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## The environmental roots of zoonotic diseases: from SARS-CoV-2 to cancer viruses. A review.

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**Article history:** Submitted June 17, 2020. Accepted in revised form December 1, 2020.

**Published online:** December 9, 2020

**Citation:** Modonesi, C. (2020). The environmental roots of zoonotic diseases: from SARS-CoV-2 to cancer viruses. A review. *Visions for Sustainability* 14, 54-65.

**DOI:** <http://dx.doi.org/10.13135/2384-8677/5319>

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**Competing Interests:** The author has declared that no competing interests exist.

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### Abstract

The destruction of natural habitats and change in land use contribute to biodiversity loss by increasing species extinction and weakening the functions of ecosystems. Ecosystems often are unsafe for humans because animals that host viruses or other pathogens become dominant within impoverished biological communities. The risk of infection propagation from one animal species to other species depends on the size of the reservoir population and the “ability” of pathogen to spillover: an event that is more likely to occur in phylogenetically related hosts. Zoonotic spillover is the transmission of pathogens to humans from vertebrate animals. If human activities contributing to the alteration of ecosystems do not slow down, the critical state of biodiversity can turn into an important driver of emerging pathogens, including viruses involved in neoplastic diseases. A radical reform of the current growth-based economic model is urgently needed to counter the unsustainable human pressure on the natural environment and the risk of new pandemics.

**Key words:** biodiversity loss; biological communities; cancer viruses; dilution effect; eco-epidemiology; economic growth; ecosystem functions; environmental health; nature/nurture debate; viral epidemics and pandemics; zoonoses.

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### Introduction

Infectious and parasitic diseases contribute to over 20% of the global disease burden, while in some areas of the planet the figure reaches over 70% (Patz et al, 2005; Engels and Savioli, 2006). The number of epidemic emergencies resulting in human and economic losses has grown considerably over the past century and Emerging Infectious Diseases (EIDs) have been widely monitored over the past two decades (Patz et al, 2005; Jones et al, 2008). The environmental disturbance due to human activities – industrial agriculture (including animal farming), soil erosion, greenhouse gas emissions, deforestation, urbanization and increased global mobility of people, goods, plants and animals – plays a crucial role in the way these diseases develop and spread worldwide (Daszak et al, 2001). Greater attention to the interaction between global change

and global health today reveals a high frequency in animal-borne diseases, in particular viral infections, while biodiversity loss is considered as a major challenge both globally and locally (Patz et al, 2005).

The mounting number of diseases has been described as a side effect of civilization, with its anthropization and transformation of the natural environment, and zoonoses are no exception (Dobson and Carper, 1996). Recent unprecedented rates of anthropogenic land use change, including agricultural conversion or intensification and habitat fragmentation, have led to a progressive ecological erosion of natural environments essential for the survival of human beings (White and Razgour, 2020).

Land use change can be an important source of zoonotic diseases due to its impact on human-wildlife interplay. By removing or reducing the natural habitats of many animal species, over-exploitation of the land leads them to live closer to human settlements (Jones et al, 2013). The problem is further complicated when an area is inhabited by one or more species that host one or more zoonotic pathogens (Jones et al, 2013). The transmission of pathogens tends to increase in response to anthropogenic impact, although for the moment this effect cannot as yet be considered universal (Gottdenker et al, 2014).

The recent history of viral epidemics related to human impact on ecosystems and wildlife is full of interesting and worrying cases, some of which are paradigmatic (Sharp et al, 2001). In the last three decades of the past century, the pandemic strain of HIV-1 was found to be closely related to a virus identified in several chimpanzee populations of the *Pan troglodytes* subspecies living in the forests of Central Africa (Cameroon, Equatorial Guinea, Gabon, Congo-Brazzaville and Central African Republic). The ape-human spillover occurred in rather critical conditions, characterized by a strong human presence in those habitats (Hahn et al, 2000). According to the so-called “hunter theory” (or “bushmeat theory”), the “jump” between species is based on the hypothesis that the virus was transmitted by chimpanzees to humans through hunting or slaughtering or consuming bushmeat (meat from wild animals). Subsequently, the virus spread to all continents through unprotected sexual habits and other dangerous human behaviours, including the consumption of injectable substances such as heroin and other drugs which have proved to be particularly devastating (Hahn et al, 2000).

In recent decades, emergencies caused by arenaviruses responsible for the spread of haemorrhagic fevers in Argentina and Bolivia have been linked to ecologically aggressive agricultural practices and the fragmentation of natural areas in order to build infrastructures and carriage roads (Mills, 2006).

In Southeast Asia, the Nipah virus, a pathogen first identified in the late 1990s that causes severe encephalitis and acute respiratory syndrome, has spread from wildlife to humans due to the expansion of industrial pig farming in a biodiversity hotspot full of frugivorous and nectarivorous bats. In that region, bats are the main natural reservoir of the virus and carrier individuals can release the pathogen through saliva, urine and feces without themselves developing any disease (Mazzola and Kelly-Cirino, 2019).

As many ecologists and epidemiologists know, the global picture of zoonotic diseases is much broader and more varied than the short repertoire described above, which only serves to understand the geographical and ecological dimension of the problem. When a forest habitat is cleaned and replaced by human settlements or domesticated environments, such as industrial crops and farms, the previous biological community is literally emptied and filled by a new environment mostly for economic purposes. In these unnatural contexts, persistent mammal species are often host to zoonotic viruses and their dominance over agricultural and peri-domestic areas increases the human risk (Hussein et al, 2016). As human activities that contribute to unsafe ecosystems continue to accelerate, interest in the role of diversity and community composition in changing disease risk will increase (Patz et al, 2004; Johnson et al, 2015). Based on such evidence, territorial surveillance actions should be implemented to promptly detect the infectious risk through appropriate environmental indicators.

However, when zoonotic outbreaks such as those discussed above occur, our attention should not be limited to their ecological and climatic triggers. It should primarily be focused on the multiple critical factors produced by the economic and cultural context. These factors could make infectious outbreaks more frequent in the future (Weiss and Cattaneo, 2017). For example,

further expansion and intensification of land use for agro-industrial purposes on a local and global scale could determine the conditions for the appearance or reappearance of new and old diseases (Lewontin and Levins, 2008).

Looking at current events, the SARS-CoV-2 pandemic requires attention to the critical connections between environmental deterioration and the emergence of pathogens, as well as the role played by intensive agriculture and land use changes in fostering the infectious outbreaks. Further scientific efforts are needed to obtain a more complete understanding of the phenomena underlying the observed health outcomes and to implement the transition toward an agroecological model of food production (Altieri and Nicholls, 2020). There is no doubt that the food demand of humanity needs an alternative agricultural paradigm, one that encourages more ecological, biodiverse, resilient, sustainable, safe and socially just forms of agriculture (Altieri and Nicholls, 2020). From a methodological point of view, the need for a systemic approach based on the integration of ecological, social and public health data clearly emerges.

### **Zoonoses: what are they and where do they come from?**

The term “zoonosis” refers to all diseases transmitted between humans and animals. Zoonoses represent a large part of recurrent and emerging infectious diseases and are now regarded as one of the major threats to health systems globally (WHO, 2014). Out of the 175 EIDs described at the turn of the millennium, 75% were animal-borne diseases (Mills, 2006). Today, about 200 zoonotic diseases are hosted by a wide variety of vertebrate species, including fishes, amphibians, reptiles, birds and mammals (Mills, 2006). Pathogens shared by wild and domestic animals cause more than 60% of infectious diseases in humans (Taylor et al, 2001). Such diseases include leptospirosis, cysticercosis and echinococcosis, toxoplasmosis, anthrax, brucellosis, anger, Q fever, Chagas disease, type A flu, Rift Valley fever, severe acute respiratory syndrome (SARS), Ebola haemorrhagic fever and HIV (Karesh et al, 2012). The most significant impact on global health is represented by about one billion cases of disease and millions of deaths that occur every year due to endemic zoonoses (ILRI, 2012). These infections are often enzootic (i.e., they remain limited to some animal populations) but sometimes they pass from animals to humans (ILRI, 2012). While animal species that share an evolutionary and/or ecological affinity with humans can transmit various viral or other zoonoses, here we focus mainly on viral ones.

Pandemics that have taken their first steps in animal populations living in perturbed habitats are common, but the underlying processes are not so clear. Recent studies show that animal species that have increased in abundance and/or have expanded their range in anthropized environments are more likely to transmit zoonotic pathogens (Pandit et al, 2018). A chain of viral infection is often the result of viral molecular changes induced by the complex interactions occurring between wildlife, domestic fauna and our species. The conditions that precede the spillover often depend on over-exploitation of the soil and the increasing pressure of economic activities on natural systems (Johnson et al, 2020). Activities such as monoculture, intensive animal farming, industrial fishing, wildlife hunting and illegal trade of protected species are typical factors that destroy natural environments by promoting conditions of ecological instability and infectious outbreaks (Johnson et al, 2020).

Many of these activities are accused of triggering a significant decline in wild populations by exacerbating the risk of extinction in already endangered species. It is worth pointing out that the epidemiological features of viral transmission at the animal-human interface have sometimes revealed dynamics that in the past have led to zoonotic spillover events (Johnson et al, 2015). This suggests that a historical perspective on how our species has managed its relationships with the animal world could be useful in order to identify and map the main factors of zoonotic risk.

### **Spillover: bats but not only bats**

While some ecological conditions make the cross-species transmission of animal viruses more likely, researchers can rarely observe animal-human spillover events leading to emerging diseases, and therefore the detailed dynamics related to these phenomena have yet to be clarified. Bats (Order *Chiroptera*) belonging to the Chinese horseshoe bat species (*Rhinolophus sinicus*) are reservoir animals of a large number of zoonotic viruses, including coronaviruses (CoV) that cause

infectious outbreaks in human populations and farm animals, such as the Severe Acute Respiratory Syndrome (SARS) (Wang and Anderson, 2019). SARS-CoV is the pneumonia virus that spread to 32 countries in 2002-2003, infecting around 8,100 people and causing 774 deaths (WHO, 2019a). Another lung disease caused by a bat-borne coronavirus (MERS-CoV) is the Middle Eastern Respiratory Syndrome (MERS), which in the first few months of 2019 killed 823 people and caused 2,374 disease cases in 27 countries (WHO, 2019b). The lesser-known coronavirus (SADS-CoV) which caused severe acute diarrhoea syndrome (SADS) in most pig farms in southern China in 2017-2018, killing over 20,000 piglets, is a further pathogen of bat origin (Zhou et al, 2018).

For many viral zoonoses, spillover is the seemingly random result of a series of events. Usually, it requires the concurrence of the following conditions: (a) a reservoir species must be present in the biological community and must be infected with the virus; (b) the virus must survive outside the reservoir species and have access to a receiving host species; (c) the receiving species must be exposed to a sufficient amount of viral source (viral load) and must also be susceptible (host competency) (Plowright et al, 2015).

Often the receiving species is an intermediate animal that lives in contact with humans, which in turn can become infected. For example, although a large variety of coronaviruses, including SARS-related coronaviruses (SARSr-CoVs), were first discovered in bats, in 2002-2003 humans were infected with SARS-CoV by civet cats (*Paguma larvata*). The same probably occurred with the MERS epidemic, spreading from bats to humans through camels (*Camelus dromedarius*) which are now the main reservoir species of that virus in the Middle East (Wang and Eaton, 2007; Wang et al, 2011). Similarly, the most recent coronavirus (SARS-Cov2) involved in the COVID-19 pandemic first appeared in bats but is suspected to have infected humans by passing through the Malayan pangolin (*Manis javanica*) (Cui et al, 2007).

It is assumed that in some cases viruses can be amplified by the intermediate host species (Drexler et al, 2012). However, the ecological events that determine the interactions between the natural reservoir and intermediate species are poorly understood, probably because the predisposing conditions and the cross-species contagion occur at different temporal, spatial and ecological scales (from within-host pathogen evolution to spatially extensive processes such as land use and climate change) (Plowright et al, 2015). Compared to other taxa of eutherian mammals, such as rodents, bats could be perceived by the non-expert reader as unusual carriers of infectious diseases; conversely, they may have played that role for a long time (Calisher et al, 2006; Luis et al, 2013). According to some investigations, many viral pathogens, including viral ancestors of measles, mumps, parainfluenza, canine distemper and hepatitis C virus, may actually have originated in bats (Drexler et al, 2012). An interesting hypothesis suggests that their immune system differs substantially from that of most mammals as an effect of flight adaptation (Zhang et al, 2013). The ability to fly, therefore, could be the key element to a better understanding of the coevolution of bats and viruses: a milestone that would have transformed bats into a natural reservoir capable of tolerating and transmitting to other animals many viral pathogens (O'Shea et al, 2014).

### **Ecology of viral zoonoses**

In general, natural habitats with a high level of biodiversity could be expected to be a favourable substrate for the development of a greater number of pathogens potentially transmissible to humans. Based on this assumption, it has also been argued that biodiversity loss could make a substantial contribution to reducing the frequency of zoonotic diseases (Wolfe et al 2005). However, a series of studies refutes that hypothesis by showing a different perspective. Disturbed environments can be unsafe for humans when animal hosts (reservoir species) become dominant within altered biological communities, thus increasing the prevalence of zoonotic pathogens (Patz et al, 2004). In these cases, the structure of the biological community is significantly different from the original and ecosystem functions are weakened (Karesh et al, 2012). The new community composition makes it possible to favour zoonotic viruses shared by *Homo sapiens* and other vertebrate hosts, which include most human EIDs agents (Taylor et al, 2001).

For example, rodent-borne haemorrhagic fever outbreaks have shown that when habitats have undergone a significant reduction in biological diversity (e.g., due to human activities), there is a greater risk of contracting viral infection compared to habitats that maintain a good level of

biodiversity (Johnson and Thielges, 2010). Interesting hypotheses have been developed to explain these dynamics, such as the so-called “dilution effect”. According to this model, there is an inverse correlation between biodiversity and disease risk. This is observed whenever a reduction in biodiversity occurs leading to an increase in the abundance of focal species potentially capable of favouring viral transmission to humans (Johnson and Thielges, 2010). Evidence from many cases of host-virus interaction highlights that the “decomplexification” of biological communities can easily turn into a threat to our species. On the other hand, in natural systems characterized by greater diversity, the susceptibility of animal species to infections (host competency) is much more variable, leading to a reduction of infection prevalence and a significantly lower risk of disease spreading.

The dilution effect is supported by well-tested data over the past 20 years (Ostfeld and Keesing, 2000). The model was formalized to explore the key points of the relationship between biological communities and human diseases. The underlying concept dates back to about a century ago and derives from the ancient practices of crop rotation and zooprophylaxis (the use of farm animals to protect humans from pathogens), typical of many rural communities around the world (Elton, 1958). In those rural societies the livestock is strategically placed around human residences to keep malaria-carrying mosquitos away from people (WHO, 1982). In other contexts, similar roles have been played by rabbits in reducing sand fly-borne leishmaniasis, cats and dogs in reducing mosquito-borne encephalitis, and lizards in reducing tick-borne Lyme disease (Hess and Hayes, 1970). It is worth noting that the dilution effect framework in zoonotic systems was developed for the tick-borne Lyme disease, an infection caused by the spirochete *Borrelia burgdorferi* (LoGiudice et al, 2003). Although the pioneering study was based on a bacterial disease, the model is equally applicable to viral zoonoses. In fact, very similar results were obtained by exploring the negative correlations between diversity and viral infections in birds, rodents, sheep, and other vertebrates (Keesing et al, 2006).

The species structure of a biological community reflects a pattern in which the reservoir animals tend to be generalists in their ecological habits, and furthermore they have a short lifespan (Karesh et al, 2012; Johnson et al, 2020). These species adapt well to disturbed environmental conditions and usually develop large populations in a rather short time. In general, larger populations are more likely to spread cycles of infection (Karesh et al, 2012). Conversely, animal species with more specific needs in the use of natural resources and small population sizes, such as many predators or species with a longer lifespan and slower reproductive cycles, tend to disappear from altered ecological situations.

Finally, it may be interesting to note that in a broader geographical context threatened species share relatively fewer viruses with humans, supporting the principle that the risk of viral spillover is influenced by the frequency of human-animal interactions (White and Razgour, 2020).

### **The blurred border between chronic and infectious diseases: viruses and cancer**

Diseases that were once believed to be non-communicable have been recognized to have infectious cofactors. Conversely, degenerative diseases and their treatments can alter individuals' immune systems leading to associated infections that put the patient at risk and make the clinical work more complicated (Modonesi et al, 2017). According to the World Health Organization (2011), many cancers are linked to chronic infections with pathogens, especially viruses. Some authors recognize in this aspect an individual susceptibility to cancer induced by infection and inflammation rather than a direct and specific relationship between viruses and carcinogenesis (Voisset et al, 2008). After all, the coexistence of animals and *Homo sapiens* goes back to the mists of time and the sharing of viruses and other microorganisms could be seen, in a sense, as an inevitable ecological implication of life on Earth, regardless of its negative effects on human health, including malignancies.

Several zoonotic viruses, basically DNA viruses and retroviruses, are involved in some way in the malignant transformation of biological tissues causing 15 to 20% of all human cancers worldwide (Parkin, 2006). The prevalence of these viruses varies in different parts of the world. Almost 30% of cancers in developing countries are linked to infectious agents, while that percentage drops to 10% in developed countries (Parkin, 2006). For example, Papillomavirus has been related to cervical cancer, Epstein-Barr virus to Burkitt lymphoma, hepatitis B and C viruses

to liver cancer, HTLV to leukemia in humans and KSHV to sarcoma of Kaposi. Other parasites linked to human tumors are bacteria like *Helicobacter pylori* (stomach cancer) and small invertebrates such as *Schistosoma hematobium* (bladder cancer) (Parsonnet, 1999).

An infectious etiology for cancer was first documented in animals during the early part of the 19th century, with the diagnosis of pulmonary adenocarcinoma in sheep caused by the Jaagsiekte sheep retrovirus (JSRV) (Tustin, 1969). With the development of biological research, it was discovered that many animal species such as rodents and other *taxa* could be hosts of viruses suspected of promoting carcinogenesis. For example, the reticuloendotheliosis virus induces cancer in chickens (avian leucosis-sarcoma). A wide variety of viruses mirroring their human analogues are spread among animals and common types include viruses of the *Polyoma*-, *Adeno*-, *Retro*-, and *Papilloma*- virus families (Hundesda et al, 2006).

Modern research into the carcinogenic potential of viruses has helped broaden conventional perspectives on the mechanisms of cancer. For example, interesting results indicate that adenoviruses, HPV (Human Papilloma Virus) and HTLV-1 (human T-lymphotropic virus) commonly block the cellular function required to establish the correct cell polarity, a property lost in almost all epithelial-derived tumor cells (Javier, 2008). These findings suggest that the loss of cell polarity directly contributes to malignant tissue transformation, showing that the investigation of viruses can clarify relevant dynamics of many human cancers (Javier, 2008). Another example comes from the “hit and run” hypothesis, according to which some viruses promote cancer by interfering with the immune system of hosts, but do not integrate into their DNA, thus contradicting the common assumption that tumor development is always the effect of a genetic change (Nevels et al, 2001). Future studies on the role of viruses in the carcinogenic process will have to address the complex nature of cancer by taking into account the interaction of biotic and abiotic factors, the multiple causation of the neoplastic transformation of cells and the related stochastic risk largely neglected by conventional mechanistic research. Currently, the ecological and biological connections between viral zoonoses and cancer, as well as the related role of animals, remain largely unclear and would require more scientific studies (Weiss, 2007).

While the general population is commonly exposed to animal viruses, many of which are known to promote cancer development in animals, a direct and mechanistic role for them in human carcinogenesis remains substantially speculative. The same infectious agent may react in different ways depending on host factors, including health status, environment, physiology, geography, seasonal variation, climate, population density, and so forth. That said, the etiological action of most viral agents in the neoplastic process deserves major attention and suggests that they commonly act within networks of multiple factors. Gene-environment interplay and epigenetic phenomena also are important pieces of a puzzle frequently missing in epidemiological studies of complex diseases such as cancer (Weiss, 2007).

Animal viruses believed to have oncogenic properties generally tend to be species-specific and do not replicate easily in human cells. However, as we have seen above, it is widely recognized that zoonotic viruses can infect different animal species, particularly when they share a common evolutionary background, contributing to the development of animal diseases. This aspect should not be overlooked, since the exchange of pathogens between domestic and wild animals can generate severe problems for humans and ecosystem health (Efird et al, 2014).

It should be remembered that infections caused by zoonotic viruses put the most fragile part of the general population at risk: primarily, elderly, young, pregnant, and immuno-compromised people. Individuals belonging to these high-risk categories should absolutely avoid any kind of exposure to pathogens, especially cancer patients who take drugs that suppress immune system. According to a recent investigation, 20% of patients who died from COVID-19 in Italy in the first half of 2020 were cancer patients (Burki, 2020). These people included subjects undergoing active chemotherapy or radical radiation therapy for lung cancer and patients with blood or bone marrow cancers.

Although biological evolution has provided adaptive immunity against many external adversities, human self-defense capacity against infections and cancer is often compromised and sometimes circumvented by the environmental pitfalls generated by our own species.

## Discussion

Unfortunately, most of the problems discussed in this paper are generally addressed in specialized scientific forums where epidemiologists, ecologists, tropical medicine experts and veterinarians present their data and opinions separately. A transdisciplinary approach is rarely implemented, and the result is that many interconnected dynamics are treated as if they belonged to different realities. The consequence of this is a fragmented and short-sighted science that neglects the fact that the two crises (biodiversity loss and disease growth) should be explored and addressed in parallel (Levins and Lopez, 1999). Nowadays, a timid consensus is slowly beginning to manifest itself within the scientific community, leading to view health and ecological calamities as the rebound effect of a broader anthropological crisis affecting most of the world. Based on present and future trends, a more effective approach for preventing zoonotic diseases will require a more extensive view of human and natural sciences, emphasizing the urgency of an integrated knowledge of the ecological, evolutionary and social phenomena occurring at the intersection between animals, humans and the environment.

Many infectious diseases have an old record of cosmopolitan appearance, disappearance and reappearance. The challenges due to economic globalization processes are connected with the scale and the speed with which people, products and pathogens can move across the planet (Institute of Medicine, 2006). The number of potentially infectious contacts has exploded as trade and transport bring goods, organisms and human beings closer than ever before. Nowadays, the duration of the longest intercontinental flight is shorter than the incubation period of a multitude of known pathogens (Institute of Medicine, 2006).

While some human health outcomes due to biodiversity loss may be directly and easily observable, others may not be so directly recognized (Mills, 2006). As reported by Patil and colleagues, according to the World Health Organization, the negative health effects of biodiversity erosion outweigh those caused by climate change (Patil et al, 2017). Even though the Convention on Biodiversity was approved and signed by nearly 200 countries in 1992, after 20 years we are witnessing the failure of the sustainability policies developed by international institutions, starting with agricultural policies for the conservation of land and biodiversity (Pe'er et al, 2014).

Healthcare professionals and public health researchers should support biodiversity conservation for its key role in promoting primary prevention and keeping human communities healthy. Engaging ecologists, epidemiologists and policymakers in a global campaign endorsed by governments and international organizations to support ecosystem health and environmental justice could be an important action for its pragmatic and ethical value. The two priorities of this initiative should be the following: i) stop plundering environmental resources; and ii) minimize the trade-offs between economic development and physical, chemical and biological deterioration of the ecosphere. In addition, it would be equally urgent to eliminate the differential exposure of population subgroups to pathogens and environmental risks, planning effective tools for the fight against poverty and access to health services.

Recent studies highlight the fundamental role of the environmental and social context as a determinant of people's health. A major aspect of many contextual variables is that they cannot be measured individually, because they are essentially properties of groups (ecological variables) (Diez-Roux, 1988). A metaphorical example can perhaps explain this principle better. If we try to study the determinants of automobile congestion that poisons our cities, investigating the characteristics of individual drivers is useless and misleading. The phenomenon can be more effectively understood by exploring the opening/closing cycles of businesses, offices and shopping centers, the location of schools, the organization of public mobility, the structure of urban spaces and other contextual variables (Giuliani and Modonesi, 2011). Likewise, the analysis of ecological variables and community factors can clarify better than an individual approach how context affects public health (Diez-Roux, 1988).

A more accurate understanding of the interaction between individuals and their environment must take into account our knowledge of the interactions between different levels of social organization and the connections between different systems (Sandberg et al, 1996). Since the relationship between our species and the environment depends on the basic rules established by the socio-economic framework, an objective and adequate evaluation of these rules is necessary when considering their effects on environment and public health. Many communicable and non-

communicable diseases are sensitive to ecological and socio-economic factors, which shows the extent to which such outcomes are avoidable or preventable. Furthermore, the most sensitive effects of these factors also vary between social groups, reflecting the wide distribution of responses to stressors to which the human population is exposed (Karpati et al, 2002).

Unfortunately, contemporary neoliberal capitalism seems to have little interest in these issues. In the richest part of the world, the fundamental principle of human organization is based on the idea that a limited planet can sustain an unlimited economic growth. Such a paradigm pursues, rather than combats, an irrational and unscientific use of ecosystems, effectively legitimizing the over-exploitation of the natural resources that sustain life on Earth, including human life.

The approach to the recent COVID-19 pandemic, as well as to the environmental health crisis of recent decades, is exactly a part of this context and, as such, it is affected by all its consequences. Its rules are based on the dogmatic supposition that the ecology of the planet is a small component of the human economy, rather than vice versa. Within this perspective, the socio-ecological roots of the good or bad health of the human population are institutionally removed. A stereotype supporting this view is that the natural world can be used by humans for exclusively economic purposes. Weak sustainability advocates who operate within this horizon are convinced that *the global economy can grow indefinitely* thanks to the power of technological innovation in overcoming natural constraints. This belief reinforces the idea that human happiness can be achieved through individual initiative and private appropriation/accumulation, regardless of any culture of solidarity, conviviality and the common good.

The ecology of the human species is becoming an increasingly critical force that destructively interacts with social and natural dynamics on a local, regional and planetary scale. To date, the leading indicator of “human temperature” worldwide is based on GDP, which overlooks the staggering costs of many human activities. It is worth noting that conventionally GDP takes no account of the role of ecosystem services and puts within the positive values column the expenses aimed to remedy depleted and degraded natural resources. Consequently, a country could clear its forests, deplete its fisheries and pollute its aquifers, thereby causing heavy damage in its ecosystems and human population, still claiming to pursue the well-being of its citizens: all while invoking that its interventions helped GDP to grow.

There is little knowledge on how macro- and micro-economic variables are related to local and global disease burden and how this relationship varies by disease and geographic area (Karpati et al, 2002). Moreover, there are still many barriers to understanding the global incidence and mortality rates for many diseases. While there is no real consensus on what the main cause of the decline in mortality over the past century could be, some authors have argued that an inverse correlation with economic growth is likely (Tapia Granados, 2005). Data from a variety of sources lead to the conclusion that only 20% of the massive international improvements in mortality that occurred between the 1930s and 1960s could be assigned to better living standards, measured in terms of per capita income (Preston, 1996). Interestingly, Sen suggested that the rate of decline of mortality in Britain between 1900 and 1970 reveals an inverse relationship with economic growth, with decades of high economic growth associated with low increases in life expectancy (Sen, 2001). Shifting our point of view slightly, the problem is that human health is both a product and a determinant of well-being and is strictly dependent on environmental health. Changes in the quality or quantity of environmental goods and services that regulate and affect the quality of food, air, water and soil can have very important impacts on human health.

The current sophistication of man-made environments reshapes biotic and abiotic characteristics and produces new patterns of human disease. Unfortunately, in recent decades, public health systems of developed countries have slowly moved away from environmental concerns, progressively narrowing their efforts on individual and genetic susceptibility to diseases, focusing intervention strategies on selective case management or specific disease prevention technologies in groups at risk.

Given that biomedical researchers are not accustomed to looking at their work within a historical and spatial perspective, usually they also neglect the ecological and evolutionary side of diseases. This bias prevents them from grasping the basic interaction between public health and contextual determinants of human diseases. Different societies living in different environmental

conditions (climate, geo-morphology, fresh waters, vegetation cover, etc.) interact with them in different ways. The structure of man-made environments and the functional correlation between their components reflect the particular patterns of interaction of anthropized (urban) contexts and public health. There is a clear indication that human ecology – in the broad sense of environmental variables, lifestyles, culture, and social organization – has a predominant role in shaping health and disease profiles.

## Conclusion

Human culture and technologies may act as a selective force affecting the environment, biology and health of *Homo sapiens* and other species. The evolution of human culture involves changes in the intergenerational transfer of ecological legacies, in the reconstruction of biological and social development's conditions, in the transmission of behavioural and symbolic information, as well as in the selective stabilization of survival practices and preferences (Jablonka, 2011). As such, human culture can be viewed as a “place” where cultural (economic, political, scientific, ideological, religious, etc.) beliefs meet with each other. This should provide the opportunity for rethinking the particular kind and scale of consequences that the human presence on Earth produces, both on the organization of the environment and on physical, mental and social health of our species.

Over the last decades we have dramatically learned that the paradigm of economic growth conceived by classical scholars is neither compatible with a public health system based on the preservation of well-being nor with a sustainable relationship between humans and the natural world. The adjective “sustainable” has often accompanied the term “degrowth” in order to stress that its meaning is linked to the improvement of well-being, social equity and the human-nature bond. Degrowth scholars are increasingly interested in the intersection between income and well-being. The Easterlin paradox refers to the lack of positive correlation over time between reported subjective well-being and income growth, at least for countries with sufficient means to meet basic needs (Easterlin, 1974). Moreover, the “threshold hypothesis” holds that, beyond a certain threshold point, economic growth does not bring about improvements in people's quality of life (Neef, 1995).

Degrowth should not be understood in its literal meaning of “negative growth of GDP”, or simply as a decline in well-being (Sekulova et al, 2013). Degrowth is a provocative word to challenge the ideology of growth (and its absurd implications) and to promote a different project of human society. “Degrowth” is an invitation to “decolonize the imaginary”, (Latouche, 2009), that is, to design different relationships with other human beings, with other creatures and, more generally, with the social and ecological environment. “In a degrowth society, everything will be different: different activities, different forms of thinking, different relations, different allocations of time between paid and non-paid work and different relations with the non-human world” (D’Alisa et al, 2014, p.4).

As Pope Francis has explicitly explained “The time has come to accept degrowth in some parts of the world, in order to provide resources for other places to experience healthy growth” (Pope Francis, 2015).

## Acknowledgements

I am grateful to my friends Michael Fox (veterinarian, USA), Alessandro Giuliani (biostatistician, IT), Giuseppe Masera (oncologist, IT), Monica Oldani (ethologist, IT), Vittorio Parisi (ecologist, IT), for the useful and comforting exchanges of ideas during the long and taxing lockdown due to the COVID19 epidemic in Italy.

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