Why do viruses mutate to a greater extent than bacteria?

The current COVID-19 pandemic has arisen because a highly virulent and contagious virus has evolved to which there is no inherent worldwide herd immunity. We have seem similar events before such as the Spanish flu epidemic in 1918 when a huge shift in the antigenic structure of the influenza virus effectively rendered all previous acquired immunity to the influenza virus redundant. An individual's immune response has effectively to start from scratch after exposure, which goes some way in explaining the high morbidity rates when these events occur.

This made me think why we don't see such large shifts in the antigenic material of the common foodborne pathogens such as *Salmonella, Listeria and STEC.* We often observe slight mutations in these bacteria which may manifest as increased resistance to antibiotics, but seldom do we see the dramatic shifts in both the structure and composition of the bacterium which effectively constitutes a new or novel organism.

I believe the reason lies in the differences in how bacteria and viruses multiply. Bacteria grow by binary fission whereby one bacterial cell simply splits down the middle, making a clone of itself which has identical genetic material. Mutations can occur, and because of the potentially rapid growth rates, these mutations are far more likely than in slower growing cell division such as seen in our own bodies. Viruses are also subject to the effects of mutations, but crucially the big difference lies in a process known as viral recombination.

Viruses infect host cells, effectively taking over the cells genetic material, turning them into virus factories which produce millions of copies of the original invading virus. Eventually the cell membrane ruptures releasing the new viruses which go on to invade other cells. The cruel irony is that the "new" virus particles have actually been made by our own cells.

Occasionally two different viruses can invade the same cell at the same time. What happens next is that both of the invading virus's genetic material can get mixed together creating a distinctly different virus with uniquely recombined and re-assorted genetic material from both of the two original invaders.

Continued....

It is therefore easy to understand why we see such dramatic shifts in viruses and the emergence of new or novel strains such as Severe Acute Respiratory Syndrome (SARS), Middle East Respiratory Syndrome (MERS) and Coronavirus disease (COVID-19).

Viral recombination stands a much better chance of happening if the virus is present not only in humans but also has an animal reservoir population in which it can infect. This enables recombination and mutations to occur away from the human population until the changes become so drastic that the existing herd immunity in the human population becomes nullified and useless. It is thought that the animal reservoir for SARS were civets (a small nocturnal mammal), for MERS it was camels and possibly bats for COVID-19.

This antigenic shift explains why we experience influenza pandemics every 10 years or so. Influenza infects many other species other than humans, and as a result they can quietly "tick over" in the animal community until a recombination occurs which either significantly alters the shape of the virus so it is no longer recognised by our antibodies or enables the virus to have increased virulence properties. This is why the new influenza pandemics are often called after the particular animal reservoir where they originated from such as swine flu or bird flu.

As well as evading our immune system, this "new" COVID-19 virus has developed spike proteins which protrude from its surface which have evolved to attach to a specific receptor site on the surface of human cells, which has significantly enhanced the virulence of this organism.

Analysis of the genetic material of COVID-19 suggests that the completely random recombination of the genetic material makes it very unlikely that this virus was man-made and had "escaped" from a laboratory as has been speculated in certain quarters.

The events of the last few months serve as a stark reminder that microbiological populations are dynamic and are constantly evolving and changing, which means that we have to develop new strategies in both the detection of, and in the treatment and management of the illnesses which they create.

Update on raw milk pathogen testing

Last month's Micro Bulletin contained an article on raw milk legislation, highlighting a study published in the Journal of Epidemiology and Infection which stated that the current testing regime in England and Wales was not fit for purpose as it did not include pathogen testing and relied only on criteria based around the indicator tests of ACC and Coliforms.

Well right on cue, on the 6th March the Food Standards Agency published a new guidance document for producers of raw drinking milk for direct human consumption. In the new guidance, testing for pathogens such as *Salmonella, Campylobacter, STEC* (Not Detected in 25ml), and *Coagulase Positive Staphylococci* (target <10,000cfu/ml) is considered as best practice. The guidance states that levels of between 2-10,000cfu/ml of *Coagulase Positive Staphylococci* may indicate poor process hygiene and temperature controls and would need to be investigated.

With regard to *Listeria monocytogenes*, the guidance quotes the criteria stipulated in the EU Regulation 2073/2005, but acknowledges that where the raw drinking milk has a shelf life of less than four days, then it can be assumed that *L monocytogenes* will not grow. However the operator must be able to demonstrate that the levels of *L monocytogenes* will not exceed 100cfu/ml at any point within its shelf life. Where a shelf life of greater than four days is contemplated, then the operator must ensure that *L monocytogenes* is absent in 25ml before it leaves the business and remain at levels of less than 100cfu/ml throughout its shelf life.

Compliance with the criteria for *L monocytogenes* must be demonstrated as part of a routing sampling and testing regime to demonstrate that the operator's food safety management scheme is effective.

What is the cost burden of foodborne illness?

Whilst the financial burden of foodborne disease should be secondary to the human consequences of the illness, it does nevertheless form a crucial part of assessing the impact of intervention strategies, and a new study performed jointly by the Food Standards Agency and university academics has attempted to put a cost burden on foodborne illness in the UK.

Based on the 2018 figures of 2.4 million foodborne cases per year, the burden for the U.K. from foodborne illness was estimated at £9.1 billion.

Norovirus imposes the most economic and societal burden at an estimated annual cost of £1.7 billion followed by *Campylobacter spp*. at £713 million and *Salmonella spp*. at £210 million. *Shiga Toxin-producing E. coli (STEC) O157* with £4 million and *Cryptosporidium* at £2.1 million have the least burden.

Individual cases of *Campylobacter*, which are common but generally not severe, impose a burden of £2,380 each while *Listeria,* the least common of the 13 pathogens measured, has a burden equivalent to £230,748 per case due to more deaths, resulting in a higher human cost.

Of the 2.4 million foodborne related cases, 16,300 needed hospital treatment and 180 deaths were reported. *Norovirus* accounts for the most illness at around 383,000 infections, followed by *Campylobacter* and *Clostridium perfringens* with around 299,000 and 85,000 respectively. *Listeria monocytogenes* has the fewest at 162 but 26 of these died so it has the highest proportion of fatalities.

Further updates on COVID-19 and the possibility of foodborne transmission?

As things are changing on a daily basis, I thought it pertinent to reiterate the message in last month's bulletin on Coronavirus and the current opinion on whether there is any evidence of foodborne spread of the disease. The European Food Safety Authority (EFSA) has once again stated that there is currently no evidence that food is a likely source or route of transmission of the virus.

The European Centre for Disease Prevention and Control (ECDC) has said that while animals in China were the likely source of the initial infection, the virus is spreading from person to person – mainly via respiratory droplets that people sneeze, cough, or exhale.

Scientists and authorities across the world are monitoring the spread of the virus and there have not been any reports of transmission through food. For this reason, EFSA is not currently involved in the response to the COVID-19 outbreaks.

Knock on effects....

More than ever, the coming months will bring immense challenges to our business. As well as coping with our own inevitable staff and supply issues, we must be prepared for a potential increase in food safety issues.

Our clients will be experiencing similar problems, and we may have a "perfect storm" of staff shortages, increased demand for certain food commodities leading to an increased workload, fatigue, sourcing ingredients from unproven suppliers and crucially a potential increase in the vulnerable groups who will be consuming the food which may prove devastating if these individuals are exposed to organisms such as *Listeria.*

Our job is vital in ensuring the safety of our community, but in the coming months it will never have been more important. To all of my colleagues working in the laboratories at this time, you have my gratitude and respect. You are doing an amazing job. Stay safe and well.