Cancer-induced cachexia occurs in 20%–80% of patients depending on the type and stage of cancer. Cachexia can reduce patient resilience and survival. This disease currently has no cure; thus, mechanisms of muscle dysfunction need to be further identified. We evaluated muscle force, mass and mitochondrial bioenergetic responses to cancer in locomotor and respiratory muscles to determine how cachexia progresses in different muscle types. In the C26 tumor-bearing mouse model, we observed that muscle weakness preceded atrophy at early stages of disease progression. In addition, locomotor muscles exhibited reduced mitochondrial respiration, whereas respiratory muscles exhibited increased markers of oxidative stress. During late-stage disease, atrophy was observed in both muscle groups, while specific force production recovered only in locomotor muscle. These findings highlight heterogeneous muscle dysfunction in the progression of cachexia that may be attributable to muscle-specific mitochondrial responses.