The adaptation of bacteria to feeding on nylon waste

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In 1975, Japanese scientists discovered bacteria that could live on the waste products of nylon manufacture as their only source of carbon and nitrogen.[1] Two species, *Flavobacterium* sp. K172 and *Pseudomonas* sp. NK87, were identified that degrade nylon compounds.

Much research has flowed from this discovery to elucidate the mechanism for the apparently novel ability of these bacteria.[2] Three enzymes are involved in *Flavobacterium* K172: F-EI, F-EII and F-EIII, and two in *Pseudomonas* NK87: P-EI and P-EII. None of these have been found to have any catalytic activity towards naturally occurring amide compounds, suggesting that the enzymes are completely new, not just modified existing enzymes. Indeed no homology has been found with known enzymes. The genes for these enzymes are located on plasmids:[3] plasmid pOAD2 in *Flavobacterium* and on two plasmids, pNAD2 and pNAD6, in *Pseudomonas*.

Apologists for materialism latched onto these findings as an example of evolution of new information by random mutations and natural selection, for example, Thwaites in 1985.[4] Thwaites' claims have been repeated by many, without updating or critical evaluation, since.

Is the evidence consistent with random mutations generating the new genes?

Thwaites claimed that the new enzyme arose through a frame shift mutation. He based this on a research paper published the previous year where this was suggested.[5] If this were the case, the production of an enzyme would indeed be a fortuitous result, attributable to 'pure chance'. However, there are good reasons to doubt the claim that this is an example of random mutations and natural selection generating new enzymes, quite aside from the extreme improbability of such coming about by chance.[6]

Evidence against the evolutionary explanation includes:

1. There are five transposable elements on the pOAD2 plasmid. When activated, transposase enzymes coded therein cause genetic recombination. Externally imposed stress such as high temperature, exposure to a poison, or starvation can activate transposases. The presence of the transposases in such numbers on the

plasmid suggests that the plasmid is designed to adapt when the bacterium is under stress.

2. All five transposable elements are identical, with 764 base pairs (bp) each. This comprises over eight percent of the plasmid. How could random mutations produce three new catalytic/degradative genes (coding for EI, EII and EIII) without at least some changes being made to the transposable elements? Negoro speculated that the transposable elements must have been a 'late addition' to the plasmids to not have changed. But there is no evidence for this, other than the circular reasoning that supposedly random mutations generated the three enzymes and so they would have changed the transposase genes if they had been in the plasmid all along. Furthermore, the adaptation to nylon digestion does not take very long (see point 5 below), so the addition of the transposable elements afterwards cannot be seriously entertained.
3. All three types of nylon degrading genes appear on plasmids and only on plasmids. None appear on the main bacterial chromosomes of either *Flavobacterium* or *Pseudomonas*. This does not look like some random origin of these genes—the chance of this happening is low. If the genome of *Flavobacterium* is about two million bp,[7] and the pOAD2 plasmid comprises 45,519 bp, and if there were say 5 pOAD2 plasmids per cell (~10% of the total chromosomal DNA), then the chance of getting all three of the genes on the pOAD2 plasmid would be about 0.0015. If we add the probability of the nylon degrading genes of *Pseudomonas* also only being on plasmids, the probability falls to 2.3×10^{-6}. If the enzymes developed in the independent laboratory-controlled adaptation experiments (see point 5, below) also resulted in enzyme activity on plasmids (almost certainly, but not yet determined), then attributing the development of the adaptive enzymes purely to chance mutations becomes even more implausible.
4. The antisense DNA strand of the four nylon genes investigated in *Flavobacterium* and *Pseudomonas* lacks any stop codons.[8] This is most remarkable in a total of 1,535 bases. The probability of this happening by chance in all four antisense sequences is about 1 in 10^{12}. Furthermore, the EIII gene in *Pseudomonas* is clearly not phylogenetically related to the EII genes of *Flavobacterium*, so the lack of stop codons in the antisense strands of all genes cannot be due to any commonality in the genes themselves (or in their ancestry). Also, the wild-type pOAD2 plasmid is not necessary for the normal growth of *Flavobacterium*, so functionality in the wild-type parent DNA sequences would appear not to be a factor in keeping the reading frames open in the genes themselves, let alone the antisense strands.

Some statements by Yomo *et al*., express their consternation:

'These results imply that there may be some unknown mechanism behind the evolution of these genes for nylon oligomer-degrading enzymes.

‘The presence of a long NSF (non-stop frame) in the antisense strand seems to be a rare case, but it may be due to the unusual characteristics of the genes or plasmids for nylon oligomer degradation.

‘Accordingly, the actual existence of these NSFs leads us to speculate that some special mechanism exists in the regions of these genes.’

It looks like recombination of codons (base pair triplets), not single base pairs, has occurred between the start and stop codons for each sequence. This would be about the simplest way that the antisense strand could be protected from stop codon generation. The mechanism for such a recombination is unknown, but it is highly likely that the transposase genes are involved.

Interestingly, Yomo *et al*. also show that it is highly unlikely that any of these genes arose through a frame shift mutation, because such mutations (forward or reverse) would have generated lots of stop codons. This nullifies the claim of Thwaites that a functional gene arose from a purely random process (an accident).

5. The Japanese researchers demonstrated that nylon degrading ability can be obtained *de novo* in laboratory cultures of *Pseudomonas aeruginosa* [strain] POA, which initially had no enzymes capable of degrading nylon oligomers.[9] This was achieved in a mere nine days! The rapidity of this adaptation suggests a special mechanism for such adaptation, not something as haphazard as random mutations and selection.
6. The researchers have not been able to ascertain any putative ancestral gene to the nylon-degrading genes. They represent a new gene family. This seems to rule out gene duplications as a source of the raw material for the new genes.[8]

P. aeruginosa is renowned for its ability to adapt to unusual food sources—such as toluene, naphthalene, camphor, salicylates and alkanes. These abilities reside on plasmids known as TOL, NAH, CAM, SAL and OCT respectively.[2] Significantly, they do not reside on the chromosome (many examples of antibiotic resistance also reside on plasmids).

The chromosome of *P. aeruginosa* has 6.3 million base pairs, which makes it one of the largest bacterial genomes sequenced. Being a large genome means that only a relatively low mutation rate can be tolerated within the actual chromosome, otherwise error catastrophe would result. There is no way that normal mutations in the chromosome could generate a new enzyme in nine days and hypermutation of the chromosome itself would result in non-viable bacteria. Plasmids seem to be adaptive elements designed to make bacteria capable of adaptation to new situations while maintaining the integrity of the main chromosome.

Stasis in bacteria

P. aeruginosa was first named by Schroeter in 1872.[10] It still has the same features that identify it as such. So, in spite of being so ubiquitous, so prolific and so rapidly adaptable, this bacterium has not evolved into a different type of bacterium. Note that the number of bacterial generations possible in over 130 years is huge—equivalent to tens of millions of years of human generations, encompassing the origin of the putative common ancestor of ape and man, according to the evolutionary story, indeed perhaps even all primates. And yet the bacterium shows no evidence of directional change—stasis rules, not progressive evolution. This alone should cast doubt on the evolutionary paradigm. *Flavobacterium* was first named in 1889 and it likewise still has the same characteristics as originally described.

It seems clear that plasmids are designed features of bacteria that enable adaptation to new food sources or the degradation of toxins. The details of just how they do this remains to be elucidated. The results so far clearly suggest that these adaptations did not come about by chance mutations, but by some designed mechanism. This mechanism might be analogous to the way that vertebrates rapidly generate novel effective antibodies with hypermutation in B-cell maturation, which does not lend credibility to the grand scheme of neo-Darwinian evolution.[11] Further research will, I expect, show that there is a sophisticated, irreducibly complex, molecular system involved in plasmid-based adaptation—the evidence strongly suggests that such a system exists. This system will once again, as the black box becomes illuminated, speak of intelligent creation, not chance. Understanding this adaptation system could well lead to a breakthrough in disease control, because specific inhibitors of the adaptation machinery could protect antibiotics from the development of plasmid-based resistance in the target pathogenic microbes.

Recommended Resources

References

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