# Seeing color traced back to genetic mutations

May 21 2012, By Faye Flam

From the inside of our heads, it feels as if colors are intrinsic aspects of the outside world and our eyes are beautifully designed to see them. But we humans are merely sampling the possible ways of sensing the spectrum of light. Most of us see more colors than dogs, but miss millions of colors that make up the world for birds, reptiles, and insects.

Why do we see the colors we do?

Some of it goes back to the types of color-absorbing pigments that we inherited from bacteria more than a billion years ago. The specific colors we see are, in part, an artifact of bacterial needs.

These ancient color-sensing pigments are tuned to two different wavelengths - shorter ones that correspond to blue and longer ones that go with yellows or reds.

All other color vision is an outgrowth of that system, said neurobiologist Jay Neitz, who works in the ophthalmology department at the University of Washington.

Over time, the genes that hold the code for our color-sensitive pigment sometimes get duplicated. The extra copies can pick up mutations that shift the wavelengths they're capable of absorbing. Such a lucky series of accidents appears to have happened very early in the evolution of animals, said Neitz, since some fish, birds, reptiles, amphibians, and insects are endowed with one or more additional color-sensitive pigments. These make up different types of light-sensitive cells called

cones. We humans have three kinds of cones, but many other animals have four. A few animals have more: One species of shrimp appears to have twelve.

Somewhere along the line, however, mammals lost all but the more ancient color-vision genes. We and a few other primates reinvented it much later, but dogs, while not color-blind, see a more limited range.

Dogs can be tested for color blindness just like people, said Neitz. They can't tell us what they're seeing, but they can show us.

He used a device in which a touchÂscreen showed three shapes, two of the same color and one of a different one. The dogs were trained to pick the different color by touching the screens with their noses. If they got it right, they were rewarded with a treat.

When they could see the colors, they almost always got it right. Knowing a treat was possible, the dogs still hazarded a guess when they couldn't see any difference, but they would get it right just a third of the time.

In that way, the dogs demonstrated they could tell blue from yellow and both of those colors from white or gray, but they could not distinguish yellow from red or green. (Cats, too, can be trained to do this, but only if researchers use treats that a cat finds irresistible).

We humans, along with apes and some monkeys, picked up a fortuitous mutation in our long-wavelength cone, adding a middle-wavelength cone that can sense green. It works in concert with the other two cones to give a whole array of different colors - green, purple, red, and many intermediate shades.

In humans, mutations on the X chromosome can interfere with this more recently acquired ability. Most people who are considered color-blind

have the same color capacity as dogs and cats, said Neitz. Men are much more likely to inherit this type of color blindness because they only have one copy of the X, while women have a second X as a backÂup.

About 8 percent of men lack the third type of cone, among them the English chemist John Dalton, who wrote in the 1700s that he saw slightly different shades of yellow when others saw red or green. Neitz points out that if Dalton couldn't distinguish these colors, it's hard to know whether his yellow looked like our yellow or our red or something else. At some point it becomes a thorny philosophical problem about the difference between reality and what individuals perceive as reality.

A few lucky mutant humans may have four different kinds of cones. They would most likely be women, who could acquire different types of cone-coding genes on each of their two X chromosomes. A few studies are under way to identify and understand the vision of such women.

Another evolutionary puzzle is why just a few primates got the green cone while most other mammals live in a less colorful world. Evolutionary neurobiologist Mark Changizi has proposed that this mutation spread because it gave our ancestors the ability to assess the health, emotional states, and fertility of fellow primates.

The mutation goes back 50 million years, so it must have spread among furry primates, but Changizi, who works for 2AI Labs in Boise, Idaho, points out that all the color-vision-endowed primates today show at least some bare skin somewhere.

Sometimes it's on the face and sometimes it's on the rear, where some primate females develop pink swellings to advertise that they have reached the fertile part of their cycle.

Others propose that color vision helped primates distinguish poisonous

snakes, or recognize edible fruits. "Maybe it's all of the above," said Neitz, which would explain why the mutation would have spread.

Neitz points out that just because an animal can benefit from a trait doesn't mean evolution will ever bestow it. A mutation has to crop up first, and perhaps the monkeys that lack a green receptor are just waiting for their lucky mutation to crop up.

A few years ago, Neitz wondered if it would be possible to give an animal that lucky mutation through gene therapy. He chose squirrel monkeys - part of the primate group that missed out on the green cone.

No one knew if the monkeys' brains would have the wiring needed to see more colors. But by giving the genetically enhanced monkeys the same kinds of color-vision tests he'd used on dogs, Neitz showed they could distinguish yellow, green, and red, just as we can.

Did that change their behavior?

One of them showed a definite preference for green M&Ms after the procedure, said Neitz. And it enjoyed green beans.