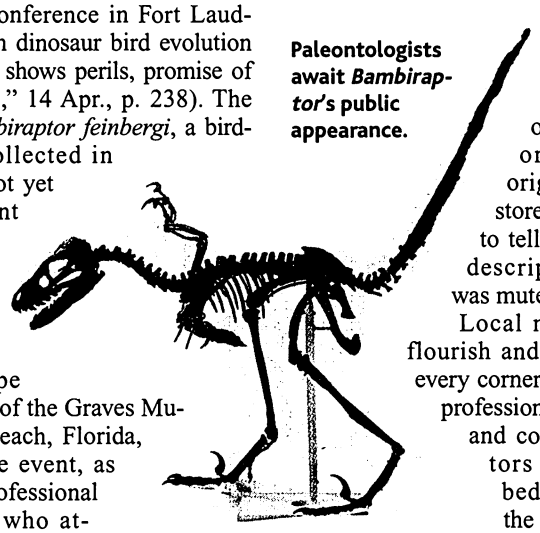
Concerns over the privately owned *Bambiraptor* fossil are raised, and it is acknowledged that "professional paleontologists and commercial collectors remain strange bedfellows." In the aftermath of a postdoc's failed attempt to sue her mentor, it is observed that "Clear rules need to be followed nationwide for conferring co-inventor status to appropriate students and postocs involved." The issue of whether a circadian period "observed under specific experimental conditions may best be referred to as the 'spontaneous' frequency of the pacemaker" or whether there is an intrinsic circadian period is discussed.

A Home for *Bambiraptor*

There were several omissions in Constance Holden's News of the Week article about the "*Bambiraptor*" conference in Fort Lauderdale, Florida, on dinosaur bird evolution ("Florida meeting shows perils, promise of dealing for dinos," 14 Apr., p. 238). The specimen of *Bambiraptor feinbergi*, a bird-like dinosaur collected in Montana, does not yet have a permanent home in a public museum, contrary to what is implied in the article. That is certainly the hope of the directorate of the Graves Museum in Dania Beach, Florida, which hosted the event, as well as of the professional paleontologists who attended the conference. Graves Museum officials arranged the private purchase of the specimen with the laudable stipulation that it would be donated to a public museum.



CREDIT: GRAVES MUSEUM

Paleontologists await *Bambiraptor*'s public appearance.

However, because the specimen is still privately owned, its publication (*1*) is problematic for many paleontologists. The Society of Vertebrate Paleontology, for example, has an ethics statement opposed to the commercial sale of important vertebrate fossils and will not publish in its journal any specimens not in the public trust. The ambiguous status of *Bambiraptor* caused a number of professional paleontologists to decline to attend the meeting.

The specimen in question was excavated and sold by then-amateur collectors. Some parts of the skull and other bones of the skeleton were severely damaged (*1*). Consequently, it is difficult to tell by comparisons whether this is a juvenile specimen of a new taxon or of a taxon that is already known. The precise systematic position of the specimen was not discussed in its description (*1*), nor was its relevance to bird origins, the origin of flight, or other larger questions advertised in the meeting's publicity. Because the specimen is mounted in a restored position, it is difficult to study the original material, or to tell what is original, what is restored, and what it has to tell us apart from its description (*1*), which was mute on these issues. Local museums should flourish and bring culture to every corner of the world. But professional paleontologists and commercial collectors remain strange bedfellows. Neither the public nor the national heritage will be served by publicizing specimens that are not collected, reposited, and documented according to the standards of professional science.

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Intellectual Property Rights

In reference to Eliot Marshall's News Focus article entitled "Patent suit pits postdoc against former mentor" (31 Mar., p. 2399), *Science* should be commended for giving appropriate prominence to such issues involving intellectual property rights in academic institutions.

The material presented in the article leads me to believe that an injustice has been meted out to Joany Chou. First, if Bernard Roizman, Chou's former lab chief, considered himself to be the "sole inventor," then why do important papers supporting the patent have other authors? Second, Judge James Zagel's ruling that the University of Chicago (UC) owns the patent is correct, but it does not debar Chou from "co-inventor status." And third, although UC officials say that "Dr. Chou has been treated fairly," I have reservations about accepting that statement; it is not unheard of for university officials to avoid treading on faculty members' toes by not supporting students and postdocs, however strong the case may be. Nor is it unheard of for faculty advisors to describe accidental discoveries as the outcome of preconceived, systematic, logical questioning in order to claim credit. In many situations, an advisor may not even be aware of important findings until a student brings them to the advisor's attention.

Clear rules need to be followed nationwide conferring co-inventor status to the appropriate students and postdocs involved, and students and postdocs need to be informed of what their share of the credit should be for discoveries that ensue from their hard labor. These steps are essential to restore the faith of junior scientists in the future of science careers, halt the perpetration of questionable scientific practices, and restore the trust between students and faculty advisors that has been greatly eroded by the lure of money.

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One thing that Marshall mentions in his article but that is worth reemphasizing is that Chou does hold a patent on the gene $\gamma_1$34.5: patent number US5,834,216, "Screening methods for the identification of inducers and inhibitors of programmed cell death (apoptosis)." Additionally, from my experience as a former member of Roizman's laboratory (student and postdoc) and as someone who has been involved in the patent process with Roizman (well before this lawsuit), for those who made an original intellectual contribution in the laboratory that was patentable, Roizman included them on the patent and they had an opportunity to negotiate with him as to how to divide the credit (future payoff, if any). A quick check of the patent database (*1*) shows 16 patents for Roizman, of which only 5 have Roizman as the sole inventor. If he wasn't intellectually honest, I would expect 16 out of 16, which is his right under the current state of affairs for postdocs and graduate students according to the rule of law.

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1. A patent database is available at www.patents.ibm.com/ibm.html

Is There an Intrinsic Period of the Circadian Clock?

In their report "Stability, precision, and near-24-hour period of the human circadian pacemaker" (25 June 1999, p. 2177), C. A. Czeisler *et al.* describe that under the experimental conditions of "forced desynchrony," the human endogenous pacemaker exhibits a period averaging 24.18 hours. They further report remarkable precision of the clock and suggest that both are "intrinsic" components of the human circadian pacemaker. Such a characterization may be misleading because it implies (and the authors articulate) that a rhythm measured under any other conditions is merely the expression of an "apparent period" of the biological clock.

It has been recognized since the late 1950s that the free-running circadian periods of laboratory animals depend on the experimental conditions under which they are measured. Indeed, one of the tenets in chronobiology is Aschoff's rule, which defines the differential responses of the circadian pacemakers of nocturnal and diurnal species to changes in light intensity (*1*). Which, then, should we call the intrinsic period of, for example, the finch's clock? That observed under constant lighting with an intensity of 0.4 lux, or the longer period that is observed when the bird is studied under 8 lux? Both are clearly endogenous periods, but it is unlikely that one reflects the essential nature of the pacemaker more so than the other. To the contrary, the essential nature of the pacemaker is reflected in its capacity to adapt to changing conditions.

As Czeisler *et al.* point out, the average free-running period of the human circadian clock (as determined by body core temperature) has been measured in a range from 24.2 to 25.1 hours. What distinguishes these various estimates of period length is the experimental conditions under which they were obtained. For example, we showed that when individuals in an otherwise traditional time-free environment took advantage of instructions to "eat and sleep when so inclined," by averaging at least one nap per subjective day, they exhibited an average period length of 24.22 hours, compared with an average period of 24.73 hours for individuals who seldom or never napped (*2*). One interpretation offered at the time to explain this finding was that some aspect of the traditional paradigm (which prohibits napping) might be responsible for "artificially lengthening the intrinsic free-running period" (*2*, p. 640). Seven years later, it seems clear that neither period estimate reflects the intrinsic period of the clock. Rather, both reflect the clock's intrinsic response to a distinct set of environmental or experimental conditions.

The forced desynchrony protocol used by Czeisler *et al.* presents the circadian system with yet another set of experimental conditions under which it must function. In this paradigm, the clock is forced to free-run against a strictly controlled background of reduced ambient light and altered subjective day lengths. The result is a strictly maintained (that is, "precise") rhythm with a characteristic period. For the authors to conclude that this particular set of conditions in some way evokes a more accurate reflection of the pacemaker's intrinsic period than other paradigms seems to beg the question.

As Aschoff emphasized 40 years ago, "The free-running period we can observe in an organism is, of course, nothing like a physical constant. Organisms as open systems are always correlated to the environment. The actual value of the rhythm, the frequency, is determined by all circumstantial conditions—external as well as internal" (*3*). To avoid any inference as to the intrinsic nature of an observed period, Aschoff suggested that an endogenous rhythm observed under specific experimental conditions may best be referred to as the "spontaneous" frequency of the pacemaker. Such a designation seems to capture more clearly the essential nature of the biological clock.

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Response

Circadian period is a fundamental, genetically inherited property of the circadian pacemaker. We do not agree with Campbell that observed circadian periods in humans are highly dependent on environmental or experimental conditions and that the forced-desynchrony protocol no more accurately reflects the intrinsic period of the human circadian pacemaker than a classical free-running paradigm. We also note that the late Jürgen Aschoff began conducting free-running studies in humans around 1960, shortly after the cited reference (*1*), and with Wever concluded more than two decades later that, "With a sample of 147 subjects, the overall mean of the [free-running circadian] period...was found to be 25.0...± 0.50 hr. The period of a free-running rhythm [in humans] is furthermore quite independent of conditions..." (*2*). In fact, on the basis of those findings, the concept that humans have an internal clock with a 25-hour period is included in numerous biology, physiology, and psychology textbooks.

Yet, in a *Science* review article shortly after the initial human free-running studies, Aschoff allowed that one (of three) possible causes of the considerably longer than 24-hour free-running circadian periods that he and others had observed in their now classical free-running experiments was "feedback between the subject's endogenous activity cycle and the self-selected periodic stimuli—that is, turning the lights on and off" (*3*), as he had seen in birds (*4*). He recognized that more data were required to "allow a final decision" on this matter (*3*).

The goal of our study was to provide those data. Individual neurons composing a central neural pacemaker of the mammalian circadian timing system (located in the suprachiasmatic nucleus of the hypothalamus) each contain a transcriptional/translational feedback oscillator or oscillators displaying a circadian period that is under genetic control; when coupled together, these ~10,000 neurons and their core oscillators form a pacemaker (*5*). We attempted to estimate the intrinsic circadian period of this central circadian pacemaker in humans, as measured immediately upon release from entrainment to the 24-hour day, by using a forced desynchrony protocol and measuring output rhythms directly driven by the pacemaker, such as melatonin. By "intrinsic," we mean the period originating from within (*6*) the circadian pacemaker, as distinct from other observed circadian periods influenced at the time of study by extrinsic resetting stimuli continuing to act on the pacemaker. This pacemaker is a dynamical system that rarely shows its intrinsic properties in humans, because it is nearly always being perturbed by light, changes in the timing of the sleep-wake cycle, transmeridian travel, etc. The pacemaker's responses to these perturbations compose the adaptiveness of the circadian pacemaker to which Campbell refers.

This adaptiveness is directly related to the wide range of observed circadian periods previously reported in humans, because in those experiments, factors that modulate the period of the pacemaker were not adequately controlled. The pacemaker's intrinsic period can only be assessed under conditions in which the main external and internal factors that have been shown to affect the clock (that is, the driving terms of