The paper provided evidence for the role of dental plaque bacteria in the initiation of human periodontal disease. Complete withdrawal of oral hygiene for three weeks invariably caused gingivitis. The number of bacteria colonizing the teeth increased dramatically and distinct changes in bacterial ecology occurred over time. Reinstitution of plaque control resulted in reestablishment of healthy gingiva. [The SC® indicates that this paper has been cited in over 455 publications since 1965.]

In a crude sense, the notion that debris on teeth plays a role in the destruction of the tissues supporting the teeth is centuries old. On the other hand, the scientific evidence to accept or reject the idea had been lacking; the etiology of periodontal disease was poorly understood and, consequently, the clinical management of this disease was generally confused and ineffective. More teeth were lost in adult life due to periodontal disease than to any other condition.

"In 1952, Jens Waerhaug of the University of Oslo had published his thesis, The Gingival Pocket, which constituted a comprehensive study of the dynamics of the gingiva in health and disease. As a recent graduate, I was afforded the opportunity to work with Waerhaug on various clinical aspects of periodontal disease for some ten years prior to assuming the chair in periodontology at the Royal Dental College in Aarhus, Denmark, in 1962. By that time, there was a fair amount of clinical experience as well as epidemiological data to indicate that a close relationship existed between deposits on teeth and destruction of the periodontal tissue and the loosening and eventual loss of teeth. The time and mind were ripe for a definitive test of the hypothesis that it was the dental plaque component of oral accumulations that is responsible for the initiation of periodontal disease.

"In the study to follow, we asked 12 healthy, young individuals with clean dentitions and normal gingiva to refrain from any measure of oral hygiene for a period of three weeks. By assessing the gradual build-up of deposits on the dentition and the response of the gingival tissues, it appeared that in all individuals the rapid accumulation of bacterial plaque elicited an inflammatory reaction in the gingival tissues, which clinically was characterized as gingivitis. The time necessary to develop gingivitis varied between ten and 21 days. Concurrent microbiological examinations showed that the number of bacteria colonizing the tooth surfaces increased dramatically with time and that distinct changes in the composition of the flora occurred as well. When good oral hygiene was re instituted, the original sparse microflora was reestablished and the inflamed gingiva reverted back to normal.

"Since this first experimental gingivitis study, we and others have reproduced the results in different age groups and in different racial groups in various geographical locations. It was soon apparent that this constituted a reproducible, low cost human experimental model for the study of the detailed microbiology of dental plaque, the immunological and other host responses to the bacterial attack, and the cytological characteristics of the lesion. Over the years a substantial amount of data has been accumulated and continues to emanate from such studies. However, beyond the scientific utility and potential of this experimental system, the study has had major clinical impact as a basic reference to the bacterial etiology of periodontal disease, its infectious nature, and the importance of oral hygiene in the control of this major dental disease."

"Personally, I find the simplicity of the model aesthetically satisfying and its lack of compromise calls for the objective truth. There is, perhaps, some intrinsic heroism in the execution of this model, but there is no permanent damage to the participant."