6. Literatur


Birnbaum LS, McDonald MM, Blair PC, Clark AM, Harris MW. Differential toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in C57BL/6J mice congenic at the Ah Locus. Fundam Appl Toxicol. 1990; 15: 186-200.


Crawford RB, Holsapple MP, Kaminski NE. Leukocyte activation induces aryl hydrocarbon receptor up-regulation, DNA binding, and increased Cyp1a1 expression in the absence of exogenous ligand. Mol Pharmacol. 1997; 52: 921-927.


Davis D, Safe S. Immunosuppressive activities of polychlorinated biphenyls in C57BL/6N mice: structure-activity relationships as Ah receptor agonists and partial antagonists. Toxicology. 1990; 63: 97-111.


De Krey GK, Kerkvliet NI. Suppression of cytotoxic T lymphocyte activity by 2,3,7,8-tetrachlorodibenzo-p-dioxin occurs in vivo, but not in vitro, and is independent of corticosterone elevation. Toxicology. 1995; 97: 105-112.


Hahn ME, Karchner SI, Shapiro MA, Perera SA. Molecular evolution of two vertebrate aryl hydrocarbon (dioxin) receptors (AHR1 and AHR2) and the PAS family. Proc Natl Acad Sci U S A. 1997; 94: 13743-13748.


Kamath AB, Camacho I, Nagarkatti PS, Nagarkatti M. Role of Fas-Fas ligand interactions in 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)-induced immunotoxicity: increased resistance of thymocytes from Fas-deficient (lpr) and Fas ligand-defective (gld) mice to TCDD-induced toxicity. Toxicol Appl Pharmacol. 1999; 160: 141-155.


Kerkvliet NI. Recent advances in understanding the mechanisms of TCDD immunotoxicity. Int Immunopharmacol. 2002; 2: 277-291.


Lawrence BP, Meyer M, Reed DJ, Kerkvliet NI. Role of glutathione and reactive oxygen intermediates in 2,3,7,8-tetrachlorodibenzo-p-dioxin-induced immune suppression in C57Bl/6 mice. Toxicol Sci. 1999; 52: 50-60.


Marcus RS, Holsapple MP, Kaminski NE. Lipopolysaccharide activation of murine splenocytes and splenic B cells increased the expression of aryl hydrocarbon receptor and aryl hydrocarbon receptor nuclear translocator. J Pharmacol Exp Ther. 1998; 287: 1113-1118.


Nessel CS, Amoruso MA, Umbreit TH, Gallo MA. Hepatic aryl hydrocarbon hydroxylase and cytochrome P450 induction following the transpulmonary absorption of TCDD from intratracheally instilled particles. Fundam Appl Toxicol. 1990; 15: 500-509.


Pollenz RS, Sattler CA, Poland A. The aryl hydrocarbon receptor and aryl hydrocarbon receptor nuclear translocator protein show distinct subcellular localizations in Hepa 1c1c7 cells by immunofluorescence microscopy. Mol Pharmacol. 1994; 45: 428-438.


Safe SH. Polychlorinated biphenyls (PCBs), dibenzo-p-dioxins (PCDDs), dibenzofurans (PCDFs), and related compounds: environmental and mechanistic considerations which support the development of toxic equivalency factors (TEFs). Crit Rev Toxicol. 1990; 21: 51-88.


Tarkowski S. WHO Coordinated intercountry studies on levels of PCDDs and PCDFs in human milk, Chemosphere 19, 1996, 995-1000.
Thurmond TS, Silverstone AE, Baggs RB, Quimby FW, Staples JE, Gasiewicz TA. A chimeric aryl hydrocarbon receptor knockout mouse model indicates that aryl hydrocarbon receptor activation in hematopoietic cells contributes to the hepatic lesions induced by 2,3,7,8-tetrachlorodibenzo-p-dioxin. Toxicol Appl Pharmacol. 1999; 158: 33-40.


U.S. EPA. Interim procedures for estimating risks associated with exposures to mixtures of chlorinated dibenzo-p-dioxines and dibenzofurans (CDDs and CDFs). 1987; EPA 625/3-87/012.


Van den Berg M, De Jongh J, Poiger H, Olson JR. The toxicokinetics and metabolism of polychlorinated dibenzo-p-dioxins (PCDDs) and dibenzofurans (PCDFs) and their relevance for toxicity. Crit Rev Toxicol. 1994; 24: 1-74.


Whitlock JP Jr, Cooper HL, Gelboin VH. Aryl hydrocarbon (benzopyrene) hydroxylase is stimulated in human lymphocytes by mitogens and benz(a)anthracene. Science. 1972; 177: 618-619.


