DYSTROGEN THERAPEUTICS

# Clinical Stage Chimeric Cell Therapy Company

**Chicago, Illinois**

**April 2023**

# Chimeric Cell Therapy Demonstrated to be Safe and Effective with Dependable Manufacturing and Cell Delivery Process

- ✔ Excellent long-term safety and tolerability profile in all patients (N=6) to date
- ✔ Consistent functional outcome improvements in all evaluated patients
- ✔ Reliable and reproducible chimeric cell manufacturing process with 100% rate of manufacturing completion
- ✔ 100% therapy administration rate

# Dystrogen Therapeutics - Clinical Stage Chimeric Cell Therapeutics Company

- *Novel chimeric cell therapy platform for musculoskeletal, neurodegenerative and hematopoietic indications*

- *Lead program, DT-DEC01,* ***in clinical trial (6 patients have been dosed to date; plan for additional 4); no AE, no SAE; efficacy demonstrated at 6 months and 12 months***

- *Discovery stage for additional indications with opportunity to broaden across a variety of disorders such as ALS, muscular dystrophies, sarcopenia/"anti-aging", post-radiation hematopoietic reconstitution*

- *Distinctive advantages of DT-DEC01 over competitive approaches for muscular dystrophy:*
  - ***Universal therapy for all Duchenne's genetic mutations***
  - ***Does not require immunosuppression, does not cause immune reaction***
  - ***Excellent safety profile - no AE or SAE noted in clinical trial to date, none in pre-clinical***
  - ***No genetic manipulation, no viral delivery required***
  - ***Novel chimeric cell approach - no other companies developing***
  - ***Technology licensed from the University of Illinois***

- *Robust IP estate with patents that expire in 2036*

# Experienced Company Founders

## Prof. Maria Siemionow MD, PhD

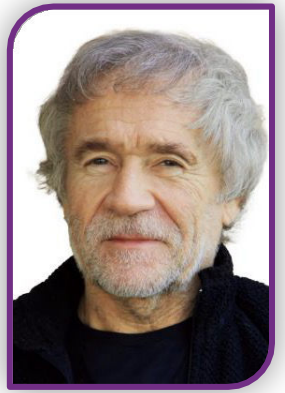
**FOUNDER**
**CHIEF MEDICAL OFFICER**

- Professor of Orthopaedics, Director of Microsurgery Research, the University of Illinois in Chicago
- Lead surgeon of team that performed the world's first near-total face transplantation, December 2008
- Over 300 scientific publications in the peer reviewed scientific journals

## Dr. Kris Siemionow MD, PhD

**CO-FOUNDER**
**CHIEF EXECUTIVE OFFICER**

- Past Chief of Spine Surgery and Associate Professor of Orthopaedics and Neurosurgery at the University of Illinois in Chicago
- Past Co-founder Holosurgical (AI for neurosurgery : acquired
- Past Co-founder Inteneural Networks (Brain MRI analytics); acquired
- Past Chief Medical Officer NASDAQ medtech
- Over 100 scientific publications



## Prof. Paul Lewicki PhD

**CO-FOUNDER**
**DIRECTOR**

- Founder and CEO of StatSoft (acquired by Dell in 2014)
- Big Data Pioneer
- Entrepreneur, large multinational company CEO (StatSoft had 30 overseas offices in all major markets and over 1 M B2B users across various industries)

CNN NATIONAL GEOGRAPHIC The New York Times

HOLOSURGICAL Surgalign INTELNEURAL

StatSoft STATISTICA

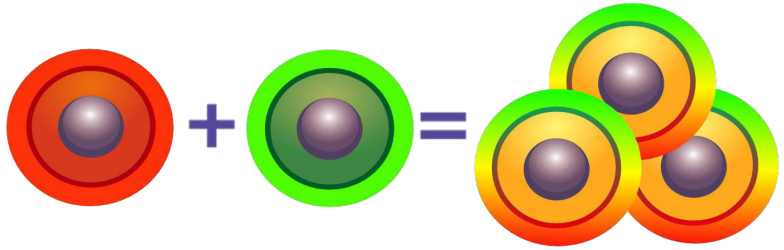
# Chimeric Cell Therapy – A Novel Approach

**Prof. Maria Siemionow**

**Inventor**

- Born out of transplantation research to identify ways to evade host rejection
- Approach for DMD catalyzed through Armed Forces Institute of Regenerative Medicine (AFIRM) collaboration
- 10+ years of scientific studies
- Patented technology by prof. Maria Siemionow (global protection)

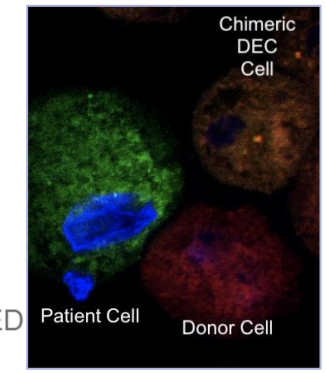
- **CHIMERIC CELLS** are created by fusing the patient's diseased cells with healthy cells from a donor. Dystrogen invented this approach



DONOR CELL STAINED **RED**

PATIENT CELL STAINED **GREEN**

CHIMERIC CELLS STAINED **ORANGE** (fusion of **RED** and **GREEN** cells)




- Chimeric cell contains **healthy organelles from donor**
- **Chimeric cells restore function and can prolong longevity**
- **"Tricks" the immune system** into accepting the cell (Trojan horse approach), as chimeric cells appear to the recipient as self
- Chimeric cells have regenerative and immunomodulatory properties
- Chimeric cells **do not create an immune response** (clinically validated)
- A novel approach to mitigate the side effects of immunosuppression and conditioning regimens as well as to **enhance cell engraftment**
- **No cancer** demonstrated in small and large animal model

11 Peer Reviewed Publications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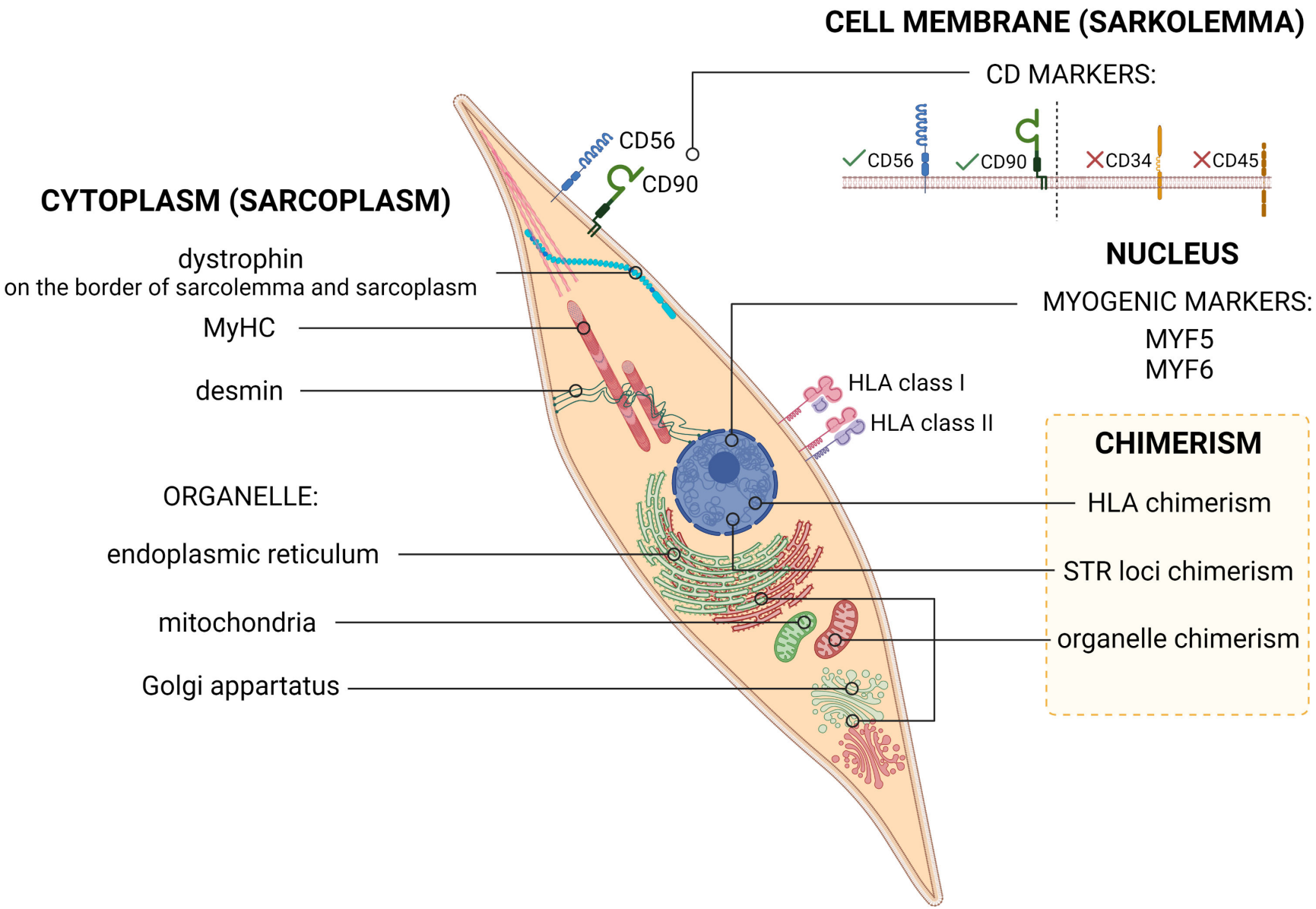
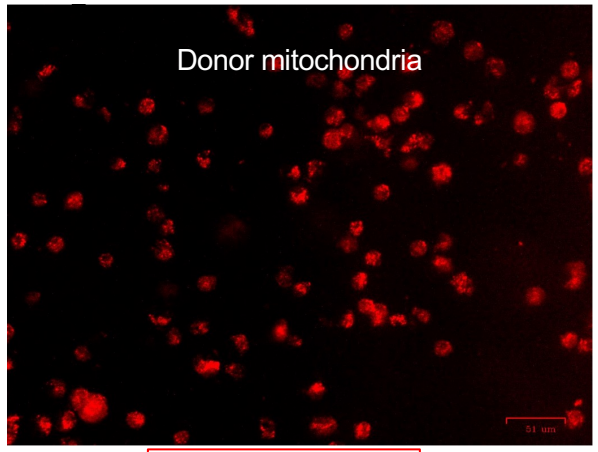
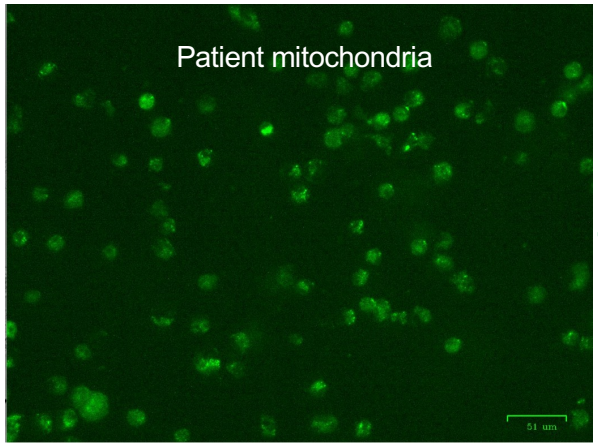
# Dystrophin Expressing Chimeric (DEC) Cell



# DT-DEC01 Cells Contain Healthy Mitochondria and other Organelles from the Donor

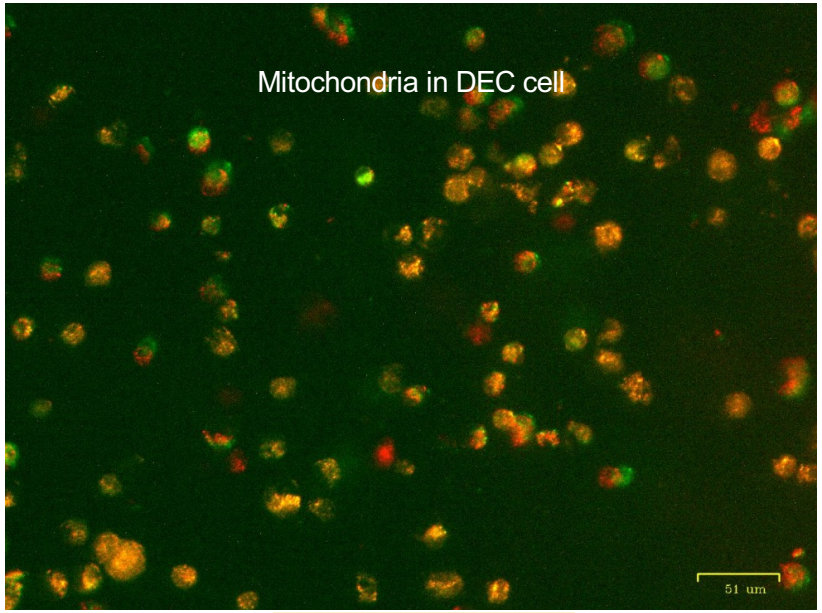



Red PKH26



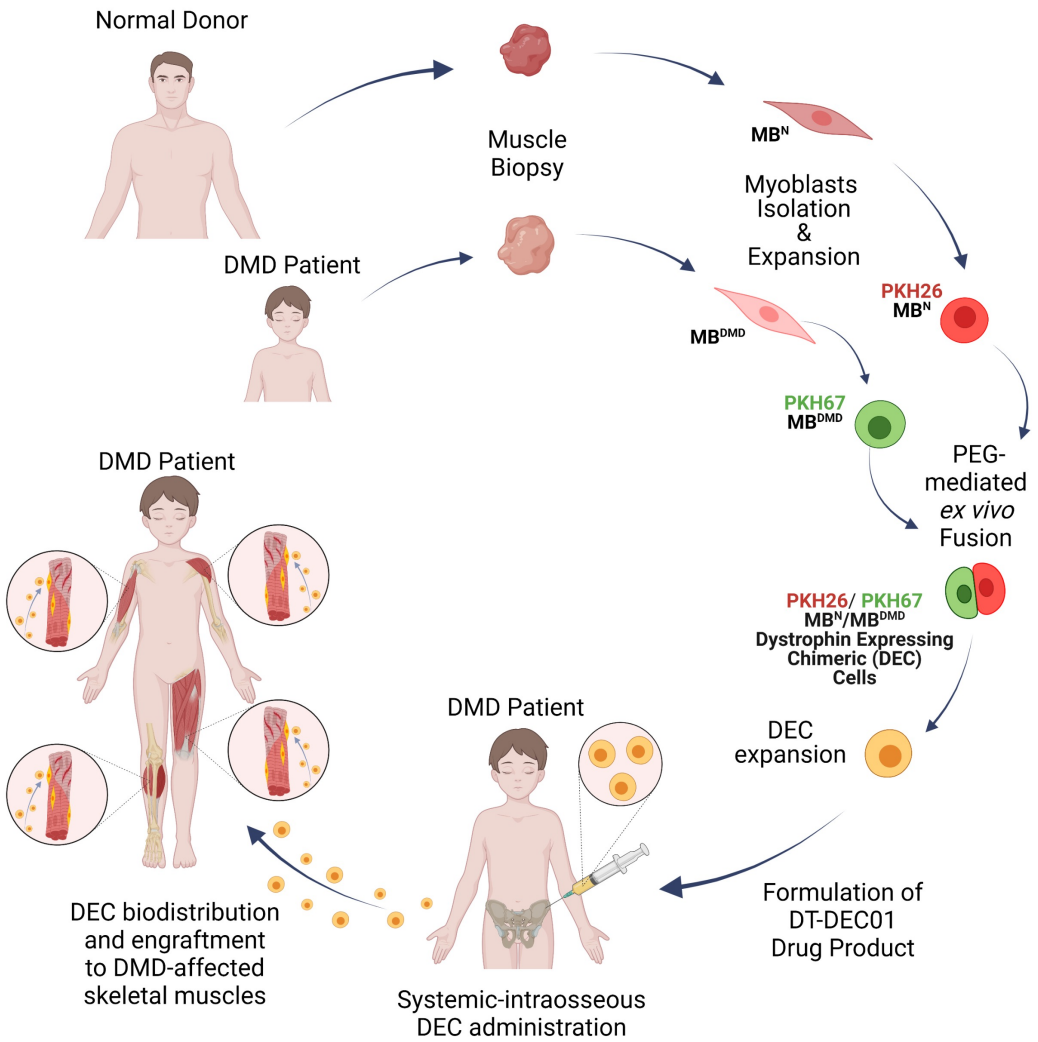

Green PKH 67

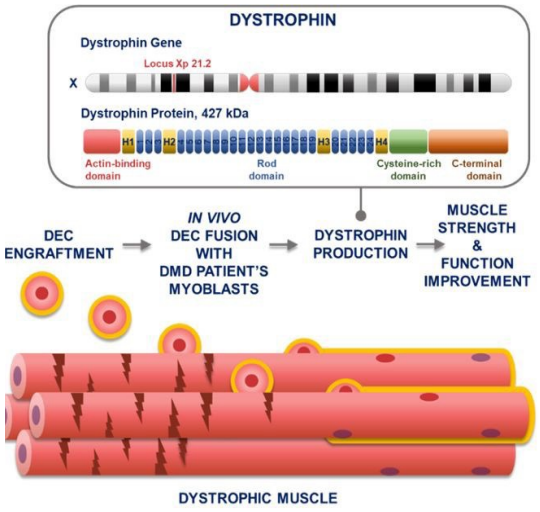
Fusion




Orange PKH 26/PKH 67

# Mechanism of Action Based on Full Length Dystrophin Gene Delivery




## Significant Advantages

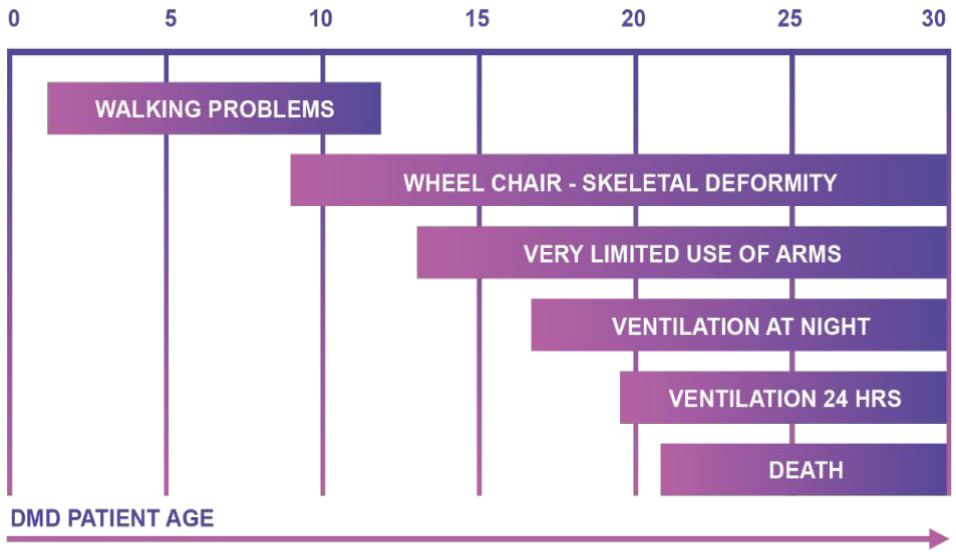
- ***Universal therapy for all Duchenne's genetic mutations***
- ***Does not require immunosuppression***
- ***Excellent safety profile beyond 12 months***
- ***Efficacy demonstrated in addition to a lack of disease progression***
- ***Novel chimeric cell approach - no other companies developing***

# Duchenne Muscular Dystrophy: A Disease Without A Cure

## Disease Overview

- Orphan disease with devastating outcomes for the patient and their families alike
- Most common lethal X-chromosome linked progressive muscle-wasting disorder
- Over 20,000 boys are living with the disease in the US and over 300,000 worldwide
- Caused by a variety of dystrophin gene mutations
- No cure currently exists
- Prior and current technologies have had safety, efficacy, and/or patient population limitations
- With no cure or viable remedies available, the massive $4+ billion DMD market is prime for disruption

## Patient Physical Degeneration



# Dystrogen Therapeutics Duchenne Muscular Dystrophy Patients Treated with DT-DEC01

| Patient Identifier | Age at Dosing | DT-DEC01 Cohort | Ambulatory Status and Genetic Profile | Follow Up Duration |
|---|---|---|---|---|
| **02B** | 7 | Low Dose (1M-2M DEC cells per kg) | **Ambulatory** (Deletion Exon 3-12) | *18 months* |
| **03B** | 16 | Low Dose (1M-2M DEC cells per kg) | **Non-Ambulatory** (Deletion Exon 48-50) | *14 months* |
| **04B** | 6 | Low Dose (1M-2M DEC cells per kg) | **Ambulatory*** (Nonsense mutation in gene DMDc.9249G>A(p.TRP3083Ter) | *13 months* |
| **05B** | 12 | Medium Dose (2M- 4M DEC cells per kg) | **Non-Ambulatory** (Deletion Exon 44) | *10 months* |
| **06B** | 6 | Medium Dose (2M- 4M DEC cells per kg) | **Ambulatory** (Deletion Exon 44) | *4 months* |
| **01B** | 7 | Medium Dose (2M- 4M DEC cells per kg) | **Ambulatory** (Deletion of exons 20-29) | *3 month* |

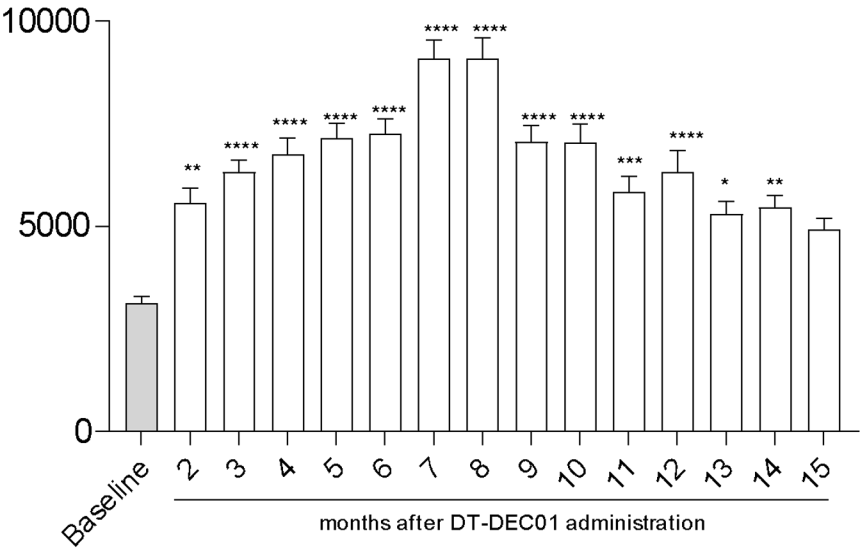
*Had recovered from Covid19 prior to dosing

# Clinical Trial Summary to Date (N=6)

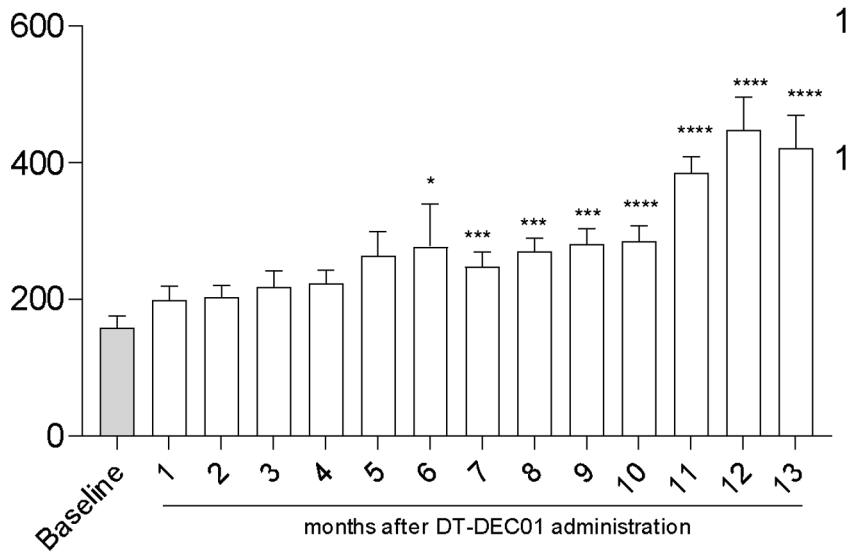
- Results continue to reinforce safety and tolerability profile of DT-DEC01 up to 14 months (eg. no AE, no SAE; negative anti-donor antibodies test up to 12 months)
- Demonstrated consistent functional outcome **improvements** in low dose cohort **at 3 months, 6 months (n=4), and 12 months (N=2)** after DT-DEC01 administration:
  - Electromyography (EMG) (objective test, not effort dependent)
  - Strength and fatigue resistance
  - Mobility and Function
  - Improvements on objective tests (EMG) correlate with improvements in strength (ex. grip strength, shoulder abduction) and functional tests (ex. **6MWT** – 6 minute walking test, **NSAA** – North Star Ambulatory Assessment, **PUL** – Performance of Upper Limb)
  - Functional improvements in clinical trial correlate with those reported in pre-clinical studies
  - Demonstrated DT-DEC01 to be a universal therapy for all Duchenne genetic mutations (5 different mutations in current trial)

# Garmin pedometer, steps per day
## (patient 2, daily arm movement)
Monthly averages with standard error

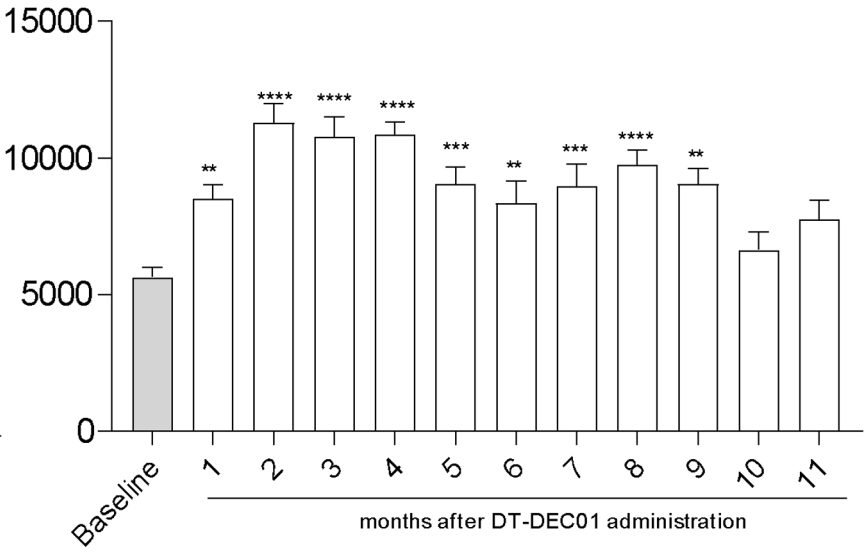
**Patient 1**
**Ambulatory**
**Steps**




**Patient 2**
**Non-Ambulatory**
**Arm movements**




**Patient 3**
**Ambulatory**
**Steps**

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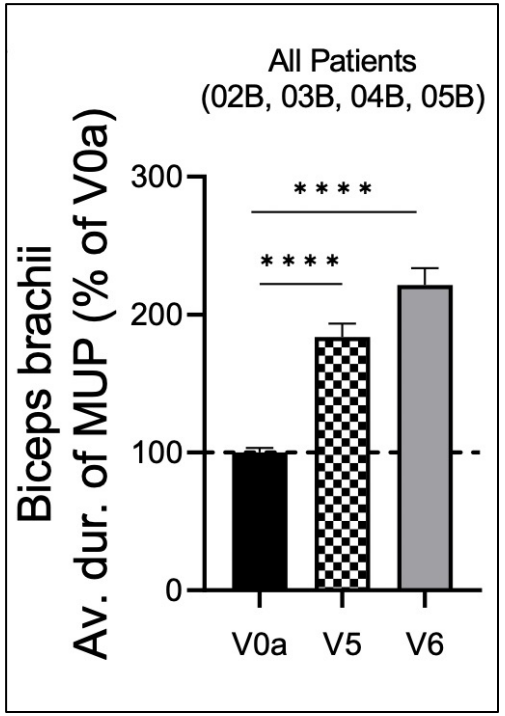
* P≤0.05, ** P≤0.01, *** P≤0.001, **** P≤0.0001, Kruskal-Wallis test

# Selected Results of Upper Extremity Strength (Grip), EMG (Biceps), and Functional Test (Performance of Upper Limb) at 6 Months Demonstrate both Clinically and Statistically Significant Improvements in Ambulatory and Non-Ambulatory patients with DMD

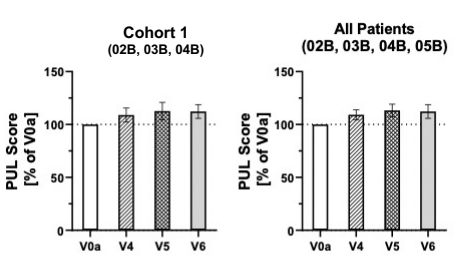
## Biceps Electromyography




**Changes in biceps motor unit potential versus V0a. Biceps brachii (at 3-months visit: 84% ± 9.6% above baseline value ; at 6-months visit: 122% ± 12.2% above baseline value )** All values: mean ± SEM

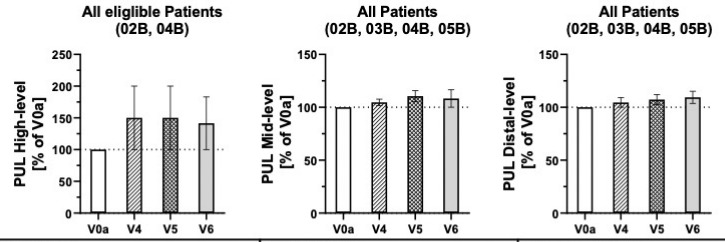
## Performance of Upper Limb

**A) Performance of Upper Limb**




| 1 Cohort, % of V0a | All Patients, % of V0a |
|---|---|
| V4 108.96% ±6.62 n=3, | V4 109.22% ±4.69 n=4, |
| V5 112.71% ±8.23, n=3; | V5 113.28% ±5.85, n=4; |
| V6 112.29% ±6.46 n=3. | V6 112.29% ±6.46 n=3. |

**B) Performance of Upper Limb – scoring in three domains**

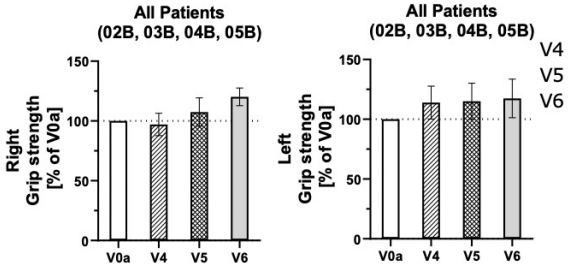



| MID-LEVEL All Patients* (02B, 04B), % of V0a | MID-LEVEL All Patients, % of V0a | DISTAL-LEVEL All Patients, % of V0a |
|---|---|---|
| V4 150.00% ±50.0 n=2, V5 150.0% ±50.00, n=2; V6 141.67% ±41.67 n=2 *Mean from 2 patients, this values are rather illustrative | V4 104.69% ±2.99 n=4, V5 110.59% ±5.31, n=4; V6 108.33% ±8.33 n=3. | V4 104.55% ±4.55 n=4, V5 107.27% ±4.75, n=4; V6 109.44% ±5.80 n=3. |

**Changes in PUL versus V0a.** All values: mean ± SEM.

## Grip Strength

**Grip strength**



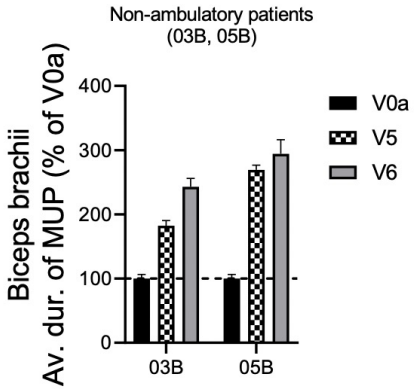

**RIGHT**
All Patients, % of V0a
V4 97.00% ±9.44 n=4,
V5 107.48% ±11.94, n=4;
V6 120.23% ±7.39 n=3.

**LEFT**
All Patients, % of V0a
V4 113.90% ±13.75 n=4,
V5 115.05% ±15.13, n=4;
V6 117.40% ±16.12 n=3.

**Figure 9. Changes in right and left grip strength versus V0a.** All values: mean ± SEM.

# Select Results of Upper Extremity Strength (Grip), EMG (Biceps), and Functional Test (Performance of Upper Limb) at 6 Months Demonstrate both Clinically and Statistically Significant Improvements Non-Ambulatory patients with DMD

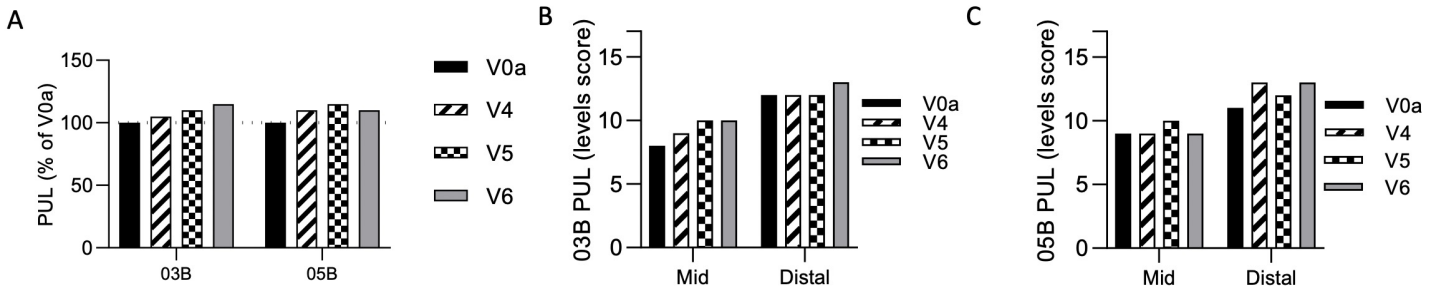
## Biceps Electromyography




**03B** biceps brachii
82% above baseline (V0a) at 3 - month (V5) visit
143% above baseline (V0a) at 6 - month (V6) visit

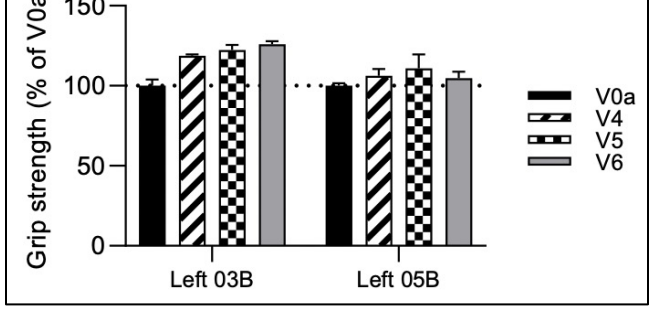
**05B** biceps brachii
170% above baseline (V0a) at 3 - month (V5) visit
194% above baseline (V0a) at 6 - month (V6) visit

## Performance of Upper Limb




The assessments of PUL in **non-ambulatory** patients: (**A**) comparison of changes in PUL total score when compared to baseline (V0a) values shows that the patient 03B improves continuously on consecutive visits (V0a: 100%, V4: 105%, V5: 110%, V6: 115%), while the patient 05B reached his maximum score on 3-months visit and then plateaued (V0a: 100%, V4: 110%, V5: 115%, V6: 110%). (**B**) PUL level scores for patient 03B – Mid and Distal demonstrate continuous improvement, (**C**) PUL level scores for patient 03B – Mid improve then plateaued and Distal demonstrate continuous improvement.

## Grip Strength




Assessments of left hand grip strength in non-ambulatory patients demonstrates improvement over baseline (V0a) at all time points

# Dystrogen Therapeutics: Pioneer of Chimeric Cell Therapy

Chimeric cell therapy represents a completely unique and novel modality

### *Novel modality for the treatment of a broad spectrum of genetic disorders*

- ✓ Initially targeting Duchenne muscular dystrophy, a rare, debilitating disease with no cure
- ✓ Also targeting neurodegenerative diseases, sarcopenia, and graft-vs-host disease
- ✓ Broad potential applicability to a variety of other genetic disorders

### *Six patients dosed with compelling initial results; enrolment ongoing*

- ✓ No safety issues and demonstrated functional improvement 6 months from dosing
- ✓ Patients demonstrating improvements in quality of motion, EMG, and other functional measures
- ✓ Additional patients identified and to be dosed

### *Orphan Drug and Rare Pediatric Disease Designations*

- ✓ Orphan and Rare Pediatric designation granted in the US for DMD
- ✓ Priority Review Voucher eligible – PRVs have sold >$100mm

### *Global DMD drug market opportunity expected to reach ~$4B by 2023(1)*

- ✓ DMD is an orphan indication with high unmet need
- ✓ Prior and current therapeutics under development have had safety and efficacy issues
- ✓ Chimeric cell therapy has numerous advantages over existing therapeutics under development

### *Robust IP protection through 2036*

- ✓ Protected by multiple layers of granted and pending patents
- ✓ Provides extensive IP protection around formulation and methods of use
- ✓ Substantial potential for patent term extension

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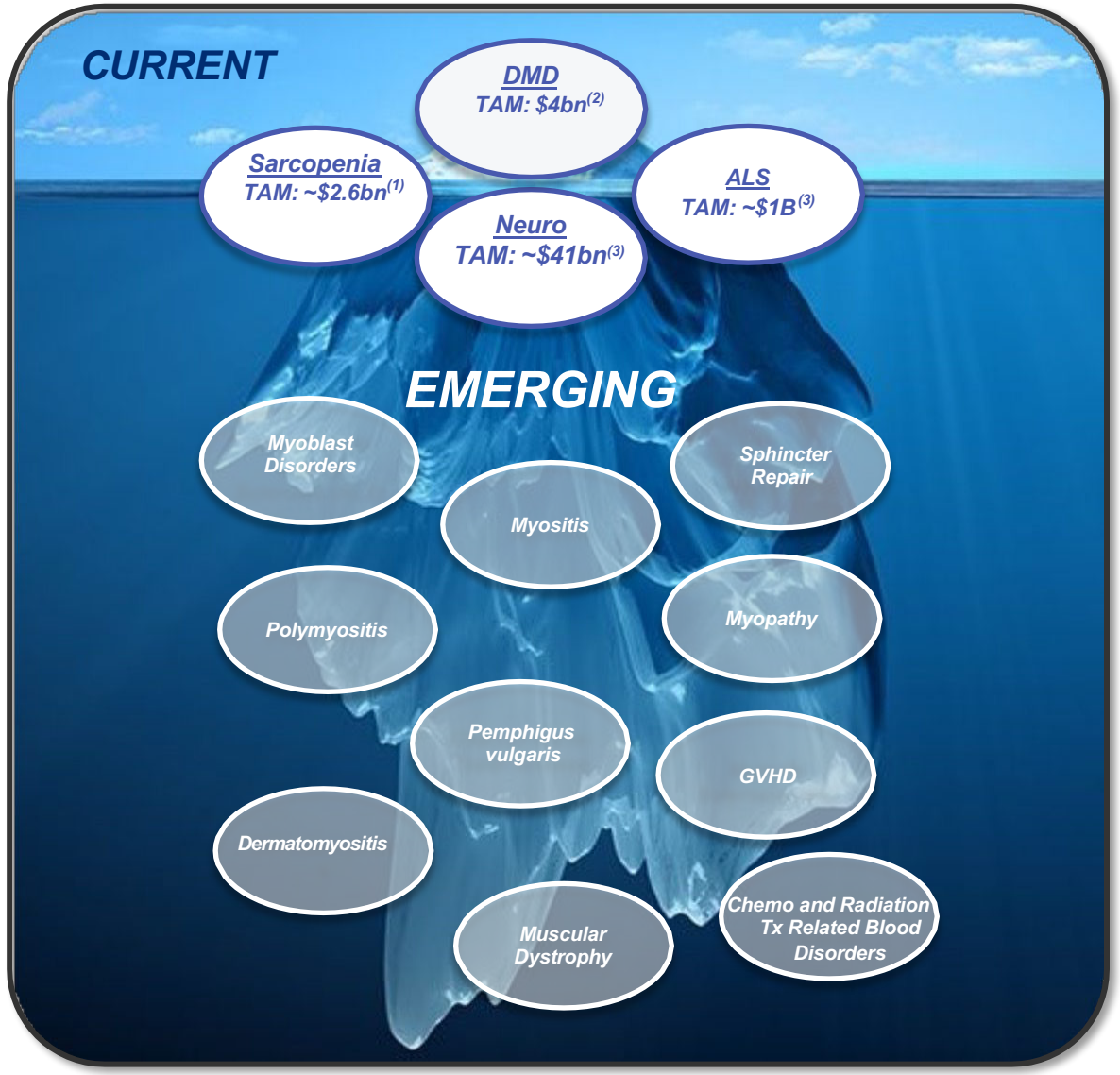
(1) Grand View Research: "Duchenne Muscular Dystrophy (DMD) Drugs Market Size, Share & Trends Analysis Report By Therapeutic Approach (Mutation Suppression, Exon Skipping, Steroid Therapy) And Segment Forecasts, 2018 – 2023" (2018).

# Platform and Technology

# Dystrogen Therapeutics Pipeline




| Program | Indications | Phase |
|---|---|---|
| DT-DEC01 | DMD | Phase 1 |
| DT-200 | ALS | Preclinical |
| DT-201 | Sarcopenia and muscle “anti-aging" | Discovery |
| DT-202 | Neurodegenerative | Discovery |

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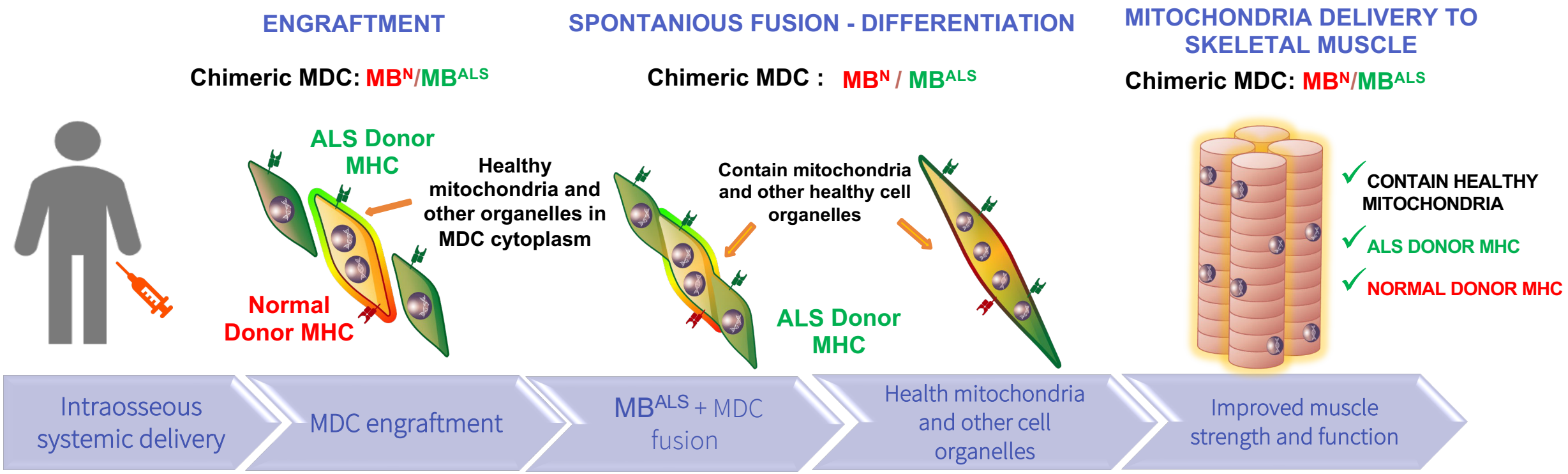
(1) Allied Market Research: “Sarcopenia Treatment Market” (2021).
(2) Grand View Research: “Duchenne Muscular Dystrophy (DMD) Drugs Market Size, Share & Trends Analysis Report By Therapeutic Approach (Mutation Suppression, Exon Skipping, Steroid Therapy) And Segment Forecasts, 2018 – 2023” (2018).
(3) Fortune Business Insights: “Neurodegenerative Diseases Drugs Market” (2019).

# Mitochondria Delivering Chimeric Cells (MDC) - Mechanism of Action in Amyotrophic Lateral Sclerosis (ALS)




Muscle metabolism and mitochondrial activity have been shown to be affected in ALS. Evidence of toxicity in skeletal muscle tissue has been demonstrated, including metabolic dysfunctions, impaired proteostasis, and deficits in muscle regeneration and RNA metabolism. The role of muscle as a secretory organ, and the negative effects on the skeletal muscle secretome has been demonstrated, including the increase in secretion of toxic factors or decrease in essential factors that have consequences for neuronal function and survival. Furthermore, ALS muscle vesicles are shown to be toxic to motor neurons, which establishes the skeletal muscle as a potential source of vesicle-mediated toxicity in ALS. Chimeric myoblasts have the potential to carry healthy mitochondria, ribosome vesicles, rough endoplasmic reticulum, Golgi apparatus, cytoskeleton, smooth endoplasmic reticulum, vacuole, cytosol, lysosome, centriole originating from the healthy donor myoblast

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1. J. Pers. Med. 2022, 12, 1868. Br J
2. Pharmacol.2021;178:1279–1297
3. Biochimica et Biophysica Acta 1802 (2010) 45–51

# Competitive Landscape

## Dystrogen Therapeutics: A Modality of its Own


(1) Sarepta is Phase 3 ready and plans to initiate its Phase 3 Study in September 2021.
(2) Solid Biosciences is Phase 1/2.

# Competitive Advantages of Chimeric Cell Therapy for DMD

| Features | DYSTROGEN DEC Therapy | Viral Gene Therapy | Steroids | ASOs | Stem Cell |
|---|---|---|---|---|---|
| Targets 100% DMD patients | ✓ | ✗ | ✓ | ✗ | ✓ |
| Functional Outcomes | ✓ | ? | ✗ | ? | ? |
| Dystrophin Expression | ✓ | ✓ | ✗ | ✗ | ? |
| No observed adverse effects | ✓ | ✗ | ✗ | ✗ | ✓ |
| No gene manipulation | ✓ | ✗ | ✓ | ✓ | ✓ |
| No need for immunosuppression | ✓ | ✓ | ✗ | ✓ | ✗ |

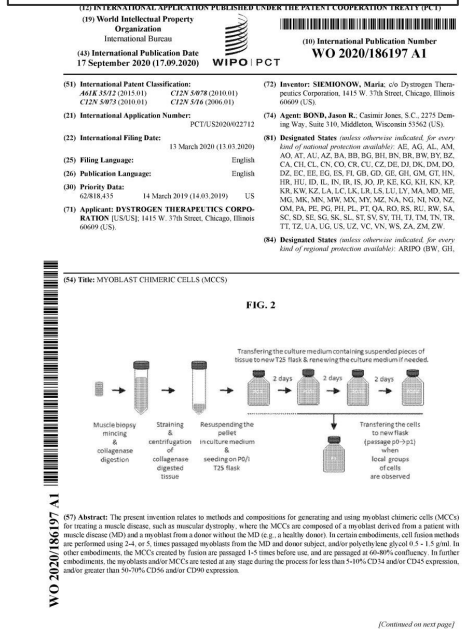
# Intellectual Property – Cellular Therapy Platform, Granted Patents

- **US Patent 11,147,840**
- Japanese Patent granted No. 2017-564642
- Canadian Patent granted No. 2988137
- Australian Patent granted No. 2016274797
- EU Patent Application No. 16808341.8 Notice of Allowance received
- PCT Application
- Filing date: 11 June 2015

  Title: Muscular Dystrophy Chimeric Cells and Method for Treating Muscular Dystrophies

  Published on: December 15, 2016 as WO/2016/201182
- Proprietary Fusion Technology Trade Secret
- Basic Science Pre-clinical Data
- Exclusive Worldwide License From University of Illinois, Chicago, USA

Myoblast chimeric cells
WO2020186197A1
2019-03-14
Priority to
US201962818435P


(12) INTERNATIONAL APPLICATION PUBLISHED UNDER THE PATENT COOPERATION TREATY (PCT)
(19) World Intellectual Property Organization
International Bureau
(43) International Publication Date
17 September 2020 (17.09.2020)
WIPO | PCT
(10) International Publication Number
WO 2020/186197 A1
(54) Title: MYOBLAST CHIMERIC CELLS (MCCS)
FIG. 2

(57) Abstract: The present invention relates to methods and compositions for generating and using myoblast chimeric cells (MCCs) for treating a muscle disease, such as muscular dystrophy, where the MCCs are composed of a myoblast derived from a patient with muscle disease (MD) and a myoblast from a donor without the MD (e.g., a healthy donor). In certain embodiments, cell fusion methods are performed using 2-4, or 5, times passaged myoblasts from the MD and donor subject, and/or polyethylene glycol 0.5 - 1.5 g/ml. In other embodiments, the MCCs created by fusion are passaged 1-5 times before use, and are passaged at 60-80% confluency. In further embodiments, the myoblasts and/or MCCs are tested at any stage during the process for less than 5-10% CD34 and/or CD45 expression, and/or greater than 50-70% CD56 and/or CD90 expression.
[Continued on next page]
WO 2020/186197 A1


(12) INTERNATIONAL APPLICATION PUBLISHED UNDER THE PATENT COOPERATION TREATY (PCT)
(19) World Intellectual Property Organization
International Bureau
(43) International Publication Date
15 December 2016 (15.12.2016)
WIPO | PCT
(10) International Publication Number
WO 2016/201182 A1
(54) Title: MUSCULAR DYSTROPHY CHIMERIC CELLS AND METHOD FOR TREATING MUSCULAR DY
(57) Abstract: A Muscul
meric Cell generated by a
myoblast with a second
chymal stem cell, or st
scribed as is the use of
treatment of a muscular d

FIG. 3A
WO 2016/201182 A1
JP 6890553 B2 2021. 6. 18
(19)日本国特許庁(JP)
(12)特 許 公 報(B2)
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最終頁に続く
(54)【発明の名称】筋ジストロフィーキメラ細胞および筋ジストロフィーの処置方法
(57)【特許請求の範囲】
【請求項1】
筋ジストロフィーを罹患した対象に由来する筋芽細胞および健康なドナーに由来する筋芽細胞間の融合を含むキメラ細胞であって、生体外融合産物である、前記キメラ細胞。
【請求項2】
筋ジストロフィーを罹患した対象に由来する筋芽細胞および健康なドナーに由来する筋芽細胞が、異なるドナーに由来する、請求項1に記載のキメラ細胞。
【請求項3】
筋ジストロフィーを罹患した対象に由来する筋芽細胞または健康なドナーに由来する筋芽細胞が、自己のものまたは同種である、請求項1に記載のキメラ細胞。
【請求項4】
健康なドナーが対象の父である、請求項1に記載のキメラ細胞。
【請求項5】
筋ジストロフィーを罹患した対象に由来するヒト筋芽細胞および健康なドナーに由来するヒト筋芽細胞間の融合を含む、請求項1に記載のキメラ細胞。
【請求項6】
請求項1に記載のキメラ細胞および薬学的に許容し得る担体を含む組成物。
【請求項7】
処置を必要とする対象に請求項1に記載のキメラ細胞の有効量を投与し、それによって対象の筋ジストロフィーを処置することを含む、筋ジストロフィーを処置する方法における

Summary -the US MDCC patent claims the cell made by an ex-vivo fusion of the first myoblast and the second myoblast, mesenchymal stem cell, or stromal cell. This is a “product-by-process” claim.

# Achievements and Upcoming Milestones

| Item | Completed | Next 6-12 Months |
|---|---|---|
| ATMP Classification – Tissue Engineering Product | ✓ | |
| Orphan Designation FDA | ✓ | |
| Rare Pediatric Disease Designation FDA | ✓ | |
| FDA PRE - IND Meeting | ✓ | |
| Launched POC Trial | ✓ | |
| First Patient Dosed | ✓ | |
| Full Data Set From First Patient | ✓ | |
| Low and Medium Dose Cohort Fully Dosed | ✓ | |
| Initiate Phase 1/2 | | |

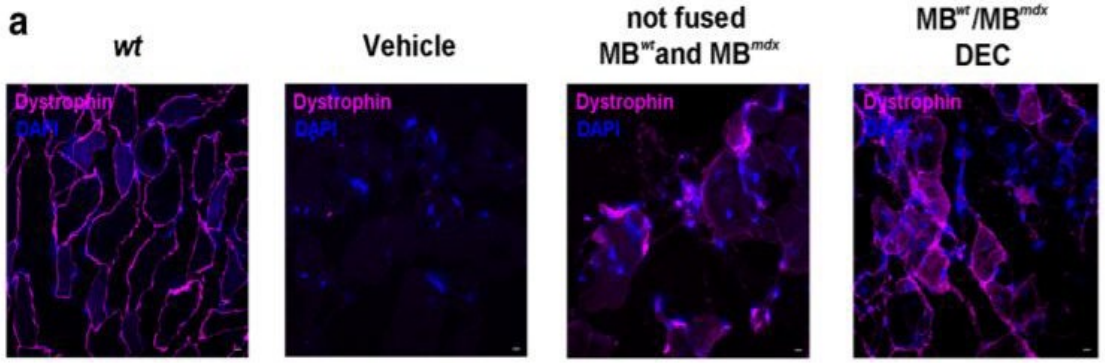
# Supportive Pre-clinical Data

# Rationale for Chimeric Cell Therapy

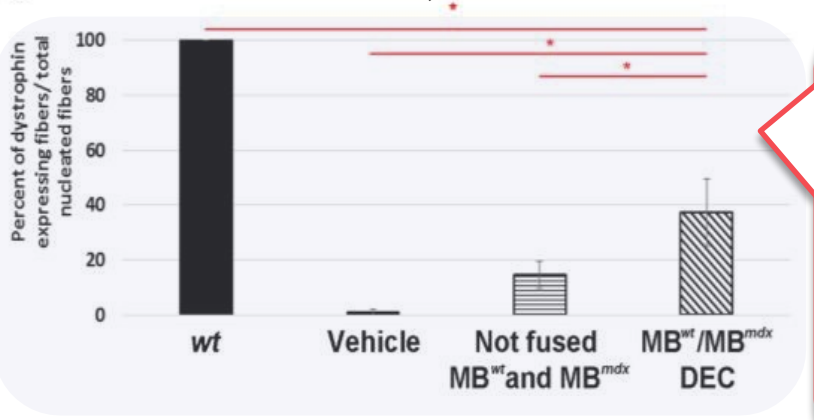
The rationale for our therapy platform is based on the principle of chimerism, a well-accepted approach for tolerance induction and prevention of rejection, which is well established in transplantation literature. Based on our 20 years of experience with the application of chimerism-based strategies for tolerance induction in bone marrow and vascularized composite allograft transplantation we have addressed the limitations of current cell therapies, by introducing chimeric cells as a novel therapeutic approach for DMD. Hence, we applied the concept of ex-vivo cell fusion technology to create human chimeric cell lines of myoblast origin as a novel, healthy myoblast delivery platform, presenting the allogeneic donor's cells as the patients' own cells in order to minimize the immune response and eliminate the need for immunosuppression. The proof-of-concept of chimeric cell therapy efficacy in DMD was demonstrated in preclinical studies in both mdx and mdx/scid mouse models of DMD, where tolerogenic and immunomodulatory properties of chimeric cell therapy and efficacy of chimeric cell engraftment were confirmed by histology and immunohistochemistry as well as significant increase of dystrophin expression correlating with long-term (180 days) improvement of function in the most DMD affected target organs - cardiac, respiratory and skeletal muscles. Based on these encouraging preclinical outcomes, we have further applied the chimeric cell concept to clinical applications as a unique platform of chimerism delivery and demonstrated excellent long-term safety and tolerability profile as well as consistent functional outcome improvements in 6 patients with DMD

# Demonstrated Dystrophin Expression While Reducing Fibrosis

Significant increase of Dystrophin expression at 30 days after DEC transplant to the gastrocnemius muscle (GM) of mdx mice




Immunofluorescence images of dystrophin expression in GM of: wild type (wt) mice (left, positive control); Dystrophin-deficient mdx mice injected with vehicle; not fused MBwt with MBmdx; MBwt/MBmdx DEC

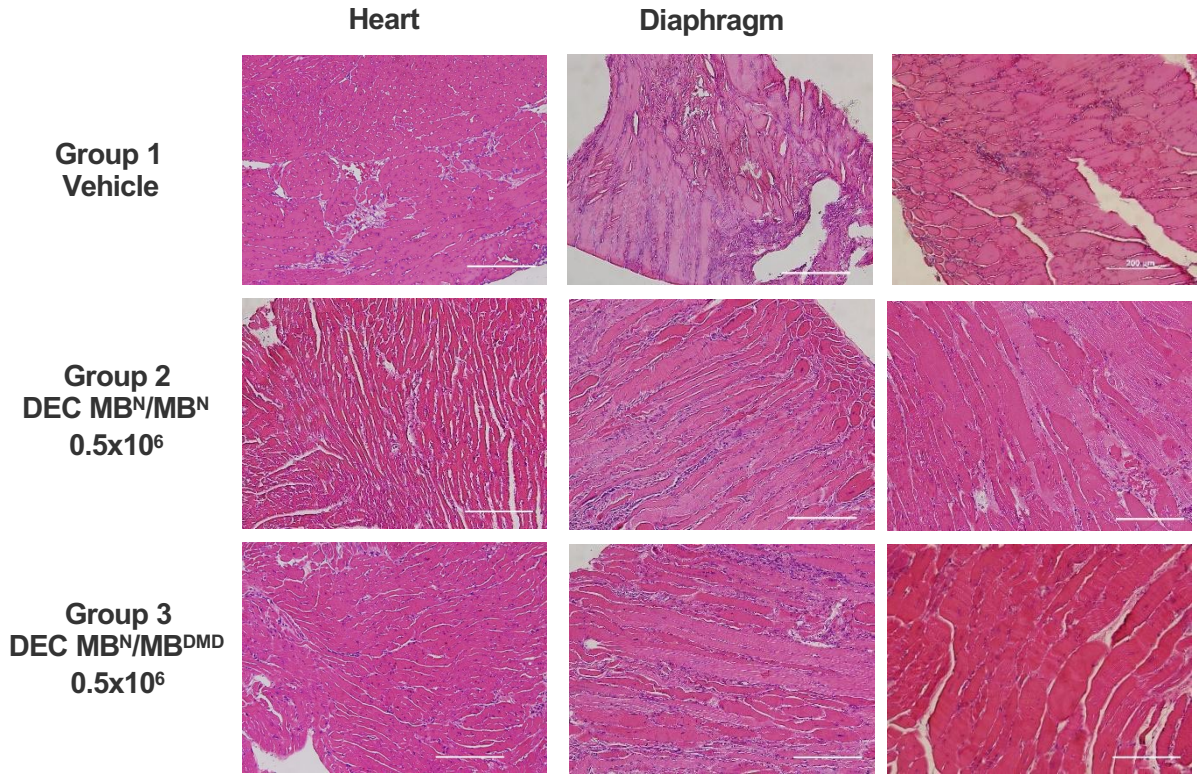



*Siemionow et al., Stem Cell Rev and Rep (2018)*

**Biological marker: 37.3% increase in Dystrophin level at 30 days**

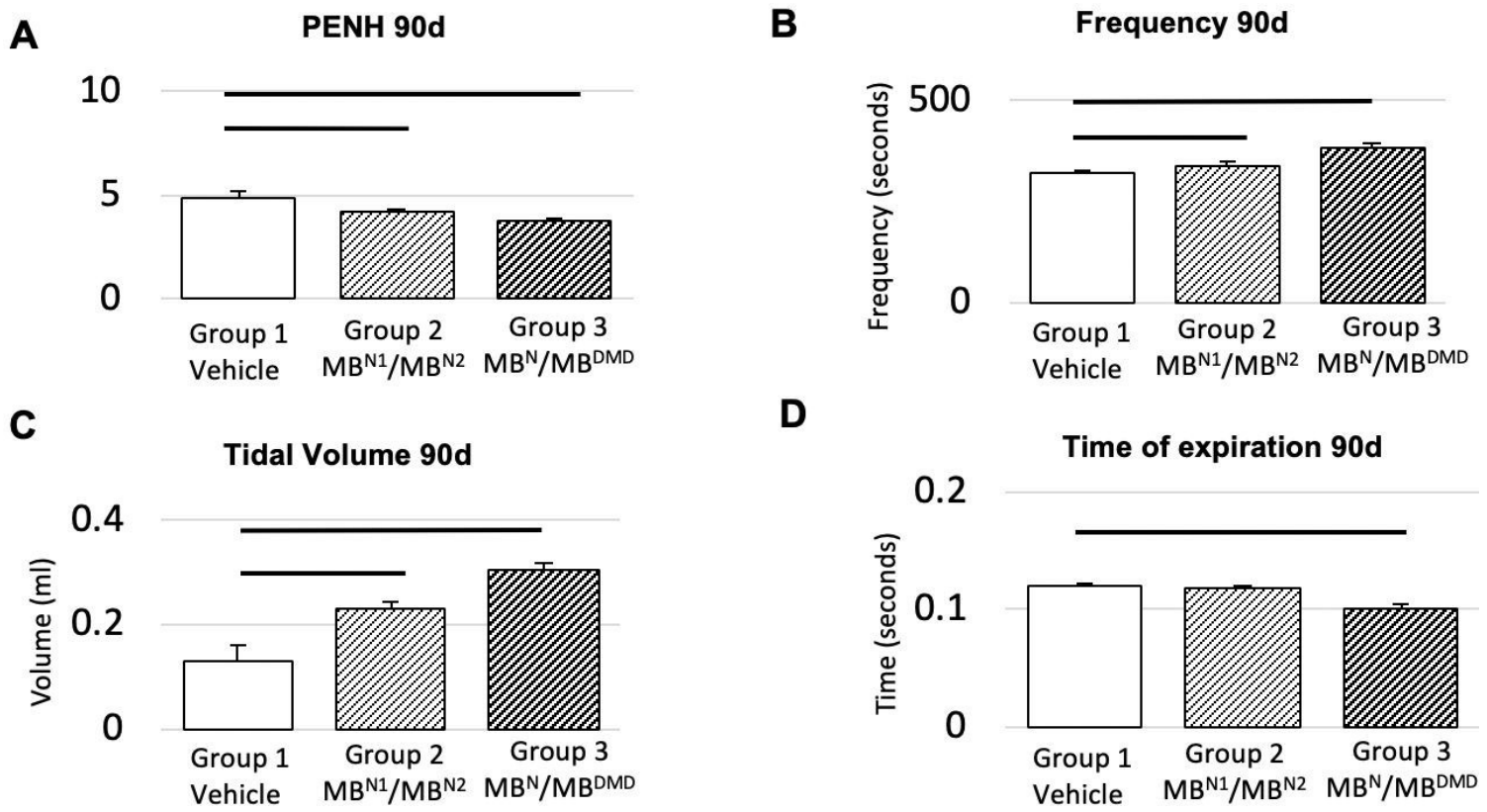
After administration to gastrocnemius muscle in DEC injected mdx host compared to vehicle and not-fused MBwt and MBmdx controls; (n=6, mean±SD, 5 ROI/3 sections/6 animal/group, (One-way ANOVA)

Tangible Improvements on a Cellular Level At 90 Days




Hematoxylin-eosin (H&E) stained cross-sections of heart, diaphragm and gastrocnemius muscle of *mdx/scid* mice **confirming reduced fibrosis** at 90 days after DEC transplant when compared to vehicle control
Magnification 10X, scale bar 200µm (Olympus, Japan)

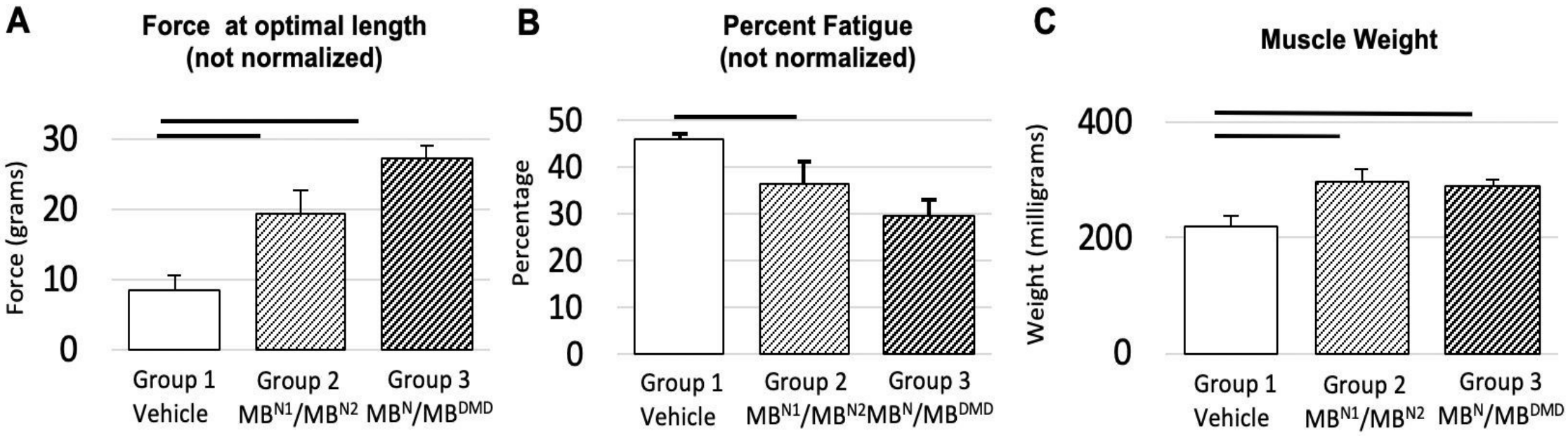
# Improved Respiratory Function after Systemic DEC Therapy in mdx/scid mice on Plethysmography




*American Society for Reconstructive Microsurgery, Ft. Lauderdale, January 10-14, 2020*

**Improved respiratory function after systemic-intraosseous transplant of DEC cells confirmed by whole-body plethysmography;**
**One-Way Anova with post-hoc Tukey's test, $p<0.05$**

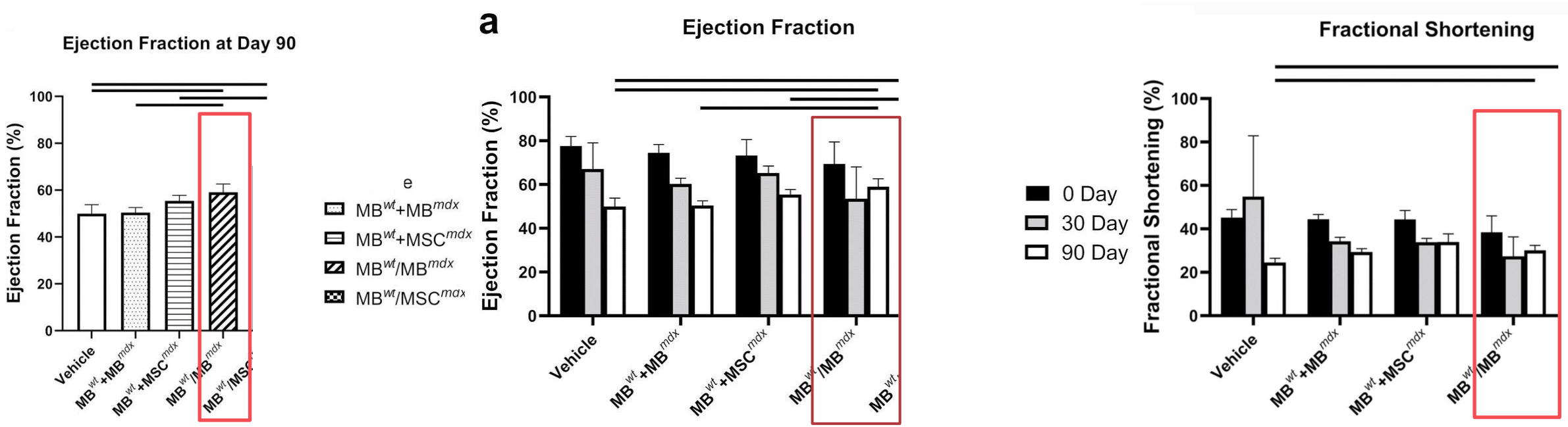
# Improved Muscle Force Function after Systemic DEC Therapy in mdx/scid mice




Reduced skeletal muscle disease after systemic-intraosseous transplant of DEC cells confirmed by ex vivo Aurora Muscle Force test;
One-Way Anova with post-hoc Tukey's test, $p<0.05$

# Improved Cardiac Outcomes After Systemic DEC Therapy in mdx/scid mice

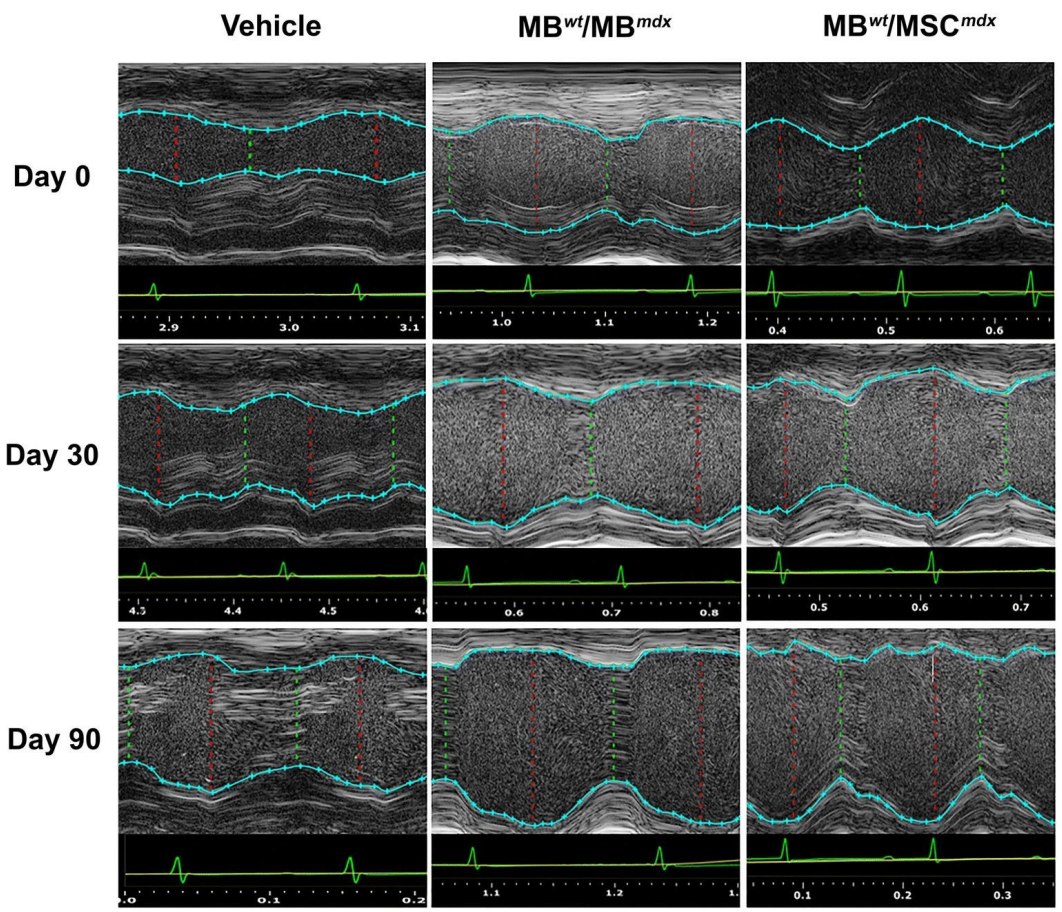
***Systemic DEC** administration demonstrated statistically **significant increase in cardiac ejection fraction at day 90 as confirmed by echocardiography***

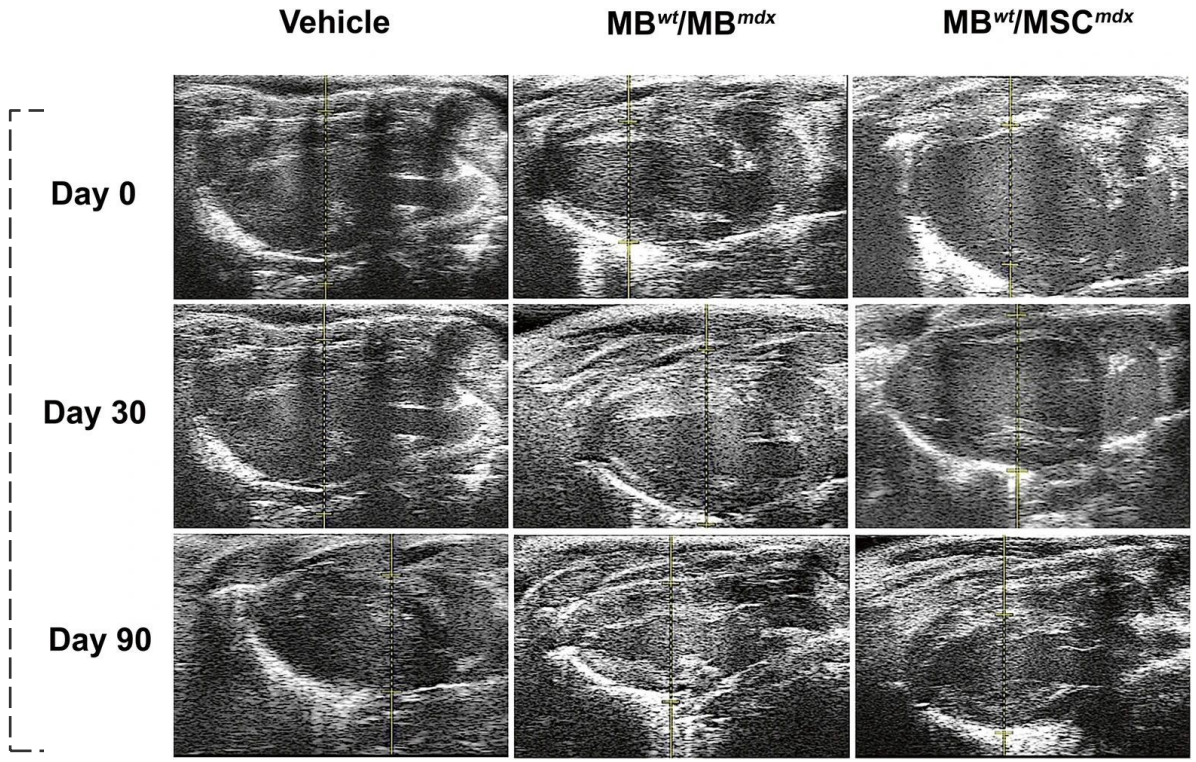



Stem Cell Reviews and Reports **volume 15**, pages 827–841 (2019)

**With a p-value of 0.031, EF was statistically significantly higher at day 90 for the mice injected with snjMB/mdxMB fused cells at an 0.05 compared to EF at day 0.**

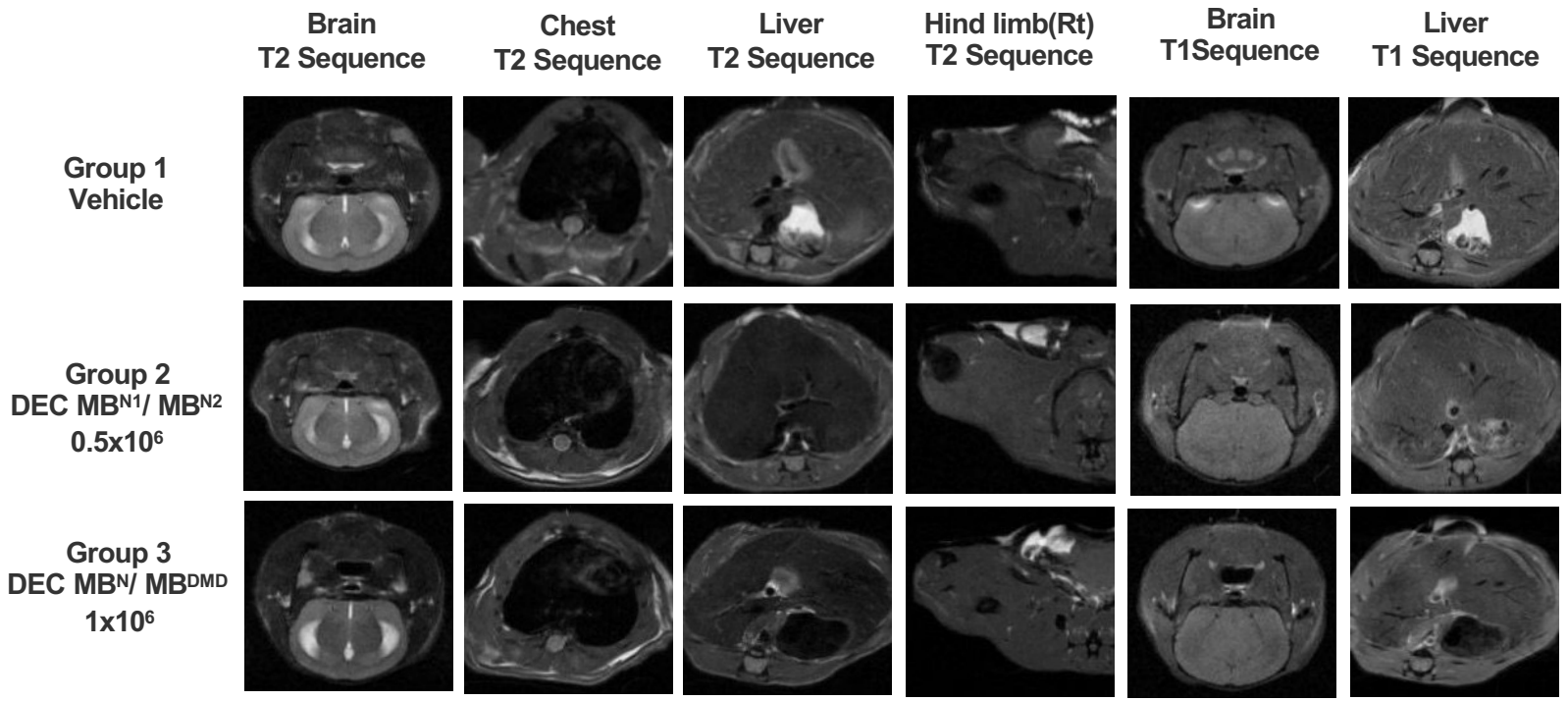
# Reduced Cardiac Disease at 90 Days after Systemic DEC Administration to the mdx mice




Confirmed by M and B-mode echocardiography

# Confirmation of DEC Safety at 90 days after Systemic Administration to the mdx/scid mice




Assessment of organs of the highest tumor risk rate and the right hind limb revealed lack of tumor or tumor-like structure formation confirming safety of DEC cell lines after transplant.
MRI evaluation was made using a standard T2 – weighted protocol and T1 – weighted scanning for brain and liver 31cm bore size 9.4 Tesla Agilent MRI System

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