in normal breast epithelial cells, placenta and the breast cancer cell line MCF7. This suggests that our present contig of cDNAs covering approximately 7.3 kb (including 300 bp of 3′ untranslated sequence) may not include the whole *BRCA2* coding sequence. The known sequence of 2,329 amino acids encoded by the *BRCA2* gene does not show strong homology to sequences in the publicly available DNA or protein databases, and therefore we have no clues to its functions. However, some weak matches were detected including, intriguingly, a very weak similarity to the *BRCA1* protein over a restricted region (amino acids 1394–1474 in *BRCA1*, and 1783–1863 in the portion of *BRCA2* shown in Fig. 2). The significance of this is unclear.

Loss of heterozygosity on chromosome 13q has been observed in sporadic breast and other cancers, suggesting that there is a somatically mutated tumour suppressor gene in this region[11–13] *BRCA2* is a strong candidate for this gene, and the analysis of a large series of cancers is underway to investigate if *BRCA2* is somatically mutated during oncogenesis.

The identification of *BRCA2* should now allow more comprehensive evaluation of families at high risk of developing breast cancer. However, the roles of environmental, lifestyle or genetic factors in modifying the risks of cancer in gene carriers are unknown, and further studies will be required before routine diagnosis of carrier status can be considered. □

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RETRACTION

Cloning and functional expression of a rat heart K_{ATP} channel

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In this letter we described the cloning and expression of an inward rectifier potassium-channel subunit from rat heart (Kir 3.4) which, when transfected into HEK293 and BHK21 cells, endowed them with ATP-sensitive potassium channels. Since this paper appeared, we have not been able regularly to reproduce those findings. In addition, the data presented by Krapivinsky *et al.*[1] presents a compelling argument that Kir 3.4 is an intrinsic component of the channel underlying I_{KACh} in atrium, and that it does not contribute to the channel underlying cardiac I_{KATP}. Therefore, we cannot support our previous statement that Kir 3.4 represents a subunit of cardiac K_{ATP} channels. □

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