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*Cover by ED EMMH illustrating S.O.S. PLANET UNKNOWN,
Drawing for the TALES THEY TELL, by Kelly Froas.*

WONDERS ARE MANY

by

L. Sprague de Camp

THIS SECTION WILL, *deo volente*, be a regular feature of this magazine. It will consist of articles and fillers on factual matters of interest to readers of imaginative fiction. It will deal with curious, controversial, and speculative aspects of science, invention, history, mythology, pseudo-science, supernaturalism, and imaginative fiction.

If anybody has a subject he would like discussed, let him write me care of the magazine. I will not promise to comply with all requests, as some topics would be less fascin-

ating to most readers than to the persons proposing them, and there are some about which nobody knows enough to say aught. But I shall be glad of suggestions.

The title of this feature-column is from Sophokles' *Antigone*. The passage continues:

Wonders are there many—none more wonderful than man./His the might that crosses seas swept white by storm winds . . .

—(Hamilton translation.)

So, let us begin with a discussion of our future.

OUR BIOLOGICAL FUTURE — I

In a well-known science-fiction story,* the hero goes three million years into the future. At that point on the time-track, he finds that man has dwindled to thirty-five super-geniuses whose skinny little bodies cannot hold up their enormous heads without props. All they do is sit and think. Horrified, the hero massacres the lot and returns to his own time, excitedly boasting of killing off this race of "monsters."

Other writers have caused man to mutate into a superman, a telepath, or (after an atomic war) a freak with three eyes or two heads. Every time one of my colleagues broods on the hydrogen-bomb menace, he writes a story wherein man has been changed by hard radiations into something halfway between a human being and an emu, an aard-vark, or an octopus.

What is really likely to happen? In recent years, the sciences of genetics and evolution-theory have made such giant strides that we can now form quite a clear idea of how we got this way and what is now befalling us.

*Harry Bates: "Alas, All Thinking!" (*Astounding Science-Fiction*, June, 1935; reprinted in *The Other Worlds and Imagination Unlimited*).

In the last two million years, we evolved from small, erect, ground-living man-apes like those found fossil in Africa. Contrary to what was once thought, the bodies of these Australopithecines had become quite human-looking at a time when their heads were still for practical purposes those of anthropoid apes.

During most of this time, our forebears were tiny isolated bands of hunters. Three forces caused them to evolve. These were mutation, selection, and genetic drift.

Mutation is a sudden change in the mechanism of heredity. As you may know, nearly all the machinery of heredity dwells in the chromosomes. These are thread-like particles in the cells of living things. Different species have different patterns of chromosomes. The chromosomes are made of hundreds of particles, called genes, strung together. Each gene is thought to be a giant molecule of protein, like a tame plant virus.

A human sperm or ovum has twenty-four chromosomes. The

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number of genes in a complete set of these chromosomes is estimated at ten-to-twenty thousand. At conception, a sperm joins an ovum, adding its twenty-four chromosomes to those of the ovum. This makes forty-eight chromosomes in the embryonic cell, with twenty-to-forty thousand genes. Then, when the cell starts to grow, it divides into two. At this time the chromosomes go through a kind of ritual-dance, which ends with each chromosome splitting lengthwise into two. They have been making duplicates of themselves out of the chemicals in the cell. One set of forty-eight chromosomes goes into one of the new cells and the rest into the other.

Every time a cell divides, this division happens again, until the grown organism comes to create sex-cells for reproductive purposes. Then the chromosomes separate once without splitting, so that each sex-cell gets only twenty-four. The 24-chromosome sex-cell is called a *gamete* and the 48-chromosome body-cell a *zygote*.

As you see, every zygote has a duplicate set of chromosomes. If we give them Roman numbers, there are two I's, two II's, and so on up to the two XXIV's. If we number the genes on each chromosome, each No. I will begin with numbers 1, 2, and so on.

The two I-1 genes of a given zygote are much alike; so are the two I-2's, the two VI-437's, and the two

XX-986's. Often they are identical, in which case the organism is called a *homozygote* with regard to that gene, whichever it be.

However, the two genes of a gene-pair may differ. In fact, there may be a number of alternative patterns of gene that can equally well occupy a given gene-site, say the I-1 spot on either No. 1 chromosome, just as you might have any of several different makes of tire on the left front wheel of your car. These different gene-types that occupy a given site in the chromosomes are called the *alleles* of the gene. When different alleles of a gene occupy the two I-1 places, the organism is called a *heterozygote* as regards that gene.

Of each such pair of genes, you get one from each parent. Each of your children will get one gene like one member of each pair you have, but it is a matter of luck which one he will get.

Genes carry particular traits. That is, each gene affects the growth of at least one part of your body and decides its size, shape, color, or other quality. Many genes affect several parts each, while many characters like the color of your skin are the result of many genes, scattered among the chromosomes, acting together. Such a group of genes that work in concert is called a *polygene*. An organ controlled by a polygene is not inherited according to Mendel's simple ratios but follows more complex

formulas. The growth of the human brain is governed by a large polygene, so that genius in men does not follow any simple one-two-one ratio, like that which obtains with eye-color in men or height in sweet peas.

Thus you are a mosaic of characters that have come down from thousands of ancestors. And now for mutations.

Mutations were called "sports" by animal-breeders long before scientists studied them. The first mutation definitely recorded was a short-legged lamb, born in 1791 into a flock owned by Seth Wright of Massachusetts.

In the 1890's, the Dutch botanist Hugo de Vries recognized mutations as part of the mechanism of evolution. De Vries supposed that all mutations were big sudden changes that made a new species all at once. He thought this because he actually found such a drastic change in the evening primrose, *Oenothera*. In fact, however, this was a special kind of mutation called *polyploidy*: doubling the number of chromosomes. It is fairly common in plants but extremely rare in animals. When animal polyploids occur, they usually leave no descendants because their offspring are sexually abnormal.

The kind of mutation that often occurs in animals is the gene-mutation or point-mutation. Although genes can be passed on down for

thousands of generations unchanged, once in a while an accident betides one. Some atoms are knocked off or twisted askew, or an extra atom is added, or the gene's position in the chromosome is changed, or the gene is duplicated or lost. If such a change occurs in a gamete, and the new gene-pattern is passed on to an offspring, the offspring will have some new character that none of its ancestors had. The same gene can change in many different ways.

One gene in a gamete does not have much chance of mutating. However, with ten to twenty thousand genes in a sex-cell, mutation is not rare. Some geneticists think the human gamete has about one chance in four of mutating during its short life. Since you get one gamete from each parent, you have twice the chance of getting a mutation from two parents as from one. In other words, you have an almost even chance of being a mutant—that is, of differing from your parents by a mutation. As each of them had the same chance of being a mutant in their turn, your chance of differing from your grandparents by one or more mutations is higher yet—between four-fifths and seven-eighths—and so on back. For practical purposes, *we are all mutants*.

Then why don't we all have two heads? Because most mutations are small—so small we can hardly detect them. In fact, many mutations

may go on all the time with such small effects that we don't even know about them. They may make your digestion work a little better or worse, or your eyesight a little keener or dimmer, or your arteries harden a little sooner or later.

In point of fact, most mutations we know about are harmful or destructive. They make your eyes and digestion and arteries worse, not better. Constructive mutations cannot be more than a fraction of 1% of the total. The bigger the mutation, the smaller its chance of being good. Most drastic mutations are lethal. They kill off the organism in embryo or in infancy.

The reason for this is simple. A gene is an enormously complex little bit of biochemical machinery, exquisitely adapted to its task of controlling the growth of some part or parts of the body, either by itself or in coöperation with other genes. To expect a big random mutation to better it is like trying to improve a watch by hitting it with a hammer.

Even if the lethal mutations be barred from consideration, nevertheless, if all non-lethal mutants are given an equal chance of survival and reproduction, the overall effect of mutation is overwhelmingly harmful to the species, because the great majority of mutations are destructive.

While the number of possible mutations is enormous, and any gene may mutate in many ways,

some genes mutate more than others, and many undergo certain mutations over and over. Geneticists estimate that the mutation causing chondrodystrophic dwarfism (fetal rickets) occurs about once in every 10,000 human births, and that one responsible for hemophilia about once in 50,000. The hemophilia that Queen Victoria passed on to her descendants in the Russian and Spanish royal families was probably such a mutation.

Although rare, constructive or beneficial mutations happen, too. That is how evolution takes place. In a wild state, living things with destructive mutations tend to die young, while those with constructives have more than their share of offspring and take the place of those without them. Thus, in Europe, the black mutation of several moths has become beneficial around sooty cities because moths that have it are harder for hungry birds to see. Therefore these black moths have taken the place of the normal speckled races. Since men have been using antibiotic drugs on bacteria and new insecticides on insects, many species of bacteria and insects have developed strains immune to these poisons.

We know some but not all of the causes of mutations. Hard radiations bring on some; not only those from man-made things like X-ray machines and atomic bombs, but also from the slight but ever-present radioactivity of the air,

from cosmic rays, and the earth, from radioactive minerals. Dosing animals like vinegar-flies with X-rays greatly speeds up their rate of mutation.

As far as evolution is concerned, though, the slow action of the weak natural radioactivity, acting over the centuries, causes far more mutations than the violent radiations from atomic explosions acting over a short time only. It has been estimated that if atomic tests keep on at their present rate for a century, the mutation-rate of men might be raised by one and a half to three percent. Most of the monsters would die young as they do now, while no one mutation will make a superman. It will not even make a genius, because the growth of the brain is governed by a large polygene, and it takes the right alleles of all the genes of the polygene acting at once to make a genius. Genius is therefore a highly unpredictable event.

An atomic war would of course dose many more people at one time with radiations. But such a war would not likely last long. Therefore its long-term genetic effect would be much less than some have feared. Civilization stands to suffer far more in such a war from the annihilation of cities and the resulting breakdown of systems and institutions than from the rise in the mutation-rate of the survivors. Careless use of X-rays by people like shoe-salesmen may present as

big a hazard to our heredity as bombs.

Besides radiations, some chemicals affect the mutation-rate in some organisms. Mustard gas, some peroxides, and ethyl sulfate raise it, at least in experimental animals. So does formaldehyde, which automobile-exhausts spew forth. So do the purine bases, a group of chemicals, including caffeine, related to uric acid. Therefore it is not impossible that drinking tea and coffee increases mutations in men. Sudden changes in temperature speed mutations in insects but are less likely to affect large animals like us because our gametes are better protected.

An agency that promotes mutations is called a *mutagen*. Recently it has been found that there are *antimutagens* as well, which lower the rate of mutation. A small class of chemicals called ribosides have this effect on bacteria. Whether there are antimutagens for higher forms of life is not yet known.

How bad is it to raise the mutation-rate? Without some mutations we could never have evolved from lizards, and without them the breed of men could not be much improved in the future.

The extra mutations caused by the mutagens of civilization are of exactly the same kind as those that have been happening for billions of years. The effect of these mutagens is to cause more mutations,

but not mutations of a different kind. So we shall not get a race of two-headed men, bombs or no bombs.

However, some geneticists think that *all* mutations caused by hard radiations are destructives. They think the impact of such rays on the genes is too violent, too much like hitting a watch with a hammer, to improve them.

Moreover, many serious defects, like mongolian idiocy and cleft palate, may not be due to mutations. They may be caused, instead, by accidental damage to the growing embryo.

Nonetheless, there is still a sinister side to mutations. Among wild animals and primitive men, destructives are always arising and then being destroyed by the secondary great evolutionary force: *selection*.

This is the "survival of the fittest" of which Darwin's follower Spencer wrote. The expression does not mean that the fittest necessarily had to prove his fitness by walloping his fellow-caveman over the head. It merely means that he had to be enough stronger, healthier, smarter, brisker, and more fertile so that on the average he would leave more descendants.

Some common human traits that do not fit very well into civilized life can be explained on the ground that they helped those who had them to survive as hunters. For instance, reading that daily chron-

icle of the crimes and follies of mankind known as a newspaper, you may wonder at the bent of men to divide into factions along any convenient line of demarcation—racial, religious, linguistic, cultural, political, or merely sentimental—and fight it out with implacable hatred and bloodthirsty ferocity.

But, in a hunting-band, this factiousness has its use. Such a band has its optimum size. Under typical hunting-culture conditions, this size is about forty to eighty people, of whom ten to twenty are adult males.

At any one time, the area that the hunters can cover is limited by the distance they can hike away from their camp, kill their game, and drag it back to camp in a couple of days. If the size of the band much exceeds the optimum, there are more mouths to feed, but the hunters cannot hunt a larger area. They can only hunt the same area more intensely, whereupon the game is killed off or flees the neighborhood, and the band starves. Therefore it is to the hunters' advantage that, when the band exceeds a certain size, factions shall arise, and the quarrels among them shall force the band to split up. This is a speculation, but a reasonable one from what we know.

Again, people of the intellectual type have, from time immemorial, bemoaned the fact that they were such a small minority, lost in a sea of brainless buffoons and ignorant

ruffians. But, while a few intellectuals are useful to the hunting-band for shamans and bards, a band made up entirely of intellectuals would probably perish, because these deep thinkers would be speculating on the origin of the cosmos or inventing the bicycle when they should be keeping their minds on spearing that salmon.

In a wild state, then, the species is kept hale and whole and betimes even bettered by the interaction of mutation and selection. Destructive mutations are always arising but are likewise being destroyed by selection. Often, however, they are not gotten rid of right away, because most mutations are *recessives*. This means that both genes of a gene-pair must belong to the mutated allele of the gene before the effects of the mutation become apparent.

Therefore, living things that carry the mutation as a recessive trait suffer from it little or not at all but can still pass it on. If the mutation is one that happens again and again, the number of heterozygotes rises until there are so many that they sometimes mate with one another instead of with the unmutated majority. When this happens, a fraction of their offspring are homozygotes displaying the mutation. If it be a destructive, they tend to die sooner than others of their species. Thus the species reaches a balance between the rate

of mutation and the rate of elimination.

Sometimes a state of things called *balanced polymorphism* comes to pass. This is when a heterozygote is more viable than either of the two kinds of homozygote: the one with the mutation and the one without. For instance, in some malarial parts of Africa, people have a high proportion of the recessive gene-allele causing sickle-cell anemia in homozygotes. The heterozygotes (with one anemia-making gene) resist falciparum malaria better than those without this allele at all. So, while selection weeds out the homozygotes of this mutation, it tends to preserve the heterozygotes. And these bring forth more anemic homozygotes.

Thus mutations put constant pressure on a species, blurring its genetic design. A common type of mutation deprives the creature of an organ or a function. The loss of a gene is likely to have this effect. If the organ is not needed, the mutation happens again and again until the type without the organ becomes the normal one.

This is the process of *rudimentation*, which results in eyeless cave-creatures and those hairless primates called men. At some stage in their evolution, our ancestors dwelt in tropical forests where hair was not needed for warmth, so the hairless mutation got rid of it everywhere, save in a few spots

where it protected tender tissues against sunburn or insect-bites.

Thus any organ or function not protected by selection is apt to be lost. Moreover, if the genes that built the organ disappear completely, the organ probably cannot be regained. Mutations do sometimes occur in reverse—that is, canceling a previous mutation—but this is rare. Our forebears failed to re-grow their pelts on moving to colder climes and had to invent clothes instead.

Contrariwise, if an animal has a prominent organ, the organ is probably useful even if we do not know just how it is used. It once was thought, for example, that the woolly mammoth's tusks were useless, since they were so long and curly that they crossed at the tips and so were no good for fighting or digging. Now it has been guessed that they were used as snow-shovels to get at food in winter.

When the pressure of selection relaxes, as it has with mankind, many destructive mutations spread through the species because those who get them are not eliminated. Every advance in medicine enables more people with defects to live and breed like everyone else.

In other words, every species is subject to constant degenerative *mutation-pressure*. In the wild state, this is counterbalanced by selection; but civilization, by mitigating the rigor of selection, allows mutation-pressure to work its effects almost

unchecked. It might mean something that color-blindness is only about one percent in Eskimos, New Guineans, and Navahos, but around seven or eight percent among the long-civilized Chinese, Europeans, and white Americans.

The remaining evolutionary force is *genetic drift*. This is the random variation of small inbreeding groups away from the original type. It comes about partly through mutations and partly as a result of the chance loss of types without regard to their adaptive value. In a large interbreeding population, different alleles of the various genes occur in various proportions. These ratios will stay about the same indefinitely unless changed by selection.

Suppose, however, that neither brown eyes nor blue eyes have any advantage over the other, which is probably true. Then a large population in which, say, one-tenth of the people have the gene that makes blue eyes, will keep this proportion for ages. But, if the group is only twenty human beings, two with blue-eye genes, an accident can easily rid the group of the blue-eye gene for good. Some odd and apparently useless racial traits of small, long-isolated populations, like the high percentage of Rh-negative blood in the Basques, may be the result of genetic drift.

[*This is the first in a series of articles on our biological future.*]