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Peripheral Interactions of Tongue Tumor and the Sensory Nervous System

Shivani Ruparel, PhD, is associate professor and director of Research in the Department of Endodontics at University of Texas Health San Antonio (UTHSCSA). She obtained her doctoral degree in cancer biology in the Department of Cellular and Structural Biology at UTHSCSA under the guidance of Dr. Robert Marciniak and Dr. Linda deGraffenried in 2009. This was complemented by her postdoctoral training, under the guidance of Dr. Ken Hargreaves, which focused on pain neuropharmacology and biochemistry. She started her independent research program in 2012 on cancer and pain. Alongside, she also obtained a master of science in clinical investigation at UTHSCSA. Her research program focuses on peripheral mechanisms of oral tumorigenesis, oral cancer-induced pain, and pain associated with cancer treatment. She was inducted to the Omikron Kappa Upsilon National Dental Society in 2018. She has been well funded throughout her career by several private and federal agencies.

Abstract: My lab focuses on peripheral interactions of oral tumors and the peripheral sensory system. Specifically, we are interested in investigating the cross-talk of tongue tumors and the peripheral sensory afferents in mediating tumor-induced pain as well as tumorigenesis. To study mechanisms of oral cancer pain, we are focusing on the role of neurotrophins like BDNF that is released from cancer cells and control activities of the lingual sensory fibers via the TrkB receptor. Additionally, we are also studying how the oral tumor microenvironment plays an important role in the interaction of the tumors with the innervating afferents. Most of my talk will focus on the role of newly identified lymphotoxin beta and its ligands in mediating oral cancer pain. While lymphotoxin-beta receptor and its ligands; LIGHT and Lymphotoxin-beta are known to play an in autoimmune and inflammatory conditions, its role in pain is entirely known. The signaling pathway is traditionally known to mediate pro-inflammatory effects; however, our data revealed that activation of LTBR reverses oral tumor-induced pain. Interestingly, we also found that the effect of LTBR pathway on nociception could be tissue specific.