RhoA, a novel tumor suppressor or oncogene as a therapeutic target?

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Journal Title: Genes & Diseases
Volume: Volume 2, Number 1
Publisher: Elsevier | 2015-03, Pages 2-3
Type of Work: Article | Final Publisher PDF
Publisher DOI: 10.1016/j.gendis.2014.10.001
Permanent URL: https://pid.emory.edu/ark:/25593/pqs3x

Final published version: http://dx.doi.org/10.1016/j.gendis.2014.10.001

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Accessed December 20, 2021 12:25 AM EST
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COMMENTARY

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mechanisms, such as nonsense or frame-shift mutations in these tumor types. The recurrent nature of RhoA mutations in AITL, PTCL and diffuse-type gastric carcinoma strongly suggests that these hotspot mutations result in a gain-of-function alteration in an unidentified signaling pathway; nevertheless, in the absence of any supporting data, the question still remains whether RhoA is an oncogene or tumor suppressor gene.

From the cancer treatment perspective, the recurrent mutational hotspots of this protein represent ideal targets for small molecule inhibitors as therapeutic reagents. If the RhoA mutants act in a dominant negative fashion, such molecules could disrupt their interaction with the wild-type protein to restore RhoA function. On the other hand, if RhoA mutants are oncogenes, the suppression of their activities by these molecules should inhibit tumorigenesis. In either case, the future development of these therapeutic reagents holds promise for cancer patients with RhoA mutations.

Acknowledgments

The authors declare no conflict of interest. We would like to thank Dr. Anthea Hammond for editing this manuscript. WZ is an Anise McDaniel Brock Scholar, a Georgia Cancer Coalition Distinguished Cancer, and an American Cancer Society Research Scholar.

References


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4 October 2014