CML news.

For people with CML & their families

Leukaemia Foundation
VISION TO CURE
MISSION TO CARE

ISSN 2208-5653

April 2018 | leukaemia.org.au | 1800 620 420

Lisa McNeil, left, with husband, Jaimie, and their children, Dallas and Madelene, at Disneyland during a trip to the U.S.

Contents

Asciminib – best treatment Lisa's had in 16 years

Lisa McNeil is happy with the delayed decision she made in 2015 to go on a Phase I trial for a new CML drug – asciminib.

"It has turned out to be a really good thing to try and I'm happy I did it," said Lisa.

"I can't pinpoint any side-effects it's giving me. It's the best drug of everything I've tried."

And Lisa has "tried nearly everything" since her diagnosis 16 years ago, just after her 30th birthday.

She was working in her husband's newly established business and raising their two young children; both have albinism and are visually impaired.

"They were tough babies who never slept well and I was constantly exhausted," said Lisa.

She thought she had chronic fatigue syndrome, then a blood test revealed a high white cell count. After a bone marrow biopsy just before Christmas 2001, she found out she had CML on December 29.

"I was sure the biopsy would be negative and was obviously upset at the diagnosis," said Lisa, now 46, of Adelaide.

Imatinib (Glivec) was being trialled at that time and wasn't widely accessible, so she began treatment with hydroxyurea briefly before going on to interferon.

That's when Lisa began to get nerve pain, which has dominated her life ever since. It took many years to be diagnosed as severe neuropathic pain. She's tried nearly every narcotic and pain is the main reason she has changed from one CML drug to another.

Lisa endured "terrible side-effects" from interferon for 12 months until "the new drug" [imatinib] became available. She was on imatinib for two years before, again due to side-effects, she moved on to dasatinib (Sprycel®) which she was on "for a long time".

"It took 10 years before I eventually got to zero [CML undetectable in her bone marrow]," said Lisa, and she was able to have a break from treatment after having a 0.00 result for two years running.

"I was experiencing such severe pain I wanted to come off my drugs to see if it made any difference, which it did," she said.

"He allowed me to do that, to try and understand what was happening with the pain. Even now, it's a big mystery.

"My doctor put me on a stop trial. I'd never stop my drugs otherwise. I only did it with his consent," said Lisa, who takes compliance to her CML treatment very seriously.

"But after six weeks, my CML levels got really high, so I had to start CML treatment again."

That's when Lisa was told about the asciminib trial, but she opted to try nilotinib (Tasigna®) instead.

Continued on page 4.

TFR on nilotinib as second-line therapy

An Australian Phase II trial has found most CML patients with a deep molecular response (DMR) to second-line therapy on nilotinib can achieve long-term treatment-free remission (TFR).

In another finding, researchers at the South Australian Health and Medical Research Institute (SAHMRI) also concluded that for patients who do not achieve sustained DMR* with imatinib, switching to nilotinib might enable more of them to become eligible for TFR.

The study comprised 163 people with chronic phase CML who had achieved a sustained DMR after switching from imatinib to nilotinib, and 126 of them (77%) were deemed eligible to stop nilotinib treatment.

At 48 weeks after stopping their tyrosine kinase inhibitor treatment, 58% of them maintained a treatment-free response, and 53% maintained it at 96 weeks.

Of the 56 patients who restarted nilotinib therapy, 55 of them regained a major molecular response and 52 regained MR4.5. None of these patients had CML progression to accelerated phase or blast crisis.

The study findings were published in *Annals of Internal Medicine*.

"We found that over 50% of patients have maintained treatment-free remission after two years of observation, suggesting that this is a reasonable option for patients who have received second-line nilotinib," said Professor Timothy Hughes, Consultant Haematologist at the Royal Adelaide Hospital and SAHMRI's Cancer Theme Leader.

"Other studies have suggested that patients who switched from imatinib to a more potent kinase inhibitor had a much lower probability of achieving treatment-free remission if the reason for switching was poor response, compared to patients who switched for poor tolerance. We didn't observe this difference in this study.

"Importantly, we also documented that over 90% of patients who needed to restart their nilotinib therapy achieved a deep molecular response within 12 months of restarting."

The researchers stressed the importance of careful patient selection to ensure the safety of treatment discontinuation.

** In CML, deep molecular response is defined as a reduction in the amount of circulating leukaemic cells from 100% down to less than 0.01% (a four log reduction in the overall number of leukaemic cells) and is the prerequisite for an attempt at TFR.*

TFR Treatment-free remission for CML PATIENTS

CML AdvocatesNetwork

#TFR4CML

Global online survey on treatment-free remission

For the last 15+years, CML has gone from a fatal disease to a chronic one where people take a tablet every day.

By adhering to their treatment on one of the targeted tyrosine kinase inhibitor (TKI) therapies, they can have a near-normal lifespan.

And now there is the possibility that some people with CML can stop their treatment and stay in treatment-free remission (TFR) for a long time.

An online global survey is collecting the thoughts, experience and outcomes of CML patients who are planning to stop or have already stopped treatment with their TKI.

This is an initiative of the international CML Advocates Network and information from the survey will be used to create education materials for people with CML and also healthcare providers during the four stages of TFR.

The TFR 4 CML patients survey is aimed at better understanding the concerns, challenges and motivations during the decision phase, the first probation stage after stopping, and when attempting TFR has either worked or failed.

The survey, which is open now and runs through to July 2018, is seeking participation by people who are thinking about stopping, those who have stopped, as well as those who have had to restart treatment due to a rising PCR*.

It takes around 20 minutes to complete the anonymous survey and the responses are confidential.

The results will be professionally analysed to obtain patient-based evidence about TFR that will support both patients and doctors.

** PCR: polymerase chain reaction – a diagnostic and monitoring test used in CML to measure response to treatment.*

Here is a link to the survey:
http://bit.ly/TFRCML

Individualised support for your CML treatment journey

PATHWAY is a support program that offers information and support for people on CML medication including those advised by their doctor that they can stop taking their CML treatment.

The program supports people with CML at each stage of their treatment, as well as their relatives and caregivers.

For more information visit:
pathwayofsupport.com.au

Whether you have just been diagnosed or have been on treatment for many years, there are tailored services and updates that may be of benefit.

The support includes a welcome call and regular follow-up phone calls from a dedicated PATHWAY registered nurse to discuss how you're getting on without treatment, as well as regular SMS notification to remind you of the recommended monitoring requirements.

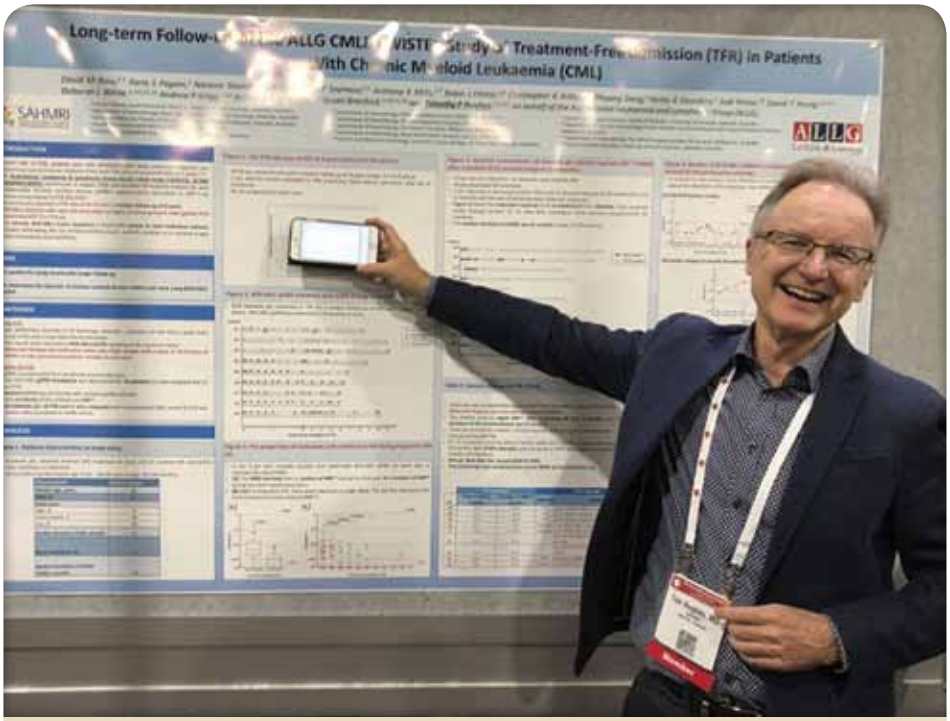
Speak to your doctor about enrolling into PATHWAY TFR.

Updated data on TFR from ASH 2017

New data presented at ASH 2017* underlined findings of previous trials and the increasing certainty on the safety of stopping CML treatment in a controlled and closely monitored clinical setting.

Uncertainty remains about factors that might predict whether a patient can successfully stop treatment or will experience a recurrence of CML leading to re-starting therapy. And there's still no clarity about the underlying biological mechanisms as to why some patients can stop successfully while others can't.

More than 2500 patients have stopped TKI treatment on STOP trials. Therapy-free remission (TFR) is achieved in 40-55% of patients after stopping TKI treatment while in deep molecular response (at least MR4, BCR-ABL below 0.01%) following several years of TKI treatment. Around 9 out of 10 patients who fail to stop treatment will lose their MMR within six months of ceasing their treatment. Current trials are trying to identify prognostic factors that predict the likelihood of an individual patient remaining in remission.



Professor Timothy Hughes with his poster at ASH 2017.

EURO-SKI

This is an ongoing trial and the largest stopping treatment study. Eligibility is based on being on any TKI treatment for at least three years and having a deep molecular response (MR4) for at least one year. Patients restart treatment if their PCR rises above MMR (BCR-ABL 0.1%). Of the 868 patients recruited, 47% remained in therapy-free remission at 36 months after stopping treatment. Even though 9 of 10 recurrences were within the first six months after stopping, there was a slow but steady decline of patients in therapy-free remission afterwards. There have been late relapses after 36 months, and a Scandinavian doctor mentioned one of his stop patients had a late recurrence after five years, which points to ongoing PCR monitoring.

Prognostic modelling on the data of 448 patients showed the best results of stopping imatinib were achieved if patients had at least 5.8 years of imatinib therapy and at least 3.1 years of deep molecular response. Similar data for dasatinib and nilotinib has not yet been analysed. A patient's prior history of suboptimal response or resistance to TKI therapy decreased the probability for therapy-free remission.

DASFREE

The dasatinib stop study is stricter than EURO-SKI, with a required deep molecular response of MR4.5 (BCR-ABL 0.0032%). Patients need to have received dasatinib for at least two years (one year less than EURO-SKI), have a deep molecular response for at least one year (same as EURO-SKI) with treatment restarted at the loss of MMR.

One-year follow-up data showed 44% had received dasatinib first-line, with the others going on this treatment after resistance or intolerance to prior treatment. The TFR rate at one year was 48-54% for first-line patients and 43% for second and subsequent lines of treatment. Median duration from discontinuation of therapy to loss of MMR was four months (range 1-16 months). All patients who had to restart treatment quickly regained MMR after therapy was reinitiated (median time to regain MMR was 1.9 months) and 90% regained at least MR4.5. The DASFREE study strongly supports the feasibility of stopping treatment in deep molecular response in first-line treatment with dastatinib and beyond.

TWISTER STOP

Long-term follow-up data in 40 imatinib-treated patients who had enrolled in the TWISTER study in 2006 and who had remained in TFR for a median of 8.6 years (range 5.8-10.8 years) showed the number of residual CML cells may further decline. A poster by Professor Timothy Hughes reported on minimal residual disease (MRD) levels in six patients over time, which decreased from a median of MR5.3 to MR6. This reduction in leukaemic cells, even after stopping imatinib treatment, could be either due to extinction of long-lived CML cells that lack the capability to self-renew, or the depletion of slowly proliferating CML precursor cells.

** The annual meeting of the American Society of Hematology, at Atlanta (U.S.), last December.*

Nilotinib label now includes stopping therapy info

The second generation CML treatment, nilotinib (Tasigna®), is the first tyrosine kinase inhibitor (TKI) to have treatment-free remission included on its product label.

Almost all patients with CML have an abnormality known as the Philadelphia chromosome (Ph+ CML), which produces a protein kinase called BCR-ABL, which causes malignant white blood cells to proliferate uncontrollably.

TKIs target and block the ability of the BCR-ABL protein kinase to send signals that drive production of these malignant cells.

Several TKIs are listed on the Pharmaceutical Benefits Scheme to treat a range of cancers in addition to CML but Tasigna is the first of them to have information on stopping therapy included on its Australian label for CML patients.

The addition of this data on the label provides people with CML and physicians with important clinical information for a potential approach for managing Ph+ CML.

Q & A: Professor Timothy Hughes

World leader in CML research, Professor Timothy Hughes has seen CML transform from "a universally fatal disease" in the 1980s, to a chronic disease requiring lifelong therapy in the 2000s, to a disease where treatment-free remission is possible for many in the current decade. He is Cancer Theme Leader at the South Australian Health and Medical Research Institute (SAHMRI), Beat Cancer Professor at the University of Adelaide, and has a host of accolades including the GSK Award for Research Excellence 2017.

Q Why did you choose a career in CML?

I specialised in CML after having the privilege of working with [the late] John Goldman in London in the 1980s. He was the leader in the field at that stage. CML was such a fascinating and challenging disease. We knew a mutant gene caused the leukaemia but couldn't convert that knowledge into treatment. I thought this was a worthwhile challenge to build my career on.

Q How has CML treatment changed?

The first agent that had an impact on survival was interferon. It was very toxic, with severe side-effects and only benefited about 25% of patients. When the tyrosine kinase inhibitors (TKIs) first became clinically available in Australia around 2000, we started seeing good responses in over 80% of patients. The responses were deeper and the tolerance of these oral agents was much better. This complete turn around in both effectiveness and tolerance is important when you're talking about a life-long therapy. The first TKI was imatinib (Glivec®) and the responses were so dramatic that a couple of CML experts, including me, proposed that perhaps some patients could eventually stop their therapy.

Q What was the initial response to treatment free remission?

The French and the Australian groups pioneered the concept of treatment-free remission (TFR) as a potential ultimate goal of CML treatment. For many years TFR was regarded as dangerous and inappropriate and something patients would find confusing, as on the one hand we tell them to take their drug every day, and on the other hand, one day we tell them to stop taking their drug. There's still a lot of concern about doing it properly, under the right conditions. The risk is that clinicians who aren't very experienced at treating CML may misinterpret or only get half the message and stop their patient's therapy after a couple of years when their responses have been good, but not as deep as you need to get successful TFR. That's something we talk about a lot at meetings and workshops; the critical requirements that make TFR a safe thing.

Q What stopping treatment trials have been held here?

We started a trial in 2006, as did the French, where patients who'd had the deepest response for at least two years actually stopped their therapy. Both trials made the same observation. Half the patients rapidly regained their leukaemia population, had to go back on therapy and got good [CML] control. And half remained in deep response, with no need for further therapy. Some patients have been off therapy for more than 10 years now, and remain in remission. So, we're starting to think of the possibility of actually curing some of these patients, but won't know that for another 10-20 years. In Adelaide, we have 80+ patients who have come off their drug and around half are still in remission. The other half have had to go back on their TKI drug and we're now looking at other ways of getting them off their therapies.

Q What's the latest results around TFR?

Our knowledge has rapidly evolved as we do more of these trials. The initial trials were only for patients who'd had a very long exposure to imatinib – at least eight years – and that's when we found out it was safe to attempt TFR. Now there are two more potent kinase inhibitors (nilotinib, dasatinib) that can be used as frontline therapy. The question we wanted to address was – do you need to wait so long before stopping these more potent TKIs, because we're achieving deep responses much earlier than we did with imatinib? We've conducted some studies and one was published last month in the *Annals of Internal Medicine* showing patients who've gone on a more potent TKI, after switching over from imatinib, are able to stop their therapy successfully as well. Another recent study shows that if you actually start [CML treatment] with the more potent drug, you can in many cases get them off their therapy within four to five years.

Continued: Asciminib – best treatment Lisa's had in 16 years

"I could have gone on the trial drug I'm on now, but I didn't want to," said Lisa.

"It hadn't been tried in humans and I felt at the time that I didn't need to put myself in that position, in case something did go wrong.

"Most people who come off their drug and relapse, like I did, will achieve total zero again within about three months," she said.

But after 12 months on nilotinib, Lisa's CML level still hadn't returned to zero, and that's when she changed her mind and decided to try asciminib.

"I thought maybe it's worth giving it a go," said Lisa who joined the asciminib trial in mid-2015.

"It was a bit scary because it was a Phase I trial. But by then people all round the world were on it. My doctor, who has conferences with other [trial] doctors every couple of weeks, didn't feel it was a risky thing for me to do."

"It's a tablet you take once a day and a bit like nilotinib, you have to fast one hour before or two hours after a meal.

"I take five tablets (200mg) before I go to sleep, so I don't get nausea, and I take painkillers every day," said Lisa.

"I've taken 3½ years and two drugs to get another zero. At my last appointment, it went up to .013, but I had a couple of 0.00s before that," she said.

"My personal aim is to get to 0.00, stay there for two years, then trial coming off the drugs again.

"I want to do that because I still have problems with my pain – it's a constant thing that bothers me more than the leukaemia. And I know my pain significantly improves when I'm not on any drugs.

"Where I'm sitting now, my [CML] levels are very low. There's no problem with living like that and the drug isn't giving me any new side-effects apart from pain and fatigue which have been ongoing issues," said Lisa, who works six hours a day most weekdays, depending on how her health is at the time.

"And even though I work, I am not actually that productive most days."

All Lisa's spare time is devoted to designing a new house, due to begin construction this year. And she's looking forward to a trip to America with her husband, Jaimie, to celebrate their 25th wedding anniversary.

"It's been a really tough journey but I learnt to be strong from my brave sister who passed away from cystic fibrosis.

"My other incentive to continue to fight comes every day from my two amazing children, who themselves face daily challenges with their vision impairment. They are such an inspiration to me.

"That's why I'm quite determined to get to zero and try coming off the drug, to see what happens again."

That's a very strong message to give to a young patient, particularly a young woman who wants to start a family but is aware that they can't attempt pregnancy while taking TKI therapy because it's teratogenic. This has been a real dilemma for many young women. Now, if they go on to more potent therapy right from the start, there's a very good chance they'll get a deep response in three to five years, and thus be eligible to attempt to achieve TFR.

"Asciminib may become the big new development of this decade and take over from the TKIs as the preferred treatment for CML."

Q Why do half the patients who stop treatment relapse?

That's something we're actively investigating. It's clear that the longer you keep a patient on their drug and the longer that they have achieved a deep response, the more likely it is that they will remain in TFR. There are competing hypotheses. One is that we're gradually getting rid of the leukaemic stem cells capable of causing relapse and eventually you no longer need the drug because you no longer have the cells present that are capable of relapsing. The other hypothesis, with equally strong evidence, is that the immune system is capable of controlling the CML cells when you get down to a very low number in the blood and bone marrow, so you no longer need TKI therapy at that stage. If that is the case, the way forward is to stimulate the immune system to allow patients to stay off their therapy long-term. There are trials starting where we use immune stimulatory drugs to see if we can achieve better success. There's a lot of interest in the checkpoint inhibitors being used in melanoma and lung cancer, which show remarkable activity because they awaken the immune system to see the cancer. There will be trials using those agents in this setting [CML] as well. The problem is, they are not without toxicity and you have to be very careful about using a toxic drug in a [CML] patient who has an excellent long-term chance of survival and whose only issue is whether they're on their drug or not. The challenge is to find drugs that are very low in toxicity but can give a meaningful boost to the immune system, to increase the chances of success when therapy is stopped.

Q What new drugs are in the pipeline?

We have 22 patients in Australia (and more than 200 people worldwide) on a Phase I trial for a very new drug, asciminib (previously called ABL-001). The TKIs (imatinib, nilotinib, dasatinib) work as small molecules that compete with ATP*, blocking kinase activity, and thus killing leukaemic cells because they are dependent on kinase activity for survival. In the process, there's some spill over of their effectiveness and activity against other normal and important kinases, which lead to side-effects (e.g., diarrhoea, fatigue, bone pain and muscle cramps). There's been a lot of interest in developing an inhibitor that doesn't have these off-target effects. Asciminib is an allosteric inhibitor – a new class of inhibitor – designed not to compete with ATP but to block this overactive kinase by binding to a different site on the protein, so you gain this incredible specificity. For someone who has come on to asciminib because they can't tolerate any of the TKIs or can't achieve a good response, this is a last chance. *(See cover story.)* We've been really excited by the results, which I first presented at the American Society of Hematology annual meeting in 2016.

Professor Timothy Hughes: "Asciminib is rapidly moving towards clinical development as a mainstream drug in CML".

"We can have fair confidence that the TGA does due diligence on the generics that are licensed in Australia and that it is safe to switch across."

We plan to publish a major paper on the trial results, which demonstrates the drug is much better tolerated than any of the TKIs and is effective in many patients who don't respond to the TKIs. Asciminib may become the big new development of this decade and take over from the TKIs as the preferred treatment. It is being developed by Novartis, the company that originally developed Glivec (imatinib) and they are now moving into Phase II and Phase III studies. We are about to start a Phase III study comparing asciminib to another TKI, so it's rapidly moving towards clinical development as a mainstream drug in CML.

Q Have many of your patients switched to generic imatinib?

Imatinib came off patent last year in Australia, allowing generic forms of the drug to be marketed in Australia at a lower cost. There was concern amongst patients about switching from the trusted drug they'd had for the last decade (Glivec) and worry that other (generic) forms of imatinib would not be effective. Numerous trials, including careful studies from Canada and Eastern European countries, where these changes came in a couple of years earlier than here, demonstrate that patients who had received good responses on the Novartis form of imatinib (Glivec), were maintaining their response when they switched to the generic form of the drug. Clinicians in Australia are generally satisfied that the generic drugs available to our patients are of good quality. We've been communicating that to patients and most are now pretty comfortable. Gradually, patients are understanding that any new drug (e.g., cholesterol-reducing statins) progress to a phase where appropriate generics are the norm. We can have fair confidence that the Therapeutic Goods Administration does due diligence on the generics that are licensed in Australia and that it is safe to switch across. *(See story on page 7.)*

** ATP: a complex organic chemical that participates in many processes.*

Part 2 of this interview with Professor Hughes will appear in the next issue of *CML News*.

CML's given Andrew "a better and more balanced life"

Andrew Bloxsom tried to delay a follow-up appointment with his GP until after a planned international business trip where he had 10 flights in eight days. Luckily he didn't.

"If I'd left the country, I probably wouldn't have come back," said Andrew, 41 of Sydney, describing the lead up to his diagnosis with CML.

It had been a Monday in May 2016 when he'd gone to his GP for a check-up because of a "little constant cough". He was due in Melbourne two days later, then travelling on to New Zealand and Singapore.

He was to have a blood test, which he put off until the next day (Tuesday) because he was "so busy", and later that afternoon he noticed five missed calls from his GP.

"My GP said he 'needed to see me first thing tomorrow'," said Andrew, an account director with an IT firm.

As his doctor was so insistent on seeing him immediately, Andrew took his wife, Tracey, to the appointment early the following morning (Wednesday), so she could take him on to the airport.

"I had my mind set on getting my flight changed to later in the day," said Andrew, but within three hours, he was in hospital!

"I felt alright and I'd done a quick race on Saturday," said Andrew, an avid cyclist who runs the junior academy at his Eastern Suburbs Cycling Club.

"But my body was starting to break down. My blood counts were so high and my spleen and kidneys were loaded up."

Six months earlier a cycling friend, William Richards, had been diagnosed with lymphoma.

"He'd said: 'you and I should ride to Canberra'. This was before I got sick."

They went ahead with these plans. Together they rode 370km over two days with three other friends, family and fellow club members in November 2016, raising $8000 for the Leukaemia Foundation.

Last November, a larger group of 12 did the ride again, this time raising $13,000 and finishing at John James Village – the Leukaemia Foundation's patient and family accommodation centre in Canberra. Andrew and William are working on the details of their Sydney 2 Canberra Leukaemia Cycle 2018, in what is fast becoming an annual ride.

Andrew has been on imatinib since starting treatment in June 2016 and after a few months he changed his medication to generic imatinib.

"After three months, when my counts weren't coming down, my dose was upped from 400 to 600mg."

It took Andrew a few months to digest having leukaemia and last year he suffered some depression.

"But at the end of the day, it's actually given me a better and more balanced life," said Andrew, who had accumulated 60 days annual leave and 40 days sick leave at the time of his diagnosis.

Dates for Andrew and William's S2C18 ride: the weekend of **27-28 October 2018**.

After completing the S2C17 ride, Andrew Bloxsom with his wife, Tracey, who rode with him last year, and their children, Millie, 10, and Bronte, 9, who both cycled the last eight kilometres, from Parliament House to the Leukaemia Foundation's John James Village.

Ken's "continuing as normal" on generic imatinib

Ken Verrier, who chose to switch to generic imatinib* nine months ago, says he feels no different and his CML levels are continuing to fall.

"I haven't had any problems at all," said Ken.

"I still have some days that are worse than others of course but it's been a pretty smooth transition."

Ken was diagnosed with CML in September 2015 soon after he moved to Cairns for a fresh start, having taken a redundancy as a sea captain in South Australia.

When he decided to shift back to Adelaide, "to be closer to family and friends and have access to a better medical system", he had already begun treatment on dasatinib. During the six months Ken was on this drug, he had a lot of side-effects – nausea, muscle pain and fatigue.

"I was feeling really yuck and when I saw another haematologist he changed my medication to imatinib," said Ken, 43, now of Port Pirie.

"At first, I still had a lot side-effects from the imatinib, but nothing like I had with dasatinib which made me pretty crook. I struggled to get out of bed and couldn't really work.

"For the last couple of years the side-effects have settled down. It's like I've got used to them," he said.

But Ken hasn't been able to return to work and is now permanently disabled due to pre-existing back issues that have been exacerbated since he got sick coupled with fatigue from the leukaemia.

When he was filling his imatinib prescription in mid-2017, a pharmacist told him about generic imatinib.

"It came with a free skin care pack," said Ken.

"I've had a problem with the skin on my arms and my face which gets very dry from the imatinib. I only have to scratch my skin and it tears and bleeds.

"These (skin) products have made a big difference. When I use them regularly my skin doesn't seem so bad."

Ken contacted his haematologist's office to let him know he was changing to a generic brand of imatinib, and to make sure there weren't any changes in the medication and that it wouldn't cause any problems.

"They said – 'it's all the same medication and it shouldn't be a problem'."

In the seven months since Ken has been on generic imatinib he said he felt no different and hadn't noticed any changes.

"I've just continued on as normal and my CML levels are still coming down.

"My doctor is very happy with the way everything is going and hopefully I'll get down to 0.00 which will be nice. It's at 0.02, I'm getting pretty close.

"It's good to see some competition in the pharmaceutical industry come in and it's much cheaper for the government through the PBS," said Ken about the availability of generic brands of imatinib.

"The cost of the generic medication I'm on is $2486, nearly $1000 cheaper than Glivec. The cost for me is $39.

"And it's working. What more can you ask for?"

** The patent for the original imatinib (Glivec®) ended in October 2016 and new brands of the drug have since entered the market.*

Ken Verrier made the change last year from original to generic imatinib.

Generic imatinib benefits Leukaemia Foundation

The Leukaemia Foundation benefits from sales of generic imatinib in a partnership with Australia's first 'for-benefit' pharmaceutical company.

For Benefit Medicines (FBM) was established under a social enterprise model with the sole purpose of distributing 100% of profits to patient support and medical research in Australia.

FBM distributes an imatinib generic drug – Cipla Imatinib. This medicine, approved by the Therapeutic Goods Administration for use in Australia, is required to meet the same strict regulations as applied to the original brand (Glivec®), with regard to quality, safety and effectiveness.

Since November last year, FBM Foundation has supported the Leukaemia Foundation through a partnership agreement where 100% of profits from sales of their brand of imatinib (Cipla Imatinib) go to the Leukaemia Foundation.

With a change to Cipla Imatinib, the Leukaemia Foundation can receive thousands of dollars which will be directed to further research based on the Leukaemia Foundation's research funding policy.

FBM's generic medicine – Cipla Imatinib – is now available on prescription from doctors and for dispensing through pharmacies. It is listed on the Pharmaceutical Benefits Scheme and is the same cost to people with CML as other current CML imatinib medications.

For more information about generic imatinib refer to the Q & A article on page 3 in *CML News*, October 2016: **http://bit.ly/CMLnews16**

NEW SOUTH WALES & AUSTRALIAN CAPITAL TERRITORY

Sydney Metro

17 Apr	10am-12pm	Liverpool Blood Cancer Information & Support Group (also 15 May, 19 Jun, 17 Jul, 21 Aug, 18 Sep, 16 Oct)
18 Apr	2-4pm	Randwick Support & Education Group (also 16 May, 20 Jun)
25 Apr	11am-1pm	Westmead Blood Cancer Education & Support Group (also 30 May, 27 Jun, 25 Jul, 29 Aug, 26 Sep, 31 Oct)
30 Apr	10-11.30am	St George Blood Cancer Education & Support Group (also 28 May, 25 Jun, 30 Jul, 27 Aug, 24 Sep, 29 Oct)

Australian Capital Territory & Southern New South Wales

5 Apr	4-6pm	Bega Valley & Sapphire Coast Blood Cancer Support Group, Merimbula (also 28 Jun, 23 Aug, 18 Oct)
9 Apr	11am-1pm	Goulburn & Surrounds Blood Cancer Support Group (also 14 May, 4 Jun, 9 Jul, 13 Aug, 10 Sep, 8 Oct)
10 Apr	10am-12pm	Canberra Blood Cancer Support Group (also 8 May, 12 Jun, 10 Jul, 14 Aug, 11 Sep, 9 Oct)
22 May	5.30-7.30pm	20/30 Chat, Garran (also 28 Aug, 27 Nov)
25 May	1.30-3.30pm	Moruya & Surrounds Blood Cancer Support Group (also 20 Jul, 21 Sep, 16 Nov)

Central Coast

26 Apr	10-11.30am	Gosford Blood Cancer Education & Support Group (also 31 May, 28 Jun, 26 Jul, 30 Aug, 27 Sep, 25 Oct)

Central West & Far West

4 Apr	10.30am-12pm	Dubbo Blood Cancer Education & Support Group (also 2 May, 6 Jun)
5 Apr	10.30am-12pm	Orange Blood Cancer Education & Support Group (also 3 May, 7 Jun)
13 Apr	10.30am-12pm	Mudgee Blood Cancer Education & Support Group (also 14 Jun, 9 Aug, 11 Oct, 13 Dec)

Hunter

3 Apr	10am-12pm	Newcastle Blood Cancer Education & Support Group, Mayfield (also 5 Jun, 7 Aug, 2 Oct, 4 Dec)
10 Apr	10am-11.30am	Port Stephens Blood Cancer Education & Support Group (also 12 Jun, 14 Aug, 9 Oct, 20 Nov)
1 May	10am-12pm	Newcastle Blood Cancer Education & Support Group, Shortland (also 3 Jul, 4 Sep, 13 Nov)

Illawarra & Shoalhaven

23 May	10am-12pm	Bowral Blood Cancer & Support Group (also 25 Jul, 26 Sep, 28 Nov)
2 Jun	12-3pm	CML Support Group, Unanderra (also 1 Sep, 1 Dec)

Mid North Coast

16 Apr	10-11.30am	Port Macquarie Blood Cancer Education & Support Group (also 21 May, 18 Jun, 16 Jul, 20 Aug, 17 Sep, 15 Oct)
26 Apr	11.30am-1pm	Coffs Harbour Blood Cancer Education & Support Group (also 24 May, 28 Jun, 26 Jul, 23 Aug, 27 Sep)

New England

10 Apr	10.30am-12pm	Armidale Blood Cancer Education & Support Group (also 8 May, 12 Jun, 10 Jul, 14 Aug, 11 Sep, 9 Oct)
17 Apr	1.30-3.30pm	Tamworth Blood Cancer Education & Support Group (also 15 May, 19 Jun, 17 Jul, 21 Aug, 18 Sep, 16 Oct, 20 Nov, 18 Dec)

SOUTH AUSTRALIA

Adelaide Metro

17 Apr	10am-12pm	Northern Adelaide Support Group, Evanston (also 19 Jun, 21 Aug, 16 Oct)
18 Apr	10.30am-12.30pm	Strathalbyn Support Group (also 16 May, 20 Jun, 18 Jul, 15 Aug, 19 Sep, 17 Oct, 21 Nov)
24 Apr	10.30am-12.30pm	Men's Group, Adelaide (also 26 Jun, 28 Aug, 30 Oct)
28 May	10am-12pm	CLL/CML Support Group (also 30 Jul, 24 Sep, 26 Nov)

Regional South Australia

10 Apr	10am-12pm	Port Lincoln Support Group (also 12 Jun, 14 Aug, 9 Oct)
4 Jun	5.30-6.30pm	Mount Gambier Support Group (also 6 Aug, 8 Oct, 3 Dec)

TASMANIA

Northern Tasmania

10 Apr	10.30am-12pm	Blood Cancer Support Group, Launceston (also 12 Jun)
25 May	9.30am-12pm	Cooking for Chemo, Burnie

Southern Tasmania

11 Apr	11am-1pm	Hobart Blood Cancer Support Group (also 8 Aug, 8 Sep, 10 Oct, 14 Nov, 5 Dec)
19 Apr	11am-12.30pm	She Shed, Battery Point (also 17 May, 21 Jun, 19 Jul, 16 Aug, 20 Sep, 18 Oct, 22 Nov)
24 Apr	11am-1pm	Cooking for Chemo (also 13 Jun)

VICTORIA

Metro Melbourne

15 May	10.15-11.45am	Northern Suburbs Blood Cancer Support Group, Hawthorn (also 17 Jul, 16 Oct)
18 May	10-11.30am	Berwick Support Group
31 May	10.15-11.45am	Bone Marrow & Stem Cell Transplant Support Group, Hawthorn (also 2 Aug, 1 Nov)

Barwon & South West

17 Apr	10-11.30am	Barwon Blood Cancer Support Group (also 19 Jun, 14 Aug, 9 Oct, 11 Dec)
30 May	10-11.30am	Warrnambool & South West Blood Cancer Support Group (also 5 Sep, 28 Nov)

Grampians

11 Apr	10-11.30am	Ballarat Blood Cancer Support Group (also 13 Jun)
17 Apr	11am-12.30pm	Horsham Blood Cancer Support Group (also 19 Jun)

Loddon/Mallee

10 Apr	10-11.30am	Bendigo Blood Cancer Support Group (also 12 Jun)
16 Apr	1.30pm	Mildura Blood Cancer Support Group (also 18 Jun)

WESTERN AUSTRALIA

Perth Metro

16 Apr	1-3pm	Perth Metro Blood Cancer Support Group

Bunbury

5 Apr	10:30am-12pm	Bunbury Regional Blood Cancer Support Group

Peel

19 Apr	10:30am-12pm	Peel Region Blood Cancer Support Group
27 Apr	1-2:30pm	Port Kennedy Blood Cancer Support Group

Join the CML Network closed group on Facebook: https://www.facebook.com/groups/CMLLFA/
Visit www.leukaemia.org.au for our latest Education and Support Program Event Calendar.
To register for an education or support event, freecall 1800 620 420 or email info@leukaemia.org.au

Leukaemia Foundation
VISION TO CURE
MISSION TO CARE

Contact us

GPO Box 9954, IN YOUR CAPITAL CITY 1800 620 420 info@leukaemia.org.au
leukaemia.org.au LeukaemiaFoundation LeukaemiaAus LeukaemiaFoundation