Uludag Univ. J. Fac. Vet. Med.
28 (2009), 2: 43-47

A Perspective On the Potential Health Risks Of Dioxin in Human Food

Özen KURŞUN[1] Firdevs MOR[2]

Geliş Tarihi: 22.09.2009
Kabul Tarihi: 26.10.2009

Abstract: It is well known that a wide variety of toxic chemicals are present in the soils, sediments, vegetation, and water. Dioxins are a heterogeneous mixture of chlorinated dibenzo-*p*-dioxins and dibenzofuran (PCDD/F) congeners. The main sources of PCDD/F and PCB are production of chloro-organics and emissions of industrial and municipal incineration and pyrolysis processes. They have been the potential for redistribution and circulation of these compounds into the environment. Transfer into human food occurs by food chain. Humans are ultimately exposed to these compounds primarily through the diet, mainly by consumption of fish, mollusks and dairy products. On the other hand, their health effects such as hyperpigmentation, impairment of immune responses, early infant deaths, hepatotoxicity, carcinogenic effects and, reproductive effects have been intensively studied recently. As growth in toxic chemicals of food, monitoring the food supply will become more important.

Key Words: Dioxin, contamination, food, human health.

Gıdalarda Bulunan Dioksinlerin Potansiyel Sağlık Riskleri

Özet: Bu derleme gıdalarda kirletici olarak bulunan dioksinlerin insanlarda oluşturduğu sağlık risklerini ortaya koymak üzere yapılmıştır. Toksik kimyasalların toprak, sediment, bitki ve suda varlığı çok iyi bilinmektedir. Dioksinler poliklorodibenzo-*p*-dioksin (PCDD/F) ve dibenzofuranların (PCB) türevleridir. Dioksinler, poliklorlu aromatik bileşiklerle benzer yapı, kimyasal ve fiziksel özeliklere sahip bir grup kimyasaldır. Bunlar yanardağ patlamaları ve orman yangınları gibi doğal olaylar sonucu veya kimyasallar, pestisitler, çelik ve boya üretimi, kağıt beyazlatılması, eksoz gazları ve atıkların yakılması gibi insan kaynaklı olaylar sonucu oluşmakta ve kimyasal işlemlerin yan ürünü olarak karşımıza çıkarlar. Örneğin klorlu atıkların yakılması ile dioksin açığa çıkmakta ve havaya karışmaktadır.

PCDD/F ve PCB, klorlu organik maddelerin üretimi, endüstriyel emisyonlar, kentsel atıkların yakılması ve pirozilin meydana getiren tüm işlemlerde ortaya çıkmaktadır. Bu toksik kimyasallar çevreye dağılarak birikmektedir. Özellikle balık, deniz kabukluları ve süt ürünleri gibi besin zincirine dahil olmak suretiyle bu gıdalarla beslenen insanlara geçmektedir. Bunlardan dioksinler, insanlarda hiperpigmentasyon, bağışıklık sisteminin baskılanması, karaciğer hasarı, karsinojenik etkiler ve üreme bozuklukları meydana getirmesi ile erken bebek ölümlerine neden olduğu son yıllarda yapılan araştırmalarla belirtilmiştir. Gıda kontrol analizlerinin yanında besinlerdeki toksik kimyasalların düzenli olarak incelenmesi oldukça önem kazanmaktadır.

Anahtar Kelimeler: Dioksin, Kontaminasyon, Gıda, İnsan sağlığı.

[1] Faculty of Veterinary Medicine, Department of Food and Tecnology, The University of Mehmet Akif Ersoy, 15100, Burdur, Turkey.

[2] Faculty of Veterinary Medicine, Department of Pharmacology and Toxicology, The University of Mehmet Akif Ersoy, 15100, Burdur, Turkey.

Introduction

Dioxins are a mixture of chlorinated dibenzo-*p*-dioxins and dibenzofuran (PCDD/F) congeners[1]. Their ubiquitous occurrence, high chemical and metabolic persistence, and potent toxicity of some of the congeners make them a well recognized class of pollutants[29].

Exposure of humans to dioxins occurs mainly (>95%) through contamination of food[13]. The inhalatory route contributes only a negligible extent. However, accidental contamination of air in occupational setting and of animal feed have led to occasional poisonings of human and livestock, respectively. The present risk assessment will briefly outline the major dioxin exposures and its toxicity, and finally characterize the health risk for humans associated with diatery uptake of a single highly contaminated meal[29]. In general, they are characterised by low solubility in water and on the contrary high solubility in organic solvents and lipids[6].

Dioxin sources

The main sources of PCDD/F and PCB are production of chloro-organics and emissions of industrial and municipal incineration and pyrolysis processes[9]. They are formed in most combustion systems. These can include waste incineration (such as MSW, sewage sludge, medical waste, and hazardous wastes), burning of various fuels, such as coal, wood, and petroleum products[32,34;38]. There is a potential for redistribution and circulation of these compounds into the environment[29]. Potential reservoirs include soils, sediments, vegetation, and water. Transfer into human food occurs by food chain[24]. Dioxin residues are present in water, soils, sediments, etc., they can transfer from waters/soils to aquatic organisms such as plankton, algae, and fish and consequently to birds and marine mammals. Humans are ultimately exposed to these compounds primarily through the diet, mainly by consumption of fish, mollusks and dairy products[5,13]. Dioxins due to early infant deaths and health risks for breast-fed newborns may be lower than previously anticipated[23].

Human Health Risk by Toxicity of Dioxin

The high metabolic stability and lipid solubility of dioxins leads to lifelong accumulation in human[30]. Dioxin is one of the most toxic chemicals known. Even the most conservative of toxicologists says, "Dioxin, has been called the most toxic synthetic chemical known to man. Dioxins, as they are commonly called, are PCDDs and PCDFs are componds whith similar chemical properties. Each compound comprises two benzene rings interconnected by oxygen atoms. In the case of PCDDs, the benzene rings are joined by two oxygen atoms. Much of the environmental behaviour of polychlorinated biphenyls (PCBs) can be related to their physical characteristics. The non-polar nature of PCBs means that they are strongly hydrophobic and thus strongly lipophilic. Dioxin toxicity in humans became almost exclusively known through high occupational exposures or by chemical catastrophes. The following changes were most prominent after human intoxications[35]:the occurrence of chloracne, increases in γ-glutamyltranspeptidase, increases in morning plasma glucose-, triglyceride- and cholesterol levels, further increases in luteinizing hormone and follicle stimulating hormone, but a decrease of testosterone levels, and finally a statistically increased incidence of diabetes[3;7;12;16;18;27].

The susceptibility of other species to the toxic effects of TCDD is variable and depends on adipose tissue to body mass ratio[11;17;31]. In addition, multiple effects on endocrine and growth factor regulated processes have been intensively studied last decade. On the other hand, their health effects such as hyperpigmentation, impairment of immune responses, hepatotoxicity[20], reproductive effects have been intensively studied recently. Their lipophilicity and resistance to degradation leads to bioaccumulation of these compounds in human tissues and fluids. PCBs and selected OCPs are considered as risk factors because of their estrogenic and anti estrogenic properties and potential to act as direct or indirect carcinogens[6;13;21;22-44]. Both estrogenic and antiestrogenic effects have been attributed to various PCBs based upon standard uterotropic animal models[14]. Nesaretnam *et al.*[25] have demonstrated that the non-*ortho* tetrachlorinated biphenyl BZ 77 can act as both an agonist and antagonist of estrogen action, and that this congener can enhance mammary carcinogenesis in the rat[26].

For that reason it seems to be very important to monitor organochlorine pollutants in human population. US Environmental Protection Agency[39;40] describes dioxin as a serious public health treat in the 1960s. The World Health Organisation[42] classes 2,3,7,8-TCDD as a “known” human carcinogen. The current PCDD/PCDF

dietary intake is below the tolerable daily intake (TDI) for dioxins established by the WHO, which for an adult of 70 kg body weight is in the range 70–280 pg TEQ, being even under the lower value of the range[41].

Dioxin contamination in human food

Bocio and Domingo[2] made research concerning the presence of dioxins in fish, seafood, oils, fats, cereals, and dairy products. Fish and seafood (33.7%), oils and fats (15.3%), cereals (14.4%), and dairy products (13.7%) were the most important contributors to this intake. Nizamlıoglu[28] found that feeds and fish meals were contaminated with PCB in Turkey. PCB levels in 121 feeds and 10 fish meals varied between 0.06-900 ppb and 447-2360 ppb respectively. Dioxins distribution in food samples obtained in different countries was shown in Table 1[2].

Tajimi et al.[36] made research the distribution of PCDD/Fs and Co-PCBs in samples of human breast milk collected in Japan. They have found toxic equivalent (TEQ) level of PCDD/Fs (the sum of PCDDs and PCDFs) was 14.9 pg TEQ/g fat, of Co-PCBs 10.6 pg TEQ/g fat, and the total sum of PCDD/Fs and Co-PCBs was 25.6 pg TEQ/g fat. The mean TEQ levels of PCDD/Fs, Co-PCBs, and total PCDD/Fs and Co-PCBs were higher in primiparae than in secundiparae. According to this research, the levels were higher in the subgroup of older mothers. Erdogrul et al.,[8] were measured in 37 individual human milk samples from Kahramanmaras region in Turkey. They were detected PCBs in 8 out of 37 samples.

Çok ve Şatiroglu[4] made research to determine the levels of PCBs in the adipose tissue of women living in Turkey. They have found PCB IUPAC numbers 138, 153 and 180 were the most abundant congeners, each accounting for > 20% of the totalPCB content of adipose tissue.

Conclusion

Chemical methods of analyses are sensitive and spesific, but can be expensive and provide little information on actual or potential biological activity of the contaminants. A government goal to efforts to improve the safety of food is to develop an economical set of monitoring and, inspection practices that will minimize the exposure of consumers to hazardous chemicals. Improved monitoring of food for dioxin and another chemical contaminants is important for minimizing the potential for adverse human health effects due to these contaminants.

Table 1. Dietary intake of PCDD/PCDFs and main food groups contributing to this intake: a summary of recent reports (2001–2005).

Country	Food groups assessed	Main food groups contributing to total PCDD/PCDF dietary intake	PCDD/PCDF intake (pg/day)	Reference
Finland	Fish and fish products, vegetables, flour, meat and meat products, eggs, milk	Fish and fish products, 82.3%; milk, 7.8%; meat and meat products, 7.2%	46 (I-TEQ)	Kiviranta et al. (19)
Japan	Fish and shellfish, meat and meat products, milk and dairy products, vegetables and fruits, pulses, oils and fats	Fish and shellfish, 37%; meat and meat products including eggs, 11.6%; vegetables and cereals, 9.1%	81.9 (WHO-TEQ)	Tsutsumi et al. (37)
United States	Fish, meat and meat products, eggs, vegetables, milk and dairy products	Meat and meat products, 32.1–36.1%; dairy products, 28.7–30.5%; vegetables, 21.3–23.6%	123, males 86, females (WHO-TEQ)	Schecter et al. (33)
Taiwan	Meat, fish and seafood, milk and milk products	Fish and shellfish, 50%	30.7, males 21.9, females (WHO-TEQ)	Hsu et al. (15)
Tarragona, Spain	Vegetables and fruits, meat and meat products, fish and seafood, milk and dairy products, pulses, eggs, oils and fats	Fish and shellfish, 33.7%; oils and fats, 15.3%; cereals, 14.4%; dairy products and cereals, 13.7%	59.6 (WHO-TEQ)	Bocio and Domingo (2)
Belgium	Meat, eggs, fish and seafood, eggs, milk and milk products	Fish and seafood, 40%; milk and milk products, 30%; meat, 21%	65.3 (WHO-TEQ)	Focant et al. (10)
Germany	Participants: 42 small children (mean body weight, 13.4 kg). Food duplicates collected on 7 consecutive days for each child	—	20.9 (I-TEQ)	Wittsiepe et al. (43)

References

1. Abad, E., Llerena, J. J., Saulo, J., Caixach, J., and Rivera, J. (2002). Study on PCDDs/PCDFs and co-PCBs content in food samples from Catalonia (Spain). Chemosphere, 46:1435-1441.

2. Bocio, A., and Domingo, J. L. (2005). Daily intake of polychlorinated dibenzo-p-dioxins / polychlorinated dibenzofurans (PCDD / PCDFs) in foodstuffs consumed in Tarragona, Spain: a review of recent studies (2001–2003) on human PCDD/PCDF exposure through the diet. Environmental Research 97 (2005) 1–9.
3. Clark, J.R., Reinert, K.H., and Dorn, P.B., 1999. Development and application of benchmarks in ecological risk assessment. Environment Toxicol. Chem. 18:1869-1870.
4. Cok, I., and Satiroglu, M. H. (2004). Polychlorinated biphenyl levels in adipose tissue of primiparous women in Turkey. Environment International 30:7 – 10.
5. Darnerud, P. O., Gunnar, E. S., Johannsson, T., Larsen, P. B., and Villuksela, M. (2001). Polybrominated diphenyl ethers: occurrence, dietary exposure and toxicology. Environ. Health Perspect. 109(1):49-68.
6. De Voogt, P., Wells, D. E., Reutergardh, L., and Brinkman, U. A. (1990). Biological activity, determination and occurrence of planar, mono- and di-ortho PCBs. Int. J. Environ. Anal. Chem. 40:1-46.
7. Dewailly, E., Ayotte, P., Bruneau, S., Gingras, S., Belles-Isles, M., and Roy, R., 2000. Susceptibility to infections and immune status in inuit infants exposed to organochlorines. Environmental Health Perspectives 108, 205–211.
8. Erdogrul, O., Covacib, A., Kurtul, N., and Schepens P.(2004).Levels of organohalogenated persistent pollutants in human milk from Kahramanmaras region, Turkey. Environment International 30:259-266.
9. Fiedler, H., 1999. Dioxin and furan inventories—national and regional emissions of PCDD/PCDF. Geneva, Switzerland.
10. Focant, J.F., Eppe G., Pirard, C., Massart, A.C., Andre´, J.E., and De Pauw, E., 2002. Levels and congener distributions of PCDDs, PCDFs and non-ortho PCBs in Belgian foodstuffs. Assessment of dietary intake.Chemosphere 48, 167–179.
11. Geyer, H.J., Scheunert, I., Rapp, K., Gebefu¨ gi, I., Steinberg, C., and Kettrup, A., 1993. The relevance of fat content in toxicity of lipophilic chemicals to terrestrial animals with special reference to dieldrin and 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). Ecotoxicology and Environmental Safety 26, 45–60.
12. Gore, R.H., 1992. The Gulf of Mexico. An assessment of the Risk assessment paradigm for ecological risk assessment. Pineapple Press, Sarasota, FL, USA, 1996.
13. Hallikainen, A., and Vartiainen, T. (1997). Food control surveys of polychlorinated dibenzo-p-dioxins and dibenzofurans and intake estimates.Food Additives and Contaminants 14, 355–366.
14. Hansen L. G. 1998. Stepping backward to improve assessment of PCB congener toxicities. Environ. Health Persp., 106(Suppl.1): 171-189.
15. Hsu, M.S., Cheng, P.S., Ma, E., Chou, U., Chen, L.P., Jone, C.H., Chou, S.S., Cheng, C.C., Yu, C.Y., Liao, C.H., and Ling, Y.C., 2002. A preliminary total diet study on PCDD/Fs-intake from food in Taiwan. Organohalogen Compd. 55, 231–234.
16. Hsu, S.T., Ma, C.I., Hsu, S.K., Wu, S.S., Hsu, N.H., Yeh, C.C., and Wu, S.B., 1985. Discovery and epidemiology of PCB poisoning in Taiwan: a four-year followup. Environmental Health Perspectives, 59: 5-10.
17. IARC, 1997. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Vol. 69. Polychlorinated dibenzo-para-dioxins.International Agency for Research on Cancer, Lyon, pp. 33–630.
18. Iscan, M. (2004). Hazard identification for contaminants Toxicology 205:195–199
19. Kiviranta, H., Hallikainen, A., Ovaskaiens, M.L., Kumpulainen, J., and Vartiainen, T. (2001). Dietary intakes of polychlorinated dibenzo-pdioxins, dibenzofurans and polychlorinated biphenyls in Finland. Food Additives Contam. 18: 945–953.
20. Kociba, R.J., Keyes, D.G., Beyer, J.E., Carreon, R.M., Wade, C.E.,Dittenber, D.A., Kalnins, R.P., Frauson, L.E., Park, C.N., Barnard,S. D., Hummel, R.A., and Humiston, C.G., 1978. Results of a two-year chronic toxicity and oncogenicity study of 2,3,7,8-tetrachlorodibenzop-dioxin in rats. Toxicology and Applied Pharmacology 46, 279–303.
21. Koopman-Esseboom, C., Morse, D.C., Weisglas-Kuperus, N., Lutkeschipholt,I.J., Van der Paauw, C.G., Tuinstra, L.G., Brouwer,A., and Sauer, P.J., 1994. Effects of dioxins and polychlorinated biphenyls on thyroid hormone status of pregnant women and their infants. Pediatric Research 36, 468–743.
22. Koopman-Esseboom, C., Weisglas-Kuperus, N., de, Ridder, M.A., Van der, Paauw, C.G., Tuinstra, L.G., and Sauer, P.J., 1996. Effects of polychlorinated biphenyl/dioxin exposure and feeding type on infants' mental and psychomotor development. Pediatrics 97, 700–706.
23. Kreuzer, P.E., Csanady, G.A., Baur, C., Kessler, W., Papke, O., Greim, H., and Filser, J.G. (1997). 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and congeners in infants: a toxicokinetic model of human lifetime body burden by TCDD with special emphasis on its uptake by nutrition. Archives of Toxicology 71, 383–400.

24. McDonald, T. A. (2002). A perspective on the potential health risks of PBDEs. Chemosphere. 46:745-755.

25. Nesaretnam K., Corcoran D., Dils R. R., and Darbre P. 1996. 3,4,3[prime],4[prime]Tetrachlorobiphenyl acts as an estrogen *in vitro* and *in vivo*. Mol. Endocrinol., 10: 923-936.

26. Nesaretnam K., Hales E., Sohail M., Krausz T., and Darbre P. 1998. 3,3[prime],4,4[prime]-Tetrachlorobiphenyl (TCB) can enhance DMBA-induced mammary carcinogenesis in the rat. Eur. J. Cancer, *34*: 389-393.

27. Newland, M. C. (2002). Neurobehavioral toxicity of methylmercury and PCBs effects-profiles and sensitive populations. Environment Toxicology and Pharmacology 00:1-10.

28. Nızamlıoğlu, F. (1995). Determination of PBC (polychlorobiphenyl) pollution in poultry feeds which is produced in some feed factories by region in Konya.

29. Parzefall, W. (2002). Risk assessment of dioxin contamination in human food. Food and Chemical Toxicology, 40:1185–1189.

30. Poiger, H., and Schlatter, C., 1986. Pharmacokinetics of 2,3,7,8-TCDD in man. Chemosphere 15, 1489–1494.

31. Riss, A., Hagenmaier, H., Weberruss, U., Schlatter, C., and Wacker, R., 1990. Comparison of P. C. D.D/PCDF levels in soil; grass; cow's milk; human blood and spruce needles in area of PCDD/PCDF contamination through emissions from a metal reclamation plant. Chemosphere 21, 1451–1456.

32. Schatzwitz, B., Brandt, G., Gafner, F., Schlump, E., Buhler, R., Hasler, P., and Nussbaumer, T. (1994). Dioxin emissions from wood combustion. Chemosphere, 29: 289-292.

33. Schecter, A., Cramer, P., Boggess, K., Stanley, J., Papke, O., Olson, J., Silver, A., and Schmitz,M. (2001). Intake of dioxins and related compounds from food in the US population. J. Toxicol. Environ. Health 63:1–18.

34. Schramm, K. W., Kaune, A., Lehnardt, R., Hofmaier, A., Henkelmann, B., and Kettrup, A. (1998). Isokinetic sampling of PCFF/F response in low and high volatile fractions of a wood incinerator. Organohalogen Comp., 36:289-292.

35. Sweeney, M.H., Fingerhut, M.A., Calvert, G.M., Piacitelli, L.A., Alderfer, R.J., Davis-King, K., Halperin, W.E., Connally, L.B., and Marlow, D.A., 1993. Noncancer health effects and exposure to 2,3,7,8-TCDD. Organohalogen Compounds 13, 369–374.

36. Tajimi, M.; Watanabe, M., Oki, I., Ojima, T., and Nakamura, Y. (2004). PCDDS, PCDDFs and Co-PCBs in human breast milk samples collected in Tokyo,Japan. Acta Paediatrica 93 (8):1098-1102.

37. Tsutsumi, T., Yanagi, T., Nakamura, M., Kono, Y., Uchibe, H., Iida, T., Hori, T., Nakagawa, R., Tobiishi, K., Matsuda, R., Sasaki, K., and Toyoda, M., (2001). Update of daily intake of PCDDs, PCDFs, and dioxin-like PCBs from food in Japan. Chemosphere 45:1129–1137.

38. USEPA, (1994a). Health Assessment Document for 2,3,7,8-Tetrachlorodi-benzo- p-dioxin (TCDD) and Related Compounds. EPA/600/Bp-92/001c Estimating Exposure to Dioxin-Like Compounds, EPA/600/6-88/005Cb,Office of Research and Development, Washington, DC.

39. USEPA, (1994b). Conbustion Emissions Technical Resource Document (CETRED), Report No. EPA 530-R-94-014, Washington, DC.

40. USEPA, (1996). Federal Register of Environmental Documents, United States Environmental Proteotion Agency.

41. Van Leeuwen, F.X.R., Feeley, M., Schrenk, D., Larsen, J.C., Farland, W., and Younes, M. (2000). Dioxins: WHO's tolerable daily intake (TDI) revisited. Chemosphere 40: 1095–1101.

42. WHO, (1997). Health criteria for polychlorinated dibenzo-*para*-dioxins and dibenzofurans, IPCS International Programme on Chemical Safety, World Health Organisation, Geneva.

43. Wittsiepe, J., Schrey, P., and Wilhelm, M., 2001. Dietary intake of PCDD/F by small children with different food consumption measured by the duplicate method. Chemosphere 43, 881–887.

44. Zober, A., Ott, M.G., Fleig, I., and Heidemann, A., 1993. Cytogenetic studies in lymphocytes of workers exposed to 2,3,7,8-TCDD. International Archives of Occupational and Environmental Health 65, 157–161.