The University of Glasgow and Kings College London received anonymous allegations of potential research misconduct involving a large number of papers related to a laboratory of a former University of Glasgow employee. Under the Russell Group Statement of Cooperation, it was agreed that the University of Glasgow would lead on the investigation.

We have followed our procedures and obligations as defined by UKRIO (UK Research Integrity Office) and laid out in our Misconduct Policy and to date have upheld the allegations related to a number of papers. Consequently, the University is in discussions to retract these papers.

The investigation is currently ongoing.

The papers concerned are:

*Paper 1 - “Integrating cardiac PIP3 and cAMP signaling through a PKA anchoring function of p110y. Molecular Cell, 8;42(1), Pages 84–95, doi: 10.1016/j.molcel.2011.01.030 (January 2011)*

*Paper 2 - “Crosstalk between PI3-Kinase and cAMP-Protein Kinase A signaling pathways at the level of a Protein Kinase B/β-arrestin/cAMP phosphodiesterase-4 complex. Molecular Cell and Biology, 30(7), pages 1660-1672, doi: 10.1128/mcb.00696-09, (April 2010).”*

*Paper 3 - “Scanning peptide array analyses identify overlapping binding sites for the signalling scaffold proteins, β-arrestin and RACK1, in cAMP-specific phosphodiesterase PDE4D5. The Biochemical Journal, 398(pt1) pages 23-36 doi: 10.1042/bj20060423 (August 2006)”*

*Paper 4 - “Phosphodiesterase-4 influences the PKA phosphorylation status and membrane translocation of G-protein receptor kinase 2 (GRK2) in HEK-293β2 cells and cardiac myocytes. The Biochemical Journal, 394(pt2) pages 427-435 doi: 10.1042/bj20051560 (March 2006)”*

*Paper 5 - “RNA Silencing Identifies PDE4D5 as the Functionally Relevant cAMP Phosphodiesterase Interacting with βArrestin to Control the Protein Kinase A/AKAP79-mediated Switching of the β2-Adrenergic Receptor to Activation of ERK in HEK293B2 Cells. The Journal of Biological Chemistry, 30;280(39) pages 33178-33189 doi: 10.1074/jbc.m414316200 (September 2005)”*

*Paper 6 - “β-Arrestin-mediated PDE4 cAMP phosphodiesterase recruitment regulates β-adrenoceptor switching from Gs to Gi. Proceedings of the National Academy of Sciences of the United States of America, 100(3) pages 940-945 doi: 10.1073/pnas.262787199 (February 2003)”*

*Paper 7 - “Phosphorylation of PDE4A5 by MAPKAPK2 attenuates fibrin degradation via p75 signalling. Journal of Biochemistry, 2019;166(1):97–106 doi:10.1093/jb/mvz016***”**