

The origin of bubonic plague

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Although some forms of the bacterium *Yersinia* are harmless, other forms have devastated human populations, causing a plague of biblical proportions (Psalm 91:3–7, 9, 10). Bubonic plague, also known as the ‘Black Death’ that killed one fourth of Europe’s population in the 1300s, appeared as a great pestilence several times in the Old Testament, including in Psalm 91 and in 2 Sam 24:14–25. Perhaps the clearest example of such a plague is recorded in 1 Sam. 6:4–19, where there is a specific reference to the tumors on people (bubos = the tumors of lymph glands) and to rats (the animal vector that carried the plague bacterium, *Yersinia pestis*.) The biblical time frame for the plagues described in 1 Samuel was about 3,000 years ago.¹ Interestingly, experts on plague ‘evolution’ estimate the emergence of *Y. pestis* at about 1,500–20,000 years ago (within an evolutionary timeframe, of course).²

Plague’s origin is multifaceted

Many infectious diseases can be traced back to the decay and corruption of the original created design of microorganisms as a result of the Fall. Corruption literally means to destroy (from the Latin *corruptus*). The origin of pathogenic (disease causing) bacteria such as *Y. pestis* is complex and multifaceted, and may be explained by a combination of genes that were lost, added and moved. The story of *Yersinia*’s degeneration into the plague pathogen may serve as a model of ‘fast’ genomic decay and corruption.²

It appears that the beginning of pathogenicity in the genus *Yersinia* started with a net loss of chromosomal DNA from its original ‘kind’ (figure 1). Later, there were minor additions of plasmid DNA³ as well as DNA from

viruses and other bacteria. A few plasmid genes for toxins (table 1) have been acquired from another existing species, but many chromosomal genes have been lost. It takes only a few such genetic changes to produce a new, extremely infectious variant,⁴ so it may have taken only hundreds or a few thousand years to produce the current bubonic plague strain that has existed for about the last 500 years.

Loss of chromosomal DNA

Researchers hypothesize that key chromosomal genes (i.e. involved in metabolic pathways) were inactivated/lost in changing from a soil inhabiting *Yersinia* to a pathogenic *Yersinia* species.² Pathogenic *Yersinia* have lost structural information and function in about 149 genes. Of these, 58 are the result of frameshift mutations,⁵ 32 have undergone deletions, and the rest are nonsense mutations.⁶ These incomplete/inactivated genes or ‘pseudogenes’ are an important feature of the *Y. pestis* genome.⁷

Wren² suggests that the genes lost in *Y. pestis* affected bioenergetic functions, including dicarboxylic

amino-acid metabolism. This reduction of metabolic pathways may have allowed the bacterium to conserve energy. The newly emerged strains (variants) were thus streamlined, which might have contributed to the development of pathogenicity (i.e. plague) due to the genes they lacked. The absence of important biosynthetic genes is believed to be a hallmark of genome decay.

Genes added and moved

The corruption by three genes of a relatively benign recent ancestor of *Y. pestis* may have played a key role in the emergence of bubonic plague. Hinnebusch and colleagues, a plague expert team at the National Institutes for Health,⁸ maintains that the acquisition of two plasmid genes (i.e. just a few discrete genetic changes) in recent times changed the fairly harmless, *Y. pseudotuberculosis*, that causes mild food poisoning, to the agent of the ‘Black Death’. A third gene (carried on plasmid pMT1) produces murine toxin, an enzyme required for the initial survival of *Y. pestis* bacilli in the flea midgut (table 1).⁷ By acquiring this last

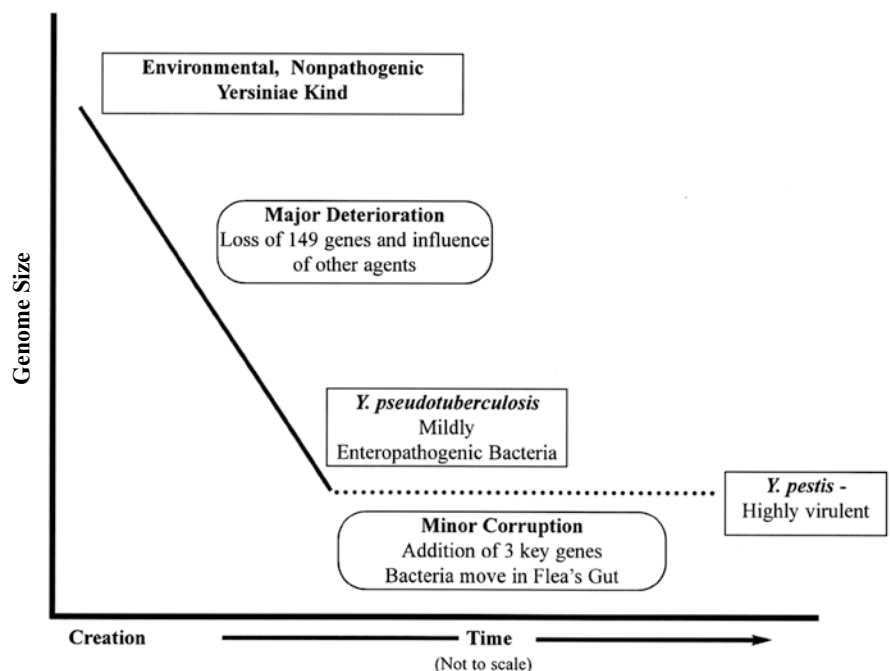


Figure 1. Origin of plague: the decay and corruption of nonpathogenic *Yersinia* to highly virulent *Y. pestis* in a few thousand years.

Table 1. Virulence-associated plasmids of *Yersinia pestis*.^{8–10}

Plasmid (size)	Transferred Factor	Action
pPla (9.6 kb)	Pla surface (protease)	activates plasminogen activator; destroys C3B; C5A (i.e. complement factors)
pYV (70.3 kb)	Yops (proteins)	interferes with phagocytosis and immune system
pMT1 (96.2 kb)	phospholipase D	bacterial transmission in fleas (Murine toxin)

gene from another organism, *Y. pestis* made a crucial shift in its host range, allowing it to survive in fleas, and devolved to relying on its blood-feeding host for transmission. This is just another example of the flexibility of many microbes in sometimes repackaging themselves into more dangerous agents of infectious disease.

This last corruption is one that distinguishes *Yersinia pestis* from all closely related, more benign bacteria such as *Y. pseudotuberculosis* and other *Yersinia* (e.g. *Y. enterocolitica*).⁷ In turn, as *Y. pestis* adapted to rely on its new blood-feeding host for transmission, the emergence of more deadly bacterial strains would have been favoured. It appears that these minor plasmid additions¹⁰ were the last changes made in an otherwise long series of genetic losses in *Y. pseudotuberculosis*' chromosome (figure 1).

One pathogenicity island was acquired by *Yersinia pestis* from a different bacterium.² This cassette of genes was not the result of evolution of new chromosomal DNA, but was an acquisition through lateral gene transfer.¹¹ It produced a corrupted message that gave bacteria a new 'position' in the gut. *Y. pseudotuberculosis*, which lacks the *hms* locus gene inhabits harmlessly the midgut of the flea. Plague bacilli, by contrast, have this inserted locus gene. Free from their original control, causing a lack of 'good' 'direction' information, the bacteria migrate from the midgut to the foregut, forming a plug of packed bacilli which is passed on to the victim when the flea feeds.

Genes, germs and Genesis

Plague bacteria are not the only microorganisms that have degenerated into disease-causing organisms. A more common recent example of a harmless bacteria 'devolving' into a pathogenic one is the intestinal *Escherichia coli* O157H7 strain that occasionally causes fatalities.¹² Other pathogenic bacteria that have undergone genomic decay include various mycoplasmas (e.g. *Mycoplasma genitalium* and *M. pneumonia*, the later causing pneumonia), and *Mycobacterium leprae* (the leprosy bacillus).^{13,14}

As we study the origin of infectious disease from a creationist, biblical perspective, bacteria provide us with a model of what may have happened to living things over time in a fallen, cursed and corrupted world. Many illnesses can probably be traced back to a loss of genetic information, plasmid acquisition, and gene translocation in organisms such as bacteria, fungi, etc. For those who know the Creator, we can rejoice that someday the Great Physician will restore all plagued bodies to a very good condition once again (Rev. 22:2–3).

References

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2. Wren, B.W., The *Yersinia*—a model genus to study the rapid evolution of bacterial pathogens, *Nature Micro. Rev.* 1:55–64, 2003.
3. Plasmids are circular, double-stranded units of DNA that replicate within cells independently of the chromosomal DNA.

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5. Frameshift mutations are changes in DNA where insertions or deletions of sequence occur that are not a multiple of three base pairs, thus disrupting the gene/protein normal code.
6. A nonsense mutation is any alteration of DNA that causes a codon representing an amino acid to be replaced by a termination codon.
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11. Lateral gene transfer is any process in which an organism transfers DNA to another cell that is not its offspring.
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