Australian Academy of Science

RUDI LEMBERG

LECTURE

Genes in Organelles: Mitochondria, Ageing, and Sex-Energy versus Fidelity

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Old Geology Building (A11)
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The primary function of mitochondria and chloroplasts is energy transduction in respiration and photosynthesis. The physico-chemical mechanisms of bioenergetics do not directly involve genes and heredity, and furthermore, redox chemistry is intrinsically mutagenic. Therefore there is a cost to having genetic systems in organelles. We propose that the benefit, which alone justifies the cost, is that expression of mitochondria and chloroplast genes is regulated directly by the function of gene products. The predicted redox regulation has been demonstrated for chloroplasts, and there is progress in identifying the mechanism by which it occurs. We propose that ageing arises from redox chemistry in mitochondria in somatic cells and male gametes, while passive, ”template” mitochondria are sequestered in female germ-lines, allowing an indefinite number of accurate mitochondria replication without damage the mitochondria, their genome, or to the cells that carry them [1]. Respiratory electron transport requires a mitochondrial genetic system [2]. However, “incorrect” electron transfers produce free radicals that cause mutation, and their frequency is itself increased by mutation. The energetic function of mitochondria is therefore

detrimental to the fidelity of their replication. Damage to somatic mitochondria DNA may accumulate within, and determine, the life-span of individual organisms. Motility of one gamete is required for fertilisation, and requires ATP. It is proposed that female gametes, which are non-motile, repress mitochondrial oxidative phosphorylation, thus protecting mitochondrial DNA for faithful transmission between generations. Male gametes, in contrast, maximise energy production for motility by sacrificing mitochondrial DNA to electron transfer and its mutagenic by-products. Male gametes then make no contribution to the mitochondrial genome of the zygote: mitochondria are maternally inherited. This testable hypothesis may explain the prevalence of anisogametic sex as a device to allow an indefinite number of accurate replications of template mitochondria, from which all somatic mitochondria derive. If the hypothesis is correct, we predict that senescence will be inherited by the progeny of reproductive cloning from somatic cells [3], and that senescence will be promoted, not postponed, in allotropic mutants where genes for respiratory-chain proteins are removed from mitochondria to the cell nucleus.

[1] Allen, JF (1996) Separate sexes and the mitochondrial theory of ageing. *J. Theor. Biol*. 180, 135-140.
[2] Allen, JF; Puthiyaveetil, S; Stroem, J and Allen, CA (2005) Energy transduction anchors genes in organelles. *BioEssays* 27, 426-435.
[3] Allen, JF and Allen,CA (1999) A mitochondrial model for premature ageing of somatically cloned mammals. *IUBMB Life* 48, 369-372.

Everyones are welcome.

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