Mechanisms of viral transmission

Most of the new viral diseases that enter the human population are enzootic viruses that have changed their hosts. These enzootic viruses tend to have a severe effect in humans. A viral disease emerges in a population through a series of steps the initial infection, the spillover, and lastly the host to host transfer. These steps are further aided or prevented by the virulence factors present in the virus versus the host or human whichever the case susceptibility. It is important to determine the source of these viruses and whether it was via an enzootic or epizootic virus. The barriers that the virus faces when trying to infect a new host are just as important as the role that the environment plays in the virus's transmissibility. There are many factors to consider when looking at viruses how viruses change hosts.

New viruses can emerge in a population through contact with an alternative host. Until recently the probability of a virus changing hosts was limited by the restricted contact between the initial host and the alternative host. An increase in contact can be accomplished by introducing the host animal to domestication or any other arena that would serve to increase the likelihood of human contact. Primates that have been infected with simian immunodeficiency virus in Africa were separated from areas of high human populations which in turn significantly limited the chances of the virus changing hosts and infecting humans.

The chances of viral contact can also be increased by changes in social and sexual behavior, increased travel, hygiene practices and the increased density of the population that work in favor of the virus and increase the chances of infection in an alternative human host. The significance of initial host to alternative host contact can be examined through the instance in

Africa where primates infected with simian immunodeficiency virus in Africa were removed from areas of high human populations and in turn significantly reduced the number of the host changes from primate to humans. The removal of the infected animal from direct human contact does not prevent transmission though intermediate hosts. In Malaysia fruit bats are the reservoirs for the virus nipah and with the large number fruit orchards near pig farms the incidence of contact between the virus and the pig is greatly increased.

When it comes to a virus’s ability to infect a new host there are new barriers that the virus must learn to penetrate. An important part of a virus’s ability to infect new hosts is its ability to infect that hosts cells. In humans the virus’s can have trouble entering the host via due to factors that fight off viral infections or something as simple as the surface of human skin can pose as a barrier for entry into an alternative host. When galactosyl producing virions which are not normally found in humans are detected in the body the galactosyl brings about an antibody response that inactivates the virus and prevents its spread. A mechanism of action such as this requires the virus’s need to rapidly adapt to bypass the barriers that are set up to prevent viral infection.

Even if the relative distance in relation between the initial host and the alternative host of a virus is close the intensity and rate of the contact between the two species is still a factor. When a virus infects a new host that is distantly or closely related to the previous host it does not mean that the host cannot also transfer the virus to more distantly related organism. Integration of a virus into a new host cell is also dependent on the receptor

binding that occurs between the virus and host cell. The changes that the virus has to undergo in order to infect the new host cells must coincide with the receptors that are found already on the host cells. A process involving the transfer of the FPV virus to infect canine involved a gain of two mutations that then allowed for it to bind to the canine transferrin receptors. These mutations allowed for the FPV virus to increase its host range successfully gain the ability to infect canines with a new form of the FPV virus CPV.

Blockades for the spread of the viral infection once it has infected the new host cells can exist in the form of proteins that prevent the spread of the virus to neighboring cells. The capsid proteins of viruses are stopped at the cytoplasm of the new host cell by TRIM5α a protein that binds to the capsid of the virus preventing its entry into the host cell. Generalist and specialist viruses are two categories for viruses that can possibly predict and help determine the ranges of hosts that a particular virus can infect; and whether or not a virus is a candidate for host switching. Generalist viruses are expected to have an increased incidence of alternative host shifting while specialist viruses are the opposite and are unable to bypass the barriers in the host cells receptors and other defenses that would require the virus to mutant in order to effectively infect the cell. Most of the specialist cells have trouble making it past the initial infection of the alternative host.

Viruses that have a wide range of hosts have a built in advantage already in that they do not have to alter in order to successfully make a change in the types of organism that they can infect. The rate of variation in a virus directly determines the adaptability of a virus into a new host. Viruses that have a high evolving rate are more likely to cross species and cause

infection in a new host due to its ability to quickly adapt to the host cell. RNA viruses do not have proofreading mechanisms as well as replication that is error prone and are in that sense much more variable than DNA viruses. DNA viruses are less variable than RNA viruses but some exception exist in that certain single stranded DNA the rate of variation may be similar to that of RNA viruses.

A reduction in virus fitness occurs when the virus undergoes mutations that are necessary in order to infect a new host. If the virus is using a intermediate host even more adaptations are required and the virus is further reduced in fitness. The addition of the intermediate hosts help to explain why the influenza A virus infects each of its hosts differently through different mechanisms. In humans for example the infection is found in the lower respiratory tract than in other hosts where it is located in the upper respiratory tract. Reassortments and recombinants aid in a virus adaptability to a new host cell by making a number of genetic changes in a shorter amount of time. The CoV virus of the bat in recombination with another virus was able to make a new virus SARS that can infect humans and other hosts.

The intermediate virus is a form of the virus that infects the intermediate host. This virus is the least stable form of the virus. The lower fitted virus loses some of the capability to infect previous parental host types efficiently in addition to the newer crossover hosts they are trying to infect. This phenomenon could account for the low percentage of viral crossover between species.

The article did a good job of following the trend and mechanism with which a virus switches hosts. More investigation should be done in the areas of the initial infection of the virus and how it crosses over. More studies should also be done on the likelihood of a virus from another animal making the host switch to infect humans and how that spread can be predicted and prevented. Further studies should be done on how the viruses that make the jump to a species that is not close in the evolutionary chain to who they normally infect to humans. A broader knowledge of how the virus adapts itself to survive in an organism that is so different from its original host also deserves further investigation. If the topics of interest listed are further studied and developed then the article would have a more focused and concise viewpoint instead of the disorganized and sometimes abandoned thread of thoughts that exist at some points within the article.