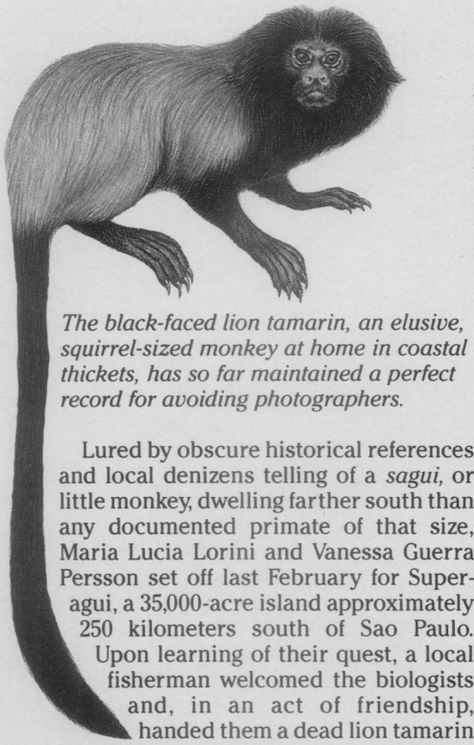
Tamarin tale: Tracking down a new species

Peering into the tangled thickets of a Brazilian coastal island, two biologists have spied colorful little monkeys where little monkeys shouldn't be. They say the creatures represent a previously undescribed species of lion tamarin, a genus that includes some of the rarest and most endangered primates in the world.



Stephen Nash/Conservation Intl.

The black-faced lion tamarin, an elusive, squirrel-sized monkey at home in coastal thickets, has so far maintained a perfect record for avoiding photographers.

Lured by obscure historical references and local denizens telling of a *sagui,* or little monkey, dwelling farther south than any documented primate of that size, Maria Lucia Lorini and Vanessa Guerra Persson set off last February for Superagui, a 35,000-acre island approximately 250 kilometers south of Sao Paulo. Upon learning of their quest, a local fisherman welcomed the biologists and, in an act of friendship, handed them a dead lion tamarin unlike any they had seen before. Within a month, the team had identified two family groups of the enigmatic monkeys of Superagui, for a total of about two dozen individuals.

The find is "one of the most amazing primatological discoveries in this century," says Russell A. Mittermeier of Conservation International in Washington, D.C., who has studied primates in coastal Brazil for the past 12 years. Identifying a new type of monkey on an inhabited island flanked north and south by mainland resort developments "is almost like finding a major new species in the suburbs of Los Angeles," he says.

Lorini and Persson, of the Natural History Museum in Curitiba, Brazil, formally announced their discovery on June 11, describing it as a new species in the bulletin of the National Museum in Rio de Janeiro. Like the three previously known species of lion tamarin, the black-faced lion tamarin, or *Leontopithecus caissara,* is a squirrel-like monkey with a small face framed by a luxuriant mane. However, it is distinct from the others in combining a black face with a golden body on a slightly larger frame, says Dante Martins Teixeira, head of vertebrate zoology at the Rio museum.

Teixeira notes that some primate taxonomists view all types of lion tamarins as one species and thus may not accept the black-faced version as a species unto itself. Mittermeier, who disputes such "lumping" of lion tamarins, says: "The four populations are each separated by at least several hundred kilometers. When populations don't overlap and are clearly morphologically distinct, then my feeling is you call them species."

Scientists are unanimous, however, in calling lion tamarins endangered. Today, these animals live wild only in remnant forest patches of rapidly developing eastern Brazil, where they collectively number in the low thousands. One, the black lion tamarin, may have dwindled to fewer than 200 individuals in the wild, Mittermeier says. The black-faced lion tamarin appears even more scarce.

Teixeira is organizing a July expedition to Superagui in hopes of learning more about the genus' newest member. He and other biologists are also pressing the Brazilian government to extend the boundaries of a national park on Superagui to include the forest where the black-faced lion tamarins live. Although the island still lacks electricity or bridges to the mainland, it does possess one dangerously attractive commodity: a beautiful beach. "There is a plan to construct a little Miami on this island," Teixeira warns. — *W. Stolzenburg*

Probing cocaine in the heart and the brain

Two recent drug studies add new details to the picture of how cocaine damages the body and point to a way of easing withdrawal pangs in addicts.

At Brookhaven National Laboratory in Upton, N.Y., researchers injected healthy volunteers with radioactively labeled cocaine at doses far too small to induce addiction or a "high." They discovered that the drug binds strongly to human heart cells, particularly in the left ventricle. Study leader Nora D. Volkow says the research, undertaken with permission from the Food and Drug Administration, suggests that cocaine overdose may pose a triple threat to the heart.

Scientists already knew that cocaine abuse can cause heart failure through its indirect effects — constricting blood vessels and manipulating the brain to disrupt normal heart rhythm. But in binding directly to cardiac tissue, cocaine may add a third lethal punch by slowing the passage of sodium ions into heart cells and/or stimulating the release of the neurotransmitter norepinephrine, which can lead to irregular heartbeat, or arrhythmia, Volkow asserts.

She and her colleagues also found that large concentrations of cocaine bind to the aorta, the major artery carrying blood from the heart. This, they say, may account for some of the blood vessel damage associated with cocaine overdose. Volkow reported the findings last week at the annual meeting of the Society of Nuclear Medicine in Washington, D.C.

In a separate study described at the meeting, Volkow's team and collaborators at the State University of New York at Stony Brook examined human brain scans highlighting nerve-cell receptors for the neurotransmitter dopamine.

Normally, dopamine spills into the gap, or synapse, between the "presynaptic" neuron that released it and a neighboring, "postsynaptic" neuron, staying there just long enough to stimulate receptors on the second neuron. The dopamine then returns to the storage compartments of the presynaptic cell, a process known as reuptake. But cocaine blocks reuptake, leaving dopamine in the synapse to repeatedly bombard the postsynaptic receptors, contributing to a temporary feeling of euphoria.

The number of postsynaptic receptors can fluctuate with changes in stimulation levels. To assess cocaine's effect on receptor abundance, the researchers gave trace amounts of a radioactively tagged compound that binds selectively to postsynaptic dopamine receptors to 10 healthy volunteers and 10 cocaine addicts who had not taken the drug for one month or less.

Addicts who had been off the drug for one week or less showed about 30 percent fewer dopamine receptors than the healthy volunteers, Volkow and her colleagues found. Those who had abstained for a full month had about the same number of receptors as the healthy controls, the researchers note in the June AMERICAN JOURNAL OF PSYCHIATRY.

Volkow conjectures that the decreased number of dopamine receptors reflects the body's attempt to balance a system gone out of control: As cocaine floods synapses with dopamine, postsynaptic receptors dwindle in number to avoid excess stimulation. But when addicts stop taking cocaine, the receptor loss can leave them temporarily starved for dopamine and craving the drug, Volkow suggests, noting that most relapses occur during the first weeks of treatment.

Other researchers have proposed similar scenarios for cocaine's influence on dopamine receptors. But until now, Volkow says, tests of those theories in humans have relied only on indirect measurements, such as correlating cocaine use with blood levels of prolactin, a dopamine-regulated hormone. The new study, which directly measured receptor abundance, suggests recovering addicts may have fewer relapses and less craving