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New research finds that enzyme is absent in Crohn's disease sufferers

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Royal Veterinary College researcher Dr David Bishop-Bailey, alongside collaborators from University College London, Queen Mary University London, the University of Umeå and the National Institute of Environmental Health Sciences / National Institutes of Health have discovered that an enzyme which helps immune cells to clear infections caused by bacteria appears to be absent in patients suffering from Crohn's disease.

It is hoped that the findings, which have been published in the journal *PLoS ONE*, will open up a new pathway to investigate for therapeutic intervention in Crohn's disease, a form of Inflammatory Bowel Disease which affects thousands of people in the UK.

Crohn's Disease is a condition that causes inflammation of the digestive system, also known as the gastrointestinal tract or gut and is associated with an inability to clear infections such as those caused by bacteria. Crohn's is sometimes described as a chronic condition, meaning that it is ongoing and usually lifelong.

Dr Bishop-Bailey and his colleagues used human cell lines and cells from volunteers and Crohn's disease patients to investigate the enzyme CYP2J2, as its role in sensing and clearing bacteria is currently not known and its regulation in human inflammatory diseases is poorly understood.

During their investigation they discovered that CYP2J2 helps immune

cells take up bacteria to remove them and that this enzyme appears to be absent in stimulated immune cells from Crohn's disease patients.

Dr Bishop-Bailey said: "We hope that this research may open up new therapeutic avenues for Crohn's disease and lead to new tests into whether mimicking or bypassing CYP2J2 by giving its enzymatic products can lead to beneficial effects for the thousands who sufferer from this disease."

More information: Bystrom, J. et al. (2013) Inducible CYP2J2 and its product 11,12-EET promotes bacterial phagocytosis: a role for CYP2J2 deficiency in the pathogenesis of Crohn's Disease? *PLoS One*, 8 (9): e75107. dx.doi.org/10.1371/journal.pone.0075107

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