WEBVISION currently enjoys.

undoubtedly embrace the vision — which will improve in our parallel cortex. We look forward to this expansion — and parallel improve processes.

Now, the availability of a selective antagonist for the V1b receptor has allowed Griebel et al. to test this idea. They used a battery of mouse and rat models of anxiety and depression to test the effects of SSR149415. In ‘classical’ models of anxiety, such as the elevated plus-maze and the light/dark test (which measures how long mice spend in a lit box as opposed to a dark one), the new compound was less effective than the benzodiazepine anxiolytic diazepam. But in models of exposure to traumatic stress, such as the social-defeat paradigm (in which a mouse is exposed to aggression from a resident mouse in a test cage, which normally increases anxiety as assessed in the elevated plus-maze), SSR149415 had clear anxiolytic effects. The authors suggest that V1b receptor antagonists might be useful as a treatment for stress disorders that result from traumatic events, rather than for generalized anxiety disorder.

To test the antidepressant effects of SSR149415, Griebel and colleagues used two models of depression: the acute forced-swimming test, and the chronic mild-stress test (in which a mouse is exposed to a sequence of mildly stressful events, such as water deprivation and restraint, for several weeks, leading to a decline in grooming that is thought to parallel the reduction in personal hygiene in depressed humans). SSR149415 reduced all of the measures of ‘depression’ in these rodent models, which are normally good predictors of antidepressant efficacy in humans.

Although these results might be therapeutically useful, they do not tell us where in the brain SSR149415 acts to reduce anxiety or depression. However, the fact that it is still effective in hypophysectomized rats indicates that the effects do not depend on blocking the hypothalamic V1a receptors, and supports the idea that the receptors in limbic structures are more important for these effects.

PSYCHIATRIC DISORDERS

Less stress?

A potential new approach to the development of anxiolytic and antidepressant drugs might emerge from results showing that an antagonist of the vasopressin V1b receptor is effective in rodent models of both anxiety and depression. Griebel et al. tested the antagonist SSR149415 in a variety of rat and mouse models, and concluded that it shows both anxiolytic- and antidepressant-like properties.

Although arginine vasopressin (AVP) is produced in the hypothalamus and is involved in the regulation of the secretion of corticotropin by the pituitary gland, the presence of AVP-containing neurons that project to the limbic system, and of vasopressin receptors (V1a and V1b) in structures such as the septum and hippocampus, has led to the idea that AVP might also be important in emotional processes such as stress responses. In support of this, a mixed (V1a/b) peptide vasopressin receptor antagonist has anxiolytic effects in rats, and stress resulting from chronic immobilization increases the levels of V1b receptor messenger RNA. These results indicate a possible role for the V1b receptor in emotional processes.

WEBWATCH

Less stress? A VP might also be important in the development of the retina.

With these solid foundations in place, the authors have started to extend the site beyond the retina to include a section on the primary visual cortex. We look forward to this expansion — and parallel improvements in our understanding of human vision — which will undoubtedly embrace the high standards of quality that WEBVISION currently enjoys.

Juan Carlos López

NEURAL INDUCTION

Tempting fate

The central tenet of the default model of neural induction states that ectodermal cells are fated to become neural unless they are instructed by bone morphogenetic proteins (BMPs) to take on an epidermal fate. This indicates that there must be transcriptional repressors that act downstream of the BMPs to inhibit the expression of neural-specification genes, and Bakkers and colleagues have now identified a strong candidate for such a repressor. The zebrafish ΔNp63 protein is a homologue of the mammalian p63, which is a close relative of the tumour-suppressor protein p53. Alternative splicing generates at least six different isoforms of ΔNp63, all of which function as transcriptional activators or repressors. Mutation of p63 in mice revealed defects in epithelial development and indicated a possible role in the maintenance of epithelial stem cells, but these new studies in zebrafish imply a much earlier role in ectodermal cell-fate choice.

First, the authors looked for evidence that ΔNp63 acts downstream of BMP signalling. They showed that the promoter region of the ΔNp63 gene contains binding sites for the BMP-signalling mediators Smad4 and Smad5. They also showed that ΔNp63 expression could be upregulated by increasing the level of BMP signalling, an effect that was abolished if the Smad binding protein p53.

Next, the authors examined the role of ΔNp63 in dorsoventral patterning. In the gastrulating zebrafish embryo, ΔNp63 transcripts are confined to the ventral ectoderm, which gives rise to epidermis. Bakkers et al. inactivated ΔNp63 at this stage of development using antisense oligonucleotides. In the resulting embryos, the neuroectoderm was expanded, and there was a concomitant reduction in expression of non-neural...
Further studies using selective vasopressin receptor antagonists, perhaps targeted to specific regions such as the amygdala, septum or hippocampus, should help to clarify the role of AVP in anxiety and stress.

Rachel Jones

ectodermal markers. Inhibition of BMP signalling also causes expansion of the neuroectoderm, and the authors found that this phenotype could be rescued by forcing the expression of ∆Np63. Overexpression of ∆Np63 in wild-type embryos, by contrast, led to a reduction in the amount of neural tissue. So, ∆Np63 acts downstream of BMP signalling and it seems to be involved in ectodermal cell-fate specification, but how does it work? Bakkers et al. found that if the DNA-binding domain of ∆Np63 was linked to the repressor domain of a different protein, overexpression of the resulting chimeric protein could produce the same phenotype as ∆Np63 overexpression. This implies that ∆Np63 normally functions as a transcriptional repressor, with its DNA-binding domain conferring target specificity.

Taken together, these lines of evidence point towards ∆Np63 acting in the ventral ectoderm, downstream of BMP signalling, to repress genes that promote neural cell-fate specification. To complete the picture, it will be necessary to identify the target genes of ∆Np63, and to find out more about the mechanisms by which it achieves this repression.

Heather Wood

HIGHLIGHTS

IN BRIEF

BEHAVIOURAL GENETICS

Influence of gene action across different time scales on behavior.


Different alleles of the foraging gene (for) in Drosophila cause flies to be either ‘rovers’ (foraging over a wide area) or ‘sitters’ (feeding more locally). Ben-Shahar et al. show that increased expression of the same gene is associated with the age-related transition from hive work to foraging in honeybees. The for gene encodes a cyclic-GMP-dependent protein kinase (PKG), and the authors found that increasing PKG activity could precocious foraging.

ADDITION

Psychostimulant-induced behavioral sensitization depends on nicotinic receptor activation.


The authors found that repeated exposure to nicotine enhanced the psychomotor effects of amphetamine, and that nicotinic antagonists prevented the development of amphetamine- and cocaine-induced behavioural sensitization. Nicotinic antagonists also prevented the increase in dopamine release that was found in the nucleus accumbens after treatment with the psychostimulant drugs. The results indicate that nicotine might enhance the long-term effects of amphetamine and cocaine, and alter their addictive properties.

NEUROLOGICAL DISEASES

Gene-microarray analysis of multiple sclerosis lesions yields new targets validated in autoimmune encephalomyelitis.


Lock et al. compared the gene expression profiles of ‘active’ and ‘silent’ multiple sclerosis (MS) lesions to identify genes that are expressed at different stages of the disease. Several genes that were differentially expressed had not previously been associated with MS. By modulating the expression of two of these genes, the authors reversed the symptoms of EAE (experimental allergic encephalomyelitis), a mouse model of MS. This approach might help in the development of therapies to treat specific aspects of MS.

PSYCHIATRIC DISORDER

Small intestinal enteropathy with epithelial IgG and complement deposition in children with regressive autism.


The authors describe a systematic study of lymphocytic colitis in children with regressive autism, comparing duodenal biopsies from these individuals with those from both normal and disease control groups. Their findings confirm the presence of a new form of enteropathy in autistic children, including increases in mucosal lymphocyte density, crypt cell proliferation, and epithelial deposition of IgG with complement C1q. They point to a possible autoimmune lesion in regressive autism.
Next in line?

The success of the kinase inhibitor Gleevec in the treatment of chronic myelogenous leukaemia — which is caused by the aberrant activity of the BCR–ABL tyrosine kinase — has given much encouragement for the development of other molecularly targeted therapies. As two reports in Cancer Cell now indicate, inhibitors of the FLT3 kinase, which is mutated in ~30% of patients with acute myelogenous leukaemia (AML), could be promising candidates for targeted treatment of this disease.

The first study involved the kinase inhibitor CT53518, which is selective for FLT3 and two other kinases, platelet-derived-growth-factor receptor (PDGFR) and KIT, in vitro. CT53518 was found to inhibit several different constitutively active FLT3 mutants that were cloned from patients with AML and expressed in Ba/F3 cells, and also to induce apoptotic cell death in human AML cell lines with mutations in FLT3. Encouraged by these observations, Kelly et al. tested CT53518 in vivo in two mouse models of mutant-FLT3-mediated AML, and found that CT53518 resulted in a significant decrease in disease progression, as assessed by spleen weight and white blood cell (WBC) count, and an increase in survival. Furthermore, CT53518 was shown to have suitable pharmacokinetic and toxicity profiles for clinical use.

The second study, by Weisberg et al., assessed the activity of the kinase inhibitor PKC412 — which targets FLT3, and also the kinases KDR, PDGFR, KIT and protein kinase C — and found it to be highly toxic to Ba/F3 cells that expressed mutant FLT3 receptors from AML patients. And in a

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mouse model of mutant-FLT3-mediated leukaemia, PKC412 treatment completely blocked the development of leukaemia, whereas all of the mice in the placebo group developed fatal disease. Moreover, spleen weights and WBC counts were also significantly lower in the treated mice.

Both of these studies strongly support the idea that FLT3 is potentially a good drug target in AML. PKC412 and CT53518 are now being evaluated in clinical trials for AML, and it seems likely that several other FLT3 inhibitors will also be clinically tested. It will be of considerable interest to compare their efficacies and toxicities, as these are likely to be influenced by the non-FLT3 targets of each drug, which might differ significantly.

References and links

Inhibition of mutant FLT3 receptors in leukaemia cells by the small molecule tyrosine kinase inhibitor PKC412. Cancer Cell 1, 433–443 (2002)

Further Reading

1

References and links

FURTHER READING

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New stress reducer

The drug SSR149415 blocks a subtype of a receptor found in brain areas associated with anxiety and depression. Researchers showed that animals given the drug were less anxious and less depressed than untreated animals. Compared to traditional antianxiety drugs, the new compound was less effective at reducing anxiety caused by dangerous situations or aversive stimuli, but it was just as effective at reducing stress caused by traumatic social encounters. However, unlike traditional anxiolytics and antidepressants that can disturb sleep, impair memory, and reduce locomotor activity, the drug has few side effects.

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New stress reducer

April 16, 2002

The drug SSR149415 blocks a subtype of a receptor found in brain areas associated with anxiety and depression. Researchers showed that animals given the drug were less anxious and less depressed than untreated animals. Compared to traditional antianxiety drugs, the new compound was less effective at reducing anxiety caused by dangerous situations or aversive stimuli, but it was just as effective at reducing stress caused by traumatic social encounters. However, unlike traditional anxiolytics and antidepressants that can disturb sleep, impair memory, and reduce locomotor activity, the drug has few side effects.

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A new drug has been shown to cut down on stress. It may offer a way to treat emotional disorders, such as anxiety and depression, researchers said in the Proceedings of the National Academy of Sciences.

The drug is called SSR149415. It was tested on mice and rats exposed to acute and chronic stress. Animals getting the drug showed less anxiety and depression than animals left to fend for themselves, said the scientists from Sanofi-Synthelabo in France.

When the researchers compared the compound to traditional anti-anxiety drugs, they found it was less effective in reducing anxiety caused by dangerous or aversive situations but worked just as well in ameliorating stress caused by traumatic social encounters. Unlike medications aimed at the same ends, the new drug has few side effects, said study co-author Guy Griebel.

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Sanofi Researchers Say Anxiety Drug Shows Promise in Mice

By Kristin Reed

Washington, April 15 (Bloomberg) – Sanofi-Synthelabo SA researchers said an experimental anti-anxiety compound appeared promising in mouse studies that will appear tomorrow in the Proceedings of the National Academy of Sciences.

Researchers said the compound, dubbed SSR149415, appeared to ease trauma-related anxiety in mice without triggering the side effects such as sleep disturbances or memory problems that often occur with antidepressants such as fluoxetine, the generic drug Eli Lilly & Co. sells as Prozac, the researchers said.

The compound is still undergoing animal testing at the company's European laboratories. If successful, SSR149415 could one day enter a global depression drug market worth about $13.4 billion in 2000, according to data compiled by IMS Health Inc.

"The present findings indicate that SSR149415 has antidepressant-like properties that are comparable in terms of efficacy of the effects to those of a classical antidepressant," Sanofi-Synthelabo researcher Guy Griebel wrote in the study.

According to the study, the drug helped animals in experiments designed to create symptoms of depression but wasn’t as effective as older medications SSR149415 appeared more promising in tests designed to create anxiety and trauma, the researchers said. The drug seemed generally free of side effects in the rodent studies.

Shares in Sanofi-Synthelabo, France's second-biggest drugmaker, fell 40 cents to 69.1 euros. Bristol-Myers Squibb Co. markets two of Sanofi's best-selling drugs, the heart treatments Plavix and Avapro, in the U.S.
Des cellules souches cérébrales adultes deviennent des neurones

Des chercheurs ont isolé des cellules souches de l’hippocampe de rats adultes et les ont mises en culture avec des cellules de soutien, des astrocytes. Les neurones développés à partir de ces cellules souches adultes ont qualifié potentiellement les mêmes propriétés que les neurones du SNC. Ils ne se divisent pas et présentent un axone et des dendrites, ils sont prêts à communiquer et ensuite à transmettre des signaux. Ils sont capables de réagir à des stimuli d’action en réponse à la stimulation synaptique, ils forment des synapses. Des informations communiquant et transmettant des informations sont intégralement intégrés dans le réseau neuronal. Enfin, ils peuvent libérer les neurotransmetteurs et communiquer en réponse aux potentiels d’action.

Il existe toutefois une différence quantitativa, les neurones ne forment pas autant de synapses que les neurones normaux, mais cette différence s’estompe à l’âge adulte.

Dans une deuxième et une troisième expérience, les chercheurs ont cultivé des cellules souches adultes de l’hippocampe avec ces mêmes cellules, que l’astrocytes, les neurones dérivés de cellules souches adultes de l’hippocampe restent actifs et fonctionnellement et fonctionnellement.

Des implications cliniques potentielles

Cela pourrait avoir des implications cliniques. Il y a beaucoup de discussions à ce sujet de savoir si les cellules souches nerveuses adultes, ainsi que les cellules souches embryonnaires pourraient être utilisées pour régénérer le tissu cérébral d’adultes, explique le Dr Steven, dans un communiqué. « Ces résultats semblaient prometteurs et que si l’organisme était exposé à ce que la thérapie par cellules souches soit possible pour traiter de telles maladies, on pourrait espérer que les cellules souches pourraient être utilisées pour régénérer le tissu cérébral et fonctionnellement et fonctionnellement. »

Art 50 peut être désormais prescrit sans limitation de durée

ECHODIAH, essai mené sur trois ans dans la coarctose, montre que la dia
cerhène (Art 50) ralentit la progression radiologique. Un symposium des Laboratoires Negma-Lerads a permis de faire le point.

Le traitement symptomatique de l’arth
erhène fait appel à des médicaments antalgiques, antinflamatoires non stéro
diens (AINS), ainsi que plus récemment inhibiteurs de COX-2. Ils soulagent la douleur à court terme, souligne le Dr T. Pham (Marseille). Les notions de tolérance et de coût entrent en ligne de compte dans le choix, poursuit-on. On accorde aussi un grand intérêt aux médicaments non pharmacologiques, qui devraient être associées en première intention aux médicaments : perte de poids (pas seulement en cas d’obésité sévère), exercice physique adapté, semelles visco
haptiques, orthèses...

L’évolution des traitements de fond de l’ar
terhène évolue. C’est le cas pour la diclofenac, inhibiteur de l’interleukine 1, dont l’effet struc
tural-modulateur sur le cartilage (1 mois) est mis en évidence par ECHODIAH (Évolut
de la fracture CHONDROMODULATEUR de la DIA
cerhène dans l’Arthrose de l’Hanche). Art 50 est désormais prescrit sans limitation de durée.

Allongement du délai de survenue du pemphigus

Le médicament de survenue du pemphigus est allongé (épargne de 350 jours sur les trois ans d’observation). Le recours à une photolyse de hanches est dimi

Une nouvelle voie de recherche sur les troubles affectifs

Le blocage du récepteur de la vasopres
sine pourrait représenter un nouveau moyen de traiter les troubles affectifs. Cela représente de dix à douze ans de recherche sur une équipe française s’est engagée. Le schéma de l’effet de la vasopresine sur le circuit émotionnel a été de façon significative. L’effet de la vasopresine sur le circuit émotionnel a été de façon significative. L’effet de la vasopresine sur le circuit émotionnel a été de façon significative. L’effet de la vasopresine sur le circuit émotionnel a été de façon significative.
Nom de code SSR149415: une nouvelle classe d'antidépresseur à venir ? - Article complété

- 16/04/2002 - Des chercheurs de Sanofi-Synthelabo relatent dans les comptes-rendus de l'académie des sciences américaine, l'essai chez le rat et la souris, d'une nouvelle molécule visant à traiter les troubles de l'émotion comme le stress et l'anxiété. Cette molécule cible les récepteurs de la vasopressine V1b impliquée dans l'emotivité et permet de réduire l'anxiété parmi des animaux soumis à un stress psychosocial. La molécule, dénommée SSR149415, prise oralement, semble aussi efficace que la fluoxétine pour réduire le stress chronique sans avoir les effets secondaires indésirables des antidépresseurs traditionnels.

L'arginine vasopressine (AVG) est un neurotransmetteur cyclique non peptidique synthétisé dans l'hippothalamus et retrouvé sous les formes V1a et V1b dans le système nerveux central, notamment dans les zones associées à l'anxiété et à la dépression, où leurs récepteurs sont présents.

Récemment, un antagoniste des récepteurs de la forme V1b de l'arginine vasopressine (SSR149415) a été mis au point dans les laboratoires du groupe pharmaceutique, et Guy Griebel (Bagneux, France) et ses collaborateurs ont décidé de le tester sur des modèles animaux de troubles du comportement liés à l'anxiété et à la dépression, induits par différents facteurs externes.

SSR149415 a produit, sur les modèles animaux classiques d'anxiété, des effets anxiolytiques à partir d'une dose de 1 mg/kg (administré per os ou en ip), mais moins importants que ceux du diazépam (une benzodiazépine) utilisé comme contrôle positif.

En revanche, dans les modèles de stress traumatiques, SSR49415 a montré des effets anxiolytiques nets et efficaces, avec une amélioration de l'anxiété, de l'état physique et du comportement stressé.

SSR49415, de plus, semble montrer moins d'effets indésirables que les antidépresseurs et anxiolytiques classiques, comme la perturbation du sommeil, la perte de mémoire et l'activité locomotrice réduite.

Source : Proc Natl Acad Sci of the USA 16 avril 2002; publication électronique avancée, DOI:10.1073/pnas092012099

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2002-04-16 09:36:46
Sanofi-Synthelabo SA/Wegelin: Neues Medikament gegen Angst

Zürich (awp 22) - Die Forscher des französischen
Pharmaunternehmens Sanofi-Synthelabo haben vielversprechende
Ergebnisse in der Entwicklung eines
Medikamentes gegen Angstzustände erzielt. Das Präparat wurde
bei Mäusen erfolgreich getestet und die Schlussfolgerungen
sollen in der am Mittwoch dieser Woche stattfindenden Sitzung
der National Academy of Sciences vorgelegt werden. Das Mittel
mit dem Testnamen SSR149415 könnte in ferner Zukunft womöglich
das Antidepressivum von Ely Lilly, Prozac, konkurrieren,
schreibt Wegelin im heutigen "Früh - Stick".

(page 3)
Nouveau medicament contre la peur
Les chercheurs de Sanofi-Synthelabo ont atteint des résultats prometteurs dans le
développement d’un médicament agissant dans les états anxieux. La molécule (SSR
149415) a donné des résultats positifs chez les souris et les résultats devraient être
présentés ce mercredi à la réunion de l’académie nationale des sciences. Elle
pourrait concurrencer éventuellement un jour le prozac.
Experimentan en Francia cun novo fármaco destinado a combate-lo estrés

Un novo medicamento, que se dirixe ó receptor da vasopresina, pode ofrecer unha nova forma de tratar alteracións emocionais, segundo informan os autores dun novo estudo publicado na última edición da revista *Proceedings*. O composto, coñecido como SSR149415, bloquea un subtipo de receptor que se atopa en áreas cerebrais asociadas coa ansiedade e a depresión. Nunca serie de experimentos que expuxeron a ratas e ratos a formas agudas e crónicas de estrés, científicos de Sanofi-Synthelabo, en Francia, amosaron que os animais nos que se lles administrou medicamento tiñan menos ansiedade e estaban menos deprimidos que os animais non tratados.

Comparado cos fármacos tradicionais contra a ansiedade, o novo composto era menos eficaz á hora de reduci-la ansiedade causada por situaacións perigosas ou a estímulos que producían aversión ou rexetamento, pero igual de eficaz á hora de reduci-lo estrés debido a encontros sociais traumáticos.
Francia.- Se desarrolla un nuevo fármaco que podría combatir el estrés

4/15/2002 11:36:37 AM
((Noticia embargada hasta las once de la noche))

MADRID 15 (EUROPA PRESS)

Un nuevo medicamento que se dirige al receptor de la vasopresina puede ofrecer una nueva forma de tratar alteraciones emocionales según informan los autores de un nuevo estudio publicado en la última edición de la revista Proceedings. El compuesto conocido como SSR149415 bloquea un subtipo de receptor que se encuentra en áreas cerebrales asociadas con la ansiedad y la depresión.

En una serie de experimentos que expusieron a ratas y ratones a formas agudas y crónicas de estrés científicos de Sanofi-Synthelabo en Francia mostraron que los animales a los que se administró el medicamento tenían menos ansiedad y estaban menos deprimidos que los animales no tratados.

Comparado con los fármacos tradicionales contra la ansiedad el nuevo compuesto era menos eficaz a la hora de reducir la ansiedad causada por situaciones peligrosas o a estímulos que producen aversión o rechazo pero igual de eficaz a la hora de reducir el estrés debido a encuentros sociales traumáticos. En un test que duró meses para medir la capacidad del nuevo medicamento de reducir los efectos del estrés crónico se vio que SSR149415 era igual de eficaz que la fluoxetina un antidepresivo común.

Sin embargo a diferencia de los ansiolíticos y antidepresivos tradicionales que alteran el sueño limitan la memoria y reducen la actividad locomotriz el nuevo fármaco posee escasos efectos secundarios. Los autores concluyen que SSR149415 puede ser útil para tratar la depresión y ciertas clases de ansiedad alteraciones que a menudo se producen al mismo tiempo en un mismo paciente.