Oral and overall health: clearing up the confusion

Précis
For several years dental researchers have been studying and reporting on links between oral and overall health, but study evidence often seems to offer conflicting information. This article aims to clear up that confusion.


After many initial studies suggested a strong association between periodontal disease and preterm low birth weight deliveries,1 an evidence-based review of all available intervention studies concluded that any potential interaction between these two conditions was minimal at best.2 More recently, an American Heart Association (AHA) review similarly suggested that the link between periodontal disease and atherosclerotic vascular disease (ASVD) may not be as robust as originally thought.3 The AHA concluded that “there is no evidence that periodontal intervention prevents ASVD or modifies its outcomes”. While admittedly contradictory, association studies support a strong link between periodontal disease and many systemic diseases, while intervention studies do not support a systemic benefit from periodontal therapy. Conflicting evidence has the potential to create confusion among practising clinicians concerning how to incorporate systemic health considerations into existing dental protocols. The intent of this article is to clear up that confusion.

Within the profession, conflicting evidence on the relationship between oral and overall health seems to be fuelling a developing struggle between those who believe that what happens in the mouth can affect the rest of the body and those who conclude that the most recent research dispels any notion that periodontal disease has systemic ramifications.4 One side is aggressively incorporating systemic disease risk management into their dental practices and suggesting to patients that adhering to treatment recommendations may also improve overall health. Meanwhile, those advocating an evidence-based approach opine that without stronger evidence no such claims should be made. Both groups are missing the point.

Why there is no ‘proof’
It should be no surprise that meta-analyses reveal that short-term mechanical bacterial removal alone does not result in dramatic alterations in the course of associated systemic diseases. The heterogeneity of the reviewed studies is vast. Meaningful combination of studies with different inclusion criteria is impossible.5 Moreover, a single treatment consisting of scaling and root planing alone (the sole intervention in most of the reviewed studies) produces little or no lasting improvement in a patient’s periodontal health. Long-term clinical studies have clearly demonstrated that the regular and effective removal of bacterial biofilms on the teeth is necessary to arrest periodontitis.6 Self-care instruction and reinforcement, follow-up monitoring and additional response-driven treatment is the standard therapy for periodontal disease. No conclusion concerning the strength of a periodontal–systemic disease link can or should be made until studies are designed that incorporate a more comprehensive and long-term approach to managing periodontal disease.

Perhaps periodontal disease should be viewed more broadly in terms of systemic...
inflammation, either as a consequence of an underlying hyperinflammatory trait or as a factor contributing to systemic inflammation. While bacteria initiate periodontal disease, it is the host’s inflammatory response to those bacteria that results in tissue destruction and potential systemic ramifications.\textsuperscript{7} Inflammation-based dissolution of connective tissue and the resultant loss of integrity of lining epithelium in the periodontal tissues open a portal of entry for periodontal bacteria, bacterial byproducts and the inflammatory mediators released in response to the pathogenic bacteria to gain entrance into the bloodstream.\textsuperscript{8} Periodontal bacteria and inflammatory mediators have repeatedly been shown to play a role in systemic disease initiation and progression.\textsuperscript{9} Despite the temptation to conclude otherwise, the lack of a demonstrable reduction in systemic disease following conventional anti-bacterial periodontal therapy does not necessarily mean that there is no relationship between oral and overall health. Rather, it may actually be a compelling reason to change our treatment approach to include addressing the host-driven inflammatory component of periodontal disease in addition to the initiating bacterial aetiology. Studying the effect that antibacterial and available host modulation therapies (aimed at reducing the inflammatory trait or as a factor contributing to systemic inflammation, either as a consequence of an underlying hyperinflammatory trait or as a factor contributing to systemic inflammation)\textsuperscript{10,11} have on systemic diseases would provide far more useful information concerning the potential systemic benefit from periodontal therapy.

The interpretation of data linking periodontal and other diseases has other shortcomings. It will never be possible to ‘prove’ or quantify the extent to which systemic disease is influenced by periodontal disease/therapy until the inflammatory burden of oral origin can be quantified. Researchers can use surrogate measurements of systemic inflammation (such as C-reactive protein, etc.) to demonstrate potential benefits of periodontal therapy. However, it is currently not possible to determine how much of the overall systemic inflammatory burden is due to periodontal involvement. The number and/or depth of periodontal pockets are typically used to separate study subjects into different disease categories. Whether such measurements truly correlate with the degree of inflammation produced orally is far from certain. Without knowing the quality or quantity of the inflammatory response and/or bacteraemia introduced into the systemic circulation when different degrees of periodontal disease persist, determination of the strength or clinical significance of the link between periodontal disease and any associated systemic disease is simply not possible. The key question concerning the systemic health benefits of a life-long commitment to minimal oral inflammation management remains unasked and unanswered.

What we can say for sure

Research advocates who use the recent evidence-based reviews to bolster their opinion that periodontal therapy has no effect on systemic disease initiation or progression should reassert their position based on the inherent characteristics of the evidence-based methodology. Evidence-based healthcare depends on the application of the best knowledge a discipline can offer.\textsuperscript{12} Systematic reviews are considered the preferred method for identifying all of the available knowledge, determining which information is ‘best’ and summarising it in a clinically useful manner.\textsuperscript{13} The initial step of a systematic review is formulation of one or more clearly-defined key questions. Conclusions from the review should likewise be well defined relative to the key question.

As such, it is misleading to conclude from the recent meta-analyses that “periodontal therapy” has not been shown to have any significant systemic effect. The evidence-based approach requires that the type of therapy provided in the studies be better defined. For example, a more descriptive conclusion would be that “limited episodes of subgingival debridement without any host modulation” have not been shown to affect the studied systemic disease.

While the current evidence is not yet sufficient to definitely conclude that oral health should be an integral part of preventive cardiology, pre-term low birth weight risk reduction, diabetes management or part of the management strategy for any inflammatory disease, the potential systemic benefit of maintaining optimal oral health should not be ignored. Although the published conclusion in the AHA statement suggests that any cardio-protective effects following periodontal therapy is minimal at best, the statement itself acknowledges the role that inflammatory mediators play in the development and/or progression of many chronic diseases. Contained in the statement is a summary of the systematic review of relevant evidence that confirms that the mouth is a source of the exact same mediators of inflammation when periodontal disease persists.\textsuperscript{14} Optimising diet, exercise, blood pressure and stress level would certainly be on any list of well-accepted health promoting behaviours. It seems reasonable (and from a public health standpoint potentially important) to add to the list a regular devotion to determining if any oral inflammation is present, and a commitment to taking the necessary steps to maximise the chance of keeping that inflammation at bay.

More simply put, it seems prudent and certainly within the realm of the evidence-based methodology to advise patients presenting for dental care that inflammation is an important factor in the development of many serious diseases, and then educate them that the mouth is a significant source of inflammation when periodontal diseases persist.

Conclusions

- A plethora of articles assesses the periodontal and systemic disease connection. Of these a large majority point to a connection, but it is not a causal relationship. Does this mean there is no a connection? We still do not know.\textsuperscript{15}
- Even though we are presented with a lot of biased, poorly executed research, it is still valid to inform patients that periodontal disease and oral inflammation can lead to increased systemic inflammation. Whether this causes cardiovascular disease, preterm low birth weight, or diabetes, etc., is still to be elucidated, but the fact is that prevention of oral inflammation is still important for oral health.\textsuperscript{16}
- What do I tell my patients? If they want to prevent oral disease
then they should see a dentist every six months, and brush and floss their teeth daily. With respect to systemic disease, I say that there is some evidence, while not conclusive, that oral inflammation may lead to an increased risk of systemic inflammation and also increased risk of a number of chronic inflammatory diseases.

References


