Researchers identify inflammatory biomarkers in T cells

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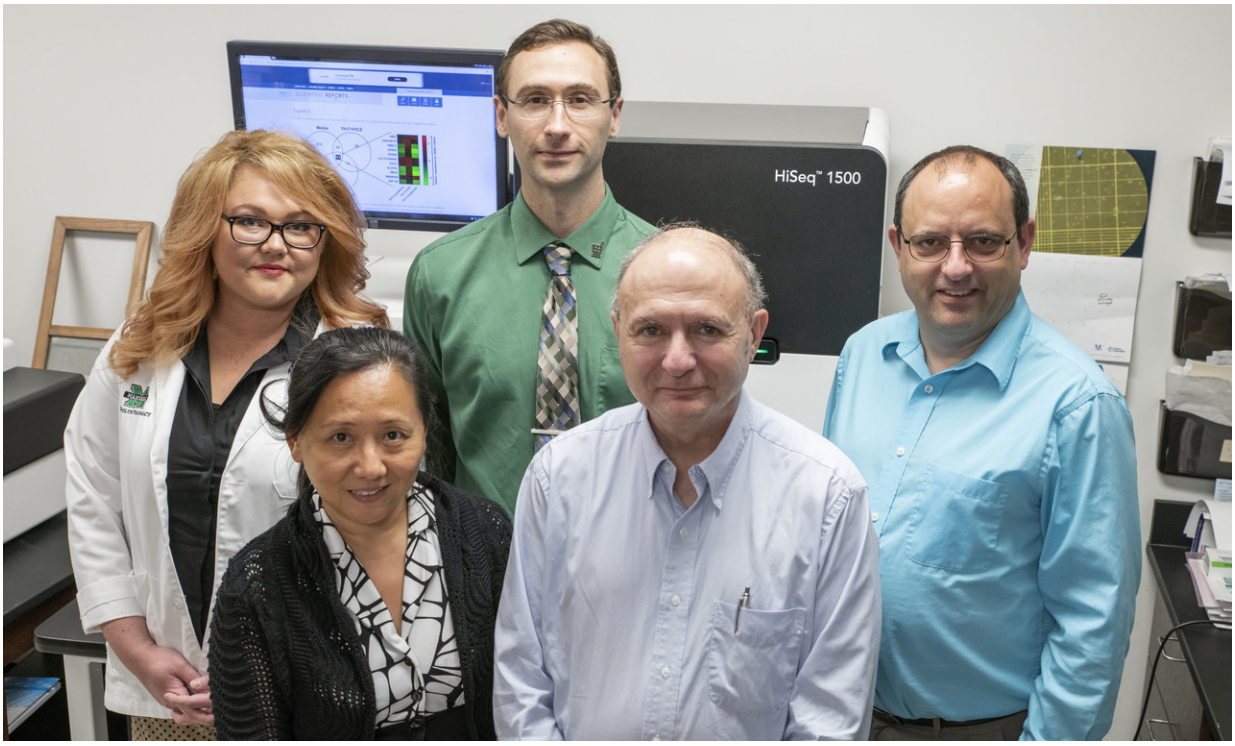

The team of researchers from the Marshall University School of Pharmacy and Marshall University Joan C. Edwards School of Medicine who identified inflammatory biomarkers in T cells includes (front row, from left) Jun Fan, Ph.D.; Donald A. Primerano, Ph.D.; (back row, from left) Bryanna Roar, third-year Pharm.D. student; Jeremy McAleer, Ph.D.; James Denvir, Ph.D. Credit: Marshall University

The Marshall University School of Pharmacy, in collaboration with the Marshall University Joan C. Edwards School of Medicine Genomics Core, recently released a new study that explores human T cell function under inflammatory conditions.

The findings are published in the July 19, 2018, edition of *Scientific Reports*, an online journal from the publishers of *Nature*.

"Our gene expression analysis of T cells provides many possible targets for studying how environmental products control T cell activation and pro-inflammatory functions," said Jeremy P. McAleer, Ph.D., lead author and assistant professor at the Marshall School of Pharmacy. "We were encouraged to find that one of these targets, named GPR68, regulates the ability of T cells to produce chemical messengers. This may have implications for diseases on mucosal surfaces such as the lungs and gastrointestinal tract."

The study examined T cells, which protect against bacteria, fungi and viruses on mucosal surfaces. When activated against harmless substances, T cells may provoke autoimmune diseases. Findings reveal that the set of genes expressed by T cells under pro-inflammatory conditions include several G-protein-coupled receptors (GPRs). Future studies will explore if blocking the GPR68 pathway can be a potential therapy for chronic inflammatory diseases.

The research team included faculty from the schools as well as a third-year pharmacy student.

More information: Jeremy P. McAleer et al. Cytokine Regulation in Human CD4 T Cells by the Aryl Hydrocarbon Receptor and Gq-Coupled Receptors, *Scientific Reports* (2018). DOI: 10.1038/s41598-018-29262-4

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