

## Errata

The authors wish to add the following acknowledgments to their article “Cancer Risks in Two Large Breast Cancer Families Linked to BRCA2 on Chromosome 13q12-13,” by Easton et al. (61:120–128), published in the July 1997 issue of the *Journal*:

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Institutes of Health and by grants DAMD 17-95-1-6266 and DAMD 17-94-J-4260 from the Department of Defense. This research was also supported by the Utah Cancer Registry, which is funded by contract NO1-CN-6700 from the National Cancer Institute, with additional support from the Utah State Department of Health and the University of Utah.

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In the September 1997 issue of the *Journal*, an error appeared in the article “Characterization of 18 New Mutations in COL7A1 in Recessive Dystrophic Epidermolysis Bullosa Provides Evidence for Distinct Molecular Mechanisms Underlying Defective Anchoring Fibril Formation,” by Hovnanian et al. (61:599–610). In Table 1 (“Characteristics of COL7A1 Mutations in 15 Unrelated RDEB Patients” [p. 603]), the nucleotide change (third column) for patient 1 should be gGGATCAAG→gG, instead of GGGATCAAG→GG. In addition, the authors add the following:

Patient 1 demonstrated a 7-bp deletion in exon 5, which led to

a TGA stop codon 22 nucleotides downstream of codon 173. Since this deletion removes the first 7 bp of exon 5, we investigated the possibility that splicing of this exon might be altered in this patient. The analysis of RT-PCR products encompassing exons 3–6 showed a band slightly smaller than that shown for the control, in addition to a band of normal size and heteroduplexes. The intensity of these bands was reduced, in comparison with that of the bands for the control. Direct sequencing of the small band showed normal splicing of exon 5 harboring the 520del7 deletion. This is consistent with the fact that this mutation leaves unchanged the G residue at the first position of the exon, a position occupied most often (55%) by a G, in vertebrates, whereas nucleotides at more internal exon positions have not been reported as consensus sequences of acceptor splice sites (Shapiro and Senapathy 1987).

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In the September 1997 issue of the *Journal*, an error appeared in the letter “Family Cell Lines Available for Research—An Endangered Resource?” by Lernmark et

al. (61:778–779). The correct URL for the Todd laboratory Website is <http://www.well.ox.ac.uk/~plyons>