Ancon sheep: just another loss mutation

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Many examples of mutations that produce phenotypic changes are 'loss mutations' in which the mutation causes the loss of a structure. Loss mutations that result in a non-functional protein or structure can be beneficial if the functional protein loss or malformation somehow benefits the organism (or, far more often, humans—as in the case of the loss of seeds in a fruit, producing a convenient seedless fruit).

One of the first and most common examples of the latter was an alleged new breed—Huxley called it a race, others labeled it a species—that resulted when Massachusetts farmer Seth Wright noticed in 1791 that he had a very short-legged sheep in his flock.[1,2] The story is usually claimed that, realizing the advantages of this trait to sheepherders, Wright bred a 'flock' of the short-legged 'species' of sheep, all of whom were unable to jump over ordinary stone walls or fences.[3,4]

Called the Ancon or Otter 'breed,' it was believed to reduce the need for tall fences, as well as reducing the number of lost sheep.[1] In addition, the short legs limited the sheep's ability to run so that, as a result, they were less active, more gentle, and gained weight far more readily then other sheep breeds.[5]

Charles Darwin and Ancon sheep

Charles Darwin was evidently the first person to use the Ancon breed as evidence for evolution. He discussed them at least three times in his published books. In the *Origin of Species,* first published in 1859, Darwin speculated that some animal variations 'have probably arisen suddenly', or by one step 'in one generation'. One example that Darwin used was 'the turnspit dog'. He then added that such 'one step' rapid evolution also is known 'to have been the case with the Ancon sheep'.[6] In another work, Darwin claimed that in a

> ' … few instances new breeds have suddenly originated; thus, in 1791, a ram-lamb was born in Massachusetts, having short crooked legs and a long back, like a turnspit-dog. From this one lamb the *otter* or *ancon* semi-monstrous breed was raised; as these sheep could not leap over the fences, it was thought that they would be valuable … . The sheep are remarkable from transmitting their character so truly that Colonel Humphreys never heard of "but one questionable case" of an ancon ram and ewe not producing ancon offspring. When they are crossed with other breeds the offspring, with rare exceptions, instead of being intermediate in character, perfectly resemble either parent; even one of the twins has resembled one parent and the second the other. Lastly, "the ancons have been observed to keep together, separating themselves from the rest of the flock when put into enclosures with other sheep".'[7]

Is the Ancon mutation beneficial?

Other evolutionists such as Kenneth Miller have also touted Ancon sheep as an example of evolutionary jumps. But this is deceptive because the condition actually is 'pathological', known as *achondroplasia* (where cartilage fails to develop, from Greek *a–*, not; *chondros*, cartilage; *plassein*, to mould or form—a form of dwarfism) or a related pathology,[8] and

> ' … would bring about the extinction of these creatures in a natural environment, rather than an advance through natural selection. The suggestion, by Miller, that the four-winged fly and the Ancon sheep present evolutionary advances was simply a deceptive ploy.'[9]

Actually, the mutation has proved lethal in a protected environment as well. Gish concludes that Ancon sheep are deformed animals, specifically, the

> ' … product of a pathological condition, called achondroplasia.

Courtesy Marcus Gellert

> In his presentation, Miller pointed out that these sheep have been bred by sheep breeders because they are short-legged and thus cannot jump fences—an advantage for those who raise sheep. What he did *not* say was that their condition is caused by a mutation which results in the failure of the cartilage between the joints to develop. There is thus little or no cartilage between the joints of their legs, causing them to be short. This abnormal condition would, of course, result in their rapid extinction in a natural environment and could never be considered an evolutionary advance.'[9]

The Ancon mutation, in harmony with our general experience with mutations, was harmful to the sheep for many reasons. Achondroplasia is a type of genetic dwarfism characterized by slow limb growth relative to the rest of the skeleton.[10]

Many other abnormalities aside from short legs have been discovered as a result of Ancon sheep postmortems. These included looser leg joint articulations, abnormal spines and skulls, flabby subscapular muscles, and crooked bent inward forelegs that caused the legs to appear like elbows while the sheep were walking.[11,12] This prominent trait is the reason for the term *Ancon* (*ancon* is the Latin transliteration of the Greek word for elbow, αγκων). The Ancon legs resemble the clubfeet condition, and, in fact, as adults were clumsy cripples that could neither run nor jump like other sheep.[13]

Conclusion

A major problem for Darwinists is that the Ancon mutation (a Mendelian recessive), as is true with most other mutations, is a loss mutation. This type

of mutation does not result in an information gain, as Darwinism requires, but an information loss (often of a complete structure or protein). A chief difficulty in arguing for macroevolution by mutations is the fact that most expressed mutations are either lethal or semi-lethal. Either they kill the organism outright, or they prove harmful, so that in the ordinary course of life they are eliminated. This includes both mutations in which the fertility rate is reduced as well as mutations that result in the loss of certain structures.

And as shown, even the rare 'beneficial' mutation, as some might consider the Ancon to be, are the result of information *loss*. Therefore they are going in the opposite direction from what goo-to-you evolution requires.[14]

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Jumping paradigms

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The paradigm that has ruled cell biology for more than a hundred years is under threat from Queensland's (Australia) wallabies. And the outcome just may provide creationists with a theory that explains the integrity of the created kinds.

In 1893, German biologist August Weismann published his 'germplasm' theory of inheritance. This says that 'germplasm' is the substance of inheritance, and it is transmitted independently of, and without interference from, the 'soma'—the body of the organism. Weismann's work refuted Lamarck's theory about characteristics acquired by the 'soma' being inherited. When the identity of the 'germplasm' was subsequently revealed to be DNA, Weismann's theory was turned into the 'central dogma' of molecular biology, which said that information could pass from the DNA to the cell, but not *vice versa*.

Although the 'central dogma' has been modified by the discovery of 'jumping genes' it still remains the major paradigm. Virtually all cell biologists today would believe that 'genes control cells'. One of the implications of this view is that there must be limits to genetic change, in order to maintain the integrity and viability of the cell. Experience with mutations shows that too much change can be fatal. If a genome were to become 'scrambled', 'completely haywire' and 'out of control' the cell should self-destruct. If 'very extreme and quite shocking' disfigurement occurred to the genome then drastic effects should result in the organism. If the equivalent of 'fifty million years of evolutionary change' (in evolution-speak) were to occur in 'five minutes' the impact on the organism should be catastrophic.

Well, apparently the rock hopper wallabies of the Queensland coast don't know this. Their genomes have suffered in exactly these ways yet the average person would think that nothing at all had happened to them!

Rock hoppers

Rock wallabies live on rock outcrops and cliff faces. Seven species occur widely scattered across Australia, mostly in geographically isolated populations, and they have distinctive colourations that tell them apart. But along the Queensland coast, another eight species live shoulder to shoulder in a linear geographic series, and they are so similar that only 'perhaps four people in the world'[1] could tell them apart by looks alone. It was not until genetic studies were carried out that the separate species were recognised.

Now in most animals the number and kind of chromosomes remains very stable. Any change to their number or structure is usually deleterious or fatal. But in macropods (wallabies and kangaroos), there is 'a tendency to play Lego® with their chromosomes, and in rock wallabies it's just gone completely haywire.'[1]

Recent studies were prompted by the curious case of 'Benny', a hybrid between two different species—the tall swamp wallaby and the tubby tammar wallaby.[2] Benny's chromosomes were found to have been seriously disfigured. Some of the centromeres (the place on the chromosome where the pairs join up) were ten times as long as normal; part of an arm of chromosome 2 had been moved to chromosome 7, and part of the X chromosome had been reversed. Analysis of Benny's DNA showed that it was 'dramatically under-methylated'. Methylation of DNA is a major method of controlling gene expression, so 'dramatically under-methylated DNA' means DNA that is 'out of control'. The researcher involved called it 'very extreme, and quite shocking'.[1]

When Benny's chromosomes with long centromeres were analysed they found pieces of retrovirus DNA repeated thousands and thousands of times. Retroviruses can insert themselves into a host's chromosomes, and on occasions may take with them a piece of the host's DNA, producing the phenomenon called 'jumping genes'.[2] The researchers suggested that perhaps