Name	DAVID LV BAUER
Position	Group Leader (1st 6)
Year joined *(Crick or founder institute)*	2020

Career History

2009-2013: DPhil, Clinical Medicine (Genetics), University of Oxford, Wellcome Trust Centre for Human Genetics & St Edmund Hall
2014-2014: US National Science Foundation Postdoctoral Fellow
2016 – 2020: Postdoctoral Research Associate, Sir William Dunn School of Pathology, University of Oxford
2020 - Present: Group Leader, the Francis Crick Institute

Major Awards, Honours and Prizes

Adjunct Fellowship, Linacre College, University of Oxford, 2016 – 2020

Award for Excellence, University of Oxford, April 2019

EPAC Junior Research Fellowship (JRF), Linacre College, University of Oxford, 2013 – 2016

Rhodes Scholarship, 2009 – 2012

Nuffield Dept. of Clinical Medicine Prize Studentship, University of Oxford, 2009 – 2013

Membership of external committees, editorial boards, review panels, SABs etc

Member, UK SARS-CoV-2 Genotype to Phenotype National Virology Consortium (G2P-UK).

Member, COVID-19 Genomics UK Consortium / G2P-UK Working Group.

Board of Directors, Creativity Foundation, 2008 – Present.

ARISE Scholarship Committee, Louis August Jonas Foundation, 2009-Present.

Lab Name	***RNA Virus Replication Laboratory***

Research programme and achievements

We study how RNA viruses replicate in order to understand how they work and to find better ways of treating diseases such as influenza and COVID-19.

Viruses are infectious parasites that can cause disease. Viruses cannot grow on their own — they must infect a 'host' cell in order to reproduce, and often cause disease in the process. All forms of life, from bacteria and fungi to plants and animals, are infected by at least one type of virus.

RNA viruses store their genome as RNA, instead of DNA. They cause a wide range of diseases in humans, from the common cold to more severe illnesses such as gastroenteritis, influenza, Ebola virus disease, measles and COVID-19.

The use of RNA (instead of DNA) by these viruses gives them unique properties. First, they all have a gene for an enzyme to copy the RNA genome, called an 'RNA-directed RNA polymerase' in the case of riboviruses. This enzyme is not found outside of these viruses, which makes it an attractive target for antiviral drugs. Second, unlike DNA, RNA can adopt specific shapes that allow the genome itself to carry out structural and enzymatic functions directly, even though it is not a protein or an enzyme in a conventional sense. All RNA viruses exploit this unique property of RNA in one way or another. At the same time, hosts (including humans) have evolved protein sensors to detect unique features of viral RNA in order to activate the immune system and fight RNA virus infections.

In order to better understand how RNA affects RNA virus replication, our laboratory borrows tools from biochemistry, molecular biology, virology, genomics, and bioinformatics. We also use these tools to design new types of antiviral drugs that can be used to block RNA virus growth, which is especially useful against new or emerging viruses for which conventional drugs or vaccines may not be available.

Research outputs

Wall, E. C. et al….Bauer DLV. (2021) *Neutralising antibody activity against SARS-CoV-2 VOCs B.1.617.2 and B.1.351 by BNT162b2 vaccination.* Lancet. DOI:10.1016/S0140-6736(21)01290-3.
Demonstrated reduction in vaccine-induced neutralising antibody titres against emerging SARS-CoV-2 Variants of Concern, as well as age-depending and time-dependent reductions in antibody titres.

Dadonaite B, Gilbertson B, Knight ML, Trifkovic S, Rockman S, Laederach A, Brown LE, Fodor E, and Bauer DLV. (2019) *The structure of the influenza A virus genome*. Nature Microbiology 4(11) 1781-1789. DOI: 10.1038/s41564-019-0513-7
Discovered that the RNA genome of influenza A viruses is much more structured than previously thought, changing the way the field thinks about reassortment (i.e. how a new pandemic could be made) and factors for pathogenicity and cytokine induction.

Bauer DLV*, et al. (2018). *Influenza Virus Mounts a Two-Pronged Attack on Host RNA Polymerase II Transcription*. Cell Reports 23, 2119–2129.e3 * corresponding author. DOI: 10.1016/j.celrep.2018.04.047
Dysregulation of host transcription during influenza virus infection surprisingly can occur independently of the viral NS1 protein's ability to interfere with host mRNA processing (as has been previously assumed), and that this dysregulation caused previously-reported 'downstreamof-gene' transcripts.

Duchi D, Bauer DLV,et al. (2016) *RNA Polymerase Pausing During Initial Transcription*. Molecular Cell 63, 939-950. DOI: 10.1016/j.molcel.2016.08.011
Discovered long-lived pausing during initial transcription in E. coli (rather than rapid abortion of transcription) at position +6 from the TSS and showed it is caused by a clash between nascent transcript and the RNAP σ70 'priming loop' domain.