Annual Report.

2015

FSHD
Global Research
Foundation Ltd

Our Mission

The Foundation is committed to advancing global medical research, education and collaboration to improve quality of life and ultimately find a cure for Facioscapulohumeral Dystrophy. Through transparency, accountability, good governance and pure passion we aim to achieve results as quickly as possible.

(02) 8007 7037 admin@fshdglobal.org www.fshdglobal.org PO Box A296, Sydney South, NSW 1235

Chairman's Address

Dear Friends,

On behalf of the Board of Directors, staff and volunteers at the FSHD Global Research Foundation, I would like to extend our appreciation and gratitude to the many people who have generously donated time, services and funds over the past year to assist the Foundation in realising our mission.

Facioscapulohumeral dystrophy (FSHD) is a debilitating muscle wasting disease that impacts both adults and children with devastating physical and psychological effect. The continued support from our community of donors gives a message of hope to families affected by this terrible disease. A message that something is being done to help find treatments and an ultimately a cure. The positive effect of this cannot be underestimated.

Since we were founded in 2007, FSHD Global has committed approximately $7.5 million to fund 33 on going medical research grants and education programs across nine countries. Within the next six months we aim to commit a further $1.5 million towards new medical research and education, focusing on improving Australia's Diagnostics, providing new funds for the development of therapeutics as well as launching a clinical trial, to build muscle strength in the body.

I am proud of the Foundation's continued transparency, accountability and innovation, leading world's best practice in the field. In a short period of time the Foundation has achieved great momentum and incredible advances, funding world leading researchers who work tirelessly to understand FSHD and find treatments and a cure.

Thank you for your ongoing support and determined effort to help us dramatically advance the pace and direction of research into FSHD. I'd like to thank you for helping make the Foundation the success it is.

FSHD is a terrible condition, but it is within our power to change the lives of future generations who are affected by FSHD.

Bill Moss AO
Chairman & Founder
FSHD Global Research Foundation

FSHD is hereditary

30% of patients have no prior family history oFSHD

It affects skeletal muscles in the body

There is no cure

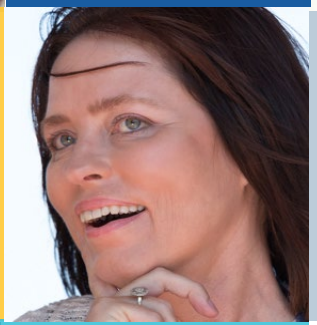
100% of all tax deductible donations fund research

The degree of muscle loss varies

It affects one in every 7,500 Australians

The age of onset can range from infancy to adulthood

No government funding in Australia



FSHD Global is an award winning charity

33 ongoing medical research grants in 9 countries

A common form of Muscular Dystrophy

Managing Directors Address

Since the beginning of the year the Foundation has been quick to achieve, innovate and motivate, setting a new tone and pace in advancing medical research and medical education across the globe.

2015 focused on increasing; fundraising, community engagement, corporate partnerships and medical innovations as we continued to grow in our search for treatments and an ultimate cure.

The 2014/2015 financial year raised a record $3 million which saw a dramatic increase on last year's revenue, thanks to support from our community, and in particular 'Donor of the Year', Babak Moini for his generous million dollar donations.

It remains our objective to fund National, International and Australian Collaborative research projects. The success of the year, led to nine (9) new medical research grants.

We funded a study to look at the bones of people with FSHD, identifying the relationship between weak muscles and weak bones, resulting in a study that will ultimately help health professionals plan treatments to help people with FSHD build their bones. In addition to funding medical research in the areas of Basic Science, Therapeutics and Diagnostics, in 2015 the Foundation gathered a group of world leading FSHD clinicians and researchers to develop a Consensus Statement identifying diagnosis and management of the disease. This paper will be published in a leading journal and become a key tool educating the medical sector about the disease, improving standard of care for Australians living with FSHD.

Actively growing our global footprint of research and education, 2015 welcomed an internal restructure and expansion of the core team behind, improving the operations, communications, brand, voice and fundraising programs that complement our overall strategy to reach a cure.

Remaining innovative and unique in our 100% donation model, the Community, Board of Directors, Scientific Committee Members, Staff and a handful of volunteers enable FSHD Global to continue to achieve key milestones and advance a global footprint on research.

Our passion, commitment and achievements during 2015, highlighted to the world we are a small organisation doing some very big things!

Natalie Moss
Managing Director
FSHD Global Research Foundation

About the Foundation

The FSHD Global Research Foundation focuses on finding treatments and a cure for FSHD. In doing so, we fund world-class medical research, awareness and education. We are also committed to complete transparency and accountability in our operations.

The Foundation was established in 2007 by Bill Moss AO, a well-known Australian businessman, philanthropist and sufferer of FSHD. Since then, we have been addressing the chronic lack of medical funding and awareness of FSHD, both in Australia and globally.

Over the past 9 years, the Foundation has committed $7.5 million to fund 33 ongoing medical research grants in 9 countries; the USA, Canada, the Netherlands, Italy, France, Belgium, Spain, New Zealand and Australia.

The FSHD Global Research Foundation does not operate like an average not for profit. **We allocate 100% of the tax deductible donations** we receive to current and future medical research grants. We are also transparent in doing so, offering all donors via the 'FSHD – Find the Cure' mobile app the opportunity to track exactly which research programs their money has been allocated and the latest milestones of those programs.

The main sources of our funding for FSHD research are individuals afflicted by FSHD, their friends, supporters, as well as corporate sponsors. All funds donated are invested through careful consideration, guided by our Scientific Advisory Boards, Board Directors and International Research Committees, ensuring FSHD Global remains a leader in discovering world's best science.

In its truest sense, the FSHD Global Research Foundation is a small organisation doing some very big things!

What is FSHD?

Facioscapulohumeral muscular Dystrophy (FSHD) is a genetic neuromuscular disease causing significant medical and health impacts on individuals, families and society. It is characterised by the progressive weakening and loss of skeletal muscles.

There is no known cure or effective treatment for FSHD.

The majority of people living with FSHD are diagnosed by the age of thirty, with an increasing proportion of children being diagnosed under the age of five. These early onset or infantile patients have more severe symptoms and added health complications.

FSHD is estimated to affect 1 in every 7,500 Australians, however, this number could be higher as FSHD is commonly misdiagnosed. These levels, make FSHD one of the most common forms of muscular dystrophy.

Despite the fact that FSHD effects around 3,200 Australians, in Australia there remains no government funding for FSHD research. Internationally the level of funding is minimal. As a result, FSHD research is estimated to lag behind research into other forms of muscular dystrophy by about twenty years.

The name of the disease comes from the areas of the body in which muscle deterioration is most commonly first observed – the face (facio), shoulders(scapulo) and upper arms (humeral). Weakness in the muscles of the eye (to open and close) and mouth (to smile, pucker and whistle) are also characteristic of FSHD in its early stages. It is common for a combination of these symptoms to form the basis of a doctor's initial diagnosis.

The rate of progression of FSHD is variable, yet it tends to be slow in most cases. From the muscles of the face and upper body, it generally moves down to the abdominal and foot-extensor muscles. Signs of deterioration include "foot drop" (significant weakness in the movement of the ankle and toes), "scapular winging" (abnormal protrusion of the shoulder blade) and difficulty reaching above shoulder level due to progressive weakness in the stabiliser muscles of the shoulder.

Many people with FSHD may experience serious speech impediments caused to the weakening of facial muscles. More than 50% of patients experience high-frequency hearing loss, and may also develop abnormalities in the blood vessels at the back of the eye leading to vision problems.

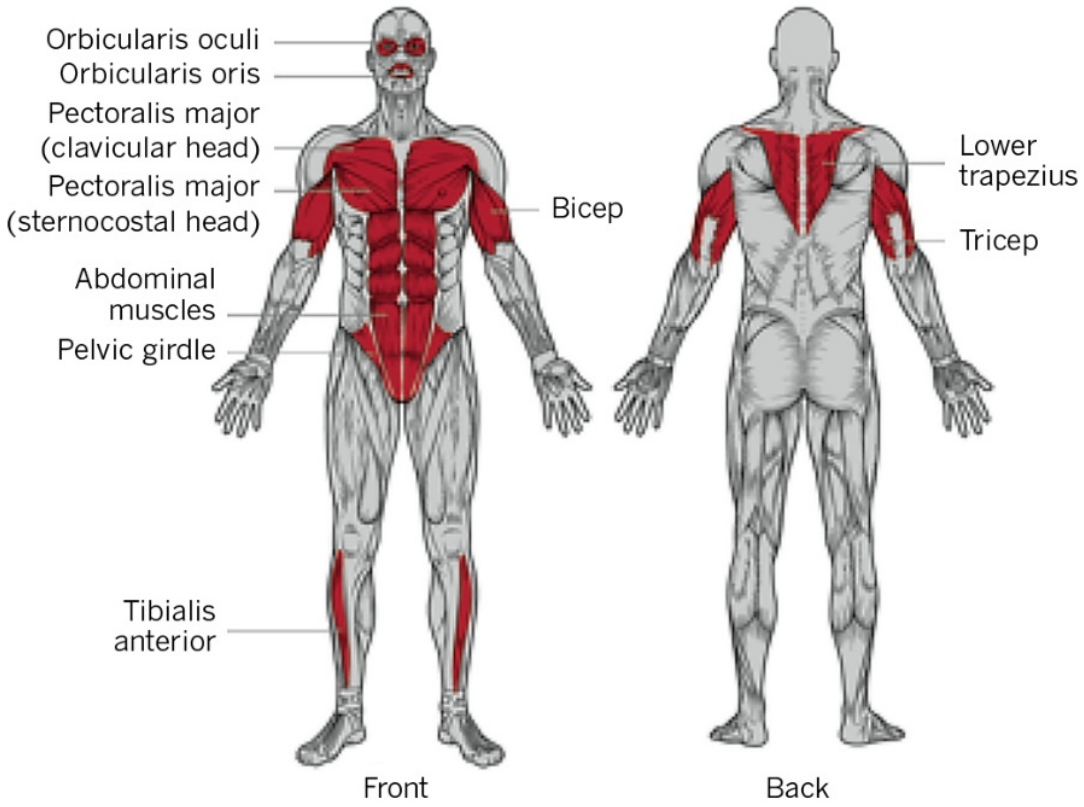
A consensus about the cause of FSHD was reached in 2014. The symptoms of FSHD are caused by the production of a protein called DUX4. This protein plays a normal role in early foetal development, but it is highly toxic when produced in adult muscle tissue. In people without FSHD, the DUX4 gene is repressed and levels of DUX4 protein are low. For people with FSHD, the DUX4 gene is not repressed and the toxic protein is produced.

While many hereditary diseases are caused by a single genetic defect, the production of DUX4 and the development of FSHD can actually result from defects in two different chromosomes. The majority of sufferers (95%) have what is called FSHD 1 which is caused by a defect on Chromosome 4. The remainder (5%) have FSHD 2 which is caused by a defect on Chromosome 18.

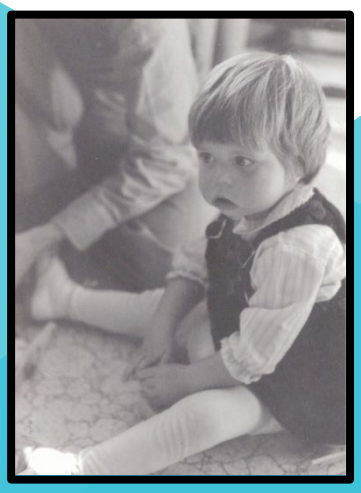
This scientific complexity, coupled with the shortage of research funding, makes the search for a cure even more challenging.

FSHD Global Research Foundation Ltd

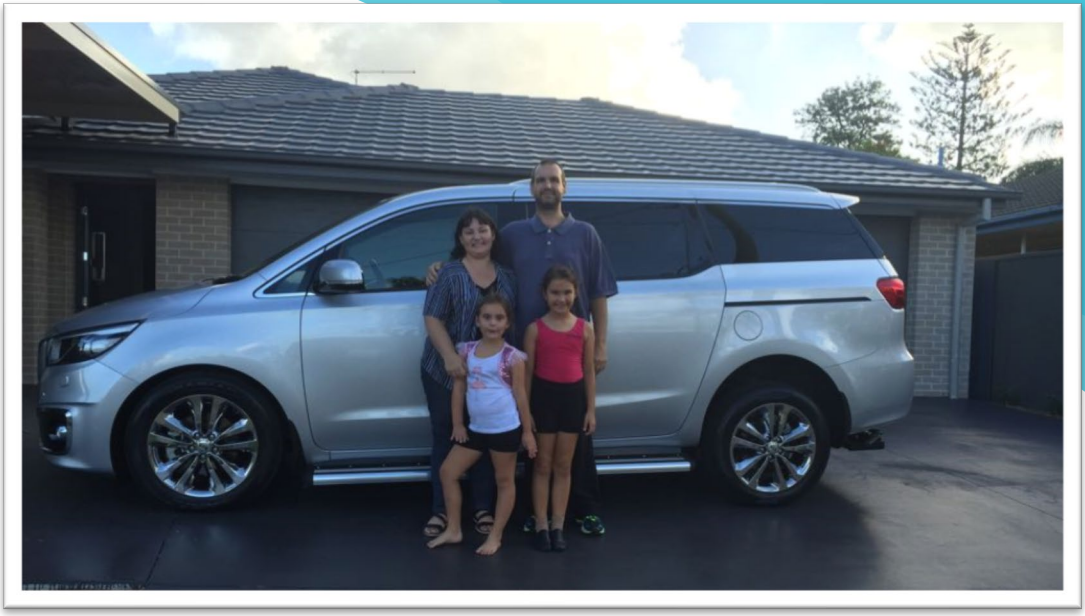
FACIOSCAPULOHUMERAL DYSTROPHY



This diagram shows the muscles that are typically affected in FSHD. Reprinted from Trends in Molecular Medicine, 2015 May; 21(5):295-306, Lek A, et al. Copyright 2015, with permission from Elsevier.



Living with FSHD: Emma's Story



Having been diagnosed with FSHD only about 3 years ago, my progression has been swift and in a lot of ways I have not been prepared, either emotionally or financially for this new life direction. About 12 months ago, I started using a wheelchair and I was most surprised to find that it has not been the negative and limiting step I had thought it would be, it has liberated me and meant I can get out and about with my family and do the things I love. Unfortunately, it has also meant that I had a lot of lifting putting it in and out of my little hatch back car. This lifting took its toll and has resulted in me dislocating my shoulder and tearing the ligaments which required surgery in late July. I can't lift the manual chair anymore, or even manage to self propel since my surgery, so the electric chair is the way forward for me. There will be no way I can fit an electric chair into my little hatch back, with even fitting the manual chair in is like playing a game of tetris.

This has been a huge threat to my independence because unless my husband is with me, I currently can't go anywhere that requires more than a very short walk. On 4th July 2015, FSHD Global Research Foundation held their annual Chocolate Ball, with the theme of "Independence Day". I was given the honour of speaking at the Chocolate Ball, sharing my story on living with FSHD. In keeping with the theme, I spoke about how I use health aids and other tools and equipment to maintain as much independence as possible. In my speech I talked about this and about how this injury has meant I now have to purchase an electric wheelchair and a new car that will accommodate it. Of course, the ultimate goal towards independence is a cure for FSHD. While we wait for that though, I would still love to be able to work, take my kids to school excursions and family outings, go shopping, go out with friends and live life to the fullest so the chair and car are both very important. The intention I had when sharing some of my personal fears, challenges and victories in living with FSHD, was to give the guests at the ball some insight into what it is like to live with this relentless condition, and help everyone there to understand what a cure will mean to me, and all FSHD patients. After my speech had finished, I was back at my table enjoying one or perhaps two glasses of Moet (just to relieve the nervous tension of course!), when our gorgeous host, Jamie Durie, made an announcement. A generous donor had pledged $150,000 to the foundation and had also decided to donate a car for me! I was absolutely speechless. In complete disbelief. I may have cried. Not only did this generous man purchase a car suitable for wheelchair conversion, he has organised for Automobility to convert my car so that it will have an electric ramp and I can drive right into the back seat and either remain in my chair for the drive, or exit the car and drive from the drivers seat. My car is in at the workshop right now and should be completed in the next month or so. I am still completely overwhelmed with gratitude

and cannot believe that anyone would be so kind and generous to someone they do not know. Even though I am working in a good job as an Accountant, this kind of car and conversion would have been well out of our reach financially. I had just accepted that the time would come where I could really only go out with my husband or another carer and that I would have to rely on public transport but with this amazing gift, I will be travelling independently and reliably for a long time yet. Attending the Chocolate Ball changed my outlook on dealing with this disease. Listening to Bill Moss share his story, the wonderful scientists and researchers who are dedicating their lives towards a cure, and of course having the opportunity to share my story was amazing. After I spoke, I had so many people come and speak to me and everyone was so supportive and positive, it was amazing. But then to also have this gift of a modified car, well that just still doesn't seem real or possible. I don't know that I will ever be able to pay it forward to this degree, but I hope that I can continue to live my life in a way that makes me a deserving recipient to this blessing. I can't wait to see it, I don't even know what colour it is! My girls were home when the representative from Automobility brought the demonstration model to our house and they are very excited about the side opening doors and the fact that I will sit in the back with them.

I am planning on attending the Chocolate Ball again next year, and by then I should have the car and my new chair so I look forward to seeing many of you there and telling you all about it. It is an amazing, inspiring and hope filled night of spectacular proportions, and for me, it has been life changing.

The Finances

For the year ended 30 June 2015

	Notes	2015	2014
		$	$
Donations		2,744,960	894,561
Fundraising		297,058	459,194
Other income	3	109,852	70,346
		3,151,870	**1,424,101**
Grants made	4	(650,139)	(490,170)
Fundraising expense		(322,652)	(381,741)
Education programs		(110,063)	(61,290)
Sponsorship		-	(90,290)
Employee expense		(347,916)	(259,051)
Other expenses		(17,244)	(16,149)
Surplus for the year		**1,703,856**	**124,791**
Other comprehensive income:			
Other comprehensive income		-	-
Other comprehensive income for the year, net of income tax		-	-
Total comprehensive income for the year		**1,703,856**	**124,791**

Assets	Notes	2015	2014
		$	$
Current			
Cash and cash equivalents	5	2,666,053	1,198,113
Trade and other receivables	6	104,059	24,538
Financial assets	7	1,174,171	642,672
Other assets	8	6,750	17,762
Current assets		3,951,033	1,883,085
Non-current			
Property, plant and equipment	9	2,925	758
Non-current assets		2,925	758
Total assets		**3,953,958**	**1,883,843**
Liabilities			
Current			
Trade and other payables	10	369,003	4,934
Provisions	11	11,436	9,246
Current liabilities		380,439	14,180
Total liabilities		**380,439**	**14,180**
Net assets		**3,573,519**	**1,869,663**
Equity			
Retained earnings		3,573,519	1,869,663
Total equity		**3,573,519**	**1,869,663**

Grants made

	2015	2014
	$	$
Australian research grants		
Monash	15,909	10,000
Concord Hospital	69,000	
Novagen/Genea	90,909	

Baker IDI	95,833	
Collaborative research grants		
Institute de Marseille Luminy / Sydney		50,000
Fodazione Centro San Raffaele Tabor, It/Sydney		100,000
Ohio WA		95,244
Lopez Castel	25,000	
Minnesota	60,000	
Children's Medical Centre	50,000	
Cooperative International Research Group	50,000	
Kennedy Kreiger Institute	36,000	
Uni. Otago NZ	10,774	
Uni Masssachuset	50,000	
Leiden Netherlands / QLD		54,869
Alberta Children's Hospital Canada / Sydney / Melbourne International Research		30,057
Fondazione Centro San Raffaele Tabor	30,000	60,000
International research grants		
Minnesota University	66,714	90,000
Total grants paid	**650,139**	**490,170**

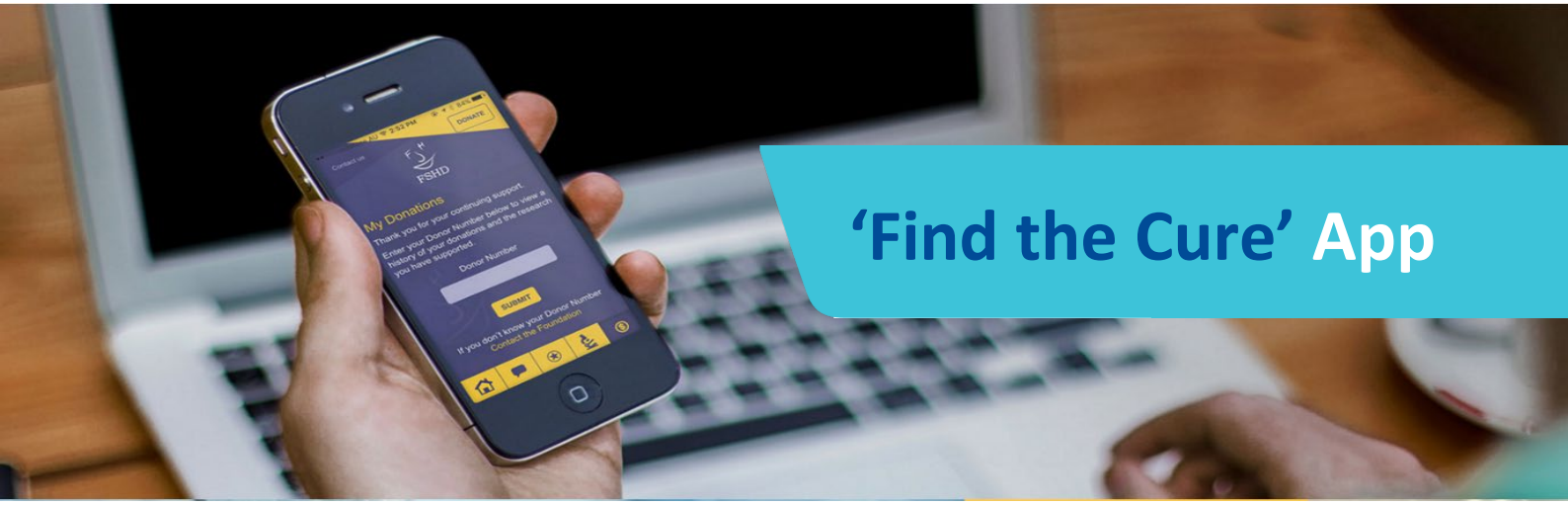
Our 100% Model

The FSHD Global Research Foundation is a pure Australian not-for-profit organisation investing 100% of all tax deductible donations funding both basic epigenetics and therapeutic clinical trials, establishing drug developments to prevent muscle wasting. With a global footprint on science, our award winning structure relies on non-tax deductible revenue and sponsorships to support all overhead expenses.

We are proud of our culture and are committed to doing even more for research. Our Board of Directors, Basic Science, Sub-committees, Patrons, Ambassadors, staff and volunteers are vastly experienced in their respective fields and continue to make excellent contributions to the Foundation.

Executed by two separate bank accounts, 100% of all tax deductible donations fund global research, with the other non-tax deductible account funding operations. To keep this sustainable pure model, the Foundation took an innovative outlook and now seeks Corporate Australia to sponsor employees, ensuring costs are kept to a minimum. This ingenious structure has enabled the Foundation to grow in scale, speed and achievement.

FSHD Global's Head Office operates from an office space donated by a legal firm in Sydney CBD. We also have branches in all states of Australia to expand our education programs nationally. The State Branches are run by volunteers many of whom are FSHD sufferers themselves.



The FSHD Global Research Foundation believes charities should operate like businesses in communicating clearly, transparently and consistently to their donors and stakeholders how donations are being spent.

This is why we built the FSHD - Find the Cure 'app' for mobile devices, allowing us to communicate more directly than ever with our donors.

This Australian first app allows every donor since our inception to track their donation and see the research grant/s to which their money has been allocated. The app also provides the latest milestones of each of these grants.

The award winning app is free to download at GooglePlay or at the Itunes Store.

Fundraising

Built as an innovative and pure charity, FSHD Global allocates **100% of all tax-deductible donations** to fund treatments and a cure for FSHD. The Foundation's operations are supported by non-tax deductible sponsorships.

Our funding is reliant on individuals, Corporate Australia and families affected by this disease. With no government support we have established a number of unique and iconic fundraising initiatives to create greater awareness and funding for this misunderstood disease.

2016's Event Calendar features both iconic annual and new events, ensuring value for money, networking and sponsorship opportunities for our community at large. Some events include the Sydney Chocolate Ball, FSHD Global Golf Tournaments, Poker Night, Luxe Luncheon, Surf Challenge, and other events.

Our most prestigious event of the year is the highly anticipated **7th Annual FSHD Sydney Chocolate Ball**, held Saturday 28th May. Hosted by Jamie Durie OAM, this extravagant night will welcome 650 guests to experience world class entertainment, a chocolate inspired menu designed by celebrity chef Luke Mangan, and an abundance of champagne.

Traditionally a sold out event, this night of nights sees support from Corporate Australia's executive management across various industries, making it one gala dinner not to be missed!

To volunteer or attend an event, please contact Natalie Livet at natalie.livet@fshdglobal.org

Meet the Team

Board Members

Bill Moss AO **Chairman**	**James Wakim** Deputy Chairman	**Natalie Moss** Managing Director	**Andrew Rigney** Director/Secretary	**Barry Robinson** Director	**David Mackay** Director	**Glenn Willis** Director
Malcolm Beville Director	**Nigel Virgo** Director	**Alan Watts** Director	**Dr Pradnya Dugal** Director	**Rohan Hardcastle** Director	**James Harvey** Alternate Director	**Bev Baker** Alternate Director

Our Core Team

Natalie Moss Managing Director	**Olivia Hibbitt** Medical Science Liason	**Natalie Livet** Director of Operations	**Stephanie Whittles** Administration & Finance Coordinator	**Danielle Thompson** Director of Events	**Jason Irvine** Partner & Relationship Manager

Scientific Working Committee

Alan Watts **Chairman**	**Olivia Hibbitt** Member	**Natalie Moss** Member	**Glenn Pilkington** Member	**Scott Baker** Member

Scientific Advisory Board: Basic, Diagnostic and Therapeutic Panel - EOI's

Bill Moss AO **Chairman**	**Dr. Alan Watts** **Chairman**	**Olivia Hibbitt** Member	**Dr. John Rasko AO** Member	**Charles Emerson** Member	**Stephen Tapscott** Member	**Baziel Van Engelen** Member
Natalie Moss Member	**Dr. Pradnya Dugal** Member	**Noel Chambers** Member	**Monique Ryan** Member	**Lucy Burns** Member	**Alexandra Belayew** Member	

FSHD
Global Research Foundation Ltd

Achievements

FSHD Global is faced with two key challenges;

- FSHD is considered a rare, orphan disease.
  - It has been our challenge to actively engage with a community who have little to no understanding of the disease, nor friends or relatives affected by FSHD
  - FSHD Global has no budget for media, advertising or brand promotion
  - FSHD Global is not a very well-known charity in the marketplace. We have raised significant profile considering our size and industry, and have achieved great media due to our medical breakthroughs, and voice on philanthropy.

Despite this, our Foundation has achieved amazing things.

- ✓ The Foundation has received $7.5 million dollars in donations since inception in 2007 – with no government support
- ✓ 100% of all tax deductible donations are invested into Medical Research and Medical Education across the globe.
- ✓ The 2015 Chocolate Ball raised over $1 million making it the most successful event to date.
- ✓ The Foundation allocated $2.3 million dollars advancing medical research in 2015, a 216% increase on previous years donation income
- ✓ Actively increased our community engagement and following, expanding our database by 15%
- ✓ The Butterfly Effect Campaign - turning $1 million into $2 million
- ✓ The current year has seen incredible advances towards clinical trials focusing on treatment and new drug developments preventing muscle wasting
- ✓ With a global footprint FSHD Global has funded 33 ongoing medical research grants across 9 countries
- ✓ The FSHD – Find a Cure' App allows donors to track exactly which grant(s) their donation has been invested in, around the world
- ✓ FSHD global is a multi-award winning Foundation
- ✓ 2015 saw a 25% increase in medical publications acknowledging the Foundation
- ✓ Our Board of Directors, National & International Science Advisory Boards, Patrons and Ambassadors receive $0 remuneration.
- ✓ Our Founder and Chair, Bill Moss AO, was a Finalist in the 'Stem Cell Person of the Year Awards' an international platform acknowledging him and the Foundation for establishing the world's first FSHD Human Embryonic Stem Cell Bank
- ✓ Education programs increased by 80% on previous year, with the highlight having established a Consensus paper on the management and treatments of FSHD, a valuable tool for educating the Australian medical sector and beyond.

Allocation of Tax Deductible Donations

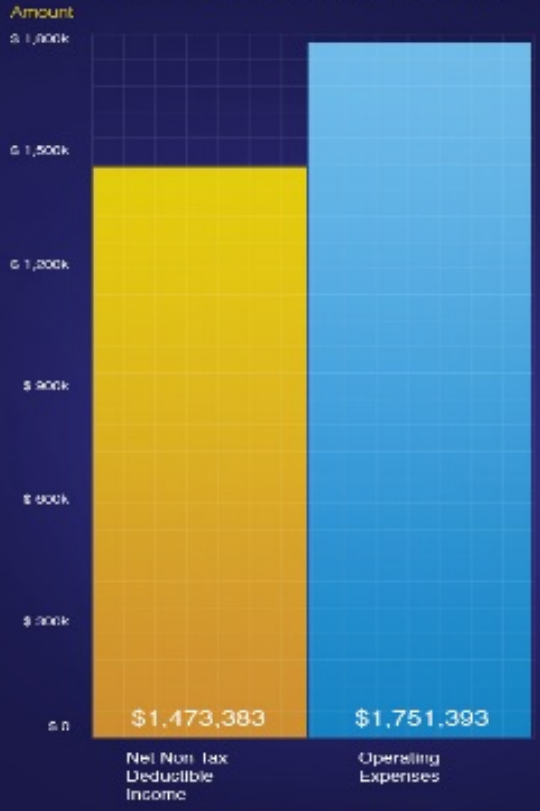
FY 2008-2015: As of 30th June

*In keeping with the Charter of the Foundation, we aim to invest 100% of all tax deductible donations into Medical Research Grants & Medical Research Education.

*Unaudited figures

Net Non Tax Deductible Income vs Operating Expenses

FY 2008-2015: As of 30th June



The 2015 Sydney Chocolate Ball was held on the 4th July, therefore the income and expenditure from this event will be included in the 2016 figures.

The Foundation continues to ensure 100% of its tax deductible donations are invested towards medical research.

*Unaudited figures

Research Funded

FY 2008-2015

- Funded for Australian research
- Funded for Australian research in collaboration with International research
- Funded for International research

*Unaudited figures

FSHD
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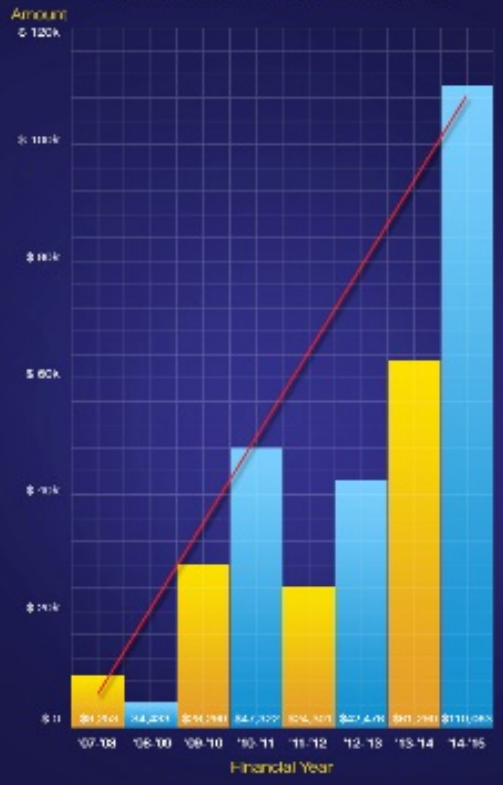
Medical Research Education Funded

FY 2008-2015. As of 30th June



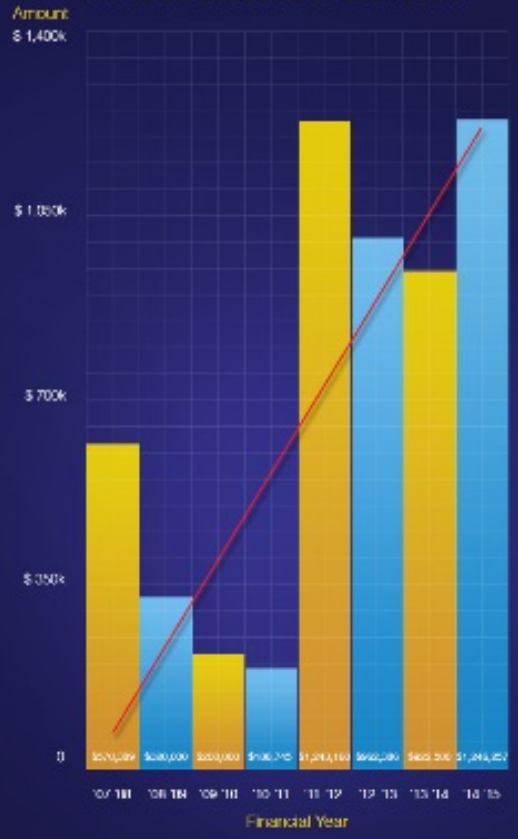
Education includes sending Australian scientists to domestic and international conferences, sponsoring international researchers to lecture in Australia and Sponsorship for worldwide scientific meetings.

$327,430 total cost of education FY 2008-2015

*Unaudited figures

Medical Research Approved

FY 2008-2015: As of 30th June



$5,653,445 Approved in total
$3,226,449 has been paid as at end of FY 14/15
$2,426,996 has been allocated for distribution over future financial years

*Unaudited figures

FSHD
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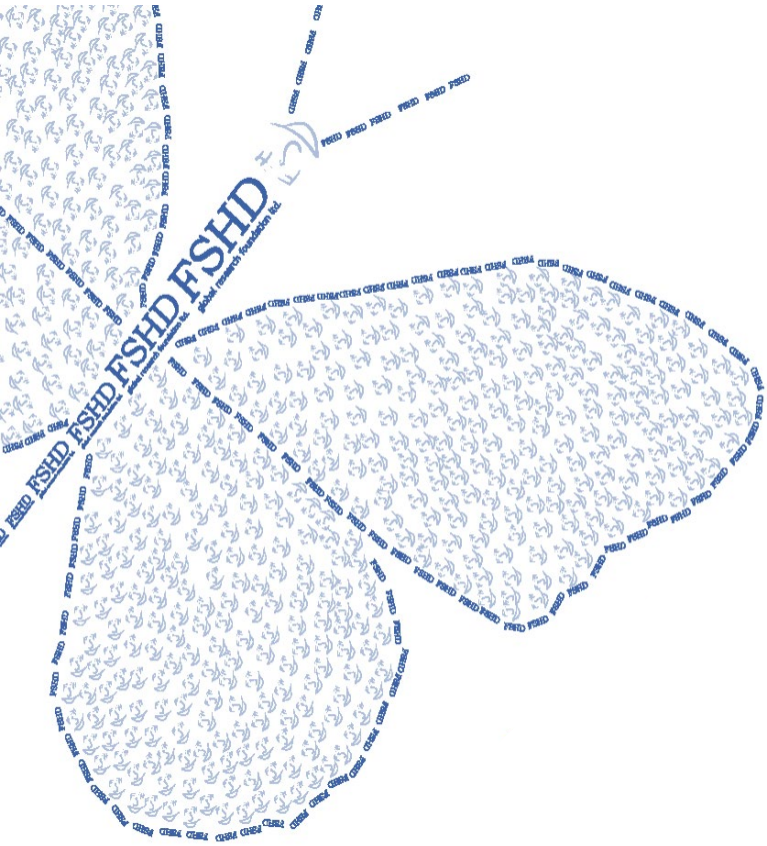
2015 Donor of the Year

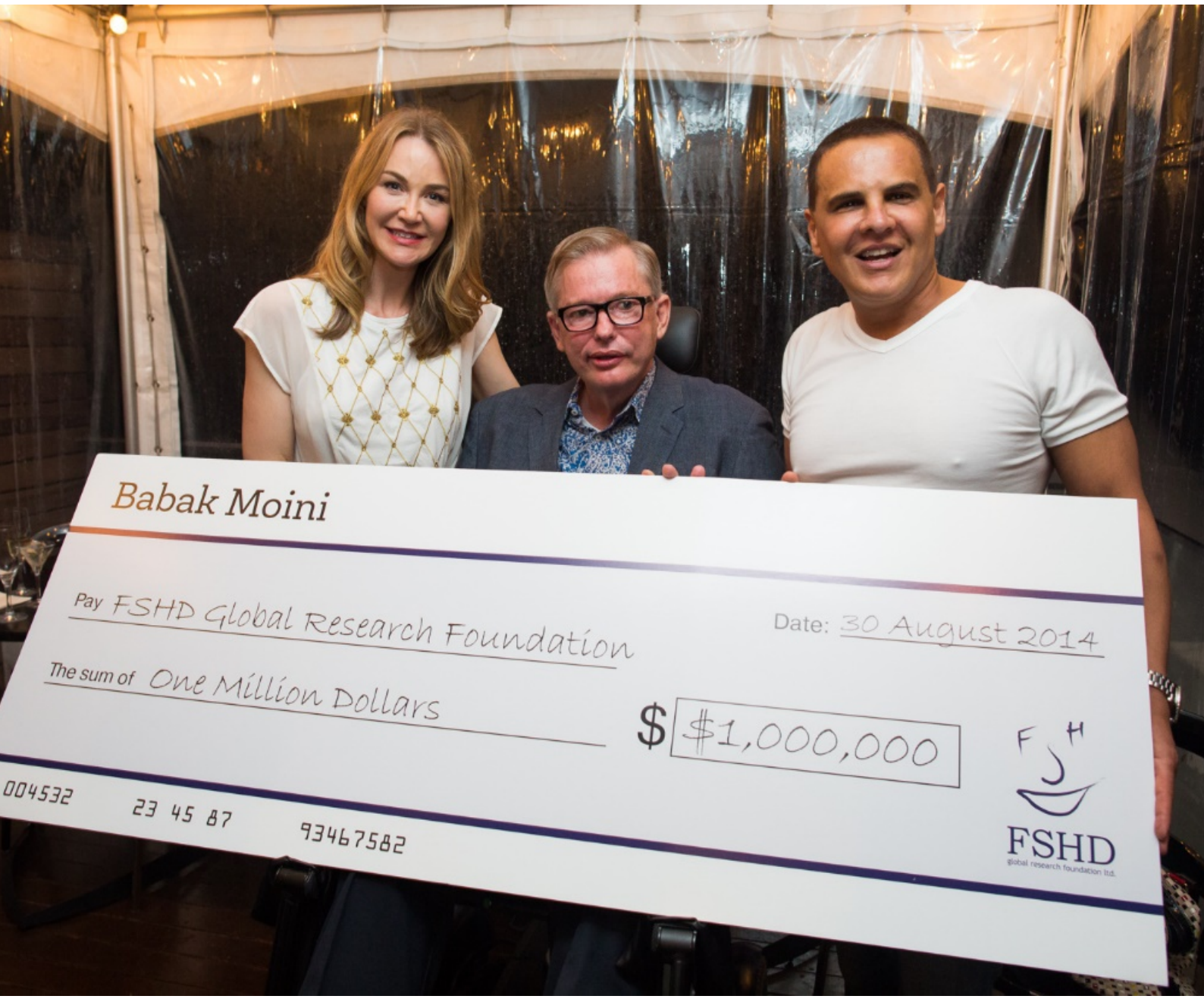
Congratulations to businessman and philanthropist Babak Moini for becoming FSHD Global's 2015 Donor of the Year. We were proud to acknowledge such a remarkable member of our community. Not only has he been an incredible support to our Foundation, he inspired our 2015 EOFY "Butterfly Effect Campaign".



Our strategy and message was simple; encourage Australians to seek transparent charities to support, challenging the culture of giving by shifting younger generations and new wealth to be creative and active in their moral commitment, giving back to society. In turn, this matching campaign successfully turned $1 million into $2 million within a two month period. This story attracted national media and enabled us to spearhead a 216% increase in donation income on 13/14 FY, raising $2.3 million, with no budget for the campaign.

Babak's partner, Rochelle Collis, has also been a huge part of the Foundation this year. She hosted the first Ladies Luxe Luncheon which was a huge success.

“I couldn’t if I tried spend more money; I now feel we have a social responsibility now we’ve got to this point, to share the wealth and invest the wealth for the betterment of society” – Babak Moini



“As long as you have a positive attitude, you can do anything...”

In loving memory of Monica Ellis

On March 4 2015, the Foundation lost its Founding Ambassador, enthusiastic supporter and valued friend, Monica Jean Ellis.

To say Monica is missed simply does not do justice to the amazing life she lead, the wonderful example she set and the richness she added to the lives of all who knew her.

Monica was diagnosed with Infantile FSHD when she was only five years old. Everything FSHD stole from her, her dreams and plans, she dreamt new dreams and made new plans.

First came the inability to blink properly and then she lost her smile. Later came the weakening in her shoulders, the inability to walk and then the inability to sit up. She lost the ability to chew and speech became difficult, but Monica refused to let this disease describe her.

It was difficult not knowing what was wrong or what could be done, and then later, when FSHD was diagnosed, there was so little knowledge regarding how this particular dystrophy progressed or how it could be treated. Lack of knowledge also meant medical professionals sometimes made wrong assumptions because they were unaware of the condition. After all, there had been no focused research on the disease for over twenty years.

She was her own person, she knew what she wanted and she refused to let what she could not do get in the way of what she could do. As she often said “having a disability just throws a spotlight on other choices”

Monica had a wicked sense of humor. Once when being pushed through the surf in her wheelchair and being teased about sharks she quipped “yeah, meals on wheels.

She was a talented artist and painted from her view of the world. From this perspective she produced the most wondrous fantasy artworks. She created “Silly” who became her alter ego. Silly did and explored the things that Monica simply was not physically able to.

Monica was, and remains an inspiration. FSHD was very cruel to her but she did not complain. She worked beside the Foundation looking for a treatment or a cure. Monica new any cure would be too late for her but she was tireless in her interest and willingness to assist.

Silly angels in a starry Christmas night...

The Science behind it all

The Foundation is currently playing a pivotal global role in the FSHD research arena. FSHD Global is the only funder of FSHD research in Australia and the largest funder of FSHD research worldwide (outside the American Government), enabling us to shift the culture of medical research encouraging commercialisation of potential treatments at a greater speed with vision and voice.

There have been many surprising outcomes from the research and education funded by FSHD Global. It is shedding light on other muscular conditions and many unrelated debilitating and life threatening diseases such as cancer.

The Foundation not only sponsors laboratories and Principal Investigators to undergo interesting research, we sponsor incredible people to speak at both worldwide and national events, educating members of both the medical and public sectors.

FSHD has no cure, limited treatments and poor diagnostics in Australia. To support clinicians who are working with people with FSHD, in September 2015, the Foundation hosted 11 prominent global leaders, bringing experts from all areas of the disease to form a consensus on current and innovative treatments, therapies and management for the disease. This collaborative paper will be published and used to educate Australian doctors and patients on symptoms and therapies to assist with managing and improving quality of life for patients. This is one example of how the Foundation is establishing effective education platforms across Australia and the world.

Our innovative structure and entrepreneurial spirit last year led the Foundation to support a socially responsible Biotech Investment Research Grant focused on finding a cure for FSHD. This biotech has aided research into the disease and bridge the gap between research and the pharmaceutical industry.

Innovation aside, research into FSHD is becoming exciting and tangible. 2016 marks the first Australian human clinical trial building muscle mass in people with muscular dystrophy. We are thrilled to announce this medical breakthrough and suggest it could assist other muscle wasting and rapidly aging diseases.

Grant 14

Principle Investigator: Dr. Francoise Helmbacher (in collaboration with Dr. Robin Fitzsimons)
Research Institution: IBDML, France
Type: International and Australian Research (Grant in Collaboration)
Primary Focus: Tissue-specific silencing of the planar cell polarity gene FAT1 as a causal mechanism for FSHD

Dr. Francoise Helmbacher's team is studying the link between a gene called FAT1 and FSHD, in collaboration with two other groups in Marseille and Paris. Although FSHD is associated with defects on chromosome 4 which causes production of DUX4 protein, other factors must contribute to appearance of FSHD symptoms. The team have observed that mice with no FAT1 gene have FSHD-like symptoms, and that one DNA abnormality in the FAT1 gene was more frequent in FSHD patients than in healthy individuals, adding to other known abnormalities in DUX4 inducing muscle wasting. To obtain further evidence that FAT1 dysfunction contributes to FSHD symptoms, they carried on additional studies. They studied the genes (DNA) in patients with FSHD-like symptoms but without the known FSHD1 or FSHD2 markers and found alterations in the FAT1 gene, affecting the FAT1 RNA that produces the FAT1 protein. Therefore, FAT1 mutations can cause FSHD-like symptoms, without DUX4 toxic protein. These FSHD-like cases are rare. Thus it is important to understand what impact FAT1 has in the more frequent FSHD1 and FSHD2 cases. The researchers then found lowered levels of FAT1 protein in fetuses and adults with FSHD and that lowered FAT1 protein correlated with FSHD disease severity and with earlier age of onset. Using muscle cells from FSHD1 patients grown in the lab, they demonstrated that this reduction was not caused by DUX4 protein, indicating that FAT1 is acting independently, but in parallel with DUX4 protein. Using FAT1 engineered mice, they showed that FAT1 also affects another type of tissue, called mesenchyme, to control muscle shape during embryonic development and correct muscle growth. Their results indicate that disrupting the FAT1 gene is an adverse factor in FSHD, correlating with symptom onset. The researchers are currently designing novel experiments to translate these results into treatments by correcting the FAT1 dysfunction, in order to alleviate the symptoms.

Grant 15

Principle Investigator: Prof. Scott Harper (in collaboration with Dr. Steve Wilton)
Research Institution: The Ohio University and Nationwide Children's Hospital Columbus, USA
Type: International and Australian Research (Grant in Collaboration)
Primary Focus: DUX4 inhibition as a therapeutic strategy for FSHD
Project goal: The overall goal of this project is to develop a translational strategy to treat FSHD

FSHD through DUX4 inhibition. FSHD is caused by expression of the DUX4 gene. This gene makes a protein called a transcription factor, which acts to turn other genes 'on' and 'off'. In FSHD, DUX4 seems to function by turning 'on' genes that are toxic in muscle. We therefore propose that treatments for FSHD should centre on inhibiting DUX4. We have developed a method to prevent DUX4 from being made in muscle, using a technology called RNA interference. This funding from FSHD Global has allowed us to show that RNA interference can reduce DUX4 toxicity. We anticipate that this treatment will best be delivered through the bloodstream, and we have

injected mice with this potential therapy at very high doses to determine if it is safe. We tested two different treatments, and we found that one showed some toxicity in the heart, spleen, and liver, while the other has so far proven safe to all organs. We are performing additional safety studies in anticipation that this RNA interference therapy could be tested in human trials.
We thank the FSHD Global Foundation and its donors for their generous support of our work.

Grant 16

Research Institution: Division of Regenerative Medicine, San Raffaele Scientific Institute
Principal Investigator: Dr Davide Gabellini (in collaboration with Australian neurologist Dr Robin Fitzsimons, Sydney Medical School, The University of Sydney)
Primary Focus: Re-creating the human chromosomal genetic defect responsible for FSHD in a mouse model.
Type: International and Australian Research Grant in collaboration
Status: Currently underway
Project goal: Creation of a mouse model that better reflects FHSD

Facioscapulohumeral muscular dystrophy (FSHD) is one of the most frequent forms of neuromuscular disease. Unfortunately, there is currently no treatment or cure for FSHD. The disease is associated with loss of genetic material (D4Z4) on chromosome 4 that is normally present in many copies toward one end of chromosome 4. FSHD patients have small D4Z4 copy number in their cells. Current animal models in mice are imperfect and the absence of an animal model that faithfully recapitulates key features of a human disease is a key step slowing down the development and test of therapeutic approaches. Unfortunately, D4Z4 repeats are present only in primates (humans, monkeys & apes) and not in laboratory animals such as mice. To solve this problem, Gabellini's group is inserting human D4Z4 and the genes that have been involved in FSHD into mouse stem cells using a technology called 'human artificial chromosomes'. This will generate animals that genetically resemble FSHD patients. This new animal model will allow scientists to study the pathways that are altered in the disease and test possible therapeutic approaches for future human clinical studies.

Dr. Gabellini's group has already transferred engineered genetic material into the intermediate mouse cells needed to finalize the production of the artificial chromosomes. These will then be transferred to mouse stem cells in order to generate "FSHD mice" and control mice which contain the human D4Z4 genetic material without the mutation responsible for FSHD.

Laboratory website: http://www.hsr.it/research/davide-gabellini/

Grant 19

Principle Investigator: Dr. Michael Kyba
Research Institution: University of Minnesota, USA
Type: International Research (Grant in Collaboration. Minneapolis & Boston)
Primary Focus: FSHD drug discovery based on chemical inhibitors of DUX4

Project goal: to identify novel small molecules that may be developed for the treatment of FSHD

Background:

The Kyba group has an ongoing research program to discover chemical inhibitors of the DUX4 protein. This work is directed towards screening and studying more complex compounds than we have evaluated previously, with the objective of discovering a compound that inhibits DUX4 primary activity, as opposed to compounds that inhibit downstream pathways. These are chemically synthesized, but more similar to natural products than to conventional drugs and have been identified through a screen of a library of complex compounds at the Broad Institute
at MIT, and to develop these leads in our laboratories here in Minnesota.

Summary of Results:

The Kyba laboratory has screened a ~35,000 compound chemical library for inhibitors of DUX4, the protein that is responsible for muscle deterioration in FSHD. They have identified several clusters of related compounds that are suitable for follow up medicinal chemistry to investigate potential as new drugs for FSHD. In the last stage of this research, they have prioritized compounds for follow up, and designed chemical synthesis routes, for the top 3 compounds. In the next phase of this research, they will synthesize one of these compounds, together with a diversity of related compounds, for further study on the inhibition of DUX4, and for testing in animals.

Grant 20

Principle Investigator: Dr. Davide Gabellini
Research Institution: San Raffaele Scientific Institute, Italy
Type: International Research (Grant in collaboration. Italy, Korea & Holland)
Primary Focus: Identification of drugs for the normalization of aberrant FSHD candidate gene expression
Project goal: to identify novel drugs that may be used to treat FSHD

The underlying genetic mechanism of FSHD has been known for 20 years. In spite of this there is no effective treatment available. One of the aspects of FSHD that complicates the development of treatments for the condition is the complexity of the issues the mutation causes. The mutation is responsible for the aberrant activity of as many as 18 different genes that code for proteins localized near the FSHD region (locus) on chromosome 4. Because of this FSHD could be caused by the cumulative effects of the combination of proteins being expressed. As a result, targeting the negative effect of just one of the proteins associated with FSHD would be unlikely to address all the symptoms of the condition. This makes the development of a therapeutic approach complicated.
A treatment allowing for a general normalization of the expression of all genes that are effected in FSHD may have a much better chance to ameliorate all the FSHD symptoms.

We have identified a non-protein-coding controlling element (called DBE-T) that behaves as a master regulator of the expression of the FSHD locus. Our results strongly suggest that by controlling the activity of DBE-T it is possible to normalize the aberrant expression of all the candidate genes in FSHD muscle cells.

Our goal is to obtain a drug to block DBE-T activity. To this aim, we are characterizing the fine details of the mechanism of action of DBE-T. In parallel, we are developing high capacity screen to identify molecules that can block the aberrant activity of DBE-T. The combination of these results will allow us to identify effective therapeutics that would prevent the aberrant protein production seen in people with FSHD.

Laboratory website: http://www.hsr.it/research/davide-gabellini/

Grant 21

Principle Investigator: Prof. Christina Mitchell
Research Institution: Monash University, Australia
Type: Australian Research Grant
Primary Focus: Drug-targeting of myoblast fusion as a treatment for FSHD
Project goal: to investigate potential therapeutics for FSHD

In previous studies funded by the FSHD Global Research Foundation, Professor Mitchell and her team reported the finding that FHL1, a positive regulator of muscle growth can reduce muscle wasting and improve the diseased muscle in FRG1 mice. FRG1 mice were engineered to have symptoms similar to human FSHD. The study identified potential new therapeutic targets for the treatment of FSHD. In recent findings, the Monash team have shown that some FSHD muscle cells have a defect in the way they grow and behave in vitro (in a test tube). These same defects can be observed in muscle cells from FRG1 mice. The group has just completed a study using an in vitro tested agent known as Tamoxifen that has potential to rectify the muscle cell defect as a therapeutic strategy against the muscle wasting that occurs in FSHD. Tamoxifen is routinely used to treat breast cancer and can be taken orally. Tamoxifen was delivered to FRG1 mice via a tamoxifen supplemented diet for a period of 12 weeks. During the study mice were assessed for weight gain and on completion muscle was collected for further analysis. Prof Mitchell and her team found that although FRG1 mice tolerated the treatment, analysis of muscle pathology revealed that tamoxifen did not improve the diseased muscle.

Grant 22

Principle Investigator: Prof. Silvère M. Van der Maarel
Research Institution: Leiden University Medical Center, Netherlands
Type: International and Australian Research (Grant in Collaboration)
Primary Focus: Increasing SMCHD1 Levels as a Therapy for FSHD

Summary:
The main aims of this project are to (a) determine the minimal SMCHD1 gene activity necessary to block the DUX4 toxic protein responsible for muscle wasting in FSHD, (b) to identify factors that regulate SMCHD1 protein levels and activity, and to identify ways to control SMCHD1 gene activity in order to treat FSHD.

Final Report:
The SMCHD1 gene on chromosome 18 encodes a protein that binds to the D4Z4 repeat on chromosome 4 to keep DUX4 repressed in muscle cells. FSHD is molecularly characterised by partial derepression of DUX4 in muscle and the presence of DUX4 in muscle is toxic to this tissue. Derepression in FSHD1 is the consequence of shortening of the D4Z4 repeat array, while in FSHD2 it is most often caused by mutations in SMCHD1.

In this project we established that 50% reduction in SMCHD1 level in muscle cells is sufficient to derepress DUX4. Conversely, a small increase in SMCHD1 levels is sufficient to repress DUX4 in FSHD1 and FHSD2 muscle cells. We have thereby provided proof-of-principle that accomplishing moderate increases in SMCHD1 levels is a plausible therapy for the majority of FSHD individuals. Our efforts are therefore directed at understanding the regulation of SMCHD1 and identifying means to increase SMCHD1 levels or activity in muscles of individuals with FHSD. To this end we have established methods and that will allow us to screen for molecules that increase SMCHD1 in muscle.

Grant 23a & b

Research Institution: Kennedy Krieger Institute, Baltimore, MD, USA & Concord Hospital, Sydney, NSW, Australia
Principle Investigator: Dr. Kathryn Wagner & Prof. Alastair Corbett
Primary Focus: Clinical Study of Bone Health in FSHD
Type: International and Australian Research Grant collaboration

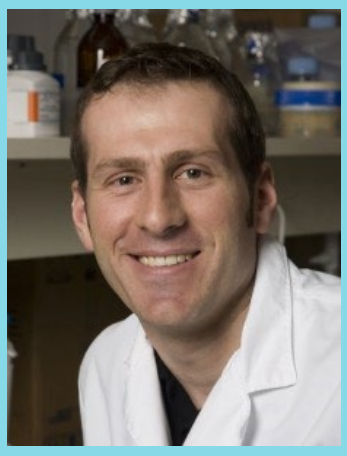
As of February 24, 2016, enrollment has been completed at the Kennedy Krieger Institute. A total of fifty participants with FSHD (27 females and 23 males) have enrolled in this clinical study at the site.

For the USA site, the age of the participants in this cross-sectional study spans from 18 to 83 years old. The allele size of the D4Z4 portion on the 4q35 chromosome in the participants ranges from 11 to 35 kb, confirming the genetic diagnosis of FSHD.

All enrollees have undergone a DEXA (dual-energy x-ray absorptiometry) scan to assess bone mineral density and lean body mass. In addition, they have had blood collected to assess bone biomarkers, and have had muscle strength and timed function testing performed.

Data from the Concord, Australian site were received on January 31, 2016. Data was collected for 52 participants, 3 of whom were confirmed to not have FSHD through gene testing. These 3 participants will be excluded. Data for the remaining 49 participants will be included in this cross-sectional study.

We have started the process of merging and cleaning the two datasets. Once a final merged dataset is available, statistical analyses will be performed to determine the prevalence of osteoporosis, fractures, abnormal bone turnover, and association with the allele size and strength testing. Descriptive statistics and bivariate associations will be explored to summarize the status of bone health among this sample of 99 participants with FSHD.



Grant 25

Research Institution: Baker IDI Heart and Diabetes Institute, Australia
Principle Investigator: Dr Paul Gregorevic
Type: Australian Research
Project title: Enhancing BMP signaling to treat FSHD

Summary:

The Team's first objective has been to develop a new mouse model in which to study how muscles are affected by FSHD, which can be used to test new therapeutic strategies. To achieve this, the Team designed a gene delivery tool that enables expression of the FSHD related gene DUX4 to be controlled in the muscles of mice. Having designed the tunable DUX4 expression system during the initial 6 months of the project, the Team has subsequently focused on defining the conditions required to reproduce intermittent DUX4 expression of varying degrees in the muscles of treated mice. This approach to controlling DUX4 expression is an important feature, as human FSHD muscles express more DUX4 than normal, but intermittently and still at low levels. Using specific conditions, the Team is now undertaking longer-term studies to a) examine how intermittent, low-level increases in DUX4 expression contribute to the development of FSHD-like symptoms, and b) profile changes in the expression of genes as a consequence of DUX4 regulation, to identify cellular processes that could be targeted by new therapeutics. With this information in hand, the Team intends to progress to the next objective of investigating whether manipulating the activity of specific signalling mechanisms of interest is protective or restorative in mouse muscles that model FSHD.

Grant 26

Research Institution: University of Massachusetts Medical School, USA

Principle Investigator: Prof Rossella Tupler

Type: International Research

Project title: Functional study of a novel candidate gene for FSHD

We have recently discovered a novel autosomal recessive form of FSHD, FSHD3, in an Italian family with only two affected sisters born from healthy parents. In autosomal recessive diseases one individual must carry two mutated copies of the same gene to have the FSHD. The genetic defect we found in FSHD3 abolishes a specific function of a protein and correlates with a very severe form of FSHD similar to that observed in rare sporadic cases. The protein we discovered in FSHD3 cooperates with SMCHD1. This is very important because the deficiency of SMCHD1 protein causes FSHD2, a sporadic form of FSHD associated with a mild disease. Interestingly FSHD3 and FSHD2 are clinically very different: FSHD3 display a very severe clinical FSHD, whereas a mild form of disease characterizes FSHD2 patients.

Thus, for the first time, we identify genetic elements that can dissect FSHD pathogenic mechanisms. From a molecular point of view, both proteins participate to the control of the activity of several genes located in various chromosomes, not only on chromosome 4q35, where the FSHD defect resides. It is therefore possible that the mutations in the FSHD3 gene can cause the very severe FSHD3 observed in the two sisters because its deficiency alters the activity of many genes contributing to muscle development and function. On the other hand, SMCHD1 deficiency might influence the activity of fewer genes and cause a milder disease, as observed in FSHD2 cases in comparison with FSHD3.

Overall, our discovery suggests that the basis of the large clinical variability observed in FSHD might lie on the number and type of genes that have an anomalous activity.

To study the effect of the FSHD3 mutation in muscle we have generated a mouse model carrying the mutation in the FSHD3 gene we found in the affected sisters from this new family (Specific Aim 1). We have generated cells with the ability of becoming various types of cells from cells donated by the two FSHD3 sisters and two healthy sibs. These cells are named iPSCs (induced Pluripotent Stem Cells) and we are now in the position of generating skeletal muscle cells in collaboration with Genea Biocells (Specific Aim 2). We have also started identifying genes that are anomalously expressed in muscle cells lacking the FSHD3 protein or SMCHD1 (Specific Aim 3).Our future work will be focused on the identification of the most important ways we should work on to impede the disease appearance and/or progression.

Grant 27

Research Institution: Children's Research Institute, DC Washington, USA
Principle Investigator: Dr. Yi-Wen Chen
Type: International Research
Project Title: Preclinical Studies of Fisetin and VBP15 in FSHD

The main goal of this study is to evaluate two compounds, fisetin and VBP15, as potential treatments of FSHD. After we discovered that both compounds suppressed DUX4 expression in muscle cells from patients, we investigated the effects of these compounds in vivo using an FSHD mouse model. In our studies, we first determined dosages and methods of delivery of these compounds. We have identified an effective dosage and delivery route for VBP15. In addition, our data suggested that VBP15 promoted muscle repair/regeneration activities in the mice. Our data from fisetin studies indicated that the delivery strategy for fisetin needed modification in order for fisetin to reach an effective level in vivo. One of the challenges of using the current mouse model is that DUX4 expression level is low, which makes it difficult to study agents that suppress expression of DUX4. Therefore, we have been searching for a better FSHD mouse model for the proposed studies. Recently, we have identified a new model which expresses DUX4 at higher level and can be adjusted as needed, therefore ideal for studying DUX4 suppression in vivo. Our next steps include developing better in vivo delivery strategies forfisetin; determining long term benefit of VBP15 in vivo; and testing both fisetin and VBP15 in the new FSHD mouse model.

Grant 32

Research Institution: University of Calgary, Canada
Principle Investigator: Dr. Jean Mah in collaboration with Prof. Monique Ryan, Royal Childrens Hospital, Melbourne, Australia
Project Status: Currently underway
Type: Australian and International Collaboration Research

The baseline motor performance of 53 study participants with infantile FSHD havebeen studied and documented.

Consistent with previous reports, age, gender, and the size of D4Z4 repeats are associated with disease severity. Earlier onset of facial weakness in the participants was associated with a greater degree of total muscle weakness. The rate at which motor function changes will be determined by follow-up evaluations.

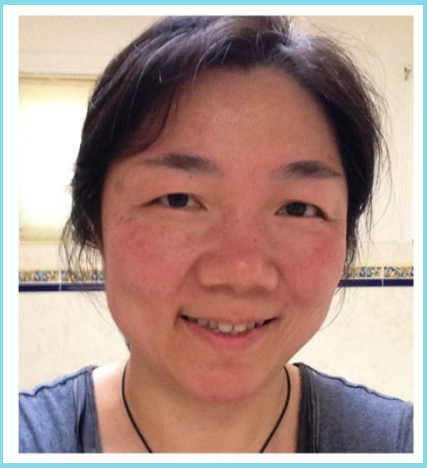
Speech impairment was a very common feature for participants with early onset FSHD. We identified the maximum phonation duration (MPD) as a sensitive tool for identifying speech-related issues in FSHD. We recommend affected individuals to seek professional counseling and to develop strategies to minimize voice strain and fatigue.

Remaining baseline data, including hearing, cognition, and ophthalmologic findings among the infantile FSHD cohort will be reported once further analysis has been undertaken.

Baseline samples from some participants were found to have a higher degree of hypomethylation which resembles the combined effects of Type 1 and Type 2 mutations. Additional samples should help verify this intriguing observation.

The longitudinal study protocol received ethics approval by the University of Calgary and the Children's National Medical Center and the protocol and operational manual have been disseminated to the twelve participating CINRG sites. Some sites are still undertaking negotiations to begin recruitment however, we anticipate having the first patient enrolled by April 2016.

A potential biomarker has been identified that significantly correlates with disease severity. Results were presented at the 2015 FSHD International Research Consortium Meeting in Boston, MA, and a manuscript is being prepared for publication. The mRNA expression profiling study of blood samples collected from affected individuals with early onset FSHD is underway. Baseline data indicated unique molecular profile that can potentially be useful as biomarkers for disease progression. To extend the expression profiling study to all 48 samples collected, we submitted a grant application to the US FSH society in 2015; however it was not funded. In the longitudinal study, we will include healthy controls in the protocol to serve as comparison for the biomarker studies. We would like to allocate $30,000 CND (out of the initial installment of $45,045.45 CND from FSHDGRF) to Dr. Chen's lab to allow for the collection and storage of blood samples for biomarker discovery. Additional funding (estimated at 120K Australian dollars including microarray profiling for 48 FSHD and 10 control samples and data validation) will be needed to continue the expression profiling and biomarker studies in infantile FSHD.



Grant 34

Research Institution: Children's Research Unit, USA
Principle Investigator: Dr Yi Wen-Chen
Project Status: Currently underway
Type: International

Gene-silencing oligonucleotides work by preventing the production of the protein product of gene transcription. This is significant in FSHD because they can be used to stop the production of toxic proteins produced through the lack of inhibition in the D4Z4 region.

Gene silencing oligonucleotides have been studied previously in models of FSHD with some promising results. The oligonucleotides that Professor Chen will be using for this newly funded grant represent a significant advancement on these early oligonucleotides. In addition, Idera Pharmaceuticals have identified four potential oligonucleotides that effectively inhibit DUX4 mRNA transcripts in cells.

The aims of this grant are to further develop these potential therapeutics in human FSHD cells and a mouse model of FSHD.

Our Sponsors

We would like to thank our sponsors who have showed his support throughout the year.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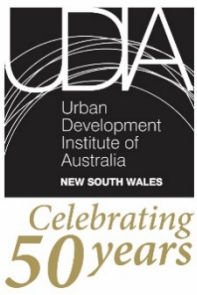
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Justin’s Story

Justin is my big brother,
When we were little we fought all the time, I thought he was bossy & mean, my poor mother.
I didn't really know what it meant that Justin had FSHD, no one seemed to know how it would affect him back when we were 12 & 13. He could still throw a hard punch and run away fast.

Later when I was married with 3 kids and Justin moved to Sydney from Canberra where we grew up, to get to warmer weather, I started to spend more time with him. He was about 28. We had always spent time together growing up, but now he needed me & mum.

He had just stopped driving. Well he hadn't really decided that, but he never drove in Sydney.
It was obvious the muscles in his legs were deteriorating.
He showed the determination and drive that he still has today in those early days in Manly.
He was still walking, but I picked him up on the days that he needed to go grocery shopping or to attend Hydrotherapy. He has lived independently all his life. I usually had 1 or 2 kids with me and Justin is a very favourite Uncle, always joking. Teaching them all the bad words and planning tricks to play on me.

Justin had dreams, he had been to University, travelled overseas, played in a band, recorded music in his own studio, and dealt every day with the slow cruel wasting of his muscles.
Justin had an office chair that he used to scoot around the house from place to place. It gave him the freedom to move without being in the dreaded wheelchair. This he was avoiding at all costs.
We had obtained a manual chair, but Justin would not use it.
Around this time Justin started a counselling diploma at the Catholic University, the biggest challenge was the transport each week for tutorials. Somehow he managed.
He excelled in this course, he wanted to help others less fortunate than himself. He believed

he had a natural gift for communicating and a truckload of empathy with young people. He is an incredibly wise and balanced person.
He started work experience with phone based counselling, this seemed a good option for someone who could not easily get to an office.
He also went and helped out at the MD Association Annual Kids camp. This was a weekend camp for kids with MD 5-14yrs.
Justin would prepare video presentations with subtle messages that appealed to the kids, it was always fun. One year he used Kung Fu Panda. It was a great visual parallel for the Kids that proved anyone can achieve great things in life overcome their doubts and fears.

This is what Justin is about, even now as he fights his body, he is not giving up. He is looking at the positive side of everything. It is a spirit I don't think I have. I admire it, but it also confuses me. I am so sad for where he has spent the last 3 months:
Prince of Wales Hospital, first in the Intensive Care High Dependency unit for 8 weeks and then in the infectious diseases Respiratory Ward. There have been some close calls, 3 near deaths in the first 10 days. And one coma that lasted about 5 hrs.

So I love him very much and respect his will to live. But I watch my mum and family visit and sit next his bed and help bath his eyes and use the suction machine to remove the mucus that is filling up his lungs. He has a feeding tube up his nose because the swallowing muscles have deteriorated so much he has been told he won't eat or drink again. To me it is a living hell, but it isn't my life. I am able to walk out of the hospital, down into the car park and drive to my house and breath in the air and thank someone that I am OK.

It takes its toll on the family though. It's just Mum and me, Dad left when the diagnosis came through, Justin aged 12, me 11. It is a tough time for a boy experiencing puberty, worse when your dad bales out and really has not much to do with you going forward.

He is not much more than a skeleton lying under a sheet, with tubes everywhere and the BPap machine and cough assist standing by. But he is thinking.
He questions everything; the doctors advice, the medication, his time at the gym in rehab. He wants to go home and live independently again. He has something to say about everything that happens in his day, from the nursing staff to the air conditioning vents. He vocal chords are effected too, so he has a raspy breathless voice, but he still has a lot to say.
He has asked to have someone to transcribe his thoughts. He wants to write. The first piece "How to Survive Hospital"

I admire him greatly and will support him whatever he chooses to do. He has always supported me and we are very close. We have spent many many afternoons and nights discussing things that puzzle and intrigue us: the complexities of life, the behaviours of people and what makes life important and meaningful. He always listens and then gives me very simple clear advice that is so wise, I go home feeling better.
Justin is about connection and authenticity. He is an honest man.

Justin’s Story by Gaenor Meakes

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