Available online at www.sciencedirect.com

ScienceDirect

Biomedical Journal

journal homepage: www.elsevier.com/locate/bj

Editorial Note: Special Edition

Legacy of the influenza pandemic 1918: Introduction

Siamon Gordon *

Graduate Institute of Biomedical Sciences, College of Medicine, Chang Gung University, Taoyuan, Taiwan
Exeter College Emeritus Fellow in Pathology, and Emeritus GlaxoWellcome Professor of Cellular Pathology, University of Oxford, UK

ARTICLE INFO

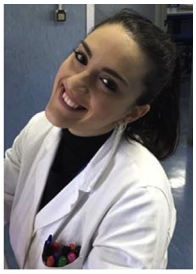
Article history:
Received 18 January 2019
Accepted 18 January 2019
Available online 28 February 2019

Keywords:
Influenza
Pandemic anniversary
Immunity

This guest edited issue contains the articles follow the earlier group of articles to mark the centennial of the influenza pandemic which swept the world in 1918, continuing into the following year. The following section provides brief personal background, perspectives and references provided by the two corresponding authors.

Carmelo Biondo, Germana Lentini, Concetta Beninati, Giuseppe Teti: The dual role of innate immunity during influenza


Dr. Biondo


Dr. Lentini


Dr. Beninati


Dr. Teti

Giuseppe Teti is currently acting as Scientific Director of Scylla Biotech S.r.l, a spin off of the University of Messina, Messina,

* *Corresponding author*. Graduate Institute of Biomedical Sciences, Chang Gung University, 259, Wenhua 1st Rd., Gueishan, Taoyuan 333, Taiwan.
E-mail address: siamon.gordon@path.ox.ac.uk.
Peer review under responsibility of Chang Gung University.
https://doi.org/10.1016/j.bj.2019.01.006

Italy involved in research on innate immunity, epitope mapping and vaccine development. A native of Naples, Italy, he obtained his medical degree at the Catholic University of Rome and worked for several years in microbiology and basic immunology at the Medical University of South Carolina in Charleston, South Carolina. He later worked at the University of Messina as Professor of Medical Microbiology.

Our understanding of the mechanisms underlying host defence against infection continues to grow at a fast pace and it is time now to consider how we can apply this knowledge to control influenza and the associated secondary bacterial infections, which still represent a major public health problem. Drs Biondo, Lentini, Beninati and Teti are interested in how the innate immune system recognizes extracellular bacteria, such as those causing secondary infections during influenza, and in identifying the major arms of the host defence system that are predominantly involved in clearing these infections [1,2]. They are now studying how previous exposure to influenza virus affects subsequent anti-bacterial activities. In the context of this broad field, Drs Biondo, Lentini, Beninati and Teti are particularly focusing on the role of nucleic acid-sensing Toll-like receptors in inducing type I interferons [1,3] and interleukin-1 beta [4,5] responses and in activating neutrophils [6,7]. While type I interferons have been traditionally viewed as playing a major role in anti-viral defences only, it was recently found that inferferon beta production occurs very early during bacterial infection and is crucial for priming phagocytes for increased antibacterial activity [8]. Drs Biondo, Lentini, Beninati and Teti are currently based at the Metchnikoff Laboratory, a public-private research unit hosted by the University of Messina in Messina, Italy.

REFERENCES

[1] Signorino G, Mohammadi N, Patane F, Buscetta M, Venza M, Venza I, et al. Role of toll-like receptor 13 in innate immune recognition of group B streptococci. Infect Immun 2014;82:5013–22.

[2] Costa A, Gupta R, Signorino G, Malara A, Cardile F, Biondo C, et al. Activation of the NLRP3 inflammasome by group B streptococci. J Immunol 2012;188:1953–60.

[3] Mancuso G, Gambuzza M, Midiri A, Biondo C, Papasergi S, Akira S, et al. Bacterial recognition by TLR7 in the lysosomes of conventional dendritic cells. Nat Immunol 2009;10:587–94.

[4] Biondo C, Mancuso G, Midiri A, Signorino G, Domina M, Cariccio VL, et al. Essential role of interleukin-1 signaling in host defenses against group B streptococcus. Mbio 2014;5:e01428–514.

[5] Biondo C, Mancuso G, Midiri A, Signorino G, Domina M, Cariccio VL, et al. The interleukin-1 beta/CXCL1/2/neutrophil axis mediates host protection against group B streptococcal infection. Infect Immun 2014;82:4508–17.

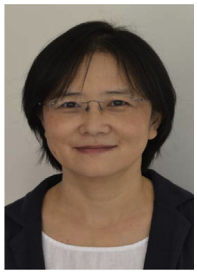
[6] Mohammadi N, Midiri A, Mancuso G, Patane F, Venza M, Venza I, et al. Neutrophils directly recognize group B streptococci and contribute to interleukin-1 beta production during infection. PLoS One 2016;11:e0160249.

[7] Biondo C, Mancuso G, Beninati C, Iaria C, Romeo O, Cascio A, et al. The role of endosomal toll-like receptors in bacterial recognition. Eur Rev Med Pharm 2012;16:1506–12.

[8] Mancuso G, Midiri A, Biondo C, Beninati C, Zummo S, Galbo R, et al. Type I IFN signaling is crucial for host resistance against different species of pathogenic bacteria. J Immunol 2007;178:3126–33.

Dannielle Wellington and Tao Dong: IFITM3: How genetics influence Influenza infection demographically

Dr. Dannielle Wellington



Dr. Tao Dong

Tao Dong is a Professor of Immunology at the MRC Human Immunology Unit and founding director (Oxford) of CAMS-Oxford Institute and Director of Center for translational Immunology, Oxford University and Senior Fellow in University College. She gained a BSc (Physiology) from Fudan University, Shanghai, China in 1987 and D. Phil in Oxford University in 1998.

Tao Dong's main research interest is to understand the key factors required for efficient viral control by the T cell response in a number of different viral infections and cancer, with the aim to augment and control the immune response as a way of improving the outcome in several important human diseases. Her group has uncovered novel mechanisms by which virus and host (HLA) genetic variation controls antigen processing and presentation to T cells, and also described how host genetic variation has an impact on HIV virus evolution and Influenza virus infection such as IFITM3.

The role of host genetic variability in the susceptibility to influenza virus has not been widely investigated due to the influence of confounding factors and a lack of conclusive association between the host and virus. However, following reports of a single nucleotide polymorphism within the anti-viral protein IFITM3 [1], Prof. Tao Dong decided to investigate the association of this SNP, rs12252, with influenza in a Chinese cohort [2]. They found a significant association between the development of severe influenza and homozygosity for the minor allele C. Consequently, this association has been confirmed in further studies [3]. The *IFITM3* rs12252-CC genotype is only common in Asian populations, suggesting that genetic variation of the host is confounded by ethnic background [4]. The mechanism of how the synonymous SNP rs12252 alters IFITM3 viral restriction is still unknown and a key focus of the research by

Prof. Dong and a postdoctoral fellow Dr Dannielle Wellington. In this review they explain the role of host genetic variation in influenza infection severity with a focus on the role IFITM3 plays, summarising what is known about IFITM3 and the open questions that surround this important anti-viral protein.

REFERENCES

[1] Everitt AR, Clare S, Pertel T, John SP, Wash RS, Smith SE, et al. IFITM3 restricts the morbidity and mortality associated with influenza. Nature 2012;484:519–23.

[2] Zhang YH, Zhao Y, Li N, Peng YC, Giannoulatou E, Jin RH, et al. Interferon-induced transmembrane protein-3 genetic variant rs12252-C is associated with severe influenza in Chinese individuals. Nat Commun 2013;4:1418.

[3] Prabhu SS, Chakraborty TT, Kumar N, Banerjee I. Association between IFITM3 rs12252 polymorphism and influenza susceptibility and severity: a meta-analysis. Gene 2018;674:70–9.

[4] Zerbino DR, Achuthan P, Akanni W, Amode MR, Barrell D, Bhai J, et al. Ensembl 2018. Nucleic Acids Res 2018;46:D754–61.