If junk DNA is useful, why is it not shared more equally?

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The presence of introns in genes requires cells to process "messenger RNA" molecules before synthesizing proteins, a process that is costly and often error-prone. It was long believed that this was simply part of the price organisms paid for the flexibility to create new types of protein but recent work has made it clear that introns themselves have a number of important functions. And so attention is gradually shifting to asking why some organisms have so few introns and others so many.

It seems likely that new introns are added to DNA when double-stranded DNA breaks – which may arise from a variety of mechanisms – are not repaired "correctly" but the newly created ends are instead joined to other fragments of DNA. Farlow and colleagues at the Institute of Population Genetics of the University of Veterinary Medicine, Vienna reasoned that introns may be lost by a similar mechanism. An examination of areas of DNA where introns are known to have been lost in organisms such as worms and flies provides support for their idea.

DNA breaks may be treated in one of two ways: correct repair (by a relatively time-consuming process known as "homologous recombination") or the rapid and error-prone joining of non-homologous ends. The two pathways are essentially separate and can compete with each other for DNA breaks to work with. The scientists at the University of Veterinary Medicine, Vienna now suggest that species-specific differences in the relative activity of these two pathways might underlie the observed variation in intron number.

The theory represents a fundamental change in the way we think about the evolution of DNA. Evolution has seen periods of large scale intron loss alternating with periods of intron gain and this has been interpreted as the result of changing selection pressure. However, the rates at which single species have gained and lost introns throughout evolution have been found to vary in parallel, consistent with Farlow's notion that the two processes are related. The new theory provides an alternative interpretation: changes in the activities of the "homologous" and "non-homologous" pathways for repairing DNA breaks could cause introns to be lost faster than they are gained, or vice versa.

The idea is consistent with what we currently know about intron numbers, which range from a handful in some simple eukaryotes to more than 180,000 in the human genome. And as Farlow says, "Linking intron gain and loss to the repair of DNA breaks offers a neat explanation for how intron number can change over time. This theory may account for the huge diversity we seen in intron number between different species."

More information: The paper DNA double-strand break repair and the evolution of intron density by Ashley Farlow, Eshwar Meduri and Christian Schlötterer is published in the January issue of the journal *Trends in Genetics* (2011, Vol. 27, pp. 1-6). www.cell.com/trends/genetics/fulltext/S0168-9525%2810%2900210-6

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