allied academies Joint Event on
Global Summit on
IMMUNOLOGY AND CELL BIOLOGY
&
Global Congress on
BACTERIOLOGY AND INFECTIOUS DISEASES
June 25-26, 2018 | Amsterdam, Netherlands
Virol Res J 2018, Volume 2

2005-2007 GLOBAL SPREAD OF H5N1: CAUSATIVE ROLE OF BODY BURDEN DIOXIN IN UP-REGULATING GENE OF INFLUENZA VIRUS NS1 PROTEIN IN CHICKEN AND HUMANS IN SOUTHEAST ASIA

I B Tsyrlov
Xenotox, Inc., USA

Agonist-induced recognition of a cognate DNA enhancer dioxin responsive element (DRE) does epitomize wide range of mammalian genes expression mediated via the Ah receptor pathway. The same was postulated also for viral DRE-containing genes expression caused by 2,3,7,8-TCDD (dioxin) in infected human cells. In this study, such mechanistic concept applied to type A influenza virus nonstructural protein 1 binding protein (NS1BP) induction in humans and chicken. The data are presented at genetic, cellular, and population levels. Primers for mutation analysis were constructed for two DRE identified within enhancer region of the IVNS1ABP gene. Treatment of HeLa cell line with 0.1 nM of dioxin resulted in substantial increase of NS1BP protein level. This might add to influenza virus A non-structural protein 1 (NS1) inhibitory effect on cellular interferons, which determines antiviral resistance of emerging H5N1 virus. 2005-2007 H5N1 outbreaks among poultry in China and Vietnam might partially relate to chicken NS1BP, as outbreaks occurred in areas highly contaminated by dioxin-like compounds. Minimal dose of TCDD upregulating human IVNS1ABP gene was estimated moderately above current TCDD blood level in general population. So, in human groups in Southeast Asia exposed to TCDD, its body burden might facilitate spreading of H5N1 if avian flu pandemic were to occur.

xenotoxit@optonline.net