might occur) would provide generation-by-generation variability in phenotype. The resulting phenotypic variation would be similar to that caused by recombination among multiple loci with many alleles, but would have a more efficient molecular basis. One stable allele associated with a spontaneously variable regulator would enable offspring to display a range of phenotypic values around a parental mean.

Significantly, such a mechanism for spontaneous, site-specific mutagenesis of a regulatory sequence would enable even a small population to extend its range of phenotypic variation, for the affected trait, within a few generations. Natural selection could establish and maintain an optimal trait distribution in a shifting environment, with no delay for the mutation of Mendelian genes or for the elimination of less fit alleles. Yet maladaptive extremes of gene expression would be unlikely to arise except from parents who were themselves far from the population mean, so genetic load would be minimal (unless, of course, the mechanism slipped out of control or exceeded some threshold, as it may have in cases of human neurological disease).

From an evolutionary perspective, nonstandard mutational mechanisms that affect specific loci can offer substantial advantages (*1*). The triple repeats that are widespread among animal genomes might represent one such mechanism. Testing the hypothesis that variable repeat length may regulate quantitative gene expression will be challenging precisely because such sequences are not stable from one generation to the next. But understanding a mechanism that could generate copious but normally benign mutation might be worth the effort. After all, it's been more than a century since Darwin promised that "a grand and almost untrodden field of inquiry will be opened, on the causes and laws of variation" (*2*).

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References

1. P. Rainey and R. Moxon, *Science* **260**, 1958 (1993); R. E. Lenski and J. E. Mittler, *ibid.*, p. 1959.
2. C. Darwin, *On the Origin of Species* (Harvard Univ. Press, Cambridge, MA, 1964), p. 486.

Genetics and Violent Crime

Peter R. Breggin writes (Letters, 3 Dec., p. 1498) that there are no known biological or genetic factors that contribute to violent crime. Yet it is well known, even by most psychiatrists, that individuals with Y chromosomes commit the overwhelming preponderance of violent crimes.

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Corrections and Clarifications

In the report "DNA sequence determination by hybridization: A strategy for efficient large-scale sequencing" by R. Drmanac *et al.* (11 June, p. 1649), the sequence of clone 8 in figure 2B (p. 1650) was inadvertently shortened by the deletion of "GA" at the seventh position from the right in the second line. In reference 20 of the same report, the probes ATATGGGG and ATGTCCTG should not have been included.