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| **Affiliation:****Address:****Contact:** |  |

**Narrative**

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| *I am a Neuroscientist and a Professor in the Department of Biochemistry at UCC as well as an active and productive researcher. My research focuses on characterization of potassium channels in glial cells with an emphasis on the functional role of these channels in homeostatic mechanisms. One of my most significant research contributions in this area is related to elucidating the role of glial Kir4.1 potassium channels in potassium and glutamate buffering. I have published more than 50 articles in peer-reviewed journals and have had NIH grant support for my research since 1996. In addition, I have had a long-standing commitment to facilitating the research endeavour at UCC. I am currently one of the PIs of the RCMI program. Prior to taking over this position, I served for 14 years as coordinator of the RCMI funded Common Instrumentation Area and Services Core facility at UCC. This is a facility that houses common use instrumentation and provides services to investigators through an electronics technician and a machinist/handyman. In addition, I was the Director of the RCMI-funded Neuroscience Research Center and am the Associate Scientific Director of the Integrative Center for Glial Research. I am a past president of the Puerto Rico Chapter of the Society for Neuroscience and have organized Conferences and Symposia including the Annual Puerto Rico Neuroscience Conference and the Annual CaribeGlia Minisymposium. These activities were facilitated by my strong collaborations with researchers from the mainland USA and Europe.* *Contributions to Science**1) One of my most significant research contributions to date is elucidating the role of glial Kir4.1 potassium channels in astrocytic potassium and glutamate buffering. I have extensive background in functional characterization of potassium channels in glial cells with an emphasis on the role of these channels in homeostatic mechanisms, but these studies were initially hampered by the lack of specific potassium channel blockers. To overcome this problem, we were one of the first groups to use siRNA technology to selectively down-regulate proteins (in this case potassium channels) in astrocytes.* *2) Diabetics are at risk for a number of serious health complications including an increased incidence of epilepsy and poorer recovery after ischemic stroke. Astrocytes play a critical role in protecting neurons by maintaining extracellular homeostasis and preventing neurotoxicity through glutamate uptake and potassium buffering. These functions are aided by the presence of potassium channels, such as Kir4.1 inwardly rectifying potassium channels, in the membranes of astrocytic glial cells. Using a variety of techniques including used q-PCR, Western blot, patch-clamp electrophysiology studying voltage and potassium step responses and a colorimetric glutamate clearance assay, we assessed Kir4.1 channel levels and homeostatic functions of rat astrocytes grown in normal and high glucose conditions. We found that hyperglycemia decreases Kir4.1 potassium channel expression and impairs homeostatic functions of astrocytes. Our results suggest that down-regulation of astrocytic Kir4.1 channels by elevated glucose may contribute to the underlying pathophysiology of diabetes-induced CNS disorders and contribute to the poor prognosis after stroke.**3) Inwardly rectifying potassium channel Kir4.1 is critical for glial function, control of neuronal excitability, and systemic K+ homeostasis. Genetic inactivation of these channels in glia impairs extracellular K+ and glutamate clearance and produces a seizure phenotype. In both mice and humans, polymorphisms and mutations in the KCNJ10 gene have been associated with seizure susceptibility. In mice, we demonstrated that there are differences in Kir channel activity and potassium- and glutamate-buffering capabilities between astrocytes from seizure resistant C57BL/6 (B6) and seizure susceptible DBA/2 (D2) mice that are consistent with an altered K+ channel activity as a result of genetic polymorphism of KCNJ10 (gene encoding Kir4.1). In addition, we investigated the functional significance of novel human mutations in Kir4.1 which have been associated with EAST/SeSAME syndrome, characterized by mental retardation, ataxia, seizures, hearing loss, and renal salt waste. All of the mutations compromised channel function, but the underlying mechanisms were different. I served as primary investigator or co-investigator on all of these studies.**4) Kir4.1 potassium channels are expressed in glial cells in the brain and retina and attempts had been made to equate properties of exogenously expressed Kir4.1 currents with those of native K+ currents in glial cells. There were nagging problems however with assigning native currents to Kir4.x channels. One major concern was that in many native tissues, the putatively correlated currents show much weaker rectification than reported for cloned Kir4.1 channels. We found that the rectification in physiological concentrations of potassium was similar to that reported in native tissue. We also found two types of block that help promote potassium uptake by the cells. When [K+]o is rapidly increased, as would occur during neuronal excitation, ‘‘fast block’’ would be relieved, promoting potassium influx to glial cells. Increase in [K+]in would then lead to relief of ‘‘slow block,’’ further promoting K+-influx.* *5) Excitotoxicity due to glutamate receptor over-activation is one of the key mediators of neuronal death after an ischemic insult. Therefore, a major function of astrocytes is to maintain low extracellular levels of glutamate. The ability of astrocytic glutamate transporters to regulate the extracellular glutamate concentration depends upon the hyperpolarized membrane potential of astrocytes conferred by the presence of K+ channels in their membranes. We have shown that TREK-2 potassium channels in cultured astrocytes are up-regulated by ischemia and may support glutamate clearance by astrocytes during ischemia. Recently, we determined the mechanism leading to this up-regulation and assessed the localization of TREK-2 channels in astrocytes after transient middle cerebral artery occlusion. We first demonstrated that TREK-2 channels were up-regulated after ischemia via a mechanism which required De novo protein synthesis, but did not require increases in TREK-2 mRNA. Immunohistochemical studies revealed TREK-2 localization in astrocytes together with increased expression of the selective glial marker, glial fibrillary acidic protein, in brain 24 hours after transient middle cerebral occlusion. Our data indicate that functional TREK-2 channels are up-regulated in the astrocytic membrane during ischemia through a mechanism requiring De novo protein synthesis. These study provides important information about the mechanisms underlying TREK-2 regulation, which has profound implications in neurological diseases such as ischemia where astrocytes play an important role.* |

**Awards and Honors**

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| **1982** |  |  *- Phi Beta Kappa* |
| **1985** |  |  *- Beal foundation Scholarship* |
| **1986** | **1990** |  *- Neuroendocrine Training Grant - Predoctoral Fellowship* |
| **1990** | **1993** |  *- Neuroscience Training Grant - Postdoctoral Fellowship* |
| **1993** |  |  *- ASPET Young Scientist Travel Award* |

**Reviews/Chapters/Editorials**

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| **1.** | *Moss, R.L., Dudley, C.A., Kim, Y.I. and Eaton, M. . Receptor-Receptor Interactions (Eds. K. Fuxe and L. Agnati). Estrogenic and antiestrogenic modulation of neuronal membrane sensitivity. 1987; 38:105-118.* |  |

**Publications**

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| **1.** | *Méndez-González MP, Rivera-Aponte DE, Benedikt J, Maldonado-Martínez G, Tejeda-Bayron F, Skatchkov SN, Eaton MJ. Downregulation of Astrocytic Kir4.1 Potassium Channels Is Associated with Hippocampal Neuronal Hyperexcitability in Type 2 Diabetic Mice. Brain Sci. 2020 Jan 30; 10(2).* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/32019062) |
| **2.** | *Rivera-Pagán AF, Méndez-González MP, Rivera-Aponte DE, Malpica-Nieves CJ, Melnik-Martínez KV, Zayas-Santiago A, Maldonado-Martínez G, Shuba YM, Skatchkov SN, Eaton MJ. A-Kinase-Anchoring Protein (AKAP150) is expressed in Astrocytes and Upregulated in Response to Ischemia. Neuroscience. 2018 08 01; 384:54-63.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/29800717) |
| **3.** | *Méndez-González MP, Kucheryavykh YV, Zayas-Santiago A, Vélez-Carrasco W, Maldonado-Martínez G, Cubano LA, Nichols CG, Skatchkov SN, Eaton MJ. Novel KCNJ10 Gene Variations Compromise Function of Inwardly Rectifying Potassium Channel 4.1. J Biol Chem. 2016 Apr 01; 291(14):7716-26.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/26867573) |
| **4.** | *Rivera-Aponte DE, Méndez-González MP, Rivera-Pagán AF, Kucheryavykh YV, Kucheryavykh LY, Skatchkov SN, Eaton MJ. Hyperglycemia reduces functional expression of astrocytic Kir4.1 channels and glial glutamate uptake. Neuroscience. 2015 Dec 03; 310:216-23.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/26404875) |
| **5.** | *Rolón-Reyes K, Kucheryavykh YV, Cubano LA, Inyushin M, Skatchkov SN, Eaton MJ, Harrison JK, Kucheryavykh LY. Microglia Activate Migration of Glioma Cells through a Pyk2 Intracellular Pathway. PLoS One. 2015; 10(6):e0131059.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/26098895) |
| **6.** | *Rivera-Pagán AF, Rivera-Aponte DE, Melnik-Martínez KV, Zayas-Santiago A, Kucheryavykh LY, Martins AH, Cubano LA, Skatchkov SN, Eaton MJ. Up-regulation of TREK-2 potassium channels in cultured astrocytes requires de novo protein synthesis: relevance to localization of TREK-2 channels in astrocytes after transient cerebral ischemia. PLoS One. 2015; 10(4):e0125195.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/25886567) |
| **7.** | *Zueva L, Rivera Y, Kucheryavykh L, Skatchkov SN, Eaton MJ, Sanabria P, Inyushin M. Electron microscopy in rat brain slices reveals rapid accumulation of Cisplatin on ribosomes and other cellular components only in glia. Chemother Res Pract. 2014; 2014:174039.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/25610648) |
| **8.** | *Huertas A, Wessinger WD, Kucheryavykh YV, Sanabria P, Eaton MJ, Skatchkov SN, Rojas LV, Maldonado-Martínez G, Inyushin MY. Quinine enhances the behavioral stimulant effect of cocaine in mice. Pharmacol Biochem Behav. 2015 Feb; 129:26-33.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/25482328) |
| **9.** | *Zueva L, Makarov V, Zayas-Santiago A, Golubeva T, Korneeva E, Savvinov A, Eaton M, Skatchkov S, Inyushin M. Müller cell alignment in bird fovea: possible role in vision. J Neurosci Neuroeng. 2014 12 01; 3(2):85-91.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/25866702) |
| **10.** | *Skatchkov SN, Woodbury-Fariña MA, Eaton M. The role of glia in stress: polyamines and brain disorders. Psychiatr Clin North Am. 2014 Dec; 37(4):653-78.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/25455070) |
| **11.** | *Zayas-Santiago A, Agte S, Rivera Y, Benedikt J, Ulbricht E, Karl A, Dávila J, Savvinov A, Kucheryavykh Y, Inyushin M, Cubano LA, Pannicke T, Veh RW, Francke M, Verkhratsky A, Eaton MJ, Reichenbach A, Skatchkov SN. Unidirectional photoreceptor-to-Müller glia coupling and unique K+ channel expression in Caiman retina. PLoS One. 2014; 9(5):e97155.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/24831221) |
| **12.** | *Kucheryavykh LY, Rolón-Reyes K, Kucheryavykh YV, Skatchkov S, Eaton MJ, Sanabria P, Wessinger WD, Inyushin M. Glioblastoma development in mouse brain: general reduction of OCTs and mislocalization of OCT3 transporter and subsequent uptake of ASP+ substrate to the nuclei. J Neurosci Neuroeng. 2014 Feb; 3(1):3-9.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/25165637) |
| **13.** | *Makarov V, Kucheryavykh L, Kucheryavykh Y, Rivera A, Eaton MJ, Skatchkov SN, Inyushin M. Transport Reversal during Heteroexchange: A Kinetic Study. J Biophys. 2013; 2013:683256.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/24307897) |
| **14.** | *Abushik PA, Sibarov DA, Eaton MJ, Skatchkov SN, Antonov SM. Kainate-induced calcium overload of cortical neurons in vitro: Dependence on expression of AMPAR GluA2-subunit and down-regulation by subnanomolar ouabain. Cell Calcium. 2013 Aug; 54(2):95-104.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/23721822) |
| **15.** | *Benedikt J, Inyushin M, Kucheryavykh YV, Rivera Y, Kucheryavykh LY, Nichols CG, Eaton MJ, Skatchkov SN. Intracellular polyamines enhance astrocytic coupling. Neuroreport. 2012 Dec 05; 23(17):1021-5.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/23076119) |
| **16.** | *Kucheryavykh LY, Kucheryavykh YV, Rolón-Reyes K, Skatchkov SN, Eaton MJ, Cubano LA, Inyushin M. Visualization of implanted GL261 glioma cells in living mouse brain slices using fluorescent 4-(4-(dimethylamino)-styryl)-N-methylpyridinium iodide (ASP+). Biotechniques. 2012 Nov; 53(5):305-9.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/23570046) |
| **17.** | *Inyushin MY, Huertas A, Kucheryavykh YV, Kucheryavykh LY, Tsydzik V, Sanabria P, Eaton MJ, Skatchkov SN, Rojas LV, Wessinger WD. L-DOPA Uptake in Astrocytic Endfeet Enwrapping Blood Vessels in Rat Brain. Parkinsons Dis. 2012; 2012:321406.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/22888467) |
| **18.** | *Seidel KN, Derst C, Salzmann M, Höltje M, Priller J, Markgraf R, Heinemann SH, Heilmann H, Skatchkov SN, Eaton MJ, Veh RW, Prüss H. Expression of the voltage- and Ca2+-dependent BK potassium channel subunits BKß1 and BKß4 in rodent astrocytes. Glia. 2011 Jun; 59(6):893-902.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/21438011) |
| **19.** | *Inyushin M, Kucheryavykh LY, Kucheryavykh YV, Nichols CG, Buono RJ, Ferraro TN, Skatchkov SN, Eaton MJ. Potassium channel activity and glutamate uptake are impaired in astrocytes of seizure-susceptible DBA/2 mice. Epilepsia. 2010 Sep; 51(9):1707-13.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/20831751) |
| **20.** | *Sala-Rabanal M, Kucheryavykh LY, Skatchkov SN, Eaton MJ, Nichols CG. Molecular mechanisms of EAST/SeSAME syndrome mutations in Kir4.1 (KCNJ10). J Biol Chem. 2010 Nov 12; 285(46):36040-8.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/20807765) |
| **21.** | *Inyushin M, Kucheryaykh Y, Kucheryavykh L, Sanabria P, Jiménez-Rivera C, Struganova I, Eaton M, Skatchkov S. Membrane potential and pH-dependent accumulation of decynium-22 (1,1''-diethyl-2,2''-cyanine iodide) flourencence through OCT transporters in astrocytes. Bol Asoc Med P R. 2010 Jul-Sep; 102(3):5-12.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/23875515) |
| **22.** | *Kucheryavykh LY, Kucheryavykh YV, Inyushin M, Shuba YM, Sanabria P, Cubano LA, Skatchkov SN, Eaton MJ. Ischemia Increases TREK-2 Channel Expression in Astrocytes: Relevance to Glutamate Clearance. Open Neurosci J. 2009 Jan 01; 3:40-47.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/19890471) |
| **23.** | *Acevedo-Torres K, Berríos L, Rosario N, Dufault V, Skatchkov S, Eaton MJ, Torres-Ramos CA, Ayala-Torres S. Mitochondrial DNA damage is a hallmark of chemically induced and the R6/2 transgenic model of Huntington''s disease. DNA Repair (Amst). 2009 Jan 01; 8(1):126-36.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/18935984) |
| **24.** | *Kucheryavykh YV, Shuba YM, Antonov SM, Inyushin MY, Cubano L, Pearson WL, Kurata H, Reichenbach A, Veh RW, Nichols CG, Eaton MJ, Skatchkov SN. Complex rectification of Müller cell Kir currents. Glia. 2008 May; 56(7):775-90.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/18293411) |
| **25.** | *Kucheryavykh YV, Pearson WL, Kurata HT, Eaton MJ, Skatchkov SN, Nichols CG. Polyamine permeation and rectification of Kir4.1 channels. Channels (Austin). 2007 May-Jun; 1(3):172-8.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/18690029) |
| **26.** | *Pearson WL, Skatchkov SN, Eaton MJ, Nichols CG. C-terminal determinants of Kir4.2 channel expression. J Membr Biol. 2006; 213(3):187-93.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/17468958) |
| **27.** | *Kucheryavykh YV, Kucheryavykh LY, Nichols CG, Maldonado HM, Baksi K, Reichenbach A, Skatchkov SN, Eaton MJ. Downregulation of Kir4.1 inward rectifying potassium channel subunits by RNAi impairs potassium transfer and glutamate uptake by cultured cortical astrocytes. Glia. 2007 Feb; 55(3):274-81.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/17091490) |
| **28.** | *Krivoi II, Drabkina TM, Kravtsova VV, Vasiliev AN, Eaton MJ, Skatchkov SN, Mandel F. On the functional interaction between nicotinic acetylcholine receptor and Na+,K+-ATPase. Pflugers Arch. 2006 Sep; 452(6):756-65.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/16636868) |
| **29.** | *Skatchkov SN, Eaton MJ, Shuba YM, Kucheryavykh YV, Derst C, Veh RW, Wurm A, Iandiev I, Pannicke T, Bringmann A, Reichenbach A. Tandem-pore domain potassium channels are functionally expressed in retinal (Müller) glial cells. Glia. 2006 Feb; 53(3):266-76.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/16265669) |
| **30.** | *Eaton MJ, Ospina CA, Rodríguez AD, Eterovic VA. Differential inhibition of nicotine- and acetylcholine-evoked currents through alpha4beta2 neuronal nicotinic receptors by tobacco cembranoids in Xenopus oocytes. Neurosci Lett. 2004 Aug 05; 366(1):97-102.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/15265598) |
| **31.** | *Eaton MJ, Veh RW, Makarov F, Shuba YM, Reichenbach A, Skatchkov SN. Tandem-pore K(+) channels display an uneven distribution in amphibian retina. Neuroreport. 2004 Feb 09; 15(2):321-4.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/15076761) |
| **32.** | *Krivoi II, Drabkina TM, Dobretsov MG, Vasil''ev AN, Kravtsova VV, Eaton MJ, Skachkov SN, Mandel F. [Functional interaction between nicotinic cholinergic receptors and Na, K-ATPase in the skeletal muscles]. Ross Fiziol Zh Im I M Sechenova. 2004 Jan; 90(1):59-72.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/15143493) |
| **33.** | *Skatchkov SN, Rojas L, Eaton MJ, Orkand RK, Biedermann B, Bringmann A, Pannicke T, Veh RW, Reichenbach A. Functional expression of Kir 6.1/SUR1-K(ATP) channels in frog retinal Müller glial cells. Glia. 2002 May; 38(3):256-67.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/11968063) |
| **34.** | *Eaton MJ, Skatchkov SN, Brune A, Biedermann B, Veh RW, Reichenbach A. SURI and Kir6.1 subunits of K(ATP)-channels are co-localized in retinal glial (Müller) cells. Neuroreport. 2002 Jan 21; 13(1):57-60.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/11924895) |
| **35.** | *Bolshakov KV, Essin KV, Buldakova SL, Dorofeeva NA, Skatchkov SN, Eaton MJ, Tikhonov DB, Magazanik LG. Characterization of acid-sensitive ion channels in freshly isolated rat brain neurons. Neuroscience. 2002; 110(4):723-30.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/11934479) |
| **36.** | *Thomzig A, Wenzel M, Karschin C, Eaton MJ, Skatchkov SN, Karschin A, Veh RW. Kir6.1 is the principal pore-forming subunit of astrocyte but not neuronal plasma membrane K-ATP channels. Mol Cell Neurosci. 2001 Dec; 18(6):671-90.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/11749042) |
| **37.** | *Skatchkov SN, Thomzig A, Eaton MJ, Biedermann B, Eulitz D, Bringmann A, Pannicke T, Veh RW, Reichenbach A. Kir subfamily in frog retina: specific spatial distribution of Kir 6.1 in glial (Müller) cells. Neuroreport. 2001 May 25; 12(7):1437-41.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/11388425) |
| **38.** | *Skatchkov SN, Eaton MJ, Krusek J, Veh RW, Biedermann B, Bringmann A, Pannicke T, Orkand RK, Reichenbach A. Spatial distribution of spermine/spermidine content and K(+)-current rectification in frog retinal glial (Müller) cells. Glia. 2000 Jul; 31(1):84-90.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/10816609) |
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| **40.** | *Durham RA, Johnson JD, Eaton MJ, Moore KE, Lookingland KJ. Opposing roles for dopamine D1 and D2 receptors in the regulation of hypothalamic tuberoinfundibular dopamine neurons. Eur J Pharmacol. 1998 Aug 21; 355(2-3):141-7.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/9760028) |
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| **42.** | *Eaton MJ, Pagán OR, Hann RM, Eterovic VA. Differential effects of dimethyl sulfoxide on nicotinic acetylcholine receptors from mouse muscle and Torpedo electrocytes. Neurosci Lett. 1997 Jul 25; 230(3):163-6.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/9272686) |
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| **46.** | *Eaton MJ, Lookingland KJ, Moore KE. The sigma receptor ligand rimcazole alters secretion of prolactin and alpha-melanocyte stimulating hormone by dopaminergic and non-dopaminergic mechanisms. Eur J Pharmacol. 1996 Mar 28; 299(1-3):171-7.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/8901020) |
| **47.** | *Wagner CK, Eaton MJ, Moore KE, Lookingland KJ. Efferent projections from the region of the medial zona incerta containing A13 dopaminergic neurons: a PHA-L anterograde tract-tracing study in the rat. Brain Res. 1995 Apr 24; 677(2):229-37.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/7552247) |
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| **49.** | *Eaton MJ, Lookingland KJ, Moore KE. Effects of the selective dopaminergic D2 agonist quinelorane on the activity of dopaminergic and noradrenergic neurons projecting to the diencephalon of the rat. J Pharmacol Exp Ther. 1994 Feb; 268(2):645-52.* | [PubMed](http://www.ncbi.nlm.nih.gov/pubmed/7906734) |
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