

Экстренное сообщение Российской Диабетической Газеты (РДГ) и Российского журнала эндокринологии, диабетологии и метаболизма...

Доктор Марк Браун и коллеги из Учебно-Исследовательского Института Клиники Кливленда, а также их научные предшественники, продемонстрировали, что имеется статистически достоверная взаимосвязь между продукцией триметиламина оксида (ТМАО) бактериями кишечника человека и развитием ожирения, инсулинорезистентности, метаболического синдрома, сахарного диабета 2 типа, сердечно-сосудистыми заболеваниями. Внимание исследователей сфокусировалось на возможностях терапевтической блокады фермента Флавин Содержащей Монооксигеназы – 3 (FMO-3) , превращающей ТМАО в активную форму. Первые результаты лекарственной блокады фермента на экспериментальных животных показали возможность защиты этих животных от развития метаболического синдрома и его смертельных последствий. Новый взгляд на синдром Ривена позволит не только разработать новые лекарства, но и более целенаправленно подбирать диеты для лиц с повышенным весом, ожирением, сахарным диабетом 2 типа путем активного влияния на состав микробиоты (микроорганизмов тела человека). О принципах составления таких диет и о их составе РДГ сообщит в ближайшее время.

Ссылка на первоисточник - журнал Cell Reports:

09.07.2017 Pathway to obesity: Study links TMAO-producing gut bacteria to obesity risk

The way bacteria in our gut metabolise the foods we eat could have a strong impact on obesity risk, while blocking certain pathways could lead to prevention, say researchers. The study, published in Cell Reports, suggests a biological link between TMAO-producing gut bacteria and obesity – adding that altering gut bacteria pathways to block production could stimulate fat tissue to prevent obesity.

Led by Dr Mark Brown from Cleveland Clinic's Lerner Research Institute, the scientists showed that blocking a specific intestinal microbial pathway involving trimethylamine oxide (TMAO) can prevent obesity and insulin resistance, as well as cause fat tissue to become more metabolically active.

Previous research involving study co-author Dr Stanley Hazen showed that high levels of TMAO, which are produced by gut bacteria during the digestion of the nutrients choline, lecithin and carnitine, are associated with a higher risk of severe cardiovascular events, such as heart attack and stroke.

"Obesity, diabetes and cardiovascular disease are strongly linked,”said Brown. “While the microbiome has been shown to affect cardiovascular disease, there is as yet no concrete evidence of precisely how gut bacteria influence obesity.”

"These findings shed light on a possible way to manipulate the microbiome with therapeutics to combat our obesity and diabetes epidemic,” he added. Study details The team focused on a host enzyme called flavin-containing monooxygenase 3(FMO3), which converts TMAO into its active form.

They discovered that mice that had a missing or deactivated FMO3 gene were protected from obesity, even when fed a high-fat, high-calorie diet. Furthermore, the FMO3-negative mice showed higher expression of genes associated with beige or brown fat cells, which are more metabolically active than white fat cells.

Observations in mice were then confirmed in work that looked at 435 patients, which showed that high levels of TMAO are associated with higher incidence of Type 2 diabetes.

“Complimentary mouse and human studies indicate a negative regulatory role for FMO3 in the beiging of white adipose tissue,”wrote the team. “Collectively, our studies reveal a link between the TMAO-producing enzyme FMO3 and obesity and the beiging of white adipose tissue.”

Given the ‘numerous strong associations’ of the gut microbe and the TMAO pathway with human disease, Hazen noted that the work has ‘broad implications’ for the development of new microbiome focused therapies which target and modulate the metabolism of our own gut bacteria.

However, he noted that further work is needed to better understand the entire pathway and the links between TMA, FMO3, TMAO and human health.

*Hazen is an inventor of a test for TMAO that was licensed to the spin-off company Cleveland HeartLab, Inc., a Cleveland Clinic. As a result, Hazen and Cleveland Clinic would benefit financially from sales of the test.

Source: Cell Reports Volume 19, Issue 12, Pages 2451–2461, June 2017, doi: 10.1016/j.celrep.2017.05.077 “The TMAO-Producing Enzyme Flavin-Containing Monooxygenase 3 Regulates Obesity and the Beiging of White Adipose Tissue” Authors: Rebecca C. Schugar, et al