Journal of Zhejiang University-SCIENCE B (Biomedicine & Biotechnology)
ISSN 1673-1581 (Print); ISSN 1862-1783 (Online)
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Letter to the Editor:

Curcumin and its emerging intraocular benefits

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doi:10.1631/jzus.B1200273
Document code: A **CLC number:** R776.1

I read with great interest the recent article "Curcumin inhibits proliferation of human lens epithelial cells: a proteomic analysis" by Hu *et al*. (2012), published in *Journal of Zhejiang University-SCIENCE B (Biomedicine & Biotechnology)*. Curcumin may be of significant benefit in other optic disorders besides cataracts.

Curcumin exerts anti-neoplastic effects in eye tumors such as retinoblastomas. It mediates these anti-cancer effects by altering the microRNAs (miRNAs) expression profile. Overall, sixteen miRNAs are down-regulated. Simultaneously five miRNAs, especially miR-22, are up-regulated in the retinoblastomas cells (Sreenivasan *et al*., 2012). The target gene of *miR-22* is erythoblastic leukemia viral oncogene homolog 3 (*Erbb3*).

Curcumin attenuates reactive oxygen species (ROS) levels and thereby affords protection to retinal pigment cells from oxidation injury. These protective effects are mediated by curcumin induced up-regulation of heme oxygenase-1 (HO-1) (Woo *et al*., 2012). The highest HO-1 expression is seen at a concentration of 15 μmol/L (Mandal *et al*., 2009). Diabetic retinopathy can be attenuated by curcumin by virtue of its anti-oxidant properties. It also indices hypoglycemia thus proving to be of further benefit in mitigating the development of diabetic retinopathy (Gupta *et al*., 2011). Curcumin decreases the elevations in vascular endothelial growth factor noted in diabetic retinopathy.

Ischemia/reperfusion injury in the retina is attenuated by curcumin administration. It mediates these protective effects by decreasing the activation of signal transducers and activators of transcription 3 (STAT3) (Wang *et al*., 2011). Simultaneously, activation of nuclear factor-κB (NF-κB) is also inhibited and further augments the protective effects of curcumin. As a result, it decreases vascular as well as neuronal degeneration in the retina. Up-regulation of p-IκBα and MCP-1 is also inhibited. These protective effects are even seen 48 h following the ischemic injury.

Curcumin also may be effective in the therapeutic management of retinitis pigmentosa. Retinitis pigmentosa secondary to P23H rhodopsin mutation may especially be amenable to curcumin therapy. Curcumin mediates these effects by decreasing stress on the endoplasmic reticulum and by improving gene expression in the retina as well as by improving retinal morphology (Vasireddy *et al*., 2011). Curcumin also dissociates protein aggregates and restores the expression of NF-κB thus further improves the pathology in retinitis pigmentiosa. These protective effects are reversed by the application of NF-κB inhibitory peptides.

Curcumin is also of benefit in proliferative vitreoretinopathy (Alex *et al*., 2010). It mediates these effects by inducing caspase 3 dependent apoptosis. Curcumin has an attenuatory effect on Bcl-2 and augments Bax levels (Lu *et al*., 2009). It also accentuates Fas-associated death domain protein (FADD) and Fas levels at the same time. As a result there is increased G_2/M phase arrest.

The above examples clearly illustrate the significant benefits of curcumin in intraocular pathologies and the need for further studies in this regard.

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