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1985 – 1990	Université Louis Pasteur School of Dental Surgery Strasbourg, France	DDS 1990 Dental Medicine
1990 – 1996	Northwestern University Chicago Illinois	PhD in Molecular and Biochemistry – Advisor Dr. Arthur Veis
1992 – 1995	Northwestern University Chicago Chicago Illinois	Certificate in Periodontics
1996 – 1998	Northwestern University Chicago Illinois	DDS 1998 Dental Medicine
1996 – 1998	Northwestern University Chicago Illinois	Dr. Arthur Veis Post-Doctoral Fellow
1999 – 2000	Oregon Health Science University Department of Periodontal and Oral Molecular Portland, Oregon	Assistant Professor
2000 – 2002	Carnegie Mellon University Bone Tissue Engineering Center Pittsburgh, Pennsylvania	Assistant Res. Scientist
2000 – 2007	University of Pittsburgh Periodontics, School of Dentistry Pittsburgh, Pennsylvania	Assistant Professor
2005 – present	University of Pittsburgh School of Dental Medicine Center for Craniofacial Regeneration Pittsburgh, Pennsylvania	Founding Director
2007 – present	University of Pittsburgh School of Dental Medicine Department of Periodontology and Preventive Dentistry Pittsburgh, Pennsylvania	Associate Professor
2013 – present	University of Pittsburgh School of Dental Medicine Pittsburgh, Pennsylvania	Associate Dean of Research
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Harnessing the Power of the Immune System to Treat Periodontal Disease

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Periodontal disease is characterized by destructive inflammation of the periodontium. Recent data in the literature has provided clues toward understanding how inflammation is involved with disease progression, leading to tissue destruction and bone resorption. It is thought that the pattern of chemokines expressed in periodontal tissue may determine the nature of leukocytes that migrate into the gingivae, and in turn, determine the pattern of cytokines produced in the periodontal environment. Ultimately, the overall balance between pro- and anti-inflammatory cytokines determines the severity of periodontitis through the modulation of the balance between the osteoclastogenic factor RANKL and OPG, its endogenous inhibitor.
Our team's objective is to understand and utilize the inflammatory and regulatory processes of the periodontium toward therapies that address the physiological cause of disease progression, namely superfluous host-response. We have focused on developing controlled release formulations to regulate the harmful inflammatory responses. Our strategy has initially targeted the release of CCL22 to recruit regulatory T-cells and more recently we have targeted other pathways of the inflammatory pathway. This data relating to regulation of both inflammation and the immune response will be presented highlighting these approaches as potential therapies for periodontal disease.