Transportation of beta-carotene to pancreas from peripheral region contributes to pancreatic cancer, triglyceride and insulin resistance, metabolic syndrome

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It is widely known that ethanol is damaging to the gastrointestinal tract and various immune, autoimmune, and metabolic diseases in people suffering from obesity, diabetes, and even inflammatory bowel disease.

Recently, Type 2 diabetes patients are getting kidney damage and symptoms resembling cardiovascular disease despite being insulin-resistant.

Scientists have shown that a catalyst is present when the digestive tract takes in sugar from food and extracts it into the blood. They hypothesize that the enzyme prostaglandin-K (PGP) is involved in this process. They also show that the leukocyte (adult white blood cells) receptor PPARÎ±, a site-specific enzyme, is located in the same place as this catalyst. PPARÎ± interacts with PGP and instructs the white blood cells to make a leukocyte channel (the commissor), releasing proinflammatory PGF on a polycarboxylated junction.

In particular, human glucocorticoid-induced endothelial cell-dependent oligodendrocyte sarcoma (GA-oESA) lesions present in the pancreas and adrenal insufficiency commonly are associated with PGP/PPARÎ± interactions.

Another complication of the combination of PPARÎ±/PGP functions is that they induce glucose release into the circulation, which further increases the incidence of obesity, Type 2 diabetes, obesity related morbidity, and chronic fatigue syndrome (CFS).

To investigate whether PGF-induced glucocorticoid-induced necrosis tumors in human adrenal insufficiency patients is caused by PGF-mediated metabolism, together with release of proinflammatory PGF from the commissor, researchers exposed 36 men with glucocorticoid-induced adrenal insufficiency and heavy drinking (a condition in which alcohol is hepatotoxic and two-thirds to three-fourths of the affected people become alcoholics), and 13 men with diabetes.

They compared all the patients with normal glucocorticoid tolerance with all patients with pancreatic oligodendrocyte sarcoma and metabolic syndrome. They also subjected the groups to dyslipidemia with severe insulin resistance and colorectal cancer.

The researchers found that after 30 days of these experiments, the patients with normal tolerance and metabolic syndrome had a lower incidence of glucocorticoid carcinoma with a three times higher incidence of metastasis. Conversely, the patients with diabetes and severe glucocorticoid tolerance had a 19-fold higher incidence of pancreatic carcinoma.

They found that PGF-induced peripheral damage is not transferred to the secondary site due to the glucocorticoid-enhanced release of proinflammatory proinflammatory (PPMA)-mediated procalcitonin-released cytokines and other proinflammatory mediators.

This study thus confirms that the glucocorticoid axis-1-GT1-PK and the proinflammatory signalling of PGF-mediated metabolic syndrome contribute to the development of pancreatic cancer. However, the glucocorticoid axis-1-GT1-K (the two segments of the three-part GT1-ÎºÎ± pathway regulating glucose absorption) exhibit inhibition of myeloid-derived suppressor cells and abnormal cytoplasmic tissue permeability and cell-to-cell communication in pancreas of diabetes patients, which prolongs hepatic toxicity. Therefore, this study emphasizes the fact that postulate about endocrine metabolic diseases appears true in the gastrointestinal tract.

A Close Up Of A Bird On A Tree Branch