Caries Risk Pyramid: A Practical Biological Approach to Caries Management by Risk Assessment

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One of the most significant advancements in dentistry during the past decade is the concept of “caries management by risk assessment” (1). There are many different ways to interpret and apply this to clinical practice, but its most relevant clinical implication is that active caries lesions should be managed as signs of an infection, rather than as localized defects of the dental tissues. The primary difference between the two types of management approach is that the first focuses on the identification and management of the etiological factors of the infection, whereas the latter involves mechanical or surgical correction of the defect. The advantage of the first approach is clear: as with any lesion of infectious origin, if the etiological factor is not identified and eliminated, the infection will continue and the lesions can recur. This approach can be relatively straightforward for the management of most infectious diseases that are caused by specific organisms. In the case of dental caries, however, the microbial etiology of the disease is not species-specific; rather the disease is the result of a disruption in the ecological balance of the dental biofilm (2, 3). Therefore, our goal should not be to eliminate any single organism, but instead to identify and modify the factors that may cause caries-promoting oral ecological shifts in individual patients (4).

A multitude of biological and psychosocial factors can directly or indirectly influence oral microbial ecology. Consequently, the most widely accepted approach for caries risk assessment involves multivariate statistical models that incorporate several caries risk factors. Invariably, the factor with the highest predictive value in these models is previous caries experience, and this limits their utility to dental professionals (5). Furthermore, the concept of using the existing level of disease to predict future disease is controversial with respect to its clinical utility. Another limitation of multivariate caries risk assessment models is the lack of simple, validated instruments for assessing complex psychosocial caries risk factors. Salivary levels of Streptococci mutans and overall acid production are the biological factors most commonly incorporated in caries risk assessment models. A number of tools for measuring these factors are commercially available, but they require long incubation periods and are reported to have low specificity. Thus, in spite of our improved understanding of the biological processes that lead to the development of dental caries, we still do not have an accurate instrument for assessing caries risk that would be suitable for use in everyday clinical dental practice or by non-dental health professionals (5, 6).

This paper proposes a novel approach for assessing caries risk based on emerging evidence (8-13), which indicates that effective monitoring of the acid: base physiology of plaque is needed to be included in caries risk assessment. Based on this evidence we are proposing a new concept, the Caries Risk Pyramid-CRP, which forms the foundation for this approach (Figure 1). The CRP model states that the etiological factors leading to the onset of dental caries, as well as the complex interactions among them, can be sequentially organized according to the level in which their influence is exerted, forming a pyramid. The tip of this pyramid (Level 4) consists of a single factor, the plaque pH, which ultimately controls the chemical phenomena that lead to caries formation (de-mineralization/re-mineralization) (7). The pH of the plaque is determined primarily by biochemical pathways that generate acid and alkali in the plaque, as well as by other potential buffering systems, and to a lesser and more transient extent by substances in the diet (Level 3). The expression and activity of these systems depends on biological factors, which include host-related factors (saliva, immune system), oral ecology and sugar availability (Level 2). The base of the pyramid (Level 1) consists of the more complex psychosocial, behavioral and genetic factors that determine the biological profile of an individual.

The hierarchical organization of the various caries risk factors in the CRP model is an innovative concept, which provides the foundation for its clinical implementation. Another innovative feature of this model is that it incorporates new risk factors that have not previously been well integrated into the existing multivariate models for caries risk assessment. These new factors are the abilities of dental plaque and saliva to generate alkali from endogenous substrates, such as urea and arginine (8-13). According to the CRP model, the ability to generate acids from

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sugars and the ability to generate alkali from endogenous sources in the dental plaque and/or in the saliva are two closely related factors, which summarize the combined effect of all other caries risk factors and are closely related to disease development (12-13). Therefore, if we develop simple, rapid methods to measure these abilities together, that could enhance the accuracy of a screening instrument for caries activity, and possibly for caries risk, without the need to evaluate simultaneously all the lower and more complex risk factors. Furthermore, if a biochemical risk factor is identified in a high risk individual, such as increased acid production or reduced alkali production, it could be linked using the CRP model to lower level biological or behavioral factors that could be more easily modified or corrected. These lower level factors are therefore more suitable targets for caries preventive approaches.

The CRP concept has the potential to shift our approach for caries risk assessment from the current complicated multivariate models to a new paradigm of just measuring acid/alkali production in plaque as the ultimate factors that determine caries activity and caries risk, and it may lead to better, more practical strategies for managing caries. This approach could be easily employed by allied health professionals and public health workers to help identify individuals who either have active caries or who are at an immediate risk to develop the disease. These individuals can then be referred to dentists for appropriate treatment. Alternatively, the CRP concept could be incorporated into existing multivariate caries risk assessment models together with other psychosocial and biological risk factors to produce a more accurate caries risk assessment tool.

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References