



# TEA: Epigallocatechin gallate (EGCG) Reduces the Risk of Cancer

Matt Johnson III  
Beloit College, Beloit, WI



## ABSTRACT

Damage to biomolecules such as DNA and lipid is produced by reactive oxygen species (ROS). ROS are involved in the process of aging and various diseases such as cancer. Epigallocatechin gallate (EGCG) is an antioxidant found in plants. I have reviewed peer-reviewed literature to investigate my hypothesis, that drinking tea will decrease the risk of getting cancer. An article reported that **EGCG** prevents chromosomal damage induced by ROS *in vitro* in tissue culture of cells. Another study showed that drinking < 10 μmol/L of EGCG will reduce the chances of cancer. However, **EGCG** itself induces chromosomal damage at 100 μmol/L (1), which is beyond physiologic levels from just drinking tea. This damage is caused by the production of H<sub>2</sub>O<sub>2</sub> by **EGCG** (1). So there may be hazard in taking supplements containing too much EGCG. These results are important because if people drink about 5 cups of tea daily, it can save millions of lives. Simply by drinking tea, a person can reduce the risk of cancer.

## INTRODUCTION

Oxidative damage to biomolecules such as DNA and lipid is produced by reactive oxygen species (ROS), and is involved in the process of aging and various diseases such as cancer (1). During oxidation of food in the body, free radicals are produced. Free radicals are single (unpaired) electrons that freely interact, producing chain reactions that damage nearby molecules and cells. The cytokinesis block micronucleus (CBMN) assay using WIL2-NS cells, a human B lymphoblastoid cell line, has been shown to be a sensitive assay system to examine ROS-induced chromosomal damage (4). Intake of antioxidants has been shown to reduce ROS-related disease in humans (1). Accordingly, antioxidants, particularly in food, have received great attention as primary preventive ingredients against various diseases. Epigallocatechin gallate (EGCG), also known as Epigallocatechin 3-gallate, shown in Figure 1, is an antioxidant found in plants. EGCG specifically is found in teas—more in green teas than black (See table 1). Green tea is produced from the unfermented leaves of *Camelia sinensis*, and polyphenols, known as catechins, constitute its principal chemical components (2). EC, ECG, EGC, and EGC-3 gallate are the major catechins contained in green tea (2). Black tea is made by extensive enzymatic oxidation of these polyphenols which converts them to theaflavins (2). Theaflavin, theaflavin-3 gallate, theaflavin-3-gallate, and theaflavin-3-3' digallate are the principal theaflavins in black tea (2). Since EGCG can trap and thus inactivate free radicals, it can lower the amount of damage to DNA and other vital molecules (2). As DNA mutations and other oxidative damage can cause cancer, drinking tea may be able to lower people's risk of disease such as cancer. In the study, the CBMN assay system in WIL2-NS cells were used to examine whether **EGCG** itself induces chromosomal damage, and whether physiological concentrations of **EGCG** protect against chromosomal damage induced by various ROS

## METHOD

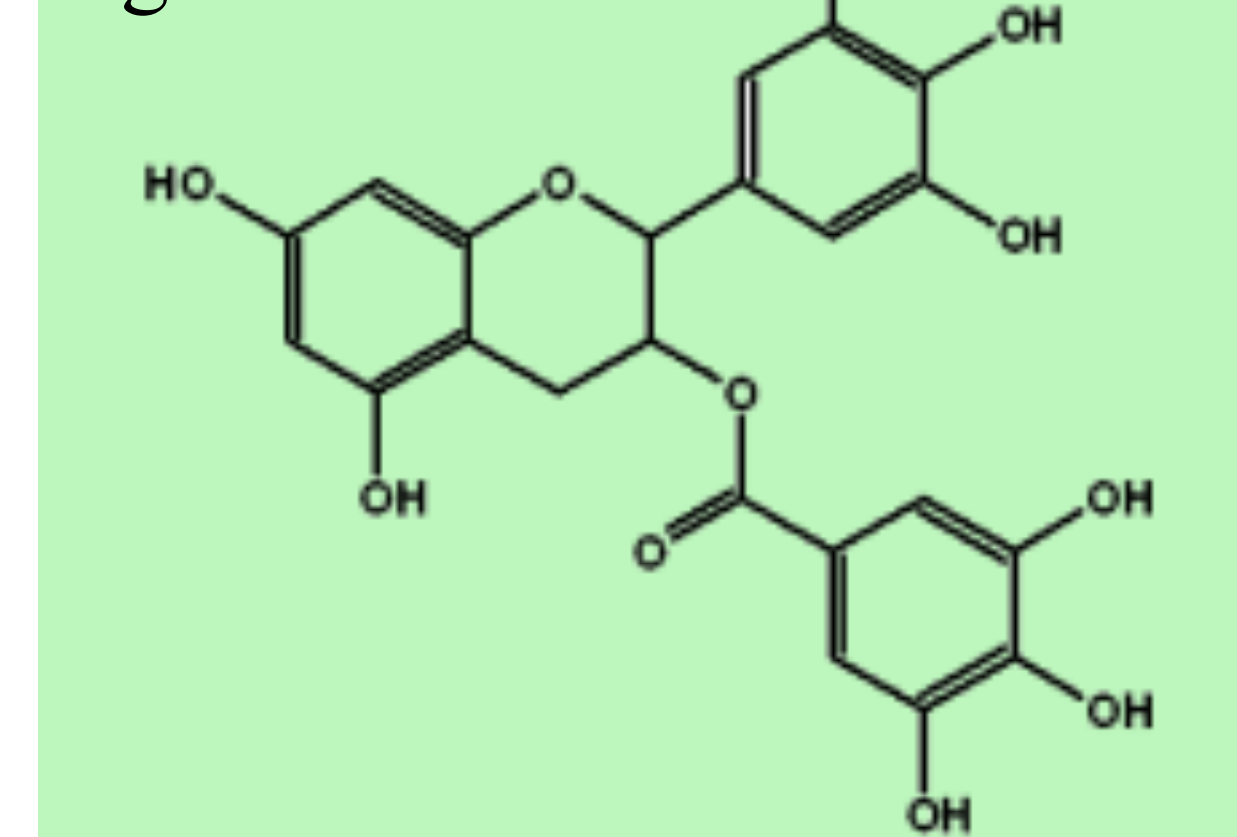
I searched many nutrition and medical peer-reviewed journals, and include data that are important. I used websites that were either credible or at least provided a list of references

## FINDINGS

Catechin is the class of molecules that include EGCG. In an *in vivo* study where humans were tested, when subjects drank an excess of catechin liquid, the concentration of **EGCG** in plasma reached a maximum between 0.3–1 μmol/L at 2 h (1).

In an *in vitro* study using tissue culture, WIL2-NS cells were treated with up to 100 μmol/L of **EGCG** to examine whether **EGCG** itself induced chromosomal damage, which was evaluated by an increase in MNed BN cells (1). **EGCG** did not induce chromosomal damage at **EGCG** concentrations < 10 μmol/L (1). However, 100 μmol/L **EGCG** induced chromosomal damage (1). The nuclear division index, a measure of cell division, decreased at **EGCG** concentrations of at least 10 μmol/L (1). When production of H<sub>2</sub>O<sub>2</sub> was also examined by incubating **EGCG** in a solution without cells, **EGCG** produced H<sub>2</sub>O<sub>2</sub> at the concentrations of at least 30 μmol/L, but not at concentrations of < 10 μmol/L or less(1). (See Table 2)

Figure 1



Epigallocatechin gallate belongs to the family of catechins. It contains 3 phenol rings and has very strong antioxidant properties.

## Epigallocatechin gallate

MW: 458.37

Formula: C<sub>22</sub>H<sub>18</sub>O<sub>11</sub>

**TABLE 1 Concentration of polyphenols and theaflavins in decaffeinated BT solids and BT and GT-diets used in the C57BL/6 mouse studies<sup>1</sup>**

Polyphenol	Green Tea -diet	Black Tea -diet
EGCG	6.51 ± 0.18	0.29 ± 0.01

<sup>1</sup> Values are means ± SD, *n* = 2.

<sup>2</sup> Below the detection limit of 0.01 mg/g diet.

**TABLE 2 Chromosomal and cellular damage and production of hydrogen peroxide induced by EGCG<sup>1</sup>**

	EGCG dose, μmol/L					
	0	0.3	1	10	30	100
Cell study <sup>2</sup>						
Chromosomal damage (MNed BN cells/1000 BN cells)	14.7 ± 2.7	8.9 ± 0.6	12.5 ± 1.8	11.3 ± 2.6	—	75.7 ± 4.1*
Nuclear division index	3.0 ± 0.2	2.8 ± 0.1	2.8 ± 0.1	2.7 ± 0.0*	—	2.3 ± 0.1*
Cell-free study <sup>3</sup>						
H <sub>2</sub> O <sub>2</sub> production (μmol/L)	ud	ud	ud	ud	2.8 ± 0.4*	10.6 ± 0.8*

<sup>1</sup> Values are means+/- SEM, *n* = 3. \* Different from 0 μmol/L EGCG.

<sup>2</sup> WIL2-NS cells were treated with various concentrations of EGCG for 60 min. The extent of chromosomal damage and nuclear division index, an indication of cellular damage, were evaluated by the CBMN assay.

<sup>3</sup> Production of H<sub>2</sub>O<sub>2</sub> in various EGCG solutions was measured in the cell-free study.

—, not determined; ud, undetectable level.

## FINDINGS (cont'd)

When WIL2-NS cells were exposed to ROS (H<sub>2</sub>O<sub>2</sub>, tert-BuOOH or superoxide) for 30 min, chromosomal damage dose-dependently increased up to the value of 150 MNed BN cells/1000 BN cells (data not shown) (1). WIL2-NS cells were pretreated with various concentrations (0, 0.3, 1 and 10 μmol/L) of **EGCG** for 60 min, and then exposed to H<sub>2</sub>O<sub>2</sub> (20 μmol/L), tert-BuOOH (1 mmol/L), or superoxide (20 mU of XO) to induce chromosomal damage (1). Regardless of the type of ROS, **EGCG** dose-dependently prevented the ROS-induced chromosomal damage. With tert-BuOOH, the increase in the chromosomal damage was significantly attenuated even at 0.3 μmol/L of **EGCG** (1). In this study, **EGCG** induced chromosomal damage at 100 μmol/L but did not induce such damage at physiological concentrations. Furthermore, physiological concentrations of **EGCG** protected against chromosomal damage induced by H<sub>2</sub>O<sub>2</sub>, tert-BuOOH and superoxide (1).

To elucidate whether the protective effect of **EGCG** was exerted inside of the cells, WIL2-NS cells were incubated with 0.3–10 μmol/L **EGCG**, washed twice with HBSS to eliminate **EGCG** outside of the cells, and then exposed to various ROS to induce chromosomal damage (1). Chromosomal damage induced by H<sub>2</sub>O<sub>2</sub> (30 μmol/L) and superoxide (20 mU of XO) was prevented by the pretreatment of cells with **EGCG**. In contrast, no preventive effect by **EGCG** was detected when cells were exposed to 1 mmol/L tert-BuOOH (1).

To examine the direct interactions between **EGCG** and ROS, each ROS was added to a solution of 3 μmol/L **EGCG**, incubated for 30 min, and then the changes in the concentration of **EGCG** were analyzed (1). The concentration of **EGCG** was not decreased by incubation with H<sub>2</sub>O<sub>2</sub>, but was significantly decreased by incubation with tert-BuOOH and superoxide (1). To further confirm the lack of interaction between **EGCG** and H<sub>2</sub>O<sub>2</sub>, changes in the concentration of H<sub>2</sub>O<sub>2</sub> were analyzed instead of changes in **EGCG**. No decrease in the concentration of H<sub>2</sub>O<sub>2</sub> was detected in mixtures of various concentrations of H<sub>2</sub>O<sub>2</sub> and **EGCG** (1).

## CONCLUSION

Tea is a cheap and safe drink that has genetic proprieties that can reduce the risk of ROS. Governments spend billions of dollars on cancer research, but this research has proven that drinking tea can be an effect means to prevent cancer. The findings reported here strongly support that **EGCG** has no genotoxicity even when an excess amount is consumed and has the beneficial effect of minimizing ROS-induced chromosomal damage, which is believed to be involved in various diseases and aging (1). These findings allow people to take a more active role in their health. Instead of waiting to get cancer, people can reduce the chances of getting it by simply drinking tea.

## LITERATURE CITED

- Sugisawa A and Umegaki K (2002) Physiological concentrations of (–)-epigallocatechin-3-O-gallate (EGCG) prevent chromosomal damage induced by reactive oxygen species in WIL2-NS cells. *J Nutr* **132**: 1836–1839.
- Leone M, Zhai D, Sareth S, Kitada S, Reed JC, Pellecchia M. Cancer prevention by tea polyphenols is linked to their direct inhibition of antiapoptotic Bcl-2-family proteins. *Cancer Res* 2003;63:8118–21.
- Henning SM, Aronson W, Niu Y, Conde F, Lee NH, Seeram NP, Lee RP, Lu J, Harris DM, et al. Tea polyphenols and theaflavins are present in prostate tissue of humans and mice after green and black tea consumption. *J Nutr.* 2006;136:1839–43.
- Umegaki, K. & Fenech, M. (2000) Cytokinesis-block micronucleus assay in WIL2-NS cells: a sensitive system to detect chromosomal damage induced by reactive oxygen species and activated human neutrophils. *Mutagenesis* 15:261-269.