BIO NEWS

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1．皮下脂肪を褐色脂肪に変える貼り薬

2017 年 12 月 28 日

南陽工科大学（NTU Singapore）の研究者らは、腹部の脂肪を減らす新治療法として、エネルギーを貯蔵する白色脂肪を、エネルギーを燃焼させる褐色脂肪に変えることで知られる薬剤を微小針のついた貼り薬を介して身体に送り込む方法を *Small Methods* 誌に発表した。それによると、マウスの皮膚に 2 分ほど押し付けることで皮膚にポリマー製微小針を埋め込み、ゆっくり分解するその微小針から β3 アドレナリン受容体刺激薬と甲状腺ホルモン T3 を経皮供給することで皮下の白色脂肪組織を、カロリーを燃やす褐色脂肪組織に変えることができた、としている。また、この皮下投与製品を使って β3 アドレナリン刺激薬を投与することで肥満マウスの体脂肪や体重の増加を防ぐことができた、としている。

英文記事：
https://www.sciencedaily.com/releases/2017/12/171228100910.htm

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New patch aims to turn energy-storing fats into energy-burning fats

A new approach to reducing bulging tummy fats has shown promise in laboratory trials

Date:

December 28, 2017

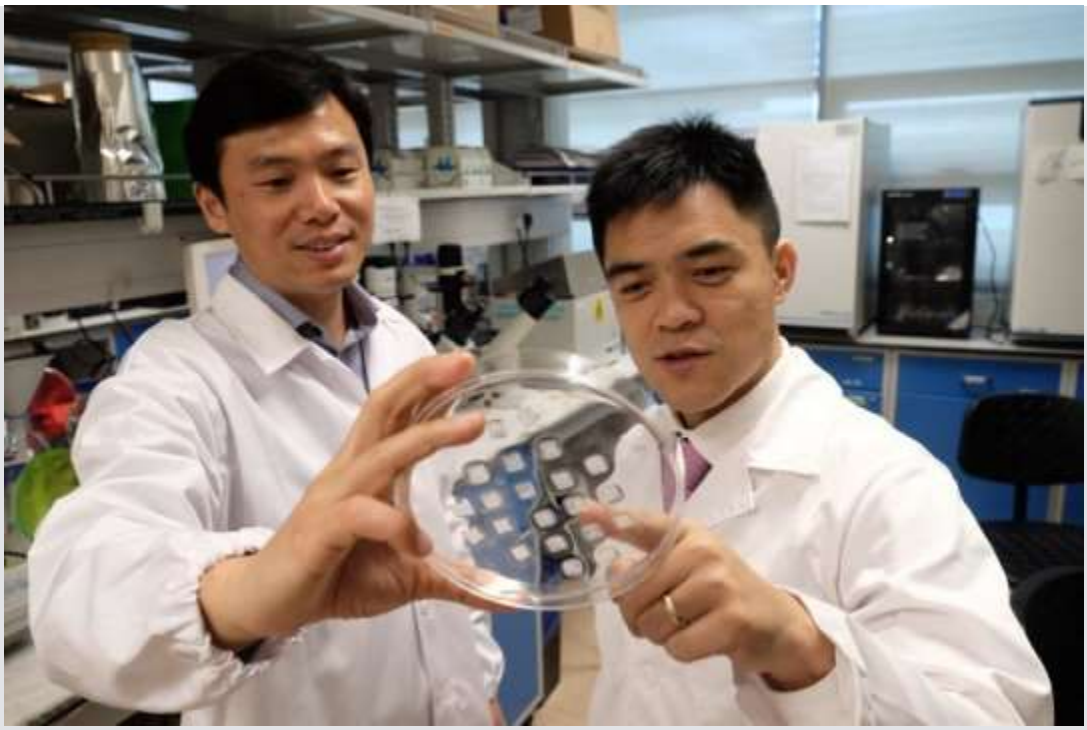
Source:

Nanyang Technological University

Summary:

A new approach to reducing bulging tummy fats has shown promise in laboratory trials. It combines a new way to deliver drugs, via a micro-needle patch, with drugs that are known to turn energy-storing white fat into energy-burning brown fat.

FULL STORY



Prof Chen Peng (left) holding the new microneedle fat burning patch with Asst Prof Xu Chenjie.

Credit: NTU Singapore

A new approach to reducing bulging tummy fats has shown promise in laboratory trials.

It combines a new way to deliver drugs, via a micro-needle patch, with drugs that are known to turn energy-storing white fat into energy-burning brown fat. This innovative approach developed by scientists from Nanyang Technological University, Singapore (NTU Singapore) reduced weight gain in mice on a high fat diet and their fat mass by more than 30 per cent over four weeks.

The new type of skin patch contains hundreds of micro-needles, each thinner than a human hair, which are loaded with the drug Beta-3 adrenergic receptor agonist or another drug called thyroid hormone T3 triiodothyronine.

When the patch is pressed into the skin for about two minutes, these micro-needles become embedded in the skin and detach from the patch, which can then be removed.

As the needles degrade, the drug molecules then slowly diffuse to the energy-storing white fat underneath the skin layer, turning them into energy-burning brown fats.

Brown fats are found in babies and they help to keep the baby warm by burning energy. As humans grow older, the amount of brown fats lessens and is replaced with visceral white fats.

Published in the journal *Small Methods* recently by NTU Professor Chen Peng and Assistant Professor Xu Chenjie, this approach could help to address the worldwide obesity problem without resorting to surgical operations or oral medication which could require large dosages and could have serious side effects.

"With the embedded microneedles in the skin of the mice, the surrounding fats started browning in five days, which helped to increase the energy expenditure of the mice, leading to a reduction in body fat gain," said Asst Prof Xu, who focuses on research in drug delivery systems.

"The amount of drugs we used in the patch is much less than those used in oral medication or an injected dose. This lowers the drug ingredient costs while our slow-release design minimises its side effects," said Asst Prof Xu.

Obesity which results from an excessive accumulation of fat is a major health risk factor for various diseases, including heart disease, stroke and type-2 diabetes. The World Health Organisation estimates that 1.9 billion adults in the world are overweight in 2016 with 650 million of them being obese.

"What we aim to develop is a painless patch that everyone could use easily, is unobtrusive and yet affordable," said Prof Chen, a biotechnology expert who researches on obesity. "Most importantly, our solution aims to use a person's own body fats to burn more energy, which is a natural process in babies."

Under the two scientists' guidance at NTU's School of Chemical and Biomedical Engineering, research fellow Dr Aung Than conducted experiments which showed that the patch could suppress weight gain in mice that were fed a high fat diet and reduce their fat mass by over 30 per cent, over a period of four weeks.

The treated mice also had significantly lower blood cholesterol and fatty acids levels compared to the untreated mice.

Being able to deliver the drug directly to the site of action is a major reason why it is less likely to have side effects than orally delivered medication.

The team estimates that their prototype patch had a material cost of about S$5 (US$3.50) to make, which contains beta-3 adrenergic receptor agonist combined with Hyaluronic acid, a substance naturally found in the human body and commonly used in products like skin moisturisers.

Beta-3 adrenergic receptor agonist is a drug approved by the Federal Drug Administration of the United States and is used to treat overactive bladders, while T3 triiodothyronine is a thyroid hormone commonly used for medication for an underactive thyroid gland.

Both have been shown in other research studies to be able to turn white fats brown, but their use in reducing weight gain is hampered by potentially serious side-effects and drug accumulation in non-targeted tissues if conventional drug delivery routes were used, such as through oral intake.

NTU's Lee Kong Chian School of Medicine Associate Professor Melvin Leow, who was not affiliated with this study, said it is exciting to be able to tackle obesity via the browning of white fat, and the results were promising.

"These data should encourage Phase I Clinical studies in humans to translate these basic science findings to the bedside, with the hope that these microneedle patches may be developed into an established cost-effective modality for the prevention or treatment of obesity in the near future," added Assoc Prof Leow, an endocrinologist.

Since the publication of the paper, the team has received keen interest from biotechnology companies and is looking to partner clinician scientists to further their research.

Story Source:

Materials provided by **Nanyang Technological University**. *Note: Content may be edited for style and length.*

Journal Reference:

1. Aung Than, Ke Liang, Shaohai Xu, Lei Sun, Hongwei Duan, Fengna Xi, Chenjie Xu, Peng Chen. **Transdermal Delivery of Anti-Obesity Compounds to Subcutaneous Adipose Tissue with Polymeric Microneedle Patches**. *Small Methods*, 2017; 1 (11): 1700269 DOI: 10.1002/smtd.201700269

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Nanyang Technological University. (2017, December 28). New patch aims to turn energy-storing fats into energy-burning fats: A new approach to reducing bulging tummy fats has shown promise in laboratory trials. *ScienceDaily*. Retrieved January 2, 2018 from www.sciencedaily.com/releases/2017/12/171228100910.htm
Nanyang Technological University. "New patch aims to turn energy-storing fats into energy-burning fats: A new approach to reducing bulging tummy fats has shown promise in laboratory trials." ScienceDaily. www.sciencedaily.com/releases/2017/12/171228100910.htm (accessed January 2, 2018).

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2. 糖尿病薬がアルツハイマー病マウスの記憶障害を有意に逆転

2017 年 12 月 31 日

Brain Research 誌で発表された英国ランカスター大学の研究で、糖尿病治療のために開発された薬剤を使用することによって、アルツハイマー病の治療において実質的な改善がもたらされる可能性があることが示された。
この薬剤は、グルカゴン様ペプチド 1（GLP-1）、グルコース依存性インスリン分泌刺激ポリペプチド（GIP）、グルカゴンの 3 つを組み合わせたもので、この「三重作用薬」の効能が、アルツハイマー病を引き起こすヒト突然変異遺伝子を発現するトランスジェニックマウス APP/PS1 で見られた、としている。また、そのトランスジェニックマウスは進行した神経変性段階の老齢のものが使用された。

英文記事：
https://www.eurekalert.org/pub_releases/2017-12/lu-dd122017.php

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PUBLIC RELEASE: 31-DEC-2017

Diabetes drug 'significantly reverses memory loss' in mice with Alzheimer's

A drug developed for diabetes could be used to treat Alzheimer's after scientists found it "significantly reversed memory loss" in mice through a triple method of action.

LANCASTER UNIVERSITY

https://www.eurekalert.org/multimedia/pub/159073.php

VIDEO: A drug developed for diabetes could be used to treat Alzheimer's after scientists found it 'significantly reversed memory loss' in mice through a triple method of action. This is the first... view more

Credit: Lancaster University

A drug developed for diabetes could be used to treat Alzheimer's after scientists found it "significantly reversed memory loss" in mice through a triple method of action.

The research, published in *Brain Research*, could bring substantial improvements in the treatment of Alzheimer's disease through the use of a drug originally created to treat type 2 diabetes.

Lead researcher Professor Christian Holscher of Lancaster University in the UK said the novel treatment "holds clear promise of being developed into a new treatment for chronic neurodegenerative disorders such as Alzheimer's disease."

Alzheimer's disease is the most common cause of dementia and the numbers are expected to rise to two million people in the UK by 2051 according to Alzheimer's Society, who part - funded the research.

Dr Doug Brown, Director of Research and Development at Alzheimer's Society, said: ""With no new treatments in nearly 15 years, we need to find new ways of tackling Alzheimer's. It's imperative that we explore whether drugs developed to treat other conditions can benefit people with Alzheimer's and other forms of dementia. This approach to research could make it much quicker to get promising new drugs to the people who need them."

Although the benefits of these 'triple agonist' drugs have so far only been found in mice, other studies with existing diabetes drugs such as liraglutide have shown real promise for people with Alzheimer's, so further development of this work is crucial."

This is the first time that a triple receptor drug has been used which acts in multiple ways to protect the brain from degeneration. It combines GLP-1, GIP and Glucagon which are all growth factors. Problems with growth factor signalling have been shown to be impaired in the brains of Alzheimer's patients.

The study used APP/PS1 mice, which are transgenic mice that express human mutated genes that cause Alzheimer's. Those genes have been found in people who have a form of Alzheimer's that can be inherited. Aged transgenic mice in the advanced stages of neurodegeneration were treated.

In a maze test, learning and memory formation were much improved by the drug which also: -

- enhanced levels of a brain growth factor which protects nerve cell functioning
- reduced the amount of amyloid plaques in the brain linked with Alzheimer's
- reduced both chronic inflammation and oxidative stress
- slowed down the rate of nerve cell loss

Professor Holscher said: "These very promising outcomes demonstrate the efficacy of these novel multiple receptor drugs that originally were developed to treat type 2 diabetes but have shown consistent neuro- protective effects in several studies."

"Clinical studies with an older version of this drug type already showed very promising results in people with Alzheimer's disease or with mood disorders"

"Here we show that a novel triple receptor drug shows promise as a potential treatment for Alzheimer's but further dose-response tests and direct comparisons with other drugs have to be conducted in order to evaluate if this new drugs is superior to previous ones."

Type 2 diabetes is a risk factor for Alzheimer's and has been implicated in the progression of the disease. Impaired insulin has been linked to cerebral degenerative processes in type 2 diabetes and Alzheimer's disease. Insulin desensitisation has also been observed in the Alzheimer's disease brain.

The desensitisation could play a role in the development of neurodegenerative disorders as insulin is a growth factor with neuroprotective properties.

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目次に戻る

3. ビフィズス菌や繊維は、内結腸粘液層の劣化を防ぐ -マウス実験

2018 年 1 月 2 日

食物繊維は栄養の重要な源泉であり、その量は体重、血糖値、インスリン感受性に影響する。*Cell Host & Microbe* 誌に掲載された University of Gothenburg の Sahlgrenska Academy の研究者らの最新の研究では、結腸の健康状態にも影響があることが示されている。

今回の研究において、低線維食を与えられたマウスで、細菌浸透性の増加、炎症性腸疾患および他の障害に対する潜在的危険性を示す腸粘液層の欠陥を発生させた、としている。また、別の実験では、繊維が枯渇した飼料を与えられたマウスが、正常に飼育されたマウスの腸内細菌の移植を受けた時、失われていた保護効果の一部を回復した、としている。

英文記事：

https://www.sciencedaily.com/releases/2018/01/180102103308.htm

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Bifidobacterium or fiber protect against deterioration of the inner colonic mucus layer

Date:

January 2, 2018

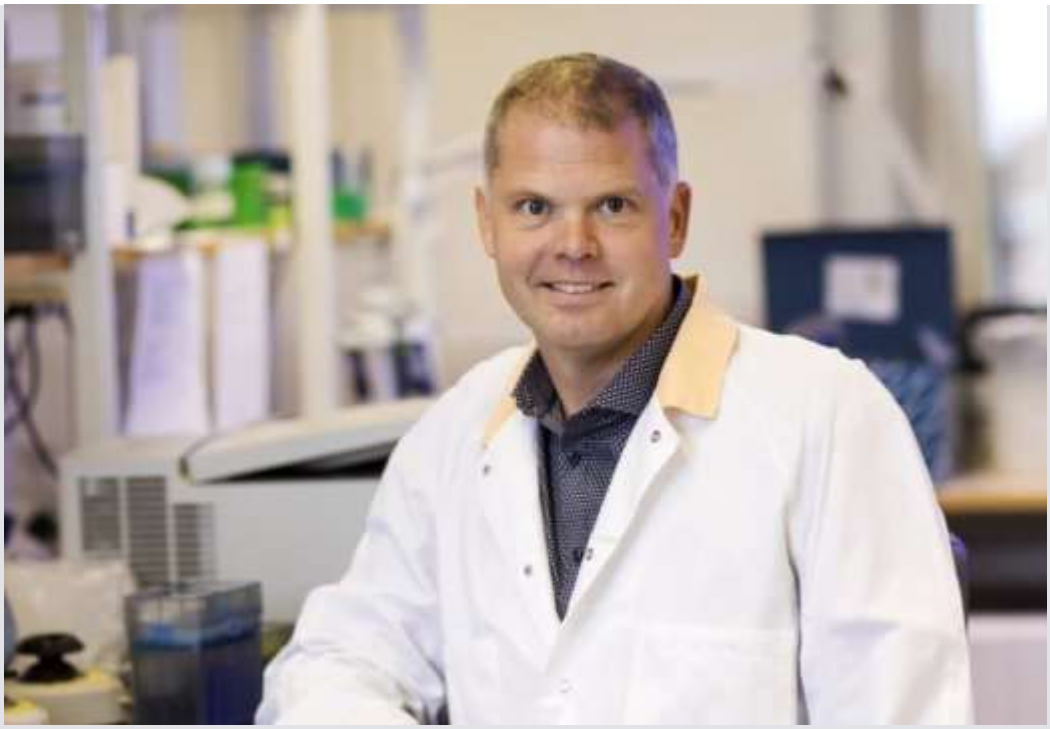
Source:

University of Gothenburg

Summary:

If you are concerned about your health, you should also think about what your gut bacteria consume. Dietary fiber is a key source for their nutrition. Thus the quantity of fiber in your diet influences your weight, blood glucose level and sensitivty to insulin is well-established. The latest research shows that colonic health is also affected.

FULL STORY



This is professor Fredrik Bäckhed, Sahlgrenska Academy, Sweden.

Credit: Johan Wingborg, University of Gothenburg

If you are concerned about your health, you should also think about what your gut bacteria consume. Dietary fiber is a key source for their nutrition. Thus the quantity of fiber in your diet influences your weight, blood glucose level and sensitivty to insulin is well-established. The latest research from Sahlgrenska Academy shows that colonic health is also affected.

Cell Host & Microbe recently published a study by scientists at Sahlgranska Academy to clarify the mechanisms for how fiber contributes to colonic health. Meanwhile, many people in contemporary society appear to be heading in a different direction altogether.

"Average fiber consumption has declined drastically in developed countries over the past few decades," Fredrik Bäckhed, Professor of Molecular Medicine, says. He studies the role of gut bacteria in metabolic disorders.

Various kinds of fiber are found in fruit, legumes, vegetables and whole grain products. Insufficient fiber consumption combined with a high-fat, high-carbohydrate diet is associated with a greater risk of inflammatory bowel disease, weight gain and diabetes.

Mice in the current study were put on a low-fiber diet. They developed defects in the inner colonic mucus layer after only three days characterized by increased bacterial penetrability, a potential risk for inflammatory bowel disease and other disorders.

"Our results demonstrate that the inner mucus layer separate gut bacteria from the body's cells," Gunnar C. Hansson, Professor of Medical and Physiological Chemistry and director of the study, says. "We clearly illustrated the rapid, process by which the mucus layer responds to dietary modifications and subsequent bacterial changes."

In a second experiment, the mice fed fiber-depleted diet received a transplant of gut bacteria from a normally fed animal and regained some of the lost protective effect.

A dietary supplement of friendly bifidobacteria stimulated growth of the mucus layer but did not prevent bacteria in the gut microbiota from approaching the body's cells. A supplement of inulin, a type of dietary fiber, addressed the latter problem but not the former.

"Low-fiber diets alter bacterial composition and influence what they produce," Professor Hansson says. "The result can be greater penetrability that affects the body's cells."

The researchers believe that fiber supplements as a method of treatment need to be investigated further. Simply enriching food with refined fiber is not recommended before more has been learned about its complex interplay with food, bacteria and the body's cells.

Story Source:

Materials provided by **University of Gothenburg**. *Note: Content may be edited for style and length.*

Journal Reference:

1. Bjoern O. Schroeder, George M.H. Birchenough, Marcus Ståhlman, Liisa Arike, Malin E.V. Johansson, Gunnar C. Hansson, Fredrik Bäckhed. **Bifidobacteria or Fiber Protects against Diet-Induced Microbiota-Mediated Colonic Mucus Deterioration**. *Cell Host & Microbe*, 2017; DOI: 10.1016/j.chom.2017.11.004

Cite This Page:

- MLA
- APA
- Chicago

University of Gothenburg. "Bifidobacterium or fiber protect against deterioration of the inner colonic mucus layer." ScienceDaily. ScienceDaily, 2 January 2018. <www.sciencedaily.com/releases/2018/01/180102103308.htm>.

目次に戻る

４．アルコールが DNA に損傷を与え癌リスクを高める仕組み -マウス研究

2018 年 1 月 3 日

アルコールとがんの関係が明らかに　DNA を損傷、2 度と戻らない状態に

1/9(火) 18:34 配信

Newsweek

アルコールとがんの関係が明らかに　luna4 -iStock

英ケンブリッジ大学の研究チームが、アルコールの摂取が、*DNA* を損傷してがんのリスクを高めると発表した

■アルコールが DNA を損傷

1 年で最もお酒を飲む機会が多くなると思われる年末年始のこの時期、お酒を愛する人たちにとって気になるニュースが報じられた。アルコールが、DNA を損傷してがんのリスクを高めるというのだ。英ケンブリッジ大学のケタン・パテル教授率いるチームが、英 MRC 分子生物学研究所で行なった研究について、科学誌「ネイチャー」に発表した。

「筋トレは、がんによる死亡リスクを 31%下げる」との研究結果

これまでも、アルコールの摂取ががんのリスクを高めることは指摘されてきた。アルコールを摂取すると、分解する過程でアセトアルデヒドが生成される。このアセトアルデヒドが DNA を損傷することは、培養細胞を使った研究で確認されていたのだ。しかしそのメカニズムははっきり分かっていなかった。今回初めて、パテル教授のチームがマウスを使い、生きている臓器の反応を確認。納得いく説明ができるようになったという。

パテル教授は、「がんの中には、幹細胞の DNA の損傷が原因でできるものもある。DNA の損傷はたまたま起こる場合もあるが、今回の研究は、アルコールが損傷リスクを高める可能性があると示唆している」と、MRC 分子生物学研究所に話している。研究チームがマウスにエタノールを投与したところ、エタノールが造血幹細胞の DNA 二重鎖を切断。細胞内の DNA 配列は、元に戻らない状態に壊されてしまったという。

■アジア人はアルコール分解がうまく機能せず

MRC 分子生物学研究所の発表文によると、人間は通常、アルコールからのダメージに対して 2 つの自己防衛機能を備えている。1 つは、アルコールを分解する過程で生成されるアセトアルデヒドに対するもの。アセトアルデヒド脱水素酵素（ALDH）が、有害なアセトアルデヒドを酢酸に分解し、細胞のエネルギー源に変える。今回の研究では、ALDH の一種、ALDH2 が欠如したマウスにアルコール（エタノール）を投与したところ、ALDH2 が機能しているマウスと比べ、DNA の損傷は 4 倍に達した。

研究チームは、この酵素が十分でなかったり欠陥があったりする人は、東南アジア人に特に多いと指摘。科学系ニュースサイトのサイエンス・アラートはこれを受けて、ALDH2 が変異している人

（つまりうまく機能しない人）の数は、アジアに 5 億 4000 万人いると具体的な数字を挙げている。

2 つめの防衛機能は、DNA の修復だ。しかしこれが常に機能するわけでもなく、中にはうまく機能しない人もいると研究チームは説明している。

■「安全な飲酒量などない」

パテル教授は、アルコールを効果的に処理できないことが、DNA 損傷のリスクを高め、特定のがんにつながる可能性があるということが今回の研究で強調された、と発表文の中で述べている。ただし、「アルコール処理や DNA 修復のシステムは完璧ではなく、こうした自己防衛機能がきちんと作用している人であっても、アルコールが原因でがんができる可能性はあることを忘れてはならない」と注意を促している。

英国のがん研究所は、アルコールとの関係が特に指摘されているがんの種類として、口腔がん、咽頭がん、食道がん、乳がん、肝臓がん、大腸がんを挙げている。そのリスクは、ワインやビール、蒸留酒などアルコールの種類とは無関係で、飲む量についても「がんに関しては安全な飲酒量などない」と断言している。ただし、英国には政府が定めた飲酒のガイドラインがあり、ここで規定している量以下であればリスクは低くなる、とがん研究所は述べている。

英国政府のガイドラインが推奨する飲酒量は、1 週間で 14 ユニット以内（1 ユニットは純アルコール 8 グラムなので 14 ユニットで 112 グラム）。英紙インディペンデントによるとこれは、4%程度のビールなら 7 パイント（約 3.3 リットル）、12%程度のワインなら通常のワイングラス（125ml）で 9 杯と 1/3 杯に相当する。

なお、厚生労働省は「節度ある適度な飲酒」を「1 日平均純アルコールで 20 グラム程度」としており、1 週間分（7 日）に換算すると英国ガイドラインより多くなっている。がんのリスクを考えて飲酒するなら、少なめに設定している英国のガイドラインも考慮に入れた方が良さそうだ。
松丸さとみ

https://headlines.yahoo.co.jp/article?a=20180109-00010003-newsweek-int&p=1

英文記事：
https://www.sciencedaily.com/releases/2018/01/180103132629.htm

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How alcohol damages DNA and increases cancer risk

Date:

January 3, 2018

Source:

Cancer Research UK

Summary:

Scientists have shown how alcohol damages DNA in stem cells, helping to explain why drinking increases your risk of cancer, according to new research.

FULL STORY

Alcohol bottle and glass (stock image).

Credit: © Ruslan Mitin / Fotolia

Scientists have shown how alcohol damages DNA in stem cells, helping to explain why drinking increases your risk of cancer, according to research part-funded by Cancer Research UK and published in *Nature* today (Wednesday).

Much previous research looking at the precise ways in which alcohol causes cancer has been done in cell cultures. But in this study, researchers have used mice to show how alcohol exposure leads to permanent genetic damage.

Scientists at the MRC Laboratory of Molecular Biology, Cambridge, gave diluted alcohol, chemically known as ethanol, to mice. They then used chromosome analysis and DNA sequencing to examine the genetic damage caused by acetaldehyde, a harmful chemical produced when the body processes alcohol.

They found that acetaldehyde can break and damage DNA within blood stem cells leading to rearranged chromosomes and permanently altering the DNA sequences within these cells.

It is important to understand how the DNA blueprint within stem cells is damaged because when healthy stem cells become faulty, they can give rise to cancer.

These new findings therefore help us to understand how drinking alcohol increases the risk of developing 7 types of cancer including common types like breast and bowel.

Professor Ketan Patel, lead author of the study and scientist, part funded by Cancer Research UK, at the MRC Laboratory of Molecular Biology, said: "Some cancers develop due to DNA damage in stem cells. While some damage occurs by chance, our findings suggest that drinking alcohol can increase the risk of this damage."

The study also examined how the body tries to protect itself against damage caused by alcohol. The first line of defence is a family of enzymes called aldehyde dehydrogenases (ALDH). These enzymes break down harmful acetaldehyde into acetate, which our cells can use as a source of energy.

Worldwide, millions of people, particularly those from South East Asia, either lack these enzymes or carry faulty versions of them. So, when they drink, acetaldehyde builds up which causes a flushed complexion, and also leads to them feeling unwell.

In the study, when mice lacking the critical ALDH enzyme -- ALDH2 -- were given alcohol, it resulted in four times as much DNA damage in their cells compared to mice with the fully functioning ALDH2 enzyme.

The second line of defence used by cells is a variety of DNA repair systems which, most of the time, allow them to fix and reverse different types of DNA damage. But they don't always work and some people carry mutations which mean their cells aren't able to carry out these repairs effectively.

Professor Patel added: "Our study highlights that not being able to process alcohol effectively can lead to an even higher risk of alcohol-related DNA damage and therefore certain cancers. But it's important to

remember that alcohol clearance and DNA repair systems are not perfect and alcohol can still cause cancer in different ways, even in people whose defence mechanisms are intact."

This research was funded by Cancer Research UK, Wellcome and the Medical Research Council (MRC).

Professor Linda Bauld, Cancer Research UK's expert on cancer prevention, said: "This thought-provoking research highlights the damage alcohol can do to our cells, costing some people more than just a hangover.

"We know that alcohol contributes to over 12,000 cancer cases in the UK each year, so it's a good idea to think about cutting down on the amount you drink."

Story Source:

Materials provided by **Cancer Research UK**. *Note: Content may be edited for style and length.*

Journal Reference:

1. Juan I. Garaycoechea, Gerry P. Crossan, Frédéric Langevin, Lee Mulderrig, Sandra Louzada, Fentang Yang, Guillaume Guilbaud, Naomi Park, Sophie Roerink, Serena Nik-Zainal, Michael R. Stratton, Ketan J. Patel. **Alcohol and endogenous aldehydes damage chromosomes and mutate stem cells**. *Nature*, 2018; DOI: 10.1038/nature25154

Cite This Page:

- MLA
- APA
- Chicago

Cancer Research UK. "How alcohol damages DNA and increases cancer risk." ScienceDaily. ScienceDaily, 3 January 2018. <www.sciencedaily.com/releases/2018/01/180103132629.htm>.

目次に戻る

5. 長時間作用して毒性も低い新型 HIV 薬がヒト化マウスでウィルスを抑制

2018 年 1 月 8 日

エール大学の研究チームは、HIV を抑制し免疫細胞を保護、さらに単回投与で数週間有効な新化合物について、HIV 治療改善の可能性を秘めているとして、米国科学アカデミー紀要で発表した。
国立衛生研究所の助成金によって支えられたこの研究は、ヒト血液細胞を移植され HIV に感染したマウスで行われた。
ヒト化マウスで、化合物は HIV 治療の重要な目標を達成：すなわち血液中のウィルスを検出できないほどに抑え、ウィルスに感染する免疫細胞を保護し、認可された HIV 薬と相乗的に働いた、としている。更に、この薬剤の単回投与の効果は、ほぼ 1 か月持続することも見出した。

英文記事：
https://www.healthcanal.com/infections/hiv-and-aids/242781-new-long-acting-less-toxic-hiv-drug-suppresses-virus-humanized-mice.html

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New long-acting, less-toxic HIV drug suppresses virus in humanized mice

3:55 January 8, 2018

A team of Yale researchers tested a new chemical compound that suppresses HIV, protects immune cells, and remains effective for weeks with a single dose. In animal experiments, the compound proved to be a promising new candidate to enhance current HIV treatment regimens — without increasing toxic side effects, the researchers said.

The finding builds on the work of senior co-authors Karen S. Anderson and William L. Jorgensen, who used computational and structure-based design methods to develop a class of compounds, that target a viral protein essential for HIV to replicate. The researchers refined this class of compounds to boost potency, lower toxicity, and improve drug-like properties in order to identify a promising preclinical drug candidate. In collaboration with Priti Kumar's lab at Yale, the drug candidate was tested in mice with transplanted human blood cells and infected with HIV.

In the humanized mice, the compound achieved key goals of HIV treatment: It suppressed the virus to undetectable levels in the blood; it protected the immune cells that the virus infects; and it worked synergistically with approved HIV medications, the researchers said.

Additionally, working with Yale drug delivery expert Mark Saltzman and his laboratory, the researchers found that the effects of a single dose of the compound — delivered in a long-acting nanoparticle form — lasted for nearly a month.

While further testing is needed, the compound has potential for improving treatment for HIV, which affects 37 million people worldwide, said Anderson. "Our drug candidate works synergistically with all current classes of HIV drugs, as well as some that are also being tested in clinical trials. It enhances their potency and could be a better combination medication."

Other Yale authors are Shalley N. Kudalkar, Jagadish Beloor, Elias Quijano, Krasimir A. Spasov, Won-Gil Lee, and José A. Cisneros.

The study, published by Proceedings of the National Academy of Sciences (PNAS), was supported by National Institute of Health grants.

Yale University

目次に戻る

6. 皮膚細胞からヒト筋肉育成に初めて成功

2018 年 1 月 9 日

デューク大学の生物医学の技術者らは、一過性の Pax7 過剰発現と 3 次元培養を介する手順により、非筋肉細胞 -元の幹細胞状態に戻された皮膚細胞- から機能するヒト筋肉を作成、これを成体マウスに移植し、少なくとも 3 週間は機能することを示した。これは、2015 年にデューク大学が発表した研究で、筋肉生検から得た細胞からヒト筋肉組織を成長させた研究成果に基づいている。
1 月 9 日の *Nature Communications* 誌に掲載されたこの研究は、筋肉以外の組織を用いて細胞のところから始めることができたことは、筋肉細胞をはるかに増殖させ、ゲノム編集や細胞治療への道を開き、希少筋肉疾患モデルを個別に開発することを可能にするものだとしている。

英文記事：
http://www.labmanager.com/news/2018/01/engineers-grow-functioning-human-muscle-from-skin-cells#.WmeKtExFxEw

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Engineers Grow Functioning Human Muscle From Skin Cells

First functioning human muscle grown from induced pluripotent stem cells holds promise for cellular therapies, drug discovery, and studying rare diseases

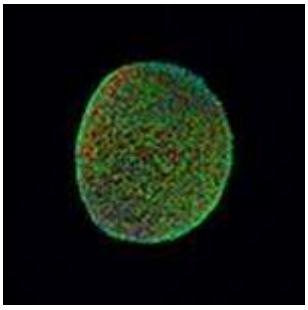
By Duke University | January 09, 2018

Duke engineers have grown the first functioning human muscle from non-muscle cells -- skin cells reverted to their primordial stem cell state. The advance holds promise for cellular therapies, drug discovery and studying rare diseases. Video Credit: Duke University

Biomedical engineers have grown the first functioning human skeletal muscle from induced pluripotent stem cells.

The advance builds on work published in 2015 when researchers at Duke University grew the first functioning human muscle tissue from cells obtained from muscle biopsies. The ability to start from cellular scratch using non-muscle tissue will allow scientists to grow far more muscle cells, provide an easier path to genome editing and cellular therapies, and develop individually tailored models of rare muscle diseases for drug discovery and basic biology studies.

The results appear online in *Nature Communications*.



A cross section of a muscle fiber grown from induced pluripotent stem cells. The green indicates muscle cells, the blue is cell nuclei, and the red is the surrounding support matrix for the cells.

IMAGE CREDIT: DUKE UNIVERSITY

"Starting with pluripotent stem cells that are not muscle cells, but can become all existing cells in our body, allows us to grow an unlimited number of myogenic progenitor cells," said Nenad Bursac, professor of biomedical engineering at Duke University. "These

progenitor cells resemble adult muscle stem cells called 'satellite cells' that can theoretically grow an entire muscle starting from a single cell."

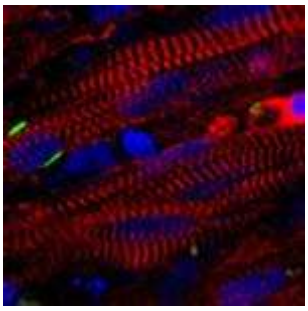
In their previous work, Bursac and his team started with small samples of human cells obtained from muscle biopsies, called "myoblasts," that had already progressed beyond the stem cell stage but hadn't yet become mature muscle fibers. They grew these myoblasts by many folds and then put them into a supportive 3-D scaffolding filled with a nourishing gel that allowed them to form aligned and functioning human muscle fibers.

In the new study, the researchers instead started with human induced pluripotent stem cells. These are cells taken from adult non-muscle tissues, such as skin or blood, and reprogrammed to revert to a primordial state. The pluripotent stem cells are then grown while being flooded with a molecule called Pax7 -- which signals the cells to start becoming muscle.

As the cells proliferated they became very similar to --but not quite as robust as -- adult muscle stem cells. While previous studies had accomplished this feat, nobody has been able to then grow these intermediate cells into functioning skeletal muscle.

The Duke researchers succeeded where previous attempts had failed.

"It's taken years of trial and error, making educated guesses and taking baby steps to finally produce functioning human muscle from pluripotent stem cells," said Lingjun Rao, a postdoctoral researcher in Bursac's laboratory and first author of the study. "What made the difference are our unique cell culture conditions and 3-D matrix, which allowed cells to grow and develop much faster and longer than the 2-D culture approaches that are more typically used."



A stained cross section of the new muscle fibers. The red cells are muscle cells, the green areas are receptors for neuronal input, and the blue patches are cell nuclei.

IMAGE CREDIT: DUKE UNIVERSITY

Once the cells were well on their way to becoming muscle, Bursac and Rao stopped providing the Pax7 signaling molecule and started giving the cells the support and nourishment they needed to fully mature.

In the study, the researchers show that after two to four weeks of 3-D culture, the resulting muscle cells form muscle fibers that contract and react to external stimuli such as electrical pulses and biochemical signals mimicking neuronal inputs just like native muscle tissue. They also implanted the newly grown muscle fibers into adult mice and showed that they survive and function for at least three weeks while progressively integrating into the native tissue through vascularization.

The resulting muscle, however, is not as strong as native muscle tissue, and also falls short of the muscle grown in the previous study that started from muscle biopsies. Despite this caveat, the researchers say this muscle still holds potential that the stronger, older relative does not.

The pluripotent stem cell-derived muscle fibers develop reservoirs of "satellite-like cells" that are necessary for normal adult muscles to repair damage, while the muscle from the previous study had much fewer of these cells. The stem cell method is also capable of growing many more cells from a smaller starting batch than the biopsy method.

Both of the advantages point toward a possibility of using this new method for regenerative therapies and for creating models of rare diseases for future studies and individualized health care.

"The prospect of studying rare diseases is especially exciting for us," said Bursac. "When a child's muscles are already withering away from something like Duchenne muscular dystrophy, it would not be ethical to take muscle samples from them and do further

damage. But with this technique, we can just take a small sample of non-muscle tissue, like skin or blood, revert the obtained cells to a pluripotent state, and eventually grow an endless amount of functioning muscle fibers to test."

The technique also holds promise for being combined with genetic therapies. Researchers could, in theory, fix genetic malfunctions in the induced pluripotent stem cells derived from a patient and then grow small patches of completely healthy muscle. While this could not heal or replace an entire body's worth of diseased muscle, it could be used in tandem with more widely targeted genetic therapies or to heal more localized problems.

The researchers are now refining their technique to grow more robust muscles and beginning work to develop new models of rare muscle diseases.

目次に戻る

7. メラノーマの新治療標的

マウス研究によって、まぜ妊婦が致命的皮膚癌からの生存優位性を有するのか説明

2018 年 1 月 16 日

ペンシルベニア大学医学部ペレルマン校の研究者らは、メラノーマのための新たな治療標的を特定した。何十年にもわたり、女性の性別と妊娠歴がメラノーマ診断後良い結果と関連しているとされてきたが、今回研究チームはメラノーマの予防効果の理由を明らかにし、今日の *eLife* 誌に発表している。
この疾患のメカニズムは、メラノサイトに見られる G タンパク質共役型エストロゲン受容体 (GPER) と呼ばれる細胞性タンパク質に関連しており、GPER が活性化され癌モデルのマウスにおいて高 PD-1 阻害剤と組み合わされた時、治療は全てのマウスの生存を劇的に延長し、又 50%において腫瘍を完全に排除した。この GPER は通常エストロゲン -この値は女性、特に妊婦で高い- によって活性化されるとしている。

英文記事：
https://www.sciencedaily.com/releases/2018/01/180116111149.htm

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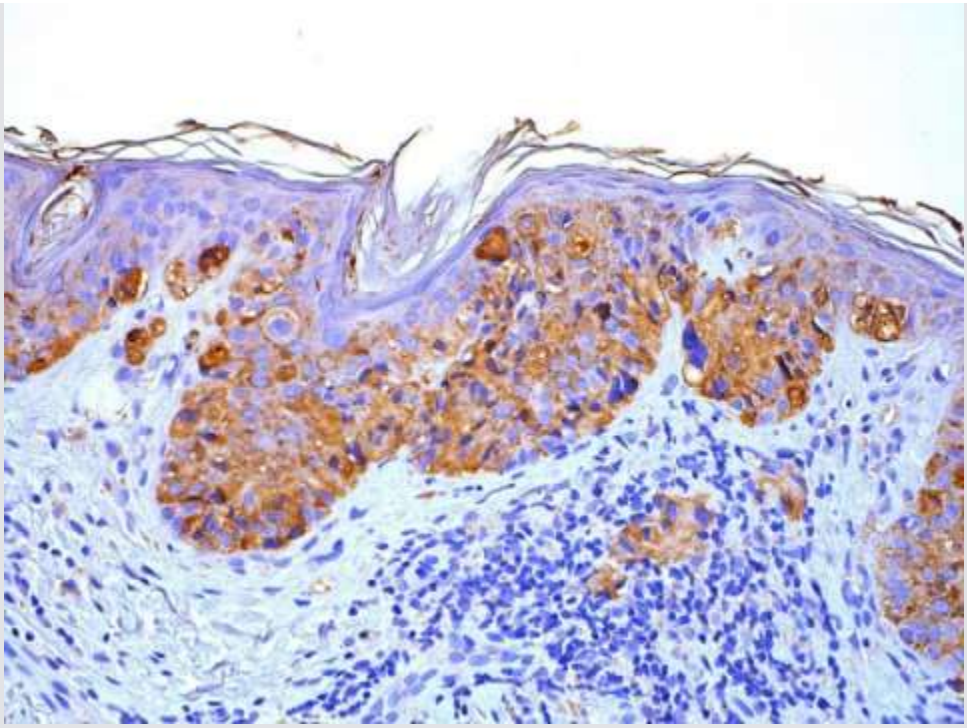
New treatment target for melanoma identified

Mouse research explains why women who have been pregnant may have survival advantage when facing deadly skin cancer

Date: January 16, 2018
Source: University of Pennsylvania School of Medicine

Summary: Researchers have identified a new therapeutic target for the treatment of melanoma. For decades, research has associated female sex and a history of previous pregnancy with better outcomes after a melanoma diagnosis. Now, a research team says it may have determined the reason for the melanoma-protective effect.

FULL STORY



GPER.

Credit: Penn Medicine

Researchers in the Perelman School of Medicine at the University of Pennsylvania have identified a new therapeutic target for the treatment of melanoma. For decades, research has associated female sex and a history of previous pregnancy with better outcomes after a melanoma diagnosis. Now, a research team from Perelman School of Medicine at the University of Pennsylvania says it may have determined the reason for the melanoma-

protective effect. The mechanism is related to a cellular protein called the G protein-coupled estrogen receptor (GPER). When GPER was activated and combined with anti PD-1inhibitor drugs in mouse cancer models, the therapy dramatically extended survival in all animals and completely eliminated the tumor in 50 percent of the mice. Researchers published their findings in the journal *eLife* today.

Melanoma is the deadliest form of skin cancer, despite accounting for only about one percent of skin cancers overall. Rates of melanoma have been rising for 30 years, and the American Cancer Society estimates there were more than 87,000 new cases in the United States in 2017. Even with recent advances in immunotherapy, the majority of patients with metastatic forms of melanoma will die from their disease.

"In melanoma and many other types of cancer, women have a better prognosis than men, and women with a history of pregnancy seem to have a better prognosis than those women that have never been pregnant" said the study's senior author Todd W. Ridky, MD, PhD, an assistant professor of Dermatology at Penn. "Decades of research certainly suggests that there is something about female sex and pregnancy hormones that helps protect against melanoma, but no one really understood how that might work."

Researchers say the key is GPER, a receptor found on melanocytes, which are pigment-producing cells in the skin. The receptor is normally activated by estrogen, which is higher in females, especially during pregnancy. Activation of GPER likely explains why many women notice that many areas of their skin gets darker during pregnancy. Previous research from the Ridky lab has shown the effects of GPER activation are totally different than the effects of classical estrogen receptor signaling, which is important in breast cancer. The team discovered that melanocytes do not even express the classical estrogen receptor, and that all estrogen effects were the result of GPER.

In melanoma specifically, once GPER is activated, the cancer cell becomes more differentiated. This means it divides less frequently, makes more pigment, and becomes more visible and vulnerable to the natural immune system. This makes it harder for the cancer to become resistant to immunotherapies.

No drugs specifically target GPER, but Ridky and his team used a lab compound called G-1, originally developed by Eric Prossnitz, PhD, at the University of New Mexico Comprehensive Cancer Center, to stimulate GPER in mice, and then used anti-PD-1 inhibitors to treat the melanoma. The approach eliminated the tumors in half of all mice. The authors note that anti-PD-1 inhibitors, when used alone in mice with melanoma, extend survival modestly, but do not completely eliminate tumors, and no animals survive long-term.

"We hope this work inspires other researchers to revisit old ideas of differentiation-based cancer therapies now that immune therapies are available," said the study's lead author Christopher A. Natale, a researcher in Ridky's lab. "It is clear that the future of cancer therapy lies in combination treatments, and differentiation drivers may be a very useful component in future cancer therapy regimens."

As Ridky points out, this represents a unique approach to immunotherapy and cancer therapy in general.

"So much of the cancer field is focused on inhibitors, but in this new treatment approach, we're actually activating something rather than blocking it," Ridky said. "We used a synthetic compound to mimic part of what happens naturally during pregnancy, and as a result, the GPER activator is very well tolerated without any obvious toxic side effects that are common with most cancer drugs."

Ridky also said this approach could be promising beyond melanoma.

"This is a receptor that is expressed in many organs, so there's a reasonable expectation that this may work in other tumor types too," Ridky said.

Although researchers did not observe any toxicities from the compound in mice, though they say they plan further toxicity studies before hopefully moving on to human trials.

Story Source:

Materials provided by **University of Pennsylvania School of Medicine**. *Note: Content may be edited for style and length.*

Journal Reference:

1. Christopher A Natale, Jinyang Li, Junqian Zhang, Ankit Dahal, Tzvete Dentchev, Ben Z Stanger, Todd W Ridky. **Activation of G protein-coupled estrogen receptor signaling inhibits melanoma and improves response to immune checkpoint blockade**. *eLife*, 2018; 7 DOI: 10.7554/eLife.31770

Cite This Page:

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- Chicago

University of Pennsylvania School of Medicine. "New treatment target for melanoma identified: Mouse research explains why women who have been pregnant may have survival advantage when facing deadly skin cancer." ScienceDaily. ScienceDaily, 16 January 2018. <www.sciencedaily.com/releases/2018/01/180116111149.htm>.

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8. サンフランシスコ ベイエリアに新たなバイオテック集積地

2018 年 1 月 16 日

カリフォルニア州には、サンディエゴとサンフランシスコ ベイエリアという 2 つの主要なバイオテクノロジー拠点がある。カリフォルニア生命科学協会のサラ・ラドクリフ社長兼 CEO によると、2015 年と 2016 年には、ベイエリアがライフサイエンス分野における雇用地域として第 1 位であったこと、2017 年はベイエリア諸郡のライフサイエンス雇用が州全体の 24%であったこと、設立された企業のほとんどが確実に成長を続けている、としている。
また、さらに多くのライフサイエンス企業を構築するための投資がベイエリア全体で継続しており、サンフランシスコ市の南に位置するサンカルロス市 (弊社所在地サニーベールから車で北に約 30 分) が新たなバイオテック集積地として歩み始めた。というのも、バイオテック企業に入居施設を提供している不動産会社 Alexandria Real Estate Equities がサンカルロスに 10 万平方メートルほどの土地を購入し、駆け出しのバイオテック企業の巣立ちまでを助けるインキュベーター施設が先月許可された。サンカルロスがバイオテクノロジー発展を受け入れる機運も静かに高まっており、新たな市場が築かれつつあるとサンカルロス市地域開発担当者 Al Savay 氏も言っている。

英文記事：
https://www.biospace.com/article/unique-new-biotech-hub-is-emerging-in-the-bay-area/?keywords=san+carlos

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Look Out: A New Biotech Hub is Emerging in the Bay Area

Published: Jan 16, 2018 By Alex Keown

It's no secret the Bay Area has been a stronghold of biotech innovation. Now it appears the area is about to get stronger as **more space for pharma and biotech companies** will soon be available down the Peninsula south of San Francisco in the city of San Carlos.

The new space for biotechs is available following investments from **Alexandria Real Estate Equities** in the city. The investment group acquired about one million square feet of office space. Alexandria is well-known for its deals with biotech companies. According to *The Daily Journal,* Alexandria acquired two buildings that both provide more than 500,000 square feet of office space in San Carlos.

The two office buildings acquired by Alexandria aren't the only indications the city is setting itself up as a potential biotech hub. The *Daily Journal* reported that QB-3 Biolabs and Dewey Land Company have acquired space in the city that will allow for the creation of a biotech incubator. The two companies also launched an incubator in San Francisco about four years ago, the *Journal* said. San Carlos Community Development Director **Al Savay** told the *Journal* that the city has "quietly been gaining momentum in the area of biotechnology growth."

While California is home to two major biotech hubs, in San Diego and the San Francisco Bay Area, **Sara Radcliffe**, president and CEO of the **California Life Sciences Association**,

told the *Journal* that growth does not appear to be slowing across the region. In 2015 and 2016, the Bay Area was the number one region in total life sciences employment.

"This last year, Bay Area counties employed 72,663 people in life sciences — 24 percent of the state's total life sciences employment. Established companies continue to grow and investments in building more life science companies have continued throughout the Bay Area," Radcliffe told the *Journal*.

Alexandria has had a busy start to 2018. At the **J.P. Morgan Healthcare Conference** in San Francisco, the group announced the launch of a new fund. Alexandria Venture Investments launched the Alexandria Seed Capital Platform, a novel and innovative seed-stage life science funding model. Alexandria Venture Investments will vet seed-stage companies of interest through the Seed Capital Platform and guide milestone-driven investments based on the quality and differentiation of a company's technology, foundational IP, business strategy, financing plan and management team, the company said in a Jan. 8 statement.

Alexandria has expanded its real estate holdings across the United States with facilities in North Carolina, New York and more. Last year, Alexandria Real Estate Equities, Inc. opened the new Alexandria LaunchLabs at the Alexandria Center for Life Science in New York City. The new innovation center includes 15,000 square feet of lab and office space that is currently the home to 13 startup companies. At capacity, the facility is expected to house about 25 companies.

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9．癌研究で使用されるマウスはどれだけヒトに近いのか？

2018 年 1 月 18 日

NIH などに資金提供を受けて行われたミシガン州立大学の研究は、癌研究に関してマウスがヒトにどれだけ近いのかという科学者の疑問に答えるものとなっている。
癌はアメリカにおける死因では、心臓病に続く第 2 位となっているが、*PLOS Genetics* 誌に掲載されたこの知見によると、マウスが実際に、ヒトの乳癌組織やその遺伝子を、肺癌、口腔癌、食道癌と同様に、以前考えられていた以上に、模倣できることを明らかにしている。
例えばヒトの乳癌では多くの亜型が存在するが、この研究では全てのサブタイプを含むマウスを調べ、マウスの腫瘍の構成と遺伝子の作用をヒト腫瘍データと比較し、特定の乳癌において遺伝子が同じように作用するだけでなく、他の癌においても遺伝子類似性が活発であることを見出した。

英文記事：
https://www.sciencedaily.com/releases/2018/01/180118142647.htm

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Can mice really mirror humans when it comes to cancer?

Date: January 18, 2018

Source: Michigan State University

Summary: A new study is helping to answer a pressing question among scientists of just how close mice are to people when it comes to researching cancer. The findings reveal how mice can actually mimic human breast cancer tissue and its genes,

even more so than previously thought, as well as other cancers including lung, oral and esophagus.

FULL STORY

A new Michigan State University study is helping to answer a pressing question among scientists of just how close mice are to people when it comes to researching cancer.

The findings, now published in *PLOS Genetics*, reveal how mice can actually mimic human breast cancer tissue and its genes, even more so than previously thought, as well as other cancers including lung, oral and esophagus.

According to the Centers for Disease Control, cancer is the second leading cause of death among Americans next to heart disease.

"Just like human breast cancer, there are many subtypes that can be found in mice," said Eran Andrechek, co-author and physiology professor whose work focuses on the genetic makeup of cancer. "Our work outlines the genetic similarities of the tissue and cells in different types of tumors and shows the strong relationships mice can have to other human cancers too."

Different tumor subtypes can include glandular, which include the mammary glands, as well as squamous, which are very rare and involve epithelial cells that line the inside of the breast.

Andrechek's federally funded study looked at mice containing all subtypes and compared the makeup of the rodent tumors and the way the genes acted, known as gene expression, to human tumor data.

He found that not only did the genes act the same in certain breast cancers but the gene similarities were active in other cancers as well.

"Groups of genes were also being expressed similarly in the lung, oral and esophageal tumors," Andrechek said. "For example, mouse mammary tumors shared a signaling pathway that is found in human lung cancer and controls how cells reproduce and move from one location to another."

Because tumors have distinct genes, the way they act or send signals can help scientists identify and define the specific kind of cancer they're dealing with in hopes of finding the right treatment.

"Our work will help scientists understand in part what makes the various tumors so unique and such a challenge to treat," Andrechek said. "But even more importantly, for patients, our ability to identify the similarities could allow treatments for other cancers like lung to be used for certain breast cancers down the road."

The study was funded by the National Institutes of Health and Worldwide Cancer Research.

Story Source:

Materials provided by **Michigan State University**. *Note: Content may be edited for style and length.*

Journal Reference:

1. Daniel P. Hollern, Matthew R. Swiatnicki, Eran R. Andrechek. **Histological subtypes of mouse mammary tumors reveal conserved relationships to human cancers**. *PLOS Genetics*, 2018; 14 (1): e1007135 DOI: 10.1371/journal.pgen.1007135

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Michigan State University. "Can mice really mirror humans when it comes to cancer?." ScienceDaily. ScienceDaily, 18 January 2018. <www.sciencedaily.com/releases/2018/01/180118142647.htm>.

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10. 今年注目すべきバイオテック新興企業 20 社～BioSpace

2018 年 1 月 2 日

BioSpace は、2015 年以降に設立された北米バイオテック新興企業について今年注目すべき 20 社のリストである NextGen Bio “Class of 2018”を発表。
このトップ 20 をはじき出すために、企業をその年齢グループに分類し、次にそれらのグループを異なるいくつかのカテゴリー、「ファイナンス」「コラボレーション」「パイプライン」「売り上げ」「編集」、で重み付けをして最終的に与えられたポイントを累積してランク付けを行っている。

英文記事：
https://www.biospace.com/article/exclusive-top-20-life-science-startups-to-watch-in-2018/?keywords=+bio+startup+2018

Top 20 Life Science Startups to Watch in 2018

Published: Jan 02, 2018 By Mark Terry

BioSpace is proud to present its NextGen Bio "Class of 2018," a list of 20 up-and-coming life science companies in North America that started up no earlier than 2015. To come up with this Top 20, BioSpace sorted companies into that age grouping, and they were then weighted by a number of different categories and finally ranked in a cumulative fashion, based on the points awarded for each category. These categories were: Finance, Collaborations, Pipeline, Sales and Editorial (view methodology below). The NextGen Bio Class of 2018 is a stellar group of companies that are already making an enormous impact on the industry now and will into the future. Congratulations to this group!

RANK	COMPANY	FOUNDED	LOCATION	POINTS
1	BlueRock Therapeutics	2016	Massachusetts	37
2	Prelude Fertility	2016	Georgia	29
3	Relay Therapeutics	2016	Massachusetts	16
4	Freenome	2016	California	15
5	Cirius Therapeutics	2016	California	12
6	Boragen	2017	North Carolina	11

7	Hyalex Orthopaedics	2017	Massachusetts	11
8	CBT Pharmaceuticals	2016	California	9
9	Corbin Therapeutics	2016	Canada	9
10	Tango Therapeutics	2017	Massachusetts	8
11	Shephard Therapeutics	2016	Massachusetts	8
12	CARMA Therapeutics	2016	Pennsylvania	6
13	Jecure Therapeutics	2017	California	5
14	Indalo Therapeutics	2017	Missouri	5
15	Fortis Therapeutics	2016	California	4
16	Surrozen	2017	California	4
17	Immune-Onc	2016	California	3
18	Fibrocor Therapeutics	2017	Canada	3
19	Apostle	2017	California	1
20	DMD Therapeutics	2015	Washington	1

1. BlueRock Therapeutics

Points: 37
Founded: 2016
Location: Ontario, Canada; Cambridge, MA, New York, NY
Notable:
· In December 2016, BlueRock launched with a $225 million Series A financing, led by Bayer AG and Versant Ventures.
· BlueRock focuses on cell therapies to regenerate heart muscle in patients who have had a heart attack or have chronic heart failure, as well as in patients with Parkinson's diseases.

· BlueRock's programs are in collaboration with Toronto-based McEwen Centre for Regenerative Medicine and University Health Network (UHN) and New York-based Memorial Sloan Kettering Cancer Center (MSKCC), and a manufacturing platform partnership with Toronto-based CCRM.
· The overall approach is focused on iPSC intellectual property invented by Nobel Prize winner Shinya Yamanaka of Kyoto University (Japan) and licensed from iPS Academia Japan.

2. Prelude Fertility

Points: 29
Founded: 2016
Location: Atlanta, Ga.
Notable:
· Prelude Fertility was founded with a $200 million investment by entrepreneur Martin Varsavsky; the company's business model is aimed at in vitro fertilization and egg freezing.
· Started with an investment in the largest in vitro fertilization clinic in the Southeast, Reproductive Biology Associates of Atlanta, and its affiliate, My Egg Bank, the largest frozen donor egg back in the U.S.

3. Relay Therapeutics

Points: 16
Founded: 2016
Location: Cambridge, Mass.
Notable:
· Relay launched in September 2016 with a $57 million Series A financing, led by Third Rock Ventures with participation from D.E. Shaw Research.
· On Dec. 14, 2017, Relay closed on a Series B round worth $63 million. The round was led by BVF Partners, with new investors GV (formerly Google Ventures), Casdin Capital, EcoR1 Capital and Section 32. Existing investors Third Rock Ventures and Alexandria Venture Investments participated.
· The company focuses on the relationship between protein motion and function.

4. Freenome

Points: 15
Founded: 2016
Location: South San Francisco, Calif.
Notable:
· Closed a $65 million Series A round led by Andreessen Horowitz and joined by GV (Google Ventures), Polaris Partners, Charles River Ventures, Eric Schmidt's Innovation

Endeavors, Spectrum 28, and Asset Management Ventures. Previous investors included Data Collective and Founders Fund.

· Is utilizing an Adaptive Genomics Engine (AGE) to evaluate genetic health and diagnoses based on cell-free DNA data in the blood for early detective of cancer and other diseases.

· Working with the University of California, San Francisco, Moores Cancer Center at UC San Diego Health and Massachusetts General Hospital.

5. Cirius Therapeutics

Points: 12

Founded: 2016

Location: San Diego, Calif.

Notable:

· Formerly known as Octeta Therapeutics, launched with a $40 million Series A in April 2017. A previous $16 million was raised by Octeta.

· Company's lead product is MSDC-0602K, a second-generation insulin sensitizer to treat NASH, which is actively enrolling patients in a Phase IIb clinical trial for NASH and liver fibrosis.

6. Boragen

Points: 11
Founded: 2017
Location: Durham, N.C.
Notable:
· Launched with a $10 million Series A that included Alexandria Venture Investments, ARCH Venture Partners, Bayer, Bill & Melinda Gates Foundation, Elanco Animal Health, Flagship Pioneering, Hatteras Venture Partners, Mountain Group Capital, Pappas Capital and Syngenta Ventures.
· The company is a small molecule development company that focuses on the unique chemical properties of Boron chemistry for crop protection and animal health, and fungicides.

7. Hyalex Orthopaedics

Points: 11
Founded: 2017
Location:
Notable: Lexington, Mass.
· Launched in May 2017 with a $16 million Series A financing led by Canaan Partners with participation from Osage University Partners and Johnson & Johnson Innovation – JJDC.
· Utilizing technology licensed from Stanford University, Hyalex developed a synthetic biomaterial that can be used in knee joint replacement.

8. CBT Pharmaceuticals

Points: 9
Founded: 2016
Location: Pleasanton, Calif.
Notable:
· CBT closed closed on a $9.75 million Series A financing in August 2016 led by Orbimed Asia, which along with $5 million in seed funding from its parent company, Crown Bioscience International, brings its operating capital to $14.75 million.
· The company has four development-stage assets, including CBT-101, an oral c-Met inhibitor that targets the epithelial to mesenchymal transition (IEMT) pathway in cancers.
· CBT's CBT-501 began enrollment in a Phase I trial in March 2017 for solid tumors in the U.S.
· CBT-101 is in a Phase I clinical trial in China with its China partner, Beijing Pearl Biotechnology Co.

9. Corbin Therapeutics

Points: 9
Founded: 2016
Location: Montreal-Quest, Que.
Notable:

· Corbin spun out of AmorChem in July 2017 with $1 million (Canadian) in seed funding.
· All rights to the USP15 technology initially held by AmorChem was transferred to Corbin.
· Corbin signed an exclusive, worldwide, license with McGill University on a USP15-based drug discovery platform in order to screen compound libraries and identify the first USP15 inhibitor for multiple sclerosis or other inflammation-based diseases.

10. Tango Therapeutics

Points: 8
Founded: 2017
Location: Cambridge, Mass.
Notable:
· Tango launched in March 2017 with a $55 million series A from Third Rock Ventures.
· The company has a product engine based on DNA sequencing and CRISPR-based target discovery, with three areas of drug development: loss of tumor suppressor gene function, multiple oncogenic drivers, and immune evasion.

11. Shepherd Therapeutics

Points: 8
Founded: 2016
Location: Aliston, Mass.
Notable:
・Founded by David Hysong after being diagnosed with adenoid cystic carcinoma (ACC) with former Genzyme senior vice president Gene Williams, to focus on rare cancers.
・Pipeline is in preclinical development in collaboration with the National Cancer Institute (NCI), University College London, and the Massachusetts Institute of Technology (MIT).

12. CARMA Therapeutics

Points: 6
Founded: 2016
Location: Philadelphia, Penn.
Notable:
・Launched with an undisclosed amount of funding through Upstart within PCI Ventures at the University of Pennsylvania to commercialize new cellular therapies based on CAR-T.
・Financing was co-led by AbbVie Ventures and HealthCap with participation by Grazia Equity and IP Group.

13. Jecure Therapeutics

Points: 5
Founded: 2017
Location: San Diego, Calif.
Notable:
· Launched with a $20 million Series A round by Versant Ventures to develop programs for non-alcoholic steatohepatitis (NASH) and fibrosis.

14. Indalo Therapeutics

Points: 5
Founded: 2017
Locaion: St. Louis, Mo.
Notable:
· A $9 million venture funding in August 2017.
· Formed by the merger of Antegrin Therapeutics and Cascadian Therapeutics to focus on fibrosis.

15. Fortis Therapeutics

Points: 4
Founded: 2016
Location: La Jolla, Calif.
Notable:
· Founded with an $18 million Series A in September 2016 led by Avalon Ventures and joined by Bregua, Lilly Asia Ventures, Osage University Partners, and Vivo Capital.

16. Surrozen

Points: 4
Founded: 2017
Location: South San Francisco, Calif.
Notable:
· Closed a $33 million Series A financing led by The Column Group in February 2017.
· One of the company's founders, Roeland Nusse, the Virginia and Daniel K. Ludwig Professor of Cancer Research and Professor of Developmental BIology at Stanford University School of Medicine, won the 2017 Breakthrough Prize in Life Sciences for his work on the Wnt pathway.

17. Immune-Onc Therapeutics

Points: 3
Founded: 2016
Location: Palo Alto, Calif.
Notable:
· A Series A financing of $7 million in September 2016 by investors that include Fame Mount Limited and CLI Ventures.
· One of the company's founders is Charlene Liao, former project team leader at Genentech.

18. Fibrocor Therapeutics

Points: 3
Founded: 2017
Location: Toronto, Ont.
Notable:
· Founded in January 2017 by Evotech AG and MaRS Innovation with $2.1 million in financing to focus on fibrosis.

19. Apostle

APOSTLE

Points: 1
Founded: 2017
Location: Sunnyvale, Calif.
Notable:
· Raised $2.35 million in one round from Amino Capital, ShangBay Capital, Westlake Ventures, and individual investors.
· Co-founder is David Dongliang Ge, former president of BioSciKin and former director of Bioinformatics at Gilead Sciences.
· Focused on developing a bioinformatics-enabled nanotechnology aimed for early cancer detection.

20. DMD Therapeutics

DMD Therapeutics

Points: 1
Founded: 2015
Location: Seattle, Wash.
Notable:
· Raised $400,000 seed money from Ryan's Quest, Michael's Cause and Pietro's Fight, to work on a potential treatment for Duchenne muscular dystrophy (DMD).
· Company's lead candidate, DMD-813, has reduced inflammation and muscle damage in mice.

· Company was spun out of the University of Washington by biotech entrepreneur Ron Berenson.

Check out last year's top 20 life science startups: NextGen Bio "Class of 2017."

Methodology: How We Ranked the "NextGen Bio Class of 2018"

· Finance: One (1) point was awarded for each $10 million in seed financing or seed money that was raised.

· Collaborations: Two (2) points were awarded if the company was a spinout from a well-known company or institution, had signed developmental or commercial agreements or partnerships. Two points for each collaboration.

· Pipeline: Two (2) points were given if the company had a compound or device in an ongoing clinical trial. Two points for each compound or device.

· Sales: Five (5) points were added if the company had an actual product or service to sell.

· Editorial: The editor awarded up to ten (10) points to some companies for either particularly interesting areas of science and technology, or for working in a particularly broad and open market. Factors such as awards for a product, founder or scientists were also taken into account.