Management of uveal melanomas: cyclooxygenase-2 as a potential molecular target

Shailendra Kapoor

Received: 30 March 2008 / Accepted: 21 May 2012 / Published online: 7 June 2012
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To The Editor

In a recent issue of International Ophthalmology, Ozdal et al. [1] wrote about cyclooxygenase-2 expression in human irradiated uveal melanomas. They reported that radiotherapy may actually decrease cyclooxygenase-2 expression in uveal melanomas. This is of great clinical significance as almost 90 % of all uveal melanomas are immunoreactive for cyclooxygenase-2 [2]. In fact, increased cyclooxygenase-2 expression by uveal melanomas is associated with a worse prognosis.

Cyclooxygenase-2 inhibitors such as celecoxib have already been shown to inhibit tumor growth in cutaneous melanomas and cyclooxygenase-2 may very well be an ideal target molecule to inhibit carcinogenesis in uveal melanomas [3]. For instance, Marshall et al. [4] have recently demonstrated the effectiveness of a topical cyclooxygenase inhibitor nepafenac in delaying tumor progression in animal models. Fernandes et al. [5] have also shown that amfenac (the active metabolite of nepafenac) increases the sensitivity of the tumor cells to radiotherapy. In fact, radiotherapy may very well have a synergistic effect with amfenac in inhibiting uveal melanoma cells. Interestingly, Souza et al. [6] have also reported increased cyclooxygenase-2 expression in retinoblastomas.

Clearly, cyclooxygenase-2 has a major role to play in the evolution and progression of ocular tumors. Given the massive potential that anti-cyclooxygenase therapies such as nepafenac and radiotherapy have in the management of uveal melanomas, there is a clear and urgent need for further human studies to fully explore their potency and effectiveness in the management of these tumors.

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S. Kapoor (✉)
University of Illinois at Chicago, Chicago, IL 60612, USA
e-mail: shailendrakaipoor@yahoo.com