Green Tea Prevents Skin Cancer by Two Mechanisms

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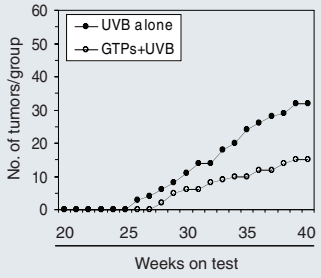
Skin cancer accounts for more new cases of cancer than all other cancers combined. Exposure to UV radiation is the most important risk factor for development of skin cancer (LeBlanc et al., 2008). Primary prevention has reduced the incidence of skin cancer in the geographic regions at highest risk, such as Australia (Geller et al., 2009), but researchers are actively seeking ways to augment prevention through chemoprevention. Candidate agents include commonly used medications and foods such as nonsteroidal anti-inflammatory agents, lipid-lowering agents, angiotensin-converting enzyme and receptor blockers, and green tea (Christian et al., 2008). Polyphenols from green tea (GTPs) have been shown to reduce UV-induced skin cancer in animal models (Mantena et al., 2005). The mechanisms by which this is achieved remain unclear, but it is thought that the ability of GTPs to reduce the inflammation associated with UV exposure may be an important factor in this process.



As reported in this issue, to study the mechanisms by which GTPs affect inflammation and DNA repair and by which GTPs may prevent skin cancers induced by chronic UV exposure, Meeran and co-workers fed GTPs through drinking water to IL-12 (an important mediator of inflammation) knockout (KO) and wild-type mice (Meeran et al., 2009). Compared with control mice, chronically UV-exposed and GTP-fed mice exhibited a delay in tumor onset, as well as smaller and fewer tumors. The benefit of GTPs was observed in IL-12 KO mice, but it was markedly less dramatic.

Mediators of inflammation, such as prostaglandin E_2 and cyclooxygenase-2 (Katiyar and Meeran, 2007), as well as markers of cell proliferation, such as proliferating cell nuclear antigen and cyclin D1, were also affected by GTP administration; these effects were weaker in IL-12 KO mice. Repletion of IL-12 in KO mice and anti-IL-12 administration in wild-type mice confirmed the role of IL-12. Because GTP benefits appeared to be only partly mediated through IL-12, DNA repair of cyclopyridine dimers was studied after acute UV exposure. GTP-fed mice showed greater DNA repair than did control mice, suggesting a dual mechanism by which GTP might reduce skin cancer development in animal models.

Through the following questions, we examine this paper in greater detail. For brief answers, please refer to http://network.nature.com/group/jidclub.

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QUESTIONS

1. What are the mechanisms by which UV induces skin cancer?
2. What is the rationale for studying green tea to prevent skin cancer?
3. What were the hypotheses in this study, and how were they tested?
4. What were the findings of this study?
5. What were the limitations of this study?
6. How might the article affect your recommendations to patients?

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