The Drunken Monkey Hypothesis

The study of fruit-eating animals could lead to an evolutionary understanding of human alcohol abuse.

By Dustin Stephens and Robert Dudley

What can a tipsy howler monkey tell science about humanity's fondness for—and problems with—alcohol? Possibly quite a lot. And that would be a good thing, considering how widespread our problems with alcohol are. In the United States alone, 14 million people are alcoholics, and several millions more are at risk. Although patterns vary from culture to culture, alcoholism is common across the globe, particularly among indigenous groups undergoing modernization, and it comes with tragic consequences: Even in the United States, abuse of alcohol is the third leading cause of preventable death.

Studying the evolutionary background of human behaviors that lead to widespread disorders has helped shed light on how those disorders emerged and became established. Similarly, placing alcoholism in an evolutionary framework might reveal how our forebears became attracted—and addicted—to alcohol. That's where the tipsy howler monkey comes into the picture. In 2004, one of us (Stephens) observed him feasting on the bright orange fruits of the *Astrocaryum* palm, in the tropical forest of Panama's Barro Colorado Island. Climbing onto the branches of a neighboring tree to reach the untouched clusters, the forager first sniffed the fruit, then frantically began to eat it, sometimes dropping partly eaten fruits onto the forest floor. Risking a thirty-foot fall and serious injury from the enormous spines of the palm tree, the monkey seemed as fearless as a drunken teenager.

The forest covering much of Barro Colorado Island, in Gatun Lake, part of the Panama Canal, is home to four species of primates, among them howler and spider monkeys. The study of these primates and their behavior may shed light on the complex relation between people and alcohol.

Our assays of the fruit he dropped suggested why: He may, in fact, have been drunk. Our calculations showed that the reckless forager had consumed the monkey equivalent of ten "standard drinks" during his twenty-minute gorging session. This measurement was the first quantitative estimate of the amount of alcohol ingested by a wild primate ever made. It also fitted nicely with the "drunken monkey" hypothesis, developed earlier by one of us (Dudley).

The hypothesis proposes that a strong attraction to the smell and taste of alcohol conferred a selective advantage on our primate ancestors by helping them locate nutritious fruit at the peak of ripeness. Millions of years later, in the Middle Ages, people learned to distill spirits, which potently concentrated the natural alcoholic content of fermented fruits and grains. The once advantageous appetite for alcohol became a danger to human health and well-being. Drawing on yeast biology, fruit ripening, biological anthropology, human genetics, and the emerging field of Darwinian medicine, the drunken monkey hypothesis could ultimately contribute to understanding—and perhaps even mitigating—the enormous damage done by alcohol.

The drunken monkey hypothesis goes like this: For 40 million years, primate diets have included substantial quantities of fruit. In the warm, humid tropics, where humans evolved, yeasts on the fruit skin and within the fruit convert sugars into various forms of alcohol, the most common being ethanol. Ethanol is a light molecule that disperses readily, and the downwind odor of ethanol is a reliable sign of ripe fruit. In the tropical forests where most primates live, the competition for ripe fruit is intense. For a hungry monkey, then, a good foraging strategy would be to follow the smell of alcohol to the fruit and eat it in a hurry. Natural selection probably favored primates with a keen appreciation for the smell and taste of alcohol. After all, they would have been quicker than their competitors to grab, if you will, the "low-hanging fruit."

We want to stress from the outset that the drunken monkey hypothesis is just that—a hypothesis. It remains far from proven, and there are experts who disagree with our assumptions. But we think the hypothesis has great potential for explaining humanity's deep and conflicting relations with alcohol. The logic of the argument, the supporting evidence, and a discussion of the areas where further work is needed all give new evolutionary and biological perspectives on what, until now, has been seen as an issue that is entirely medical and sociological in nature.

An impressive body of evidence indicates that contemporary primate diets are dominated by plant materials. In many primate groups those materials take the form of ripe (and probably alcohol-containing) fruits. Fossilized teeth show that fruit has been a major component of the primate diet since the mid- to late Eocene Epoch, between 45 million and 34 million years ago. Some of our closest relatives—chimpanzees, orangutans, and certain populations of gorillas—eat diets based primarily on fruit.

Spider monkey (Ateles geoffroyi) feeds on a brightly colored fruit from the Astrocaryum *palm tree, growing in the forest of Barro Colorado.*

To be sure, our own ancestors long ago left fruit behind as the main source of their nutrition. By the time the genus *Homo* appears in the fossil record, between 1 and 2 million years ago, fruit had been marginalized, and largely replaced by meat and by foods such as roots and tubers. But even though our early hominid ancestors stopped relying heavily on fruit, humanity shares a deep evolutionary background with other primates. It seems likely that the taste for alcohol arose during that long shared prologue.

Consider the evidence.

The place to begin is the relation between ripe fruit and alcohol. Yeasts that occur on fruit consume sugar molecules in the fruit as a source of energy, in a process known as anaerobic fermentation ("anaer-

*Chimpanzee (*Pan troglodytes*) feeds on ripe mangoes, already fallen to the ground. The diets of the closest primate relatives of human beings—namely, orangutans, chimps, and some populations of gorillas—are based on plant materials, mostly fruits.*

obic," because it takes place in the absence of oxygen) [*see diagram on opposite page*]. As the fruit ripens, and the yeast enzymes get going, the ethanol content of the fruit rises rapidly. For example, the unripe fruit of the *Astrocaryum* palm contains no ethanol; ripe hanging fruit is about 0.6 percent ethanol by weight; overripe fruit, often fallen to the ground, can have an ethanol content of more than 4 percent. The howler monkey that Stephens observed on Barro Colorado Island was feasting on fruit near its peak ripeness—when its ethanol content is about 1 percent.

What is the evidence that our primate relatives (or other organisms, for that matter) hone in on alcohol as a nutritional signpost? It is known that fruit flies of the genus *Drosophila*, a laboratory workhorse in genetics, follow increasing concentrations of ethanol vapor as a way to find the ripe fruit within which they lay their eggs. The fruit is an excellent food source for the fly larvae when they hatch.

A similar sensory mechanism is likely at play in other species, including primates. Alcohol-driven fruit "binges" similar to the one seen on Barro Colorado have been observed several times in howler and spider monkeys. In each instance, the monkey risked life and limb while eating quickly from bunch after bunch of *Astrocaryum* fruits, sometimes committing its full weight to a fruit cluster without so much as a prehensile tail secured as a backup. Other observations from the rainforest describe what seems to be fruit-induced intoxication in butterflies, fruit flies, a variety of birds, fruit bats, elephants, and several other primate species.

It is possible, of course, that drunken behavior is simply an accident without a deep evolutionary context. Maybe rainforest fauna just like to have fun. But some evidence implies that the connection between alcohol and nutrition is deeper than that, at least for primates. Initial observations of monkeys on Barro Colorado show that they prefer ripe palm fruits with moderate levels of alcohol. They avoid unripe fruits—with no alcohol—as well as overripe fruits—with more alcohol but less sugar (by then, most of the sugar has been converted to alcohol). Anecdotally, we note that people, too, often drink alcohol while eating, suggesting that drink with food is a natural combination. And various experiments have shown that drinking an aperitif increases both the time spent eating and the number of calories consumed at a sitting.

If there really is an evolutionary connection between alcohol and primate nutrition, an important conclusion follows: Alcohol—at least in moderation—cannot be entirely inimical to health. If it were, a good nose for alcohol would not have conferred selective advantage on our primate forebears; in fact, it would have damaged them.

In any event, a wide range of evidence suggests that moderate consumption of alcohol is healthful for widely divergent organisms. Fruit flies, for instance, live longer and have more offspring when they are experimentally exposed to vapors containing intermediate levels of ethanol than they do when exposed to a lot of it or to none at all. The biological mechanisms underlying those effects remain unclear, but as we noted earlier, the ability to sense and respond to the scent of ethanol plays an important role in the life cycle of the fruit fly. The health benefits of consumption may be connected in some way to the role that ethanol has in the fly's life cycle.

In people, too, moderate alcohol consumption seems to be more salubrious than too much or too little. Much of the evidence, however, for the health benefits of moderate drinking arises out of the risk factors for heart disease, which may not be relevant to the evolutionary argument. (The protection alcohol confers against heart disease may come from counteracting the effects of the high-fat diet we adopted long after our ancestors' fruit-eating days were past.) Still, other evidence suggests, circumstantially at least, that intermediate levels of alcohol consumption have benefits beyond their effects on the heart. A recent epidemiological study of Finnish civil servants showed that the workers who took the fewest official sick days were moderate consumers of alcohol.

To prove the drunken monkey hypothesis, it is not enough to show that alcohol is beneficial—or at least not damaging—to health. One also has to demonstrate that a varied group of genes is related to alcohol consumption. Only by operating on a variety of genes could evolution have selected the fittest of our primate forebears. Here we are on firmer ground. There is unquestionably a wide variation among human beings in the genes that underlie alcohol metabolism and, consequently, in individual appetites for alcohol.

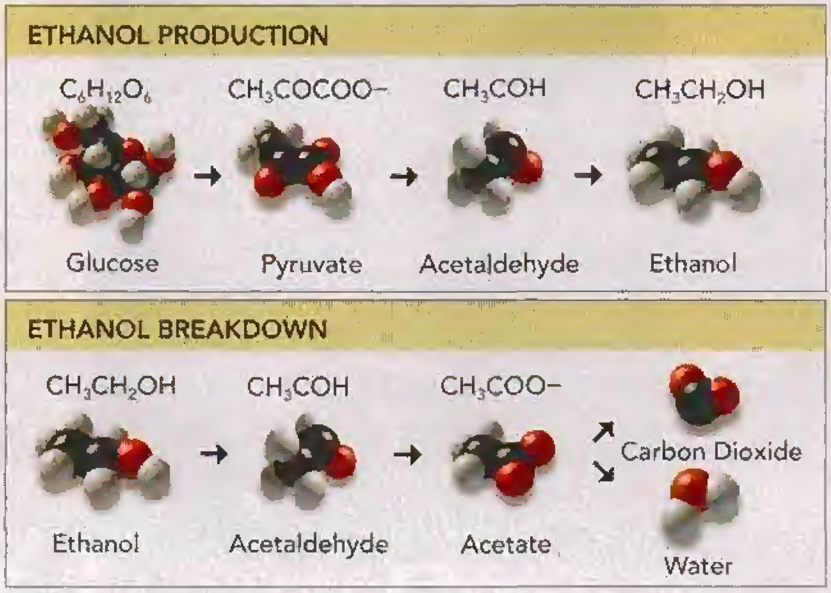
The genes in question encode two enzymes that metabolize alcohol and its breakdown products; the enzymes are known as alcohol dehydrogenase and aldehyde dehydrogenase. But the genes vary from person to person, and that genetic variation becomes manifest as a wide variation among the gene-encoded enzymes in their efficiency at clearing alcohol or its toxic breakdown product, acetaldehyde, from the blood. Elevated levels of acetaldehyde cause headache, nausea, palpitations, and flushing. Given such a suite of unpleasant effects, it would be surprising if people who have inefficient acetaldehyde-clearing enzymes were eager to get tipsy. And sure enough, studies of East Asian populations, where the less-efficient enzymes are common, confirm that guess. In Japan, alcoholics are more likely to have rapid and efficient versions of the enzymes than nonalcoholics.

To sum up: A variety of direct and circumstantial evidence suggests that in our deep evolutionary background, alcohol and nutrition (and consequently, alcohol and survival) were intertwined. For some of our close genetic relatives, rainforest observations show that they remain intertwined to this day. Furthermore, some evidence shows that intermediate levels of alcohol consumption are beneficial to human health. But if evolution has rendered alcohol so good for us, why is it now such a plague?

The answer, we think, lies in a mismatch between our species' long evolutionary prelude and the techno-cultural environment we have created in the past few centuries. Until well into the first millennium A.D.—following millions of years of primate evolution—the amount of alcohol our ancestors could consume was strictly limited. As we have noted, even overripe fruits have an ethanol content of only about 4 percent, and they are not the ones favored by monkeys.

That picture did not change substantially even when modern humans, some 10,000 years ago, learned to control fermentation. As agriculture took root, barley and wheat became plentiful, which in turn provided good substrates for beer. Archaeological evidence from the same period indicates that wine was also being made. In fact, until industrialization made water filtration practical, alcoholic drinks are thought to have been more widely consumed in many cultures than water was.

But the alcoholic drinks of today—and the alcoholism that accompanies them—are, in evolutionary terms, recent innovations. Yeasts stop making ethanol when its concentration reaches between 10 and 15 percent by weight. Hence drinks made using natur-



Schematic chemical reaction pathways show major steps in the production and metabolic breakdown of ethanol (no attempt has been made to "balance" the chemical reactants and products). Yeasts growing on and within tropical fruits transform glucose and other sugars from the fruits into ethanol through fermentation (upper diagram). When animals consume ethanol, they break it down into carbon dioxide and water (lower diagram). Acetaldehyde, an intermediate compound in the metabolism of ethanol, is toxic.

Fruitbats (upper image), fruitflies, and waxwings (lower image), all fruit-eating animals, have been anecdotally described in various states of apparent inebriation. The anecdotes suggest that alcohol consumption may be widespread among a broad range of species.

al yeasts are limited in alcohol content. Beer and wine made before the invention of chemical distillation (in central Asia around A.D. 700) probably were no more than 5 percent ethanol. No harder stuff was available.

The invention of distillation, which had reached Europe in the Middle Ages, radically changed humanity's relationship with alcohol. Drinks whose ethanol content was much higher than 5 or even 12 percent suddenly became widely available. From the vantage of the drunken monkey hypothesis, the results were predictable: wide availability of potent drink led straight to extreme forms of alcohol abuse.

From the evolutionary perspective taken by Darwinian medicine, alcoholism is one of the "diseases of nutritional excess" that arises from a mismatch between prehistoric and contemporary environments. Perhaps the most striking example of such a disease is the ongoing epidemic of obesity. In 1962, the late geneticist James Neel predicted that as high-fat, high-calorie Western foods became available to tribal peoples, their incidence of obesity, heart disease, and adult-onset diabetes would sharply increase. The rationale for Neel's hypothesis was that "thrifty" genes, which had been advantageous in sequestering scarce calories, had turned deleterious when fats and sugars become readily available. The high rates of diabetes among Pima Indians, Micronesian Nauruans and Australian Aborigines have confirmed his predictions.

Neel's prescient hypothesis, now clearly relevant to human populations in the developed world as well, fits nicely with the drunken monkey hypothesis. The increased alcohol concentration of booze made possible by industrial distillation played right into a genetically rooted appetite for alcohol that had been present for millions of years—and had served a valuable survival function for our forerunners as they climbed through the rainforest canopy. And just as with obesity, heart disease, and diabetes, alcoholism has become a risk for anyone with access to the fruits of contemporary culture.

The drunken monkey hypothesis, like many another productive scientific idea, raises more questions than the evidence so far in its favor can answer. How, precisely, do primates locate ripe fruit? What are the typical alcohol concentrations in the fruits they eat? Does alcohol act as a stimulant for primate feeding? How often do primates become intoxicated as a result of eating fruit? And what are the beneficial effects of alcohol on human beings and related species?

We are working to answer some of these questions, and we encourage our colleagues to answer others. It is still just the beginning of what we believe will be (forgive the pun) a fruitful avenue of research into human prehistory. And perhaps the knowledge gained will ultimately suggest strategies for stemming the tragic damage to our species wrought all too commonly by alcoholism. ☐