Original Article

Investigating and combatting the key drivers of viral zoonoses in Africa: an analysis of eight epidemics

Investigar e combater os principais promotores de zoonoses virais na África: uma análise de oito epidemias

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Abstract

Investigating the interplay of factors that result in a viral zoonotic outbreak is difficult, though it is increasingly important. As anthropogenic influences shift the delicate balance of ecosystems, new zoonoses emerge in humans. Sub-Saharan Africa is a notable hotspot for zoonotic disease due to abundant competent mammalian reservoir hosts. Furthermore, poverty, corruption, and an overreliance on natural resources play considerable roles in depleting biological resources, exacerbating the population's susceptibility. Unsurprisingly, viral zoonoses have emerged in Africa, including HIV/AIDS, Ebola, Avian influenza, Lassa fever, Zika, and Monkeypox. These diseases are among the principal causes of death in endemic areas. Though typically distinct in their manifestations, viral zoonoses are connected by underlying, definitive factors. This review summarises vital findings on viral zoonoses in Africa using nine notable case studies as a benchmark for future studies. We discuss the importance of ecological recuperation and protection as a central strategy to control zoonotic diseases. Emphasis was made on moderating key drivers of zoonotic diseases to forestall future pandemics. This is in conjunction with attempts to redirect efforts from reactive to pre-emptive through a multidisciplinary "one health" approach.

Keywords: zoonoses, epidemic, pandemic, omicron, ecological restoration, HIV, Ebola, Lassa fever, monkeypox, Rift Valley fever, West Nile virus.

Resumo

Investigar a interação de fatores que resultam em um surto zoonótico viral é difícil, embora seja cada vez mais relevante. À medida que as influências antropogênicas mudam o delicado equilíbrio dos ecossistemas, novas zoonoses surgem em humanos. A África Subsaariana é um ponto crítico notável para doenças zoonóticas devido a abundantes reservatórios mamíferos competentes. Além disso, a pobreza, a corrupção e o excesso de confiança nos recursos naturais desempenham papéis consideráveis no esgotamento dos recursos biológicos, exacerbando a suscetibilidade da população. Sem surpresa, zoonoses virais surgiram na África, incluindo HIV/AIDS, Ebola, gripe aviária, febre de lassa, zika e varíola dos macacos. Essas doenças estão entre as principais causas de morte em áreas endêmicas. Apesar de serem tipicamente distintas em suas manifestações, as zoonoses virais na África usando nove estudos de caso notáveis como referência para estudos futuros. Discutimos a importância da recuperação e proteção ecológica como estratégia central para o controle de doenças zoonóticas. Foi dada ênfase à moderação dos principais impulsionadores de doenças zoonóticas para prevenir futuras pandemias. Isso ocorre em conjunto com tentativas de redirecionar os esforços de reativos para preventivos por meio de uma abordagem multidisciplinar de "uma só saúde".

Palavras-chave: zoonoses, epidemia, pandemia, ômicron, restauração ecológica, HIV, Ebola, febre de Lassa, varíola dos macacos, febre do Vale do Rift, vírus do Nilo Ocidental.

1. Introduction

An overwhelming portion of human diseases circulate through non-human hosts, and about 60% have been directly linked to zoonotic origins. If phylogeny is traced, it is expected to unearth zoonotic viral transmission as far as 200,000 years ago (Forni et al., 2022). Even so, investigating the interplay of factors that result in a viral zoonotic outbreak is difficult, though its necessity should not be understated (Holmes, 2022). As anthropogenic

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influences shift the delicate balance of ecosystems, new zoonoses emerge in humans. These once-rare events have been on an incline (Williams et al., 2021), with devastating ecological, social, and economic consequences (Chauhan et al., 2020). Sub-Saharan Africa has notable hotspots for zoonotic disease due to abundant competent mammalian reservoir hosts.

Furthermore, poverty, corruption, and an overreliance on natural resources play considerable roles in depleting biological resources, exacerbating the population's susceptibility (Chauhan et al., 2020). By 1986, Africa had lost 65% of its wildlife habitat hotspots of exotic plants and animals, and current estimates report a 50% habitat alteration in 33 African countries, and up to 70% in 20 countries (NCBI, 2021). Significant viral zoonoses have emerged in Africa, including HIV/AIDS, Ebola, Lassa fever, Zika, and Monkeypox (Brady et al., 2012; Bhatt et al., 2013; Berry et al., 2020; Zhu et al., 2020). Azuma et al. (2020) reported on case studies of viral zoonoses in Africa, including the year it was first recorded and the mortality rate. HIV was first reported in 1980 with a mortality rate of 10,700,000, Ebola in 1976 with 12,930, Monkeypox in 1970 with 5,000, Rift Valley fever in 1977 with 3,000, West Nile virus in 1999 with 2,330, Zika virus in 1947 with 50, Lassa fever in 1969 with 250,000, and COVID-19 in 2020 with 4,000,000 (Ceballos et al., 2015).

These diseases are among the principal causes of death in endemic areas (WHO, 2020; Cavalerie et al., 2021). Though typically distinct in their manifestations, viral zoonoses are connected by underlying, definitive factors (Visher et al., 2021). Other authors have attempted to link the occurrences of zoonotic outbreaks in Africa to establish causality (Smith et al., 2003; Al-Tawfiq and Memish, 2014; Kansky et al., 2016; NCBI, 2021). However, the wealth of information, already skewed with socio-economic biases, grossly understates the African situation (Altizer et al., 2011). This review summarises vital findings on viral zoonoses in Africa using eight notable case studies as a benchmark for future studies (Akinduti et al., 2021; Akinduti et al., 2022). We propose two practical steps to alleviate, control and perhaps, prevent future outbreaks within the continent. Our contribution is valuable to prior attempts to redirect zoonotic disease control efforts from reactive to pre-emptive through a multidisciplinary "one health" approach (Amenu et al., 2019; Williams et al., 2021; Obafemi et al., 2021).

2. Theories of Zoonotic Emergence

According to a fact sheet documented by WHO (2020), zoonotic diseases (zoonosis) are infectious diseases caused by pathogens migrating and rapidly circulating through the human population from different species. Though viruses are ubiquitous, only an estimated 0.001% constitute significant health and economic concerns to man and his environment (Visher et al., 2021). Several forces amplifying this minority include viral disease etiology, epidemiology, host biochemistry, and distribution. The exact model in which this occurs is still a subject of scientific speculation as particular details may vary between pathogens and outbreak episodes (Andersen et al., 2015; Azuma et al., 2020). Current predominant hypotheses offer solutions to two central questions: What is the origin of viral zoonotic outbreaks? Secondly, what forces influence their adaptation and outbreak in new populations?

2.1. Spillover model and circulation model

The origin of viral zoonotic outbreaks is a primary contention in zoonotic studies. Scientists have investigated the timeline and phylogeny of zoonotic pathogens, and several have linked them to animal populations, especially birds, bats, rodents, and monkeys (Kansky et al., 2016; Baud et al., 2020). Lassa virus is linked to multimammate rats: Mastomys natalensis (Wille et al., 2021). HIV-1 is adapted from the Simian Immunodeficiency Virus in Rhesus macaques and Sooty mangabeys (Weldemhret, 2021). Zika Virus was initially isolated from a febrile Rhesus macaque (Pierson and Diamond, 2018). Several studies also implicate non-human primates as the reservoir of diseases like Monkeypox, Ebola, and COVID-19 (Bausch and Schwarz, 2014; Lu et al., 2015; Jin et al., 2021; Obafemi et al., 2021; Akinduti et al., 2021; Akinduti et al., 2022). Thus, before or during human outbreaks, the enzootic and sylvatic cycles of several zoonotic viruses have been thoroughly demonstrated.

Negative laboratory tests indicate that several organisms, genetically similar and dissimilar to man, react diversely to human-adapted viral strains (Bhowmick et al., 2020; Williams et al., 2021). Zika Virus strains isolated from humans tested in bats, mice, and rats resulted in a less than 2 log increase in viral load (Bolles et al., 2011). Horses experimentally infected with the NY99 strain of West Nile virus were asymptomatic, failing to develop the viral load necessary for transmission (Bongaarts, 2009; Briggs et al., 2010). Numerous studies like this diminish the probability of particular lineages of viral zoonoses. Without notable adaptation, rapid propagation of viruses in all living species is unlikely. Therefore, from an anthropocentric perspective, tracing a virus's origin leaves room for fewer explanations. They either emerged from a singular interspecies transmission or as a recombination of several inter- and intraspecies transmissions.

The concept of diseases existing in distinct species populations can be traced before man to the "Spillover model" described by Daszak et al. (2000) represented a phenomenon where an infection circulates within a particular species before inadvertently overflowing into others. The spillover effect is a population-scale interpretation of disease transmission. It alludes to the presence of a threshold the virus must overcome before interspecies transmission occurs (Brink and Eva, 2009). This closed system approach may be helpful when describing a community of cells that constitute a singular organism, but not in the context of multi-individual living communities. The primary critique of this model is that its linear approach rarely accurately captures the nuances of "chance" present in nature (Burniston et al., 2015). Identifying a singular trans-species event that precedes a "Spillover" might be tedious at best.

Genetic and physiological variations between species, described in this context as "species barriers," may require viruses to adapt to new populations. Earlier infections may not necessarily translate into disease if resolved by the immune system. Even when diseases occur, symptoms may be absent or poorly identified where present. Unidentifiable incidents like this are often left unresolved or misdiagnosed (Butler, 2012; Wille et al., 2021). In poorer continents like Africa, such occasions may not even be reported (Butler, 2012). Viruses probably circulate within the population long before they are considered or characterised (Visher et al., 2021). Within the open system of natural habitat, anthropogenic activities interfere with organisation, so infected non-human hosts may more readily contact other species, including humans. This is the basis of what Frutos et al. (2021) described as the "circulation model". This model posits that divergent viral sub-populations evolve independently within organisms of different species encountered randomly. Repeated infections, which may not result in diseases, constitute the stuttering phase of an outbreak and may persist until the virus attains optimal virulence and transmissibility. An outbreak occurs if this unlikely event coincides with a hotspot of susceptible species.

2.2. Dilution, amplification and coevolution

Understanding the forces that influence viral adaptation in new populations is fundamental and imperative. When viruses migrate into novel populations, survival necessitates evolution (Lion and Metz, 2018). Events of viral adaptations can be navigated using key hypotheses such as "the dilution effect" (Chlebicz and Śliżewska, 2018; CDC, 2020), "the amplification effect" (Colwell et al., 2008; CBD, 2017), and "the coevolution effect" (Dietrich et al., 2015; Engineering and Technology, 2020). The dilution effect proposes that the likelihood of disease outbreaks is limited or hindered in greater species diversity. It suggests that fragmented habitats support the evolution of pathogens (Dudas et al., 2017) due to reduced biodiversity.

Conversely, the amplification effect proposes that species diversity enhances pathogenic evolution and the outbreak of diseases (Ceballos et al., 2015). This is based on the rationale that higher species diversity increases the likelihood of a suitable host for given zoonoses. According to Lemieux et al. (2022), both theories act in tandem with reality to produce a net diluted or amplified effect. The coevolution effect hypothesised by Zohdy et al. (2019) proposes that within the diverging communities created by habitat fragmentation, obligate parasites and hosts facilitate the evolution of pathogens. This enhances their overall diversity and virulence, thereby reducing spillover. The subtle effect of competing factors in individual ecosystems (including host-pathogen ecophysiology, the composition of reformed communities, and the study scale) affects all findings in this area of study. This complicates the process of pinpointing causality.

3. Overview of Emergence Timeline

The Human Immunodeficiency Virus has persisted in Africa for centuries, where it existed amongst primates (Chimpanzees and Gorillas) as Simian Immunodeficiency Virus (SIV) (Sharp and Hahn, 2011). This intra-specie transfer eventually led to the outbreak from 1938 to 1960 (Sharp and Hahn, 2011; Sousa et al., 2017). HIV-1-M and HIV-1-N, in particular, can be traced from south-central and southeast Cameroonian Chimpanzees, while HIV-1-O HIV-1-P from gorillas in Western lowlands (Sousa et al., 2017). HIV 2 has been generally linked to independent transmission from Mangabey monkeys in West Africa. Significant overlap between HIV and SIV geographic distribution strongly indicates multiple subspecies transfer (Baral and Phaswana-Mafuya, 2012). Some studies have suggested that these transfers began in the Congo Basin River in Africa (Barboza et al., 2018; Everard, 2020)

Monkeypox is an Orthopoxvirus first clinically documented in 1958 from skin lesions believed to have originated from non-human primates (Everard et al., 2020). The first clinical report was of an infant in the Demographic Republic of Congo in 1970, and the disease subsequently spread sporadically around the country, as well as many others in Central and West Africa (Gottwalt, 2013; Golin et al., 2020). Subsequent outbreaks occurred in the US and globally in 2003. This was linked to a cross mutation from the vaccine for smallpox, as both are of the Poxviridae family and are closely related genetically (Gould and Higgs, 2009). Reports state that smallpox vaccination was at least 85% effective against monkeypox (Gugnani and Muotoe-Okafor, 1997; Grace et al., 2012). Before the 2010s, with more infections than seen in the last half-century, monkeypox occurrences have been few and far between (Guine et al., 2021). However, cases have been repeatedly reported to show a gradual rise since 1970 (Hudson et al., 2002; Guzman et al., 2016). Within the last decade, 98.5% of confirmed and reported monkeypox cases occurred in the Demographic Republic of Congo, with the rest distributed between Nigeria, Cameroon, the Central African Republic, Congo and South Sudan (Shuman, 2010; Jaramillo et al., 2019; Sijtsma et al., 2020). The biggest singular outbreak occurred in Nigeria in 2017 and Cameroon in 2018, which were previously inhabited by the virus, thus raising concerns about new mutations that might have caused its re-emergence. The same strain, with a few novel mutations, was identified earlier this year in non-endemic locations like the United Kingdom and the United States. Investigations regarding this development are undergoing (Kaler et al., 2022).

The Coronaviruses were first isolated in 1966 from a sample culture of invalids with the common cold. Over two decades later, the first four cases of COVID-19 were relayed in Wuhan City, Hubei Province, China, on 29 December 2019 (Saadat et al., 2020). Until recently, the four different subfamilies identified include alpha-, beta-, gamma-, and delta-coronaviruses (Johnson et al., 2015). The beta coronaviruses result in profound diseases, including more mortality than the other genotypes of this virus. In contrast, alpha-coronaviruses cause mild symptoms and can even be asymptomatic in some cases (Kularatne et al., 2018). The B.1.1.529 variation, discovered in South Africa, was fundamentally delineated by the World Health Organization (WHO) on 24 November 2021. A detailed epidemiological

characterisation of the South African situation showed three distinct peaks, the latest predominantly the Delta variant.

The Ebola virus was first isolated in 1976 in the Demographic Republic of Congo and Sudan, with its most significant outbreak occurring in 2014 (Leroy et al., 2007). A few weeks after initial isolation, the first outbreak, with over 318 reported cases of 88% fatality, was documented in Guinea. Over 20 outbreaks occurred in the interim, with a spread of about 1740 confirmed cases (Loh et al., 2013). In 2011, several instances of EBOV were reported in Uganda following the death of one patient that endangered over 20 more (Londono-Renteria et al., 2016). Similar occurrences were documented in Sierra Leone in 2014 through a singular recorded event that led to a series of reported cases (Kreuels et al., 2014). Sporadic cases of EBOV from 2011-2014 were common in West African countries. Before the first quarter of 2014, 111 reported cases and about 79 resulting deaths had been documented in Sub-Saharan Africa, with over 23 total outbreaks (Baize et al., 2014; Shultz et al., 2016). Believed to have been contracted from Savannah forest, the 2014 outbreak is traced to a single report in a Guinean village in December 2013. With a spread of 28,600 confirmed cases in three central African countries-Liberia, Sierra Leone and Guinea, it quickly became a public health emergency several times more severe than all other EBOV outbreaks (Shultz et al., 2016). Since March 2016, when the outbreak was declared curtailed, reports of annual cases have continued to trickle in (Mabogunje, 1995; Strle and Stanek, 2009; Shapiro, 2017).

Lassa virus was popularised after its isolation from infected patients in Borno State, Nigeria, in 1969 and its clinical description in 1970. The outbreak occurred minimally but consistently for 54 years, except from 1981 to 1988 (Agbonlahor et al., 2021). In the succeeding decades, less than 10% of total disease cases were discovered outside West Africa, predominant in several other countries, including Sierra Leone, Liberia, Mali and Guinea (Bonwitt et al., 2016). With a persistent hike since early 2005, the largest outbreak occurred between 2017-2020, affecting over 20 states in Nigeria. This outbreak has been depicted with the lowest fatality of about 4.8% compared to 49-86% of the rates in the epidemics of the early 2000s (Agbonlahor et al., 2021).

Zika virus was isolated from a febrile rhesus monkey in the Zika forest by scientists in 1947 (Schwartz, 2017). The virus was neurotrophic and found to induce illness and death in lab-tested mice (Dick et al., 1952). The first documentation of the zika virus isolated from a human occurred in Nigeria in 1954 (Macnamara, 1954; Tian et al., 2018), where the malaria-like and yellow-fever-like symptoms sparked debates about its validity. It has also raised further questions about the prevailing misdiagnosis of the virus within the continent (Gubler et al., 2017). Zika virus is endemic to African countries but has also been isolated in several parts of Asia (Musso and Gubler, 2016). It was initially isolated outside Africa from Malaysian mosquitos in 1966 and subsequent seroprevalence in Asia between 1950 to 1970 likely indicates its presence. In 2007, the first Zika virus outbreak was reported in Yap Island, Micronesia, preceding the epidemics in Polynesia and South Pacific in 2013-2014 (Duffy et al., 2009; Setti et al.,

2020). Alongside its geographical migration, the virus has evolved into two major lineages linked to numerous global outbreaks in the last ten years (Musso and Gubler, 2016).

West Nile virus was initially isolated in 1937 in Uganda during a Yellow Fever campaign and has since evolved into several lineages discovered in Australia and various European countries (Smithburn et al., 1940). It is a Flavivirus endemic to several Sub-Saharan African, European, Asian and Middle Eastern countries. As it is vector-borne, the virus is greatly influenced by the geographical distribution and migration of the Culex and Aedes mosquitoes (Calistri et al., 2010). Of the nine known lineages of the West Nile virus, only 3: L1, L2 and L8, are found in Botswana, Congo, Senegal, South Africa, Madagascar, Uganda and the Central Republic of Africa (Fall et al., 2017). Phylogenetic research has traced a shared ancestry back to the 16th century (Pachler et al., 2014). In 1960, L1 migrated through Northern and most countries in Central Africa (Sule et al., 2018). Since then, West Nile has caused outbreaks in Algeria (1994), Morocco (1997), and Tunisia (2007). The second and largest outbreak occurred in 2004 in Northern Africa and Hungary, chiefly driven by L1 and L2 (Fall et al., 2017).

The first report of Rift Valley fever occurred following the death of 4,700 livestock on a farm in the Great Rift Valley, Kenya, in 1931 (Daubney et al., 1931), spreading slowly into many countries in Southern Africa (Swanepoel and Coetzer, 2004). Over two decades later, the first outbreak occurred in South Africa, killing over 100,000 livestock, which was recognised after the death of several animal care doctors (Mason, 2016). In 1977, the largest outbreak occurred in Egypt, resulting in over 500 deaths and 200,000 human infections (Meegan et al., 1981; Sambri et al., 2013; Roberts, 2018). In 1997, an outbreak occurred in Kenya and again in Eastern Africa between 2007-2008, resulting in about 700 deaths (Bird et al., 2008). Subsequent episodes in the continent have been few and far between, making the first appearance outside Africa in 2000 (CDC, 2000; Sampathkumar and Sanchez, 2016; Seah and Agrawal, 2020).

4. Drivers of Zoonosis in Africa

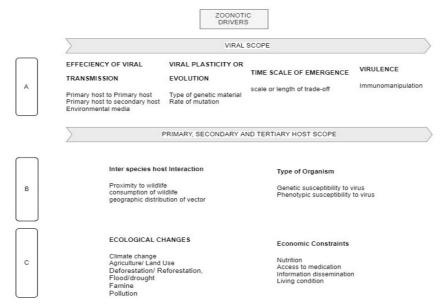
Direct and indirect drivers of African zoonoses can be distinguished into three broad categories: A as "the proximate drivers", B as "the primary distal drivers", and C as "the secondary distal drivers" (Figure 1).

4.1. Viral scope

4.1.1. Viral transmission

From an anthropocentric perspective, three significant players may be considered (excluding the virus) to be involved in viral zoonoses transmission: primary host (non-human species or reservoir), intermediate host (vector) and secondary host (human population). Hence, propagation within the secondary host population typically occurs through four major pathways:

Pathway 1: Primary host to secondary host. Pathway 2: Intermediate host to secondary host. Pathway 3: Secondary host to secondary host.





Pathway 4: Environmental media to a secondary host. Since viruses are rarely constrained within any specific population (Memish et al., 2013), there are likely as many potential hosts as available hosts. Survival within new populations generally requires an adequate infected host interacting, primarily via body fluids, with a vulnerable host. Viruses need efficiency in pathways 2 and 3 to emerge, thus, an evolution to sustain pathway 3 ensures longevity (Mishra et al., 2020). Pathway 3 sustaining viruses are costly to contain and have been linked to lower death rates, and acute infection (Visher et al., 2021). Transmission has contributed significantly to the emergence of each viral case study analysed - this includes both positive and negative influences. The RO value is also used here to characterise transmission efficiency, measuring the average incidence following the introduction of a virus into a new community.

HIV strains are known to infect humans in Africa widely: HIV 1 and HIV 2, both of which have been traced back to independent pathway 1 transmissions (Sharp and Hahn, 2011; Baral and Phaswana-Mafuya, 2012; Jin et al., 2021). The epidemic groups of HIV-1 and 2 all successfully maintain pathway 3, which accounts for 86% of incidences (Royce et al., 1997; Jin et al., 2021). Consequently, HIV has an R0 value of 4.6, surviving for many centuries in human society (Sousa et al., 2017). Viruses that operate through pathway 2 are also dominant in Africa (Kilpatrick and Randolph, 2012; Dzingirai et al., 2017). They are commonly constrained to the geographical location of the vector or reservoir host, but many studies indicate that common arthropod vectors are adequately bred within the continent (Venter, 2018; Mubemba et al., 2022). The success of Rift Valley, West Nile and Zika virus is attributable to this factor (Bird et al., 2008; Gubler et al., 2017; Sule et al., 2018). However, these viruses are not exclusively transmitted this way and often employ Pathways 1 and 4. Monkeypox, Ebola and Lassa virus is commonly associated with pathway 1 and demonstrates higher virulence and lower longevity (Shultz et al., 2016; Kamara et al., 2020; Peter et al., 2022). Rift Valley virus is primarily transmitted via the Aedes mosquitos, though it may also be maintained from man to man by sexual transmission (Pienaar and Thompson, 2013; Mweya et al., 2013; Meegan and Bailey, 2019). It has an R0 value of 2.5 and may replicate continuously in the cells of blood, eyes and sperm. It requires multiple primate and non-primate hosts to complete its life cycle, which severely constrains transmission (Meegan and Bailey, 2019). West Nile and Zika viruses are primarily transmitted through pathway 2, with various species of the Culex and Aedes Mosquitoes. West Nile virus may also be transmitted intravenously during pregnancy and contact with infected body fluids (Córdoba et al., 2007; Watts et al., 2020). Birds are regarded as the reservoir host, so that transition into a secondary host is likely to occur after a mosquito bite a bird with West Nile disease. Horses, birds and man are considered dead-end hosts, as interspecies transmission within these populations is inefficient (Bosco-Lauth and Bowen, 2019). Similarly, the Zika virus undergoes sylvatic and enzootic phases, circulating through primates and the arthropod vector that bites them (Bonyah et al., 2017). The Ebola virus outbreak of 2013-2014 was chiefly a function of pathway 2, which is majorly linked to body fluids. This outbreak was largely successful, with an R0 rate of 1.5-2.5 (WHO, 2020). Lassa fever Infection transmission is primarily through pathway 1, particularly from multimammate rodents (*Mastomys spp.*) that carry and transmit the human-compatible LASV. Pathway 3 transmission, though still plausible, is less evident (Agbonlahor et al., 2021).

4.1.2. Virulence

This refers to the ability of a virus to successfully circumvent the host immune system to induce disease and eventual death. Virulence will vary greatly depending on independent pathogen adaptations and the response of infected hosts (Bonneaud and Longdon, 2020). Several studies suggest that viruses adapt towards virulence optimal for longevity. However, this theory is subject to the unique pathology of each virus (Pagán et al., 2014; Jaderyan and Khotanlou, 2016; Cressler et al., 2016; Bonneaud and Longdon, 2020). Greater virulence implies higher mortality levels, which affects the availability of hosts and limits proliferation (Mishra and Mishra, 2020).

Furthermore, higher virulence is likely to create a socio-economic bias, leading to proactive societal practices (Pagán et al., 2014; Bonneaud and Longdon, 2020). Alternatively, low virulence will strengthen host immunity and significantly lead to extinction (Visher et al., 2021). Optimal virulence requires enough stealth and adaptability to manipulate host immunity and enhance spread while limiting mortality. Several reports indicate that viral zoonoses in Africa have corresponded to these findings (Cressler et al., 2016; Grubaugh et al., 2020; Visher and Boots, 2020). Diseases with more optimal fatality rates and generic symptoms like Monkeypox, Rift Valley and West Nile fever seem to be positively impacted, and others with Ebola are negatively impacted (Bonneaud and Longdon, 2020; Wang et al., 2020).

Ebola is a highly virulent RNA virus, with outbreaks linked to pathogens of the genus Ebolavirus: the Sudan Ebola virus and the Zaire Ebola virus (Changula et al., 2013; White et al., 2019). The rates of mortality ranged between 49-70% in multiple outbreaks. This has been attributed to its expert evasion of adaptive immunity (Mollentze and Streicker, 2020). Thus, the disease has also experienced more rigorous human interventions and shorter timescales of circulation (Bonneaud and Longdon, 2020). Monkeypox has a fatality rate of ~10%, a fair value that leans more to <5% in current estimates. Each notable species varies in virulence- the Congo Basin clade supersedes the West African clade and all previously identified clades and is responsible for the US outbreak of 2003. Monkeypox manipulates the immune system by restricting T-cell production and T-cell-mediated action by up to 80% (Wheat, 2006; Kaler et al., 2022). This immunomodulation has been only discovered in the Congo variant (Kumar et al., 2022). The Central African variant has also been seen to regulate apoptosis and alleviate the transcription and expression of immune-related genes. Unfortunately, their larger size also limits immune system evasion, which is combated with virulence proteins: viro-mimic, viro-stealth and virotransducer proteins (Harapan et al., 2022). These actions produce a net negative effect on its virulence (Kumar et al., 2022). There is evidence that sufficient immunity against Lassa fever will be generated because of a single infection, and chronic infection is rarely established. About 80% of reported LASV infections are mild or asymptomatic cases, with non-specific conditions such as common fever and weakness. These individuals recover to total health (Purushotham et al., 2019). This amount may double if the likelihood of misdiagnosis in these areas is considered (Olowookere et al., 2014; Mrema, 2020). In patients already hospitalised, the mortality rate is 15-25%, which rises to about 35% for non-endemic countries

(Houlihan and Behrens, 2017; Buba et al., 2018). The LASV attacks the macrophages and dendritic cells of the innate immune system, and ser seroprevalence is associated with a LASV-specific memory T-cell (Langwig et al., 2015; Sullivan et al., 2020). HIV is particularly efficient in immune manipulation. Reports have suggested that initial outbreak success is due to its ability to transmit highly variant strains between interspecies and work in tandem with other pathogens to evolve (Sousa et al., 2017; Walker et al., 2018) favourably. West Nile disease typically begins and remains asymptomatic or produces mild febrile symptoms. It less frequently progresses to the more virulent later phases, when it attacks the central nervous system (Wilby, 2009; Ciota et al., 2013; Witkowski et al., 2014; Prow et al., 2016). Earlier symptoms of Rift Valley disease are non-specific, including fever and nausea. Later symptoms include severe hepatitis, abortion or malformations of the fetus, and neural disorders (Meegan and Bailey, 2019). Zika virus is symptomatic in 20% of infected cases and rarely leads to death. The disease is considerably associated with poor neurological development in infants (Schwartz, 2017).

4.1.3. Evolution

Viruses may initially possess traits that allow circulation in new populations, but more often, they need to evolve to improve propagation in a community. An interspecies transfer is, therefore, more probable the closer the organisms are genetically (WHO, 2003; Mullins et al., 2010). The rate of evolution and the likelihood of success depends mainly on the rate of mutation and the degree of selective pressure towards those mutations (Visher et al., 2021). The former is a function of the virus's genetic structure and the life cycle stage, while the latter is a function of community structure, gene pool and several infected individuals (Lion and Metz, 2018; Parsons et al., 2018; Guth et al., 2019). Pathogens often develop functional traits through low-impact mutations that precede an outbreak (Bull and Ebert, 2008; Visher et al., 2021). Within this trial stage, frequent failure is essential. Thus, RNA viruses often evolve faster (Bull and Ebert, 2008; WHO, 2009; Acevedo et al., 2019). Reports on Monkeypox, Ebola virus, and COVID-19 have implicated notable evolution as key drivers of African outbreaks.

Monkeypox is a double-stranded DNA. Unlike other DNA viruses that replicate in the nucleus, poxviruses like this can utilise cell proteins to carry cytoplasmic replication. The prevalence of monkeypox infection in individuals born during the discontinuation of smallpox vaccination depicts a corresponding decline in cross-protective immunity it provided (Myers et al., 2013; Kaler et al., 2022; Kumar et al., 2022). Comprehensive laboratory analysis of various Lassa fever cases following this time also indicates a marked heterogeneity in strains, even amongst individuals within the same zone (Langwig et al., 2015; Hallam et al., 2018). This implies rapid and multiple evolutions leading to its outbreak (Hallam et al., 2018). The genetic material of the Ebola virus is also a single RNA, thus undergoing rapid and frequent mutations (Shoemaker et al., 2012; Zinsstag et al., 2012). Some reports show strong genetic similarities between samples collected during the outbreak

that depict a singular source of infection (NCBI, 2021). The time scale of viral evolution occurs differently than the data may imply. Rates may spike within an outbreak and slows down between or before an outbreak (Gire et al., 2014). It is more likely that lack of sampling is the point of contention. Typically, EBOV RNA viruses evolve through rapid transcription, creating multiple error-prone models, which are often eradicated through natural selection (Lu et al., 2015). It is unclear what advantage the Ebola virus disease outbreak of 2013-2014 had, but it likely evolved to improve transmissibility or virulence or migrated into a peculiar, more suitable demographic. Alpha and beta coronaviruses emanated from mammals, particularly bats, while gamma and delta coronaviruses originated from birds and pigs (Normile, 2008; Ceballos et al., 2015). The genome of these viruses varies from 26 kb to 32 kb. Scientific evidence shows that the newly isolated South African variant, Omicron, has a colossal number of concerning transmutations (Ceballos et al., 2015; Reed, 2018; WHO, 2020; NCBI, 2021). Empirical evidence from polymerase chain reaction diagnostics suggests an increased risk of reinfection with this variant compared to other variants of concern (Saadat et al., 2020). Several studies are currently in progress to evaluate the rate of mutations in this variant. However, pieces of evidence gathered so far are indicative of a detrimental transformation in COVID-19 epidemiology (Novelli et al., 1998; Saadat et al., 2020). Consequently, the WHO has designated it a global concern (WHO, 2020).

4.2. Host scope: primary

4.2.1. Interspecies host interaction

The interface between various organisms, particularly wild ones, increases the risk of pathway 1 disease transmission. Of the endemic regions, the seroprevalence of viral zoonoses varies greatly, with West African forest areas the most prominent zones (NCBI, 2021). It is believed to be associated with the availability and proximity to wildlife habitat, particularly rodents, i.e. the hypothesised reservoir host (Ganjeer et al., 2021). The LASV rats are asymptomatic and highly fertile. Therefore, they are numerous in endemic regions and are unlikely to be identified as diseased (Bonwitt et al., 2016; Wu et al., 2017; Reed, 2018). This may facilitate consumption and, thus, the spread of infection. Transmission routes may be through the skin, gastrointestinal tract and respiratory tract. In a study of 36 primary and secondary cases of LASV, 36% of infection was linked to nosocomial and lack of PPE by healthcare professionals and morticians, and 16% was traced to contact with peridomestic rodents (Wolf et al., 2020). Another study indicated that areas with infected individuals were multiple times more likely to have a rat infestation than areas without (Tambo et al., 2018; WHO, 2018).

Four (4) formational genes encode the spike protein, a small membrane protein, the nucleocapsid protein, and the integument glycoprotein with an additional membrane glycoprotein in the HCoV-OC43 and HKU1 betacoronaviruses (Saadat et al., 2020). The unexpurgated genome of SARS-CoV-2 is 96% homogenous to the coronavirus in bats (Saadat et al., 2020; Obafemi et al., 2021). This gives empirical justification that the covid-19 pandemic might have originated from bats, although some scientists suspect pangolin may be the source. For example, new buildings constantly dislodge bats from their natural habitats in the wild into the city. Other dislodged wild animals have been frequently spotted on the streets (Li et al., 2020). Their droppings and fluids in human societies may be potential sources of new viral infections (Reed, 2018; CDC, 2020; WHO, 2020).

4.2.2. Type of host

The role of host response is a significant component of a viral outbreak. Though the exact reactions are not yet accurately determined, it is well known that factors like health, socio-economic status and type of treatment are incredibly influential. Individuals with poor diets and sanitary culture or immunocompromised are more at risk and experience enhanced fatality (Grace et al., 2012; Moretti et al., 2013; Matilla et al., 2018; Launay et al., 2021). Healthcare workers are at the frontline of patient care and are more vulnerable (Vonesch et al., 2019). Similarly, individuals who have been vaccinated may experience cross-immunity towards related viruses (León-Figueroa et al., 2022). Precise mapping of vulnerable demographics may be determined case by case.

Monkeypox has notably affected individuals born after monkeypox vaccination discontinuation (Kaler et al., 2022). HIV transmission heavily relies on the viral load, which is influenced by the type of variant, the stage of its lifecycle and the presence of other pathogens, which help to boost the load (Nguyen et al., 2019). Infections like genital ulcer disease (GUD) and Syphilis, which cause inflammation and amplify HIV load, likely contributed to the initial outbreak (Maan et al., 2021). War-ranging areas, work associated with frequent sexual practice, paternal transmission and men without circumcision have been deemed more vulnerable to the spread of the HIV-2 Disease in Western Africa (Odilara et al., 2006). Inadequate use of PPE, lack of proper safety protocol and sexual promiscuity are also substantial risk factors associated with HIV (Vu et al., 2018). Alternatively, the presence of CCR5 receptors and vaginal lactobacilli diminishes HIV susceptibility (Wada, 2018; Ñahui Palomino et al., 2019).

4.3. Host scope: secondary and tertiary

According to Conticini et al. (2020), increased pollution could boost the propagation of zoonotic viruses. Thus, countries with high air pollution levels have been more significantly impacted by outbreaks than the less polluted ones. It has been severally documented that altering aquatic habitats through organic pollutants enables the pathogens of water-borne diseases such as diarrhoea, cholera, dysentery, and so forth, contributing significantly to a sharp rise in infant mortality (UN, 2019; Ong et al., 2020; Zhang et al., 2022). These challenges have marked alterations in ecosystems, thus shaping the behavioural evolution of wildlife, pathogens and viruses (Markovchick-Nicholls et al., 2008). Consequently, these impacts have influenced the emergence and dispersal of infectious diseases (Oladele et al., 2018), threatening public health (Olival and Hayman, 2014). Viral zoonosis initially penetrates the population from rural settlements. They suggest that primary local hotspots are produced when milder deforestation rates combine with proximity to a fragmented society. Zhang et al. (2022) fault the predominant poverty and corruption. The bioresource bases of forest ecosystems and inland and coastal mangroves are vital for the rural poor in developing nations. As agrarian societies, African countries depend primarily on biodiversity resources for nutrition and medicine (Langwig et al., 2015; Reed, 2018). Biodiversity, therefore, supports 70-80% of the food requirements of 70% of locales, while about 30-50% of urban and peri-urban areas depend on natural resources for their nutritional support (Parola and Raoult, 2001; Spencer et al., 2020). Impoverished African societies, therefore, rely on illegal activities such as poaching, logging for fuelwood, and abuse of land, air and water bodies for survival (Root et al., 2003; Dilley et al., 2005; Parrish et al., 2008; Schwarzenbach et al., 2010). Generally, poverty plays a multifunctional role in the epidemiology of infectious diseases. Poverty causes malnutrition which is responsible for immunosuppression, hence the susceptibility of the impoverished population (Kelt and Hafner, 2010). Poverty thus increases the direct interactions of humans with the habitats, thereby compromising the endemic barrier. Some of the economic-motivated interactions of humans with nature include hunting, domestication, transportation, slaughter, and sale of wild animals. This may enable the viruses in domesticated animals, fluids, or wastes to break the ecological barrier and adapt to human societies (CSIS, 2020; NCBI, 2021; Zhang et al., 2022).

5. Approaches to Combating Viral Zoonoses

The planet is in a state of dynamic equilibrium as the inherent life support systems continue to compensate for the predominant anthropogenic perturbations to ensure the continuity of essential ecological services (Powers, 2015). Viruses continue to evade prevention and control measures by undergoing mutations that evolve new variants with more significant morbidity. As the world anticipates a new pandemic by the newly evolved virus called Omicron, it becomes imperative to explore scientific measures to mitigate the outbreak and possible ones in the future (Richmond and Baglole, 2003; Real and Biek, 2007; Reperant, 2010).

5.1. The ecological barrier restoration approach

Drastic urbanisation encroaches on natural vegetation, promotes human population clusters, and closes the gap between humans and animals. These, in turn, increase the likelihood of transmission through breeding, slaughter, transportation, and sale of animals (Pearce-Duvet, 2006; UNEP, 2007; Weaver and Lecuit, 2015; Steiner, 2020). The prevalence of emerging viral variants crossing the ecological barrier is strongly linked to ecological processes that are intensively affected by the consequences of anthropogenic activities, characterised by global climate change, invasions of wildlife habitats, unsustainable agricultural practices, and dramatic urbanisation (Daszak et al., 2013; Gao et al., 2013; Pike et al., 2014; Zumla and Hui, 2019; NCBI, 2021). In this context, the focus is to restore the potentiality of the ecological barriers to impede viral transmission from innate hosts to humans (NCBI, 2021). The burdens of viral mediation in the wild, intermediate, and domesticated assemblages, as well as the environmental media, are also to be put into perspective (Parsons et al., 2018; Zhu et al., 2020; NCBI, 2021; Zhang et al., 2022).

A comparative assessment of the corona variants has characterised Omicron as a coronavirus of concern with enhanced transmissibility, acridness and inimical change in COVID-19 epizootic, which may present a dramatic transformation in clinical disease presentation, thus, causing global health concerns of greater magnitude than ever seen (UN DECADE, 2019; CDC, 2020; UNEP, 2020). This may impair the efficacy of diagnostics, therapeutics, and vaccines; and the effectiveness of social measures. Based on current zoonotic trends, this may soon become common. Within the scope of this report, we consider the viral outbreaks as a reaction of nature (CDC COVID-19 Response Team, 2020; UNESCO, 2020). We presuppose that the features of HIV, Lassa fever, Ebola and COVID-19 relate to ecological indices. Applied ecology could help solve critical problems that the global invasion of SARS-CoV-2 has unravelled (CDC COVID-19 Response Team, 2020; Li et al., 2020). Conscious efforts towards restoring the endemic and cross-species barriers may hold the hope for the safe and sustainable coexistence of all species on earth. Ground-breaking success in applied ecology and milestones in the strategic manipulation of the endemic barriers, particularly cross-species barriers, may proffer novel solutions and conquest against the coronaviruses of concern, including the newly evolved Omicron (Langwig et al., 2015). Therefore, it is imperative to survey further the empirical upshots of anthropogenic activities on the integrity of environmental barriers and fill the knowledge gap on the transmission mechanisms of emerging coronaviruses across the barriers (NCBI, 2021).

5.2. The viral surveillance approach

Peculiarities in viral behaviours are studied for disease profiling and control (Petersen et al., 2013; Wille et al., 2021). Several factors constrain these reports, resulting in an exclusive representation of viral subpopulations of socio-economic relevance (Wille et al., 2021). The issue is compounded when we consider that even predominantly employed surveillance techniques poorly capture the viral richness of this subset of the virosphere (Childs et al., 2007; CBD, 2017; Wille et al., 2021). Our understanding of proximate causes may be insufficient, so our findings on some distinct zoonotic outbreak episodes chronicle commonalities that characterise successful outbreaks (Ceballos et al., 2015). We recommend enhancing surveillance and a detailed chronological investigation to understand the dynamics of viral mutants better. For this approach, the public availability of a database on complete genome sequences and the associated metadata variances of interest and concern is essential (Langwig et al., 2015).

5.3. Further recommendations

The paucity of information on the epidemiology of the variants of concern characterises the limitations of this report. Hence, we recommend further field investigations, improved diagnostic methods, and laboratory assessments to revamp mastery of the potential impacts of Omicron on COVID-19 epidemiology, morbidity, immune responses, antibody neutralisation, or other vital characteristics. We recommend rigorous studies to rejuvenate all crossspecies barriers discussed in this report. Restoration of the endemic barrier is recommended to forestall future epidemic outbreaks through more rigorous applied ecological research paths. A detailed, comprehensive analysis of the interconnections amongst viruses, their hosts, and environmental media can ignite superior cognisance into the effects of ecological barrier atrophy on the spread of coronaviruses, as well as the influential prime factors. Policies and strategies are required to encourage reforestation and ecological restoration to re-establish lost forests, vegetation, and other ecosystems. Mitigation of unregulated wildlife trade and sanctions for dairy and wet handling is required worldwide. Awareness creation, public sensitisation, and protection of natural habitats are also necessary measures to prevent future disease outbreaks.

5.4. Conclusion

The ecological barrier is an essential natural component of the ecosystem which protects human society from viral transmissions from natural or intermediate hosts. The protective integrity of the ecological barrier determines the emergence of infectious viruses in human society. Future studies must fill the knowledge gap on dynamic processes and ecophysiological conditions in crossing over viruses beyond the ecological barrier. This knowledge is critical to preventing and controlling emerging epidemics. The dominant influential constituents affecting the ecological barrier comprise virus-specificity, contact probability and frequency, and transmission routes. Ultimately, prevention of future viral outbreaks through ecological restoration, integrated with other sustainable mitigation measures such as wearing well-fitting masks, hand hygiene, social distancing, enhanced indoor ventilation, prohibiting crowds, and vaccination.

We propose two practical steps to alleviate, control and perhaps, prevent future outbreaks within the continent. Our contribution is valuable to prior attempts to redirect zoonotic disease control efforts from reactive to preemptive through a multidisciplinary "one health" approach (Williams et al., 2021).

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