Overweight and obesity are public health concerns of epidemic proportions, increasing the risk of chronic illnesses such as diabetes and cardiovascular, and various types of cancer. Obesity has been linked to over 13 different cancers, including liver cancer, which comprises about 40% of all cancers diagnosed in the U.S., making it the second leading cause of cancer after smoking. Obesity-related body fatness has been identified as a risk factor in the development of non-alcoholic fatty liver disease and its progression to hepatocellular carcinoma. Obesity is associated with changes in physiological functions of adipose tissue resulting in excess production of growth hormones and adipocytokines, and chronic inflammation which fan the flames of cancer. The primary role of my laboratory is to characterize and assess the physiological impact of adipocytokines on liver cancer; a type of cancer that has not decreased in incidence or mortality. Specifically, we are interested in visfatin and resistin; two inflammation-associated adipocytokines which are found at higher levels in both the obese state and liver cancer. Using an in vitro model of obesity and liver cancer, our laboratory has demonstrated that visfatin and resistin differentially activate pro-tumorigenic Akt and Erk signaling pathways leading to adverse physiological changes in liver cancer cells. By identifying the key pathways underlining the obesity-liver cancer link, studies can be designed to investigate the reversibility of the pro-tumorigenic effects of obesity.