Periodontal disease is one of the most prevalent diseases of the dentition and is responsible for most of the teeth lost by adults. It has been known for some time to be associated with oral microorganisms. The microbial population of the oral cavity is one of the most complex of the human body, comprising over 300 species. Some of these grow in vitro with difficulty or not at all. Until recently, the lack of suitable culture techniques coupled with gaps in our knowledge of the taxonomy of the oral microbiota gave the erroneous impression that the composition of the microbiota in periodontally healthy patients was identical to that of patients with periodontal disease. Periodontal disease was believed to be the result of an increased mass of bacteria rather than qualitative differences in the composition of the microbiota.

Already in the early 1960s, as a postdoctoral student at the Harvard School of Dental Medicine, I had noted that electron microscopic observations of microorganisms associated with periodontal disease, notably necrotizing ulcerative gingivitis ("trench mouth"). did not correspond to the culture data. Many microbial forms, readily identifiable microscopically, appeared to escape cultivation, with the result that microbial proportions observed microscopically bore little relationship to those reported on the basis of culture analyses. Furthermore, differences appeared to exist in the composition of bacterial deposits recovered from periodontally diseased vs. healthy teeth, an observation that also appeared to be at odds with the culture data. These observed differences between microscopic and culture data generated lively discussion with some of my fellow postdocs, notably S. Socransky at the Forsyth Dental Infirmary (now Dental Center) in Boston.

Studies undertaken in the early 1970s2 began to shed some light on the mechanisms of plaque growth. At that time, it was generally believed that microbial deposits thickened by appositional growth. These studies provided evidence of internal growth by bacterial division and clear evidence of bacterial succussions during plaque maturation. Shortly thereafter, we began to examine the microscopic structure of dental plaques on condemned teeth with known periodontal conditions. Teeth that were scheduled for extraction were carefully extracted and processed for microscopic examination of the associated microbiota. The study graphically demonstrated for the first time that qualitative differences existed between health-associated microbiotas and those associated with various forms of periodontal disease and that the differences were much more dramatic than those suggested by the culture data from healthy and diseased microbial samples. These results also constituted the first convincing evidence that specific microorganisms might be associated with clinically distinct periodontal diseases. These findings provided added impetus to improve culture techniques and to pursue taxonomic studies of the periodontal microbiota in periodontal health and disease. These efforts eventually led to the confirmation of the original microscopic observations of microbial diversity in different disease entities and opened up new approaches to the diagnosis of periodontal infections and their treatment.3

The aesthetic appeal of some of the transmission electron micrographs also may have accounted in part for the popularity of this particular paper.