Name	EACHAN JOHNSON
Position	Group Leader (1st 6)
Year joined *(Crick or founder institute)*	2021

Career History

2009-2014: DPhil Chemical Biology, Departments of Chemistry and Biochemistry, University of Oxford
2014-2020: Broad Institute of MIT and Harvard; Department of Molecular Biology, Massachusetts General Hospital; Department of Genetics, Harvard Medical School

Major Awards, Honours and Prizes

2016-2017: BroadNext10 Catalytic Steps Award
2019: Partners Healthcare Innovator Award

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

Lab Name	***Systems Chemical Biology of Infection and Resistance Laboratory***

Research programme and achievements

We are developing precision molecular tools to study how pathogenic bacteria survive and infect, enabling the design of new antimicrobial therapies.

As soon as new antimicrobial drugs are discovered and used in the clinic, pathogenic bacteria inevitably evolve resistance, driving an unsustainable cycle threatening the twentieth century's improvements to public health.

Antibiotics revolutionised modern medicine, but once again millions of lives are threatened by pathogenic bacteria like *M. tuberculosis*, which causes tuberculosis, the deadliest infectious disease and one of the top 10 causes of death worldwide.

Working at the interface of genetics, chemistry, and machine learning, we use chemical 'probes' to systematically and precisely disrupt the cellular machinery of *M. tuberculosis* and study the consequences of this disruption on its ability to survive, infect, and resiliently evolve resistance.

With this approach, we seek to bridge the gap between understanding pathogen biology and designing new therapeutic strategies.

Research outputs

Johnson EO, et al. (2019) ***Large-scale chemical-genetics yields new M. tuberculosis inhibitor classes*****. Nature, 571, 72–78. DOI: 10.1038/s41586-019-1315-z**
Development of a new phenotypic chemical screening strategy in M. tuberculosis that, for the first time, provided mechanism of action information for active compounds in primary phenotypic screening data and sensitively detected bioactive small molecules in new regions of chemical space, enabling compound prioritization based on putative targets instead of simply on potency. Highlighted in Nature Reviews Drug Discovery, Nature Chemical Biology and Biochemistry

Johnson EO, Office E, Kawate T, Orzechowski M, Hung DT. (2019) ***A large-scale chemical-genetic strategy to design antimicrobial combination chemotherapy for Mycobacterium tuberculosis*****. ACS Infectious Diseases, 6(1), 56–63. 10.1021/acsinfecdis.9b00373**
Retrospectively mined the PROSPECT data, for another structurally distinct compound inhibiting EfpA. Resistance conferring mutations of the two compounds are mutually exclusive, and resistance to one compound causes hypersensitivity to the other, in some cases completely suppressing emergence of cross-resistance.