range and compete with their parents and each other. This could explain the unexpected negative correlation between litter size and ECF[1].

Second, it might be expected that competition between siblings should occur post-weaning and before dispersal at or around sexual maturity in dispersing species[10]. Age at maturity is highly correlated with body size[13], but age at weaning is only weakly correlated with body size[14]. Therefore, the offspring of larger species will tend to remain together in the natal area even after they have been weaned. This will not be true for smaller species. The offspring of species with greater age at maturity are, therefore, likely to encounter greater sibling competition than those which mature earlier.

In conclusion, Burt and Bell's test of the Tangled Bank and Red Queen hypotheses using ECF frequencies was carried out using only partially valid hypotheses. I am not convinced that either theory can currently be discounted as accounting for the evolution of sex and recombination.

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SIR—Burt and Bell[1] used a correlation of excess chiasma frequency with age at maturity to support the Red Queen model over the Tangled Bank model as the selective factor maintaining genetic recombination. The theoretical basis of this has been discussed[2–5]. The test raises the practical questions of the suitability of the organisms used and the extent to which excess chiasma frequency is influenced by selection for variation among offspring.

Mammals are rather unrepresentative species and have good powers of dispersal, which reduces sib-competition. In some species such as chimpanzees *Pan troglodytes* and rhesus macaques *Macaca mulata* (both used by Burt and Bell), relatives of one sex cooperate (negative competition) to defend territories. This makes it unlikely that they will show good evidence of sib-competition. Sedentary organisms with a large brood size would be better. Plants could provide the best examples, as beside sexual species, partially asexual, outbreeding and selfing species could be used to compare excess chiasmata with age at maturity.

An increase in chiasmata does not necessarily increase recombination. For example, replacing a single interstitial chiasma with two near the telomeres because of interference reduces real recombination. Also, excess chiasma frequency is not entirely independent of chromosome number. It is uncommon for bivalents to have more than six chiasmata and excess chiasmata per bivalent seems to be a better parameter to measure. Dipteran flies all have a low haploid number and low recombination index irrespective of a generation time of days or years, typically with no recombination in males[6]. Differences in length between individual bivalents may also have an effect if recombination levels which ensure a crossover in short bivalents tend to produce several crossovers in long bivalents.

Chiasma frequency variation and distribution between sexes and species of annual temperate grasshoppers with similar karyotypes suggests that recombination has little to do with age at maturity, one year from egg laying. Some species have many excess chiasmata, whereas in others males have a single, terminally localized chiasmata in each bivalent arm so that there is no recombination in most of the genome. The females may show similar, opposite, or no localization[7], and more or less excess chiasmata. Similar sex-specific differences are shown by *Triturus* (newts)[8] and *Equus caballus* (horse)[9]. In lepidoptera, females (the heterogametic sex) lack recombination. Selection for or against recombination cannot explain differences between sexes as chromosomes from both sexes end up together in each offspring. Physiological differences must be the prime factor, if only by preventing changes in recombination in one sex. Meiosis is a long process and spermatogenesis may take weeks to complete. We have suggested[7] that reduced recombination may streamline sperm production in some species, particularly in insects where females need only mate once for life, and the males are relatively small and fast maturing. A similar effect might apply to the mammals used by Burt and Bell[1], presumably mainly males, since quantitative data on female meiosis is rare. Those with rapid maturation probably have more mating opportunities per week than slow maturing types, and may produce more sperm per kg body weight per unit time.

A phenotypic character like excess chiasma number can occur under several different selection pressures, and a universal explanation is unlikely. The relative effect of the Red Queen and Tangled Bank models will depend upon the ecology of individual species. The response may be limited by other factors. The risk in accepting Burt and Bell's conclusions is that the other factors affecting chiasma frequency will be ignored. As a general rule in evolution, if something is possible and selectively advantageous, some organism somewhere will be doing it. But two species may not do it for the same reasons.

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Protein kinase C and cyclic AMP pathways cross-talk

SIR—Levitzki[1] argues that in intact cells the most likely mechanism by which activation of protein kinase C by phorbol esters potentiates the synthesis of cyclic AMP is via G_i, the inhibitory G protein of adenylate cyclase. The phosphorylation of the α subunit of G_i by protein kinase C, which has been observed in platelet membranes[2], would remove tonic inhibition of cyclase activity. In support of his argument Levitzki states that the effect of phorbol esters is quantitatively similar to that of pertussis toxin[3], which ADP-ribosylates and inactivates G_i. We would like to present an alternative hypothesis based on our recent studies with cultured cells[4].

These findings suggest that there is a G protein that is sensitive to pertussis toxin and that may act, at least in some cells, as a mediator of 'cross-talk' between protein kinase C and adenylate cyclase activity. In its phosphorylated form the G protein would activate the catalytic subunit and its phosphorylation by protein kinase C would be prevented by pertussis toxin. Note that there are a number of recently identified pertussis toxin substrates to which no function has yet been ascribed[5].

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