developed premenopausal breast cancer. Her mother and two of her siblings, who were mutation carriers, also had premenopausal breast cancer. This latest event suggests that other, possibly additional, genes may be involved in this family, the only classical LFS family to date reported to carry a *CHEK2* mutation.

Over the last 8 years, much data have accumulated on the clinical expression of *CHEK2* mutations and we should therefore re-evaluate the original claim. There is one predominant founder mutation in *CHEK2* (1100delC) which is carried by 0.7% of the Northern European population. In contrast, the LFS is believed to affect one in 20 000 births—or approximately 100 times less.¹⁰ If Bell and colleagues are correct, would we not expect the number of *CHEK2* mutations in LFS to greatly exceed the number of *TP53* mutations? There are two relevant questions here: (1) do families with *CHEK2* mutations manifest the cancers which are associated with the classical LFS (or LFL); and (2) are the cancers found to be associated with *CHEK2* mutations also found in LFS? A detailed analysis of 67 *CHEK2* families¹¹ failed to identify an increased risk of cancer for any site other than breast cancer—notably, no increases in sarcomas or adrenocortical cancers were seen. A study of 4008 Polish cancer cases found excess risks for cancers of the prostate, breast, colon, kidney and thyroid among carriers of *CHEK2* mutations¹²—none of these sites (other than breast) is featured in LFS or LFL.

Based on these and other analyses we believe that *CHEK2* mutations are not associated with the cancer types seen in the LFS or LFL (other than breast cancer) and it is no longer reasonable to consider *CHEK2* mutations to be a cause of LFS.

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CORRECTION

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The journal apologises for an error that occurred in this paper on page 496, in the figure 3 caption, which should read as follows:

(B) Timed growth assay. The mean percent growth in copper/iron-limited media of *ccc2Δ* transformed with *S637L* and *A1362D*, compared with *ccc2Δ* transformed with wild-type *ATP7A*, was 73% and 17%, respectively. The mock-transformed *ccc2Δ* strain (quadrant 1) failed to grow.