In the spring of 1986, a neurologist received an extraordinary letter. The writer, Mr. I., had been recently concussed in a car accident. Within days of the crash, his vision had become "that of an eagle" — he could see the smallest details from a city block away. "BUT," he wrote, "I AM ABSOLUTELY COLORBLIND."

Mr. I. did not notice this change until after leaving the hospital. Eager to return to work as an artist, he set out for his painting studio only to find the road shrouded in mist, despite the sunny weather. Alarmed, he rushed toward his studio and was ticketed for running two red lights that he could not see. At last he entered his workspace, seeking comfort in a room he expected to be filled with vibrant paintings. Instead, his artwork was gone. The painter's gaze darted over the slate-toned walls, trying to understand. Though the canvasses hung where he had left them, all their colors had drained away, leaving only black, white, and gray. He closed his eyes and tried to picture the studio as it should have been, but even his memory was void of color. As far as his brain was concerned, the world had always been shades of slate and lead.

Total colorblindness due to brain damage, a phenomenon known as acquired cerebral achromatopsia, is not only incredibly rare — it's irreversible. For people accustomed to the world of color, its effects can be devastating. Mr. I. was disgusted by his new reality. Without color, art and life felt meaningless. Profoundly disturbed by the altered appearance of food, he struggled to eat. Choosing clothes was impossible. Human skin, now rat-colored, was repulsive. He visited specialist after specialist to no avail and gradually sank into despair. That spring, while the world around him delighted at the sight of bluebells and roses, Mr. I. sat at his gray desk and wrote the letter that would lead him to neurologist Oliver Sacks.

As far as his brain knew, the world had always been shades of slate and lead

Dr. Sacks and his team instantly recognized the importance of Mr. I. Uncovering the cause of his achromatopsia would yield valuable information on how the brain processes color. The task, then, was to determine which part of the painter's visual system had been altered in the crash. In a normal human visual system, light enters the eye and hits light-sensitive cells in the retina called photoreceptors. Activated photoreceptors send a signal back through the retina, down through the optic nerve pathway, and into the visual cortex of the brain. The brain then assembles the information to decide what it is seeing.

All photoreceptors are either rods or cones, both essential for normal vision. Rods are responsible for seeing in low light, providing night vision. Cones perceive color and function best in bright light, which is why it becomes hard to see color in the dark. Humans possess three types of cones, each sensitive to different wavelengths of light. Short cones show the most sensitivity to blue light, medium to green, and long to red. (Cone names do not reflect the sizes of the cones themselves, but rather the size of the wavelengths they receive.) If one type of cone is mutated but still present, a person may be able to perceive that color, albeit weakly. If a cone type is missing entirely, the corresponding color cannot be seen.

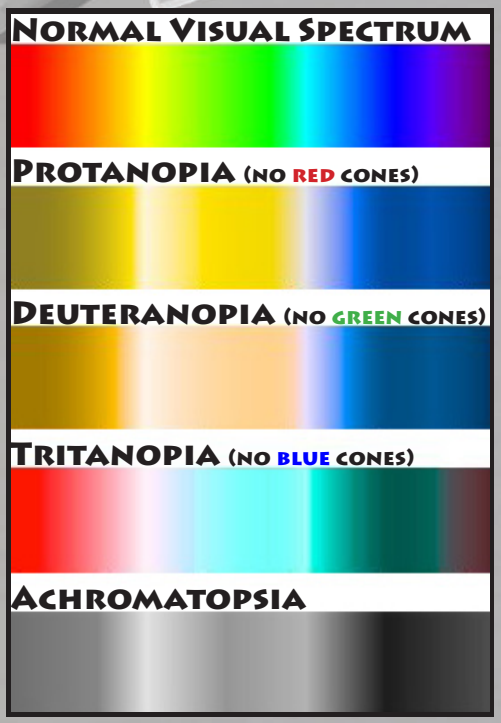
Most cases of colorblindness involve only type of one cone, usually red or green. Lack of either red or green cones restricts a person's vision to a blue-yellow spectrum. Due to their similar effects, both conditions are grouped together under the name red-green colorblind. Tritanopia, the loss of blue cones, is far rarer. Tritanopes confuse blue with green and yellow with violet, seeing the world as largely pink and turquoise.

Colorblindness occurs in one in twelve men and one in two hundred women. It is usually genetic, though not all types are

inherited the same way. Both red and green colorblindness are sex-linked, meaning the relevant genes are located on a sex chromosome — in this case, the X chromosome. People assigned as female at birth generally have two X chromosomes, so if one carries a defunct gene, the other can compensate, preventing expression of the abnormal trait. People assigned male at birth, however, have only one X chromosome; if those genes code for colorblindness, nothing can prevent it, which explains the higher prevalence of red-green colorblindness in assigned men relative to assigned women. Tritanopia, by contrast, has no association with sex chromosomes and thus appears with equal frequency in all sexes. It is a dominant trait, meaning one copy of the gene is enough to produce the condition in all cases.

Then there is achromatopsia. Here, it is crucial to distinguish between achromatopsia that is present from birth (congenital) and achromatopsia that has been acquired, such as that of Mr. I. In people born with achromatopsia, all types of cone cells are either nonfunctional or absent, resulting in complete loss of color vision. Unlike Mr. I., born achromats do not describe their world as gray. To them, the concept of gray is as foreign as any other color. At least four genes have been implicated in congenital achromatopsia, which affects approximately one in thirty-three thousand people and, like tritanopia, is not sex-linked.

But what could cause the total colorblindness of Mr. I.? The sudden onset of achromatopsia indicated degeneration of the eye was not to blame, since degeneration requires a longer timespan. Moreover, his cones were functioning perfectly, as evidenced by his ability to see clearly in daylight. If only his rod cells had remained, their inability to function in regular light would have caused Mr. I. to find



daylight unbearable, even painful. This extreme sensitivity to light, called photophobia, is often found in people born totally colorblind, but not in those whose total colorblindness is acquired, since their cones are intact. Our painter's problem, then, lay not within his eyes.

Thus, the only explanations not ruled out were damage to the signal pathway between Mr. I's eyes and brain, or the visual cortex itself. When a visual signal leaves an eye through the optic nerve it passes through the thalamus, a relay station within the brain that decides where the signal goes next. Damage to the thalamus can cause achromatopsia, but such damage is usually internal, such as a tumor, since the location of the thalamus protects it from external harm. The visual cortex rests at the back of the head, making it far more vulnerable; it is easy to imagine Mr. I. injuring this part of his brain when he was rammed by the force of an oncoming truck. In the case of our colorblind painter, all tests conducted by Dr. Sacks suggested damage to the part of the brain that specializes in understanding color, an area of the visual cortex called V4.

Though colorblindness is often viewed as a disability, it can be advantageous in some cases. People who exhibit red-green colorblindness are not easily fooled by camouflage, which made them valuable soldiers in the World War II. Though achromatopsia would not be useful in such a circumstance, Mr. I., whose story was chronicled by Dr. Sacks in the phenomenal book An Anthropologist on Mars, grew to accept and even relish his new way of life. He became, in his own words, a "night person," taking nocturnal walks through city streets and delighting in his superior night vision. He returned to painting, 15 to 18 hours each day, but only in black and white. That was all the color he needed.