the dynamical system) are absent or distributed uniformly across the circadian cycle. Thus, for example, the circadian period of the perch-hopping rhythm in the finch observed under 8 lux is further removed from the "intrinsic" circadian period of the finch's circadian pacemaker than the period observed under 0.4 lux, because light imposes a direct drive onto the dynamical system. This is why most genetic studies of circadian period in mammals are carried out in constant darkness.

The efficacy of the forced desynchrony protocol in removing or uniformly distributing these driving factors—as predicted by a mathematical model of this dynamical system (*7*)—is demonstrated by our observation that the observed period of the pacemaker was nearly identical in forced desynchrony protocols with markedly different cycle lengths—for example, 11, 20, 28, or 42.85 hours—and with markedly different levels of physical activity. This is in contrast to the cited "eat and sleep when so inclined" paradigm, in which Campbell *et al.* reported that even though all of the participants were given the same instructions, the circadian period averaged 24.73 hours among those who chose to not nap during the experiment and 24.22 hours among those who did nap (*8*). As Campbell *et al.* noted at the time, their observation thus raised the possibility that intrinsic circadian period differed in nappers compared with non-nappers. In contrast, we consistently observed a near-24-hour intrinsic circadian period (averaging 24.18 ± 0.04 hours), despite the fact that none of the individuals in our experiments was allowed to nap. We thus conclude that the reason Campbell *et al.* observed the near-25-hour period among non-nappers (*8*) was because their sleep episodes and associated light-dark cycles were less evenly distributed across circadian phases, resulting in feedback resetting effects on the circadian pacemaker, rather than representing a systematic difference between those population groups.

Although we claimed to have estimated the intrinsic period of the human circadian pacemaker using this protocol, we do not contend that the period of the human circadian pacemaker is invariant. It has been known for 30 years that prior entrainment influences the intrinsic period of the pacemaker (an aftereffect of entrainment that can last for months) in mammalian species. In fact, as we noted in our report, the slightly longer circadian period observed in blind individuals may in part be a reflection of the absence of such an aftereffect of entrainment to the 24-hour day in some blind people. Such aftereffects do not invalidate the concept of a genetically determined circadian period; natural selection most certainly acted on the parameters of circadian pacemakers in organisms that were entrained to a 24-hour light-dark cycle. We chose to assess the intrinsic period of the human circadian pacemaker immediately on release from entrainment to a 24-hour day because it is this "aftereffected" period that is most relevant for understanding entrainment to the 24-hour day.

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References

1. J. Aschoff, *Pfluegers Arch.* **270**, 9 (1959).
2. ——— and R. Wever, *Biological Rhythms: Handbook of Behavioral Neurobiology*, J. Aschoff, Ed. (Plenum, New York, 1981), p. 311.
3. J. Aschoff, *Science* **148**, 1427 (1965).
4. ———, U. V. Saint Paul, R. Wever, *Z. Vergl. Physiol.* **58**, 304 (1968).
5. E. D. Herzog, J. S. Takahashi, G. D. Block, *Nature Neurosci.* **1**, 708 (1998).
6. *Webster's New Collegiate Dictionary*, 10th ed.
7. E. B. Klerman, D.-J. Dijk, R. E. Kronauer, C. A. Czeisler, *Am. J. Physiol.* **270**, R271 (1996).
8. S. S. Campbell, D. Dawson, J. Zulley, *Sleep* **16**, 638 (1993).

CORRECTIONS AND CLARIFICATIONS

Letters: Response by John Olney under title "Induced damage in the developing brain" (12 May, p. 977). It was an editorial error to list only John Olney as the author of the response to letters by R. W. Montgomery and by A. Sharma and S. Kumar. Olney was the corresponding author for the report under discussion by C. Ikonomidou *et al.*, "Ethanol-induced apoptotic neurodegeneration and fetal alcohol syndrome" (11 Feb., p. 1056). The author list should have been as follows: C. Ikonomidou, P. Bittigau, M. J. Ishimaru, D. F. Wozniak, C. Koch, K. Genz, M. T. Price, V. Stefovska, F. Hörster, T. Tenkova, K. Dikranian, J. W. Olney. *Science* regrets the error.

News Focus: "Science and policy clash at Yucca Mountain" by Richard A. Kerr (28 Apr., p. 602). John Greeves is with the Nuclear Regulatory Commission (NRC). All appearances of "NRC" referred to the Nuclear Regulatory Comission.

News of the Week: "Pruned sanctions list points to closer ties" by Jeffrey Mervis (14 Apr., p. 244). The director of the Aeronautical Development Establishment, a defense institute that remains on the banned list, was misidentified. His correct name is Krishnapuram Gopalakrishnan Narayanan.