NEONATAL AND CHILDHOOD TEETH IN RELATION TO IN UTERO EXPOSURE TO PCBS/PCDFS - THE OBSERVATIONS FROM YU-CHENG CHILDREN IN TAIWAN

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Introduction

Children with in utero exposure to polychlorinated biphenyls (PCBs), from maternal ingestion of PCBs-contaminated oil in the Japanese Yusho episode, were reported to bear abnormal-shaped tooth roots, retarded eruption and reduced numbers of permanent teeth[1]. Early eruption of deciduous teeth was also reported in these Yusho children[2,3,4]. Based on the observation of 18 Yucheng children exposed in utero to PCBs in Taiwan, about 50% of the girls and 10% of the boys had missing permanent teeth germ[5]. However, the neonatal teeth were not described in the above studies. We conducted this study to determine the effect of in utero PCB exposure on neonatal and childhood teeth, both deciduous and permanent, in Yucheng children.

Methods and Materials

In 1979, over 2000 Taiwanese people ingested rice oil contaminated with PCBs and their heat degradation products, mainly PCDFs. We followed cognitive development yearly for 116 Yucheng children who were in utero during or after the period of oil contamination since 1986. An unexposed child was selected as control for each Yucheng child, matching for 1979 neighborhood, age, sex, mother's age, parents' educational level and occupation. In February 1992, Yucheng and their control children were recruited for dental examination. Complete dental evaluations were carried out by the same experienced dentist, who was blinded to the children's exposure status. Each subject's dental history was recorded with particular attention to neonatal and deciduous teeth. Dental X-ray films were read blindly by two skillful dentists with agreement upon the diagnosis achieved. ANOVA T-test and chi-square test, including Fisher's exact 2-tail test, were utilized for the comparisons between the Yucheng and the control group. JMP software, a SAS window package, was adopted for all the analyses.

Results and Discussion

The types of tooth defects and related factors in PCBs-exposed and control groups of both genders are summarized in table 1. Age and gender distributions were similar between exposed and control groups. In the exposed groups, there were 5/38 (13%) girls and 2/35 (5.7%) boys developed neonatal teeth and none was observed in the control group. Similar findings were reported in a study by Rogan et al[6] in which 11 in 127 children (8.7%) bore teeth at neonatal period. The percentages of teeth with congenitally missing and rotation were found to be significantly increased in the exposed group ($p<0.05$). The numbers of teeth with developmental defects, such as fusion, microdontia, peg teeth, enamel hypoplasia, and impaction, were also greater in exposed than in control group ($p<0.1$). In addition to neonatal teeth development, girls tended to have greater numbers of teeth with cross bites, rotation, pigmentation at tongue, gingivitis and hyperplasia than boys, particularly among exposed subjects.

Table 1. Distribution of defects and related factors in exposed and control groups by gender

Factor/Defects	Exposed (n=73)			Control (n=75)			P[1]
	Boy(35)	Girl(38)	Tot.(%)	Boy(34)	Girl(41)	Tot.(%)	
Age (SD)	9.34(2.0)	9.28(2.0)	8.6(2.4)	9.4(2.1)	9.3(2.1)	9.3(2.1)	N.S.
Congenital Missing (%)	10(28)	9(24)	21(29)	1(2.9)	1(2.4)	2(2.7)	<0.01
Neonatal Teeth(%)	2(5.7)	5(13.1)	7(9.6)	0	0	0	<0.01
-Supernumberous(%)	1(2.8)	2(5.3)	3(4.1)	0	0	0	<0.1
-Deciduous(%)	1(2.8)	2(5.3)	3(4.1)	0	0	0	<0.1
No. of Deciduous Teeth (SE)	8.3(1.1)	7.8(1.2)	8.0(0.81)	9.2(1.1)	7.7(1.1)	8.4(0.80)	N.S.
No. of Permanent Teeth (SE)	14.9(1.3)	16.5(1.4)	15.7(0.98)	14.8(1.4)	16.6(1.4)	15.8(0.96)	N.S.
Estimated Dental Age (SD)	8.4(2.6)	8.7(2.5)	8.0(2.8)	8.4(2.8)	8.8(2.7)	8.6(2.7)	N.S.
Tooth Chipping (%)	1(2.8)	0	1(1.4)	0	0	0	N.S.
Open bite(%)	1(2.8)	1(2.6)	2(2.7)	0	1(2.4)	1(1.3)	N.S.
Cross bite[2](%)	8(23)	13(34)	25(34)	3(8.8)	10(24)	13(17)	<0.1
Developmental Defects[3](%[4])	10(28)	6(16)	18(25)	0	4(9.8)	4(5)	<0.1
Rotation(%)	1(2.8)	12(32)	14(19)	0	2(4.9)	2(2.7)	<0.05
Tonguetie & Pigmentation(%)	1(2.8)	2(5.3)	3(4.1)	1(2.9)	0(0)	1(1.3)	<0.2
Gingivitis & Hyperplasia(%)	2(5.7)	4(10.5)	6(8.2)	1(2.9)	2(4.9)	4(4)	N.S.

[1] Comparisons between the exposed and control groups (boys and girls combined), Chi-square test or Fisher's exact 2-tail test for cells' numbers less than 5. N.S. for $p>0.2$.

[2] The anterior and posterior cross bites were combined because of same pattern in the difference between the 2 groups.

[3] Other developmental defects combining fusion, microdontia, peg teeth, enamel hypoplasia, and impaction because of the same pattern (higher rates were found in exposed group but not significant when showing separately).

[4] Total numbers of teeth with the defects were divided to the total numbers of subjects showing in percentages. This is applied to the 3 defects below.

The mean numbers of teeth in exposed and control groups according to age was presented in figure 1. Our study showed that exposed groups had a significantly smaller number of deciduous teeth than control group at the age of 9 and 10 years (left panel). This might have resulted from earlier eruption and utilization, or weaker structure, of deciduous teeth in the exposed groups. Normally the first and second deciduous molars could sustain until the age of 10~11 years[7]. In the exposed group, however, these teeth were lost at a younger age. We also observed that the exposed group had significantly greater numbers of permanent teeth than the control group at the age of 9 and 10 years (right panel). This might result from earlier loss of deciduous teeth and earlier eruptions of permanent teeth in the these exposed children. In addition, the numbers of permanent teeth tended to be greater in the control than in the exposed groups after the age of 11 years. This might be the result of a greater number of caries, extraction, or tooth-loss in these children. Congenital defects of permanent teeth germ were reported in Yucheng children previously[2]. The difference in the number of permanent teeth between exposed and control groups may increase after the age of 12 years. It is worthwhile to follow these children to determine whether such difference persists and/or increases.

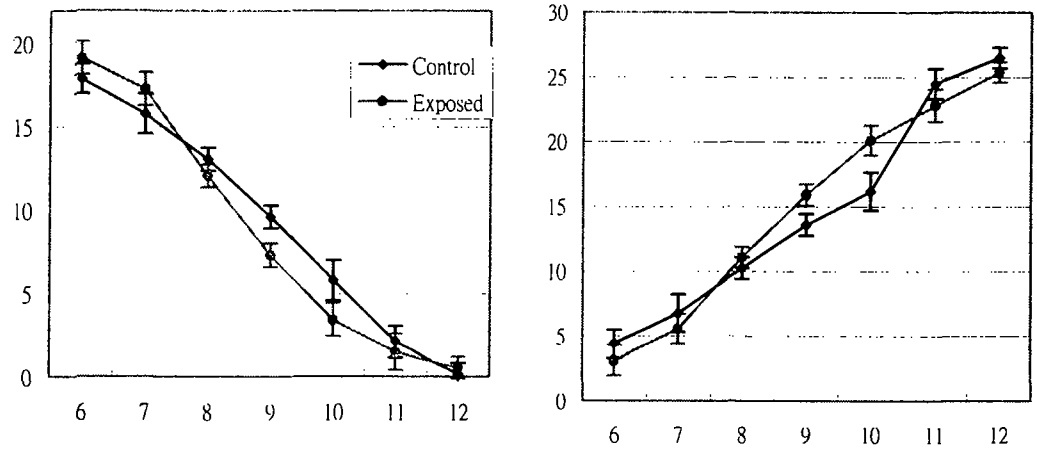

Figure 1. Mean numbers of teeth in exposed and control groups according to age (left panel: *deciduous* teeth; right panel: *permanent* teeth).

Our present study demonstrated that PCBs/PCDFs might induce neonatal teeth development as well as abnormal deciduous teeth defects. However, the mechanism of this toxicity is still obscure and needs further investigation. Dental defects have been suggested to be a potential indicator for PCDD/Fs exposure[8]. In addition, abnormal calcification of the skull has been reported in Yusho

babies[5]. Since PCBs/PCDFs is a known endocrine disruptor, its disruption on hormones, such as estrogen and alternated calcium metabolism[9], may be considered as a mechanism underlying such developmental defects.

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References

1. Fukuyama F., Anan Y., Akamine A. and Aono M. (1979) Fukuoka Acta Med. 70, 187.
2. Funatsu I., Yamashita F., Yoshikane T., Funatsu T., Ito Y. and Tsugawa S. (1971) Fukuoka Acta Med. 62, 139.
3. Yamaguchi A., Yoshimura T. and Kuratsune M. (1971) Fukuoka Acta Med. 62, 117.
4. Yamashita F. and Hayashi M. (1985) Environ Health Perspect. 59, 41.
5. Lan S.-J., Yen Y.-Y., Ko Y.-C. and Chen E.-R. (1989) Bull Environmen Contam Toxicol. 42, 931.
6. Rogan W.J., Gladen B.C., Hung K.-L., Koong S.-L., Shih L.-Y., Taylor J.S., Wu Y.-C., Yang D., Ragan B. and Hsu C.-C. (1988) Science. 241, 334.
7 Stewart R.E., Barber T.K., Troutman K.C. and Wei S.H.Y. (1982), Pediatric dentistry - Scientific foundations and clinical practice, Mosby, St. Louis
8. Alaluusua S., Lukinmaa P.-L., Torppa J., Tuomisto J. and Vartiainen T. (1999) Lancet. 353, 206.
9. Porterfield, S.P. (2001) in: Endocrine Physiology (Porterfield, S.P., Ed.), Mosby, London, 107-129.