Evaluation of a Caries Risk Assessment Model in an Adult Population

by

Ferne Kraglund

A thesis submitted in conformity with the requirements for the degree of

Master of Science

Graduate Department of Dentistry (Dental Public Health)

University of Toronto

© Copyright by Ferne Kraglund (2009)
Evaluation of a Caries Risk Assessment Model in an Adult Population

Ferne Kraglund
Master of Science
Graduate Department of Dentistry (Dental Public Health)
University of Toronto
2009

Abstract

The purpose of this study was to evaluate a multifactorial caries risk assessment model by comparing the risk factor scores with caries increment. Secondary data analysis included a chart review of 434 dental patients in which basic caries risk scores, total risk score and caries increment were collected. ANOVA and logistic regression were conducted to assess the statistical significance of the difference between caries increments across categories of risk factors. Mean tooth caries increments of the risk groups were 0.20±0.8 (low), 1.13±1.59 (moderate), 2.09±2.63 (high), which were statistically significant ($p<0.001$). Past and present caries experience, carbohydrates, mucogingival bleeding index, and stimulated salivary flow showed statistically significant associations with caries increment ($p<0.05$) but the total risk score proved to be the most powerful predictor of future caries activity ($p<0.001$). The multifactorial etiology of caries necessitates the use of a risk assessment model that includes various factors that contribute to caries development.
Acknowledgements

I would like to express my sincere gratitude to a number of individuals who have contributed to the successful completion of this thesis. Firstly, I would like to thank my supervisor, Dr. Hardy Limeback, for his advice, encouragement, and mentorship. His humour and open door policy made the process one that was truly enjoyable.

I would also like to thank Dr. David Locker and Dr. Laura Dempster for participating as supervisory committee members and providing valuable feedback and support.

A special thank you must be given to Marika Banfi for helping me to learn the ins and outs of the dental software and for introducing me to various staff members; this reduced many nightmares during my data collection.

I would like to thank my friends and the staff in the Department of Community Dentistry for making these last two years memorable- thank you for your listening ears and for making me laugh when I needed it most!

This project would not have been possible without the love and support of my family and friends, both new and old. Most importantly, I need to thank my husband, James, for allowing me to uproot the family in order to pursue my dreams and goals. I am forever grateful for his unconditional support, his ability to keep me grounded, and for understanding the bigger picture. Lastly, I would like to thank my canine study buddy, Rigby, for providing snuggles and company during my study sessions.
Table of Contents

Abstract .................................................................................................................................. ii

Acknowledgements ................................................................................................................ iii

Table of Contents ................................................................................................................... iv

List of Tables and Figures ....................................................................................................... vi

Literature Review ...................................................................................................................1
  Dental Caries...................................................................................................................... 1
  Caries Distribution ......................................................................................................... 2
  Treatment of Dental Caries .......................................................................................... 4
  Modern Caries Management ......................................................................................... 6
  Caries Risk Assessment .............................................................................................. 7
  Risk Assessment Models ............................................................................................. 10
  Selection of Risk Assessment Instrument .................................................................. 13
  Risk Factors .................................................................................................................... 15
    *Streptococcus mutans* and *Lactobacillus* ......................................................... 16
  Other Salivary Factors ............................................................................................... 18
  Fluoride Exposure ........................................................................................................ 19
  Plaque ............................................................................................................................ 20
  Fermentable Carbohydrates ....................................................................................... 21
  Previous Caries Experience ......................................................................................... 23
  Sociodemographic Indicators ...................................................................................... 23
  Overall ............................................................................................................................ 25
  Multifactorial Caries Risk Assessment Models ............................................................ 26
  Cariogram ..................................................................................................................... 27
  ‘Caries Risk and Preventive Needs Assessment’ Instrument ........................................ 28

Statement of the Problem .................................................................................................... 30
  Objectives ....................................................................................................................... 30
  Hypothesis ....................................................................................................................... 30

Methodology ....................................................................................................................... 31
  Study Design .................................................................................................................. 31
  Eligibility Criteria ......................................................................................................... 31
  Sample Size .................................................................................................................... 31
  Data Collection ............................................................................................................. 32
  Data Analysis ................................................................................................................ 34

Results ................................................................................................................................. 36

Discussion ........................................................................................................................... 44
  Study Limitations .......................................................................................................... 50
LIST OF TABLES AND FIGURES

Tables

Table 1. Caries-related factors from the ‘Caries Risk and Preventive Needs Assessment’ form................................................................................................................................................................. 33

Table 2. Caries risk frequencies, sample characteristics and mean caries increments ± standard deviation.............................................................................................................................................................................. 36

Table 3. ANOVA analysis of various caries risk factors and ‘Total Risk Score’ versus mean caries increment..................................................................................................................................................................................39

Table 4. Adjusted odds ratios (OR) for the various caries risk groups in the logistic regression model........................................................................................................................................................................................................41

Table 5. Adjusted odds ratios (OR) for various factors when all independent variables except the ‘Total Risk Score’ were included in the logistic regression model......................................................... 42

Table 6. Comparison of sensitivity (Se), specificity (Sp), positive predictive (PPV) and negative predictive (NPV) values of the caries risk assessment model using two different cut-off points........................................................................................................................................................................43

Figure

Figure 1. Caries incidence by risk classification as designated by the caries risk assessment form........................................................................................................................................................................................................................................38
LITERATURE REVIEW

Dental Caries

Dental caries is a chronic, transmissible disease of multifactorial etiology. It has long been accepted that there are a large number of factors involved in the process of caries development. It is, however, the interaction of three principal factors: destructive microflora (plaque), host susceptibility (teeth and saliva), and a substrate (cariogenic diet), as described by Keyes’ three-circle diagram,\(^1\) that determine if the disease will occur.\(^2\) Demineralization of the tooth surface results when the cariogenic biofilm exists in an oral environment that is more pathological than protective.\(^3\) The specific manner in which etiological factors influence the disease process is complex, including host and pathogen adaptations, and is not fully understood.\(^4\) A fourth factor, time, was later incorporated into the diagram to account for the delay in the process of disease development. Dental caries is a slowly-progressive disease; it is not the consequence of a singular event but rather a sequel of processes occurring over a period of time.\(^5\)

One of the current approaches to better understanding dental caries is the consideration of its microbiological origin. Bacterial biofilms are ubiquitous in nature and have been found to be involved in a wide variety of microbial infections in the body, including the formation of dental plaque. A biofilm is a structured community of microorganisms encapsulated within a self-developed polymeric matrix that adheres to a living or inert surface.\(^6\) It is a sophisticated ecosystem with its own infrastructure, including metabolic and waste channels, and mechanisms in which bacteria may share genetic material and communicate with one another.\(^7\)
When the oral environment favours these bacteria, they shift from normal healthy microflora to the acidogenic (acid-forming) and acidoduric (tolerate living in acidic environments) microorganisms that are associated with dental caries. Bacteria in biofilms, such as dental plaque, are better able to survive and exhibit stronger resistance to various environmental factors as they are 1000 times more resistant to antibodies, antibiotics, and antimicrobial products. These attributes lead to persistent bacterial infections that will undoubtedly represent a new challenge in the treatment of dental caries.  

Caries Distribution

The prevalence of dental caries among children and adolescents living in industrialized nations declined in the 1970’s and 1980’s. It was suggested by most experts that regular exposure to fluoride was the most significant contribution to this decline in caries. The decline has since stabilized in many countries; however, in some areas, there are reports that the prevalence of caries is again on the rise. It was reported in *Oral Health in America: A Report of the Surgeon General* that dental caries is the single most common chronic disease of childhood, with a prevalence rate five times greater than that of asthma. Due to the universal nature of this disease, management of dental caries, typically in the form of operative procedures, remains the most routine practice in the dental office.

The diminished pervasiveness and severity of dental caries in many developed countries, along with an increasing number of dentate elderly retaining their teeth longer, have brought about a noticeably skewed distribution of disease in the population. From the North Carolina risk assessment studies, it was shown that the pattern of dental caries has changed such that the high
caries minority is suffering very different patterns of caries prevalence, risk, and activity than the low caries majority that have no or few cavitated lesions.\textsuperscript{20-22} It has been estimated that approximately 60-75\% of the caries occurs in only 20-25\% of the population. In addition, findings from the National Preventive Dentistry Demonstration Program showed that most severe disease was limited to only 5\% of the children.\textsuperscript{23}

With the earlier high prevalence of dental caries observed in western civilization between the 1950’s and 1980’s, most of society was categorized as having the disease.\textsuperscript{24} This led health professionals to utilize a population-based approach in which its goal was to alter the distribution of disease by controlling the underlying determinants of dental caries in the entire population.\textsuperscript{25-27} Fluoridation of public water systems is an example of a population-based strategy that provides preventive treatment for all members of society. Whereas water fluoridation has in the past been shown to be a successful and cost-effective population-wide strategy,\textsuperscript{28} the appropriateness of providing costly preventive measures (e.g. sealants) to whole populations has been questioned.\textsuperscript{11}

Consequently, the low prevalence and skewed distribution of disease have led some investigators to argue for a high risk targeted approach to diagnosing and treating dental caries.\textsuperscript{23,29} Using the high risk approach, the goal would be to identify highly susceptible persons and to use efficacious individual-based preventive measures to diminish their risk.\textsuperscript{24} This method operates to decrease the risk of a small number of highly vulnerable individuals to include them into the majority of the population with no or few caries.\textsuperscript{22,30} In order to use a high risk approach, one must have accurate and feasible measures for identifying those individuals that are exceedingly
prone to dental caries. A caries risk assessment may aid in the identification of etiological factors so that suitable preventive treatment may be rendered for that particular individual.

There is still much debate in the scientific community as to the appropriate way to approach the diagnosis and treatment of dental caries. Batchelor and Sheiham have argued in favour of a population approach as opposed to a high risk approach because the latter would fail to deal with the majority of new carious lesions in the population. On the contrary, Axelsson et al. have had much success using targeted approaches for prevention of dental diseases. Utilizing a high risk strategy does not imply that the general population does not require preventive dental care, but rather that the intensity of treatment should vary depending on the need. A joint approach for caries prevention in which both strategies are used may maximize the advantages of both methods by addressing those most in need, while acquiring smaller, but still significant, changes in the population’s distribution of disease.

**Treatment of Dental Caries**

Historically, it was thought that dental caries was a progressive disease that inevitably led to the eventual loss of a tooth unless a dentist intervened surgically. The conventional method of dealing with dental decay involved detection of the carious lesions, followed by drilling and filling. Although treating dental caries by restorative means will offer relief from pain and restore function to the tooth, it will likely not prevent the lifelong continuation of the disease process and will undoubtedly allow recurrent decay necessitating further surgical interventions. Restoration of the carious lesions removes areas of cariogenic microorganisms but it does not alter the risk level of the patient. Research demonstrates that placing dental
restorations contributes very little to the management of the caries disease process as there is no measurable effect on the cariogenic bacterial load in the mouth once restorative procedures are completed.\(^\text{37-39}\)

A great deal of dental work is focused on treating the symptoms of this bacterial infection rather than focusing on the causative factors. Restorations, by themselves, are incapable of modifying the etiological factors of dental caries in order to eradicate caries-forming bacteria.\(^\text{19,40}\) When health professionals are dealing with other systemic diseases, measures to eradicate the causes of the disease are utilized, such as immunizations and antibiotics. Dental professionals need to consider dental caries in the same manner and treat the disease rather than just the clinical manifestations of the disease. It is believed that our current understanding of the caries disease process is strong enough to accomplish this.\(^\text{41}\)

Numerous dental researchers have advocated following the medical model for dental caries. This approach entails regarding dental caries as a disease process, to manage its etiological factors, and to employ prevention strategies rather than simply repairing the damage caused by the disease.\(^\text{42-44}\) Medical management of dental caries is not only possible, but it has been shown to provide superior outcomes as compared to surgical intervention alone. It was demonstrated that patients treated via a caries risk assessment and medical model approach had developed significantly fewer new carious lesions than patients being treated solely with the conventional surgical approach (i.e. drill and fill).\(^\text{7}\)
Modern Caries Management

Modern caries management is based on evidence-based dentistry, with a more intense focus on prevention. Comprehensive caries control involves focusing on the whole patient to manage the individual risk factors of the patient to promote and maintain optimum oral health. Preventive dentistry is thus characterized by risk factor management in which we hope to maximize the protective factors while minimizing the pathological factors.

Modern management of dental caries includes the following sequence of treatment:

1. Detection of carious lesions at an earlier stage (incipiencies, non cavitated lesions);
2. Diagnosis of the caries disease process;
3. Identification of the patient’s caries risk factors;
4. Treatment planning, including tooth restoration, risk factor modification/elimination, arresting active lesions, and preventing future lesions; and
5. Monitoring the caries risk status of the patient.

Measures of caries control involve identifying the disease process and the risk factors; this is achieved by first performing a caries risk assessment for the patient in order to identify his/her risk factors for caries development. Once the risk factors have been detected, the dental professional provides preventive measures aimed at remineralizing incipient lesions, suppressing microbial levels, and preventing the appearance of new lesions. The clinician uses a variety of behavioural, chemical, and minimally-invasive surgical techniques to bring back a positive balance between the pathologic and protective factors that favour a healthy oral environment.

Caries control measures may include restorative treatment (with or without fluoride releasing...
materials), sealants, oral hygiene instruction, patient education, dietary analysis and modification, fluoride treatments (e.g. gel, varnish), xylitol chewing gum, and antimicrobial therapies (e.g. chlorhexidine gluconate).45

With a better understanding of the caries process, there comes a change in operative dentistry philosophy. Although there is considerable variability between dentists, there is more emphasis on preventive dentistry than ever before.35 The shift in emphasis appears to be occurring in dental schools in which curriculum and practical skills are focused more on caries risk assessment, modern management of the caries disease (including minimally invasive dentistry), and delayed restoration of teeth until the surfaces have become cavitated (or are likely to become cavitated). Restorations are only placed after all practical efforts of prevention and remineralization have been attempted.45,46

**Caries Risk Assessment**

Caries risk assessment (CRA) is the process of collecting data regarding various factors (e.g. bacterial level) and indicators (e.g. previous caries experience) to predict caries activity in the immediate future.47

Formal CRA has been described as a four-step process:

1. Identification of measurable risk factors;
2. Development of a multifactorial tool;
3. Risk assessment to determine a patient’s risk profile; and
4. Application of preventive measures tailored to the risk profile.24
For the past thirty years, researchers have focused on developing an instrument that is easy to administer, simple, quick and accurate. The risk assessment tool should estimate caries risk, identify the primary etiological factors, provide an inventory of the patient’s current preventive practices, and serve as a guide for selecting specialized preventive care tailored to that individual’s needs.²,⁴⁸ It is likely that most dentists incorporate some informal CRA into their practice based on their overall impression of the patient and previous caries experience.⁹ Research has shown that experienced clinicians are often able to assess caries risk very quickly and accurately.⁴⁹,⁵⁰ Although determination of the overall risk level may be relatively easy, pinpointing the specific factors associated with the disease process often proves to be more difficult. For that reason, it is worthwhile for dental practitioners to conduct a formal CRA to determine the precise factors involved in the patient’s disease progression.¹²

Performing a CRA assists practitioners to provide their patients appropriate levels of preventive care and to eliminate wasteful use of resources.⁵¹,⁵² By matching the person’s risk level to his/her proposed preventive therapy, the profession stands a greater chance of positively impacting their patients’ oral health. If dental professionals were able to identify, in advance, individuals at the greatest risk of developing dental caries, the cost of caries prevention procedures could be markedly reduced and their efficiency greatly increased.²²

Dental caries management by formal risk assessment represents a significant change in the mindset of the profession and should be incorporated into daily practice as dictated by practices of evidence-based standards of care. CRA should be routinely built into preliminary and recall examinations as the findings help to guide the patient’s designated course of treatment. This is
especially important before extensive prosthodontic, restorative, and orthodontic treatment is undertaken to ensure a favourable prognosis.⁵,³⁷

CRA may be valuable in the clinical management of caries by helping dental clinicians to:
1. Categorize the level of the patient’s risk of developing caries to control the intensity of treatment rendered.⁹,⁵³
2. Pinpoint main etiological factors that contribute to the development of decay and thus determine appropriate form of therapy.⁹,³⁶
3. Assist in restorative treatment decisions (e.g. choice of restorative material).⁹,⁵³
4. Improve prognosis of planned therapeutic care.⁹,⁵⁴
5. Provide information on what additional diagnostic tests and screening are required.⁹,⁵³,⁵⁴
6. Educate and motivate patients to improve and maintain optimum oral health.⁹,¹²,³⁷
7. Guide timing of subsequent recall appointments.¹²,⁵³

CRA tools screen people based on risk factors and predictors and classify patients into one of three risk categories- low, moderate, or high.⁹,⁵⁴,⁵⁵ Ordinarily, if new caries have developed since the last examination, the patient is categorized as either moderate or high risk depending on the interval since the last examination, and the number and severity of carious lesions. If, however, the patient is caries-free since the last examination, his/her risk level would be designated as low or moderate risk depending on his/her oral hygiene status, fluoride exposure, and microbiological count.²
Reaching a consensus on the moderate risk group represents the greatest diagnostic challenge. It can be rather simple to identify low risk, low caries and high risk, high caries patients. However, it is much more complicated to recognize moderate risk individuals that may have exhibited little or no disease for long periods of time followed by a sudden development of carious lesions. It is for these patients, along with people who are at risk without any apparent signs or symptoms of disease, that benefit the most from identification from CRA.\textsuperscript{36,37}

Although dental caries has long been established to be a disease of multifactorial etiology, many of the traditional caries prediction models have focused on individual factors associated with high caries activity.\textsuperscript{23} More recently, the multifactorial etiology of caries points in the direction of constructing a more promising risk assessment model that includes the various factors that contribute to the development of caries as no single test can simultaneously measure the three principal components of dental caries: host resistance, cariogenecity of the diet, and microbial pathogens.\textsuperscript{2,26,56}

**Risk Assessment Models**

Two types of variables can be used in the development of multivariable caries risk models: risk factors and risk indicators or sometimes called etiologic and non-etiologic factors, respectively.\textsuperscript{52} A risk factor is an environmental, behavioural, biologic or lifestyle exposure or characteristic that increases the probability of a disease occurring.\textsuperscript{27,57} They are part of the causal chain of disease development because they satisfy the conditions of causality, such as strength of association, temporal relationship, consistency of association, dose-response relationship, and
biological plausibility. Collection of information regarding true risk factors, such as *Streptococcus mutans*, during risk assessment may help clinicians plan preventive therapy.

A risk predictor, on the other hand, is normally a biologic marker that is indicative of the disease process, but is not thought to be etiological for that disease. It is often used synonymously with risk marker in the literature. Some risk indicators for caries, such a previous caries experience, can be powerful predictors for future lesion development without being a direct cause of the disease but offer little direction in prescribing preventive measures. Risk factors and indicators are most commonly pathological in nature (i.e. associated with disease occurrence); however, they may also be protective if they decrease an individual’s probability of developing the disease (e.g. fluoride exposure).

There are two frameworks that can be used for the development of CRA instruments: the risk model and the prediction model. The risk model, or sometimes called the etiologic model, is employed when you want to identify the risk factors for the disease to implement the most effective prevention and treatment interventions. It contains only true risk factors and is typically simple to use, but it is not intended to predict future caries risk. Because of its simplicity and stability across different subgroups of the population, it is often used for screening in public health domains.

In contrast, a prediction model uses both risk factors and risk predictors to maximize its ability to identify low and high risk individuals (i.e. maximize sensitivity and specificity). While risk predictors (e.g. baseline caries) will not influence the incidence of disease, they can be strong
predictors that are inexpensively and easily obtained. Risk factors (e.g. *Streptococcus mutans* counts) are often more costly to measure; however, they tend to be more reliable in caries prediction.\textsuperscript{27,59}

Often investigators want to use a combination risk and prediction model that encompasses both risk factors and risk indicators. The variables directly involved in the caries process either as a protective or risk factor include, but are not limited to, specific microorganisms, dental plaque, type and frequency of carbohydrates and sugars in the diet, and fluoride exposure. Conversely, risk indicators, such as previous dental experience, are often included in CRA tools as they are indirectly related to the occurrence of dental caries without participating in the actual development of carious lesions.\textsuperscript{26,27} There are some CRA instruments that support both types of models. The Cariogram\textsuperscript{60} and the CRA form developed at the University of Toronto\textsuperscript{55} do just that; they act as prediction models in that they can identify those who are high risk and they are also risk models because they can identify the risk factors involved in order to facilitate appropriate intervention planning.

Bratthall and Hänsel Petersson conducted an internet-based search for risk models. Although they found numerous reports dealing with caries prediction-based models using one or a few risk factors, there have been few attempts made in cariology to construct any practical and comprehensive risk assessment instruments.\textsuperscript{61} It was concluded at the 2001 National Institutes of Health conference that “caries is an etiologically complex disease process. It is likely that numerous microbial, genetic, immunological, behavioural, and environmental contributors to risk are at play in determining the occurrence and severity of clinical disease. Assessment tools
based on a single risk indicator are therefore unlikely to accurately discriminate between those at high and low risk. Multiple indicators combined on an appropriate scale and accounting for possible interactions, will certainly be required”. Caries risk analyses using a combination of variables have shown superior results than single factors.

**Selection of Risk Assessment Instrument**

The underlying principle of risk assessments is that individuals with elevated levels of risk factors will receive more aggressive preventive interventions. Because of this, examiners must be cautious in how they categorize patients in the various risk levels. One must decide at what stage a risk factor changes from low to moderate to high risk categorization. This can prove to be a difficult task as many etiological factors do not have a clear demarcation between absence and presence, but normally range from excellent to poor (e.g. oral hygiene) or from low to high (e.g. *Lactobacillus* counts).

Regrettably, no perfect risk factors are available for caries risk assessment; clinicians must be willing to accept a certain proportion of errors in the prediction of future caries activity. This remains true for medical conditions as well. For example, Hausen used data from the Kuopio Ischaemic Heart Disease Risk Factor Study to construct a logistic risk function. He discovered from the Receiver Operating Characteristic (ROC) curves, a summary of the predictive power of a multiple level risk factor, that the individual risk assessments of acute myocardial infarction and dental caries are equally inaccurate.
In creating a CRA tool, a trade-off between sensitivity (percentage of truly diseased persons who test positively) and specificity (percentage of persons without the disease who test negatively) must be made. In doing so, a balance is struck between the number of false positives (i.e. patients assumed to be high risk but do not develop caries) and false negatives (i.e. patients assumed to be low risk but develop caries). It is imperative that the values of sensitivity and specificity remain high to ensure that a patient labelled as high risk truly has the disease and the threat of identifying low risk individuals as diseased should be minimal.

Because it is impractical to believe that a diagnostic test would have sensitivity (Se) and specificity (Sp) values of 100%, different criteria and cut-off points have been established by researchers investigating caries risk. Wilson and Ashley suggested that Se and Sp values each be set at 80% to be considered an acceptable test for caries risk. Alternatively, it has been recommended by Fleiss and Kingman that the combined Se and Sp of a risk model should be at least 160%, which has become the gold standard amongst many researchers. Unfortunately, this benchmark value has only been achieved by a small number of caries prediction instruments.

Caries risk prediction remains an inexact science despite the sizeable amount of research devoted to the topic. Examiners must appreciate that a certain proportion of errors will be made and risk misclassification may result in providing inappropriate management, including elements of over- and undertreatment. When considering the cut-off points of Se and Sp and the trade-off between them, one must weigh the consequences of having too many false positives or false negatives.
If the CRA tool is to be used at the public health level for mass screenings, it may be advantageous to have a higher specificity. Public health administrators would want to avoid false positives and as a result the overtreatment of individuals that do not necessarily require the preventive services. This is especially crucial in environments where resources are scarce. Avoiding false positives would also be desirable in situations where the recommended disease management is invasive or costly to the patient.53,54

On the contrary, it may be more advantageous from an ethical and economical standpoint to increase the sensitivity of the test in order to avoid false negatives. Failure to identify those at risk for caries development may result in unnecessary treatment in the future that may be more costly and painful to the patient due to the progression of undiagnosed disease. By raising the sensitivity of the risk model, the number of false positives would increase. Although it may result in overtreatment of some patients, if the clinician uses an appropriate preventive strategy, this would result in little to no harm to the patient in that dental caries would not be permitted to start or progress. The patient would, however, experience some economical loss for the cost of the preventive therapy.53

**Risk Factors**

A multitude of caries risk factors and indicators have been identified over decades of research. For example, in a systematic review of risk factors for dental caries in young children, Harris *et al.* found that 106 risk factors were significantly related to the prevalence of dental caries.69 With so many factors, it can seem daunting to decide which variables should be chosen for
inclusion in the CRA instrument. The risk factors selected for research are typically dictated by the purpose of the study as there are very few standardized CRA instruments available.70

The few CRA models currently in use are recent additions to the discipline and tend to focus on the principal factors associated with caries development, namely diet, microbial pathogens, and host susceptibility factors.70 The University of Toronto’s CRA model is congruent with these instruments (e.g. Cariogram, CAMBRA- Caries Management by Risk Assessment71) in that it maintains its focus on the basic caries risk elements that can easily be identified in the dental clinic and modified through preventive care practices.

The most commonly used caries risk factors and indicators in multifactorial CRA models include levels of cariogenic bacteria (i.e. *Streptococcus mutans* and *Lactobacillus*), salivary factors (e.g. flow rate and buffering capacity), carbohydrate intake, oral hygiene, fluoride exposure, previous caries experience, and socioeconomic characteristics.48,61,72 Each of these caries risk predictors will be discussed in the following sections.

*Streptococcus mutans and Lactobacillus*

Dental caries is an infectious disease of microbial origin; the etiologic agents are regular inhabitants of the oral cavity that cause demineralization of dental hard tissue when their pathogenecity and proportions are altered in response to environmental conditions.71 Microorganisms such as *Streptococcus mutans* (SM) and *Lactobacillus* (LB) have acquired a significant advantage over other oral acidogenic bacteria due to their acidoduric nature. Not only are SM able to survive in an acidic environment, but they have also adapted the ability to
increase their rate of acid production, thus driving the pH in the oral cavity lower and forming a cariogenic plaque.\textsuperscript{24}

Whereas SM are the primary initiators of the formation of carious lesions,\textsuperscript{73} LB contribute substantially to the propagation of the lesion due to their ability to survive at a lower pH than SM. In addition, SM have evolved the capacity to store energy for occasions when fermentable carbohydrates are scarce in the oral cavity. This incredible adaptation allows oral SM levels to remain relatively constant regardless of dietary modifications. LB have yet to develop this ability and thus LB counts are often used to determine a patient’s compliance to dietary changes.\textsuperscript{45}

Traditionally, SM and LB counts have been the principal biological factors used for prediction of future caries experience.\textsuperscript{23,56,59,68,74-77} Studies have shown that not only are these microorganisms related to the incidence of dental caries, but that children with high levels of these pathogens develop a significantly greater number of carious lesions than children with low levels.\textsuperscript{78} Nevertheless, salivary levels of SM and LB have been more successful in identifying low risk children than those at an elevated risk for developing dental caries.\textsuperscript{29,79}

These salivary tests aid dental professionals in identifying the two extremes in a disease susceptible population but are less effective in predicting caries in moderate risk groups. The accuracy of tests for SM in predicting future caries in the whole population is less than 50%.\textsuperscript{2} Unfortunately, despite their prevalent use in CRA’s, the predictive power of microbiologic tests
remains uncertain at the individual level as well.\textsuperscript{29,72,79} With the exception of findings in young children, salivary levels of SM have been disappointing with regard to risk assessment.\textsuperscript{48}

Salivary tests for LB are even less sensitive than tests for caries prediction than SM. This is to be expected as LB are not primarily responsible for the initiation of dental caries but they are found in large quantities when a considerable amount of carbohydrates have been consumed.\textsuperscript{9} LB counts are commonly analyzed to reflect dietary changes and the test results can be useful to motivate patients and to monitor changes in oral hygiene, diet, and microbial therapies.\textsuperscript{26,29,47}

**Other Salivary Factors**

Saliva serves multiple protective functions against the initiation and progression of dental caries. It assists to clear food particles and bacteria from the oral cavity and it buffers the acids produced by microorganisms in dental plaque.\textsuperscript{2,9} The number of individuals suffering from a reduced salivary flow rate is increasing, especially in the elderly population. Xerostomia (dry mouth) may be the consequence of a variety of conditions including radiation therapy to the head and neck region and medical ailments such as Sjögren’s syndrome, Parkinson’s disease, and uncontrolled diabetes mellitus.\textsuperscript{29,80}

Xerostomia, however, is most commonly attributed to the side effects of many frequently prescribed medications including antihistamines, anticholinergics, and tricyclic antidepressants. Although xerostomia has long been known to be a risk factor for individuals of any age, the elderly are especially susceptible to salivary changes due to the large number of medications they are often required to take.\textsuperscript{81} This can be especially problematic for this cohort as they are
generally retaining more teeth than they have previously but suffer from unfavourable salivary conditions, which puts them at an even higher risk of developing dental caries.\textsuperscript{82,83}

Individuals with chronically reduced salivary function have been found to have a significant increase in caries activity. Many dentists rely on the patient’s complaint of xerostomia to diagnose hyposalivation; however, this subjective complaint often does not correlate with objective findings of reduced salivary flow. Testing an individual’s unstimulated salivary flow rate can be accomplished easily in clinical practice and it has a strong predictive validity for assessing caries risk. The stimulated flow rate, using paraffin wax, is also customarily measured to conclude if preventive strategies based on salivary stimulation (e.g. chewing sugarless gum) will benefit the patient.\textsuperscript{2,9}

**Fluoride Exposure**

Topical and systemic fluoride exposure, oral hygiene habits, and diet are often not strong predictive factors for caries development but they are often still included in CRA instruments because they may be prescriptive for the preventive actions recommended. Determining a problem in one or more of these areas will aid the dentist and patient to customize a care plan using these elements to alter other caries risk variables, such as bacterial and salivary factors.\textsuperscript{63}

In order to prevent dental caries, it has been recommended that a constant, low ambient level of fluoride should be maintained in the oral environment.\textsuperscript{29,84} Although both pre-eruptive (systemic) and post-eruptive (topical) mechanisms of action continue to be suggested, the literature supports the latter.\textsuperscript{85}
Fluoride works via three mechanisms:

1. Inhibition of demineralization- Fluoride becomes incorporated into the enamel hydroxyapatite crystal and reduces enamel solubility in the presence of acids (pre- and post-eruptive);

2. Enhancement of remineralization- Low levels of fluoride available in the oral cavity enhance remineralization during repeated cycles of demineralization and remineralization in the early stages of the caries process (post-eruptive);

3. Inhibition of bacterial enzymes- Fluoride inhibits glycolysis, the process by which fermentable carbohydrates are metabolized by cariogenic bacteria to produce acid (post-eruptive).  

The considerable reduction in the prevalence of dental caries from 1960 to 1990 was related initially to the introduction of fluoride into the public water system and to the subsequent use of topical fluoride through fluoridated toothpaste and professionally applied delivery systems. Its use has been shown to prevent and arrest carious lesions and its protective mechanism allows for more conservative management strategies in the prevention and treatment of dental caries. 

When completing a CRA, the various sources of fluoride must be taken into account such as fluoridated drinking water, food and drinks, fluoridated toothpaste and mouthrinse, and professionally applied topical fluoride.

**Plaque**

Because dental caries is a microbiological disease, a prerequisite for caries development is the presence of dental plaque on the teeth, and unless this biofilm is present caries will not occur, regardless of any other risk factors. Researchers have failed to demonstrate a consistent relationship between dental plaque scores and caries. Not all patients with poor plaque control
inevitably develop caries; however, those who clean their teeth infrequently or ineffectively may be at higher risk for developing carious lesions.\textsuperscript{9,12} Furthermore, conditions that hinder long-term maintenance of good oral hygiene, such as mental and physical disabilities and oral appliances, are positively associated with a higher caries risk.\textsuperscript{62}

The inconsistent relationship between oral hygiene and dental caries prevalence may be due to the manner in which data is collected. Many risk assessment models use plaque indices that were developed for the study of periodontal disease to record the oral hygiene status of patients. This can be an inaccurate means of recording because they are often based on smooth surface scores, whereas the majority of caries occurs in the pits and fissures or the interproximal surfaces of teeth.\textsuperscript{2,48}

**Fermentable Carbohydrates**

Consumption of sugar and carbohydrates is considered an important etiological factor in the development of dental caries.\textsuperscript{15,87} The role of diet is primarily local in nature rather than systemic as bacteria metabolize carbohydrates and sugar, producing acidic by-products that cause the demineralization of the enamel surface. Whether this disease activity proceeds to a carious lesion depends on various dietary elements, as well as the patient’s oral hygiene and exposure to fluoride.\textsuperscript{88}

There are several dietary elements that need to be addressed when assessing a patient’s caries risk level. Whether or not a food is cariogenic depends on a number of factors specific to the individual who eats it, namely the predominant oral bacteria in plaque, salivary flow rate and
buffering capacity, and fluoride availability in the oral cavity.\textsuperscript{89} The clinician must also take into account the retentiveness of the food, protective elements in food (e.g. fluoride, calcium, phosphate), the frequency of meals and snacks, sugar-containing non-foods (e.g. lozenges, gum, medications), and patterns of consumption (e.g. sipping sugared drinks over a long period of time).\textsuperscript{2,9} Patients are typically asked to fill out a 24-hour diet diary and may be requested to complete an additional dietary record of up to a week if the practitioner believes the patient to be at high risk for dental caries.

Assessing diet alone is usually inadequate at predicting caries. Studies in humans have not found a consistent relationship between consumption of cariogenic foods and dental caries experience.\textsuperscript{29,48} In a longitudinal study by Burt \textit{et al.}, the between meal sugar consumption was found to be only marginally related to interproximal caries increment but not at all related to caries in the pits and fissures.\textsuperscript{90}

Dental caries is a multifactorial disease and thus caries risk is not always directly correlated to fermentable carbohydrate consumption. For example, it was found that children developed very few caries if they had good oral hygiene irrespective of their dietary intake but if oral hygiene was poor, a high sugar intake revealed an increase in caries prevalence.\textsuperscript{91} It is thus more prudent to consider dietary factors in association with other caries factors such as oral hygiene practices and fluoride exposure.
**Previous Caries Experience**

Without a doubt, previous caries experience remains the most powerful single predictor of future caries development.\(^{19,31,52}\) It is the most common risk indicator used by dentists in both clinical practice and in CRA research as it provides the strongest predictive ability.\(^{26,47,51,59,75}\) Studies of children and adolescents show that individuals developing carious lesions early in life tend to develop more caries in the coming years.\(^{53,61,70,92,93}\) This tendency has also been demonstrated with an increase in caries risk among children whose mothers have caries and adults are more likely to develop root caries if they have existing coronal caries.\(^{9,94}\)

Previous caries experience is often used in prediction models as it is fast, simple and inexpensive to record. However, it cannot specify the particular risk factors that are causing the dental caries and, therefore, it cannot be used alone to specify appropriate preventive strategies directed at eliminating or modifying the patient’s risk for caries development.\(^{56,61}\) Documenting caries experience over the past one to two years, and current disease activity, tends to be more indicative of the patient’s true caries risk level. It has been shown that short-term predictions (i.e. less than 2 years) are more reliable than long-term predictions of risk (i.e. more than 5 years). Dental caries develop under precise oral conditions and these circumstances are more likely to change during studies of longer duration due to lifestyle or behavioural modifications.\(^{70}\)

**Sociodemographic Indicators**

Some researchers take the patient’s age into account when assessing caries risk as teeth are exposed to different levels of the oral environment at various stages throughout life. For example, it has been shown that newly erupted teeth are more susceptible to development of
carious lesions due to their limited exposure to topical fluoride.\textsuperscript{2} Dental caries used to be considered a disease of childhood. This belief arose when the prevalence of caries was much higher and when few children reached adulthood caries-free. This is no longer the case and the caries disease process is spread out more throughout life.\textsuperscript{81} Adults of all ages still develop coronal caries and thus dental caries must now be considered a lifetime disease.\textsuperscript{76} Currently age, as a risk indicator, is considered to be less critical in the prediction of caries. The interaction of the principal risk factors (diet, bacteria, and host factors) takes precedence over an individual’s age for appropriately categorizing caries risk level.\textsuperscript{2,19,70}

Some investigators use other demographic risk indicators in their caries risk models, such as gender. Women, in both childhood and adulthood, tend to present with higher DMF (Decayed, Missing, Filled) scores than men. However, females generally tend to have superior oral hygiene and fewer missing teeth than males. Therefore, it is unlikely that women have higher caries susceptibility than men, but rather it is a combination of seeking out more dental care and/or earlier tooth eruption in the case of children and adolescents.\textsuperscript{2,81}

A person’s medical status is an indicator that incorporates some of the same elements as a few other risk factors, such as bacterial load and salivary characteristics. Xerostomia and lack of physical or cognitive abilities will alter the saliva and bacterial counts in an individual’s oral cavity, especially in the elderly and special needs patients, and thus resulting in a higher risk for caries activity. Medical status is not often formally assessed because it is indirectly considered by its effect on stronger predicting etiologic risk factors such as salivary flow rate.\textsuperscript{12,83}
Socioeconomic status (SES) is a broad measure of individual or family’s relative economic and social ranking with regards to factors such as income, education, and occupation.\textsuperscript{81} A large number of reports over the past few decades have demonstrated that social and behavioural factors are associated with dental caries and some studies have specifically indicated that dental caries can now be regarded as a disease of poverty.\textsuperscript{95-97} The sharpest decline in caries prevalence has been in the upper SES groups, while reductions in disease rates in lower SES groups have been much more modest.\textsuperscript{48}

There is much discussion on whether or not to include socioeconomic variables in CRA instruments. Certainly these indicators will often select for high risk individuals as they will be more inclined to develop higher levels of tooth decay than people living under less extreme conditions.\textsuperscript{98} But much like medical status, they do not indicate which risk factors are responsible for the development of disease and they are often indirectly considered with stronger etiological factors.\textsuperscript{61} There is little doubt that dietary and health practices are affected by education, income and environment;\textsuperscript{29} however, bacterial levels and cariogenic diet are normally already considered to be direct causes of caries and are regularly assessed. It, therefore, may be redundant to assess both socioeconomic variables and biological factors in the same CRA model.

**Overall**

It was concluded at the risk assessment conference at the University of North Carolina that clinical variables were stronger predictors of dental caries than non-clinical variables. Past experience of caries activity was the most significant indicator of future caries development, along with fluoride exposure, microbial agents, tooth morphology, and socioeconomic status.\textsuperscript{70}
Given that dental caries is a multifactorial disease, it only makes sense to use multiple predictors in order to accurately predict risk for the disease. It has been established that analyses applying a combination of biological and social factors have shown better results than any single risk factor studies. Work must be continued in this area to determine which caries risk factors and indicators are most effective for defined populations.

**Multifactorial Caries Risk Assessment Models**

It has been noted by a couple of researchers that the majority of CRA studies have been conducted in children and adolescent populations. There are relatively few studies involving adult subjects and those that do exist focus mainly on older adults (aged 50+ years) and the development of root caries. We are only just beginning to see studies that investigate general caries activity in populations that include younger adults. This is promising because the younger adult population may express different disease factors due to lifestyle changes they encounter early into adulthood, such as living away from home for the first time and changes in dental care utilization and insurance patterns.

The following are two examples of multifactorial caries risk assessment instruments. Firstly, the Cariogram is a widely available tool that has been validated and has received much attention in the discipline of cariology. It has been used extensively to identify caries risk factors for a variety of populations globally. Secondly, the caries risk assessment form (Caries Risk and Preventive Needs Assessment) from the Faculty of Dentistry at the University of Toronto is a university-developed model utilized by the students in the dental school clinic.
**Cariogram**

The Cariogram, developed in 1996, was originally conceived as an educational model aiming to demonstrate the multifactorial etiology of dental caries in a simple manner. It is a graphical picture illustrating the interactions of caries-related factors and the overall risk profile of the patient. In the beginning, the pie chart presentation included three components: diet, bacteria and susceptibility. Based on this model, an interactive computer program was developed in 1997. Changes made to the program included the addition of two more sections to the pie chart—‘circumstances’ and ‘chance of avoiding caries’. The circumstances sector included factors that did not participate directly in the development of caries but were risk predictors of dental caries, such as past caries experience and systemic diseases.

The program prompts the clinician to enter a weight (0 to 3, with ‘0’ representing a low risk and ‘3’ representing a high risk) for nine risk factors (caries experience, related general diseases, dietary contents, dietary frequency, plaque amount, *Streptococcus mutans*, fluoride, saliva secretion, and saliva buffering capacity) and a clinical judgement score. An algorithm was constructed such that all the factors entered into the model could be weighed and the patient’s chance of avoiding caries could be calculated. This was represented as the final pie piece in the diagram. With this interactive program, it is possible to demonstrate to the patient how their caries risk can change as a result of various actions. Additionally, the patient’s risk profile can be saved or printed and the program offers recommendations for preventive measures that should be adopted to avoid new caries activity.
‘Caries Risk and Preventive Needs Assessment’ Instrument

The Faculty of Dentistry at the University of Toronto developed its own caries risk assessment instrument (Caries Risk and Preventive Needs Assessment) and implemented it into their school-based dental clinic in 1996 (Appendix). It was introduced to the undergraduate students as a preventive dentistry assignment to help students focus their attention on their patients’ caries-preventive needs. The CRA form was intended to provide a guide for students to consider the various factors that may influence caries risk, as well as the preventive measures available to reduce the risk of disease.55

There are five sections of the form:

1. Basic patient data- includes information regarding medical conditions, disabilities, and oral appliances that may influence the patient’s overall caries risk.

2. Basic caries risk factors- eleven factors (e.g. past caries and oral hygiene) are assessed and given a score (‘0’ for low, ‘1’ for moderate, or ‘2’ for high risk). A total risk score is calculated by summing the scores of the eleven risk factors.

3. Caries preventive factors- provides an inventory of the preventive practices routinely used by the patient.

4. Supplementary caries susceptibility tests- provides suggestions for additional diagnostic tests and procedures that could be used to investigate the patient’s caries risk level.

5. Assessment summary- provides space to summarize the patient’s risk factors and to propose preventive treatment.
The risk factors selected for assessment in this instrument were obtained from the literature. Although this CRA tool has been used for more than a decade, it has never been evaluated. It is unknown whether the instrument can accurately assess caries risk level and which risk factors and/or indicators are the best predictors of caries activity in this population.
Statement of the Problem

Although dental caries has long been established to be a disease of multifactorial etiology, many of the traditional caries prediction models have focused on individual factors associated with high caries activity. More recently, the multifactorial etiology of caries points in the direction of constructing a more promising risk assessment model that includes the various factors that contribute to the development of caries as no single test can simultaneously measure the three principal components (host, bacteria, and diet) of dental caries. The Faculty of Dentistry at the University of Toronto has developed and implemented their own caries risk assessment form to assess patients’ risk for development of caries and to determine their oral health preventive needs. This multifactorial model, however, has never been evaluated to determine if it can accurately predict caries risk levels or if the overall risk score predicts future dental caries more accurately than any single factor measured.

Objectives

1. To determine if the caries risk assessment model (‘Caries Risk and Preventive Needs Assessment’) could correctly assign the appropriate risk categorization to adult patients.
2. To determine if caries increment differed across various degrees of risk for each basic caries risk factor and the overall risk score.
3. To determine which risk factors are the strongest predictors of future caries development.

Hypothesis

A caries risk assessment model that evaluates multiple factors simultaneously is able to predict caries increment more accurately than a single factor.
Methodology

Study Design
This research paper was designed as a quality assurance study. It consisted of secondary data analyses of measures collected from a chart audit of patient records from the Faculty of Dentistry, University of Toronto. The purpose of the study was to examine disease activity and risk factors for dental caries at baseline (i.e. when patients were first admitted to the Faculty’s dental clinic) and subsequent dental caries experience at follow-up (i.e. during recall appointments or after years of treatment). Study approval was obtained from the University of Toronto’s Research Ethics Board.

Eligibility Criteria
To be eligible for study participation, the subject in the patient chart had to meet the following criteria: 18 years of age or older at baseline, dentate, and have had a caries risk assessment completed at the University of Toronto dental clinic. Subjects must also have returned to the dental clinic for an oral health re-evaluation (e.g. recall or oral diagnosis examination) in which dental caries was re-assessed.

Sample Size
Using a computer-generated sample size calculator (with a significance level of 0.05 and power of 80%), it was determined that a sample size of 375 patients was required. A list of potential candidates was derived from axiUm, a dental computer software program, using the dental recall code (01202) to ensure that patients had returned to the clinic for a re-assessment of dental caries. Any repetitions of names in the list (i.e. those patients that had returned for multiple recall
appointments) were removed from the list to equalize the opportunity for all patients in the list to be randomly selected for the study. This produced a directory of nearly 5900 potential patients, each with a unique number. A computer random integer generator was utilized to select the patients. These patient files were inspected electronically via axiUm to ensure that a complete exam (including a caries risk assessment) was performed and that a recall examination was subsequently completed.

The final study population consisted of 434 individuals; however, only 186 of these comprised data from complete caries risk assessment forms. The remainder of the sample included all the necessary data except two of the caries risk factors, *Streptococcus mutans* and *Lactobacillus* counts. Customarily, microbial tests are often not completed due to patient refusal and/or the students are told (at the time of the assessment) that they do not have to do the bacterial tests on each patient unless they consider the patient to be at high risk or that it would make a great teaching tool to help improve the patient's home care.

**Data Collection**

Secondary data analysis included a chart review of active and inactive University of Toronto adult dental patients. All information was previously recorded by dental students and was extracted from the patient’s paper chart housed at the Faculty of Dentistry, University of Toronto or from the patient’s electronic chart, accessed through axiUm. Scores from the eleven ‘Basic Caries Risk Factors’ and the patient’s ‘Total Risk Score’ were extracted from the university-developed caries risk assessment form, Caries Risk and Preventive Needs Assessment (Appendix). The various caries risk factors are given an individual score according to
predetermined scales (‘0’ for low risk, ‘1’ for moderate risk, and ‘2’ for high risk). The manner in which the student selects the appropriate level of risk is described for each factor in Table 1.

The overall caries risk score is obtained through the addition of the individual scores for each variable. Depending on the ‘Total Risk Score’ of the eleven criteria, the patient’s caries risk falls into one of three categories: low (0-3), moderate (4-6), or high (≥7) caries risk.

### Table 1. Caries-related factors from the ‘Caries Risk and Preventive Needs Assessment’ form

<table>
<thead>
<tr>
<th>Factor</th>
<th>Information and data collected</th>
<th>Score&lt;sup&gt;a&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Caries Activity</strong></td>
<td>Present caries activity at baseline, including coronal, root and incipient caries; data from clinical examination and radiographs</td>
<td>0: None</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1: One</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2: Two or more</td>
</tr>
<tr>
<td><strong>Past Caries (DMFS)</strong></td>
<td>Past caries experience at baseline, including fillings and missing teeth due to caries; data from clinical examination and radiographs</td>
<td>0: None or pit and fissure only</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1: Proximal</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2: Free, smooth surface or lower anterior</td>
</tr>
<tr>
<td><strong>Root Caries Index</strong></td>
<td>Number of teeth with carious roots (decayed or filled) divided by number of teeth with exposed roots</td>
<td>0: 0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1: Intermediate</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2: ≥0.3</td>
</tr>
<tr>
<td><strong>Usual Fluoride Exposure</strong></td>
<td>Estimation of the number of fluoride sources to which the patient is exposed</td>
<td>0: ≥2/day</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1: 1/day</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2: &lt;1/day</td>
</tr>
<tr>
<td><strong>Carbohydrate Between Meals Frequency</strong></td>
<td>Estimation of the number of carbohydrates consumed between meals</td>
<td>0: &lt;1/day</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1: 2/day</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2: ≥3/day</td>
</tr>
<tr>
<td><strong>Oral Hygiene</strong></td>
<td>Clinical estimation of oral hygiene by visual inspection only</td>
<td>0: Good</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1: Fair</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2: Poor</td>
</tr>
<tr>
<td><strong>Modified Gingival Bleeding Index</strong></td>
<td>Total score from all proximal sites that gave a response to Stimudent probing. 0=no symptoms; 1=inflammation and no bleeding; 2=inflammation and slight bleeding; 3=inflammation and profuse bleeding</td>
<td>0: 0-4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1: 5-8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2: ≥9</td>
</tr>
<tr>
<td><strong>Stimulated Salivary Flow (mL/min)</strong></td>
<td>Estimation of salivary secretion rate (paraffin-stimulated saliva)</td>
<td>0: ≥0.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1: Intermediate</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2: ≤0.5</td>
</tr>
<tr>
<td><strong>Resting Salivary</strong></td>
<td>Estimation of salivary secretion rate at</td>
<td>0: ≥0.3</td>
</tr>
<tr>
<td>Flow (mL/min)</td>
<td>rest</td>
<td>1: Intermediate</td>
</tr>
<tr>
<td>-----------------------</td>
<td>------------</td>
<td>-----------------</td>
</tr>
<tr>
<td><strong>Mutans streptococci (CFU/mL)</strong></td>
<td>Estimation of levels of Mutans streptococci in saliva</td>
<td>0: ≤1×10^5</td>
</tr>
<tr>
<td><strong>Lactobacilli (CFU/mL)</strong></td>
<td>Estimation of levels of lactobacilli in saliva</td>
<td>0: ≤1×10^4</td>
</tr>
<tr>
<td><strong>Total Risk Score</strong></td>
<td>Addition of all above scores</td>
<td>0-3: Low caries risk</td>
</tr>
</tbody>
</table>

* A score of ‘0’ denotes a low risk for caries, ‘1’ for moderate risk, and ‘2’ for high risk.

The patients’ caries increments, as identified from paper charts, electronic records, and radiographs, were estimated for the time interval extending from the initial oral diagnosis appointment to the recall examination. Only dentinal caries (i.e. radiolucency that has broken through the dentin-enamel junction) were recorded as a carious lesion. In addition, the patient’s age and gender were collected for the logistic regression analysis.

**Data Analysis**

Statistical analyses for associations between individual caries risk factors and the ‘Total Risk Score’ and caries increment were performed. Statistical tests were two-tailed and the level of significance was set at \( p < 0.05 \). Data were analyzed using SPSS, version 17.0 (SPSS, Chicago, IL, USA). Three levels of analyses included: 1) univariate description of variables using percent frequencies for risk factors and mean ± standard deviation for caries increments; 2) bivariate associations between the eleven ‘Basic Caries Risk Factors’ and the ‘Total Risk Score’ and caries increment using one-way ANOVA analyses, with Tukey’s HSD post-hoc test; and 3) multivariate analysis using logistic regression (Forward: Wald), in which all the significant
variables from the bivariate analysis, as well as gender and age, were entered into the model. Age was divided into three groups (18-39; 40-59; 60+ years) for logistic regression. The low risk group of each variable, the female gender and the 60+ age group were used as references in the logistic regression analysis.

In addition, the sensitivity (Se), specificity (Sp), positive predictive value (PPV) and negative predictive value (NPV) of the caries risk assessment tool were calculated using the ‘Total Risk Score’ as the predictor variable for the development of caries. Because there were three risk levels, Se, Sp, PPV, and NPV were calculated at two different cut-off points- one combined the low and moderate risk groups, while the other combined the moderate and high risk groups.
Results

The frequency of subjects within the three caries risk categories, descriptive sample characteristics (age, gender, recall interval), and caries increments (by tooth) are presented in Table 2. Approximately one-fifth of the participants were classified as low risk for future development of dental caries. The remainder of the subjects were fairly equally distributed in the moderate (38.2%) and high (40.6%) risk groups. The sample population consisted of fewer males (43.1%) than females and the mean age of the sample was 58.7±15.2 years of age, with the age of participants ranging from 24 to 95 years. There was much consistency across these two factors (i.e. age and gender) in the moderate and high risk groups. The recall period, which is defined as the time from the initial caries risk assessment to the follow-up appointment at which caries diagnosis was performed, was collected from a random sample of 60 charts. The recall interval ranged from 24.80±6.38 to 27.95±5.26 months across the risk groups but the difference was not statistically significant.

Table 2. Caries risk frequencies, sample characteristics and mean caries increments ± standard deviation (SD)

<table>
<thead>
<tr>
<th>Risk Classification</th>
<th>Low</th>
<th>Moderate</th>
<th>High</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of individuals (%)</td>
<td>92 (21.2)</td>
<td>166 (38.2)</td>
<td>176 (40.6)</td>
<td>434 (100)</td>
</tr>
<tr>
<td>No. of males (%)</td>
<td>29 (31.5)</td>
<td>76 (45.8)</td>
<td>82 (46.6)</td>
<td>187 (43.1)</td>
</tr>
<tr>
<td>Mean age (years)±SD</td>
<td>62.3±13.1</td>
<td>57.4±14.7</td>
<td>58.2±16.3</td>
<td>58.7±15.2</td>
</tr>
<tr>
<td>Mean recall interval±SD (months)</td>
<td>27.95±5.26</td>
<td>24.80±6.38</td>
<td>26.85±4.87</td>
<td>26.53±5.93</td>
</tr>
<tr>
<td>Mean caries increment±SD (tooth)</td>
<td>0.20±0.80</td>
<td>1.13±1.59</td>
<td>2.09±2.63</td>
<td>1.32±2.10</td>
</tr>
</tbody>
</table>

*a Based on a random sample of 60 charts (20 from each caries risk group)
The number of carious teeth diagnosed at the recall appointment ranged from zero to 17 across the sample. The mean caries increment noted from the time of the initial examination to the recall visit was 1.32±2.10 for the total sample. A trend could be noted in which caries increment increased with elevated risk classification (0.20±0.80 for low risk, 1.13±1.59 for moderate risk, and 2.09±2.63 for high risk).

The participants’ caries experience, expressed as caries incidence from the initial examination to subsequent follow-up examination, is displayed in Figure 1. Nearly half of the sample (49.3%) had developed new carious lesions. Of the individuals deemed at a low risk for future caries activity by the risk assessment form, only 8.7% had new caries. Caries experience increased significantly in the other risk categories with 51.8% of moderate risk and 68.2% of high risk participants presenting with new caries at their recall examination. The trend of increased caries experience with elevated risk classification was statistically significant ($p<0.001$).
Figure 1. Caries incidence by risk classification as designated by the caries risk assessment form

The frequencies of each level (low, moderate, and high) of the various caries risk factors experienced by the subjects are listed in Table 3. The most prevalent descriptors of this population were: 61.3% experienced two or more carious lesions at the time of the initial examination; 75.3% had proximal carious lesions in the past; 84.8% had no root caries; 77.4% were exposed to two or more sources of fluoride daily; 39% consumed less than one carbohydrate snack between meals; 56.5% had fair oral hygiene and 53% had little to no gingival bleeding. In addition, the majority of the sample had low level bacterial counts- 68.8% of the population for *Streptococcus mutans* and 65% of the population for *Lactobacillus*; and stimulated and resting salivary rates were normal for 86.6% and 91.9% of the population, respectively.
Table 3. ANOVA analysis of various caries risk factors and ‘Total Risk Score’ versus mean caries increment

<table>
<thead>
<tr>
<th>Factor (Score)</th>
<th>Prevalence N (%)</th>
<th>Caries Increment (tooth) ± SD</th>
<th>Significant Differencesa</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present Caries</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None (0)</td>
<td>85 (19.6)</td>
<td>0.55±1.23</td>
<td>0 vs. 2; ( p&lt;0.001 )</td>
</tr>
<tr>
<td>One (1)</td>
<td>83 (19.1)</td>
<td>0.80±1.40</td>
<td>1 vs. 2; ( p=0.001 )</td>
</tr>
<tr>
<td>Two or more (2)</td>
<td>266 (61.3)</td>
<td>1.73±2.38</td>
<td></td>
</tr>
<tr>
<td>Past Caries</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None or pit and fissure only (0)</td>
<td>29 (6.7)</td>
<td>0.31±0.60</td>
<td>0 vs. 2; ( p&lt;0.001 )</td>
</tr>
<tr>
<td>Proximal (1)</td>
<td>327 (75.3)</td>
<td>1.24±1.83</td>
<td>1 vs. 2; ( p=0.005 )</td>
</tr>
<tr>
<td>Free, smooth or lower anterior (2)</td>
<td>78 (18.0)</td>
<td>2.05±3.08</td>
<td></td>
</tr>
<tr>
<td>Root Caries Index</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 (0)</td>
<td>368 (84.8)</td>
<td>1.17±1.76</td>
<td>0 vs. 1; ( p=0.014 )</td>
</tr>
<tr>
<td>Intermediate (1)</td>
<td>52 (12.0)</td>
<td>2.04±2.84</td>
<td>0 vs. 2; ( p=0.050 )</td>
</tr>
<tr>
<td>≥0.3 (2)</td>
<td>14 (3.2)</td>
<td>2.50±4.83</td>
<td></td>
</tr>
<tr>
<td>Usual Fluoride Exposure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥2/day (0)</td>
<td>336 (77.4)</td>
<td>1.19±1.80</td>
<td>No significant differences between groups</td>
</tr>
<tr>
<td>1/day (1)</td>
<td>77 (17.8)</td>
<td>1.64±2.70</td>
<td></td>
</tr>
<tr>
<td>&lt;1/day (2)</td>
<td>21 (4.8)</td>
<td>2.19±3.49</td>
<td></td>
</tr>
<tr>
<td>Carbohydrate Between Meals Frequency</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1/day (0)</td>
<td>169 (39.0)</td>
<td>1.07±1.78</td>
<td>0 vs. 2; ( p=0.005 )</td>
</tr>
<tr>
<td>2/day (1)</td>
<td>133 (30.6)</td>
<td>1.15±1.84</td>
<td>1 vs. 2; ( p=0.025 )</td>
</tr>
<tr>
<td>≥3/day (2)</td>
<td>132 (30.4)</td>
<td>1.82±2.59</td>
<td></td>
</tr>
<tr>
<td>Oral Hygiene</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Good (0)</td>
<td>113 (26.0)</td>
<td>0.65±1.26</td>
<td>0 vs. 1; ( p=0.002 )</td>
</tr>
<tr>
<td>Fair (1)</td>
<td>245 (56.5)</td>
<td>1.44±2.20</td>
<td>0 vs. 2; ( p&lt;0.001 )</td>
</tr>
<tr>
<td>Poor (2)</td>
<td>76 (17.5)</td>
<td>1.92±2.51</td>
<td></td>
</tr>
<tr>
<td>Modified Gingival Bleeding Index</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-4 (0)</td>
<td>230 (53.0)</td>
<td>0.93±1.80</td>
<td>0 vs. 1; ( p=0.017 )</td>
</tr>
<tr>
<td>5-8 (1)</td>
<td>146 (33.6)</td>
<td>1.52±1.88</td>
<td>0 vs. 2; ( p&lt;0.001 )</td>
</tr>
<tr>
<td>&gt;9 (2)</td>
<td>58 (13.4)</td>
<td>2.38±3.10</td>
<td>1 vs. 2; ( p=0.019 )</td>
</tr>
<tr>
<td>Stimulated Salivary Flow (mL/min)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥0.7 (0)</td>
<td>376 (86.6)</td>
<td>1.17±1.76</td>
<td>0 vs. 1; ( p=0.001 )</td>
</tr>
<tr>
<td>Intermediate (1)</td>
<td>42 (9.7)</td>
<td>2.43±3.26</td>
<td></td>
</tr>
<tr>
<td>≤0.5 (2)</td>
<td>16 (3.7)</td>
<td>2.00±4.02</td>
<td></td>
</tr>
<tr>
<td>Resting Salivary Flow (mL/min)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥0.3 (0)</td>
<td>399 (91.9)</td>
<td>1.18±1.79</td>
<td>0 vs. 1; ( p&lt;0.001 )</td>
</tr>
<tr>
<td>Intermediate (1)</td>
<td>28 (6.5)</td>
<td>3.36±4.25</td>
<td>1 vs. 2; ( p=0.028 )</td>
</tr>
<tr>
<td>≤0.1 (2)</td>
<td>7 (1.6)</td>
<td>1.14±1.68</td>
<td></td>
</tr>
<tr>
<td>Mutans streptococci (CFU/mL)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤1x10^5 (0)</td>
<td>128 (68.8)</td>
<td>1.20±1.82</td>
<td>0 vs. 2; ( p=0.001 )</td>
</tr>
<tr>
<td>Intermediate (1)</td>
<td>39 (21.0)</td>
<td>2.13±3.21</td>
<td></td>
</tr>
<tr>
<td>≥1x10^6 (2)</td>
<td>19 (10.2)</td>
<td>3.42±4.35</td>
<td></td>
</tr>
</tbody>
</table>
Utilizing one-way ANOVA analyses, the mean caries increments of the risk categories were compared across the eleven ‘Basic Caries Risk Factors’ and the ‘Total Risk Score’ recorded at baseline. The results of this analysis are presented in Table 3. A post-hoc test, Tukey-HSD, was conducted to determine between which groups statistically significant differences lied. All factors except fluoride exposure displayed statistically significant results between at least two of the caries risk classifications. The most common difference found was between those in the low risk group as compared to the high risk group. All variables, with the exception of fluoride exposure, stimulated and resting salivary flow, showed statistically significant differences between the low and high risk categories. Only two factors, Modified Gingival Bleeding Index (MGBI) and the ‘Total Risk Score’ exhibited differences between all risk groups.

The adjusted odds ratios for the three caries risk classifications are illustrated in Table 4. The adjusted odds ratios were calculated twice. Because not all of the participants had *Streptococcus mutans* and *Lactobacillus* counts done during their caries risk assessment appointment, there were two possible scenarios. The first was the full sample (N=434) in which the microbial counts were excluded from the analysis and the second was a partial sample (N=186) in which only
those individuals with complete caries risk assessments (i.e. obtained microbial counts) were included in the analysis. Although the magnitude of the figures differed slightly, the trend of increased odds of developing new caries with an elevated risk categorization persisted across both samples. For example, in the full sample analysis, patients in the moderate risk group were 12 times more likely to develop caries than individuals in the low risk group. The high risk group had 25 times greater risk of developing caries than the lowest risk group. All findings were highly statistically significant.

**Table 4.** Adjusted odds ratios (OR) for the various caries risk groups in the logistic regression model

<table>
<thead>
<tr>
<th>Risk Category</th>
<th>Full sample (N=434)</th>
<th>Partial sample (N=186)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Adjusted OR</td>
<td>95% CI</td>
</tr>
<tr>
<td>Low</td>
<td>1 (reference)</td>
<td>-</td>
</tr>
<tr>
<td>Moderate</td>
<td>12.04</td>
<td>5.40-26.85</td>
</tr>
<tr>
<td>High</td>
<td>25.44</td>
<td>10.87-59.53</td>
</tr>
</tbody>
</table>

* a 95% Confidence Interval

Logistic regression analysis was carried out to determine which variables were the strongest predictors of caries activity when all factors were considered simultaneously. Age and gender were also added to the model to ensure they were not confounding factors. The ‘Total Risk Score’ ($p<0.001$) and between meals carbohydrate frequency ($p=0.019$) were the only significant factors associated with the presence of caries. Because the overall risk score was by far the most powerful predictor of future caries development, the model was run again, excluding the ‘Total Risk Score’, to see what other variables were associated with caries activity. The results of this
logistic regression analysis are presented in Table 5. Other factors that proved to be significantly associated (i.e. \( p<0.05 \)) with caries increment included present caries activity, past caries experience, carbohydrate frequency, MGBI, and stimulated salivary flow.

Table 5. Adjusted odds ratios (OR) for various factors when all independent variables except the ‘Total Risk Score’ were included in the logistic regression model

<table>
<thead>
<tr>
<th>Risk Category</th>
<th>Adjusted OR</th>
<th>95% CI (^a)</th>
<th>( p ) value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Present Caries</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>1 (reference)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>One</td>
<td>2.42</td>
<td>1.19-4.90</td>
<td>0.014</td>
</tr>
<tr>
<td>Two or more</td>
<td>3.42</td>
<td>1.88-6.23</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Past Caries</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None or pit and fissure only</td>
<td>1 (reference)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Free, smooth, or lower anterior</td>
<td>5.93</td>
<td>2.07-16.98</td>
<td>0.001</td>
</tr>
<tr>
<td><strong>Carbohydrate Between Meals Frequency</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1/day</td>
<td>1 (reference)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>( \geq 3 )/day</td>
<td>1.76</td>
<td>1.05-2.94</td>
<td>0.032</td>
</tr>
<tr>
<td><strong>Modified Gingival Bleeding Index</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-4</td>
<td>1 (reference)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>5-8</td>
<td>2.71</td>
<td>1.69-4.35</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>&gt;9</td>
<td>2.34</td>
<td>1.21-4.51</td>
<td>0.011</td>
</tr>
<tr>
<td><strong>Stimulated Salivary Flow (mL/min)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( \geq 0.7 )</td>
<td>1 (reference)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>( \leq 0.7/ \geq 0.5 )</td>
<td>3.48</td>
<td>1.55-7.82</td>
<td>0.003</td>
</tr>
</tbody>
</table>

\(^a\) 95% Confidence Interval

Sensitivity (Se), specificity (Sp), positive predictive value (PPV), and negative predictive value (NPV) of the caries risk assessment tool were compared for two cut-off points in Table 6. When the moderate risk group was combined with the high risk group rather than the low risk group, Se and NPV both increased significantly (Se 56 to 96% and NPV 63.6 to 91.3%). Conversely, Sp and PPV both decreased, 75 to 38% and 68.2 to 55.4%, respectively.
Table 6. Comparison of sensitivity (Se), specificity (Sp), positive predictive (PPV) and negative predictive (NPV) values of the caries risk assessment model using two different cut-off points

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Caries</th>
<th>No Caries</th>
<th>Se (%)</th>
<th>Sp (%)</th>
<th>PPV (%)</th>
<th>NPV (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>High vs. Moderate/Low</td>
<td>120</td>
<td>94</td>
<td>56</td>
<td>75</td>
<td>68.2</td>
<td>63.6</td>
</tr>
<tr>
<td></td>
<td>56</td>
<td>164</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High/Moderate vs. Low</td>
<td>206</td>
<td>8</td>
<td>96</td>
<td>38</td>
<td>55.4</td>
<td>91.3</td>
</tr>
<tr>
<td></td>
<td>164</td>
<td>84</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Discussion

The University of Toronto’s multifactorial caries risk assessment (CRA) tool was able to accurately categorize an individual’s risk for future caries development, as demonstrated by two trends that emerged from the data. Firstly, as the caries risk elevated, the number of teeth affected by caries also increased. The number of carious teeth rose proportionately across the risk groups (0.20 for low, 1.13 for moderate and 2.09 for high risk groups). Secondly, as the caries risk rose, the number of individuals displaying caries at the follow-up exam also increased. Approximately 9% of the low risk subjects presented with caries at the recall exam, while nearly 70% of the high risk group exhibited new carious lesions. Predicting caries in the moderate risk group proved to be more challenging; just over half of the moderate risk group presented with dental decay at the follow-up appointment.

Defining the appropriate criteria to select moderate risk individuals represents the utmost diagnostic challenge as it is difficult to know where to set the cut-off point between risk categorizations. Often it can be rather straightforward to identify low risk and high risk individuals; however, it is much more complicated to recognize moderate risk individuals that may have exhibited little or no dental disease for long periods of time followed by a sudden burst of caries activity.36,37

All the risk factors, except usual fluoride exposure, were found to be significantly associated with caries increment in the bivariate analysis. This was not surprising as dental caries is a disease of multifactorial etiology and thus there were many elements related to the development of caries in this population. It should be noted, however, that the majority of the differences
were found solely between the high and low risk groups. Again, this demonstrates the ease in identifying low risk, low caries and high risk, high caries patients, while detecting a moderate risk for caries development proves to be a more complex task.

Usual fluoride exposure was the only variable that was not significantly associated with dental caries in this study. Typically, exposure to various sources of fluoride has been shown to be a protective factor and helps to prevent the formation or progression of caries development.\textsuperscript{118} However, it is becoming increasingly difficult to distinguish between various levels of fluoride exposure due to the “halo effect”, which is the impact of individuals indirectly benefitting from the anti-carious effects of fluoride through the consumption of processed foods and beverages that have been produced using fluoridated water.\textsuperscript{28}

The effect of fluoride exposure is a preventive factor that is best identified at the population level rather than at the individual level. An inverse relationship between caries experience and fluoride exposure has been established at the population level.\textsuperscript{29} Individuals living an area with fluoridated public water, such as Toronto, are all exposed to levels of fluoride that provide anti-caries protection. Supplementary sources of fluoride (e.g. toothpaste, mouthrinses) may not provide much additional benefit in the way of caries prevention if they consume fluoridated water and foods and beverages manufactured with fluoridated water. Because individuals from all risk groups may already be ingesting optimal caries-inhibitory levels of fluoride, no differences were found between caries increments for the various levels of fluoride exposure in this study.
The overall risk score from the CRA instrument, which is the summation of multiple risk factors, was by far the strongest predictor of future caries activity. This supports the current literature which suggests that due to the multifactorial etiology of dental caries, the identification of several risk factors would increase the probability of formulating an accurate risk profile for our patients. Multifactorial CRA tools are becoming more popular and their use more prevalent as they present an overall picture of the interaction of the multitude of caries risk factors.

The dated method of relying on a single factor to dictate the development of a multifactorial disease often would lead to inaccurate predictions. For example, while consumption of fermentable sugars certainly plays a key role in the development of caries, reliance on this single factor as a predictor for future development of caries would often be misleading. Many individuals remain caries-free despite a high intake of sugar (high risk behaviour), likely due to the interaction of several low risk behaviours, such as good oral hygiene habits and regular exposure to fluoride.

When all variables (except overall risk score) were considered in the logistic regression analysis, caries experience (past and present), carbohydrate frequency, MGBI, and stimulated salivary flow were significantly associated with caries activity. From the literature, it is clear that past caries experience remains the most powerful single predictor of future caries development. It is the most common risk indicator used in clinical practice and in research due to its strong predictive ability. Although caries experience is a powerful indicator of caries activity, it cannot specify the particular risk factors that are causing the dental caries and,
therefore, it cannot be used alone to specify appropriate preventive strategies directed at eliminating or modifying the patient’s risk for caries development.37,56,61

Dental caries occurs as a result of the interaction of a dietary substrate, microbial pathogens, and host factors. In this study, between meal carbohydrate frequency, which is a dietary indicator, and MGBI and stimulated salivary flow rate, both indicators of host susceptibility, were all significant factors in caries development. Contrarily, bacterial counts (i.e. *Streptococcus mutans* and *Lactobacillus*) were not found to be significantly associated with caries in this population. Historically, when studied in groups, caries experience has been found to be related to *Streptococcus mutans* counts; however, bacterial counts by themselves are poor predictors of caries activity at the individual level.81 In the current study, 51 patients had low levels of *Streptococcus mutans* and *Lactobacillus* at baseline but still presented with dental caries at follow-up. Similarly, ten patients had high counts of these microbial pathogens at their initial examination but were caries-free at their recall appointment. This indicates that bacterial counts alone cannot predict future caries experience very accurately and thus we must rely on the overall risk score (i.e. the interaction of multiple factors) to assess risk level. Additionally, some researchers have suggested that microbial tests are not cost-effective and contribute only marginally to the prediction of future dental caries development if other clinical and sociodemographic data are available.50,119

The reason for the lack of association between high salivary bacterial counts and future caries in this study may also have been due to insufficient power. Of the 434 charts reviewed, only 186 of the caries risk assessment forms had bacterial counts. The lack of bacterial counts is often due to
patient refusal (infrequent) or that students have chosen not to do them. It is not a mandatory part of the caries risk assessment and students may choose to do the procedure if they feel that their patient is high risk or that it could be used as a teaching tool to help improve their patient’s home care. Microbial counts can be an important element to consider when customizing a caries management plan that corresponds to the patient’s individual needs (e.g. prescribing an antimicrobial rinse such as chlorhexidine). This same issue has been observed in a U.S. university dental clinic.\textsuperscript{35} Using the CAMBRA risk assessment tool, students were instructed that bacterial assessments were indicated when a patient was classified as high risk. From an overall sample of 1218 charts, only 46 patients had \textit{Streptococcus mutans} and \textit{Lactobacillus} counts recorded. This was thought to be due to the extra charge incurred by patients for this service.

The calculation of sensitivity, specificity, positive and negative predictive values is the preferred method for evaluation of disease prediction models.\textsuperscript{39} Because the university’s risk assessment instrument classifies caries risk into three categories, 2 different cut-off points were possible. The moderate risk group was combined with either the low risk or high risk group. Intuitively, it makes more sense to combine the moderate and high risk groups as they would encompass the individuals requiring increased preventive care to avoid caries. When the moderate and high risk groups were amalgamated, the sensitivity (96\%) and negative predictive value (91.3\%) of the model were extremely high. This translates to a caries risk instrument that is able to detect disease (i.e. caries) when is truly present in the majority of cases and that it will be correct most of the time in predicting dental caries. Eriksen, in his review of multifactorial CRA’s, found that several factors considered together produced a higher sensitivity than each factor alone.\textsuperscript{72}
The combined sensitivity and specificity of the CRA (131 or 134) did not reach the gold standard of 160 as proposed by Fleiss and Kingman but few do.\textsuperscript{31} When that recommendation was originally made, it did not take into account the fact that errors relating to poor sensitivity have consequences that are entirely different from those of poor specificity.\textsuperscript{120} There is always a trade-off between sensitivity and specificity, and consequently positive and negative predictive values, in prediction models. When considering suitable prediction values, one must consider the disease in question as well as the consequence of misclassification of patients by the instrument. If one was administering this caries risk assessment tool on a population-wide basis (i.e. in public programs), a test providing higher specificity (i.e. fewer false positives) would be favoured. This would avoid the treatment of individuals that do not require preventive services and thus saving scarce resources for other needs.\textsuperscript{53,54}

On the other hand, it may stand that the goal of the risk assessment instrument would be to identify all those at risk for caries and thus the model would need to possess higher sensitivity, such as is the case with the CRA under current investigation. The disadvantage of this is that the number of false negatives would increase (i.e. those identified as high risk for caries but will not actually develop the disease). This would result in overtreatment in which patients receive preventive services that they do not necessarily need. For some procedures, patients may suffer an economical loss; however, the harm of providing preventive care is minimal and is therefore preferred to the threat of failing to identify those at risk for caries, ultimately resulting in needless dental treatment in the longterm.\textsuperscript{53}
Study Limitations

The majority of the limitations of the study relate to its design. The study sample included only patients that attended the university dental clinic (i.e. sample of convenience); therefore, they may not have been representative of the general population. Secondly, although the study was a retrospective analysis of caries that developed over time (with a prospective component in its design), it consisted of an analysis of secondary data collected from dental charts. Inherently, secondary data analysis suffers from many potential weaknesses, such as unwanted and unknown biases, the need to tailor research questions to fit the data, and the inability to manage the quality of the data. There was no control over what and how the data were collected which led to difficulties in finding a sufficient number of charts that possessed microbial counts. In addition, there is no guarantee that all the students consistently evaluated all the caries factors or diagnosed dental caries in the same fashion. This could only be overcome if the collection of data had been completed by a small number of calibrated investigators. Due to time constraints, data collection was not carried out in this manner.

In addition, the recall period varied for the patients. Due to the limited number of charts available for review, it was not possible to narrow a recall frequency, therefore, this period ranged from 6 months to 7 years. The recall period from a random sample of 60 charts (20 from each risk group) was collected to determine if the discrepancy in the recall intervals may have led to statistically significant differences between caries increments scores of the risk groups. From this limited sample, no statistically significant difference was found between the recall periods across the various caries risk groups. The interval ranged between 24.80±6.38 and
27.95±5.26 months, with an average of 26.53±5.93 months between the initial caries risk assessment and the follow-up appointment.

To overcome these difficulties, an ideal study would have consisted of a team of calibrated examiners collecting measurements of all the basic caries risk factors at baseline and an assessment of dental caries within a specified period of time. The recall period would ideally be two to three years after baseline factors were collected. This time period balances between two principles: dental caries formation versus lifestyle change. Dental caries is a slowly progressing disease that takes two years for an initial lesion to progress through the enamel and requires in the region of three to four years to extend into the dentin.\textsuperscript{121,122} Documenting past and current caries activity tends to be indicative of the patient’s true caries risk level. It has been shown that short-term predictions (i.e. less than 2 years) are more reliable than long-term predictions of risk (i.e. more than 5 years). Dental caries develop under precise oral conditions and these circumstances are more likely to change during studies of longer duration due to lifestyle or behavioural modifications.\textsuperscript{9,70}

**Research Implications**

In addition to the aforementioned ideal study, other research relating to the topic of caries risk assessment should be conducted. A study investigating what preventive care was administered once patients’ risk for caries was assessed, as well as whether or not the provision of preventive services decreased future caries experience. In general, more research regarding caries risk in the adult population, but not limited to the elderly, needs to be conducted. The majority of studies in this area have also restricted their analysis to the development of root caries in seniors.
While this research is certainly warranted, investigators need to branch out to look at caries risk factors and indicators in younger adults, especially in the context of lifestyle changes. For example, one could explore dental caries and the factors associated with young adults living away from home for the first time. Young adults may move away from home to go to school or work elsewhere which may lead to changes in dietary and oral hygiene habits, thus resulting in possible shifts in risk levels and dental disease patterns.  

\(^7,^{1,7}\)
CONCLUSIONS

1. The caries risk assessment model (‘Caries Risk and Preventive Needs Assessment’) appropriately categorizes an individual’s risk for future caries development as evidenced by increased caries activity as the risk level rises.

2. All factors except fluoride exposure displayed statistically significant differences between the caries increments of the various risk levels. The most common difference found was between those in the low risk group as compared to the high risk group.

3. Past and present caries experience, carbohydrate between meal frequency, mucogingival bleeding index, and stimulated salivary flow showed statistically significant associations with caries increment \((p<0.05)\) but the total risk score proved to be the most powerful predictor of future caries activity \((p<0.001)\).

This study has confirmed the hypothesis that while numerous risk factors were significantly associated with caries development, the multifactorial caries risk assessment model (i.e. the ‘Total Risk Score’) was a stronger predictor of future dental caries experience than any single caries risk factor. Utilization of a global risk score necessitates a “bigger picture” mentality and allows for a more thorough assessment of the various elements involved in this multifactorial disease. The University of Toronto’s caries risk assessment instrument has shown that it can aid clinicians to assign patients to the appropriate risk level. This model is simple but yet comprehensive as it incorporates elements of the three principal etiological factors of dental caries, namely dietary substrate, microbial pathogens, and host susceptibility. The risk factors
can be easily measured in a clinical setting and are issues that can easily be attended to through preventive care (e.g. dietary analysis, oral hygiene instruction). Finally, the caries risk assessment model serves as an educational tool for both students and patients as it measures and identifies risk factors that are pertinent to the individual patient’s oral health needs.
REFERENCES


Appendix
**Caries Risk and Preventive Needs Assessment**

### Conditions Which Will Likely Increase Caries Risk:

<table>
<thead>
<tr>
<th>Medical Condition Summary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical Disability?</td>
</tr>
<tr>
<td>Low Dental IQ?</td>
</tr>
<tr>
<td>Major Lifestyle Change?</td>
</tr>
<tr>
<td>Negative Attitude?</td>
</tr>
<tr>
<td>Workplace/Home Environment</td>
</tr>
<tr>
<td>Intra-oral Appliance Planned</td>
</tr>
</tbody>
</table>

### Rating of the Basic Caries Risk Factors:

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Rating</th>
<th>Patient Score</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral Hygiene</td>
<td>poor</td>
<td>fair</td>
<td>good</td>
</tr>
<tr>
<td>MGBI (modified gingival bleeding index)</td>
<td>&gt; 9</td>
<td>5-8</td>
<td>0.4</td>
</tr>
<tr>
<td>Salivary Tests</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stimulated Flow (SSF) ml/min</td>
<td>≤ 0.5</td>
<td>intermediate</td>
<td>≥ 0.7</td>
</tr>
<tr>
<td>Resting Flow (if SSF &lt; 0.7) ml/min</td>
<td>≤ 0.1</td>
<td>intermediate</td>
<td>≥ 0.3</td>
</tr>
<tr>
<td>Mutans streptococci CFU/ml</td>
<td>≥ 1x10^3</td>
<td>intermediate</td>
<td>≤ 1x10^3</td>
</tr>
<tr>
<td>Lactobacilli CFU/ml</td>
<td>≥ 1x10^3</td>
<td>intermediate</td>
<td>≤ 1x10^4</td>
</tr>
</tbody>
</table>

### Total Risk Score

**Patient's Caries Risk**

- High (≥ 7)
- Mod (4-6)
- Low (0-3)

---

I: divide # teeth with carious roots (decayed or filled) by # teeth with exposed roots

GBI: use Student test numbers collected from all facial interprox. locations (0=no symptoms: 1=inflammation and no bleeding; 2=inflammation and slight bleeding; 3=inflammation and profuse bleeding)

**Preventive Dentistry March 2002**
Detailed Description of Current Caries Preventive Practices

Current Preventive Practices: What tooth paste is currently used and how often? What, if any, other fluoridated products are used? What are the missed during OH? What technique of OH is used? How frequent is floss used? What professional preventive procedures are being carried out?

Supplementary Analyses/Tests that may be required

<table>
<thead>
<tr>
<th>TEST REQUIRED?</th>
<th>Dietary Analysis* (2-day)</th>
<th>Plaque Index (Loe &amp; Silness)</th>
<th>Salivary Buffering Capacity</th>
</tr>
</thead>
<tbody>
<tr>
<td>YES □ NO □</td>
<td></td>
<td>YES □ NO □</td>
<td></td>
</tr>
</tbody>
</table>

*Required when Dietary Questionnaire Score ≥2/day

Assessment Summary

<table>
<thead>
<tr>
<th>Patient’s Caries Risk: □ High □ Mod □ Low</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Main factors contributing to patient’s caries risk:</td>
</tr>
<tr>
<td>2. Potential impact of planned dental treatment on caries risk:</td>
</tr>
<tr>
<td>3. Details of the preventive measures needed beyond those already in place. (include concentrations)</td>
</tr>
<tr>
<td>4. Details of caries preventive follow-up planned.</td>
</tr>
</tbody>
</table>

Preventive Dentistry March 2002
DIETARY FREQUENCY QUESTIONNAIRE

Student_________________________________________ Date (d/m/y)______________________
Patient_________________________________________ Chart #______________________

Ask your patient each question "for a typical day" and enter responses below.
(Do not have patient fill this in)

How many times a day do you:

1. drink sugared tea or coffee between meals? □
2. drink regular pop, kool-aid, lemonade, fruit drinks sweetened with sugar, or natural fruit juice between meals? □
3. chew regular (sugar-containing) gum? □
4. eat mints, lozenges, candies, candy bars etc? □
5. eat sweetened baked goods (cookies, cake etc)? □
6. eat other snacks (not listed above) between meals? □
7. use sweet-tasting medicines? □

Total frequency per day

Enter above *Total on the appropriate line of Assessment of Caries Risk Factors & Preventive Practices form (Page 1)

8. Do you follow a special diet of any kind? (Specify.__________________________) Yes ☐ No ☐

9. What is your most common snack item? ____________________________________________

10. How often do you have a snack before going to bed? ___________/week

11. Do you normally brush with fluoride toothpaste after a bedtime snack? □ ☐

12. Do you sip a sweetened drink (coffee, tea, carbonated beverage) or eat a sugary snack over an extended period of time (45 minutes or more)? □ ☐

If Yes, name snack item(s)?
(consider this response when completing your Assessment Summary on the form Assessment of Caries Risk Factors & Preventive Practices - Page 2)

* If the total frequency of between meal snacks is 2 or more, then a more comprehensive dietary analysis is required. Have the patient complete a 3-day diet record. This will provide information on the quality of the patient's current diet, including the nature and distribution of between-meal snacks and it will help identify those patients who will benefit from a preventive diet counselling. A Qualitative Dietary Analysis is only carried out for these patients.

3-day Diet Record Form
☐ Given to Patient_________________________ ☐ Returned by Patient_________________________

Preventive Dentistry 8/02