Understanding the body’s response to aspirin

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University scientists have made an important breakthrough in understanding how different individuals respond to aspirin.

This is significant because aspirin is a widely used cardiovascular preventive drug and also has an emerging role in cancer treatment and prevention.

Understanding how people respond to aspirin is key in terms of knowing who will benefit from it.

The research, led by Professor Valerie O’Donnell from the University’s Systems Immunity Research Institute showed for the first time that there is a direct link between energy generation and fast changes in the levels of cellular lipids (fats), in specialised blood cells called platelets that are essential for blood clotting.

“Our research shows a new link between energy metabolism and inflammation as well as giving early insights into the fundamentals of precision medicine regarding the variation of the lipidome among individuals,” said Professor O’Donnell.

The research, which also involved Professor Victor Darley-Usmar, Director of the University of Alabama at Birmingham Mitochondrial Medicine Laboratory, and Professor Robert Murphy in the Department of Pharmacology, University of Colorado Denver, USA, found more than 5,600 lipids in platelets and worked out the donor variation with aspirin treatment of a subset that are generated when the cells are inflammatory activated.

Published in the journal *Cell Metabolism*, the research is the first comprehensive lipidomic profile of human platelets in response to stimulation and aspirin treatment.

Professor Mike Murphy, Medical Research Council Mitochondrial Biology Unit, Cambridge, said: “This work led by Professor O’Donnell is a technical tour de force, providing a wonderful resource for other biomedical researchers. A particularly important aspect is the focus on platelets, which are readily available from patients’ blood in diagnosis, prognosis or as a biomarker in assessing therapies.

“In addition to its future use, this work also demonstrated an unexpected link between mitochondrial fat metabolism and platelet activation during inflammation.”

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