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Ruth Francis Nature Publishing Group

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Confinement of antihydrogen for 1,000 seconds DOI: 10.1038/nphys2025

Atoms made of a particle and an antiparticle are unstable, usually surviving less than a microsecond. Antihydrogen, made entirely of antiparticles, is believed to be stable, and it is this longevity that holds the promise of precision studies of matter–antimatter symmetry.

We have recently demonstrated trapping of antihydrogen atoms by releasing them after a confinement time of 172ms. A critical question for future studies is: how long can anti-atoms be trapped? Here, we report the observation of anti-atom confinement for 1,000 s, extending our earlier results by nearly four orders of magnitude. Our calculations indicate that most of the trapped anti-atoms reach the ground state.

Further, we report the first measurement of the energy distribution of trapped antihydrogen, which, coupled with detailed comparisons with simulations, provides a key tool for the systematic investigation of trapping dynamics. These advances open up a range of experimental possibilities, including precision studies of charge–parity–time reversal symmetry and cooling to temperatures where gravitational effects could become apparent.

Upping the anti

DOI: 10.1038/nphys2025

The creation, trapping and storage of antihydrogen atoms for up to 1,000 seconds is reported online in *Nature Physics* this week. This achievement not only represents the longest time period so far that antihydrogen has been captured, but it also brings us closer to answering the question: 'Do matter and antimatter obey the same laws of physics?'

Antimatter particles are routinely produced in particle accelerators as well as in space, but holding onto them, particularly the neutral ones, is the main difficulty. This is because antimatter and matter will annihilate on contact and conventional containers are made of matter. The ALPHA collaboration at CERN demonstrated last year that they could instead use a magnetic trap to capture antihydrogen particles, and managed to store them for 172 milliseconds. The team now increase that period by more than 5,000-fold, meaning that the antihydrogen atoms have time to reach their ground state, rather than only existing in the highly excited states created by previous experiments, in which they are quickly annihilated. Such long storage times allowed the first measurements of the characteristics of trapped anti-atoms, which provide information about the formation dynamics of antihydrogen atoms and their kinetic energy distribution.

Improved traps will potentially provide plenty of interaction time for future experiments to probe the anti-atoms' quantum nature with lasers or microwaves, or to cool them down to study the gravitational effects on antimatter.

What was the strategy?

- Work with authors and press officers
- Press briefing under embargo telephone
- CERN website updated as paper published
- Twitter feeds and responding to comments
- Facebook group posting and responding to comments



La nave 'Enterprise', dirigida por **el capitan Kirk en la ya clásica serie de ciencia ficción Star Trek**, se impulsaba gracias a la energía proporcionada por la antimateria presente en el Universo. Es sólo ciencia ficción, porque en la realidad la antimateria es, cuanto menos, difícil de encontrar.

Sin embargo, el experimento Alpha del acelerador de partículas del CERN ha conseguido **atrapar átomos de antimateria durante más de 1.000 segundos** (16 minutos): el tiempo suficiente para empezar a estudiar sus propiedades en detalle. El hallazgo ha sido publicado en la revista 'Nature Physics'.

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SCIENTISTS have successfully stored anti-matter for a record 16 minutes. Boffins at the European nuclear research facility, CERN, created 300 atoms of anti-hydrogen and stopped them from imploding for more than quarter of an hour. This is 5,000 times longer than they had previously managed to bottle the elusive substance.

The feat is so incredible because anti-matter is destroyed as soon as it comes into contact with its nemesis, matter - which is anything known to man, even thin air.

It is hoped the anti-matter might help physicists increase their understanding of the nature and origins of the universe.

De novo cardiomyocytes from within the activated adult heart after injury

DOI:10.1038/nature10188

A significant bottleneck in cardiovascular regenerative medicine is the identification of a viable source of stem/progenitor cells that could contribute new muscle after ischaemic heart disease and acute myocardial infarction. A therapeutic ideal—relative to cell transplantation—would be to stimulate a resident source, thus avoiding the caveats of limited graft survival, restricted homing to the site of injury and host immune rejection. Here we demonstrate in mice that the adult heart contains a resident stem or progenitor cell population, which has the potential to contribute bona fide terminally differentiated cardiomyocytes after myocardial infarction. We reveal a novel genetic label of the activated adult progenitors via reexpression of a key embryonic epicardial gene, Wilm's tumour 1 (Wt1), through priming by thymosin β 4, a peptide previously shown to restore vascular potential to adult epicardium-derived progenitor cells with injury. Cumulative evidence indicates an epicardial origin of the progenitor population, and embryonic reprogramming results in the mobilization of this population and concomitant differentiation to give rise to de novo cardiomyocytes. Cell transplantation confirmed a progenitor source and chromosome painting of labelled donor cells revealed transdifferentiation to a myocyte fate in the absence of cell fusion. Derived cardiomyocytes are shown here to structurally and functionally integrate with resident muscle; as such, stimulation of this adult progenitor pool represents a significant step towards resident-cell-based therapy in human ischaemic heart disease.

Stem cells: Mending a broken heart (AOP) *PRESS BRIEFING*

DOI: 10.1038/nature10188

A small peptide can boost the formation of new heart muscle cells (cardiomyocytes) after a heart attack, according to research in mice published this week in *Nature*. These findings indicate that resident progenitor cells can be encouraged to differentiate into cardiomyocytes after heart damage and may have important implications for cardiovascular regenerative medicine.

One of the biggest hurdles for effective cell-based therapy in cardiovascular regenerative medicine is the identification of an appropriate source of resident cells that can give rise to functional cardiomyocytes. Thymosin b4, previously shown to encourage regrowth of blood vessels and improve heart function after injury in mice, can activate progenitor cells that could differentiate into new cardiomyocytes, Paul Riley and colleagues report. Evidence suggests that these progenitor cells might be derived from the epicardium — the outer layer of heart tissue. However, the researchers can not entirely rule out a contribution from a non-epicardial source.

The new cardiomyocytes structurally and functionally integrate with resident muscle and could potentially replace damaged muscle following a heart attack. Stimulation of a progenitor pool as shown by Riley and coworkers could prove valuable in the development of resident-cell-based therapy in human ischaemic heart disease.

What was the strategy?

- British Heart Foundation made online video
- Broadcasters assisted with patient interviews/filming in the lab
- Press briefing in London
- Two authors talk at Science Festival as paper published
- Tweets
- Press release
- Facebook group posting and responding to comments

CIENCIA

Corazones reparados por células madre

Logran regenerar tejido cardiaco dañado tras un infarto en un experimento con ratones

ANGELES LOPEZ / Madrid Desde que se sabe que existen célu-las madre en diferentes partes del carepo humano, los especialistas en cardiología han desartollado numerosos estudios con dos objetivos. El primero, demostrar que también en el coranón existen esas celulas, capaces de convertirse en cualquier otro tipo celular. Por otro lado, también buscan una forma de utilizarlas como terapia para regenerar el tejido cardiaco tras un infarto.

En definitiva, se persigue un método eficar y seguro que transforme esas celulas primarias en nuevos componentes del músculo cardiaco para restablecer la función de la zona infartada. Ahora un trabajo, desurrollado por investigadores del University College London (Reino Unido) y publicado on la revista Nature, parece haber dado en el clavo de esa búsqueda. Aunque de momento los resultados sólo se han probado en catones, por lo que habra our esperar años para ver su anno ann freatmannos

Estos científicos se centraron en las critulas madre del epicardio, que se encuentran en la membrana externa que recubre al corazón. En el

Se 'fabricó' nuevo núsculo cardiaco n células de los mios animales

en som camaces de transforinteres que cuando nos were and addition by no pue-

E La 'fabricación' de nuevo músculo cardiaco

2

EL INFARTO DE MIOCARDIO

E EL EXPERIMENTO

Varios días antes

riesgo de sutrirlo.

de sufrir un infarto,

dosis de la molécula

tirosina beta 4 (T B4)

en ratones sanos con

. Tirosina

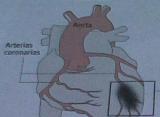
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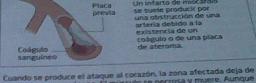
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FUENTE NUTURE

beta 4

invectaron varias





recibir sangre y oxigeno. El músculo se necrosa y muere. Aunque el corazón sigue funcionando, esa zona no se mueve y trabaja peor.

Area infartada

Un infarto de miocardio

La molécula T B4 activó las células progenitoras del epicardio (membrana externa del corazón), que empezaron a producir células adultas del miocardio (músculo del corazón). Esas células se integraron en la zona dañada.

Membranas del corazón

Epicardio Miocardio Células de miocardio

EL MUNDO

molécula llamada timosina beta 4 (TB4) en corazones sanos de ratones adultos y luego volvieron a inocular varias dosis de recuerdo en los ratones que sufrieron un infarto. Esta sustancia desencadenó la activación de las células madre del epilo cardiaco y a integrarse con el resto de compañeras sanas.

«Nuestras investigaciones anteriores probaron que era posible regenerar vasos sanguíneos en corazones adultos, pero teníamos dudas de si ocurriria lo mismo en el ra reparar corazones dañados por un infarto y podria tener un mayor impacto en futuras terapias orientadas a tratar la insuficiencia cardiaca», explica el catedrático Paul Riley, del Instituto de Salud Infantil del UCL y principal autor del es-

Para Francisco Fernández Avi-Ma, Jefe del Servicio de Cardiología del Hospital Gregorio Marahon da Madrid, este estudio «confirma que en el corazón adulto existen céluias madre que pueden activar-se y regenerar el tejido destruido. Estos resultados abren una espe ranza a, en el futuro, poder utilizar moléculas activadoras que, admi-nistradas a pacientes de alto riesgo, puedan evitar o atenuar la des trucción de tejido cardiaco en caso de infarton

No obstante, a pesar de los excelentes resultados, todavia queda mucho trabajo por delante. Aunqui la molécula TB4 permitió la forma ción de células adultas del múscu cardiaco, su número fue limita Por este motivo, los investigado planean seguir estudiando el m nismo por el que funciona esta tancia para desarrollar un m más eficaz de transformar la las madre en células especia v tener una alternativa que da trasladar a los humanos

A esas limitaciones hay los posibles riesgos. «Si e cula es capaz de transf células madre en otras

Futuros traba deben descar riesgo de qu generen tun

