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Viral hit-and-run

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A mammalian complex containing three nuclear proteins - Mre11, Rad50 and NBS1 - plays a critical role in repairing double-strand breaks in DNA and maintaining telomeres. In the July 18 Nature, Matthew Weitzman and colleagues at the Salk Institute in California, describe how viral oncoproteins can cooperate to disable the Mre11-Rad50-NBS1 complex (*Nature* 2002, 418:348-352). They demonstrate that polypeptides encoded by the adenovirus E4 early region are necessary to prevent concatemerization of the double-stranded DNA viral genome. They found that the E4 proteins target the human Mre11-Rad50-NBS1 complex by increasing the degradation of its components and changing their cellular localization. Two adenoviral proteins, E4orf6 and E4orf3, are needed to regulate this degradation and mislocalization. Sustained expression of the E4 proteins is not required for viral transformation, leading the authors to suggest that down-regulation of the host DNA-damage machinery accounts for the 'hit-and-run' behaviour of these viral oncoproteins.

References

1. The hMre11/hRad50 protein complex and Nijmegen breakage syndrome: linkage of double-strand break repair to the cellular DNA damage response.

2. *Nature*, [http://www.nature.com]

3. Salk Institute , [http://www.salk.edu]

4. "Hit-and-run" transformation by adenovirus oncogenes.