

Effects of Brazilin and Haematoxylin on the Lipidperoxidation in the Rat Liver Mitochondria

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The major sites of lipidperoxidation-damage within the cell are at biomembranes, especially those of subcellular organells such as mitochondria and microsomes whose membranes contain relatively large amount of polyunsaturated fatty acids.¹⁾ Mitochondria are the power plants of eukaryotic cells. Hence their damage by lipidperoxidation can profoundly affect cellular function.²⁾

Lipidperoxidation correlates with swelling and finally with lysis and disintegration of the mitochondria. Lipidperoxidation in mitochondria can be initiated by redox-agents such as ferrous iron, ascorbic acid, glutathion and some hepatotoxic chemicals such as CCl₄, ethanol etc.^{3) 4)} In our unpublished study we confirmed that brazilin and haematoxylin prevent the hepatic damage induced by CCl₄.

In the view of this connection we started to investigate the protecting effects of brazilin and haematoxylin on the lipidperoxidation in the rat liver mitochondria.

Female Sprague-Dawley rats weighing 200±20g were obtained from the Experimental Animal Breeding Center of the Seoul National University. Laboratory chow of Sam-Yang Industry LTD. were used for the experiment. SD-rats

Table I: Grouping and treatment of rats.

Group	No. of animal	Treatment
Control-group	5	only saline
CCl ₄ -group	5	0.5ml CCl ₄ /100g bd. wt
Ethanol-group	5	1.5ml ethanol/100g bt. wt
Brazilin-CCl ₄ -group	5	103mg brazilin/kg bd. wt +0.5ml CCl ₄ /100g bt. wt
Brazilin-ethanol-group	5	103mg brazilin/kg bd. wt +1.5 ethanol/100g bd. wt
Haematoxylin-CCl ₄ -group	5	109mg haematoxylin/kg bd. wt +0.5ml CCl ₄ /100g bd. wt
Haematoxylin-ethanol-group	5	109mg haematoxylin/kg bd. wt +1.5ml ethanol/100g bd. wt

* bd. wt=body weight

bred in the same condition were adapted in the experimental neighoring environment for a week. Thirty-five healthy female Sprague-Dawley rats were grouped and treated as shown in Table I.

Brazilin and haematoxylin were suspended in saline and administered intraperitoneally daily for 2 days. After the rats were fasted for 3 hours, ethanol and carbon tetrachloride were administered orally. An equivalent volume of 0.9% saline was administered to the control group.

Preparation of mitochondrial fraction and assay of malondialdehyde(MDA) contents in mitochondria were measured as previously described.⁵⁾ The statistical significance of the diffe-

Table II: Antilipidperoxidation effects of brazilin and haematoxylin in the mitochondria of CCl₄ and ethanol treated rat livers.

Group	MDA-contents in the rat liver mitochondria (\bar{x} moles/mg protein)	Inhibition(%)
Control group	1.176 \pm 0.054	—
CCl ₄ group	4.307 \pm 0.156	—
Brazilin-CCl ₄ group	2.179 \pm 0.179*	68
Haematoxylin-CCl ₄ group	1.745 \pm 0.102*	82
Ethanol group	2.903 \pm 0.131	—
Brazilin-ethanol group	1.624 \pm 0.097*	74
Haematoxylin-ethanol group	1.199 \pm 0.116*	98

Statistical significance: * $p < 0.001$

rences between values of treated animals versus control animals was evaluated by the student's T-test.

As shown in Table II. Brazilin and haematoxylin showed marked suppressing effects on the mitochondrial lipidperoxidation induced by CCl₄ and ethanol. In the CCl₄-treated group brazilin and haematoxylin exhibited 68% and 82% inhibitory effects on the lipidperoxidation respectively compared to 74% and 98% inhibitory effects in the ethanol treated group.

Haematoxylin represented more potential inhibitory effect on the mitochondrial lipidperoxidation induced by both CCl₄ and Ethanol than brazilin. This more potential antiperoxidation-activity of haematoxylin as compared with brazilin seems to be due to an additional hydroxyl group in catechol orientation but the underlying mechanism of activity and structure-activity-relationship should be elucidated by further studies. In conclusion these inhibitory

effects of brazilin and haematoxylin suggest a possibility to be applied as antilipidperoxidants.

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