

MATERIAL SAFETY DATA SHEET

SECTION I - INFECTIOUS AGENT

NAME: Human parainfluenza virus

SYNONYM OR CROSS REFERENCE: Human parainfluenza virus (hPIV)

CHARACTERISTICS: Human parainfluenza viruses (hPIV1-4) were originally classified as members of the genus Paramyxovirus within the family Paramyxoviridae; however, according to new findings hPIVs are now divided into two genera: Respirivirus (hPIV 1 and 3) and Rubulavirus (hPIV 2 and 4), under the subfamily Paramyxovirinae. Virus particles are enveloped, pleomorphic, and about 150 to 300 nm in diameter. The lipid envelop is derived from the cell membrane of the infected cell and contains specific glycoprotein spikes which include the hemagglutinin-neuraminidase and the "fusion" proteins. The nucleocapsid core is herringbone-like in shape, and is filamentous since it is studded with the "phosphoprotein" and the "large" protein. The viral genome consists of a linear, non-segmented, negative-sense RNA molecule, usually containing about 15,000 nucleotides.

SECTION II - HAZARD IDENTIFICATION

PATHOGENICITY/TOXICITY: hPIVs generally cause upper and lower respiratory infections. hPIVs are the second most common cause of lower respiratory disease in young children after respiratory syncytial virus. hPIVs primarily attack the cell within the respiratory epithelium. Infected cells are distinguishable from normal cells due to their change in morphology, including focal rounding and increase in size of the cytoplasm and nucleus. In some cases, formation of multinucleated giant cells is also observed. Common symptoms of illness caused by hPIVs include rhinorrhea, cough, croup (acute laryngotracheobronchitis), bronchiolitis, and pneumonia. In some cases high body temperatures of up to ~40 °C are also observed. Croup in children is mainly caused by hPIV1; however, hPIV2 has also been shown to cause croup. Bronchiolitis and pneumonia are caused by all four types of hPIVs, but more cases have been associated with hPIV 1 and 3; however, more cases among hospitalized children are caused by hPIV3. Immunocompromised children and adults may develop more severe symptoms, which may prove fatal. hPIVs have also been linked to acute and chronic neurological diseases, including febrile seizures, encephalitis, ventriculitis, and cluster headaches. Other conditions such as development of apnea and bradycardia due to hPIV infection have also been reported in rare cases in infants. Viremia is most prominent in infected children and the immunocompromised where it has been shown to extend to a few

months in some rare cases. Primary infections tend to be mild or asymptomatic and repeated infections are often needed before any protection develops. Immunity, however, is not long-lasting, as evident from susceptibility to subsequent infections during adulthood.

EPIDEMIOLOGY: hPIVs are common community-acquired respiratory pathogens responsible for upper and lower respiratory infections throughout the world without any ethnic, socioeconomic, gender, age, or geographic boundaries; however, morbidity and mortality rates are higher in developing countries as compared to developed countries. The majority of infections and deaths are observed among young infants, immunocompromised, and elderly individuals. Malnutrition, overcrowding, vitamin A deficiency, lack of breast feeding, and environmental contaminants are factors that can predispose to these infections. It has been estimated that 12% of the 500,000 to 800,000 lower respiratory infection (LRI) cases reported annually in USA are caused by hPIV1-3. It has also been estimated that, worldwide, 10% of the total LRIs in preschool children are caused by hPIVs and 25 to 30% of these result in death. Nosocomial infections are also common, especially among young infants; with hPIV3 being the most frequently transmitted among the four hPIVs. Although the four serogroups of hPIV1-4 have different seasonal peaks, infections caused by these viruses tend to be diagnosed throughout the year. hPIV1 causes biennial epidemics which peak during the fall season. During these epidemics the majority of infections (50%) occur in children aged 7 to 36 months and peaking during the second and third year of life. hPIV2 also causes biennial infections, either with hPIV1 or during alternate years from hPIV1, or annual epidemics, which peak during fall to early winter. The majority of infections (60%) caused by hPIV2 occur in children younger than 5 years of age and peak between the first two years of life. Outbreaks caused by hPIV3 tend to occur yearly and peak during early spring to summer (for North America and Europe). The majority of these infections (40%) occur in children during the first year of life. Little is known about the epidemiology of hPIV4 due to the small number of studies conducted. Generally, it has been noted that the rate of infection is relatively the same in age groups from young infants to adults. An outbreak of hPIV4 within a developmental disabilities unit involving 38 institutionalized children and 3 staff members has also been described.

HOST RANGE: Humans. hPIVs have also been shown to infect many other animals under natural and experimental conditions. Infection has been induced in hamsters, guinea pigs, adult ferrets, non-human primates (chimpanzees, macaques, and squirrel, owl, patas, and rhesus monkeys); however, these infections are almost always asymptomatic.

INFECTIOUS DOSE: Unknown; however, evidence exists that the infectious dose for hPIV1 is small (80 TCID₅₀ of hPIV1). The National Institutes of Health lists the infection dose for parainfluenza 1 to be \geq 1.5 viral units, administered through nasal drops.

MODE OF TRANSMISSION: hPIVs can be transferred through direct person-to-person contact (with infected secretions) and via respiratory droplets. Some sources, however, suggest that person-to-person transfer through contact is less likely since hPIVs do not survive well outside the host, and instead transfer through contaminated surfaces is more likely.

INCUBATION PERIOD: Incubation period for infection by hPIVs is about 2 to 4 days.

COMMUNICABILITY: hPIVs are transmitted between humans through direct person-to-person contact. They are also transmitted by large-droplet spread. The exact period of communicability is not known; however, hPIV3 (the most infective hPIV) is known to shed from the oropharynx for about 3 to 10 days during initial infection. Shedding rates are lower for subsequent infections. In rare cases, hPIV3 has been observed to shed for periods as long as 3 to 4 weeks.

SECTION III - DISSEMINATION

RESERVOIR: Infected humans.

ZOONOSIS: None.

VECTORS: None

SECTION IV - STABILITY AND VIABILITY

DRUG SUSCEPTIBILITY: No antiviral drugs with proven clinical efficacy against hPIVs are currently available. It has been suggested that hPIVs may be susceptible to ribavirin, some interferons, and some protein inhibitors; however, further testing is required to evaluate their efficacy.

SUSCEPTIBILITY TO DISINFECTANTS: hPIVs may be sensitive to hypochlorites (1% sodium hypochlorite), formaldehyde (18.5 g/L; 5% formalin in water), 2% glutaraldehyde, and iodophores (1% iodine). Common detergents, disinfectants, or antiseptic agents are usually efficient enough to remove hPIVs from contaminated surfaces.

PHYSICAL INACTIVATION: hPIVs are sensitive to temperatures of >37 °C, when significant decrease in viral survival is observed, and are almost completely inactivated at 50 °C for about 15 minutes. They are

most stable at 4 °C or under freezing conditions. Viral infectivity is also rapidly lost at pH 3.0 to 3.4, under low humidity, and upon virus desiccation. They are also inactivated by ether.

SURVIVAL OUTSIDE HOST: Existing evidence shows that hPIV1-3 can survive for up to 10 hours on nonporous surfaces and 4 hours on porous surfaces. Survival rate on human skin has been shown to be lower, as hPIV3 loses more than 90% infectivity within the first 10 minutes when placed on fingers. Viral infectivity can be maintained for extended periods of time, up to 26 years for hPIV1, if frozen with the addition of various reagents such as 0.5% bovine serum albumin, skim milk, 5% dimethyl sulfoxide, or 2% chicken serum.

SECTION V - FIRST AID / MEDICAL

SURVEILLANCE: Monitor for symptoms of disease. Isolation of viruses using tissue culture is considered the gold standard among detection techniques for hPIVs. hPIVs demonstrate the best growth in primary monkey kidney (PMK) cell-lines. Secondary cell lines such as LLC-MK2 are also used. Detection of hPIVs in tissue cultures is performed through immunofluorescence (IF) assays, currently the most rapid test to detect hPIVs in tissue cultures. Other tests used for hPIV diagnosis include serological and nucleic acid tests. Serological tests, such as enzyme-linked immunosorbent assays (ELISA), are used to detect antibodies to hPIV. A major problem among these tests is with heterologous cross-reactivity, due to closely related hPIV serogroups, which makes it difficult to differentiate between different serogroups of hPIV during acute infection. Nucleic acid tests (RT-PCR) are generally more sensitive and are used to detect hPIV RNA.

Note: All diagnostic methods are not necessarily available in all countries.

FIRST AID/TREATMENT: Treatment is mainly for symptoms. Immunotherapy may be considered for patients with severe disease.

IMMUNISATION: None available to date; however, several different attempts are being made to prepare a viable vaccine. Currently, two vaccines under investigation include intranasally administered bovine PIV3 (bPIV3) vaccine and cold-adapted PIV3 vaccine; however, further testing and clinical trials are required. hPIV3 is being targeted since it is considered to be the most virulent form of hPIV.

PROPHYLAXIS: None available to date.

SECTION VI - LABORATORY HAZARDS

LABORATORY-ACQUIRED INFECTIONS: No cases of laboratory-acquired infections have been reported to date.

SOURCES/SPECIMENS: Nasopharyngeal samples and secretions (throat swabs, nasopharyngeal swabs, nasal washes, and nasal aspirations) are the primary source of hPIVs. hPIVs have also been isolated from cerebrospinal fluid in some rare cases of meningitis.

PRIMARY HAZARDS: Contact with environmentally contaminated surfaces, direct person- to-person contact with infected secretions, and inhalation of infected respiratory droplets.

SPECIAL HAZARDS: None

SECTION VII - EXPOSURE CONTROLS / PERSONAL PROTECTION

RISK GROUP CLASSIFICATION: Risk Group 2

CONTAINMENT REQUIREMENTS: Containment Level 2 facilities, equipment, and operational practices for work involving infected or potentially infected materials, animals, or cultures.

PROTECTIVE CLOTHING: Lab coat. Gloves when direct skin contact with infected materials or animals is unavoidable. Eye protection must be used where there is a known or potential risk of exposure to splashes.

OTHER PRECAUTIONS: All procedures that may produce aerosols, or involve high concentrations or large volumes should be conducted in a biological safety cabinet (BSC). The use of needles, syringes, and other sharp objects should be strictly limited. Additional precautions should be considered with work involving animals or large scale activities.

SECTION VIII - HANDLING AND STORAGE

SPILLS: Allow aerosols to settle. While wearing protective clothing, gently cover the spill with absorbent paper towel and apply appropriate disinfectant, starting at perimeter and working towards the centre. Allow sufficient contact time before clean up.

DISPOSAL: Decontaminate, either by steam sterilization, incineration, or chemical disinfection, before disposal.

STORAGE: The infectious agent should be stored in appropriately labeled sealed containers at -70 or -20 °C in a non-frost-free freezer.

SECTION IX - OTHER INFORMATION

The above information is believed to be accurate but does not purport to be all inclusive and shall be used only as a guide. The information in this document is based on the present state of our knowledge and is applicable to the product with regard to appropriate safety precautions. It does not represent any guarantee of the properties of the product. ViraTree and its Affiliates shall not assume any liability whatsoever for the accuracy or completeness of the information contained herein. See www.viratree.com and packing slip for additional terms and conditions of sale.