

ORIGINAL ARTICLE

Prevalence of non-cavitated lesions and progression, regression, and no change from age 9 to 23 years

Mahrukh Zafar BDS, MS^{1,2}  | Steven M. Levy DDS, MPH^{1,3}  |
John J. Warren DDS, MS¹  | Xian Jin Xie PhD^{1,4,5} | Justine Kolker DDS, MS, PhD⁶ |
Chandler Pendleton MS¹

¹Department of Preventive and Community Dentistry, College of Dentistry, University of Iowa, Iowa City, Iowa, USA

²Department of Community Dentistry, Rawal Institute of Health Sciences, Islamabad, Pakistan

³Department of Epidemiology, College of Public Health, University of Iowa, Iowa City, Iowa, USA

⁴Division of Biostatistics and Computational Biology, Iowa Institute for Oral Health Research, University of Iowa, Iowa City, Iowa, USA

⁵Department of Biostatistics, College of Public Health, University of Iowa, Iowa City, Iowa, USA

⁶Department of Operative Dentistry, College of Dentistry, University of Iowa, Iowa City, Iowa, USA

Correspondence

Mahrukh Zafar, Department of Preventive and Community Dentistry, College of Dentistry, University of Iowa, Iowa City, IA, USA.
Email: mzafar@uiowa.edu; mahrukhz18@gmail.com

Funding information

Delta Dental of Iowa Foundation; U.S. National Institute of Health, Grant/Award Numbers: M01-RR00059, R01-DE09551, R01-DE12101, R03-DE023784, R56-DE012101, UL1-RR024979, UL1-TR000442, UL1-TR001013; Roy J. Carver Charitable Trust

Abstract

Objectives: Some non-cavitated caries lesions (D₁), the initial stage of caries, progress to cavitation. This article reports participant-level and surface-level D₁ prevalence and changes in status of D₁ lesions through different periods from age 9 to 23.

Methods: The Iowa Fluoride Study (IFS) participants were followed longitudinally; all permanent tooth surfaces were examined clinically for caries at ages 9, 13, 17, and 23 using standardized criteria for sound (S), questionable (D₀), non-cavitated (D₁), cavitated (D₂₊), filled (F), or missing due to decay (M). D₁ lesions at the beginning of each interval were reassessed at each follow-up age to determine transitions (to the 5 categories or no transition).

Results: The sample had relatively high socioeconomic status (SES), with about 52%–55% high SES, 32–35% middle SES, and 12–13% low SES. Person-level prevalences of D₁ lesions were 23%, 38%, 60%, and 45% at ages 9, 13, 17, and 23, respectively. Surface-level prevalences were less than 1% at ages 9 and 13, 3% at 17, and 2% at 23. Thirteen percent of D₁s at age 9 progressed at 13, 18% progressed from 13 to 17, and 11% progressed from 17 to 23. The percentages regressing (to sound or D₀) were 72%, 54%, and 72%, respectively.

Conclusion: Non-cavitated lesions were more prevalent at age 17 than at ages 9, 13, and 23. The high rates of regression compared to progression or no change suggest that many non-cavitated lesions do not progress to cavitated lesions and could be reversed; therefore, surgical intervention should not be the treatment of choice for incipient lesions.

KEYWORDS

adolescents, non-cavitated lesions, prevalence, progression, transition, white spot lesions

INTRODUCTION

Half of the world's population (3.58 billion people) is affected by oral diseases, and dental caries of the permanent teeth was determined to be the most prevalent oral health condition worldwide [1]. Oral scientists and dental practitioners have advocated various strategies for

appropriate management of various stages of caries. New caries management protocols have been developed considering the various levels of caries risk [2, 3], combining aspects of both therapeutic and preventive strategies for caries management [4]. The term “minimally invasive dentistry” has also become very popular [5, 6]. This conservative approach toward dental caries is based on the

This is an open access article under the terms of the [Creative Commons Attribution-NonCommercial](https://creativecommons.org/licenses/by-nc/4.0/) License, which permits use, distribution and reproduction in any medium, provided the original work is properly cited and is not used for commercial purposes.

© 2022 The Authors. *Journal of Public Health Dentistry* published by Wiley Periodicals LLC on behalf of American Association of Public Health Dentistry.

facts that dental caries is preventable and costly to treat [6]. It highlights the significant role of social and other determinants, beside the biological process of caries development [6]. Therefore, it is emphasized that, since dental caries is preventable, arrestable, and reversible, we should focus more on preventing disease progression rather than treating it with restorations at later stages [7, 8].

The caries status of a given tooth may or may not change over time. A sound tooth can remain sound or develop a caries lesion, while a carious lesion can either maintain its state, progress into a deeper lesion or regress to an arrested or sound status. This fate of a caries lesion is dependent on the balance between remineralization and demineralization [7, 9]. The initial stage of caries is the non-cavitated enamel lesion which is not painful [10] and can gradually progress to cavitation if remineralization measures are not taken to halt its progression. Machiulskiene et al. [11] used the term “initial caries lesion” for them and defined them as early caries lesions that could have been present in the mouth for a lifetime. The term is defined on the basis of the stage of severity, rather than lesion activity. Failure to assess and report these non-cavitated enamel lesions underestimates the total caries prevalence and results in failure to identify potential, future oral health problems for patients [12].

Non-cavitated lesions are significantly more prevalent than cavitated carious lesions [10, 12] and data suggest that only a small proportion of individuals remain unaffected by tooth decay after taking into account non-cavitated lesions [13, 14]. Moreover, considering the fact that caries management strategies in dentistry recently have become more focused toward prevention and conservative approaches, assessing non-cavitated lesions is essential for complete understanding of the true treatment needs of patients and populations [15]. Even though there is some research published on similar topics worldwide [14–24], there is none specifically related to the prevalence and progression of non-cavitated carious lesions in permanent teeth, particularly in the United States [17, 25, 26]. Therefore, it is important to study caries progression and differences by age. The purpose of this article is to report on the prevalence of non-cavitated lesions and their progression and regression across different periods from age 9 to 23.

METHODS

This article is based on secondary analyses of data from the Iowa Fluoride Study (IFS), a prospective cohort study that followed a birth cohort recruited from 1992 to 1995 in the maternity wards of 8 Iowa hospitals. The study followed the cohort for 23 years and regularly collected fluoride exposure, dietary and other related information associated with dental fluorosis and caries. The primary goals of the IFS were to estimate fluoride intakes

of participants and to study associations between these intakes and dental fluorosis and dental caries [27].

Approval for the study was obtained from the University of Iowa Institutional Review Board (IRB) initially in 1991 and annually, as well as when there were any changes to the study protocol or study personnel [28]. Parental informed consent was obtained at recruitment and at each examination through age 17, and participant assent at ages 13 and 17 and consent at 23 were obtained.

The participants underwent dental examinations for the assessment of permanent tooth dental caries up to 4 times during the study period at approximately ages 9 (mixed dentition, but only permanent teeth used in these analyses), and 13, 17, and 23 (permanent dentition) using IFS non-cavitated/cavitated criteria [17] modified from Pitts' criteria [29]. The examinations were conducted by 5 trained and calibrated examiners over the course of study, with a subset of 3 examiners per wave and 2 examiners the same at each subsequent examination. The IFS scoring criteria defined five categories: sound (S), questionable (D_0), non-cavitated (D_1), and cavitated (D_{2+} for enamel and dentin combined) caries lesions, and filled (F); sealed status was scored separately, but not considered directly in the assessments. In the absence of any lesions on the same surface as a sealant, sealed surfaces were considered sound, so instances where a surface that initially had a D_1 lesion, but later had a sealant, were considered regression. (Table 1) Most examinations were conducted at the University of Iowa Clinical Research Center, with some completed at remote locations in Des Moines and Waterloo, Iowa. The clinical examinations were conducted using portable equipment without radiographs. A lighted dental mirror system (DenLiteB, Welch-Allyn Medical Products, Inc., Skaneateles Falls, NY) and compressed air were used to improve visualization, and dental explorers were used only to confirm questionable findings. All children's teeth were brushed prior to the examinations [17].

Sociodemographic information was collected in 2007 by questionnaire when the sample was about 12–15 years of age. The questionnaires collected information about sex, race/ethnicity, annual family income, and maternal educational level. Composite socioeconomic status (SES) was defined based on annual family income and maternal educational level. Low SES families were defined as those with family income <\$40,000 and any level of maternal education less than graduate/professional school. Middle SES families had annual family income <\$40,000 and maternal education level of graduate/professional school or annual family income from \$40,000–\$79,999 and any level of maternal education less than graduate/professional school. High SES families had a family income between \$40,000 and \$79,999 and maternal education level of graduate/professional school or annual family income of \geq \$80,000, irrespective of maternal educational level.

TABLE 1 Scoring criteria for the Iowa Fluoride Study [Color table can be viewed at wileyonlinelibrary.com]

0 – No Caries [^]	50 – Filled, No Decay [#]	60 – Sealant, Fully Retained and No Decay [^]
10 - Pit and Fissure Questionable D ₀ [§]	11 - Pit and Fissure Incipient Caries D ₁ ^{**}	12 - Pit and Fissure Frank Caries D ₂ ^Δ
20 - Smooth Surface Questionable D ₀ [§]	21 - Smooth Surface Incipient Caries D ₁ ^{**}	22 - Smooth Surface Frank Caries D ₂ ^Δ
30 - Filled and Separate Questionable Lesion	31 - Filled and Separate D ₁ Decayed (Incipient)	32 - Filled and Separate D ₂ Decayed (Frank) [#]
40 - Filled and Questionable Recurrent Lesion	41 - Recurrent D ₁	42 - Recurrent D ₂
70 - Sealant, Fully Retained and Separate Questionable Lesion [§]	71 - Sealant, Fully Retained and Separate D ₁ ^{**}	72 - Sealant, Fully Retained and Separate D ₂ ^Δ
73 - Sealant, Fully Retained and Recurrent Questionable Lesion	74 - Sealant, Fully Retained and Recurrent D ₁	75 - Sealant, Fully Retained and Recurrent D ₂
90 - Sealant, Partially Retained and Separate Questionable Area [§]	91 - Sealant, Partially Retained and Separate D ₁ ^{**}	92 - Sealant, Partially Retained and Separate D ₂ ^Δ
93 - Sealant, Partially Retained and Recurrent Questionable Area	94 - Sealant, Partially Retained and Recurrent D ₁	95 - Sealant, Partially Retained and Recurrent D ₂
80 - Sealant, Partially Retained and No Decay [^]	99 - Unable to Score/Excluded	

Note: Colored codes used in this study (The IFS scores that were not used in this study were not coded with any color): ■, [^]No caries/sound; ■, [§]Questionable (D₀); ■, ^{**}Non-cavitated enamel lesion (D₁); ■, ^ΔCavitated enamel lesion (D₂₊); ■, [#]Filled (F).

For dental examination of both pit and fissure surfaces and smooth surfaces, the study criteria clearly differentiated among various stages of developing caries lesions. Radiographs were not used and the criteria used by the study did not distinguish between D₂ (enamel) and D₃ (dentin) lesions, so they were combined as D₂₊. Cavitated-level caries status included D₂₊, M, and F surfaces (D₂₊MFS) [28, 30], where F were filled with no recurrent or separate decay and M were missing due to caries. In brief, D₁ lesions were characterized by chalky-white appearance or staining in the pits and fissures without loss of enamel, while “questionable” (D₀) lesions were recorded when lesions were present, but did not meet the criteria for D₁ lesions, such as in the case of arrested lesions.

This study assessed the prevalence of different surface lesions, as well as transitions of non-cavitated (D₁) lesions, at both the participant-level and surface-level. At the participant-level, prevalence was defined as the percentage of participants with at least one non-cavitated (D₁) lesion. At the surface-level, it was the percentage of tooth surfaces/zones with D₁ lesions. Tooth

surfaces (buccal, lingual, distal, mesial, and occlusal) were scored separately and, for the maxillary first and second molars, the occluso-mesial and occluso-distal pits were recorded separately as occlusal zones. The transitions were reported into the six categories as described above (S, D₀, remained D₁, D₂₊, F, or M), as well as three broad categories (progression, no change, and regression). “Progression” of non-cavitated caries lesions was defined as a change from D₁ to D₂₊, F, or M. The surfaces that maintained their status as a D₁, that is, neither showed progression nor regression/arrest, were categorized as “no change.” Finally, the “regression” category represented lesions that reverted from D₁ to S or D₀. These transitions were determined separately for the following five time periods: 9–13 years, 13–17 years, 17–23 years, 9–23 years (for early erupting teeth only), and 13–23 years (for late-erupting teeth only). Prior to each of the four exams, examiners were retrained and recalibrated, and during each exam period they conducted duplicate exams on a subset of participants to assess inter-examiner agreement. Kappa scores for inter-examiner agreement accounting for multiple

examiners were calculated. Data analysis for descriptive frequencies and kappa statistics were calculated using R software, version 3.6.2 [31].

RESULTS

The study sample of 629 participants at age 9 had relatively equal sex distribution (49% males and 51% females). It was predominantly white and non-Hispanic (95%). The sociodemographic data collected in 2007 showed that the majority of these participants' mothers (71%) had some education beyond high school (17% had some college education, 23% a 2-year-college degree or completed technical/beauty school, and 31% a 4-year college degree). Over the study period, about 52%–55% of the samples were high SES, 32%–35% were middle SES, and 12%–13% were low SES.

Inter-examiner agreement caries kappa scores at the person- and permanent tooth surface-levels were 0.74 and 0.47 at age 9, 0.74 and 0.70 at age 13, 0.53 and 0.74 at age 17, and 0.74 and 0.83 at age 23 years, respectively.

The sample sizes for each cross-sectional assessment, along with the numbers and percentages of participants with at least one D₁ lesion, are shown in Table 2. At age 9, 23% of the 629 participants ($n = 146$) had at least one D₁ lesion; at age 13, 38% ($n = 209$) of 549 had at least one D₁; at age 17, 60% of 464 ($n = 279$) had at least one D₁; and at age 23, 45% ($n = 153$) of 342 had at least one D₁ lesion.

Table 3 shows the prevalence of zone/surface-level lesions by caries category at each examination. The numbers (and percentages of all sites examined) with D₁ were

307 (0.9%), 607 (0.9%), 1856 (3.1%), and 926 (2.1%) at ages 9, 13, 17, and 23 years, respectively. At all ages, the highest prevalence of D₁ lesions was recorded on molars (95% at age 9, 62% at age 13, 58% at age 17 and 63% at age 23) and buccolingual surfaces (54% at age 9, 58% at age 13, 67% at age 17, and 59% at age 23).

Table 4 shows the frequency distributions of D₁ transitions for the five time periods. Of 222 D₁ lesions at age 9 by age 13, 60.8% ($n = 135$) regressed to sound, 11.3% ($n = 25$) regressed to questionable (D₀), 15.3% ($n = 34$) remained non-cavitated (D₁), 1.8% ($n = 4$) progressed to cavitation (D₂₊), and 10.8% ($n = 24$) were filled (F). During this time period, fillings accounted for 85.7% of progression, while cavitation contributed 14.3%.

From age 13 to 17, 34.4% of 459 D₁s ($n = 158$) regressed to S and 20.0% ($n = 92$) regressed to D₀, 28.3% ($n = 130$) remained D₁, 3.7% ($n = 17$) progressed to D₂, and 13.5% ($n = 62$) progressed to F. Fillings accounted for 78.6% of D₁ lesion progression, while cavitation contributed 21.5%.

From age 17 to 23, 51.6% of 1125 D₁ lesions ($n = 580$) transitioned to S and 19.6% ($n = 221$) to D₀, 17.6% ($n = 199$) remained D₁, 3.7% ($n = 42$) became D₂₊, 7.0% ($n = 79$) became F, and 0.4% ($n = 4$) were missing (M) due to caries. Most progression was fillings (63.2%), with 33.6% untreated decay (D₂₊) and 3.2% missing (M) (Table 3).

For the early erupting teeth from age 9 to 23, there was a higher rate of progression from the D₁ category during this longer follow-up period of 14 years at about 31.9% (with 97.7% of this progression being fillings) and a lower percentage of lesions regressing, 60.9% (with 81.3% transitioning to S and 18.6% to D₀).

For the late-erupting teeth followed for 10 years from age 13 to 23, results were similar. The rate of progression was 28.3% (with 92.3% of progression being fillings) and 55.0% regressed (with 71.1% transitioning to S and 28.9% to D₀).

The rates of caries progression for 17–23 years interval among D₁ lesions were highest on the occlusal surfaces (i.e., 25% on occlusal (O) versus 18% on mesiodistal (M/D) and 5% on buccolingual (B/L) surfaces) and molar teeth (14% on molars versus 7% on non-molars), while the rates of regression were highest on the buccal and lingual surfaces (i.e., 76% on B/L versus 67% on M/D and

TABLE 2 Frequency distributions of participants examined and those with at least one D₁ lesion at each examination (ages 9, 13, 17, and 23 years)

Age (years)	Total sample size	At least 1 D ₁ lesion (sample size)	
		Number	Percentage
Age 9	629	146	23.2%
Age 13	549	209	38.1%
Age 17	464	279	60.1%
Age 23	342	153	44.7%

TABLE 3 Frequency distributions of zone/surface-level lesions by caries categories at each examination (age 9, 13, 17, and 23 years)

Caries categories	Age 9 (n, %)	Age 13 (n, %)	Age 17 (n, %)	Age 23 (n, %)
D ₀	77 (0.2%)	482 (0.7%)	1739 (2.9%)	1125 (2.6%)
D ₁	307 (0.9%)	607 (0.9%)	1856 (3.1%)	926 (2.1%)
D ₂	67 (0.2%)	60 (0.1%)	149 (0.3%)	188 (0.4%)
F	261 (0.7%)	605 (0.9%)	1465 (2.5%)	1578 (3.6%)
S	35,013 (98.0%)	66,809 (97.4%)	54,593 (91.3%)	40,132 (91.2%)
M	0 (0%)	0 (0%)	0 (0%)	35 (0.1%)
Total	35,726 (100%)	68,563 (100%)	59,802 (100%)	43,984 (100%)

TABLE 4 Frequency distribution of D₁ lesion transitions at follow-up to regression (sound (S), arrested or questionable D₀), no change (D₁), or progression (cavitation (D₂), filled (F), or missing due to caries (M))

Frequency distribution of D ₁ lesion transitions	Regression		No change	Progression			Total
	S	D ₀	D ₁	D ₂	F	M	
Status at age 13 of D ₁ lesions at age 9	135 (60.8%)	25 (11.3%)	34 (15.3%)	4 (1.8%)	24 (10.8%)	–	222
Status at age 17 of D ₁ lesions at age 13	158 (34.4%)	92 (20.0%)	130 (28.3%)	17 (3.7%)	62 (13.5%)	–	459
Status at age 23 of D ₁ lesions at age 17	580 (51.6%)	221 (19.6%)	199 (17.6%)	42 (3.7%)	79 (7.0%)	4 (0.4%)	1,125
Status at age 23 of D ₁ lesions at age 9 ^a	70 (49.6%)	16 (11.3%)	10 (7.1%)	1 (0.7%)	44 (31.2%)	–	141
Status at age 23 of D ₁ lesions at age 13 ^b	54 (39.1%)	22 (15.9%)	23 (16.6%)	2 (1.5%)	36 (26.1%)	1 (0.7%)	138

^aEarly erupting teeth only.

^bLate-erupting teeth only.

59% on O) and teeth except molars (i.e., 77% on non-molars versus 68% on molars).

Overall, for all incidence intervals, only a small percentage progressed to decayed, missing, or filled. Specifically, the percentages of no-change were 15%–28%, percentages of regression were 34%–61%, and percentages of progression to D₂ were quite low, from 1.8% to 3.7% (i.e., excluding progression reported as fillings).

DISCUSSION

The primary goals of this article were to report the prevalence of non-cavitated carious lesions (at ages 9, 13, 17, and 23) and progression of non-cavitated carious lesions longitudinally [from ages 9–13, 13–17, 17–23, 9–23 (early erupting teeth), and 13–23 (late-erupting teeth)] for participants in the IFS.

The percentages of participants with at least one non-cavitated D₁ lesion were 23% at age 9, 38% at age 13, 60% at age 17, and 45% at age 23, respectively. The substantially higher rate at age 17 could be due in part to the higher likelihood of D₁ lesions surrounding orthodontic brackets or recently removed brackets. The lower rate at age 23 may be due to a multi-year remineralization period after removal of brackets. Another partial explanation could be the dietary choices at these ages, with higher amounts of processed food and junk food being consumed in adolescence before the age 17 exams and reductions from age 17 to 23.

There were no studies found for comparison of these person-level results, since all studies reported prevalence at the tooth and/or surface level only. Therefore, the following discussion is for prevalence and results at the surface-level.

The prevalence of non-cavitated D₁ lesions at the zone/surface-level was ~1% at ages 9 and 13, 3% at age 17, and 2% at age 23. The highest zone/surface-level D₁ rate was at age 17 and the rate at age 23 also was much higher than at ages 9 and 13. These rates were somewhat lower when compared to other studies. Nuca et al. [15] reported 18% of sites (*n* = 1765) to be incipient white

spot lesions at age 12–13 years based on combined clinical and radiographic examinations. Acevedo et al. [14] reported that, based on clinical examinations, approximately 1.73% (*n* = 3117) were non-cavitated lesions out of 180,074 surfaces evaluated in schoolchildren 11–13 years of age. Zenkner et al. [16] reported that, out of 1152 total sites examined at baseline (mean 13.3 years), 47% (*n* = 550) were inactive non-cavitated lesions. At follow-up (mean age 17.6 years), 60% (*n* = 692) of the 1152 sites had inactive non-cavitated lesions and 3% (*n* = 36) had active non-cavitated lesions. The higher prevalences of D₁ lesions in other studies were probably due to methodological differences and their samples having lower SES than ours.

This study found that the rates of D₁ lesion progression for different age intervals varied from 11% to 32%, with higher rates for longer follow-up periods: 13% for 9–13, 19% for 13–17, 11% for 17–23, 32% for 9–23 (early erupting teeth), and 29% for 13–23 (late-erupting teeth). However, the rates of regression were relatively high for all age intervals as compared to no change and progression. The rates of regression for different age-intervals varied from 54% to 72%: 72% for 9–13, 54% for 13–17, 72% for 17–23, 61% for 9–23 (early erupting), and 55% for 13–23 (late-erupting). In summary, rates of D₁ lesion regression were always higher than rates of progression, but higher rates of progression were recorded for longer follow-up periods.

Most lesion progression was the result of new fillings, as overall progression ranged 11%–32%, but excluding fillings (i.e., including only D₁ to D₂₊), progression rates were less than 4%. This shows that the overall rates of progression (due to high rates of fillings alone i.e., 7%–28%) reported by this study might over-estimate the progression of true disease. Since the restorations were also counted as progression, but decisions to restore were made based on the subjective judgments of clinicians, there are no study data available for these to identify the “true need” for a filling. Moreover, the sample with relatively high SES having high rates of treatment could be another possible explanation of high rates of filling. Our results for progression of D₁ lesions are generally consistent with the patterns of the few other studies [18, 20],

with differences probably due to study differences in ages, duration of follow-up, and assessment methodologies [18, 20]. Specifically, Mejare et al. [18] reported radiographic progression of 19% of approximal enamel lesions from age 12 to 21 years. Martignon et al. [19] reported that 50% of lesions located in the outer half of enamel at baseline (age 20 years) progressed to cavitation and 10% were filled at follow-up (age 26 years). Hintze [20] reported progression to cavitation of 22% and to filling of 8% of the enamel lesions (from baseline mean age 14.5 years to follow-up 17.6 years). Zenkner et al. [21] reported that, out of 539 inactive enamel lesions at baseline, 3% progressed to active lesions and 1% were filled after 1 year (baseline median age 13) based on thorough clinical examinations [16, 21].

Our study recorded “no change” over 6 years from age 17 to 23 in 18% ($n = 203$) of the surfaces with a D_1 lesion at baseline, that is, 18% of lesions remained non-cavitated from baseline to follow-up after 6 years. These results are consistent with Hintze [20] who reported no change in 13% of the radiographic approximal enamel lesions (from baseline mean age 14.5 to follow-up mean age 17.6). However, Zenkner et al. [21] reported very high rates for the “no change” category of D_1 lesions in school-children (median age 13.3 years) after one-year of follow-up, with 96% ($n = 518$) of 539 inactive enamel lesions that remained the same. Their high rates of “no change,” much higher than our study, could be explained by the short follow-up duration of 1 year, since the same cohort was reported to have 64% of “no change” when followed up for 4–5 years (mean age 17.9 years) [16, 21].

Finally, the rates of regression of D_1 lesions were very high, ranging from 54% to 72%. These results could be explained by the high socioeconomic status of our sample, as well as the high rate of exposure to fluoridated water and almost universal use of fluoridated dentifrice. Moreover, some of these could be attributed to reversal of diagnoses owing to different examiners at different examinations. Only a few studies in the literature have reported on regression of D_1 lesions, and the results were highly variable. Warren et al. [17] reported high rates of regression to sound/arrested or no change for D_1 lesions (63%) from baseline (mean age 5.2 years) to follow-up (mean age 9.2 years) among IFS participants. David et al. [22] reported that 25% of lesions (D_{1-5} MFS) had either a reversal or no increment in caries from age 12 to 18. Martignon et al. [19] reported regression in 6% of 70 enamel lesions from age 20 to 26 years. Chestnutt et al. [23] reported extremely low clinical reversals, with a reversal rate of <1%, from decayed (DMFS) to sound after 3 years (baseline 12.5 years). Lith et al. [24] reported on regression from age 6 to 20, with 4% of all reversals from dentin to enamel and 95% within enamel. Zenkner et al. [16] found that 10% of inactive non-cavitated lesions became sound and 23% of inactive cavitated lesions became inactive non-cavitated lesions after 4–5 years of follow-up.

This study had several strengths, including a moderately large sample with much data concerning a cohort followed for 23 years in the IFS. It is the only study we are aware of that assessed the progression of non-cavitated caries lesions longitudinally from age 9 to 23. An additional strength of the study was the assessment of the regression of non-cavitated caries lesions, since most of the published literature related to caries outcomes has not explored caries regression. Lastly, the examiners in the IFS had formal training, calibration, and recalibration exercises and inter-examiner reliability was favorable.

There also were several limitations to the study. First, the IFS study participants were a relatively homogeneous group, with the large majority being non-Hispanic Whites (95%) and from relatively higher SES families (35% from middle and 53% from high SES groups) at age 9, with mostly relatively low disease levels. Therefore, the participants are not representative of the general U.S. population, limiting generalizability. Furthermore, there was loss of subjects to follow-up over time. Second, a large percentage of non-cavitated sites with caries progression presented as fillings (not frank cavitation), so the study is not reporting on the natural history of caries progression and is instead largely reflecting dentists' treatment. Third, there were no study data included on dentists' diagnostic practices or decision-making concerning dentists' preventive, remineralization, or restorative approaches. This is important, as non-cavitated caries lesions can sometimes be challenging to differentiate from questionable lesions and stained tooth surfaces, as well as from dentinal caries without enamel cavitation. Thus, dentists may have restored some non-cavitated D_1 lesions that had not actually progressed. Thus, there is possible over-estimation of caries progression results due to the relatively high number of fillings done (based on subjective assessment of the dentists). Fourth, the dental exams were only done at the beginning and end of the 4- to 6-year study periods, without any examinations in between, so we cannot assess the timing of change in the D_1 lesions within the 4- to 6-year intervals. Also, the time between exams varied slightly from person to person, so the incidence period also varied for each person. Fifth, the IFS caries assessment criteria did not distinguish between cavitated enamel and cavitated dentin lesions, limiting reporting of the level of progression to a cavitated outcome. The study did not use radiographs to assess caries; therefore, it is likely that the caries outcomes were underestimated, particularly on the proximal surfaces. Sixth, the examiners varied at different examinations, with only one examiner present during all five examinations. However, in order to reduce the impact of inter-examiner differences and to ensure consistency of results, at least two examiners from each wave continued to be part of each subsequent examination. Lastly, the study did not account for information on important changes during adolescence and young adulthood, such as changes in dietary choices, orthodontic treatment, and

other lifestyle habits. All of these might have affected the results and could partially explain why there was a higher rate of D₁ lesions at age 17 compared with the other ages.

CONCLUSION AND FUTURE RECOMMENDATIONS

This study reported that non-cavitated lesions were more prevalent at age 17 than at ages 9, 13, and 23. For all time periods, there were higher rates of regression and no change at follow-up as compared to only a fraction of lesions that showed progression to a cavitated state. The high rates of regression, even after 4 to 14 years of follow-up, further highlight the importance of the non-cavitated stage. It reinforces that the non-cavitated lesions can be reversed and that the provider should not assume surgical intervention to be the treatment of choice for incipient lesions.

Future studies should include non-cavitated lesions in their analyses and report them separately from cavitated lesions. This will add useful information to the literature and hopefully encourage enhanced use of remineralization and preventive dentistry approaches and reductions in the unnecessary restoration of non-cavitated lesions.

ACKNOWLEDGMENTS

The funding and support for the Iowa Fluoride Study was provided by U.S. National Institute of Health grants R03-DE023784, R01-DE12101, R01-DE09551, R56-DE012101, UL1-RR024979, UL1-TR000442, UL1-TR001013, and M01-RR00059, the Roy J. Carver Charitable Trust, and the Delta Dental of Iowa Foundation.

CONFLICT OF INTEREST

The authors declare that they have no competing interests to declare.

ORCID

Mahrukh Zafar  <https://orcid.org/0000-0002-3493-662X>
 Steven M. Levy  <https://orcid.org/0000-0001-9150-4199>
 John J. Warren  <https://orcid.org/0000-0002-0090-9973>

REFERENCES

- GBD 2016 Disease and Injury Incidence and Prevalence Collaborators. Global, regional, and national incidence, prevalence, and years lived with disability for 328 diseases and injuries for 195 countries, 1990–2016: a systematic analysis for the global burden of disease study 2016. *Lancet*. 2017;390:1211–59.
- Summitt JB. Conservative cavity preparations. *Dent Clin*. 2002;46(2):171–84.
- Murdoch-Kinch CA, Mclean ME. Minimally invasive dentistry. *J Am Dent Assoc*. 2003;134(1):87–95.
- Tyas MJ, Anusavice KJ, Frencken JE, Mount GJ. Minimal intervention dentistry—a review* FDI Commission project 1–97. *Int Dent J*. 2000;50(1):1–12.
- Kumar A, Yadav N, Singh S, Chauhan N. Minimally invasive (endoscopic-computer assisted) surgery: technique and review. *Ann Maxillofac Surg*. 2016;6(2):159–64.
- Nassar HM. Dental caries preventive considerations: awareness of undergraduate dental students. *Dent J*. 2020;8(2):31.
- Martignon S, Gomez J, Tellez M, Ruiz JA, Marin LM, Rangel MC. Current cariology education in dental schools in Spanish-speaking Latin American countries. *J Dent Educ*. 2013;77(10):1330–7.
- Ekstrand KR, Ricketts DN, Kidd EA. Occlusal caries: pathology, diagnosis and logical management. *Dent Update*. 2001;28(8):380–7.
- Featherstone JD. The caries balance: the basis for caries management by risk assessment. *Oral Health Prev Dent*. 2004;2:259–64.
- Kidd EAM. The diagnosis and management of the 'early' carious lesion in permanent teeth. *Dent Update*. 1984;11(2):69–70, 72–4, 76–8.
- Machiulskiene V, Campus G, Carvalho JC, Dige I, Ekstrand KR, Jablonski-Momeni A, et al. Terminology of dental caries and dental caries management: consensus report of a workshop organized by ORCA and Cariology research group of IADR. *Caries Res*. 2020;54(1):7–14.
- Amarante E, Raadal M, Espelid I. Impact of diagnostic criteria on the prevalence of dental caries in Norwegian children aged 5, 12 and 18 years. *Community Dent Oral Epidemiol*. 1998;26(2):87–94.
- Gomez J. Detection and diagnosis of the early caries lesion. *BMC Oral Health*. 2015;15(1):S3.
- Acevedo AM, Montero M, Machado C, Saez I, Rojas-Sanchez F, Kleinberg I. Dental caries experience in school children and the impact of non-cavitated lesions on the caries index. *Acta Odontol Latinoam*. 2013;26(1):8–14.
- Nuca C, Aniarizi C, Gaita A, Diaconu C. Salivary fluoride concentration after professional topical fluoride applications. *OHDMSC*. 2003;2(4):38–41.
- Zenkner JEA, Nora AD, Alves LS, Carvalho J, Wagner MB, Maltz M. Long-term follow-up of inactive occlusal caries lesions: 4-5-year results. *Clin Oral Invest*. 2019;23:847–53.
- Warren JJ, Levy SM, Broffitt B, Kanellis MJ. Longitudinal study of non-cavitated carious lesion progression in the primary dentition. *J Public Health Dent*. 2006;66(2):83–7.
- Mejare I, Stenlund H, Kallestal C. Incidence and progression of approximal caries from 11 to 22 years of age in Sweden: a prospective radiographic study. *Caries Res*. 1999;33(2):93–100.
- Martignon S, Chavarria N, Ekstrand KR. Caries status and proximal lesion behaviour during a 6-year period in young adult Danes: an epidemiological investigation. *Clin Oral Invest*. 2010;14:383–90.
- Hintze H. Caries behaviour in Danish teenagers: a longitudinal radiographic study. *Int J Paediatr Dent*. 1997;7(4):227–34.
- Zenkner JEA, Carvalho JC, Wagner MB, Alves LS, de Oliveira RS, Rocha RO, et al. One-year evaluation of inactive occlusal enamel lesions in children and adolescents. *Clin Oral Invest*. 2016;20:133–9.
- David J, Raadal M, Wang NJ, Strand GV. Caries increment and prediction from 12 to 18 years of age: a follow-up study. *Eur Arch Paediatr Dent*. 2006;1(1):31–7.
- Chestnutt IG, Schafer F, Jacobson APM, Stephen KW. Incremental susceptibility of individual tooth surfaces to dental caries in Scottish adolescents. *Dent Oral Epidemiol*. 1996;24(1):11–6.
- Lith A, Lindstrand C, Grondahl HG. Caries development in a young population managed by a restrictive attitude to radiography and operative intervention: II. A study at the surface level. *Dentomaxillofac Rad*. 2002;31(4):232–9.
- Ismail AI, Brodeur JM, Gagnon P, Payette M, Picard D, Hamalian T, et al. Prevalence of non-cavitated and cavitated carious lesions in a random sample of 7-9-year-old schoolchildren in

- Montreal, Quebec. *Community Dent Oral Epidemiol.* 1992;20(5): 250–5.
26. Ferreira Zandoná A, Santiago E, Eckert GJ, Katz BP, Pereira de Oliveira S, Capin OR, et al. The natural history of dental caries lesions: a 4-year observational study. *J Dent Res.* 2012;91(9): 841–6.
27. Levy SM, Warren JJ, Davis CS, Kirchner HL, Kanellis MJ, Wefel JS. Patterns of fluoride intake from birth to 36 months. *J Public Health Dent.* 2001;61(2):70–7.
28. Chankanka O, Cavanaugh JE, Levy SM, Marshall TA, Warren JJ, Broffitt B, et al. Longitudinal associations between children's dental caries and risk factors. *J Public Health Dent.* 2011;71(4):289–300.
29. Pitts NB. Diagnostic tools and measurements—impact on appropriate care. *Community Dent Oral Epidemiol.* 1997;1:24–35.
30. Broffitt B, Levy SM, Warren JJ, Cavanaugh JE. Factors associated with surface-level caries incidence in children aged 9 to 13: the Iowa Fluoride Study. *J Public Health Dent.* 2013;73(4): 304–10.
31. R Core Team. R: a language and environment for statistical computing. Vienna, Austria: R Foundation for Statistical Computing; 2019. Available from: <https://www.R-project.org/>. Accessed 20 Nov 2020.

How to cite this article: Zafar M, Levy SM, Warren JJ, Xie XJ, Kolker J, Pendleton C. Prevalence of non-cavitated lesions and progression, regression, and no change from age 9 to 23 years. *J Public Health Dent.* 2022;82(3): 313–20. <https://doi.org/10.1111/jphd.12538>