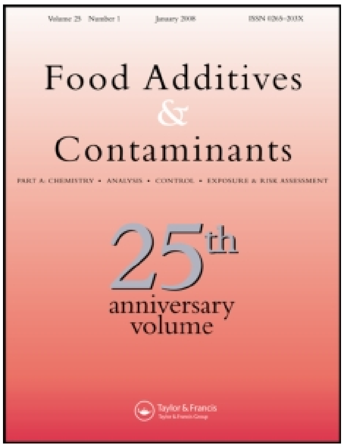


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# Decreasing concentrations of PCDD/Fs in pork based on Korean monitoring in years 2002 and 2005

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# Decreasing concentrations of PCDD/Fs in pork based on Korean monitoring in years 2002 and 2005

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The congener-specific profiles of PCDD/Fs in domestic and imported pork monitored in South Korea in years 2002 and 2005 were compared. Total concentrations of PCDD/Fs decreased from 2002 to 2005, but displayed a similar pattern of congeners. In neither 2002 nor 2005 were either 2,3,7,8-TCDD or 2,3,7,8-TCDF detected. The mean concentrations of PCDD/Fs in domestic and imported pork were 8.29 pg $g^{-1}$ fat from 106 samples in 2002 and 4.03 pg $g^{-1}$ fat from 90 samples in 2005. However, the contribution of PCDDs increased about four times with respect to toxic equivalent (TEQ) level and about 1.5 times in terms of concentration in the monitoring results from 2005 compared with 2002, and the PCDF contribution decreased substantially. This suggests that the main source of dioxins in pork probably changed to a larger portion coming from animal feeds than environmental sources of exposure. The estimated human intakes of PCDD/Fs originating from pork in the South Korean diet were calculated as 0.029 and 0.019 pg TEQ $kg^{-1}$ body weight $day^{-1}$ for the upper bound exposure in 2002 and 2005, respectively. The values represent low intakes when compared with the both Korean tolerable daily intake (TDI) (4 pg TEQ $kg^{-1}$ body weight $day^{-1}$) and World Health Organization TDI (1–4 pg TEQ $kg^{-1}$ body weight $day^{-1}$).

**Keywords:** dioxin; PCDD/Fs; congener; pork; monitoring; dietary intake



## Introduction

Dioxins (polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans, PCDD/Fs) are a group of toxic and persistent organic pollutants (POPs) that cause potential risks for human health. Levels of dioxins in the environment, food, and human body have undergone a several-fold reduction in human exposure and body burdens since 1970, suggesting that efforts to control emissions of dioxins and to reduce exposures have been successful (Hays and Aylward 2003; Charnley and Doull 2005). In Korea, a similar trend is observed in the data from the Ministry of Environment that dioxin emissions from incinerators have been reduced by about 80% during the last few years (Korea Ministry of Environment 2005). The US Environmental Protection Agency (USEPA) has estimated that more than 90% of the human exposures to dioxins occur through food consumption, primarily from food of animal origin (USEPA 2004).

According to a previous study, toxic equivalent (TEQ) levels of PCDD/Fs in pork were higher than in beef or chicken (Kim et al. 2003, 2007). The amount of pork consumption (46.14 g/person $day^{-1}$) has been more than twice the beef consumption (17.39 g/person $day^{-1}$) and almost three times the chicken consumption (15.81 g/person $day^{-1}$) in 2005 in South Korea (Korea Rural Economic Institute 2005). Also, the intake of fat from pork was much higher (about five times) than that from beef or chicken in 2005. Therefore, pork is a primary concern for PCDD/Fs exposure in the Korean diet.

In this study, the monitoring results for the levels of PCDD/Fs in pork in 2005 were compared with the results in 2002 which was previously published (Kim et al. 2007), and the dietary intakes of PCDD/Fs from pork were calculated for both years to indicate if a trend could be observed.

## Materials and methods

### *Materials*

PCDD/Fs and $^{13}C$-labelled standards were obtained from Cambridge Isotope Laboratories (CIL, Andover, MA, USA). High-performance liquid chromatography (HPLC)-grade hexane, nonane, methylene chloride, ethyl acetate, benzene, and toluene were obtained from J.T. Baker (Phillipsburg, NJ, USA).

During 2005, 60 domestic and 30 imported pork samples were collected and analysed. The domestic samples were collected from slaughterhouses nationwide in South Korea. The imported samples were

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collected from randomly selected products by an Automated Import Information System (AIIS) developed by the National Veterinary Research and Quarantine Service, Korea. The imported pork originated from Austria, Australia, Belgium, Canada, Chile, Denmark, France, Hungary, the Netherlands, Poland, and the USA.

The samples collected and analysed in 2002 were from 48 domestic pork and 58 imported pork originated from Austria, Australia, Belgium, Canada, Denmark, France, Hungary, Mexico, Poland, Sweden, and the USA (Kim et al. 2007).

### *Sample preparation and analysis*

Fat was melted from the samples in an oven at 80°C. One sample in a beaker with a glass cover put in an oven at a time to avoid cross-contamination. For analysis, an isotope dilution method was used based on USEPA 1613B protocol. The detailed procedure is described in a previous report (Kim et al. 2007). Extraction was carried out with hexane after spiking $^{13}$C-labeled PCDD/F standards into the proper amount of melted fat. Clean-up was performed using Power-Prep$^{TM}$ (Fluid Management System, Waltham, MA, USA) and $^{37}Cl_4$-2,3,7,8-TCDD was added before the clean-up to check the efficiency of the clean-up process. The extract was concentrated and analysed by HR-GC/MS (Autospec Ultima, Micromass, Manchester, UK) with a capillary column DB-5MS (60 m $\times$ 0.25 mm i.d., 0.25 µm film thickness; J&W Scientific, Folsom, CA, USA). Spiked corn oil matrix blank was also tested and the recoveries fell in the range of 70–98%.

## Results and discussion

Congener-specific concentrations of PCDD/Fs in pork are presented in Table 1. The monitoring results from 2002 and 2005 were from 106 and 90 samples of pork, respectively, which included domestic and imported products. 2,3,7,8-TCDD and 2,3,7,8-TCDF were not found in any of the samples. The concentrations of PCDD/Fs decreased based on the 2005 results compared with 2002 except for 1,2,3,4,7,8-HxCDD and 1,2,3,7,8,9-HxCDD. The mean concentrations of PCDD/Fs in domestic and imported pork were 8.29 pg g$^{-1}$ fat from 106 samples (48 domestic and 58 imported) in 2002 and 4.03 pg g$^{-1}$ fat from 90 samples (60 domestic and 30 imported) in 2005. A single analysis of each sample was made. The average TEQ levels of PCDD/Fs in 2002 were ranged from not detected (n.d.) to 1.36 pg TEQ g$^{-1}$ fat in domestic pork and from n.d. to 1.11 pg TEQ g$^{-1}$ fat in imported pork. The average TEQ levels in 2005 were ranged from n.d. to 0.98 pg TEQ g$^{-1}$ fat in domestic pork and from n.d. to 0.85 pg TEQ g$^{-1}$ fat in imported pork. Thus, noticeable decreases of PCDD/Fs in pork by concentration and by TEQ level were observed in the

Table 1. Mean concentration of polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans (PCDD/Fs) (pg g$^{-1}$ fat) in pork from monitoring years 2002 and 2005.

| | 2002 | | 2005 | |
|---|---|---|---|---|
| Congener | Domestic ($n=48$) | Imported ($n=58$) | Domestic ($n=60$) | Imported ($n=30$) |
| 2,3,7,8-TCDD | n.d. (<0.009) | n.d. (<0.009) | n.d. (<0.009) | n.d. (<0.009) |
| 1,2,3,7,8-PeCDD | n.d. (<0.008) | n.d. (<0.008) | n.d. (<0.004) | 0.03 |
| 1,2,3,4,7,8-HxCDD | 0.05 | 0.08 | 0.39 | 0.25 |
| 1,2,3,6,7,8-HxCDD | n.d. (<0.012) | 0.06 | 0.01 | n.d. (<0.009) |
| 1,2,3,7,8,9-HxCDD | n.d. (<0.011) | n.d. (<0.011) | n.d. (<0.005) | 0.03 |
| 1,2,3,4,6,7,8-HpCDD | 1.03 | 0.68 | 0.26 | 0.07 |
| OCDD | 3.49 | 3.39 | 3.09 | 1.72 |
| 2,3,7,8-TCDF | n.d. (<0.009) | n.d. (<0.009) | n.d. (<0.007) | n.d. (<0.007) |
| 1,2,3,7,8-PeCDF | 0.01 | n.d. (<0.008) | 0.01 | n.d. (<0.005) |
| 2,3,4,7,8-PeCDF | 0.12 | 0.03 | 0.09 | 0.02 |
| 1,2,3,4,7,8-HxCDF | 0.67 | 0.11 | 0.17 | 0.01 |
| 1,2,3,6,7,8-HxCDF | 0.38 | 0.19 | 0.08 | 0.05 |
| 2,3,4,6,7,8-HxCDF | 0.16 | 0.28 | 0.05 | n.d. (<0.006) |
| 1,2,3,7,8,9-HxCDF | 0.04 | 0.33 | n.d. (<0.004) | n.d. (<0.004) |
| 1,2,3,4,6,7,8-HpCDF | 2.73 | 0.45 | 0.48 | 0.18 |
| 1,2,3,4,7,8,9-HpCDF | 0.09 | 0.45 | n.d. (<0.010) | n.d. (<0.010) |
| OCDF | 0.24 | 1.63 | 0.19 | 0.08 |
| Sum of PCDDs | 4.57 | 4.21 | 3.75 | 2.10 |
| Sum of PCDFs | 4.44 | 3.47 | 1.07 | 0.34 |
| Sum of PCDD/Fs | 9.01 | 7.68 | 4.82 | 2.44 |
| TEQ (PCDD/Fs) | 0.23 | 0.14 | 0.13 | 0.08 |

Note: *n*, Numbers of samples; n.d., not detected. The limit of detection (LOD) is given in parentheses.

2005 results versus those from 2002. These concentrations were calculated using zero for non-detects, and toxic equivalency factors, TEFs (World Health Organization (WHO) 1998) were applied for the TEQ levels. The US Food and Drug Administration (USFDA) has stated that assuming non-detects are equal to half of the limit of detection (LOD/2) or LOD may overestimate the levels of PCDD/Fs in food and lead to overestimates of dietary exposure, too (USFDA 2004). The LODs for the PCDD/Fs ranged from 0.004 to 0.020 pg g$^{-1}$ fat in this study.

Table 2 presents the reported average TEQ levels of PCDD/Fs in pork from several countries. The TEQ levels in this study might be closer to the levels of the other countries if the calculation of 'n.d.' values was treated in the same way.

Figure 1 presents the contribution of PCDDs and PCDFs in pork consumed in South Korea at the TEQ level compared with concentration. The contribution of PCDDs increased four times in the TEQ level and 1.5 times in the concentration from the monitoring results in 2005 compared with that in 2002. Thus, the cause in the overall decrease in the concentrations is from the greatly decreasing PCDF levels.

Figure 2 shows the changes of dioxin contamination with respect to the contributions of PCDDs and PCDFs in domestic and imported pork. The results indicate that the pattern of dioxins contamination in domestic pork became closer to the pattern in imported pork, although the ratio of domestic samples monitored increased in 2005. The samples of domestic pork occupied 55% and 67% of the total samples in 2002 and 2005, respectively. The percentage contribution of PCDDs to the total concentrations increased about five times in both domestic and imported pork from 2002 to 2005. The contribution of PCDFs was

Table 2. Reported toxic equivalent (TEQ) levels of polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans (PCDD/Fs) in pork in various countries.

| Country | pg TEQ g$^{-1}$ fat | Remarks | References |
|---|---|---|---|
| Korea | 0.13[a] | n.d. = 0, 2005 | Present study |
| Korea | 0.23[a] | n.d. = 0, 2001–2002 | Kim et al. (2007) |
| Belgium | 0.17 | n.d. = LOD, 2000–2001 | Focant et al. (2002) |
| Greece | 0.39 | n.d. = LOD, 2002 | Papadopoulos et al. (2004) |
| Netherlands | 0.47 | PCDD/Fs plus dioxin-like PCBs, 1999 | Baars et al. (2004) |
| Portugal | 0.47 | n.d. = LOD, 2004 | De Sousa Martins et al. (2005) |
| Slovakia | 0.3 | 2001 | Chovancovaá et al. (2005) |
| Spain | 0.3 | 2002 | Bocio and Domingo (2005) |
| USA | 0.16 | n.d. = 0, 2002–2003 | Huwe et al. (2004) |

Note: [a]Domestic pork in South Korea; n.d., Not detected; PCB, polychlorinated biphenyl.

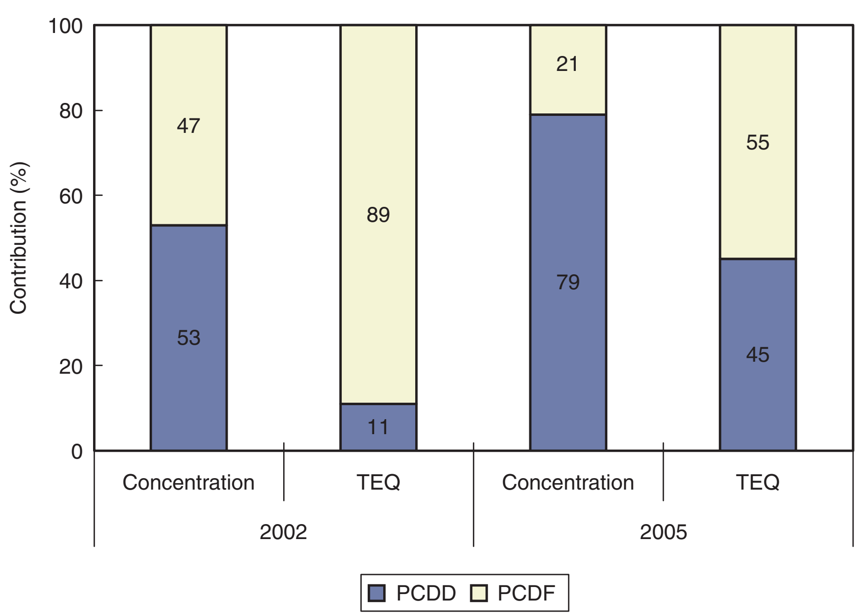


Figure 1. Contribution of polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) at concentration versus toxic equivalent (TEQ) level in pork consumed in Korea.

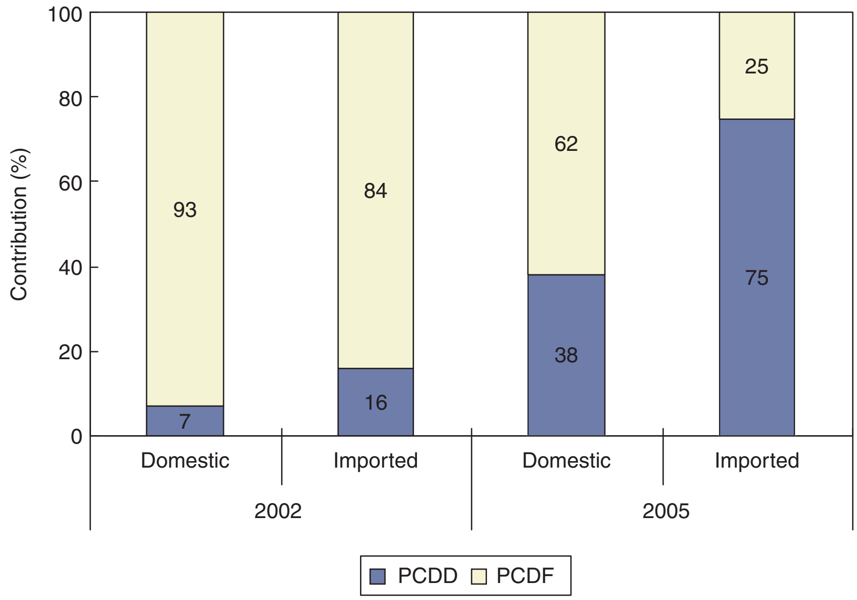


Figure 2. Contribution of polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) at toxic equivalent (TEQ) level in domestic and imported pork.

Table 3. Estimated dietary intakes of polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans (PCDD/Fs) from domestic and imported pork in 2002 and 2005.

| | Concentration (pg TEQ $g^{-1}$ fat) | | | | Dietary intake of PCDD/Fs (pg TEQ $kg^{-1}$ body weight $day^{-1}$)[b] | |
|---|---|---|---|---|---|---|
| Year | Lower bound[c] | Upper bound[c] | Daily consumption[a] (g) | Fat content[a] (g) | Lower bound | Upper bound |
| 2002 | 0.18 | 0.22 | 45.21 (3.0)[d] | 7.93 (9.3)[d] | 0.024 | 0.029 |
| 2005 | 0.11 | 0.14 | 46.14 (3.0) | 8.10 (9.1) | 0.015 | 0.019 |

Notes: [a]Data are from the Food Balance Sheet published by the Korea Rural Economic Institute (2002 and 2005).
[b]Body weight = 60 kg.
[c]Lower and upper bounds were calculated with non-detects equal to zero and LOD, respectively.
[d]Percentage of the total consumption of food or fat are given in parentheses.
TEQ, toxic equivalent.



more dominant in domestic and imported pork in 2002, however, PCDDs occurred in 75% of the imported pork samples in 2005. This change probably means that the direct effect of environmental sources to the residual dioxins in pork became less than that of feed-related sources (Abad et al. 2002; Eljarrat et al. 2002; Kim et al. 2007).

The estimated dietary intakes of PCDD/Fs from pork in Korea are presented in Table 3. The TEQ levels of PCDD/Fs were calculated for the lower and the upper bound with non-detects equal to zero and LOD, respectively. The figures for daily consumption of pork and its fat content were taken from Food Balance Sheet published by Korea Rural Economic Institute. The estimated dietary intakes of PCDD/Fs in upper bound were 0.029 in 2002 and 0.019 pg TEQ $kg^{-1}$ body weight $day^{-1}$ in 2005. The dietary intakes decreased over 35% from 2002 to 2005 even though pork consumption increased. A tolerable daily intake (TDI) established by the WHO is 1–4 pg TEQ $kg^{-1}$ body weight $day^{-1}$. Therefore, the dietary intakes of PCDD/Fs in pork from this study make up only 2.9% in 2002 and 1.9% in 2005 of the lowest WHO-TDI (1 pg TEQ $kg^{-1}$ body weight $day^{-1}$). Korean daily intake of PCDD/Fs from pork was 0.5% of the total intake of PCDD/Fs from food compared with 33.8% of fish, 7.4% of shellfish, 22.3% of cereals, and 19.3% of vegetables (Lim et al. 2002).

## Conclusions

PCDD/Fs in pork were found to decrease in the monitoring results from 2005 compared with the results from 2002. The decreasing trend for pork in this study does not necessarily represent decreasing of PCDD/Fs in the environment or animal feeds. However, those changes in concentrations first appear in pork, which has a relatively short life span until slaughter. Although Koreans eat pork with a high fat content, the estimated dietary intakes of PCDD/Fs through pork were very low. The results of this study give a positive indication, even though further monitoring is needed to confirm that the decreasing trend extends to the sources of PCDD/Fs in livestock and other types of meat.

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