PublisherInfo				
PublisherName		BioMed Central		
PublisherLocation		London		
PublisherImprintName		BioMed Central		

BRCA2 loss in Fanconi Anemia

ArticleInfo		
ArticleID	:	4504
ArticleDOI		10.1186/gb-spotlight-20020614-01
ArticleCitationID	\Box	spotlight-20020614-01
ArticleSequenceNumber	\Box	170
ArticleCategory	$\begin{bmatrix} \vdots \end{bmatrix}$	Research news
ArticleFirstPage	\Box	1
ArticleLastPage	\Box	2
ArticleHistory	:	RegistrationDate : 2002–6–14 OnlineDate : 2002–6–14
ArticleCopyright		BioMed Central Ltd2002
ArticleGrants	\Box	
ArticleContext	\Box	130593311

Jonathan B Weitzman

Email: jonathanweitzman@hotmail.com

Fanconi Anemia (FA) is a recessive cancer susceptibility syndrome. Six FA genes have been cloned and encode proteins involved in a DNA-damage response pathway. In the June 13 ScienceXpress, Howlett *et al.* report the characterization of mutations in cells from the FA subtypes B and D1 (*ScienceXpress* 13 June 2002, DOI:10.1126/science.1073834). They discovered biallelic mutations in the *BRCA2* breast cancer susceptibility gene; the mutations create frameshifts resulting in truncated BRCA2 protein. Howlett *et al.* show that restoring *BRCA2* expression could rescue the phenotype of FA cells and restore resistance to DNA-damaging agents. The authors propose a model linking FA-associated genes to the regulation of a common DNA-damage response pathway.

References

- 1. Fanconi anemia and DNA repair
- 2. Interaction of the Fanconi anemia proteins and BRCA1 in a common pathway.
- 3. ScienceXpress, [http://www.sciencexpress.org]