

DESIGN VISUALS

Area 1: Fence panels

Area 2 and 3: Wall vinyl

Area 4: Column Vinyl

Area 1: Fence panels (02)

Proposed Artwork

Artwork dimensions

Width: 1422.4 mm

Height: 1056.6 mm

Height from concrete wall

1000mm

Artwork material

Dibond print on aluminium

Type sizes

Title questions: 300pt

Intro: 80pt

Body: 50pt

Max Letter Height

75mm

Typeface

Marfa Black

Marfa Bold

Marfa Regular

WHY DO WE HAVE A NEW FLU JAB EACH YEAR?

In 1933, scientists at the National Institute for Medical Research discovered that flu was caused by a virus. Four years later, they showed that all flu viruses are not the same.



When early vaccine trials began, researchers grew flu in hens' eggs (because viruses can only reproduce in living things). Their studies suggested each annual outbreak was caused by a different strain of the flu virus.

To keep pace, the Worldwide Influenza Centre opened in 1948. Researching, collecting and sharing information about active flu strains, the Centre enabled vaccine manufacturers to target jabs against the right strain every year.

Today...

The Centre is now located in the Francis Crick Institute (the building behind you), where it plays a critical role within a global surveillance system coordinated by the World Health Organization. Centre researchers studying the physical structures of active flu strains are also advancing our understanding of COVID-19 variants.

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Area 1: Fence panels (03)
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WHAT HAPPENS TO OUR BODIES AT EXTREMES?

Before 1953, every attempt to climb Mount Everest had failed. Tenzing Norgay and Edmund Hillary finally made it to the top after a scientist investigated how climbers were affected by high altitudes.



Griffith Pugh from the National Institute for Medical Research designed the 1953 expedition's schedule for acclimatising to altitude, and advised when and how oxygen tanks should be used.

The first person to understand that dehydration slows oxygen movement around the body, Griffith recommended climbers drank six times more water than usual. He even designed and tested the warm, lightweight boots worn on the first successful climb.

Today...

The National Institute for Medical Research is now part of the building behind you: the Francis Crick Institute.

Inside, Peter Ratcliffe studies how cells sense and respond to low oxygen levels, a situation seen in anaemia, lung diseases and some cancers. In 2019, Peter won a Nobel Prize for this work.

Area 1: Fence panels (04)
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WHY ARE SOME INFECTIOUS DISEASES SO STUBBORN?

In the 1950s, doctors realised that a previously successful drug targeting leprosy wasn't working properly any more. Finding out what had happened transformed leprosy into the curable disease it is today.



In 1964, working with the Sungai Buloh leprosy settlement in Malaysia, researchers from the National Institute for Medical Research proved that the leprosy bacterium had changed to become resistant to the standard drug treatment.

The findings led the World Health Organization to recommend a 'cocktail' of several drugs for all new patients. Thanks to this multidrug therapy, leprosy has been all but wiped out in many countries. Where it still exists, it can be cured.

Today...

The National Institute for Medical Research is now part of the Francis Crick Institute (the building behind you).

Inside, Luiz Carvalho studies how antibiotics work against tuberculosis and how tuberculosis bacteria resist treatment. Luiz's research may lead to more effective drugs and more manageable treatment regimes for tuberculosis patients.

Area 1: Fence panels (05)

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WHAT IF WE COULD MAKE A WORKING, WEARABLE ORGAN?

In the 1970s, the Sony Walkman music player provided inspiration for another portable device: a lightweight, wearable pump that transformed the lives of people with diabetes.



Diabetes is a condition in which your body can't make enough insulin, or your insulin doesn't work properly. The pump, developed at the National Institute for Medical Research, provided regular doses of this essential hormone, mimicking the processes of a human pancreas.

Small enough to carry around (about the size of one of today's mobile phones), this technology gave people more control over their condition – and their lives – than ever before.

Today...

The National Institute for Medical Research is now part of the Francis Crick Institute (the building behind you).

Inside, Vivian Li is using new technologies to make lab-grown intestines that could be used to replace damaged intestinal tissue in patients with certain digestive disorders.

Area 1: Fence panels (06)

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WHY DOESN'T EVERYONE GET CANCER?

In 1979, David Lane from the Imperial Cancer Research Fund discovered a protein that – when working properly – stops cancers from growing.



While investigating a virus known to cause cancer in rodents, David discovered p53, a protein that usually prevents tumour growth. We now know that p53 is damaged or missing in most human cancers.

Nicknamed the 'guardian of the genome', this superhero protein continually monitors our cells. It sounds the alarm when damage is detected, calls in reinforcements and sees off cells that can't be repaired. The discovery of p53 has sparked many new lines of research, which may lead to game-changing therapies.

Today...

The Imperial Cancer Research Fund laboratories are now part of the building behind you: the Francis Crick Institute.

Inside, Karen Vousden's lab studies how p53 operates and how it can be controlled. Some of this research involves developing molecules that could be used to reactivate p53 – and its therapeutic benefits – within tumours.

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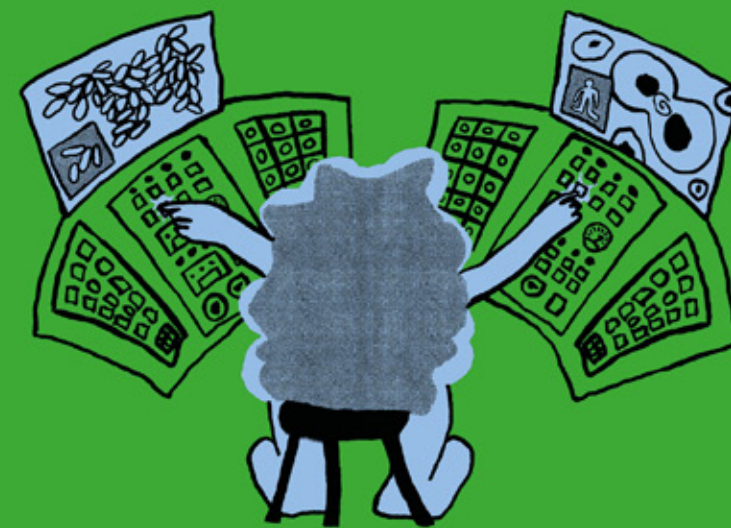
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WHAT COULD WE POSSIBLY HAVE IN COMMON WITH YEAST?

In the 1980s, researchers discovered something we share with yeast and almost every other living thing: a gene that tells each cell in our bodies when to divide and grow.



We all start life as a single cell, which grows and then divides over and over again. In 1987, Paul Nurse and Melanie Lee from the Imperial Cancer Research Fund discovered the human version of a gene known to trigger this process in yeast. Scientists soon realised that the process of cell division is amazingly similar between yeast, humans and everything in between.

Because it's easier to study complex processes in simple organisms like yeast, scientists could answer important questions about how cells grow in humans much more quickly, saving years of research.

Today...

The Imperial Cancer Research Fund laboratories are now part of the building behind you, where Paul (who won a Nobel Prize for his work) is now Director.

Inside, Frank Uhlmann uses yeast to study how cells organise and separate their DNA when they divide, helping us understand how this process might go wrong in human development and disease.

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Area 1: Fence panels (08)

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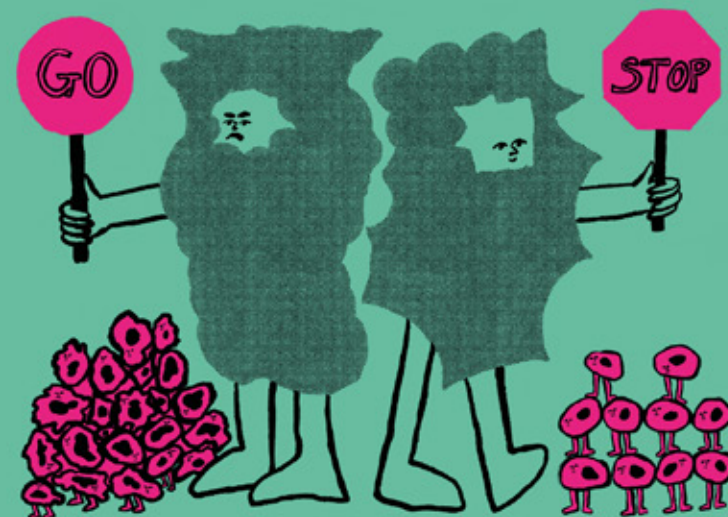
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HOW CAN WE IMPROVE CANCER TREATMENT?

In the 1980s, scientists discovered that the proteins transmitting signals to help our bodies heal could also cause cancer. This revolutionary news led to a new era of personalised cancer drugs.



Julian Downward's and Mike Waterfield's discoveries at the Imperial Cancer Research Fund laboratories showed genes involved in wound healing can mutate to cause the uncontrollable growth we see in cancer. Scientists realised that turning these genes off could stop cancer cells from growing.

Fourteen years later, breast-cancer drug Herceptin was launched. Herceptin binds to and inactivates a protein needed by tumours, inhibiting their growth, and is just one of a range of targeted cancer therapies based on this principle.

Today...

Some of the Imperial Cancer Research Fund's work carries on in the building behind you, where Julian still works.

Inside, Ilaria Malanchi and Erik Sahai study how interactions between cancer cells and healthy cells affect tumour growth. Their work could lead to other improved cancer therapies.

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Area 1: Fence panels (09)

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WHAT DECIDES THE DEVELOPMENT OF TESTES OR OVARIES?

In 1990, researchers at the Imperial Cancer Research Fund and the National Institute for Medical Research together tracked down a gene that plays a pivotal role in the initiation of sex development.



Though scientists knew that embryos with a Y chromosome usually went on to develop testes and then other male organs, no one knew what sparked this process until Peter Goodfellow and Robin Lovell-Badge identified a likely candidate gene, which they called SRY.

Robin and Peter made headline news by showing that if this Y chromosome gene was introduced into XX mouse embryos, which normally develop ovaries, they developed testes instead. In humans, the activity or absence of SRY during pregnancy has a similarly significant impact.

Today...

The Imperial Cancer Research Fund and National Institute for Medical Research are now part of the Francis Crick Institute (the building behind you), where Robin still works.

Inside, James Turner studies genes on the X and Y chromosomes, exploring their role in development, disease and infertility.

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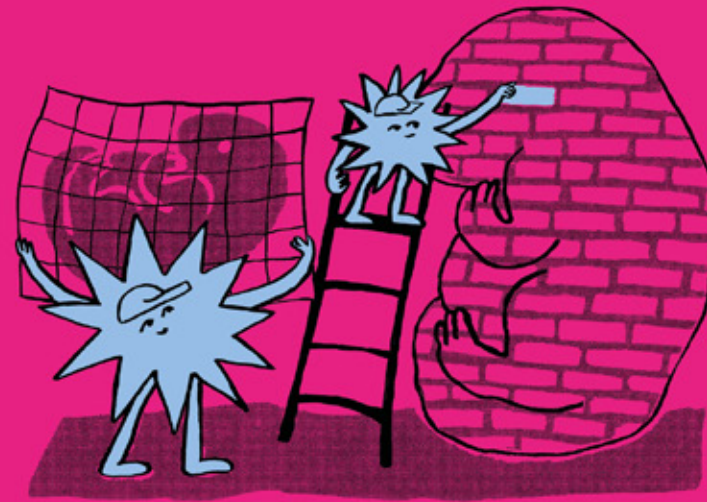
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HOW DOES AN EMBRYO KNOW ITS HEAD FROM ITS TAIL?

In the 1990s, a steady-handed scientist combined skills in microsurgery and genetics to overturn our understanding of how embryos develop.



Cells in the early embryo must decide whether to become bone, muscle, or an organ, and something must tell each cell where to go and which part of the body to make.

For 70 years, scientists believed that cells inside the embryo guided this entire process. Then, Rosa Beddington from the National Institute for Medical Research made a surprising discovery: she showed that tissue outside the embryo instructs the head to form. By studying mice, Rosa showed that faulty genes in this tissue could have severe consequences for the developing embryo.

Today...

The National Institute for Medical Research is now part of the Francis Crick Institute (the building behind you), where Kathy Niakan's lab is mapping the complex web of factors that control the fate of each cell shortly after a human egg is fertilised.

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Area 1: Fence panels (11)
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WHY ARE SOME PEOPLE MORE LIKELY TO GET CANCER?

In 2005, a discovery about how broken DNA is repaired helped explain why some people are at greater risk of developing certain cancers.



Keeping our DNA from harm is vitally important, and so our bodies constantly monitor it for damage. Usually, any problems are spotted and automatically repaired, but if the repair process goes wrong we may develop cancer.

Steve West's research at the CRUK London Research Institute showed how a protein called BRCA2 directs other proteins to repair broken DNA. People with a faulty copy of the BRCA2 gene may end up with damaged DNA, which puts them at increased risk of developing particular types of cancer.

Today...

The CRUK London Research Institute built a world-renowned programme for DNA repair research, under the leadership of Nobel Prize laureate Tomas Lindahl. This continues in the building behind you, where Steve still works.

Fellow Crick scientist Simon Boulton also explores how cells repair DNA damage. This work helps us understand more about cancer, ageing and infertility.

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