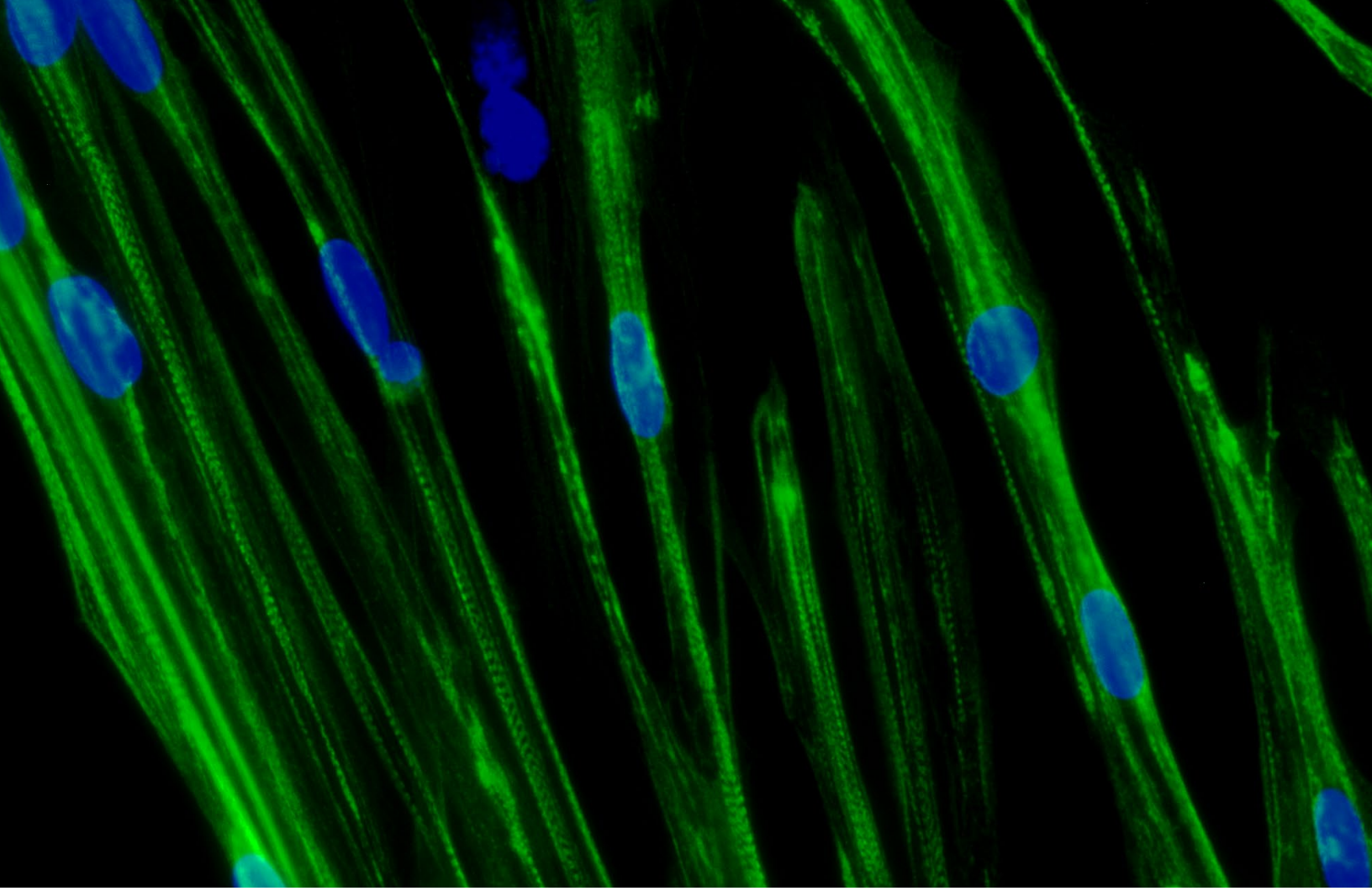


ioSkeletal Myocytes™

Human iPSC-derived skeletal myocytes

Learn more about ioSkeletal Myocytes

ioCells™

About the cells

ioSkeletal Myocytes, are human iPSC-derived skeletal myocytes precision reprogrammed using opti-ox™ technology. Cells are delivered cryopreserved and upon revival, mature rapidly to form elongated, striated, multinucleated muscle cells that contract within 10 days. The cells are easy to culture, consistently exhibit high population purity and express key myofilament proteins.

ioSkeletal Myocytes are a reliable source of highly-defined and consistent human muscle cells that provide a valuable tool for various areas of research, including mechanistic and functional studies, disease modelling and drug development.

Benchtop benefits

CONSISTENT

Lot-to-lot reproducibility and homogeneity results in a stable human model for the study of muscle and neuromuscular disorders.

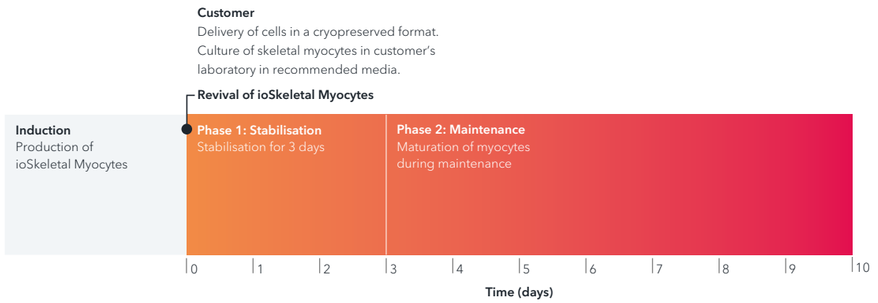
DEFINED

Striated, multinucleated skeletal myocytes form within 10 days post-revival and are characterised by ICC and gene expression.

FUNCTIONAL

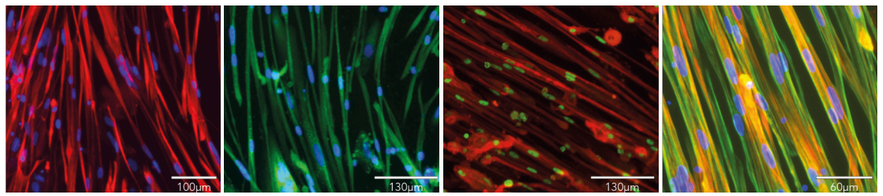
Cells contract in response to chemical and electrical stimulation and react to pharmacological inhibitors and activators.

ioSkeletal Myocytes are delivered cryopreserved and ready to plate

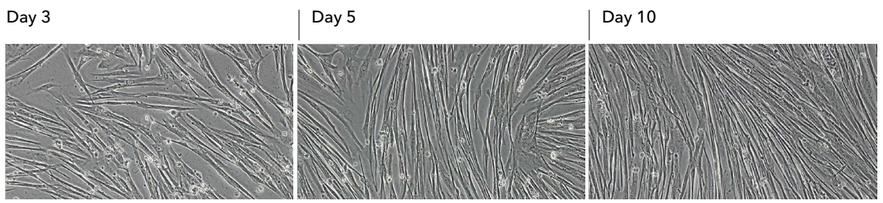


ioSkeletal Myocytes are highly characterised and defined, so you know exactly what is in every vial

ioSkeletal Myocytes show robust protein expression of components of the contractile apparatus including desmin, dystrophin, and myosin heavy chain (MHC), as well as myogenin and troponin, with visible striated fibres, and multinucleation. Cells show classical myocyte morphology and form elongated, multinucleated myocytes over 10 days post revival.



A. Desmin/DAPI B. Dystrophin/DAPI C. Myogenin/MHC D. Troponin/Phalloidin/DAPI



Robust human skeletal muscle cell model suitable for functional studies

Cells contract in 2D culture in response to increased extracellular potassium levels and electrical stimulation

A. Immunofluorescence staining of ioSkeletal Myocytes revealing robust expression of sarcomere structures.

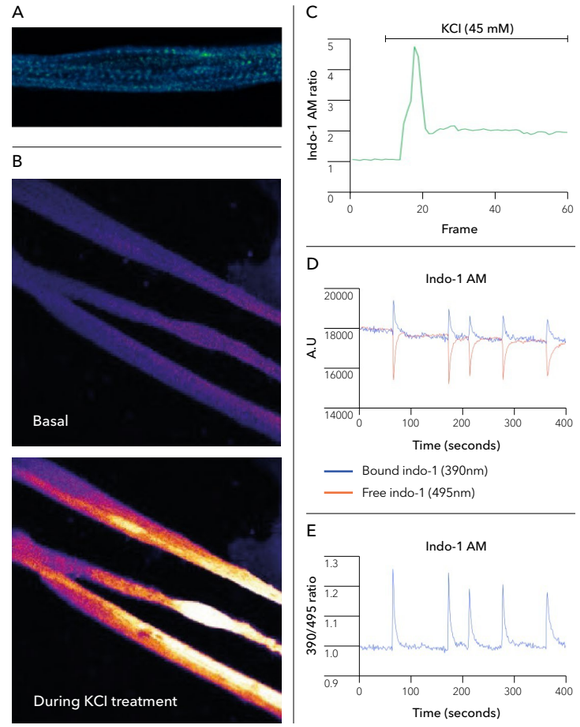
B. Contraction is stimulated by depolarisation of the cells using potassium chloride (KCl), and the consequent increase in intracellular calcium is detected using calcium binding indicator dye Indo-1 AM. ioSkeletal Myocytes incubated with Indo-1 AM (5 µM) and 0.02% Pluronic F127; cells were excited at UV spectra (355 nm).

C. Changes in Indo-1 AM ratio shows calcium influx induced by 45 mM KCl.

D. Contraction is induced by electrical stimulation and the cells release and sequester calcium repeatedly, demonstrating they can withstand repeated electrical stimuli whilst maintaining their ability to regulate intracellular calcium signalling.

E. The data in D is shown as a ratio of bound to free Indo-1; electrical stimulation, 2 Hz, 6 v, 2 ms.

Airyscan Z-series stacks, data courtesy of Gabriel E. Valdebenito and Michael R. Duchen, 2021. UCL, UK



Functional 3D muscle microtissues respond to electrical and pharmacological stimuli

ioSkeletal Myocytes form functional 3D skeletal muscle microtissues that respond to electrical and pharmacological stimuli

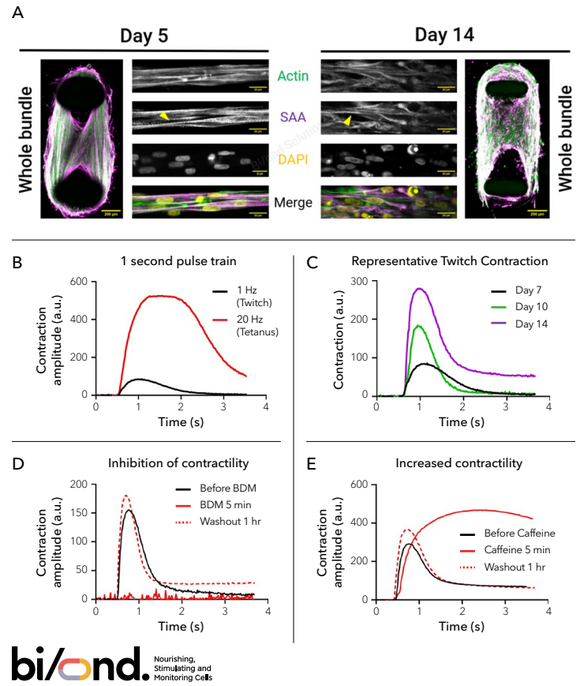
A. ioSkeletal Myocytes were successfully cultured in 3D on Bi/ond's MUSbit™ microchip over 14 days. Cells express muscle cell markers and show cross-striation of sarcomeric alpha actinin (SAA) (yellow arrows).

B-C. Muscle bundles show twitch and tetanic forces at day 7 and become stronger over time.

D. Contraction is inhibited when the bundle is electrically stimulated following treatment with BDM, a non-selective skeletal muscle myosin-II ATPase inhibitor.

E. Contractility is increased when the bundle is electrically stimulated following addition of caffeine, which stimulates calcium release from the sarcoplasmic reticulum.

Images and data courtesy of Bi/ond Solutions B.V., Delft, The Netherlands.



bi/ond. Nourishing, Stimulating and Monitoring Cells

ioSkeletal Myocytes form the isogenic control for DMD exon 44 and exon 52 deletion disease models, enabling investigation of disease-related phenotypes

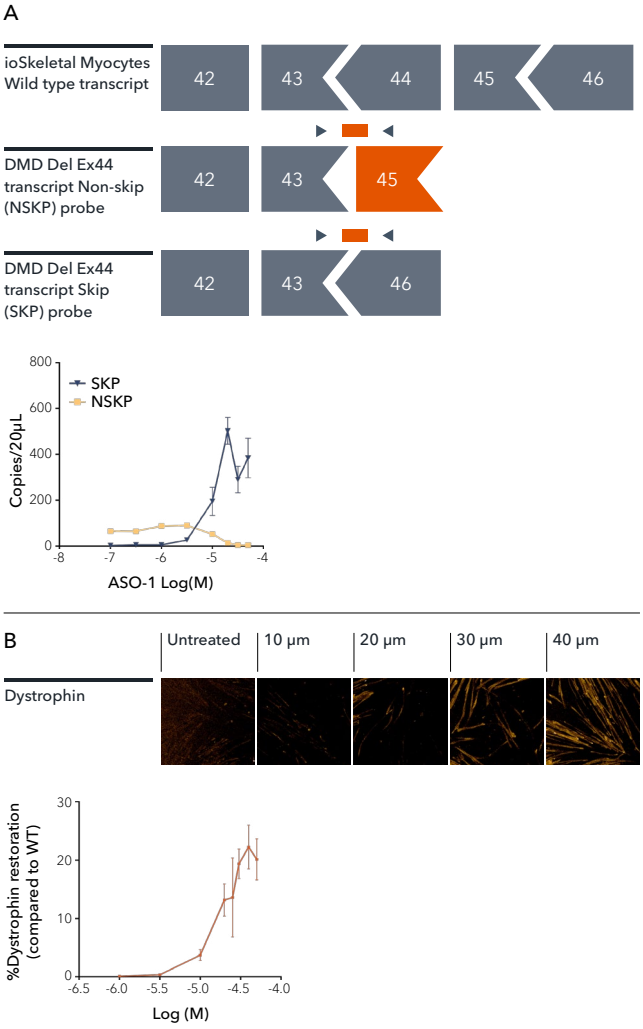
Restoration of dystrophin by ASO-mediated exon skipping in DMD Exon 44 Deletion disease model cells

A. Dystrophin mRNA restoration. PCR primers and fluorescent labelled probes were designed to amplify the region coding exons 43-45 (NSKP) or exons 43-46 (SKP). The graph shows a concentration-dependent increase in the SKP transcript (blue) and a decrease in the NSKP transcript (yellow), indicating that ASO treatment successfully created an in-frame mRNA transcript for dystrophin.

B. Dystrophin protein restoration. High content image analysis demonstrated that ASO treatment restored dystrophin protein expression in a concentration-dependent manner.

Data courtesy of Charles River Laboratories.

charles river



Who we are

bit.bio combines the concepts of cell programming and biology to provide human cells for research, drug discovery and cell therapy, enabling a new generation of medicines.

This is possible with our precision human cellular reprogramming technology opti-ox™ – a gene engineering approach that enables unlimited batches of any human cell to be manufactured consistently at scale

For general information, email info@bit.bio

To learn more, visit www.bit.bio

Product information

Cat code
io1002

Starting material
Human iPSC line

Karotype
Normal (46, XY)

Seeding compatibility
6, 12, 24, 48, 96 and 384 well plates

Shipping info
Dry ice

Donor
Caucasian adult male (skin fibroblast)

Vial size
Small: >2.5 x 10^6 viable cells
Large: >5 x 10^6 viable cells

Quality control
Sterility, protein expression (ICC) and gene expression (RT-qPCR)

Differentiation method
opti-ox™ cell reprogramming

Recommended seeding density
100,000 cells/cm^2

User storage
LN2 or -150°C

Format
Cryopreserved cells

Product use
ioCells™ are for research use only

Applications
Functional and mechanistic studies in 2D and 3D cultures
Disease modelling for neuromuscular disorders and muscular dystrophies
Drug discovery and development

bit.bio
THE CELL CODING COMPANY