Testimony of Daniel Paul Perez, Co-Founder & Director Emeritus FSHD Society before
U.S. House Appropriations Subcommittee on Labor, HHS, Education and Related Agencies
May 26, 2022

Honorable Chairwoman DeLauro, Ranking Member Cole, and distinguished members of the Subcommittee, thank you. We are requesting the FY2023 appropriation of an *amount* of **$30 million** for the *agency* U.S. DHHS National Institutes of Health (**NIH**) to sustain and increase its 'dedicated' research *program* on facioscapulohumeral muscular dystrophy (*hereafter called* **FSHD**).

Madam Chairman, this is my sixty-fourth (64th) testimony before the U.S. Congress' Appropriations Subcommittee on this matter. I have been professionally engaged in FSHD research since 1987, with a focus on funding research on the fundamental pathophysiology, molecular biology and genetics of the disease. I am co-founder, Director emeritus, past -Chairman, -President & CEO, and -CSO of FSHD Society, and have been involved in the evolution and design of FSHD research, gene mapping and genetics more or less from its inception to the present. My work and the FSHD Society's funding spans nearly every research lab working on FSHD, the tactical and strategic planning that have led to understanding how FSHD1[1] and FSHD2[2,3] work, the advancement of the Muscular Dystrophy Community Assistance, Research and Education Amendments of 2001 (MD-CARE Act, Public Law 107-84), advocacy and policy, and the relationship of the scientific community to the societal context in which FSHD is embedded.

I am approaching near thirty (30) years of testifying as a patient with FSHD for Appropriation of funding for FSHD. I have had this disease for twice that time. That is a long time to live with a disease of this burden. I have now seen, experienced the entire effects, and borne almost the full brunt of what FSHD can do to you. FSHD is a heritable disease and one of the most common neuromuscular disorders with a prevalence of 1:8,000.[4] It affects 934,000 children and adults of both sexes worldwide. FSHD is characterized by progressive loss of skeletal muscle strength that is asymmetric in pattern and widely variable. Muscle weakness typically starts at the face, shoulder

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girdle and upper arms, often progressing to the legs, torso and other muscles. In addition to affecting any skeletal muscle, it can bring with it respiratory failure and breathing issues[5-7], mild-profound hearing loss[8], eye problems and cardiac bundle blockage and arrhythmias[9,10]. FSHD causes significant disability and death according the U.S. Centers for Disease Control and Prevention (CDC), National Center on Birth Defects and Developmental Disabilities, Atlanta, and others.[11,12]

The NIH is currently the principal worldwide source of funding of biomedical research on FSHD. Currently annual funding dedicated for FSHD listed in NIH RCDC is $10 million. Given the remarkable advances and momentum in FSHD research in the past eight years; it is appalling that FSHD funding has not grown according to NIH RCDC. This indicates a mismatch between NIH funding mechanisms and the external community working on FSHD.

Figure 1. **FSHD Research Dollars (millions) & FSHD Percentage of Total NIH Muscular Dystrophy Funding**

Sources: NIH/OD Budget Office & NIH OCPL & NIH RePORT / RCDC (e=estimate, a=actual)

Fiscal Year	**2010**	**2011**	**2012**	**2013**	**2014**	**2015**	**2016**	**2017**	**2018**	**2019**	**2020**	**2021e**	**2022e**
All MD ($ millions)	$86	$75	$75	$76	$78	$77	$79	$81	$81	$83	$95	$97	$102
FSHD ($ millions)	$6	$6	$5	$5	$7	$8	$9	$11	$11	$10	$9	$9	$10
FSHD (% total MD)	7%	8%	7%	7%	9%	10%	11%	14%	14%	12%	9%	9%	10%

Currently active projects listed in NIH RePORT as being applicable to FSHD are $17.507 million FY2022 (04May) (source: NIH Research Portfolio Online Reporting Tools (RePORT) 'FSHD or facioscapulohumeral or landouzy-dejerine'). This includes dedicated funds in NIH RCDC.

In my role, I provide initiator and seed funding to bring new ideas and researchers online and am asked to evaluate and compare the various research projects we have funded and understand their commonalities and differences. I have, if I had to estimate, made a fairly extensive study of six hundred FSHD projects and proposals. My effort to solve FSHD has been persistent in yielding refined advances, it is novel and original, and is clearly driven by an overall sense of goals and concepts that are meaningful to patients and their families. The research we've started is forward-looking, as illustrated by the number of industry and philanthropic partners/researchers that have picked up on this seminal work and contributed to it.

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FSHD is associated with epigenetic changes on the tip of human chromosome 4q35 in the D4Z4 DNA macrosatellite repeat array region leading to an inappropriate gain of expression (function) of the D4Z4-embedded double homeobox 4 (DUX4) gene[13-16]. DUX4 is a transcription factor that kick starts the embryonic genome during the 2- to 8-cell stage of development[17-19]. Ectopic expression of DUX4 in skeletal muscle is associated with the disease and the disease's pathophysiology that leads to muscle death. DUX4 is never expressed in 'healthy' muscle. FSHD has had few clinical trials[20-26,27,28], and currently there is no cure or therapeutic option available to patients. DUX4 requires and needs to activate its direct transcriptional targets for DUX4-induced gene aberration and muscle toxicity[29-41]. The genetics of FSHD are so remarkable, that NIH Director Dr. Francis Collins said on the front page of the New York Times, "If we were thinking of a collection of the genome's greatest hits, this [FSHD] would go on the list."[42] Add to this that FSHD was the sole disease mentioned in the recent tour de force Science publication 'The complete sequence of a human genome.' FSHD was highlighted as having a bunch of newly assembled paralogs in the assembly; some of which showed evidence of being transcriptionally active. Great exposure for FSHD![43]

Blocking DUX4's DNA, DUX4's RNA or DUX4's protein ability to activate its targets has profound therapeutic relevance[44-47]. The scientific community has in recent years pioneered inroads to treating FSHD using the enormous potential of genomic sequencing, genomic medicine, gene editing and next generation diagnostics. All with the central paradigm of the reduction of: DUX4, DUX4 expression, DUX4 protein activity, or the effects of DUX4-mediated toxicity. Strategies include modulating DUX4 repressive pathways, targeting DUX4 mRNA, DUX4 protein, or cellular downstream effects of DUX4 expression. Our FY2022 testimony on FSHD was very comprehensive in scope with a call to action in research areas from bench-side to clinic. Rather than restating – we ask the Committee to urge NIH to move forward on the many priorities listed.

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In FY2023, we/I additionally request NIH to make an immediate and targeted push to answer the following three questions. Answers to these questions will help remove the obstacles to measuring disease progression, help measure if novel therapeutics are making a difference in stopping the disease and elucidate if muscle can grow again and be restored.

Figure 2. **Three Key Research Questions**

How does DUX4 expression lead to pathophysiology? We know a lot of what can happen when DUX4 is expressed in a cell (mostly forced experimental expression), but not a lot about what happens when an FSHD muscle cell expresses DUX4 that leads to pathology.

Can FSHD muscle pathology be reversed once DUX4 expression starts the pathogenic cascade in a particular muscle? This is a key question when looking to improve outcomes with either muscle building or DUX4 halting therapies.

Is there a systemic effect of local DUX4 expression that leads to amplification of muscle decline, either immune or some extracellular signaling? Answering this question will help delineate where along the travels of DUX4 from its birth and death in muscle we can intercept and control the disease process.

New data/information generated on the basic mechanism of DUX4 and how it causes muscle disease has the potential to focus the design of future clinical trials on muscles and measurements that will increase the rigor of the design and decrease the number of individuals necessary for initial tests of drug activity. It is absolutely necessary to increase our resolution, clarity and understanding of what DUX4 is and what it does to muscle in FSHD. The gains in this area will effectively unpin or untether FSHD from the difficulty category of "slowly progressing neuromuscular diseases remaining recalcitrant" to timely ascertainment that a clinical intervention can work.

We request for FY2023, increasing NIH FSHD research funding/appropriation of the standard portfolio to $25 million. The growth has been slow, continuous and prone to year-to-year up/down fluctuations according to NIH. Additionally, we request a one-time boost of $5 million to solicit applications to answer the three questions of key import. FSHD needs an infusion of NIH grants both submitted and funded. NIH needs to add exploratory / developmental research grants (parent R21) and research project grants (parent R01). This is NIH's wheelhouse and forte without a doubt.

Madam Chairman, thank you for this opportunity to update you on FSHD with this testimony.

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REFERENCES

1. Gould T, Jones TI, Jones PL. Precise Epigenetic Analysis Using Targeted Bisulfite Genomic Sequencing Distinguishes FSHD1, FSHD2, and Healthy Subjects. Diagnostics (Basel). 2021 Aug 13;11(8):1469. doi: 10.3390/diagnostics11081469. PMID: 34441403; PMCID: PMC8393475. (2021).
2. Goossens R, Tihaya MS, van den Heuvel A, Tabot-Ndip K, Willemsen IM, Tapscott SJ, González-Prieto R, Chang JG, Vertegaal ACO, Balog J, van der Maarel SM. A proteomics study identifying interactors of the FSHD2 gene product SMCHD1 reveals RUVBL1-dependent DUX4 repression. Sci Rep. 2021 Dec 8;11(1):23642. doi: 10.1038/s41598-021-03030-3. PMID: 34880314; PMCID: PMC8654949. (2021).
3. Wang, L. H. & Tawil, R. Facioscapulohumeral Dystrophy. Curr. Neurol. Neurosci. Rep. 16, 66 (2016).
4. Deenen, J. C. W. et al. Population-based incidence and prevalence of facioscapulohumeral dystrophy. Neurology 83, 1056–9 (2014).
5. Lu-Nguyen N, Malerba A, Antoni Pineda M, Dickson G, Popplewell L. Improving Molecular and Histopathology in Diaphragm Muscle of the Double Transgenic ACTA1-MCM/FLExDUX4 Mouse Model of FSHD with Systemic Antisense Therapy. Hum Gene Ther. 2022 Apr 28. doi: 5.1089/hum.2021.251. Epub ahead of print. PMID: 35078334. (2022).
6. Teeselink S, Vincenten SCC, Voermans NC, Groothuis JT, Doorduin J, Wijkstra PJ, Horlings CGC, van Engelen BGM, Mul K. Long-term follow-up of respiratory function in facioscapulohumeral muscular dystrophy. J Neurol. 2022 Feb 11:1–8. doi: 10.1007/s00415-022-10990-7. Epub ahead of print. PMID: 35147730; PMCID: PMC8831680. (2022).
7. Henke C, Spiesshoefer J, Kabitz HJ, Herkenrath S, Randerath W, Brix T, Görlich D, Young P, Boentert M. Respiratory muscle weakness in facioscapulohumeral muscular dystrophy. Muscle Nerve. 2019 Dec;60(6):679-686. doi: 10.1002/mus.26717. Epub 2019 Oct 23. PMID: 31566774. (2019).
8. Fuccillo E, Frezza E, Massa R, Di Girolamo S. Response To Letter To The Editor "Auditory Dysfunction In Facioscapulohumeral Muscular Dystrophy Type 1: Beyond The Inner Ear Involvement" By Gheller et al. Otol Neurotol. 2022 Mar 1;43(3):e392-e393. doi: 10.1097/MAO.0000000000003423. PMID: 34772889. (2022).
9. van Dijk GP, van der Kooi E, Behin A, Smeets J, Timmermans J, van der Maarel S, Padberg G, Voermans N, van Engelen B. High prevalence of incomplete right bundle branch block in facioscapulohumeral muscular dystrophy without cardiac symptoms. Funct Neurol. 2014 Jul-Sep;29(3):159-65. PMID: 25473735; PMCID: PMC4264782. (2014).
10. Ducharme-Smith A, Nicolau S, Chahal CAA, Ducharme-Smith K, Rehman S, Jaliparthy K, Khan N, Scott CG, St Louis EK, Liewluck T, Somers VK, Lin G, Brady PA, Milone M. Cardiac Involvement in Facioscapulohumeral Muscular Dystrophy (FSHD). Front Neurol. 2021 May 24;12:668180. doi: 10.3389/fneur.2021.668180. (2021).
11. Blokhuis AM, Deenen JCW, Voermans NC, van Engelen BGM, Kievit W, Groothuis JT. The socioeconomic burden of facioscapulohumeral muscular dystrophy. J Neurol. 2021 May 27. doi: 10.1007/s00415-021-10591-w. (2021).
12. Wallace B, Smith KT, Thomas S, Conway KM, Westfield C, Andrews JG, Weinert RO, Do TQN, Street N; Muscular Dystrophy Surveillance, Tracking, and Research Network (MD STARnet). Characterization of individuals with selected muscular dystrophies from the expanded pilot of the Muscular Dystrophy Surveillance, Tracking and Research Network (MD STARnet) in the United States. Birth Defects Res. 2021 Apr 15;113(7):560-569. doi: 10.1002/bdr2.1764. (2020).
13. Mocciaro E, Runfola V, Ghezzi P, Pannese M, Gabellini D. DUX4 Role in Normal Physiology and in FSHD Muscular Dystrophy. Cells. 2021 Nov 26;10(12):3322. doi: 10.3390/cells10123322. PMID: 34943834; PMCID: PMC8699294. (2021).
14. Jagannathan S. The evolution of DUX4 gene regulation and its implication for facioscapulohumeral muscular dystrophy. Biochim Biophys Acta Mol Basis Dis. 2022 May 1;1868(5):166367. doi: 10.1016/j.bbadis.2022.166367. Epub 2022 Feb 11. PMID: 35158020. (2022).
15. Vuoristo S, Bhagat S, Hydén-Granskog C, Yoshihara M, Gawriyski L, Jouhilahti EM, Ranga V, Tamirat M, Huhtala M, Kirjanov I, Nykänen S, Krjutškov K, Damdimopoulos A, Weltner J, Hashimoto K, Recher G, Ezer S, Paluoja P, Palovita P, Takegami Y, Kanemaru A, Lundin K, Airenne TT, Otonkoski T, Tapanainen JS, Kawaji H, Murakawa Y, Bürglin TR, Varjosalo M, Johnson MS, Tuuri T, Katayama S, Kere J. DUX4 is a multifunctional factor priming human embryonic genome activation. iScience. 2022 Mar 22;25(4):104137. doi: 10.1016/j.isci.2022.104137. PMID: 35402882; PMCID: PMC8990217.
16. Wong CJ, Whiddon JL, Langford AT, Belleville AE, Tapscott SJ. Canine DUXC: Implications for DUX4 retrotransposition and preclinical models of FSHD. Hum Mol Genet. 2021 Dec 9:ddab352. doi: 10.1093/hmg/ddab352. Epub ahead of print. PMID: 34888646.
17. Hendrickson, P. G. et al. Conserved roles of mouse DUX and human DUX4 in activating cleavage-stage genes and MERVL/HERVL retrotrans. Nat. Genet. 49, 925–934 (2017).
18. Whiddon, J. L., Langford, A. T., Wong, C.-J., Zhong, J. W. & Tapscott, S. J. Conservation and innovation in the DUX4-family gene network. Nat. Genet. 49, 935–940 (2017).
19. De Iaco, A. et al. DUX-family transcription factors regulate zygotic genome activation in placental mammals. Nat. Genet. 49, 941–945 (2017).
20. Tawil, R. et al. A pilot trial of prednisone in facioscapulohumeral muscular dystrophy. FSHDY Group. Neurology 48, 46–9 (1997).
21. Passerieux, E. et al. Effects of vitamin C, vitamin E, zinc gluconate, and selenomethionine supplementation on muscle function and oxidative stress biomarkers in patients with facioscapulohumeral dystrophy: a double-blind randomized controlled clinical trial. Free Radic. Biol. Med. 81, 158–69 (2015).
22. Kissel, J. T. et al. Randomized, double-blind, placebo-controlled trial of albuterol in facioscapulohumeral dystrophy. Neurology 57, 1434–40 (2001).
23. Elsheikh, B. H. et al. Pilot trial of diltiazem in facioscapulohumeral muscular dystrophy. Neurology 68, 1428–9 (2007).
24. Wagner, K. R. et al. A phase I/IItrial of MYO-029 in adult subjects with muscular dystrophy. Ann. Neurol. 63, 561–71 (2008).
25. Statland JM, Campbell C, Desai U, Karam C, Díaz-Manera J, Guptill JT, Korngut L, Genge A, Tawil RN, Elman L, Joyce NC, Wagner KR, Manousakis G, Amato AA, Butterfield RJ, Shieh PB, Wicklund M, Gamez J, Bodkin C, Pestronk A, Johnson NE, Mathews KD, Miller B, Leneus A, Fowler M, van de Rijn M, Attie KM. Randomized phase 2 study of ACE-083, a muscle-promoting agent, in facioscapulohumeral muscular dystrophy. Muscle Nerve. 2022 Apr 15. doi: 10.1002/mus.27558. Epub ahead of print. PMID: 35428982. (2022).
26. Mellion ML, Ronco L, Berends CL, Pagan L, Brooks S, van Esdonk MJ, van Brummelen EMJ, Odueyungbo A, Thompson LA, Hage M, Badrising UA, Raines S, Tracewell WG, van Engelen B, Cadavid D, Groeneveld GJ. Phase 1 clinical trial of losmapimod in facioscapulohumeral dystrophy: Safety, tolerability, pharmacokinetics, and target engagement. Br J Clin Pharmacol. 2021 Dec;87(12):4658-4669. doi: 10.1111/bcp.14884. Epub 2021 May 14. PMID: 33931884. (2021).
27. Ghasemi M, Emerson CP Jr, Hayward LJ. Outcome Measures in Facioscapulohumeral Muscular Dystrophy Trials. Cells. 2022 Feb 16;11(4):687.. PMID: 35203336; PMCID: PMC8870318. (2022).
28. Gros M, Nunes AM, Daoudlarian D, Pini J, Martinuzzi E, Barbosa S, Ramirez M, Puma A, Villa L, Cavalli M, Grecu N, Garcia J, Siciliano G, Solé G, Juntas-Morales R, Jones PL, Jones T, Glaichenhaus N, Sacconi S. Identification of Serum Interleukin 6 Levels as a Disease Severity Biomarker in Facioscapulohumeral Muscular Dystrophy. J Neuromuscul Dis. 2022;9(1):83-93.. PMID: 34459413; PMCID: PMC8842759. (2022).
29. Rickard, A. M., Petek, L. M. & Miller, D. G. Endogenous DUX4 expression in FSHD myotubes is sufficient to cause cell death and disrupts RNA splicing and cell migration pathways. Hum. Mol. Genet. 24, 5901–14 (2015).
30. Sandri, M. et al. Caspase 3 expression correlates with skeletal muscle apoptosis in Duchenne and facioscapulohumeral muscular dystrophy. A potential target for pharmacological treatment? J. Neuropathol. Exp. Neurol. 60, 302–12 (2001).
31. Block, G. J. et al. Wnt/β-catenin signaling suppresses DUX4 expression and prevents apoptosis of FSHD muscle cells. Hum. Mol. Genet. 22, 4661–72 (2013).
32. Statland, J. M. et al. Immunohistochemical Characterization of Facioscapulohumeral Muscular Dystrophy Muscle Biopsies. J. Neuromuscul. Dis. 2, 291–299 (2015).
33. Kowaljow, V. et al. The DUX4 gene at the FSHD1A locus encodes a pro-apoptotic protein. Neuromuscul. Disord. 17, 611–23 (2007).
34. Bosnakovski, D. et al. An isogenetic myoblast expression screen identifies DUX4-mediated FSHD-associated molecular pathologies. EMBO J. 27, 2766–79 (2008).
35. Wallace, L. M. et al. DUX4, a candidate gene for facioscapulohumeral muscular dystrophy, causes p53-dependent myopathy in vivo. Ann. Neurol. 69, 540–52 (2011).
36. Geng, L. N. et al. DUX4 activates germline genes, retroelements, and immune mediators: implications for facioscapulohumeral dystrophy. Dev. Cell 22, 38–51 (2012).
37. Yao, Z. et al. DUX4-induced gene expression is the major molecular signature in FSHD skeletal muscle. Hum. Mol. Genet. 23, 5342–52 (2014).
38. Homma, S., Beermann, M. Lou, Boyce, F. M. & Miller, J. B. Expression of FSHD-related DUX4-FL alters proteostasis and induces TDP-43. Ann. Clin. Transl. Neurol. 2, 151–66 (2015).
39. Jagannathan, S. et al. Model systems of DUX4 expression recapitulate the transcriptional profile of FSHD cells. Hum. Mol. Genet. 25, 4419–4431 (2016).
40. Jones, T. I. et al. Facioscapulohumeral muscular dystrophy family studies of DUX4 expression: evidence for disease modifiers and a model of pathogenesis. Hum. Mol. Genet. 21, 4419–30 (2012).
41. Campbell AE, Shadle SC, Jagannathan S, Lim JW, Resnick R, Tawil R, van der Maarel SM, Tapscott SJ. NuRD and CAF-1-mediated silencing of the D4Z4 array is modulated by DUX4-induced MBD3L proteins. Elife. 2018 Mar 13;7. pii: e31023. doi: 10.7554/eLife.31023 (2018).
42. Kolata, G., Reanimated 'Junk' DNA Is Found to Cause Disease. New York Times, Science. Published online: August 19, 2010 http://www.nytimes.com/2010/08/20/science/20gene.html (2010).
43. Nurk S, Koren S, Rhie A, Rautiainen M, Bzikadze AV, Mikheenko A, Vollger MR, Altemose N, Uralsky L, Gershman A, Aganezov S, Hoyt SJ, Diekhans M, Logsdon GA, Alonge M, Antonarakis SE, Borchers M, McCoy RC, Dennis MY, Alexandrov IA, Gerton JL, O'Neill RJ, Timp W, Zook JM, Schatz MC, Eichler EE, Miga KH, Phillippy AM. The complete sequence of a human genome. Science. 2022 Apr;376(6588):44-53. doi: 10.1126/science.abj6987. Epub 2022 Mar 31. PMID: 35357919 (2022).
44. Jagannathan S1,2,3, Ogata Y4, Gafken PR4, Tapscott SJ3, Bradley RK1. Quantitative proteomics reveals key roles for post-transcriptional gene regulation in the molecular pathology of facioscapulohumeral muscular dystrophy. Elife. 2019 Jan 15;8. pii: e41740. doi: 10.7554/eLife.41740 (2019).
45. Bouwman LF, den Hamer B, van den Heuvel A, , Tapscott SJ, Rigo F, van der Maarel SM, de Greef JC. Systemic delivery of a DUX4-targeting antisense oligonucleotide to treat facioscapulohumeral muscular dystrophy. Mol Ther Nucleic Acids. 2021 Sep 27;26:813-827. doi: 10.1016/j.omtn.2021.09.010. PMID: 34729250; PMCID: PMC8526479. (2021).
46. Soliman HAN, Toso EA, Darwish IE, Ali SM, Kyba M. Antiapoptotic Protein FAIM2 is targeted by miR-3202, and DUX4 via TRIM21, leading to cell death and defective myogenesis. Cell Death Dis. 2022 Apr 25;13(4):405. doi: 10.1038/s41419-022-04804-x. PMID: 35468884; PMCID: PMC9038730. (2022).
47. Guo D, Daman K, Chen JJ, Shi MJ, Yan J, Matijasevic Z, Rickard AM, Bennett MH, Kiselyov A, Zhou H, Bang AG, Wagner KR, Maehr R, King OD, Hayward LJ, Emerson CP Jr. iMyoblasts for ex vivo and in vivo investigations of human myogenesis and disease modeling. Elife. 2022 Jan 25;11:e70341. doi: 10.7554/eLife.70341. PMID: 35076017; PMCID: PMC8789283. (2022).

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