Bio News – March, 2021

In-Vivo Science International, Inc.

今月の企業関連ニュース/他

1/28　Pfizer も変異株に対応できるように必要に応じて COVID-19 ワクチンに手を加える

Pfizer の CEO・Albert Bourla 氏は、新たに出回る新型コロナウイルス変種が見つかったらワクチンの効果をその都度検証し、Moderna のように必要とあらば変種に対応できるようにワクチンに手を加えていく、また、SARS-CoV-2 の根絶は見込んでおらず、SARS-CoV-2 感染がインフルエンザのように落ち着くのを助ける役割を製薬会社は担う、と述べている。
又、Pfizer はそういう状況を見越して保管がより容易なワクチンの開発に取り組んでいる。

1/28　韓国、高齢者への AstraZeneca 製コロナワクチン接種を見直し

韓国政府は28日、一般向けの新型コロナウイルスワクチン接種を第3・四半期に開始すると発表した。ただ、65歳以上に対する効果が限定的と独紙ハンデルスブラットおよびビルトで報じられた英アストラゼネカ製のコロナワクチンを高齢者に接種することは見直す。

1/29　国内流行とは別タイプのウイルス確認　東京医科歯科大

東京医科歯科大は 29 日、いま国内で広まっているものとは異なるタイプの新型コロナウイルスが患者 3 人から見つかったと発表。英国や南アフリカなどで報告されている変異ウイルスとも異なるタイプ。感染力や重症化のしやすさなどは、国内で流行しているものと大きく変わらないと考えられるという。

1/29　海水から淡水をつくる高性能な膜　神戸大が開発

1/29　英国試験で Novavax のワクチンが COVID-19 発症 89%予防～南ア試験の効果は低い

1/29　J＆Jのコロナワクチン、有効性 66%　来週にも緊急使用申請へ

米医薬品・日用品大手J＆Jは29日、開発中の新型コロナウイルスワクチンについて、有効性が66%だったと発表した。来週にもFDAに緊急使用を申請するほか、その他の国や欧州連合（EU）でも近く申請する。

https://jp.reuters.com/article/health-coronavirus-vaccines-johnson-john-idJPKBN29Y224

1/29　ハンガリー、中国製コロナワクチンを承認 EU 加盟国初

ハンガリーは 29 日、欧州連合（EU）加盟国では初めて、中国の製薬大手・中国医薬集団（シノファーム、Sinopharm）製の新型コロナウイルスワクチンを承認。

1/30　「がん論文不正」新たに2本発覚の阪大病院、臨床研究中止

1/31　投与 1 回きりで常温保存の COVID-19 ワクチン開発にゲイツ財団が 210 万ドル提供

1 か月は常温保存可能で投与 1 回きりの新型コロナウイルス感染症（COVID-19）予防ワクチンを開発する Mass General Brigham 育ちの取り組み AAVCOVID にゲイツ財団（Bill & Melinda Gates Foundation）が最大 210 万ドルを提供。

2/1　J&J と Novavax の COVID-19 ワクチンの南アフリカ変異株への効果はどちらも低め

2/2　検体と混ぜるだけで COVID-19 感染を知らせる発光蛋白質ができた

2/2　AstraZeneca の COVID-19 ワクチンの投与量ミスは被験者に通知されていなかった

2/3　Pfizer/BioNTech の一番乗り COVID-19 ワクチンの今年の売り上げ予想 150 億ドル

2/4　インド政府の調査によって同国の 4 人に 1 人が COVID-19 を経ていると推定された

2/5　米国の数少ない黒人 CEO の一人・Ken Frazier 氏が Merck & Co を引退

2/5　仏マクロン大統領、中国製ワクチンの危険性を警告 新たな変異助長の恐れも

2/6　J&J が COVID-19 ワクチンを FDA に認可申請

2/9　Novavax の COVID-19 ワクチンの認可審査が英国、米国、欧州、カナダで開始

2/10　先月イスラエルで新型コロナウイルス感染して死んだ 97%超はワクチン非接種

2/10　Tmunity（ペンシルベニア州フィラデルフィア市）がペンシルバニア大学から固形癌治療メソセリン CAR-T を更に取得

2/11　AstraZeneca の COVID-19 ワクチンを WHO が推奨

2/11　シークエンス装置の Pacific Biosciences（カリフォルニア州メンローパーク市）にソフトバンクが 9 億ドル投資

2/12　コウモリのコロナウイルスは直接ヒトに感染しうる

新型コロナウイルス（SARS-CoV-2）やコウモリ由来の SARS-CoV-2 近縁コロナウイルスを含むコロナウイルス幾つかをマウスに移植したヒト肺組織に感染させたところどのコロナウイルスも難なく増えうることが示され、コウモリの間に広まるコロナウイルスはコウモリとヒトを橋渡しする動物なしで直接ヒトに感染しうる模様。

2/14　移植患者の抗ウイルス薬抵抗性 CMV 血症が武田薬品の maribavir で 2 倍以上解消

2/16　世界の新規感染、5 週間で半減　昨年 10 月以来の少なさ

世界保健機関（WHO）のテドロス・アダノム事務局長は 15 日の記者会見で、新型コロナウイルスの世界の新規感染の報告が 5 週連続で減り、5 週間で半減したと明らかにした。1 月 4 日からの 1 週間は新規感染の報告が 500 万件以上あったが、2 月 8 日からの 1 週間は約 260 万件まで減り、昨年 10 月以来の少なさとなった。

2/16　AstraZeneca の COVID-19 ワクチン、145 か国への供給開始

2/16　ワクチン副反応、SNSで調査へ　厚労省

厚生労働省は、新型コロナウイルスのワクチンを接種した人に対して、副反応の調査をSNSを通じて行うことを決めた。

2/17　南アフリカは変異株に弱い AstraZeneca の COVID-19 ワクチンを返却したい

Economic Times によると、買ったはいいものの新型コロナウイルス変異株 501Y.V2 に殆ど無効の AstraZeneca ワクチンを南アフリカが製造元 Serum Institute of India に返したがっている。

2/18　別の変異コロナウイルスを確認　国立感染研が報告

国立感染症研究所は 18 日、変異した新型コロナウイルスについて、従来の英国由来、南アフリカ由来、ブラジル由来の三つの変異株とは異なるタイプのウイルスを国内で確認したと明らかにした。どの国に由来するかは不明。変異の仕方から、免疫の効果が弱まる可能性があるが、感染力が強くなる性質はないという。

2/18　製薬、慈善団体グループが世界中で忘れられた病気を追いかけている Adjuvant Capital のために 3 億ドル調達

これらのグループには、Merck、Novartis、IFC、the Bill & Melinda Gates Foundation が含まれる。

2/18　アフリカのザンビアの首都ルサカ -昨夏の死者の 20%から SARS-CoV-2 検出

2/19　米平均寿命、1 年縮む コロナ流行の 20 年上半期

CDC が発表した統計によると、同期の平均寿命は 77.8 歳で、2019 年の 78.8 歳からちょうど 1 年縮み、2006 年以降で最短となった。最も大きな影響を受けたのが少数派で、非ヒスパニック系黒人の寿命は 3 年、ヒスパニック系の寿命は約 2 年それぞれ縮んだ。

2/20　新型コロナウイルス中和抗体を見つけた Immunome の株価が 76%も上昇

2/20　Pfizer が COVID-19 ワクチンの標準的な冷凍庫での保管の認可を FDA に申請

Pfizer/BioNTech の新型コロナウイルス感染症予防ワクチン BNT162b2 がより一般的な冷凍庫温度である零下 25～15℃で合計 2 週間安定なことを示したデータが米国 FDA に提出された。

2/21　コロナワクチン接種、死亡防ぐ効果 99%　イスラエル

イスラエル保健省は 20 日、ファイザー製ワクチンを 2 回接種してから 2 週間が経った場合、未接種の場合と比べて、死亡に至ることを防ぐ効果が約 99%だったと発表した。重症化や感染についても、95%を超える高い予防効果がみられたという。

2/22　健常人にあえて COVID-19 を取り付かせてワクチン効果を調べる試験を英国が許可

感染を確立しうる最小量の新型コロナウイルス（SARS-CoV-2）を健康な若い成人にあえて取り付かせる試験が英国の倫理検討で許可され、今月中に始まると同国政府が先週 17 日に発表した。

2/22　絶滅危惧種クロアシイタチのクローン誕生、30 年以上前の細胞から、保護に光

世界で初めて体の細胞からクローンがつくられた哺乳類、ヒツジのドリーのことは耳にしたことがあるが、今回は、クロアシイタチのエリザベス・アン。米国在来種の絶滅危惧種では初のクローン。今回、カリフォルニア州のサンディエゴ動物園の保体であるサンディエゴ動物園の研究者らが、死後長期間冷凍保存されていた野生の個体の細胞を使い、クローン化に成功した。

2/23　紫外線に当たると発光する齧歯類見つかる

ウサギっぽいが実はネズミの仲間の「トビウサギ」。アフリカ南部に生息している野生動物で、毛色はいたって地味。ところが、この地味な薄茶色が紫外線に照らされると、赤・オレンジ・ピンク色に一変することがわかった。最近の研究で明らかになったこのトビウサギの華麗なる変貌は、まだ哺乳類では数少ない生体発光の一例。
生体発光とは、生物が太陽光から吸収した紫外線をカラフルな輝きとして放つ現象で、これまで魚類・両生類・爬虫類・鳥類、そして極小のクマムシなどに確認されていた。生体発光する哺乳類はまだ確認された例が少なく、有袋類のオポッサム、齧歯類のアメリカモモンガ、単孔類のカモノハシは光ることがわかっていた。

2/24　UCB（本社：ベルギー ブリュッセル）の COVID-19 以外の治療薬探しにも Microsoft が協力する

2/24　前 FDA 長官 Stephen Hahn 氏がパーキンソン病薬開発会社の取締役に

FDA 長官の座を降りた Stephen Hahn 氏がフィラデルフィアのパーキンソン病薬開発会社 Blackfynn の取締役に加わる。

2/24　Pfizer、１１歳以下のワクチン臨床試験へ…安全性の検証急ぐ

2/24　毛髪作る「毛包」大量増幅法を開発 理研など、脱毛治療に望み

毛髪を作る器官「毛包」を繰り返し再生させる細胞を、能力を保ったまま体外で大量に増やす方法を開発したと、理化学研究所などの研究グループが発表。マウスの実験で効果や安全性を確認しており、臨床研究を準備済みで共同研究企業を探しているという。脱毛症治療への応用が実現すれば世界初の、複数種の細胞からなる器官丸ごとの再生医療となる。

2/25　J&J の COVID-19 ワクチンを FDA が有効で安全と判断～早ければ今週中承認

2/25　J＆J 製ワクチン、コロナ重症化予防に高い効果

FDA は 24 日、米医薬品大手 J＆J が開発した接種 1 回型の新型コロナウイルスワクチンについて、重症化予防での高い効果が確認されたと発表。

2/25　大腸を使って小腸の再生成功　動物（ラット）実験で　慶応大などのチーム

2/25　イスラエルで Pfizer の COVID-19 ワクチン二回接種が無症状感染の 9 割を恐らく予防

2/25　SoftBank が上場バイオテック企業（DNA 配列を読む Pacific Biosciences of California）に数十億ドルを投資するらしい/Bloomberg

2/26　Moderna が COVID-19 ワクチンの今年の売上を Pfizer を上回る 184 億ドルと予想

2/26　米国が COVID 後遺症を post-acute sequelae of SARS-CoV-2 infection（PASC）と命名

2/26　初めて利益が出る見通しの Moderna の最高医学責任者 Tal Zaks 氏が 9 月に辞任

2/27　運動選手の頭部の衝撃から脳を守る首巻き装置 Q-Collar を FDA が承認

2/28　J&J 製新型コロナワクチン許可　米で 3 例目、1 回接種は初

2/28　外国からの研究費、開示を義務付けへ…先端技術の流出防止・虚偽報告に罰則も -科学技術省

政府は、国から資金援助を受ける研究者に対し、外国を含めた資金提供状況の開示を義務付ける方針を固めた。公的な研究費に関する指針を年内に改定する。資金源の透明性を高め、先端技術の海外流出を防ぐ狙いがある。

企業関連ニュース/他のトップページに戻る

今月の研究関連ニュース/他

1. 栄養や交際が鎌状赤血球症のマウスの痛みを軽減
   セロトニンを高める抗うつ薬デュロキセチンはオピオイドの代替薬になる可能性

2. 腸内の特定の細菌が、母親マウスに子供を無視するよう促す
   特定の微生物における、動物の行動への影響についての驚くべき発見

3. 東南アジアのコウモリとセンザンコウは SARS-CoV-2 関連のコロナウイルスを持つ

4. 長時間の運動中に身体が新しい満腹因子を生成

5. ホルモンが高脂肪食マウスの筋肉喪失を防ぐ
   サルコペニアなどの筋肉を消耗する状態の治療法開発への知恵

6. バクテリオファージ療法が薬剤耐性クレブシエラニューモニエと闘う -マウス実験

7. 脂肪細胞が心不全に対する身体の反応に影響
   マウスでの実験結果が心不全治療における新しい研究分野への扉を開ける

8. 新生児マウスにはあって、ヒトにはないスキル、マイクロチップでその秘密を探る

1. 栄養や交際で鎌状赤血球症のマウスの痛みが軽減
セロトニンを高める抗うつ薬デュロキセチンがオピオイドの代替薬になる可能性

日付：2021 年 2 月 1 日
ソース：カリフォルニア大学アーバイン校
概要：

米国疾病予防管理センターによると、鎌状赤血球症は、約 10 万人のアメリカ人が罹患している痛みを伴う生涯にわたる状態であり、その大部分の罹患者はアフリカ系アメリカ人である。オピオイドは、痛みを治療する最も一般的な方法だが、それらは中毒性があり、過剰摂取は米国の主要な死因であるため、代替案が調査されている。
今回、カリフォルニア大学アーバイン校とミネソタ大学の研究者らは、セロトニンを増やすことで、豊富な食事と交際が鎌状赤血球症のマウスの痛みを軽減できることを発見した。彼らはまた、セロトニンレベルを高める抗うつ薬であるデュロキセチンが、慢性疼痛の治療においてオピオイドの代替となる可能性があることを発見した。
この研究結果は、2 月 1 日の「Scientific Reports」誌に掲載されている。

研究関連ニュース/他のトップページに戻る

＜英文＞Nutrition, companionship reduce pain in mice with sickle cell disease, UCI-led study finds | EurekAlert! Science News

NEWS RELEASE 1-FEB-2021

NUTRITION, COMPANIONSHIP REDUCE PAIN IN MICE WITH SICKLE CELL DISEASE, UCI-LED STUDY FINDS

Serotonin-boosting antidepressant duloxetine had same effect, could be opioid alternative

UNIVERSITY OF CALIFORNIA - IRVINE

Research News

Irvine, Calif. -- Researchers from the University of California, Irvine and the University of Minnesota have found that an enriched diet and companionship can reduce pain in mice with sickle cell disease by increasing serotonin. They also discovered that duloxetine, an antidepressant that boosts serotonin levels, could be an alternative to opioids in treating chronic pain.

"Finding safe and effective alternatives to opioids is a research priority, especially for patients with sickle cell disease," said Dr. Keith Hoots, director of the Division of Blood Diseases and Resources at the National Heart, Lung, and Blood Institute. "It's encouraging to see a dose-response relationship to nutrients and companions in mice, which guides future research about the role foods and friends may have in helping humans manage chronic pain."

Scientists have long suspected that feeling pain is connected to a person's mood or state of mind, but the link has never been proven beyond doubt. This study, published Feb. 1 in *Scientific Reports*, supports that connection.

"Part of pain is perception," said Kalpna Gupta, a visiting professor of medicine at UCI and the study's lead author. "For instance, if I start watching a comedy, suddenly I might forget that I was hurting; mood and a feeling of well-being block pain from being perceived."

Sickle cell disease is a painful, lifelong condition that affects about 100,000 Americans, according to the Centers for Disease Control and Prevention, the majority of whom are African American. Opioids are the most common method of treating their pain. But because they're addictive, and overdoses are a leading cause of death in the United States, Gupta's team has been investigating alternatives.

"By suggesting a safe alternative to opioids for pain control, the findings of this study have the potential to change the practice of pain management and save lives," said study co-author Dr. Rajendra Badgaiyan, a professor of psychiatry at the University of Minnesota at the time who's now at the University of Texas Health Science Center in San Antonio.

Love conquers pain

The researchers found that mouse models of sickle cell disease in a "happy environment" experienced less pain from the disorder. Over a four-week period, pain was reduced for male mice that were placed with female companions and received a diet rich in protein, fatty acids and amino acids.

Their pain decreased as the diet and companionship caused their brains to secrete serotonin. Researchers quantified the mice's pain according to the frequency with which they lifted their paws in response to stimuli and by their ability to exert force.

To confirm that serotonin caused the reduction in pain, the mice were given duloxetine, an antidepressant that works by raising the amount of serotonin in the brain. The drug decreased pain levels for both male and female mice.

The researchers believe that adapting these conditions for humans would produce the same results. Gupta is recommending that duloxetine be used in clinical trials for sickle cell patients as an alternative to opioids for pain relief.

Combating pain and prejudice

In the United States, the stigma associated with opioids can make it difficult for sickle cell patients to lessen their pain.

"Patients with sickle cell disease often have to navigate the complex social prejudices of the healthcare system in addition to dealing with severe pain," said Varun Sagi, a medical student at the University of Minnesota and a contributing author on the study. "Finding alternatives to opioids could help alleviate this burden."

Duloxetine could provide an easier-to-obtain alternative. Likewise, as in the mice, building strong relationships and improving nutrition might also stimulate serotonin production in humans and thus reduce their pain.

As the COVID-19 pandemic persists, social isolation can make relationship-building a challenge, and as unemployment continues to hover around 7 percent, many Americans lack food security. As the study suggests, this can impact pain for sickle cell patients and others, so Gupta stressed the importance of developing strategies to enhance social interaction and nutrition for those affected.

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This project was supported by National Institutes of Health grants UO1 HL117664 and RO1 HL147562.

About the University of California, Irvine:

Founded in 1965, UCI is the youngest member of the prestigious Association of American Universities. The campus has produced three Nobel laureates and is known for its academic achievement, premier research, innovation and anteater mascot. Led by Chancellor Howard Gillman, UCI has more than 36,000 students and offers 222 degree programs. It's located in one of the world's safest and most economically vibrant communities and is Orange County's second-largest employer, contributing $5 billion annually to the local economy. For more on UCI, visit http://www.uci.edu.

Media access: Radio programs/stations may, for a fee, use an on-campus ISDN line to interview UCI faculty and experts, subject to availability and university approval. For more UCI news, visit news.uci.edu. Additional resources for journalists may be found at communications.uci.edu/for-journalists.

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2. 腸内の特定の細菌が、母親マウスに子供を無視するよう促す
特定の微生物における、動物の行動への影響についての驚くべき発見

日付：2021 年 2 月 2 日
ソース：ソーク研究所
概要：

科学者らが身体にコロニーを形成する微生物（総称して微生物叢と呼ばれる）について学ぶにつれて、非常に興味深い領域の 1 つは、これらの微生物が脳に及ぼす影響である。ソーク研究所の科学者らが主導した新しい研究では、メスのマウスの腸に住んでいる、子孫を無視する大腸菌の菌株が特定され、その研究成果が 1 月 29 日の「Science Advances」誌に発表されている。
研究チームが、腸内に単一の大腸菌株を持っているマウスのグループを調査していたところ、O16：H48 MG1655 と呼ばれる特定の大腸菌株を持つマウスは、発育阻害のある子孫を産んだ。更なる調査により、栄養不良のためにマウスが小さいことが明らかになった。子供の行動は正常で、母親が作ったミルクも正常で健康的な組成で正常な量で生産されていたにもかかわらず、この特定のバクテリアが定着すると、母親の行動が悪くなることに気付いた。母マウスは子マウスを無視していたのである。追加の実験により、IGF-1 と呼ばれる成長因子を与えるか、子マウスを適切に世話することができる母マウスに引き渡すことによって、子マウスが発育阻害から救われることが明らかになった。これにより、発育阻害の原因は、子マウス自身の何かではなく、母マウスの行動にあることが確認された、としている。

研究関連ニュース/他のトップページに戻る

＜英文＞Specific bacteria in the gut prompt mother mice to neglect their pups: Researchers make unexpected discoveries about how a particular microbe influences animal behavior -- ScienceDaily

SPECIFIC BACTERIA IN THE GUT PROMPT MOTHER MICE TO NEGLECT THEIR PUPS

Researchers make unexpected discoveries about how a particular microbe influences animal behavior

Date:

February 2, 2021

Source:

Salk Institute

Summary:

As scientists learn more about the microorganisms that colonize the body -- collectively called the microbiota -- one area of intense interest is the effect that these microbes can have on the brain. A new study has identified a strain of E. coli bacteria that, when living in the guts of female mice, causes them to neglect their offspring.

FULL STORY

As scientists learn more about the microorganisms that colonize the body -- collectively called the microbiota -- one area of intense interest is the effect that these microbes can have on the brain. A new study led by Salk Institute scientists has identified a strain of *E. coli* bacteria that, when living in the guts of female mice, causes them to neglect their offspring.

The findings, published January 29, 2021, in the journal *Science Advances*, show a direct link between a particular microbe and maternal behavior. Although the research was done in mice, it adds to the growing body of science demonstrating that microbes in the gut are important for brain health and can affect development and behavior.

"To our knowledge, this is the first demonstration that the intestinal microbiota is important for promoting healthy maternal behavior and bonding between mom and offspring in an animal model," says Professor Janelle Ayres, Laboratory Head of Salk's Molecular and Systems Physiology Laboratory and senior author of the paper. "It adds to the ever-growing evidence that there's a gut-brain connection, and that microbes are important for regulating the behavior of the host that they're inhabiting."

The ways in which the microbiota can impact mental health and neurological disorders is a growing area of research. The makeup of the gut microbiota in people has been linked to depression, anxiety, autism and other conditions. But it has been difficult to study how individual strains of bacteria exert their influence on human behavior, a connection often called the microbiota-gut-brain axis.

In her lab, Ayres uses mice to study how body systems and the brain interact with each other to promote health. This includes focusing on how body processes are regulated by microbes and the ways in which microbes affect growth and behavior. In the current experiments, she and her team were investigating groups of mice that each had a single strain of *E. coli* in their gut. Mice with one particular strain of *E. coli*, called O16:H48 MG1655, mothered offspring that had stunted growth. Further examination revealed that the mice were smaller because they were malnourished.

"We found that the pups' behavior was normal, and the milk made by the mothers was of normal, healthy composition and was being produced in normal amounts," Ayres says. "We eventually figured out that being colonized with this particular bacteria led to poor maternal behavior. The mice were neglecting their pups."

Additional experiments revealed that the mice could be rescued from stunted growth, either by giving them a growth factor called IGF-1 or handing them off to foster mouse mothers that could take care of them properly. This confirmed that the cause of stunted growth was coming from the mothers' behavior rather than something in the pups themselves.

"Our study provides an unprecedented understanding of how the intestinal microbiota can disrupt maternal behavior and how this can negatively impact development of an offspring," says first author Yujung Michelle Lee, a former graduate student in Ayres' lab and now a postdoctoral fellow at Genentech. "It is very interesting to me that establishment of a healthy mother-infant relationship is driven by factors beyond hormones, and that the microorganisms residing in our bodies play a significant role in it."

Ayres and her team plan to study how these microbes provoke changes in mouse behavior. Early findings suggest the bacteria might be affecting levels of serotonin, the hormone associated with feelings of happiness and well-being, but more work is needed.

"It's very hard to study these relationships in humans, because the human microbiota contains hundreds of different species of microorganisms," says Ayres, who holds the Helen McLoraine Developmental Chair. "But once we understand more about the mechanisms in animal models, we may be able translate our findings to humans to determine whether the microbes and their effects might be the same."

The O16:H48 MG1655 strain has been found in human guts and was previously believed to have no positive or negative effects.

Story Source:

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

Journal Reference:

1. Yujung Michelle Lee, Andre Mu, Martina Wallace, Jivani M. Gengatharan, Annalee J. Furst, Lars Bode, Christian M. Metallo, Janelle S. Ayres. **Microbiota control of maternal behavior regulates early postnatal growth of offspring**. *Science Advances*, 2021; 7 (5): eabe6563 DOI: 10.1126/sciadv.abe6563

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3. 東南アジアのコウモリとセンザンコウは SARS-CoV-2 関連のコロナウイルスを持つ

日付：2021 年 2 月 9 日
ソース：Duke-NUS 医科大学
概要：

世界保健機関（WHO）は、SARS-CoV-2 の起源と早期感染について武漢での調査を継続しているが、シンガポールのデューク NUS 医科大学とタイのチュラロンコン大学の科学者らが主導した新しい研究では、SARS-CoV-2 関連コロナウイルス（SC2r-CoV）は、タイの動物の間で流行している、としている。
本日「Nature Communications」誌に発表されたこの研究は、ウイルスに対する高レベルの中和抗体が東南アジアの国で見付かったコウモリとセンザンコウの両方に存在したことを報告している。更に、この研究は、この地域でより多くの SC2r-CoV が発見される可能性が高いことを示しており、コウモリの個体数が多い東南アジアは、そのようなウイルスのホットスポットである可能性が高いかもしれない、としている。

研究関連ニュース/他のトップページに戻る

＜英文＞Bats and pangolins in Southeast Asia harbor SARS-CoV-2-related coronaviruses, reveals new study (medicalxpress.com)

FEBRUARY 9, 2021

BATS AND PANGOLINS IN SOUTHEAST ASIA HARBOR SARS-COV-2-RELATED CORONAVIRUSES, REVEALS NEW STUDY

by Duke-NUS Medical School

Credit: CC0 Public Domain

While the World Health Organization (WHO) continues its mission to Wuhan investigating the origin and early transmission of SARS-CoV-2, a new study led by scientists from Duke-NUS Medical School, Singapore, and Chulalongkorn University, Thailand, shows that SARS-CoV-2-related coronaviruses (SC2r-CoVs) are circulating in animals as far away as Thailand. The study, published in *Nature Communications* today, reported that high levels of neutralizing antibodies against the virus were present in both bats and pangolins found in the Southeast Asian country. The study further indicates that more SC2r-CoVs are likely to be discovered in the region. Southeast Asia with its large and diverse bat populations may be a more likely hotspot for such viruses.

"This is an important discovery in the search for the origin of SARS-CoV-2, which was made possible by rapid application of cutting-edge technology through transparent international collaboration," said Dr. Supaporn Wacharapluesadee, from Thai Red Cross Emerging Infectious Diseases Health Science Centre, Faculty of Medicine, Chulalongkorn University, Bangkok Thailand.

In the study, the team examined Rhinolophus bats in a Thai cave. SARS-CoV-2 neutralizing antibodies were detected in bats of the same colony and in a pangolin at a wildlife checkpoint in Southern Thailand.

"Our study extended the geographic distribution of genetically diverse SARS-CoV-2-related coronaviruses from Japan and China to Thailand over a 4,800-km range. Cross-border surveillance is urgently needed to find the immediate progenitor virus of SARS-CoV-2," said Dr. Chee Wah Tan, Senior Research Fellow with Duke-NUS' Emerging Infectious Diseases (EID) program and co-author of this study.

The team conducted serological investigations using the SARS-CoV-2 surrogate virus neutralization test (sVNT) developed at Duke-NUS in early 2020.

"Our study demonstrates that our SARS-CoV-2 surrogate virus neutralization test, developed mainly for determining neutralizing antibodies in humans to monitor vaccine efficacy and detect past infections, can also be critical for tracing the animal origin and animal-human spillover events," said Professor Wang Linfa from Duke-NUS' EID program and corresponding author of this study.

Prof Wang's team invented the sVNT assay, trade named cPass, which has been granted Emergency Use Authorisation by the US FDA to determine SARS-CoV-2-neutralizing antibodies in human sera, due to its good performance concordance with live virus-based assays.

"Studies like this are crucial in furthering our understanding of the many SARS-CoV-2-related viruses that exist in the wild. This work is also timely as investigations into the origins of SARS-CoV-2 are ongoing and may provide further leads on the origin of this outbreak. Such studies also play a key role in helping us be better prepared against future pandemics as they provide a more detailed map of zoonotic threats, " said Prof Patrick Casey, Senior Vice Dean for Research at Duke-NUS.

Explore further

Follow the latest news on the coronavirus (COVID-19) outbreak

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More information: Wacharapluesadee, S et al. Evidence for SARS-CoV-2 related coronaviruses circulating in bats and pangolins in Southeast Asia. *Nat Commun* 12, 972 (2021). doi.org/10.1038/s41467-021-21240-1

Journal information: Nature Communications

Provided by Duke-NUS Medical School

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4. 長時間の運動中に身体が新しい満腹因子を生成

日付:2021 年 2 月 16 日
ソース:コペンハーゲン大学
概要:

研究者らが関心を持っている新たな抗肥満薬候補の 1 つは、齧歯類に投与されると食欲と体重を低下させるホルモン GDF15 であるが、今回コペンハーゲン大学の研究者らによって、長時間の激しい運動中に、おそらく生理学的ストレス信号として、身体が大量の GDF15 を生成することが発見された。
そこで研究者らは、薬物として投与される GDF15(薬理学)と、激しい運動に反応して自然に放出される GDF15(生理学)の根本的な違いをよく理解することに焦点を当てた。
齧歯類とサルにこのホルモンが薬理学的に投与されると、食欲を低下させるだけでなく、吐き気および病気を促進する。又、GDF15 が生理学的に自然放出された時にどのように機能するかについては今までほとんど知られていないため、研究者らはヒトとマウスにおいて実験をした。ヒトにおいては、2 時間を超える運動で、GDF15 の循環が 4～5 倍増加した。マウスにおいては、GDF15 を薬物として投与した場合と同様に、食欲が低下した。
研究者らは、GDF15 がヒトの行動に影響を与えるかどうかを理解するには、さらに多くの研究が必要である、として、今後の研究によって、身体によって生成されたときの GDF15 の効果を明らかにしたい、としている。

研究関連ニュース/他のトップページに戻る

＜英文＞The body produces new satiety factor during prolonged exercise | EurekAlert! Science News

NEWS RELEASE 16-FEB-2021

THE BODY PRODUCES NEW SATIETY FACTOR DURING PROLONGED EXERCISE

UNIVERSITY OF COPENHAGEN - THE FACULTY OF HEALTH AND MEDICAL SCIENCES

Research News

A drug that helps us to eat less could help the more than 650 million people around the world who live with obesity. One of the emerging drug candidates that interest researchers is the hormone GDF15 that, when given to rodents, lowers their appetite and body weight. New research from the University of Copenhagen finds that the body produces large amounts GDF15 during extended bouts of vigorous exercise, presumably as a physiological stress signal.

This finding highlights central differences between GDF15 given as a drug (pharmacology), and GDF15 released naturally in response to vigorous exercise (physiology). This is an important distinction in understanding GDF15's role in appetite regulation and energy balance, with implications for its role as a possible anti-obesity drug.

"Whether there are any physiological conditions that implicates GDF15 as a regulator of energy metabolism remains an unsolved mystery," says Associate Professor Christoffer Clemmensen from the Novo Nordisk Foundation Center for Basic Metabolic Research (CBMR) at the University of Copenhagen.

Christoffer Clemmensen, PhD student Trine Sand Nicolaisen and Assistant Professor Anders Bue Klein led the research in collaboration with the Department of Nutrition Exercise and Sports at the University of Copenhagen and the findings were published in Nature Communications.

Their goal was to better understand the physiological role of GDF15 in energy metabolism and behavior. Recent findings in rodents and monkeys suggest that the hormone, when administered pharmacologically, lowers appetite but also promotes nausea and sickness. Other studies have shown that the drug metformin promotes weight loss by increasing the levels of GDF15.

Different pharmocological and physiological effects of GDF15

Little is known about how GDF15 functions when released naturally by the body, however. The researchers set out to fill this knowledge gap with a series of experiments on humans and mice. Among their main findings was that prolonged exercise beyond two hours in humans results in a four to five-fold increase in the circulation of GDF15, suggesting that GDF15 functions as an exercise-induced stress signal.

To test this idea, the researchers used animal models. They found that giving GDF15 to mice as a drug clearly lowered their motivation to exercise and reduced their appetite. But when the mice were vigorously exercised, to stimulate the physiological release of GDF15, it did induce the same response on behavior and food intake.

These findings underscore a difference between physiological GDF15 and pharmacological GDF15. Christoffer Clemmensen stresses that more studies are needed to understand this mismatch and whether GDF15 also has behavioral effects in humans. He and his team will now focus on clarifying the effects of GDF15 when produced by the body.

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5. ホルモンが高脂肪食マウスの筋肉喪失を防ぐ
サルコペニアなどの筋肉を消耗する状態の治療法開発への知恵

日付：2011 年 2 月 23 日
ソース：南カリフォルニア大学（USC）
概要：

USC の新しい研究は、体重増加を防ぎ、代謝を正常化することが知られているホルモンが、マウスの健康な筋肉を維持するのにも役立つ、として今月「American Journal of Physiology-Endocrinology and Metabolism」誌で発表されている。
この研究では、高脂肪食のマウスを運動の効果を模倣することが知られているミトコンドリア由来ペプチドである MOTS-c で治療すると、筋肉の成長を阻害するタンパク質であるミオスタチンのレベルが低下するため、肥満に関連する筋萎縮を防ぐことができた、としている。研究者らはまた、ヒトの MOTS-c レベルが高いほどミオスタチンのレベルが低いことと相関している、としている。他にもいくつかのミオスタチン阻害剤が同定されているものの、それらはまだ臨床試験で筋肉の消耗状態にうまく対処することができていないため、USC の研究者によると、これは筋肉量の改善だけでは不十分である可能性がある。彼らは、ミトコンドリア機能を高めることも重要であると信じており、MOTS-c 由来の治療法はこの点で特に有望である可能性がある、としている。

研究関連ニュース/他のトップページに戻る

＜英文＞Hormone helps prevent muscle loss in mice on high fat diets, USC study finds | EurekAlert! Science News

NEWS RELEASE 22-FEB-2021

HORMONE HELPS PREVENT MUSCLE LOSS IN MICE ON HIGH FAT DIETS, USC STUDY FINDS

Researchers also identify molecular pathway that can inform development of treatments for muscle-wasting conditions such as sarcopenia.

UNIVERSITY OF SOUTHERN CALIFORNIA

Research News

A new study suggests that a hormone known to prevent weight gain and normalize metabolism can also help maintain healthy muscles in mice. The findings present new possibilities for

treating muscle-wasting conditions associated with age, obesity or cancer, according to scientists from the University of Southern California Leonard Davis School of Gerontology.

The research, published this month in the *American Journal of Physiology-Endocrinology and Metabolism*, addresses the related problems of age and obesity-induced muscle loss, conditions which can lead to increased risk of falls, diabetes and other negative health impacts. It also adds to a growing number of findings describing beneficial effects of MOTS-c, a mitochondrial-derived peptide that is known to mimic the effects of exercise.

In this study, treating mice on a high-fat diet with MOTS-c helped prevent obesity-associated muscle atrophy by decreasing levels of myostatin, a protein that inhibits muscle growth--myostatin levels were 40% lower in MOTS-c treated mice compared to control mice. The researchers also found that higher MOTS-c levels in humans were correlated with lower levels of myostatin.

The mice findings show MOTS-c improves not only metabolic function but muscle mass as well.

Through molecular analysis, the researchers also identified the specific signaling pathway regulated by MOTS-c, demonstrating for the first time "that MOTS-c modulates the CK2-PTEN-AKT-FOXO1 pathway to inhibit myostatin expression and muscle wasting," and suggesting that the exercise mimetic effect of MOTS-c may be derived from its previously unknown role as a myostatin inhibitor, according to the paper.

"Knowing the signaling pathway affected by MOTS-c is really important to the discovery of possible treatments," says corresponding author Su Jeong Kim, a research associate professor at the USC Leonard Davis School. "This insight provides a target for potential drug development efforts and can be rapidly translated into clinical trials of MOTS-c and related analogues."

Though several other myostatin inhibitors have been identified, they have yet to successfully reduce muscle wasting conditions in clinical trials. This may be because improving muscle mass alone is not enough, say the USC researchers. They believe boosting mitochondrial function is also key and say that MOTS-c-derived treatments could be especially promising in this regard.

Co-corresponding author Pinchas Cohen, professor of gerontology, medicine and biological sciences and dean of the USC Leonard Davis School, along with Changhan David Lee, assistant professor at the USC Leonard Davis School, first described MOTS-c and its effects on metabolism in 2015. Their mice studies have shown that MOTS-c administration improves both high-fat diet- and aging-induced insulin resistance as well as exercise capacity and median life span.

"Taken together, our work suggests that MOTS-c can address mitochondrial dysfunction," says Cohen. "This study can help improve healthy aging by opening up new avenues for research on

how to treat conditions such as insulin resistance-induced skeletal muscle atrophy as well as other muscle-wasting conditions, including sarcopenia."

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This study was conducted in collaboration with Hiroshi Kumagai (lead author) and Noriyuki Fuku from the Graduate School of Health and Sports Science, Juntendo University, Chiba, Japan; Ana Raquel Coelho and Paulo J. Oliveira from the Center for Neuroscience and Cell Biology, University of Coimbra, Portugal; Hirofumi Zempo from Tokyo Medical and Dental University, Japan; Seiji Maeda, Faculty of Health and Sport Sciences, University of Tsukuba, Japan; and Junxiang Wan, Hemal Mehta , Kelvin Yen, and Amy Huang of the USC Leonard Davis School of Gerontology.

This work was supported by a Glenn/AFAR Postdoctoral Fellowship Program for Translational Research on Aging to Kim, by R01AG061834, P01AG034906, R56AG062693 and an AFAR BIG AWARD grants to Cohen, by the Fundação Luso-Americana para o Desenvolvimento (FLAD) Healthcare 2020 grant to Oliveira, and by a PhD fellowship from Portuguese FCT SFRH/BD/103399/2014) to Coelho. Cohen is a co-founder, stockholder and board member of Cohbar Inc. CohBar is developing analogues of mitochondrial peptides including of MOTS-c for metabolic diseases of aging and is in clinical trials with a MOTS-c analogue.

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6. バクテリオファージ療法が薬剤耐性クレブシエラニューモニエと闘う –マウス実験

日付：2021 年 2 月 23 日
ソース：NIH/国立アレルギー感染症研究所
概要：

抗生物質の代わりにウイルスを使用して厄介な薬剤耐性菌を飼いならすのは、バクテリオファージとか「ファージ療法」として知られる有望な戦略である。国立衛生研究所の科学者らは、2 つの異なるバクテリオファージウイルスを使用して、多剤耐性クレブシエラニューモニエ配列タイプ 258（ST258）に感染した研究用マウスの治療に成功した。
細菌 K.pneumoniae ST258 は、抗生物質耐性の最大の脅威として米国の疾病管理予防センターのリストにも載っている。高い罹患率と死亡率は、未治療の K.pneumoniae 感染症に関連している。

研究関連ニュース/他のトップページに戻る

＜英文＞Mouse study shows bacteriophage therapy could fight drug-resistant Klebsiella pneumoniae -- ScienceDaily

MOUSE STUDY SHOWS BACTERIOPHAGE THERAPY COULD FIGHT DRUG-RESISTANT KLEBSIELLA PNEUMONIAE

Date:

February 23, 2021

Source:

NIH/National Institute of Allergy and Infectious Diseases

Summary:

Using viruses instead of antibiotics to tame troublesome drug-resistant bacteria is a promising strategy, known as bacteriophage or 'phage therapy.' Scientists have used two different bacteriophage viruses individually and then together to successfully treat research mice infected with multidrug-resistant Klebsiella pneumoniae sequence type 258 (ST258).

FULL STORY

Using viruses instead of antibiotics to tame troublesome drug-resistant bacteria is a promising strategy, known as bacteriophage or "phage therapy." Scientists at the National Institutes of Health have used two different bacteriophage viruses individually and then together to successfully treat research mice infected with multidrug-resistant *Klebsiella pneumoniae* sequence type 258 (ST258). The bacterium *K. pneumoniae* ST258 is included on a CDC list of biggest antibiotic resistance threats in the United States. High rates of morbidity and mortality are associated with untreated *K. pneumoniae* infections.

Phage therapy has been pursued for about a century, though conclusive research studies are rare and clinical results -- from a handful of reports -- have provided mixed results. In the new paper published in the journal *mBio*, the NIH scientists note that phages are of great interest today because of a dearth of alternative treatment options for drug-resistant infections. Bacterial resistance has emerged against even the newest drug combinations, leaving some patients with few or no effective treatment options.

In research conducted in Hamilton, Montana, at Rocky Mountain Laboratories -- part of the NIH's National Institute of Allergy and Infectious Diseases -- and in collaboration with the National Cancer Institute in Bethesda, Maryland, scientists completed a series of studies on research mice infected with ST258. They treated the mice with either phage P1, phage P2, or a combination of the two, all injected at different times following ST258 infection. The scientists had isolated phages P1 and P2 in 2017 from raw sewage that they screened for viruses that would infect ST258 -- an indication that phages can be found just about any place. Phages P1 and P2 are viruses from the order Caudovirales, which naturally infect bacteria.

Each of the three experimental treatment regimens helped the mice recover from ST258 infection. The scientists noted that the dose of phage provided was less vital to recovery than was the timing of when the dose was received. Mice treated 1 hour after infection showed the strongest recovery, followed by those treated eight hours after infection and then those treated at 24 hours. Control mice treated with saline all quickly developed severe disease and died.

The scientists also checked the blood and tissue of phage-treated mice for the presence of ST258 bacteria and found there were significantly fewer bacteria at all time points regardless of the treatment method used, as compared to control mice.

Unfortunately, the scientists also found that ST258 bacteria recovered in the blood and tissue samples of phage-treated mice already had begun developing phage resistance, a finding they are continuing to investigate. The group also is studying how phage therapy results compare between samples of ST258-infected mouse blood and human blood, and are examining whether components of human blood can interfere with phage efficacy.

This study represents a first step in evaluating the use of phage therapy for treatment of severe *K. pneumoniae* ST258 infection in humans.

Story Source:

Materials provided by **NIH/National Institute of Allergy and Infectious Diseases**. *Note: Content may be edited for style and length.*

Journal Reference:

1. Shayla Hesse, Natalia Malachowa, Adeline R. Porter, Brett Freedman, Scott D. Kobayashi, Donald J. Gardner, Dana P. Scott, Sankar Adhya, Frank R. DeLeo. **Bacteriophage Treatment Rescues Mice Infected with Multidrug-Resistant Klebsiella pneumoniae ST258**. *mBio*, 2021; 12 (1) DOI: 10.1128/mBio.00034-21

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7. 脂肪細胞が心不全に対する身体の反応に影響
マウスでの実験結果が心不全治療における新しい研究分野への扉を開ける

日付：2021 年 2 月 23 日
ソース：アルバータ大学（カナダ エドモントン）
概要：

アルバータ大学の研究者らは、心不全の際に身体が脂肪細胞から血流に放出する脂肪の量を制限することで、患者の状態を改善できることを発見した。
心不全などのストレス時、身体はストレスホルモンを放出するが、心臓はこれ以上機能することができず、実際にはより速くポンプをかけることによってさらに損傷を受けるため、身体はより多くのストレスホルモンを放出し、プロセスがカスケードし、心臓機能が低下し続ける。心不全の一般的な治療法がベータ遮断薬であるのはこのためで、ベータ遮断薬は、心臓に対するストレスホルモンの影響を遮断するように設計されている。また、ストレスホルモンの放出は、脂肪細胞の貯蔵沈着物から血流への脂肪の放出を引き起こし、脂肪分解と呼ばれるプロセスとして身体に余分なエネルギーを提供する。研究チームは、心不全の間、マウスの脂肪細胞も身体全体で炎症を起こし、通常よりも早く脂肪を動員して放出し、心臓と身体の残りの部分に炎症を引き起こすことを発見した。この炎症は心臓にさらなるストレスを与え、カスケード効果を高め、損傷を増やし、心臓機能を低下させる。
今回マウスの脂肪細胞から脂肪の動員を阻害することができる薬によって、実際に炎症によって引き起こされる損傷から心臓を保護することに成功した、としている。
この研究成果は「American Journal of Physiology」誌に掲載されている。

研究関連ニュース/他のトップページに戻る

＜英文＞Fat cells may influence how the body reacts to heart failure: Promising results in mice open door to new areas of research in treating patients with heart failure. -- ScienceDaily

FAT CELLS MAY INFLUENCE HOW THE BODY REACTS TO HEART FAILURE

Promising results in mice open door to new areas of research in treating patients with heart failure.

Date:

February 23, 2021

Source:

University of Alberta Faculty of Medicine & Dentistry

Summary:

Researchers have found that limiting the amount of fat the body releases into the bloodstream from fat cells when in heart failure could help improve outcomes for patients.

FULL STORY

University of Alberta researchers have found that limiting the amount of fat the body releases into the bloodstream from fat cells during heart failure could help improve outcomes for patients.

In a recent study published in the *American Journal of Physiology*, Jason Dyck, professor of pediatrics in the Faculty of Medicine & Dentistry and director of the U of A's Cardiovascular Research Centre, found that mice with heart failure that were treated with a drug blocking the release of fat into the bloodstream from fat cells saw less inflammation in the heart and throughout the body, and had better outcomes than a control group.

"Many people believe that, by definition, heart failure is only a condition of the heart. But it's much broader and multiple organs are affected by it," said Dyck, who holds the Canada Research Chair in Molecular Medicine and is a member of the Alberta Diabetes Institute and the Women and Children's Health Research Institute. "What we've shown in mice is that if you can target fat cells with a drug and limit their ability to release stored fat during heart failure, you can protect the heart and improve cardiac function.

"I think it really opens the door for other avenues of investigation and therapies for treating heart failure," Dyck noted.

During times of stress, such as heart failure, the body releases stress hormones, such as epinephrine and norepinephrine, to help the heart compensate. But because the heart can't function any better -- and is in fact damaged further by being forced to pump faster -- the body releases more stress hormones and the process cascades, with heart function continuing to decline. This is why a common treatment for heart failure is beta-blocker drugs, which are designed to block the effects of stress hormones on the heart.

The release of stress hormones also triggers the release of fat from its storage deposits in fat cells into the bloodstream to provide extra energy to the body, a process called lipolysis. Dyck's team found that during heart failure, the fat cells in mice were also becoming inflamed throughout the body, mobilizing and releasing fat faster than normal and causing inflammation in the heart and rest of the body. This inflammation put additional stress on the heart, adding to the cascade effect, increasing damage and reducing heart function.

"Our research began by looking at how the function of one organ can affect other organs, so I thought it was very fascinating to find that a fat cell can influence cardiac function in heart failure," Dyck said. "Fortunately, we had a drug that could inhibit fat mobilization from fat cells in mice, which actually protected the hearts from damage caused by inflammation."

Dyck points out that although his results are promising, more work is needed to better understand the exact mechanisms at play in the process and develop a drug that could work in humans.

"This work is a proof-of-concept showing that abnormal fat-cell function contributes to worsening heart failure, and now we're working on understanding the mechanisms of how the drug works to limit lipolysis better," he said. "Once we get that, that's the launchpad for making sure it's safe and efficacious, then advancing it to our chemists, and then maybe some early trials in humans."

Dyck said the findings -- and a better understanding of how organ functions affect other organs -- could be used to develop new approaches to several other diseases.

"We know that people have high rates of lipolysis when they have heart failure, so I presume this approach would benefit all types of heart failure," he said. "But if you consider that inflammation is associated with a wide variety of different diseases, like cancer, diabetes or other forms of heart disease, then this approach could have a much wider benefit."

Dyck's research was funded by the Heart and Stroke Foundation and the Canadian Institutes of Health Research.

Story Source:

Materials provided by **University of Alberta Faculty of Medicine & Dentistry**. Original written by Ryan O'Byrne. *Note: Content may be edited for style and length.*

Journal Reference:

1. Shingo Takahara, Mourad Ferdaoussi, Nikola Srnic, Zaid H. Maayah, Shubham Soni, Anna K. Migglautsch, Rolf Breinbauer, Erin E. Kershaw, Jason R. B. Dyck. **Inhibition of ATGL in adipose tissue ameliorates isoproterenol-induced cardiac remodeling by reducing adipose tissue inflammation**. *American Journal of Physiology-Heart and Circulatory Physiology*, 2021; 320 (1): H432 DOI: 10.1152/ajpheart.00737.2020

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8. 新生児マウスにはあって、ヒトにはないスキル、マイクロチップでその秘密を探る

日付：2021 年 2 月 25 日
ソース：ニューサウスウェールズ大学
概要：

新生児マウスには、ヒトが羨む特別なスキルがある。それは、生後 7 日間は、損傷した心臓組織を再生する能力がある、ということである。ヒトの場合、受けた心臓の怪我は、永久的な損傷に繋がる可能性がある。もし私達が新生児マウスのように心臓修復する方法を学ぶことができたら？
UNSW シドニーが率いる研究チームは、科学者らがマウスの心臓細胞の再生能力を研究するのに役立つマイクロチップを開発し、このマイクロチップが新しい再生心臓医学研究への道を開くのに役立つ可能性がある、としている。

研究関連ニュース/他のトップページに戻る

＜英文＞Baby mice have a skill that humans want, and this microchip might help us learn it -- ScienceDaily

BABY MICE HAVE A SKILL THAT HUMANS WANT, AND THIS MICROCHIP MIGHT HELP US LEARN IT

Date:

February 25, 2021

Source:

University of New South Wales

Summary:

A new microchip could help scientists uncover secrets of heart regeneration in baby mice.

FULL STORY

Baby mice might be small, but they're tough, too.

For their first seven days of life, they have the special ability to regenerate damaged heart tissue.

Humans, on the other hand, aren't so lucky: any heart injuries we suffer could lead to permanent damage. But what if we could learn to repair our hearts, just like baby mice?

A team of researchers led by UNSW Sydney have developed a microchip that can help scientists study the regenerative potential of mice heart cells. This microchip -- which combines microengineering with biomedicine -- could help pave the way for new regenerative heart medicine research.

"We've developed a simple, reliable, cheap and fast way to identify and separate these important mouse heart cells," says lead author Dr Hossein Tavassoli, a biomedical engineer and stem cell researcher at UNSW Medicine & Health who conducted this work as part of his doctoral thesis.

"Our method uses a microchip that's easy to fabricate and can be made in any laboratory in the world."

The process for identifying and separating mice heart cells is rather complex.

First, scientists need to separate the *right kind* of heart cells (called proliferative cardiomyocytes) from other types of cells present in the heart.

Their next challenge is keeping the cells alive.

"Newborn mice heart cells (called proliferative cardiomyocytes) are very sensitive," says Dr Vashe Chandrakanthan, a senior research fellow at UNSW Medicine & Health and co-senior author of the study.

"Only about 20 per cent usually survive the conventional isolation and separation process. If we want to study these cells, we need to isolate them before they undergo cell death."

Dr Tavassoli says that this new method is much more efficient.

"We reduced the stress applied on these cells by minimising the isolation and processing time," he says. "Our method can purify millions of cells in less than 10 minutes.

"Almost all of the cells survived when we used our microfluidic chip -- over 90 per cent."

The spiral-shaped device is a microfluidic chip -- that is, a chip designed to handle liquids on tiny scale. It filters cells according to their size, separating the cardiomyocytes from other cells. The chip costs less than $500 to produce, making it cheaper than other isolation and separation methods.

This tool will make it easier for researchers to study how baby mice repair their hearts -- and whether humans might be able to use the same technique.

"Heart disease is the number one killer in the world," says Dr Tavassoli. "In Australia, someone dies of heart disease every 12 minutes, and every four hours a baby is born with a heart defect.

"We hope that our device will help accelerate heart disease research."

Characterising mice heart cells

Once the heart cells were separated from other cells with the help of their chip, the researchers seized the opportunity to study the cells' physico-mechanical properties -- that is, the way they respond to force.

This involved asking questions like 'How do these individual heart cells beat?', 'Do the cells have distinct features?' and 'What are their differences in size, shape and elasticity?'.

The findings could provide new insights for developing materials that repair heart tissue, like cardiac patches, scaffolds and hydrogels.

"The fast, large-scale characterisation of cells' physico-mechanical features is a relatively new field of research," says Dr Tavassoli, who originally trained as an engineer before specialising in medicine.

"This is the first time microfluidic technology has been used to study mechanical properties of baby mouse heart cells."

A multipurpose microchip

Dr Chandrakanthan says that even though the microchip was created for baby mouse heart cells, it could potentially be adapted for use in other types of cell applications.

"The principles are compatible with isolating cardiomyocytes from mouse heart cells of all ages," he says.

"We could potentially also use this method to separate not only the heart cells, but all sorts of cells from different organs."

Dr Tavassoli says this method could also help other areas of medical research, including cardiac biology, drug discovery and nanoengineering. He is currently conducting research at the Garvan Institute and Lowy Cancer Research Centre on how this method could help cancer diagnosis.

"This microchip opens up the opportunity for new discoveries by researchers all over the world," he says.

Story Source:

Materials provided by **University of New South Wales**. Original written by Sherry Landow. *Note: Content may be edited for style and length.*

Journal Reference:

1. Hossein Tavassoli, Prunella Rorimpandey, Young Chan Kang, Michael Carnell, Chris Brownlee, John E Pimanda, Peggy P.Y. Chan, Vashe Chandrakanthan. **Label-Free Isolation and Single Cell Biophysical Phenotyping Analysis of Primary Cardiomyocytes Using Inertial Microfluidics**. *Small*, 2020; 17 (8): 2006176 DOI: 10.1002/smll.202006176