



Review

An updated min-review on environmental route of the SARS-CoV-2 transmission

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ABSTRACT

The risk of newly emerging diseases is constantly present in a world where changes occur significantly in climatic, commercial, and ecological conditions, in addition to the development of biomedical investigations in new situations. An epidemic respiratory disease instigated by a new *coronavirus* was initially identified in and has resulted in the current global dissemination. This viral strain and its related disease has been termed “SARS-CoV-2” and “coronavirus disease 2019” (abbreviated “COVID-19” or “2019-nCoV”), respectively, which is transmitted simply between individuals. The World Health Organization (WHO) announced the COVID-19 outbreak as a pandemic on March 11, which necessitates a cooperative endeavour globally for mitigating the spread of COVID-19.

The absence of previous, and minimum present-day information, particularly concerning the path of contagion have precluded the control of this disease. The present article, therefore, describes the SARS-CoV-2 paths of contagion such as drinking water, solid waste, sewer water, ambient air, and the rest of emerging likely paths.

1. Introduction

As classified by the International Committee for Taxonomy of Viruses (ICTV), *coronaviruses* (CoV) are a member of the genus *Coronavirus* in the *Coronaviridae* family (Gorbalenya et al., 2020). Pleomorphism is seen in the RNA of all CoVs viral strains, characterized by having crown-shaped peplomers 80–160 nm in size (Fehr et al., 2017; Fehr and Perlman, 2015; Sahin et al., 2020). The viruses have a positive-sense, single-stranded ribonucleic acid (RNA) genome with a length in the range of 26–32 kilobases (kb) hence containing the biggest RNA of viral genome (Ahmad and Rodriguez-Morales, 2020; Fehr et al., 2017; Roosa et al., 2020). Recombination of CoVs occur at intensely high rates since there are constant development of transcription errors and RNA-related

RNA polymerase (RdRP) vaults (Sahin et al., 2020). Such a high mutation rate renders *coronaviruses* zoonotic pathogens found in humans and a variety of animals with a wide-ranging clinical properties, from an symptomless period to the need for hospital care in the intensive care unit, which causes infections in the respiratory, gastrointestinal, hepatic, and neurologic systems (Desforges et al., 2019; Fehr et al., 2017; Roosa et al., 2020; Rothan and Byrareddy, 2020). The viruses are sometimes capable of causing more severe disease in young, elderly, or immunocompromised people (Rothan and Byrareddy, 2020; Tang et al., 2020). These viral strains are subdivided into four major subclasses of alpha, beta, gamma, and delta. Alpha and beta coronaviruses have their mammalian (bats) origins, whereas gamma and delta coronaviruses are of swine and avian origins (Burrell et al., 2017; Tao et al., 2017; Wang

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et al., 2019). In humans, infection is caused by seven coronaviruses, namely HCoV-229E (*alpha coronavirus*), HCoV-NL63 (*alpha coronavirus*), HCoV-OC43 (*beta coronavirus*), HCoV-HKU1 (*beta coronavirus*), Middle East Respiratory Syndrome, or MERS-CoV (*beta coronavirus*), Severe Acute Respiratory Syndrome, or SARS-CoV (*beta coronavirus*) (Burrell et al., 2017), and the Novel Coronavirus (2019-nCoV) detected recently in 2019 (Ahmad et al., 2020; Lippi et al., 2020), known as SARS-CoV-2, which causes the Coronavirus Virus Disease of 2019 (COVID-19). Four of seven common human CoVs (HCoVs) result in common self-limited upper respiratory diseases: HCoV-229E, HCoV-OC43, HCoV-NL63, and HCoV-HKU1 (Chang et al., 2020). Of more than 60 coronaviruses (CoVs) isolates obtained from bats (BtCoV), the majority belong to the genus *betacoronavirus*. Bats function as huge (and very movable) CoV sources; most of bat species possess their specific exclusive BtCoV, which suggests that they have coevolved over a highly prolonged history (Payne, 2017). In humans, coronaviruses fall into the range of viruses causing the common cold and also more serious respiratory diseases, specially, severe acute respiratory syndrome (SARS, 2002) and the Middle East respiratory syndrome (MERS, 2014), both of which are zoonotic diseases initially detected by the culture of viruses from patients with common colds in the mid-1960s (Li et al., 2019; Lu et al., 2020).

1.1. The host of SARS-CoV-2

As suggested by evolutionary assays by the use of ORF1a/1b, S, and N genes, SARS-CoV-2 is probably a new coronavirus with independent transfer from animals to humans (Chen et al., 2020a; Lai et al., 2020). A close identity was established between SARS-CoV-2 and two bat-SARS-like CoV (*bat-SL-CoV ZC45* and *bat-SL-CoV ZXC21*), particularly the sequence identities of the E gene in *bat-SL-CoV ZC45* (GenBank accession no. MG772933.1), *bat-SL-CoV ZXC21* (GenBank accession no. MG772934.1) existed 98.7% among 13 gene sites, but it was more apart from SARS-CoV (~79% similarity) and Middle East respiratory syndrome coronavirus (MERS-CoV) (~50% resemblance) (Jiang et al., 2020; Lu et al., 2020; Ren et al., 2020). An analysis of data obtained from the initial outbreak using a full-length genome study revealed that the SARS-CoV-2 sequence had a 79.5% sequence identity in common with SARS-CoV (Zhou et al., 2020). Sequence and phylogenetic analyses illustrated that SARS-CoV-2 had a longer spike protein, a dissimilar relative phylogeny of the complete RNA-reliant RNA polymerase gene, and a differing apparent genetic distance (Ahmed et al., 2020; Zhou et al., 2020), indicating that SARS-CoV-2 is a novel *betacoronavirus*, not the SARS-CoV (Ahmed et al., 2020). Nevertheless, SARS-CoV-2 and SARS have a common ancestor to the bat *HKU9-1 coronavirus* (Xu et al., 2020c). In Wuhan, SARS-CoV-2 is a member of the genera *beta coronavirus* and genome analysis indicates that SARS-CoV-2 has closer relations to *bat-SARS-like (SL)-ZC45* and *bat-SL-ZXC21* (Zhou et al., 2020). Even so, the dissimilarities are also possibly suggestive of additional intermediate hosts present between bats and humans. An investigational group observed that 70% of pangolins possessed β -CoV. A coronavirus isolate obtained from the pangolins contained a genome with a high similarity to that from SARS-CoV-2, and a genome sequence resemblance of 99% indicates that pangolin is likely the intermediate host of SARS-CoV-2 (Peng et al., 2020b).

1.2. Diseases caused by coronaviruses

Among the seven subtypes of coronaviruses capable of infecting humans, *beta-coronaviruses* can possibly induce serious disease and mortalities. The initial isolates of coronaviruses were obtained from fowls with respiratory disease (infective bronchitis) in the 1930s (Burrell et al., 2017). Until 2002, *human coronaviruses* (HCoVs) were linked only to mild respiratory tract disease, which were estimated to cause 15%–25% of the entire common colds (Geller et al., 2012). This underwent a change in 2002 upon identification of a *human coronavirus* as the causative agent of an emerging disease named SARS. The SARS spread was

taken under control, but a different new CoV isolate was collected from patients admitted to hospitals with severe respiratory disease in Saudi Arabia during 2014 (Fehr et al., 2017; Roosa et al., 2020; Zumla and Hui, 2014). Since the majority of affected patients were the inhabitants or travellers to Middle East countries, the newly detected disease was termed MERS and the causative *coronavirus* was named HCoV-MERS (Zumla and Hui, 2014). The transmission of most animal and human CoVs occurs via the faecal-oral path, and their primary proliferation place is in epithelial cells, in which virus reproduction induces local respiratory symptoms or diarrhoea (Burrell et al., 2017). Yet, CoVs is capable of causing acute to lethal disease. Besides seasonal flu, the recounted pathogens of pneumonia consist of *adenovirus*, *coronavirus 229E/NL63/OC43*, *human bocavirus*, *human metapneumovirus*, *parainfluenza virus 1/2/3*, *rhinovirus*, and *respiratory syncytial virus A/B* (Chou et al., 2019; Lee et al., 2019a, 2019b; Su et al., 2019). Additionally, these viruses are able to induce co-infection in the situation of public-derived bacterial pneumonia (Lai et al., 2020).

1.3. The transmission routes

Due to the relationship of the SARS-CoV-2 to the genus *betacoronavirus*, the diseases and transmission paths for the genus are depicted in Fig. 1.

The usual spread paths of the novel coronavirus are spreading directly (cough, sneeze, and droplet inhalation transmission) and contact transmission (contact with oral, nasal, and eye mucous membranes) (Otter et al., 2016; Peng et al., 2020b; Tellier et al., 2019). Eye contact can likely pave the ground effectively for the viral entry into the body (Peng et al., 2020a, 2020b; To et al., 2020). Despite an insignificant risk of contact with SARS-CoV-2 from the faeces of a patient, there are proofs indicating that SARS-CoV-2 is likely to result in enteric contagion and be found in faeces (Gu et al., 2020). About 2–10% of patients with established COVID-19 manifested with diarrhoea two reports indicated that SARS-CoV-2 viral RNA segments were detected in the stool of COVID-19 patients (Chen et al., 2020b; Huang et al., 2020; Wang et al., 2019, 2020a). Up to now, there is only one report on the culture of SARS-CoV-2 from a single stool sample (Holshue et al., 2020). Investigations observing viral nucleic acids in these patients, additional studies are required to discover the existence of viral elements and nucleic acid levels for demonstrating faecal-oral contagion (Xiao et al., 2020). Moreover, research has demonstrated that respiratory viruses are transmittable among individuals via contacting directly or indirectly, or coarse or small droplets, and SARS-CoV-2 is transmittable through direct or indirect salivary route (To et al., 2020). To be precise, aerosols denote particles suspending in a gas (Tellier et al., 2019). As shown by a study in 2010, the likelihood of transmitting influenza by aerosols could be reduced by improving ventilation design and prevention of generating aerosols (Wong et al., 2010). It is usually believed that transmitting by aerosols is plausible due to the high risk of cross-infection among physicians, nurses, and personnel (Hoseinzadeh et al., 2013, 2014, 2017). Research suggests that SARS-CoV-2 is probably air-transmitted via aerosols produce during therapeutic actions (Huang et al., 2020; van Doremalen et al., 2020; Wax and Christian, 2020). Remarkably, a case of 2019-nCoV infection reported in Germany reveals that SARS-CoV-2 may also transmitted via contacting with symptomless patients (Rothe et al., 2020). Even so, transmissions through aerosol and faecal-oral routes are those making people concerned, necessitating to be confirmed with additional studies. Fomite transmission, i.e. viral dissemination via a material, including a door handgrip, door-bell, or inhalator, also has a critical contribution to the virus spread (Kraay et al., 2018; WHO, 2020). Additionally, postnatal infection with SARS-CoV-2-borne pneumonia in neonates could be boosted by the contact levels of faecal infection, aerosol transmission, and contacting closely with the mother (Meng et al., 2020). Up to now, no investigations are available reporting COVID-19 transmission via exposure to blood (Zhou et al., 2020).

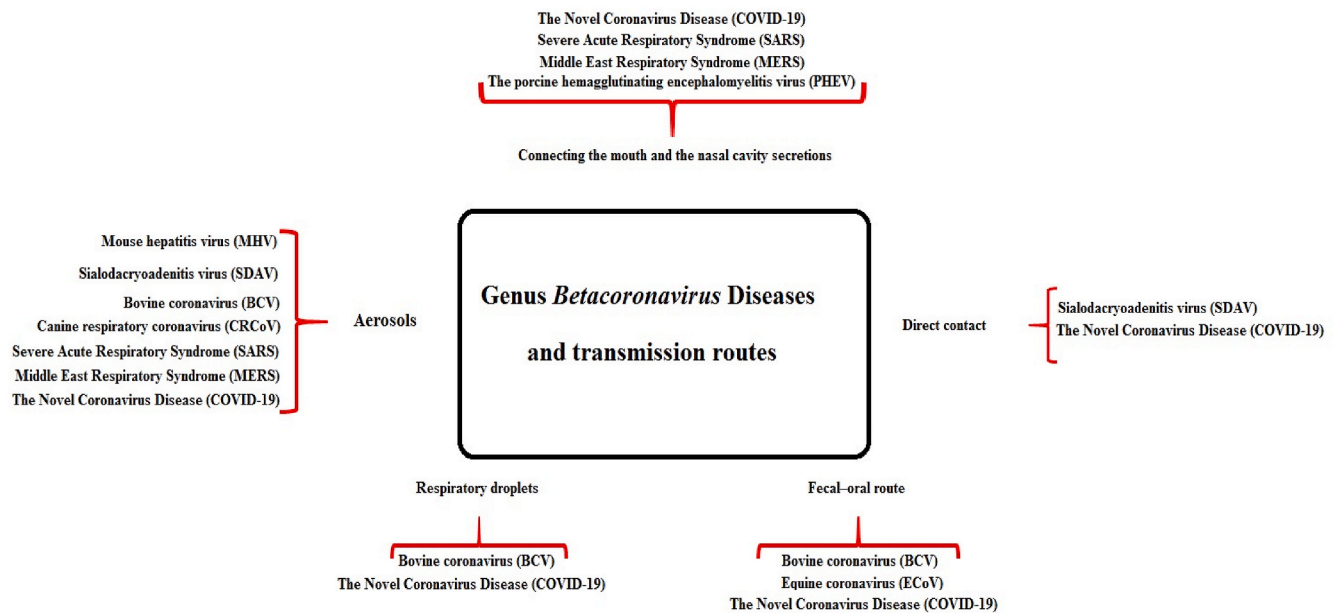


Fig. 1. Betacoronavirus diseases and transmission paths (the routes for COVID-19 under study and not yet confirmed) (Burrell et al., 2017; MacLachlan and Dubovi, 2017) for betacoronavirus except SARS-CoV-2.

2. Environmental transmission possibility

Besides public movement and person-to-person connections, viral contagion and survivability can be impacted by environmental players (e.g., influenza), but no research has yet studied the topic about this new pathogen. Absolute humidity, described as the water content in ambient air, is a robust determining factor in the environment for other viral spreads (Hoseinzadeh et al., 2017; Wolkoff, 2018; Yang and Marr, 2012). For instance, influenza viruses live lengthier on surfaces or in droplets in cold and dry air, which increases the possibility of succeeding contagion. It is, therefore, imperative to apprehend the impacts of environmental elements on the progressive outburst to endorse making decisions on controlling the disease, particularly in places in which there is a likely underestimation of the transmission risk, including in moist and warmer areas. This paper focuses on drinking water, sewage systems, solid waste, and ambient air as potential paths of contagion in the environment.

2.1. Transmission by drinking water

As a tiny infective agent, a virus proliferates only within the organismal living cells but not within contaminated cells, or in the event of contaminating cells. Viruses are found independently in the form of particles. Waterborne viruses are different with regard to their genome contents and capsid proteins, but such viruses have some common attributes making them of specific interest concerning the risk of disease epidemics related to drinking water infections (Gall et al., 2015). Water-spread viral pathogens, categorized in terms of moderate to high health importance by the WHO, consist of *adenovirus*, *astrovirus*, *hepatitis A* and *E viruses*, *rotavirus*, *norovirus*, and other *caliciviruses*, as well as *enteroviruses* including *coxsackieviruses* and *polioviruses* (Gall et al., 2015). Also, viruses of urine urinary excretion (e.g. *polyomaviruses* and *cytomegalovirus*) have the potential to be disseminated via water (Gall et al., 2015; Goetsch et al., 2018; WHO, 2011). Other viruses (e.g. *influenza* and *coronaviruses*) have been considered as organisms being transmittable via drinking water, but there is indecisive evidence (Gall et al., 2015; WHO, 2011). Besides, the COVID-19 virus has not been shown to be present in drinking water sources, and existing documentation indicate a low risk to water sources (WHO, 2020). Extreme

numbers of viruses are excreted in fecal matter even in a symptomless manner. For instance, up to 10^{11} *norovirus* particles are detectable per gram of faeces (Gall et al., 2015). Additionally, non-enfold viruses can survive in water for prolonged time-periods (Firquet et al., 2015; Pinon and Vialette, 2018). With taking account of these features, insufficient sanitization of faecal-infected drinking water facilitates epidemics of viral gastroenteritis from consumption. Remarkably, drinking water is also capable of transmitting viruses through inhalation or aspiration (e.g., water-bathing) or contacting with skin and eyes (e.g., swimming), which cause respiratory and ophthalmic contaminations (Pinon and Vialette, 2018). COVID-19 virus is an encased virus with a brittle exterior membrane. Encased viruses have in general less environmental stability with more susceptibility to such oxidants as chlorine (Pinon and Vialette, 2018). Although no documentation is available hitherto on the COVID-19 virus survivability in water or sewerage (WHO, 2020), the virus can probably be deactivated considerably quicker than non-encased human intestinal viruses (e.g., *adenovirus*, *norovirus*, *rotavirus*, and *hepatitis A*) with confirmed transmission ability through water. Heller et al. by considering the detection of SARS-CoV-2 in stools and sewage, proposed the faecal-oral transmission of COVID-19 from environment to human (Heller et al., 2020). According to other concordant investigations, the *human coronavirus* illustrated a 99.9% die-out from 2 days to 2 weeks at 23 °C and 25 °C, respectively, (WHO, 2020). The die-out is facilitated by heat, high or low pH, sunshine, and commonly used sanitizers (e.g., chlorine) (Pinon and Vialette, 2018).

2.1.1. Water treatment process against viruses

Commonly used water purification methods globally are physical removal of pathogenic agents by treating conventionally and inactivation of pathogenic agents via application of ultraviolet light or chemical oxidizing agents including chlorine, chloramines, ozone, and chlorine dioxide. Due to the tiny size of viruses (mostly with a diameter ranging between 5 and 400 nm), though some *paramyxoviruses* may be up to 14,000 nm long, traditional purification, including filtration (Fig. 2), is not effective in physical removal of viruses.

Fujioka et al. reported bacterial removal rates of 95.2 %-99.3% during full-scale water filtering (Fujioka et al., 2019). Thus, viral removals are not expectedly lower than bacterial ones (>0.2 µm in size) due to the size. The use of sanitizers is greatly dependent upon water

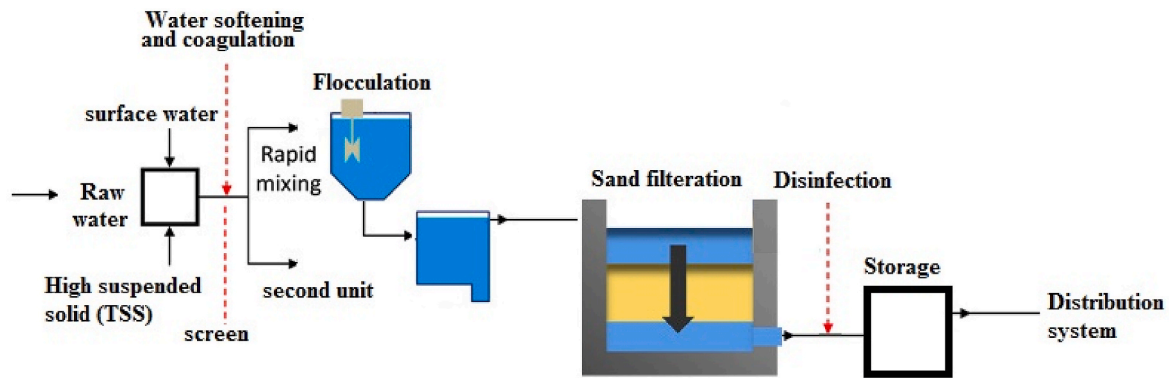


Fig. 2. Schematic flow diagram of a drinking water treatment plant.

chemistry and regional rules. Free chlorine (i.e., the sum of hypochlorous acid and hypochlorite ions produced by dissolving and hydrolysing chlorine gas in water) is the disinfection agent with the uppermost usage around the world (Hoseinzadeh, 2019). This powerful oxidizing agent inactivates the majority of viruses (Pinon and Vialette, 2018). Nonetheless, treatment with free chlorine may form regularized toxic disinfection by-products (DBPs). *Adenovirus* has susceptibility to deactivation with free chlorine with high resistance to deactivation using both monochloramine and UV light (Pinon and Vialette, 2018). Chlorine dioxide and ozone are also robust oxidizing agents, both of which can effectively control viruses (Gall et al., 2015). Irrespective of the sanitization utilized at a drinking water facility, the travel of purified water from the treatment plant to the tap can lead to cross-contamination all over the extent of water dissemination substructure because of cavitation and random depressurisation; thus, it is necessary to utilize alternative sanitizers in distribution systems. Free chlorine and monochloramine are the only two sanitizers that can maintain remains in the distribution system. In spite of more powerful disinfecting effect of free chlorine in terms of inactivating pathogens, monochloramine leaves residues with a higher stability in distribution systems hence both are useable. *Human coronavirus* can persist for only two days in dechlorinated tap water and hospital effluent at 20 °C (WHO, 2020). A main barricade to extensive use of water quality settings is that not a single sanitization technique is effectual towards the whole viruses. To have efficient concentrated sanitization, free chlorine remaining concentration must be ≥ 0.5 mg/L following a minimum of 30 min after contact time at pH < 8.0 (WHO, 2011). A chlorine residue should remain all over the distribution system.

2.1.2. Detection of viruses in drinking water

Water purification facilities routinely examine the existence of faecal coliforms in water sources, but they do not examine the existence of infective viruses due to either impossibility or unfeasibility of detecting or propagating infective viral particles cost-effectively and in a well-timed fashion. Whereas, viral proteins or genomes can be detected swiftly utilizing enzyme-linked immunosorbent assay (ELISA) or quantitative polymerase chain reaction (qPCR)-based technologies, respectively, the techniques cannot discriminate between infective and non-infective viral particles. Integrated cell culture-PCR (ICC-PCR) lowers the time demands of conventional plaque tests allowing infective viruses to proliferate in host cells, but a cell culture used in the technique is not even practical at water treatment facilities (Gall et al., 2015). Despite advancements obtained in collecting viruses from huge volumes of water, it remains as a fast way of detecting survivable viruses. Multiple existing barricades preclude detection of viruses in drinking water. To consider technologically, viral replication needs using tissue culture, a system requiring elevated time, workforce, skill, and costly apparatus. Additionally, some of these viruses are not culturable simply (*adenovirus* serotypes 40 and 41) or at all (*human norovirus*, *hepatitis A virus*) in culture media. Accordingly, conventional viral development tests

(plaque assays) are either inaccessible or highly time-taking rendering them impractical for water treatment facilities. Generally, the potential of SARS-CoV-2 transmission via water sources is low (Cahill and Morris, 2020) but more research need to study the viability of the virus and contamination of water source that can transmit the SARS-CoV-2 to humans.

2.2. Transmission by sewerage systems

The majority of faecal-oral-spread viruses have high resistance in the water milieu, in which they are likely to present improved survival in spite of the sanitization procedures usually utilized to treat drinking water and sewerage (Hoseinzadeh, 2019). The bulk of such viruses are non-encased viruses, such as *norovirus*, *enterovirus*, *Aichi virus*, *parvovirus*, *hepatitis A* and *E viruses* (HAV and HEV, respectively), *astrovirus*, *rotavirus*, and *adenovirus* (Ad) (Gall et al., 2015). These viruses are detectable in effluents as people excrete about 100g of stool daily, and 10^5 – 10^9 intestinal viral particles per gram of stool are released every day from a diseased person (Wigginton et al., 2012). Since 10^7 – 10^{13} viral particles are excreted daily from an diseased person (Wigginton et al., 2012), a possibly helpful tool is to analyse entering effluent to disclose the existence and quantification of defecated human pathogenic agents, thereby providing an estimate of the numbers of diseased individuals (La Rosa et al., 2020). If the virus is present, it will not indicate a relation between the viral sequences in effluent and those from patients of the same sampling time and area (Hellmér et al., 2014). Multiple virus recognition methods were designed for detecting *poliovirus* in effluent in accordance to the WHO polio removal program (Matrajt et al., 2018). Seven weekly combined untreated effluent samples from Ryaverket (a large wastewater treatment plant in Gothenburg) were analysed to determine the existence of seven differing intestinal viruses: *norovirus*, *astrovirus*, *rotavirus*, *adenovirus*, *Aichi virus*, *hepatitis A virus*, and *hepatitis E virus* (Hellmér et al., 2014). The virus is detectable prior to occurring an endemic, as reported by a research on *norovirus*, because the virus may be defecated in stool prior to the incidence of indications (Carter, 2005), which is 1–2 days for *norovirus* GII and 4–5 days for *astrovirus* (Lee et al., 2013) whereas, the defecation duration is lengthier for *hepatitis A* and *E viruses*, occurring for about 7 weeks (Richardson et al., 2001). No documentation is available hitherto about the COVID-19 virus transmission through sewer systems, with or without wastewater treatment. Besides, no proof can be found regarding the counteraction with SARS by effluent and wastewater treatment staffs. Genetically compared effluent viruses with those from clinical periodic cases and endemics can put forward a model to understand the epidemiology of intestinal viral pathogens in the population. Thus, surveillance of effluent regularly for such viruses can offer an ahead-of-time alarm of a potential forthcoming outbreaks. The encumbrance of contagion in a special population can also be estimated by the use of typing as an instrument. Based on a research work in Helsinki, Finland, *poliovirus* was detectable in effluent

if only 1 in 10,000 residents defecated the virus (Hovi et al., 2001). Even so, it may identify genomes from non-infective viral particles as well (Hellmér et al., 2014). A study conducted in Spain on six wastewater treatment plants in an area with lowest COVID-19 prevalence revealed that two secondary water sample were polluted. Comparison of the data with data of confirmed COVID-19 cases at municipally level revealed that people were shedding SARS-CoV-2 RNA in their faeces even before the first cases were reported by local or national authorities (Randazzo et al., 2020). In case of identifying only viral segments, it will continue providing an acceptable denotation about the types of viruses circulating in the community and those infecting individuals not seeking therapeutic care. It is recommendable to utilize this procedure for forecasting COVID-19 in the public (Randazzo et al., 2020). As a fraction of a merged population health strategy, effluent passed through sewer systems should undergo ideal treatment in decently established and properly controlled consolidated wastewater treatment utilities. Finally, it is possible to consider a decontamination phase if available sewer water purification plants are not subjected to optimisation to for viral removal. It is essential to follow the best applications to protect the professional health of labours at sanitization treatment installations. Labours need to put on suitable personal protective equipment (PPE), including protecting outer wear, gloves, boots, goggles, or face shields, masks, regularly hand hygiene, and avoiding the touch of the eyes, nose, and mouth with dirty hands.

2.3. Transmission by healthcare waste

Leftovers created during health care activities possess great potentiality for contamination, and its insufficient managing practices exposes health care personnel (e.g., physicians, nurses, and laboratory staffs), waste carriers, and patients in hospitals to health risks both directly and indirectly (Amsalu et al., 2016). Health-care waste (HCW) carries a vast array of pathogenic agents. Professional risk associated with COVID-19 contact is a main concerning issue, particularly in developing countries in which people do not rigorously follow a protocol for waste treatment. Documentations are absent about human exposure directly and insecurely throughout treatment of HCW resulting in the COVID-19 endemic. A report by the WHO (2004) indicate that urban and solid HCW have identical contents of microorganisms, and that 2% of blood-stained waste tested positively for *hepatitis viruses*, *poliovirus*, and *echovirus* identified in the defiled diapers of household waste (WHO, 2004). Pathogenic viruses, such as *NoVs* and *hepatitis B virus*, have been identified in human tissue waste (WHO, 2004) (WHO, 2004). There is little information suggesting that viral RNA was detectable in the plasmas or sera of COVID-19 infected people (Chang et al., 2020). Even so, observations of researches conducted experimentally demonstrated that some viruses (e.g. respiratory syncytial virus) have partial survivability under settings possibly found in a variety of wastes (Park et al., 2009), and that viral loads are usually low. Thus, an essential issue is that labours follow group and personal protecting procedures including gloves, respiratory masks, glasses, and overalls that are previously supplied in all working environment security plans (Carducci et al., 2013). Since minor amounts of information are available from individual plants, it is necessary to perform more inclusive surveillance examinations. All HCW created throughout the care of COVID-19 patients need to be gathered securely in engineered vessels and bags, handled, and then carefully discarded and/or treated, preferably in situ (WHO, 2020). In case of moving wastes ex situ, its treatment and destruction place and mechanism must be understood critically. Prior to treatment of HCW, it is necessary to wear suitable PPE (boots, apron, long-sleeved gown, thick gloves, mask, and goggles), and perform appropriate hand sanitation after elimination. To gain more data, the reader is referred to the WHO Safe Management of Wastes from Health-Care Activities guidance (WHO, 2014). An emphasis is placed on necessary trainings in preventing contagion, in particular concerning waste treatment and removal. It is imperative to follow the most acceptable operations to

safely manage HCW, such as assignment of accountability and providing adequate human and material resources for safe removal of waste.

2.4. Transmission by inanimate surfaces

Non-encased viruses (e.g. *coxsackieviruses*, *rotavirus*, or *poliovirus*) can be viable for prolonged times on surfaces whereas, encased viruses, such as *H1N1* and *human coronaviruses*, are still infective on surfaces after few days. A variety of environmental situations and parameters including heat, moisture, pH, and surface type influence the viability of desiccated viruses (Firquet et al., 2015). The ingredients of the media can affect the viability of viruses as well. Environmental surfaces probably contribute to the spread of hospital-derived viral contaminations (Ryu et al., 2020; Wang et al., 2020b; Ye et al., 2020). At times of endemics in health care services, samples taken from surfaces discovered SARS-CoV nucleic acids on surfaces and non-living substances (Casanova et al., 2010), suggesting that surfaces could be sources of virus spread for SARS-CoV-2. Environmental viral tests identify them in protein-laden media (such as serum) and protein-poor media, including water. Hydrophobic substances prevent the spread of droplets, and their non-porous feature improves viral survivability (Firquet et al., 2015). Survival duration of the COVID-19 causing virus on surfaces is not certainly known, but it apparently behaves the same as other *coronaviruses*. Recently, reviewing the viability of *human coronaviruses* on surfaces revealed high survivability, in the range of 2 h–9 days (WHO, 2020). Casanova et al. (2010) demonstrated that infective SARS-CoV survived for a period of 28 days at 4 °C, and the deactivation was lowermost at 20% relative humidity (RH). Faster deactivation occurred at 20 °C than at 4 °C at all moisture levels; the viruses were viable for 5–28 days, and a low RH led to the slowest deactivation. Deactivation of viruses was more rapid at 40 °C than at 20 °C (Casanova et al., 2010). The association between deactivation and RH was not monotone, and low RH (20%) and high RH (80%) resulted in better persistence or a higher protecting impact than at mild RH (50%) (Casanova et al., 2010). Deactivation is dividable into two steps of initial represented by water loss because of evaporating free water from the surface (Zhao et al., 2012), exposing viruses to a liquid-air interface, which leads to viral deactivation. The discrepancy can be attributed to rehydration, noticeably inactivating non-lipid viruses, including *poliovirus* (Zhao et al., 2012). Even so, iterative drying cannot influence non-encased viruses. Encased viruses showed more sensitivity than non-encased ones in the second stage of viral survival, which began once the liquid could not be seen on the lids anymore. Probably, the viruses survived for days or even weeks on dry hydrophobic surfaces (Firquet et al., 2015; WHO, 2011). Media constituents and component contents had a clear contribution upon exposure of virus suspensions to desiccation (Firquet et al., 2015; WHO, 2011). *Coronaviruses* are encased, positive-sense, and single-stranded RNA viruses. They typically show vulnerability to acid-pH, basic-pH, and heat, but apparently have more stability at 4 °C. The lipid bilayer casing of these viruses has rather sensitivity to dryness, heat, and deteratives; thus, their sterilisation is simpler than non-encased viruses (Firquet et al., 2015; WHO, 2011). Several studies report that a succeeding elevation in solute concentrations in droplets could modulate the survivability of viruses against desiccation. Because the contents of media has a contribution the survivability of viruses subjected to desiccation, further investigations are necessary on the viral viability in natural media (clinical or environmental), rather than determined media. Contamination of surfaces in hospital was investigated by et al. (Razzini et al., 2020) which reported that 35% of COVID-19 patient's ward, 50% undressing room and no clean areas were contaminated with COVID-19. The most contaminated surfaces were hand sanitizer dispensers, medical equipment, medical equipment touch screens, and shelves for medical equipment, bedrails and door handles, respectively. Kampf et al. presented evidence that efficient inactivation of *coronaviruses* would be possible by surface sanitizers containing 62%–71% ethanol, 0.5% hydrogen peroxide, or 0.1% sodium hypochlorite for

approx. 1 min, but other biocides, e.g. 0.05%-0.2% benzalkonium chloride or 0.02% chlorhexidine digluconate, presented lower effectiveness (Kampf et al., 2020).

2.5. Transmission by ambient air

Various pathogens are present in the air and have the potential to be spread over extended spaces (Hoseinzadeh et al., 2017), which comprise *influenza virus*, *SARS virus*, *Mycobacterium tuberculosis*, foot and mouth disease, and several other ones. The majority of patients with COVID-19 were affected severely by acute respiratory infection, including fever, cough, and shortness of breath, leading to death of most of such people. Given that respiratory spread and the spread of survivable virus placed on the surfaces is possible, the viability of air-transmitted viral aerosolised should be importantly investigated in ambient air (Morawska and Cao, 2020). Felipe Falcão Sobral et al. found the negative association between air temperature and COVID-19 cases. In addition, they found rainfall as an important climate factor in SARS-CoV-2 transmission (Sobral et al., 2020). Furthermore, the impact of weather on COVID-19 pandemic in Turkey assessed by Mehmet Şahin. He found inverse correlation between wind speed, air humidity and temperature as the weather parameters with COVID-19 cases (Şahin, 2020). The similar results obtained by Wu et al. (2020); Yao et al. (2020) as well. A study (Xu et al., 2020a) showed a significant relation (Poisson regression model) between air quality index (AQI) that determine the ambient air quality with COVID-19 cases for Chinese cities. A recent extensive research has studied the persistence of diverse strains of air-conveyed *influenza virus* and (Pyankov et al., 2012) observed that infectious virus were present following 90 min of aerosolisation. Air temperature and humidity, microbial resistance to external physical and biological stresses, and solar intensity have been introduced to be some significant factors of air that affect the persistence and distribution of microorganisms (Pyankov et al., 2012). Cases may be transmitted continually and grow (exponentially) and rapidly within a spectrum of humidity varying from cold and dry provinces in China, including Jilin and Heilongjiang, to hot regions, such as Guangxi, and Singapore (Luo et al., 2020). A conclusion can be drawn that climatic changes (i.e., rises of temperature and humidity upon reaching spring and summer months in the Northern Hemisphere) will not essentially result in reductions in case frequencies with no execution of widespread public health interventions. Carducci et al. (2013) reported that totally 30% (12/40) of air samples and 13.5% (5/37) of surface samples obtained from the solid waste removal location were positive for the virus. Pyankov et al. (2018) investigated the persistence of aerosolised *MERS-CoV* in ambient air. They detected a rather high count of viral particles at 25 °C, as opposed to influenza strains, with over 63% of living particles staying in the air following 60 min of aerosolisation. The inactivation was much more effectual at 38 °C, with merely 4.7% live viruses observed after completing the 60 min run. The reported findings illustrate that the strain is capable of surviving during prolonged times and has the potential of spreading because of respiratory contagion even under tropical and arid weather situations relating to the Middle Eastern area, the place of origin for this virus. Doremalen et al. found identical persistence of *HCoV-19* and *SARS-CoV* under the settings examined experimentally (van Doremalen et al., 2020). According to their findings, *HCoV-19* retained its viability in aerosols in the course of their experimental period (3 h) with a reduced in infective titre between $10^{3.5}$ and $10^{2.7}$ TCID₅₀/L, to the same as that noticed for *SARS-CoV*, from $10^{4.3}$ to $10^{3.5}$ TCID₅₀/mL. The above surveys suggest that aerosol and fomite spread of *HCoV-19* is probable because the virus is able to retain its viability in aerosols for several hours and on surfaces for a number of days. Ogen studied the nitrogen dioxide (NO₂) levels of ambient air as a causal factor to coronavirus (COVID-19) fatality in Turkey. The results showed as the NO₂ can lead to lung inflammatory and may be increasing susceptibility to air pollution, so chronic airway disease due to long-term exposure to air pollutants can increase the COVID-19 fatality (Ogen,

2020). In regard with impact of particulate matter, Mehmood et al. stated that although the short- and long-term exposure with PM_{2.5} resulted in higher incidence of lethality of COVID-19, however, estimation of PM_{2.5} incidence is required to experimental and epidemiological studies (Mehmood et al., 2020). Correia et al. addressed indoor and environmental transmission of the SARS-CoV-2 through Heating, Ventilation and Air Conditioning Systems (HVAC) (Correia et al., 2020). By controlling this transmission routs the pandemic control of COVID-19 can be more effective. However, Faridi et al. (2020) did not found viral RNA in air samples taken from patient room with confirmed COVID-19 at distance of 2–5 m from the beds.

3. New ways of SARS-CoV-2 transmission that maybe facilitated by angiotensin-converting enzyme 2 (ACE2)

The commonly observed indications of COVID-19 at disease incidence are fever, fatigue, dry cough, myalgia, and dyspnoea (Wang et al., 2020a). Few patients could also present headache, dizziness, abdominal pain, diarrhoea, nausea, and vomiting (Wang et al., 2020a). Illness incidence may result in continuous respiratory inability because of alveolar harm and even demise. The expression and dissemination of angiotensin-converting enzyme 2 (ACE2) in the human body can be indicative of the possible entrance of SARS-CoV-2 that play as a receptor (Bosso et al., 2020). High ACE2 expression was detected in type II alveolar cells (AT2) of the lung, oesophagus, upper and stratified epithelial cells, absorptive enterocytes from the ileum and colon, cholangiocytes, myocardial cells, kidney proximal tubule cells, and bladder urothelial cells (Xu et al., 2020b; Zhang et al., 2020). These observations revealed that the organs with high ACE2-expressing cells would be regarded as those with high potential risk for SARS-CoV-2 contamination (Xu et al., 2020b). Xu et al. modelled the spike protein of the receptor for SARS-CoV-2 and found that ACE2 might be the receptor for this viral strain (Xu et al., 2020b). Likewise, ACE2 is a receptor for SARS-nCoV and NL63 (Cao et al., 2020). Based on their model, the coupling strength is greater between SARS-CoV-2 and ACE2 than the limit needed for viral contamination, though it is poorer than that between SARS-nCoV and ACE2. Zhou et al. carried out viral contamination assays and detected that ACE2 would be necessary for entering SARS-CoV-2 into HeLa cells (Zhao et al., 2020). Such information demonstrate that ACE2 may be the receptor for SARS-CoV-2. Zhao et al. examined normal lung tissue cells of eight healthy persons and noticed that the only Asian donor presented over five times the ACE2 expressing cell ratio as white and African American donors. These findings suggest that the Asian population is likely to be increasingly susceptible, though such a conclusion requires further documentation. Hao Xu et al. (2020b) presented evidence of ACE2 expression on the mucosa of the oral cavity. It is interesting that this receptor contained a high bulk of epithelial cells of the tongue, which have justified the basic mode of action by which the oral cavity has the potential of a high risk for SARS-CoV-2 contamination sensitivity; such discoveries also present proof of forthcoming preventing policies in dental clinical practice (Peng et al., 2020b) and everyday life. The observations also imply that ACE2 has a significant contribution to cellular entrance (Choi et al., 2020); hence, ACE2-expressing cells can serve as target cells, and have susceptibility to SARS-CoV-2 contamination. It may be the SARS-CoV-2 virus uses the ACE2 receptor to improvement access the cell interior that the virus can do replication there easily (Li et al., 2020).

4. Conclusion

The accessible data about environmental spread paths for SARS-CoV-2 that have not been earlier investigated to date were reviewed here, indicating the availability of scarce or unavailable documentation concerning thereof. Despite the unconfirmed contagion of SARS-CoV-2 via drinking water, sewer systems, and ambient air revealed by accessible guidelines, reviewed data strengthen the suspicion by highlighting the

robust potential of environmental spread via these paths hence necessitating extra studies. Moreover, the ACE2 that was recognised as a cellular doorway for SARS-CoV-2 entrance the cells by some studies, which may be useable for identifying novel paths of SARS-CoV-2 contagion.

Data availability statement

All datasets generated for this study are included in the article/supplementary material.

Author contributions

EH did conception and design, acquisition of data, or analysis and interpretation of data, and writing. MT did conception and design, interpretation of data, and drafting the article or revising it critically for important intellectual content. MF, SJ, FM and HH did revising it critically for important intellectual content and final approval of the version to be published.

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Declaration of competing interest

We declare there is no competing interest.

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