VIRUS SURVIVAL IN THE ENVIRONMENT WITH SPECIAL ATTENTION TO SURVIVAL IN SEWAGE DROPLETS AND OTHER ENVIRONMENTAL MEDIA OF FECAL OR RESPIRATORY ORIGIN
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INTRODUCTION

Viruses are submicroscopic, obligate intracellular parasites that are biologically active only within their host. They range in size from about 20 to 300 nm and consist of a nucleic acid genome surrounded by a protein coat; some have an additional outer layer composed of lipoprotein, called the envelope. Viruses are shed from infected hosts into the environment and while outside of their cellular hosts and in the environment they have the potential to survive, persist and be transported by various routes to again reach other susceptible hosts. Not all viruses released into the environment are, however, successful in surviving and reaching new susceptible hosts. Viruses can be considered biochemical complexes that behave as colloidal particles in the environment, where they can interact with various environmental media and matrices, be subjected to various transport processes as well as sequestration and degradation processes. Therefore, viruses present in environmental media and matrices can be transported, diluted sequestered, inactivated and degraded before they ever reach a susceptible host to infect.

The extent to which viral pathogens of humans and animals persist in the environment to reach other hosts is of considerable public health interest and concern. Viruses can be transmitted by a variety of routes, including direct and indirect contact, vector transmission, and vehicle transmission. For many human and animal viruses, vehicle transmission includes respiratory transmission by droplets and aerosols, and fecal-oral transmission via water, food, and fomites, such as environmental surfaces. Consequently, for viruses transmitted by the respiratory and fecal-oral routes, transport and persistence in environment is directly related to the potential for and risk of transmission, host exposure, infection and disease. Viral persistence, survival and transport can vary greatly with virus type and environmental conditions. This review will summarize the factors influencing virus persistence, survival and transport in the environment, especially for viruses transmitted by the fecal-oral and respiratory routes and with special consideration of the SARS virus and other Coronaviruses. Data will be presented for environmental persistence of a range of virus types in different environmental media and matrices, including air, water, waste and surfaces, and under different environmental conditions influencing virus survival, persistence and transport.

ENTERIC VIRUSES AND OTHER FECALLY SHED VIRUSES OF HUMANS AND ANIMALS OF PUBLIC HEALTH CONCERN FOR POSSIBLE FECAL TRANSMISSION

A wide variety of different viruses, representing nearly all of the families of animal viruses, can be present in human animal fecal wastes and urine. Especially important are a variety of non-enveloped enteric and respiratory viruses. These include adenoviruses, astroviruses, caliciviruses, papovaviruses, parvoviruses, picornaviruses (enteroviruses and hepatitis A virus), and other non-enveloped viruses that can be shed in faeces (and in some cases urine) from infected individuals and can be present in fecal wastes and fecally contaminated environmental samples. Also fecally shed (or shed from the urinary tract) and present in faeces and fecally contaminated environmental samples are a variety of enveloped viruses, including arenaviruses, bunyaviruses, coronaviruses, filoviruses, flaviviruses, herpesviruses, orthomyxoviruses, paramyxoviruses, pestiviruses, poxviruses, retroviruses, rhabdoviruses, togaviruses and these viruses can be in fecal wastes of human an animals. Some of the main viruses and virus groups of importance in human fecal wastes and in animal manures (faeces and urine) are shown in Table 1. Many of the viruses listed here are important pathogens of their human and animal hosts, although some of them do not always cause severe illness or high mortality rates. The impact on health of many of the human viruses belonging to these families is reasonably well understood and documented. However, the significance to human health of many of the non-human animal viruses belonging to these virus families is less well understood and remains uncertain or unknown for many of them. It is notable, however, that zoonotic viruses of humans continue to be discovered or appear to re-emerge as important human pathogens. Recent among these are the SARS virus (a coronavirus), monkeypox virus (a poxvirus), and hepatitis E virus, an unclassified virus recently implicated in human hepatitis illness from consumption of raw venison. Many of the viruses found in faeces and combined fecal and urine wastes are non-enveloped, relatively persistent in the environment and resistant to treatment and control processes. Enveloped viruses also can be present in human fecal wastes and urine and in animal manures (urine and faeces) at high concentrations. Furthermore, many of these enveloped viruses also can persist for considerable periods of time in these wastes and in contaminated environmental media. Therefore, the factors that influence virus survival in the environment are of considerable interest because they play a role in the extent to which the viruses shed into environmental media will again pose risks of further human exposure, infection and illness.

FACTORS AFFECTING VIRUS SURVIVAL IN THE ENVIRONMENT

Introduction

Numerous physical, chemical and biological factors influence virus persistence in the environment. In all environmental media or matrices, virus type and composition is an important factor in survival, transport and persistence. A number of the important physical, chemical and biological factors influencing virus survival in the environment are summarized in Table 1. Some of the primary factors affecting the transport, survival and fate of viruses in liquid environmental matrices or media are temperature, ionic strength, chemical constituents, microbial antagonism, the sorption status of the virus and the type of virus. Of the chemical constituents in liquid or semi-solid (faeces, human night soil, biosolids, animal manures, etc.) in environmental matrices, the amount and type of organic matter and specific antiviral chemicals, such as ammonia at elevated pH levels (pH > 8.5), play a role in virus survival. Of the physical factors influencing virus survival in liquid media, temperature, sunlight and virus association with solids are among the most important factors influencing survival.

Viruses can occur in the airborne state in two major forms: as droplets that are relatively large (>20 μmeters in diameter) and largely liquid and as aerosols that are relatively small (5 μmeters or less) that may be composed of either mostly liquid or solid materials. Particles in the range of about 5 to 20 μmeters also can remain airborne for extended periods of time, especially if they are mostly composed of water, which tends to evaporate, thereby causing the particles to become even smaller in size over time. An important difference in physical properties of these airborne virus-containing particles is that the droplets are so large that they settle out or sediment within short distances (meters) from their source while the aerosol particles can remain airborne for extended periods of time and travel long distances. For virus-containing aerosols (virus bioaerosols), the most important factors affecting virus survival are temperature, relative humidity (RH), moisture content of the aerosol particles, composition of the suspending medium, sunlight exposure, air quality (especially poorly characterized air pollutants, such as "open air factor"), and type of virus (enveloped or hydrophobic versus non-enveloped and hydrophilic). Aerosol particle size also plays a role in viral persistence in aerosols and in the deposition of the particles in the respiratory tract.

On surfaces the most important factors that affect virus survival are the type of virus and surface, relative humidity, moisture content (water activity), temperature, composition of the suspending medium, light exposure and presence of antiviral chemical or biological agents.

Virus Family, Type and Composition

Viruses are diverse in their sizes, shapes, surface properties and internal composition. Table 2 summarizes the physical and genetic characteristics of some of the most environmentally relevant or important human and animal virus families. As previously noted, viruses are essentially composed of a nucleic acid surrounded by a protein coat, and depending on the virus family (or type) they may or may not have a lipoprotein envelope. For the non-enveloped viruses, the capsid is the outermost layer that is responsible for virus attachment to cells and is in contact with the environment. For enveloped viruses the envelope is in contact with the environment and the virus-specific glycoproteins within the envelope are responsible for virus attachment to host cells. For both enveloped and non-enveloped viruses, the surface chemistry of the virion is complex. The surface can be considered a mosaic consisting of both hydrophilic and hydrophobic domains with a variety of both charged and uncharged functional groups capable of interacting with solvents (such as water), other solutes, surfaces (solids) and chemical ligands. Morphologically, viruses are either icosahedral, helical, ovoid, bullet-shaped or pleomorphic. Virus morphology is a function of the capsid morphology and the presence or absence of an envelope. As previously indicated the physical size of viruses range from a low of about 18-25nm for parvoviruses to about 350nm for poxviruses.

The SARS virus and other coronaviruses, which are of particular interest in this report, are enveloped, have a helical nucleocapsid, are circular to pleomorphic in shape and about 120-140 nm in diameter. The surface projections of SARS virus and other coronaviruses are distinctive, club–shaped peplomers composed of different types of proteins. The surface projections are surrounded by a prominent fringe; they are spaced widely apart and evenly dispersed. The surface projections comprise S-proteins responsible for attachment to cells, hemagglutination and membrane fusion and hemagglutinin esterase proteins (HE), which form short projections (in some species). The nucleocapsids of coronaviruses are helical and either straight or bent; 9 nm in diameter, or 11–13 nm in diameter, depending on the specific virus. The molecular mass (Mr) of coronavirions is 400 x 10^6. The virions have a buoyant density in CsCl of 1.23–1.24 g cm^{-3} and in sucrose of 1.15–1.19 g cm^{-3}. The sedimentation coefficient is 300–500 S_{20w}. Under *in vitro* conditions some coronaviruses are stable in an acid environment of pH 3. Like many other

viruses, some coronaviruses are more stable in presence of Mg^{++}. For example, heat inactivation of mouse hepatitis virus is reduced in the presence of Mg^{++}.

Viral Capsids: Viral capsids are typically either helical (filamentous) or icosahedral (round). Viral capsids are made up of one or more structural proteins and consequently have electrically charged surfaces due to the ionic functional groups (primarily carboxyl, amino, sulfhydryl, etc.) of the acidic and basic amino acids comprising the virion proteins. The iso-electric point of a virus (or any particle) refers to the point at which there is no net surface charge on the particle {Gerba, 1984 #556}. This does not mean that there is no charge on the surface, rather that the ionic functional groups of positive and negative charge or pockets of them are charge-balanced across the surface as a whole. The iso-electric points vary greatly even between strains of a particular virus type, indicating that the degree to which they will sorb to different materials (such as soil particles and other surfaces) will also vary {Gerba, 1984 #556}. Additionally, a virus may have more than one iso-electric point. An example of this is poliovirus in which a conformational change in the protein capsid at a particular pH exposes different amino acid functional groups to the surface, thus leading to the existence of the viruses with one of two iso-electric points {Mandel, 1971 #496}. Most viruses have iso-electric points in the acid pH region, below the neutral pH of 7.0.

The pH of fecal matter, most domestic sewage, many natural water and most drinking water supplies is generally in the range of neutral pH and therefore, above the iso-electric points of most viruses. Consequently, viruses are generally thought to be negatively charged in faeces, sewage, most natural waters, and most water supplies. As previously noted, the electrical potential or charge on the surface of a virus is dependent on the relative presence of surface amine, R-functional, carboxyl groups and sulfhydryl groups in the amino acids of the virus capsid proteins. The gain and loss of protons is controlled by the individual dissociation constants of the termini and functional groups of the amino acids (which range from 1.8 to 2.4 for the α-COOH groups, 8.8 to 10.8 for the $\alpha\text{-}NH_3^+$ groups, and 3.9 to about 13 for the R-functional groups) {Murray, 1996 #566}. Viruses are generally considered to be hydrophilic, however regions of hydrophobicity on virus capsids have been shown to exist {Wait, 1983 #564}. This is due to the amino acid composition and the conformational folding of capsid proteins. Some amino acids, such as alanine, leucine and tyrosine, are more hydrophobic than others, like arginine, glycine, lysine, and threonine.

Viral Envelopes and Glycoprotein spikes: Some virus types, such as the alphaviruses and orthomyxoviruses, acquire a lipoprotein envelope as they bud through the host plasma membrane. Other viruses, like the coronaviruses acquire a lipoprotein envelope by budding through the membrane of various host cell organelles (in the case of coronaviruses the endoplasmic reticulum). Additionally, most enveloped viruses, such as the coronaviruses, have additional surface proteins or glycoproteins that function in their attachment to host cells and in other interactions. The surfaces of the enveloped viruses are more hydrophobic than those of the non-enveloped viruses because the lipoprotein bilayer is highly hydrophobic. However, the virus-specific glycoproteins embedded in the lipid bilayer of the virion enveloped are hydrophilic to varying degrees, and they contribute to the hydrophilic properties of the virion surface. As noted above these proteins and glycoproteins have a key role to play in virion attachment and other interactions with host cell surfaces (cellular receptors), antibodies and environmental surfaces.

Viral Nucleic Acids: The type of viral nucleic acid is characteristic of a virus family and may be either RNA or DNA, single- or double stranded, linear or circular, in a single piece of made up of discrete segments. Single stranded RNA or DNA genomes may be either positive sense or negative sense. A positive sense genome is one which has the same orientation as mRNA.

Further, viral genomes may be either linear or circular. The size of viral genomes ranges from ~5kb for parvoviruses to ~248kb for cytomegaloviruses. The genomes of coronaviruses are not segmented and consist of a single molecule of linear positive-sense single-stranded RNA. The complete genomes are 25000–33000 nucleotides long. The 5'–end of coronavirus genomes has a methylated nucleotide cap, and the 3'–terminus has a poly (A) tract.

Effects of virus type on survival in the environment: Considerable differences have been observed in the stability and inactivation rates of viruses in faeces, sewage, biosolids, animal manures, natural waters and other environmental media. Differences in survival have been observed not only among viruses of different families and genera, but also among viruses of the same family, genus and even among similar types or strains of virus {Denis, 1975 #504;Block, 1983 #485}. Such differences between virus types have been observed for survival in aerosols and on surfaces. A few examples of such differences in different environmental media are reported below.

In a variety of water samples, including fresh and saline waters, poliovirus has been demonstrated to be more persistent than either herpes simplex virus or HIV. HIV was reduced by 1 $\log_{10}$ after less than two days in tap water, sewage and seawater at 16^{o}C. Herpes was reduced by 1 $\log_{10}$ in less than a day and a half in all water samples. Depending on sample, it took 23-59 days for poliovirus to be reduced by 1 $\log_{10}$ {Slade, 1989 #335}. In another study with higher incubation temperatures (25^{o}C), HIV was reduced by 1 $\log_{10}$ after only two hours and by 3 $\log_{10}$ after eight hours, whereas polio persisted for 24 hours {Moore, 1993 #334}. However, a third study by Casson *et al.* (1992) reported that cell-free HIV was reduced only by about 1- to 2-$\log_{10}$ after 48 hours in water and wastewater (primary and secondary effluent) at 25^{o}C.

Thermal sensitivity of pseudorabies virus has been shown to be strain dependent, and thermal sensitivities have been used to group individual strains of virus {Platt, 1979 #409;Golais, 1975 #410;Bartha, 1969 #411}.

During aerosol studies on influenza survival, avian and equine strains were found to be more resistant to inactivation than human or swine strains {Mitchell, 1972 #305}. Detection of infectious human and swine strains was found to be less than or equal to 15 hours, whereas equine and avian strains were detectable for 20-30 hours and 24-36 hours, respectively. Furthermore, differences in the stability of aerosolized influenza at intermediate relative humidity have been reported in several studies. Several studies report a transition between low and high inactivation rates at intermediate humidity {Harper, 1963 #435;Harper, 1961 #322;Hemmes, 1960 #423}. Other studies report the greatest inactivation at intermediate humidity {Hood, 1963 #436;Shechmeister, 1950 #437}. These differences in reported inactivation at intermediate humidity have been attributed possibly to the hosts used for passage of the viruses and the differences in the host matrices comprising the viruses being studied {Schaffer, 1976 #296}. Influenza was raised in bovine, human, and chicken cell cultures, as well as embryonated eggs. Viruses aerosolized in cell culture fluids were most stable at low relative humidity, minimally stable at intermediate humidity, and moderately stable at high relative humidity. On the other hand after, some preparations of viruses aerosolized in allantoic fluid were equally stable at intermediate and high relative humidity.

Other studies have reported shifts or variations in the survival patterns of different viruses with multiple passages in tissue cultures {Hearn jr., 1965 #438;Hinshaw, 1976 #295}. The sensitivity of Yellow Fever virus to inactivation at intermediate humidity has been shown to increase with multiple passage in HeLa cell culture {Hearn jr., 1965 #438}. Schaffer *et al*. suggest that this phenomenon may be due to a difference in structural lipids or other related structural factors

between the propagating host cells {Schaffer, 1976 #296}. Differences in the suspending medium may also explain discrepancies between virus survival studies {Benbough, 1971 #306}.

In studies examining the persistence of representatives of several families and genera of viruses (adenovirus, poliovirus, coxsackievirus, vaccinia virus, and herpes simplex type 1) on inanimate surfaces, survival was found to vary with virus type {Mahl, 1975 #366}. Adenoviruses were most persistent, surviving for greater than 12 weeks at low humidity. With the exception of herpes simplex virus at high humidity and temperature, the coxsackieviruses studied were generally found to be the least persistent, persisting for less than three weeks regardless of temperature and humidity.

Virus Detection in Human and Animal Wastes and in Environmental Samples

Although enteric and respiratory viruses have been studied for their survival, transport and fate in human fecal and respiratory wastes, animal fecal wastes (manures), waste treatment and management systems, and environmental media, much of what has been done has relied on spiking samples with high concentrations of test viruses to follow their survival, transport and fate (Turner *et al*., 1999a, 1999; 1999c). Some research has been done to develop, evaluate and apply standardized and consistent methods to recover, detect and quantify seeded and naturally occurring human and animal enteric viruses in untreated and treated fecal wastes, animal manures and fecally contaminated environmental samples, including aerosolized viruses (virus bioaerosols) (Bosch, 1998; Cole and Cook, 1998; Douwes *et al*., 2003; Gerba and Goyal, 1992; Metcalf *et al*., 1995; Straub and Chandler, 2003; Straub *et al*., 1993; Theron and Cloete, 2002). Viruses have been recovered from faeces, animal manures, treated wastes, foods and environmental samples containing high concentrations of viruses by simple extraction or elution procedures, and sometimes, no attempt has been made to further concentrate or purify the viruses (Deng and Cliver, 1992). However, in many environmental media, adequate virus detection requires that the viruses be concentrated from the samples and separated and purified from sample constituents that can interfere with virus detection. Viruses in treated animal manures generally are recovered and concentrated by the same methods used to recover human enteric viruses in municipal biosolids. Sometimes viruses in sample extracts either after concentration or with no concentration, are inoculated directly into cell cultures for virus detection and assay by infectivity. For example, viruses in swine waste slurry extracts have been treated with fluorocarbon (solvent) extraction to improve recoveries and reduce cytotoxicity prior to direct inoculation into cell cultures (Turner *et al*., 1999). In many cases viruses are concentrated and purified before inoculation into cell culture for virus assay or before virus detection by molecular methods targeting the viral nucleic acid. Some of the sample processing steps used to recover, concentrate and purify non-enveloped viruses, such as solvent and detergent treatments and exposure to pH extremes, are, however, not applicable to enveloped viruses because they will inactivate them.

Detecting viruses by detecting their nucleic acids, with or without nucleic acid amplification, requires further steps to extract the viral nucleic acids and to remove interfering sample materials that can interfere with viral nucleic acid amplification and detection. Detecting viruses in environmental samples by detecting their nucleic acids has the limitation of not being able to conclusively determine if the detected virus nucleic acids are from infectious viruses. Viruses of inactivated viruses can be detected by these nucleic acid methods (Sobsey *et al*., 1998). To overcome this problem, nucleic acid detection is being used in conjunction with virus propagation in cell cultures in order to detect the nucleic acids of only those viruses that are infectious and successfully propagate in cell cultures (Chung *et al*., 1996; Reynolds *et al*., 1996). Another approach to increase the likelihood of detecting intact and potentially infectious viruses in cell

cultures is to pre-treated the virions with proteolytic enzymes and nucleases prior to nucleic acid extraction, amplification and detection, thereby eliminating the detection of free nucleic acids or nucleic acids associated with damaged, inactivated virions (Nuanualsuwan and Cliver, 2002).

Many different of methods have been used to recover and detect viruses in fecal wastes of humans and animals and in different fecally contaminated environmental samples. In addition, a number of different methods also have been developed to recover, detect and characterize enteric and respiratory viruses and other microbes in aerosols and other airborne particles (Alvarez *et al*., 1995; Cole and Cook, 1998; Douwes *et al*., 2003). Furthermore, there is lack of performance data on their recovery and detection efficiency, especially for the many different families, genera and specific types of viruses that can be present in faeces, respiratory secretions, manures and other fecally contaminated materials and environmental samples. The variability and often unknown or poorly characterized and quantified performance characteristics (e.g., recovery and detection efficiencies) of the methods is a major source of uncertainty and variability in the available data on virus occurrence, persistence, survival and fate in fecal material and fecally contaminated environmental samples and it confounds and limits the ability to understand, interpret and compare these data.

Bacteriophages of fecal bacteria present in human and animal faeces are considered candidate viral indicators of the presence of viruses in such wastes and in fecally contaminated environmental samples, and the reductions of viruses by treatment processes, management systems in environmental media (Grabow, 2001; IAWPRC Study Group, 1991; Leclerc *et al*., 2000). Of the bacteriophages present in animal faeces and manures, the bacteriophages that have received the most attention as indicator viruses are the somatic and male-specific coliphages. These are the same types of bacterial viruses that have received similar attention as enteric virus indicators in human and animal fecal waste, sewage and biosolids. The extent to which coliphages are reliable indicators of the presence, survival, transport and fate of human and animal enteric viruses in faeces, manures, and in environmental media impacted by fecal wastes is relevant to both animal and human fecal waste sources. Currently, bacteriophages of fecal waste origin are being used to detect, characterize and quantify the presence, survival, transport and fate of human and animal viral pathogens in fecally contaminated environmental media. However, the extent to which they can reliably and quantitatively predict the presence, survival, transport and fate of different viral pathogens in a variety of environmental media under a range of environmental conditions is uncertain at this time. This is an active area of environmental virology research that can possibly contribute to better understanding of the behavior of newly discovered and emerging pathogens in fecally contaminated environmental media.

Factors Affecting Virus Survival in Liquid and Semi-solid Media (Faeces, Animal Manure, etc.)

Temperature: Temperature is one of the most important factors determining virus survival and persistence. Viral persistence may be affected by elevated temperatures through several mechanisms, including protein denaturation, RNA damage, and influence on microbial or enzymatic activity {Melnick, 1980 #391;Deng, 1995 #237;Dimmock, 1967 #325}. Early studies indicated that the primary reason for viral inactivation at high temperatures is damage to virion proteins, while at low temperatures it is damage to viral RNA. However, it was assumed that damage to both protein and RNA occurred at all temperatures (Dimmock, 1967). Other studies have documented temperature-related structural changes in the poliovirus capsids in soil, leading to inactivation of the virus through dissociation of the viral capsid and degradation of the viral

RNA {Yeager, 1979 #375;Yeager, 1979 #376}. As temperature increases, virus survival typically decreases {Yates, 1990 #551;Hurst, 1980 #552}.

A variety of enteric viruses have been examined for the effects of temperature on their survival in a range of media or environmental matrices. The effects of temperature on the persistence of both non-enveloped and enveloped viruses has been examined in liquid and dried animal wastes under a number of different conditions. Even enveloped viruses can be relatively persistent in animal manure. One of the more environmentally persistent animal viruses is Aujeszky's disease virus, also called pseudorabies virus, which is a herpesvirus with a wide animal host range. The survival of the enveloped Aujeszky's disease virus in pig slurry was investigated during anaerobic storage at 5, 20, 35, 40, 45, 50 and 55 ^{o}C using 100-ml laboratory models simulating the conditions in slurry tanks during winter and summer seasons and during anaerobic digestion in batch reactors. These environmental conditions are likely to be worst cases for virus survival, because they are intended to promote anaerobic treatment and degradation of the animal waste as part of a waste management system. As expected, virus inactivation rates increased with increasing temperature. Virus was inactivated at 5 and 20 ^{o}C in 15 weeks and 2 weeks, respectively. At 35 ^{o}C (mesophilic conditions) the virus was inactivated in 5 hours and at 55 ^{o}C (thermophilic conditions) no virus could be detected after 10 minutes. Although Aujeszky's disease virus was capable of persisting for considerable periods (days) in swine fecal wastes at mesophilic and lower temperatures, it was inactivated rapidly at a thermophilic temperature of 55^{o}C (Botner, 1991). However, it is noteworthy that this enveloped virus and member of the Herpesviridae family was quite persistent in an infectious form for periods of hours to weeks at ambient environmental temperatures.

In another study, the persistence of a bovine enterovirus (BEV) (ECBO-virus strain LCR-4) and a field isolate of Aujeszky's disease virus was determined in liquid cattle manure stored at temperatures of 4 and 20^{o}C for up to 26 weeks (Biermann *et al*., 1990). On the day of inoculation each sample had a titer of 5 $\log_{10}$ ID50/ml. Aujeszky's disease virus inactivation was >5 $\log_{10}$ after 16 weeks at 20^{o}C but only 3.25 $\log_{10}$ after 26 weeks at 4^{o}C. Enterovirus inactivation at 20^{o}C was only 2 $\log_{10}$ after 26 weeks. Therefore, as expected, the non-enveloped (BEV) virus was more persistent than the enveloped virus (Aujeszky's disease virus).

The survival of the bacterium *Salmonella anatum*, pseudorabies virus (PRV), and porcine reproductive and respiratory syndrome virus (PRRSV) in swine slurry was studied at temperatures of 4, 25, and 37 ^{o}C) and at pH 4.0, 7.0, and 10.0 (Ajariyakhajorn *et al*., 1997). These microbes survived longest at 4^{o}C and pH 7, with *S. anatum* surviving longest at 56 days and pseudorabies and PRRS viruses inactivated by 8 and 14 days, respectively. These results suggest that some viruses are not more persistent than bacteria under certain environmental conditions in animal manures.

Most studies examining virus inactivation or persistence have done so under laboratory conditions, which raises concerns that they may not be representative of conditions in the field. Therefore, the persistence of five animal viruses, representing picorna-, rota-, parvo-, adeno-, and herpesviruses, and the coliphage f2 was determined in the field by exposing the viruses to different animal wastes using a filter sandwich technique (Pesaro *et al*., 1995). This method attempts to model the natural state of virus adsorption onto or incorporation into suspended solids, which may prolong virus survival. The "filter sandwiches" consist of viruses adsorbed to nylon filters that are sandwiched between layers of polycarbonate (PC) filters. The outer PC filters of the sandwich are either porous (15 nm in diameter) to allow passage of solutes or poreless (solid) to allow no passage of solutes. Using both types of PC filters it was possible to differentiate between overall virus inactivation (from temperature and antiviral chemicals diffusing into the

sandwich) and the effect of veridical agents that act through temperature effects only (solid PC membranes). The virus-filter sandwich set-ups can be placed in manure storage or treatment systems in the field in order to study virus survival under the realistic conditions of actual field operations. Depending on ambient temperature, pH, and type of animal waste, time in days required for a 90% virus titer reduction varied widely, ranging from less than 1 week for herpesvirus to more than 6 months for rotavirus. Virus inactivation was faster in liquid cattle manure, a mixture of urine and water (pH > 8.0), than in semiliquid wastes that consisted of mixtures of faeces, urine, water, and bedding materials (pH < 8.0). It was concluded that unidentified virucidal agents that permeated poreless PC membranes contributed substantially to virus inactivation. Also, substances that protect rotavirus and possibly other viruses from inactivation also appear to be present in animal wastes. These apparently protective substances were not specifically identified in this study, but they are hypothesized to be similar to the previously identified detergents that thermally stabilized rotaviruses and other viruses in sludge (Ward and Ashley, 1978; 1980). Overall, the results of the study indicate that both enveloped and non-enveloped viruses contained in animal fecal waste (manure) may persist for prolonged periods of time if stored under nonaerated conditions. However, it was further concluded that at ambient temperatures (<20 ^{o}C), thermal effects were only indirectly related to viral inactivation and that other factors were more directly contributing to virus inactivation{Pesaro, 1995 #236}.

A study on the survival of several types of enteroviruses at various temperatures found that while differences were detected between different enteroviruses, all viruses persisted greater at lower temperatures (-20C and 1C) than at higher temperatures 22C {Hurst, 1988 #331}. Certain virus types within a single virus family, such as HAV (genus Hepatovirus within the Picornaviridae family), are more heat stable than other genera of this family and consequently they survive longer in the environment. However even relatively thermo-stable viruses, such as HAV, have shown greater reductions at temperatures of 30°C than at 20°C or 10°C {Nasser, 1993 #329}. Nasser *et al*. (1993) showed that poliovirus 1 and HAV are more rapidly inactivated in a variety of aqueous samples (groundwater, PBS, wastewater, and sterilized wastewater) at 30°C as compared to 10°C or 20°C {Nasser, 1993 #329}. A similar temperature effect on inactivation was found for poliovirus, HAV, and rotavirus incubated at 25C and 4C in seawater {Chung, 1993 #333}. The persistence of Coxsackie A9 in stream water, brackish water, and sea water has also been demonstrated to be dependent on temperature {Nasser, 2003 #224}. Additionally, poliovirus and hepatitis A virus have been shown to be more persistent at 4ºC than at room temperature in mineral water. Both viruses were very persistent at 4ºC with minimal reductions after 1 year {Biziagos, 1988 #330}. Rotavirus has been shown to be most stable at 4ºC, less stable at 20ºC, and least stable at 37ºC {Moe, 1982 #277}. Foot and Mouth Disease Virus (FMDV), a picornavirus, can persist for >185 days in frozen, snow-covered soil and infect exposed cattle (Bartley et al., 2002)

Temperatures above 55ºC have been demonstrated to rapidly inactivate poliovirus (≤30 minutes) in both growth media and sewage {Clarke, 1961 #392}. At lower temperatures, virus survival in a variety of environmental media is much longer. For example, poliovirus survived for up to 66 days at 4ºC in digested sludge {Wellings, 1976 #377}. In septage, poliovirus inactivation also has been shown to be dependent on temperature, with inactivation at 55ºC being more rapid (>5 $\log_{10}$ within 15 minutes) than inactivation at 50ºC (<2 $\log_{10}$ in 90 minutes) {Stramer, 1984 #266}.

Canine coronavirus survives well frozen at –20^{o}C, but does not survive well above 4 ^{o}C{Tennant, 1994 #238}. Presence of fecal material greatly reduced survival between +20C to -70C. Mouse hepatitis virus 2 (MHV2) was not completely inactivated during heating to 56 ^{o}C for 30 minutes{Hirano, 1978 #573}. MHV2 also survived heating to 50 ^{o}C for 15 minutes, and 37 ^{o}C

for 60 minutes in the presence of 1M magnesium (as $MGCl_2$ or $MgSO_4$) at pH 3.0 to 9.0. Reductions of MHV2 were 2 log_{10} after 30 days at 4 ^oC and 2.6 log_{10} after 24 hours at 37 ^oC.

Temperature has also been shown to be an important factor in the survival of non-enteric viruses, like human immunodeficiency virus (HIV) and hepatitis B virus (HBV). Results are somewhat inconsistent in the magnitude of HIV persistence in environmental media as a function of temperature, but all studies show increased inactivation at higher temperatures. Studies examining the survival of HIV in phosphate buffer solution containing 2% fetal bovine serum indicate that HIV is stable (<0.5 log_{10} after 6 months) at temperatures of -20ºC or below {Tjotta, 1991 #369}. Additionally, HIV was show to have half-lives of 9 days, 30 hours, and 24 hours at temperatures of 4ºC, 20ºC, and 37ºC, respectively {Spire, 1985 #412;Barre-Sinoussi, 1985 #413;Tjotta, 1991 #369}. Some previous studies reported that 30 minutes treatment at temperatures above 56ºC was sufficient to fully inactivate HIV {Harada, 1985 #415;Einarsson, 1989 #414;Spire, 1985 #412}. In contrast, Tjotta et al. found an appreciable fraction of HIV survival after 30 minutes at 56-64ºC {Tjotta, 1991 #369}. Other studies have also determined longer exposure or higher temperatures are necessary to inactivate HIV {Yoshikura, 1989 #418;Hilfenhaus, 1987 #416;Prince, 1986 #417}.

Other viruses have also been shown to be inactivated by exposure to elevated temperatures. Ebola virus has been demonstrated to be thermally inactivated by treatment for 1 hour at 58-60ºC, and by treatment at 75ºC for 30 minutes {Bray, 1999 #482;Chepurnov, 1995 #481}. Hepatitis B virus can be inactivated by treatment at 98ºC for 1 minute {Bond, 1981 #341}. However, prolonged treatment (10 hours) at 60ºC will not achieve complete inactivation {Shikata, 1978 #473}.

Lower temperatures generally enhance virus survival and most viruses can be stored frozen to maintain their infectivity for long periods of time{Chepurnov, 1995 #481;Block, 1978 #567}. However, freezing temperatures may cause structural damage and thus decrease survival {Hurst, 1980 #552;Gerba, 1984 #554}. Freezing and thawing was found to reduce HIV titers, by 0.2 to 0.3 log_{10} {Tjotta, 1991 #369}. Enteroviruses also survive long term storage when frozen to ultracold conditions. However, it has been shown that up to 40% of poliovirus may be inactivated by freezing to -26ºC and then thawing to room temperature {Block, 1978 #567}. The reported differences in the effects of freezing and thawing on the virus type (e.g., poliovirus) may be due to type or strain differences of the virus, the temperatures to which they are frozen, the rate of freezing and thawing and the medium in which they are frozen.

In summary, virus persistence or survival in stored or conveyed liquid and semi-solid human and animal wastes can lead to environmental contamination if the wastes are not contained and properly treated and disposed of. Viruses in human and animal fecal wastes and fecally contaminated water generally are inactivated more rapidly at higher temperatures. At moderate and low temperatures (and intermediate pH levels of 5-9), viruses can persist for considerable periods of time that may range from hours to days to weeks to even months in the case of the most persistent viruses. The rate of inactivation and the extent of survival varies, depending on virus type, temperature, suspending medium and other environmental conditions.

pH & Salt Content: Both direct and indirect effects on virus survival may be caused by pH. Directly, pH may effect the conformation of the capsid proteins {Fujioka, 1975 #497;Mandel, 1971 #496}. Indirectly, pH will influence virus adsorption and elution from other particles {Bitton, 1980 #498}. Poliovirus is stable at a wide range of pH (3.8 to 8.5) for weeks {Pollard, 1949 #465}. Studies indicate that the reduction of Rotavirus (strain Wa) in water is a function of the pH of the water {Pancorbo, 1987 #398}. The pH of tissue culture medium was determined to

be an important factor in survival of HIV. At 4ºC the half life of HIV at pH 7.1 was determined to be 9 days, while at pH 7.35 the half-life was determined to be 15 hours {Tjotta, 1991 #369}. The effect of pH on the inactivation of herpes simplex virus in tissue culture medium appears to be influenced by temperature {Lancz, 1979 #471}. At ultracold temperatures, no effect of pH was shown. However, increasing pH from 6.3 to 7.8 at 6º C had a negative effect on herpes virus survival. The magnitude of the anti-viral pH effect increased with increasing temperature.

Ionic strength or salinity has variable effects on virus survival. Studies have reported enhanced virus inactivation in saline solution compared to distilled water {Dimmock, 1967 #325; Salo, 1976 #503}. Other studies have reported a protective effect from increased ionic strength {Cords, 1977 #501;Rao, 1984 #543}. Divalent cations may have a stabilizing effect on non-enveloped as well as enveloped viruses {Yana Moto, 1962 #500;Wallis, 1962 #499}. In buffered solution, the presence of 2M $MgCl_2$ has been demonstrated to have a protective effect for poliovirus against thermal inactivation at temperatures above 40ºC. The effect was not as pronounced for rhinovirus {Dimmock, 1967 #325;Wallis, 1961 #405;Melnick, 1963 #404;Majer, 1965 #403}. Still other studies report no significant effect of salinity on survival of certain viruses {Lo, 1976 #502;Dinchere, 1992 #542}. Differences in the effect of salinity on survival have also been reported between virus types {Lo, 1976 #502}. However, minor differences in salinity of seawater from different geographical regions have been found to correlate poorly with differences in virus survival {Fujioka, 1980 #285}. Perhaps other differences in seawater composition besides salinity (such as enzymes and microbial communities) have greater effects on virus inactivation.

The survival of herpesvirus of turkeys (HVT; the Marek's disease vaccine virus) in commercial diluents has been examined for the effects of medium composition, osmolality, temperature and rate of temperature change {Colwell, 1975 #430}. Osmolality of vaccine diluents were shown to become important in virus survival above the normal range of 288 to 372 mOsm/kg, and appeared to be more important than pH. Cell-associated Marek's disease (MD) vaccine was suspended at dilutions normally used for vaccination in seven commercially available diluents and in tryptose phosphate broth. The stability of diluted vaccines was determined by assay in cell cultures subjected to 0 to 37^{o} C for 0 to 90 minutes. Optimum holding temperatures for MD vaccine virus survival varied with the specific diluents employed. Some diluents afforded greatest survival when dilution was at 0^{o} C and held at 0^{o} C, while others performed best when dilution was at 25^{o} C followed by cooling and holding at 0^{o} C. Diluents which allowed greatest survival when tested at 37^{o} C also performed well under other temperature regimes. Additives to the suspending medium that increased the osmolality to 745 mOsm/kg and higher markedly reduced vaccine virus survival. The adverse effects of high osmotic pressure were accentuated by extended holding time, elevated incubation temperature, and physical manipulations, including mechanical mixing or expressing through a syringe and needle.

The pH and salt content of water and other environmental media also can indirectly influence virus survival by influencing the extent of virus adsorption to other particles and to other surfaces. At pH levels typically found in natural waters, viral adsorption is thought to be governed by phenomena involving the diffuse double layer that comprises the charged surface of the virion. Being charged and at least somewhat hydrophilic, viruses have a surface potential or electrical charge as do other colloidal particles in aqueous media. The cation concentration and pH of the medium both affect virus adsorption by determining the thickness of the diffuse layer, thus promoting or inhibiting van der Waals attraction forces {Gerba, 1984 #556}. The adsorption of viruses generally increases as pH decreases and as cation concentrations, especially the concentrations of multivalent cations, increase {Gerba, 1984 #556}. Salt concentrations have been demonstrated to play a factor in the adsorption of poliovirus type1 and echovirus type 7 to

solid waste components {Sobsey, 1975 #297}. Viruses were shown to adsorb efficiently in the presence of high concentrations of dissolved salts, while in the absence of dissolved salts efficient viral adsorption was not observed.

Microbial Antagonism: The effects of natural biological activity on the survival of viruses is well documented in surface waters, fecal wastes and soils. Viruses consistently demonstrate less persistence in natural waters and other environmental media and matrices compared to the same media that have been sterilized or pasteurized. Studies generally have shown that viruses are more persistent in autoclaved or filter-sterilized waters, than in their unsterilized counterparts {Magnusson, 1967 #514;Lycke, 1965 #521;Matossian, 1967 #515;Shuval, 1971 #517;Hermann, 1974 #519;O'Brien, 1977 #520}. Bacteria and other microbial predators (ciliated grazers, amoebae, rotifers, fungi, and aerobic bacteria) play a role in viral inactivation, either through production of metabolites that adversely affect the virus particles, or by direct use of the virion as a nutrient source. Several organisms, including members of the genera *Vibrio*, *Flavobacterium*, and *Klebsiella*, have been isolated from natural waters that demonstrate antiviral properties {Katayoshi, 1983 #548;Girones, 1989 #544;Katznelson, 1978 #518;Kelly, 1961 #507;Mitchell, 1971 #508;Gunderson, 1967 #506}. However, a few studies have found no difference between the inactivation rates in sterilized and unsterilized seawater {Leogrande, 1969 #505;Metcalf, 1967 #516}. In those studies, other factors may have outweighed the antiviral effect of the biological activity of the water, or perhaps the enzymatic activity of the water was not effected by autoclaving. Enzymatic activity of natural waters, in particular proteolytic enzymes, have also been implicated in the inactivation of viruses {Sobsey, 1973 #509;Ehresmann, 1977 #510;Cliver, 1972 #511;Hermann, 1973 #512;Grabow, 1975 #513}.

The effect of microbial antagonism on virus survival may also be observed in wastewater and other environmental media, such as soil. In studies on the inactivation of hepatitis A virus in mixed septic tank effluent and animal waste, HAV was shown to be inactivated much more rapidly in raw mixed waste than in autoclaved wastes, suggesting that microbial activity is a factor in viral inactivation {Deng, 1995 #237}. Similarly, HAV was more readily inactivated in mixed wastes that had been filtered to remove bacteria than in autoclaved wastes, suggesting that microbial metabolites or other thermo-labile substances may play a role in viral inactivation {Deng, 1995 #237}. In marine and estuarine waters, studies have demonstrated antiviral activity against enteroviruses (poliovirus type 1, coxsackievirus B4, and echovirus 7) associated with the natural microbiological community {Fujioka, 1980 #285}. Microbial activity in water has also been shown to negatively affect the persistence of rotaviruses in raw and treated fresh waters {Raphael, 1985 #373}. Another study demonstrated that survival of poliovirus in sandy loam was negatively affected by aerobic soil organisms {Hurst, 1988 #331}.

The survival of Poliovirus and Reovirus was studied in sterile and non-sterile soil suspensions {Sobsey, 1980 #283}. Both viruses survived better in sterile suspensions than in their corresponding non-sterile counterparts. Poliovirus survival in sterile soil suspensions ranged from 18 to 167 days, with a mean of 95 days, while in non-sterile soil suspensions poliovirus survival was 9 to 80 days with a mean of 42 days. Reovirus survived 8 to 257 days in sterile soil suspensions (mean of 123 days), while in non-sterile soil suspensions survival was 9 to 110 days **(**mean of 60 days). The survival of tobacco mosaic virus has also been shown to be affected by the presence of aerobic microorganisms in the soil {Cheo, 1980 #534}. Katayoshi *et al*., (1983) have isolated a phototropic bacterium (*Rhodopseudomonas capsulata*) capable of producing a substance with a virucidal effect on coliphages and polioviruses.

The degree to which microbial antagonism is a factor in virus inactivation in groundwater is still unclear. Some studies suggest that a microbially active groundwater environment consistently

decreases virus survival, while others suggest no clear trend {Sobsey, 1980 #283;Yates, 1990 #551}. However, it is clear that soil bacteria produce proteolytic and other enzymes that appear to decrease virus survival {Yates, 1990 #551}. Other studies also have shown a greater viral persistence in sterile samples than non-sterilized samples, indicating a role of antagonistic microflora in the survival of viruses in water {LaBelle, 1980 #332}. Viruses differ in their susceptibility to inactivation by these enzymes, however, and this makes it difficult to compare the results of groundwater studies in which different viruses were used.

Enzymatic degradation and other chemical effects: The role of enzymes from microbes and possibly other sources in water, wastes, soils and other media on virus inactivation has been studied for several decades. Many bacteria produce proteolytic enzymes inactivating enteric viruses, including certain bacterial species that are prevalent members of the mesophilic flora of water, for example *B. subtilis* (Cliver and Herrmann, 1972). Laboratory experiments have demonstrated that cell-free filtrates of bacterial cultures have an antiviral activity and that such an activity is inhibited by protease inhibitors. Their results suggested that enzymes present in the cell-free filtrates are responsible for viral inactivation (Cliver and Herrmann, 1972; Deng and Cliver, 1992; 1995). It also appears that viruses may serve as a nutrient source for bacteria, as indicated by the recovery in bacterial cells of radioactively labeled viral capsid proteins (98). A comprehensive study was carried out to determine the properties of agents responsible for loss of virus infectivity in mixed-liquor suspended solids (MLSS) of activated sludge. Initial experiments revealed that model enteric viruses (poliovirus-1 and rotavirus SA-11) were irreversibly inactivated in MLSS and released their RNA genomes. Enteric viruses belonging to other genera (echovirus-12, coxsackievirus A13, reovirus-3) were also shown to lose infectivity in MLSS. Although the virucidal activity decreased at reduced temperatures, MLSS still retained significant activity at 4^{o} C. The virucidal agents in MLSS were stable for months at 4^{o} C, but their activity decreased approximately 50% during four days of aeration at 26^{o} C. Primary effluent, the nutrient source for activated sludge, also contained virucidal activity. It was concluded that microorganisms are responsible for this antiviral activity because treatments that resulted in the inactivation or removal of microorganisms also caused a loss of virucidal activity. It was further suggested that the virucidal components of microorganisms are either short-lived or active only while bound to the organisms themselves. In a study on the survival of five animal viruses in animal waste, it was suggested that at ambient environmental temperatures, volatile and non-volatile microbial metabolites, high ambient pH, and other factors are important contributing factors to the inactivation of viruses (Pesaro *et al*., 1995). Therefore, it appears that chemically mediated microbial antagonism decreases virus survival due to the action of enzymes, the release of metabolic products and the use of viruses and their components as a nutrient source {Cliver, 1972 #419;Lipson, 1985 #420}.

Adsorption and its influence on virus persistence and survival. Several types of forces may play a part in viral adsorption. These may include ionic attractions, covalent reactions with certain chemicals, hydrogen bonding, hydrophobic interactions, double layer interactions, and van der Waals attractions. The current theory of virus adsorption suggests that the most important of these forces are the double layer interactions and van der Waals attractions {Gerba, 1984 #556}.

The diffuse double layer theorized independently by Gouy and Chapman, and later by Debye and Huckel in the early 1900s is based on the observation that individual colloidal particles may carry a charge, although the colloidal system (particles and suspending medium) remains neutral. The theory was refined by Verwey and Overbeek to describe the interactions between two particles. This theory was demonstrated to apply to poliovirus by Murray and Parks in 1978 {Murray, 1980 #557}. The theory essentially states that as a particle develops a surface charge, a discrete layer of counter ions forms compactly around the particle. This layer is referred to as the Stern Layer.

The ions in this layer only partially counter balance the surface charge on the particle. There is a second more diffuse layer of counter ions that also forms around that particle which also helps to neutralize the surface charge. This layer is called the Gouy Layer. The thickness of the Gouy Layer determines how close individual particles may come to each other. Under conditions where the Gouy Layer is sufficiently compact, two particles (or a particle and a surface) may approach each other close enough for their van der Waals forces to hold them together. Van der Waals Forces are electrical attractions between the instantaneous dipole moments of two molecules {Stumm, 1992 #558}.

Hydrophobic interactions may also play an important part in virus adsorption {Bales, 1991 #559}. Viral surfaces may have both hydrophilic and hydrophobic domains. The hydrophobicity of a viral particle depends on how these domains are balanced and oriented on the surface, which for non-enveloped viruses is directly related to the amino acid composition of the capsid proteins. Consequently, some viral particles are more hydrophobic than others. For solubilization of a hydrophobic substance in water to occur a thermodynamic barrier must be overcome. The hydrogen bonding network within the water must be reorganized to maximize the hydrogen bonding in water around the substance. Consequently, hydrophobic substances are forced together, not by a mutual attraction but because it is thermodynamically unfavorable for them to be solubilized in water. The presence of chaotropic agents will allow hydrophobic compounds to be solubilized in water. Chaotropes are small compounds that disrupt the thermodynamic barrier by decreasing the ordered structure of water. Anti-chaotropes, on the other hand, increase the ordered structure of hydrogen bonding in water, thus increasing the barrier preventing hydrophobic compound solubilization {Gerba, 1984 #556}.

Chemical binding by covalent or ionic attractions is another possible attachment mechanism. Chemical binding is often associated with virus inactivation. Chemical binding of viruses may cause conformational changes in the virus capsid that render the virus inactive or perhaps open the capsid releasing the more easily degradable nucleic acid. {Yeager, 1979 #560}. Adsorption of microbes is generally a weak attachment and microbes have been demonstrated to be eluted or desorbed by low ionic strength rainwater {Reneau, 1989 #561}. Several factors may affect the adsorption of viruses to other particles or surfaces. The pH of the system probably is the most important factor influencing virus adsorption. Ionic strength and cation content also influence virus adsorption. The isoelectric points of the viruses and the other particles or surfaces, and the strength of the charge on the virus and these other surfaces (zeta potential) are also factors affecting virus adsorption.

Viruses adsorb rapidly and efficiently to particulate matter. In wastewater, particle association of viruses ranges from 16 to 100 percent, depending on virus type and the type and level of treatment {Bitton, 1984 #267;Pancorbo, 1981 #399;Wellings, 1976 #377}. One gram of activated sludge particles may adsorb greater than 10^8 poliovirus particles {Nakajima, 2003 #401}.

Adsorption: Adsorption status has been shown to greatly affect virus survival. Generally, a virus that is adsorbed has a better chance of survival than one that is free {Gerba, 1975 #555}. Adsorption of a viral particle may protect it against disruption by steric effects {Liew, 1980 #495}. Adsorption to activated sludge particles has been shown to increase the persistence of poliovirus infectivity over a period of 28 days, and also provide a protective effect against elevated temperatures {Nakajima, 2003 #401}. In small batch virus survival studies, Reovirus was found to survive longer in solutions containing soil than in soil-free samples {Sobsey, 1980 #283}. Adsorption to marine sediments has been shown to increase the persistence of poliovirus and echovirus (from 1 hour to >4 days and 1.4 days to 6 days, for 99% inactivation respectively),

although differences were observed between locations {LaBelle, 1980 #332}. Reovirus 3 and bacteriophages T1 and T7 were demonstrated to persist longer when adsorbed to clay materials {Stotzky, 1980 #533}. For poliovirus, HAV, rotavirus, and bacterial viruses suspended in marine water samples with and without sediment, all viruses, except rotavirus, were more rapidly inactivated in samples with sediment than without {Chung, 1993 #333}.

Aggregation or clumping of viruses has been shown to affect virus inactivation kinetics by chemical disinfection. In water, virions on the inside of poliovirus aggregates have been show to be protected from disinfection by bromine {Young, 1977 #294}.

However, a protective affect is not always derived from adsorption. In the case of viral adsorption to certain metallic oxides, the sorption may be so great that the virus is effectively inactivated {Gerba, 1984 #556}.

Organic Matter Content: The presence of organic matter has been shown to improve the persistence of viruses. Suspension of sandy soils in septic liquor led to greater virus survival than suspension in groundwater or riverwater {Yeager, 1979 #376}. In a recent study, the adsorption of poliovirus to activated sludge particles was shown to be protective. After a one hour incubation at 55ºC, 60 percent of particle associated poliovirus was infectious, whereas only 0.6 percent of unattached viruses were infectious {Nakajima, 2003 #401}. Hepatitis A virus has been demonstrated to be more persistent in mineral water in the presence of protein {Biziagos, 1988 #330}. The association of poliovirus with organic solids in fecal suspensions during chlorination has been shown to increase viral survival. The most significant protective effect of organic material is by increasing the chlorine demand of the solution. However, lesser protection is also afforded by occluding the virus from the disinfectant {Hejkal, 1979 #289}. Hepatitis A virus inactivation by free chlorine was much greater for dispersed viruses than for viruses that were cell-associated (Sobsey *et al*., 1991). Greater survival of cell-associated HAV was attributed to protection of the viruses within the cell by cellular organic matter and cellular structures such as membranes.

Antiviral chemicals: The effect of natural and synthetic detergents on bacteriophages survival has recently been investigated {Chattopadhyay, 2002 #226}. In these studies, a non-ionic detergent (Triton X-100) had a minimal negative effect on the survival of either bacteriophages T2 or ΦX-174 except at very high concentrations. Both anionic and cationic detergents had greater negative impacts on the survival of the viruses. Natural organic matter and a biosurfactant were also demonstrated to negatively impact the survival of the bacteriophages. Anionic detergents and ammonia have been demonstrated to have rapid virucidal activity at specific pH conditions. Below pH 6 and above pH 8, sodium dodecyl sulfate, an anionic detergent, may have potent virucidal activity against poliovirus type 1 {Ward, 1979 #393}. Ammonia (NH_3) has virucidal action, while Ammonium (NH_4^+) is not a virucide against enteroviruses {Ward, 1977 #394}. The effects of chemical germicides on virus survival has been extensively studied, but this topic will not be reviewed extensively here because it has been comprehensively reviewed elsewhere (Prince and Prince, 2001; Sattar and Springthorpe, 2001; Soule *et al*., 1998).

Light: Light has both direct and indirect mechanisms of virucidal activity. The direct activity is likely due to radiation at wavelengths below 370 nm (ultraviolet radiation) being absorbed by proteins and nucleic acids. These effects include breaking of chemical bonds and covalent bond formation, such as pyrimidine dimer formation between adjacent pyrimidines in nucleic acids, nucleic acid strand breaks and changes in virion conformation {Attree-Pietri, 1970 #488;Bitton, 1979 #487;Hill, 1970 #486}. The most active UV wavelengths against viruses and other microbes are in lower ranges of UV B (280-320 nm and UVC (185-280 nm) because wavelengths

in the range to 200 to 280 nm are highly absorbed by nucleic acids. UVA (320-400 nm) is the most abundant UV in sunlight, but it less virucidal than the lower wavelengths. The extent to which UV radiation in sunlight inactivates viruses in water and other aqueous media has not been extensively studied for enteric viruses. However, UV radiation in sunlight is thought to contribute substantially to the inactivation of marine viruses in seawater (Wilhelm *et al*., 2002).

Indirectly, light may trigger the formation of reactive oxidants, or activate photoreactive chemicals {Zafiriou, 1977 #489;Wallis, 1979 #490;Mohr, 1997 #538;Teschner, 1978 #539;Mueller-Breitkreutz, 1995 #540;Jockusch, 1996 #541}. In addition, light may stimulate the growth of antagonistic microflora and fauna {Fukada, 1968 #492;Malherbe, 1967 #491}. Some strains of algae, including *microcytis* and *anabaena*, appear to protect viruses when exposed to light {Bitton, 1979 #487}. Particles such as clay particles can offer some protection against photoinactivation of viruses {Bitton, 1979 #487}.

Factors Affecting Survival in Aerosols

Introduction: Many of the same factors that influence virus survival in water, wastes and other aqueous environmental media also influence virus survival in aerosols. Virus type, virus physical state (dispersed, aggregated, or solids-associated), temperature, particles and suspended matter, organic matter, salts, pH, specific antiviral chemicals, and UV radiation in sunlight all influence virus survival in the aerosol state. In addition to these factors, other factors also become important in influencing virus survival in aerosols.

Relative Humidity and Moisture content: The effect of relative humidity on the survival of viruses in the environment varies with virus type. Generally, viruses with higher lipid content tend to be more persistent at lower relative humidity, while viruses with lesser or no lipid content are more stable at higher relative humidities {Assar, 2000 #323}. Early studies indicated that with a few exceptions, orthomyxoviruses and poxviruses seemed generally sensitive to high humidity, whereas lower humidities favored persistence {Songer, 1967 #318;Webb, 1963 #321;Loosli, 1943 #320;Harper, 1961 #322}. One study indicated that exposure to humidity levels of 80-90% for as short as 30 minutes could render the influenza virus noninfectious to mice, while exposure to lower humidity levels (17-24%) provided the greatest maintenance of infectivity {Loosli, 1943 #320}. Studies have indicated that persistence of non-enveloped viruses like poliovirus and rotavirus is greater at mid to high relative humidities (50-80%) than at low humidities {Harper, 1961 #322;Ijaz, 1985 #265}. However, other studies have concluded that viruses are least persistent at intermediate humidity and more persistent at the extremes. In other studies the survival of some viruses have been shown to be independent of relative humidity{Elazhary, 1979 #288}. Stability of T3, T5, and T7 coliphages has been shown to be related to relative humidity, whereas stability of T2 and T4 coliphages was shown not to be dependent on relative humidity {Hemmes, 1959 #408;Ehrlich, 1964 #406;Happ, 1966 #407;Webb, 1963 #321}.

Picornaviruses and adenoviruses, both of which are non-enveloped virus groups, survive better at high relative humidity {Harper, 1961 #322;Miller, 1967 #426}. In studies examining the survival of rhinovirus types 2 and 14 suspended in buffer in scintillation vials and incubated for 24 hours under wet and drying conditions at three temperatures (6, 23, 37ºC), viral inactivation correlated to evaporation of the suspending media {Reagan, 1981 #363}. Coxsackieviruses that were allowed to dry during incubation were rapidly inactivated {McGeady, 1979 #364}. The influence of relative humidity on the survival of a picornaviruses in the encephalomyocarditis group in aerosols has been examined at 27ºC{Akers, 1968 #315;de Jong, 1970 #459;de Jong, 1974 #452}. Encephalomycarditis viruses survived best under relative humidity conditions above 60%. Survival was the worst between 40 and 60% relative humidity {Akers, 1968 #315}. Similar

influence of relative humidity on survival has also been shown for other strains within the encephalomyocarditis group of picornaviruses (Columbia-SK, Maus Eberfield, and another strain of mengovirus) {Akers, 1966 #319}. Survival of infectious picornaviral RNA was found to be stable at all humidity levels, suggesting that inactivation was attributable to damage of the intact virion {Akers, 1968 #315}.

Consistent with previous studies, Abad et al. found that non-enveloped viruses, including HAV, generally survive longer under conditions of high humidity and low temperature {Sobsey, 1988 #476;Sattar, 1988 #478;Buckland, 1962 #470;Abad, 1994 #239}. On the other hand, Mbithi *et al.* reported that the influence of relative humidity on HAV survival is different from that of other non-enveloped viruses {Mbithi, 1991 #249}. Their results indicated that HAV was most persistent under low relative humidity conditions at least on non-porous material. However, the period of virus exposure to relative humidity conditions in the study of Abad et al. (1994) was of greater duration than that of Mbithi et al. (1991).

Abad et al. also found that rotavirus persisted to a greater extent on porous material at high relative humidity compared to lower relative humidities{Abad, 1994 #239}. In contrast, previous studies have found rotavirus to persist the greatest extent either both at high and low relative humidity, or at low relative humidity {Sattar, 1986 #260;Moe, 1982 #277}. These reported differences in rotavirus persistence can not be readily explained. Perhaps they are due to differences in the methodologies between these studies. For example, Sattar et al. suspended their virus in infant stool and Abad et al. use a healthy adult stool.

Measles and influenza, both enveloped viruses, survive best in aerosols at low relative humidity {de Jong, 1964 #424;Hemmes, 1960 #423}. Similarly, Japanese encephalitis virus has been demonstrated to be most stable in stabilized aerosols at low relative humidity, with half lives of 28, 38, and 62 minutes at 80, 55, and 30%, respectively {Larson, 1980 #281}. Some enveloped viruses have been demonstrated to be least persistent at intermediate relative humidity. Semliki Forest virus in aerosols has been demonstrated to be most vulnerable to inactivation at intermediate RH {de Jong, 1976 #462}. SFV has also been shown to be sensitive to small pressure fluctuations {Druett, 1973 #463}.

Vesicular exanthema virus and feline calicivirus, both non-enveloped and members of the Caliciviridae family, are most stable in aerosols under low (20-30%) or high (70-80%) relative humidity, and least stable at intermediate relative humidity (40-60%) {Donaldson, 1976 #439}. In contrast, at 22ºC, the best survival of psuedorabies virus, an enveloped virus also called Aujesky's disease virus and a member of the Herpesviridae family, was shown to occur at intermediate (50%) relative humidity (half life of 36.1 minutes){Schoenbaum, 1990 #250}. Survival times of this virus were lower and similar at high and low humidity, with half lives of 17.4 and 18.8 minutes, respectively. At 4ºC, the pattern of survival was similar with pseudorabies viruses persisting better at 50% humidity than at 80%.

In a study on the aerosol stability of bovine parainfluenza virus type 3, no significant difference was observed in virus survival during stabilization of the aerosols (during spraying; zero to seven minutes) when there were changes in suspending media, temperature, or relative humidity {Elazhary, 1979 #288}. This appears to be in agreement with another study on parainfluenza performed at room temperature, but is somewhat inconsistent with the results reported by Donaldson and Ferris, where bovine strains of parainfluenza virus were sensitive to high humidity during stabilization of the aerosols {Donaldson, 1976 #439;Miller, 1967 #426}. During the aging of the parainfluenzavirus aerosols (from 1 to 3 hours), virus survival was shown to be greatest under low temperature conditions regardless of relative humidity and suspending media. During

aging of aerosols at 32° C and 30% relative humidity, parainfluenza virus was less stable in Eagle's minimum essential medium than in nasal secretion from a noninfected calf, but at 6 °C and 30% relative humidity, the virus was more stable in Eagle's minimum essential medium. At 32º C, inactivation of bovine parainfluenza virus was greater at high relative humidity (90%) compared to low relative humidity (30%) {Elazhary, 1979 #288}. A similar relative humidity-dependent inactivation was observed at room temperature {Miller, 1967 #426}.

Other studies also report that the effects of relative humidity on virus survival can be influenced either positively or negatively by temperature. For example, simian virus 40 has been demonstrated to display increased inactivation at 50-60% relative humidity only at high temperature (32º C) {Akers, 1973 #446}. At 20ºC human coronavirus was found to be most stable at intermediate humidity, but was also relatively stable at low humidity {Ijaz, 1985 #261}. At 6º C the inactivation pattern at low and medium humidity was similar to that observed at 20º C, but survival was enhanced. In contrast to the poor survival of human coronavirus at 20º C and high (80%) humidity, virus survival at 6º C and 80% humidity was very similar to the best survival at intermediate humidity {Ijaz, 1985 #261}.

Temperature: Generally virus survival varies inversely with temperature. However the degree to which virus survival in aerosols is affected by temperature depends on the virus type and relative humidity. Early studies found Yellow Fever virus (YFV) survival was not significantly affected by temperature, from freezing to 27º C {Mayhew, 1968 #324}. Similarly, Venezuelan Equine Encephalitis (VEE) virus has been reported to be stable in air from -40 ° C to 26 ° C {Ehrlich, 1971 #326}. Both YFV and VEE are enveloped viruses and their patterns of survival at different temperatures in may not be the same for non-enveloped viruses, especially when the viruses are on surfaces. In studies examining the survival of Coxsackie B3, a non-enveloped enterovirus in the Picornaviridae family, suspended in Tris buffer and applied to various surfaces (paper, plastic, and glass), the virus was found to be stable at 6º C and room temperature (23º C), while at 37º C rapid inactivation was observed {McGeady, 1979 #364}. Lower air temperature was also found to enhance the survival of aerosolized human rotavirus at low and medium relative humidity, but not at high relative humidity {Ijaz, 1985 #263}. Colder temperatures have also been shown to enhance rhinovirus survival at high relative humidities {Karim, 1985 #262}.

Temperature also has been shown to be a factor in the aerosol survival of herpesviruses. Psuedorabies virus in aerosolized form survives better at 4º C than at 22º, although not significantly {Schoenbaum, 1990 #250}. Herpesvirus of turkeys (HVT) survived better at 0º C than at either 25 ° or 37º C. HVT was reduced by 0.3 to >2.0 $\log_{10}$ at 37º C after only 90 minutes {Colwell, 1975 #430}.

Composition of Suspending Media: The stability of virus infectivity in aerosols at different relative humidities is affected by solutes in the suspending media used for aerosolization. That is, certain solutes in the medium from which virus aerosols are generated influence the survival of aerosolized viruses. Benbough found that the removal or reduction of salts from the solution used to aerosolize Langat virus (Flaviviridae family) was protective of virus infectivity at moderate relative humidity {Benbough, 1971 #306}. In contrast, Semliki Forest virus (Togaviridae family) was affected by salts in the solution used for aerosolization only at high relative humidity. However in further studies, the removal of salts from the suspending media resulted in improved survival of Semliki Forest virus in aerosols under all humidity conditions tested {Benbough, 1969 #309}.

Poliovirus and T coliphages (T1, T2, and T7) were found to survive best at high relative humidity and their survival at low relative humidity was found to decrease further when solutes were

removed from the virus suspension from which aerosols were generated {Benbough, 1971 #306; Strange, 1972 #302}.

However, salts may be toxic to certain viruses in aerosols and frozen or freeze-dried stocks by modifying the rehydration rate{Benbough, 1971 #306}. Other potential mechanisms for salt toxicity on frozen viruses have also been described, and include the ability of strong salt solutions to dissolve lipoprotein {Lovelock, 1957 #428;Greiff, 1966 #427}.

The effects of the composition of the suspending medium from which viruses are aerosolized are difficult to predict and characterize because the effects vary depending on relative humidity and a number of the constituents within the suspending medium. For example, Influenza A virus, strain WSNH, propagated in bovine, human and chick embryo cell cultures and aerosolized from the cell culture medium, was maximally stable at low relative humidity (RH), minimally stable at mid-range RH, and moderately stable at high RH. Most lots of WSNH virus propagated in embryonated eggs and aerosolized from the allantoic fluid were also least stable at mid-range RH, but two preparations after multiple serial passage in eggs showed equal stability at mid-range and higher RH levels. Airborne stability varied from preparation to preparation of virus propagated both in cell culture and embryonated eggs. There was no apparent correlation between airborne stability and protein content of spray fluid above 0.1 mg/ml, but one preparation of lesser protein concentration was extremely unstable at 50-80% RH. Polyhydroxy compounds exerted a protective effect on airborne stability. Removal of calcium and magnesium from preparations of influenza was shown to have variable effects on virus survival, with greater virus survival at intermediate humidity being documented in some cases, and no effect seen in other cases. Replacement of the divalent cations had no effect on virus survival{Schaffer, 1976 #296}. Adjustment of the pH in allantoic influenza preparations had no appreciable effect on the survival of influenza at intermediate humidity {Schaffer, 1976 #296}.

T3 coliphage is rapidly inactivated in an aerated solution of 2.6M NaCl. Inactivation does not occur with out aeration suggesting that the air water interface is important for inactivation. Addition of 1% peptone provided a protective effect against inactivation by aeration. Similar protection was afforded to aerosolized T3 by peptone {Trouwborst, 1974 #299}. These findings are consistent with previous studies suggesting that certain viruses, both enveloped, hydrophobic viruses as well as non-enveloped, hydrophilic viruses, have a tendency to migrate to the surface of the air-water interface in liquid solutions or in aqueous aerosol particles. At the air-water interface, these viruses are inactivated by shear forces and other forces that alter virus structures. Indeed, it is well documented that viruses become inactivated and proteins lose activity upon exposure to air-water interfaces (AWIs) (Adams, 1948; James and Augenstein, 1966; Trouwborst *et al*., 1972; Trouwborst, and de Jong, 1973; Trouwborst *et al*., 1974). These phenomena will be discussed in more detail in a later section of this report.

The stability of viruses in the environment relates is also related to the partition of bound and unbound water, as well as the other constituents of the aerosol particle containing the virus, and the surrounding atmosphere {Benbough, 1971 #306}. Consequently the survival of the viruses may depend on the water activity of the aerosol solution. The water activity of the aerosol particle is also related to its composition.

In a study examining the effect of suspending media on survival of infectious bovine rhinotracheitis virus, IBR virus was shown to survive spraying of the aerosol best at 32º C than at low temperature 6º C in both Eagle's minimal essential medium (EMEM) and bovine nasal secretions at 30% relative humidity {Elazhary, 1979 #290}. At 90% percent humidity, survival of IBR virus was better in bovine nasal secretion at 6º C. The authors hypothesized that the

reason for the better survival at the higher temperature during spraying of the aerosol might be due to transitory freezing and thawing events occurring at 6º C that were damaging to the virus. Generally virus survival was found to be better in bovine nasal secretion during spraying, but survival was better in Eagle's Minimum Essential Medium in stabilized aerosols. The authors suggest that the better survival in bovine nasal secretion during spraying may be due to greater protein and lower salt content.

In a similar study examining the aerosol stability of bovine adenovirus, EMEM was found to be generally more protective during aerosol stabilization than bovine nasal secretions at both 30 and 90% relative humidity {Elazhary, 1979 #444}. Also, EMEM was generally more protective during aging of the aerosols, although inactivation rates were similar to those in bovine nasal secretions. Generally bovine adenovirus was found to be most persistent in aerosols at high humidity and low temperature {Elazhary, 1979 #444}. These results are consistent with other studies reporting that other non-enveloped viruses, including human adenovirus type 7, are most stable in aerosols under conditions of high humidity and low temperature {Davis, 1971 #445;Miller, 1967 #426}.

The previously noted effects of protein and organic matter content of the suspending media on virus survival in aerosols has also been reported in other studies. Addition of salt and protein (in the form of BSA) to coxsackieviruses suspended in Tris buffer provided a protective effect even at 37ºC{McGeady, 1979 #364}. Salt and protein have also been observed to provide some protection against dehydration and thermal inactivation for Rhinovirus {Reagan, 1981 #363}. Removal of protein from the suspending medium was shown to increase the inactivation of SFV at high relative humidity{Benbough, 1971 #306}. Addition of protein to aerosolization solution also resulted in improved survival of SFV, especially at high relative humidity {Benbough, 1969 #309}. Influenza virus from a cell culture preparation with a protein content of 0.075 mg/ml was very unstable in aerosolized form {Schaffer, 1976 #296}. Airborne stability of influenza was improved at 0.1 mg/l protein in the solution from which the aerosol was generated, but it was not further improved by protein content greater than 0.1 mg/ml.

The effects of organic matter on virus survival in aerosols and on surfaces also have been studied for rotaviruses. Human rotavirus aerosolized in a 10% fecal suspension was found to have improved survival compared to virus aerosolized in tryptose phosphate buffer {Ijaz, 1985 #263}. Studies also have been performed to examine the protective effects of organic suspensions on the survival of human rotavirus (CJN) during drying {Ward, 1991 #370}. Previous studies had shown fecal matter to be protective of rotavirus against inactivation in a dried state {Moe, 1982 #277;Sattar, 1986 #260;Woode, 1975 #477}. However, Ward *et al*. found fecal matter to be only slightly more protective than distilled water {Ward, 1991 #370}. Other organic suspensions (tryptose, tryptose phosphate, and gelatin) were found to be much more protective, especially non-fat dry milk. The reason for the apparent discrepancy in the protective effect of fecal matter compared to other organics tested may have been due to the presence of neutralizing antibody in the faeces (coproantibodies); or perhaps seeded fecal material may not be as protective as naturally shed fecal material containing viruses{Ward, 1991 #370}.

Addition of porcine mucin to suspending medium was demonstrated to improve the aerosol survival of psuedorabies virus {Schoenbaum, 1990 #250}. Mucus is composed mucin (a glycoprotein usually present in mucus as a polymer or complex) and cells, and is intimately associated with viruses originating from nasal and nasopharyngeal surfaces.

The addition of polyhydroxy compounds has been found to protect against or overcome the negative effects of salt on the survival of viruses in aerosols {Benbough, 1971 #306;Webb, 1967

#317}. Addition of polyhydroxy compounds (inositol, glucose, raffinose) improved survival at low and moderate relative humidity, but they were ineffective at high relative humidity {Benbough, 1969 #309}. Addition of inositol to the aerosolized solution stabilized RSV at all lower humidity levels tested {Webb, 1963 #321}. Inositol (6%) has been shown to stabilize survival of influenza at intermediate humidity, however inactivation was enhanced at high relative humidity. The presence of inositol had no effect on virus survival at low humidity compared to samples lacking inositol {Schaffer, 1976 #296}. Sucrose was shown to be equally or more effective than inositol in stabilizing influenza at intermediate humidity, and it did not increase inactivation at high relative humidity. Glucose was less stabilizing than sucrose at the same concentration. Dimethylsulfoxide did not show a significant stabilizing effect on influenza survival.

In another study, inositol was found not to protect vesicular exanthema virus (VEV) from inactivation under unfavorable relative humidity {Ferris, 1980 #284}. NaCl was found to increase VEV survival at intermediate relative humidity. Inclusion of glucose, glycerol, DMSO, and BSA were not found to improve survival at intermediate humidity.

Inositol is thought to replace structural water molecules thus stabilizing the virus {Webb, 1963 #321}. Aerosolization in citrate buffer reversed the survival pattern for RSV with survival becoming greatest at low humidity (>50% survival at five hours in 20% humidity) and poor at high humidity (above 60% humidity less than 10% of virus remained at 5 hours) {Webb, 1963 #321}. Addition of serum to salt buffer counteracted the protective effect on RSV survival at lower humidity {Webb, 1963 #321}.

Oxygen and Air Ions: Atmospheric oxygen was found not to effect aerosolized viruses {Benbough, 1971 #306}. Similarly, Webb found that oxygen concentration had little effect on survival of bacteriophages T7 and F2 in aerosols {Webb, 1967 #317}. However, the airborne stability of T1 coliphage was shown to be affected by air ions {Happ, 1966 #407}.

OAF: Open air has virucidal activity {May, 1969 #442;Benbough, 1971 #431}. The term "open air factor" (OAF) was coined in studies to explain the significantly lower survival observed for aerosolized bacteria in open air as compared to clean laboratory air {Druett, 1973 #456;Cox, 1995 #458;Mohr, 1991 #454}. It has been proposed that the OAF may be attributed to reactions between ozone and olefins, or a combination of factors including pollutant concentration, relative humidity, pressure fluctuations, and air ions {Mohr, 1991 #454;Cox, 1987 #457;Cox, 1995 #458;Druett, 1968 #440;De Mik, 1977 #443}. The reactive products of ozone and olefin also have virucidal properties {Druett, 1972 #441}. Bacteriophage ΦX174 is relatively stable in clean air of high humidity. In a study examining the effect of ozone and olefins on bacteriophage ΦX174, ozone alone was found to have a minimal effect on virus survival with a maximum effect observed at 30ppb{De Mik, 1977 #293}. In the presence of various olefin compounds, however, viral inactivation increased with increasing ozone concentrations.

The influence of OAF and daylight on the survival of foot and mouth disease (FMD) virus was examined by Donaldson and Ferris {Donaldson, 1975 #298}. OAF was not shown to be a significant factor in inactivation of FMD virus in aerosols. Semliki forest virus and T coliphages, on the other hand, were inactivated at a considerably greater rate in open air, as opposed to enclosed aerosol studies {Benbough, 1971 #431}. SFV has been demonstrated to be rapidly inactivated by OAF {Benbough, 1971 #431}.

Other Antiviral chemicals: Berendt and Dorsey examined the effects of light and sodium fluoresce in on the recovery of Venezuelan equine encephalitis virus (VEE) after one hour at 30

or 60% relative humidity{Berendt, 1971 #328}. Inactivation of aerosolized VEE in a dark environment at 30 and 60% relative humidity was found to be less than or equal to 0.1 $\log_{10}$ reduction. Under simulated solar radiation, inactivation increased to 3.7 and 4.2 $\log_{10}$ reduction at 30% and 60 %, respectively. The addition of sodium fluorescein had a negative effect on the survival of VEE in non-irradiated aerosols at 60% relative humidity, and under simulated solar conditions, no VEE could be detected after 30 minutes at either humidity level{Berendt, 1971 #328}.

Light: As previously noted, the main antiviral component of sunlight is UV radiation, primarily UVA (320-400 nm). Virus susceptibility to UV exposure also appears to vary with virus type. Early studies suggested that viruses, such as coxsackie, influenza, sindbis, and vaccinia, are highly susceptible (>3 $\log_{10}$ inactivation) to UV radiation at an exposure of 0.03 watt-minutes/ft^3. Adenoviruses, on the other hand, were demonstrated to be more resistant (<2 $\log_{10}$ inactivation) {Jenson, 1964 #327}. Venezuelan equine encephalitis virus has been demonstrated to be highly sensitive to radiation {Berendt, 1971 #328}. Compared to other viruses (vaccinia, herpes simplex, fowl plaque, louping-ill, Borna disease, vesicular stomatitis, influenza, Newcastle Disease, and canine distemper), FMD virus has been show to be relatively photoresistant {Skinner, 1954 #433;Perdau, 1933 #432;Donaldson, 1975 #298}. Whereas other viruses were inactivated by exposure to daylight for 4 hours, FMD virus was stable. Other picornaviruses have also been shown to be relatively photoresistant. Over a similar four hour period, poliovirus was not inactivated, while Sindbis, Murray Valley encephalitis virus, influenza, and rabbit pox viruses were inactivated {Appleyard, 1967 #434}.

Aerosol Particle Size: Aerosol particle size may effect the survival of airborne viruses, with more rapid inactivation in smaller particles than large ones {Moe, 1983 #273}.
Respiratory syncytial virus (RSV) in small particle aerosols is rapidly inactivated {Rechsteiner, 1969 #472}. Naneva et al. (1983) also evaluated the stability Aujeszky's disease virus in aerosol particles of different size, with 5 min. exposure at 80-100 per cent relative humidity, and a temperature range of 14-21.2^{o} C. The sizes of all aerosol particles were >5 micron, and they differed according to the composition of the suspending medium. Viruses differed in their survival in different aerosols, but the effects of different aerosol particle size and different particle composition were not resolved. Therefore, the effects particle size alone could not be specifically determined. Highest virus stability (11.2% survival) was in a 0.2 M buffered saline containing 10% peptone and 0.8% saccharose at pH 7.

Aerosol Collection Method: Rehydration of aerosol particles upon collection may be responsible for either inactivation or protection of viruses in aerosols and the effects can differ for enveloped and non-enveloped viruses. Salts and other solutes may protect T coliphages and poliovirus by influencing the rehydration rate during collection {Benbough, 1971 #306}. With some viruses (e.g. poliovirus, FMDV, and bacteriophages T3) prehumidification prior to or during aerosol particle collection has been demonstrated to increase the level of infectious virus that may be recovered {Hatch, 1969 #448;Warren, 1969 #449;Dubovi, 1976 #450;Benbough, 1971 #306;Barlow, 1972 #451;de Jong, 1974 #452;de Jong, 1975 #453}. This suggests that inactivation occurs during aerosol particle collection unless steps are taken to protect the virus during collection by prehumidification. However, prehumidification during aerosol particle collection does not appear to significantly effect recovery of vesicular exanthema virus or mengovirus {Ferris, 1980 #284;Warren, 1969 #449}. {de Jong, 1970 #459}. Prehumidifcation was not shown to improve recovery of infectious encephalomyocarditis virus from aerosols {de Jong, 1970 #459}.

Factors Affecting the Survival of Viruses on Surfaces and in Soil

Introduction: The same factors that influence virus survival in water, wastes and other aqueous environmental media and in aerosols also influence virus survival on surfaces and in soils. Virus type, virus physical state (dispersed, aggregated, or solids-associated; especially and the extent and state of virus adsorption), temperature, particles and suspended matter, organic matter, salts, pH, specific antiviral chemicals, UV radiation in sunlight, relative humidity, moisture content and water activity all influence virus survival on surfaces and in soils.

Adsorption State: The extent and state of virus adsorption has an important influence on virus survival on surfaces and in soils. Most studies have shown that the survival of some viruses is increased with increased adsorption to soil or surfaces{Hurst, 1980 #552}.

However, the phenomena influencing virus interactions with and survival on surfaces and in soils are somewhat similar to those influencing the survival of viruses at the air-water interface, but they are further influenced by the solid surface with which the aqueous medium and the air are in contact. As previously noted, studies have shown that viruses become inactivated and proteins lose activity upon exposure to air-water interfaces (AWIs). However, more recent studies by Thompson et al. (1998) suggest that factors other than the AWI alone are responsible for viral inactivation when the viruses are in a three-part system consisting an aqueous medium, a surface and air. This is referred to as a triple-phase-boundary [TPB]) system and can be depicted as a line or boundary (perimeter) at which an AWI meets a surface, such as a soil particle or table top.

Thompson et al. hypothesize that viruses in solution reach the AWI, where they adsorb, via convection and diffusion. This adsorption is dominated by electrostatic, hydrophobic, hydration, and capillary forces; solution ionic strength; pH; and various other factors. As a virus adsorbs to the AWI, hydrophobic domains on the protein surface (e.g., the capsid of a non-enveloped virus) partition out of the solution and into the more nonpolar gas phase. They suggest that such exposed domains on the virus capsid are susceptible to forces at the TPB which are not present at the AWI itself. Unlike the AWI, the balance of forces at the TPB will be influenced by the surface characteristics of the solid (surface or soil). The forces acting on the aqueous droplet will balance at equilibrium, and these are the solid-air, solid-water, and air-water surface tensions are described by a contact angle, which is cosine of the angle of contact between liquid and solid. Forces influencing an exposed virus particle at the TPB are much different from those at the bulk AWI and are dictated by the type of surface with which the virus containing aqueous medium and air are in contact. The orientations of water molecules at large surfaces are different from those in bulk water, especially for hydrophobic surfaces. Therefore, it is proposed that virus particles partitioned at the TPB experience destructive forces as a result of the reconfiguration of water molecules near the surface. Virus surface proteins projecting into the gas phase may also interact with the surface at the TPB. As the AWI is shears away from a surface, such as during evaporation or transport, partitioned virus particles can undergo shear stress. Such forces can cause structural changes to a virus capsid, resulting in loss of infectivity. Hence, it is proposed that the main site of phage inactivation is the interface at which the liquid, solid, and gas phases meet.

These TPB effects are more likely to occur when the surface is hydrophobic, as was demonstrated in the study by Thompson et al. (1998). In comparing the inactivation of MS2 coliphage in an experimental system consisting of air-water-solid interfaces with glass and polypropylene (PP) surfaces, inactivation was much greater in the presence of the PP surface than the glass surface. Glass is hydrophilic, with a contact angle against water of 45°, while PP is a hydrophobic organic polymer with a contact angle of 108°. There was a lack of MS2 inactivation in the presence of a hydrophilic glass surface, indicating that the forces responsible for virus loss are greater in the

hydrophobic PP system, or if they are present in the glass system they do not influence virus fate in the same manner.

While the above phenomena suggest that air-water-solid interfaces can contribute to virus inactivation, it has been repeatedly observed that viruses adsorbed to soils and surfaces are inactivated at lower rates than they are when simply in an aqueous suspension alone. Therefore, there must be other explanations for the apparent protective effects of virus adsorption to surfaces and soils. It is possible that adsorption to some surfaces stabilizes virus structures or that virus sites for attack by antiviral agents are blocked due to their adsorption to the surface. The protective as well as antagonistic factors influencing virus survival on surfaces will be discussed in later sections of this report.

Relative Humidity: The effect of relative humidity on the survival of viruses on surfaces is virus type dependent. The half-life of HIV in phosphate buffer with 2% fetal bovine serum at room temperature was similar both dried and in solution {Tjotta, 1991 #369}.
Similarly, the inactivation rate of hepatitis B virus was demonstrated not to be affected by relative humidity {Favero, 1974 #343}. However, Mahl and Sadler generally found that the viruses they tested survived longer at low humidity than at mid or high RH. However, coxsackie virus, vaccinia, and adenovirus appeared to be minimally affected by relative humidity {Mahl, 1975 #366}. Studies examining the loss of infectious titer due to drying of rhinovirus have reported greater losses at 84% relative humidity than at 20% relative humidity {Buckland, 1962 #470}. Herpes Simplex Virus type 1 has been demonstrated to be very susceptible to desiccation in studies examining its survival on surfaces with reductions of 2 $\log_{10}$ accompanying evaporation of the suspending medium {Bardell, 1994 #336}.

Rotaviruses in faeces have been shown to be most stable at higher relative humidities (>75%). On surfaces, rotavirus was also more stable at very low relative humidities (<14%) than at moderate humidities (15-74%) {Moe, 1982 #277}. There results are in contrast to studies, in which rotavirus in aerosols was rapidly inactivated at high relative humidity and 22ºC. Other studies with rotavirus aerosols have shown a similar sensitivity of various rotaviruses to high relative humidity {Sattar, 1984 #269;Ijaz, 1985 #265;Ijaz, 1985 #263}. It is likely that methodological or virus strain differences are responsible for the different patterns of inactivation between these studies on surfaces versus those in aerosols. Given the proposed differences in mechanisms of virus inactivation at air-water interfaces (AWI) and in triple phase boundary (TPB) systems consisting of air-water-surfaces, such differences in rotavirus survival with respect to relative humidity are plausible.

Some studies on the survival of foot and mouth disease (FMDV) have assumed that relative humidity was only important in aerosol transport and not in the survival of FMDV on surfaces {McColl, 1995 #235}. However, this was contradicted by the differences in virus survival observed in dry rather than wet hay and bran {Bartley, 2002 #225;McColl, 1995 #235}.

Vaccinia virus has been shown to be generally not as stable at high humidity (78%) as lower humidity on wool or cotton {Sidwell, 1966 #464}. Poliovirus survival on fabrics also was also dependent on relative humidity, with greatest persistence under low humidity conditions. Initial inactivation was less extensive under high relative humidity, but the subsequent rate of inactivation was higher than at low relative humidity{Dixon, 1966 #350}.

Moisture content: The extent of virus survival in the subsurface seems to relate directly to the amount of moisture in the soil {Vaughn, 1983 #530}. As a general rule, pathogens survive longer under moist conditions {Gerba, 1984 #554;Hurst, 1980 #552}. As soil moisture levels decrease,

viruses may be inactivated by desiccation or enhanced predation due to a thinner moisture layer with biological activity around individual soil particles. Inactivation of poliovirus type 1, echovirus type 1, and indigenous enteroviruses in the soil of a wastewater infiltration basin has been shown to be dependent on the rate of moisture loss, with drying cycles increasing virus inactivation {Hurst, 1980 #282}. Drying of soil is virucidal to both poliovirus and coxsackievirus {Yeager, 1979 #376}.

Temperature: Temperature plays an important role in virus survival in soils and on surfaces, however the degree of the effect is virus type dependent. As in other environmental media viruses in soils and on surfaces are inactivated more rapidly as temperature increases. For example, poliovirus type 1 and Coxsackievirus B1 were more rapidly inactivated in soil as temperature increased {Yeager, 1979 #376}.

Under low moisture conditions, such as on surfaces, temperature can either protect against or enhance the virucidal effects of relative humidity. Temperature has been shown to have a variable effect on the survival of different viruses on glass surfaces {Mahl, 1975 #366}. Whereas coxsackie virus and adenovirus survival was not affected by increased temperature, vaccinia survival at 25º C was greater than at 37º C, independent of relative humidity. Poliovirus and herpes simplex virus survival at mid (55%) and high (93-96%) relative humidity was also greater at 25º C than at 37º C, but at low humidity, temperature was not a factor. Influenza virus was shown to survive less well at 37º C than at 22º C on glass slides {Edward, 1941 #466}. Reduction of the temperature to 4ºC from 22ºC, has been shown to have a protective effect on human rotavirus survival on surfaces especially at high relative humidity {Sattar, 1986 #260}.

The effect of air temperature as well as relative on survival of hepatitis A virus on environmental surfaces has been studied (Mbithi et al., 1991)**.** Stainless steel disks contaminated with fecally suspended hepatitis A virus (HAV; strain HM-175) were held at RH levels of 25, 55, 80 and 95% and at air temperatures of 5, 20, or 35^{o} C. HAV survival was inversely proportional to the level of RH and temperature, and the half-lives of the virus ranged from greater than seven days at the 25% RH and 5^{o} C to about 2 h at the 95% RH and 35^{o} C. In parallel tests with fecally suspended Sabin poliovirus (PV) type 1 at the 25 and 95% RH, all PV activity was lost within four and twelve hours, respectively. HAV could therefore survive much better than PV on nonporous environmental surfaces. Moreover, the ability of HAV to survive better at low levels of RH is in direct contrast to the behavior of enteroviruses.

Laundering of poliovirus contaminated fabrics was shown to significantly reduce viral contamination {Sidwell, 1971 #344}. Water temperature was an important factor in virus inactivation with greater inactivation observed at higher temperatures {Sidwell, 1971 #344}.

Suspending Media: Similar to virus survival in aerosols, the composition of the suspending medium may effect the virus survival on surfaces. When applied to countertops in water, rotavirus was inactivated with in 45 minutes, while poliovirus and bacteriophages survived to 90 minutes with reductions of 1.5 and 3 $\log_{10}$, respectively {Keswick, 1983 #270}. Application in 10% stool suspension improved survival of SA11 and f2 coliphage to 90 minutes with inactivation of about 3 and 1.5 $\log_{10}$, respectively. The inactivation kinetics of poliovirus were initially increased, but overall virus survival did not appear to be improved. The presence of fecal material was also shown to have a protective effect on the survival of rotavirus on non-porous surfaces, as determined by similar experiments using tryptose phosphate buffer to suspend the viruses {Sattar, 1986 #260}.

Type of Surface: Little effect on virus survival has been documented for the type non-porous surfaces used in most studies of virus survival. For example, no difference in adenovirus persistence was observed on different non-porous surfaces (glass, vinyl asbestos tile, ceramic tile, or stainless steel) {Mahl, 1975 #366}. On the other hand, Herpes simplex virus type 2 survival on surfaces was shown to be related to the surface type {Larson, 1985 #355}.

The effect of surface type on virus survival is much more pronounced with porous surfaces. Differences have been documented between different fabrics of the same material, and for different materials {Sidwell, 1966 #464;Dixon, 1966 #350}. A "wash and wear" fabric finish was found to negatively effect the persistence of both vaccinia and poliovirus. Fabrics containing synthetic fibers have been shown to allow a greater viral persistence than seen in cotton fabrics {Sidwell, 1970 #346}. Viral contamination on cotton, wool and synthetic fabrics has been shown to be transferable to sterile fabrics by dry contact {Sidwell, 1970 #346}. Poliovirus could be isolated from laundry rinse water, and from previously sterile fabrics co-washed with the contaminated fabrics.

The survival of viruses in soil has been shown to be soil type dependent {Sobsey, 1980 #283;Meschke, 1999 #571}. Composition of soil surface to which viruses are adsorbed can effect their viability. In studies by Murray et al. no loss of infectivity was observed when sorbed viruses were recovered from silicates and iron oxides, whereas recovery from other metal oxides (CuO, MnO_2, and Al_2O_3) revealed that viruses were physically disrupted and non-infectious {Murray, 1980 #536;Murray, 1979 #535}.

The amount of organic matter in a system can influence virus adsorption to soils and surfaces. Generally, organic matter will have a low isoelectric point and thus carry a net negative surface charge at most naturally occurring pH levels. Consequently, organic matter can compete with viruses for adsorption sites on soil particles, thus negatively impacting virus survival {Gerba, 1985 #562;Gerba, 1984 #554}. This may also be the reason why soils with high organic matter content are relatively poor adsorbers of virus {Gerba, 1984 #556;Sobsey, 1986 #563}. However, it has been suggested that soils with low clay content require conditioning with treated effluents (and thus organic matter) for efficient virus removal {Vaughn, 1983 #530}. Paradoxically, organic matter also has been shown to increase survival of certain enteric bacteria and viruses in soil, perhaps by forming protective barriers around viral particles, or by acting as targets or substrates for enzymes and other antiviral chemicals. Organic matter competition for adsorption sites on soils may increase virus mobility, through reduction of virus adsorption. This is one reason that organic matter (e.g. beef extract) is often included in eluants used to recover adsorbed viruses {Wait, 1983 #564}.

Light: The effects of light on the survival of viruses on surfaces has not been extensively studied. The few studies reported indicate generally less virus survival when surfaces are exposed to light. Foot and mouth disease virus persisted longer in the dark than in the light on hay {Bartley, 2002 #225}. Viruses on inanimate surfaces exposed to light survived less than one week, whereas those maintained in the dark survived between 3 and 6 weeks {Mahl, 1975 #366}. Light had no effect on virus survival at low humidity. At high humidity, exposure to light had a significant negative impact on adenovirus survival {Mahl, 1975 #366}.

Antiviral Chemicals: Studies found that fabrics impregnated with quaternary ammonium compounds had a virucidal effect on vaccinia, but not poliovirus {Sidwell, 1967 #348}. Type of detergent during laundering (ionic or anionic) was not a significant factor in viral inactivation on fabrics {Sidwell, 1971 #344}. The effect of chemical disinfectants on viruses on surfaces has been extensively studied because of its importance in preventing the spread of viruses in

healthcare settings, food establishments and other settings. This subject will not be reviewed here because it has been reviewed extensively elsewhere (Sattar *et al*., 2001; 1994). However, it is important to note that disinfection of human enteric viruses (HAV and rotavirus) on fomites (polystyrene) by several commercially available disinfectants has been reported to be poor, with less than 3 $\log_{10}$ inactivation (Abad *et al*., 1997). Furthermore, suspension tests performed with the same disinfectants showed different virus inactivation rates, thus failing to provide a reliable indication of the extent of virus disinfection on fomites.

VIRUS SURVIVAL TIMES AND INACTIVATION KINETICS IN DIFFERENT ENVIRONMENTAL MEDIA

Introduction

In the following section of this report data are presented in tabular form and in text on the survival times of different viruses in various environmental media and under different environmental conditions such as temperature and pH. The purpose of these data compilations is to provide some quantitative information on the extent of virus survival and the rates and extent of virus inactivation in various environmental media and at different conditions.

Survival Studies in Liquid Media

Table 3. summarizes some survival data for human and animal viruses in various liquid and semi-sold media, including faeces, sludges and soil materials. Additional study descriptions are included below by type of medium.

Distilled and Drinking Water: Keswick and Gerba cite virus survival for up to 200 days in drinking water {Keswick, 1980 #531}. Rotavirus has been demonstrated to be stable in drinking water (loss of less than 1 $\log_{10}$) for up to 64 days at 4° C, while at 20° C virus infectivity was reduced by 2 $\log_{10}$ after the same period {Sattar, 1984 #374}. Rotavirus has been shown to be less stable in distilled water than in drinking water, with 1 $\log_{10}$ loss of infectivity after less than one hour {Sattar, 1984 #374}. Herpes Simplex Virus has been shown to persist up to 24 hours in distilled water, while in tap water it persisted less than 4 hours, and in spa water was immediately inactivated {Nerurkar, 1983 #361}. In sterile water at pH 7, Schwartzbrod et al. found poliovirus could survive for 296 days at a temperature of 18-23º C, and longer at a temperature of 4ºC {Schwartzbrod, 1975 #537}. Pseudorabies virus has been shown to be persistent in chlorinated water for less than 1 day {Schoenbaum, 1990 #250}. Astrovirus persistence has been documented to be as long as 90 days in dechlorinated drinking water at both 4 and 20ºC. Astrovirus was also relatively resistant to chlorination, persisting at detectable levels in water with a free chlorine level of 1.0 mg/L, although a 4 $\log_{10}$ reduction was seen {Abad, 1997 #483;Abad, 1994 #484}.

Fresh Surface Water: Data compiled by Sattar (19??) on virus survival in surface waters indicates that many enteric viruses require weeks to months to be reduced by 99 to 99.99% (2 -4 $\log_{10}$). Virus survival has been reported to be as long as 188 days in surface water {Keswick, 1980 #531}. Coxsackie A9 has been reported to survive for up to 30 days in 15°C stream water and seawater with reductions of 2.0 $\log_{10}$ {Nasser, 2003 #224}. Human astrovirus survival in water has been shown to be similar to that of the enteric adenoviruses and human rotavirus {Abad, 1997 #483; Abad, 1994 #484}. During a waterborne outbreak of Norovirus, epidemiologic evidence suggested that the source of the virus was water from a frozen river that had been contaminated upstream 4 months earlier {Kukkula, 1999 #233}.

Soil and Groundwater: In a study by Landry et al., virus survival in groundwater was calculated to be at least 200 days based on the velocity of groundwater movement {Vaughn, 1983 #530}. Wellings et al. report the survival of enteroviruses for at least 28 days in a Florida cypress dome {Wellings, 1975 #378}. Other researchers have reported the survival of poliovirus in sand for up to 91 days {Lefler, 1974 #402}. Anecdotal evidence from an outbreak at a caravan park in Australia, suggests that enteroviruses and caliciviruses may persist in groundwater and sediments for at least 4 weeks {McAnulty, 1993 #244}. Other evidence suggests that viruses may persist in sediments and that sediments may serve as a reservoir for viral infection {Ellender, 1987 #389}. Bagdarsar'yan reported survival of echovirus 7 for up to 170 days in seeded sandy soil during winter months {Bagdasar'yan, 1964 #532}. Tierney et al. reported survival of viruses in sewage sludge, effluent irrigated soil, or sludge amended soils for 11 days in the summer and up to 96 days under winter conditions {Tierney, 1977 #421}. Pseudorabies virus was found to be persistent in well water for up to 7 days {Schoenbaum, 1990 #250}. Hurst *et al.* found that viral inactivation rates during sludge treatment were slight. However during field application and drying, inactivation rates ranged from 1.5-2.3 $\log_{10}$ over a period of seven days . However, enteric viruses have been isolated from dried sludge and field sprayed sludge {Wellings, 1976 #377}.

Sea and Brackish Water: Coxsackie A9 has been reported to survive for up to 30 days in seawater with 2.2 $\log_{10}$ reduction in titer, and in brackish water there was no observed reduction in infectivity titer (<0.1 $\log_{10}$) {Nasser, 2003 #224}. Callahan *et al*. (1995) studied the survival of two groups of enteric bacteriophages, F-specific coliphages (FRNA phages) and somatic *Salmonella* bacteriophages (SS phages) and two human enteric viruses, hepatitis A virus (HAV) and poliovirus type 1 (PV-1), in coastal seawater from three geographic areas (Southern California, Hawaii, and North Carolina) at 20^{o} C. Concentrations of all four viruses decreased over 30 days from their initial titers and there was little difference in the survival of a particular virus among the three seawaters. However, the extent of reduction varied among the four viruses. Survival was greater for the SS phages than for any of the other viruses, with an estimated 4 $\log_{10}$ reduction time of about 10 weeks. FRNA phages and PV-1 were inactivated rapidly, with 4 $\log_{10}$ reductions in ˜ 1 week. HAV reductions were intermediate between SS phages and FRNA phages, with 4 $\log_{10}$ reductions in about four weeks.

The survival of four strains of feline calicivirus in marine water was measured diluting virus stock 1/10 with marine water maintained at 4, 10 and 20 ^{o}C (Kadoi and Kadoi, 2001). Considerable amounts of infective viruses remained at 10 and 4^{o} C for a period of 30 days.

Wait and Sobsey (2001) studied the survival of enteric bacteria (*Escherichia coli*, *Salmonella typhi*, *Shigella sonnei*), and enteric viruses (poliovirus type 1 and the parvovirus Minute Virus of Mice in Atlantic Ocean seawater. Seeded seawater was incubated in the laboratory at 6, 12, 20 and 28°C for up to 40 d. *In-situ* survival studies were done seasonally (winter, spring, summer and fall) using seeded microbial dialysis equipment placed in the Atlantic Ocean off coastal North Carolina at water depths of 3-10 m. In laboratory studies all test microbes survived longer at lower temperatures with typical times for 90% inactivation (T_{90}) of 1-3 d at the highest temperature and >10 d at the lowest temperature. Of the microbes tested, *E. coli* survived least well while *S. typhi* and *Sh. sonnei* survived similar to or greater than enteric viruses. Parvovirus survival was similar to that of poliovirus. Overall, microbial survival in seawater was greater under laboratory conditions than under in-situ conditions. There was no clear association between microbial survival and water temperature.

Enveloped virus also have been shown to survive for extended periods of time in water. For example, bovine diarrhea virus, a member of the Flaviviridae family and Pestivirus genus, has

been shown to persist in various types of water for periods ranging from 6±24 days at 20°C (Pagnini et al., 1984). Kocan et al. (2001) studied the survival of viral hemorrhagic septicemia virus (VHSV) (Rhabdoviridae family). VHSV could be recovered for up to 40 h in natural filtered seawater (27 ppt) with a 50% loss of infectivity after approximately 10 h at 15 degrees C. Addition of 10 ppb North Slope crude oil to the seawater had no effect on virus survival. However, when various concentrations of teleost ovarian fluid were added to seawater, virus could be recovered after 72 h at 0.01% ovarian fluid and after 96 h at 1.0%. When cell culture medium supplemented with 10% fetal bovine serum was added to the seawater, 100% of the virus could be recovered for the first 15 d and 60% of the virus remained after 36 days. Ovarian fluid occurs naturally during spawning events, so its prolongation of virus survival may have practical implications.

Faeces, Wastewater and Sludges: Both non-enveloped and enveloped viruses can survive for extended time periods in untreated as well as treated faeces, wastewater, animal waste slurry and sludges. Studies on the survival of viruses in human fecal wastes and agricultural animal wastes have indicated that persistence is dependent on several factors, including the virus type, waste type, temperature and other environmental conditions and processes. In both human and animal waste treatment processes virus pathogen concentrations are reduced at various rates and to various extents depending on conditions, but they are not completely eliminated. Furthermore, they may be enriched in certain treated or separated waste fractions (such as waste solids) by sedimentation and solids-liquids separation processes {Strauch, 1991 #248}.

Studies by Pesaro *et al*., concluded that viral persistence in animal wastes was highly dependent on virus type, waste type and environmental conditions. Depending on ambient temperature, pH, and type of animal waste, time in day required for a 90% (1 $\log_{10}$) reduction of virus titer varied widely, ranging from less than 1 week for herpesvirus (an enveloped virus) to more than 6 months for rotavirus (a non-enveloped virus). Virus inactivation progressed substantially faster in liquid cattle manure, a mixture of urine and water (pH > 8.0), than in semiliquid wastes that consisted of mixtures of faeces, urine, water, and bedding materials (pH < 8.0). Overall, non-enveloped viruses such as encephalomyocarditis virus, bovine rotavirus, bovine adenovirus and bovine parvovirus were more persistent than an enveloped virus, with 1 $\log_{10}$ reduction times ranging from 9.4 to 197 days, depending on waste source and virus type. Bovine herpesvirus was reduced by 1 $\log_{10}$ within 2.5 days {Pesaro, 1995 #236}.

Other studies also report survival times of up to months for non-enveloped viruses. Fu et al. (1989) reported that a cytopathic porcine rotavirus (British isolate SW20/21) kept at room temperature for four months survived with titers reduced by 2 $\log_{10}$. Foot and Mouth Disease Virus, a picornavirus, can survive for 100 days in fecal slurry (Bartley *et al*., 2002). Other studies also report the survival of enveloped viruses for periods of up to days or weeks. Pseudorabies virus was persistent in swine urine, lagoon liquid, and waste pit effluent for 2 weeks, 2 days, and <1 day, respectively {Schoenbaum, 1990 #250}. Classical swine fever virus, member of the Flaviviridae family and Pestivirus genus, can survive for 15 days or more in manure, with inactivation occurring more rapidly in the liquid phase of slurry than in the solid phase (Have, 1984).

In biologically treated sludges from municipal wastewater plants using extended-aeration or oxidation ditch processes, indigenous viruses have been shown to survive up to 35 days at 5º C {Berg, 1988 #256}. Hepatitis A virus has been shown to resist drying and storage for at least 1 month in faeces {McCaustland, 1982 #274}. At 4ºC, poliovirus persisted in sand saturated in septic liquor for 180 days with <2 $\log_{10}$ reduction {Yeager, 1979 #376}. Under conditions of higher temperature, drying conditions, or suspension in groundwater, persistence was not as great.

Studies on virus survival in undisturbed soil cores which had been amended with sewage sludge have indicated that enteroviruses may survive for up to 35 days depending on temperature and moisture conditions {Bitton, 1984 #267}. Polioviruses have been shown to persist within septic tank sludge for at least 100 days {Stramer, 1984 #400}. In sewage sludge samples and sewage sludge eluates, indigenous viruses have been shown to remain stable (less than 1 log10 reduction) for up to 27 days at room temperature, and at temperatures of 2º C or -70º C viruses were shown to be stable for greater than 160 days {Hurst, 1986 #258;Hurst, 1986 #259}.

JC virus (JCV) particles have been detected in urban sewage of diverse geographical areas and some JCV isolates from sewage are infectious (Bofill-Mas and Girones, 2003; Bofill-Mas et al., 2003). The oral route could be significant for transmission of JCV infections because JCV virions have demonstrated relative resistance in the environment and to some of the conditions present in the gastrointestinal tract. It has been suggested that the archetypal strains commonly detected in the environment may be implicated in the transmission of JCV among the population.

Mineral Water: In mineral water, HAV and poliovirus 1 have been demonstrated to persist for up to 1 year at 4^{o}C, while at room temperature poliovirus persisted for 300 days and HAV remained infective longer {Biziagos, 1988 #330}. Noroviruses have also been found to persist in mineral water {Beuret, 2000 #570;Beuret, 2002 #569}.

Other Environmental Liquid Media: Virus survival in mock solid waste leachates has been shown to be temperature dependent with considerable survival at temperatures of 4ºC (<1 $\log_{10}$ reduction of poliovirus in 27 days), lesser survival at 20º C (~2 $\log_{10}$ reduction in 27 days), and least survival at 37º C (>2 $\log_{10}$ within 6 days) {Sobsey, 1975 #297}. However, greater inactivation in mock leachates than in glycine controls incubated at the same temperature indicates factors other than temperature also affect virus survival in solid waste leachates {Sobsey, 1975 #297}.

In saliva, HIV may persist either wet or dry for up to 7 days {Barre-Sinoussi, 1985 #340}. Studies examining the survival of Pseudorabies virus in buffered solutions found the virus was stable for at least 10 days in PBS and saline solution {Schoenbaum, 1990 #250}.

Exposure to blood containing viruses, such as Hepatitis B Virus, Hepatitis C Virus and HIV, and other pathogens is a risk in healthcare settings and in a variety of other environmental settings and exposure circumstances (Sattar and Springthorpe, 1991; Sattar et al., 2001). Studies by van Bueren et al. (1994) found that cell-free and cell-associated human immunodeficiency virus cultures suspended in 10% serum remained infectious for several weeks at room temperature. Virus stability was further increased when cell-associated virus was suspended in neat serum. Therefore, even enveloped viruses in blood can remain infectious for long periods of time. Weber et al. (1999) assessed the virucidal activity of three disinfectants (sodium hypochlorite, a phenolic, and a quaternary ammonium compound) in the presence and absence of blood against vaccine strain of poliovirus type 1 (prototype for relatively resistant hydrophilic viruses) and herpes simplex virus (HSV) type 1 (prototype for relatively susceptible lipophilic viruses). Viral survival was tested at room temperature and times of 0.25. 0.5, 1, 2, 5 and 10 minutes. In the absence of blood, complete inactivation of HSV was achieved within 30 seconds with 5,000 (1:10 dilution of bleach) and 500 (1:100 dilution of bleach) ppm chlorine, 1:10 and 1:128 diluted phenolic (use dilution), and 1:10 and 1:128 diluted quaternary ammonium compound (use dilution). In the presence of 80% blood, only 5,000 ppm hypochlorite, 1:10 phenolic, and 1:10 or 1:128 quaternary ammonium compound were effective. In the absence of blood, complete inactivation of poliovirus was achieved within 30 seconds by 5,000 and 500 ppm chlorine and

1:10 quaternary ammonium compound. In the presence of 80% blood, no solution tested was capable of completely inactivating poliovirus within ten minutes. It was recommended that hypochlorite at a final concentration of 5,000 ppm (1:10 dilution) should be used to decontaminate blood spills.

Survival Studies in Aerosols

Table 4 summarizes some data for human and animal virus survival in aerosols under various conditions. Additional study descriptions are included below by typical viral transmission routes.

Laboratory studies: The following generalizations can be made about aerosol survival of viruses: 1) viruses with lipids in their outer coat or capsid are more stable at low relative humidity (RH) than at high RH, 2) viruses without lipids are most stable in aerosols at high RH than at low RH, 3) when viruses can no longer be detected after aerosol generation, viral nucleic acid can be isolated and may still be active (suggesting inactivation occurs by altering of coat proteins), 4) pre-humidification upon sampling increases the recovery of non-enveloped viruses {Mohr, 1991 #454}. The mechanisms of virus inactivation in aerosols are poorly documented. However, two mechanisms of inactivation have been proposed for bacteriophage T3 in aerosols: 1) at high relative humidity surface inactivation, and 2) at low humidity removal of structural water molecules {Trouwborst, 1974 #299}.

Several groups report biphasic inactivation kinetics for aerosolized viruses {Hinshaw, 1976 #295;Schaffer, 1976 #296;Larson, 1970 #230;Songer, 1967 #318}. These inactivation kinetics can be explained by an initial rapid loss of viral titer as the aerosol is generated and as the newly formed aerosol is dried and equilibrates with the environment. The second phase is a more gradual long-term inactivation rate once the aerosol has been generated and become stabilized in the airborne state{Schaffer, 1976 #296}. At 24ºC and 35% relative humidity, murine leukemia virus demonstrated an initial 2 $\log_{10}$ inactivation during aerosolization. However, the virus was relatively stable for at least 35 minutes after stabilization of the aerosol, showing inactivation rates of 1% per minute {Larson, 1970 #230}. At lower relative humidity, reovirus demonstrates a rapid initial inactivation followed by a more gradual inactivation rate {Adams, 1982 #460}.

Early studies showed the survival of detectable amounts of vaccinia virus, Venezuelan equine encephalitis virus (VEE), and influenza A virus in aerosols for up to 23 hours under various temperature (10, 22, and 32ºC) and relative humidity (20, 50, and 80 %) conditions {Harper, 1961 #322}. Poliovirus was also examined in these studies, but only at 22º C. Vaccinia could be detected at 23 hours under all temperature and humidity conditions tested. Survival of VEE in detectable amounts was also documented at 23 hours under all conditions but the highest humidity and highest temperature. Trace levels of influenza also survived for 23 hours at low temperature and any humidity, and at higher temperatures at low humidity. VEE and influenza could be detected in all other samples for greater than six hours. Greatest survival of vaccinia, VEE and influenza was documented at low temperature and low humidity, with 66%, 26%, and 61% remaining at 23 hours, respectively. Poliovirus demonstrated a different survival pattern than the other viruses tested, showing greatest survival under high humidity. Other early studies on poliovirus and influenza virus in aerosols also found different survival patterns of the two viruses with regard to relative humidity {Hemmes, 1960 #423}. While poliovirus was still found to be most stable at high humidity, rapid decay below 50% humidity was not observed.

Pigeon pox virus has been shown to be stable in aerosols for up to five hours with >70% of virus remaining regardless of humidity {Webb, 1963 #321}. Rous Sarcoma virus was shown to be

most stable in water aerosols at high humidity (>60%) with up to 85% of virus remaining at five hours {Webb, 1963 #321}. Below 60% humidity, inactivation of RSV was greater.

In a study examining the aerosol stability of Newcastle Disease Virus, vesicular stomatitis virus, infectious bovine rhinotracheitis virus, and bacteriophages T3, loss of infectious titer during generation of aerosols has been shown to be consistently less at 90% relative humidity (RH) than at 10% or 35% RH for all viruses {Songer, 1967 #318}. However during aerosol aging at 23ºC, Songer demonstrated that Newcastle disease virus and vesicular stomatitis virus survived best at low humidity (10%). On the other hand infectious bovine rhinotracheitis virus and bacteriophages T3 survived aerosol aging better at 23ºC and 90% humidity. In another study examining the effect of temperature and relative humidity on the survival of infectious bovine rhinotracheitis virus, the virus was shown to be the most persistent in stabilized aerosols under conditions of high relative humidity and low temperature {Elazhary, 1979 #290}. In contrast, Donaldson and Ferris found IBR virus to be less stable at high relative humidity and room temperature {Donaldson, 1976 #439}.

Three strains of New Castle Disease Virus (NCDV) were found to survive best at high humidity (>70%). At least 1% of virus from each strain could be detected 4 hours after aerosolization. One strain persisted in detectable amounts for up to 16 hours post-aerosolization {Hugh-Jones, 1973 #300}. Using a micro-thread technique developed by May and Druett, survival of NDV was also documented for up to 1 hour in open air with variable humidity and wind conditions {Hugh-Jones, 1973 #300;May, 1968 #429}.

Mayhew, et al. found that the survival of Yellow Fever Virus in aerosols for up to 60 minutes was not significantly affected by temperature or humidity. However, a slight decrease in survival was noted at the 27º C and 50% humidity {Mayhew, 1968 #324}.

Aerosolized Semliki Forest Virus has been shown to survive best (<1 $\log_{10}$ inactivation after 24 hours) at low humidity (20%){Benbough, 1969 #309}. Inactivation increased above 50% relative humidity and was greatest (5 $\log_{10}$ inactivation in less than 24 hours) at high relative humidity (90%).

Bovine rotavirus in aerosols has been shown to survive best under high relative humidity and low temperature conditions{Moe, 1983 #273;Ijaz, 1985 #265}. However, simian rotavirus SA11 was reported to be most stable at intermediate (50%) relative humidity{Sattar, 1984 #269}. SA11 also is stable at low (25 %) humidity, but at high humidity (80%) SA11 is rapidly inactivated (no infectious virus detected at 24 hours post-aerosolization). Other experiments documented detection of infectious SA11 in aerosols after as long as 223 hours {Sattar, 1984 #269}. A similar inactivation pattern in relation to relative humidity has been demonstrated for human and calf rotaviruses {Ijaz, 1985 #265;Ijaz, 1985 #263}. The apparent inconsistencies of more recent results for rotavirus survival in aerosols compared to previous studies by Moe and Harper, may be explained by methodological differences including a longer sampling period, a longer aerosol stabilization period and perhaps differences in the strain of virus used {Ijaz, 1985 #265;Moe, 1983 #273}.

Reovirus may be shed in two forms, infectious virions (IV) and potentially infectious virions (PIV). Aerosol survival studies have shown that both forms are stable at high relative humidity {Adams, 1982 #460}. Other studies have examined the aerosol survival of IV and PIV for multiple strains of reovirus (Type 1 Lang, Type 2, Jones, and Type 3 Dearing) at three temperatures and four relative humidities {Mohr, 1984 #461;Mohr, 1991 #454}. These studies

found greater inactivation at intermediate RH (40-60%). Generally inactivation of IV was greater for type 2 and type 3, whereas inactivation of PIV was greater for type 1.

Rhinovirus 14 in aerosols was found to persist well at high relativity humidity, with 30% of virus remaining infectious after 24 hours at 20ºC {Karim, 1985 #262}. At low and intermediate humidity, rhinovirus was rapidly inactivated.

Human coronavirus (229E) has been demonstrated to be very persistent in aerosols {Ijaz, 1985 #261}. After 6 days in aerosol at 20ºC and 50% relative humidity, 20% of human coronavirus was demonstrated to be infectious.

Studies have shown that despite the loss of infectivity of Picornaviruses in aerosols, intact and active nucleic acid may be recovered. Although poliovirus is rapidly inactivated at low relative humidity, active nucleic acid may still be extracted and free RNA may remain active for a short period {de Jong, 1970 #459}. Similarly, after the infectivity of encephalomyocarditis virus was lost, active nucleic acid could be recovered {de Jong, 1970 #459;de Jong, 1974 #452}.

Survival Studies on Surfaces

Table 5. summarizes some data for human and animal virus survival on surfaces under various conditions. Additional study descriptions are included below by typical viral transmission route.

Enteric viruses: In studies of surface survival, poliovirus was found to persist at up to 20 weeks on wool blanket fabric {Dixon, 1966 #350}. Survival of poliovirus was not as great on wool gabardine fabric or on cotton fabrics. An initial rapid inactivation of poliovirus was documented followed by a more gradual rate of decline. FMD, a picornavirus of animals, has been found to persist on wool at detectable levels for at least 31 days at 4º C {McColl, 1995 #235}. Inactivation of FMD on wool was shown to be inversely related to temperature and at 18º C, FMDV was shown to persist as long as 14 days. Other studies have documented the survival of enteroviruses for 2-12 days on painted wood, glass, and cotton fabric {Kiselva, 1968 #474}. At 20º C enterovirus 70 has been shown to persist best on surfaces at very high relative humidity (95%). The inactivation rate increases with decreasing humidity and at low humidity the virus is quickly inactivated, >2.5 $\log_{10}$ reduction per hour. At higher temperatures, inactivation rates greatly increased, even at high (80%) relative humidity. However extreme high (>95%) relative humidity was still protective of enterovirus infectivity. Similar results have been shown for coxsackievirus B3 virus as well.

Hepatitis A is persistent on environmental surfaces, with recovery of infectious virus documented at 96 hours from stainless steel {Mbithi, 1991 #249}. Infectious HAV has been recovered from polystyrene surfaces after 1 month {Sobsey, 1988 #476}. HAV has also been shown to survive well in faeces {McCaustland, 1982 #274}. HAV survival on skin has also been examined. A rapid initial inactivation associated with drying of the inoculum (0.5 $\log_{10}$) was observed within the first 60 minutes, after which the virus was relatively stable {Ansari, 1991 #247}. A study on the persistence of a number of taxonomically different virus types found that HAV and rotavirus when dried on fomite surfaces are generally more resistant to inactivation than poliovirus or enteric adenovirus {Abad, 1994 #239}.

In a one-week parallel study, astrovirus type 2 was shown to persist longer than Adenovirus or poliovirus on surfaces {Abad, 2001 #229}. However it was not as persistent as HAV and rotavirus. Lower temperature (4ºC, compared to 20ºC) has been shown to improve the survival of astrovirus and adenovirus, but not of rotavirus. Astrovirus is sensitive to high humidity,

especially at 20ºC. Fecal matter was shown to be somewhat protective of astrovirus infectivity against high humidity at 4ºC.

The survival of human rotavirus has been examined on several surfaces, including stainless steel, glass, plastic, cloth, paper, and the hands of volunteers {Sattar, 1986 #260;Ansari, 1988 #255;Keswick, 1983 #270;Moe, 1983 #273}. Keswick et al. showed that rotavirus SA11, poliovirus type 1 and bacteriophage f2 were capable of surviving drying on countertop surfaces between 45 to 90 minutes {Keswick, 1983 #270}. The survival of purified human rotavirus has been shown to be similar to that of purified bovine rotavirus when dried onto coverslips and maintained at 20ºC and 75% relative humidity {Moe, 1983 #273}. However, human rotavirus in faeces was significantly more persistent. In more recent studies, rotavirus has been found to be very stable on non-porous surfaces, with inactivation of less than 1 $\log_{10}$ in ten days, depending on relative humidity {Sattar, 1986 #260}. At low and intermediate relative humidity, rotavirus infectivity was reduced <1 $\log_{10}$ on all surfaces examined after 10 days. Rotavirus can also persist on non-porous surfaces at 36º C for up to six days {Sattar, 1986 #260}. The survival of human rotavirus has also been studied on the hands of volunteers {Ansari, 1988 #255}. At 60 minutes after inoculation, infectious virus was reduced by 0.4 $\log_{10}$, while after 260 minutes, infectious virus was reduced by 1.1 $\log_{10}$. Unlike results from studies on non-porous surfaces, results on the survival of human rotavirus on porous surfaces were variable, depending on the environmental surface and exposure conditions {Sattar, 1986 #260}. No infectious virus could be recovered from contaminated poster card. However, infectious rotavirus could occasionally be recovered from experimentally contaminated writing paper and paper currency. Recovery from cotton cloth was highly variable, but survival to ten days at 4ºC was documented in one set of experiments.

A number of studies have reported virus survival on foods. Croci *et al*. (2002) studied the survival of HAV on vegetables that has been experimentally surface-contaminated with HAV and stored at 4^{o} C for up to nine days. Virus was consistently detected on lettuce, with only a 2 $\log_{10}$ decrease over time. Greater decreases were observed for carrots and fennel, with complete inactivation at day 4 and 7, respectively. In a recent study, poliovirus survival was determined on fresh produce stored at refrigeration temperature for 2 weeks (Kurdziel et al., 2001). D-values (time for 90% or 1 $\log_{10}$ reduction) were: lettuce, 11.6 days; green onion, no decline; white cabbage, 14.2 days; fresh raspberries, no decline; and frozen strawberries, 8.4 days. Therefore, enteric viruses such a poliovirus can persist on fresh fruits and vegetables for at least several days under conditions commonly used for storage in households. FMDV can persist for 105 days in hay and 140 days in bran (Bartley, 2002).

Enveloped viruses also have been shown to persist in foods. Classical swine fever virus (Flaviviridae family, Pestivirus genus), like many enveloped viruses, is considered relatively labile in the environment. It shows short but variable survival times in the environment, depending on physical conditions. For example, Edwards, summarizing data from studies by Slavin (1938), notes that bricks or chopped hay, exposed to air but protected from direct sunlight and rain, still contained infectious classical swine fever virus (Family Flaviviridae, genus Pestivirus) after 7 days but not after 14 days (Edwards, 2000). Classical swine fever virus can persist for long periods in the cold, is relatively stable between pH 3 and 11 and is partially resistant to a moderate temperature of 56^{o}C. The virus can remain viable for prolonged periods in favorable circumstances as found, for example, in stored meat, and it can survive smoking and curing procedures (Edwards, 2000; Panina *et al.,* 1992).

Respiratory viruses: Rhinovirus type 39 has been shown to persist for up to 72 hours non-porous surfaces {Hendley, 1973 #357}. Infectious rhinovirus has also been recovered for up to three

hours from porous surfaces, such as natural and synthetic fabrics, and paper products. Survival of rhinovirus on skin has also been documented up to 3 hours {Hendley, 1973 #357}. Rhinovirus in nasal secretions has been shown to be inactivated by 0.4 to 2 $\log_{10}$ when dried on skin or other surfaces {Reed, 1975 #365}. Sattar et al. have examined the survival of human rhinovirus 14 on stainless steel discs under various conditions of relative humidity {Sattar, 1987 #372}. Rhinovirus was found to survive best at high relative humidity, with 1.2 $\log_{10}$ reduction after 24 hours when suspended in tryptose phosphate buffer. A similar pattern of inactivation was observed when rotavirus was applied to stainless steel surfaces in bovine mucin, however rates of inactivation were initially much higher. Bovine mucin was found to better approximate nasal secretions and results were more consistent with those of previous studies {Buckland, 1962 #470; Reed, 1975 #365}.

Influenza (PR-8 strain) has been demonstrated to survive for several weeks on dust, cotton sheets, and glass slides at 22ºC {Edward, 1941 #466}. Survival of influenza has also been studied on skin and glass slides up to 200 minutes {Parker, 1944 #467}.

Respiratory syncytial virus (RSV) suspended in minimal essential media or pooled adult nasal secretions can survive up to 8 hours on countertops, 5 hours on rubber gloves, 2.5 hours on cloth gowns, and 1 hour on paper towels or skin {Hall, 1980 #356}. The pattern of survival for RSV in nasal secretions obtained from confirmed RSV infected infants on the same surfaces was similar to seeded virus in negative nasal secretions, but of shorter duration {Hall, 1980 #356}. Respiratory syncytial virus in tissue culture homogenate was shown to be reduced by 1 $\log_{10}$ when dried on polythene surfaces and was reduced another 1 log10 after 24 hours {Kingston, 1968 #468}.

Human parainfluenza viruses types 1, 2, and 3 have been shown to persist at room temperature on a plastic surface under dry conditions for periods of up to four, seven and twelve days, respectively {Parkinson, 1983 #362}. At temperatures of -22 to -33º C (midsummer conditions at South Pole), parainfluenza virus type 1 survival on plastic was as long as 7 days, while the survival of types 2 and 3 was as long as 17 days. Parainfluenza types 1, 2, and 3 were found to persist similarly on non-porous and porous surfaces {Brady, 1990 #251}. Viruses persisted for at least 10 hours on non-porous surfaces as long as the surface remained moist. When surfaces dried, infectious virus was still recoverable at 2 hours. On adsorptive surfaces, parainfluenza viruses could be recovered up to 4 hours post-inoculation. Drying of the material appeared to be associated with increased viral inactivation. Survival of parainfluenza viruses for 1 hour on skin has also documented {Brady, 1990 #251;Ansari, 1991 #247}.

Human coronavirus has been shown to survive up to six days in saline solution, but was found to survive for a shorter period in culture medium with or without added cells{Sizun, 2000 #572}. After drying on surfaces at 21ºC and 55-70% relative humidity, strain HCoV-229E survived for up to 3 to 6 hours with reductions of 1-2 $\log_{10}$ on aluminum foil, latex gloves, and sterile sponges{Sizun, 2000 #572}. Under similar conditions, strain HCoV-OC43 persistence was <1 to 2 hours with for similar (1-2 $\log_{10}$) reductions. Porcine reproductive and respiratory syndrome virus (PRRS) is a member of the Arteriviridae from the order Nidovirales, like the Coronaviridae. PRRS appears to be very sensitive to drying on non-porous (plastic, stainless steel, and rubber) and porous fomites (alfalfa, wood shavings, denim cloth, corn, starter feed, and straw), as no virus could be recovered from any of these materials after the day of application {Pirtle, 1996 #358}. However, the virus did however persist from 9 to 11 days in water.

Viruses infecting by the ocular route: At room temperature, adenovirus type 19 has been shown to survive on porous (cloth and paper) and non-porous (metal and plastic) for 7-35 days post

inoculation {Nauheim, 1990 #354}. Maximum survival was demonstrated on plastic. Various strains of adenovirus types 5, 8, and 19 have been shown to persist on metal and plastic surfaces for up to 49 days {Kowalski, 1998 #352}. Inactivation was greatest during the first 7 days and the rate of inactivation declined after 14 days. Adenovirus type 3 and echovirus type 1 have been shown to persist for up to ten and 30 days, respectively, on dried films of adhesive gum on envelopes {Selwyn, 1965 #379}.

Bloodborne viruses: Favero et al. examined the inactivation of hepatitis B antigen in blood on stainless steel and cotton surfaces and found hepatitis B titers to be stable (<0.5 $\log_{10}$ reduction) for up to 14 days {Favero, 1974 #343}. Studies have shown that hepatitis B virus retains its infectivity for up to seven day even when dried in human plasma {Bond, 1981 #341}. Differences have been documented in the survival of cell-free and cell-associated HIV diluted on surfaces{van Bueren, 1994 #242}. Cell free virions were found to be much more persistent than cell-associated virus when dried onto glass coverslips at room temperature, with >70 hours for 1 $\log_{10}$ inactivation of the former, as compared to 1 $\log_{10}$ within 18 hours for the latter. As previously noted in an earlier section of this report, HIV infectivity in liquid suspensions was generally more persistent than viruses dried on surfaces and cell-associated virions were found to be more persistent than cell-free virions. The inactivation of HIV on coins at room temperature is thought to be due partially to drying and partially to metallic nature of the coins.

Dermal Route: A number of studies have documented the survival of herpes simplex virus type 1 on human skin for at least 2 hours {Bardell, 1989 #339;Turner, 1982 #475}. Herpes simplex virus type 1 has been shown to be reduced by 1.5 $\log_{10}$ on plastic door handles and chrome plated wash basin tap handles over a two -our period {Bardell, 1990 #338}. Herpes Simplex Virus has been demonstrated to survive on plastic surfaces for less than 6 hours in a humid atmosphere at 37 – 40º C {Nerurkar, 1983 #361}. Herpes simplex virus type 2 from genital lesions has been shown to persist on fomite surfaces for as long as 72 hours {Larson, 1985 #355}. Gauze was the surface on which the virus was shown to be most persistent. Survival on toilet seats was up to 4 hours. Herpes simplex type 2 was found to persist better on skin than metal {Graham, 1996 #479}. HSV2 persisted on skin for up to 90 minutes. Herpes simplex virus and vaccinia viruses have been isolated from floor sweepings for up to 2 weeks after patients with generalized eruptions vacated the room {Kibrick, 1959 #469}. Vaccinia has been shown to persist on wool for up to 14 weeks at 25ºC under conditions of low humidity (35%) {Sidwell, 1966 #464}. Vaccinia was also recovered from cotton, although for shorter time periods (six to twelve weeks, depending on fabric type).

The survival of Pseudorabies virus was examined on the surface of a range of materials commonly found in swine-raising environments {Schoenbaum, 1990 #250}. Pseudorabies virus was found to be relatively stable in saline solution (up to ten days with less than 2 $\log_{10}$ inactivation) on both whole corn and steel. On polypropylene, vinyl, loam soil, and meat and bone mea, virus inactivation rates were more rapid with a 7 $\log_{10}$ reduction within seven days. For straw, concrete, and pelleted feed, virus dropped below detectable levels within four days. For wood, faeces, green grass, alfalfa, and denim fabric, virus dropped to below detectable levels in one to two days.

Purified human and bovine papilloma viruses dried in tubes have been shown to persist up to one week at room temperature {Roden, 1997 #367}. Bovine papilloma virus and human papilloma virus 16 were also been show to be relatively thermo-stable, surviving treatment at 56º C for one hour, unlike cottontail rabbit papillomavirus and HPV-11, which were more labile {Bonnez, 1994 #480;Roden, 1997 #367}.

The SARS Associated Coronavirus (SARS-CoV) and the Environment

The virus associated with sudden acute respiratory syndrome has been identified as a novel coronavirus {Ksiazek, 2003 #381;Drosten, 2003 #382}. As previously noted, Coronaviruses are circular to pleoporphic enveloped viruses with a positive sense, single stranded RNA genome, a helical nucleocapsid and they are about 120-140 nm in size. Coronaviruses have previously been associated with both respiratory and enteric illnesses in a range of animals. Infection for the SARS-CoV appears to occur primarily by the respiratory route, but a fecal-oral route of infection may also be possible. Epidemiologic evidence suggests that the major mode of transmission for SARS-CoV is by close personal contact with an infected individual, by droplet secretions (Seto et al., 2003){Poutanen, 2003 #383}. However, exposure to bioaerosols or contact with environmental surfaces contaminated with respiratory secretions or other bodily fluid (e.g. urine or faeces) may also play a role in transmission {Tsang, 2003 #386}.

Concern over the environmental transmission of SARS-CoV arose after clusters of cases were recognized in a Hong Kong hotel and in the Amoy Gardens apartment, and it was further heightened after other buildings with multiple cases were identified. SARS-CoV has been isolated from faeces, urine, and respiratory secretions of infected individuals {Peiris, 2003 #385}. Additionally, SARS-CoV has been isolated from a number of animal samples, including the Palm Civet, indicating a possible animal reservoir or source {Stein, 2003 #388}. Further, SARS-CoV has been detected in number of environmental samples including: a swab of the toilet rim in the flat of an infected individual; surface and gut swabs of cockroaches; rodent droppings; throat and anal swabs of cats and dogs; the soil stack on the rooftop of a building with multiple cases; and swabs from the bathroom floor, internal surface of balcony window, and wall between kitchen and bathroom in rooms adjacent to the room of an infected individual in a building with multiple cases {Tsang, 2003 #386}.

Prevailing hypothesis for environmental transmission in the Amoy Gardens building and other nearby buildings are that: (1) virus-contaminated sewage droplets were sucked back into bathrooms by powerful bathroom fans through dried-up floor drains, then escaped through windows and rose as a plume in a narrow light well (chimney effect); (2) passive carriage by pests, such as cockroaches and rodents; and (3) fecal-oral transmission through contact with contaminated surfaces and other fecally contaminated vehicles and fomites. Recently another hypothesis has been proposed: human infection of roof rats with subsequent spread by infected rat vectors (Ng et al., 2003). None of these hypotheses are mutually exclusive and potentially all of them could be correct. However, there are inadequate data to fully elucidate, document and characterize the roles of various indirect personal contact, zoonotic and environmental transmission routes for the SARS Co-V.

Very little is known about the survival, persistence environmental fate of SAR-CoV. Information is very limited on the environmental stability of coronaviruses in general. Laboratory studies on the environmental stability of the SARS-CoV and its susceptibility to various disinfectants are currently be performed by the WHO Laboratory Network. Initial results as summarized on the WHO web site are presented in Table 6. However, initial results are difficult to interpret because of inadequately described methods and the format in which results are presented. No final virus concentrations were reported so survival at the indicated time can not be fully interpreted on a quantitative basis. However, the SARS-CoV appears to survive in urine for at least 1 day, while in fecal samples it may persist for up to 4 days {WHO, 2003 #387}. Stability in virus culture medium is much greater, with persistence of greater than 21 days being reported {WHO, 2003 #387}. The virus appears to be more stable at higher pH levels. The stability of the virus appears to be inversely related to temperature (i.e. the virus is relatively stable at temperatures $\leq 4°C$ and

is readily inactivated at temperatures above 37°C) {WHO, 2003 #387}. The SARS-CoV appears to be susceptible to laboratory fixatives, like acetone and formaldehyde, and is readily inactivated by alcohol and bleach {WHO, 2003 #387}. On surfaces, the virus is typically more stable in sterilized stool than in PBS (or tissue culture medium) {WHO, 2003 #387}. Survival on surfaces is greater than or equal to the survival expected in stool or culture medium {WHO, 2003 #387}. Anecdotal evidence indicates that viral genomic material may be detected in environmental samples, such as carpets, despite disinfection efforts{Tsang, 2003 #386}. This is consistent with previous studies indicating that RT-PCR may detect inactivated viruses {Sobsey, 1998 #568}.

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

A variety of different kinds of viruses, both enveloped and non-enveloped and representing many taxonomic groups, are likely to be present in human and animal fecal wastes, combined fecal and urine wastes (sewage, nightsoil, slurry, etc.), and respiratory secretions entering sewage and other domestic waste streams. New viruses, such as the SARS Coronavirus, Hantavirus Pulmonary Syndrome Virus (Bunyaviridae family) and Nipah Virus (Paramyxoviridae family), continue to be recognized in human and in animal populations and the host ranges of many of these viruses are uncertain. There are concerns that some of these recently discovered animal viruses do infect or may be able to infect humans or that they have the potential to recombine with human and other animal viruses and produce new virus strains capable of infecting humans or having greater potential to cause human infection and illness. Examples of viruses of particular concern in this regard include the SARS Coronavirus, hepatitis E virus, Hantavirus Pulmonary Syndrome virus, caliciviruses, orthhomyxoviruses (influenza viruses) and paramyxoviruses (such as Nipah Virus).

The persistence of enteric and respiratory viruses in various fecally contaminated environmental media has been studied, but reliable quantitative information is lacking for many of the recently recognized and emerging viruses, especially in certain environmental media and under certain environmental conditions that represent potential reservoirs and vehicles of transmission. Therefore, there are considerable uncertainties about the extent to which some human animal viruses survive in the environment, are released into the environment and are available to be transported for further human or animal exposure, contact and transmission.

The survival times and kinetics of inactivation of some human and animal viruses in various environmental media under certain environmental conditions has been determined, primarily in laboratory and less frequently in pilot scale or field studies. Further studies are recommended to better characterize virus survival times and inactivation rates, particularly for the newly recognized and emerging viruses of concern. Of particular importance are determining the media and environmental conditions that lead to or facilitate virus persistence for periods of time that provide for their further transmission, identifying media and conditions where virus reductions occur and quantifying the rates and extents of virus reductions in specific environmental media and under specific environmental conditions.

In general, many different kinds of viruses can persist in and on environmental media, including liquid and solid media and in the airborne state, with half-lives of hours, days, weeks or even months. The extent of persistence depends on the type of virus, it physical state (dispersed, aggregated, cell-associated, membrane-bound, adsorb to other solids, etc.), the medium in which it is present (faeces, respiratory secretions, tissues, other liquids or solids, air, etc. and prevailing environmental conditions that influence virus survival. The environmental conditions influencing virus survival generally include: temperature; pH and other physical and chemical properties of the medium in which the viruses are present, such as moisture content, organic matter, particulates, salt concentration, protective ions, and antiviral chemicals such as proteolytic

enzymes; antiviral microbial activity, and light. On environmental surfaces and in aerosols additional environmental factors also influence virus survival, such as relative humidity and physico-chemical forces at air-water and air-water-solid interfaces.

Quantitative information is needed on the survival, persistence and inactivation rates of many enteric and respiratory viruses, including the SARS Coronavirus, in various sources of fecal and respiratory material at ambient environmental conditions in wet states (as faeces, sewage, respiratory secretions, etc.), when subjected to drying and desiccation, when deposited on various porous and non-porous surfaces, when aerosolized, when subjected to mesophilic and thermophilic biological processes in both natural environments and in engineered treatment systems, when subjected to chemical treatments such as germicides, disinfectants and other waste treatment chemicals (e.g., lime or other alkaline material treatments), and when subjected to physical process such as sedimentation, filtration, UV irradiation, sonication and pressure changes, shearing forces and extreme changes in temperature.

Also needed are definitive, reliable, robust and quantitative methods to recover and detect viruses in human and animal fecal wastes and fecally contaminated environmental media, especially for enveloped viruses such as the SARS Coronavirus. Such methods are needed to determine virus presence in fecal and other wastes and in fecally contaminated environmental media and the extent to which these viruses are transported, removed or inactivated under natural environmental conditions or in waste treatment processes and management systems. The development, evaluation and application of reliable, sensitive and affordable methods to recover and detect human and animal viruses in fecal wastes and in fecally contaminated environmental media is recommended.

In principle, methods are available to recover and detect some human and animal viral pathogens as well as indicator viruses, such as somatic and male-specific coliphages, in human and animal wastes and fecally contaminated environmental media. However, these methods have not been applied to some of the viruses of interest and concern, such as the SARS Coronavirus, and they have not been adequately verified and collaboratively tested in some of the fecal wastes and fecally contaminated environmental media of interest and concern. Such validation, verification and performance characterization studies are recommended. Also recommended are comparative studies on the removal, inactivation and fate of indicator viruses and human and animal viral pathogens in fecal wastes and fecally contaminated environmental media, including waste treatment processes and management systems. If such studies show that the indicator viruses reliably reflect or predict the responses of the human and animal enteric viruses, than it may become possible to use them in practical, rapid and affordable monitoring and surveillance activities to assess environmental survival and persistence, the presence of enteric viruses in environmental media, the efficacy of treatment processes and systems to reduce viruses, and to determine the virological quality of treated liquid and solid residuals, such as sewage effluents and biosolids.

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Table 1. SOME IMPORTANT FACTORS INFLUENCING VIRUS SURVIVAL IN FAECES AND FECALLY CONTAMINATED ENVIRONMENTAL MEDIA

Factor	Effects
Physical	
Heat or thermal effects	Increasing inactivation at higher temperature; pasteurize
Desiccation or drying	Increased inactivation at lower moisture content or relative humidity; effects of RH differ between enveloped and non-enveloped viruses
Aggregation	Clumping protects viruses from inactivating agents
Asdorption to particles or surfaces	Adsorption protect viruses from inactivating agents; some specific chemical surfaces (heavy metals) are virucidal
Encapsulation or embedding	Viruses within membranes or larger particles are protected from inactivation
Chemical	
Hydrogen ions; pH	Viruses survive best near neutral pH and worst at pH extremes
Organic matter	Many viruses are stabilized and protected by dissolved, colloidal and solid organic matter, including fecal organics and natural organic matter (humic materials)
Ammonia	NH_3 has virucidal activity; manifest at higher pH (>pH 8)
Salts and Ionic Strength	Increased concentrations of salts (e.g., sodium chloride) are antiviral for many viruses; some viruses are destabilized and inactivated by water lacking stabilizing salts (such as NaCl) ions such as Mg++
Enzymes	Proteases and nucleases contribute to virus inactivation
Biological	
Microbial activity	Biological treatment and microbial activity/metabolism in soils, sediments, water; several contributing mechanisms
Proteolytic activity	Proteolytic enzymes inactivate/denature virion proteins
Microbial predation	Engulfment, ingestion, etc. by protozoa, helminths, etc.
Biofilms	Virus adsorption to biofilms can be protective or microbial activity in biofilms can cause virus inactivation and degradation

Table 2. SOME IMPORTANT ANIMAL VIRUSES CAPABLE OF INFECTING HUMANS AND POTENTIALLY PRESENT IN HUMAN AND ANIMAL FAECES AND FECALLY CONTAMINATED WASTES
((NOTE: TABLE IS INCOMPLETE; WILL ADD MORE VIRUS FAMILIES)

Virus or Virus Group	**Family**	**Virion Properties***	**Animal Hosts? Y/N (animals)**	**Disease in Animal Hosts**	**Human Infection/ Human Disease**	**Trans-mission Routes**	**Presence in Faeces**
Entero-viruses	Picorna-viridae	NE, icos., 27 nm, ss +RNA	Yes (Bovine, porcine, avian)	Yes in some	Yes for human types maybe for others	Fecal-oral and respiratory	Yes
Calici viruses	Caliciviridae	NE, icos., 35 nm, ss +RNA	Yes (Bovine, porcine, avian)	Yes in some	No, but maybe	Fecal oral and respiratory	Yes
Reovir uses	Reoviridae	NE, icos, 75 nm, dsRNA, 10 seg.	Yes (Wide host range for some)	Some	Some yes, infection/ No illness	Fecal-oral; respiratory	Yes
Rotavi ruses	Reoviridae	NE, icos, 75 nm, dsRNA, 11 seg.	Yes (Found in many animals)	Yes, some	No, but maybe for some	Fecal-oral; possibly respiratory	Yes
Adeno-viruses	Adenoviridae	NE, 70 nm, icos., ds L DNA	Yes (In many animals)	Yes, some	Unknown	Fecal-oral and respir-atory	Yes
Herpes-viruses	Herpesviridae	E, ~175 nm ~S/icos., ds L DNA	Yes, (In many animals)	Yes, some	Unknown	Respirator y	Yes
Myxo viruses	Orthomyxo viridae	E, ~100 nm, ~S&F/H, -RNA, 7-8 seg.	In many animals	Yes, some	Yes, some; No, others	Respirator y	Yes
Pestivi ruses	Filoviridae	E, 800 x 80, H/H, -ss RNA	In many animals	Yes, some	No,	Fecal-oral and respiratory	Yes, some
Coron	Coronavirid	E, 75-	In many	Yes,	Some Yes	Respirator	Yes

a-viruses	ae	160 nm, S/H, +ssRNA	animals	some	(SARS virus); Many No	y; possibly fecal-oral for some	
Hepatitis A Virus	Picornaviridae (Hepatovirus genus)	NE, 27 nm, S/icos., +ssRNA	No, except monkeys; perhaps other primates	Yes, experimentally	Yes		Yes
Hepatitis E Virus	Uncertain	NE, 32 nm, S/icos., +ssRNA	Swine, rats, chickens, deer, and others	Yes, but mild effects	Yes	Respiratory and enteric	Yes

E = enveloped, NE = non-enveloped, nm = nanometer, F = filamentous virion shape, S = spherical virion shape, H = helical virion shape or after a "/" = helical nucleocapsid, icos = icosahedral virion shape or after a "/", icosahedral nucleocapsid, ds = double-stranded, ss = single-stranded, L = linear, "+" = plus-sense nucleic acid, "-" = minus-sense nucleic acid, seg. = segmented.

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Biofilms	Virus adsorption to biofilms can be protective or microbial activity in biofilms can cause virus inactivation and degradation

TABLE 3. VIRUS SURVIVAL IN LIQUID AND SEMI-SOLD MEDIA, INCLUDING FAECES, SLUDGES AND SOIL MATERIALS

Virus	Medium	log10 reduction	Time	Temp	pH	Conductivity	Turbidity	Other Conditions	Citation
Rotavirus	Tapwater	0.7	64 days	4ºC	8.3 start (7.9 end)			10 fold bacterial growth over time	Sattar, 1984
Rotavirus	Tapwater	2	64	20ºC	8.3 start (7.9 end)			60 fold bacterial growth over time	Sattar, 1984
Rotavirus	Distilled Water	1	<1 hr						Sattar, 1984
NLV	GW and Sediment		4 weeks					anecdotal	McAnulty, 1993
Enterovirus	GW and Sediment		4 weeks					virus isolation from sediment following repair of leaking septic	McAnulty, 1993
Coxsackie A9	Stream Water	1.9	30 days	15ºC		7.9 7 (ms)		39 TPC bacteria 1.5E6	Nasser, 2003
Coxsackie A9	Stream Water	4	30 days	30ºC		7.9 8 (ms)		39 TPC bacteria 1.5E6	Nasser, 2003
Coxsackie A9	Brackish water	0.05	30 days	15ºC		8.5 13.9 (ms)		13.7 TPC bacteria 5.1E4	Nasser, 2003
Coxsackie A9	Brackish water	4.5	30 days	30ºC		8.5 13.9 (ms)		13.7 TPC bacteria 5.1E4	Nasser, 2003
Coxsackie A9	Seawater	2.2	30 days	15ºC		8.4 55.5 (ms)		1.7 TPC bacteria 48	Nasser, 2003
Coxsackie A9	Seawater	5.3	30 days	30ºC		8.4 55.5 (ms)		1.7 TPC bacteria 48	Nasser, 2003
Poliovirus Type 1	Seawater	1.5 - 4.0	4	24ºC		220,000-240,000 (µs/cm)		Increasing rate of reduction with increasing fecal contamination	Fujioka, 1980
Poliovirus Type 1	Fresh water	0.7	4	24ºC		470 (µs/cm)			Fujioka, 1980
Poliovirus Type 1	Estuarine water	2.2	4	24ºC		200,000 (µs/cm)			Fujioka, 1980
Poliovirus Type 1	PBS	0.5	4	24ºC		73,000 (µs/cm)			Fujioka, 1980
Coxsackie B4	Marine water	> 2.0	2						Fujioka, 1980
Coxsackie b4	PBS	stable	2						Fujioka, 1980
Echovirus 7	Marine Water	> 2.0	2						Fujioka, 1980
Echovirus 7	PBS	stable	2						Fujioka, 1980
Rotavirus	River Water	< 2	64 days	4ºC	7.2 start (7.8 end)		3-14 NTU	26-40 fold bacterial growth over time	Raphael, 1985
Rotavirus	River Water	2 - 3	64 days	20ºC	7.2 start (7.8 end)			>1000 fold bacterial growth over time	Raphael, 1985
Rotavirus	Tap Water	< 1	64 days	4ºC	8.4 start (7.8 end)			Total Cl 0.05 mg/L, Final TPC bacteria 47-98ml	Raphael, 1985
Rotavirus	Tap Water	1 - 1.5	64 days	20ºC	8.4 start (7.8 end)			Total Cl 0.05 mg/L, Final TPC bacteria upto 376ml	Raphael, 1985
Rotavirus	0.22 µm Filtered RiverWater	<0.5	64 days	4ºC	7.6 start (7.8 end)			No detectable TPC bacteria	Raphael, 1985
Rotavirus	0.22 µm Filtered RiverWater	0.1 - 0.3	64 days	20ºC	7.6 start (7.8 end)			No detectable TPC bacteria	Raphael, 1985
Aujeszky's Disease virus	ensilage	> 7	7			3.93			Martinez-Gamba, 2001
Blue eyed Disease virus	ensilage	> 7	7			3.7			Martinez-Gamba, 2001
Aujeszky's Disease virus	Pig Feces		3 - 15 weeks						Martinez-Gamba, 2001
African Swine Fever Virus	Pig Feces		60 days						Martinez-Gamba, 2001
FMDV	Pig Feces		21 - 103 days						Strauch, 1994
Pig Parvovirus	Pig Feces		14 weeks						Mengling, 1999
Poliovirus Type 1	Mixed Human and Pig Wastes		4 weeks						Ming, 1995
Hepatitis A virus	Mixed Human and Pig Wastes		5 weeks						Ming, 1995
Poliovirus Type 1	Mineral Water	5.25	270 days	23ºC ± 3ºC	7.0 start			total mineralization at 180ºC = 110 mg/L	Bitzlagos, 1988
Hepatitis A Virus	Mineral Water	5.25	330 days	23ºC ± 3ºC	7.0 start			total mineralization at 180ºC = 110 mg/L	Bitzlagos, 1988
Poliovirus Type 2	Mineral Water	1.16	1 year	4ºC	7.0 start			total mineralization at 180ºC = 110 mg/L	Bitzlagos, 1988
Hepatitis A Virus	Mineral Water	0.68	1 year	4ºC	7.0 start			total mineralization at 180ºC = 110 mg/L	Bitzlagos, 1988
Herpes Simplex Virus Type 1	Saliva on Chrome Tap Handle	3.1	2 hours	21 - 24ºC		7		Saliva Evaporated at ~40 minutes; accompanied by 2 log_{10} reduction	Bardell, 1993
Herpes Simplex Virus Type 1	Saliva on Plastic Doorknob	2.8	2 hours	21 - 24ºC		7		Saliva Evaporated at ~40 minutes; accompanied by 2 log_{10} reduction	Bardell, 1993
Herpes Simplex Virus Type 1	Saliva on Chrome Tap Handle	0.2	2 hours	21 - 24ºC		7		Saliva Maintained in Liquid State	Bardell, 1993
Herpes Simplex Virus Type 1	Saliva on Plastic Doorknob	0.2	2 hours	21 - 24ºC		7		Saliva Maintained in Liquid State	Bardell, 1993
Herpes Simplex Virus Type 1	Tap Water on Chrome Tap Handle	3.1	2 hours	21 - 24ºC		6.5		Water Evaporated at ~30 minutes; accompanied by 2 log10 reduction	Bardell, 1993
Herpes Simplex Virus Type 1	Distilled Water on Chrome Tap Handle	3.5	2 hours	21 - 24ºC		6.5		Water Evaporated at ~30 minutes; accompanied by 2 log10 reduction	Bardell, 1993
Herpes Simplex Virus	Distilled Water	> 3	24 hours	37 -40ºC	6.9 - 7.0			<0.1 ppm Cl; <0.1 ppm Br; steady reduction over 24 hours	Narukar, 1993
Herpes Simplex Virus	Tap Water	3	4 hour	37 -40ºC	6.7 - 6.85			4.0 ppm Cl; <0.01 ppm Br; ~2.5 log_{10} reduction within 30 min	Narukar, 1993
Herpes Simplex Virus	Spa Water	3 - 4	Immediate	37 -40ºC		4.8		12 ppm Cl; 18 ppm Br	Narukar, 1993
Herpes Simplex Virus	Spa Water	3 - 4	Immediate	37 -40ºC		8.2		20 ppm Cl; 40 ppm Br	Narukar, 1993
Herpes Simplex Virus	Plastic Surfaces in Humid Atmosphere	> 4	< 6 hours	37 -40ºC					Narukar, 1993
Norovirus (Genogroup II)	Surface Water		4 months	0 - 4 ºC				Epidemiologic evidence suggests survival and mobility in ice covered river	Kukkula, 1999
Coliphage F2	Positive Charged Nylon Filter Membrane in PBS	no observable	11 weeks	4ºC		7.3			Pesaro, 1995
Bovine Parvovirus (Haden strain)	Positive Charged Nylon Filter Membrane in PBS	no observable	6 - 7 weeks	4ºC		7.3			Pesaro, 1995
Encephalomyocarditis virus	Positive Charged Nylon Filter Membrane in PBS	no observable	6 - 7 weeks	4ºC		7.3			Pesaro, 1995
Bovine Rotavirus	Positive Charged Nylon Filter Membrane in PBS	no observable	6 - 7 weeks	4ºC		7.3			Pesaro, 1995
Bovine Adenovirus	Positive Charged Nylon Filter Membrane in PBS	no observable	6 - 7 weeks	4ºC		7.3			Pesaro, 1995
Bovine Herpesvirus 2	Positive Charged Nylon Filter Membrane in PBS	1.5	7 - 7 weeks	4ºC		7.3			Pesaro, 1995
Coliphage F2	Positive Charged Nylon Filter Membrane in Cattle Liquid Manure	1	9.4 days	15.9 (10.5 - 19.9)ºC		8.7		Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Bovine Parvovirus (Haden strain)	Positive Charged Nylon Filter Membrane in Cattle Liquid Manure	1	8.2 days	15.9 (10.5 - 19.9)ºC		8.7		Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Encephalomyocarditis virus	Positive Charged Nylon Filter Membrane in Cattle Liquid Manure	1	27.5 days	15.9 (10.5 - 19.9)ºC		8.7		Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Bovine Rotavirus	Positive Charged Nylon Filter Membrane in Cattle Liquid Manure	1	20.3 days	15.9 (10.5 - 19.9)ºC		8.7		Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Bovine Adenovirus	Positive Charged Nylon Filter Membrane in Cattle Liquid Manure	1	8.4 days	15.9 (10.5 - 19.9)ºC		8.7		Persistence was longer when factors other than temperature eliminated	Pesaro, 1995

TABLE 3. VIRUS SURVIVAL IN LIQUID AND SEMI-SOLD MEDIA, INCLUDING FAECES, SLUDGES AND SOIL MATERIALS (CONTINUED)

Virus	Medium	log10 reduction	Time	Temp	pH	Conductivity	Turbidity	Other Conditions	Citation
Bovine Herpesvirus 2	Positive Charged Nylon Filter Membrane in Cattle Liquid Manure	1	< 2.5 days	15.9 (10.5 - 19.9)°C	8.7			Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Coliphage F2	Positive Charged Nylon Filter Membrane in Cattle Manure	1	34.7 days	16.2 (11.2 - 19.0)°C	6.9 (6.6 - 7.1)			Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Bovine Parvovirus (Haden strain)	Positive Charged Nylon Filter Membrane in Cattle Manure	1	78.1 days	16.2 (11.2 - 19.0)°C	6.9 (6.6 - 7.1)			Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Encephalomyocarditis virus	Positive Charged Nylon Filter Membrane in Cattle Manure	1	197 days	16.2 (11.2 - 19.0)°C	6.9 (6.6 - 7.1)			Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Bovine Rotavirus	Positive Charged Nylon Filter Membrane in Cattle Manure	1	38 days	16.2 (11.2 - 19.0)°C	6.9 (6.6 - 7.1)			Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Bovine Adenovirus	Positive Charged Nylon Filter Membrane in Cattle Manure	1	22.9 days	16.2 (11.2 - 19.0)°C	6.9 (6.6 - 7.1)			Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Bovine Herpesvirus 2	Positive Charged Nylon Filter Membrane in Cattle Manure	1	< 2.5 days	16.2 (11.2 - 19.0)°C	6.9 (6.6 - 7.1)			Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Coliphage F2	Positive Charged Nylon Filter Membrane in Swine Manure	1	23 days	12.7 (9.5 - 17.7)°C	7.4 (7.0 - 7.8)			Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Bovine Parvovirus (Haden strain)	Positive Charged Nylon Filter Membrane in Swine Manure	1	28.9 days	12.7 (9.5 - 17.7)°C	7.4 (7.0 - 7.8)			Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Encephalomyocarditis virus	Positive Charged Nylon Filter Membrane in Swine Manure	1	104 days	12.7 (9.5 - 17.7)°C	7.4 (7.0 - 7.8)			Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Bovine Rotavirus	Positive Charged Nylon Filter Membrane in Swine Manure	1	29.3 days	12.7 (9.5 - 17.7)°C	7.4 (7.0 - 7.8)			Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Bovine Adenovirus	Positive Charged Nylon Filter Membrane in Swine Manure	1	37.3 days	12.7 (9.5 - 17.7)°C	7.4 (7.0 - 7.8)			Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Bovine Herpesvirus 2	Positive Charged Nylon Filter Membrane in Swine Manure	1	< 2.5 days	12.7 (9.5 - 17.7)°C	7.4 (7.0 - 7.8)			Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Coliphage F2	Positive Charged Nylon Filter Membrane in Mixed Manure	1	27.3 days	14.8 (11.0 - 17.7)°C	7.9 (6.9 - 8.7)			Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Bovine Parvovirus (Haden strain)	Positive Charged Nylon Filter Membrane in Mixed Manure	1	44.6 days	14.8 (11.0 - 17.7)°C	7.9 (6.9 - 8.7)			Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Encephalomyocarditis virus	Positive Charged Nylon Filter Membrane in Mixed Manure	1	61.8 days	14.8 (11.0 - 17.7)°C	7.9 (6.9 - 8.7)			Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Bovine Rotavirus	Positive Charged Nylon Filter Membrane in Mixed Manure	1	24.5 days	14.8 (11.0 - 17.7)°C	7.9 (6.9 - 8.7)			Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Bovine Adenovirus	Positive Charged Nylon Filter Membrane in Mixed Manure	1	34.9 days	14.8 (11.0 - 17.7)°C	7.9 (6.9 - 8.7)			Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Bovine Herpesvirus 2	Positive Charged Nylon Filter Membrane in Mixed Manure	1	< 2.5 days	14.8 (11.0 - 17.7)°C	7.9 (6.9 - 8.7)			Persistence was longer when factors other than temperature eliminated	Pesaro, 1995
Hepatitis A virus	PBS	1	217.4 days	5°C	6.9 start (6.85 end)				Deng, 1995
Hepatitis A virus	Septic Tank Effluent (STE)	1	58.5 days	5°C	6.9 start (7.22 end)			Total Solids = 2,015 mg/L	Deng, 1995
Hepatitis A virus	20% STE Mixed with 80% Dairy Cattle Manure Slurry	1	34.6 days	5°C	6.9 start (7.04 end)			Total Solids = 20,125 mg/L	Deng, 1995
Hepatitis A virus	20% STE Mixed with 80% Swine Manure Slurry	1	48.5 days	5°C	6.9 start (7.58 end)			Total Solids = 23,375 mg/L	Deng, 1995
Hepatitis A virus	PBS	1	90.1 days	22°C	6.9 start (6.20 end)				Deng, 1995
Hepatitis A virus	Septic Tank Effluent (STE)	1	35.1 days	22°C	6.9 start (7.15)			Total Solids = 2,015 mg/L	Deng, 1995
Hepatitis A virus	20% STE Mixed with 80% Dairy Cattle Manure Slurry	1	23.0 days	22°C	6.9 start (7.6 end)			Total Solids = 20,125 mg/L	Deng, 1995
Hepatitis A virus	20% STE Mixed with 80% Swine Manure Slurry	1	17.1 days	22°C	6.9 start (7.0 end)			Total Solids = 23,375 mg/L	Deng, 1995
Hepatitis A virus	20% STE Mixed with 80% Dairy Cattle Manure Slurry	1	8.3 days	28°C	6.9				Deng, 1995
Hepatitis A virus	20% STE Mixed with 80% Swine Manure Slurry	1	8.1 days	28°C	6.9				Deng, 1995
Hepatitis A virus	20% STE Mixed with 80% Dairy Cattle Manure Slurry	1	6.8 days	37°C	6.9				Deng, 1995
Hepatitis A virus	20% STE Mixed with 80% Swine Manure Slurry	1	6.8 days	37°C	6.9				Deng, 1995
Poliovirus Type 1	Septic Tank Effluent (STE)	1.5	28 days	20°C					Green, 1975
Poliovirus Type 1	Septic Tank Effluent (STE)	0.4	28 days	7°C					Green, 1975
Poliovirus Type 1	Cell Culture Growth Medium	6	5 minutes	60°C					Wiley, 1969
Poliovirus Type 1	Cell Culture Growth Medium	6	30 minutes	55°C					Wiley, 1969
Poliovirus Type 1	Composted Sewage Solids	> 0.15	< 1 hour	55 - 60°C				Available virus recovery methods were inadequate to quantify reduction beyond 0.15 $\log_{10}$	Wiley, 1969
Poliovirus	Raw Sewage	0.9	5 minutes	Room Temperature					Clarke, 1961
Poliovirus	Raw Sewage	2	45 minutes	Room Temperature					Clarke, 1961
Poliovirus Type 1	Mock Solid Waste Leachates	0.5 - 1.5	27 days	4°C	5.1 - 7.5	8590 - 9080 mg NaCl/L	6.5 - 13 FTU	dependent on leachate type	Sobsey, 1975
Poliovirus Type 1	Mock Solid Waste Leachates	1.7 - 2.4	28 days	20°C	5.1 - 7.5	8590 - 9080 mg NaCl/L	6.5 - 13 FTU	dependent on leachate type	Sobsey, 1975
Poliovirus Type 1	Mock Solid Waste Leachates	2.2 - 4.4	6 days	37°C	5.1 - 7.5	8590 - 9080 mg NaCl/L	6.5 - 13 FTU	dependent on leachate type	Sobsey, 1975
Poliovirus Type 1	0.05 M Glycine	< 0.5	27 days	4°C					Sobsey, 1975
Poliovirus Type 1	0.05 M Glycine	< 1	27 days	20°C					Sobsey, 1975
Poliovirus Type 1	0.05 M Glycine	1 - 2.7	6 days	37°C					Sobsey, 1975
Poliovirus Type 1	Sewage Sludge Amended Fine Sand	5 - 6	35 days	23.5 - 29°C	5.7 - 7.8	105 - 1360 (µs/cm)		35 day persistence during heavy rainfall	Bitton, 1984
Poliovirus Type 1	Sewage Sludge Amended Fine Sand	6	< 21 days	18 - 27°C	6.9 - 6.9	190 - 1200 (µs/cm)		Last positive detection at 8 days; dry conditions	Bitton, 1984
Echovirus Type 1	Sewage Sludge Amended Fine Sand	4	< 21 days	19 - 27°C	6.9 - 6.9	190 - 1200 (µs/cm)		Last positive detection at 8 days; dry conditions	Bitton, 1984

TABLE 3. VIRUS SURVIVAL IN LIQUID AND SEMI-SOLD MEDIA, INCLUDING FAECES, SLUDGES AND SOIL MATERIALS (CONTINUED)

Virus	Medium	log10 reduction	Time	Temp	pH	Conductivity	Turbidity	Other Conditions	Citation
Culturable Enteric Viruses	Extended Aeration Sewage Sludge	Stable	up to 38 days	6°C	6.4 - 7.4			Assay of indigenous viruses	Berg, 1986
Culturable Enteric Viruses	Oxidation Ditch Sewage Sludge	Stable	up to 15 days	6°C	6.3 - 6.9			Assay of indigenous viruses	Berg, 1986
Culturable Enteric Viruses	Land Disposed Sludge	1.5 - 2.3	7 days	20 - 31°C				depending on moisture loss	Hurst, 1978
Culturable Enteric Viruses	Sludge Treatment (Activation, Settling, Thickening)	0.2	9 hours	21 - 31°C					Hurst, 1978
Poliovirus Type 1	Digested Sludge	2	1 day	28°C					Ward, 1976
Poliovirus Type 1	Digested Sludge	2	3 days	20°C					Ward, 1976
Indigenous Viruses	Land Disposed Sludge	2	9 days						Farrah, 1981
Indigenous Viruses	Sludge lagoon		7 months					lagoon frozen for 6 months	Sattar, 1974
Poliovirus Type 1	Digested Sludge		≥ 66 days	4°C				Completely digested with 40 day retention time at 34°C	Wellings, 1976
Reovirus	Digested Sludge		≥ 66 days	4°C				Completely digested with 40 day retention time at 34°C	Wellings, 1976
Echovirus 22/23 complex	Digested Sludge		≥ 65 days	4°C				Completely digested with 40 day retention time at 34°C	Wellings, 1976
PoliovirusType 2	Digested Sludge		≥7 days	4°C				Completely digested with >60 day retention time at 34°C	Wellings, 1976
Poliovirus Type 3	Digested Sludge		≥7 days	4°C				Completely digested with >60 day retention time at 34°C	Wellings, 1976
Echovirus 22/23 complex	Digested Sludge		≥8 days	4°C				Completely digested with >60 day retention time at 34°C	Wellings, 1976
Poliovirus 3	Field Sprayed Sludge		≥ 48 hour						Wellings, 1976
Echovirus Type 7	Sludge from Drying Bed		≥ 13 day						Wellings, 1976
Rotavirus	Feces	2.8	13 days	20°C				13 % relative humidity	Moe, 1982
Rotavirus	Feces	4.3	11 days	20°C				33 % relative humidity	Moe, 1982
Rotavirus	Feces	4.1	9 days	20°C				55 % relative humidity	Moe, 1982
Rotavirus	Feces	2.9	13 days	20°C				75% relative humidity	Moe, 1982
Rotavirus	Feces	1.9	13 days	20°C				92% relative humidity	Moe, 1982
Rotavirus	Feces	2*	33 days	4°C				12 - 14% relative humidity; * Time for 2 log10 reduction calculated from reported Inactivation rates	Moe, 1982
Rotavirus	Feces	2*	25 days	4°C				33 - 34% relative humidity; * Time for 2 log10 reduction calculated from reported Inactivation rates	Moe, 1982
Rotavirus	Feces	2*	17 days	4°C				51 - 59% relative humidity; * Time for 2 log10 reduction calculated from reported Inactivation rates	Moe, 1982
Rotavirus	Feces	2*	33 days	4°C				75 - 76% relative humidity; * Time for 2 log10 reduction calculated from reported Inactivation rates	Moe, 1982
Rotavirus	Feces	2*	59 days	4°C				92 - 94% relative humidity; * Time for 2 log10 reduction calculated from reported Inactivation rates	Moe, 1982
Rotavirus	Feces	2*	10 days	20°C				13 - 14% relative humidity; * Time for 2 log10 reduction calculated from reported Inactivation rates	Moe, 1982
Rotavirus	Feces	2*	6 days	20°C				34 - 34% relative humidity; * Time for 2 log10 reduction calculated from reported Inactivation rates	Moe, 1982
Rotavirus	Feces	2*	4 days	20°C				52 - 59% relative humidity; * Time for 2 log10 reduction calculated from reported Inactivation rates	Moe, 1982
Rotavirus	Feces	2*	10 days	20°C				75 - 76% relative humidity; * Time for 2 log10 reduction calculated from reported Inactivation rates	Moe, 1982
Rotavirus	Feces	2*	15 days	20°C				93 - 94% relative humidity; * Time for 2 log10 reduction calculated from reported Inactivation rates	Moe, 1982
Rotavirus	Feces	2*	3 days	20°C				14 - 14% relative humidity; * Time for 2 log10 reduction calculated from reported Inactivation rates	Moe, 1982
Rotavirus	Feces	2*	2 days	37°C				35 - 34% relative humidity; * Time for 2 log10 reduction calculated from reported Inactivation rates	Moe, 1982
Rotavirus	Feces	2*	1 day	37°C				53 - 59% relative humidity; * Time for 2 log10 reduction calculated from reported Inactivation rates	Moe, 1982
Rotavirus	Feces	2*	2 days	37°C				77 - 76% relative humidity; * Time for 2 log10 reduction calculated from reported Inactivation rates	Moe, 1982
Rotavirus	Feces	2*	6 days	37°C				94 - 94% relative humidity; * Time for 2 log10 reduction calculated from reported Inactivation rates	Moe, 1982
Poliovirus Type 1 (CHAT)	Creek Water								Pancorbo, 1987
Poliovirus Type 1 (CHAT)	Secondary Effluent (pre-chlorination)								Pancorbo, 1987
Poliovirus Type 1 (CHAT)	Lake Water								Pancorbo, 1987
Poliovirus Type 1 (CHAT)	Groundwater								Pancorbo, 1987
Poliovirus Type 1 (CHAT)	Chlorinated Tap Water								Pancorbo, 1987
Rotavirus Type 2 (strain Wa)	Creek Water								Pancorbo, 1987
Rotavirus Type 2 (strain Wa)	Secondary Effluent (pre-chlorination)								Pancorbo, 1987
Rotavirus Type 2 (strain Wa)	Lake Water								Pancorbo, 1987
Rotavirus Type 2 (strain Wa)	Groundwater								Pancorbo, 1987
Rotavirus Type 2 (strain Wa)	Chlorinated Tap Water								Pancorbo, 1987
Poliovirus Type 1 (CHAT)	Septage	> 2	90 minutes	50°C					Stramer, 1984a
Poliovirus Type 1 (CHAT)	Septage	5 - 6	15 - 30 minutes	55°C					Stramer, 1984a
Poliovirus Type 1	Septic Tank Sludge		100 days						Stramer, 1984b
Poliovirus Type 1 (Sabin)	Activated Sludge Solids (suspended in Dulbecco's MEM)	2.5	28 days						Nekjma, 2003
Poliovirus Type 1 (Sabin)	Dulbecco's MEM	3.7	28 days						Nekjma, 2003

TABLE 3. VIRUS SURVIVAL IN LIQUID AND SEMI-SOLD MEDIA, INCLUDING FAECES, SLUDGES AND SOIL MATERIALS (CONTINUED)

Virus	Medium	log10 reduction	Time	Temp	pH	Conductivity	Turbidity	Other Conditions	Citation
Indigenous Viruses	Beef Extract Eluate of Sewage Sludge	1	375 days	-70°C					Hurst, 1988a
Indigenous Viruses	Beef Extract Eluate of Sewage Sludge	1	198 days	2°C					Hurst, 1988a
Indigenous Viruses	Beef Extract Eluate of Sewage Sludge	1	27 days	23°C					Hurst, 1988a
Indigenous Viruses	Beef Extract Eluate of Sewage Sludge (concentrated by organic flocculation)	1	246 days	-70°C					Hurst, 1988a
Indigenous Viruses	Beef Extract Eluate of Sewage Sludge (concentrated by organic flocculation)	1	132 days	2°C					Hurst, 1988a
Indigenous Viruses	Beef Extract Eluate of Sewage Sludge (concentrated by organic flocculation)	1	22 days	23°C					Hurst, 1988a
Indigenous Viruses	Sewage Sludge	1	153	-70°C					Hurst, 1988b
Indigenous Viruses	Sewage Sludge	1	180	2°C					Hurst, 1988b
Indigenous Viruses	Sewage Sludge	1	26	23°C					Hurst, 1988b
Hepatitis A virus	Dried Feces		> 30 days	25°C				42% relative humidity	McCausland, 1982
Indigenous Enteroviruses	Florida Cypress Dome		> 28 days						Wellings, 1975
Poliovirus Type 1	Sand Moistened with Oxidation Pond Effluent		91 days						Lefler, 1974
Poliovirus Type 1	Dry Sterile Sand		77 days						Lefler, 1974
Poliovirus Type 1 (LSc)	Seawater (viruses suspended in dialysis tubing)	2	1 - 2 days	30 - 33°C	7.85 - 8.9		5.4 - 11		Labelle, 1980
Echovirus Type 1 (Farouk)	Seawater (viruses suspended in dialysis tubing)	2	< 1 day	30 - 33°C	7.85 - 8.9		5.4 - 11		Labelle, 1980
Poliovirus Type 1 (LSc)	Seawater and Sediment (viruses suspended in dialysis tubing)	2	2 - 3 days	30 - 33°C	7.85 - 8.9		5.4 - 11		Labelle, 1980
Echovirus Type 1 (Farouk)	Seawater and Sediment (viruses suspended in dialysis tubing)	2	4 days	30 - 33°C	7.85 - 8.9		5.4 - 11		Labelle, 1980
Poliovirus Type 1 (LSc)	Sterile Artificial Seawater	2	2 - 5 days	30 - 33°C	7.85 - 8.9		5.4 - 11		Labelle, 1980
Echovirus Type 1 (Farouk)	Sterile Artificial Seawater	2	3 days	30 - 33°C	7.85 - 8.9		5.4 - 11		Labelle, 1980
Poliovirus Type 1 (LSc)	Sterile Artificial Seawater and Sediment	2	≥ 5 days	30 - 33°C	7.85 - 8.9		5.4 - 11		Labelle, 1980
Echovirus Type 1 (Farouk)	Sterile Artificial Seawater and Sediment	2	4 days	30 - 33°C	7.85 - 8.9		5.4 - 11		Labelle, 1980
Rhinovirus strain HGP	0.1 M Tris Buffer	2	2 minutes	55°C	7.4 - 7.6				Dimmock, 1967
Rhinovirus strain HGP	0.1 M Tris Buffer	2	10 minutes	50°C	7.4 - 7.6				Dimmock, 1967
Poliovirus Type 1 (LSc)	0.1 M Tris Buffer	6	10 minutes	50°C	7.4 - 7.6				Dimmock, 1967
Poliovirus Type 1 (LSc)	0.1 M Tris Buffer	2	> 5 hours	47°C	7.4 - 7.6				Dimmock, 1967
Rhinovirus strain HGP	0.1 M Tris Buffer	4	< 10 minutes	45°C	7.4 - 7.6				Dimmock, 1967
Poliovirus Type 1 (LSc)	0.1 M Tris Buffer	4	50 hours	45°C	7.4 - 7.6				Dimmock, 1967
Rhinovirus strain HGP	0.1 M Tris Buffer	4	3 days	40°C	7.4 - 7.6				Dimmock, 1967
Poliovirus Type 1 (LSc)	0.1 M Tris Buffer	4	8 days	40°C	7.4 - 7.6				Dimmock, 1967
Rhinovirus strain HGP	0.1 M Tris Buffer	3	2 days	35°C	7.4 - 7.6				Dimmock, 1967
Poliovirus Type 1 (LSc)	0.1 M Tris Buffer	6	30 days	35°C	7.4 - 7.6				Dimmock, 1967
Rhinovirus strain HGP	0.1 M Tris Buffer	3	7 days	30°C	7.4 - 7.6				Dimmock, 1967
Poliovirus Type 1 (LSc)	0.1 M Tris Buffer	4	> 30 days	30°C	7.4 - 7.6				Dimmock, 1967
Rhinovirus strain HGP	0.1 M Tris Buffer	2	15 days	20°C	7.4 - 7.6				Dimmock, 1967
Poliovirus Type 1 (LSc)	0.1 M Tris Buffer	2	60 days	20°C	7.4 - 7.6				Dimmock, 1967
Coliphage T3	Stock Suspension	0.3	42	37°C					Warren, 1969
Coliphage T3	Stock Suspension	0.3	72 days	31°C					Warren, 1969
Coliphage T3	Stock Suspension	0.4	72 days	21°C					Warren, 1969
Coliphage T3	Stock Suspension	2	72 days	4°C					Warren, 1969
Coliphage T3	Stock Suspension	stable	72 days	-20°C					Warren, 1969
Coliphage T3	Aerosol (in rotating drum)	stable	240 minutes	21°C				95% RH, Immediate 1 log10 loss then stable	Platt, 1979
Coliphage T3	Aerosol (in rotating drum)	0.6	240 minutes	21°C				80% RH, Immediate 2 log10 loss then 0.6 log10 after 240 minutes	Platt, 1980
Coliphage T3	Aerosol (in rotating drum)	0.6	240 minutes	21°C				50% RH, Immediate 5 log10 loss then 0.6 log10 after 240 minutes	Platt, 1981
Coliphage T3	Aerosol (in rotating drum)	1.6	240 minutes	21°C				30% RH, Immediate 6 log10 loss then 1.6 log10 after 240 minutes	Platt, 1982
Coliphage T3	Aerosol (in rotating drum)	> 3.6	240 minutes	21°C				10% RH, Immediate 4.6 log10 loss then below detection before 180 minutes	Platt, 1983
Pseudorabies virus	Saline G (phenol red-phosphate buffer)	2	30 - 130 minutes	48°C	6.9			Dependent on strain, T99 calculated from inactivation rates	Platt, 1984
Coxsackie B3 (Nancy)	Tris Buffer w/wo Sat and BSA on Paper, Plastic or Glass	0 - 0.2	24 hours	6°C	7			Salt and protein offered protective effect; higher inactivations under drying conditions	McGeady, 1979
Coxsackie B3 (Nancy)	Tris Buffer w/wo Sat and BSA on Paper, Plastic or Glass	0 - 1.8	24 hours	23°C	7			Salt and protein offered protective effect; higher inactivations under drying conditions	McGeady, 1979
Coxsackie B3 (Nancy)	Tris Buffer w/wo Sat and BSA on Paper, Plastic or Glass	0.3 - 6	24 hours	37°C	7			Salt and protein offered protective effect; higher inactivations under drying conditions	McGeady, 1979
Rhinovirus Type 2 strain HGP	Tris Buffer w/wo Sat and BSA	0	24 hours	6°C	7			wet or drying conditions	Reagan, 1981
Rhinovirus Type 14 strain 1059	Tris Buffer w/wo Sat and BSA	0 - 0.1	24 hours	6°C	7			wet or drying conditions	Reagan, 1981
Rhinovirus Type 2 strain HGP	Tris Buffer w/wo Sat and BSA	0.1 - 0.3	24 hours	23°C	7			underwet conditions	Reagan, 1981
Rhinovirus Type 14 strain 1059	Tris Buffer w/wo Sat and BSA	0.2 - 0.3	24 hours	23°C	7			underwet conditions	Reagan, 1981
Rhinovirus Type 2 strain HGP	Tris Buffer w/wo Sat and BSA	0.5 - 1.0	24 hours	37°C	7			underwet conditions	Reagan, 1981
Rhinovirus Type 14 strain 1059	Tris Buffer w/wo Sat and BSA	0.3 - 0.6	24 hours	37°C	7			underwet conditions	Reagan, 1981
Rhinovirus Type 2 strain HGP	Tris Buffer w/wo Sat and BSA	0.2 - 0.5	24 hours	23°C	7			under drying conditions	Reagan, 1981
Rhinovirus Type 14 strain 1059	Tris Buffer w/wo Sat and BSA	1.3 - 1.7	24 hours	23°C	7			under drying conditions	Reagan, 1981
Rhinovirus Type 2 strain HGP	Tris Buffer w/wo Sat and BSA	2.0 - 3.2	24 hours	37°C	7			under drying conditions, slight protective effect from salt and protein	Reagan, 1981

TABLE 3. VIRUS SURVIVAL IN LIQUID AND SEMI-SOLD MEDIA, INCLUDING FAECES, SLUDGES AND SOIL MATERIALS (CONTINUED)

Virus	Medium	log10 reduction	Time	Temp	pH	Conductivity	Turbidity	Other Conditions	Citation
Rhinovirus Type 14 strain 1059	Tris Buffer w/wo Salt and BSA	2.6 - 4.6	24 hours	37°C		7		underdying conditions, slight protective effect from salt and protein	Reagan, 1981
Adenovirus Type 19 (Kowalski strain)	Topical Fluorescein Ophthalmic Solution	2.25	21 days	25°C					Kowalski, 1999
Adenovirus Type 28 (Cray strain)	Topical Fluorescein Ophthalmic Solution	2.25	28 days	25°C					Kowalski, 1999
Herpes Simplex virus 1 (McRae)	Topical Fluorescein Ophthalmic Solution	2 - 4	< 1 hour						Romanowski, 1999
Herpes Simplex virus 1 (McRae)	Proparacaine Hydrochloride Ophthalmic Solution	2 - 4	< 1 hour						Romanowski, 1999
Herpes Simplex virus 1 (McRae)	Artificial Tears Ophthalmic Solution	3	5 days					Inactivation leveled off after 5 days, recovery to at least 7 days depending on initial dose	Romanowski, 1999
Pseudorabies virus	PBS	1	10 days	25°C					Schoenbaum, 1991
Pseudorabies virus	Saline G	1	10 days	25°C					Schoenbaum, 1991
Pseudorabies virus	Swine Nasal Washings	7	2 days	25°C					Schoenbaum, 1991
Pseudorabies virus	Swine Saliva	7	4 days	25°C					Schoenbaum, 1991
Pseudorabies virus	Swine Urine	7	14 days	25°C					Schoenbaum, 1991
Pseudorabies virus	Swine Lagoon Water	4	2 days	25°C					Schoenbaum, 1991
Pseudorabies virus	Swine Pit Effluent	> 3	< 1 day	25°C					Schoenbaum, 1991
Pseudorabies virus	Swine Bile	> 3	< 1 day	25°C				very rapid	Schoenbaum, 1991
Pseudorabies virus	Chlorinated Water	4	1 day	25°C					Schoenbaum, 1991
Pseudorabies virus	Well Water	4	7 days	25°C					Schoenbaum, 1991
Pseudorabies virus	Steel	2	7 days	25°C				In saline G; 7 log10 Inactivation in 2-4 days in saliva or nasal washings	Schoenbaum, 1991
Pseudorabies virus	Concrete	7	1 - 4 days	25°C				Depending on Diluent, greater survival in saliva	Schoenbaum, 1991
Pseudorabies virus	Polypropylene	7	3 - 7 days	25°C				Depending on Diluent, greatest survival in saline G	Schoenbaum, 1991
Pseudorabies virus	Vinyl Rubber	7	2 - 7 days	25°C				Depending on Diluent, greatest survival in saline G	Schoenbaum, 1991
Pseudorabies virus	Denim Cloth	7	< 1 day	25°C					Schoenbaum, 1991
Pseudorabies virus	Loam Soil	7	2 - 7 days	25°C				Depending on Diluent, greatest survival in saliva, poorest in nasal washings	Schoenbaum, 1991
Pseudorabies virus	Green Grass	7	1 - 2 days	25°C				Depending on Diluent, greater survival in saliva	Schoenbaum, 1991
Pseudorabies virus	Whole Corn	1	7 days	25°C				In saline G; 7 log10 Inactivation in 2-4 days in saliva or nasal washings	Schoenbaum, 1991
Pseudorabies virus	Pelleted Feed	7	1 - 3 days	25°C				Depending on Diluent, greater survival in saline G	Schoenbaum, 1991
Pseudorabies virus	Meat and Bone Meal	7	2 - >4 days	25°C				Depending on Diluent, greatest survival in saline G	Schoenbaum, 1991
Pseudorabies virus	Alfalfa	7	< 1 day	25°C					Schoenbaum, 1991
Pseudorabies virus	Straw	7	3 - 4 days	25°C				Depending on Diluent, greater survival in saliva and nasal washings	Schoenbaum, 1991
Pseudorabies virus	Wood	7	1 - 2 days	25°C				Depending on Diluent, greater survival in saliva	Schoenbaum, 1991
Pseudorabies virus	Swine Feces	7	1 - 2 days	25°C					Schoenbaum, 1991
HIV (LAV/HTLV-III)			30 minutes	56°C					Spire, 1985
HIV (LAV/HTLV-III)			30 minutes	60°C					Ehrnsson, 1989
HIV (LAV/HTLV-III)			30 minutes	56°C					Heraida, 1986
HIV (LAV/HTLV-III)			> 48 hour	30°C					Barre-Sinoussa, 1984
HIV (LAV/HTLV-III)	Saliva (maintained wet or dried)	stable	4 days	20 - 22°C				Only slight decrease after 7 days	Barre-Sinoussa, 1985
HIV (LAV/HTLV-III)	50% human plasma		> 15 days	23 - 27°C					Resnick, 1986
HIV (LAV/HTLV-III)	50% human plasma	7	11 - 15 days	36 - 37°C					Resnick, 1987
HIV (LAV/HTLV-III)	50% human plasma	7	5 hours	54 - 56°C					Resnick, 1988
HIV (LAV/HTLV-III)	Dried Preparation of H9 cells	7	1 - 3 days	24 - 27°C					Resnick, 1989
HIV-1	Phosphate Buffer with 2 % FBS	0.3	24 hours	37°C		7.1			Tjotta, 1991
HIV-1	Phosphate Buffer with 2 % FBS	0.3	30 - 35 hours	20 - 22°C		7.1			Tjotta, 1991
HIV-1	Phosphate Buffer with 2 % FBS	0.3	9 days	4°C		7.1		rate pH dependent	Tjotta, 1991
HIV-1	Phosphate Buffer with 2 % FBS	0.1 - 0.2	6 months	-20°C		7.1			Tjotta, 1991
HIV-1	Phosphate Buffer with 2 % FBS	stable	6 months	-75°C		7.1			Tjotta, 1991
Poliovirus Type 1 (LSc)	Sterile Soil Suspensions, 5% wt/vol	2	18 - 167 days	20°C	6.5; except Organic Muck, 5.5)			Depending on soil type	Sobsey, 1980
Poliovirus Type 1 (LSc)	Non-sterile Soil Suspensions, 5% wt/vol	2	9 - 80 days	20°C	6.5; except Organic Muck, 5.5)			Depending on soil type	Sobsey, 1980
Reovirus Type 3 (Dearing)	Sterile Soil Suspensions, 5% wt/vol	2	8 - 257 days	20°C	6.5; except Organic Muck, 5.5)			Depending on soil type	Sobsey, 1980
Reovirus Type 3 (Dearing)	Non-sterile Soil Suspensions, 5% wt/vol	2	9 - 110 days	20°C	6.5; except Organic Muck, 5.5)			Depending on soil type	Sobsey, 1980
Poliovirus Type 1 (LSc)	Sandy Loam Soil	1	455 days (Calculated value, actual observance to 75 days)	1 °C				Aerobic; sterile	Hurst, 1988
Poliovirus Type 1 (LSc)	Sandy Loam Soil	1	152 days(Calculated value, actual observance to 75 days)	1 °C				Aerobic; non-sterile	Hurst, 1988
Poliovirus Type 1 (LSc)	Sandy Loam Soil	1	688 days(Calculated value, actual observance to 75 days)	1 °C				Anaerobic; sterile	Hurst, 1988
Poliovirus Type 1 (LSc)	Sandy Loam Soil	1	323 days(Calculated value, actual observance to 75 days)	1 °C				Anaerobic; non-sterile	Hurst, 1988
Poliovirus Type 1 (LSc)	Sandy Loam Soil	1	31 days	23°C				Aerobic; sterile	Hurst, 1988
Poliovirus Type 1 (LSc)	Sandy Loam Soil	1	10 days	23°C				Aerobic; non-sterile	Hurst, 1988
Poliovirus Type 1 (LSc)	Sandy Loam Soil	1	30 days	23°C				Anaerobic; sterile	Hurst, 1988
Poliovirus Type 1 (LSc)	Sandy Loam Soil	1	33 days	23°C				Anaerobic; non-sterile	Hurst, 1988

TABLE 3. VIRUS SURVIVAL IN LIQUID AND SEMI-SOLD MEDIA, INCLUDING FAECES, SLUDGES AND SOIL MATERIALS (CONTINUED)

Virus	Medium	log10 reduction	Time	Temp	pH	Conductivity	Turbidity	Other Conditions	Citation
Poliovirus Type 1 (LSc)	Sandy Loam Soil	1	3 days	37°C				Aerobic; sterile	Hurst, 1988
Poliovirus Type 1 (LSc)	Sandy Loam Soil	1	1.4 days	37°C				Aerobic; non-sterile	Hurst, 1988
Poliovirus Type 1 (LSc)	Sandy Loam Soil	1	1.7 days	37°C				Anaerobic; sterile	Hurst, 1988
Poliovirus Type 1 (LSc)	Sandy Loam Soil	1	3.5 days	37°C				Anaerobic; non-sterile	Hurst, 1988
Poliovirus Type 1 (LSc)	Rapid Infiltration Basin Soil During Application of Wastewater	1	25 days						Hurst, 1980
Echovirus Type 1 (Farouk)	Rapid Infiltration Basin Soil During Application of Wastewater	1	6.7 days						Hurst, 1980
Poliovirus Type 1 (LSc)	Rapid Infiltration Basin Soil During Drying Phase	1	9.1 days						Hurst, 1980
Echovirus Type 1 (Farouk)	Rapid Infiltration Basin Soil During Drying Phase	1	2 days						Hurst, 1980
Poliovirus Type 1	Effluent Irrigated Soil (Summer)		11 days						Tierney, 1977
Poliovirus Type 1	Effluent Irrigated Soil (Winter)		96 days						Tierney, 1977
Poliovirus Type 1	Sand and Sandy Loam (Saturated with groundwater)	1.4 - 2.5	155 days	4°C	7.7 - 8.0			Depending on soil type	Yeager, 1979
Poliovirus Type 1	Sand and Sandy Loam (Saturated with groundwater)	3.5 - 4.0	21 - 39 days	22°C	7.7 - 8.0			Depending on soil type	Yeager, 1979
Coxsackie virus B1	Sand and Sandy Loam (Saturated with groundwater)	0.2 - 0.5	6 days	22°C	7.7 - 8.0			Depending on soil type	Yeager, 1979
Poliovirus Type 1	Sand and Sandy Loam (Saturated with groundwater)	3.7 -4.5	7 - 11 days	37°C	7.7 - 8.0			Depending on soil type	Yeager, 1979
Herpesvirus of Turkeys	Commercial Vaccine Diluents	0.3 - >2	90 minutes	37°C	6.8 - 7.4			Depending on Diluent	Colwell, 1975
Herpesvirus of Turkeys	Commercial Vaccine Diluents	0.2 - 2	60 minutes	37°C	6.8 - 7.4			Depending on Diluent	Colwell, 1975
Herpesvirus of Turkeys	Commercial Vaccine Diluents	0 - 0.1	60 minutes	0°C	6.8 - 7.4			Depending on Diluent	Colwell, 1975
Herpesvirus of Turkeys	Commercial Vaccine Diluents	0 - 0.4	60 minutes	25°C	6.8 - 7.4			Depending on Diluent	Colwell, 1975
Foot and Mouth Disease Virus	Aerosol on Microthreads In Open Air	0 - 0.8	90 minutes	1 - 12°C				Relative Humidity: 60-95%, Wind: calm to strong	Donaldson and Ferris, 1975
EchovirusType 1	Adhesive Gum		1 - 10 days					Depending on Type of Adhesive Gum	Selwyn, 1965
Adenovirus Type 3	Adhesive Gum		30 minutes - 6 days					Depending on Type of Adhesive Gum	Selwyn, 1965
Herpes Simplex Virus Type 2	Tissue Culture Medium	0.5	72 hours	35°C	6.3			Decreased Inactivation with lower temperature, Increased Inactivation at higher pH	Lanoz, 1979
Hepatitis B virus	1:10 Serum Dilution		1 minute	98°C					Bond, 1981
Aujeszky's Disease virus	Feces		3 - 15 weeks						Strauch, 1991
Borna Virus	Feces		22 days						Strauch, 1991
Marek's Disease Virus	Feces		7 days						Strauch, 1991
Teschen Virus	Feces		3 - 25 days						Strauch, 1991
African Swine Fever Virus	Feces		50 - 150 days						Strauch, 1991
Foot and Mouth Disease Virus	Feces		21 - 103 days						Strauch, 1991
Newcastle Disease Virus	Feces		22 days						Strauch, 1991
Cell-Free Human Immunodeficiency Virus	Complete Medium with 10% Fetal Calf Serum	1	122 hours						van Beusen, 1994
Cell-Associated Human Immunodeficiency Virus	Complete Medium with 10% Fetal Calf Serum	1	146 hours						van Beusen, 1994
Cell-Associated Human Immunodeficiency Virus	FCS heat	1	308 hours						van Beusen, 1994
Porcine Reproductive and Respiratory Syndrome Virus	City Water	3	11 days	26°C					Pirtle, 1996
Porcine Reproductive and Respiratory Syndrome Virus	Well Water	2.3	9 days	26°C					Pirtle, 1996
Porcine Reproductive and Respiratory Syndrome Virus	Saline G	2	6 days	26°C					Pirtle, 1996
Porcine Reproductive and Respiratory Syndrome Virus	PBSS	1	4 days	26°C					Pirtle, 1996
Porcine Reproductive and Respiratory Syndrome Virus	Swine Saliva	3	< 1 day	26°C					Pirtle, 1996
Porcine Reproductive and Respiratory Syndrome Virus	Swine Urine	3	< 1 day	26°C					Pirtle, 1996
Porcine Reproductive and Respiratory Syndrome Virus	Fecal Slurry	3	< 1 day	26°C					Pirtle, 1996
Foot and Mouth Disease Virus	Bovine Feces		78 days	4°C					Parker, 1971; as reported by Bartley, 2002
Foot and Mouth Disease Virus	Bovine Slurry		100.9 days	4°C					Hess, 1995; as reported by Bartley, 2002
Foot and Mouth Disease Virus	Bovine Slurry		79.7 days	17°C					Hess, 1995; as reported by Bartley, 2002
Foot and Mouth Disease Virus	Bovine Slurry		< 12 days	30°C				87 to 90% relative humidity	Bubnow, 1968; as reported by Bartley, 2002
Foot and Mouth Disease Virus	Bovine Slurry		<8 days	32°C					Bubnow, 1968; as reported by Bartley, 2002
Foot and Mouth Disease Virus	Bovine Slurry		3 - 5 days	34°C					Bubnow, 1968; as reported by Bartley, 2002
Astrovirus Type 4	Dechlorinated Drinking Water	2	60 days	4°C	8.38 [illegible] µS		0.51 NTU		Abad, 1997
Astrovirus Type 4	Dechlorinated Drinking Water	3.3	90 days	4°C	8.38 [illegible] µS		0.51 NTU		Abad, 1997
Astrovirus Type 4	Dechlorinated Drinking Water	3.2	60 days	20°C	8.38 [illegible] µS		0.51 NTU		Abad, 1997
Astrovirus Type 4	Dechlorinated Drinking Water	5	90 days	20°C	8.38 [illegible] µS		0.51 NTU		Abad, 1997
Astrovirus Type 4	Drinking Water; 0.5 mg/L Free Chlorine	3	120 minutes	20°C	7.5				Abad, 1997
Astrovirus Type 4	Drinking Water; 1.0 mg/L Free Chlorine	4.17	120 minutes	20°C	7.5				Abad, 1997
Enteric Adenoviruses 40 and 41	Dechlorinated Drinking Water	3.2	60 days	20°C					Abad, 1994, as reported Abad, 1997
Enteric Adenoviruses 40 and 41	Drinking Water; 0.5 mg/L Free Chlorine	2.5	120 minutes	20°C					Abad, 1994, as reported Abad, 1997
Enteric Adenoviruses 40 and 41	Drinking Water; 1.0 mg/L Free Chlorine	3	120 minutes						Abad, 1994, as reported Abad, 1997
Human Rotavirus Type 3	Dechlorinated Drinking Water	3.2	60 days	20°C					Abad, 1994; as reported Abad, 1997
Poliovirus Type 3	Lee River Water	3	9 weeks	5°C					Poynter, 1968
Poliovirus Type 3	Lee River Water	3	11 days	20°C					Poynter, 1968
Echovirus Type 5	Sea Water	1		3 - 5°C					Won, 1973
Echovirus Type 5	Sea Water	5		22°C					Won, 1973
Poliovirus Type 1	Surface Water	2	3 days	23 - 27°C					Obrien, 1977
Poliovirus Type 1	Surface Water	2	25 days	18 - 20°C					Leverzan, 1980
Poliovirus Type 1	Surface Water	2	15 days	19 - 25°C					Hermann, 1974
Poliovirus Type 1	Surface Water	2	6 days	16 - 23°C					Cubbage, 1979
Poliovirus Type 1	Surface Water	2	6 - 9 days	20°C					Hurst, 1980
Poliovirus Type 1	Autoclaved Seawater	2	8 days	21 - 25°C					Davis, 1973

TABLE 3. VIRUS SURVIVAL IN LIQUID AND SEMI-SOLD MEDIA, INCLUDING FAECES, SLUDGES AND SOIL MATERIALS (CONTINUED)

Virus	Medium	log10 reduction	Time	Temp	pH	Conductivity	Turbidity	Other Conditions	Citation
Poliovirus Type 1	Autoclaved Seawater	2	> 78 days	18ºC					Leagrande, 1969
Poliovirus Type 1	Autoclaved Seawater	2	12 - 24 days	22ºC					Dents, 1977
Poliovirus Type 1	Seawater	2	23 days	18ºC					Leagrande, 1969
Poliovirus Type 1	Seawater	2	2 - 3 days	18 - 20ºC					Magnusson, 1967
Poliovirus Type 1	Seawater	2	4 - 5 days	25ºC					Metcalfan, 1967
Poliovirus Type 1	Seawater	2	7 days	15ºC					Shuval, 1971
Poliovirus Type 1	Seawater	2	10 days	24ºC					De Flora, 1975
Poliovirus Type 1	Seawater	2	3 - 4 days	24ºC					Akin, 1974; as reported by Black, 1983
Poliovirus Type 1	Seawater	2	3 - 12 days	22ºC					Dents, 1977
Poliovirus Type 1	Seawater	2	3 - 4 days	18 - 20 ºC					Smith, 1978
Poliovirus Type 1	Seawater	2	1 - 4 days	24ºC					Fujioka, 1980
Poliovirus Type 1	Seawater	2	4 - 5 days	20ºC					Hurst, 1980
Poliovirus Type 1	Seawater	2	11-12 days	25ºC					Chung, 1992
Poliovirus Type 1	Seawater - Sterile	1	18.6 days	25ºC					Dinchera, 1992
Hepatitis A virus	Seawater	2	19 - 20 days	25ºC					Chung, 1992
Coxsackie B3	Seawater	2	12 days	19 - 21ºC					Vaughn, 1975
Coxsackie B4	Seawater	2	28 days	4 - 15ºC					Vaughn, 1975
Poliovirus	Drinking Water - Sterile	2	66 days	25ºC					Quignon, 1992; as cited in Schwartzbrod, 1995
Poliovirus	Drinking Water - Sterile	2	505 days	4ºC					Quignon, 1992; as cited in Schwartzbrod, 1995

TABLE 4. VIRUS SURVIVAL IN AEROSOLS

Virus	Medium	Time	Temp (°C)	pH	RH Low %	%Remaining	logic Reduction	RH Med %	%Remaining	logic Reduction	RH High %	%Remaining	logic Reduction	Other Conditions	Citation
Vaccinia	McIlvaines Citric Acid/di-sodium phosphate buffer with 1% horse serum	23 hours	11	7.2	20	66	0.47	50	69	0.39	80	13	0.1		Harper, 1961
Vaccinia	McIlvaines Citric Acid/di-sodium phosphate buffer with 1% horse serum	23 hours	22	7.2	20	15	0.07	50	12	0.06	80	Trace			Harper, 1961
Vaccinia	McIlvaines Citric Acid/di-sodium phosphate buffer with 1% horse serum	23 hours	32	7.2	20	13	0.06	50	Trace		80	Trace			Harper, 1961
VEE	20% egg yolk in Sorensons buffer	23 hours	11	7.2	20	26	0.13	50	11	0.05	80	6.2	0.03		Harper, 1961
VEE	20% egg yolk in Sorensons buffer	23 hours	22	7.2	20	1.7	0.01	50	0.1	0.00	80	0.1	0.00		Harper, 1961
VEE	20% egg yolk in Sorensons buffer	23 hours	32	7.2	20	0.17	0.00	50	Trace		80	Nil			Harper, 1961
Influenza A (PR8)	0.2% casein in McIlvaine's buffer	23 hours	11	7.4	20	61	0.41	50	19	0.09	80	3	0.01		Harper, 1961
Influenza A (PR8)	0.2% casein in McIlvaine's buffer	23 hours	22	7.4	20	22	0.11	50	Trace		80	Nil			Harper, 1961
Influenza A (PR8)	0.2% casein in McIlvaine's buffer	23 hours	32	7.4	20	1.3	0.01	50	Nil		80	Nil			Harper, 1961
Poliovirus Type 1 (Brunhilde)	PBS	23 hours	22	7.4	20	1.1	0.00	50	Trace		80	85	0.82		Harper, 1961
Pigeon Pox virus	Water	5 hours	not reported		30%	78	0.66	60	> 80		90	> 80			Webb, 1963
Rous Sarcoma Virus	Water	5 hours	not reported		30%	< 1 (at 3 hours)	> 2	60	> 60		90	> 80			Webb, 1963
Rous Sarcoma Virus	Citrate Buffer	5 hours	not reported	7	30%	50%		60	10		90	< 5			Webb, 1963
New Castle Disease Virus (GB Texas Strain)		5 hours	4		10			35		5	90				Songer, 1967
New Castle Disease Virus (GB Texas Strain)		91 minutes	23		10	< 1		35		2	90	1			Songer, 1967
Infectious Bovine Rhinotracheitis Virus		91 minutes	23		10	2.5		35		3	90	<1			Songer, 1967
Vesicular Stomatitis Virus		92 minutes	23		10	1.25		35		2.25	90	1.25			Songer, 1967
Bacteriophage T3		93 minutes	23		10	5.1		35		5 (by 60 minuts	90	stable			Songer, 1967
Yellow Fever Virus (Asibi)	Infective Plasma	60 minutes	-1		30	4		50	3.2		80	3.8			Maynew, 1968
Yellow Fever Virus (Asibi)	Infective Plasma	60 minutes	10		30	5.1		50	3		80	4.2			Maynew, 1968
Yellow Fever Virus (Asibi)	Infective Plasma	60 minutes	27		30	2.5		50	1.4		80	4.5			Maynew, 1968
Mengovirus (37A)		2 hours	27		25		3	50		>5	70	stable			Akers, 1968
Semliki Forest Virus	Water	24 hours	22		20	49		65	28		88	0.4			Benbough, 1969
Murine Leukemia Virus	10% cell free spleen extract	35 minutes	24					35		2.0				Inactivation was during Aerosol generation	Larson, 1970
Influenza A (swine types)	Allantoic Fluid	15 hours	21		15									To last detection	Mitchel, 1972
Influenza A (equine types)	Allantoic Fluid	20 - 30 hours	21		15									To last detection	Mitchel, 1972
Influenza A (human types)	Allantoic Fluid	≤ 15 hours	21		15									To last detection	Mitchel, 1972
Influenza A (avian types)	Allantoic Fluid	24 - 36 hours	21		15									To last detection	Mitchel, 1972
VEE	0.002 M Phosphate Buffer with 0.3% BSA	1 hour		7.5	30	90	0.0	60	78	0.1				In the Dark	Berendt, 1971
VEE	0.002 M Phosphate Buffer with 0.3% BSA	2 hour		7.5	30	0.02	3.7	60	0.006	4.2				Exposure to 584 mcal/cm²/min	Berendt, 1971
Langat Virus	Clarified Culture Fluid	60 minutes			30	>10	1.00	50	<1	2.0	90	>10	1		Benbough, 1971
Langat Virus	Desalted Clarified Culture Fluid	3 hours			30	50	0.30	50	40	0.4	90	10	1		Benbough, 1971
T7 Coliphage	Clarified Lysates	1 hour			30	None Detected	>3	50	0.1	3.0	90	Stable	0		Benbough, 1971
T1 Coliphage	Clarified Lysates	1 hour			30	90	0.0	50	Stable	0	90	Stable	0		Benbough, 1971
T1 Coliphage	Desalted Clarified Lysates	1 hour			30	10	1.00	50	80	0.1	90	Stable	0		Benbough, 1971
Poliovirus Type 1 (LSc)	Clarified Culture Fluid	1 hour			30	2	1.70	50	0.2	2.7	90	80	0.1		Benbough, 1971
T7 Coliphage	Purified Lysates	30 minute						55	0.02	3.7					Stange, 1972
New Castle Disease Virus (Herts 33/56)	Allantoic Fluid	4 hours	20		50	2	1.70	60	9	1.05	70	15	0.82		Hugh-Jones, 1973
New Castle Disease Virus (Eastwood 67)	Allantoic Fluid	4 hours	20		50	3	1.52	60	3	1.52	70	7	1.15		Hugh-Jones, 1973
New Castle Disease Virus (Essex 70)	Allantoic Fluid	4 hours	20		50	1	2.00	60	4	1.40	70	9	1.05		Hugh-Jones, 1973
T3 Coliphage	0.1 M NaCl	30 minutes	20		30		3	60		4	90		3		Trouwborst, 1974
T3 Coliphage	0.1 M NaCl + 0.1% peptone	30 minutes	20		30		3.5	60		4.1	90		1.2		Trouwborst, 1974
Moloney Murine Sarcoma Virus	Cell Culture Fluid		21		25		5	57		5	75		5	Almost all of inactivation during stabilization of aerosol	Hinshaw, 1976
Murine Leukemia Virus	Cell Culture Fluid		21		25		5	57		5	75		5	Almost all of inactivation during stabilization of aerosol	Hinshaw, 1976
Bovine Rhinotracheitis Virus	Eagle's Minimal Essential Medium	3 hours	32		30		3.1				90		2.1		Elazhary, 1979
Bovine Rhinotracheitis Virus	Eagle's Minimal Essential Medium	3 hours	6		30		2.9				90		1.2		Elazhary, 1979

TABLE 4. VIRUS SURVIVAL IN AEROSOLS (CONTINUED)

[redacted]	0.01% Lysine	32 minutes	20				70		0.9	87		0.75		de Mk, 1977
Bovine Adenovirus 3 (WBR-1)	Eagle's Minimal Essential Medium	3 hours	32	30		5.6				90		1.7		Elzaahary, 1979
Bovine Adenovirus 3 (WBR-1)	Eagle's Minimal Essential Medium	3 hours	6	30		2.6				90		1.1		Elzaahary, 1979
Bovine Parainfluenza Type 3	Eagle's Minimal Essential Medium	3 hours	32	30		4.2				90		5.2		Elzaahary, 1979
Bovine Parainfluenza Type 4	Eagle's Minimal Essential Medium	3 hours	6	30		2.2				90		1.4		Elzaahary, 1979
Japanese B Encephalitis Virus	Stock Suspension diluted with BHI and 1FCS	60 minutes	24	30		0.3	65		0.6	80		1		Larson, 1980
Bovine Rotavirus	Cell Culture Fluid	60 minutes	10	20		0.45	60		1.02	90		0.03	Survival up to 2 hours documented at 20%	Moe, 1983
Bovine Rotavirus	Cell Culture Fluid	60 minutes	20	20		0.69	60		1.72	90		0.19	Survival up to 2 hours documented at 20%	Moe, 1983
Bovine Rotavirus	Cell Culture Fluid	60 minutes	30	20		0.84	60		2.39	90		0.32	Survival up to 2 hours documented at 20%	Moe, 1983
Rotavirus SA11	Tryptose Phosphate Broth with Rhodamine and Antifoam C	72 hours	20	25	20	0.70	60	60	0.3	80	< 1 within 30 minutes	> 2		Sattar, 1984
Calf Rotavirus (C-486)	Tryptose Phosphate Broth with sodium fluorescein and Antifoam A	24 hours	20	30	40	0.40	60	68	0.24	80	< 5	1.5		Ijaz, 1985
Poliovirus Type 1 (Sabin)	Tryptose Phosphate Broth with sodium fluorescein and Antifoam A	24 hours	20	30	None Detected at 15 minutes		60	None Detected at 15 minutes		80	20	0.70		Ijaz, 1985
Human Rotavirus (Wa)	Tryptose Phosphate Broth with sodium fluorescein and Antifoam A	72 hours	20	30	20	0.7	60	60	0.3	80	< 5	> 1.5		Ijaz, 1985
Human Rotavirus (Wa)	Tryptose Phosphate Broth with sodium fluorescein and Antifoam A	24 hours	6	30	55	0.26	60	75	0.12	80	< 10	1		Ijaz, 1985
Human Rotavirus (Wa)	10% Feces from confirmed rotavirus patient	24 hours	20				60	70	0.15					Ijaz, 1985
Human Rotavirus (Wa)	Tryptose Phosphate Broth with sodium fluorescein and Antifoam A	24 hours	20				60	60	0.3					Ijaz, 1985
Rhinovirus 14	Tryptose Phosphate Broth with sodium fluorescein and Antifoam A	24 hours	20	30	(<0.25% detectable in first air sample)		60	(<0.25% detectable in first air sample)		80	30	0.52		Karim, 1985
Human Coronavirus (229E)	Tryptose Phosphate Broth with sodium fluorescein and Antifoam A	72 hours	20	30	50	0.3	60	68	0.2	80	3 (at 24 hours)	1.5		Ijaz, 1985
Human Coronavirus (229E)	Tryptose Phosphate Broth with sodium fluorescein and Antifoam A	24 hours	6	30	65	0.2	60	82	0.1	80	85	0.1		Ijaz, 1985
Poliovirus Type 1 (Sabin)	Tryptose Phosphate Broth with sodium fluorescein and Antifoam A	24 hours	20	30	None detected		60	None Detected		80	30	0.5		Ijaz, 1985
Pseudorabies Virus (IowaWilson/S62/66)	Stock Solution with Porcine Mucin and Antifoam	60 minutes	22	25	11	1.0	65	40	0.4	85	10	1.0		Schoenbaum, 1990
Pseudorabies Virus (IowaWilson/S62/66)	Stock Solution with Porcine Mucin and Antifoam	60 minutes	4	could not be achieved			65	68	0.2	85	23	0.6		Schoenbaum, 1990
Reovirus Type 1 (Lang)		150 minutes	2	30	78	0.1	60	85	0.1	80	95	0.0		Mohr, 1991
Reovirus Type 1 (Lang)		150 minutes	14	20	80	0.1	60	60	0.2	80	80	0.1		Mohr, 1991

TABLE 5. VIRUS SURVIVAL ON SURFACES

Virus	Suspending Medium	Surface	$\log_{10}$ reduction	Time	Temp (C)	Relative Humidity (%)	Other Conditions	Citation
Echovirus Type 1	Adhesive Gum	Paper	N.R.	6 - 30 days			Depending on Suspending Medium	Selwyn, 1985
Adenovirus Type 3	Adhesive Gum	Paper	N.R.	1 - 10 days				Selwyn, 1985
Vaccinia (Lederle)	Cell Culture Suspension	Wool	4 - 5	6-14 weeks	25	35	Depending on method of application and fabric type	Sidwell, 1966
Vaccinia (Lederle)	Cell Culture Suspension	Cotton	4 - 5	6-10 weeks	25	35	Depending on method of application and fabric type	Sidwell, 1966
Vaccinia (Lederle)	Cell Culture Suspension	Wool	4 - 5	2 - 4 weeks	25	78	Depending on method of application and fabric type	Sidwell, 1966
Vaccinia (Lederle)	Cell Culture Suspension	Cotton	4 - 5	1 - 2 weeks	25	78	Depending on method of application and fabric type	Sidwell, 1966
Poliovirus Type 2 (MEF-1)	Cell Culture Suspension	Wool	> 5	10 - 20 weeks	25	35	Depending on method of application and fabric type	Dixon, 1966
Poliovirus Type 2 (MEF-1)	Cell Culture Suspension	Cotton	> 5	1 - 4 weeks	25	35	Depending on method of application and fabric type	Dixon, 1966
Poliovirus Type 2 (MEF-1)	Cell Culture Suspension	Wool	> 5	6 - 8 weeks	25	78	Depending on method of application and fabric type	Dixon, 1966
Poliovirus Type 2 (MEF-1)	Cell Culture Suspension	Cotton	> 5	1 - 2 weeks	25	78	Depending on method of application and fabric type	Dixon, 1966
Rhinovirus Type 39	Hanks Balanced Salt Solution or 0.85% saline	Plastic	3 - 4	24 - 72 hours	23	ambient	Depending on initial dose applied	Hendley, 1973
Rhinovirus Type 39	Mucus or Diluted Mucus	Plastic	1 - 4	3 - 24 hours	23	ambient	Depending on initial dose applied	Hendley, 1973
Rhinovirus Type 39	Hanks Balanced Salt Solution or 0.85% saline	Skin	N.R.	1 - 3 hours	23	ambient	Depending on initial dose applied	Hendley, 1973
Rhinovirus Type 39	Hanks Balanced Salt Solution or 0.85% saline	Formica, Stainless Steel, Varnished Wood	0.5	3 hours	23	ambient		Hendley, 1973
Rhinovirus Type 39	Hanks Balanced Salt Solution or 0.85% saline	Nylon, Acetate, Orlon, Dacron, Wool, Silk	1	3 hours	23	ambient		Hendley, 1973
Rhinovirus Type 39	Hanks Balanced Salt Solution or 0.85% saline	Cotton, Rayon	> 2.5	3 hours	23	ambient		Hendley, 1973
Rhinovirus Type 39	Hanks Balanced Salt Solution or 0.85% saline	Facial Tissue, Paper Towel	> 2.8	3 hours	23	ambient		Hendley, 1973
Hepatitis B virus	Blood	Stainless Steel and Cotton Swabs	0.1	72 hours	25	42		Favero, 1974
Hepatitis B virus	Blood	Stainless Steel and Cotton Swabs	0.2	14 days	25	42		Favero, 1974
Adenovirus Type 2	Cell Culture Suspension	Glass, Vinyl Asbestos Tile, Ceramic Tile, Stainless Steel	6	<1 week - <6 weeks	37	93	Greater survival under dark conditions	Mahl, 1974
Adenovirus Type 2	Cell Culture Suspension	Glass, Vinyl Asbestos Tile, Ceramic Tile, Stainless Steel	0.5 - 1	8 weeks	37	7	Light was not a factor	Mahl, 1974
Adenovirus Type 2	Cell Culture Suspension	Glass	2.75	12 weeks	25	7	Best survival at lowest RH, survival to 8 weeks at mid and high RH	Mahl, 1974
Adenovirus Type 2	Cell Culture Suspension	Glass	2.75	12 weeks	37	3	Best survival at lowest RH, survival to 8 weeks at mid and high RH	Mahl, 1974
Herpes Simplex Virus Type 1	Cell Culture Suspension	Glass	0.2	8 weeks	25	7	Best survival at lowest RH, survival <1 week at mid and high RH	Mahl, 1974
Herpes Simplex Virus Type 1	Cell Culture Suspension	Glass	0.2	8 weeks	37	3	Best survival at lowest RH, survival <1 day at mid and high RH	Mahl, 1974
Poliovirus Type 2 (MEF-1)	Cell Culture Suspension	Glass	4	8 weeks	25	7	Best Survival at lowest RH, survival to [illegible] at mid and high RH	Mahl, 1974
Poliovirus Type 2 (MEF-1)	Cell Culture Suspension	Glass	2.9	8 weeks	37	3	Best Survival at lowest RH examined, survival for < 1 week at mid and high RH	Mahl, 1974
Vaccinia (Lederle)	Cell Culture Suspension	Glass	4	5 weeks	25	7, 98	Similar survival at high and low RH, survival at mid RH to 4 weeks	Mahl, 1974
Vaccinia (Lederle)	Cell Culture Suspension	Glass	3.5 - 4.5	4 weeks	37	3, 55, 93	Similar survival at all RH levels examined	Mahl, 1974
Coxsackie Virus B3	Cell Culture Suspension	Glass	2.5 - 4.25	2 weeks	25	7, 55, 98	Similar survival at all RH levels examined	Mahl, 1974
Coxsackie Virus B3	Cell Culture Suspension	Glass	2 - 4	2 weeks	37	3, 55, 93	Similar survival at all RH levels examined	Mahl, 1974
Rhinovirus Type 2 (HGP)	Cell Culture Vials with 2% bovine serum, diluted with Hank's saline or nasal mucus and Hank's saline	Skin	0 - 0.7	3 hours				Reed, 1975
Rhinovirus Type 2 (HGP)	Cell Culture Vials with 2% bovine serum, diluted with Hank's saline or nasal mucus and Hank's saline	Stainless Steel	1	24 hours				Reed, 1975
Rhinovirus Type 2 (HGP)	Cell Culture Vials with 2% bovine serum, diluted with Hank's saline or nasal mucus and Hank's saline	Enamel Table Top	0.7	24 hours				Reed, 1975
Rhinovirus Type 2 (HGP)	Cell Culture Vials with 2% bovine serum, diluted with Hank's saline or nasal mucus and Hank's saline	Plastic	< 0.5	24 hours				Reed, 1975
Rhinovirus Type 2 (HGP)	Cell Culture Vials with 2% bovine serum, diluted with Hank's saline or nasal mucus and Hank's saline	Stainless Steel	1.75	< 4 days				Reed, 1975
Respiratory Syncytial Virus	Vedium and Pooled Nasal Secretions	Countertops	5	7 - 8 hours	24	35 - 50		Hall, 1980
Respiratory Syncytial Virus	Vedium and Pooled Nasal Secretions	Rubber Gloves	5	5 hours	24	35 - 50		Hall, 1980
Respiratory Syncytial Virus	Vedium and Pooled Nasal Secretions	Cloth Gowns	5	2.5 hours	24	35 - 50		Hall, 1980
Respiratory Syncytial Virus	Vedium and Pooled Nasal Secretions	Paper Towels	5	1 hour	24	35 - 50		Hall, 1980
Respiratory Syncytial Virus	Vedium and Pooled Nasal Secretions	Skin	5	2 hour	24	35 - 50		Hall, 1980
Hepatitis B Virus	Human Plasma	Sterilized Tubes		7 days	25	42	Incubated in Dark	Bond, 1981
Rotavirus SA11	Sterile Water	Countertops	4	45 minutes				Keswick, 1983
Poliovirus Type 1 (LSc)	Sterile Water	Countertops	1.5	90 minutes				Keswick, 1983
Bacteriophage F2	Sterile Water	Countertops	3	90 minutes				Keswick, 1983
Rotavirus SA11	10% Stool Suspension	Countertops	3	90 minutes				Keswick, 1983
Poliovirus Type 1 (LSc)	10% Stool Suspension	Countertops	1.5	90 minutes				Keswick, 1983
Bacteriophage F2	10% Stool Suspension	Countertops	1.5	90 minutes				Keswick, 1983
Parainfluenza Virus Type 1	Hanks Balanced Salt Solution with 0.5% Gelatin	Plastic Petri Plate	3.75	4 days	21	1.5g water/m^3		Parkinson, 1983
Parainfluenza Virus Type 1	Hanks Balanced Salt Solution with 0.5% Gelatin	Plastic Petri Plate	3.75	7 days	21	1.5g water/m^3		Parkinson, 1983
Parainfluenza Virus Type 2	Hanks Balanced Salt Solution with 0.5% Gelatin	Plastic Petri Plate	5.58	7 days	21	1.5g water/m^3		Parkinson, 1983
Parainfluenza Virus Type 2	Hanks Balanced Salt Solution with 0.5% Gelatin	Plastic Petri Plate	5.58	17 days	-22 to -33	0.706 to 0.247g water/m^3		Parkinson, 1983
Parainfluenza Virus Type 3	Hanks Balanced Salt Solution with 0.5% Gelatin	Plastic Petri Plate	5.38	12 days	-22 to -33	0.706 to 0.247g water/m^3		Parkinson, 1983
Parainfluenza Virus Type 3	Hanks Balanced Salt Solution with 0.5% Gelatin	Plastic Petri Plate	5.38	17 days	-22 to -33	0.706 to 0.247g water/m^3		Parkinson, 1983
Herpes Simplex Virus Type 2	Direct from Genital Lesions	Gauze		72 hours				Larson, 1985
Herpes Simplex Virus Type 2	Direct from Genital Lesions	Speculum		18 hours				Larson, 1985
Herpes Simplex Virus Type 2	Direct from Genital Lesions	Gloves		1 hour				Larson, 1985
Herpes Simplex Virus Type 2	Direct from Genital Lesions	Toilet Seat		4 hours				Larson, 1985
Herpes Simplex Virus Type 2	Direct from Genital Lesions	Plastic Container		2 hours				Larson, 1985
Human Rotavirus (Wa)	10% Fecal Suspension in Saline	Stainless Steel	0.01	10 days	4	50		Sattar, 1986
Human Rotavirus (Wa)	10% Fecal Suspension in Saline	Stainless Steel	0.8	10 days	4	85		Sattar, 1986

TABLE 5. VIRUS SURVIVAL ON SURFACES (CONTINUED)

Virus	Suspending Medium	Surface	log_{10} reduction	Time	Temp (°C)	Relative Humidity (%)	Other Conditions	Citation
Human Rotavirus (Wa)	10% Fecal Suspension in Saline	Stainless Steel	0.33	10 days	22	25		Sattar, 1986
Human Rotavirus (Wa)	10% Fecal Suspension in Saline	Vinyl	0.25	10 days	22	25		Sattar, 1986
Human Rotavirus (Wa)	10% Fecal Suspension in Saline	Vilar	0.31	10 days	22	25		Sattar, 1986
Human Rotavirus (Wa)	10% Fecal Suspension in Saline	Glass	0.48	10 days	22	25		Sattar, 1986
Human Rotavirus (Wa)	10% Fecal Suspension in Saline	Stainless Steel	0.46	10 days	22	50		Sattar, 1986
Human Rotavirus (Wa)	10% Fecal Suspension in Saline	Vinyl	0.72	10 days	22	50		Sattar, 1986
Human Rotavirus (Wa)	10% Fecal Suspension in Saline	Vilar	0.72	10 days	22	50		Sattar, 1986
Human Rotavirus (Wa)	10% Fecal Suspension in Saline	Glass	0.69	10 days	22	50		Sattar, 1986
Human Rotavirus (Wa)	10% Fecal Suspension in Saline	Stainless Steel	3.482	2 days	22	85		Sattar, 1986
Human Rotavirus (Wa)	10% Fecal Suspension in Saline	Vinyl	3.128	2 days	22	85		Sattar, 1986
Human Rotavirus (Wa)	10% Fecal Suspension in Saline	Vilar	3.736	2 days	22	85		Sattar, 1986
Human Rotavirus (Wa)	10% Fecal Suspension in Saline	Glass	4.816	2 days	22	85		Sattar, 1986
Human Rhinovirus Type 14	Tryptose Phosphate Buffer	Stainless Steel	1.68	24 hours	20	20		Sattar, 1987
Human Rhinovirus Type 14	Tryptose Phosphate Buffer	Stainless Steel	1.92	24 hours	20	50		Sattar, 1987
Human Rhinovirus Type 14	Tryptose Phosphate Buffer	Stainless Steel	1.2	24 hours	20	80		Sattar, 1987
Human Rhinovirus Type 14	Bovine Mucin	Stainless Steel	2.64	24 hours	20	20		Sattar, 1987
Human Rhinovirus Type 14	Bovine Mucin	Stainless Steel	2.64	24 hours	20	50		Sattar, 1987
Human Rhinovirus Type 14	Bovine Mucin	Stainless Steel	2.16	24 hours	20	80		Sattar, 1987
Human Rhinovirus Type 14	Undiluted Nasal Discharge	Stainless Steel	27.6	24 hours	20	20		Sattar, 1987
Human Rhinovirus Type 14	Undiluted Nasal Discharge	Stainless Steel	10.32	24 hours	20	50		Sattar, 1987
Human Rhinovirus Type 14	Undiluted Nasal Discharge	Stainless Steel	9.12	24 hours	20	80		Sattar, 1987
Enterovirus 70 (J67071)	PBS	Stainless Steel	28	10 hours	20	20		Sattar, 1988
Enterovirus 70 (J67071)	PBS	Stainless Steel	6.5	10 hours	20	50		Sattar, 1988
Enterovirus 70 (J67071)	PBS	Stainless Steel	3.2	10 hours	20	80		Sattar, 1988
Enterovirus 70 (J67071)	PBS	Stainless Steel	0.6	10 hours	20	95		Sattar, 1988
Enterovirus 70 (J67071)	PBS	Stainless Steel	29.8	10 hours	33	80		Sattar, 1988
Enterovirus 70 (J67071)	PBS	Stainless Steel	1	10 hours	33	95		Sattar, 1988
Enterovirus 70 (J67071)	PBS	Stainless Steel	1.3	10 hours	35	95		Sattar, 1988
Human Rotavirus (Wa)	10% Fecal Suspension in Saline	Skin	0.4	60 minutes	22	50		Ansari, 1988
Human Rotavirus (Wa)	10% Fecal Suspension in Saline	Skin	1.1	240 minutes	22	50		Ansari, 1988
Herpes Simplex Virus Type 1	Stock Suspension	Skin	1.5	2 hours	35			Bardell, 1989
Herpes Simplex Virus Type 1	Stock Suspension	Glass	0	2 hours	35		Vaintained in Liquid State	Bardell, 1989
Herpes Simplex Virus Type 1	Stock Suspension	Glass	1.3	2 hours	35		Allowed to Dry, 1.2 log_{10} Inactivation accompanies drying	Bardell, 1989
Parainfluenza Types 1, 2 & 3	Minimal Essential Medium with Earle's Salts	Stainless Steel		10 hours	22		Vaintained in Liquid State; Survival to less than 4 hours when dried	Brady, 1990
Parainfluenza Types 1, 2 & 3	Minimal Essential Medium with Earle's Salts	Laminated Plastic		8 hours	22		Vaintained in Liquid State; Survival to 1 hour when dried	Brady, 1990
Parainfluenza Types 1, 2 & 3	Minimal Essential Medium with Earle's Salts	Facial Tissue		2 hours	22			Brady, 1990
Parainfluenza Types 1, 2 & 3	Minimal Essential Medium with Earle's Salts	Cloth Gown		4 hours	22			Brady, 1990
Parainfluenza Types 1, 2 & 3	Minimal Essential Medium with Earle's Salts	Lab Coat		4 hours	22			Brady, 1990
Parainfluenza Types 1, 2 & 3	Minimal Essential Medium with Earle's Salts	Skin		1 hour	22			Brady, 1990
Herpes Simplex Virus Type 1	Culture Growth Medium	Plastic Doorknobs and Chrome Plated Tap Handles	1.5	2 hours	25			Bardell, 1990
Adenovirus Type 19 (VR1098)	Tissue Culture Medium	Metal	1.1	10 days				Naumann, 1990
Adenovirus Type 19 (VR1098)	Tissue Culture Medium	Plastic	2.4	35 days				Naumann, 1990
Adenovirus Type 19 (VR1098)	Tissue Culture Medium	Paper	2.0	8 days				Naumann, 1990
Adenovirus Type 19 (VR1098)	Tissue Culture Medium	Cloth	1.6	6 days				Naumann, 1990
Hepatitis A Virus (HV-175)	10% Fecal Suspension in Saline	Stainless Steel	0.15	4 hours	5	25		Wehrl, 1991
Hepatitis A Virus (HV-175)	10% Fecal Suspension in Saline	Stainless Steel	0.38	96 hours	20	25		Wehrl, 1991
Hepatitis A Virus (HV-175)	10% Fecal Suspension in Saline	Stainless Steel	1.0	96 hours	35	25		Wehrl, 1991
Hepatitis A Virus (HV-175)	10% Fecal Suspension in Saline	Stainless Steel	0.20	4 hours	5	55		Wehrl, 1991
Hepatitis A Virus (HV-175)	10% Fecal Suspension in Saline	Stainless Steel	0.52	96 hours	20	55		Wehrl, 1991
Hepatitis A Virus (HV-175)	10% Fecal Suspension in Saline	Stainless Steel	1.2	96 hours	35	55		Wehrl, 1991
Hepatitis A Virus (HV-175)	10% Fecal Suspension in Saline	Stainless Steel	0.28	4 hours	5	80		Wehrl, 1991
Hepatitis A Virus (HV-175)	10% Fecal Suspension in Saline	Stainless Steel	0.93	96 hours	20	80		Wehrl, 1991
Hepatitis A Virus (HV-175)	10% Fecal Suspension in Saline	Stainless Steel	3.2	96 hours	35	80		Wehrl, 1991
Hepatitis A Virus (HV-175)	10% Fecal Suspension in Saline	Stainless Steel	0.30	4 hours	5	95		Wehrl, 1991
Hepatitis A Virus (HV-175)	10% Fecal Suspension in Saline	Stainless Steel	1.3	96 hours	20	95		Wehrl, 1991
Hepatitis A Virus (HV-175)	10% Fecal Suspension in Saline	Stainless Steel	2	4 hours	35	95		Wehrl, 1991
Poliovirus Type 1 (LSc)	10% Fecal Suspension in Saline	Stainless Steel	2.4	6 hours	20	25		Wehrl, 1991
Poliovirus Type 1 (LSc)	10% Fecal Suspension in Saline	Stainless Steel	1.2	12 hours	20	95		Wehrl, 1991
Human Rotavirus (CJN)	Distilled Water	Petri Plates	4	6 days	28	50		Ward, 1991
Human Rotavirus (CJN)	Non-fat Dry Milk	Petri Plates	0.5	6 days	28	50		Ward, 1991
Parainfluenza Virus Type 3	Saline Containing Bovine Mucin	Stainless Steel	0.9	60 minutes	22	50		Ansari, 1991
Parainfluenza Virus Type 3	Saline Containing Bovine Mucin	Skin	> 2	60 minutes	22	50		Ansari, 1991
Rhinovirus Type 14	Saline Containing Bovine Mucin	Stainless Steel	0.2	20 minutes	22	50		Ansari, 1991
Rhinovirus Type 14	Saline Containing Bovine Mucin	Skin	0.4	60 minutes	22	50		Ansari, 1991
Hepatitis A Virus (HV-175)	10% Fecal Suspension in Saline	Skin	0.5	4 hours				Wehrl, 1992
Adenovirus Type 5 (McElwain)	Minimal Essential Medium with Earle's Salts, 8% Fetal Bovine Serum	Aluminum Foil		42 days	22	ambient		Gordon, 1993
Adenovirus Type 8 (Ohsy)		Aluminum Foil		28 days	22	ambient		Gordon, 1993
Adenovirus Type 19 (Kowalski)		Aluminum Foil		49 days	22	ambient		Gordon, 1993
Adenovirus Type 5 (McElwain)		Plastic		49 days	22	ambient		Gordon, 1993
Adenovirus Type 8 (Ohsy)		Plastic		49 days	22	ambient		Gordon, 1993
Adenovirus Type 19 (Kowalski)		Plastic		49 days	22	ambient		Gordon, 1993
Adenovirus Type 5 (VR-5)		Aluminum Foil		35 days	22	ambient		Gordon, 1993
Adenovirus Type 8 (VR-1085)		Aluminum Foil		35 days	22	ambient		Gordon, 1993
Adenovirus Type 19 (VR264)		Aluminum Foil		49 days	22	ambient		Gordon, 1993
Adenovirus Type 5 (VR-5)		Plastic		49 days	22	ambient		Gordon, 1993
Adenovirus Type 8 (VR-1085)		Plastic		49 days	22	ambient		Gordon, 1993
Adenovirus Type 19 (VR264)		Plastic		49 days	22	ambient		Gordon, 1993
Cell-Free Human Immunodeficiency Virus	Complete Medium with 10% Fetal Calf Serum	Glass	1	> 70.2 hours	24			van Beuren, 1994
Cell-Associated Human Immunodeficiency Virus	Complete Medium with 10% Fetal Calf Serum	Glass	1	17.5 hours	24			van Beuren, 1994
Herpes Simplex Type 1	Saliva	Penny	5.6	2 hours	22			Bardell, 1994
Herpes Simplex Type 1	Saliva	Nickel	4	2 hours	22			Bardell, 1994
Herpes Simplex Type 1	Saliva	Dime	3.7	2 hours	22			Bardell, 1994
Herpes Simplex Type 1	Saliva	Quarter	3.9	2 hours	22			Bardell, 1994
Herpes Simplex Type 1	Saliva	Glass	0	2 hours	22		Vaintained in a liquid state	Bardell, 1994
Herpes Simplex Type 1	Saliva	Glass	2.6	2 hours	22		Allowed to Dry	Bardell, 1994
Herpes Simplex Type 1	Saliva	Penny	1.9	2 hours	22		Vaintained in a liquid state	Bardell, 1994
Herpes Simplex Type 1	Saliva	Nickel	1.4	2 hours	22		Vaintained in a liquid state	Bardell, 1994
Herpes Simplex Type 1	Saliva	Dime	1.4	2 hours	22		Vaintained in a liquid state	Bardell, 1994
Herpes Simplex Type 1	Saliva	Quarter	2	2 hours	22		Vaintained in a liquid state	Bardell, 1994
Hepatitis A Virus (HV-175)	PBS	Aluminum	1.7	60 days	4	90		Abad, 1994
Human Rotavirus (Ito p13)	PBS	Aluminum	1.7	60 days	4	90		Abad, 1994
Poliovirus Type 1 (LSc)	PBS	Aluminum	5.5	< 10 days	4	90		Abad, 1994
Adenovirus Type 40	PBS	Aluminum	5.3	15 days	4	90		Abad, 1994
Bacteriophage B40-8	PBS	Aluminum	4.9	60 days	4	90		Abad, 1994
Hepatitis A Virus (HV-175)	PBS	Aluminum	1.7	60 days	20	85		Abad, 1994
Human Rotavirus (Ito p13)	PBS	Aluminum	2	60 days	20	85		Abad, 1994
Poliovirus Type 1 (LSc)	PBS	Aluminum	4	< 5 days	20	85		Abad, 1994
Adenovirus Type 40	PBS	Aluminum	4	15 days	20	85		Abad, 1994
Bacteriophage B40-8	PBS	Aluminum	3.2	60 days	20	85		Abad, 1994

TABLE 5. VIRIUS SURVIVAL ON SURFACES (CONTINUED)

Virus	Suspending Medium	Surface	log_{10} reduction	Time	Temp (°C)	Relative Humidity (%)	Other Conditions	Citation
Hepatitis A Virus (HV-175)	PBS	Aluminum	2.2	60 days	20	50		Abad, 1994
Human Rotavirus (Ito p13)	PBS	Aluminum	2.5	60 days	20	50		Abad, 1994
Poliovirus Type 1 (LSc)	PBS	Aluminum	5.5	< 3 days	20	50		Abad, 1994
Adenovirus Type 40	PBS	Aluminum	4	15 days	20	50		Abad, 1994
Bacteriophage B40-8	PBS	Aluminum	3.7	60 days	20	50		Abad, 1994
Hepatitis A Virus (HV-175)	PBS	China	0.9	60 days	4	90		Abad, 1994
Human Rotavirus (Ito p13)	PBS	China	2.2	60 days	4	90		Abad, 1994
Poliovirus Type 1 (LSc)	PBS	China	5.3	60 days	4	90		Abad, 1994
Adenovirus Type 40	PBS	China	5.7	60 days	4	90		Abad, 1994
Bacteriophage B40-8	PBS	China	1.2	60 days	4	90		Abad, 1994
Hepatitis A Virus (HV-175)	PBS	China	2	60 days	20	85		Abad, 1994
Human Rotavirus (Ito p13)	PBS	China	2	60 days	20	85		Abad, 1994
Poliovirus Type 1 (LSc)	PBS	China	5.9	30 days	20	85		Abad, 1994
Adenovirus Type 40	PBS	China	5.1	30 days	20	85		Abad, 1994
Bacteriophage B40-8	PBS	China	2.3	60 days	20	85		Abad, 1994
Hepatitis A Virus (HV-175)	PBS	China	2.1	60 days	20	50		Abad, 1994
Human Rotavirus (Ito p13)	PBS	China	2.1	60 days	20	50		Abad, 1994
Poliovirus Type 1 (LSc)	PBS	China	5	< 3 days	20	50		Abad, 1994
Adenovirus Type 40	PBS	China	> 6	15 days	20	50		Abad, 1994
Bacteriophage B40-8	PBS	China	2.7	60 days	20	50		Abad, 1994
Hepatitis A Virus (HV-175)	PBS	Latex	< 2	60 days	4	90		Abad, 1994
Human Rotavirus (Ito p13)	PBS	Latex	2	60 days	4	90		Abad, 1994
Poliovirus Type 1 (LSc)	PBS	Latex	5.7	30 days	4	90		Abad, 1994
Adenovirus Type 40	PBS	Latex	5.5	15 days	4	90		Abad, 1994
Bacteriophage B40-8	PBS	Latex	3.5	60 days	4	90		Abad, 1994
Hepatitis A Virus (HV-175)	PBS	Latex	2.5	60 days	20	85		Abad, 1994
Human Rotavirus (Ito p13)	PBS	Latex	1.9	60 days	20	85		Abad, 1994
Poliovirus Type 1 (LSc)	PBS	Latex	5.7	15 days	20	85		Abad, 1994
Adenovirus Type 40	PBS	Latex	5.7	15 days	20	85		Abad, 1994
Bacteriophage B40-8	PBS	Latex	3.8	60 days	20	85		Abad, 1994
Hepatitis A Virus (HV-175)	PBS	Latex	4	60 days	20	50		Abad, 1994
Human Rotavirus (Ito p13)	PBS	Latex	2.9	60 days	20	50		Abad, 1994
Poliovirus Type 1 (LSc)	PBS	Latex	5.6	30 days	20	50		Abad, 1994
Adenovirus Type 40	PBS	Latex	5.6	30 days	20	50		Abad, 1994
Bacteriophage B40-8	PBS	Latex	3	60 days	20	50		Abad, 1994
Hepatitis A Virus (HV-175)	PBS	Paper	2.5	60 days	4	90		Abad, 1994
Human Rotavirus (Ito p13)	PBS	Paper	2	60 days	4	90		Abad, 1994
Poliovirus Type 1 (LSc)	PBS	Paper	3.5	60 days	4	90		Abad, 1994
Adenovirus Type 40	PBS	Paper	4.1	60 days	4	90		Abad, 1994
Bacteriophage B40-8	PBS	Paper	3.6	60 days	4	90		Abad, 1994
Hepatitis A Virus (HV-175)	PBS	Paper	3.7	60 days	20	85		Abad, 1994
Human Rotavirus (Ito p13)	PBS	Paper	2	60 days	20	85		Abad, 1994
Poliovirus Type 1 (LSc)	PBS	Paper	5	60 days	20	85		Abad, 1994
Adenovirus Type 40	PBS	Paper	5	60 days	20	85		Abad, 1994
Bacteriophage B40-8	PBS	Paper	3.6	60 days	20	85		Abad, 1994
Hepatitis A Virus (HV-175)	PBS	Paper	3.5	60 days	20	50		Abad, 1994
Human Rotavirus (Ito p13)	PBS	Paper	2.8	60 days	20	50		Abad, 1994
Poliovirus Type 1 (LSc)	PBS	Paper	5.8	60 days	20	50		Abad, 1994
Adenovirus Type 40	PBS	Paper	5.8	60 days	20	50		Abad, 1994
Bacteriophage B40-8	PBS	Paper	3.6	60 days	20	50		Abad, 1994
Foot and Mouth Disease Virus (O_1-BFS)	Tissue Culture Medium	Merino Greasy Wool	4.9	7.0 day	4		Estimated Time to Limit of Detection	McColl, 1995
Foot and Mouth Disease Virus (O_1-BFS)	Tissue Culture Medium	Merino Greasy Wool	5.7	12 days	18		Estimated Time to Limit of Detection	McColl, 1995
Foot and Mouth Disease Virus (O_1-BFS)	Tissue Culture Medium	Merino Greasy Wool	5.84	68 hours	37		Estimated Time to Limit of Detection	McColl, 1995
Foot and Mouth Disease Virus (^{TA}I 1/90)	Tissue Culture Medium	Merino Greasy Wool	3.2	48 days	4		Estimated Time to Limit of Detection	McColl, 1995
Foot and Mouth Disease Virus (^{TA}I 1/90)	Tissue Culture Medium	Merino Greasy Wool	3.9	12 days	18		Estimated Time to Limit of Detection	McColl, 1995
Foot and Mouth Disease Virus (^{TA}I 1/90)	Tissue Culture Medium	Merino Greasy Wool	2.2	33 hours	37		Estimated Time to Limit of Detection	McColl, 1995
Foot and Mouth Disease Virus (O_{Lausy})	Tissue Culture Medium	Merino Greasy Wool	4.1	7.2 days	4		Estimated Time to Limit of Detection	McColl, 1995
Foot and Mouth Disease Virus (O_{Lausy})	Tissue Culture Medium	Merino Greasy Wool	4.2	11 days	18		Estimated Time to Limit of Detection	McColl, 1995
Foot and Mouth Disease Virus (O_{Lausy})	Tissue Culture Medium	Merino Greasy Wool	3.7	57 hours	37		Estimated Time to Limit of Detection	McColl, 1995
Porcine Reproductive and Respiratory Syndrome Virus	Undiluted Stock	Plastic	2	< 1 day	26			Pirtle, 1996
Porcine Reproductive and Respiratory Syndrome Virus	Undiluted Stock	Boot Rubber	2	< 1 day	26			Pirtle, 1996
Porcine Reproductive and Respiratory Syndrome Virus	Undiluted Stock	Stainless Steel	2	< 1 day	26			Pirtle, 1996
Herpes Simplex Virus Type 2 (333)	Earle's Balanced Salts	Skin	0.2	50 minutes	32	50		Graham, 1998
Herpes Simplex Virus Type 2 (333)	Earle's Balanced Salts	Metal	0.7	50 minutes	22	35		Graham, 1998
Herpes Simplex Virus Type 2 (333)	Earle's Balanced Salts	Metal	> 2	50 minutes	32	50		Graham, 1998
Human Papilloma Virus Type 16 (BPV-1 Pseudotype)	Dulbecco's PBS	Tubes	0.5	7 days				Roden, 1997
Bovine Papillomavirus Type 1	Dulbecco's PBS		0.5	7 days				Roden, 1997
Astrovirus Type 2 (L13745)	20% Fecal Suspension	China	3	60 days	4	90		Abad, 2001
Astrovirus Type 2 (L13745)	20% Fecal Suspension	China	4.5	7 days	20	90		Abad, 2001
Astrovirus Type 2 (L13745)	PBS	China	4	60 days	4	90		Abad, 2001
Astrovirus Type 2 (L13745)	PBS	China	3	7 days	20	90		Abad, 2001
Astrovirus Type 2 (L13745)	20% Fecal Suspension	Paper	3	60 days	4	90		Abad, 2001
Astrovirus Type 2 (L13745)	20% Fecal Suspension	Paper	2.2	7 days	20	90		Abad, 2001
Astrovirus Type 2 (L13745)	PBS	Paper	3	3 days	4	90		Abad, 2001
Astrovirus Type 2 (L13745)	PBS	Paper	3	60 days	20	90		Abad, 2001
Foot and Mouth Disease Virus		Soil Surface Under Snow		184 to 195 days	-17 to 5.1°C			Podrezova, 1969; as reported by Bartley, 2002
Foot and Mouth Disease Virus		Soil Surface		> 24 to 36 days	3 to7.5°C	65 to 73		Podrezova, 1969; as reported by Bartley, 2002
Foot and Mouth Disease Virus		Soil Surface, Under leaves, Plant Stems		<5 days to <24 hour	16.6 to 30.6°C	69		Podrezova, 1969; as reported by Bartley, 2002
Foot and Mouth Disease Virus		Soil Surface		< 2 days	up to 34°C ambient and up to 50°C soil	51		Podrezova, 1969; as reported by Bartley, 2002
Foot and Mouth Disease Virus		Soil 1-2cm under Surface		≥ 2 days	up to 34°C ambient and up to 50°C soil	51		Podrezova, 1969; as reported by Bartley, 2002
Foot and Mouth Disease Virus		Straw, Hay, Bran, Cow Hair, Wool, Sand		14 - 168 days	16.7°C	43-68		Bedson, 1927; as reported by Bartley, 2002
Foot and Mouth Disease Virus		Damp Hay, Damp Bran		< 5 days	16.7°C	~100		Bedson, 1927; as reported by Bartley, 2002
Foot and Mouth Disease Virus		Wool		2.5	21°C			Elsner, 1980; as reported by Bartley, 2002

TABLE 6. SURVIVAL OF THE SARS CORONVIRUS UNDER DIFFERENT CONDITIONS

Survival of SARS-CoV

Substrate	Initial viral count (log_{10} PFU)	Specified Conditions	Survival Time	Method of Detection
Solution				
pH effects				
Virus spiked in baby stool[1]	1.00E+03	pH 6-7	3 hours	cell culture
Virus spiked in normal stool[1]	7.50E+03	pH 8	6 hours	cell culture
Virus spiked in diarrheal stool[1]	7.50E+03	pH 9	4 days	cell culture
Temperature effects				
stool[2]	1.00E+03	Room Temperature	≥ 2 days	cell culture
urine[2]	1.00E+03	Room Temperature	≥ 24 hours	cell culture
Virus culture medium + 1% bovine serum[2]	1.00E+04	30-37 °C	≥ 1 hour	cell culture
Virus culture medium + 1% fetal calf serum[2]	1.00E+04	56°C	degradation of titre over time, 10000 infectious units in 15 minutes	cell culture
Virus culture + 2% bovine serum[3]	1.00E+06	-80°C	≥ 4 days	cell culture and RT-PCR
Virus culture + 2% fetal calf serum[3]	1.00E+06	4°C	≥ 4 days	cell culture and RT-PCR
Virus culture + 2% fetal calf serum[3]	1.00E+06	37 °C	< 4 days	cell culture and RT-PCR
Virus culture + 2% fetal calf serum[3]	1.00E+05	56°C	< 30 minutes	cell culture and RT-PCR
Virus culture[4]	1.00E+06	4°C	≥ 21 days	cell culture
Virus culture[4]	1.00E+06	-80°C	≥ 21 days	cell culture
Disinfectant Effects				
Virus in acetone[2]	1.00E+06	Room Temperature	less than 5 minutes	cell culture
Virus in 10% formaldehyde[2]	1.00E+06	Room Temperature	less than 5 minutes	cell culture
Viru in 10% Clorox[2]	1.00E+06	Room Temperature	less than 5 minutes	cell culture
Virus in 75% ethanol[2]	1.00E+06	Room Temperature	less than 5 minutes	cell culture
Virus in 2% phenol[2]	1.00E+06	Room Temperature	less than 5 minutes	cell culture
Surfaces				
Virus culture medium + 1% bovine serum[5]	1.00E+03	Plastic Surface at Room Temperature	≥ 2 days	cell culture
Virus in PBS[5]*	9.00E+04	plastered wall	24 hours	cell culture
Virus in PBS[5]*	9.00E+04	plastic surface	36 hours	cell culture
Virus in PBS[5]*	9.00E+04	formica	36 hours	cell culture
Virus in PBS[5]*	9.00E+04	stainless steel	36 hours	cell culture
Virus in PBS[5]*	9.00E+04	wood	12 hours	cell culture
Virus in PBS[5]*	9.00E+04	cotton cloth	12 hours	cell culture
Virus in PBS[5]*	9.00E+04	pig skin	≥ 24 hours	cell culture
Virus in PBS[5]*	9.00E+04	glass slide	72 hours	cell culture
Virus in PBS[5]*	9.00E+04	paper file cover	24 hours	cell culture
Virus in Sterilized Stool[5]	9.00E+04	plastered wall	36 hours	cell culture
Virus in Sterilized Stool[5]	9.00E+04	plastic surface	72 hours	cell culture
Virus in Sterilized Stool[5]	9.00E+04	formica	36 hours (72 hours)†	cell culture
Virus in Sterilized Stool[5]	9.00E+04	stainless steel	72 hours (36 hours)†	cell culture
Virus in Sterilized Stool[5]	9.00E+04	wood	24 hours	cell culture
Virus in Sterilized Stool[5]	9.00E+04	cotton cloth	24 hours	cell culture
Virus in Sterilized Stool[5]	9.00E+04	pig skin	≥ 24 hours	cell culture
Virus in Sterilized Stool[5]	9.00E+04	glass slide	96 hours	cell culture
Virus in Sterilized Stool[5]	9.00E+04	paper file cover	36 hours	cell culture

Adapted from WHO SARS website. * Reported as virus in tissue culture medium by Tsang, 2003. †Discrepancy in results as reported by Tsang, 2003

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[2] Queen Mary Hospital, The University of Hong Kong, Hong Kong, SAR China

[3] National Institute of Infectious Diseases, Tokyo, Japan

[4] University of Marburg, Germany

[5] Chinese University, Hong Kong