Bacterial genome decay from a baraminological viewpoint

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Bacterial genome decay (BGD) is common across bacteria, many aspects of which support the biblical creation/Fall model. The phenomenon can be traced through species comprising different baramins, with a common ancestor only a few thousand years old. Pseudogenization, genomic deletions, and the spread of insertion elements, chromosomal rearrangements, and genome downsizing are common, which are the opposite of what would supposedly happen during evolution.

We present a survey of the scientific literature describing BGD in the *Bordetella*, *Mycobacterium*, and *Yersinia* baramins plus their statistical genomic characterization. These baramins are contrasted with bacteria with highly reduced genomes and organelles. We also examine the consequences of BGD for the minimal organism question and the endosymbiotic theory. Based on a minimum gene estimate for the last universal common ancestor (LUCA) of 1,340, the probability for the evolution of the first non-parasitic cellular organism is approximately $10^{-167,500}$. Gene density, gene loss and the differences in genetic code separate organelles from bacteria with reduced genomes.

In conclusion, the study of BGD can be helpful in statistically characterizing baramins. It can delineate at what stage individual species are in this process within baramins. Genome size, gene number, and GC% can also help delineate individual baramins from one another.

Studying the decay of bacterial genomes is an interesting part of the creation/evolution debate. Certain aspects of bacterial genomes highlight natural processes that are more in line with the creation/devolution model compared to the evolutionary model of gradual upwards progression. The very fact that the scientific literature speaks about the genome decay in bacterial genomes strikes a massive blow to gradual upwards evolutionism and supports biblical creation, which states that genome decay is the result of the Curse.

First of all, bacterial genuses (such as *Mycobacterium* or *Bordetella*) correspond to a large degree to the biblical baramins, or created kinds. Secondly, these taxonomical groups all have a common ancestor dating back only a few thousand years, which fits quite well within the biblical timescale. Thirdly, these bacterial genuses also have undergone genetic bottlenecks in the recent past, along with other species, such as humans, during the Genesis Flood. Lastly, their genomes have undergone a process of decay, involving processes such as pseudogenization, deletions, spread of insertion elements, chromosomal rearrangements, and genome downsizing.[1]

For example, Wood[2] describes genome decay in the genomes of two *Mycoplasma* and *Ureaplasma* species. The 475 genes of *M. genitalium* are contained in the genome of *M. pneumonia*, with only a slight difference in gene order. However, only 53% of the genes in *Ureaplasma urealyticum* are found in either of the previous species. As genome decay progressed, these bacteria lost their genes and became more dependent on their hosts for survival (*M. genitalium* and *U. urealytica* are both found in the urinary tract of humans).

It is interesting to note that in these genuses there are species, the genomes of which are in different stages of decay. This work describes these processes in a number of bacterial genuses, such as *Mycobacterium*, *Bordetella*, and *Yersinia*. Some of the species in these genuses cause illnesses such as leprosy, tuberculosis, Buruli ulcer, whooping cough, and the black plague. Related genetic phenomena, such as genetic entropy and the minimal genome, are also discussed from this aspect.

The *Bordetella* baramin

A study by Parkhill *et al*.[3] examined three species of the *Bordetella* baramin: *B. bronchiseptica*, *B. pertussis*, and *B. parapertussis*, all of which colonize the respiratory tracts of animal species. *B. parapertussis* infects both sheep and humans, causing whooping cough in humans. Both species of bacteria have descended from the strains of the species *B. bronchiseptica*, which colonizes a broad range of hosts. During this process they underwent genome downsizing and changes in their virulence factors as well as changes in genes coding for enzymes in metabolic pathways. These species are thought to have originated from a common ancestor only a few thousand years ago due to lack of allelic polymorphisms.[4]

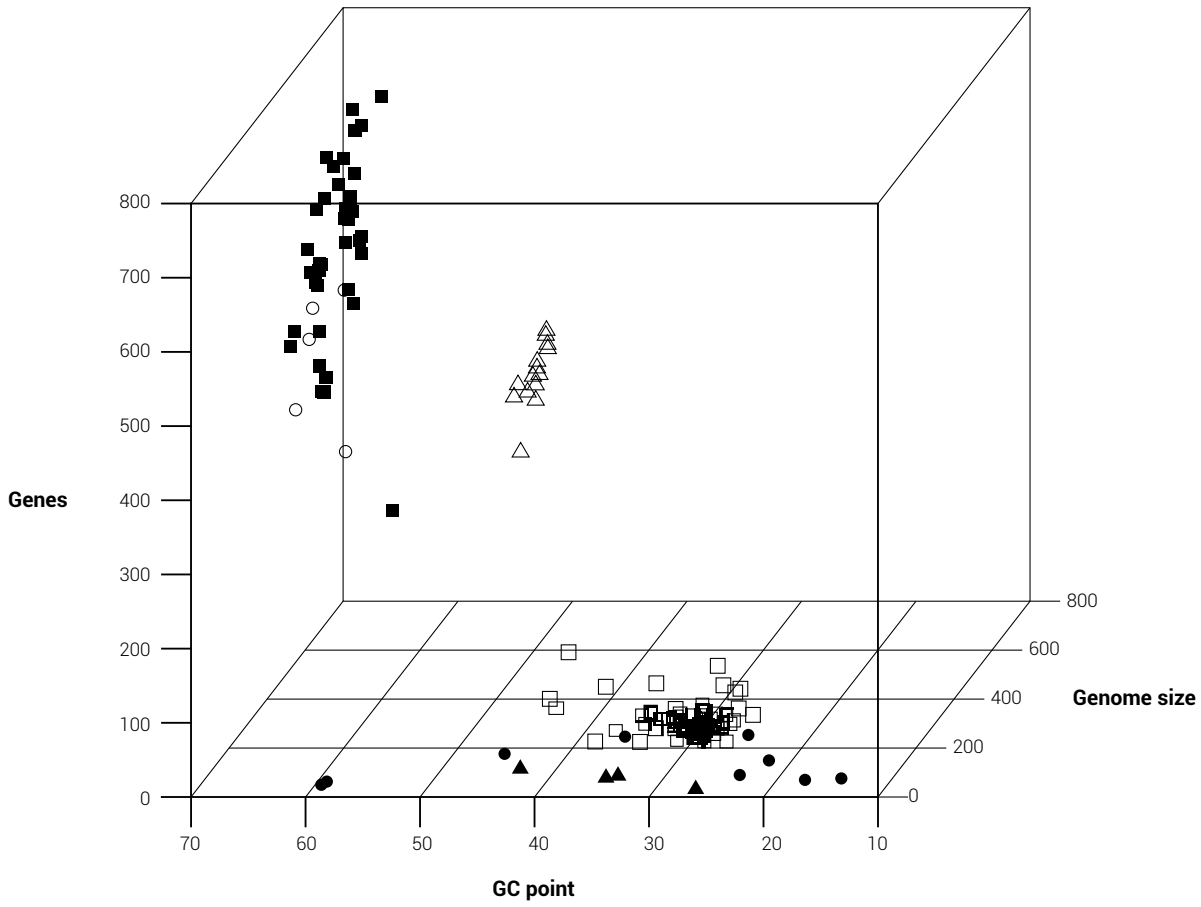

Figure 1. The number of genes as a function of genome size and GC%. Black square: *Mycobacterium* (40 data points), White circle: *Bordetella* (5 data points), White triangle: *Yersinia* (16 data points), White square: *Mycoplasma* (67 data points), Black circle: bacteria with reduced genomes (11 data points), Black triangle: organelles (4 data points).

In a study of 132 strains from these three species covering 32 different sequence types (ST), Diavatopoulos *et al*.[5] observed that *pertussis* and *parapertussis* have only appeared recently, implying that they descended from strains of *B. bronchiseptica*. From a bird's eye view, the genome sizes have decreased from the direction of *bronchiseptica* to *pertussis* and *parapertussis*. The number of coding sequences has decreased, whereas the number of pseudogenes has increased, a hallmark of genome decay. For exact numbers, see table 1,[6] where data is also given for the other groups. The genome sizes and number of genes for this as well as the other two baramins can be seen in figure 1. Here the *Bordetellae* baramin clearly separates from the other groups. Members of the *Mycobacterium* and *Bordetellae* baramin are intermingled with each other, but can be expected to separate from each other if more characteristics are taken into account besides such general ones like genome size or GC%. The correlation between the genome size and the number of genes in the *Bordetellae* baramin is 0.99.

Six hundred and two of the genes in *bronchiseptica* are unique to that species only. Besides this, there are several prophage sequences within the genome of *bronchiseptica* which were independently deleted—in spite of random chance—in the genomes of *pertussis* and *parapertussis*. This is evident because the edges of these prophage sequences are different in both species. The much smaller genome of *pertussis* is due to the widespread insertions of insertion sequence elements (ISE) (238 of them) into different parts of the *pertussis* genome, causing deletion and genomic rearrangements.

Many of the genes lost in the *pertussis* and *parapertussis* genomes are involved in utilization of alternative nutrient sources, such as autotransporter genes, some of which are involved in iron uptake, for example. Other genes include virulence factors, such as a type-IV pilus biosynthesis operon that is lacking from the other two genomes. Others include flagella, which are present in *bronchiseptica*, but not the other two species. This trend also exists in the *Yersinia*

baramin,[7] which will be described later. Finally, the genome of *bronchiseptica* contains a gene which codes for a type II-polysacharide capsule, which contributes to survival in the environment.

In sum, in the *Bordetella* baramin the loss of genes due to genome decay is concomitant with the change in their pathogenicity. Loss of flagella or polysaccharides surface structures reduces the number of surface molecules which could be targeted by the human immune system. Herewith, the range of hosts is constricted as a byproduct of the process of genome decay. The resulting *Bordetella* species can hardly be candidates for evolution after losing so many genes in order to specialize to a tighter niche of a smaller number of host species.

The *Mycobacterium* baramin

A similar process of genome decay has been observed in species of *Mycobacterium*. For example, *M. ulcerans*, a bacterium which causes skin lesions is prevalent in poor, rural areas and is transmitted by water insects. The *ulcerans* genome is regarded as an intermediate phase in genomic reduction towards the ultra-decayed *leprae* genome. Compared to the *marinum* genome, which is 6.6 Mbp coding for 5,426 genes, the *ulcerans* genome is 5.8 Mbp, and contains 4,160 CDs and 771 pseudogenes, with 1,064 Kbp of deletions compared to *marinum*,[1] and another 1,232 Kbp lost in *ulcerans* residing in 157 regions. However, a stretch of 475 Kbp was identified in *ulcerans* which is missing from *marinum*. These include sequences coming from the IS2404 and IS2606 insertion elements. The correlation between the genome size and the number of genes in the *Mycobacterium* baramin is 0.97.

According to a study involving 128,463 SNPs in the genomes of different *M. ulcerans* populations, it was shown by phylogenetic analysis that the *M. ulcerans* populations have all descended from a common *M. marinum* progenitor.[8] This involved the accumulation of insert sequences and pseudogenes, and large genome deletions which occurred in the *marinum* genome in its transition towards the *ulcerans* genome. These deletions took along with them genes involved in intermediary metabolism and respiration. It is within two distinct lineages of the *ulcerans* genomes that large-scale genomic rearrangements took place.[9] Pseudogenes accumulated in the *ulcerans* genome include otsB genes, involved in trehalose metabolism, and gltA genes, which are involved in glutamine synthesis. Another gene, crtB, which is involved in the production of light-responsive carotenoid pigments, which protect *marinum* from direct sunlight, has a corresponding non-functional ortholog in *ulcerans*, implying that it has to adapt to another more protected ecological niche, hindering it from evolution as in the case of *B. pertussis* and *parapertussis*. The *marinum* genome has 192 CDs involved in substrate transport, whereas *ulcerans* has only 128. *Marinum* has more than 590 oxireductases, whereas *ulcerans* has more than 400. *Marinum* has 27 PKS genes in its genome, which are responsible for the production of secondary metabolites, whereas *ulcerans* has only 12. For example, the *cycdA* ortholog of the *cycdABCD* cytochrome is missing in *ulcerans*.[1]

Individual members of this baramin can be seen with members of the *Bordetella* baramin in figure 1.

Other species include *M. tuberculosis* and *M. leprae*, which cause tuberculosis and leprosy, respectively. Leprosy is a disease thought to have originated either in East Africa or India some 4,000 years ago, well within the biblical timescale.[10] Leprosy is still prevalent today, with reservoirs in Brazil and India, with 220,000 deaths reported in 2011. A most recent common ancestor of all extant and archaic *M. leprae* genomes existed some 1,400 to 2,700 years ago; *M. tuberculosis* is thought to have arisen from a cow pathogen, *M. bovis,* some 8,000–10,000 years ago. The *leprae* genome is much smaller compared to other *Mycobacterium* genomes (about 3.2 Mbp) and has a small number of active genes (~1,600), and a high number of pseudogenes (~1,300).[11,12,13] The M. *leprae* genome is so reduced, and it is has become so specialized, that the bacterium (an obligate parasite) can only be cultured in nine-banded armadillo. It grows so slowly in laboratory conditions that its cell division rate is 13 days. The bacterium has a propensity for infecting Schwann cells, and has lost three Mce operons encoding invasins, which are virulence factors.[11]

What makes the *M. leprae* genome interesting is its small genomic variance; leprosy genomes are >99.9% similar, and have only 807 polymorphic sites, and only 4–5 subtypes. They have found that ancient leprosy genomes are very similar to modern ones. Thus, due to this similarity if they can replicate factors and conditions that halted the spread of leprosy in the 14th century, then this could possibly put an end to the modern leprosy epidemic.[14,15] The *leprae* genome is very stable,[16] and is thus at the end stage of the genome reduction process.

The *Yersinia* baramin

Bacteria such as *Yersinia pestis*, *pseudotuberculosis*, *fredericksenii, kristensenii, ruckeri,* and *enterocolitica* belong to the *Yersinia* baramin.[17] The bacterium *Y. pestis* is famous for causing three major pandemics throughout human history, the Justinian plague, the Black Death (bubonic plague), and the Chinese plague in the 19th century,[18] and has been classified into four biovars (Antiqua, Medievalis, Orientalis, and Pestoides), according to its spread.[19] Comparisons between four published genomes

Table 2. Different stages in bacterial genome reduction.

Stage	Characteristics	Typical genome size	Example species
Free-living stage	Few pseudogenes or mobile elements, stable genome	>1Mbp, >1,400 genes	*Prochlorococcus marinus*
Host-restricted pathogens	High number of pseudogenes, mobile elements, instable genome, reducing in size	<1Mbp, >500Kbp, >500 genes	*Mycobacterium ulcerans, leprae*
Endosymbionts	Highly reduced, stable genome, few pseudogenes or mobile elements	<500 Kbp, 200–500 genes	*Carsonella ruddii*

of *pseudotuberculosis* and of 133 *pestis* strains show that *pseudotuberculosis* is more variable, and that *pestis* developed only more recently.[20,21] During its descent from the ancestral *Yersinia* species, *Y. pestis* has undergone lateral gene transfer, pseudogenization of some 149 genes, spread of insertion elements, genome size reduction, and genome rearrangement.[22] Changes in the pathogenicity of *pestis* compared to *pseudotuberculosis* include genes involved in cell adhesion to the host gut, such as YadA and invasin, as well as gene coding for the flagellum and chemotaxis. *Y. enterocolitica* is motile in this respect.[7] Genes such as hmsT, iucD, or phoP involved in biofilm production, iron acquisition, or adaptation to the intracellular environment have also been implicated in gene loss.[23]

Members of the *Yersinia* baramin can also be seen to form a tight cluster by themselves in figure 1. This is due to their characteristically low GC% compared to the other three baramins. The correlation between the genome size and the number of genes in the *Yersinia* baramin is 0.97.

This divergence is estimated to have occurred approximately 6,500 years ago (nicely fitting into the biblical timescale), with little sequence diversity having accrued during this period.[24,25] This age might be as young as 1,500 years (with the Justinian plague in ad 541). The age can also possibly be set to 3,350 years, during the time of the prophet Samuel. 1 Sam. 6:4–19 describes a plague which was caused by mice as vectors in the land of the Philistines. Verse 4–5 state the following:

> "Then said they, What shall be the trespass offering which we shall return to him? They answered, Five golden *emerods*, and five golden *mice*, according to the number of the lords of the Philistines: for one *plague* was on you all, and on your lords. Wherefore ye shall make images *of your emerods, and images of your mice that mar the land*; and ye shall give glory unto the God of Israel: peradventure he will lighten his hand from off you, and from off your gods, and from off your land."

Here the emerods in the King James version are thought to be tumors, caused by the black plague. It was about this time that *Rattus rattus*, the black rat, which is the vector for *Y. pestis*, arrived in the Middle East, and which are also interestingly noted in the passage from 1 Samuel. 1 Sam. 5:9 states: "And it was so, that, after they had carried it about, the hand of the LORD was against the city with a very great destruction: and he smote the men of the city, both small and great, and *they had emerods in their secret parts*." Here the secret parts refer to the groin where plague tumors regularly broke out.

According to some interpretations of Revelation, the black plague could correspond to the first of the seven plagues, correlating the Bible with science and history: "And the first went, and poured out his vial upon the earth; and there fell a noisome and grievous sore upon the men which had the mark of the beast, and upon them which worshipped his image" (Rev. 16:2). Indeed, one of the characteristic symptoms of bubonic plague is the coloured sores which appear on victims' skin.

Trypanosomatids

Interestingly enough, the processes described in the previous bacterial baramins are also present in nearly all eukaryotic superkingdoms. This process includes pronounced genes loss, elimination of repetitive elements, and reduction of average intron sizes.[26] For example, the trypanosome *Trypanosoma cruzi*, a single-celled eukaryote and the etiologic agent of Chagas disease, has a genome of 60.3 Mbp, and an estimated 12,000 genes as well as 3,590 pseudogenes, which are a clear sign of genomic decay. About half of its genome is made up of repetitive sequences such as surface proteins, retrotransposons, and subtelomeric repeats. In comparison, *T. brucei* has a genome of 26.1 Mbp and only 8,800 non-redundant protein-coding genes and 500 pseudogenes. Its GC% is also smaller, 46.4%.[27] 103 long non-coding RNA (lncRNA) transcripts were shown not to have any protein-coding potential. While this makes up a substantial part of the pseudogenes in *T. brucei*, still a large number of pseudogenes remain which are truly functionless.

Some important genes which are either lost or have been pseudogenized include a photolyase homolog, and a number of endonucleases. The enzymatic machinery for

Table 3. Correlation between genome size, GC%, and gene number in different groups of bacteria and organelles.

	Genome size vs GC%	Genome size vs gene number	GC% vs gene number
Mycobacterium	0.43	0.97	0.41
Bordetella	0.55	0.99	0.51
Yersinia	-0.25	0.97	-0.28
Mycoplasma	-0.10	0.75	0.04
Bacteria with reduced genomes	-0.23	0.99	-0.27
Organelles	0.95	-0.54	-0.26

non-homologous end-joining is also seemingly missing. Homologs for genes such as MCM10, CDT1, DBF4, and CDC7 are missing. Important signaling molecules, such as serpentine receptors, heterotrimeric G proteins, as well as some PK genes are also missing. This gives the picture that trypanosomes are undergoing the process of genome reduction, similar to bacteria, hallmarked by the spread of repetitive elements and retrotransposons, and the loss of genes involved in DNA repair.[28]

General genetic trends in bacterial genomes undergoing genomic decay

A description of the different stages in bacterial genome decay can be seen in table 2. From table 3 we can also confer that there is a very tight correlation with genome size and gene number. Besides gene loss, other trends are present in the genomes of bacteria. For example, recombination of genetic material occurs between closely related species, members of the same baramin. However, this does not count as production of new genetic material, as only existing genetic material is shifted, recycled, and repackaged. Horizontal gene transfer of plasmids and genes between species is also widely cited, but is in principle the same as recombination; no new genetic material is created during the process. Gene duplication is also cited for the creation of new genetic material. Gene duplication and divergence is not observable per se, as it is a process that requires the pseudogenization and sequential change of one copy of the duplicated gene into an entirely new gene.[29] However, we can completely disprove this notion by taking the following facts into consideration: it has been observed that two *strains* of the bacterium *E. coli*, that of K-12 and O157, differ from one another by *1.4 Mbp, which is one-quarter of its genome.*[30] This means that roughly one fourth of the genes between these two strains would have disappeared due to this deletion. You could also think of this in an inverse manner: the O157 strain of *E. coli* has roughly 1,000 more genes (larger than the number of genes in the genomes of even some so-called minimal organisms, which we will discuss later) than the K-12 strain, *yet they are both strains of the same species.* Gene duplication and divergence can only achieve this much! This situation is similar to that in plants, not just bacteria.[31]

Despite the glaring evidence for genome decay, a paper in the secular journal *Genome Research* states, "The loss of genes is not necessarily associated with the loss of DNA. In fact, *the half-life of a pseudogene in some eukaryotic species may be hundreds of millions of years*.[32] Yet, according to Graur *et al.,* "*it has been observed* in bacteria that the tendency of these nonfunctional regions is to disappear from the genome in short periods of time [emphasis added]".[13] So, observation of nature trumps secular religious beliefs. These observations come from a paper by Nilsson *et al*[33], which describe how methyl-directed DNA mismatch repair (MMR) protects chromosomal structure due to its antirecombinational activity. According to Nilsson:

> "… sixty lineages were each serially passaged for 1,500 generations, and deletions, totaling 224,873 bp, were found in four of the lineages. From these numbers, we calculated the arithmetic mean DNA loss rate in the mutS lineages to be ~2.5 bp per chromosome per generation [i.e. 224,873 bp (total amount of DNA deleted) / (60 (number of lineages) x 60 (number of serial passages) x 25 (number of generations of growth per serial passage))]."

They assume that with a generation time of 1 day for a single bacterium, a genome with a functional MMR system can be reduced by 1 Mbp in 50,000 years, and without an MMR system in 1,000 years. The replication time of bacteria here is overstated; a study of *Bacillus* species[34] shows the replication time is more like 3 hours (8 times in a day), reducing the deletion time from 50,000 years to 6,250 years (8 times less) with an MMR, and to 125 years without one. These dates are within the biblical timescale.

Genetic entropy in bacteria

What then will be the ultimate end of all this genome decay? If given enough time, bacterial genomes could erode so much that they would suffer a mutational meltdown. However, this is not so, as shown in a paper by Carter.[35] On the one hand, bacteria have smaller genomes and replicate

very quickly—a mutation or genomic decay would be felt very rapidly if bacteria didn't have very large population sizes and also lower mutation rates (around 10^{-10} compared to 10^{-6}–10^{-8} in eukaryotes). Thus, if mutations did accrue, these bacteria would quickly be replenished by ones without the mutation.

Nevertheless, genetic entropy does accrue, due to the Curse, and even bacterial genomes decay to the point that some bacterial species are what is known as minimal organisms, the genomes and number of genes of which are both extremely small. The number of genes in minimal organisms is so small that they are just enough so that the organism is able to survive (again, the exact opposite of gradual, upwards evolution). Some exist outside the host cell, whereas others have become intracellular endosymbionts, such as *Rickettsia prowazekii*, the aphid endosymbiont *Buchnera aphidicola*, and the psyillid symbiont *Carsonella ruddii*, which holds the record for the smallest-sized genome ever, at just 160 Kbp.[36,37] Other endosymbiont bacteria colonize plant and fungal species as well.

The minimal organism question

This is even more significant to the origins debate in that evolutionists must necessarily push for a minimal organism with as small a genome as possible. This is because if the number of genes is small enough, then it would be a lot easier for them to explain how a small number of genes were formed in the primordial chemical soup during abiogenesis.[38,39] Evolutionary theory here is at a definite disadvantage, because endosymbiont species are ruled out from the outset, since they must necessarily colonize larger organisms such as insects in order for them to survive (which weren't present in the chemical soup). As of yet, the smallest free-living genome that evolutionists could define is that of *Actionmarina minuta*, with approximately 800 genes and a genome size less than 1Mbp.[40] A minimal estimate for the gene content of the hypothetical/imaginary evolutionary superstar organism LUCA (last universal common ancestor) is around 1,340 genes.[41] This number was determined after analyzing 37,402 genes across 184 genomes, including genes from eukaryotic organisms with which to reflect LUCA's gene content most accurately.

This allows for a calculation of the probability of the first primordial cell arising from the chemical soup through random mutations. After looking at proteins from completed genomes representing one million sequences, scientists have been able to recognize 50,000 protein families; up from earlier estimates.[42,43] If we assume, conservatively speaking, that a protein has only 100 amino acids, then this means that the probability of a random sequence of 100 amino acids constituting a viable protein sequence is $6 \cdot 10^4 / 20^{100} \approx 3.9 \cdot 10^{-126}$. However, if we need 1,340 proteins (the number of proteins in the genome of LUCA) to be available all at once, then *the probability is further reduced to* $\approx (3.9 \cdot 10^{-126})^{1,340} \approx 10^{-167,500}$. This is the probability that a single-celled organism can arise from the chemical soup, and it is clearly unfeasible.

These bacteria themselves are generally cultivable only under laboratory conditions, or hardly at all. We know that besides evolutionists, there were no laboratories present during what is believed to be chemical evolution. In general, the smallest genome size of heterotrophic bacteria is around 1,300 genes; the minimum number of genes is still larger for free-living photoautotrophs, which code for more genes for the protein apparatus which transforms sunlight into energy. For example, *Prochlorococcus marinus*, the most abundant photosynthetic organism on Earth has around 1,700 genes. Many of these reduced genome non-endosymbiont bacterial species also depend upon the presence of other organisms to produce raw material that they themselves need for their metabolism.[44] These minimal organisms in themselves, by definition, constitute an irreducibly complex system, which is a hallmark of design and points to the existence of the supernatural Creator of the Bible. A good estimate of the minimum number of genes necessary for a stable single-celled organism would be between 1,400–1,500,[39] which is approximately the same number of genes which were estimated for LUCA. The minimal genome size for free-living bacteria is also around 1 Mbp.[45] In figure 1 we can see that there is a great gulf in the size of the genome and the number of genes between free-living bacteria and bacteria with reduced genomes, including endosymbiont species.

The case of endosymbiont bacteria is in itself noteworthy, since precisely due to endosymbiosis, genes unnecessary for living in intracellular are thus free to mutate, diverge, and differentiate as freely as possible, since all selection pressure has been relaxed on them. In other words, we have a scenario very much akin to the duplication of a great number of genes which are now free to mutate into newer genes. In this way we can test the 'duplication and divergence' hypothesis of evolution. Yet here in all cases the genomes of these endosymbiont bacteria we see genome reduction, loss of genes, pseudogenization, decrease in gene density, and the spread of transposons and other repetitive sequences. Even worse for evolution, Kuo *et al.*[46] hypothesize that the ratio of non-synonymous mutations to synonymous ones (d_N/d_S) are consistently larger in organisms with smaller genomes. This also goes along with a general trend that A+T content also increases in genomes undergoing genomic reduction.[47] For example, there is a moderate correlation between genome size and GC% (which is the opposite of A+T content) in *Mycobacterium* (0.43) and *Bordetella* (0.55) and organelles (0.95), according to Table 3. This relation also

holds moderately to the relationship between GC% and gene number. Here the correlation is 0.51 for *Mycobacterium*, and 0.51 for *Bordetella*. This reflects mutational bias rather than adaptation through selection based on random mutations.[48] This is because if mutations were truly random, the A+T content in bacterial genomes would not change but stay roughly the same.

This AT mutational bias is universal in bacteria, and even in strict symbionts, as a high GC–>AT transition ratio was measured in the endosymbiont species *Hodgkinia cicadicola*. Here 115 of 167 SNPs were shown to transition from GC to AT (68.9%).[49] The GC% of the genome of this organism is 58.4%, which is relatively high for endosymbionts. The mystery here for evolutionists is how could this species' genome have such a high GC% along with such a high AT mutation bias? An answer is made plausible according to creation science if this one species was created in a separate baramin which had high GC%.

No help for the endosymbiotic theory

It turns out that many features of reduced bacterial genomes do not help the endosymbiotic theory. In the case of *Carsonella ruddii* we can see which genes count as being dispensable to a bacterial endosymbiont (and conversely, indispensable to free-living bacteria). Many genes involved in replication, transcription, and translation are lost. Histone-like and single-stranded proteins, gyrase is lost; 9 aminoacyl-tRNA synthetases and 15 out of 50 essential ribosomal components are lost. Furthermore, it has lost the capability of synthesizing three essential amino acids.[35] According to Bennett *et al.*[50],

> "… the propogation and establishment of obligate symbionts in successive host generations requires symbiont cell replication and division. However, this remains one of the major essential functions typically lost from bacterial symbionts with small genomes."

This is remarkable, in that according to some evolutionary theories, the mitochondrion is derived from bacterial species which have undergone extremely massive genome reduction so as to result in organelles of eukaryotic cells. However, it is a well-known fact that mitochondria and chloroplasts divide and segregate within eukaryotic daughter cells.[51] This poses a problem for the endosymbiotic theory of evolution which states that organelles came from such endosymbiont bacteria with highly reduced genomes.

In general, the distribution of genes in bacterial genome is linear, with approximately one gene per Kbp of sequence. As we can see in table 3, the correlation between genome size and gene number is very significant, whereas for organelles, the correlation is moderately negative (-0.54). Insect symbionts have retained core enzymes for chromosome replication, translation, and transcription, such as the replicative DNA polymerase *dnaE*, whereas in organelles, these genes have been lost.[52] However, the types of genes as well as the gene density are different in organellar genomes. For example, 90% of many plant mitochondrial genomes are noncoding,[53] and can vary in size from 300 Kbp to 2.9 Mbp in the mitochondria of *Cucurbitaceae*.[54] Even some viruses have extremely large genomes, such as Mimiviridae, with genomes over 1Mbp with over 1,000 genes.[55] Interestingly enough, these authors hypothesize that the common ancestor [the archebaramin] of the cucurbits had an unusually large mitochondrial genome which itself underwent genomic contraction. Add to this the fact that the 'universal' genetic code is not really universal; codons are differently assigned in a number of organellar genomes.[56] This is important to note, since if codons are re-assigned to different amino acids, this disrupts fluid evolution from one protein sequence to another through random base pair mutations. Genome expansion due to introns and repeat elements also occurs in chloroplast genomes, although to a lesser extent than mitochondrial genomes.[57] These considerations prove that mitochondrial genomes are much too different from the genomes of bacteria with reduced genomes. Therefore we cannot conclude that mitochondria arose from the reduction of such bacteria.

Conclusion

Bacterial genome decay is a widespread phenomenon among different groups of bacteria whereby the size of the genome and the number of genes are continuously and starkly reduced. These groups of bacteria correspond to the created kinds of Genesis, and these genomic decay processes include pseudogenization, deletions, spread of insertion elements, non-random changes in base composition, and chromosomal rearrangements.

The species belonging to these created kinds have undergone genomic bottlenecks, and their common ancestor, in the case of a few of these created kinds examined in this review date back to only a few thousand years, consistent with the Bible. The process of genomic decay has also been observed in another major domain of life, in single-celled eukaryotes, and also does not necessarily need millions of years to occur. During this process many kinds of genes, involved in motility, metabolism, transcription, and replication are lost. From this, two things follow: a change in pathogenicity of the microorganism and a specialization to the host organism. High specialization due to gene loss makes it highly unlikely or impossible for the bacteria to be able to 'evolve' any further.

Further genome reduction in bacteria eventually leads to the minimal organism, which is defined as an organism which is capable of living freely in its environment without depending on a host organism for survival. Currently, the free-living bacterium with the smallest number of genes is *Actionmarina minuta*, with 800 genes. Advanced genome reduction leads to endosymbiosis with a host organism, where the bacteria receive nutrients and protection from the host in exchange for secondary metabolites or other nutrients. The minimal organism question is a great impediment for evolution, since it cannot explain how 800 fully functional genes arose through abiogenesis. Life does not come from non-life. This paper also presents a calculation for the possibility of abiogenesis to be $10^{-167,500}$.

The types of genes and gene density in organelles are different than those in bacteria. Organelle genomes also show signs of having undergone contractions, and also use different codons than do bacteria, which prove that organelles did not originate from bacteria. Since the differences in genome size and number of genes is so large between free-living and endosymbiont species, we can conclude that these are two major apobaramins, distinct from one another.

All in all, bacterial genomic decay goes counter to evolution, is a recent and fast-happening phenomenon, happening separately in different kinds of bacteria and other organisms, and is fully consistent with biblical creation.

Materials and methods

The data points depicted on figure 1, and also listed in table 1,[6] were taken from McCutcheon, 2011 and www.ncbi.nlm.nih.gov/genome. Here genome size, GC%, the number of genes, and the number of pseudogenes were from the representative genome information for each species from NCBI. Only those species were analyzed which had complete data. A data frame for R was made by creating a txt file with the genome size, GC%, and gene number for each species as well as a number denoting which group the species belonged to (*Bordatella*, *Mycoplasma*, *Yersinia*, or bacteria with reduced genomes, organelles).

Figure 1 was generated with the scatterplot3d command using R version 3.1.0. The following commands were used:

```
library("scatterplot3d")
pch=c(0,1,2,15,16,17)
bgd<-read.table("BGDdata.txt",header=T)
scatterplot3d(bgd[,1:3], pch=bgd[,4], angle=240)
```

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