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General review

The COVID-19 pandemic, an environmental neurology perspective

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ABSTRACT

Neurologists have a particular interest in SARS-CoV-2 because the nervous system is a major participant in COVID-19, both in its acute phase and in its persistent post-COVID phase. The global spread of SARS-CoV-2 infection has revealed most of the challenges and risk factors that humanity will face in the future. We review from an environmental neurology perspective some characteristics that have underpinned the pandemic. We consider the agent, SARS-CoV-2, the spread of SARS-CoV-2 as influenced by environmental factors, its impact on the brain and some containment measures on brain health. Several questions remain, including the differential clinical impact of variants, the impact of SARS-CoV-2 on sleep and wakefulness, and the neurological components of Long-COVID syndrome. We touch on the role of national leaders and public health policies that have underpinned management of the COVID-19 pandemic. Increased awareness, anticipation and preparedness are needed to address comparable future challenges.

1. Introduction

The World Federation of Neurology (WFN) Environmental Neurology Specialty Group has been concerned with the neurological aspects of the COVID-19 pandemic, which was officially declared by the World Health Organization on March 11, 2020. Given the involvement of the central nervous system (CNS) in coronavirus-associated Severe Acute Respiratory Syndrome (SARS), in the Middle East Respiratory Syndrome (MERS) and in early reports on the neurological complications of SARS-CoV-2, we had reviewed with a clinical approach the

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subject in depth [1] and along with others called for the development of national and regional registries to report neurological disorders in COVID-19 [2–4].

In this review, we point to some characteristics of the pandemic from an environmental neurology perspective. We consider the agent, SARS-CoV-2, the spread of SARS-CoV-2 as influenced by environmental factors, its impact on the brain in the short- and long-term, and some containment measures on brain health. We also address from a public health policy perspective, some aspects of the management of the COVID-19 crisis, and our community responsibility to improve the anticipation, mitigation and preparedness for foreseeable environmental disasters.

2. The agent, SARS-CoV-2 and its variants

We know that viruses pose a major threat to human health and notably to the human nervous system, which is targeted by half of all known viruses. Infectious diseases, notably zoonoses and epizootics, have and will continue to influence the destiny of *Homo sapiens* [5]. History shows that viral infections were responsible for several major reductions of the human population. Numerous studies published in the early 21st century have documented several recent virus-driven epidemics (e.g., SARS, Ebola, H1N1, Zika) and anticipated the socio-economic impacts of a pandemic. Therefore, the COVID-19 pandemic was absolutely not a surprise for virologists and health-crisis managers across the world; they warned many times about the threat of pandemics, notably the risks related to RNA respiratory viruses [6,7] and in particular to SARS-like coronaviruses [8]. Fan and colleagues [8] issued this stark warning in March 2019: ''It is generally believed that bat-borne CoVs will re-emerge to cause the next disease outbreak. In this regard, China is a likely hotspot. The challenge is to predict when and where, so that we can try our best to prevent such outbreaks.'' By December 2019, the first cases of COVID-19 were hospitalized in the Chinese city of Wuhan.

2.1. *SARS-CoV-2 discovery*

The new coronavirus responsible for the COVID-19 pandemic was successfully grown, sequenced and identified by three different Chinese teams, which published their results in January 2020. The International Committee on Taxonomy of Viruses (ICTV) named the emerging virus, Severe Acute Respiratory Syndrome CoronaVirus-2 (SARS-CoV-2), underlining its close relationship with SARS-CoV (now known as SARS-CoV-1) that was responsible for the SARS epidemic in 2003–2004. Concurrently, the World Health Organization (WHO) named the SARS-CoV-2-associated disease ''COVID-19''[1].

2.2. *The emergence*

The challenging question of the origin of SARS-CoV-2 is the subject of ongoing investigations. A One Health [9] approach to this question may help because it recognizes the interconnection and biological exchanges among people, animals, plants, and their shared environment. Since the precise origins, reservoirs, intermediate hosts, and routes to human transmission of numerous viruses are commonly ignored, progress in understanding the natural history of SARS-CoV-1 is remarkable [10]. The horseshoe bat appears to be the SARS-CoV-1 reservoir, and the palm civet an intermediate host [10]. As certain animal groups are susceptible to coronavirus infection (e.g., bats, camels, cats, cattle, mink), the risk of coronavirus spread from animals to humans is to be considered whatever the mechanism, spillover or circulation [11].

The question of a relationship between climate change and the rise of infective coronaviruses involves the distribution of bats which, as noted above, are considered reservoirs for these viruses. Climate change has shifted the global distribution of bat populations: ''The southern Chinese Yunnan province and neighboring regions in Myanmar and Laos form a global hotspot of climate change-driven increase in bat richness. This region coincides with the likely spatial origin of bat-borne ancestors of SARS-CoV-1 and SARS-CoV-2'' [12].

The human role in the emergence and spread of zoonotic infections is huge. Humans are responsible for habitat fragmentation, deforestation, biodiversity loss, intensive agriculture, livestock farming, uncontrolled urbanization, pollution, climate change, and bushmeat hunting and trading, all of which are relevant to the emergence of pathogens that can impact human health [13]. Vast numbers of germs circulate undetected in fauna and flora, many and perhaps most of which remain to be discovered. In addition, we know that viral infection (e.g., Epstein–Barr virus or Herpes viruses) does not necessarily trigger overt disease, which is the usual warning sign leading to a medical or veterinary response [11]. Additionally, certain infectious agents, such as the Measles virus, may mutate in the human body, develop a clinically silent latent infection in the brain, only to be expressed later in the form of a devastating brain disease (Sub-Acute Sclerosing Panencephalitis). Fortunately, there is no evidence that SARS-CoV-2 establishes a persistent infection. Whatever the debated origin of the virus [14] and its more contagious recent mutants, this is the second time in less than 20 years that an atypical coronavirus pneumonia with human-to-human transmission has appeared in China. The human disease named COVID-19 officially began in late November 2019 and, thereafter, spread across the world.

2.3. *A neurological concern*

The neurologists interest in SARS-CoV-2 lies in the characteristics of virus-cell interaction that allows the virus to entery CNS tissue. The primary binding targets of the virus are the angiotensin-converting enzyme-2 cell membrane receptor (ACE2) and the transmembrane serine protease 2 (TMPRSS2), both of which are less expressed by brain cells compared to those of the heart, lung and nose [15,16]. Therefore, SARS-CoV-2 must interact with other receptors e.g., Neuropilin 1 (NRP1), Cathepsin L (CTSL) and furin which are expressed at much higher levels in the brain [16,17]. Qiao and colleagues [15] have shown, with cell lines and mouse tissue, that the CD147

[1] www.who.int/emergencies/diseases/novel-coronavirus-2019/technical-guidance/naming-the-coronavirus-disease-(covid-2019)-and-the-virus-that-causes-it.

receptor (which belongs to the Ig superfamily) is more expressed than ACE-2 in brain cells compared to lung and intestine cells. Although debated, the role of the CD147 receptor in the entry of SARS-CoV-2 is recognized [17].

A second concern is related to the ongoing evolution of the virus responsible for COVID-19, evolving from the Wuhan wild-type SARS-CoV-2, since the first infections, to numerous lineages or variants due to mutations that routinely occur during replication of its genome [18–20]. For example, 5,775 distinct genome variants, including 2,969 missense mutations, 1,965 synonymous mutations, 484 mutations in the non-coding regions, 142 non-coding deletions, 100 in-frame deletions, 66 non-coding insertions, 36 stop-gained variants, 11 frameshift deletions and two in-frame insertions were identified through May 1, 2020 [20]. Most mutations have little to no impact on the virus's properties, e.g., transmissibility, interaction with host immunity, and infection severity [18]. Newly detected variants are clinically documented allowing a classification based on additional information as country first detected ; virus spike mutations of interest, year and month of virus first detection,performance of vaccines and medicines, and diagnostic tools [18,19,21]. In May 2021, WHO defined three main categories of SARS-CoV-2, namely variants of concern (VOC), variants of interest (VIC), and variants under monitoring (VUM). A VOC has one or more mutations that may impact its epidemiologic, immunologic and pathogenic properties [20]. WHO recommended using the Greek alphabet a to name several variants, e.g., Alpha, Beta, Gamma, Delta, Omicron. The emergence of these variants, especially the latter, poses a major health threat and challenge for the control of the pandemic [22], at least in the short term. The pathogen's diversity and dynamics also illustrate the adaptation-driven evolution of the virus [23]. The various clades (lineages), have different geographical distributions and may perhaps trigger different clinical presentations, for example clades G and GV showed a significantly higher prevalence among asymptomatic patients or those with mild symptoms [23]. Comparison among recent lineages shows that some variants are more transmissible and fatal than their ancestor [24]. For example, Delta is more transmissible and increases the hospitalization and mortality rate when compared to Alpha [25], whereas Omicron is highly transmissible but, at this writing, is thought primarily to infect the upper respiratory tract, and less the lungs, thus resulting in a milder disease in most patients [26].

A third question concerns the capacity of the new variants to impact the nervous system. The 2020 Wuhan wild-type virus triggered the quasi-pathognomonic symptoms of anosmia and dysgeusia in most countries; however, in China, these symptoms were uncommonly reported (around 5%). One hypothesis is that the Chinese people were infected by another clade [27]. Symptoms have changed with the new variants. For example, anosmia is uncommon when infected by Omicron; in the ZOE COVID study, anosmia affected around 19% of study participants compared to 60% related to infection by wild type or Alpha variants [28]. The cell-entry of Omicron differs from that of the other SARS-CoV-2 variants; its binding capacity-capacit y to ACE2 is lower and Omicron also uses an endocytic pathway [29]. Unknown is w hether and how these characteristics affect the ability of this variant to infect brain cells . .

3. The infected human host

3.1. *Impact on the human brain, the neurological manifestations of COVID-19*

While involvement of the CNS in coronavirus-associated Severe Acute Respiratory Syndrome (SARS) and the Middle East Respiratory Syndrome (MERS) were well-known [30], the pathophysiologic mechanisms are still unclear [31]. SARS-CoV-2 appears to be a neurotropic virus able to infect neurons and glia [32], although viral particles have been rarely found in human brain and its presence in the cerebrospinal fluid is inconstant [16,32]. Additionally, evidence is largely lacking for direct CNS invasion of SARS-CoV-2 as a primary cause of neurologic sequelae [31]. Nevertheless, whether infection occurs directly or indirectly via the hematogenous route, or whether the resulting immunological response (cytokine storm) [16] plays the major role, there is a large spectrum of neural targets, including the brain, medulla oblongata, spinal cord, peripheral nerves and muscles. Thirty percent of symptomatic COVID-19 patients present with CNS involvement during the acute phase of the disease. Anosmia and dysgeusia are considered quasi pathognomonic symptoms of COVID-19, with smell and taste disorders occurring in up to 80% of patients [33]. Neurological symptoms affect 73% of hospitalized patients [30].

3.2. *Impact on the human brain and mind: neuropsychological manifestations of COVID-19*

After the acute phase of the illness, 10 to 30% of COVID-19 patients continue to experience pulmonary, cardiovascular and/or neurological symptoms, sometimes for several months. The so-called ''long-haulers'' with this post-acute syndrome (''Long-COVID'') often have multiple symptoms referable to the nervous system, including chronic fatigue (30–78%), headache (18–50%) cognitive symptoms (e.g., attention disorder, memory loss, anxiety), sleep disorder (11–65%) and smell/taste dysfunction (10–43%) [34]. Among the vast published literature on Long-COVID are papers describing findings with positron-emission tomography/computed tomography (PET/CT) that have documented changes in the regional uptake of 2-desoxy-2-fluoro-D-glucose (FDG). At the onset of COVID-19, four patients with predominant frontal lobe cognitive impairment, whose cerebrospinal fluid was RT-PCR negative for SARS-CoV-2, showed hypometabolism and cerebellar hypermetabolism [35]. Persistent functional complaints of 35 patients with Long-COVID correlated with evidence of brain hypometabolism involving the olfactory gyrus and connected limbic/paralimbic regions, extending to the brainstem and the cerebellum [36]. A serial study of seven patients with heterogenous encephalopathy at onset of COVID-19 showed a consistent pattern of hypometabolism in the frontal cortex, anterior cingulate, insula and caudate nucleus. Six months later, the majority of these patients had improved clinically but cognitive and emotional disorders of varying severity remained in association with prefrontal, insular and subcortical 18F-FDG-PET/CT abnormalities [37]. Other studies have used this type of brain imaging to document such biological markers of Long-COVID [38,39].

Sleep disorders may occur during the disease course and/or after recovery [1,40,41]. Most studies of this phenomenon have relied on questionnaires administered to the general population and/or specific human groups (e.g., students, medical staff, etc.). A number of studies conducted around the world found that sleep quality deteriorated during lockdown episodes, especially among women. This was the case in France [42], where people over 18 years of age were asked whether they encountered sleep problem at the beginning (1,005 persons), the end (2,003 participants) and following (1,736 people) the Spring 2020 lockdown. Sleep problems diminished towards the end of the quarantine period and vanished progressively afterwards. In addition to insomnia, alterations in circadian sleep schedules were revealed through a bedtime phase delay of two hours (from 22:00 to midnight) and a similar trend for rise time [43]. In 368 Saudi Arabia university students in quarantine, nightmares (a REM sleep parasomnia) were experienced in 31.8% of the participants, of whom 44.4% suffered new-onset nightmares [44]. These figures were increased when academic exams were conducted during quarantine, indicating that nightmares were dependent on stressor intensity. Using natural language processing tools, dream reports were analyzed [45] and revealed higher proportions of words related to anger and sadness, in support of mental suffering. We may thus conclude that the measures adopted by authorities in every country have led to changes in sleep behavior and/or quality. Nevertheless, the sleep of COVID-19 patients has been widely overlooked. An early meta-analysis [46] included 31 publications on 5,153 patients with confirmed COVID-19. The authors found that 47% of patients experienced anxiety, 45% depression and 34% sleep disturbances (defined as poor sleep quality, sleep initiation or maintenance, excessive somnolence, sleep-schedule disorders or insomnia). However, objective findings on sleep illness are rare. One patient with PCR-confirmed COVID-19 presenting fever, asthenia and insomnia declared restless leg syndrome that was in favor of iron dysmetabolism [47]. In turn, sleep-related disorders may also influence COVID-19 morbidity and mortality. Obstructive sleep apnea (OSA) syndrome was identified early as a risk factor [48], although obesity is commonly observed in OSA patients and represents in itself a major risk factor of morbidity and mortality in COVID-19 patients. Circadian rhythm alterations, associated with the psychological problems imposed by the COVID-19 pandemic, compromise the quality of sleep and the immune system, as sleep influences immunity maintenance and immune responses [49].

Our group and others also alerted the scientific community on disorders potentially appearing after recovery. The possible occurrence of narcolepsies was argued [50], as such a syndrome had occurred in Chinese children after the 2009 H1N1 influenza outbreak, as well as in children in Europe and Brazil after pediatric vaccination. Such disorders of sleep and circadian clock are also observed in other infectious diseases, such as African sleeping sickness [51].

3.3. *Human vulnerabilities and susceptibilities*

3.3.1. *The human susceptibility to SARS-CoV-2: the severity of COVID-19*

The COVID-19 pandemic has revealed and highlighted specific vulnerabilities of several human populations to virus infection. Biological vulnerability in humans is linked with age, gender, genetic and ethnic background, and preexisting comorbid illness.

3.3.1.1. Genetic factors. Genetic factors may modulate susceptibility/resistance to SARS-CoV-2 infection and the severity of the resulting illness [52,53]. They include gene polymorphisms and comorbidities, such as the association of ACE1 with cardiovascular disease, hypertension, diabetes, chronic kidney disease, and obesity, and of alpha-1 anti-trypsin deficiency with lack of control over inflammatory mediators [52]. The polymorphism of genes encoding ACE2 and the transmembrane protease serine 2 (TMPPSS2) influence individual susceptibility to COVID-19 [54]. With regard to blood group phenotype, there is weak evidence that subjects with group-O are less susceptible than non-O individuals to SARS-CoV-2 infection, while the O type has no detectable effect on COVID-19 severity [55]. A recent review examined the susceptibility to SARS-CoV-2 and the polymorphism of a large number of genes involved in the different stages of virus-cell interactions [56]. ''Classical twin studies indicated 31% heritability for predicted COVID-19'' [53]. Substantially higher death rates from COVID-19 among Africans and African-Americans may be related to several gene polymorphism, e.g., *androgen receptor* gene (AR), *ACE* gene, ACE2 expression, *apolipoprotein E* gene (APOE), in African-American males. A lower frequency of a gene variant encoding for the IFIH1 protein, which is involved in innate immunity, has also been shown [53].

3.3.1.2. Evolutionary history. The evolutionary history of *Homo sapiens* provides some interesting clues as to the differential susceptibility among people with disparate ethnic backgrounds and geographical origins [57]. Some Neanderthal ancestry genes located on chromosome 12 (OAS locus) may be protective (20% reduction of relative risk) [58] while other Neanderthal-inherited genes located on chromosome 3 may increase the risk of severe COVID-19 [59]. An ACE2 haplotype present among 20% and 60% of European and South Asian populations respectively, has been linked to a lower fatality rate among South Asians [57]. Evolutionary genetics has shown that genomes of East Asian populations bear the signature of a *circa* 25,000-year-old coronavirus epidemic; this interaction between ancient coronaviruses and human hosts, shown by the presence of selected gene variants encoding virus-interacting proteins, suggests adaptive mechanisms in East Asian populations [60].

3.3.1.3. Neutralizing immunoglobulin G autoantibodies. In addition to genetic abnormalities involving the innate immune system, neutralizing immunoglobulin G (IgG) autoantibodies (ABs) against various interferons (omega, alpha, both and I INF) have been observed. These ABs account for life-threatening COVID-19 in at least 2.6% of women and 12.5% of men [61]. In addition to the biased sex ratio, the ABs against I IFN offer an explanation for the higher risk of critical COVID-19 in patients over 65. Rare before age 65, the ABs are more often detected from 4% (range 70 to 79 years) to 7% thereafter [62]. Therefore, 25% of severe cases of COVID-19

might be explained by genetic and immunologic abnormalities of the host[2].

3.3.1.4. Gender-associated risks for severe and fatal forms. Epidemiological studies have pointed to gender-associated risks for severe and fatal forms of COVID-19, such that the risk is double for adult males vs. females. Biological factors include sex hormones that differentially regulate the innate and adaptative immunological system, its interaction with adiposity, the entry of SARS-CoV-2 into cells (ACE2, TMPPSS), and probably the susceptibility to tissue injury [63]. Several studies point to an inherited X-linked recessive *TLR7* gene deficiency in a small percentage of younger-adult COVID-19 cases with severe forms of COVID-19 [62,64]. The *TLR7* gene is involved in the innate immunity response; its loss-of-function impairs the types I and II interferon pathway [64]. Other important components of innate immunity are the mucin proteins that offer first-line protection to all epithelial cells, in combination with mucosal antibodies, the secretory IgA. In the case of SARS, epidemiological data suggested sex-differences, as human females may be more resistant than human males. However, until now, no data on this topic are available for SARS-CoV-2 [65].

3.3.1.5. Senior patients. Early victims of COVID-19 were senior patients (death occurred mostly in >80-year-old persons) and those with multiple risk factors, such as hypertension, obesity, diabetes and cardiovascular disease. Surprisingly, children appeared less prone to illness, such that the death rate in this group has been relatively very low, although rare fatal complications such as Multisystem Inflammatory Syndrome (MIS) and Kawasaki syndrome have occurred. Several hypotheses have been proposed to explain the age-related differential biological vulnerability to SARS-CoV-2, with particular focus on the density of the ACE-1 virus receptor and variability in the body's immune response to the virus [66].

3.3.2. *Is there a specific neurological vulnerability?*

3.3.2.1. The brain's susceptibility to SARS-CoV-2: olfactory epithelium and vascular endothelia. COVID-19 related loss of smell and taste is significantly less likely affected in East Asian and African American individuals than among Europeans, a phenomenon linked to the UGT2A1/UGT2A2 locus [67]. These genes code for enzymes expressed in the olfactory epithelium involved in the clearance of odorants. Loss of smell is related to damage of the cilia and olfactory epithelium but not to an infection of olfactory neurons. Recent evidence suggests that SARS-CoV-2 enters and accumulates in olfactory support cells, specifically, sustentacular cells, which unlike olfactory neurons, abundantly express the viral cell-entry proteins, ACE2 and TMPPSS2 [67].

The seven-fold increased risk of stroke in COVID-19 appears to be associated with a specific vulnerability of the brain's endothelial cells [68] resulting in dysfunction of the endothelium [67].The direct infection, which may trigger localized phenomena including thrombosis and cellular permeability, occurs through a flow-dependent expression of ACE2, as ACE2 is physiologically poorly expressed in these cells. ''Viral S protein binding triggers a unique gene expression profile in brain endothelia that may explain the association of SARS-CoV-2 infection with cerebrovascular events'' [68]. ACE2 expression is also increased in case of arterial hypertension [68].

3.3.2.2. Neurological diseases as risk factor for COVID-19 severity and complications. There is no evidence at this time that preexisting neurologic diseases increase the risk of the occurrence of neurologic complications in COVID-19 [31]. However, underlying neurological diseases constitute a risk factor for developing a severe form of COVID-19, particularly those with significant bulbar and respiratory weakness (e.g., neuromuscular disorders) or other neurologic disability [31]. Obviously, such factors increase COVID-19 severity, worsening the underlying disease and provoking a higher mortality rate [31]. Disabilities related to cognitive impairment [31], Alzheimer's disease [69] and Multiple Sclerosis (MS), assessed by the EDSS score [70], are independent risk factors for severe COVID-19. However, no association has been found between MS Disease Modifying Treatments and COVID-19 severity [71]. A preexisting mental illness worsens clinical outcomes in COVID-19, with a doubled rate of more severe course and mortality compared to patients with no mental disorder [72]. Another hypothetical concern is related to the risk of neurodegenerative disease, triggered or induced by the SARS-CoV-2 infection, leading to a delayed disorder, such as Parkinson's disease or dementia [1,69,73].

3.3.3. *The human way-of-life: the risk of exposure and infection by SARS-CoV-2*

Vulnerability also takes into account the probability of exposure/infection by SARS-CoV-2 as a function of personal occupation, housing conditions, education, and other factors. Thus, this pandemic has shown that COVID-19 has a very unequal occurrence in populations across the world; risk factors are linked to socio-economic status, including those in poverty and low-class jobs, and to minorities, migrants and refugees [74]. The built environment and high urban population density in megacities also favor infectious disease transmission [75]. Health policy has had to address cultural behaviors as well as religious beliefs and practices. Several communities have ignored or rejected common health-protection advice and mandates issued by medical and state authorities [76]. To the contrary, most religious leaders have encouraged those in their charge to accept public health rules issued by authorities [77].

As pointed out before, pandemics reveal societal vulnerabilities linked with lifestyle and values. Most societies and communities have adopted a lifestyle based on individualism, ''liberty'' and freedom of movement. Today life is characterized by an extraordinary increase of rapid and widespread transportation means, temporary or permanent national and international migration, and progressive urbanization resulting in crowded megacities; all of these behaviors can promote the spread of infectious agents and associated human and animal diseases. Socioeconomic and demographic drivers relevant to viral transmission from

[2] https://presse.inserm.fr/un-quart-des-formes-severes-de-covid-19-sexpliquent-par-une-anomalie-genetique-ou-immunologique/43635/.

wildlife to humans and among humans include ecosystem conversion, meat consumption that requires deep changes in land-use and agricultural practices, urbanization, and connectivity among cities [78]. Mass gathering, which is known to favor the spread of epidemic diseases, is another societal trend [79]. There is little doubt that Superspreading social or cultural Events (SSE) have been a major cause for the rapid spread of SARS-CoV-2 in the human population [80]. Several SSE-associated cases have been scrutinized, for example those in Boston USA [81] and in Austria [82]; these share common characteristics [80]: ''Closed environments, environments with poor ventilation, crowded places, and long durations of potential exposure''. Recognized since the SARS epidemic, the rapid worldwide spread of SARS-CoV-2 was related to international air traffic patterns and the intermingling of people in airport hubs. As in the case of SARS, the diffusion of SARS-CoV-2 may be modelled as ''a function of airline network accessibility'' [83]. On the other hand, while in-flight transmission of SARS-CoV-2 among airline passengers has occurred, this has not become a major problem apparently because face masking is enforced and the flow of air and its filtration reduce ambient contamination .

4. Virus transmission from human-to-human and the role of environmental factors

4.1. *Airborne transmission*

Although SARS-CoV-2 is an airborne transmissible virus, the routes of human contamination have been debated, especially by the WHO World Health Organization. Acceptance that COVID-19 results from human contact with an aerosol-transmissible virus, rather than from contact with surfaces contaminated by droplets and fomites, took a surprisingly long time, despite several publications [84,85].

4.2. *The human-to-human contamination*

A remarkable aspect of the COVID-19 pandemic is the variation in human-to-human virus transmission, favored at the individual level by virus superspreaders and at the community level, by SSEs, phenomena that were well-known before the COVID-19 pandemic [86,87]. These facts have major public health consequences as SARS-CoV-2 transmission is stochastic [80]. Secondary viral transmission is described by the mathematical overdispersion k parameter. Thus, with $k = 0.1$, around 10% of contaminated people are responsible for 70–80% of all secondary transmissions [88]; in other words, a single superspreader can infect a disproportionate number of contacts [87]. If correct, this means that most individuals (40–70%) did not infect anybody else [89]. These data were obtained from study of subjects infected with the Wuhan SARS-CoV-2 wild type.

Early in the pandemic, attention was drawn to the k parameter [90]; unfortunately, identification of superspreaders has been retrospective [87]. Their biological capacities remain unknown [87] even though several hypotheses have been proposed, including an ability to propagate the virus better because of higher viral particle emission [80], i.e., loud speaking resulting in the exhalation of more virus-loaded air [89] and, of course, the number, proximity and duration of interpersonal contacts [80] in indoor vs. outdoor settings with/without masking. When tracing is possible, identification of critical factors promoting viral transmission may lead to public health protective measures [80].

The transmissibility of the different variants (Alpha, Delta and Omicron) versus wild-type SARS-CoV-2 must be taken into account, as these variants have a higher transmissibility and higher secondary infection rate. A Thai retrospective study found that the secondary infection rate in households increased from 16.6% (known rate for the wild-type) to 48% (for certain contemporaneous variants) [91]. At a Finnish hospital, an index case triggered an outbreak due to the Delta variant, infecting 58 patients and 45 health care workers (HCW). ''Transmission occurred despite the use of personal protective equipment by the HCW, and a high two-dose COVID-19 vaccination coverage'' [92]. So-called breakthrough cases, in which SARS-CoV-2 provoked COVID-19 in fully vaccinated people, seem to be increasingly common with the Omicron variant, although individual susceptibility will vary with the vaccine type, the number of doses, and the time since vaccination.

4.3. *COVID-19, air pollution and weather-climate*

Among the huge number of publications related to COVID-19, a search in ''PubMed'' (30 December, 2021) using the search terms ''COVID-19 and air pollution'' and ''COVID-19 and climate'' retrieved 1,364 and 2,067 references, respectively. Here, we shall briefly examine the impact of air pollution and/or meteorological conditions on the survival, spread and infectivity of the SARS-CoV-2, and their impact on the evolution and clinical presentation of COVID-19.

At the beginning of the pandemic in 2020, Italian and Chinese scientists, joined by other Western countries teams, concluded that air pollution seemed to favor the aerosolized spread of SARS-CoV-2 [93–95]. High rates of air pollution, notably due to particulate matters (PM), may also lower the host's immune system and thereby favor viral infection. Air pollution is a major risk factor for several non-communicable diseases, creating a preexisting poorer health condition that is linked with an increased susceptibility to SARS-CoV-2, COVID-19 severity, hospitalization and risk of death [93–95]. Among air pollutants, PM_{10} (particles 10 μm in diameter) may serve as carriers attaching viruses that spread in ambient air. As inhaled PM carries microorganisms inside the body, PM act as a cofactor and may contribute to the accentuated cardiovascular effects of COVID-19. PM reportedly may promote clinical severity and increase the risk of death. In the USA, an ecological regression analysis showed that an increase of 1 $\mu g/m^3$ of $PM_{2.5}$ was associated with an 11% increase in the COVID-19 death rate [96]. Another gaseous pollutant, NO_2, a marker for traffic-related air pollution, is also involved in the pandemic. In Los Angeles County neighborhoods, the annual NO_2 level is associated with COVID-19 incidence and mortality [97].

These findings were discussed by the European Union's Joint Research Centre (JRC) team [98]. In October 2020, the JRC issued a 36-page report that analyzed the literature published

between March and July 2020. The report found that ''a significant impact of outdoor air pollution on the spreading or severity of the disease has not been demonstrated yet'' because of methodological shortcomings in many articles. However, the report attributed 7% of COVID-19 deaths to air pollution. Reviewing several articles issued in the first semester of 2020, Bourdrel and colleagues [99] concluded that the relationship (association and/or causality) between air pollution and the pandemic remained unclear, although the health effects of air pollution are supported by experimental and epidemiological data [100].

Presently, interest in the contribution of environmental factors to COVID-19 is extended to all meteorological and air pollution conditions [101–103]. Thus, it is established that the virus favors cold, dry and polluted air. Another aspect is the inequity in human exposure to polluted air, which is higher in industrial regions (e.g., the Po Valley in Italy) versus rural areas [93] and also in some American counties inhabited by ethnic communities (Blacks and Latinos) and lower socioeconomic groups [95,96]. This factor also contributes to the differential COVID-19 mortality rate among different populations worldwide.

Along with air pollution, meteorological characteristics (temperature, humidity, wind speed, UV radiation) have often been investigated ''based on unreliable data and questionable modelling techniques that did not account for numerous factors that were co-occurring at the beginning of the pandemic'' [104] leading to incomplete and even false policy conclusions [105]. Therefore, the World Meteorological Organization (WMO) established a COVID-19 Task Team [103] to review available data. Elaborated since September 2020 and published in January 2021, the WMO report stated notably: ''Epidemiological studies of COVID-19 have, to date, offered mixed results regarding the meteorological sensitivity of the virus. Laboratory studies of SARS-CoV-2 have yielded some evidence that the virus survives longer under cold, dry and low ultraviolet radiation conditions'' [103]. The role of meteorological factors is now accepted; for example, their role in the USA is higher in northern than southern counties. They act on SARS-CoV-2 transmissibility, which is moderately associated with cold and dry weather and low levels of ultraviolet radiation, with humidity playing the largest role [106].

Mathematical modelling using a logarithmic regression to analyze the worldwide distribution of COVID-19 cases (less prevalent in countries closer to the equator, where heat and humidity are higher) predicted in January 2021 that the ''threat of epidemic resurgence may increase during winter'' and conversely decrease in summer, although not vanishing [107]. The resurgence of Delta and the exponential spread of Omicron are consistent with this prediction. ''An increase in absolute latitude by 1 °C is associated with a 4.3% increase in COVID-19 per 10^6 inhabitants'' [107], confirming that SARS-CoV-2 is sensitive to temperature and longer sunlight exposure.

4.4. *The possible effects of global chemical contamination*

During the first pandemic wave, the high rate of COVID-19 deaths in some parts of Europe (Northern Italy, France, Spain, and UK) compared to other regions (Germany, Switzerland, Austria, and Denmark) was reported to correlate with relative levels of environmental contamination (air pollution, pesticides, dioxins, chlorinated water), which may affect immune function [108]. The efficacy and persistence of post-vaccination immunity to SARS-CoV-2 should consider the impact of exposure to environmental pollutants that may modify immune responses [100].

5. Environmental and health consequences of non-pharmaceutical interventions

5.1. *Lockdown, air pollution and stroke*

The first prolonged lockdown of populations designed to slow and stop the spread of COVID-19 drastically decreased air pollution worldwide [109]. However, the decrease was differential, with a drop of nitrite oxides (NOx) related to car traffic, but the persistence of the pollution by PM produced by building heating as well as agricultural practice. In France, an unexpected and marked reduction in mortality rate, mostly in low COVID-19-incidence departments, occurred during the first quarantine period [110,111], being associated with a drop in the stroke-hospitalization rate [110,112]. However, the interaction between reduced air pollution in March 2020 and the increase in the risk of stroke induced by SARS-CoV-2 infection, has not been properly addressed [112].

Substantial human health benefits related to cardiovascular disease morbidity and mortality were linked with the COVID-19-related lockdown in China, where levels of vehicular air pollution (NOx, PM 2.5) were also substantially reduced [113]. Additionally, China's strict lockdown and quarantine policies, border and travel controls, prompt virus tracing and widespread immunization of its population, greatly reduced the number of COVID-19 cases, hospitalizations and deaths relative to the experience of Europe and the USA. By the end of 2021, the U.S. population had experienced > 150-fold the number of reported COVID-19-related deaths than the official number in China, a country with a population > 4 times that of the U.S. [114].

5.2. *Psychologic stress and crisis management*

Neurologists cannot ignore the major impact of the COVID-19 pandemic on mental health and sleep quality (*vide supra*). Already recognized (in 2019) as leading causes of the global health-related burden, depressive and anxiety disorders increased worldwide in 2020 in prevalence and burden [115]. In their review, Brooks and colleagues anticipated the psychological impact of COVID-1, based on previous knowledge (specifically the societal disruption that lockdown, infection, and quarantine would cause). ''Most of the adverse effects come from the imposition of a restriction of liberty'' [116]. They pointed also to populations at-risk, namely health workers and people with preexisting psychiatric illness. They recommended several measures to reduce the expected psychological consequences of lockdowns. Clearly, crisis management can be a direct cause of psychosocial distress.

6. Crisis management and responsibilities: should we enlarge the scope?

Decision-making during the COVID-19 pandemic was influenced by gender, determining differences in public health measures taken by female versus male leaders [117,118]. In the Spring 2020, lower death rates were observed in countries with governments led by women as in Denmark, Finland, Germany, Norway, Iceland, New Zealand and Taiwan, as well as in several U.S. and Brazil states. By contrast , several male leaders lacked (and still lack) anticipation or denied the magnitude of the health crisis. Women politicians showed greater empathy [117] and use of less aggressive words and war metaphors [119] than their male counterparts.

Widely broadcasted, communication from political leaders led to the false belief that the pandemic was caused by an unknown virus. For Frankel [120] at the Harvard Kennedy School and knowledgeable analyst; however, the viral pandemic represented a ''known-unknown'' phenomenon not an ''unknown-unknown'' entity, and was likely to occur. Some governments and so-called experts released false opinions to the media to hide ignorance and lack of preparation [121], not considering the fake news peddled by some social networks. The critical importance of aerosol transmission of respiratory viruses was originally overlooked [84], although Flügge droplets were described in the 1890s, and SARS-CoV-1 was known to diffuse indoor via airborne aerosols [122], reaching distances $>$ 60 m [123].

The medico-scientific community also failed to anticipate the debilitating post-infectious syndrome, Long-COVID, although the comparable Myalgic Encephalomyelitis/Chronic-Fatigue Syndrome (ME/CFS) had been described six decades ago [124] and also acknowledged in SARS [125]. A long and indefensible debate about treatments, notably for outpatients with benign/mild COVID-19, took place in the early phase of the pandemic [126]. Unethical practices, such as therapeutic abstention, were not exempt of health consequences regarding lack of treatment, postponed hospitalization and increased morbidity and mortality [127].

Public health policies were blurred by inertia, ideology and ignorance (the devastating three Is, recognized in the fight against global poverty [128]). On January 15, 2020, six days after WHO announced a coronavirus-related outbreak in Wuhan (China), the World Economic Forum published their 2020 Global Risk Report [129] and deplored the unpreparedness against outbreaks of new emerging infectious diseases. The unpreparedness was such that the health threat was minimized by ''experts'', journalists and prominent politicians [130,131], except for some countries [132]. As a consequence, successes and failures should be scrutinized carefully by experts [133].

Teaching environmental health, risk-management and decision-making under uncertainty [134] should be considered as an absolute necessity for political leaders and decision-makers, and a large panel of disciplines (neuroscience, anthropology, virology, immunology, veterinary medicine, biology, but also political sciences). This represents a unique opportunity to reach a better level of awareness, anticipation and preparedness for future environmental health shocks. Anticipation, mitigation, and avoidance of foreseeable disasters will require increased surveillance, detection and identification of a new disease surfacing in humans and/or animals. Therefore, as proposed by the Lancet COVID-19 Commission [135], the teaching procedure should promote, via an independent international council, the evaluation of public health policies and that of the benefit/risk ratio of the different treatments and vaccines.

7. Conclusions

The COVID-19 pandemic may be seen as a global human health catastrophe triggered by an environmental agent and amplified by human behavior. Analysis of the acute, chronic and potential long-term neurological impact of COVID-19 is susceptible to the methods employed in environmental neurology. This holistic approach is praised by Horton in his editorial ''COVID-19 is not a pandemic'' but ''a syndemic characterized by biological and social interactions, as well as a broad array of human beliefs and behaviors that increase individual susceptibility to adverse health outcomes'' [136]. The link between the COVID-19 pandemic, environmental factors, climate and health has also been highlighted by the HERA-COVID-19 working group [137].

Changing humanity and its foolish behaviors is a utopian dream. However, two possible approaches exist: top-down regulation and bottom-up pressures. Societal awareness and activist protestation can both offer creative solutions and influence decision-makers' policies. Anticipation is mandatory if we acknowledge Walt's statement that another pandemic ''is not a matter of if but when; we as individuals, our nation, and the entire global community need to be prepared'' [138]. Are our societies prepared to meet this challenge? With the laudable and necessary goal of preserving the brain health of individuals and populations worldwide, how can our community best prepare and mitigate the next predictable pandemic or ecological disaster? In any case, the neurology community should enhance its preparedness by improving surveillance and research in prevention, diagnostic, treatment and communication tools and procedures to improve the ways to face a future crisis such as the COVID-19 pandemic.

Since the acceptation of this article, important articles were published, related to Schizophrenia as risk factor for severe COVID-19 patients [139] and to the delay of the WHO agreement about the role of bioaerosols in the propagation of SARS-CoV-2 [140].

Disclosure of interest

The authors declare that they have no competing interest.

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