2009 Distinguished Public Lecture

The Neurobiology of Consciousness

Christof Koch PhD
California Institute of Technology (Caltech)

Author of The Quest for Consciousness

May 5, 2009
Retama Auditorium
1604 Campus UTSA

5:30p Reception
6pm Lecture
free parking
public welcome

Half a century ago, many did not think it was possible to understand the secret of life. Then Watson and Crick discovered the structure of DNA, forever changing biology. We are facing a similar pursuit in determining the material basis of consciousness. How does the unmistakable smell of dogs after they have been in the rain, or the awfulness of throbbing tooth pain emerge from networks of neurons and their associated synaptic processes?

In this public lecture, Dr. Koch will summarize what is known about the neurobiology of consciousness, argue that attention is distinct from consciousness, outline the limits to our knowledge, and describe ongoing experiments using visual illusions to manipulate the relationship between physical stimuli and their associated conscious percepts via fMRI and single unit recordings in human medial temporal lobe. He will conclude with a discussion of a promising information-theoretical approach to consciousness that is grounded in circuit complexity.

Christof Koch is the Lois and Victor Troendle Professor of Cognitive and Behavioral Biology, and Professor of Computation and Neural Systems, at Caltech.
Stigma, Secrecy & Medical History: What we can learn from Huntington’s Disease

The UTSA Neurosciences Institute, American Studies Program, & Honors College present:

Alice Wexler PhD
Author of
Mapping Fate
&
The Woman Who Walked into the Sea

November 10, 2009
Retama Auditorium UC 2.02.02
1604 Campus, UTSA

4:30pm Reception
5pm Lecture

Illnesses and disabilities perceived as hereditary, especially those affecting mental functions, have long been stigmatized in western culture. Still, in the US in the 19th and 20th centuries, the historical representations and experiences of families and individuals with inherited disorders have varied considerably across different social locales.

In this Lecture, Dr. Alice Wexler will draw on the example of Huntington’s disease, a late-onset inherited neurological and psychiatric illness, to suggest the ways in which both biology and history, including the eugenics movement of the twentieth century, helped shape the perception and experience of hereditary disease and disability in the past, and how this history may offer insights for policy in the present.

Alice Wexler is Research Scholar at the UCLA Center for the Study of Women. She is a recipient of the 2009 American Medical Writer's Association Book Award.

Sponsored by the UTSA Neurosciences Institute American Studies Program & Honors College
please see http://neuroscience.utsa.edu for more info
Expansions on a Dream: From Cause to Cure of Huntington’s Disease

The UTSA Neurosciences Institute presents:

Nancy Wexler PhD
Director, Hereditary Disease Foundation
Professor of Neurology & Neuropsychology
Columbia University

November 11, 2009
Main Building 0.104
1604 Campus, UTSA

5:30pm Reception
6pm Lecture

In 1968, a biomedical detective story began with one family’s personal heartache. It eventually stretched from the poorest villages of Venezuela to the most advanced research laboratories in the world.

In this public lecture, Dr. Nancy Wexler relates her personal and scientific journey toward discovery of the gene that causes the profoundly debilitating neurological disorder Huntington’s Disease, and the ongoing search for its cure.

Nancy Wexler is the Higgins Professor of Neuropsychology in the Departments of Neurology and Psychology at the College of Physicians and Surgeons at Columbia University and President of the Hereditary Disease Foundation.

Sponsored by the UTSA Neurosciences Institute and the UTSA Specialized Neuroscience Research Program
please see http://neuroscience.utsa.edu for more info
Studies in Venezuela led to the discovery of the gene for the crippling neurodegenerative disorder Huntington's Disease. Since then, studies of postmortem human brains, transgenic animals and cell cultures have led to a new understanding of the potential mechanisms underlying the disease. Now we have entered a new phase in the story of Huntington's, in which biomarkers of the disease are being discovered that will change the effectiveness of our interventions.

Dr. Young will discuss the clinical, genetic, pathological and pathogenic mechanisms that play a role in Huntington's disease, and how these findings are being applied to new clinical tools that have the potential for restoring the lives of those stricken with the disease, and those living at risk.

Anne B. Young is the Julieanne Dorn Professor of Neurology at Harvard Medical School.
Learned Birdsong & the Neurobiology of Human Language

Erich D. Jarvis PhD
Duke University Medical Center/ Howard Hughes Medical Institute

The brains and behavior of song-learning birds and humans have key similarities and differences. Vocal learners (including songbirds, parrots, hummingbirds, and humans), have a unique forebrain system that controls learned vocalizations (such as language in humans), whereas those species that produce unlearned, innate vocalizations have only a brainstem vocal system. Dr. Jarvis’s research finds that the song learning systems of the three distantly related vocal learning bird groups are all similar to each other, and each are embedded within a motor system involved in limb and body movements. These pathways also have parallels with those for spoken language in humans. Further, this behavioral and brain pathway convergence is associated with changes in neural connectivity and neural protection genes in song learning birds and humans.

In this lecture, Dr. Jarvis will describe a motor theory for the origin of vocal learning and other complex behavioral traits. He posits that an ancient brain system used to control movement and motor learning, such as learning how to walk, fly, or gesture. It duplicated and then diverged to directly connect to the brainstem vocal motor neurons that now generate learned song or spoken language. In this manner, the evolution of brain pathways for vocal learning may have evolved independently of a common ancestor in distantly related birds and in humans, but dependent on deep homology of pre-existing genes and a pre-existing motor learning pathway.

Dr. Jarvis is an HHMI Investigator and Associate Professor of Neurobiology at Duke University Medical Center.
Most people have experienced indigestion after a meal; if that meal included a novel taste, a lasting memory is often formed leading to avoidance of that taste in the future. Avoiding tastes that make you sick is of obvious advantage just as avoiding things that cut, burn or shock you are avoided or at least treated with caution. How does one learn to avoid bad situations?

In this lecture, Dr. Palmiter will describe how his laboratory is using exciting new viral and genetic manipulations in the mouse to directly access the neural circuits and signaling molecules that underlie acquisition of aversive memories. His lab has identified a neural circuit that suppresses appetite in response to “noxious” signals, like indigestion. Pairing artificial activation of specific neurons within this circuit with a novel taste, elicits avoidance for that taste in the future. Thus, his lab can generate a “false memory” of indigestion without the mouse ever becoming ill. Moreover, if his lab presents mice with a novel taste and prevents activation of those specific neurons in response to illness, the mice do not form a taste memory. Mice, like people, do not like to be shocked; mice respond to a shock, or environments associated with a shock, by ‘freezing in their tracks’. The same circuit that is used to learn to avoid foods that make a mouse sick is used to learn to ‘freeze’ in response to cues that predict a shock. Dr. Palmiter will discuss the challenge of understanding how the brain receives unpleasant sensations and creates memories that allow an animal to avoid or cope with those bad events.

*Dr. Palmiter is an HHMI Investigator and Professor of Biochemistry at The University of Washington School of Medicine, Seattle.*
The analogy of the adult brain as a computer is wrong. The adult brain is made of living cells with genomic diversity that can change their connections and numbers based on experience, a process called "neural plasticity." The same mechanisms that drive differences between species can change the DNA in each cell within an individual, leading to genomic diversity.

Dr. Gage's work concentrates on the adult central nervous system and unexpected plasticity and adaptability to environmental stimulation that remains throughout the life of all mammals. In addition, he models human neurological and psychiatric disease in vitro using human stem cells. His lab studies the genomic mosaicism that exists in the brain as a result of mobile elements that are active during neurogenesis.

Fred H. Gage, Ph.D., a Professor in the Laboratory of Genetics, joined The Salk Institute in 1995. He received his Ph.D. in 1976 from The Johns Hopkins University. He is a member of the most prestigious international scientific societies, and has served as President of both the Society for Neuroscience and the International Society for Stem Cell Research.
The hippocampus is essential to episodic memory, which is characterized by our ability to recall the spatial and temporal organization of events that constitute a specific past experience. An understanding of how the hippocampus supports episodic memory would benefit by using an animal model to identify neural coding mechanisms for the spatial and temporal organization of memories within the hippocampus.

With regard to spatial organization, I will describe a recent study using Representational Similarity Analysis on neural ensembles showing that the hippocampus creates a highly organized, hierarchical network representation of features of events and the places and contexts in which they occurred. With regard to temporal organization, I will present evidence that the hippocampus is critical for memory of order of events in unique experiences in animals, as it is in humans. Furthermore, I will describe recent evidence that hippocampal “time cells” (as contrasted with the famous hippocampal “place cells”) encode specific moments in the course of temporally extended experiences, and time cell ensembles encode specific memories and predict memory success. These findings support an emerging view that the hippocampus serves episodic memory by creating a scaffold for the organization of events within their spatial and temporal context, and the relevance of this model to cognitive aging will be discussed.
Parkinson’s disease is the second most common neurodegenerative disorder in the U.S. There are no proven strategies for preventing it or slowing its progression. But there is hope that a new treatment is on the horizon.

This talk will outline the rationale for a large Phase 3 clinical neuroprotection trial in early stage Parkinson’s disease patients with the drug isradipine that is being sponsored by the National Institutes of Health. The core idea behind the trial is that some neurons in the brain act as look-outs or sentries – they are always watching for events that could hurt or help us – and that this constant vigilance causes them to wear out as we age. The goal of the drug therapy is to remodel these neurons in a way that allows them to ‘take a break’ and rest without compromising their ability to do their job in the less threatening, more predictable world we live in today.

D. James Surmeier, PhD
Chair, Department of Physiology
Nathan Smith Davis Professor
Director, Morris K. Udall Center for Excellence in Parkinson’s Disease Research
Feinberg School of Medicine, Northwestern University

April 10, 2017
UC Ballroom I
1604 Campus,
UTSA

5:00p Reception
5:30p Lecture

free parking*
public welcome

*See http://neuroscience.utsa.edu for lecture details and free parking instructions

Sponsored by The UTSA Neurosciences Institute
The UTSA Neurosciences Institute presents its
2018 Distinguished Public Lecture

In Search of a Cure for Alzheimer’s Disease

George Perry, PhD
Professor of Biology
Semmes Foundation Distinguished University Chair in Neurobiology
Chief Scientific Officer, Brain Health Initiative
University of Texas at San Antonio

November 12, 2018
UC Ballroom I
1604 Campus,
UTSA

5:00p Reception
5:30p Lecture

free parking
public welcome

In fewer than 40 years, Alzheimer’s disease has transformed from a condition known by few outside of neurology to arguably the most impactful disease in the world. Nearly 6 million Americans are affected; annual costs are estimated at $277 billion and are growing rapidly as our population ages. The talents of thousands of neuroscientists and billions spent on research is contrasted with drugs which offer only moderate symptomatic relief rather than a cure.

Alzheimer’s disease is a complex neurodegenerative disease involving neuronal death, amyloidosis, cytoskeletal abnormalities, oxidative stress, mitochondria changes, metabolic abnormalities, each of which is being tested individually to explain the disease. In this lecture, Dr. Perry will present Alzheimer’s disease as a multifaceted condition that must first be understood in order to be cured.

George Perry is the Semmes Foundation Distinguished University Chair in Neurobiology at UTSA and the Chief Scientific Officer of the UTSA Brain Health Initiative. He is an authority on fundamental mechanisms of Alzheimer’s Disease, member of various prestigious scientific societies, and journals and recipient of numerous scientific awards.
Spinal muscular atrophy (SMA) Type I is the leading genetic cause of infant mortality globally. It results from a defect in the SMN1 gene, and about 1 in 50 Americans are genetic carriers. In the absence of a functional SMN1 gene, infants rapidly lose the motor neurons required for breathing and swallowing. By 24 months of age, they typically require permanent ventilation.

Dr. Brian Kaspar’s research on single-doses SMA gene therapy has informed an ambitious new strategy for a one-time, foundational gene-replacement therapy designed to repair the genetic root cause of SMA Type 1 in humans.

In his lecture, Dr. Kaspar will take us on his journey from bench to cure by giving us a window on the basic and translational research that has led to the first-in-human clinical trial of an SMA gene-therapy. He will discuss the process for determining dose and pre-clinical efficacy, as well the key safety studies that enabled this groundbreaking Phase 1/2 clinical trial.

Dr. Kaspar is Scientific Founder and Chief Scientific Officer at AveXis, Inc., a clinical-stage gene therapy company focused on the treatment of neurological diseases. He was formerly Associate Professor of Pediatrics & Neuroscience at The Ohio State University College of Medicine and the Grant Morrow, III, MD Endowed Chair in Pediatric Research at Nationwide Children’s Hospital.
Autism impacts 1 in 50 children, with boys 4 times more likely to be diagnosed than girls. It is a complex disorder characterized by repetitive behaviors, restricted interests and deficits in language and social communication. Research has revealed that genes linked to autism spectrum disorders regulate the function of connections between neurons in the brain.

Dr. Ted Abel’s studies on the genes linked to autism provide a neurobiological explanation for many of the challenges faced by individuals with autism as they seek to navigate the complex world that surrounds them.

In his lecture, Dr. Abel will discuss the specific molecular signaling processes he has identified that underlie reward learning selectively in males. Reward learning is critical for navigating the complex world around us and is mediated by specific neural circuits, termed cortico-striatal circuits, which are known to be altered in individuals with autism. Interestingly, these same neural circuits mediate the development of social communication, language, repetitive behaviors and restricted interests, all the very core symptoms of autism.

Edwin G. (Ted) Abel, Ph.D. is the founding director of the Iowa Neuroscience Institute & Chair of the Department of Neuroscience and Pharmacology in the Carver College of Medicine. He is a pioneer in defining the molecular mechanisms of long-term memory storage, and identifying how these processes go awry in neurodevelopmental and psychiatric disorders.
The UTSA Neurosciences Institute & Brain Health Consortium present

Spring 2022 Distinguished Public Lecture

APRIL 25TH, 2022

LOCATION:
Denman Ballroom
Student Union 2.01.28
1604 Campus UTSA

CRISPR Correction of Neuromuscular and Heart Disease

Eric Olson is the founding Chair of the Department of Molecular Biology at UT Southwestern Medical Center. He also directs the Hamon Center for Regenerative Science and Medicine and the Wellstone Center for Muscular Dystrophy Research at UT Southwestern. He holds the Robert A. Welch Distinguished Chair, the Pogue Chair Distinguished Chair in Cardiac Birth Defects and the Annie and Willie Nelson Professorship in Stem Cell Research.

Dr. Olson and his trainees discovered many of the key genes and mechanisms responsible for development and disease of the heart and other muscles. His most recent work has provided a new strategy for correction of Duchenne muscular dystrophy using CRISPR gene editing.

Dr. Olson will discuss the latest advances in CRISPR gene editing technology and its application to the correction of monogenic neuromuscular disorders. Opportunities and obstacles in the path to clinical translation will also be discussed.

Speaker: Eric Olson, PhD
Professor and Chair
UT Southwestern Medical Center