

UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE PATENT TRIAL AND APPEAL BOARD

MARINUS PHARMACEUTICALS, INC.,
Petitioner

v.

OVID THERAPEUTICS, INC.,
Patent Owner

Case PGR2023-00020
Patent 11,395,817

DECLARATION OF DR. MICHAEL A. ROGAWSKI

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I, Michael A. Rogawski, declare as follows:

I. INTRODUCTION

1. I am being offered as an expert on behalf of Petitioner Marinus Pharmaceuticals, Inc. (“Marinus”) to provide opinions and testimony in support of Marinus’s Petition for Post-Grant Review of U.S. Patent No. 11,395,817.

II. QUALIFICATIONS AND PROFESSIONAL EXPERIENCE

2. A copy of my CV, which describes my qualifications, is attached as Exhibit A to this declaration.

3. I am currently a professor in the Department of Neurology of the School of Medicine at the University of California, Davis (“UC Davis”). I also hold the appointment of professor in the Department of Pharmacology of the School of Medicine. Prior to my appointment at UC Davis in January 2007, I served as senior investigator and chief of the Epilepsy Research Section at the National Institute of Neurological Disorders and Stroke. In July 2021, UC Davis awarded me the honorary title of Distinguished Professor.

4. I am also a clinical neurologist at UC Davis Health and I see patients suffering from neurological disorders, including epilepsy, at the UC Davis Medical Group’s Midtown Clinic in Sacramento.

5. I received my M.D. from Yale University School of Medicine in 1980. I also received a Ph.D. in pharmacology from Yale University in 1980.

6. I completed an internship in internal medicine at Yale University School of Medicine from 1980–81. I then did my residency and fellowship in neurology at The Johns Hopkins Hospital from 1982–85.

7. After residency, I was a senior staff fellow at the National Institute of Health from 1986–89. I was a member of the Active Medical Staff, Clinical Center at the National Institute of Health from 1987–2006.

8. During my entire career as a neurologist, I have been interested in the study of seizure disorders and their treatment.

9. More specifically, my research interests include cellular neurophysiological studies of ion channels as they relate to the mechanisms of action of antiseizure medications; the use of animal models for the development of new therapies for the treatment of neurological conditions (including seizures and status epilepticus); and clinical studies of new treatments for seizures and epilepsy.

10. My work on AMPA receptors and neurosteroids have led to new treatment approaches for seizures and epilepsy, and to Zulresso® (brexanolone), which is an FDA-approved treatment for postpartum depression.

11. During my career, I have authored more than 170 peer-reviewed articles; nearly 100 reviews, book chapters, and books; and have been granted 8 patents by the United States Patent and Trademark Office, most of which relate to the treatment of neurological disorders or the development of pharmaceuticals to treat neurological disorders, including epilepsy and seizures. My CV contains a comprehensive list of peer-reviewed articles, book chapters, invited lectures, scientific presentations, and courses taught that demonstrate my expertise in research on treatments for neurological conditions, including epilepsy, seizures, and status epilepticus.

12. I have received a number of awards over the course of my career. I provide a list of those awards in my CV (attached as Exhibit A). I highlight several here. I have received the NIH Director's Award, the Epilepsy Research Award for Outstanding Contributions to the Pharmacology of Antiepileptic Drugs from the American Society for Pharmacology and Experimental Therapeutics, and the Service Award from the American Epilepsy Society. In October 2018, the American Association for the Advancement of Science elected me as a Fellow in the Neuroscience Section for my contributions in the fields of neuroscience, neuropharmacology, and neurology, particularly related to the treatment of epilepsy.

13. I have also been elected to full membership in Sigma Xi, which is a scientific research honor society. I have also been elected as a fellow of the American Neurological Association, the American Epilepsy Society, and the National Academy of Inventors.

14. I have served on the editorial boards of a number of scientific journals, including *Molecular Pharmacology*, *Epilepsia*, *Epilepsy Research*, *Synapse*, *CNS Drug Reviews*, *Current Neuropharmacology*, *Pharmacology & Therapeutics*, *BMC Pharmacology*, *Cellular and Molecular Neurobiology*, and *Nature Scientific Reports*, and was executive editor of *Neuropharmacology* and associate editor of *Neurotherapeutics*. Many of those publish extensively on treatments for neurological disorders, including seizure disorders. I am also one of the founders of *Epilepsy Currents*, which is the journal of the American Epilepsy Society.

15. In the past, I have served on the board of directors of the American Epilepsy Society and have been a member of advisory panels to the National Institutes of Health and the Food and Drug Administration. I was also the president of the American Society for Experimental Neurotherapeutics from 2012 to 2014.

III. TECHNOLOGY BACKGROUND

16. A seizure is defined as an abnormal electrical discharge in the brain that causes alteration in consciousness, sensation, or behavior. (Reddy & Kuruba, Ex. 1004, at 18287.) Millions of people worldwide experience seizures. One of the most common seizure disorders is epilepsy, which is a chronic condition in which patients experience recurrent, unprovoked seizures. (*Id.*)

17. Status epilepticus (abbreviated “SE”) is a neurologic condition in which a patient experiences a prolonged seizure or a series of multiple seizures without recovery in between them. (Reddy & Kuruba, Ex. 1004, at 18287.) Historically, the definition of SE was based on the seizure being “long enough” to cause damage, which was originally defined as 60 minutes and later reduced to 30 minutes. (Seinfeld, Ex. 1005, at 1.)

18. More recently, before the 2016–17 timeframe, it came to be understood that seizures, particularly convulsive seizures (i.e., seizures that involve repeated, rapid and rhythmic contraction of the muscles in the extremities or trunk), that last for 5 minutes or longer are likely to be prolonged and not resolve on their own. (*Id.* at 1–2.) Thus, intervention at the 5-minute mark is appropriate. (*Id.*; Zhang, Ex. 1009, at ¶ [0001].)

19. SE can be caused by many underlying conditions, including stroke, traumatic brain injury, central nervous system infection, alcohol or drug withdrawal, or the cessation of antiseizure medications. (Imtiyaz, Ex. 1006, at 1; Seinfeld, Ex. 1005, at ¶ 2.) It can also occur with no known cause. (Seinfeld, Ex. 1005, at 2.)

20. SE is not a form of epilepsy, although people with epilepsy may experience SE. Specifically, SE is distinct from epileptic conditions like CDKL5 and PCDH19, which are rare types of epilepsy with a genetic basis. Moreover, treatments for conditions like CDKL5 and PCDH19, and indeed most epilepsies, seek to *prevent* the occurrence of seizures, whereas treatments for SE seek to *stop* (*terminate*) the prolonged or repeated seizure condition that the patient is experiencing. (I note that people with CDKL5 and PCDH19 or other forms of epilepsy may also experience SE, in which case they require additional treatment to stop the SE, as is the case generally in SE.)

21. SE, particularly convulsive SE, is a serious condition that constitutes a life-threatening medical emergency and is generally treated in the hospital setting. The cause of the SE, the patient's age, and underlying medical conditions all have an impact on the outcome for the patient. (Seinfeld, Ex. 1005, at 7.) Overall, convulsive SE has a mortality rate from 7%–39%. (*Id.*)

22. SE can be characterized by certain behavioral manifestations evolving over time (also referred to as semiology), which in the case of convulsive status epilepticus includes generalized tonic-clonic movements of the extremities and impaired mental status. In addition, SE has electrophysiological correlates detectable on an electroencephalogram (EEG) recording. (Wylie, Ex. 1017; Trinka et al., Ex. 1018).

23. SE can cause significant damage to the brain, which may be associated with cognitive and other neurological impairments. (Reddy & Kuruba, Ex. 1004, at 18284.) SE may also lead to the development of epilepsy. Currently, the initial therapy for convulsive SE is with a benzodiazepine such as lorazepam, midazolam or diazepam. (Reddy & Kuruba, Ex. 1004, at 18287.) However, the efficacy of benzodiazepines decrease dramatically as the duration of SE increases and, in some cases, there is a complete loss of efficacy. (*Id.*) In most cases, a second drug therapy is administered, such as phenytoin, fosphenytoin, levetiracetam or valproic acid, but the initial and second therapies are not always able to successfully terminate the seizures. (*Id.*)

24. SE in which the seizures fail to stop with such treatment is referred to as refractory SE. (*Id.*) Refractory SE constitutes up to 40% of cases. (*Id.*) There

is a need for treatments that effectively terminate SE, particularly in refractory cases. (Imtiyaz, Ex. 1006, at 1.)

25. Researchers often use animal models to identify treatments that may be effective in terminating SE in humans. When such a potential treatment is identified, it may be advanced to human clinical trials, which are required for regulatory approval before the treatment becomes available for use in medical practice. (Imtiyaz, Ex. 1006, at 1.)

26. In the selection of an animal model to identify treatments for SE, researchers consider the extent to which the model mimics the various clinical features of SE in humans. One desirable characteristic is that the model exhibits seizures that last longer than 5 minutes, so as to satisfy the definition of SE described in ¶ 17 above. An additional desirable characteristic is that the model should reproduce the neuropathological injuries that are observed in specific brain regions of patients who have experienced SE. (Imtiyaz, Ex. 1006, at 1–2.) Finally, if the intent is to identify treatments for refractory SE, the model should simulate the pharmacoresistance to certain antiseizure medications that is characteristic of refractory SE. (*Id.*)

27. One animal model used to evaluate potential treatments for SE is the rat pilocarpine model, which was developed in the 1980s. (Turski, Ex. 1007,

throughout.) The rat pilocarpine model is generally accepted as a model to identify and evaluate potential treatments for SE. The literature consistently explains that “the pilocarpine model of status epilepticus is a well-established, clinically translatable model that satisfies all the criteria for an animal model of status epilepticus.” (Imtiyaz, Ex. 1006, at 1; *see also* Reddy & Kuruba, Ex. 1004, at 18291–2.)

28. In the rat pilocarpine model, rats are treated with a high dose of pilocarpine, which induces convulsive SE (which can also be referred to as CSE). (Reddy & Kuruba, Ex. 1004, at 18292; Turski, Ex. 1007, at 154.) Pilocarpine is typically administered by injection. (*Id.*) Rats may be pretreated with a peripherally acting muscarinic cholinergic antagonist such as scopolamine methylnitrate to limit any peripheral effects of pilocarpine. (*Id.* at 18293.) The pilocarpine, which is a cholinergic agonist, induces convulsive seizures within about 30 minutes, and those seizures persist over time so that the animals are in a state of SE. (*Id.*; Imtiyaz, Ex. 1006, at 2–3.)

29. To allow for continuous EEG recording to assess the absence or presence of electrographic SE, the rats being evaluated in the pilocarpine model may be pre-implanted with electrodes before they are dosed with pilocarpine. (Reddy & Kuruba, Ex. 1004, at 18293.)

30. After its initial development, the rat pilocarpine model underwent further refinements that led to the rat lithium-pilocarpine model. (Imtiyaz, Ex. 1006, at 3.) In that refinement, rats are first dosed with lithium chloride and then with pilocarpine. (*Id.*) In lithium pretreated rats, a lower dose of pilocarpine is required to induce SE, the time to SE onset may also be reduced, and the frequency of occurrence of SE may be increased. (*Id.*) The lithium and pilocarpine treatments generate the same behavioral and electrographic SE as in the pilocarpine model without lithium pretreatment. (*Id.*)

31. It is recognized in the field that the rat pilocarpine model and the related rat lithium-pilocarpine model are both translationally relevant models for identifying treatments for SE, in part because “studies have found similar electrophysiological and morphological abnormalities in the rat hippocampus of the pilocarpine model of SE ... and human SE.” (Imtiyaz, Ex. 1006, at 2 (citing sources).)

32. In common usage, test substances or vehicle are administered at times (typically 15 min, 30 min or 60 min) after the onset of SE in the rat pilocarpine or lithium-pilocarpine SE models while monitoring the behavior of the animal and the EEG power in various frequency bands. If the test substance causes a statistically significant reduction in the EEG power in the frequency bands in relation to

vehicle this indicates that the test substance at the dose administered is effective in treating the SE condition. Alternatively, if the test substance protects, prevents, halts, or blocks convulsive behavioral seizures it is effective in treating the SE condition.

33. I have been involved in studies that have used the rat pilocarpine model to evaluate potential treatments for SE and, in my opinion, it is well-recognized in the art as satisfying the accepted criteria for a model that predicts if a therapy could be useful in the treatment of SE in humans. (*See generally, e.g.,* Kokate, Ex. 1008.)

IV. GANAXOLONE DEVELOPMENT

34. Ganaxolone, a synthetic neurosteroid compound that is a GABA_A receptor positive modulator, is currently under development by Marinus for the treatment of SE. (Zhang, Ex. 1009, ¶¶ [0026]–[0027]; Marinus June 22, 2016, Press Release, Ex. 1022.)

35. Early on, Marinus evaluated various dosage forms of ganaxolone, including oral dosage forms, for the treatment of epilepsy. (Shaw, Ex. 1010, ¶¶ [0002], [00174].) Marinus also measured the blood levels of ganaxolone administered to 6 healthy subjects as an oral suspension, finding that it resulted in a “C_{max} of 37 ± 25 ng/ml and an AUC_(0–24) of 184 ± 104 ng*h/ml.” (*Id.*, ¶ [00546].)

36. Marinus also sought to develop ganaxolone specifically for SE. (April 15, 2016 Press Release, Ex. 1011.) As patients with SE are often unconscious and unable to swallow, oral administration may be difficult or impossible. Therefore, oral dosage forms are not typically used in the treatment of SE. The most common route of administration of drugs used in the treatment of SE is intravenous (IV). Accordingly, in Marinus's research on ganaxolone to treat SE, ganaxolone was administered IV.

37. By 2016, Marinus had done substantial research using the rat lithium-pilocarpine model to assess the efficacy of IV-administered ganaxolone for SE in humans, had published patent applications (*see, e.g.*, Zhang, Ex. 1009), and had also presented the results of its research at various times, including at the 2016 American Academy of Neurology Annual Meeting. (*See, e.g.*, AAN Meeting Brochure, Ex. 1012, at 46, 97, 126; Saporito Poster 1, Ex. 1013, and Saporito Poster 2, Ex. 1014.) The American Academy of Neurology (AAN) is a professional organization, which at year end in 2016, had 26,000 US members, including 14,000 neurologists and 910 researchers. (2016 AAN Annual Report, Ex. 1019, page 19.) The 2016 annual meeting where the Saporito posters were presented was held April 15–21, 2016, at the Vancouver Convention Center in Vancouver, B.C. and had 11,670 attendees who were able to study the 2,700 poster

presentations available for public viewing at the meeting. (2016 AAN Annual Report, Ex. 1019, page 12.) The Saporito posters were among those on public display and freely available to all 11,670 attendees.

38. Marinus's research examined the impact of IV-administered ganaxolone on both the EEG (electrical) and behavioral manifestations of SE. As to the electrical effects, the research found that the ganaxolone "elicits a sustained (≥ 5 hours) block of SE independent of treatment time after SE onset," indicating that the drug effectively terminated and treated SE as assessed by EEG. (Saporito Poster 2, Conclusion, Pages 1 and 5 of Ex. 1014.)

39. As to the behavioral effects, the research found that IV-administered ganaxolone "halted CSE [convulsive status epilepticus] and produced a dose-dependent reduction in seizures associated with CSE when administered at 3 different doses over four separate time points after the first observed convulsive seizure." (Saporito Poster 1, Conclusions, Pages 1 and 5 of Ex. 1013.) It also enhanced survival, measured 24 hours after CSE onset. (*Id.*)

40. Seeing these results in the rat pilocarpine model, persons skilled in the art would have reasonably believed that ganaxolone would show efficacy in treating human SE as well, for the reasons discussed above.

41. Marinus’s clinical work on the use of ganaxolone to treat SE in humans also began in 2016 and is ongoing. On April 14, 2016, the FDA granted Marinus Orphan Drug Designation for ganaxolone to treat SE. (April 15, 2016 Press Release, Ex. 1011.) On June 22, 2016, Marinus dosed its first patient in a Phase I Clinical Trial, representing the first clinical study in healthy volunteers intended to advance ganaxolone as an FDA-approved treatment for SE. (June 22, 2022, Press Release, Ex. 1022.) Marinus reported at the 6th London-Innsbruck Colloquium on Status Epilepticus and Acute Seizures held April 6–8, 2017, in Salzburg, Austria, that ganaxolone “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.” (Tsai, Ex. 1020.)

V. THE ’817 PATENT

42. United States Patent 11,395,817 (“the ’817 patent,” Ex. 1001), titled Methods and Compositions for Treatment of Epileptic Disorders, was filed on September 17, 2020 and issued on July 26, 2022. (*Id.*, cover sheet.) The patent states that it claims priority to earlier filings, including provisional applications filed on August 11, 2016 and April 26, 2017. (Ex. 1001 at 1:18–20.)

43. The '817 patent discloses methods of using various allosteric modulators, with or without gaboxadol, to treat a wide variety of epileptic disorders. SE is listed as one of the many epileptic disorders that the methods discussed in the patent are intended to treat. (Ex. 1001 at 2:13–35.)

44. One type of allosteric modulator discussed in the '817 patent is a neurosteroid. Ganaxolone is among the examples of neurosteroids listed in the '817 patent. (*Id.* at 6:22–26, 2:45–3:36.)

45. The '817 patent contains four Examples. Of these, Examples 3 and 4 relate to ganaxolone (as opposed to gaboxadol). Example 3 of the '817 patent presents an example showing the evaluation of the ability of allopregnanolone, ganaxolone, and gaboxadol to block benzodiazepine-resistant SE. Example 3 uses the rat lithium-pilocarpine model to perform this evaluation. (Ex. 1001 at 35:31–37:15.) Example 4 does not contain any data, but describes further studies that could be carried out to assess synergistic activity between gaboxadol and either allopregnanolone, ganaxolone, or lorazepam in the rat lithium-pilocarpine model. (Ex. 1001 at 37:17–38:40.)

46. In Example 3, ganaxolone was administered intraperitoneally to rats, and the results are summarized in Table 2 as well as Figures 3, 4, 5, 6A, and 6B. (Ex. 1001 at 35:31–37:15.) For these experiments, rats were administered

ganaxolone intraperitoneally 30 minutes after the first observed convulsive seizure. The rats were observed and scored for seizure severity for 120 minutes (2 hours) post drug administration. (*Id.*) At the highest dose of ganaxolone (20 mg/kg), 4/13 rats were protected against further convulsive seizure after 10 min of drug administration, suggesting efficacy of ganaxolone for treatment of SE. (Ex. 1001 at 36:4–24, Table 2, Figure 3.) At the 0.5, 2, and 5 mg/kg doses, there was no protection. ('817 patent at Table 2, Figure 3.) At 10 mg/kg, the level of protection was not statistically significant. (*Id.*)

47. The remainder of the data in Example 3 relates to 24-hour survival ('817 patent at Table 2, Figure 4) and the number of post-treatment seizures ('817 patent at Figure 5). There were no significant effects at any dose of ganaxolone on 24-hour survival (only gaboxadol at 10 mg/kg showed a statistically significant effect). For groups treated with ganaxolone doses of 2 mg/kg to 20 mg/kg (but not the group treated with a ganaxolone dose of 0.5 mg/kg), the number of post-treatment seizures is seen to be numerically less than the value indicated with the dotted line and marked VEH, which is presumably the number of post-treatment seizures in a control (vehicle-treated) group. There is no indication on the figure or in its description if the values are statistically different from the control values.

VI. LEGAL STANDARDS

48. In the following section, I describe my understanding of the legal standards that apply to written description and enablement, as explained to me by Marinus's counsel.

A. Anticipation

49. It is my understanding that a prior art reference "anticipates" a claim, and thus renders the claim unpatentable, if all elements of the claim are disclosed in that single prior art reference, either explicitly or inherently (i.e., necessarily present or implied). It is also my understanding that a prior art reference must enable the subject matter of the claims in order to anticipate the claims.

B. Obviousness

50. I understand that a patent claim is invalid as obvious if the differences between the claimed subject matter and what is taught by the prior art are such that the subject matter as a whole would have been obvious to a person having ordinary skill in the art at the time that the invention was made. I understand that four factual inquiries guide this determination: (1) the scope and content of the prior art; (2) the differences between the claims and the prior art; (3) the level of ordinary skill in the pertinent art; and (4) relevant secondary considerations (or objective criteria), such as unexpected results, commercial success, long-felt but unsolved

needs, failure of others, copying by others, licensing, acclaim and awards, teaching away or skepticism in the art, and contemporaneous invention by others.

51. I understand that the obviousness analysis is done from the perspective of a person of ordinary skill in the art at the time of the invention. I understand the relevant time period here to be the 2016–2017 timeframe.

52. I further understand that a person of ordinary skill in the art is presumed to have knowledge of the relevant prior art at the time of the claimed invention, and that the scope of the prior art comprises any prior art that was reasonably pertinent to the particular problems the inventor faced.

53. I understand that a claimed invention may be found obvious based on a combination of prior art references. Further, I understand that there need not be an explicit teaching, suggestion, or motivation to combine those references in order to determine that an invention is obvious. Nonetheless, I understand it is important that one of ordinary skill in the art have a reason to combine the elements found in the prior art in the way the claimed new invention does.

54. I understand that obviousness does not require absolute predictability of success. Rather, it is my understanding that a patent claim is obvious so long as there was a reasonable expectation of success.

C. Enablement

55. It is my understanding that a claim is enabled if a person of ordinary skill in the art could make and use the claimed invention without undue experimentation. I understand that some amount of experimentation is permissible, so long as that experimentation would not be considered “undue.” I also understand that the test for whether experimentation is “undue” is not quantitative, and that a significant amount of experimentation is permissible if it is either routine and if the specification provides a reasonable amount of guidance. I understand that in certain circumstances, even extensive experimentation is permissible if it involves repeating commonly used techniques.

VII. PERSON OF ORDINARY SKILL IN THE ART

56. In my opinion, a hypothetical person of ordinary skill in the art (“POSITA”) of the ’817 patent at the time of its filing would have had a medical degree, or a Ph.D. in pharmacology or other field related to the development of pharmaceuticals, and several years of experience as a practicing neurologist treating patients with neurological disorders, including seizure disorders, and/or several years of experience in researching treatments for such seizure disorders.

VIII. OPINIONS ON INVALIDITY

57. In the following sections, I provide my opinions on anticipation, obviousness, and enablement.

58. As described in detail in these sections, in my opinion, the '817 patent is anticipated by several of the prior art Marinus publications and is also obvious over combinations of those references, as laid out below. The references show that before Ovid filed the application for its '817 patent Marinus had already conducted significant research on the use of IV-administered ganaxolone to treat SE, had shown positive results in the well-established rat lithium-pilocarpine model, had obtained regulatory approval to study ganaxolone for the treatment of SE in humans, and had undertaken a Phase I clinical trial of ganaxolone with the intention of advancing ganaxolone as a commercial treatment for SE.

59. If Ovid takes the position that the references that I have discussed do not anticipate and/or render obvious the claims of the '817 patent because they are not enabled, then, in my opinion, the claims of the '817 patent cannot be enabled.

A. Claims 1–3, 10–17, and 19–31 Are Anticipated by the Zhang Publication

60. In my opinion, claims 1–3, 10–17, and 19–31 of the '817 patent are anticipated by the Zhang publication from Marinus (Ex. 1009), which gives examples that teach the skilled person how to carry out the claimed methods.

1. Claim 1

61. Claim 1 recites “A method of treating status epilepticus comprising administering to a patient in need thereof a pharmaceutical composition comprising ganaxolone or a pharmaceutically acceptable salt thereof.”

62. The ’817 patent defines “patient” broadly, as “includ[ing], but not limited to, primates such as humans, canines, porcine, ungulates, rodents, poultry, and avian.” (Ex. 1001 at 32:65–67.) Therefore, in addition to specifically relating to humans, the claims also relate to rats, which are rodents.

63. Zhang discloses “methods of treating status epilepticus . . . comprising administering an effective amount of [a] substituted β -cyclodextrin-ganaxolone injectable formulation to a patient suffering from [the same].” (Zhang, Ex. 1009, ¶ [0069].)

64. Because “[g]anaxolone is very poorly soluble in water” and is “difficult to formulate as an aqueous injectable,” Zhang teaches pharmaceutical compositions of ganaxolone “formulated as an aqueous injectable by complexing [it] with a substituted β -cyclodextrin, such as [CAPTISOL®, also referred to as sulfobutyl ether β -cyclodextrin].” (Zhang, Ex. 1009, ¶¶ [0005], [0031].) Zhang includes examples (Examples 1–6), showing how to make the disclosed formulations. (Zhang, Ex. 1009, ¶¶ [0104]–[0115].)

65. Zhang also includes examples that demonstrate the use of its ganaxolone formulations. (Zhang, Ex. 1009, ¶¶ [0116]–[0136].) Example 7 discloses plasma concentration profiles of ganaxolone over time in rats infused with IV-administered ganaxolone-CAPTISOL® (Zhang, Ex. 1009, ¶ [0116], Fig. 2.), and Example 8 describes the effects of ganaxolone in the rat lithium-pilocarpine model of SE (Zhang, Ex. 1009, ¶¶ [0117]–[0136].)

66. Furthermore, as described in Example 8, rats were administered a CAPTISOL®-ganaxolone formulation intravenously at 12 and 15 mg/kg, either 15 or 60 minutes after the onset of SE. (Zhang, Ex. 1009, ¶ [0117].) Therapeutic activity of ganaxolone in the treatment of SE was assessed by determining changes in EEG power for five hours after onset of SE. (Zhang, Ex. 1009, ¶ [0118].) Ganaxolone at both 12 and 15 mg/kg “produced a very strong reduction in SE amplitude overall.” (Zhang, Ex. 1009, ¶ [0134].)

67. In particular, when dosed 15 minutes after the onset of SE, ganaxolone reduced EEG power across all frequency ranges tested to baseline levels or lower for up to 5 hours. (Zhang, Ex. 1009, ¶ [0135].) Even when dosed 60 minutes after SE onset, ganaxolone also strongly reduced EEG power (not below baseline for up to 70 Hz, but dropping below baseline for 70–96 Hz). (Zhang, Ex. 1009, ¶ [0136].) This reduction in EEG power indicates to a person of

ordinary skill that ganaxolone is an effective method for treating SE. Tables 6 and 7, as well as Figures 3 and 4, present details of these results.

68. As discussed above (Section IV), seeing those results, persons skilled in the art would have reasonably believed that ganaxolone would show success in treating human SE as well.

69. Thus, in my opinion, the Zhang publication anticipates claim 1, with respect to either a rat or a human patient.

2. Claims 2 and 3

70. Claims 2 and 3 depend upon claim 1 and recite “wherein the patient is administered ganaxolone or a pharmaceutically acceptable salt thereof” either “twice daily” (for claim 2) or “three times daily” (for claim 3).

71. Zhang discloses that “[s]eizure disorders that may be treated with the substituted β -cyclodextrin-ganaxolone injectable formulation include status epilepticus . . .” and that “treating a patient suffering from seizures . . . include administering multiple injections of the substituted β -cyclodextrin-ganaxolone injectable formulation over a period of 1 to 10 days.” (Zhang, Ex. 1009, ¶¶ [0070], [0074].) As for the specific dosing, Zhang teaches that the injections “may be given at intervals of 1 to 24 hours,” and specifically discusses dosing schedules “in which the substituted β -cyclodextrin-ganaxolone injectable formulation is injected

every 1 hr, 2 hrs, 4 hrs, 6 hrs, 8 hrs, 12 hrs, or 24 hrs.” (*Id.*, ¶ [0074].) Dosing every 12 hours equates to twice daily, and dosing every 8 hours equates to three times daily.

72. Therefore, in my opinion, Zhang anticipates claims 2 and 3.

3. Claims 10–17

73. Claims 10–17 each depend on claim 1 and recite “wherein the patient is administered [X] mg ganaxolone or a pharmaceutically acceptable salt thereof,” where “X” is 600 mg (claims 10 and 11), 50 mg (claim 12), 100 mg (claim 13), 200 mg (claim 14), 300 mg (claim 15), 400 mg (claim 16), or 500 mg (claim 17). Claims 10 and 12–17 do not specify how or over what timeframe the recited amount is administered. Claim 11 specifies that the patient is administered the 600 mg “three times daily.”

74. In general, small molecule drugs may be administered to patients either as a flat-fixed dose (i.e., without respect to the weight of the patient), or based on the weight of the patient. In some cases, a drug may even be administered to a patient based on that individual patient’s blood levels.

75. Claims 10–17, however, do not specify how the dose is determined, only that the patient be administered a particular amount of ganaxolone, in mg (and, in the case of claim 11, a certain number of times per day).

76. As described above, SE is a serious and potentially life-threatening medical emergency that requires immediate treatment with a rapidly acting medication that will stop the seizures as quickly as possible, and which can be readily administered to a patient experiencing prolonged seizure(s). Therefore, in the ordinary treatment of SE and as described in Zhang, ganaxolone is administered as a bolus dose by non-oral infusion, typically intravenous or through other non-oral routes, so that therapeutic plasma concentration levels are reached as quickly as possible. (See, for example, Zhang, Ex. 1009, at ¶¶ [0018], [0021], [0081], [0082]). In some cases in the treatment of SE, therapeutic plasma concentrations must be maintained for prolonged periods of time. The required duration may be longer than the time plasma concentration persists at therapeutic levels following the bolus dose. Therefore, Zhang recites that in some cases, “multiple bolus doses are given over 1 to 10 days at intervals of 1 to 24 hours,” and that, “[i]n certain embodiments each bolus dose comprises about 1 mg/kg to about 20 mg/kg ganaxolone.” (Zhang, Ex. 1009, at ¶ [0085]).

77. While, in these passages, Zhang describes weight based dosing (as opposed, for example, to flat-fixed dosing), a person of skill in the art would readily be able to calculate the total dose in mg. For example, based on Zhang’s

descriptions, one could calculate the total dose in mg for a typical human adult patient, weighing in at 70 kg, as shown in the table below.

Claim(s)	Amount Administered (total mg)	Amount Administered to a 70 kg patient (in mg/kg)
10, 11	600	8.57
12	50	0.71
13	100	1.43
14	200	2.86
15	300	4.29
16	400	5.71
17	500	7.14

78. As is readily apparent from this table, Zhang's bolus doses of 1 mg/kg to 20 mg/kg, for a 70 kg patient, encompass each of the amounts recited in claims 10–17, with the exception of 50 mg (claim 12). However, a person of skill in the art would immediately understand that, for example, when administering a 1 mg/kg dose to a patient weighing 50 kg (a human child, perhaps), a total of 50 mg would be administered.

79. With respect to claim 11, which adds that the 600 milligrams is administered three times daily, Zhang teaches that multiple bolus doses (such as a 8.57 mg/kg dose administered to a 70 kg patient), can be administered at intervals of 1 to 24 hours, which includes every 8 hours (i.e., three times daily).

80. Therefore, in my opinion, Zhang anticipates claims 10–17.

4. Claims 19 and 20

81. Claims 19 and 20 of the '817 patent, which each depend on claim 1, recite patient outcomes. Claim 19 recites “wherein the pharmaceutical composition provides reduction in the frequency of seizures.” Claim 20 recites “wherein the pharmaceutical composition provides reduction in the frequency of seizures, the severity of seizures, or a combination thereof.”

82. Zhang discloses “methods of treating status epilepticus . . . comprising administering an effective amount of the substituted β -cyclodextrin-ganaxolone injectable formulation to a patient suffering from [the same].” (Zhang, Ex. 1009, ¶ [0069].) It also states that SE is a “serious seizure disorder.” (*Id.*, ¶ [0001].)

83. Zhang also teaches that “treatment” includes “relieving a condition caused by the disease or disorder, or reducing the symptoms of the disease or disorder.” (Zhang, Ex. 1009, ¶ [0025].)

84. As discussed above, Example 8 in Zhang tests ganaxolone’s effect on SE using the rat pilocarpine model. Ganaxolone at both the 12 and 15 mg/kg doses “produced a very strong reduction in SE amplitude overall.” (Zhang, Ex. 1009, ¶ [0134].)

85. In addition, Zhang teaches “[w]hen dosed 15 minutes after SE-onset, [ganaxolone] reduced EEG power across all frequency ranges to levels at or below baseline for at up to 5h. From 5-70 Hz, EEG power was reduced to near the baseline level and remained there for 5 h post dosing.” (Zhang, Ex. 1009, ¶ [0135].)

86. Further, Zhang teaches “[w]hen dosed at 60' post SE-onset, EEG power was also strongly reduced, but not below the baseline level for frequencies up to 70 Hz. From 70-96 Hz, EEG power appeared to drop below baseline from 1 to 4 h post dosing.” (Zhang, Ex. 1009, ¶ [0136].)

87. The statistically significant reduction in EEG power produced by ganaxolone (to baseline or below) in the examples would be understood by a person skilled in the art as indicating that ganaxolone terminated (stopped) SE, which is equivalent to a reduction in the frequency and severity of seizures. More specifically, when SE is terminated, the frequency of seizures is zero, which is the greatest reduction that can be obtained. Similarly, when SE is terminated, the severity has been reduced to the greatest extent possible.

88. Thus, in my opinion, Zhang anticipates claims 19 and 20 of the '817 patent.

5. Claim 21

89. Claim 21 of the '817 patent recites a “method of treating status epilepticus comprising administering to a patient in need thereof a pharmaceutical composition comprising ganaxolone or a pharmaceutically acceptable salt thereof wherein the pharmaceutical composition is administered intravenously.”

90. Claim 21 has all the same limitations that appear in claim 1 (and already discussed above) but adds the requirement that the composition is administered intravenously. Zhang teaches that its ganaxolone formulations “include parenteral formulations suitable for intravenous infusion.” (Zhang, Ex. 1009, ¶ [0029].) And example 8 shows the intravenous administration of ganaxolone formulations using the rat lithium-pilocarpine model of SE. (*Id.* at ¶¶ [0117]–[0136].)

91. Thus, in my opinion, the Zhang publication also anticipates claim 21.

6. Claim 22

92. Claim 22 recites “wherein the pharmaceutical composition provides improvement to the patient after administration for more than 8 hours.”

93. Zhang does not expressly state the composition provides improvement for more than 8 hours after administration. In Zhang’s Example 8, the rats experiencing SE were sacrificed after 5 hours and, so, even though some effects of

ganaxolone were sustained for the entire 5 hours, because the experiment was stopped at 5 hours, there is no data demonstrating a response “for more than 8 hours.”¹ To the extent that improvement to the patient were to occur after that time period, however, it would be the natural result of administering the ganaxolone formulations in the manner taught in Zhang.

94. Moreover, Zhang states: “[m]ethods of treatment also include administering multiple injections of the β -cyclodextrin-ganaxolone injectable formulation over a period of 1 to 10 days. The injections may be given at intervals of 1 to 24 hours.” (Zhang, Ex. 1009, ¶ [0074].) A skilled artisan would immediately recognize that if a single bolus treatment for SE suppresses seizures for up to 2 hours as ganaxolone is clearly shown to do in Tables 6 and 7, then the treatment could be repeated at intervals of 2 hours to provide sustained treatment for more than 8 hours.

¹ The '817 patent, like Zhang, does not expressly show improvement for more than 8 hours after administration. In Example 3 of the '817 patent, the only improvement shown for the rats is protection after 2 hours (see Section V, above). Therefore, as discussed below, if Zhang does not anticipate claim 22 or render it obvious, then, in my opinion, the '817 patent's description does not enable it.

95. Thus, in my opinion, the Zhang publication also anticipates claim 22.

7. Claim 23

96. Claim 23, which depends on claim 21, recites “wherein the pharmaceutical composition comprises about 30 mg ganaxolone.”

97. The Zhang publication teaches administering its β -cyclodextrin ganaxolone formulation at a range of concentrations from 0.025 mg/mL to about 15 mg/mL. (Zhang, Ex. 1009, ¶ [0033].) Claim 23 does not specify how much of the composition to administer to reach 30 mg ganaxolone. But at the upper end of the concentration range taught in Zhang, 2 mL of the composition would comprise about 30 mg ganaxolone.

98. Therefore, in my opinion Zhang also anticipates claim 23.

8. Claims 24-31

99. Claims 24–31 each depend on claim 21 and recite “wherein the patient is administered about [X1] mg to [X2] mg/day ganaxolone,” where “X1” and “X2” are low and high amounts of a range, respectively. Claims 24–31 do not specify how the recited amount is administered.

100. In general, small molecules drugs may be administered to patients either as a flat-fixed dose (i.e., without respect to the weight of the patient), or

based on the weight of the patient. In some cases, a drug may even be administered to a patient based on that individual patient's blood levels.

101. Claims 24–31, however, do not specify how the dose is determined, only that the patient be administered a daily amount of ganaxolone within a particular range.

102. As described above, SE is a serious medical emergency requiring immediate treatment. Therefore, in most cases and as described in Zhang, ganaxolone is administered as an intravenous bolus to allow a therapeutic plasma concentration to be reached as quickly as possible, and which can be readily administered to a patient experiencing prolonged seizure(s). (*See, e.g.*, Zhang, Ex. 1009, at ¶¶ [0018], [0081]–[0089]). Zhang recites that in some cases, “multiple bolus doses are given over 1 to 10 days at intervals of 1 to 24 hours,” and that, “[i]n certain embodiments each bolus dose comprises about 1 mg/kg to about 20 mg/kg ganaxolone.” (Zhang, Ex. 1009, at ¶ [0085]). In other cases, ganaxolone is administered by continuous infusion, with or without an initial bolus dose. (Zhang, Ex. 1009, at ¶ [0086]).

103. While, in these passages, Zhang describes weight-based dosing (as opposed, for example, to flat-fixed dosing), a person of skill in the art would readily be able to calculate the total daily dose in mg. For example, assuming a

single bolus dose per day as taught by Zhang, one could calculate the total dose in mg per day for an average human patient, weighing 70 kg, as shown in the table below.

Claim(s)	Amount Administered Per Day (range in mg)	Amount Administered to a 70 kg Patient Per Day (range in mg/kg)
24	150–1000	2.14–14.29
25	500–1000	7.14–14.29
26	625–650	8.93–9.29
27	650–675	9.29–9.64
28	675–700	9.64–10
29	625–700	8.93–10
30	700–725	10–10.36
31	1025–1050	14.64–15

104. As is readily apparent from this table, Zhang’s bolus doses of 1 mg/kg to 20 mg/kg, for a 70 kg patient, encompass each of the amounts recited in claims 24–31.

105. Therefore, in my opinion, Zhang anticipates claims 24–31.

B. Claims 1–3, 10–17, and 19–31 Would Have Been Obvious Over the Zhang Publication

106. In addition, it is my opinion that claims 1–3, 10–17, and 19–31 of the ’817 patent would have been obvious over the Zhang Publication.

107. If the Zhang Publication does not anticipate all the limitations of claims 1–3, 10–17, and 19–31, it at least suggests them by the same teachings I laid out above in Section VI. C.

108. Based on the teachings in Zhang, those of skill in the art would have had a reasonable expectation of success in achieving the claimed methods. As discussed above in Section IV, the rat lithium-pilocarpine model was, and is, a widely accepted model to identify potential treatments for SE. Example 8 of Zhang teaches that IV-administered ganaxolone terminates SE, i.e., ganaxolone gave a positive result in the model.

109. Thus, a person of skill in the art reading Zhang would at least have had a reasonable expectation of success in achieving the claimed methods of treating SE with ganaxolone, whether in a rat patient or a human patient. Therefore, in my opinion, claims 1–3, 10–17, and 19–31 would have been obvious over Zhang.

C. Claims 1–3, 10–17, and 19–31 Would Have Been Obvious Over the Zhang Publication Combined with 2016 AAN Saporito Poster 1 and/or 2016 AAN Saporito Poster 2.

110. In my opinion, claims 1–3, 10–17, and 19–31 would also have been obvious over the Zhang publication in combination with either or both of 2016 AAN Saporito Poster 1 (Ex. 1013) and 2016 AAN Saporito Poster 2 (Ex. 1014),

each of which reports on studies conducted by Marinus to investigate the effects of IV-administered ganaxolone in the rat lithium-pilocarpine model.

111. A person skilled in the art reading Zhang would have had reason to look for other references, particularly those that also detail Marinus's work related to the use of ganaxolone, the same drug discussed in Zhang, to treat SE, one of the main conditions addressed in Zhang. 2016 AAN Saporito Poster 1 and 2016 AAN Saporito Poster 2 are both such references, and each provides data on the effects of ganaxolone in the rat lithium-pilocarpine model of SE.

112. 2016 AAN Saporito Poster 1 (Ex. 1013) is titled "Ganaxolone Administered Intravenously Prevents Behavioral Seizures and Promotes Survival in the Rat Lithium-Pilocarpine Model of Status Epilepticus." In the study described in the poster, rats were administered lithium chloride (127 mg/kg) and pilocarpine (50 mg/kg) to induce convulsive SE (CSE). (2016 AAN Saporito Poster 1 at Methods, Pages 1 and 4 of Ex. 1013.) Rats were then administered ganaxolone (formulated at a concentration of 2.5 mg/mL in 30% Captisol®) at a dose of 6, 9, or 12 mg/kg at the time of seizure onset (time zero) and 15, 30, and 60 minutes after seizure onset. (*Id.*, Pages 1 and 2–3 of Ex. 1013.)

113. The data presented in the poster show that IV administered ganaxolone "halted CSE and produced a dose-dependent reduction in seizures

associated with CSE when administered at 3 different doses over four separate time points after the first observed convulsive seizure.” (*Id.*, at Conclusions, Pages 1 and 5 of Ex. 1013.) That data is presented in Figure 2, which shows the percentage of rats that were protected from seizure with the vehicle, the three different doses of ganaxolone, and the one dose of allopregnanolone tested, administered at time zero, 15 minutes, 30 minutes, and 60 minutes. After the administration of each agent, animals were monitored for 2 hours. (*Id.*, all Pages of Ex. 1013.) Animals that did not exhibit a convulsive seizure during that time period were categorized as protected. (*Id.*, at Methods, Pages 1 and 2–3 of Ex. 1013.)

114. The poster also shows that the IV administered ganaxolone promotes survival, measured 24 hours after the onset of CSE. That data is presented in Figure 3 (on Pages 1, 3, and 5 of Ex. 1013).

115. As discussed above, those of skill in the art understood on the priority date of the '817 patent, and still understand, the rat lithium-pilocarpine model to be a widely accepted model to identify potential treatments for SE. Halting behavioral SE in the model indicates that a test substance is effective in treating the SE condition. Thus, in my opinion, 2016 AAN Poster 1 (Ex. 1013) teaches a

method of treating SE by administering to a patient in need thereof a pharmaceutical composition of ganaxolone.

116. To the extent that the Zhang publication alone (Ex. 1009) does not render obvious claims 1–3, 10–17, and 19–31, those of skill in the art would have looked to the data in 2016 AAN Saporito Poster 1 (Ex. 1013) for additional confirmation that ganaxolone would have been reasonably expected to treat SE.

117. 2016 AAN Saporito Poster 2 (Ex. 1014) is titled “Intravenous Administration of Ganaxolone Attenuates Electroencephalographic Seizures in a Diazepam Resistant Model of Status Epilepticus.” Like the study on 2016 AAN Saporito Poster 1 (Ex. 1013), rats were administered lithium chloride followed by pilocarpine to induce SE. (2016 AAN Saporito Poster 2, at Methods, Pages 1 and 4 of Ex. 1014.) The poster explains that “[s]tatus epilepticus can be modeled in experimental animals by administration of lithium and pilocarpine.” (*Id.*, at Overview, Pages 1 and 2 of Ex. 1014.) In this study, the rats had been pre-implanted with cortical electrodes to measure electrical activity consistent with seizures. (*Id.*, at Methods, Pages 1 and 4 of Ex. 1014.) Ganaxolone was administered via IV bolus injection 15 or 60 minutes after the detection of SE onset. (*Id.*)

118. The results of the study showed that IV administration of ganaxolone elicited a sustained block of SE as assessed by EEG when it was administered either 15 or 60 minutes after onset of SE. (*Id.*, at Conclusions, Pages 1 and 5 of Ex. 1014.) The term “block” would be interpreted by a skilled artisan to mean terminate or stop SE. Therefore, the results demonstrated that ganaxolone gave a positive result in the model, which is widely accepted to identify potential treatments for SE. The data are shown in Figures 3 and 4.

119. Thus, in my opinion, 2016 AAN Poster 2 teaches a method of treating SE by administering to a patient in need thereof a pharmaceutical composition of ganaxolone.

120. To the extent that the Zhang publication alone (Ex. 1009) does not render obvious claims 1–3, 10–17, and 19–31, those of skill in the art would have looked to the data in 2016 AAN Saporito Poster 2 (Ex. 1014), which comes from the same company’s research, deals with the same drug, and seeks to assess the potential of that drug in the treatment of the same condition, for additional confirmation that ganaxolone would have been reasonably expected to treat SE.

121. Therefore, in my opinion, claims 1–3, 10–17, and 19–31 of the ’817 patent would have been obvious over Zhang (Ex. 1009) in combination with 2016 AAN Saporito Poster 1 (Ex. 1013) and/or 2016 AAN Saporito Poster 2 (Ex. 1014),

both of which provide additional evidence that a person of skill in the art would draw upon to form a reasonable expectation of achieving the claimed methods.

D. Claims 1–3, 10–17, and 19–31 Would Have Been Obvious Over the Zhang Publication Combined with Marinus’s Orphan Drug Press Release and/or Marinus’s Phase I Press Release.

122. In my opinion, claims 1–3, 10–17, and 19–31 would also have been obvious over the Zhang publication in combination with either or both of Marinus’s Orphan Drug Press Release (Ex. 1011) and Marinus’s Orphan Drug Press Release (Ex. 1022), each of which reports on Marinus’s clinical development of ganaxolone for the treatment of SE.

123. As discussed in detail above, Zhang teaches “methods of treating status epilepticus . . . comprising administering an effective amount of the substituted β -cyclodextrin-ganaxolone injectable formulation to a patient suffering from [the same],” and includes positive data for ganaxolone in the rat lithium-pilocarpine model of SE demonstrating that ganaxolone is useful in the treatment of SE. (Zhang, Ex. 1009, ¶¶ [0069], [0117]–[0136].)

124. A person of skill in the art reading Zhang would have had reason to look for additional publications related to the treatment of SE by administering ganaxolone to a patient in need. Marinus’s Orphan Drug and Phase I Press Releases are such publications.

125. Marinus's Orphan Drug Press Release reports that Marinus received Orphan Drug Designation for its IV formulation of ganaxolone, for the treatment of SE. (Ex. 1011) A person skilled in the art would know that, to obtain Orphan Drug Designation, the FDA Office of Orphan Drug Products requires the submission of significant data supporting the rationale for the use of the drug to treat the proposed disease or condition. Moreover, by law, FDA must refuse to grant Orphan Drug Designation if "[t]here is insufficient information about the drug, or the disease or condition for which it is intended, to establish a medically plausible basis for expecting the drug to be effective in the prevention, diagnosis, or treatment of that disease or condition." (21 C.F.R. § 316.20(b)(4); 21 C.F.R. § 316.25 (explaining refusal to grant orphan-drug designation.) Thus, the skilled artisan would have recognized that at the time of designation, sufficient information about ganaxolone had been provided to the FDA to establish a medically plausible basis for ganaxolone to be effective in the "Treatment of status epilepticus," which was its publicly disclosed Orphan Drug Designation.

126. Marinus's Phase I Press Release reports that Marinus had dosed its first patient in a Phase I clinical study conducted at the Duke Clinical Research Unit, Duke University Medical Center, Durham, North Carolina. Since this study was conducted in the United States and ganaxolone was not an approved drug, the

FDA permitted the study to be undertaken under an IND. In order for the FDA to permit a clinical study to be undertaken under an IND, those of skill in the art understand that the sponsor must provide evidence that “the compound exhibits pharmacological activity that justifies commercial development.” (See <https://www.fda.gov/drugs/investigational-new-drug-ind-application/drug-development-and-review-definitions>.) Since the purpose of Marinus’s trial was to advance commercial development of ganaxolone for the treatment of SE, a person of skill in the art would have recognized that the FDA had determined that IV ganaxolone was reasonably likely to be successful in the treatment of SE in humans.

127. Even if Zhang alone were not enough to render these claims obvious, which I believe that it is, the Marinus Press Releases demonstrate that it had been publicly recognized and also recognized by regulators that IV ganaxolone is reasonably likely to be a successful method for treating SE.

128. Therefore, in my opinion claims 1–3, 10–17, and 19–31 would have been obvious over Zhang in combination with Marinus’s Orphan Drug Press Release and/or Marinus’s Phase I Press Release.

E. Claims 4–9 and 18 Would Have Been Obvious Over the Zhang Publication in Combination with Shaw

129. In my opinion, Claims 4–9 and 18 would also have been obvious over Zhang in combination with Shaw (Ex. 1010).

130. Shaw (Ex. 1010) is a Marinus patent publication titled “Ganaxolone Formulations and Methods for the Making and Use Thereof,” which was published on May 31, 2007.

131. Shaw describes “ganaxolone formulations . . . as well as methods of making ganaxolone formulations and their use in the treatment of epilepsy-related and other central nervous system disorders.” (Shaw, Ex. 1010, ¶ [0002].) It also provides examples of making various oral formulations of ganaxolone. (Shaw, Ex. 1010, ¶ [00426].)

132. Shaw contains data showing that ganaxolone, administered to 6 healthy subjects as an oral suspension, resulted in a “C_{max} of 37 ± 25 ng/ml and an AUC₍₀₋₂₄₎ of 184 ± 104 ng*h/ml.” (Shaw, Ex. 1010, ¶ [00546].)

133. As discussed in detail above, Zhang discloses “methods of treating status epilepticus . . . comprising administering an effective amount of the substituted β-cyclodextrin-ganaxolone injectable formulation to a patient suffering from [the same]” and includes positive data for ganaxolone in the rat lithium-

pilocarpine model of SE, demonstrating that ganaxolone is effective in the treatment of SE. (Zhang, Ex. 1009, ¶¶ [0069], [0117]–[0136].)

134. A person of skill in the art would have had good reason to combine Shaw and Zhang, both of which include studies by Marinus, and both of which study the same drug (ganaxolone) for seizure conditions.

135. Claim 1 of the '817 patent recites “A method of treating status epilepticus comprising administering to a patient in need thereof a pharmaceutical composition comprising ganaxolone or a pharmaceutically acceptable salt thereof.” Claims 4 and 5 depend from claim 1 and recite particular dosage forms for the ganaxolone. In particular, claim 4 requires that the pharmaceutical composition is an “oral suspension,” and claim 5 requires that it is an “oral capsule.”

136. Shaw specifically teaches those two types of dosage forms for ganaxolone, explaining that that its “pharmaceutical ganaxolone compositions . . . can be formulated into any suitable dosage form, including but not limited to . . . aqueous oral suspensions” and “solid dosage forms including oral solid dosage forms” such as “capsules.” (Shaw, Ex. 1010, ¶ [00174].)

137. Relevant to the oral dosing requirements for claims 4 and 5, Zhang teaches that “[g]anaxolone has a relatively long half-life - approximately 20 hours in human plasma following *oral administration*,” [emphasis added] and that it “has

a short $T_{\max}$, which means that therapeutic blood levels are reached quickly.”

(Zhang, Ex. 1009, ¶ [0027].) Thus, Zhang suggests to those of skill in the art that oral administration is appropriate for ganaxolone.

138. The teachings of Shaw and Zhang would have given a person of skill in the art a reasonable expectation of success in achieving the methods recited in claims 4 and 5. Thus, in my opinion, claims 4 and 5 would have been obvious over the combination of Shaw and Zhang.

139. Claim 6 depends on claim 4 (which requires an “oral suspension”) and recites that the ganaxolone or pharmaceutically acceptable salt thereof is administered in an amount of up to 1,800 mg/day.

140. Shaw teaches that “[in one] aspect, the invention is directed to a liquid pharmaceutical oral suspension comprising ganaxolone . . . based on a dose of 200 mg ganaxolone.” (Shaw, Ex. 1010, ¶ [0068].) It also discloses that its ganaxolone formulations can be administered “once-a-day, twice-a-day (b.i.d), or three times a day (t.i.d.).” (*Id.*, ¶ [00174].) If 200 mg ganaxolone is administered one, two, or three times a day, as taught in Shaw, the amount of ganaxolone administered for all three of those dosing frequencies fall within the claimed range of “up to 1,800 mg/day.”

141. Thus, in my opinion, the combination of Shaw and Zhang renders claim 6 obvious.

142. Claims 7–9 each depend on claim 1 and recite various doses and administration schedules of an “oral suspension” comprising ganaxolone or a pharmaceutically acceptable salt thereof, including the requirements that the suspension comprise 50 mg/mL ganaxolone, that it is administered three times per day, and that the patient is administered an amount of up to 1,800 mg/day.

143. Shaw teaches that “[i]n one embodiment, a ganaxolone formulation is administered as an aqueous oral suspension at a concentration of about 25 mg/ml to about 100 mg/ml final concentration,” and that these suspensions “can be administered both as a single dose per day or given multiple times within a 24 hour period,” including “three times a day.” (Shaw, Ex. 1010, ¶ [00289].) As one example, Shaw teaches administering 4 ml of a 50 mg/ml suspension. (*Id.*, ¶ [00546].) Administered three times daily, 4 ml of a 50 mg/ml suspension would result in administration of 600 mg per day, which is within the “up to 1,800 mg” range of the claims.

144. Thus, Shaw teaches administration of 50 mg/ml ganaxolone (claim 7), administration of that 50 mg/ml three times daily (claim 8) and administration of up to 1,800 mg/day (claim 9). In my opinion, the teachings of Shaw, in

combination with the teachings of Zhang already discussed, including its teachings about using ganaxolone to treat SE, would have made claims 7–9 obvious to a person skilled in the art.

145. Claim 18 depends on claim 1 and recites that the patient is administered 200–600 mg ganaxolone or a pharmaceutically acceptable salt thereof as an oral capsule.

146. Shaw teaches that “[i]n various other embodiments of the present invention, the amount of ganaxolone administered to a subject via a solid dosage form to achieve a therapeutically effective concentration ganaxolone is typically in the range of 50 mg to about 800 mg.” (Shaw, Ex. 1010, ¶ [00288].) Those “solid dosage forms include oral solid dosage forms” such as “capsules.” (*Id.*, ¶ [00174].) And, as discussed above, Shaw teaches that its formulations can be administered “three times a day.” (*Id.*)

147. Thus, for the same reasons discussed above for claims 4–9, in my opinion, claim 18 would also have been obvious over the combination of Shaw and Zhang.

F. Claims 4–9 and 18 Would Have Been Obvious Over the Zhang Publication in Combination with Shaw and Marinus’s Orphan Drug Press Release and/or Marinus’s Phase I Press Release

148. In my opinion, Claims 4–9 and 18 would also have been obvious over Zhang and Shaw in further combination with Marinus’s Orphan Drug Press Release (Ex. 1011) and/or Marinus’s Phase I Press Release (Ex. 1022).

149. As discussed above, The Marinus Press Releases demonstrate that it had been publicly recognized and also recognized by regulators that IV ganaxolone is reasonably likely to be a successful method for treating SE.

150. Thus, if the combination of Zhang and Shaw is not sufficient to render the claims obvious, which I believe that it is, Marinus’s Orphan Drug Press Release and/or Marinus’s Phase I Press Release provided additional support that the use of IV administered ganaxolone in the treatment of SE in human patients was reasonably likely to be successful.

151. Thus, in my opinion, claims 4–9 and 18 would also have been obvious over Zhang and Shaw, combined with Marinus’s Orphan Drug Press Release and/or Marinus’s Phase I Press Release.

G. All Claims of the '817 Patent Lack Enablement if the Prior Art is Not Sufficient to Anticipate them or Render them Obvious

152. If Ovid takes the position that the references that I have discussed do not anticipate and/or render obvious the claims of the '817 patent because they are not enabled, then, in my opinion, the claims of the '817 patent cannot be enabled.

153. Claim 1 of the '817 patent recites “a method of treating status epilepticus comprising administering to a patient in need thereof a pharmaceutical composition comprising ganaxolone or a pharmaceutically acceptable salt thereof.”

154. The prior art discussed above demonstrates, based on extensive experimentation in the rat lithium-pilocarpine model, that ganaxolone can treat SE. And, in particular, that ganaxolone administered intravenously was effective for treating SE. As is also discussed above, based on the results seen in the rat lithium-pilocarpine model, a person of skill in the art would reasonably have expected ganaxolone to be effective for treating SE in humans.

155. As described above, the '817 patent also includes an experimental Example that utilizes the rat lithium-pilocarpine model to investigate the effect of administering ganaxolone. In this case, intraperitoneally.

156. In contrast to the extensive experimental evidence presented in Marinus's publications, the '817 patent's one example shows only that a single dose of ganaxolone (20 mg/kg) at one time point (30 min after the first convulsive

seizure) halted convulsive SE in just 4 out of 13 (30.8%) of the rats. The '817 patent does not contain any other data demonstrating that ganaxolone is effective in treating SE. In sum, there is more extensive and convincing evidence in the prior art, which all comes from Marinus's research, demonstrating that there will be a reasonable likelihood of success in treating SE with ganaxolone than there is in the '817 patent.

157. It is my opinion, as discussed above, that the claims of the '817 patent are anticipated and/or rendered obvious over the prior art publications. In short, Marinus's publications, which were published before the '817 patent was filed, contain data that is more extensive and more compelling with respect to the treatment of SE with ganaxolone than the disclosure in the '817 patent. If the description and data demonstrated in the prior art is not sufficient to anticipate and/or render obvious the claims of the '817 patent, then the less compelling description and disclosure of the '817 patent cannot be enough to support the conclusion that a person of skill in the art would understand that the inventors had invented what is claimed.

158. Some of the dependent claims of the '817 patent recite particular dosage forms (claims 4 and 6–9: oral suspension; claims 5, and 18: oral capsule; claims 21–31: intravenous). The '817 patent (unlike Marinus's publications), does

not contain any working examples of treating SE (in a rat model or otherwise) using any of those dose forms. In the '817 patent's Example 3, the rats were administered ganaxolone intraperitoneally. Therefore, to the extent that Marinus's publications do not render these dosage forms anticipated or obvious, the '817 patent does not enable them.

159. Other of the dependent claims recite particular concentrations (claim 23), dosing amounts (claims 6, 7–18 and 24–31) and/or administration schedules (claims 2, 3, 8, 9, and 11). A person of skill in the art would have arrived at these dosing amounts and administration schedules based on Marinus's publications. If not, however, a person of skill in the art would certainly not have arrived at them based on the only dose (20 mg/kg) for which data presented in the '817 patent (Example 3) could possibly suggest efficacy for treating SE in a rat. Therefore, to the extent that Marinus's publications do not render these concentrations, doses, and administration schedules anticipated or obvious, the '817 patent does not enable them.

160. Finally, some of the dependent claims recite particular outcomes (claims 19, 20, and 22). Again, the disclosure in the '817 patent is not more compelling than that in the Marinus publications. Therefore, to the extent that

Declaration of Dr. Michael A. Rogawski
in support of Petition for Post-Grant Review
of U.S. Patent No. 11,395,817

Marinus's publications do not render these outcomes anticipated or obvious, the
'817 patent does not enable them.

I declare under the penalty of perjury that the foregoing is true and correct.

Executed on March 10, 2023.



Michael A. Rogawski

Exhibit A

MICHAEL ANDREW ROGAWSKI

EDUCATION

Undergraduate and Graduate

- 1974 B.A., *magna cum laude*, Amherst College.
1980 M.D., Yale University School of Medicine.
1980 Ph.D., Pharmacology, Yale University.

Postgraduate Research and Clinical Training

- 1980–1981 Intern (Internal Medicine), Yale University School of Medicine (Waterbury Hospital).
1982–1985 Resident and Fellow (Neurology), The Johns Hopkins Hospital.
1981–1984 Medical Staff Fellow (Pharmacology Research Associate Training Program), Laboratory of Neurophysiology, National Institute of Neurological and Communicative Disorders and Stroke, National Institutes of Health.

Special Courses

- 1981 Electrophysiology of the Mammalian Brain Slice, Cold Spring Harbor Laboratory.
1983 Electrophysiological Methods Used in Analyzing the Mode of Action of Transmitters, Cold Spring Harbor Laboratory.
1986 Fundamental Neurobiological Approaches to Disease: Epilepsy, Rockefeller University.

PROFESSIONAL APPOINTMENTS

- 1986–1989 Senior Staff Fellow, Medical Neurology Branch, National Institute of Neurological and Communicative Disorders and Stroke, National Institutes of Health.
1987–2006 Active Staff, Clinical Center, National Institutes of Health.
1989–1990 Medical Officer – Research, National Institute of Neurological Disorders and Stroke, National Institutes of Health.
1990–2006 Senior Investigator and Chief, Epilepsy Research Section, National Institute of Neurological Disorders and Stroke, National Institutes of Health.
2007– Professor and Chair (until 2012), Department of Neurology, University of California, Davis; and Pharmacology and Toxicology Graduate Group; Neuroscience Graduate Group; Molecular, Cellular and Integrative Physiology Graduate Group; and Center for Neuroscience.
2008– Special Government Employee, Peripheral and Central Nervous System Drugs Advisory Committee, Center for Drug Evaluation and Research, Food and Drug Administration.
2012–2014 President, American Society for Experimental Neurotherapeutics.
2012– Co-Director, CounterACT Center of Excellence, University of California, Davis.
2016– Professor, Department of Pharmacology, University of California, Davis.
2017– Co-Principal Investigator (Principal Investigator since 2018), University of California Drug Discovery Consortium.

HONORS AND AWARDS

- 1974 Phi Beta Kappa, Society of Sigma Xi
1991 NINDS Merit Award
1992 NINDS Equal Opportunity Achievement Award
1993 NIH Director's Award

1994	Epilepsy Research Award, American Society for Pharmacology and Experimental Therapeutics
2000	British Pharmacological Society Lecture in Honor of Retirement of Brian Meldrum
2001	Killam Lecture, Montreal Neurological Institute
2003	Elected to Active Membership, American Neurological Association
2006	Certificate of Appreciation, Society for Neuroscience
2009	William G. Lennox Lecture, American Epilepsy Society
2011	Service Award, American Epilepsy Society
2015	Neuroendocrine Research Award, American Academy of Neurology
2017	Physician-Scientist Award, Epilepsy Foundation Greater Los Angeles
2018	Elected Fellow, American Association for the Advancement of Science (Section on Neuroscience)
2018	Elected Full Member, Sigma Xi, the Scientific Research Honor Society
2019	Innovator of the Year, UC Davis Chancellor's Innovation Awards
2019	The Theodore Koppányi Lectureship in Pharmacology, Department of Pharmacology & Physiology, Georgetown University
2020	Elected Fellow, National Academy of Inventors
2021	Designated honorary title of Distinguished Professor (Above Scale)

PROFESSIONAL ACTIVITIES

Professional Society Memberships

Society for Neuroscience
American Society for Pharmacology and Experimental Therapeutics
American Academy of Neurology
American Epilepsy Society
American Society for Experimental Neurotherapeutics
American Neurological Association (Elected Fellow)

Editorial Board Memberships

1975–1979 *Yale Journal of Biology and Medicine*
1988– *Cellular and Molecular Neurobiology*
1992–1999 *Jasper's Basic Mechanisms of the Epilepsies, Third Edition* – Advisory Board)
1992–2008 *Neuropharmacology* – Executive Editor (1996–2008)
1993–2004 *Synapse*
1994–1998 *Epilepsy Advances*
1994–1997 *Epilepsia*
1994– *Molecular Pharmacology*
1999–2010 *CNS Neuroscience & Therapeutics* (formerly *CNS Drug Reviews*)
2000–2004 *Pharmacology and Therapeutics*
2001– *BMC Pharmacology and Toxicology* (formerly *BMC Pharmacology*)
2001– *Epilepsy Currents* – Founder, Associate Editor (2001–2008), Co-Chief Editor (2008–2012)
2001–2006 *Epilepsy Research*
2002– *Current Neuropharmacology*
2005– *Neurotherapeutics, The Journal of the American Society for Experimental NeuroTherapeutics* – Associate Editor (2006–2012)
2006–2012 *Jasper's Basic Mechanisms of the Epilepsies, Fourth Edition* – Co-editor)
2011– *Scientific Reports* (Nature Publishing Group)
2011– *The Scientific World Journal (Pharmacology Domain)*
2012– *PeerJ* – Academic Editor
2016– *Epilepsia Open* – Associate Editor

Review Committee Memberships

1992–1993	Reviewers Reserve, Alcohol, Drug Abuse and Mental Health Administration
1993–1997	Regular Member, Neuropharmacology and Neurochemistry Study Section (NPNC), NIH
1996	Reviewer, Pharmacology Research Associate Program, NIGMS
1998	Review Panel Member, Advanced Anticonvulsant Program, U.S. Army Medical Research Institute of Chemical Defense
2000	Chair, Peer Review Panel, Neurotoxin Exposure Treatment Research Program, U.S. Army Research and Materiel Command
2001	Member, Molecular, Cellular and Developmental Neuroscience 5 (MDCN-5) Study Section, Center for Scientific Review, NIH
2002	Chair, Peer Review Panel, Neurotoxin Exposure Treatment Research Program (Pathogenesis and Therapeutics in Parkinson's and other Neurodegenerative Diseases), U.S. Army Research and Materiel Command
2002–2005	Scientific Advisory Board, CURE – Citizens United for Research in Epilepsy
2002	Review Committee Member, Research Grants and Fellowships Committee, Professional Advisory Board, Epilepsy Foundation
2004	Review Committee Member, Partnership for Pediatric Epilepsy Research, Epilepsy Foundation
2005	Reviewer, Epilepsy Research Foundation (U.K.)
2005	Chair, Peer Review Panel, Epilepsy and Traumatic Brain Injury, Peer Reviewed Medical Research Program, Office of Congressionally Directed Medical Research Programs (CDMRP), U.S. Army Research and Materiel Command
2005	Chair, Traumatic Brain Injury and Epilepsy Scientific Advisory Board, CURE – Citizens United for Research in Epilepsy
2006	Reviewer, Mental Retardation and Developmental Disabilities Research Centers 2006 Special Emphasis Panel ZHD1 DSR–H(30), National Institute of Child Health and Human Development, NIH
2006	Reviewer, Countermeasures Against Chemical Threats (CounterACT) Special Emphasis Panel ZNS2 SRB-R(22), National Institute of Neurological Disorders and Stroke
2007	Reviewer, Prevention of Epilepsy After Traumatic Brain Injury Program, CURE – Citizens United for Research in Epilepsy
2008	Reviewer, CURE – Citizens United for Research in Epilepsy
2008	Reviewer, Brain Disorders and Clinical Neurosciences Fellowships Special Emphasis Panel ZRG1 F01-V(20), Center for Scientific Review, NIH
2008–2009	Consultant, Peripheral and Central Nervous System Drugs Advisory Committee, FDA
2009	Site Visit Reviewer, Program Project Grant “Anesthetics: Cellular and Molecular Actions” (5 PO1 GM47969-18; Program Director: J. H. Steinbach), Washington University, St. Louis
2009	Reviewer, Challenge Grants Panel ZRG1 ETTN-A (58), Center for Scientific Review, NIH
2009	Reviewer, NSD-A Study Section ZNS1 SRB-R (39), National Institute of Neurological Disorders and Stroke, NIH
2009	Reviewer, CURE – Citizens United for Research in Epilepsy
2009	Reviewer, Neuroprotection and Neurodegeneration Special Emphasis Panel ZRG1 MDCN-G (02), Center for Scientific Review, NIH
2010, 2012	Consultant, Division of Metabolism and Endocrinology Products, FDA
2011	Reviewer, ZNS1 SRB-B (31), National Institute of Neurological Disorders and Stroke Special Emphasis Panel, NIH
2011	Reviewer, ZNS1 SRB-P (62), National Institute of Neurological Disorders and Stroke Special Emphasis Panel, NIH
2012	Reviewer, ZNS1 SRB-B (33), NIH Epileptogenesis Centers Without Walls Planning Grant Study Section, National Institute of Neurological Disorders and Stroke, NIH

2012	Reviewer, ZRG1 MDCN-B (91), Drug Discovery for the Nervous System, Center for Scientific Review, NIH
2013	Consultant, Neurological Devices Advisory Panel, Center for Devices and Radiological Health, FDA
2013	Reviewer, Peer Review Panel, Psychological Health and Traumatic Brain Injury Research Program, Congressionally Directed Medical Research Programs (CDMRP)
2015	Reviewer, Peer Review Panel, Chronic Migraine and Post-Traumatic Headache Preapplication, Congressionally Directed Medical Research Programs (CDMRP)
2016	Chair, Peer Review Panel, 2015 Epilepsy Research Program, Congressionally Directed Medical Research Programs (CDMRP)
2016	Chair, ZNS1 SRB-T (22), Small Molecule Drug Discovery and Development (BPN), National Institute of Neurological Disorders and Stroke, NIH
2017	Reviewer, ZRG1 PSE-N (02), Behavioral Genetic, Neurological and Musculoskeletal Epidemiology, NIH
2017	Chair, Peer Review Panel, Focused Program – Pain Medicine, Peer Reviewed Medical Research Program, U.S. Army Medical Research and Materiel Command (USAMRMC), Congressionally Directed Medical Research Programs (CDMRP)
2018	Reviewer, ZRG1 EMNR-S 55, Translational Research in Pediatric and Obstetric Pharmacology and Therapeutics Special Emphasis Panel, Center for Scientific Review, NIH
2018	Reviewer, ZNS1 SRB-T(34), Blueprint Neurotherapeutics Network Review Panel, National Institute of Neurological Disorders and Stroke, NIH
2019	Reviewer, ZNS1 SRB-T(37), Blueprint Neurotherapeutics Network Review Panel, National Institute of Neurological Disorders and Stroke, NIH
2019	Reviewer, ZNS1 SRB-T (41), Blueprint Neurotherapeutics Network Review Panel, National Institute of Neurological Disorders and Stroke, NIH
2021	Reviewer, ZRG1 ETTN-S (14 B), Small Business: Drug Discovery Involving the Nervous System, Special Emphasis Panel, Center For Scientific Review, NIH
2022	Chair, Peer Review Panel, Idea Development - Research Partnership - 4 (ID-RP-4), Peer Reviewed Medical Research Program, U.S. Army Medical Research and Materiel Command (USAMRMC), Congressionally Directed Medical Research Programs (CDMRP)
2022	Reviewer and Co-Chair, Reviewer, ZRG1 ETTN-S (14), Small Business: Drug Discovery Involving the Nervous System, Special Emphasis Panel, Center For Scientific Review, NIH
2022	Reviewer, ZNS1 SRB-K (35), R13 Review, Special Emphasis Panel, National Institute of Neurological Disorders and Stroke, NIH
2022	Chair, ZRG1 NV-S 14 B, Small Business: Drug Discovery Involving the Nervous System, Special Emphasis Panel, Center for Scientific Review, NIH

Committee Memberships

1991–2004	Technology Advisory Committee, NINDS
1992–1994	Long Range Planning Committee, American Epilepsy Society
1993–1994	Technology Transfer Policy Board, NIH
1993–1994	Program Committee, Winter Conference on Brain Research
1994–1995	Program Committee, 4th International Congress on Amino Acids
1995–1997	Program Committee, American Epilepsy Society
1996–1997	Chair, Program Committee, American Epilepsy Society (and Member, Annual Meeting Committee and Annual Course Committee)
1998–2005	Ad Hoc Committee on Electronic Initiatives, Society for Neuroscience
1999–2003	Chair, Technology Committee, American Epilepsy Society
2000–2003	Board of Directors, American Epilepsy Society
2003–2005	Scientific Advisory Board, CURE – Citizens United for Research in Epilepsy

- 2004 Program Committee, American Society for Experimental NeuroTherapeutics
- 2005–2006 Chair, Publications Committee, American Epilepsy Society
- 2006 Information Technology Committee, Society for Neuroscience
- 2005–2015 Board of Directors, American Society for Experimental NeuroTherapeutics
- 2015– Epilepsy Research Benchmark Stewards Committee, American Epilepsy Society

Meetings Organized

- 1993 Antiepileptic Drugs in the 1990s: Current and Investigational Therapies (Focus on Epilepsy II), Whistler, B.C., Canada (with M. Avoli and B.J. Wilder).
- 1995 Regulation of Ion Channel Gene Expression by Seizures, Investigators Workshop, Annual Meeting, American Epilepsy Society, Baltimore, MD.
- 1996 *Neuropharmacology* Symposium on Potassium Channels (satellite to Society for Neuroscience Meeting sponsored by Elsevier Science Ltd.), Washington, D.C. (with I. Levitan).
- 1999 Generalized Epilepsies: From Genes to Clinic (Focus on Epilepsy V), Québec City, Canada (with M. Avoli, C. Marescaux and G. Avanzini).
- 2001 Treatment of Epileptic Syndromes: From Molecular Targets to Quality of Life (Focus on Epilepsy VI), Château Mont-Tremblant, Québec, Canada (with M. Avoli, C. Wasterlain and G. Avanzini).
- 2003 Amygdala, Seizures and Epileptogenesis (Focus on Epilepsy VII), Montreal Neurological Institute, Montreal (with M. Avoli).
- 2004 National Capital Cure Epilepsy Conference, American Association for the Advancement of Science, Washington, D.C. (with S. Axelrod).
- 2004 Disorders of Impulsivity and Amygdala Neurocircuitry, Uniformed Services University of the Health Sciences, Bethesda, MD (conference steering committee).
- 2005 Antiepileptic Drugs: Not Just for Epilepsy, Annual Meeting, American Society for Experimental NeuroTherapeutics, Washington, D.C.
- 2008 New Horizons in the Development of Antiepileptic Drugs IV: Nontraditional Approaches to Treat Epilepsy, Clontarf Castle, Dublin, Ireland (with G. Holmes).
- 2009 Fourth International Workshops, Jasper's Basic Mechanisms of the Epilepsies, Yosemite National Park, California (with J.L. Noebels, M. Avoli, R.W. Olsen, A.V. Delgado-Escueta).
- 2019– Fifth International Workshops, Jasper's Basic Mechanisms of the Epilepsies, Yosemite National Park, California (with J.L. Noebels, M. Avoli, A. Vessani, A.V. Delgado-Escueta).

Graduate Student Thesis Committee Member and Major Professor

- 1991–1993 R. Alberto Travagli, Department of Pharmacology, Georgetown University School of Medicine.
- 1994–1996 Jon Poling, Department of Pharmacology, Georgetown University Medical Center.
- 1994–1996 Wolfram A. Gottschalk, Department of Physiology and Biophysics, Georgetown University School of Medicine.
- 2003–2005 Natalie White, Department of Pharmacology, Texas Tech University.
- 2011–2014 Emir Leal Leon, Pharmacology and Toxicology Graduate Group, University of California, Davis.
- 2015–2017 Pearl Chen, Molecular, Cellular and Integrative Physiology Graduate Group, University of California, Davis.

INVITED PRESENTATIONS

- 1977 "Hirschsprung's Disease in the 70's," Surgical Grand Rounds, Yale-New Haven Hospital (with R.J. Touloukian).
- 1978 "Hirschsprung's Disease: Absence of Serotonergic Neurons in the Aganglionic Colon," Ninth Annual Meeting of the American Pediatric Surgical Association, Hot Springs, Virginia. Awarded best scientific paper.
- 1978 "Response of Central Monoaminergic Neurons to the Ergots Lisuride and LSD: Single Cell Studies," Greater Hartford Chapter of the Society for Neuroscience, University of Hartford.
- 1980 "Norepinephrine and Serotonin Have Opposite Effects on the Excitability of Lateral Geniculate Neurons," Ninth Annual Meeting, New England Pharmacologists, University of Connecticut, Storrs.
- 1980 "Physiology of Norepinephrine in the Brain: Results from Single Neuron Recording," Amherst College, Amherst, Massachusetts.
- 1981 "An α -Adrenoceptor in Brain which Modulates Neuronal Excitability," Laboratory of Biochemical Genetics, National Heart, Lung and Blood Institute, NIH.
- 1982 "An Alpha-receptor which Modulates Neuronal Excitability: Studies on the Noradrenergic Pathway to the Lateral Geniculate Nucleus," Physiology Department, Armed Forces Radiobiology Research Institute.
- 1982 "An α -Adrenoceptor which Modulates Neuronal Excitability: Studies on the Physiology of the Noradrenergic Pathway to the Lateral Geniculate," Physiology Department, University of Maryland School of Medicine, Baltimore.
- 1982 "Cholecystokinin Octapeptide Enhances the Excitability of Cultured Mouse Spinal Neurons," Third Winter Neuropeptide Conference, Breckenridge, Colorado.
- 1982 "Alpha-Adrenoceptor Modulation of Neuronal Excitability in the Lateral Geniculate," Neuroscience Lecture Series, NIH.
- 1982 "Electrophysiological Analysis of Brain Serotonin Receptors," Biochemical Pharmacology Discussion Group, New York Academy of Sciences.
- 1982 "Physiological Analysis of Brain Norepinephrine Receptors," Brain Research Institute, Zurich.
- 1982 "Physiological Analysis of Brain Norepinephrine Receptors: Mechanism of Action of Antidepressants," Psychiatric Grand Rounds, Department of Psychiatry, University of California, Irvine.
- 1982 "Alpha-Adrenoceptor Modulation of Neuronal Excitability: Studies on the Noradrenergic Pathway to the Lateral Geniculate," Department of Neurology, University of California, Los Angeles.
- 1983 "Norepinephrine and the Lateral Geniculate Nucleus," Department of Cell Biology and Anatomy, Johns Hopkins University School of Medicine, Baltimore, Maryland.
- 1984 "Single Channel Properties of a Voltage-Activated Potassium Conductance in Cultured Hippocampal Neurons," UCLA Symposia on Molecular and Cellular Biology, Keystone, Colorado.
- 1984 "Potassium Conductances that Regulate the Excitability of Central Neurons: Voltage Clamp and Single-Channel Recordings," Department of Neurology, University of Pennsylvania, Philadelphia.
- 1985 "Electrical Excitability of Cultured Neurons from the Central Nervous System: Studies Using Voltage Clamp and Patch Clamp Techniques," Department of Anatomy, Emory University School of Medicine, Atlanta, Georgia.

- 1985 “Electrophysiological Analysis of Brain Noradrenergic Receptors,” Department of Neurology, University of Massachusetts Medical Center, Worcester.
- 1985 “Potassium Channels and the Excitability of Mammalian Central Neurons,” Medical Neurology Branch, National Institute of Neurological and Communicative Disorders and Stroke, NIH.
- 1985 “Excitability Mechanisms in CNS Neurons: From EEG to Patch Clamp,” Neurology Grand Rounds, The Oregon Health Sciences University (Good Samaritan Hospital), Portland.
- 1986 “Electrophysiological Analysis of Brain Noradrenergic Receptors,” Parkinson’s and Brain Peptides Seminar, Department of Neurology, Columbia University, College of Physicians and Surgeons, New York.
- 1986 “Voltage-Dependent K^+ Channels in Mammalian CNS Neurons,” Department of Neurobiology, University of California, San Francisco.
- 1986 “Potassium Channels in Neural and Endocrine Cells,” Center for Neurobiology, The Mount Sinai School of Medicine, New York.
- 1987 “Excitability Mechanisms in CNS Neurons: From EEG to Patch Clamp,” Neurology Grand Rounds, Georgetown University Hospital, Washington, D.C.
- 1988 “Cellular Pharmacology of Phencyclidine: Effects on NMDA-Receptors and Voltage-Dependent K^+ Channels,” Department of Pharmacology, St. Louis University School of Medicine, St. Louis, Missouri.
- 1988 “Actions of Phencyclidine (PCP): Receptors, Physiology, and Behavior,” Neuroscience Lecture, Amherst College, Amherst, Massachusetts.
- 1989 “New Research Strategies for Antiepileptic Therapy,” Current and Future Trends in Anticonvulsant, Anxiety and Stroke Therapy, Princeton Drug Research Symposia, Princeton, New Jersey.
- 1989 “Excitatory Amino Acids, Seizures and Antiepileptic Drugs,” NINDS Grand Rounds, NIH.
- 1990 “Techniques in Cellular Neurophysiology Short Course,” Potomac Chapter, Society for Neuroscience, Bethesda, Maryland.
- 1990 “Glyburide-Sensitive Voltage-Gated K^+ Channels in Hippocampal Cells,” Sulfonylureas: Molecular Mechanisms of Action Workshop, NIH.
- 1990 “Electrophysiological Approaches to Understanding Hallucinogens,” NIDA Addiction Research Center, Baltimore, Maryland.
- 1990 “Novel Strategies for Antiepileptic Drug Development,” ICI Pharmaceuticals, Wilmington, Delaware.
- 1990 “Pharmacological Regulation of Voltage-Dependent Potassium Channels in Central Neurons,” Department of Pharmacological and Physiological Sciences, University of Chicago.
- 1990 “Excitatory Amino Acid Receptors: Novel Research Strategies for Antiepileptic Therapy,” Excitatory Amino Acid Receptors in the Brain: Functions and Disorders, Montréal Neurological Institute.
- 1991 “Therapeutic Potential of Weak Uncompetitive NMDA Antagonists for Neurological Disorders,” Parke-Davis Pharmaceutical Research Division, Warner-Lambert Co., Ann Arbor, Michigan.
- 1991 “Interaction of Dissociative Anesthetics with Voltage-Dependent K^+ and Ca^{2+} Channels and NMDA Receptors in Hippocampal Neurons: Diverse Blocking Mechanisms,” Department of Physiology, University of Maryland School of Medicine, Baltimore.
- 1991 “Therapeutic Potential of Weak Uncompetitive NMDA Antagonists for Neurological Disorders,” Neurogen Corporation, Baltimore, Maryland.

- 1991 “Pharmacological Regulation of K⁺ Channels in CNS Neurons,” SmithKline Beecham Pharmaceuticals, Philadelphia, Pennsylvania.
- 1991 “Prospects for the Development of Non-Competitive NMDA Antagonists in the Treatment of Epilepsy,” Focus on Epilepsy: Cellular Mechanisms and Pharmacological Approaches, St. Adèle, Quebec City.
- 1991 “Anticonvulsant 1-Phenylcyclohexylamine Analogs: Uncompetitive NMDA antagonists with Less Motor Toxicity than PCP,” Third French-U.S. Joint Seminar, Multiple *Sigma* and PCP Receptor Ligands, La Grande Motte, France.
- 1991 “Symposium on Receptors and Disease: The Glutamate Receptor and Epilepsy” (discussant of presentation by Dr. J.O. McNamara), 116th Annual Meeting of the American Neurological Association, Seattle, Washington.
- 1991 “Ion Channels, Drugs and the Treatment of Epilepsy,” Department of Neuroscience, Johns Hopkins University School of Medicine, Baltimore, Maryland.
- 1991 “Glyburide Sensitive K⁺ Channels in Central Neurons,” Miles Laboratories, West Haven, Connecticut.
- 1991 “Mechanisms of Action of Antiepileptic Drugs,” Montreal Neurological Institute, Montreal.
- 1991 “Potassium Channels and Epilepsy,” Investigator’s Workshop, Annual Meeting, American Epilepsy Society, Philadelphia, Pennsylvania.
- 1992 “Anticonvulsant Drugs,” Neurology Grand Rounds, VA Medical Center, Washington, D.C.
- 1992 “Ion Channels as Targets for Stroke Therapy: Focus on Excitatory Amino Acid Receptors and ATP-Sensitive K⁺ Channels,” Wyeth-Ayerst Research, Princeton, New Jersey.
- 1992 “Theoretical and Practical Considerations in the Use of Excitatory Amino Acid Antagonists in Epilepsy Therapy,” XVIIIth C.I.N.P. Congress, Nice, France.
- 1992 “Cellular Electrophysiological Studies on Noncompetitive NMDA and Non-NMDA Antagonists: Implications for Neurological Therapeutics,” First International Congress of the Polish Neuroscience Society, Warsaw.
- 1992 “Potential Antiepileptic Drugs that Act on Excitatory Neurotransmitter Systems,” Excitatory and Inhibitory Neurotransmitters in Epilepsy and New Anti-Epileptic Drugs,” Annual Meeting, American Epilepsy Society, Seattle, Washington.
- 1993 “Strategies for Antiepileptic Drug Development,” Future Applications of Cellular Biology, Marion Merrell Dow, Kona, Hawaii.
- 1993 “ATP-Sensitive K⁺ Channels,” Winter Conference on Brain Research, Whistler, B.C., Canada.
- 1993 “Therapeutic Potential of Noncompetitive Excitatory Amino Acid Antagonists,” Lilly Research Laboratories, Eli Lilly and Company, Indianapolis, Indiana.
- 1993 “Antiepileptic Drug Mechanisms: Voltage-dependent Na⁺ Channels,” 20th International Epilepsy Congress, Oslo Norway.
- 1993 “Uncompetitive NMDA Antagonists,” Antiepileptic Drugs in the 1990s: Current and Investigational Therapies (Focus on Epilepsy II), Whistler, B.C., Canada.
- 1994 “Noncompetitive Antagonism of AMPA and NMDA Receptors by 2,3-Benzodiazepines and Channel Blockers,” Neuroscience Seminar Series, Uniformed Services University of the Health Sciences, Bethesda, Maryland.
- 1994 “New Anticonvulsant Mechanisms: K⁺ Channels,” International Union of Pharmacologists, 125th Meeting, Toronto, Canada.

- 1994 "Cellular Physiology of Antiepileptic Drugs: Clues for Improved Therapies," New Frontiers in the Neuroscience of Epilepsy, Bowman Gray School of Medicine, Wake Forest University, Winston-Salem, North Carolina.
- 1994 "The Pharmacology of NMDA Antagonists," Behavioral Pharmacology Research Unit, The Johns Hopkins University School of Medicine, Baltimore, Maryland.
- 1994 "Excitatory Amino Acid Antagonists for Epilepsy Therapy: New Approaches," Ninth Rinshoken International Conference, Tokyo.
- 1995 "Potassium Channels Modulators in Neurological Disease," IBC USA Conferences, Washington, D.C.
- 1995 "New Developments in Antiepileptic Drugs," Neurology Grand Rounds, University of Maryland, School of Medicine, Baltimore, Maryland.
- 1995 "Promise and Progress in the Treatment of Seizure Disorders," Winter Conference on Brain Research, Steamboat Springs, Colorado.
- 1995 "Pharmacological Properties of AMPA Receptor Isoforms," Neuroscience Seminar, University of Massachusetts Medical Center, Worcester, Massachusetts.
- 1995 "Barbiturates, Neuroactive Steroids and Dicarbamates: New Directions for Antiepileptic Drug Development," GABA and Epileptogenesis (Focus on Epilepsy III), Whistler, B.C., Canada.
- 1995 "Excitatory Amino Acids and Epilepsy: Etiology and Therapy (Drug Targeting AMPA Receptor Subtypes)," Fourth International Congress on Amino Acids, Vienna, Austria.
- 1995 "Excitatory Amino Acids, Epilepsy and Remacemide." and "Rational Development of Antiepileptic Drugs," Twenty-first International Congress on Epilepsy, Sydney, Australia.
- 1995 "Pharmacology and Gating of AMPA Receptors," Membrane Ion Channels: Structure and Function, Fogarty International Center, National Institutes of Health, Bethesda, Maryland.
- 1995 "Modulation of GABA_A Receptors by Neuroactive Steroids: Epilepsy Therapy," Pre- and Postsynaptic Modulation of the GABA_A Receptors: Molecular Pharmacology and Therapeutic Implication, Satellite Symposium, 25th Annual Meeting, Society for Neuroscience, San Diego, California.
- 1996 "K⁺ Channels as Drug Targets in the Treatment of Neurological Diseases," Voltage-Gated Ion Channel Modulators, IBC USA Conferences, Philadelphia.
- 1996 "The NMDA Receptor as Target for AEDs," Molecular Targets and Cellular Mechanisms of Antiepileptic Drugs, Genoa, Italy.
- 1996 "AMPA Receptors as Antiepileptic Drug Targets," Epilepsy: Advances in Understanding and Latest Drug Developments, IBC USA Conferences, Orlando, Florida.
- 1996 "Ion Channels and AED Development," Third International Symposium on Basic Mechanisms of the Epilepsies, San Diego, California.
- 1996 "Mechanism Specific Pathways for New AED Discovery," Antiepileptic Drug Trials III (University of Pennsylvania School of Medicine), Charleston, South Carolina.
- 1996 "Basic Mechanisms of Epilepsy," Neurology Resident Lecture, The Johns Hopkins University School of Medicine, Baltimore.
- 1996 "Pharmacological Properties and Mechanisms of Action of the New Antiepileptic Drugs," Third Eilat Conference on New Antiepileptic Drugs, Eilat, Israel.
- 1996 "Basic Mechanisms of Epilepsy: What Can We Modulate with Antiepileptic Drugs?" New Antiepileptic Drug Development – Focus on Mechanism of Action, Annual Meeting, American College of Clinical Pharmacology, Philadelphia.

- 1996 "Approaches to Hormonal Therapy of Epilepsy," Investigator's Workshop, Annual Meeting, American Epilepsy Society, San Francisco, California.
- 1997 "Neuroactive Steroids and Epilepsy," Department of Pharmacology and Experimental Therapeutics, Boston University School of Medicine.
- 1997 "Mechanisms of AED Action: Present & Future," Symposium on Epilepsy, Millennium Pharmaceuticals, Cambridge, Massachusetts.
- 1997 "The GABA Hypothesis," 8th International Bethel-Cleveland Clinic Epilepsy Symposium, Bielefeld, Germany.
- 1997 "Antiepileptic Drugs for the 21st Century," Spring Epilepsy Research Conference, Grand Cayman, British West Indies.
- 1997 "New Strategies for Antiepileptic Drug Discovery," Lilly Research Laboratories, Eli Lilly and Company, Indianapolis, Indiana.
- 1977 "Modulation of AMPA Receptors," Department of Psychiatry, University of Illinois School of Medicine, Chicago.
- 1977 "Neuroprotection Strategies: Excitatory Amino Acid Systems," Focus on Epilepsy IV: Seizure-Induced Brain Damage, Hôtel Le Chantecler, Saint-Adèle, Canada.
- 1997 "Anticonvulsants for the 21st Century," Plenary Lecture in Symposium "Emerging Strategies for the 21st Century," Child Neurology Society Annual Meeting, Phoenix, Arizona.
- 1998 "Glutamate Receptors as Targets of Antiepileptic Drugs," Department of Pharmacology, Uniformed Services University of the Health Sciences, Bethesda, Maryland.
- 1998 "Sodium and Potassium Channels as Targets for Antiepileptic Drugs," Department of Pharmacology and Toxicology, University of Utah, Salt Lake City, Utah.
- 1998 "Preclinical AED Development: Model Specific vs. Mechanism Specific," Antiepileptic Drug Trials IV (University of Pennsylvania School of Medicine), Charleston, South Carolina
- 1998 Neurology Grand Rounds, Ottawa General Hospital, Ottawa, Canada
- 1998 "Cellular Mechanisms of Antiepileptic Drug Action," Department of Neurology, Yale University School of Medicine, New Haven, Connecticut.
- 1998 "Glutamate Neurotransmission in Epilepsy: New Treatment Strategies," The Glutamate Cascade: Common Pathways of Central Nervous System Disease States, National Institutes of Health, Bethesda, Maryland.
- 1998 "Targeting Ion Channels for Neuroprotection in Epilepsy," Nerve Cell Death and Neuroprotection in Epilepsy, Program in Neuroscience, University of Maryland, Baltimore.
- 1998 "ADCI: An NMDA Receptor and Sodium Channel Antagonist with Broad Spectrum Anticonvulsant Activity and Favorable Toxicity Characteristics," Fourth Eilat Conference on New Antiepileptic Drugs, Eilat, Israel.
- 1998 "Mechanisms of Action of Antiepileptic Drugs," Merritt-Putnam Lectures on Epilepsy (sponsored by American Epilepsy Society), Costa Mesa, California.
- 1998 "Kainate Receptors and Synaptic Plasticity in the Amygdala," *Neuropharmacology* Symposium on Ionotropic Glutamate Receptors (satellite to Society for Neuroscience Meeting), Los Angeles.
- 1998 "AMPA Receptor Antagonists and Allosteric Modulators in Epilepsy," Excitatory Amino Acids - Ten Years Later, Manaus, Brazil.
- 1998 "Progesterone Metabolites as Modulators of Seizure Susceptibility," Symposium, Annual Meeting, American Epilepsy Society, San Diego.

- 1999 “Neurosteroids, GABA and Seizures,” Department of Neurology, Children’s National Medical Center, Washington, D.C.
- 1999 “New Developments in Epilepsy Therapy: Potential Ion Channel Modulators,” DuPont Pharmaceuticals Company, Wilmington, Delaware.
- 1999 “Channel Blocking NMDA Receptor Antagonists,” Institute for Biological Sciences, National Research Council, Ottawa, Canada.
- 1999 “Therapeutic Agents Targeting Brain Glutamate Systems,” Special Keynote Presentation, New Developments in Glutamate Pharmacology, IBC USA Conferences, Orlando, Florida.
- 1999 “AMPA and Kainate Receptors as Antiepileptic Drug Targets,” The Sangamon County Chapter of the Society for Neuroscience, Southern Illinois University School of Medicine, Springfield, Illinois.
- 1999 “Therapeutic Agents Targeting Brain Glutamate Systems,” Department of Pharmacology, University of Colorado Health Sciences Center, Denver, Colorado.
- 1999 “Ligand-gated Ion Channels As Therapeutic Targets,” Generalized Epilepsies: From Gene to Clinic (Focus on Epilepsy V), Québec City, Canada.
- 1999 “Low Affinity Channel Blockers: Past Present and Future,” 6th International Congress on Amino Acids, University of Bonn, Germany.
- 1999 “Neurosteroids in Epilepsy,” Interdisciplinary Program in Neuroscience, Georgetown University.
- 1999 “Molecular Targets for Neuroprotection,” Speaker and Chair, Symposium on Neuroprotection: Implications for the Future Clinical Management of Epilepsy, Annual Meeting, American Epilepsy Society, Orlando.
- 2000 “New Developments in Antiepileptic Drugs,” Maryland Psychiatric Research Center, University of Maryland, Catonsville.
- 2000 “New Advances in Epilepsy Treatment,” Spring Brain Conference, Sedona, Arizona.
- 2000 “Therapeutic Applications of Potassium Channel Modulators,” Plenary Speaker and Chair, Potassium Channels: Structure, Function and Therapeutic Utilities, Keystone Symposia on Molecular & Cellular Biology, Granlibakken Resort, Tahoe City, California.
- 2000 “Ion Channels and Epilepsy,” ICAgen, Research Triangle Park, North Carolina.
- 2000 “Neuroactive Steroids,” Fifth Eilat Conference on New Antiepileptic Drugs, Eilat, Israel.
- 2000 “GABA Involvement and the Case for Neurosteroids,” University of Washington Pediatric Epilepsy Research Symposium on Epilepsy, Infantile Spasms and Developmental Encephalography, Seattle, Washington.
- 2000 “NMDA, AMPA and Kainate Antagonists as Potential Antiepileptic Drugs,” British Pharmacological Society Lecture in Honor of Retirement of Brian Meldrum, University of Bradford.
- 2001 “GluR5 Kainate Receptors and Epileptogenesis in the Amygdala,” Killam Lecture, Montreal Neurological Institute, McGill University, Montréal, Québec, Canada.
- 2001 “Novel Approches to Targeting Glutamate and GABA Receptors,” Treatment of Epileptic Syndromes: From Molecular Targets to Quality of Life (Focus on Epilepsy VI), Mont-Tremblant, Québec, Canada.
- 2001 “Kainate Receptors and Synaptic Plasticity in the Amygdala,” Department of Neuroscience, Albert Einstein College of Medicine, Bronx, New York.
- 2001 “The Mechanisms of Old and New Antiepileptic Drugs: Have Previous Strategies Preselected Drugs Which Act by Old Mechanisms?” New Horizons in the Development of Antiepileptic Drugs, Satellite to Annual Meeting, American Epilepsy Society, Philadelphia.

- 2001 "A Model of Catamenial Epilepsy: Role of Neurosteroids," Neuroendocrinological Aspects of Epilepsy, Annual Meeting, American Epilepsy Society, Philadelphia.
- 2002 "Hormonal Regulation of Seizures," Neurology Grand Rounds, The Ohio State University, Columbus.
- 2002 "Neurosteroids and Epilepsy," Department of Pharmacology, University of Tennessee Health Science Center, Memphis.
- 2002 "Women's Issues in Epilepsy" (half-day course), 54th Annual Meeting, American Academy of Neurology, Denver.
- 2002 "A New Look at the Role of Kainate Receptors in Epilepsy," 35th Winter Conference on Brain Research, Snowmass Village, CO.
- 2002 "Neurological Diseases and Neuroprotection Therapeutic Strategy: Role of Antiepileptic Drugs," satellite symposium to "Sixth Eilat Conference on New Antiepileptic Drugs," Taormina, Sicily.
- 2002 "Kainate Receptor Mediated Long-Term Plasticity, Seizures and Epileptogenesis in the Amygdala," The Amygdala in Brain Function: Basic and Clinical Approaches, New York Academy of Sciences, Galveston Island, Texas.
- 2002 "Hormonal Regulation of Seizure Susceptibility: Catamenial Epilepsy and Stress," Neurology Grand Rounds, The University of Washington School of Medicine, Seattle.
- 2002 "Targeting Glutamate Receptors for Neuroprotection in Epilepsy: The Calcium Connection," Neuroprotection in Epilepsy, Montefiore Medical Center and Albert Einstein College of Medicine, New York.
- 2003 "Hormonal Regulation of Seizure Susceptibility," Epilepsy Research Seminar Series, Division of Neurology, Children's Hospital of Philadelphia and University of Pennsylvania.
- 2003 "Molecular Targets for Drug Development for Neuroprotection: AMPA, NMDA, Kainate and K^+ , Ca^{++} , Na^+ Channels as Potential Targets," Clinical Trials In Neuroprotection, University of Pennsylvania, Key Biscayne, Florida.
- 2003 "Neurosteroid Regulation of Seizure Susceptibility: Catamenial Epilepsy and Stress," XXXIII Annual Congress of the International Society of Psychoneuroendocrinology, Pisa, Italy.
- 2003 "Women's Issues in Epilepsy" (half-day course), 55th Annual Meeting, American Academy of Neurology, Honolulu.
- 2003 "Tolerance: Neuronal Mechanisms," Long-Term Efficacy of Antiepileptic Drugs and the Phenomenon of Tolerance, Medical College of Wisconsin, Milwaukee.
- 2003 "Role of Kainate Receptors in Synaptic Plasticity and Epileptogenesis," Amygdala, Seizures and Epileptogenesis (Focus On Epilepsy VII), Montréal Neurological Institute, Montréal.
- 2003 "Understanding the Optimal Treatment Approach to Alzheimer's Disease: The NMDA Receptor as a Novel Target," 11th National Alzheimer's Disease Education Conference, Chicago.
- 2003 "Beyond Acetylcholine: Novel Targets in Alzheimer's Disease," U.S. Geriatric and Long-Term Care Congress, San Francisco.
- 2003 "Hormones and Epilepsy," Neurology Grand Rounds, Georgetown University, Washington, D.C.
- 2003 "Neurosteroid Modulation of Seizure Susceptibility," Departments of Pharmacology and Neurology, Medical College of Ohio.
- 2003 "Neurosteroid Update: Integrating the Basic and Clinical Data," 42nd Annual Meeting, American College of Neuropsychopharmacology, San Juan.

- 2004 “Neurosteroids and the Regulation of Seizure Susceptibility,” Center for Neuroscience Research, Children’s Research Institute, Children’s National Medical Center, Washington, D.C.
- 2004 “Cognitive Neuropsychiatry of Glutamate,” Society for Behavioral and Cognitive Neurology Workshop, 15th Annual Meeting, American Neuropsychiatric Association, Bal Harbour, Florida.
- 2004 “What is the Rationale for New Treatment Strategies in AD?,” Assessing Current Practice in Alzheimer’s Disease, American Association for Geriatric Psychiatry 17th Annual Meeting, Baltimore.
- 2004 “Kainate Receptors in the Amygdala,” The Synapse: Molecular Mechanisms of Plasticity, Celebrating the Opening of the Porter Neuroscience Research Center, Harbourtowne Conference Center, St. Michaels, Maryland.
- 2004 “Neurosteroids and Epilepsy,” Pharmacology Department Seminar, Uniformed Services University of the Health Sciences, Bethesda.
- 2004 “How Do Hormones Influence Seizures?” National Capital Cure Epilepsy Conference, American Association for the Advancement of Science, Washington, D.C.
- 2004 “Catamenial Epilepsy,” NINDS Grand Rounds, Clinical Center, National Institutes of Health, Bethesda.
- 2004 “Physiology of Kainate Receptors and their Role in Synaptic Plasticity and Epileptogenesis in the Amygdala,” Department of Physiology and Neuroscience Center, Dartmouth Medical School, Hanover, New Hampshire.
- 2004 “Women’s Issues in Epilepsy” (half-day course), 56th Annual Meeting, American Academy of Neurology, San Francisco.
- 2004 “Catamenial Epilepsy,” Neurology Grand Rounds, George Washington University, Washington, D.C.
- 2004 “Role of Neurosteroids in the Regulation of Seizure Susceptibility,” Department of Neuroscience Seminar, Albert Einstein College of Medicine, Bronx, New York.
- 2004 “The ABCs of Alzheimer’s Disease Management: Understanding the Latest Advances and Rationale of Pharmacological Management,” Canadian Association of Geriatric Psychiatry, Montreal.
- 2004 “Neurosteroids and Epilepsy – How Menstruation, Stress and Hypogonadism Influence Seizure Susceptibility,” Epilepsy Center, Barrow Neurological Institute, Phoenix, Arizona.
- 2004 “The Role of Kainate Receptors in Epilepsy,” Investigator’s Workshop, Annual Meeting, American Epilepsy Society, New Orleans, Louisiana.
- 2004 “Molecular Mechanisms of Action of Antiepileptic Drugs in the Treatment of Bipolar Disorder: How Can More Effective Agents Be Developed?,” American College of Neuropsychopharmacology Annual Meeting, San Juan, Puerto Rico.
- 2005 “Neurosteroids and Epilepsy,” Grand Rounds, Department of Neurology, New York Presbyterian Hospital–Cornell Medical Center.
- 2005 “Neurosteroids and Epilepsy – How Menstruation, Stress and Hypogonadism Influence Seizure Susceptibility,” Grand Rounds, The Neurosciences Institute, Albany Medical Center.
- 2005 “Modulation of GABA_A Receptors by Neurosteroids,” Frontiers in Neuroscience III, Montreal Neurological Institute, Mont-Tremblant, Québec, Canada.
- 2005 “Memantine: Mechanism of Action in Alzheimer’s Disease,” Cloister Eberbach, Eltville, Germany.

- 2005 “Episodic Nervous System Disorders: Pathophysiology to Neurotherapeutics,” symposium on “Antiepileptic Drugs: Not Just for Epilepsy,” American Society for Experimental NeuroTherapeutics Annual Meeting, Washington, D.C.
- 2005 “Molecular Targets vs Models for New Drug Development,” Antiepileptic Drug Trials VIII (University of Pennsylvania Medical Center), Sonesta Beach Resort, Key Biscayne, Florida.
- 2005 “Epilepsy,” Demystifying Medicine Lecture Series, Office of Intramural Research, Office of the Director, NIH.
- 2005 “Avenues of Neuroprotection in Epilepsy,” Disease Modification in Epilepsy, Comprehensive Epilepsy Center, Weill Medical College/New York-Presbyterian Hospital, New York.
- 2005 “Special lecture: Modulation of Ion Channel by Antiepileptic Drugs,” and “Neurosteroids and Epilepsy – How Menstruation, Stress and Hypogonadism Influence Seizure Susceptibility,” Korean Epilepsy Society, Seoul.
- 2005 “Neurosteroids and Epilepsy,” Neurology Grand Rounds, Keck School of Medicine, University of Southern California, Los Angeles.
- 2005 “Neurosteroids and Epilepsy – How Menstruation, Stress and Hypogonadism Influence Seizure Susceptibility,” Department of Pharmacology, School of Medicine, University of Maryland at Baltimore.
- 2005 “Neurosteroids and Brain Excitability,” Neurosteroids – Effects and Clinical Consequences, 26th International Epilepsy Congress, Paris.
- 2005 “Kainate Receptors in the Amygdala: Role in Seizures and Epileptogenesis,” Epilepsy Research Seminar, Department of Pharmacology, and Neurology Grand Rounds, Department of Neurology, Emory University, Atlanta.
- 2005 “Why Women With Epilepsy Tend to Have Seizures Around the Time of Menstruation,” Neuroscience Program, Williams College, Williamstown, Massachusetts.
- 2005 “Antiepileptic Drugs in the Preclinical Pipeline,” New Horizons in the Development of Antiepileptic Drugs: Innovative Strategies, Satellite to Annual Meeting, American Epilepsy Society, Washington, D.C.
- 2005 “Stress, Seizures and the GABA System,” Plenary Symposium on Maladaptive Neuroplasticity in Stress and Epilepsy, Annual Meeting, American Epilepsy Society, Washington, D.C.
- 2006 “Which Molecular Targets Exist for New Drugs Preventing or Reversing Epilepsy and Drug Resistance?” UCB Research Day – Prevention or Reversal of Epilepsy and Pharmacoresistance: Potential for Future Drug Discovery and Development, Chateau du Lac, Genval, Belgium.
- 2006 “Pathophysiology of Episodic Neurological Disorders,” Continuing Medical Education Conference on Depression in Neurologic Disease, Weill Medical College of Cornell University, New York.
- 2006 “Lessons from Epilepsy,” Symposium on “Predicting Treatment Efficacy to Analgesic Drugs in Neuropathic Pain: Lessons from the Worlds of Epilepsy and Depression,” 25th Annual Scientific Meeting of the American Pain Society, San Antonio, Texas.
- 2006 “Neurosteroids in Seizure Disorders and Depression and the Development of Neurosteroid Analogs,” Symposium on “Neurosteroids as Candidate Modulators of Pathophysiology and Novel Therapeutic Agents,” 61st Annual Convention, Society of Biological Psychiatry, Toronto, Canada.
- 2006 “Hormonal Regulation of Seizure Susceptibility,” Department of Biomedical Sciences, University of Modena and Reggio Emilia, Modena, Italy.

- 2006 “Neurosteroids and Epilepsy – How Menstruation, Stress and Hypogonadism Influence Seizure Susceptibility,” Grand Rounds, Department of Neurology, Jefferson Medical College, Thomas Jefferson University, Philadelphia.
- 2006 “Neurosteroids and Epilepsy – How Menstruation, Stress and Hypogonadism Influence Seizure Susceptibility,” Grand Rounds, Department of Neurology, West Los Angeles Healthcare Center, Veterans Affairs Greater Healthcare System, Los Angeles.
- 2007 “Novel Antiepileptic Drugs,” Ion Channel Modulators and Antiepileptics, Innovative Drug Development For Headache Disorders, 16th International Headache Research Seminar, University of Copenhagen, Glostrup Hospital, Copenhagen.
- 2007 “Convection Enhanced Delivery of Peptide Toxins for the Treatment of Focal Epilepsies,” 4th Conference on Epileptogenesis, Santa Croce in Fossabanda, Pisa, Italy.
- 2007 “Endogenous Neurosteroids as Regulators of the Transition to the Ictal State,” IX Workshop on Neurobiology of Epilepsy (WONOE 2007), Sheraton Langkawi Resort, Teluk Nibong, Langkawi Island, Malaysia.
- 2007 “Hormones and Epilepsy: How Menstruation, Stress, and Hypogonadism Influence Seizure Susceptibility,” NIH Clinical Center Grand Rounds, National Institutes of Health, Bethesda, Maryland.
- 2007 “Neurosteroids as Therapeutic Agents: Seizure Protection and Antiepileptogenesis,” 27th International Epilepsy Congress, Suntec Singapore International Convention & Exhibition Centre, Singapore.
- 2007 “Recent Advances in Antiepileptic Drug Mechanisms: New Molecular Targets,” 27th International Epilepsy Congress, Suntec Singapore International Convention & Exhibition Centre, Singapore.
- 2007 “Anatomy and Physiology of Neuron-Specific K⁺ Channels,” 27th International Epilepsy Congress, Suntec Singapore International Convention & Exhibition Centre, Singapore.
- 2007 “Neurosteroids and Epilepsy – How Menstruation, Stress and Hypogonadism Influence Seizure Susceptibility,” UCI Epicenter, University of California, Irvine.
- 2007 “Neurosteroids as Therapeutic Agents: Seizure Protection, Antiepileptogenesis, Clinical Trials with Ganaxolone,” 46th Annual Meeting American College of Neuropsychopharmacology, Boca Raton Resort & Club, Boca Raton, Florida.
- 2008 “Neurosteroids as Regulators of Seizure Susceptibility: Seizure Protection, Antiepileptogenesis, Clinical Trials with Ganaxolone,” 41st Winter Conference on Brain Research, Snowbird, Utah.
- 2008 “Convection-Enhanced Delivery,” 4th Workshop on New Horizons in the Development of Antiepileptic Drugs, Clontarf Castle, Dublin.
- 2008 “Revolutionary Treatments: New CNS Drug Targets and Breakthrough Epilepsy Treatment Strategies,” Epilepsy Pipeline Update: Portal into CNS Opportunities, Epilepsy Therapy Project, Hotel Monaco, San Francisco.
- 2008 “Basic Science Mini-Symposium: Future Directions (Rational Polytherapy: The Ketogenic Diet Plus),” International Symposium on Dietary Therapies for Epilepsy and Other Neurological Disorders, Ritz Carlton Hotel, Phoenix.
- 2008 “Neurosteroids and Epilepsy – How Menstruation, Stress and Hypogonadism Influence Seizure Susceptibility,” Neurology Grand Rounds, University of California, Los Angeles.
- 2008 “New Epilepsy Treatment Approaches – Focus on Convection-Enhanced Delivery,” Center for Integration of Medicine & Innovative Technology/Neurotechnology Industry Organization Epilepsy Innovation Summit, Westin Copley Place, Boston.

- 2008 “Epilepsy and Migraine,” Ninth Eilat Conference on New Antiepileptic Drugs, Sitges, Spain.
- 2008 “Posttraumatic Epilepsy: Update on the Problem and the Status of Animal Models,” University of California Neurotrauma Symposium, Quail Lodge, Carmel, California.
- 2008 “Neurosteroids and Epilepsy – How Menstruation, Stress and Hypogonadism Influence Seizure Susceptibility,” Neurology Grand Rounds, Stanford University, Palo Alto, California.
- 2008 “Neuroendocrinology in Epilepsy – Brain, the Third Gonad: How CNS Steroids Interact with Peripheral Steroids to Affect Seizures,” Neuroendocrinology Special Interest Group, Annual Meeting, American Epilepsy Society, Seattle, Washington.
- 2009 “Convection-Enhanced Delivery for the Treatment of Partial Seizures,” Pipeline Session, American Society for Experimental NeuroTherapeutics, 11th Annual Meeting, Crystal Gateway Marriott, Arlington, Virginia.
- 2009 “New mechanisms vs. old models: Is it time for out of the box thinking?,” Antiepileptic Drug Trials X (University of Pennsylvania School of Medicine), Biltmore Hotel, Coral Gables, Florida.
- 2009 “New Approaches to Epilepsy Therapeutics,” Fourth International Workshops, Jasper’s Basic Mechanisms of the Epilepsies, Yosemite National Park, California.
- 2009 “Neurosteroids and Epilepsy — How Menstruation, Stress and Hypogonadism Influence Seizure Susceptibility,” Johns Hopkins Neurology Grand Rounds, Baltimore, Maryland.
- 2009 “Neurosteroids and Epilepsy — How Menstruation, Stress and Hypogonadism Influence Seizure Susceptibility,” Department of Anesthesiology, Washington University, St. Louis.
- 2009 “New Molecular Targets for Antiepileptic Drugs,” 13th Annual Scientific Meeting of the Epilepsy Association of Thailand, Novotel Hotel, Bangkok.
- 2009 “From NINDS Lab to Clinical Trial: A Case Study in the Translation of a Novel Epilepsy Drug” and “Pharmaceutical Development in the Academic Setting,” Second Annual Summer Course for Clinical and Translational Research in the Neurosciences, American Neurological Association, Vail Marriott Mountain Resort, Vail, Colorado.
- 2009 “Migraine and Epilepsy,” 14th International Headache Congress, American Headache Society and the International Headache Society, Philadelphia, Pennsylvania.
- 2009 “Migraine, Epilepsy and Migralepsy: Myths and Realities,” 7th Headache Research Summit, National Headache Foundation, Hyatt Regency Hotel, Chicago, Illinois.
- 2009 “Migraine and Epilepsy,” West Los Angeles VA Healthcare Center Neurology Grand Rounds, Los Angeles, California.
- 2009 “The Future of Epilepsy Therapy,” William G. Lennox Lecture, 63rd Annual Meeting, American Epilepsy Society, Boston, Massachusetts.
- 2010 “The Science of Epilepsy: Mechanisms of Action of Drugs and Biologics” and “Intrapulmonary Propofol Analogs,” 2010 Epilepsy Pipeline Update: Portal Into CNS, Epilepsy Therapy Project, San Francisco.
- 2010 “Neurosteroids and Epilepsy — How Menstruation, Stress and Hypogonadism Influence Seizure Susceptibility,” Neurology Grand Rounds, University of California, San Diego.
- 2010 “The Other Adrenal Effect — The DOC Hypothesis,” 2010 Infantile Spasms Summit: the Unique Mechanistic Properties of ACTH, Hôtel Plaza Athénée, New York.
- 2010 “Emerging Antiepileptic Drug Mechanisms — New Molecular Targets,” 9th European Congress on Epilepsy, Rhodes, Greece.
- 2010 “Neuroprotection in Epilepsy: Promise or Pipe Dream?,” Keynote Address, 2010 Epilepsy Update, Barrow Neurological Institute, Phoenix.

- 2010 “Central Nervous System Channels: Review of the Basics,” Channel Surfing: Impact On Treatment Strategies, AET Symposium, American Epilepsy Society, San Antonio, Texas.
- 2010 “Neurosteroids in Men with Epilepsy,” Hormonal Issues in Men with Epilepsy, Neuroendocrine Special Interest Group, 64th Annual Meeting, American Epilepsy Society, San Antonio, Texas.
- 2011 “Neurosteroids for Epilepsy, Traumatic Brain Injury and Fragile X Syndrome,” Panel: Translational Studies of Neuroactive Steroids: Targets, Biomarkers and Potential Therapeutics for Neuropsychiatric Disease, 2011 Winter Conference on Brain Research Keystone, Colorado.
- 2011 “Migraine,” The Fourth Annual Headache Cooperative of the Pacific Winter Symposium, Ojai Valley Inn, Ojai, California.
- 2011 “Pulmonary Drug Delivery to Treat Epilepsy and Migraine,” Pipeline Session, American Society for Experimental NeuroTherapeutics, 12th Annual Meeting, Bethesda, Maryland.
- 2011 “Complexities of Migraine Headaches,” 4th Annual Chair Summit, Loews Annapolis Hotel, Annapolis, Maryland.
- 2011 “Migraine and Epilepsy – Shared Mechanisms within the Family of Episodic Disorders,” NINDS Grand Rounds Lecture Series, NIH Clinical Center, Bethesda, Maryland.
- 2011 “Neurobiological Mechanisms of Comorbidities,” Investigators’ Workshop (organizer), 65th Annual Meeting, American Epilepsy Society, Baltimore, Maryland.
- 2011 “Migraine and Epilepsy – Shared Mechanisms within the Family of Episodic Disorders,” Neurology Grand Rounds, University of Vermont College of Medicine, Burlington.
- 2012 “Inhaled Treatments to Stop Seizures Before they Spread,” Epilepsy Pipeline Update, Epilepsy Therapy Project, Hyatt Embarcadero Hotel, San Francisco.
- 2012 “New Approaches to Epilepsy Therapy,” Department of Neurology, University Medical Center Magdeburg A.ö.R, Otto-von-Guericke-University, Magdeburg, Germany.
- 2012 “Intracranial Delivery of Antiseizure Substances,” Deutsche Gesellschaft für Epileptologie (German Society for Epileptology) Annual Congress, Kultur- & Kongresszentrum Liederhalle, Stuttgart, Germany.
- 2012 “Neurosteroids and Epilepsy – How Menstruation, Stress and Hypogonadism Influence Seizure Susceptibility,” Academia Sinica, Taipei, Taiwan.
- 2012 “New Approaches to Epilepsy Therapy,” National Taiwan University Hospital, Taipei, Taiwan.
- 2012 “Neurotherapeutics Discovery and Development in the Academic Setting: A New Paradigm,” Neurotranslation: Neurologists and Neuroscientists Defining the Next Generation of CNS Therapies, American Academy of Neurology 64th Annual Meeting, New Orleans, Louisiana.
- 2012 “Mechanisms and Models for Migraine,” Eleventh Eilat Conference on New Antiepileptic Drugs, Eilat, Israel.
- 2012 “How to Do Translational Neurology in Academia,” American Neurological Association Fourth Annual Translational and Clinical Research Course in the Neurosciences, San Francisco.
- 2012 “New Directions in Epilepsy Treatment,” Encuentro de Expertos en Epilepsia VII, Casa America, Madrid, Spain.
- 2012 “Intrinsic Severity As a Determinant of Antiepileptic Drug Refractoriness,” Second Halifax International Epilepsy Retreat “Pharmacoresistance in Epilepsy,” Oak Island Resort & Marina, Nova Scotia, Canada

- 2012 “AMPA Receptors: A New Target in the Treatment of Epilepsy,” 10th European Congress on Epileptology, London.
- 2012 “Migraine and Epilepsy – Shared Mechanisms within the Family of Episodic Disorders,” Neurology Grand Rounds, New York Medical College, Valhalla, New York.
- 2012 “How do we treat epileptic seizures with medications?,” Living with Epilepsy, a conference about epilepsy (sponsored by Stanford University), Li Ka Shing Center for Learning and Knowledge, Stanford University, Stanford, California.
- 2013 “Neurosteroids and Epilepsy – How Menstruation, Stress and Hypogonadism Influence Seizure Susceptibility,” Basic Medical Sciences Seminar Series, University of Arizona College of Medicine, Phoenix.
- 2013 “Neuroactive Steroids for Status Epilepticus,” The 4th London-Innsbruck Colloquium on Status Epilepticus and Acute Seizures, Radisson Blu Hotel & Conference Centre, Salzburg, Austria.
- 2013 “Research Update,” New York University Comprehensive Epilepsy Center, NYU Langone Medical Center, New York.
- 2013 “Neurosteroids as Drug Targets in Epilepsy,” Organization for the Study of Sex Differences, Annual Meeting, Sheraton Lincoln Harbor Hotel, Weekawken, New Jersey.
- 2013 “Preclinical Considerations on a Unified Indication,” Antiepileptic Drug and Device Trials XII (University of Pennsylvania School of Medicine), Turnberry Isle Miami Hotel, Aventura, Florida.
- 2013 “The Role of AMPA Receptors in Excitatory Neurotransmission and Epileptic Activity,” 30th International Epilepsy Congress, Montreal
- 2013 “Update on Epilepsy Treatment,” Epilepsy Grand Rounds, George Washington University, Washington, D.C.
- 2013 “Neurosteroids and Epilepsy,” Longwood Medical Area Grand Rounds, Brigham and Women’s Hospital, Harvard Institute of Medicine, Boston.
- 2013 “The Newly Approved Antiepileptic Drug Perampanel – How It Was Developed, How It Works and How to Use It,” Neurology Grand Rounds, Massachusetts General Hospital, Boston.
- 2013 “New and old AEDs with different modes of action – How do their different targets impact clinical outcomes?” Training Day, Finnish Epilepsy Society, Scandic Marski, Helsinki.
- 2013 “Advancing Translation in Epilepsy: Countering the Positive Results Bias,” Epilepsy Special Interest Group, American Neurological Association 2013 Annual Meeting, New Orleans.
- 2013 “Neuroactive Steroids Ganaxolone and Allopregnanolone in the Treatment of Epilepsy, Traumatic Brain Injury, and Neurobehavioral Disorders,” 52nd American College of Neuropsychopharmacology Annual Meeting, The Westin Diplomat, Hollywood, Florida.
- 2014 “Human Studies with Therapeutic Neurosteroids: An Update,” Duke Neurosteroid Congress, Washington Duke Inn & Golf Club, Durham, North Carolina.
- 2014 “Inhaled Treatments to Stop Seizures Before they Spread,” Epilepsy Pipeline Update, Epilepsy Therapy Project, Hyatt Regency, San Francisco.
- 2014 “Should the Current Approach to Therapy Discovery be Modified,” Symposium on Conquering Pharmacoresistant Epilepsy: Debates, Twelfth Eilat Conference on New Antiepileptic Drugs, Melia Castilla, Madrid, Spain.
- 2014 “Propofol Hemisuccinate,” Twelfth Eilat Conference on New Antiepileptic Drugs, Melia Castilla, Madrid, Spain.

- 2015 “Neurosteroids in Epilepsy – From Menstrual Seizures to Super-Refractory Status Epilepticus,” Keynote, EpLINK Science Innovation Team, Ontario Brain Institute, Toronto.
- 2015 “Treatment of Super-refractory Status Epilepticus,” Harvard Medical School Neurology Program at Beth Israel Deaconess Medical Center and Boston Children’s Hospital, Harvard Institute of Medicine, Boston.
- 2015 “Propofol Hemisuccinate,” The 5th London-Innsbruck Colloquium on Status Epilepticus and Acute Seizures,” Imperial College, London.
- 2015 “Debate: Preclinical Randomized Controlled Trials: Can They Increase the Likelihood of Success in Human Trials?,” Antiepileptic Drug and Device Trials XIII, Turnberry Isle Miami Hotel, Aventura (North Miami Beach), Florida.
- 2015 Propofol Hemisuccinate,” Antiepileptic Drug and Device Trials XIII, Turnberry Isle Miami Hotel, Aventura (North Miami Beach), Florida.
- 2015 “Pathophysiology of Seizures and New Treatment Strategies,” Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, Oklahoma.
- 2015 “Antiepileptic Drugs: Needs for New Treatments and Targets,” Therapy Development in the Era of Team Science and Big Data: What will the Future Bring to the Patient with Epilepsy, College of Pharmacy, University of Utah, Park City, Utah.
- 2015 “AMPA Receptors: Synaptic Physiology, Role in the Circuitry of Epilepsy, and Clinical Translation,” 2015 Ion Channel Symposium, Zhejiang University, Hangzhou, P.R. China.
- 2015 “Propofol Hemisuccinate for the Acute Treatment of Migraine,” 9th Annual Pain & Migraine Therapeutics Summit, Washington, D.C.
- 2015 “Publication Bias: When Data is AWOL,” Translational Symposium, 69th Annual Meeting, American Epilepsy Society, Philadelphia.
- 2016 “Propofol Hemisuccinate,” Epilepsy Foundation Pipeline Conference, San Francisco
- 2016 “Propofol Hemisuccinate: An Inhaled Treatment for Epilepsy and Migraine,” 2016 Annual Meeting, American Society for Experimental Neurotherapeutics, Bethesda, Maryland
- 2016 “AMPA Receptors, A New Molecular Target for Epilepsy Therapy,” Clinical Neuroscience Grand Rounds, Boston University School of Medicine, Boston
- 2016 “Allopregnanolone in the Treatment of Status Epilepticus,” Second Neurosteroid Congress, Duke University, Washington Duke Inn & Golf Club, Durham, North Carolina
- 2016 “Novel Drugs and Future Hopes: Human Perspective,” 2nd International Conference on One Medicine One Science, Team Science Case Study: Canine and Human Epilepsy, a Model for Bidirectional Benefit, The Commons Hotel, University of Minnesota, Minneapolis
- 2016 “Management of Tonic-clonic Seizures: Does Mechanism Matter?” Tonic-clonic Seizures: Realities, Controversies, Therapies, American Academy of Neurology Annual Meeting, Stanley Park Ballroom, Westin Bayshore Hotel, Vancouver, Canada.
- 2016 2016 ACVIM Forum / Scientific Sessions, American College of Veterinary Internal Medicine, Colorado Convention Center.
- 2016 CURE Day of Science, California Academy of Science, San Francisco.
- 2016 Basic Neuroscience of EEG, CNSF Congress Neurology Resident Course – Neurophysiology/Electroencephalography, Quebec City Convention Centre, Quebec City, Quebec.
- 2016 Debate 2: “Is Knowledge of Mechanism of Action Essential for the Development of New AEDs?” and “Propofol Hemisuccinate: An Inhaled Treatment for Epilepsy,”

- Thirteenth Eilat Conference On New Antiepileptic Drugs and Devices (EILAT XIII), Madrid, Spain.
- 2016 “Neurosteroids in the Treatment of Epilepsy and Status Epilepticus,” Excitatory-Inhibitory Signaling Balance as Therapeutic Target in Epilepsy, Montreal Neurological Institute, Montreal.
- 2016 “New Antiepileptic Drugs with Potential for Therapy-resistant Epilepsy,” Chair Symposium and “Allopregnanolone Potential in the Treatment of Super-refractory Status Epilepticus,” Novel Therapies for Status Epilepticus (parallel session), 12th European Congress on Epileptology, Kongresové centrum Praha, Prague.
- 2016 “Refractory Status Epilepticus: Mechanisms and Management,” Faculty Speaker Interactive Lunch Workshop, 141st Annual Meeting of the American Neurological Association, Baltimore, Maryland.
- 2016 “AMPA Receptors in Epilepsy Therapy,” 26th Elsevier Neuropharmacology Conference, Ionotropic Glutamate Receptors, Paradise Point, San Diego.
- 2016 “The newer medications have not helped our refractory patients.” Annual Course, Annual Meeting, American Epilepsy Society, Houston, Texas.
- 2017 “New Therapies for Super-Refractory Status Epilepticus (SRSE)” and “New Antiepileptic Drugs and Potential for Therapy Resistant Epilepsy,” Israeli League Against Epilepsy Conference, Leonardo City Tower, Ramat Gan, Israel.
- 2017 “Drug Development for Seizures and Status Epilepticus at UC Davis,” Inaugural UC-Wide Drug Discovery Symposium, California Nanosystems Institute, University of California, Los Angeles.
- 2017 “Propofol hemisuccinate. An Inhaled and Oral Treatment for Acute Seizures,” The 6th London-Innsbruck Colloquium on Status Epilepticus and Acute Seizures, Salzburg, Austria.
- 2017 “Neuroendocrine Effects on Headache,” Neuroendocrine Update: Nuts and Bolts of What You need to Know, 69th Annual Meeting, American Academy of Neurology, Boston.
- 2017 “Neurobiology of Antiepileptic Drugs,” 9th National Epilepsy Congress, Philippine League Against Epilepsy, Marco Polo Hotel, Cebu City, Philippines.
- 2017 “Novel Treatments for Refractory Status Epilepticus” (Session on Super-refractory Status Epilepticus) and “Glutamate Receptors in Epilepsy” (Plenary session 5: Targeted Therapy in Epilepsy – The Role of Glutamate), ILAE British Chapter Annual Scientific Meeting, Queens Hotel, Leeds, United Kingdom.
- 2017 “Molecular Targets for Antiseizure Drugs,” Genentech Inc., South San Francisco.
- 2018 “Glutamate receptor antagonists in epilepsy therapy: Does mode of action matter?” 11th Valentin Symposium, Berlin.
- 2018 “Propofol Prodrug: Oral Treatment for Acute Repetitive Seizures,” Epilepsy Foundation Pipeline Conference, San Francisco.
- 2018 “Neurosteroids and the Treatment of Status Epilepticus,” Center for Orphan Drug Research, Institute for Therapeutics Discovery & Development, University of Minnesota, Minneapolis.
- 2018 “Propofol Prodrug: Oral Treatment for Acute Repetitive Seizures,” 2018 Annual Meeting American Society for Experimental Neurotherapeutics.
- 2018 “Ganaxolone in the Treatment of CDKL5 Deficiency Syndrome,” 2018 Annual Meeting American Society for Experimental Neurotherapeutics.
- 2018 “Update on Parenteral Brexanolone,” Third Neurosteroid Congress, Duke University, Washington Duke Inn & Golf Club, Durham, North Carolina.

- 2018 “Propofol Prodrug,” Fourteenth Eilat Conference on New Antiepileptic Drugs and Devices (EILAT XIV), Madrid, Spain.
- 2018 “Anti-ictal Versus Mechanism Targeted Therapies,” Epilepsy Foundation 2018 Research Roundtable for Epilepsy: Evolving Concepts in Endpoints and Populations in Epilepsy Trials, Washington, DC.
- 2018 “Targeting Novel Receptors,” Targeted Therapy, 13th European Congress on Epilepsy, Vienna, Austria.
- 2018 “Pharmacological Thoughts for SLC6A1,” SLC6A1 Connect Roundtable: Developing a Unified Scientific Approach to Treatment Discovery in SLC6A1, American Epilepsy Society Meeting, New Orleans.
- 2019 “New Drugs for Seizures and Epilepsy,” Basic Science Seminar Series, Department of Pathology & Microbiology, University of Nebraska Medical Center, Omaha.
- 2019 “Molecular Therapeutics of Epilepsy,” Fifth International Workshops, Jasper’s Basic Mechanisms of the Epilepsies, Yosemite Lodge, Yosemite National Park, California.
- 2019 “Neuroendocrine Effects on Headache,” Neuroendocrine Update: Nuts and Bolts of What You need to Know, 71st Annual Meeting, American Academy of Neurology, Philadelphia.
- 2019 “Do Neurosteroids Have Any Future in the Treatment of Status Epilepticus?,” and “Novel Acute Seizure Treatments,” 7th London-Innsbruck Colloquium on Status Epilepticus and Acute Seizures, Francis Crick Institute, London.
- 2019 “DBF (diazepam buccal film) for Acute Repetitive Seizures,” Antiepileptic Drug and Device Trials XV, JW Marriott Turnberry Isle Miami Hotel, Aventura, FL.
- 2019 “Neuroactive Steroids for the Treatment of Neurological and Psychiatric Disease,” The Theodore Koppanyi Lectureship in Pharmacology, Department of Pharmacology & Physiology, Georgetown University.
- 2019 “Update: Pharmacological Thoughts for SLC6A1,” SLC6A1 Connect Roundtable, American Epilepsy Society Meeting, Baltimore.
- 2020 “Creation of the First Neurosteroid Drug,” Israeli League Against Epilepsy Conference, Dan Hotel, Tel Aviv.
- 2020 “Diazepam Buccal Film for Acute Repetitive Seizures,” Fifteenth Eilat Conference on New Antiepileptic Drugs and Devices (EILAT XV).
- 2020 “Screening Across the Board: Where are We and Where Should We be Going?” Epilepsy Foundation Pipeline Conference (virtual).
- 2020 “Diazepam Buccal Film,” Epilepsy Foundation Pipeline Conference (virtual).
- 2020 “AMPA Receptors in Epilepsy and as Targets for Antiepileptic Drugs,” China Association Against Epilepsy (virtual).
- 2021 “Neurosteroids in the Treatment of Status Epilepticus,” Claude G. Wasterlain, MD Festschrift in Recognition of 45 Years of Contributions to Veterans & Science, West Los Angeles Veterans Administration.
- 2022 “Neurosteroids in the Treatment of Epilepsy and Status Epilepticus,” Neurology Grand Rounds, Stanford University.
- 2022 “Neurosteroids in the Treatment of Epilepsy and Status Epilepticus,” Neurology Grand Rounds, St. Louis University.
- 2022 “Update on Neuroactive Steroids in the Treatment of Status Epilepticus,” The 8th London-Innsbruck Colloquium on Status Epilepticus and Acute Seizures, Salzburg, Austria.
- 2022 “Role of AMPA Receptors in Epilepsy and as a Target for Antiseizure Medications,” CURE Epilepsy Frontiers in Epilepsy Research Seminar Series, Texas A & M University College of Medicine.

- 2022 “Pharmacological Thoughts for SLC6A1,” SLC6A1 Symposium, American Epilepsy Society Annual Meeting.
- 2022 “Refractory Epilepsy: Molecular Mechanisms,” An Update on Pharmacological Treatments for Refractory Epilepsy and its Prevention, American Epilepsy Society Annual Meeting, Nashville, TN.

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II. REFEREED REVIEWS

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VI. ABSTRACTS

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