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Abstract

Tea consumption as a beverage is very common in various parts of the world. It has attained a worldwide liking and measure of social status in many parts. Tea contains various chemicals which have positive effects on health from heart to skin. It has been associated with the cure of aging to potent anticancer agent also. Considering these facts an attempt was made to establish a relation between tea and oral health. Tea has its effects on oral microorganisms, anticariogenic properties, and reduction of gingivitis as well as periodontitis. A cup of tea immediately after lunch had reduced dental caries in children and rinsing with 0.2% Chinese green tea decreased plaque and the gingival index significantly. Tea has been found to be effective against oral cancer, precancerous lesions and conditions as well. Hence tea has been rightly said as a functional food for health. Green tea has shown to have bactericidal effects on Porphyromonas gingivalis and Prevotella species. The gingival inflammation is reduced and a marked reduction in pocket size has been noticed. Tea selectively induces p57 and apoptosis as well as inhibits the growth and invasion of oral carcinoma.

Keywords: tea, oral cancer, antimicrobial properties, catechins, EGCG, gingivitis, periodontitis

1. Introduction

Ancient Chinese and Japanese medicines have emphasized the fact that green tea consumption could heal wounds and cure diseases. In 2737 BC Chinese had a belief on tea for its healing powers. Lu Yu who was a scholar in China, who had written a treatise in AD 780, entitled Cha Ching, states that ‘tea tempers the spirits, harmonizes the mind, dispels the lassitude, relieves fatigues, awakens thought, prevents drowsiness, refreshes the body and clears the perspective faculties’ [1]. In the ancient system of Medicine, Ayurveda had listed tea in the group of medicaments as ‘rasayanas' that bring about positive health, resistance to diseases and assured full lifespan of quality living, unlike drugs that cure after disease has struck [2].

Tea has been considered a desired drink worldwide. The world consumed 2.9 million tones of tea in 2016 which was more when compared to 1.6 in 2002 and this may shoot to 3.3 million tones in 2021 (Euromonitor data). More than half of tea is contributed by Asia and the top three markets by per capita consumption of tea are Turkey, Ireland and UAE [3]. Different forms of tea are obtainable in the market claiming varied health benefits. These categories are based on the oxidation process. There are six different types of tea produced [4]:
Tea has shown to have various benefits on Health and oral health. Tea reduces the risk of several major lifestyle related diseases which include cancer, arteriosclerosis and cardiovascular diseases, neural and obesity problems, diabetes, diseases of the kidneys and liver, pulmonary ailments, flu, SARS and even AIDS. The impact on oral health though less researched has been beneficial.

2. Tea and oral health

Oral/dental diseases are a costly burden to health care services, accounting for between 5 and 10% of total health care expenditures and exceeding the cost of treating cardiovascular disease, cancer and osteoporosis in industrialized countries [5]. In low-income countries, the cost of traditional restorative treatment of dental disease would probably exceed the available resources for health care. Dental health promotion and preventive strategies are clearly more affordable and sustainable. Although not life-threatening, dental diseases have a detrimental effect on quality of life in childhood through to old age, having an impact on self-esteem, eating ability, nutrition and health.

Oral health is related to diet in many ways, for example, through nutritional influences on craniofacial development, oral cancer and oral infectious diseases [6]. Both animal studies and experimental investigations in humans have shown that black tea extract increases plaque fluoride concentration and reduces the cariogenicity of a sugar rich diet. The protection against the causative organisms of dental caries is well documented by Banerjee in 1990 [7]. Sava et al. showed that the melanin-like pigment from black tea has immunostimulant activity [8].

2.1 Anticariogenic effects

Dental caries is a transmissible microbial disease affecting the hard tissues of teeth caused by acids from bacterial metabolism leading to demineralization and dissolution of enamel and dentin. The bacteria responsible of producing organic acids as a by-product of their metabolism of fermentable carbohydrates. The caries process is a continuum in the oral cavity resulting from many cycles of demineralization and remineralization [9].

The studies of cariostatic effects of tea were started in the 1940s and 1950s showing fluoride to be the active component [10]. Reports by many researchers have showed that the tea consumption leads to reduction in dental caries in humans and experimental animals, that tannins and fluoride were the reason for this inhibitory effect [11–14]. Despite the positive animal data supporting the positive relationship between tea and dental caries prevention, relatively little attention has
been given to this field of research. Green tea extracts, or polyphenols, have been reported to inhibit \textit{in vitro} growth, acid production and water insoluble glucan synthesis by glucosyltransferase enzyme of \textit{Streptococcus mutans} [12, 15–18]. Similar findings have been reported for oolong tea by Nakahara et al., in 1993 and Ooshima et al., in 1993 [18, 19]. Taiwanese green, black and oolong teas have also been shown to inhibit \textit{in vitro} growth of selected cariogenic and periodontal pathogens [20, 21]. In an adult human study by Wu et al. [22] rinsing with black tea ten times a day for 7 days resulted in significantly less pronounced pH fall, a lower plaque index ($P < 0.05$) and lower numbers of mutants streptococci and total oral streptococci in plaque but not in saliva. Fluoride concentrations in plaque and saliva increased, reaching a maximum at day 7. Black tea and its polyphenols may benefit human oral health by inhibition of dental plaque, acidity and cariogenic microflora.

Animal studies have shown that specific pathogen-free (SPF) rats infected with \textit{S. mutans} and then fed a cariogenic diet containing green tea polyphenols demonstrated significantly lowered caries scores [17]. Supplementing drinking water of rats with 0.1% green tea polyphenol along with a cariogenic diet also significantly reduced total fissure caries lesions [23]. Animal studies using oolong tea gave similar results and it was suggested that active substances may affect bacterial virulence factors other than the glucosyltransferase enzymes [24]. Caries were found to be significantly lower among children who drank a cup of tea immediately after lunch and the tea polyphenols, rather than fluoride, were found to be responsible for the anticariogenic effects [25]. Another study reported that rinsing with 0.2% Chinese green tea while brushing decreased plaque and the gingival index significantly [26].

Tea drinking has been attributed as one of the factors in the declining prevalence of caries in Tunisia [27]. Tea extracts have also been shown to inhibit human salivary amylase and tea consumption may reduce the cariogenic potential of starch-containing foods, such as biscuits and cakes, because tea may reduce the tendency for these foods to serve as slow-release sources of fermentable carbohydrate [28]. It is likely that cariogenic challenge in a cariogenic diet may be overcome by the simultaneous presence of green tea in the diet. An anticariogenic potential of black tea has been suggested in various \textit{in vitro} studies. [20, 29, 30]. Black tea and its polyphenols inhibited growth, acid production, metabolism and glucosyltransferase enzyme activity of Mutans Streptococci and dental plaque bacteria.

Tea is a source of fluoride (F) as well as many other dietary trace elements. The caries-preventive effect of teas was first believed to be due to its fluoride content. More recent studies, however, have pointed out that the polyphenol contents of tea may affect plaque formation and metabolism as well [31]. The commercial tea plant, \textit{Camellia sinensis}, takes up F from the soil by passive diffusion and concentrates it in the leaves by transpiration [32]. Due to differing soils, types of tea leaves, infusion times and methods of analysis, a great deal of variation in tea content has been found. Coupled with the various drinking habits among different people, it is very difficult to calculate the contribution of tea to total fluoride intake. A recent animal study showed that rats consuming black tea (prepared from fluoride-free water) over a 2-week period had a significantly lower rate of caries than those consuming non-fluoridated water. Furthermore, the caries scores in the group receiving tea were significantly greater than those in the group receiving fluoridated water. The authors suggested that black tea consumption attenuates the development of caries in young, caries-prone rats [14]. Wei et al. found a 15-min infusion to result in a mean fluoride concentration of 1.75 p.p.m. for 15 Chinese teas, 1.24 p.p.m. for 11 Ceylon/Indian teas and a negligible F amount for six herbal teas [32]. The bioavailability of fluoride in tea has been said to be approximately 85%. A review by Kavanagh and Renehan lists over 10 papers that measured the fluoride content of various teas [33]. Several studies have also calculated the total F intake from tea consumption.
When using the assumptions of 2.5 cups/day, 150 ml per cup and 2.2 p.p.m. F diluted in half with milk for children, an ingested range of 0.1 mg to 1.08 mg was found. Wei et al. estimated a daily F intake at a level of 1.05 mg [32]. As well as teas are unlikely to cause fluorosis by themselves, but they may be significant contributors to the total fluoride intake of children.

2.2 Tea and gingivitis/periodontal disease

Periodontal disease (PD) is one of the most omnipresent diseases of mankind, which is also second most common oral disease worldwide, after dental caries. This is a chronic condition in which a multiple and complex group of inflammatory diseases are affecting the periodontal complex i.e., tissue that surround and support the teeth (Periodontium, bone, gingival fibers). Negligence towards this condition may show further deterioration of periodontium leading to progressive loss of the alveolar bone around the teeth and subsequent loss of teeth. In fact, PD remains the most common cause of tooth loss in the world today; in the United States, it has a prevalence of 30–50% of the population and can affect up to 90% of the population worldwide [34].

Researchers have observed that for every one cup of green tea consumed per day, there was a decrease in the indicators of gingival inflammation, in turn reducing the periodontal disease. Green tea catechin has been shown to be bactericidal against Porphyromonas gingivalis and Prevotella species in vitro. A local slow delivery system of green catechin along with mechanical treatment was found to be effective in improving periodontal status. There was a reduction pocket size and the suppression of peptidase activities in the gingival crevicular fluid [35]. Tea catechins containing the galloyl radical (Epicatechin Gallate [EGC] and Epigallocatechin Gallate [EGCG]) possess the ability to inhibit both eukaryotic and prokaryotic cell-derived collagenase, an enzyme that plays an important role in the disruption of the collagen component in the gingival tissues of patients with periodontal disease [36–38]. Catechin derivatives have been reported to inhibit certain proteases and toxic metabolites of P. gingivalis and may reduce periodontal breakdown [39, 40]. Green tea catechins EGC have also been shown to inhibit protein tyrosine phosphatase in Prevotella intermedia [41]. Zhu et al. have shown that purified tea polyphenols inhibited in vitro growth and H2S production of P. gingivalis and Fusobacterium nucleatum associated with human halitosis [42].

The molecular and cellular effects of green tea on oral cells of smokers were researched [43]. A recent human study investigated the effect of tea polyphenols in the form of chew candies on gingival inflammation over a 4-week period [44]. The approximal plaque index (API) and sulcus bleeding index (SBI) were determined at the end of day 7 and day 28. These authors suggested that tea polyphenols might exert a positive influence on gingival inflammation.

2.3 Oral cancer

Oral cancer is a global public health problem and relevant to dentists due to proximity of this area to the work carried out by them. It is located within the top 10 ranking incidence of cancers and despite the progress in research and therapy, survival has not improved significantly in the last years, representing a continuing challenge for biomedical science. Oral cancer is a malignant neoplasia, which arises on the lip or oral cavity. It was traditionally defined as a squamous cell carcinoma (OSCC), as 90% of cancers are histologically originated in the squamous cells in the oral cavity [45].

The influence of tea on oral cancer as well as various studies has been summarized in Tables 1–3 [44, 46–75]. Green tea polyphenols are found to induce
<table>
<thead>
<tr>
<th>Sl no.</th>
<th>Author</th>
<th>Type of study</th>
<th>Sample</th>
<th>Country</th>
<th>Intervention</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>The Indian-US Head and Neck Cancer Cooperative Group, 1997</td>
<td>Population based chemo preventive experimental trial—6 months</td>
<td>64</td>
<td>India</td>
<td>Green—3.6 g per day and 5.4 g per day</td>
<td>Feasible</td>
</tr>
<tr>
<td>2.</td>
<td>Khafif A et al., 1998</td>
<td>In vitro experimental trial</td>
<td>(−)-epigallocatechin-3-gallate (EGCG) from green tea</td>
<td>A reduction of 4.4–8.5-fold in cell cycle of cancer cells</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Khafif A et al., 1998</td>
<td>In vitro experimental trial—mice</td>
<td>EGCG (−)-epigallocatechin-3-gallate</td>
<td>Inhibited cancer cells growth</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Yang CS, Lee MJ, Chen L., 1999</td>
<td>In vivo experimental trial—human</td>
<td>Green tea</td>
<td>Increase in (−)-epigallo-catechin (EGC; 11.7–43.9 microg/ml), EGC-3-gallate (EGCG; 4.8–22 microg/ml), and (−)-epicatechin (EC; 1.8–75 microg/ml) levels in saliva</td>
<td></td>
<td></td>
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<tr>
<td>5.</td>
<td>Li N et al., 1999</td>
<td>Double blind randomized controlled human clinical trial—6 months</td>
<td>Tea—3 g/day</td>
<td>Reduction in size in 37.9% of leckoplakic patients</td>
<td></td>
<td></td>
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<tr>
<td>6.</td>
<td>Li N, Han C, Chen J, 1999</td>
<td>In vivo animal experimental trial—hamsters</td>
<td>1.5% green tea, 0.3% tea pigments, and 0.5% mixed tea</td>
<td>Reduced buccal pouch tumor burden and the incidence of dysplasia and oral carcinoma (P &lt; 0.01)</td>
<td></td>
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</tr>
<tr>
<td>7.</td>
<td>Li N et al., 1998</td>
<td>Randomized control trial—human—6 months</td>
<td>Mixed tea—3 g/day oral and 0.1% topical</td>
<td>Significant decrease in micronuclei (P &lt; 0.001)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8.</td>
<td>Masuda M, Suzui M, Weinstein IB, 2001</td>
<td>In vitro experimental trial</td>
<td>Egcg—10 microg/ml</td>
<td>Growth inhibition (P &lt; 0.001)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9.</td>
<td>Hsu SD et al., 2002</td>
<td>In vitro experimental trial</td>
<td>Tea extracts, green tea polyphenols, (−)-epigallocatechin-3-gallate (EGCG)</td>
<td>Selectively induce apoptosis only in oral carcinoma cells, EGCG inhibit the growth and invasion of oral carcinoma cells</td>
<td></td>
<td></td>
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<tr>
<td>10.</td>
<td>Li N et al., 2002</td>
<td>In vivo experimental trial—hamsters</td>
<td>0.6% green tea powder, 0.6% green tea powder + 10 mumol curcumin</td>
<td>Decreased the number of visible tumors and the tumor volume. Suppression of cell proliferation, induction of apoptosis, and inhibition of angiogenesis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11.</td>
<td>Masuda M et al., 2003</td>
<td>In vitro experimental trial</td>
<td>10 or 30 microg of EGCG</td>
<td>50% inhibition of growth of carcinoma cells</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12.</td>
<td>Lee MJ et al., 2004</td>
<td>In vivo cross over Clinical trial—human</td>
<td>2 g of black tea, 2 g of green tea</td>
<td>Concentrations of catechins (C(max) = 131.0–2.2 micro M) and theaflavins (C(max) = 1.8–0.6 micro M) were observed in saliva in the 1st hour</td>
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<tr>
<td>Sl no.</td>
<td>Author</td>
<td>Type of study</td>
<td>Sample</td>
<td>Country</td>
<td>Intervention</td>
<td>Outcome</td>
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</tr>
<tr>
<td>13</td>
<td>Srinivasan P, Sabitha KE, Shyamaladevi CS</td>
<td><em>In vivo</em> experimental trial—rats</td>
<td>1 month</td>
<td></td>
<td>Green tea polyphenols (GTP) (200 mg/kg)</td>
<td>Enhances the cellular thiol status thereby mitigate oral cancer</td>
</tr>
<tr>
<td>14</td>
<td>Babich H et al., 2005</td>
<td><em>In vitro</em> experimental trial</td>
<td></td>
<td></td>
<td>Catechin gallate (CG), epigallocatechin gallate (EGCG), epigallocatechin (EGC), catechin (C) and epicatechin (EC)</td>
<td>Reduced carcinoma HSC-2 cells of oral cavity</td>
</tr>
<tr>
<td>15</td>
<td>Gonzalez de Mejia E et al., 2005</td>
<td><em>In vitro</em> controlled experimental study</td>
<td></td>
<td></td>
<td>Yerba mate tea products</td>
<td>Inhibition of topoisomerase II (cancer cell proliferation)</td>
</tr>
<tr>
<td>16</td>
<td>Hsu S et al., 2005</td>
<td><em>In vitro</em> experimental study</td>
<td></td>
<td></td>
<td>Tea polyphenols</td>
<td>p21WAF1 is involved in EGCG-induced growth arrest of OSC2 cells</td>
</tr>
<tr>
<td>17</td>
<td>Halder A et al., 2005</td>
<td><em>In vivo</em> human experimental trial—1 year</td>
<td>82</td>
<td>India</td>
<td>Black tea</td>
<td>Significant decrease in the micronuclei frequency and chromosomal aberrations, which correlated with the clinical improvement</td>
</tr>
<tr>
<td>18</td>
<td>Hua Y et al., 2006</td>
<td><em>In vitro</em> experimental trial</td>
<td></td>
<td></td>
<td>Tea polyphenols</td>
<td>The human telomerase reverse transcriptase (hTERT) gene in the Tca8113 cancerous cell line was less (0.1 g/l, TP 0.05 g/l) when compared to controls (0.32 ± 0.05, 0.41 ± 0.04 and 0.72 ± 0.05, respectively) (P &lt; 0.05)</td>
</tr>
<tr>
<td>19</td>
<td>Ko SY et al., 2007</td>
<td><em>In vitro</em> experimental trial—hamsters</td>
<td></td>
<td></td>
<td>Green tea</td>
<td>Amyloid precursor protein (APP) expression was also significantly increased in MBN-induced HBP carcinomas but was significantly reduced by tea intake (P &lt; 0.0001)</td>
</tr>
<tr>
<td>20</td>
<td>Tsao AS et al., 2009</td>
<td>Randomized control human trial—12 weeks</td>
<td></td>
<td></td>
<td>Green tea extracts at 500, 750, or 1000 mg/m² or placebo thrice daily</td>
<td>The OPL clinical response rate was higher in all GTE arms (n = 28; 50%) versus placebo (n = 11; 18.2%; P = 0.09). However, the two higher-dose GTE arms [38.8% (750 and 1000 mg/m²), 36.4% (500 mg/m²), and 18.2% (placebo); P &lt; 0.03] had higher responses, improved histology (21.4% versus 9.3%; P = 0.65)</td>
</tr>
</tbody>
</table>
Apoptosis (programmed cell death) in many types of tumor cells, including oral cancer cells. However, how the normal cells escape the apoptotic effect has not still been understood by the researchers. The effect of extracts and polyphenols of green tea as well as \((-\text{-epigallocatechin-3-gallate (EGCG) which is the most potent green tea polyphenol on normal human keratinocytes and oral carcinoma cells were assessed through assays for cell growth, invasion, combined with apoptosis. It was shown that the green tea and its constituents selectively induce apoptosis, whereas EGCG usually inhibits the growth and invasion of oral carcinoma cells. This difference in the identification of normal cells and malignant cells by green tea and its constituents was attributed to the induction of p57, a cell cycle regulator.

### Table 2.

**Descriptive studies of tea and oral cancer.**

<table>
<thead>
<tr>
<th>Sl no.</th>
<th>Author</th>
<th>Type of study</th>
<th>Risk</th>
<th>Sample</th>
<th>Country</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Bundgaard T, et al., 1995</td>
<td>Case control study</td>
<td>OR = 0.45</td>
<td>561 Cases—161 Controls—400</td>
<td>Denmark</td>
<td>Squamous cell carcinoma occurrence</td>
</tr>
<tr>
<td>2.</td>
<td>Ariyawardana A et al., 2007</td>
<td>Cross sectional study</td>
<td>12,716</td>
<td>Sri Lanka</td>
<td>46.1 per 1000 for leukoplakia and 16.4 per 1000 for oral submucous fibrosis</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Ide R et al., 2007</td>
<td>Longitudinal study—10.3 years</td>
<td>HR—0.51 0.60 0.31</td>
<td>Japan</td>
<td>37 oral cancer cases. Did not suggest a prominent inverse association of green tea consumption with oral cancer, although there was a tendency for a reduced risk in women</td>
<td></td>
</tr>
</tbody>
</table>

### Table 3.

**Review.**

<table>
<thead>
<tr>
<th>Sl no.</th>
<th>Author</th>
<th>Type of study</th>
<th>Input</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Weisburger JH, Chung FL, 2002</td>
<td>Review</td>
<td>Tea</td>
<td>Chemo preventive effects of tea and mechanisms</td>
</tr>
<tr>
<td>3.</td>
<td>Lodi G et al., 2004</td>
<td>Review</td>
<td>Tea</td>
<td>Leukoplakia</td>
</tr>
<tr>
<td>5.</td>
<td>Klass CM, Shin DM</td>
<td>Review</td>
<td>Green tea</td>
<td>Premalignant lesions</td>
</tr>
<tr>
<td>6.</td>
<td>Boehm K et al., 2009</td>
<td>Systematic review</td>
<td>Tea</td>
<td>Reduced oral cancer</td>
</tr>
<tr>
<td>7.</td>
<td>Yang CS et al., 2008</td>
<td>Review</td>
<td>Tea and tea polyphenols</td>
<td>Reduced carcinogenesis</td>
</tr>
</tbody>
</table>
p57 mediated survival pathway in normal epithelial cells is the reason for the chemopreventive effects of green tea polyphenols in normal cells, while oral carcinoma cells undergo an apoptotic pathway. Regular consumption of green tea has favorable effects in the prevention of oral cancer. The oxidative stress and inflammation in the oral cavity may be reduced in the presence of green tea polyphenols (Figure 1). Green tea prevents the transformation of healthy cells to malignant cells and locally facilitates the induction of apoptosis in oral cancer cells.

3. Conclusions: tea—a multi edged sword

Tea is rich in the beneficial polyphenols and similar components that can supplement the recommended 5–10 vegetables and fruits per day. Tea is not a drug. It is a health food, akin to Rasayanas known to the ancient Indians. It has shown its effect on various diseases and oral diseases.

The above review also leads us to conclude that this popular beverage can regress tumors directly by inhibiting tumor angiogenesis, blocking metastasis and inducing apoptosis in cancer cells. Thus, tea, previously considered only as a popular beverage, can now emerge as a ‘multiedged sword’ against the various diseases.

Although there has been a substantial amount of research related to the study of teas and their health benefits, it has been difficult to compare data between laboratories due to the lack of standardization in experimental procedures. Teas used in studies often differed in their types, sources, method of manufacture and procedures for extraction. Analytical data of tea preparations were often not specified or provided, making the comparison of in vitro or in vivo data difficult among laboratories. Improvement in this aspect and encouragement in designing new multidisciplinary research approaches will strengthen our knowledge concerning this ancient beverage with its many health attributes.

At present, the use of tea in clinical application is still a long way from reality, and further controlled clinical trials in humans are warranted. Furthermore, consumption of tea may have added oral health benefits by controlling ‘through prevention’ the most prevalent infectious disease of humankind, namely caries considering the Indian scenario. With the added dental health implication among many other bioregulatory functions, tea can be considered as a functional food for oral health.

Figure 1.
The effect of tea on cancer.
Acknowledgements

I acknowledge the various resources from which I have gathered the information.

Conflict of interest

No conflict of interest.

Author details

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