

Vitamin C Deficiency and Evolution

A Creationist Response

Lately, one of the most-used arguments for common ancestry between human beings and apes depends on our shared vitamin C deficiency. While this fact is not commonly known, almost all living creatures are capable of producing their own ascorbic acid (vitamin C). Apparently, only humans, apes and guinea pigs do not possess this ability. In and of itself, this might not be a particularly compelling bit of evidence. However, genetic studies seem to have discovered that the genes necessary for production of ascorbic acid are present in each of these cases, but are damaged in such a way that they no longer function. The “rubble” of the once-working genes is clearly identifiable. Since this is the case, it does not seem plausible that humans, apes and guinea pigs were originally designed by God without this trait. If that were the case, then we would not expect to have the non-functioning genetic material present. Evolutionists charge that this would be “wasteful” and “inefficient” and therefore an unworthy design to attribute to a perfect Creator. Most creationists would be inclined to agree, if the present genetic arrangement was actually equivalent to the original design.

Leaving guinea pigs aside for a moment, let us consider the argument as it is advanced as evidence of common ancestry. Actually, I have never seen the argument presented in a formal manner. In most cases, the use of vitamin C deficiency goes something like this:

Apes and humans requir[e] vitamin C in their diets. How loving of God to give people without adequate diets scurvy! For not only can most mammals synthesise their own, yet we cannot; we do have the gene for this — but it is broken! And it is rendered non-functional by precisely the same mutation in all the great apes. Coincidence?

(from the website, *Some More of God's Greatest Mistakes*, <http://www.freewebs.com/oolon/SMOGGM.htm>)

In order to be as generous as possible, let me represent the claim as a formal argument. I will attempt to structure it so that it is as strong an argument as possible:

A) Humans and apes both have the same genetic defect.

B) This shared defect is best explained by common ancestry.

Divine design is a problematic explanation and therefore should be rejected.
(see above)

Parallel mutation must be rejected. Parallel mutation refers to the idea that humans and each species of apes each separately experienced the same mutation. This is considered unlikely since this defect is almost entirely restricted to apes and humans. If the conditions which lead to the mutation are common, then many other kinds of animals ought to have the same genetic defect and this is not the case.

Common ancestry is the only other possibility. Common ancestry refers to the idea that apes and humans have a common ancestor in which the defect was first present. This explains why primates alone are both vitamin C deficient and in possession of the broken genes that once produced ascorbic acid.

C) Since A is true and B is true, humans and apes have a common ancestor.

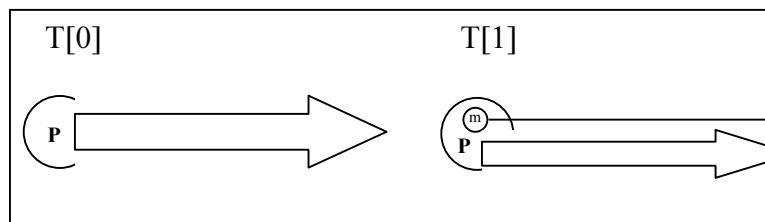
The conclusion listed above is valid, but it is true only if both A and B are true. Is it certain that common ancestry for apes and humans is the best explanation for our shared deficiency? No. At best, this argument is *probabilistic*; that is, it argues that C is the most probable conclusion.

As we will soon see, however, C is not the most probable conclusion. There are two reasons for this. The first has to do with the anomalous vitamin C deficiency in the guinea pig. The second reason has to do with the nature of the concept of common ancestry. Let's look at each of these in turn.

The existence of the same genetic defect in guinea pigs cannot be explained by reference to common ancestry. According to evolutionary theory, guinea pigs and primates would have had to split off of the mammal branch before acquiring the mutation. If the defect had occurred earlier on the biological tree, then all mammals (or at least a large segment of them) ought to have the same defect, yet they do not. Since at least two mammal families, but not all mammal families, have the defect, then this must be the result of either intentional design in those families, which does not seem plausible, or the result of *parallel mutation*. Since common ancestry cannot be the explanation for the genetic defect shared by apes and guinea pigs, then what requires it to be an explanation for the similarity between humans and primates? Even from an evolutionary perspective, where humans are simply one branch of the primate line, parallel mutation must be admitted as a possible explanation since this mechanism is clearly in operation.

In any event, the very nature of common ancestry, as advanced by evolutionists, would seem to refute the argument for common ancestry. The original entrance of this mutation into primates had to be limited. That is, at some point after primates split off from the larger mammalian branch, *a small number of individual primates, if not a single individual*, acquired the mutation. Mutations do not affect *every* individual organism within the population at the same time and in the same way. Mutations do not affect whole populations at all. Rather, they affect individuals within a population. It is not conceivable that every member of the primate population acquired the same mutation at the same time, otherwise we are really arguing for some sort of parallel mutation.

Let us call the time of the acquisition of this mutation by one or a small number of organisms, *Time 1* (T[1]). The important thing to note is that, at T[1], only a small percentage of the total population of primates (P) had the mutation (m):



In this scenario, what we would expect to find today, at T[n] would be a primate branch in which, broadly speaking, some individuals have the mutation and some do not. Thus, we would expect to find one of two things. One option would be a primate species which could not produce ascorbic acid in contrast to the other primate species which could. In other words, the primate sub-population *m* would have split off at some time to become a distinct species within the larger primate family. The second option would be to have the mutation present in some individual members of each species which emerged from the T[1] population. For example, we would expect to find some human beings who could still produce their own ascorbic acid and some who could not. The same would be true for all the other primate species (and guinea pigs). There is only one way, at least according to evolutionary theory, in which the mutation possessed by a small percentage of the population would become characteristic of all primates: it would have to be an advantage of some sort. The “law” of survival of the fittest holds that

genetic mutations become characteristic of whole populations, rather than of some individuals within the population, only when the mutation conveys some advantage on its possessor. This advantage then allows them to become, over time, the dominant variant within the population, eventually displacing all other variants.

If this were the case, however, then we would be suggesting that the inability to produce ascorbic acid would be an *advantage*. Yet, this is precisely the opposite of what evolutionists are suggesting. Their point is that the inability to produce vitamin C is a flaw in primates' genetic makeup and thus evidence of chance working on a common ancestor, which evidence is seen as refutation of the idea of a perfect Designer. But as we've just seen, simple chance could not produce the mutation in a whole population. Once anyone argues that the mutation was an advantage, the very reason the mutation was raised in the first place is demolished and the argument becomes absurd.

The only way around this objection is to suggest that the mutation happened much earlier on the biological tree, when the number of organisms within the target population was quite small. This would seem to require then, that the mutation be present in all mammals today, or at least in a larger number of the species. You can't have it both ways. Either the mutation entered when the population was very small and thus should be present in more mammals, or the mutation entered later and thus should only be present in particular species of primates or spread out among individuals within the primate family as a characteristic, much like blue eyes or blond hair.

So where does that leave us? As we saw above, one might argue that the mutation actually provides some benefit, but again, this undermines the function of the argument as evolutionists are using it. In any event, it does appear to be a disadvantage not to be able to synthesize ascorbic acid. If this were not the case, then we might be tempted to argue that God designed humans, other primates and guinea pigs this way. However, the apparent presence of the "broken" genes does seem to imply that there is a loss of function here. So, barring both design and common ancestry as viable answers to this question, we are left with the concept of parallel mutation.

Given the considerations we've just looked at, parallel mutation would appear to be the most likely candidate. The existence of the defect in guinea pigs would seem to require, even from an evolutionary perspective, that such parallel mutation does in fact occur. Furthermore, the fact that the genetic design of apes, and human beings is so similar, seems to suggest that perhaps there is something about our DNA arrangement that is susceptible to this particular mutation, given either present or past environmental conditions.

From a Christian perspective, what we are saying is this: Human beings, apes and guinea pigs were all designed to be able to synthesize their own ascorbic acid. At some point, however, environmental conditions changed (say, after the Fall or after the Flood) in such a way that some sort of damage occurred to the DNA sequence in humans, apes and guinea pigs. Why this damage was limited to these particular organisms is unknown, but this does not provide any great difficulty. Imagine a fleet of sports cars and SUV's driving down a broad highway at 100 m.p.h. Now, imagine that the highway takes a sudden jog to the right, so our hypothetical fleet attempts to make the sharp turn. The cars do all right, but the SUV's flip over. Why? Because conditions have changed in such a way that the top-heavy vehicles were being asked to do something they weren't designed for. Was this a design flaw? Well, it *could* be, but this assessment isn't necessary since what we are really dealing with is a failure resulting from conditions for which the original design was not intended.

Something similar is in view here. Animals weren't originally designed for the condi-

tions of a post-Fall world, so we would expect a certain amount of breakdown, malfunction and extinction given the present conditions. Why should only humans, apes and guinea pigs have this particular vitamin C “breakdown?” That’s impossible to say. My best guess would simply be that God designed humans and these other animals in such a way that they possess a degree of genetic similarity that happened to be susceptible to this particular damage under post-Fall conditions.

In any event, the evolutionary argument that the broken genes described here prove common ancestry for apes and human beings clearly does nothing of the sort. If anything, the current genetic state of affairs would appear to be inexplicable from an evolutionary perspective.