INTRODUCTION:
The re-tear rates after arthroscopic rotator cuff repair surgery for degenerative rotator cuff tears are reported to range from 10-94%[1]. There have been findings which suggest that oxidative stress in tissues plays a role in degenerative rotator cuff tears[2]. Mitochondria, while producing reactive oxygen species during the process of energy generation, possess the anti-oxidative super-oxide dismutase (SOD), which helps in mitigating self-damage. Increase in reactive oxygen species due to endogenous or exogenous factors can lead to mitochondrial DNA damage, resulting in a decrease in mitochondrial function. This causes a reduction in ATP production capability and excessive production of reactive oxygen species, ultimately leading to tissue damage and degeneration. To the best of our knowledge, there are scant reports investigating the decline in mitochondrial function in relation to human rotator cuff tears. Meanwhile, recent attention has been drawn to the Stump classification using MRI[2] fat-suppressed imaging[3]. It has been reported that the re-tear rate following arthroscopic rotator cuff repair surgery is significantly higher in Stump type 3, and this is expected to serve as an indicator of vulnerability that can be evaluated preoperatively. This study evaluated the mitochondrial function in the degeneratively torn human rotator cuff tissues of Stump classification types 1 and 3 and investigated the relationship between mitochondrial function and degenerative rotator cuff tears.

MATERIALS AND METHODS:
The subjects were 10 non-traumatic human tendon rupture surgical specimens in total, including 5 cases of Stump type 1 (average age 64.5 years) and 5 cases of Stump type 3 (average age 65.5 years). Histological evaluation by the modified Bonar Score, immunohistochemical staining, TUNEL staining, SOD activity by a kit, and evaluation of the expression of mitochondrial function-related genes such as ATP5A and SOD2, and apoptosis-related gene expression such as BAX, Bcl2 by real-time PCR were performed on the supraspinatus tendon stumps collected during arthroscopic rotator cuff tear surgery. Furthermore, the morphology of the mitochondria in each case was observed using an electron microscope.

RESULTS:
Compared to Stump type 1 tissue specimens, Stump type 3 tissue specimens had a significantly higher modified Bonar Score, lower expression of mitochondrial function genes by PCR(Fig1), and significantly more TUNEL-positive cells(Fig2). Additionally, SOD activity in the tissue was lower in Stump type 3. Evaluation with an electron microscope revealed a greater number of mitochondria with reduced cristae in the Stump type 3 specimens(Fig3).

DISCUSSION:
Stump type 3 showed advanced degeneration by histological evaluation, and mitochondrial dysfunction was suggested by PCR and SOD activity. Also, there was increased apoptosis observed from the increase in the expression of apoptosis-related genes such as BAX, Bcl2 and the results of TUNEL staining. From the above, it was considered that tendon vulnerability in Stump type 3 is related to mitochondrial dysfunction.

SIGNIFICANCE:
This study provides compelling evidence linking mitochondrial dysfunction to tendon vulnerability in Stump type 3 degenerative rotator cuff tears, presenting a potential diagnostic and therapeutic target.

REFERENCE: