Supplementary Information (SI) for Nanoscale.
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SUPPLEMENTARY DATA

I. TEM image of R-HAp Nps

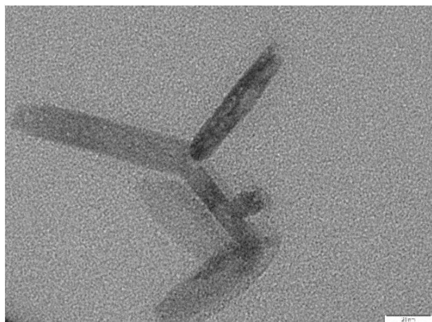

Figure (i): HR-TEM micrograph of HAp nanoparticles

II. Transfection efficiency of R-HAp Nanoparticles in C2C12 cells and Human Skeletal Muscle cells

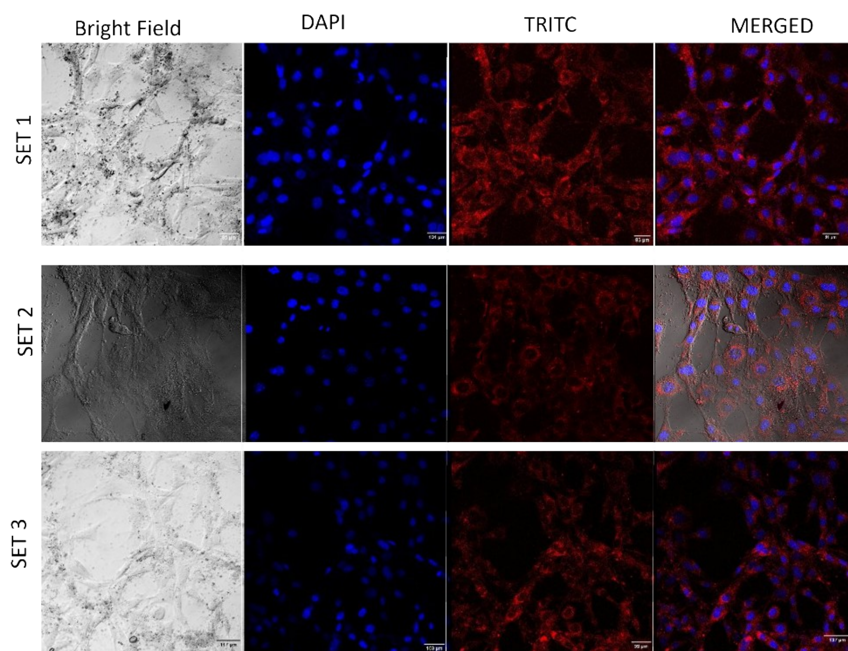

Figure ii (a): Confocal images for C2C12 cells transfected with DMD plasmid using R-HAp nanoparticles.

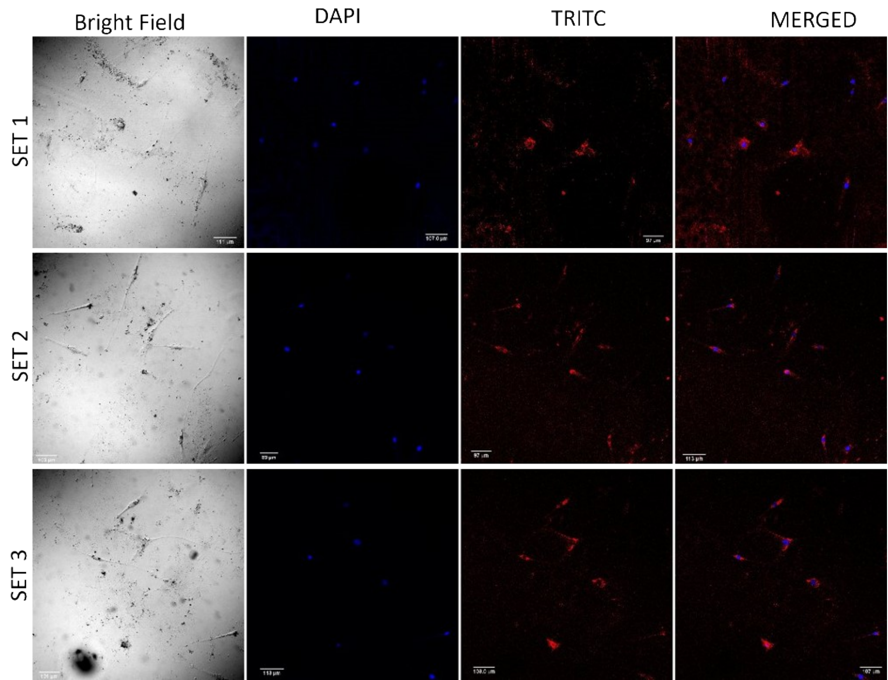

Figure ii (b): Confocal images for human skeletal muscle cells transfected with DMD plasmid using R-HAp nanoparticles.

III. WESTERN BLOT

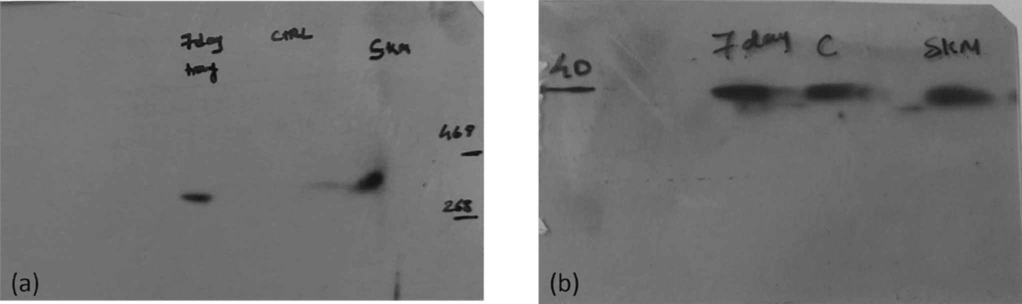

Figure iii (a) Western Blots of (a) Dystrophin (b) β-actin

IV. EXON 44 MUTATION

Methodology : To confirm that the exon 44 mutation in DMD positive cells did not affect the mRNA splicing in patient cells, we designed primers spanning from exon 43-45 (Primer

sequences are provided below). The expected product size is 236 base pairs. Since the length of exon 44 is 148 bp, exon 44 skipping would have produced a shorter product length (88 bp) and would have been detected by RT-PCR analysis using these primers. cDNA was synthesized from mRNA extracted from the normal human skeletal muscle cells and DMD patient cells followed by PCR amplification using these primers (Annealing temperature: 54°C). GAPDH expression was analyzed as an internal control.

Target Gene	Gene Primer	Product size
Exon 44	F: 5'AGG AAG CTC TCT CCC AGC TT-3' R: 5' CCCAATGCCATCCTGGAGTT-3'	236

Note: The primers used for GAPDH were the same as described before.

Results:

The product length obtained on amplification from patient cells was the same as compared to the normal skeletal muscle cells (236 bp) as seen from Figure (iv), which included exon 44. No shorter product in the patient sample was evident by this analysis, although the extent of expression was lower.

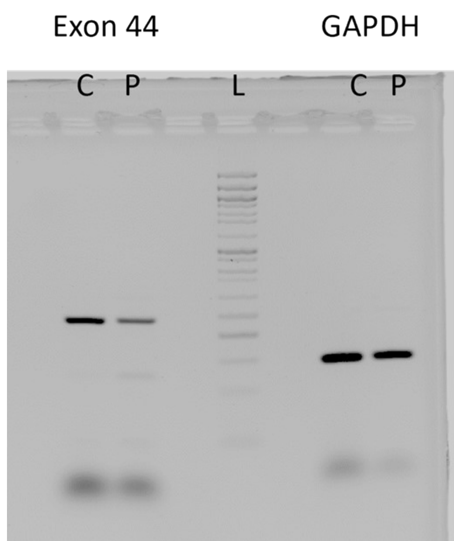

Figure iv: RT-PCR analysis with primers spanning from Exon 43-45. C: Normal human skeletal muscle cells, P: DMD patient cells