that are ubiquitous in plants and many of them have been used as traditional medicines for thousands of years (Aggarwal and Shishodia, 2006). As early as 2500 years ago,
Hippocrates recognized and professed the importance of various foods both natural and those derived from human skill in the primary constitution of the person. Some phytochemicals derived in spices and herbs as well as other plants possess substantial cancer preventive properties (Lai and Roy, 2004; Surh, 2002, 2003).

Ginger (Zingiber officinale Rosc.), belonging to a tropical and sub-tropical family – Zingiberaceae, originating in South-East Asia and introduced to many parts of the globe, has been cultivated for thousands of years as a spice and for medicinal purposes (Park and Pizzuto, 2002). The ginger plant has a perennial, tuberous root or rhizome; the stems are erect, oblique, round, annual, and invested by the smooth sheaths of the leaves, 2 or 3 feet in height. Ginger rhizome is typically consumed as a fresh paste, dried powder, slices preserved in syrup, candy (crystallized ginger) or for flavoring tea. In many countries, especially in India and China, fresh ginger is used to prepare vegetable and meat dishes and as a flavoring agent in beverages and many other food preparations. The underground stem or rhizome of this plant has been used as a medicine in Asian, Indian, and Arabic herbal traditions since ancient times (Altman and Marcussen, 2001). It has been used extensively for more than 2500 years in China for headache, nausea and colds (Grant and Lutz, 2000) and in Mediterranean (Sharma and Clark, 1998) and Western parts in herbal medicine practice for the treatment of arthritis, rheumatological conditions and muscular discomfort (Bordia et al., 1997; Langner et al., 1998). Its use in inflammatory conditions is consistent with anti-inflammatory activities of its components in vitro (Kiuchi et al., 1982; Mascolo et al., 1989). It has also been suggested for the treatment of various other conditions, including atherosclerosis, migraine headaches, rheumatoid arthritis, high cholesterol, ulcers, depression, and impotence (Liang, 1992). In addition to these medicinal uses, ginger continues to be valued around the world as an important cooking spice and is believed to help the common cold, flu-like symptoms, and even painful menstrual periods (Awang, 1992). Due to these properties, it has gained considerable attention as a botanical dietary supplement in the USA and Europe in recent years, and especially for its use in the treatment of chronic inflammatory conditions (Srivastava and Mustafa, 1992; Kiuchi et al., 1992; Srivastava, 1984; Tjendraputra et al., 2001; Park and Pizzuto, 2002; Aggarwal and Shishodia, 2004). Several population-based studies shows that people in South East Asian countries have a much lower risk of colon, gastrointestinal, prostate, breast, and other cancers than their Western counterparts (Dorai and Aggarwal, 2004), and it is thought that constituents of their diet may play significant role in protection. Indeed, phenolic substances present in fruit and vegetables, and in medicinal plants, have cancer chemopreventive activities, both in vitro as well as in vivo animal models (Surh, 1999; Mahmoud et al., 2000; Kim et al., 2004; Murakami et al., 2004). These agents are believed to suppress the transformative, hyperproliferative, and inflammatory processes that initiate carcinogenesis, as well as the later steps of carcinogenesis, namely angiogenesis and metastasis.

Some phenolic substances present in ginger, generally, possess strong anti-inflammatory and anti-oxidative properties and exert substantial anti-carcinogenic and anti-mutagenic activities (Surh, 2002; Surh et al., 1998, 1999). The aim of this review is to provide a critical insight on the cancer preventive potential of ginger, covering its basic chemistry and biochemical activity, epidemiological investigations, laboratory studies, as well as possible directions for future research.

1.1. Chemistry of ginger

The sensory perception of ginger in the mouth and the nose arises from two distinct groups of chemicals:

**Volatile oils:** The volatile oil components in ginger consist mainly of sesquiterpene hydrocarbons, predominantly zingiberene (35%), curcumene (18%) and farnesene (10%), with lesser amounts of bisabolene and b-sesquiphellandrene. A smaller percentage of at least 40 different monoterpenoid hydrocarbons are present with 1,8-cineole, linalool, borneol, neral, and geraniol being the most abundant (Govindarajan, 1982). Many of these volatile oil constituents contribute to the distinct aroma and taste of ginger.

**Non-volatile pungent compounds:** This species contains biologically active constituents including the non-volatile pungent principles, such as the gingerols, shogaols, paradols and zingerone that produce a "hot" sensation in the mouth. The gingerols, a series of chemical homologs differentiated by the length of their unbranched alkyl chains, were identified as the major active components in the fresh rhizome (Govindarajan, 1982). In addition, the shogaols, another homologous series and the dehydrated form of the gingerols, are the predominant pungent constituents in dried ginger (Connell and Sutherland, 1969). Paradol is similar to gingerol and is formed on hydrogenation of shogoal. The major constituents of ginger are shown in Fig. 1.

**Other constituents:** In addition to the extractable oleoresins, ginger contains many fats, waxes, carbohydrates, vitamins and minerals. Ginger rhizomes also contain a potent proteolytic enzyme called zingibain.

1.2. In vitro cancer chemopreventive effects

In a study, Unnikrishnan and Kuttan (1988) found that alcoholic extracts of the ginger were more cytotoxic to Dalton’s lymphoma ascites tumor cells and human lymphocytes in vitro and Chinese hamster ovary cells and Vero cells in tissue culture than aqueous extracts. It was observed by the researchers that alcoholic extracts inhibited cell growth at concentrations of 0.2–1 mg/ml in vitro and 0.12–0.3 mg/ml in tissue culture as well as inhibited thymidine uptake into DNA. *Helicobacter pylori* is the
primary etiological agent associated with peptic ulcer disease and the development of gastric and colon cancer. The anti-\textit{H. pylori} effects of ginger and its constituents were tested \textit{in vitro} by Mahady et al. (2003). It was found that gingerol inhibited the growth of \textit{H. pylori} CagA+ strains \textit{in vitro} and this activity may contribute to its chemopreventive effects against colon cancer. The results of a study done by Kim et al. (2005a) demonstrated that [6]-gingerol inhibited angiogenesis of human endothelial cells and caused cell cycle arrest in the G1 phase through the down-regulation of cyclin D1. 6-Gingerol inhibited nitric oxide synthase expression in LPS-treated cell lines (Ippoushi et al., 2003) as well as the EGF-induced cell transformation and AP-1 activation in JB6 cells (Bode et al., 2001). The inhibition of the AP-1 transcriptional complex by [6]-gingerol, in human skin keratinocytes cell lines was also reported by Davies et al. (2005).

Some compounds present in ginger may exert cancer preventive effects by inducing apoptosis in cancerous or transformed cells. The oleoresin from the root of ginger contains [6]-gingerol, the major pharmacologically active component and lesser amounts of a structurally related vanilloid, [6]-paradol. Two studies suggest that these compounds suppress proliferation of human cancer cells through the induction of apoptosis (Lee and Surh, 1998; Lee et al., 1998) and were found to exert inhibitory effects on the viability of human HL-60 (promyelocytic leukemia) cells (Lee and Surh, 1998). Results of the study show that the cell viability was decreased by 35% and 89% after 5 h of treatment with 20 \textmu M and 100 \textmu M [6]-paradol, respectively (Lee and Surh, 1998). In another study, it was observed that at low concentrations (up to 25 \textmu M), [6]-paradol induced apoptosis in JB6 cells, but concentrations of 50 \textmu M or greater resulted in apparent necrotic cell death (Huang et al., 1996). Bode and his co-workers (2001), reported that [6]-paradol exerts its primary inhibitory effect on cell transformation through the induction of apoptosis. [6]-Paradol and other structurally related derivatives like [10]-paradol, [3]-dehydroparadol, [6]-dehydroparadol, and [10]-dehydroparadol, induced apoptosis in an oral squamous carcinoma cell line, in a dose-dependent manner through a caspase-3-dependent mechanism (Keum et al., 2005).
2002). It was also noted that [10]-paradol and [10]-dehydroparadol exhibited a similar extent of cytotoxicity to that of [6]-paradol. Exposure of Jurkat human T cell leukemia cells to various ginger constituents galanals A and B (isolated from the flower buds of Japanese ginger) resulted in apoptosis mediated through the mitochondrial pathway (Miyoshi et al., 2003). The authors concluded that apoptosis is accompanied by a down-regulation of antiapoptotic protein Bcl-2 and an enhancement of proapoptotic protein Bax expression. Effect of [6]-gingerol or [6]-paradol on cell proliferation was also assessed by determining DNA synthesis using a radioisotope-labeled precursor. When judged by nuclear incorporation of [3H]thymidine, the inhibitory effect of [6]-paradol on DNA synthesis was found to be more effective than that of [6]-gingerol at all concentrations (Lee and Surh, 1998). It was observed by the researchers that after 5 h incubation with 10 and 50 µM of [6]-gingerol, incorporation of [3H]thymidine was suppressed by 44% and 86%, respectively, while the same concentrations of [6]-paradol reduced the DNA synthesis by 74% and 99%, respectively. The rhizome of ginger has been reported to possess antitumor promotional potential as determined by abrogation of activation induced by a phorbol-ester promoter, 12-O-hexadecanoylphorbol-13-acetate (HPA) induced Epstein–Barr virus (EBV) activation in Raji cells in short term in vitro assay (Koshimizu et al., 1988). In another short term in vitro assay involving Epstein–Barr virus early antigen (EBV-EA) activation in Raji cells promoted by phorbol ester, 12-O-tetradecanoylphorbol-13-acetate (TPA) and ginger extract exhibited potent anti-EBV-EA activity (Vimala et al., 1999; Kapadia et al., 2002). The rhizome extract, that exhibited EBV activation inhibitory activity, had no cytotoxic effect in Raji cells (Vimala et al., 1999). β-Elemene is a novel anticancer drug, which is extracted from the ginger plant. It triggers apoptosis in non-small-cell lung cancer cells through a mitochondrial release of the cytochrome c-mediated apoptotic pathway. Wang et al. (2005) showed that β-elemene induced caspase-3, -7 and -9 activities, decreased Bcl-2 expression, caused cytochrome c release and increased the levels of cleaved caspase-9 and poly (ADP-ribose) polymerase in cells. Certain bioactive components of ginger rhizomes have been characterized by spectroscopic analysis such as zingerone and dehydrozingerone, which also exhibit potent antioxidant and tyrosinase inhibition activities (Kuo et al., 2005). Zingerone was also found to inhibit liver microsomal lipid peroxidation at higher concentrations (Reddy and Lokesh, 1992) and to act as an effective scavenger of superoxide anions as measured by nitroblue tetrazolium reduction in a xanthine-xanthine oxidase system (Krishnakanta and Lokesh, 1993).

Gingerdione is one of the components from ginger. 1-(3,4-Dimethoxyphenyl)-3,5-dodecen-1-ol (I6), a gingerdione derivative, synthesized by Hsu and his co-workers, has been demonstrated to be an effective anti-tumor agent in human leukemia cells. Results of the above study suggested that it induces G1 arrest in human leukemia HL 60 cells through down-regulation of G1-associated cyclin D2, cyclin E and cdc25A and up-regulation of CDKI, p15 and p27, and may contribute to I6-mediated cell cycle arrest along with apoptosis by the decrease in Bcl-2 expression and activation of caspase-3 activity (Hsu et al., 2005).

Nakamura et al. (2004) investigated the phase II detoxification enzyme induction of zerumbone (ZER), a sesquiterpene compound occurring in tropical ginger (Zingiber zerumbet Smith), in rat normal liver epithelial cell line RL34. Exposure of cells to ZER resulted in significant induction of glutathione S-transferase and nuclear localization of the transcription factor Nrf2. This study also implied an antioxidant role for this detoxification system activation by ZER in the neutralization of lipid peroxidation in hepatocytes, providing a new insight for cancer prevention.

1.3. In vivo cancer preventive effects

Ginger has been found to be anticarcinogenic via multiple pathways. Although chemopreventive activities of ginger have been examined (Koshimizu et al., 1988; Katyar et al., 1996), very little information is available in the literature with regard to the effects of individual constituents of ginger on experimental carcinogenesis.

Antioxidant and anti-inflammatory activity: The identification of plant-derived compounds or phytochemicals having the capacity to interfere with carcinogenic processes has been receiving increased interest among researchers. Many herbs and spices are known to possess an array of biochemical and pharmacological activities including antioxidant and anti-inflammatory properties that are believed to contribute to their anticarcinogenic and antimutagenic activities. Since tumor promotion is closely linked to inflammation and oxidative stress, a compound that exhibits anti-inflammatory and/or antioxidant properties could act as anticarcinogenic agent. Masuda et al. (2004) determined the structures of more than 50 antioxidants isolated from the rhizomes of ginger. [6]-Gingerol, the major pungent principle constituent of ginger, has been found to possess substantial antioxidant activity. This has clearly been demonstrated by inhibition of phospholipid peroxidation induced by the FeCl3-ascorbate system (Aeschbach et al., 1994) and its inhibitory effect on xanthine oxidase system (Chang et al., 1994) which is responsible for the generation of reactive oxygen species, such as superoxide anion. In a study, Guh et al. (1995) reported concentration-dependent (0.5–10 µM) inhibition by [6]-gingerol of arachidonic acid-induced platelet aggregation and formation of thromboxane B2 and prostaglandin D2. Gingerol, shogaol and other structurally related substances in ginger inhibit prostaglandin and leukotriene biosynthesis through suppression of 5-lipoxygenase or prostaglandin synthetase (Kiuchi et al., 1982; Kiuchi et al., 1992; Flynn et al., 1986). The ethanol extract of ginger reduced carrageenan-induced paw edema (Mascolo et al., 1989). Water and alcoholic extracts of
Ginger have been shown to possess potent antioxidant activity and prevent lipid peroxidation (Shobana and Naidu, 2000). The rhizome of ginger contains pungent vanillyl ketones, including [6]-gingerol and [6]-paradol, and these have been reported to possess a strong anti-inflammatory activity and suppress TNF-α production in TPA-treated female ICR mice (Surh et al., 1999).

Zerumbone, found in subtropical ginger, exhibits anti-proliferative and anti-inflammatory activities and mediates its activity through the modulation of NF-κB activation (Takada et al., 2005). It inhibits the activation of NF-κB and NF-κB-regulated gene expression induced by carcinogens and this inhibition may provide a molecular basis for the prevention and treatment of cancer by zerumbone.

Skin cancer: The rhizome of ginger and its major pungent principle constituent [6]-gingerol have been shown to suppress promotion of mouse skin carcinogenesis in laboratory animals (Katiyar et al., 1996; Park et al., 1998). The inhibitory activity of ginger extracts in tumor initiation and promotion is due to its pungent vanillyl ketones, including [6]-gingerol and [6]-paradol (Surh et al., 1999).

Park et al. (1998) reported that [6]-gingerol inhibited TPA skin tumor promotion in addition to the inhibition of epidermal ornithine decarboxylase activity in ICR mice. In a study, Surh et al. (1999) found anti-tumor-promoting properties of both [6]-gingerol and [6]-paradol. Topical application of [6]-gingerol or [6]-paradol 30 min prior to TPA, attenuated the skin papillomagenesis initiated by 7,12-dimethylbenz[a]anthracene (DMBA) in female ICR mice and significantly inhibited the tumor-promoter-stimulated inflammation, TNF-alpha production, and activation of epidermal ornithine decarboxylase. Another study revealed that topical application of the ethanol extract of ginger resulted in suppression of TPA mediated induction of ornithine decarboxylase and its mRNA expression in SENCAR mouse skin (Katiyar et al., 1996). It was also found that ethanol extracts of ginger have anti tumor promoting effects in mouse skin tumorigenesis model and was concluded that animals pretreated with ginger extracts showed substantially lower tumor body burdens compared with non-ginger treated controls. Pre-application of ginger extract to mouse skin afforded significant inhibition of TPA caused epidermal edema (56%) and hyperplasia (44%) (Katiyar et al., 1996). Topical application of [6]-gingerol inhibited TPA-induced COX-2 expression along with suppressed NF-κB DNA binding activity in mouse skin (Kim et al., 2004). [6]-Paradol and its synthetic nonpungent analog, [6]-dehydroparadol, significantly decreased the incidence and the multiplicity of skin tumors initiated by DMBA and promoted by TPA. Topical application of [6]-paradol and its derivatives inhibited TPA-induced ear edema and H₂O₂ production and myeloperoxidase activity in the dorsal skin of mice (Chung et al., 2001). The anti-tumor initiating and promoting activities of zerumbone in mouse skin were evaluated by Murakami et al. (2004) using a conventional 2-stage carcinogenesis model. Results indicated the prevention of both tumor initiation and promotion process, through the induction of anti-oxidative and phase II drug metabolizing enzymes as well as attenuation of pro-inflammatory signaling pathways. They further showed that single topical application of zerumbone (2 μM) 24 h before application of DMBA markedly suppressed skin tumor incidence by 60% and average number of tumors by 80%. Repeated pretreatment (16 nM) twice weekly during the post-initiation phase reduced the number of TPA-induced tumors by 83%.

Gastrointestinal cancer: Ginger is often prescribed in Chinese and Japanese medicine for a variety of gastrointestinal disorders. In the US, ginger is often promoted as a digestive aid and treatment for abdominal pain, indigestion and ulcers. Although human clinical trials have not been conducted, several animal studies support some of these claims. To the best of our knowledge, there is only one report that deals with the intestinal chemopreventive activity of gingerol assessed in experimental animals (Yoshimi et al., 1992). They showed that gingerol, when given to male F344 rats in the diet at a concentration of 0.02% for 3 weeks, significantly reduced the multiplicity of azoxymethane-induced intestinal carcinogenesis. However, it is unclear which stage of carcinogenesis is suppressed by gingerol in this experiment because the carcinogen was administered during the gingerol treatment.

Several observations made in the past are suggestive of anti-ulcerogenic effect of ginger and its constituents. Studies on the cytoprotective and gastric anti-ulcer properties of ginger have been carried out in albino rats using cytodestructive effects produced by 80% ethanol, 0.6 M HCl, 0.2 M NaOH and 25% NaCl (al-Yahya et al., 1989) as well as those induced by non-steroidal anti-inflammatory drugs. The results of this study demonstrated that the extract (500 mg/kg, orally) exerted highly significant cytoprotective effects on gastric lesions. Yamahara et al. (1988) reported that crude acetone extract of ginger, isolated zingiberene (the main terpenoid from acetone extract), as well as [6]-gingerol significantly inhibited gastric lesions induced by HCl and ethanol in rats. The orally administered acetone extract at 1000 mg/kg body weight (bw), zingiberene at 100 mg/kg bw and 6-gingerol, at 100 mg/kg bw significantly inhibited gastric lesions by 97.5, 53.6 and 54.5%, respectively. These results are suggestive of zingiberene and 6-gingerol being the important constituents in stomachic medications containing ginger.

Similarly, Chinese investigators utilized various experimental gastric ulcer models with orally administered dry and roasted ginger decoctions and found that roasted ginger has an obvious inhibitory tendency against stomach damage (Wu et al., 1990). In a study, Yoshikawa and his co-worker in Japan (1994) identified a new compound [6]-gingesulfonic acid in ginger extracts, which was found to be effective in preventing ulcers in an experimental rat model.

These protective effects on the gastric mucosa seem to involve an increased mucosal resistance or potentiation of some defensive factor or mechanism against noxious
chemicals. Clearly, more work is necessary to further substantiate these animal studies and to clarify which ginger constituents are active and by what mechanisms.

Colon cancer: Ginger and its components have been used traditionally for the treatment of gastrointestinal ailments such as motion sickness, dyspepsia and hyperemesis gravidarum, and is also reported to have colon cancer chemopreventive activity. Gingerol also inhibited the growth of human colorectal cancer cells, (Bode, 2003). The author claimed that these results strongly suggest that ginger compounds may be effective chemopreventive and/or chemotherapeutic agents for colorectal carcinomas. In his first experiment, mice were fed with ginger before and after tumor cells were injected. In the second set of experiments, ginger was fed only after their tumors had grown to a certain size. The efficacy of ginger was found to be significant in both cases. In a recent study its modifying potential on the process of colon carcinogenesis induced by 1,2-dimethylyhydrazine (DMH) was investigated in male Wistar rats using the aberrant crypt foci assay by Dias et al. (2006). Results showed that dietary intake of ginger does not significantly change the proliferative or apoptosis indexes of the colonic crypt cells.

The effect of ginger on the initiation and post-initiation stages of DMH-induced colon carcinogenesis in male Wistar rats was studied by Manju and Nalini (2005) where the results showed a lower incidence of tumors. It was further concluded by the researchers of same group that ginger supplementation suppressed colon carcinogenesis by reducing lipid peroxidation and significantly enhancing the enzymatic and non-enzymatic antioxidant levels.

Breast cancer: The effects of chronic treatment with hot water extract of ginger rhizome on spontaneous mammary tumorigenesis have been examined in mice. In mice given free access to extract of ginger (0.125%) in drinking water, the development of mammary tumors was significantly inhibited (Nagasawa et al., 2002).

Effects on angiogenesis and metastasis: Angiogenesis, the formation of new blood vessels from pre-existing endothelium, is fundamental in a variety of physiological and pathological processes such as chronic inflammation, and tumor progression. Metastasis reported that [6]-gingerol, a major pungent ingredient present in ginger, has potent anti-angiogenic activity in vitro and in vivo (Kim et al., 2005a). These results points towards a possible role of [6]-gingerol in preventing cancers from becoming malignant, presumably by selective inhibition of angiogenesis formation at the tumor site. Gingerol suppressed experimental metastases in tumor-bearing mice and results suggested that [6]-gingerol may inhibit tumor growth and metastasis via its anti-angiogenic activity (Kim et al., 2005a,b). Furthermore, [6]-gingerol has been shown to inhibit pulmonary metastasis in mice implanted with B16 melanoma cells, probably through stimulation of the host’s immune functions (Suzuki et al., 1997).

2. Conclusion

Overall, a significant number of in vitro and laboratory animal studies provide substantial evidences that ginger and its organic pungent vallinoid compounds are effective inhibitors of the carcinogenic process. The use of this ancient medicine for gastrointestinal problems (stimulation of digestion) has been given scientific approval. Today, medicinal ginger is used mainly for prevention of the symptoms of travel sickness and in cancer chemoprevention. Due to its abundance, low cost and safety in consumption, ginger has been the subject of intensive scientific research over the past two decades, which has shown anticarcinogenic, antibacterial, antifungal, hypoglycemic, and anti-atherosclerotic activity. Several mechanisms are likely to account for this protection. The benefits provided by ginger must be viewed as part of the entire diet, since several dietary constituents can influence the degree of protection. Future research should focus on how genetic variability and daily environmental factors influence the anticaner benefits attributed to ginger and its pungent vallinoid components. Further studies on determining the anticaner activity of ginger and its active components should ideally include human intervention trials to investigate its effectiveness against human cancers and other diseases.

References


