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Dioxin and BoHV-1 Infection

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Introduction

2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD), commonly known as dioxin, is a widespread, persistent, and toxic environmental pollutant, which can suppress immune response thereby leading to an increased susceptibility to infectious agents, including Bovine herpesvirus-1 (BoHV-1). BoHV-1 is an important cattle pathogen that can cause infectious bovine rhinotracheitis (IBR), infectious pustular vulvovaginitis (IPV), conjunctivitis and abortions. Following infection in bovine cells (MDBK), TCDD significantly enhances BoHV-1 replication, through an anticipation of BoHV-1-induced apoptosis, by interfering with iron homeostasis and stimulating the expression of bICP0, the major regulatory protein of BoHV-1. As consequence, TCDD may be an additional threat for BoHV-1 infection in animals.

Dioxin is a persistent environmental contaminant which may induce toxicity, both in humans and in animals, like chloracne, hepatotoxicity, reproductive toxicity, carcinogenesis and thymic atrophy. Moreover, it has the potential to provoke immune suppression and increased susceptibility to several viruses both *in vivo* and *in vitro* [1,2]. It has been observed that TCDD enhances mortality in mice infected with herpes simplex II virus, human Coxsackie virus B3 or with different subtypes of influenza A viruses. TCDD also increases virus replication in cells infected with human immunodeficiency virus-1, cytomegalovirus or BoHV-1 [1,2]. BoHV-1, a member of the alpha-herpesvirinae subfamily, is an important pathogen that, in cattle, can cause infectious bovine rhinotracheitis, genital disorders, conjunctivitis, abortions and shipping fever. BoHV-1 initiates the disorder through immune suppression which could render the animals more susceptible to secondary bacterial infections, leading occasionally to death [3].

Following infection in permissive cells (MDBK) BoHV-1 induces apoptosis, which occurs during the late stages of infection [4,5]. While TCDD is able to stimulate BoHV-1-induced apoptosis by stimulating the activation of caspases and modulation of Bcl-2 family members [5]. Interestingly, TCDD drastically decreases telomerase activity when virus-induced apoptosis takes place [6]. The presence of dioxin also up-regulates the levels of infected cell protein 0 encoded by BoHV-1 (bICP0) [7], the main transcriptional regulatory protein of BoHV-1.

BoHV-1, like DNA viruses, requires an iron-replete host to efficiently replicate, so that iron bioavailability is an important component of viral virulence [8]. Analysis of the effects of TCDD on iron metabolism during BoHV-1 infection indicates a modulation of the levels of iron regulatory proteins, transfer in receptor 1 and ferritin. Those changes induce an expansion of the free iron pool, which may promote the onset of BoHV-1 infection and render bovine cells more vulnerable to the virus [9].

Overall, the involvement of multiple pathways may contribute to induce an increase of virus replication during TCDD exposure [10]. Intriguingly, a recent survey in cattle indicated a significant prevalence of IBR on sera collected from farms in dioxin contaminated areas, compared to samples collected in uncontaminated areas [11]. Hence, TCDD may act as an additional risk factor in promoting of viral disease and economic losses in the cattle industry.

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