V.S. Yu¹*, L.W. Khin², C.S. Hsu¹,
R. Yee¹, and H.H. Messer³

¹Faculty of Dentistry, National University of Singapore, Singapore; ²Dean's Office Medicine Investigational Medicine Unit, Department of Surgery, Yong Loo Lin School of Medicine, National University of Singapore, Singapore; and ³Melbourne Dental School, University of Melbourne, Melbourne, Australia; *corresponding author, denyshv@nus.edu.sg

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ABSTRACT

Persistent apical periodontitis related to a nonvital tooth that does not resolve following root canal treatment may be compatible with health and may not require further intervention. This research aimed to develop a Deterioration Risk Score (DRS) to differentiate lesions requiring further intervention from lesions likely to be compatible with health. In this cross-sectional study, patient records (2003-2008) were screened for root-filled teeth with periapical radiolucency visible on periapical radiographs taken at treatment and at recruitment at least 4 yr later. The final sample consisted of 228 lesions in 182 patients. Potential demographic and treatment risk factors were screened against 3 categorical outcomes (improved/unchanged/deteriorated), and a multivariate independent multinomial probit regression model was built. A 5-level DRS was constructed by summing values of adjusted regression coefficients in the model, based on predicted probabilities of deterioration. Most lesions (127, 55.7%) had improved over time, while 32 (14.0%) remained unchanged, and 69 (30.3%) had deteriorated. Significant predictors of deterioration were as follows: time since treatment (relative risk [RR]: 1.11, 95% confidence interval [CI]: 1.01-1.22, $p = .030$, rounded beta value = 1, for every year increase after 4 yr), current pain (RR: 3.79, 95% CI: 1.48-9.70, $p = .005$, rounded beta value = 13), sinus tract present (RR: 4.13, 95% CI: 1.11-15.29, $p = .034$, rounded beta value = 14), and lesion size (RR: 7.20, 95% CI: 3.70-14.02, $p < .001$, rounded beta value = 20). Persistent apical periodontitis with DRS <15 represented very low risk; 15-20, low risk; 21-30, moderate risk; 31-40, high risk; and >40, very high risk. DRS could help the clinician identify persistent apical periodontitis at low risk for deterioration, and it would not require intervention. When validated, this tool could reduce the risk of overtreatment and contribute toward targeted care and better efficiency in the timely management of disease.

KEY WORDS: decision making, periapical periodontitis, endodontics, root canal therapy, probability, regression analysis.

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Risk Score Algorithm for Treatment of Persistent Apical Periodontitis

INTRODUCTION

Persistent apical periodontitis (AP) is defined as an apical lesion related to a nonvital tooth that does not resolve following root canal treatment, most commonly due to persistence of virulent pathogens that survive biomechanical debridement (Byström and Sundqvist, 1981; Sjögren and Sundqvist, 1987; Nair, 2006; Siqueira and Rôças, 2009). It can remain asymptomatic or progress to symptomatic AP, which affects daily living and prompts patients to seek treatment (Iqbal *et al.*, 2009; Yu *et al.*, 2012a; Grönholm *et al.*, 2013). Although accepted quality guidelines (European Society of Endodontology, 2006) recommend intervention (retreatment or apical surgery) for AP persisting for 4 yr or more, asymptomatic persistent AP may be compatible with health if the tooth remains functional (Friedman and Mor, 2004) and may even be capable of further healing over prolonged periods beyond 4 yr (Molven *et al.*, 2002; Fristad *et al.*, 2004; Yu *et al.*, 2012b).

When asymptomatic AP is associated with a technically inadequate root filling, dentists are more inclined toward further intervention (Morgental *et al.*, 2012); otherwise, many dentists question the benefit of intervention (Kvist and Reit, 2002) and disagree on the need for, as well as the mode of, further treatment (Reit and Gröndahl, 1984; Rawski *et al.*, 2003). Although a number of tooth-level clinical parameters, such as periodontal status and quality of root fillings, are commonly considered in decision making (von Arx *et al.*, 2014), the decision of whether to intervene is largely subjective. Integration of clinical findings and the assessment of prognosis are strongly influenced by personal observation and experience (Reit and Kvist, 1998; Azarpazhooh *et al.*, 2013).

In view of the challenges involved, a risk score based on routine clinical and radiographic information to predict progression of persistent lesions will aid objective decision making regarding further intervention. Various scoring systems have been developed to identify individuals at risk for a range of diseases. Examples include the Laboratory Risk Indicator for Necrotizing Fasciitis score to differentiate necrotizing fasciitis from other severe soft tissue infections for timely and predictable treatment in the emergency room (Wong *et al.*, 2004), as well as risk scores to identify women at risk for adverse pregnancy outcome (Burstyn, 2010), men and women at risk for advanced periodontitis (Gursoy *et al.*, 2011), and children at risk for childhood caries (Gao *et al.*, 2010) for targeted allocation of resources. However, none have been developed to guide clinicians in the decision to intervene or not for asymptomatic persistent AP.

Comprehensive predictive studies investigating factors associated with healing/nonhealing after root canal treatment have been published (Friedman *et al.*, 2003; Farzaneh *et al.*, 2004; Ørstavik *et al.*, 2004; Marquis *et al.*, 2006; de Chevigny *et al.*, 2008; Ng *et al.*, 2011a, 2011b) and are not addressed in this article. The aim of this study was to identify potential predictors for persistent lesions to improve (**I**), remain unchanged (**U**), or deteriorate (**D**) in a cohort of patients with AP that had persisted at least 4 yr and to develop a risk score to differentiate lesions requiring further intervention from lesions likely to be compatible with health, for cost-effective and evidence-based treatment planning.

MATERIALS & METHODS

Selection of Cases

This study conformed to STROBE guidelines for cross-sectional human observational study. Ethical approval was obtained from the Institutional Review Board (NUS-IRB 10-171), and written informed consent was obtained from all patients taking part in the study. The selection of cases, data collection, and scoring of lesion progression included in the final cohort have been described in detail (Yu *et al.*, 2012a, 2012b). In brief, patients who received root canal treatment for AP from general dentists, endodontic specialists, undergraduate dental students, and endodontic residents at a university-based dental center from 2003 to 2008 whose AP had not resolved at short-term recall were screened for AP at least 4 yr later. At the time of screening, treatments performed before 2003 in the same dental center were also included if inclusion and exclusion criteria were fulfilled. Inclusion criteria included primary or secondary (nonsurgical) root canal treatment, with radiographs clearly showing the entire lesion before treatment and presenting with a widened periodontal ligament space with discontinuity of the lamina dura or a larger radiolucency associated with any root of a root-filled tooth at least 4 yr after treatment. Cases without good-quality treatment radiographs and teeth with periodontal bone loss, vertical root fracture, or a history of traumatic luxation injuries or surgical endodontic procedures were excluded. The patients recruited were at least 21 yr old and were healthy or had controlled medical conditions.

For recruitment, 2 calibrated endodontists independently evaluated treatment and reviewed radiographs using 2 × magnification against a fluorescent x-ray viewing box in a darkened room. Radiographs were taken with the long-cone technique per the long-cone positioning device (Dentsply Rinn XCP, Elgin, IL). Disagreements were resolved by discussion and consensus. The final sample of 228 lesions in 182 patients included 151 persistent lesions in 114 patients previously studied for risk of painful exacerbation and characteristics of lesion progression (Yu *et al.*, 2012b).

Data Collection

An interviewer-administered structured questionnaire collected patient information on post-treatment experience of pain and flare-up, as well as the self-reported experience of a swelling or

sinus tract. Dental records corroborated patient recollections of flare-ups and provided historical and demographic data. Clinical and periapical radiographic examination provided details of clinical signs and symptoms; periapical lesion characteristics including size, border, and location; and type and quality of endodontic treatment and postendodontic restoration.

Scoring of Lesion Progression

By reading past treatment and current review periapical radiographs (all taken with a long-cone positioning device) under similar conditions of a 2× magnification against a fluorescent x-ray viewing box in a darkened room, 2 calibrated endodontists independently scored initial and persistent lesion sizes based on 4 categories—(1) <2 mm, (2) 2–4.9 mm, (3) 5–9.9 mm, and (4) ≥10 mm—in the largest dimension and scored lesion progression as 3 categorical outcomes: **I**, lesion improved (decreased in size); **U**, lesion remained unchanged (no change in size); and **D**, lesion deteriorated (increased in size; Yu *et al.*, 2012b). The calibration process was previously reported; weighted Cohen kappa score for interexaminer reliability was 0.716, and weighted Cohen kappa scores for intraexaminer reliability were 0.638 and 0.821 (Yu *et al.*, 2012a). Any disagreements were resolved by discussion and consensus before reporting the final score.

Model Building and Risk Score Algorithm

The independent multinomial probit stepwise regression procedure (Stata 12.1, StataCorp LP, USA) was selected since outcome categories were discrete variables with more than 2 outcome categories that did not have a natural ordering, and outcome categories were identified per radiographic evaluation of lesion progression. Potential demographic and treatment risk factors were screened against the 3 categorical outcomes (**I**, **U**, **D**) according to univariate independent multinomial probit regression. Regression coefficients with standard errors adjusted for clustering were estimated for both **D** and **U**, with **I** as base outcome. The final fitted model was constructed on the basis of multivariate independent multinomial probit regression with backward procedures where variables with $p \leq .1$ in the univariate analysis and variables with clinical importance were considered for inclusion, to not miss any potentially important predictors. To choose among competing models, the preferred final fitted model was based on the log likelihood chi-square value. The effect size on clinical endpoints was quantified according to the relative risk (RR) estimate and its associated 95% confidence interval (CI).

To assess risk of deterioration for treatment prescription, a risk score algorithm was constructed by rounding up values of regression coefficients in the final fitted model, based on predicted probabilities that were calculated *via* a postestimation command. A 5-level Deterioration Risk Score (DRS) was constructed—very low risk, ≤20% predicted probability; low risk, 21%–40%; moderate risk, 41%–60%; high risk, 61%–80%; very high risk, >80%—and a decision tree described.

Model and risk score performance was evaluated per receiver operating characteristic analysis. Sensitivity, specificity, and positive and negative predictive values were calculated accord-

Table 1. Screening Model of Patient Demographics and Potential Clinical and Radiographic Risk Factors for Lesion Remaining Unchanged and Deteriorating^a

Variable	Improved	Unchanged	<i>p</i> Value ^b	Deteriorated	<i>p</i> Value ^b	Total
Patient and treatment factors						
Sex						
Male	62 (48.8)	16 (50.0)	.859	38 (55.1)	.424	116 (50.9)
Female	65 (51.2)	16 (50.0)		31 (44.9)		112 (49.1)
Race						
Chinese	110 (86.6)	28 (87.5)	.872	61 (88.4)	.706	199 (87.3)
Non-Chinese	17 (13.4)	4 (12.5)		8 (11.6)		29 (12.7)
Tooth type						
Anteriors + premolars	72 (56.7)	14 (43.8)	.14	24 (34.8)	.005	110 (48.2)
Molars	55 (43.3)	18 (56.2)		45 (65.2)		118 (51.8)
Restoration type						
Crown	93 (73.2)	25 (78.1)	.49	59 (85.5)	.05	177 (77.6)
Others	34 (26.8)	7 (21.9)		10 (14.5)		51 (22.4)
Intracanal post present						
No	106 (83.5)	27 (84.4)	.943	55 (79.7)	.496	188 (82.5)
Yes	21 (16.5)	5 (15.6)		14 (20.3)		40 (17.5)
Root-filling length						
Ideal	107 (84.3)	19 (59.4)	.004	56 (81.2)	.532	182 (79.8)
Not ideal	20 (15.7)	13 (40.6)		13 (18.8)		46 (20.2)
Root-filling density						
Dense and tapered	94 (74.0)	21 (65.6)	.328	43 (62.3)	.107	158 (69.3)
Voids present / poorly condensed	33 (26.0)	11 (34.4)		26 (37.7)		70 (30.7)
Iatrogenic error						
No	112 (88.2)	27 (84.4)	.458	55 (79.7)	.122	194 (85.1)
Yes	15 (11.8)	5 (15.6)		14 (20.3)		34 (14.9)
Self-reported experience						
Pre-treatment pain						
No	85 (66.9)	20 (62.5)	.56	38 (55.1)	.129	143 (62.7)
Yes	42 (33.1)	12 (37.5)		31 (44.9)		85 (37.3)
Post-treatment pain						
No	92 (74.8)	26 (81.3)	.592	38 (57.6)	.034	156 (70.6)
Yes	31 (25.2)	6 (18.7)		28 (42.4)		65 (29.4)
Post-treatment sinus tract						
No	123 (96.9)	29 (90.6)	.124	55 (79.7)	.002	207 (90.8)
Yes	4 (3.1)	3 (9.4)		14 (20.3)		21 (9.2)
Past dental records						
History flare-up						
No	124 (97.6)	31 (96.9)	.654	60 (87.0)	.007	215 (94.3)
Yes	3 (2.4)	1 (3.1)		9 (13.0)		13 (5.7)
Care provider						
Endodontist	87 (68.5)	26 (81.3)	.168	49 (71.0)	.701	162 (71.1)
Others	40 (31.5)	6 (18.7)		20 (29.0)		66 (28.9)
Current findings						
Age, yr						
20-40	26 (20.5)	5 (15.6)	.434	12 (17.4)	.416	43 (18.9)
41-65	80 (63.0)	23 (71.9)		51 (73.9)		154 (67.5)
>65	21 (16.5)	4 (12.5)		6 (8.7)		31 (13.6)
Restoration quality						
Adequate	94 (74.0)	27 (84.4)	.244	49 (71.0)	.675	170 (74.6)
Deficient margins	33 (26.0)	5 (15.6)		20 (29.0)		58 (25.4)
Tooth is painful now						
No	123 (96.9)	28 (87.5)	.036	59 (85.5)	.005	210 (92.1)
Yes	4 (3.1)	4 (12.5)		10 (14.5)		18 (7.9)

(continued)

Table 1. (continued)

Variable	Improved	Unchanged	<i>p</i> Value ^b	Deteriorated	<i>p</i> Value ^b	Total
Percussion pain						
No	92 (72.4)	22 (68.8)		38 (55.1)		152 (66.7)
Yes	35 (27.6)	10 (31.2)	.552	31 (44.9)	.022	76 (33.3)
Palpation pain						
No	117 (92.1)	26 (81.3)		59 (85.5)		202 (88.6)
Yes	10 (7.9)	6 (18.7)	.079	10 (14.5)	.122	26 (11.4)
Biting pain						
No	110 (88.0)	26 (83.9)		51 (75.0)		187 (83.5)
Yes	15 (12.0)	5 (16.1)	.439	17 (25.0)	.021	37 (16.5)
Sinus tract present						
No	125 (98.4)	30 (93.8)		57 (82.6)		212 (93.0)
Yes	2 (1.6)	2 (6.2)	.102	12 (17.4)	<.001	16 (7.0)
Lesion size, mm						
1-1.9	84 (66.1)	16 (50.0)		8 (11.6)		108 (47.4)
≥2	43 (33.9)	16 (50.0)	.038	61 (88.4)	<.001	120 (52.6)
Lesion location ^c						
Apical or lateral only	118 (92.9)	28 (87.5)		45 (65.2)		191 (83.8)
Lesion involving ≥2 locations	9 (7.1)	4 (12.5)	.187	24 (34.8)	<.001	37 (16.2)
Time since treatment, yr ^d						
Mean ± SD	5.62 ± 2.66	6.06 ± 3.35	.349	6.87 ± 3.71	.007	
Median (min-max)	5 (4-18)	5 (4-19)		5 (4-21)		

^aValues in *n* (%) unless noted otherwise.^bStandard error adjusted for 182 clusters in patients.^cSome large lesions extended beyond its primary region to involve surrounding root surfaces and even the furcation.^dContinuous variable.**Table 2.** Full and Final Model of Potential Risk Factors Using Independent Multinomial Probit Regression: Lesion Remained Unchanged (**U**) and Deteriorated (**D**)^a

Variables for Risk Assessment: Condition	Full Model			
	Unchanged (U)		Deteriorated (D)	
	Coefficient	<i>p</i> Value	Coefficient	<i>p</i> Value
Self-reported conditions				
Post-treatment pain: Yes	-0.567	.192	0.455	.169
Post-treatment sinus tract: Yes	0.643	.366	0.334	.562
Conditions verified by dental records				
History of flare-up: Yes	-0.541	.499	0.286	.659
Static factors since treatment				
Tooth type: Molars	0.169	.644	0.283	.389
Restoration type: Not crowns	-0.268	.474	-0.398	.263
Root-filling length: Not ideal	1.012	.008	-0.295	.421
Root-filling density: Voids present or poorly condensed	0.045	.896	0.423	.207
Current findings				
Time since treatment: annual increase after 4 yr ^b	0.039	.504	0.104	.019
Tooth is painful now: Yes ^c	1.495	.025	0.489	.406
Percussion pain: Yes	-0.059	.87	0.564	.062
Palpation pain: Yes	0.443	.426	-0.312	.478
Biting pain: Yes	0.355	.467	0.4	.29
Sinus tract present: Yes ^d	0.759	.322	1.273	.118
Lesion size: ≥2 mm ^e	0.517	.117	1.671	<.001
Lesion location: Lesion involving ≥2 locations	-0.141	.794	0.681	.088

^aBase outcome = lesion improved. For all *p* values, standard error adjusted for 182 clusters in patients. CI, confidence interval; RR, relative risk.^bFinal fitted model for lesion deteriorated (**D**): adjusted RR (95% CI): 1.11 (1.01-1.22); adjusted coefficient: 0.103; *p* = .031; risk score: 1.^cFinal fitted model for lesion deteriorated (**D**): adjusted RR (95% CI): 3.79 (1.48-9.70); adjusted coefficient: 1.332; *p* = .005; risk score: 13.^dFinal fitted model for lesion deteriorated (**D**): adjusted RR (95% CI): 4.13 (1.11-15.29); adjusted coefficient: 1.418; *p* = .034; risk score: 14.^eFinal fitted model for lesion deteriorated (**D**): adjusted RR (95% CI): 7.20 (3.70-14.02); adjusted coefficient: 1.974; *p* < .001; risk score: 20.

Deterioration Risk Score (DRS)	Predicted Probability (95% CI) (%)	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Very Low	7.8 (6.7-8.8)	3.0	96.2	25.0	69.5
Low	37.2 (35.2-39.3)	15.9	84.3	30.6	69.8
Moderate	47.5 (45.7-49.3)	2.9	97.5	33.3	69.8
High	71.3 (70.3-72.3)	5.8	99.4	80.0	70.9
Very High	85.8 (84.7-86.9)	1.4	100.0	100.0	70.0

Predicted risk DRS	No. of Cases with persistent AP (%)				
	Very Low <15	Low 15-20	Moderate 21-30	High 31-40	Very High >40
Improved AP in cohort (n=127)	83 (65.4)	18 (14.2)	24 (18.9)	2 (1.5)	0 (0)
Unchanged AP in cohort (n=32)	15 (46.9)	8 (25.0)	6 (18.8)	3 (9.3)	0 (0)
Deteriorated AP in cohort (n=69)	7 (10.1)	12 (17.4)	31 (44.9)	16 (23.3)	3 (4.3)

Figure 1. Model and Deterioration Risk Score performance. Receiver operating characteristic curve of the bootstrap sample compared favorably with that of the original full sample. Sensitivity and specificity, positive and negative predictive values (PPV and NPV) of the predicted probabilities, and distribution of apical periodontitis (AP) with predicted risk were described.

ing to DRS with corresponding probability cutoff points. Internal validation of the final fitted model was performed by drawing a bootstrap random sample of 90% of this developmental cohort, running the same multivariate independent multinomial regression model on this bootstrap sample, and comparing it against the original sample, as well as performing 500 replications of the final fitted model.

RESULTS

Characteristics of Persistent Endodontic Lesions and Potential Risk Factors

Of 331 patients screened, 182 consecutive patients with 228 lesions fulfilled the inclusion criteria and were recruited (Table 1). Three patients contributed 4 teeth each; 5 patients, 3 each; 27 patients, 2 each; and the remaining 147, 1 tooth each. Median time since treatment was 5 yr (minimum 4 yr, maximum 21 yr). Patients received treatment for molar teeth (51.8%) and

nonmolar teeth (48.2%). Most teeth were restored with crowns (77.6%) with adequate margins (74.6%), and most root fillings had ideal length (79.8%) and density (69.3%).

Just over half the lesions ($n = 127$, 55.7%) had improved over time; 32 (14.0%) remained unchanged; and 69 (30.3%) had deteriorated. Almost half the lesions ($n = 108$, 47.4%) were small but demonstrated a distinct loss of radiographic bony architecture and lamina dura. The remaining 120 lesions ranged from 2 mm to >10 mm, and none of the largest lesions were associated with improved lesions. Sex and race distributions among the 3 outcomes were similar, with almost equal numbers of male and female patients and >85% being of Chinese descent in each outcome. Patients who were 41 to 65 yr old made up the majority (67.5%). Age, race, and sex did not appear to be associated with lesion progression ($p > .05$).

Patient-reported post-treatment pain (65 cases), sinus tracts (21 cases), and flare-up after treatment (13 cases) were infrequent but significantly more common among **D** lesions ($p = .034$, $p = .002$, and $p = .007$, respectively). Lesions were mostly asymptomatic at recall (92.1%), but a complaint of pain at review appeared to be significantly associated with **U** lesions ($p = .036$) and **D** lesions ($p = .005$). A sinus tract was present at recall in only 16 cases (7.0%), 12 of which were found in **D** lesions ($p < .001$). Lesions >2 mm in diameter and lesions that persisted for longer periods were associated with **D** lesions ($p < .001$ and $p = .007$, respectively).

Potential risk factors that were clinically and statistically important ($p \leq .1$) for both **D** lesions and **U** lesions were selected for multivariate analysis and model building (Table 2).

Final Fitted Model

Significant predictors of **D** were time since treatment (RR: 1.11, 95% CI: 1.01-1.22, $p = .030$, for every year increase after 4 yr), "tooth is painful now" (RR: 3.79, 95% CI: 1.48-9.70, $p = .005$), sinus tract present (RR: 4.13, 95% CI: 1.11-15.29, $p = .034$), and lesion size (RR: 7.20, 95% CI: 3.70-14.02, $p < .001$). The only significant predictor of lesion **U** was "tooth is painful now" (RR: 3.15, 95% CI: 1.05-9.46, $p = .040$). As **U** was not a clinically important outcome in decision making and the number with outcome **U** was small, no further analysis was performed.

Deterioration Risk Score

Estimated risk scores based on rounded-up beta values of significant predictors for **D** as identified in Table 2 were as follows: time since treatment (every year of persistence beyond 4 yr), 1; a positive pain complaint at recall, 13; presence of a sinus tract, 14; radiographic lesion ≥ 2 mm, 20. A decision tree based on DRS (summation of significant predictors of deterioration) and the corresponding predicted probabilities of deterioration (Figures 1 and 2) proposed that a persistent AP presenting with very low risk would not require further endodontic intervention and could receive a definitive coronal restoration at any time; that with moderate risk might benefit from further intervention; and that with high or very high risk of deterioration should receive further intervention.

Figure 2. Decision tree for intervention of persistent apical periodontitis (AP). Deterioration Risk Score (DRS) for each persistent AP is derived from the sum of risk scores: (a) + (b) + (c) + (d).

DISCUSSION

Endodontic treatment provides predictable healing in a majority of cases (Friedman, 2008). AP associated with symptoms after treatment or a root filling of poor technical quality generally results in prompt intervention. Also, long-term recall of patients with endodontically treated teeth tends to be low (Friedman, 2008). Identifying a large cohort of suitable patients with persistent AP following root canal treatment (and for which a complete treatment record was available) was a challenge. As a result, a relatively small sample of 228 cases of persistent AP was identified in 182 patients at the end of a 33-month recruitment period involving the scrutiny of more than 2,500 patient records.

To minimize confounding by factors influencing treatment outcome, this cohort included only AP that was persistent, by conventional definition, for at least 4 yr after treatment (European Society of Endodontology, 2006). Results of the multivariate analysis showed that every year of persistence beyond 4 yr increased the risk of lesion **D** by 11% (95% CI: 1%-22%; Table 2), whereas time since treatment did not affect the risk of a lesion **U**. A trend was observed where smaller

lesions at the final examination were associated with outcomes **I** and **U**, while larger lesions were associated with the outcome **D**, consistent with the natural progression of disease following intervention. No lesion >10 mm was found in the **I** outcome, and only 1 such lesion was found in the **U** outcome. Because of the low cell numbers in 2 of the 3 outcomes, it was necessary to group lesion sizes ≥ 2 mm together for a clinically and statistically meaningful analysis of its effect on lesion progression. Collectively, lesions ≥ 2 mm in size were strongly associated with a **D** lesion.

Our final fitted model based on independent multinomial probit regression was appropriate for handling 3 discrete outcomes without a natural order and described the data well. Internal validation of the model by 500 bootstrap resampling of the original cohort and by comparing model performance of a 90% bootstrap sample against the original sample demonstrated that model and risk score performance was satisfactory, suggesting that the DRS has potential applications in (1) minimizing risk of overtreatment among teeth compatible with health; (2) identifying high-risk AP for timely intervention; (3) providing an evidence-based estimate of the risks and benefits of intervention *versus* further monitoring for shared decision making and

personalized medicine; and (4) contributing toward clinical practice guidelines. However, external validation of this model would have to be carried out in a separate clinical cohort.

It would be necessary to bear in mind some potential limitations of the DRS in clinical practice. A majority of the persistent AP in our cohort was associated with dense root filling of adequate extensions so that the DRS cannot be injudiciously applied to AP with poor technical quality. Pain is a predictor in the DRS, but it is a subjective experience and may be moderated by social and cultural norms and expectations (Yu *et al.*, 2012a). Clinical judgment is needed to determine the dimensions of a radiographic lesion, and this may influence the performance of the DRS model. In comparison with the model built on the final score agreed by both evaluators after discussion (mentioned in Materials & Methods), our preliminary analyses suggest that area under the curve for models built on independent evaluation of outcome *D* was much lower (0.6882 and 0.6297). In optimal patient care, the needs for dental rehabilitation are varied, and treatment objectives have to serve the needs of each patient. The DRS could be used as an adjunct in decision making, but it should not replace clinical acumen. If clinical suspicion of deterioration is high, intervention should be recommended regardless of the risk score.

In conclusion, this model and its decision tree could help the clinician identify persistent AP at low risk for deterioration that will not require intervention. When validated, this tool could reduce the risk of overtreatment and contribute toward targeted care and better efficiency in the timely management of disease.

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