

UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE PATENT TRIAL AND APPEAL BOARD

OVID THERAPEUTICS INC.,
Petitioner,

v.

MARINUS PHARMACEUTICALS, INC.,
Patent Owner.

Case IPR2024-00726
Patent 11,110,100 B2

DECLARATION OF RAMAN SANKAR, M.D., PH.D.

Declaration of Raman Sankar, M.D., Ph.D.
in support of Petition for an Inter Partes Review
of U.S. Patent No. 11,110,100 B2

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EXHIBIT LIST

Exhibit	Description
1001	U.S. Patent No. 11,110,100 B2 to Marinus Pharmaceuticals, Inc. (“the ‘100 patent”)
1002	Declaration and Curriculum Vitae of Dr. Raman Sankar, M.D., Ph.D. (the “Sankar Declaration”)
1003	U.S. Patent No. 11,395,817 B2 (“During”)
1004	NCT03350035 Clinical Trial Study Details, ClinicalTrials.gov (Nov. 17, 2017), https://clinicaltrials.gov/study/NCT03350035?term=NCT03350035&rank=1&tab=history&a=1 (last visited Sept. 28, 2023) (“NCT03350035”)
1005	J. Tsai et al., Phase I study to determine the pharmacokinetics, pharmacodynamics, and safety of IV ganaxolone in healthy adults, Final Programme of the 6th London-Innsbruck Colloquium on Status Epilepticus and Acute Seizures, 74-75 (Apr. 6-8, 2017) (“Tsai 2017”)
1006	J. Tsai et al., Phase I Study to Determine the Pharmacokinetics, Pharmacodynamics, and Safety of IV Ganaxolone in Healthy Adults 80 Annals of Neurology S77-78 (Supp. 20 2016) (“Tsai 2016”)
1007	Ganaxolone Phase 1 Data Supports Progressing to Phase 2 in Patients with Status Epilepticus, Clinical Leader (Oct. 19, 2016), https://www.clinicalleader.com/doc/ganaxolone-phase-data-progressing-phase-patients-epilepticus-0001 (last visited Sept. 28, 2023) (“Clinical Leader 2016”)
1008	R. Eugene Ramsay et al., Pharmacokinetic and Pharmacodynamic (PK/PD) Relationship of Intravenous Ganaxolone in Refractory Status Epilepticus (Nov. 25, 2019), https://aesnet.org/abstractslisting/pharmacokinetic-and-pharmacodynamic-(pk/pd)-relationship-of-intravenous-ganaxolone-in-refractory-status-epilepticus (“Ramsay 2019”)

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1009	A. Husain et al., Intravenous Ganaxolone Achieves Rapid and Dose-Dependent Sustained Improvement in EEG Seizure Burden in Patients with Refractory Status Epilepticus, 37 J. Clin. Neurophysiol. 332 (July 2020) (“Husain 2020”)
1010	Treatment of Refractory Status Epilepticus Prevents Need for Induced Coma with Intravenous Anesthetics in Phase 2 Trial, Practical Neurology (Oct. 18, 2019), https://practicalneurology.com/news/treatment-of-refractory-status-epilepticus-prevents-need-for-induced-coma-with-intravenous-anesthetics-in-phase-2-trial (last visited Sept. 28, 2023) (“Practical Neurology 2019”)
1011	R. Eugene Ramsay, Pharmacokinetic and Pharmacodynamic (PK/PD) Relationship of Intravenous Ganaxolone in Refractory Status Epilepticus, Marinus Pharmaceuticals (Dec. 7, 2019), https://marinuspharma.com/wp-content/uploads/2020/09/AES-2019-GNX-in-RSE-Ph2-Poster-vFINAL.pdf (last visited Sept. 28, 2023) (“AES Poster 2019”)
1012	Doodipala S. Reddy et al., Experimental Models of Status Epilepticus and Neuronal Injury for Evaluation of Therapeutic Interventions, 14 Int’l J. Mol. Sci. 18284-318 (2013) (“Reddy”)
1013	Marinus Pharmaceuticals Receives FDA Orphan Drug Designation for Ganaxolone IV to Treat Status Epilepticus, Marinus Pharmaceuticals (Apr. 15, 2016), https://ir.marinuspharma.com/news/news-details/2016/Marinus-Pharmaceuticals-Receives-FDA-Orphan-Drug-Designation-for-Ganaxolone-IV-to-Treat-Status-Epilepticus/default.aspx (last visited Sept. 28, 2023)
1014	Marinus Corporate Presentation June 2023, Marinus Pharmaceuticals (June 2023), https://s25.q4cdn.com/443656056/files/doc_financials/2023/Corporate-Deck-June-FINAL.pdf (last visited June 19, 2023)
1015	Eugen Trinka et al., A definition and classification of status epilepticus – Report of the ILAE Task Force on Classification of Status Epilepticus, 56 Epilepsia 1515-1523 (2015) (“Trinka 2015”)

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1016	Henrikas Vaitkevicius, Intravenous ganaxolone for the treatment of refractory status epilepticus: Results from an open-label, dose-finding, phase 2 trial, 63 Epilepsia 2381-91 (2022) (“Vaitkevicius 2022”)
1017	John D. Hixson, Stopping Antiepileptic Drugs: When and Why?, 12 Current Treatment Options in Neurology 434-442 (2010) (“Hixson 2010”)
1018	Simon Shorvon, The Management of Status Epilepticus, 70 J. Neurol. Neurosurg. Psychiatry ii22-ii27 (Supp. II 2001) (“Shorvon 2001”)
1019	Durgul Ozdemir et al., Efficacy of continuous midazolam infusion and mortality in childhood refractory generalized convulsive status epilepticus, 14 Seizure 129-132 (2005) (“Ozdemir 2005”)
1020	Edward P. Monaghan et al., Initial Human Experience with Ganaxolone, a Neuroactive Steroid with Antiepileptic Activity, 38 Epilepsia 1026-1031 (1997) (“Monaghan 1997”)
1021	Richard B. Carter et al., Characterization of the Anticonvulsant Properties of Ganaxolone (CCD 1042; 3 α -Hydroxy-3 β -methyl-5 α -pregnan-20-one), a Selective, High-Affinity, Steroid Modulator of the γ -Aminobutyric Acid _A Receptor, 280 J. Pharmacol. Experimental Therap. 1284-1295 (1997) (“Carter 1997”)
1022	WO 2007/062266 A2 (“Shaw”)
1023	Michael R. Sperling et al., Randomized, double-blind, placebo-controlled phase 2 study of ganaxolone as add-on therapy in adults with uncontrolled partial-onset seizures, 58 Epilepsia 558-564 (2017) (“Sperling 2017”)
1024	Marinus Pharmaceuticals Doses First Subject in Phase 1 Clinical Trial for Ganaxolone IV, Marinus Pharmaceuticals (June 22, 2016), https://ir.marinuspharma.com/news/news-details/2016/Marinus-Pharmaceuticals-Doses-First-Subject-in-Phase-1-Clinical-Trial-for-Ganaxolone-IV/default.aspx (last visited Sept. 28, 2023) (“Marinus’s Phase 1 Press Release”)

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1025	Julia Tsai et al., Phase 1 Study to Determine the Pharmacokinetics, Pharmacodynamics, and Safety of IV Ganaxolone in Healthy Adults (“2016 Phase 1 Poster”)
1026	Eileen Broomall et al., Pediatric Super-Refractory Status Epilepticus Treated with Allopregnanolone, 76 Ann Neurol. 911-915 (2014) (“Broomall 2014”)
1027	Dorota Zolkowska et al., Intramuscular allopregnanolone and ganaxolone in a mouse model of treatment-resistant status epilepticus 59 Epilepsia 220-227 (Supp. 2 2018) (“Zolkowska 2018”)
1028	Henrikas Vaitkevicius et al., First-in-man allopregnanolone use in super-refractory status epilepticus, 4 Annals of Clinical Translational Neurol. 411-414 (2017) (“Vaitkevicius 2017”)
1029	Excerpts from the File History of the ’100 Patent
1030	Raman Sankar, Treatment of status epilepticus: Physiology, pharmacology, and future directions, Epilepsia Open 1-8 (May 2023), https://onlinelibrary.wiley.com/doi/epdf/10.1002/epi4.12725 (“Sankar 2023”)
1031	U.S. Provisional Application No. 62/882,648 (“the ’648 Provisional Application”)
1032	WO 2021/026124 A1 (“WO ’124”)
1033	Marinus Pharmaceuticals Presents Clinical Data of Ganaxolone in Pediatric Epilepsies at Antiepileptic Drug and Device Trials XIII Conference, Marinus Pharmaceuticals (May 18, 2015), https://ir.marinuspharma.com/news/news-details/2015/Marinus-Pharmaceuticals-Presents-Clinical-Data-of-Ganaxolone-in-Pediatric-Epilepsies-at-Antiepileptic-Drug-and-Device-Trials-XIII-Conference/default.aspx (last viewed Sept. 28, 2023)
1034	Geoffrey M. Currie, Pharmacology, Part 2: Introduction to Pharmacokinetics, 46 J. Nuclear Med. Tech. 221-30 (Sept. 2018) (“Currie 2018”)

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1035	Excerpts from the File History of U.S. Patent Application No. 15/673,737
1036	141st Annual Meeting of the American Neurological Society, Annals of Neurology, S1-S265, https://onlinelibrary.wiley.com/doi/10.1002/ana.24759 (last accessed Feb. 8, 2024)

I. Introduction

1. I, Raman Sankar, M.D., Ph.D., have been retained by Ovid Therapeutics Inc. (“Ovid” or “Petitioner”) as an independent expert in the field of clinical neurology. My qualifications in this area are set forth in my curriculum vitae in **Appendix A**. I am being compensated for the time I spend on this matter, but no part of my compensation depends on the outcome of this proceeding.

II. Professional Background and Qualifications

2. I am a neurologist with over 35 years of experience treating people with epilepsy and other neurological disorders. I see approximately 40-50 patients per month who are suffering from epilepsy. I currently have around 100-120 active patients under my care, including those who have suffered from status epilepticus (“SE”).

3. I am currently a Professor of Neurology and Pediatrics and Chief Emeritus of Pediatric Neurology at the David Geffen School of Medicine at the University of California, Los Angeles (“UCLA”).

4. I am also an attending neurologist at UCLA’s Mattel Children’s Hospital and the UCLA Medical Center Hospital and Clinics, providing patients with consult and in-patient services and epilepsy/long-term monitoring.

5. I earned a B.S. in Chemistry in 1965 and a B.S. Tech. in Technology of Pharmaceuticals and Fine Chemicals in 1968 from the University of Bombay, and a

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Ph.D. in Pharmaceutical Sciences (Medicinal Chemistry) in 1974 from the University of Washington. From 1974-1975, I conducted postdoctoral research at the University of Washington School of Pharmacy. I was an Assistant Professor of Medicinal Chemistry from 1975-1980 and an Associate Professor from 1980-1982 at Xavier University College of Pharmacy. I was also an Adjunct Assistant Professor of Pharmacology at Tulane Medical School from 1979-1986.

6. I earned an M.D. in 1986 from Tulane Medical School. From 1986-1988, I was a Pediatric Intern-Resident at Children's Hospital of Los Angeles. From 1988-1989, I was a Resident in the Department of Neurology at the UCLA School of Medicine. From 1989-1991, I was a fellow in the Division of Pediatric Neurology at the UCLA School of Medicine. I am licensed to practice medicine in California. I am a Diplomate of the American Board of Psychiatry and Neurology with a special qualification in Child Neurology.

7. I became a Professor of Neurology at UCLA in 2005. Previously, I was a Clinical Instructor of Pediatric Neurology at the UCLA School of Medicine from 1991-1992, an Assistant Professor at the UCLA School of Medicine from 1992-1999, and an Associate Professor at the UCLA School of Medicine from 1999-2005. I also served as the Chief and Rubin Brown Endowed Chair of the Division of Pediatric Neurology at the UCLA School of Medicine from 2005-2022.

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8. I have served on the Editorial Board of several journals related to epilepsy and neurology, including *Epilepsia*, *Epilepsy & Behavior*, *Journal of Pediatric Epilepsy*, *Contemporary Neurology*, and *Frontiers in Bioscience*.

9. I have performed extensive research in the fields of epilepsy and neurology, publishing over 200 peer-reviewed journal articles and book chapters. Additionally, I have sponsored over 60 candidates for post-doctoral residency and 25 postdoctoral fellows.

10. I have received professional awards for my clinical work, research, and teaching, including the Founders Award of the American Epilepsy Society, which is the highest honor bestowed by the society and recognizes outstanding lifetime achievement in the field of epilepsy. I have also received awards and recognition for my clinical work, research, and teaching, including election as a Fellow of the American Epilepsy Society (FAES) in 2018, election to the American Pediatric Society in 2013, appointment to the Commission on Neurobiology of the International League Against Epilepsy (ILAE) in September 2009, and election as a Fellow of the American Academy of Neurology (FAAN) in 2004. My research has been supported by grants from the National Institutes of Health, the Epilepsy Foundation of America, the Epilepsy Foundation of Greater Los Angeles, and awards from Valeant Pharmaceuticals, Sigma-Tau Pharmaceuticals, Inc.,

Cyberonics, Inc., and Johnson & Johnson Pharmaceutical Research Institute. Teaching awards that I have received include the Excellence in Teaching Award for the Department of Neurology from Resident Physicians in 2004 and the Lawrence Ferring Faculty Award for Teacher of the Year at the Xavier University College of Pharmacy in 1982.

III. Materials Considered

11. I have considered all the documents I cite in this Declaration and all the documents cited in the Petition for an Inter Partes Review of U.S. Patent No. 11,110,100 (“the ’100 patent”) in forming my opinions. I refer to the prior art references and other documents cited in this Declaration using the same exhibit numbers and terminology as presented in the Petition. I may consider additional documents and information in forming any supplemental opinions. To the extent I consider additional documents or information, including any expert declarations in this proceeding, I may offer further opinions.

IV. Background of the Technology

A. Status Epilepticus

12. Epilepsies and seizure disorders fall within the field of neurology and cover a variety of neurological disorders characterized by abnormal electrical activity in the brain that causes seizures. A seizure refers to an atypical electrical discharge in the brain that results in changes in consciousness, sensation, and

behavior. Ex. 1012 at 18285. Epilepsy is a chronic condition characterized by recurring, unprovoked seizures. Ex. 1012 at 18285.

13. Status epilepticus (“SE”) is a specific type of seizure disorder that is defined as continuous seizure activity or multiple seizures lasting longer than 30 minutes without regaining consciousness. Ex. 1012 at 18287. SE is a life-threatening condition that is associated with high mortality and, if not treated immediately, could result in brain damage, cognitive impairment, or death. Ex. 1013 at 1. SE is known to be “the second most common neurologic emergency in the U.S.” with “150,000 cases per year.” Ex. 1014 at 22. As a result, SE is generally treated in a hospital or emergency room setting.

14. SE can be characterized by various behavioral signs or symptoms that evolve over time (i.e., semiology). For example, convulsive SE may result in excessive abnormal muscle contractions and/or impaired mental status. SE can also have electroencephalographic correlates detectable on an electroencephalogram (“EEG”) recording. Ex. 1015 at 1519. The results of the EEG can affect the choice and aggressiveness of treatment, prognosis, and clinical approaches. Ex. 1015 at 1518. Moreover, the EEG pattern and semiology are highly dynamic in SE and may change over short periods of time in certain patients. Ex. 1015 at 1518.

15. SE does not fall neatly within the category of seizure disorders—it is a neurologic emergency that has limited and drastic treatment options. Ex. 1016 at 2382. Antiseizure medicines (“ASMs”) are the first line of defense against SE, but 30-57% of patients fail to respond to first-line therapy. Ex. 1016 at 2382. Moreover “53-55% of patients with SE do not respond to either first- or second-line treatment.” Ex. 1016 at 2382 These patients are generally provided an additional round of second-line ASMs or are placed in a medically-induced coma. Ex. 1016 at 2382; *see also* Ex. 1012 at 18287 (reporting up to 40% of SE patients suffer from refractory SE and fail to respond to standard antiepileptic treatments).

16. For those patients who have refractory SE (“RSE”) because they do not respond to first or second-line ASMs, the next treatment options involve administering certain drugs using an intravenous bolus dose followed by continuous infusion for a period of around 12 hours, along with monitoring the patient’s EEG readout for seizure activity and adjusting the rate of infusion as necessary to control seizures. This would be followed by tapering of the drug. A bolus dose is a certain amount of the drug administered within a specific amount of time to quickly raise the drug’s plasma concentration in the blood to an effective level. Then, continuous infusion is used to maintain that plasma concentration level. The use of a bolus dose followed by continuous infusion is common in emergency situations, where starting

with a continuous infusion would take too long to reach a steady-state plasma concentration. The continuous infusion is then tapered (i.e., reduced or decreased) to avoid the risk of withdrawal seizures or recurrence of SE. Ex. 1017 at 439 (“Intuitively, it is inappropriate to abruptly discontinue any AED because of an increased risk of withdrawal seizures or even status epilepticus.”).

17. For example, in 2001, propofol was used to treat RSE according to the following administration:

2 mg/kg *iv bolus*, repeated if necessary, and then followed by a *continuous infusion* of 5-10 mg/kg/h initially, reducing to a 1-3 mg/kg/h. When seizures have been *controlled for 12 h*, the drug dosages should be *slowly tapered* over 12h.

Ex. 1018 at ii24 Fig. 2 (emphasis added). Similarly, thiopental was administered, in conjunction with monitoring EEG, to treat RSE as set out below:

100-250mg *iv bolus* given over 20 s, with further 50 mg boluses every 2-3 min until seizures are controlled, followed by a *continuous iv infusion* to maintain a burst suppression pattern on the EEG (usually 3-5 mg/kg/h). Thiopental should be *slowly withdrawn 12 h after the last seizure*.

Ex. 1018 at ii24 Fig. 2 (emphasis added).

18. In 2009, researchers studied the efficacy of midazolam for treating childhood refractory generalized convulsive SE. Ex. 1019 at 129. In this study, subjects with RSE were administered midazolam as a “0.2 mg/kg . . . bolus, followed

by a continuous infusion starting at 1 mg/kg/min up to 5 mg/kg/min increasing by 1 mg/kg/min every 15 min until complete control of seizures was achieved.” Ex. 1019 at 130. The midazolam infusion rate was “gradually decreased until tapering was completed when patient had remained seizure free for a period of 24 h.” Ex. 1019 at 130. EEG was used “after the seizures had been controlled to monitor the suppression of seizure discharge.” Ex. 1019 at 130.

B. Development of Allosteric Modulators of GABA_A to Treat Seizure Disorders, Including SE

1. Ganaxolone

19. Ganaxolone is a neuroactive steroid that allosterically modulates the γ -aminobutyric acid type A (GABA_A) receptor complex in the brain. Ex. 1020 at 1026. Researchers have evaluated ganaxolone for its antiseizure effects for over 20 years. *See, e.g.*, Ex. 1020; Ex. 1021.

20. In 1997, researchers at Baxter, recognizing ganaxolone’s potential for antiepileptic activity, reported initial experience with ganaxolone in humans. *See generally* Ex. 1020. In the study, 96 healthy volunteers were administered ganaxolone orally with doses ranging from 50 to 1,500 mg, and the researchers “systematically characterized” the pharmacokinetics (“PK”) of ganaxolone and documented adverse events associated with the drug. Ex. 1020 at 1026. The PK analysis revealed a “linear and proportional increase in the area under the curve

(AUC) and $C_{\max}$ values with increasing dose within the expected therapeutic dose range.” See Ex. 1020; at 1026, 1031 (finding that the “[p]harmacokinetic analysis demonstrated that ganaxolone administered either as single doses or after once-daily dosing for 14 days results in predictable and proportional plasma concentrations over the dose range of 50-500 mg/daily”).) The maximum daily dose administered in the study was 1,500 mg/day. Ex. 1020 at 1031.

21. In 2007, Marinus evaluated different dosage forms of ganaxolone for treating epilepsy and other central nervous system disorders, as described in Shaw. Ex. 1022 at ¶¶ [0002], [00174]. For example, Marinus administered 200 mg of ganaxolone as an oral suspension (50 mg/mL) to six healthy subjects to analyze ganaxolone’s pharmacokinetic properties. Ex. 1022 at ¶ [00546]. This resulted in a “mean $C_{\max}$ of 37 ± 25 ng/ml and an $AUC_{(0-24)}$ of 184 ± 104 ng*h/ml.” Ex. 1022 at ¶ [00546].

22. In 2017, Marinus reported the results of a Phase 2 study (that had occurred earlier) of the safety and efficacy of ganaxolone as an add-on therapy in adults with partial onset seizures, demonstrating that 1,500 mg/day ganaxolone was effective at reducing partial-onset seizure frequency. Ex. 1023 at 558, 561 (“Decreases in baseline in mean weekly seizure frequency were observed in all weeks except week 7 in the ganaxolone group.”), 562 (“ganaxolone at a dose of

1,500 mg/day resulted in a significant reduction in mean weekly seizure frequency compared to placebo”). Ganaxolone was titrated starting at 600 mg/day for 2 days, followed by 900 mg/day for 2 days, 1,200 mg/day for 2 days, and then 1,500 mg/day, after which the 1,500 mg/day dose was administered for an 8-week maintenance period. Ex. 1023 at 559. The study found that the doses of ganaxolone were “generally safe and well tolerated” and exhibited “no clinically relevant laboratory or [electrocardiography (“ECG” or “EKG”)] changes during the study.” Ex. 1023 at 558, 563. The study also notes that it was known that “[f]ollowing oral administration, ganaxolone is rapidly absorbed” and that the plasma concentration “sharply decreases . . . over the first 12 h and then by a terminal half-life of approximately 20 hours.” Ex. 1023 at 559.

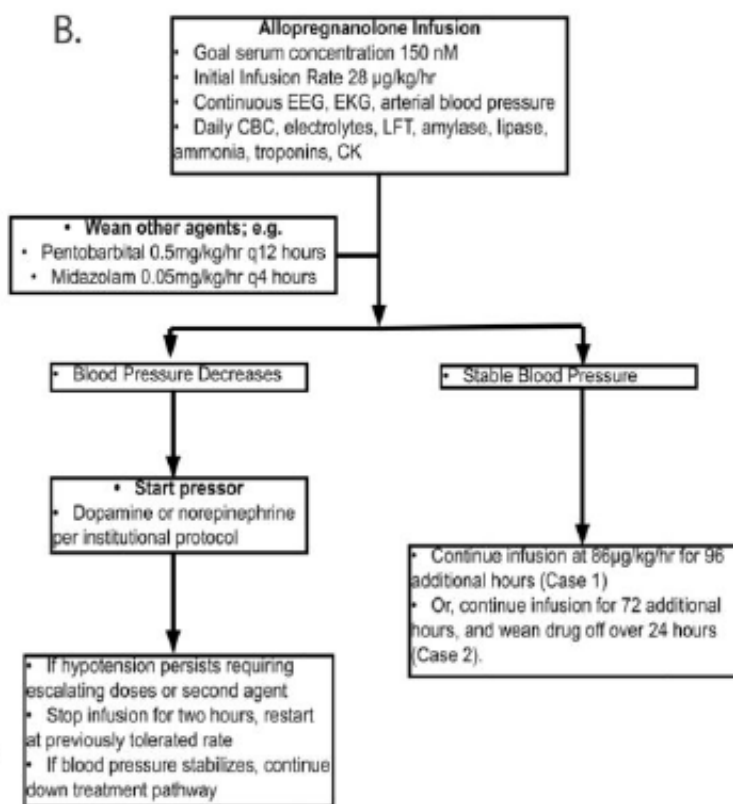
23. In 2016, Marinus began clinical trials for an IV formulation of ganaxolone for the treatment of SE. Ex. 1024 at 1. Marinus’s Phase 1 trial was designed to investigate the safety, pharmacokinetics, and pharmacodynamics (“PD”) of IV ganaxolone administered as both an ascending bolus dose (Stage 1 with dose cohorts A, B, C, and D) and as a bolus dose followed by a continuous infusion (Stage 2 with dose cohort A). Ex. 1024 at 1; Ex. 1025 at 1. In a poster describing the Phase 1 study that Marinus scientists presented at the 141st Annual Meeting of the American Neurological Association from October 16-18, 2016, the authors indicated

that the doses for cohorts A and B of the bolus dose were based on Marinus's "GNX focal onset seizure study," described above. Ex. 1025 at 1. Moreover, the poster indicates that "[a]n interim PK analysis was conducted after each cohort and this PK data was used to predict the PK to inform dose and infusion rate in subsequent cohorts, as well as dosing in Stage 2." Ex. 1025 at 1.

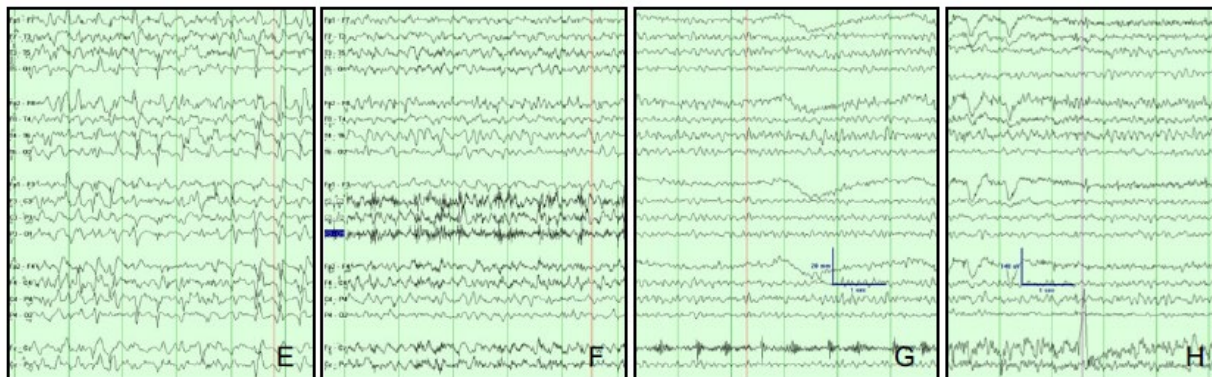
2. Allopregnanolone

24. Allopregnanolone is another neurosteroid and allosteric modulator of GABA_A receptors that has been tested in SE patients because of its antiseizure activity. Ex. 1026 at 1. Ganaxolone is the 3 β -methyl analog of allopregnanolone. Ex. 1027 at 220.

25. Broomall 2014 reports the use of continuous infusion of an IV solution of allopregnanolone to treat SE in two pediatric patients. Ex. 1026 at 1. In the first patient, an IV solution of allopregnanolone (0.5 mg/mL in 0.9% NaCl with 6% Captisol) was infused over 5 days according to the protocol in Fig. 1B (shown below). Ex. 1026 at 3. In the second patient, allopregnanolone was also infused according to the protocol in Fig. 1B but was tapered off between hours 96 and 120. Ex. 1026 at 3. The target infusion rate for both patients was 86 μ g/kg/h, which would result in a steady-state plasma level of 150 nM. This target steady-state level is equivalent to 48 ng/mL.



26. In another report, Vaitkevicius 2017, two adult SE patients were given allopregnanolone as a 120-hour continuous infusion. Ex. 1028 at 411. The first patient received a total of 670.8 mg of allopregnanolone as a continuous infusion of 5.6 mg/h over 5 days. Ex. 1028 at 412. Panels E-H in Figure 2 of Vaitkevicius 2017 (below) show EEG patterns demonstrating reduced seizure activity at 12, 24, 36, and 48 hours after discontinuation of phenobarbital while on allopregnanolone infusion. Ex. 1028 at 413.



27. A 2018 study in a mouse model of SE also sought to assess the potential of allopregnanolone and ganaxolone to treat SE when administered intramuscularly and found that both neurosteroids were effective in terminating behavioral seizures. Ex. 1027 at 221, 225. In the study, 3 mg/kg of both allopregnanolone and ganaxolone terminated SE in 92% and 75% of animals, respectively, and prevented mortality in 85% and 50% of animals. Ex. 1027 at 220. The plasma $C_{\max}$ values for allopregnanolone and ganaxolone at the same dose (3 mg/kg) were also found to be comparable at 645 ng/mL and 550 ng/mL, respectively. Ex. 1027 at 225 (Table 1).

V. The '100 Patent

A. The Specification

28. U.S. Patent No. 11,110,100 (“the '100 patent”) is titled “Ganaxolone for use in treatment of status epilepticus” and was filed on February 26, 2021. Ex. 1001. The '100 patent states that it claims priority to earlier filings, including International Patent Application No. PCT/US2020/044843, filed on Aug. 4, 2020,

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and U.S. Provisional Application No. 62/882/648, filed on Aug. 5, 2019. Ex. 1001 at 1:4-8.

29. The '100 patent discloses methods of treating SE with ganaxolone by administering an intravenous bolus and a continuous intravenous infusion. Ex. 1001 at 3:27-30. The specification states that “[s]uppression of SE typically breaks the seizures (i.e., suppresses or reduces seizure activity)” and that “[c]linically, suppression of SE can be reduction in seizure burden (i.e., the percent of time during which there is electrographic seizure activity).” Ex. 1001 at 16:7-13.

30. The '100 patent contains three examples. Example 1 reports the results of a Phase 1 clinical trial that investigated the safety, pharmacokinetics, and pharmacodynamics of an intravenous bolus of ganaxolone and continuous intravenous infusion of ganaxolone in healthy subjects. Ex. 1001 at 47:19-24.

31. Example 2 details an “ongoing, double-blind, randomized placebo-controlled Phase 2 study designed to evaluate the safety, tolerability, and efficacy of adjunctive IV GNX in subjects with SE who have failed initial treatment with benzodiazepines and at least one AED” and presents preliminary results from 5 subjects in the study. Ex. 1001 at 47:50-54:50. The '100 patent states that during the study, “subjects will receive a study drug bolus dose with a concurrent continuous infusion treatment with the goal of administering a therapeutic dose as quickly as

possible to stop the seizures and prevent permanent neurological damage.” Ex. 1001 at 47:62-67. “The primary objective of the study is to establish that IV GNX given concomitantly with second-line IV AED therapy is safe and effective in stopping SE that has already failed at least one second-line IV AED Therapy and prevents escalation of treatment requiring an IV anesthetic drug (a third-line treatment) for seizure suppression.” Ex. 1001 at 48:20-26.

32. Example 3 relates to “[a]n open-label, dose range finding study in which IV ganaxolone was added to standard-of-care IV AEDs in SE patients who had failed initial benzodiazepines and at least one 2nd line IV AED but had not progressed to IV anesthetics.” Ex. 1001 at 54:54-58. This study’s primary objective was to “establish that IV ganaxolone was safe and effective when added to standard of care IV AED therapy in stopping SE and preventing the escalation of treatment to IV anesthesia for seizure suppression.” Ex. 1001 at 54:58-62. The ’100 patent provides the following preliminary results from the study:

Preliminary results from this open-label, dose-finding Study 1042-SE-2001 in RSE patients showed that all patients (100%; n=17) did not require escalation of treatment to an IV anesthetic drug for SE control within the first 24-hours after initiation of GNX treatment, thereby meeting the primary endpoint. 16 patients (94%) achieved and maintained SE cessation within the first 24-hours following initiation of GNX treatment. 14 patients (82%) did not require additional IV AEDS or IV anesthetics for status relapse up to 4 weeks.

Ex. 1001 at 56:19-30.

B. The Claims

33. The '100 patent contains 21 claims. I have been advised that 1 of the 21 claims (claim 1) is an independent claim because it does not refer back to or depend from any other claim.

34. Independent claim 1 reads as follows:

1. A method of treating status epilepticus (SE), comprising administering to a subject in need thereof an effective amount of ganaxolone to suppress SE, wherein the effective amount of ganaxolone is administered as an intravenous bolus plus continuous infusion to produce and maintain a ganaxolone plasma concentration of about 500 ng/ml to about 1000 ng/ml for about 8 to about 12 hours.

Ex. 1001 at 57:9-15

35. I understand that the remaining 20 claims are dependent claims because they refer back to or depend from independent claim 1. I note that dependent claims 2, 3, 11 and 12 recite modifications to the administration of the intravenous bolus dose (dosage and duration). For example, I have provided claim 2 below:

2. The method of claim 1, wherein the intravenous bolus comprises about 5 mg to about 40 mg of ganaxolone.

Ex. 1001 at 57:16-17

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36. Dependent claims 4, 5, 7, and 9 recite modifications to the administration of the of the continuous intravenous infusion (dosage and duration).

For example, I have presented claim 4 below:

4. The method of claim 1, wherein the continuous intravenous infusion comprises infusion of about 20 mg of ganaxolone per hour to about 80 mg of ganaxolone per hour.

Ex. 1001 at 57:20-22

37. Dependent claims 6, 8, 10, 13, 15, 17, and 20 recite decreasing or tapering the amount of the continuous intravenous infusion. For example, I have presented claim 6 below:

6. The method of claim 5, wherein the amount of ganaxolone infused into the subject by the continuous intravenous infusion is decreased from about 80 mg to about 40 mg per hour to about 20 mg per hour.

Ex. 1001 at 57:27-30

38. Dependent claims 14, 18, 19, and 21 specify that the subject has RSE.
For illustration purposes, claim 14 is presented below:

14. The method of claim 1, wherein the subject has refractory status epilepticus.

Ex. 1001 at 58:15-16

39. Dependent claim 16 recites that “the total daily dose of ganaxolone administered is from about 500 mg to about 900 mg.” Ex. 1001 at 57:22-24.

VI. Legal Standards for Patentability

A. Obviousness

40. I understand that a claimed invention cannot be patented if the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art. In applying this standard, I understand that I must consider: (i) the level of ordinary skill in the art; (ii) the scope and content of the prior art; (iii) the differences between the claimed invention and the prior art; and (iv) any secondary considerations indicating the invention would not have been obvious.

41. I also understand that a claim can be obvious in light of either a single prior art reference or multiple prior art references. To be obvious, there must have been a motivation for a POSA to modify or combine the references to achieve the claimed invention.

42. I understand that the reason to combine references may come from a teaching, suggestion, or motivation, the references themselves, the knowledge of a POSA, or the nature of the problem to be solved; and it may be explicit or implicit. I also understand it is improper to rely on hindsight in making the determination of obviousness.

43. I also understand that obviousness does not require a certainty of success or absolute predictability of success, but only a reasonable expectation of

success. Likewise, I understand that obviousness cannot be avoided by showing some degree of unpredictability so long as there was a reasonable probability of success.

B. Priority

44. I understand that a subject patent can assert priority to a patent application filed earlier than the subject patent.

45. However, I understand that such an assertion does not necessarily mean the subject patent is entitled to the date of the earlier-filed patent application.

46. In this regard, I understand that the subject patent is only entitled to the date of the earlier-filed patent application if the earlier-filed patent application sufficiently discloses the invention claimed in the subject patent in a manner compliant with 35 U.S.C. § 112(a).

47. I understand that § 112(a) requires that the earlier-filed patent application (i) reasonably conveys to one of ordinary skill in the art that the inventors were in possession of the claimed subject matter (i.e., the “written description support” requirement) and (ii) enables a POSA to practice the claimed invention without undue experimentation (i.e., the “enablement” requirement).

48. Lastly, I understand that a broad disclosure of a given concept in an earlier-filed patent application often does not provide written description support for a narrower invention relating to that given concept.

VII. Person of Ordinary Skill in the Art

49. I have been instructed that a person of ordinary skill in the art (“POSA”) is a legal fiction that is used in many aspects of patent law. I have been instructed that a POSA is a hypothetical person at the time of the invention who is presumed to be aware of all pertinent prior art, thinks along conventional wisdom in the art, and is a person of ordinary creativity.

50. Based on my years of experience in the relevant field, it is my opinion that as of August 5, 2019, a POSA would have possessed a high level of skill, such as having an advanced degree, such as a medical degree or a Ph.D. in the field of pharmacology or other fields related to the development of pharmaceuticals, and have several years of experience as a practicing neurologist treating patients who suffer from seizure disorders including SE and/or several years of experience in researching treatments for such seizure disorders. A POSA would be familiar with the pharmacokinetics (“PK”) and pharmacodynamics (“PD”) of pharmaceuticals. A POSA would easily have understood the prior art references I discuss in this Declaration and would have had the capability to draw inferences from them.

51. As an expert in the relevant field since before 2019, I am qualified to provide an opinion as to what a POSA would have understood, known, or concluded as of 2019. I have reviewed the materials, conducted my analyses, and formed my opinions in my Declaration through the eyes of the POSA as of August 5, 2019. My definition of a POSA would not change if the relevant date were August 4, 2020.

VIII. Claim Construction

52. I understand that I should read claims 1-21 of the '100 patent in accordance with the ordinary and customary meaning that their terms would have been understood by a POSA unless that ordinary and customary meaning is changed by the '100 patent's specification or prosecution history.

53. I have construed the terms in claims 1-21 of the '100 patent as having their ordinary and customary meaning.

A. “to produce and maintain a ganaxolone plasma concentration of about 500 ng/ml to about 1000 ng/ml”

54. I understand that certain claim terms may be considered non-limiting if the claim terms merely express a purpose and only state an intended result of the claimed method. That is, claim language without any bearing on the claimed methods should be deemed non-limiting when the language does not result in a manipulative difference in the steps of the claim.

55. In my opinion, a POSA would have understood that the claim term “to produce and maintain a ganaxolone plasma concentration of about 500 ng/ml to about 1000 ng/ml” in independent claim 1 is non-limiting.

56. The language of claim 1 requires administering “an effective amount of ganaxolone to suppress SE” and further specifies that the effective amount should be “administered as an intravenous bolus plus continuous infusion.” Thus, the claim language is clear: the dosing amount must be an “effective amount of ganaxolone to suppress SE.” To determine what amount of ganaxolone is effective to suppress SE, a POSA would have administered ganaxolone as a bolus followed by continuous infusion and then monitored the patient’s electroencephalography (“EEG”) activity to confirm the suppression of SE. A POSA would have understood that the claimed plasma concentration (i.e., “about 500 ng/ml to about 1000 ng/ml”) does not further limit “an effective amount of ganaxolone to suppress SE.” Therefore, the recited plasma concentration adds nothing to these steps and is superfluous.

57. Additionally, a POSA would not have looked to blood-plasma concentration levels of ganaxolone in practice when treating a patient with SE. This is because it could take many hours (or even days) to receive the results of testing required to determine ganaxolone plasma concentration in the patient’s blood.

58. The '100 patent's specification also links "effective amount" to SE suppression. In particular, the specification states that:

During the treatment, the subject can be monitored for plasma concentration of neurosteroid (e.g. ganaxolone) and *using EEG for seizure activity*. If the subject appears to show signs of SE re-lapse, *the amount of ganaxolone administered can be adjusted accordingly*. For instance, an additional intravenous bolus can be provided or the amount of ganaxolone can be increased. The amount of neurosteroid (e.g. ganaxolone administered during infusion is typically adjusted by increasing or decreasing the infusion rate.

Ex. 1001 at 17:6-11, 23:64-24:1 ("Electroencephalogram (EEG) can be used to detect signs of SE re-lapse. Plasma concentration of ganaxolone can alternatively or in combination with EEG be used to detect signs of SE re-lapse or a subject that experiences SE re-lapse."). Thus, the '100 patent's specification confirms that determining whether an effective amount of ganaxolone has been administered to suppress SE is based on monitoring EEG for seizure activity and then adjusting the dose if needed.

59. Thus, based on the above, a POSA would have understood that the ganaxolone plasma concentration recited in independent claim 1 is non-limiting.

IX. Claims 1-21 of the '100 Patent Are Not Entitled to the Priority Date of the '648 Provisional Application

60. The '100 patent claims priority to PCT Application No. PCT/US2020/044843, filed on August 4, 2020, which claims priority to U.S.

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Provisional Application No. 62/882,648 (“the ’648 application”), filed on August 5, 2019.

61. To the extent the ganaxolone plasma concentration recited in independent claim 1 is construed to be limiting or not a natural result of an obvious method, it is my opinion that independent claim 1 and dependent claims 2-21 of the ’100 patent should not be afforded their claimed priority date of August 5, 2019, because the ’648 application does not provide adequate written description to support the limitation requiring “to produce and maintain a ganaxolone plasma concentration of about 500 ng/ml to about 1000 ng/ml for about 8 hours to about 12 hours.”

62. No data in the provisional application would have demonstrated to a POSA that ganaxolone plasma concentration was maintained at “about 500 ng/ml to about 1000 ng/ml for about 8 hours to about 12 hours” in any patient.

63. Example 2 of the ’648 application provides the protocol for a Phase 2 clinical trial and pharmacokinetic modeling data from 5 subjects, which “suggested that exposure levels of GNX at approximately 1000 ng/mL were associated with rapid and complete SE suppression, and lower GNX exposures at <500 ng/mL were associated with seizure relapse.” Ex. 1031 at ¶¶ [0188]-[0203]. However, none of the experiments discussed in Example 2 maintained a ganaxolone plasma

concentration of about 500 ng/ml to about 1000 ng/ml for about 8 to about 12 hours. In fact, in four of the five subjects, seizure activity relapsed about 5-6 hours after the infusion started and once plasma concentrations of ganaxolone decreased < 500 ng/mL. Ex. 1031 at ¶ [0189]. And, in the fifth subject, ganaxolone plasma concentration had decreased to < 500 ng/mL by the fourth hour after the infusion started. ('648 application, Fig. 10.) These experiments therefore provide no support for persistent suppression of SE, which was “unexpectedly” achieved with the claimed plasma concentration and time period.

64. The provisional application also includes trial results from a single patient who was subjected to a modified protocol that would allow the total daily dose of ganaxolone to increase from 500 mg to 715 mg and would purportedly allow for ganaxolone exposure to be maintained at >500 ng/ml for approximately 8 hours. Ex. 1031 at ¶¶ [0204], [0208]. This single patient “experienced rapid RSE cessation following GNX treatment . . . and was successfully weaned off GNX without seizure relapse.” Ex. 1031 at ¶ [0204]. The provisional application expressly admits, however, that “pharmacokinetic data were *unavailable at the time of this report.*” Ex. 1031 at ¶ [0204], (emphasis added).

65. Notably, the provisional application is missing Example 3 from the '100 patent, which is related to a Phase 2 Open-Label Dose Range Study. *See* Ex.

1001 at 54:52-57:7. Only in Example 3 of the '100 patent do the inventors state that “[t]he major difference between the low [500 mg/day] and high [713 mg/day] dosing cohorts was the duration of predicted plasma GNX levels >500 ng/ml (4 vs. 8-hours, respectively).” Ex. 1001 at 55:56-58.

66. Thus, it is my opinion that no data in the provisional application would have demonstrated to a POSA that the ganaxolone plasma concentration was maintained at “about 500 ng/ml to about 1000 ng/ml for about 8 hours to about 12 hours” in any patient. Therefore, because claims 1-21 of the '100 patent do not have adequate written description support in the '648 application, I understand that the claims are not entitled to the August 5, 2019 priority date, and the earliest priority date they may be afforded is the priority date of International Patent App. No. PCT/US2020/044843, filed on August 4, 2020.

X. The Pertinent Prior Art

A. During (Ex. 1003)

67. During is U.S. Patent No. 11,395,817, which issued on July 26, 2022, and claims priority to a provisional application filed on August 11, 2016. I understand that During is prior art to the '100 patent regardless of whether the '100 patent is afforded its August 5, 2019 priority date.

68. During describes “methods of using allosteric modulators and/or gaboxadol or pharmaceutically acceptable salts thereof for the treatment of epileptic

disorders in a subject in need thereof.” Ex. 1003 at Abstract. Allosteric modulators include the neurosteroid ganaxolone. Ex. 1003 at 6:22-23.

69. During discloses that “a method of treating an epileptic disorder such as status epilepticus includes administering ganaxolone or a pharmaceutically acceptable salt thereof to a patient in need thereof.” Ex. 1003 at 8:11-14. During discloses that ganaxolone can be administered in doses by weight of the patient ranging from:

10 mg/kg to 40 mg/kg, e.g., 11 mg/kg to 39 mg/kg, 12 mg/kg to 38 mg/kg, 13 mg/kg to 37 mg/kg, 14 mg/kg to 36 mg/kg, 15 mg/kg to 35 mg/kg, 16 mg/kg to 34 mg/kg, 17 mg/kg to 33 mg/kg, 18 mg/kg to 32 mg/kg, 19 mg/kg to 31 mg/kg, 20 mg/kg to 30 mg/kg, 21 mg/kg to 29 mg/kg, 22 mg/kg to 28 mg/kg, 23 mg/kg to 27 mg/kg, or 24 mg/kg to 26 mg/kg.

Ex. 1003 at 8:14-22.

70. During also provides additional doses of ganaxolone as:

50 mg, 75 mg, 100 mg, 125 mg, 150 mg, 175 mg, 200 mg, 225 mg, 250 mg, 275 mg, 300 mg, 325 mg, 350 mg, 375 mg, 400 mg, 425 mg, 450 mg, 475 mg, 500 mg, 525 mg, 550 mg, 575 mg, 600 mg, 625 mg, 650 mg, 675 mg, 700 mg, 725 mg, 750 mg, 775 mg, 800 mg, 825 mg, 850 mg, 875 mg, 900 mg, 925 mg, 950 mg, 975 mg, 1000 mg, 1225 mg, 1250 mg, 1275 mg, 1300 mg, 1325 mg, 1350 mg, 1375 mg, 1400 mg, 1425 mg, 1450 mg, 1475 mg, 1500 mg, 1525 mg, 1550 mg, 1575 mg, 1600 mg, 1625 mg, 1650 mg, 1675 mg, 1700 mg, 1725 mg, 1750 mg, 1775 mg, 1800 mg, 1825 mg, 1850 mg, 1875 mg, 1900 mg, 1925 mg, 1950 mg, 1975 mg, or 2000 mg.

Ex. 1003 at 8:22-34.

71. According to During, ganaxolone “can be administered, e.g., once daily, twice daily, three times daily, or four times daily” and can be administered “as soon as possible after the onset of seizures.” Ex. 1003 at 8:39-44.

72. During also more broadly discloses that “an allosteric modulator or a pharmaceutically acceptable salt thereof is administered at dosages ranging from about 0.001 mg/kg to about 30 mg/kg of body weight of a patient in need thereof.” Ex. 1003 at 6:47-50. With respect to these allosteric modulators, During discloses specific exemplary daily dose ranges of:

500 mg to 525 mg/day, 525 mg to 550 mg/day, 550 mg to 575 mg/day, 575 mg to 600 mg/day, 600 mg to 625 mg/day, 625 mg to 650 mg/day, 650 mg to 675 mg/day, 675 mg to 700 mg/day, 700 mg to 725 mg/day, 725 mg to 750 mg/day, 750 mg to 775 mg/day, 775 mg to 800 mg/day, 800 mg to 825 mg/day, 825 mg to 850 mg/day, 850 mg to 875 mg/day, 875 mg to 900 mg/day, 900 mg to 925 mg/day, 925 mg to 950 mg/day, 950 mg to 975 mg/day, 975 mg to 1000 mg/day.

Ex. 1003 at 7:49-57.

73. Example 3 of During provides a detailed description of the “[e]valuation of the ability of allopregnanolone, ganaxolone, and gaboxadol to block benzodiazepine-resistant status epilepticus.” Ex. 1003 at 35:33-37:15. In the study, allopregnanolone, ganaxolone, and gaboxadol were administered to rats in doses of 0.5 mg/kg, 2 mg/kg, 5 mg/kg, 10 mg/kg, or 20 mg/kg. Ex. 1003 at 36:22-24. As

shown by Table 2 and Figures 3 and 4, administration of 20 mg/kg ganaxolone was effective to induce significant protection against SE, characterized by no observation of further convulsive seizures after the drug had been administered for 10 min. At a lower dose (10 mg/kg), ganaxolone also afforded protection against SE despite not reaching a statistically significant threshold. Both 10 mg/kg and 20 mg/kg of ganaxolone treatment also promoted 24-hour survival of the experimental animals, although the effects were not statistically significant.

74. The claims of During also recite methods of treating SE by administering intravenous ganaxolone at doses of “about 150 mg to 1000 mg/day,” “about 500 mg to 1000 mg/day,” “about 625 mg to 650 mg/day,” “about 650 mg to 675 mg/day,” “about 675 mg to 700 mg/day,” “about 625 mg to 700 mg/day,” “about 700 mg to 725 mg/day,” “about 1025 mg to 1050 mg/day.” Ex. 1003 at 40:18-33.

B. Clinical Trial NCT03350035 (Ex. 1004)

75. Clinical Trial NCT03350035 was first published on ClinicalTrials.gov on November 17, 2017. A POSA would have considered ClinicalTrials.gov to be a reliable and trustworthy source for information related to scheduled, ongoing, and completed clinical trials. A POSA would have considered the posting dates cited on clinicaltrials.gov to be trustworthy and authoritative. Like most other POSAs, I

regularly consult ClinicalTrials.gov for the details of clinical trials relevant to my practice and have done so for years. *See, e.g.*, Ex. 1030 at 5.

76. A POSA also would have understood that ClinicalTrials.gov allows the user to view historical versions of clinical trial protocols. The version of Clinical Trial NCT03350035 I have considered was first submitted on November 17, 2017, and first posted on November 22, 2017. Ex. 1004 at 2. I understand that Clinical Trial NCT03350035 is prior art to the '100 patent regardless of whether the '100 patent is afforded its August 5, 2019 priority date.

77. Clinical Trial NCT03350035 describes “a double-blind, randomized, placebo-controlled study to evaluate the safety, tolerability, and efficacy of adjunctive IV ganaxolone in subjects with SE.” Ex. 1004 at 3. In particular, Clinical Trial NCT03350035 states that ganaxolone was administered as an “IV loading dose with continuous infusion (maintenance dose) for 2-4 days followed by a 24-hour taper.” Ex. 1004 at 3-4.

C. Marinus’s Phase 1 Clinical Trial Results

1. Tsai 2017 (Ex. 1005)

78. Tsai 2017 is abstract P72 in the Final Programme of the 6th London-Innsbruck Colloquium on Status Epilepticus and Acute Seizures and is titled “Phase I study to determine the pharmacokinetics, pharmacodynamics, and safety of IV ganaxolone in healthy adults.” Ex. 1005 at 74-75. Tsai 2017 was published in April

2017. I understand that Tsai 2017 qualifies as prior art to the '100 patent regardless of whether the '100 patent is afforded its August 5, 2019 priority date.

79. Tsai 2017 describes the results of a Phase 1 study that evaluated the “safety, pharmacokinetics (PK), and pharmacodynamics (PD) of [ganaxolone (GNX)] IV administered as a bolus dose and continuous infusion to healthy patients.” Ex. 1005 at 74. Tsai 2017 describes the protocol followed in this Phase 1 study as assessing “PK changes in EEG, BIS, and sedation scores, and safety . . . following a single bolus and bolus dosing followed by continuous infusion of GNX IV.” Ex. 1005 at 74.

80. Tsai 2017 reports the pK results of the Phase 1 study as showing that “GNX concentrations in plasma were generally proportional to the administered dose. Potential anti-convulsant plasma concentrations were achievable with a bolus dose. After the IV bolus dose or cessation of the IV infusion dose, concentrations of GNX in plasma declined in a multi-phasic manner.” Ex. 1005 at 75. Tsai 2017 concludes that “GNX administered as a rapid IV bolus or bolus with continuous infusion was generally safe and well tolerated,” and that “there were no safety issues that limit getting to a potentially efficacious plasma concentration in a short period of time” through the bolus dose, and that “[p]redictable PK from this study enables dosing guidance for GNX IV.” Ex. 1005 at 75.

2. Tsai 2016 (Ex. 1006)

81. Tsai 2016 is abstract S208 in Volume 80, Supplement 20 of the 2016 Annals of Neurology Journal and is titled “Phase I Study to Determine the Pharmacokinetics, Pharmacodynamics, and Safety of IV Ganaxolone in Healthy Adults.” Ex. 1006 at S77-78. Tsai 2016 was published in 2016. I understand that Tsai 2016 is prior art to the ’100 patent regardless of whether the ’100 patent is afforded its August 5, 2019 priority date.

82. Tsai 2016 describes the results of a Phase I study, whose objective was “to evaluate the safety, pharmacokinetics (PK), and pharmacodynamics (PD) of ganaxolone (GNX) IV administered as a bolus dose and continuous infusion to healthy participants.” Ex. 1006 at S77-S78. Tsai 2016 describes the method of the study as proceeding in two stages, where Stage 1 “will determine the PK, change in quantitative and qualitative EEG (PD), and safety . . . following single bolus doses of IV GNX” and Stage 2 “will determine the PK, safety, and PD of bolus IV dosing followed by continuous infusion.” Ex. 1006 at S78.

3. Clinical Leader 2016 (Ex. 1007)

83. Clinical Leader 2016 is a news article titled “Ganaxolone Phase 1 Data Supports Progressing to Phase 2 in Patients with Status Epilepticus.” Ex. 1007 at 1. The Clinical Leader 2016 Press Release was published on October 19, 2016. I

understand that Clinical Leader 2016 is prior art to the '100 patent regardless of whether the '100 patent is afforded its August 5, 2019 priority date.

84. Clinical Leader 2016 states that the Phase I study “was designed to determine the pharmacokinetics (PK), pharmacodynamics (PD), and safety of ganaxolone IV administered as an ascending bolus dose (Stage 1) or continuous infusion (Stage 2).” Ex. 1007 at 1. The press release also states that Marinus had “announced that in its Phase 1 dose-escalation study, ganaxolone intravenous (IV) achieved dose levels targeted for efficacy in patients with status epilepticus (SE) and other indications.” Ex. 1007 at 1. Marinus’s chief medical officer, Albena Patroneva, is quoted in the press release as stating that Marinus is “pleased with the results from our Phase 1 study which has provided predictable PK to enable dosing of ganaxolone IV in clinical studies in SE.” Ex. 1007 at 1. The press release describes the results of the study as demonstrating that “[e]very dose regimen of ganaxolone IV administered, either bolus or continuous infusion, was generally safe and well tolerated and reached targeted dose levels in a short period of time.” Ex. 1007 at 1. Moreover, “[g]anaxolone IV plasma concentrations were generally proportional to the administered dose, with potential anti-convulsant plasma concentrations achievable with a bolus dose. Additionally, the continuous infusion of ganaxolone IV achieved the targeted PK levels.” Ex. 1007 at 1. Clinical Leader 2016 also notes

that the data from the Phase I study “was presented by Dr. Julia Tsai, senior director of clinical development and project management at Marinus, in a poster entitled, ‘Phase 1 study to determine the pharmacokinetics, pharmacodynamics, and safety of IV ganaxolone in healthy adults,’ at the 141st Annual Meeting of the American Neurological Association (ANA), October 16-18, 2016.” Ex. 1007 at 1.

D. Phase 2 Clinical Trial Results

1. Practical Neurology 2019 (Ex. 1010)

85. Practical Neurology 2019 is an article titled “Treatment of Refractory Status Epilepticus Prevents Need for Induced Coma with Intravenous Anesthetics in Phase 2 Trial.” Ex. 1010 at 1. Practical Neurology 2019 was published on October 18, 2019. I understand that Practical Neurology 2019 is prior art to the ’100 patent should the ’100 patent not be afforded its August 5, 2019 priority date.

86. Practical Neurology 2019 describes the results of a Phase 2 clinical trial. The article describes the results of the study as follows:

In a phase 2 clinical trial, treatment with ganaxolone prevented all 17 participants from needing IV anesthetics for induced coma within 24 hours of treatment initiation. . . . A target dose of ganaxolone, supported by pharmacokinetic (PK) data, has been identified for advancement to phase 3 clinical trials.

The PK data show that the target dose of ganaxolone (713 mg/day) achieved plasma drug concentrations greater than 500 ng/mL for approximately 8 hours, the longest and highest duration of the 3 dosing groups. A dose-response

relationship among the dose groups was apparent, with the target dose demonstrating the best and most durable efficacy for the period beginning with the commencement of the ganaxolone infusion through the 24-hour period that followed discontinuation of ganaxolone therapy.

Ex. 1010 at 1.

87. The results of the Phase 2 study “were presented at the [N]eurocritical Care Society 17th Annual Meeting in Vancouver, BC.” Ex. 1010 at 1.

2. Ramsay 2019 (Ex. 1008)

88. Ramsay 2019 is an abstract titled “Pharmacokinetic and Pharmacodynamic (PK/PD) Relationship of Intravenous Ganaxolone in Refractory Status Epilepticus. Ex. 1008 at 1. Ramsay 2019 was published on November 25, 2019. I understand that Ramsay 2019 is prior art to the ’100 patent should the ’100 patent not be afforded its August 5, 2019 priority date.

89. Ramsay 2019 describes “PK/PD evidence of a rapid, durable, and drug exposure-dependent clinical response to IV GNX administration in patients with refractory status epilepticus (RSE) in a Phase 2 open-label study.” Ex. 1008 at 1-2. During the study, “two dose levels of IV GNX were assessed in subjects with RSE.” Ex. 1008 at 2. Cohort A and Cohort B “were dosed with 500 mg/day and 714 mg/day of continuous IV GNX, respectively, for up to four days.” Ex. 1008 at 2. The infusion was designed to “produce initial high plasma levels of GNX for several hours followed by stepped decreases of GNX dosing.” Ex. 1008 at 2. A POSA would have

recognized that such an intravenous infusion would be achieved through administering a bolus dose, which would “produce initial high plasma levels,” followed by continuous infusion, which would allow for “stepped decreases.”

90. The results of the study demonstrated that “RSE was stopped rapidly (within minutes) upon the start of IV GNX in all subjects who failed 1-4 IV ASMs.” Ex. 1008 at 2. Ramsay 2019 associates this response with “plasma GNX levels >500 ng/mL.” Ex. 1008 at 2.

91. In Cohort A, ganaxolone plasma concentrations were “maintained ~500 ng/mL for approximately 4 hours with good seizure suppression.” Ex. 1008 at 2. “Seizure relapse or abnormal EEG activity was noted at ~5-6 hours into the infusion in four out of 5 subjects which corresponded to when GNX plasma levels decreased to < 500 ng/mL.” Ex. 1008 at 2. In Cohort-B, ganaxolone plasma levels were “able to be maintained ~500 ng/mL for ~8 hrs. Good seizure control was maintained in all subjects throughout the entire dosing period even when GNX plasma levels decreased < 500 ng/mL and no subjects progressed to IV anesthetics.” Ex. 1008 at 2.

92. Ramsay 2019 concluded that “the durability of pharmacological response appears to correlate with GNX exposure and ability to maintain target plasma drug levels for an adequate amount of time.” Ex. 1008 at 2.

3. AES 2019 Poster (Ex. 1011)

93. Marinus's website includes a poster under the heading "AES 2019" that has the same name as the Ramsay 2019 abstract, i.e., "Pharmacokinetic and Pharmacodynamics (PK/PD) Relationship of Intravenous Ganaxolone in Refractory Status Epilepticus." Ex. 1011 at 1. This is the same poster that was presented with the Ramsay 2019 abstract on December 7, 2019. Ex. 1008 at 1 (noting that the data underlying Ramsay 2019 was presented on December 7, 2019, at 6 pm). I understand that the AES 2019 Poster is prior art to the '100 patent should the '100 patent not be afforded its August 5, 2019 priority date.

94. The AES 2019 Poster presents the results of the phase 2 study described in Ramsay 2019. The AES 2019 Poster discloses results from three dose groups and concludes that "Target Dose [713 mg/day] Achieves 500 ng/mL for ~8 hours." Ex. 1011 at 1.

4. Husain 2020 (Ex. 1009)

95. Husain 2020 is abstract F53 titled "Intravenous Ganaxolone Achieves Rapid and Dose-Dependent Sustained Improvement in EEG Seizure Burden in Patients with Refractory Status Epilepticus." Ex. 1009 at 332. Husain 2020 is published in Volume 37, Number 4 of the Journal of Clinical Neurophysiology and was submitted for the 2020 American Clinical Neurophysiology Society (ACNS) Annual Meeting. Ex. 1009 at 332. Husain 2020 published on July 1, 2020. I

understand that the Husain 2020 is prior art to the '100 patent should the '100 patent not be afforded its August 5, 2019 priority date.

96. Husain 2020 describes a phase 2, open-label, dose-escalation study where IV ganaxolone was used to treat RSE patients. Ex. 1009 at 332. Husain 2020 discloses that “evaluable patients were allocated to three dose cohorts: 500 mg/day GNX (Low, n=4), 650 mg/day GNX (Medium, n=4), or 713 mg/day GNX (Target, n=7).” Ex. 1009 at 332.

97. Husain 2020 reports that “[i]n all dose groups, a rapid reduction in seizure burden of ~45% was seen within 0-5 minutes of GNX administration and further enhanced to at least an 80% sustained reduction within 10-15 minutes post-GNX initiation.” Ex. 1009 at 332. Husain 2020 also notes that “[o]nly evaluable patients in the Target dose achieved at least an 80% sustained reduction in seizure burden for the entire analysis window (24 hrs) whereas seizure activity partially returned in the Low and Medium doses at times aligning with decreased GNX exposure.” Ex. 1009 at 332.

XI. Claims 1-21 of the '100 Patent Are Unpatentable as Obvious

A. All Claims of the '100 Patent Are Unpatentable Even If They Are Entitled to the Priority Date of the '648 Application

1. Ground 1: All Claims of the '100 Patent Would Have Been Obvious Over During and NCT03350035.

98. As part of my obviousness analysis, I considered whether a POSA would have been motivated to combine the disclosures of During and NCT03350035 as of August 5, 2019. For the reasons I describe below, I conclude that a POSA would have had several reasons to combine the disclosures of During and NCT03350035 to arrive at the claimed invention with a reasonable expectation of success.

a. Claim 1

i. A POSA Would Have Been Motivated to Combine During and NCT03350035

99. Both During and NCT03350035 relate to treating epileptic disorders, including SE. Ex. 1003, 8:11-14, 8:22-34, 35:31-37:15; Ex. 1004, 3-4.

100. During teaches administering ganaxolone to treat SE. Ex. 1003 at, 8:11-14, 8:22-34, 35:31-37:15. In particular, Example 3 of During describes an evaluation of ganaxolone's ability to block benzodiazepine-resistant SE and concludes that there was significant improvement in seizure protection at 20 mg/kg ganaxolone. Ex. 1003 at 35:33-35. During's claims also recite methods of treating SE by

administering the following doses of ganaxolone: “about 150 mg to 1000 mg/day,” “about 500 mg to 1000 mg/day,” “about 625 mg to 650 mg/day,” “about 650 mg to 675 mg/day,” “about 675 mg to 700 mg/day,” “about 625 mg to 700 mg/day,” “about 700 mg to 725 mg/day,” “about 1025 mg to 1050 mg/day.” Ex. 1003 at 40:18-33.

101. Further, the prior art demonstrated that 1,500 mg/day ganaxolone (after titrating up from 600 mg/day) administered orally was effective at reducing partial-onset seizure frequency. *See, e.g.*, Ex. 1023, 561, 562. The prior art directly taught that doses of ganaxolone in partial-onset seizure studies inform those for doses of ganaxolone for treating SE. Ex. 1025, 1 (Table 1 stating “Cohort B” dose is “Therapeutic dose based on GNX (FOS [focal/partial onset seizure]); over 2 min”). A POSA would have been motivated to target even lower doses based on the ganaxolone’s oral administration because a POSA would have known that ganaxolone administered intravenously would have higher bioavailability than ganaxolone administered orally. Ex. 1034, 222. A POSA thus would have been motivated to select a lower dose of ganaxolone within the range of 600 mg/day to 1,500 mg/day to suppress SE, e.g., During’s disclosed doses of 625-725 mg/day or 1025-1050 mg/day ganaxolone.

102. NCT03350035 is a study protocol to evaluate the safety, tolerability, and efficacy of adjunctive IV ganaxolone in subjects with SE. In Clinical Trial

NCT03350035, ganaxolone was administered as an “IV loading dose with continuous infusion (maintenance dose) for 2-4 days followed by a 24-hour taper.” Ex. 1004 at 3-4. A POSA would have understood that a “loading dose” is the same as a “bolus dose.”

103. Thus, During specifically addresses the use of ganaxolone to treat SE, including specific dose ranges, and NCT03350035 also specifically addresses the use of ganaxolone to treat SE as an IV loading dose (or bolus) with continuous infusion.

104. Based on During’s disclosures (directed to ganaxolone’s evaluation of the ability of ganaxolone to block benzodiazepine-resistant SE and disclosure of dose ranges) and the disclosures of NCT03350035 (directed to the administration of ganaxolone as an IV bolus dose with continuous infusion in subjects with SE), a POSA would have been motivated to use ganaxolone to suppress SE by administering ganaxolone as an intravenous bolus dose followed by continuous infusion.

ii. A POSA Would Have Had a Reasonable Expectation of Success in Combining During and NCT03350035

105. I considered whether claim 1 would have been obvious over During and NCT03350035. Based on the combination of During and NCT03350035, a POSA

would have expected that ganaxolone administered as an IV bolus dose with continuous infusion would suppress SE and produce and maintain a ganaxolone plasma concentration of about 500 ng/ml to about 1000 ng/ml for about 8 hours to about 12 hours.

106. As explained above, During describes administering ganaxolone to treat and suppress SE, including by administering the following doses of ganaxolone: “about 150 mg to 1000 mg/day,” “about 500 mg to 1000 mg/day,” “about 625 mg to 650 mg/day,” “about 650 mg to 675 mg/day,” “about 675 mg to 700 mg/day,” “about 625 mg to 700 mg/day,” “about 700 mg to 725 mg/day,” “about 1025 mg to 1050 mg/day.” Ex. 1003 at 8:11-14; 40:18-33. NCT03350035 specifically addresses the use of ganaxolone as an IV loading dose (i.e., bolus) with continuous infusion in humans with SE. Ex. 1004 at 3-4.

107. The prior art thus expressly describes a method of treating SE by administering ganaxolone as an intravenous bolus followed by continuous infusion. Ex. 1003, 8:11-14, 35:33-37:15, 40:18-33; Ex. 1004, 3-4. During further expressly describes “an effective amount of ganaxolone to suppress SE.” For instance, During discloses that doses of ganaxolone to treat SE can range from 50 mg to 2000 mg (Ex. 1003, 8:22-34) and can be “administered parenterally in escalating doses as soon as possible after onset of seizures” (*id.*, 8:42-44).

108. A POSA also would have understood that the doses in During encompass doses that are effective for suppressing SE based on earlier studies for treating SE and other seizure disorders using ganaxolone or its analog, allopregnanolone. For example, as described in Section IV.B.1, Marinus reported the results of a phase 2 study of ganaxolone as an add-on therapy in adults with partial-onset seizures, which demonstrated that 1,500 mg/day ganaxolone (after titrating up from 600 mg/day) administered orally was effective at reducing partial-onset seizure frequency. Ex. 1023 at 558, 561, 562. And the prior art directly taught that ganaxolone doses in partial-onset seizure studies inform those for SE. Ex. 1025, 1. A POSA therefore would have reasonably expected that a dose of 1,500 mg/day would be effective to suppress SE.

109. Dosing of allopregnanolone further would have given a POSA a reasonable expectation that During's dose range of 50 mg to 2000 mg encompasses doses that are effective to suppress SE. As explained in Section IV.B.2, ganaxolone is an analog of allopregnanolone and both are allosteric modulators of the GABA_A receptor. Ganaxolone was also "designed to have similar GABA modulation effects as allopregnanolone without unwanted steroidal side effects." Ex. 1033 at 1.

110. Allopregnanolone doses had also previously been correlated to ganaxolone doses in the SE mouse model. *See, e.g.*, Ex. 1027, 223, 225. In 2018, a

study into the mouse model of SE by Zolkowska confirmed that allopregnanolone and ganaxolone administered at the same dose terminated SE and found that the same dose of each drug achieved “comparable plasma levels . . . (C_{max} , 645 ng/mL for allopregnanolone and 550 ng/mL for ganaxolone).” Ex. 1027 at 223. And in 2017 study found that 670.8 mg allopregnanolone administered as a 5.6 mg/h continuous infusion over 5 days “successfully treated” two patients with super-refractory SE (“SRSE”). Ex. 1028 at 411-12.

111. This information related to allopregnanolone (i.e., that it has comparable plasma concentration levels in treating SE as ganaxolone and successfully treated patients with SE using 670.8 mg of intravenous allopregnanolone) also would have provided a POSA with a starting point to determine an amount of ganaxolone effective to suppress SE and a reasonable expectation of doing so. Ex. 1027; Ex. 1028.

112. A POSA therefore would have reasonably expected that doses falling within During’s express range of 50 mg to 2000 mg would have suppressed SE. Ex. 1003, 8:22-34. Any optimization required to be performed within this range would have been routine to a POSA.

113. As discussed above in Section VIII, the ganaxolone plasma concentration of “about 500 ng/ml to about 1000 ng/ml” should be construed as non-

limiting. To the extent that the ganaxolone plasma concentration is limiting, this is simply the expected and natural result from administering an effective amount of ganaxolone to suppress SE, which, as discussed, would have been obvious.

114. The dosing regimen described in NCT03350035 (i.e., a bolus followed by continuous infusion) is not a new dosing regimen in the field of treating SE. As I explained in Section IV.A, patients who did not respond to first or second-line ASMs would have been administered certain drugs intravenously using a bolus dose, followed by continuous intravenous infusion and tapering. As this administration is occurring, a POSA would have monitored the patient's EEG readout for seizure activity and adjusted the dose of the drug to control seizures. This was a common approach used to treat SE with drugs such as propofol, thiopental, and midazolam. A POSA would have thus been motivated to apply NCT03350035's dosing regimen to the doses in During with a reasonable expectation of successfully suppressing SE. By monitoring the patient's EEG readout, a POSA would have been able to optimize the doses of ganaxolone administered via intravenous bolus and continuous intravenous infusion to successfully suppress SE.

115. To the extent considered a limitation, a POSA would have known that the ganaxolone plasma concentration levels recited in claim 1 necessarily flow from the dosing method disclosed by NCT03350035 and at the dosage ranges disclosed

in the prior art, including in During. In other words, administering ganaxolone as an intravenous bolus plus continuous infusion, as disclosed in NCT03350035, at the dosage ranges disclosed in the prior art, including in During, would necessarily and inherently achieve the plasma concentration range disclosed in claim 1. The limitation in claim 1 of “about 500 ng/ml to about 1000 ng/ml for about 8 hours to about 12 hours” thus necessarily follows from administering a dose effective to suppress SE.

116. Thus, given the disclosures of During and NCT03350035, and in light of the background art as explained above, a POSA would have been motivated to administer ganaxolone to treat SE according to Claim 1 with a reasonable expectation of success. Therefore, claim 1 would have been obvious.

b. Dependent Claims 2, 3, 11, and 12 Would Have Been Obvious in View of During and NCT03350035

117. I also considered whether claims 2, 3, 11, and 12 would have been obvious over During and NCT03350035. Dependent claims 2, 3, 11, and 12 recite further limitations on, and requirements for, the intravenous bolus dose of ganaxolone. Specifically, claim 2 further requires that the intravenous bolus comprises about 5 to about 40 mg of ganaxolone; claim 3 further requires that the intravenous bolus comprises 30 mg of ganaxolone; claim 11 requires the intravenous bolus to produce a ganaxolone plasma concentration of about 500 ng/ml to about

1000 ng/ml; and claim 12 requires that the intravenous bolus be administered for about 1 minute to about 5 minutes. During and NCT03350035 render these dependent claims obvious for the same reasons discussed above in Ground 1.

118. During states that “dosages” of allosteric modulators, including ganaxolone, may include “1 mg to 30 mg.” Ex. 1003, 6:51-54. This range of “1 mg to 30 mg” directly overlaps with the bolus doses of claims 2 (from about 5 mg to about 40 mg) and 3 (about 30 mg). Additionally, a bolus is used for the rapid achievement of the desired effects. As described above, a bolus dose is administered when a fast response is required, especially in emergency situations, such as SE. *See* Section IV. A POSA would have been motivated to use a bolus dose to suppress SE, i.e., to achieve plasma concentration levels required to achieve the desired clinical effect. Marinus’s own Phase 1 Clinical Trial Results demonstrated that “potential anti-convulsant plasma concentrations were achievable with a bolus dose.” Ex. 1007 at 1.

119. It would have been a routine practice for a POSA to determine the amount of a bolus dose of ganaxolone. A POSA treating a patient with SE would have relied on EEG readouts (as described above in Section IV), which measure electrical activity in the brain and are routinely used to monitor seizures, to make adjustments to the bolus dose to effectively achieve the desired outcome, i.e., the

suppression of SE. This is especially true because ganaxolone had already been shown to be safe and well tolerated at doses up to 1,500 mg/day (*see, e.g.*, Ex. 1023 at 558, 563 (finding that 1,500 mg/day ganaxolone exhibited “no clinically relevant laboratory or [electrocardiography (“ECG”)] changes during the study”)). Further, the time period of administering a bolus dose “for about 1 minute to about 5 minutes” in claim 12 is a standard time period for administering bolus doses.

120. As described in Section XI.A.1.a.i, a POSA would have targeted doses of ganaxolone between 600 mg/day to 1,500 mg/day for suppressing SE based on this study demonstrating ganaxolone’s efficacy in treating partial-onset seizures. In addition, it would have been routine and obvious for a POSA to optimize the ganaxolone dosing regimen—in particular, the amount of the intravenous bolus dose—based on the target dose, prior art disclosures, and the standard practices a POSA would use to suppress SE.

121. Thus, upon reading During and NCT03350035, the specific amount and timing of the intravenous bolus disclosed in claims 2, 3, 11, and 12, would have been obvious to a POSA.

c. Dependent Claims 4, 5, 7, and 9 Would Have Been Obvious in View of During and NCT03350035

122. I also considered whether claims 4, 5, 7, and 9 would have been obvious over During and NCT03350035. Dependent claims 4, 5, 7, and 9 recite further

requirements for, and modifications to, the rate of continuous intravenous infusion of ganaxolone. During and NCT03350035 render these dependent claims obvious for the same reasons discussed above in Ground 1.

123. As explained above in Section X.A.1.a.i, a POSA would have been led to target a dose between 600 mg/day and 1500 mg/day of ganaxolone to suppress SE. After administering the intravenous bolus as described above, a POSA would then be able to determine an hourly rate of infusion based on the EEG readout.

124. Further modification to arrive at the rates of continuous intravenous infusion recited in claims 4 (about 20 mg/hr to about 80 mg/hr), 5 (about 80 mg/hr for at least 2 hours), 7 (50 mg/hr for about 6 hours to about 10 hours), and 9 (20 mg/hr for about 12 hours to about 24 hours) would have been a routine practice for a POSA at the relevant time. Additionally, a POSA treating an SE patient regularly adjusts the rate of continuous intravenous infusion based on various metrics, including EEG readouts (as described above in Section IV)), which measure electrical activity in the brain and are routinely used to monitor seizures. Based on the EEG of a patient, a POSA would know to modify the rate of continuous intravenous infusion of ganaxolone for a subject to more effectively suppress SE.

125. It would have been routine and obvious to a POSA to optimize the ganaxolone dosing regimen—in particular, the rate of continuous intravenous

infusion—based on the target dose, prior art disclosures and the standard practices a POSA would use to suppress SE.

126. Thus, upon reading During and NCT03350035, the hourly rate of ganaxolone infused into the subject by continuous intravenous infusion, and any modifications thereof, including the further limitations recited in claims 4, 5, 7, and 9, would have been obvious to a POSA.

d. Dependent Claims 6, 8, 10, 13, 15, 17, and 20 Would Have Been Obvious in View of During and NCT03350035

127. I also considered whether claims 6, 8, 10, 13, 15, 17, and 20 would have been obvious over During and NCT03350035. Dependent claims 6, 8, 10, 13, 15, 17, and 20 recite a reduction or decrease in the amount of ganaxolone infused into the subject by continuous intravenous infusion. During and NCT03350035 render these dependent claims obvious for the same reasons discussed above in Ground 1.

128. At the relevant time period, it was well known in the art that a reduction or decrease in the amount of an antiepileptic drug infused into a subject by continuous intravenous infusion, often called a taper, was desirable. *See supra* Section IV.A. This is to reduce the risk of withdrawal seizures or even a recurrence of SE. *Id.*

129. As described in Section XI.A.1.a.i, a POSA would have targeted doses of ganaxolone between 600 mg/day to 1,500 mg/day for suppressing SE based on

this study demonstrating ganaxolone's efficacy in treating partial-onset seizures. In addition, it would have been routine and obvious to a POSA to optimize the ganaxolone dosing regimen—in particular, the rate of taper—based on the target dose, prior art disclosures and the standard practices a POSA would use to suppress SE.

130. Thus, upon reading During and NCT03350035, it would have been obvious to a POSA to include a reduction or decrease in the amount of ganaxolone infused into the subject by continuous intravenous infusion as recited in claims 6, 8, 10, 13, 15, 17, and 20.

e. Dependent Claims 14, 16, 18, 19, and 21 Would Have Been Obvious in View of During and NCT03350035

131. I also considered whether claims 14, 16, 18, 19, and 21 would have been obvious over During and NCT03350035. Dependent claims 14, 18, 19, and 21 recite that a subject in need “has refractory status epilepticus.” Dependent claim 16 recites that the total daily dose of ganaxolone administered is from about 500 mg to about 900 mg. During and NCT03350035 render these dependent claims obvious for the same reasons discussed above in Ground 1.

132. In addition, it would have been obvious to a POSA that a subject in need would include a patient with RSE. Indeed, During specifically discloses administering ganaxolone to treat SE, including RSE. Ex. 1003 at 2:13-22, 4:6-16,

10:28-42. It also would have been well known to a POSA at the relevant time that a subset of patients with SE have RSE. Thus, upon reading During and NCT03350035, it would have been obvious to a POSA that subjects in need of the disclosed ganaxolone treatment method includes patients with RSE.

133. In addition, it would have been routine and obvious for a POSA to optimize the ganaxolone dosing regimen—in particular, the total daily dose—based on the prior art disclosures and the standard practices a POSA would use to suppress SE.

134. Thus, upon reading During and NCT03350035, the claimed inventions of claims 14, 16, 18, 19, and 21 would have been obvious to a POSA.

2. Ground 2: All Claims of the '100 Patent Would Have Been Obvious in View of During and Marinus's Phase 1 Clinical Trial Results

a. Claim 1

i. A POSA Would Have Been Motivated to Combine During and Marinus's Phase 1 Clinical Trial Results

135. As part of my obviousness analysis, I considered whether a POSA would have been motivated to combine the disclosures of During and Marinus's Phase 1 Clinical Trial Results (i.e., Tsai 2016, Tsai 2017, and Clinical Leader 2016) as of August 5, 2019. For the reasons I describe below, I conclude that a POSA

would have had several reasons to combine the disclosures of During and the Phase 1 Clinical Trial Results to arrive at the claimed invention.

136. Both During and Marinus's Phase 1 Clinical Trial Results relate to the use of ganaxolone to treat epileptic disorders, including SE.

137. During teaches administering ganaxolone to treat SE. Ex 1003 at 8:11-14. In particular, Example 3 of During describes an evaluation of ganaxolone's ability to block benzodiazepine-resistant SE and concludes that there was significant improvement in seizure protection at 20 mg/kg ganaxolone. Ex. 1003 at 35:33-35. During's claims also recite methods of treating SE by administering the following doses of ganaxolone: "about 150 mg to 1000 mg/day," "about 500 mg to 1000 mg/day," "about 625 mg to 650 mg/day," "about 650 mg to 675 mg/day," "about 675 mg to 700 mg/day," "about 625 mg to 700 mg/day," "about 700 mg to 725 mg/day," "about 1025 mg to 1050 mg/day." Ex. 1003 at 40:18-33.

138. Marinus's Phase 1 Clinical Trial Results (i.e., Tsai 2016, Tsai 2017, and Clinical Leader 2016) describe a Phase 1 study to evaluate the safety, pharmacokinetics (PK), and pharmacodynamics (PD) of ganaxolone administered as a bolus dose and continuous infusion to healthy participants. Ex. 1006 at S77-S78; Ex. 1005 at 74; Ex. 1007 at 1. Tsai 2016 states that one of the stages of the Phase 1 study "will determine the PK, safety, and PD of bolus IV dosing followed

by continuous infusion.” Ex. 1006 at S78. Tsai 2017 reports the pK results of the Phase 1 study as showing that ganaxolone concentrations in plasma “were generally proportional to the administered dose. Potential anti-convulsant plasma concentrations were achievable with a bolus dose. After the IV bolus dose or cessation of the IV infusion dose, concentrations of GNX in plasma declined in a multi-phasic manner.” Ex. 1005 at 75. Tsai 2017 concludes that “GNX administered as a rapid IV bolus or bolus with continuous infusion was generally safe and well tolerated” and that “[p]redictable PK from this study enables dosing guidance for GNX IV.” Ex. 1005 at 75. Similar results were presented in Clinical Leader 2016. Ex. 1007 at 1. Therefore, a POSA would have understood that these disclosures demonstrate that there was nothing unpredictable about dosing ganaxolone as a bolus dose followed by continuous infusion. Moreover, a POSA would have been motivated to optimize the dose to suppress SE.

139. Thus, During specifically addresses the use of ganaxolone to treat SE, including specific dose ranges, and Marinus’s Phase 1 Clinical trials specifically addresses the use of ganaxolone as an IV loading dose (or bolus) with continuous infusion.

140. A POSA also would have targeted doses of ganaxolone between 600 mg/day to 1,500 mg/day for suppressing SE based on this study demonstrating

ganaxolone's efficacy in treating partial-onset seizures. Based on this direction, a POSA would have been motivated to select from During about 625-725 or 1025-1050 mg/day of ganaxolone to suppress SE. Additionally, because ganaxolone is administered intravenously to treat SE, a POSA would have been motivated to select a lower dose within the range of 600-1,500 mg/day because a POSA would have understood that a drug administered intravenously has 100% bioavailability because it is delivered directly to the bloodstream, while a drug administered orally has lower bioavailability. *See, e.g.*, Ex. 1034 at 222.

141. Based on During's disclosures (directed to ganaxolone's evaluation of the ability of ganaxolone to block benzodiazepine-resistant SE and disclosure of dose ranges) and Marinus's Phase 1 Clinical Trial Results (directed to the administration of ganaxolone as an IV loading dose with continuous infusion), a POSA would have been motivated to use ganaxolone to suppress SE by administering ganaxolone as an intravenous bolus dose followed by continuous infusion. These disclosures also would have motivated a POSA to optimize the dose to suppress SE.

ii. A POSA Would Have Had a Reasonable Expectation of Successfully Combining During and Marinus's Phase 1 Clinical Trial Results

142. I considered whether claim 1 would have been obvious over During and Marinus's Phase 1 Clinical Trial Results. Based on the combination of During and the Phase 1 Clinical Trial Results, a POSA would have expected ganaxolone administered as an IV bolus dose with continuous infusion would suppress SE and produce and maintain a ganaxolone plasma concentration of about 500 ng/ml to about 1000 ng/ml for about 8 hours to about 12 hours.

143. As explained above, During discloses administering ganaxolone to treat SE, including by administering the following doses of ganaxolone: "about 150 mg to 1000 mg/day," "about 500 mg to 1000 mg/day," "about 625 mg to 650 mg/day," "about 650 mg to 675 mg/day," "about 675 mg to 700 mg/day," "about 625 mg to 700 mg/day," "about 700 mg to 725 mg/day," "about 1025 mg to 1050 mg/day." Ex. 1003 at 8:11-14; 40:18-33.

144. The Phase 1 Clinical Trial Results specifically address the use of ganaxolone as an IV bolus dose with continuous infusion to suppress SE. Ex. 1005 at 74-75; Ex. 1006 at S77-S78. Tsai 2016 discloses that Stage 2 of the study "will determine the PK, safety, and PD of bolus IV dosing followed by continuous infusion." Ex. 1006 at S78. Tsai 2017 describes the results of a Phase 1 study that

evaluated the “safety, pharmacokinetics (PK), and pharmacodynamics (PD) of [ganaxolone (GNX)] IV administered as a bolus dose and continuous infusion to healthy patients” and concluded that ganaxolone administered as a bolus followed by continuous infusion was “generally safety and well tolerated; there were no safety issues that limit getting to a potentially efficacious plasma concentration in a short period of time” and that “[p]redictable PK from this study enables dosing guidance for GNX IV.” Ex. 1005 at 74-75.

145. As discussed above in Section XI.A.1.a.ii, a POSA also would have considered dosing of ganaxolone and other GABA_A modulators like allopregnanolone when determining an effective dose of ganaxolone for suppressing SE.

146. For example, as described in Section IV.B.1, Marinus reported the results of a Phase 2 study of ganaxolone as an add-on therapy in adults with partial-onset seizures, which demonstrated that 1,500 mg/day ganaxolone (after titrating up from 600 mg/day) was effective at reducing partial-onset seizure frequency. Ex. 1023 at 558. It also reported that, following oral administration, ganaxolone is rapidly absorbed and the plasma concentration sharply decreases over the first 12 h and then by a terminal half-life of approximately 20 hours. Ex. 1023 at 558.

147. A POSA also would have had a reasonable expectation that these ranges reflect effective amounts of ganaxolone to suppress SE because of (1) During's finding that ganaxolone was effective to induce significant protection against SE in the SE mouse model (Ex. 1003 at ¶ [0180]) and (2) Marinus's own Phase 1 work, which reported that the study "provided predictable PK to enable dosing of ganaxolone IV in clinical studies in SE" that was "based on" Marinus's partial-onset seizure study (Ex. 1007 at 1; Ex. 1025 at 1). Additionally, a POSA would have had a reasonable expectation of success because the Phase 1 study also found that ganaxolone plasma concentrations were "generally proportional to the dose administered" and that the bolus dose achieved "potential anti-convulsant plasma concentrations"

148. Moreover, and as explained in Section IV.B.2, a POSA would have considered previous administrations of allopregnanolone to treat SE when determining doses of ganaxolone effective to suppress SE. Ganaxolone is an analog of allopregnanolone and both are allosteric modulators of the GABA_A receptor. Ex. 1033, 1. Marinus's Press Releases also state that ganaxolone was "designed to have similar GABA modulation effects as allopregnanolone without unwanted steroidal side effects." Ex. 1033 at 1.

149. A 2017 study found that 670.8 mg allopregnanolone administered as a 5.6 mg/h continuous infusion over 5 days “successfully treated” two patients with super-refractory SE (“SRSE”). Ex. 1028 at 411-12. A year later, a study into the mouse model of SE confirmed that allopregnanolone and ganaxolone administered at the same dose terminated SE and found that the same dose of each drug achieved “comparable plasma levels . . . (C_{max} , 645 ng/mL for allopregnanolone and 550 ng/mL for ganaxolone). Ex. 1027 at 223.

150. This information related to allopregnanolone (i.e., that it has comparable plasma concentration levels in treating SE as ganaxolone and successfully treated patients with SE using 670.8 mg of intravenous allopregnanolone) also would have provided a POSA with a starting point to determine an amount of ganaxolone effective to suppress SE and a reasonable expectation of doing so.

151. To the extent that During and Marinus’s Phase 1 Clinical Trial Results do not disclose the ganaxolone plasma concentration ranges of claim 1, this limitation would have been obvious because a POSA would have recognized that administering ganaxolone as an intravenous bolus plus continuous infusion at the dose ranges described in During, would produce and maintain a ganaxolone plasma concentration of about 500 ng/ml to about 1000 ng/ml for about 8 hours to about 12

hours in a typical patient. Indeed, Marinus's Phase 1 Clinical Trial Results specifically discloses that ganaxolone demonstrated "[p]redictable PK." Ex. 1015 at 75; Ex. 1007 at 1. A POSA thus would have known that the blood plasma concentration levels disclosed in claim 1 necessarily flow from the dosing method disclosed by Marinus's Phase 1 Clinical Trial Results at the dosage ranges described in During.

152. Further, and as discussed above in Section IV.B, Marinus reported the results of a Phase 2 study of ganaxolone as an add-on therapy in adults with partial-onset seizures, demonstrating that 1,500 mg/day ganaxolone was effective at reducing partial-onset seizure frequency. Ex. 1023 at 558. It was also known that following oral administration, ganaxolone is rapidly absorbed and the plasma concentration sharply decreases over the first 12 h and then by a terminal half-life of approximately 20 hours. Ex. 1023 at 558. This study demonstrates the efficacy of ganaxolone in seizure disorders and identifies a range of oral doses that were being used successfully to treat epilepsy disorders. A POSA thus would have been led by the oral doses disclosed in Marinus's Phase 2 study to target a dose between 600 mg/day and 1500 mg/day. Such a range would have been obvious for a POSA to try in light of the prior art, as described above.

153. In other words, administering ganaxolone as an intravenous bolus plus continuous infusion, as disclosed in Marinus's Phase 1 Clinical Trial Results, and at the dose ranges disclosed in During and in Sperling 2017, would inherently achieve the plasma concentration range disclosed in claim 1. The claim 1 limitation of "about 500 ng/ml to about 1000 ng/ml" thus necessarily follows from administering a dose effective to suppress SE, and a POSA would have known this as of August 5, 2019.

154. Because a POSA would have known that administering ganaxolone as an intravenous bolus plus continuous infusion at the doses disclosed in the prior art would produce a ganaxolone plasma concentration of about 500 ng/ml to about 1000 ng/ml for about 8 hours to about 12 hours in a typical patient, it would have been obvious to administer ganaxolone to suppress SE in a manner that achieves the pK ranges of Claim 1. Thus, given the disclosures of During and Marinus's Phase 1 Clinical Trial Results, a POSA would have been motivated to administer ganaxolone to treat SE according to claim 1 with a reasonable expectation of success.

b. Dependent Claims 2, 3, 11, and 12 Would Have Been Obvious in View of During and Marinus's Phase 1 Clinical Trial Results

155. I also considered whether claims 2, 3, 11, and 12 would have been obvious over During and Marinus's Phase 1 Clinical Trial Results. During and the

Phase 1 Clinical Trial Results render these dependent claims obvious for the same reasons discussed above in Ground 1.

156. As described in Section XI.A.1.a.i, a POSA would have targeted doses of ganaxolone between 600 mg/day to 1,500 mg/day for suppressing SE based on this study demonstrating ganaxolone's efficacy in treating partial-onset seizures. In addition, it would have been routine and obvious for a POSA to optimize the ganaxolone dosing regimen—in particular, the amount of the intravenous bolus dose—based on the target dose, prior art disclosures, and the standard practices a POSA would use to suppress SE.

157. For the reasons I describe above, *see* Sections XI.A.1.b and XI.A.2.a.ii, I conclude that a POSA would have been motivated to combine the disclosures of During and Marinus's Phase 1 Clinical Trial Results to arrive at the claimed invention of claims 2, 3, 11, and 12 with a reasonable expectation of success.

c. Dependent Claims 4, 5, 7, and 9 Would Have Been Obvious in View of During and Marinus's Phase 1 Clinical Trial Results

158. I also considered whether claims 4, 5, 7, and 9 would have been obvious over During and Marinus's Phase 1 Clinical Trial Results. During and the Phase 1 Clinical Trial Results render these dependent claims obvious for the same reasons discussed above in Ground 1.

159. As described in Section XI.A.1.a.i, a POSA would have targeted doses of ganaxolone between 600 mg/day to 1,500 mg/day for suppressing SE based on this study demonstrating ganaxolone's efficacy in treating partial-onset seizures. For the reasons I describe above, *see* Section XI.A.1.c, it would have been routine and obvious to a POSA to optimize the ganaxolone dosing regimen—in particular, the rate of continuous intravenous infusion—based on the target dose, prior art disclosures and the standard practices a POSA would use to suppress SE.

160. Thus, for the reasons I describe above, *see* Sections XI.A.1.c and XI.A.2.a.ii, I conclude that a POSA would have been motivated to combine the disclosures of During and Marinus's Phase 1 Clinical Trial Results to arrive at the claimed invention of claims 4, 5, 7, and 9 with a reasonable expectation of success.

d. Dependent Claims 6, 8, 10, 13, 15, 17, and 20 Would Have Been Obvious in View of During and the Phase 1 Clinical Trial Results

161. I also considered whether claims 6, 8, 10, 13, 15, 17, and 20 would have been obvious over During and the Phase 1 Clinical Trial Results. During and the Phase 1 Clinical Trial Results render these dependent claims obvious for the same reasons discussed above in Ground 1.

162. As described in Section XI.A.1.a.i, a POSA would have targeted doses of ganaxolone between 600 mg/day to 1,500 mg/day for suppressing SE based on

this study demonstrating ganaxolone's efficacy in treating partial-onset seizures. In addition, it would have been routine and obvious to a POSA to optimize the ganaxolone dosing regimen—in particular, the rate of taper—based on the target dose, prior art disclosures and the standard practices a POSA would use to suppress SE.

163. For the reasons I describe above, *see* Sections XI.A.1.d and XI.A.2.a.ii, I conclude that a POSA would have been motivated to combine the disclosures of During and the Phase 1 Clinical Trial Results to arrive at the claimed invention of claims 6, 8, 10, 13, 15, 17, and 20 with a reasonable expectation of success.

e. Dependent Claims 14, 16, 18, 19, and 21 Would Have Been Obvious in View of During and the Phase 1 Clinical Trial Results

164. I also considered whether claims 14, 16, 18, 19, and 21 would have been obvious over During and the Phase 1 Clinical Trial Results. Dependent claims 14, 18, 19, and 21 recite that a subject in need “has refractory status epilepticus.” Dependent claim 16 recites that the total daily dose of ganaxolone administered is from about 50 mg to about 900 mg. During and the Phase 1 Clinical Trial Results render these dependent claims obvious for the same reasons discussed above in Ground 1.

165. As described in Section XI.A.1.a.i, a POSA would have targeted doses of ganaxolone between 600 mg/day to 1,500 mg/day for suppressing SE based on this study demonstrating ganaxolone's efficacy in treating partial-onset seizures.

166. For the reasons I describe above, *see* Sections XI.A.1.e and XI.A.2.a.ii, I conclude that a POSA would have been motivated to combine the disclosures of During and the Phase 1 Clinical Trial Results to arrive at the claimed invention of Claims 14, 16, 18, 19, and 21 with a reasonable expectation of success.

B. All Claims of the '100 Patent Are Unpatentable Over Marinus's Own Phase 2 Publications

1. Ground 3: All Claims of the '100 Patent Would Have Been Obvious in View of During, NCT03350035, and the Phase 2 Clinical Trial Results

a. Claim 1

i. A POSA Would Have Been Motivated to Combine During, NCT03350035, and the Phase 2 Clinical Trial Results

167. As part of my obviousness analysis, I considered whether a POSA would have been motivated to combine the disclosures of During, NCT03350035, and the Phase 2 Clinical Trial Results as of August 4, 2020. For the reasons I describe below, I conclude that a POSA would have had several reasons to combine the disclosures of During, NCT03350035, and the Phase 2 Clinical Trial Results to arrive at the claimed invention.

168. During, NCT03350035, and the Phase 2 Clinical Trial Results relate to the use of ganaxolone to treat SE.

169. During teaches administering ganaxolone to treat SE. Ex. 1003 at 8:11-14. Example 3 of During describes an evaluation of ganaxolone's ability to block benzodiazepine-resistant SE and concludes that there was significant improvement in seizure protection at 20 mg/kg ganaxolone. Ex. 1003 at 35:33-35. During's claims also recite methods of treating SE by administering the following doses of ganaxolone: "about 150 mg to 1000 mg/day," "about 500 mg to 1000 mg/day," "about 625 mg to 650 mg/day," "about 650 mg to 675 mg/day," "about 675 mg to 700 mg/day," "about 625 mg to 700 mg/day," "about 700 mg to 725 mg/day," "about 1025 mg to 1050 mg/day." Ex. 1003 at 40:18-33.

170. NCT03350035 is a study to evaluate the safety, tolerability, and efficacy of adjunctive IV ganaxolone in subjects with SE. In Clinical Trial NCT03350035, ganaxolone was administered as an "IV loading dose with continuous infusion (maintenance dose) for 2-4 days followed by a 24-hour taper." Ex. 1004 at 3-4. A POSA would have understood that a "loading dose" is the same as a "bolus dose."

171. The Phase 2 Clinical Trial Results (i.e., Practical Neurology 2019, Ramsay 2019, AES Poster 2019, and Husain 2020) all describe the results of a Phase

2 clinical trial for treating RSE with ganaxolone. Each also reports that the target dose (713 mg/day) achieved plasma concentrations above 500 ng/ml for approximately 8 hours. Ex. 1010 at 1-2 (“The PK Data show that the target dose of ganaxolone (713 mg/day) achieved plasma drug concentrations greater than 500 ng/mL for approximately 8 hours.”); Ex. 1008 at 2 (describing subjects dosed with 713 mg/day ganaxolone had plasma levels that were “able to be maintained ~500 ng/mL for ~8 hrs” and had “[g]ood seizure control”); Ex. 1011 at 1 (concluding “Target Dose [713 mg/day] Achieves 500 ng/mL for ~8 hours”); Ex. 1009 at 3 (noting that “[o]nly evaluable patients in the Target dose achieved at least an 80% sustained reduction in seizure burden for the entire analysis window (24 hrs) whereas seizure activity partially returned in the Low and Medium doses at times aligning with decreased GNX exposure”).)

172. Thus, During specifically addresses the use of ganaxolone to treat SE, including specific dose ranges, NCT03350035 also specifically addresses the use of ganaxolone to treat SE as an IV loading dose (or bolus) with continuous infusion, the Phase 2 Clinical Trial Results specifically addresses administering 713 mg/day ganaxolone to achieve a plasma concentration of > 500 ng/ml for about 8 hours and suppressing SE.

173. Based on During's disclosures (directed to ganaxolone's evaluation of the ability of ganaxolone to block benzodiazepine-resistant SE and disclosure of dose ranges), the disclosures in NCT03350035 Clinical Trial (directed to the administration of ganaxolone as an IV bolus dose with continuous infusion in subjects with SE) and the Phase 2 Clinical Trial Results (directed to administering 713 mg/day ganaxolone to achieve a plasma concentration of > 500 ng/ml for about 8 hours and suppressing SE), a POSA would have been motivated to use ganaxolone to suppress SE.

ii. A POSA Would Have Had a Reasonable Expectation of Success in Combining During, NCT03350035, and the Phase 2 Clinical Trial Results

174. I considered whether Claim 1 would have been obvious over During, NCT03350035, and the Phase 2 Clinical Trial Results. Based on the combination of During, NCT03350035, and the Phase 2 Clinical Trial Results, a POSA would have expected ganaxolone administered as an IV loading dose with continuous infusion would suppress SE and produce and maintain a ganaxolone plasma concentration of about 500 ng/ml to about 1000 ng/ml for about 8 hours to about 12 hours.

175. As explained above, During teaches administering ganaxolone to treat SE, including by administering the following doses of ganaxolone: "about 150 mg to 1000 mg/day," "about 500 mg to 1000 mg/day," "about 625 mg to 650 mg/day,"

“about 650 mg to 675 mg/day,” “about 675 mg to 700 mg/day,” “about 625 mg to 700 mg/day,” “about 700 mg to 725 mg/day,” “about 1025 mg to 1050 mg/day.” Ex. 1003 at 8:11-14, 40:18-33.

176. NCT03350035 specifically addresses the use of ganaxolone as an IV loading dose (i.e., bolus) with continuous infusion in subjects with SE.

177. And the Phase 2 Clinical Trial Results describe the results of a Phase 2 clinical trial for treating RSE with ganaxolone. (Ex. 1008 at 1-2; Ex. 1009 at 332; Ex. 1010 at 1-2; Ex. 1011 at 1.) Each also reports that the target dose (713 mg/day) achieved plasma concentrations above 500 ng/ml for approximately 8 hours. Ex. 1010 at 1-2 (“The PK Data show that the target dose of ganaxolone (713 mg/day) achieved plasma drug concentrations greater than 500 ng/mL for approximately 8 hours.”); Ex. 1008 at 2 (describing subjects dosed with 713 mg/day ganaxolone had plasma levels that were “able to be maintained ~500 ng/mL for ~8 hrs” and had “[g]ood seizure control”); Ex. 1011 at 1 (concluding “Target Dose [713 mg/day] Achieves 500 ng/mL for ~8 hours”); Ex. 1009 at 332 (noting that “[o]nly evaluable patients in the Target dose achieved at least an 80% sustained reduction in seizure burden for the entire analysis window (24 hrs) whereas seizure activity partially returned in the Low and Medium doses at times aligning with decreased GNX exposure”).)

178. Because a POSA would have known that administering ganaxolone as an intravenous bolus plus continuous infusion at or around 713 mg/day would produce and maintain a ganaxolone plasma concentration of about 500 ng/ml to about 1000 ng/ml for about 8 hours to about 12 hours in a typical patient, it would have been obvious to administer ganaxolone to treat SE in the manner recited in claim 1. Thus, given the disclosures of During, NCT03350035, and the Phase 2 Clinical Trial Results, a POSA would have been motivated to administer ganaxolone to treat SE according to claim 1 with a reasonable expectation of success.

b. Dependent Claims 2, 3, 11, and 12 Would Have Been Obvious in View of During, NCT03350035, and the Phase 2 Clinical Trial Results

179. I also considered whether claims 2, 3, 11, and 12 would have been obvious over During, NCT03350035, and the Phase 2 Clinical Trial Results. During, NCT03350035, and the Phase 2 Clinical Trial Results render these dependent claims obvious for the same reasons discussed above in Ground 1.

180. As described in Section XI.A.1.a.i, a POSA would have targeted doses of ganaxolone between 600 mg/day to 1,500 mg/day for suppressing SE based on this study demonstrating ganaxolone's efficacy in treating partial-onset seizures. In addition, it would have been routine and obvious for a POSA to optimize the ganaxolone dosing regimen—in particular, the amount of the intravenous bolus

dose—based on the target dose, prior art disclosures, and the standard practices a POSA would use to suppress SE.

181. For the reasons I describe above, *see* Sections XI.A.2.b and XI.B.1.a, I conclude that a POSA would have been motivated to combine the disclosures of During, NCT03350035, and the Phase 2 Clinical Trial Results to arrive at the claimed invention of claims 2, 3, 11, and 12 with a reasonable expectation of success.

c. Dependent Claims 4, 5, 7, and 9 Would Have Been Obvious in View of During, NCT03350035, and the Phase 2 Clinical Trial Results

182. I also considered whether claims 4, 5, 7, and 9 would have been obvious over During, NCT03350035, and the Phase 2 Clinical Trial Results. During, NCT03350035, and the Phase 2 Clinical Trial Results render these dependent claims obvious for the same reasons discussed above in Ground 1.

183. As described in Section XI.A.1.a.i, a POSA would have targeted doses of ganaxolone between 600 mg/day to 1,500 mg/day for suppressing SE based on this study demonstrating ganaxolone’s efficacy in treating partial-onset seizures. For the reasons I describe above, *see* Sections X.A.4 and XI.D.2, it would have been routine and obvious to a POSA to optimize the ganaxolone dosing regimen—in particular, the rate of continuous intravenous infusion—based on the target dose, prior art disclosures and the standard practices a POSA would use to suppress SE.

184. Thus, for the reasons I describe above, *see* Sections XI.A.2.c and XI.B.1.a, I conclude that a POSA would have been motivated to combine the disclosures of During, NCT03350035, and the Phase 2 Clinical Trial Results to arrive at the claimed invention of claims 4, 5, 7, and 9 with a reasonable expectation of success.

d. Dependent Claims 6, 8, 10, 13, 15, 17, and 20 Would Have Been Obvious in View of During, NCT03350035, and the Phase 2 Clinical Trial Results

185. I also considered whether claims 6, 8, 10, 13, 15, 17, and 20 would have been obvious over During, NCT03350035, and the Clinical Trial 2 Study Results. During, NCT03350035, and the Phase 2 Clinical Trial Results render these dependent claims obvious for the same reasons discussed above in Ground 1.

186. As described in Section XI.A.1.a.i, a POSA would have targeted doses of ganaxolone between 600 mg/day to 1,500 mg/day for suppressing SE based on this study demonstrating ganaxolone's efficacy in treating partial-onset seizures. In addition, it would have been routine and obvious to a POSA to optimize the ganaxolone dosing regimen—in particular, the rate of taper—based on the target dose, prior art disclosures and the standard practices a POSA would use to suppress SE.

187. For the reasons I describe above, *see* Sections XI.A.2.d and XI.B.1.a, I conclude that a POSA would have been motivated to combine the disclosures of During, NCT03350035, and the Phase 2 Clinical Trial Results to arrive at the claimed invention of claims 6, 8, 10, 13, 15, 17, and 20 with a reasonable expectation of success.

e. Dependent Claims 14, 16, 18, 19, and 21 Would Have Been Obvious in View of During, NCT03350035, and the Phase 2 Clinical Trial Results

188. I also considered whether claims 14, 16, 18, 19, and 21 would have been obvious over During, NCT03350035, and the Phase 2 Clinical Trial Results. Dependent claims 14, 18, 19, and 21 recite that a subject in need “has refractory status epilepticus.” Dependent claim 16 recites that the total daily dose of ganaxolone administered is from about 50 mg to about 900 mg. During, NCT03350035, and the Phase 2 Clinical Trial Results render these dependent claims obvious for the same reasons discussed above in Ground 1.

189. As described in Section XI.A.1.a.i, a POSA would have targeted doses of ganaxolone between 600 mg/day to 1,500 mg/day for suppressing SE based on this study demonstrating ganaxolone’s efficacy in treating partial-onset seizures. In addition, it would have been routine and obvious for a POSA to optimize the

ganaxolone dosing regimen—in particular, the total daily dose—based on the prior art disclosures and the standard practices a POSA would use to suppress SE.

190. For the reasons I describe above, *see* Sections XI.A.2.e and XI.B.1.a, I conclude that a POSA would have been motivated to combine the disclosures of During, NCT03350035, and the Phase 2 Clinical Trial Results to arrive at the claimed invention of claims 14, 16, 18, 19, and 21 with a reasonable expectation of success.

XII. No Secondary Considerations of Nonobviousness

191. I understand that during prosecution Dr. Joseph Hulihan, one of the named inventors of the '100 patent, submitted a declaration that the inventors of the '100 patent found that “[i]t was completely unexpected that providing ganaxolone at a serum concentration of about 500 ng/ml to about 1000 ng/ml for a short duration of only about 8 to about 12 hours, resulted in persistent SE suppression. In fact, it was completely contrary to our expectations.” Ex. 1029 at 580, ¶ 10.

192. In my opinion, this result would not have been surprising or unexpected to a POSA in view of During’s disclosure of similar doses of ganaxolone to treat SE (e.g., “625 to 700 mg/day,” “700 mg to 725 mg/day,” or “1025 to 1050 mg/day”) that would inherently reach the same ganaxolone plasma concentration that Dr. Hulihan describes as unexpected. Thus, it would not have been unexpected for a

Declaration of Raman Sankar, M.D., Ph.D.
in support of Petition for an Inter Partes Review
of U.S. Patent No. 11,110,100 B2

POSA to achieve suppression of SE by administering an effective amount of ganaxolone via an intravenous bolus plus continuous infusion to produce and maintain a ganaxolone plasma concentration of about 500 ng/ml to about 1000 ng/ml for about 8 hours to about 12 hours.

193. I am not aware of any other alleged secondary considerations of nonobviousness that are pertinent to the obviousness inquiry in this case. I have been instructed that, to the extent that Marinus raises any secondary considerations of nonobviousness in its response, I will have the chance to evaluate them and submit a reply. This applies to each obviousness ground (Grounds 1, 2, and 5).

I hereby declare that all statements made herein of my own knowledge are true and that all statements made upon information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like so are made punishable by fine or imprisonment, or both, under § 1001 of Title 18 of the United States Code.

Declaration of Raman Sankar, M.D., Ph.D.
in support of Petition for an Inter Partes Review
of U.S. Patent No. 11,110,100 B2

Respectfully submitted,



Digitally signed by Raman Sankar, MD, PhD
DN: cn=Raman Sankar, MD, PhD, o=UCLA, ou=Pediatric
Neurology, email=RSankar@ucla.edu, c=US
Date: 2024.02.18 16:43:36 -08'00'

18 FEB 2024
Date: _____

Raman Sankar, M.D., Ph.D.

Appendix A

RAMAN SANKAR, PhD, MD, FAAN, FAES
Division of Pediatric Neurology
David Geffen School of Medicine at UCLA
UCLA Mattel Children's Hospital and UCLA Children's Discovery and Innovation Institute
Room 22-474 MDCC
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310-794-1014; Fax 310-825-5834
Admin: Raquel Gallegos (RGallegos@mednet.ucla.edu): 310-206-6870

CURRICULUM VITAE

ACADEMIC POSITIONS:

2022 – present	Professor and Emeritus Chief
2005 – 2022	Professor and Chief, Rubin Brown Endowed Chair Division of Pediatric Neurology David Geffen School of Medicine at UCLA
1999 – 2005	Associate Professor Director of Residency Training in Child Neurology David Geffen School of Medicine at UCLA
1992 – 1999	Assistant Professor UCLA School of Medicine
1991 – 1992	Clinical Instructor of Pediatric Neurology UCLA School of Medicine
1989 – Present	Consulting Pediatric Neurologist Kern County Medical Center (affiliated with UCLA) Bakersfield, California
1997 – 1999	Consulting Pediatric Neurologist Bakersfield Family Medical Center, Bakersfield, California
1979 - 1986	Adjunct Assistant Professor of Pharmacology Tulane University School of Medicine, New Orleans. Lectures to pharmacology graduate students
1982 - 1986	Adjunct Associate Professor of Medicinal Chemistry Xavier University College of Pharmacy, New Orleans
1975 - 1982	Associate Professor of Medicinal Chemistry (80-82) Assistant Professor of Medicinal Chemistry (75-80) Xavier University College of Pharmacy, New Orleans Taught various courses in medicinal chemistry and directed undergraduate research
1974 - 1975	Postdoctoral Research Associate School of Pharmacy, University of Washington

EDUCATION:

Post-Doctoral:	1989 - 1991	Fellow Division of Pediatric Neurology UCLA School of Medicine, Los Angeles, CA
	1988 - 1989	Resident Department of Neurology UCLA School of Medicine, Los Angeles, CA
	1986 - 1988	Pediatric Intern-Resident Children's Hospital of Los Angeles Los Angeles, California
Medical:	1982 - 1986	Tulane Medical School New Orleans, Louisiana
Graduate:	1968 - 1974	University of Washington, Seattle, Washington Ph.D. in Pharmaceutical Sciences (Medicinal Chemistry)
Undergraduate:	1965 - 1968	University of Bombay Bombay, India B.S. Tech. Technology of Pharmaceuticals and Fine Chemicals
	1961 - 1965	University of Bombay Bombay, India B.S. Major: Chemistry, Minor: Physics

MEDICAL LICENSES AND CERTIFICATIONS:

California License: A 043996; Louisiana License: 018831

DEA License: BS1107172

Diplomate of the American Board of Psychiatry and Neurology: **Neurology with Special Qualification in Child Neurology.** Certificate Number: 985 (1995); Recertified, 2005, **2016**

HONORS:

Academic Merit Scholarship, University of Bombay (1965-1968)

Rubenstein Fellow, School of Pharmacy, University of Washington, Seattle, Washington

Lawrence Ferring Faculty Award for the teacher of the year at the College of Pharmacy, Xavier University, New Orleans, 1982

Rho Chi, National Pharmaceutical Honor Society

Sigma Xi, The Scientific Research Society

New Orleans Pediatric Society Award, Tulane University Medical School Graduation, 1986

John Hughlings Jackson Fellow of the Epilepsy Foundation of America, 1992-93.

Award for "Volunteer of the Year" by the Epilepsy Foundation of Los Angeles and Orange Counties, July 9, 1995.

Special Recognition by the Board of Directors of the Venice Family Clinic (Summer 1996).

Listed in The Best Doctors in America, Woodward/White, Inc. 1996, again in 1998 – excerpt appeared in the Los Angeles Magazine, November 1996 as one of the "Best Doctors in LA." Since then, continuous listing in several lists.

Visiting Professor, *Massachusetts General Hospital & Tufts New England Medical Center*, Boston, March 1997.

Visiting Professor, *University of Manitoba*, Pediatrics & Neurology, Winnipeg, Manitoba, April 2000.

Visiting Professor, *University of Michigan* School of Medicine, Department of Neurology, Ann Arbor, MI, July 2000.

Elected to the Professional Advisory Board of the *Epilepsy Foundation*, April 2000.

Elected Member, Grants and Fellowships Committee, Epilepsy Foundation, April 2000.

Visiting Professor, *Yonsei University*, Seoul, South Korea, September 2001.

Visiting Professor, *Oregon Health Sciences University*, Portland, Oregon, September 18-19, 2002.

William F. Friedman Lecturer, David Geffen School of Medicine at UCLA, January 24, 2003.

Selected to NINDS,NIH, Special Emphasis Panel, ZNS1 SRB-W (07), on "Model Validation for Antiepileptogenic and Resistant Epilepsy Therapies," April 22, 2004

Selected as participant in an NINDS, NIH Workshop on "Animal Models of the Childhood Epilepsies," May 13-14, 2004.

Excellence in Teaching Award, from the Resident Physicians, Department of Neurology, June 23, 2004.

Elected *Fellow of the American Academy of Neurology* (FAAN), Fall 2004.

Invited to participate in the *Gordon Research Conference on Neuronal Synchronization*, Aug 2006.

Presentation at the NINDS, NIH, Workshop on Neonatal Seizures, June 31, 2007.

Visiting Lecturer Pro Tempore, The Neurological Institute, The *Cleveland Clinic Educational Foundation*, Cleveland, Ohio, July 25, 2008.

Visiting Professor, *Institute of Physiology, Academy of Sciences of the Czech Republic*, September 29, 2008, Prague, Czech Republic.

Professor Jagannathan Oration at the *Institute of Neurology, Madras Medical College*, February 5, 2009, Chennai, India.

Appointed to the Commission on Neurobiology of the *International League Against Epilepsy (ILAE)*, September 2009.

Visiting Professor, *King Edward Memorial Hospital – Seth Gordhandas Sunderdas Medical College, University of Bombay*, January 30, 2010, Mumbai, India.

Visiting Professor and **Eric Lothman Memorial Lecturer**, Department of Neurology, *University of Virginia*, April 23, 2010, Charlottesville, Virginia.

Listed in Southern California Super Doctors, 2011, 2012, 2013, 2014, 2015, 2016 (dating back to the nineties)

Listed in Castle Connolly America's Best Doctors (reprinted as Southern California's Top Doctors in Los Angeles Magazine), 2011, 2012, 2013, 2014, 2015, 2016 (dating back to the mid-nineties)

Visiting Professor at the Department of Neurology at the *Mayo Clinic*, Rochester, Minnesota, June 27-28, 2011.

Elected to American Pediatric Society, February, 2013.

Dr. Balagopal Raju oration at the *Institute of Child Health & The Hospital for Children, Madras Medical College*, Chennai, India, February 9, 2015.

The first **Professor Yukio Fukuyama Memorial Lecturer** at the 58th Annual Meeting of the *Japanese Society of Child Neurology*, Tokyo, Japan, June 4, 2016.

William Friedman Lecturer at the Pediatric Grand Rounds, *David Geffen School of Medicine at UCLA and Mattel Children's Hospital UCLA*, Los Angeles, California, January 13, 2017.

Edward Senz Lecturer at *UCSF Benioff Children's Hospital*, Oakland, California, November 27, 2018.

Elected as *Fellow of the American Epilepsy Society (FAES)*, December 2018.

Founders Award of the American Epilepsy Society – the highest honor bestowed by the Society, *In recognition of outstanding lifetime achievement in the field of epilepsy*. Awarded at the Annual Meeting in New Orleans, Louisiana, December 3, 2018.

Visiting Professor, Children's Hospital of Omaha, University of Nebraska, November 11, 2021.

RESEARCH INTERESTS:

Neurochemistry of GABA systems during status epilepticus in the developing rat brain.

Effects of seizures and chronic epilepsy in the developing rat brain. New models for chronic, recurrent seizures based on electrophysiologic (perforant path stimulation) and chemical (lithium-pilocarpine) models of experimental seizures.

Studies on seizure-induced apoptosis, necrosis, neurogenesis and epileptogenicity in the developing brain.

Glucose transporters in the developing brain – role in hypoxia-ischemia, seizures and epileptogenesis. Consequence of GLUT3 gene deficiency on cerebral structure and function (in collaboration with Dr. Sherin Devaskar's laboratory).

Developmental aspects of traumatic brain injury and aberrant plasticity (with Drs. Giza and Hovda).

Studies on the duration of status epilepticus as a modifier of neuronal injury and epileptogenicity in the developing brain.

Study of inflammation as a modifier of seizure-induced neuronal injury in the developing brain.

Explorations in neuroprotective interventions in the developing brain.

Development of fast throughput models to facilitate screening of candidate compounds for development-specific antiepileptogenic activity.

Seizures and behavioral consequences in the developing and adult rat brain – what is the neurochemical connection? Role of galanin and serotonin in brainstem structures (with Dr. Mazarati). These studies parallel clinical studies relating epilepsy, cognition and behavior under the direction of Dr. Rochelle Caplan.

GLUT3 gene disruption produces features of autism in the rodent (with Drs. Sherin Devaskar and Alcino Silva).

Neurochemistry of depression associated with epileptogenesis – studies of behavior, physiology, and serotonergic systems in kindling model and in post-status epilepticus epileptogenesis.

Plasticity of KCNQ channels during epileptogenesis in the immature brain.

Antiepileptogenesis and disease modification by modulation of neuroinflammation.

Epilepsy and comorbidities, including autism and ADHD, in rodent models.

Study of the safety and efficacy Lenti-D modified, single dose, autologous stem cells phase 2/3 trial for the treatment of childhood cerebral adrenoleukodystrophy (Multicenter study, PI Kohn, D)

Efficacy and Safety of Synthetic Cannabidiol in Infantile Spasms (Co-PI, sponsored by Insys Therapeutics)

Safety of Intranasal Diazepam in Cluster Seizures (PI, Acorda Therapeutics)

Studies on monoaminergic systems in mediating comorbidities of epilepsy in rodent models of epilepsy.

Role of intestinal microbiome in mediating the impact of stress on epileptogenesis.

Post-traumatic depression and epilepsy – mechanistic studies in rodents.

GRANT SUPPORT:

1992 - 93: John Hughlings Jackson Fellowship of the Epilepsy Foundation of America (\$30,000 for Research/Clinical Award).

1994 - 97: NINDS - NIH - NS28383, for "Study of growth cone markers and neurotrophins in human epileptic tissue," of Pediatric Epilepsy Surgery Research Program Project (\$58,329 Coinvestigator with Dr. H.I. Kornblum, Project 3 of Program Project Grant. Principal Investigator: W. Donald Shields, MD)

1995 - 2000: NINDS - NIH – KO8 NS01792 "Effect of Seizures on Brain Maturation," Clinical Investigator Development Award (\$360,500 for 5 years).

1998 – 2000: Sigma-Tau Pharmaceuticals, Inc., "Carnitine Supplementation in Patients on the Ketogenic Diet for the Treatment of Intractable Epilepsy," (\$93,000).

1999 – 2004: NICHD – NIH – HD38600 "Neuroplasticity of brain asphyxiated Infants : Efficacy of intervention" (5% effort, Dr. Lina Badr, P.I., transferred to Dr. Meena Garg).

2002 – 2004: Cyberonics, Inc., "A Study of Neuropsychiatric and Cognitive Function in Children with Epilepsy Treated with Vagus Nerve Stimulation," Rochelle Caplan, PI (\$60,000)

2003 – 2008: NINDS, NIH –1R01 NS045911 "Childhood Absence Epilepsy: Treatment, Pharmacokinetics-Pharmacodynamics-Pharmacogenetics" PI: Tracy A. Glauser, M.D., (A multicenter project with Raman Sankar, MD, PhD as the PI at UCLA. 5% effort).

2004 – 2008: NINDS, NIH – 1R01 NS046516 "Epileptogenicity in the Developing Brain" (Raman Sankar, PI, 35% effort, \$208,125 Direct Costs for 1st year).

2007 – 2008: Award from Valeant Pharmaceuticals for the "Anticonvulsant Development Program," (Raman Sankar and Andrey Mazarati, Co-PIs, \$70,000).

2007 – 2008: Award from Johnson & Johnson Pharmaceutical Research Institute "Evaluation of the Antiepileptogenic Potential of Carisbamate in the Rapid Kindling Model" (Raman Sankar and Andrey Mazarati, Co-PIs, \$88,500)

2007 – 2009: Marinus Pharmaceuticals, Inc. A double-blind, placebo-controlled dose-ranging clinical study to evaluate the safety, tolerability and antiepileptic activity of ganaxolone in treatment of patients with infantile spasms. (Raman Sankar, PI, \$306,380)

2007 – 2009: Marinus Pharmaceuticals, Inc. An open-label clinical study to evaluate the safety and antiepileptic activity of ganaxolone in the treatment of patients diagnosed with infantile spasms. (Raman Sankar, PI, \$152,967).

2008 – 2010: NINDS, NIH - R21 NS059505 "Validation of high-throughput screening of candidate antiepileptogenic drugs" (Raman Sankar 10% effort; Andrey Mazarati, PI). (Score 128, 4th percentile, effective Feb 15, 2008).

2009 – 2011: NIMH, NIH – R21 MH079933 "Neurobiological Basis of Depression as a Comorbidity in Epilepsy" (Raman Sankar 10% effort; Andrey Mazarati, PI) – (\$247,500 funded, April 20, 2009).

2009 – 2010: Epilepsy Foundation of America, "Hippocampal Neuroinflammation as a Mechanism of Depression in Epilepsy" (Raman Sankar, Co-I, Andrey Mazarati, PI), (\$74,000,funded June 2009,).

2010 – 2014: NINDS, NIH – R01 NS065783 "Mechanisms of Comorbidity between Epilepsy and Depression" (Raman Sankar Co-I; Andrey Mazarati, PI).

2009-2011: Berlin Heart, Inc. "Study of Berlin Left Ventricular Assist Device in Pediatric Patients" (Juan Alejos, PI. My role: Study Neurologist)

2010 – 2011: Pfizer, Inc. A Placebo-controlled, multiple dose study to evaluate the safety, tolerability and pharmacokinetics of pregabalin in pediatric patients with partial onset epilepsy.

2010 – 2012: Pfizer, Inc. A Placebo-controlled, multiple dose study to evaluate the safety and tolerability of flexible doses of pregabalin in pediatric patients with partial onset epilepsy.

2010 – 2012: Epilepsy Foundation of Greater Los Angeles. Award of \$110,000 for the support of the Ketogenic and Other Dietary Therapies program at Mattel Children's Hospital.

2011 – 2013: Epilepsy Foundation of Greater Los Angeles, \$140,000. Julie and Stephen DesJardins Pediatric Epilepsy Fellowship Award – will be used to support Dr. Lekha Rao.

2012 – 2014: Epilepsy Foundation of Greater Los Angeles, \$140,000. James Abrahams – Charlie Foundation Pediatric Epilepsy Fellowship Award – will be used to support Dr. Nicole Cobo.

2011 – 2018: NIH-NINDS, 1U10NS077355-01 (Dobkin, Grill) \$200,000 annual. Investigator in the NEURONEXT clinical trial network formed by the NINDS to facilitate improved and more efficient clinical trials for neurological diseases and disorders. Twenty-five sites make up the network, which will conduct up to seven clinical studies per year over a seven year funding cycle.

2012 – 2015: NIH-NINDS, P20 NS080181 (Engel PI, Sankar Co-I) \$1,406,772 (total, multi center, multi-investigator), “Epilepsy Research Centers without Walls” program.

2014 – 2019: NIN-NINDS, 1 U10NS086497 (Drs. Jeffrey Saver & Gene Sung, PIs; Sankar, Site PI for Pediatrics) Los Angeles Southern California StrokeNet Consortium.

2014 – 2016: Bluebird Bio. “An open label, non-randomized, single dose, multi-center phase 2/3 study of the safety and efficacy of Lenti-D modified autologous stem cells for the treatment of subjects with childhood cerebral adrenoleukodystrophy (CCALD)” . (Ami Shah, PI. My role: Study Neurologist)

2014 – 2015: Upsher-Smith Laboratories. (Sankar Site PI) An Open-Label Safety Study of USL261 (intranasal midazolam) in the Outpatient Treatment of Adolescent and Adult Subjects with Seizure Clusters.

2015 – 2016: Acorda Therapeutics (Sankar, Site PI) An Open-Label, Safety and Tolerability Study of Chronic Intermittent Use of Diazepam Nasal Spray in Adolescents and Adults with Cluster Seizures

2015 – 2016: GW Pharmaceuticals (Co-PI with Dr. Hussain) A Double-blind, Placebo-Controlled, Two-part Study to Investigate the Dose-ranging Safety and Pharmacokinetics, followed by the Efficacy and Safety of Cannabidiol (GWP42003-P) in Children and Young Adults with Dravet Syndrome.

2014 – 2019: NIH-NINDS (Lead Co-PIs: Dr. Samden Lhatoo, Case Western Reserve University & dr. Jeffery Noebels, Baylor College of Medicine). SUDEP Translational Research Alliance (SUTRA). (Centers without Walls for Collaborative Research in the Epilepsies: Sudden Unexpected Death in Epilepsy (SUDEP) – The Center for SUDEP Research).

NS090407 Autonomic and Imaging Biomarkers of SUDEP, Sutra 3 of 7

PIs, Samden Lhatoo, Beate Diehl, Ronald Harper (Professor Ronald Harper, UCLA PI, Raman Sankar, co-investigator for pediatric patients at UCLA, 5% effort).

DoD W81XWH-18-1-0612 Galanopoulou (PI) 09/01/2018-08/31/2020
Vulnerabilities, Risks and Interplay of Post-Traumatic Depression and Epilepsy: Role of Monoaminergic Circuits
Role: Investigator

SK Life Science, Inc/Premier Research: Phase I, Open-Label Study of Carisbamate in Adult and Pediatric Subjects with Lennox-Gastaut Syndrome. Protocol # YKP509C001
Raman Sankar, Site PI. 09/2019 – 12/2020

SK Life Science, Inc/Premier Research: Phase I, Open-Label Study of Carisbamate in Adult and Pediatric Subjects with Lennox-Gastaut Syndrome. Protocol # YKP509C002 – Extension Phase
Raman Sankar, Site PI. 09/2019 – 12/2020

Sponsor: Marinus Pharmaceuticals, A double-blind, randomized, placebo-controlled trial of adjunctive ganaxolone treatment in female children with protocadherin 19 (PCDH19)-related epilepsy followed by long-term open-label treatment - Violet Study
Role Sub-Investigator 09/2019 – 12/2020

Sponsor: Marinus Pharmaceuticals, A Double-blind, Randomized, Placebo-controlled Trial of Adjunctive Ganaxolone Treatment in Children and Young Adults with Cyclin-dependent Kinase-like 5 (CDKL5) Deficiency Disorder (CDD) Followed by Long-term Open-label Treatment - Marigold Study
Role Sub-Investigator 09/2019 – 12/2020

Sponsor: Eisai Pharmaceuticals, Multicenter, Double-Blind, Randomized, Placebo-Controlled, Parallel-Group Study with Open-Label Extension Phase of Lorcaserin as Adjunctive Treatment in Subjects with Dravet Syndrome.

Role: Site PI	02/2021 – 06/2023
DoD CDRMP IDA ERP EP180003 Gut Microbiome and Post-Traumatic Epilepsy: Biomarker and Mechanistic Implications Role: Senior Investigator	Mazarati (PI) 09/01/2019-08/31/2022
DoD RPA EP210022 Leveraging innate immunity to prevent post-traumatic epilepsy Role: Co-investigator (0.5 calendar month)	Mazarati & Galanopoulou (PIs) 07/01/2022-06/30/2024
DoD RPA EP210041 Modeling the impact of trauma experience upon post traumatic outcomes and epilepsy Role: Co-investigator (0.5 calendar month)	Mazarati (PI) 07/01/2022-06/30/2024
NINDS-NIH R01 NS127524-01 Translational platform for epilepsy therapy and biomarker discovery Role: Co-investigator (1.0 calendar month)	Mazarati (PI/multi-PI grant) 04/01/2022-03/31/2027

CLINICAL / TEACHING ACTIVITIES:

Attending Neurologist, UCLA Children's Hospital, UCLA Medical Center Hospital and Clinics.
Consult- and in-patient services; Epilepsy / Long-Term Monitoring.

Attending Neurologist, Multidisciplinary Clinic for Tuberous Sclerosis, run by the Division of Genetics and the Neuropsychiatric Institute, UCLA Medical Center (1994 - 1996).

Consulting Neurologist, Kern Medical Center, Bakersfield, CA (1989 – 2012).

Consulting Neurologist, Bakersfield Family Medical Center (1997 – 99).

Consulting Neurologist, Venice Family Clinic, Venice, CA (volunteer service, 1990 - 2000).

Preceptor for Child Psychiatry fellows at Kern Medical Center since inception of that training program in 2009.

OTHER EDUCATIONAL AND MENTORING ACTIVITIES:

1975 - 1984	Regular contributor to American Council on Pharmaceutical Education (ACPE) approved continuing education programs to practicing pharmacists sponsored by Xavier University, New Orleans, Louisiana.
1989 - Present	Contributor to educational programs in the UCLA Graduate School of Nursing and nursing educational programs sponsored by the Pediatric Critical Care Nursing Division at UCLA Center for the Health Sciences.
1991 - Present	Regular lecturer to residents in the Departments of Neurology and the Department of Pediatrics at Kaiser Permanente Medical Center.
1992 - Present	Participation in the undergraduate Student Research Program.
1993 - Present	Pediatric Resident Mentor Program (Drs D. Balke, L. Tseng-Ong, V. Cheung, S. Spence, K. Maher, R. Chong)

1994 - 97	Member, Doctoral Dissertation Committee of Kerry Thompson, Candidate for PhD in the UCLA Neuroscience Program.
1996 – 2007	Faculty, Pediatric Board Review Course, UCLA Department of Pediatrics and Mattel Children's Hospital at UCLA.
1996, 97 Summers	Participating faculty in Pediatric Board Review courses by the Osler Institute.
1998, 99 January	Faculty in Osler Review for Neurology Boards, ABPN Part II.
2003 – 2004	Contributing Faculty, UCLA School of Dentistry Resident Fellow Program, DS 30701
2003 - 2006	Member, Doctoral Dissertation Committee of Gene Gurkoff, Candidate for PhD in the UCLA Neuroscience Program.
2006 – 2008	Member, Doctoral Dissertation Committee of Stéphane Auvin at the University of Lille Medical School, Lille, France.
2004 - 2006	Member, Scholarship Oversight Committee, Camille Fong, MD, Neonatology fellow.
2007 – 2008	Preceptor to visiting scientist, Dr. Young-Se Kwon, Inha University, Incheon, South Korea.
2007 – 2009	Research mentor and Member, Scholarship Oversight Committee, Edward Evans, MD, Neonatology fellow.
2008 - present	Preceptor to post-doctoral fellow, Eduardo Pineda, PhD.
2010 – 2011	Doctoral dissertation committee, Madhuri Sudan, PhD candidate in the School of Public Health.
2013 – 2014	Research co-mentor (with Dr. Mazarati) and Member, Scholarship Oversight Committee, James Washington, MD, Neonatology fellow.
2013 – 2015	Research co-mentor (with Dr. Mazarati) to Udaya Kumar, PhD, post-doctoral fellow in the Developmental Epilepsy Laboratories.
2014 – 2015	Research co-mentor (with Dr. Mazarati) to Dana Shprung.
2014 – 2015	Research co-mentor (with Dr. Mazarati) to Dr. Eunice Hagen.
2014 – 2016	Research co-mentor (with Dr. Mazarati) to Dr. Elena Minakova.
2018 - 2020	Research Mentor to Dr. Kathryn Sims

MEMBERSHIPS IN PROFESSIONAL SOCIETIES:

Society for Neuroscience (Member since 1979)
 American Academy of Neurology (Member since 1988, Elected Fellow, 2004)
 American Epilepsy Society (Member since 1991)
 Child Neurology Society (Member since 1992)
 American Pediatric Society (Elected 2013)
 International Child Neurology Association

SERVICE TO EDITORIAL BOARDS, PROFESSIONAL ORGANIZATIONS

Editorial Board, *Epilepsia*, 2007 – 2010.

Editorial Board, *Epilepsy & Behavior*, 2011 – 2019.

Editorial Board, *Journal of Pediatric Epilepsy*, 2015 – ongoing.

Contributing Editor, *Epilepsy Currents*, appointed in 2001-2005 (Blackwell Publishing, on behalf of the American Epilepsy Society)

Consulting Editor, Neurosciences, *Pediatric Research* (2004-2005)

Member, Editorial Board, *Contemporary Neurology* (MIT Press, Peer-reviewed, Electronic)

Member, Editorial Board, *Frontiers in Bioscience* (Peer-reviewed, Electronic)

Ad hoc reviewer for:

1. Acta Neurologica Scandinavica
2. Annals of Neurology
3. Biochemical and Molecular Medicine
4. Brain Research
5. Cellular and Molecular Life Sciences
6. Epilepsia
7. Epilepsy Research
8. Epileptic Disorders
9. Journal of Cerebral Blood Flow and Metabolism
10. Journal of Neurochemistry
11. Journal of Neuroscience
12. Journal of Pediatrics
13. Journal of Pediatric Research
14. Lancet Neurology
15. Life Sciences
16. Mental Retardation and Developmental Disabilities Research Reviews
17. Molecular Brain Research
18. Molecular Genetics and Metabolism
19. Nature Neurology
20. Neurobiology of Disease
21. Neurology
22. Neuroscience
23. Neuroscience Letters
24. Pharmacology, Biochemistry, and Behavior
25. Journal of Pediatric Psychology

Ad hoc grant reviewer for the Medical Research Council of New Zealand (1996).

Chaired a platform session (Session 661) at the 27th Annual Meeting of the Society for Neuroscience, October 25-30, 1997, New Orleans: Epilepsy: Basic Mechanisms I

Co-chaired a platform session (Session L) at the Annual Meeting of the American Epilepsy Society, December 5-10, 1997, Boston: Behavior; Neuropsychology; Language and Aphasia

Co-chaired a platform session at the 50th Annual Meeting of the American Academy of Neurology, April 25 – May 2, 1998, Minneapolis: Antiepileptic Drugs:

Member, Research Review Board, Alternating Hemiplegia Foundation

Selected to represent pediatric neurology in the Medical Expert Panel, ABC News (1999 -)

Reviewed Neuroscience Abstracts for the Western Regional Meeting (Feb 2000) of the American Federation for Medical Research

Ad hoc consultant to Ortho-McNeil, Parke-Davis, Cyberonics, Ortho-McNeil, Novartis, Xcel, Élan, UCB, and Valeant Pharmaceuticals.

Member, Awards Committee, Epilepsy Section, American Academy of Neurology (1999 -)

Member, Committee on Continuing Education, American Epilepsy Society (1999 -)

Member, Research Committee, Child Neurology Society (2000 - 2005)

Member, Professional Advisory Board, Epilepsy Foundation (2000 - present)

Ad hoc grant reviewer for the Wellcome Trust, 183 Euston Road, London NW1 2BE, UK (2000)

Ad hoc grant reviewer for the Veterans Administration Merit Review Grants (2001)

Research Grants Review for the Epilepsy Foundation, Professional Advisory Board Meeting, Las Vegas, October 26, 27 (2001)

Member, Annual Meeting Committee, American Epilepsy Society (2001 - 2002)

Grants and Fellowships Committee, Epilepsy Foundation PAB, Washington D.C., October 18,19 (2002).

Examiner, American Board of Psychiatry and Neurology, Part II (orals), May 2002.

SRB Study Section for NINDS, NIH, Special Emphasis Panel: ZNS1 SRB-W (07) (April 2004)

Selected as participant in an NINDS, NIH Workshop on "Animal Models of the Childhood Epilepsies," May 13-14, 2004.

NINDS, NIH, Special Emphasis Panel, ZNS1 SRB - G(03), Mechanisms and Treatment of Epilepsy, November 2005.

NINDS, NIH Study Section, Neurological Sciences and Disorders – C (NSDC), *ad hoc*, September 2005, February 2006.

NINDS, NIH, Fellowship Review Committee ZNS1 SRB-M, April, 2006.

NINDS, NIH, Loan Repayment Program Application Review ZNS1 SRB-M 35, April, 2006.

Examiner, American Board of Psychiatry and Neurology, Part II (orals), September 10-12, 2004.

Tuberous Sclerosis Complex Investigator's Meeting, New York, New York, November 19-21, 2004.

Examiner, American Board of Psychiatry and Neurology, Part II (orals), January 14-16, 2005.

Reviewer for Research Grants submitted to the Child Neurology Foundation, 2005.

Scientific Review Board, **CURE** (Citizens United for Research in Epilepsy), 2003, 2004, 2005.

Scientific Review Board, American Epilepsy Society, Early Career Physician Scientist Award, 2005-2008.

Appointed to the Research Council of the Professional Advisory Board of the Epilepsy Foundation, April, 2006.

NINDS, NIH Study Section, Neurological Sciences and Disorders – C (NSDC), Appointment 2006, term expiring in June 2010.

Epilepsy Foundation Youth Research Initiative – work group member, appointed Fall 2006.
Committee on Special Interest Discussion Groups, American Epilepsy Society, and Chair, SIG on Basic Science, 2006.

Steering Committee for Regional Education Programs, American Epilepsy Society, 2006

Organizing Committee (Committee of the International League Against Epilepsy) for the Workshop on the Neurobiology of Epilepsy 2007 in Langkawi, Malaysia, satellite to the International Epilepsy Congress in Singapore, 2007.

Committee on Special Interest Discussion Groups, American Epilepsy Society, and Chair, SIG on Basic Science, 2007.

Elected member of the Epilepsy Research Foundation Board of Directors as an Epilepsy Foundation representative, 2008.

Pediatric Topics Selection Committee for the Annual Meeting of the American Epilepsy Society, 2008 – present.

Committee on Special Interest Discussion Groups, American Epilepsy Society, and Chair, SIG on Basic Science, 2008.

Scientific Organizing Committee (Committee of the International League Against Epilepsy) for the Workshop on the Neurobiology of Epilepsy 2009 (June 23-26) in Pécs, Hungary, satellite to the International Epilepsy Congress June 26-30, Budapest, Hungary.

Appointed to the Commission on Neurobiology of the International League Against Epilepsy (2009 – 2013).

Chair, Scientific Review Committee, NINDS, NIH, Panel ZNS1 SRB-B 24 for the Epilepsy EUREKA Program, November 8, 2010.

Appointed Chair, Special Interest Group Oversight Committee, American Epilepsy Society, 2011-14.

Member, Scientific Organizing Committee (Committee of the International League Against Epilepsy) for the Workshop on the Neurobiology of Epilepsy 2011 in Grottaferrata, Italy, August 23-26, satellite to the International Epilepsy Congress, August 26-31, 2011, Rome, Italy.

Participant, Council on Education, American Epilepsy Society, September 19, 2011.

Member of the Research Grant Review Panel, Epilepsy Foundation, October, 2011.

Chair, Scientific Review Committee, NINDS, NIH, Panel ZNS1 SRB-B (32) Epilepsy EUREKA Program, December, 2010.

Member, Scientific Review Committee, NINDS, NIH, Panel ZNS1 SRB-B (35) Epilepsy EUREKA Program, December, 2011.

Member, Scientific Organizing Committee (Committee of the International League Against Epilepsy) for the Workshop on the Neurobiology of Epilepsy 2013 in Lake Dupuis, Quebec, Canada, June 19-22, 2013, satellite to the International Epilepsy Congress, June 22-27, Montreal, Canada.

Member, Scientific Review Committee, NINDS, NIH, Special Emphasis Panel/Scientific Review Group 201401 ZNS1 SRB-G (71), NSD-K SEP, November, 2013.

Grant Review for *Fédération pour la Recherche sur le Cerveau*, (Federation for Brain Research – FRC), Paris, France, November, 2014.

Member, Scientific Organizing Committee (Committee of the International League Against Epilepsy) for the Workshop on the Neurobiology of Epilepsy 2015 in Heybeliada Island, Turkey, August 31 – September 4, satellite to the International Epilepsy Congress, Sept 4-10, 2015, Istanbul, Turkey.

Member, Scientific Review Committee, NINDS, NIH, Blueprint Neurotherapeutics – ZNS1 SRB-T (07), November, 2015.

Member, Self-Assessment for the Maintenance of Certification Committee of the American Epilepsy Society (appointed April 2016)

Grant reviewer (ad hoc) for the Thrasher Foundation for Pediatric Research (April 2016)

Member, Organizing Committee for the *9th International Epilepsy Colloquium on Surgical and Targeted Treatments for Acquired Lesions* at the Church House Conference Centre in Westminster, London on 22 - 24 June, 2016.

Member, Scientific Organizing Committee (Committee of the International League Against Epilepsy) for the Workshop on the Neurobiology of Epilepsy 2017 in Mon St Benet, Barcelona, Spain, August 28-Sept 1, satellite to the International Epilepsy Congress, Sept 1-5, 2019, Barcelona, Spain.

Member, Scientific Review Committee, NINDS, NIH, NST Review Group, February 2018, June 2018.

Scientific Organizing Committee (Committee of the International League Against Epilepsy) for the *Workshop on the Neurobiology of Epilepsy 2019* in Ayuthaya, Thailand, June 16-20, satellite to the International Epilepsy Congress, June 20-25, 2019, Bangkok, Thailand.

Member, Scientific Review Committee, NINDS, NIH, Special Emphasis Panel/Scientific Review Group 2020/01 ZNS1 SRB-G (27) (SIREN Program), December 20, 2019.

Reviewer for grant application to Science Foundation Ireland, January 2020.

Member, Scientific Review Committee, NINDS, NIH, Special Emphasis Panel, 2021/10 ZNS1 SRB-T (48) Blueprint Neurotherapeutics Network Small Molecule Drug Discovery for Disorders of the Nervous System, June 18, 2021.

Member, Scientific Program Committee, Child Neurology Society (December 2019 – December 2022)

Member and Special Advisor to the Commission on Neurobiology of the *International League Against Epilepsy (ILAE)*, Appointment: 2021-2025.

Member, Epilepsy Therapies Symposium Committee, *American Epilepsy Society* (January 1, 2022 – December 31, 2024).

Member, Scientific Review Committee, NINDS, ZNS1 SRB-T (53), Blueprint Neurotherapeutics Network Small Molecule Drug Discovery for Neurological Disorders, 06/10/2022.

Member, Scientific Organizing Committee (Committee of the International League Against Epilepsy) for the *Workshop on the Neurobiology of Epilepsy 2022* (July 4-8) in Talloires, France, satellite to the European Epilepsy Congress (July 8-12) in Geneva, Switzerland.

Member, Scientific Review Committee, NINDS, ZNS1 SRB-Q(07), Blueprint Neurotherapeutics Network Small Molecule Drug Discovery for Neurological Disorders, 06/14-15/2023.

UNIVERSITY / DEPARTMENTAL SERVICE

Faculty Advisor to medical school freshmen (1994 -1995).

Presentations to the Pharmacy and Therapeutics Committee on nomination of new anticonvulsants to the hospital formulary (1994: Felbamate; 1995: Lamotrigine).

Pharmacy and Therapeutics Committee, UCLA Medical Center (1994 - present).

Ad hoc Committee for the Development of Filmless Radiology (Department of Pediatrics (1994-95).

Ad hoc Committee for Evaluating the Role of Information Systems in Research Support (Department of Pediatrics, 1997).

Education Liaison Committee, Department of Pediatrics (1996).

Information Technology Task Force - Strategic Planning, UCLA Medical Enterprise (1997).

Member: World Wide Web Advisory Committee, Department of Pediatrics (1998 -)

Ad hoc Rehabilitation and Subacute/Long-term Care Advisory Committee for the UCLA Healthcare Network (1998)

Department of Pediatrics Grand Rounds Committee (1999- present)

In-patient Charge Document Committee (1999 - 2000)

Grant Reviewer: UCLA AIDS Institute.

Ad hoc Review Committee of the Council on Academic Personnel (CAP), January 2001, May 2002.

Mentor to Junior Faculty: John Stern, MD, Department of Neurology; Mini Mehra, MD, Department of Pediatrics (both beginning in 2001)

General Clinical Research Center Advisory Board, October 2002 – present

Residency Training Committee, Department of Neurology, 2002 – 2005

Department of Pediatrics Group Practice (DPGP) Executive Board, 2004 - 2008.

Review & Appraisals Committee, Department of pediatrics, 2005 – 2016.

Neurology Residency Selection Committee, 2009 – 2010.

Departmental representative to the Legislative Assembly of the University Senate, appointed 2014.

Member, Pediatric Neurosurgeon Search Committee, UCLA, 2015-16.

Member, Neurology Chair Search Committee, UCLA, 2015-16.

Member, Neurosurgery Chair Search Committee, UCLA, 2017.

Member, Committee to Evaluate candidates for Endowed Chairs in Developmental & Behavioral Pediatrics, Fall 2019.

COMMUNITY SERVICE

Consulting pediatric neurologist for the Venice Family Clinic, 1991 – 1999.

Member, Board of Directors, Epilepsy Foundation of Los Angeles, Orange, San Bernardino, and Ventura Counties, Elected 1995.

Member, Professional Advisory Board, Epilepsy Foundation of Greater Los Angeles (2003-present)

Member, Professional Advisory Board, Epilepsy Foundation of America (2005-presentt)

Guest speaker at the Family Weekend Camp organized by the Epilepsy Foundation of Los Angeles, Orange, San Bernardino, and Ventura Counties. Fall 1993 - 2000.

Participant / Speaker at the UCLA symposium "Treatment of Epilepsy Today". Held at the UCLA Conference Center on November 20, 1993.

Speaker at the Medical Education Conference for patients and their families, sponsored by the National Tuberous Sclerosis Association: "Neurological Aspects of Tuberous Sclerosis," Los Angeles, June 11, 1994.

Speaker at the UCLA Conference "Treating Epilepsy in 1995: A Seminar for Patients," UCLA Conference Center on April 29, 1995.

Speaker at the Medical Education Conference for patients and their families, sponsored by the National Tuberous Sclerosis Association: "New Treatment Options for Seizure Disorders," Long Beach, October 21, 1995.

Speaker at a program sponsored by "Parents Helping Parents" on the Ketogenic Diet: "Role of Carnitine Supplementation in the Ketogenic Diet," San Jose, May 17, 1997.

Speaker at the Medical Education Conference for patients and their families, sponsored by the National Tuberous Sclerosis Association: "New anticonvulsants for patients with tuberous sclerosis." Los Angeles, November 21, 1998.

Speaker at the Epilepsy Education Conference sponsored by the Epilepsy Foundation: "The Ups and Downs of Epilepsy Treatments for Children," at Anaheim, March 27, 1999.

Speaker at the Epilepsy Education Conference for families: Vagus Nerve Stimulation in Medically Refractory Epilepsies of Childhood," Santa Monica, May 26, 1999.

Various national media interviews on epilepsy such as National Public Radio (1994), Home Show (ABC TV, 1993), Beyond Belief (CBS TV, Dec 1994), local cable channel dedicated to the Asian community (KSCI, Ch. 18, Los Angeles, 1994), Evening News (Fox TV, Ch. 11, Los Angeles (March 11, 1996), The Learning Channel (National Cable Channel, March 2, 1997).

Speaker at the Program for Families with Epilepsy sponsored by the Epilepsy Foundation, September 7, 2002.

Volunteer and speaker at *Family Weekend Camp*, Epilepsy Foundation, September 21, 2002.

Teleconference for Parents, sponsored by the Epilepsy Foundation, November 9, 2002.

Volunteer and speaker at *Family Weekend Camp*, Epilepsy Foundation, September 20, 2003.

Speaker at the *Epilepsy Brain Storm Summit 2003*, An All-day Program for Families, Marriage and Family Therapists, Licensed social Workers, Nurses, and Nursing Home Administrators sponsored by the Epilepsy Foundation, held at the Loyola Marymount University Campus, November 8, 2003.

Speaker at a family support group meeting of the *Tuberous Sclerosis Alliance*, January 24, 2004.

Speaker at the *Epilepsy Brain Storm Summit 2004*, *Epilepsy Foundation of Los Angeles*, Cathedral of Our Lady of Angels, November 12, 2004, Los Angeles, California.

Participant in the *Tuberous Sclerosis Alliance's* West Coast Regional TSC Conference, February 19-20, 2005 in Riverside, California.

Speaker and Small Group Discussant at "Not Another Moment Lost ... to Childhood Epilepsies," a conference for physicians and families of patients organized by the *Epilepsy Foundation of Los Angeles*, April 2-3, 2005 in Lakewood, California.

Session Co-Chair on the Low-glycemic Index Diet for the Treatment of Epilepsy at the *Epilepsy Brain Storm Summit 2005*, *Epilepsy Foundation of Los Angeles*, Cathedral of Our Lady of Angels, November 12, 2005, Los Angeles, California.

Invited speaker, *Annual Meeting of the Tuberous Sclerosis Alliance*, July 12-15, 2006, Chicago, Illinois.

Speaker, Family weekend Camp of the *Epilepsy Foundation of Greater Los Angeles*, Sept 16, 2006, Topanga, California.

"Epilepsy: Diagnosis, Treatment, and Future Options" to the *Central Coast Epilepsy Network*, Oct 9, 2006, Templeton, California.

Presenter and consultant, *2007 Sturge-Weber Foundation International Conference*, July 12-15, Anaheim, California.

Member, Board of Directors, *Epilepsy Partnership India Trust* (EPI Trust).

Speaker, Family weekend Camp of the *Epilepsy Foundation of Greater Los Angeles*, Sept 15, 2007, Topanga, California.

Round Table on "Epilepsy during Infancy" at the *Epilepsy Brain Storm Summit* of the *Epilepsy Foundation*, November 15, 2008, Los Angeles, California.

"New Medications for Epilepsy," at the *Epilepsy Brain Storm Summit* of the *Epilepsy Foundation*, November 14, 2009, Los Angeles, California.

"From the Newly Diagnosed to the Refractory Patient: Current Concepts in Rationalization of AED Therapy" and "The New Kids on the Block: Understanding the New AEDs," presentations at the Long Distance Epilepsy Education and Awareness Program (LEAP), a Multi Center Epilepsy Symposium organized by the *Epilepsy Partnership India (EPI) Trust*, January 31, 2010, Mumbai, India.

"Ketogenic and Other Dietary Therapies for Epilepsy," at the *Epilepsy Brain Storm Summit* of the *Epilepsy Foundation*, November 6, 2010, Los Angeles, California.

Expert testimony at the California Senate Subcommittees in support of SB. 161 to support seizure rescue therapy in schools. Subcommittee on Education (March 16, 2011), Subcommittee on Health (April 6, 2011), Sacramento, California. (http://www.chinohills.com/news-articles-details/Huff_Legislation_Overcomes_Union_Opposition-1893).

Radio interviews on Senate Bill 161 (State of California) on the Andy Caldwell Show with Mr. Joe Armendariz on September 16, 2011 (Simulcast on KZSB 1290, Santa Barbara and KUHL 1440, Santa Maria)

Speaker at the Epilepsy Parents' Forum organized by the *Epilepsy Foundation of Greater Los Angeles* at the Annenberg Community Beach House, January 5, 2013, Santa Monica, California.

Speaker at the *Epilepsy Foundation of Greater Los Angeles* Family Day Camp at Camp Tumbleweed, September 7, 2013, Brentwood, California.

Appearance on Univision Spanish TV News on cannabis products in severe childhood epilepsies: <http://noticias.univision.com/video/393035/2014-02-24/primer-impacto/salud/tratamiento-epilepsia-con-marihuana?hash=9cfbb82b1ce00faa8f7cee39d8652045> February 24, 2014.

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"Medical issues After Hemispherectomy: Headaches & Migraines," at the *Annual Meeting of the Hemispherectomy Foundation and the Brain Recovery Project*, July 12, 2014, Anaheim, CA.

Multiple presentations and role as co-organizer in the "Epilepsy Summit 2015" by the *Epilepsy Foundation of Greater Los Angeles*, March 26-28, 2015, Manhattan Beach, CA.

Presentation at the End Epilepsy Workshop and Social Event, organized by the *Epilepsy Foundation of Greater Los Angeles*, May 7, 2016, at the Sunset canyon Recreational Center, UCLA Campus.

Presentation on Pediatric Epilepsy Surgery at the "End Epilepsy Summit 2017" by the *Epilepsy Foundation of Greater Los Angeles*, April 28, 2017, Universal City, Los Angeles, CA.

"Emerging Therapies for SCN1A (Dravet syndrome) Disorders," at the *Day of Dravet: Los Angeles*, organized by the Dravet Syndrome Foundation, September 14, 2019, Los Angeles, California.

Program Moderator for "*CURE Day of Science: Epilepsy Conversations*," at the Children's Hospital of Orange County, organized by *Citizens United for Research in Epilepsy* (CURE), September 21, 2019, Orange, California.

Speaker at Epilepsy Awareness Day at Disneyland (EADDL) on "Your Child's EEG," November 4, 2019, Anaheim, California.

Medication-Resistant Epilepsy – Epilepsy Expert Series on Zoom, Epilepsy Support Network of Orange County, March 12, 2021.

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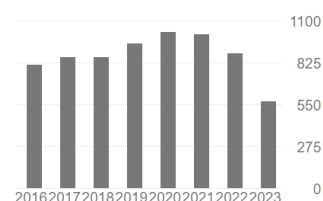
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144. Phillips D, Arndt D, Michon A, Minarik S, **Sankar R**. Dravet Syndrome Associated with a Novel GABRG2 Variant (R323W) Treated With Clobazam. Annual Meeting of the American Epilepsy Society, Abstr. 3.160, 2016, Houston, Texas.
145. Bernardo D, Nariai H, Hussain SA, **Sankar R**, Wu JY. Interictal scalp fast ripples: an epileptogenic marker by visual and semi-automatic detection. Annual Meeting of the American Epilepsy Society, Abstr. 1.108, 2017, Washington DC.
146. Nariai H, Bernardo D, **Sankar R**, Fallah A, Wu JY, Hussain SA. Inter-rater reliability in visual identification of high frequency oscillations on electrocorticography and scalp EEG. Annual Meeting of the American Epilepsy Society, Abstr. 1.148, 2017, Washington DC.
147. Bajammal R, Nariai H, Numis AL, Rajaraman RR, **Sankar R**, Hussain SA. Ideal duration of video-EEG to confirm response to therapy in the treatment of infantile spasms. Annual Meeting of the American Epilepsy Society, Abstr. 2.160, 2017, Washington DC.
148. Eliyan Y, Bhatt R, **Sankar R**, Hussain SA. Amantadine treatment for children with Electrical Status Epilepticus in Sleep (ESES). Annual Meeting of the American Epilepsy Society, Abstr. 2.188, 2017, Washington DC.
149. Rajaraman RR, **Sankar R**, Hussain SA. The Successful Use of Pure Cannabidiol for Treatment of Super-Refractory Status Epilepticus. Annual Meeting of the American Epilepsy Society, Abstr. 2.189, 2017, Washington DC.
150. Elsanadi R, Bajammal R, Murata K, **Sankar R**, Hussain SA. Efficacy of vagus nerve stimulation for treatment of epileptic spasms. Annual Meeting of the American Epilepsy Society, Abstr. 2.190, 2017, Washington DC.
151. Vaisleib I, Duchowny M, **Sankar R**, Grinnell T, Vieira M, Blum DE. Analysis of sodium levels and hyponatremia events in clinical trials of eslicarbazepine acetate in pediatric patients (aged 4–17 years) Annual Meeting of the American Epilepsy Society, Abstr. 2.278, 2017, Washington DC.
152. Hussain SA, Dlugos DJ, Cilio MR, Parikh N, **Sankar R**. A phase-2 study of pharmaceutical grade synthetic cannabinoid oral solution for the treatment of refractory infantile spasms. Annual Meeting of the American Epilepsy Society, Abstr. 3.278, 2017, Washington DC.
153. Mazarati A, Medel-Matus J, Santana-Gomez C, Shin D, Staba R, **Sankar R**. Kindling epileptogenesis in rats is modified by microbiome allotransplant from donors that have undergone brain fluid percussion injury. American Epilepsy Society, Abstr. 1.001, 2018, New Orleans, LA.
154. Bernardo D, Nariai H, Hussain S, **Sankar R**, Wu J. Investigation of phase amplitude coupling of interictal scalp HFOs in children with epileptic spasms. American Epilepsy Society, Abstr. 2.098, 2018, New Orleans, LA.
155. Yoo J, Garg M, **Sankar R**, Rao L. Incidence of later onset seizures in term babies who underwent therapeutic hypothermia for hypoxic-ischemic encephalopathy. American Epilepsy Society, Abstr. 2.159, 2018, New Orleans, LA.
156. Nariai H, Cao D, Bernardo D, Sankar R, Hussain S, Wu J. Prevalence of interictal scalp EEG high-frequency oscillations in pediatric epilepsy monitoring unit. American Epilepsy Society, Abstr.3.169, 2018, New Orleans, LA.
157. Truty R, Patil N, **Sankar R**, Sullivan J, Millichap J, Carvill G, Entezam A, Esplin ED, Fuller A, Hogue M, Johnson B, Khouzam A, Kobayashi Y, Lewis R, Nykamp K, Riethmaier D, Westbrook J, Zeman

- M, Nussbaum RL, Aradhya S. Possible precision medicine implications in a large cohort with childhood epilepsy. *American Epilepsy Society*, Abstr., 2019, Baltimore, MD.
158. Mazarati AM, Medel-Matus J-S, Shin D, **Sankar R**. Kindling of limbic seizures after lateral fluid percussion injury. *American Epilepsy Society*, Abstr. 1.439, 2019, Baltimore, MD.
 159. Bernardo D, Tung K, Nariai H, **Sankar R**, Scalzo F, Hussain S. Predicting short-term outcome in infantile spasms with machine learning. *American Epilepsy Society*, Abstr. 2.077, 2019, Baltimore, MD.
 160. Nariai H, Hussain SA, Bernardo D, Motoi H, Sonoda M, Asano E, Bragin A, Staba R, Hu H, Elashoff D, **Sankar R**, Wu JY. Automated analysis of scalp EEG high frequency oscillations and cross-frequency coupling in children with infantile spasms. *American Epilepsy Society*, Abstr. 2.076, 2019, Baltimore, MD.
 161. **Sankar R**, Kirkham F, Holmes GL, Pina-Garza JE, Wheless J, Grinnell T, Cantu D, Gama H, Moreira J, Tosiello R, Blum D. Long-term safety and tolerability of adjunctive eslicarbazepine acetate in pediatric patients (aged 4–17 years) with focal seizures. *American Epilepsy Society*, Abstr. 1.309, 2019, Baltimore, MD.
 162. Heesch J, Weng J, Rajaraman RR, Nadkarni D, Numis AL, **Sankar R**, Hussain S. Risk of epileptic spasms induction by sodium channel blockade. *American Epilepsy Society*, Abstr. 2.234, 2019, Baltimore, MD.
 163. Nadkarni D, Hussain SA, Sharifi-Hannauer P, **Sankar R**, Reyes BL, Nariai H. Predictive value of overnight continuous EEG monitoring in determining epilepsy risk in children with autism spectrum disorder. *American Epilepsy Society*, Abstr. 1.29, 2019, Baltimore, MD.
 164. Katsarou A, Medel JS, Shin D, Liu WD, Li Q, Gnanapackiam DM, **Sankar R**, Mazarati A, Moshé S, Galanopoulou A. Depressive-like impairments in a rat model of post-traumatic epilepsy. *American Epilepsy Society*, Abstr.3.445, 2019, Baltimore, MD.
 165. Mazarati A, Medel-Matus J-S, Lagishetty V, Santana-Gomez C, Shin D, Jacobs J, Staba R, **Sankar R**. Traumatic Brain Injury, Perturbations in Gut Microbiome and Posttraumatic Epilepsy. *American Epilepsy Society*, Abstr. 1.399, 2021, Chicago, IL.
 166. Nariai H, Lu Q, Monsoor T, Hussain S, Qiao J, Salamon N, Fallah A, Sim MS, Asano E, **Sankar R**, Staba R, Engel JJr, Speier W, Roychowdhury V. Refining Epileptogenic High-frequency Oscillations Using Deep Learning: A Reverse Engineering Approach. *American Epilepsy Society*, Abstr. 2.059, 2021, Chicago, IL.
 167. Murata K, Fallah A, Nariai H, Hussain S, Elashoff D, Brook J, **Sankar R**, Engel JJr, Wu, J. Utilization of High-Frequency Oscillations in Pediatric Epilepsy Surgery: A Prospective, Double-blinded, Randomized Controlled Pilot Study. *American Epilepsy Society*, Abstr. 2.262, 2021, Chicago, IL.
 168. Edmonds B, Miyakoshi M, Ahn S, Phillips HW, **Sankar R**, Fallah A, Matsumoto J, Nariai H. Characteristics of Ictal Thalamic EEG in Pediatric-onset Neocortical Focal Epilepsy. *American Epilepsy Society*, Abstr. 1.161, 2022, Nashville, TN.
 169. Ngo J, Zhang Y, Chung H, Monsoor T, Hussain S, Matsumoto J, Walshaw P, Fallah A, Sim MS, **Sankar R**, Staba R, Engel J, Speier W, Roychowdhury V, Nariai H. Characterization of Physiological High-frequency Oscillations Obtained from Chronic Intracranial EEG in Children Using Deep Learning. *American Epilepsy Society*, Abstr. 1.192, 2022, Nashville, TN.
 170. Ahn S, Rajaraman R, Rao L, Hussain S, Lerner J, **Sankar R**, Matsumoto J, Fallah A, Nariai H. Bithalamic Responsive Neurostimulation for Pediatric-onset Medication-resistant Epilepsy. *American Epilepsy Society*, Abstr. 2.047, 2022, Nashville, T

INVITED PRESENTATIONS:**International:**

1. "Why are Children not just Small Adults?" presented at the *International Conference on Clinical Issues in Epilepsy*, June 11, 1993, Los Angeles, California.
2. Session Moderator, "Where We're Going: The Future for Antiepileptic Drugs," at the symposium *Managing Seizures in Children: Designer Drugs or the Cutting Edge?* March 17-18, 1994, Palm Desert, California.
3. "Presurgical Evaluation: Choosing the Right Candidate," at the symposium *Managing Seizures in Children: Designer Drugs or the Cutting Edge?* March 17-18, 1994, Palm Desert, California.
4. "Structural and Functional Imaging in Defining Subtle Cortical Abnormalities," at *New Developments in Pediatric Epilepsy Surgery*, a satellite symposium of the joint *Child Neurology Society / International Child Neurology Association* meeting, September 30, 1994, Santa Monica, California.
4. Afternoon session moderator, *New Developments in Pediatric Epilepsy Surgery*, a satellite symposium of the joint *Child Neurology Society / International Child Neurology Association* meeting, September 30, 1994, Santa Monica, California.
5. "Differential effects of status epilepticus (SE) on GABA synthesis in the rat cortex and hippocampus during development," Focus on Epilepsy III, GABA and Epileptogenesis, an International Conference, Whistler, British Columbia, Canada, May 13-16, 1995.
6. "Ontogeny of Status Epilepticus-Associated Brain Damage," at *Status Epilepticus - Mechanisms and Management, An International Symposium*, February 6-8, 1997, Santa Monica, California.
7. "Ontogeny of Seizure-Associated Brain Injury," at *Childhood Epilepsy and Brain Development, An International Conference*, April 2-5, 1997, Strasbourg-Obernai, France.
8. "Use of Serum Neuron-Specific Enolase as a Marker for Seizure-Induced Brain Damage," at the *Workshop on the Neurobiology of Epilepsy - WONOEP IV*, June 25-27, 1997, Adare, County Limerick, Ireland.
9. "Does neuronal dysfunction impair maturation and functions of subcortical structures including myelin?" Plenary session at the 24th Annual Meeting of the Neuropediatric Society of Germany, Austria, and Switzerland, October 15-18, 1998, Hamburg, Germany.
10. "Acute and Chronic Effects of Status Epilepticus in Developing Brains," at the *Van Gogh Symposium on Temporal Lobe Epilepsy (A Merritt-Putnam Symposium)*, April 9-10, 1999, Los Angeles, California.
11. "Patterns of immediate early gene expression predict age- and model-dependent plasticity following experimental status epilepticus," at the *Workshop on the Neurobiology of Epilepsy – WONOEP V*, September 8-10, 1999, Český Krumlov, Czech Republic.
12. Visiting Professor with presentations to Neuroscience, Neurology, and Pediatrics, and Neurology case conferences at the *University of Manitoba*, April 27-28, 2000, Winnipeg, Manitoba.
13. "Catastrophic Childhood Epilepsies," "Anticonvulsant Pharmacology," and "Seizure-induced Injury and Plasticity in the Developing Brain," at the *Third Russian-American Symposium on the Clinical and Social Aspects of Epilepsy*, at the N.N. Burdenko Research Institute of Neurological Surgery, August 31, 2000, Moscow, Russian Federation.
14. "The potential role of topiramate in disease modification and neuroprotection," at the *Asia Pacific Roundtable*, November 10, 2000, New Delhi, India.

15. "Epileptogenesis in the Developing Brain," in a Plenary Symposium on Abnormal Plasticity in Epileptogenesis at the 3rd Congress of the *Asian and Oceanian Epilepsy Organizations*, November 11, 2000, New Delhi, India.
16. "Topiramate: Rationale for Early Use in Epilepsy," at the Plenary Session on Pediatric Epilepsy sponsored by Janssen-Cilag at the 3rd Congress of the *Asian and Oceanian Epilepsy Organizations*, November 11, 2000, New Delhi, India.
17. "Seizure-Induced Injury, Neurogenesis, and Plasticity in the Developing Brain," at the *Workshop on the Neurobiology of Epilepsy - WONOEP VI*, May 9, 2001, Iguaçu, Brazil.
18. "Studies on the Effect of Seizures on the Developing Brain," at the Young Investigators Seminar, 24th *International Epilepsy Congress*, May 13-18, 2001, Buenos Aires, Argentina.
19. Visiting Professor, *Yonsei University*, September 19, 2001, Seoul, South Korea.
20. "Neuroprotection by AEDs in the Developing Brain," at a symposium sponsored by the *Taiwanese Epilepsy Association*, September 22, 2001, Taipei, Taiwan.
21. "Seizure-induced Epileptogenicity in the Developing Brain," at a symposium at *Kaohsiung Medical University*, September 23, 2001, Kaohsiung, Taiwan.
22. "The Ketogenic Diet in the Treatment of Childhood Epilepsy," for the *Chilean League Against Epilepsy*, October 2, 2001, Santiago, Chile.
23. "Epilepsy and Migraine," "Seizures and the Developing Brain," Plenary Presentations at the *XIX Congreso de la Sociedad de Psiquiatría y Neurología de la Infancia y Adolescencia*, October 4-6, 2001, Viña del Mar, Chile.
24. Invited Discussant on severe myoclonic epilepsy of infancy versus GEFS+ syndrome in an international symposium-workshop on "*Myoclonic Epilepsies of Infancy, Childhood, Adolescence & Adulthood*," December 5,6, 2002, Seattle, Washington.
25. "Traumatic Brain Injury Exacerbates Seizure-Induced Hilar Injury in the Developing Rat Brain," *Spring Epilepsy Research Conference*, May 1, 2003, Grand Cayman, Cayman Islands.
26. "Traumatic Brain Injury and Seizures Interact to Produce Hilar Injury in the Immature Rat Brain," *Workshop on the Neurobiology of Epilepsy - WONOEP VII*, October 7-11, 2003, Ericeira, Portugal.
27. "Current Concepts in Epileptogenesis," and "Genetics of Epilepsy," Invited presentations at the 8th *Asian and Oceanian Congress of Child Neurology*, October 7-10, 2004, New Delhi, India.
28. "Children with CSWS – Variability in the Syndrome," invited presentation at the *International Symposium on Epileptic Syndromes in Infancy and Early Childhood (ISES) - Evidence-based Taxonomy and its Implications in the ILAE Classification*, April 29 - May 1, 2005, Tokyo, Japan.
29. "Modifiers of Seizure-Induced Brain Injury in the Developing Brain" at the 8th *Workshop on the Neurobiology of Epilepsy - WONOEP VIII* August 24-26, 2005, Château Villiers-le-Mahieu, France.
30. "Therapeutic Decision Making in Pediatric Epilepsy," to the *Malaysian Pediatric Association*, April 25, 2006, Kuala Lumpur, Malaysia.
31. "Seizure-Induced Injury and Epileptogenicity in the Developing Brain," at the Department of Neurology, *Yonsei University School of Medicine*, April 27, 2006, Seoul, South Korea.
32. "Inflammation Exacerbates Status Epilepticus-induced Injury in the Developing Rat Brain," invited presentation at the *International Symposium on Status Epilepticus in Infants and Young*

- Children (ISSE)- Basic Mechanisms, Clinical Evaluation, Prognosis and Treatment*, April 29 - 30, 2006, Osaka, Japan.
33. "Status Epilepticus," at *Current Trends in Epilepsy: An International Symposium by AIIMS*, Nov 4, 2006, New Delhi, India.
 34. Sankar R and Perucca E, Moderators of a debate between Patrick Kwan (in support of monotherapy) and Sunita Dergalust (in support of polypharmacy) at *Current Trends in Epilepsy: An International Symposium by AIIMS*, Nov 5, 2006, New Delhi, India.
 35. "Status Epilepticus: Pathophysiologic Mechanisms," and "Status Epilepticus: Treatment Options," at the *9th Asian and Oceanian Congress of Child Neurology (AOCCN)*, January 24-27, 2007, Cebu, Philippines.
 36. "Inflammation Modifies Status Epilepticus-Induced Hippocampal Injury During Development," at the *1st London Colloquium on Status Epilepticus*, April 12-14, 2007, London, United Kingdom.
 37. "Back to the Future with Rapid Kindling – Utility in Developmental Studies" and chairing a session on the "Mechanisms of Interictal to Ictal Transition" at the *9th Workshop on the Neurobiology of Epilepsy - WONOE IX*, July 3-6, 2007, Teluk Nibong, Langkawi Island, Malaysia.
 38. "Selecting Appropriate AEDs for Children" at the *International Epilepsy Congress*, Special Symposium organized by UCB, July 9, 2007, Singapore.
 39. "Basic Mechanisms of Teratogenesis by AEDs," at the meeting of the *5th Mediterranean Neuroscience Association*, September 26, 2007, Catania, Sicily.
 40. "Status Epilepticus: Pathophysiology and Management" at the *XXV Congreso de la Sociedad de Psiquiatría y Neurología de la Infancia y Adolescencia*, October 17-20, 2007, Termas de Chillán, Chile.
 41. "Neonatal Seizures: Pathophysiology and Therapeutics," at the *XXVII Annual Convention of the National Neonatology Forum*, December 15, 2007, Pune, India.
 42. "Ketogenic Diet in a Pill: Is This Possible?" at an *International Symposium on Dietary Therapies in Neurology*, April 4, 2008, Scottsdale, Arizona.
 43. "Experimental Studies of Depression as a Comorbidity of Epilepsy," *Institute of Physiology, Academy of Sciences of the Czech Republic*, September 29, 2008, Prague, Czech Republic.
 44. "Neurobiology of Depression in Epilepsy," *6th Mediterranean Neuroscience Congress and the 11th Etrnean Epilepsy Workshop*, October 4, 2008, Catania, Sicily, Italy.
 45. "Experimental Approach to Studying Depression in Epilepsy," "*Sapienza*" *Neuroscience Meetings*, Consiglio Nazionale delle Ricerche, *Universita di Roma*, October 6, 2008, Rome, Italy.
 46. "Pediatric Epilepsy Syndromes – Update on Genetics and Management," *The 5th International Neurology Forum*, December 13, 2008, Ho Chi Minh City, Vietnam.
 47. "Advances in Pediatric Epilepsy," Professor Jagannathan Oration at the *Institute of Neurology, Madras Medical College*, February 5, 2009, Chennai, India.
 48. "Pediatric Epilepsy Syndromes and Treatment Options," at ECON 2009, 10th Annual Conference of the *Indian Epilepsy Association and the Indian Epilepsy Society*, February 7, 2009, Tirupati, India.
 49. "Therapeutic Decision Making in Pediatric Epilepsy," Annual Meeting of the *Delhi Neurological Society* held at the *All India Institute of Medical Sciences*, February 8, 2009, New Delhi, India.

50. "Ketogenic Diet and Other Dietary Therapies for Epilepsy," and "Optimal Treatment of Pediatric Epilepsy at the 10th Asian & Oceanian Congress of Child Neurology (AOCCN) and 32nd Congress of the Korean Child Neurology Society, June 10-13, 2009, Daegu, South Korea.
51. "Evaluation of age-specific targets for antiepileptogenic therapy," at the 10th Workshop on the Neurobiology of Epilepsy - WONOEP X, June 24-26, 2009, Pécs, Hungary.
52. "From the Newly Diagnosed to the Refractory Patient: Current Concepts in Rationalization of AED Therapy" and "The New Kids on the Block: Understanding the New AEDs," presentations at the Long Distance Epilepsy Education and Awareness Program (LEAP), a Multi-Center Epilepsy Symposium organized by the Epilepsy Partnership India (EPI) Trust, January 31, 2010, Mumbai, India.
53. "Clinical and Scientific Perspectives on Epilepsy among Neurometabolic Diseases in Children," at the International Symposium on Epilepsy in Neurometabolic Diseases - 13th Annual Meeting of the Infantile Seizure Society and the 14th Annual Meeting of the Taiwan Child Neurology Society, March 27, 2010, Taipei, Taiwan.
54. "Pharmacologic treatment of common pediatric epilepsy syndromes," Management of the most common comorbidities in Pediatric Epilepsy," and "Is monotherapy the best strategy in the treatment of refractory epilepsy in clinical practice? In a symposium on *Strategies for Optimizing the Treatment of Pediatric Epilepsy* at the Annual Meeting of the Colombian Child Neurology Society, May 1, 2010, Caratgena, Colombia.
55. "Can animal models be used to study whether epilepsy has an impact on depression?" at an Epilepsy & Behavior conference: *Epilepsy and Depressive Disorders*, September 12-13, 2010, Chicago, Illinois.
56. "Status epilepticus and Inflammation in immature rats," at, Immunity and Inflammation in Epilepsy: Mechanistic Insights and Therapeutic Perspectives," at the Mario Negri Istituto di Recherche Farmacologiche, September 16-18, 2010, Milan, Italy.
57. "Epilepsy and the Maturing Brain," at the II Reunión Internacional Universitaria de Neurología sponsored by La Universidad Autónoma de San Luis Potosi, September 23-25, 2010, San Luis Potosi, Mexico.
58. "Risk reduction in epilepsy - Cellular perspective: Neuroprotection," at the Annual Scientific Meeting of the UK Chapter of the International League Against Epilepsy, October 13-15, 2010, Brighton, England.
59. "Inflammation and the Epilepsies – a Complex Theatre," at the Pediatric Epilepsy Training 3 (PET3) course organized by the British Pediatric neurology Association, March 29, 2011, Edinburgh, Scotland.
60. "Depression in Epilepsy: Explorations into the Neurobiological Connection," Pediatric Neurology Grand Rounds at the Alberta Children's Hospital, University of Calgary, July 5, 2011, Calgary, Alberta, Canada.
61. "Depression in Epilepsy: Explorations into the Neurobiological Connection," Pediatric Neurology Grand Rounds at the BC Children's Hospital, University of British Columbia, July 6, 2011, Vancouver, British Columbia, Canada.
62. "Seizure mechanisms and action of antiepileptic drugs," at the Advanced International Course: bridging Basic with Clinical Epileptology-4, sponsored by the International League Against epilepsy at Venice International University, July 23, 2011, San Servolo (Venice), Italy.
63. "Optimizing treatment of epilepsy syndromes and the use of newer AEDs" at the Annual Conference of the Indian Academy of Pediatrics – Child Neurology Chapter, October 9, 2011, Mumbai, India.

64. "Treatment of Status Epilepticus," at the Annual Conference of the *Indian Academy of Pediatrics – Child Neurology Chapter*, October 9, 2011, Mumbai, India.
65. "Cortical Dysplasia in Epilepsy," at the Annual Conference of the *Indian Academy of Pediatrics – Child Neurology Chapter*, October 9, 2011, Mumbai, India.
66. "Inflammation, Epilepsy & Behavior," at the *Prague Symposium of Child Neurology and Developmental Epileptology*, November 4-5, 2011, Prague, Czech Republic.
67. "Epilepsy Syndromes-Their Relevance to the Practicing Neurologist," at the *Neurology Update Mumbai 2012*, February 17-19, 2012, Mumbai, India.
68. "Therapeutics in New Onset Epilepsy-The Old Order Changeth?" at the *Neurology Update Mumbai 2012*, February 17-19, 2012, Mumbai, India.
69. "Refractory Status Epilepticus," at the *Neurology Update Mumbai 2012*, February 17-19, 2012, Mumbai, India.
70. "Experimental Models of Developmental Symptomatic Epilepsy," at the *Latin American Summer School in Epilepsy VI (LASSE VI)*, February 25, 2012, São Paulo, Brazil.
71. "Depression in Epilepsy: Explorations into the neurobiological Connection," at the *Latin American Summer School in Epilepsy VI (LASSE VI)*, February 26, 2012, São Paulo, Brazil.
72. "Effect of Stress on the Hippocampus in Epilepsy and Depression," at the *2nd International Congress on Epilepsy, Brain and Mind*, March 29-31, 2012, Prague, Czech Republic.
73. "Future Targets for Antiseizure Therapy in the Neonate: Preclinical and Clinical Observations," at the *International Symposium on Neonatal Seizures, the 15th Annual Meeting of the Infantile Seizure Society*, April 12-14, 2013, Tokyo, Japan.
74. "Medically Refractory Epilepsy: Selection for Pediatric Epilepsy Surgery," at the *XXII Reunión Annual, Soceidad Mexicana de Neurologia Pediatrica*, May 22-25, 2013, Oaxaca, Mexico.
75. "Epilepsy and the Comorbidities: Exploring the Connection," at the *XXII Reunión Annual, Soceidad Mexicana de Neurologia Pediatrica*, May 22-25, 2013, Oaxaca, Mexico.
76. "Epilepsy and the Comorbidities," Introductory presentation on a session *co-chaired by* Raman Sankar, MD, PhD (Los Angeles) and Christoph Helmstaedter, MD, PhD (Bonn) at the *European Forum on Epilepsy Research* (sponsors, International League Against Epilepsy and the International Bureau of Epilepsy), May 27, Dublin, Ireland.
77. "A Short History of the Workshop on the Neurobiology of Epilepsy," at the *WONOE XII*, June 20, 2013, Lake Dupuis, Quebec, Canada.
78. "Evolution of AEDs and Beyond" and "Epileptic Encephalopathies," at the *Colloquium on Drug Resistant Epilepsy* at the *National Institute for Mental Health and Neuro Sciences (NIMHANS)*, August 16-18, 2013, Bangalore, India.
79. "New Antiepileptic Therapy for Children 2014: Disappointments & Promises," *International Symposium on Epilepsy Syndromes*, June 25, 2014, Cappadocia, Turkey.
80. "The Impact of Epilepsy on Children," at the *International Conference on Epilepsy: Opportunities and Challenges*, October 31 – November 1, 2014, New Delhi, India.
81. "Common Epilepsy Syndromes of Childhood," "Severe Epileptic Syndromes of Infancy and Childhood," "Interictal and Ictal Signatures of Neonatal and Childhood Seizures," "Genetic Generalized Epilepsies," and Epilepsy and Common Comorbidities: the Biological Basis," at the

- ECON 2015, *16th Joint Annual Conference of the Indian Epilepsy Association and the Indian Epilepsy Society*, February 6-8, 2015, Chennai, India.
82. "Pharmacologically Refractory Epilepsy in Infancy and Childhood," at the *XXIV Reunión Annual, Sociedad Mexicana de Neurología Pediátrica*, May 21, 2015, Guadalajara, Mexico.
 83. "Medically Refractory Epilepsy – Etiologies, Evaluations, and Management Approaches," at the Annual Symposium of the Epilepsy Center, *Second Affiliated Hospital of the Zhejiang University (SAHZU) College of Medicine*, July 25, 2015, Hangzhou, Peoples Republic of China.
 84. "Epilepsy and Comorbidities: Investigations on the Neurobiological Connection," at the *Zhejiang University College of Medicine*, July 28, 2015, Hangzhou, Peoples Republic of China.
 85. "Introduction to the Session on Biomarkers of Comorbidities" at the 13th Workshop on the Neurobiology of Epilepsy (WONOE XIII), September 2, 2015, Heybeliada Island, Turkey.
 86. "A Personal Tribute Professor Yukio Fukuyama," at the *International Symposium on Benign Epilepsy Syndromes*, September 26, 2015, Tokyo, Japan.
 87. "Challenges and Opportunities in Drug Development for Severe Pediatric Epilepsy Syndromes," at the *UCB Epilepsy Summit*, October 1, 2015, Braine l'Alleud, Belgium.
 88. "Standard of Care in the U.S. for Infantile Spasms," at the *Radiprodil Advisory Board Meeting*, October 2, 2015, Leuven, Belgium.
 89. "Pharmacologic and Molecular Genetic Update: Rolandic epilepsy," at the 58th Annual Meeting of the *Japanese Society of Child Neurology*, June 3, 2016, Tokyo, Japan.
 90. "Progress and Challenges in the Understanding of Epileptic Encephalopathies," the **First Professor Yukio Fukuyama Memorial Lecture** at the 58th Annual Meeting of the *Japanese Society of Child Neurology*, June 4, 2016, Tokyo, Japan.
 91. "Acute Encephalopathy with Inflammation-mediated Status Epilepticus," at the *International Symposium on Acute Encephalopathies in Infancy (ISAE2016)*, July 3, 2016, Tokyo, Japan.
 92. "Epilepsy Genetics – Applying Data to Actions," at Genes and Epilepsy: At a Crossroads When Timely Diagnosis of CLN2 Disease matters," at the Annual Meeting of the *Child Neurology Society*, October 27, 2016, Vancouver BC, Canada.
 93. "Autism Spectrum Disorders and Epilepsy: A Mechanistic Update," at the *14th Asian and Oceanian Congress of Child Neurology (AOCCN 2017)*, May 11, 2017, Fukuoka, Japan.
 94. "Pathophysiology of Status Epilepticus," Plenary lecture at the *Joint Child Neurology Society of the Philippines – Philippines League Against Epilepsy (CNSP – PLAE) Symposium on Status Epilepticus & the 9th National Epilepsy Congress*, July 17, 2017, Cebu, Philippines.
 95. "Epilepsy Neurogenetics: Earlier diagnosis of CLN2 disease in clinical practice," in a symposium on CLN2 disease at the *International Epilepsy Congress*, September 3, 2017, Barcelona, Spain.
 96. "New Options in Australia for the Treatment of Pediatric Epilepsy," at the *Royal Children's Hospital*, September 10, 2018, Melbourne, Australia.
 97. "Epilepsy, Inflammation, and the Development of Comorbidities," at the *7th Annual Scientific Meeting of the Australia New Zealand Child neurology Society (ANZCNS)*, September 13, 2018, Perth, Australia.
 98. "Pathophysiology of Status Epilepticus," at the *7th Annual Scientific Meeting of the Australia New Zealand Child Neurology Society (ANZCNS)*, September 13, 2018, Perth, Australia.

99. "New Neurobiology-based Classification of Status Epilepticus and Mechanisms of Drug Resistant Status Epilepticus," at the *19th International Symposium on Severe Infantile Epilepsies*," *Ospedale Pediatrico Bambino Gesù, Università Urbaniana*, September 20, 2018, Rome, Italy.
100. "Early Onset Epilepsies," Session Co-chair with Prof Hideo Yamanouchi at the *19th International Symposium on Severe Infantile Epilepsies*," *Ospedale Pediatrico Bambino Gesù, Università Urbaniana*, September 21, 2018, Rome, Italy.
101. "Impact of Comorbidities on the Quality of Life in Pediatric Epilepsy," at the *Advances in Neonatology and Pediatrics*, March 10, 2019, Mumbai, India.
102. "Evaluation of Children for Epilepsy Surgery," at the *Advances in Neonatology and Pediatrics*, March 10, 2019, Mumbai, India.
103. "Precision Medicine in Epilepsy: Opportunities and Challenges," at the *Deenanath Mangeshkar Hospital*, March 11, 2019, Pune, India.
104. "An Overview of Epileptic Encephalopathies," Keynote Address at the *International Symposium on Developmental Epileptic Encephalopathies*, June 19, 2020 (Virtual Meeting by Zoom due to Covid-19 limitations), Okayama, Japan.
105. "Advanced Tools for the diagnosis of structural epileptogenic substrates" Co-chaired session with Dr. Terence O'Brien (July 7, 2022) at the XVI Workshop on the Neurobiology of Epilepsy – WONOE 2022, July 4-8, 2022, Talloires, France. (Also served on the Scientific Organizing Committee)

National:

1. "Role of Subcortical Structures in Infantile Spasms: An FDG-PET Study Showing Activation of the Lenticular Nuclei and the Brainstem," Neurology Grand Rounds, Spring 1991, *University of North Carolina*, Chapel Hill, North Carolina.
2. "Advances in the Management of Catastrophic Epilepsy in Children," presented to the pediatric house-staff and attending physicians at *Tripler Army Medical Center*, June 3, 1993, Honolulu, Hawaii.
3. "Current Concepts in the Treatment of Epilepsy," presented to the medical staff of *Straub Clinic and Hospital*, June 4, 1993, Honolulu, Hawaii.
4. "Antiepileptic Therapy: Arrival of the New Agents," presented at the Second Annual Symposium of the *Epilepsy Foundation of Greater Chicago, Recent Advances in Clinical Epilepsy*, March 5, 1994, Chicago, Illinois.
5. "Microscopic Cortical Dysplasia in Infantile Spasms: Evolution of White Matter Signal Changes," special seminar at the *Massachusetts General Hospital, Harvard Medical School*, May 31, 1994, Boston, Massachusetts.
6. "Patient Re-evaluation: Impact of New Generation Antiepileptic Agents," *Tufts Medical School - New England Medical Center*, June 1, 1994, Boston, Massachusetts.
7. "Developmental Aspects of Neuroimaging in Infantile Spasms," *Boston Children's Hospital, Harvard Medical School*, June 3, 1994, Boston, Massachusetts.
8. "Classification, Etiology and neurophysiology of Cerebral Palsy," at *Management of Children with Spastic Cerebral Palsy*, A conference for neurologists, pediatricians, neurosurgeons, orthopedic surgeons and physical therapists, February 3-6, 1995, Ensenada, Mexico.

9. "Neuroimaging of the Developing Brain in Intractable Epilepsy," at the *Children's Hospital of Pittsburgh / University of Pittsburgh School of Medicine*, February 15, 1995, Pittsburgh, Pennsylvania.
10. "Neuroimaging of the Developing Brain in Intractable Childhood Epilepsy," Grand Rounds of the Department of Neurology, *University of Washington School of Medicine*, May 18, 1995, Seattle, Washington.
11. "Seizure Disorders: Current Management and Approaches to Treatment," "Meningitis and Other CNS Infections," and "Headaches and Head Injuries: Assessment and Management," at *Clinical Issues in Pediatrics*, a national conference, August 17-18, 1995, Jackson Hole, Wyoming.
12. "Neuroimaging and the Developing Brain: Detection of Microscopic Cortical Dysplasia," Grand Rounds of the Department of Neurology, *Stanford University School of Medicine*, November 8, 1995, Palo Alto, California.
13. "Preclinical and Clinical Pharmacology of New Generation Anticonvulsants," Neurology, Neurosurgery Grand Rounds, *Santa Clara Valley Medical Center*, November 9, 1995, San Jose, California.
14. "Classification, Etiology and neurophysiology of Cerebral Palsy," at *Management of Children with Spastic Cerebral Palsy*, A conference for neurologists, pediatricians, neurosurgeons, orthopedic surgeons and physical therapists, March 22-24, 1995, Ensenada, Mexico.
15. Invited faculty for "Pediatric Epilepsy Case Studies After Dinner Seminar" at the 48th Annual Meeting of the *American Academy of Neurology*, March 25, 1996, San Francisco, California.
16. "Neurotransmitters, Ion Channels, and Antiepileptic Drugs," *Children's Hospital*, March 29, 1996, San Diego, California.
17. "Child Neurology for the Pediatric Practitioner," at the Pediatric Board Review Course by the Osler Institute, July 14, 1996, Los Angeles, California.
18. "Weakness, Hypotonia, and Ataxia," and "Congenital Brain Malformations, Metabolic and Neurodegenerative Diseases," at the Pediatric Board Review Course by the UCLA Children's Hospital, September 6, 1996, Los Angeles, California.
19. Visiting Professor with the Division of Pediatric Neurology, *Tufts University Medical School, New England Medical Center*, March 5, 1997, Boston, Massachusetts.
20. "Ontogeny of Status Epilepticus (SE) and SE-induced Brain Damage," Neurology Grand Rounds at the *Massachusetts General Hospital / Harvard Medical School*, March 6, 1997, Boston, Massachusetts.
21. Faculty, *Pediatric Board Review Course*, UCLA Department of Pediatrics and Mattel Children's Hospital at UCLA, September, 1997, Los Angeles, California.
22. "Neurotransmitters, Ion Channels, and Antiepileptic Drugs," *Children's Hospital and Medical Center*, October 18, 1997, Seattle, Washington.
23. "Seizure-induced Apoptosis and Necrosis in the Developing Rat Brain," Grand Rounds at the Department of Neurology, *University of Arizona*, October 24, 1997, Tucson, Arizona.
24. "Treatment of Status Epilepticus in Children," at the Pediatric Neurology Conference for Primary Care Physicians sponsored by *Barrow Neurological Institute*, October 25, 1997, Phoenix, Arizona.
25. "Seizure-induced Brain Damage in the Developing Brain," *Children's Hospital and Regional Medical Center, and the University of Washington*, April 8, 1998, Seattle, Washington.

26. "Patterns of Status Epilepticus-Induced Neuronal Injury during Development." Grand Rounds of the Department of Neurology, *University of Washington School of Medicine*, June 11, 1998, Seattle, Washington.
27. Faculty, *Pediatric Board Review Course*, UCLA Department of Pediatrics and Mattel Children's Hospital at UCLA, September, 1998, Los Angeles, California.
28. "Carnitine and the Ketogenic Diet," at *The Ketogenic Diet: Models and Mechanisms: A Regional Workshop*, Battelle Conference Center, September 18, 1998, Seattle, Washington.
29. "Epilepsy syndromes, Genetics, and Treatment," for *Washington, Oregon Neurology Group*, April 10, 1999, Skamania Lodge, Stephenson, Washington.
30. "Neuromuscular Disorders," at the 4th Annual Primary Care Update, presented by the *UCLA School of Medicine*, April 16-18, 1999, Las Vegas, Nevada.
31. "Do Seizures Cause Brain Damage?" at the Update in Child Neurology & Psychiatry, presented by *Children's Hospital & Regional Medical Center* and the *University of Washington*, May 8, 1999, Seattle, Washington.
32. Faculty, *Pediatric Board Review Course*, UCLA Department of Pediatrics and Mattel Children's Hospital at UCLA, September, 1999, Los Angeles, California.
33. "Rational Drug Sequencing in Children to Separate Medically Refractory from Medically Responsive Epilepsy," at the Epilepsy Educator' Conference, October 23, Palm Beach, Florida.
34. "Pathology of Childhood Epilepsy: Effects of Maturation on Neuroimaging," at the *Pediatric Symposium on Brain Imaging Techniques in Children with Epilepsy*, Annual Meeting of the *American Epilepsy Society*, December 3, 1999, Orlando, Florida.
35. "Seizure-Induced Neuronal Injury in the Maturing Brain," at the symposium *Neuroprotection: Implications for the Future Clinical Management of Epilepsy*, Annual Meeting of the *American Epilepsy Society*, December 5, 1999, Orlando, Florida.
36. "Are Infrequent Seizures Harmful?" Speaker at the participatory roundtable, Annual Meeting of the *American Epilepsy Society*, December 5, 1999, Orlando, Florida.
37. "Seizure-Induced Injury and Plasticity in the Immature Brain," Grand Rounds at the Department of Neurology, School of medicine, *St. Louis University*, January 13, 2000, St. Louis, Missouri.
38. "Advances in the Treatment of Pediatric Seizure Disorders," Grand Rounds at the Department of Pediatrics, College of Medicine, *University of Iowa*, January 14, 2000, Iowa City, Iowa.
39. "Rational Use of Anticonvulsants in Pediatric Epilepsy," at a continuing education program sponsored by the *University of South Florida*, February 26, 2000, Tampa, Florida.
40. "Impact of Seizures on the Developing Brain: Injury and Plasticity," Grand Rounds of the Department of Neurology, *State University of New York*, April 5, 2000, Syracuse, New York.
41. "The Impact of AEDs on Brain Development: Neurocompromise vs. Neuroprotection," in a symposium on the "Clinical Uses of Antiepileptic Drugs," sponsored by the *Children's Hospital & Regional, Medical Center, University of Washington School of Medicine, and the Regional Epilepsy Center, Harborview Medical Center*, May 5, 2000, Seattle, Washington.
42. Several topics on pediatric epilepsy at the "Pediatric Epilepsy Preceptorship" sponsored by the *Bowman Gray Medical School of Wake Forest University*, June 22-23, 2000, Winston-Salem, North Carolina.

43. Visiting Professor, presentations to the epilepsy faculty, Department of Neurology, case conferences, and Grand Rounds of the Neurosurgery Department, University of Michigan, July 17-19, 2000, Ann Arbor, Michigan.
44. Faculty, *Pediatric Board Review Course*, UCLA Department of Pediatrics and Mattel Children's Hospital at UCLA September, 2000, Los Angeles, CA.
45. Symposium Chair for "Pediatric Epilepsy: From Bench to Clinical Practice" and speaker: "Pediatric Epilepsy for the Adult Neurologist," at the *Annual Meeting of the American Epilepsy Society*, December 2, 2000, Los Angeles, California.
46. Chairman, Special Interest Group on "Basic Neuroscience" at the *Annual Meeting of the American Epilepsy Society*, December 5, 2000, Los Angeles, California.
47. "Seizures in the Developing Brain," Grand Rounds at the *Primary Children's Hospital, University of Utah*, Jan 10, 2001, Salt Lake City, Utah.
48. "AED Use in Pediatrics," and "Oxcarbazepine and Zonisamide," in a Continuing Medical Education program on New Anticonvulsants at the *Case Western Reserve University School of Medicine*, February 17, 2001, Cleveland, Ohio.
49. "Pharmacology and Therapeutics of AEDs in Pediatric Patients," in a Continuing Medical Education program on Pediatric Neurology at *University of Southern Florida School of Medicine*, March 10, 2001, St. Petersburg Beach, Florida.
50. Several lectures and workshop leader at the *Kiffin Penry Memorial Epilepsy Mini-fellowship* sponsored by *Wake Forest University School of Medicine*, March 15-18, 2001, Winston-Salem, North Carolina.
51. "Prevention of Epileptogenesis as a Goal for Treatment with AEDs," *Wake Forest University School of Medicine, Bowman Gray Campus*, June 7, 2001, Winston-Salem, North Carolina.
52. "Pharmacologic Treatment of Childhood Epilepsy," *Cook's Children's Medical Center*, August 8, 2001, Fort Worth, Texas.
53. Faculty, *Pediatric Board Review Course*, UCLA Department of Pediatrics and Mattel Children's Hospital at UCLA, September, 2001, Los Angeles, California.
54. "Treatment of Pediatric Epilepsy – Present and Future," at Southern Regional Epilepsy Symposium, September 7-8, 2001, Asheville, North Carolina.
55. "Applications of Positron Emission Tomography in Epilepsy and Autism," Breakfast Symposium, *Annual Meeting of the Child Neurology Society*, October 20, 2001, Victoria, British Columbia, Canada.
56. "Drug Selection in Gender- and Age-Based Population: Pediatric Patients," in "A Contemporary Assessment of seizures and Epilepsy." *A Merritt-Putnam Lecture Series* sponsored by the *American Epilepsy Society*. Program was developed by Drury I, Ferrendelli JA, Ramsay RE and **Sankar R**. November 10, 2001, Detroit, Michigan.
57. "Epilepsy and the Developing Brain," Neurology grand Rounds, *University of Medicine and Dentistry of New Jersey*, January 16, 2002, and the VA Hospital, Orange, New Jersey.
58. "The Developing Brain – Seizures, Injury, Antiepileptic Drugs," Neurology Grand Rounds, *School of Medicine, State University of New York Downstate*, January 18, 2002, Brooklyn, New York.

59. "Use of AEDs in Neurological Disorders," Grand Rounds at the *South Beach Psychiatric Hospital*, January 18, 2002, Staten Island, New York.
60. "The Developing Brain – How to Optimize the Choice of AEDs?" Grand rounds of the Department of Neurology, *Loma Linda University School of Medicine*, February 6, 2002, Loma Linda, California.
61. Several lectures and workshop leader at the Kiffin Penry Memorial Epilepsy Mini-fellowship sponsored by *Wake Forest University School of Medicine*, February 28 – March 2, 2002, Winston-Salem, North Carolina.
62. "Pediatric Epilepsy Syndromes," and Breakfast Case Studies at the *Uniformed Services Pediatric Seminar* sponsored by the *American Academy of Pediatrics*, March 14, 2002, San Diego, California.
63. "Epilepsy and the developing Brain," at the *Children's Hospital of Wisconsin*, March 22, 2002, Milwaukee, Wisconsin.
64. "Selection of Anticonvulsants in the Pediatric Patient," *Merritt-Putnam Lectures on Epilepsy*, American Epilepsy Society, May 4, 2002, Baltimore, Maryland.
65. "Selection of Anticonvulsants in the Pediatric Patient," *Merritt-Putnam Lectures on Epilepsy*, American Epilepsy Society, May 18, 2002, San Francisco, California.
66. "Seizure-Induced Injury and Plasticity in the Developing Brain," Neurology Grand Rounds at *University of Arizona*, May 31, 2002, Tucson, Arizona.
67. Several lectures and workshop leader at the Kiffin Penry Memorial Epilepsy Mini-fellowship sponsored by *Wake Forest University School of Medicine*, June 20-22, 2002, Winston-Salem, North Carolina.
68. "Status Epilepticus Induced Epileptogenicity in the Developing Brain," Grand Rounds of the Department of Neurology, School of Medicine, *University of Utah*, July 10, 2002, Salt Lake City, Utah.
69. "Pediatric Epilepsy Syndromes – a Current Perspective," in the 2002 Epilepsy Symposium presented by the *University of New Mexico School of Medicine*, August 24, 2002, Santa Ana Pueblo, New Mexico.
70. Faculty, *Pediatric Board Review Course*, UCLA Department of Pediatrics and Mattel Children's Hospital at UCLA, September, 2002, Los Angeles, California.
71. "Medically Refractory Pediatric Epilepsy," "Seizures and the Developing Brain," and "A New Look at Pediatric Epilepsy Syndromes," presentations to medical students and pediatric residents, Pediatric and Neurology Grand Rounds, respectively, at *Oregon Health Sciences University*, September 18-19, 2002, Portland, OR.
72. "Pharmacology of Anticonvulsant Action," to the Adult and Pediatric Epilepsy Groups at *Duke University*, November 10, 2002, Durham, North Carolina.
73. Invited speaker in a Participatory Roundtable on "Neurodevelopmental Effects of Epilepsy on Learning/Behavior and Brain Structure in Humans and Animals," at the Annual Meeting of the *American Epilepsy Society*, December 8, 2002, Seattle, Washington.
74. Several lectures and workshop leader at the Kiffin Penry Memorial Epilepsy Mini-fellowship sponsored by *Wake Forest University School of Medicine*, June 20-21, 2003, Winston-Salem, North Carolina.
75. One of two principal guest faculty (several lectures on epilepsy, anticonvulsant drugs and on management of status epilepticus) at the "Current Concepts in Child Neurology: San Diego 2003," a

- Continuing Medical Education Program offered every three years by the *Children's Hospital and Health Center of San Diego*, June 27-29, 2003, San Diego, California.
76. "Brain Injury and Plasticity in the Developing Rat," Grand Rounds in the Department of Neurology at the *Medical University of South Carolina*, July 10, 2003, Charleston, South Carolina.
 77. "How Do Seizures Affect the Infant Brain?" Pediatric Grand Rounds at the *Pritzker School of Medicine*, University of Chicago, September 4, 2003, Chicago, Illinois.
 78. "Seizures and the Developing Brain: Injury, Neurogenesis and Circuit Rearrangement," Neurology Grand Rounds at *Rush Medical School and Rush Presbyterian St. Luke's Hospital*, September 4, 2003, Chicago, Illinois.
 79. Faculty, *Pediatric Board Review Course*, UCLA Department of Pediatrics and Mattel Children's Hospital at UCLA, September, 2003, Los Angeles, California.
 80. Faculty in Neurology at "PREP: The Course," sponsored by the *American Academy of Pediatrics*, September 13 – 16, 2003, Salt Lake City, Utah.
 81. "Pediatric Seizure Disorders and Their Treatment," Pediatric Grand Rounds at *Blank Children's Hospital*, October 24, 2003, Des Moines, Iowa.
 82. "Seizure Disorders: How to Diagnose & When to Refer?" Seminar at the National Conference and Exhibition of the American Academy of Pediatrics, November 4, 2003, New Orleans, Louisiana.
 83. "Classification of Status Epilepticus," Spoke in and moderated a Participatory Roundtable at the *Annual Meeting of the American Epilepsy Society*, December 7, 2003, Boston, Massachusetts.
 84. "Early Treatment of Epilepsy: What If the First Drug Fails?" Presentation to Epilepsy/Clinical Neurophysiology Fellows in Training at the TOPScholars Symposium, *Annual Meeting of the American Epilepsy Society*, December 9, 2003, Boston, Massachusetts.
 85. "Monotherapy with Antiepileptic Drugs in Pediatric Epilepsy," at a special symposium at the *Annual Meeting of the Southern Clinical Neurological Society*, January 16, 2004, St. Thomas, Virgin Islands.
 86. "Neonatal Seizures: A Challenge in Bench to Bedside Translation," Presentation to the Division of Pediatric Neurology at *University of California San Francisco*, March 10, 2004, San Francisco, California.
 87. "Monotherapy versus Polypharmacy in the Treatment of Epilepsy," at *Oakland Children's Hospital*, March 17, 2004, Oakland, California.
 88. "Molecular Targets of Anticonvulsant Action," at the Department of Neurology, *Rush Medical School*, June 24, 2004, Chicago, Illinois.
 89. "AES and AAN Recommendations on New AEDs: Practice parameters," Commentary and a Discussion on "New Paradigm for Treatment of New Onset Epilepsy" at *Children's Memorial Hospital*, June 24, 2004, Chicago, Illinois.
 90. "Seizures and the Developing Brain: Cellular Injury, Neurogenesis and Circuit Rearrangement," at the *Children's Hospital of Wisconsin*, June 25, 2004, Milwaukee, Wisconsin.
 91. "Molecular Targets for Anticonvulsant Pharmacotherapy in the Mature and Developing Brain," Department of Neurology Grand Rounds at the *Medical University of South Carolina*, August 12, 2004, Charleston, South Carolina.
 92. Faculty, *Pediatric Board Review Course*, UCLA Department of Pediatrics and Mattel Children's Hospital at UCLA, September, 2004, Los Angeles, California.

93. "Seizure-Induced Injury and Plasticity in the Developing Brain," Neurology Grand Rounds at *University of Texas Medical School*, September 9, 2004, San Antonio, Texas.
94. "A New Paradigm for the Treatment of Epilepsy," at *Wilford Hall Medical Center, Lackland Air Force Base*, September 9, 2004, San Antonio, Texas.
95. "Selecting Pharmacotherapy: Decision Making in Pediatric Epilepsy," at a satellite symposium on *Current Guidelines for the Treatment of Pediatric Epilepsy* at the *Annual Meeting of the Child Neurology Society*, October 16, 2004, Ottawa, Canada.
96. "Genetics of the Epilepsies," and "New Recommendations for Pharmacotherapy of Childhood Epilepsy Syndromes," at *New Horizons in Epilepsy*, a conference at the *University of Oklahoma (OUHSC)*, November 5, 2004, Oklahoma City, Oklahoma.
97. "New AEDs; is Rational Polypharmacy Feasible?" at the *Annual Neurology Conference of the Army Medical Department (AMEDD)*, November 16, 2004, Silver Springs, Maryland.
98. "Current Concepts in Epileptogenesis," at the *Floating Hospital for Children, New England Medical Center*, November 19, 2004, Boston, Massachusetts.
99. "Refractory Status Epilepticus: Why does it Occur and What to Do About It?" at the XVII Penry Minifellowship at the *Annual Meeting of the American Epilepsy Society*, December 4, 2004.
100. Introductory presentation and symposium moderator for the satellite symposium on "Neuroprotection in Epilepsy," at the *Annual Meeting of the American Epilepsy Society*, December 5, 2004.
101. "Mechanisms of AED Action and an Approach to Rational Polypharmacy," at the Winter meeting of the *Texas Neurological Society*, February 11, 2005, Austin, Texas.
102. "Molecular Targets of Antiepileptic Drugs: Potential for Novel Therapeutic Regimens," Neurology Grand Rounds at *St. Louis University School of Medicine*, February 17, 2005, St. Louis, Missouri.
103. "Therapeutic Advances in the management of Childhood Epilepsy," to the Division of Pediatric Neurology at *Cardinal Glennon Children's Hospital*, February 18, 2005, St. Louis, Missouri.
104. "Pharmacologic Diversity in Anticonvulsant Medications," presentation to neurology residents at *University of South Alabama*, March 29, 2005, Mobile, Alabama.
105. "Bench to Bedside Challenges in the Management of Neonatal Seizures," Neurology Grand Rounds, *University of Alabama at Birmingham*, March 30, 2005, Birmingham, Alabama.
106. "Seizure-induced Brain Injury in the Developing Brain," Neuroscience Grand Rounds, Barrow Neurological Institute, May 18, 2005, Phoenix, Arizona.
107. "Seizures and the Developing Brain: Scientific and Clinical Aspects," Neurology Grand Rounds, *Wayne State University School of Medicine*, May 20, 2005, Detroit, Michigan.
108. "Consequences of Status Epilepticus in the Developing Brain," Neurology Grand Rounds, *Boston University Medical Center*, May 31, 2005, Boston, Massachusetts.
109. "Studies on Seizure-induced Brain Injury as a Function of Seizure Duration," Epilepsy Research Conference, *Massachusetts General Hospital, Harvard medical School*, May 31, 2005, Boston, Massachusetts.
110. Case conference in pediatric epilepsy surgery," *Children's Medical Center, University of Texas Southwestern Medical School*, July 20, 2005, Dallas, Texas.

111. "Optimization of Pharmacotherapy in Epilepsy," to residents from *University of Texas, San Antonio* Department of Neurology, July 21, 2005, San Antonio, Texas.
112. Faculty, *Pediatric Board Review Course*, UCLA Department of Pediatrics and Mattel Children's Hospital at UCLA, September, 2005, Los Angeles, California.
113. "Consequences of Seizures on the Developing Brain," at the symposium on *Localization-related Epilepsy in Children – Annual Meeting of the Child Neurology Society*, September 29, Los Angeles, California.
114. "Narrow vs. Broad Spectrum AEDs in Focal Epilepsy: Can Mechanism Guide Optimal Use?" at the symposium on the *Role of AEDs in Pediatric Focal Epilepsies - Annual Meeting of the Child Neurology Society*, September 29, Los Angeles, California.
115. "Medically Refractory Epilepsy," a seminar at the *National Conference & Exhibition of the American Academy of Pediatrics*, October 10, 2005, Washington, DC.
116. "Optimization of Therapy in Epilepsy: Impact of New AEDs," at the *Department of Neurology, Harborview Hospital, University of Washington*, November 8, 2005, Seattle, Washington.
117. "Optimization of Pharmacotherapy in Epilepsy," at *Madigan Army Medical Center*, November 9, 2005, Tacoma, Washington.
118. "Selecting Pharmacotherapy: Decision Making in Pediatric Epilepsy," at *Rainier School, Division of Developmental Disabilities, Washington State Department of Social & Health Services*, November 9, 2005, Buckley, Washington.
119. "Of Science and Semantics: Concept of Impending Status Epilepticus," at the Clinical Roundtable, *Annual Meeting of the American Epilepsy Society*, December 3, 2005, Washington D.C.
120. "On Presynaptic versus other sites of action of AEDs," Invited discussant at the Basic Science Special Interest Group Round Table, *Annual Meeting of the American Epilepsy Society*, December 6, 2005, Washington D.C.
121. "Mechanisms of Epileptogenesis as a Basis for the Design of Rational Therapeutics," Neuroscience Grand Rounds at the *Children's Hospital of Philadelphia – University of Pennsylvania*, February 23, 2006, Philadelphia, Pennsylvania.
122. "The Place for Polypharmacy in Pediatric Epilepsy," Invited plenary lecture at the Annual Winter Meeting of the *Texas Neurological Society*, February 24, 2006, Austin, Texas.
123. "Modifiers of Seizure-Induced Brain Injury in the Developing Brain," Neuroscience Grand Rounds at the *McKnight Mind-Brain Research Institute*, Department of Neurology, *University of Florida*, March 7, 2006, Gainesville, Florida.
124. "Epileptogenic Mechanisms and Rational Design of Antiepileptic Therapy," Presentation to neurology residents and faculty at the *Pritzker School of Medicine, University of Chicago*, March 27, 2006, Chicago, Illinois.
125. "Mechanisms of Epileptogenesis as a Basis for the Design of Rational Therapeutics," Presentation at Neurology Grand Rounds at the *Upstate Medical Center, State University of New York*, April 19, 2006, Syracuse, New York.
126. "Therapeutics of Epilepsy: Relationship to Mechanisms of Epileptogenesis," at the weekly epilepsy conference at the *Miami Children's Hospital*, May 31, 2006, Miami, Florida.
127. "Epileptogenic Mechanisms and Therapeutics in Partial and Generalized Epilepsies," *Children's Hospital and Regional Medical Center*, June 16, 2006, Seattle, Washington.

128. "Neurobiology of Depression in Epilepsy," at a symposium on the *Behavioral Aspects of Epilepsy*, June 16-17, 2006, Boise, Idaho.
129. Several lectures and workshop leader at the *Kiffin Penry Memorial Epilepsy Mini-fellowship* sponsored by *Wake Forest University School of Medicine*, August 10-12, 2006, Winston-Salem, North Carolina.
130. Faculty, *Pediatric Board Review Course*, UCLA Department of Pediatrics and Mattel Children's Hospital at UCLA, September, 2006, Los Angeles, California.
131. "Mechanisms of Epilepsy and Antiepileptic Therapy," *Cardinal Glennon Children's Hospital, St. Louis University School of Medicine*, Sept 8, 2006, St. Louis, Missouri.
132. "Do Neonatal Seizures Cause Brain Injury?" at the 11th Annual Pediatric Neurosciences Symposium, *Barrow Neurological Institute*, Oct 28, 2006, Phoenix, Arizona.
133. Hot Topics Symposium at the 1st *North American Regional Epilepsy Congress*, Session co-chaired with Dr. Andrey Mazarati, Dec 1, 2006, San Diego, California.
134. Chair, Basic Mechanisms of Epilepsy Special Interest Group, 1st *North American Regional Epilepsy Congress*, Dec 1, 2006, San Diego, California.
135. Speaker at the Neuropharmacology Roundtable, 1st *North American Regional Epilepsy Congress*, Dec 2, 2006, San Diego, California.
136. "New Options for Management of Primary Generalized Tonic Clonic Seizures in Children," at *San Diego Children's Hospital*, March 13, 2007, San Diego, California.
137. "Seizure Mechanisms and AED Pharmacology," Neurology Grand Rounds at *Downstate Medical Center of the State University of New York*, April 27, 2007, Brooklyn, New York.
138. "Generators of Focal and Generalized Seizures – Channels, Networks and AEDs," Neurology Grand Rounds at *University of Cincinnati*, May 2, 2007, Cincinnati, Ohio.
139. "Medically Refractory Epilepsy," at the *Children's Hospital of Cincinnati*, May 2, 2007, Cincinnati, Ohio.
140. "Update on the Pharmacology of Antiepileptic Drugs," at the *Children's Hospital of Columbus*, May 3, 2007, Columbus, Ohio.
141. "Explorations in Translational Epilepsy Research," at the Department of Neurology, *Medical University of South Carolina*, May 21, 2007, Charleston, South Carolina.
142. "Seizures, AEDs, and the Developing Brain," invited presentation at the *NINDS, NIH, Workshop on Neonatal Seizures*, May 31-June 1, 2007, Bethesda, Maryland.
143. "Decision making in Pediatric Epilepsy Therapeutics," at *Children's Hospital of the King's Daughters*, July 27, 2007, Norfolk, Virginia.
144. Faculty, *Pediatric Board Review Course*, UCLA Department of Pediatrics and Mattel Children's Hospital at UCLA, September 4, 2007, Los Angeles, California.
145. "Animal Models in the Development of AEDs," moderated the Basic Science Special Interest Group at the *Annual Meeting of the American Epilepsy Society*, November 30, 2007, Philadelphia, Pennsylvania.

146. "Discontinuation of AEDs – what do we know from animal models?" presentation at the Annual Course at the *Annual Meeting of the American Epilepsy Society*, December 2, 2007, Philadelphia, Pennsylvania.
147. "Comorbidities of Epilepsy: Development of an Animal Model of Epileptogenesis-associated Depression," *Pediatric Neurology Grand Rounds at Texas Children's Hospital – Baylor College of Medicine*, March 12, 2008, Houston, Texas
148. Several lectures and workshop leader at the *Kiffin Penry Memorial Epilepsy Mini-fellowship* sponsored by *Wake Forest University School of Medicine*, March 27-29, 2008, Winston-Salem, North Carolina.
149. "Priorities in Epilepsy Therapy Development," at *Gillette Children's Specialty Hospital*, April 11, 2008, St. Paul, Minnesota.
150. "Investigations in Antiepileptogenic Therapy and the Neurobiology of Depression," at *Children's National Medical Center*, May 15, 2008, Washington DC.
151. "Therapy Targeting Antiepileptogenesis in the Developing Brain," Epilepsy Grand Rounds at *Johns Hopkins University School of Medicine*, May 28, 2008, Baltimore, Maryland.
152. "Rapid Kindling in the Investigation of Antiepileptogenesis and the Mechanism of Depression in Epilepsy," *University of Maryland School of Medicine*, May 28, 2008, Baltimore, Maryland.
153. "Translational Epilepsy Research," Neurology Grand Rounds at *George Washington University School of Medicine*, May 29, 2008, Washington DC.
154. "Pediatric Epilepsy Syndromes – Diagnosis and Management," Neurology Grand Rounds at *Howard University School of Medicine*, May 29, 2008, Washington DC.
155. "Curing Epilepsy 2007 - NINDS Benchmarks for Research: Progress at UCLA" Epilepsy Grand Rounds at the *Neurologic Institute, Cleveland Clinic Educational Foundation*, July 25, 2008, Cleveland, Ohio.
156. "Investigations on Depression in Epilepsy," at the *Behavioral Aspects of Neurological Disease Conference*, October 11, 2008, Sun Valley, Idaho.
157. "Future of Epilepsy Therapeutics: Testing Development-Specific Targets," at a breakfast symposium at the Annual Meeting of the *Child Neurology Society*, November 6, 2008, Santa Clara, California.
158. "Translational Epilepsy Research – Antiepileptogenic Therapeutics and Modeling Comorbidities," Grand Rounds of the Department of Neurology, *State University of New York*, November 12, 2008, Syracuse, New York.
159. "New Treatments for Epilepsy on the Horizon," at the *Advances in the Management of Epilepsy and the Epilepsy Clinic Symposium* at the *Annual Meeting of the American Epilepsy Society*, December 5, 2008, Seattle, Washington.
160. "New Treatments for Pharmacological Treatment of Epilepsy," at the *Advances in the Management of Epilepsy* sponsored by *Wake Forest University School of Medicine* Feb 28, 2009 at Asheville, North Carolina.
161. Several lectures and workshop leader at the *Kiffin Penry Memorial Epilepsy Mini-fellowship* sponsored by *Wake Forest University School of Medicine*, May 21-22, 2009, Winston-Salem, North Carolina.
162. "Studies in Antiepileptogenic Therapeutics During Development and Modeling Comorbidities," *Division of Pediatric Neurology, University of Wisconsin*, May 29, 2009, Madison, Wisconsin.

163. "Studies in Antiepileptogenic Therapeutics During Development and Modeling Comorbidities," *Children's Hospital of Milwaukee, Medical College of Wisconsin*, May 29, 2009, Milwaukee, Wisconsin.
164. "Psychiatric Comorbidity of Neurologic Disease: Modeling Depression in Epilepsy," Epilepsy Grand Rounds at the *University Hospital*, Department of Neurology, *Case Western Reserve University School of Medicine*, November 9, 2009, Cleveland, Ohio.
165. "Advances in Pediatric Epilepsy Therapy," at the Children's Neurology Practice Group, *San Diego Children's Hospital*, April 13, 2010, San Diego, California.
166. "Extending the Eric Lothman Legacy: Rapid Kindling in Developmental Epilepsy Research," Epilepsy Grand Rounds and ***Eric Lothman Memorial Lecture*** at the Department of Neurology at the *University of Virginia*, April 23, 2010, Charlottesville, Virginia.
167. "Modeling Depression associated with Epilepsy in the Laboratory," at the Department of Neurology, *Phoenix Children's Hospital*, May 12, 2010, Phoenix, Arizona.
168. "Psychiatric Comorbidity of Neurologic Disease: Modeling Depression in Epilepsy," Neurology Grand Rounds at the School of Medicine, *University of Colorado*, July 28, 2010, Denver, Colorado.
169. "An Update on New Anticonvulsants for Use in Children with Epilepsy," at the weekly educational conference in pediatric neurology, *University of Colorado*, July 30, 2010, Denver, Colorado.
170. "Depression in Epilepsy: Explorations into the Neurobiological Connection," Neurology Grand Rounds at the *Mayo Clinic*, June 27, 2011, Rochester, Minnesota.
171. "Challenges in Pediatric epilepsy management," at the *First New Horizons in Neuroscience Symposium at the Texas Tech University Health Sciences Center*, March 10, 2012, Lubbock, Texas.
172. "Depression and Epilepsy: How are they Connected?" at the *Children's Medical Center – University of Texas Southwestern Medical Center*, May 23, 2012, Dallas, Texas.
173. "Anticonvulsant Drugs and the Neonatal Brain," at the *Focus on Epilepsy at Dell Children's Hospital*, July 7, 2012, Austin, Texas.
174. "Neurobiology of Depression in Epilepsy," Neurology Grand Rounds at *University of Texas San Antonio*, August 17, 2012, San Antonio, TX.
175. "New Options for Treating Lennox-Gastaut Syndrome," *Oakland Children's Hospital*, August 22, 2012, Oakland, California.
176. "Choices in Treatment of Epilepsy with Modulation of GABA signaling," presentation at the annual meeting of the *Epilepsy Foundation of Georgia Professional Advisory Board Meeting*, November 5, 2012, Atlanta, Georgia.
177. "Neurobiology of Depression in Epilepsy," Neurology Grand Rounds at *Detroit Medical Center, Wayne State University School of Medicine*, November 16, 2012, Detroit, Michigan.
178. Speaker at the Neonatal Seizures Special Interest Group at the *Annual Meeting of the American Epilepsy Society*, December 3, 2012, San Diego, California.
179. "Quest for Neuroprotective and Antiepileptogenic Therapy," at the Pediatric Neurology Grand Rounds at the *Primary Children's Medical Center, University of Utah School of Medicine*, March 6, 2013, Salt Lake City, Utah.

180. "New AEDs: What Advances do they bring to Therapeutics?" at the *7th Annual Greater Mid-South Pediatric Neurology Update by the Neuroscience Institute of Le Bonheur Children's Hospital*, May 3, 2013, Memphis, Tennessee.
181. "Epilepsy and Co-morbidities – Exploring the Connection," at the *7th Annual Greater Mid-South Pediatric Neurology Update by the Neuroscience Institute of Le Bonheur Children's Hospital*, May 4, 2013, Memphis, Tennessee.
182. "Biomarkers for Epileptogenesis & Comorbidities," Introductory presentation in a Scientific Symposium (Chair: Raman Sankar), *Annual Meeting of the American Epilepsy Society*, December 10, 2013, Washington DC.
183. "Viral Infections during Pregnancy: A Risk Factor for Autism and Epilepsy in the Offspring?" at the *Annual Meeting of the American Academy of Neurology*, May 1, 2014, Philadelphia, PA.
184. "Medical issues After Hemispherectomy: Headaches & Migraines," at the *Annual Meeting of the Hemispherectomy Foundation and the Brain Recovery Project*, July 12, 2014, Anaheim, CA.
185. "Headaches and Migraines," "Febrile Convulsions Update," "Benign Childhood Epilepsy Syndromes," "Sports Concussion Evaluation and Management," "Evaluation of the Child with Developmental Delay," and "Seminar on Refractory Epilepsy," at the *Aloha Update: Pediatrics 2014* (sponsored by the *Children's Hospital of Los Angeles* and the *American Academy of Pediatrics*, October 18-24, 2014, Kauai, HI.
186. "Updates on AED Options," at the *Advances in the Management of Epilepsy* course for CME at the *Annual Meeting of the American Epilepsy Society*, December 4, 2015, Philadelphia, Pennsylvania.
187. "New AEDs: Are They Really Different?" at the *Seattle Children's Hospital-University of Washington School of Medicine*, February 16, 2016, Seattle, Washington.
188. "Comorbidities in Epilepsy: Research on Mechanisms," at the Grand Rounds of the Department of Neurology, *Duke University School of Medicine*, April 27, 2016, Durham, North Carolina.
189. "Mechanistic Basis for Rational Therapy & Polypharmacy in Epilepsy," in *Rational Polytherapy as an Epilepsy Treatment Strategy* at the *Annual Meeting of the American Epilepsy Society*, December 2, 2016, Houston, Texas.
190. "Neurogenetics, Physiology and Pharmacology of a Pediatric Epilepsy Syndrome," at the Grand Rounds of the *Department of Pediatrics, Beaumont Hospital*, August 2, 2018, Royal Oak, Michigan.
191. "Precision Medicine for Epilepsy: Opportunities and Challenges," at the *Epilepsy Grand Rounds, Cleveland Clinic Foundation*, August 10, 2018, Cleveland, Ohio.
192. "A Benign Pediatric Epilepsy Syndrome and its Atypical Evolution: Neurogenetics and Therapeutics," a lecture to Epilepsy Fellows and faculty at the *Cleveland Clinic Foundation*, August 10, 2018, Cleveland, Ohio.
193. "Epilepsy, Depression and ADHD: Central Role of Neurotransmitter Systems," presentation at an Investigators Workshop on "Melancholia in Epilepsy" – *Annual Meeting of the American Epilepsy Society*, December 8, 2019, Baltimore, Maryland.
194. "Genetic Testing in Pediatric Epilepsy – Advancing Patient Care," Neurology Grand Rounds at the *School of Medicine at State University of New York (SUNY) Downstate*, May 22, 2020, Brooklyn, NY (Zoom presentation during Covid Lockdown).
195. "Cannabidiol: Mechanism of Action to Clinical Use," at Pediatric Epilepsy: New and Novel Approaches to Care, Live Virtual Conference at *Beaumont Children's Hospital*, August 13-14, 2021, Royal Oak, Michigan.

196. "Status Epilepticus – Urgency to Treat and Emerging Drug Treatments," at Pediatric Epilepsy: New and Novel Approaches to Care, Live Virtual Conference at *Beaumont Children's Hospital*, August 13-14, 2021, Royal Oak, Michigan.
197. "Neurogenetics and Neuropharmacology of a Pediatric Epilepsy Syndrome," *Children's Hospital of Omaha*, University of Nebraska, November 11, 2021, Omaha, Nebraska.
198. "Mechanisms of Action of Anti-Seizure Medications," virtual program at *Cohen Children's Hospital of the Long Island Jewish Medical Center*, January 14, 2022, New Hyde Park, New York.
199. "Emerging Targets for Antiseizure Medications," at *Johns Hopkins All Children's Hospital (and Johns Hopkins, Baltimore by Zoom)*, March 10, 2023, Tampa, Florida.
200. "Established and Emerging Targets for Antiseizure Therapeutics," virtual program - Neuroscience Grand Rounds at Banner Health System - University of Arizona, March 24, Virtual
201. "Established and Emerging Targets for Antiseizure Therapeutics," Neuroscience Grand Rounds at *Beaumont Health System*, March 29, 2023, Royal Oak, Michigan.
202. "Classical and Emerging Targets for Antiseizure Medication Activity," Lecture during Visiting Professorship at *Virginia Commonwealth University* School of Medicine, Department of Neurology, June 21, 2023, Richmond, Virginia.

Regional:

1. "Diagnosis and Management of Seizures in the Pediatric Patient," presented to the pediatric staff at *Southern California Permanente Medical Group*, May 13, 1992, Los Angeles, California.
2. "Status Epilepticus in the Pediatric Patient," Grand Rounds of the Department of Pediatrics, *UCLA School of Medicine*, August 7, 1992, Los Angeles, California.
3. "Cortical Microdysgenesis: Evolution of Changes in the White Matter Signal in the MRI with Development in Children with Infantile Spasms" and "Amino acid Neurotransmitters in Status Epilepticus in the Developing Rat Brain" - Presentation to the Neurology Department at the *Children's Hospital of Los Angeles*, June 22, 1993, Los Angeles, California.
4. "Pediatric Epileptic Syndromes" presented to the combined pediatric medical staff of Santa Monica Hospital and St. John's Medical Center, Santa Monica, at *Santa Monica Hospital*, June 23, 1993, Santa Monica, California.
5. "Diagnosis and Management of Headaches" - Pediatric Grand Rounds, *Olive View Medical Center*, July 21, 1993, Sylmar, California.
6. "Neuromuscular Diseases in Infants and Children" - presented to the pediatric staff at *Southern California Permanente Medical Group* on August 18, 1993, Los Angeles, California.
7. "Pediatric Epileptic Syndromes" and "Clinical Research Vignette on Infantile Spasms," Grand Rounds of the Department of Pediatrics, *UCLA School of Medicine*, August 20, 1993, Los Angeles, California.
8. "Pediatric Experience with Felbamate, a New Anticonvulsant," one of the several presentations on Felbamate at the Grand Rounds of the Department of Neurology, *UCLA School of Medicine*, October 6, 1993, Los Angeles, California.

9. "Developmental Changes in Amino Acid Neurotransmitters in the Rat caused by Status Epilepticus," one of the several presenters on "Issues in Pediatric Epilepsy" at the Grand Rounds of the Department of Neurology, *UCLA School of Medicine*, October 1993, Los Angeles, California.
10. "Epilepsy in Infants, Children, and Adolescents," Grand Rounds of the Department of Pediatrics, *White Memorial Hospital*, December 2, 1993, Los Angeles, California.
11. "Seizure Disorders," presented to the medical staff of *La Palma Intercommunity Hospital*, February 28, 1994, La Palma, California.
12. Workshop on "Common Neurologic Problems in the Adolescent Patient" - *Society for Adolescent Medicine*, Annual Meeting, March 16, 1994, Los Angeles, California.
13. "Neurological Aspects of Tuberous Sclerosis," *Medical Education Conference sponsored by the National Tuberous Sclerosis Association*, June 11, 1994, University of California, Los Angeles, Los Angeles, California.
14. "Re-evaluation of Patients with Epilepsy in the Light of Newly Released Antiepileptic Agents," *Daniel Freeman Hospital*, June 24, 1994, Marina del Rey, Los Angeles, California.
15. "Pharmacology of New Anticonvulsants," Department of Neurology, *UCLA School of Medicine*, August 31, 1994, Los Angeles, California.
16. "MRI in Cortical Dysplasia," to Epilepsy/Clinical Neurophysiology fellows, Department of Neurology, *UCLA School of Medicine*, September 8, 1994, Los Angeles, California.
17. "Epilepsy and the Adolescent," Department of Adolescent Medicine, *Children's Hospital of Los Angeles / USC School of Medicine*, September 9, 1994, Los Angeles, California.
18. "Headaches in the Pediatric Patient," presented to the pediatric staff at *Southern California Permanente (Kaiser) Medical Group*, November 16, 1994, Los Angeles, California.
19. "Current Management of Epilepsy," presented to the medical staff at *Pacifica Hospital of the Valley*, November 18, 1994, Sylmar, California.
20. "Anticonvulsant Drugs: Old and New," at *Living Well with Epilepsy: Current Practice, New Hope*, A continuing education seminar for nurses, November 19, 1994, Los Angeles, California.
21. "Update on the Treatment of Epilepsy," presented to the medical staff at *Robert F. Kennedy Medical Center*, January 5, 1995, Hawthorne, California.
22. "Antiepileptic Therapy: Pharmacologic Principles," a continuing education seminar for clinical pharmacists, February 23, 1995, Pasadena, California.
23. "Headaches: Update on the Understanding of Mechanisms and Management," at the Department of Neurology, *Southern California Permanente (Kaiser) Medical Group*, March 1, 1995, Los Angeles, California.
24. "Neuroimaging and the Developing Brain: Detection of Microscopic Cortical Dysplasia," Grand Rounds of the Department of Neurology, *UCLA School of Medicine*, April 5, 1995, Los Angeles, California.
25. "Epilepsy: Classification, Syndromes; Diagnosis," and "Antiepileptic Drugs," at the *UCLA / Parke-Davis Epilepsy Training Seminar* for the Parke-Davis Neuroscience Medical Team, *UCLA Medical Center*, April 11, 1995, Los Angeles, California.
26. "GABA Metabolism, Development, and Status epilepticus," Grand Rounds of the Department of Pediatrics, *UCLA School of Medicine*, April 14, 1995, Los Angeles, California.

27. "Pharmacology of New Anticonvulsants," at *Fairview Developmental Center*, May 10, 1995, Costa Mesa, California.
28. "Pharmacology of New Anticonvulsants," to *Orange County Society of Health-System Pharmacists*, May 10, 1995, Santa Ana, California.
29. "New Generation Anticonvulsants: Pharmacology and Clinical Applications," at *Lanterman Regional Developmental Center*, June 16, 1995, Pomona, California.
30. "An approach to the Diagnosis and Management of Headaches in the Adolescent Patient," at the Department of Adolescent Medicine, *Children's Hospital of Los Angeles*, August 4, 1995, Los Angeles, California.
32. "A Comprehensive Approach to the Classification, Diagnosis, and Management of Seizure Disorders," a continuing education program sponsored by the *Kern County Department of Pediatrics*, September 19, 1995, Bakersfield, California.
33. "Neurotransmitters, Ion Channels, and Antiepileptic Drugs," Grand Rounds at the Department of Neurology, *Harbor - UCLA Medical Center*, December 15, 1995, Torrance, California.
48. "A Review of New Generation Anticonvulsants," Meeting of the *Orange County Neurologic Society*, March 12, 1996, Costa Mesa, California.
49. "Diagnosis and Management of Headaches," Grand Rounds of the Department of Pediatrics, *Olive View - UCLA Medical Center*, March 27, 1996, Sylmar, California.
50. "Carnitine, Inborn Errors of Metabolism, and Valproic acid," at the Department of Neurology, *Kaiser Permanente Medical Center*, October 2, 1996, Los Angeles, California.
38. "Management of Intractable Epilepsy in the Developmentally Disabled Patient," at the *Lanterman Development Center*, December 13, 1996, Pomona, California.
39. "New Treatment Options in Pediatric Epilepsy," at the Merritt-Putnam Epilepsy Speakers Bureau, February 21-23, 1997, Santa Fe, New Mexico.
40. "Classification, Etiology and neurophysiology of Cerebral Palsy," at *Cerebral Palsy*, A conference for neurologists, pediatricians, neurosurgeons, orthopedic surgeons and physical therapists, March 1, 1997, Los Angeles, California.
41. "Carnitine and the Patient with a Seizure Disorder," at the Department of Neurology of the *West Los Angeles VA Hospital*, May 16, 1997, Los Angeles, California.
42. "Disorders of Lipid β -Oxidation: Neurologist's Perspective," at the *Annual Neurology Symposium* for *Kaiser Permanente Medical Group*, September 18, 1997, Pasadena, California.
43. "Seizure-related Brain Damage in the Immature Brain," Neurology Grand Rounds, *Harbor-UCLA Medical Center*, September 19, 1997, Torrance, California.
44. "Epilepsy Treatment Options in Special Populations: The Adolescent Patient," at the *Merritt-Putnam Epilepsy Speakers Bureau*, September 27, 1997, La Jolla, California.
45. "Update on new Antiepileptic Drugs," Continuing Education program for Pharmacists, *Kaiser Permanente Medical Center*, October 7, 1997, West Los Angeles, California.
46. "Diagnosis and Management of Headaches and Head Injuries," at *Nursing Issues in Ambulatory Pediatrics*, October 23, 1997, Anaheim, California.

47. "Pediatric Epilepsy," lecture to medical residents at *White Memorial Hospital*, February 19, 1998, Los Angeles, California.
48. "Basic and Clinical Pharmacology of Antiepileptic Drugs," at the Department of Neurology, *Children's Hospital of Los Angeles*, February 24, 1998, Los Angeles, California.
49. "Classification and Etiology of Cerebral Palsy," at the 2nd Annual Margaret Jones Conference, *Recent Advances in the Treatment of Cerebral Palsy, UCLA Medical Center*, March 28, 1998, Los Angeles, California.
50. "Classification, Etiology, and Emerging Approaches to Prevention, of Cerebral Palsy," at the *Advanced Perinatal/Neonatal Nursing Conference*, May 14, 1998, Oxnard, California.
51. "Status Epilepticus in Children," Grand Rounds at *Miller Children's Hospital/Long Beach Memorial Hospital*, May 15, 1998, Long Beach, California.
52. "Evaluation and Management of a Child with a Seizure Disorder," Grand Rounds of the *Department of Family Medicine*, UCLA School of Medicine, June 19, 1998, Los Angeles, California.
53. "Mechanism of Action of New Antiepileptic Drugs," and "Alternative Therapies: The Ketogenic Diet," at the *Merritt-Putnam Lectures on Epilepsy*, July 11, 1998, Santa Monica, California. Also chaired this half-day program with a nationally selected faculty.
54. "Perspectives on Neurontin® In Epileptic Syndromes of Infancy and Childhood," *Merritt-Putnam Speaker's Bureau Meeting*, October 3, 1998, Palm Springs, California.
55. "Advances in Epilepsy Therapy: A Pediatric Perspective," at the *Annual Meeting of the California Society of Health System Pharmacists*, October 22-25, 1998, Anaheim, California.
56. "Epilepsy Surgery: Indications in Adults and Children," at the *Merritt-Putnam Lectures on Epilepsy*, Oct 31, 1998, Costa Mesa, California.
57. "Recent Advances in the Treatment of Seizure Disorders," at Neurology Grand Rounds, *Cottage Hospital*, November 23, 1998, Santa Barbara, California.
58. "Treatment of Seizure Disorders in Children and Young Adults," at *Family Medicine Grand Rounds*, Loma Linda University, December 4, 1998, Loma Linda, California.
59. "Pediatric Epilepsy Syndromes and their Treatment," *Department of Pediatrics, Cedars Sinai Medical Center*, January 8, 1999, Los Angeles, California.
60. "The Ups and Downs of Epilepsy Treatments for Children," *Epilepsy Education Conference* sponsored by the Epilepsy Foundation, March 27, 1999, Anaheim, California.
61. "Advances in the Treatment of Epilepsy," *Department of Family Medicine, Arrowhead Regional Medical Center*, April 6, 1999, San Bernardino, California.
62. "Genetics of Epilepsy" and "Comprehensive Review of Anticonvulsants" for a *Ortho-McNeil Pharmaceutical Company* sponsored preceptorship for West Coast sales personnel, held at UCLA, April 7 and 8, 1999, Los Angeles, California.
63. "Metabolic and Neurodegenerative Disorders," Neurology Grand Rounds, *Harbor-UCLA Medical Center*, May 21, 1999, Torrance, California.
64. "Treatment of Epilepsy: Considerations in the Management of Special Populations," at the *First Annual Western CNS Conference* of the Institute of Medical Studies, July 18, 1999, Yosemite National Park, California.

65. "Epilepsy 2000: New understandings and Emerging Treatments," Pediatric Grand Rounds, *UCLA School of Medicine*, February 11, 2000, Los Angeles, California.
66. "Advances in the Diagnosis and Treatment of Epilepsy, 2000," Family Medicine Grand Rounds, *Loma Linda University school of Medicine*, March 10, 2000, Loma Linda, California.
67. "Epilepsy at the Turn of the Millennium: Advances in Therapeutics," Grand Rounds, Department of Neurology, *UCLA School of Medicine*, May 10, 2000, Los Angeles, California.
68. "Status Epilepticus in the Pediatric Patient," Pediatric Grand Rounds, *UCLA School of Medicine*, August 11, 2000, Los Angeles, California.
69. "Floppy Infant, Stumbling Child: Weakness in the Pediatric patient," Pediatric Grand Rounds, *UCLA School of Medicine*, January 5, 2001, Los Angeles, California.
70. "Seizures in the Developing Brain: Injury, Neurogenesis, and Plasticity," Grand Rounds of the Department of Neurology, *UCLA School of Medicine*, January 31, 2001, Los Angeles, California.
71. "Seizure-Induced Epileptogenicity in the Developing Brain," Grand Rounds of the Department of Neurology, *Keck School of Medicine, University of Southern California*, February 6, 2001, Los Angeles, California.
72. "GABA, Glycine, and Animal Models of Epilepsy," Lecture to Neurology Resident Lecture Series, Department of Neurology, *UCLA School of Medicine*, February 7, 2001, Los Angeles, California.
73. "Neonatal Seizures: Scientific and Clinical Challenges in Management," to the Division of Neonatology, Department of Pediatrics, *UCLA School of Medicine*, March 27, 2001, Los Angeles, California.
74. "Impact of Seizures on the Immature Brain," to the Division of Pediatric Neurology, *Children's Hospital of Los Angeles*, March 27, 2001, Los Angeles, California.
75. "Pediatric Seizure Syndromes," and "Pharmacology of Antiepileptic Drugs," *VA Greater Los Angeles Healthcare System, West Los Angeles VA Hospital*, March 30, 2001, Los Angeles, California.
76. "Drug Selection in Gender- and Age-Based Population: Pediatric Patients," in "A Contemporary Assessment of seizures and Epilepsy." *A Merritt-Putnam Lecture Series sponsored by the American Epilepsy Society*. Program developed by Drury I, Ferrendelli JA, Ramsay RE and Sankar R. Also chaired the program on: September 29, 2001, Los Angeles, California.
77. "A Contemporary Look at Pediatric Epilepsy Syndromes," Neurology Grand Rounds, *Harbor-UCLA Medical Center*, May 10, 2002.
78. "Epilepsy in Childhood and Adolescence," in a Symposium on *Women and Epilepsy for Healthcare Professionals*, June 29, 2002, Santa Monica, California.
79. "Brain Injury from Seizures in the Developing Brain," at the *UC Neurotrauma Meeting*, Aug 8, 2002, Silverado Resort, Napa, California.
80. "Neurochemistry of GABA and Glycine," "Animal Models of Epilepsy," and "A Neurologist's Approach to Metabolic Disease," Lectures to Neurology Resident Lecture Series, Department of Neurology, *UCLA School of Medicine*, December 4, 2002, Los Angeles, California.
81. "Through the Glass, Darkly: Reflections & Observations on My Decade at UCLA Caring for Children with Epilepsy," **Tenth Annual William F. Friedman Lecture**, *Mattel Children's Hospital and David Geffen School of Medicine at UCLA*, January 24, 2003, Los Angeles, California.

82. "Migraines and Epilepsy – What is the Relationship?" Grand Rounds presentation at the Neurology Grand Rounds at the *West Los Angeles Veterans Administration Hospital*, January 31, 2003, Los Angeles, California.
83. "A Young Boy with Medically Refractory Epilepsy," Clinico-Pathological Conference, Neurology Grand Rounds, *David Geffen School of Medicine at UCLA*, March 5, 2003, Los Angeles, California.
84. "Antiepileptic Drugs, Ion Channels, and Other Targets for Anticonvulsant Action," Lecture given as part of Neurology Resident Lecture Series, Department of Neurology, *David Geffen School of Medicine at UCLA*, August 27, 2003, Los Angeles, California.
85. "Hypotonia and Weakness" and "Ataxia, Congenital Malformations, Neurocutaneous Syndromes," lectures at the *UCLA Pediatric Board Review*, September 9, 2003, Los Angeles, California.
86. "Markers and Consequences of Status Epilepticus in the Immature Brain," Neurology Grand Rounds, *David Geffen School of Medicine at UCLA*, September 10, 2003, Los Angeles, California.
87. "Pharmacologic Basis of Action of Antiepileptic Drugs," Neurology Grand Rounds at *Harbor-UCLA Medical Center*, November 21, 2003, Torrance, California.
88. "Diagnosis and Treatment of Epilepsy: The Science and The Art," Pediatric Grand Rounds at *Mattel Children's Hospital and David Geffen School of Medicine at UCLA*, February 13, 2004, Los Angeles, California.
89. "Metabolic Disease – A Neurologist's Perspective," Lecture given as part of Neurology Resident Lecture Series, Department of Neurology, *David Geffen School of Medicine at UCLA*, February 18, 2004, Los Angeles, California.
90. "Pediatric Epilepsy: An Update for the Primary Care Physician," Grand Rounds of the Department of Pediatrics, *Huntington Memorial Hospital*, March 24, 2004, Pasadena, California.
91. "Floppy Infant, Stumbling Child," Lecture to pediatric residents at *Mattel Children's Hospital and David Geffen School of Medicine at UCLA*, May 26, 2004, Los Angeles, California.
92. "Headaches and Migraines in Children," Grand Rounds, Department of Pediatrics at the *West Los Angeles Kaiser Permanente Medical Group*, May 29, 2004, Los Angeles, California.
93. "Neonatal Seizures: A Bench to Bedside Dilemma," at *Mattel Children's Hospital and David Geffen School of Medicine at UCLA*, July 29, 2004, Los Angeles, California.
94. "Medically Refractory Epilepsy of Childhood – the Role of Ketogenic Diet," Pediatric Grand Rounds at the *Children's Hospital of Orange County*, August 18, 2004, Orange, California.
95. "Pediatric Epilepsy," and "Treatment of Childhood Headaches and Migraines," Lectures to Pediatric Medical Staff at *Southern California Kaiser Permanente Medical Center*, August 18, 2004, Los Angeles, California.
96. "Hypotonia and Weakness" and "Ataxia, Congenital Malformations, Neurocutaneous Syndromes," lectures at the *UCLA Pediatric Board Review*, September 7, 2004, Los Angeles, California.
97. "A Child with Seizures: A Problem for the Neurologist," at the Pediatric Neurosurgery Symposium: Bridging the Gap Between Clinicians and Surgeons," *Mattel Children's Hospital and David Geffen School of Medicine at UCLA*, September 18, 2004, Los Angeles, California.
98. "Pathophysiology of Seizures," Lecture given to the Division of Critical Care at *Mattel Children's Hospital and David Geffen School of Medicine at UCLA*, September 22, 2004, Los Angeles, California.

99. "Overview of Pediatric Epilepsy," Presentation to pediatric house-staff at *Mattel Children's Hospital and David Geffen School of Medicine at UCLA*, September 28, 2004, Los Angeles, California.
100. "Neonatal Seizures: Lessons from the Laboratory," at the Second California Pediatric Neurology Colloquium at *Children's Hospital and Health Center*, October 30, 2004, San Diego, California.
101. "Pediatric Epilepsy," Grand Rounds, Department of Pediatrics at the *West Los Angeles Kaiser Permanente Medical Group*, Jan 19, 2005, Los Angeles, California.
102. "Pediatric Epilepsy Syndromes," at Pediatric Grand Rounds at *Olive View Medical Center*, Jan 19, 2005, Sylmar, California.
103. "Novel Molecular Targets for Antiepileptic Drugs: Implications for Polypharmacy," Neurology Grand Rounds of the *UCSD School of Medicine at the Veteran's Administration Hospital*, February 25, 2005, La Jolla, California.
104. "Epilepsy Syndromes of Infancy, Childhood and Adolescence," at the *Kaiser Permanente Medical Center*, March 4, 2005, Panorama City, California.
105. "Traditional and Novel Molecular Targets of Antiepileptic Drug Action," presentation to neurology house-staff and medical students at David Geffen School of Medicine at UCLA, June 15, 2005, Los Angeles, California.
106. "Pediatric Epilepsy: Diagnosis and Treatment," to the attending faculty physicians of the General Pediatric Services at the *Children's Hospital of Los Angeles*, July 25, 2005, Los Angeles, California.
107. "Optimization of Therapy in Pediatric Epilepsy," to pediatrics house staff and faculty at *Miller Children's Hospital at Long Beach Memorial Medical Center*, August 5, 2005, Long Beach, California.
108. "Hypotonia and Weakness" and "Ataxia, Congenital Malformations, Neurocutaneous Syndromes," lectures at the *UCLA Pediatric Board Review*, September 12, 2005, Los Angeles, California.
109. "Approach to the Floppy Infant and the Stumbling Child," Pediatric Grand Rounds at *Mattel Children's Hospital and David Geffen School of Medicine at UCLA*, September 16, 2005, Los Angeles, California.
110. "Mechanisms of Epileptogenesis and AED Mechanisms," Neurology Conference at *Harbor UCLA Medical Center*, September 21, 2005, Torrance, California.
111. "The Floppy Infant," "Headaches in the Pediatric Patient," "The Ataxic Child," and "Neurotherapeutics of Epilepsy and Migraine," at the *Current Advances in Pediatrics* course sponsored by the *California Chapter 4 of the American Academy of Pediatrics*, October 21-23, Irvine, California.
112. "Emerging Concepts in the Management of Epilepsy," dinner presentation to neurologists from the *Southern California Kaiser Permanente Medical Group (Baldwin Park, Pasadena and Sunset)*, November 28, 2005, Pasadena, California.
113. "Mechanisms of Epileptogenesis as a Basis for the Design of Rational Therapies," Neuroscience Grand Rounds at the *Cedars-Sinai Medical Center*, February 8, 2005, Los Angeles, California.
114. "Neonatal Seizures: Pathophysiology and Management," Neonatology Lecture Series, *Mattel Children's Hospital at UCLA*, February 28, 2006, Los Angeles, California.
115. "Seizure Mechanisms and Antiepileptic Drug Mechanisms," Neuroscience Grand Rounds at *Loma Linda University School of Medicine*, August 28, 2006, Loma Linda, California.

116. "Hypotonia and Weakness" and "Ataxia, Congenital Malformations, Neurocutaneous Syndromes," lectures at the *UCLA Pediatric Board Review*, September 6, 2006, Los Angeles, California.
117. "Neonatal Seizures: Translational Gap in Management," Pediatric Grand Rounds at *Mattel Children's Hospital at UCLA*, December 15, 2006, Los Angeles, California.
118. "Evidence-based Treatment Plan for Pediatric Epilepsy," *San Diego Children's Hospital*, March 13, 2007, San Diego, California.
119. "Treatment of Epilepsy," in a case-based Regional CME Program sponsored by the *American Epilepsy Society*, March 24, 2007, Los Angeles, California.
120. "What is New in Pediatric Epilepsy," Grand Rounds at the *Children's Hospital of Los Angeles*, August 17, 2007, Los Angeles, California.
121. "Hypotonia and Weakness" and "Ataxia, Congenital Malformations, Neurocutaneous Syndromes," lectures at the *UCLA Pediatric Board Review*, September 4, 2007, Los Angeles, California.
122. Faculty, *Pediatric Update, The 64th Brennmann Lectures of the Los Angeles Pediatric Society*, September 7-9, 2007, Anaheim, California.
123. "NICU Neurology Consult: Neonatal Seizures," at the Academic Day for Neonatologists of Southern California, November 8, 2007.
124. "Headaches and Migraines," "Cerebral Palsy," "Evaluation of the Floppy Infant," and "Pediatric Epilepsy Workshop" at the Clinical Pediatrics program of the *American Academy of Pediatrics, California, Chapter 2*, February 15-17, 2008, Palm Springs, California.
125. "Hypotonia and Weakness" and "Ataxia, Congenital Malformations, Neurocutaneous Syndromes," lectures at the *UCLA Pediatric Board Review*, September 3, 2008, Los Angeles, California.
126. "Short and Long Term Sequelae of Hypoxic-Ischemic Encephalopathy," at a *Symposium on Hypoxic-ischemic Encephalopathy, Mattel Children's Hospital at Ronald Reagan UCLA Medical Center*, September 18, 2008, Los Angeles, California.
127. "Advances in the Diagnosis and Prevention of Brain Injury," at the *15th Annual Conference of the California Association of Neonatologists and AAP District IX Section on Perinatal Pediatrics on Current Topics and Controversies in Perinatal and Neonatal Medicine*, March 7, 2009, Coronado, California.
128. "Hypotonia and Weakness" and "Ataxia, Congenital Malformations, Neurocutaneous Syndromes," lectures at the *UCLA Pediatric Board Review*, September 9, 2009, Los Angeles, California.
129. "Infant presenting with Congenital Hypotonia," Clinico-pathological Conference (CPC), Division of Neonatology, Department of Pediatrics, October 6, 2009, *David Geffen School of Medicine at UCLA*, Los Angeles, California.
130. "Psychiatric Comorbidity of Neurologic Disease: Modeling Depression in Epilepsy," Neurology Grand Rounds, Department of Neurology, *David Geffen School of Medicine at UCLA*, October 7, 2009, Los Angeles, California.
131. "Neuroprotection in Clinical Pediatric Practice," Pediatric Grand Rounds at *Harbor-UCLA Medical Center*, May 27, 2010, Torrance, California.
132. "Depression in Epilepsy: the Neurobiological Connection," Neurology Grand Rounds at the *Keck School of Medicine at the University of Southern California*, June 8, 2010, Los Angeles, California.

133. "Hypotonia and Weakness" and "Ataxia, Congenital Malformations, Neurocutaneous Syndromes," lectures at the *UCLA Pediatric Board Review*, September 8, 2010, Los Angeles, California.
134. "Common Childhood Seizure Syndromes: Recognition and Management," Pediatric Grand Rounds at *Miller Children's Hospital*, September 10, 2010, Long Beach, California.
135. "Depression in Epilepsy – the Neurobiological Connection," Neurology Grand Rounds at the *Cedars-Sinai Medical Center*, June 3, 2011, Los Angeles, California.
136. "Hypotonia and Weakness" and "Ataxia, Congenital Malformations, Neurocutaneous Syndromes," lectures at the *UCLA Pediatric Board Review*, September 7, 2011, Los Angeles, California.
137. "Hypotonia and Weakness" and "Ataxia, Congenital Malformations, Neurocutaneous Syndromes," lectures at the *UCLA Pediatric Board Review*, September 7, 2012, Santa Monica, California.
138. "What is New with Drugs for Pediatric Epilepsy?" at the *Paul H. Crandall Symposium on Epilepsy Surgery* at the Faculty Center, UCLA, September 20, 2012, Los Angeles, California.
139. "Evaluation and Management of Altered Mental status in the Pediatric Patient," at the Annual Pediatric Symposium of *Kaiser Permanente Medical Group*, November 2, 2012, Long Beach, California.
140. "Pediatric Epilepsy: A Syndrome-based Approach," Grand Rounds at the *Children's Hospital of Los Angeles*, November 9, 2012, Los Angeles, California.
141. "Inflammation, Epilepsy, and the Development of Comorbidities," at the Neurology Grand Rounds of the *University of California Irvine School of Medicine* held at the *UC Irvine Douglas Hospital*, January 18, 2013, Orange, California.
142. "Hypotonia and Weakness" and "Ataxia, Congenital Malformations, Neurocutaneous Syndromes," lectures at the *UCLA Pediatric Board Review*, September 3, 2013, Los Angeles, California.
143. "Hypotonia and Weakness" and "Ataxia, Congenital Malformations, Neurocutaneous Syndromes," lectures at the *UCLA Pediatric Board Review*, September 5, 2014, Marina del Rey, California.
144. "Epilepsy and Comorbidities: The Neurobiological Connection," special lecture at the *Children's Hospital of Los Angeles*, June 22, 2015, Los Angeles, California.
145. "Hypotonia and Weakness" and "Ataxia, Congenital Malformations, Neurocutaneous Syndromes," lectures at the *UCLA Pediatric Board Review*, September 11, 2015, Marina del Rey, California.
146. "Comorbidities in Epilepsy: Research on Mechanisms," Neurology Grand Rounds at the *Harbor-UCLA Medical Center*, May 20, 2016, Torrance, California.
147. "Precision Medicine for Epilepsy: Opportunities and Challenges," **William F. Friedman Lecture**, *Mattel Children's Hospital and David Geffen School of Medicine at UCLA*, January 13, 2017, Los Angeles, California.
148. "Subunit-selective Benzodiazepine Actions: Relevance to Therapeutics," Neurology Grand Rounds at the *VA Greater Los Angeles Healthcare System*, January 13, 2017, Los Angeles, California.
149. "Genetics and Epilepsy: Opportunities and Challenges for Precision Medicine," **Edward Senz Lecture** at the *UCSF Benioff Children's Hospital*, November 27, 2018, Oakland, California.
150. "Recognizing the Role of AMPA Receptor Signaling in Epilepsy and its Therapeutic Potential," Neurology Grand Rounds at the *VA Greater Los Angeles Healthcare System*, December 7, 2018, Los Angeles, California.

151. "Emerging Therapies for SCN1A (Dravet syndrome) Disorders," at the *Day of Dravet: Los Angeles*, organized by the *Dravet Syndrome Foundation*, September 14, 2019, Los Angeles, California.
152. Program Moderator for "CURE Day of Science: Epilepsy Conversations," at the *Children's Hospital of Orange County*, organized by the *Citizens United for Research in Epilepsy (CURE)*, September 21, 2019, Orange, California.
153. "Neurotransmitter Systems in Neurology and Psychiatry," didactic lecture to Neurology Residents at *Cedars Sinai Medical Center*, December 20, 2019, Los Angeles, California.
154. "Neurobiology of Comorbidities of Epilepsy," Neurology Grand Rounds at *Cedars Sinai Medical Center*, December 20, 2019, Los Angeles, California.
155. "Endocannabinoids and Cannabidiol in Epilepsy," Neurology Grand Rounds at Loma Linda Medical Center, March 9, 2022, Loma Linda, California.
156. "New Classification of Pediatric Epilepsy Syndromes," didactic lecture to Neurology Residents at Cedars Sinai Medical Center, January 13, 2023, Los Angeles, California.

September 15, 2023