Developmental dental defects in children exposed to PCBs

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Introduction

Developing enamel is sensitive to a wide range of local and systemic disturbances. Because of the absolute metabolic stability of its structure, changes in enamel during its development are permanent in nature.

Polychlorinated biphenyls (PCB) have been shown to disturb tooth development in experimental animals[1], but only limited amounts of data exist on their adverse effects in humans. Dental changes such as mottled, chipped, carious, and neonatal teeth have been reported in accidentally exposed humans[2,3]. Nevertheless, co-contamination with polychlorinated dibenzo-furans (PCDFs) was largely responsible for the overall toxicity[4]. Alaluusua et al.[5] found that developmental dental defects were correlated with the total exposure to polychlorinated aromatic hydrocarbons via mother´s milk. The correlation was strong with exposure to prevailing levels of polychlorinated dibenzo-p-dioxins (PCDD) and furans (PCDF) but weak with exposure to PCBs alone. In our previous study we have shown developmental dental defects in children exposed to PCBs alone[6], suggesting that the developing human teeth are vulnerable to PCBs.

In the Michalovce region of eastern Slovakia, PCBs from a chemical plant manufacturing Delors contaminated the surrounding district[7]. The total serum PCB levels in samples from the general population there exceeded by several times the background levels in subjects living in a comparable unexposed Svidnik district. PCB levels in breast milk samples in the Michalovce region were the highest in Slovakia[8]. Levels of toxic polychlorinated aromatics (PCDFs, PCNs, and planar PCBs) in technical Delors were high[9].

The aim of this study was to evaluate the effects of long-term exposure to PCBs, measured at the individual level, on developmental dental defects in children in eastern Slovakia.

Methods and Materials

We examined 432 children aged 8-9 years (208 from the Michalovce, and 224 from the Svidnik area). Dental examinations were carried out in September 2002 by two calibrated dentists, using a standard mouth mirror and dental probe. The teeth were not cleaned or dried prior to examination. Developmental defects of enamel were assessed using the Developmental Defects of Enamel (DDE) Index[10] on buccal surfaces of permanent teeth. Three main types of developmental defects of enamel were recorded; demarcated opacities, diffuse opacities, and hypoplasia. The extent of the defects was recorded in thirds of the surface area. Questionnaires that were completed by the parents provided information on various confounding factors and modifiers (e.g. place of residence during tooth development, parity, duration of breast-feeding, children's diseases, medications, fluoride intake). Analyses of blood samples for organochlorines were made by high resolution gas chromatography using electron capture detection.

All the data were analysed using the SPSS 9.0 statistical software package.

Results and Discussion

The description of the children population examined, and ranges of the quartiles of the sum of serum PCBs concentration ng/g serum lipids are presented at the Dioxin 2004.

Fig. 1. Percentage of teeth with any enamel defect with different extensions on labial surfaces according to the serum total PCB concentration categorized in quartiles

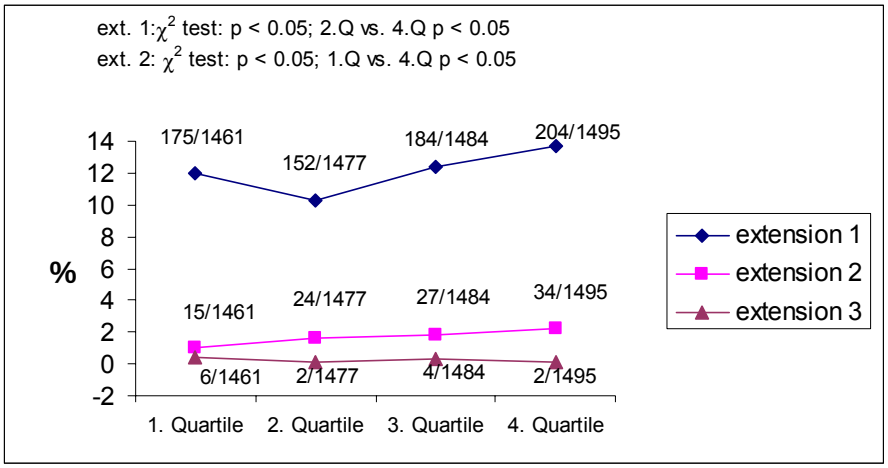
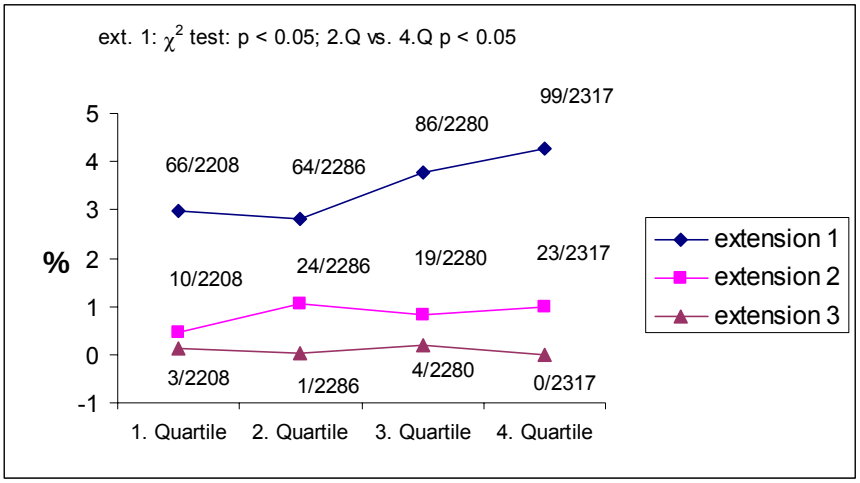


Fig. 2. Percentage of teeth with demarcated opacity with different extensions according to the serum total PCB concentration categorized in quartiles



The percentage of teeth affected with developmental dental defect was significantly higher in higher quartiles. Furthermore, the extent of the defects was also significantly greater (Fig. 1). When excluding diffuse opacities and hypoplasia from the final statistical analysis, there was a significantly higher percentage of teeth affected with demarcated opacities in the children in higher quartiles (Fig. 2). The results are in accordance with our previous study[6] where the difference in prevalence of dental defects between PCB exposed and unexposed children was mostly due to demarcated opacities.

This study demonstrated a dose-response relationship between PCB exposure and developmental dental defects in children. It confirmed our previous findings that long-term exposure to PCBs may cause developmental dental defects. Further evaluation of the mechanism of this toxicity is needed.

Acknowledgements

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References

1 Hashiguchi I., Akamine A., Hara Y., Maeda K., Anan H., Abe T., Aono M. and Fukuyama H. (1985) Fukuoka Igaku Zasshi 76, 221.

2 Rogan W.J., Gladen B.C., Hung K.L., Koong S.L., Shih L.Y., Taylor J.S., Wu Y.C., Yang D., Ragan N.B. and Hsu C.C. (1988) Science 241, 334.

3 Wang S.L., Chen T.T., Hsu J.F., Hsu C.C., Chang L.W., Ryan J.J., Guo Y.L. and Lambert G.H. (2003) Environ. Res. 93, 131.

4 Masuda Y. (1996) Chemosphere 32, 583.
5 Alaluusua S., Lukinmaa P.L., Torppa J., Tuomisto J. and Vartiainen T. (1999) Lancet 353, 206.
6 Jan J., Vrbic V. (2000) Caries Res. 34, 469.
7 Kočan A., Petrík J., Jursa S., Chovancová J. and Drobná B. (2001) Chemosphere 43, 595.
8 Petrík J., Drobná B., Kočan A., Chovancová J. and Pavúk M. (2001) Fresenius Environ. Bull. 10, 342.
9 Taniyasu S., Kannan K., Holoubek I., Ansorgova A., Horii Y., Hanari N., Yamashita N. and Aldous K.M. (2003) Environ. Pollut. 126, 169.
10 FDI Commission on Oral Health, Research & Epidemiology. (1992) Int. Dent. J. 42, 411.