The accuracy of caries risk assessment in children attending South Australian School Dental Service

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The accuracy of caries risk assessment in children attending South Australian School Dental Service

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ABSTRACT:

Objectives: To determine the accuracy of caries risk assessment conducted by clinicians during routine practice in providing care for children in the South Australian School Dental Service (SA SDS).

Methods: Baseline data on caries experience, clinicians' ratings of caries risk status and child demographics were obtained for all SA SDS patients aged 5–15 years examined during 2002–05. Children's caries incidence rate, calculated using examination data after a follow-up period of 6–24 months from baseline, was used as the gold standard to compute the sensitivity and specificity of clinicians' baseline ratings of caries risk. Multivariate binomial regression models were used to evaluate effects of children's baseline characteristics on sensitivity and specificity.

Results: A total of 133 clinicians rated caries risk status of 71,430 children during 2002 to 2005. The observed sensitivity and specificity were 0.48 and 0.86 respectively (Se+Sp=1.34). Caries experience at baseline was the strongest factor influencing accuracy in multivariable regression model. Among children with no caries experience at baseline, overall accuracy (Se+Sp) was only 1.05, whereas it was 1.28 among children with at least one tooth surfaces with caries experience at baseline.

Conclusions:

Clinicians' accuracy in predicting caries risk during routine practice was similar to levels reported in research settings that simulated patient care. Accuracy was acceptable in children who had prior caries experience at the baseline examination, while it was poor among children with no caries experience.

Introduction

Despite effective population oral health preventive programs in many western countries, dental caries among children remains a major public health issue [1]. Certain groups of children develop high levels of the disease that compromise their quality of life and place a substantial burden on the health care system [2-4]. High-risk strategy is another potential approach to address the skewed distribution of dental caries in the population. It is prudent that, in order for the high-risk strategy to work, risk status of children would need to be identified as correctly as possible and appropriate preventive care would then be applied to them [5].

Several studies have reported the clinicians' ability to accurately identify risk for chronic dental diseases such as caries and periodontal disease [6, 7]. These studies reported a reasonable level of accuracy, which was measured by combined sensitivity and specificity. They also reported a wide variation in clinician accuracy. These studies were conducted under special circumstances where examiners were dentists specially trained in using risk assessment criteria. Those circumstances may provide a "proof of principle" for the validity of caries risk assessment. However, there is little information available about accuracy in routine practice by clinicians who are not specially trained in caries risk assessment (CRA). This information is important, as the accuracy of CRA in routine practice will have implications for oral health outcomes and cost of dental care.

Studies have shown that clinicians' subjective estimate of a child's risk of developing caries was the single best predictor of DMFS/dmfs increment in a multivariate model adjusting for other factors [8]. This finding implied that caries risk could be reasonably predicted with information routinely available to clinicians at the time of examination, without the need for expensive or time consuming biological methods that have been promoted for caries risk

assessment. This conclusion has been supported by studies from Finland where dentists achieved high specificity (Sp=0.90), although low sensitivity (Se=0.44), in predicting caries risk using their subjective judgement alone [6]. In that study, the combined sensitivity and specificity was 1.34 which was reasonable. Importantly, some individual dentists predicted caries with a high combined sensitivity and specificity that approached a score of 1.60 [6].

Other studies have investigated factors that might contribute to clinicians' judgements about caries risk by studying child-related factors associated with clinicians' assessment of caries risk. A study of South Australian children reported that clinicians' assessment of caries risk was strongly associated with the caries experience of a child's teeth present at the time of assessment. For example, among 6-year-olds, mean dmfs of high-risk children was almost 50 times higher than mean dmfs of low-risk children (9.91 and 0.20 respectively). Among 12-year-olds, mean DMFS of the high-risk children was 5 times greater than that of the low-risk children [9]. However, that cross-sectional study did not investigate accuracy of clinicians' risk assessment, as judged against children's subsequent rate of caries development.

South Australia currently has a highly developed public dental programme for the provision of dental care for school children. The service is provided mainly by dental therapists. Every child in South Australia is eligible for care in the school dental service. Children are invited to enrol in the South Australian School Dental Service (SA SDS) when they start school at age five years. Enrolment can occur at any time throughout their schooling. Children are able to access any clinic in the SA SDS system. Coverage of the SDS system was over 65% of the state's primary school child population [10] between 2002 and 2005, the period used for the current study. Dental care was fully subsidised for children in primary school (aged 5–12 years, approximately).

In the early 1990s, the SA SDS adopted the risk assessment strategy as the approach to individual patient management in the SDS clinic and implemented it as a personalised dental

care program [11]. At each dental examination, the children would be classified as having either high-, medium- or low-risk of developing caries. The assessment would be based on past history and the current oral health status as determined at the current oral examination. Clinical guidelines for classification of risk status were developed within each SADS health region. The decision regarding risk level was made by the dentist or dental therapist who assessed and provided care for the child. Once the risk status of a child had been assigned, appropriate services (treatments or preventions) and recall interval would be determined for the individual. Preventive services such as fissure sealants and professionally applied fluoride are more routinely provided to high-risk children as compared with low-risk children. Oral health education was provided to all children. However, the accuracy of the caries risk assessment system in general and the performance of individual clinicians in their attempts to predict caries have not been examined. In addition, knowing an individual clinician's accuracy would help to further explore clinician factors that might influence the accuracy of the CRA. Such an understanding would help to assess the effectiveness of the risk-based prevention strategy at a program level and help to deliver better dental care to children in South Australia. Therefore, this study aimed to quantify the accuracy of CRA in routine practice and initially explore factors that are associated with the accuracy of CRA in the SA SDS.

Methods

The sampling frame for this study was children aged 5 to 15 years examined in the SA SDS between 2002 and 2005. In SA, since 2001, patient data has been captured in electronic patient records (TITANIUM) as part of routine clinic practice. Clinician had been trained in a small group De-indentified records were extracted with only identification number by SA SDS and transferred to the Australian Research Centre for Population Oral Health (ARCPOH) at the University of Adelaide. Ethical approval was received from the University

of Adelaide's Human Research Ethics Committee and the South Australian Dental Service. Informed parental consent was obtained for the clinical data extraction. Analysis was conducted with SAS 9.1.

Data collection

The baseline and follow-up examination

The first task during data management was to select from the dataset each child's first examination in the four year period ("baseline" examination). Dates from that baseline examination were used to select a subsequent follow-up examination, if one existed. The first available re-examination of a child that was made 6 or more months after his/her baseline examination was chosen as the follow-up examination for that child. Data from the baseline and the selected follow-up examination were used for the analysis. Children with only one examination during the study period were excluded from the analysis.

Data items

Information recorded during each examination and exported from TITANIUM included: caries experience of each tooth surface; child's risk status at baseline examination as classified by the examining clinician; and socio-demographic parameters such as child sex, residency location, country of birth and Indigenous status. Clinician code was also provided in the dataset.

Individual tooth surfaces were classified as decayed, filled or missing because of caries. An additional code designated surfaces that contained fissure sealants and that were otherwise sound and not restored. Five surfaces were coded for all teeth including molars incisors and canines. For both dentitions, additional guidelines were used to distinguish between teeth missing due to caries and teeth that might have been exfoliated or were extracted for orthodontic reasons [12]. The SA SDS clinical staff (where the vast majority are dental therapists) were trained in assessment and recording of dental caries following the guidelines

developed by ARCPOH researchers based on WHO guidelines [13] and National Institute of Dental Research [14] protocol. However, there were no additional procedures for calibrating examiners.

Computation of dmfs and DMFS indices

Tooth surface-level data from baseline examinations were used to compute decayed, missing or filled tooth surfaces (dmfs/DMFS). Deciduous dmfs was calculated for children aged 5 to 10 years and permanent DMFS was calculated for children aged 6 to 15 years. The dmfs and DMFS scores were calculated as sum of decayed, missing or filled tooth surfaces due to caries of the deciduous or permanent dentition. For each child total number of dmfs +DMFS was calculated.

Computation of caries incidence density

Net caries increment (NCI) was computed using a De Paola grid [15]. The De Paola grid documented the status of all surfaces at the two examinations, and mapped the changes in status that were caries increments. It also identified reversals in caries status or false decrements. Such false negatives were used to estimate the error associated with apparent positive increments. This method assumed that the number of errors due to false increments was equivalent to the number of errors due to false decrements and the resulting net caries increment represented the corrected estimate of true caries activity. In order to calculate surface–years at risk, the De Paola grid was also used to measure tooth surface time at risk for each individual [16].

$$\text{Incidence density (ID)} = \frac{\text{Number of new carious events during the study period (NCI)}}{\text{Total number of surface years at risk in the study}} X100$$

Caries incidence density for each individual at the tooth-surface level was computed according to the above equation. In this study children were aged between 5 and 15 years old. Most of children aged between 6 and 11 have a mixed dentition. A large proportion of clinician's risk assessment and predictions of caries development was for children with mixed

dentitions in this study. Therefore, incidence density was calculated as combined incidence density for both deciduous and permanent dentitions. For children who were 11 years or older at baseline, increment in the deciduous teeth was considered as zero.

Calculation of sensitivity and specificity

In order to achieve the aim of study in measuring the accuracy of CRA in routine practice and to enable comparison to that reported in other studies, sensitivity and specificity were used as the measures of caries risk assessment accuracy.

Incidence density (observed rate of caries development) was dichotomised, classifying children as having a high caries rate if they developed at least 1.2 new carious events per 100 surface-years at risk. Children with a lower rate of caries were classified as having a low caries rate. The dichotomized rate was then used as the gold standard for calculating sensitivity and specificity. The cut-off of 1.2 new carious events per 100 surface-years at risk was selected because the resulting proportion of children with a high caries rate was equivalent to or concordant with the proportion of children judged by clinicians to be at high risk at baseline.

Sensitivity and specificity were calculated using contingency tables that cross-classified children according to clinician's baseline risk assessment and observed rate of caries development (incidence density). Sensitivity and specificity were calculated for each clinician who had examined at least 20 children with both baseline and follow-up data during study period. In order to calculate sensitivity and specificity, low- and medium- categories of risk assigned by clinicians at baseline were aggregated into one group.

Variables used in the study

Risk status at the baseline examination was used as an independent variable. Socio-demographic characteristics of children recorded routinely during the examination were used as additional explanatory variables. Sex, age in years, Indigenous status, country of birth

(Australia or elsewhere) were collected for every child. Fluoridation status of the area of residence was assigned based on level of fluoride in public water supplies. Children were also classified into two groups with/without caries based on their caries experience (dmfs+DMFS) at baseline examination.

Analysis

Before developing a regression model, a description of this child population was carried out. A number of multivariate regression models were generated using SAS PROC GENMOD. The purpose of these models was to estimate associations between child-level and clinician-level characteristics associated with each measure of accuracy: sensitivity and specificity. Separate models estimated probabilities (either sensitivity or specificity) using the binomial distribution and an identity link. Level of significance; direction and magnitude of the effect of each factor were examined.

In the models for sensitivity, the probability of being predicted as high-risk of developing caries at baseline among children who had developed a high caries rate was estimated. The intercept of this model was the estimated sensitivity in the population when all child-level factors were fixed at the selected reference category for that factor. Reference categories were selected to represent either the largest number of children from among two or more categories of a child-level characteristic, or to represent a conventional demographic group. The estimates of individual factors indicated direction and magnitude of effect of those factors. If the estimate of a factor has a negative value indicated that sensitivity or specificity was lower in the non-reference category than in the reference category.

In the models of specificity, probability of being predicted as low risk at baseline among children who had a low rate of the disease during the follow-up was estimated. Similarly, direction and magnitude of the effect of individual factors were presented as the estimates of the models.

Results

A total of 133 clinicians examined 71,430 children with two or more examinations within the time interval at least six months apart. The majority of clinicians practised as dental therapists (83%) while the remainder 17% were dentists. Some 88.6% of clinicians were females and 79.4% were Australian-born. The modal age group was 41–50 years (41.3% of clinicians) and only 12.7% were aged 23–30 years. Nearly one half of respondents had 21–30 years experience working in dentistry. More than half of respondents reported working part-time, whilst only 2.4% reported they were employed on a casual basis (data not shown). The distribution of children by sex and residential location were similar to distribution of children for the whole state of South Australia. Two thirds of the children were from metropolitan areas. Just under half of the children had experience of deciduous or permanent caries at baseline (Table 1).

Time interval between two examinations ranges from 6 months to 4 years. Around 12% children had follow-up after less than 12 months and 90% children of this group were classified as medium or high risk (Table 1). Only 2% of children had follow-up examination after 4 years.

Children who were classified at baseline as high-risk of developing caries had significantly higher mean baseline dmfs and DMFs compared with those who were classified as medium- or low-risk of developing caries (7.52 versus 1.36 and 0.20 for deciduous dmfs; 1.30 versus 0.57 and 0.29 for permanent DMFS) (Table 2). High-risk children developed nine and two times higher caries incidence density for deciduous dentition compared to low- and medium-risk children, respectively (Table 2). Similarly, for permanent dentition, the mean of incidence density was 0.99 among high-risk children, five times higher than that among low risk children (ID=0.17). High-risk children developed almost nine times higher caries

incidence density for combined deciduous and permanent dentition compared to low -risk children (Table 2).

Sensitivity of individual clinicians ranged from 0 to 0.92 with the mean of 0.46 (Figure 1). Over half of the clinicians had sensitivity within the range of 0.40 to 0.60, while only 14% of clinicians achieved high sensitivity of 0.7 or more (Figure 1). Individual clinicians had a mean of specificity of 0.86 with minimum of 0.61 and maximum of 1.00. Some 82% of clinicians had specificity within the range of 0.80 to 1.00 (Figure 2). The level of overall accuracy for each clinician was assessed by combining sensitivity and specificity (se+sp). The majority of clinicians had a combined (se+sp) ranging from 1.30 to 1.50. There were 5% of clinicians that had low combined (se+sp). There were also 5% of clinicians that achieved a combined (se+sp) over 1.50 (Figure 3).

Sensitivity and specificity of the caries risk assessment among SA SDS children was 0.47 and 0.86, respectively (Table 3). The combined sensitivity and specificity was 1.33. More than half of the children who developed a high rate of caries within one recall cycle were not classified as being high-risk at baseline.

Both sensitivity and specificity was influenced by children's caries experience at baseline. Among children without caries experience at baseline, sensitivity was low (se=0.07) while specificity was almost perfect (sp=0.98). Among children with caries experience at baseline, sensitivity improved significantly compared with that among the no caries experience group (0.57 versus 0.07 respectively), while specificity decreased from 0.98 among group without caries experience to 0.72 among the group with caries experience (Table 3).

A number of other child-level factors were found associated with sensitivity and specificity scores of caries risk assessment in multivariate models (Table 4). The directions of association between sensitivity and specificity with every variable were reversed except the level of fluoride concentration in the water. Examining children with no caries experience

reduced the sensitivity by 0.48 and increased the specificity by 0.26. Children's age was also another factor that influenced the accuracy of caries risk assessment. Examining younger children increased sensitivity by 0.11 while decreasing specificity by 0.03 (Table 4).

Discussion

In this study caries, clinicians were moderately accurate in correctly identifying children who later developed a low rate of caries (Sp=0.86). However, they had lower accuracy in correctly identify children who later developed a high rate of caries (Se=0.47). Among children with caries experience at their baseline examination, there was greater accuracy in correctly predicting a high rate of caries (Se=0.57) although it came at the expense of reduced accuracy in correctly predicting a low rate of caries (Sp=0.71). However, accuracy was scarcely better than chance (Se+Sp=1.05) among children with no caries experience at their baseline examination.

Several aspects of the study setting and the study methods need to be considered when interpreting the results. As the study was based on routine practice, children received dental care, including caries prevention such as fissure sealants, fluoride varnish and dental health education, according to their level of assessed risk. A certain proportion of the predicted disease could have been prevented by the interventions provided. This would result in an underestimation of caries risk assessment accuracy (sensitivity and specificity). Further, when assigning a child's risk status clinicians might form their judgements after considering the child's likely clinical status over a longer period than that observed in this study. This again might contribute to underestimation of accuracy with the methods used here.

A useful risk assessment program is one with high sensitivity and specificity. However, with the inherent trade-off between sensitivity and specificity, it might be impractical to achieve both high sensitivity and specificity simultaneously. The overall caries risk assessment accuracy over one recall interval of SA SDS was 1.33. This level of accuracy was similar the

level reported in a Finnish study [6], which was conducted in similar primary care environment (children aged 5–17 year old assessed by dentists and dental hygienists working in a healthcare centre). The current study, therefore, further validated a realistic level in "real life" accuracy. The components of clinicians' accuracy were similar in both studies, with Se=0.44 and Sp=0.90 in Finish study and Se=0.47 and Sp=0.86 in this South Australian study.

In the North Carolina study [8, 17], the highest CRA's accuracy level reached 1.45 (Se=0.60 and Sp=0.85) among children in grade 1 and grade 5. Clinicians in the current study had lower accuracy, especially lower sensitivity. However, conditions in the current study were different to that of the North Carolina research study, where a more rigid research protocol was applied. Clinicians in the North Carolina study were specifically trained for the purpose of caries risk assessment. In the current study, clinicians practiced CRA as a routine part of their daily clinical practice that involved ongoing clinical patient care, including examination visits, preventive and restorative services and recalls. Therefore, it was expected that the caries risk assessment process may have lower accuracy level compared to that observed in the North Carolina study.

Clinicians in this studied varied considerably in their accuracy of caries risk prediction. Some clinicians predicted caries with a high combined sensitivity and specificity that approached a score over 1.50, while some clinicians had a much lower accuracy. This finding is comparable with a results reported in Finland by Alanen [6]. The variation among clinician's accuracy of CRA indicates that there are some clinician related factors that might account for the different in the level of accuracy.

A study evaluating predictive accuracy of caries risk assessment performed during routine clinical practice among adult patients also reported a level of overall accuracy that was similar to the level observed in this study [18]. The observed sensitivity score in this current

study was slightly lower compared with that in the other study (0.47 versus 0.57 respectively). However, caries risk assessment in this South Australian study was performed in a population including children with a mixture of deciduous dentition, mixed dentition phase and permanent dentition in contrast to the above study where adults with permanent dentition were examined.

The difficulty in individual assessment of future caries risk is widely accepted. This was evident in this current study. However, at the group level risk assessment is much stronger. The fact that children in the low risk group developed significantly fewer new carious lesions than children in high risk group (Table 2) revealed that risk assessment at the group level was far more accurate.

The stratified analysis (Table 3) revealed some level of interaction between clinicians' ability to predict caries and the true risk of developing caries. Clinicians were more likely to assign high risk status to children with caries experience at baseline, who in turn, might truly be at higher risk of developing new caries. Hence, the non-stratified analysis was a mixed effect of the abovementioned issues. The stratified analysis isolated the clinicians' true capacity to predict caries, and in both strata, the accuracy level was lower than the non-stratified estimates of accuracy.

The stratified analysis, confirmed by the multivariate analysis, indicated the strong effect of previous caries experience on accuracy of caries risk assessment. This finding was reported in the previous assessment of caries risk assessment. It means that caries risk assessment in the current form may have little value among children who have no previous caries experience.

There may be some criticism that the data used to measure the outcome variable, dental caries rate, were collected by un-calibrated clinicians. However, those clinicians were similarly trained and used uniform clinical manuals to perform the examinations. In addition, the protocol was developed by experienced oral epidemiologists from the University of Adelaide

in collaboration with South Australian Dental Service clinical leaders. This approach was also consistent with a recent statement by Hausen et al [19] that "In large enough settings, data obtained from patient records could possibly be used as a replacement for separate surveys". Also, analyses were based on the presence/absence of cavitated caries lesion (either filled or not), which is reliable [20].

Time interval between examinations in purposively-designed caries risk assessment studies was often set to be uniform. Caries increment is time-dependent. Therefore, net caries increment was often the "gold standard" of choice in those studies. This was not possible in this study, where the time interval between examinations varied considerably among children. For that reason, incidence density was used as the "gold standard" in this study which helped to overcome that problem.

To conclude, this study provided evidence that routine caries risk assessment performance among children in the SA SDS was accurate at the group level. However, at the individual level accuracy was lower, especially among those children with no previous caries experience. This study confirms that caries risk assessment within SDS is only a method to justify when place children into different recall interval.

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Contributorship Statement

The authors declared that they all have contributed to the manuscript and have approved the final version.

Competing Interests

None

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Table 1: Study sample characteristics

Variables (effective n)	Study sample characteristics (%)
Total (71,430)	
Sex (71,430)	
Boy	50.8
Girl	49.2
Born in Australia (62,341)	
Yes	95.7
No	4.3
Indigenous identity (59,954)	
Indigenous	2.1
Non-Indigenous	97.9
Residential location (67,305)	
Adelaide (Capital City)	66.5
Other areas	33.5
Risk status at baseline (71,430)	
Low-risk	21
Medium-risk	57.9
High-risk	21.1
Caries status at baseline (71,430)	
DMFS+dmfs=0	53.3
DMFS+dmfs>0	46.7
Time interval between Two examination (71,430)	
6-12 months	12.18
12-18 months	44.5
>18 months	43.32

Some children had missing information on several variables

Table 2: Caries experience at baseline and caries increment during the followup by baseline risk

Baseline caries experience	Low-risk at baseline Mean (95%CI)	Medium-risk at baseline Mean (95%CI)	High-risk at baseline Mean (95%CI)
Baseline dmfs	0.20 (0.19-0.22)	1.36 (1.33-1.39)	7.52 (7.40-7.65)
Baseline DMFS	0.29 (0.27-0.31)	0.57 (0.56-0.59)	1.30 (1.25-1.34)
Baseline DMFS + dmfs	0.49 (0.47-0.52)	1.93 (1.90-1.96)	8.82 (8.70-8.95)
Caries rate (Incidence density*)			
Deciduous dentition	0.32 (0.30–0.34)	0.84 (0.82–0.85)	2.83 (2.77–2.88)
Permanent dentition	0.17 (0.16–0.17)	0.37 (0.36–0.37)	0.99 (0.97–1.02)
Combined	0.20 (0.19-0.21)	0.52 (0.51-0.53)	1.74 (1.71-1.77)

*Incidence Density: newly-affected surfaces per 100 surface-years at risk

95% CI: 95% confidence intervals. Estimates were judged to be statistically significant if its 95% CIs did not overlap.

Table 3: Overall sensitivity and specificity among children without/with caries experience at baseline

Risk status at baseline	Follow-up		Total
	Incidence density* (Gold standard)		
	High rate ≥ 1.2	Low or medium rate <1.2	
Among all children			
High, n (col. proportion)	6,997 (**0.47**)[a]	8,051 (0.14)	15,048
Low /Medium n (col. proportion)	7,831 (0.53)	48,551 (**0.86**)[b]	56,382
Total, n	14,828	56,602	71,430
	sensitivity + specificity = **1.33**		
Among children without caries experience at baseline			
High, n (col. proportion)	186 (**0.07**)[a]	609 (0.02)	795
Low /Medium, n (col. proportion)	2,650 (0.93)	29,898 (**0.98**)[b]	32,548
Total, n	2,836	30,507	33,343
	sensitivity + specificity =**1.05**		
Among children with caries experience at baseline			
High, n (col. proportion)	6,811 (**0.57**)[a]	7,442 (0.29)	14,253
Low /Medium, n (col. proportion)	5,181 (0.43)	18,653 (**0.71**)[b]	23,834
Total, n	11,992	26,095	38,087
sensitivity + specificity **=1.28**			

*Incidence density: newly-affected surfaces per 100 surface-years at risk

a Sensitivity

b Specificity

Figure 1: Individual clinician's sensitivity

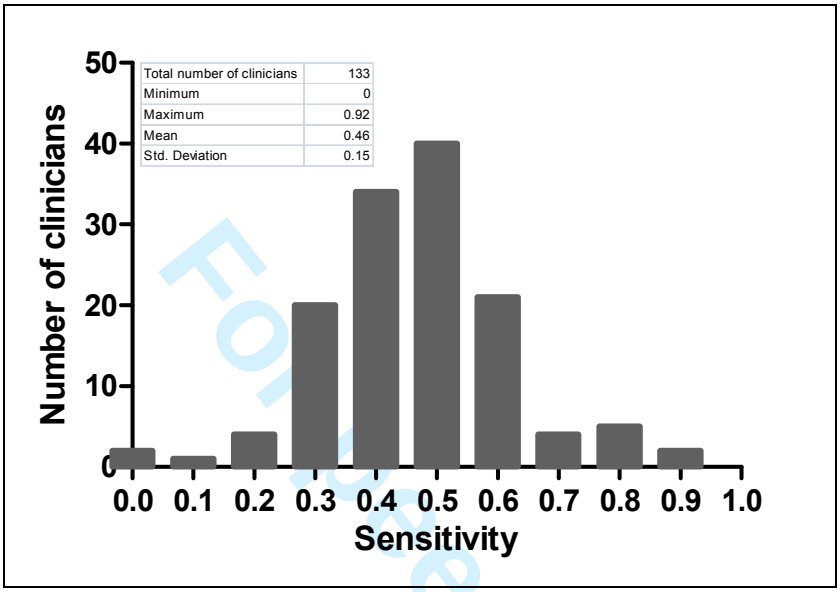

Figure 2: Individual clinician's specificity

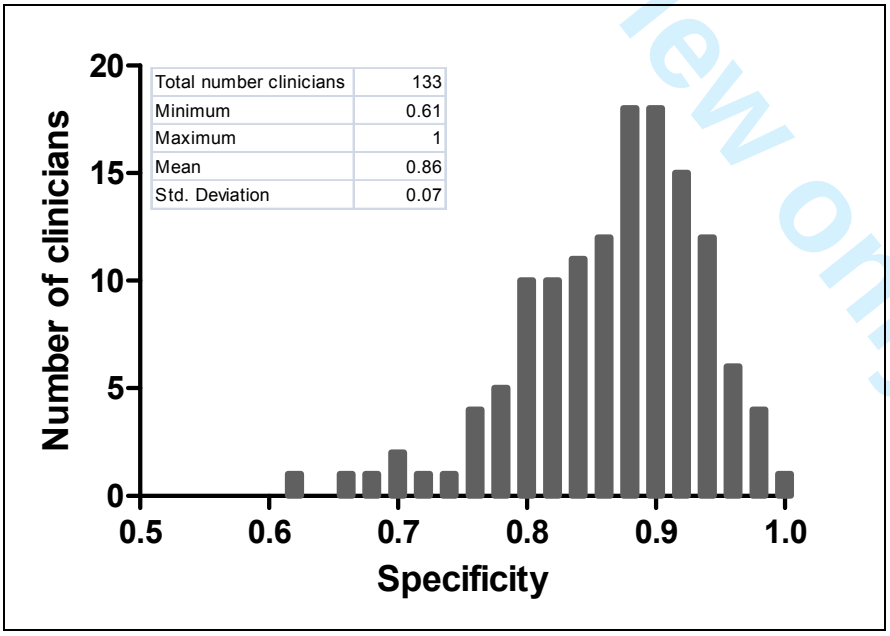

Figure 3: Individual clinician combined sensitivity + specificity

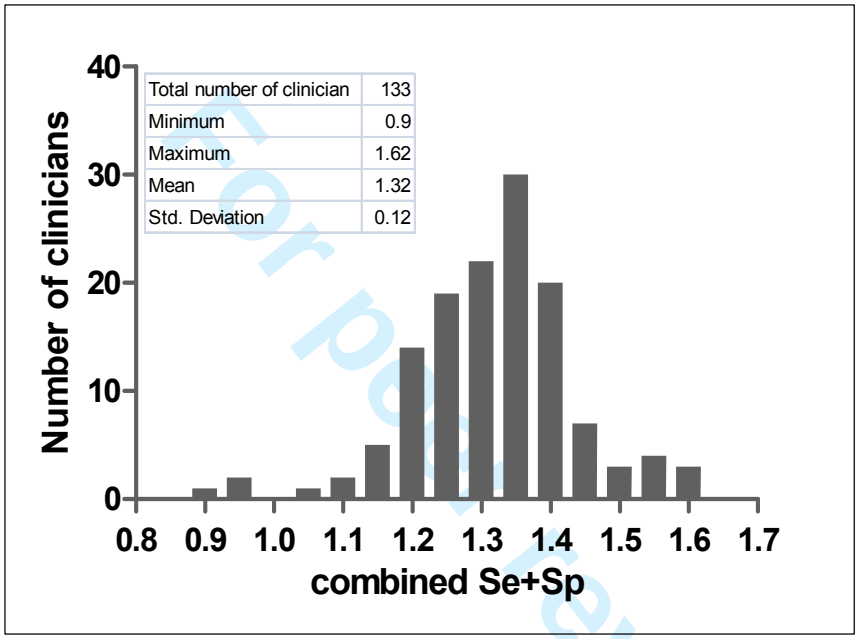

Table 4: Multivariate binomial regression model for sensitivity and specificity by child-level factors

	Sensitivity		Specificity	
	Estimate	**P**	**Estimate**	**P**
Intercept	0.58	<0.001	0.71	<0.001
Child's sex				
Boy	0.02	0.006	0.00	0.0119
Girl	ref		ref	
Child's country of birth				
Australia	-0.09	<0.001	0.02	0.0013
Overseas	ref		ref	
Child's residence				
Fluoridated area	-0.01	0.2867	0.00	0.4326
Non-fluoridated area	ref		ref	
Child's baseline caries experience				
DMFS + dmfs=0	-0.48	<0.001	0.26	<0.001
DMFS + dmfs>0	ref		ref	
Child's Indigenous status				
Yes	0.07	0.0042	-0.04	0.006
No	ref		ref	
Child's age				
5-7 years	0.11	<0.001	-0.03	<0.001
8-12 years	0.01	0.2624	0.00	0.049
13-15 years	ref		ref	

The accuracy of caries risk assessment in children attending South Australian School Dental Service

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The accuracy of caries risk assessment in children attending South Australian School Dental Service

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ABSTRACT:

Objectives: To determine the accuracy of the caries risk assessment system and performance of clinicians in their attempts to predict caries for children during routine practice.

Design: Longitudinal study

Setting and participants: Data on caries risk assessment conducted by clinicians during routine practice while providing care for children in the South Australian School Dental Service (SA SDS) were collected from electronic patient records. Baseline data on caries experience, clinicians' ratings of caries risk status and child demographics were obtained for all SA SDS patients aged 5–15 years examined during 2002–05.

Outcome measure: Children's caries incidence rate, calculated using examination data after a follow-up period of 6–48 months from baseline, was used as the gold standard to compute the sensitivity and specificity of clinicians' baseline ratings of caries risk. Multivariate binomial regression models were used to evaluate effects of children's baseline characteristics on sensitivity and specificity.

Results: A total of 133 clinicians rated caries risk status of 71,430 children during 2002 to 2005. The observed sensitivity and specificity were 0.48 and 0.86 respectively (Se+Sp=1.34). Caries experience at baseline was the strongest factor influencing accuracy in multivariable regression model. Among children with no caries experience at baseline, overall accuracy (Se+Sp) was only 1.05, whereas it was 1.28 among children with at least one tooth surfaces with caries experience at baseline.

Conclusions: Clinicians' accuracy in predicting caries risk during routine practice was similar to levels reported in research settings that simulated patient care. Accuracy was acceptable in children who had prior caries experience at the baseline examination, while it was poor among children with no caries experience.

Keywords: Caries Risk Assessment, Sensitivity, Specificity, School Dental Service

Strength and limitation of study:

This study measured accuracy in caries risk assessment in a real-life clinical situation. Most previous studies of caries risk assessment used data collected in the purposively designed trials where specific criteria were used to control for possible variation. Such studies provided only "proof-of-principle" evidence of the caries risk assessment process.

Clinicians in this study were not specifically trained for a caries risk assessment trial. Their performance of caries risk assessment was based on their education, experience and perception, and practice regulations applied at their clinics. This is unavoidable in any real-life clinical situation. Findings of this study therefore provide evidence of the practice of caries risk assessment in the South Australian Dental Service

Introduction

Despite effective population oral health preventive programs in many western countries, dental caries among children remains a major public health issue [1]. Certain groups of children develop high levels of the disease that compromise their quality of life and place a substantial burden on the health care system [2-4]. High-risk strategy is another potential approach to address the skewed distribution of dental caries in the population. It is prudent that, in order for the high-risk strategy to work, risk status of children would need to be identified as correctly as possible and appropriate preventive care would then be applied to them [5].

Several studies have reported the clinicians' ability to accurately identify risk for chronic dental diseases such as caries and periodontal disease [6, 7]. These studies reported a reasonable level of accuracy, which was measured by combined sensitivity and specificity. They also reported a wide variation in clinician accuracy. These studies were conducted under special circumstances where examiners were dentists specially trained in using risk assessment criteria. Those circumstances may provide a "proof of principle" for the validity of caries risk assessment. However, there is little information available about accuracy in routine practice by clinicians who are not specially trained in caries risk assessment (CRA). This information is important, as the accuracy of CRA in routine practice will have implications for oral health outcomes and cost of dental care.

Studies have shown that clinicians' subjective estimate of a child's risk of developing caries was the single best predictor of DMFS/dmfs increment in a multivariate model adjusting for other factors [8]. This finding implied that caries risk could be reasonably predicted with information routinely available to clinicians at the time of examination, without the need for expensive or time consuming biological methods that have been promoted for caries risk

assessment. This conclusion has been supported by studies from Finland where dentists achieved high specificity (Sp=0.90), although low sensitivity (Se=0.44), in predicting caries risk using their subjective judgement alone [6]. In that study, the combined sensitivity and specificity was 1.34 which was reasonable. Importantly, some individual dentists predicted caries with a high combined sensitivity and specificity that approached a score of 1.60 [6].

Other studies have investigated factors that might contribute to clinicians' judgements about caries risk by studying child-related factors associated with clinicians' assessment of caries risk. A study of South Australian children reported that clinicians' assessment of caries risk was strongly associated with the caries experience of a child's teeth present at the time of assessment. For example, among 6-year-olds, mean dmfs of high-risk children was almost 50 times higher than mean dmfs of low-risk children (9.91 and 0.20 respectively). Among 12-year-olds, mean DMFS of the high-risk children was 5 times greater than that of the low-risk children [9]. However, that cross-sectional study did not investigate accuracy of clinicians' risk assessment, as judged against children's subsequent rate of caries development.

South Australia currently has a highly developed public dental programme for the provision of dental care for school children. The service is provided mainly by dental therapists. Every child in South Australia is eligible for care in the school dental service. Children are invited to enrol in the South Australian School Dental Service (SA SDS) when they start school at age five years. Enrolment can occur at any time throughout their schooling. Children are able to access any clinic in the SA SDS system. Coverage of the SDS system was over 65% of the state's primary school child population [10] between 2002 and 2005, the period used for the current study. Dental care was fully subsidised for children in primary school (aged 5–12 years, approximately).

In the early 1990s, the SA SDS adopted the risk assessment strategy as the approach to individual patient management in the SDS clinic and implemented it as a personalised dental

care program [11]. At each dental examination, the children would be classified as having either high-, medium- or low-risk of developing caries. Clinical guidelines for classification of risk status were developed within each SADS health region. The broad guideline basically asked clinician to consider patient as high, medium or low risk if patient satisfied one or more factors on the list such as having past or current caries experience, active decalcified lesion, and severe hypoplasia. Clinicians also were instructed to look for dietary habits, oral hygiene habits, fluoride exposure and social history factors while making decision on child risk level. The decision regarding risk level was left to be made based on the subjective estimate of the dentist or dental therapist who assessed and provided care for the child. Once the risk status of a child had been assigned, appropriate services (treatments or preventions) and recall interval would be determined for the individual. Preventive services such as fissure sealants and professionally applied fluoride are more routinely provided to high-risk children as compared with low-risk children. Oral health education was provided to all children. Recall interval could range from 10 to 15 months for high risk children and 18 to 24+ months for low risk children. However, the accuracy of the caries risk assessment system in general and the performance of individual clinicians in their attempts to predict caries have not been examined. In addition, knowing an individual clinician's accuracy would help to further explore clinician factors that might influence the accuracy of the CRA. Such an understanding would help to assess the effectiveness of the risk-based prevention strategy at a program level and help to deliver better dental care to children in South Australia. Therefore, this study aimed to quantify the accuracy of CRA in routine practice and initially explore factors that are associated with the accuracy of CRA in the SA SDS.

Methods

The sampling frame for this study was children aged 5 to 15 years examined in the SA SDS between 2002 and 2005. In SA, since 2001, patient data has been captured in electronic

patient records (TITANIUM) as part of routine clinic practice. Clinician had been trained in a small group. De-identified records were extracted with only identification number by SA SDS and transferred to the Australian Research Centre for Population Oral Health (ARCPOH) at the University of Adelaide. Ethical approval was received from the University of Adelaide's Human Research Ethics Committee and the South Australian Dental Service. Informed parental consent was obtained for the clinical data extraction. Analysis was conducted with SAS 9.1.

Data collection

The baseline and follow-up examination

The first task during data management was to select from the dataset each child's first examination in the four year period ("baseline" examination). Dates from that baseline examination were used to select a subsequent follow-up examination, if one existed. The first available re-examination of a child that was made 6 or more months after his/her baseline examination was chosen as the follow-up examination for that child. Data from the baseline and the selected follow-up examination were used for the analysis. Children with only one examination during the study period were excluded from the analysis.

Data items

Information recorded during each examination and exported from TITANIUM included: caries experience of each tooth surface; child's risk status at baseline examination as classified by the examining clinician; and socio-demographic parameters such as child sex, residency location, country of birth and Indigenous status. Clinician code was also provided in the dataset.

Individual tooth surfaces were classified as decayed, filled or missing because of caries. An additional code designated surfaces that contained fissure sealants and that were otherwise sound and not restored. Five surfaces were coded for all teeth including molars incisors and

canines. For both dentitions, additional guidelines were used to distinguish between teeth missing due to caries and teeth that might have been exfoliated or were extracted for orthodontic reasons [12]. The SA SDS clinical staff (where the vast majority are dental therapists) were trained in assessment and recording of dental caries following the guidelines developed by ARCPOH researchers based on WHO guidelines [13] and National Institute of Dental Research [14] protocol. However, there were no additional procedures for calibrating examiners.

Computation of dmfs and DMFS indices

Tooth surface-level data from baseline examinations were used to compute decayed, missing or filled tooth surfaces (dmfs/DMFS). Deciduous dmfs was calculated for children aged 5 to 10 years and permanent DMFS was calculated for children aged 6 to 15 years. The dmfs and DMFS scores were calculated as sum of decayed, missing or filled tooth surfaces due to caries of the deciduous or permanent dentition. For each child total number of dmfs +DMFS was calculated.

Computation of caries incidence density

Net caries increment (NCI) was computed using a De Paola grid [15]. The De Paola grid documented the status of all surfaces at the two examinations, and mapped the changes in status that were caries increments. It also identified reversals in caries status or false decrements. Such false negatives were used to estimate the error associated with apparent positive increments. This method assumed that the number of errors due to false increments was equivalent to the number of errors due to false decrements and the resulting net caries increment represented the corrected estimate of true caries activity. In order to calculate surface–years at risk, the De Paola grid was also used to measure tooth surface time at risk for each individual [16].

$$\text{Incidence density (ID)} = \frac{\text{Number of new carious events during the study period (NCI)}}{\text{Total number of surface years at risk in the study}} X100$$

Caries incidence density for each individual at the tooth-surface level was computed according to the above equation. In this study children were aged between 5 and 15 years old. Most of children aged between 6 and 11 have a mixed dentition. A large proportion of clinician's risk assessment and predictions of caries development was for children with mixed dentitions in this study. Therefore, incidence density was calculated as combined incidence density for both deciduous and permanent dentitions. ID was calculated following the above formula, total number of new carious lesions including both deciduous and permanent dentition divided by total number of surface year at risk in both deciduous and permanent dentition. For children who were 11 years or older at baseline, increment in the deciduous teeth was considered as zero.

Calculation of sensitivity and specificity

In order to achieve the aim of study in measuring the accuracy of CRA in routine practice and to enable comparison to that reported in other studies, sensitivity and specificity were used as the measures of caries risk assessment accuracy.

Incidence density (observed rate of caries development) was dichotomised, classifying children as having a high caries rate if they developed at least 1.2 new carious events per 100 surface-years at risk. Children with a lower rate of caries were classified as having a low caries rate. The dichotomized rate was then used as the gold standard for calculating sensitivity and specificity. The cut-off of 1.2 new carious events per 100 surface-years at risk was selected because the resulting proportion of children with a high caries rate was equivalent to or concordant with the proportion of children judged by clinicians to be at high risk at baseline.

Sensitivity and specificity were calculated using contingency tables that cross-classified children according to clinician's baseline risk assessment and observed rate of caries development (incidence density). Sensitivity and specificity were calculated for each

clinician who had examined at least 20 children with both baseline and follow-up data during study period. In order to calculate sensitivity and specificity, low- and medium- categories of risk assigned by clinicians at baseline were aggregated into one group.

Variables used in the study

Risk status at the baseline examination was used as an independent variable. Socio-demographic characteristics of children recorded routinely during the examination were used as additional explanatory variables. Sex, age in years, Indigenous status, country of birth (Australia or elsewhere) were collected for every child. Fluoridation status of the area of residence was assigned based on level of fluoride in public water supplies. Children were also classified into two groups with/without caries based on their caries experience (dmfs+DMFS) at baseline examination.

Analysis

Before developing a regression model, a description of this child population was carried out. A number of multivariate regression models were generated using SAS PROC GENMOD. The purpose of these models was to estimate associations between child-level and clinician-level characteristics associated with each measure of accuracy: sensitivity and specificity. Separate models estimated probabilities (either sensitivity or specificity) using the binomial distribution and an identity link. Level of significance; direction and magnitude of the effect of each factor were examined.

In the models for sensitivity, the probability of being predicted as high-risk of developing caries at baseline among children who had developed a high caries rate was estimated. The intercept of this model was the estimated sensitivity in the population when all child-level factors were fixed at the selected reference category for that factor. Reference categories were selected to represent either the largest number of children from among two or more categories of a child-level characteristic, or to represent a conventional demographic group. The

estimates of individual factors indicated direction and magnitude of effect of those factors. If the estimate of a factor has a negative value indicated that sensitivity or specificity was lower in the non-reference category than in the reference category.

In the models of specificity, probability of being predicted as low risk at baseline among children who had a low rate of the disease during the follow-up was estimated. Similarly, direction and magnitude of the effect of individual factors were presented as the estimates of the models.

Results

A total of 133 clinicians examined 71,430 children with two or more examinations within the time interval at least six months apart. The majority of clinicians practised as dental therapists (83%) while the remainder 17% were dentists. Some 88.6% of clinicians were females and 79.4% were Australian-born. The modal age group was 41–50 years (41.3% of clinicians) and only 12.7% were aged 23–30 years. Nearly one half of respondents had 21–30 years working in dentistry. More than half of respondents reported working part-time, whilst only 2.4% reported they were employed on a casual basis (data not shown). The distribution of children by sex and residential location were similar to distribution of children for the whole state of South Australia. Two thirds of the children were from metropolitan areas. Just under half of the children had experience of deciduous or permanent caries at baseline (Table 1). Time interval between two examinations ranges from 6 months to 4 years. Around 12% children had follow-up after less than 12 months and 90% children of this group were classified as medium or high risk (Table 1). Only 2% of children had a follow-up examination later than 4 years. These children were excluded from the analysis.

Children who were classified at baseline as high-risk of developing caries had significantly higher mean baseline dmfs and DMFs compared with those who were classified as medium- or low-risk of developing caries (7.52 versus 1.36 and 0.20 for deciduous dmfs; 1.30 versus

0.57 and 0.29 for permanent DMFS) (Table 2). High-risk children developed nine and two times higher caries incidence density for deciduous dentition compared to low- and medium-risk children, respectively (Table 2). Similarly, for permanent dentition, the mean of incidence density was 0.99 among high-risk children, five times higher than that among low risk children (ID=0.17). High-risk children developed almost nine times higher caries incidence density for combined deciduous and permanent dentition compared to low -risk children (Table 2).

Sensitivity of individual clinicians ranged from 0 to 0.92 with the mean of 0.46 (Figure 1). Over half of the clinicians had sensitivity within the range of 0.40 to 0.60, while only 14% of clinicians achieved high sensitivity of 0.7 or more (Figure 1). Individual clinicians had a mean of specificity of 0.86 with minimum of 0.61 and maximum of 1.00. Some 82% of clinicians had specificity within the range of 0.80 to 1.00 (Figure 2). The level of overall accuracy for each clinician was assessed by combining sensitivity and specificity (se+sp). The majority of clinicians had a combined (se+sp) ranging from 1.30 to 1.50. There were 5% of clinicians that had low combined (se+sp). There were also 5% of clinicians that achieved a combined (se+sp) over 1.50 (Figure 3).

Sensitivity and specificity of the caries risk assessment among SA SDS children was 0.47 and 0.86, respectively (Table 3). The combined sensitivity and specificity was 1.33. More than half of the children who developed a high rate of caries within one recall cycle were not classified as being high-risk at baseline.

Both sensitivity and specificity was influenced by children's caries experience at baseline. Among children without caries experience at baseline, sensitivity was low (se=0.07) while specificity was almost perfect (sp=0.98). Among children with caries experience at baseline, sensitivity improved significantly compared with that among the no caries experience group

(0.57 versus 0.07 respectively), while specificity decreased from 0.98 among group without caries experience to 0.72 among the group with caries experience (Table 3).

A number of other child-level factors were found associated with sensitivity and specificity scores of caries risk assessment in multivariate models (Table 4). The directions of association between sensitivity and specificity with every variable were reversed except the level of fluoride concentration in the water. Examining children with no caries experience reduced the sensitivity by 0.48 and increased the specificity by 0.26. Children's age was also another factor that influenced the accuracy of caries risk assessment. Examining younger children increased sensitivity by 0.11 while decreasing specificity by 0.03 (Table 4).

Discussion

In this study caries, clinicians were moderately accurate in correctly identifying children who later developed a low rate of caries (Sp=0.86). However, they had lower accuracy in correctly identify children who later developed a high rate of caries (Se=0.47). Among children with caries experience at their baseline examination, there was greater accuracy in correctly predicting a high rate of caries (Se=0.57) although it came at the expense of reduced accuracy in correctly predicting a low rate of caries (Sp=0.71). However, accuracy was scarcely better than chance (Se+Sp=1.05) among children with no caries experience at their baseline examination.

Several aspects of the study setting and the study methods need to be considered when interpreting the results. As the study was based on routine practice, children received dental care, including caries prevention such as fissure sealants, fluoride varnish and dental health education, according to their level of assessed risk. A certain proportion of the predicted disease could have been prevented by the interventions provided. This would result in an underestimation of caries risk assessment accuracy (sensitivity and specificity). In this population, high-risk children were significantly more likely to receive fissure sealants

compared with the children who had lower risk. Therefore, the more intensive use of fissure sealants in high-risk children was expected to reduce the rate of new caries during the follow-up period. High-risk children who received fissure sealants had a lower rate of caries development than high-risk children who did not receive sealants. Similarly, low-risk children who received sealants had a lower rate of caries development than those low-risk children who did not receive sealants. This was expected to result in lower accuracy among the children who received sealants than among children who did not receive new sealants. However, the observed overall accuracy was virtually identical between the two groups (data not shown).

A possible reason for this finding might be that the effect of fissure sealant on caries increment in this child population was small. Also, the difference in the underlying rate of caries increment between the high-risk and the low-risk groups was substantial. The preventive effect of fissure sealants was not enough to offset the difference in caries rate between the two risk groups. It was possible to conclude that the accuracy of caries risk assessment in this study population was not significantly biased by the preventive treatment provided to the high-risk children.

Further, when assigning a child's risk status clinicians might form their judgements after considering the child's likely clinical status over a longer period than that observed in this study. This again might contribute to underestimation of accuracy with the methods used here.

A useful risk assessment program is one with high sensitivity and specificity. However, with the inherent trade-off between sensitivity and specificity, it might be impractical to achieve both high sensitivity and specificity simultaneously. The overall caries risk assessment accuracy over one recall interval of SA SDS was 1.33. This level of accuracy was similar the level reported in a Finnish study [6], which was conducted in similar primary care

environment (children aged 5–17 year old assessed by dentists and dental hygienists working in a healthcare centre). The current study, therefore, further validated a realistic level in "real life" accuracy. The components of clinicians' accuracy were similar in both studies, with Se=0.44 and Sp=0.90 in Finish study and Se=0.47 and Sp=0.86 in this South Australian study.

In the North Carolina study [8, 17], the highest CRA's accuracy level reached 1.45 (Se=0.60 and Sp=0.85) among children in grade 1 and grade 5. Clinicians in the current study had lower accuracy, especially lower sensitivity. However, conditions in the current study were different to that of the North Carolina research study, where a more rigid research protocol was applied. Clinicians in the North Carolina study were specifically trained for the purpose of caries risk assessment. In the current study, clinicians practiced CRA as a routine part of their daily clinical practice that involved ongoing clinical patient care, including examination visits, preventive and restorative services and recalls. Therefore, it was expected that the caries risk assessment process may have lower accuracy level compared to that observed in the North Carolina study.

Clinicians in this studied varied considerably in their accuracy of caries risk prediction. Some clinicians predicted caries with a high combined sensitivity and specificity that approached a score over 1.50, while some clinicians had a much lower accuracy. This finding is comparable with a results reported in Finland by Alanen [6]. The variation among clinician's accuracy of CRA indicates that there are some clinician related factors that might account for the different in the level of accuracy.

A study evaluating predictive accuracy of caries risk assessment performed during routine clinical practice among adult patients also reported a level of overall accuracy that was similar to the level observed in this study [18]. The observed sensitivity score in this current study was slightly lower compared with that in the other study (0.47 versus 0.57

respectively). However, caries risk assessment in this South Australian study was performed in a population including children with a mixture of deciduous dentition, mixed dentition phase and permanent dentition in contrast to the above study where adults with permanent dentition were examined.

The difficulty in individual assessment of future caries risk is widely accepted. This was evident in this current study. However, at the group level risk assessment is much stronger. The fact that children in the low risk group developed significantly fewer new carious lesions than children in high risk group (Table 2) revealed that risk assessment at the group level was far more accurate.

The stratified analysis (Table 3) revealed some level of interaction between clinicians' ability to predict caries and the true risk of developing caries. Clinicians were more likely to assign high risk status to children with caries experience at baseline, who in turn, might truly be at higher risk of developing new caries. Hence, the non-stratified analysis was a mixed effect of the abovementioned issues. The stratified analysis isolated the clinicians' true capacity to predict caries, and in both strata, the accuracy level was lower than the non-stratified estimates of accuracy.

The stratified analysis, confirmed by the multivariate analysis, indicated the strong effect of previous caries experience on accuracy of caries risk assessment. This finding was reported in the previous assessment of caries risk assessment. It means that caries risk assessment in the current form may have little value among children who have no previous caries experience.

There may be some criticism that the data used to measure the outcome variable, dental caries rate, were collected by un-calibrated clinicians. However, those clinicians were similarly trained and used uniform clinical manuals to perform the examinations. In addition, the protocol was developed by experienced oral epidemiologists from the University of Adelaide in collaboration with South Australian Dental Service clinical leaders. This approach was

also consistent with a recent statement by Hausen et al [19] that "In large enough settings, data obtained from patient records could possibly be used as a replacement for separate surveys". Also, analyses were based on the presence/absence of cavitated caries lesion (either filled or not), which is reliable [20].

Time interval between examinations in purposively-designed caries risk assessment studies was often set to be uniform. Caries increment is time-dependent. Therefore, net caries increment was often the "gold standard" of choice in those studies. This was not possible in this study, where the time interval between examinations varied considerably among children. The time factor was important in computation of caries increment. Children in the general population may have different time intervals between their dental visits that may affect the amount of disease development during the recalls. The children in this study were of different age groups who would naturally have different numbers of teeth, deciduous and permanent, present in their mouth and hence, being at risk for having caries. One advanced technique that was used in this study was the calculation of incidence density. The incidence density calculated in this study can adjust for different time intervals and number of teeth present in the mouth. The time and number of tooth surfaces present indicate the level of risk exposure for a child during the study period. Variation in risk exposure level was appropriately handled. For that reason, incidence density was used as the "gold standard" in this study which helped to overcome that problem.

This study provided evidence that routine caries risk assessment performance among children in the SA SDS was relatively accurate at the group level. However, at the individual level accuracy was lower, especially among those children with no previous caries experience. This study findings are in accordance with the existing studies that we cannot predict caries very well and that there is a large difference between caries-free and caries-active

populations. It also suggests that it might be time for researchers to minimise the search for more information on how to predict caries, as caries risk prediction is only for clinical management, and to pay more attention to research and providing an evidence based approach to population prevention strategies according to caries-free and caries-active status. An explicit decision about CRA should be made in the future: CRA is a population oral health prevention strategy or CRA is a clinical monitoring strategy [21].

To conclude, this study confirms that caries risk assessment within SDS is only a method to justify when place children into different recall interval.

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Contributorship Statement

The authors declared that they all have contributed to the manuscript and have approved the final version.

Data Sharing Statement

Data is stored at Australian Research Centre for Population Oral Health. Access to the data can be requested.

Competing interests

None

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Table 1: Study sample characteristics

Variables (effective n)	Study sample characteristics (%)
Total (71,430)	
Sex (71,430)	
Boy	50.8
Girl	49.2
Born in Australia (62,341)	
Yes	95.7
No	4.3
Indigenous identity (59,954)	
Indigenous	2.1
Non-Indigenous	97.9
Residential location (67,305)	
Adelaide (Capital City)	66.5
Other areas	33.5
Risk status at baseline (71,430)	
Low-risk	21
Medium-risk	57.9
High-risk	21.1
Caries status at baseline (71,430)	
DMFS+dmfs=0	53.3
DMFS+dmfs>0	46.7
Time interval between Two examination (71,430)	
6-12 months	12.18
12-18 months	44.5
>18 months	43.32

Some children had missing information on several variables

Table 2: Caries experience at baseline and caries increment during the followup by baseline risk

Baseline caries experience	Low-risk at baseline Mean (95%CI)	Medium-risk at baseline Mean (95%CI)	High-risk at baseline Mean (95%CI)
Baseline dmfs	0.20 (0.19-0.22)	1.36 (1.33-1.39)	7.52 (7.40-7.65)
Baseline DMFS	0.29 (0.27-0.31)	0.57 (0.56-0.59)	1.30 (1.25-1.34)
Baseline DMFS + dmfs	0.49 (0.47-0.52)	1.93 (1.90-1.96)	8.82 (8.70-8.95)
Caries rate (Incidence density*)			
Deciduous dentition	0.32 (0.30–0.34)	0.84 (0.82–0.85)	2.83 (2.77–2.88)
Permanent dentition	0.17 (0.16–0.17)	0.37 (0.36–0.37)	0.99 (0.97–1.02)
Combined	0.20 (0.19-0.21)	0.52 (0.51-0.53)	1.74 (1.71-1.77)

*Incidence Density: newly-affected surfaces per 100 surface-years at risk

95% CI: 95% confidence intervals. Estimates were judged to be statistically significant if its 95% CIs did not overlap.

Table 3: Overall sensitivity and specificity among children without/with caries experience at baseline

Risk status at baseline	Follow-up		Total
	Incidence density* (Gold standard)		
	High rate ≥ 1.2	Low or medium rate <1.2	
Among all children			
High, n (col. proportion)	6,997 (**0.47**)[a]	8,051 (0.14)	15,048
Low /Medium n (col. proportion)	7,831 (0.53)	48,551 (**0.86**)[b]	56,382
Total, n	14,828	56,602	71,430
	sensitivity + specificity = **1.33**		
Among children without caries experience at baseline			
High, n (col. proportion)	186 (**0.07**)[a]	609 (0.02)	795
Low /Medium, n (col. proportion)	2,650 (0.93)	29,898 (**0.98**)[b]	32,548
Total, n	2,836	30,507	33,343
	sensitivity + specificity =**1.05**		
Among children with caries experience at baseline			
High, n (col. proportion)	6,811 (**0.57**)[a]	7,442 (0.29)	14,253
Low /Medium, n (col. proportion)	5,181 (0.43)	18,653 (**0.71**)[b]	23,834
Total, n	11,992	26,095	38,087
sensitivity + specificity **=1.28**			

*Incidence density: newly-affected surfaces per 100 surface-years at risk

a Sensitivity

b Specificity

Figure legends

Figure 1: Individual clinician's sensitivity

Figure 2: Individual clinician's specificity

Figure 3: Individual clinician combined sensitivity + specificity

Table 4: Multivariate binomial regression model for sensitivity and specificity by child-level factors

	Sensitivity		Specificity	
	Estimate	**P**	**Estimate**	**P**
Intercept	0.58	<0.001	0.71	<0.001
Child's sex				
Boy	0.02	0.006	0.00	0.0119
Girl	ref		ref	
Child's country of birth				
Australia	-0.09	<0.001	0.02	0.0013
Overseas	ref		ref	
Child's residence				
Fluoridated area	-0.01	0.2867	0.00	0.4326
Non-fluoridated area	ref		ref	
Child's baseline caries experience				
DMFS + dmfs=0	-0.48	<0.001	0.26	<0.001
DMFS + dmfs>0	ref		ref	
Child's Indigenous status				
Yes	0.07	0.0042	-0.04	0.006
No	ref		ref	
Child's age				
5-7 years	0.11	<0.001	-0.03	<0.001
8-12 years	0.01	0.2624	0.00	0.049
13-15 years	ref		ref	

The accuracy of caries risk assessment in children attending South Australian School Dental Service

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Running title: **caries risk assessment in South Australian School Dental Service**

ABSTRACT:

Objectives: To determine the accuracy of the caries risk assessment system and performance of clinicians in their attempts to predict caries for children during routine practice.

Design: Longitudinal study

Setting and participants: Data on caries risk assessment conducted by clinicians during routine practice while providing care for children in the South Australian School Dental Service (SA SDS) were collected from electronic patient records. Baseline data on caries experience, clinicians' ratings of caries risk status and child demographics were obtained for all SA SDS patients aged 5–15 years examined during 2002–05.

Outcome measure: Children's caries incidence rate, calculated using examination data after a follow-up period of 6–48 months from baseline, was used as the gold standard to compute the sensitivity and specificity of clinicians' baseline ratings of caries risk. Multivariate binomial regression models were used to evaluate effects of children's baseline characteristics on sensitivity and specificity.

Results: A total of 133 clinicians rated caries risk status of 71,430 children during 2002 to 2005. The observed sensitivity and specificity were 0.48 and 0.86 respectively (Se+Sp=1.34). Caries experience at baseline was the strongest factor influencing accuracy in multivariable regression model. Among children with no caries experience at baseline, overall accuracy (Se+Sp) was only 1.05, whereas it was 1.28 among children with at least one tooth surfaces with caries experience at baseline.

Conclusions: Clinicians' accuracy in predicting caries risk during routine practice was similar to levels reported in research settings that simulated patient care. Accuracy was acceptable in children who had prior caries experience at the baseline examination, while it was poor among children with no caries experience.

Keywords: Caries Risk Assessment, Sensitivity, Specificity, School Dental Service

Strength and limitation of study:

This study measured accuracy in caries risk assessment in a real-life clinical situation. Most previous studies of caries risk assessment used data collected in the purposively designed trials where specific criteria were used to control for possible variation. Such studies provided only "proof-of-principle" evidence of the caries risk assessment process.

Clinicians in this study were not specifically trained for a caries risk assessment trial. Their performance of caries risk assessment was based on their education, experience and perception, and practice regulations applied at their clinics. This is unavoidable in any real-life clinical situation. Findings of this study therefore provide evidence of the practice of caries risk assessment in the South Australian Dental Service

Introduction

Despite effective population oral health preventive programs in many western countries, dental caries among children remains a major public health issue [1]. Certain groups of children develop high levels of the disease that compromise their quality of life and place a substantial burden on the health care system [2-4]. High-risk strategy is another potential approach to address the skewed distribution of dental caries in the population. It is prudent that, in order for the high-risk strategy to work, risk status of children would need to be identified as correctly as possible and appropriate preventive care would then be applied to them [5].

Several studies have reported the clinicians' ability to accurately identify risk for chronic dental diseases such as caries and periodontal disease [6, 7]. These studies reported a reasonable level of accuracy, which was measured by combined sensitivity and specificity. They also reported a wide variation in clinician accuracy. These studies were conducted under special circumstances where examiners were dentists specially trained in using risk assessment criteria. Those circumstances may provide a "proof of principle" for the validity of caries risk assessment. However, there is little information available about accuracy in routine practice by clinicians who are not specially trained in caries risk assessment (CRA). This information is important, as the accuracy of CRA in routine practice will have implications for oral health outcomes and cost of dental care.

Studies have shown that clinicians' subjective estimate of a child's risk of developing caries was the single best predictor of DMFS/dmfs increment in a multivariate model adjusting for other factors [8]. This finding implied that caries risk could be reasonably predicted with information routinely available to clinicians at the time of examination, without the need for expensive or time consuming biological methods that have been promoted for caries risk

assessment. This conclusion has been supported by studies from Finland where dentists achieved high specificity (Sp=0.90), although low sensitivity (Se=0.44), in predicting caries risk using their subjective judgement alone [6]. In that study, the combined sensitivity and specificity was 1.34 which was reasonable. Importantly, some individual dentists predicted caries with a high combined sensitivity and specificity that approached a score of 1.60 [6].

Other studies have investigated factors that might contribute to clinicians' judgements about caries risk by studying child-related factors associated with clinicians' assessment of caries risk. A study of South Australian children reported that clinicians' assessment of caries risk was strongly associated with the caries experience of a child's teeth present at the time of assessment. For example, among 6-year-olds, mean dmfs of high-risk children was almost 50 times higher than mean dmfs of low-risk children (9.91 and 0.20 respectively). Among 12-year-olds, mean DMFS of the high-risk children was 5 times greater than that of the low-risk children [9]. However, that cross-sectional study did not investigate accuracy of clinicians' risk assessment, as judged against children's subsequent rate of caries development.

South Australia currently has a highly developed public dental programme for the provision of dental care for school children. The service is provided mainly by dental therapists. Every child in South Australia is eligible for care in the school dental service. Children are invited to enrol in the South Australian School Dental Service (SA SDS) when they start school at age five years. Enrolment can occur at any time throughout their schooling. Children are able to access any clinic in the SA SDS system. Coverage of the SDS system was over 65% of the state's primary school child population [10] between 2002 and 2005, the period used for the current study. Dental care was fully subsidised for children in primary school (aged 5–12 years, approximately).

In the early 1990s, the SA SDS adopted the risk assessment strategy as the approach to individual patient management in the SDS clinic and implemented it as a personalised dental

care program [11]. At each dental examination, the children would be classified as having either high-, medium- or low-risk of developing caries. Clinical guidelines for classification of risk status were developed within each SADS health region. The broad guideline basically asked clinician to consider patient as high, medium or low risk if patient satisfied one or more factors on the list such as having past or current caries experience, active decalcified lesion, and severe hypoplasia. Clinicians also were instructed to look for dietary habits, oral hygiene habits, fluoride exposure and social history factors while making decision on child risk level. The decision regarding risk level was left to be made based on the subjective estimate of the dentist or dental therapist who assessed and provided care for the child. Once the risk status of a child had been assigned, appropriate services (treatments or preventions) and recall interval would be determined for the individual. Preventive services such as fissure sealants and professionally applied fluoride are more routinely provided to high-risk children as compared with low-risk children. Oral health education was provided to all children. Recall interval could range from 10 to 15 months for high risk children and 18 to 24+ months for low risk children. However, the accuracy of the caries risk assessment system in general and the performance of individual clinicians in their attempts to predict caries have not been examined. In addition, knowing an individual clinician's accuracy would help to further explore clinician factors that might influence the accuracy of the CRA. Such an understanding would help to assess the effectiveness of the risk-based prevention strategy at a program level and help to deliver better dental care to children in South Australia. Therefore, this study aimed to quantify the accuracy of CRA in routine practice and initially explore factors that are associated with the accuracy of CRA in the SA SDS.

Methods

The sampling frame for this study was children aged 5 to 15 years examined in the SA SDS between 2002 and 2005. In SA, since 2001, patient data has been captured in electronic

patient records (TITANIUM) as part of routine clinic practice. Clinician had been trained in a small group. De-identified records were extracted with only identification number by SA SDS and transferred to the Australian Research Centre for Population Oral Health (ARCPOH) at the University of Adelaide. Ethical approval was received from the University of Adelaide's Human Research Ethics Committee and the South Australian Dental Service. Informed parental consent was obtained for the clinical data extraction. Analysis was conducted with SAS 9.1.

Data collection

The baseline and follow-up examination

The first task during data management was to select from the dataset each child's first examination in the four year period ("baseline" examination). Dates from that baseline examination were used to select a subsequent follow-up examination, if one existed. The first available re-examination of a child that was made 6 or more months after his/her baseline examination was chosen as the follow-up examination for that child. Data from the baseline and the selected follow-up examination were used for the analysis. Children with only one examination during the study period were excluded from the analysis.

Data items

Information recorded during each examination and exported from TITANIUM included: caries experience of each tooth surface; child's risk status at baseline examination as classified by the examining clinician; and socio-demographic parameters such as child sex, residency location, country of birth and Indigenous status. Clinician code was also provided in the dataset.

Individual tooth surfaces were classified as decayed, filled or missing because of caries. An additional code designated surfaces that contained fissure sealants and that were otherwise sound and not restored. Five surfaces were coded for all teeth including molars incisors and

canines. For both dentitions, additional guidelines were used to distinguish between teeth missing due to caries and teeth that might have been exfoliated or were extracted for orthodontic reasons [12]. The SA SDS clinical staff (where the vast majority are dental therapists) were trained in assessment and recording of dental caries following the guidelines developed by ARCPOH researchers based on WHO guidelines [13] and National Institute of Dental Research [14] protocol. However, there were no additional procedures for calibrating examiners.

Computation of dmfs and DMFS indices

Tooth surface-level data from baseline examinations were used to compute decayed, missing or filled tooth surfaces (dmfs/DMFS). Deciduous dmfs was calculated for children aged 5 to 10 years and permanent DMFS was calculated for children aged 6 to 15 years. The dmfs and DMFS scores were calculated as sum of decayed, missing or filled tooth surfaces due to caries of the deciduous or permanent dentition. For each child total number of dmfs +DMFS was calculated.

Computation of caries incidence density

Net caries increment (NCI) was computed using a De Paola grid [15]. The De Paola grid documented the status of all surfaces at the two examinations, and mapped the changes in status that were caries increments. It also identified reversals in caries status or false decrements. Such false negatives were used to estimate the error associated with apparent positive increments. This method assumed that the number of errors due to false increments was equivalent to the number of errors due to false decrements and the resulting net caries increment represented the corrected estimate of true caries activity. In order to calculate surface–years at risk, the De Paola grid was also used to measure tooth surface time at risk for each individual [16].

$$\text{Incidence density (ID)} = \frac{\text{Number of new carious events during the study period (NCI)}}{\text{Total number of surface years at risk in the study}} X100$$

Caries incidence density for each individual at the tooth-surface level was computed according to the above equation. In this study children were aged between 5 and 15 years old. Most of children aged between 6 and 11 have a mixed dentition. A large proportion of clinician's risk assessment and predictions of caries development was for children with mixed dentitions in this study. Therefore, incidence density was calculated as combined incidence density for both deciduous and permanent dentitions. ID was calculated following the above formula, total number of new carious lesions including both deciduous and permanent dentition divided by total number of surface year at risk in both deciduous and permanent dentition. For children who were 11 years or older at baseline, increment in the deciduous teeth was considered as zero.

Calculation of sensitivity and specificity

In order to achieve the aim of study in measuring the accuracy of CRA in routine practice and to enable comparison to that reported in other studies, sensitivity and specificity were used as the measures of caries risk assessment accuracy.

Incidence density (observed rate of caries development) was dichotomised, classifying children as having a high caries rate if they developed at least 1.2 new carious events per 100 surface-years at risk. Children with a lower rate of caries were classified as having a low caries rate. The dichotomized rate was then used as the gold standard for calculating sensitivity and specificity. The cut-off of 1.2 new carious events per 100 surface-years at risk was selected because the resulting proportion of children with a high caries rate was equivalent to or concordant with the proportion of children judged by clinicians to be at high risk at baseline.

Sensitivity and specificity were calculated using contingency tables that cross-classified children according to clinician's baseline risk assessment and observed rate of caries development (incidence density). Sensitivity and specificity were calculated for each

clinician who had examined at least 20 children with both baseline and follow-up data during study period. In order to calculate sensitivity and specificity, low- and medium- categories of risk assigned by clinicians at baseline were aggregated into one group.

Variables used in the study

Risk status at the baseline examination was used as an independent variable. Socio-demographic characteristics of children recorded routinely during the examination were used as additional explanatory variables. Sex, age in years, Indigenous status, country of birth (Australia or elsewhere) were collected for every child. Fluoridation status of the area of residence was assigned based on level of fluoride in public water supplies. Children were also classified into two groups with/without caries based on their caries experience (dmfs+DMFS) at baseline examination.

Analysis

Before developing a regression model, a description of this child population was carried out. A number of multivariate regression models were generated using SAS PROC GENMOD. The purpose of these models was to estimate associations between child-level and clinician-level characteristics associated with each measure of accuracy: sensitivity and specificity. Separate models estimated probabilities (either sensitivity or specificity) using the binomial distribution and an identity link. Level of significance; direction and magnitude of the effect of each factor were examined.

In the models for sensitivity, the probability of being predicted as high-risk of developing caries at baseline among children who had developed a high caries rate was estimated. The intercept of this model was the estimated sensitivity in the population when all child-level factors were fixed at the selected reference category for that factor. Reference categories were selected to represent either the largest number of children from among two or more categories of a child-level characteristic, or to represent a conventional demographic group. The

estimates of individual factors indicated direction and magnitude of effect of those factors. If the estimate of a factor has a negative value indicated that sensitivity or specificity was lower in the non-reference category than in the reference category.

In the models of specificity, probability of being predicted as low risk at baseline among children who had a low rate of the disease during the follow-up was estimated. Similarly, direction and magnitude of the effect of individual factors were presented as the estimates of the models.

Results

A total of 133 clinicians examined 71,430 children with two or more examinations within the time interval at least six months apart. The majority of clinicians practised as dental therapists (83%) while the remainder 17% were dentists. Some 88.6% of clinicians were females and 79.4% were Australian-born. The modal age group was 41–50 years (41.3% of clinicians) and only 12.7% were aged 23–30 years. Nearly one half of respondents had 21–30 years working in dentistry. More than half of respondents reported working part-time, whilst only 2.4% reported they were employed on a casual basis (data not shown). The distribution of children by sex and residential location were similar to distribution of children for the whole state of South Australia. Two thirds of the children were from metropolitan areas. Just under half of the children had experience of deciduous or permanent caries at baseline (Table 1). Time interval between two examinations ranges from 6 months to 4 years. Around 12% children had follow-up after less than 12 months and 90% children of this group were classified as medium or high risk (Table 1). Only 2% of children had a follow-up examination later than 4 years. These children were excluded from the analysis.

Children who were classified at baseline as high-risk of developing caries had significantly higher mean baseline dmfs and DMFs compared with those who were classified as medium- or low-risk of developing caries (7.52 versus 1.36 and 0.20 for deciduous dmfs; 1.30 versus

0.57 and 0.29 for permanent DMFS) (Table 2~~Table 2~~). High-risk children developed nine and two times higher caries incidence density for deciduous dentition compared to low- and medium-risk children, respectively (Table 2~~Table 2~~). Similarly, for permanent dentition, the mean of incidence density was 0.99 among high-risk children, five times higher than that among low risk children (ID=0.17). High-risk children developed almost nine times higher caries incidence density for combined deciduous and permanent dentition compared to low - risk children (Table 2~~Table 2~~).

Sensitivity of individual clinicians ranged from 0 to 0.92 with the mean of 0.46 (Figure 1). Over half of the clinicians had sensitivity within the range of 0.40 to 0.60, while only 14% of clinicians achieved high sensitivity of 0.7 or more (Figure 1~~Figure 1~~). Individual clinicians had a mean of specificity of 0.86 with minimum of 0.61 and maximum of 1.00. Some 82% of clinicians had specificity within the range of 0.80 to 1.00 (Figure 2~~Figure 2~~). The level of overall accuracy for each clinician was assessed by combining sensitivity and specificity (se+sp). The majority of clinicians had a combined (se+sp) ranging from 1.30 to 1.50. There were 5% of clinicians that had low combined (se+sp). There were also 5% of clinicians that achieved a combined (se+sp) over 1.50 (Figure 3~~Figure 3~~).

Sensitivity and specificity of the caries risk assessment among SA SDS children was 0.47 and 0.86, respectively (Table 3~~Table 3~~). The combined sensitivity and specificity was 1.33. More than half of the children who developed a high rate of caries within one recall cycle were not classified as being high-risk at baseline.

Both sensitivity and specificity was influenced by children's caries experience at baseline. Among children without caries experience at baseline, sensitivity was low (se=0.07) while specificity was almost perfect (sp=0.98). Among children with caries experience at baseline, sensitivity improved significantly compared with that among the no caries experience group

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(0.57 versus 0.07 respectively), while specificity decreased from 0.98 among group without caries experience to 0.72 among the group with caries experience (Table 3~~Table 3~~).

A number of other child-level factors were found associated with sensitivity and specificity scores of caries risk assessment in multivariate models (Table 4). The directions of association between sensitivity and specificity with every variable were reversed except the level of fluoride concentration in the water. Examining children with no caries experience reduced the sensitivity by 0.48 and increased the specificity by 0.26. Children's age was also another factor that influenced the accuracy of caries risk assessment. Examining younger children increased sensitivity by 0.11 while decreasing specificity by 0.03 (Table 4~~Table 4~~).

Discussion

In this study caries, clinicians were moderately accurate in correctly identifying children who later developed a low rate of caries (Sp=0.86). However, they had lower accuracy in correctly identify children who later developed a high rate of caries (Se=0.47). Among children with caries experience at their baseline examination, there was greater accuracy in correctly predicting a high rate of caries (Se=0.57) although it came at the expense of reduced accuracy in correctly predicting a low rate of caries (Sp=0.71). However, accuracy was scarcely better than chance (Se+Sp=1.05) among children with no caries experience at their baseline examination.

Several aspects of the study setting and the study methods need to be considered when interpreting the results. As the study was based on routine practice, children received dental care, including caries prevention such as fissure sealants, fluoride varnish and dental health education, according to their level of assessed risk. A certain proportion of the predicted disease could have been prevented by the interventions provided. This would result in an underestimation of caries risk assessment accuracy (sensitivity and specificity). In this population, high-risk children were significantly more likely to receive fissure sealants

compared with the children who had lower risk. Therefore, the more intensive use of fissure sealants in high-risk children was expected to reduce the rate of new caries during the follow-up period. High-risk children who received fissure sealants had a lower rate of caries development than high-risk children who did not receive sealants. Similarly, low-risk children who received sealants had a lower rate of caries development than those low-risk children who did not receive sealants. This was expected to result in lower accuracy among the children who received sealants than among children who did not receive new sealants. However, the observed overall accuracy was virtually identical between the two groups (data not shown).

A possible reason for this finding might be that the effect of fissure sealant on caries increment in this child population was small. Also, the difference in the underlying rate of caries increment between the high-risk and the low-risk groups was substantial. The preventive effect of fissure sealants was not enough to offset the difference in caries rate between the two risk groups. It was possible to conclude that the accuracy of caries risk assessment in this study population was not significantly biased by the preventive treatment provided to the high-risk children.

Further, when assigning a child's risk status clinicians might form their judgements after considering the child's likely clinical status over a longer period than that observed in this study. This again might contribute to underestimation of accuracy with the methods used here.

A useful risk assessment program is one with high sensitivity and specificity. However, with the inherent trade-off between sensitivity and specificity, it might be impractical to achieve both high sensitivity and specificity simultaneously. The overall caries risk assessment accuracy over one recall interval of SA SDS was 1.33. This level of accuracy was similar the level reported in a Finnish study [6], which was conducted in similar primary care

environment (children aged 5–17 year old assessed by dentists and dental hygienists working in a healthcare centre). The current study, therefore, further validated a realistic level in "real life" accuracy. The components of clinicians' accuracy were similar in both studies, with Se=0.44 and Sp=0.90 in Finish study and Se=0.47 and Sp=0.86 in this South Australian study.

In the North Carolina study [8, 17], the highest CRA's accuracy level reached 1.45 (Se=0.60 and Sp=0.85) among children in grade 1 and grade 5. Clinicians in the current study had lower accuracy, especially lower sensitivity. However, conditions in the current study were different to that of the North Carolina research study, where a more rigid research protocol was applied. Clinicians in the North Carolina study were specifically trained for the purpose of caries risk assessment. In the current study, clinicians practiced CRA as a routine part of their daily clinical practice that involved ongoing clinical patient care, including examination visits, preventive and restorative services and recalls. Therefore, it was expected that the caries risk assessment process may have lower accuracy level compared to that observed in the North Carolina study.

Clinicians in this studied varied considerably in their accuracy of caries risk prediction. Some clinicians predicted caries with a high combined sensitivity and specificity that approached a score over 1.50, while some clinicians had a much lower accuracy. This finding is comparable with a results reported in Finland by Alanen [6]. The variation among clinician's accuracy of CRA indicates that there are some clinician related factors that might account for the different in the level of accuracy.

A study evaluating predictive accuracy of caries risk assessment performed during routine clinical practice among adult patients also reported a level of overall accuracy that was similar to the level observed in this study [18]. The observed sensitivity score in this current study was slightly lower compared with that in the other study (0.47 versus 0.57

respectively). However, caries risk assessment in this South Australian study was performed in a population including children with a mixture of deciduous dentition, mixed dentition phase and permanent dentition in contrast to the above study where adults with permanent dentition were examined.

The difficulty in individual assessment of future caries risk is widely accepted. This was evident in this current study. However, at the group level risk assessment is much stronger. The fact that children in the low risk group developed significantly fewer new carious lesions than children in high risk group (Table 2~~Table 2~~) revealed that risk assessment at the group level was far more accurate.

The stratified analysis (Table 3) revealed some level of interaction between clinicians' ability to predict caries and the true risk of developing caries. Clinicians were more likely to assign high risk status to children with caries experience at baseline, who in turn, might truly be at higher risk of developing new caries. Hence, the non-stratified analysis was a mixed effect of the abovementioned issues. The stratified analysis isolated the clinicians' true capacity to predict caries, and in both strata, the accuracy level was lower than the non-stratified estimates of accuracy.

The stratified analysis, confirmed by the multivariate analysis, indicated the strong effect of previous caries experience on accuracy of caries risk assessment. This finding was reported in the previous assessment of caries risk assessment. It means that caries risk assessment in the current form may have little value among children who have no previous caries experience.

There may be some criticism that the data used to measure the outcome variable, dental caries rate, were collected by un-calibrated clinicians. However, those clinicians were similarly trained and used uniform clinical manuals to perform the examinations. In addition, the protocol was developed by experienced oral epidemiologists from the University of Adelaide in collaboration with South Australian Dental Service clinical leaders. This approach was

also consistent with a recent statement by Hausen et al [19] that "In large enough settings, data obtained from patient records could possibly be used as a replacement for separate surveys". Also, analyses were based on the presence/absence of cavitated caries lesion (either filled or not), which is reliable [20].

Time interval between examinations in purposively-designed caries risk assessment studies was often set to be uniform. Caries increment is time-dependent. Therefore, net caries increment was often the "gold standard" of choice in those studies. This was not possible in this study, where the time interval between examinations varied considerably among children. The time factor was important in computation of caries increment. Children in the general population may have different time intervals between their dental visits that may affect the amount of disease development during the recalls. The children in this study were of different age groups who would naturally have different numbers of teeth, deciduous and permanent, present in their mouth and hence, being at risk for having caries. One advanced technique that was used in this study was the calculation of incidence density. The incidence density calculated in this study can adjust for different time intervals and number of teeth present in the mouth. The time and number of tooth surfaces present indicate the level of risk exposure for a child during the study period. Variation in risk exposure level was appropriately handled. For that reason, incidence density was used as the "gold standard" in this study which helped to overcome that problem.

This study provided evidence that routine caries risk assessment performance among children in the SA SDS was relatively accurate at the group level. However, at the individual level accuracy was lower, especially among those children with no previous caries experience. This study findings are in accordance with the existing studies that we cannot predict caries very well and that there is a large difference between caries-free and caries-active

populations. It also suggests that it might be time for researchers to minimise the search for more information on how to predict caries, as caries risk prediction is only for clinical management, and to pay more attention to research and providing an evidence based approach to population prevention strategies according to caries-free and caries-active status. An explicit decision about CRA should be made in the future: CRA is a population oral health prevention strategy or CRA is a clinical monitoring strategy (21).

To conclude, this study confirms that caries risk assessment within SDS is only a method to justify when place children into different recall interval.

Acknowledgments

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Table 1: Study sample characteristics

Variables (effective n)	Study sample characteristics (%)
Total (71,430)	
Sex (71,430)	
Boy	50.8
Girl	49.2
Born in Australia (62,341)	
Yes	95.7
No	4.3
Indigenous identity (59,954)	
Indigenous	2.1
Non-Indigenous	97.9
Residential location (67,305)	
Adelaide (Capital City)	66.5
Other areas	33.5
Risk status at baseline (71,430)	
Low-risk	21
Medium-risk	57.9
High-risk	21.1
Caries status at baseline (71,430)	
DMFS+dmfs=0	53.3
DMFS+dmfs>0	46.7
Time interval between Two examination (71,430)	
6-12 months	12.18
12-18 months	44.5
>18 months	43.32

Some children had missing information on several variables

Table 2: Caries experience at baseline and caries increment during the followup by baseline risk

	Low-risk at baseline	Medium-risk at baseline	High-risk at baseline
Baseline caries experience	Mean (95%CI)	Mean (95%CI)	Mean (95%CI)
Baseline dmfs	0.20 (0.19-0.22)	1.36 (1.33-1.39)	7.52 (7.40-7.65)
Baseline DMFS	0.29 (0.27-0.31)	0.57 (0.56-0.59)	1.30 (1.25-1.34)
Baseline DMFS + dmfs	0.49 (0.47-0.52)	1.93 (1.90-1.96)	8.82 (8.70-8.95)
Caries rate (Incidence density*)			
Deciduous dentition	0.32 (0.30–0.34)	0.84 (0.82–0.85)	2.83 (2.77–2.88)
Permanent dentition	0.17 (0.16–0.17)	0.37 (0.36–0.37)	0.99 (0.97–1.02)
Combined	0.20 (0.19-0.21)	0.52 (0.51-0.53)	1.74 (1.71-1.77)

*Incidence Density: newly-affected surfaces per 100 surface-years at risk

95% CI: 95% confidence intervals. Estimates were judged to be statistically significant if its 95% CIs did not overlap.

Table 3: Overall sensitivity and specificity among children without/with caries experience at baseline

Risk status at baseline	Follow-up		Total
	Incidence density* (Gold standard)		
	High rate ≥ 1.2	Low or medium rate <1.2	
Among all children			
High, n (col. proportion)	6,997 (**0.47**)[a]	8,051 (0.14)	15,048
Low /Medium n (col. proportion)	7,831 (0.53)	48,551 (**0.86**)[b]	56,382
Total, n	14,828	56,602	71,430
	sensitivity + specificity = **1.33**		
Among children without caries experience at baseline			
High, n (col. proportion)	186 (**0.07**)[a]	609 (0.02)	795
Low /Medium, n (col. proportion)	2,650 (0.93)	29,898 (**0.98**)[b]	32,548
Total, n	2,836	30,507	33,343
	sensitivity + specificity =**1.05**		
Among children with caries experience at baseline			
High, n (col. proportion)	6,811 (**0.57**)[a]	7,442 (0.29)	14,253
Low /Medium, n (col. proportion)	5,181 (0.43)	18,653 (**0.71**)[b]	23,834
Total, n	11,992	26,095	38,087
sensitivity + specificity **=1.28**			

*Incidence density: newly-affected surfaces per 100 surface-years at risk

a Sensitivity

b Specificity

Figure 1: Individual clinician's sensitivity

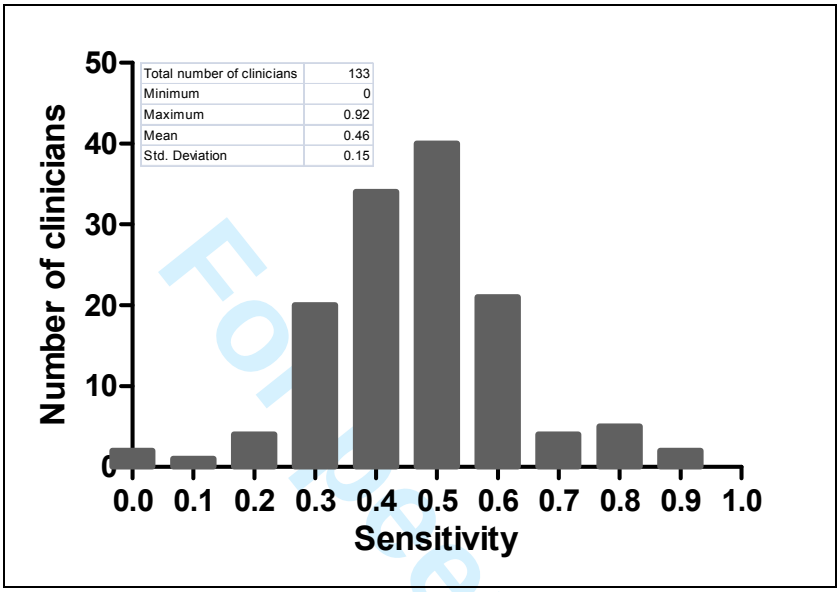


Figure 2: Individual clinician's specificity

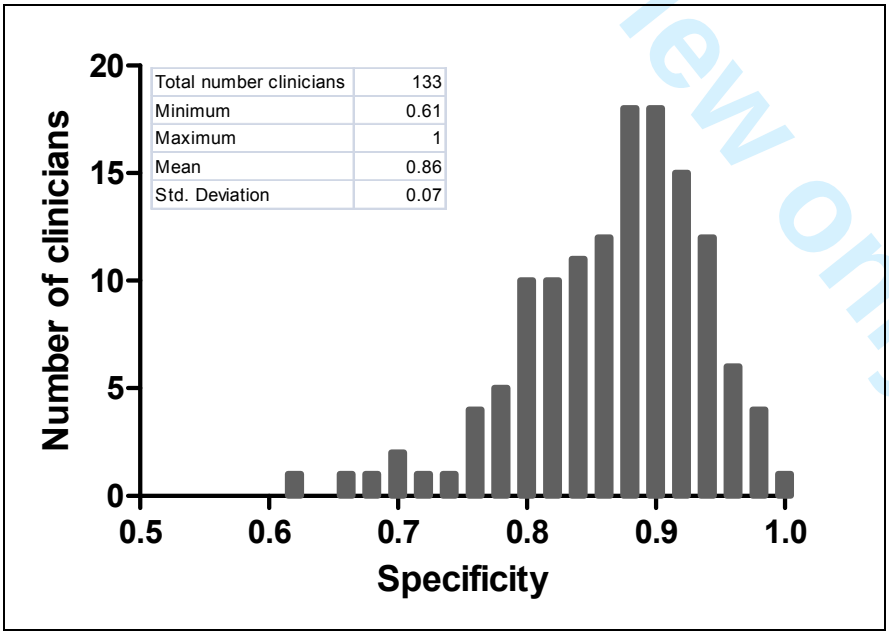


Figure 3: Individual clinician combined sensitivity + specificity

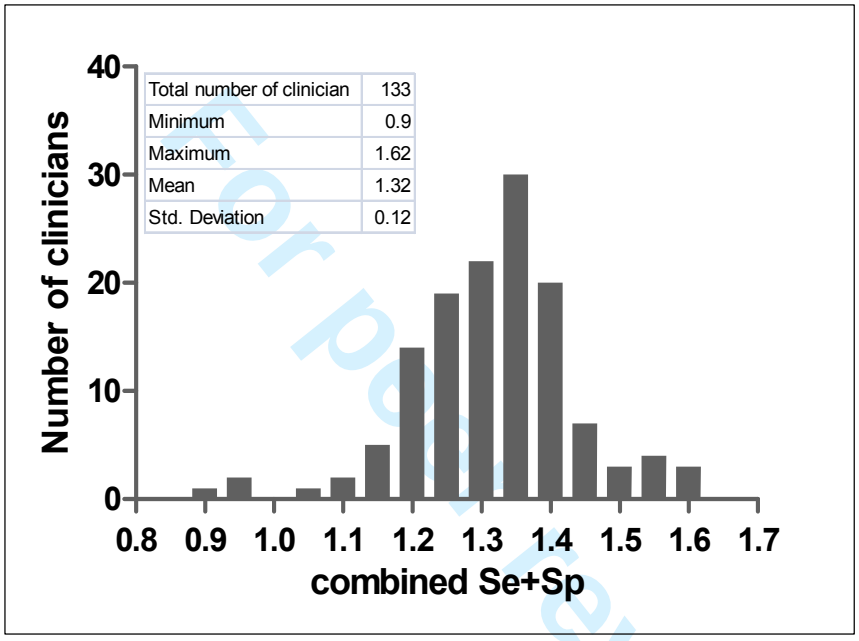
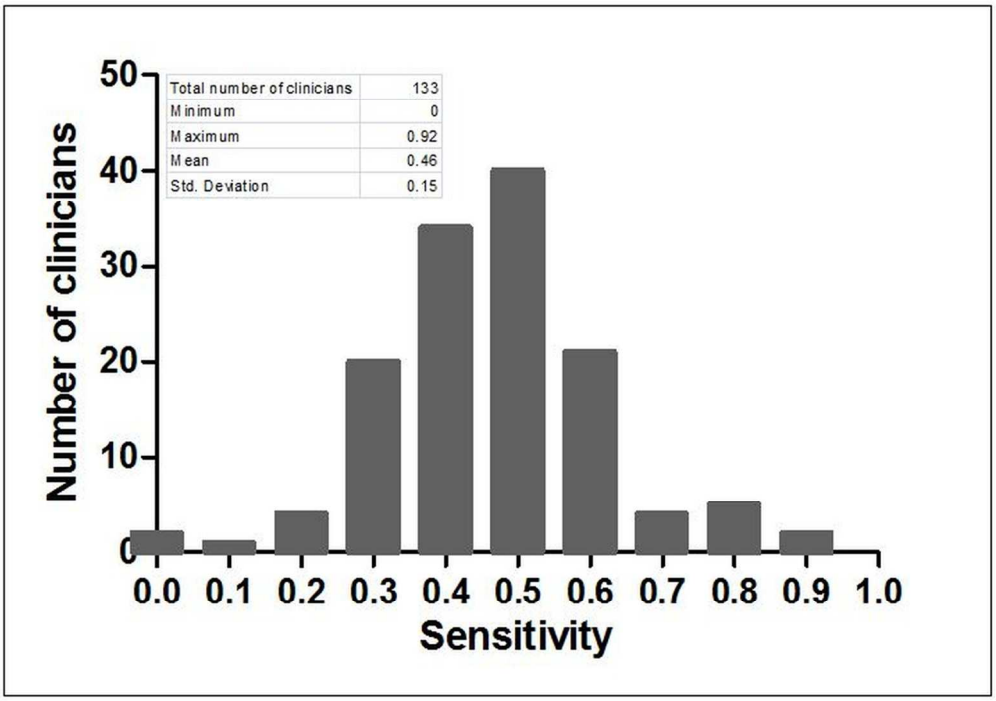


Table 4: Multivariate binomial regression model for sensitivity and specificity by child-level factors

	Sensitivity		Specificity	
	Estimate	**P**	**Estimate**	**P**
Intercept	0.58	<0.001	0.71	<0.001
Child's sex				
Boy	0.02	0.006	0.00	0.0119
Girl	ref		ref	
Child's country of birth				
Australia	-0.09	<0.001	0.02	0.0013
Overseas	ref		ref	
Child's residence				
Fluoridated area	-0.01	0.2867	0.00	0.4326
Non-fluoridated area	ref		ref	
Child's baseline caries experience				
DMFS + dmfs=0	-0.48	<0.001	0.26	<0.001
DMFS + dmfs>0	ref		ref	
Child's Indigenous status				
Yes	0.07	0.0042	-0.04	0.006
No	ref		ref	
Child's age				
5-7 years	0.11	<0.001	-0.03	<0.001
8-12 years	0.01	0.2624	0.00	0.049
13-15 years	ref		ref	



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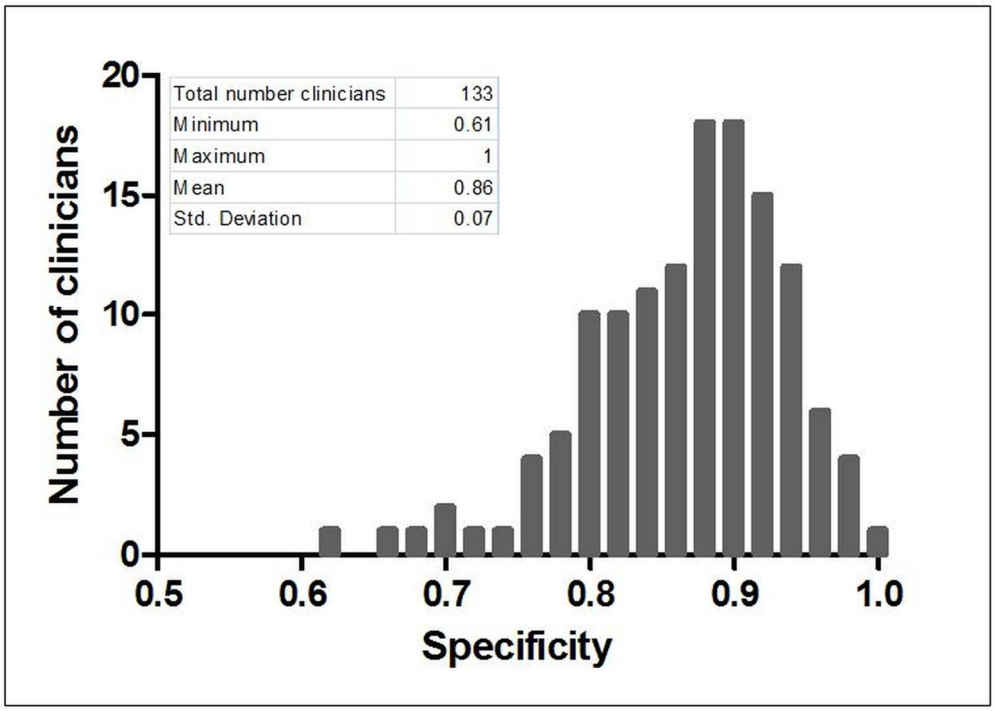

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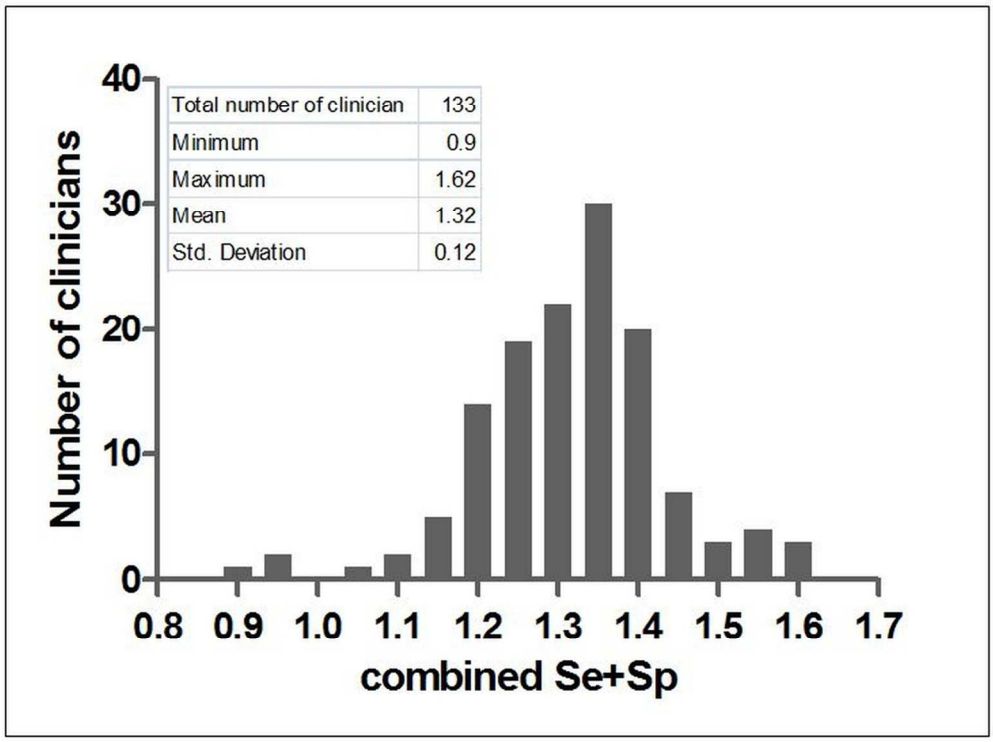

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STROBE Statement—checklist of items that should be included in reports of observational studies

	Item No	Recommendation
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract **DONE**
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found **DONE**
Introduction		
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported **DONE**
Objectives	3	State specific objectives, including any prespecified hypotheses **DONE**
Methods		
Study design	4	Present key elements of study design early in the paper **YES**
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection **YES**
Participants	6	(*a*) *Cohort study*—Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up **YES**
		(*b*) *Cohort study*—For matched studies, give matching criteria and number of exposed and unexposed *Case-control study*—For matched studies, give matching criteria and the number of controls per case
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group **DONE**
Bias	9	Describe any efforts to address potential sources of bias **YES**
Study size	10	Explain how the study size was arrived at **YES**
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why **YES**
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding **YES**
		(*b*) Describe any methods used to examine subgroups and interactions
		(*c*) Explain how missing data were addressed
		(*d*) *Cohort study*—If applicable, explain how loss to follow-up was addressed *Case-control study*—If applicable, explain how matching of cases and controls was addressed *Cross-sectional study*—If applicable, describe analytical methods taking account of sampling strategy
		(*e*) Describe any sensitivity analyses

Continued on next page

Results		
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed **YES**
		(b) Give reasons for non-participation at each stage
		(c) Consider use of a flow diagram
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders **YES**
		(b) Indicate number of participants with missing data for each variable of interest **YES**
		(c) *Cohort study*—Summarise follow-up time (eg, average and total amount)**YES**
Outcome data	15*	*Cohort study*—Report numbers of outcome events or summary measures over time **YES**
		Case-control study—Report numbers in each exposure category, or summary measures of exposure
		Cross-sectional study—Report numbers of outcome events or summary measures
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included **YES**
		(*b*) Report category boundaries when continuous variables were categorized **YES**
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses
Discussion		
Key results	18	Summarise key results with reference to study objectives **YES**
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias **YES**
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence **YES**
Generalisability	21	Discuss the generalisability (external validity) of the study results **YES**
Other information		
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based **YES**

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.