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Long-Term Survival of Enamel-Defect-Affected Teeth

Chuen Lin Hong Jonathan Mark Broadbent William Murray Thomson

Department of Oral Sciences, Faculty of Dentistry, University of Otago, Dunedin, New Zealand

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Epidemiology · Caries prediction · Developmental defects

Abstract
There has been considerable research focussed on the occurrence and aetiology of developmental defects of enamel, but less is known about the extent to which enamel-defect-affected teeth may be at greater risk for dental caries. The Dunedin Multidisciplinary Health and Development Study is a prospective cohort study of 1,037 children born in Dunedin, New Zealand, between April 1, 1972, and March 31, 1973. Participants were examined for the presence of developmental defects of enamel at the age of 9 years and then repeatedly for the occurrence of dental caries through to the age of 45 years. After controlling for confounding variables, incisor teeth affected by demarcated opacities at the age of 9 were 3.4 times more likely to be restored than teeth unaffected by defects. Incisors with diffuse opacities and hypoplasia or combinations of defects were 2.8 times more likely to be restored. Molars with enamel defects of any type did not have any significantly different risk for being subsequently restored or lost due to caries than unaffected molars, except those affected by diffuse opacities, which were at 0.4 times the risk of being lost due to caries. Dental clinicians should be aware that enamel-defect-affected teeth are not necessarily at greater risk for tooth loss due to caries in the long term, but permanent incisors affected by enamel defects are at higher risk of receiving restorative intervention.

Introduction

A considerable body of research has investigated the occurrence and aetiology of developmental defects of enamel, but less research has considered the extent to which enamel-defect-affected teeth may be at greater risk for dental caries. Much of what is known about this association comes from cross-sectional studies among children [King and Wei, 1986; Ellwood and O'Mullane, 1996; Li et al., 1996; Mackay and Thomson, 2005; Carvalho et al., 2011; Vargas-Ferreira and Ardenghi, 2011; Vargas-Ferreira et al., 2014, 2015; Costa et al., 2017]. A 2015 systematic review and meta-analysis considered findings from seven cross-sectional studies and concluded that defect-affected permanent teeth have over twice the risk for dental caries than unaffected teeth [Vargas-Ferreira et al., 2015]. Another review considered outcomes for defect-affected primary teeth and found that they had 3.3 times the odds (95% confidence interval, CI, 2.4–4.6) of being affected by dental caries than unaffected teeth.

karger@karger.com
www.karger.com/cre

Chuen Lin Hong
Department of Oral Sciences
Faculty of Dentistry, University of Otago
Dunedin 9054 (New Zealand)
c.hong@otago.ac.nz

Teeth with hypoplastic defects had 4.3 times the odds (95% CI 2.2–8.2) of being affected by caries than sound teeth, while those with diffuse defects had an only 1.4 times (95% CI 1.2–1.8) greater risk [Costa et al., 2017].

These cross-sectional studies suggest that teeth affected by enamel defects are more susceptible to dental caries, but do not help explain whether this risk is life-long or limited to the short term following tooth eruption. Four longitudinal studies have considered the occurrence of dental caries among defect-affected teeth [Lai et al., 1997; Oliveira et al., 2006; Hong et al., 2009; Targino et al., 2011], but the duration of follow-up for the occurrence of dental caries among these studies was only 36–60 months, and all four studies considered only primary teeth. The most recent of these studies followed 224 Brazilian children from 12 to 54 months of age and found that those with enamel defects had twice the risk of developing dental caries [Targino et al., 2011].

Caries may progress rapidly, with teeth affected shortly after eruption, or it may develop too slowly to be detected in a short-term study. The greater caries susceptibility of enamel-defect-affected teeth may mean that these teeth are more likely to be restored at some stage during the life course. Restorations do prolong the time before eventual failure of a tooth, but the porous structure of hypomineralised enamel makes bonding of restorations unreliable [William et al., 2006a]. Past studies have demonstrated lower bonding strength of resin composites to hypomineralised enamel than occurs with sound enamel [Chay et al., 2014; William et al., 2006b]. Accordingly, a restored defect-affected tooth may require repeated restorative interventions [Jälevik and Klingberg, 2002]. Repeated restoration failure may hasten the eventual loss of a caries-affected tooth. No studies have evaluated the extent to which enamel defects may increase the risk of caries-associated tooth loss into middle age.

Additionally, enamel defects have a wide range of clinical presentations. Enamel defects can be categorised as diffuse opacities, demarcated opacities or hypoplastic defects. It may present as a well-circumscribed area or be widespread across the surface of a tooth. Affected teeth may be discoloured or differ in texture and/or translucency. These presentations are not likely to have negative aesthetic impact for posterior teeth but may be expected to do so for affected maxillary anterior teeth [Mackay and Thomson, 2005]. Defect-affected incisors may become carious or be restored at an earlier age than sound teeth, affecting their appearance, but the extent to which this is a problem is unclear. Enamel defects do not necessarily affect a child's perception of his/her own oral health but may affect oral function [Vargas-Ferreira and Ardenghi, 2011]. Other research on the implications of enamel defects for dental aesthetics has found that mild dental fluorosis can diminish with time [Do et al., 2016] and may even have a positive impact on oral health-related quality of life [Do and Spencer, 2007].

Accordingly, this birth cohort study investigated the longer-term restorative fate of teeth with enamel defects noted in childhood.

Materials and Methods

The "Dunedin Multidisciplinary Health and Development Study" is a longitudinal study of human development and health, of a birth cohort born in Dunedin, New Zealand [Poulton et al., 2015]. The cohort consists of children born at Queen Mary Maternity Hospital in Dunedin, New Zealand, between April 1, 1972, and March 31, 1973. Perinatal data were collected at the time of birth, and the cohort for the longitudinal study was defined at the age of 3 years. The cohort of 1,037 children were assessed within a month of their third birthdays and then at the ages of 5, 7, 9, 11, 13, 15, 18, 21, 26, 32, 38 and 45 years. Over 90% of study members self-identify as being of New Zealand European origin. The study protocol was approved by the Health and Disability Ethics Committees, Ministry of Health, New Zealand. Study members gave informed consent before participating.

At the age of 9, study members were examined for the presence of enamel defects using the Developmental Defects of Enamel index [Suckling et al., 1985; Mohamed et al., 2010]. Each fully erupted tooth was examined for demarcated opacities, diffuse opacities, hypoplasia, other defects or combinations of defects. For the current study, we considered defects on the buccal surfaces of the eight incisor teeth and defects on either the buccal and lingual surfaces of the permanent first molars. Defects were categorised as "demarcated opacities," "diffuse opacities" and "hypoplasia/other defects" (combined due to the small number of teeth with hypoplasia and other defects or combinations of defects). Defects of <2 mm diameter were excluded and, if a tooth was affected by more than one type of enamel defect it was grouped into "hypoplasia/other defects."

In addition to the examination for developmental defects of enamel at the age of 9 years, clinical examinations of oral health were conducted at ages 5, 9, 15, 18, 26, 32, 38 and 45 by calibrated examiners. Teeth were examined for dental caries and restorations, with four surfaces (buccal, lingual, distal and mesial) for incisors and a fifth surface (the occlusal) included for permanent first molars. An estimate of caries-associated tooth loss was obtained by observing the presence or absence of each tooth at each assessment and ascertaining reasons for its absence at each age by asking the study member and considering the previous status of the tooth. At the age of 45, the intra-examiner reliability in scoring of count of decayed, missing and restored tooth surfaces was 0.99 for each of the three examiners. Inter-examiner reliability intraclass correlation coefficient scores were 0.97 (examiners 1 and 2), 0.95 (examiners 1 and 3) and 0.99 (examiners 2 and 3). The nature of data collection meant that the exact time the tooth became carious, lost or restored could not be determined exactly. For the purposes of analysis, we assumed that the event of interest occurred at the time of the examination (for those who were

examined at all phases before the event of interest occurred), or where there were missing data due to study members not attending an assessment phase, the midpoint (in years) between the most recent assessment phase when the event of interest occurred and the most recent assessment phase was used instead. The survival time was defined as: (i) the time by which the tooth was restored or restored and decayed (incisors: buccal, permanent first molars: any surfaces) or (ii) the time the tooth was recorded as missing due to caries, or the time from baseline examination at the age of 9 to the last follow-up visit in cases of censoring.

Study members were allocated to life course plaque trajectories using group-based trajectory analysis [Jones et al., 2001]. Three plaque trajectories had previously been identified using the Simplified Oral Hygiene Index [Greene and Vermillion, 1964] measurements at the ages of 5, 9, 15, 18, 26 and 32, as previously reported by Broadbent et al. [2011]. Socio-economic status (SES) was measured according to the New Zealand Socio-Economic Index 2006 [Milne et al., 2013], a six-group occupation-based measure of SES. Examples of occupations in the six categories include: 6 = medical practitioner, legal professional; 5 = financial broker, engineering professional; 4 = database administrator, electrician; 3 = printing trades worker, personal assistant; 2 = office cashier, floor finisher; 1 = cleaner, truck driver. The dental visiting pattern was determined using the study members' self-reported dental visiting behaviours at the ages of 26, 32, 38 and 45. Study members were asked whether they usually visited the dentist for a check-up or only for a dental problem, together with the number of months since their last visit. For each of the ages, routine attenders were identified as those who (a) usually visited for a check-up and (b) had made a dental visit during the previous 12 months. Those who were identified as routine attenders at 50% or more of the assessments (at which they participated) were classified into the "routine attenders" group.

In this study, analyses were limited to the eight incisors (teeth 11, 12, 21, 22, 31, 32, 41, 42) and four permanent first molars (16, 26, 36, 46) because canines and premolars have usually not fully erupted by the age of 9. Bivariate tests for the statistical significance of associations between categorical variables were conducted using the χ^2 test by tooth type (incisors and molars). The underlying risk of failure outcomes (tooth loss and restoration) would depend on person characteristics and can differ among study members. As such, we fitted shared frailty survival models which adjusted the survival estimates for both within- and between-person differences. To investigate whether enamel defects pose a risk for restorations and tooth loss due to caries, hazard ratios and CIs were calculated for enamel defects using Cox proportional hazard regression models. These models accounted for confounders such as SES, sex, oral hygiene and the use of dental services. Separate survival analyses were conducted for (a) buccal restorations by all incisors, (b) buccal restorations by upper incisors, (c) caries-related tooth loss by molars and (d) restoration of any surfaces by molars. All analyses were conducted using IC STATA 14 software (Stata, TX, USA).

Results

The participation rate in the Dunedin Multidisciplinary Health and Development Study was 92.3% (n = 955) at the age of 9 years, and 696 were assessed for the presence of enamel defects. At the age of 45, the participation rate was 94.1% (n = 938 of the surviving 997 study members), and 896 were dentally examined. Of the 696 study members dentally examined for enamel defects at the age of 9, 37 were not dentally examined at two or more phases since the age of 9 and were excluded. Subsequent analyses were limited to 659 study members. Just over half (52.8%) were male, of medium childhood SES (64.8%) and were routine attenders in adulthood (60.9%). No differences existed by sex, SES and use of dental services in adulthood between included and excluded study members.

Prevalence of Study Members with 1+ Developmental Defects of Enamel

At the age of 9 years, more than half of the study members (55.8%) had at least one enamel-defect-affected incisor or permanent first molar. Almost half (45.1%) had at least one enamel defect of at least 2 mm in diameter on an incisor tooth's buccal surface. One in 3 study members (33.2%) had at least one permanent first molar with an enamel defect.

Prevalence of Teeth with Developmental Defects of Enamel

Of the 4,940 incisors examined, nearly 1 in 5 had an enamel defect (n = 853, 17.3%). Enamel defects on the incisor teeth most frequently presented as diffuse opacities (9.4% of teeth), followed by demarcated opacities (4.0%) and hypoplastic/other defects (3.9%). Of the 2,611 permanent first molars examined, 1 in 5 (n = 533, 20.4%) was affected on either the buccal or lingual side (or both). Diffuse opacities (9.8% of teeth) were the most common defects followed by demarcated opacities (8.0%) and hypoplastic/other defects (2.6%).

Risk for Restoration and Caries-Related Tooth Loss

Table 1 shows the bivariate associations between the presence of enamel defects (age 9) and restoration status and caries-related tooth loss (age 45) by tooth type (incisors or molars). Incisors affected by enamel defects at the age of 9 were significantly more likely to have a buccal restoration. Insufficient permanent incisors (n = 7) were missing due to caries to justify further analysis of incisor tooth loss by the presence of defects. Defect-affected permanent first molars were more likely to be restored and missing due to caries at the age of 45.

The proportions of incisors and permanent first molar teeth restored from the age of 9 to 45 are shown in Figures 1 and 2, respectively. Among incisors, proportionally

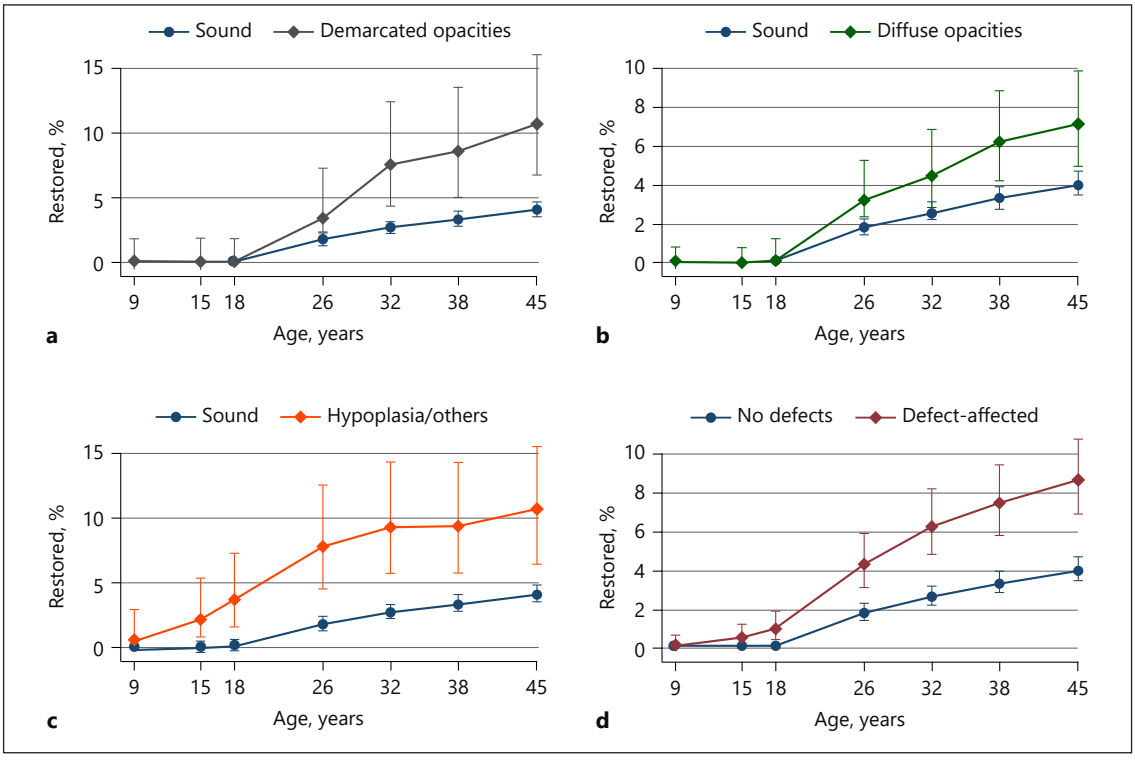

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Fig. 1. Percentage of incisors restored (95% CI) by defect types from the ages of 9 to 45. **a** Sound vs. demarcated opacities. **b** Sound versus diffuse opacities. **c** Sound versus hypoplasia. **d** Sound versus any defects.

Table 1. Types of enamel defect at the age of 9 by restoration status and caries-related tooth loss at the age of 45 (data are the number of teeth)

Defect type at the age of 9	Tooth status by the age of 45			
	incisors		molars	
	missing due to caries, *n* (%)	restored, *n* (%)	missing due to caries, *n* (%)	restored, *n* (%)
Sound	120 (2.9)[a]	166 (4.1)[b]	236 (11.4)[b]	1,395 (75.7)[a]
Demarcated opacity	1 (0.5)	21 (10.8)	31 (14.9)	141 (79.7)
Diffuse opacity	1 (0.2)	33 (7.1)	12 (4.7)	177 (72.5)
Hypoplasia/others	5 (2.6)	20 (10.3)	8 (11.6)	57 (93.4)
All combined	127 (2.6)	240 (4.9)	287 (11.0)	1,770 (76.2)

[a] χ^2 test, $p < 0.005$. [b] χ^2 test, $p < 0.001$.

more demarcated opacities and hypoplasia/other defect-affected teeth were restored on the buccal surfaces. By the age of 45, 10.8% (95% CI 6.8–16.0) and 10.3% (95% CI 6.4–15.5) of incisors with demarcated opacities and hypoplasia/other defects had been restored, while this was the case for only 4.1% (95% CI 3.5–4.7) of sound incisors.

Among molar teeth, significant differences in the proportion restored were observed for demarcated opacities (ages 9–18) and hypoplasia/other (across all ages). By the age of 45, 94.2% (95% CI 85.8–98.4) of molars with hypoplasia or other defects were restored, and this was 76.5% (95% CI 74.6–78.3) for unaffected teeth.

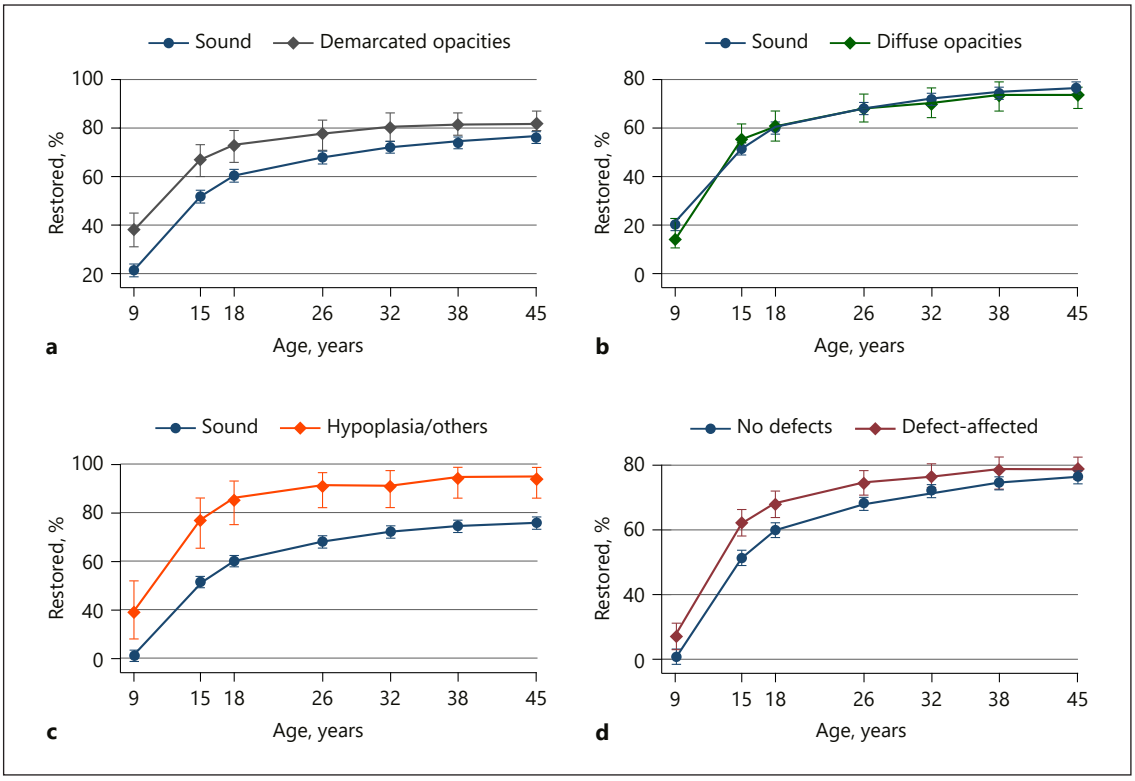

Fig. 2. Percentage of molars restored (95% CI) by defect types from the ages of 9 to 45. **a** Sound versus demarcated opacities. **b** Sound versus diffuse opacities. **c** Sound versus hypoplasia. **d** Sound versus any defects.

Association of Restoration and Caries-Related Tooth Loss: Survival Analysis

After controlling for confounding variables, incisor teeth affected by demarcated opacities at the age of 9 were 3.4 times more likely to be restored than teeth unaffected by defects (Table 2). Incisors with diffuse opacities and hypoplasia or combinations of defects were 2.8 times more likely to be restored. Because teeth in the aesthetic zone may be restored at an earlier age for aesthetic reasons, separate survival analysis was conducted for the four upper incisors. Where demarcated opacities or hypoplasia/other enamel defects were recorded on the buccal surfaces at the age of 9, the upper incisors were 2.7 more likely to be restored. No differences were noted for upper incisors with diffuse opacities. Molars affected by diffuse opacities were 0.4 times more likely to be affected by caries than teeth without developmental defects. Molars with enamel defects at the age of 9 were no different to sound teeth in restoration status by the age of 45. Caries-related tooth loss of molars was more common among those who were problem-oriented dental visitors.

Discussion/Conclusion

This study found that permanent incisor teeth affected by developmental defects of enamel at the age of 9 (regardless of type of defect) were 3 times more likely to have been restored by the age of 45 years than teeth with developmentally normal enamel. Permanent first molars affected by developmental defects of enamel were not at a greater risk of being restored or lost due to dental caries by the age of 45 years.

In the current study, analyses were limited to incisor and permanent first molar teeth, and defects <2 mm were excluded. Thus, the prevalence of enamel defects may be underestimated. However, we did not set out to describe the occurrence of developmental defects of enamel in the entire permanent dentition. Furthermore, permanent first molars and permanent central incisors are most commonly affected by developmental defects of enamel [Seow et al., 2011]. Study members of this study were dentally examined for enamel defects at the age of 9. The permanent first molar erupts at about the age of 6 years. Ma-

Table 2. Survival analysis models[a] for teeth being restored (incisors: buccal surface and permanent first molars: all surfaces) and missing due to caries (permanent first molars only) by the age of 45

	Incisors, HR (95% CI)		Molars, HR (95% CI)	
	all incisors restored or decayed	upper incisors restored or decayed	missing due to caries	restored or decayed
Sex				
Male	–	–	–	–
Female	1.50 (1.02–2.22)[b]	1.51 (0.96–2.38)	1.49 (0.99–2.25)	0.95 (0.74–1.22)
Defect type				
No defect	–	–	–	–
Demarcated opacities	3.39 (2.00–5.76)[b]	2.71 (1.49–4.93)[b]	1.32 (0.82–2.13)	0.86 (0.62–1.18)
Diffuse opacities	2.75 (1.66–4.55)[b]	1.58 (0.91–2.74)	0.40 (0.18–0.85)[b]	0.89 (0.65–1.22)
Hypoplasia/others	2.80 (1.50–5.24)[b]	2.72 (1.34–5.50)[b]	1.07 (0.42–2.75)	1.51 (0.83–2.74)
Plaque group				
Low	–	–	–	–
Moderate	0.77 (0.42–1.41)	0.68 (0.34–1.37)	0.49 (0.28–0.86)[b]	0.72 (0.46–1.14)
Heavy	0.53 (0.27–1.05)	0.43 (0.19–0.95)[b]	0.17 (0.09–0.33)[b]	0.63 (0.38–1.02)
Childhood SES				
High	–	–	–	–
Medium	0.88 (0.50–1.55)	0.94 (0.49–1.83)	1.38 (0.71–2.69)	1.22 (0.87–1.71)
Low	1.02 (0.51–2.04)	1.27 (0.57–2.84)	2.29 (1.09–4.80)[b]	1.41 (0.91–2.18)
SES at the age of 45				
High	–	–	–	–
Medium	1.66 (1.04–2.64)[b]	1.66 (0.97–2.84)	2.31 (1.35–3.96)[b]	1.06 (0.81–1.39)
Low	1.77 (0.96–3.26)	1.66 (0.81–3.40)	4.62 (2.50–8.54)[b]	1.01 (0.68–1.51)
Dental attendance in adulthood				
Routine attenders	–	–	–	–
Episodic users	1.19 (0.79–1.80)	1.05 (0.65–1.70)	1.94 (1.21–3.11)[b]	1.09 (0.85–1.40)

HR, hazard ratio; CI, confidence interval; SES, socio-economic status. [a] Cox proportional hazard model. [b] $p < 0.05$.

honey and Morrison [2011] reported that 33.9% of children with demarcated opacities experienced posteruptive breakdown on the affected teeth. It is possible that some defect-affected teeth, especially those more severely affected, may have been restored and/or could have been masked by caries in the 3-year posteruption window prior to examination. However, the reported prevalence of demarcated opacities at the tooth level in this study is comparable to other New Zealand studies of 9- to 10-year-olds. Schluter et al. [2008] found that 3% of teeth had demarcated opacities while Mackay and Thomson [2005] and de Liefde and Herbison [1985] reported a prevalence of 8.3 and 8.9%, respectively.

In this study, some study members were not examined at every age, leading to some missing data. Thus, the year of tooth loss or restoration will be more accurately recorded for those who participated at all ages than those who missed certain assessments. However, the study aimed to identify the long-term survival of enamel defect-affected teeth, not dental caries experience specifically. Jälevik and Klingberg [2012] reported that children with severe molar incisor hypomineralisation on their permanent first molars were more likely to require repeated dental treatments. The current investigation did not account for possible "re-restoration" of the teeth. The nature of the caries data collection meant that it is difficult to account for restoration replacement over the life course. Hence, findings of this study do not fully reflect the possible failure of restorations into mid-life nor does it reflect the possible greater rate of recurrent caries around dental restorations on affected teeth. Restoration of a tooth is not the "end point" of treatment since once restored, the tooth enters a restorative cycle which may end in tooth mortality, which is what this study aimed to investigate.

The long-term survival of enamel defect-affected teeth from childhood to early adulthood has been underresearched in the past. Previous work has investigated the association between enamel defects and dental caries during childhood and adolescence [King and Wei, 1986; Li et

al., 1995; Mackay and Thomson, 2005; Hong et al., 2009; Vargas-Ferreira et al., 2014], but none has considered whether enamel defects affect the risk that a tooth will be subsequently affected by dental caries or its sequelae through into adulthood. The current findings do not provide support for the notion that enamel defects increase the risk of tooth loss; rather those of lower adult SES or who were episodic users of dental care had a greater risk of losing teeth due to caries. These findings are similar to previously identified risk factors for tooth loss [Eklund and Burt, 1994; Thomson et al., 2000]. Permanent first molars with diffuse opacities were less likely to be missing due to caries. Diffuse opacities are indicative of higher fluoride exposures in childhood [Suckling and Pearce, 1984; Broadbent et al., 2005; Mackay and Thomson, 2005]. Favourable exposure to fluoride during childhood from community water fluoridation (or topical fluoride sources which continued into adulthood) may explain the lack of tooth loss seen among study members with diffuse opacities.

Defect-affected permanent first molars were not more likely to be restored, but such an association was found for incisors. Because the aesthetic impact of these defects was not directly assessed, we can infer only that there may have been an aesthetic motivation for the restorations of those incisor tooth surfaces. At each assessment from the age of 9 to 45 years, proportionally more defect-affected incisors had been restored. The survival modelling findings suggested defect-affected incisors to be more likely to be restored at all ages, with certain types of defect having a higher risk than others. The hazard ratio was almost 1.5 times greater for demarcated opacities than diffuse opacities, suggesting that the former have a greater aesthetic impact. Diffuse opacities fade with time and become less noticeable, through the posteruptive processes of normal enamel wear, enamel maturation and further mineralisation [Do et al., 2016; Wong et al., 2016]. They are also often very mild, and some evidence suggests that they are not undesirable [Do and Spencer, 2007] and, so, may be less likely to be restored. Our findings showing that upper incisors with diffuse opacities were less likely to be restored than teeth with demarcated opacities or hypoplasia/other defects were consistent with this. The presence of hypoplasia/combination of other defects was also associated with a higher likelihood of restoration of the incisors, but this was lower than that for teeth affected by demarcated opacities. From an aesthetic view, hypoplastic incisor teeth where the surfaces have thinner enamel and may present as linear pits, fissures or grooves on tooth surfaces should be a strong motivator for aesthetically driven dental treatment.

Enamel defect-affected teeth are not necessarily at greater risk for tooth loss due to caries in the long term, but enamel defects of the permanent incisors are associated with a higher rate of restorative intervention. Further longitudinal research into the long-term survival of enamel defect-affected teeth is needed to investigate the extent of restorations on affected teeth. Clinicians should consider strategies to delay the initial restoration of teeth affected by developmental defects of enamel and consider non-restorative strategies for improving the appearance of defect-affected anterior teeth.

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Statement of Ethics

Ethical approval was obtained from the Health and Disability Ethics Committees, Ministry of Health, New Zealand (17/STH/25/AM05).

Disclosure Statement

None of the authors reported any conflict of interest.

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Author Contributions

All authors contributed to the conception, design, data acquisition, analysis and interpretation, drafted and critically revised the manuscript. All authors gave final approval and agree to be accountable for all aspects of the work.

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