Investigation on early pathogenesis of KHVD in carp (Cyprinus carpio L.)

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SPF Carp (Cyprinus carpio L.), free from koi herpesvirus (KHV, CyHV-3) and carp pox virus (CyHV-1), were infected with KHV, isolate KHV-E (D 182, provided by Dr. Keith Way, CEFAS, UK), by immersion with 103 TCID50 / ml for 1 hour at 20[U+FFFD]C. After incubation, carp were divided into two groups. Carp were checked twice a day for KHVD. Samples were collected lethally (skin, gill, spleen, kidney, gut, liver and brain) and non-lethally (gill, fins and skin swabs, blood for leukocytes separation) from both groups 1 to 8 hours post infection. From the 1st dpi, samples were collected from both groups until the 4th dpi, where 100% mortality occurred in group 1. Then only group 2 was sampled further on. Mortality started in group 2 from 7th dpi.
KHV copy number quantification was carried out by real-time PCR, modified according to Gilad et al., 2004 and Bergmann et al., 2010. In both cases of KHVD development, the virus was found to be at very high levels of infection to the skin and gill mucus in the first two hours. Up to 4 hours p.i. a massive decrease of viral DNA took part on those superficial organs. There was obviously no replication in these tissues as KHV mRNA was not detected by RT-PCR. Internally KHV replication took place first in gut tissue, then in leukocytes and was brought via blood stream into all other organs. An enrichment of viral DNA was detected in spleen but with an explosive replication in kidney tissue at this stage of infection. During acute KHVD development, a 10,000 times higher viral DNA load was observed from 4th dpi followed by an enormous increase of KHV DNA in all organ tissues, swabs and leukocytes preparations.