Hesperornis (Fig. 3*b*) and *Sphenosuchus*[2,3] had perilymphatic ducts which issued laterally from the lagenar recess into a fenestra pseudorotunda. The fenestra probably arose from a canal for the ninth cranial nerve which was 'captured' by a lateral shift of the perilymphatic duct. This eliminated the necessity for an unossified vestibular region in the braincase wall (as the duct no longer used this space to leave the otic capsule) and allowed the complete internal ossification of the adult otic capsule, a feature already present in *Hesperornis* and the Triassic crocodile, *Notochampsa* (BMNH R 8503). Although other living vertebrates (mammals and lizards) also have round windows, only in birds and crocodiles is this feature formed, embryologically, by a subcapsular process. This process is a cranial rib, ossified in the adult, which divides the metotic fissure and forms the floor and posterior wall of the fenestra[12]. In the adult it also forms a laterally directed flange, fused to the combined exoccipital/opisthotic bone at the back of the middle ear cavity, and supporting the secondary tympanic membrane (Fig. 1*d*). Such similarity in the developmental process would be unlikely if the fenestra pseudorotunda of birds and crocodiles was independently derived.

An additional feature shared by birds and crocodilians is a system of pneumatic spaces in the bones surrounding the middle ear (Fig. 3). An upper pneumatic chamber joins the middle ear cavity beneath the lateral shelf of the squamosal bone at its junction with the prootic and exoccipital/opisthotic bones. Lying just above the principal braincase articulation with the quadrate, this cavity extends medially and dorsally into the supraoccipital and parietal bones. A second chamber, below and behind the quadrate articulation, extends into the base of the paroccipital process. These periotic sinuses also occur in Mesozoic birds (*Hesperornis*) and Triassic crocodilians (*Notochampsa*), but are absent in theropod dinosaurs and other sauropsid reptiles. Functionally, periotic pneumatic cavities represent an enlargement of the air cushion which lies medial to the tympanic membrane. They are thus analogous to the enlarged auditory bullae or pneumatic petrosal bones of mammals. The dampening effect of the air cushion is such that a large middle ear cavity is better suited for low frequency sound, and a small cavity is better suited for high frequency[20,21].

These advanced features in the ear region support a hypothesis of common ancestry for crocodiles and birds, independent of both saurischian and ornithischian dinosaurs. A hypothetical common ancestor having a fenestra pseudorotunda and periotic pneumatisation, but also having the primitive features retained by either birds or crocodiles (for example, cranial kinesis and 'semi-improved'[22] locomotion), would currently be classified within the paraphyletic grade, Pseudosuchia. A theory of a pseudosuchian origin for birds is, at the very least, a reasonable alternative to the dinosaur model proposed by Ostrom.

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Transplantation of a circadian pacemaker in *Drosophila*

A MAJOR question in the physiology of activity rhythms is the nature of the coupling between the pacemaker and the motor system. There is good evidence for humoral coupling in several species. In the sparrow, pineal organs from donor birds maintained in a light–dark cycle different from that of the host were transplanted into the anterior chamber of the eye of recipient birds made arrhythmic by pinealectomy. In this case the activity rhythm of the host was restored and its phase was determined by a diffusible humoral factor from the implanted pineal[1]. In the mollusc *Aplysia* humoral coupling between the circadian pacemaker and the output cells is thought to control locomotor activity, although electrical coupling has not been ruled out[2]. In insects, however, experiments involving cockroaches and silkmoths indicate that an intact electrical connection between the brain and thoracic ganglia is required for rhythmic activity. The pacemaker controlling the locomotor activity rhythm in the cockroach appears to reside in the optic lobes, and disruption of the electrical pathway between the optic lobes and thoracic ganglia results in arrhythmicity[3–6]. Initial transplant experiments with this insect indicated that activity could be hormonally controlled by the sub-oesophageal ganglion[7]. While these results have not been repeatable by other laboratories[8–10], evidence from parabiosis experiments does suggest some humoral influence on the activity rhythm[11]. Surgical experiments using giant silkmoths have also demonstrated the need for an intact neural connection between brain and thorax for expression of the flight activity rhythm[12]. Thus, until now, there has been no unambiguous evidence for humoral control of activity rhythms in insects. Here we show that a short-period mutant brain can produce a short-period activity rhythm when implanted into the abdomen of a genetically arrhythmic host. In this instance, there were no functional neural connections between the implanted brain and the locomotor system of the recipient, so the action of the brain must be mediated by humoral influences.

Adult brains from short-period mutant (per^s) animals which normally exhibit 18- to 20-h activity rhythms in constant environmental conditions[13] were transplanted into the abdomens of arrhythmic mutant (per^o) adults whose activity in these conditions is aperiodic. Of 55 individuals surviving the operation, 4 showed short-period activity rhythms for at least three consecutive cycles (four activity bursts) (Table 1). Figure 1*a* shows the activity of the A-21-24 arrhythmic host containing a

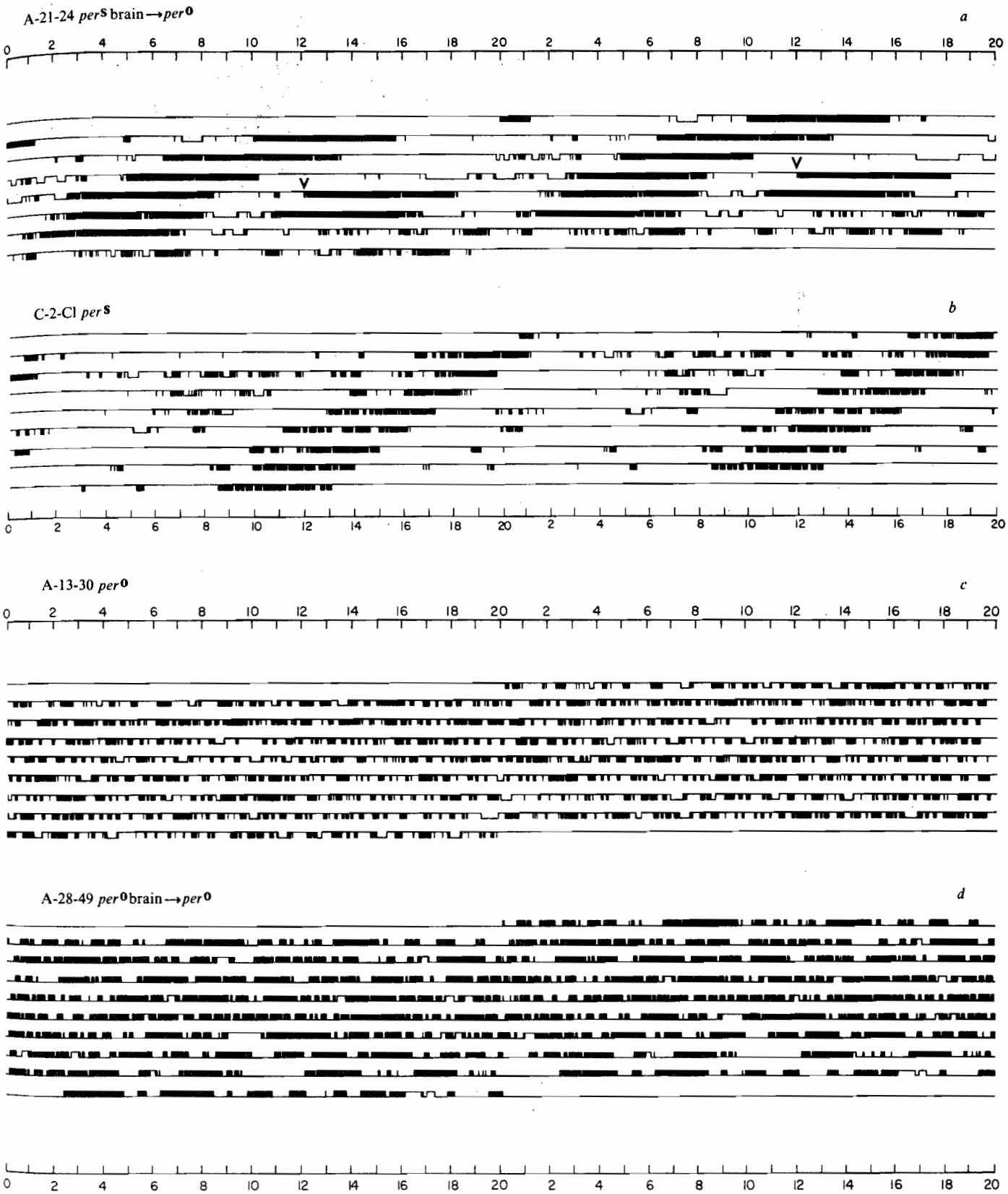

Fig. 1 Locomotor activity of individual adult *Drosophila melanogaster*. *a*, A *per*° fly implanted with a *per*^{s} brain; *b*, an unoperated *per*^{s} fly; *c*, an unoperated *per*° fly; *d*, a *per*° fly implanted with a *per*° brain. Event recorder data of activity monitored in IR light using a device similar to that described previously[13]. Data plotted modulo 20 h. Each line indicates the start of successive 20-h intervals. Successive intervals are also plotted to the right of the immediately preceding interval for visual continuity. Female flies bearing the *per*° or *per*^{s} mutation were raised in identical 12 h white light–12 h dark cycles. One to two days after eclosion, brains from *per*° and *per*^{s} donors were dissected in *Drosophila* Ringer's solution[15] and injected with Ringer's into the abdomens of *per*° hosts, using a glass micropipette with a small constriction. Host animals were kept in a moist environment for 1 d to facilitate wound healing. Two to four days later, surviving hosts and unoperated *per*° and *per*^{s} controls which had been raised in the same light–dark cycle were inserted into the activity monitor. Locomotor activity was usually recorded for an initial 2-d entrainment period which continued the previous light–dark cycle, using long-wave UV light. Flies were then maintained in constant conditions (24 °C) for an additional 7 to 10 d during which locomotor activity was monitored in IR light. Events were also counted each hour and the digital data used for periodogram analysis.

Table 1 Activity rhythms in *per*° hosts implanted with a *per*ˢ brain

Host	No. of cycles	Average period* (h)
A-21-24	6	17
A-23-79	5	16
A-23-76	4	10 and 20
A-23-27	3	18

* Average period obtained by periodogram analysis of digital activity data.

*per*ˢ brain in its abdomen. A clear short-period rhythm persists for five cycles, with a sixth cycle that is less clear. (An additional activity component with a similar period appears briefly in the record, shown by the arrow; the beginning of this activity burst coincided with a food change and probably resulted from the mechanical disturbance or the stimulus of the fresh food.) The period of the activity onsets and offsets increases slightly during the course of the record, possibly because of degenerative changes in the implanted brain. The end of the first clear activity burst occurs 69 h after the end of the last entraining light pulse, consistent with the proposition that the phase of the rhythm was set by the last light signal seen by the donor animal.

In addition to A-21-24, three other *per*° hosts with implanted *per*ˢ brains showed short-period rhythmicity (Table 1). One of these, A-23-76, showed evidence of a bimodal rhythm. Activity rhythms of wild-type and *per*ˢ flies are typically bimodal in a light–dark cycle[14] and occasionally bimodal in constant environmental conditions (R. K., unpublished results).

Figure 1*b* shows the activity rhythm of an unoperated *per*ˢ fly for comparison. In addition to the similarity in periodicity of the rhythms in Fig. 1*a* and *b*, the duration of the activity bursts (5–7 h) in both records is typical of the reduced activity span in *per*ˢ mutants compared with wild-type (10–13 h). Unoperated *per*° flies were always aperiodic (Fig. 1*c*)[13,14]. Furthermore, none of the *per*° hosts containing implanted *per*° brains showed any evidence of persistent rhythmicity ($n = 48$, Fig. 1*d*). Therefore implantation of extra brain tissue is not in itself sufficient for rhythmic activity.

After the activity of the *per*° hosts implanted with *per*ˢ brains had been measured, surviving animals were dissected and the transplanted brains examined. Transplanted brains were morphologically recognisable in ~70% of surviving hosts 2 weeks after the operation. The remaining donor brains probably degenerated soon after the operation. Eleven donor and host brains from animals whose activity was not monitored were stained using a silver impregnation technique 2 to 4 weeks after the operation and examined under the light microscope for degeneration. All donor brains and some host brains showed some degree of cell death and neuropil degeneration, resulting in vacuolation of the tissue. The low percentage of transplants showing short-period activity rhythms may be due in large part to degeneration which ensues after dissection and implantation.

We conclude that a brain isolated from a short-period mutant fly can produce its characteristic activity rhythm when implanted into a genetically arrhythmic host. It is highly unlikely that the donor brain in the abdomen could establish functional neural connections with the host nervous system in the thorax during these brief experiments, so the conferred rhythmicity must be due to a diffusible humoral factor secreted from the donor brain. The factor then acts on the nervous system of the host, either directly on the motor centres or indirectly via the host brain, to elicit the locomotor activity rhythm. The induced activity rhythms in successful transplants were of short-period and not wild-type, so it is unlikely that the donor brain somehow complemented the defect in the host's arrhythmic pacemaker system, allowing it to function normally. The fact that activity records of successful transplants do not show aperiodic background activity at all times indicates that the factor from the implanted *per*ˢ brain is inhibitory, limiting the activity of the *per*° host to discrete periodic intervals, with an absence of activity during intervening periods.

The demonstration of a humoral component in the control of *Drosophila* activity rhythms does not, of course, rule out the possibility of an additional neuronal pathway in intact flies.

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Androgen-insensitive rats are defeminised by their testes

SOME chromosomal XY male humans[1], mice[2,3] and rats[4] are born with a vagina, labia and clitoris instead of scrotum and penis. The expression of the female phenotype in these individuals is thought to be due to an insensitivity to androgenic hormones which are secreted by the testes. This phenomenon has been termed the androgen-insensitivity syndrone or testicular feminisation. Typically, androgen-insensitive humans are identified and adopt a female gender identity[5]. Androgen-insensitive rats, on the other hand, seem to be incapable of showing a high degree of feminine behaviour in adulthood even when given oestrogen and progesterone[6,7]. It is shown here that androgen-insensitive male rats are capable of responding to ovarian hormones with the display of feminine sexual behaviour if they are castrated at birth but not when castrated 10 d after birth. Although these rats are insensitive to their testicular secretions in terms of genital differentiation, the neural substrate of sexual behaviour is differentiated by the presence of testes during the first few days after birth.

Androgen-insensitive male rats of the Stanley-Gumbreck strain ($n = 16$) and their male littermates ($n = 19$) were castrated within 24 h of birth or 10 d after birth at the International Foundation for the Study of Rat Genetics and Rodent Control (Introgen), Oklahoma City. At weaning, the rats were sent to our animal facilities and were maintained in pairs on a reversed light–dark cycle with *ad libitum* food and water.

At 75–98 d of age, the subjects were administered 2 μg of oestradiol benzoate 48 and 24 h before injection of 500 μg of progesterone; testing began 3–4 h after the progesterone injection. Each subject was placed in a glass observation cage with a sexually vigorous male rat. The male was allowed to mount the subject 10 times. The occurrence of each lordotic response was recorded and a lordosis quotient (*LQ*) was determined ((number of lordotic responses per mount with thrusts) × 100). Each subject was tested twice during a 2-week period.