Errata

Table 3

Association of the Truncating Mutation E265X and the Missense variant R462Q of the RNASEL Gene with Patients with BPH, Unselected PRCA, or HPC

| Patient or Family Sample and Mutation | No. of Carriers/ Total (Frequency) | OR | 95% CI | P |
|---------------------------------------|---------------------------------------|------|------------|------|
| E265X: | . 1 77 | | | |
| Controls | 10/566 (1.8%) | 1.00 | | |
| Patients with BPH | 7/223 (3.1%) | 1.80 | .68-4.79 | .24 |
| Patients with unselected PRCA | 10/492 (2.0%) | 1.15 | .48-2.80 | .75 |
| All patients with HPC | 5/116 (4.3%) | 2.51 | .84-7.47 | .1 |
| Two affecteds | 1/64 (1.6%) | .88 | .11-7.01 | .91 |
| Three affecteds | 2/31 (6.5%) | 3.83 | .80-18.31 | .09 |
| Four or more affecteds | 2/21 (9.5%) | 5.85 | 1.20-28.87 | .03ª |
| R462Q homozygotes: | | | | |
| Controls | 23/176 (13.1%) | 1.00 | ••• | |
| Patients with unselected PRCA | 24/167 (14.4%) | 1.12 | .60-2.07 | .73 |
| All patients with HPC | 15/66 (22.7%) | 1.96 | .95-4.03 | .07 |
| Two affecteds | 2/19 (10.5%) | .78 | .17-3.61 | .75 |
| Three affecteds | 7/26 (26.9%) | 2.45 | .93-6.47 | .07 |
| Four or more affecteds | 6/21 (28.6%) | 2.66 | .94–7.55 | .07 |

^a Statistically significant.

In the May 2002 issue of the *Journal*, in the article entitled "Germline Alterations of the *RNASEL* Gene, a Candidate *HPC1* Gene at 1q25, in Patients and Families with Prostate Cancer," by Rökman et al. (70:1299–1304), four of

the odds ratios and their corresponding 95% CI figures were incorrect. The corrected table 3 is shown here. The authors regret these errors and thank Professor Henrik Grönberg for bringing these mistakes to their attention.

In the June 1999 issue of the *Journal*, in the article entitled "Mutational Analysis of the Defective Protease in Classic Late-Infantile Neuronal Ceroid Lipofuscinosis, a Neurodegenerative Lysosomal Storage Disorder" by Sleat et al. (64:1511–1523), we reported in error that the cell line GUS16776 lacked CLN2 protease activity.

Subsequent reanalysis of this cell line, which was derived from a patient originally diagnosed with late-infantile neuronal ceroid lipofuscinosis, has revealed the activity of the *CLN2* gene product, tripeptidyl peptidase I, to be normal in this cell line; thus, a defect in a gene other than *CLN2* is likely. The authors regret this error.