The prevalence of molar incisor hypomineralization (MIH) in a group of children in a highly polluted urban region and a windfarm-green energy island

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Backround. Children's developing teeth may be sensitive to environmental pollutants such as polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans. The term molar incisor hypomineralization (MIH) was introduced to describe the clinical appearance of enamel hypomineralization of systemic origin affecting one or more permanent first molars (PFMs) that are associated frequently with affected incisors.

Aim. The aim of this study was to determine the prevalance of MIH in children from the most industrialized and polluted region and the most green-energy island of Turkey.

Design. In September 2007, a retrospective study was initiated in two elementary schools: one, a

group of children (N = 153) who fitted the criteria from Tavsancil, Kocaeli (N = 109) and the other from Bozcaada island, Canakkale (N = 44). The soil samples were collected from selected regions in order to determine the contamination levels in a heavily industrialized area and a non-industrialized area.

Results. Prevalance of MIH in children in Bozcaada island was 9.1%, while prevalance of MIH was 9.2% in Tavsancil. The PCDD/F levels in soil samples collected from Bozcaada and Tavsancil were determined as 1,12 and 8,4 I-TEQ ng/kg dry soil, respectively (P < 0.001).

Conclusions. In this preliminary study with a small study population, prevalence of MIH did not seem to be associated with the levels of PCDD/Fs in the environment.

Introduction

Environmental toxins such as chemicals have adverse health effects on the human body. Polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are among the most toxic chemicals in the environment. These compounds originate from chemical manufacturing, especially of herbicides and chlorinated phenols; combustion processes, especially incinerators; metallurgical processes; and paper and pulp processing¹. Generation and release of PCDD/Fs have created great public concern because of their acute and chronic health effects on immune, nervous, endocrine, and reproductive systems, and potential carcinogenic effects^{2–4}.

Kocaeli, the most industrialized bay region of Turkey, the next state to Istanbul, has undergone dense industrialization followed by a rapid increase in population and an uncontrolled urbanization since the 1960s. These led to serious environmental pollution in air, water, and soil in the vicinity of the city. Because of the absence of environmental measures, these problems have increased gradually until the 1990s, while positive measures are currently established within EU proceedings. Kocaeli, with a population density of 344 people/km², has many sources of potential environmental pollutants such as two very busy traffic arteries, three major tyre factories, a car factory, a medical waste incinerator, a pulp and paper

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mill, several petrochemical plants, and the largest petroleum refinery of Turkey. A current study showed that the levels of PCDD/F pollution in Kocaeli are comparable to the levels observed in various urban areas in Europe. PCDD/F concentrations in surface soils ranged between 0.4 pg I-TEQ/kg and 4.27 pg I-TEQ/kg, while those in ambient air were between 23 fg/m^3 and 563 fg/m^3 . The rates were higher than the recommended deposition rates in relation to the tolerable daily intake range of PCDD/Fs for humans⁵. In 2004, the estimated PCDD/F doses that will be experienced by the receptor groups and subgroups as a result of the PCDD/F emissions from the incinerator in Kocaeli, were found to be $3.01 \times 10^5 - 0.081$ pg TEQ/kg bw/day for infants and 2.31×10^6 and 0.008 pg TEQ/kg bw/day for the adults in the region⁶.

On the other hand, Bozcaada is a small touristic island (next to Troy) at the most Western point of Turkey, in the Aegean sea. While organic agriculture, fishing, and vineyards are popular on the island, 100% of the energy supply is based on wind farms heading for a pollutant-free region (recently energy was supplied from Çanakkale on the mainland). The biggest available wind energy power plant in Turkey is at present the Bozcaada Wind Energy Plant (BORES) which was constructed in 2000⁷. There are no visible sources producing PCDD/ Fs such as arteries, factories, and plants on the island (Fig. 1).



Fig. 1. Regional map. 1, Bozcaada, Çanakkale; 2, Tavsancil, Kocaeli; 3, Istanbul.

Children's developing teeth may be sensitive to environmental pollutants such as PCDD/Fs. The term molar incisor hypomineralization (MIH) was introduced in 2001 to describe the clinical appearance of enamel hypomineralization of systemic origin affecting one or more permanent first molars (PFMs) that are associated frequently with affected incisors⁸. It has also been referred to as 'hypomineralized' PFMs⁹, 'idiopathic enamel hypomineralization'^{10,11}, 'dysmineralized' PFMs¹², 'nonfluoride hypomineralization^{'13,14}, and 'cheese molars'^{15,16}. The condition is attributed to disrupted ameloblastic function during the transitional and maturational stages of amelogenesis^{10,17}. By the age of 9, children affected with MIH had been obligated to undergo dental treatment of their PFM's nealy ten times as often as the healthy controls¹⁷. Recently, Kotsanos et al.¹⁸ stated that children exhibiting MIH have 11 times greater probability of undergoing restorative treatment in their PFM's compared with children of a control group. Moreover, fillings and sealants in MIH-affected children have over three times a greater probability of needing re-treatment than interventions on children of the control group. The present situation shows how important it is to prevent children from MIH, while attempts to understand its aetiology become vital. In the literature, limited factors associated with MIH include systemic conditions and environmental insults influencing natal and early development^{15,19}.

The high prevalence of hypomineralized dental defects among normal children may be a sign of exposure to PCDD/Fs. Because the dental hard tissues do not undergo remodelling, defects that occurred in infancy can be diagnosed even after many years. In the late 1970s, an increasing number of children showing extensive and severe idiopathic hypomineralization of the enamel of incisors and PFMs was reported in Sweden. Regarding this situation, an epidemiologic study was initiated to analyse the prevalence, extent, and severity of enamel hypomineralization in Swedish children born in 1966–1974. It was found that 15.4% of the children born in 1970 showed such changes, while corresponding figures for children born in 1966, 1969, 1971, 1972, and 1974 were 6.3, 7.3, 7.1, 5.2, and 4.4, respectively¹¹. In Finland,

it was recently shown that in a normal child population, PCDDs and PCDFs in mother's milk may cause mineralization defects in the child's PFM teeth undergoing mineralization during the first 2 years of life^{20,21}. In a Slovenian epidemiologic study on children of 8– 15 years old, pre- and postnatally exposed to polychlorinated biphenyls, mainly PCBs, Jan and Vrbic²² found that significantly more developmental defects of enamel were found in a contaminated region than in the control area.

The aim of this study was to determine the prevalance of MIH in two groups of children from the most industrialized and polluted region, and the most green-energy-producing island of Turkey.

We hypothesized that environmental factors may cause MIH; furthermore, we expected to see an increased prevalance of MIH in a highly industrialized urban population compared to a windfarm-green energy island.

Methods

In September 2007, a retrospective study was initiated in two elementary schools (selected on the basis of the application to the National Ministry of Education), on a group of children from Tavsancil, Kocaeli, and Bozcaada island; Canakkale (there is only one elementary school, including all schoolchildren on the island) in Turkey. The following procedures were performed at the schools selected. Schoolchildren were included with respect to the following criteria: (i) 7- to 10-year-old children; (ii) no systemic disease history; (iii) life-long residents in the selected regions (children born and living in the study area); (iv) children's mothers and fathers should have permanently lived in the respective area for at least 5 years before the child's birth, while breastfeeding was also practiced in the same region; and (v) four PFMs and eight permanent incisors erupted.

From the schoolchildren examined, 153 children (109 from Tavsancil, and 44 from Bozcaada) met the criteria. All parents gave informed consent. Dental examinations were carried out in September – November 2007 by two calibrated paediatric dentists (OOK and EC), using a standard mouth mirror and dental

probe. The location of demarcated opacities and enamel breakdown was recorded on a specially designed patient research data sheet. All the participating children received toothbrushes from the paediatric dentists, and were instructed to brush their teeth in their classroom. After thorough brushing, the four PFMs and eight erupted permanent incisors were examined wet for demarcated opacities, PEB, and atypical restorations under portable light source²³. Criteria for the diagnosis of demarcated opacities, post-eruption breakdown, atypical restorations, and extracted PFMs caused by MIH were developed by Weerheijm et al.²³. Demarcated opacities were defined as defects of altered enamel translucency; the defective enamel is white-cream or yellowbrown in colour, of normal thickness with a smooth surface, and has a distinct boundary adjacent to normal enamel^{9,24}. Dentitions with generalized opacities present on all teeth (such as in several forms of amelogenesis imperfecta), rather than limited to the PFMs and permanent incisors, are not considered to have MIH²³. Details of medical history on various confounding factors (such as birth details; place of residence during tooth development; mothers' detailed data; duration of breastfeeding; respiratory, ear, or other infections; hospitalization: fever > 39 °C, medications, fluoride, metal exposure) of MIH patients had been recorded after personal interviews with patients' mothers. Where mothers had been unsure, the patients' health data booklet with all medical information recorded by a paediatrician was inspected.

Detection of PCDD/Fs

Because of their lipophilic nature, PCDD/Fs accumulate in matrices rich in organic matter, such as soil, aquatic sediment, and biota. Soil is one of the main reservoirs for long-term PCDD/F deposition. PCDD/F compounds released into the atmosphere from different emission sources reach the soil by dry/wet deposition processes. As a result of cumulative deposition, concentrations of PCDD/Fs in soil increase, and then enter the food chain through which the major part of the human exposure occurs. In addition, PCDD/Fs are highly stable (persistent) compounds in soil with half lives between 10 and 100 years. Therefore, PCDD/F levels in soil are a good indicator of long-term PCDD/F exposure in an area. Moreover, determination of PCDD/F levels in soil is a widely accepted method of identifying background PCDD/F pollution. The soil samples were taken from two different locations: Tavsancil and Bozcaada. In each location, five different samples were taken from undisturbed areas to indicate a long-term deposition of PCDD/Fs. The samples were mixed and homogenized prior to analyses. The sampling points were especially selected from agriculturally and socially dense areas for each region.

Because the half-lives of PCDD/Fs are relatively long and their solubility in water is very low, the concentrations of these compounds in soil change very slowly. Because of durability and low mobility of PCDD/Fs, changes occur generally during soil cultivation. Previous studies show that the majority of PCDD/Fs in soils have been found in upper layers of soil (e.g. $0-10 \text{ cm}^{25}$. In this study, 500 g of soil samples was taken from depths of 5 cm from the residential area. The samples were then sieved through a 2-mm-mesh screen to obtain a homogenous distribution; the water content was determined and then stored at -20 °C until analyses. About 40 g of soil sample was used for PCDD/F analyses. The EPA Method 8280 was applied for extraction and cleaning of samples²⁶.

All PCDD/F analyses were made by a highresolution GC/low resolution MS (HP 6890 GC and HP 5973 N MS) with DB-dioxin column ($60 \text{ m} \times 0.25 \text{ mm} \times 0.25 \text{ Lm}$) in Kocaeli University Gas Chromotography Laboratory, except for 2,3,7,8 TCDD congener.

The temperature regime for GC was as follows: At the beginning, the initial temperature (180 °C) of the capillary column was held for 2 min, then increased to 220 °C at a rate of 5 °C per minute and held for 2 min. Then, the temperature was increased to 270 °C at a rate of 2, 5 °C per minute, and held for 40 min. The injection volume was 2 μ L by splitless injection. The MS interface, quadropole, and ion source temperatures were 280 °C, 106 °C, and 150 °C, respectively. The MS was operated under negative chemical ionization (70 eV electron energy) conditions with methane as the reagent gas and helium as the carrier gas. 2,3,7,8-TCDD was analysed by GC/MS (HP 6890 GC and HP 5973 N MS) operated in EI mode with DB-5 MS column (60 m × 0.25 mm × 0.25 Lm). Toxic equivalents (TEQs) of the congeners were calculated using the International Toxicity Equivalency (I-TEQ) factors²⁷. Congener concentrations measured below the detection limit were assumed as half of the corresponding detection limit in the I-TEQ calculations. The level of fluoride in drinking water is low in both regions (in Bozcaada, it is 0.080 mg/L and in Tavsancil is 0.070 mg/L)²⁸.

Statistical method

The data were processed with the SPSS software (version 14.5, Chicago, IL, USA). Fisher's exact test, chi-square, and Mann–Whitney U-tests were applied. A P value < 0.05 was considered as statistically significant.

Results

In total 153 schoolchildren, 44 from Bozcaada island and 109 from Tavsancil were examined. The inter-examiner reliability kappa statistics of the two examiners was 0.90. Prevalance of MIH in children in Bozcaada island was 9.1%, while prevalance of MIH in Tavsancil was 9.2%.

Of the four children affected with MIH in Bozcaada island, all had only demarcated opacities, but no breakdown or restorations. Two had lesions in both incisors and molars, and two children had a single molar and incisor affected. In Tavsancil, of the ten children affected with MIH, seven (75%) had only demarcated opacities, while three had breakdown. Distribution of the subjects with different affected teeth is presented in Table 1.

Tooth 26 was the most affected PFM, and tooth 46 the least. There was no significant difference between affected PFMs and incisors in both regions. Tooth 41 was the most affected incisor, and tooth 42 and 32 the least affected incisors by MIH. Regarding gender, there were no statistical differences between MIH (6 F, 8 M) and non-MIH (72 F, 67 M) groups.

	Bozcaada (n = 44)			Tavsancil (<i>n</i> = 109)		
Affected	Girls	Boys n	Total n	Girls	Boys n	Total n
Single molar	0	0	0	2	1	3
Single molar + incisors	0	2	2	0	2	2
Only two to four molars	0	0	0	2	0	2
Molars + incisors	1	1	2	1	2	3
Total children with MIH	1	3	4	5	5	10

Table 1. Prevalance of molar incisor hypomineralization (MIH) in children.

Fisher's exact test, chi-square NS, P > 0.05.

	Bozcaada		Tavsancil	
Infections in first 3 years of life	MIH n (%)	Non-MIH n (%)	MIH n (%)	Non-MIH n (%)
Upper airway infections				
Respiratory				
Bronchitis		7 (17.5)		6 (6.1)
Otitis media		1 (2.5)		1 (1.0)
Urinary infections				1 (1.0)
Gastrointestinal infections			1 (10)	2 (2.0)
Viral infections	1 (25.0)	3 (7.5)	2 (20)	7 (7.1)
Fever > 39 °C		2 (5.0)		10 (10.1)
Allergy, asthma		1 (2.5)	1 (10.0)	2 (2.0)
Dental infections				4 (4.0)
Anaemia				2 (2.0)
Dermatologic disease				2 (2.0)
No disease history	3 (75.0)	27 (67.5)	6 (60.0)	66 (66.6)

Table 2. Distribution of infant disease-
related factors.

Fisher's exact test, chi-square NS, P > 0.05.

Regarding diseases in the first 3 years of life, on Bozcaada island, 25% of MIH and 37.5% of non-MIH children had a disease history. In Tavsancil, 40% of MIH and 33.4% of non-MIH children had a disease history, and there was no significant relation between disease history and MIH in either region. Regarding systemic diseases reported in MIH and non-MIH children, no statistical differences were found (Table 2).

Regarding preterm birth data, only three children (non-MIH) were stated to be born preterm with very low birth weight (under 1500 mg). None of the MIH children were born preterm.

With regard to breastfeeding duration, there were no significant differences between MIH and non-MIH children in both regions.

The PCDD/F concentrations of soil samples are presented in Table 3. The PCDD/F levels of soil samples collected from Bozcaada and Tavsancil were determined as 1, 12 and 8,4 I-TEQ ng/kg dry soil, respectively. (P < 0.001) These results are comparable with the reference values of PCDD/F concentrations in the soils for agricultural and horticultural land uses and soil reclamation. According to the limit values proposed by the Federal Health Office of Germany²⁹, the PCDD/F level of the Bozcaada sample fall below the limit value of 5 pg I-TEQ/g dry soil. On the other hand, the PCDD/ F level in the Tavsancil sample is within the range of 5-40 pg I-TEQ/g dry soil used for the cultivation of certain vegetables.

As Table 3 shows, OCDD and other highly chlorinated congeners $(P_{6,7})$ are the major contributors to the total PCDD/Fs. The relative

Table 3. The polychlorinated dibenzo-*p*-dioxin (PCDD)/F concentrations in soil samples.

Congener	Bozcaada (ng/kg dry soil)	Tavsancil (ng/kg dry soil)
2,3,7,8 TCDD	0.08 ^a	0.10 ^a
1,2,3,7,8 PCDD	0.03ª	0.73
1,2,3,4,7,8 HxCDD	0.03ª	0.04ª
1,2,3,6,7,8 HxCDD	0.01ª	0.51
1,2,3,7,8,9 HxCDD	0.06ª	0.08 ^a
1,2,3,4,6,7,8 HpCDD	0.06ª	6.76
OCDD	1.57	38.60
2,3,7,8 TCDF	0.03ª	1.17
1,2,3,7,8 PeCDF	0.16	0.97
2,3,4,7,8 PeCDF	1.41	9.90
1,2,3,4,7,8 HxCDF	0.20	3.10
1,2,3,6,7,8 HxCDF	0.72	7.30
2,3,4,6,7,8 HxCDF	1.90	11.00
1,2,3,7,8,9 HxCDF	0.02 ^a	0.35
1,2,3,4,6,7,8 HpCDF	0.02ª	46.20
1,2,3,4,7,8,9 HpCDF	0.53	0.83
OCDF	0.04	3.56
Sum of 17 congeners	6.87	131.2
Sum of PCDD congeners	1.84	46.82
Sum of PCDF congeners	5.03	84.38
PCDF/PCDD	2.7	1.8
I-TEQ	1.12	8.4

*The concentration of congeners measured below the detection limits was accepted as half of the detection limit. Mann–Whitney U-test P < 0.001.

contribution of OCDD to total PCDD/F concentration is calculated to be about 22% and 29% for Bozcaada and Tavsancil samples, respectively. On the other hand, the highest congener concentration was observed for 2,3,4,6,7,8 HxCDF for Bozcaada and 1,2,3,4,6,7,8 HpCDF for Tavsancil samples, with the contributions of 27% and 35% to total PCDD/F concentration, respectively. The congener distributions reflect the wide variety of the industrial facilities in Tavsancil.

The most toxic dioxin congener, 2,3,7,8tetrachlorodibenzo-*p*-dioxin (TCDD) was below the detection limit in this study; therefore, the concentration of this congener was assumed as half of the detection limit. According to this assumption, concentrations of 2,3,7,8 TCDD in soils are 0087 ng/kg dry soil and 0105 ng/kg dry soil for Bozcaada and Tavsancil samples, respectively. On the other hand, the contribution of this congener to the total PCDD/F concentration is lower than that of highly chlorinated congeners.

Lower chlorinated PCDD/Fs are generally found in vapor phase, and they can fall into

the transformation reactions (photolytic degradation, gas phase reactions, etc.). Therefore, the relative concentrations of highly chlorinated congeners on particles may increase. Low concentration of less chlorinated congeners ($P_{4,5}CDD/Fs$) indicates the existence of longtime depositions⁵. Hence, the results of this study show presence of a long-time deposition in both samples.

According to the total concentrations of PCDD/Fs in dry soils, the sum of PCDF concentrations is higher than PCDDs for both samples. Ratios of PCDF/PCDD are equal to 2,7 and 1,8 for Bozcaada and Tavsancil samples, respectively.

In this study, while environmental conditions significantly differed in the two regions, no relationship was detected with MIH prevalence.

Discussion

Every year, 370 000 people in Europe die prematurely from diseases linked to environmental pollution³⁰. Since 2001, all European Union member and accession (Croatia and Turkey) countries act on persistent organic pollutants which require measures to reduce or eliminate, inter alia, release of dioxins and PCBs³¹.

PCDD/Fs are among the most toxic chemicals in the environment⁵. Additionally, the contribution of other chemicals with dioxinlike toxicity (dioxin-like PCBs and polybrominated dibenzo - dioxins and furans - PBDD/Fs) to the total PCDD/F toxicity should be noted. It has been stated that it is highly possible that these chemicals play a significant role in total PCDD/Fs intake⁶. Finally, the presence of many other pollutants (PCBs, PAHs, heavy metals, etc.) in the environment at significant levels is highly probable in Kocaeli, the most industrialized area of Turkey. At present, no environmental adverse report has been detected from Bozcaada island. The island may seem to be isolated but with respect to dioxins no region in the world can be assumed to be unaffected. Dioxins are persistent and semivolatile compounds, so they can be transported in the air over long distances. Long-range atmospheric transport and subsequent atmospheric deposition have been recognized as the most important pathways of chemicals to migrate from their places of origin to remote areas, although other transport pathways have been discovered in recent years, for example, migration via seabirds and ocean currents³². Even in remote areas like the arctic or antarctic, dioxins can be found in air, biota, and soil. Bozcaada Meteorological Institute states that predominant wind directions in this area are north and north-east heading for Istanbul, while the main air masses reaching Bozcada island are mainly from the Balkan peninsula since 1992 (40–45%)³³.

Teeth develop as a result of a series of inductive, sequential, and reciprocal interactions between the ectoderm and the subjacent mesenchyme³⁴. Tooth development is genetically regulated but sensitive to environmental disturbances. Aberrations in the function of tooth-forming cells lead to permanent morphologic consequences. Regarding environmental pollution, PCDD/Fs may be another target for MIH.

Living and dietary conditions in Europe may be a factor for MIH. Recently, a North African study showed that MIH was rare (2.9%) in Libya³⁵. This prevalence, however, was clearly higher in comparable studies performed in North Europe: Finland (19.3%, and 17%)^{14,36}; Germany (6%)³⁷; Sweden (4%–15%)¹¹, (18%)³⁸; Lithuania (9.7%)³⁹; the Netherlands (10%)¹⁶; and South Europe: Italy (13.7%)⁴⁰, Turkey (14.9%)⁴¹. In this study, prevalence of MIH in children in Bozcaada island and Tavsancil were also both over 9%, representing similiar data.

The present concern over the increasing level of tropospheric sulphur dioxide and other gases in urban areas is essentially because of their role in causing detrimental effects of air pollutants on human health and aquaticterrestrial ecosystems. Regarding other toxins such as chemicals, associations have been made between the presence of polyhalogenated aromatic hydrocarbons, mainly PCDDs in breast milk, and enamel hypomineralization in both clinical and laboratory studies^{20,21}.

The dietary habits of the children living on the island may be different from those living in the urban region. Consumption rates of fruits and vegetables, and the locally grown fraction of these food groups have a significant role in total PCDD/F exposure. The relative

importance of exposure pathways was evaluated in terms of their contribution to the overall PCDD/F risk was recently shown for the Kocaeli urban region which includes Tavsancil⁶. The most significant exposure pathway for the receptor groups in the age groups older than 6 years because of the dominance of these products in the diet has been calculated. In the urban settings, it is constituted mainly by the consumption of fruits (24%). fruiting vegetables (12%), and leafy vegetables (9%). The contribution of the ingestion of poultry products is estimated to be between 2% and 4% for the receptors in the age groups older than 6 years, and more than 90% of this pathway results from the ingestion of eggs. The percentage of the meat and meat products does not exceed 6%. The contribution of the ingestion of a plant product pathway increases as the age is increased, in opposition to the ingestion of a milk pathway. Ingestion of milk was estimated to be responsible for over 90% of total intake for infants, while its contribution decreases to 12%, for the adult receptors in the urban area. For the non-school children (ages 1–6), consumption of milk, other dairy, and vegetable products has higher contribution percentages as compared to the consumption of animal products. For infants, about 90% of the PCDD/F intake results from the ingestion of milk. People living in Tavsancil have lifestyles and dietary habits similar to those who live in a rural region. Therefore, the living and dietary habits of people in both Taysancıl and Bozcaada island are by and large the same. Furthermore, the daily diet in both areas is heavily dependent on locally grown food. On the other hand, the determination of PCDD/F intakes, related exposure pathways, and risk assessment are complex processes that depend on many parameters. Although soil contamination plays a central role in the exposure assessment, a detailed exposure study would be useful for drawing better conclusions on the MIH prevalence and PCD/F exposure relationship. Persistence and accumulation of PCDDs in tissue lipids and in the food chain may result in chronic low-level exposure in humans⁴². Karademir *et al.*⁴³ stated that PCDD/ F intakes for rural receptors in Kocaeli were estimated to be three times higher than those

for urban and semi-urban Kocaeli receptors due mainly to the consumption of locally grown food. In this study, because the PCDD/ F level in Tavsancil soil is seven times higher than that of Bozcaada soil, the difference could be attributed to the effect of long industrialization and urbanization periods on the environmental pollution in Kocaeli.

The effects of long-term exposure to PCBs on developmental dental defects of primary and permanent teeth in children in eastern Slovakia demonstrated a dose-response relationship between PCB exposure and developmental enamel defects of permanent teeth in children where the prevalences of demarcated opacities and hypoplasias were especially increased⁴⁴. On the other hand, children living by the heavily polluted Kymijoki River in Southern Finland had no increase in the prevalence of developmental dental defects, nor were human milk PCDD/F concentrations higher than the concentrations in samples from two other areas in Finland¹³. Regarding other potential hazards, 25 years after the dioxin accident in Seveso, Italy, subjects from the contaminated areas were found to show enamel defects and developmental dental aberrations that were associated with childhood exposure to toxicity of 2,3,7,8-TCDD⁴⁵. In this study, children aged 7-10 years are selected. Therefore, at present, it is not possible to determine the PCDD/F intake caused by breast milk feeding. PCDD/F level in the soil was considered as an indicator of long-term PCDD/F pollution. Also, we did not receive ethical clearance from Yeditepe University Ethics Committee to examine MIH detected children's serum PCDD levels. None of the children and their mothers had ever been exposed to a main toxic hazard, any dioxin, or environmental accident.

The prevalence of MIH in two groups of children from the most industrialized and polluted region, and the most green-energy producing island of Turkey were nearly the same whereas PCDD levels were significantly different. Our hypothesis that environmental factors cause MIH was not supported. Furthermore, we expected to see an increased prevalence of MIH in a highly industrialized urban area and a decreased prevalence on a windfarm-green energy island. MIH prevalence was, however, found to be the same in both regions.

What this paper adds

• Although MIH is a multifactorial disturbance, there are some studies finding a relationship between environmental pollutants and MIH. However, in this preliminary study with a small study population, the prevalence of MIH did not seem to be associated with the levels of PCDD/Fs in the environment.

Why this paper is important to paediatric dentists

• Paediatric dentists should be aware of environmental conditions and their effects on dental and oral tissues, whereas more research should be carried out on MIH and environmental conditions.

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References

- 1 Anonymous. European Commission DG Environment Releases of Dioxins and Furans to Land and Water in Europe. Brussels: EU Commission, 1999.
- 2 Alcock R, Gemmill R, Jones KC. Improvements to the UK PCDD/PCDF and PCB atmospheric emission inventory following an emission measurement programme. *Chemosphere* 1999; **38**: 759–770.
- 3 WHO. *PCDD/Fs and Their Effects on Human Health.* 1999. Fact Sheet No. 225.
- 4 USEPA. Screening level ecological risk assessment protocol for hazardous waste combustion facilities. EPA 530-D-99–001B, 1999.
- 5 Bakoglu M, Karademir A, Durmusoglu E. Evaluation of PCDD/F levels in ambient air and soils and estimation of deposition rates in Kocaeli, Turkey. *Chemosphere* 2005; **59**: 1373–1385.
- 6 Karademir A. Health risk assessment of PCDD/F emissions from a hazardous and medical waste incinerator in Turkey. *Environ Int* 2004; **30**: 1027–1038.
- 7 World Energy Council Turkish National Committee (WECTNC). Turkey Energy Report, Ankara, Turkey, 2000.
- 8 Weerheijm KL, Jälevik B, Alaluusua S. Molar–incisor hypomineralization. *Caries Res* 2001; **35**: 390–391.

- 9 Jälevik B, Noren JG. Enamel hypomineralization of permanent first molars: a morphological study and survey of possible aetiological factors. *Int J Paediatr Dent* 2000; **10**: 278–289.
- 10 Fearne J, Anderson P, Davis GR. 3D X-ray microscopic study of the extent of variations in enamel density in first permanent molars with idiopathic enamel hypomineralization. *Br Dent J* 2004; **196**: 634–638.
- 11 Koch G, Hallonsten AL, Ludvigsson N, Hansson BO, Holst A, Ullbro C. Epidemiologic study of idiopathic enamel hypomineralization in permanent teeth of Swedish children. *Community Dent Oral Epidemiol* 1987; **15**: 279–285.
- 12 Croll TP. Creating the appearance of white enamel dysmineralization with bonded resins. *J Esthet Dent* 1991; **3**: 30–33.
- 13 Hölttä P, Kiviranta H, Leppäniemi A, Vartiainen T, Lukinmaa PL, Alaluusua S. Developmental dental defects in children who reside by a river polluted by dioxins and furans. *Arch Environ Health* 2001; **56**: 522–528.
- 14 Leppäniemi A, Lukinmaa PL, Alaluusua S. Nonfluoride hypomineralizations in the permanent first molars and their impact on the treatment need. *Caries Res* 2001; **35**: 36–40.
- 15 van Amerongen WE, Kreulen CM. Cheese molars: a pilot study of the etiology of hypocalcifications in first permanent molars. *J Dent Child* 1995; **62**: 266–269.
- 16 Weerheijm KL, Groen HJ, Beentjes VE, Poorterman JH. Prevalence of cheese molars in 11-year-old Dutch children. J Dent Child 2001; 68: 259–262.
- 17 Jälevik B, Klingberg GA. Dental treatment, dental fear and behaviour management problems in children with severe enamel hypomineralization of their permanent first molars. *Int J Paediatr Dent* 2002; **12**: 24–32.
- 18 Kotsanos N, Kaklamanos EG, Arapostathis K. Treatment management of first permanent molars in children with molar-incisor hypomineralisation. *Eur J Paediatr Dent* 2005; 6: 179–184.
- 19 Jälevik B, Noren JG, Barregård L. Etiologic factors influencing the prevalence of demarcated opacities in permanent first molars in a group of Swedish children. *Eur J Oral Sci* 2001; **109**: 230–234.
- 20 Alaluusua S, Lukinmaa PL, Torppa J, Tuomisto J, Vartiainen T. Developing teeth as biomarker of dioxin exposure. *Lancet* 1999; **353**: 206.
- 21 Alaluusua S, Lukinmaa PL, Koskimies M, *et al*. Developmental dental defects associated with long breastfeeding. *Eur J Oral Sci* 1996; **104**: 493–497.
- 22 Jan J, Vrbic V. Polychlorinated biphenyls cause developmental enamel defects in children. *Caries Res* 2000; 34: 469–473.
- 23 Weerheijm KL, Duggal M, Mejare I, *et al.* Judgement criteria for molar incisor hypomineralization (MIH) in epidemiologic studies: a summary of the European meeting on MIH held in Athens, 2003. *Eur J Paediatr Dent* 2003; **4**: 110–113.
- 24 Commission on Oral Health Research & Epidemiology. A review of the Developmental Defects of Enamel Index (DDE Index). Commission on Oral Health,

Research & Epidemiology. Report of an FDI Working Group. *Int Dent J* 1992; **42**: 411–426.

- 25 Roots O, Henkelmann B, Schramm KW. Concentrations of polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans in soil in the vicinity of a landfill. *Chemosphere* 2004; **57**: 337–342.
- 26 U.S. EPA. The analysis of polychlorinated dibenzo*p*-dioxins and polychlorinated dibenzofurans by high resolution gas chromatography/low resolution mass spectrometry (HRGC/LRMS). US EPA, Method No: 8280a. 1996.
- 27 NATO/CCMS (North Atlantic Treaty Organization/ Committee on Challenges of Modern Society). Scientific Basis for the Development of International Toxicity Equivalency Factor (I-TEF) Method of Risk Assessment for Complex Mixtures of Dioxins and Related Compounds. Report No. 187, 1988.
- 28 Turkish Ministry of Health. *Water Fluoride Map of Turkey*. Ankara: Ministry Press, 2003.
- 29 Eljarrat E, Caixach J, Rivera J. Levels of polychlorinated dibenzo-*p*-dioxins and dibenzo-*p*-furans in soil samples from Spain. *Chemosphere* 2001; **44**: 1383– 1387.
- 30 Commision of the European Communities. 2006 Environment Policy Review SEC 487. Brussels: EU Commission, 2007.
- 31 European Commission. Dioxins & PCBs: Environmental Levels and Human Exposure in Candidate Countries Reference: ENV.C.2/SER/2002/0085. Brussels: EU Commission, 2004.
- 32 Cheng H, Zhang G, Jiang JX, *et al.* Organochlorine pesticides, polybrominated biphenyl ethers and lead isotopes during the spring time at the Waliguan Baseline Observatory, northwest China: implication for long-range atmospheric transport. *Atmos Environ* 2007; **41**: 4734–4747.
- 33 Karanfil V. *Annual Reports of Bozcaada Meteorological Institute. 1992–2007.* Çanakkale, Turkey: Bozcaada meteorological institute, 2007.
- 34 Thesleff I, Vaahtokari A, Partanen A-M. Regulation of organogenesis: common molecular mechanisms regulating the development of teeth and other organs. *Int J Dev Biol* 1995; **39**: 35–50.
- 35 Fteita D, Ali A, Alaluusua S. Molar–incisor hypomineralization (MIH) in a group of school-aged children in Benghazi, Libya. *Eur Arch Paediatr Dent* 2006; **7**: 92–95.
- 36 Alaluusua S, Lukinmaa PL, Vartiainen T, Partanen M, Torppa J, Tuomisto J. Polychlorinated dibenzo-*p*dioxins and dibenzofurans via mother's milk may cause developmental defects in the child's teeth. *Environ Toxicol Pharmacol* 1996; 1: 193–197.
- 37 Dietrich G, Sperling S, Hetzer G. Molar incisor hypomineralization in a group of children and adolescents living in Dresden (Germany). *Eur J Paediatr Dent* 2003; **4**: 133–137.
- 38 Jälevik B, Klingberg G, Barregard L, Noren JG. The prevalence of demarcated opacities in permanent first molars in a group of Swedish children. *Acta Odontol Scand* 2001; **59**: 255–260.

- 39 Jasulaityte L, Veerkamp JS, Weerheijm KL. Molar incisor hypomineralization: review and prevalence data from the study of primary school children in Kaunas/Lithuania. *Eur Arch Paediatr Dent* 2007; **8**: 87– 94.
- 40 Calderara PC, Gerthoux PM, Mocarelli P, Lukinmaa PL, Tramacere PL, Alaluusua S. The prevalence of molar incisor hypomineralisation (MIH) in a group of Italian school children. *Eur J Paediatr Dent* 2005; 6: 79–83.
- 41 Kuscu OO, Caglar E, Sandalli N. The prevalence and aethiology of molar–incisor hypomineralization (MIH) in a group of children. Istanbul. *Eur J Ped Dent* 2008;
 9: 139–144.
- 42 Gao Y, Sahlberg C, Kiukkonen A, et al. Lactational

exposure of Han/Wistar rats to 2,3,7,8-tetrachlorobenzo-*p*-dioxin interferes with enamel maturation and retards dentin mineralization. *J Dent Res* 2004; **83**: 139–144.

- 43 Karademir A, Durmusoglu E, Bakoglu M. Health risk assessment of background PCDD/F exposure levels in Kocaeli, Turkey. *J Environ Sci Health A Tox Hazard Subst Environ Eng* 2007; **42**: 729–739.
- 44 Jan J, Sovcikova E, Kocan A, Wsolova L, Trnovec T. Developmental dental defects in children exposed to PCBs in eastern Slovakia. *Chemosphere* 2007; **67**: S350–S354.
- 45 Alaluusua S, Calderara P, Gerthoux PM, et al. Developmental dental aberrations after the dioxin accident in Seveso. Environ Health Perspect 2004; 112: 1313–1318.

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