

hydrocarbon biofuels from algae in 5 to 10 years (22).

Hydrocarbons derived from biomass are attractive because of their high energy density and compatibility with existing energy infrastructure. If recent technological innovations result in competitive production costs, hydrocarbons rather than ethanol will likely be the dominant biofuel.

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BEHAVIOR

Monkeys Like Mimics

Josep Call and Malinda Carpenter

Human adults routinely engage in unconscious (automatic) bodily mimicry of each other, and this has many positive social consequences: When others mimic us, we like them more, empathize with them more, and are more helpful and generous toward them (1). On page 880 in this issue, Paukner *et al.* (2) show that these social consequences of mimicry may have deeper evolutionary roots than previously thought.

Adding to previous findings that apes (3, 4) and macaque monkeys (5) recognize when they are being imitated, Paukner *et al.* demonstrate that imitation recognition has consequences for nonhuman primates in terms of their later affiliative behavior toward the imitator. They found that capuchin monkeys are more likely to sit closer to and exchange tokens with a human who imitated their actions than one who did not.

Most previous experimental work on imitation in nonhuman primates has focused on the instrumental function of imitation: learning new behaviors, typically to extract food from an unfamiliar apparatus. By contrast, Paukner *et al.* look at imitation in nonhuman primates from a more social and interpersonal perspective. In humans, imitation is not only a way of acquiring new behaviors, but also is a way of connecting with others and aligning



Imitation. Mimicking allows the acquisition of new behaviors and facilitates connections with others.

oneself with them—of communicating one's likeness and affinity to others (6). This social function of imitation is apparent from a very early age, when human children copy others closely (see the figure), even in problem-solving tasks when copying the particular actions the demonstrator used often is not necessary to achieve the instrumental goal (7). Both adults and children copy others more often when social goals are important (8, 9).

However, as Paukner *et al.* acknowledge, whereas the capacity to recognize imitation now appears to be widespread among primates, imitation itself is thought to be relatively rare in monkeys and apes, and certainly far less prevalent than in humans (10). It is thus unclear whether monkeys actually copy each other enough in their natural social

Human and nonhuman primates recognize when they are being imitated. Is there a social advantage to that?

groups for imitation recognition to serve the affiliative functions Paukner *et al.* postulate. Nonhuman primates do show contagious yawning (11) and some facial mimicry (12), but it is unknown how common and wide-ranging automatic bodily mimicry and imitation are for social functions in the natural social lives of nonhuman primates. Traditionally, researchers have used the distribution of grooming among individuals to assess affiliative networks in apes' and monkeys' social groups. Although it is conceivable that "mimicry networks" could accomplish a similar function, it is also possible that mimicry does not indicate affiliation per se; rather, it could indicate a different type of social information such as dominance. Individuals who copy others might be perceived as subordinate and therefore safer to approach. This could explain why monkeys in the Paukner *et al.* study approached the imitating partner (or avoided the nonimitating partner) to exchange tokens. Future studies will be needed to ascertain precisely what type of social information nonhuman animals extract from social mimicry.

If nonhuman primates do copy others' behaviors for social functions, this raises intriguing questions. Can monkeys mimic tactically, as humans do (8), to ingratiate themselves with others? This could be a powerful tool for developing, maintaining, and assessing social bonds with others. And does being mimicked have other, more actively prosocial consequences, as it does in humans (13)? For

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example, nonhuman primates who have been mimicked may help or share resources with others more often than those who have not been mimicked.

The finding of a link between imitation recognition and affiliative behavior in nonhuman primates highlights the need for more research into the social functions of imitation in nonhumans. We also need to explain why human imitation goes so far beyond automatic mimicry, in our “over-imitation” as children (14), our conformity to the majority’s way of doing things, our learning of actions conventionally and normatively (15), and our faithful transmis-

sion of such an extensive variety of cultural artifacts, rituals, and customs. An enhanced motivation to be like others may be what has boosted our imitation to such high levels.

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PHYSIOLOGY

How Much Sleep Do We Need?

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Sufficient sleep is necessary for optimal daytime performance and well-being, yet there is a large difference in how much sleep people need, ranging from less than 6 to more than 9 hours. People at all points along this range exhibit no noticeable differences in health and waking performance. Those of us who envy short sleepers would like to reduce sleep duration to the minimum necessary for normal functioning, but do we know what this minimum is? Short sleepers are found in families, as are long sleepers, which suggests a genetic basis for sleep duration. On page 866 of this issue, He *et al.* (1) add new evidence by showing that a mutation in a transcriptional factor, DEC2, is associated with short sleep in humans and mice.

Sleep and wakefulness are thought to be controlled by two independently regulated but interacting processes: a circadian program that sets the timing of sleep onset and offset, and a homeostatic process that tracks sleep need (2, 3). The homeostatic process is examined by measuring slow-wave electrical activity in the brain—which is derived from recordings of spontaneous brain activity as an electroencephalogram—to quantify oscillations in the 0.5 to 4.5 Hz frequency range (delta oscillations). These oscillations characterize a phase of sleep called non-rapid eye movement sleep. Slow-wave activity is thought to reflect the intensity of sleep. Although this intensity

measure is directly and predictably correlated with the duration of preceding waking, it only marginally predicts sleep duration, which indicates that sleep loss is primarily recovered by increasing sleep intensity and not necessarily by sleep duration.

By sequencing candidate genes in a family in which two individuals (a mother and her daughter) have short sleep durations (average 6.25 hours versus 8.06 hours in nonaffected individuals), He *et al.* identified a point mutation in hDEC2. DEC2 is a transcription factor involved in cell proliferation and differentiation, response to hypoxia, and circadian rhythms. The mammalian molecular circadian clock involves a heterodimer of transcriptional regulatory proteins containing BMAL1 and either CLOCK or NPAS2. This complex

Mutations that affect sleep duration are a starting point for understanding sleep regulation and function.

activates a negative feedback loop involving expression of the *Cryptochrome* (*Cry1* and *-2*) and *Period* (*Per1* and *-2*) genes (4). Although DEC1 and *-2* were also proposed to act as negative regulators of the circadian clock (5), their precise role is still unclear. For example, mice deficient for both *Dec1* and *-2* show intact circadian rhythmicity with only a slightly longer period of their rest-activity cycle and an attenuated entrainment response to light (6). By contrast, the gene *Clockwork Orange* (*cwo*) in the fly *Drosophila melanogaster*, which is the closest hDEC homolog, cooperates with the clock protein PER to repress *Clock*-mediated transcription of target genes (7). Flies lacking *cwo* have a longer circadian period and become arrhythmic in constant darkness, indicating that the fly homolog of hDEC is a core clock gene (7).

The mutation identified by He *et al.* causes a proline-to-arginine amino acid change at position 385 of hDEC2 (P385R) and was not found in 250 control subjects. The authors genetically engineered mice to express the P385R mutant form of hDEC2 and found that their activity period was ~1.2 hours longer than in normal mice. This activity period was even longer (~2.5 hours) when the P385R variant was expressed in mice lacking endogenous DEC2 expression, confirming a dominant



Sleep genes. Sleep amount, like weight and height, is a quantitative phenotype normally distributed in the population. Total daily sleep duration has an estimated heritability of ~50% in humans and mice, suggesting complex underlying genetics with contributions from numerous genes. But mutations in single genes that yield dramatic effects cannot be excluded.

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