AML NEWS

For people with AML & their families

Leukaemia Foundation
VISION TO CURE
MISSION TO CARE

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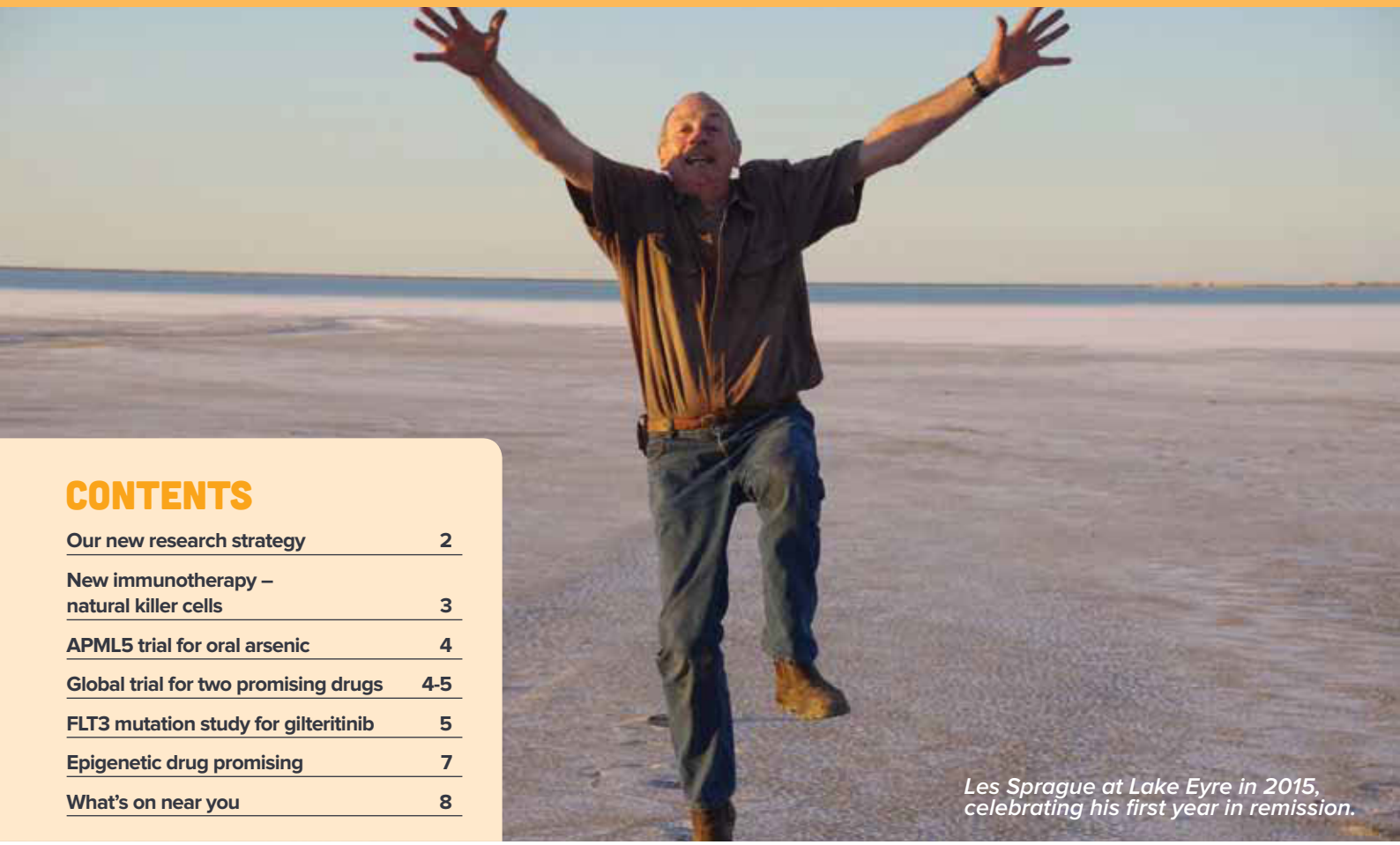

Les Sprague at Lake Eyre in 2015, celebrating his first year in remission.

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AFTER SURVIVING AML LES SAID "THANKS" BY RAISING FUNDS AND AWARENESS

It was during a Saturday afternoon snooze in April 2014 when Les Sprague, 71, got a phone call and the "total blinking shock" of being told he could have leukaemia!

He had been only weeks away from heading off to Vietnam with his wife, Paula, when he was overcome with a feeling of "absolute tiredness". He had nosebleeds, hot flushes and was losing weight as well.

"I felt pretty exhausted the whole time and couldn't understand why," said Les, a visual artist, now aged 75.

"Obviously I couldn't go to Vietnam. I had to do something."

Thinking it was a passing virus Les had a blood test on a Friday.

"To my horror, a pathologist rang from Melbourne the next afternoon while I was lying down.

"He said 'you've got some very low blood counts and better get to the emergency department. It looks like you've got leukaemia.

"'Get an ambulance, you can't drive', he said, in case I had an accident because any sort of bleeding could be dangerous," explained Les, who lives on a small farm on the edge of the forest at Enfield, near Ballarat (Vic).

"I drove. It was only 25km. I was terribly upset and when I rang my wife who was coming home from a work event, I could hardly even breathe the word 'leukaemia' because I knew what leukaemia was."

Les spent that night in hospital at Ballarat and the next day was transferred by ambulance to a Melbourne hospital. After a range of diagnostic and stress tests, he was told 'if we don't do anything, you've got a life expectancy of two to three weeks!'.

"The haematologist also said, 'let's hope and pray you've got inversion 16', which meant nothing to me at the time. It's a chromosomal thing, which means you respond to a certain cytotoxic drug and that's a good thing, and miraculously that's what I had," said Les.

"Those first hours and days were totally disorientating and de-stabilising. There was shock and also the understanding that my death would mean the tragic loss of the relationship between my wife and I."

Continued on page 6.

OUR NEW RESEARCH STRATEGY INVESTS IN INNOVATION

The Leukaemia Foundation's new research strategy supports medical research that drives rapid advancements in treatments, encourages the careers of promising scientists and discovers new diagnostics and novel therapies. Giving Australians access to global clinical trials is another key aim.

CEO, Bill Petch said this new research framework came from consulting with the Leukaemia Foundation's stakeholders over the last two years.

"We have turned our attention to investment in innovation – in diagnosis, treatments and improving quality of life across the blood cancer spectrum," said Bill.

"And by forging new, strong research partnerships with leading research agencies, including HSANZ, Cancer Australia, the Centre for Blood Transplant and Cell Therapy's Centre of Research Excellence, our new research program is powered for maximum impact.

"Our research priorities are understanding the biology of blood cancers, tailor-made therapies to treat each patient's cancer and the psychosocial aspects of blood cancer.

"Other key areas will include innovative clinical trials, new therapies and prevention research which includes investigating risk factors and possible causes of blood cancers."

Bill Petch, Leukaemia Foundation CEO.

Our current (2017-2019) multi-million dollar funding commitment to research will grow over coming years in line with generous support from the community.

To find out more about our new research program visit: leukaemia.org.au/research

GVHD RESEARCH BRINGS NEW HOPE

There is an urgent need for new treatment approaches to better prevent and treat graft versus host disease (GVHD).

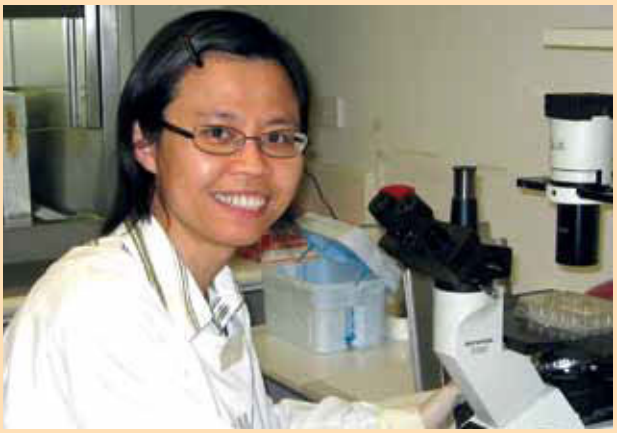
In response, the Leukaemia Foundation has announced a $1 million investment to the Centre for Blood Transplant and Cell Therapy (CBTCT).

The CBTCT is a newly funded Centre of Research Excellence (CRE) endorsed by the National Health and Medical Research Council (NH&MRC) to develop a world class, multi-centre approach to design and deliver improved therapies for people with blood cancers.

The Leukaemia Foundation is a major partner and the only non-government organisation to support this project to better prevent and treat GVHD and maintain and/or augment immunity to leukaemia.

Better outcomes for GVHD will be achieved by:

- changing clinical practice for treating transplant recipients who have GVHD,
- discovering biomarkers with diagnostic, prognostic and predictive power, to prevent GVHD, and
- using novel agents, such as CAR T-cell therapy, immunomodulatory and immunotherapy agents and genetically modified T-cells, to reduce blood cancer relapse and improve patient survival.



Dr Siok Tey – one of five chief Australian investigators for the CBTCT.

ABOUT GVHD

A bone marrow (or peripheral blood stem cell) transplant (BMT or SCT) is a possible treatment for AML.

GVHD may occur after an allogenic BMT or SCT (in which someone receives bone marrow tissue or stem cells from a donor). These donated cells view the recipient's body as foreign and attack it.

GVHD affects most (50-70%) of BMT or SCT recipients, with 20% of them developing severe acute GVHD that doesn't respond to conventional treatment.

Together with opportunistic infections, GVHD accounts for most transplant-related deaths.

Q&A WITH KIMBERLY STEGMAIER

There is much that excites U.S. physician/scientist, Kimberly Stegmaier, about advances in AML research. The Professor of Pediatrics at Harvard Medical School, Boston, was one of the international speakers at the Leukaemia Foundation-hosted New Directions in Leukaemia Research conference, in Brisbane earlier this year. *AML News* spoke to her following her presentation, 'Clinical translation of AML genomics'.

To read this story, visit: bit.ly/ProfessorStegmaier

UNLEASHING FULL POTENTIAL OF NK CELLS TO TREAT AML

A major finding by basic science researcher, Rebecca Delconte, during her Leukaemia Foundation PhD scholarship, utilises natural killer cells in a potential new form of immunotherapy to treat AML.

The 28 year-old, who works in the Huntington lab at the Walter and Eliza Hall Institute (Melbourne), has already achieved so much.

Halfway through her scholarship (2015-2017), Rebecca's research was published in the prestigious journal, *Nature Immunology* in 2016. A drug company is now searching for a compound to take her discovery to the pre-clinical stage. She has completed her PhD and recently accepted a position at one of the largest research institutes in the U.S., to continue her research work on a specialised type of immune cell – natural killer cells (NK cells), and AML.

"... it looks as though it will be a very translatable finding, which is really exciting."

"I could never have achieved all these things without my supervisor, Associate Professor Nicholas Huntington, the lab, and the Leukaemia Foundation – it's never a solo project," said Rebecca whose field is immunotherapy.

During her scholarship, she worked on NK cells, which she describes as "the sentinels of your immune system".

"While many forms of immunotherapy have been largely successful, they mainly focus on another immune cell – T-cells – and they can have quite severe off-target effects.

"Because we know how good NK cells are at fighting tumours, we wanted to find an immunotherapy directed against NK cells instead of T-cells," Rebecca explained.

"In healthy individuals, NK cells constantly circulate in the body searching for cells that are stressed, mutated or transformed – cells which could be the beginning of tumour formation.

"The job of NK cells is to clear out those cells, to prevent cancer.

"Obviously people still do get sick and still get cancer, so we wanted to try and manipulate NK cells to make them better at their job," she said.

"We looked at how they respond to different stimuli and found a protein, called CIS, that is crucial for growth factor signaling in NK cells.

"NK cells need a particular growth factor to survive and CIS regulates how NK cells see that growth factor.

"We found that if we targeted that protein (CIS) – by getting rid of it – the NK cells responded better to the growth factor. This caused them to grow and do their anti-tumour job better –10 times better than normal!

"This was the major finding – that removing this protein from NK cells enhances their anti-tumour activity.

"We showed this in several solid tumour cancer models. I also looked at AML and found AML survival was increased when you remove CIS from NK cells. It was a really, really potent response.

"And it looks as though it will be a very translatable finding, which is really exciting," said Rebecca, the lead author on the *Nature Immunology* paper that has since generated lots of interest, particularly from pharmaceutical companies.

"We have since partnered with one company, to find a drug that can specifically target CIS.

"If there is a drug that can target CIS, we could first test it in pre-clinical models, then hopefully in a Phase I clinical trial.

"That's the blue sky goal, which is still a little while away."

While these experiments are underway, Rebecca has looked into the mechanisms behind the anti-tumour response "to understand why NK cells without that protein are so much better at killing tumour cells".

"There seems to be a number of reasons why," she said.

"Not only do NK cells missing CIS kill more tumour cells, they also grow much faster, so there are more of them available to do the anti-tumour job.

"Our aim is to remove CIS from NK cells, to pump them up and make them better at what they do.

"By targeting CIS, we're unleashing the full potential of the body's own NK cells to then go and attack the AML.

Rebecca Delconte.

"Seeing the translational potential of this basic biological finding has been what has renewed my passion for basic biological research," said Rebecca.

In a partnership with the HSANZ, the Leukaemia Foundation is funding new PhD scholarships as part of its new research strategy. ***(See story on page 2.)***

"I wouldn't have done this research and it might not have happened if the Leukaemia Foundation hadn't funded me."

Read how Rebecca Delconte's PhD scholarship from the Leukaemia Foundation changed the course of her research career. Next year she takes up a post-doctoral position at the Memorial Sloan Kettering Cancer Center in the U.S. to continue working in basic biological research "where the biggest discoveries are still yet to be made".

Visit: bit.ly/BasicBiologicalResearch

TWO TARGETED THERAPIES OFFER PROMISE FOR NEWLY DIAGNOSED

A Phase III international clinical trial for two promising drugs "heralds a new era in precision medicine for people with AML" according to Associate Professor Paula Marlton.

The Head of Leukaemia and Lymphoma Services at Princess Alexandra Hospital (Brisbane) is leading the trial in Australia when it opens early next year.

Two small molecule inhibitors – ivosidenib and enasidenib – will be tested in combination with standard chemotherapy for people newly diagnosed with AML who have either the IDH1 or IDH2 mutations.

This is the first time a targeted therapy has been available to these patient groups, which together make up around 20% of the AML patient population.

Ivosidenib inhibits the activity of the mutated IDH1 gene product and enasidenib targets the related product from mutant IDH2.

"A lot of work has been done on these drugs in the relapsed and refractory setting. Those patients normally do very poorly and their outlook is grim," said Assoc. Prof. Marlton.

"The Phase I/II results for each drug showed responses that were meaningful for quite a few patients in this difficult setting.

"For enasidinib, which is in more advanced clinical development, the overall response rate (ORR) was 40%, with almost 20% achieving a complete response (CR).

"This is certainly remarkable as these were patients essentially out of options who participated in a Phase I trial hoping to benefit from a new targeted agent aimed at addressing one of the mutations in their relapsed leukaemia.

"Enasidinib gave them a meaningful extension of life and in many cases an improvement in quality of life," said Assoc. Prof. Marlton.

"Even among those patients who didn't achieve a CR, many benefited in terms of a reduction in transfusion requirements.

"Their haemoglobin levels and white blood counts went up, so they got less infections, their platelet counts went up, requiring fewer transfusions to prevent bleeding, and their blast count (leukaemia count) went down.

"Those are meaningful responses for a group of patients with a huge unmet need.

APML5 TRIAL RECRUITING EARLY–2019

The APML5 trial is the first time in the world a particular capsule formulation of oral arsenic – arsenic trioxide (ATO) – has been studied specifically to optimise treatment for APML.

Principal investigator Professor Harry Iland said the Australasian Leukaemia & Lymphoma Group study, which opened in June last year, "was a different trial from usual".

"This Phase I trial is not an efficacy trial, it's a pharmacokinetic study, and it's constructed in two parts," he said.

"Standard treatment for APML involves treatment with IV ATO, asll-trans retinoic acid (ATRA), and sometimes chemotherapy. APML induction lasts 4-5 weeks, depending on the white cell count at presentation, followed by seven months of consolidation that is based around four cycles of arsenic. Each cycle lasts four weeks, with a four-week break between cycles.

"Before you go on to the APML5 trial, you need to have been on a standard regimen for induction and be in remission," said Prof. Iland.

"This trial only starts at the beginning of consolidation."

The arsenic is traditionally given daily by intravenous infusion (IV) for five days a week for four weeks in each cycle of consolidation.

"This trial's methodology is the same but in the first week of each cycle we're collecting blood samples and doing pharmacokinetic studies, to measure the amount of arsenic in the blood.

"And in two of those four cycles, instead of using IV arsenic, we're using a new oral formulation of arsenic trioxide.

"Our overall aim is to identify a dose of oral arsenic that gives us the same amount of arsenic in the blood as if you gave it by IV infusion.

"Then we can plan a full Phase II or III study that would be a direct comparison of IV versus oral arsenic and which would look at quality of life as well as efficacy and safety.

"Intravenous arsenic is very cumbersome. It's a two-hour infusion every day for 80 days spread over seven months, so it's a massive disruption for patients and a huge resource waste for the hospital.

"If we can eventually switch over to oral arsenic, that would be a tremendous improvement in the way patients are treated over such a long period of time.

"We have to do this long study, where we still rely mainly on IV arsenic for safety reasons, but we still get two valuable weeks of oral arsenic exposure when we can do the blood testing and pharmacokinetic studies to compare it with the IV arsenic given to the same patients.

Professor Harry Iland.

"By giving the bulk of their treatment in the conventional way with IV arsenic, we know they are being treated appropriately, but at the same time include a little bit of the new agent that we can study and directly compare to the standard treatment."

Accrual for the first part of this Phase I trial, with eight "evaluable patients", has been completed. The trial is on hold as some of these patients are still going through consolidation treatment.

"We have some early data but not enough yet to recommend a dose of oral arsenic for the second part of the trial, scheduled to open early next year at 15 Australian sites, with a planned enrolment of 20 patients."

"The ivosidenib trial showed very similar promising results, with similar outcomes in terms of the improvement in transfusion requirements, blood counts and the responses," said Assoc. Prof. Marlton.

Almost 42% of patients responded to this treatment, with 22% achieving a CR.

The FDA in the U.S. has approved both drugs for relapsed or refractory AML.

"Now these drugs have moved to the frontline setting and are being trialed in combination with standard chemotherapy for newly diagnosed AML," said Assoc. Prof. Marlton.

"Knowing these trials were coming, we have been collecting information on our patients' IDH status. This requires mutation analysis of their leukaemia cells when they first present [at diagnosis].

"It involves taking leukaemia cells from their diagnostic bone marrow and performing a mutation analysis for the genes in question [IDH1 and IDH2], using genomic sequencing techniques.

"This is referred to as precision medicine and it involves a huge paradigm shift in our approach to AML which is the past has been 'one size fits all'. Now we are hiving off groups of patients with specific mutations," said Assoc. Prof. Marlton.

"We've already been separating those with FLT3 mutations *(see story below)* and now this group of patients too – the 8-10% with IDH1 and ~12 % with IDH2 – and that approach will continue as new drugs are developed.

"Having rapid, accurate, robust genomic testing available at diagnosis is very important, to slot patients very quickly into the appropriate treatment.

"The reason for screening everyone, regardless of the trial availability, is to have this information for the future so that, if they unfortunately go on to relapse, we may be able to access these drugs through a compassionate access program. In addition, we are gearing up for this frontline study," she said.

Assoc. Prof. Paula Marlton.

The study is being run out of Holland and Germany and is a collaboration between the lead study groups, HOVON – the Dutch equivalent of the Australasian Leukaemia and Lymphoma Group (ALLG) – and Germany's AML Study Group.

"Both are highly renowned throughout the world and the study has been extended to global co-operative groups including, for the first time, Australia, through the ALLG.

"This new collaboration is an exciting new era in global clinical trials in AML," said Assoc. Prof. Marlton.

"AML is a relatively rare condition and we have to work cooperatively as a global community to try and advance the therapies for AML patients, especially now we are treating small subsets of patients with specific mutations with a specific precision therapy."

For more information about the HOVON 150 trial, visit: bit.ly/HOVON150Trial, contact Assoc. Prof. Paula Marlton on (07) 3176 2111 or speak to your haematologist.

MIDOSTAURIN TRIAL PAVED WAY FOR NEXT GENERATION GILTERITINIB STUDY

Midostaurin (Rydapt®) is the first targeted therapy to be approved by the Therapeutic Goods Administration for people newly diagnosed with AML who have the FLT3 mutation.

This recommendation was based on data from the RATIFY trial.

Australian patients were among 3277 patients aged under 60 years screened with a molecular test to identify whether they had the FLT3 mutation, which occurs in about a third of all AML patients.

There were 717 patients with the mutation on the Phase III trial who were randomised to midostaurin, or a placebo, in combination with standard intensive chemotherapy.

The trial resulted in an improvement in overall survival for patients with the FLT3 mutation who were on the midostaurin arm. Median survival was increased from 44% to 51% at four years.

"This is the first targeted therapy approved for patients with FLT3 mutation," said Associate Professor Wei, haematologist at the Alfred Hospital and Monash University.

"It is likely that midostaurin will be a new standard of care for patients with the FLT3 mutation and there are patients already around the country who are receiving this drug through an access program.

"This study, along with the parallel IDH1/IDH2 HOVON trials targets three mutations that affect more than half of all AML patients."

"We hope that this drug will be available through the PBS."

Leading on from the RATIFY trial, Assoc. Prof. Wei is now collaborating with the HOVON group in Europe for a trial investigating a potentially more potent FLT3 inhibitor, to open in Australia next year.

Participating patients will have FLT3 identified, then will be randomised to either the new drug (gilteritinib) or midostaurin, both in combination with standard intensive chemotherapy.

Dr Wei said the HOVON study would make both FLT3 inhibitors available after a stem cell transplant as well.

"It will be a big study, available at multiple sites here through the Australasian Leukaemia & Lymphoma Group," said Assoc. Prof. Wei.

"It is the beginning of a new era for more personalised and targeted therapies to improve patient outcomes in AML, and hopefully there will be more therapies to come."

This study, along with the parallel IDH1/IDH2 HOVON trials *(see story above)* targets three mutations that affect more than half of all AML patients.

"We're trying to make FLT3 and IDH screening universal and standard for all patients in Australia," said Assoc. Prof. Wei.

CONTINUED: AFTER SURVIVING AML LES SAID "THANKS"

Paula immediately took time off work as a teacher to be with Les "for as long as it took" which turned out to be four months, and the Spragues remained in Melbourne while Les had four cycles of intensive treatment.

"As we lived way up in the provinces, we were hardly likely to go back and forwards [to Melbourne] every day," said Les.

"Paula was put in touch with the Leukaemia Foundation and they quickly found a unit and made that available to us for the entire time of my treatment.

"It was a haven. Paula would go home at the end of each day and just collapse. It was very unsettling for her to have me with such a debilitating disease so suddenly," he said.

"The unit was comfortable, close, and I was able to go there between cycles of chemotherapy, to recover before going back to hospital."

Despite his fitness, and being told he had the body of a 60-65 year old, Les was too old to have a stem cell transplant. So his haematologist advised that Les was to get 'the strongest dose without actually killing him'.

"And it worked," said Les.

"The first treatment nearly killed me and was terribly painful – it was touch and go."

Les believes he survived AML by finding out everything he could about his blood cancer and not "feeling like a victim".

"I saw people lying in hospital passively copping whatever was going to be given to them, and we didn't do that. We got ourselves in a position where we could be active members of the team treating this thing.

"I made sure I knew the names of all the nurses, the registrars, the cleaners, everyone who had anything to do with us and that made it easier for them to see me not just as a patient but as a person," said Les.

During his second cycle of chemotherapy, Les got a serious infection. Initially thought to be a haemorrhoid, it turned out to be an infection in his blood, from his bowel, because his immune system had failed.

"It was my insistence and Paula's that they properly diagnose this and I think that turned the corner for me and helped save my life. I had to have surgery to fix it.

"I was as weak as a kitten during treatment but luckily I keep pretty fit and that was important, but there were days when I thought, nah, I'm not going to get through this," said Les.

He finished his treatment and went home in July 2014.

"I've had no more chemo or anything since then. Just close surveillance," said Les, who has got on with his life, continuing his work as an artist along with the "millions of jobs that have to be done even on a small farm".

"The Leukaemia Foundation was so good to us," said Les.

"I thought it would be good to do something for them and to raise awareness of what the Leukaemia Foundation does for people, particularly in regional Australia, to make their treatment more palatable or easier.

"I'm an artist and people like art. We decided to say 'thank you' in a way that meant something to us."

Paula and Les in their Leukaemia Foundation unit in Melbourne, 2014, and Les' lucky king parrot mascot.

Last year, in Ballarat, Les held a sell-out exhibition of his drawings, paintings, etchings and photographs, predominantly of the outback and covering his work over 30 years. He also published a 2018 calendar, with help from a team of enthusiastic supporters, and the Spragues donated a generous percentage of sales from both to the Leukaemia Foundation – $8000.

Earlier this year, Les and Paula finally got to go to Vietnam.

AML NEWS – A TURNING POINT FOR PAULA

When Les was first diagnosed, finding and reading a copy of *AML News* in the hospital ward in Melbourne was critical for Paula Sprague.

"We knew about leukaemia but we didn't know what AML was. Yet AML was well known and widespread enough that there was actually a newsletter about it!" said Les.

"Paula found out a lot of research was going on and an organisation [the Leukaemia Foundation] supported people like me, and their families.

"That newsletter was an important part of Paula being able to deal with this and a turning point for her in marshalling support for me.

"It's so true that a good news story is what people need when they've been sandbagged by this very fast moving disease."

EPIGENETIC DRUG TRIAL RECRUITING FOR RELAPSED OR REFRACTORY AML

A Phase II trial for a 'first in class' epigenetic drug that already has shown promising results is recruiting in Australia for people with relapsed or refractory AML.

This drug, called IBET*, was developed by GlaxoSmithKline in collaboration with work performed in Professor Mark Dawson's laboratory, with funding support from the Leukaemia Foundation.

Prof. Dawson said results from the international Phase I study of 40 patients from five hospitals in Australia, the U.S. and the UK, had proven the drug was "safe and tolerable".

"The results of that clinical trial will be reported later this year," said Prof. Dawson, a clinician-scientist at the Peter MacCallum Cancer Centre (Melbourne).

Prof. Dawson received a $1 million five-year senior research fellowship from the Leukaemia Foundation in 2013.

"It has been invaluable in helping me re-establish the lab in Melbourne," he said.

In 2014, Prof. Dawson, along with PhD student, Dr Chun Fong, moved from the University of Cambridge (UK) to the Peter MacCallum Cancer Centre.

"In the last five years the lab has grown from one to 28 people," said Prof. Dawson.

"The importance of the Leukaemia Foundation in providing the platform to build that lab cannot be overstated.

"It has been absolutely critical. They have not only supported me, they've supported many members of my lab."

Three members of Prof. Dawson's lab also have received research grants from the Leukaemia Foundation: Dr Chun Fong, PhD scholarship (clinical), $180,000, 2013-2015; Mr Dean Tyler, PhD scholarship top up grant, $14,151, 2015-2017; and Dr Omer Gilan, postdoctoral fellowship, $300,000, 2015-2017.

"They helped establish this critical mass for me to build the research team and in terms of what they have achieved, it's fair to say they have achieved enormously on a world scale."

Dr Chun looked at how leukaemias develop resistance to therapy.

"Insights from his work have been far-reaching, published in one of the world's premier scientific journals, *Nature*, and cited more than 100 times since its publication," said Prof. Dawson.

"It has been referred to as a seminal body of work because it established major principles that previously eluded us, including the fact that resistance often emerges from leukaemia stem cells, and almost as importantly, resistance can emerge in the absence of new mutations.

"Those two key insights have provided ongoing work in the lab and Dr Fong used his Leukaemia Foundation scholarship to build that body of work and it provided him with his own platform to get independent funding.

"He has moved on to be the head of myeloid malignancies at the Austin Hospital and has started his own area of research."

Dean Tyler developed a new chemical method to show "at an unprecedented level" how drugs work within cells.

"We often make the assumption that drugs are equally distributed around the body and work effectively, regardless of the tissue in which the cancer is harboured," said Prof. Dawson.

"Dean's work, published in an equally prestigious journal, *Science*, really challenged that dogma and showed, using new methods, that we can now trace and identify exactly where in the cell the drug is going and why that drug is able to turn a gene on or off. And it showed that some tissues, such as the bone marrow, which often harbours leukaemia cells, does not necessarily get the level of drug exposure one had previously assumed.

"This really helps us develop better strategies for the development of leukaemia," Prof. Dawson said.

Another premier journal, *Nature Structural and Molecular Biology*, published Dr Gilan's work, which showed, in a very aggressive leukaemia (often seen in the paediatric patient population) how two key proteins, that regulate the expression of genes that sustain the leukaemia, interact and work together.

"Those insights ultimately will lead to new therapies in the long run to really tackle these two key proteins and hopefully give a better outcome for adults and children who have that particular AML subtype."

Professor Mark Dawson.

Prof. Dawson said that, in addition to establishing a successful team and doing groundbreaking research, "we've taken that research into the clinical arena".

"We've taken the new molecules we have discovered to run the clinical trial which involves testing a first-in-class drug that affects the way genes are turned on and off in cancer cells.

"The drug is a bromodomain inhibitor that targets a critical protein, called BRD4, which is an essential regulator of malignant gene expression programs in cells.

"It is an epigenetic therapy," said Prof. Dawson.

"Epigenetic proteins regulate access to our DNA. Our DNA contains genes that need to be expressed, the DNA itself is replicated, and the DNA is repaired after DNA damage. And all the proteins that control access to DNA, to enable those DNA-templated processes (of gene expression, gene repair and replication) are called epigenetic proteins.

"No single drug has ever cured AML. It is only cured as part of combination drug regimens. The important thing here is how best to use this drug to combat AML and that is a study that is ongoing.

"By itself, IBET has significant promise, but where it will have its best effects, is in combination with other agents and really working that out is what the next few years of our research is going to do," said Prof. Dawson.

Speak to your haematologist about your eligibility for this trial. More information on the trial is available on the ClinTrial Refer ANZ app. This can be downloaded from the App Store (iPhone, iPad users) or Google Play (Android users).

** also known as GSK525762.*

WHAT'S ON NEAR YOU

NEW SOUTH WALES & AUSTRALIAN CAPITAL TERRITORY		
Sydney Metro		
26 Nov	10-11.30am	St George Blood Cancer Education & Support Group
28 Nov	11am-1pm	Westmead Blood Cancer Education & Support Group
18 Dec	10am-12pm	Liverpool Blood Cancer Information & Support Group
Australian Capital Territory & Southern New South Wales		
12 Nov	11am-1pm	Goulburn & Surrounds Blood Cancer Support Group (also 10 Dec)
13 Nov	10am-12pm	Canberra Blood Cancer Support Group (also 11 Dec)
16 Nov	1.30-3.30pm	Moruya & Surrounds Blood Cancer Support Group
27 Nov	5.30-7.30pm	20/30 Chat, Garran
Central Coast		
1 Nov	10.30-11.30am	Orange Blood Cancer Support Group (also 6 Dec)
7 Nov	10.30am-12pm	Dubbo Blood Cancer Support Group (also 5 Dec)
29 Nov	10-11.30am	Gosford Blood Cancer Education & Support Group
Central West & Far West		
13 Dec	10.30am-12pm	Mudgee Blood Cancer Education & Support Group
Hunter		
13 Nov	10am-12pm	Newcastle Blood Cancer Education & Support Group, Shortland
4 Dec	10am-12pm	Newcastle Blood Cancer Education & Support Group, Mayfield
Illawarra & Shoalhaven		
7 Nov	10.30am-12.30pm	Wollongong Blood Cancer Information & Support Group, Fig Tree (also 5 Dec)
12 Nov	10am-12pm	Shoalhaven Blood Cancer Information & Support Group, Bomaderry (also 10 Dec)
Mid North Coast		
19 Nov	10-11.30am	Port Macquarie Blood Cancer Education & Support Group (also 17 Dec)
New England		
13 Nov	10.30am-12pm	Armidale Blood Cancer Education & Support Group (also 11 Dec)
20 Nov	1.30-3.30pm	Tamworth Blood Cancer Education & Support Group (also 18 Dec)
Southern NSW		
28 Nov	10am-12pm	Bowral Blood Cancer & Support Group
QUEENSLAND		
Brisbane Metro		
3 Nov	9am-12pm	It's all about me, Nutrition
17 Nov	9am-3pm	It's all about me, Exercise & Relaxation
1 Dec	10am-12pm	20/30 Chat, Brisbane

QUEENSLAND *(continued)*		
Regional Queensland		
9 Nov	10am-12pm	Coffee, Cake & Chat, Sunshine Coast
15 Nov	10am-12pm	Coffee, Cake & Chat, Gold Coast
7 Dec	10am-12pm	Coffee, Cake & Chat, Toowoomba
SOUTH AUSTRALIA		
Adelaide Metro		
13 Nov	10am-12pm	Women & Blood Cancer Support Group
21 Nov	10.30am-12.30pm	Strathalbyn Support Group
Regional South Australia		
8 Nov	10am-12pm	Southern Community Blood Cancer Support Group
3 Dec	5.30-6.30pm	Mount Gambier Support Group
TASMANIA		
Southern Tasmania		
22 Nov	11am-12.30pm	She Shed and Man Cave, Battery Point
5 Dec	11am-1pm	Hobart Blood Cancer Support Group Xmas Party
11 Dec	10.30am-12pm	Northern Tasmania Blood Cancer Support Group
VICTORIA		
Melbourne Metro		
1 Nov	10.15-11.45am	Bone Marrow & Stem Cell Transplant Support Group, Hawthorn
9 Nov	10-11.30am	Berwick Support Group
22 Nov	10-11.30am	Eastern Melbourne Blood Cancer Support Group
Barwon & South West		
28 Nov	10-11.30am	Warrnambool & South West Blood Cancer Support Group
11 Dec	10-11.30am	Barwon Blood Cancer Support Group
Grampians Region		
11 Dec	10-11.30am	Ballarat Support group
Loddon-Mallee Region		
3 Dec	10-11.30am	Bendigo Support Group
WESTERN AUSTRALIA		
Perth Metro		
19 Nov	1-3pm	Perth Metro Blood Cancer Support Group
Albany		
7 Nov	10am-12pm	Albany Blood Cancer Support Group
Bunbury		
1 Nov	10.30am-12pm	Bunbury Blood Cancer Support Group
Peel		
15 Nov	10.30am-12pm	Peel Blood Cancer Support Group

Leukaemia Foundation
VISION TO CURE
MISSION TO CARE

Join the AML Network closed group on Facebook: facebook.com/groups/AMLLFA/
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

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