BIO NEWS

March, 2017

In-Vivo Science International Inc.
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Sunnyvale, CA 94089 USA

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米国科学アカデミー（National Academy of Sciences：NAS）は、2017 年 2 月 14 日、重篤な遺伝性疾患を予防する目的に限り、生殖細胞のゲノム編集を

認めるとする報告書「Human Genome Editing: Science, Ethics, and Governance」を公表した。

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1．薬物検査で死亡する実験動物を減少させる方法

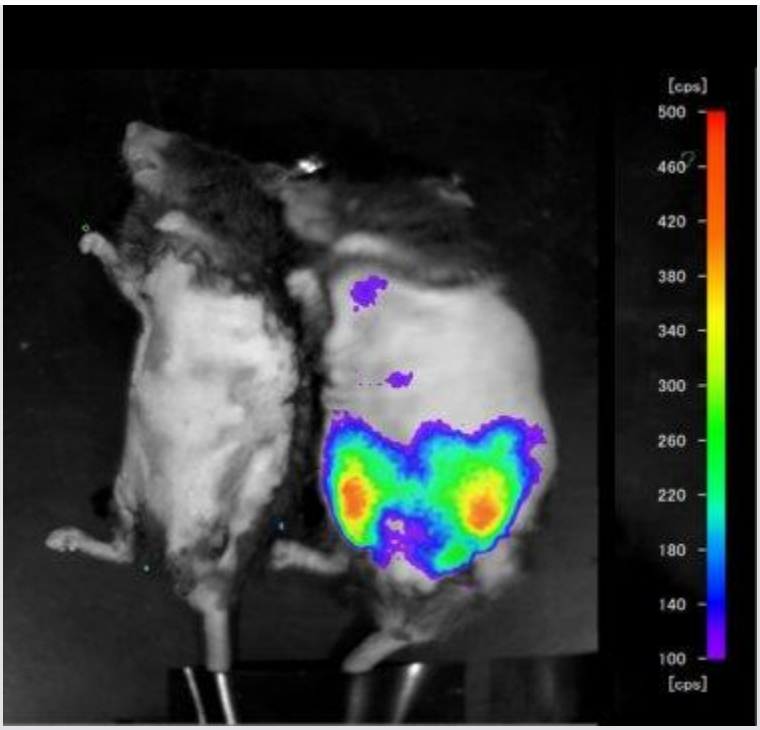
2017 年 1 月 31 日
世界的に肥満のレベルが高まる中で、2 型糖尿病、心血管疾患、癌を含む代謝障害も影響を受けており、今まで脂肪組織の炎症分析に大量の実験用マウスが使用されてきている。今回、健康食品や医薬品の抗炎症特性を評価する方法として、実験用マウスを犠牲にしない方法が、広島大学生活圏科学研究科のチームによって開発された。
研究チームは、ホタルから取り出した発行ルシフェラーゼ遺伝子を用いて、炎症を起こしている脂肪組織に光を発するキメラ遺伝子を作成。このキメラ遺伝子をマウスの受精卵に注入することにより、トランスジェニックマウスのパッチを作成し、その結果炎症検出に当たってマウスを殺さなくても済む方法が誕生した、としている。

[英文記事]：
https://www.sciencedaily.com/releases/2017/01/170131075324.htm
以下抜粋

Scientists aim to reduce animals killed in drug testing

Date:
January 31, 2017
Source:
Hiroshima University
Summary:
A non-invasive way to assess the anti-inflammatory properties of fortified health foods and medications has now been developed by researchers. The team believes their technique for examining fatty tissues will greatly reduce the numbers of lab mice sacrificed and could revolutionize medicinal drug testing.

FULL STORY



Mouse exhibiting presence of inflamed fatty tissue using bioluminance imaging.

Credit: Image courtesy of Hiroshima University

Associate Professor Noriyuki Yanaka and researchers at Hiroshima University have developed a non-invasive way to assess the anti-inflammatory properties of fortified health foods and medications.

The team from HU's Graduate School of Biosphere Science believe their technique for examining fatty tissues will greatly reduce the numbers of lab mice sacrificed and could revolutionize medicinal drug testing.

With obesity levels soaring globally, so too are associated metabolic disorders including type-2-diabetes, cardiovascular disease, and cancers. Over-nutrition strains the body and can physically damage the bodies naturally occurring fatty tissue. The body responds to this with an influx of macrophage white-blood cells -- disease-fighting cells that physically engulf pathogens.

While in the healthy body this should prove a positive action, previous animal studies show that macrophages can cause serious problems in obese fatty tissues. Macrophages damage already strained fat cells further, leading to inflammation and the risk of life-threatening diseases developing.

It is this phenomenon that the team at Hiroshima University are interested in observing -- however, their concern is not just for humans leading sedentary modern lives.

Until now, analysis of inflammation in fatty tissue required large numbers of lab mice to be terminated regularly.

Not only does this regular termination restrict long-term observation, it also leads to many mice dying in vain, as unsatisfactory observations require fresh batches of mice to be prepared.

Professor Yanaka, conscious of the high numbers of lab mice euthanized in these studies has proposed a method that reduces not only the numbers of rodents sacrificed but also makes for more-satisfactory observations -- a solution that works better for all concerned.

By investigating a spectrum of lab mouse genes, and their activation in different murine tissues, the researchers at HU have been able to isolate one that is highly expressed in obese fatty tissues where macrophages are causing inflammation havoc.

That gene, Serum Amyloid A3 (*Saa3*), "turns-on" and can be detected when fatty tissue becomes inflamed, enabling a non-invasive drug testing method to be developed.

To achieve this Professor Yanaka's team attached the light-emitting luciferase gene, sourced from fireflies, to Saa3's promoter region -- creating a chimeric gene that emits light in fatty tissue suffering inflammation.

By injecting this chimeric gene into fertilized mouse eggs the team created a batch of transgenic mice -- some of which were fed a natural-wild diet, and others a high-fat diet. Examination of them, while alive, using bioluminescence imaging showed the obese mice exhibiting high concentrations of light-emittance in areas where inflamed fatty tissue was found to be present -- the natural-wild-diet fed mice exhibited no light emittance, thus a non-invasive method for detecting inflammation in fatty tissue was born.

Professor Yanaka stresses this method does not negate the need for terminating lab mice entirely as some require termination for further analysis. However, the process is much more selective and termination only occurs when it is most useful to the researchers. He says the numbers of lab mice required will drop dramatically and this could have far-reaching ramifications:

"Animal experimentation is a serious social problem in developed countries, with many animals being sacrificed in order to develop medicines, cosmetics and functional foods for humans.

"What we are proposing is a way to reduce not only the number of animals which are used in experiments, but also animal experiments themselves."

Story Source:

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

Journal Reference:

1. Yohei Sanada, Takafumi Yamamoto, Rika Satake, Akiko Yamashita, Sumire Kanai, Norihisa Kato, Fons AJ van de Loo, Fusanori Nishimura, Philipp E. Scherer, Noriyuki Yanaka. **Serum Amyloid A3 Gene Expression in Adipocytes is an Indicator of the Interaction with Macrophages**. *Scientific Reports*, 2016; 6: 38697 DOI: 10.1038/srep38697

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2. 脳内の「やる気スイッチ」を発見、慶應義塾大ほか共同研究グループ – マウス実験

2017 年 2 月 2 日

慶應義塾大学と生理学研究所は 2 月 2 日、「脳内にあるやる気スイッチを発見した」と発表した。共同研究グループが、マウスを用いた実験で意欲障害の原因となる脳内の部位を特定した。意欲障害の治療法につながる成果だという。

研究成果を発表したのは、慶應義塾大学医学部精神・神経科学教室の田中謙二准教授、三村將教授、生理学教室の岡野栄之教授、北海道大学大学院医学研究科の渡辺雅彦教授、防衛医科大学校の太田宏之助教、大学共同利用機関法人自然科学研究機構 生理学研究所の佐野裕美助教らの共同研究グループ。

意欲障害は、いわゆる「やる気がない」という症状で、認知症や脳血管障害、脳外傷など脳の障害では、高い頻度で認められる。だが、原因やメカニズムは、脳が広範囲に障害を受けたときに起こること以外わかっていなかった。

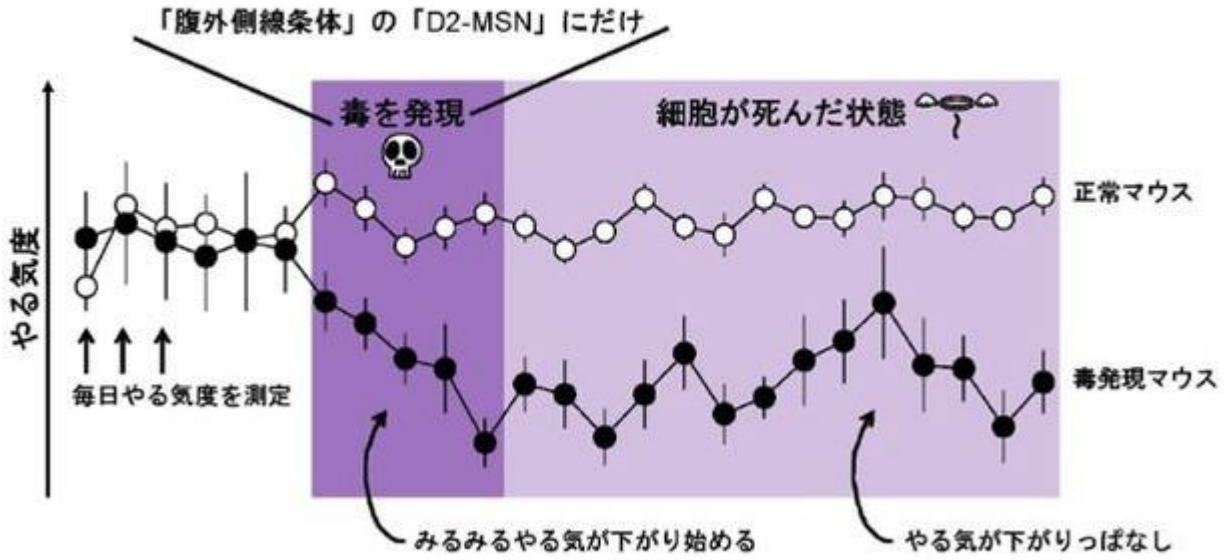
共同研究グループでは、運動制御や報酬を計算する脳部位である大脳基底核・線条体を構成する細胞集団、ドパミン受容体 2 型陽性中型有棘ニューロン（D2-MSN）に注目。D2-MSN だけに神経毒を発現させて細胞死させたマウスと正常なマウスを比較して、意欲評価の実験を行った。

その結果、線条体腹外側という脳領域の限られた細胞集団が障害を受けるだけで意欲が障害されること、この細胞集団が健康でないと意欲を維持できないことを発見した。やる気を生むためには、ほかにもいくつかの部位が必要であると想像されているが、今回の研究では初めて、やる気を維持する脳部位・細胞種を明確に示せたという。

今後は、意欲障害モデル動物を用いて、これまで治療法がまったくわかっていなかった脳損傷後の意欲障害における治療法や改善する薬剤を探索することが可能になるという。

研究成果は、2 月 1 日に総合科学雑誌「Nature Communications」に掲載された。

（図 1）


「腹外側線条体」の「D2-MSN」にだけ
毒を発現
細胞が死んだ状態
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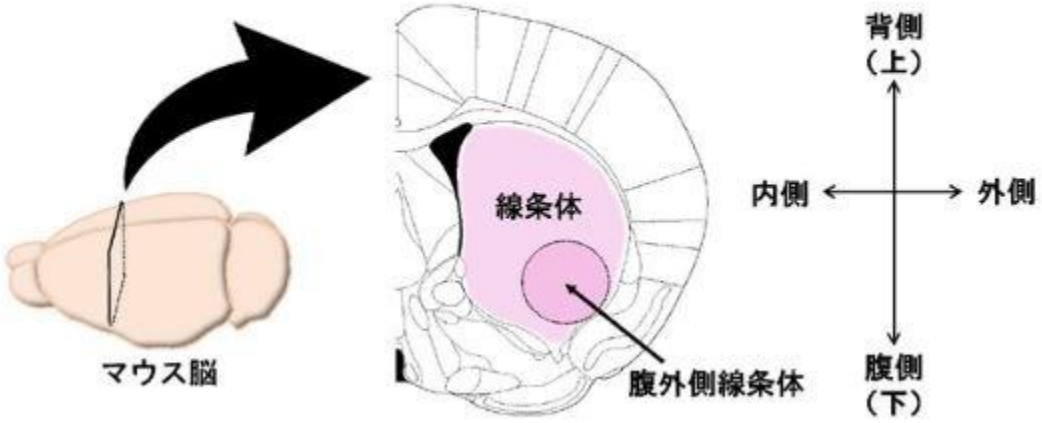
（図 3）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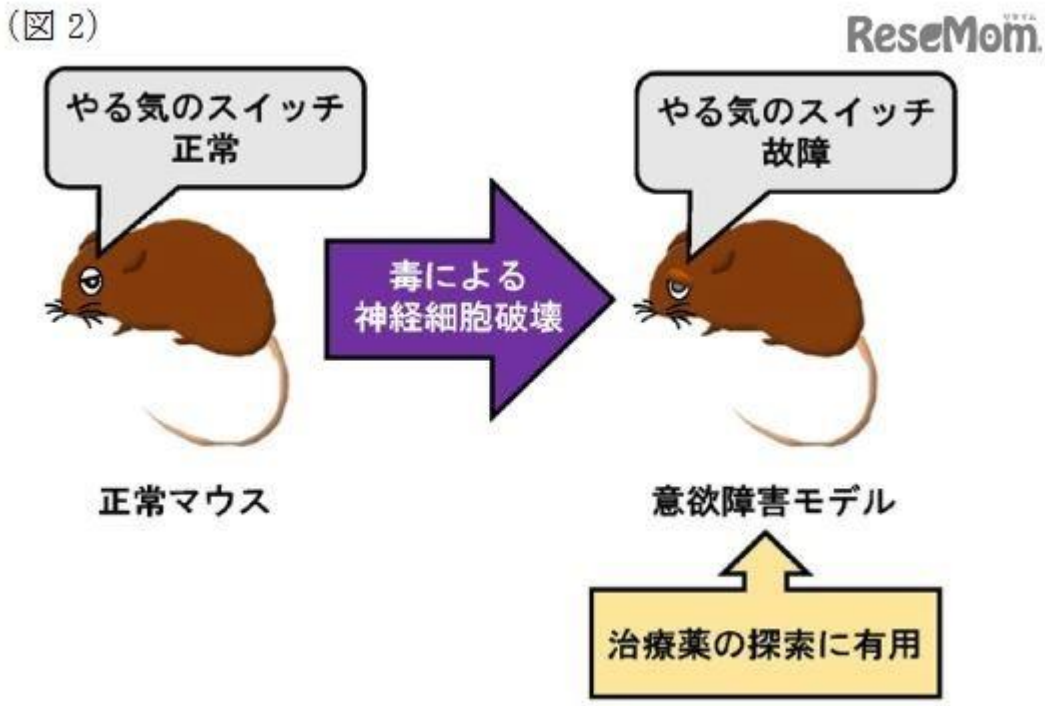

ReseMom より転載
http://resemom.jp/article/2017/02/02/36305.html
https://www.keio.ac.jp/ja/press-releases/files/2017/2/2/170202-1.pdf

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3．ヒト化マウスモデル市場は、2021 年には 1 億 1,600 万ドルに到達予想

2017 年 2 月 7 日

Medical Health & Life Science Research News

Humanized mouse model market is expected to reach USD 116.0 million by 2021 according to new research report

WhaTech Channel: Medical Market Research
Published: 07 February 2017
Submitted by Rohan Salgarkar WhaTech Premium
News from MarketsandMarkets - Business Research

Humanized Mouse Model Market report categories the global market by Type (Genetic, Cell-based (CD34, PBMC, BLT)), Application (Hematopoiesis, Neuroscience, Oncology, Immunology & Infectious diseases), End User(Pharmaceutical & Biotech Companies, CRO)) & Geography

The report **"Humanized Mouse Model Market by Type (Genetic, Cell-based (CD34, PBMC, BLT)), Application (Neuroscience, Hematopoiesis, Oncology, Immunology & Infectious diseases) & End User(Pharmaceutical & Biotech Companies, CRO) - Global Forecast to 2021"**, analyzes and studies the major market drivers, restraints/challenges, and opportunities.

***Browse 69 market data tables and 47 figures spread through 182 pages and in-depth TOC on* "Humanized Mouse Model Market by Type (Genetic, Cell-based (CD34, PBMC, BLT)), Application (Neuroscience, Hematopoiesis, Oncology, Immunology & Infectious diseases) & End User(Pharmaceutical & Biotech Companies, CRO) - Global Forecast to 2021"**

This report studies the global humanized mouse modelmarket for the forecast period of 2016 to 2021. This market is expected to reach USD 116.0 Million by 2021 from USD 73.3 Million in 2016, growing at a CAGR of 9.6%.

The global humanized mouse model market is segmented on the basis of type, application, end user, and region.

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On the basis of type, the humanized mouse model market is categorized into genetic and cell-based humanized mouse model.

The genetic models segment is estimated to account for the largest share of the global humanized mouse model market, by type.

The cell-based models segment is projected to grow at the highest CAGR between 2016 and 2021, owing to the growing applications of cell-based humanized mouse models. The cell-based mouse model segment is further segmented into CD34, PBMC, and BLT humanized mouse.

In 2016, the CD34 model segment is expected to command the largest share of the global cell-based humanized mouse model market and grow at the highest CAGR during the forecast period. CD34 mouse models are used as in vivo platforms for analyzing the safety and effectiveness of potential new drugs that can modulate the immune system.

Additionally, they are used for long-term studies in the fields of immuno-oncology, infectious disease, and graft versus host disease. Thus, the growing application areas of CD34 models are expected to trigger the demand for these models in the coming years.

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On the basis of application, the humanized mouse model market is segmented into oncology, immunology & infectious diseases, neuroscience, toxicology, hematopoiesis, and other applications (which include rare diseases, graft-versus-host diseases, cardiovascular diseases, and regenerative medicine). In 2016, the oncology segment is expected to command the largest share of the market.

On the basis of end user, the humanized mouse model market is segmented into pharmaceutical & biotechnology companies, contract research organizations (CROs), and academic & research institutions. In 2016, the pharmaceutical & biotechnology companies segment is expected to command the largest share of the market.

Key players in the global humanized mouse model market include The Jackson Laboratory (U.S.), Taconic Biosciences, Inc. (U.S.), Harbour Antibodies BV (China), HuMurine Technologies, Inc. (U.S.), Vitalstar Biotechnology Co. Ltd. (China), Crown Bioscience, Inc. (U.S.), ingenious targeting laboratory (U.S.), Axenis S.A.S (France), TRANS GENIC, Inc. (Japan), genOway S.A. (France), and Horizon Discovery Group plc (U.K.).

REPORT SUMMARY:

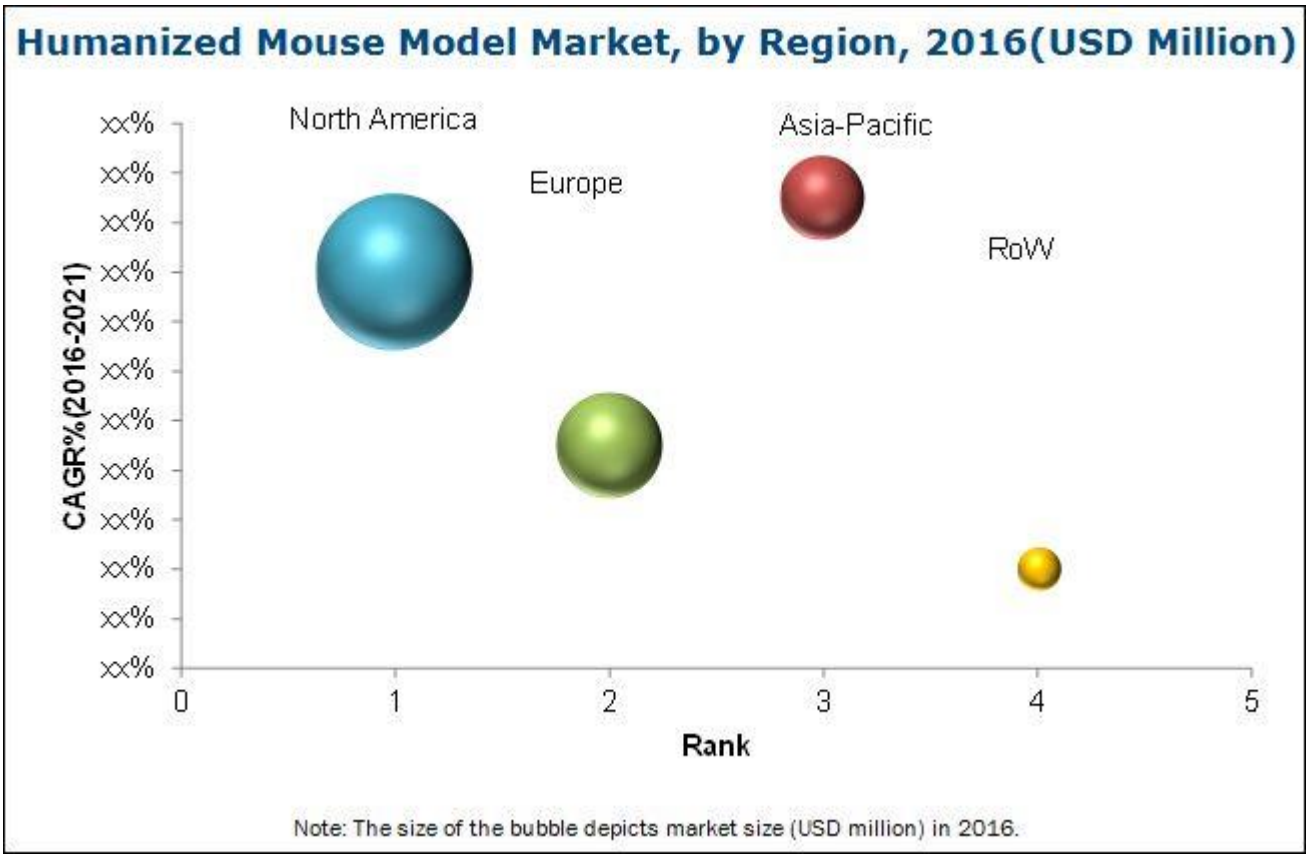
The humanized mouse model market is projected to reach USD 116.0 Million by 2021 from 73.3 Million in 2016, growing at a CAGR of 9.6% during the forecast period. The growth of the overall market can be contributed to surge in the number of research activities involving humanized mouse models, increased R&D activities by pharmaceutical and biotechnology companies, and growing adoption of personalized medicine. In addition to this, continuous support for research activities in the form of investments and grants is further stimulating the market growth.

The global humanized mouse model market is categorized on the basis of type, application, end user, and region. On the basis of type, the market is categorized into genetic and cell-based humanized mouse model. The genetic segment is estimated to account the largest share of the global market, by type. The cell-based humanized mouse model segment is further divided into CD34, PBMC, and BLT humanized mouse model. In 2016, the CD34 segment is expected to command the largest share of the cell-based humanized mouse model market. CD34 mice models are used as in vivo platforms for analyzing the safety and effectiveness of potential new drugs that can modulate the immune system. Additionally, they are used for long-term studies in the fields of immuno-oncology, infectious disease, and graft versus host disease. Thus, the growing application areas of CD34 models are expected to trigger the demand for these models in the coming years.

On the basis of application, the global humanized mouse model market is segmented into oncology, immunology and inflammation, neuroscience, hematopoiesis, toxicology, and other applications. The oncology segment is estimated to command the largest share of the global market in 2016. This segment is projected to grow at the highest CAGR during the forecast period. The growth of this segment can be attributed to increasing research activities and growing funding from various governments to carry out research studies on cancer.

On the basis of end user, the market is segmented into pharmaceutical & biotechnology companies, contract research organizations (CROs), and academic & research institutions. In 2016, the pharmaceutical & biotechnology companies segment is expected to command the largest share of the market.

North America is expected to account for the largest share of the global humanized mouse model market. The large share of this segment can be attributed to rising biomedical research and increased R&D spending by pharmaceutical companies. In the coming years, the humanized mouse model market is expected to witness the highest growth rate in the Asia-Pacific region. The high growth in the region can be attributed to rising focus on personalized medicine in China, increasing investments from government and private sector in China’s life sciences sector, research in regenerative medicine in Japan, increase in animal research in Malaysia, growth in the pharmaceutical industry in India, growth in translational and biomedical research in Singapore, and rising pharmaceutical and biotechnology R&D activities in South Korea.



Key players in the global humanized mouse model market include The Jackson Laboratory (U.S.), Taconic Biosciences, Inc. (U.S.), Harbour Antibodies BV (China), HuMurine Technologies, Inc. (U.S.), Vitalstar Biotechnology Co. Ltd. (China), Crown Bioscience, Inc. (U.S.), ingenious targeting laboratory (U.S.), Axenis S.A.S (France), TRANS GENIC, Inc. (Japan), genOway S.A. (France), and Horizon Discovery Group plc (U.K.).

To speak to our analyst for a discussion on the above findings, click **Speak to Analyst**

https://www.whatech.com/market-research/medical/258939-humanized-mouse-model-market-is-expected-to-reach-usd-116-0-million-by-2021-according-to-new-research-report

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４．マウス研究によると、重度の新生児黄疸は予防できる

2017 年 2 月 6 日
新生児は、分子ビリルビンを分解する酵素はすぐには活性化せず、結果としてビリルビンの蓄積が黄疸を引き起こす。今回、カリフォルニア大学サンディエゴ校保健科学部の研究者らは、ビリルビン分解酵素を阻害するタンパク質を同定した。この阻害剤をブロックし、酵素の活性を回復させることができれば、重度の黄疸を予防/治療する新たなアプローチを提供できる、としている。
ヒトの新生児における UGT1A1 と呼ばれるこの分解酵素の役割の理解を深めるために、マウスの天然 UGT1A1 遺伝子をヒト遺伝子と置き換えたところ、正常なマウスは誕生時に黄疸を発症しないが、「ヒト化」マウスは重篤な新生児高ビリルビン血症を引き起こした。また、マウスの腸組織から NCoR1 遺伝子を欠失させた時、UGT 1A1 遺伝子が活性化され、過剰なビリルビンを分解し高ビリルビン血症の徴候を排除した、としている。
この研究は、2 月 6 日の米国科学アカデミー紀要に公開されている。

[英文記事]:
https://www.sciencedaily.com/releases/2017/02/170206155945.htm
以下抜粋

Severe newborn jaundice could be preventable, mouse study shows

Date:
February 6, 2017
Source:
University of California San Diego Health Sciences
Summary:
A protein that inhibits the enzyme that breaks down bilirubin in newborns has now been identified by researchers. Methods that block this inhibitor, and thus restore the enzyme's activity, could provide a new therapeutic approach for preventing or treating severe newborn jaundice.

FULL STORY

For many newborn babies, an enzyme that breaks down the molecule bilirubin doesn't activate right away. The resulting bilirubin buildup can lead to jaundice, a

typically harmless condition that causes a baby's skin to temporarily appear yellow. In some cases, however, bilirubin can accumulate to toxic levels in the brain. Researchers at University of California San Diego School of Medicine have identified a protein that inhibits the bilirubin-breakdown enzyme. Methods that block this inhibitor, and thus restore the enzyme's activity, could provide a new therapeutic approach for preventing or treating severe jaundice.

The study is published February 6 by the Proceedings of the National Academy of Sciences. "This is the first report that describes the molecular processes that dictate the onset and control of the most medically worrisome form of jaundice in newborns, a condition known as severe neonatal hyperbilirubinemia," said study co-author Robert Tukey, PhD, professor of pharmacology at UC San Diego School of Medicine. "This new information will help us look for drugs or dietary therapeutics that alleviate the early onset of bilirubin toxicity."

At birth, newborns are suddenly exposed to unprecedented levels of oxygen, resulting in the rapid but temporary destruction of red blood cells and spillage of excess bilirubin in the bloodstream. If not properly broken down by an enzyme called UDP-glucuronosyltransferase 1A1 (UGT1A1), bilirubin continues to accumulate. High bilirubin levels in the brain can lead to encephalopathy, seizures, life-long brain damage and even death.

To better understand UGT1A1's role in human newborns, Tukey's collaborator and senior author Shujuan Chen, PhD, assistant professor of pharmacology at UC San Diego School of Medicine, replaced the native UGT1A1 gene in mice with the human version of the gene. While normal mice don't develop jaundice at birth, the researchers found that "humanized" mice developed severe neonatal hyperbilirubinemia and some of the resulting health consequences.

Tukey, Chen and team also discovered that the UGT1A1 gene is turned off in liver tissue in newborn humanized mice, as in humans, but also repressed in the gastrointestinal tract. They eventually identified the cause of UGT1A1's inhibition in humanized newborn mice -- a repressor protein called nuclear corepressor protein 1 (NCoR1).

When the researchers deleted the NCoR1 gene from the mice's intestinal tissue, the UGT1A1 gene was activated. Newly restored UGT1A1 broke down the excess bilirubin, eliminating signs of severe neonatal hyperbilirubinemia in the humanized mice.

"Since we now know that intestinal tissue is at least partly responsible for regulating bilirubin toxicity, we're hopeful that oral therapeutics could be developed to block the onset of severe neonatal hyperbilirubinemia," said Chen.

In countries with adequate health care systems, severe neonatal hyperbilirubinemia can be managed with phototherapy and blood transfusions. However, in many parts of the world, such as sub-Saharan Africa, South Asia and other places where preterm births are on the rise, rapid bilirubin rise often goes untreated. Each year, more than 1 million newborns worldwide experience severe neonatal hyperbilirubinemia.

Story Source:

Materials provided by University of California San Diego Health Sciences. Original written by Heather Buschman, PhD. Note: Content may be edited for style and length.

Journal Reference:
Shujuan Chen et al. Intestinal NCoR1, a regulator of epithelial cell maturation, controls neonatal hyperbilirubinemia. PNAS, February 2017 DOI: 10.1073/pnas.1700232114

目次に戻る

5. 腸内細菌がアルツハイマー病を加速 - マウス実験

2017 年 2 月 10 日

健康なマウスと罹患したマウスの両方を研究することにより、アルツハイマー病に罹患したマウスは健康なマウスに比べて腸内細菌の組成が異なることが、スウェーデンのルンド大学の新しい研究で示された。

研究者らは、腸内細菌とその病気との関係をさらに研究するために、腸内細菌を全く欠いたマウスでアルツハイマー病を研究。細菌を完全に欠損したマウスでは、アルツハイマー病の場合に神経線維に形成される塊である脳内ベータアミノロイド斑の量が非常に少なかった。また、罹患したマウスの腸内細菌叢を無菌マウスに移した場合、脳内ベータアミノロイド斑が発生したことを発見した。

腸内細菌がアルツハイマー病の発症を加速すると示したこの論文は、*Scientific Reports* 誌 2 月 8 日オンライン版に掲載されている。

[英文記事]:
https://www.sciencedaily.com/releases/2017/02/170210085532.htm
以下抜粋

Gut bacteria may play a role in Alzheimer's disease

Date:
February 10, 2017
Source:
Lund University
Summary:
New research has shown that intestinal bacteria can accelerate the development of Alzheimer's disease. According to the researchers behind the study, the results open up the door to new opportunities for preventing and treating the disease.

FULL STORY

New research from Lund University in Sweden has shown that intestinal bacteria can accelerate the development of Alzheimer's disease. According to the researchers behind the study, the results open up the door to new opportunities for preventing and treating the disease.

Because our gut bacteria have a major impact on how we feel through the interaction between the immune system, the intestinal mucosa and our diet, the composition of the gut microbiota is of great interest to research on diseases such as Alzheimer's. Exactly how our gut microbiota composition is composed depends on which bacteria we receive at birth, our genes and our diet.

By studying both healthy and diseased mice, the researchers found that mice suffering from Alzheimer's have a different composition of gut bacteria compared to mice that are healthy. The researchers also studied Alzheimer's disease in mice that completely lacked bacteria to further test the relationship between intestinal bacteria and the disease. Mice without bacteria had a significantly smaller amount of beta-amyloid plaque in the brain. Beta-amyloid plaques are the lumps that form at the nerve fibres in cases of Alzheimer's disease.

To clarify the link between intestinal flora and the occurrence of the disease, the researchers transferred intestinal bacteria from diseased mice to germ-free mice, and discovered that the mice developed more beta-amyloid plaques in the brain compared to if they had received bacteria from healthy mice.

"Our study is unique as it shows a direct causal link between gut bacteria and Alzheimer's disease. It was striking that the mice which completely lacked bacteria developed much less plaque in the brain," says researcher Frida Fåk Hållenius, at the Food for Health Science Centre.

"The results mean that we can now begin researching ways to prevent the disease and delay the onset. We consider this to be a major breakthrough as we used to only be able to give symptom-relieving antiretroviral drugs."

The research is a result of an international collaboration between Associate Professor Frida Fåk Hållenius and doctoral student Nittaya Marungruang, both at the Food for Health Science Centre in Lund, and a research group at the Ecole Polytechnique Federale de Lausanne in Switzerland. The collaboration has now expanded to include researchers from Germany and Belgium in connection with receiving a SEK 50 million EU grant.

The researchers will continue to study the role of bacteria in the development of Alzheimer's disease, and test entirely new types of preventive and therapeutic strategies based on the modulation of the gut microbiota through diet and new types of probiotics.

Story Source:

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

Journal Reference:

1. T. Harach, N. Marungruang, N. Duthilleul, V. Cheatham, K. D. Mc Coy, G. Frisoni, J. J. Neher, F. Fåk, M. Jucker, T. Lasser, T. Bolmont. **Reduction of Abeta amyloid pathology in APPPS1 transgenic mice in the absence of gut microbiota**. *Scientific Reports*, 2017; 7: 41802 DOI: 10.1038/srep41802

Cite This Page:

- MLA
- APA
- Chicago

Lund University. "Gut bacteria may play a role in Alzheimer’s disease." ScienceDaily. ScienceDaily, 10 February 2017. <www.sciencedaily.com/releases/2017/02/170210085532.htm>.

目次に戻る

6. 幹細胞を再プログラミング化して膵細胞に – 糖尿病治療に光 – マウス実験

2017 年 2 月 13 日
ヘルムホルツ協会の Max Delbrück 分子医学研究センター (MDC) の研究チームは、糖尿病患者がインスリン注射ではなく細胞療法によって治療できる方法を、マウス実験によって示すことに成功した。
その方法は、TGIF2 と呼ばれる単一の遺伝子の活性を変化させることによるもので、膵臓の組織において活性であるこの遺伝子の追加コピーをマウスの肝臓から取り出した細胞に与えた場合、細胞はまず肝臓の性質を失い、次に膵臓の特性を獲得した。更にその改変された細胞を糖尿病マウスに移植したところ、マウスの血糖値がすぐに改善されたとしている。
この研究成果は、*Nature Communications* の 2 月 13 日オンライン版で公開されている。

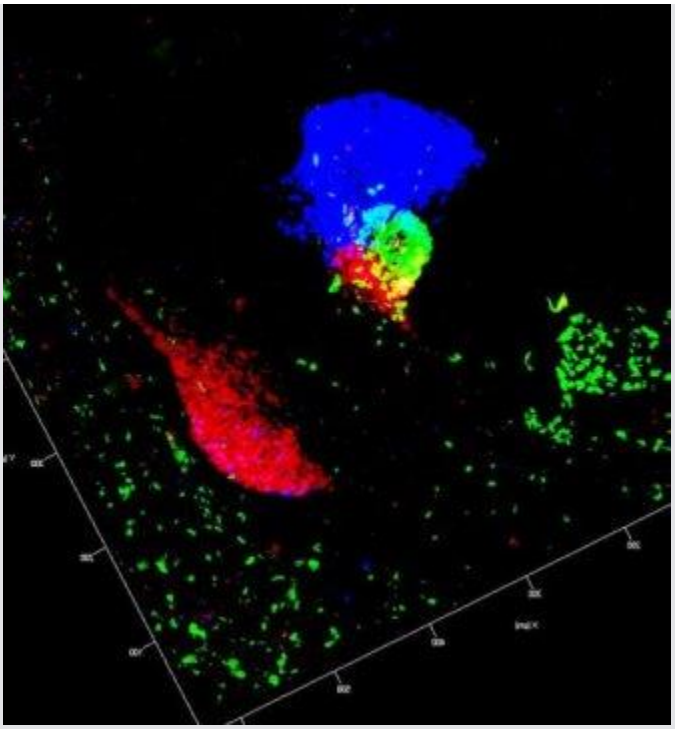
[英文記事]:
https://www.sciencedaily.com/releases/2017/02/170213083736.htm
以下抜粋

Inducing an identity crisis in liver cells may help diabetics

First successful reprogramming of liver cells to pancreas progenitor cells based on a single factor

Date:
February 13, 2017
Source:
Max Delbrück Center for Molecular Medicine in the Helmholtz Association
Summary:
It is now possible to reprogram cells from the liver into the precursor cells that give rise to the pancreas by altering the activity of a single gene. A team of researchers has now accomplished this feat in mice. Their results should make it feasible to help diabetic patients through cell therapy.

Share:
FULL STORY



This is a 3-D map of liver and pancreatic buds in a mouse embryo. Cells of the pancreas are marked in red and green, while liver cells appear in blue.

Credit: Francesca Spagnoli, MDC

It is now possible to reprogram cells from the liver into the precursor cells that give rise to the pancreas by altering the activity of a single gene. A team of researchers at the Max Delbrück Center for Molecular Medicine in the Helmholtz Association (MDC) has now accomplished this feat in mice. Their results should make it feasible to help diabetic patients through cell therapy.

In patients suffering from type I diabetes, their immune system turns against their own bodies and destroys a type of pancreatic cell called islet cells. Without these cells, the pancreas is unable to produce the hormone insulin and blood glucose rises, which leads to diabetic disease. At that point, such patients need to inject insulin for the rest of their lives.

A way to provide a lasting help to the afflicted may be to grow new pancreatic cells outside of the body. MDC group leader and researcher Dr. Francesca has been pursuing the idea of reprogramming liver cells to become pancreatic cells. Dr. Spagnoli's team has now succeeded in thrusting liver cells into an "identity crisis" -- in other words, to reprogram them to take on a less specialized state -- and then stimulate their development into cells with pancreatic properties.

Promising success in animal experiments

A gene called TGIF2 plays a crucial role in the process. TGIF2 is active in the tissue of the pancreas but not in the liver. For the current study Dr. Nuria Cerda Esteban, at the time a PhD student in Dr. Spagnoli's lab, tested how cells from mouse liver behave when they are given additional copies of the TGIF2 gene.

In the experiment, cells first lost their hepatic (liver) properties, then acquired properties of the pancreas. The researchers transplanted the modified cells into diabetic mice. Soon after this intervention, the animals' blood glucose levels improved, indicating that the cells indeed were replacing the functions of the lost islet cells. The results bring cell therapies for human diabetic patients one step closer to reality.

The obvious next step is to translate the findings from the mouse to humans. The Spagnoli lab is currently testing the strategy on human liver cells in a project funded in 2015 by the European Research Council. "There are differences between mice and humans, which we still have to overcome," Spagnoli says. "But we are well on the path to developing a 'proof of concept' for future therapies."

Story Source:

Materials provided by **Max Delbrück Center for Molecular Medicine in the Helmholtz Association**.
Note: Content may be edited for style and length.

Journal Reference:

1. Nuria Cerdá-Esteban, Heike Naumann, Silvia Ruzittu, Nancy Mah, Igor M. Pongrac, Corinna Cozzitorto, Angela Hommel, Miguel A. Andrade-Navarro, Ezio Bonifacio, Francesca M. Spagnoli. **Stepwise reprogramming of liver cells to a pancreas progenitor state by the transcriptional regulator Tgif2**. *Nature Communications*, 2017; 8: 14127 DOI: 10.1038/ncomms14127

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to pancreas progenitor cells based on a single factor. *ScienceDaily*. Retrieved February 13, 2017 from www.sciencedaily.com/releases/2017/02/170213083736.htm
Max Delbrück Center for Molecular Medicine in the Helmholtz Association. "Inducing an identity crisis in liver cells may help diabetics: First successful reprogramming of liver cells to pancreas progenitor cells based on a single factor." ScienceDaily. www.sciencedaily.com/releases/2017/02/170213083736.htm (accessed February 13, 2017).

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7. 併用免疫療法でマウスの脳腫瘍を死滅

2017 年 2 月 15 日
SMAC Mimetics (SMs) として知られる薬剤と免疫チェックポイント阻害剤 (ICIs) の組み合わせは、マウス実験で癌腫瘍細胞の死滅率を増加させた。
カナダ オタワのオンタリオ州子供病院 (Children's Hospital of Eastern Ontario: CHEO) 研究センターの研究者らは、2014 年、SMs を免疫刺激剤またはウィルス療法と組み合わせることによって、どちらの薬剤よりも優れた腫瘍殺滅効果が得られることを発見した。今回は、更に SMs は ICIs と強力な相乗効果を発揮することを発表した。
この研究の科学的根拠は、*Nature Communications* に掲載されている

[英文記事]:
https://www.sciencedaily.com/releases/2017/02/170215101427.htm
以下抜粋

Researchers kill brain cancer in mice with combination immunotherapies

Date:
February 15, 2017
Source:
Children's Hospital of Eastern Ontario Research Institute

Summary:
A combination of drugs known as SMAC Mimetics and immune checkpoint inhibitors (ICIs) amplifies kill rates of cancer tumor cells in laboratory testing.

Share:
FULL STORY

A promising combination of immunotherapies delivers a one-two punch to brain cancer tumours with high cure rates in mice.

Credit: © BillionPhotos.com / Fotolia

A promising combination of immunotherapies delivers a one-two punch to brain cancer tumours with high cure rates in mice, scientific evidence published in *Nature Communications* says.

Researchers at the Children's Hospital of Eastern Ontario (CHEO) in Ottawa found that a combination of drugs known as SMAC Mimetics and immune checkpoint inhibitors (ICIs) amplifies kill rates of cancer tumour cells in laboratory testing. Researchers also discovered a new mechanism by which the combination promotes long-term immunity against glioblastoma tumours. The combination therapy also proved to be highly effective against breast cancer and multiple myeloma.

"These findings represent a significant evolution in our research and the field of immunotherapy. We are the first in the world to show the synergistic tumour-killing impact of combining SMAC Mimetics with

immune checkpoint inhibitors for glioblastoma," said Dr. Robert Korneluk, distinguished professor at the University of Ottawa and senior scientist at the CHEO Research Institute. "You could say it takes two to tango. We believe that it takes a combination strategy to impact cancer cure rates."

In 2014, a team of scientists led by Dr. Korneluk discovered that combining SMAC Mimetics with immune stimulators or live virus therapies had a synergistic or amplified tumour-killing effect that was greater than either agent on its own. Today's news shows that SMAC Mimetics also have a powerful synergistic effect with ICIs, relatively new drugs that are showing great promise in the clinic. SMAC Mimetics known as LCL161 and Birinapant were combined with ICI antibodies targeting PD-1 and CTLA-4 immune checkpoints.

Eric Lacasse, a scientist at the CHEO Research Institute, said: "Two drug companies have initiated human clinical trials this year to assess the impact of this combination of SMAC Mimetics and ICIs on patients with a variety of cancers. Although it could be years before any clinical trials begin for adults or children with the deadly brain cancer, glioblastoma, we're looking forward to seeing how scientific evidence from these experimental treatments adds to our knowledge. It's an exciting, exploratory field and we hope we've hit a home run."

Shawn Beug, lead author of the 2014 and 2017 papers, said: "This research heightens our understanding of the mechanics behind this double-whammy effect, which both enhances the immune response and weakens tumour cells to immune attack. We're hoping that more oncologists and biotech companies test out this combination in clinical trials as we continue to decipher how SMAC Mimetics encourage the immune system to kill cancer cells."

Story Source:

Materials provided by **Children's Hospital of Eastern Ontario Research Institute**. *Note: Content may be edited for style and length.*

Journal Reference:

1. Shawn T. Beug, Caroline E. Beauregard, Cristin Healy, Tarun Sanda, Martine St-Jean, Janelle Chabot, Danielle E. Walker, Aditya Mohan, Nathalie Earl, Xueqing Lun, Donna L. Senger, Stephen M. Robbins, Peter Staeheli, Peter A. Forsyth, Tommy Alain, Eric C. LaCasse, Robert G. Korneluk. **Smac mimetics synergize with immune checkpoint inhibitors to promote tumour immunity against glioblastoma**. *Nature Communications*, 2017; 8 DOI: 10.1038/ncomms14278

- Chicago

Children's Hospital of Eastern Ontario Research Institute. "Researchers kill brain cancer in mice with combination immunotherapies." ScienceDaily. ScienceDaily, 15 February 2017. <www.sciencedaily.com/releases/2017/02/170215101427.htm>.
Children's Hospital of Eastern Ontario Research Institute. (2017, February 15). Researchers kill brain cancer in mice with combination immunotherapies. *ScienceDaily*. Retrieved February 16, 2017 from www.sciencedaily.com/releases/2017/02/170215101427.htm
Children's Hospital of Eastern Ontario Research Institute. "Researchers kill brain cancer in mice with combination immunotherapies." ScienceDaily. www.sciencedaily.com/releases/2017/02/170215101427.htm (accessed February 16, 2017).

目次に戻る

8. 肥満解消を複雑にするメカニズムの研究

2017 年 2 月 15 日
新陳代謝を増加させる方法があるか？
スクリプス研究所 (TSRI) の Anutosh Chakraborty 助教授による過去の研究において、動物モデルで蓄積された脂肪の分解を遅らせ体重増加を促すたんぱく質を特定した。今回、彼とその同僚は、IP6K1 として知られるこのたんぱく質の遺伝子を欠損させることによって、肥満や糖尿病から動物モデル (ノックアウトマウス) を保護することに成功した、と発表した。また、IP6K1 が外的条件には関係なく、より多くのエネルギーを燃焼させるのを妨げる、ともしている。この論文は、*Molecular Metabolism* 誌に掲載されている。

[英文記事]:
https://www.sciencedaily.com/releases/2017/02/170215130445.htm
以下抜粋

Scientists take aim at obesity-linked protein

Date:

February 15, 2017

Source:

Scripps Research Institute

Summary:

Scientists have shown that deleting the gene for this protein, known as IP6K1, protects animal models from both obesity and diabetes.

Share:

FULL STORY

Scientists are working to understand the mechanisms that make weight loss so complicated. Exercise burns calories, of course, but scientists are also looking at how the body burns more energy to stay warm in cold temperatures.

Is there a way to get metabolism to ramp up -- even when it's not cold out?

TSRI Assistant Professor Anutosh Chakraborty is on a mission to answer this question. His past research revealed a new therapeutic target in this battle -- a protein that actually promotes fat accumulation in animal models by slowing stored energy (fat) breakdown and encouraging weight gain.

Now, in a study recently published online in the journal *Molecular Metabolism*, Chakraborty and his colleagues have shown that deleting the gene for this protein, known as IP6K1, protects animal models from both obesity and diabetes. This protective effect is seen regardless of diet, even at what's known as a thermoneutral temperature (around 86?F). This means inhibiting IP6K1 should help animals burn more energy, regardless of outside conditions.

"In genetically altered animal models that lack IP6K1, we found that deletion dramatically protects these knock-out mice from diet-induced obesity and insulin resistance regardless of the temperature in the environment," Chakraborty said. "When we inhibited the enzyme with chemical compounds, the results were similar."

Why Temperature Matters

Temperature is important in the study of obesity because an animal in lower temperatures will rapidly lose weight as it burns more energy to try to maintain core body temperature.

Because humans can maintain their body temperatures in a number of ways -- clothing, for example -- any pathway that reduces body weight at higher temperatures is a highly encouraging target in human obesity.

The new study suggests a future pharmaceutical may be able to target IP6K1 to mimic the energy burning seen at relatively lower temperatures.

"If we delete IP6K1, the animals gain less body weight because they simply expend more energy -- regardless of temperature. That's important because blocking weight gain by enhancing energy expenditure in a thermoneutral environment is harder and thus, targeting IP6K1 is expected to be successful in ameliorating obesity in humans," said Chakraborty.

"If you're developing an anti-obesity drug based on inhibiting IP6K1, our new findings shows that there are potentially very few restrictions for its use -- a subject would lose weight even on a high-fat diet, and nobody would have to sit in a refrigerator to make it work," he added.

Story Source:

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

Journal Reference:

1. Qingzhang Zhu, Sarbani Ghoshal, Richa Tyagi, Anutosh Chakraborty. **Global IP6K1 deletion enhances temperature modulated energy expenditure which reduces carbohydrate and fat induced weight gain**. *Molecular Metabolism*, 2017; 6 (1): 73 DOI: 10.1016/j.molmet.2016.11.010

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Scripps Research Institute. "Scientists take aim at obesity-linked protein." ScienceDaily. ScienceDaily, 15 February 2017. <www.sciencedaily.com/releases/2017/02/170215130445.htm>.
Scripps Research Institute. (2017, February 15). Scientists take aim at obesity-linked protein. *ScienceDaily*. Retrieved February 16, 2017 from www.sciencedaily.com/releases/2017/02/170215130445.htm
Scripps Research Institute. "Scientists take aim at obesity-linked protein." ScienceDaily. www.sciencedaily.com/releases/2017/02/170215130445.htm (accessed February 16, 2017).

目次に戻る

9. 光るマウスから新しい遺伝子治療技術

2017 年 2 月 16 日

スタンフォード大学の化学者と遺伝子治療専門家の合同研究により、改変されたたんぱく質のコードをマウスの細胞に挿入する新しい方法が生まれた。これがヒトでも成功すれば、この技術はワクチンや癌治療に有効であるだろう、としている。
米国科学アカデミー紀要に掲載されたこの研究では、蛍を光らせるたんぱく質を作り出す支持を運び、麻酔をしたマウスの細胞内にそれらの支持を届ける化合物を精製し、実際にマウスを暗闇の中で光らせることに成功した。
この成功は、遺伝子治療における大きな前進であり、この技術がヒトでも通用するなら、病気の細胞に治療用たんぱく質を挿入する新しい方法を提供できることになる、としている。

[英文記事]:
https://www.sciencedaily.com/releases/2017/02/170216144029.htm
以下抜粋

Glowing mice suggest new gene therapy technique

Date:
February 16, 2017
Source:
Stanford University
Summary:
A collaboration between chemists and gene therapy experts produced a new way of inserting the code for modified proteins into the cells of mice. If successful in humans, the technique could be useful for vaccines or cancer therapies.

FULL STORY

Colin McKinlay and Jessica Vargas are co-lead authors of research that could mark a significant step forward for gene therapy by providing a new way of inserting therapeutic proteins into diseased cells.

Credit: L.A. Cicero

Timothy Blake, a postdoctoral fellow in the Waymouth lab, was hard at work on a fantastical interdisciplinary experiment. He and his fellow researchers were refining compounds that would carry instructions for assembling the protein that makes fireflies light up and deliver them into the cells of an anesthetized mouse. If their technique worked, the mouse would glow in the dark.

Not only did the mouse glow, but it also later woke up and ran around, completely unaware of the complex series of events that had just taken place within its body. Blake said it was the most exciting day of his life.

This success, the topic of a recent paper in *Proceedings of the National Academy of Sciences*, could mark a significant step forward for gene therapy. It's hard enough getting these protein instructions, called messenger RNA (mRNA), physically into a cell. It's another hurdle altogether for the cell to actually use them to make a protein. If the technique works in people, it could provide a new way of inserting therapeutic proteins into diseased cells.

"It's almost a childlike enthusiasm we have for this," said chemistry Professor Robert Waymouth. "The code for an insect protein is put into an animal and that protein is not only synthesized in the cells but it's folded and it becomes fully functional, capable of emitting light."

Although the results are impressive, this technique is remarkably simple and fast. And unlike traditional gene therapy that permanently alters the genetic makeup of the cell, mRNA is short-lived and its effects are temporary. The transient nature of mRNA transmission opens up special opportunities, such as using these compounds for vaccination or cancer immunotherapy.

Making a protein

Gene therapy is a decades-old field of research that usually focuses on modifying DNA, the fundamental genetic code. That modified DNA then produces a modified mRNA, which directs the creation of a modified protein. The current work skips the DNA and instead just delivers the protein's instructions.

Previous work has been successful at delivering a different form of RNA -- called short interfering RNA, or siRNA -- but sending mRNA through a cell membrane is a much bigger problem. While both siRNA and mRNA have many negative charges -- so-called polyanions -- mRNA is considerably more negatively charged, and therefore more difficult to sneak through the positively charged cell membrane.

What the researchers needed was a positively charged delivery method -- a polycation -- to complex, protect and shuttle the polyanions. However, this alone would only assure that the mRNA made it through the cell membrane. Once inside, the mRNA needed to detach from the transporter compound in order to make proteins.

The researchers addressed this twofold challenge with a novel, deceptively straightforward creation, which they call charge-altering releasable transporters (CARTs).

"What distinguishes this polycation approach from the others, which often fail, is the others don't change from polycations to anything else," said chemistry Professor Paul Wender, co-author of the paper. "Whereas, the ones that we're working with will change from polycations to neutral small molecules. That mechanism is really unprecedented."

As part of their change from polycations to polyneutrals, CARTs biodegrade and are eventually excreted from the body.

The power of collaboration

This research was made possible through coordination between the chemists and experts in imaging molecules in live animals, who rarely work together directly. With this partnership, the synthesis, characterization and testing of compounds could take as little as a week.

"We are so fortunate to engage in this kind of collaborative project between chemistry and our clinical colleagues. It allowed us to see our compounds go from very basic building blocks -- all the way from chemicals we buy in a bottle -- to putting a firefly gene into a mouse," said Colin McKinlay, a graduate student in the Wender lab and co-lead author of the study.

Not only did this enhanced ability to test and re-test new molecules lead to the discovery of their charge-altering behavior, it allowed for quick optimization of their properties and applications. As different challenges arise in the future, the researchers believe they will be able to respond with the same rapid flexibility.

After showing that the CARTs could deliver a glowing jellyfish protein to cells in a lab dish, the group wanted to find out if they worked in living mice, which was made possible through the expertise of the Contag lab, run by Christopher Contag, professor of pediatrics and of microbiology and immunology. Together, the multidisciplinary team showed that the CARTs could effectively deliver mRNA that produced glowing proteins in the thigh muscle or in the spleen and liver, depending on where the injection was made.

A bright future ahead

The researchers said CARTs could move the field of gene therapy forward dramatically in several directions.

"Gene therapy has been held up as a silver bullet because the idea that you could pick any gene you want is so alluring," said Jessica Vargas, co-lead author of the study, who was a PhD student in the Wender lab during this research. "With mRNA, there are more limitations because the protein expression is transient, but that opens up other applications where you wouldn't use other types of gene therapy."

One especially appropriate application of this technology is vaccination. At present, vaccines require introducing part of a virus or an inactive virus into the body in order to elicit an immune response. CARTs could potentially cut out the middleman, directly instructing the body to produce its own antigens. Once the CART dissolves, the immunity remains without any leftover foreign material present.

The team is also working on applying their technique to another genetic messenger that would produce permanent effects, making it a complementary option to the temporary mRNA therapies. With the progress already made using mRNA and the potential of their ongoing research, they and others could be closer than ever to making individualized therapeutics using a person's own cells. "Creating a firefly protein in a mouse is amazing but, more than that, this research is part of a new era in medicine," said Wender.

Story Source:

Materials provided by **Stanford University**. Original written by Taylor Kubota. *Note: Content may be edited for style and length.*

Journal Reference:

1. Colin J. McKinlay, Jessica R. Vargas, Timothy R. Blake, Jonathan W. Hardy, Masamitsu Kanada, Christopher H. Contag, Paul A. Wender, Robert M. Waymouth. **Charge-altering releasable transporters (CARTs) for the delivery and release of mRNA in living animals**. *Proceedings of the National Academy of Sciences*, 2017; 114 (4): E448 DOI: 10.1073/pnas.1614193114

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10. ポリオ様疾患研究のための新マウスモデル

2017 年 2 月 23 日
コロラド大学 Anschutz Medical Campus の科学者らは、2014 年に 120 人の子供達を麻痺させたポリオ様の疾患を研究するための最初の動物モデルを開発した。
研究者らは、2014 年の流行時に回収されたエンテロウィルス D68 または EV-D68 として知られるウィルス株が、ヒトの症例のいくつかの側面においてよく似た麻痺性疾患を引き起こす可能性があることをこのマウスモデルで示した。
この研究は、オンライン オープンアクセス ジャーナル *PLOS Pathogens* の 2 月 23 日号に掲載されている。

[英文記事]:
https://www.sciencedaily.com/releases/2017/02/170223142133.htm
以下抜粋

Researchers develop model for studying rare polio-like illness

Date:
February 23, 2017
Source:
University of Colorado Anschutz Medical Campus
Summary:
Scientists have developed the first animal model for studying paralysis caused by virus linked to a polio-like illness that paralyzed 120 children in 2014.

FULL STORY

Scientists, led by researchers at the University of Colorado School of Medicine, have developed the first animal model for studying paralysis caused by virus linked to a polio-like illness that paralyzed 120 children in 2014.

Working with mice in the laboratory of Kenneth Tyler, MD, chairman of the Department of Neurology, Alison Hixon, an MD/PhD candidate at the CU School of Medicine, and a team of researchers were able to demonstrate that several strains of the virus, known as enterovirus D68, or EV-D68, recovered during the 2014 epidemic can cause a paralytic illness in mice that resembles several aspects of the human cases.

"This is a really important breakthrough as it gives us a model to both study therapeutics and to understand how the disease develops," Tyler said. The results are published in the Feb. 23 edition of *PLOS Pathogens*, a peer-reviewed online open-access journal.

In the fall and winter of 2014, the United States experienced an epidemic of acute flaccid myelitis (AFM) cases in children coincident with a nationwide outbreak of EV-D68 respiratory disease. EV-D68 had previously been a rare cause of illness in the United States. Up to half of the 2014 AFM patients had RNA from EV-D68 detected in their respiratory secretions. The connection between EV-D68 and AFM led to the current research studies.

AFM appears as sudden onset of limb weakness, similar to polio, and medical imaging tests often reveal damage within the nervous system, particularly in the spinal cord. The effects of AFM can be devastating, leaving victims permanently disabled. Since 2014, the Centers for Disease Control and Prevention has been actively investigating the illness and has continued to receive reports of sporadic cases of AFM. In 2016, a total of 132 people in 37 states across the country were confirmed to have AFM.

"There are currently no established treatments for EV-D68," said Hixon. "A mouse model is an important first step in screening potential drug and vaccine therapies. Our results suggest that that there may be potentially effective strategies to treat or prevent EV-D68 and that's particularly important because we saw a surge in AFM cases in 2016."

Story Source:

Materials provided by **University of Colorado Anschutz Medical Campus**. *Note: Content may be edited for style and length.*

Journal Reference:

1. Alison M. Hixon, Guixia Yu, J. Smith Leser, Shigeo Yagi, Penny Clarke, Charles Y. Chiu, Kenneth L. Tyler. **A mouse model of paralytic myelitis caused by enterovirus D68**. *PLOS Pathogens*, 2017; 13 (2): e1006199 DOI: 10.1371/journal.ppat.1006199

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