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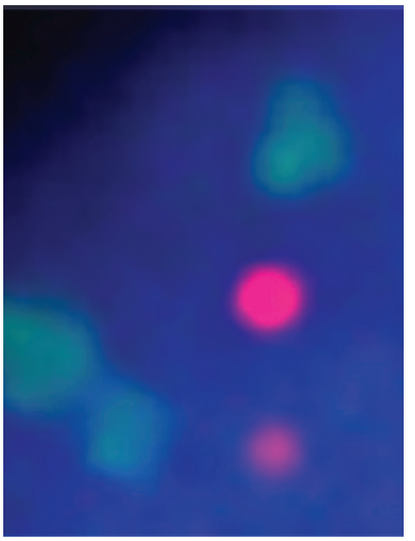
ABOUT THE COVER

Following treatment with a G-quadruplex ligand, telomestatin, glioma stem cells rapidly developed punctate nuclear 53BP1 foci. Of note, some of these foci colocalized with nontelomeric DNA, thereby representing both telomeric and nontelomeric dysfunction-induced foci, a hallmark of deprotected DNA damage. The loss of tumor stemness is likely associated with a failure in the DNA damage response elicited by telomestatin in glioma stem cells. For details, see the article by Miyazaki and colleagues on page 1268 of this issue.



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