Many paths lead to high-altitude adaptation

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One marvellous characteristic of living things is the ability to respond to their environment in a way that sustains life and allows for procreation. This is clear evidence of a wise Creator who blessed living creatures to reproduce and fill the earth (Genesis 1:21–22, 27–28; 8:17; Isaiah 45:18). Of great interest to creation biologists is the underlying design that has enabled living creatures to adapt to numerous, varied environments in a bit over 4,300 years since the Flood.

Using the history in Genesis and observations from the world around us, creation scientists recognize that within various created kinds of plants and animals, considerable diversity has arisen.[1] While it is well known that substantial variation has arisen in domestic species (e.g. dogs) within the last few hundred years, evolutionists imagined that the process is orders of magnitude slower in the wild. Given the biblical timeframe, this is clearly not the case. In recent decades, scientific studies have confirmed many examples of rapid diversification in the wild.[2] Creation geneticists have begun to explore some of the mechanisms that appear to be involved.[3]

As more studies uncover the rapid pace of changes and their underlying mechanisms, it becomes increasingly apparent that God designed His creatures with an amazing ability to adapt to a wide variety of challenges that He knew they would encounter in this world. A brief glimpse of this can be seen in organisms that have adapted to living at high altitudes.

Taking the high road

It is well known that high-altitude environments pose serious challenges because of reduced (partial pressure of) oxygen and generally cooler temperatures. For warm-blooded animals, mammals and birds, this can impair not only normal activity, but also the maintenance of a constant body temperature. Indeed, ascending to very high altitudes too rapidly can cause severe sickness or death (acute mountain sickness).[4]

Yet God, in his providence, has designed our bodies, and those of other creatures, with the inherent ability to adapt. Most people can travel by plane to cities well over a kilometre above sea level (e.g. Quito, Ecuador, 2,850 m; Bamyan, Afghanistan, 2,550 m; Mexico City, 2,250 m; Santa Fe, NM, 2,194 m), and with proper precautions (e.g. avoiding heavy exertion for the first few days and increasing fluid intake) can remain healthy and adjust well. Here the underlying adaptive changes are epigenetic, involving changes in gene expression that allow our bodies to adjust to environmental challenges, including high altitudes. This is known as *physiologic adaptation*.

Physiologic adaptation to high altitude is not a matter of making a single adjustment. Instead, multiple, complex, well-coordinated changes in gene expression result when the body detects the drop in oxygen. The obvious immediate changes are an increase in respiratory and heart rate. Yet these cannot be maintained for extended periods, so this is followed by other changes which are more sustainable. Some of these changes have been studied in detail.[4]

Another immediate change to the reduced oxygen at high altitudes is an increase in hypoxia-inducible factors (HIFs), transcription factors regulating a variety of genes involved in the response. One of the genes upregulated by HIFs is the erythropoietin (EPO) gene. Within a day or two, EPO levels peak. Among the functions of EPO is stimulating erythropoiesis, or the production of red blood cells. Within a few weeks, there is a substantial increase in circulating red blood cells to help deliver sufficient oxygen to the tissues.[4]

Adaptive physiological changes are even more dramatic in young animals that develop under high-altitude conditions. Studies in beagles showed that adaptive development in the lungs and associated vasculature increased diffusion, resulting in greater adaptation than seen in adults. This is from altered gene expression during development, which is also epigenetic in nature (i.e. gene sequence is not changed to accomplish this). In some cases, epigenetic changes can be heritable, or passed on to offspring. It is currently unknown if this may also play a role in high-altitude adaptation.[4]

Yet God's provision does not stop with physiologic adaptation as described above. In populations of people and animals that have lived at high altitudes for many generations, adaptive genetic changes have also been documented.

Parallel paths with divergent steps

There are interesting patterns that may be seen with *genetic adaptation*; often the same genes are involved in multiple, independent adaptions to a specific environment.[5] This is true in birds that have adapted to high altitudes. One study compared 28 high-altitude adapted avian species with their nearest lowland relatives. In most cases there were nonsynonymous mutations (i.e. those causing an amino change) affecting one of the several hemoglobin subunits which resulted in a higher oxygen affinity, corresponding to an adaptive phenotype.[6] The question is: is the same mutation occurring repeatedly, or are different mutations in hemoglobin genes responsible for the same adaptive phenotype in these species that independently colonized high-altitude environments?

In most cases different amino acid changes in hemoglobin were

responsible for this adaptive phenotype. This indicates that different mutations were responsible, and a variety of changes in the hemoglobin gene can lead to an adaptive result. So, a parallel adaptive path (increasing oxygen affinity of hemoglobin through mutation in hemoglobin genes) usually had different molecular steps. How amazing that the design of hemoglobin allows for these adaptive possibilities! Certainly designed mechanisms must also be in place for them to arise in a timely fashion (i.e. over a limited number of generations, within hundreds or thousands of years)!

The study revealed a few cases where the same amino acid change was responsible for the increased oxygen affinity of hemoglobin. The most notable was among hummingbirds and one passerine species where a substitution to the amino acid serine (N83S in the β^A-globin subunit) resulted in the increased affinity of hemoglobin to oxygen. The authors were suspicious that the genetic background would influence the effectiveness of a particular amino acid change. Through site-directed mutagenesis this mutation was produced on other genetic backgrounds, and it did not result in the same adaptive change in oxygen affinity. Thus, at least in some cases, a change that is effective in one lineage of organisms may not be in another.[6]

It should be noted that evolutionists usually consider the same mutation in different species as very strong evidence of common descent. This is because they assume the underlying mutations are random and in no way *designed* to occur in a manner that is biased to be adaptive. However, parallel changes have been increasingly documented, making it clear that mutations are not always random. This erodes arguments for universal common descent.[7] Many mutations are clearly biased, and it would appear that this is often in a way that promotes genetic adaption.[8]

Different paths to adaptation

The same gene is not always used in adaptation. Human populations that are adapted to high altitudes have mutations in different genes that allow them to thrive under these hypoxic conditions. For example, in Tibetans, three genes (EGLN1, PHD2, and EPAS1) that are part of the pathways used to adapt physiologically (EGLN1 affects HIF levels, PHD2 codes for part of a HIF molecule, and EPAS1 affects erythropoiesis), were found to have an altered sequence.[4] Yet, different genes appeared to play a prominent role in the genetic adaptation of Ethiopian highlanders and Andeans.

It is important to recognize that such adaptations aren't always due to one or a few genes with a major impact on the affected physiological pathway. Instead, many genes may be involved, which all contribute a little to the adaptation. Many genes with little effects are hard to identify, but continued studies in humans have now recognized over 1,000 genes that appear to be involved to some extent. Further analysis showed that 64 of these genes had undergone changes in at least two of the three high-altitude human populations (i.e. Tibetans, Ethiopians, and Andeans). Interestingly, well over a hundred of the genes implicated in high altitude adaptation are involved in the same biological processes: the circulatory system, angiogenesis, and erythrocyte homeostasis/oxygen transport.[9] This is clear evidence of design that allows for different genes in relevant pathways to be modifiable, facilitating genetic adaptation.

Summary

The ability of living things to adapt to challenging environments, such as the hypoxic conditions at high altitudes, is truly awe-inspiring. These adaptive changes require the existence of complex, well-integrated pathways to control all of the essential functions of life. Yet, this is not a rigid complexity, but one that allows for changes that are useful, both physiologically and genetically. This staggeringly complex design of life clearly points to a wise Creator, who through wisdom and foreknowledge, created life to fill the earth.

References

1. Two examples were reviewed in: Lightner, J.K., Karyotypic and allelic diversity within the canid baramin (Canidae), *J. Creation* **23**(1):94–98, 2009; Lightner, J.K., Identification of a large sparrow-finch monobaramin in perching birds (Aves: Passeriformes), *J. Creation* **24**(3):117–121, 2010.
2. Some of the best studied examples in vertebrates involve fish trapped in fresh water lakes and lizards on islands. Two specific examples are:

   Lescak, E.A., Bassham, S.L., Catchen, J, *et al.*, Evolution of stickleback in 50 years on earthquake-uplifted islands, *PNAS* **112**(52):E7204–7212, 2015.

   Kolbe, J.J., Leal, M., Schoener, T.W. *et al.*, Founder effects persist despite adaptive differentiation: a field experiment with lizards, *Science* **335**(6072):1086–1089, 2012.
3. Many were discussed in detail in the excellent series on Evidence for the Design of Life: Terborg, P., Evidence of the design of life: part 1—genetic redundancy, *J. Creation* **22**(2):79–84, 2008; Terborg, P., Evidence of the design of life: part 2—baranomes, *J. Creation* **22**(3):67–76, 2008; Terborg, P., Evidence of the design of life: part 3—an introduction to variation-inducing genetic elements, *J. Creation* **23**(1):99–106, 2009; Terborg, P., Evidence of the design of life: part 4—variation-inducing genetic elements and their function, *J. Creation* **23**(1):107–114, 2009.
4. Reviewed in Lightner, J.K., Adaptation of endotherms to high altitudes, *CRSQ* **50**(3):132–140, 2014.
5. Martin, A. and Orgogozo, V., The loci of repeated evolution: a catalog of genetic hotspots of phenotypic variation, *Evolution* **67**(5):1235–1250, 2013.
6. Natarajan, C., Hoffman, F.G., Weber, R.E. *et al.*, Predictable convergence in hemoglobin function has unpredictable molecular underpinnings, *Science* **354** (6310):336–339, 2016.
7. Terborg, P., An illusion of common descent, *J. Creation* **24**(2):122–127, 2010.
8. Another example in a colour-coding gene is discussed in Lightner, J.K., Genetics of coat color I: the melanocortin 1 receptor, *Answers Research J.* **1**:109–116, 2018.
9. Azad, P., Stobdan, T., Zhou, D., Hartley, I., Akbari, A., Bafna, V., and Haddad, G.G., High-altitude adaptation in humans: from genomics to integrative physiology, *J. Molecular Medicine* **95**(12):1269–1282, 2017.