MEDWIN PUBLISHERS
Committed to Create Value for Researchers

Anthropology and Ethnology Open Access Journal
ISSN: 2639-2119

Coevolution and Adaptation of Viral Infections, Susceptible Populations, Evolution of Disease and Variant Creation Related to SARS-CoV-2

Caldararo N*

Department of Anthropology, San Francisco State University, USA

***Corresponding author:** Niccolo Caldararo, Ph.D, Department of Anthropology, San Francisco State University, Department of Biology, City College of San Francisco, USA, Email: cald@sfsu.edu

Review Article
Volume 6 Issue 1
Received Date: February 10, 2023
Published Date: April 04, 2023
DOI: 10.23880/aeoaj-16000201

Abstract

Covid-19 has created a major health challenge as a pandemic and demonstrates the threats that human populations face with increased population numbers and density of living situations. Pathogen adaptations to the human immune system are a treat to social complexity and evolution. Present theory argues that human social complexity has advanced in population density due to epidemiologic transition. Globalization and increase human needs for resources with increased density may be reaching limits of the potential for further transitions. The spread of the disease has resulted from mutations making the virus more easily transmitted. Analysis of the viral genome also shows substantial recombination among viral species, quasi-species and variants since an evolutionary event some 5,000 years ago that created several coronavirus groups. Rapid evolution due to recombination and purifying selection of the viral genome, pose a substantial difficulty in producing vaccines and methods of preventing transmission without onerous social distancing and other practices. Typical aspects of the natural history of pathogen infection in mammalian populations that limit the spread, duration and severity of disease (immunity, etc.) appear to be undermined by these features of viral mutation and gene exchange. Breakthrough infections, reinfection after disease and viral escape of vaccines due to evolution of mutations, indicate the possibility of endemic coronavirus in human populations.

Keywords: Covid-19; Coronaviruses; Recombination; Mutation; Immunity

Introduction

Theory in anthropology utilizes modifications of Omran AR [1] model of epidemiologic transitions [2]. In this view human health and disease and economic development (technological and structural) are mediated where high burden fatality of infectious disease, e.g. childhood diseases, viral and bacterial are replaced by non-communicable diseases. This view is in contradiction to some theories in medical science where early human disease burden was characterized by chronic illnesses of mainly non-communicable nature [3]. Medical anthropologists questioned the theory based on the lack of evidence of pre-Neolithic populations [4]. A number of viruses have appeared in the 20th century that challenged the future of dense urban living; these include Influenza, Polio, HIV and Covid-19. One generally attacked individuals of reproductive age, a second, attacked children and individuals of reproductive age, the third adapted to the human immune system causing a general destruction of its capacity to respond and the last

preferentially attacked those over 55 years [5,6].

The natural history of pathogens in animal populations depends on the capability of the virus, bacteria or other agent to infect, developing a means of utilizing the organic material of the host for its own survival and propagation of its kind. It also depends on the physiology and biochemistry of the host, especially its ability to prevent infection or to fight off one once begun. A successful infection in one host can lead to that of other susceptible hosts until the population of those susceptible is exhausted. Here the outcome can be varied, the infection could lead to host death, which would leave the pathogen with no long term opportunities, or to chronic infection as the host fails to develop immunity to dislodge the pathogen. Infection may be temporary, lasting until immunity develops and yet allowing for the pathogen to produce multiple copies of itself and pass on to other hosts.

Successful infection of a host and the production of copies of the pathogen allow for the spread of a disease to susceptible hosts in a population. Often the time of such multiplication depends on the immune system of the host and its behavior and how fast the pathogen can reproduce itself. If it fails to achieve sufficient spread in the host population it will die off unless it has a reserve ability, as in some pathogens if it can survive in some hosts as a latent presence, not producing disease in the host (or a very limited one) and not capable of infecting other hosts as in tuberculosis. For example, some genetic variations in cell responses to virus infection support higher numbers of virus replication and survival in epithelial tissue allowing the rhinovirus that causes the common cold to more easily infect some individuals than others [7]. Some pathogens can use one host as a temporary location for development of stages of its natural history and be passed on to other hosts, often of different species [8].

Yearly appearance of flu and the common cold represent endemic infections that require a reserve host (with some flu viruses, pigs or birds) and each year results in not only new strains of the virus that has been produced by mutations, but new individuals of host populations and loss of immunity by most. While rhinoviruses are often the agents causing the common cold, coronavirus variants (often called serotypes) are also causal pathogens of the cold [9]. In 2017 Wong and his associates [10], published a study showing variation in strains of coronaviruses in their receptor-binding apparatus. Jones S, et al. [11] in studying 9 years of flu virus in India found that strain variation produced more efficient adaptations to human immunity, a clear example of pathogen evolution.

Viral Genome, Recombination and Mutation

When the Covid-19 (SARS-CoV-2) genome and physiology have been examined, the spike S protein has been the major focus of attention. Changes in this protein have allowed the virus to gain access to host cells and to evade host immune responses and, with recent variants like Delta, vaccine design. The Covid-19 genome shows extensive adaptability to mammalian cells and a propensity for recombination across host species boundaries [12] and evidence of purifying selection (mutation driven by selection pressures) (Figure 1).

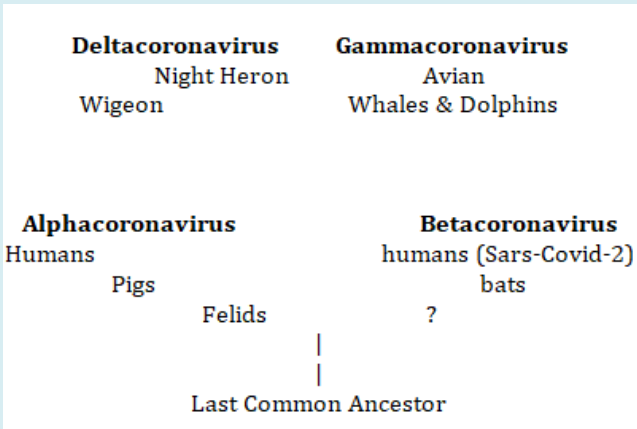

Figure 1: Derived from Devika S, et al. [12] separation of 4 groups dates to 2400 to 3000 B.C.E.

Viral recombination has taken place between strains and led to exchange of genetic material that over time has produced the Covid-19 strain that is quite infective and pathogenic. Of the two types of mutations that can take place, non-synonymous and synonymous, the former change the sequence and type of amino acid used in the assembly of a functional protein. The latter, the synonymous mutation simply substitutes an amino acid that results in the usual protein product. In Covid-19, there is a constant editing process that corrects non-synonymous mutations. While mutation rates can usually be determined by comparing sequences of DNA or RNA or protein structure of amino acids, in the coronaviruses, due to the substantial recombination (blocks of recombined genes) and number of recombination events in the past, evolutionary rates of change have been obscured [13].

Natural History of Viral Infection and Disease

We are experiencing an unusual situation with the Covid-19 pandemic. While most viral infections will increase in a population rapidly, the reproduction rate (r) will vary with the means of transmission and opportunity, attacking all susceptible individuals until it exhausts these hosts. Covid-19 has a more sinister strategy. We have seen in the past virulent viral disease change over time, and variants

be produced as in Polio and HIV [14-16]. Covid-19 has gone beyond the usual mechanism of mutational energy as outlined above. It is known that disease infection in populations can suppress other pathogen spread and disease expression, as during the First World War when measles was suppressed but not diphtheria (caused by a bacillus) in the Australian population (Figure 2), perhaps due to the virulent flu of the time. Virus-virus interactions have long been suspected to affect transmission and spread, but only recently has some definite evidence developed [17]. Wave-like interactions result as diseases affect human populations, often due to changes in human behavior, climate or disease interactions.

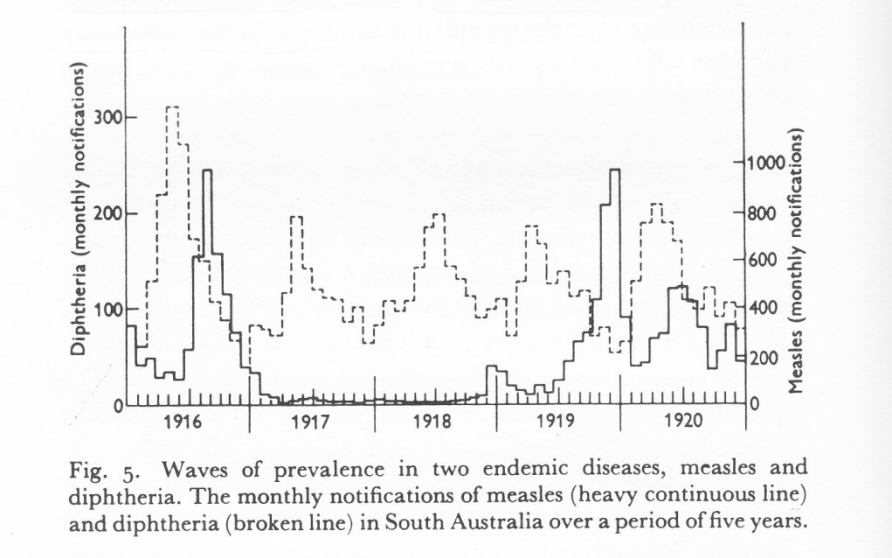

Fig. 5. Waves of prevalence in two endemic diseases, measles and diphtheria. The monthly notifications of measles (heavy continuous line) and diphtheria (broken line) in South Australia over a period of five years.

Figure 2: From Burnet M [14] showing the wave-like interaction of endemic measles and diphtheria over time during the First World War.

We have seen a similar reduction of flu cases in the Northern Hemisphere as well as a number of other respiratory diseases, but the reductions in the Southern Hemisphere have been more dramatic. This includes respiratory syncytial virus (RSV) Metapneumovirus. Some of this for flu may be due to increased vaccination, as Australia nearly doubled the number by May of 2020 over 2019. For South America and South Africa, however, that cannot be a factor [18].

In the past, changes in locality, environment, technology and social density have had impacts on the health of human populations and brought new challenges to longevity and chronic disease, as well as new disease conditions [3,4,19]. An effective example is the invention and spread of agriculture that resulted in clearing land for forest and irrigation. Standing water promoted the success of populations of the anopheles mosquito and the spread of the malarial plasmodium (like P. falciparum) among human populations [20]. While density of human populations can concentrate wastes producing septic conditions, which promote disease, disease among human populations can introduce mass psychogenic disease. Human efforts to avoid disease have varied over time, ecology and cultural theory of disease [6] and some, like quarantine and social distancing, parallel the efforts of other social animals where we find rejection at the hive or nest and dispersal [21].

Urban life and especially globalization have made the spread of disease faster than human populations can respond to them. The development of avoidance methods, vaccines and cures require significant time to design, test and implement. At the same time the spread of a disease agent via air, sea and land has become nearly immediate. This includes not only the movement of humans as tourists and business professionals, but also the transport of goods [22]. Few shipping containers are examined for pathogens, infected animals or insects [23,24]. Examination for contamination of food and other material is seldom undertaken either at depots of shipping or receiving. Only 3% of all containers entering the USA are inspected [25]. The potential for transmission of disease is significant.

Evolutionary Co-Evolution, Advantage and Adaptation

Over the past 400 plus million years the vertebrate immune system has been in a constant dance of development with pathogens. Recently some viruses like HIV and CMV have evolved to break through the defenses of our immune system, to evade detection and/or to attack the immune system and

disarm it. This evolution has been co-evolutionary, where pathogens evolve modifications in how they present to our immune cells and the complexity of its communication system. Some of this co-evolution has been related to how we live and how we have changed as mammals, Primates and hominids [5].

Covid-19 presents a significant evolutionary challenge, as in the past three years it has produced a number of variants, as noted above, that are capable of efficient mutation allowing greater infectivity, but also with a bewildering number of means of inducing disease in hosts. Not only does the virus attack cells of the bronchial epithelial type, pneumocytes and upper respiratory tract cells resulting in often severe conditions sometimes promoted by excessive inflammatory responses [26], but it can cause a variety of organ involvement including conditions in the brain that result in long Covid "brain fog." The virus has changed type of host, from elderly and adults to children in its course [27].

The rapid mutation rate of the virus, the exchange of genetic material via recombination and the concentration of mutational hot spots in the S gene area for implantation efficiency, all make Covid-19 a most dangerous challenge to the human immune system. The new Mu, or B.1.621 lineage of Covid-19 has more than 19 mutations, most located in the S gene area of its genome [28]. It was detected originally in Columbia in June of 2021, and has now spread to some 39 other countries [29]. Another troubling aspect of the Covid-19 epidemic is the appearance of reinfection of people who had the disease [30]. The current dominant sub variant as of February 2023 is XBB.1.5 with 66% of cases followed by BQ.1.1 with about 20% according to CDC data. XBB.1.5 also called Kraken, has a greater ability to infect than previous strains due to a combination of mutations. This wave structure of the Covid-19 family of variants has generally produced more infectious forms and not more lethal ones. Such a strategy provides the virus with an adaptive sustainability within the regional and local population of host immune defense variation. It is a process of coevolution.

Usually a body having a viral disease produces sufficient neutralizing antibodies that the virus can be suppressed and eliminated from the host. The reinfection of individuals with Covid-19 seems to indicate that this may be compromised in some individuals. In addition, "breakthrough" cases where an individual contracts Covid-19 after being vaccinated are an indication of both failure of the production of sufficient antibody to suppress active infection, and of virus escape of active neutralizing antibody from vaccine design. This again, undermines the usual natural history of infection where the pool of susceptible individuals is depleted and the virus exhausts potential sources of infection.

At present there is no evidence of latent, undetectable infection in people who have had the disease, as in AIDS. However, Covid-19 variants appear able to reactivate other latent viral infections [31]. Nevertheless, the future of Covid-19 and other coronaviruses presents a significant public health challenge. The continued production of variants, occurrence of reinfection and breakthrough cases is worrying at best [32]. Greater efforts at quarantine are unlikely to be successful as people are showing increased resistance to these measures. The development of new treatments for infective disease could be one solution, but such outcomes require significant research, testing and manufacturing delays. Luckily people who have been vaccinated develop less severe disease, yet existing treatments appear to require early intervention as with dexamethasone or remdesivir and are limited due to type of delivery or side effects. New potential treatments, as is the case with anakinra and tocilizumab show promise in reducing death, but more research is necessary as is availability of drugs [33].

The possibility that Covid-19 and other coronaviruses will become endemic diseases with recurrent waves of mass population involvement as well as long-term debilitation of significant numbers of adults is a real problem. Human intervention has changed some fatal diseases like AIDS into chronic ones. Influenza now is a chronic yearly disease attenuated by vaccine in the number of deaths and severity of symptoms. While humanity has gone through a dramatic population change since industrialization and the application of public health measures, current urban density has created new conditions for disease as has the increase exploitation of wild fauna for food. The one clear conclusion from the current costs in terms of lives and disruption is that world health systems are inadequate to meet the challenge, especially in a globalized world [34]. More investments in hospitals and medical research and testing laboratories are needed.

References

1. Omran AR (1971) The epidemiologic transition: A theory of the epidemiology of population change. Milbank Memo Fund Quest 49(4): 509-538.

2. Zuckerman MK, Kristen NH, Ronald B, Amelagos GJ (2014) The evolution of disease: anthropological perspectives on epidemiologic transitions. Global Health Action 7: 10.

3. Greaves MF (2000) Cancer: The Evolutionary Legacy. Oxford University Press, USA, pp: 276.

4. Horacio F (1997) Evolution of Sickness and Healing. Berkeley (Edn.), University of California Press.

5. Caldararo N (1996) The HIV/AIDS epidemic: its evolutionary implications for human ecology with special reference to the immune system. Sci Total Environ 191(3): 245-269.

6. Caldararo N (2013) Some Evolutionary Aspects of Disease Avoidance. Saarbrucken (Edn) Scholars Press.

7. Foxman EF, James AS, Fitzgerald ME, Wasik BR, Hou L, et al. (2015) "Temperature-dependent innate defense against the common cold virus limits viral replication at war temperature in mouse airway cells. Proc Natl Acad Sci USA 112(3): 827-832.

8. Chandler AC, Clark PR (1961) Introduction to Parasitology. In: 10th (Edn.), John Wiley & Sons, New York, USA.

9. Mims CA, David OW (1984) Viral Pathogenesis and Immunity. Blackwell, Oxford, UK.

10. Wong AH M, Aidan CAT, Zhou D, Satkunarajah M, Chen K, et al. (2017) "Receptor-binding loops in alphacoronavirus adaptation and evolution". Nature Communications 8 (1735).

11. Jones S, Sathi SN, Wang Y, Prasad R, Rayen S, et al. (2019) "Evolutionary, genetic, structural characterization and its functional implications for the influenza A (H1N1) infection outbreak in India from 2009 to 2017". Scientific Reports 9(14690).

12. Devika S, Soojin VY (2021) On the origin and evolution of Sars- Cov-2. Experimental and Molecular Medicine 53: 537-547.

13. Boni MF, Lemey P, Jiang X, Lam TY, Perry BW, et al. (2020) Evolutionary origins of the SARS-CoV-2 sarbecovirus lineage responsible for the COVID-19 pandemic. Nat Microbiol 5: 1408-1417.

14. Burnet M (1966) Natural History of Infectious Disease. Cambridge University Press, USA.

15. Guillot S, Caro V, Cuervo N, Korotkova E, Combiescu M, et al. (2000) Natural genetic exchanges between vaccine and wild poliovirus strains in humnas. J Virol 74(18): 8434-8443.

16. Nowak MA (1992) What is a quasispecies?. Trends Ecol & Evol 7 (4): 118-121.

17. Sema N, Mair C, Louise M, Reeve R, Johnson PCD, et al. (2019) Virus-virus interactions impact the population dynamics of influenza and the common cold. PNAS 116(52): 27142-27150.

18. Nicola J (2020) How Covid-19 is changing the cold and flu season. Nature 588: 388-390.

19. McNeill WH (1976) Plagues and Peoples. The Hispanic American Historical Review Duke University Press 60(1): 97-99.

20. Janko MM, Irish SR, Reich BJ, Peterson M, Stephanie MD, et al. (2018) The links between agriculture, Anopheles mosquitoes, and malaria risk in children younder than 5 years in the Democratic Republic of the Congo: a population-based, cross-sectional, spatial study. Lancet Planet Health 2(2): E74-E82.

21. Caldararo N (2020) Covid-19, isolation, mammalian dispersal patterns, urban density, social distancing and mass psychogenic disease. Insights of Anthropology 4(2): 300-324.

22. Kulisch E (2016) U.S. Lawmakers say with new technology, its time to inspect all inbound containers. Westar.

23. Marchioro M, Andrea B, Faccoli M (2020) Light traps in shipping containers: a new tool for the early detection of insect alien species. Journal of Economic Entomology 113(4): 1718-1724.

24. Food and Agriculture Organization of the UN (2016) Floating threat: sea containers spread pests and diseases.

25. Montaigne F (2004) Policing America's Ports. The Smithsonian Magazine.

26. Kovski PV, Annika K, Steiner S, Stalder H, Thiel V (2021) Coronavirus biology and replication: implications for SARS-CoV-2. Nature Reviews Microbiology 19: 155-170.

27. Centers for Disease Control (2022) Post-Covid conditions.

28. PANGO Characteristic mutations in lineage, B.1.621 lineage report.

29. Rettner R (2021) New 'mu' coronavirus variant could escape vaccine-induced immunity, WHO says. LiveScience.

30. Cavanaugh AM, Spicer KB, Thoroughman D, Glick C, Winter K, (2021) Reduced Risk of Reinfection with SARS-CoV-2 After COVID-19 Vaccination-Kentucky, May-June 2021. Morbidity and Mortality Weekly Report 70(32): 1081-1083.

31. Apostolou E, Rizwan M, Moustardas P, Sjorgren P, Bertilson BC, et al. (2022) Saliva antibody-fingerprint of reactivated latent viruses after mild/asymptomatic

COVID-19 is unique in patients with myalgic-encephalomyelitis/chronic fatigue syndrome. Front Immunol, Sec Viral Immunology 13.

32. Centers for Disease Control, (2021) Covid-19 vaccine breakthrough case investigation and reporting.

33. Kyriazopoulou E, Poulakou G, Giamarellos Bourboulis EJ, Milionis H, Metallidis S, et al. (2021) Early treatment of COVID-19 with anakinra guided by soluble urokinase plasminogen receptor plasma levels: a double-blind, randomized controlled phase 3 trial. Nature Medicine 27: 1752-1760.

34. Ferreira FHG (2021) Inequality in the time of Covid-19. International Monetary Fund.