



# GLUTATHIONE PEROXIDASE ACTIVITIES IN LEUKEMIA, LIVER AND COLON CANCER PATIENTS.

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## Abstract

Plasma level of glutathione peroxidase(GP<sub>x</sub>) in fifty(50) patients attending University teaching hospital and Federal Medical Centre in Ekiti State suffering from leukemia, liver and colon cancers were measured using high sensitive enzyme linked Immunosorbent assay(ELISA). The result shows a significant decrease ( $P < 0.05$ ) in the plasma GP<sub>x</sub> level of all the cancer types; leukemia ( $10.0 \pm 9.65^a$ ), liver cancer( $33.2 \pm 5.92^b$ ) and colon cancer( $37.0 \pm 9.65^a$ ) when compared with the control subjects( $135.0 \pm 10.8^c$ ). Though, antioxidant enzyme depletion had been implicated with increased oxidative damage. Thus, low levels of GP<sub>x</sub> in the cancer patients as observed from this study could also be due to high level of oxidative stress in cancer cells.

Key words: glutathione peroxidase, antioxidant, plasma, leukemia, liver and colon cancer.

## Introduction

Glutathione peroxidase(GP<sub>x</sub>) is the general name of an enzyme family with peroxidase activity whose main biological role is to protect the organism from oxidative damage. Its biochemical

function is to reduce hydroperoxides to their corresponding alcohols and to reduce free hydrogen peroxide to water. (Bakan *et al*, 2003).

Hydrogen peroxide is a harmful by- products of many normal metabolic processes, which must be quickly converted into other less dangerous substances to prevent damage. In this regard, GPx is frequently used by cells to rapidly catalyze the decomposition of hydrogen peroxide ( $H_2O_2$ ) into less reactive gases, that is, oxygen and water molecules. Glutathione peroxidase (GPx) has seen distinguished to be only human enzyme known to require the element selenium for its activity which also removes  $H_2O_2$  by using reduced glutathione (GSH) (Pujari *et al* , 2011).

Selenium plays a vital role in GPx antioxidant activity which exerts cancer-preventive events and anti-tumorigenic activity (Rotruck *et al*, 2003) by protecting mammalian epithelial cells from oxidative DNA damage (Hayes and Mclellan, 2009), inhibiting initiation phase of carcinogenesis, stimulated DNA repair and regulating apoptosis(Hayes and Mclellan, 2009). Thus many of the pathological consequences of selenium deficiency are associated with oxidative damage to tissues.

The manipulation of GPx activities of changing selenium levels in diet and by molecular over expression or knock out techniques indicate they have more subtle functions(Pujari *et al*, 2011). When selenoperoxidase activity is manipulated by dietary means, there is coordinate regulation of several other selenium containing proteins, notably the iodothyronine deoiochinases and the thioredoxin reductases. These other selenoproteins can have a profound effect upon metabolism and will greatly alter the context in which changes in selenoperoxidase activity are expressed (Mills, 2007).

Thus, the enzymes levels of GPx is altered to considerable extent in various diseased states exhibiting either elevation or depletion in its activity. (Vasudevan *et al*,2005). Hence, this study measures the enzyme levels in leukemia, a general term that indicate different cancers that occur in the bone marrow,which is responsible for supplying blood cells.

Liver cancer- cancer which originates in the liver and colon cancer or cancer that starts in the colon or rectum, since 90.9% of deaths as a result of cancer has been associated to common environmental factors which include tobacco, diet, obesity and environmental pollution. (Anand *et al*,2008)

## Materials and methods

A total number of 50 patients was involved in this study. 5ml of Venous blood was collected from the patients and normal subjects. The collected blood samples were dispensed into lithium heparine bottles and was centrifuged for fifteen minutes at 1000g. The plasma obtained was assayed immediately using Sandwich-ELISA method as described by Rotruck et al (1973)

## RESULTS

Table 1.0

Showing GPx activity( $\mu$ L) in leukemia,liver and colon cancer patients with the control subjects

Leukemia	Liver cancer	Colon	Control
10.0 $\pm$ 9.65 <sup>a</sup>	33.2 $\pm$ 5.92 <sup>b</sup>	37.0 $\pm$ 9.65 <sup>a</sup>	135.0 $\pm$ 10.8 <sup>c</sup>

P< 0.05, superscript a,b and c indicate significant difference.

## Discussion

Table 1.0 shows the mean plasma GPx activities in Leukemia, liver and colon cancer patients (10.0 $\pm$  9.65<sup>a</sup>, 33.2 $\pm$ 5.92<sup>b</sup> and 37.0 $\pm$ 9.65<sup>a</sup>) respectively as compared with the control subjects (135.0 $\pm$ 10.8c)

The results indicate a significant decrease (P<0.005), in the plasma activity of GPx in all the cancer patients when compared to the compared to the control subjects. The significant decrease observed in the GPx level in all the cancer type could be as a result of increased production of Reactive Oxygen Species (ROS) in the system (Bakan *et al*, 2003).

However, GPx activity in leukemia patient was found to be lower than other cancer types. This could be due persistent oxidative stress occurs in leukemia more than in other cancer types leading to an exponential decrease in GPx activity.

Thus, this study may also indicate a possible link between decreased antioxidants and increased levels of cells alteration due to oxidative damage, explaining why haemolytic anemia occurs in leukemia.

## CONCLUSION

This study shows a strong association between cancer cells and oxidative stress, which causes a marked decrease in the activity of the antioxidant enzyme (GPx). Increased oxidative stress (as observed in cancer cells), arises due to overproduction of reactive oxygen species (ROS), which is a contributing factor to the decreased activity of antioxidant enzymes in the body.

## References

- Anand P., Kunnumakkara A.B., Kunnumakara A.B., Sundaram C., Harikumar K.B., Tharakan S.T., Lai O.S., Sung B. and Aggarwal B.B. (2008). "Cancer is a preventable disease that requires major lifestyle changes". *Pharm. Res.* 25 (9): 2097–116.
- Bakan N., Taysi S., Yilmaz O., Bakan E., Kuskay S., Uzun N., and Gungdogdu M. (2003). Glutathione peroxidase, glutathione reductase, Cu-Zn superoxide dismutase activities, nitric oxide and malondialdehyde concentrations in serum of patients with chronic lymphocytic leukemia. *Clin-Chim-Acta.* Dec; 338(1-2):143-49.
- Hays J.D and Mclellan L.I. (2009). Glutathione and glutathione-dependent enzymes represent a cor-ordinately regulated defence against oxidative stress. *Free Radic. Res.* 31: 273-300.
- Mills G.C., (2007). Hemodlobin catabolism. Glutathione peroxidase: an erythrocyte enzyme which protects hemoglobin from oxidative breakdown. *J. Biol. Chem.* 229:189-19.
- Pujari K.N., Aruna Kulkarni. V.B Tuljapurkar, M.K., Suryawanshi R.M and Joshi A.M (2011). Lipid peroxidation and antioxidant enzymes in chronic leukemia. *Spectrum: Journal of Medical Research.* :(1&2) :60-63.
- Rotruck, J.T., Ashton, K. E., Becker, A. E., Holland, J.F., and Reamy B. V (2003): Selenium: biochemical role as a component of glutathione peroxidase. *Science*, 5; 334–36.
- Rotruck, J.T., Pope, A.L., Ganther, H.E., Swanson, A.B., Hafeman, D.G., and Hoekstra, W.G. (1973): Selenium: biochemical role as a component of glutathione peroxidase. *Science*, 179: 588–590.
- Vasudevan D.M., Wenger A. and Sreekumari S. (2005). Free radicals and antioxidants: Textbook of Biochemistry. Jaypee 4th Ed. Page 338-42.