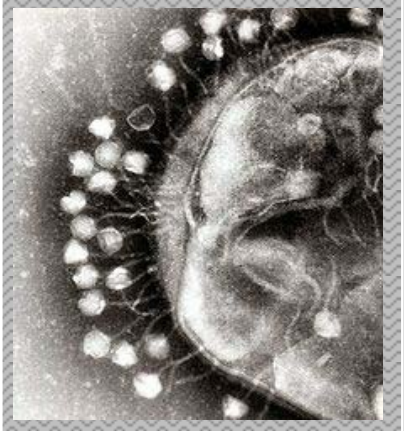

EMGEN Newsletter

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INSIDE THIS ISSUE:

Eastern Mediterranean Health Genomics and Biotechnology Network (EMGEN) was created in 2004 with collaboration of representatives of selected center of excellence in (health related) molecular biology, biotechnology & genomics in the Eastern Mediterranean region by recommendations and efforts of WHO/EMRO.

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Role of NGAL for the early detection of acute kidney injury

*An article entitled "Role of NGAL for the early detection of acute kidney injury " aims to identification of NGAL as a marker in acute kidney injury. The study was carried out by **Zohreh Rostami**; she is working in Nephrology and Urology Research Center, Baqiyatallah University of Medical Sciences, Tehran. Iran; and the paper was published in International Journal of Nephrol Urology, 2010; 2 (3): 387-389*

Dr. Zohreh Rostami

The incidence of acute kidney injury (AKI) is estimated to be from 5% of hospitalized patients to 30–50% of patients in intensive care units, and nowadays there is significant evidence of an increase in its incidence [1, 2]. However, the lack of early, predictive and non-invasive biomarkers may lead to delayed initiation of potentially useful therapies for this common clinical setting [3]. The diagnosis of AKI is usually based on increases in serum creatinine levels; however, it is not a good indicator of acute deterioration of kidney function. In addition, serum creatinine concentration is greatly influenced by numerous non-renal factors (such as lean muscle mass, race, age, gender, hydration status, medications, muscle metabolism, and protein intake). Measurement of serum creatinine is not a preferred marker of renal function in AKI, since these patients are not in steady state. Therefore, changes in serum creatinine may appear much later than renal injury. Thus, considerable rises in serum creatinine is often not apparent until 48–72 h after the initial insult to the kidney. In addition, no significant changes in serum creatinine concentrations may be seen until about 50% of renal function has already been lost. In other words the renal disease can exist with minimal or no changes in serum creatinine because of renal reserve, enhanced tubular secretion of creatinine, and compensated lower rates of glomerular filtration [1, 2, 4, 5].

Biomarkers for early AKI diagnosis represent a unique opportunity for an intervention to save the kidney from additional insults and avoid tissue damage. If we wait for the present paraclinical data which can certainly help, we will always be late. Therefore, we need a novel and more sensitive biomarker for the diagnosis of AKI in order to treat it as soon as possible [5]. This biomarker must also be specific, practically easy

to detect and measure, reproducible, feasible at bedside, correlative with severity, quantitatively describing the intensity of renal damage even when typical clinical signs are absent and is appropriate to indicate the initiation of the therapy [5]. Fortunately, promising methods such as functional genomics and proteomics have discovered new candidates as biomarkers [3, 6], and among them neutrophil gelatinase-associated lipocalin (NGAL), which is a small molecular size (25 kDa) protein and resistance to degradation [7], is a promising biomarker. Several studies have reported that in patients with AKI, NGAL rises very high compared to matching controls [6, 8, 9]. In addition, NGAL enhancement occurs in different studies at 24 to 48 hours sooner than the increase in creatinine. Both urinary and plasma NGAL are excellent early markers of AKI with an area under the receiver operator characteristic curve (AUC) in the range of 0.9. Nowadays, many studies are being performed to illuminate exact correlation between NGAL and AKI [5]. By the way further studies are required to depict, does the NGAL actually enable to distinguish between various AKI subtypes (prerenal, intrinsic renal, or postrenal)? Is NGAL more efficient than other methods (BUN/Cr, urine osmolality, fractional excretion of Na, needle biopsy, etc) in evaluation of AKI subtypes? Can we hope to not to need an invasive procedure for the early detection of AKI etiology in future?

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Proteopathy

In medicine, **proteopathy** alludes to a class of diseases in which definite proteins become structurally irregular, and so disturb the purpose of cells, tissues and organs of the body. Often the proteins fail to crease into their normal pattern; in this misfolded situation, the proteins can become toxic in some technique (a gain of toxic function) or they can lose their normal purpose. The proteopathies (also known as proteinopathies, protein conformational disorders, or protein misfolding diseases), combine such diseases as Alzheimer's disease, Parkinson's disease, type 2 diabetes, amyloidosis, selective hyperproteolytic diseases, and an extensive variety of other diseases.

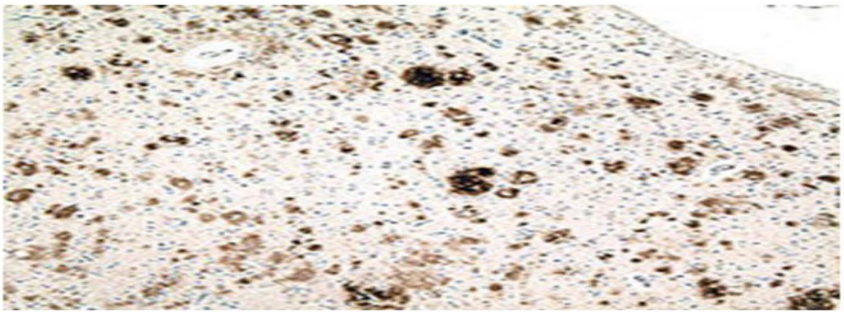
The idea of proteopathy can mark out its genesis to the mid-19th century, when, in 1854, Rudolf Virchow made the word amyloid "starch-like" to explain a substance in rational corpora amylases that exhibited a chemical reaction like that of cellulose. In 1859, Friedreich and Kekule confirmed that, rather than add up to cellulose; in fact "amyloid" is rich in protein. following study has shown that many different proteins can form amyloid, and that all amyloids have in ordinary birefringence in cross-polarized light after mark with the coloring Congo Red, as well as a fibrillar ultrastructure when viewed with an electron microscope. Furthermore, confirmation has emerged those small, non-fibrillar protein aggregates recognized as oligomers are toxic to the cells of an exaggerated organ, and that amyloidogenic proteins in their fibrillar form may be comparatively benevolent.

Pathophysiology

In the majority, if not all proteopathies, modify in the 3-dimensional conformation increases the propensity of a specific protein to bind to it. In this aggregated figure, the protein is opposed to permission and can obstruct with the normal ability of the exaggerated organs. In some cases, misfolding of the protein results in a loss of its characteristic purpose, such as in cystic fibrosis, which is caused by a imperfect cystic fibrosis transmembrane conductance regulator (CFTR) protein. Because proteins split an ordinary structural characteristic known as the polypeptide spine, all proteins have the possible to misfold under some situation.

However, only a comparatively few proteins are connected to proteopathic disorders, perhaps due to structural idiosyncrasies of the susceptible proteins. For instance, proteins that are moderately unbalanced as monomers (that is, as single, unbound molecules) are more expected to misfold into an irregular conformation. Almost all instances, the disease-causing molecular pattern involves an augment in beta-sheet secondary structure of the protein. The irregular proteins in some proteopathies have been exposed to fold into multiple 3-dimensional shapes; this variation, proteinaceous structures are distinct by their different pathogenic, biochemical, and conformational assets. They have been most methodically studied with regard to prion disease, and are alluded to as protein strains.

The possibility that proteopathy will extend is augmented by certain risk factors that encourage the self-assembly of a protein. These combine destabilizing changes in the main amino acid sequence of the protein, post-translational modifications (such as hyperphosphorylation), changes in temperature or pH, an augment in construction of a protein, or a decrease in its approval. Advancing age is a strong risk factor, as is shocking brain injury. In the aging brain, numerous proteopathies can extend beyond. For instant, besides Aβ-amyloidosis, many Alzheimer patients have attendant synucleinopathy (Lewy bodies) in the brain.



Micrograph of a part of the cerebral cortex from a patient with Alzheimer's disease, immunostained with an antibody to Aβ (brown), a protein fragment that accumulates in senile plaques and cerebral amyloid angiopathy.

Seeded Induction of Proteopathy

Some proteins can be encouraged to form abnormal assemblies by contact to the same (or similar) protein that has folded into a disease-causing agreement, a procedure called 'seeding' or 'permissive templating. In this technique, the disease condition can be brought about in a vulnerable host by the introduction of diseased tissue extract from an afflicted contributor. The best known form of such incitable proteopa

-thy is prion disease, which can be transmitted by revelation of a host organism to purified prion protein in a disease-causing conformation. There is now confirmation that other proteopathies can be induced by a similar method, counting AA amyloidosis, apolipoprotein AII amyloidosis, Aβ amyloidosis, and tauopathy. In all of these cases, an abnormal form of the protein itself appears to be the pathogenic mediator. In some cases, the statement of one kind of protein can be experimentally induced by aggregated assemblies of other proteins that are rich in β-sheet arrangement, maybe because of structural approvingly of the protein molecules. For instance, amyloid A (AA) amyloidosis can be enthused in mice by such varied macromolecules as silk, the yeast amyloid Sup35, and curli from the bacterium Escherichia coli. In addition, apoplipoprotein AII amyloid can be induced in mice by a diversity of β-sheet rich amyloid fibrils, and intellectual tauopathy can be induced by brain extracts that are rich in aggregated Aβ. In common, such heterologous seeding is less competent than is seeding by a tainted form of the same molecule.

List of Proteopathies:

Proteopathy	***Major aggregating protein***
Alzheimer's disease	Amyloid β peptide (Aβ); Tau protein
Cerebral β-amyloid angiopathy	Amyloid β peptide (Aβ)
Critical illness myopathy (CIM)	Hyperproteolytic state of myosin ubiquitination
Retinal ganglion cell degeneration in glaucoma	Amyloid β peptide (Aβ)
Prion diseases (multiple)	Prion protein
Parkinson's disease and other synucleinopathies (multiple)	α-Synuclein

Tauopathies (multiple)	Microtubule-associated protein tau (Tau protein)
Frontotemporal lobar degeneration (FTLD) (Ubi+, Tau-)	TDP-43
FTLD-FUS	Fused in sarcoma (FUS) protein
Amyotrophic lateral sclerosis (ALS)	Superoxide dismutase, TDP-43, FUS
Huntington's disease and other triplet repeat disorders (multiple)	Proteins with tandem glutamine expansions
Familial British dementia	ABri
Familial Danish dementia	ADan
Hereditary cerebral hemorrhage with amyloidosis (Icelandic) (HCHWA-I)	Cystatin C
CADASIL	Notch3
Alexander disease	Glial fibrillary acidic protein (GFAP)
Seipinopathies	Seipin
Familial amyloidotic neuropathy, Senile systemic amyloidosis	Transthyretin
Serpinopathies (multiple)	Serpins
AL (light chain) amyloidosis (primary systemic amyloidosis)	Monoclonal immunoglobulin light chains
AH (heavy chain) amyloidosis	Immunoglobulin heavy chains
AA (secondary) amyloidosis	Amyloid A protein
Type II diabetes	Islet amyloid polypeptide (IAPP; amylin)
Aortic medial amyloidosis	Medin (lactadherin)
ApoAI amyloidosis	Apolipoprotein AI
ApoAII amyloidosis	Apolipoprotein AII

ApoAIV amyloidosis	Apolipoprotein AIV
Familial amyloidosis of the Finnish type (FAF)	Gelsolin
Lysozyme amyloidosis	Lysozyme
Fibrinogen amyloidosis	Fibrinogen
Dialysis amyloidosis	Beta-2 microglobulin
Inclusion body myositis/myopathy	Amyloid β peptide (Aβ)
Cataracts	Crystallins
Medullary thyroid carcinoma	Calcitonin
Cardiac atrial amyloidosis	Atrial natriuretic factor
Pituitary prolactinoma	Prolactin
Hereditary lattice corneal dystrophy	Keratoepithelin
Cutaneous lichen amyloidosis	Keratins
Mallory bodies	Keratin intermediate filament proteins
Corneal lactoferrin amyloidosis	Lactoferrin
Pulmonary alveolar proteinosis	Surfactant protein C (SP-C)
Odontogenic (Pindborg) tumor amyloid	Odontogenic ameloblast-associated protein
Seminal vesical amyloid	Semenogelin I
Cystic Fibrosis	cystic fibrosis transmembrane conductance regulator (CFTR) protein
Sickle cell disease	Hemoglobin

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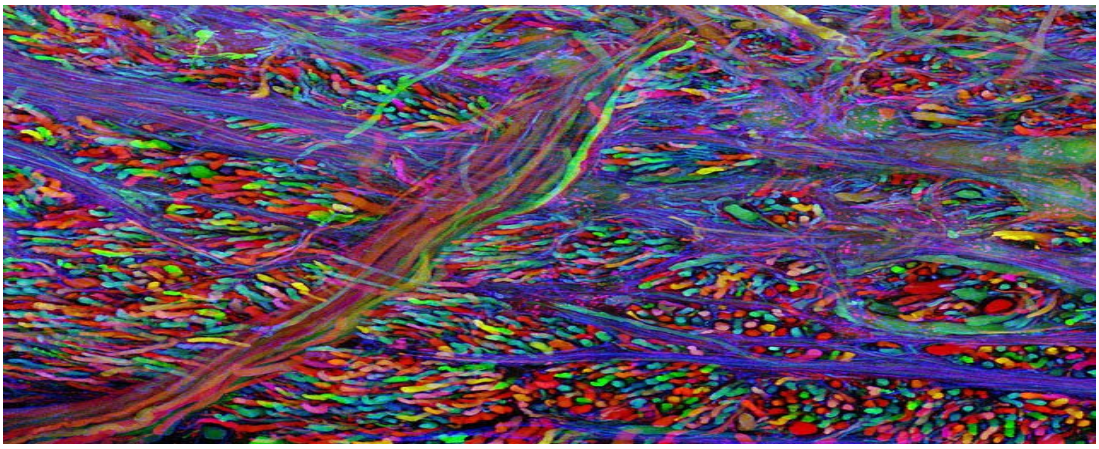
Brainbow

Brainbow is an expression used to explain the process by which individual neurons in the brain can be eminent from adjacent neurons via fluorescent proteins. By arbitrarily expressing assorted ratios of red, green, and blue derivatives of green fluorescent protein in individual neurons, it is probable to standard each neuron with a characteristic color. This procedure has been a major donation to the field of connectomics, or the study of neural relations in the brain.

At first, the technique was developed in the spring of 2007 by a team led by both professors of Molecular & Cellular Biology in the Department of Neurobiology at Harvard Medical School; Jeff W. Lichtman and Joshua R. Sanes. Their expression of the technique in mice first published in the November 1, 2007 issue of the journal Nature. The innovative technique has been recently modified for use with other model organisms as well as Drosophila melanogaster and Caenorhabditis elegans.

While previous classification techniques allowable for the mapping of only a few neurons, this new method allows more than 100 differently mapped neurons to be concurrently and differentially illuminated in this way. The resultant images can be fairly affecting and have in fact won awards in science photography conflict.



Brainbow image of hippocampal neurons. Courtesy of Jeff Lichtman/Harvard University

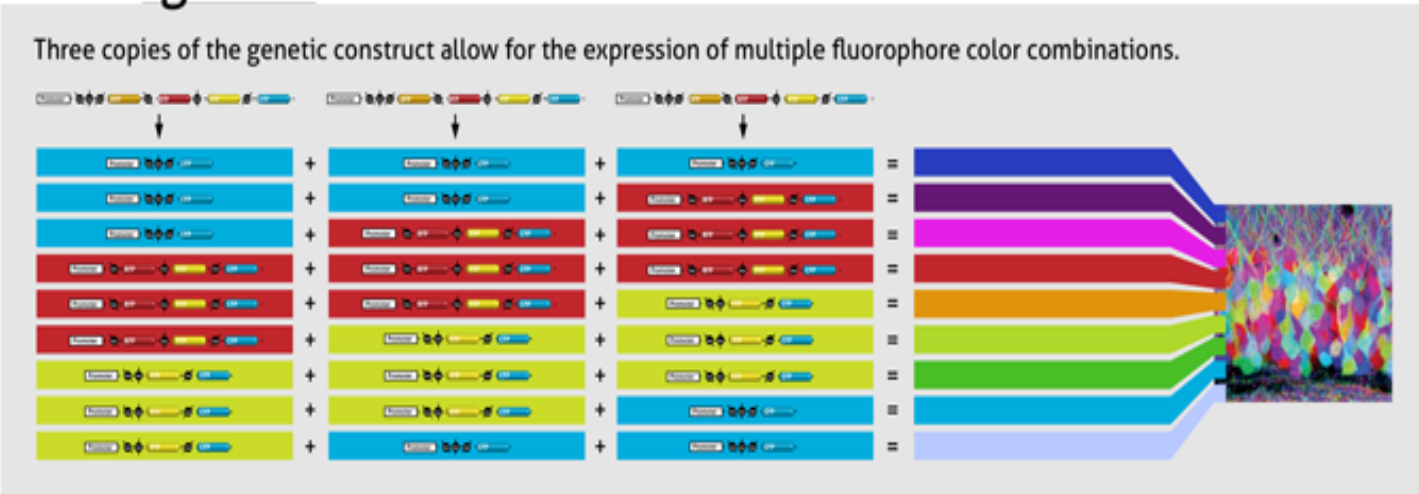
History and Development

A team of researchers in the Department of Neurobiology at Harvard Medical School in 2007 developed the Brainbow neuroimaging technique. Professors Jeff W. Lichtman and Joshua R. Sanes, both of whom concentrate in Molecular and Cellular Biology and are highly famous for their work, led this specific group of scientists. The team assembled Brainbow using a two-step method: first, a precise genetic construct was begetting that could be recombined in numerous preparations to create one of either three or four colors based on the exacting fluorescent proteins (XFPs) being appliance. Subsequently, numerous copies of the same transgenic construct were i into the genome of the target species, resulting in the random expression of different XFP ratios and subsequently causing different cells to exhibit a variety of colorful hues.

Brainbow was initially shaped as a development over more customary neuroimaging techniques, such as Golgi staining and dye injection, both of which obtainable severe confines to researchers in their aptitude to imagine the complicated architecture of neural circuitry in the brain. although elder techniques were only able to coloring cells with a constricted variety of colors, often utilizing bi- and tri-color transgenic mice to reveal incomplete information in regards to neuronal arrangements, Brainbow is much more supple in that it has the ability to fluorescently label individual neurons with up to about 100 different hues so that scientists can recognize and even distinguish between dendritic and axonal processes. By illuminating such comprehensive information about neuronal connectivity and patterns, sometimes even in vivo, scientists are often able to deduce information concerning neuronal connections and their following impact upon performance and function. Thus, Brainbow packed the void that earlier neuroimaging methods were unable to complete.

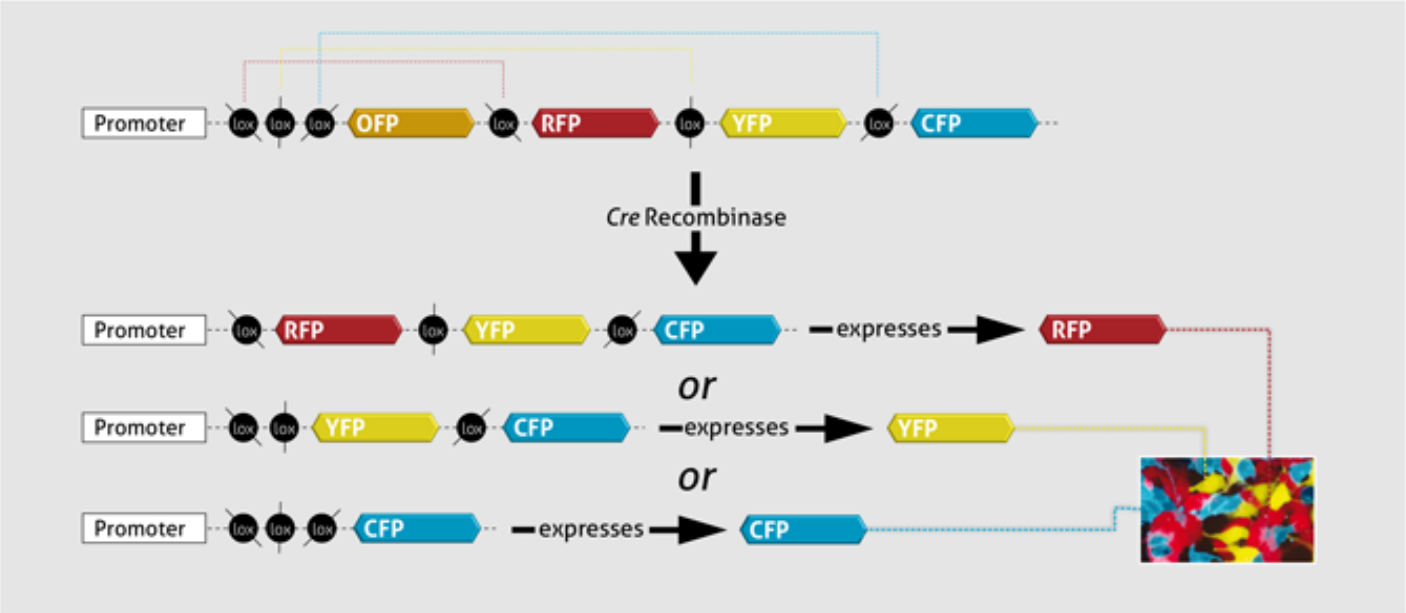
With the new arrival of Brainbow in neuroscience, researchers are now able to create precise maps of neural circuits and better examine how these relate to a variety of mental actions and their linked behaviors (ie. Brainbow reveals information about the interconnections between neurons and their following connections that affect generally brain functionality). As a further extrapolation of this technique, Brainbow can therefore also be used to revise both neurological and psychological disorders by analyzing differences in neural maps.

Building Brainbow



Lawson Kurtz et al./Duke University

Basic Genetic Construct



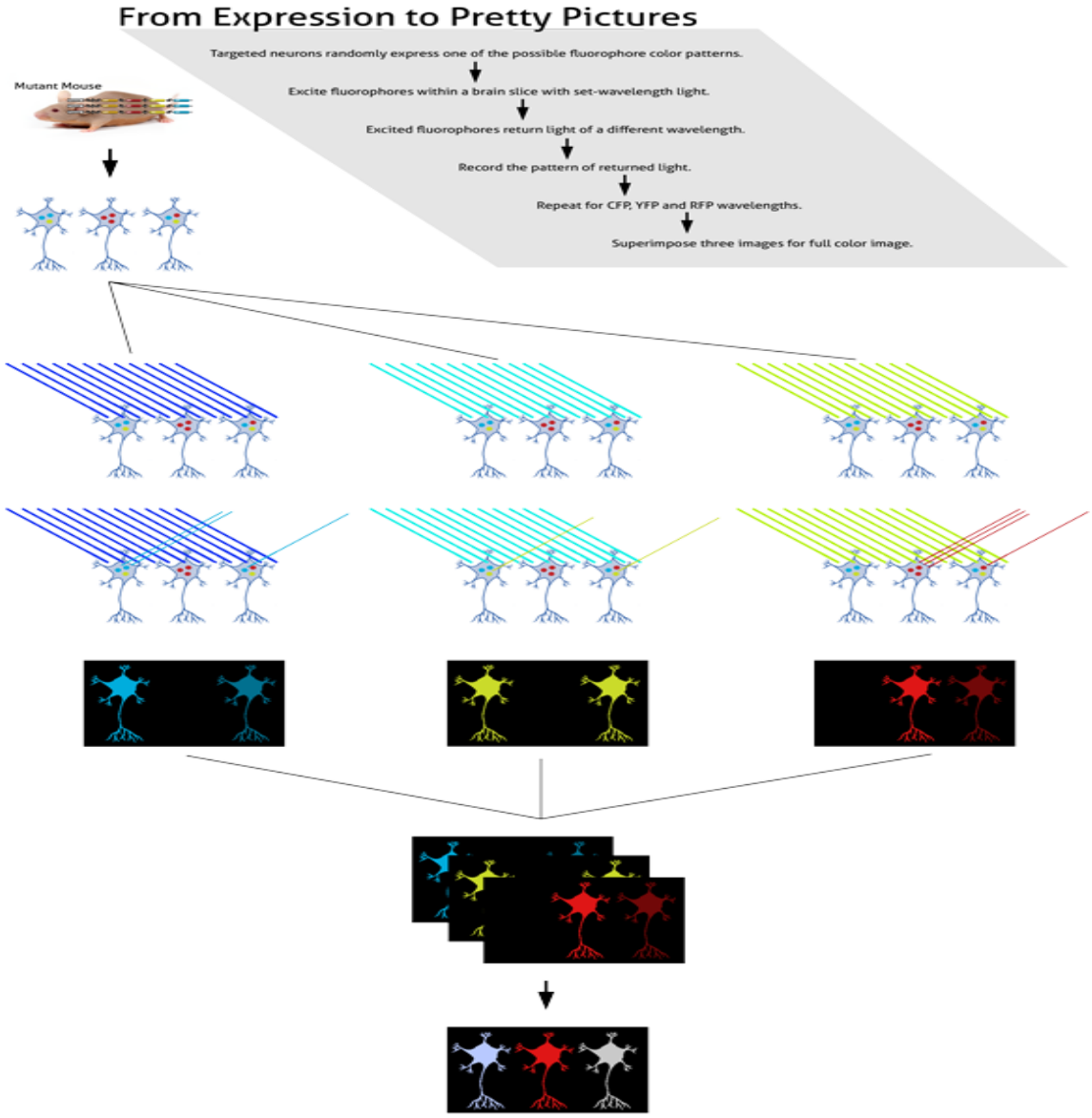
The basic Brainbow1 genetic construct. Lawson Kurtz et al./Duke University

Methods

Brainbow techniques depend on the Cre-Lox recombination, in which the protein Cre recombinase drives inversion or removal of DNA between loxP sites. The unique Brainbow technique includes both Brainbow-1 and Brainbow-2, which use diverse forms of cre/lox recombination. Brainbow-1 uses DNA constructs with dissimilar fluorescent protein genes (XFPs) alienated by mutant and canonical forms of loxP. This creates a set of equally elite removal possibilities, since cre mediated recombination occurs only between indisti -guishable loxP sites.

After recombination occurs, the fluorescent protein that is left straight after the promoter is exclusively uttered. Thus, a construct with four XFPs alienated by three different loxP sites, three removal events, and the unique construct can create four different fluorescent proteins. Brainbow-2 uses Cre deletion and inversion to permit numerous expression potential in a given construct. In one DNA section with two oppositely leaning XFPs, Cre will encourage a random inversion event that leaves one fluorescent protein in the correct direction for expression. If two of these invertible sequences are allied, three different inversion events are probable. When deletion events are also measured, one of four fluorescent proteins will be expressed for a given mixture of Cre excisions and inversions. For both Brainbow-1 and-2, the expression of a given XFP is a stochastic, or accidental, event. Brainbow is implemented in vivo by crossing two transgenic organism strains: one that expresses the Cre protein and another that has been transected with some versions of a loxP/XFP construct. By manifold copies of the transgene allows the XFPs to join in a way that can give one of about 100 different colors. Thus, each neuron is labeled with a dissimilar hue based on its given combinatorial and stochastic expression of fluorescent proteins. In order to clarify disparity XFP expression patterns into an observable form, brain slices are imaged with confocal microscopy. When bare to a photon with its meticulous excitation wavelength, each fluorophore emits a signal that is collected into a red, green, or blue channel, and the resulting light grouping is analyzed with data analysis software. Superimposition of differentially colored neurons allows illustration unraveling of complex neural circuits.

Brainbow has been mostly tested in mice to date; however, the essential technique described above has also been customized for use in more new studies since the start of the unique method introduced in 2007.



Creating Brainbow images. Lawson Kurtz et al./Duke University

Mice

The mouse brain has 4,000,000 neurons and is more akin to a human brain than both drosophila and other usually used organisms to model this method, such as C. elegans. For the first time the Brainbow technique of neuroimaging was successfully employed in Mice. Livet et al. (2007) developed two versions of Brainbow mice by means of Brainbow-1 and Brainbow-2, which are demonstrated above. In using these

techniques to make a complete map and path the axons of a mouse muscle, it is essential to collect tens of thousands of images and accumulate them into masses to create a complete schematic. It is then possible to draw each motor axon and its synaptic associates to make a total connectome of the muscle.

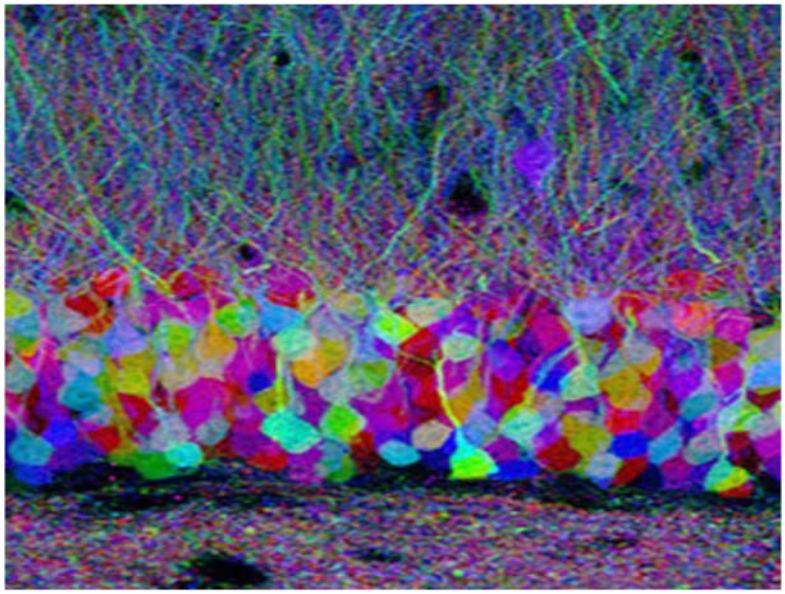
Drosophila

The difficulty of the Drosophila brain, consisting of about 100,000 neurons, makes it a brilliant contender for implementing neurophysiology and neuroscience techniques like Brainbow. In fact, Stefanie Hampel et al. (2011) joint Brainbow in combination with genetic targeting tools to recognize individual neurons within the Drosophila brain and a variety of neuronal lineages. One of the genetic targeting tools was a (UAS) -GAL4 double expression system that controls the expression of UAS-Brainbow and targets the expression to small groups of neurons. Utilizing 'Flip Out' techniques augmented the cellular decree of the reporter construct. The expression of fluorescent proteins, as with the unique Brainbow, depended on Cre recombination matching with coordinated lox sites. Hampel et al. (2011) also developed their own difference of Brainbow (dBrainbow), based on antibody labeling of epitopes rather than endogenous fluorescence. Two copies of their construct yield six bright, divisible colors. This, along with simplifications in color task, enabled them to view the trajectories of each neuron over long distances. Specially, they traced motor neurons from the antennal lobe to neuromuscular junctions, allowing them to recognize the specific muscle targets of individual neurons. Eventually, this method provides the aptitude to efficaciously map the neuronal circuitry in Drosophila so that scientists are able to expose more information about the brain structure of this invertebrate and how it relates to its resulting behavior.

Limitations

Just as any method, Brainbow has a number of limitations that required performing it. For example, the process of propagation at least two strains of transgenic animals from embryonic stem cells is both time consuming and multifaceted. Even if two transgenic species are successfully shaped, not all of their progeny will show the recombination. Thus, this requires wide planning prior to performing a trial. As well, due to the accidental nature in the expression of the fluorescent proteins, scientists are powerless to exactly control the

labeling of neural circuitry, which may consequence in the poor recognition of specific neurons. The use of brainbow in mammalian populations is also disadvantaged by the unbelievable variety of neurons of the central nervous system. The pure density of neurons coupled with the attendance of long tracts of axons make performance larger areas of the CNS with high decree difficult. Brainbow is most helpful when examining single cell resolution next to the backdrop of a complex multicellular setting. However, due to the resolution limits of optical microscopy, decisive recognition of synaptic connections between neurons is not easily talented. This issue is rather avoided by the use of synaptic markers to addition the use of optical microscopy in viewing synaptic relations.



Brainbow image of the dentate gyrus. Courtesy of Jeff Lichtman/Harvard University

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Scientists find out 'Fickle' DNA Changes in Brain

The scientists, who include a husband-and-wife team, establish evidence of an epigenetic change called demethylation in the non-dividing brain cells' DNA, demanding the scientific creed that even if the DNA in non-dividing adult neurons changes on occasion from methylated to demethylated state, it does so very uncommonly. They provide ultimate evidence suggestive of that DNA demethylation happens in non-dividing neurons, and it happens on a large scale.

They used electric shock to arouse the brains of live mice. A few hours after commanding the brain motivation, the scientists analyzed two million of the similar type of neurons from the brains of stimulated mice, focusing on what happens to one structure block of DNA -- cytosine -- at 219,991 sites. These sites assumed about one percent of all cytosines in the whole mouse genomes. The scientists used the latest DNA sequencing skill and compared neurons in mice with or without brain stimulation. About 1.4 percent of the cytosines deliberate showed rapid active demethylation or became newly methylated.

Because DNA demethylation can happen inertly during cell division, the scientists targeted emission to the sections of mouse brains they were studying, enduringly preventing passive cell division, and still found evidence of DNA demethylation. This confirms, they say, that the DNA methylation changes they deliberate occurred separately of cell division.

Their finding opens up new opportunities to understand if these epigenetic modifications are potential drug targets for treating depression and endorse renewal.

This research was supported by the National Institutes of Health, a McKnight Scholar Award, the Brain and Behavior Research Foundation, the Adel son Medical Research Foundation, and the Johns Hopkins Brain Science Institute.

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Guo JU., Ma DK., Mo H., Ball MP., Jang MH., Bonaguidi MA., Balazer JA., Eaves HL., Xie B., Ford E., Zhang K., Ming Gl., Gao Y., Song H., (2011). Neuronal activity modifies the DNA methylation landscape in the adult brain. *Nature Neuroscience*; 14 (10): 1345-1351.

Powerful Antibody-Based Strategy Suggests a New Therapeutic Approach to Diabetes and Obesity

Type 2 Diabetes, the most ordinary form, is a disease of insulin resistance. In the disease condition, an insulin molecule docks at a cell, but the cellular mechanism that sends its signal does not work correctly. This is why just adding more insulin will not solve the problem. What you really need to do is to encourage insulin signaling within the cell to favor the phosphorylation proceedings triggered by the insulin receptor. For over a decade, scientists have known that when PTP1B is concealed in mice by "knocking out" the gene that encodes it, the mice are rendered apparently resistant to corpulence induced by a diet that is high in carbohydrate and fat; importantly, these mice show augmented compassion to the beneficial effects of insulin and do not expand diabetes. This could indicate that producing an inhibitor of PTP1B may be an effective approach to regenerate a normal response in insulin-resistant, diabetic patients. The question for drug developers has been how to repress PTP1B in people, in the specific background of its role in the insulin signaling pathway. Gene knockouts in humans, whether in exact cell types or systemically, are not possible with recent technology. As well, a variety of obstacles have also banned scientists from inhibiting PTP1B biochemically with small-molecule drugs.

Previous studies have distinct the three-dimensional arrangement of PTP1B. From this, they know that the active site of the enzyme-- the place on the molecule where the elimination of phosphate from target substrates is catalyzed-- is a deep cleft. The development of molecules that block the active site, and thereby inhibit the purpose of an enzyme, is a standard plan for drug design. However, this profound active-site cleft in PTP1B has obtainable a major confront to drug developers because it is highly emotional. As a result any small molecules that attach there competently tend also to be highly charged and so will not get across cell membranes. This limits their drug development potential. The scientists have recognized a new targeting approach that sidesteps some of the key impediments confrontations in trying to develop an active site-directed, drug-like PTP1B inhibitor. The approach is described in a paper published September 29 in the journal Cell.

Reference:

Haque A., Andersen JN., Salmeen A., Barford D., Tonks NK., (2011). Conformation-Sensing Antibodies Stabilize the Oxidized Form of PTP1B and Inhibit its Phosphatase Activity. *Cell*; 147 (1): 185-198.

Microorganisms in Industry and Environment

This book (World Scientific publisher, New Jersey, 2011) aims to distribute the most current investigate in applied microbiology obtainable.

This volume offers an appealing examination of microbiology from technical and industrial research to consumer products in a collection of more than 150 papers written by leading experts in the field, who afford critical insights into numerous topics, review current research and discuss future directions to stimulate further deliberations. This book also serves as an update on the most significant current microbial study, by providing a complete overview of cutting-edge topics in a single volume, where readers can also gain insights into how microbiology can resolve problems in everyday settings.

Though mainly intended for microbiologists interested in knowing the latest developments in agriculture, environmental, food, industrial, medical and pharmaceutical microbiology and microbial biotechnology, this book is also a great source of reference for scientists and researchers concerned in advancements in applied microbiology.

Edited by:

A Mendez-Vilas *(Formatex Research Center, Spain & University of Extremadura, Spain)*

Readership:

Microbiologists; biotech researchers; researchers in cell/molecular biology, biophysics, physiology, genetics, pharmacology, biochemistry and agriculture.

Announcement

http://www.vipca.at/Molecol/

The International Conference Molecular Ecology (February 4 - 7, 2012) will cover the recent advances in following research areas:

- Population and Landscape Genetics
- Molecular Tools in Ecology
- Genetic Analysis of Populations
- Genetic Variation and Diversity
- Molecular Evolution
- Phylogenetics and Phylogeography
- Molecular Approaches to Behavioral Ecology
- Conservation Genetics & Genomics
- Applied Molecular Ecology

http://www.keystonesymposia.org/Meetings

Announcement

http://www.bioinfoconf.org

http://www.mohb.org/2012

Center for Genomics and Public Health

In this issue, we would like to introduce The Center for Genomics and Public Health Website (http://depts.washington.edu/cgph/), which is a training and resource center at the University of Washington. The Center serves as a local center of expertise in genomics and public health with a focus on translating genomic information into applied public health knowledge. Also the Center affords scientific assistance to state and local public health agencies and assembles genomics into programs and practice. This website is a very useful portal, which consist of five different information sections:

· *Genetic Testing*

· *Risk Benefit for Genetics*

· *Family History*

· *Health Disorders*

· *Training and Resources*

Genetic Testing contains interesting information about Genomics and Genetics and Genetic testing which determine an individual's physical character, growth, and much other individuality. It can be useful for detecting variations in genes that could increase vulnerability to certain diseases or conditions.

The next section is "***Risk Benefit for Genetics***", gives the information about formal clinical risk-benefit structure to facilitate the translational pathway for genomic technologies using three case studies. Specially, these case studies are:

1. *Increasing a quantitative risk-benefit structure for evaluating the scientific utility of genetic tests in collaboration with stake-holder groups, utilizing warfarin pharmacogenomics as a case example.*
2. *Appraising the generalizability of the risk-benefit structure by applying it to gene expression profiling in women with early stage breast cancer Appraising the generalizability of the risk-benefit structure by applying it to Factor V Leiden testing for pregnant women with clotting or unfavorable pregnancy outcomes.*

Family History section provides related questions on the Behavioral Risk Factor observation System Survey (BRFSS), the National Health and Nutrition assessment Survey, and the health styles survey for public Health care professionals.

The next part is "***Health Disorders***" Contains interesting information to improve our understanding of multifaceted diseases such as asthma, diabetes, cardiovascular health (familial hypercholesterolemia) and obesity, as well as develop medical treatment of these diseases.

You can find below information In ***Training and Resources*** section, which is one of the most useful parts of this website:

- *Technical Assistance*
- *Online Training*
- *Center Publications*
- *Links to Other Resources*
- *Other Publications*
- *In the News*

Cover Picture

Title: *Aspergillus niger*

Aspergillus niger is a fungus and one of the most ordinary species of the genus *Aspergillus*. It causes an illness called black mold on convinced fruits and vegetables such as grapes, onions, and peanuts, and is an ordinary pollutant of food. It is everywhere in soil and is usually reported from inside environments, where its black colonies can be puzzled with those of *Stachybotrys*

Reference: http://www.wikipedia.org/wiki/Aspergillus_niger

Title: *Agroinfiltration*

Agroinfiltration is a technique in plant biology to persuade transient expression of genes in a plant or to create a preferred protein or antibodies. The advantage of agroinfiltration when compared to customary plant transformation is speed and expediency. Recombinant antibodies can be used to identify, treat and put off disease by exploiting their specific antigen-binding behavior. A large number of drugs currently in growth are recombinant antibodies and most of these are shaped in cultured rodent cells. Though such cells produce genuine functional products, they are expensive, difficult to scale-up and may hold human pathogens. Plants represent a gainful, suitable and safe option production system and are slowly gaining receipt.

Reference: http://www.wikipedia.org/wiki/Agroinfiltration

Title: *Bacteriophage*

Bacteriophages are an ordinary and varied group of viruses and are the most plentiful form of biological body in aquatic environments – there are up to ten times more of these viruses in the load than there are bacteria, attainment levels of 250,000,000 bacteriophages per millilitre of seawater. These viruses contaminate specific bacteria by binding to surface receptor molecules and then entering the cell. Within a short quantity of time, in some cases just minutes, bacterial polymerase starts translating viral mRNA into protein. These proteins go on to turn out to be either new virions within the cell, assistant proteins, which help meeting of new virions, or proteins involved in cell lyses. Viral enzymes help in the breakdown of the cell membrane, and in the case of the T4 phage, in just over twenty minutes after inoculation over three hundred phages could be free.

Reference: http://www.wikipedia.org/wiki/Bacteriophage