Viking Genes Newsletter Summer 2026

Viking Genes

Viking Genes Homepage

Welcome to your Summer Newsletter

Welcome to the Summer edition of the Viking Genes newsletter!

We begin with news from Edinburgh Medical School's 300th anniversary, Professor Jim Flett Wilson's Inaugural Lecture in Edinburgh, and a remarkable research focus and lived experience story on neuromyotonia from the island of Flotta.

There is a fascinating story of a founder ancestor in Shetland with haemophilia, and an important multi-generational volunteer story from Shetland that highlights why the Shetland Community Project is necessary.

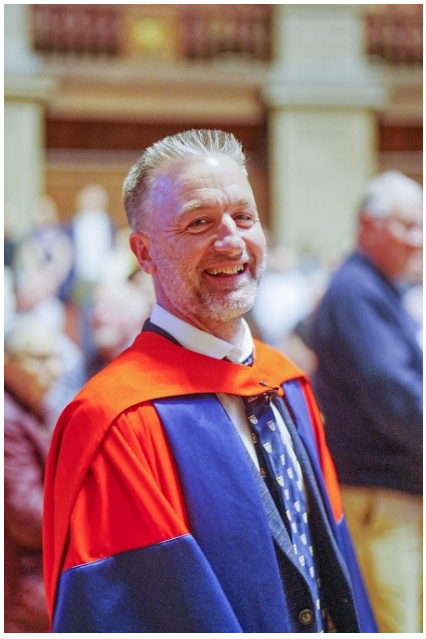
We have a summary of a research paper on the "sunshine vitamin".

To round things off, we bring you an update on the Viking Genes fundraising campaign, and news of three public awareness talks in the Hebrides in June.

Read on to find out more.

Edinburgh Medical School 300 & Prof Jim Flett Wilson's Inaugural Lecture

We are delighted to announce that Professor Jim Flett Wilson has been chosen as one of the 300 faces of Edinburgh Medical School. If you have recently been to Edinburgh, you might have spotted him on a billboard in Princes Street!



The Faculty of Medicine at the University of Edinburgh was founded in 1726, making 2026 its 300th anniversary. Celebrations included the 300 Alumni Weekend which coincided with the College of Medicine and Veterinary Medicine Inaugural Lecture Showcase on Friday 24th April at McEwan Hall in Edinburgh. The double-header featured Professors Kenny Bailie and Jim Flett Wilson as they shared their career and research journey so far to a capacity audience.

Professor Jim Flett Wilson (pictured after his lecture) treated the audience, which included family, friends, esteemed colleagues, alumni and Viking Genes supporters, to a unique mixture of family trees and genetic research. Prof Wilson's academic family tree was quite something!

You can watch footage of Professors Kenny Bailie and Jim Flett Wilson's brilliant inaugural lectures on the College of Medicine & Veterinary Medicine website via the link below.

EMS Alumni Lectures 2026

Flotta Neuromyotonia – The Story So Far

"Flett fae Flotta" (Flett from Flotta) is a well-known 4/4 march composed by Pipe Major Donald MacLeod. The tune, memorable for its simple major-key melody, is named after William Arnot Flett (pictured in 1942), a soldier from Flotta, one of the Orkney Islands.

Flett served with the Seaforth Highlanders, as did MacLeod. According to the story, the tune was composed during a 1940s train journey from Inverness to London while the two were guarding regimental silver. Passing time by playing their practice chanters, they decided to write a tune together; MacLeod then used Flett's name and home island as its title.

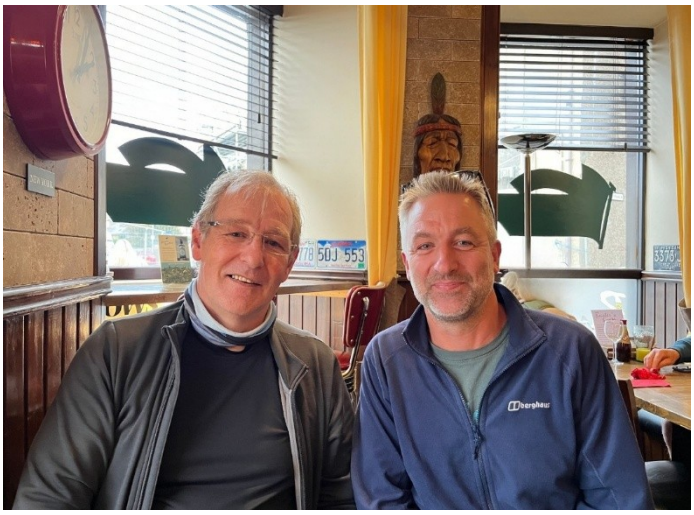
Alternative versions of the tale suggest the march imitates Flett's distinctive walking gait, which may be a tantalising clue as to why Professor Jim Flett Wilson was contacted in 2023 by Stuart and Chloe Flett.

In 2024, the Viking Genes (Wilson Group) began studying the Flett family from Flotta (Orkney) with a suspected inherited muscle-and-nerve condition. The first family member assessed was diagnosed with neuromyotonia, a very rare condition that can cause severe cramps, muscles that are slow to relax after movement, and enlarged muscles. Across at least six generations, relatives have received diagnoses including motor neurone disease, multiple sclerosis and Parkinson's disease, and others have had similar symptoms, often starting in the teenage years. The team suspects these may all be different labels for the same underlying inherited genetic cause.

Sixteen family members were whole-exome sequenced through Viking Genes. A symptoms questionnaire, reviewed by a consultant neurologist, was completed by thirteen relatives. The team used two approaches side by side: (1) looking for rare genetic variants that were present in affected relatives but not in unaffected relatives, and (2) carrying out parametric linkage analysis to pinpoint chromosome regions that affected relatives are likely to share because they inherited them from the same ancestor (identical-by-descent). The variant analysis ruled out known pathogenic coding variants in genes previously associated with neuromyotonia and related neuromuscular conditions. The results suggest that the genetic cause is likely a type of variant that is not captured by standard whole-exome sequencing, so further analysis is required.

The next steps are to raise enough funds so additional family members, some who display many symptoms, and other closely related individuals who have no symptoms (but also contribute to the ability to detect genetic signals), can be whole-genome sequenced. This will focus on the linked regions and look for structural and non-coding variants across the genome. If a causal variant is identified, Viking Genes can screen for its presence among the approximately 4,000 individuals of Orcadian heritage recruited in Viking Genes, potentially identifying additional affected individuals, and contributing to our understanding of neuromuscular disease in this population.

Living with Neuromyotonia by Stuart Flett



My name is Stuart Flett and I'm 60 years of age. I have a condition known as neuromyotonia. It is an extremely rare neuromuscular disorder but thankfully isn't considered to be a neurodegenerative disease or life-shortening. There are thought to be fewer than 200 cases recorded in medical literature, only 10% of which are considered to be hereditary. In my case the condition is thought to be inherited. Clearly there will be many more cases, but as it's so difficult to diagnose, those undiagnosed individuals don't appear in the data.

My background is that both of my paternal grandparents are Orcadian. My granny's family were from Westray and my grandad's family were from Flotta. My dad and his 5 siblings were born in Orkney, but the whole family moved to England. That's where my dad met my mum, who was English. So that makes me 50% Orcadian.

The symptoms of neuromyotonia are as follows; when I contract a muscle, for example by straightening my arm or leg, reaching across my chest to reach for my seat belt, or getting dressed and bending over to put on socks and shoes, the muscles are foreshortened and lock up. The affected muscle doesn't automatically relax and remains activated. If I don't force the muscle to relax by stretching, it builds into a powerful and often painful cramp. Once I manage to get the muscle to relax, I experience a phenomenon known as myokymia. With myokymia, the muscles ripple and undulate which has the outward appearance of having worms crawling under the skin. It is often referred to as 'continuous muscle fibre activity'. All of my muscle groups are affected, from my face to my feet. As a result, movement is challenging, tiring and painful. One of the most alarming features for onlookers is when my throat muscles clamp up; it stops me from talking, sometimes mid-word and I have the outward appearance of choking. I can deal with it by taking shallow controlled breaths until it passes. I also have the propensity to make a kissing shape with my lips and one cheek will lift giving the appearance of winking. It can get me in trouble with the ladies and can misconstrue things somewhat. I also have periods where swallowing is problematic. This is caused by my swallowing muscles going a bit haywire.

Myokymia can also cause physical damage to the muscles, which was indicated in my blood tests by elevated creatine kinase. My kidneys have to clear out the damaged muscle from my system, and consequently I always feel washed out, akin to having a hangover.

An 'upside' to the condition is that my muscles are much more defined and pronounced, caused by the continuous muscle fibre activity. It truly is continuous and persists during sleep. I also experience extreme night cramps and sweats. One of the dangers associated with neuromyotonia is that the unusual muscle behaviour persists during general anaesthesia. There is also a potential risk of a life-threatening reaction to anaesthesia called malignant hyperthermia, so special precautions should be taken when considering surgery. I wear a medical alert talisman in the event that I'm unable to communicate my condition.

Neuromyotonia also means I'm unusually strong, without having to engage in any form of training. My consultant said "Well, you're doing a workout just sitting in your chair". When I was a teenager, I was accused of using anabolic steroids. Folk said that I couldn't achieve that level of muscle size, definition and strength without engaging in weightlifting.

I was first aware of unusual symptoms when I was about 7 years old. I started experiencing myokymia in my face, along with unusual facial muscle spasms. I was diagnosed with Bell's Palsy at the time and underwent intensive facial physiotherapy. After a few weeks, things normalised and I didn't give it any more thought. Could this have been the start of my neuromyotonia symptoms?

As I started to grow, my physique began to develop. At secondary school, I could climb the gym ropes hand over hand without using my legs. I thought that was how you were supposed to do it. It wasn't until I watched the others really struggling, that I realised that I was somewhat different. I was experiencing bad muscle cramps but just tried to shake it off until they became so problematic that I had to consult my doctor. I was around 15 at the time. I wasn't aware at the time that other family members had visited the same doctor with the same symptoms. The doctor had written in my notes "History of MND on father's side. This is ridiculous, what is it with this family?" Perhaps if he'd considered that there was something genetic going on, things would have made more sense.

I was subsequently referred to a university hospital that specialised in neurological conditions. They took muscle samples and subjected me to all kinds of tests and investigations. They had me up on stage in front of a load of medical students, having me parade up and down in my underpants, pointing out my unusual gait and drawing attention to my muscles with a pointy stick. They were using medical terminology that I didn't understand. Upon being discharged I was prescribed with a powerful muscle relaxant and told to come back if things became unmanageable. I just got on with life.

In 1989 my dad, who is also an unusually powerful man, had been attacked at his home by 3 career criminals, intent on robbing him. To cut a long story short, they fled, bruised, bleeding and empty handed. A cousin remarked "Did they no ken that it's no wise tae **** with a Flett?". I was involved in the police investigation and found it very interesting. So, at the age of 24 I joined the police.

I was enjoying an interesting career, managing to mask my symptoms to the best of my ability, then one day I was working with a doctor and he asked, "Do you have myotonia?". I'd never heard the term before. He explained that he'd only seen one case in his career, and that was when he was studying medicine, when they'd presented a patient to the students to point out what to look for. He explained that my gait, the way I held my pen, along with my general physique were exactly the same as the subject they'd observed. He suggested that I ask my own doctor. I did just that, but my doctor stated "Oh, that's really rare, you won't have that" and sent me packing.

One day while at work, I was restraining a rather large body builder and had to use all of my strength. This resulted in my pectoral muscles locking up and I was unable to let go of him. I was experiencing extreme pain in my pectoral muscles. With experiencing extreme 'chest pain', I was sent to the hospital to investigate coronary involvement. Once any coronary involvement had been ruled out, it left them scratching their heads a bit, so they called in a neurologist. There were many steps which I'll skim over,

involving MRI scans and lumbar punctures, but it ultimately resulted in a referral to the university hospital where I'd been seen 25 years earlier.

I actually saw the same consultant, but he was now the professor. He remembered me and explained how they had suspected I had a genetic condition known as myotonia congenita. He explained that back in the 80s genetic testing wasn't available for myotonia congenita, but he could tell just by looking at me that I had this condition. I remembered the police doctor asking me if I had myotonia and being dismissed by my own GP. I felt vindicated at that point. It's an autosomal dominant inherited condition, passed down by one parent, with a 50/50 chance of an offspring inheriting the gene variant. He asked whether I'd be prepared to travel to London to see a particular professor who would be very interested in seeing me and would in turn conduct the genetic test to confirm myotonia congenita. I said yes, and he immediately picked up the phone and called his counterpart in London and said "I have a very interesting gentleman sitting in front of me who has the appearance of Arnold Schwarzenegger. I think he has myotonia congenita, can I send him down to you so you can apply your expertise?"

So that was me off to London. They conducted the gene test for myotonia congenita which to everyone's surprise was negative. I had however piqued their interest, and they enrolled me in a research programme. I was subjected to a bunch of tests and investigations, one of which detected the unique signature of neuromyotonia. It is difficult to differentiate between myotonia congenita and neuromyotonia, so it's quite understandable why they initially misdiagnosed me.

Neuromyotonia is an extremely rare condition which is normally autoimmune and acquired rather than hereditary. I was screened for all the known neuromyotonia antibodies, but the tests were all negative. As there were very few documented cases of the condition being inherited, and to date, the gene variant responsible hasn't been identified, they were unable to conduct a specific gene test to confirm the diagnosis.

I was placed under the care of a consultant who has a special interest in inherited neuromuscular disorders. Since then, they have cast a wide net and searched for variants in hundreds of genes known to cause any kind of neuromuscular disease using NHS 'gene panels' but haven't found anything of significance. The consultant repeated the previous tests which had detected neuromyotonia and on several occasions detected "unequivocal neuromyotonia". So, I definitely had neuromyotonia, but without the recognised autoimmune markers. That was certainly a challenge for the experts. She asked me to compile a family tree and was extremely interested to find that I had Orcadian heritage. Things got remarkably interesting from that point onwards.

My dad, although he was unusually strong, had relatively mild cramps but pointed out that at the age of 12 he had been struck down with extreme body wide muscle cramps and hyperhidrosis which resulted in him being bedridden. He was diagnosed with rheumatic fever at the time, but tests later in life excluded that diagnosis. After over a year of being bedridden, he was nursed back to health by his parents. His dad had seen the same thing happen to one of his siblings at around the same age. She had suffered widespread cramps and sweating which progressed to muscle wasting. Sadly, at the age of 14 she died. At the time, her death was attributed to TB, but could this have been a misdiagnosis like my dad?

A number of paternal aunts, uncles and cousins have experienced unusual neuromuscular symptoms, particularly muscle cramps and unusual strength. Interestingly, my dad's eldest sister wasn't a Flett. She was born before my grandad met my grandmother, so was a half-sister to my dad. None of her children, or descendants have reported any neuromuscular symptoms. This helps the geneticists to establish that the gene variant has passed down through the Flett lineage.

I went back a generation. My grandad from Flotta was struck down by a neuromuscular disorder and ended up being a wheelchair user. In addition to his sister who died young having been bedridden with muscle

problems, two of his sisters had been diagnosed with motor neurone disease, and one sister was bedridden for decades with neuromuscular issues.

Then there's his brother, William Arnot Flett. He emigrated to Australia, so we don't know how he fared in his twilight years, although anecdotally he had Parkinson's disease. He was however quite famous in Orkney, particularly among bagpipe players. He was a giant of a man with herculean strength; his party piece was lifting a plough over his head at his homestead on Flotta. He was a piper and joined up with the Seaforth Highlanders, subsequently going to fight in World War II. My grandad referred to him as "that great big clumsy gowk" due to his propensity to break unbreakable household objects. Interestingly both my daughter and I have the same trait.

After the war, he continued as a piper with the Seaforth Highlanders. There is a famous pipe tune which is a staple for a marching pipe band. Readers will almost certainly have heard the tune. It's called "Flett from Flotta". The tune was composed as a tribute to William Arnot Flett and is written to emulate his unusual gait. Remember that the police doctor had identified myotonia in me by observing my unusual gait? Things were starting to make sense.

I went back another couple of generations and gathered anecdotes. The family history is littered with tales of family members engaged in feats of superhuman strength, most notably my grandad's grandad. He, along with 5 other family members had put to sea in an open boat to rescue the crew of a stricken steamer which had run aground on a neighbouring uninhabited Switha island. He was 60 years old at the time, but led the rescue, having to row several miles in a snowstorm to affect the rescue. The rescuers received a medal, presented in person by The King for their act of bravery and endurance. Again, I've learned that he was a very powerful and unusually strong individual. He lived to the age of 80 but spent the last decade of his life being immobile due to debilitating neuromuscular symptoms. His death was attributed to motor neurone disease. He was born in 1845. I have been unable to establish anything about the health of previous generations, but there have certainly been issues of neuromuscular disorders going as far back as 1845.

Once I'd managed to document things as far as I could, I presented my family history to my neuromuscular consultant. She was very interested, particularly in the cases of MND which had been documented. Neuromyotonia is actually classed a subcategory of MND but wasn't fully documented until the mid-20th century. She pointed out that diagnosis was in its infancy back then, and there is every possibility that those family members who had been diagnosed with MND could possibly have been suffering from neuromyotonia.

By this time, my daughter Chloe, who was in her early teens, began to display symptoms of neuromyotonia, but didn't let on. She didn't want to worry me. Then her boyfriend let it slip one day when he saw me struggling with a particularly debilitating muscle cramp. He said "Oh, that happens to Chloe too". So, the cat was out of the bag so to speak.

Chloe set out with an intention to contribute to the solution and gained the necessary qualifications to gain a place at a Russell Group University to study medical biochemistry. She subsequently gained a PhD with a focus on ovarian cancer and took up a position as a researcher at the very institute in London where I was diagnosed, having switched fields to neuromuscular disease.

During her PhD, keeping up to date with the latest ovarian cancer research, she saw a story on BBC news about the new *BRCA1* variant discovered in Westray by Professor Jim Wilson and the Viking Genes team. After looking further into his work, she decided to reach out to him and offer our genomes to the Viking Genes programme, hinting at our muscle disease afflicting the Fletts from Flotta. It turned out that through the Viking Genes study, Jim had already gathered DNA from many of my Orcadian relatives, so had the

foundation to expand the study into neuromyotonia. He is now collaborating with my neuromuscular professor in London.

We travelled to Orkney to meet with Jim to discuss the way forward. Chloe and Jim were talking in scientific terminology that was alien to me, so I just nodded in what I thought were the appropriate places. We discussed which family members who weren't currently in the study that we could recruit into the research. I felt useful again as that was something I could arrange.

I'm a member of a support group for folk with neuromyotonia, and a common thread is the frustration expressed by group members at the apparent lack of research being conducted into this debilitating disorder. It has been encouraging to reassure group members that there is lot of research ongoing in the background. This has given hope for group members who are struggling to get a diagnosis. As a result, funds amounting to tens of thousands of pounds have been donated by group members to further the Viking Genes study, specifically in relation to inherited neuromyotonia.

It is an exceedingly difficult condition to diagnose, particularly the inherited version of neuromyotonia. Patients often have to undergo years of testing to exclude more sinister varieties of motor neurone disease, and other degenerative conditions. To be able to screen for a gene variant which makes people susceptible to developing neuromyotonia would be a game changer, giving individuals access to treatments much earlier and reducing the anxiety associated with the fear of having a neurodegenerative disease. Furthermore, it would identify their propensity to develop malignant hyperthermia during general anaesthesia.

My family are firmly behind the research being undertaken by Professor Wilson and his team and are extremely grateful for the tireless work which is being undertaken. In 2026, Chloe was formally diagnosed with neuromyotonia.

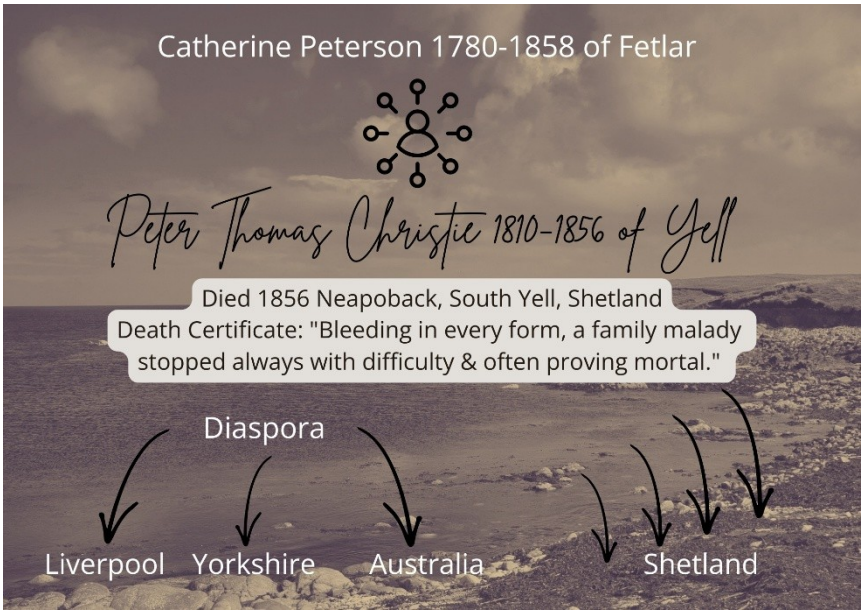
If you would like to support our research into Flotta Myotonia

Please visit our Donate page and select the Viking Genes Orkney Fund

[Donate | VIKING Genes | Viking Genes]

Haemophilia 1856

A genealogist friend of Jim's noticed an unusual entry in a death certificate from Yell in 1856, for a Peter Christie. He died of internal bleeding from the bowels, but the extra note was of particular interest: "Bleeding in every form, a family malady stopped always with difficulty & often proving mortal." This was clearly haemophilia, the inherited disorder where the body's ability to make blood clots is impaired, resulting in bleeding, which was commonly fatal.



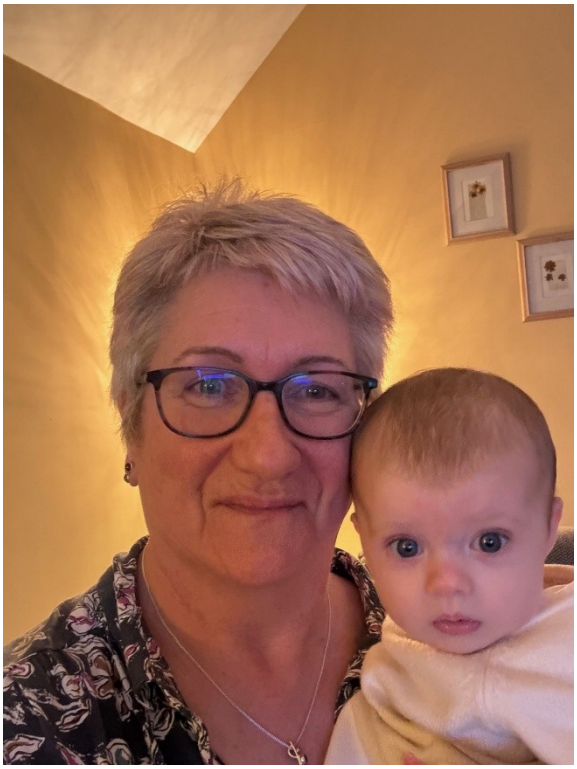
Because of how it is inherited, it is mostly a disease of men. The most famous carriers were various descendants of Queen Victoria, including Tsarevich Alexei Romanov of Russia. The fact it was understood to be a family malady at that time shows that Peter was not the first in his family to suffer from the disease, and he would have inherited the gene variant from his mother Catherine Peterson 1780-1858 of Fetlar.

Jim filed this information away, until early this year, when someone from Yorkshire contacted him describing her Shetland heritage and the fact that her grandmother Millie, who was born in Liverpool, had three haemophiliac sons, one dying aged 29, one in his 60s and one from infected blood, aged 63. Could we shed any light on it? Her grandmother's parents were both born in Shetland and when we looked into it, lo and behold, her grandmother's grandfather was the very same Peter Christie from the 1856 death certificate!

We could see exactly how this has been inherited down the generations. We can also predict that there are likely to be more haemophiliac descendants, as Peter had 6 further daughters, who would all have been carriers. Many of them moved away to Liverpool or Australia. In turn, half of the sons of these seven sisters would be expected to be haemophiliacs, and it will carry on down the generations, just as it did in the Royal families of Europe, where ten individuals were affected over the generations.

If you know of any haemophiliacs with Shetland ancestors, Viking Genes would be interested to hear from you. Please get in touch by emailing viking@ed.ac.uk.

Volunteer Story – Elaine's Story



Elaine has an alteration to a gene called *KCNQ1*, which increases the chance of having a heart condition called Long QT Syndrome. She found out after receiving a letter from Prof Jim Flett Wilson in 2024. Her result led to seven positive family members being found, who needed to be checked by a cardiologist.

Back in 2010 I took part in the original Viking Genes project and was surprised in the beginning of 2024 to get a letter from Professor Wilson saying that they'd gotten funding to look into the health side of volunteer genes. I was asked if I would like to take part and I thought why not. Then, to my surprise I got a letter in April 2024 to say that I may have an alteration to a gene called *KCNQ1* which increases the chance of having a heart condition called Long QT Syndrome. He went on to explain that some may never have any symptoms from it, but it can cause irregular heart rhythms, which can be life-threatening. If it is detected it can be treated with medication, or occasionally surgery, therefore it was a very treatable condition.

It has been a bit upsetting for the family, especially the grandchildren having to take medication at such a young age, but if it helps to protect them through life then it's worth it. My younger daughter was told that young boys are very susceptible to the symptoms. Precautions we all have to take are to avoid sudden shocks, cold water swimming and be wary of certain medications.

“Viking Genes is a life-saving project for sure.”

To read more visit: Elaine's story | VIKING Genes | Viking Genes

How You’re Helping Research – Vitamin D

Vitamin D is often described as the “sunshine vitamin” because our bodies produce it when skin is exposed to sunlight. It plays an important role in bone health and many other aspects of wellbeing, including helping to protect against Multiple Sclerosis. Yet even people who live in the same place, experience similar weather, and lead similar lifestyles can have very different vitamin D levels in their blood. A large genetic study helps to explain why.

Scientists have long known that both genes and environment influence vitamin D levels. Previous genetic studies identified more than 140 genetic variants linked to vitamin D, but these explained only a small part of the differences between people, much less than sunlight exposure. This raised an important question: do genes and sunlight act independently, or do they work together?

A major challenge has been how sunlight exposure is measured. Many studies simply rely on the season when a blood sample is taken as a stand in for ultraviolet B (UVB) exposure. However, UVB levels vary greatly from day to day and place to place due to factors such as latitude, cloud cover, and weather. Grouping all of this variation into broad seasons can hide important biological effects.

To overcome this problem, researchers used historical satellite data to calculate personalised UVB exposure for each participant based on where they lived and the sunlight they experienced in the months before their blood was taken. This produced a much more accurate estimate of real sunlight exposure than had been used before.

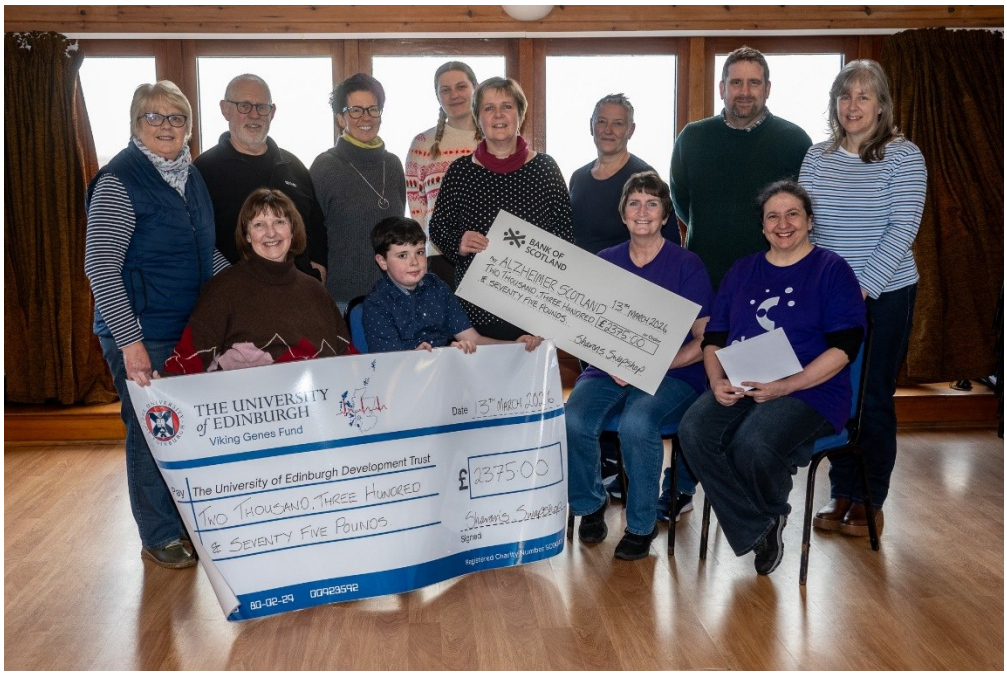
The results showed that genes and sunlight strongly interact. In total, more than 300 genetic variants were linked to vitamin D levels, including 162 that had not been identified previously. Importantly, genetic effects were much more visible in people with higher UVB exposure; rather than cancelling out genetic differences, sunlight revealed them. To check the validity of the findings, the analysis was repeated in the data from ORCADES, which provided replication.

The study also found links between vitamin D, fat and cholesterol metabolism, and genes that control the body’s internal biological clock. Several key signals came from circadian rhythm genes, suggesting vitamin D may be involved in how the body responds to daily and seasonal changes in the environment.

To read more (including link to the paper) visit:

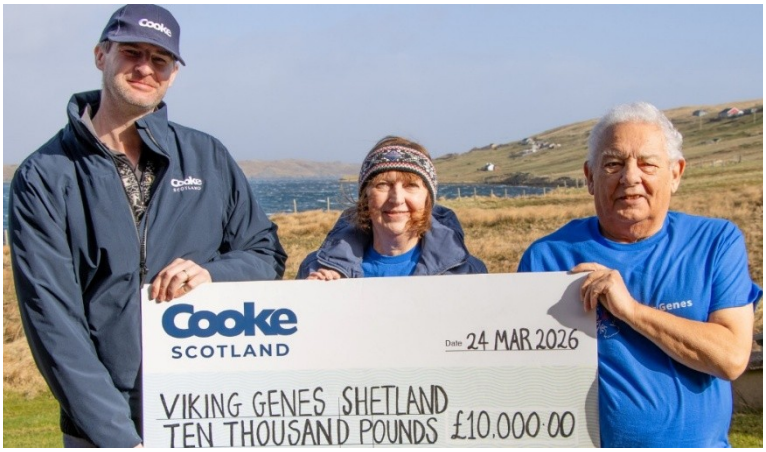
How sunlight, genes and the body clock shape our vitamin D levels

Fundraising Update - £10k from Cooke Scotland & Sharon does it again!



Sharon Deyell's wonderful fundraising continues after the hugely popular Sharon's Swapshop event in January. Viking Genes, Alzheimer Scotland and Clan Cancer Support were each given £2,375 after the long-running event in Aith Hall earlier this year. We can't thank you enough, Sharon!

Cooke Scotland's Community Benefit Fund has awarded the University of Edinburgh's Development Trust (Viking Genes Fund) £10,000 in support of the Viking Genes Shetland Community Screening Project.



Professor Jim Flett Wilson said: "It is fantastic to see this donation from Cooke Scotland, which will help in getting the Viking Genes Fund to the level where we can start to appoint staff."

Sandra Laurenson OBE, chair of Viking Genes Shetland, commented: "Cooke Scotland's commitment to the local community is very welcome. This generous donation will help empower people from across the isles to learn if they are at risk of one of the genetic variants more common in Shetland, improving their healthcare and saving lives."

Katrine Johnson, Unst Office Manager and member of Cooke Scotland's Shetland Community Benefit Fund Committee, said: "We are proud to support the Viking Genes study and its important work in Shetland. As a family company rooted in rural and coastal communities, we're pleased this funding will be directed towards research with lasting impact that has such clear relevance and benefit for the people living in the isles where we call home, and for future generations."

If you can help us to fundraise for our screening projects, let's get talking, we are grateful for all the community help. Find out what materials and resources we can provide to help you fundraise for Viking Genes.

Resources | VIKING Genes | Viking Genes

We hope the resources, provided by Fundraise Your Way, the official fundraising platform of the University of Edinburgh, help you to get started. Please get in touch if you need anything else by emailing viking@ed.ac.uk.

Noticeboard – Talks in the Hebrides

Professor Jim Flett Wilson will be in Barra on Thursday 4th June and South Uist and Benbecula on Friday 5th June with Torcuil Crichton, MP for Na h-Eileanan an Iar (Western Isles). This is part of a series of public awareness talks to share findings from the Viking Genes 'Hebrides Study' and make the case for community screening in the Outer Hebrides.

BARRA

THURSDAY 4th June

19:30-21:00

Castlebay Community Hall

SOUTH UIST

FRIDAY 5th June

12:30-14:00

Cnoc Soilleir, Daliburgh,

Isle of South Uist, HS8 5SS.

BENBECULA

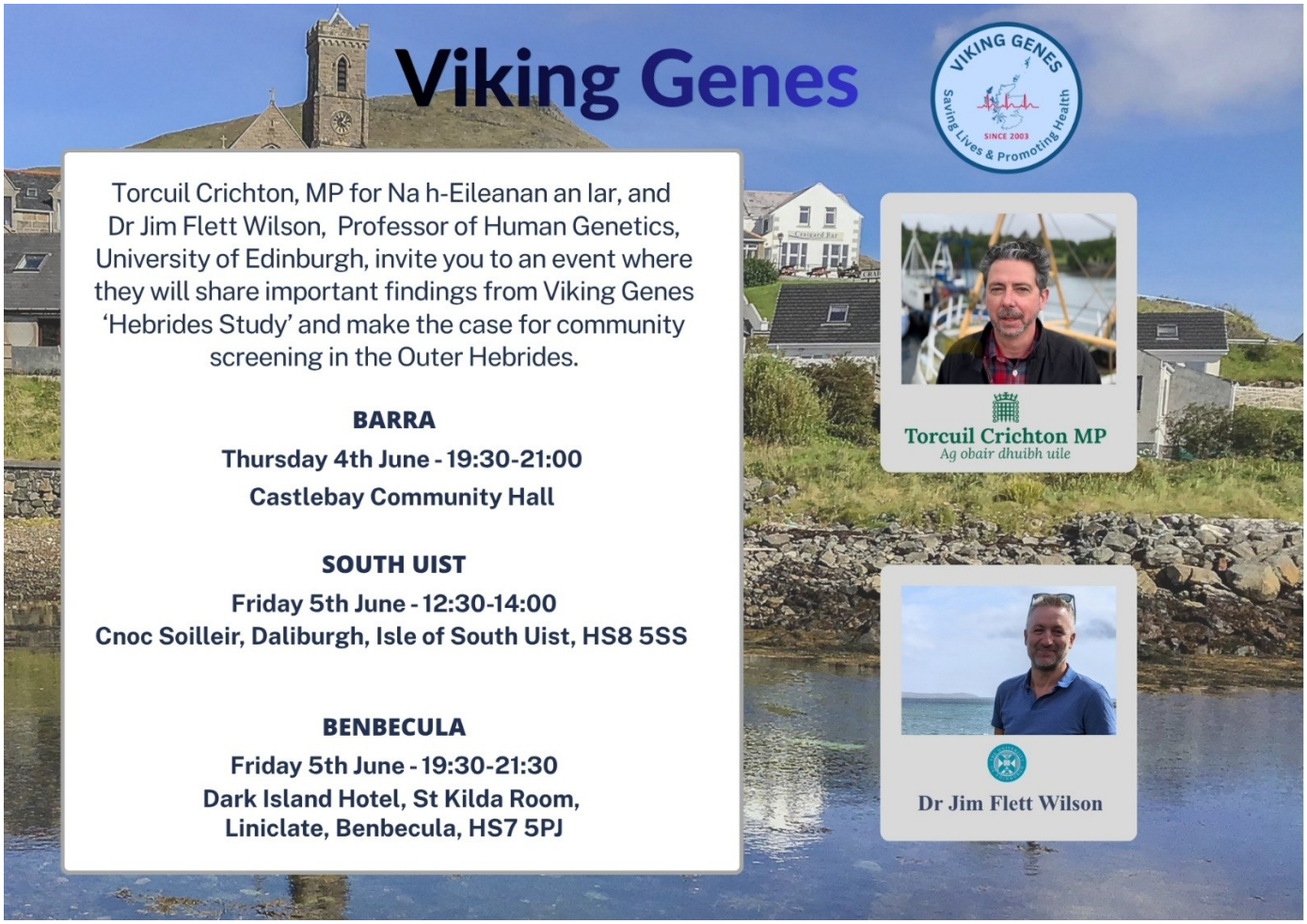
FRIDAY 5th June

19:30-21:30

Dark Island Hotel, St Kilda Room,

Liniclate, Benbecula, HS7 5PJ

If you can attend, keep a look out for our first official Viking Genes badge. While stocks last, pick one up for free and wear it to show that you support our life-saving work.



Viking Genes continues to grow on social media.

We now have over 4,300 followers on Facebook. Thank you!

Follow us on:

Facebook

Instagram

Contact Us

Our research team is based at the University of Edinburgh, in the Usher Institute.

If you ever have any questions, you can email us at viking@ed.ac.uk

Your Data Privacy

We want to make sure you're aware of how we protect your data when conducting our research. For more information about how we use your data and keep it safe, please see our Privacy Policy at www.viking.ed.ac.uk/privacy-notice, or let us know if you'd like to have a copy posted to you.

Craig

Jim

Ben

THE UNIVERSITY of EDINBURGH | usher institute | Viking Genes