September 15, 2017

Dear Regina,

Welcome to BioMarketing Insight's monthly newsletter.

We have a new look to our newsletter. Love to receive your feedback.

Last month I covered part one of a two part series on Obesity entitled "Can't Stop Eating? Maybe Your Brain is Telling You to Eat." If you missed last month's article, click here to read it. This month we'll cover part two of the series, entitled "White Fat is Bad, Brown Fat is Good, Can We Convert White to Brown Fat?"

Read on to learn more about this topic and other current news. The next newsletter will be published on October 15th, 2017.

We encourage you to share this newsletter with your colleagues by using the social

Please email me, Regina Au, if you have any questions, comments, or suggestions.

Sincerely,
Regina Au
Principal, New Product Planning/
Strategic Commercial Consultant
BioMarketing Insight

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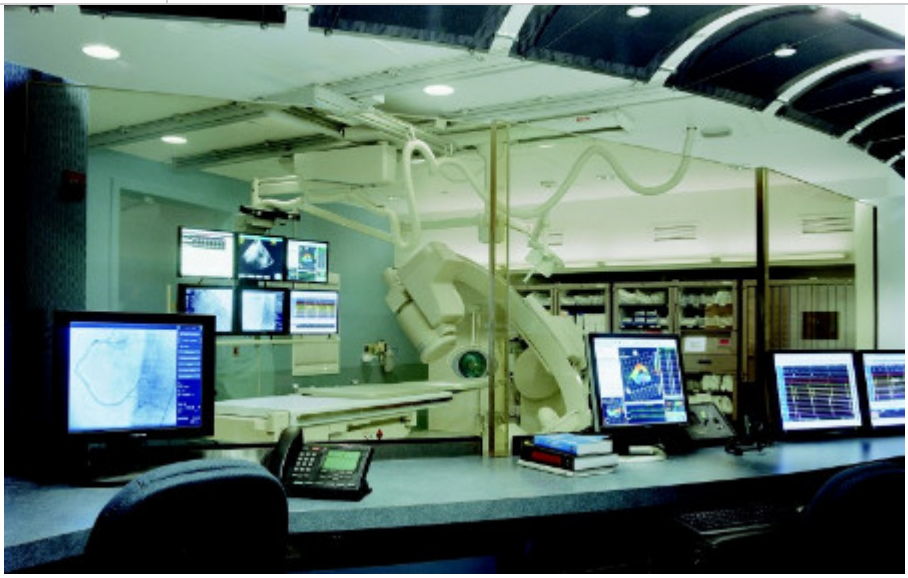

Developing a Product? Commercializing a Product?

If you are developing a product and have not conducted the business due diligence to determine commercial viability or success, contact me for an appointment. For successful commercial adoption of your product or looking to grow your business, contact me for an appointment.

For more information on our services, click on the links below:

Product Development
Market Development
Marketing Strategies

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Innovation Navigation: Road Map from Idea to a Successful Company

I am pleased to announce that I will be speaking at The Boston Entrepreneur's Network (ENET) meeting on Tuesday, September 19th, 2017, from 6:00 - 8:45 PM at Pivotal Labs,

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Why Our Microbiome is Important to Our Physiology and Diseases

I am pleased to announce that my article entitled "Why Our Microbiome is Important to Our Physiology and Diseases" was published in the International Journal of Clinical Pharmacology & Pharmacotherapy. This article reviews the results of the Human Microbiome Project and the factors that affect our microbiome in relation to our healthy state and dysbiosis or disease state. To read the article, click here.

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Immunooncology: Can the Right Chimeric Antigen Receptors T-Cell Design Be Made to Cure All Types of Cancers

I am pleased to announce that my article on "Immunooncology: Can the Right Chimeric Antigen Receptors T-Cell (CAR-T) Design Be Made to Cure All Types of Cancers and Will It Be Covered?" has been published in Journal of Pharmaceutics. This article reviews the mechanism, design and administration of CAR-T cells, and whether payers will pay for this new technology. To read the article, click here.

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Lucille Ball (left) and Vivian Vance "I Love Lucy", CBS-TV (1951-1957)

White Fat is Bad, Brown Fat is Good, Can We Convert White to Brown Fat?

We know that too much fat is not good. But, what is the difference between white and brown fat and why is one bad and the other good? Scientists have been studying fat for decades and found that the type of fat makes a difference in your metabolism. White fat, or white adipocytes, get their color from triglycerides and other unhealthy substances and their only function is to store energy. Too much white fat can lead to obesity which in turn

So how do we get more brown fat instead of white fat? Scientists at the Perelman School of Medicine at the University of Pennsylvania have identified a signaling pathway that may turn white fat into brown. The pathway activates a "browning program" in white adipocytes that gives them the characteristics of energy-burning brown fat, according to a press release from the university.

"It's conceivable that one would be able to target this pathway with a drug, to push white fat to become brown fat and thereby treat obesity," said Zoltan Arany, an associate professor of cardiovascular medicine at Penn and the senior author of the study.

Arany and his team discovered that the protein Folliculin (FLCN), in conjunction with the protein complex called mTOR, suppressed browning activity in white fat cells. When the gene that makes FLCN was deleted in mice, their white cells started to brown, producing visibly more mitochondria that convert energy into heat. This finding was prompted from earlier research, where the team had discovered that activating the mTORC1 and mTORC2 protein complexes could instigate the “beiging” of white fat in mice, which helped burn fat, and control blood sugar and cholesterol.

However, the quest to turn white fat into brown fat has not been easy. In 2015, Ember Therapeutics—which had been launched in 2012 with $34 million from Third Rock Ventures, quietly closed its doors after its efforts to use the protein BMP-7 to stimulate the production of brown fat failed.

On the positive side, other theories to solve this problem are still being researched. In 2015, a team at UC Berkeley developed a scaffold comprised of stem cells programmed to transform white fat into beige fat. When they put the scaffold into mice and fed them a high-fat diet, they discovered that the animals gained half as much weight as control mice. Their blood sugar and fatty acid levels were also improved.

Another theory from scientists at Florida’s Sanford Burnham Prebys Medical Discovery Institute discovered that Natriuretic peptides (NPs) may be the key to turning unhealthy white fat into energy-burning brown fat.

It is believed that NPs send signals to white fat in the adipose tissue to turn harmful white fat into energy-burning brown fat. It is also believed that NP signaling may even play a role in protecting against metabolic disease.

they dial up NP signaling in adipose tissue of mice, they can prevent obesity and increase sugar uptake in brown fat.

When receptors that normally remove NPs from circulation were deleted from adipose tissue in mice, and the animals were fed a high-fat diet, they not only showed improved insulin sensitivity, they also suffered less inflammation and burned more energy.

"Usually when you feed mice high-fat diets they get fatty liver," said senior author Sheila Collins, Ph.D., professor in the Integrative Metabolism Program at Sanford Burnham, in a press release. "In mice without NRPCs in adipose tissue the liver was completely clean and completely devoid of stored lipids, which I'm sure contributes to their improved overall metabolic performance."

This could explain why in previous research it was shown that naturally thin people have higher levels of NPs in their blood.

Harvard University researchers took a different approach and discovered an enzyme that protects cells from harmful fats, that could lead to improved treatments for obesity and its related diseases.

Triglycerides are broken down into fatty acids and then transported out of fat cells to other tissues in the body for energy. However, any remaining fatty acids can damage the endoplasmic reticulum, a cell component that makes cellular products, such as proteins and fats.

Re-esterification is a process where any leftover fatty acids in the fat cells turn back into triglycerides thereby protecting the endoplasmic reticulum and warding off inflammation in body fat.

The Harvard team discovered that the enzyme diacylglycerol acyltransferase (DGAT1) drives re-esterification just like the “cellular police officer” to protect the endoplasmic reticulum from fatty acids. This answers why some triglycerides, after being broken down into fatty acids, reassemble themselves.

"To better understand what happens when cells are overwhelmed with fat during obesity, we first have to understand how the system normally deals with fluctuations in lipids," said Tobias Walther, professor of genetics and complex diseases at Harvard University’s Chan School of Public Health and co-senior author of the study, in the statement. "Our findings

Closing Thoughts

Over the years, there have been many different approaches to combat obesity, from drugs and supplements for appetite suppression or reduction in fat absorption, to medical devices such as weight-loss balloons that fill the stomach to curb one's appetite for food.

In addition to research on converting white fat to brown fat, researchers at John Hopkins recently found that removing the enzyme O-GlcNAc transferase (OGT) from mice caused them to overeat, indicating that a key signal to stop eating in the nerve cells of the brain had been turned off by the absence of OGT. This could also be the beginning of the discovery of new drugs that control appetite.

It appears that there are many mechanisms that may contribute to obesity, as discussed in this article on converting white fat to brown fat and last month's article on how hunger promoting neurons in our brain expresses the gene for Agouti-related protein (AGRP), plays a role in maintaining body weight that can induce food intake even when we are full. So perhaps we need to take a multi-prong approach as well and shift to a personalized approach, as one size does not fit all.

There is an increasing interest in this field because obesity is becoming an epidemic not only in the US, but also across many developed countries as well. Obesity and the risk of developing more serious diseases is what will drive the healthcare costs up exponentially, due to the care and management of these diseases, not the cost of drugs, as it's has been publicized in the news lately.

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Should you have any questions or need of assistance with your business due diligence, determining your product's value proposition and economic value of your product, feel free to contact me at 781-935-1462 or regina@biomarketinginsight.com.
