ALL news.

For people with ALL & their families

Leukaemia Foundation
VISION TO CURE
MISSION TO CARE

ISSN 2202-2392 (online)

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

Contents

Dr Kylie Mason and Liam in 2009 after her brain tumour diagnosis – a long-term effect of the radiotherapy that was part of her ALL treatment as a teenager.

Kylie's experiences as an ALL patient, doctor and researcher

Leading clinical haematologist and medical researcher, Dr Kylie Mason*, knows what it is like to be taken from your normal day-to-day life after being told you have a life-threatening disease.

This has happened to her twice. Once when she had leukaemia as a teenager, and again when she found out she had a brain tumour, when her son was young.

"Everything else just stops and your normal life becomes less relevant," said Kylie, 44, of Melbourne, mother of two, Liam, nine and Eliza, seven.

"Having leukaemia in my formative years has significantly affected how I approach my life and my thinking about what's important.

"Things can change, so I never put off until tomorrow what I can do today because I don't know what tomorrow brings. I say – hope for the best, but plan for the worst."

Kylie was diagnosed with ALL towards the end of Year 10, when she was 15, in 1988. She'd quit gymnastics "because I wasn't coping so well with it", then noticed she was short of breath, tired and generally unwell.

"Having leukaemia in my formative years... affected how I approach my life..."

She ignored a couple of "episodes" during physical fitness testing at school and didn't tell her parents about collapsing during a 100m sprint, or waking with a mouthful of blood.

"My brother and I were due to go on tour to Perth with the school band when he saw me after a shower" said Kylie, who played clarinet and piano.

"He went to Mum and told her I was covered in bruises and wasn't managing at school.

"I went to the GP, had blood tests, and that afternoon Mum got a call from the doctor who said: 'come in, bring Kylie and a packed bag – she needs to go to hospital'."

Within a few hours Kylie was in the children's cancer ward having a bone marrow test and, just days later, started intensive chemotherapy. She spent the next three weeks in hospital as well as most of the following six months.

Story continued on pages 4 & 5.

New AYA trial for newly diagnosed opens mid-2018

A new national study for adolescents and young adults (AYA) newly diagnosed with ALL begins recruitment mid-year.

The Australasian Leukaemia & Lymphoma Group (ALLG) ALL9 trial follows on from previous clinical trials targeting this age group.

While improvements in outcomes for AYAs have been associated with adopting paediatric-inspired multi-agent chemotherapy, this group still fails to achieve the same outcomes as those seen in children.

"This is likely due to these patients being less tolerant of intensive chemotherapy and differences in disease biology," said Delaine Smith, ALLG CEO.

High-risk genetic mutations are seen more commonly in AYA ALL and are associated with high rates of minimal residual disease (MRD) and poorer outcomes when treated using standard therapy.

The design of the ALLG ALL9 study addresses both issues. It incorporates a novel immune-based therapy (blinatumomab) which is a bi-specific monoclonal antibody with demonstrated activity in relapsed/refractory ALL that is highly efficacious in eliminating MRD.

Ms Smith said the study also would lead to a better understanding of how to successfully incorporate immune-based therapy into standard of care protocols in AYA ALL, as well as having a significant impact on the outcomes.

The trial regimen is expected to reduce toxicity and improve quality of life.

The target accrual is 85 patients over three years, with 65% expected to be aged 16-25. Dr Matthew Greenwood is the lead chief investigator.

Genomic analysis of T-ALL aids development of new therapies

Genomic sequencing analysis of hundreds of people with T-lineage ALL has provided a detailed genomic landscape that will inform treatment strategies and the development of targeted drugs.

Professor Charles Mullighan*, who received funding* from the Leukaemia Foundation in 2013 when he was based in Adelaide, and who now lives in the U.S., led the project's consortium of 39 researchers.

"This first comprehensive and systematic analysis in a large group of patients revealed new mutations that are biologically significant as well as new drug targets that could be clinically important," said Prof. Mullighan of St Jude Children's Research Hospital (Memphis).

"Leukemias typically arise from multiple genetic changes that work together.

"Most previous studies have not had the breadth of genomic data in enough patients to identify the constellations of mutations and recognise their associations," he said.

"The study data is available to researchers worldwide."

The genomes of 264 children and young adults with T-ALL – the largest group ever analysed – identified 106 driver genes; those whose mutations trigger the malfunctions that block normal T-cell development and give rise to cancer. Half of those mutated genes had never been identified in childhood T-ALL.

All the patients received uniform treatment so the researchers could draw meaningful associations between the genetics of their cancer and the response to different treatments, which will enable better diagnosis and treatment of T-ALL with existing drugs.

Significant unexpected findings included more than half of the new targets and mutations being previously unrecognised, and some mutations were exclusively found in some subtypes of T-ALL, but not others.

The new genomic analysis confirmed T-ALL is driven by mutations in known signalling pathways including JAK–STAT, Ras and PTEN–PI3K, and many more genetic mutations were identified in those pathways, offering more targets for drugs to shut down the cancerous cells.

The researchers also found cases where the same T-ALL subtype had mutations in different pathways triggered by the same cancer-causing founding mutation.

"This finding suggests we can target these subtypes with an inhibitor drug for one of the pathways and it's likely to be effective," said Prof. Mullighan.

Professor Charles Mullighan.

Genetic engineering will be used to create mouse models that more accurately reflect human cancer, taking into account the multitude of new mutations revealed in this analysis.

The study is further evidence that through systematically studying a large enough population with careful, detailed genomic analysis, new mutational patterns of collaboration or exclusion across multiple genes unique to each T-ALL subtype can be discovered.

The study data is available to researchers worldwide to explore the newly mapped T-ALL genomic landscape.

* Professor Charles Mullighan and Professor Deborah White, of the SA Pathology and Centre for Cancer Biology, known now as the South Australian Health and Medical Research Institute (SAHMRI) received a $100,000 Grant-in-Aid from the Leukaemia Foundation in 2013 for *Improving outcomes for children with high-risk acute lymphoblastic leukaemia.*

Sequencing study reveals new targeted ALL therapies

The world's most in-depth protein sequencing analysis of T-cell ALL cells by Newcastle biomedical scientist, Dr Matthew Dun, has led to identifying new targeted treatments for children and adolescents.

And the most promising potential treatment is the Australian-developed drug, venetoclax.

Six years ago, when Dr Dun started his post-doctoral studies, he had an idea – "we needed to sequence the proteins rather than just the genes, to give biological meaning to the gene mutations that give rise to cancer".

"Proteins are the functional unit of the cell. They control how the cell functions, moves, communicates, grows, divides, activates and deactivates", said Dr Dun, who went about getting the training, the samples and the collaborators necessary to learn how to sequence proteins.

"And it's kept going from there," said Dr Dun, a Senior Lecturer at the Hunter Medical Research Institute at the University of Newcastle.

He went to Denmark to learn high resolution sequencing techniques, enabling him to sequence all of the proteins from a cell and their activated versions, known as phosphoproteins, that control how a cell grows, divides and replicates – "all the hallmarks of a cancer cell"; uncontrolled proliferation.

"As part of my Cancer Institute NSW research fellowship, I was sequencing proteins from AML patients' leukaemia cells at diagnosis, with the goal of identifying new and better drug targets.

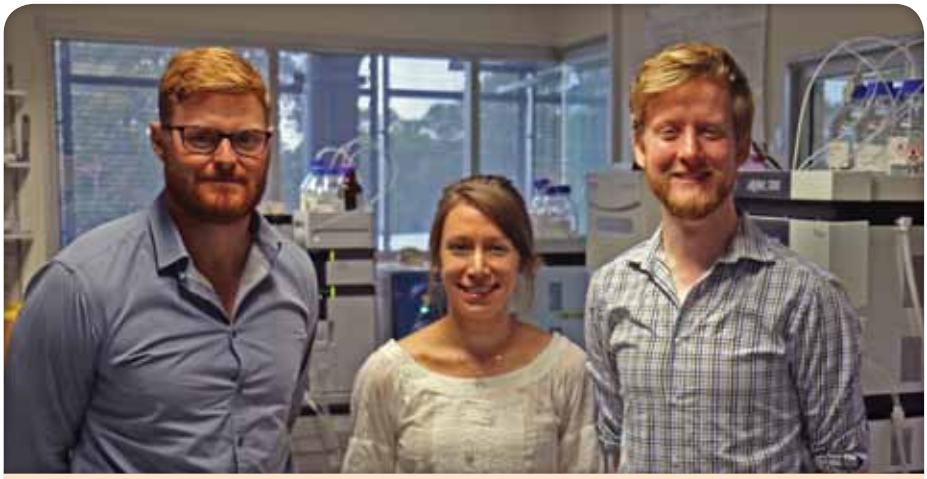
"On diagnosis, here in Newcastle we get leukaemia cells from the bone marrow of patients and subject them to both protein sequencing and gene sequencing.

"I'm trying to identify the proteins in each patient that relate to the gene mutations of the patients, as most of the new personalised therapies target proteins not DNA.

"That's the paradigm for my research group – to search for molecular drug targets and design leukaemia-specific therapies," said Dr Dun who has become an internationally recognised expert in protein sequencing.

"While working at the University of Southern Denmark, a friend and colleague at Belgium's University of Leuven and Leuven Cancer Centre, working in a prestigious ALL research group run by Professor Jan Cools, invited me to view their facilities and to present my data on AML patients," said Dr Dun.

Subsequently, the Belgium group asked Dr Dun to provide a read-out of the proteins in T-cell ALL samples harbouring a recurring genetic mutation to the protein



Dr Matt Dun and his two PhD students, Heather Murray and David Skerrett-Byrne, with the mass spectrometer behind them.

JAK3, known as JAK3-mutant ALL. This mutation causes the developing blood stem cells to stop maturing.

"I agreed to do the deep sequencing and this allows us to dissect and identify the proteins that drive the leukaemia. From that, we could determine how the cancer cells are controlled by JAK3," said Dr Dun.

He won a grant to buy a mass spectrometer, the study started in 2014 and a Belgium PhD student was sent out to Australia for six weeks.

"I sequenced the proteins and phosphoproteins responsible for uncontrolled growth of the leukaemia and analysed the data. The results were quite interesting, and so I was invited to present at a conference in Belgium the following year and met again with the guys at the University of Leuven," he said.

After further data analysis, some new proteins and pathways were identified that could be targeted with drugs not previously thought to be appropriate for ALL.

"Next we validated the drug targets, testing them in the lab and in animal models, and prepared and submitted a paper to the international journal, *Leukemia*.

"That's the paradigm for my research group – to search for molecular drug targets and design leukaemia-specific therapies."

"We received favourable reviews from four expert reviewers and had 100 days to answer their questions," said Dr Dun.

This involved a huge amount of further work looking at additional samples, to substantiate the findings in a separate patient population, before the study data was published last year.

"This is the most in-depth protein and phosphoprotein sequencing analysis of T-cell ALL cells anywhere in the world," said Dr Dun.

"It was an excellent collaboration between my protein sequencing skills and the Belgium team's ALL knowledge and resources.

"This research changes the way we develop new treatment ideas.

"It provides the most information on the proteins and pathways that lie directly downstream of JAK3 and identifies potential therapeutic approaches for ALL patients who don't do so well with standard therapies.

"We know now, as a result of our sequencing, the proteins activated downstream of the mutant JAK3, so we can specifically select proteins that have known inhibitors, to see if we can inactivate the cells in a different way," said Dr Dun.

JAK3 is mutated in 16% of people with T-cell ALL, which is a more rare and more aggressive form of ALL, compared to B-cell ALL, and this patient group does worse than the other sub-types.

"At this stage, all patients receive the same therapy," said Dr Dun.

"Now, we're saying, if we hit patients with JAK3 mutant T-ALL with a JAK3 specific inhibitor plus this novel inhibitor, we're going get a much more beneficial effect and significantly reduce the side-effects if we're targeting two cancer-specific proteins.

"This is called a novel novel therapeutic approach.

"That's what we did in our study and we absolutely ablated [destroyed the function of] the survival the survival of these patients' ALL cells.

"The new step is to use the results in clinical trials for patients who are refractory to all other therapies who harbour JAK3 mutations, to test the combinations of JAK3 inhibitors and other inhibitors, such as venetoclax, and those already FDA approved for other indications."

Dr Dun said protein sequencing will become a standard part of diagnosis.

Continued: Kylie's experiences as an ALL patient, doctor and researcher

"Children and teenagers with ALL these days still have intensive treatment and still get very unwell, but they can spend more time at school and less time in hospital because of the better supportive treatments available now," said Kylie.

"They don't have the bad side-effects like vomiting that we had back in the 1980s because we've got much better antibiotics and antiemetics (nausea and vomiting drugs) now," she said.

Despite spending so much time away from school, Kylie was determined not to repeat year 11 or 12.

"And it was important at the time to keep up with my friends," she said.

"I worked hard, studied over the school holidays and concentrated on what I had to do. And I was clever about my subject choices, like doing clarinet, which only had one lesson a week and gave me flexibility.

"I had wanted to be a Commonwealth coroner. I liked the intrigue and detective idea of this role that investigates deaths," said Kylie, who later found out this was a legal, rather than medical, job.

"Before I got sick I was keen on doing something that was medical science based.

"In year 12, when I was deciding what I should do [career-wise], I had a long chat with my haematologist (who subsequently went on to refer patients to me!) and other doctors and nurses.

"Many felt medicine might be a bit tough going, but everyone was very encouraging, so I gave it a go," said Kylie.

She finished school on time and got into medicine at the University of Melbourne, aged 17 and having finished treatment.

She was 35 when she completed her medical studies and training 18 years later, after a six-year medical degree and 12 years of specialist training and further study, including a PhD.

"That's standard training to become a haematologist, but no one explains this to you when you're at high school!"

Kylie has "quite a few ALL patients", works across all areas of blood cancer, including non-malignant blood disorders, works on several clinical trials, and specialises in late effects, "monitoring people like myself".

"Up to 90% of childhood or adult cancer survivors go on to have significant long-term side-effects of cancer treatment, and among the group I was treated with, many have had second tumours or other major medical problems."

In 2009, Kylie was diagnosed with a brain tumour; the result of radiotherapy that was a standard part of treatment in the 1980s/early-1990s. She had a major operation to remove the tumour, which thankfully was benign, and constantly has her brain, thyroid and heart monitored.

"When I was treated, everyone with ALL was treated the same. It was difficult to determine who was likely to do well or not so well," said Kylie.

"... we can stratify people to receive more or less treatment, or more targeted treatment, according to their risk factors."

"The principles of treatment are similar today, but the doses are varied and radiotherapy hasn't been used in most patients for about 20 years.

"More is known about the cytogenetic and molecular changes that contribute to diseases like ALL. This provides a better idea about prognosis and we can stratify people to receive more or less treatment, or more targeted treatment, according to their risk factors.

"This means there are more survivors and today's survivors are a lot less likely to have some of the long-term side-effects than my treatment cohort," she said.

In mid-1988, after Kylie was diagnosed with ALL: "I'm just starting to lose my hair".

"And very small amounts of disease [minimal residual disease] can be tracked, now, so we can work out if the leukaemia is 100% gone or not, when we may need to try some extra treatment.

"Whereas, we just had to wait to see if we would relapse. They [the doctors] thought we were in remission, not knowing there may be this tiny amount of leukaemia there that they couldn't see.

"I haven't relapsed but I've had a lot of infections," said Kylie.

In one instance, the medication used to treat a lung infection caused a complication, called aplastic anaemia [a rare blood disorder].

"I eventually recovered but there was a time when I was in hospital a lot and quite unwell with no working bone marrow, so I needed a lot of blood and platelet transfusions."

Kylie's career highlight was being on the research team that developed the drug called venetoclax that was recently licensed in Australia for chronic lymphocytic leukaemia.

"It will make a huge difference to thousands of people around the world and may have applications in other blood cancers," she said.

Another "big thing", as part of her PhD research, was the discovery of the protein that is important in keeping platelets alive for longer.

"This has huge implications in understanding platelet biology and the potential to develop drugs for blood clots and heart disease, and for use in blood banking.

"There's lots happening; it's a good time to be a researcher in this area, but far and away the biggest hurdle is funding."

"My overall aim is to make a difference... so patients in the future will have better long-term outcomes."

Kylie, and her fellow chief investigators, received three Grants-in-Aid from the Leukaemia Foundation, in 2010, 2012 and 2014, totaling $300,000.

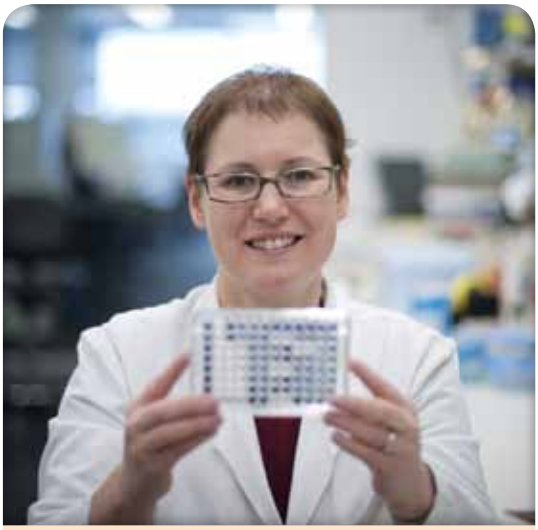
"These grants contributed a big part to the venetoclax program, without which certain parts of the project wouldn't have been done as fast, or at the time they were done, or even done at all. All parts of that project were important and made a difference," said Kylie.

"My overall aim is to make a difference – from my clinical work on a one-to-one patient basis, with a group of patients, through to research – so patients in the future will have better long-term outcomes.

"Everyone – patients, carers and doctors – has such stressful, busy lives.

"There's no doubt that taking some time out, whether it's meditation or exercise or spending some time with your kids and keeping life in perspective is important for everyone.

"I exercise, not too much, and love being outdoors. Because of my busy lifestyle – I'm often on call and have young kids – I need something that gives me time out without punishing me for ignoring it for a long time. For me that's my garden."



Dr Kylie Mason – on the job as a clinical haematologist and medical researcher.

* Dr Kylie Mason is a clinical haematologist and Head of Late Effects (Integrated Department of Haematology, Peter MacCallum and Royal Melbourne Hospital), clinician researcher (University of Melbourne) and honorary senior research fellow (Sydney Medical School, University of Sydney).

New risk scoring system more accurate at predicting relapse

A new Australian-developed risk scoring system for childhood ALL, based on missing DNA fragments or 'microdeletions', provides an early warning that treatment may not be working.

This scoring system could make a big difference to the success of childhood ALL treatment.

In an international study that tested 475 children with non-high-risk B-cell precursor ALL (BCP-ALL) at six hospitals in Australia and New Zealand, two genes were found to be important predictors of relapse – IKZF1 and P2RY8-CRLF2.

Both deletions have a significant effect on relapse-free survival, event-free survival, and overall survival, and were even more effective when combined with two other test results – minimal residual disease (MRD) and National Cancer Institute (NCI) risk.

A risk stratification score (RS0, RS1 or RS2+) that used the presence of either or both deletions, as well as MRD and NCI was then developed to calculate a risk score for each patient of '0' (no risk factors) to '2+' (several).

When combined, these measures identified a sub-group of patients previously classified as medium-risk who were likely to relapse.

Children with a '2+' score were found to be most likely to relapse within seven years after treatment started, while those with a '0' score were least likely.

The risk score was validated in an independent cohort of medium risk BCP-ALL patients from The Netherlands.

The findings were published in the *British Journal of Haematology*.

The improved stratification provides doctors with a more accurate way to categorise patient risk than the current approach.

If the new risk score system is adopted in the future, doctors could give children with a 2+ risk more intensive treatment, with the aim of improving their survival.

According to co-author Dr Toby Trahair, a paediatric haematologist and oncologist at the Children's Cancer Institute and Kids Cancer Centre, it meant some children would now receive more intensive treatment earlier.

"It's much better to prevent relapse by treatment rather than having to treat somebody at relapse," said Dr Trahair.

"... more accurate risk prediction will ensure the best therapy and the lowest chance of relapse."

"So in the next generation of clinical trials and treatment for children with ALL, we will build in strategies to try and identify these higher risk patients at early time points," said Dr Trahair.

Dr Toby Trahair.

He said the new risk model relied on laboratory techniques that were easy to use and would be easy to implement.

"The technology used to identify small deletions is increasingly available and this paper is important because it shows we need to be looking for these microdeletions in ALL," said Dr Trahair.

"Much more accurate risk prediction will ensure the best therapy and the lowest chance of relapse.

"It's an exciting new area. Some patients with microdeletions have genetic changes that are targetable. A sub-group we are starting to call Philadelphia-like leukaemia often has targetable genetic changes, some of which respond to targeted treatments."

CAR T-cells – 'living drugs' that target cancer cells – approved in the U.S.

Last September, the first-ever CAR T-cell therapy to receive FDA approval in the U.S. was tisagenlecleucel (Kymriah™) for the treatment of relapsed B-cell ALL in children and young people.

The following month, the second immunotherapy treatment to be FDA-approved, axicabtagene ciloleucel (Yescarta™), was for adults with another blood cancer – aggressive forms of non-Hodgkin lymphoma.

These and similar gene therapies under development have been heralded as game-changers that have reshaped thinking about cancer treatment

CAR-T (chimeric antigen receptor T-cell) immunotherapy was pioneered by Dr Carl June of the University of Pennsylvania and involves removing a patient's immune T-cells from their body and reprogramming the cells to find and kill cancer cells.

Until now, the dominant strategies for cancer treatment have been slash, burn and poison (surgery, radiation and chemotherapy) which destroy tumours but also cause a lot of collateral damage, and the tumour often returns.

In contrast, the immune system is designed by nature to identify and destroy enemy cells only and to keep on destroying them while minimising collateral damage.

These new immunotherapy strategies don't target cancer itself but work on the body's ability to fight it. Administered in tablet or IV form, they trigger the immune system to fight cancer cells while keeping healthy cells intact.

CAR T-cell trials have generated impressive early results and considerable promise in people with blood cancers. The global 25 centre ELIANA study, resulted in 83% of children with ALL who had relapsed or failed chemotherapy achieving a complete or partial remission three months after their infusion. And after 12 months, 79% of them were still alive.

The treatment involves collecting T-cells from a simple blood draw, then genetically engineering them in a laboratory to produce chimeric antigen receptors (CARs) on their surface. These engineered CAR T-cells are then infused into the patient.

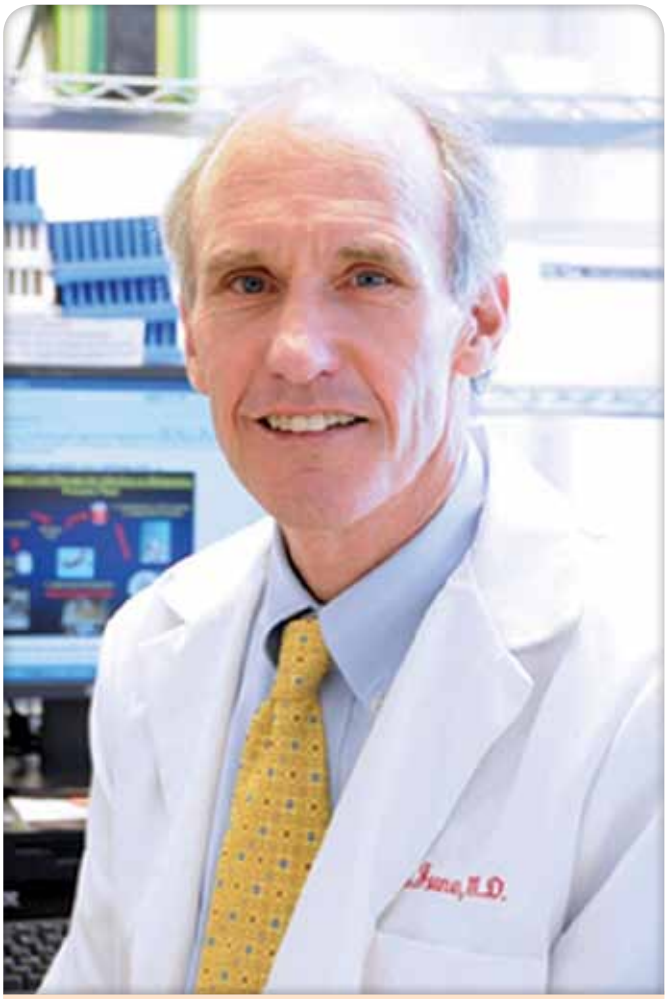
The CARs enable the T-cells to specifically target and eliminate the cancer cells, just as T-cells would normally target and eliminate virus-infected cells. Finding targets specific to each cancer is the key.

Haematologist and cancer researcher, Dr Kenneth Micklethwaite, of Sydney's Westmead Institute for Medical Research (WIMR), who is leading a trial for a similar therapy in Australia, described immunotherapy as "revolutionary".

"The results have been remarkable in trials overseas. Patients with very active leukaemia, who would have no other options, have gone into a complete remission within a month after receiving the CAR T-cells," he said.

A challenge in using CAR T-cells is to make the technology accessible on a large scale, given that each patient needs their own batch of T-cells engineered; a process that takes several weeks.

Funding access to such innovative therapies requires a paradigm shift. The Leukaemia Foundation is actively working with industry and government to ensure Australians can access this treatment once approved for use here.



CAR T-cell therapy pioneer, Dr Carl June.

Researchers at WIMR, led by Dr Micklethwaite, are trialling a new way of manufacturing the CAR T-cells in Australia that bypasses the viral delivery and will drastically reduce the cost of the therapy.

The Phase I study, called CARTELL, is testing the safety of the WIMR-developed CAR T-cells in patients with persistent and relapsed B-cell leukaemia and lymphoma who have previously had a stem cell transplant.

"What we've been working on over the last five years is a simple and inexpensive way of making these CAR T-cells using a non viral system of genetic modification. This involves taking two pieces of DNA and cutting and pasting the receptor into the DNA," said Dr Micklethwaite.

"We've got a simple and robust process that enables us to make these cells quickly for around $10,000 per patient – a fraction of the cost of the CAR T-cell products approved by the FDA in the U.S.

"If we can provide a less expensive (and) as effective alternative that is locally produced, it gives us some degree of control over the process and will provide this therapy to patients sooner."

There are risks with these 'living drugs'. Complications seen in trials overseas include cytokine release syndrome, where the T-cells become over activated and release inflammatory chemicals that damage healthy cells.

The treatment also can cause neurotoxicity that damages the nervous system and can cause confusion, memory loss, convulsions and other cognitive dysfunctions.

The winning strategy for immunotherapy will likely involve a combination of the old and the new, such as radiotherapy to rouse the immune system combined with immuno-drugs. Tailoring the treatment to each patient also is crucial because each cancer requires a different plan.

Lauren Krelshem was the first Australian to be treated with this new immunotherapy. Read how she is travelling, almost two years after her CAR T-cell infusion in 2016 – see adjoining page.

Lauren's well, grateful and excited about her future

It's nearly two years since Lauren Krelshem became the first person in Australia to have CAR T-cell therapy on an international Phase II paediatric clinical trial in Melbourne.

"That was the only treatment left for me," said Lauren, 23. She has lived with refractory B-cell ALL since her diagnosis as a seven year old.

"If it wasn't for this treatment I wouldn't be here… that's the truth of it. I think it's amazing – the idea that this [therapy] could become something bigger than just treating patients with ALL.

"The fact it worked is such a blessing and I'm so grateful I have this chance to live life where it takes me now," said Lauren, rather than living based on life and death decisions about treatment options.

"Now every day is about freedom and choice which is sort of scary but also really enlightening and exciting."

In June last year, Lauren chose to move out of home and interstate, leaving her family in Adelaide for Melbourne. This met her medical obligations on the trial, which continues until March 2020, and her career aspirations to be an actor.

"I wanted to explore my own growth a bit more, but didn't realise how hard the recovery process would be," said Lauren.

Not only is she dealing with fatigue and a suppressed immune system which means there are times when she can't work, she is learning how to be financially independent and managing living on her own in a share house.

Lauren went into remission just a month after being infused with her own genetically modified T-cells (known now as Kymriah™) in February 2016.

The first week after receiving the lifesaving treatment was "horrible, like you've caught the worst flu you can ever imagine".

"If it wasn't for this treatment I wouldn't be here… "

"After three weeks I was feeling more alive again, not like chemo where you have good and bad days. And once I felt better, I continued to feel better every single day, and there were no side-effects, like graft versus host disease," she said.

While there are risks involved in having this highly personalised CAR T-cell therapy, Lauren has had no toxic side-effects.

She had to return to Melbourne, from hometown Adelaide, once once a month for blood tests and a two-hour infusion of immunoglobulin, and for the first year, she had a bone marrow biopsy every two months.

Her haematologist visits have just dropped back to once a quarter and dosages of her "recovery drugs" – antifungal, antibacterial, antiviral tablets and hormone replacement therapy – are slowly being reduced.

Now Lauren takes 10 tablets a day and, at this stage, the monthly immunoglobulin infusions are destined to be life-long. They replace the cancerous cells that are killed by the highly specific therapy.

"If I get a temperature of 38 degrees, I have to be admitted to hospital for antibiotics," said Lauren. This has happened every two to three months until recently.

"My blood counts are coming slowly back to normal. My haemoglobin is normal but my platelets and neutrophils are still playing catch-up.

"Melbourne is definitely the place I need to be right now," she said.

"I've made some friends, gone out for a few drinks, dinner, and I love the brunches, and I've been to the beach."

Now Lauren's health is "extremely better" and she's settled into Melbourne, she plans to "live happily" and get on with her goals, and top of the list is to start acting classes.

Lauren Krelshem: "my favourite character-type shot. It's very me… the curiosity, inner strength and determination with that hint of sadness or struggle/discomfort is what I like about it".

NEW SOUTH WALES & AUSTRALIAN CAPITAL TERRITORY

Sydney Metro

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

Australian Capital Territory & Southern New South Wales

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

Central Coast

22 Feb	10-11.30am	Gosford Blood Cancer Education & Support Group (also 29 Mar, 26 Apr, 31 May, 28 Jun, 26 Jul, 30 Aug)

Central West & Far West

1 Feb	10.30am-12pm	Orange Blood Cancer Education & Support Group (also 1 Mar, 5 Apr, 3 May, 7 Jun)
7 Feb	10.30am-12pm	Dubbo Blood Cancer Education & Support Group (also 7 Mar, 4 Apr, 2 May, 6 Jun)
8 Feb	10.30am-12pm	Mudgee Blood Cancer Education & Support Group (also 13 Apr, 14 Jun, 9 Aug, 11 Oct, 13 Dec)

Hunter

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

Illawarra & Shoalhaven

28 Mar	10am-12pm	Bowral Blood Cancer & Support Group (also 23 May, 25 Jul, 26 Sep, 28 Nov)

Mid North Coast

19 Feb	10-11.30am	Port Macquarie Blood Cancer Education & Support Group (also 19 Mar, 16 Apr, 21 May, 18 Jun, 16 Jul)
22 Feb	11.30am-1pm	Coffs Harbour Blood Cancer Education & Support Group (also 22 Mar, 26 Apr, 24 May, 28 Jun, 26 Jul)

New England

13 Feb	10.30am-12pm	Armidale Blood Cancer Education & Support Group (also 13 Mar, 10 Apr, 8 May, 12 Jun, 10 Jul, 14 Aug)
20 Feb	1.30-3.30pm	Tamworth Blood Cancer Education & Support Group (also 20 Mar, 17 Apr, 15 May, 19 Jun, 17 Jul, 21 Aug)

SOUTH AUSTRALIA

Adelaide Metro

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

Regional South Australia

5 Feb	TBA	Mount Gambier Support Group (also 26 Mar, 4 Jun, 6 Aug, 8 Oct, 3 Dec)
13 Feb	10am-12pm	Port Lincoln Support Group (also 10 Apr, 12 Jun, 14 Aug, 9 Oct)

TASMANIA

Northern Tasmania

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

Southern Tasmania

15 Feb	11am-12.30pm	She Shed, Battery Point (also 15 Mar, 19 Apr, 17 May, 21 Jun, 19 Jul, 16 Aug, 20 Sep, 18 Oct, 22 Nov)
11 Apr	11am-1pm	Hobart Blood Cancer Support Group (also 8 Aug, 8 Sep, 10 Oct, 14 Nov, 5 Dec)
24 Apr	11am-1pm	Cooking for Chemo (also 13 Jun)

VICTORIA

Metro Melbourne

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

Barwon & South West

6 Feb	10-11.30am	Barwon Blood Cancer Support Group (also 6 Mar, 17 Apr)
21 Feb	10-11.30am	Warrnambool & South West Blood Cancer Support Group

Grampians

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

Hume

8 Feb	10-11am	Shepparton Blood Cancer Support Group
13 Feb	10-11am	Wangaratta Blood Cancer Support Group
27 Feb	10-11am	Echuca Blood Cancer Support Group

Loddon/Mallee

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

Join the ALL Network closed group on Facebook: https://www.facebook.com/groups/ALLLFA/
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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