The Robert Larner, M.D. College of Medicine
THE UNIVERSITY OF VERMONT

DAVID C. HILL-EUBANKS, PhD
CURRICULUM VITAE

Position: Faculty Scientist
Department of Pharmacology

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EDUCATION

Year	Instituition	Degree	Area of Degree, Accolades
1998	University of Vermont	PhD	Pharmacology
1979	Rice University	BA	Biology, *cum laude*

LICENSES, CERTIFICATION

None (N/A)

FACULTY POSITIONS HELD

Years	Institution	Academic Title	Department
2018-Present	University of Vermont	Faculty Scientist	Pharmacology
2002-2019	University of Vermont	Res. Asst. Prof.	Pharmacology

OTHER POSITIONS AND MAJOR ADMINISTRATIVE POSITIONS HELD

KEYWORDS/AREAS OF INTEREST

Muscle, Smooth/physiology; Myocytes, Smooth Muscle/physiology; Vascular system/physiology; Endothelium, Vascular/physiology; Calcium signaling; ion channels/physiology; electrophysiology; receptors, G-protein-coupled/physiology; Signal transduction; Neurovascular coupling; Microcirculation/physiology; Cerebrovascular Circulation/physiology; Cerebral Blood Flow/physiology; Cerebral small vessel diseases.

SUMMARY OF PROFESSIONAL ACTIVITIES- OVERALL

As a Faculty Scientist (formerly Research Assistant Professor), I have not had formal teaching or clinical responsibilities; accordingly, my professional efforts have been devoted entirely to research. Over the course of my career, I have pursued an unconventional but deeply productive trajectory that has brought me from coagulation biochemistry to receptor pharmacology, smooth muscle physiology, vascular biology, and ultimately to the molecular and network-level mechanisms governing neurovascular coupling. This path has been non-linear by design. Rather than concentrating narrowly on a single domain, I have consistently sought out problems at disciplinary boundaries, where conceptual cross-fertilization is not only possible but necessary. This broad scientific perspective has proven to be one of my most valuable research assets. Working in a field as conceptually fragmented as neurovascular coupling—where competing models often appear to describe different realities—demands the ability to integrate mechanistic insights across scales and systems. My background in Ca^{2+} signaling, ion channel physiology, G-protein-coupled receptor (GPCR) structure–function relationships, and vascular smooth muscle biology has allowed me to do exactly that. It has positioned me to contribute meaningfully to projects spanning the spectrum from molecular mechanisms to emergent microvascular behaviors, and to help frame these findings in ways that resonate with diverse audiences. Several long-standing research themes illustrate the through-lines linking these seemingly eclectic threads. Early work on the kinetics of factor VIII activation and its regulation by von Willebrand factor established a foundation in mechanistic biochemistry. Studies of GPCRs employing unbiased mutagenesis and functional screening identified structural motifs governing G-protein engagement and constitutive signaling—work that informed my later interest in receptor-dependent Ca^{2+} signaling. Investigations of Ca^{2+}-dependent transcriptional pathways, Ca^{2+} sparks and excitability in visceral smooth muscle, and microdomain-restricted TRPV4 signaling in resistance arteries provided conceptual framing for subsequent studies of how excitable and non-excitable cells convert ionic events into physiological outcomes. More recent efforts have elucidated how capillary endothelial Kir channels enable the brain's vast capillary network to detect neuronal activity and communicate this to upstream arterioles, how contractile pericytes regulate blood flow distribution within the arteriole–capillary transition zone, and how defects in extracellular matrix signaling involving a TIMP3/ADAM17/HB-EGF cassette contribute to impaired neurovascular coupling in CADASIL and related cerebral small vessel diseases. Each project reflects a commitment to understanding how local signaling events shape integrated vascular behavior. In addition to my research contributions, I have played a major role in shaping the presentation of our group's work through writing and editorial leadership. This role is further highlighted by a series of invited reviews on Ca^{2+} signaling (PMID: 21709182), purinergic signaling (PMID: 20660658), NFAT regulation in smooth muscle (PMID: 12586440), and the role of TRP channels in flow-induced dilation and myogenic tone (PMID: 25180264). Taken together, these activities reflect a career dedicated to advancing vascular biology through rigorous experimentation, conceptual integration, and clear scientific communication.

SUMMARY OF ACCOMPLISHMENTS

- **Factor VIII/von Willebrand factor binding and the kinetics of factor VIII activation:** These studies, which are routinely cited in coagulation and thrombosis textbooks, investigated proteolytic activation of factor VIII (fVIII), a component of the coagulation cascade, showing that release of fVIII from the fVIII/von Willebrand factor (vWF) complex, a prerequisite for fVIII to exert its procoagulant activity, is dependent on a single cleavage in the light chain of the fVIII molecule that is catalyzed by vWF.
- **Structure and function of G-protein–coupled receptors:** This series of studies used an unbiased approach—random saturation mutagenesis coupled with functional screening—to identify structural features of a G-protein–coupled muscarinic receptor (m5) that define the G-protein binding domain and establish a structural basis for constitutive receptor activity.

- **Ca^{2+}-dependent transcriptional regulation in smooth muscle:** This work placed activation of the Ca^{2+}-dependent transcription factor, NFAT (nuclear factor of activated T cells), in smooth muscle cells in context with previously established activation paradigms in neurons and non-excitable cells, showing that basic features of the process are fundamentally different in smooth muscle.
- **Ca^{2+} sparks & urinary bladder smooth muscle excitability:** This work analyzed communication pathways in urinary bladder and corpus cavernosum smooth muscle, focusing on areas of functional convergence and divergence, with a specific emphasis on cGMP-dependent protein kinase (PKG)-dependent processes.
- **Regulation of myoendothelial function by signaling microdomains:** These studies investigated the role of an ion channel signaling network within myoendothelial projection microdomains in regulating normal myoendothelial function, demonstrating that Ca^{2+} influx via a very small number of TRPV4 (transient receptor potential vanilloid type 4) channels is sufficient to maximally dilate mestenteric arteries through activation of small- and intermediate-conductance K^+ channels and subsequent endothelial-dependent hyperpolarization of smooth muscle.
- **K^+ sensing & electrical signaling by brain microvascular endothelial cell Kir channels in neurovascular coupling:** These studies investigated the role of capillary endothelial cell strong inwardly rectifying K^+ (Kir) channels in mechanisms that link neuronal activity to localized increases in blood flow (neurovascular coupling), demonstrating that these channels initiate a propagating retrograde electrical signal that dilates upstream arterioles, enabling the vast capillary network to sense changes in neuronal activity and direct blood flow towards active brain regions.
- **Contractile pericytes as regulators of blood flow distribution in brain capillary networks:** This work identified a parenchymal arteriole-proximate region of the capillary bed, generally termed the arteriole-capillary transition (ACT) zone, as an important site of pericyte-mediate control over blood flow, demonstrating that junctional contractile pericytes in this region are capable of differentially controlling blood flow at capillary branches and facilitating $K_{IR}2.1$ channel-mediated increases in blood flow towards stimulation sites.
- **Molecular basis of vascular impairments in cerebral small vessel diseases (cSVDs):** These studies, employing a mouse model of CADASIL (cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy)—the most common genetic form of cSVD and a model of more common sporadic forms—have demonstrated that vascular deficits, including diminished arteriolar tone and impaired neurovascular coupling, are early manifestations of the disease that appear before the characteristic infarcts and cognitive deficits and have further shown that a common derangement in TIMP3/ADAM17/HB-EGF signaling is responsible for both channelopathy-like defects via different proximate mechanisms.

PROFESSIONAL SERVICE

N/A

TEACHING

N/A

CURRICULUM DEVELOPMENT

N/A

POSTGRADUATE AND OTHER COURSES

N/A

PREDOCTORAL STUDENTS SUPERVISED OR MENTORED

Dates	Name	Program/School	Role	Current Position
2004-2008	Jeff Layne	Pharmacology	Thesis committee	Industry scientist

DISSERTATION/THESIS COMMITTEE MEMBERSHIP
Jeff Layne (~2004-2008)

POSTDOCTORAL FELLOWS AND RESIDENTS DIRECTLY SUPERVISED OR MENTORED
None

INFORMAL TEACHING

Year	One Sentence Description of Information Teaching
All	***Without formal teaching responsibilities, I have focused on informal mentorship, helping trainees develop proficiency in writing high-quality manuscripts and crafting competitive grant applications***

FACULTY MENTORED
N/A

OTHER VISITING FACULTY SUPERVISED
N/A

TEACHING AIDS
N/A

TEACHING AWARDS AND NOMINATIONS
N/A

SUMMARY OF TEACHING ACTIVITIES
Inferrable from the above.

RESEARCH AND SCHOLARLY ACTIVITIES

RESEARCH AWARDS AND GRANTS

Ongoing Research Support
Foundation Leducq “International Network of Excellence on Brain Endothelium: A Nexus for Cerebral Small Vessel Disease” (M. Dichgans & M.T. Nelson, Co-PI)
Award period: 01/01/2023–12/31/2028
Amount: $847,300
Role: Writer/Principal Editor
Description: This proposal uses single-cell to network-level approaches to explore brain capillary endothelial cell dysfunction as a common theme in cerebral small vessel diseases.

NIH R01 NS128963-01 “Lesions and loss of smooth muscle cells in the brain underlies small vessel disease” (M.T. Nelson & A. Joutel, Co-PI)
Award period: 08/01/2022–07/31/2027
Amount: $3,306,200
Role: Co-Investigator, Writer/Principle Editor
Description: This application tests the contribution of brain microvascular mural cell (arteriolar smooth muscle cells and contractile ACT zone pericytes) degeneration, which occurs early in the progression of ischemic cerebral small vessel disease (cSVD), to the pathogenesis of cSVDs.

1 R35 HL 140027-01 (M.T. Nelson, PI) *Capillaries as a sensory web that controls cerebral blood flow in health and disease*
Award period: 1/1/2019–12/31/2025
Amount: $6,355,741
Role: Co-Investigator; Writer/Principal Editor
Description: This program, which extends the research in 1R01 HL121706 and 1R01 HL131181, seeks to integrate diverse aspects of cerebral blood flow regulation to provide a coherent view of the physiology of the brain vasculature.

Completed Research Support
Horizon 20/20 European Union 666881; In partnership with 11 European Institutions (M.T. Nelson, PI for WP1 & Co-PI for WP2-5) *Small vessel disease in a mechanistic perspective: Targets for intervention*
Award period: 01/01/16–12/31/2020
Amount: $493,095
Role: Co-Investigator; Writer/Principal Editor
Description: Exploration of affected pathways and mechanisms in the prevention of stroke and dementia.

1R01HL131181-01 (M.T. Nelson, PI) *K^+ sensing & electrical signaling by Kir channels in brain vasculature*
Award period: 02/01/16–01/31/20
Amount: $1,523,405
Role: Co-Investigator; Writer/Principal Editor
Description: To explore the role of the strong inward-rectifier K+ (Kir2) channels in the endothelium of the brain vasculature as a sensor of neuronal activity-derived K^+ that transmits an electrical signal to cause upstream dilation of PAs and surface arteries, and coordinate blood flow within the brain.

NIH R01 HL121706/1-5 (M.T. Nelson, PI) *Regulation of myoendothelial function by signaling microdomains in hypertension*
Award period: 7/1/2014–6/30/2019
Amount: $1,250,000.00
Role: Co-Investigator; Writer/Principal Editor
Description: To elucidate the role of a local, bidirectional, ion channel signaling network within myoendothelial projection microdomains in regulating normal myoendothelial function and to investigate its disruption in hypertension.

NIH R37 DK053832/15-19 (M.T. Nelson, PI) *Ca^{2+} sparks & urinary bladder smooth muscle excitability*
Award period: 8/1/1998–5/31/2019
Amount: $1,145,490.00
Role: Co-Investigator; Writer/Principal Editor
Description: To extend the analysis of communication pathways in urinary bladder and corpus cavernosum smooth muscle, focusing on areas of functional convergence and divergence.

Pending *[Competitive renewal just submitted.]*
R01 NS 1106562 (M.T. Nelson, PI) *Ion channel dysfunction in small vessel disease in the brain*
Award period: 04/15/19–03/31/24
Amount: $2,555,093
Role: Co-Investigator, Writer/Principal Editor
Description: The goal of this proposal is to study cerebral blood flow regulatory signaling mechanisms in small vessel disease by examining their operation in complex segments of the brain vasculature *in vitro*, *ex vivo*, *in vivo*, and *in silico*.

Selected Unfunded Grant Submissions (include scoring if available)

SCHOLARSHIP

Peer Reviewed Publications
Overall *h*-index: 38
Research Interest (RI) Score: 2,293

Original Research

1. Luehrsen K.R., Nicholson D.E., **Eubanks D.C.,** and Fox G.E. (1981) An archaebacterial 5s RNA contains a long insertion sequence. *Nature* 293:755. PMID: 6169998.
2. Duvernay V.H., **Eubanks D.C**., Perales R., Prestayko A.W., and Crooke S.T. (1982) The anti-tumor effects of anthracyclines: 2. The stereospecificity of the carbomethoxy group at position 10 of the class II anthracycline molecule. *Mol Pharm* 21:196-203. PMID: 6957713.
3. Mong S., **Eubanks D.C.**, Prestayko A.W., and Crooke S.T. (1982) Characterization of *in vitro* deoxyribonucleic acid breakage and crosslinking induced by bis-(isopropylamine) transdihydroxy-cis dichloroplatinum (IV). *Biochemistry* 21:3174-3180. PMID: 7049238.
4. Lollar P., **Hill-Eubanks D.C.,** and Parker C.G. (1988) Association of the factor VIII light chain with von Willebrand factor. *J Biol Chem* 263:10451-10455. PMID: 3134349.
5. **Hill-Eubanks D.C.**, Parker C.G., and Lollar P. (1989) Differential proteolytic activation of factor VIII-von Willebrand factor complex by thrombin. *Proc Natl Acad Science* 86:6508-6512. PMID: 2505252.
6. **Hill-Eubanks D.C.** and Lollar, P. (1990) von Willebrand factor is a cofactor for thrombin-catalyzed cleavage of the factor VIII light chain. *J Biol Chem* 265:17854-17858. PMID: 2120219.
7. Tecle H., Lauffer D.J., Davis R.E., Mirzadegan T., Moreland D.W., Swarz R.D., Thomas A.J., Raby C., **Eubanks D.**, Brann M.R. and Jaen J.C. (1995) (R)-(-)-(Z)-1-azobicyclo[2.2.1]heptan-3-one, O-(3-methyl-5-aryl-2-penten-4-ynyl) oxines: Potent muscarinic agonists. Bioorg & Med Chem Letters 5:637:642.
8. Jaen J., Barrett S., Brann M., Callahan M., Davis R., Doyle P., **Eubanks D.**, Lauffer D., Lauffer L., Lipinski W., Moreland D., Nelson C., Raby C., Schwarz R., Spencer C., and Tecle H. (1995) In vitro and in vivo evaluation of the subtype-selective muscarinic agonist PD 15382. Life Sciences 56:845-852. PMID: 10188784.

9. Messier T.L., Dorman C.M., Brauner-Osborne H., **Eubanks D.,** and Brann M.R. (1995) High throughput assays of cloned adrenergic, muscarinic, neurokinin and neurotrophin receptors in living mammalian cells. *Pharmacology & Toxicology* 76:308-311. PMID: 7567780.

10. Burstein E.S., Spalding T.A., **Hill-Eubanks D.,** and Brann M.R. (1995) Structure-function of muscarinic receptor coupling to G proteins. Random saturation mutagenesis identifies a critical determinant of receptor affinity for G proteins. *J Biol Chem* 270:3141-3146. PMID: 7852396.

11. Spalding T.A., Burstein E.S., Brauner-Osbourne H., **Hill-Eubanks D.,** and Brann M.R. (1995) Pharmacology of a constitutively active muscarinic receptor generated by random mutagenesis. *Pharm and Exp Therapeutics* 275:1274-1279. PMID: 8531092.

12. **Hill-Eubanks D.**, Burstein E.S., Spalding T.A., Brauner-Osbourne H., and Brann M.R. (1996) Structure of a G-protein-coupling domain of a muscarinic receptor predicted by random-saturation mutagenesis. *J Biol Chem* 271:3058-3065. PMID: 8621701.

13. Tecle H., Barrett S.D., Lauffer D.J., Augelli-Szafran C, Brann M.R., Callahan M.J., Caprathe B.W., Davis R.E., Doyle P.D., **Eubanks D**., Lipiniski W., Mirzadegan T., Moos W.H., Moreland D.W., Nelson C.B., Pavia M.R., Raby C., Schwarz R.D., Spencer C.J., Thomas A.J., Jaen J.C. (1998) Design and synthesis of m1-selective muscarinic agonists: (R)-(-)-(Z)-1-Azabicyclo[2.2.1]heptan-3-one, O-(3-(3'-methoxyphenyl)-2-propynyl)oxime maleate (CI-1017), a functionally m1-selective muscarinic agonist. J Med Chem 41(14):2524-36. PMID: 9651157.

14. Stevenson A.S., Gomez M.F., **Hill-Eubanks D.C.**, and Nelson M.T. (2001) NFAT4 movement in native smooth muscle: a role for differential calcium signaling. *J Biol Chem* 276:15018-24. PMID: 11278965.

15. Gomez M.F., Stevenson A.S., Bonev A.D., **Hill-Eubanks D.C.**, and Nelson M.T. (2002) Opposing actions of inositol 1,4,5-trisphosphate and ryanodine receptors on nuclear factor of activated T-cell regulation in smooth muscle. *J Biol Chem* 277:37756-64. PMID: 12145283.

16. Gomez M.F., Gonzalez Bosc L.V., Stevenson A.S., Wilkerson M.K., **Hill-Eubanks D.C.**, and Nelson M.T. (2003) Constitutively elevated nuclear export activity opposes Ca^{2+}-dependent NFATc3 nuclear accumulation in vascular smooth muscle: Role of JNK2 and Crm-1. *J Biol Chem* 278:46847-53. PMID: 12954637.

17. Gonzalez Bosc L.V., Wilkerson M.K., Eckman D.M., Bradley K.N., **Hill-Eubanks D.C.** and Nelson M.T. (2004) Intraluminal pressure is a stimulus for NFATc3 nuclear accumulation: role of calcium, endothelium-derived nitric oxide, and cGMP-dependent protein kinase. *J Biol Chem* 279:10702-9. PMID: PMID: 14688253.

18. Gonzalez Bosc L.V., Layne J.J., Nelson M.T., and **Hill-Eubanks D.C.** (2005) Nuclear Factor of Activated T-cells and Serum Response Factor cooperatively regulate the enhancer activity of a CArG box-containing α-actin intronic element. *J Biol Chem* 280:26113-20. PMID: 15857835.

19. Layne J.J., Werner M.E., **Hill-Eubanks D.C**., and Nelson M.T. (2008) NFATc3 Regulates BK Channel Function in Murine Urinary Bladder Smooth Muscle. *Am J Physiol* 295:C611-C623. PMCID: PMC2544435.

20. Sonkusare S.K., Bonev A.D., Ledoux J., Liedtke W., Kotlikoff M.I., Heppner T.J., **Hill-Eubanks D.C.**, and Nelson M.T. (2012). Elementary Ca^{2+} signals through endothelial TRPV4 channels regulate vascular function. *Science* 336(6081):597-601. PMCID: PMC3715993.

21. Dabertrand F., Hannah R.M., Pearson J.M., **Hill-Eubanks D.C.**, Brayden J.E., and Nelson M.T. (2013) Prostaglandin E_2, a postulated astrocyte-derived neurovascular coupling agent, constricts rather than dilates parenchymal arterioles. *J Cereb Blood Flow Metab* 33(4):479-82. PMCID: PMC3618402.

22. Dunn K.M., **Hill-Eubanks D.C.**, Liedtke W.B., and Nelson M.T. (2013) TRPV4 channels stimulate Ca^{2+}-induced Ca^{2+} release in astrocytic endfeet and amplify neurovascular coupling responses. *Proc Natl Acad Sci USA* 110(15):6157-62. PMCID: PMC3625327.

23. Longden T.A., Dabertrand F., **Hill-Eubanks D.C.**, Hammack S.E., and Nelson M.T. (2014) Stress-induced glucocorticoid signaling remodels neurovascular coupling through impairment of cerebrovascular inwardly rectifying K^+ channel function. *Proc Natl Acad Sci USA* 111(20):7462-7. PMCID: PMC4034203.

24. Gonzales A.L., Yang Y., Sullivan M.N., Sanders L., Dabertrand F., **Hill-Eubanks D.C.,** Nelson M.T., and Earley S. (2014) A PLCγ1-dependent, force-sensitive signaling network in the myogenic constriction of cerebral arteries. *Sci Signal* 7(327):ra49. PMCID: PMC4170587.

25. Sonkusare S.K., Dalsgaard T., Bonev A.D., **Hill-Eubanks D.C.**, Kotlikoff M.I., Scott J.D., Santana L.F., and Nelson M.T. (2014) AKAP150-dependent cooperative TRPV4 channel gating is central to endothelium-dependent vasodilation and is disrupted in hypertension. *Sci Signal* 7(333):ra66. PMCID: PMC4403000.

26. Dabertrand F., Krøigaard C., Bonev A.D., Cognat E., Dalsgaard T., Domenga-Denier V., **Hill-Eubanks D.C.**, Brayden J.E., Joutel A., and Nelson M.T. (2015) A potassium channelopathy-like defect underlies early-stage cerebrovascular dysfunction in a preclinical model of small vessel disease. *Proc Natl Acad Sci USA 112(7):E796-805*. PMCID: PMC4343103.

27. Collier D.M., **Hill-Eubanks D.C.,** and Nelson M.T. (2015) Orchestrating Ca^{2+} influx through Ca(V)1.2 and Ca(V)3.x channels in human cerebral arteries. *J Gen Physiol* 145(6):481-3. PMCID: PMC4442783.

28. Ye W., Chang R.B., Bushman J.D., Tu Y.H., Mulhall E.M., Wilson C.E., Cooper A.J., Chick W.S., **Hill-Eubanks D.C.,** Nelson M.T., Kinnamon S.C., and Liman E.R. (2016) The K^+ channel KIR2.1 functions in tandem with proton influx to mediate sour taste transduction. *Proc Natl Acad Sci USA* 113(2):E229-38. PMCID: PMC4720319.

29. Villalba N., Sackheim A., Nunez I., **Hill-Eubanks** D., Nelson M.T., Wellman G.C., and Freeman K. (2016) Traumatic brain injury causes endothelial dysfunction in the systemic microcirculation through arginase-1-dependent uncoupling of endothelial nitric oxide synthase. *J Neurotrauma* 33(1): 113-121. PMCID: PMC5198065.

30. Heppner T.J., Tykocki N.R., **Hill-Eubanks D.,** and Nelson M.T. (2016) Transient contractions of urinary bladder smooth muscle are drivers of afferent nerve activity during filling. *J Gen Physiol*. 147(4):323-35. PMCID: PMC4810069.

31. Povlsen G.K., Longden T.A., Bonev A.D., **Hill-Eubanks D.C.,** and Nelson MT. (2016) Uncoupling of neurovascular communication after transient global cerebral ischemia is caused by impaired parenchymal smooth muscle Kir channel function. *J Cereb Blood Flow Metab*. 36(7):1195-201. PMCID: PMC4929704.

32. Khavandi K., Baylie R.L., Sugden S.A., Ahmed M., Csato V., Eaton P., **Hill-Eubanks D.C.,** Bonev A.D., Nelson M.T., and Greenstein A.S. (2016) Pressure-induced oxidative activation of PKG enables vasoregulation by Ca^{2+} sparks and BK channels. *Sci Signal.* 11;9(449). PMCID: PMC5154376.

33. Longden T.A., Dabertrand F., Koide M., Gonzales A.L., Tykocki N.R., Brayden J.E., **Hill-Eubanks D.,** Nelson M.T. (2017) Capillary K^+-sensing initiates retrograde hyperpolarization to increase local cerebral blood flow. *Nat Neurosci*. 20(5):717-726. PMCID: PMC5404963

34. Baylie R., Ahmed M., Bonev A.D., **Hill-Eubanks D.C.,** Heppner T.J., Nelson M.T., and Greenstein A.S. (2017) Lack of direct effect of adiponectin on vascular smooth muscle cell BKCa channels or Ca^{2+} signaling in the regulation of small artery pressure-induced constriction. *Physiol Rep*. 5(16). PMCID: PMC5582259.

35. Harraz O.F., Longden T.A., Dabertrand F., **Hill-Eubanks D.,** Nelson M.T. (2018) Endothelial GqPCR activity controls capillary electrical signaling and brain blood flow through. PIP_2 depletion. *Proc Natl Acad Sci U S A.* 115(15):E3569-E3577. PMCID: PMC5899484.

36. Harraz O.F., Longden T.A., **Hill-Eubanks D.,** Nelson M.T. (2018) PIP_2 depletion promotes TRPV4 channel activity in mouse brain capillary endothelial cells. *Elife.* Aug 7. PMCID: PMC6117155.

37. Csato V., Kadir SZSA, Khavandi K., Bennett H., Sugden S., Gurney A.M., Pritchard H.T., **Hill-Eubanks D.,** Eaton P., Nelson M.T., and Greenstein A.S.. (2019) A Step and a Ceiling: mechanical properties of Ca^{2+} spark vasoregulation in resistance arteries by pressure-induced oxidative activation of PKG. *Physiol. Rep.* 7(22):e14260. PMCID: PMC6883097.

38. Gonzales A.L., Klug N.R., Moshkforoush A., Lee J.C., Lee F.K., Shui B., Tsoukias N.M., Kotlikoff M.I., **Hill-Eubanks D.,** Nelson M.T. (2020) Contractile pericytes determine the direction of blood flow at capillary junctions. *Proc Natl Acad Sci USA* 117(43):27022-27033. PMCID: PMC7604512.

39. Mughal A., Harraz O.F., Gonzales A.L., **Hill-Eubanks D.**, Nelson M.T. (2021) PIP_2 Improves Cerebral Blood Flow in a Mouse Model of Alzheimer's Disease. *Function* (Oxf) 2(2):zqab010. PMCID: PMC7955025.

40. Dabertrand F., Harraz O.F., Koide M., Longden T.A., Rosehart A.C., **Hill-Eubanks D.C.,** Joutel A., Nelson M.T. (2021) PIP_2 corrects cerebral blood flow deficits in small vessel disease by rescuing capillary Kir2.1 activity. *Proc Natl Acad Sci U A* 118(17):e2025998118. PMCID: PMC8092380.

41. Longden T.A., Mughal A., Hennig G.W., Harraz O.F., Shui B., Lee F.K., Lee J.C., Reining S., Kotlikoff M.I,. König G.M., Kostenis E., **Hill-Eubanks D.**, Nelson M.T. (2021) Local IP_3 receptor-mediated Ca^{2+} signals compound to direct blood flow in brain capillaries. *Sci. Adv.* 7(30):eabh0101. PMCID: PMC8294755.

42. Koide M., Harraz O.F., Dabertrand F., Longden T.A., Ferris H.R., Wellman G.C., **Hill-Eubanks D.C.**, Greenstein A.S., Nelson M.T. (2021) Differential restoration of functional hyperemia by antihypertensive drug classes in hypertension-related cerebral small vessel disease. *J Clin Invest* 131(18):e149029. PMCID: PMC8439604.

43. Sancho M., Klug N.R., Mughal A., Koide M., Huerta de la Cruz S., Heppner T.J., Bonev A.D., **Hill-Eubanks D.**, Nelson M.T. (2022) Adenosine signaling activates ATP-sensitive K^+ channels in endothelial cells and pericytes in CNS capillaries. *Sci. Signal.* 15(727):eabl5405. PMCID: PMC9623876.

44. Harraz O.F., Klug N.R., Senatore A.J., **Hill-Eubanks D.C.,** Nelson M.T. (2022) Piezo1 Is a Mechanosensor Channel in Central Nervous System Capillaries. *Circ Res.* 130(10):1531-1546. PMC9106929.

45. Taylor J.L., Pritchard H.A.T., Walsh K.R., Strangward P., White C., **Hill-Eubanks D.,** Alakrawi M., Hennig G.W., Allan S.M., Nelson M.T., Greenstein A.S. (2022) Functionally linked potassium channel activity in cerebral endothelial and smooth muscle cells is compromised in Alzheimer's disease. *Proc Natl Acad Sci USA* 119(26):e2204581119. PMCID: PMC9245656.

46. Klug N.R., Sancho M., Gonzales A.L., Heppner T.J., O'Brien R.I.C., **Hill-Eubanks D.,** Nelson M.T. (2023) Intraluminal pressure elevates intracellular calcium and contracts CNS pericytes: Role of voltage-dependent calcium channels. *Proc Natl Acad Sci USA.* 120(9):e2216421120. PMCID: PMC9992766.

47. Taylor J.L., Walsh K.R., Mosneag I.E., Danby T.G.E., Luka N., Chanda B., Schiessl I., Dunne R.A., **Hill-Eubanks D.,** Hennig G.W., Allan S.M., Nelson M.T., Greenstein A.S., Pritchard H.A.T. (2023) Uncoupling of Ca^{2+} sparks from BK channels in cerebral arteries underlies hypoperfusion in hypertension-induced vascular dementia. *Proc Natl Acad Sci USA* 15;120(33):e2307513120. PMCID: PMC10433456.

48. Ferris H.R., Stine N.C., **Hill-Eubanks D.C**., Nelson M.T., Wellman G.C., Koide M. (2023) Epidermal growth factor receptors in vascular endothelial cells contribut to functional hypermia in the brain. *Int. J. Mol. Sci.* 24(22):16284. PMCID: PMC10671586.

49. Dupré N., Gueniot F., Domenga-Denier V., Dubosclard V,. Nilles C., **Hill-Eubanks D.**, Morgenthaler-Roth C., Nelson M.T., Keime C., Danglot L., Joutel A. (2024) Protein aggregates containing wild-type and mutant NOTCH3 are major drivers of arterial pathology in CADASIL. *J. Clin. Invest.* 134(8):e175789. PMCID: PMC11014667.
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In Review

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In Preparation

None

Non-Peer Reviewed Publications

Review Articles

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2. Brann M.R., Jørgensen H.B., Burstein E.S., Spalding T.A., Ellis J., Jones S.V., **Hill-Eubanks D**. (1993) Studies of the pharmacology, localization, and structure of muscarinic acetylcholine receptors. *Ann N Y Acad Sci* 707:225-36. PMID: 9137555.
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4. Heppner T.J., Herrera G.M., Bonev A.D., **Hill-Eubanks D.**, and Nelson M.T. (2003) Ca^{2+} sparks and K(Ca) channels: novel mechanisms to relax urinary bladder smooth muscle. *Adv Exp Med Biol* 539:347-57. PMID: 15088917.
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9. Harraz O.F., **Hill-Eubanks D.**, Nelson M.T. (2020) PIP2: A critical regulator of vascular ion channels hiding in plain sight. *Proc Natl Acad Sci USA* 117(34):20378-20389. PMCID: PMC7456132.

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Books and Chapters

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2. **Hill-Eubanks D.C.**, Werner M.E., Heppner T.J., and Nelson M.T. (2011) Calcium signaling in smooth muscle. *Cold Spring Harb Perspect Biol* **3**(9):a004549. PMCID: PMC3181028.

Other Scholarly Publications

Abstracts
None

Patents Issued or Pending
None

Other Creative Activities
None

Quality Improvement and Patient Safety Activities
None

SUMMARY OF SCHOLARLY ACTIVITIES
Inferrable from the above.

INVITED PRESENTATIONS
None