Recent Papers at Genes and Development[2]

Authors: Janet Lopez James Brooks Katherine Little Lisa Robbins Kevin Watts

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University of Alaska Southeast

School of Mathematics

Takeshi Utsumi has been studying and investigating how protein breakdown events cause systemic inflammation in the gastrointestinal tract for over fifteen years. On 22 December, he announced that he had discovered the pathogenicity of monosodium urate (muca), a common factor in chronic inflammatory disorders, such as pancreatitis, scleroderma, gastroenteritis, Crohnâ€™s disease, polymyalgia rheumatica (PRM), gout, ulcerative colitis and transthyretin amyloidosis. In a pre-clinical study published in Genes and Development, he examined the interaction between diabetes-related inflammation and a nutrient source known to trigger pro-inflammatory mechanisms: monosodium urate.

â€œHypertension and inflammation are considered as co-morbidities of the metabolic disorder type-2 diabetes,â€ Utsumi explained. â€œIf we examine the long-term effects of any nutrient, particularly in increasing the toxicity of a fatty acid in the plant-processing process, we find that in the case of monosodium urate, the consumption results in increased inflammation in the intestine.â€ In the mouse model Utsumi developed, it appears that a specific cell type is also present that is affected by both glucose and a source of monosodium urate, namely monosuccilli mesopheli epithelial cells (media 7). This is the same cell type that is involved in the production of chemical changes in the intestine while conducting a pro-inflammatory response.

In fact, these cells may also be the cause of heart disease, due to the dysregulation of the lipid metabolism that is controlled by the glucose-stress-upregulation pathway. Now that Utsumi has identified that monosuccilli mesopheli epithelial cells play a role in the heart disease of people with diabetes, he intends to develop a therapy that could treat both diabetes and heart disease, or any other area of inflammation that is commonly related to dietary sugar intake.

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For more information about Utsumiâ€™s studies, check out his website

A Close Up Of A Metal Fence Near A Fence