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COLOR PERCEPTION: AN ONGOING CONVERGENCE OF REDUCTIONISM AND PHENOMENOLOGY

Scientific explanation rests on two major approaches. The more familiar is the *reductionist or mechanistic explanation* in which complex processes or things are analyzed into components and these in turn are reassembled into a working facsimile or restoration of the original object or process. A good example of this is the work done with viruses which can be dissociated into their components (a nucleic acid and one or more proteins) and then reassembled in test-tube conditions without the need for a living host to provide support. At a cellular level such tour de force cases are rare. For the reductionist scientist this is mostly a matter of the cell's complexity. The second method is sometimes called *historical explanation*. This is used to interpret time-bound processes. Much of embryology would use this method to explain how an organ forms from its rudiments or from a few initiating cells at some earlier stage of development. A good example of this is a human pseudohermaphrodite with 46, XX karyotype (usually a normal female) who has a penis and a split scrotum leading to a vaginal passageway and internal uterus, oviducts, and ovaries. As one follows the fate of embryonic sexual rudiments and the influence of hormones at particularly sensitive stages one can recreate how such a contradiction to expectation arises. It can be duplicated experimentally in mammals which have a similar sex-determining mechanism. Time also plays an important role in the evolution of species or the divergence of related members of a species. Thus one can study sequences of nucleotides on the Y chromosome or sequences of nucleotides in mitochondria to work out the ancestry of human tribes, ethnic groups, and racial groups. Their origins can then be traced to a common ancestor out of Africa. One can limit the search to the common ancestor from Asia of all native tribes in North and South America. The underlying assumptions for this historical explanation involve mutation frequency and the spread of populations carrying adaptive or neutral mutant changes. Although one cannot do experimental evolution as readily as one can do experimental embryology, studies of Darwin's finches at the molecular level corroborate field studies that speciation is an on-going process of Darwinian small variations undergoing constant natural selection.

A.-T. Tymieniecka (ed.), Analecta Husserliana LXXVII, 3–11.

The reductionist or evolutionary scientist who uses these methods rejects explanations that assume *vitalism* (some nonmaterial essence that animates living matter), *holism* (some irreducible property of structure that cannot be explained by analysis of the components of a complex system), *emergence* (some unique property at higher levels of organization that is not explainable by the physics and chemistry of the level immediately preceding it), or the *supernatural* (some conscious activity of a divine creator or similar entity known only by faith).

The scientist's daily practice of reductionism and historical explanation does not exclude what can be called *the phenomenological interpretation* or description of reality. The complex world that philosophers like Merleau-Ponty describe is filled with nuances, subject to subtle changes, subjective, and sometimes ambiguous. A good example of this is our perception of color. We appreciate Merleau-Ponty's descriptions of the changing color of an object throughout the day from dawn to dusk, at night, and under artificial lighting. Color seems to change for an object in the context of its surroundings. A child (and sometimes we) often mistake colors that seem obvious to others. Moods alter our sensitivity to color and those in fabric arts deal with hundreds of shades and hues that most of us cannot name. Scientists do not reject these perceptions but they certainly cannot demonstrate a reductionist or historical interpretation that accounts for these observations. Instead scientists assume a complexity that will someday yield to more complete analysis. In many ways, I share that faith with my fellow scientists.

Are complex phenomena just complex? Are the blurring of interpretations and contradictions that we encounter primarily associated with our imperfect understanding of how our nervous systems work? I will argue that in the fifty years since Merleau-Ponty described color perception in phenomenological terms, the field of vision research has changed enormously. I will show that the interpretation of color vision is yielding to a reductionist interpretation although I acknowledge the story is far from complete. Enough has happened, however, to suggest that there is a convergence of the two outlooks and in not too many years there will be few of the phenomenological puzzles that cannot be explained by either the reductionist or historical explanations of traditional science. For my argument, I will survey what is known of color vision from the views of genetics, cell biology, biochemistry, molecular biology, psychology, evolution, and neurobiology. At the time Merleau-Ponty was active only the genetics and psychology was available and even there the knowledge was vastly less complete than it is now.

COLOR VISION: DESCRIPTIVE LEVEL

What we call red, blue, or green the physicist would describe as a response to measurable wavelengths of light separated into a spectrum. Red is perceived at 560 nm, blue at 440 nm, and green at 535 nm. It does not matter how different people see that color (we have no way of entering their minds) but we do know that when light of that wavelength is projected on a white screen, persons who are genetically normal for color perception, about 92% of males and 99.9% of females, all will respond with the same word. The perception of color is associated with retinal cells called cones. The perception of black and white is associated with retinal cells called rods. The cones tend to cluster centrally and the rods peripherally. Cone cells contain pigments called opsins. There are two major genes on the X chromosome (at Xq28 or near the tip of the long arm of the X) for red and green. Mutations in the red pigment cones cause protanopia (severe) or protanomaly (mild) defects in discriminating red (not seen) from green. Mutations in the green pigment cones cause deuteranopia (severe) or deuteranomaly (mild) and the persons with deutan defects cannot distinguish green from red. These are diagnosed by a variety of instruments and tests, one of the most familiar and accessible being Ishihara charts that use color dots that appear as numbers to those with full color vision or that appear as lacking numbers or having an altered number for those with protan or deutan defects. These color deficiencies are usually found in males because females are generally carriers and color deficient females only rarely arise from a carrier mother and a father with a color deficiency mutation. Both protanopes and deuteranopes have perfect yellow and blue discrimination.

The color seen by color-deficient males is known because occasional males are born with a mosaicism in which one eye is partially or completely red-green color deficient and the other eye is normal. Such males can score normally on Ishihara charts with the good eye but score as a typical deteranope or protanope with the defective eye. The mosaicism arises from a new mutation that arose after fertilization in one line of the cells of the embryo.

Less common forms of color deficiency include achromatopsia, in which no color is perceived and vision is monochromatic. Such individuals have poor daytime vision but excellent nighttime vision where they make use of their rod cells and can even perceive more stars in the night sky than those with normal functioning cone cells. One form of that disorder is associated with a mutation on 2q11 (on the second chromosome long arm near the centromere). The Pingalese in the Caroline Islands of the Pacific have a

founder effect (past ancestor) in which the gene was introduced by its early Polynesian settlers and then proliferated and became homozygous among its many cousin marriages. The achromatopsia members work at night and sleep during the day and build their homes in the more densely canopied growth of the inland jungle.

There is a defect of the blue cone receptor that leads to confusion of yellow and blue colors. It is associated with a gene on chromosome 7 and it is very rare and only one in 60,000 births produce such a child.

SOME HISTORICAL ASPECTS OF COLOR VISION

The first detailed description of what was called color blindness was by John Dalton [1766–1844], better known as the founder of the atomic theory of chemistry with its valence associations of atoms. In 1798 he published studies of his own color deficiency and those of members of his family, and he recognized that it was associated with males. A crimson ribbon had the same color others called "mud." He could not distinguish the color difference between red sealing wax and a green laurel leaf. He believed, falsely, that his vitreous humor had some blue pigment to it and that this was the cause of his defect, and he willed his eyes to be slit open upon his death. His vitreous humor was reported to be normal (colorless) and the remains of his eyes were preserved in a bottle. In 1802 Thomas Young proposed three primary colors, red, green, and blue, from which all other colors could be produced. This was confirmed by combinations of these colors from prisms. Over a century later the cones for recognizing primary colors were found to have pigments sensitive to red, green, and blue. In 1996 the desiccated eyes of Dalton were used to isolate his DNA. From it scientists could identify the defect he had. He had deuteranopia, a defect in the perception of green pigment. It is a triumph of reductionist scientific explanatory power when a genetic mutation can be examined a century or more after a person's death and reveal the precise lesion that caused his color deficiency.

A study of the Xq28 region revealed that there are usually several copies (up to four) of the deutan gene and only one copy of the protan gene in humans. The red, green, and blue cone genes have similar sequences and probably arose from a common ancestor in the vertebrates. New World monkeys have blue and red cones. Old World monkeys and apes have red, green, and blue like humans. About 40 million years ago after the old and new monkeys split into two populations, a duplication arose in the red cone and differentiated through mutations into a pigment capable of detection of

green color. Tandem duplication leading to gene evolution is a major mechanism of evolution first described by H. J. Muller ("all genes arise from pre-existing genes") in 1935. Duplications are inherently unstable and tend to throw off returns to unduplicated genes and proliferation of gene number. In *Homo* this process has led to a multiplication of the deutan gene. Those with two or three copies of the deutan gene usually have the same capacity to read Ishihara charts as those with four.

GENETICS OF COLOR DEFICIENCY

The overwhelming number of mutations that lead to color deficiency in the red-green system are not substitutions of single nucleotides but intragenic rearrangements. This arises from the inherent instability of the tandem duplication which undergoes an unequal crossing over during the production of eggs or sperm. The recombinant products that form the color deficiency are fusions of gene parts, usually a left piece of the red gene with a right piece of one of the green genes. Sometimes it is the reverse. The resulting hybrid gene leads to defective mutation if it is in second place, following the red gene. If it is in third place following an intact red and green gene, the hybrid gene is not expressed. This shows that the red and green genes are normally read and function in tandem. We can designate the recombinant hybrid molecules as follows: If the DNA is read from its chemical start at the 5′ end of the pigment genes and ends at its 3′ end, then the normal reading would be 5′RED-GREEN-GREEN3′ for the tandem genes on Xq28. The protanope or protanomalous individual has something like 5′RE/EEN-GREEN3′ where the segment D-GR is missing or deleted by unequal crossing over. By greatly diminishing the size of the RED gene, it no longer functions or only partially functions. In the deuteranope or deuteranomalous male the condition would look like 5′RED-GRE/EEN3′ where a segment, EN-GR, has been excised by unequal crossing over. Persons who are 5′RED-GREEN-GRE/EEN3′, where a recombinant segment follows the two normal red and green genes, would have normal vision.

Another feature of interest is that a woman who receives a deutan gene from her mother's egg and a protan mutation from her father's sperm has normal color vision. This shows that the red and green genes are separate and not just variant forms of the same gene. They produce what is called genetic complementation and act like two separate heterozygous pairs of genes. The story is actually more complicated than logic suggests because of a complication about our X chromosomes that the public knows little about