Duchenne muscular dystrophy (DMD) is caused by the absence of functional dystrophin protein resulting in a host of secondary pathologies, however, the role of autophagy in dystrophic pathology is unclear. We hypothesized that autophagic dysfunction would increase with disease severity. To test this hypothesis markers of autophagy initiation and flux were measured in the diaphragms of 7-week and 17-month old dystrophin deficient mdx and healthy C57 mice. In the mdx diaphragm upstream activation was suppressed, however, initiation was strongly induced compared to healthy muscle, independent of disease severity. Despite this, similar increases in LC3II and accumulation of p62 in dystrophic muscle indicate autophagic flux is impaired independent of disease progression. Histological analysis showed p62 accumulation in dystrophic muscle along with suppression of the lysosomal marker Lamp2, indicating autophagosomes are accumulating without subsequent lysosomal degradation. Surprisingly, confocal microscopy revealed extracellular p62 positive foci of similar size to autophagosomes, raising the possibility autophagosomes escape from dystrophic muscle. To further investigate the fate of these autophagosomes differential centrifugation of media from C2C12 myotubes was used to isolate released vesicles of different sizes and then imaged by scanning electron microscopy. We identified vesicles similar in morphology and size to autophagosomes. To confirm that part of the extracellular vesicle population released from muscle includes autophagosomes, healthy and dystrophic diaphragms were incubated in an oxygenated Krebs bath for 2 hours, and vesicles were isolated from the Krebs buffer and incubated with a dye that fluoresces upon incorporation into autophagosomes. Using flow cytometry, we confirmed that healthy muscle releases autophagosomes, but significantly more are released from dystrophic muscle. In conclusion, autophagic dysfunction occurs in dystrophic diaphragm independent of disease severity. Further, we propose healthy muscle releases autophagosomes and that this process becomes dysfunctional in dystrophin-deficient skeletal muscle and may contribute to disease severity.