stable in the extreme[16] and isoperiodic only in the most unusual conditions[4,17].

In conclusion, we would like to emphasise that Winfree's elegant experiments have revealed certain aspects of the dynamic behaviour of the circadian regulator of the *Drosophila* eclosion rhythm and represent a prime example of the use of mathematical models for understanding the nature of biological mechanisms. It is only unfortunate that the paper chose to emphasise especially in its title and section titles only two of the many hypotheses still available. Furthermore, most of these hypotheses are very "clocklike" as long as the term "clock" is used to mean a general time-keeping mechanism.

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WINFREE REPLIES—There seem to be three issues, all resolvable. First, Aldridge and Pavlidis[1] rightly observe that my experiments[2] exclude only "quickly recovering" limit cycle models, by virtue of showing no change in a measure of the amplitude of a circadian oscillator during 48 h after each of 30 different adjustments of that amplitude. "No change" means roughly "less than ± 10%". They rightly object that my adjective "quickly recovering", used everywhere else, was deleted from a section heading which then read "limit cycle excluded experimentally"—whereas a very slow recovery of amplitude remains unexcluded, as does a very slow decay of amplitude.

On the other hand, according to my data, the spiralling of trajectories in the descriptive metaphor of their Fig. 1 is definitely too fast: almost all trajectories strike arc *AD* within 48 h, ensuring complete recovery to a standard cycle, contrary to observations.

Second, Aldridge and Pavlidis agree that my experiments suggest a long-lasting heterogeneity of state among two-to-many similar circadian mechanisms with each animal. I chose to emphasise phase incoherence and that this could derive from relative independence of these autonomous (possibly cellular) mechanisms. Assuming such independence and persistent incoherence in a population, the metaphor of Fig. 1 (among others) is perfectly compatible with the emphasised peculiarities of the circadian rhythm of *Drosophila*. This was intended to be the main point of my "Unclocklike . . ." paper in *Nature*[3]. Aldridge and Pavlidis chose to emphasise an alternative possibility, that some oscillators in the population are switched off, the others remaining synchronous. This model abandons Fig. 1 for a more complicated drawing with two limit cycles (one unstable) and supposes rapidly synchronising interactions among cells. I agree that this is a sensible alternative, which I overlooked. When a histological assay of circadian state becomes available, the two models—one predicting phase heterogeneity and independence, one predicting amplitude heterogeneity and coupling—should be clearly discriminable.

The third point is terminological. It is usual for familiar words, adopted into a specialised area, to change their connotation as perspectives change and scholars make more refined distinctions. As adopted in the 1950s "clock" connoted little more than adaptive stability of period. But by the mid-1960s much of the literature makes sense only if a more restrictive implicit connotation is recognised, namely what is distinguished in the cell-cycle literature[4] as a "simple clock". This is a mechanism which, like a commercial clock or a music box, unlike a dynamical oscillator, can change only its phase, having no states off a unique causal cycle. In pointing to the singularity and to amplitude lability as two "unclocklike" features of the circadian mechanism, I have adhered to this more restrictive usage, distinguishing "clocks" from "dynamical oscillators". In contrast, Aldridge and Pavlidis apparently use "clock" to mean "dynamical oscillator" as opposed to such alternative periodic mechanisms as a sequential state machine with one fixed cycle. Thus the "clocklike" features of their paper[1] are the same as the "unclocklike" features of mine[3]! I notice that Pittendrigh has simply abandoned the word in entitling a current preprint ". . . circadian pacemakers".

I am delighted to have this critical exchange, and wish there were a lot more of it in the circadian literature.

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Structure of the galactic magnetic field

RECENTLY Somogyi[1] has presented arguments indicating that the power spectrum of the turbulent galactic magnetic field is of the form $k^{-\alpha}$, where k is the wavenumber and $\alpha = 1.0$–1.8 depending on the model of cosmic-ray transport assumed. This spectrum is relevant for scales of 0.001–1 pc, and was obtained by noting the dependence of the cosmic-ray anisotropy and pathlength on energy. I wish to show that a similar conclusion follows from a related but independent argument based solely on the average cosmic-ray pathlength and observed properties of the large scale galactic magnetic field[2].

Consider a simple one-dimensional model for the diffusion of cosmic rays perpendicular to the plane of the Galaxy, with constant diffusion coefficient $K_\perp$ and free escape at the boundaries $z = +L$ and $z = -L$. For observers near the galactic disk ($z = 0$) it is straightforward to show that the mean path λ is given by

$$\lambda = nm_{\rm H}c\langle t\rangle = (5/12)nm_{\rm H}cL^2/K_\perp \quad (1)$$

where n is the interstellar density of hydrogen, $m_{\rm H}$ is the mass of a hydrogen atom, c is the speed of light, and $\langle t\rangle$ is the average cosmic-ray lifetime[2,3]. From equation (1), one may infer a value of $K_\perp$, given the other parameters, and for those listed in Table 1, the value of $K_\perp$ varies from 1.2×10^{26}–1.2×10^{28} cm^2 s^{-1}, with a nominal value of $\sim10^{27}$ cm^2 s^{-1}. This value corresponds to the 'observed' value of the cosmic-ray diffusion coefficient for those particles (with energies ~5 GeV per nucleon) which are responsible for spallation.

Next consider the derivation of the diffusion coefficient from the properties of the random interstellar magnetic field. Using standard diffusion theory[4,5], in the limit that the gyroradius r_g of the particle is much smaller than the magnetic-field correlation length l_o, one obtains[2,6]

$$K_\perp = F(\alpha)cl_o(r_g/l_o)^{2-\alpha} \quad (2)$$

Here $F(\alpha)$ is a factor of order unity[2], and it has been assumed that the magnetic-field power spectrum is flat for wave