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Viruses Follow an Evolutionary Pathway

Urmi Chouhan, Kshipra Pandey and Ritu Patel*

Department of Bioscience, Indrashil University, Mehsana, Gujarat , India

ABSTRACT

Inherently, viruses are parasites that live inside cells. Viruses compete with their hosts to drive their own genomes and evolutionary paths, but they are evolutionarily autonomous despite their dependency on host cells. In at least two dimensions, viruses exhibit an immense diversity that surpasses that of their biological hosts. First, viruses efficiently use every information transmission scheme that is possible with the two types of nucleic acids. In contrast, cellular life forms use a consistent method of genetic information storage and expression with double- stranded (ds) DNA genome transcribed into various RNAs, including mRNAs that are translated into proteins. Secondly, virus gene sequences can span relatively larger parts of the sequence space because they usually evolve at a considerably faster rate than their biological homologs. It can be challenging to identify the deep evolutionary linkages and ancestry of virus genes because a single family of viruses might have sequence diversity greater than that of entire domains of cellular life. Identifying the deep evolutionary linkages and ancestry of virus genes can be extremely difficult. In this review, we will be highlighting the evolutionary history of viruses and their pathways alongside.

***Corresponding author**

Ritu Patel, Department of Bioscience, Indrashil University, Mehsana, Gujarat 382740, India.

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Introduction

Viruses, ubiquitous entities existing at the intersection of life and non-life, exhibit a remarkable capacity for evolutionary adaptation, shaping their genomic architecture in response to selective pressures over time. The study of virus evolution is pivotal not only for comprehending the dynamic interplay between these microorganisms and their hosts but also for devising effective strategies in the face of emerging infectious diseases. This intricate dance of genetic changes, environmental interactions, and host responses propels viruses along complex evolutionary pathways, influencing their transmissibility, virulence, and host range. At the molecular level, viruses undergo rapid genetic diversification driven by mutation, recombination, and reassortment. These mechanisms, coupled with selective forces imposed by host immune responses and antiviral interventions, engender a continuous arms race between viruses and their hosts. Understanding the nuances of these evolutionary trajectories is paramount for predicting the emergence of novel viral strains with altered biological properties, which may pose challenges for public health systems [1]. This exploration delves into the underlying principles that govern the evolutionary dynamics of viruses, shedding light on the mechanisms that drive their adaptive changes. By dissecting the molecular intricacies of viral evolution, we aim to unravel the enigma surrounding the emergence of new viral variants and to pave the way for proactive strategies aimed at mitigating the impact of evolving viral threats on global health.

Evolutionary Biology of Viruses

The evolutionary biology of viruses stands as a captivating realm within the broader tapestry of life's dynamic processes. Viruses, despite their simple structure and lack of cellular autonomy, exhibit an unparalleled ability to navigate the intricate pathways of adaptation and evolution. At the heart of this evolutionary odyssey lies the genetic malleability of viral genomes, perpetuated by mechanisms such as mutation, recombination, and reassortment. Mutation, the primary engine of genetic diversity, empowers viruses to explore vast sequence space, affording them the plasticity necessary for rapid adaptation. Concurrently, recombination and reassortment events, often spurred by interactions between different viral strains or host species, foster the exchange of genetic material, generating novel genomic configurations with the potential to confer advantageous traits. The selective pressures exerted by host immune responses and antiviral interventions orchestrate a perpetual tug-of-war between viruses and their hosts. This selective landscape drives the fixation of beneficial mutations and the culling of deleterious ones, sculpting viral populations in response to the ever-changing biological milieu [2]. The evolutionary biology of viruses assumes particular significance in the context of emerging infectious diseases, where understanding the forces shaping viral genomes becomes paramount for predicting and managing outbreaks. The complex interplay between viral evolution and host dynamics underscores the need for a holistic approach to studying the ecological and genetic factors influencing the trajectory of viral adaptation. By delving into the evolutionary intricacies of viruses, researchers not only unravel the mysteries of their origin and diversification but also gain insights critical for devising effective preventive and therapeutic strategies [3].

Diversification based on Host Evolution

The diversification of viruses constitutes a global challenge. Over the past decade, empirical evidence has substantiated the assertion that viruses stand as the most abundant biological entities ubiquitously distributed across the Earth. Profound reservoirs of viral-like particles (VLPs) are discernible in both marine and terrestrial environments, predominantly mirroring the morphology of tailed DNA viruses prevalent in bacterial populations. Furthermore, certain environmental viruses exhibit unexpected dimensions and intricacy, exemplified by phycodnaviruses infecting algae and mimivirus affecting amoeba- a remarkable 1.2 megabase (Mb) DNA virus capable of encoding nearly 1000 genes [4]. Consequently, viruses manifest as an extensive and heterogeneous repository of untapped genetic diversity. Despite this wealth of genomic resources, the evolutionary dynamics governing this viral population and its consequential impact on host organisms remain inadequately elucidated. It is plausible that the expansive reservoir of viral genes exerts an influence on host evolution, given the well- documented colonization of prokaryotic genomes by prophages. Consequently, this expansive viral gene pool establishes direct connections with prokaryotes, intertwining with the broader framework of the 'tree of life.' The investigation into virus evolution now stands as an integral extension of the overarching paradigm of biological evolution [5].

An Evolutionary History of Viruses

Primarily, the evolutionary trajectory of viruses adheres to the foundational Darwinian principles shared with host evolution, encompassing mechanisms of variation and natural selection. Nevertheless, viruses exhibit a polyphyletic nature due to their diverse origins. Classifiable into six major categories (+RNA, –RNA, dsRNA, retro, small DNA, large DNA), these viruses lack common genes, indicating their absence of a shared ancestor. However, within these categories, conserved hallmark genes (e.g., capsid proteins, Rd RNA pol, RT, primase, helicase, and RCR initiation proteins) form monophyletic clusters, potentially tracing back to primordial host/viral gene pools.

Notably, certain DNA viruses display replication strategies and polymerases distinct from those of their hosts, relying on these hallmark genes. This implies an ancient yet intricately linked evolution between viruses and their hosts. In contrast to host evolution, viral quasispecies showcases a population-based adaptability, expanding the concept of natural selection to encompass populations with otherwise unfit genomes. Viruses can transcend typical host- species barriers, fostering reticulated evolution in extensive gene pools. Recombination at high rates is observed among bacterial DNA viruses and +RNA viruses infecting diverse host species. Furthermore, viruses defy conventional notions of death and extinction by reconstructing genomes from fragments and rectifying lethal damage through multiplicity reactivation. Damaged (defective) viruses play a role in both virus and host evolution, with such defects identified in various virus types and host genomes (e.g., defective prophage or defective endogenous retroviruses). The impact of defective viruses on host survival is evident, thereby expanding upon and challenging the Darwinian principles governing host evolution. The systematic exploration of virus evolution necessitated the advent of sequencing technologies, enabling the quantification of mutations and genetic variations within viral populations [6]. The foundational principles of natural selection, fitness, and the propagation of advantageous variations were well-established in evolutionary biology literature well before the maturation of virology. Consequently, mathematical models such as Fisher population genetics, concerned with gene frequency in sexually reproducing species populations, emerged as directly applicable to virus evolution, mirroring the evolutionary dynamics of host genes [7]. Viral fitness was commonly delineated through relative replication rates (replicative fitness), occasionally incorporating considerations of host virulence and disease [8]. Despite these efforts, a comprehensive definition of viral fitness remains elusive, as expounded below.

In the 1940s, pioneering quantitative assessments of virus mutation rates were conducted, notably with bacterial phage. These mutation rates were articulated through sets of ordinary differential equations, forming the basis for subsequent development of quasispecies equations applied to error-prone RNA genomes. However, the conceptualization of a virus species poses a challenge to evolutionary paradigms and prompts a re-evaluation of how kinship in viruses is defined. Unlike host species defined by sexual reproduction, a virus species is presently defined as a polythetic class—an assemblage of related components with non-universal shared elements (e.g., host range, genome relatedness, antigenic properties). Lacking a specific defining characteristic or gene, a virus species does not necessitate sexual exchange for classification. This definition, akin to delineating a 'heap,' is inherently fuzzy, unmistakable yet resistant to precise specification by a fixed number of components. Molecular characterizations of numerous virus populations align with this inherently fuzzy species definition. The ensuing challenge involves deciphering viral evolutionary patterns within the context of these ambiguous definitions [9]. Nevertheless, discernible patterns in virus evolution persist, suggesting an ancient lineage possibly extending into the primordial RNA world.

Replication Prone to Errors and the Concept of Quasispecies

During the 1970s, Manfred Eigen and Peter Schuster formulated a foundational theoretical model elucidating virus evolution. They introduced a set of ordinary differential equations, defining what they termed 'quasispecies.' These equations, derived from measurements of phage mutation rates, delved into the consequences of high error rates inherent in RNA replication—a process characterized by error-prone, non-correcting mechanisms. The resulting population, termed quasispecies, represented a community of individuals arising from error- laden replication. It's important to note that the term 'quasispecies' describes a chemically diverse set of molecules and was not initially intended to connote a biological species involving genetic exchange. However, as discussed earlier, the ambiguous definition of virus species and quasispecies exhibits some overlap, contributing to conceptual confusion.

Several foundational premises underpinned the development of this theory

(1) individual products within the population are considered independently, interacting solely as individuals

(2) the system operates away from equilibrium, with resources not posing limitations.

Replicative fitness, providing a mathematical definition based on relative replication, described the growth of favorable types. The original equations represented an idealized, generalized system of infinite population size and aren't directly applicable to the real world. Nevertheless, they offer valuable insights into real-world systems, although they do not account for variable mortality, interference, exclusion, competition, complementation, and persistence, and their impact on nonreplicative fitness definitions [10].

Mortality and fitness present intriguing aspects in the context of viruses. For instance, an interfering defective virus might be considered nonviable but can disrupt and lead to the extinction of the wild-type template replication within the quasispecies. In some instances, the quasispecies equations seemingly align mathematically with classical Wright and Fisher population genetics equations, as applied by Kimura and Maruyama to asexual haploid populations at the mutation-selection balance. However, these approaches originate from distinct perspectives, and the assumption of high error rates in the quasispecies equations significantly influenced experimentation and our current comprehension of virus evolution. This has yielded counterintuitive conclusions, such as the selection of 'the fittest' compared to the consensus character of the master template.

Quasispecies from viruses with high error rates, like HIV-1, might comprise all mutant progeny RNAs, rendering the consensus template (mean, fittest, or master template) potentially nonexistent. In classical population genetics, an asexual clonal population would be expected to fix the clonal sequence, a phenomenon not observed in quasispecies. The initial laboratory measurements of viral quasispecies were conducted using Qβ RNA polymerase in vitro, estimating error rates ranging from 10^(-3) to 10^(-4) substitutions per site per year— applicable to most RNA viruses. Qβ replication generated a characteristic genetic spectrum through the replication of numerous nonviable mutants. Additional laboratory measurements have demonstrated that quasispecies can exhibit significant adaptive fitness beyond the cloned master template and display memory, retaining information from prior selections in a minority of the population. Moreover, complementation, interference, competition suppression, and extinction have been observed in various quasispecies, challenging the original assumption of genome independence in these equations. Despite these revelations, the concept of quasispecies remains highly valuable, not solely as a theoretical construct. For instance, the live poliovirus vaccine exemplifies a heterogeneous quasispecies, harboring a minority of neurovirulent variants suppressed by the majority of avirulent viruses [11]. A key aspect of the quasispecies concept is its ability to elucidate high adaptability within a population containing numerous, even lethal, mutations. Intriguingly, this aligns with the proposed early RNA world, suggesting a collective quasispecies environment.

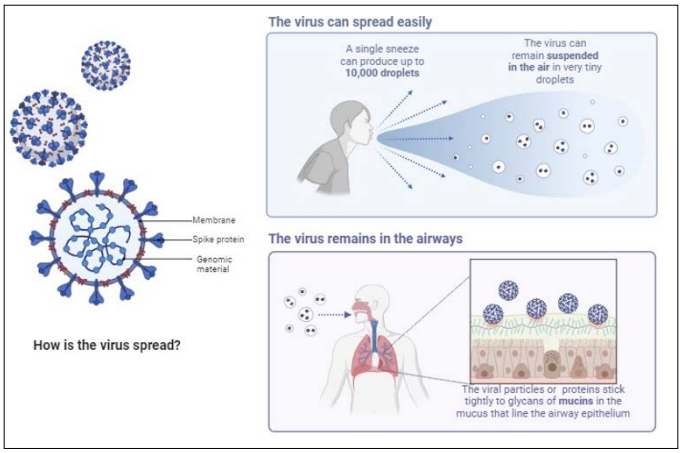

Figure 1: Elaboration of Viral Spread

Perpetual Adaptation

A tangible illustration of the potentially unceasing adaptation of viruses is depicted in Figure 2. The monitoring of the HA and NA genes of the human influenza A virus over several decades reveals a dynamic scenario. The predominant master template of the virus within the human population undergoes continual alterations, driven by immune selection and stochastic viral immigration. This ongoing evolution necessitates annual adjustments to the vaccine composition, as illustrated in the figure. Although this population dynamic has been consistently maintained in the human populace, all previous iterations essentially vanish without reemergence. It's noteworthy that not all RNA virus populations exhibit this continuous change or the anticipated diversity associated with quasispecies.

Even within influenza virus A, certain avian isolates from their natural hosts (waterfowl) display genetic stability. Some RNA and retroviruses, despite possessing high error rates, can uphold stable populations within specific hosts. For instance, the measles virus demonstrates much less antigenic drift in human infections compared to influenza virus A. The Hepatitis G virus, a prevalent human virus and a distant relative of HCV, exhibits minimal variation even within isolated human populations. Filoviruses such as Ebola virus and Marburg virus reveal no genetic variation in Zaire isolates spanning two decades. Similarly, the Hendra virus isolated from Australian fruit bats and the Nipah virus from Malaysian fruit bats exhibit limited genetic diversity. Arenaviruses and hantaviruses also maintain genetic stability within their natural rodent hosts. The underlying reasons for such population stability have yet to be comprehensively assessed [12]. Purifying selection seems likely in some cases, such as measles, while in others, factors like persistence and low replication rates appear to be influential. For example, simian foamy virus (SFV) and human T-lymphotropic virus II (HTLV- II) exhibit a generation of only about 10^(-8) substitutions per site per year, largely attributed to their low replication rates.

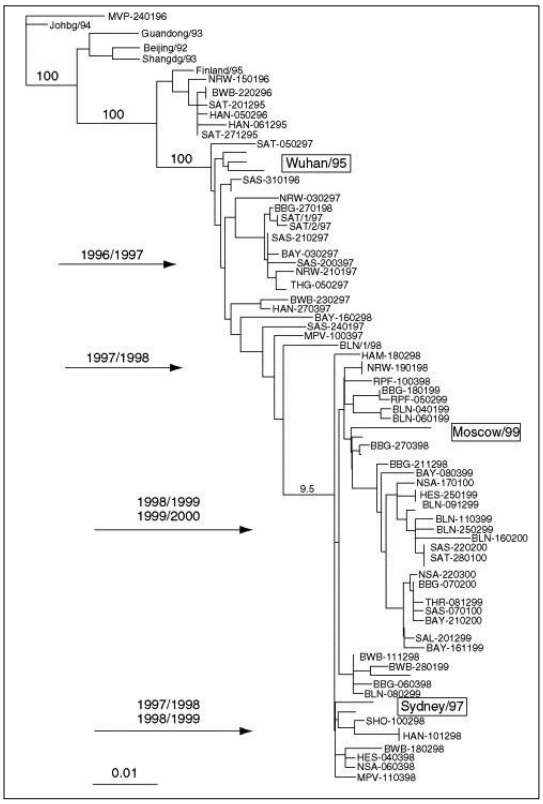

Figure 2: The Phylogenetic Tree Depicting Annual Variations in Influenza A/H3N2 Viruses, based on the Hemagglutinin Gene, is provided. The Locations of Designated Vaccine Strains are Highlighted within the Tree for clarity [13].

Correspondence of Virus-Host Interactions and RNA Resilience

Numerous instances now demonstrate coevolution between RNA viruses and their host species, indicating notably gradual rates of virus evolution. This observation poses a challenge to the quasispecies theory, as the congruence in error rates seems incongruent. For instance, the coevolution of Hantavirus (genus Bunyavirus) with its rodent host implies an enduring association spanning approximately 20 million years [14]. Similarly, Arenaviruses (single- stranded RNA bisegmented ambisense) exhibit coevolution patterns in both Old and New World rodents. Of particular significance is the potential for these viruses to serve as the origin of five hemorrhagic human fevers, including the notorious Lassa virus. In these instances, it becomes evident that the virus establishes a persistent, asymptomatic infection within its natural host, with human disease arising from a species jump.

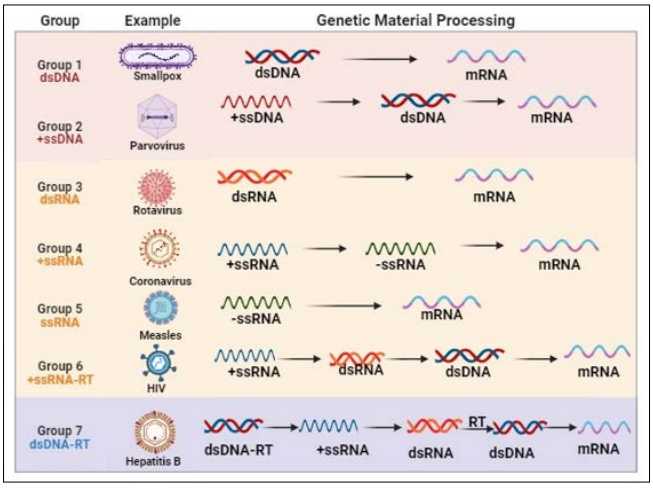

Figure 3: Classification of Viruses

Evolutionary Patterns in RNA Viruses

The occasionally pronounced divergence observed in RNA virus sequences has prompted some to posit that most family lineages seem to be approximately 10,000 years old, a proposition diverging from older estimations. Notably, +RNA viruses exhibit a striking diversity in genomes and replicator mechanisms, with substantial evidence of recombination and a proclivity for crossing host barriers. Approximately 38 families of +RNA viruses, often characterized by up to four segments, are recognized. Four distinct replicase classes, sharing a common genetic plan, exist in these viruses, featuring three helicase superfamilies, two protease superfamilies, and two jelly roll capsid domains. Generally, capsid and RdRpol sequences are congruent, barring exceptions like the Luteoviridae and Tetraviridae families, which appear to have undergone recombination between these two gene lineages. The smallest+RNA virus identified belongs to the Leviviridae bacterial virus family, encompassing only four genes, reflecting a seemingly ancestral +RNA virus. Notably, no RNA virus has been discovered to infect archaebacteria to date. In contrast, the largest +RNA viruses belong to the Coronavirus genus, with genomes ranging from 27 to 32 kilobase pairs. Recently described+RNA viruses in the Marnaviridae family, infecting bats and marine organisms, appear to be foundational to the evolution of picornaviruses [15]. Despite the potential for high variability, certain +RNA virus populations can exhibit stability. For instance, the dengue virus (Flaviviridae) demonstrates low rates of amino acid substitution, attributable to strong selective constraints likely stemming from acute arbovirus infection, high error rates, and involvement of multiple tissues and vector transmission. Negative-strand RNA viruses exhibit distinct evolutionary patterns traced through their polymerase genes, with highly conserved gene orders. Unsegmented viruses, such as rhabdoviruses, lyssaviruses, and paramyxoviruses, show minimal recombination and tend to vary through point mutations and deletions. Despite the potential for high error rates and quasispecies generation in laboratory settings, natural isolates, including lyssaviruses and measles virus, tend to exhibit relative homogeneity. For instance, lyssaviruses display a slow evolutionary rate (5 × 10^(-5) substitutions/site/year), possibly influenced by persistent infections in their natural hosts. Conversely, the measles virus, strictly human-specific and acute, likely maintains stability through purifying selection [16].

Trends I Evolutionary Dynamics of DNA Viruses: Tailed Bacteriophages

The extensive DNA viruses infecting bacteria, archaea, and eukaryotes exhibit evolutionary connections. Despite limited sequence conservation between bacterial T4 phage, archaeal halophages, algal-infecting Phycodnaviridae, and vertebrate eukaryotic herpesviruses, similarities in gene programs, DNA polymerase types, capsid structures, and capsid assembly suggest a common ancestor. For instance, both the Enterobacteria phage T4 and herpes simplex virus 1 (HSV-1) display T = 1 capsid symmetry with 60 copies of capsid protein. While bacterial DNA viruses likely represent the ancestral lineage, the origins of these phages seem obscured within the primordial gene pool. These DNA viruses often possess large genomes, unsustainable by error-prone replication, prompting the adoption of error-correcting DNA replication with error rates approximating those of host cells (10^(-8)). Characterizations of giant bacterial phage genomes (e.g., Bacillus megaterium phage G with around 600 genes) and algal phycodnaviruses have been achieved. Even larger DNA viruses infecting amoeba, such as the Acanthamoeba polyphaga mimivirus, coding for over 1000 genes, are abundant in certain water habitats.

The tailed bacterial phages, constituting a vast genetic reservoir (~10^31 in total), have been termed the "dark matter of genetics," given their numerical dominance, equivalent to approximately 10^24 productive infections per second globally [17]. Host-restricted phage lineages typically conserve core proteins, especially capsid genes, while broader T-even phages may not conserve any hallmark genes. Hallmark genes, when present, are often identified through conserved domains within proteins, such as replication and structural proteins, with replicator strategy and gene order frequently conserved. Phages also tend to retain genes active against other phages, such as those involved in DNA modification, lambda RexA, and T4 rII. Although phage genomes often evolve as mosaics, incorporating sharp boundaries between genes and protein domains, recombination plays a pivotal role in generating gene and subgene domain variation. Current sequencing efforts have revealed 350 full genomes of tailed phages and 400 prophages from bacterial genomes. Comparative genomics, particularly in lactobacterial phages, indicates that most phage genomes evolve as mosaics, with sharp boundaries between genes and protein domains. Recombination between lytic, temperate, and cryptic prophages contributes to this variation. While some phages employ specific mechanisms for generating gene diversity, such as bordetella phages using reverse transcription (RT) for surface receptor diversity, most diversity arises from recombination [18]. Two broad patterns of phage variation have been observed, corresponding to host-unassociated lytic and host-associated (congruent) temperate phages. In various bacterial genomes, including Escherichia coli (ECOR collection), cyanobacteria, and Bacillus subtilis, prophage colonization patterns significantly contribute to genetic distinctions among closely related host

strains. The prevailing understanding for tailed bacterial phages suggests that they are not products of host genome reduction.

Considering the Emergence of Coronavirus through Evolution

To gain a more profound understanding of evolution, one can investigate the latest historical record, exemplified by the global pandemic, the coronavirus outbreak. Three major outbreaks of the coronavirus, a zoonotic virus known to cause respiratory disease, have been reported since 2002, including SARS-CoV, MERS-CoV, and the most recent 2019-nCoV, or more recently known as SARS-CoV-2. Bats are known to be the primary animal reservoir for coronaviruses [19]. However, in the past few decades, the virus has been able to mutate and adapt to infect humans, resulting in an animal-to-human species barrier jump. The emergence of a novel coronavirus poses a serious global public health threat and possibly carries the potential of causing a major pandemic outbreak in the naïve human population. The recent outbreak of COVID-19, the disease caused by SARS-CoV-2, in Wuhan, Hubei Province, China has infected over 36.5 million individuals and claimed over one million lives worldwide, as of 8 October 2020. The novel virus is rapidly spreading across China and has been transmitted to 213 other countries/territories across the globe. Researchers have reported that the virus is constantly evolving and spreading through asymptomatic carriers, further suggesting a high global health threat [20].

Coronaviruses derive their name from the Latin word "corona," signifying crown or halo, referencing the distinctive crown-like spikes observable on their surface through electron microscopy. These enveloped viruses carry a non-segmented, single-stranded, positive-sense RNA genome, approximately 32 kilobases in size, representing the largest known genome among RNA viruses [21]. Classified under the subfamily Coronavirinae within the Coronaviridae family, belonging to the order Nidovirales, coronaviruses encompass four genera: Alphacoronavirus, Betacoronavirus, Deltacoronavirus, and Gammacoronavirus. The SARS-CoV-2 strain falls within the Betacoronavirus genus, as determined by genome sequence analysis [22]. Notably, the coronavirus genome possesses a 5' cap and a 3' poly(A) tail, facilitating its function as an mRNA upon infecting the host cell for the translation of replicase polyproteins crucial for viral replication [23].

These viruses are primarily harbored in animal reservoirs, such as bats, mice, rats, chickens, dogs, cats, horses, and camels [24]. Recently, coronaviruses have demonstrated the capacity to initiate epidemics by adapting to humans through zoonotic transmission, akin to the Zika virus outbreak in 2015 [25, 26]. Bats, identified as the primary carriers and reservoirs for various viruses, including coronaviruses, present a heightened risk of species barrier crossing due to their significant congregations and long-distance travel capabilities [27]. Human coronaviruses were initially identified in the 1960s, and to date, seven distinct strains have been reported. Among these, the common strains-229E, NL63, OC43, and HKU1—typically cause mild respiratory tract infections globally. It is noteworthy that animal-origin coronaviruses may undergo evolution and adaptation to infect humans, leading to the emergence of novel viruses and the potential for pandemic outbreaks. Examples include SARS-CoV, MERS-CoV, and the more recent SARS-CoV-2, all crossing the animal-to-human species barrier and causing more severe symptoms in affected individuals [28]. In general, RNA viruses, exemplified by coronaviruses, influenza viruses, and HIV, exhibit exceptionally high mutation rates attributed to their replication mechanisms and the absence of proofreading activity in viral RNA polymerase. Mutations, serving as fundamental units of evolution, enable natural selection for traits advantageous to the virus, including heightened virulence, adaptability, and evolvability [29]. According to a phylogenetic investigation by Lu et al. SARS-CoV-2 is believed to have crossed the species barrier from bats at the Huanan South China Seafood Market in Wuhan, Hubei Province, China [30]. The study highlighted a higher genomic sequence similarity between SARS-CoV-2 and the SARS-like bat coronaviruses RaTG13 (96.2% identity) from bats in Yunnan, compared to SARS-CoV (79%) or MERS-CoV (51.8%), implicating bats as the primary source of SARS-CoV-2 (refer to Table 2). Due to the diverse range of animals traded at the Huanan Seafood Market, researchers propose that an intermediary animal, such as snakes, pangolins, birds, or other mammals, might have played a role in facilitating the emergence of the ongoing COVID-19 outbreak.

A recent investigation by Tang et al. proposed that SARS-CoV-2 is undergoing continuous evolution [31]. The study, comparing the genomes isolated from 100 patients, revealed 149 mutation sites, with two SNPs displaying strong linkage and suggesting two distinct SARS-CoV-2 subtypes. The first SNP, identified at 8782 (orf1ab: T8517C, synonymous), and the second at 28,144 (ORF8: C251T, non-synonymous—S84L) categorized the genomes into S or L types based on the second SNP. Genomic alignment with related viruses indicated the S type as the ancestral form, while the L type was more prevalent, particularly in Wuhan.

The coronavirus spike protein, a pivotal viral surface protein governing attachment and host cell entry, stands as a compelling target for evolutionary investigations due to its role in influencing host selectivity, susceptibility, and viral infectivity [32,33]. Additionally, as the primary target of the host immune system, the spike protein undergoes rapid molecular evolution and selective pressure. A study by Zhang et al. highlighted the emergence of the D614G mutation in the SARS-CoV-2 spike protein, resulting in reduced S1 shedding and increased infectivity [34]. Further research on the evolutionary dynamics of the SARS-CoV-2 spike protein through epidemiological surveillance is recommended to elucidate the association between SARS-CoV-2 mutations and virulence.

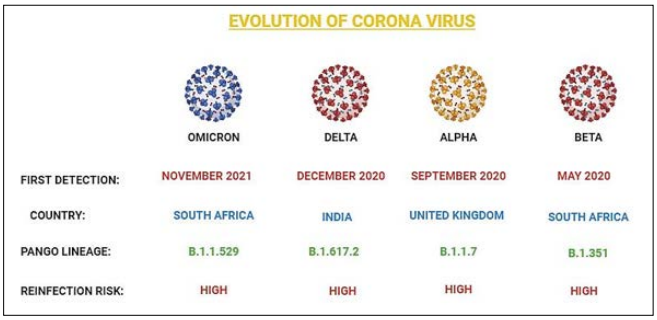

Figure 4: Evolution of Coronavirus

Conclusion

A persistent challenge in the realm of virus evolution is comprehending the origins of novel viral pathogens. The unpredictable and stochastic nature of such adaptations that lead to increased virulence poses difficulties in making accurate predictions, given the ambiguous connection between virulence and evolutionary processes. For instance, foreseeing the genetic alterations that transformed the SARS virus, persisting in bat populations, into an acute human pathogen remains elusive. The understanding of viral fitness, selection, and how these factors

evolve from persistent states to acute species jumps is yet to be fully articulated. Despite this, several variables contribute to the likelihood of viral emergence, with virus ecology playing a pivotal role. Considerations such as the population density and dynamics of the new host, as well as the ecological interactions between emerging and established viral hosts, often prove critical. Additionally, interactions between viruses themselves can play a crucial role, facilitating recombination and/or reassortment or diminishing immunological selective barriers through immunosuppression. The emergence of HIV-1 from various persistent SIVs in African monkeys, passing through chimpanzees to cause a new human disease, exemplifies the complexities involved. Similarly, the potential emergence of pandemic human influenza from avian (Anatiformes) sources, such as H5N1, remains a significant concern. Consequently, the field of virus evolution continues to captivate researchers as they strive to predict, control, and ultimately eliminate viral agents of disease.

References

1. Nahmias AJ, Reanney DC (1977) The evolution of viruses. Annual Review of Ecology and Systematics 8: 29-49.
2. Holland J, Domingo E (1998) Origin and evolution of viruses. Virus genes 16: 13-21.
3. Villarreal LP (2008) The widespread evolutionary significance of viruses. Origin and Evolution of Viruses 477-516.
4. Lauring AS (2020) Within-host viral diversity: a window into viral evolution. Annual Review of Virology 7: 63-81.
5. Castel G, Razzauti M, Jousselin E, Kergoat GJ, Cosson JF (2014) Changes in diversification patterns and signatures of selection during the evolution of murinae-associated hantaviruses. Viruses 6: 1112-1134.
6. Pavesi A (2021) Origin, evolution and stability of overlapping genes in viruses: a systematic review. Genes 12: 809.
7. Goldhill DH, Turner PE (2014) The evolution of life history trade-offs in viruses. Current opinion in virology 8: 79-84.
8. Villarreal LP (2008) Evolution of viruses. Encyclopedia of virology pp: 174-184.
9. Shankarappa RAJ, Margolick JB, Gange SJ, Rodrigo AG, Upchurch D, et al. (1999) Consistent viral evolutionary changes associated with the progression of human immunodeficiency virus type 1 infection. Journal of virology 73: 10489-10502.
10. Domingo E, Perales C (2018) Quasispecies and virus. European Biophysics Journal 47: 443-457.
11. Andino R, Domingo E (2015) Viral Quasispecies. Virology 479: 46-51.
12. De Filippis VR, Villarreal LP (2000) An introduction to the evolutionary ecology of viruses. Viral ecology pp: 125-208.
13. Schweiger B, Zadow I, Heckler R (2002) Antigenic drift and variability of influenza viruses. Medical microbiology and immunology 191: 133-138.
14. Brinkmeyer Langford C, Amstalden K, Konganti K, Hillhouse A, Lawley K, et al. (2021) Resilience in Long-Term Viral Infection: Genetic Determinants and Interactions. International Journal of Molecular Sciences 22: 11379.
15. Greenbaum BD, Levine AJ, Bhanot G, Rabadan R (2008) Patterns of evolution and host gene mimicry in influenza and other RNA viruses. PLoS pathogens 4: e1000079.
16. Reanney DC (1982) The evolution of RNA viruses. Annual Reviews in Microbiology 36: 47-73.
17. Hendrix RW, Hatfull GF, Smith MC (2003) Bacteriophages with tails: chasing their origins and evolution. Research in microbiology 154: 253-257.
18. De Souza RF, Iyer LM, Aravind L (2010) Diversity and evolution of chromatin proteins encoded by DNA viruses. Biochimica et Biophysica Acta (BBA)-Gene Regulatory Mechanisms 1799: 302-318.
19. Pyrc K, Berkhout B, Van der Hoek L (2007) The novel human coronaviruses NL63 and HKU1. Journal of virology 81: 3051-3057.
20. Patel RB, Patel BB (2022) An analysis of the COVID-19 situation in India in terms of testing, treatment, vaccine acceptance and National Economic Performance. International Journal of Public Health 67: 1604975.
21. Smith EC, Denison MR (2013) Coronaviruses as DNA wannabes: a new model for the regulation of RNA virus replication fidelity. PLoS pathogens 9: e1003760.
22. Weiss SR, Navas-Martin S (2005) Coronavirus pathogenesis and the emerging pathogen severe acute respiratory syndrome coronavirus. Microbiology and molecular biology reviews 69: 635- 664.
23. Sawicki SG, Sawicki DL, Siddell SG (2007) A contemporary view of coronavirus transcription. Journal of virology 81: 20-29.
24. Van Der Hoek, Pyrc L, Jebbink K, Vermeulen-Oost MF, Berkhout W, et al. (2004) Identification of a new human coronavirus. Nature medicine 10: 368-373.
25. Sharma A, Lal SK (2017) Zika virus: transmission, detection, control, and prevention. Frontiers in microbiology 8: 110.
26. Sui J, Aird DR, Tamin A, Murakami A, Yan M, et al. (2008) Broadening of neutralization activity to directly block a dominant antibody-driven SARS-coronavirus evolution pathway. PLoS pathogens 4: e1000197
27. Kahn JS, McIntosh K (2005) History and recent advances in coronavirus discovery. The Pediatric infectious disease journal 24: S223-S227.
28. Chowell G, Blumberg S, Simonsen L, Miller MA, Viboud C (2014) Synthesizing data and models for the spread of MERS-CoV, 2013: key role of index cases and hospital transmission. Epidemics 9: 40-51.
29. Duffy S (2018) Why are RNA virus mutation rates so damn high? PLoS biology 16: e3000003.
30. To KK, Hung IF, Chan JF, Yuen KY (2013) From SARS coronavirus to novel animal and human coronaviruses. Journal of thoracic disease 5: S103.
31. Tang X, Wu C, Li X, Song Y, Yao X (2020) On the origin and continuing evolution of SARS-CoV-2. National science review 7: 1012- 1023.
32. Li F (2016) Structure, function, and evolution of coronavirus spike proteins. Annual review of virology 3: 237-261.
33. De Haan CA, Te Lintelo E, Li Z, Raaben M, Wurdinger T, et al. (2006) Cooperative involvement of the S1 and S2 subunits of the murine coronavirus spike protein in receptor binding and extended host range. Journal of virology 80: 10909-10918.
34. Zhang L, Jackson CB, Mou H, Ojha A, Rangarajan ES, et al. (2020) The D614G mutation in the SARS-CoV-2 spike protein reduces S1 shedding and increases infectivity. Bio Rxiv https://doi.org/10.1101%2F2020.06.12.148726.

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