Establishment and characterization of a novel murine model for head and neck cancer cachexia

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Cancer cachexia is the metabolic wasting syndrome that the cancer will release a lot of cytokines and result in metabolic abnormalities and anorexia. The rate of the cancer death has been revealed almost 30% in the cachexia patients. Cancer cachexia can vary according to tumor type, site, mass, and host genotype. Clinical studies showed that more than 60% of head and neck cancer (HNC) patients might develop cancer cachexia. Several animal models have been established to elucidate the importance of pro-cachectic cytokines, such as TNF-α and interleukin 6 (IL-6) in the pathogenesis of cancer cachexia. Unfortunately, the pathogenesis of HNC cachexia is still unknown. Our preliminary results demonstrated that IFIT2, an interferon-induced protein with tetratricopeptide repeat 2 (IFIT2) depletion enhances expression of TNF-α, a well-known cancer-cachexia related cytokine in HNC cells. Thus, this study aims to explore the effect of IFIT2 depletion on HNC cachexia. To the end, a murine model was established by injecting the IFIT2 depleted HNC cells. Moreover, the body weight and survival rate were significantly decreased in IFIT2-depleted cells bearing mice as compared to control mice. The quadriceps had a 28.6% reduction in cachectic mice. Similarly, the gastrocnemius had a 33.3% reduction in cachectic mice. These results suggest that IFIT2-depleted HNC cells bearing mice may act as a model for studies on HNC cachexia.