

## Physiological Evaluation to the Antioxidant Efficacy of Quercetin Against Ascorbic Acid in Rats

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### Abstract:

Fortyeight male rats were adopted to estate the antioxidant efficacy difference between the flavonoid Quercetin against the vitamin Ascorbic acid when these animals are being subjected to the potent oxidant agent; the hydrogen peroxide. The results elucidated that, H<sub>2</sub>O<sub>2</sub> could cause a significant elevations in malondialdehyde, glutathione peroxidase, catalase, superoxide dismutase, aminotransferases, alkaline phosphatase, total cholesterol, triglycerides, low and very low density lipoproteins. The Quercetin and Ascorbic Acid, could cause significant declinations and improvement of the above mentioned parameters, with approximately little supremacy for Quercetin, as it is seen in details of the results.

### Introduction

Quercetin is a type of flavonoids known for its antioxidant capacity in diets (1). Quercetin can be existed in many vegetables and fruits like onions, beans, broccoli and apples (2). Quercetin antioxidant capacity was found to be four times that of vitamin E and it is the most vast spread antioxidant in the nature (3). Quercetin was documented to be effective as anticancer, anti-inflammatory, and immune booster (4). Ascorbic acid; Vitamin C, is a well-known for being potent antioxidant which present in many citrus fruits beside the vegetables and of a great value for different body systems (5). Oxidative stress is being induced experimentally exploiting the oxidant capacity of hydrogen peroxide; the H<sub>2</sub>O<sub>2</sub>, where it is a potent oxidant agent known for its damaging ability on cells via the generating of free radicals (6). This research study was accomplished to estate which of the two antioxidants, the quercetin and the ascorbic acid, is more potent as an antioxidant against the oxidative stress caused by hydrogen peroxide.

### Material and Methods

#### 1- Groups of animals

Forty eight adult male rats, of weights 200 – 250 grams, were adopted to perform this experimental study. The animals were randomly allocated into four groups of twelve rats to each after being acclimatized in typical conditions for two weeks. The groups of the animals were:

1- Control, animals were maintained on a standard food for one month.



2- First treatment; (T1), animals were maintained on a standard food plus 0.1% of hydrogen peroxide  $H_2O_2$  in drink water for one month.

3- Second treatment; (T2), animals were maintained on a standard food enriched with 80mg/kg/diet of Quercetin plus 0.1% of hydrogen peroxide  $H_2O_2$  in drink water for one month.

4- Third treatment; (T3), animals were maintained on a standard food enriched with 400mg/kg/diet of Ascorbic acid plus 0.1% of hydrogen peroxide  $H_2O_2$  in drink water for one month. The doses of  $H_2O_2$ , Quercetin and Ascorbic acid were chosen depending upon previous studies of (7, 8, 9 and 10) respectively.

## **2- Blood samples collection**

To obtain sera, blood samples were put in gel tubes after being collected from the heart directly while the rats were being anesthetized by chloroform at the end of experimental period.

## **3- Tests of the study**

A special device; chemical autolyzer, DONGI; (DONGI, 120; ITALIA), was exploited to accomplish and obtain results of all the required test parameters for this study.

## **4- Statistics**

ANOVA; one way test, was used to get the least significant difference (LSD) among groups. The numbers in the tables; refers to the means  $\pm$  standard deviations, and the letters upon the numbers, refer to the significant differences at ( $P \leq 0.05$ ).

## **Results and Discussion**

One can readily see, table 1; that the hydrogen peroxide  $H_2O_2$  could cause significant elevations of the malondialdehyde; MDA, glutathione peroxidase; GPx, superoxide dismutase; SOD, and the catalase CAT comparing with the control and the other treatment groups. Quercetin caused significant declination of MDA comparing with the  $H_2O_2$  group, and the same was true for Ascorbic Acid, but the latter was significantly higher than the control and the Quercetin groups. GPx, was significantly declined by both the Quercetin and the Ascorbic Acid comparing with the  $H_2O_2$  group without a significant difference with the control. The SOD, was significantly declined by both the Quercetin and the Ascorbic Acid, but, the Quercetin was significantly higher than both the control and the Ascorbic Acid groups. The CAT, was significantly declined by both the Quercetin and the Ascorbic Acid without significant difference comparing with the control.

The hepatic aminotransferases; the alanine aminotransferase; ALT, and the aspartate aminotransferase; AST, beside the alkaline phosphatase; ALP, were significantly elevated by the  $H_2O_2$ , as it is clearly obvious, table 2. Both the Quercetin and the Ascorbic Acid caused significant declination of the AST and the ALT, but this declination was still higher than that



of the control significantly. The ALP was significantly declined by both the Quercetin and the Ascorbic Acid, comparing with H<sub>2</sub>O<sub>2</sub>, but without significance comparing with the control.

All the lipid profile parameters, were significantly elevated by the H<sub>2</sub>O<sub>2</sub> comparing with all the groups, as in table 3. The Quercetin and the Ascorbic Acid, were both capable of causing significant declination of the lipid profile parameters to the limit of no significance with the control.

The findings in this article, coincide with those of (11, 12 and 13) considering the antioxidant properties of Quercetin and Ascorbic Acid. The hydrogen peroxide; H<sub>2</sub>O<sub>2</sub>, is famous among scientific fields by its properties as a strong oxidant agent, causing deleterious vast devastating effects on cellular extra and infrastructures with liberating vast range of free radicals leading for elevated values of the biomarker intermediate compound of lipid annihilation; the MDA besides clear elevations of other markers like ALT, AST, ALP, and lipid profile (14 and 15). The ameliorating effects of both Ascorbic Acid and Quercetin can be explained as the following concerns. Quercetin plays a potent role as antioxidant throughout inhibition to the oxygen reactive species. By this, Quercetin help the cells as a protector (16). Quercetin was reported to be a scavenger against these radicals, pertaining to its high antioxidant properties, mentioned to be higher than these of Ascorbic Acid and vitamin E (17, 18). Quercetin is also documented to be a protector against lipid peroxidation, therefore; it provide a shield like role against elevation of lipid profile (19). Ascorbic Acid, on the other hand, has a very potent properties as an antioxidant, providing it with great capability of protecting the cells and the body as a whole against damages caused by harmful radicals or substances, and it is of great potency in modulating and promoting immunity, lipid profile and other systemic parameters (20, 21, 22 , 23 and 24 ).

**Table 1. Role of Quercetin and Ascorbic Acid on oxidative status.**

Groups	MDA (µm / L)	GPx (µm / L)	SOD (µm / L)	CAT (IU/ml)
Control	c 1.7 ±0.4	b 82.6 ±2.2	d 31.7 ±1.5	b 2.3 ±0.5
T1 (H <sub>2</sub> O <sub>2</sub> )	a 5.3 ±0.7	a 128.5 ± 2	a 71.3 ±1.5	a 6.7 ±0.7
T2 (H <sub>2</sub> O <sub>2</sub> +Quercetin)	c 1.8 ±0.3	b 83.7 ±2.7	b 37.1 ±1.1	b 2.4 ±0.5
T3 (H <sub>2</sub> O <sub>2</sub> +Ascorbic Acid)	b 2.3 ±0.4	b 84.3 ± 2	c 35.8 ±1.1	b 2.8 ±0.6
LSD	0.5	44.2	1.3	3.9

**Table 2. Role of Quercetin and Ascorbic Acid on hepatic enzymes.**



Groups		AST (U/L)		ALT (U/L)		ALP (U/L)
Control	d	60.6 ±1.3	c	29.4 ±1.2	b	113.1 ±3.6
T1 (H <sub>2</sub> O <sub>2</sub> )	a	84.4 ±1.9	a	51.8 ±5.7	a	137.5 ±3.4
T2 (H <sub>2</sub> O <sub>2</sub> +Quercetin)	c	65.6 ±2.2	b	32.5 ±3.3	b	116.6 ±3.0
T3 (H <sub>2</sub> O <sub>2</sub> +Ascorbic Acid)	b	70.3 ±6.1	b	33.4 ±4.1	b	117.1 ±1.6
LSD		4.7		3.1		20.4

**Table 3. Role of Quercetin and Ascorbic Acid on lipid profile.**

Groups		TC (mg/dl)		TGs (mg/dl)		HDL (mg/dl)		LDL (mg/dl)		VLDL (mg/dl)
Control	c	74.2 ±4.05	b	58.4 ±4.9	a	42.3 ±2.1	b	20.1 ±2.8	b	11.3 ±1.4
T1 (H <sub>2</sub> O <sub>2</sub> )	a	93.1 ±6.5	a	73.9 ±3.3	c	20.8 ±2.4	a	57.5 ±8.0	a	16.8 ±0.9
T2 (H <sub>2</sub> O <sub>2</sub> +Quercetin)	b	82.05 ±5.8	b	62.3 ±2.6	b	35.8 ±3.4	b	31.7 ±6.1	b	11.4 ±1.5
T3 (H <sub>2</sub> O <sub>2</sub> +Ascorbic Acid)	b	84.1 ±6.6	b	60.05 ±5.1	b	33.8 ±5.1	b	30.9 ±10.8	b	12.4 ±1.4
LSD		7.85		11.6		6.5		25.8		4.4

### Conclusion

Ascorbic Acid and Quercetin have a potent antioxidant activity with a proximately similar potency, with a little supremacy to the Quercetin.

### References



- 1- Paolillo R, Carratelli C, Rizzo A. (2011). Effect of resveratrol and quercetin in experimental infection by Salmonella enteric serovar Typhimurium. *Int Immunopharmacol*; 11: 149-156.
- 2- Pandey KB, Rizvi S. (2009). Current understanding of dietary polyphenols and their role in health and disease. *Curr Nutr Food Sci*; 5: 249-263.
- 3- Neha P, Sangai V. (2012). Quercetin ameliorates bisphenol A-induced toxicity in mice. *Acta Poloniae Pharmaceutica – Drug Research*, Vol. 69 No. 3 pp. 557-563.
- 4- Tieppo J, Vercelino R, Dias S, Silva F, Silveira R, Marroni A. (2007). Evaluation of the protective effects of quercetin in the hepatopulmonary syndrome. *Food Chem Toxicol*; 45: 1140-1146.
- 5- Carr C, Frei B. (1991). Towards a new recommended dietary allowance for vitamin C based on antioxidant and health effects in humans. *Am J Pathol*, 18:499.
- 6- Aruoma O. (1998). Free radicals, oxidative stress and antioxidants in human health and disease. *JAOCS*, 2: 199-210.
- 7- Ganie S, Haq E, Hamid A, Masood A, Zargar M. (2011). Long dose exposure of hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>) in albino rats and effect of Podophyllum hexandrum on oxidative stress. *European Review for Medical and Pharmacological Sciences*, 15: 906-915.
- 8- Al-Uboody WS, Al-Diwan MA. (2015). Ameliorating and protective role of quercetin against o-anisidine toxicity on some reproductive aspects of laboratory male rats (*Rattus norvegicus*). *Bas.J.Vet.Res*, 14(1): 250-261.
- 9- Al-Uboody WS, Al-Diwan MA. (2015). Study the protective role of quercetin against o-anisidine toxicity on some hematological parameters of laboratory male rats (*Rattus norvegicus*). *Bas.J.Vet.Res*, 14(1): 262-273.
- 10- Al-Uboody WSH, Ishraq JH, Al-Diwan MA. (2011). Effect of acute exposure of lead acetates on the morphology of liver and kidney of mice (*Mus musculus*) and the role of vitamin C as a repairing agent. *Bas.J.Vet.Res*, 10(1): 43-55.
- 11- Hashim WS, Mshary GS, Al-Samawy ERM, Waad SK. (2019). Physiological Study about Rosuvastatin and Lovastatin as Compared with Quercetin in Rats (*Rattus norvegicus*). *Journal of Global Pharma Technology*, Vol. 11, Issue 05 (Suppl.), 28-32.
- 12- Jabbar AA, Al Uboody WSH, Waad SK, Abbas MQ. (2018). Physiological Study about the Hematological Effects of Rosuvastatin and Lovastatin as Compared with Sophoretin in Rats (*Rattus Norvegicus*). *Journal of Global Pharma Technology*, 10 (08):314-318.
- 13- Wu L, Xu W, Li H, Dong B, et al. (2022). Vitamin C Attenuates Oxidative Stress, Inflammation, and Apoptosis Induced by Acute Hypoxia through the Nrf2/Keap1 Signaling Pathway in Gibel Carp (*Carassius gibelio*). *Antioxidants*, 11(5), 935; <https://doi.org/10.3390/antiox11050935>.
- 14- AC MF, ME H, R M. (1984). Cell killing and DNA damage by hydrogen peroxide are mediated by intracellular iron. *Biochem J*, 218: 273-275.
- 15- JA I, SM C, S L. (1988). Toxic DNA damage by hydrogen peroxide through the Fenton reaction in vivo and in vitro. *Science*, 240: 640-642.



16. Fiorani M, Accorsi A, Cantoni O. (2003). Human red blood cells as a natural flavonoid reservoir. *Free Radic. Res*, 37:1331-8.
17. Jamshidzadeh A, Mehrabadi RA. (2010). Protective effect of quercetin on oxidative stress in glucose-6-phosphate dehydrogenase-deficient erythrocytes in vitro. *Iranian Journal of Pharmaceutical Research*, 9 (2): 169-175.
18. Haraguchi H, Saito T, Ishikawa H, et al. (1996) Antiperoxidative components in *Thymus vulgaris*. *Planta Med.*, 62:217-21.
19. Abd El-Baky EA. (2011). Quercetin protective action on oxidative stress, sorbitol, insulin resistance and cell function in experimental diabetic rats. *IJPSR. II (II)*:11-18.
- 20- Kumari J, Sahoo PK. (2005). High dietary vitamin C affects growth, non-specific immune responses and disease resistance in asian catfish, *Clarias batrachus*. *Mol. Cell. Biochem*, 280, 25–33.
- 21- Ming J, Xie J, Xu P, Ge X, Liu W, Ye J. (2012). Effects of emodin and vitamin C on growth performance, biochemical parameters and two HSP70s mRNA expression of Wuchang bream (*Megalobrama amblycephala* Yih) under high temperature stress. *Fish Shellfish Immunol*, 32, 651–661.
- 22- Ai QH, Mai KS, Tan BP, Xu W, Zhang WB, Ma HM, Liufu ZG. (2006). Effects of dietary vitamin C on survival, growth, and immunity of large yellow croaker, *Pseudosciaena crocea*. *Aquaculture*, 261, 327–336.
- 23- Ahmed, Q., Rahim, S., & Hameed, A. (2022). The effect of hydroxytyrosol (hxt) and local olive oil (loo) on oxidative stress and histopathological changes in the liver resulting from induced hyperlipidaemia in male rats. *International Journal of Medical Sciences*, 5(1), 43-54.
- 24- Abdulaali Azeez, A., Mohammed Mustafa, E., & Mahrouf Ali Shoshin, O. (2021). Effect of *Camellia sinensis* on Fat Peroxidation and Ox-LDL in Rats. *Archives of Razi Institute*, 76(4), 949-955.

