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The Historical/Evolutionary Cause and Possible Treatment of Pandemic Covid-19 (Sars-Cov-2, 2019-Coronavirus)

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Abstract

Background: A virus is a small infectious agent that replicates only inside the living cells of an organism. Viruses can infect all types of life forms, from animals and plants to microorganisms, including bacteria and archaea. In evolution, viruses are an important means of horizontal gene transfer, which increases genetic diversity in a way analogous to sexual reproduction.Influenza (Including (COVID-19), is an infectious disease caused by an influenza virus. Some viruses especially smallpox, throughout history, has killed between 300-500 million people in its 12,000-year existence.As modern humans increased in numbers, new infectious diseases emerged, including SARS-CoV-2. We have two groups of virus, RNA and DNA viruses. The most brutal viruses are RNA ones like COVID-19 (Sars-CoV-2).

Introduction: Coronaviruses are a group of viruses that cause diseases in mammals and birds. In humans, coronaviruses cause respiratory tract infections that are typically mild, such as some cases of the common cold (among other possible causes, predominantly rhinoviruses), though rarer forms can be lethal, such as SARS, MERS, and COVID-19. Symptoms vary in other species: in chickens, they cause an upper respiratory tract disease, while in cows and pigs they cause diarrhea. Coronaviruses constitute the subfamily Orthocoronavirinae, The genome size, coronaviruses ranges from approximately 27 to 34 kilobases, the largest among known RNA viruses.

Discussions and Results:We have researched from the first virus in the planet to the last mutated version which is SARS-COV-2. We have collected many informative data in tables and figures to reach the main cause of 2019-Coronavirus and calculated the probability and estimated deaths in the current time. We have discussed about the possible treatment and prevention of the virus and did algebraic calculations on the epidemiology, the size and even the future of this pandemic. The only era which any virus had not been epidemic, were through world war 2, were the German scientists had found the way to fight any viral infections which is very important and can help scientists to reach the main treatment of the new 2019-Coronavirus. We have sorted the deadly and non-deadly coronaviruses and explained how this epidemic had begun through Evolutionary Medicine (EM). The result of the article is that 16% of the whole population in the world has been contaminated which is 1248000000 of 7.8 billion people world-wide. SARS-CoV-2 is an RNA Virus. its nucleic acid is single-stranded RNA (ssRNA). The polarity of this virus is positive-sense ((+) ssRNA). Positive-sense viral RNA is similar to mRNA and thus can be immediately translated by the host cell. Recombination in RNA viruses appears to be an adaptation for coping with genome damage. Recombination can occur infrequently between animal viruses of the same species but of divergent lineages. The resulting recombinant viruses may sometimes cause an outbreak of infection in humans. RNA viruses have very high mutation rates This is one reason why it is difficult to make effective vaccines to prevent diseases caused by RNA viruses. The resulting recombinant viruses causes an outbreak of infection in humans.

Conclusion: In conclusion, the mutation of the SARS-CoV and influenza viruses through Drift and Reassortment is the main cause

of SARS-CoV-2 through natural selection, Lamarckian Evolution and coevolution which caused this RNA virus so powerful, unpredicted and different in the genome size and nations worldwide. The first Pandemic of Influenza was first detected in 1732 and this virus evolved through natural selection till 2019 which caused the worldwide pandemic of SARS-CoV-2. Based on many studies, inhalation of Ozone plus Sulfur Dioxide, increasing the amounts of L-Glutathione (Which is low in children and older adults and this is the main reason why older adults and children die from this disease.) plus Viral Phage Therapy (VPT) which we discussed fully in this article can be the possible prime treatment of SARS-CoV-2 infection. The seasonal temperature cannot be useful in controlling/reducing the pandemic of this virus since the natural selection, Lamarckian Evolution and high mutation of the virus helps its survival. No antiviral drugs will be useful against SARS-CoV-2 because of high rate of mutation and primarily adaptation of the virus to the drugs and even the environmental Temperature.

Keywords: COVID-19 (SARS-CoV-2);Drift; Fibonacci Series; Influenza; Lamarckian Evolution; L-Glutathione; Natural Selection;Ozone; Positive-Sense ((+) ssRNA Sulfur Dioxide; Reassortment; Pandemic); Viral Phage Therapy

Introduction

Brief History of Viruses

Prehistory: The world, new infectious diseases emerged, including those caused by viruses[1]. Earlier, humans lived in small, isolated communities, and most epidemic diseases did not exist[2,3]. Smallpox, which is the most lethal and devastating viral infection in history, first emerged among agricultural communities in India about 11,000 years ago[4]. The virus, which only infected humans, probably descended from the poxviruses of rodents [5]. Humans probably came into contact with these rodents, and some people became infected by the viruses they carried. When viruses cross this so-called "species barrier", their effects can be severe, [6] and humans may have had little natural resistance. Contemporary humans lived in small communities, and those who succumbed to infection either died or developed immunity.

This acquired immunity is only passed down to offspring temporarily, by antibodies in breast milk and other antibodies that cross the placenta from the mother's blood to the unborn child's. Therefore, sporadic outbreaks probably occurred in each generation. In about 9000 BC, when many people began to settle on the fertile flood plains of the River Nile, the population became dense enough for the virus to maintain a constant presence because of the high concentration of susceptible people [7]. Other epidemics of viral diseases that depend on large concentrations of people, such as mumps, rubella and polio, also first occurred at this time [8]. The Neolithic age, which began in the Middle East in about 9500 BC, was a time when humans became farmers [9].

This agricultural revolution embraced the development of monoculture and presented an opportunity for the rapid spread of several species of plant viruses [10]. The divergence and spread of sobemoviruses-southern bean mosaic virus-date from this time[11]. The spread of the potyviruses of potatoes, and other fruits and vegetables, began about 6,600 years ago. [10] About 10,000 years ago the humans who inhabited the lands around the Mediterranean basin began to domesticate wild animals. Pigs, cattle, goats, sheep, horses, camels, cats and dogs were all kept and bred in captivity[12]. These animals would have brought their viruses with them[13]. The transmission of viruses from animals to humans can occur, but such zoonotic infections are rare and subsequent human-to-human transmission of animal viruses is even rarer, although there are notable exceptions such as influenza. Most viruses are species-specific and would have posed no threat to humans[14].

The rare epidemics of viral diseases originating in animals would have been short-lived because the viruses were not fully adapted to humans [15] and the human populations were too small to maintain the chains of infection[16]. Other, more ancient, viruses have been less of a threat. Herpes viruses first infected the ancestors of modern humans over 80 million years ago[17]. Humans have developed a tolerance to these viruses, and most are infected with at least one species[18]. Records of these milder virus infections are rare, but it is likely that early hominids suffered from colds, influenza and diarrhoea caused by viruses just as humans do today. More recently evolved viruses cause epidemics and pandemics[17]. The influenza virus is one that seems to have crossed the species barrier from ducks and water fowl to pigs and from there to humans. It is possible that a fatal plague in the Middle East at the time of the late 18th Dynasty was associated with this transmission at Amarna [19-20].

Influenza: Influenza, commonly known as the flu, is an infectious disease caused by an influenza virus [21-25]. Symptoms can be mild to severe [25]. The most common symptoms include: high fever, runny nose, sore throat, muscle and joint pain, headache, coughing, and feeling tired just like Sars-CoV-2[21]. These symptoms typically begin two days after exposure to the virus and most last less than a week [21]. The cough, however, may last for more than two weeks [21]. In children, there may be diarrhea and vomiting, but these are not common in adults [26]. Diarrhea and vomiting occur more commonly in gastroenteritis, which is an unrelated disease and sometimes inaccurately referred to as stomach flu or the 24-hour flu[26]. Complications of influenza may include viral pneumonia, secondary bacterial pneumonia, sinus infections, and worsening of previous health problems such as asthma or heart failure [22,25]. Three of the four types of influenza viruses affect humans: Type A, Type B, and Type C[22,27]. Type D has not been known to infect humans, but is believed to have the potential to do so [27,28]. Usually, the virus is spread through the air from coughs or sneezes [21]**(Table 1-7)**. This is believed to occur mostly over relatively short distances [29]. It can also be spread by touching surfaces contaminated by the virus and then touching the eyes, nose, or mouth [25,29,30-35]. A person may be infectious to others both before and during the time they are showing symptoms [25,36-40]. The infection may be confirmed by testing the throat, sputum, or nose for the virus [45-50]. A number of rapid tests are available; however, people may still have the infection even if the results are negative [22]. A type of polymerase chain reaction that detects the virus's RNA is more accurate [22]**(Figure 1-3)**.

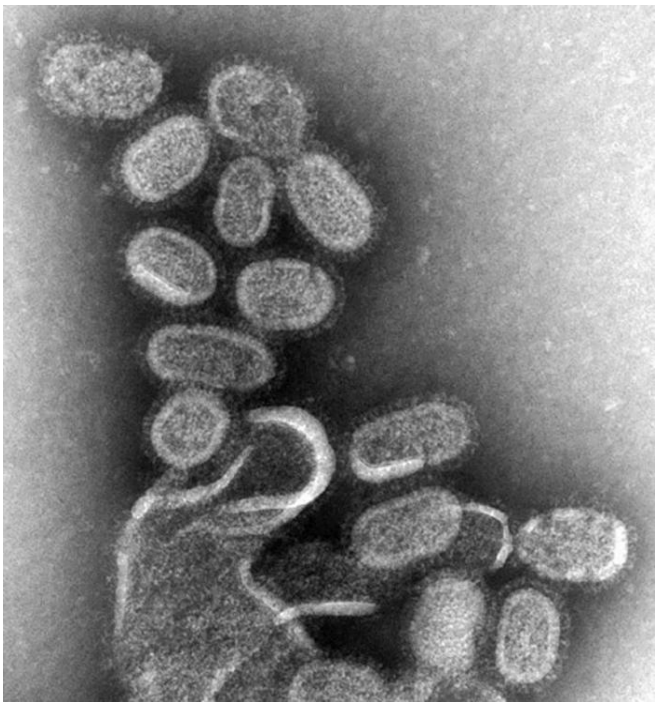

Figure 1: Influenza virus, magnified approximately 100,000 times, Specialty: Infectious disease, Symptoms: Fever, runny nose, sore throat, muscle and joint pain, headache, coughing, feeling tired, Usual onset: One to four days after exposure, Duration: 1 week, Causes: Influenza viruses.Prevention: Hand washing, influenza vaccine, surgical masks, Frequency: 3-5 million severe cases per year. Deaths: Up to 650,000 respiratory deaths per year[1-3].

Symptom	Sensitivity	Specificity
Fever	68-86%	25-73%
Cough	84-98%	7-29%
Nasal congestion	68-91%	19-41%
Note: All three findings, especially fever, were less sensitive in people over 60 years of age.		

Table 1: Most sensitive symptoms for diagnosing influenza [1-3].

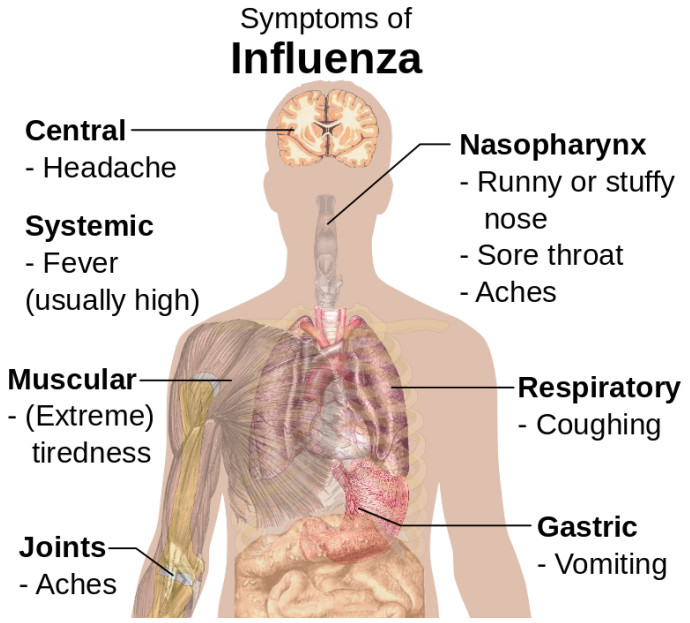

Figure 2: Symptoms of influenza, with fever and cough the most common symptoms [20-22].

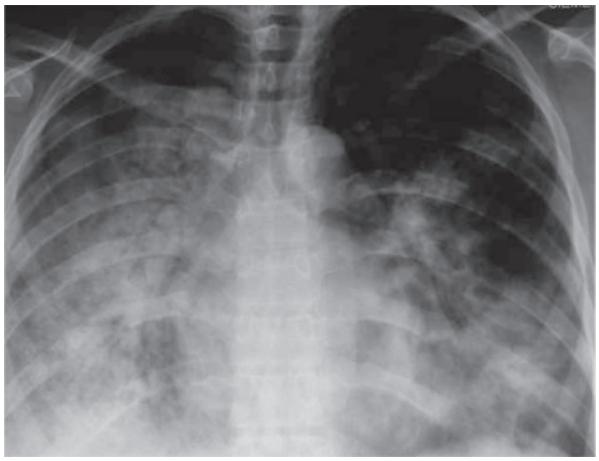

Figure 3: 29 yr. old with H1N1 with high inflammation in the lungs and respiratory systems.

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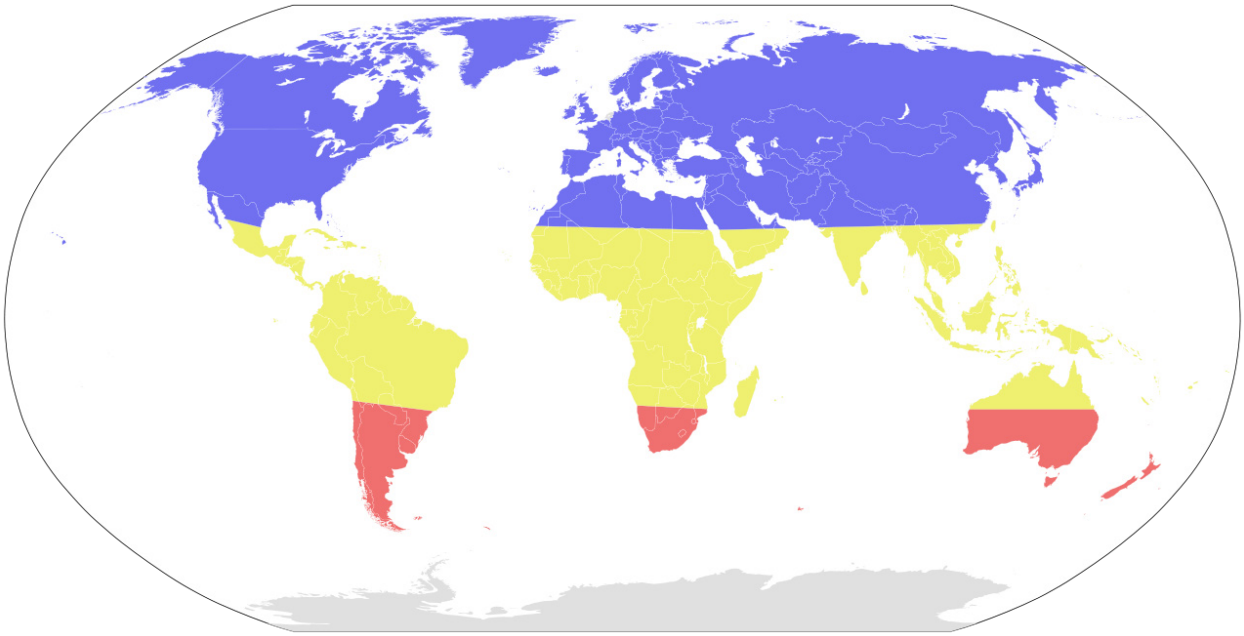

Figure 4: Seasonal risk areas for influenza: November-April (blue), April-November (red), and year-round (yellow) [21].

List of epidemics of Plagues

Death toll (estimate)	Location	Date	Event	Disease
75,000-100,000	Greece	429-426 BC	Plague of Athens	Unknown, possibly typhus, typhoid fever or viral hemorrhagic fever
5-10 million	Roman Empire	165-180 (possibly up to 190)	Antonine Plague	Unknown, possibly smallpox
	Europe	250-266	Plague of Cyprian	Unknown, possibly smallpox
25-50 million; 40% of population	Europe, Egypt and West Asia	541-542	Plague of Justinian	Plague
	Rome	590	Roman Plague of 590	Plague
> 100,000	Ctesiphon, Persia	627-628	Plague of Sheroe	Plague
	British Isles	664-689	Plague of 664	Plague
	Japan	735-737	735-737 Japanese smallpox epidemic	Smallpox
	Byzantine Empire, West Asia, Africa	746-747	Plague of 746-747	Plague
20-200 million; 20-60% of European population	Europe, Asia and North Africa	1331-1353	Black Death	Plague
				Y. pestis
> 10,000	England and later continental Europe	1485-1551	Sweating sickness (multiple outbreaks)	Unknown, possibly an unknown species of hantavirus

Table 2: 15th century and earlier.

Death toll (estimate)	Location	Date	Event	Disease	Ref.
5-15 million (80% of population)	Mexico	1545-1548	Cocoliztli Epidemic of 1545-1548	Possibly Salmonella enterica	[50-53]
2-2.5 million (50% of population)	Mexico	1576-1580	Cocoliztli epidemic of 1576	Possibly Salmonella enterica	[50-53]
280,000	Italy	1629-1631	Italian plague of 1629-1631	Plague	[54-60]
100,000	England	1665-1666	Great Plague of London	Plague	[61-67]
76,000	Austria	1679	Great Plague of Vienna	Plague	-
40,000	France	1668		Plague	[68]
30-90% of population	Southern New England, especially the Wampanoag people	1616-1619	1616 New England epidemic	Unknown cause. Latest research suggests epidemic(s) of leptospirosis with Weil syndrome. Classic explanations include yellow fever, bubonic plague, influenza, smallpox, chickenpox, typhus, and syndemic infection of hepatitis B and hepatitis D.	[58-59]
24,148[32]	Netherlands	1663-1664	-	Plague	-
> 20,100 in London	London	1563-1564	1563 London plague	Plague	-
> 19,900 in London and outer parishes	London	1592-1593	1592-93 London plague	Plague	-
	Seneca nation	1592-1596	-	Measles	[54]
	Spain	1596-1602	-	Plague	[55]
	South America	1600-1650	-	Malaria	[56]
	England	1603	-	Plague	[57]
	Egypt	1609	-	Plague	-
	Wyandot people	1634	-	Smallpox	[61]
	Thirteen Colonies	1633	Massachusetts smallpox epidemic	Smallpox	-
	England	1636		Plague	[62]
	China	1641-1644		Plague	[63]
	Spain	1647-1652	Great Plague of Seville	Plague	
	Central America	1648		Yellow fever	[64]
	Italy	1656	Naples Plague	Plague	[65]
	Thirteen Colonies	1657	-	Measles	[61]
	Spain	1676-1685	-	Plague	[67]
	Thirteen Colonies	1687	-	Measles	[68]
	Thirteen Colonies	1690	-	Yellow fever	-

Table 3: 16-17th centuries.

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-	Canada, New France	1702-1703	-	Smallpox	[69]
> 18,000 (36% of population)	Iceland	1707-1709	Great Smallpox Epidemic	Smallpox	-
	Denmark,Sweden	1710-1712	Great Northern War plague outbreak	Plague	-
	Thirteen Colonies	1713-1715	-	Measles	[70]
	Canada, New France	1714-1715	-	Measles	[71]
	France	1720-1722	Great Plague of Marseille	Plague	[72]
	Thirteen Colonies	1721-1722	-	Smallpox	[73]
	Thirteen Colonies	1729	-	Measles	[74]
	Spain	1730	-	Yellow fever	
	Thirteen Colonies	1732-1733	-	Influenza	[75]
	Canada, New France	1733	-	Smallpox	[76]
> 50,000	Balkans	1738	Great Plague of 1738	Plague	
	Thirteen Colonies	1738	-	Smallpox	[77]
	Thirteen Colonies	1739-1740	-	Measles	
	Italy	1743	-	Plague	[78]
	Thirteen Colonies	1747	-	Measles	
	North America	1755-1756	-	Smallpox	
	North America	1759	-	Measles	[79]
	North America, West Indies	1761	-	Influenza	
	North America, present-day Pittsburgh area.	1763	-	Smallpox	[80]
> 50,000	Russia	1770-1772	Russian plague of 1770-1772	Plague	
	Pacific Northwest natives	1770s	-	Smallpox	[81]
	North America	1772	-	Measles	
> 2,000,000	Persia	1772	1772 Persia plague	Plague	[8]

	England	1775-1776		Influenza	[82]
	Spain	1778	-	Dengue fever	[83]
	Plains Indians	1780-1782	North American smallpox epidemic	Smallpox	[84]
	Pueblo Indians	1788	-	Smallpox	[85]
	United States	1788	-	Measles	
	New South Wales, Australia	1789-1790	-	Smallpox	[86]
	United States	1793	-	Influenza and Epidemic Typhus	
	United States	1793-1798	Yellow Fever Epidemic of 1793, resurgences	Yellow fever	[87]

Table 4: 18th century.

Death toll (estimate)	Location	Date	Event	Disease	Ref.
1,000,000	Russia	1852-1860	Third cholera pandemic	Cholera	[88]
1,000,000	Worldwide	1889-1890	1889-1890 flu pandemic	Influenza	[83]
> 100,000	Asia, Europe	1816-1826	First cholera pandemic	Cholera	[89]
> 100,000	Asia, Europe, North America	1829-1851	Second cholera pandemic	Cholera	[89]
40,000	Fiji	1875	1875 Fiji Measles outbreak	Measles	[81]
> 20,000	Canada	1847-1848	Typhus epidemic of 1847	Epidemic typhus	[69]
> 9,000	India, Germany	1881-1896	Fifth cholera pandemic	Cholera	[89]
4,737	Copenhagen, Denmark	1853	Cholera epidemic of Copenhagen 1853	Cholera	[73]
3,164	Montreal	1885	-	Smallpox	Timeline
> 3,000	Central Coast, British Columbia	1862-1863	-	Smallpox	[78]

	England	1854	Broad Street cholera outbreak	Cholera	[74]
	Spain	1800-1803		Yellow fever	[86]
	Ottoman Empire, Egypt	1801	-	Bubonic plague	[87]
	United States	1803	-	Yellow fever	-
	Egypt	1812	-	Plague	-
	Ottoman Empire	1812	-	Plague	[88]
	Malta	1813	-	Plague	-
	Romania	1813	Caragea's plague	Plague	-
	Ireland	1816-1819	-	Typhus	-
	United States	1820-1823	-	Yellow fever	-
	Spain	1821		Yellow fever	[90]
	New South Wales, Australia	1828	-	Smallpox	[91]
	Netherlands	1829	Groningen epidemic	Malaria	-
	South Australia	1829	-	Smallpox	[92]
	Iran	1829-1835	-	Bubonic plague	[93]
	Egypt	1831	-	Cholera	[94,95]
	Plains Indians	1831-1834	-	Smallpox	-
	England, France	1832	-	Cholera	-
	North America	1832	-	Cholera	[96]
	United States	1833	-	Cholera	-
	United States	1834	-	Cholera	-
	Egypt	1834-1836		Bubonic plague	[96,97]
	United States	1837	-	Typhus	-
	Great Plains	1837-1838	1837-38 smallpox epidemic	Smallpox	[98]
	Dalmatia	1840	-	Plague	-
616	South Africa	1840	-	Smallpox	-
	United States	1841	-	Yellow fever	-
	United States	1847	-	Yellow fever	-
	Worldwide	1847-1848	-	Influenza	[99]
	Egypt	1848	-	Cholera	[96,97]
	North America	1848-1849	-	Cholera	-
	United States	1850	-	Yellow fever	-
	North America	1850-1851	-	Influenza	-
	United States	1851	-	Cholera	[100]
	United States	1852	-	Yellow fever	
	Ottoman Empire	1853	-	Plague	[101]
	United States	1855	-	Yellow fever	
	Worldwide	1855-1960	Third plague pandemic	Bubonic plague	[102]
	Portugal	1857	-	Yellow fever	
	Victoria, Australia	1857	-	Smallpox	[103]
	Europe, North America, South America	1857-1859	-	Influenza	[104-111]
	Middle East	1863-1879	Fourth cholera pandemic	Cholera	[112]
	Egypt	1865	-	Cholera	[96,97]
	Russia, Germany	1866-1867	-	Cholera	-
	Australia	1867	-	Measles	-
	Iraq	1867		Plague	[105]
	Argentina	1852-1871	-	Yellow fever	[106]
	Germany	1870-1871	-	Smallpox	-
	Russian Empire	1877	-	Plague	[108]
	Egypt	1881	-	Cholera	[96,97]
	West Africa	1900	-	Yellow fever	

Table 5: 19th century.

Death toll (estimate)	Location	Date	Event	Disease	Ref.
	Congo Basin	1896-1906		Trypanosomiasis	[109]
> 800,000	Europe, Asia, Africa	1899-1923	Sixth cholera pandemic	Cholera	[85]
113	San Francisco	1900-1904	-	Bubonic plague	[110]
	Uganda	1900-1920	-	Trypanosomiasis	[111]
	Egypt	1902	-	Cholera	[96,97]
22	India	1903	-	Bubonic Plague	[112]
4	Fremantle	1903	-	Bubonic plague	[113]
40,000	China	1910-1912	1910 China plague	Bubonic plague	[114]
1.5 million	worldwide	1915-1926	1915 Encephalitis lethargica pandemic	Encephalitis lethargica	[115]
up to 100,000,000	worldwide	1918-1920	Spanish flu	Influenza Spanish Flu Virus	[116]
	Russia	1918-1922	-	Typhus	-
30	Los Angeles	1924	1924 Los Angeles pneumonic plague outbreak	Pneumonic plague	-
43	Croydon, UK	1937	Croydon epidemic of typhoid fever	Typhoid fever	[117]
-	Egypt	1942-1944	-	Malaria	[96,97]
-	China	1946	-	Bubonic plague	
-	Egypt	1946	-	Relapsing fever	[96,97]
-	Egypt	1947	-	Cholera	[96,97]
2,000,000	worldwide	1957-1958	Asian flu	Influenza	[118]
	worldwide	1961-1975	Seventh cholera pandemic	Cholera	[110]
4	Sweden	1963		Smallpox	[119,120]
1,000,000	worldwide	1968-1969	Hong Kong flu	Influenza	[118]
5	Netherlands	1971		Poliomyelitis	[121]
35	Yugoslavia	1972	1972 outbreak of smallpox in Yugoslavia	Smallpox	-
	United States	1972-1973	London flu	Influenza	[122]
15,000	India	1974	1974 smallpox epidemic of India	Smallpox	[123]
> 32,000,000	worldwide(commenced in Congo Basin)	1960-present	HIV/AIDS pandemic	HIV/AIDS	[124]
	South America	1990s	-	Cholera	-
52	India	1994	1994 plague epidemic in Surat	Plague	[125]
231	worldwide	1996-2001	-	vCJD	-
-	West Africa	1996	-	Meningitis	-
105	Malaysia	1998-1999	1998-99 Malaysia Nipah virus outbreak	Nipah virus infection	[126]
	Central America	2000	-	Dengue fever	[127]

Table 6:20thcentury.

Death toll (estimate)	Location	Date	Event	Disease	Ref.
> 400	Nigeria	2001	-	Cholera	[128]
	South Africa	2001	-	Cholera	[129]
349	China	2002-2004	SARS outbreak	SARS corona-virus	[130]
299	Hong Kong	2002-2004	SARS outbreak	SARS corona-virus	[131]
37	Taiwan	2002-2004	SARS outbreak	SARS corona-virus	[132]
44	Canada	2002-2004	SARS outbreak	SARS corona-virus	[133]
33	Singapore	2002-2004	SARS outbreak	SARS corona-virus	[134]
-	Algeria	2003	-	Plague	[135]
-	Afghanistan	2004	-	Leishmaniasis	[136]
-	Bangladesh	2004	-	Cholera	[137]
-	Indonesia	2004	-	Dengue fever	[138]
-	Senegal	2004	-	Cholera	[139]
7	Sudan	2004	-	Ebola	[140]
	Mali	2005		Yellow fever	[141]
27	Singapore	2005	2005 dengue outbreak in Singapore	Dengue fever	[142]
	Luanda, Angola	2006	-	Cholera	[143-166]
61	Ituri Province, Democratic Republic of the Congo	2006	-	Plague	[167,168]
17	India	2006	-	Malaria	[169]
> 50	India	2006	2006 dengue outbreak in India	Dengue fever	[169]
	India	2006	Chikungunya outbreaks	Chikungunya virus	[170]
> 50	Pakistan	2006	2006 dengue outbreak in Pakistan	Dengue fever	[171]
	Philippines	2006	-	Dengue fever	[172]
187	Democratic Republic of the Congo	2007	Mwekaebola epidemic	Ebola	[173]
	Ethiopia	2007	-	Cholera	[174]
49	India	2008	-	Cholera	[175]
10	Iraq	2007	2007 Iraq cholera outbreak	Cholera	[176]
	Nigeria	2007	-	Poliomyelitis	[177]
	Puerto Rico; Dominican Republic; Mexico	2007	-	Dengue fever	[178]
	Somalia	2007	-	Cholera	[179]
37	Uganda	2007	-	Ebola	[180]
-	Vietnam	2007	-	Cholera	[181]
-	Brazil	2008	-	Dengue fever	[182]
-	Cambodia	2008	-	Dengue fever	[183]
-	Chad	2008	-	Cholera	[184]

-	China	2008-2017	-	Hand, foot and mouth disease	[185]
-	Madagascar	2008	-	Bubonic plague	[186]
-	Philippines	2008	-	Dengue fever	[187]
-	Vietnam	2008	-	Cholera	[188]
4,293	Zimbabwe	2008-2009	2008-2009 Zimbabwean cholera outbreak	Cholera	[189]
18	Bolivia	2009	2009 Bolivian dengue fever epidemic	Dengue fever	[190]
49	India	2009	2009 Gujarat hepatitis outbreak	Hepatitis B	[191]
-	Queensland, Australia	2009	-	Dengue fever	[192]
	Worldwide	2009	Mumps outbreaks in the 2000s	Mumps	
931	West Africa	2009-2010	2009-2010 West African meningitis outbreak	Meningitis	[193]
203,000	Worldwide	2009	2009 flu pandemic	Influenza	[194,195]
9,985 (May 2017)	Hispaniola	2010-present	Haiti cholera outbreak	Cholera	[196,197]
> 4,500 (February 2014)	Democratic Republic of the Congo	2011-present	-	Measles	[198][199]
170	Vietnam	2011-present	-	Hand, foot and mouth disease	[200,201]
> 350	Pakistan	2011	2011 dengue outbreak in Pakistan	Dengue fever	[202]
171 (as of 10 January 2013[update])	Darfur Sudan	2012	2012 yellow fever outbreak in Darfur, Sudan	Yellow fever	[203]
862 (as of 13 January 2020[update])	Worldwide	2012-present	2012 Middle East respiratory syndrome coronavirus outbreak	Middle East respiratory syndrome	[204-206]
142	Vietnam	2013-2014	-	Measles	[207]
>> 11,300	Worldwide, primarily concentrated in West Africa	2013-2016	Ebola virus epidemic in West Africa	Ebola virus disease, Ebola virus virion	[208-210]
183	Americas	2013-2015	2013-14 chikungunya outbreak	Chikungunya	[211]
40	Madagascar	2014-2017	2014 Madagascar plague outbreak	Bubonic plague	[212]
36	India	2014-2015	2014 Odisha jaundice outbreak	Primarily Hepatitis E, but also Hepatitis A	[213]
2,035	India	2015	2015 Indian swine flu outbreak	Influenza A virus subtype H1N1	[214][215][216]
~53	Worldwide	2015-2016	2015-16 Zika virus epidemic	Zika virus	[217]
100's (as of 1 April 2016[update])	Africa	2016	2016 yellow fever outbreak in Angola	Yellow fever	[218]
3,886 (as of 30 November 2019[update])	Yemen	2016-present	2016-17 Yemen cholera outbreak	Cholera	[219]
64 (as of 16 August 2017[update])	India	2017	2017 Gorakhpur Japanese encephalitis outbreak	Japanese encephalitis	[220]
18 (as of February 2020[update])	India	2018	2018 Nipah virus outbreak in Kerala	Nipah virus infection	[221]

2,253 (as of 20 February 2020[update])	Democratic Republic of the Congo & Uganda	August 2018-present	2018-19 Kivu Ebola epidemic	Ebola virus disease	-
>6,000 (by January 2019)	Democratic Republic of the Congo	2019-present	2019 measles outbreak in the Democratic Republic of the Congo	Measles	-
83	Samoa	2019-present	2019 Samoa measles outbreak	Measles	-
3,360 (as of 6 March 2020[update])	Worldwide	2019-present	2019-20 coronavirus outbreak	COVID-19	-

Table 7: 21st century.

Coronaviruses are a group of viruses that cause diseases in mammals and birds. In humans, coronaviruses cause respiratory tract infections that are typically mild, such as some cases of the common cold (among other possible causes, predominantly rhinoviruses), though rarer forms can be lethal, such as SARS, MERS, and COVID-19. Symptoms vary in other species: in chickens, they cause an upper respiratory tract disease, while in cows and pigs they cause diarrhea. Coronaviruses constitute the subfamily Orthocoronavirinae, in the family Coronaviridae, order Nidovirales, and realm Ribavirin [31,32]. They are enveloped viruses with a positive-sense single-stranded RNA genome and a nucleocapsid of helical symmetry. The genome size of coronaviruses ranges from approximately 27 to 34 kilobases, the largest among known RNA viruses[33].

Evolution

The most recent common ancestor (MRCA) of all coronaviruses has been placed at around 8000 BCE [34]. The MRCAs of the Alpha-coronavirus line has been placed at about 2400 BCE, the Beta-coronavirus line at 3300 BCE, the Gamma-coronavirus line at 2800 BCE, and the Delta-coronavirus line at about 3000 BCE. It appears that bats and birds, as warm-blooded flying vertebrates, are ideal hosts for the coronavirus gene source (with bats for Alpha-coronavirus and Beta coronavirus, and birds for Gamma-coronavirus and Delta-coronavirus) to fuel coronavirus evolution and dissemination [35]. Bovine coronavirus and canine respiratory coronaviruses diverged from a common ancestor in 1951 [36]. Bovine coronavirus and human coronavirus OC43 diverged around the 1890s. Bovine coronavirus diverged from the equine coronavirus species at the end of the 18th century [37]. The MRCA of human coronavirus OC43 has been dated to the 1950s [38]. MERS-CoV, although related to several bat coronavirus species, appears to have diverged from these several centuries ago [39]. The human coronavirus NL63 and a bat coronavirus shared an MRCA 563-822 years ago [40]. The most closely related bat coronavirus and SARS-CoV diverged in 1986 [41]. A path of evolution of the SARS virus and keen relationship with bats have been proposed. The coronaviruses have been coevolved with bats for a long time and the ancestors of SARS-CoV first infected the species of the genus Hipposideridae, subsequently spread to species of the Rhinolophidae and then to civets, and finally to humans[42-43]. Alpaca coronavirus and human coronavirus 229E diverged before 1960 [44].

Discussion

By mentioning all the facts, evolution and all the similarities from the viruses, we observe that all kinds of Flu including COVID-19 which have been caused many deaths through epidemic and pandemic of these giant but very small objects, all the viruses from the first one before the rise of the human in evolution, had been existed. Through my deep research, the first virus was or blue-green algae, pre-date viruses and are one of the oldest forms of life on Earth**(Figure 4)**.

Figure 4: Blue-green algae, pre-date viruses and are one of the oldest forms of life on Earth.

Results

The researchers developed algorithms to compare the protein shapes of 3,460 viruses and 1,620 cells. They found that 442 protein folds were shared between cells and viruses, but 66 folds were unique to viruses. To make sense of the data, the team arranged the protein folds into a tree that grew a new 'branch' every time a new type of protein fold evolved. Wherever possible, the team used fossil evidence to put an approximate date on the budding of specific branches. For example, one particular protein

fold was first seen in cyanobacteria (blue-green algae), and later appeared in all its descendants. By comparing when cyanobacteria first appeared in the fossil record (2.1 billion years ago) to when its offspring later emerged, they could establish this particular fold appeared around 2 billion years ago. According to Caetano-Anolles's microbial family tree, viruses are ancient, however; they were not the first form of life. In fact, his family tree suggests viruses and bacteria share a common ancestor - a fully functioning, self-replicating cell that lived around 3.4 billion years ago, shortly after life first emerged on the planet. From this cell, bacteria have evolved in the direction of increasing complexity, while viruses have gradually shed genes they found they didn't need, until they could no longer even reproduce on their own. A key step in the virus evolutionary journey seems to have come about around 1.5 billion years ago and that's the age at which the 66 virus-specific protein folds came on the scene. These changes are to proteins in the virus' outer coat-the machinery viruses use to break into host cells. For Charleston, the remarkable point about the study is how many proteins today's bacteria and viruses have in common.Viral evolution, Primordial cellular origins and late adaptation to parasitism, Mob Genet Elements**(Figure 5)**.

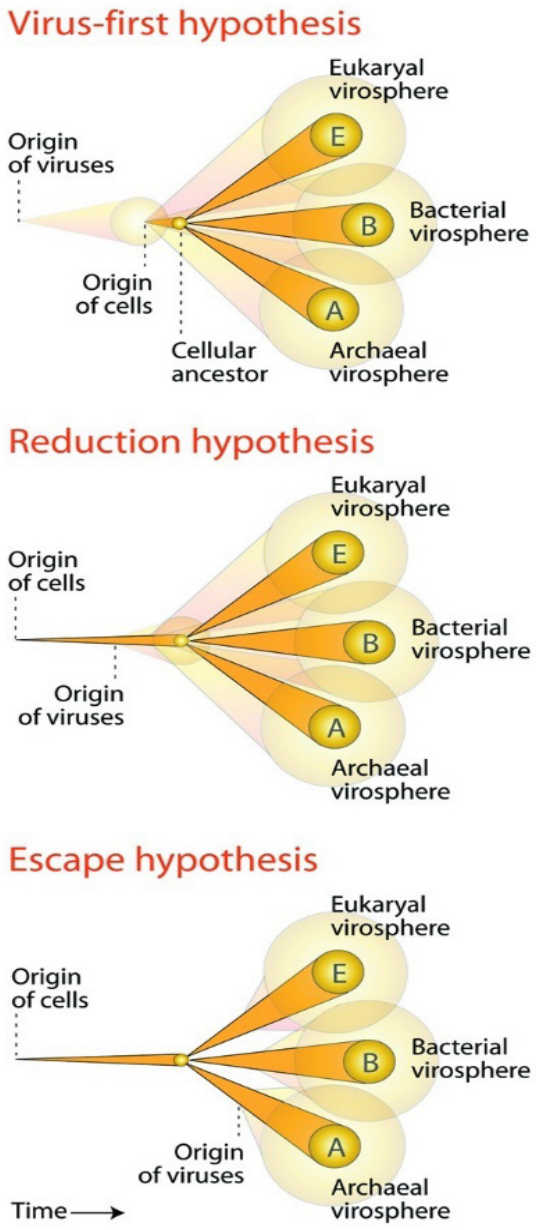

Figure 5: The origin of the viruses was before the beginning the cellular ancestor of any species. Based on **(Figure 5)**and the mentioned era of the virus evolution, they have been there since 3.5 billion years ago when cyanobacteria begin to be existed. The main problem is to find what or which kinds of matter can fight this machine-like organism.

Drift

New viruses can also emerge by drift. Drift can refer to genetic drift or antigenic drift. [45] Mutation and selection for the most advantageous variation of the virus takes place during this form of evolution. Natural Selection and Lamarckian Evolution both caused the evolution of several viruses like Coronaviruses fast enough[45]. Antigenic mutants can evolve quickly due to the high mutation rate in viruses. The cause of the antigenic drift lies in the mechanisms of RNA synthesis itself. Mutations arise very easily simply due to the error prone RNA polymerase and its lack of proofreading mechanisms. These mutations lead to subtle changes in the HA and NA genes which completely changes the infectious capabilities of the virus. These changes allow for almost endless possibilities for new viral strains to arise [46,47] and it is the antigenic drift of the HA and NA genes that allow for the virus to infect humans that receive vaccines for other strains of the virus[48]. This evolution occurs under the pressure of antibodies or immune system responses. Seven strains of human coronaviruses are known and four of them produce the generally mild symptoms of the common cold: 1) Human coronavirus OC43 (HCoV-OC43), 2) Human coronavirus HKU1, 3) Human coronavirus NL63 (HCoV-NL63, New Haven coronavirus), 4) Human coronavirus 229E (HCoV-229E) and three coronaviruses that are potentially fatal: 5) Middle East respiratory syndrome-related coronavirus (MERS-CoV), previously known as novel coronavirus 2012 and HCoV-EMC, 6) Severe acute respiratory syndrome coronavirus (SARS-CoV or "SARS-classic"), 7) Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), previously known as 2019-nCoV or "novel coronavirus 2019"[45].

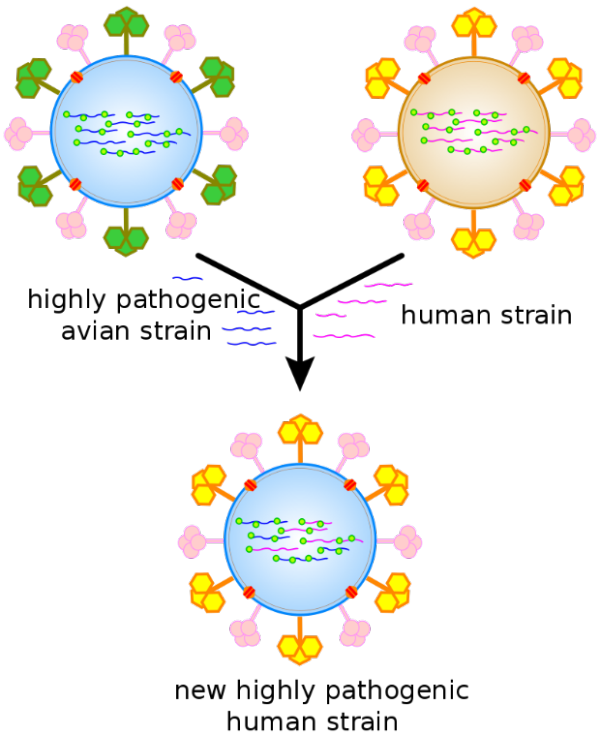

Figure 6: An antigenic shift, or Reassortment, which has the main result of novel and highly pathogenic strains of SARS-CoV-2 and other DNA and RNA viruses like All types of fatal Influenza.

Reassortment

Reassortment, also known as antigenic shift, allows new viruses to evolve under both natural conditions and in artificial cultures[45].Reassortment occurs in a similar way as chromosome crossover events, as two different viral strains may come in contact and transfer some of their genetic information. This crossing-over event creates a mixture of the two viral strains, which may replicate as one hybrid virus that expresses traits from both original viruses[46]. The mechanism of the evolutionary force of antigenic shift allows influenza viruses to exchange genes with strains that infect different species. Under this mechanism, a human influenza virus could exchange genes with an avian strain, and that is how pandemic strains arise. There have been three occurrences of pandemics caused by antigenic shift since 1900, and it has happened again today as we know as SARS-CoV-2[47].In fact, the 1957 evolution of the H2N2 virus is thought to be a result of Reassortment[45]. In this case, human H1N1 strains and avian influenza A genes were mixed [45]. Infecting tissue cultures can demonstrate how pathogenic qualities can evolve for a particular species even though the reasserted virus may be nonpathogenic for another species [45]. A prime example of evolution under natural conditions is the Reassortment of two avian influenza strains that were discovered in dead seals back in 1979 [45].

Based on research by an Inhalation of ozone and sulfur dioxide inhibited influenza virus growth in the nose of mice. Ozone inhalation caused the more pronounced inhibition of influenza virus growth: 0.6 ppm ozone for 3 hours' post-virus exposure almost completely inhibited influenza virus growth in the nose, whereas sulfur dioxide (6 ppm for 7 days) causes only partial inhibition of influenza growth in the nose. Neither gas altered the propagation of influenza virus in the lungs of mice. Vesicular stomatitis virus (VSV) growth was either unaffected by exposure to ozone (0.9 ppm for 3 hours) or, when ozone exposure preceded VSV exposure, the virus may have grown to slightly higher titer. The inhibitory effect of ozone and sulfur dioxide on influenza virus growth in nasal epithelium suggests a competitive interaction between the chemical inhalant, the virus, and host tissues, with net consequences for the pathogenesis of this disease. If the effects of these inhalants are to be properly interpreted, they should be determined for all major regions of virus growth and inhalant deposition. Therefore; inhalation of Ozone plus Sulfur Dioxide may inhibit the influenza virus growth in humans as well.

Another research indicates that Influenza virus was rendered noninfectious for mice by in vitro exposure to relatively high concentrations of sulfur and nitrogen mustards. Concentrations of the mustards that abolished infectivity did not reduce the ability of the virus to agglutinate erythrocytes. The parenteral administration of nitrogen mustard had no effect on the course of infection with influenza virus in mice.

There are several researches on the compound L-Glutathione which contains high sulfur amounts and it is normally produced by liver but the amounts vary in ages and sexes in its compound and inhibits viral infection. These researches has done in in Vivo and in Vitro with controlled groups double blind and received totally positive results and the treatment of the patients with several most dangerous viral diseases including Coronaviruses.

Sulfur and Viruses

Scientists have known about viruses that attack bacteria since the early twentieth century. In 1917, Felix d'Herelle, a French-Canadian microbiologist, and his colleagues successfully isolated phages that kill bacteria like E. coli, salmonella, and dysentery, and doctors used his so-called phage therapies to treat disease. But phage research lost steam in the 1950s in the face of penicillin and other powerful new antibiotics.Bacteria are living micro-organism which can be affected by viruses. The adaptation of bacteria to fight viruses are higher than human living cells. Therefore; this is the most important research on Phages against viruses which should be considered by scientists to fight against all viruses including 2019-Coronavirus. Paula Dalcin Martins and his colleagues reached positive results using Sulfur against many viruses. Basically If two viruses share a good number of genes, they are probably related like MERS, SARS and 2019-Coronavirus.The fewer genes they share, the more divergent their evolutionary paths. The most impressive article about sulfur against viruses has been published by Matthew B Sullivan which indicated Sulfur can kill and terminate any known viruses in eukaryotic host cells. Based on Sullivanstatements and articles, Sulfur and Phage Therapies can be one of the main treatment of viral diseases through coevolution hypothesis (coevolution occurs when two or more species reciprocally affect each other's evolution through the process of natural selection like viruses and mammals.) including SARS-CoV-2**(Figure 7-10)**.

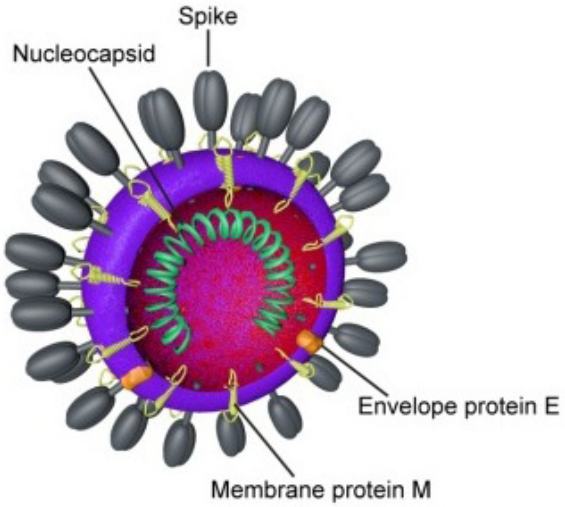

Figure 7: Diagram of coronavirus Virion structure.

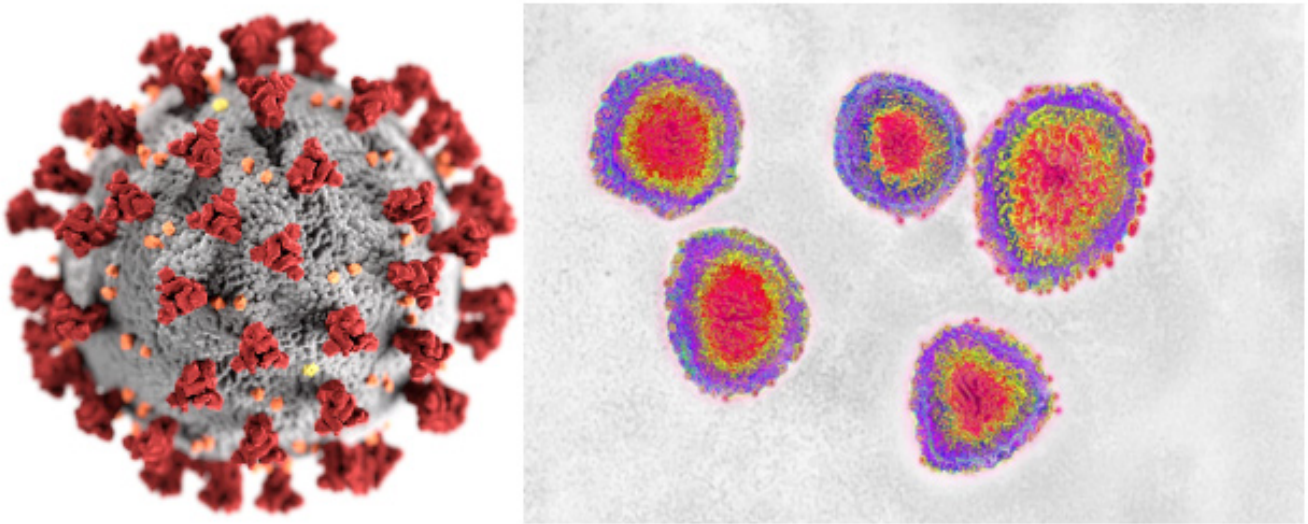

Figure 8: Illustration of a SARS-CoV-2 Virion.

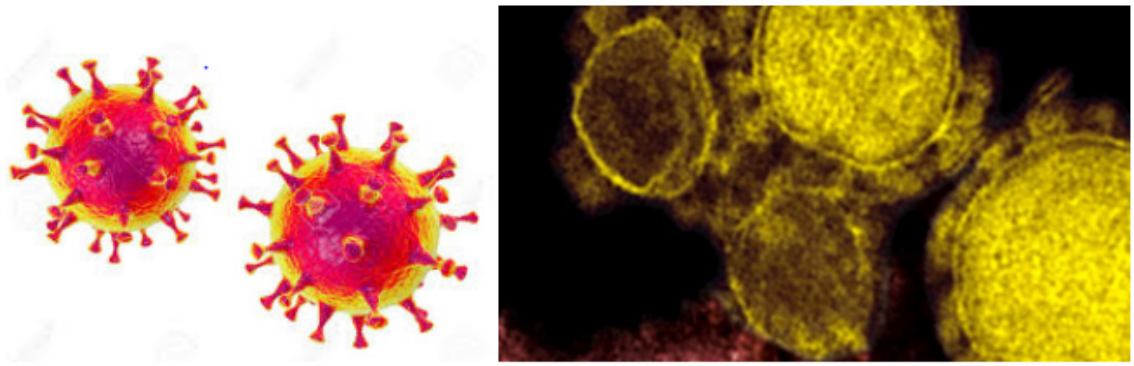

Figure 9:MERS virus, Middle-East Respiratory Syndrome coronavirusVirion, (Camel Flu).

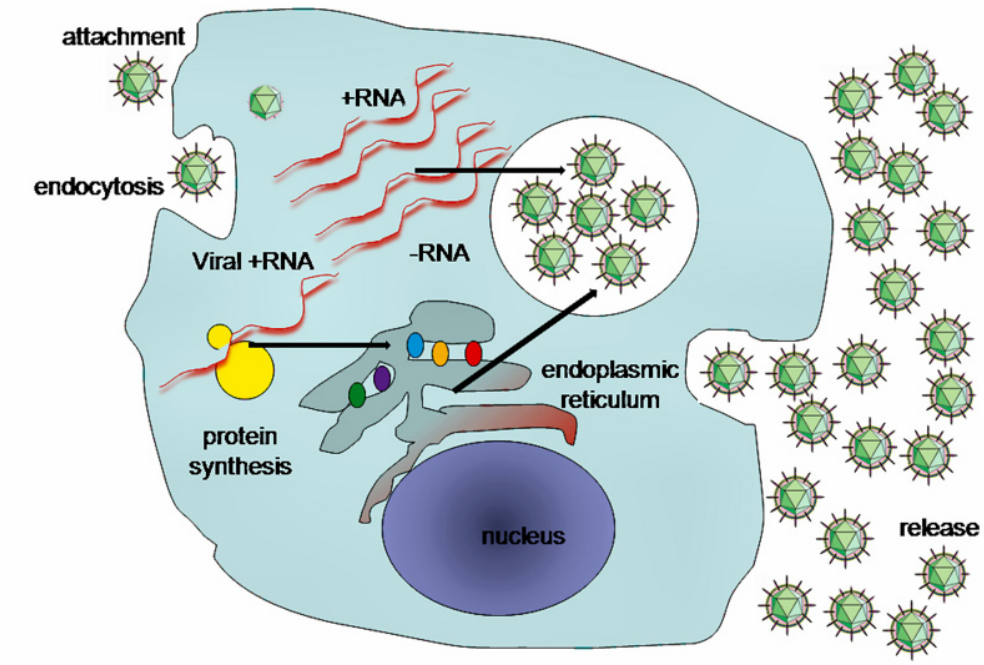

Figure 10: A typical virus replication cycleincluding Sars-Cov-2019.2019-nCoV is sufficiently divergent from SARS-CoV to be considered a new human-infecting betacoronavirus. Although our phylogenetic analysis suggests that bats might be the original host of this virus, an animal sold at the seafood market in Wuhan might represent an intermediate host facilitating the emergence of the virus in humans. Importantly, structural analysis suggests that 2019-nCoV might be able to bind to the angiotensin-converting enzyme 2 receptor in humans. The future evolution, adaptation, and spread of this virus warrant urgent investigation.

In Taxonomy, the family Coronaviridae is organized in 2 sub-families, 5 genera, 23 sub-genera and about 40 species: Coronaviridae - Orthocoronavirinae -Letovirinae-Alphaletovirus-Milecovirus and then Microhylaletovirus[3].

MERS-CoV and Severe acute respiratory syndrome (SARS) are betacoronavirus derived from bats [2].Between November 2002 and July 2003, an outbreak of SARS in southern China caused an eventual 8,098 cases, resulting in 774 deaths reported in 17 countries (9.6% fatality rate), with the majority of cases in mainland China and Hong Kong. No cases of SARS have been reported worldwide since 2004. In late 2017[1-3]. Chinese scientists traced the virus through the intermediary of civets to cave-dwelling horseshoe bats in Yunnan province[220]. Therefore; through the lines above, SARS and COVID-19 outbreak was first observed in China.The coronaviruses HCoV-229E, -NL63, -OC43, and -HKU1 continually circulate in the human population and cause respiratory infections in adults and children world-wide [221].

In the new Coronavirus, Genetic recombination which is the process by a strand of DNA that is broken and then joined to the end of a different DNA molecule. This can occur when viruses infect cells simultaneously and studies of viral evolution have shown that recombination has been rampant in the species studied. [230]. Recombination is common to both RNA and DNA viruses [108,220]. SARS-CoV-2 has undergone genetic change by several mechanisms. These include a antigenic drift process where individual bases in the RNA mutate to other bases. the mutations of this virus is silent means it does not change the protein that the gene encodes. And confers evolutionary advantages such as resistance to antiviral drugs [210-220]. Therefore; we cannot mention there would be any possible antiviral drugs to resist this kind of virus even in the future.

SARS-CoV-2 is an RNA Virus. its nucleic acid is single-stranded RNA (ssRNA). The polarity of this virus is positive-sense ((+) ssRNA). Positive-sense viral RNA is similar to mRNA and thus can be immediately translated by the host cell. Recombination in RNA viruses appears to be an adaptation for coping with genome damage. Recombination can occur infrequently between animal viruses of the same species but of divergent lineages. The resulting recombinant viruses may sometimes cause an outbreak of infection in humans. RNA viruses have very high mutation rates. This is one reason why it is difficult to make effective vaccines to prevent diseases caused by RNA viruses[215,216].

Mathematical Calculation of SARS-CoV-2 and Future Concerns: The Factorial formula is:

Factorial Formula

Factorial formula by **Product**

$$n! = \prod_{k=1}^{n} k$$

Factorial Formula by **Recurrence Relation**

$$n! = \begin{cases} 1 & if\ n = 0, \\ (n-1)! * n & if\ n > 0 \end{cases}$$

The size of SARS-CoV-2 is approximately 0.3 microns[237]. With conversion of Micrometer into nanometer, 0.3 will be 300 nm in size. In page 21, we have described the Antigenic Drift of viruses especially SARS-CoV-2. The mathematical calculation of how many types of this virus exists is: 3 × 4, since we have 4 types of mild coronavirus and 3 types of deadly with severe symptoms. So the numbers of different coronaviruses will be 3 × 4=12. Therefore; based on each type of virus is different from another one, we use Factorial to measure all types of coronavirus which exists on earth till now without the concern of the mutation in vivo of the virus. It is 12!=479001600. Overall, the number of different SARS-CoV-2 is about 479001600. Each of these kinds of viruses when get inside their hosts also mutates. Therefore; Each of these coronaviruses get inside their host will have different symptoms from mild into severe based on the host's immune system response and other factors. Hence; all these SARS-CoV-2, will mutate and possible severe symptoms will occur based on the Reassortment of the virus which have been described in page 22.The probability of each person in the world who may contaminate with this virus is 7.8 Billion/479001600 equals 16.2838704505.

This is the probability from the first time outbreak of the virus means approximately 16% of the population infected by the virus. So the near future of death or infected people from SARS-CoV-2 will be 1248000000. Based on high rates of mutation of 2019-Corovirus, each carrier of this deadly virus may/will contaminate another living human. Therefore; all people will carry this virus in the near future. We do not know the time range of this highly pandemic, but the lesson that we have learnt from the history is that the only way to fight the virus will not producenew vaccines, since the adaptation and the mutation of the virus is higher than any knownones on earth. The only answer to end the pandemic, is the adaptation of human race to SARS-CoV-2 through Lamarckian Evolution. This infectious disease with 7.8 billion people living on earth is like playing with dices and the people contamination is random in mathematical Theorems. Some may adapt to the virus, some will die and the rest will be the carrier, therefore; the graph we predict is like The bean machine, a device invented by Francis Galton shown below**(Figure 11)**:

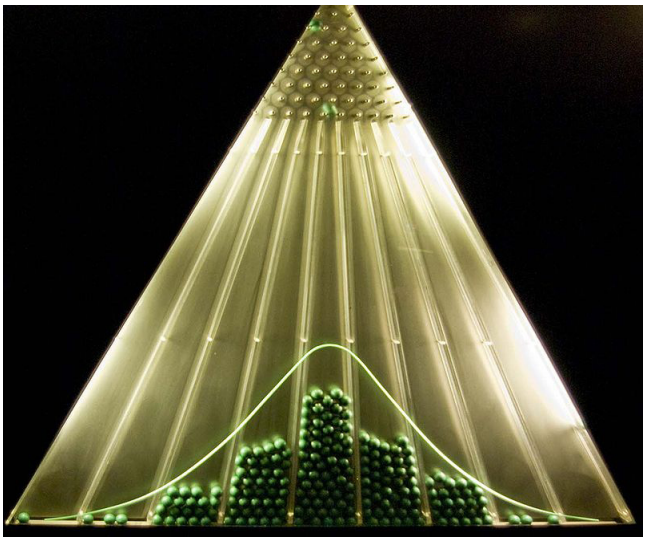

Figure 11: The bean machine, a device invented by Francis Galton, can be called the first generator of normal random variables. This machine consists of a vertical board with interleaved rows of pins. Small balls are dropped from the top and then bounce randomly left or right as they hit the pins. The balls are collected into bins at the bottom and settle down into a pattern resembling the Gaussian curve.

According to the central limit theorem (more specifically, the de Moivre-Laplace theorem), the binomial distribution approximates the normal distribution provided that the number of rows and the number of balls are both large. Varying the rows will result in different standard deviations or widths of the bell-shaped curve or the normal distribution in the bins [219].

Citation: Niknamian S and Zaminpira S (2020) The Historical/Evolutionary Cause and Possible Treatment of Pandemic Covid-19 (Sars-Cov-2, 2019-Corona-virus). Ann Med &Surg Case Rep: AMSCR-100050

2019-Corovirus Relation to Fibonacci Sequence: The Fibonacci numbers, commonly denoted Fn, is a function called the Fibonacci sequence, means each number is the sum of the two preceding numbers, starting from 0 and 1:

$$F_0 = 0, \quad F_1 = 1,$$
$$F_n = F_{n-1} + F_{n-2},$$
$$0,\ 1,\ 1,\ 2,\ 3,\ 5,\ 8,\ 13,\ 21,\ 34,\ 55,\ 89,\ 144,\ \ldots$$

And the sequence is: We have collected the infected people with COVID-19. If we suppose each infected individual infects one other individual each day and at the end of day-two recovers fully or dies (we are interested in the number of people who are actively infected and alive). So the time stamps are the beginning of day-one, at the end of day-one and at the end of day-two. According to our model, an infected individual can infect one other person at the end of day-one and at the end of day-two. If we go on through time (days), the table below shows the result of infected people **(Figure 12-14)**.

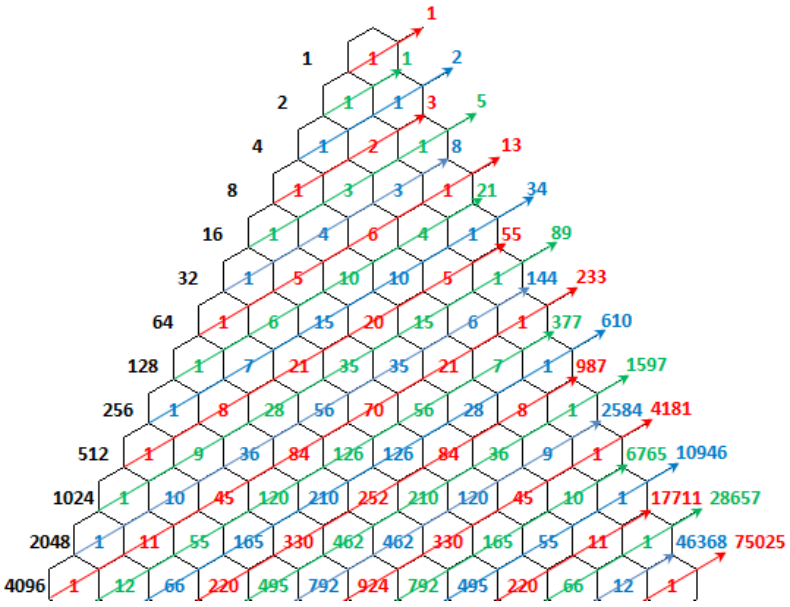

Figure 12: The right vertical numbers shows the infected people per day.

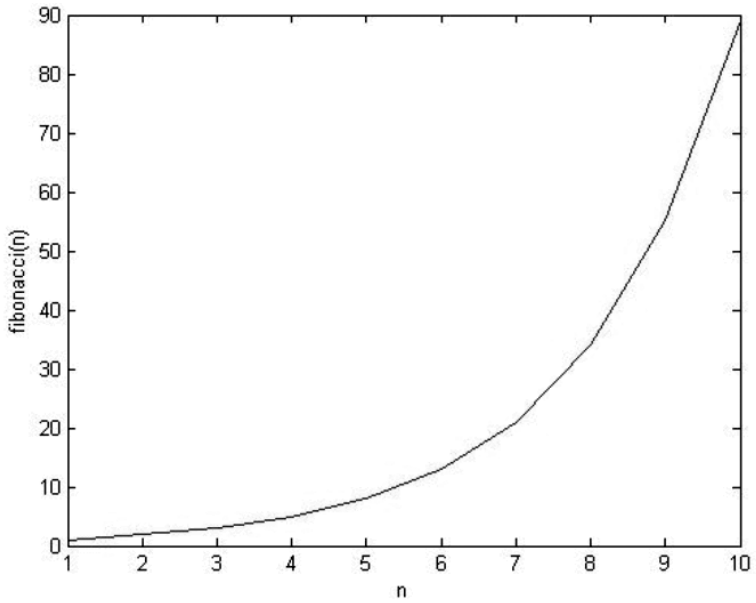

Figure 13: The number of infected people through time (day) by COVID-19.

Citation: Niknamian S and Zaminpira S (2020) The Historical/Evolutionary Cause and Possible Treatment of Pandemic Covid-19 (Sars-Cov-2, 2019-Coronavirus). Ann Med &Surg Case Rep: AMSCR-100050

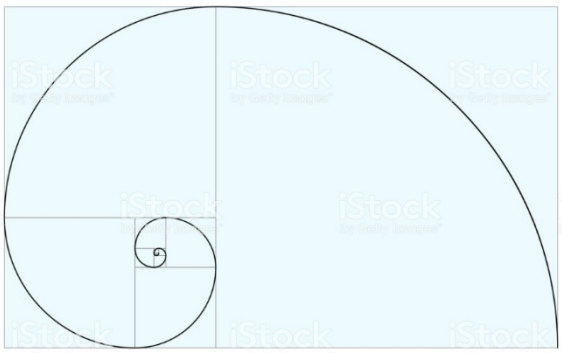

Figure 14: The Graph of Fibonacci and Pandemic of COVID-19.

Therefore; the formula of infected people per day is: $F_n = F_{n-1} + F_{n-2}$(for n > 2), which is the formula of Fibonacci series.

Viruses Related to COVID-2019

There are some viruses that causes respiratory infection. Enteroviruses, Poliovirus, coxsackie, echovirus and Human orthopneumovirus. Hence; none of them has been related to 2019-Coronavirus. Since their Signs and symptoms, Structure, Genome, Evolution, Taxonomy and Transmission are not similar to the new COVID-2019. The only virus that has been correlated with COVID-2019 is Rhinovirus. The rhinovirus is the most common viral infectious agent in humans and is the predominant cause of the common cold and flu. Rhinovirus infection proliferates in temperatures of 33-35 °C (91-95 °F), the temperatures found in the nose. The three species of rhinovirus (A, B, and C) include around 160 recognized types of human rhinovirus that differ according to their surface proteins (serotypes) They are lytic in nature and are among the smallest viruses, with diameters of about 30 nanometers. By comparison, other viruses, such as smallpox and vaccinia, are around ten times larger at about 300 nanometers (the same diameter as COVID-2019 which is 300 nanometers in size)[220].

There are two modes of transmission: via aerosols of respiratory droplets and from fomites (contaminated surfaces), including direct person-to-person contact. Rhinoviruses are spread worldwide and are the primary cause of the common cold. Symptoms include sore throat, runny nose, nasal congestion, sneezing and cough. Those most affected by rhinoviruses are infants, the elderly, and immune compromised people (Most elderly and Children). Human rhinoviruses preferentially grow at 32 °C (89 °F), notably colder than the average human body temperature of 37 °C (98 °F); hence the virus's tendency to infect the upper respiratory tract, where respiratory airflow is in continual contact with the (colder) extrasomatic environment [221].

Rhinoviruses have single-stranded positive sense RNA genomes of between 7200 and 8500 nt in length. At the 5' end of the genome is a virus-encoded protein, and like mammalian mRNA, there is a 3' poly-A tail. Structural proteins are encoded in the 5' region of the genome and nonstructural at the 3' end. This is the same for all picornaviruses. The viral particles themselves are not enveloped and are icosahedral in structure. The viral proteins are translated as a single, long polypeptide, which is cleaved into the structural and nonstructural viral proteins [212]. Human rhinoviruses are composed of a capsid that contains four viral proteins, VP1, VP2, VP3 and VP4.VP1, VP2, and VP3 form the major part of the protein capsid. The much smaller VP4 protein has a more extended structure, and lies at the interface between the capsid and the RNA genome. There are 60 copies of each of these proteins assembled as an icosahedron. Antibodies are a major defense against infection with the epitopes lying on the exterior regions of VP1-VP3. Human rhinovirus is most contagious during the autumn and winter months. The virus can remain activated for up to 3 hours outside of a human host. Once the virus is contracted, a person is most contagious within the first 3 days. Preventive measures such as regular vigorous hand washing with soap and water may aid in avoiding infection. Avoiding touching the mouth, eyes and nose, the most common entry points for rhinovirus, may also aid in prevention. Droplet precautions, which take the form of a surgical mask and gloves, is the method used in major hospitals.[243] [214,215].As mentioned, through our analysis, the most related virus to COVID-2019, is Rhinovirus which has very similar in Structure, Genome, Size and symptoms of the SARS-CoV-2. There has not been an effective anti-viral vaccination of this virus either.

There is a high possibility of mutation in this virus into 2019-Coronavirus. In 2018, a new series of anti-rhinoviral compounds were reported by researchers at Imperial College London and colleagues at the University of York and the Pirbright Institute. These molecules target human N-myristoyltransferase, an enzyme in the host cell which picornavirus requires in order to assemble its viral capsid, and thus generate an infectious virion. The lead compound in this series, IMP-1088, very potently inhibited host myristoylation of viral capsid protein and prevented infectious virus formation, rescuing the viability of cells in culture which had been exposed to a variety of rhinovirus serotypes, or to related picornaviruses including poliovirus and foot-and-mouth-disease virus. Because these compounds target a host factor, they are broadly active against all serotypes, and it is thought to be unlikely that they can be overcome by resistance mutations in the virus.

The main Cause of COVID-19 Pandemic

Through the lines and statements in this article, we hypothesize that the prime cause of SARS-CoV-2 is:

- The coevolution and Red Queen effect between mammals including humans and viruses. coevolution occurs when two or more species reciprocally affect each other's evolution through the process of natural selection and the Red Queen effect which is an evolutionary hypothesis that proposes organisms must constantly adapt, evolve, and proliferate in order to survive while pitted against ever-evolving opposing organisms in a constantly changing environment, as well as to gain reproductive advantage. Increasing human population, antibiotic resistance, antiviral resistance, malnutrition, immune

deficiency (basically nutrition low in sulfur), low adaptation of human in their environment (Lamarckian Evolution) andhigh adaptation of viruses with human genome [220].

- The main cause of SARS-CoV-2 is through natural selection and Lamarckian Evolution Both which caused this RNA virus so powerful and different in the nations worldwide. The first Pandemic of Influenza was detected in 1732 and this virus evolved through natural selection till 2019 through Drift and Assortment of SARS-CoV-1 and Influenza viruses which caused the worldwide pandemic of SARS-CoV-2. [45,46]. There may be a correlation between Rhinoviruses and COVID-19 which has not been studied yet and should be considered [221].

The Possible Treatment of COVID-19 (SARS-CoV-2)

Based on our research/Review of many major researches, there has not been any antiviral vaccines against these viruses and no antiviral drugs will be useful against COVID-19 since the adaptation and high mutation of this virus will not let these drugs be effective. The prime possible treatment Inhalation of ozone and sulfur dioxide, increasing L-Glutathione Plus Viral Phage Therapy (VFT) is the possible prime treatment of this disease. Finding the right Phage Therapy is very hard in any viral infections since this therapy was mainly used against bacterial infections. However; through our research and findings we have concluded that substantial dose-dependent inhibition by T4 phage of adsorption of HAdV to both A549 and HEK293 cell lines in vitro LPS was without effect. Moreover, T4 phage protected A549 cells from anHAdV-induced cytopathic effect. These data suggest that T4 phage could be considered as a potential novel antiviral agent. The capacity of the phage to inhibit HAdV infection at the stage of viral replication suggests that phage could also interfere with viruses using cellular receptors other than those used by HAdV. Notably, the anti-viral effect of phage was measured as the decrease of the end-point HAdV infectious titer. Further in vitro studies were carried out on the effects of T_4 and the staphylococcal phage A5/80 on HAdV DNA synthesis (real-time PCR) and the expression of its early and late genes (measured at the level of mRNA synthesis) in an A549 cell culture. Continuous incubation of adenovirus-infected cells with T_4 phage significantly reduced the level of adenoviral DNA synthesis (this effect was not observed with the staphylococcal phage). Incubation with a high titer of T_4 phage was required for inhibition of HAdV early gene expression. Late adenoviral gene expression was reduced by preincubation (application of phage prior to HAdV infection) and Coincubation with both phages when a low HAdV titer was used. In contrast, when a high HAdV titer was applied, both incubation and preincubation with T4 phage were inhibitory whilst a staphylococcal phage was inhibitory only when continuous incubation with cells was applied. Those results suggest that the inhibitory effect of phage on HAdV infection may differ and may be partially explained by their influence on the expression of early and late adenoviral genes. When the effect of both phages on the expression of genes involved in antimicrobial immunity by the A549 cell line from human lung was studied, the most striking phenomenon was marked (>10-fold) enhancement of a gene coding for interleukin-2 (Il-2) by a staphylococcal phage (Borysowski et al., 2018). This effect is of particular interest in the context of the well-known role of NK cells in the immune response to viruses and the ability of Il-2 to induce those cells - even in ultra-low doses. Interestingly, a resident NK cell population is present in the human lung and may provide early and important control of viral infection (Cooper et al., 2018). Moreover, NK cells also exhibit activity against a variety of bacteria, e.g., by secretion of the soluble molecules perforin and granulysin. Mice infected with Shigella and lacking B and T cells but with normal NK cells have higher survival rates and lower bacterial titers than mice which lack all three cell types. This suggests that phage-induced Il-2 dependent activation of NK-mediated antimicrobial activity may contribute to beneficial effects of PT, especially during prolonged phage administration cumulative median duration of successful phage therapy is 43 days. NK cells could be a promising agent in antimicrobial immunotherapy, as data strongly suggest that these cells are active against not only viral but also bacterial and fungal pathogens.

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Conclusion

In this Research/Review articles, we have gone through all the cases and epidemic of all the fatal diseases from the first time mentioned in history including Flu/Influenza pandemicuntil 2020. Based on our research, the only period of time when there was no outbreak of influenza, RNA viruses or infectious diseases were during World War 2, where German scientists controlled the epidemic of any virus infectionswhich should be noted by the scientists and researchers. The mutation of the SARS-CoV and Influenza viruses is the main cause of SARS-CoV-2 through natural selection and Lamarckian Evolution Both which caused this RNA virus so powerful and different in the nations worldwide. The first Pandemic of Influenza was first detected in 1732 and this virus evolved through natural selection till 2019 which caused the worldwide pandemic of SARS-CoV-2. Based on many studies, inhalation of Ozone plus Sulfur Dioxide, increasing the amounts of L-Glutathione (Which is low in children and older adults) plus Viral Phage Therapy (VPT)can be the prime treatment of SARS-CoV-2 infection. The seasonal temperature cannot be useful in controlling or reducing the pandemic of this virus since the temperature rises gradually and the natural selection, Lamarckian Evolution and high mutation rate of the virus helps its survival. In our mathematical calculation, the number of infected people is/will be 1248000000 which should not be ignored by the scientists. We also introduced the Fibonacci Model in calculating the infected/

will be infected people. Through our research we have found that no antiviral drugs will be useful against SARS-CoV-2 because of high rate of mutation and adaptation of the virus to the drugs and even the environment temperature.

References

1. McMichael AJ (2004) Environmental and social influences on emerging infectious diseases: past, present and future. Philos Trans R SocLond B BiolSci 359: 1049-1058.
2. Hughes AL, Irausquin S, Friedman R (2010) The evolutionary biology of poxviruses. Infect Genet Evol 10: 50-59.
3. Georges AJ, Matton T, Courbot-Georges MC (2004) Monkey-pox, a model of emergent then reemergent disease. Méd Mal Infect 34: 12-19.
4. Gibbs AJ, Ohshima K, Phillips MJ, Gibbs MJ (2008) The prehistory of potyviruses: their initial radiation was during the dawn of agriculture. PLoS ONE 3: e2523.
5. Fargette D, Pinel-Galzi A, Sérémé D, Lacombe S, Hébrard E, Traoré O, Konaté G (2008) Diversification of rice yellow mottle virus and related viruses spans the history of agriculture from the neolithic to the present. PLOS Pathog 4: e1000125.
6. Zeder MA (2008) Domestication and early agriculture in the Mediterranean Basin: origins, diffusion, and impact. Pro Natl AcadSci USA 105: 11597-11604.
7. White DW, Suzanne Beard R, Barton ES (2012) Immune modulation during latent herpesvirus infection. Immunol Rev 245: 189-208.
8. Martin P and Martin-Granel E (2006) 2,500-year evolution of the term epidemic. Emerg Infect Dis 12 : 976-80.
9. World Health Organization (2018) Influenza (Seasonal).
10. Fauci AS, Hauser SL, Jameson JL, Kasper DL, Longo DL, et al.(2012) Chapter 187: Influenza. Harrison's principles of internal medicine 18th ed. New York: McGraw-Hill. ISBN 978: 174889-174896.
11. Jefferson T, Del Mar CB, Dooley L, Ferroni E, Al-Ansary LA, et al. (2011) Physical interventions to interrupt or reduce the spread of respiratory viruses. Cochrane Database Syst Rev 2011: CD006207.
12. Up to 650 000 people die of respiratory diseases linked to seasonal flu each year (2019) World Health Organization (WHO) (Press release).
13. Key Facts About Influenza (Flu) (2014) Centers for Disease Control and Prevention (CDC).
14. Duben-Engelkirk PG and Engelkirk J (2011) Burton's microbiology for the health sciences (9thed). Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins. Pp: 314.
15. "Types of Influenza Viruses Seasonal Influenza (Flu) (2018) Centers for Disease Control and Prevention (CDC).
16. Su S, Fu X, Li G, Kerlin F, Veit M (2017) Novel Influenza D virus: Epidemiology, pathology, evolution and biological characteristics. Virulence 8: 1580-1591.
17. Call S, Vollenweider M, Hornung C, Simel D, McKinney W (2005) Does this patient have influenza?. JAMA 293: 987-997.
18. Flu Symptoms & Diagnosis (2020) Centers for Disease Control and Prevention (CDC).
19. Flu Symptoms & Complications (2019) Centers for Disease Control and Prevention (CDC).
20. King AM, Lefkowitz E, Adams MJ, Carstens EB (2017) Taxonomy of Viruses. 9th Report of the International Committee on. Oxford: Elsevier. Pp: 806-828.
21. International Committee on Taxonomy of Viruses (2010) ICTV Master Species List 2009-v10" (xls).
22. Sexton NR, Smith EC, Blanc H, Vignuzzi M, Peersen OB, et al. (2016) Homology-Based Identification of a Mutation in the Coronavirus RNA-Dependent RNA Polymerase That Confers Resistance to Multiple Mutagens. Journal of Virology 90: 7415-7428.
23. Wertheim JO, Chu DK, Peiris JS, Kosakovsky Pond SL, Poon LL (2013) A case for the ancient origin of coronaviruses. J Virol 87: 7039-7045.
24. Woo PC, Lau SK, Lam CS, Lau CC, Tsang AK, et al. (2012) Discovery of seven novel Mammalian and avian coronaviruses in the genus deltacoronavirus supports bat coronaviruses as the gene source of alphacoronavirus and betacoronavirus and avian coronaviruses as the gene source of gammacoronavirus and delta coronavirus. J Virol 86: 3995-4008.
25. Bidokhti MR, Tråvén M, Krishna NK, Munir M, Belák S, et al. (2013) Evolutionary dynamics of bovine coronaviruses: natural selection pattern of the spike gene implies adaptive evolution of the strains. J Gen Virol 94: 2036-2049.
26. Vijgen L, Keyaerts E, Moës E, Thoelen I, Wollants E, et al. (2005) Complete genomic sequence of human coronavirus OC43: molecular clock analysis suggests a relatively recent zoonotic coronavirus transmission event. Journal of Virology 79: 1595-1604.
27. Lau SK, Lee P, Tsang AK, Yip CC, Tse H, et al. (2011) Molecular epidemiology of human coronavirus OC43 reveals evolution of different genotypes over time and recent emergence of a novel genotype due to natural recombination. J Virol 85: 11325-11337.
28. Lau SK, Li KS, Tsang AK, Lam CS, Ahmed S, et al. (2013) Genetic characterization of Betacoronavirus lineage C viruses in bats reveals marked sequence divergence in the spike protein of pipistrellus bat coronavirus HKU5 in Japanese pipistrelle: implications for the origin of the novel Middle East respiratory syndrome coronavirus. J Virol 87: 8638-8650.
29. Huynh J, Li S, Yount B, Smith A, Sturges L, et al. (2012) Evidence supporting a zoonotic origin of human coronavirus strain NL63. J Viro l86: 12816-12825.
30. Vijaykrishna D, Smith GJ, Zhang JX, Peiris JS, Chen H, et al. (2007) Evolutionary insights into the ecology of coronaviruses. J Virol 81: 4012-4020.
31. MeriadegAr G, Puechmaille SJ, Gonzalez, JP, Emma T, Pattamaporn K, et al. (2011) SARS-Coronavirus ancestor's foot-prints in South-East Asian bat colonies and the refuge theory". Infection, Genetics and Evolution 11: 1690-1702.
32. Cui J, Han N, Streicker D, Li G, Tang X, et al. (2007) Evolutionary relationships between bat coronaviruses and their hosts. Emerging Infectious Diseases 13: 1526-1532.
33. Liu S, Kang J, Chen J, Tai D, Jiang W, et al. (2009) Panorama phylogenetic diversity and distribution of type A influenza virus. PLoS ONE 4: 1-20.
34. Scholtissek C (1995) Molecular evolution of influenza viruses. Virus Genes 11: 209-215.
35. Peng J, Yang H, Jiang H, Lin YX, Lu CD, et al. (2014) The origin of novel avian influenza A (H7N9) and mutation dynamics for its human-to-human transmissible capacity. PLoS ONE 9: e93094.

Citation: Niknamian S and Zaminpira S (2020) The Historical/Evolutionary Cause and Possible Treatment of Pandemic Covid-19 (Sars-Cov-2, 2019-Coronavirus). Ann Med &Surg Case Rep: AMSCR-100050

36. Clancy S (2008) Genetics of the influenza virus. Nature Education 1: 83.

37. LevittEB (1999) Plague of Athens: Another Medical Mystery Solved at University of Maryland. University of Maryland Medical Center.

38. Papagrigorakis MJ, Yapijak C, Synodinos PN, Baziotopoulou-Valavani E (2006) DNA examination of ancient dental pulp incriminates typhoid fever as a probable cause of the Plague of Athens. Int J Infect Dis 10: 206-214.

39. Olson PE; Hames CS; Benenson AS; Genovese EN (1996) The Thucydides syndrome: Ebola déjà vu? (or Ebola reemergent?). Emerging Infect. Dis 2: 155-156.

40. Stathakopoulos DCH (2007) Famine and Pestilence in the late Roman and early Byzantine Empire. Pp: 432.

41. Rosen W (2007) Justinian's Flea: Plague, Empire, and the Birth of Europe. Pp: 3.

42. Andrew E (2007) Byzantine Rome and the Greek Popes. Lexington Books. Pp: 358.

43. Shahraki AH, Carniel E, Mostafavi E (2016) Plague in Iran: Its history and current status. Epidemiol Health 38: e2016033.

44. Maddicott JR (1997) Plague in seventh century England. Past & Present 156: 7-54.

45. Suzuki A (2011) Smallpox and the epidemiological heritage of modern Japan: Towards a total history. Med Hist 55: 313-318.

46. Princeton, New Jersey: Checkmark Books. Pp: 213.

47. Turner D (1990) The Politics of Despair: The Plague of 746-747 and Iconoclasm in the Byzantine Empire1. Annual of the British School at Athens 85: 419-434.

48. Austin Alchon S (2003) A pest in the land: new world epidemics in a global perspective. University of New Mexico Press. Pp: 21.

49. Heyman P, Simons L, Cochez C (2014) Were the English Sweating Sickness and the Picardy Sweat Caused by Hantaviruses?. Viruses 6: 151-171.

50. American plague (2000) New Scientist.

51. Acuna-Soto R, Romero LC, Maguire JH (2000) Large epidemics of hemorrhagic fevers in Mexico 1545–1815. The AmJ Trop Med Hyg 62: 733-739.

52. Acuna-Soto R, Stahle DW, Cleaveland MK, Therrell MD (2002) Mega drought and Mega death in 16th Century Mexico". Emerg Infect Dis 8: 360-362.

53. Vågene ÅJ, Herbig A, Campana MG, Robles G, Nelly M, et al.(2018) Salmonella enterica genomes from victims of a major sixteenth-century epidemic in Mexico. Nature Ecology & Evolution. 2: 520-528.

54. Payne SG (2014) A History of Spain and Portugal. Pp: 382.

55. Griffing SM; Gamboa D, Udhayakumar V (2013) The history of 20th century malaria control in Peru. Malaria Journal 12: 303.

56. Bell WG (2004) The great Plague in London-Folio society by arrangement with Random House. Pp: 3-5.

57. Marr JS and Cathey JT (2010) New Hypothesis for Cause of Epidemic among Native Americans, New England, 1616-1619. Emerging Infectious Diseases 16: 281-286.

58. Mann CC (2005) Native intelligence.

59. Hays JN (2005) Epidemics and pandemics their impacts on human history. Santa Barbara, Calif.: ABC-CLIO. Pp: 103.

60. Johansen BE (2015) American Indian Culture: From Counting Coup to Wampum [2 volumes]: From Counting Coup to Wampum. ABC-CLIO. Pp: 88.

61. Newman Kira LS (2012) Shutt up: bubonic plague and quarantine in early modern England. J SocHist 45: 809-834.

62. Timothy B (1999) The Confusions of Pleasure: Commerce and Culture in Ming China. University of California Press. Pp: 163.

63. Rogers DJ, Wilson AJ, Hay SI, Graham AJ (2006) The Global Distribution of Yellow Fever and Dengue. AdvParasitol 62: 181-220.

64. Scasciamacchia S, Serrecchia L, Giangrossi L, Garofolo G, Balestrucci A, et al. (2012) Plague Epidemic in the Kingdom of Naples, 1656-1658. Emerging Infectious Diseases journal 18: 186-188.

65. Berger St (2020) Measles: Global Status: GIDEON Informatics Inc. Pp: 564.

66. Ross D (2011) UK travel and Heritage-Britain Express UK travel guide. The London Plague of 1665.

67. Great Plague of 1665-1666. The National Archives.

68. Jones C (1996) Plague and Its Metaphors in Early Modern France. Representations 53: 97-127.

69. Casey J (1999) Early Modern Spain: A Social History. Psychology Press. Pp: 305.

70. Purvis TL (2014) Colonial America To 1763. Infobase Publishing. Pp: 400.

71. Desjardins B (1996) Demographic Aspects of the 1702–1703 Smallpox Epidemic in the St. Lawrence Valley. Canadian Studies in Population 23: 49-67.

72. Morens DM (2015) The Past Is Never Dead-Measles Epidemic, Boston, Massachusetts, 1713. Emerg Infect Dis 21: 1257-1260.

73. Mazan R, Gagnon A, Desjardins B (2009) The Measles Epidemic of 1714–1715 in New France. Canadian Studies in Population 36: 295-323.

74. Devaux CA (2013) Small oversights that led to the Great Plague of Marseille (1720-1723): Lessons from the past. Infect Genet Evol 14: 169-185.

75. Cook ND and Lovell WG (2001) Secret Judgments of God: Old World Disease in Colonial Spanish America. University of Oklahoma Press. Pp: 127.

76. Gagnon A and Mazan R (2009) Does exposure to infectious diseases in infancy affect old-age mortality? Evidence from a pre-industrial population. Social Science & Medicine. 68: 1609-1616.

77. Krebsbach S (1996) The Great Charlestown Smallpox Epidemic of 1760. The South Carolina Historical Magazine 97: 30-37.

78. Tognotti E (2013) Lessons from the History of Quarantine, from Plague to Influenza A. Emerg Infect Dis 19: 254-259.

79. LeMay MC (2016) Global Pandemic Threats: A Reference Handbook: A Reference Handbook. ABC-CLIO. Pp: 227.

80. Ranlet P (2000) The British, the Indians, and Smallpox: What Actually Happened at Fort Pitt in 1763?. Pa His 67: 427-441.

81. Greg L (2008) Smallpox epidemic ravages Native Americans on the northwest coast of North America in the 1770s.

Citation: Niknamian S and Zaminpira S (2020) The Historical/Evolutionary Cause and Possible Treatment of Pandemic Covid-19 (Sars-Cov-2, 2019-Coronavirus). Ann Med &Surg Case Rep: AMSCR-100050

82. Prichard A and Fothergill J (1894) Influenza in 1775. The Lancet 143: 175-176.

83. Rohé GH and Robin A (1908) Text-book of Hygiene: A Comprehensive Treatise on the Principles and Practice of Preventive Medicine from an American Standpoint. Davis. Pp: 428.

84. Houston CS and Houston S (2000) The first smallpox epidemic on the Canadian Plains: In the fur-traders' words. Can J Infect Dis 11: 112-115.

85. Waldman C and Braun M (2009) Atlas of the North American Indian. Infobase Publishing. Pp: 295.

86. Warren C (2013) Smallpox at Sydney Cove- who, when, why?. Journal of Australian Studies. 38: 68-86.

87. Tiger mosquitoes and the history of yellow fever and dengue in Spain.

88. Davidson A (1894) Hygiene & diseases of warm climates. Journal of Mental Science 40: 286-287.

89. Odessa (1812) Plague and Tyranny at the Edge of the Empire.

90. Hays JN (2005) Epidemics and pandemics: their impacts on human history. ABC-CLIO 1: 513.

91. Chisholm H (1911) Yellow Fever. Encyclopædia Britannica. 28 (11thed). Cambridge University Press. Pp: 910-911.

92. Aboriginal Health History.

93. Barry L (2014) South Australian History Timeline (19th Century).

94. Kuhnke L (1990) Lives at Risk: Public Health in Nineteenth-Century Egypt. ark.cdlib.org Archived 2008-11-20 at the Wayback Machine, Berkeley: University of California Press.

95. Gallagher N (1990) Egypt's Other Wars: Epidemics and the Politics of Public Health. Syracuse University Press, c1990. Published by the American University in Cairo Press. Pp: 248.

96. Wilford JN (2008) How Epidemics Helped Shape the Modern Metropolis". The New York Times.

97. www.nlm.nih.gov .

98. Gallagher CSSR and John A (1936) The Irish Emigration of 1847 and Its Canadian Consequences. CCHA 3: 43-57.

99. Peacock TB (1849) On the Influenza, or Epidemic Catarrhal Fever of 1847-8. Pp: 182.

100. Daly WJ (2008) The Black Cholera Comes to the Central Valley of America in the 19th Century -1832, 1849, and Later. TransAm Clin and ClimatolAssoc 119: 143-153.

101. Yandell DW (2018) The American Practitioner 1877. Monthly Journal of Medicine and Surgery. Pp: 396.

102. John S (1855) On the mode of communication of cholera. John Churchill.

103. Pryor EG (1975) The great plague of Hong bong. Journal of the Hong Kong Branch of the Royal Asiatic Society. 15: 61-70.

104. Australian Medical Pioneers Index (AMPI) - Colonial Medical Life.

105. Beveridge WIB (1978) Influenza, the Last Great Plague an Unfinished Story of Discovery. Epidemiology. Pp: 132.

106. Swanky T (2016) The Smallpox War in Nuxalk Territory. British Columbia, Canada: Dragon Heart. Pp: 93.

107. https://www.1902encyclopedia.com/

108. https://www.infobae.com/def/desarrollo/2020/03/28/la-fiebre-amarilla-una-epidemia-que-revelo-lo-peor-de-buenos-aires/

109. https://archive.org/details/EncyclopaediaBritannicaDict.a.s.l.g.i.11thed.chisholm.1910-1911-1922.33vols/page/n9/mode/2up

110. Great B (1893) Further report and papers on epidemic influenza, 1889–92: with an introduction by the medical officer of the Local Government Board. Eyre. Pp: 154.

111. World Health Organization (2018) Human African trypanosomiasis.

112. Echenberg M (2007) Plague Ports: The Global Urban Impact of Bubonic Plague: 1894-1901. Sacramento: New York University Press. Pp: 231.

113. Fevre EM, Coleman PG, Welburn SC, Maudlin I (2004) Reanalyzing the 1900-1920 sleeping sickness epidemic in Uganda". Emerg Infect Dis 10: 567-573.

114. Texas Department of State Health Services, History of Plague-Plague Through the Ages.

115. Watson WA (1903) Report on the outbreak of plague at Fremantle.

116. Foster HD and Hoffer A (2007) Hyperoxidation of the Two Catecholamines, Dopamine and Adrenaline: Implications for the Etiologies and Treatment of Encephalitis Lethargica, Parkinson's Disease, Multiple Sclerosis, Amyotrophic Lateral Sclerosis, and Schizophrenia". Oxidative Stress and Neurodegenerative Disorders. Elsevier Science BV. Pp: 369-382.

117. Patterson KD and Pyle GF (1991) The geography and mortality of the 1918 influenza pandemic. Bulletin of the History of Medicine 65: 4-21.

118. Ravenel MP (1938) The Croydon Epidemic of Typhoid Fever. Am J Public Health Nations Health 28: 644-646.

119. Paul WE (2003) Fundamental immunology. Lippincott Williams & Wilkins. Pp: 1701.

120. Spross Å (2018) Såkanvaccinutrotasjukdomar" (in Swedish). Uppsala Nya Tidning.

121. World Health Organization (2013) Public Health Agency of Sweden.

122. https://www.anderetijden.nl/programma/1/Andere-Tijden/aflevering/340/Polio-in-Staphorst

123. www.smallpoxhistory.ucl.ac.uk.

124. UNAIDS REPORT ON THE GLOBAL AIDS EPIDEMIC (2010) Report on the global AIDS epidemic.

125. Dutt AK, Akhtar R, McVeigh M (2006) Surat Plaque of 1994 re-examined. Southeast Asian J Trop Med Public Health 37: 755-760.

126. Looi LM and Chua KB (2007) Lessons from the Nipah virus outbreak in Malaysia. Malays J Pathol29: 63-67.

127. Dengue in the Americas: The Epidemics of 2000.

128. Nigeria cholera outbreak kills 400.

129. Cholera Spreads Through South Africa Townships.

130. www.ncbi.nlm.nih.gov

131. How Hong Kong Beat SARS: Lessons Learned.

132. Hsieh YH, King CC, Chen CW, Ho MS, Lee JY, et al. (2005) Quarantine for SARS, Taiwan". Emerg Infect Dis 11: 278-282.

Citation: Niknamian S and Zaminpira S (2020) The Historical/Evolutionary Cause and Possible Treatment of Pandemic Covid-19 (Sars-Cov-2, 2019-Coronavirus). Ann Med &Surg Case Rep: AMSCR-100050

133. Tam T (2018) Fifteen years post-SARS: Key milestones in Canada's public health emergency response. Can Commun Dis Rep 44: 98-101.

134. World Health Organization (2003) Severe Acute Respiratory Syndrome.

135. Bertherat E, Bekhoucha S, Chougrani S, Razik F, Duchemin JB, et al. (2007) Plague Reappearance in Algeria after 50 Years, 2003. Emerg Infect Dis 13: 1459-1462.

136. World Health Organization action in Afghanistan aims to control debilitating leishmaniasis.

137. Faruque SM, Islam MJ, Ahmad QS, FaruqueASG, Sack DA, et al.(2005) Self-limiting nature of seasonal cholera epidemics: Role of host-mediated amplification of phage. ProcNatl AcadSci USA 102: 6119-6124.

138. www.who

139. Cholera Outbreak in Senegal in 2005: Was Climate a Factor? "Ebola virus disease" (Press release). World Health Organization.

140. Yellow fever epidemic in Kayes.

141. Koh BK, Ng LC, Kita Y, Tang CS, Ang LW, et al. (2008) The 2005 dengue epidemic in Singapore: Epidemiology, prevention and control. Ann Acad Med Singapore 37: 538-545.

142. Worst cholera outbreak in Angola. Machine, BBC.

143. www.who.int

144. Malaria Epidemic Sweeps Northeast India.

145. Dengue epidemic threatens India's capital.

146. World Health Organization (2006) Chikungunya in India.

147. Khan E, Siddiqui J, Shakoor S, Mehraj V, Jamil B, et al. (2007) Dengue outbreak in Karachi, Pakistan, 2006: Experience at a tertiary care center. Trans R Soc Trop Med Hyg. 101: 1114-1119.

148. www.researchgate.net

149. Mourners die as fever grips Congo.

150. Xan Rice (2007) Fatal outbreak not a cholera epidemic, insists Ethiopia.

151. Cholera death toll in India rises. BBC News.

152. Cholera outbreak in Iraq growing. Associated Press.

153. Vaccine-linked polio hits Nigeria. BBC News.

154. Dengue fever epidemic hits Caribbean, Wayback Machine, Reuters.

155. Somalia cholera death fears grow.

156. Thousands hit by Brazil outbreak of dengue. CNN.com

157. Cambodia suffers worst dengue epidemic, 407 dead, Wayback Machine. Reuters.

158. Cholera epidemic in western Chad kills 123.

159. Huang J, Liao Q, Ooi MH, Cowling BJ, Chang Z (2018) Epidemiology of Recurrent Hand, Foot and Mouth Disease, China, 2008-2015. Emerging Infectious Diseases 24: 432-442.

160. Dengue cases in Philippines rise by 43 percent: government.

161. Cholera Country Profile: Zimbabwe. World Health Organization - Global Task Force on Cholera Control. 31 October 2009.

162. www.ncbi.nlm.nih.gov

163. McCredie J (2009) Dengue fever epidemic hits northern Australia. BMJ 338: b967.

164. Odigwe C (2009) West Africa has worst meningitis epidemic for 10 years. BMJ 338: b1638.

165. First Global Estimates of 2009 H1N1 Pandemic Mortality Released by CDC-Led Collaboration. Centers for Disease Control and Prevention. 2012-06-25.

166. 2009 Swine-Flu Death Toll 10 Times Higher Than Thought. LiveScience.com.

167. www.mspp.gouv.ht

168. www.npr.org

169. Democratic Republic of Congo: More measles vaccinations needed. Médecins Sans Frontières (MSF) International.

170. Vietnam on alert as common virus kills 81 children.

171. Nuyen NTB; Pham HV, Hoang CQ, Nguyen TM, Nguyen LT, et al. (2014) Epidemiological and clinical characteristics of children who died from hand, foot and mouth disease in Vietnam, 2011. BMC Infect Dis. 14: 341.

172. www.emro.who.int

173. Yuill TM, Woodall JP, Baekeland S (2013) Latest outbreak news from ProMED-mail. Yellow fever outbreak-Darfur Sudan and Chad". International Journal of Infectious Diseases. 17: e476-e478.

174. Donnelly CA, Malik MR, Elkholy A, Cauchemez S, Van Kerkhove MD (2019) Worldwide Reduction in MERS Cases and Deaths since 2016. Emerg Infect Dis 25: 1758-1760.

175. World Health Organization (2019) Middle East Respiratory Syndrome coronavirus (MERS-CoV).

176. World Health Organization (2018) Middle East respiratory syndrome coronavirus (MERS-CoV)-United Arab Emirates.

177. Vietnam measles outbreak kills more than 100 people, mostly children. Sydney Morning Herald.

178. www.cdc.gov

179. World Health Organization (2016) Situation summary Latest available situation summary.

180. Gignoux E, Idowu R, Bawo L, Hurum L, Sprecher A, et al. (2015) Use of Capture-Recapture to Estimate Underreporting of Ebola Virus Disease, Montserrado County, Liberia. Emerg Infect Dis 21: 2265-2267.

181. Pan American Health Organization (2015) Number of reported cases of epidemic chikungunya arthritis in the Americas.

182. World Health Organization (2017) Plague-Madagascar.

183. Bhubaneswar (2015) Odisha grapples with jaundice outbreak. Deccan Herald.

184. Press Trust of India (2015) Swine flu deaths at 1895; number of cases near 32K mark. The Indian Express.

185. India struggles with deadly swine flu outbreak. BBC News.

186. Kindhauser MK, Allen T, Frank V, Santhana RS, Dye C (2017) Zika: the origin and spread of a mosquito-borne virus. World Health Organization.

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187. Yellow fever-countries with dengue. ProMED-mail. International Society for Infectious Diseases.

188. WHO Regional Office for the Eastern Mediterranean Cholera Situation in Yemen November 2019.

189. www.indiatvnews.com

190. Nipah virus contained, last two positive cases have recovered: Kerala Health Min. The News Minute.

191. Operations Dashboard for ArcGIS.

192. World Health Organization. Ebola Virus Disease Outbreak Uganda Situation Reports.

193. DR Congo measles: More than 6,000 dead in world's worst outbreak". BBC News.

194. Two more deaths from measles in samoa over new year period. Radio New Zealand.

195. Tracking coronavirus: Map, data and timeline. Bnonews.

196. www.medixocentre.com

197. McKie R (2017) Scientists trace 2002 Sars virus to colony of cave-dwelling bats in China. The Guardian.

198. Corman VM, Muth D, Niemeyer D, Drosten C (2018) Hosts and Sources of Endemic Human Coronaviruses. Adv Virus Res 100: 163-168.

199. Worobey M and Holmes EC (1999) Evolutionary aspects of recombination in RNA viruses. J GenVirol. 80:2535-2543.

200. Lukashev AN (2005) Role of recombination in evolution of enteroviruses. RevMed Virol 15: 157-167.

201. Umene K (1999) Mechanism and application of genetic recombination in herpesviruses. Rev Med Virol 9: 171-182.

202. Sandbulte MR, Westgeest KB, Gao J, Xu X, Klimov AI, et al. (2011) Discordant antigenic drift of neuraminidase and hemagglutinin in H1N1 and H3N2 influenza viruses. Proc Natl AcadSci USA 108: 20748-20753.

203. Moss RB, Davey RT, Steigbigel RT, Fang F (2010) Targeting pandemic influenza: a primer on influenza antivirals and drug resistance. The Journal of Antimicrobial Chemotherapy 65: 1086-1093.

204. Barr JN and Fearns R (June 2010) How RNA viruses maintain their genome integrity. J Gen Virol 91: 1373-1387.

205. Su S, Wong G, Shi W, Liu J, Lai AC, Zhou J, et al. (June 2016) Epidemiology, Genetic Recombination, and Pathogenesis of Coronaviruses. Trends Microbiol 24: 490-502.

206. Wu F, Zhao S, Yu B, Chen YM, Wang W, et al. (2020) A new coronavirus associated with human respiratory disease in China. Nature 579: 265-269.

207. Galton F (1889) Natural Inheritance. Macmillan.

208. www.galtonboard.com

209. Nicola Davison (2017) Why can't we cure the common cold?. The Guardian.

210. Jacobs SE, Lamson DM, St George K, Walsh TJ (2013) Human rhinoviruses. Clinical Microbiology Reviews 26: 135-162.

211. Robert B Couch (2005) Rhinoviruses: Replication. Encyclopedia of Life Sciences. John Wiley.

212. Rossmann MG, Arnold E, Erickson JW, Frankenberger EA, Griffith JP, Hecht HJ, et al. (1985) Structure of a human common cold virus and functional relationship to other picornaviruses. Nature 317: 145-153.

213. Smith TJ, Kremer MJ, Luo M, Vriend G, Arnold E, Kamer G, et al. (1986) The site of attachment in human rhinovirus 14 for antiviral agents that inhibit uncoating. Science 233: 1286-1293.

214. Farr BM, Gwaltney JM, Adams KF, Hayden FG (July 1984) Intranasal interferon-alpha 2 for prevention of natural rhinovirus colds". Antimicrob Agents and Chemother 26: 31-34.

215. Mousnier A, Bell AS, Swieboda DP, Morales-Sanfrutos J, Pérez-Dorado I, et al. (2018) Fragment-derived inhibitors of human N-myristoyltransferase block capsid assembly and replication of the common cold virus. NatChem 10: 599-606.

216. Mary E (2009) Rhinovirus strains' genomes decoded; cold cure-all is unlikely: The strains are probably too different for a single treatment or vaccine to apply to all varieties, scientists say. Los Angeles Times.

217. Katpally U, Fu TM, Freed DC, Casimiro DR, Smith TJ (2009) Antibodies to the buried N terminus of rhinovirus VP4 exhibit cross-serotypic neutralization. Jo Virol 83: 7040-7048.

218. Carroll L (1991) 2: The Garden of Live Flowers". Through the Looking-Glass.

219. Van Valen L (1973) A new evolutionary law. Evolutionary Theory 1: 1-30.

220. Bell G (1982) The Masterpiece of Nature: The Evolution and Genetics of Sexuality. University of California Press, Berkeley. Pp: 378.

221. Magain N and Sérusiaux E (2014) Do photobiont switch and cephalodia emancipation act as evolutionary drivers in the lichen symbiosis? A case study in the Pannariaceae (Peltigerales). PLoS ONE 9: e89876.

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