Cell biology of canine parvovirus (cpv) essay

Parvoviruses are among the smallest animal DNA viruses which contain a protein coat (capsid) and a single strand of DNA inside (Vihinen-Ranta et al. 2004). The Parvoridae family is further divided into two subfamilies and these are the Parvovirinae, which infects vertebrates, and the Densovirinae, which infects invertebrates most specifically insects. The subfamily Parvovirinae can be classified into three genera and these include Parvovirus (autonomous parvovirus), Dependovirus, and Densovirus (cited in Siegel et al. 985, Berns 1996). The autonomous parvoviruses are the ones that infect humans as well as other animals such as dogs, cats, mice, and swine (Suikkannen, 2003). Canine Parvovirus belongs to the genera Parvovirus is closely related to feline panleukopenia virus (FPV) with over 98% similarity in their DNA sequences within the capsid protein gene.

Similarity in the DNA sequence of CPV and FPV is mainly attributed to the mutation in a strain of FPV. The mutation in the strain of FPV allowed the virus to extend its host range to canine species (Vihinen-Ranta et al. 2004). The Canine Parvovirus, specifically CPV type 2 emerged in 1978 as a new virus which infects dogs all over the world. The virus became prevalent to canids such as dogs, coyotes and wolves.

Canine Parvovirus causes vomiting, intestinal infection resulting in severe diarrhea, anorexia, rapid dehydration, and death as a result of dehydration. The stool usually contains blood and has a very characteristic odor (hemorrhagic enteritis) . The virus may affect all ages of Canidae (Canine family) but, puppies 6 to 16 weeks of age are more susceptible since puppies younger than this usually acquire protection by the antibodies they received in the mother’s milk. These antibodies can be acquired by the puppies from

the mother’s milk on the first two days; this is denoted as Colostrum. CPV can be transmitted through oronasal exposure to contaminated stool, hair coat, and formites or vectors such as contaminated instruments, insects, and rodents (Skellenger, 2008). Canine Parvovirus is considered to be a host specific virus since it only affects the Canidae or canine family. The host specificity of the CPV can be explained in the cellular level. In order for a viral particle to infect the host cell, it must begin with attachment to the host cell, penetration into the cytosol, uncoating and injection its nucleic acids inside the cell, and targeting the genome and any required accessory proteins toward the correct cellular organelle or compartment for replication (Vihinen-Ranta, 2004).

The virus, Canine Parvovirus in particular can accomplish it through its nonenveloped capsid (protein coat) which is very specific to what host cells it can attach to (Hueffer et al. 003). The entry and infection of the viral particles with nonenveloped capsid is initiated with receptor-mediated endocytosis unlike to those enveloped viruses which entry is initiated with glycoprotein-mediated fusion of the viral envelope with the cellular membrane. The specificity of CPV is controlled on its cell binding abilities.

The CPV use the feline transferrin receptor (TfR) for the binding and infection of feline cells. Binding of CPV to the canine TfRs is controlled by the interactions between the apical domain of the TfR and a raised region of the capsid. Aside form the raised region of the capsid, there are other two regions where the host range-controlling residues are located. Meanwhile, it should be noted that it is the sequences of the residues of the capsid that controls the host specificity of the CPV. The three residues are found to be

different from FPV isolates and CPV type 2. Alterations in the three residues of the capsid allowed the virus to infect canine cells (Hueffer et al. 2003).

CPV specifically binds to the canine transferrin receptor which is only present on canine cells, therefore, causing infection only to canine family (Hueffer et al. 2004).