Diseases and Illnesses – did God create them?

Nancy Darrall

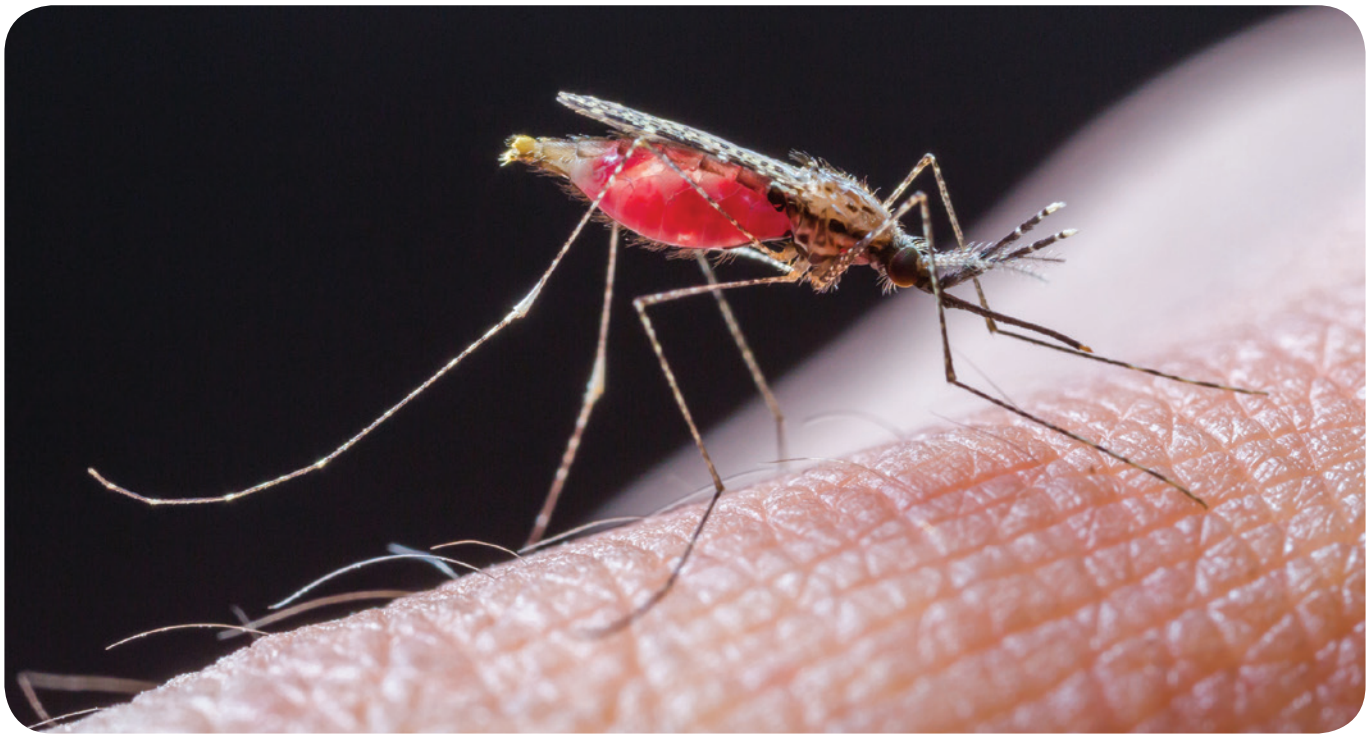
In Scripture, we read that the original creation was "good" and "very good". Yet today many suffer illnesses from a minor cold to life-threatening malaria, and crops can be slightly below standard or totally devastated by blight, rust or mildew. We can trace the origin of illnesses and disease to Genesis 3 and the Fall, but there is no reference to a further creation here after God finished his work of creation in Genesis 1. On the other hand, their creation within the time frame of Genesis 1 and 2, before sin entered the world, would contradict the description of the created order as "good" and "very good" (a well-discussed topic, e.g. Wilson, 2004). This article provides a fresh look at this dilemma, drawing examples from the scientific literature.

Malaria and Mosquitos – A Case Study

Introduction

The sequencing of the human genome and subsequent explosion of interest in sequencing a wide variety of plants and animals has provided a wealth of new information to look again at these issues. Malaria provides a good case study as it involves the unicellular pathogen, a vector, a host and a variety of environmental factors. After discussing this disease, some other cases are examined.

Malaria is one of the most devastating diseases of the tropics. Estimates suggest that over 650 million people are infected with malaria in the world and that about a million people die every year. The parasite that causes the illness (*Plasmodium* spp.) lives in the mid-gut of the mosquito and is transmitted to humans when an infected female mosquito pierces someone to get a blood meal. Once a person is infected, the parasite lives in and feeds on their red blood cells. There it reproduces and the next generation of the parasite is released into the blood stream to infect other cells. A further feed by a mosquito transfers



Mosquito sucking blood ID 30286852. © www.dreamstime.com

the parasite back into the mosquito so completing the life cycle.

This parasite is widespread in wild-living chimps and gorillas where there appear to be no ill effects, although primates in captivity infected with human strains do develop symptoms (Paupy, 2013).

Gene Loss and Gene Duplication make *Plasmodium* Dependent on Host

Plasmodium is a single-celled organism, a member of the Chromoalveolates which contains predominantly free-living forms but also some parasites. DNA sequencing reveals great similarities between the free-living and parasitic members and points to a single ancestor living in primates that later diversified. It is thought likely that cross infection with humans occurred by close contact with primates, possibly through keeping them as pets.

DNA in the mitochondria.

Mitochondria are structures (plastids) in cells primarily involved in energy release. The DNA in the mitochondria of *Plasmodium* spp. has undergone dramatic changes in size, organization, and encoding functions compared with the free-living organisms. It is unusually small and has been described as a relic. It contains only three genes, which means that the genes coding for many key metabolic components are absent. Interestingly, those three genes are present as multiple repeats, the same as the genes found in very similar, free-living dinoflagellates. For example there are 150 copies of one gene in *Plasmodium yoelii* making up an amazing 3% of the DNA. These genes code for proteins that are essential for the release of energy within cells. The high numbers of repeats can be considered an adaptation to life in a host, by increasing the activity of these enzymes. However, they are merely copies of genes that are already present in the free-living forms (Foth *et al.,* 2005; Vaidya and Mather, 2009).

DNA in the chloroplast.

Chloroplasts are another type of plastid within the cells of plants and algae. Their primary role is to capture energy from light to synthesize carbohydrates. In *Plasmodium* there is a "relict plastid" (apicoplast), so similar that it is thought to be "a legacy of the malaria parasite's distant photosynthetic ancestry" (Ralph, 2004). It encodes only 30 enzymes. A large proportion of the genes are devoted to evading the immune reaction of the host and enabling host-parasite interactions. For example, it produces chemicals called terpenes through metabolic pathways normally found in chloroplasts. These are breathed out by the host and are attractants for the mosquito, so enhancing disease transmission from one person to another (McSpadden Gardener, 2004; Kelly *et al.,* 2015; Kohler *et al.,* 1997).

Increased Activity of Gene in a Mosquito responsible for Switch to Human Host

One mosquito subspecies, *Aedes aegypti aegypti,* lives in forested areas. It prefers to feed on forest animals and appears to do so without causing harmful infections. However, another subspecies, *A. aegypti formosus,* prefers human blood and has spread yellow fever, dengue, and chikungunya viruses widely across the world. This subspecies seems to have emerged recently and contains a number of mutations. Fourteen genes were strongly linked to the preference for humans. One of these, *Or4,* is highly expressed in the mosquitos that crave human blood (McBride *et al.,* 2014). Here we have increased activity of a gene that is already present causing host-switching and the emergence of these diseases in humans.

The Mosquito's Gut Flora are Gate-Keepers of the Malaria Disease

The human body is host to a variety of organisms including a diverse and complex microbial community. A healthy gut flora is known to protect against a variety of diseases, such as illness from *Clostridium difficile,* by direct inhibition of those bacteria that cause diseases as well as by stimulating the immune system of the host. The gut of insects is no different. Two bacteria, *Wolbachia* and *Chromobacterium,* are amongst those often found in the gut of insects. Such insect-bacteria associations are known to affect the ability to transmit malaria and also the dengue virus to humans. *Wolbachia* directly inhibits survival of both the larval and adult stages of *Plasmodium* in mosquitos. It does this by activating immune responses and producing substances that directly inhibit the pathogens. As the gut flora is inherited maternally, there are trial programmes in northern Australia to inoculate mosquitos with *Wolbachia* to block disease transmission to humans (Hoffman and Turelli, 2013; Ramirez *et al.,* 2014).

So, infection with a *Plasmodium* sp., which has degenerate DNA and the lack of a normal member of the gut flora, *Wolbachia,* can transform the mosquito into the carrier of a devastating disease.

Environmental and Behavioural Factors affect Malaria Transmission

There are over 500 different species of mosquitos but only about a dozen carry the malarial parasite. All adult mosquitos feed on the nectar and honeydew of plants and this provides enough nourishment for both males and females to live. However, before they lay eggs, females of those mosquito species that cannot store enough food supplies from their larval stage require a blood meal. The same slender, sharp mouthparts used for penetrating plant tissues are used for sucking up blood. The saliva of blood-feeding mosquitos contains a wide variety of components that work synergistically to counter the defence mechanisms in animals and plants, to aid digestion and to enable the mosquito to draw up fluids. The contents of the saliva are adjusted by the mosquito depending on the food source (see Chagas *et al.,* 2014).

Since only a minority of mosquitos require a blood meal, it is thought to be an adaptation by a few species in conditions where their plant food sources are now less rich in nutrients. An interesting parallel is found in the kea, a parrot that has colonised the harsh mountains of the Southern Alps on South Island, New Zealand. Parrots generally feed on plants, and the closely related parrot on North Island is no exception. However, those kea that have colonised the poorly vegetated Southern Alps have developed carnivorous strategies, as in the winter, when food is scarce and the climate harsh, many can die. Some kea will dig chicks of sooty shearwaters from their burrows to eat. Others have adapted to feed on carcasses of animals in sheep farming areas. Some even attack live merino sheep in the high hills, feeding on the fat layer beneath their woollen coats. In these conditions, the environmental stress has led to a change in behaviour; the same beak that is used

to dig up plants and forage for fruit and nuts is able to attack the shearwater chicks and sheep. The total destruction of soil and plant communities at the Flood in the time of Noah is likely to have precipitated many such changes in feeding habits as the post-Flood world would have been a very impoverished place. It is notable that Noah was given animals for food after the Flood, suggesting that plants alone would not provide adequate nutrition.

Same Patterns – Other Diseases

Gene Loss

Leprosy and tuberculosis (TB) are two devastating diseases and the bacteria responsible belong to the genus *Mycobacterium.* While some are free-living organisms with large genomes, (e.g. *M. smegmatis,* 7Mb), leprosy is caused by a member of this genus with a much reduced genome, only 3.27 Mb. Comparisons show that it is closely related but has lost more than 2,000 active genes through deletion or decay, eliminating many important metabolic functions and their regulatory networks. Only 391 soluble proteins are produced, compared with an estimate of over 6,000 from the very similar, free-living *M. smegmatis.* Similarly, *M. tuberculosis,* the cause of TB, has a genome of 4.4Mb and only 1,800 soluble proteins. Here are two examples of diseases where originally free-living bacteria now have a degenerate genome and so need to live within the human body to survive.

Reduced genome size is not restricted to bacteria that cause harmful diseases; it is also found in bacteria that form mutually beneficial partnerships with their hosts, such as the members of our gut flora. One example is *Escherischia coli,* which is beneficial in several ways including provision of Vitamin K, assisting our immune system and breaking down waste. In the protective environment of the host, many substances needed by the bacteria are readily available from the host. This means that the bacterial genes to synthesize them are redundant and are eliminated. The consequences are positive or deleterious only from the perspective of the host; we call them symbionts or pathogens respectively depending on whether disease symptoms occur (see Cole *et al.,* 2001; Francis 2003: Pirofski and Casedevall, 2012).

Gene Duplication

Disease symptoms are often caused by overproduction of substances normally present in cells to levels that are harmful or toxic. This does not require new genes; it is achieved by multiple copies of the same gene, or a change in the regulation of gene activity, or both. This process is not limited to pathogens; symbionts often have multiple copies of genes which code for nutrients that the host cannot synthesize.

Animal diseases. The pathway of pathogen development from a free-living organism can be traced in *Toxoplasma gondii,* responsible for toxoplasmosis, a disease we commonly associate with cats. The degree of virulence of this pathogen is largely due to a gene, *ROP-5,* which belongs to the kinase family of enzymes found in plants, animals and yeasts. Firstly, there was a mutation of an original kinase gene, a key regulator of cell function, into a pseudokinase. Later amplification and diversification of this gene gave rise to a whole family of similar genes encoding the pseudokinases that are injected directly into host cells causing the disease symptoms (Reese *et al.,* 2011). Deletion of this gene cluster causes complete loss of virulence.

Toxins in venom. Venom toxins are found in a wide range of animals including snakes and are injected into another animal or human for defence or predation. They contain proteins and other substances that are similar to the products of genes required for the normal housekeeping functions of cells. These genes have been recruited and then modified and/or amplified, sometimes into large families of genes, which means that far greater quantities of the venomous protein(s) can be produced. In some cases, gene products are assembled in different ways (alternative splicing) to produce toxins. These genes encode a variety of functional activities and potencies and they act by blocking metabolism, destroying substances or preventing normal physiological responses (see Casewell *et al.,* 2013; Fry, 2005).

Plant diseases can devastate crops, Early tomato blight. © Makaid, www.istockphoto.com

Plant diseases. Repetition of sections of the genome is also a key feature of plant pathogens. Akin to our gut flora, plant tissues are colonised with a wide variety of micro-organisms. Some are beneficial to plant growth, such as nitrogen-fixing bacteria in legumes, while a few cause serious diseases such as rust fungi on cereals, mildews and potato blight. Sequenced genomes of pathogens are very similar to those of the free-living forms. One notable difference is that there has been extensive proliferation of regions of repetitive DNA. In the fungus that causes potato blight, *Phytophthora infestans,* these regions can comprise up to 74% of the genome. The DNA codes for enzymes important in breaking down the cell wall of the host; the more copies, the greater amount of enzyme produced, and so the greater virulence of the disease. This process of gene amplification is not confined to disease development and symbiosis. It is one of a whole raft of important, beneficial processes that allows organisms to adapt to stressful environments and survive in extreme or variable conditions, for example in soils with high levels of heavy metals (see Rafaelle and Kamoun, 2012).

Other Processes – Acquisition of Plasmids

Organisms such as bacteria and fungi undergo sexual reproduction by exchanging genetic material during temporary joining or permanent fusion. This allows plasmids, which are small pieces of DNA carrying additional information such as antibiotic resistance, to transfer as well. Increasingly, these transfers are known to be important in the rise of new diseases such as the transformation of *E. coli,* a beneficial member of our gut flora, into a dangerous pathogen causing diarrhoea and other, sometimes fatal, infections. The plasmid responsible is thought to have come from the disease-causing bacteria, *Vibrio cholera,* within the human intestine.

The bacterium that causes anthrax contains two plasmids. It belongs to the *Bacillus* cereus group of organisms. They are ubiquitous in the natural world, primarily in the soil but also commonly as part of the gut microflora of invertebrates. Some are intimately associated with plant roots in a symbiotic relationship and can promote plant growth, improve drought tolerance and suppress disease and insect damage. Many forms of *B. cereus* are known, varying greatly in lifestyle but with only very minor genetic differences in their circular chromosome. It is the genetic content of the acquired plasmids they contain that determines their ecology and their ability to cause disease. Anthrax contains two plasmids known as pXO1 and pXO2 that are responsible for their pathology. Another *B. cereus,* containing a variant of the plasmid pXO1, has been isolated as a cause of food-borne toxins responsible for gastrointestinal diseases. A single section of DNA in the pXO1 plasmid produces a family of peptides ranging from non-toxic to highly toxic (see Sozhamannan, 2006; Wood, 2002). There is evidence that during the process of peptide synthesis from the DNA of this plasmid, proof reading is not as rigorous as usual and this is responsible for the diversity and enormous variation in disease symptoms.

Plasmids can influence a wide variety of traits, not just disease virulence and antibiotic resistance. They may be hugely beneficial, transferring traits necessary for accommodation to a symbiotic lifestyle as well as enabling free-living bacteria to have the versatility to adapt to, and remain fit in, changed environmental conditions (see Dobrindt *et al.,* 2004; Rasko and Rosovitz, 2007; Schuh and Fischetti, 2009). However, bacteria that have a symbiotic lifestyle can more readily become pathogens because they already have the features necessary for life in close proximity to a host. All that is required is that the symbiont gains a further trait causing damage to the host as in the case of the toxic peptides produced by pathogenic *E. coli.*

Combinations of Genetic and Environmental Factors

Most pathogens and symbionts have probably adapted to living within a host by a combination of gene loss, gene duplication and plasmid acquisition. Damaged and poor environments also play a big role. A couple of examples will suffice.

Buruli ulcer is an emerging disease associated with disturbed tropical wetlands, a good example of multiple changes in the transformation of a free-living organism into a serious pathogen. The causative agent has been called *Mycobacterium ulcerans.* It has an almost identical genome sequence to *M. marinum,* a ubiquitous fast-growing species living in water where it is associated with plants and algae. In contrast, *M. ulcerans* is associated with slowly flowing or stagnant watercourses. It has lost around 23% of its genes and acquired a virulence plasmid that carries multiple copies of a gene giving it the ability to produce the polyketide mycolactone. Polyketides are one of a large class of compounds produced by bacteria, fungi and plants. It is the high levels of mycolactone produced by the multiple gene copies in this pathogen that are toxic, not only suppressing the innate inflammatory responses to infection but also causing tissue damage. Mild ulceration of the skin can progress to massive tissue destruction but with minimal inflammation (Demangel, 2009).

A very similar set of changes has been uncovered in a fungus disease of poplar trees. Horizontal transfer of DNA from fungi associated with wood decay has combined with up-regulation of genes encoding hemicellulose-degrading enzymes. This has allowed the fungus to colonize poplar woody stems causing the lethal canker on leaves, stems and branches (Dhillon *et al.,* 2015).

Another example concerns *Yersinia* species, most of which are free-living bacteria that do not cause disease. However, three species are known to be human pathogens: the plague bacillus *Y. pestis,* and two others that live in the gut, *Y. enterocolitica* and *Y. pseudotuberculosis.* A survey of the currently known species shows acquisition of virulence plasmid(s) followed by further changes, including functional gene loss and reduced metabolic flexibility, compared with free-living forms *en route* to pathogenicity (Reuters *et al.,* 2014).

Lyme disease is an increasingly common and potentially serious disease we can pick up from ticks harbouring the bacterium, *Borrelia burgdorferi.* This pathogen contains at least 17 linear and circular plasmids, the combined size of which is more than half as big as its chromosome. They include sections of multiple repeats of plasmid genes. The biological significance of these

plasmids is not yet clear but is thought to enable the bacterium to evade host defences. Environmental factors are also important in controlling infection. Where native vertebrate diversity is high, the ticks feed from a wider variety of hosts, many of whom are poor reservoirs for the pathogens because of their natural immunity. So, this disease was unknown until recently. However, when diversity is reduced to a few species, such as deer, sheep and people, infection is more likely, as the pathogen can adapt to these few hosts and build up a reservoir for future transmissions (Fraser *et al.*,1997).

Environment and Behaviour – A Balanced Ecosystem is Protective

The ecosystem of the human body. Some people are carriers of disease; the pathogens live in them but there are no disease symptoms, whereas in others there can be a catastrophic reaction. In TB, it is the immune reaction of the host that results in the disease symptoms of severe lung inflammation and tissue damage. In whooping cough it is the strong inflammatory response to the diphtheria toxin that causes the fatal obstruction to breathing. These diseases would not exist if the immune response was less or if the bacteria did not produce the toxins; here the relationship would be neutral or symbiotic. A healthy human gut flora is a good example of such a relationship. Another important example is the community of bacteria, fungi and animals in the soil.

Soil organisms. An ecosystem where there is a diverse array of organisms protects from parasites, pests and diseases. In the soil, bacteria can multiply to very high levels around and also between the cells in the roots of plants where there is an ample supply of nutrients. These bacteria (rhizobia) protect plants from fungal pathogens, bacterial diseases, insect attack and infestations with nematodes. Some soils are so well-endowed that they are known as disease suppressive soils. Although rare, they provide examples of what a healthy soil could provide for plants and crops. Rhizobia suppress disease through competition for nutrients, production of antimicrobial compounds and enzymes that destroy the cell walls of fungi and nematodes.

One example is that of muskmelons, which have been grown since antiquity in the Châteaurenard region of France with little trouble from *Fusarium* wilt even though the fungus is present. In nearby regions the disease is so severe that sometimes cultivation of the crop is abandoned. Plant growth-promoting bacteria are now being exploited commercially for protection against various pests and diseases. However, resistance tends to decrease with time showing that by and large our present-day soils are not able to support the microbial population necessary to support healthy plant growth (Persello-Cartieaux *et al.,* 2003; Ramamoorthy *et al.,* 2001; Schroth and Hancock, 1982). This is a predictable outcome of the biblical account of the worldwide flood at the time of Noah. The flood covered the land surface of the whole earth for a large part of a year. The soils created in the beginning would have been completed destroyed by this event. Soils formed afterwards would have come from flood deposits of rotting animal and plant material mixed with mud, silt and sand.

Conclusions

Organisms living in close association with a host obtain a variety of nutrients from them and so no longer need to synthesise them. The respective genes are then lost which means that those organisms are then no longer able to survive without the host as free-living organisms. When both benefit from the association it is known as symbiotic, but if other genetic changes occur as discussed above the host is harmed and we describe the outcome as a disease.

Gene mutations, amplification and/or changes to the activity of a gene(s) lead to excessive levels of normal metabolites, causing such things as the damage in diseases and the toxicity of snake venom. Similarly, host over-reaction or susceptibility can lead to disease symptoms.

Plasmids with benign metabolic functions in a non-pathogenic organism can be transferred into an organism that is closely associated with a host. These products can then have harmful effects in the host and a disease is born.

Competition from members of a diverse free-living and symbiotic ecosystem can minimise the survival of pathogenic forms, both in living organisms and in the soil. Reduced diversity, or an imbalance, provides conditions for disease build-up and infection to occur. The Noachian Flood would have destroyed the original soils and left an impoverished ground surface vulnerable to such problems.

This article provides examples of how diseases may arise from the degeneration of free-living forms: by small changes in DNA, by duplications, by lax proof-reading of genes and their products, and by the transfer of genetic material between organisms. It is not necessary to invoke their creation after the Fall or their existence before the Fall. ■

Infectious diseases cause suffering and death to millions in the world today.

References

Casewell, N.R. *et al.* (2013). Complex cocktails: the evolutionary novelty of venoms. *Trends in Ecology and Evolution,* 28(4): 219-229.

Chagas, A.C. *et al.* (2014). Collagen-binding protein, Aegyptin, regulates probing time and blood feeding success in the dengue vector mosquito, *Aedes aegypti. Proceedings of the National Academy of Sciences,* 111: 196946–6951.

Cole, S.T. *et al.* (2001). Massive gene decay in the leprosy bacillus. *Nature,* 409: 1007-1011.

Demangel, C., Stinear, T.P. and Cole, S.T. (2009). Buruli ulcer: reductive evolution enhances pathogenicity of *Mycobacterium ulcerans. Nature Reviews Microbiology,* 10: 50-60.

Dhillon, B. *et al.* (2015). Horizontal gene transfer and gene dosage drives adaptation to wood colonization in a tree pathogen. *Proceedings of the National Academy of Sciences,* 112(11): 3451–3456.

Dobrindt, U. (2004). Genomic islands in pathogenic and environmental microorganisms. *Nature Reviews Microbiology,* 2: 414-424.

Foth, B.J. *et al.* (2005). The malaria parasite *Plasmodium falciparum* has only one pyruvate dehydrogenase complex, which is located in the apicoplast. *Molecular Microbiology,* 55 (1): 39–53.

Francis, J. (2003). The Organosubstrate of Life: A creationist perspective of microbes and viruses. https://answersingenesis.org/biology/microbiology/the-organosubstrate-of-life (accessed 16 April 2015)

Fraser, C.M. *et al.* (1997). Genomic sequence of a Lyme disease spirochaete. *Borrelia burgdorferi. Nature,* 390: 580-586.

Fry, B.G. (2005). From genome to "venome": Molecular origin and evolution of the snake venom proteome inferred from phylogenetic analysis of toxin sequences and related body proteins. *Genome Research,* 15: 403–420.

Hoffman, A.A. and Turelli, M. (2013). Facilitating Wolbachia introductions into mosquito populations through insecticide-resistance selection. *Proc. R. Soc.* B, 280: 20130371

Kelly, M. *et al.* (2015). Malaria parasites produce volatile mosquito attractants. *ASM Mbio.,* 6(2): e1-6.

Kohler, S. *et al.* (1997). A plastid of probable green algal origin in apicomplexan parasites. *Science,* 275: 1485-1489.

McBride, C.S. *et al.* (2014). Evolution of mosquito preference for humans linked to an odorant receptor. *Nature,* 515: 222-227.

McSpadden Gardener, B. (2004). Ecology of *Bacillus* and *Paenibacillus* spp. in agricultural systems. Phytopathology, 94:1252-1258.

Paupy, C. *et al.* (2013). *Anopheles moucheti* and *Anopheles vinckei* are candidate vectors of ape *Plasmodium* parasites, Including *Plasmodium praefalciparum* in Gabon. PLOS ONE, 8(2): e57294.

Persello-Cartieaux, F. *et al.* (2003). Tales from the underground: molecular plant–rhizobacteria interactions. *Plant, Cell and Environment,* 26: 189–199.

Pirofski, L. and Casadevall, A. (2012). Q&A: What is a pathogen? A question that begs the point. *BMC Biology,* 10: 6.

Rafaelle, S. and Kamoun, S. (2012). Genome evolution in filamentous plant pathogens: why bigger can be better. *Nature Reviews Microbiology AOP,* 1-14. published online 8 May 2012;

Ralph, S.A. (2004).Tropical infectious diseases: Metabolic maps and functions of the Plasmodium falciparum apicoplast. *Nature Reviews Microbiology,* 2: 203-216.

Ramamoorthy, V. *et al.* (2001). Induction of systemic resistance by plant growth promoting rhizobacteria in crop plants against pests and diseases. *Crop Protection,* 20: 1-11.

Ramirez, J. L. *et al.* (2014). *Chromobacterium* Csp_P reduces malaria and dengue infection in vector mosquitoes and has entomopathogenic and in vitro anti-pathogen activities. *PLOS Pathogens,* 10(10): e1004398

Rasko, D.A. and Rosovitz, M.J. (2007). Complete sequence analysis of novel plasmids from emetic and periodontal *Bacillus cereus* isolates reveals a common evolutionary history among the *B. cereus*-group plasmids, including *Bacillus anthracis pXO1. Journal of Bacteriology,* 189(1): 52–64.

Reese, M.L. *et al.* (2011). Polymorphic family of injected pseudokinases is paramount in *Toxoplasma* virulence. *Proceedings of the National Academy of Sciences,* 108(23): 9625–9630. Reuter, S. *et al.* (2014). Parallel independent evolution of pathogenicity within the genus *Yersinia. Proceedings of the National Academy of Sciences,* 111(18): 6768-6773.

Schuh, R. and Fischetti, V.A. (2009). The secret life of the anthrax agent *Bacillus anthracis*: bacteriophage-mediated ecological adaptations. *PLOS One,* 4(8): e6532.

Schroth, M.N. and Hancock, J.G. (1982). Disease-Suppressive Soil and Root-Colonizing Bacteria. *Science,* 216: 1376-1381.

Sozhamannan, S. *et al.* (2006). The *Bacillus anthracis* chromosome contains four conserved, excision-proficient, putative prophages. *BMC Microbiology* 6:34.

Vaidya and Mather, M.W. (2009). Mitochondrial evolution and functions in malaria parasites. *Annu. Rev. Microbiol.,* 63: 249–67.

Wilson, G. (2004). The origins of natural evil. In: *Discovering the Creator, Proceedings Of The Third Biology Study Group,* USA, June 9, 2004, (R.W. Sanders, ed.), Occasional Papers of the BSG, No. 4: p6.

Wood, T.C. (2002) The terror of anthrax in a degrading creation. http://www.icr.org/article/terror-anthrax-degrading-creation/ (accessed 16 April 2015)