Spermatogenesis 2:4, 304-304; October/November/December 2012; © 2012 Landes Bioscience

Testin and its emerging modulatory role in systemic carcinogenesis

Shailendra Kapoor

Private Practice; Chicago, IL USA

I read with great interest the recent article by Mruk et al.[1] Testin may suppress tumor growth in a number of systemic malignancies.

Downregulation of the testin gene contributes to the development of head and neck malignancies. Better survival rates are seen in individuals with head and neck malignancies that exhibit high testin levels in comparison to those who exhibit low testin levels.[2] Interestingly, downregulation of testin is more likely in those with a family history of head and neck malignancies.

Similarly, in gastric tissue testin acts as a tumor suppressor gene.[3] Nearly, 72% of gastric carcinomas exhibit downregulated testin levels.[4] The testin promoter usually undergoes hypermethylation in gastric carcinomas. While upregulation of testin attenuates tumor growth in gastric carcinomas, its downregulation enhances gastric carcinogenesis. In fact, a poor clinical outcome is seen in gastric malignancies with downregulated testin levels. Testin is thus emerging as a valuable prognostic indicator in gastric carcinomas. Patient with testin positive gastric malignancies tend to have a longer median survival in comparison to those with testin negative tumors.

Recent studies suggest that tumor growth in both breast cancers as well as endometrial carcinomas can be attenuated following adenovirus mediated transduction of the testin gene.[5] Tumor attenuation and apoptosis in breast carcinomas is mediated through caspase dependent mechanisms.

The above data clearly illustrates the importance of assessing testin expression in systemic malignancies and the need for further studies in this regard.

References

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Correspondence to: Shailendra Kapoor; Email: shailendrakapoor@yahoo.com
Submitted: 10/09/12; Accepted: 11/01/12
DOI: http://dx.doi.org/10.4161/spmg.22744