Antioxidant effect of curcumin in selenium induced cataract of Wistar rats

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Wistar rat pups treated with curcumin, a natural constituent of Curcuma longa before being administered with selenium showed no opacities in the lens. The lipid peroxidation, xanthine oxidase enzyme levels in the lenses of curcumin and selenium co-treated animals were significantly less when compared to selenium treated animals. The superoxide dismutase and catalase enzyme activities of curcumin and selenium co-treated animal lenses showed an enhancement. Curcumin co-treatment seems to prevent oxidative damage and found to delay the development of cataract.

Keywords: Antioxidant, Curcumin, Selenium toxicity, Cataract, Lipid peroxidation, Xanthine oxidase, Superoxide dismutase, Catalase.

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Curcumin is the colouring principle of turmeric a fleshly rhizome (Curcuma longa) used as a spice in Indian Cuisine. Curcumin is anti-inflammatory and anti-oxidative in nature. Blind staggers has been reported in animals that eat a limited number of selenium accumulator plants over a period of weeks or months, the affected animals had impaired vision and other pathological conditions like laboured breathing, abnormal movement and diarrhea. Selenium acts as a pro-oxidant generating free radicals. Selenium cataract is an animal model of cataract produced in suckling rat pups. Selenium (30 μM) administration to 10 days old Wistar rat pups causes cataracts. In the present investigation the antioxidative nature of curcumin especially with reference to its delaying property in selenium induced cataractous lenses of Wistar strain albino male rats was studied.

Materials and Methods

Healthy Wistar strain albino mother rats with 2-3 day old pups were procured from National Infra Structural Facility for Laboratory Animals, National Institute of Nutrition, Hyderabad and maintained in standard laboratory conditions. The animals were housed in screen bottomed cages in a room lit for 12 hr daily with standard fluorescent light and maintained at 22°C and provided with standard rat feed pellets (Hindustan Lever Ltd) and water, ad libitum.

Single sub-cutaneous injections of 30 μM of selenium as sodium selenite / kg body weight on day 10 post-partum and 75 mg/kg body weight of curcumin (natural extract) procured from Sigma Chemicals, were administered orally in gum acacia suspension. The animals were administered with curcumin one hour before being treated with selenium. The control animals were given sub-cutaneous injection of physiological saline and gum acacia suspension orally.

The dosage was administered between 0900-1000 hrs everyday to avoid variations that could arise due to circadian rhythms. The animals were divided into 7 groups (group-I, control, group-II 10 days selenium exposure, group-III selenium and curcumin exposure for 10 days, group-IV 20 days selenium exposure, group-V 20 days selenium and curcumin exposure, group-VI 30 days selenium exposure, group-VII 30 days selenium and curcumin exposure).

For biochemical estimations 12 lenses of six animals were pooled together from each group and the experiments repeated six times. Malondialdehyde the by-product of lipid peroxidation was estimated by the method of Bhuyan et al.. Catalase enzyme (E.C.1.11.1.6) activity was estimated by the method of Aebi. Xanthine oxidase (XOD; E.C. 1.2.3.2) activity was assayed by the method of Srikanthan and Krishna Murthy as described by Govindappa and
Swami. Superoxide dismutase (SOD; E.C. 1.15.1.1) activity was assayed according to the method of Marklund and Marklund. The data were statistically analysed using one-way analysis of variance (ANOVA).

**Results and Discussion**

Lenses of control animals reveal no opacities (Fig.1a and b) and it was evident that the selenium administered rats developed cataract (Fig.1c and d). No opacification was observed in the groups of rats, which were co-treated with curcumin (Fig. 1e and f).

The results of biochemical estimations are given in Table 1.

Higher doses of selenium are pro-oxidant in nature. The significant increase in lipid peroxidation level and that of cataracts in the present investigation could be due to the oxidative insult induced by the selenium.

In human cataractous lenses oxidative insult on the lens and its components have been observed and lipid peroxidation products are implicated in cataractogenesis. Malondialdehyde causes protein aggregation and oxidation of crystallins and membrane components is recognized as an early fundamental change in cataractous lens.

Xanthine oxidase and xanthine generated toxic derivatives of oxygen and the increase in XOD activity could be linked to the pro-oxidant nature of selenium. The significant reduction in the levels of lipid peroxidation and xanthine oxidase enzyme in

![Fig. 1—Close up of rat eye of 20-day old control animal; 30-day old control animal; (c) experimental animal after selenium treatment for 20-day (group-IV) showing nuclear cataract; (d) experimental animal after selenium treatment for 30-day (group-VI) showing nuclear and cortical cataract; (e) experimental animal after selenium and curcumin co-treatment for 20-day (group-V) showing no visible opacities; and (f) experimental animal after selenium and curcumin co-treatment for 30-day (group-VII) showing no visible opacities.]

Table 1—Oxidative and anti-oxidative enzymes in lens of Wistar rats after selenium exposure and curcumin and selenium exposure.  

<table>
<thead>
<tr>
<th>Group</th>
<th>LPO (μ mol MDA/g tissue)</th>
<th>XOD (μ mol/ mg protein)</th>
<th>Catalase (U/g tissue)</th>
<th>SOD (U/g tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>10.36±0.015</td>
<td>0.2±0.001</td>
<td>1.52±0.089</td>
<td>3.11±0.94</td>
</tr>
<tr>
<td>Group-II</td>
<td>18.13±0.015</td>
<td>0.35±0.003</td>
<td>1.38±0.003</td>
<td>2.75±0.023</td>
</tr>
<tr>
<td>(10-day selenium exposure)</td>
<td>(+75)</td>
<td>(+75)</td>
<td>(-12.1)</td>
<td>(-11.5)</td>
</tr>
<tr>
<td>Group-III</td>
<td>10.11±0.011</td>
<td>0.25±0.001</td>
<td>1.42±0.029</td>
<td>3.56±0.148</td>
</tr>
<tr>
<td>(10-day selenium+curcumin exposure)</td>
<td>(+2.41)</td>
<td>(+63)</td>
<td>(-9.55)</td>
<td>(-15.11)</td>
</tr>
<tr>
<td>Group-IV</td>
<td>20.11±0.013</td>
<td>0.47±0.003</td>
<td>0.96±0.006</td>
<td>2.39±0.007</td>
</tr>
<tr>
<td>(20-day selenium exposure)</td>
<td>(+94.11)</td>
<td>(+135)</td>
<td>(-38.85)</td>
<td>(-23.15)</td>
</tr>
<tr>
<td>Group-V</td>
<td>14.1±0.005</td>
<td>0.38±0.003</td>
<td>1.07±0.061</td>
<td>2.8±0.051</td>
</tr>
<tr>
<td>(20-day selenium+curcumin exposure)</td>
<td>(+36.19)</td>
<td>(+90)</td>
<td>(+31.84)</td>
<td>(+9.96)</td>
</tr>
<tr>
<td>Group-VI</td>
<td>23.0±0.006</td>
<td>0.66±0.003</td>
<td>0.91±0.041</td>
<td>1.42±0.006</td>
</tr>
<tr>
<td>(30-day selenium exposure)</td>
<td>(+122)</td>
<td>(+230)</td>
<td>(+42.03)</td>
<td>(+54.34)</td>
</tr>
<tr>
<td>Group-VII</td>
<td>12.15±0.03</td>
<td>0.41±0.0005</td>
<td>0.92±0.024</td>
<td>1.79±0.043</td>
</tr>
<tr>
<td>(30-day selenium+curcumin exposure)</td>
<td>(+17.27)</td>
<td>(+105)</td>
<td>(-99.41)</td>
<td>(-42.44)</td>
</tr>
</tbody>
</table>

All values are significant at P<0.001  
XOD: μ moles of formazan formed/ hr/mg protein  
LPO: μ moles of MDA/g wt. of tissue  
Catalase: Units/mg protein (Specific Activity)  
SOD: Units/mg protein (Specific Activity)
selenium and curcumin co-treated rat lenses could be due to the anti-oxidative property of curcumin and its fee-radical scavenging nature.\(^{2,7,28}\) Superoxide dismutase and catalase constitute the enzymatic defense mechanism against oxidative damage. SOD and catalase enzyme activities decreased in selenium exposed rats. But in selenium and curcumin co-treated rats, an increase in the activity levels of these enzymes was observed and this perhaps may be a biochemical strategy to reduce the lipid peroxidation as suggested by Reddy and Lokesh\(^{28}\) and Venkatesan, et al.\(^{25,26}\). This is also in conformity with the findings of Pandya et al.,\(^{31}\) who described prevention of naphthalene induced oxidative cataract by low levels of dietary curcumin and also cataracts induced by 4-hydroxy nonenal. The present findings suggest that curcumin has a role in preventing oxidative damage. The exact mechanism(s) of curcumin’s action in preventing formation of opacities is not known very clearly at present and further studies in this direction would throw more light on the protective role of curcumin in prevention of oxidative damage in general and delaying of cataract formation in particular.

References