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The specific autophagic elimination of mitochondria (mitophagy) plays the role of quality control for this organelle. Deregulation of mitophagy leads to an increased number of damaged mitochondria and triggers cell death. The deterioration of mitophagy has been hypothesized to underlie the pathogenesis of several neurodegenerative diseases, most notably Parkinson disease. Although some of the biochemical and molecular mechanisms of mitochondrial quality control are described in detail, physiological or pathological triggers of mitophagy are still not fully characterized. Here we show that the induction of mitophagy by the mitochondrial uncoupler FCCP is independent of the effect of mitochondrial membrane potential but dependent on acidification of the cytosol by FCCP. The ionophore nigericin also reduces cytosolic pH and induces PINK1/PARKIN-dependent and -independent mitophagy. The increase of intracellular pH with monensin suppresses the effects of FCCP and nigericin on mitochondrial degradation. Thus, a change in intracellular pH is a regulator of mitochondrial quality control.