— not for want of trying.

We failed to find salicin (present in the food plant) in the tissues of the viceroy (M. R. and R. Nash, unpublished); nor was pyrazine present in its offensive, aposematic odour³. However, we did find it contained a high concentration of carotenoids (938.7 µg per g) characteristic of models rather than mimics⁴ and we remarked once again that "the Viceroy may well be a müllerian rather than a batesian mimic".

The identification of self-secreted active deterrents in Lepidoptera, some of which also sequester toxins from their food plants, is rather difficult. We found a lethal protein in *Arctia caja* (which sequesters and stores cardiac glycosides and pyrrolizidin alkaloids from its food plants) which we named cajan⁵ and another lethal substance in white butterflies (which sequester mustard-oil glycosides from Cruciferae) which we named pierin⁶. But who is going to hit the jackpot by isolating and identifying limenitins?

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SIR — Vane-Wright suggests¹ that one of the most telling points against batesian mimicry is likelihood, on the grounds that chemical defence is more likely to evolve because it provides more reliable protection. We are not aware of any evidence that this argument is valid. Evolution is opportunistic and has to make do with the genetic variation that is actually available, and biochemical and ecological constraints on the synthesis and storage of chemical defences may, for all we know, greatly exceed those affecting colour patterns. No doubt comparative and phylogenetic studies could establish that chemical defences do evolve more frequently than mimetic resemblances. But this is a historical and statistical necessity as unpalatability must exist before mimicry can evolve, and the evolution of mimicry is itself an improbable event.

Ritland and Brower note the obvious dangers of extrapolating from a laboratory experiment to the field². It would clearly be highly desirable to use field tests to discriminate between batesian and müllerian mimicry. Such tests are possible if frequency-dependent predation of different colour forms can be detected. In müllerian mimicry, predation is negatively frequency-dependent (rare forms attacked) whereas in batesian mimicry it is positively frequency-dependent (common forms attacked). We believe that the use of this criterion has been wrongly neglected in favour of laboratory experiments and chemical investigations. Batesian³

and müllerian⁴ mimicry can be distinguished in this way. Where naturally occurring colour variants are lacking, artificially altered butterflies can be used⁴.

Finally, we wish to draw attention to the existence of rule-breaking mimics⁵, in which there is widespread biogeographical mismatching in the distributions of mimics and models. The study of mimicry has long since reached the point where the exception is far more interesting than the rule, and we believe that further investigation of such cases may reveal much about the circumstances under which functional mimicry can evolve.

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Virus ecology

SIR — Suttle *et al.*¹ suggest that in addition to grazing and limitation of nutrients, infection of phytoplankton by marine viruses could be a factor regulating their production. The evidence for this hypothesis is the huge amounts of virus particles found in marine waters, and the finding that up to 20-fold concentrations of such can reduce the photosynthesis of marine phytoplankton under experimental conditions¹. Although this hypothesis is attractive, we think that one important aspect of this phenomenon has not been considered. When a new, highly virulent virus type is introduced into a population of initially susceptible individuals, an enormous selection pressure for virus-resistant variants is generated. A well-known example of this phenomenon is the artificial introduction of myxoma virus into Australia to help control the rabbit pest problem². In this giant field experiment, genetic resistance to myxomatosis increased so that the mortality rate after infection with a particular strain of virus decreased from 90 to 25 per cent within a few years.

This phenomenon is not restricted to virus–animal systems. Shortly after his discovery of bacteriophages³, d'Herelle suggested that bacterial viruses might serve a useful therapeutic purpose; but he was never able to prove his idea experimentally¹. The

suggestion that exogenous marine viruses should continuously be able to kill a large proportion of a phytoplankton community seems to us as unlikely as the idea that bacteriophages should eliminate pathogenic or other bacteria within a given ecological niche. Therefore, if marine viruses are involved in the control of primary plankton production, this probably occurs only if a new virulent virus strain emerges, and the effects on primary production would most likely only be transient, owing to the rapid evolution and reproduction of resistant phytoplankton strains.

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SUTTLE *ET AL.* REPLY — Olofsson and Kjelleberg doubt that viruses could reduce primary productivity because of selection for virus-resistant variants. This point is an important aspect of viral ecology and we agree that resistant phytoplankton would arise if put under strong selective pressure by a viral pathogen.

The greatest impact of viruses on photosynthetic rates probably occurs in specific situations, such as during the demise of large monospecific phytoplankton blooms. Resistant variants would not prevent a transitory decrease in productivity as the infection propagated, but would presumably protect the host species from local extinction. In non-bloom situations, viruses probably remove a portion of the primary productivity, as lytic viruses occur in sea water and must be produced at the expense of hosts. As viral infections propagate rapidly when host density becomes high⁵, viruses may also indirectly affect primary productivity by modifying community structure. In other situations viruses could stimulate primary productivity by causing cell lysis and enhancing the rates of nutrient recycling, analogous to the effect of grazers in some planktonic communities⁶. Observations that aquatic viruses infect particular hosts over wide geographic areas^{7,8}, that a significant portion of marine cyano-

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