cate that year-class formation occurs as a consequence of changes in life-cycle length.

The question that now needs to be addressed is, are these pre-1868 cicadas genetically 13-year or 17-year? If we can obtain alcoholic or dried specimens from 1829, 1842 or 1855, we can determine their mitochondrial genotype through DNA amplification and sequencing as we have done for other preserved periodical cicadas (Simon and Pääbo, unpublished data). A search is underway; anyone with information on such specimens is urged to contact us.

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Parasites and sexual selection

SIR—Recent discussions about the prevalence of blood parasites and colour in birds[1,2] have resurrected a debate which began in 1982 (ref. 3) but aroused little interest among parasitologists who had hoped that the whole matter would die quickly and quietly. In my view, all this work is strong on evolutionary theory and statistics but so weak on basic parasitology that the fundamental basis upon which the edifice has been erected is suspect.

The basis of the work has been published data on parasite numbers in stained blood smears, which are notoriously difficult to interpret even by experts. Overall, a single blood smear presents only a tiny window through which real infection can be glimpsed. It requires an almost superhuman effort to examine the whole of a blood smear properly, so short cuts are usually taken[4]. Furthermore, given the many factors that affect infection, the true prevalence of any blood parasite cannot be determined by single blood smears. It requires several sequential smears taken over a long period of time to be fairly sure, and an immunological or molecular test to be virtually certain that a host is infected with a particular parasite. In this context, Zuk[2] finds it intriguing that rarely trapped birds show a stronger correlation between brightness and parasites more than commonly caught birds. The explanation for this is simple — and here I plead guilty — for when examining blood smears, one takes much more care if they come from rarer birds because these are more likely to produce observations justifying publication.

We know practically nothing about the pathology of many blood parasites that infect birds, and what we do know comes largely from laboratory observations or anecdotal evidence. Some parasites can kill their hosts, but some hosts exhibit a phenomenon known as premunition during which a tolerable infection prevents the establishment of a less tolerable one. Furthermore, other concomitant infections cannot be ignored because blood parasites are markedly affected by the presence of other parasites, bacteria or viruses. Practically nothing is known about immunity to parasites in passerines and it is futile to try to make any assumptions about the ways in which these blood parasites affect the lives of their hosts.

It therefore does not seem surprising that there is no agreement as to whether there is any connection between the colour of birds and the presence of blood parasites. To a parasitologist, the only suggestion that makes any sense is that of Read and Harvey[1] who suggest a phylogenetic relationship. It would be interesting to see this followed up in a systematic way.

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HAMILTON AND ZUK REPLY—Before responding to Cox, it is necessary to deal with inaccuracies in Read and Harvey's response[1] to Zuk[2]. Her claim (on which both of us, as authors of the original paper[3], agreed) was that Read and Harvey's reanalysis[4] in fact produces significant correlations between male brightness and parasite prevalence in passerine birds. This is not wrong. Read and Harvey[1] evidently mean that it would be wrong to claim such correlation after their procedures for removing 'taxonomic association' have been applied, but Zuk's letter was not referring to this case but to ordinary cross-species comparison, as in our original paper[3]. Almost every dataset has some potential classification underlying it which has not yet been 'controlled'. It is not wrong to say that a study on a school class reveals a 'significant correlation of exam mark with writing speed' just because there are several ethnic groups in the class. 'Controlling' an underlying complex of relationships in such a case may clarify and it may also deny evidence of an important cause. However, even had we made the claim that Read and Harvey criticize we would still be half right: their report tells that on the side of the Atlantic where the new scorers were treating familiar birds the association within taxa had $P=0.008$.

They seem to reject this because it reduces if species with small sample sizes are excluded. That new fact is very interesting but does not contradict either our actual claim or the stronger one they treat as made. As to reasons, we suggested one which Harvey and Read reject. Whether that argument is wanting or not, a point not clear to us, another that can be independent or auxilliary is confidently suggested by Cox in his letter in this issue. Thus, it now seems that the most carefully assessed bird species — those rarely caught, on Cox's authority — after scoring by local ornithologists and statistical removal of taxonomic artefact, accord very well with our expectation[4,5]. Thus the combination of data and analysis which all five contributors to this correspondence most nearly agree to be valid rejects no association with $P=0.003$ (two-tailed) and shows association in the direction we predicted[4].

That more infections are apparently added for showy birds than for dull when smears are carefully searched is also interesting. Low-level infections in showy birds fit well with theory because sexually selected species, co-evolving faster, may be better able to keep them low. Read and Harvey also suggest this in a different context, claiming we discounted the possibility. We did not, although we do expect sexual selection only to be able to diminish parasite incidence, not eliminate species.

Cox's comments on the weaknesses of the data are similar to those we found in most of the original papers. However, all authors who reported data had enough faith in their methods to think it worthwhile to tabulate numbers of birds caught and cases found. Provided there is no reason to expect a capricious relationship between observed and true incidence our test can proceed. It is remarkable and reassuring that from such weak data all the 'showiness' calculations, now done for three continents with contributions from three sets of scorers, have uncovered correlations of the same type.

We share Cox's wish for better understanding of the parasite conditions. We would especially like to see chronic virus conditions included in cross-species surveys. We do not agree with Cox, however, that it is futile to assume that passerine parasitaemias are usually detrimental, even though some studies on particular genera have failed to show this[6]. Both the known immense impact of disease associated with parasitaemia in humans and domestic animals, and a well-supported theory of how virulence might be maintained when parasites have mobile vectors[7], reinforce this assumption. Even if it is true that certain infections can be