Prospective longitudinal study of early childhood caries and developmental defects of enamel on permanent successors in children in the Czech Republic

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Abstract

Aim This study investigates the influence of early childhood caries (ECC) on the occurence of developmental defects of enamel (DDE) on permanent teeth.

Methods Study design: This prospective original study was conducted to monitor the relationship between ECC in primary dentition and DDE as their consequences on permanent successors. One hundred and forty children were recruited and included in the study group with diagnosed ECC (ECCG) (N=60) or the control group (CG) (N=80). Deciduous upper central incisors were assessed for existing dental caries according to the dmft index and the modified pufa score and permanent incisors were examined using the modified DDE index.

Results Prevalence of DDE was 19.3% in the whole sample; in ECCG it was 24.2% and 12.6% in CG. The most common defect was diffuse opacity with a prevalence of 44.8% in ECCG and 72% in CG. Prevalence of hypoplasia was 13.8% in ECCG, in CG it was present as a combination defect with a prevalence of 4%. The extent of less than 1/3 of labial aspect was in 69% of defects in ECCG, in CG it was 52%. The location of defects in the incisal third was 48.3% in ECCG and 32% in controls.

Conclusion A statistically significant relationship between ECC and DDE was not observed. The results were compared with studies about the relationship between ECC and DDE.

KEYWORDS Developmental enamel defects (DDE); Early childhood caries (ECC); Permanent teeth.

Introduction

Developmental enamel defects (DDE) are visible deviations from the normal translucent appearance of the enamel. They can be classified into three types (the modified DDE index) according to their macroscopic appearance in hypoplasia, demarcated and diffuse opacity [Clarkson and O'Mullane, 1989; Rai et al., 2018]. The prevalence of DDE ranges from 21% to almost 100% in the permanent dentition [Vargas-Ferreira and Ardenghi, 2011] and 3.9–81.5% in the deciduous dentition [Costa et al., 2017; Basha et al., 2014]. There are many factors that can lead to changes in the normal appearance of the enamel. A special group is presented by defects caused by excessive fluoride intake. Defects of nonfluoride origin caused by systemic factors include genetic anomalies, congenital malformations, hereditary metabolic disorders, infectious diseases, neurological disorders, endocrinopathies, nephro- and enteropathy, liver diseases, neonatal disorders, nutritional deficiencies and intoxications [Pindborg, 1982]. Among local aetiologic factors, the permanent tooth germ damage by a temporary tooth injury or presence of periapical infection represent the most important ones. Experimental studies in animals supported by human authopsy reports have shown an association of artificially induced inflammation of the periapical tissue in primary dentition with defects of permanent tooth enamel formation [Broadbent et al., 2005].

Due to the high prevalence of caries in the paediatric population (e.g. 36–85% in Asia, 44% in India, 8.2% in Italy, 60–65% of children in the Czech Republic), higher incidence of DDE on permanent teeth [Vandana et al., 2018; Broukal et al., 2009; Colombo et al., 2019; Ivančáková et al., 2007; Alazmah, 2017; Meriç et al., 2020] can be expected. This means an aesthetic handicap with an impact on the quality of life of children related to their oral health [Hazar at al., 2018; Vargas-Ferreira and Ardenghi, 2011], as well as possible DDE-associated complications [Vargas-Ferreira et al., 2015], such as eruption failure, calcification in the pulpal chamber or more frequent development of apical periodontitis in these teeth [Hazar et al., 2018; Figueiredo et al., 2011; Lo et al., 2003]. The link between early childhood caries (ECC) in deciduous dentition and higher occurence of DDE in permanent dentition as its consequence was firstly demonstrated by Lo et al. [2003] in a study of Chinese children from a nonfluoridated area. There are only two aepidemiological studies dealing with ECC and DDE as its consequences [Broadbent et al., 2005; Lo et al., 2003]. Despite several animal studies [Purdell-Lewis and Suckling,

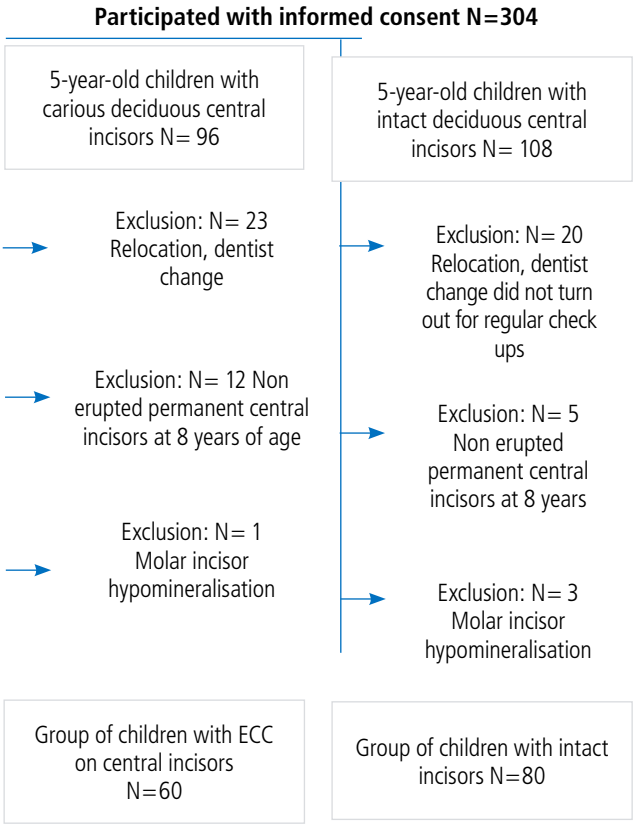

FIG. 1 Flow chart diagram of patients recruitment.

1987] there is only limited aepidemiological evidence of relationship between ECC and higher occurence of DDE. There is a lack of clinical information about caries in primary dentition and enamel defects on their successors in other populations, but there are many studies about DDE in primary dentiton and dental caries as their consequences.

The aim of this study was to analyse whether there is a relationship between EEC in Czech population and its progression to inflammatory complications determined by the modified pufa score and the occurence of DDE on a permanent successor. We monitored the frequency of DDE occurrence in the ECC group and control group, the extent and the location of DDE on thirds of the dental crown.

Methods

Prior to the study, approval was obtained from the Ethics Committee of the Faculty Hospital Olomouc and the Faculty of Medicine and Dentistry, Palacký University in Olomouc (Czech Republic) (ref. No. 50/13). The study population was selected from 5-year-old patients of the Institute of Dentistry and Oral sciences, Faculty of Medicine and Dentistry, Palacký University Olomouc (Czech Republic), who presented for regular examination from 1/1/2010 to 31/12/2014 (N=304). The participation was strictly voluntary and the informed consent from legal guardian was obtained before they were included in the study.

During first examination the medical history was taken, through a questionnaire about nutrition habits (breastfeeding, diet with regard to sugars, oral hygiene), family background, socio-economical status and dmft [Klein and Palmer, 1938; Paglia et al., 2016; Colombo et al., 2019], and modified pufa index [Figueiredo et al., 2011; Monse et al., 2010] was recorded. The patients were consecutively distributed in two groups according to the carious status (dmft index) of deciduous incisors: early childhood caries group (ECCG) with dmft index >4 and control group (CG) with dmft index ≤4 and intact incisors. Groups were monitored during the regular dental examinations until the eruption of permanent upper central incisors, up to 8 years of age. After the eruption, the presence, extent and location of DDE (Table 1) were clinically evaluated by the modified DDE index [Clarkson and O'Mullane, 1989]. All indexes (dmft index, pufa score and modified DDE index) in both groups were provided by single examiner (EM).

A patient was excluded from the study if one or more of the following conditions were observed: history of dental traumatic injury, molar incisor hypomineralisation (MIH), serious systemic illness, unerupted permanent upper central incisors at 8 years, inconsistent dental appointments/check-ups, relocation or dentist change. At the final examination the ECCG consisted of 60 patients and CG of 80 patients. The recruitment of patients is summarised in Figure 1. Both groups were monitored from the age of 5 years until the full eruption of permanent upper central incisors at the age of 8 years.

The obtained data were statistically processed using the IBM SPSS Statistics version 22 statistical software (IBM, USA). To compare the ECCG with the CG in qualitative parameters, a chi-square test was used, respectively Fisher's exact test. For comparison of quantitative parameters, the Mann-Whitney U test was used. Tests were performed at a significance level lower than 0.05.

Results

The ECCG was composed by 60 children, 25 boys (41.7%) and 35 girls (58.3%). The 80 children of CG counted 37 boys (46.3%) and 43 girls (53.8%). In total, 140 children, 62 boys (44.3%) and 78 girls (55.7%) participated. The chi-square test did not show a significant difference between the groups in this parameter (gender).

The following results were obtained by evaluating anothe parameter: the modified pufa score (c=caries, p=pulpal involvement, f+a=periodontitis) on teeth 51, 61 in ECCG. Out of a total 120 evaluated temporary incisors, 58 (48.4%) were affected by caries, 10 (8.3%) by pulpal involvement, and 52 (43.3%) incisors were affected by inflammatory complications of dental caries dignosed as apical periodontitis. Totally identical was the modified pufa score for the right and left deciduous incisors separately.

The permanent upper central incisors were evaluated by the modified DDE index after the eighth year of age. The prevalence of DDE in the whole population examined of 140 children was 19.3% (54 defects). In the ECCG, 29 permanent incisors (24.2%) were diagnosed to have an enamel defect, in the CG DDE was found on 25 permanent incisors (15.6%). Data are summarised in Table 1. The location and the extent of DDE in ECCG and CG are summarised in Table 2, but in general DDE in ECCG were smaller and more often located in the incisal third of the dental crown, while enamel defects in CG were in 48% of cases larger than 2/3 of crown and 28% of DDE were located over the whole

	Diffuse opacity N (%)	Demarcated opacity N (%)	Hypoplasia N (%)	Combination N (%)	Total teeth with defects N (%)	Total teeth with defect N (%)
Tooth 11 ECCG N=60	6 (10%) 20.7%	3 (5%) 10.3%	2 (3.3%) 6.9%	3 (5%) 10.3%	14 (23.3%) 48.3%	29 (24.2%) 100%
Tooth 21 ECCG N=60	7 (11.7%) 24.1%	5 (8.4%) 17.2%	2 (3.3%) 6.9%	1 (1.3%) 3.4%	15 (25.0%) 51.7%	
Tooth 11+21 ECCG	13 44.8%	8 27.5%	4 13.8%	4 13.7%	29 100%	
Tooth 11 CG N=80	8 (10.1%) 32%	2 (2.6%) 8%	0	0	10 (12.5%) 40%	25 (15.6%) 100%
Tooth 21 CG N=80	10 (12.6%) 40%	4 (5%) 16%	0	1 (1.3%) 4%	15 (18.7) 60%	
Tooth 11+21CG N=160	72% 18	24% 6	0	4% 1	100% 25	
All teeth 11+21 N=280	31 57.4% 11.1%	14 25.9% 5%	4 7.4% 1.4%	5 9.3% 1.8%	54 (100%) All teeth with defect	280 (100%) All teeth in study 19,3%

TABLE 1 Results of development defects of enamel (DDE = development defects of enamel, EECG = early childhood caries group, CG = control group).

	E up to 1/3	E up to 2/3	E more than 2/3	E total	L incisal 1/3	L middle 1/3	L cervical 1/3	L i+m 1/3	L c+m1/3	L entire surface
DDE in ECCG N=29	20 69%	6 20.7%	3 10.3%	29 100%	15 51.7%	2 6.9%	0	10 34.5%	0	2 6.9%
DDE in CG n=25	13 52%	7 28%	5 20%	25 100%	8 32%	0	0	10 40%	0	7 28%
ECCG vs. CG p-value	0.402				0.083					

TABLE 2 The extent (E) and location (L) of development defects of enamel (DDE) in early childhood caries group (ECCG) and control group (CG) (i-incisal, m-middle, c-cervical 1/3 of tooth surface), Fisher's exact test.

labial surface. Teeth no. 11 and no. 21 were evaluated by the modified DDE index. No significant difference was found between ECCG and CG for defects present on tooth no. 11 (p = 0.059) or tooth no. 21 (p = 0.540). The results are displayed in Table 3.

The final summary shows that the prevalence of DDE on permanent teeth no. 11 and no. 21 in ECCG was 23.3% and 25.0%, whereas in the CG the DDE prevalence was 12.5% and 18.7%. Although there is a percentage difference in the incidence of DDE in the ECCG and CG, this difference is not statistically significant (p>0.05).

The modified pufa score of deciduous predecessors and the type of DDE of permanent incisors were compared to assess the association between the type of DDE and the severity of damage of the deciduous predecessor. A statistically not significant relationship was demonstrated (Table 4).

Discussion

Statistically significant relationship between ECC and the development of DDE on permanent successors was not proven, although the higher prevalence of defects as well as the difference in the extent and in the location of defects in ECC group was detected (Fig. 2). The results of this prospective original study was compared with only two available retrospective studies about the relationship between ECC and DDE as their consequences [Lo et al., Broadbent et al.2005]. The results of this study show that there seems to be an association between ECC and DDE formation, but the probable role of dental caries in the creation of enamel defects is not so unequivocal.

Tooth decay in pre-school children is still a serious problem in the Czech Republic with a prevalence of 60–65% [Broukal et al., 2009; Ivančáková et al., 2007; WHO Oral health Country/Area Profile Programme, 2004]. The ECC prevalence worldwide ranges from 1% in Australia, 6.8–12% in England, 8.2% in Italy, 11–53.1% in USA to 70% in developing countries and disadvantaged groups of developed countries [Vandana et al., 2018; Gussy et al., 2006; Colombo et al., 2019; Chen et al., 2019]. The results of the cohort studies of Lo et al. [2003] and Broadbent et al. [2005] support the conclusions from earlier studies about the existence of the relationship between ECC and DDE as its consequences on the permanent successor [Pindborg, 1983; Turner, 1906; Turner, 1912]. Broadbent et al. [2005] found a 25.7% prevalence of DDE in his study of the ECC and DDE relationship in the upper central and lateral incisors, which is comparable to the prevalence of DDE in ECCG of 24.2% in our study. The prevalence of DDE in permanent dentition of all 140 examined children was 19.3%. Broadbent´s prevalence is naturally higher, because teeth with history of dental trauma were not excluded. The percentage representation of each type of DDE is similar in both works, where results are compared (Table 5). Although the prevalence of DDE in CG (15.6%) was lower than in ECCG (24.2%), a statistical significance was not reached (Table 1). When considering

		Groups				Total	
		ECCG		CG			
		count	%	count	%	count	%
Modif. DDE index Tooth 11	Healthy (0)	46	76.7%	70	87.5%	116	82.9%
	Dem. opacity- white (1)	3	5.0%	1	1.3%	4	2.9%
	Dem. opacity- yellow (2)	0	0.0%	1	1.3%	1	0.7%
	Diff. opacity- line (3)	3	5.0%	1	1.3%	4	2.9%
	Diff. opacity- mottled (4)	3	5.0%	4	5.0%	7	5.0%
	Diff. opacity- continuous (5)	0	0%	3	3.8%	3	2.1%
	Hypolasia- pits (7)	2	3.3%	0	0%	2	1.4%
	Combination A (Dem. + Diff.op)	2	3.3%	0	0%	2	1.4%
	Combination C (Diff. op. + hypoplasia)	1	1.7%	0	0%	1	0.7%
Total		60	100%	80	100%	140	100%
		Groups				Total	
		ECCG		CG			
		count	%	count	%	count	%
Modif. DDE index Tooth 21	Healthy (0)	45	75%	65	81.3%	110	78.6%
	Dem. opacity- white (1)	4	6.7%	2	2.5%	6	4.3%
	Dem. opacity- yellow (2)	1	1.7%	2	2.5%	3	2.1%
	Diff. opacity- line (3)	2	3.3%	1	1.3%	3	2.1%
	Diff. opacity- mottled (4)	4	6.7%	7	8.8%	11	7.9%
	Diff. opacity- continuous (5)	1	1.7%	2	2.5%	3	2.1%
	Hypolasia- pits (7)	2	3.3%	0	0%	2	1.4%
	Combination A (Dem. + Diff.op)	1	1.7%	0	0%	1	0.7%
	Combination B (Dem. op.+ hypoplasia)	0	0%	1	1.3%	1	0.7%
Total		60	100%	80	100%	140	100%

TABLE 3 The Modified DDE index clinically evaluated for tooth 11 and 21 (Fisher´s exact test).

DDE type teeth 11, 21 in ECCG	pufa score teeth 51,61				
	Caries (c)		Root (p)+ peridontitis (f+a)		Summary
1 +2 Demarcated opacity	3	38%	5	63%	8
3+4+5 Diffuse opacity	7	54%	6	46%	13
7 Hypoplasia	0	0%	4	100%	4
Combination A (Dem. op. + Diff. op)	2	100%	0	0%	2
Combination C (Diff. op.+ Hypoplasia)	1	50%	1	50%	2
Summary	13	45%	16	55%	29

TABLE 4 Comparison of all types of development defects of enamel (DDE) on permanent incisors vs. the pufa score of deciduous predecessors in early childhood caries group (ECCG).

the results, it must be taken into account that in the study of Lo et al. [2003] the entire deciduous dentition and permanent incisors, canines and premolars were included, whereas this study evaluates only deciduous central incisors and their permanent successors. Excluding deciduous molars and premolars as their permanent successors significantly reduces the number of detected DDE. Another possible reason why this study failed to demonstrate a statistically significant difference in DDE prevalence is the relatively small sample size, because the study is prospective and original and it was complicated to acquire a vast sample. The study of Broadbent et al. [2005] included 663 children and that of Lo et al. [2003] 250 children (Table 2), but both studies were retrospective where previous studies were followed-up and data analysed secondarily. In our study, the detailed condition of dentition of all children was evaluated only by one operator. As in the work of Lo et al. [2003] there was no difference between prevalence of DDE and gender. From the results it appears that an association between the ECC and the presence of DDE on their successors exists (Table 4), however the probable role of ECC in the DDE formation does not seem to be essential [Costa, 2017].

The relationship between the degree of severity of ECC determined by the modified pufa score and the DDE type on a permanent successor were examined. Hypoplasia is the most serious type of DDE and is associated with severe damage of ameloblasts. The results show that hypoplasia is associated with the most severe units of the modified pufa score (p)+(f+a). This association was also observed in a work by Broadbent et al. [2005], where statistical significance was not achieved. The second defect, often associated with local irritation caused by the spreading of infection, is demarcated opacity. This work failed to prove the association between ECC and demarcated opacity formation in the permanent dentition, although the incidence of demarcated opacity was 63% after more severe damage (p)+(f+a) of the temporary predecessor (Table 4).

Diffuse opacity is a defect associated with a systemic aetiology rather than a locally acting pollutant, most often involving excessive exposure to fluoride ions during the amelogenesis process [Clarkson and O'Mullane, 1989; Broadbent et al., 2005; Lo et al., 2003]. All patiens in this study were from the same geographical area and the same

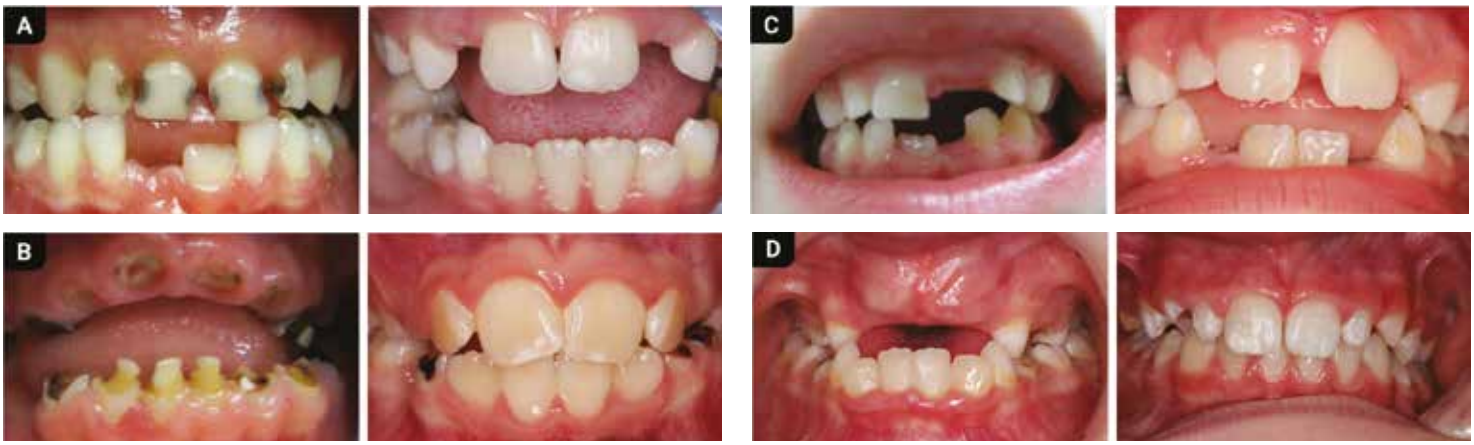

FIG. 2 Case reports of patients with EEC scored according to the modified pufa score (caries/pulpal involvement/periodontitis) in deciduous dentition and DDE on permanent incisors as their consequences.
Patient A with diagnosed ECC and the modified pufa score "caries" and diffuse opacities on permanent teeth 11,21.
Patient B with the modified pufa score "pulpal involvement" and demarcated opacities on permanent teeth 11,21.
Patient C with ECC on palatal facets scored modified pufa score as "caries" and enamel hypoplasia on tooth 11.
Patient D with extracted deciduous teeth 52,51,61,62 due to inflammation complications, "periodontitis" according to the modified pufa score and a combined enamel defect on permanent tooth 11 (diffuse+ demarcated opacity) and diffuse opacity on tooth 21.

fluoride intake can be assumed. The prevalence of diffuse opacities in this study was almost identical in both groups and no association with ECC was found (Table 4). Importantly, in the Czech Republic drinking water has not been fluoridated since 1989. The level of fluorides in drinking water in Olomouc and its vicinity is <0.31 ppm.

The extent of DDE was another evaluated parameter, which was determined by the modified DDE index. In the ECCG the extent in 16.7% of defects was less then 1/3 of the labial tooth surface, in the CG it was only 8.1%, the other extents of DDE were similar in both groups. Defects of the extent less than 1/3 of the tooth are small-size DDE, mostly hypoplasia and demarcated opacity, which are associated with a short and intense impact on the course of amelogenesis. On the contrary, diffuse opacity is a larger lesion caused by a long-term excess fluoride intake [Lo et al., 2003; Broadbent et al., 2005]. DDE on permanent teeth, whose deciduous predecessor suffered from ECC, were in general of smaller sizes and occurred more frequently than DDE in the CG. DDE on teeth with intact predecessors were percentually more extensive (20% of DDE in CG were larger than 2/3 of labial facet, in ECCG it was only 10%), less frequent in appearance, and hypoplasia as the most serious enamel defect occurred in one case and only as part of a combined defect (Table 2).

The last observed parameter was DDE location. In the ECCG, 27 (93.1%) of the 29 DDE were located to incisal 2/3 of dental crown and two defects were located across the entire labial surface. In the CG 18 defects (72%) out of a total of 25 were located in incisal 2/3 dental crowns and the remaining 7 defects were located throughout the whole labial area (Table 2). Langlais et al. [2009] showed the association between the locationof the DDE in the thirds of the labial area of central incisors with the expected time of its creation. Based on this fact we can estimate that almost all DDE of the ECCG occurred before two years of age, and the remaining two DDE are related to the long-lasting noxae until the third year of age. Within the CG, we estimate that 18 (72%) lesions in the incisal 2/3 of the tooth surface were caused by an insult in the first two years of age, and 7 defects located on the entire facet were related to a nox lasting from birth to 3 years of age.

The aim of this study was to examine the relationship between ECC and the development of DDE on a permanent successors, which was not statistically significant. The higher prevalence of DDE on permanent teeth in a group of children with ECC in temporary dentition as well as the difference in extent and location of the defects did not reach statistical

	Patients teeth, tooth type	Teeth with DDE	DDE p (%)	Diffuse opacity p (%)	Demarcated opacity p (%)	Hypoplasia p (%)	Combined defects p (%)
Lo et al. (2003)	250 5639 i, c, p	X	x	x	56.8	10.8	x
Broadbent et al. (2005)	663 2652 i1, i2	616	25.7	15.2	6	2.2	2.3
Míšová et al. (2019)	140 280 i1	54	19.3	11.1	5	1.4	1.8

TABLE 5 The comparison of findings in similar studies observing development defects of enamel (DDE) (p= prevalence)

significance. The probable role of ECC in DDE formation was not significant and it will be necessery to arrange more studies in order to further elucidate this problematic in future.

Bullet points

ECC is one of most common and most expensive diseases in children with potential risk of damage to the permanent teeth.

There are only two aepidemiological studies dealing with ECC and DDE as its consequences and we have only limited aepidemiological evidence of the relationship between ECC and DDE.

Acknowledgments

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