

#1

COMPLETE

**Collector:** Web Link 1 (Web Link)  
**Started:** Thursday, July 26, 2018 8:03:42 AM  
**Last Modified:** Thursday, July 26, 2018 8:21:09 AM  
**Time Spent:** 00:17:27  
**IP Address:** 69.74.180.17

---

Page 1: ASNM BOARD ELECTION CANDIDATE FORM

**Q1 Name (as you would like it to appear on the ballot)**

Marat Avshalumov

---

**Q2 Credential(s)**

PhD, DABNM, CNIM

---

**Q3 Current Position and Organization**

Chief Neurophysiologist

---

**Q4 Education**

Education

1995 Rostov State University, Russia Ph.D. in Biochemistry/ Neurochemistry

1991 Dagestan State University, Russia Master of Science, Department of Biochemistry with Highest Honors

Related coursework:

2006 Clinical trials: structure and management, NYU School of Medicine, New York, New York

2005 From Idea to IPO: the technology venture (Case Studies), New York Academy of Sciences, New York, New York

---

**Q5 Professional Affiliations**

Scientific societies memberships

2012 – present International Society for Intraoperative Monitoring

2008 – present American Society of Neurophysiologic Monitoring

1999 – present American Society for Neuroscience

---

**Q6 Publications, Awards and Appointments**

Professional Experience

Chief Neurophysiologist

09/2009 – Present

Director of Operations, Neurophysiology Monitoring Unit,  
Neurological Surgery, P.C., Rockville Centre, NY

Assistant Professor and Assistant Director Intraoperative Unit Department of Neurosurgery

## ASNM 2018 Candidate Form

Assistant Professor and Assistant Director Intraoperative Unit Department of Neurosurgery  
Mount Sinai School of Medicine, New York, New York 09/2007 - 09/2009

Research Assistant Professor Department of Neurosurgery  
New York University School of Medicine, New York, New York 09/2005 – 03/2007

Postdoctoral Fellow/Research Scientist,  
New York University School of Medicine, New York, New York 09/1998 – 09/2005

### Adjunct Appointments

Assistant Professor, Laboure College, Milton, Massachusetts 2014 - Present  
Assistant Professor, New York University School of Medicine  
New York, New York 2007 - Present

### Awards

2009 M. Stecker Award, ASNM Vancouver, Canada

### Bibliography

#### Peer-reviewed Articles

1. Avshalumov, M. V., Chen, B. T., Rice, M. E. Mechanisms underlying H<sub>2</sub>O<sub>2</sub>-mediated inhibition of synaptic transmission in rat hippocampal slices. *Brain Res.* 2000; 882:86-94.
2. Chen, B. T., Avshalumov, M. V., Rice, M. E. H<sub>2</sub>O<sub>2</sub> is a novel, endogenous modulator of synaptic dopamine release. *J. Neurophysiol.* 2001; 85:2468-2476.
3. Chen, B. T., Avshalumov, M. V., Rice, M. E. Modulation of somatodendritic dopamine release by endogenous H<sub>2</sub>O<sub>2</sub>: susceptibility in substantia nigra but resistance in VTA. *J. Neurophysiol.* 2002; 87:1155-1158.
4. Avshalumov, M. V., Rice, M. E. NMDA-receptor activation mediates hydrogen peroxide-induced pathophysiology in rat hippocampal slices. *J. Neurophysiol.* 2002; 87:2896-2903.
5. Rice, M. E., Forman, R. E., Chen, B. T., Avshalumov, M. V., Cragg, S. J., Drew, K. L. Brain antioxidant regulation in mammals and anoxia-tolerant reptiles: balanced for neuroprotection and neuromodulation. *Comp. Biochem. Physiol. (Part C)* 2002; 133:515-525.
6. Avshalumov, M. V., Chen, B. T., Marshall, S. P., Peña, D. M., Rice, M. E. Glutamate-dependent inhibition of dopamine release in striatum is mediated by a new diffusible messenger, H<sub>2</sub>O<sub>2</sub>. *J. Neurosci.* 2003; 23:2744-2750.
7. MacGregor, D. G., Avshalumov, M. V., Rice, M. E. Brain edema induced by in vitro ischemia: causal factors and neuroprotection. *J. Neurochem.* 2003; 85: 1402-1411.
8. Avshalumov, M. V., Rice, M. E. Activation of ATP-sensitive K<sup>+</sup> (K<sub>ATP</sub>) channels by H<sub>2</sub>O<sub>2</sub> underlies glutamate-dependent inhibition of striatal dopamine release. *Proc. Natl. Acad. Sci. (U.S.A.)* 2003; 100:11729-11734.
9. Avshalumov, M. V., MacGregor, D. G., Sehgal, L. M., Rice, M. E. The glial antioxidant network and neuronal ascorbate: protective yet permissive for H<sub>2</sub>O<sub>2</sub> signaling. *Neuron Glia Biol.* 2004; 1: 365-376.
10. Avshalumov, M. V., Chen, B. T., Koós, T., Tepper, J. M., Rice, M. E. Endogenous hydrogen peroxide regulates the excitability of midbrain dopamine neurons via ATP-sensitive potassium channels. *J. Neurosci.* 2005; 25:4222-4231.
11. Bao, L., Avshalumov, M. V., Rice, M. E. Partial mitochondrial inhibition causes suppression of striatal dopamine release and depolarization of medium spiny neuron via H<sub>2</sub>O<sub>2</sub> elevation in the absence of ATP depletion. *J. Neurosci.* 2005; 25: 10029-10040.
12. Chen, B. T., Moran, K. A., Avshalumov, M. V., Rice, M. E. Limited regulation of somatodendritic dopamine release by voltage-sensitive Ca<sup>2+</sup> channels contrasted with strong regulation of axonal dopamine release. *J. Neurochem.* 2006; 96: 645-655.
13. Fedirko, N., Avshalumov, M. V., Rice, M. E., Chesler, M. Regulation of postsynaptic Ca<sup>2+</sup> influx in hippocampal CA1 pyramidal neurons via extracellular carbonic anhydrase. *J. Neurosci.* 2007; 27: 1167-1175.
14. \*Avshalumov, M. V., \*Patel, J. C., Rice, M. E. AMPA receptor-dependent H<sub>2</sub>O<sub>2</sub> generation in striatal medium spiny neurons, but not dopamine axons: one source of a retrograde signal that can inhibit dopamine release. *J. Neurophysiol.* 2008; 100: 1590-1601 (\*co-

## ASNM 2018 Candidate Form

first authors).

15. Patel, J. C., Witkovsky, P., Avshalumov, M. V., Rice, M. E. Mobilization of calcium from intracellular stores facilitates somatodendritic dopamine release. *J. Neurosci.* 2009; 29: 6568-6579.
16. Bao, L., Avshalumov, M. V., Patel, J. C., Lee, C. R., Miller, E. W., Chang, C. J., Rice, M. E. Mitochondria are the source of hydrogen peroxide for dynamic brain-cell signaling. *J. Neurosci.* 2009; 29: 9002-9010.
17. Li, X., Patel, J. C., Wang, J., Avshalumov, M. V., Nicholson, C., Buxbaum, J. D., Elder, G. A., Rice, M. E., Yue, Z. Enhanced motor performance and striatal dopamine transmission caused by LRRK2 overexpression in mice is eliminated by familial Parkinson's Disease mutation G2019S. *J. Neurosci.* 2010; 30: 1788-1797.
18. Pan, Y., Chau, L., Liu, S., Avshalumov, M. V., Rice, M. E., Carr, K. D. A food restriction protocol that increases drug reward decreases TrkB in the ventral tegmental area, with no effect on BDNF or TrkB protein levels in dopaminergic forebrain regions. *Neuroscience* 2011; 197: 330-338.
19. Rice, Margaret E.; Avshalumov, Marat V.; Patel, Jyoti C.; Patel, Jyoti C; Rossignol, Elsa; Rice, Margaret E; Machold, Robert P. 'Opposing regulation of dopaminergic activity and exploratory motor behavior by forebrain and brainstem cholinergic circuits'. *Nature communications.* 2012 (3):1172-1172 (# 932562).
20. Rice, Margaret E.; Avshalumov, Marat V.; Patel, Jyoti C.; Khan, Raymond; Kirschenbaum, Linda A; Larow, Catherine; Astiz, Mark E. 'The effect of resuscitation fluids on neutrophil-endothelial cell interactions in septic shock'. *Shock.* 2011 36(5):440-444 (# 946962).
21. Panov F, Tagliati M, Ozelius LJ, Fuchs T, Gologorsky Y, Cheung T, Avshalumov M, Bressman SB, Saunders-Pullman R, Weisz D, Alterman RL. Pallidal deep brain stimulation for DYT6 dystonia. *J Neurol Neurosurg Psychiatry.* 2012 Feb;83(2):182-7. Epub 2011 Sep 23.

### Reviews and Book Chapters

1. Rice, M. E., Chen, B. T., Avshalumov, M. V. Modulation of dopamine release by endogenous H<sub>2</sub>O<sub>2</sub>: implications for Parkinson's disease. In: *Monitoring Molecules in Neuroscience 2003*, Kehr, J., Fuxe, K., Ungerstedt, U., Svensson, T.H., eds. Stockholm: Karolinska University Press, 2003: 206-208.
2. Cragg, S. J., Rice, M. E. Somatodendritic dopamine release in midbrain. In: *Dendritic Neurotransmitter Release*, Ludwig, M., ed. Springer, New York, 2005: 69-83.
3. Rice, M. E., Avshalumov, M. V. Somatodendritic H<sub>2</sub>O<sub>2</sub> from medium spiny neurons inhibits axonal dopamine release. In: *Dendritic Neurotransmitter Release*, Ludwig, M., ed. New York: Springer, 2005: 301-313.
4. Rice, M. E., Patel, J., Bao, L., Pearson, Z. S., Shashidharan, P., Walker, R. H., Chen, B. T., and Avshalumov, M. V. Regulation of dopamine release and dopamine cell activity by endogenous H<sub>2</sub>O<sub>2</sub>: implications for basal ganglia function. In: *The Basal Ganglia VIII*, Bolam, J. P., Ingram, C., and McGill, P., eds. New York: Springer, 2005; 177-186.
5. Rice, M. E., Avshalumov, M. V., Patel, J. C. Hydrogen peroxide as a diffusible messenger: evidence from voltammetric studies of dopamine release in brain slices. In: *Electrochemical Methods in Neuroscience*, Michael, A. C. and Borland, L. M. eds. Boca Raton, Florida: CRC Press, 2007; 205-232.
6. Avshalumov, M.V., Bao, L., Patel, J. C., and Rice, M. E. H<sub>2</sub>O<sub>2</sub> signaling in the nigrostriatal dopamine pathway via ATP-sensitive potassium channels: issues and answers. *Antioxid. Redox. Signal.* 2007; 9: 219-231.
7. Avshalumov, M. V., Patel, J. C., Bao, L., MacGregor, D. G., Sidló, Z., Rice, M. E. Diffusible hydrogen peroxide generated by synaptic activity inhibits axonal dopamine release in striatum. In: *Beyond the Synapse: Cell-Cell Signaling in Synaptic Plasticity*, Fields, R. D. ed. London: Cambridge University Press, 2008: 181-192.
8. Patel, J. C., Witkovsky, P., Avshalumov, M. V., Lee, C. R., Rice, M. E. New insights into the physiological regulation of somatodendritic dopamine release. In: *Monitoring Molecules in Neuroscience 2010*, Westerink, B., Clinckers, R., Smolders, I., Sarre, S., Michotte, Y. eds. Brussels, Vrije Universiteit Brussel, 2010: 130-132.
9. Rice, M. E., Lee, C. R., Bao, L., Avshalumov, M. V., Stouffer, M. A., Carr, K. D., Witkovsky, P., Patel, J. C. Regulation of striatal dopamine release by metabolic signals. In: *Monitoring Molecules in Neuroscience 2010*, Westerink, B., Clinckers, R., Smolders, I., Sarre, S., Michotte, Y. eds. Brussels, Vrije Universiteit Brussel, 2010: 137-139.

Abstracts (National and International Meetings)

## ASNM 2018 Candidate Form

1. Avshalumov, M., Chen, B. T., Kume-Kick, J., Rice, M. E. Inhibition of evoked fields in rat hippocampal slices by H<sub>2</sub>O<sub>2</sub> is mediated by hydroxyl radical, but not by altered presynaptic Ca<sup>2+</sup> entry. Soc. Neurosci. Abstr. 1999; 25:1500.
2. Chen, B. T., Avshalumov, M. V., Rice, M. E. Calcium modulates the inhibition of striatal dopamine release by H<sub>2</sub>O<sub>2</sub>. Soc. Neurosci. Abstr. 1999; 25: 1501.
3. Avshalumov, M. V., Rice, M. E. NMDAR activation by H<sub>2</sub>O<sub>2</sub> in hippocampal slices. Soc. Neurosci. Abstr. 2000; 26:353.
4. MacGregor, D. G., Avshalumov, M. V., and Rice, M. E. Mechanisms of edema formation in in vitro ischemia. Soc. Neurosci. Abstr. 2001; 27:436.16.
5. Chen, B. T., Avshalumov, M., and Rice, M. E. Modulation of somatodendritic dopamine release by endogenous H<sub>2</sub>O<sub>2</sub>: implications for Parkinson's disease. Soc. Neurosci. Abstr. 2001; 27:654.16.
6. Avshalumov, M. V., MacGregor, D.G., Rice, M. E. Glial antioxidant network prevents H<sub>2</sub>O<sub>2</sub>-induced oxidative damage in brain slices. Soc. Neurosci. Abstr. 2001; 27:715.17.
7. Avshalumov, M. V., Chen, B. T., Marshall, S.P., Pena, D.M., Rice, M.E. (2002) Glutamate regulation of dopamine release in striatum is mediated by H<sub>2</sub>O<sub>2</sub>. Dopamine 2002, Portland, Oregon, Poster 1.4, p 54.
8. Chen, B. T., Avshalumov, M. V., Moran, K.A., Rice, M. E. Calcium channel blockade inhibits synaptic but not somatodendritic dopamine release. 2002 Abstract Viewer/Itinerary Planner, Washington, DC: Society for Neuroscience, 2002; 440.7.
9. Avshalumov, M. V. Chen, B. T. Marshall, S. P. Pena, D. M., Rice, M. E. H<sub>2</sub>O<sub>2</sub>-sensitive potassium channels mediate the regulatory effect of glutamate on dopamine release in striatum. 2002 Abstract Viewer/Itinerary Planner, Washington, DC: Society for Neuroscience, 2002; 550.8.
10. Avshalumov, M. V., MacGregor, D. G., Rice, M. E. Glial antioxidant network prevents H<sub>2</sub>O<sub>2</sub>-induced oxidative damage in guinea pig brain slices. Brit. J. Pharmacol. 2002; 137:137P Suppl. S.
11. MacGregor, D. G., Avshalumov, M. V., Rice, M. E. Mechanisms underlying rat cerebral oedema formation in in vitro ischaemia Brit. J. Pharmacol. 2002; 137: 138P Suppl. S.
12. Avshalumov, M. V., Chen, B. T., Rice, M. E. Presence of different sulfonylurea receptors in the nigrostriatal pathway defines the sensitivity of dopamine release to H<sub>2</sub>O<sub>2</sub>. 2003 Abstract Viewer/Itinerary Planner, Washington, DC: Society for Neuroscience, 2003; 440.5.
13. Patel, J., Avshalumov, M. V., Rice, M. E. Glutamate-dependent inhibition of striatal dopamine release is mediated by H<sub>2</sub>O<sub>2</sub> generated in medium spiny neurons but not in dopamine terminals. 2004 Abstract Viewer/Itinerary Planner, Washington, DC: Society for Neuroscience, 2004; 46.6.
14. Avshalumov, M. V., Rice, M. E. Physiological consequences of H<sub>2</sub>O<sub>2</sub> generation in midbrain dopamine neurons. 2004 Abstract Viewer/Itinerary Planner, Washington, DC: Society for Neuroscience, 2004; 753.22.
15. Patel, J., Avshalumov, M. V., Rice, M. E. Glutamate-dependent generation of H<sub>2</sub>O<sub>2</sub> occurs in medium spiny neurons but not in dopamine terminals. International Basal Ganglia Society 8th Triennial Meeting: Crieff, Scotland Abstract Book; 2004; P70.
16. Avshalumov, M. V., Rice, M. E. H<sub>2</sub>O<sub>2</sub> generation in midbrain dopamine neurons: physiological consequences. International Basal Ganglia Society 8th Triennial Meeting: Crieff, Scotland Abstract Book; 2004; P80.
17. Avshalumov, M. V., Rice, M. E. H<sub>2</sub>O<sub>2</sub> is an endogenous modulator of midbrain dopamine neuron activity via KATP channels. J. Neurochem. 2005; 94 (Suppl. 2): 116.
18. Bao, L., Avshalumov, M. V., Rice, M. E. Mitochondrial inhibition causes functional dopamine denervation via increased H<sub>2</sub>O<sub>2</sub>, not decreased ATP. 2005 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2005; 426.8.
19. Bao, L., Avshalumov, M. V., Rice, M. E. Modulatory H<sub>2</sub>O<sub>2</sub> that mediates glutamate-dependent modulation of striatal dopamine release is from mitochondria. Dopamine 50 years Symposium, Göteborg, Sweden, May 2007.
20. Patel, J. C., Sideris, A., Avshalumov, M. V., Rice, M. E. Somatodendritic but not axonal dopamine release is enhanced by caffeine: a mechanism involving mobilization of intracellular calcium stores? Dopamine 50 years Symposium, Göteborg, Sweden, May 2007.
21. Patel, J. C., Witkovsky, P., Avshalumov, M. V., Rice, M. E. Regulation of somatodendritic dopamine release by IP<sub>3</sub> receptor and ryanodine receptor dependent intracellular calcium stores. 2008 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2008: 34.10.

**Q7 PROFILE QUESTIONS:**How do you feel you can contribute to the leadership of ASNM? What strengths/passions/talents do you hold that would benefit ASNM?

The last 9 years of my life have been very exciting: I made a transition from academia from the Mount Sinai school of medicine to Chief Neurophysiologist and Director of operations for an IOM practice (which I spearheaded from the ground up) in a private neurosurgical group. I also run an IOM training online and on-site program for international (Australia, Bulgaria and Russia) IOM professionals. These domestic and international experiences required me to develop the ability to negotiate differing points of view and build effective teams from different parties with diverging ideas - that requires both flexibility and the ability to stand your own ground. In a changing environment around IOM as a part of our healthcare system, I truly believe that these strengths of mine will help ASNM to overcome challenges that society is currently facing. My passion is to see ASNM as "all inclusive", catering to both small and big players in the IOM field, with an emphasis on the role of the neurophysiologist as a critically important member of the ASNM.

---

**Q8** With changes in health care service delivery and reimbursement, how do you feel you can contribute to keep ASNM moving forward in the right direction?

- It is important that the ASNM remain a leader in education in the field of IONM at all levels, including neurophysiologists and physicians. The society should also reach out to educate insurance providers and governmental regulations to improve patient safety and lower healthcare costs
- The only way to keep moving forward is as a united group of health care professionals. This must include IOM professionals in academic centers, small IOM groups, and large IOM companies. As a PHD degree holder, I feel that I can contribute towards the further consolidation between physician and non-physician IOM professionals.
- ASNM is a great society, however, I am sure that ASNM will benefit from more intense interaction with the relevant societies in the USA and internationally. This should include not only IOM groups, but also surgical societies.
- I believe that it is time for the ASNM to focus on the specific education of the different patients and patient interests groups

Together these will result not only in an increase of the ASNM influence and recognition in the field of intraoperative monitoring, but it will also positively affect the growth of the ASNM

---

**Q9** ASNM constantly seeks ideas of how to better serve our membership through education, resources, representation to other professional entities, connections and networking or other means of advancement. What do you think ASNM could offer its members that would provide value?

To better serve the membership:

- ASNM needs to advocate for collection of IONM data that can help us understand its clinical value for different surgical procedures. It is important to realize that this data must result in peer-reviewed publications. This will result in making the case for medical necessity to all parties: surgeons, patients, government legislative bodies, and insurance companies.
  - Based on the "functional and active" clinical outcome database, ASNM should forge consensus and uniformity on monitoring modalities and methods. This will result in improved patient care, helping with the reimbursement process, as well as building a strong defense against litigation. IOM field needs a standardization of operating procedures.
  - Continuously improve the educational programs that are run by ASNM. My belief is that educational programs, at least some, should be based on unedited, raw data clinical cases, presented by technologists. Bringing up "not so ideal" cases to learn from mistakes and errors.
-

## ASNМ 2018 Candidate Form

**Q10** Personal Statement: Please provide any additional information to the members.

It is an Honor to be nominated to run for the ASNМ board. At the same time, it is a great responsibility. However, if one would like to participate in the joint effort to move the IOM field and the ASNМ forward to success and recognition, one has to get actively involved in such an effort. So, I decided to run for it.

---