report, "Less can be more, U.K. study finds," by B. Shouse (17 Aug., p. 1238). Semple's presentation compared the relative coverage and rates of misassembly across several draft human genome assemblies for a region of chromosome 4p linked with bipolar affective disorder. The assemblies examined included releases from the National Center for Biotechnology Information (http://www.ncbi.nlm.nih.gov/), the University of California at Santa Cruz (http://genome.ucsc.edu/), and the public release from Celera Genomics (CG) (http://public.celera.com/). There were inaccuracies in the article relating to the data analyzed, the results presented, and the attributed collaborators in this work, and we wish to set the record straight.

First, this work was performed by Colin Semple in collaboration with Kathryn Evans and Stewart Morris in the laboratories of David Porteous in the Molecular Medicine Centre, University of Edinburgh.

Contrary to the report by Shouse, we have not undertaken large-scale sequencing of the region; rather, our group constructed a contig of BAC/PAC clones across the region (*1*). The comparisons between draft genome assemblies exploited the high-resolution physical mapping data from this contig. Each assembly was assessed with respect to the proportion of International Human Genome Sequencing Consortium (IHGSC) sequence data included from the region (constituting our estimate of coverage) and also for the number of deviations from the marker order seen in the contig (our estimate of the rate of misassembly). The region examined was estimated to be 5.8 megabases in size, not 6.9, as stated in the article. An important conclusion from the presentation was that the available assemblies vary widely by both measures. No conclusions were drawn, or could be drawn, as to the total amounts of sequence from the region produced by CG and IHGSC. The next generation of common, complex disease mapping studies will rely on linkage disequilibrium mapping using accurate, high-resolution maps of regions conferring susceptibility. Detailed descriptions of these regions cannot be derived from an inaccurate assembly of the available genomic sequence. Thus, our data emphasize that, until a region of interest is covered by finished sequence, a high-resolution physical map of the region is indispensable to correct the misassemblies present in all publicly available draft human genome assemblies.

Shouse also says that "Celera's approach of breaking the whole genome into random fragments for sequencing yielded better data than the map-directed approach used by [the Human Genome Project]." However, this is a false dichotomy because CG used mapping and sequence data from IHGSC in the production of their published assembly. Therefore, our comparisons, which only examined the published CG assembly, shed no light on the issue of whether it is better to start with a clone-based physical map of the genome or with whole-genome shotgun sequencing.

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References and Notes
1. K. L. Evans *et al.*, *Genomics* **71**, 315 (2001).

CORRECTIONS AND CLARIFICATIONS

RESEARCH ARTICLE: "Resilient quantum computation" by E. Knill, R. Laflamme, W. H. Zurek (16 Jan. 1998, p. 342). In both the legend and the text discussion of Fig. 1, the double exponents in the expression $c^{(2h-1)}p^{(2h)}$ were omitted. The correct expression is $c^{(2^h-1)}p^{(2^h)}$.