

Yet another way of getting more from less

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The Human Genome Project turned up some 30,000 supposed protein-coding genes.¹ But protein studies had already suggested that we humans can produce over 100,000 different proteins. As Harrison *et al.*, said, '... the human proteome size is likely to be significantly larger than approximately 90,000.'² So where do the extra proteins come from?

Known sources of extra variety include:

1. DNA editing. This occurs with the production of antibodies in the immune system. Two mechanisms appear to operate; one involving recombination and the other a targeted hypermutation of the DNA coding for antibodies.^{3,4} This incompletely understood mechanism allows cells in the lymphatic system to quickly generate new antibodies to deal with pathogens not encountered before. Some apologists for the evolutionary philosophy have claimed this as an example of information arising from a random process, but it is irrelevant to biological evolution.⁵
2. Messenger RNA (mRNA) editing. The exon / intron arrangement of genes in eukaryotes allows for rearrangement of the exons to produce different proteins. For example, the cSlo gene produces at least 576 different proteins in the hair cells of the inner ear of chickens.⁶ These variant proteins are involved in facilitating hearing by the tuning of the hair cells to different frequencies. Such high levels of alternative splicing are apparently common in nerve cells.⁴ Researchers do not know yet how cells regulate such mRNA editing to generate different proteins. If each of the 30,000 putative genes

produced only four different proteins by mRNA editing, this would increase the number of possible proteins to 120,000.

Now researchers have found, in humans, a third means of getting more peptides: protein editing.⁷ Already known to operate in plants and unicellular organisms, it also occurs in the production of peptides that label aberrant cells for destruction by killer T-cells in humans. In a mind-boggling sequence of events, many details of which still remain obscure, cells recognize foreign (e.g. bacterial) proteins or wayward self-proteins, such as the excess production of a certain protein in a cancerous cell, and mark them for destruction. The proteasome⁸ degrades the marked proteins into short pieces (oligopeptides). Then a special antigen-processing transport system moves the pieces to the endoplasmic reticulum where some of the peptides bind to MHC (Major Histocompatibility Complex) class I molecules. These complexes are then conveyed to the cell surface, where the MHC molecules present the peptides, stimulating killer T-cells to destroy the cell.

The researchers found that the peptides presented by the MHC came from a consistent joining together of the two ends of the aberrant protein—protein editing. This is the first time this has been shown to occur in vertebrates.

The astonishing complexity of living things never ceases to amaze me. The protein editing system adds another dimension of complexity to the immune system of vertebrates. The killer T-cell system appears to be a good example of an irreducibly complex system. Because the system has to be complete to contribute to fitness, a step-wise process of small lucky mutations selected by natural selection, per Dawkins, cannot work. Attributing such incredibly integrated complex systems to chance, the only alternative to design, defies basic logic.

References

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Insurmountable problem

'You can collect lists of conserved genes, but once you get those lists, it's very hard to get at the mechanisms [of evolution] ...'

'Macroevolution is really at a dead end.'

Jeffery, W.,
Quoted in, Pennisi, E.,
Evo-devo enthusiasts get down to detail,
Science 298(5595):7–9
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