The authors have attempted to compare aging mitochondria with mitochondria in Friedreich ataxia (FA). There are certain identities that are of interest though FA is not due to aging, or accelerated aging. The paper shows major deficiencies that would preclude publication. Throughout the manuscript, Friedreich ataxia is misspelled as Friedrich ataxia. In the abstract, the authors write that the frataxin gene is located in the mitochondria. The frataxin gene resides in the nuclear genome, not in mitochondrial DNA. FA may not be a neurodegenerative disease though this language is widely used. It is a developmental disease though some lesions are truly atrophic, hence, degenerative. Also in the introduction, what is the evidence for protein misfolding in FA? The mutation is GAA trinucleotide repeat expansion, not just "GAA repeats". The three illustrations are attractive but should be labeled by figure1, figure 2, and figure 3; and they should display legends. The authors expect too much from  readers who do not work with mitochondria. Not all readers will understand the abbreviations of ROS. VOmax, OXPHOS. T2D, and ECT. A list of abbreviations would be helpful, and at a minimum,
abbreviations should follow full spellings when used first in the text. The paper is not paginated, Therefore, this reviewer has labeled them as 1-11. On page 6, paragraphs 2 and 3, a duplication has occurred, namely, "decreased cellular respiration, O2 utilization, and decreased ATP production" (para 2) and "lower rate of oxygen consumpiton" (para 3). Figure 3 displays images of heart, brain, and pancreas to its right, without explanation. On page 7, para 3, the statement that "cardiomyopathy is one of the primary derangements seen in FA" is difficult to understand. Why is cardiomyopathy "primary"? Finally, on page 8, the authors assert that FA mitochondria and aging mitochondria share "irregular iron homeostasis" but such evidence is insufficient, notably when the roles of hepcidin and ferroportin are omitted. This reviewer recommends more attention to the notion that iron homeostasis may antedate mitochondrial aging and mitochondrial dysfunction in FA.