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REVIEW ARTICLE

CLASSICAL CONCEPTS OF PERIODONTOLOGY

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ABSTRACT

The maturity of a scientific discipline is marked by knowledge of its roots, knowledge of evolution of concepts from ancient times to present and an intelligent look into the future. Newer thoughts have been superimposed on the matrix of older ideas. The important discoveries and decisions of past centuries became the springboard for inventions and advances of the field in coming centuries. So, periodontology in modern era presents the blend of the thoughts from the times periodontal disease was first noticed in history. This review article is intended to compile the various concepts regarding etiology, pathogenesis and treatment of periodontal diseases that changed with time and gave rise to present concepts and also provide a view of future possibilities.

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INTRODUCTION

Periodontology is the scientific study of the periodontium in health and disease ([Glossary of Periodontal terms, 2001](#)). Periodontal diseases have plagued humans and their phylogenetic ancestors for a very long time. Since the times history first mentioned periodontal disease, the concepts regarding etiology, pathogenesis, treatment and prevention of periodontal diseases are in a state of flux ([Tatakis and Kumar, 2005](#)). In a sense, the newest or dominant concepts rest on a foundation of the still valid components of the older or previous thoughts. As said,

*"Change is one thing, progress is another,
Change is scientific, progress is ethical,
Change is indubitable whereas progress is a matter of
controversy."*

-Bertrand Russell

With all this, the article aims to bring together the classical concepts of periodontology regarding etiology, pathogenesis, treatment and prevention of periodontal diseases.

CLASSICAL CONCEPTS OF ETIOLOGY

The basic concept of etiology of periodontal diseases consisted of two camps namely: the "localists" and the "generalists" or "constitutional" camps ([Hujoel et al., 2000](#)).

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The Localist group: For the ease of simplification the local-cause theory can be summarized into four axioms as follows (formulated over two centuries ago)

- Periodontal disease is independent of systemic disease manifestations.
- Periodontal disease had a local etiology or accidental origin occasioned by teeth and no internal or general cause.
- Local, intraoral interventions can prevent and successfully treat periodontal disease.
- Local treatments can provide systemic health benefits.

The generalists camp: The generalists said that systemic conditions were the immediate cause of periodontal disturbances. They hypothesized that primary cause of periodontal disease are remote from the oral cavity and are only amenable to chronic disease management unless the remote cause are pinpointed and intervened upon ([Hujoel et al., 2000](#)). However, Irving Glickman in 1959 considered that these two factors are actually interrelated. He concluded that gradations in the proportion of local and systemic disease causing factors determine the nature and course of periodontal disease.

Over the coming decades, periodontal disease became more redefined as an infectious disease and Harold Loe in 1960s provided experimental evidence for a *direct relationship between the presence of dental bacterial plaque and gingivitis* which affected most aspects of clinical periodontology prevailing at that time ([Theilade et al., 1966](#)).

Sigmund Socransky in 1970 further advocated that periodontal infections are caused by organised communities of bacteria in which some microorganisms are more important than others from an etiologic perspective and this belief made Walter Loesche in 1976 to advocate the 'Specific Plaque hypothesis' over the 'Non Specific hypothesis' (Loesche, 1976).

The non specific hypothesis describes that the periodontal disease results from the "elaboration of noxious products produced by the entire plaque flora". The Specific plaque concept predicts that the plaque harbouring specific bacterial pathogens results in a periodontal disease because these organisms produce substances that mediate the destruction of host tissues (Teughels et al., 2012). However, Marsh and co-workers in 1990s gave the ecologic plaque hypothesis which states that both the amount of dental plaque and the specific microbial composition contribute to the transition from health to periodontal disease.

By 1987 the majority of clinicians and authors accepted the concept that most forms of destructive periodontal disease followed the Infection-Host Response paradigm. In summary, during (1962-1987) the important concepts in periodontal etiology were as follows:

- Chronic gingivitis and experimental gingivitis appear to be inflammatory reactions to qualitative changes in the composition of dental plaque at affected sites.
- Only a small percentage of sites with untreated chronic gingivitis progress to periodontitis. This progression is not inevitable.
- The progression of periodontitis, or the development of increased clinical attachment loss, appears to be episodic or occurs in bursts.
- Periodontosis (juvenile periodontitis) was shown to be an infection and not a degenerative condition.
- The most common form of destructive periodontitis (i.e. chronic inflammatory periodontitis) is a group of infections caused by dental plaque in susceptible individuals.
- The concept emerged that a relatively small number of specific bacteria appear to be the main etiologic agents of adult periodontitis.
- Hard and soft tissue damage observed in periodontal disease was primarily caused by inflammatory and immunologic host responses to infecting agents (Armitage, 2000).

From 1990 to 2012 most of the research dealing with the conceptual issues in periodontal etiology focused on confirmation of the concepts that were introduced during two and a half decades from 1962-1990. In 2012, the prevailing concepts in periodontal etiology were essentially the same as those were followed in 1990. In retrospect it is somewhat disappointing that paradigm shifting discoveries have not occurred in the past 23 years. Nevertheless, there have been scientific advances and technological developments that have slightly modified or refined the concepts that govern the field of periodontal etiology. The current concept concerning the periodontal diseases considers that three groups of factors will determine occurrence of active periodontal destruction in a subject:

- The susceptible host
- The presence of pathogenic species
- The absence/small proportion of beneficial bacteria

CLASSICAL CONCEPTS OF PATHOGENESIS

From the pages of history we now know that the concepts of etiology of periodontal diseases shuffled with time, and the same happened with the concepts of pathogenesis of periodontal disease. The prehistoric era had little knowledge about the mechanism of periodontal diseases. The early assumptions held that disease or injury was a deserved evil visited upon an individual by the deity for infarctions in thought or deed (Gold, 1985). In 1728 Pierre Fauchard recognized the relationship between oral hygiene and the etiology and wrote:

"Little or no care as to the cleanliness of the teeth is ordinarily the cause of the maladies that destroy them."

But with time the developments in the medical and technical fields opened new methods to understand the disease mechanism. In the 18th century, the first scientific studies were presented by Morgagni GB and Bichat that hinted the correlation between clinical symptoms and autopsy findings. Morgagni's descriptions clearly distinguished the important findings from the significant details, presenting the clinical signs and symptoms, the treatment performed, and the autopsy findings with an interpretation of their relationship to the clinical picture. Bichat's contribution was to extend the Morgagni's concept of organ pathology to the organ constituents, namely the tissues. He discovered that organs are made out of tissues and that the tissues are the origin of pathological changes (Merrit, 1921).

- In the 19th century, three major developments in medical science had a particular impact on Periodontics: 1. Discovery of anaesthesia, 2. Development of germ theory of disease, and 3. Discovery of X rays.
- During those days periodontal pathogenesis was dominated by the premise that local factors lead to the destruction of periodontal tissues. Researchers believed that most of the destruction of periodontal tissues during the course of the disease was due to inflammatory or degenerative/atrophic processes. Until the late 1960s, researchers based their studies primarily on observations and individual interpretations of the histologic changes in the diseased tissues.
- In the 1960s, the major development was the demonstration that bacteria in dental plaque cause human gingivitis and periodontitis.
- In the 1970s, specific species of predominantly gram-negative, anaerobic bacteria were associated with these diseases
- The 1980s, saw strong documentation of the association of specific bacteria with active tissue destruction, beginning characterization of the immune response to antigens and mitogens of the infecting bacteria and beginning elucidation of the role of cytokines and prostaglandins in the pathogenesis

- In 1982, Page RC and Schroeder HE stated that periodontitis is not a single homogeneous disease but rather consists of a family of closely related diseases each of which may vary somewhat in etiology, natural history and response to therapy. Nevertheless, a common underlying chain of events in the pathogenesis is shared by all forms of the disease. The histopathological and ultrastructural features and pathways of tissue destruction as well as healing and regeneration are very similar if not identical. The same basic pathological mechanisms underlie all forms of bacterially induced periodontitis.
- “The research in 1990s about pathogenesis of periodontal disease discovered that bacteria are essential but insufficient for disease, that bacteria account for a relatively small portion of the variance in susceptibility for disease expression, and that hereditary factors alone can account for up to roughly 50% of the variance. This was thus the decade of the paradigm shift; this was the “*decade of the host and disease modifiers*”. In addition, this shift provides a new perspective on the distinctly different roles of the bacteria, the host and risk factors and indicators in the disease process.
- A useful conceptual model was also introduced in 1997 to explain the pathogenesis of periodontal diseases (Fig 1). However, the model presented by Page RC and Kornmann in 1997 continues to be refined.

CLASSICAL CONCEPTS OF TREATMENT

Non surgical treatment: As a result of the confusion regarding the etiology of periodontitis for many years, treatment included a range of therapies including dietary changes, gingival massage, local application of caustic chemicals, removal of local irritants and surgical resection of affected tissues etc (Armitage and Robertson, 2009). On June 10 and 11, 1869, the following resolutions were passed giving credit to Riggs JW for "originating and first publicly describing a new treatment for the cure of inflammation of the gum." Thorough curettement of the alveolar process formed a prominent feature in the treatment and, as practised by Riggs, made of it heroic operation.

At the beginning of the 20th century, there were two major approaches to treatment of periodontitis.

- One approach involved the use of surgical resection of periodontal pockets followed by curettage of the underlying bone.
- Proponents of the second approach held that the disease was caused by local irritation from dental calculus, and the underlying bone was not affected. Practitioners like Riggs and Younger WJ favoured the nonsurgical removal of acquired deposits followed by a rigorous program of oral hygiene.

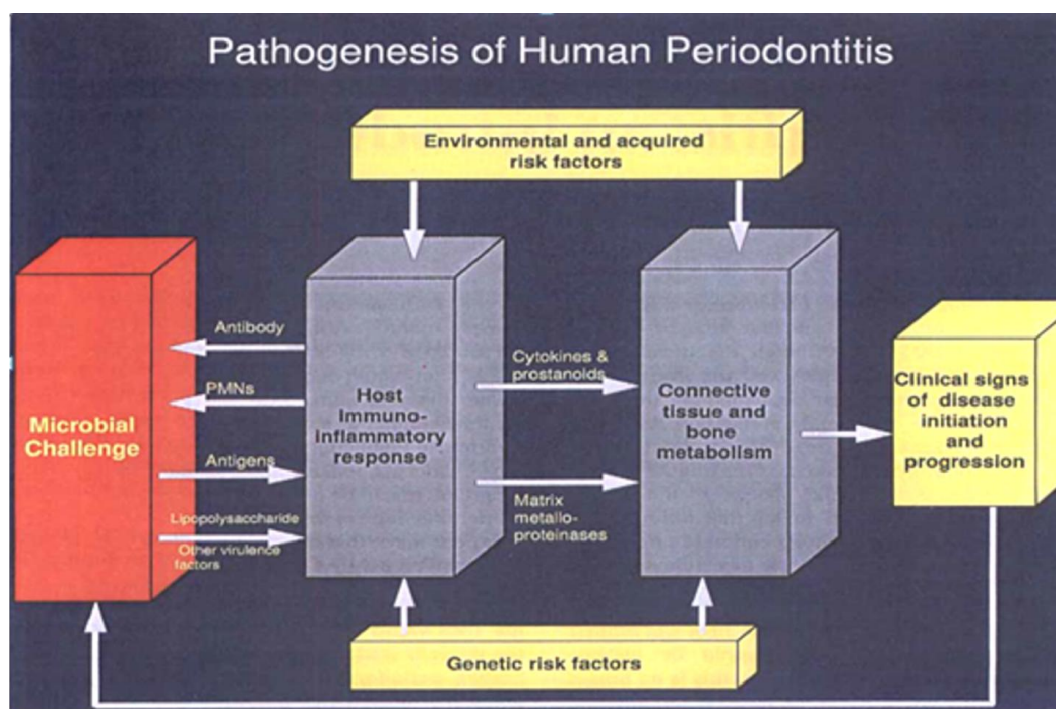


Fig. 1. Schematic illustration of the pathogenesis of periodontitis

Further Offenbacher in 2008 (Fig 2) suggested a Biological system model which includes a person level, a genetic/epigenetic level, the biological phenotype and ultimately the clinical phenotype. This model had more comprehensive view of the disease as a complex regulatory network, in which aspects of the specific genetic factors, environmental factors that an individual is exposed were mentioned to determine the development of the disease (Offenbacher *et al.*, 2008).

In the 1970s and 1980s nonsurgical periodontal therapy was performed using predominately hand instrumentation, with the aim being to remove supragingival and subgingival calculus and plaque, and contaminated root cementum. The use of ultrasound in dentistry was proposed by Catuna in 1953 for the process of cutting teeth, further work undertaken by Zinner in 1955 showed that ultrasound could be used to remove deposits from the teeth. Syzmid and McColl in 1960 accepted the use of ultrasound instruments for scaling and stated that the

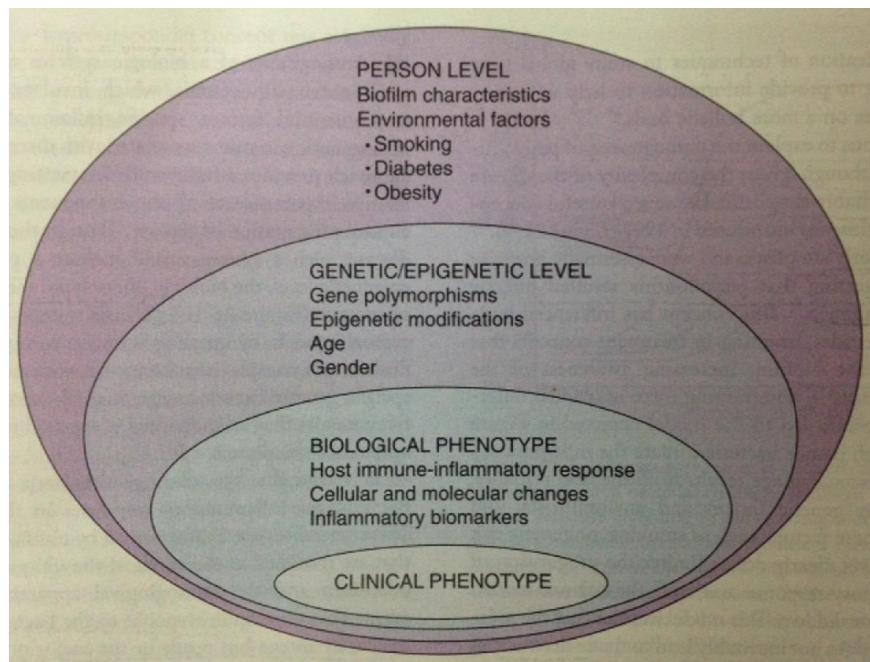


Fig. 2. A biologic systems model for representing periodontitis

instruments are acceptable alternatives to hand scalers. It was stated that the instruments were acceptable alternatives to hand scalers in 1960 as they were found to be as effective in the removal of calculus.

In the 1970s, scaling and root planning combined with gingival curettage was a common procedure for periodontal therapy. In the early 1980s Echeverria B and Caffesse RG, challenged the value of gingival curettage and concluded that gingival curettage did not result in any additional improvement (Gemmell *et al.*, 2000). In 1968 Bader HI and Paul Goldhaber found that tetracyclines are excreted in the gingival fluid and this increased the interest in the use of antibiotics in periodontal therapy (Carranza, 2003). However, the concept of local delivery to the periodontal pocket and the first systems of this were developed by Max Goodson, at the Forsyth Dental Infirmary in Boston in 1979. The concept of Host Modulation was first introduced to dentistry by Williams (1990) and Golub *et al* (1992). A variety of drug classes have been evaluated as host response modulators, including the nonsteroidal anti-inflammatory drugs, bisphosphonates, and tetracyclines.

In 1995, the concept of full mouth disinfection was introduced as a potentially more effective non-surgical periodontal therapy than the conventional quadrant-by-quadrant scaling approach. But if targets are not achieved by non-surgical means, a surgical approach may be indicated. Surgical therapy was also adopted by many clinicians in history. Surgical treatment: Most of the progress in the periodontal surgery in last years of 19th century came from Germany and other European countries, and is associated with three names: Robert Neumann, Leonard Widman, and Cieszynski A. In the 1920's, a controversy centering on the priority of periodontal flap surgery involved Cieszynski, Widman and Neumann, each claiming to have been the first to publish this material (Widman L 1923, Neumann R 1923, Cieszynski A 1926).

of vertical incisions, not bisecting the interdental papilla followed by crevicular incisions to the bone margin to separate a flap that was then elevated to gain clear view of the entire field of operation. While the flap was held open with the retractors, all granulations and calculus were removed and the bone margin smoothed with chisels and burs to reshape it to its normal topography. Neumann later followed Weski, advocating transformation of all vertical bone losses into horizontal ones, meaning to eliminate all vertical losses. Neumann's flap operation had wide diffusion in North and South America in 1920s.

He outlined his treatment methods which included full radiographic survey and clinical examination, instructions in oral hygiene, meticulous scaling and root planing, as well as flap surgery in cases of abscess or deep granulation tissue. Leonard Widmann (1917) gave Widman's technique similar to Neumann's and recommended surgery to obtain access to root deposits for their complete elimination of granulation tissue. In 1918, Arthur Zentler, described a technique similar to Neumann's, as two parallel vertical incisions and scalloped incisions "following the original festoons of the gums, lifting a flap to allow root scaling and curettage to remove all granulation tissue from the pocket area and underside of the flap, and trimming and smoothing, with chisel and mallet, all infected bone. He then cut away with scissors the margin of flap and sutured the vertical and interdental incisions.

Zentler claimed that treatment produced a successful and permanent cure in short number of visits, and was not painful (Carranza, 2003). William Ziesel in 1920 presented a gingivectomy technique using specially designed instruments. In 1926, James LZemsky presented a technique that he called *open view* operation, which was a flap technique with removal of "infected and sharp edges of bone." Olin Kirkland in 1932 presented *modified flap* operation.

Toward the late 1930s and 1940s, the procedures were refined and the aggressive curettage of the osseous margin – an attempt to remove “diseased bone”-was ceased (Carranza, 2003). In 1950 Henry M. Golman described gingivoplasty. A few years later in 1949, not knowing Carranza’s work, Sauls Schluger described a similar technique, with the indication of bone contouring in isolated infrabony pockets on facial or lingual surfaces, interdental craters, and deep pockets on the mesial aspect of tilted second molars where the first molar has been lost and not replaced. A new approach to the treatment of osseous defects was introduced in 1923, seeking to rebuild rather than eliminate them by surgical recontouring, had begun to appear with the work of Zoltan Hegedus, who proposed to rebuild the lost alveolar bone by “utilizing the bone-building and regenerative property of periosteum.

In the 1970s and 1980s from some important clinical trials it was established that nonsurgical periodontal therapy is effective in eliminating inflammation in deep pockets and in improving clinical attachment levels. However, despite best efforts at meticulous nonsurgical instrumentation, residual plaque and calculus may still be found. It was accepted that in situations where signs of inflammation persist, surgical therapy may be indicated. According to Lindhe *et al* in 1982, a concept of “critical probing depth” was developed for decision making following the completion of a hygienic phase [initial periodontal therapy (non-surgical therapy + oral hygiene instruction)]. The critical probing depth represents a baseline probing-depth value above which the outcome of a therapy will result in attachment gain and below which the outcome of therapy will result in clinical attachment loss. The critical probing depth for nonsurgical therapy (scaling and root planing) is 2.9 mm. This means that below this probing depth the site would lose clinical attachment as a result of therapy. However, above this value clinical attachment gain will result. On the other hand, for the access flap therapy, the critical probing depth is 4.2 mm. Again, this means that open flap debridement is only beneficial above this value, while below this value, attachment loss may result. The next major advance in periodontal regeneration was the proof of principle introduction of guided tissue regeneration (GTR) procedures in 1982. Nyman S and colleagues in 1982 placed a barrier membrane between the periodontal flap and a tooth scheduled for extraction in a patient with severe periodontitis.

As the field of periodontics matured from 1970 to 2000, investigators in many controlled studies evaluated the effects of periodontal flap procedures alone compared with flap procedures combined with the insertion of various bone-replacement graft materials. In a systematic review and metaanalysis of these studies, Reynolds MA and colleagues in 2003 concluded that bone-replacement grafts resulted in statistically significantly increased bone and clinical attachment levels and reduced probing depths compared with flap procedures alone (Reynolds *et al.*, 2003) and According to Dentino, *et al* in 2013, The surgical approaches have been utilized in treatment of periodontal disease for decades, and are broadly classified as: Access procedures, Resective procedures and Regenerative procedures and The pocket depth and bone architecture will dictate which surgical approach is indicated (Heitz-Mayfield and Lang, 2000).

CLASSICAL CONCEPTS OF PREVENTION

Although there are various techniques of therapy for periodontal disease but according to phrase “*Prevention is better than cure*”, various methods to prevent periodontal diseases have been practised since prehistoric times. Early tooth cleaning devices included toothpicks (metallic and wood) and wooden chew sticks. Tooth picks were used by Greeks and Romans, probably before them by Babylonians and Chinese. The Chinese were amongst the first people to use the chewstick as a toothpick and toothbrush to clean the teeth and massage the gingival tissues. The Romans were also very much interested in oral hygiene. The use of the toothbrush was also mentioned in the writings of many of the Roman poets. In the thirteenth to seventeenth centuries toothpicks were made of precious metals and jewels, being ornamental in addition to utilitarian. The use of a chew stick, called *sivak* recommended at that time is still used by people in Asia and Africa (Carranza, 2003).

The toothbrush appeared about the year 1600 in China, was first patented in America in 1857 and has since undergone little changes. Chinese dentist would clean teeth with hairs of pigs and paste them on bamboo sticks or animal bones. Europeans would brush their teeth by dipping linen cloth or sponges dipped in sulfur oils and salt solutions and rubbing away all the tooth grime. William Addis became the first person to mass produce modern toothbrushes. He used cow hair drilled and tied on to cow bones. A suggestion regarding the benefits of flossing dates back to the early 19th century, when it was believed that irritating matter between teeth was the source of dental disease.

Then, Alfred Fones, in 1934 described the circular technique known as the Fones method. Paul R. Stillman published extensively on periodontal diseases and their treatment. In 1932 he outlined the so-called Stillman method. In 1939, Isador Hirshfeld described the importance of flossing and the technique for its use, including the use of floss holders, but flossing did not come into practice until gingival massage was proven to be unimportant in maintaining gingival health. Powered toothbrushes were invented in 1939. Numerous types have been marketed since then, with technological advances that have made them a valuable part of the oral hygiene armamentarium.

The various methods used today to prevent periodontal diseases include Mechanical plaque control and Chemical plaque control. Mechanical plaque control is achieved by the use of: Toothbrushes, Dentifrices and Interdental aids. Chemical preventive agents: These agents are viewed as adjuncts and not replacements for effective mechanical plaque control. They are preventive agents, not therapeutic agents. The American Dental Association has accepted two agents as plaque control agents: Chlorhexidine and Essential oil rinse. Additional oral hygiene aids have been developed in an attempt to augment the effect of toothbrushing on reducing interdental plaque. The oral irrigator was introduced in 1962 but there is contradictory evidence about the usefulness of supragingival irrigation in reducing plaque formation and prevention of periodontal diseases.

FUTURE TRENDS

The future advances in the concepts of periodontal diseases demand the more relevant ability to detect microorganisms that could not be cultivated so far. The recognition of the beneficial activity of several groups of commensal species might open new strategies for understanding of etiology and treatment of periodontal disease. Greater awareness of the role of the host response in the periodontal disease will further improve the understanding of the severity of periodontal diseases. Until recently, it is not possible to conduct in depth studies of full spectrum of host microbe interactions in health and disease (Armitage, 2000). So, the current intense research to understand the impact of the human microbiome on health and disease will lead to major conceptual changes of periodontal etiology in the coming decades.

New technologies have been developed or are in development that could be used to treat periodontitis. Not all of these technologies will bear fruit; however, those that do will provide clinicians of the twenty-first century with more effective means of non-surgical treatment of periodontitis than are currently available. In recent years, refinement of periodontal surgical techniques has been possible with development of new instrumentation and the use of illumination and magnification. Minimally invasive periodontal surgical approaches and microsurgical techniques are currently being evaluated and may show advantages (Heitz-Mayfield, 2000). Nanotechnology is another growing field that is potentially altering the ways to treat diseases through novel and advanced diagnostic and therapeutic methods (Heitz-Mayfield, 2000). The use of laser light in treatment of periodontal diseases and Photodynamic therapy are also fields of great potential light but the technology appears to require more development and testing before it can be considered a substitute for classic mechanical debridement (Heitz-Mayfield, 2000).

Further advances in the host modulation area of therapy await better classification of diseases and identification of the multifaceted molecular basis for the biofilm / host imbalances that produce destructive inflammation. Vaccination against bacterial/viral infectious diseases has progressed immensely throughout the 20th century. Periodontal diseases are one such group of infectious bacterial diseases, against which vaccine research is still going on. The complexities in the etiopathogenesis of the periodontal diseases have been the prime obstacle in the hunt for vaccine.

The most dramatic potential for future control or virtual elimination of periodontal disease may emanate from advances in understanding the role of heredity in determining susceptibility to and the severity of disease. Although this field is still in its infancy, enough is known already to glimpse the future. Under the new paradigm, periodontics is rapidly changing from diagnosing and treating existing disease to prevention and health promotion. Reduction of risk becomes the primary objective of intervention for individuals and populations.

Identifying the factors that place individuals and groups at enhanced risk and managing risk as a means of prevention and treatment are of ever increasing importance. Some risk factors are immutable to change; others are not.

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