Associations between PCDD/Fs, PCBs, OCPs and PBDEs in breast milk and thyroid, growth, and steroid sex hormones

Lin D. Y.[1], Chao H.R.[1], Wang S.L.[2], Päpke O.[3]

[1]Department of Environmental Science and Engineering, National Pingtung University of Science and Technology, Neipu, Pingtung; [2] Division of Environmental Health and Occupational Medicine, National Health Research Institutes, Miaoli; [3] ERGO Research Laboratory, Hamburg, Germany

Abstract

Polychlorinated dibenzo-*p*-dioxins (PCDDs, dioxins), polychlorinated dibenzofurans (PCDFs), polychlorinated biphenyls (PCBs), organochlorine pesticides (OCPs), and polybrominated diphenyl ethers (PBDEs) are environmental endocrine disruptors. We examined relationships between PCDD/PCDFs, PCBs, OCPs, and PBDEs exposure and thyroid, growth, and steroid sex hormones in the sera and cord blood in 42 pregnant women from central Taiwan between 2000 and 2001. Before factors analysis, Concentrations of transthyretin in maternal sera were increased with the increasing of 2,3,3',4,4',5'-HxCB (157) (β = 0.011, p = 0.014). Higher concentrations of T_3 in cord blood were correlated concentrations higher total PCDDs TEQ levels (β = 3.48, p = 0.007). The levels of T_3U in cord blood were increased with decreased 1,2,3,4,7,8-Hexa-CDD levels(β = -0.406, p = 0.004). Meanwhile, after factors analysis, CORTISOL concentrations in cord blood were decreased with elevated levels of Heptachlor epoxide+DDE+DDT (β = -4.22, p = 0.046). IGF1 concentrations were decreased with higher level of DDE+Total non-*ortho* PCBs (β = -20.3, p = 0.026).

Introduction

Polychlorinated dibenzo-*p*-dioxins (PCDDs, dioxins), polychlorinated dibenzofurans (PCDFs), polychlorinated biphenyls (PCBs), organochlorine pesticides (OCPs), and polybrominated diphenyl ethers (PBDEs) are ubiquitous, persistent organic pollutants that act as endocrine disruptors in eco- and biosystems. Various industries, the combustion of solid wastes, and forest fires generate these xenobiotic pollutants. Human milk surveys have established a high body burden for these substances in long-industrialized countries, such as those in Europe[1]. These endocrine disrupting effects upon thyroid function might significantly affect growth and development during fast growth stages of the central nervous system in the human fetus[2,3]. Previous studies only discussed the relationships unique environmental hormones to thyroid, growth, and steroid sex hormones, however, this study tries to discus the statistical relationship with environmental hormones and physical hormones.

Materials and Methods

The study participants were selected from the cohort of the dioxin survey described previously[4]. Subjects were healthy pregnant women recruited from a medical center in the suburban area of Taichung, located in central Taiwan, from December 2000 to November 2001. A mother collected her milk in a chemical-free bottle and froze it in the refrigerator at home. The milk samples of 25 ml were shipped frozen to the ERGO laboratory in Germany for chemical analysis. These environmental hormones were quantified and identified by high resolution gas chromatography equipped with high resolution mass spectrometry (HRGC/HRMS: HP GC5890 II / VG-AutoSpec). Physical hormones were quantified and identified by radioimmunoassay methods.

Spearmen's rank correlation coefficients were initially used to find the correlations between environmental hormones and physical hormones. The measurements of PBDEs were examined to fulfill the normal distribution by the Kolmogorov-Smirnov method. By the linear stepwise regression and factors analyses, we find the relationships with environmental hormones and physical hormones.

Results and Discussion

After factors analyses, BDE-(28+47) had a negative correlation and 2,3,3',4,4',5'-HxCB (157) had a positive correlation with transthyretin levels in maternal blood（p =0.002）. Levels of BDE-99+α- Chlordane+DDE had a negative correlation with TBG levels in cord blood（p =0.013）. PBDEs might affect thyroid hormones, transthyretin and TBG. Higher levels of BDE-(196+197+207) had a positive correlation with free T_4 in cord blood（p =0.046）. PBDEs might bind to the receptor of TBG to induce the secretion of free T_4 in cord blood. PCDD/Fs+PCBs has a positive correlation with T_3 in cord blood（p =0.015）. higher levels of heptachlor epoxide+DDE+DDT had a negative association with CORTISOL in cord blood（p =0.0046）. Hexa-CDD had a negative related to T_3U in cord blood（p =0.003）. BDE196+2,3,3',4,4'-PeCB (105) was in relation to TSHr in maternal blood（p =0.017）. The fact is still unknown based on the results of our epidemiological studies, but these statistical results might offer the new concepts for animal or human studies in the future.

Table1. Significant predictors for transthyretin, cTBG, mTSHr, cT_3, and cT_3U by a multiple linear stepwise regression after factor analysis

	β	p value	A	p value	Adjusted R^2	p value
m[c]Transthyretin						
BDE-(28+47)	-2.10	0.004	13.2	<0.001	0.414	0.002
2,3,3',4,4',5'-HxCB (157)	1.39	0.030				
c[d]TBG						
BDE-99+α- Chlordan+DDE	-15.0	0.013	69.2	<0.001	0.192	0.013
cfree T_4						
BDE-(196+197+207)	0.095	0.046	0.739	<0.001	0.179	0.046
cT_3						
PCDD/Fs+PCBs [a]	7.87	0.015	59.0	<0.001	0.224	0.015
cCORTISOL						
Heptachlor epoxide+DDE+DDT	-4.22	0.046	21.4	<0.001	0.108	0.046
cIGF1						
DDE+Total non-*ortho* PCBs	-20.3	0.026	86.7	<0.001	0.276	0.026
cT_3U						
Hexa-CDD [b]	-2.68	0.003	28.7	<0.001	0.411	0.003
mTSHr						
BDE196+2,3,3',4,4'-PeCB (105)	-0.806	0.017	1.84	<0.001	0.471	0.017

[a] 2,3,7,8-Tetra-CDD, 1,2,3,7,8-Penta-CDD、1,2,3,4,7,8-Hexa-CDD、1,2,3,6,7,8-Hexa-CDD、2,3,4,7,8-Penta-CDF、1,2,3,6,7,8-Hexa-CDF、3,3',4,4',5,5'-HxCB (169)、PCDD/Fs+PCBs TEQ、Total PCDDs TEQ、Total PCDFs TEQ.

[b] 1,2,3,4,7,8-Hexa-CDD, 1,2,3,6,7,8-Hexa-CDD.

[c] m：maternal venous serum

[d] c：cord serum

References

1. Schecter AJ, Piskac AL. Lexington, *KY:University Press of Kentucky* 2001；161–168.

2. Koopman-Esseboom C, Morse D-C, Weisglas-Kuperus N, Lutkeschipholt I-J, Van der Paauw C-G, Tuinstra L-G, *Pediatr Res* 1994；36:468–473.

3. Pluim H-J, Koppe J-G, Olie K, Vd Slikke J-W, Kok J-H, Vulsma T. *Lancet* 1992；339:1303.

4. Chao HR, Wang SL, Lee CC, Yu HY, Lu YK, Päpke O. *Food Chem Toxicol* 2004；42:1299–1308.