<table>
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<tr>
<th>Year</th>
<th>Moderator(s)</th>
<th>Title</th>
<th>Description</th>
<th>Objectives</th>
<th>Speakers</th>
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<tr>
<td>2019</td>
<td>Melissa Barker-Holik, MD, PhD; Angela Birnbaum, PhD; Helen Scherfan, PhD</td>
<td>Seizures in Seniors: How Do We Identify New and Innovative Therapies for this Growing Patient Demographics?</td>
<td>This workshop will address the specific therapeutic needs of the fastest growing patient demographics with epilepsy: the elderly. Despite the greater incidence of epilepsy in the elderly, as well as the overlapping pathology and low risk and benefits associated with antiepileptic drug treatment, many patients are not adequately treated.</td>
<td>To discuss the unique needs of elderly patients with epilepsy; to understand the challenges of therapeutic development and novel targets for treatment; to share evidence-based guidelines for the management of seizures in the elderly.</td>
<td>Eric Rosenberg, MD; Silvia M. Bagdade, PharmD, PhD; and David Alcantara Gonzalez, MD</td>
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<td>2019</td>
<td>Melanie Boly, PhD</td>
<td>Neurostimulation and Neuroimaging of Subcortical Arousal Circuits in Epilepsy</td>
<td>This workshop will address new exciting data and techniques for imaging networks and uncovering mechanisms of therapeutic modulation by subcortical stimulation devices in epilepsy. We will provide a comprehensive update of the neuroscience of arousal and consciousness as a general field, and as applied to epileptic seizure networks, in the context of other altered states such as anesthesia, coma, or sleep. We will focus on high field fMRI and multi-site stimulation in animal models to allow unprecedented visualization of cortical and limbic network changes with subcortical stimulation - with the ability to modulate seizure threshold or reverse seizure-induced loss of consciousness.</td>
<td>To describe the role of subcortical mechanisms in epileptic seizures, and sleep; to discuss specific case studies of subcortical stimulation - with ability to modulate seizure threshold or reverse seizure-induced loss of consciousness.</td>
<td>Melanie Boly, PhD; Jin Hyung Lee, PhD; and Hal Blumenfeld, MD</td>
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<td>2019</td>
<td>Hajime Takano, PhD</td>
<td>Recent Advances in Microelectrode Array Technology and Its Applications</td>
<td>This workshop will address emerging microelectrode array (MEA) technologies and their applications. Transient electrode arrays are of interest to many researchers who work on in vitro neuronal imaging and optogenetic stimulation. In addition, in late 2018, the novel, extremely high density silicon electrode array, “Neurops”， became available to the general research community. Emerging innovations are not limited to the forefront of in vivo electrophysiology.</td>
<td>To discuss the advantages and limitations of MEA concepts also in vitro assay systems.</td>
<td>Flavia Vitale, PhD; Timothy Harris, PhD; and Ikuo Suzuki, PhD</td>
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<td>2019</td>
<td>Douglas Coulier, PhD</td>
<td>Circuit Based Therapies in Epilepsy</td>
<td>This workshop will address novel circuit therapies in epilepsy. Conventional systemic antiepileptic drug therapy immunes the entire brain, affecting both normal and pathologic circuits, causing both therapeutic and negative outcomes. In addition, the neuronal circuit disruptions underlying phenotypic expression in epilepsy are mechanistically complex. Targeting individual symptoms has proven insufficiently effective in ameliorating seizures or restoring cognitive function in chronic epilepsy.</td>
<td>To recognize the promise of circuit-based therapies in controlling seizures and correcting epilepsy co-morbidities; to describe several novel intervention strategies targeting specific neuronal circuits effective in the control of seizures and co-morbidities in epilepsy; to recognize how circuit-based therapies in epilepsy overcome some common issues in current therapeutic strategies.</td>
<td>Robert F. Hunt, PhD; Julia Kahn, BS; Dimitri Kulmann, MD</td>
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<td>2019</td>
<td>Jeanne T. Fair, PhD; and Daniel Lowenstein, MD</td>
<td>Gut Microbiome and Epilepsy: Paradigm Shifts for Understanding and Treating Epilepsy</td>
<td>This workshop will address the timely and emerging topics of the gut microbiota in epilepsy. Several publications within last year have suggested that an unhealthy gut microbiota may play a role in the development and progression of epilepsy. The role of the gut microbiota and gut-brain axis interactions have never been discussed at previous IW workshops. Here, we will discuss the role of the gut microbiome in the development of epilepsy and the potential implications of these discoveries for treatment.</td>
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<td>Gloria Choi, PhD; Elain Hsiao, PhD; and Audrey Macarati, PhD</td>
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<td>2019</td>
<td>Gemma Canit, PhD; and Heather Moffett, MD, PhD</td>
<td>Poison Exons: From Development and Disease to Therapeutic Target</td>
<td>This workshop will address the role of poison exons in neuronal development, in the development of genetic epilepsies through aberrant splicing, and as potential therapeutic targets for genetic epilepsies. Poison exons, or nonsense mediated decay (NMD) exons, are small exonic regions that when spliced into an RNA transcript lead to premature truncation of a protein. Inclusion of poison exons occurs during specific times in neuronal development and splicing occurs in a cell-specific manner. Of the many genes implicated in epilepsy, these include CACNA and make them candidates for harboring disease-causing mutations, but also for targeted RNA-based therapeutics.</td>
<td>To discuss the role of poison exons in neuronal development and their potential as therapeutic targets.</td>
<td>Gemma Canit, PhD; Leri L. Isom, PhD; and Xiaochang Zhang, PhD</td>
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<td>2019</td>
<td>Chris Dula, PhD; and Laura Ewold, PhD</td>
<td>Seeing the Forest or the Trees: Does Synaptogenetic and Cellular Heterogeneity Support Pathological Network Activity Level?</td>
<td>This workshop will address the topic of oligonucleotide-based therapies for the treatment of epilepsy. Oligonucleotides are artificial DNA sequences which work by binding to target RNAs (e.g. mRNA) to disrupt their function. This IW will focus on exploring this nascent field in epilepsy, which offers virtually unlimited potential to treat genetic and acquired epilepsies and is now moving to clinical trials. It will provide an overview of the state of the art, specific use of ODNs in distinct forms of epilepsy and open the pipeline from proof-of-concept to clinical trials as well as explore risks and limitations.</td>
<td>To explore new and emerging applications of oligonucleotides for the treatment of epilepsy and the mechanisms that control poison exon inclusion/exclusion; to recognize the role of poison exon inclusion/exclusion in genetic epilepsies, identify patients that should be screened for these variants, and the sequencing approaches needed to identify these variants; to describe how eliminating the inclusion of poison exons is a novel therapeutic target disease with a special focus on the benefits of this approach (e.g., almost limitless precision therapy) and limitations (delivery) of oligonucleotide “antisense” therapies for epilepsy ranging from those in late proof-of-concept, those in preclinical development and those now in clinical trials; to discuss and exchange ideas on the road to translation, the current and future “pipeline”; to view the future of how diseases could be translated using nanotechnologies.</td>
<td>Heinz Beck, MD; Ka Yang, PhD; and Kirshna Jayant, PhD</td>
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<td>2019</td>
<td>Vaidhna Krishman, MD, PhD</td>
<td>On Melancholia in Epilepsy: Mechanistic Insights into the Comorbidity of Epilepsy with Depression and Anxiety Disorders</td>
<td>This workshop will address recent translationally relevant mechanistic insights into the occurrence of mood and anxiety disorders in persons with epilepsy, which contribute substantially to disability and impairments in quality of life and which remain a key NINDS/ADHS research benchmark. We will emphasize preclinical developments in our understanding of the shared anatomical, cellular and molecular substrates that may predispose individuals to epilepsy and comorbid mood/anxiety disorders. Following our presentations, we will host a discussion that will resolve around strategies for risk stratification and treatment.</td>
<td>To identify key neuromotorial projections/pathways implicated in the genesis of mood/anxiety disorders in the context of epilepsy; to appreciate the rich and diverse mechanisms by which stress may impact seizure likelihood or seizure thresholds; to acknowledge the utility of novel behavioral assays currently implemented to assess depression- and anxiety-like behavior in rodent models of epilepsy spectrum disorders.</td>
<td>Vaidhna Krishman, MD, PhD; Raman Sankar, MD, PhD; and Jamie L. Maguire, PhD</td>
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<td>2019</td>
<td>David C. Hensch, PhD</td>
<td>Oligonucleotide Therapies for Epilepsy: A New Era in Precision Medicine?</td>
<td>This workshop will address the topic of oligonucleotide-based therapies for the treatment of epilepsy. Oligonucleotides are artificial DNA sequences which work by binding to target RNAs (e.g. mRNA) to disrupt their function. This IW will focus on exploring this nascent field in epilepsy, which offers virtually unlimited potential to treat genetic and acquired epilepsies and is now moving to clinical trials. It will provide an overview of the state of the art, specific use of ODNs in distinct forms of epilepsy and open the pipeline from proof-of-concept to clinical trials as well as explore risks and limitations.</td>
<td>To identify the community on the issues of using oligonucleotides to target disease with a special focus on the benefits of this approach (e.g., almost limitless precision therapy) and limitations (delivery) of oligonucleotide “antisense” therapies for epilepsy ranging from those in late proof-of-concept, those in preclinical development and those now in clinical trials; to discuss and exchange ideas on the road to translation, the current and future “pipeline”; to view the future of how diseases could be translated using nanotechnologies.</td>
<td>Cristina Reschke, PhD; Steve Petrou, PhD; and Clare Walshe, MD, PhD</td>
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**Note:** The table above summarizes the workshops and their key topics, objectives, and speakers. Each workshop focuses on a specific area related to epilepsy, such as therapeutic strategies, circuit therapies, and genetic aspects.
The Storm before the Quiet: Basic mechanisms of SUDEP

This workshop will address how ultra-slow changes in membrane potential and extracellular voltage may reveal common mechanisms that link epileptic seizures with migraine. Migraine is a comorbidity of epilepsy, and spreading depression (the depression in spread of spreading depression, and spreading depression; 3) Perform long-term DC recordings in animal models and clinically in human patients.

2018

Jeffrey Noebels, MD, PhD

The Storm before the Quiet: Basic mechanisms of SUDEP

1) Recognize the linkages between epilepsy and migraine; 2) Explain the similarities and differences between spreading depression; and spreading depression. The workshop will show how DC recordings are required to measure directly the ultra-slow components of spreading depression and postictal depression, and will discuss new techniques that can be used chronically in animal models and clinically in human patients.

2019

F. Edward Dudek, PhD, and Bruce Gluckman, PhD

Ultra-slow and DC Recordings to Study Seizures, Migraine, and Spreading Depression

This workshop will address how ultra-slow changes in membrane potential and extracellular voltage may reveal common mechanisms that link epileptic seizures with migraine. Migraine is a comorbidity of epilepsy, and spreading depression (the depression in spread of spreading depression, and spreading depression; 3) Perform long-term DC recordings in animal models and clinically in human patients.

2018

Jeffrey Noebels, MD, PhD

Do rodent models of generalized absence seizures represent human disease or simply normal neuronal activity?

1) Identify descending forebrain pathways activated by seizures that may transmit detrimental signals to brain circuits necessary for regulation in the medulla. 2. Define pathophysiology of specific SUDEP linked genes that affect cardiovascular and respiratory functions. 3) Develop experimental models of SUDEP to examine the role of specific SUDEP linked genes in the mechanism of SUDEP. 

2019

John Huguenard, PhD

Eliana Scemes, PhD

Astrocytes in Epilepsy: cause or consequence?

It is known that some of the best treatments for epilepsy and migraine are directed at astrocytes, the most abundant cell type in the CNS. Recent studies have shown that astrocytes play a critical role in the pathogenesis of both seizures and migraine. However, the mechanisms by which astrocytes contribute to these disorders are not well understood. This workshop will explore the role of astrocytes in the pathogenesis of epilepsy and migraine, and how targeting astrocytes may offer new therapeutic strategies for these disorders.
Inflammation and epilepsy: where do we stand and where do we go from here?

2017

Luca Bartolini

Inflammation and epilepsy: where do we stand and where do we go from here?

This session will focus on discussing the crucial role of inflammation in epileptogenesis. This mechanism is not targeted by conventional antiepileptic drugs and may contribute to the high number of refractory epilepsy cases. We will present the results of a cross-sectional study analyzing the potential role of mRNA and protein expression and the immune response in children with various forms of seizures, we will discuss evidence for inflammation in the pathophysiology of epilepsy, including immunological aspects of epileptogenesis and role of biomarkers; finally, we will exchange views on approaches to novel therapeutic trials of antiepileptogenic and immunomodulatory treatments under clinical development.

1. Assess the role of oral infections in the pathogenesis of epilepsy and as a possible trigger for inflammatory mechanisms and as a possible trigger for inflammatory mechanisms and as a possible trigger for inflammatory mechanisms and as a possible trigger for inflammatory mechanisms; 2) Evaluate future directions for trials of antiseptic and anti-inflammatory therapies in epilepsy.

2017

Mikhail Hildebrand

Somatic mutation: the 'hidden genetics' of brain malformations

Somatic mutations are genetic alterations acquired in somatic cells, especially in germline tissues of the brain. This is a very interesting field of research, especially in the context of brain tumors. Somatic mutations can be an important cause of brain malformations and other neurological disorders. In this talk, we will discuss the role of somatic mutations in the development of brain malformations and their implications for the development of novel therapies and biomarkers.

3) Downregulation of KCC2 in mutiple models of seizure–related status epilepticus

2017

Peyman Golshani

In-vivo imaging of network dynamics in epilepsy

New tools now enable high speed simultaneous imaging of activity in hundreds of neurons in behaving animals. We will highlight 3 notions of how these new approaches can be used to fill the activity pattern of large populations of precisely identified neurons in models of temporal lobe and generalised epilepsy. These approaches can be used to discover dysfunction in key cell types that may be driving initiation of seizures or network dysfunction during cognition in epilepsy.

2017

Christina Gross

MicroRNA-induced silencing in epilepsy: potential therapeutic target and biomarker

MicroRNAs (miRNAs) are small non-coding RNAs that play important roles in the regulation of gene expression. They are involved in various biological processes, including development, metabolism, and disease. In this talk, we will discuss the role of microRNAs in the regulation of gene expression in epilepsy, and their implications for the development of novel therapies and biomarkers.

1) Highlight how current studies have elucidated the activity patterns of large populations of precisely identified neurons in models of temporal lobe and generalised epilepsy. These approaches can be used to discover dysfunction in key cell types that may be driving initiation of seizures or network dysfunction during cognition in epilepsy.

2017

Sarah Maddox

Data-driven computational modeling of epileptic network structure

Recent efforts in computational modeling of large-scale brain dynamics have begun to take a data-driven approach, incorporating structural and/or functional brain networks derived from patient data into the model. In this workshop, we will focus on approaches that are either structurally or functionally based and are sensitive to brain dynamics. The workshop will include presentations by some of the leaders in this new direction of computational research (Vitor Jima, Markus Kaiser, and Ankth Khamlichi), and discuss how the integration of data-driven network research with brain imaging data and computational modeling can lead to a better understanding of the underlying mechanisms of epilepsy.

1) Highlight how data-driven network-based modeling can lead to the development of personalized models of epilepsy; 2) Discuss the potential of applying machine learning algorithms to identify biomarkers and develop new approaches for understanding the mechanisms of epilepsy.

2017

Bret Smith

Are animal models of post-traumatic epilepsy translational?

The incidence of post-traumatic epilepsy is increasing, and there is a need for better models to understand the mechanisms and develop treatments for this condition. In this workshop, we will focus on the translatability of animal models of post-traumatic epilepsy and discuss the challenges and opportunities for translational research. The workshop will include presentations by some of the leaders in this area, including Robert Hunt and Annapurna Poduri, and discuss how these models can be used to understand the mechanisms of post-traumatic epilepsy and develop new treatments.

3) To discuss the different novel functions recently described for microRNAs and their impact on the development of post-traumatic epilepsy.

2017

Joaquim Lugo

From inflammation to phagocytosis: how microglia shape vulnerable neuronal networks in epilepsy

When their microenvironment is challenged with events such as seizures microglia cells can develop an inflammatory or phagocytic response. It is widely known that microglia-mediated neuroinflammatory mechanisms play a role in the emergence of seizures and in epilepsy; however, the contribution of their phagocytic response to seizures and epilepsy is less known. Emerging evidence suggests that microglial phagocytic signaling cascades contribute to the activity-dependent modulation of neuronal networks under physiological and pathological conditions. This workshop will begin by first reviewing the contributions of the microglial-mediated neuroinflammatory alterations to the hippocampal network.

1) To discuss recent progress in mechanisms and functions of microRNA-induced silencing in epilepsy; 2) Discuss potential and challenges of microRNA-mediated mechanisms for therapy and biomarker development in epilepsy.

2017

Chris Dulla

NMDA receptors in epilepsy: mutations, orbital circuits, and personalized medicine

NMDA receptors are critical for synaptic plasticity and memory, but also contribute to multiple aspects of abnormal neuronal hyperexcitability, neuronal homeostasis, and seizure generalization. Last, disrupted glutamate signaling acting through NMDA receptors can induce excitotoxicity, interfere with important spontaneous network activity in the developing brain, and disturb cellular and circuit maturation. Targeting NMDA receptors therapeutically, however, has been a significant challenge due to the complexity of the receptor's functions.

1) To discuss microglial-mediated inflammatory and phagocytic mechanisms to the neuronal connectivity in the hippocampus in both human and experimental epilepsy.

2017

Shilpa Kadam

KCC2 hypofunction in epilepsy: developing novel therapeutic strategies to modulate KCC2-CO-transporter 2 (KCC2) function

The KCC2 chloride co-transporter is the chief Cl- extruder in CNS neurons. KCC2 is known to co-localize with GABAA receptors but is also expressed at excitatory synapses. KCC2 plays significant roles in dendritic spine morphogenesis and glutamatergic synaptic function. KCC2 activity is both positively and negatively regulated by many pathways including phosphorylation of different sites on its protein structure. KCC2 hypofunction has now been shown by many independent research groups to play a critical role in the emergence of seizures by multiple independent research groups. More interestingly, enhancing or preserving KCC2 function during seizures can positively impact seizure outcomes and therefore highlight the need to develop true enhancers of KCC2 as a novel therapeutic strategy.

1) To discuss the concept and development of microRNA-mediated regulation in epilepsy

1) Understand the role neuronal potassium chloride cotransporter in seizure suppression; 2) To discuss how a complex systems approach can be applied to genetics and neurobiology and thereby understand the concept of system level mechanisms.

1) To understand the complex systems scientific approach and be able to identify differences between this approach and a traditional reductionist approach. To understand how a complex systems approach can be applied to genetics and neurobiological mechanisms.

2) Discuss the importance of recent identification of disease-causing KCC2 mutations in humans with epilepsy in infancy and migrating focal seizures and the role of KCC2 in proper brain development.

2) To discuss the role neuronal potassium chloride cotransporter in seizure suppression.
2016 Long-Jun Wu
Glial mechanisms of epilepsy

In the normal brain, glial cells, specifically astrocytes and microglia, contribute to neurovascular coupling by participating in the regulation of vascular tone and neuronal excitability. Recent evidence suggests that glial cells also play a role in the pathophysiology of epilepsy. The mechanisms underlying the altered glial function in epilepsy are not well understood. This symposium will provide an overview of the current understanding of glial cell function and their role in epilepsy. It will also discuss recent advances in the use of glial cells and their derivatives as therapeutic tools for epilepsy.

Moderators: Julia Kahn, Ph.D., Christophe Bernard, Ph.D.; Speakers: Long-Jun Wu, Ph.D., Devin Binder, Ph.D., Karen Wipke, Ph.D.

2016 Helen Scharfman
Role of aberrant neurogenesis in epilepsy

Neurogenesis in the adult brain is established in all mammals including humans, where it has been suggested to contribute to the development of epilepsy and related disorders. Recent evidence has demonstrated that aberrant neurogenesis may play a role in the pathogenesis of epilepsy. This symposium will focus on the role of neurogenesis in epilepsy, including its potential as a therapeutic target.

Moderators: Helen Scharfman, Ph.D.; Speakers: Helen Scharfman, Ph.D., Kyung-Ok Cho, M.D., Ph.D., Steve Danner, Ph.D.

2016 Viji Santhakumar
Peripheral imaging biomarkers in epilepsy

The role of peripheral imaging biomarkers in the diagnosis and management of epilepsy is an area of increasing interest. This symposium will discuss the use of peripheral imaging biomarkers in the management of epilepsy, including their potential for early detection and monitoring of therapeutic effects.

Moderators: Viji Santhakumar, Ph.D.; Speakers: Jenny Hoeh, Ph.D., Jack Parent, M.D., Robert Hunt, Ph.D.

2016 Jenny Hoeh
Emerging strategies using stem cells to prevent epilepsy

Stem cell research offers new opportunities for the development of therapies for epilepsy. This symposium will focus on emerging strategies using stem cells to prevent epilepsy, including recent advances in stem cell technologies and their potential for the treatment of childhood epilepsy.

Moderators: Jenny Hoeh, Ph.D.; Speakers: Jenny Hoeh, Ph.D., Jack Parent, M.D., Robert Hunt, Ph.D.

2016 Christian Dullé
Novel intracellular signaling cascades and epilepsy: is there an untapped therapeutic potential?

Intracellular signaling cascades play a critical role in the development and progression of epilepsy. Recent advances in understanding these cascades offer new opportunities for the development of targeted therapies. This symposium will focus on novel intracellular signaling cascades and their potential therapeutic applications.

Moderators: Christian Dullé, Ph.D.; Speakers: Gaia Novarino, Ph.D., Michèle Jacob, Ph.D., Angelique Bordy, Ph.D.

2016 Dr. Jana Velikova
Autistic traits in epilepsy models: Why, when and how?

Autistic traits have been observed in epilepsy models, but the underlying mechanisms are not well understood. This symposium will focus on the relationship between autistic traits and epilepsy, including potential mechanisms for their co-occurrence and the implications for treatment.

Moderators: Jana Velikova, M.D., Ph.D.; Speakers: Melissa Benson, Ph.D., Jürgen Schieken, M.D., Pierre-Pascal Lenck-Santini, Ph.D.

2016 Devin K. Binder (co-organizer: Viji Santhakumar)
Neurovascular Unit in Seizures and Epilepsy

The neurovascular unit (NVU) is a complex network of neurons, astrocytes, pericytes, and perivascular cells. Recent discoveries have highlighted the critical role of the NVU in the regulation of cerebral blood flow and metabolism, and its dysfunction has been implicated in the pathogenesis of epilepsy. This symposium will focus on the role of the NVU in epilepsy, including mechanisms and potential therapeutic targets.

Moderators: Devin Binder, Ph.D., Viji Santhakumar, Ph.D.; Speakers: Nicola Martin, Ph.D., Todd Flax, Ph.D., G. Campbell Tenkay, Ph.D.

2016 Dan Xu, Ph.D. and Soo-Ikyung Koh, M.D. Ph.D.
Novel immunomodulatory Therapies in Epilepsy

The immune system plays a critical role in the pathogenesis of epilepsy. Recent advances in immunology have revealed new targets for the development of immunomodulatory therapies. This symposium will focus on novel immunomodulatory therapies for epilepsy, including their potential for the treatment of refractory epilepsy.

Moderators: Dan Xu, Ph.D., Soo-Ikyung Koh, M.D. Ph.D.; Speakers: Dan Xu, Ph.D., Eileen A. Anderson, M.D. Ph.D., Tereza Baroza, Ph.D.
Whole exome sequencing (WES) has become a practical and affordable clinical test for many patients with presumed epilepsy, given that activation of PI3K/AKT/mTOR and FMRP is enhanced by seizures and in some models of chronic epilepsy (Cohen et al., 2015; Wilcox et al., 2015; Wilcox and Krueger, 2015). Whole exome sequencing in the epilepsies: in epilepsy, ion channels, and behavioral comorbidities.

Inflammation in epilepsy: From brain chemistry to clinical interventions. Dr. Schiff will present a unified theory based on experimental and computational modeling work has given the field new insight into how seizures begin, are sustained, and terminate. Furthermore, novel imaging, electrophysiological, and biochemical studies have identified metabolic pathways which affect excitability. In this workshop we will discuss the hypothesis that slow metabolic processes underlie the idiopathic nature of seizures. Dr. Christoph Bernard (INSERM) will present the role of slow molecular, including mitochondrial-mediated, processes as a common denominator between comorbidities and comorbidities with metabolic origin. However, substantial evidence suggest that pathological inflammatory signaling also may contribute to the epileptogenic process. The main objectives of the session are to: 1) Describe the interplay between extracellular potassium concentration and excitatory neuronal activity; 2) Understand the role of different potassium channels in different cell types in shaping seizure dynamics; 3) Discuss novel data identifying mechanisms by which metabolic changes lead to altered excitability. The overall goal of this Investigator’s Workshop is to identify critical barriers and potential solutions to developing an antiepileptogenic drug, using tuberous sclerosis complex (TSC) as a model disease for testing such an approach. Such increases in extracellular potassium can in turn alter the activity of neurons. In this Investigator’s Workshop, we will highlight recent advances in understanding the mechanical relationship between potassium concentration and seizures. The session will be chaired by Omar Ahmed, a young investigator who has worked extensively on inhibitory-excitatory neuronal signaling in human brain tissues by Drs. Steven Schiff, Ali Agha, and Bernardo Rudy, all renowned cellular and computational physiologists with massive amounts of experience in understanding seizure dynamics. Dr. Schiff will present a unified theory based on experimental and computational genomics, drug discovery, and translational medicine.

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1. Highlight novel information about epileptogenesis, focusing on unexplained data; 2. Introduce concepts from animal studies, e.g., neurosciences, biochemistry, and epigenetics; 3. Inform our understanding of the epileptogenic process.

Kevin Eis, Jack Parent, Lori Isom

1. Provide an introduction to the methodology of neurological disease modeling with iPSCs; 2. Demonstrate the utility of patient-derived neurons to model epilepsy.

Christopher Reid, Dennis Dlugos, Peter Crino

To familiarize participants with respiratory, cardiac and arousal mechanisms for SUDEP.

Gordon F Buchanan, MD, PhD, Jeffrey L Noebels, MD, PhD, George R Robinson, MD, PhD

To facilitate discussions on large-scale collaborations among three national and international consortia with complementary approaches to epilepsy gene discovery. 2. To review the development and application of novel analytical approaches that have been created in response to a need to unravel the genetics of rare epilepsies and the common general genetic epilepsies (GGEs). 3. To discuss emerging results of gene discovery research and potential clinical applications.

Ssale Petrovski, Roland Krause, Patrick Coutoise

- To describe the unique opportunities presented through large-scale collaborations among three national and international consortia with complementary approaches to epilepsy gene discovery. 2. To review the development and application of novel analytical approaches that have been created in response to a need to unravel the genetics of rare epilepsies and the common general genetic epilepsies (GGEs). 3. To discuss emerging results of gene discovery research and potential clinical applications.

Mohamed Elwazi, MD, George Washington University, Washington, DC; Dominique Durand, PhD, Case Western Reserve University, Cleveland, Ohio; G. Campbell Teskey, PhD, University of Calgary, Calgary, Alberta, Canada

No credentials at this time. 1. Uncover novel genetic variants that are understood as an ictal core generating excitatory barrages, and a surrounding ictal penumbra where inhibition restrain seizure progression. 2. Explain how brain function can become compromised widely, both within the core and also in the penumbra. Specifically, we will discuss the impact on the subcortical arious systems. 3. Discuss how these new discoveries can optimize associated therapies and novel experimental designs where low frequency stimulation can be investigated thoroughly.

Andrew Trelawny (Newcastle University, UK), Hal Blumenfeld (Yale University), Catherine Schoen (Columbia University)

- To describe the unique opportunities presented through large-scale collaborations among three national and international consortia with complementary approaches to epilepsy gene discovery. 2. To review the development and application of novel analytical approaches that have been created in response to a need to unravel the genetics of rare epilepsies and the common general genetic epilepsies (GGEs). 3. To discuss emerging results of gene discovery research and potential clinical applications.

Mohamed Elwazi, MD, George Washington University, Washington, DC; Dominique Durand, PhD, Case Western Reserve University, Cleveland, Ohio; G. Campbell Teskey, PhD, University of Calgary, Calgary, Alberta, Canada

1. Highlight novel discoveries in the molecular pathogenesis of infantile spasms. 2. Investigate signaling pathways which are disrupted in IS models. 3. Identify novel therapeutic strategies for treating IS.

Chris Dula, Jeff Noebels, John Swann

- To highlight the role of extracellular molecular actions in the physiological mechanisms by which excessive cellular energy demand activates master regulators of gene expression.

Katja Kubic, Jong Rho, R. Coppari (McNight as backup)- expert on ischemia.
Mitochondrial abnormalities in malformations of cortical development

Mitochondrial abnormalities have been reported in patients with treatment-resistant epilepsy. This workshop will explore neurological, neuropathological, and biochemical methods for evaluation of mitochondrial abnormalities in resected brain tissue. Investigation of mitochondrial abnormalities in cortical malformations may provide new therapeutic targets, biomarkers, and understanding of epileptogenesis in patients with intractable epilepsy.

Ed Cooper
The KCNQ2-associated epilepsy and encephalopathy spectrum: bedside to bench

Ed Cooper, Baylor College Medicine

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Ed Cooper

2013

Chris Dulla
Astrocyte control of the extracellular environment – pathological and therapeutic implications

Alexander Rotenberg, Chris Dulla, Daniela Kaufer, Harold Sontheimer

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2013

Michael Wong
Dendritic injury in epilepsy: mechanisms and consequences

Anne Anderson, John Swayne, Michael Wong

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2013

Aristea Galanopoulou, Karen Wilcox
Translating severe terminology, modeling, and detection from rodents to humans: is consensus possible?

Aristea Galanopoulou, Karen Wilcox, Discussants: Solomon Moshe, Alexis Aristarkhov, Brian Litt

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2013

Steve White, PhD and Nicholas Crino, M.D., Ph.D.
Translational Neurogenetics of Hemimegalencephaly

Harvey Samet M.D., Erin Heimann Ph.D., Gregory Heuer M.D., Ph.D.

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<table>
<thead>
<tr>
<th>Year</th>
<th>Name</th>
<th>Title</th>
<th>Co-authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>2012</td>
<td>Alica Goldman</td>
<td>Predictive Genes, Basic Mechanisms, and Clinical Biomarkers of SUDEP</td>
<td>Jeff Noebels, Jack Parent (speakers)</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Alica Goldman, MD, PhD (introducer), George Richardson, MD, PhD (speaker)</td>
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<td></td>
<td></td>
<td></td>
<td>Lisa Bateman (speaker)</td>
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<tr>
<td>2012</td>
<td>Scott Baraban &amp; Ed Dudek</td>
<td>Swimming toward a new path for drug discovery in epilepsy: an open discussion of novel approaches using zebrafish, mice and induced pluripotent stem cells</td>
<td>4th speaker: Jack Parent</td>
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<td></td>
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<td></td>
<td>H. Steve White, Kevin Staley, Scott Baraban</td>
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<tr>
<td>2012</td>
<td>Talke Z. Baram, MD, PhD</td>
<td>It takes two to tango: Dance of neuronal ion channels and their auxiliary subunits</td>
<td>Nick Poolos? Ed Cooper?</td>
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<td></td>
<td>Yow Noam (PhD Dec 2011), Lara Iosam, PhD, Dane Chetkovich, MD, PhD</td>
</tr>
<tr>
<td>2012</td>
<td>Scott Baraban</td>
<td>What’s Next? A Young Investigator Workshop</td>
<td>Andy Cole, Merci De Curtis, Ed Dudek, John Ebersole, Brian Uit</td>
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<td>Maximo Avoli, Kevin Staley, Christophe Bernard + 4th speaker Elaine Willi</td>
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<tr>
<td>2012</td>
<td>Christophe Bernard</td>
<td>What does it mean to be interictal spikes - do we have a predictive value?</td>
<td>Maximo Avoli, Kevin Staley, Christophe Bernard + 4th speaker Elaine Willi</td>
</tr>
<tr>
<td>2012</td>
<td>Sam Berkovic</td>
<td>Massively Parallel Sequencing in Epilepsy</td>
<td>Peter De Jonghe (Antwerp); co-chair with Sam Berkovic</td>
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<td>Heather Mefford (Seattle), David Goldstein (Duke), Ingo Helbig (Kiel, Germany, also a junior investigator)</td>
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<tr>
<td>2012</td>
<td>Aristeo Galanopoulos</td>
<td>Validation of epilepsy biomarkers in humans: goals, successes, challenges</td>
<td>4th speaker: John W. Miller, M.D., Ph.D., Director Regional Epilepsy Center, UW, 2nd TBA</td>
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<td>Ramondo D’Ambrosio, Ph.D., Remen Diaz-Aristizabal, M.D., Ph.D., Harry Sontheimer, Ph.D.</td>
</tr>
</tbody>
</table>

**Notes:**
- The table includes information from the 2012 American Epilepsy Society Annual Meeting.
- The page contains a mix of titles, descriptions, and discussions related to various topics in epilepsy research, including genetic factors, therapeutic approaches, and biomarker validation.
- The discussions cover a range of topics, from predictive gene studies to the utilization of novel approaches using zebrafish and induced pluripotent stem cells.
- The event featured talks by a diverse group of experts, including MDs, PhDs, and other professionals involved in epilepsy research.
- The aim appears to be fostering collaboration and advancing the field through innovative research and discussions.
- The meeting highlighted the importance of interdisciplinary approaches and the need for predictive markers in epilepsy management.
- The setting was conducive to networking and knowledge sharing among the scientific community.
- The table entry reflects the comprehensive nature of the meeting, encompassing both theoretical and practical aspects of epilepsy research.
The extracellular matrix in epilepsy

Recent data has suggested that one of the regulators of synaptic plasticity is the extracellular matrix. It appears to stabilize the neural microenvironment and synaptic plasticity. Following head injury, stroke and status epilepticus there is a breakdown in the extracellular matrix due to upregulation of proteases. Recent work in transgenic animals has suggested that the destruction of the extracellular matrix contributes to epileptogenesis.

Paulaite Micarea will talk about the perineuronal net following status epilepticus at different ages, Chris Ilomemadu will talk about NMDA- NR2 subunit expression in the extracellular matrix and contribution to epileptogenesis. This is an important: have been back and forth with several European and Japanese groups and still working on the final part.

Brain pH in the generation and suppression of seizures

Pharmacological interventions that affect brain pH are critical for the generation and suppression of seizures. Recent studies have revealed a key role for cerebral acidosis, which can be induced by hypoglycemia, ischemia, or histamine, in the generation of both acute and chronic seizures. The mechanisms underlying this pH-dependent epileptogenesis are crucial for developing effective therapeutic strategies.

Saúl Mullén, Steven Petrus, Kai Kaia

Cell signaling pathways and epileptogenesis

Cell signaling pathways play a crucial role in the regulation of neuronal excitability and synaptic plasticity. Disruptions in these pathways can contribute to the development and maintenance of epilepsy. Recent studies have shed light on the complex interplay between cell signaling and epileptogenesis, highlighting potential therapeutic targets for the treatment of this devastating condition.

Andrey M. Mazurkiewicz, M.D., Ph.D., Gregory L. Holmes, M.D., Carl Stafstrom, M.D., Ph.D.

Neurobiological Mechanisms of Comorbidities

Understanding the neurobiological mechanisms underlying comorbid conditions is essential for improving treatment strategies. The workshop will focus on elucidating the mechanisms that link epilepsy with mood disorders, sleep disorders, and other psychiatric conditions. By integrating basic science knowledge and clinical insights, participants will gain a comprehensive understanding of these complex interactions.

Daniela Kaufner, Amy Brooks-Kayal, Michael Wong

The importance of subcortical structures in epilepsy

Subcortical structures, including the basal ganglia, thalamus, and brainstem, play a crucial role in the generation and propagation of seizures. Recent advances in neuroimaging techniques have allowed for the precise localization of these structures, providing insights into their functional contributions to epileptic activity.

Russell Ferland, PhD, Hal Blumenfeld, MD, PhD, Carl Fairgeld, PhD

Novel therapeutic target identification for seizures from the ketogenic diet research

The ketogenic diet has been a subject of extensive research in epilepsy, providing insights into potential therapeutic targets. Through the analysis of observational data, a candidate model for the mechanism of action has been proposed, which could serve as a foundation for future research.

Manish Patel

The "methylation hypothesis": does epigenetic chromatin modification play a role in epileptogenesis?

Epigenetic modifications, such as DNA methylation, play a critical role in the regulation of gene expression and neuronal plasticity. In epilepsy, these modifications can contribute to abnormalities in neuronal function and connectivity, ultimately leading to seizure activity.

Irfan A. Qureshi, Mark F Mehlert, Sebastian Jesberger

Mechanisms of high frequency activity in epileptic foci

High frequency activity, also known as fast ripples, is a hallmark of epileptic activity and is associated with pathological neuroplasticity. Understanding the mechanisms underlying fast ripples is crucial for developing targeted therapeutic strategies.

John Jefferys

Neuronal death and pediatric epilepsy: Any effect of early-life seizure? A cause-or-not-of-later epilepsy?

Pediatric seizures can have long-term consequences for neuronal development and the emergence of epilepsy. The workshop will focus on recent studies that have shed light on the potential impact of early-life seizures on later-onset epilepsy.

Ed Dudek, Anatol Bragin

What’s Next? A Young Investigator Workshop

The Young Investigator Workshop aims to foster the development of young researchers in the field of epilepsy. Participants will have the opportunity to present their work, receive feedback from experienced colleagues, and network with peers.

Arne Anderson, Ivan Soltesz

TBD - recent EF prodacx awardee, TBD - recent EF prodacx awardee, TBD - recent EF prodacx awardee

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Adenosine and epilepsy - promising start into a Neuronal Coupling and Excitability (NCHE), Molecular Neurobiology, and Pharmacology 2009 Kevin Staley Imaging synchrony with activity-dependent reorganization of the brain’s network. This will be important to seizure generation and epileptogenesis. Most investigators do not have access to high bandwidth human EEG recordings or the computational resources required to analyze them. There are many unanswered questions regarding the significance of high bandwidth EEG in epilepsy that cannot be addressed by qualitative studies of small data sets (unpublished observations). The NCHE has developed a high bandwidth EEG database and is developing tools that will allow directed analysis of specific questions.

2009 Jack Parent and Scott Baraban Stem Cells and Epilepsy 2010 Astrid NEHLIG, INSERM U 666, France 2010 Christophe Bernard Epigenetic mechanisms of epileptogenesis 2010 Anatol Bragin 2009 Theodore H. Schwartz Optical imaging of epilepsy - hemodynamic methods and computational models 2010 Deliev Beison 2010 Lisa R. Merlin, MD Inducible regulation of mGlul-mediated epileptogenesis Group I mGluR activation has been shown to have epileptogenic potential. A variety of intracellular pathways converge to internalize receptors, with both positive and negative effects on this mGluR-induced excitation, suggesting the possibility for targeted antiepileptic therapies

2010 Verena C Wimmer The emerging role of the axon initial segment in epileptogenesis Decision what to hit for a potential action potential (AP) or not. In most neurons, this decision is made at the axon initial segment (AIS), a specialized domain of the axon proximal to the soma where APs are initiated. Recently, the AIS has been in the spotlight of scientific interest because of its important roles in neuronal output and protein trafficking. The AIS also serves as a critical synaptic and nuclear compartmentalization and its cell type specific properties and unique forms of plasticity have only recently been explored. Here, we will review the central role of the AIS in the excitability of neurons and its contribution to epileptogenesis.

2010 Asla Pitkanen Peptidopathy, chemoencephalopathy or bad network: what causes epilepsy in Alzheimer’s disease? For a long time, patients with Alzheimer’s disease (AD) have been known to have myoclonic seizures. Recently it was hypothesized that daily cognitive fluctuations in AD could relate to the occurrence of undiagnosed hyperexcitability. This idea is supported by a recent study demonstrating that a subpopulation of patients with AD and myoclonus have high frequency oscillations (HFOs) in the range of 50-100 Hz. The focus of this workshop is recent translational research on adenosine, a powerful anticonvulsant neuromodulator in epilepsy, and new insights into the ongoing metabolic and astrocytic regulation of adenosine, as well as advances in adenosine-releasing cells and polymers, bringing new promise to adenosine-based therapeutic strategies.

2010 Elizabeth Powell "Interneuronopathies" - Diversity in the phenotypes of genetic mutations that alter intracellular calcium handling in epilepsy. Hyperexcitability has been associated with both up- and downregulation of Ih. Despite the relatively well-characterized role of Ih in cellular excitability, its contribution to network activity in epilepsy is not well characterized. Ih-based methods using stem-cell cultures are dependent on the donors and the memories of their human development.

2010 Anato Bregin Early Detection of Epileptogenesis and Search for Effective Protective Treatment in Experimental Models and in Clinic Recent evidence suggests that high frequency oscillations, microseizures and human microelectrode recordings may be precursors of epileptic events. In some circumstances these effects precede the onset of seizure activity. Whether optical imaging has provided insights into neurovascular coupling mechanisms and changes in the extracellular environment associated with microseizures in the human brain is uncertain. Nevertheless, optical mapping will be clinically useful to supplement or even replace electrical recordings. There are many unanswered questions regarding the significance of high bandwidth EEG in epilepsy that cannot be addressed by qualitative studies of small data sets (unpublished observations). The NCHE has developed a high bandwidth EEG database and is developing tools that will allow directed analysis of specific questions.

2010 Roth Otten, Ph.D. Neurobiological Mechanisms in Genetic Focal Epilepsies: The Case of LGI1 For a long time, patients with Alzheimer’s disease (AD) have been known to have myoclonic seizures. Recently it was hypothesized that daily cognitive fluctuations in AD could relate to the occurrence of undiagnosed hyperexcitability. This idea is supported by a recent study demonstrating that a subpopulation of patients with AD and myoclonus have high frequency oscillations (HFOs) in the range of 50-100 Hz. The focus of this workshop is recent translational research on adenosine, a powerful anticonvulsant neuromodulator in epilepsy, and new insights into the ongoing metabolic and astrocytic regulation of adenosine, as well as advances in adenosine-releasing cells and polymers, bringing new promise to adenosine-based therapeutic strategies.

2010 Astrid NIEHL, INSERM U 666, Strasbourg, France The endocannabinoid system and temporal lobe epilepsy The endocannabinoid system (ECS) is a complex and redundant network of cell-cell interactions that is involved in several physiological and pathological processes. ECS interactions are particularly strong in the hippocampus, where the ECS is the most predominant in the brain. ECS interactions are particularly strong in the hippocampus, where the ECS is the most predominant in the brain.

2010 David Prince Control of synapse formation and epilepsy Synapses are the main channels for signal transmission in the brain. Recent findings suggest that the properties of individual synapses can be altered by experience and environmental factors. These changes in synaptic properties are believed to underlie learning and memory. The study of synaptic plasticity is essential for understanding the mechanisms underlying these processes.

2009 Andre Lagrange Epilepsy and Depression: The Two Faces of ROR1 (an Development Neurogenesis) Depressive symptoms are present in 50% of patients with epilepsy. The reason for this is unknown. However, it is possible that the same mechanisms that cause epilepsy and depression are involved. In this study, we investigated the expression of ROR1 in the hippocampus of mice with epilepsy and depression. Our results suggest that ROR1 expression is increased in both conditions.

2009 Jack Parent and Scott Baraban Stem Cells and Epilepsy 2009 Theodore H. Schwartz Optical imaging of epilepsy - hemodynamic methods and computational models 2009 Kevin Staley Imaging synchrony with activity-dependent reorganization of the brain’s network. This will be important to seizure generation and epileptogenesis. Most investigators do not have access to high bandwidth human EEG recordings or the computational resources required to analyze them. There are many unanswered questions regarding the significance of high bandwidth EEG in epilepsy that cannot be addressed by qualitative studies of small data sets (unpublished observations). The NCHE has developed a high bandwidth EEG database and is developing tools that will allow directed analysis of specific questions.
The impact of neuroinflammation on neuronal excitability and excitotoxicity

Neuroinflammation plays a critical role in the onset and maintenance of epilepsy. This is supported by the recent finding that genetic deletion of astrocyte Chemoattractant Receptor Fragment 5 (CRF5) results in a neuroprotective effect in a model of status epilepticus. Understanding the mechanisms through which inflammation modulates neuronal excitability and excitotoxicity is essential for the development of novel anti-epileptic therapies.

Neuro-glial signaling and epileptogenesis

Epileptogenesis involves the interaction between neurons and glial cells, which can contribute to the development of epilepsy. Recent studies have shown that glial cells play a crucial role in the initiation and progression of seizures. For example, astrocytic swelling and reactive gliosis have been implicated in the spread of electrical activity in the brain and the development of epilepsy.

Tuberous sclerosis: a genetic disorder with seizures

Tuberous sclerosis is a genetic disorder characterized by seizures, developmental delay, and skin lesions. Recent studies have identified mutations in the TSC1 and TSC2 genes, which lead to the production of abnormal proteins that disrupt normal cellular function. This disorder is associated with neuronal hyperexcitability and is a potential model for understanding the mechanisms underlying epilepsy.

Febrile seizures and their regulation

Febrile seizures are a common feature of many familial epilepsy syndromes. Recent research has shown that the development of febrile seizures is influenced by a combination of genetic and environmental factors. Understanding the mechanisms through which fever triggers seizures is crucial for the development of effective treatment strategies.

Epilepsy and anticonvulsant injury

The use of anticonvulsant drugs is a common treatment for epilepsy, but these medications can also cause neuronal injury. Studies have shown that anticonvulsants can induce apoptosis and necrosis in neuronal cells, leading to the development of secondary epilepsy. Understanding the mechanisms through which anticonvulsants induce neuronal injury is essential for the development of safer and more effective treatments.

The dentate "gate": what is it, what regulates it?

The dentate gyrus has been hypothesized to function as a "gate" that regulates the relay of pathological, synchronous afferent input into the hippocampus. Recent studies have shown that the dentate gyrus is involved in the propagation of seizures, and its dysfunction is associated with the development of epilepsy. Understanding the mechanisms through which the dentate gyrus regulates epileptogenesis is crucial for the development of novel anti-epileptic therapies.

Human/animal neuroimaging in the study of epileptogenesis

Human/animal neuroimaging techniques are being used to study the mechanisms underlying epileptogenesis. These techniques allow researchers to visualize the structural and functional changes that occur in the brain during the development of epilepsy. Understanding the mechanisms through which these changes occur is essential for the development of effective treatment strategies.

The catastrophic epilepsies of childhood: a review

The catastrophic epilepsies of childhood are among the most devastating and least understood neurologic disorders. Recent studies have shown that these disorders are associated with genetic mutations and neurodevelopmental abnormalities. Understanding the mechanisms through which these disorders develop is crucial for the development of effective treatment strategies.
Raised extracellular potassium: convulsant and anticonvulsant mechanisms

Membrane depolarization and neuronal bursting are known to be brought on by raised potassium. Diffusion of extracellular potassium can cause spread of epileptogenic activity. There is now increasing evidence that raised potassium can also play a very significant role in seizure cessation. This can be through depolarization blockades, loss of plasticity and bestowing a refractory state which may benefit the cessation of epileptiform activity.

The proliferating brain and its role in epileptogenesis

The focus will be on neuronal and glial progenitors, astrocytes, activated microglia and proliferating endothelial cells describing their morphological and functional changes in epileptogenic brain. The implications of these changes on neuronal network excitability and epileptogenesis. Experimental evidence in animal models and observations in human patients will be discussed.

Surrogate markers in animal models of epilepsy

It is necessary to have a method to detect seizures in animal models. This can be achieved by monitoring EEG and other electro-physiological variables. A variety of surrogate markers have been proposed including: electrophysiological metrics, ERPs, local field potentials, neurochemical measures and receptor binding. The use of these markers in assessing the efficacy of experimental treatments will be reviewed.

Mitochondrial trafficking and morphology in healthy and injured neurons

Mitochondrial shape changes during basal neuronal activity (Bennett), Mitochondrial trafficking and morphology in healthy and injured neurons (I.J. Reynolds, Merck), Mitochondrial dysfunction in temporal lobe epilepsy (W. Kuhn, Univ. Bern), Mitochondrial dysfunction during neuronal activations in the anoxia-hypoxia neural model (T. Longmore)

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The role of opioids in epilepsy

Do seizures beget seizures?

Gene Therapies and Epilepsy: New Therapeutic Directions

New Therapeutic Directions

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H-channelopathy workshop

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Epileptogenesis of cortical dysplasia: Compare and contrast animal models with mechanisms gleaned from human studies

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<tr>
<td>2004</td>
<td>Kevin M. Kelly, M.D., Ph.D.</td>
<td>Models of epilepsy in aging</td>
<td>Eric M. Biallock, Ph.D., Peter R. Patrylo, Ph.D., Kevin M. Kelly, M.D., Ph.D.</td>
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<td>2004</td>
<td>Mauroso Avuti, M.D., Ph.D.</td>
<td>Plasticity of chloride transport and GABA signaling</td>
<td>Melanie Woodin, Ph.D., Richard Milles, Ph.D., Francisco J. Alvarez-Leefmans, M.D., Ph.D.</td>
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<td>Jong M. Rho, M.D.</td>
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<td>Margaret P. Jacobs</td>
<td>Creating new animal models of the childhood epilepsies</td>
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<td>James O. McNamara, M.D.</td>
<td>Neurotrophins: Epileptogenesis and electroconvulsive seizures</td>
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<td>The role of tonic GABA inhibition and control of brain excitability</td>
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