Teeth developing in bird embryos—does it prove evolution?

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> 'Birds with teeth turn the clock back 70m years … And they could help to cure baldness.'

So trumpeted *TimesOnline* (UK), 4 June 2003. But, as commonly happens, the title of the actual paper that inspired this statement was much less exciting:

> 'Development of teeth in chick embryos after mouse neural crest transplantations.'[1]

The researchers transplanted the part of a mouse embryo that normally produces teeth into the part of a chick's embryo that produces the head of the bird. They found that the mouse parts continued their development, forming the beginnings of teeth, some surviving up to 18 days.

Using staining techniques with microscopy (histochemistry), the authors demonstrated the activities of various genes considered to be involved in tooth formation, both in the mouse tissue and the surrounding chick tissue. Mouse genes known to be involved in tooth formation were active in the tooth germs (beginning teeth) and certain chick genes were active in the chick tissue surrounding the (mouse) tooth germs.

Significantly, no enamel began to develop on the teeth. The authors argued that the genes active in the chick epithelium were necessary for the mouse tooth germs to develop, but enamel formation needs specific genes present in *mouse* epithelium. This brings into question just how much the chick genes were helping in the tooth development.

One could dispute the researchers' argument for the involvement of any chick genes *specific to teeth* in the development of the teeth. One of the genes in particular, *shh,* is widely expressed during development; it is not peculiar to teeth.[2] Others, such as *BMP4* and *FGF8,* are expressed during normal chick embryo development, as the researchers stated in their paper. *BMP4, FGF8* and *Pitx2* are involved, interacting together in a broad spectrum of developmental processes other than tooth formation.[3]

The activity of chick genes close to the tooth germs may have been due to the foreign mouse cells stimulating metabolic activity in the adjacent chick cells. This has not been ruled out in the experimental design.

Note that bird chicks develop an egg tooth that is used to break the egg shell during hatching, so birds must have the genes needed to specify the development of a tooth-like appendage to the beak. So it would be unsurprising if some of these genes could assist in the development of the mouse teeth, but the chick genes studied are not specific to tooth development of any type.

Some fossil birds such as *Archaeopteryx* did have teeth. Others didn't, just as some reptiles have teeth (e.g. crocodiles); others don't (e.g. turtles). Most mammals have teeth; some don't (e.g. several anteaters and the baleen whales). In creating various kinds of animals, God could have used the same basic design, switching on/off information for making teeth. This would make sense for an intelligent designer. Engineers take this approach in designing different machines.

Natural processes could be responsible for the loss of teeth in some species derived from the original created kinds that had teeth—just as mutations created a wingless 'species' of beetle.[4] A similar principle applies to antibiotic resistance in microbes—when it is due to mutation, it involves *loss* of function, contrary to common hype. Such degenerative processes reign in the fallen world we now live in.

Baseless pop media claims

The *TimesOnLine* article claimed that 'researchers managed to reawaken a gene that has lain dormant in birds for at least 70 million years'. Actually, the researchers made no such claim in their paper. And even in the evolutionary scenario, if genes had 'lain dormant' for over 70 million years, there would not be much chance of them still being functional because mutations would have destroyed their latent functionality. Note that a gene without any *current* function for millions of years could mutate without any detriment. Therefore natural selection could not eliminate any mutants, as it could for any gene that was still operational. So a gene that was dormant would be mutated over these allegedly vast time spans, and the information would be corrupted, with no chance of being reactivated. No, the genes involved are genes that have functions in normal chick embryo development. At the most, the researchers showed that some genes that normally have other functions may assist in the expression of the tooth-forming genes in the mouse tissue.

But so what if tooth genes really had been re-awakened? If they had, it would speak *against* the assumed millions-of-years scenario, for reasons stated above. But evolutionists need to show the origin of *new* information to demonstrate the plausibility of their belief system, not that information has been lost. Mutations and natural selection result in loss of information, which can sometimes be adaptive, but this does not in any way validate the microbes-to-man scheme for the origin of all living things by natural processes, because this requires the addition of loads of new genetic information (to put feathers onto reptiles, for example).

The researchers' own summary of their work is wholly unremarkable, non-sensational:

> 'These results show that, although within a species cranial neural crest cells do not appear to be prepatterned with respect to their skeletal fates, they do contain the information to interpret generic epithelial signals and to behave in a species specific way.'

All-in-all, this is yet another case of much ado about nothing. To claim that this research shows some

awakening of bird tooth genes inactive for 70 million years of evolutionary dream time, and/or that it proves that birds had reptile ancestors, is plainly going way beyond the facts. What has been experimentally demonstrated is in no way inconsistent with the biblical account of creation.

References

1. Mitsiadis, T.A., Chéraud, Y., Sharpe, P. and Fontaine-Pérus, J., Development of teeth in chick embryos after mouse neural crest transplantations, Proc. *Nat. Acad. Science USA* **100**(11):6541–6545, 2003.
2. It is involved in many developmental processes in many different species, from insects to humans. *Shh* is implicated in left-right axis formation, and the development of limb buds, brain, eyes and lungs, and much else. See, for example, Oldak, M., Grzela, T., Lazarczyk, M., Malejczyk, J. and Skopinski, P., Clinical aspects of disrupted Hedgehog signaling (Review), *Int J. Mol Med* **8**(4):445–52, 2001.
3. For example, Lu, M.F., Pressman, C., Dyer, R., Johnson, R.L. and Martin, J.F., Function of Rieger syndrome gene in left-right asymmetry and craniofacial development, *Nature* **401**(6750):276–278, 1999.
4. Wieland, C., Beetle bloopers—even a defect can be an advantage sometimes, *Creation* **19**(3):30, 1997.