atmospheric pCO_2 variations of about two-thirds the magnitude of the about 90 p.p.m. glacial–interglacial pCO_2 difference[6]. Models to explain these glacial–interglacial variations in atmospheric CO_2 have centred on changes in ocean circulation[7], but no entirely satisfactory explanation has yet been proposed. Recent evidence that volcanic eruptions may follow a ~23,000-yr cycle[8] (perhaps as a result of changes in sea level)[9] suggests possible orbital pacemakers for volcanic CO_2 emissions. Could a few volcanoes such as Etna significantly affect the atmospheric CO_2 concentration on the 10^4–10^5-yr timescale?

KEN CALDEIRA
Earth System Science Center and Department of Geosciences, The Pennsylvania State University, University Park, Pennsylvania 16802, USA
MICHAEL R. RAMPINO
Earth Systems Group, Department of Applied Science, New York University, New York, New York 10003, USA

1. Allard, P. *et al. Nature* **351**, 387–391 (1991).
2. Berner, R. A., Lasaga, A. C. & Garrels, R. M. *Am. J. Sci.* **283**, 641–683 (1983).
3. Chester, D. K., Duncan, E. M., Guest, J. R. & Kilburn, C. R. J. *Mount Etna: the Anatomy of a Volcano* (Stanford University Press, 1985).
4. Caldeira, K. & Rampino, M. R. *Geophys. Res. Lett.* **15**, 987–990 (1991).
5. Berner, R. A. *Am. J. Sci.* **291**, 339–376 (1991).
6. Barnola, J. M., Raynaud, D., Korotkevich Y. S. & Lorius, C. *Nature* **329**, 408–414 (1987).
7. Broecker, W. S. & Denton, G. *Quart. Sci. Rev.* **9**, 305–341 (1990).
8. Paterne, M., Labeyrie, J., Guichard, F., Mazaud, A. & Maitre, F. *Earth. planet. Sci. Lett.* **98**, 166–174 (1990).
9. Rampino, M. R., Self, S. & Fairbridge, R. W. *Science* **206**, 826–829 (1979).

More questions on forest decline

SIR — Ross and De Serves[1] criticize the recent proposal by Becker *et al.*[2] that H_2O_2 formation via the reaction of terpenes with ozone in the presence of water could make a significant contribution to the mixed-layer H_2O_2 budget, thereby contributing to forest decline. We agree that the yields reported by Becker *et al.* are too low to make a significant contribution to the H_2O_2 budget of the mixed layer compared to H_2O_2 formation via peroxy radical disproportionation.

Becker *et al.* in their reply[3] to the criticism by Ross and De Serves suggest that the true yields may be much larger than their reported values because, for the high concentrations of reactant used by them, the reaction of the Criegee biradicals with aldehydes produced in the reaction would compete with their reaction with water. But the independence of the H_2O_2 yields over a wide reactant concentrations range, as reported by Becker *et al.*, implies that such a competition cannot be important under their conditions. Our recently reported significantly higher yields for the formation of H_2O_2 via the new process[4] suggests that under some conditions that contribution of this process to the mixed layer H_2O_2 budget may not be negligible. Using fluxes for terpenes quoted by Ross and De Serves, for example, and assuming that about 25% of the β-pinene and about 50% of the other terpenes are oxidized by reaction with ozone[5], the rate of H_2O_2 formation is estimated to be about 52 parts per 10^{12} by volume per hour (p.p.t.v. h^{-1}) in the mixed layer. This can be compared to 95 p.p.t.v. h^{-1} calculated for the disproportionation process for a HO_2 concentration of 20 p.p.t.v. (ref. 6).

Becker *et al.*[3] suggest that our observations of high H_2O_2 yields are due to interference from ozone in the wet chemical system used for peroxide analysis. There is no basis for this conclusion. Previous studies have shown that interference by ozone using the fluorimetric method for H_2O_2 determination is very small[7]. This was confirmed in our experiments. Introduction of 150 parts per 10^9 ozone into the reaction chamber in the absence of alkene initially indicated the formation of about 70 p.p.t.v. of H_2O_2 which rapidly decreased to about 30 p.p.t.v. This corresponds to less than 1% of the H_2O_2 formed in the experiments in the presence of alkene.

R. SIMONAITIS
K. J. OLSZYNA
J. F. MEAGHER
Atmospheric Science Department, Tennessee Valley Authority, Muscle Shoals, Alabama 35660, USA

1. Ross, H. B. & De Serves, C. *Nature* **352**, 672 (1991).
2. Becker, K. H., Brockmann, K. J. & Bechara, J. *Nature* **346**, 257–258 (1990).
3. Becker, K. H., Brockman, K. J. & Bechara, J. *Nature* **352**, 672–673 (1991).
4. Simonaitis R., Olszyna, K. J. & Meagher, J. F. *Geophys. Res. Lett.* **18**, 9–12 (1991).
5. Atkinson, R., Hasegawa, D. & Ashmann, S. M. *Int. J. Chem. Kinetics* **22**, 871–887 (1990).
6. Trainer, M. *et al. J. geophys. Res.* **92**, 11879–11894 (1987).
7. Lazrus, A. L. *et al. Analyt. Chem.* **58**, 594–597 (1986).

Maintenance of MHC polymorphism

SIR — Although the finding of Hill *et al.*[1], that specific human class I and class II MHC alleles are associated with resistance to *Plasmodium falciparum* malaria, is of undoubted importance, their interpretation of the evolutionary mechanisms underlying their findings is questionable. Hill *et al.* give the impression that their data may favour the hypothesis of frequency-dependent rather than overdominant selection as the mechanism of maintenance of MHC polymorphisms. In fact, their data are not consistent with the former hypothesis, and their study does not constitute a true test of the latter.

The overdominance hypothesis[2,3] depends on the exposure of the population to two or more pathogens (either separate species or antigenically distinct strains of the same species) to which different MHC alleles confer resistance. We previously wrote: "As a particular class I antigen may provide enhanced recognition of a particular pathogen, individuals expressing various types of class I antigen may have a selective advantage in a population exposed to a diverse array of pathogens; such individuals will be those which are heterozygous at most or all loci"[3]. On this hypothesis, one would expect to find (as Hill *et al.* report in the case of HLA-Bw53) that heterozygotes for an allele conferring resistance to a single pathogen are no more resistant to that pathogen than are homozygotes for the same allele. In fact, *P. falciparum* populations are polymorphic with respect to proteins that elicit an immune response[4], and it is conceivable that different MHC alleles confer resistance to different strains of this parasite. But because Hill *et al.* did not consider polymorphism in *P. falciparum* and did not study other pathogen species, their results are not directly relevant to the overdominance hypothesis.

In the case of class II MHC haplotypes, Hill *et al.* present evidence that, among individuals having the protective DRw13.02 haplotype, there is a higher proportion of heterozygotes among those with severe malarial anaemia than among mild controls. This suggests that the fitness of Drw13.02 homozygotes is higher than that of DRw13.02 heterozygotes. If this is true, it has nothing to do with the maintenance of MHC polymorphism. In the absence of other factors, such as the presence of other pathogens to which other haplotypes confer resistance, these apparent fitness values amount to an absolute advantage for the DRw13.02 haplotype. Therefore, if no other factors are involved, the result should eventually be fixation of the DRw13.02 haplotype.

Furthermore, it is worth noting that the data of Hill *et al.* provide no support for the hypothesis of frequency-dependent selection. The only model of frequency-dependent selection that is as effective as that of overdominant selection for maintaining the type of multiallelic, long-term polymorphism characteristic of the MHC is a model of rare allele advantage[5]. But the data of Hill *et al.* for both class I and class II show that the protective alleles have high frequen-