Human papillomavirus (HPV) is a large family of DNA tumor viruses. The two genera of HPV most associated with disease are the alpha and beta genera (α- and β-HPVs). α-HPVs cause cancers throughout the anogenital tract as well as in the head and neck. These infections are extremely common with over 14 million new infections each year in the US alone. Worldwide, they kill someone every two minutes. β-HPVs are even more common, with majority of adults showing signs of past or current infection. Unlike α-HPVs, β-HPVs are cutaneous viruses and less clearly tumorigenic. Despite these differences, both genera of HPV manipulate host DNA damage responses to facilitate their replication. We have used a combination of \textit{in vitro}, \textit{in vivo} and \textit{in silico} approaches to characterize the consequences of HPV-driven changes in host DNA repair. A β-HPV protein (β-HPV E6) reduces repair signaling by destabilizing a histone acetyltransferase (p300) making UV-damage more mutagenic. This has been hypothesized to augment the oncogenic potential of UV and contribute to non-melanoma skin cancer development. α-HPVs express two oncogenes (α-HPV E6 and E7). α-HPV E7 induces a replication stress, while α-HPV E6 prevents cells from responding to that stress. This sensitizes cervical cancers (caused almost exclusively by α-HPV infections) to drugs that cause replication stress.

Zoom information posted below. Nick is currently a candidate for tenure and promotion. If you would like to visit with Dr. Nick Wallace, please contact him at nwallac@ksu.edu.