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TO THE EDITOR:

The call for caries diagnosis by caries lesion activity assessment is growing. Lesion progression monitoring is difficult and requires careful and time-consuming record-keeping, whereas a single time-point activity assessment makes immediate treatment decisions, mainly involving preventive care, possible. Nyvad and co-workers presented a formal visual/tactile scoring system for lesion activity (1999). They also recently presented a clinical validation of this method for non-cavitated enamel lesions (2003), an excellent effort. Using their data, we would like to extend their analysis.

Nyvad and co-workers analyzed the results from a previous study involving two groups: a group that received daily supervised toothbrushing with a fluoride-containing toothpaste, and a control group that received neither supervised brushing nor fluoride. Baseline and three-year lesion assessments were available. The authors present their case as involving both construct validity and predictive validity. Most important is the case the authors present for the predictive validity of the assessment. The 'proof of the pudding' of caries activity measurement must surely lie in the validation through observed caries progression. The authors conclude that, in the control group, lesions diagnosed at baseline as active, non-cavitated (ANC) have a relative risk (RR) of 1.24 to progress in a three-year period to a cavity or filling or be extracted (CFE) as compared with lesions that, at baseline, were diagnosed as inactive, non-cavitated (INC). For the fluoride group, this RR was 1.04.

We were interested in what happened to the non-cavitated lesions that did *not* progress. Therefore, we transformed the percentages in Table 1 of Nyvad *et al.* (2003) to numbers, using the totals given. The results may deviate from the original ones by a few units. From these results, we calculated

Table 1. Calculation of the Relative Risk (RR) that an ANC vs. an INC Lesion Will Progress to Either a Cavity, Be Filled, or Extracted, or Regress to Sound between Baseline and Three-year Follow-up in the Control and the Fluoride Groups

Baseline Assessment	Number of Lesions		Fractions		RR
	INC	ANC	INC	ANC	
Three-year changes					
<i>Control group</i>					
Regression to sound	62	83	0.18	0.22	1.25
Remained non-cavitated	209	188	0.60	0.51	0.84
Progression to CFE	76	101	0.22	0.27	1.24
Total	347	372	1.00	1.00	1.00
<i>Fluoride group</i>					
Regression to sound	404	275	0.25	0.36	1.45
Remained uncavitated	953	360	0.59	0.47	0.81
Progression to CFE	259	123	0.16	0.16	1.01
Total	1616	758	1.00	1.00	1.00

the numbers and fractions of ANC and INC lesions that progressed to CFE, that remained non-cavitated, and that regressed to sound. From those, we determined the relative risks of ANC lesions compared with INC lesions. These calculations are presented in the Table.

We find a RR for progression of 1.01 and 1.24 for the F-group and the control group, respectively, which agrees with the values in the paper. The discrepancy between 1.01 and the value of 1.04 in the paper can be ascribed to our necessarily inaccurate determination of the numbers in the categories. In addition, we find RR values for regression to sound of 1.45 in the F-group and 1.25 in the control group. This does imply that, in the control group (the non-F group), **the RR of regression is as large as the RR of progression**. In the fluoride group, the RR of regression is even higher. This implies that so-called active lesions are not specifically caries-active (suggesting only progression), but rather are more prone to change in general than are so-called inactive lesions. This is supported by data included in Table 1 of Nyvad and co-workers (2003), where, in the fluoride group, 53% of INC lesions remained INC, while only 19% of ANC lesions remained ANC. For the control group, these numbers were 42% and 28%, respectively.

The factors that cause this difference in susceptibility are as yet unknown. They may, for instance, involve the 'openness' of the lesion surface, the age of the lesion, or its progression stage. At this moment, we therefore suggest only that the terminology be changed to **unstable vs. stable** lesions.

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THE AUTHORS REPLY:

In their letter, which we have read with great interest, ten Bosch and Huysmans observe that in our Control group (Nyvad *et al.*, 2003), the RR for progression of active non-cavitated (ANC) lesions vs. inactive non-cavitated (INC) lesions is as large as the RR for regression ($RR_{\text{progression}} = 1.25$)

vs. $RR_{\text{regression}} = 1.24$). They furthermore note that, as concerns our Fluoride group, the $RR_{\text{regression}}$ (1.45) is even greater than the $RR_{\text{progression}}$, which they have calculated as 1.01. Based on an assumption that an active caries lesion cannot, by virtue of the definition of activity, regress, ten Bosch and Huysmans therefore suggest that the "active" vs. "inactive" caries lesion terminology be changed to one using "unstable" vs. "stable" lesions.

We are in concert with neither the argument used nor the conclusions drawn, for the following reasons: First, it seems to escape ten Bosch and Huysmans that there is a three-year lapse between the baseline recordings and the follow-up data. While we might agree with their premise that an active lesion cannot regress over a very short period, we find it difficult to accept that an initially active non-cavitated lesion should not be able to turn sound (regress) at some time during a three-year follow-up period. Similarly, we see no reason to expect that an initially inactive lesion should remain unchanged forever. In fact, lesion regression has been shown to be a commonly occurring phenomenon in longitudinal caries studies, even when there is no fluoride available in the lesion environment (Backer Dirks, 1966; Pot *et al.*, 1977). We have recently identified some biological factors that may influence the dynamic balance of lesion transitions in caries (Baelum *et al.*, 2003).

Second, in our understanding, the argumentation put forward by ten Bosch and Huysmans is clearly contradictory to well-known facts about the mode of action of fluoride in caries lesion control. Hence, fluoride is believed to exert its cariostatic effect mainly on the ongoing caries process (active lesion) (Fejerskov *et al.*, 1981) by inhibiting demineralization and promoting remineralization (ten Cate and Featherstone, 1996). During such processes, lesions may lose the clinical characteristics that warrant the diagnosis of active caries.

Even if the premise put forward by ten Bosch and Huysmans is accepted, we have difficulties in understanding how the proposed terminology could contribute to an improved understanding of the caries lesion transition dynamics, or just be better. If "unstable" is better than "active" because the term "unstable" allows for progression as well as regression, then "stable" is worse than "inactive" because it allows for neither progression nor regression.

On a methodological note, we find it relevant to point out that the RR results presented by ten Bosch and Huysmans could

have been obtained more easily—and perhaps more accurately—directly from our Table 1. Hence, $RR_{\text{regression, fluoride}} = 1.44$ (ten Bosch and Huysmans report 1.45) can be obtained directly from Table 1 as the ratio of 36% to 25%. Similarly, $RR_{\text{regression, control}} = 1.22$ (ten Bosch and Huysmans report 1.25) is obtained as the ratio of 22% to 18%. The progression RRs may similarly be calculated as: $RR_{\text{progression, fluoride}} = 16\%/16\% = 1.00$, and $RR_{\text{progression, control}} = 27\%/22\% = 1.23$.

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