MY PULMONARY FIBROSIS JOURNEY

SEAN TULLY

PREFACE

I am not a physician. Nor am I a scientist. Everything below stems from my direct experience as a patient, which I have tried to describe as accurately as possible. Nothing in this essay should in any way be considered advice, nor as recommendations. I am not giving any advice nor making any recommendations. Every person needs to consult their own doctor before adjusting anything they do regarding their own health.

DIAGNOSIS

In July 2021, around the 11th anniversary of my father's death, I stumbled into the same terminal diagnosis my father had been given, which led to his death - Idiopathic Pulmonary Fibrosis (IPF). While the timeline from diagnosis to passing is highly variable, most studies indicate a 3-5 year expected lifespan post diagnosis. My father died 5 years post his diagnosis, in line with those studies. It was a time for reflection.

How did I find out? I had feared I would be diagnosed with IPF for many years. My general physician was excellent and knew my concerns. Then, in July 2021, I happened to have a lower abdominal CAT scan for another indication when he saw the results and didn't like the look of the bottom of my lungs. He said the bottom of my lungs looked "fuzzy". He called me in to talk with him and ordered a chest scan. Eight years earlier, in late 2013 I had an X-ray which showed bi-apical pleural thickening (the outer membrane of both lungs appeared to be thickening). In early 2014 I went to see my father's pulmonologist. I didn't know that a study had recently been completed, called the Panther Study, which showed that the standard of care for IPF patients, which my father had been on, was shown to likely be accelerating death. My Dad's pulmonologist didn't look happy to see me. He put an oximeter on my finger and asked me to walk up and down a set of stairs in a stairwell near his office. My blood oxygen level didn't change much. He said I was fine and not to

worry about it. No CAT scan, no pulmonary function tests, no follow up. My interpretation has always been that even if he determined that I had early- stage disease at that time, there was nothing he could do, so maybe he felt there was no point in investigating further.

After my GP diagnosed me in 2021, it was time to see a pulmonologist. I eventually saw four. It took months. I filled out several questionnaires. No, I wasn't a smoker and never was. No, I didn't own or live with any birds. No, my employment didn't expose me to any physical environmental issues of any concern, although my career in finance was intense, stressful, required frequent business travel and I was certainly sleep deprived. Yes, I had a family history of IPF. I saw pulmonologists at three different hospitals in quick succession, all with the same results. My CAT scan showed fibrosis, my lung diffusing capacity of carbon dioxide was well below the normal range and a faint crackle could be heard in my lungs via stethoscope, all indicating I had IPF. A dear friend, doctor and researcher sequenced my DNA looking for a genetic marker known for the disease. There was none.

My Dad, Michael J Tully Sr. Diagnosed with IPF in 2005, passed from IPF 2010.

A TIME FOR REFLECTION AND DECISIONS

It was time for action. I called my brothers and let them know. I knew by then that IPF was highly familial and was concerned they might be ill as well and might not even know it. They didn't like the news, or my suggestion that they should see pulmonologists. Who would? They then both went to see a pulmonologist months later (it can take months to get an appointment). CAT scans and pulmonary function tests indicated my younger brother Tom had IPF as I did, and my older brother was diagnosed with Interstitial Lung Abnormalities, potentially a very early stage of the same disease. Tom was unfortunately much further advanced than I was. A little over a year later he entered the transplant list at Columbia, and in September 2023 with the hard work of the incredibly talented and meticulous team at Columbia, he had a single lung transplant in order to remain among us. While Tom's transplant has been a very rough roller coaster ride, with multiple surgeries and weeks in hospital after his initial three-week hospital stay for the transplant, today he is doing well. Fingers crossed.

At my time of my diagnosis, I had a stressful, 10-12 hour a day job as an executive at a publicly traded company, I was overweight, didn't exercise regularly and was sleep deprived. I traveled about three times a month, usually across time zones. I was devoted to and loved my job, but

believing I likely had less than five years to live, it was time to reflect and to change my direction. Finding out that I would likely die from suffocation, as my father did, was life changing. It was time to focus on my health. Might I be able to extend my life further? There were two drugs approved for IPF but they were difficult to tolerate, caused enormous GI issues, and they didn't slow the disease down much. As well, while much had been learned since my father died, there was still very little understanding of what might be causing the disease and how to stop it.

My brother Tom Tully. Diagnosed December '21, received lung transplant September '23.

In very early 2022 I approached my boss and let him know it was time for me to retire. I didn't share that I had been diagnosed with a terminal illness. He asked me to stay on another 15 months or so in order to complete a major transition, the move of the US interest rate derivatives marketplace from LIBOR to SOFR and the global US dollar lending market to Term SOFR. Within finance space, interest rate, banking and Federal Reserve space this was a very big deal. At that time most US dollar corporate lending and US floating rate mortgages were based on US dollar LIBOR and it was going away. I was proud of my team at CME Group and proud of the work we did. I agreed to stay until the job was done. Today over $9 trillion in US dollar debt references CME Term SOFR, an index my team and I created and launched during that time. In early 2022 very little, if any, US debt yet referenced our CME term SOFR.

I decided not to go public with my terminal diagnosis. I didn't want to be "the dead guy walking", I wanted to continue to be an effective executive. I told just two people at work, who I knew would find my sudden and frequent doctor visits odd, and would need an explanation for the inevitable mood challenges I would face. In addition, I started on **OFEV** while still working and that brought significant GI challenges for at least the first 6 months, especially when I traveled to London, Singapore and Tokyo. The two people I shared my health situation with at work were a huge support- I'm not sure what others thought. Some suspected I was very ill, and they were right! I wasn't ready to "come out".

A CALL TO ACTION

While working hard at my job, I also committed to doing everything possible to understand the disease, to support research towards an effective therapy and to extend my lifespan without a transplant. In doing so, I also wanted to help my other family members that might someday be diagnosed as I was, and to help all patients with this devastating disease. I committed to use my brain, my body (to experiment on) and my retirement funds.

I saw four pulmonologists, as I said earlier. I began reading everything I could about IPF and googling and searching for more information. In 2022, being interested in learning more about the disease and in helping to fund research, I reached out to the Pulmonary Fibrosis Foundation and asked how I could work with them to help drive research towards a cure forward. They agreed to create a Tully family familial pulmonary fibrosis grant award and to send out a request for proposals which would be reviewed by the PFF scientific advisory board for the most promising proposal. The selected proposal would receive $250,000 over the next two years to support the research project. We received a number of great proposals and the scientific advisory committee struggled to select the best one from the top two contenders. On the spot I agreed to double my donation and to fund both of them.

I began attending medical conferences, starting with the IPF summit in Boston in August 2023 and the Pulmonary Fibrosis Foundation (PFF) Summit in November 2023 in Orlando Florida. At the Boston IPF Summit in 2023 I was very lucky to meet many doctors and scientists, and, especially the Three-Lakes team. Three Lakes had been doing an enormous amount supporting research and I very much had wanted to meet them. That's where I met Cheryl Nickerson-Nutter, a former Pfizer scientist and today the Executive Director of Three Lakes 12-20. At the PFF Summit I also met another patient, Michael Breslin. Mike introduced himself as he saw I was asking a lot of what he thought were good questions. We became close friends quickly. From November of 2023 till May 0f 2025, Cheryl, Mike and I (we called ourselves "the three amigos") went across the country together to a number of conferences and we met every Tuesday virtually, sharing what we had learned that week, how we were doing and making plans to support research. Devastatingly, in June 2025 Mike passed from pulmonary fibrosis.

From right to left – Cheryl Nutter, Mike Breslin and myself, dinner after ATS May 2024.

I read everything I could get my hands on that was scientifically based, have now attended about a dozen medical conferences on IPF to try to better understand what is known and learned everything I could about how I might change my behaviors in order to slow disease progression.

In 2021 my dear friend Dr Richard Smith from the Carver Medical school at the University of Iowa sequenced my entire exome (DNA sequencing). He didn't find any of the known genetic markers for disease. Richard sent the data to my pulmonologist, Dr Christine Garcia at Columbia, who confirmed her lab didn't see any markers in my data either. Dr Garcia also sent my brothers and my blood samples to John's Hopkins for telomere length testing. Extremely short telomeres, telomeres in the shortest 10% of their age cohort, are common in IPF patients. The telomere testing showed my brother Tom and I had extremely short telomeres, my brother's even shorter than mine, and Dr Garcia thought this was important. Might our short telomeres be driving the DNA damage response, cellular senescence, increased complement activation, inflammation, be harming alveolar cell regeneration, depleting sirtuin and NAD+ supplies, and further damaging mitochondria and driving our disease?

I certainly didn't know and still don't know, but I have hunches.

In early 2023, through Dr Garcia at Columbia, I was also given the chance to join a phase 3 clinical trial for a new IPF drug being developed by Boehringer Ingelheim. I jumped at the chance and joined that trial. I am grateful to have been given the opportunity and I've been in that trial ever since.

CHANGING TO A HEALTHIER LIFESTYLE, HOPING TO MAINTAIN MY TELOMERES

Around the time of the PFF Summit in Orlando, November 2023, I read "The Telomere Effect" by Elizabeth Blackburn Ph.D. and Elissa Epel, Ph.D. Elizabeth Blackburn received the Nobel Prize for her work on telomeres and she collaborated with Elissa Epel on much of the research. In the book they cite many scientific studies that describe new behaviors that have shown promise for maintaining telomere length. I then made it my goal to adopt as many of these habits as possible immediately. Here are new habits I adopted:

Meditation- I had previously taken a mindfulness-based stress reduction certification course based on Jon Kabat Zinn's teachings but was not as disciplined in practicing everyday as I should have been. I then later also took a Transcendental Meditation course in order to continue my training. Meditation is a relaxation technique that has shown to reduce cortisol levels, and studies have shown high stress was harmful to telomere length. So, I doubled down on meditation aiming to meditate for 20 minutes two times daily.

Sleep - During my career in finance I was sleep deprived, getting up for many years at 5:30 AM to get the 6:07 train to the office, and often not getting home till 8:00 or 9:00 PM not yet having eaten dinner. I regularly took red-eye flight to Europe. I averaged less than 6 hours of sleep a night. By mid-summer 2023 I had retired and committed to getting at least 8 hours of sleep every night.

Exercise - While I rode my bike regularly in good weather on weekends during my career, often riding 40-60 miles a weekend, I did nothing during the week. I committed to doing 30 minutes of cardio at least 5 days a week and to doing some weight training. Today I have a trainer and try to do at least 3 one-hour sessions of weight training every week in addition to 5 days of cardio

per week. Studies have shown that at least 150 minutes of cardio per week helps to maintain telomere length.

BMI, belly fat and weight loss - When I was diagnosed my BMI was 28.6. I've lost 25 pounds since and my BMI is down to 25. Between counting calories and exercising I've reduced my belly fat, although it is still a work in progress. According to the "Telomere Effect" the greater the belly fat the greater the risk of telomere shortening. I reduced my belly fat and need to reduce it further.

Alcohol - I enjoy a good glass of wine and was consuming 1 to 2 glasses of wine per night. Now I try to keep it to 1- 2 glasses per week.

Red meat - I used to have 5 or more servings of red meat per week. Now it's down to 1 or 2 servings per week. I also generally avoid high fat foods like bacon and sausage, although I indulge very occasionally. Today I eat more fish, chicken and vegetable based proteins. For snacks I try to lean towards fresh fruit, edamame, chick pea, lentil, nut and dried fruit snacks in place of potato chips, corn chips and pretzels.

Fish oil and Omega 3 - I started supplementing with fish oil/ Omega3 every day.

Vitamins - I started taking a daily 50+ multivitamin everyday plus vitamin C and vitamin D.

Green Tea - For an afternoon pick me up I now reach for a matcha as often as I reach for a coffee. Both coffee and green tea have strong antioxidants but there is reason to think green tea, and particularly EGCG, which comes from green tea, may be helpful for IPF patients.

Supporting more research, attending more conferences, listening to webinars, asking MDs and PhDs more questions and adding more supplements.

As I mentioned earlier, since my diagnosis, I've attended a dozen conferences and had the pleasure of meeting with and asking dozens questions to many incredible doctors, researchers, biotechs and pharma companies. We are all united in our goal to help patients and to accelerate the development of better therapies. I am grateful for everything that they do, and for helping to educate me.

In addition to the "The Telomere Effect", I have read other books that have inspired me. One of these was "Lifespan" by David Sinclair. Pulmonary fibrosis often occurs late in life and appears in many cases to be a disease of aging, and David Sinclair, while sometimes a controversial figure, is one of the most respected authorities on aging. I've read the book multiple times. Among its many valuable lessons is that DNA damage, which can be caused by short telomeres can also exacerbate the depletion of NAD+, which drives the operation of mitochondria and fuels Sirtuins, which can play a crucial role in the DNA damage response. Another great book is "Mitochondria and the future of medicine". There are a number of theories of aging, including the information / DNA damage theory and the free radical / mitochondrial theory. There is evidence to support both of these theories in the development of IPF.

In early 2024 I listened to a webinar hosted by PF Warriors, a pulmonary fibrosis patient advocate organization. The webinar was led by Dr Paul Noble of Cedars Sinai in Los Angeles. Dr

Noble was presenting the results from some new research which he and his team had performed to an audience of patients. The results showed that in pre-clinical studies using aged mouse models and human patient tissue, there is a depletion of Alveolar type 1 and type 2 cells. Zinc plus Nicotinamide Riboside (NR) seemed to restore Sirtuin 1 activity and AT2 cell progenitor function in both models. NR is a precursor of NAD+, which fuels Sirtuin 1. Sirtuin 1 and NAD have both been shown to be depleted in IPF patients. Short telomeres can drive the DNA damage response, reducing the available NAD+ in aging people and particularly in IPF patients. These insufficient NAD+ levels can lead to mitochondrial damage. It's known that the mitochondria in alveolar cells of IPF patients were generally very damaged. So, zinc plus NR might help restore alveolar type II mitochondria, might restore alveolar type II cell progenitor function, might help restore normal alveolar repair and might improve lung function.

To learn more about this work, Mike Breslin, Cheryl and I spoke with Dr Noble. Cheryl Nickerson-Nutter, former Executive Vice President of Research and Development at Three Lakes Foundation, helped to set up the call. The Three Lakes Foundation developed many great connections and resources and funded more than $20 million of IPF research. The new foundation, Three Lakes 12-20 will continue this work to accelerate therapies for pulmonary fibrosis.

Speaking with Dr Noble, and asking simple questions, I became convinced that supporting his idea of a human trial using zinc and Tru-Niagens Nicotinamide Riboside (NR) was a great project to support. In our very first conversation I asked how much more money they needed to run a human trial. They indicated $150,000 was still needed for the planned phase 1 trial. I immediately offered to fill the gap with my own funds so that he and Dr Tanzira Zaman at Cedars Sinai could immediately get started with their trial plans.

Being a participant in the Boehringer Trial at that time, and based on the recommendation of Dr Paul Nobel, I asked the trial coordinator if I could take these over-the-counter supplements: Zinc, Nicotinamide Riboside (NR), Resveratrol (RES) and Quercetin (QUE), without interfering with the trial results. The trial coordinator checked and let me know that it was fine. These are safe and commonly used over the counter supplements. I waited until after my final 12-month trial measurement visit, as I understood it, the official last data collection the FDA would use for the trial, before starting on these new over the counter supplements.

In late April 2024 based on Dr Nobles research I added the following to my daily pill taking.

- Tru-Niagen Nicotinamide Riboside (500 mg) twice daily
- Zinc picolinate (30 mg) once daily
- Resveratrol (500 mg) - twice daily
- Quercetin (250 mg) - two, twice daily

In addition to Dr Noble's recommendation, there are many scientific, pre-clinical studies which indicate that all of these supplements may be very promising for patients.

Alveolar type II (ATII) cells have been shown to be massively depleted in pulmonary fibrosis. ATII cells are the workhorse of the alveoli, producing and secreting surfactant that is vital for preventing alveolar collapse, regulating fluid in the alveoli, contributing to innate immune response and acting as the key cell alveolar repair. ATII cells act as progenitor, or stem cell for

both ATII cells and alveolar type I cells (ATI). ATI cells are responsible for the exchange of oxygen from the inhaled breath to the blood, and for carbon dioxide from the blood to the outgoing breath. IPF patients have depleted ATI and ATII cells, their ATII cells no longer repair the alveoli normally, and they stop acting as progenitor cells. ATII cells of patients have been shown to have highly dysfunctional mitochondria, short telomeres, depleted NAD+, depleted sirtuin1and to be senescent.

All of the following reasons have been found in studies for ATII cells no longer being healthy in pulmonary fibrosis patients: upregulated CD38 (an NAD hydrolase, which depletes NAD+), short telomeres, dysfunctional mitochondria, DNA damage, senescence, NAD+ depletion, Sirtuin 1 depletion, excess unfolded proteins, the integrated stress response, etc.

Idiopathic Pulmonary Fibrosis: Let's Keep the Focus on the A(ge)TII cell
https://www.atsjournals.org/doi/10.1164/rccm.202204-0703ED

Studies have also linked NAD+ depletion to falling Sirtuin 1 levels, an important epigenetic actor, which aids with DNA damage response, mitochondrial function, telomere length maintenance and ATII cell progenitor function. Further, short telomeres have been shown to suppress sirtuin levels, which reduces their ability to maintain telomeres, further reducing telomere length in a vicious cycle.

Telomere Dysfunction Induces Sirtuin Repression that Drives Telomere-Dependent Disease
https://pubmed.ncbi.nlm.nih.gov/30930169/

The role and regulation of SIRT1 in pulmonary fibrosis
https://pubmed.ncbi.nlm.nih.gov/38393490/

Therefore, there may be promise in increasing NAD+ levels and activating Sirtuin 1 in patient cells, in order to restore, or at least better maintain telomere length, improve mitochondrial function and restore ATII cell progenitor function and health.

In fact, Dr Noble's lab has shown through mouse experiments and patient organoid cells that zinc plus NR supplementation do exactly that.

The ZIP8/SIRT1 axis regulates alveolar progenitor cell renewal in aging and idiopathic pulmonary fibrosis - PubMed
https://pubmed.ncbi.nlm.nih.gov/35389887/

How do Resveratrol and Quercetin add value? Both have been shown to improve mitochondrial function, to activate sirtuin 1 and in numerous pre-clinical studies to be potentially promising for patients.

Potential of resveratrol in the treatment of interstitial lung disease - PMC

Protective effects and mechanism of resveratrol in animal models of pulmonary fibrosis: a preclinical systematic review and meta-analysis

https://www.frontiersin.org/journals/pharmacology/articles/10.3389/fphar.2025.1666698/full

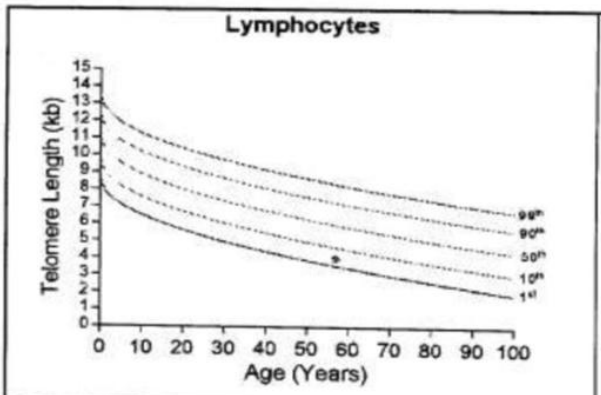
Use quercetin for pulmonary fibrosis: a preclinical systematic review and meta-analysis
https://pubmed.ncbi.nlm.nih.gov/40038212/https://pubmed.ncbi.nlm.nih.gov/40038212/

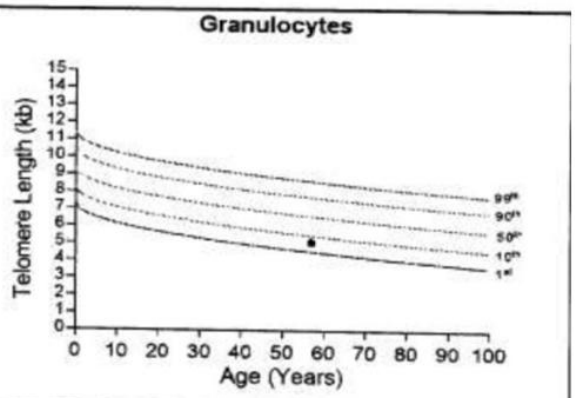
Quercetin in Idiopathic Pulmonary Fibrosis: Another Brick in the Senolytic Wall - PMC

Post March of 2025, in an open label extension, I started taking a higher dose of the IPF trial drug. I had also read research indicating NR was safe up to 3,000 mg per day, Resveratrol was safe up to 1,500 mg per day, zinc up to 50 mgs per day and Quercetin up to 1,000 mgs per day. Having experienced positive results thus far, I increased my supplement dosages in May 2025.

My hope in sharing some of the things I've been doing since my diagnosis is that they might be helpful to other patients suffering from this devastating disease. I also hope that in using my own body as an experiment through time, that my results might help doctors, researchers, biotechs and big pharma. As I said in the preface, nothing here should be construed as advice, I am not a physician. All patients should consult with their physicians. Before I took the initial supplements I consulted with my physician and the trial coordinator for the trial I was participating in and got their OK.

Here are my results. First, it appears my telomere lengths, particularly of my lymphocytes and granulocytes may have increased in length or at least did not shorten further. Below you'll find my telomere length reports from John's Hopkins from 2021 and then from 2025. Assuming the assays are accurate it looks like there may have been some lengthening over the past 4 years which is highly encouraging since a decline would have been expected. I do not know the standard error of these tests so the results need to be taken "with a grain of salt".





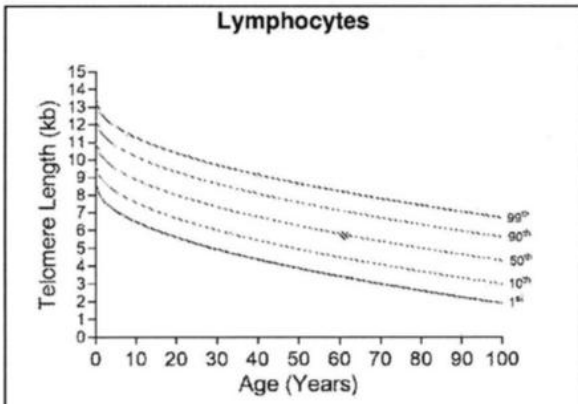
Telomere Length (kb):	4	**Telomere Length (kb):**	5.1
Number of events:	10084	**Number of events:**	2986
Median for Age (kb):	5.95	**Median for Age (kb):**	6.57
Delta Telomere Length (kb):	-1.95	**Delta Telomere Length (kb):**	-1.47

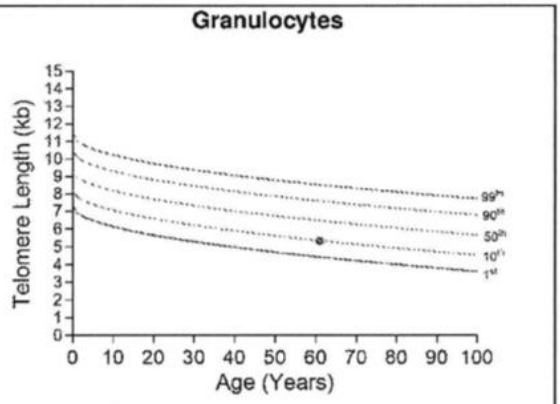
Interpretation

Telomere length falls between the 1st and 10th percentiles for age in both lymphocytes and granulocytes.

Telomere length measured at Johns Hopkins in September, 2021

Telomere length measured at Johns Hopkins in March, 2025



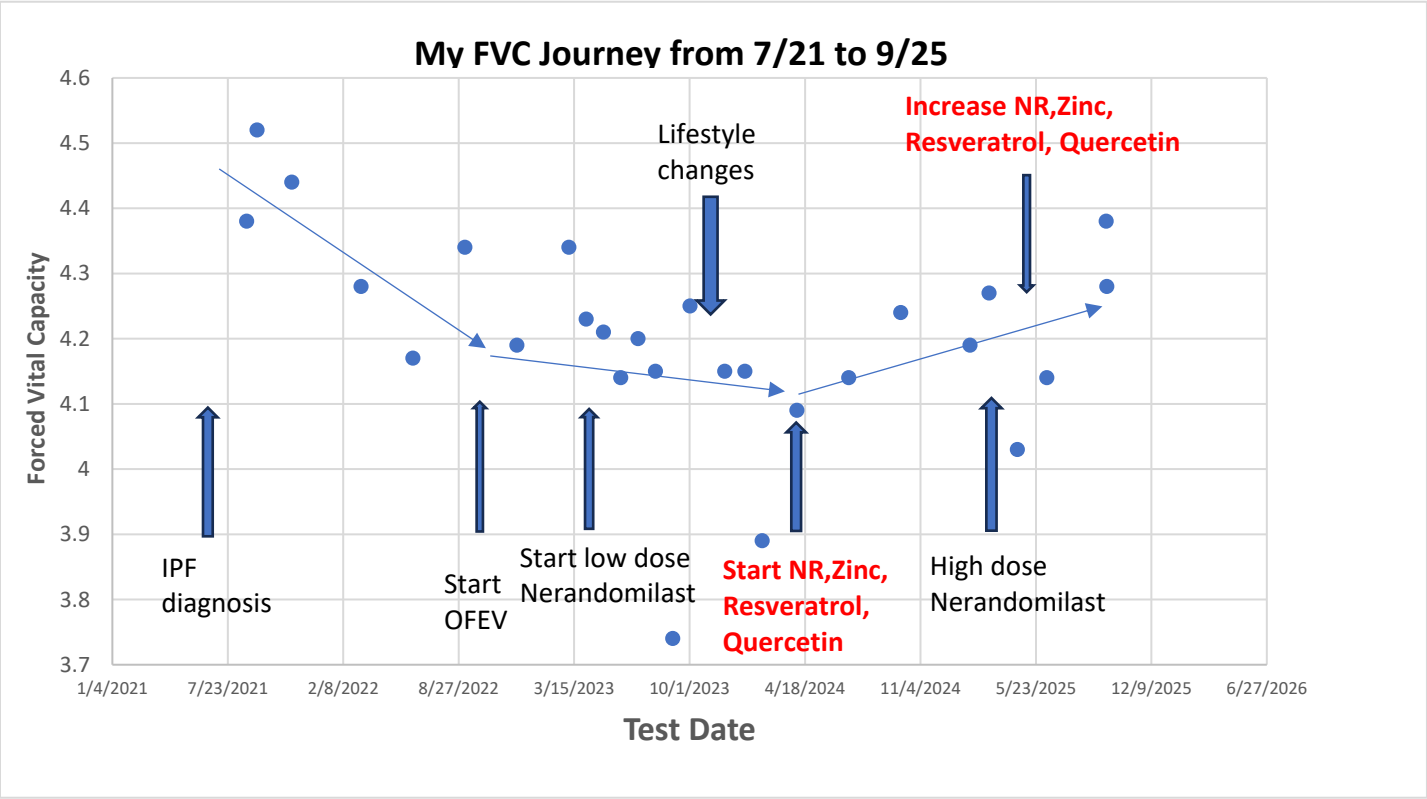


Telomere Length (kb):	5.71	**Telomere Length (kb):**	5.33
Number of events:	5267	**Number of events:**	1677
Median for Age (kb):	5.78	**Median for Age (kb):**	6.48
Delta Telomere Length (kb):	-0.07	**Delta Telomere Length (kb):**	-1.15

Interpretation

Lymphocyte telomere length falls near the 50th percentile.
Granulocyte telomere length falls near the 10th percentile.

Below is a chart of all of the forced vital capacity (FVC) tests I've done since I was diagnosed in 2021. FVC is considered a "gold standard" in measuring disease progression in pulmonary fibrosis and is the measure the FDA prefers for IPF drug endpoints to determine efficacy.



It appears my FVCs were declining rapidly from my time of diagnosis in 2021 until late in 2022. The decline appeared to lessen after I started taking OFEV. It makes sense that OFEV and Nerandomilast might act like tapping a brake, slowing further fibrotic scarring. It's only been 17 months since I started taking the supplements in April 2024 but my FVC readings appear to be improving, despite the variability. I have had a CAT scan done annually every year since I was diagnosed. My CAT scan from early 2025 was the first where the commentary coming back from the radiologist indicated no further scaring. This was the first CAT scan that I have ever had done that did not indicate increased scarring in the commentary.

What happens to my FVC next? I have no idea. I'm excited for the Cedars Sinai trial on Zinc and NR to get started. I'm hopeful the FDA will approve Nerandomilast and I'm grateful to be a participant. There also appears to be evidence that all the actions I've been taking, including exercise, taking anti-fibrotics and taking the supplements recommended by Dr Noble may be helping to improve my health. I'm not a physician and as I said earlier, none of what I have provided here should be considered a recommendation. Each patient needs to discuss anything they might do with their own physician. I am very excited about the trials myself and the Three Lakes 12-20 team are sponsoring. These include Zinc and NR at Cedars Sinai, the Thymidine trial launched in collaboration with Team Telomere at Boston Children's Hospital, the EGCG trial, and the Precisions trial. I am also encouraged by all of the other clinical trials and novel research being conducted in the labs around the world!

My hope is that we can get more support for research, more research done, better understand this devastating disease, and take more shots on goal. My vision is that in 10 years from now, rather than pulmonologists grieving every time they diagnose a new patient, they will look confidently in the eye of the patient, knowing how they will treat them, and confident that the treatments available to those patients will prevent them from dying from this disease.

Thank you!