Pattern of programmed cell death in bat wing membrane—support for evolution?

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What distinguishes the front foot of a mouse and the wing of a bat? What similarities and differences are there anatomically and developmentally? It might seem obvious that the contrast between such diverse structures is wholly due to their singular functional designs and purposes. A mouse uses its forepaws for weight bearing, running, climbing and to manipulate food items whereas a bat's wings are sophisticated, foldable aerofoils which facilitate powered flight and exquisite manoeuvrability. For the evolutionist, however, these are homologous structures—that is, different though they undoubtedly are, the elongated digits[1] of a bat wing correspond to the digits ('toes') of a mouse in a phylogenetic sense. Phrased colloquially, the fact that each of these mammals has five digits is said to be due to their common ancestry. But since this assertion of homology is axiomatic by nature (merely stating evolutionary belief), evolutionists are always keen to provide zoological evidence for its alleged veracity. A recent example of such an attempt—concerning the developmental control of programmed cell death in bat wing digits and interdigital tissue—has appeared in the prestigious *Proceedings of the National Academy of Sciences*.[2]

Molecular signals & apoptosis

For some years, it has been known that programmed cell death (apoptosis) occurs during limb development of certain vertebrates in order to remove the nascent interdigital tissue, thereby 'sculpting' out the digits.[3] The researchers focused their attention on the cellular fate of cells in the interdigital areas of the developing forelimbs of bat embryos (*Carollia* sp.; native to Trinidad). Cultured limbs, at different stages of development, were examined for expression of various genes and proteins known/suspected to have a role in digit elongation and/or apoptosis. Fore-limbs and hind-limbs were compared since it is only in the latter that significant apoptosis occurs, resulting in free digits.

The researchers demonstrated conclusively that, as expected, various bone morphogenetic proteins (Bmps) were expressed in the interdigital areas; these tend to induce cell death. They also detected significant expression of a potent Bmp inhibitor, *Gremlin,* in the forelimb interdigital regions. However, measurable levels of *Msx* gene (a downstream target of Bmp signalling) hinted that a role for *Gremlin* in blocking Bmp-mediated cell death (thereby promoting wing membrane development) was not the whole story. It turned out that Bmp-inhibition alone was insufficient to prevent interdigital apoptosis and the researchers were able to show that the activation of Fgf (particularly *Fgf8*) signalling cooperated with reduced Bmp signalling. Fgfs seem to be survival signals for tissues in creatures as diverse as chicks, mice and men,[4] so it was interesting that they were also found to have a role in bats in helping to prevent wing membrane apoptosis. Furthermore, by perturbing the balance of Bmp signalling and Fgf signalling experimentally, the authors of this study were able to promote extensive programmed cell death of the forelimb interdigital areas. So far, so good.

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A narrative gloss[5] on the data

But then the authors made a leap from their data by making the following statement:

> 'The development of the chiropatagium [forewing membrane] depends partly on the retention of early interdigit tissue. Our data suggest that the modulation of Bmp and Fgf signaling plays a critical role in this process and may have been involved in the evolution of the wing membrane, with one of the key events being the acquisition of *Fgf8* signaling. Our data provide a molecular insight into the evolution of powered flight in bats.'

However, I must respectfully disagree—the data suggests nothing of the sort but is merely being interpreted in this way to fit the authors' belief in evolution. Retention of interdigital

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 discusses 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'

Jonathan Sarfati

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.
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> '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