

Re: Article: Unravelling new complexity in the genome

Source: <http://sci.tech-archive.net/Archive/sci.bio.evolution/2007-08/msg00092.html>

- *From:* "John W Edser" <edser@xxxxxxxxxxxxxxxx>
 - *Date:* Wed, 15 Aug 2007 16:59:15 -0400 (EDT)
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"Robert Karl Stonjek" <rstonjek@xxxxxxxxxxxxxxxx> wrote:–

A major surprise emerging from genome sequencing projects is that humans have a comparable number of protein-coding genes as significantly less complex organisms such as the minute nematode worm *Caenorhabditis elegans*. Clearly something other than gene count is behind the genetic differences between simpler and more complex life forms.

JE:–

I identify two "other than gene count " possibilities:–

1) CORRECTING non additive gene interactions from its present status as "inherited" but not "heritable" to "inherited" and "heritable".

The

pioneering work of C. H. Waddington demonstrated over 50 years ago that non additive gene associations remain heritable. His synthetic theory (I posted Waddington's synthetic model twice to sbe) incorporated this principle was based on two basic concepts:

a) Canalisation: a non additive incredibly higher rate of heritable, phenotypic conservation.

b) Assimilation: a non additive amazingly rapid rate of heritable phenotypic change.

Both remain ignored within today's synthetic models.

The high gearing of genetic epistasis provides the best of both words for nature: higher rates of phenotypic conservation when things stay the same and lightning fast rates of phenotypic change when things change making the traditional model look like a model T ford. The problem is non additive epistasis remains deleted from today's, oversimplified, uncorrected, synthetic theory only because it was inconvenient for the mathematics.

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2) Actually incorporating and not just ignoring gene imprinting within synthetic theory as a powerful above the gene (epigenetic) heritable mechanism which can act as a fine tuner of the DNA/RNA system allowing a parents adaptation to be passed on to offspring as an epistatic set of genes in which some are inherited turned on and others, turned off.

Increased functional and cellular complexity can be explained, in large part, by how genes and the products of genes are regulated.

JE:–

Yes, where most regulation of one gene by another remains non additive and therefore deleted within popular, uncorrected, oversimplified Neo Darwinian models.

A University of Toronto–led study published in the latest issue of *Genome Biology* reveals that a step in gene expression (referred to as alternative splicing) is more highly regulated in a cell and tissue–specific manner than previously appreciated and much of this additional regulation occurs in the nervous system. The alternative splicing step allows a single gene to specify multiple protein products by processing the RNA transcripts made from genes (which are translated to make protein).

JE:–

Such a massive non additive effect remains deleted from Neo Darwinian models.

"We are finding that a significant number of genes operating in the same biological processes and pathways are regulated by alternative splicing differently in nervous system tissues compared to other mammalian tissues," says lead investigator Professor Benjamin Blencowe of the Banting and Best Department of Medical Research and Centre for Cellular and Biomolecular Research (CCBR) at the University of Toronto

According to Blencowe, it is particularly interesting that many of the genes have important and specific functions in the nervous system, including roles associated with memory and learning. However, in most cases the

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investigators working on these genes were not aware that their favorite genes are regulated at the level of splicing. Blencowe believes that the data his group has generated provides a valuable basis for understanding molecular mechanisms by which genes can function differently in different parts of the body.

JE:–

Leaving the gene centric theory dead in the water.

Blencowe attributes these new findings in part to the power of a new tool that he, together with his colleagues including Profs. Brendan Frey (Department of Electrical and Computer Engineering) and Timothy Hughes (Banting and Best, CCBR), developed a few years ago. This tool, which comprises tailored designed microarrays or "gene chips" and computer algorithms, allows the simultaneous measurement of thousands of alternative splicing events in cells and tissues. "Until recently researchers studied splicing regulation on a gene by gene basis. Now we can obtain a picture of what is happening on a global scale, which provides a fascinating new perspective on how genes are regulated," Blencowe explains.

JE:

Suddenly the mechanism of non additive genetic epistasis has become observable. The heritability of genetic epistasis is just the heritability of basic pattern no matter if some genes become added or deleted.

snip for brevity<

Thanks Robert,

Regards,

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