Anti iron toxicity and Antioxidant Effect of *Camellia sinensis*

D. DIVYA, V. SIVAKUMARI AND R. SATHYA PRIYA

Asian Journal of Environmental Science (June to November, 2009) Vol. 4 No. 1 : 58-61

See end of the article for authors' affiliations

Correspondence to :
V. SIVAKUMARI
Department of Environmental and Herbal Sciences, Tamil University, THNJAVUR (T.N.) INDIA

SUMMARY

The study was carried out to evaluate the anti iron toxic and antioxidant activity effect of *Camellia sinensis* on ferrous sulphate induced albino rats. The phytochemical activity of *Camellia sinensis* showed the presence of tannins, alkaloids, flavonoids, steroids, amino acids, reducing sugar and carbohydrate. SGOT (Serum Gluatamate Oxaloacetate Transminase), SGPT (Serum Gluatamate Pyruvate Transminase), Gamma Glutamyl Trasferase (GGT), MDA (malonaldehyde), SOD (Superoxide Dismutase) were also estimated. The result indicated that ethanol extract of *Camellia sinensis* to ferrous sulphate induced rats decreased the level of SGPT from iron intoxication.

Key words : *Camellia sinensis*, Antioxidant activity, Ferrous sulphate, Malonaldehyde

Iron poisoning is a common problem in all developed and many developing countries. In the United Kingdom, it accounts for 15 to 20% of all medical emergency admission to hospitals. Iron poison is one of the most common causes of childhood poisoning death (Litovitz and Manoguerra, 1992; Andrews, 1999).

Iron has several vital functions in the body as a carrier of oxygen to the tissue from the lungs, as a transport medium for electrons within cells and as an integrated part of important enzyme reaction in various tissues. The main part of iron in the body is present in the red cells as hemoglobin, which is a molecule composed of 4 units, each containing one haem and protein chain (Thakerngpol *et al*., 1990; Chapman and Hall, 1995).

The normal level of iron in male is 50-160 mg/dl and the value of iron in female is 45-150 mg/dl. If the iron content is deficient, it leads to anaemia. But if the iron is excess in body, it leads to iron toxicity, *i.e*., iron overload. Iron overload is known as hemochromatosis. The body normally absorbs less iron if its stores are full, but some individuals are poorly defended against iron toxicity. Once considered rare, iron overload has emerged as an important disorder of iron metabolism (Nichaus and Sanuelsson, 1968).

MATERIALS AND METHODS

The samples were collected from Tamil University Herbal Garden. Colleted plants were carefully examined and identified with the help of regional floras (Gamble, 1975; Mathew, 1983; Nair and Hendry, 1983; Henry *et al*., 1987). Specimens were further confirmed with reference to Herbarium sheet available in the Botanical Survey of India, Southern Circle, Coimbatore. The plant samples were collected and dried under shade. These powdered materials were used for further physio-chemical, phyto-chemical and florescent analyses. Iron in the form of ferrous sulphate (500 mg) was dissolved in distilled water and given orally to rats to develop iron toxicity. In experiment, 16 albino rats were used. The rats were divided into the following 4 groups of 4 rats each.

Group I: Normal animals received with standard feed and water to allow adlibitum

Group II: Iron treated experimental control rats. [Ferrous sulphate (500mg/10ml H_2O) given orally for 20 days].

Group III: Along with ferrous sulphate the ethanol extract of *Camellia sinensis* (1gm / ml) given orally for 20 days.

Group IV: Ethanolic extract of leaf (1g/ml) given orally for 20 days.

After treatment for 20 days, the animals were sacrified with ether anesthesia. The blood was collected by cardiac puncture and then centrifuged at 2000 rpm and stored under 4^{o}C until analysis. The liver homogenate was also taken for the biochemical analysis. SGOT, SGPT, GGT, MDA, SOD were estimated (Kakkar and Dar Viswanathan, 1999, Samman,

Accepted : ***March, 2009***

2001). Tannins, phlobotannins, saponin, alkaloids, flavonoids, steroids, terpenoids, reducing sugar, amino acid and carbohydrate were also estimated (Sinha, 1972; Trease and Evans, 1978; Chung, 1995).

RESULTS AND DISCUSSION

The present study was carried out to evaluate the antioxidant activity and anti iron toxic effect of *Camellia sinensis* on ferrous sulphate induced albino rats. The effectiveness of drug was screened through SGOT, SGPT, MDA, SOD, catalase and gamma glutamyl transferase. The observations were made on different groups of experimental animals. The results obtained were compared with the control groups. The results were expressed as mean ± standard deviation.

The SGOT level in group I was 84.57±2.53 (IU/L). group II rats showed a significant increase in the level of SGOT when compared to group I rats. Decreased SGOT level was observed in group III. This was lower than group II. The level of SGOT in group IV was similar to that of group I. Administration of ethanol extract of *Camellia sinensis* to ferrous sulphate induced rats attained near normal level (Table 1).

In group I rats, the level of SGPT was 65.65±1.12(IU/L). Group II rats showed a significant increase in the level of serum glutamate pyruvate transminase when compared to group I rats. In group III rats, decreased level of SGPT was observed when compared to group II rats. In group IV rats, a significant decrease in the level of SGPT was observed when compared to group III rats. The data revealed that the level of SGPT in group IV was near to normal. Administration of ethanol extract of *Camellia sinensis* to ferrous sulphate induced rats decreased the level of SGPT from iron intoxication (Table 2).

Table 1 : The level of serum glutamate oxaloacetate transaminase in control and treated rats

Sr. No.	Groups	SGOT (IU/L)
1.	Group – I	84.57±2.53
2.	Group – II	106.50 ± 1.15
3.	Group – III	85.06 ±1.65
4.	Group – IV	83.80 ±5.45

Values expressed as mean ± S.D for 4 animals in each group

In group I rats, the level of GGT was 50.6±1.35 (IU/L). Group II rats showed a significant increase in the level of GGT when compared to group I rats. When compared to group II rats, the level of GGT significantly decreased in group III rats. The level of GGT in group IV rats was 48.7 ±0.78 which was near to the level in group I. From the data in Table 3 it was revealed that the administration of *Camellia sinensis* normalized the SGPT level in group III (Table 3).

Table 2 : The level of serum glutamate pyruvate transaminase in control and treated rats

Sr. No.	Groups	GGT (IU/L)
1.	Group – I	65.65±1.12
2.	Group – II	95.67±1.35
3.	Group – III	71±0.15±1.25
4.	Group – IV	70.1±1.25

Values expressed as mean ± S.D for 4 animals in each group

The level of malonaldehyde in group II rat wass 3.5±0.76 Nanomoles / mg and showed a significant increase in the level of malonaldehyde when compared to group I rats. In rats administrated with ferrous sulphate and ethanolic extract of *Camellia sinensis*, the level of malonaldehyde significantly decreased. The level in group IV rats was near to the level of group I. Ingestion of ethanolic extract of *Camellia sinensis* decreased the level of malonaldehyde (Table 4).

Table 3 : The level of gamma glutamate transaminase in control and treated rats

Sr. No.	Groups	SGOT (IU/L)
1.	Group – I	50.6±1.35
2.	Group – II	85.6±0.35
3.	Group – III	60.7±0.43
4.	Group – IV	48.7±0.78

Values expressed as mean ± S.D for 4 animals in each group

The activity of SOD in group II was observed as 3.12±0.20 (mm/mg) which was lower when compared to the level of 6.43±0.34 (mm/mg) observed in group I. Group III rats showed the increased activity of SOD (5.80 mm/mg) which was higher when compared to group II (3.12 mm/mg). In group IV the activity of SOD was near to group I. *Camellia sinensis* restored the activity of SOD is group III animals which intoxicated with ferrous sulphate (Table 5).

Table 4 : The level of malonaldehyde in control and treated rats

Sr. No.	Groups	Malonaldehyde (Nanomoldes/mg)
1.	Group – I	1.50±0.07
2.	Group – II	3.5±0.76
3.	Group – III	2.0±1.15
4.	Group – IV	1.75±0.65

Values expressed as mean ± S.D for 4 animals in each group

The activity of catalase in group II wass 38.65± 4.54 which was lower than group I. When compared to group

Table 5 : The activity of superoxide in control and treated rats

Sr. No.	Groups	SOD (μm/mg)
1.	Group – I	6.43±0.34
2.	Group – II	3.12±0.20
3.	Group – III	5.80±0.78
4.	Group – IV	6.75±0.78

Values expressed as mean ± S.D for 4 animals in each group

II rats, the activity of catalase significantly increased in group III rats. In group IV rats there was significantly increase in the activity of catalase when compared to group III rats. This revealed that group IV values were near to normal. The administration of *Camellia sinensis* restored the activity of catalase in group IV (Table 6).

Table 6 : The activity of catalase in control and treated rats

Sr. No.	Groups	Catalase (μm/mg)
1.	Group – I	58.3±2.91
2.	Group – II	38.65±4.54
3.	Group – III	46.04±6.38
4.	Group – IV	59.4±3.5

Values expressed as mean ± S.D for 4 animals in each group

In the phytochemical study, qualitative analysis of various constituents in *Camellia sinensis* was made (Table 7). The tannins, saponin, alkaloids, flavonoids, steroids, terpenoids, reducing sugar, amino acid and carbohydrate were present in the *Camellia sinensis* while phlobotannins were absent (Table 7).

Iron overload is most often diagnosed when tissue damage occurs, especially in iron – storing organs, such as liver. Iron toxicity is not always due to an increase in dietary iron with acute iron poisoning; much of the damage to the gastrointestinal tract and liver may be a result of high localized iron concentration and free radical production, leading to hepatotoxcity via lipid peroxidation and the destruction of the hepatic mitochondria. It is present in tissues such as heart, liver, skeletal muscle, kidney, brain, etc. The elevation of SGOT could be due to toxic injury in the liver cells by iron overload (Chatterjea, 1999; Jung, 2001).

Table 7 : Qualitative analysis of *Camellia sinensis*

Sr. No.	Constituents	*Camellia sinensis*
1.	Tannins	+
2.	Phlobatannins	-
3.	Saponin	+
4.	Allkaloids	+
5.	Flavonoids	+
6.	Steroids	+
7.	Terpenoids	+
8.	Reducing Sugar	+
9.	Amino acids	+
10.	Carbohydrate	+

+ Presence of constituents
- Absence of constituents

The liver is the major site of iron storage and as such is susceptible to damage from iron toxicity. Iron deposits are in the kupffer cells as well as in hepatocytes. This causes hepato cellular injury as indicated by a massive increase in the activity of SGOT. The minimal elevation of SGOT also may be found in association with biliary tract obstruction. Higher level suggests the development of cholangitis with resultant hepatic cell necrosis (Pryor and Stanley, 1975; Farmer and Davoine, 2007).

SGPT is found exclusively in the cytosol of the hepatocytes. Presence of SGPT in serum is directly related to membrane damage. SGPT is the cytosolic enzyme, which is more specific for liver than SGOT. Clinical applications of SGPT assay are confined mainly to elevation of hepatic disorders. Hepatocellular disorder causes higher elevation of this enzyme (Moore and Roberts, 1998; Anderson, 2001). This is due to administration of ethanolic extract of *Camellia sinensis*. By this way it prevents the damage of liver cell and decreases the level of SGPT (Katiyar and Elmets, 2001; Garrow *et al*., 2000). So, finally if the principle of herbal treatment is advocated to iron toxicity, then it will definitely be a great boon and contribution to the suffering humanity.

Authors' affiliations

D. DIVYA AND R. SATHYA PRIYA, Rabiammal Ahamed Maideen College for Women, TIRUVARUR (T.N.) INDIA

REFERENCES

Anderson, S. (2001). Green tea catechins partially protect DNA from OH radial – induced strand breaks and base damage through fast chemical repairs of DNA radicals. *Carcinogenesis*. **23**(8): 1189 – 1193.

Andrews, N.C. (1999). Disorders of iron metabolism. *N. Engl. J. Med*., **3** (26): 41-44.

Chapman, A. and Hall, R. (1995). Iron nutritional and physiological significance. *The British Nutrition Foundation*. 51-56pp.

Chatterjea, M.N. (1999). *Text book of medicinal biochemistry,* second Ed. Jaypee Brothers Medical Publishers. New Delhi. 830 pp.

Chung, R.T. (1995). Complication of chronic liver disease care. *Clin. Microbiol*., **11** : 431.

Farmer, E.E. and Davoine, C. (2007). Reactive electrophile species. *Curr. Opin. Plant Bio1*., **10**(4):380-386

Gamble, J.S. (1975). *Flora of the Presidency of Madras,* Botanical Survey of India. Vol. 1-3.

Garrow, J.S., James, W.P.T. and Ralph, A. (2000). *Human nutrition and dietetics*. 10th Edition.

Henry, A.N., Kumari, G.R. and Chitra, V. (1987). *Flora of Tamil Nadu, India.* Series I, Vol II. BSI Southern circle, Coimbatore, India.

Jung, A. (2001). A major component of green tea inhibits tumor growth by inhibiting VEGF induction in human colon carcinoma cells. *Br. J. cancer,* **84** (6): 844 – 850.

Kakkar, P. and Dar Viswanathan, P.N. (1999). Amoudified spectro photometric assay of superoxide dismutase (SOD). *Indian J. Biochem. Biophy*., 130-132.

Katiyar, S.K. and Elmets, C.A. (2001). Greentea polyphenolic antioxidant and skin photo protection. *Internat. J. Oncol.*, **18** (6): 1307 – 1313.

Litovitz, T. and Manoguerra, A. (1992). Comparison of pediatric poisoning hazards: An analysis of 3.8 million exposure incidents. *Pediatrics*, **89**: 12-15.

Mathew, K.C. (1983). *The flora of the Tamil Nadu Carnatic*. Rapinet Herbarium. Tiruhirappalli.

Moore, K. and Roberts, L. J. (1998). Measurement of lipid peroxidation. *Free Radic. Res*., **28** (6) : 659-71.

Nair, N.C. and Hendry, A.N. (1983). *Flora of Tamil Nadu, India*. Vol. I. Series I. BSI, Southeren circle, Coimbatore, India.

Nichaus, W.G. and Sanuelsson, B. (1968). Formation of malonaldehyde from phospholipids arachidonate during microsomal lipid peroxidation *Eur. J. Biochem.*, **6**: 126-130

Pryor, W.A. and Stanley, I.P. (1975). A suggested mechanism for the production of malonaldehyde during the antioxidation of PUFA, Non enzymatic production of prostoglandins endoperoxides during autooxidation. *J. Org. Chem.*, **40**(24): 3615-3617.

Samman, S. (2001). Green tea extract added to foods reduces nonheme – iron absorption. *AM. J. Clin. Nutr*., **73**(3): 607 – 612.

Sinha, A.K. (1972). Colorimetric assay of catalase. *Anal. Biochem*., **47**:389-394.

Thakerngpol, K., Fucharoen, S. and Boonyaphipat, P. (1990). Liver injury due to iron overload. *N. Engl, J. Med*., **3** (26): 49-53.

Trease, H.S. and Evans, H.C. (1978). *Text book of Pharmacognosy*. Nineth Edition, Bailiar Zindall and Co., London. 25-54pp.