

webbing by the mechanisms that they have elucidated only provides an explanation for how a bat's forewing develops in the embryo. Their claim begs many questions of its own. *Fgf8* is a complex molecule and an explanation would be needed for: (a) how the gene(s) for its production arose by random mutations; (b) how the molecular targets for *Fgf8* arose in the bat wing tissues; (c) the origin of the cooperative effect between the emergent *Fgf8* molecule and the Bmps; (d) how these molecules became key components of the whole apoptosis machinery; (e) the elongation of the digits themselves; etc. A bat wing is irreducibly complex at the macro, the micro and the molecular levels in spite of evolutionists' protestations to the contrary. The researchers' neo-Darwinian claims are nothing more than wishful thinking. In fact, contrary to their own expectations, they admit that their findings provide no support for 'a conserved mechanism for maintaining interdigit tissue across amniotes' (Abstract).

The experimental research reported by these researchers is fascinating in itself and certainly worthy of the attention of their peers. Central to their research was the status of apoptosis, a phenomenally sophisticated and tightly controlled process, involving a bewildering array of molecular components, whose alleged conservation during evolution beggars belief.⁶ To show that the retention of interdigital bat wing membrane is due to the prevention of apoptosis advances our understanding of its role in wing development but is quite unhelpful to the authors' own evolutionary speculations. They have demonstrated the system complexity responsible for normal wing development: co-expression of *Gremlin* and *Fgf8* and inhibition of Bmps conspire to prevent programmed cell death of interdigital tissue but *not* the digits themselves. Logically, disruptions of this system of complex molecular signalling between interdependent components would likely lead to abnormal wing development and a flightless bat that cannot

feed—hardly convincing evidence for the system's random, piecemeal assembly over time. In conclusion, borrowing from the authors' own words in their paper, 'The evolution of flight in bats is a matter of conjecture.' To argue 'In the beginning, God created ...' is no more presupposed (and no less scientific) than to contend for a naturalistic origin for the Chiroptera.

References and notes

1. Actually, digits 2, 3, 4 and 5 are considerably elongate but the first digit (the 'thumb') is much shorter and, unlike the other four, plays no part in supporting the wing membrane.
2. Weatherbee, S.D., Behringer, R.R., Rasweiler IV, J.J. and Niswander, L.A., Interdigital webbing retention in bat wings illustrates genetic changes underlying amniote limb diversification, *PNAS* **103**(41):15103–15107, October 10, 2006.
3. For instance, see references 51 and 52 of: Bell, P., Apoptosis: Cell 'death' reveals creation, *Journal of Creation* **16**(1):90–102, 2002. Among other things, this paper illustrates details of the cellular program of apoptosis, the mechanisms involved and some of its diverse functions within normal living organisms. See also the erratum for Figure 1 of this paper, *Journal of Creation* **16**(3):126, 2002.
4. In humans, mutations are known that activate Fgf receptors, thus preventing programmed cell death and leading to joined fingers and toes (syndactyly). See Wilkie, A.O. *et al.*, Apert syndrome results from localized mutations of FGFR2 and is allelic with Crouzon syndrome, *Nature Genetics* **9**(2):165–172, 1995.
5. Subtitle inspired from a statement made by Philip Skell about the invocation of Darwinian evolution after the scientific research has been completed but for which it 'provided no discernable guidance'; see Why do we invoke Darwin? *The Scientist* **19**(16):10, 29 August 2005.
6. Bell, P.B., The non-evolution of apoptosis, *Journal of Creation* **18**(1):86–96, 2004.

More marvellous machinery: 'DNA scrunching'

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Some of the most startling discoveries in the last few decades have improved our understanding of the amazing complexity of the cell. This includes the world's tiniest machines.¹ But not only are there machines, but also their *blueprint*—the message molecule DNA.² DNA's function is to store and transmit genetic information, but it can't work without many molecular machines. However, as the noted philosopher of science, Sir Karl Popper (1902–1992), commented:

'What makes the origin of life and of the genetic code a disturbing riddle is this: the genetic code is without any biological function unless it is translated; that is, unless it leads to the synthesis of the proteins whose structure is laid down by the code. But ... the machinery by which the cell (at least the non-primitive cell, which is the only one we know) translates the code consists of at least fifty macromolecular components *which are themselves coded in the DNA*. Thus the code can not be translated except by using certain products of its translation. This constitutes a baffling circle; a really vicious circle, it seems, for any attempt to form a model or theory of the genesis of the genetic code.

'Thus we may be faced with the possibility that the origin of life (like the origin of physics) becomes an impenetrable barrier to science, and a residue to all attempts to reduce biology to chemistry and physics.'³

Transcription tricks

Now Richard H. Ebright and his team from Rutgers University have discovered more intricacies in the

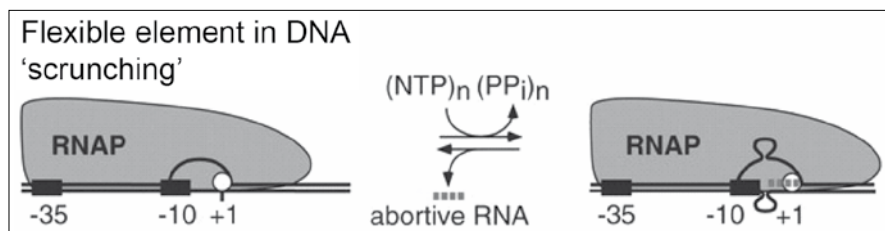


Figure 1. The ‘scrunching’ model for RNAP–active-centre translocation during abortive initial transcription.^{5,6}

process of *transcription*,⁴ where information from the right part of the DNA is copied onto a strand of messenger RNA (mRNA).^{5,6} Indeed, it is this mRNA that is translated into proteins in the complex machines known as ribosomes.^{7–9}

DNA is double stranded, so must first be unwound, so that the right strand can be copied onto mRNA, in a sense like a photographic negative. So the machine, called RNA polymerase (RNAP), first locks on to the start of the gene. Ebricht and colleagues demonstrated what happens next with two complementary techniques, single-molecule fluorescence resonance energy transfer (FRET)⁶ and single-molecule DNA nanomanipulation,⁵ and were able to rule out other ideas of how it works.

The next stage is that the anchored RNAP then reels in the DNA—*scrunching* (figure 1).¹⁰ This unwinds the double strand so the messenger RNA copy can be formed off one of them. Also, the unwinding stores energy, just like winding the rubber band of a rubber-band-powered airplane. And just like the toy plane, this energy is eventually released, with the machine then breaking free of its starting point and shooting forward. This also rewinds the unwound DNA (‘unscrunching’) which escapes from the back of the machine.

Ebricht states that this research should also enable them to develop antibacterial agents that target the bacterial version of this machine.⁴

Evolutionary conundrum

This discovery provides yet more support for Popper’s bafflement. The instructions to build RNAP are

themselves encoded in the DNA. But the DNA could not be transcribed into the mRNA without the elaborate machinery of RNAP. And this is also an example of *irreducible complexity* because it would not be able to perform its function unless every feature was working fully. There would be no use being able to dock onto the right spot of the gene and getting stuck there, or unwinding the DNA without being able to wind it back.

Furthermore, RNAP uses ATP as an energy source to achieve its feats. And ATP is made by another nanomachine, the ATPase complex, which is a rotary motor.¹ This is also coded on the cell’s DNA.

Natural selection is no answer, because this means differential reproduction, i.e. fully formed self-reproducing entities that can pass on the information that codes for their features. But until RNAP is fully formed, the coding would not work at all, being unable to get past first base (pun intended). Thus Darwinian evolution could not even have got off the starting block.

References

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2. Sarfati, J., DNA: marvellous messages or mostly mess? *Creation* **25**(2):26–31, 2003; <www.creationontheweb.com/message>.
3. Popper, K.R., ‘Scientific reduction and the essential incompleteness of all science’; in Ayala, F. and Dobzhansky, T., (Eds.), *Studies in the Philosophy of Biology*, University of California Press, Berkeley, p. 270, 1974. We now know that there are many more than 50 ‘macromolecular components’ necessary.
4. *PhysOrg.com*, Nanotech tools yield DNA transcription breakthrough, <www.physorg.com/news82910354.html>, 16 November 2006.

5. Revyakin, A., Liu, C., Ebricht, R.H. and Strick T.R., Abortive initiation and productive initiation by RNA Polymerase involve DNA scrunching, *Science* **314**(5802):1139–1143, 17 November 2006.
6. Kapanidis, A.N., Margeat, E., Sam On Ho, Kortkhonja, E., Weiss, S. and Ebricht, R.H., Initial transcription by RNA polymerase proceeds through a DNA-scrunching mechanism, *Science* **314**(5802):1144–1147, 17 November 2006.
7. In ‘higher’ organisms (eukaryotes), it is necessary to remove non-coding parts called *introns* and splice the coding parts (*exons*) together. This requires elaborate machinery called a *spliceosome*—a scientific paper was entitled, ‘Mechanical devices of the spliceosome: motors, clocks, springs, and things’, (Staley, J.P. and Guthrie, C., *Cell* **92**(3):315–326, 1998). This is assembled on the intron, chops it out at the right place and joins the exons together. This must be in exactly the right direction and place, because it makes a huge difference if the exon is joined even one ‘letter’ off.
8. Andrew Fire and Craig Mello won the 2006 Nobel Prize for Physiology and Medicine for their 1998 discovery of RNA interference, where double-stranded RNA can silence the gene, i.e. block protein synthesis. Many of our genes encode small RNA molecules called microRNAs that contain pieces of the code of other genes. These can form a double-stranded structure with mRNA, stopping its translation into proteins, thus instigating ‘RNA interference’. <nobelprize.org/nobel_prizes/medicine/laureates/2006/press.html>, 11 December 2006.
9. RNA interference involving micro-RNAs has major implications against dismissing much of our DNA as ‘junk’—see Woodmorappe, J., Junk DNA indicted, *Journal of Creation* **18**(1):28–33, 2004.
10. Roberts, J.W., RNA Polymerase, a scrunching machine, *Science* **314**(5802):1139–1143, 17 November 2006 (comment on refs 5 and 6).