Editorial

Role of novel multi omics in periodontal disease

Ramesh Babu Mutthineni [1],*

[1] *Dept. of Periodontics, Mamata Dental College, Telangana, India*

A R T I C L E   I N F O

Understanding the origins and mechanisms of a disease is crucial for developing successful treatments. This can be challenging to do, especially in places of the body like the oral cavity where a wide variety of microorganisms are already resident. Complex biofilm patterns on teeth and soft tissues make it difficult to distinguish between primary pathogens and nonpathogens, making it difficult to identify the underlying causes of periodontitis.

In the early 1990s, specialised molecular approaches were developed, allowing for the simultaneous identification and quantification of numerous species in tens of thousands of plaque samples. They also improved the diversity of microbes that could be enumerated precisely, opening the door to research into species that had been difficult to culture. Checkerboard DNA-DNA hybridization is one such method, and its findings have considerably advanced our knowledge of the microbiological consequences of various treatment protocols and helped define microbiological end objectives for periodontal therapy. Beginning in the early 2000s, DNA-sequencing technologies allowed for considerably broader and more in-depth study of the oral microbiota. Studies employing Sanger sequencing, cloning, and sequencing laid the groundwork for an exciting era of discovery in oral microbiology in relation to health and disease. The subsequent development of next-generation sequencing technology paves the way for comprehensive oral microbial surveys based on useful marker genes like 16S ribosomal RNA (rRNA), community gene inventories (metagenomics), and functional research (metatranscriptomics). Researchers have recently adopted this method to assess the impact of different therapies on the oral microbiome.[1] As it becomes apparent that the variety of oral microbial communities is more complex than previously assumed, a new era of periodontal microbiology study has begun. In addition, there are likely yet undiscovered and uncultured diseases waiting to be discovered.[2]

Omic Technology In Periodontal Clinical Practice

Loss of teeth is linked to periodontal disease, the most common chronic inflammatory disease affecting humans. Patients with periodontitis need to be identified in nonspecialist and/or nondental settings, and there is a need for simple, objective diagnostic procedures. Diagnosis requires clinical expertise, training, and specialised equipment.

Such diagnostic tests may be developed through the discovery of biomarkers by means of omic technologies including genomics, epigenomics, transcriptomics, proteomics, metabolomics, and microbiomics.

According to the official definition published by the National Institute of Health, a biomarker is "a trait that is objectively measured and analysed as an indicator of normal biologic processes, pathogenic processes, or pharmacologic reactions to a therapeutic intervention" (NIH, USA).

* *Corresponding author.*
  E-mail address: rameshbabu297@gmail.com (R. B. Mutthineni).

Six distinct classes of biomarkers have been identified, according to recent research by Bensalah et al.[3]

1. Early disease detection
2. Early diagnosis of disease's presence or absence
3. A prognosis for the course of the disease and possible patient stratification to enable individualised medical interventions (especially in periodontology for persons who are more likely to experience recurrence of the condition);
4. Anticipating the course of treatment;
5. Identifying patients who will benefit from a specific treatment; and
6. Surrogate end-points

If a biomarker or panel of biomarkers is going to be employed in a clinical setting, it needs to be able to outperform current assays in terms of sensitivity, specificity, and diagnostic accuracy, as well as be objective, reproducible, easy to use, inexpensive, and widely applicable. Most relevant biomarkers have been discovered in the past either by serendipity or after rigorous investigation of candidates generated by hypothesis-driven research. Many prospective biomarkers are generated using preclinical in vitro models, but only a subset of those go on to generate assays that are utilised to evaluate a modest sample of patients in phase 1 studies. It takes more than raw data to prove a biomarker works; a well-designed, carefully-executed experiment is required. Fewer phase 1 studies exist than the equivalent phase 2 and 3 trials, which are conducted on large populations of people living in the community. Therefore, these state-of-the-art biomarker technologies should be used in bigger multi-center, multi-omic systems biology experiments.

The whole phenotype of periodontal disease is influenced by multiple levels of biology, including genes, transcripts, proteins, and metabolites. When compared to genomics, which examines the fixed encoding of the genome, it is important to remember that transcriptomics, proteomics, and metabolomics evaluate the dynamic expression of genes. They therefore consider not only environmental factors but also influences of nature and nurture. Transcriptomics is affected by translation and activation, proteomics explains post-translational changes and protein splice variants, and metabolomics shows off reaction products. As we move from genomics to transcriptomics, proteomics, and metabolomics, our focus moves from hypothetical outcomes to those that really occurred. Genomic, transcriptomic, and proteomic discoveries have been tremendously facilitated by the relatively regular structure of the DNA, RNA, and proteins being studied. There are also many other differences between the studies that make up each of these areas of inquiry. However, the huge variety of tiny molecules included in metabolomics exhibit a wide range of acidity, alkalinity, and other physicochemical properties. This points to a rise in the analytical difficulty and complexity of metabolomics. It's important to keep in mind that each level might have an effect on the others via feedback loops and regulatory processes, so you can't pick one as being simpler than the others. More than 30,000 genes and transcripts, over 100,000 proteins (including post-translational modifications), and over 6,500 metabolites have been predicted for the human body. However, the investigator encounters challenges when thinking about the different molecular species present in each location.[4]

In conclusion, our approach to patient care in routine practise has not changed as a result of OMICS expertise. First, it dispels the myth that open-ended culture and molecular target approaches' definitions of the primary goals of periodontal therapy are flawed. This is significant because it allows researchers to expand the range of animals they can study rather than wasting time disputing earlier findings. Second, it supports the idea that periodontitis is a complicated ecological issue that requires the control or eradication of pathogens and the recolonization of the mouth cavity with species beneficial to periodontal health. Future randomised clinical trials could monitor these species/genera in a more thorough manner when new diseases and helpful species were discovered in these research. Third, although still preliminary, the metatranscriptomic data imply that the interaction between the host and the oral microbiome may represent our greatest chance to apply individualised periodontal therapy. The future of improved periodontal therapy may involve therapeutic procedures that target particular bacterial protein products in patients with particular genetic profiles, for instance.

1. Conflict of Interest

None.

References

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Author biography

Ramesh Babu Mutthineni, Professor ⓘ https://orcid.org/0000-0001-6904-8385