guanine have half lives of about a year, uracil about 12 years, and cytosine only 19 days.[8] Intense heating also readily destroys many of the complex amino acids such as serine and threonine.[9] Another problem is that the exclusive 'left-handedness' required for life is destroyed by heating, i.e. the amino acids are *racemized*.[10] But this was not put to the test because the Japanese team used the simplest amino acid, glycine, which is the only achiral amino acid used in living systems. It seems incomprehensible that after designing this experiment with such care other amino acids would not have been tested. The fact that they are all known to undergo various non-peptide bond reactions has surely not escaped the researchers' attention.

3. The longest polymer (or rather, oligomer) formed was hexa-glycine. Most enzymes, however, have far more than six amino acid residues — usually hundreds. And even the hexaglycine produced was found only in minuscule amounts.
4. This experiment gave a simple homo-oligomer, i.e. all monomers are the same. But life requires many polymers in *precise sequences* of 20 *different* types of amino acids. Thus Matsuno's experiments offer not the slightest explanation for the complex, high-information polymers of living organisms.

Conclusion

As the non-creationist information theorist Hubert Yockey observed over 20 years earlier (and he has not revised his opinion since):

'Research on the origin of life seems to be unique in that the conclusion has already been authoritatively accepted .... What remains to be done is to find the scenarios which describe the detailed mechanisms and processes by which this happened.

One must conclude that, contrary to the established and current wisdom a scenario describing the genesis of life on earth by chance and natural causes which can be accepted on the basis of fact and not faith has not yet been written.'[11]

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Genomic imprinting

Pierre Jerlström

Virtually everyone has heard of Dolly the sheep, cloned from DNA isolated from mammary gland cells.[12] Cloning (or making a genetically identical copy of an organism) by replacing the DNA of an egg cell with non germ-cell DNA, is promising to become a very lucrative business for the generation of improved domestic animal strains. But difficulties in the cloning of other mammals such as cows, mice, goats and monkeys shows that Dolly's easy success may have been somewhat of a fluke.[3] Also, as now discovered with Dolly, successful clones may have short life-spans due to the inheritance of 'pre-aged' genes from 'old' parent cells.[4]

Currently, the process of cloning is a health risk, often proving lethal to the pregnant mothers and to the clones themselves — on the way to a successful clone there are lots of placental and embryonic defects resulting in death of the foetuses or death of the animal shortly after birth.[5] But why does this occur? Scientists are actively trying to unravel this problem, and have recently become aware of the importance of **genomic imprinting.**

Offspring normally have two copies of virtually all their genes, one complement from each parent. But in many sets, one of the genes carries a biochemical mark that keeps it switched off. This mark is established during the development of egg and sperm cells to distinguish between the maternal and the paternal copies. The imprinted mark is maintained through embryo development but is erased in the gonads (testicles and ovaries) to allow fresh imprinting for the next generation of offspring.[6]

So how does imprinting occur? Researchers are still uncertain of the hows and whys. But the biochemical process of **methylation** appears to be important in imprinting, since all imprinted genes have DNA sequences that are methylated (called differentially methylated regions or DMRs; see

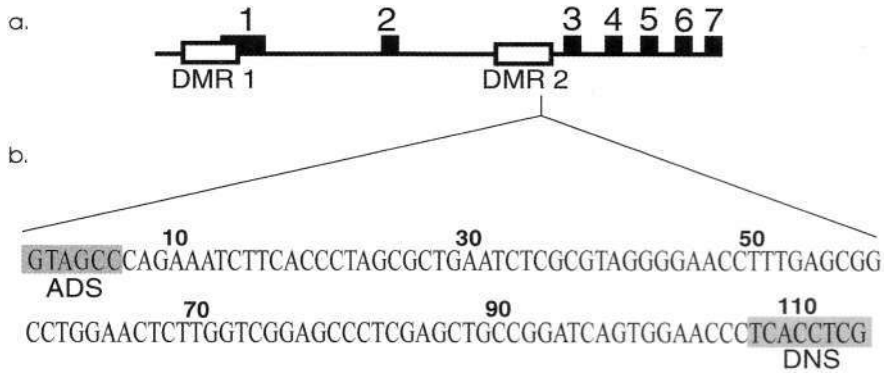

Figure 1. Methylation of the mouse Igf2r gene, encoding the receptor for insulin-like growth factor type-2. a) Diagrammatic representation of the Igf2r gene, showing two methylated regions (DMR1 and DMR2), designated by open boxes. Black boxes designate exons. b) Nucleotide sequence of imprinting box within DMR2. Sequences for allele-discrimination signal, ADS, and de novo *methylation signal, DNS, are highlighted. The ADS prevents DMNR2 methylation in the paternal copy of the Igf2r gene (after Birger* et al..*).*[8]

Figure l),[7] and mutated mice that have lost the ability to methylate DNA, or have DNA with deleted DMRs, lose imprinting.[8]

Methylation is carried out by specific transmethylase enzymes that recognise the sequences surrounding the nucleotide bases adenine or cytosine on which they act (Figure 2).[9] By binding certain proteins, methyl groups aid in folding the DNA into tight coils, thereby blocking transcription or the copying of DNA into RNA and thus protein expression from the genes.[10]

So scientists have now become aware that our simplistic 20th century belief that all life is just a result of genes is an oversimplification, and that the results of the inheritance of additional factors such as methylation need to be incorporated in the definition of genes.[10]

It is clear that for the successful development of an embryo, a balance between the genes of *both* parents is required — some cloning experiments mimic experiments where imprinting has been disrupted, because the embryo's genes come from a single parent. Scientists are therefore recognising that imprinting *'is nature's way of ensuring that every baby has two parents.'*[3] This is further evidence for the completeness of God's design at the molecular level in mammals and specifically in man, and his safeguard for the family unit.

As man pursues his futile quest for immortality without a Creator, the goal of human cloning may soon be a reality. The fact that cloning experiments result in such a tremendous number of casualties and deformities is disturbing and human cloning is clearly against God's will, as God regards man as a living soul even before he is born:

> *'... the Lord hath called me from the womb; from my mother hath he made mention of my name.' (Isaiah 49:1)*
>
> *'Before I formed thee in the belly I knew thee; and before thou earnest forth out of the womb I sanctified thee* and / *ordained thee ....' (Jeremiah 1:5)*

Imprinting is also responsible for the unsuccessful mating of two different mouse species, *Peromyscus polionotus* and *P. maniculatus.'* [11] In this experiment the researchers found that both parental copies of some imprinted genes were active, resulting in malformed foetuses. In addition, which imprinted gene was affected depended on which species contributed the egg and the sperm. Offspring with P. *maniculatis* as the mother produced offspring 40% of the normal size and with low expectation of survival in the wild, but when this species acted as the father the foetuses were oversized and died.[12]

The researchers concluded that

> *' "imprinting recognition proteins " in the egg of one species can't read all the imprinting marks on the DNA of another. ... This inability of one species to recognise the imprinting of another appears to create a strong reproductive barrier between them'.'*[11]

The inability of species to hybridise and produce fertile offspring has been interpreted as a possible contribution to the evolution of mammals, *'... since reproductive isolation is one characteristic of species that have diverged'.* [11] However, there is no need for evolutionary explanation to account for speciation:

> *'Loss of information through mutations, natural selection and genetic drift [random sampling of information in small isolated populations] can sometimes result in different small populations losing such different information that they will no longer interbreed. For example, changes in song or colour might result in birds no longer recognizing a mate, so they no longer interbreed. Thus a new "species " is formed.'*[13]

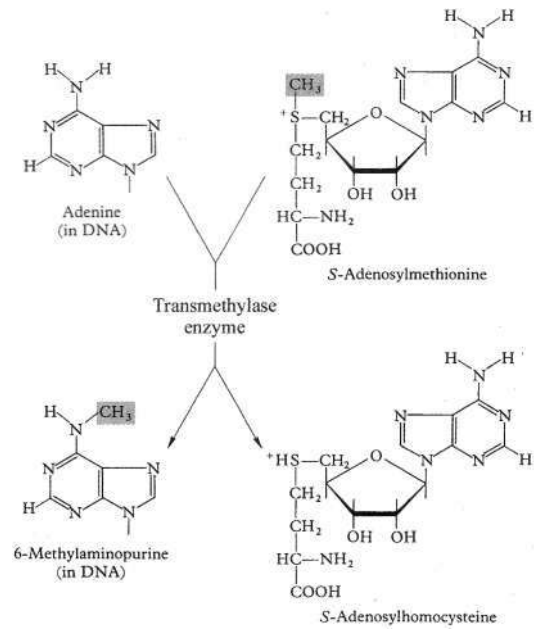

Figure 2. *Transmethylation of adenine into 6-methylaminopurine; the transferred methyl group is highlighted (from Stent and Calender).*[9]

Information-losing mutations in genes coding for such imprinting recognition proteins may be a better mechanism to help explain rapid speciation after the Flood within a Genesis creation model (e.g. dog kind giving rise to coyotes, wolves, etc.).

It is important to note that speciation occurs via the sorting out or loss of pre-existing genetic information, and not the particle-to-people evolution proposed by evolutionists, which requires the generation of *new* information.

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Very rapid emplacement of Columbia River basalts in non-turbulent flow

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Flood basalts, sometimes extending over 100,000 km^2 and a few kilometres thick, are found in many areas of the world.[1] There are no modern analogs for such continental flood basalts, and the origin and emplacement of the lava is poorly understood.[2] The most studied flood basalt is the Columbia River Basalt Group (CRBG) in eastern Washington, northern Oregon, and western Idaho, USA. The CRBG is composed of about 300 remarkably homogeneous flows, a few as large as 2000 km^3. Although many believe that most of the basalts erupted over 2.5 million years, a number of researchers found evidence for rapid emplacement of each flow — of the order of days to a week or two.[3]

Recently, some scientists have attempted to slow this emplacement time to months or years.[4] For instance, Thordarson and Self,[5] and Self *et al*;[6] claim that the 1300 km^3 Roza Flow of the CRBG would have been emplaced in 5 to 15 years based mainly on finding what they believe are pahoehoe (ropy) lava lobes at the base and top of many CRBG flows. However, Anita Ho and Katharine Cashman challenge this claim by quantitative evidence for very rapid emplacement.[7]

Ho and Cashman used a 'geothermometer', based on the MgO content of volcanic glass, to measure the cooling of the 1600 km^3 Ginkgo Flow of the CRBG along its 500 km flow path. The flow cooled only 10 to 20 °C over 500 km — a rate of only 0.02 to 0.04 °C/km! This compares to a cooling rate of 1 to 4.5 °C/km measured on Hawaiian áá [rough, jagged] flows and 0.6 to 1.0 °C/km observed in active Kilauea lava tubes.[8] The extremely low cooling rate of the Ginkgo Flow suggests two possibilities: 1) the flow was extraordinarily rapid, or 2) transport was extremely thermally efficient. Ho and Cashman choose the latter because of the great thickness of the flow. Both could be correct.[9]

Ho and Cashman also calculated a range of flow viscosities of the Ginkgo Flow based on the observed crystallinity of 10-20% and the slight temperature change with distance. From these calculations, they deduce that flow must have been laminar, otherwise turbulence would have caused a much greater heat loss due to a higher exposed surface area. In laminar flow, the calculated viscosities resulted in a flow velocity of 1-8 m/s, which represents a total emplacement time of 18 hours to 6 days for the Ginkgo Flow. However, these estimates are based on their highest calculated viscosity, which is three times their lowest estimate. There are also other factors that would allow a higher flow velocity, such as the presence of bubbles. So flow velocity could be significantly faster than 8 m/s. If the Ginkgo flow was extruded in 1 day and this rate continued, the whole CRBG could be emplaced in as little as 100 days.

Ho and Cashman, unable to shake their uniformitarian bias, suggest that emplacement could have been either by fast laminar flow under an insulating crust or by a slower, inflating flow. The latter is similar to flows observed on Hawaii but seems incongruous with the quantitative data presented. Laminar flow under an insulating crust requires a crust that rapidly cools, but how can a 30-70 m thick lava creep slowly enough for an insulating crust to form, when the evidence indicates rapid flow? It seems more likely that emplacement was very rapid and non-turbulent.

There are still a number of mysteries associated with flood basalts. In a catastrophic flood