A new date for handover of the Pears Building by Willmott Dixon to the Royal Free Charity and UCL has been agreed.

Provided there are no new issues posed by the pandemic, it’s hoped to hand over the new home for the UCL Institute of Immunity and Transplantation by the end of the year, provided there are no more delays caused by the pandemic.

On pages 2-4 we describe the crucial research work underway among our research teams as they join scientists across the world to help unravel the workings of the coronavirus and find a vaccine.
The project has adapted well to the additional health and safety requirements associated with lockdown. Initially the number of workers had to be reduced to accommodate social distancing rules but following an increase in welfare facilities giving more space, the site is now running at full capacity and we are achieving the required turnover.

The scaffolding on the west side has been removed, revealing the building façade. Much of the site hoarding has also been removed and replaced by wire mesh fencing to allow the landscaping work to start.

Discussions are underway with Camden Council regarding its refurbishment of the public footpath between the new building and Hampstead Green – when this starts, the footpath will be temporarily closed.

Inside the building, work on the laboratories has proceeded as planned with most of the flooring now down and installation of the laboratory furniture well underway.

The frame of the feature staircase has been installed in the atrium as well as the atrium pods - square meeting areas that are suspended in the atrium.

Most of the engineering plant has been installed on level 5 and on the roof, and the solar power equipment is expected imminently.

The installation of the external louvres has been delayed by the need to amend the louvre fins from wood to aluminium (ie non-combustible) following the Grenfell Tower fire and permission is being sought from Camden Council under a section 73 application. We hope to have the application approved shortly.

The remaining tower crane has now been dismantled and the roof is watertight – a key milestone.
IIT research re Co-19

David Lowe, consultant immunologist

**Usual area of research:** primary immune deficiency

**Covid work:** searching for an early treatment

There has been much coverage of the exciting discovery that a cheap, readily available steroid can dramatically reduce the risk of death in the sickest patients. But Dr Lowe is focusing on a drug that might be effective earlier in the disease, keeping patients out of hospital.

“In my normal research into treatments for patients with immune deficiencies and chronic infections, I have used an antiviral called favipiravir. When Covid came along various research teams, including ours, started looking at whether it could help and preliminary studies in China indicate that it might.”

The antiviral remdesivir has also been found to be useful for treating Covid but it must be given intravenously and is therefore used only for patients who are in hospital.

“Antivirals work best if we get them into the patient early and this is even more true for Covid-19 patients as the virus replicates particularly quickly in the first few days of infection when they’re not feeling that ill but may go on to become very sick and need admission to hospital.”

He has secured a grant to do a randomised clinical trial comparing favipiravir, an HIV drug, the combination of both drugs and a placebo. “The measure will be the amount of virus in the saliva after five days of treatment. If it works, we would hope to roll it out to high-risk groups in the hope of preventing serious deterioration in their condition.”

Claudia Mauri, professor of immunology

**Usual area of research:** the role of regulatory B cells in autoimmune disease

**Covid work:** What lessons can be learned from children who have been exposed to the virus?

It is well known that most children who contract the virus develop a very mild form of Covid-19.

Prof Mauri is looking at the role of B-cells, which produce the antibodies which protect us from infections and which influence the body’s immune response to a threat such as a virus. She thinks they may help explain why people of different ages respond so differently.

“It is very clear that every decade adds more risk of developing severe disease,” said Prof Mauri. “We want to understand what it is in the B-cells which allows them to clear the infection, which is missing in the elderly.”

She and her team are working on the hypothesis that a child’s immune system is very “naïve” and as a result their immune system can mount a more rigorous response to the threat.

“What may happen is that as we age, the immune system generates different antibodies in response to a variety of infections, they become more specialised and won’t necessarily promptly react to a new virus like Covid-19. But children’s antibodies are less specialised and can fight it off more effectively.”

Prof Mauri’s team is part of a consortium of British, Italian, African and Chilean researchers looking at how age differences affect the risk of developing severe disease.

“There will be more crises like these. This is the third in the past 10 years and there are obviously questions we have to answer before we can deal with them effectively. For example, if the reservoir is in bats, why doesn’t it kill them? How do they clear the infection?”

“The level of collaboration between scientists across the world at the moment is unprecedented and I am really hopeful we’re going to find some answers more quickly than we would in normal circumstances.”

Mala Maini, professor of viral immunology and consultant specialising in viral hepatitis

**Usual area of research:** novel immunotherapy for hepatitis B virus and liver cancer

**Covid work:** How T-cells affect response to Covid-19 and how cholesterol and statins may affect outcomes

Prof Maini and her team work on immune responses to viruses – usually hepatitis B but now also Covid-19 – and focus on how the immune system, particularly T and B-cells, respond to threats.

“T and B cells can either control viruses or sometimes they fail to do this and instead contribute to the disease by initiating organ damage,” said Prof Maini.

“We want to look at the differences in T-cell responses specifically able to target Covid-19 in those who are exposed to the infection, those who get infected but stay well and those who get very ill.”

She and her team are also very interested in how high cholesterol levels, and drugs used to treat the condition, may affect how patients respond to the virus.
Laura McCoy, Medical Research Council career development fellow

Usual area of research: how antibodies work against viruses, particularly in patients with HIV infection.

Covid work: studying how good coronavirus antibodies are at preventing re-infection.

Focusing on HIV antibodies, Dr McCoy’s laboratory aims to understand how antibodies work against viruses. Turning to the Covid-19 crisis, she and her team have developed methods to measure the amount of coronavirus antibodies in the blood.

“These tests measure antibodies that stick to the outer surface of the virus and so have the potential to block the virus from infecting human cells,” said Dr McCoy. “We’re now working on understanding how good these antibodies are at preventing infection in a test tube and investigating whether we can use some of the antibodies already identified in a test to detect viral proteins in saliva.”

Her team have used their test to measure the levels of coronavirus antibodies in NHS workers and will continue to measure antibody levels in the same group of patients over the next three months.

“We’ll also measure how good their antibodies are at blocking infection in the laboratory. If people in the study are re-infected by coronavirus then we can look at the number and quality of antibodies and use this information to understand whether antibodies can protect from infection in the real world as well as in the lab. This will be crucial to the evaluation of vaccines.”

She is most interested in understanding the “rules” of antibody development. Is the type of antibody response you make driven by the shape of the viral protein, by the amount of virus you are infected with or by where in the body you are infected, for example?

“There has been a huge focus on antibodies against coronavirus during the emergency and I hope our work will help the medical community to halt the spread of the virus and to better define the rules of what makes a good antibody response against other viruses.”

Matthew Reeves, molecular virologist

Usual area of research: molecular virology and the immune response.

Covid work: The measurement of antibody responses to new vaccines to decide which are likely to work.

Matthew Reeves’ lab is interested in vaccines, more than 100 of which are in development in response to the pandemic.

“Many vaccines are successful because they can show the immune system how to make antibodies to prevent virus infection before the person is infected with the virus,” said Dr Reeves. “So, along with colleagues, we are trying to develop ways to rapidly measure these antibody responses in response to new vaccines to help us decide which vaccines are going to be the most promising.”

He and his team have applied this approach to analyse the antibody response made by people infected with SARS-Cov-2 – the coronavirus. “We know people have very different outcomes when infected with the virus and that is likely to be linked to how their immune system responds to the infection.

“We can learn from those people who are controllers of the infection what an optimal immune response may look like and use that to help us design vaccines.”

Working with colleagues at UCL, Sussex and Bern, Switzerland, he and his colleagues aim to have the evidence to support the further development of a vaccine by September. “We’ll then be in a position to analyse how people respond to the vaccine. What we want to do is demonstrate that the vaccine induces the ‘right’ antibody response.”

He said that one of the most interesting aspects of the emergency to a scientist is what made this virus so good at jumping the species barrier. “Viruses often try to jump between species but usually the jump into a new species is too big and the virus fizzes out because it can’t replicate and transmit effectively. But this virus has achieved this and so the question is how?”

Lucy Walker, professor of immune regulation

Usual area of research: why the immune system sometimes attacks our own bodies, eg in type 1 diabetes and arthritis.


The widely differing ways in which people have responded to infection by the SARS-CoV-2 virus is at the heart of much research into this new disease and the work by Prof Walker and her team is also focused on this aspect.

She is applying her knowledge of autoimmune conditions, particularly type 1 diabetes and arthritis, which arise when the immune system mistakenly attacks healthy cells in the body.

“Having an effective immune response is important for fighting the coronavirus that causes COVID-19,” said Prof Walker. “In many people, the immune system stops the virus in its tracks, sometimes with few or no symptoms. However, in others, the immune response becomes dysregulated, in other words, it gets out of hand.”

Co-operation between different immune cells is key for fighting the virus. But if these interactions are not properly regulated, the immune response can cause damage to multiple tissues, as seen in patients with severe Covid-19.

“In this group of patients, it’s the body’s response, rather than the virus, that ultimately causes the harm. My group would like to understand the dysregulated immune response in Covid-19, drawing on our expertise in autoimmune diseases.”