like the latter two groups, mammals lack functional extraocular photoreceptors (*28*); thus, redundancy in photoreception is confined to the retina. One challenge is to determine the relative contributions of melanopsin, rod/cone opsins, cryptochromes, and other currently uncharacterized photopigments in communicating photic information to the circadian system.

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Melanopsin (*Opn4*) Requirement for Normal Light-Induced Circadian Phase Shifting

Satchidananda Panda[1,2] Trey K. Sato,[1,2] Ana Maria Castrucci,[3,4] Mark D. Rollag,[3] Willem J. DeGrip,[5] John B. Hogenesch,[1] Ignacio Provencio,[3]* Steve A. Kay[1,2]*

The master circadian oscillator in the hypothalamic suprachiasmatic nucleus is entrained to the day/night cycle by retinal photoreceptors. Melanopsin (*Opn4*), an opsin-based photopigment, is a primary candidate for photoreceptor-mediated entrainment. To investigate the functional role of melanopsin in light resetting of the oscillator, we generated melanopsin-null mice (*Opn4*$^{-/-}$). These mice entrain to a light/dark cycle and do not exhibit any overt defect in circadian activity rhythms under constant darkness. However, they display severely attenuated phase resetting in response to brief pulses of monochromatic light, highlighting the critical role of melanopsin in circadian photoentrainment in mammals.

Photoentrainment of circadian rhythms can occur in the absence of classical visual photoreceptors (rods and cones) (*1*, *2*) but not in animals without eyes (*3*, *4*). Therefore, nonvisual ocular photoreceptor(s) must mediate light entrainment. Recently, melanopsin has been suggested as a candidate circadian photopigment in mammals on the basis of several lines of evidence (*5*–*10*). First, it is expressed in retinal ganglion cells (RGCs), which directly project to the suprachiasmatic nucleus (SCN) and express the neuropeptide pituitary adenylyl cyclase activating peptide (PACAP) (*11*). PACAP has also been implicated in circadian photoreception (*12*). Furthermore, physically isolated melanopsin-containing RGCs depolarize in response to direct illumination with a spectral sensitivity that closely matches the behavioral action spectrum of circadian photoentrainment in rodents (*8*, *10*). To formally investigate the role of melanopsin in light resetting of the circadian clock in mammals, we have generated melanopsin-null mice (*Opn4*$^{-/-}$) by replacing exon 1 of the melanopsin gene with a neomycin-resistance gene by homologous recombination in embryonic stem cells (fig. S1). We verified interruption of the *Opn4* gene immunohistochemically with an antiserum to melanopsin (figs. S1 and S2). The targeted locus exhibits normal autosomal Mendelian inheritance, and the *Opn4*$^{-/-}$ mice are apparently healthy with anatomically normal eyes and no obvious developmental defects.

Photoreceptors can contribute to circadian oscillation in three ways: (i) as oscillator components (*13*, *14*), (ii) in acute light suppression of activity (masking) (*15*), and (iii) in light entrainment of the clock (*16*). To determine whether melanopsin is required for normal functioning of the circadian oscillator, we characterized locomotor activity rhythms in driven and free-running conditions in *Opn4*$^{-/-}$ mice and littermate controls (Fig. 1, A and B) (*17*). *Opn4*$^{-/-}$ mice entrained to a 12-hour white light (800 lux)/12-hour dark (LD) cycle (*18*) and exhibited no detectable defect in locomotor activity rhythms in constant darkness (DD). During entrainment, the phase angle of activity onset in relation to the LD cycle was similar in both wild-type and *Opn4*$^{-/-}$ mice. In DD, the free-running period length (τ) of the locomotor activity rhythm in the knockout mice was not significantly different from that of wild-type littermates (Fig. 2D). Total activity and the length of the activity phase during a

[1]Genomics Institute of the Novartis Research Foundation, 10675 John J. Hopkins Drive, San Diego, CA 92121, USA. [2]Department of Cell Biology, The Scripps Research Institute, 10550 North Torrey Pines Road, San Diego, CA 92037, USA. [3]Department of Anatomy, Physiology, & Genetics, Uniformed Services University, 4301 Jones Bridge Road, Bethesda, MD 20814, USA. [4]Department of Physiology, University of Saõ Paolo, Post Office Box 11176, Saõ Paolo 05422, Brazil. [5]Department of Biochemistry, University of Nijmegen, Post Office Box 9101, 6500 HB Nijmegen, The Netherlands.

*To whom correspondence should be addressed. E-mail: stevek@scripps.edu, provenci@bob.usuhs.mil

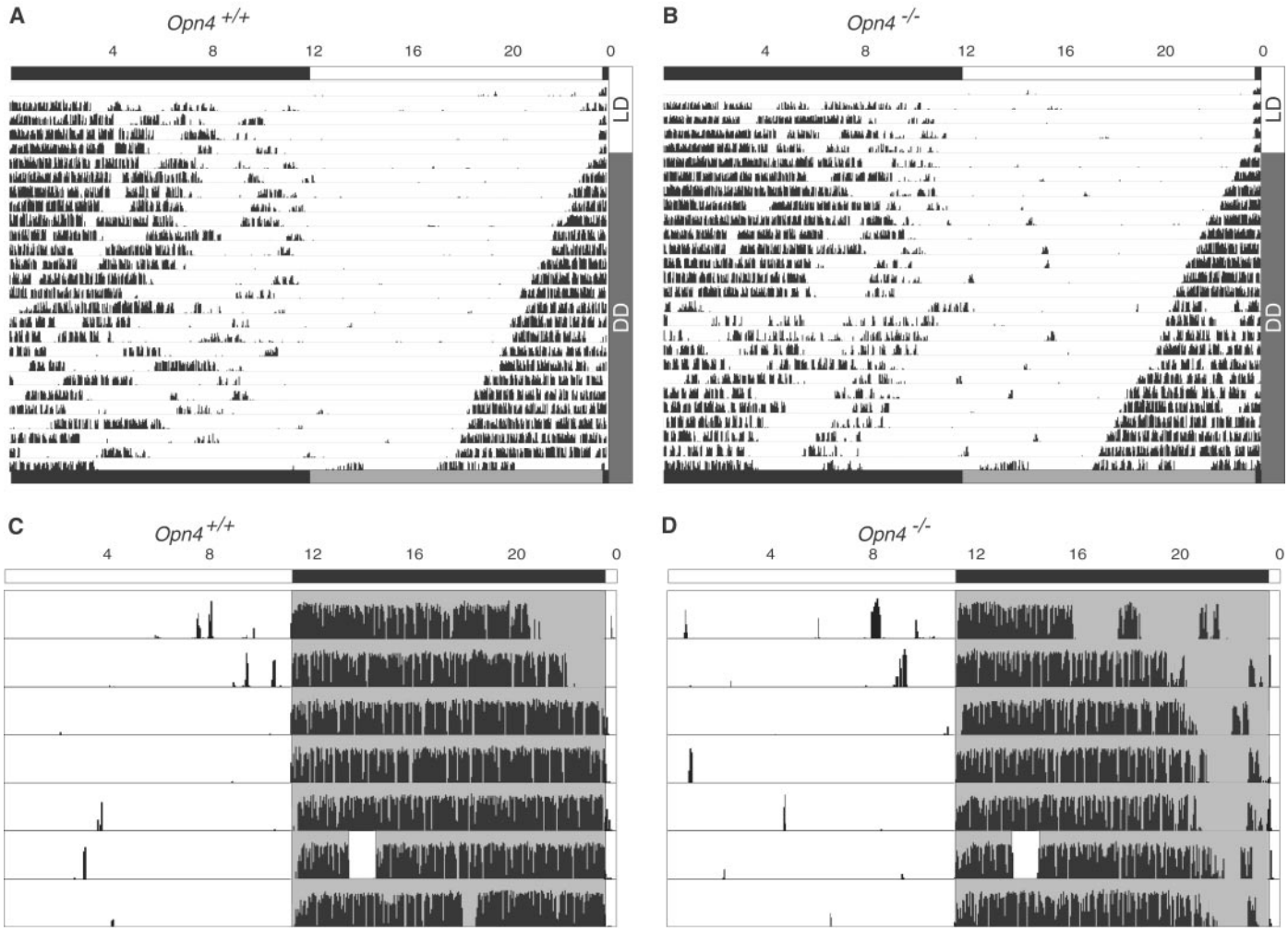

Fig. 1. *Opn4*$^{-/-}$ mice exhibit normal circadian behavioral rhythms and light suppression of activity. Littermate *Opn4*$^{+/+}$, *Opn4*$^{+/-}$, and *Opn4*$^{-/-}$ mice were entrained to a LD cycle and then allowed to free run under constant darkness. The respective period lengths (means ± SD) under constant darkness were 23.7 ± 0.14 (*Opn4*$^{+/+}$), 23.6 ± 0.14 (*Opn4*$^{+/-}$), and 23.7 ± 0.12 (*Opn4*$^{-/-}$) hours with no significant difference among genotypes (two-tailed, equal variance *t* test; $n = 6$ to 12 per genotype group). Representative activity traces of *Opn4*$^{+/+}$ (**A**) and *Opn4*$^{-/-}$ (**B**) mice are shown. Activity traces from the last 4 days of entrainment (LD) and 22 days of constant darkness (DD) are shown. Each horizontal line represents data from a single day. Consecutive days are plotted beneath each other, and 1-min bins of activity are represented as deflections from the horizontal line. Normal light suppression of activity in *Opn4*$^{+/+}$ (**C**) and *Opn4*$^{-/-}$ (**D**) mice is shown. A 300-lux white light pulse (*18*) was administered during the dark phase of entrainment. The light pulse acutely suppressed activity compared with activity at a similar phase in the preceding or following days (9 *Opn4*$^{+/+}$ and 11 *Opn4*$^{-/-}$ animals tested). Representative records from one *Opn4*$^{+/+}$ and one *Opn4*$^{-/-}$ animal are shown. White background indicates the light phase and gray background indicates the dark phase. The light pulse was administered on day 6 at 2 hours after lights off.

circadian cycle were also similar in all three genotypes (*19*), suggesting no significant defect in the functioning of the core oscillator or in the locomotor activity output of the clock. Taken together, these data suggest that melanopsin does not participate in the normal functioning of the core oscillator.

In addition, we tested whether masking contributes to the photoentrainment of *Opn4*$^{-/-}$ mice. Animals were entrained to a normal LD cycle and subjected to 1 hour of 300 lux of white light (*18*) during the dark phase (*17*). This analysis revealed light suppression of activity in both wild-type and *Opn4*$^{-/-}$ mice (Fig. 1, C and D), showing that masking is preserved in *Opn4*$^{-/-}$ mice.

To test the role of melanopsin in photoentrainment of the clock, we evaluated the phase-shifting effect of a brief pulse of monochromatic light on activity rhythms in DD. The magnitude of light-induced phase shifts of circadian locomotor activity rhythms depends on the spectral composition and irradiance of the stimulus. In addition, the magnitude and direction of the phase shift also depend on the circadian phase at which the stimulus is administered (*20*). Electrophysiological responses of melanopsin-positive RGCs have an action spectrum with a peak at 480 nm (*8*, *10*), coincident with the behavioral action spectrum for circadian photoentrainment. We hypothesized that any light input defect in the *Opn4*$^{-/-}$ mice would be most pronounced at this wavelength of light. Therefore, we administered a 15-min pulse of monochromatic light (480 nm with a 10-nm half-peak bandwidth) (*17*) of varying irradiance in early subjective night 3 hours after the onset of activity under constant darkness (circadian time 15). This is a time when normal mice exhibit robust phase delays in response to light. We then evaluated the phase-shifting effect of the light pulse by examining the phase of the onset of activity over subsequent days. *Opn4*$^{-/-}$ mice had significantly attenuated phase delays compared with wild-type animals (Fig. 2, A to C). Because the *Opn4*$^{-/-}$ mice displayed a τ in DD indistinguishable from the wild-type control (Fig. 2D), this difference in the phase-shifting response was unlikely to be due to a difference in the phase of the oscillator when the light pulse was administered. Instead, the attenuation in phase shifting likely resulted from reduced sensitivity of the photic input pathway in the *Opn4*$^{-/-}$ mice.

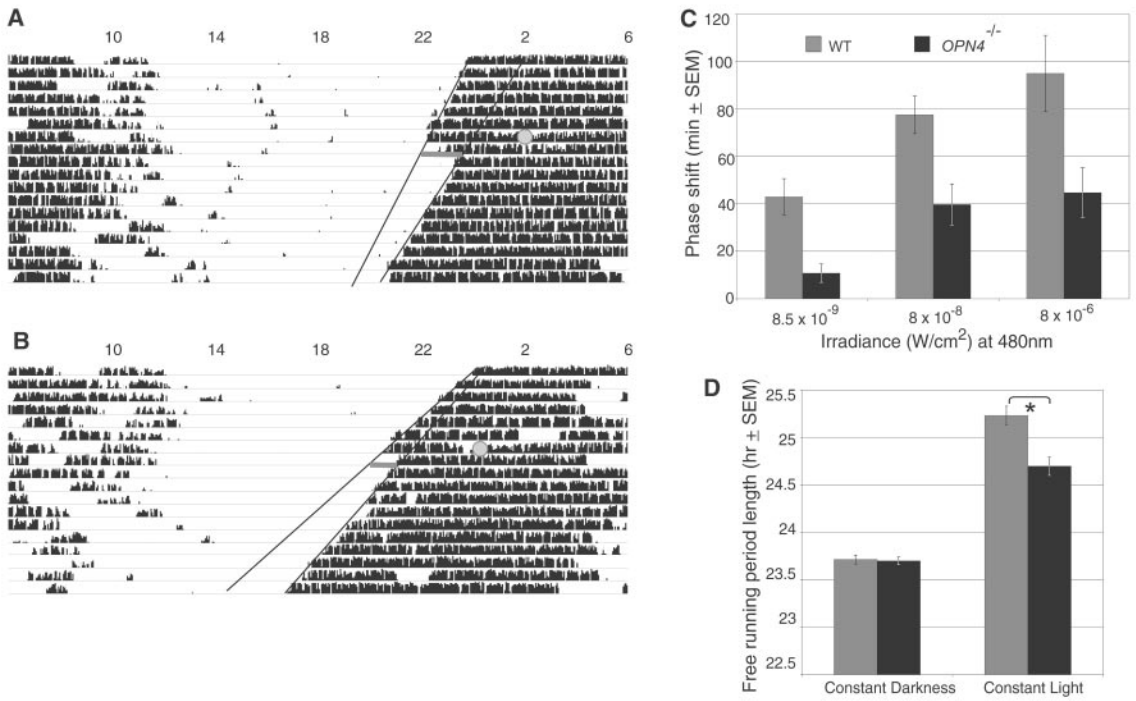

Fig. 2. Attenuated circadian light input in *Opn4*$^{-/-}$ mice. A single 15-min pulse of monochromatic light of 480 nm (circle) was administered 3 hours after the onset of activity, which produced a smaller phase shift in the activity rhythm of the *Opn4*$^{-/-}$ animal (**B**) than in the wild-type littermate (**A**). The phase shift (gray bar) on the day after the light pulse was determined with Clocklab software (Actimetrics, Evanston, IL). (**C**) The light-induced phase-shift defect in the null mice is evident at all irradiance levels tested. Means (±SEM) of phase-shift measurements for the *Opn4*$^{-/-}$ mice (black) and the littermate wild-type mice (gray) are shown (n = 5 to 17 mice per group). Data were analyzed by t test (two tailed, equal variance), and a statistically significant difference ($P < 0.05$) was observed between the genotypes at all irradiances tested. Sham animals handled in the same way but that did not receive a light pulse show no significant difference between genotypes. (**D**) An attenuated lengthening of period was observed in *Opn4*$^{-/-}$ mice (black) relative to wild-type mice housed in constant light but not in constant darkness. Asterisk indicates a statistically significant difference ($P < 0.05$).

To confirm this deficit in photic input to the clock in *Opn4*$^{-/-}$ mice, we assessed the role of melanopsin in light modulation of the circadian oscillator in constant light (LL). In most organisms, LL τ is a function of the intrinsic τ of the oscillator as well as light input to the clock. Genetic studies in plants and flies have shown that disrupting circadian light input pathways results in a circadian period length in LL that is closer to the DD period (*21*, *22*). In rodents under constant light conditions, photic input to the oscillator makes progressive phase adjustments, resulting in a longer LL τ relative to DD τ (*23*). Consistent with the role of melanopsin in light input to the oscillator, when we placed *Opn4*$^{-/-}$ mice in constant light (100 lux of white light) (*18*), their period length was shorter than that of the wild-type mice maintained in constant light (Fig. 2D). Therefore, the phase-shifting deficit in *Opn4*$^{-/-}$ mice accounts for a significant portion of the light input to the core oscillator under white light.

The residual phase resetting and LL period lengthening of *Opn4*$^{-/-}$ mice under LL (relative to DD τ) suggest that melanopsin-independent mechanisms participate in the photic entrainment of the oscillator. Several explanations exist for such a mechanism. First, developmental compensation may account for the residual phase-shifting effects found in *Opn4*$^{-/-}$ mice. Second, although the core oscillator role of cryptochromes confounds the genetic analysis of their role in photoentrainment, cryptochrome-deficient mice display a deficit in transcriptional responses to light in the SCN (*24*) and, therefore, may still mediate circadian light input. Third, genetic and anatomic studies in visual photoreceptor–deficient mice rule out their requirement for normal phase-shifting responses to light (*2*, *4*). Nevertheless, the visual photoreceptors may mediate light signaling to the oscillator, and melanopsin may be epistatic to them. Finally, uncharacterized photoreceptors may be responsible for the residual phase-shifting in *Opn4*$^{-/-}$ mice. In that regard, melanopsin may not function as a classical phototransducing photopigment, but it could function as a photoisomerase-like RGR opsin (*25*). Nevertheless, our results show that these mechanisms are not sufficient to fully compensate for the loss of melanopsin.

In summary, our observations show that melanopsin is required for normal circadian photoentrainment and, importantly, that other mechanisms for light input to the clock also play a role. Light entrainment in mammals then resembles the mechanisms in plants and flies (*22*, *26*, *27*), where independent photoreceptors with overlapping roles may function to adapt the organism to the natural changes in light quality and irradiance.

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Discounting and Reciprocity in an Iterated Prisoner's Dilemma

D. W. Stephens,* C. M. McLinn, J. R. Stevens

The Iterated Prisoner's Dilemma (IPD) is a central paradigm in the study of animal cooperation. According to the IPD framework, repeated play (repetition) and reciprocity combine to maintain a cooperative equilibrium. However, experimental studies with animals suggest that cooperative behavior in IPDs is unstable, and some have suggested that strong preferences for immediate benefits (that is, temporal discounting) might explain the fragility of cooperative equilibria. We studied the effects of discounting and strategic reciprocity on cooperation in captive blue jays. Our results demonstrate an interaction between discounting and reciprocity. Blue jays show high stable levels of cooperation in treatments with reduced discounting when their opponent reciprocates, but their levels of cooperation decline in all other treatment combinations. This suggests that stable cooperation requires both reduced discounting and reciprocity, and it offers an explanation of earlier failures to find cooperation in controlled payoff games.

The Prisoner's Dilemma illustrates the economic barriers to cooperative action. In this game, the defecting (noncooperative) option is always the best choice for a single play of the game, even though both players could do better if they cooperated. Axelrod and Hamilton (*1*) argued that cooperation could be a game theoretical equilibrium if (i) the game was played repeatedly and (ii) the players adopted a reciprocating strategy. In their argument, repetition and reciprocity combine to make mutual cooperation a viable strategy, because although a defector will receive an immediate reward, reciprocity means that it will suffer for this choice in the long run.

Although theoreticians have exploited this paradigm with great success, it has been markedly less successful empirically (*2–5*). Nonhuman animals show a strong tendency to defect in experimentally created Prisoner's Dilemmas (*6–9*). These studies raise important questions, because we cannot usually confirm that the payoffs in naturalistic studies conform to the Prisoner's Dilemma. This uncertainty has led to controversy in some cases (*10–12*), and in others, it has led to questions about whether simpler explanations of observed behavior might not be more appropriate (*5, 13, 14*). More than 20 years after Axelrod declared the Prisoner's Dilemma to be "the *E. coli* of social psychology" (*15*), there is still no single unambiguous case of stable nonhuman cooperation in a verifiable Prisoner's Dilemma.

One possible explanation for the fragility of cooperation in the Iterated Prisoner's Dilemma (IPD) is strong temporal discounting. In theory, animals should cooperate in an IPD because cooperation leads to higher payoffs in the long run, but animals may not value these long-term benefits because they strongly discount the future. Psychological studies support this idea. In these studies (*16–18*), experimentalists offer animals a choice between small immediate and large delayed food rewards. These experiments show very strong preferences for immediacy. Fitted discounting functions suggest that the first second of delay reduces the value of food reward by as much as 50% (*19*). These data, therefore, suggest that animal discounting may be much stronger than rates typically assumed by economists and other students of human behavior [e.g., 4% per year (*20*)].

An alternative explanation of the fragility of cooperative equilibria might hold that animals fail to cooperate, not because they discount strongly, but because they do not implement the appropriate strategy. In the IPD framework, the opponent's reciprocation means that cooperation now enhances long-

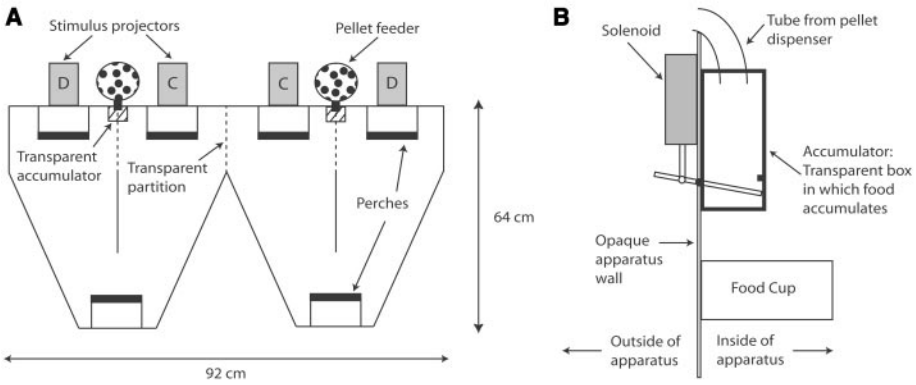

Fig. 1. (**A**) Top view of apparatus. The apparatus consists of side-by-side compartments, each in the shape of a V. Each compartment is equipped with three perches. Each perch has a microswitch that reports its status to a controlling computer. Each compartment houses a single bird, one of which is designated the subject and the other is designated the stooge. The subject chooses freely, but the stooge follows an experimentally imposed strategy. At the beginning of a play, the birds wait on their respective rear perches (at the apex of the V). At a programmed time, the controlling computer switches on stimulus lights on the front panel signaling that a trial has begun. The subject may hop forward to one of the two front perches to indicate its choice. A hop on the inside perch indicates a cooperate (or "C") choice, whereas a hop on the outside perch indicates a defect (or "D") choice. The stooge only sees one stimulus light and must hop on the associated perch. The apparatus is designed with transparent partitions across the front and opaque partitions elsewhere so that the birds can see each other after they have made a choice (hopped to the front), but not before. When both birds occupy one of the front perches, the pellet dispensers deliver food into the accumulators. (**B**) Accumulator. A transparent plastic box, front and center in each compartment, received food from the pellet dispenser. The bottom of the box was a flap that could be opened by the controlling computer. Thus, during accumulated treatments, subjects could see their food gains but not consume them until the flap was opened.

Department of Ecology, Evolution and Behavior, University of Minnesota, 1987 Upper Buford Circle, St. Paul, MN 55108, USA.

*To whom correspondence should be addressed. E-mail: dws@forager.cbs.umn.edu