NEUROBIOLOGÍA | Técnicas de imagen

La arquitectura del cerebro

Imágenes obtenidas de las fibras neuronales del cerebro. | Science

- Dos estudios mejoran el conocimiento sobre el tejido neuronal y su función
- La información genética condiciona la estructura de la corteza cerebral

Ángeles López | Madrid
Actualizado lunes 09/04/2012 08:00 horas

Un damero de ajedrez más que un enmarañado ovillo. Así podría resumirse la forma en que las fibras nerviosas se organizan en el cerebro y así es como las podemos ver en las fotografías que un grupo de expertos del Hospital General de Massachusetts (EEUU) ha obtenido con diferentes técnicas de imagen. Su trabajo, junto con el de otro grupo de la Universidad de California que ha analizado cómo la información genética contribuye en la conformación de la corteza cerebral, nos permite conocer mejor la estructura de ese órgano que todavía sigue siendo un gran desconocido.

Aunque se conoce que en la médula espinal y en el tronco del encéfalo, las fibras nerviosas se organizan formando fascículos paralelos derivados de cómo se organiza el sistema nervioso en el desarrollo embrionario, se desconoce cómo es ese patrón de haces en el lóbulo frontal y otras regiones corticales. Hasta ahora sólo se había podido estudiar pequeñas fracciones de esas 'carreteras' neuronales en cerebros simples, pero no de humanos. A través de un nuevo escáner de imagen de resonancia magnética de difusión se puede ahora visualizar la distribución de esas fibras en todas las zonas del tejido cerebral.

"Hemos trabajado durante años en esta tecnología para establecer un

Imagen del cerebro de un mono que se asemeja a una rejilla de cables eléctricos. 'Science'

mapa de la arquitectura de las fibras del cerebro [...] Cada secuencia de fibras se conforma como una rejilla tridimensional. Esas fibras, en la parte superior del cerebro, son como un grupo de cables organizados en hojas dobladas, de tal manera que las fibras sólo van en las dos direcciones de las hojas y en otra perpendicular a ellas. Por lo tanto, todas las conexiones del cerebro siguen estas tres direcciones. En la vida embriológica seguir estas direcciones es más simple, pero el cerebro adulto tiene muchos pliegues, con lo que las tres direcciones terminan haciendo curvas", explica Van Weeden, principal investigador del estudio, publicado en la revista 'Science'.

Un orden establecido

"Los libros clásicos nos dan la idea de que las fibras nerviosas carecen de orden en su recorrido, formando un aparente ovillo de fibras entremezcladas, pero en el cerebro hay tanta fibra que por fuerza tiene que haber un orden, para que no haya un caos. Es un principio esperable, si queremos que los diversos cerebros se comporten parecidamente. No vemos fibras que crucen regiones en oblícuo, igual que un coche no atraviesa los edificios, sino que sigue un orden establecido por reglas de tráfico y la estructura de las calles. Lo que pasa en el cerebro es que las calles y casas están deformadas, porque hay muchos pliegues debidos a crecimiento desigual (como en un cuadro de Dalí), pero las fibras se apañan para caminar por esas calles de manera ordenada, aunque tengan un recorrido deformado. Esto es lo que acaban de demostrar estos científicos para la zona del cerebro donde menos se esperaba que fuera así", explica Luis Puelles, del departamento de Anatomía Humana y Psicobiología de la Universidad de Murcia.

Gracias a esta tecnología, se puede conocer cómo crece el cerebro. "Las conexiones del cerebro maduro parecen reflejar los tres principales ejes establecidos en el desarrollo embrionario", afirma este investigador. Otro dato que han podido comprobar en este trabajo es que las fibras nerviosas no están aisladas sino que se relacionan entre sí. La tercera conclusión que podría extraerse de la investigación, en la que han analizado tejido cerebral de cuatro tipos de monos y de personas vivas, es que esta estructura fibrilar se mantiene entre las especies, es decir, que los cerebros más simples y los más complicados conservan una estructura similar. "En definitiva, ahora tenemos una manera de ver la conectividad del cerebro como un todo, no como áreas aisladas", señala Weeden.

Tal y como explica el investigador español Alberto Ferrús, del departamento de Neurobiología Molecular, Celular y del Desarrollo del Instituto Cajal del Consejo Superior de Investigaciones Científicas (CSIC), "al estudiar esas vías [de fibras] se ve una organización ortogonal [en ángulo recto], esta organización permite decir que la parte más compleja (cortical) del cerebro sigue el mismo patrón de crecimiento que cualquier otra parte del sistema nervioso. Hasta hace poco se pensaba que la corteza era una estructura desarrollada súbitamente en los mamíferos, siendo inicialmente de estructura homogénea. Se hacían deducciones singulares como que 'la corteza es lo que nos hace humanos' [ya que la mente emerge de su función]. Estos estudios demuestran que la estructura de la corteza en primates y el hombre no es tan homogénea sino que está organizada en módulos, cada uno con una pauta de conectividad especializada, y que es comparable no obstante con la de otras especies".

Genes y neuronas

El segundo trabajo que publica la revista 'Science' va en la misma línea de intentar conocer mejor la estructura del cerebro humano. En este caso, investigadores de la Universidad de California, utilizando un mapeo masivo de la información genética característica de distintas partes de la corteza cerebral, con datos obtenidos mediante una poderosa técnica de resonancia magnética en 406 pares de gemelos adultos (para reducir la variabilidad), han completado un novedoso atlas génico de la corteza cerebral, la capa más superficial del cerebro.

Al comparar gemelos monocigotos con gemelos dicigotos, los autores observaron cómo la influencia genética se comparte separadamente en grupos de áreas que conforman 12 regiones de la corteza. Estas 12 regiones coinciden a grandes rasgos con sectores con una función ya definida en estudios previos.

Comprobaron, por tanto, que la estructura regional funcionalmente especializada de la corteza cerebral está genéticamente controlada y se organiza de una manera jerárquica (varias subáreas forman un área, y varias áreas forman una región).

"Si podemos comprender la base genética del cerebro, podemos tener una mejor idea de como se desarrolla y funciona, información que, a la larga, podemos utilizar para mejorar tratamientos de múltiples enfermedades", concluye Chi-Hua Chen, principal autor de la investigación.

Puelles aclara que este trabajo es "la primera evidencia, de una forma fidedigna, de que también para construir la parte más compleja del cerebro se toma información del genoma. Probablemente estarán implicados miles de genes. Es interesante que haya un orden genético, y, aunque es hermoso verlo confirmado, no nos sorprende, porque sería difícil que fuera de otra manera. El cerebro es lo más ordenado que conocemos. Lo más sorprendente es hasta qué punto encaja bien este análisis con lo que ya se iba sabiendo de funciones diferenciadas en la corteza. Los grupos de áreas cerebrales determinadas en este estudio se agrupan por funciones que resultan predeterminadas por la estructura génica. Los estudios anatómicos y funcionales previos ya habían adelantado particiones similares, pero este nuevo enfoque ha llegado a parecidos resultados desde un ángulo totalmente nuevo. Esto indica que diversos enfoques científicos están llegando a un consenso, lo cual es todo un hito histórico".

Repercusión de los resultados

Los dos trabajos que publica 'Science' indican, según Puelles, que "estamos empezando a comprender el cerebro, a ver 'las piezas de la máquina' que dependen de los genes, hemos llegado a un dintel de conocimiento a partir del cual podemos empezar a profundizar. Evidentemente, los genes no lo controlan todo al detalle (no hay suficientes, teniendo en cuenta que se calculan 10.000.000.000.000.000 sinapsis -o contactos entre neuronas- y sólo hay unos 20.000 genes), por lo cual, más allá de las 'piezas' en cuestión, hasta llegar a la mente, se intercala la microestructura que creamos individualmente al ir viviendo (a este nivel esperaremos diferencias entre los individuos) Por otra parte, sabiendo qué genes son los que predominan en las diversas áreas cerebrales podemos estudiar algunas enfermedades cerebrales de causa genética con mayor detalle".

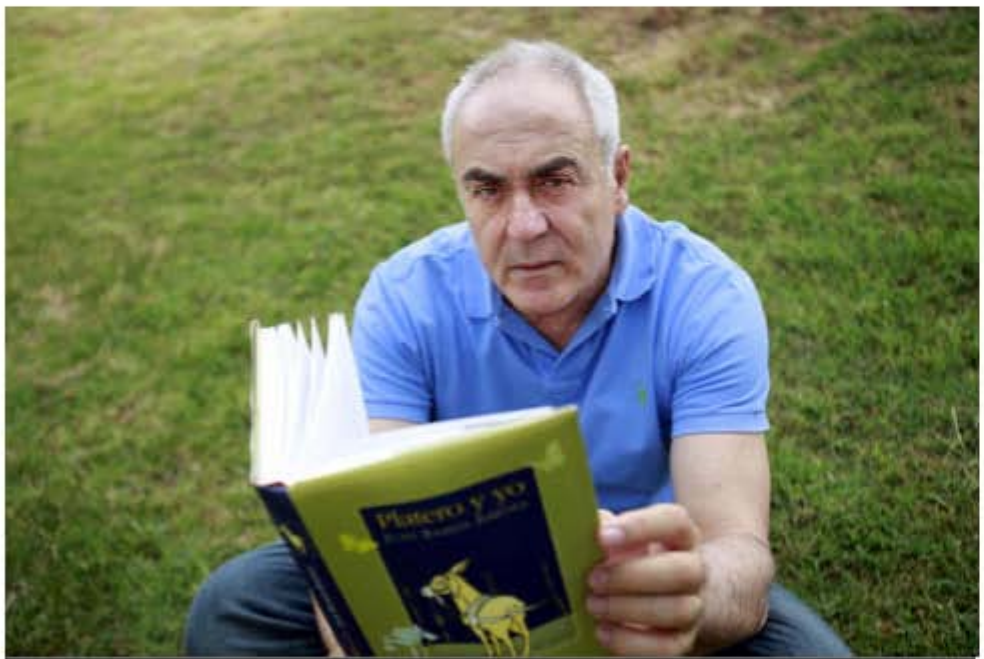
Sin embargo, no opina lo mismo Javier de Felipe Oroquieta, del Laboratorio Caja de Circuitos Corticales del Centro de Tecnología Biomédica de la Universidad Politécnica de Madrid, quien recuerda que ha habido mucha disparidad en los mapas citoarquitectónicos que han sido elaborados por diferentes investigadores. "Me parecen interesantes estos trabajos pero es difícil de entender el significado de esas diferencias. Cuando uno estudia el cerebro hay muchos niveles de análisis, éste es un nivel macroscópico, pero yo propongo el ultramicroscópico, con el que se pretende conocer el mapa de las sinapsis o sinaptoma", señala este experto.

No obstante, este investigador reconoce que el trabajo de Weeden, aun

siendo grosero, es al mismo tiempo maravilloso. De momento lo que se ha podido mostrar es la disposición conjunta de muchas fibras nerviosas, como si viéramos la carretera que une Barcelona con Madrid junto a otras que se cruzan con ella, pero nada más. Necesitamos saber más de lo que pasa entre medias, de las neuronas, de los circuitos, de la sinapsis...".

En la misma línea se expresa Ferrús: "Incluso cuando queremos colocar una función en una estructura todavía no sabemos cuáles son las unidades de esa función. Necesitamos un código para codificar, pero no sabemos ni con qué, ni cómo hacerlo. Estamos muy por detrás de los bioquímicos de los años 50 tras el descubrimiento de la estructura del ADN".

Más optimista se muestra Puelles quien señala que, a pesar de que "estos dos estudios no son rompedores, sí son aclaradores y confirmadores de que se estaba pensando en la dirección adecuada. Ahora podemos plantear nuevas ideas y experimentos más productivos. Es como cubrir una etapa".



De Felipe recomienda 'Platero y yo': "Me ha hecho reflexionar sobre la evolución del cerebro y el gran enigma de los otros mundos mentales, los de los otros animales". Imagen: Olmo Calvo | SINC

Javier de Felipe: "Platero y yo' representa una fuente inagotable de inspiración científica"

Fuente: SINC
Autor: SINC
Localización: España
Fecha: 29 junio 2012 14:20
Categoría SINC: Humanidades

Con Javier de Felipe (Madrid, 1953) estrenamos la nueva serie de reseñas de libros escritas por investigadores para SINC. El neurobiólogo recomienda *Platero y yo*, de Juan Ramón Jiménez: "Aunque no es un libro científico, trata sobre la relación del hombre con un animal. Este libro me ha servido de inspiración para meditar sobre los diversos mundos mentales". De Felipe es codirector de Cajal Blue Brain, un gran proyecto para simular el cerebro de los mamíferos por ordenador.

Descargar

Javier de Felipe: "Seguro que colonizaremos el espacio"

El eminente neurobiólogo investiga la neocorteza cerebral, donde se halla lo que nos hace ser humanos

Vida | 05/07/2012 - 00:23h

Javier de Felipe durante su visita al Museu Blau de Barcelona R.Q

RAQUEL QUELART | Sigue a este autor en **Twitter** o **Google +**

Somos nuestro cerebro, pero sin embargo conocemos tan poco de él… La necesidad de saber más sobre este órgano y, por lo tanto, de conocernos mejor a nosotros mismos, ha llevado a centenares de expertos de todo el mundo a centrar sus investigaciones en esta parte del cuerpo humano. Este es el caso del neurocientífico Javier de Felipe (Madrid, 1953), especialista en el estudio microanatómico del cerebro, profesor de investigación en el Instituto Cajal (CSIC) y uno de los mayores expertos en la investigación de la corteza cerebral. De Felipe ha colaborado con la NASA en el proyecto NEUROLAB (1998) y, actualmente, forma parte del proyecto Cajal Blue Brain, una iniciativa que desde 2002 pretende aunar los esfuerzos de investigadores de todo el mundo con el fin de entender la estructura cerebral. Los resultados podrían aportar soluciones para enfermedades como el alzheimer y la esquizofrenia.

El neurocientífico ha visitado recientemente Barcelona en el marco de una conferencia -"Sobre la belleza, el arte y la evolución del cerebro"- que tuvo lugar en el Museu Blau y que se enmarca en el conjunto de actividades que se celebran con motivo del año de la neurociencia.

- ¿Qué objetivo persigue el estudio Cajal Blue Brain?

- Es el estudio de las columnas corticales, que es la estructura elemental del funcionamiento de la corteza cerebral, la región del cerebro más relacionada con lo que nos hace ser humanos. El problema es que no sabemos cómo se estructuran las columnas ni cuántas células ni conexiones tienen. Disponemos de una información muy incompleta.

-¿En qué se diferencia de otros proyectos?

- Es el primer intento exhaustivo de hacer ingeniería inversa del cerebro, es decir, desmontarlo

LO MÁS »

LO MÁS VISTO

1 Catalunya pide prestados 9.073 millones de euros al Fondo de Liquidez en 2013

2 Un asteroide de 45 metros 'rozará' la Tierra el próximo 15 de febrero

3 La gran ola de Nazaré

4 Una profesora de matemáticas crea polémica tuiteando fotos desnuda

5 "El 90% de los suicidios tiene un trasfondo o causa laboral"

LO MÁS COMENTADO

Ir a Lo más

para observar sus partes. El cerebro es una máquina muy compleja formada por miles de millones de conexiones y tenemos que trazar el mapa, ver cómo se han creado estos circuitos y, luego, recrear estos procesos mentales en un ordenador.

- ¿Por qué el estudio es interdisciplinar?
- El problema que tenemos en neurociencia es que cada laboratorio trabaja con una pequeña porción de información, pero muchas veces no la pueden usar otros científicos. Con el Cajal Blue Brain nos juntamos muchos expertos de diversos campos, desde ingenieros a informáticos, para avanzar más rápido. Analizamos la estructura con un detalle altísimo, y mediante programas de computación transformamos la información biológica en datos matemáticos y realizamos simulaciones. Hemos conseguido crear neuronas virtuales y queremos hacer lo mismo con las columnas, lo que nos permitirá llevar a cabo miles de experimentos en segundos.

- ¿Se podrían aplicar estos conocimientos al ámbito de la robótica?
- Sí, claro. Nuestro cerebro tiene una capacidad enorme de procesar información, de hacer tareas muy complejas de manera simplificada, utilizando atajos, que debemos averiguar cómo son. Hay que imitar el cerebro para aprender cómo resuelve los problemas. Por ejemplo, a un ordenador le puedes enseñar a multiplicar y lo puede hacer más rápido que un cerebro humano, pero no es capaz de reconocer caras de manera instantánea; el cerebro utiliza 12 watios para procesar una información, mientras que una supercomputadora necesita millones de watios.

- ¿Qué pueden aportarnos estos conocimientos?
- Si queremos conocernos a nosotros mismos, debemos conocer cómo es nuestro cerebro, cómo funciona, cómo ha sido diseñado. Es curioso que esta idea filosófica-científica no esté arraigada en la sociedad, ya que se suele separar cerebro y mente.

- ¿La complejidad de la neocorteza nos diferencia de otras especies?
- No, no es la complejidad ni el tamaño del cerebro.

- Entonces, ¿qué nos hace ser distintos?
- A pesar de estar en el siglo XXI, es un misterio que todavía no ha sido resuelto, no sabemos lo que nos hace distintos de otras especies. Una gran parte de los científicos piensa que la diferencia radica en la complejidad, en el mayor número de columnas. No somos muchos los que defendemos lo contrario, es decir, que lo que nos hace ser diferentes es la estructura, que hay células que son distintas, que cada especie animal tiene un cerebro propio.

- Sin embargo, la mayoría de los experimentos se realiza con animales.
- Lo que se estudia en la rata se extrapola al cerebro humano y eso se emplea para modelos de alzehimer, esquizofrenia, autismo y muchas otras enfermedades. Es un problema porque resulta que, aunque hay cosas idénticas entre el hombre y el ratón -como las estructuras de la neocorteza-, existen muchas diferencias y eso no se acepta fácilmente.

- ¿A qué animal nos parecemos más?
- Al chimpancé, con el que compartimos un antepasado común hace seis millones de años. Pero, ¿cuánta información de un animal de experimentación puedes extrapolar al cerebro humano? Yo creo que es relativamente poca.

- ¿Somos superiores al resto de animales?
- Simplemente somos distintos, pero tenemos una capacidad de abstracción superior a la del resto de especies. En otro aspecto, como el olfativo, nos dominan claramente los perros, que también tienen un cerebro muy complejo y se deprimen y sufren. Por ejemplo, otros animales son buenos detectando sonidos, otros con los colores…

- La creatividad emana de nuestra neocorteza cerebral. ¿Por qué el hombre es capaz de crear arte?
- Es un gran misterio, es interesante porque la estética no es necesaria para la supervivencia, pero sin embargo nos produce un placer intelectual increíble y nos hace sentir mejor por razones desconocidas. Sabemos que se activan las zonas del cerebro que están relacionadas con los circuitos de recompensa, al igual que ocurre cuando tomamos drogas. Somos el único animal capaz de modificar las cosas para crear belleza, música, pintar… Una reflexión: durante miles y miles de años el hombre ha sido incapaz de crear arte, ya que este ha surgido relativamente hace poco tiempo.

- ¿Es quizá porque nuestro cerebro ha evolucionado?
- No, el cerebro es el mismo desde que apareció el homo sapiens, hace 200.000 años. Los primeros indicios de capacidad de abstracción se han encontrado en Sudáfrica, datan de hace 100.000 años y son unas incisiones geométricas que ya indican un pensamiento simbólico; en Austria se ha descubierto una escultura de 35.000 años de antigüedad, un hombre con cabeza de león, que es lo más antiguo que se conoce de creatividad artística; incluso, hay un estudio

AL MINUTO »

16:02 Montserrat Caballé anula su concierto del 23 de febrero en Granollers por...

16:00 El Barça, favorito en el clásico para las casas de apuestas

15:51 El 'Monstruo de las Galletas' reivindica el robo del emblema de Bahlsen

15:50 Navarro pide a Mas celebrar la cumbre anticrisis antes de los presupuestos

15:48 Merkel reclama ante Morsi respeto a los derechos humanos y diálogo

Ir al minuto

que demuestra que algunas de las pinturas de las cuevas de Altamira tienen 41.000 años.

- ¿Cuál es la conclusión?
- ¡Hemos perdido 160.000 años de creatividad, antes no ha habido nada!

- ¿Alguna explicación?
- La evolución ha sido cultural, no biológica. Un recién nacido en nuestra sociedad enseguida aprende a hablar y leer, pero si naciera en otro sitio donde no hubiera cultura, sería incapaz de hablar e, incluso, podría tener un retraso mental. Nuestro cerebro nace con unas capacidades enormes que son desconocidas, todavía no sabemos adónde vamos a llegar biológicamente.

- Entonces, es la capacidad de abstracción lo que marca la diferencia del ser humano.
- Una expresión tan habitual como "vuelvo mañana" es un pensamiento muy complejo, y que yo te lo diga a través de un sonido y que tú lo entiendas es impresionante. Es una adquisición humana muy tardía. Si un homo sapiens de hace 200.000 años naciera hoy, sería como nosotros, y al contrario, Einstein, Goya, Picasso y Dalí nunca hubieran sido lo que fueron de haber nacido en la época en que no existía el esqueleto intelectual. Nuestro cerebro es una máquina que se autorepara y aprende de sí misma, es como una esponja del entorno.

- Usted se muestra convencido de que el hombre colonizará el espacio, ¿el cerebro cambiará?
- Si eso ocurriera, el ser humano se convertiría en una especie nueva. En un experimento que hicimos con la NASA observamos cambios en los circuitos de la neocorteza de animales que viajaron al espacio durante su proceso de maduración cerebral. Esos cambios permanecieron en los animales que regresaron a la Tierra.

- ¿Esto qué significa?
- Nuestro cerebro ha evolucionado en este planeta, somos terrícolas, si pasamos a otro entorno, con el tiempo desarrollaremos otras habilidades distintas. Los viajes espaciales los vemos todavía como ciencia ficción, pero el desarrollo tecnológico de estos últimos 100 años es espectacular, por lo que no sabemos dónde vamos a estar dentro de medio mileno. Seguro que colonizaremos el espacio. Pero, claro, si vas a Marte o más allá no es para volver, no vale la pena pasar media vida en una nave.

- Claro.
- Los que nazcan en otro planeta tendrán su colonia de individuos que puede que se comuniquen con la Tierra, o no. Nuestro cerebro se adaptará o a lo mejor no podrá hacerlo y esos individuos acabarán desapareciendo. Pero no sería sorprendente que al estar en un entorno distinto al que fuimos creados hace miles de años, se produzcan cambios y mutaciones y al final nos convirtamos en una especie distinta.

- ¿Qué es lo más increíble del cerebro para usted?
- Que el cerebro soy yo, que una materia me hace sentirme ser yo. Fíjate que nosotros hemos surgido de la materia y que a lo largo de la evolución del tiempo se han ido formando estructuras hasta llegar al cerebro humano.

Scooped by sonia ramos

Alzheimer's disease i
alleviated: Promising
approach for humans

From www.scienceda

“ ScienceDaily (N
typical of Alzheime
in mice by blockad
research team fro
and the University of Zurich has just pu
in fighting Alzheimer's disease in the c
approach promises potential in prevent
disease has already set in.”

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Los pacientes con es
presentan alteracione
sistemas inmune e in

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Una investigación
Investigaciones Ci
Universitario Marq
los pacientes con
en la expresión de
inflamatorio respe
publicado en la re
lista de 200 genes asociados a la enfe
test de diagnóstico pre sintomático.
FUENTE | CSIC 22/11/2012

La esquizofrenia, una enfermedad men
consecuencias, tiene una incidencia de
estimado en el 80%. Con el fin de cara
información funcional sobre las bases
investigadores secuenciaron el product
en sangre de 32 pacientes de esquizof
comparación de los perfiles de expresi
implicados en siete procesos biológicos
respuesta inmune innata, la respuesta
respuesta a las heridas.
"Pese al reducido número de individuo
modelo de desarrollo neurológico de la

Jornada Formativa “COMUNICAR LA NEUROCIENCIA”

From www.aecomunicacioncientifica.org - August 28, 2012 5:10 PM

MARTES 21 DE AGOSTO DE 2012 13:16
En el Año de la Neurociencia en España, la Asociación Española de Comunicación Científica organiza ‘Comunicar la neurociencia’, una jornada formativa dirigida a profesionales del periodismo científico y la divulgación de toda España, así como a científicos interesados en comunicarse con la sociedad.
Fechas
3 de octubre de 2012
Lugar
Aula magna del edificio Paraninfo de la Universidad de Zaragoza. Plaza Basilio Paraíso nº 4 50005 Zaragoza.
Organiza
Asociación Española de Comunicación Científica (AECC).
Colaboran
Universidad de Zaragoza, Sociedad Española de Neurociencia (SENC) y Fundación Ibercivis.
Contenidos (programa provisional)
09:00 | Recepción y acreditación de asistentes.
09:30 | Presentación: Antonio Calvo Roy (presidente de la AECC) y Manuel J. López Pérez (rector de la Universidad de Zaragoza).
10:00 – 14:00 | Diálogos del cerebro
Actividad en formato de conversaciones acerca de la neurociencia y los problemas y retos que plantea a la hora de comunicarla. Protagonizados por 6 prestigiosos científicos y 6 periodistas. Cada “pareja” cuenta con 20-25 minutos.
Alberto Ferrús (Instituto Cajal, CSIC) - Pilar Perla (Tercer Milenio, Heraldo de Aragón)
Javier de Felipe (Instituto Cajal, CSIC) - Gonzalo Casino (periodista científico)
Francisco Mora (Universidad Complutense de Madrid/Universidad de Iowa)– José Pardina (director de Muy Interesante)
Luis M. Martínez Otero (Instituto de Neurociencias de Alicante) – Malen Ruíz de Elvira (El País)
Ignacio Morgado (Instituto de Neurociencia de la Universidad Autónoma de Barcelona) – Lorena Sánchez (redactora jefe de QUO)
Javier Mínguez (Universidad de Zaragoza)– Mónica Salomone (periodista científica).
* Nota: Habrá un descanso a media mañana en la sesión “Diálogos del cerebro” para tomar café, seguido de una dinámica de 10 minutos sobre hemisferios cerebrales.
DESCANSO PARA LA COMIDA
16:00 – 17:30 | Taller “Poniendo en práctica la divulgación de la neurociencia en multiformato” (Pilar Perla y Elena Sanz, con puesta en común del trabajo de los asistentes)
17:30 – 19:45 | Casos de neurodivulgación
“El mal del cerebro”, documental de Antonio Martínez Ron (La información)
"Making-off de la neurona de 15 metros para STAS, Alicante (2012)".
Ricardo Domínguez. El Caleidoscopio, spin-off de la Universidad Miguel Hernández.
Programa “Dona tu Neurona” en los Museos Científicos Coruñeses.
Francisco Armesto.
"Ciencia ciudadana también para la neurociencia". Fermín Serrano. Fundación ibercivis. Instituto de Biocomputación y Física de Sistemas Complejos.
Minidocumental “Envejecer con lucidez. Construyendo reserva cognitiva”, dirigido por María Teresa Fernández Turrado, del Departamento de

durante las dos últimas décadas. El mo
sería consecuencia de una exposición
que producirían una vulnerabilidad inm
vulnerabilidad se pondrá de manifiesto
los individuos afectados serían más su
problemas inmunológicos que contribui
investigador del CSIC Jesús Sainz, de
Biotecnología de Cantabria, centro mix
Cantabria.
DIAGNÓSTICO PRECOZ
Según sus autores, los resultados de e
pruebas de diagnóstico antes de que e
enfermedad mediante el empleo del pe
Además, añade el investigador del CS
los procesos moleculares que causan l
de nuevos fármacos y el reposicionam
actualidad".
J. Sainz, I. Mata, J. Barrera, R. Perez-
Rodríguez, B. Crespo-Facorro. Inflamm
have significantly altered expression in
DOI: 10.1038/mp.2012.165.

Fuente: http://www.madrimasd.org/infor
id=54944&origen=RSS

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Rescooped by sonia ramos fr

Cyberbullying only ra
sole factor identified i
suicides

From www.sciencedaily.com - October 22, 2012 5:20 PM

“ Cyberbullying – the use of the Internet, phones or other technologies to repeatedly harass or mistreat peers – is often linked with teen suicide in media reports. However, new research shows that the reality is more complex. ”

Via Dimitris Agorastos

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Psicología y Sociología de la Universidad de Zaragoza. Fruto del Taller de Guión y Producción del Documental Científico organizado por la Unidad de Cultura Científica de la Universidad de Zaragoza.
"Una neurobióloga divulgando". Mara Dierssen. Centro de Regulación Genómica de Barcelona, presidenta de la Sociedad Española de Neurociencia.
"Dona tu risa a la ciencia". Raquel del Moral, investigadora del Estudio neurocomputacional de la risa: aplicación a nuevas tecnologías de diagnóstico psiquiátrico. Instituto Aragonés de Ciencias de la Salud.
"La neurociencia, a escena con los títeres de la Tía Elena”. Adolfo Ayuso, autor de la obra “Cajal, el rey de los nervios".
20:00 | Teatro científico "Protón. La fascinante historia de una partícula inmortal... o casi", del neurocientífico y divulgador Xurxo Mariño (abierto al público general).
Inscripción
Los interesados en inscribirse pueden hacerlo mandando un correo con su nombre, apellidos, DNI y profesión a cerebro@aecomunicacioncientifica.org , indicando en el asunto "Curso neurociencia". La secretaría le remitirá los datos para hacer efectivo el ingreso de la inscripción. Las plazas son limitadas.
El precio de inscripción es de 20 euros para socios de la AECC y para miembros de la comunidad universitaria de la Universidad de Zaragoza, y de 40 euros para el público general.
El coste de inscripción para los miembros de la SENC, de la ACCC y del Instituto Aragonés de Ciencias de la Salud será de 30 euros.

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comportamientos repetitivos o
a Efe Christos Gkogkas, autor principal
la relación entre la síntesis incorrecta
descubrieron por sorpresa que un
do en el desarrollo del autismo.El
científicos ir un paso más allá y
diante un compuesto diseñado en un
cáncer, que redujo los niveles de
padecen algún trastorno del espectro
dades que afecta a todo tipo de etnias
cuatro veces más frecuente entre

n/ciencia-y-
s-autistas-en-ratones-controlando-
fKixIbQ8mMctc6/

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From www.centrodeinnovacionbbva.com - October 22, 2012 9:04 AM

“ ¿Qué hace al hombre ser humano? El neurocientífico Javier DeFelipe nos planteará respuestas a esta compleja pregunta en torno a uno de los objetivos de la neurociencia: comprender los mecanismos biológicos responsables de la actividad mental humana.”

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Brain’s language center has multiple roles - MIT News Office

From web.mit.edu - October 16, 2012 12:21 PM

Neuroscientists Find the Molecular “When” and “Where” of Memory Formation

From www.nyu.edu - October 16, 2012 8:07 PM

“ RT @TapasDeCiencia: Los neurocientíficos encuentran el 'cuándo' y 'dónde' molecular de la formación de la memoria.**”**

October 15, 2012

Neuroscientists from New York University and the University of California, Irvine have isolated the “when” and “where” of molecular activity that occurs in the formation of short-, intermediate-, and long-term memories. Their findings, which appear in the journal the Proceedings of the National Academy of Sciences, offer new insights into the molecular architecture of memory formation and, with it, a better roadmap for developing therapeutic interventions for related afflictions.
“Our findings provide a deeper understanding of how memories are created,” explained the research team leader Thomas Carew, a professor in NYU’s Center for Neural Science and dean of NYU’s Faculty of Arts and Science. “Memory formation is not simply a matter of turning molecules on and off; rather, it results from a complex temporal and spatial relationship of molecular interaction and movement.”
Neuroscientists have previously uncovered different aspects of molecular signaling relevant to the formation of memories. But less understood is the spatial relationship between molecules and when they are active during this process.
To address this question, the researchers studied the neurons in Aplysia californica, the California sea slug. Aplysia is a model organism that is quite powerful for this type of research because its neurons are 10 to 50 times larger than those of higher organisms, such as vertebrates, and it possesses a relatively small network of neurons—characteristics that readily allow for the examination of molecular signaling during memory formation. Moreover, its coding mechanism for memories is highly conserved in evolution, and thus is similar to that of mammals, making it an appropriate model for understanding how this process works in humans.
The scientists focused their study on two molecules, MAPK and PKA, which earlier research has shown to be involved in many forms of memory and synaptic plasticity—that is, changes in the brain that occur after neuronal interaction. But less understood was how and where these molecules interacted.

The study’s other co-authors were Xiaojing Ye, a postdoctoral fellow in NYU’s Center for Neural Science, Andreea Marina, an undergraduate at UC Irvine at the time of the study. The research was conducted at NYU’s Center for Neural Science and UC Irvine’s Center for Neurobiology of Learning and Memory.
This work was supported by grants RO1 MH 041083 and RO1 MH 081151 from the National Institute of Mental Health, part of the National Institutes of Health, and a grant IOB-0444762 from the National Science Foundation.

Fuente: http://www.nyu.edu/about/news-publications/news/2012/10/15/neuroscientists-find-the-molecular-when-and-where-of-memory-formation.html

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Rescooped by sonia ramos from Social Neuroscience

La gente que nos disgusta hace

“ Neuroscientists find Broca’s area is really two subunits, each with its own function.**”**

Anne Trafton, MIT News Office

October 16, 2012
A century and a half ago, French physician Pierre Paul Broca found that patients with damage to part of the brain’s frontal lobe were unable to speak more than a few words. Later dubbed Broca’s area, this region is believed to be critical for speech production and some aspects of language comprehension.
However, in recent years neuroscientists have observed activity in Broca’s area when people perform cognitive tasks that have nothing to do with language, such as solving math problems or holding information in working memory. Those findings have stimulated debate over whether Broca’s area is specific to language or plays a more general role in cognition.
A new study from MIT may help resolve this longstanding question. The researchers, led by Nancy Kanwisher, the Walter A. Rosenblith Professor of Cognitive Neuroscience, found that Broca’s area actually consists of two distinct subunits. One of these focuses selectively on language processing, while the other is part of a brainwide network that appears to act as a central processing unit for general cognitive functions.
“I think we’ve shown pretty convincingly that there are two distinct bits that we should not be treating as a single region, and perhaps we shouldn’t even be talking about ‘Broca’s area’ because it’s not a functional unit,” says Evelina Fedorenko, a research scientist in Kanwisher’s lab and lead author of the new study, which recently appeared in the journal Current Biology.

Fuente: http://web.mit.edu/newsoffice/2012/brocas-area-multiple-roles-1016.html

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Scooped by sonia ramos

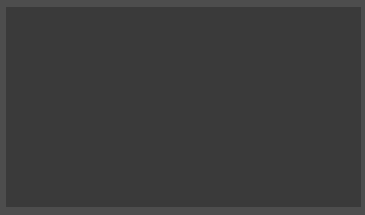
La neurocirugía del futuro llega a España

From www.lavozdigital.es - October 15, 2012 9:50 AM

La resonancia magnética en tiempo real para operar un tumor cerebral ya es una realidad gracias a una tecnología pionera que reduce el riesgo para los pacientes
28.09.12 - 03:03 - RUBÉN CAÑIZARES

La sanidad española y los pacientes que sufren algún tipo de tumor cerebral están de enhorabuena. El Hospital HM Universitario Sanchinarro es ya el segundo hospital a nivel nacional (cuarto en Europa y noveno en todo el mundo y el primero en la comunidad de Madrid) en ofrecer a sus pacientes el conjunto tecnológico de resonancia magnética intraoperatoria PoleStar N30 iMRI con navegador integrado StealthStation i7. ¿Cómo se traducen estos alambicados términos? En una innovación tecnológica que posibilita la realización de una resonancia magnética en tiempo real durante la intervención quirúrgica de un tumor cerebral que permite al neurocirujano localizar la lesión con una precisión milimétrica y, con ello, minimizar los riesgos para el paciente. Además, permite reducir el tiempo de hospitalización y la probabilidad de tener que ser sometido a una nueva intervención.

que nuestro cerebro funcione más lento



From actualidad.rt.com - October 16, 2012 9:29 AM

Nuestra simpatía o disgusto con alguien influye en cómo nuestro cerebro procesa sus acciones...

Via Benjamín Villasana

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The Changing Brain - B.S. Shankaranarayana Rao, National Institute of Mental Health and Neuro Sciences | Somerco

From www.somerco.com - October 8, 2012 1:31 PM

The Changing Brain - B.S. Shankaranarayana Rao, National Institute of Mental Health and Neuro Sciences
2012-05-04 (18:23)
Neural Plasticity and Brain Repair Mechanisms
B.S. Shankaranarayana Rao
Department of Neurophysiology
National Institute of Mental Health and Neuro Sciences
NIMHANS Deemed University
Until recently, the brain was considered unique in its lack of ability to repair itself once it had matured to adulthood. But recent evidences clearly demonstrated the ability of adult brain to modify its connections leading to recovery of functions by plasticity mechanisms. Neuronal plasticity is an extraordinary property of the brain and refers to the morphological, biochemical, behavioral and electrophysiological alterations in both the adult and developing nervous system. Thus, it is becoming increasingly clear that neuroplasticity contributes to the regenerative and continuing mechanism for adaptive reorganization of the brain. This unique property of the nervous system may be responsible for the recovery of functions in several brain disorders including stress and depression. Stress is a condition that seriously perturbs physiological and psychological homeostasis, resulting in disorders ranging from anxiety to post traumatic stress disorder. Severe traumatic or repeated stress can result in long-term deleterious effects leading to depression and cognitive deficits. Prolonged stress causes cognitive dysfunction associated with altered neurotransmission and formation of new neurons. On the other hand, brain stimulation rewarding and social enriched experiences results in formation of new connections, enhances neurotransmission and facilitates cognition by inducing progressive plasticity. Accordingly, the role of brain stimulation, social enrichment and pharmacological manipulation of transmitter systems in ameliorating stress and depression-induced cognitive dysfunctions was evaluated. We found reversal of stress and depression-induced cognitive impairment and associated brain deficits. These studies indicate that the rewiring and remodeling of neural circuits in the adult brain is possible because of plasticity at multiple levels of neural organization. Thus, understanding the mechanisms of plasticity will provide an insight in developing new therapeutic strategies in treating neurological and psychiatric disorders.

Fuente: http://www.somerco.com/file/11212

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El doctor Jorge Diamantopoulos, jefe del Servicio de Neurocirugía de HM Hospitales, explica lo que supone esta notable mejora tecnológica: «Esta nueva técnica nos va a ayudar a ser muchos más precisos. Hasta ahora, solo podíamos hacer uso de los sistemas de navegación que utilizábamos para guiarnos por el cerebro durante el preoperatorio. Este sistema tenía un pequeño inconveniente: cuando nosotros comenzábamos la intervención y abríamos el cerebro, como no es un órgano estático, corríamos el riesgo de que el paciente sufriera un ‘brain shift’, es decir, el desplazamiento de la masa cerebral. De este modo, existía la posibilidad de que las imágenes que nos marcaba el navegador del cerebro del paciente ya no fueran del todo correctas. Ahora, al hacer una resonancia magnética intraoperatoria (RNMi), sabemos siempre donde estamos localizados. Por ello, en todo momento, vamos a distinguir el tejido sano del tejido tumoral mediante la visión en tiempo real del tumor. Sabremos si quedan restos y si debemos seguir extirpando. Para tener una idea del avance que supone, con esta nueva técnica se disminuye el numero de reintervenciones entre un 30 y un 60%». En España, se diagnostican cada año 3.500 nuevos casos de tumores cerebrales primarios y entre 12.000 y 14.000 casos de metástasis cerebrales. Según los registros oficiales, un 25% de la población española tiene algún tipo de cáncer, y los tumores cerebrales tienen una incidencia del 7.5 por cada 100 mil habitantes, lo que supone el 2% de los cánceres en adulto y cerca de un 15% en los niños y jóvenes menores de 15 años. Este tipo de tumor afecta más a los hombres jóvenes y es la segunda causa de muerte entre niños de 0 a 5 años, después de la leucemia. De ahí la importancia de este avance tecnológico.

Noticia completa en la fuente: http://www.lavozdigital.es/cadiz/20120928/mas-actualidad/salud/investigacion/neurocirugia-futuro-llega-espana-201209280259.html

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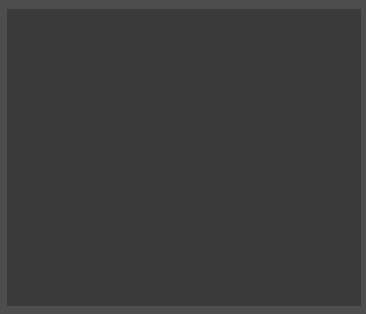
Rescooped by sonia ramos from Psychology and Brain News

Hearing brains are ‘deaf’ to disappearance of sounds, study reveals



From www.psypost.org - October 8, 2012 8:56 AM

“ Our brains are better at hearing new and approaching sounds than detecting when a sound disappears, according to a study published today funded by the Wellcome Trust.”

Via Dimitris Agorastos

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Descubierta la causa de que el estrés acabe en depresión / Noticias / SINC - Servicio de Información y

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Rescooped by sonia ramos from Diabetes Hoy

Relacionan el nivel sanguíneo de glucosa con la pérdida de volumen cerebral - Revista de Neurología

From www.revneurol.com - October 7, 2012 1:07 PM

Las personas con un nivel sanguíneo de glucosa en el extremo superior del rango normal podría tener un mayor riesgo de contracción cerebral, como ocurre con el envejecimiento y enfermedades como la demencia, según un estudio australiano.
En la investigación participaron 266 personas de 60 a 64 años, cognitivamente sanas y sin diabetes mellitus tipo 2, que tenían un nivel de azúcar en la sangre dentro del rango normal, según la definición de la Organización Mundial de la Salud. Los participantes se sometieron a resonancias magnéticas cerebrales al inicio del estudio y, de nuevo, cuatro años después.
Las personas con mayores niveles de glucosa en sangre dentro del intervalo normal eran más propensas a mostrar una pérdida de volumen cerebral en las áreas del hipocampo y la amígdala, áreas que están implicadas en la memoria y las habilidades cognitivas.
Tras controlar por edad, hipertensión arterial, tabaquismo, consumo de alcohol y otros factores, se observó que un nivel sanguíneo de azúcar en el rango alto de la normalidad influía en un 6-10% en la contracción del volumen cerebral.

Via C8 MediSensors ES

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Rescooped by sonia ramos from Records y curiosidades sobre la ciencia

Comunicar la Neurociencia, un evento con mucho cerebro

Noticias Científicas

From www.agenciasinc.es - October 2, 2012 8:54 AM

Un estudio de la Universidad de Washington describe el mecanismo molecular por el cual el estrés puede conducir a la depresión. El trabajo explica cómo un grado severo de este trastorno afecta a la liberación del neurotransmisor dopamina en el núcleo accumbens, una región del cerebro relacionada con la recompensa y el placer.

SINC | 19 septiembre 2012 19:00
ardiaca, aparece sudoración en las manos y la boca se seca.
Cuando este estrés se mantiene en el tiempo, la situación se vuelve crónica y puede desembocar, entre otras patologías, en una depresión.
Ahora, científicos de la Universidad de Washington (EE UU) han descubierto el mecanismo molecular que explica esta relación entre un estrés prolongado con los trastornos depresivos.
La investigación, publicada en la revista Nature, muestra como antes de llegar a una situación crónica, cuando el estrés es agudo o puntual, se produce la secreción del neurotransmisor CRH (hormona liberadora de corticotropina).
Esta molécula provoca la liberación de dopamina, comúnmente asociada con el sistema cerebral del placer.
Antes de llegar a una situación crónica, cuando el estrés es agudo o puntual, se produce la secreción del neurotransmisor CRH
Pero, tal y como señala a SINC Paul Phillips, autor principal del estudio, “tras el estrés prolongado, esta función reguladora de la CRH se pierde, y no se libera más dopamina, lo que puede conducir a la depresión. La función no vuelve a recuperarse en meses”.

Referencia bibliográfica:
Julia C. Lemos, Matthew J. Wanat, Jeffery S. Smith, Beverly A. S. Reyes, Nick G. Hollon, Elisabeth J. Van Bockstaele, Charles Chavkin, Paul E. M. Phillips “Severe stress switches CRF action in the nucleus accumbens from appetitive to aversive”. Nature. doi:10.1038/nature11436

Noticia completa en la fuente: http://www.agenciasinc.es/Noticias/Descubierta-la-causa-de-que-el-estres-acabe-en-depresion

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Rescooped by sonia ramos from Psychology and Brain News

Autistic adults have unreliable neural responses, research team finds

From www.sciencedaily.com - September 21, 2012 10:01 AM

“ New research by neuroscientists takes the first step toward deciphering the connection between general brain function and the emergent behavioral patterns in autism.”

Via Dimitris Agorastos

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From aecomunicacioncientifica.org - October 1, 2012 9:43 AM

Asociacion Española de Comunicacion Cientifica...

Via Isabel Etayo

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Describen un mecanismo que explicaría las resistencias al tratamiento farmacológico de la esquizofrenia - Genoma España

From www.gen-es.org - September 18, 2012 3:13 PM

“ Describen un mecanismo que explicaría las resistencias al tratamiento farmacológico de la esquizofrenia http://t.co/3bEIPnnG...”

Una investigación, en la que participa la Universidad del País Vasco (UPV/EHU), describe por qué algunos medicamentos para la esquizofrenia no son efectivos en uno de cada tres pacientes.
Un consorcio de grupos de investigación formado por la UPV/EHU, la Facultad de Medicina Mount Sinai de Nueva York, la Universidad de Arizona, el Instituto de Tecnología de Massachusetts y el Centro de Investigación Biomédica en Red (CIBER) de Salud Mental podría haber descubierto por qué ciertos fármacos utilizados en el tratamiento de la esquizofrenia no son efectivos en un tercio de los pacientes.
La investigación, publicada en septiembre por Nature Neuroscience, abre el camino al diseño y uso de nuevos medicamentos para esta enfermedad crónica que afecta al 1% de la población mundial.
Los investigadores intentaban hallar las alteraciones epigenéticas (alteraciones en los genes provocadas por factores externos) involucradas en la resistencia al tratamiento con antipsicóticos atípicos, el más usual en la esquizofrenia. Descubrieron que, con el paso del tiempo, en el cerebro de los esquizofrénicos se produce el incremento de la enzima HDAC2, y que

Rescooped by sonia ramos from All About Food

Brain diabetes: the ultimate food scare - opinion - 03 September 2012 - New Scientist

From www.newscientist.com - September 18, 2012 12:14 PM

“ Brain diabetes: the ultimate food scare”
03 September 2012
Magazine issue 2880. Subscribe and save
For similar stories, visit the Food and Drink and The Human Brain Topic Guides
Big trouble lies ahead if Alzheimer's is proven to be a form of diabetes

THE human brain evolved to seek out foods high in fat and sugar. But a preference that started out as a survival mechanism has, in our age of plenty, become a self-destructive compulsion.
It is well known that bad diets can trigger obesity and diabetes. There is growing evidence that they trigger Alzheimer's disease too, and some researchers now see it as just another form of diabetes (see "Food for thought: Eat your way to dementia").
If correct, this has enormous, and grave, implications. The world already faces an epidemic of diabetes. The prospect of a parallel epidemic of Alzheimer's is truly frightening, in terms of human suffering and monetary cost.
This outcome will not be easily averted. Few people need to be told that too much high-fat, high-sugar food is a health hazard. And yet sales of fast food remain healthy (or should that be hefty?). Part of the reason is "future discounting", another evolved feature of the human brain that makes us value short-term rewards over long-term risks.
What can be done? One option is to call in the lawyers. Some moderately successful attempts have already been made to sue food companies for their role in creating the obesity epidemic. If a causal link between fatty, sugary food and Alzheimer's can be established, it is highly likely that more lawsuits will follow. Such actions have their place, but this is a laborious and expensive way to enact change.

Read more: http://www.newscientist.com/article/mg21528801.100-brain-diabetes-the-ultimate-food-scare.html

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esa enzima sobreexpresada es la responsable de los cambios genéticos que provocan una menor eficacia de los fármacos.
Es decir, la propia medicación prescrita para tratar la esquizofrenia podría ser la responsable de la resistencia al tratamiento crónico.

Fuente: SINC
Tags: biomedicina, esquizofrenia, resistencia tratamiento, hdac2
Permalink: http://www.gen-es.org/?iid=describen-un-mecanismo-que-explicaria-las-resistencias-al-tratami&itid=1&lan=es

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Mendeley y la ciencia abierta en 2012. Más que un gestor de bibliografías | Infobiblio

From www.infobiblio.es - September 18, 2012 10:42 AM

Submitted by mladt on Mon, 09/17/2012 - 19:44 002 011 Ciencia abierta Redes científicas Redes sociales Software bibliográfico
El producto Mendeley es al mismo tiempo una red social y una buena herraienta bibliográfica para investigadores, con casi 2 millones de usuarios, más de 65 millones de referencias de artículos y recibe actualmente más 100 millones de consultas anuales. Tech Europe, un blog de The Wall Street Journal le dedicó una importante entrada en agosto pasado sobre el desarrollo de sus aplicaciones abiertas y su utilización por los investigadores: UK Start-up Opens Up Science Research.

Fuente: http://www.infobiblio.es/mendeley-y-la-ciencia-abierta-en-2012-mas-que-un-gestor-de-bibliografias

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Descubren los principios que crean conexiones entre las neuronas

FISICA Y QUIMICA PARA ESTUDIANTES: ¿Cómo funciona el DNA?

From fqruben.blogspot.fr - September 18, 2012 9:33 AM

lunes, 17 de septiembre de 2012

¿Cómo funciona el DNA?
Si pincháis aqui podréis ver un video de cómo se pliega el DNA dentro de las células y cómo se replica, formando una copia de sí mismo. Este video es más completo, e incluye la traduccion y la biosintesis de proteinas. Aqui más.
Con esto empiezo el nuevo curso, que promete ser largo y lleno de sorpresas.

Publicado por Ruben Martín en 7:16:00 p.m.

Etiquetas: 1ºBachFyQ, 2ºBachQuim, opinion

Fuente: http://fqruben.blogspot.fr/2012/09/como-funciona-el-dna.html

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El CSIC y U-tad colaborarán en el diseño de contenidos digitales - csic.es

From www.csic.es - September 13, 2012 10:03 AM

“ Firman un convenio para aplicar las neurociencias a este sector
El Consejo Superior de Investigaciones Científicas (CSIC) y U-tad, Centro Universitario de Tecnología y Arte Digital, han firmado un convenio de colaboración para aplicar el conocimiento sobre el cerebro y la conducta al desarrollo de productos y servicios digitales. Este trabajo conjunto permitirá nuevos avances en el conocimiento del cerebro que podrán aplicarse al desarrollo de productos y servicios con fines educativos, comerciales o sociales, desde aplicaciones para dispositivos móviles a videojuegos, entre

From www.abc.es - September 18, 2012 9:31 AM

“ Un equipo de científicos recreó por ordenador un microcircuito neuronal y lo comparó con mapas de experimentos reales, descubriendo que eran muy parecidos...**”**

EFE / MADRID
Día 17/09/2012 - 22.13h

Un grupo de científicos de la Escuela Politécnica Federal de Lausana (EPFL) ha conseguido dar un paso adelante hacia la comprensión de cómo funciona el cerebro, gracias a la identificación de los «principios clave» que determinan las conexiones que se producen entre las neuronas. «Lo que creemos es que cada neurona crece independiente de las demás y que, cuando todas han crecido, simplemente se producen colisiones accidentales entre ellas y se forman las sinapsis (la conexión entre dos neuronas)», explicó el director del proyecto Blue Brain, Henry Markram.
El equipo de Markram llegó a esta conclusión después de recrear en un ordenador un microcircuito neuronal compuesto por 10.000 células de este tipo (el cerebro humano se compone de unos 100.000 millones) y observar que las conexiones que predecía el modelo eran muy similares a las que se comprobaron en un circuito cerebral equivalente procedente de un mamífero real.
Según el director del proyecto, «la explicación de cómo conectar las neuronas era uno de los grandes retos. Sabemos cómo se tocan unas neuronas con otras y cómo forman las sinapsis, pero no sabíamos la regla general que siguen millones de neuronas». «Podría pensarse que una neurona, a través de químicos, es atraída y repelida hasta que encuentra a otra neurona y establece una conexión con ella, pero es mucho más simple», aseguró Markram, aludiendo así a la hipótesis de la quimioafinidad, que establece que estas células siguen unas señales químicas que les indican con qué otras conectarse.

Más información en la fuente original:
http://www.abc.es/20120917/ciencia/abci-principios-conexiones-neuronas-201209172201.html

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8 argumentos neurocientíficos para una educación mejor - El caparazon

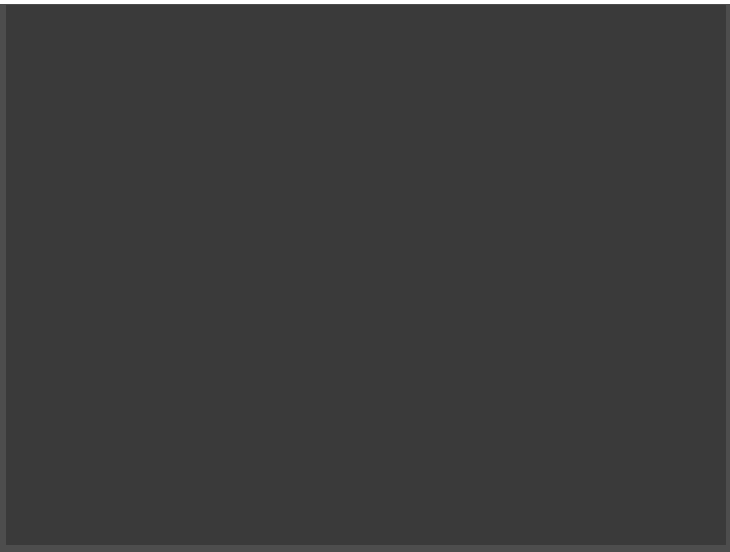
otros.**”**

“
El acuerdo impulsará la investigación y el desarrollo de las neurociencias en el ámbito digital a partir de cuatro ejes de actuación: el académico, la investigación, el desarrollo de productos y servicios, y la divulgación de conocimiento. También facilitará la formación de investigadores, y la difusión entre la comunidad científica de los hallazgos de estos trabajos.
”
El convenio permitirá al CSIC promover la participación en programas nacionales y europeos de financiación de la I+D+i, a los que U-tad contribuirá aportando recursos humanos y técnicos. Asimismo, los alumnos del centro universitario participarán en proyectos de I+D, y podrán cubrir necesidades de personal técnico, vinculado con el sector digital, que surjan en el Instituto de Neurociencias de Alicante, un centro mixto del CSIC y la Universidad Miguel Hernández de Elche.
“El Instituto de Neurociencias está encantado de intentar que el conocimiento que generamos en torno al funcionamiento del cerebro pueda ser integrado en la creación de contenidos de rápida proyección social, porque demuestra que la investigación básica puede ser eficientemente trasladable”, indica Juan Lerma, director del centro ubicado en Alicante.
Según el vicepresidente de Organización y Relaciones Institucionales del CSIC José Ramón Urquijo: “Este acuerdo pone de manifiesto el interés del CSIC por el establecimiento de cooperaciones con entidades privadas que refuercen sus objetivos”.
”

“
Dos ámbitos y un beneficio mutuo
”
Los contenidos digitales y las neurociencias tienen muchos puntos en común. El uso de simuladores, la telemedicina, el 3D, o la realidad virtual son desde hace años recursos habituales en el campo de la salud y la ciencia. Las neurociencias y el neuromárketing han dado pasos de gigante gracias al desarrollo de contenidos y herramientas digitales que pueden medir factores impensables hasta hace pocos años.
La industria de contenidos digitales puede utilizar los conocimientos de las neurociencias para incrementar la efectividad y el impacto de sus productos y servicios en la conducta, como sucede en la industria de los videojuegos.
“Con este acuerdo, U-tad y el CSIC van a impulsar el desarrollo de productos digitales basados en un conocimiento más científico del funcionamiento del cerebro. Las neurociencias utilizan la tecnología digital para avanzar en sus investigaciones, al igual que otros muchos sectores tan diversos como la aviación, el cine o la banca, lo que demuestra la importancia y transversalidad que tiene el estudio de los contenidos digitales. Tenemos numerosas líneas de trabajo comunes que recibirán un impulso considerable”, señala Ignacio Pérez Dolset, fundador de U-tad.
Los contenidos digitales y las neurociencias tienen puntos en común. El uso de simuladores, la telemedicina, el 3D, o la realidad virtual son herramientas ya habituales en la medicina. Las neurociencias y el neuromárketing han dado pasos de gigante gracias al desarrollo de contenidos y herramientas digitales que pueden medir factores impensables hasta hace pocos años. Estos conocimientos se pueden aplicar al diseño y creación de contenidos digitales para mejorar su efectividad e impacto.
“Cuando hablamos de las múltiples aplicaciones laborales de nuestra oferta académica, hay que recordar que áreas como la investigación en medicina y salud utilizan desde hace años las más innovadoras tecnologías digitales, y que la puesta en marcha de productos interactivos es clave para muchos científicos. Tenemos numerosos puntos de desarrollo comunes que recibirán un impulso considerable tras la firma de este acuerdo”, señala Ignacio Pérez Dolset, fundador de U-tad.
”

El Consejo Superior de Investigaciones Científicas (CSIC) es la mayor institución pública dedicada a la investigación en España y la tercera de Europa.

U-tad, Centro Universitario de Tecnología y Arte Digital es el primer centro universitario especializado 100% en la formación de las tres grandes áreas asociadas a la cadena de valor de los contenidos digitales: el arte, la



From www.dreig.eu - September 13, 2012 9:56 AM

8 ARGUMENTOS NEUROCIENTÍFICOS PARA UNA EDUCACIÓN MEJOR
Dolors Reig (dreig) | Wednesday, September 12th, 2012

Me llamaba la atención de nuevo el punto de vista neuroeducativo presente en la infografía explicada que os dejo. En un mundo que requiere aprendizaje permanente vale la pena aplicar lo que ya sabemos sobre la forma en que aprendemos. Dejo al final, porque creo que podría tratarse perfectamente de una serie, alguna referencia más al respecto:
-Tutorías cognitivas: en la intersección entre neurociencia y educación y similar, creo, q lo que métodos como Knewton proponen, se trata de sistemas de aprendizaje basados en “learning by doing” y en el cognitivismo, utilizando sistemas de inteligencia artificial para ajustarse mejor a las necesidades de los estudiantes.
-Cambiar los horarios en secundaria: sabemos que los patrones de sueño cambian con la edad, que los adolescentes necesitan dormir más que otros grupos de población y no son especialmente matutinos. Empezar las clases un poco más tarde, incluso solamente 30 minutos resulta en diferencias importantes en cuestión de humor y atención, cosas que sabéis tan necesarias también en esas épocas.
-Variedad: La repetición es importante para la memoria pero parece que espaciar los aprendizajes en el tiempo favorece el aprendizaje significativo. El cerebro funciona mejor de ese modo, elevando los niveles de atención cuando programamos lo que en formación corporativa se ha denominado “píldoras” cortas. También es importante variar entre formatos y métodos, atendiendo a los distintos estilos de aprendizaje que unos estudiantes que, recordémoslo, acostumbran a lidiar con material extraordinariamente diverso y enriquecido en el mundo postdigital, pueden preferir.

Post completo en la fuente: http://www.dreig.eu/caparazon/2012/09/12/8-argumentos-neuroeducacione/

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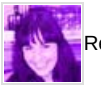

Rescooped by sonia ramos from Psychology and Brain News

Brain filter for clear information transmission: Neuronal inhibition is key for memory formation

From www.sciencedaily.com - September 10, 2012 9:37 AM

“ Every activity in the brain involves the transfer of signals between neurons. Frequently, as many as one thousand signals rain down on a single neuron simultaneously.”

Via Dimitris Agorastos

dirección y gestión, y la ingeniería y programación.

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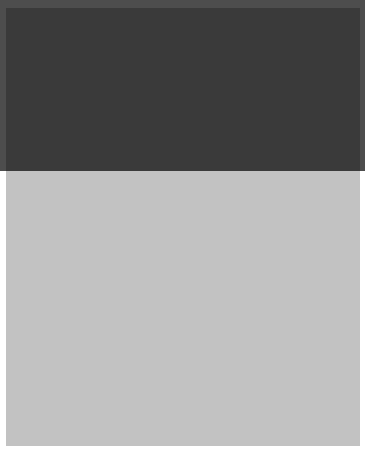
Descubren la importancia de una proteína en el desarrollo del sistema nervioso / Noticias / SINC - Servicio de Información y Noticias Científicas



From www.agenciasinc.es - September 12, 2012 8:24 PM

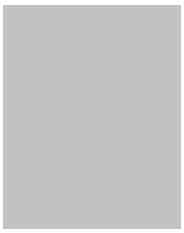
“ SINC, Servicio de Información y Noticias Científicas, plataforma multimedia de comunicación científica (Descubren la importancia de una proteína en el desarrollo del sistema nervioso http://t.co/cEIaVnTK...)...”

Investigadores españoles han analizado el papel de la proteína RhoE en el correcto desarrollo del sistema nervioso. Las conclusiones, a partir del cultivo en el laboratorio de las neuronas de ratones que no expresaban dicha proteína, revelan que este modelo animal presentaba evidentes alteraciones en el cerebro como retraso en el crecimiento de las neuronas, daño neuromotor y neuromuscular, y desarrollo anormal del sistema nervioso.

Científicos del Centro de Investigación Príncipe Felipe (CIPF), de la Universidad CEU-Cardenal Herrera y de la Universidad de Valencia han descubierto la implicación de una proteína llamada RhoE en el desarrollo del sistema nervioso y las alteraciones que su carencia induce en las neuronas.

El estudio, publicado en el Journal of Neurochemistry, se ha llevado a cabo a partir del cultivo en el laboratorio de las neuronas de un ratón que no expresa la proteína RhoE. Los investigadores han estudiado los efectos de la falta de esta proteína y han descubierto importantes alteraciones en el desarrollo neuronal.
Los investigadores han estudiado los efectos de la falta de esta proteína y han descubierto importantes alteraciones en el desarrollo neuronal
Rosa Guasch, investigadora del CIPF y autora principal del artículo, apunta que “este modelo animal presenta evidentes alteraciones en el cerebro como son un retraso en el crecimiento de las neuronas, así como daño neuromotor y neuromuscular, y en definitiva un desarrollo anormal del sistema nervioso”.
La proteína RhoE pertenece a la familia de proteínas “Rho” - homólogas del oncogen Ras-, cuya función principal es la de organizar el citoesqueleto celular. Precisamente por su papel en la restructuración del citoesqueleto, este tipo de proteínas estan implicadas en diversos procesos celulares como la proliferación, migración, secreción, etc. En los últimos años se está resaltando su importancia en diversos aspectos del crecimiento neuronal.

Referencia bibliográfica:
Peris B, Gonzalez-Granero S, Ballester-Lurbe B, García-Verdugo JM, Pérez-Roger I, Guerri C, Terrado J, Guasch RM. "Neuronal polarization is impaired



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in mice lacking RhoE expression". J Neurochem. 2012 Jun;121(6):903-14. doi: 10.1111/j.1471-4159.2012.07733.x. Epub 2012 Apr 13.

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Hablamos con Javier DeFelipe sobre Innovación y Neurociencia

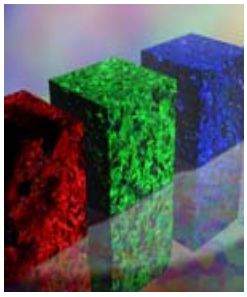
19 octubre, 2012 21:19 joju

Tras la conferencia **Innovation & Neuroscience: Innovación en la ciencia: qué nos hace ser humanos,** impartida por el profesor Javier DeFelipe en el Centro de Innovación BBVA el 18 de octubre, se realizó una pequeña entrevista que se ha editado con imágenes del evento.

Javier deFelipe refuerza la idea de la necesidad del reconocimiento de imagen y posproceso gráfico en el avance de la ciencia, así como el estudio interdisciplinar como modelo de trabajo.

> *¿Qué hace al hombre ser humano? El neurocientífico Javier DeFelipe nos ha planteado respuestas a esta compleja pregunta en torno a uno de los objetivos de la neurociencia: comprender los mecanismos biológicos responsables de la actividad mental humana.*

Hemos tenido la oportunidad de entrevistarle tras la ponencia y averiguar aún más cosas sobre nuestro cerebro.



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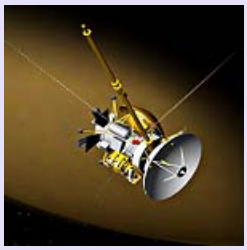

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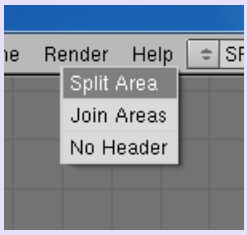

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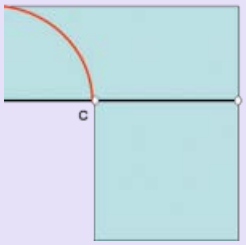

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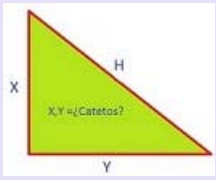

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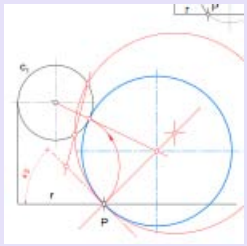

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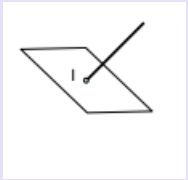

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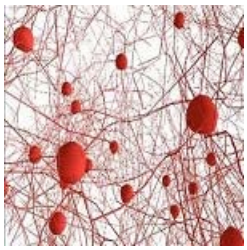

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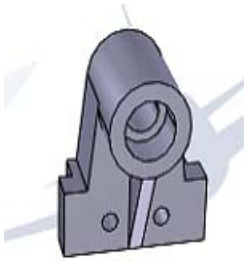

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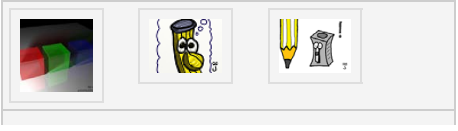

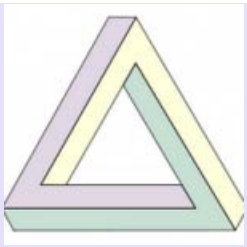

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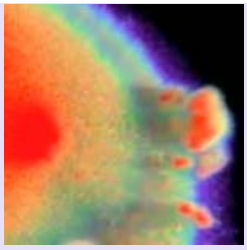
Felices fiestas CAJAL...



Alexander Tsiaras: de la ...

About joju

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Subdirector del Dpto. DVA

Coordinador del grupo de innovación educativa Visual Graphics Group (VGG)

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Alexa

Autor

José Juan Aliaga Maraver

ENTREVISTA A JAVIER DE FELIPE

Javier de Felipe: "Seguro que colonizaremos el espacio"

El eminente neurobiólogo investiga la neocorteza cerebral, donde se halla lo que nos hace ser humanos

Vida | 05/07/2012 - 00:23h

Javier de Felipe durante su visita al Museu Blau de Barcelona R.Q

RAQUEL QUELART | Sigue a este autor en **Twitter** o **Google +**

Somos nuestro cerebro, pero sin embargo conocemos tan poco de él… La necesidad de saber más sobre este órgano y, por lo tanto, de conocernos mejor a nosotros mismos, ha llevado a centenares de expertos de todo el mundo a centrar sus investigaciones en esta parte del cuerpo humano. Este es el caso del neurocientífico Javier de Felipe (Madrid, 1953), especialista en el estudio microanatómico del cerebro, profesor de investigación en el Instituto Cajal (CSIC) y uno de los mayores expertos en la investigación de la corteza cerebral. De Felipe ha colaborado con la NASA en el proyecto NEUROLAB (1998) y, actualmente, forma parte del proyecto Cajal Blue Brain, una iniciativa que desde 2002 pretende aunar los esfuerzos de investigadores de todo el mundo con el fin de entender la estructura cerebral. Los resultados podrían aportar soluciones para enfermedades como el alzheimer y la esquizofrenia.

El neurocientífico ha visitado recientemente Barcelona en el marco de una conferencia -"Sobre la belleza, el arte y la evolución del cerebro"- que tuvo lugar en el Museu Blau y que se enmarca en el conjunto de actividades que se celebran con motivo del año de la neurociencia.

- ¿Qué objetivo persigue el estudio Cajal Blue Brain?

- Es el estudio de las columnas corticales, que es la estructura elemental del funcionamiento de la corteza cerebral, la región del cerebro más relacionada con lo que nos hace ser humanos. El problema es que no sabemos cómo se estructuran las columnas ni cuántas células ni conexiones tienen. Disponemos de una información muy incompleta.

-¿En qué se diferencia de otros proyectos?

- Es el primer intento exhaustivo de hacer ingeniería inversa del cerebro, es decir, desmontarlo

LO MÁS »

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LO MÁS COMENTADO

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para observar sus partes. El cerebro es una máquina muy compleja formada por miles de millones de conexiones y tenemos que trazar el mapa, ver cómo se han creado estos circuitos y, luego, recrear estos procesos mentales en un ordenador.

- ¿Por qué el estudio es interdisciplinar?
- El problema que tenemos en neurociencia es que cada laboratorio trabaja con una pequeña porción de información, pero muchas veces no la pueden usar otros científicos. Con el Cajal Blue Brain nos juntamos muchos expertos de diversos campos, desde ingenieros a informáticos, para avanzar más rápido. Analizamos la estructura con un detalle altísimo, y mediante programas de computación transformamos la información biológica en datos matemáticos y realizamos simulaciones. Hemos conseguido crear neuronas virtuales y queremos hacer lo mismo con las columnas, lo que nos permitirá llevar a cabo miles de experimentos en segundos.

- ¿Se podrían aplicar estos conocimientos al ámbito de la robótica?
- Sí, claro. Nuestro cerebro tiene una capacidad enorme de procesar información, de hacer tareas muy complejas de manera simplificada, utilizando atajos, que debemos averiguar cómo son. Hay que imitar el cerebro para aprender cómo resuelve los problemas. Por ejemplo, a un ordenador le puedes enseñar a multiplicar y lo puede hacer más rápido que un cerebro humano, pero no es capaz de reconocer caras de manera instantánea; el cerebro utiliza 12 watios para procesar una información, mientras que una supercomputadora necesita millones de watios.

- ¿Qué pueden aportarnos estos conocimientos?
- Si queremos conocernos a nosotros mismos, debemos conocer cómo es nuestro cerebro, cómo funciona, cómo ha sido diseñado. Es curioso que esta idea filosófica-científica no esté arraigada en la sociedad, ya que se suele separar cerebro y mente.

- ¿La complejidad de la neocorteza nos diferencia de otras especies?
- No, no es la complejidad ni el tamaño del cerebro.

- Entonces, ¿qué nos hace ser distintos?
- A pesar de estar en el siglo XXI, es un misterio que todavía no ha sido resuelto, no sabemos lo que nos hace distintos de otras especies. Una gran parte de los científicos piensa que la diferencia radica en la complejidad, en el mayor número de columnas. No somos muchos los que defendemos lo contrario, es decir, que lo que nos hace ser diferentes es la estructura, que hay células que son distintas, que cada especie animal tiene un cerebro propio.

- Sin embargo, la mayoría de los experimentos se realiza con animales.
- Lo que se estudia en la rata se extrapola al cerebro humano y eso se emplea para modelos de alzehimer, esquizofrenia, autismo y muchas otras enfermedades. Es un problema porque resulta que, aunque hay cosas idénticas entre el hombre y el ratón -como las estructuras de la neocorteza-, existen muchas diferencias y eso no se acepta fácilmente.

- ¿A qué animal nos parecemos más?
- Al chimpancé, con el que compartimos un antepasado común hace seis millones de años. Pero, ¿cuánta información de un animal de experimentación puedes extrapolar al cerebro humano? Yo creo que es relativamente poca.

- ¿Somos superiores al resto de animales?
- Simplemente somos distintos, pero tenemos una capacidad de abstracción superior a la del resto de especies. En otro aspecto, como el olfativo, nos dominan claramente los perros, que también tienen un cerebro muy complejo y se deprimen y sufren. Por ejemplo, otros animales son buenos detectando sonidos, otros con los colores…

- La creatividad emana de nuestra neocorteza cerebral. ¿Por qué el hombre es capaz de crear arte?
- Es un gran misterio, es interesante porque la estética no es necesaria para la supervivencia, pero sin embargo nos produce un placer intelectual increíble y nos hace sentir mejor por razones desconocidas. Sabemos que se activan las zonas del cerebro que están relacionadas con los circuitos de recompensa, al igual que ocurre cuando tomamos drogas. Somos el único animal capaz de modificar las cosas para crear belleza, música, pintar… Una reflexión: durante miles y miles de años el hombre ha sido incapaz de crear arte, ya que este ha surgido relativamente hace poco tiempo.

- ¿Es quizá porque nuestro cerebro ha evolucionado?
- No, el cerebro es el mismo desde que apareció el homo sapiens, hace 200.000 años. Los primeros indicios de capacidad de abstracción se han encontrado en Sudáfrica, datan de hace 100.000 años y son unas incisiones geométricas que ya indican un pensamiento simbólico; en Austria se ha descubierto una escultura de 35.000 años de antigüedad, un hombre con cabeza de león, que es lo más antiguo que se conoce de creatividad artística; incluso, hay un estudio

AL MINUTO »

16:02 Montserrat Caballé anula su concierto del 23 de febrero en Granollers por...

16:00 El Barça, favorito en el clásico para las casas de apuestas

15:51 El 'Monstruo de las Galletas' reivindica el robo del emblema de Bahlsen

15:50 Navarro pide a Mas celebrar la cumbre anticrisis antes de los presupuestos

15:48 Merkel reclama ante Morsi respeto a los derechos humanos y diálogo

Ir al minuto

que demuestra que algunas de las pinturas de las cuevas de Altamira tienen 41.000 años.

- ¿Cuál es la conclusión?
- ¡Hemos perdido 160.000 años de creatividad, antes no ha habido nada!

- ¿Alguna explicación?
- La evolución ha sido cultural, no biológica. Un recién nacido en nuestra sociedad enseguida aprende a hablar y leer, pero si naciera en otro sitio donde no hubiera cultura, sería incapaz de hablar e, incluso, podría tener un retraso mental. Nuestro cerebro nace con unas capacidades enormes que son desconocidas, todavía no sabemos adónde vamos a llegar biológicamente.

- Entonces, es la capacidad de abstracción lo que marca la diferencia del ser humano.
- Una expresión tan habitual como "vuelvo mañana" es un pensamiento muy complejo, y que yo te lo diga a través de un sonido y que tú lo entiendas es impresionante. Es una adquisición humana muy tardía. Si un homo sapiens de hace 200.000 años naciera hoy, sería como nosotros, y al contrario, Einstein, Goya, Picasso y Dalí nunca hubieran sido lo que fueron de haber nacido en la época en que no existía el esqueleto intelectual. Nuestro cerebro es una máquina que se autorepara y aprende de sí misma, es como una esponja del entorno.

- Usted se muestra convencido de que el hombre colonizará el espacio, ¿el cerebro cambiará?
- Si eso ocurriera, el ser humano se convertiría en una especie nueva. En un experimento que hicimos con la NASA observamos cambios en los circuitos de la neocorteza de animales que viajaron al espacio durante su proceso de maduración cerebral. Esos cambios permanecieron en los animales que regresaron a la Tierra.

- ¿Esto qué significa?
- Nuestro cerebro ha evolucionado en este planeta, somos terrícolas, si pasamos a otro entorno, con el tiempo desarrollaremos otras habilidades distintas. Los viajes espaciales los vemos todavía como ciencia ficción, pero el desarrollo tecnológico de estos últimos 100 años es espectacular, por lo que no sabemos dónde vamos a estar dentro de medio mileno. Seguro que colonizaremos el espacio. Pero, claro, si vas a Marte o más allá no es para volver, no vale la pena pasar media vida en una nave.

- Claro.
- Los que nazcan en otro planeta tendrán su colonia de individuos que puede que se comuniquen con la Tierra, o no. Nuestro cerebro se adaptará o a lo mejor no podrá hacerlo y esos individuos acabarán desapareciendo. Pero no sería sorprendente que al estar en un entorno distinto al que fuimos creados hace miles de años, se produzcan cambios y mutaciones y al final nos convirtamos en una especie distinta.

- ¿Qué es lo más increíble del cerebro para usted?
- Que el cerebro soy yo, que una materia me hace sentirme ser yo. Fíjate que nosotros hemos surgido de la materia y que a lo largo de la evolución del tiempo se han ido formando estructuras hasta llegar al cerebro humano.

This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Neurology				
Epilepsy; seizures	MicroRNA-134 (miR-134)	Mouse studies suggest inhibiting miR-134 could help treat and prevent seizures. In mice, hippocampal neurons damaged by prolonged seizures had higher miR-134 levels than undamaged neurons. In mice, an antagomir against miR-134 decreased seizure activity and seizure-associated neuronal damage compared with a nontargeting antagomir. Next steps include developing an miR-134 inhibitor and evaluating it in additional animal seizure models. *SciBX* 5(26); doi:10.1038/scibx.2012.681 Published online June 28, 2012	Patent application filed covering inhibitors of miR-134 to treat or prevent seizure-related disorders and neurologic injuries; available for licensing from the Royal College of Surgeons in Ireland **Contact:** Gearóid Tuohy, Royal College of Surgeons in Ireland, Dublin, Ireland e-mail: gearoidtuohy@rcsi.ie	Jimenez-Mateos, E.M. *et al. Nat. Med.*; published online June 10, 2012; doi:10.1038/nm.2834 **Contact:** David C. Henshall, Royal College of Surgeons in Ireland, Dublin, Ireland e-mail: dhenshall@rcsi.ie

Nature Reviews Neuroscience | AOP, published online 4 July 2012; doi:10.1038/nrnIB1

IN BRIEF

NEUROIMMUNOLOGY

Harnessing adaptive immunity for AD

Immunotherapy has potential as a disease-modifying approach to the treatment of Alzheimer's disease. In this study, Santuccione *et al.* found that among individuals with Alzheimer's disease, those who exhibit slower cognitive decline have higher blood levels of antibodies specific for the neuronal cytoskeletal protein ankyrin G (ANKG; also known as ANK3). ArcAβ mice (a transgenic mouse model of Alzheimer's disease) that were vaccinated with ANKG had fewer and smaller amyloid plaques than unvaccinated animals, as well as less spine loss. Thus, taking advantage of the adaptive immune response to ANKG could be a potential approach for Alzheimer's disease immunotherapy.

ORIGINAL RESEARCH PAPER Santuccione, A. C. *et al.* Active vaccination with ankyrin G reduces β-amyloid pathology in APP transgenic mice. *Mol. Psychiatry* 12 Jun 2012 (doi:10.1038/mp.2012.70)

NEUROLOGICAL DISORDERS

Focus on miR-134 for seizures

The numerous changes in synaptic physiology and structure that occur in epilepsy are not effectively targeted by the currently available anti-epileptic therapies. Here, Jimenez-Mateos *et al.* found that miR-134 — a brain-specific microRNA reported to be involved in regulating spine morphology — was upregulated in the brain of patients with epilepsy and in a mouse model of epilepsy. Pharmacological silencing of miR-134 in mice through the use of antagomirs attenuated seizure severity during status epilepticus and reduced cell death and classical hallmarks of epilepsy, such as astrogliosis.

ORIGINAL RESEARCH PAPER Jimenez-Mateos, E. M. *et al.* Silencing microRNA-134 produces neuroprotective and prolonged seizure-suppressive effects. *Nature Med.* 10 Jun 2012 (doi:10.1038/nm.2834)

SYNAPTIC PHYSIOLOGY

No return for spiking axons

Under certain circumstances, action potentials can be initiated in the distal axon, but how the back-propagation of this activity to the soma is prevented is unknown. In this study, pharmacologically induced gamma oscillations in mouse hippocampal slices produced ectopic action potentials in distal axons of CA3 pyramidal cells. Back-propagation of these spikes to the soma was reduced by GABA release from axo-axonic cells at the axon initial segment during fast oscillations, thus enabling functional compartmentalization of axonal and somatic activity.

ORIGINAL RESEARCH PAPER Dugladze, T. *et al.* Segregation of axonal and somatic activity during fast network oscillations. *Science* **336**, 1458–1461 (2012)

NEUROLOGICAL DISORDERS

SHANK2 misbehaves in autism

Numerous genetic studies have reported an association between *SHANK2* and autism spectrum disorders (ASDs). Won *et al.* generated mutant mice carrying a human ASD-associated microdeletion in *Shank2*. These mice exhibited a marked reduction in hippocampal NMDA receptor-mediated long-term potentiation and long-term depression and showed autistic-like behaviours (such as altered social interaction). Importantly, these behavioural deficits were attenuated by pharmacological modulation of metabotropic glutamate receptors that indirectly enhance NMDA receptor function, suggesting a possible future therapeutic approach for ASDs.

ORIGINAL RESEARCH PAPER Won, H. *et al.* Autistic-like social behaviour in *Shank2*-mutant mice improved by restoring NMDA receptor function. *Nature* **486**, 261–265 (2012)

Gardaí in Waterford are seeking assistance in tracing John Swift, 57, from the city, who is missing since July 19. He was last reported seen at his home that morning.

He is described as being 6ft 1in, with a broad build and blue eyes. He was wearing a green and cream check shirt, blue jeans, and sandals.

It is thought Mr Swift may be driving a wine-coloured Volvo S40.

Contact Waterford gardaí at 051 305300, the Confidential Line at 1800 666111, or any station.

Preventing epileptic fits

Irish scientists have found a new way to stop epileptic fits. Epilepsy affects 37,000 people in Ireland — about 1% of the population, and there is no cure.

One in three people who suffer from epilepsy are either drug resistant or do not respond well to medication.

Now neuroscientists at the Royal College of Surgeons in Ireland are using a drug that blocked a newly discovered gene linked to epilepsy, mircoRNA-134, from working properly.

First US woman in space dies

Sally Ride, the first US woman to travel into space, died yesterday after a 17-month battle with pancreatic cancer, according to her organisation, Sally Ride Science, in San Diego. She was 61.

Ride, a physicist, blasted off in the US space shuttle Challenger on Jun 18, 1983.

"Sally's historic flight into space captured the nation's imagination and made her a household name," Sally Ride Science said in a statement.

Trains pledge

Iarnród Éireann has pledged to improve its on-board announcement systems on both Dart and commuter train services.

It has confirmed an initial investment of €120,000 to renovate the outdated announcement systems on a number of Dart services.

Crash couple still critical

The Irish couple whose

permits termination if a mother's life is at risk, including from suicide.

The expert group of medical and legal experts is due to present a range of options to the Government in September, but already rifts are appearing between the Coalition partners, and also within the parties.

The Supreme Court ruled 20 years ago that the Human Rights in 2010.

But the Government could take a less liberal view and Europe Minister Lucinda Creighton, when asked when the Government would act on the Supreme Court's ruling and introduce legislation, said: "The Constitution can always be changed."

The Fine Gael minister said having ministers and

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"I think it
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speculate."

Taoiseach Enda Kenny before his address to the MacGill Summer School

Senator wants candida

Fianna Fáil senator Averil Power called for candidates for public office to sign up to a legally-binding ethics pledge which could be monitored by an independent electoral commission.

Her comments came as academic Elaine Byrne suggested that whistleblowers needed to receive recognition for their work.

Both were speaking at the MacGill Summer School in Donegal while debating tribunal reports.

Ms Power said fines or sanctions could be set for those who breached any standards. "The days of TDs, senators, or the other public representatives being able to lawyer up in order to dodge legitimate questions have to be ended," she said.

"That must apply whether the
are scruffy-looking new deputie
or sitting taoisigh."

She said her own party had bee
hurt by the damning findings i
the Mahon Tribunal plannin
report, and insisted ordinary part
members never sought one cer
working in politics.

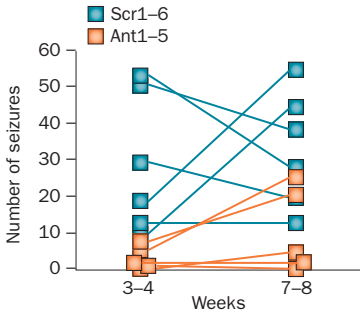
The Seanad spokeswoman o
education criticised the Coalitio
for its inadequate response to th
Moriarty probe, the inquiry int

Burton atta

EPILEPSY

Silencing of miR-134 suppresses seizures in mice

A research team in Dublin, Ireland has shown that a specific microRNA (miRNA) is upregulated in the brains of mice and humans with epilepsy, and could represent a new target for interventions to treat this condition. In a study reported in *Nature Medicine*, David Henshall and colleagues found that silencing of miR-134 after induction of status epilepticus in mice dramatically reduced the subsequent occurrence of both spontaneous seizures and epilepsy-associated pathology.



Effects of miR-134 depletion on spontaneous seizure rates in mice 1 month and 2 months post-status epilepticus. Graph shows data from five mice injected with an antagomir targeting miR-134 (Ant1–5) and six mice injected with a nontargeting scrambled sequence (Scr1–6). Image courtesy of D. C. Henshall.

MiRNAs are noncoding RNA molecules that regulate protein synthesis by binding to mRNAs, and are being implicated in an increasing number of neurological disorders. "MiR-134 was on our shortlist because of work showing that it regulated dendrites," explains Henshall. "This seemed an interesting miRNA to focus on as dendrites are major sites of excitatory synaptic contact and are known to be changed in epilepsy."

Henshall's team generated a mouse model of status epilepticus by injecting the glutamate receptor agonist kainic acid into the amygdala. The resulting mice exhibited increased levels of miR-134 in the CA1 and CA3 regions of the hippocampus ipsilateral to the injection site. Consistent with this observation, miR-134 was also found to be upregulated in brain tissue from patients who had undergone surgery to treat refractory temporal lobe epilepsy (TLE).

To establish whether the elevated miR-134 levels were pathogenic, the researchers used an miRNA inhibitor known as an antagomir to silence miR-134 in their mouse model. The Ant-134 antagomir was administered via intracerebroventricular injection 1 h after induction of status epilepticus. "The result was stunning—over 90% fewer epileptic seizures during 2-week recordings," says Henshall. "Even when we waited 1 or 2 months, the animals were having far fewer seizures."

In parallel with the reduction in seizure frequency, the antagomir was shown to protect against the development of TLE-associated pathology, which includes neuronal loss, gliosis and mossy fibre rearrangment. Intriguingly, the antagomir also produced a reduction in dendritic spine density in CA3 pyramidal neurons—an effect that, the researchers speculate, might contribute to seizure suppression.

"This is the first study to show an effect of a single miRNA on seizures directly," concludes Henshall. "The antagomirs are a very potent antiseizure tool that works in a completely different way from current antiseizure drugs—they could be useful for drug-resistant epilepsy or refractory status." His team now plans to monitor mice beyond 3 months after Ant-134 treatment to establish whether suppression of seizures persists once miR-134 levels have returned to normal.

Heather Wood

Original article Jimenez-Mateos, E. M. *et al.* Silencing microRNA-134 produces neuroprotective and prolonged seizure-suppressive effects. *Nat. Med.* doi:10.1038/nm.2834

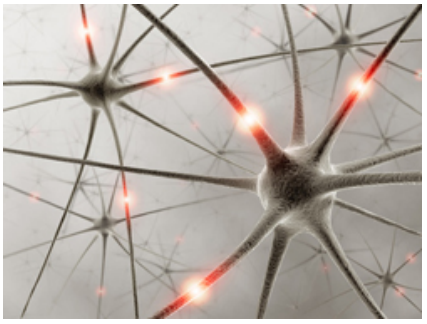
IN BRIEF

Estudian el efecto de las placas de beta amiloide en cerebros con Alzhéimer

Investigadores del Laboratorio Cajal de Circuitos Corticales de la UPM avanzan en la comprensión del modo en que la acumulación de placas de beta amiloide afecta a los axones de las neuronas.

29.10.12

Tratar de llegar a comprender mejor los cambios que se producen en el cerebro afectado por la enfermedad de Alzheimer es uno de los objetivos de un estudio llevado a cabo por un equipo de investigadores del Laboratorio Cajal de Circuitos Corticales de la Universidad Politécnica de Madrid (UPM). Una de las características fundamentales de la enfermedad de Alzheimer es la acumulación en el cerebro de una sustancia llamada beta amiloide. Por ello, el objetivo de los investigadores era determinar si la presencia de placas de esta sustancia provocaba alteraciones en las neuronas.



En una investigación con ratones de laboratorio a los que se les ha inducido la producción de beta amiloide , los expertos han analizado mediante microscopía confocal (1), las características morfológicas así como la inervación GABAérgica (2) del segmento inicial del axón de un tipo específico de neuronas: las neuronas piramidales. El segmento inicial del axón (3) es una región de especial interés ya que está implicada en la generación de los potenciales de acción necesarios para la transmisión de información entre las neuronas.

Como resultado de su trabajo, los investigadores constataron que la estructura del segmento inicial del axón no se ve afectada por la acumulación de beta amiloide. Sin embargo, , el estudio demostró que la inervación GABAérgica disminuye cuando el segmento inicial del axón contacta con las placas de beta amiloide o pasa cerca de ellas, lo que puede ayudar a explicar la hiperactividad neuronal que presentan algunos pacientes de Alzheimer .

Para Javier De Felipe, director del área de Neurobiología del Proyecto Cajal Blue Brain (UPM-CSIC), el trabajo supone “un paso más hacia una mejor comprensión de los fenómenos que ocurren en un cerebro con la enfermedad de Alzheimer. Cuanto más se conozca sobre las causas y consecuencias, más cerca estaremos de desarrollar un tratamiento eficaz para esta enfermedad”.

Los investigadores (Gonzalo Leon-Espinosa, Javier DeFelipe y Alberto Muñoz) centrarán ahora su estudio en “la influencia de las placas de beta amiloide sobre otros componentes del segmento inicial del axón, como son los canales iónicos necesarios para la generación de los potenciales de acción”, continúa Muñoz. Además, pretendemos estudiar el papel que desempeña la hiperfosforilación de tau (la otra característica patológica de la enfermedad) sobre diversos aspectos del segmento inicial del axón, para conocer mejor cómo afecta todo ello a la transmisión de información en el cerebro”, concluyen.

Más información sobre el Laboratorio Cajal de Circuitos Corticales

Dirigido por Javier DeFelipe, el Laboratorio Cajal de Circuitos Corticales tiene una dilatada experiencia en el análisis de la organización microanatómica y neuroquímica de la corteza cerebral, mediante la utilización de una variedad de técnicas, entre las que se incluyen inyecciones intracelulares, técnicas histoquímicas e inmunocitoquímicas para microscopía óptica y electrónica, y métodos de reconstrucción 3D para microscopía óptica y electrónica.

A partir de 2006 comenzó a centrarse en el estudio de la enfermedad de Alzheimer, y se continúan los estudios sobre la microestructura de la corteza cerebral normal. En 2009 se inicia una nueva etapa con la participación en el proyecto Blue Brain cuyo origen se remonta al año 2005, cuando L’École Polytechnique Fédérale de Lausanne (Suiza) y la compañía IBM anunciaron conjuntamente el ambicioso proyecto de crear un modelo funcional del cerebro utilizando el superordenador Blue Gene, de IBM.

A finales de 2006, el proyecto Blue Brain había creado un modelo de la unidad funcional básica del cerebro, la columna neocortical. Sin embargo, las metas marcadas por el proyecto imponían su conversión en una iniciativa internacional. En este contexto surge en España el proyecto Cajal Blue Brain, cuyos objetivos se encuadran en dos ejes principales: La microorganización anatómica y funcional de la columna neocortical y el desarrollo de tecnología biomédica (fundamentalmente informática). La contribución del laboratorio de DeFelipe consiste en participar en los estudios microanatómicos y funcionales de la columna neocortical y coordinar las tareas de investigación realizadas en otros laboratorios nacionales e internacionales. Esta coordinación es fundamental para establecer una base de datos homogeneizada que permite estandarizar y aprovechar al máximo los datos generados por cada grupo de investigación.

Notas:

(1) Microscopio confocal: El microscopio confocal es un microscopio que emplea una

técnica óptica de imagen para incrementar el contraste y/o reconstruir imágenes tridimensionales utilizando un "pinhole" espacial (colimador de orificio delimitante) para eliminar la luz desenfocada o destellos de la lente en especímenes que son más gruesos que el plano focal.

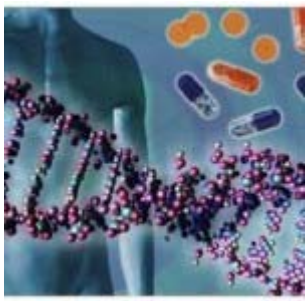
(2) Inervación Gabaérgica: Los circuitos neuronales son modulados en función del balance entre las señales excitadoras e inhibidoras, estas últimas generadas desde distintas poblaciones de interneuronas GABAérgicas. En la corteza cerebral, cada una de estas interneuronas inerva selectivamente diferentes dominios de células neuronales piramidales (como por ejemplo el segmento inicial del axón). Por tanto, la actividad cortical depende, entre otros factores, de GABA (Gamma-aminobutirato), un importante neurotransmisor del cerebro en mamíferos.

(3) Axón: Se llama axón a una prolongación larga y delgada de las neuronas que se origina en una región especializada llamada eminencia axónica o cono axónico, a partir del cuerpo central de la neurona, o a veces de una dendrita (una de las prolongaciones cortas de las neuronas). Su función es el transporte de orgánulos y sustancias, y la conducción del impulso nervioso.

by Chuck Carmen

New Epilepsy Gene Identified; Possible New Treatment Option

July 23, 2012 *in* *Epilepsy*, *Global*, *Neurology*, *News*, *Research*, *Seizures*, *Technology*, *Treatment*



New research conducted by neuroscientists from the Royal College of Surgeons in Ireland (RCSI) published in *Nature Medicine* has identified a new gene involved in epilepsy and could potentially provide a new treatment option for patients with epilepsy.

The research focussed on a new class of gene called a 'microRNA' which controls protein production inside cells. The research looked in detail at one particular microRNA called 'microRNA-134' and found that levels of microRNA-134 are much higher in the part of the brain that causes seizures in patients with epilepsy. Read the rest of this entry →

No Comments »

Community Learning New

by Chuck Carmen

Switching Epilepsy Medications

July 23, 2012 *in* Epilepsy, Medicine, Treatment

Fifty percent of all patients with newly diagnosed epilepsy will become seizure-free with the first epilepsy drug they try. For the rest, it's try, try again: switching epilepsy medications, adjusting to side effects, and waiting to make sure the new drug works. Others find their seizures are controlled, but they can't tolerate the medication's side effects and need to switch drugs.

Before you ask your doctor if your medication should be switched, make sure you are taking your current medication exactly as prescribed. Missing doses, splitting pills, or not following instructions to the letter could affect your control and the side effects you experience. If you are already complying with your doctor's instructions, but still are having breakthrough seizures, talk to your neurologist or epileptologist (specialists who are experts in treating epilepsy). Your doctor will evaluate whether you should switch medications. Read the rest of this entry →

1 Comment »

by Chuck Carmen

Treating epilepsy while trying to conceive

July 23, 2012 *in* Epilepsy, Global, News, Women and Epilepsy

*

Forums

Community Discussions
Teachers
Support
Parents

News

Select Month

Groups

Newest | Active | Popular

Epilepsy Association of Central Florida

active 9 hours, 48 minutes ago

EpilepsyU

active 11 hours, 45 minutes ago

Working with epilepsy

active 15 hours, 54 minutes ago

Parents of School Children

active 5 days, 10 hours ago

Side effects of medications

active 1 week, 2 days ago

Community Learning News

In the United States, epilepsy affects nearly one million women of childbearing age.

“Many women with epilepsy experience seizure exacerbations around the time of their menstrual periods, and that is thought to do to the cyclic hormones during the menstrual cycle,” said Dr. Cynthia Harden, director of the Comprehensive Epilepsy Care Center at North Shore-LIJ Health System. “We are working on ways to treat specifically those hormone-exacerbated seizures for women with epilepsy.” Read the rest of this entry →

No Comments »

by Chuck Carmen

The CURE Introduce 2012 Research Awards.

July 19, 2012 *in* *Epilepsy*, *Global*, *Medicine*, *Neurology*, *News*, *Research*, *Technology*, *Treatment*

Susan Axelrod and her organization the CURE (Citizens United for Research in Epilepsy) are thrilled to introduce the second wave of research award recipients for 2012 as they mover forward to fund innovative projects and their efforts to find cures for epilepsy. To see the recipients and a summary of their research read more. Read the rest of this entry →

No Comments »

Topics

how my twins got cured of epilepsy.
School Counselor
Sabrina's mom
Night Driving
Who do you see for your epilepsy?
Pre-existing condition for insurance
Spouses of people with epilepsy
Introduce yourself
Bug(s) Found
I have Epilepsy

My Points

You need to be logged in to view your points.

Community Learning News

by *Chuck Carmen*

Leading an active life with epilepsy possible, but requires caution!

July 19, 2012 *in* *Epilepsy*, *Global*, *News*

Austin “DJ” Brooks was never one to sit still. He loved to be active, even seeking out extreme activities like riding dirt bikes and racing stock cars. The 24-year-old Oxford man was an epileptic, but it was rare for him to have a grand mal seizure, often marked by a loss of consciousness and violent muscle contractions.

Given the rarity of his seizures, Brooks likely felt there was no reason to keep his epilepsy from letting him live an active life, and many experts probably would have agreed with him. But in late June, Brooks, who was reportedly reeling in a bass while fishing on Swan Lake in Oxford with his girlfriend, had a seizure, lost control of his body and fell out of the canoe he was in and drowned. Read the rest of this entry →

1 Comment »

by *Chuck Carmen*

Tampa General Tops in US News Florida Hospital List – Fl. Hospital Second

July 18, 2012 *in* *Global*, *News*

U.S. News and World Reports has taken its national rankings system to the state level, naming Tampa General Florida’s best hospital.

Community Learning News

Tying for #2 were Florida Hospital in Orlando and Shands Hospital in Gainesville.

The magazine's "best hospitals" list this year is different from in the past, when it was based almost wholly on reputation. Now reputation accounts for only 32.5 percent of the score; more objective measures of patient safety and outcomes, based on federal data, now account for the majority of the score, as HealthLeaders Media reports.

The top 10 hospitals in the state were listed in the publication's national rankings for various specialties. Tampa General was recognized in the nation's top 50 for nine specialties, according to a news release from TGH. Read the rest of this entry →

No Comments »

by Chuck Carmen

Kerry Kennedy says seizure caused car wreck

July 18, 2012 *in* *Epilepsy*, *News*

New York (CNN) – Kerry Kennedy, the daughter of the late Robert F. Kennedy and ex-wife of New York Gov. Andrew Cuomo, said a partial seizure is the cause of a Friday morning car crash. At a press conference on Tuesday, Kennedy also said the seizure might have been from a previous injury she suffered on the right side of her brain.

Kennedy, 52, was found by state police behind the wheel of her damaged Lexus SUV. A New York State Police press release said the SUV collided with a tractor-trailer. Read the rest of this entry →

Community Learning News 1 Comment »

by *Epilepsy Uni*

Hero Hank: 4-year-old fights serious form of epilepsy

July 17, 2012 *in* *Childhood Epilepsy*, *Epilepsy*, *Febrile Seizures*, *Media*, *Neurology*, *Parents*, *Seizures*, *Social Media*

Photo: Provided
Hank Kovach was recently the guest of honor at a Joliet Slammers minor-league baseball game. The Chicago resident is shown with Slammers mascot J.L. Bird. The team donated money to the Doose Syndrome Epilepsy Alliance.

The 4-year-old boy is the son of Jerry and Megan Turner Kovach, of Chicago. Jerry is a native of Streator and a graduate of Woodland High School. Hank's grandparents include Marilyn Kovach, of Streator, and the late William Kovach.

Hank has been diagnosed with Doose Syndrome, a rare and catastrophic form of childhood epilepsy.

Also known as myoclonic astatic epilepsy, Doose Syndrome oftenisresistant to medication and difficult to treat. It usually arises spontaneously with no known origin and seizures occur throughout the brain

without a focal point. Many children can have hundreds of seizures per day. Children who have Doose Syndrome have a significantly increased risk of sudden unexplained death in epilepsy patients.

Hank stopped babbling at about 9 months old, and that's when his parents began to seek answers.

"Our pediatrician assured us that we were overreacting and said that we shouldn't compare Hank to his older sister, Ruby, but we just felt something was off," Megan Turner Kovach told The Times. "Hank was not crawling or walking well after one year of age and we became concerned at how quiet he became."

Hank was 18 months old when he had his first febrile seizure. His doctor again reassured Jerry and Megan and sent them home, saying most children have seizures with a high temperature.

"We began to become extremely concerned by this," Megan said. "Hank's first febrile seizure lasted over five minutes. It was one of the most frightening experiences we have been through as parents."

The Kovaches say they now know Hank was having a variety of seizures, such as myoclonic muscle spasms and head drops—up to hundreds per day. They believe Hank's initial developmental delays resulted from undiagnosed seizures. After Hank was hospitalized a second time for having a series of severe seizures, the family was referred to the pediatric epilepsy center at Children's Memorial Hospital in Chicago.

Despite finally having a diagnosis and guidance, Hank went into convulsion status twice and while waiting for the paramedics the first time, the family almost lost Hank.

"Those moments were the scariest of our lives as we had to witness our little boy's complexion quickly turn purple and grey from

head to toes as he stopped breathing,"Megan said. "Thankfully, he began breathing again as the paramedics arrived to transport him to the hospital."

The couple said as a result of these instances, Hank seems to have lost some of his speaking and cognitive skills.

Prior to diagnosis, Hank had been receiving treatment for developmental speech, physical and motor skill delays through the state's early intervention program. Now the main focus is on seizure control.

While Hank has been prescribed several types of anti-seizure medications, they carry harmful side effects, which have impacted Hank. His epileptologist recommended the ketogenic diet as an alternative treatment.

"Their philosophy is that if the diet works for the patient, then weaning of medications should take place until eventually the ketogenic diet becomes the medication itself to control seizures,"Jerry said. "Hank is one of the fortunate ones who reacted positively to the ketogenic diet."

Still, there are health risks to being on the high-fat, adequate-protein, low-carbohydrate diet long term. Hank is followed by a dietitian and epileptologist. Children on the ketogenic diet have to be monitored closely. If they eat food that contains sugars and carbohydrates it could lead to a recurrence of seizures.

Today the family is hopeful that Hank's seizures will not return and his brain will continue to heal. He has been showing gains with his development as well, although there are no guarantees he will catch up with his peers.

The family is grateful to have a strong support network. Jerry is one of 12 children, while Megan is one of 13.

"During Hank's hospital stays we were able to count on the support of our families and

friends, which included visits to Hank's hospital room,"Megan said. "They also have embraced the seriousness of his diagnosis and treatment. We know of many people who do not have this level of support and often tell us their own family members were not even supporting the need to monitor what their children were eating and did not believe in the ketogenic diet."

The Kovaches have learned to value their time together even more throughout Hank's illness.

"As a family, it has brought us closer together,"Jerry said. "We do not take anything for granted, and we appreciate the smallest of things that Hank shows in terms of his childhood development, such as learning a new sign to communicate his basic needs and wants to us."

Hank's R Hero Fundraising Benefit

The Turner and Kovach families are hosting a benefit for Doose Syndrome Epilepsy Alliance

- **What:** Pork-chop dinner, silent auction.
- **When:** Saturday, July 28, 5 to 10 p.m.
- **Where:** Gardner American Legion.

Donations can be mailed to:

Hank's R Hero
Campus State Bank
P.O. Box 127
Campus, IL 60920
(Make checks payable to DSEA)

Print this story

Tags: Doose Syndromw, Febrile, ketogenic diet, myoclonic

No Comments »

by *Epilepsy Uni*

Twins bond in the gift of the other: Hailey is there for Olivia, born with

epilepsy and cerebral palsy

July 16, 2012 *in* *Epilepsy*, *Family and Friends*, *Local*, *Media*, *Neurology*, *News*, *Parents*, *Seizures*, *Social Media*, *Support*

This touching story comes out of CLEARWATER, FL and was published yesterday by the Tampa Bay Times. What a great story to start the week!

Click here to view the video!

It was just after the 2 p.m. opening time and already Hailey Scheinman's lemonade stand had attracted a crowd.

The 7-year-old turned around to brush the sleep from her twin sister's eye. Olivia sat quietly in a hot pink wheelchair that was decorated with flowers and butterflies.

Hailey's best friend arrived to help, and the pair jumped up and down at the end of the driveway.

"Lemonade! Lemonade!"

Hailey's friend said goodbye a couple of hours later, and Hailey stood at the edge of the driveway watching the girl's car disappear down the street.

Hailey retreated to the front porch and counted the profits: $142.65, all for her sister's physical therapy.

Community Learning News

What makes a good sister?

Read the rest of this entry →

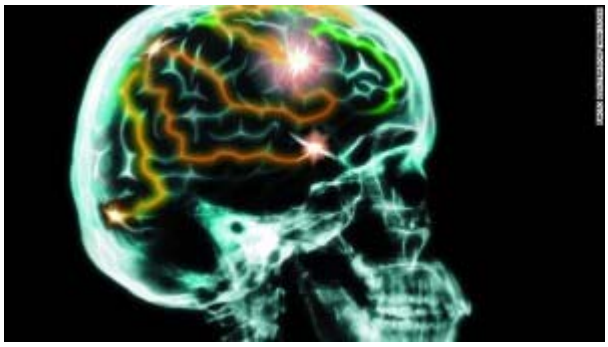
Tags: Cerebral Palsy, Epilepsy, Twins

1 Comment »

by *Epilepsy Uni*

The Cause of Temporal Lobe Epilepsy Identified!

July 16, 2012 *in* *Epilepsy*, *Global*, *Medicine*, *News*, *Research*, *Seizures*



Tokyo: Japanese researchers claim they have pinned down the cause of temporal lobe epilepsy, a chronic neurological condition that affects one per cent of world`s adults.

A group of researchers at the University of Tokyo say their research found that the condition is caused by excitatory neural signaling generated in the brain when a child experiences a seizure along with a fever from a cold or flu, news agency Kyodo reported.

The team, including Yuji Ikegaya, associate professor in the university`s graduate school of pharmaceutical sciences, said it could pave the way for finding a treatment to prevent epilepsy.

Epilepsy affects around one per cent of adults around the world. In Japan, more than one million people are believed to have the condition.

Symptoms of temporal lobe epilepsy often manifest in adulthood. While this form of

epilepsy has been ascribed to febrile seizure, its exact mechanism is unknown.

The research findings were reported in the online version of Nature Medicine, a biomedical research journal yesterday.

Tags: Casue, research, Temporal Lobe Epilepsy

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Research finds new gene involved in epilepsy

July 23rd, 2012

Published on July 23, 2012 at 8:36 AM

New research conducted by neuroscientists from the Royal College of Surgeons in Ireland (RCSI) published in Nature Medicine has identified a new gene involved in epilepsy and could potentially provide a new treatment option for patients with epilepsy.

The research focussed on a new class of gene called a ‘microRNA’ which controls protein production inside cells. The research looked in detail at one particular microRNA called ‘microRNA-134′ and found that levels of microRNA-134 are much higher in the part of the brain that causes seizures in patients with epilepsy.

By using a new type of drug-like molecule called an antagomir which locks onto the ‘microRNA-134′ and removes it from the brain cell, the researchers found they could prevent epileptic seizures from occurring.

Professor David Henshall, Department of Physiology & Medical Physics, RCSI and senior author on the paper said ‘We have been looking to find what goes wrong inside brain cells to trigger epilepsy. Our research has discovered a completely new gene linked to epilepsy and it shows how we can target this gene using drug-like molecules to reduce the brain’s susceptibility to seizures and the frequency in which they occur.”

Dr Eva Jimenez-Mateos, Department of Physiology & Medical Physics, RCSI and first author on the paper said “Our research found that the antagomir drug protects the brain cells from toxic effects of prolonged seizures and the effects of the treatment can last up to one month.”

Source: http://www.nature.com/nm/journal/vaop/ncurrent/abs/nm.2834.html

Follow this link:
Research finds new gene involved in epilepsy

Tags: a-new-class, a-new-gene, a-new-type, author-on-the, department, ireland, medical-physics, nature-medicine-, paper, physiology, professor-david, removes-it-from, researchers, using-drug-like | Comments Closed|

Researchers identify several gene mutations responsible for medulloblastoma

July 23rd, 2012

Researchers at the Stanford University School of Medicine and Lucile Packard Children’s Hospital have identified several gene mutations responsible for the most common childhood brain tumor, called

medulloblastoma, adding evidence to the theory that the diagnosis is a group of genetically distinct cancers with different prognoses. These and accompanying findings are likely to lead to less-toxic, better-targeted treatment approaches over the next two years, the researchers said.

“We tend to treat all medulloblastomas as one disease without taking into account how heterogeneous the tumors are at the molecular level,” said Yoon-Jae Cho, MD, an assistant professor of neurology and neurological sciences at Stanford, a pediatric neurologist at Packard Children’s and the senior author of the new research. “This paper represents a finer-grained view of the genetic landscape of these tumors and provides us with some leads on how to develop new therapies.”

The research, which will appear online in Nature July 22, is part of a large, ongoing effort to characterize genetic errors in medulloblastoma. Two companion studies on which Cho is a co-author will be published simultaneously with his paper. The three papers came from a consortium that involves scientists at Stanford, Packard Children’s, the Broad Institute, Children’s Hospital Boston, the Dana-Farber Cancer Institute, the German Cancer Research Center, Brandeis University and the Hospital for Sick Children in Toronto.

Current treatment for medulloblastoma, which originates in the cerebellum and affects about 250 U.S. children each year, begins with surgery to remove as much of the tumor as possible. Patients then receive a combination of radiation and chemotherapy, but the treatments are not tailored to the tumor’s genetic characteristics.

Cho’s team extracted DNA from 92 medulloblastoma tumors and compared it with DNA from matched blood samples from the same patients, uncovering 12 significant “point mutations” – single-letter errors in the genetic code – that occurred frequently in the brain cancer. A handful of the mutations had been previously identified in smaller studies of medulloblastoma, but several mutations were novel in both medulloblastoma and in cancer.

Among the newly identified mutations was one in an RNA helicase gene, DDX3X, which Cho said is the second-most common mutation in medulloblastoma tumors. “Mutations in this gene have now also been identified in other tumor types, such as chronic lymphocytic leukemia, and head and neck tumors,” he said.

However, the researchers found that it was rare for the same gene mutated in several different patients’ tumors. More commonly, mutations involving a set of genes regulating a single biological pathway were found in the tumors – a pattern that is emerging across cancer genome sequencing efforts.

Go here to read the rest:
Researchers identify several gene mutations responsible for medulloblastoma

Tags: broad-institute, children, dna, medicine, nature, paper, research, researchers, same, school, stanford, theory, tumor, tumors | Comments Closed|

Genetic mutations that cause common childhood brain tumors identified

July 23rd, 2012

ScienceDaily (July 22, 2012) Researchers at the Stanford University School of Medicine and Lucile Packard Children’s Hospital have identified several gene mutations responsible for the most common childhood brain tumor, called medulloblastoma, adding evidence to the theory that the diagnosis is a group of genetically distinct cancers with different prognoses. These and accompanying findings are likely to lead to less-toxic, better-targeted treatment approaches over the next two years, the researchers said.

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However, the researchers found that it was rare for the same gene mutated in several different patients' tumors. More commonly, mutations involving a set of genes regulating a single biological pathway were found in the tumors — a pattern that is emerging across cancer genome sequencing efforts.

Though no single tumor in the study carried all 12 mutations, the researchers were able to categorize the tumors according to which mutations they possessed. "We now understand that there are certain tumors with particular genetic signatures that are really resistant to standard treatments," Cho said. Children with medulloblastoma do not routinely have their tumors' genetic signatures characterized, but Cho believes that such characterization coupled with targeted therapies could greatly enhance tumor treatment.

About two-thirds of medulloblastoma patients now survive five years past diagnosis, but many survivors suffer lasting physical or intellectual side effects from their cancer treatments. Drugs tailored to a tumor's genetic profile have the potential to save more patients while reducing side effects, Cho said.

Several of the mutations discovered affect cellular signals that switch large groups of genes on and off. "The dysregulation of these 'epigenetic programs' is becoming a common theme not only in medulloblastoma but across cancer," Cho said. Such pathways may be good targets for cancer drugs; indeed, drugs targeting one such pathway (histone methyltransferases) are currently in pre-clinical development, while agents against another pathway (Hedgehog signaling pathway) are entering phase-2 clinical trials for medulloblastoma.

View original post here:
Genetic mutations that cause common childhood brain tumors identified

Tags: dna, genetic, hospital-boston, researchers, school, science, study, theory, tumor, tumors | Comments Closed|

Matching Cancer Drugs with Gene Targets

July 20th, 2012

With the new tools, researchers can compare patterns of drug activity and gene expression, not only to each other but also to other patterns of interest.

The newly updated software, called CellMiner, was built for use with the NCI-60, one of the most widely utilized collections of cancer cell samples employed in the testing of potential anti-cancer drugs. The tools, available free, provide rapid access to data from 22,379 genes catalogued in the NCI-60 and from 20,503 previously analyzed chemical compounds, including 102 U.S. Food and Drug Administration-approved drugs.

The study, written by the scientists that developed the tools at the National Cancer Institute (NCI), part of the National Institutes of Health, appeared in the July 16, 2012, issue of Cancer Research.

"Previously you would have to hire a bioinformatics team to sort through all of the data, but these tools put the entire database at the fingertips of any researcher," explained Yves Pommier, M.D., Ph.D., of the NCI's Center for Cancer Research. "These tools allow researchers to analyze drug responses as well as make comparisons from drug to drug and gene to gene."

Genomic sequencing and analysis have become increasingly important in biomedicine, but they are yielding data sets so vast that researchers may find it difficult to access and compare them. As new technologies emerge and more data are generated, tools to facilitate the comparative study of genes and potentially promising drugs will be of even greater importance. With the new tools, researchers can compare patterns of drug activity and gene expression, not only to each other but also to other patterns of interest. CellMiner allows the input of large quantities of genomic and drug data, calculates correlations between genes and drug activity profiles, and identifies correlations that are statistically significant. Its data integration capacities are easier, faster, and more flexible than other available methods, and these tools can be adapted for use with other collections of data.

Researchers looking at a particular drug can use the tools to access data from previous experiments done on that drug and analyze how the drug relates to other drugs and various gene profiles. As a case example for this study, the researchers compared drug activity levels and gene expression patterns from previous research to identify an investigational compound, called NSC732298, which is not currently being studied for colon cancer, but could be a potential therapy for the disease based on a CellMiner gene-drug match. In the same exercise, the researchers were able to identify that a second investigational drug that is being tested for colon cancer, called selumetinib, might also be effective against melanoma.

“We’re looking forward to seeing how other people are going to use this tool to look at gene co-regulation, regulation of gene expression, and the relationship between gene expression and cancer,” said Pommier.

This work was supported by NCI’s Center for Cancer Research and Division of Cancer Treatment and Diagnosis under intramural project number ZIA BC 006150.

Related Tool Link http://discover.nci.nih.gov/cellminer

Read the original here:
Matching Cancer Drugs with Gene Targets

Tags: a-are-generated, drugs, july, people--, related, researchers, scientists, study, tool-, work | Comments Closed|

New gene mutations linked to ALS and nerve cell growth dysfunction

July 15th, 2012

Public release date: 15-Jul-2012 [| E-mail | Share]

Contact: NINDS press team nindspressteam@ninds.nih.gov 301-496-5751 NIH/National Institute of Neurological Disorders and Stroke

Researchers have linked newly discovered gene mutations to some cases of the progressive fatal neurological disease amyotrophic lateral sclerosis ALS, also known as Lou Gehrig’s disease. Shedding light on how ALS destroys the cells and leads to paralysis, the researchers found that mutations in this gene affect the structure and growth of nerve cells.

ALS attacks motor neurons, the nerve cells responsible for controlling muscles. People with ALS experience such early symptoms as limb weakness or swallowing difficulties. In most people, the disease leads to death three to five years after symptoms develop, usually as a result of respiratory failure.

Scientists at the University of Massachusetts Medical School, Worcester, collaborated with international ALS researchers to search for gene mutations in two large families with an inherited form of ALS. The researchers used a technique to decode only the protein-encoding portions of DNA, known as the exome, allowing an efficient yet thorough search of the DNA regions most likely to contain disease-causing mutations. This deep sequencing of the exome led to the identification of several different mutations in the gene for profilin (PFN1) which were present only in the family members that developed ALS. Further investigations of 272 other familial ALS cases across the world showed that profilin mutations were also found in a small subset (about 1 to 2 percent) of the familial ALS cases studied.

The protein profilin is a key part of the creation and remodeling of a nerve cell’s scaffolding or cytoskeleton. In fly models, disrupting profilin stunts the growth of axons the long cell projections used to relay signals from one neuron to the next or from motor neurons to muscle cells. After identifying the PFN1 mutations in ALS patients,

the researchers demonstrated that these mutations inhibited axon growth in laboratory-grown motor neurons as well. They also found that mutant profilin accumulated in clumps in neural cells, as has been seen for other abnormal proteins associated with ALS, Parkinson's and Alzheimer's. Neural cells with PFN1 mutations also contained clumps of a protein known as TDP-43. Clumps of abnormal TDP-43 are found in most cases of ALS, further linking profilin to known ALS mechanisms.

John Landers, Ph.D., associate professor of neurology at the University of Massachusetts Medical School, described how studying ALS in large families is challenging. "ALS is a late-onset, rapidly progressive disease. Unless you've been following a family for decades, it is hard to get DNA samples to study," Dr. Landers said. "We were very fortunate to obtain the DNA samples with the help of our research collaborators and the affected families."

Over a dozen genes have been linked to ALS, and these findings support existing studies which suggest that cell cytoskeleton disruptions play a major role in ALS and other motor neuron diseases. Motor neurons are large cells with long axons that connect to muscle, and cytoskeleton proteins are especially important in the transport of proteins along the axon to the remote parts of the neuron. This information could be useful in developing strategies for detection and treatment of ALS.

"In all of the causative genes that we identify, we look for common pathways," Dr. Landers said. "Every time we are able to identify a new gene, we have another piece of the puzzle. Each one of these genes helps us to understand what's going on. The more of these we can find, the more we're going to know about what's going wrong in ALS."

Familial ALS accounts for 10 percent of all ALS cases, but the majority of ALS cases are sporadic, where the cause is unknown. Even though this new mutation is linked to familial ALS, it reveals information about the mechanisms underlying motor neuron degeneration in general, and also may have broader implications for understanding sporadic ALS.

See the rest here:
New gene mutations linked to ALS and nerve cell growth dysfunction

Tags: a-major-role, a-protein-known, a-small-subset, exome, family, gene, identification, information, researchers, scientists, university | Comments Closed|

Alzheimer's Research Uncovers New Gene Mutation

July 13th, 2012

July 12, 2012

By Christy Lewis

MEDFORD, Ore. — After years of fighting Alzheimer's disease, researchers spotted a gene that could change the future of Alzheimer's treatment.

Here are the basics of it all: researchers found a rare gene mutation that protects and stops the build up of a protein called beta amyloid. The production of the amyloids has been a possible cause for the disease, but this new discovery confirms that idea.

Not only does this discovery demonstrate a way to slow the symptoms of Alzheimer's, but it also verifies everything researchers have been working on. It means they're moving in the right direction toward better Alzheimer's treatment.

The gene could potentially lead to successfully preventing the disease or slowing down the production of the amyloid proteins. But the discovery doesn't provide help for those who already suffer from the disease right now.

Researchers say it could be years before anyone reaps the benefits of this discovery, but it certainly provides a positive outlook down the road for folks who'll likely get the disease.

Read the original:

Alzheimer's Research Uncovers New Gene Mutation

Tags: a-rare-gene, also-verifies, alzheimer, benefits, beta-amyloid-, discovery, disease, new-discovery, preventing-the, protein-called, researchers, stops-the-build, symptoms, the-production | Comments Closed|

Genetic mutation may protect against Alzheimer's

July 13th, 2012

Some drugs in development target what scientists have thought is the root cause of Alzheimer's – the buildup in the brain of amyloid protein. Newly published research supports such treatments.

Researchers have discovered for the first time a genetic mutation that may protect people against the degenerative brain disease that affects almost 30 million people worldwide.

Results from key Alzheimer's studies could decide future of treatment Watch: An Alzheimer's researcher who is also a patient

In the U.S. there's about 5.4 million people with the disease, but barring a medical breakthrough, researchers estimate by 2050 that the number of people with Alzheimer's will grow to 16 million.

For the new research, published in the July 11 issue of Nature, a team of scientists from Iceland took a closer look at what's called amyloid-beta precursor protein (APP).

APP was discovered about 25 years ago in patients with rare inherited forms of Alzheimer's that is developed in middle age, reports Nature News. According to the researchers, APP breaks down into amyloid-beta, which shows up as plaques in the brain that are a telltale marker of the disease. Scientists have debated whether the plaque buildup contributes to causing the disease or is caused by Alzheimer's.

The scientists in the new study compared the complete genome sequences of nearly 1,800 elderly Icelanders with their medical histories and discovered a genetic mutation in a gene that produces APP. The mutation was found to slow plaque formation in the brain by 40 percent.

Looking for the mutation in 400,000 Scandinavians, the researchers estimated that people who possess the mutation are more than five times as likely to reach 85 without developing Alzheimer's disease. People without Alzheimer's who had the mutation were also less likely to experience cognitive decline that comes with aging, suggesting that memory loss with aging and Alzheimer's disease may share a root cause.

"Pathologists have always suspected that there was a substantial overlap between Alzheimer's disease and normal age-related changes," study author Kri Stefnsson, chief executive of deCODE Genetics in Reykjavik, Iceland, told Nature News.

Since drug companies are currently investigating drugs that target amyloid production, as CBS This Morning reported in May, the new study may come as a sigh of relief for many scientists.

The rest is here:
Genetic mutation may protect against Alzheimer's

Tags: a-closer-look, a-root-cause-, disease, genetics-, july, medical, morning, nature, nature-news, researchers, telltale-marker, the-researchers, times-as-likely | Comments Closed|

Skin cancer-promoting gene discovered

July 11th, 2012

London, July 11 (ANI): Researchers have discovered a gene that plays a central role in black skin cancer, also known as melanoma.

Read the original:
Skin cancer-promoting gene discovered

Tags: a-central-role, also-known, ani, black-skin, central-role, have-discovered, known-as-melanoma, researchers | Comments Closed|

Gene Discovered By Scientists Linked To Facial Abnormalities

July 10th, 2012

Editor's Choice Main Category: Genetics Article Date: 09 Jul 2012 – 12:00 PDT

Current ratings for: Gene Discovered By Scientists Linked To Facial Abnormalities

The finding was published in The American Journal of Human Genetics and was conducted by Dr. Hyung-Goo Kim, molecular geneticist at the Medical College of Georgia at Georgia Health Sciences University and his team.

The researchers discovered the PHG21A mutated gene in patients with Potocki-Shaffer syndrome, a rare disorder that can result in significant abnormalities, like a small head and chin as well as intellectual disability.

The researchers conducted experiments in zebrafish, which developed similar head and brain abnormalities to those found in humans and discovered that their findings were confirmed when they suppressed the PHF21A gene in zebrafish.

Dr. Kim explained:"With less PHF21A, brain cells died, so this gene must play a big role in neuron survival."

To reconfirm their finding, the team inserted the gene back into the malformed fish, which subsequently became normal. The gene was also found in the craniofacial area of normal mice. Even though it is impossible to cure humans just by re-inserting the normal gene as is possible in zebrafish, the researchers believe that their finding will, in the future, allow genetic screening and possibly early intervention during fetal development, as well as treatments to increase PHF21A levels. In addition, the finding provides more insight into a better understanding of face, skull and brain formation.

The team focused on the gene when they used a distinctive chromosomal break found in patients with Potocki-Shaffer syndrome as a starting point. Chromosomes, i.e. packages of DNA and protein, are not supposed to break. However, when they do, they can damage nearby genes. Co-author of the study, Dr. Lawrence C. Layman, who is Chief of the MCG Section of Reproductive Endocrinology, Infertility and Genetics, explained: "We call this breakpoint mapping and the breakpoint is where the trouble is."

Damaged genes can no longer retain their optimum function. In PHF21A's case for instance the functionality is reduced to about half of the norm.

Layman continues: "When you see the chromosome translocation, you don't know which gene is disrupted. You use the break as a focus then use a bunch of molecular techniques to zoom in on the gene."

Go here to read the rest:
Gene Discovered By Scientists Linked To Facial Abnormalities

Tags: a-big-role, a-focus-then, a-rare-disorder, finding, focused-on-the, functionality, gene, genetics-, georgia, georgia-health, medical-college, researchers, team | Comments Closed|

Sea urchins could contain the genetic key to curing some diseases

July 10th, 2012

(Phys.org) — The purple sea urchin could help develop cures for diseases such as Alzheimers and cancer, scientists at the University of St Andrews have discovered.

Creatures, such as the sea urchin and sponge, have been discovered to have a special genetic sequence previously only thought to be used by certain viruses.

Now these sea creatures could inform scientists how to produce a therapeutic response in our own cells.

This latest finding builds on the earlier discovery of a short genetic sequence (2A) caused by viruses which can be used to return cells to a stem-cell like state allowing them to be manipulated and used for special treatments.

Martin Ryan, Professor of Translational Virology at the University of St Andrews, was the key researcher in that discovery.

He said: You could put two or more different genes into one cell, but each individual gene would be expressed at very different levels.

This process allows you to daisy-chain multiple genes into a single gene, but the different proteins made from each part of the new gene are expressed at the same level and within the same cell – which is a massive step forward.

This sequence was first discovered in Foot-and-Mouth Disease Virus, but we now know it is found in many other types of virus. This sequence has been used (by other researchers) in human gene therapy clinical trials to treat a number of cancers: metastatic melanoma, for example. It has also been used to produce human pluripotent stem cells a very important step in regenerative medicine, a treatment in which damaged tissues can be replaced.

It is now possible to take cells from a patient and drive them back into a stem cell state. These patient-specific stem cells could be used to treat a very wide range of diseases – Parkinsons Disease, Alzheimers, heart disease among others.

Prof Ryan added: Since our initial discovery, over the last four or five years the use of this sequence has gone through the roof. There have been more than 560 academic papers published using this new biotechnology.

Read more:
Sea urchins could contain the genetic key to curing some diseases

Tags: a-massive-step, a-patient-and, a-stem-cell, a-urchin-and, a-very-wide, alzheimers, individual-gene, mouth-disease, professor, researchers, same, sequence-, special-genetic, virology-at-the, within-the-same | Comments Closed|

Smoking linked to ectopic pregnancy

July 8th, 2012

ISTANBUL, Turkey, July 7 (UPI) — Cigarette smoke reduces the production of a Fallopian tube gene, which helps explain the link between smoking and ectopic pregnancy, Scottish researchers say.

Drs. Andrew Horne and Colin Duncan of the Medical Research Council Centre for Reproductive Health in Edinburgh, Scotland, said ectopic pregnancy — when the embryo implants in the Fallopian tube — is the most common cause of maternal death in early pregnancy.

Ectopic pregnancy occurs in up to 2 percent of all pregnancies.There is no way to prevent the condition, which must be treated by abdominal surgery or, if the ectopic is small and stable, by injection of a drug called methotrexate.

Horne and colleagues exposed cells from the Fallopian tube to a breakdown product of nicotine — cotinine. They then showed that cotinine had a negative effect on genes known to be associated with cell death, or apoptosis, and in particular with a particular gene.

In a further study the researchers showed that the gene's reduced production in the Fallopian tube of women who were smokers.

"The research is exciting because it provides new scientific evidence to help understand why women who smoke are more likely to have ectopic pregnancies," Horne said. "It appears that smoking reduces the production of genes, which are involved in the control of cell death and promote an environment in the Fallopian tube which is attractive to the developing embryo."

The findings were presented at the European Society of Human Reproduction and Embryology in Istanbul,

Turkey.

See the original post here:
Smoking linked to ectopic pregnancy

Tags: a-drug-called, a-further-study, andrew-horne, associated-with, council, developing, ectopic, european, horne, medical, production, researchers, scotland, study, turkey | Comments Closed|

Genetic 911: Study examines how cells exploit gene sequences to cope with toxic stress

July 4th, 2012

Toxic chemicals wreak havoc on cells, damaging DNA and other critical molecules. A new study from researchers at MIT and the University at Albany reveals how a molecular emergency-response system shifts the cell into damage-control mode and helps it survive such attacks by rapidly producing proteins that counteract the harm.

Peter Dedon, a professor of biological engineering at MIT, and colleagues had previously shown that cells treated with poisons such as arsenic alter their chemical modification of molecules known as transfer RNA (tRNA), which deliver protein building blocks within a cell. In their new paper, appearing in the July 3 issue of Nature Communications, the research team delved into how these modifications help cells survive.

The researchers found that toxic stresses reprogram the tRNA modifications to turn on a system that diverts the cell's protein-building machinery away from its routine activities to emergency action. "In the end, a stepwise mechanism leads to selective expression of proteins that you need to survive," says Dedon, senior author of the Nature Communications paper.

The findings offer insight into not only cells' response to toxins, but also their reactions to all kinds of stimuli, such as nutrients or hormones, Dedon says. "We're proposing that any time there's a stimulus, you're going to have a reprogramming [of tRNA] that causes selective translation of proteins you need for the next step in whatever you're going to do," he says.

Lead author of the paper is recent MIT PhD recipient Clement Chan. Other MIT authors are postdocs Yan Ling Joy Pang and Wenjun Deng and research scientist Ramesh Indrakanti. Authors from the University at Albany are Thomas Begley, an associate professor of nanobioscience, and research scientist Madhu Dyavaiah.

A new role for RNA

Transfer RNA is made of 70 to 90 ribonucleotide building blocks. After synthesis, the ribonucleotides usually undergo dozens of chemical modifications that alter their structure and function. The primary job of tRNA is to bring amino acids to the ribosomes, which string them together to make proteins.

In a 2010 paper, Dedon and colleagues exposed yeast cells to different toxic chemicals, including hydrogen peroxide, bleach and arsenic. In each case, the cells responded by uniquely reprogramming the location and amount of each tRNA modification. If the cells lost the ability to reprogram the modifications, they were much less likely to survive the toxic attack.

In the new study, the researchers focused on a particular tRNA modification, known as m5C, which occurs when cells encounter hydrogen peroxide, a chemical produced by white blood cells.

They first discovered that this modification occurs predominantly in one of the tRNAs that carry the amino acid leucine. Every amino acid is encoded by three-letter sequences in the genome called codons. Each tRNA corresponds to one amino acid, but most amino acids can be coded by several tRNA sequences. For example, leucine can be coded by six different genome sequences: TTA, TTG, CTT, CTC, CTA and CTG.

Read more:
Genetic 911: Study examines how cells exploit gene sequences to cope with toxic stress

Tags: cell, cells, clement-chan-, dna, havoc-on-cells, july, madhu-dyavaiah-, modification, modifications,

researchers, ribonucleotides, such-as-arsenic, university | Comments Closed|

Genetic 911: Cells' emergency systems revealed

July 4th, 2012

ScienceDaily (July 3, 2012) oxic chemicals wreak havoc on cells, damaging DNA and other critical molecules. A new study from researchers at MIT and the University at Albany reveals how a molecular emergency-response system shifts the cell into damage-control mode and helps it survive such attacks by rapidly producing proteins that counteract the harm.

Peter Dedon, a professor of biological engineering at MIT, and colleagues had previously shown that cells treated with poisons such as arsenic alter their chemical modification of molecules known as transfer RNA (tRNA), which deliver protein building blocks within a cell. In their new paper, appearing in the July 3 issue of Nature Communications, the research team delved into how these modifications help cells survive.

The researchers found that toxic stresses reprogram the tRNA modifications to turn on a system that diverts the cell's protein-building machinery away from its routine activities to emergency action. "In the end, a stepwise mechanism leads to selective expression of proteins that you need to survive," says Dedon, senior author of the Nature Communications paper.

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Continued here:
Genetic 911: Cells' emergency systems revealed

Tags: a-2010-paper, carry-the-amino, location, madhu-dyavaiah-, modification, researchers, such-as-arsenic, thomas-begley | Comments Closed|

Lotion May Treat Skin Diseases

July 3rd, 2012

Imagine a lotion that can treat irreversible genetic skin diseases like psoriasis or life-threatening skin cancers like

melanoma. Researchers at Northwestern University say they're another step closer to creating a treatment that will naturally slip through the skin and genetically alter cells to treat a particular skin disease.

Using creams and lotions to target a particular problem area is seen as a great advantage among many dermatologists in treating a localized skin problem.

"We like to treat skin diseases with topical creams so that we avoid side effects from treatments taken by mouth or injected," said Dr. Amy Paller, chair of dermatology and professor of pediatrics at Northwestern University Feinberg School of Medicine.

But the difficulty among researchers has been creating a gene-altering topical agent that can successfully penetrate the skin to specifically treat genetic skin diseases.

"The problem is that our skin is a formidable barrier," Paller said. "Genetic material can't get through the skin through regular means."

Using nanotechnology, the researchers packaged gene-altering structures on top of tiny particles of gold designed to target epidermal growth factor receptor, a genetic marker associated with many types of skin cancers. The structure is designed to sneak through the skin and latch onto targets underneath without eliciting an immune response.

The researchers mixed the structure into the ointment Aquaphor, which is commonly used among many patients who have dry skin or irritation.

The researchers then rubbed the ointment onto the mice and onto human skin tissue and saw the gene-altering structure in the lotion successfully penetrated the skin and was able to shut down the potentially cancer-causing protein, according to the findings published Monday in the journal Proceedings of the National Academy of Sciences.

The preliminary study is regarded as the first to deliver topical gene therapy effectively with no toxic effects.

Topical steroids are the most commonly used cream-based treatment for skin problems such as psoriasis. While they can treat inflammation or other effects of a skin disease, they do not treat the underlying mechanism that's causing the problem, Paller said. And in cases like melanoma, the diseased cells are often surgically removed from the skin, leaving scarring.

Read more:
Lotion May Treat Skin Diseases

Tags: commonly-used, feinberg-school, from-the-skin, journal, national, ointment, regarded-as-the, researchers, skin, structure, the-researchers, through-regular, underlying | Comments Closed|

Gene Mutations Associated With Enlarged Brain Size, Disorders

July 2nd, 2012

July 2, 2012

Lawrence LeBlond for redOrbit.com Your Universe Online

At least three genetic mutations found in the human brain have been linked to enlarged brain size (megalencephaly) and a number of disorders, including cancer, epilepsy and autism, according to new research led by Seattle Childrens Research Institute.

The mutations were found in the genes AKT3, PIK3R2 and PIK3CA. The mutations were also linked to vascular disorders and skin growth disorders, said the researchers. The study, published in the online edition of the journal Nature Genetics on June 24, offers important implications for the future of medicine through the research findings.

Study leaders, geneticist William Dobyns, MD, and Jean-Baptiste Rivire, PhD, discovered through their research

additional proof that the genetic makeup of a person is not completely determined at the moment of conception. The new evidence ties in with previous research that recognized that genetic changes can occur after conception, although considered quite rare.

The researchers also discovered the genetic causes of these human diseases, including developmental disorders, may also directly lead to new possibilities for treatment.

AKT3, PIK3R2 and PIK3CA are found in all humans, but only when they are mutated do they lead to the diseases and disorders. PIK3CA is known as a cancer-related gene, and appears to make cancer more aggressive. Boston Childrens Hospital researchers recently found a common link between the PIK3CA gene and a rare condition known as CLOVES syndrome.

James Olson, MD, PhD, a pediatric cancer expert at Seattle Childrens and Fred Hutchinson Cancer Research Center acknowledged the two decades-worth of work that led to the findings.

This study represents ideal integration of clinical medicine and cutting-edge genomics, said Olson, who was not involved in the latest research. I hope and believe that the research will establish a foundation for successfully using drugs that were originally developed to treat cancer in a way that helps normalize intellectual and physical development of affected children.

He noted that the team did an excellent job by deep sequencing exceptionally rare familial cases and unrelated cases to identify the culprit pathway. He further noted that the three genes all encode core components of the phosphatidylinositol-3-kinase/AKT pathway, the culprit pathway, as referenced by his work.

Follow this link:
Gene Mutations Associated With Enlarged Brain Size, Disorders

Tags: culprit, diseases, genetic, known-as-cloves, nature, researchers, universe, work | Comments Closed|

Stem-cell research leaders to meet in NUIG

July 2nd, 2012

The Irish Times – Monday, July 2, 2012

LORNA SIGGINS

WORLD leaders in stem-cell technology are due to exchange knowledge of potential treatments at a conference opening in NUI Galway today.

Researchers from NUIG, University College Cork and NUI Maynooth will participate in the event, which has been billed as the first major conference on stem-cell therapy in Ireland.

Prof Anthony Hollander of the University of Bristol, England who was one of a team which successful created and then transplanted the first tissue-engineered trachea or windpipe is among a number of international speakers presenting findings.

The gathering will focus on the realities of stem-cell treatment, Prof Frank Barry, director of NUIGs National Centre for Biomedical Engineering Science has said.

The therapy is complex and controversial, and sometimes exaggerated claims are made, he said.

The researchers are specialists in Mesenchymal, or adult, stem cells, and will be concentrating on what is likely in the future, he added.

The list of conditions which could be treated successfully by stem cells is small, but growing, Prof Barry said.

Leukaemia and other diseases of the blood appear to respond best.

Read this article:

Stem-cell research leaders to meet in NUIG

Tags: billed-as-the, bristol, college-cork, diseases, first, focus-on-the, gathering-will, irish, irish-times, national-centre, realities--, researchers, science | Comments Closed|

Gene therapy for smoking kills pleasure of nicotine

July 1st, 2012

By Jon Bardin, Los Angeles Times / For the Booster Shots blog 7:01 p.m. EST, June 29, 2012

A new vaccine may help prevent the brain stimulation that keeps smokers from being able to quit. (Francine Orr / Los Angeles Times / Jun 29, 2012)

Cant kick cigarettes? A vaccine may one day help by preventing nicotine from reaching its target in the brain, according to research published this week.

Most smoking therapies do a poor job of stopping the habit 70% to 80% of smokers who use an approved drug therapy to quit relapse. Scientists say this is because the targets of existing therapies are imperfect, only slightly weakening nicotines ability to find its target in the brain.

So some scientists have been trying a different approach creation of a vaccine. It would work like this: People would inject the vaccine like a shot, and the vaccine would create nicotine antibodies, molecules that can snatch up nicotine from the bloodstream before it reaches the brain. The vaccine could be used by smokers who want to quit or people who are worried about getting addicted to cigarettes in the future.

Researchers have tried to create vaccines in the past, but the ones theyve come up with have not been particularly effective. The authors of the new study say this may be because previous vaccines just didnt create enough antibodies to get rid of all the nicotine.

The new report, published in the journal Science Translational Medicine, attempts to solve this problem via gene therapy, in which a new gene is inserted into the body to do a particular job.

First the scientists at Weill Cornell Medical College in New York City put a gene that produces a nicotine antibody into mice. The gene was taken into the mices livers, and the liver started producing the antibody. Once produced, the antibody connected with nicotine, trapping it and preventing it from making its way to the brain, where it would otherwise have caused the pleasurable, addictive effects it is so known for.

Because of this trick, the researchers say that the new vaccine should only have to be injected once, and it will work for life, continuing to produce new antibodies in the liver.

The vaccine was effective: When mice were given nicotine intravenously, ones with the vaccine had a 47-fold drop in levels of nicotine in the blood compared with ones that hadnt received the vaccine. The antibody had successfully captured the nicotine in the bloodstream before it could reach the brain.

Continued here:
Gene therapy for smoking kills pleasure of nicotine

Tags: a-47-fold-drop, bloodstream, journal, liver, medical-college, medicine, researchers, science, scientists, trapping-it-and, vaccine, weill-cornell | Comments Closed|

Stem Cells From Muscular Dystrophy Patients Transplanted Into Mice

June 30th, 2012

Editor’s Choice Main Category: Muscular Dystrophy / ALS Also Included In: Transplants / Organ Donations
Article Date: 29 Jun 2012 – 11:00 PDT

Current ratings for: Stem Cells From Muscular Dystrophy Patients Transplanted Into Mice

A new study published in Science Translational Medicine reveals that researchers have, for the first time, managed to turn fibroblast cells, i.e. common cells within connective tissue, from muscular dystrophy patients into stem cells and subsequently changed these cells into muscle precursor cells. After modifying the muscle precursor cells genetically, the researchers transplanted them into mice.

In future, this new technique could be used in order to treat patients with the rare condition of limb-girdle muscular dystrophy, which primarily affects the shoulders and hips, and maybe other types of muscular dystrophies. The method was initially developed in Milan at the San Raffaele Scientific Institute and was completed at UCL.

Muscular dystrophy is a genetic disorder, which typically affects skeletal muscles. The condition leads to severely impaired mobility and can, in severe cases result in respiratory and cardiac dysfunction. At present, there is no effective treatment for the condition. A number of new potential therapies, including cell therapy, are entering clinical trials.

The scientists of this study concentrated their research on genetically modifying mesoangioblasts, i.e. a self-renewing cell that originates from the dorsal aorta and differentiates into most mesodermal tissues, which demonstrated its potential for treating muscular dystrophy in earlier studies.

Given that the muscles of patients with muscular dystrophy are depleted of mesonangioblasts, the researchers were unable to obtain sufficient numbers of these cells from patients with limb-girdle muscular dystrophy, and therefore “reprogrammed” adult cells from these patients into stem cells, which enabled them to prompt them to differentiate into mesoangioblast-like cells.The team then genetically corrected these ‘progenitor’ cells by using a viral vector, and injected them into mice with muscular dystrophy so that the cells targeted damaged muscle fibers.

In a mice study, the same process demonstrated that dystrophic mice were able to run on a treadmill for longer a longer time than dystrophic mice that did not receive the cells.

Research leader, Dr Francesco Saverio Tedesco, from UCL Cell & Developmental Biology, who led the study, explained:

Professor Giulio Cossu, also an author at UCL, concluded:

Read the original:
Stem Cells From Muscular Dystrophy Patients Transplanted Into Mice

Tags: a-mice-study, a-treadmill-for, a-viral-vector, cells, condition-leads, from-the-dorsal, muscular, muscular-dystrophy, research, researchers | Comments Closed|

A tomato by any other gene: Just as sweet?

June 29th, 2012

New research shows that the bland flavor of a popular variety of firm tomatoes is caused by a genetic switch. Locating this switch may enable scientists to create good-tasting and good-shipping tomatoes in the future.

Using genetics, scientists have been able to dig up the dirt on why homegrowntomatoes taste so much sweeter than the ones in the supermarket.

Researchers found a genetic switch responsible for some of the sugar production within atomato. A new study in Friday’s edition of Science found that the common type oftomatobred for firmness and good shipping also inadvertently turns off the sugar-producing switch. That makes it less sweet and blander than garden varieties.

University of California Davis plant scientist Ann Powell said knowing the genetics behind the sugar-making could lead someday to development of sweetertomatoesthat also travel well.

View original post here:

A tomato by any other gene: Just as sweet?

Tags: dirt-on-why, genetics-, makes-it-less, popular-variety, research-shows, researchers, said-knowing, science, shipping-also, supermarket, turns-off-the, university | Comments Closed|

A tomato by any other gene: Just as sweet? (+video)

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Tags: california, firm-tomatoes, genetic-switch, genetics-, makes-it-less, popular-variety, researchers, science, shipping-also, sugar, supermarket, turns-off-the, university | Comments Closed|

Biology's Master Programmers

June 29th, 2012

Photographs by Mark Ostow

George Church is an imposing figureover six feet tall, with a large, rectangular face bordered by a brown and silver nest of beard and topped by a thick mop of hair. Since the mid-1980s Church has played a pioneering role in the development of DNA sequencing, helpingamong his other achievementsto organize the Human Genome Project. To reach his office at Harvard Medical School, one enters a laboratory humming with many of the more than 50 graduate students and postdoctoral fellows over whom Church rules as director of the school's Center for Computational Genetics. Passing through an anteroom of assistants, I find Church at his desk, his back to me, hunched over a notebook computer that makes him look even larger than he is.

Church looms especially large these days because of his role as one of the most influential figures in synthetic biology, an ambitious and radical approach to genetic engineering that attempts to create novel biological entitieseverything from enzymes to cells and microbesby combining the expertise of biology and engineering. He and his lab are credited with many of the advances in harnessing and synthesizing DNA that now help other researchers modify microrganisms to create new fuels and medical treatments. When I ask Church to describe what tangible impact synthetic biology will have on everyday life, he leans back in his chair, clasps his hands behind his head, and says, "It will change everything. People are going to live healthier a lot longer because of synthetic biology. You can count on it."

Such grandiosity is not uncommon among the practitioners of synthetic biology. Ever since Church and a few other researchers began to combine biology and engineering a dozen years ago, they have promised it would "change everything." And no wonder. The very idea of synthetic biology is to purposefully engineer the DNA of living things so that they can accomplish tasks they don't carry out in nature. Although genetic engineering has been going on since the 1970s, a rapid drop in the cost of decoding and synthesizing DNA, combined with a vast increase in computer power and an influx into biology labs of engineers and computer scientists, has led to a fundamental change in how thoroughly and swiftly an organism's genetics can be modified. Church says the technology will eventually lead to all manner of breakthroughs: we will be able to replace diseased tissues and organs by reprogramming cells to make new ones, create novel microbes that efficiently secrete fuels and other

chemicals, and fashion DNA switches that turn on the right genes inside a patient's cells to prevent arteries from getting clogged.

Even though some of these applications are years from reality, Church has a way of tossing off such predictions matter-of-factly. And it's easy to see why he's optimistic. The cost of both decoding DNA and synthesizing new DNA strands, he has calculated, is falling about five times as fast as computing power is increasing under Moore's Law, which has accurately predicted that chip performance will double roughly every two years. Those involved in synthetic biology, who often favor computer analogies, might say it's becoming exponentially easier to read from, and write into, the source code of life. These underlying technology trends, says Church, are leading to an explosion in experimentation of a sort that would have been inconceivable only a few years ago.

Up to now, it's proved stubbornly difficult to turn synthetic biology into a practical technology that can create products like cheap biofuels. Scientists have found that the "code of life" is far more complex and difficult to crack than anyone might have imagined a decade ago. What's more, while rewriting the code is easier than ever, getting it right isn't. Researchers and entrepreneurs have found ways to coax bacteria or yeast to make many useful compounds, but it has been difficult to optimize such processes so that the microbes produce significant quantities efficiently enough to compete with existing commercial products.

Church is characteristically undeterred. At 57, he has survived cancer and a heart attack, and he suffers from both dyslexia and narcolepsy; before I visited him, one of his colleagues warned that I shouldn't be surprised if he fell asleep on me. But he has founded or taken an advisory role in more than 50 startup companiesand he stayed awake throughout our time together, apparently excited to describe how his lab has found ways to take advantage of ultrafast sequencing and other tools to greatly speed up synthetic biology. Among its many projects, Church's lab has invented a technique for rapidly synthesizing multiple novel strings of DNA and introducing them simultaneously into a bacterial genome. In one experiment, researchers created four billion variants of E. coli in a single day. After three days, they found variants of the bacteria in which production of a desired chemical was increased fivefold.

The idea, Church explains, is to sort through the variations to find "an occasional hopeful monster, just as evolution has done for millions of years." By mimicking in lab experiments what takes eons in nature, he says, he is radically improving the odds of finding ways to make microbes not just do new things but do them efficiently.

A DNA Turn-On

In some ways, the difficulties researchers have faced making new, more useful life forms shouldn't come as a surprise. Indeed, a lesson of genome research over the last few decades is that no matter how elegantly compact the DNA code is, the biology it gives rise to is consistently more complex than anyone anticipated. When I began reporting the early days of gene discovery 30 years ago, biologists, as they often do, thought reductively. When they found a gene involved in disease, the discovery made headlines. Scientists said they believed that potent new medicines could soon deactivate malfunctioning versions of genes, or that gene therapy could be used to replace them with healthy versions in the body.

Go here to see the original:
Biology's Master Programmers

Tags: a-decade-ago, a-dozen-years, a-heart-attack, a-single-day-, church, dna, office, researchers, school, technology-, time, tools | Comments Closed|

Researchers develop vaccine to treat nicotine addiction

June 28th, 2012

Researchers have developed a vaccine that successfully treated nicotine addiction in mice, according to a study published Wednesday in Science Translational Medicine.

With just a single dose, the vaccine protected mice against nicotine addiction for the rest of their lives, the researchers said. The vaccine works by prompting the animals liver to act as a factory that continually produces antibodies. The antibodies then absorb the nicotine as soon as it hits the bloodstream, preventing it from reaching the brain or the heart.

According to the studys lead investigator, Dr. Ronald Crystal, chairman and professor of Genetic Medicine at Weill Cornell Medical College, it normally takes nicotine about six to 10 seconds to cross the bloodstream, reach the brain and bind to receptors. This is what produces the calm or relaxed feelings that drive nicotine addiction. By blocking nicotine from reaching the brain, the antibodies prevent those pleasurable feelings from occurring.

“As far as we can see, the best way to treat chronic nicotine addiction from smoking is to have these Pacman-like antibodies on patrol, clearing the blood as needed before nicotine can have any biological effect,” Crystal said in a released statement.

Importantly, the vaccine allows the body to build up its own immunity against nicotine, making it more effective and consistent than vaccines developed in the past.

Crystal said previous nicotine vaccines likely failed because they directly injected nicotine antibodies into the body, rather than prompting the body to build its own antibodies. This meant these passive vaccines had to be injected multiple times, because they only lasted for three to four weeks, and the dosage level required may have varied from person to personparticularly if the person started smoking again.

On the other hand, the researchers knew the second main type of vaccines, known as active vaccines, wouldnt protect against nicotine addiction either. Active vaccinesused to protect people against viruses such as polio or the mumpswork by introducing a piece of a virus into the body, which in turn prompts the body to develop a lifelong immune response against the invading agent. However, nicotine molecules are too small for the immune system to recognize.

As a result, the researchers had to develop a third kind of vaccine: a genetic vaccine, which works by binding the genetic sequence of a nicotine antibody to a non-harmful virus. The virus is directed to go to the liver cells, and the genetic sequence of the antibody then inserts itself into those cells, causing the cells to produce a stream of the antibodies along with the other molecules they make.

We can target almost any organ [with this type of vaccine], but the reason for using the liver is that it is a very good secretory organ, Crystal told FoxNews.com. The liver is very good at making and secreting many proteins, so we just genetically modified the liver cells to also make antibodies against nicotine.

Crystal said he first thought of the concept behind the vaccine a few years ago while passing by a newsstand. I saw a magazine cover that said something along the lines of Addiction: We Need Vaccines and got this idea to use gene therapy.

See the original post:
Researchers develop vaccine to treat nicotine addiction

Tags: animals, body, brain, crystal, into-the-body, liver, medical-college, other, person, researchers, ronald-crystal, science, the-researchers, vaccine | Comments Closed|

Gene therapy curbs nicotine addiction in mice

June 28th, 2012

Forget patches: gene therapy could suppress cigarette cravings by preventing the brain from receiving nicotine. The treatment is effective in mice, but with gene therapy still not fully tested in people, human trials and treatments are a long way off.

For drug users who really can’t quit, vaccination might one day be an option, and several groups have attempted to develop such treatments.

But nicotine vaccines have mostly flopped. This is because nicotine is a very small molecule, so the immune system has difficulty recognising the drug and making antibodies that bind it. Physicians can inject antibodies directly into a patient, but this treatment quickly becomes expensive because the antibodies don’t last long.

Ronald Crystal of Weill Cornell Medical College in New York and his team decided to bypass that problem by putting the gene for a nicotine antibody right into the body.

They selected the strongest antibody against nicotine from a mouse and isolated the gene that produced it. They then placed this gene into a carrier called adeno-associated virus (AAV), which is widely used for gene therapy.

When the researchers injected the virus and its cargo into nicotine-addicted mice, the rodents' livers took up the virus, began making antibodies and pumped them into the bloodstream. The researchers injected two cigarettes' worth of nicotine into AAV-infected mice. The antibodies were able to bind 83 per cent of the drug before it reached the brain.

Without their drug, the mice's behaviour changed. Nicotine usually causes mice to "chill out", Crystal says, but the researchers found that the treated mice stayed active and their heart rates stayed normal when they received nicotine.

Eighteen weeks later, the mice's livers were still making the antibody, suggesting that the therapy might render nicotine useless to smokers for long periods.

Jude Samulski at the University of North Carolina at Chapel Hill, who was part of the team that developed AAV as a gene therapy vector, says he's "ecstatic" that the vector has come so far. He calls the research "a gorgeous piece of work" that has "leapfrogged" the difficulties faced by vaccines.

But he has doubts about whether gene therapy is well-tested enough to be used to treat nicotine addiction. So far, AAV has been clinically tested in people with HIV or terminal cancer where potential benefits far outweigh the risks. "It's ahead of its time. In 10 years there may be enough safety data," he says. "Quitting smoking might be easier."

Read the original here:
Gene therapy curbs nicotine addiction in mice

Tags: a-gene-therapy, brain, difficulties, drug, gene, mice, north, research, researchers, samulski-at-the, university | Comments Closed|

Gene Therapy Against Nicotine May Someday Help Smokers Quit

June 28th, 2012

By Elizabeth Lopatto – 2012-06-27T18:00:00Z

An experimental vaccine againstnicotine, delivered using gene therapy, prevents the substance from reaching the brain and may make quitting easier for smokers, a study using mice indicates.

A single dose of vaccine allowed the liver to produce antibodies that stopped most of the nicotine from getting to the brain, according to a study in the journal Science Translational Medicine. The concentration of nicotine in the brains of treated mice was just 15 percent of that in untreated ones.

Of the more than 4,000 chemicals in cigarette smoke, it is nicotine that leads to addiction, the researchers wrote. Keeping the substance away from the brain might stymie nicotines addictive power by preventing smokers from enjoying their cigarettes, giving them no incentive to relapse, said Ronald Crystal, one of the studys researchers.

This looks really terrific if youre a mouse, but the caveat is that they arent small humans, said Crystal, the chairman of genetic medicine at Weill Cornell Medical College in New York, in a telephone interview.

The gene therapy delivers the vaccine to the liver using a virus engineered not to be harmful. The gene sequence for the antibodies is inserted into liver cells, which then begin to create antibodies to nicotine.

The antibody is floating around like Pac-Man in the blood, Crystal said. If you give the nicotine and the anti-nicotine gobbles it up, it doesnt reach the brain.

The idea of vaccines against nicotine has emerged before, in the form of injections used to trigger an immune response. Those methods proved ineffective, according to the researchers. They turned to gene therapy to trigger production of antibodies.

About 20 percent of U.S. adults are smokers, and most relapse shortly after quitting.

We dont have very effective therapies, Crystal said. The problem is even with the drugs we have now, 70 percent of people go back to smoking within 6 months of trying to quit.

Follow this link:
Gene Therapy Against Nicotine May Someday Help Smokers Quit

Tags: chairman, cigarettes, crystal, from-the-brain, journal, medical, medical-college, researchers, substance, weill-cornell, with-the-drugs | Comments Closed|

ESCs and iPSCs demonstrate similar survival and neural differentiation capabilities

June 27th, 2012

Researchers in Japan who evaluated the risks and efficacy of transplanting two varieties of stem cells into mouse cochlea have concluded that both adult-derived induced pluripotent stem (iPS) cells and mouse embryonic stem (ES) cells demonstrate similar survival and neural differentiation capabilities. However, there is a risk of tumor growth associated with transplanting iPS cells into mouse cochleae. Given the potential for tumorigenesis, they concluded that the source of iPS cells is a critical issue for iPS cell-based therapy.

Their study is published in a recent issue of Cell Transplantation (21:4), now freely available on-line at http://www.ingentaconnect.com/content/cog/ct/,

“Hearing loss affects millions of people worldwide,” said Dr. Takayuki Nakagawa of the Department of Otolaryngology, Graduate School of Medicine, Kyoto University, Japan. “Recent studies have indicated the potential of stem-cell based approaches for the regeneration of hair cells and associated auditory primary neurons. These structures are essential for hearing and defects result in profound hearing loss and deafness.”

The authors noted that embryonic stem cells have previously been identified as promising candidates for transplantation, however they have also been associated with immune rejection and ethics issues. Consequently, this study compared the survival and neural differentiation capabilities of ES and three clones of mouse iPS cells.

“Our study examined using induced pluripotent stem cells generated from the patient source to determine if they offer a promising alternative to ES cells,” explained Dr. Nakagawa. “In addition, the potential for tumor risk from iPS cells needed clarification.”

Four weeks after transplantation, the researchers found that the majority of cochleae that had been transplanted exhibited the settlement of iPS or ES-derived neurons. However, there was a difference in the number of cells present based on cell lines. They noted that the number of cells able to be transplanted into cochleae is limited because of the cochleae’s tiny size. Thus, the number of settled cells is low.

More:
ESCs and iPSCs demonstrate similar survival and neural differentiation capabilities

Tags: based-on-cell, department, japan, medicine, mouse-cochleae-, number, otolaryngology, potential, researchers, settlement, source, stem-cell-based, study, the-potential, transplantation | Comments Closed|
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Gene discovery offers hope for new drug for epilepsy

Gabrielle Monaghan

[illegible] neuroscientists have discovered a gene linked to epilepsy that could respond to drug therapy to prevent seizures.

Scientists at the Royal College of Surgeons in Ireland (RCSI) claim research demonstrates how this gene can be targeted with drug-like molecules to lessen the brain's susceptibility to seizures and the frequency with which they occur.

At present, a third of the 40,000 people in Ireland with epilepsy continue to have seizures despite being prescribed medication. Along with the physical symptoms, more than half of Irish sufferers have experienced social stigma because of their condition, according to a survey released earlier this year by Epilepsy Ireland, Ireland's epilepsy association. Some two of these surveyed said they did not feel comfortable revealing their condition to employers and colleagues.

This identification of a new gene responsible for epilepsy was outlined in a study published in Nature Medicine, an international journal. The research was conducted by neuroscientists at the RCSI, with the help of clinicians at Beaumont Hospital in Dublin and brain structure experts at the Cajal Institute in Madrid.

The team investigated a new class of gene called "microRNA", which regulates key molecules that control protein production inside cells and is thought to be related to some neurological disorders. The researchers examined a specific molecule in the gene, known as "microRNA-134", and found that its levels were much higher in the part of the brain that causes seizures in patients with epilepsy.

As a result of this discovery, the scientists said a new type of drug-like molecule called an antagomir. This molecule appears to work by latching on to the microRNA-134 gene and silencing it from the brain cell. By using this strategy in mice, the researchers found they could prevent epileptic seizures from happening.

Eva Tiraboschi-Moreno, a researcher at the RCSI's department of physiology and medical physics, said: "Our research found the antagomir drug prevents the brain cells from toxic effects of prolonged seizures and the effects of the treatment can last up to one month."

David Greenfield, a professor at the same department, said there were up to 40% fewer epileptic seizures over two-week periods. Tests where the treatment was given months after the first drug therapy, there were the fewest seizures.