river valleys leading into Assyria, which is northern Iraq ... not ending up in Shinar, southern Iraq.

So it seems likely that they did not travel far to establish the base camp. If it was indeed near the Ark, then the site of the Ark is in the Zagros Mountains. That is where Babylonian and Assyrian legends put it.[10]

Where in the Zagros Mountains?

The mountains east of southern Iraq are desolate and sparsely populated. Some of them are high. One that strikes my eye on the map is Zard Kuh, or Zardeh Kuh (I think 'Kuh' means mountain in the local language), 4,547 m in altitude at roughly 50.05°E longitude, 32.4°N latitude. It seems to be significantly higher than other peaks near it. A river near it leads down to the plain. However, there are many other possibilities. I would look along any modern or ancient river valley that emerges onto the plain of southern Iraq, preferring mountains that are relatively close to the plain (figure 2).

Warning to Ark searchers: the area is extremely dangerous, being fought over by Kurds, Iraqis, and Iranians. It may be that God is using those means to keep the site of Noah's Ark from being revealed to the world until the time it suits Him.

References

1. Some histories suggest that the traditional mountain acquired the name 'Ararat' only as recently as AD 700. Often ancient Flood legends name a spectacular mountain near the people whose legend it is as the place where the vessel (sometimes a log, canoe, or cube) that saved their ancestors came to rest. Agri Dagh is one of the most spectacular local mountains—lofty, snow-capped, majestic, and isolated—that the Armenians could choose for their legend.
2. One was a pioneer creationist geologist, Clifford Burdick, in an early reference I no longer have at hand. Another is creationist geophysicist John Baumgardner, who visited the traditional site and told me that the mountain is without doubt a post-Flood volcano.
3. Zimansky, P.E., *Ancient Ararat: A handbook of Urartian Studies*, Caravan Books, Delmar, NY, 1998.
4. Lang, D.A., *Armenia: Cradle of Civilization*, 3rd ed., George Allen & Unwin, London, Map I, p. 86, 1980. Note location of the ancient Urartian site at Saqqiz (or Saqqez), in the Zagros Mountains at about the latitude of Mosul, Iraq.
5. As Genesis 8:11 indicates, trees would begin sprouting (for example from floating twigs, branches, or seeds present with them) immediately after the Flood. But it would take a number of years to establish mature forests again.
6. Pierce, L., In the days of Peleg, *Creation* **22**(1):46–49, December 1999; creation.com/in-the-days-of-peleg.
7. Holladay, W.L., *A Concise Hebrew and Aramaic Lexicon of the Old Testament*, W.B. Eerdmans Publishing Company, Grand Rapids, MI, pp. 200–201, 1974: 'basic meaning *out of, away from*'.
8. Also, there are no suitably high mountains, only a barren desert, just to the west of southern Iraq.
9. Archer Jr, G., Harris, R.L. and Waltke, B.K. (Eds.), *Theological Wordbook of the Old Testament*, 2 vols., Moody, Chicago, IL, 1980. Examples of the use of the word *nāsa'* as 'setting out' (KJV "took their journey") include Gen. 46:1, Exo. 16:1 and Num. 1:51.
10. Speiser, E.A., Southern Kurdistan in the annals of Ashurbanipal and today, *Annual of the American Schools of Oriental Research* **17/18**:1–43, 1926–1927. On p. 18, Speiser says that the Assyrians and the Babylonians regarded 'Mount Nisir' as the site of Noah's Ark. Speiser identifies Mt. Nisir with Pir Omar Gudrun (or Pira Magrun, or other variations). The ~2,750-m mountain is at the western edge of the Zagros range, 32 km northeast of the town of Sulaymaniyah (spelling varies), Iraq. It is often snow-capped and visible from 160 km away, well-known to the Babylonians.

The diminishing returns of beneficial mutations

Shaun Doyle

Beneficial mutations are often seen as the engine of microbes-to-man evolution.[1] However, beneficial mutations by themselves don't solve the problem of how to generate biological information (i.e. specified complexity[2]) *de novo*.[3] For that to occur, mutations not only have to be beneficial, but they have to add biological information. However, practically all beneficial mutations observed have been *losses* of specified complexity,[4] with only a tiny handful of highly disputable examples of mutations that increase information ever found (e.g. bacteria that digest nylon,[5] citrate[6] or xylitol[2]).

Epistasis: how do mutated genes interact?

However, mutations need to be more than beneficial and net-information-increasing to produce new coordinated structures and systems, as microbes-to-man evolution requires. Mutations don't act alone; the effect of a mutation on an organism's phenotype depends on other genes, and mutations in those genes. This is called *epistasis*, and describes the effects of one gene upon another in the process of gene expression. It is determined by assessing the difference between (1) the cumulative effect of several mutations on a given trait and (2) the sum of the effects of the individual mutations on that same trait (which assumes that there is no epistasis because mutations affect a given trait *independently*). Any difference suggests epistasis is occurring. Epistasis is an important consideration for evolution because the ways that mutations interact will determine if they could possibly build new structures in a stepwise manner.

For microbes-to-man evolution to occur, mutations need to be not just information-increasing and beneficial,

they also need to work together. This also has to be the main dominant trend in adaptive evolution so that the mutations can together produce new biological structures and systems. This phenomenon is called *synergistic epistasis* (SE), where the combined effect of mutations is greater together than the sum of their individual effects. This is obviously a good situation for beneficial mutations, but very bad for harmful mutations. SE of harmful mutations can result in *synthetic lethality*, where the combined effects of several harmful mutations are compounded by each other's presence, resulting in such an informationally deleterious effect that it kills the organism.[7] So evolution needs SE to be common *only in beneficial mutations*; it works against evolution when it occurs in harmful mutations.

Antagonistic epistasis (AE) is the opposite of SE. It occurs when mutations have a *negative* influence on each other, such that their combined effect is *less* than the sum of the effect of the individual mutations. For harmful mutations, this is a good thing because it mutes the effect of individual mutations and stalls error catastrophe.[8] This is no help for evolution in the long run, since they are still *harmful* mutations. However, AE presents problems for evolution if it occurs in beneficial mutations. The benefits of individual mutations are muted by other beneficial mutations, resulting in a decreasing rate of fitness gain with every beneficial mutation added.

How not to work together

Two recent studies investigated the effects that beneficial mutations have on each other and yielded very similar results. One study, by Khan *et al*., looked at the combined effect on fitness (by comparing the reproductive rate and morphology of the mutants with the wild types) from some of the earliest beneficial mutations to occur in Richard Lenski's "Long Term Evolution Experiment" on 12 *Escherichia coli* populations.[9] This is the same experiment in which an *E. coli* population produced the ability to utilize citrate under aerobic conditions, which it couldn't before. This was widely hailed as an example of 'evolution', but it actually involved a breakdown in regulation, which increased citrate-utilizing biochemistry that was already present in the bacteria.[6] Another study, by Chou *et al*., published in the same issue of *Science*, looked at the effect beneficial mutations have on each other in an engineered strain of *Methylobacterium extorquens*.[10]

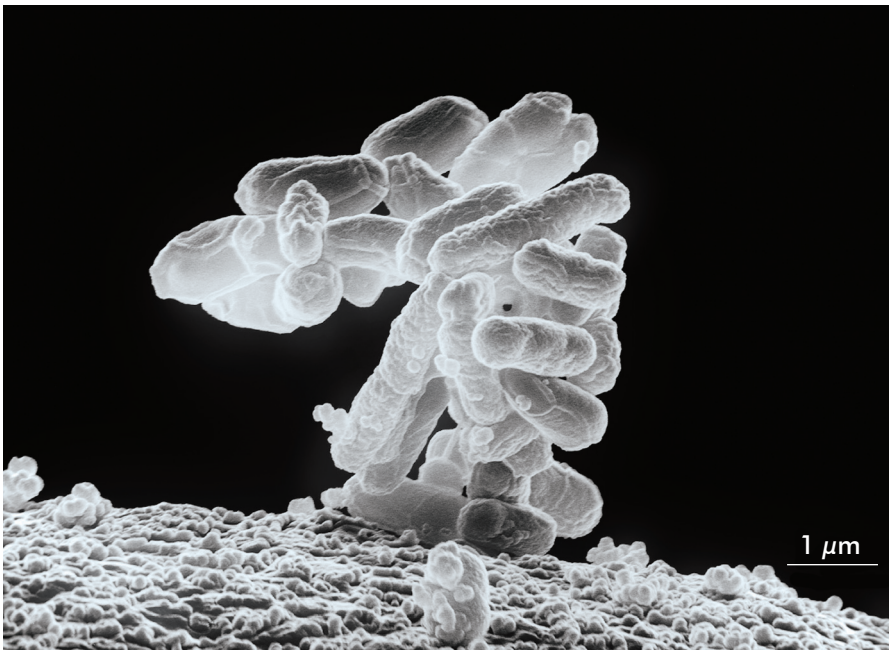

Photo by Eric Erbe

Figure 1. Low-temperature electron micrograph of a cluster of *E. coli* bacteria, magnified 10,000 times. Each individual bacterium is oblong shaped.

Both studies found that beneficial mutations, to be defined below, interacted under an overall trend of *antagonistic* epistasis. Khan *et al*., in comparing their study with that of Chou *et al*., pointed out that the results of both studies were virtually identical:

> "Note that similar trends were seen by Chou *et al*. … That study, like ours, found that four mutations interacted to yield diminishing fitness returns, whereas one mutation had the opposite effect."[11]

Therefore, the cumulative effect of the "beneficial" mutations together was smaller than it would be if the mutations were considered independently—i.e. they display an overall trend of AE. Some individual mutations displayed synergistic epistasis, but they were a minority, and were not enough to reverse the overall antagonistic trend.

Khan *et al*. explain this as a result of environmental adaptation:

> "Mechanisms that may explain this deceleration include reductions in the number and effect-size of beneficial mutations as a population becomes better adapted to its environment … In other words, epistasis acts as a drag that reduces the contribution of later beneficial mutations."[11]

But is this the case? No doubt this is a fair assessment of these results as far as they go. These experiments were done in strictly controlled environmental conditions, so the range of questions that can be answered is limited. However, these results didn't take into account environmental flexibility and change. Khan *et al*. observed examples of previous mutations that stymied the adaptive capabilities of some lines relative to others in the population.[12] This suggests that, because the 'beneficial' mutations destroy information and

because there is only a finite amount of information in the genome, the population as a whole loses the ability to adapt to new environments in the future.[13]

What is a beneficial mutation?

Both studies stated they were studying *beneficial* mutations. But what do they mean by beneficial? Are these mutations *universally* beneficial, or only within a certain environmental context? These may seem like trite questions, but they become immensely important when we consider the context of these studies. As stated above, these are laboratory studies conducted in *strictly controlled* environments, so the mutations observed are only known to be 'beneficial' within a strict environmental context.

Moreover, Chou *et al*. conducted their experiments on an *engineered* bacterial strain that, even without mutations, grew three times *slower* than the wild-type in the same environment.[14] In the engineered strain, Chou *et al*. eliminated an essential metabolic pathway and replaced it with another from a different species. All the 'beneficial' mutations in the engineered strain were merely compensating for the loss of the native metabolic pathway. The same mutations in the wild type would most likely be *harmful*. This displays the amazing amount of contingency built into these cells, but fails to support evolution because evolution needs a *net* benefit to have any plausibility.

Finally, a beneficial mutation is not necessarily a mutation that increases specified complexity.[15] Something is beneficial if it confers a growth advantage, not simply if it adds information. This points to an important issue: mutations not only have to add information to support evolution, but they also have to be selectable. Since mutations (apart from a few trivial examples) are universally losses of specified complexity, and natural selection is incredibly slow and weak, beneficial mutations are ultimately no help to evolution.

Genetic entropy and the mystery of epistasis

These studies reflect a universally consistent trend in lab experiments on adaptation:

> "The most consistent finding across studies of laboratory-evolved populations has been a *rapid deceleration* of the rate of fitness increase."[14]

The two scientific reports discussed above are in line with those consistent results, and serve as further confirmation of the concept of universal genetic entropy, as described in Dr John Sanford's landmark book: *Genetic Entropy and the Mystery of the Genome*.[16] Sanford pointed out that the genome is in a state of inexorable decay because of mutation accumulation. Mutations are occurring at a much greater rate than previously imagined, and most of these mutations are 'near neutral'; i.e. their deleterious effect is not enough for natural selection to be able to get rid of them. So they keep accumulating, and together, their cumulative deleterious effect is profound. It will eventually lead to the extinction of all multicellular life. If beneficial mutations generally get in the way of each other, their combined effects cannot stop this process of decay in the genome.[17] Evolution by mutations thus has three equally fatal strikes against it:

1. too few mutations are beneficial,
2. practically all mutations destroy specified complexity even if they confer greater survivability in a specific environment,
3. 'beneficial' mutations display an overall trend of working against each other (antagonistic epistasis).

While mutations may be of limited benefit to a single organism in a limited context (e.g. sickle cell anaemia can protect against malaria even though the sickle cell trait is harmful), mutations are no benefit whatsoever for microbes-to-man evolution, whether individually or together.

References

1. Williams, A., Mutations: evolution's engine becomes evolution's end! *J. Creation* **22**(2):60–66, 2008; creation.com/mutations-are-evolutions-end.
2. Batten, D., How is information content measured? 10 September 2001; creation.com/how-is-information-content-measured.
3. Wieland, C., Beetle bloopers, *Creation* **19**(3):30, 1997; creation.com/beetle-bloopers.
4. Wieland, C., The evolution train's a-comin', *Creation* **24**(2):13–16, 2002; creation.com/the-evolution-trains-a-comin.
5. Batten, D., The adaptation of bacteria to feeding on nylon waste, *J. Creation* **17**(3):3–5, 2003; creation.com/nylon.
6. Batten, D., Bacteria 'evolving in the lab'? 14 June 2008; creation.com/citrate.
7. Kaelin Jr, W.G., The concept of synthetic lethality in the context of anticancer therapy, *Nature Reviews Cancer* **5**:689–698, 2005.
8. *Error catastrophe* occurs when harmful mutations accumulate faster than natural selection can weed them out. It leads to a continuous fitness decline in every generation. If not reversed, it will result in the extinction of the population.
9. Khan, A.I., Dinh, D.M., Schneider, D., Lenski, R.E. and Cooper, T.F., Negative epistasis between beneficial mutations in an evolving bacterial population, *Science* **332**:1193–1196, 2011.
10. Chou, H.-H., Chiu, H.-C., Delaney, N.F., Segrè, D. and Marx, C.J., Diminishing returns epistasis among beneficial mutations decelerates adaptation, *Science* **332**:1190–1192, 2011.
11. Khan *et al.*, ref. 9, p. 1195.
12. Khan *et al.*, ref. 9, p. 1193.
13. Anderson, K.L. and Purdom, G., A Creationist Perspective of Beneficial Mutations in Bacteria; in: Snelling, A.A. (Ed.), *Proceedings of the Sixth International Conference on Creationism*, Creation Science Fellowship, Pittsburgh, PA, and Institute for Creation Research, Dallas, TX, pp. 73–86, 2008.
14. Chou *et al.*, ref. 10, p. 1191.
15. Carter, R. Can mutations create new information? *J. Creation* **25**(2):92–98, 2011.
16. Sanford, J., *Genetic Entropy and the Mystery of the Genome*, 3rd ed., FMS Publications, New York, 2008.
17. Thomas, B., The cost of adaptations limits evolution, *ICR News*, 13 June 2011; icr.org/article/cost-adaptations-limits-evolution/.