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BRIEF BIOSKETCH

I focus on the physiology and treatment of abdominal and pelvic pain, with a particular emphasis on dysmenorrhea, or menstrual pain. Dysmenorrhea is the leading cause of school and work absenteeism and is a significant risk factor for chronic visceral pain among reproductive-aged women. My laboratory is at the forefront of this field, having developed innovative animal models, pioneered new diagnostic methods, and conducted groundbreaking treatment studies.

Our overarching goal is to systematically unravel the pathophysiology of dysmenorrhea and to create novel, effective treatments. Simultaneously, I am dedicated to mentoring the next generation of investigators, equipping them with the tools and knowledge to transform the study of visceral pain. Given the scarcity of laboratories committed to eradicating dysmenorrhea, our research is crucial in addressing one of the most common sources of suffering and a significant factor in gender disparity worldwide.

ACADEMIC APPOINTMENTS

2004-2009	Postdoctoral Scholar, Department of Neurobiology, Pharmacology and Physiology, University of Chicago, Chicago, IL
2009-2010	Research Associate (Assistant Professor), Department of Neurobiology, University of Chicago
2011-2019	Assistant Professor (part-time), Department of Obstetrics and Gynecology, University of Chicago
2019-	Research Associate Professor, Department of Obstetrics and Gynecology, University of Chicago
2010-	Research Scientist, Co-founder of GyRL, Department of Obstetrics and Gynecology, Endeavor Health/NorthShore University HealthSystem

ACADEMIC TRAINING

1994-1998	B.S., Computer Science, University of Wisconsin-Madison, Madison, WI
1998-2004	Ph.D., Neuroscience, The University of Pennsylvania, Philadelphia, PA

BOARD CERTIFICATION AND LICENSURE

2010-	CITI Certification in IRB compliance for Clinical Research, Good Clinical Practice & IACUC Leadership
2012-	Federal and Illinois DEA licensure for research-class drugs

SCHOLARSHIP

(a) Peer-reviewed publications in the primary literature, exclusive of abstracts:

1. Lytton WW, Hellman KM, Sutula TP. 1998. Computer Models of Hippocampal Circuit Changes of the Kindling Model of Epilepsy. Artificial Intelligence in Medicine 13(1-2):81-97. http://dx.doi.org/10.1016/S0933-3657(98)00005-0

2. Graves LA, Hellman KM, Veasey S, Blendy JA, Pack AI, Abel T. 2003. Genetic Evidence for a Role of CREB in Sustained Cortical Arousal. Journal of Neurophysiology 90(2):1152-1159. http://jn.physiology.org/cgi/content/full/90/2/1152

3. Ouyang M, Hellman KM, Abel T, Thomas SA. 2004. Adrenergic Signaling Plays a Critical Role in the Maintenance of Waking and in the Regulation of REM Sleep. Journal of Neurophysiology 92(4):2071-2082 http://jn.physiology.org/cgi/content/full/92/4/2071

4. Keeley MB, Wood MA, Isiegas C, Stein J, Hellman KM, Hannenhalli S, Abel T. 2006. Differential Transcriptional Response to Non-Associative and Associative Components of Classical Fear Conditioning in the Amygdala and Hippocampus. Learning and Memory 13(2):135-142. http://learnmem.cshlp.org/content/13/2/135.long

5. Brink TS, Hellman KM, Lambert AM, Mason P. 2006. Raphe Magnus Neurons Help Protect Reactions to Visceral Pain from Interruption by Cutaneous Pain. Journal of Neurophysiology 96(6):3423-3432. http://jn.physiology.org/cgi/content/full/96/6/3423

6. Hellman KM, Abel T. 2007. Fear Conditioning Increases NREM sleep. Behavioral Neuroscience 121(2):310-323. http://psycnet.apa.org/journals/bne/121/2/310.pdf

7. Hellman KM, Brink TS, Mason P. 2007. Activity of Murine Raphe Magnus Cells Predicts Tachypnea and On-going Nociceptive Responsiveness. Journal of Neurophysiology 98(6):3121-3133. http://jn.physiology.org/cgi/content/full/98/6/3121

8. Hellman KM, Mendelson SJ, Mendez-Duarte MA, Russell JL, Mason P. 2009. Opioid Microinjection into Raphe Magnus Modulates Cardiorespiratory Function in Mice and Rats. American Journal of Physiology 297(5): R1400-8 http://ajpregu.physiology.org/cgi/reprint/00140.2009v1.pdf

9. Hellman KM, Hernandez P, Young A, Park A, Abel T. 2010. Genetic Evidence that Protein Kinase A Regulates Thalamocortical Oscillations during NREM Sleep. Sleep 33(1):19-28. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2802244/

10. Tu FF, Hellman KM, Backonja M. 2011. Gynecological Management of Neuropathic Pain. American Journal of Obstetrics & Gynecology 205(5):435-443. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3205239/

11. Hellman KM, Mason P. 2012. Opioids Disrupt Pro-nociceptive Modulation Mediated by Raphe Magnus. Journal of Neuroscience 40:13668-13678. http://www.jneurosci.org/content/32/40/13668.long

12. Tu FF, Epstein AE, Pozolo KE, Sexton DL, Melnyk AI, Hellman KM. 2013. A Noninvasive Bladder Sensory Test Supports a Role for Dysmenorrhea Increasing Bladder Noxious Mechanosensitivity. Clinical Journal of Pain 29(10):883-890. http://dx.doi.org/10.1097/AJP.0b013e31827a71a3

13. Westling AM, Tu F, Griffith JW, Hellman KM. 2013. The Association of Dysmenorrhea with Noncyclic Pelvic Pain Accounting for Psychological Factors. American Journal of Obstetrics & Gynecology 209(5): 422.e1-422.e10 http://dx.doi.org/10.1016/j.ajog.2013.08.020

14. Hellman KM, Patanwala IY, Pozolo KE, Tu FF. 2015. Multimodal Nociceptive Mechanisms Underlying Chronic Pelvic Pain. American Journal of Obstetrics & Gynecology 213(6):827.e1-9. http://dx.doi.org/10.1016/j.ajog.2015.08.038

15. Tu FF, Kane J, Hellman KM. 2016. Noninvasive Experimental Bladder Pain Assessment in Painful Bladder Syndrome. British Journal of Obstetrics & Gynecology 124(2):283-291. http://dx.doi.org/10.1111/1471-0528.14433

16. Hellman KM, Yu PY, Oladosu, FA Segel C, Han A, Prasad PV, Jilling T, Tu FF. 2017. The Effects of Platelet-Activating Factor on Uterine Contractility, Perfusion, Hypoxia, and Pain in Mice. Reproductive Sciences 25(3):384-394 http://doi.org/10.1177/1933719117715122

17. Oladosu FA, Tu FF, Hellman KM. 2017. Nonsteroidal Anti-inflammatory Drug Resistance in Dysmenorrhea: Epidemiology, Causes, and Treatment. American Journal of Obstetrics & Gynecology 218(4):390-400 http://doi.org/10.1016/j.ajog.2017.08.108

18. Hellman KM, Kuhn CS, Tu FF, Dillane KE, Shlobin NA, Senapati S, Zhou X, Li W, Prasad PV. 2018. CINE MRI During Spontaneous Cramps in Women with Menstrual Pain. (2018) American Journal of Obstetrics & Gynecology. 218(5):506.e1-506.e8 http://doi.org/10.1016/j.ajog.2018.01.035

19. Zuckerman RM, Silton RL, Tu FF, Eng JS, Hellman KM. 2018. Somatic Symptoms in Women with Dysmenorrhea and Noncyclic Pelvic Pain. Archives of Women's Mental Health. 21: 533-541 http://doi.org/10.1007/s00737-018-0823-4

20. Hellman KM, Datta A, Steiner ND, Kane J, Garrison EF, Clauw DJ, Tu FF. 2018. Identification of Experimental Bladder Sensitivity Among Dysmenorrhea Sufferers. American Journal of Obstetrics & Gynecology. 219(1):84.e1-84.e8. http://doi.org/10.1016/j.ajog.2018.04.030

21. Oladosu FA, Tu FF, Farhan S, Garrison EF, Steiner ND, Roth GE, Hellman KM. 2018. Abdominal Skeletal Muscle Activity Precedes Spontaneous Menstrual Cramping Pain in Primary Dysmenorrhea. American Journal of Obstetrics & Gynecology. 219(1):91.e1-91.e7. http://doi.org/10.1016/j.ajog.2018.04.050

22. Oladosu FA, Hellman KM, Ham PJ, Kochlefl L, Datta A, Garrison EF, Steiner ND, Roth GE, Tu FF. 2019. Persistent Autonomic Dysfunction and Bladder Sensitivity in Primary Dysmenorrhea. Scientific Reports 18;9(1):2194. http://doi.org/10.1038/s41598-019-38545-3

23. Tu FF, Datta A, Atashroo D, Senapati S, Roth G, Clauw D, Hellman KM. 2020. Clinical Profile of Comorbid Dysmenorrhea and Bladder Sensitivity: A Cross-Sectional Analysis. American Journal of Obstetrics & Gynecology. 222(6): 594.e1-e11 http://10.1016/j.ajog.2019.12.010

24. Oladosu FA, Tu FF, Garfield LB, Garrison EF, Steiner ND, Roth GE, Hellman KM. 2020. Low Serum Oxytocin Concentrations are Associated with Painful Menstruation. Reproductive Sciences. 27(2):668-674. http://doi.org/10.1007/s43032-019-00071-y

25. Hellman KM, Roth GE, Dillane, KE, Garrison EF, Clauw D, Tu FF. 2020. Dysmenorrhea Subtypes Exhibit Differential Quantitative Sensory Assessment Profiles. PAIN. 161(6):1227-1236. http://doi.org/10.1097/j.pain.0000000000001826

26. Oladosu FA, Tu FF, Garrison EF, Dillane, KE, Roth GE, Hellman KM. 2020. Low Serum Naproxen Concentrations Are Associated with Minimal Pain Relief: A Preliminary Study in Women with Dysmenorrhea. Pain Medicine. http://doi.org/10.1093/pm/pnaa133

27. Kantarovich D, Vollbrecht HB, Cruz SA, Castillo H, Lee CS, Kushner J, Leng JX, Morgan VK, Hellman KM. 2020. Wikipedia: A Medical Student Educational Project to Edit Wikipedia in Preparation for Practicing Evidence-Based Pain Medicine. Journal of Medical Education and Curricular Development. 21(11): 3102-3108 https://doi.org/10.1177%2F2382120520959691

28. Britto K, Shilpa I, Hellman KM, Laveaux S. 2021. Racial Distribution and Characterization of Pelvic Organ Prolapse in a Hospital-Based Subspecialty Clinic. Female Pelvic Medicine & Reconstructive Surgery. Female Pelvic Medicine & Reconstructive Surgery 27.3: 147-150. https://doi.org/10.1097/SPV.0000000000001016

29. Wilson SH, Hellman KM, James D, Adler AC, Chandrakantan A. 2021. Mechanisms, Diagnosis, Prevention and Management of Perioperative Opioid-induced Hyperalgesia. Pain Management 11(4):405-417. https://doi.org/10.2217/pmt-2020-0105

30. Tu FF, Hellman KM, Roth GE, Dillane KE, Walker LS. 2022. Noninvasive Bladder Testing of Adolescent Females to Assess Visceral Hypersensitivity. Pain. Jan 1; 163(1):100-109.

https://doi.org/10.1097/j.pain.0000000000002311

31. Hellman KM, Oladosu FA, Garrison EF, Dillane, KE, Roth GE, Tu FF. 2021. Circulating Sex Steroids and Bladder Pain Sensitivity in Dysmenorrhea. Molecular Pain 17: 17448069211035217
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8549469/

32. Kmiecik MJ, Tu FF, Silton RL, Dillane KE, Roth GE, Harte SE, Hellman KM. 2021. Cortical Mechanisms of Visual Hypersensitivity in Women at Risk for Chronic Pelvic Pain. Pain. Epub 2021 August 27.
https://doi.org/10.1101/2020.12.03.20242032

33. Wilson SH, Hellman KM, James D, Adler AC, Chandrakantan A. 2021. "Mechanisms, Diagnosis, and Medical Management of Hyperalgesia: An Educational Review" Current Anesthesiology Reports. 2021 April 11(4):405-417
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8023328/

34. Kantarovich D, Dillane KE, Garrison EF, Oladosu FA, Schroer MS, Roth GE, Tu FF, Hellman KM. 2021. Development and Validation of a Real-time Method Characterizing Spontaneous Pain in Women with Dysmenorrhea. Journal of Obstetrics and Gynecology Research. Apr;47(4):1472-80.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8317258/

35. Oh H, Ehrenpreis ED, Tu FF, Dillane KE, Garrison EF, Leloudas N, Prasad PV, Hellman KM. 2022. Menstrual Cycle Variation in MRI-based Quantification of Intraluminal Gas in Women with and without Dysmenorrhea. Frontiers in Pain. May 11;3:720141.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9130698/

36. Schrepf A, Hellman KM, Bohnert AM, Williams DA, Tu FF. 2022. Generalized Sensory Sensitivity is Associated with Comorbid Pain Symptoms: A Replication Study in Women with Dysmenorrhea. PAIN. 164(1): 142-148.
https://doi.org/10.1097/j.pain.0000000000002676

37. Shlobin AE, Tu FF, Sain CR, Kmiecik MJ, Kantarovich D, Singh L, Wang CE, Hellman KM. 2023. Bladder Pain Sensitivity Is a Potential Risk Factor for Irritable Bowel Syndrome. Digestive Disease Science July. 68(7):3092-3102
https://pubmed.ncbi.nlm.nih.gov/36879177/

38. Kmiecik MJ, Tu FF, Clauw DJ, Hellman KM. 2023. Multimodal Hypersensitivity Derived from Quantitative Sensory Testing Predicts Pelvic Pain Outcome: An Observational Cohort Study. Pain September 1;164(9):2070-2083.
https://pubmed.ncbi.nlm.nih.gov/37226937/

39. Tu FF, Hellman KM, Darnell SE, Harber KA, Bohnert AM, Singh L, Walker LS. 2024. A Multidimensional Appraisal of Early Menstrual Pain Experience. American Journal of Obstetrics and Gynecology Jan 28: S0002-9378(24)00058-9
https://pubmed.ncbi.nlm.nih.gov/38290643/

40. Cockrum RH, Tu FF, Kierzkowska O, Leloudas N, Pottumarthi PV, Hellman KM. 2024 Ultrasound and MRI-based Investigation of the Role of Perfusion and Oxygen Availability in Menstrual Pain. Jan 29:S0002-9378(24)00059-0. https://pubmed.ncbi.nlm.nih.gov/38295969/

41. Medved M, Harmath CB, Siblini H, Giurcanu M, Kulkarni K, Hellman KM, Madueke-Laveaux OS. Quant Imaging Med Surg. 2024 Jul 1;14(7):4362-4375. doi: 10.21037/qims-23-1663. https://www.ncbi.nlm.nih.gov/pmc/articles/ PMC11250352/

42. Reina E, Hellman KM, Kmiecik MK, Tu FF, Associations between menstrual pain and sexual function: The role of visceral hypersensitivity on developing sexual pain" *in press* Journal of Sexual Medicine.

(b) Non-peer-reviewed original articles

1. Mason P, Hellman KM. Review of Unmasking the Tonic-aversive State in Neuropathic Pain. 2009. Faculty of 1000: 12(11): 1364-1366 http://f1000.com/prime/1168048

2. Mason P, Hellman KM. Review of Changes in Expression of NMDA-NR1 Receptor Subunits in the Rostral Ventromedial Medulla Modulate Pain Behaviors. 2010 October; Faculty of 1000: 151(1): 155-161 http://f1000.com/prime/6657956

3. Mason P, Hellman KM. Review of Acetate Causes Alcohol Hangover Headache in Rats. 2010. Faculty of 1000: 5(12): e15963 http://f1000.com/prime/8249956

4. Hellman KM. Summary of Relevant Findings at SFN. 2016. Pelvic Pain Special Interest Group Newsletter (January 2016)

5. Hellman KM, Tu FF. Reply to Ruan et al. Multimodal Nociceptive Mechanisms Underlying Chronic Pelvic Pain. American Journal of Obstetrics & Gynecology, 250(1): 132 – 133. http://dx.doi.org/10.1016/j.ajog.2016.02.051

6. Hellman K, Tu F. Primary Dysmenorrhea: Diagnosis and Therapy. Obstetrics & Gynecology. (2021) 137(4):752. https://doi.org/10.1097/aog.0000000000004341

7. Hellman KM, Tu FF, Hal. How Many Types of Pelvic Pain are There? Pain (2024). *In press*

(c) Posters, Talks, or Published Abstracts:

1. Hellman KM, Lytton WW, Kornguth SE, Sutula TP. 1996. Computer Modeling of Dentate Gyrus used to Devise Physiological Tests of Connectivity. Talk: Society of Neuroscience. Washington DC.
2. Lytton WW, Hellman KM, Sutula TP. 1996. Computer Network Model of Mossy Fiber Sprouting in Dentate Gyrus. Talk: American Epilepsy Society.
3. Lytton WW, Hellman KM, Lynch MW, Sutula TP. 1997. Sparse Inter-blade Sprouting Enhances Sustained Activity in Computer Model of Kindled Dentate Gyrus Slices. Talk: American Epilepsy Society.

4. Hellman KM, Finkel LH, Contreras D. 1999. Computer Modeling of Compartmental Neurons used to Estimate Sodium Channel Density. Talk: Society for Neuroscience. Miami, FL.
5. Hellman KM, Graves, L, Pack A, Abel T. 2001. Alterations in Sleep After Fear Conditioning. Talk: APSS. Chicago, IL.
6. Hellman KM, Ouyang M, Abel T, Thomas SA. 2003. Adrenergic Signaling Plays a Critical Role in the Maintenance of Waking and in the Regulation of REM Sleep. Talk: APSS. Chicago, IL.
7. Hellman KM, Young A, Abel T. 2004. Genetic Evidence that Protein Kinase A Regulates Thalamocortical Oscillations during NREM Sleep. Talk: APSS. Philadelphia, PA.
8. Hellman KM, Abel T. 2004. Fear Conditioning Increase NREM Sleep and Delta Oscillations. Talk: APSS. Philadelphia, PA.
9. Hellman KM, Brink TS, Mason P. 2005. The Response to Cutaneous, Not Visceral Stimulation, Predicts the Effects of Opioids on Raphe Magnus Neurons. Talk: Society for Neuroscience. Washington DC.
10. Hallman KM, Mendelson SJ, Mason P. 2007. Nonserotonergic Neurons Within the Raphe Magnus Regulate Respiratory Rate and Contribute to Opioidergic Respiratory Depression. Talk: Society for Neuroscience. San Diego, CA.
11. Ohlson E, Mendez-Duartes MA, Mendelson SJ, Hellman KM, Mason P. 2008. Naloxone Methiodide Pre-treatment Attenuates Respiratory Depression Consequent to Systemic Morphine. Talk: Society for Neuroscience. Washington DC.
12. Hellman KM, Mendelson SJ, Mendez-Duartes MA, Russel J, Ohlson E, Mason P. 2008. Opioid Signaling within Raphe Magnus Elicits Respiratory Depression in Mice, but Not Rats. Talk: Society for Neuroscience. Washington DC.
13. Hellman KM, Mason P. 2009. Anti-Lock Analgesia: A Pulsatile Model for Descending Opioid Modulation Suggested by Recordings of Medullary Neurons in Unanesthetized Mice. Talk: Society for Neuroscience. Chicago, IL.
14. Teo S, Khosla N, Hellman KM, Mason P. 2010. A Potential Role for Nociceptive-modularly Neurons in the Raphe Magnus in Intradermal Serotonin-induced Hyperalgesia. Talk: Society for Neuroscience. San Diego, CA.
15. Hellman KM, Tu FF. 2011. A New Mouse Model Supports a Role for TRPV1 in Uterine Nociception. Talk: Society for Neuroscience. Washington DC.
16. Hellman KM, Carrow J, Sandwick V, Yu P, Edelmuth A, Segel S. Tu F. 2011. Deciphering the Role of Uterine Contractility and Blood Perfusion in Uterine Pain. Talk: International Pelvic Pain Society Annual Fall Meeting. Las Vegas, NV.
17. Yu P, Jiling T, Segel C, Tu FF, Hellman KM. 2012. Deciphering the Roles of Inflammation, Neural Activity, Blood Perfusion and Uterine Contractility in Uterine Pain. Talk: Society for Gynecological Investigation. San Diego, CA.
18. Westling A, Tu FF, Griffith J, Hellman KM. 2012. Severity of Dysmenorrhea with Overall Elevated Pain Report. Talk: Society for Gynecological Investigation. San Diego, CA.
19. Yu P, Segel S, Jiling T, Tu FF, Hellman KM. 2012. Deciphering the Physiological Explanation for Uterine Pain. Talk: World Congress on Pain. Milan.
20. Hellman KM, Tu FF, Epstein AE, Sexton DL, Melnyk AI. 2012. A Novel Noninvasive Bladder Sensory Test Supports a Role for Dysmenorrhea Increasing Bladder Nociception. Talk: World Congress on Pain. Milan.
21. Tu FF, Hellman KM, Yu PY, Pozolo K. 2012. Reduced Pain Thresholds Among Women with Chronic Pelvic Pain and Dysmenorrhea. Talk: World Congress on Pain. Milan.
22. Hellman KM, Yu P, Segel C, Tu FF. 2012. Channel Rhodopsin Simulation Demonstrates a Role for Brainstem Modulation in Uterine Nociceptive Physiology. Talk: Society for Neuroscience. New Orleans, LA.

23. Hellman KM, Tu FF, Epstein AE, Sexton DL, Melnyk AI. 2012. A Novel Noninvasive Bladder Sensory Test Supports a Role for Dysmenorrhea Increasing Bladder Nociception. Talk: International Pain Society. Chicago, IL.
24. Tu FF, Hellman KM, Yu PY, Pozolo K. 2012. Reduced Pain Thresholds Among Women with Chronic Pelvic Pain and Dysmenorrhea. Talk: International Pelvic Pain Society. Chicago, IL.
25. Westling A, Tu FF, Griffith J, Hellman KM. 2012. Severity of Dysmenorrhea is Correlated with Overall Elevated Pain Report. Talk: International Pelvic Pain Society. Chicago, IL.
26. Hellman KM, Senapati S, Tu FF. 2012. Optical Diagnostics of Pain Targets during Laparoscopic Surgery. Talk: AAGL. Las Vegas, NV.
27. Hellman KM, Tu FF. 2012. Electrosnographic Diagnostics for Pelvic Pain. Talk: Translational Research to Inform Modern Medicine Symposium. Chicago, IL.
28. Senapati S, Tu FF, Hellman KM. 2013. Anxiety, Sleep, Pain Sensitivity and the Response to Laparoscopic Management of Pelvic Pain. Talk: AAGL. Washington DC.
29. Tu FF, Hellman KM. 2013. Myosonographic Diagnostics for Pelvic Pain. Talk: the 60th Annual Meeting of the Society for Gynecological Investigation. Orlando, FL.
30. Tu FF, Pozolo K, Resnick J, Borushko E, Hellman KM. 2013. Comparative Study of Clinical vs. Quantitative Measures of Pelvic Sensitivity. Washington DC.
31. Tu FF, Hellman KM. 2013. The Role of Visceral Motor Reflexes in Menstrual Pain. Talk: Society for Neuroscience.
32. Hellman KM, Tu FF. 2014. Behavioral and Physiological Characterization of the Molecules Involved in Menstrual Pain. Talk: Society for Neuroscience.
33. Tu F, Hellman KM, Kane K, Resnick J, Pozolo K, Borushko E. 2014. A Novel Visceral Sensitivity Task Evaluates Bladder Hyperalgesia Independent of Psychological Factors. Talk: International Association for the Study of Pain 15th World Congress on Pain. Buenos Aires, Argentina.
34. Zuckerman R, Kane J, Silton RL, Hellman KM, Tu FF. 2015. Somatic Complaint in Dysmenorrhea and Other Visceral Pains. Talk: Society for Neuroscience.
35. Dillane K, Harte SE, Polnazsek K, Tu FF Silton RL, Hellman KM. 2015. Neural Correlates of Sensory Amplification in Dysmenorrhea and Other Visceral Pain Conditions. Talk: Society for Neuroscience.
36. Polnazsek K, Dillane K, Tu FF, Hellman KM, Silton RL. 2015. The Effects of Depression, Dysmenorrhea, and Sensory Amplification on Resting State Brain Activity. Talk: Society for Neuroscience,
37. Rosenbaum J, Kuhn C, Tu FF, Hellman KM. 2015. Ultrasonographic Investigation of the Mechanisms Involved in Menstrual Cramps. Abstract: Journal of Minimally Invasive Gynecology. Talk: American Association of Gynecologic Laparoscopists 45th Global Congress of Minimally Invasive Gynecology. Las Vegas, NV.
38. Kuhn C, Senapati SS, Tu FF, Hellman KM. 2016. The Relationship Between fMRI Events Resembling Sustained Uterine Contractions and Spontaneous Menstrual Cramping Report. Talk: International Pelvic Pain Society. Chicago, IL.
39. Senapati SS, Tu FF, Kuhn C, Hellman KM. 2016. Functional Phenotyping of Menstrual Pain. Talk: American Association for Gynecologic Laparoscopists 45th Global Congress on Minimally Invasive Gynecology. Orlando, FL.
40. Zuckerman R, Hellman KM, Silton RL, Tu FF. 2016. The Distinct Roles of Somatization in Menstrual and Non-menstrual Pelvic Pain. Talk: International Pelvic Pain Society. Chicago, IL.
41. Polnazsek KL, Silton RL, Dillane K, Harte S, Tu F, Hellman K. 2016. Neural Correlates of Sensory Amplification in Women with Dysmenorrhea. Talk: Society for Affective Science. Chicago, IL.

42. Hellman KM, Silton RL, Dillane K, Polnaszek K, Harte S, Tu FF. 2016. Cortical Mechanisms of Sensory Amplification in Visceral Pain Conditions. Talk: IASP. Yokohama, Japan.
43. Hellman KM, Gebhart G, Garrison E, Steiner N, Kane J, Tu F. 2017. Development of a Paradigm to Prevent Bladder Pain. Talk: 3rd World Congress on Abdominal and Pelvic Pain Annual Meeting. Washington DC.
44. Laus K, Gebhart G, Garrison E, Steiner N, Tu F, Hellman K. 2017. Noninvasive Objective Measurement of Uterine Perfusion/Oxygenation and the Effects of Naproxen. Talk: 3rd World Congress on Abdominal and Pelvic Pain Annual Meeting. Washington DC.
45. Oladosu F, Gebhart G, Garrison E, Steiner N, Tu F, Hellman K. 2017. Menstrual Pain Unresponsive to Naproxen is Related to Low Naproxen Serum Levels. Talk: 3rd World Congress on Abdominal and Pelvic Pain Annual Meeting. Washington DC.
46. Domokos F, Polnaszek KL, Kahrilas IJ, Bryant FB, Tu FF, Hellman KM, Silton RL. 2017. Savoring Past and Present Positive Events Boosts Positive Affect for Individuals with Pain Symptoms. Talk: Midwestern Psychological Association. Chicago, IL
47. Cockrum RH, Tu FF, Roth GE, Garrison EF, Oladosu FA, Hellman KM. 2018. Development of a Method to Characterize Vascular Contributions to Cramping Pain in Dysmenorrhea. Abstract: The Journal of Minimally Invasive Gynecology Nov-Dec 25(7): S200 AAGL
48. Hellman KM, Laus K, Oladosu F, Garrison E. 2018. Noninvasive Objective MRI Method to Evaluate Uterine Hemodynamic Function and the Effects of Naproxen. Abstract: Reproductive Sciences 25: 118A-119A.
49. Hellman KM, Tu F, Garrison E, Oladosu F. 2018. Identification of a Preclinical Phenotype for the Development of Painful Bladder Syndrome in Women with Isolated Dysmenorrhea. Abstract: Reproductive Sciences 25:221A.
50. Oladosu F, Garrison E, Hellman K. 2018. NSAID-Resistant Menstrual Pain is Due to Low Serum Levels of NSAIDs. Abstract: Reproductive Sciences 2018 25:145A.
51. Kuhn C, Hellman K, Tu F. 2018. Cine MRI During Spontaneous Cramps in Women with Menstrual Pain. Talk: American Obstetrics and Gynecologists 66th Annual Clinical and Scientific Meeting. Austin, TX.
52. Roth G, Tu F, Oladosu F, Garrison E, Steiner N, Hellman K. 2018. Multisensory Hypersensitivity: A Possible Risk Factor in the Transition from Episodic to Chronic Pain Among Women with Dysmenorrhea. Talk: International Association for the Study of Pain 17th World Congress on Pain. Boston, MA.
53. Oladosu F, Tu F, Garrison E, Ham P, Steiner N, Roth G, Hellman K. 2018. The Interplay Between Heart Rate Variability and Conditional Pain Modulation in Dysmenorrhea. Talk: International Association for the Study of Pain 17th World Congress on Pain. Boston, MA.
54. Oladosu FA, Hellman KM, Garrison EF, Steiner ND, Roth GE, Tu FF. 2018. The Interplay between Heart Rate Variability and Conditioned Pain Modulation in Dysmenorrhea. Talk: International Association for the Study of Pain 17th World Congress on Pain. Boston, MA.
55. Oladosu FA, Tu FF, Garrison EF, Steiner ND, Roth GE, Hellman KM. 2018. Menstrual Pain Unresponsive to Naproxen is Due to Low Naproxen Serum Levels. Talk: University of Chicago Post-Doctoral Symposium. Chicago, IL.
56. Shlobin A, Tu FF, Oladosu FA, Garrison EF, Steiner ND, Roth GE, Hellman KM. 2018. Mechanisms Underlying the Comorbidity of Dysmenorrhea and IBS. Talk: International Pelvic Pain Society Annual Meeting. Chicago, IL.

57. Hellman K, Oladosu F, Tu F. 2019. (133) Preliminary Analysis of the Relationship between Refractory Menstrual Pain and Naproxen Metabolism. Abstract: The Journal of Pain 20.4: S9-S10. American Academy of Pain Medicine.
58. Roth GE, Hellman KM, Silton RL, Tu FF. 2019. Dimensional Analysis of Psychological Symptoms in Multimodal Experimental Pain Sensitivity in Women with Pelvic Pain. Talk: Annual Meeting of the Society for Research in Psychopathology. Buffalo, NY.
59. Hellman KM, Roth GE, Dillane KD, Steiner NS, Tu FF. 2019. Women with Heightened Dysmenorrhea and Silent Bladder Sensitivity Exhibit Broad Abnormalities in Pain Experience and Quantitative Sensory Testing. Talk: International Pelvic Pain Society. Toronto, Canada.
60. Tu FF, Schroer M, Ashenafi G, Hellman KM. 2020. Exploring the Neuro-immune Mechanisms Causing Distinctive Nociceptive Profiles in Women with Endometriosis vs. Pure Dysmenorrhea. Talk: Psychoneuroimmunology Research Society. Los Angeles, CA. (Canceled due to Covid – 19).
61. Ashenafi G, Schroer M, Tu FF, Hellman KM. 2020. The Contribution of Central vs. Peripheral Nociceptive Mechanisms and Psychosocial Factors to Pain Interference in Women with Menstrual Pain. Talk: Chicago Chapter for Neuroscience. Chicago, IL. (Canceled due to Covid – 19).
62. Tu FF, Schroer M, Ashenafi G, Hellman KM. 2020. Comparative Assessment of Nociceptive Mechanisms in Endometriosis vs. Primary Dysmenorrhea. Talk: AAGL. Virtual.
63. Brito K, Iver S, Glass D, Helllman K, Laveaux S. 2020. Racial Distribution and Characterization of Pelvic Organ Prolapse. Talk: American Urogynecological Society.
64. Cockrum R, Tu FF, Garrison EF, Dillane KE, Roth GE, Hellman KM. 2020. A Novel Method for Characterizing Spontaneous Menstrual Cramps with Uterine Artery Doppler Velocimetry. Talk: International Pelvic Pain Society.
65. Khosla K, Caldwell HD, Farhan S, Hellman KM, Tu FF. 2021. Characterizing Pain and Medication Use for Primary Dysmenorrhea. Talk: American Congress of Obstetrics and Gynecology.
66. Darnell S, Tu FF, Hellman KM, Walker LS. 2021. Noninvasive Bladder Test for Adolescents to Assess Visceral Hypersensitivity. Talk: International Association for the Study of Pain.
67. Reina E, Matteson K, Hellman K, Raker C. Tu F. 2021. Comparative Self-reported Sexual Functioning Relative to Pelvic Pain Burden. Oral Presentation: International Pelvic Pain Society 24th Annual Scientific Meeting. Baltimore, MD.
68. Hellman KM. 2022. Mechanistic Characterization of Uterine Pain. Talk: Massachusetts Institute for Technology: Center for Gynopathology Research.
69. Hellman KM. 2022. Menstrual Pain and Fibroids: The Science of Why it Hurts and How to Make it Hurt Less. Talk: Annual Fibroid Summit.
70. Hellman KM. 2022. The Role of Sex Hormones in Visceral Sensitivity. Plenary Lecture: United States Association for the Study of Pain Annual Meeting: Sex Differences Special Interest Group.
71. Hellman KM. 2022. Advances in Methods to Understanding the Transition from Episodic to Chronic Visceral Pain in Dysmenorrhea. Symposium: United States Association for the Study of Pain Annual Meeting.
72. Darnell SE, Tu FF, Hellman KM, Walker LS. 2022. Psychosocial and Visceral Pain Predictors of Early Menstrual Pain Experience. Poster: International Association for the Study of Pain (IASP) World Congress on Pain. Toronto, Canada.

73. Kmiecik MJ, Tu FF, Clauw DJ, Hellman KM. 2022. Multimodal Hypersensitivity Predicts Pelvic Pain Outcome 4 Years Later. Poster: International Association for the Study of Pain (IASP) World Congress on Pain. Toronto, Canada.
74. Darnell SE, Tu FF, Harber KE, Sain C, Kmiecik MJ, Hellman KM. 2022. Menstrual, Sensory and Psychological Factors Predict New Onset Chronic Pelvic Pain. Poster: International Association for the Study of Pain (IASP) World Congress on Pain. Toronto, Canada.
75. Kmiecik MJ, Tu FF, Darnell S, Harber K, Hellman KM. 2022. Early Cortical Mechanisms of Visual Discomfort in Premenarchal Adolescents. Poster: Society for Neuroscience Annual Meeting. San Diego, CA.
76. Hellman KM, Tu FF. 2022. Spinal Cord Imaging in Vestibuloydia. Webinar: International Pelvic Pain Society.
77. Hagy H, Rea E, Crowley SJ, Bohnert AM, Tu FF, Hellman KM. 2022. On the Brink: Sleep as a Protective Factor for Psychological Well-Being Among Peri-menarcheal Adolescent Girls. Talk: Society of Behavioral Medicine.
78. Siblini H, Medved M, Harmath C, Kulkarni K, Giger ML, Hellman KM, Laveaux S. 2022. Feasibility of Combined Magnetic Resonance (MR) Methods to Determine Predictive Factors of Uterine Fibroid (UF) Growth. Talk: Society for Reproductive Investigation.
79. Medved M, Harmath C, Kulkarni K, Giger ML, Hellman KM, Madueke-Laveaux S. 2022. Quantitative MRI of Uterine Fibroids and Correlation with Fibroid Size – A Pilot Study. Talk: American Roentgen Ray Society.
80. Hagy H, Bohnert AM, Tu FF, Hellman KM. 2022. Associations of Wake Time, Physical Activity and Task Switching in Adolescent Girls. Talk: International Neuropsychological Society.
81. Bohnert AM, Hagy HA, Rea E, Crowley SJ, Tu FF, Hellman KM. 2023. Variability in Sleep Among Girls During Early Adolescence: The Role of Environmental Sensitivity and Affect. Talk: Society of Behavioral Medicine.
82. Hagy HA, Rea E, Crowley SJ, Bohnert AM, Tu FF, Hellman KM. 2023. On the Brink: Sleep as a Protective Factor for Psychological Well-being Among Peri-menarcheal Adolescent Girls.
83. Julliete K, Tu FF, Hellman KM. 2023. Validation of a Functional MRI Method to Characterize Candidate Mechanisms in Menstrual Pain. Talk: International Pelvic Pain Society.
84. Medved M, Harmath C, Kulkarni K, Giger ML, Hellman K, Madueke-Laveaux OS. 2023. Quantitative MRI of Uterine Fibroids for Prediction of Growth Rate. Talk: ARRS.
85. Medved M, Harmath C, Kulkarni K, Giger ML, Hellman K, Madueke-Laveaux OS. 2023. Quantitative and Morphological Characterization of Uterine Fibroids on MRI for Prediction of Growth Rate. Talk: RSNA.
86. Osborne,NO, Hellman KM, Burda EM, Darnel SE, Schrepf AS, Tu FF. Multimodal Hypersensitivity Predicts the Development of Widespread Body Pain in Adolescents. United States Association for the Study of Pain 2024.
87. Osborne,NO, Hellman KM, Burda EM, Darnel SE, Schrepf AS, Tu FF.Predictors of widespread body pain development during the menarchal transition. International Association for the Study of Pain 2024.
88. Julliete K, Tu FF, Taytum K, Hirsch E, Hellman KM. A Functional MRI Method to Characterize Uterine Contractility and Menstrual Pain.Central Association of Obstetrics and Gynecology 2024.

(d) Books:

As a reviewer of scientific content:

1. Hellman, KM. 2011. Mason P. pp. 665, Oxford University Press. New York, NY.

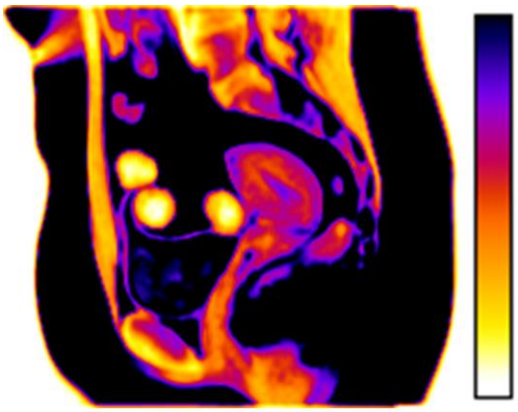
(e) Book chapters:

1. Hellman, KM, Abel, T. 2003. Molecular Mechanisms of Memory Consolidation. In: Maquet, P, Smith, C, Stickgold, R. 2003. Sleep and Brain Plasticity. Chapter 15, Oxford University Press, New York, NY.

(f) Other works that are publically available (websites, interviews, publications in the popular press, testimony, computer programs, protocols, reagents, inventions, patents not listed above, etc.)

2014 Interview on the Pelvic Messenger blogtalk RadioShow: The Holy Grail of Prevention of Pelvic Pain

2018 May 2018 Cover Issue of American Journal of Obstetrics and Gynecology:



Single pseudocolor frame from a new method for studying causes of menstrual pain using cine MRI. The full sequence shows myometrial activity before the perception of a self-reported menstrual cramp indicative of a potential cause of dysmenorrhea. [Image enhanced in ImageJ for cover artwork]

2018 Video on “The Mechanisms of Menstrual Pain” available on American Journal of Obstetrics and Gynecology

2018 IASP Global Year for Excellence in Pain Education Webinar: Abdominal and Pelvic Pain: Scientific Progress Vis-à-vis Clinical Evaluation and Management

2022 Annual Fibroid Summit: Menstrual Pain and Fibroids: The Science of Why it Hurts and How to Make it Hurt Less.

(g) Works in review, in preparation, etc. not yet publically available [list ONLY if available for BSD review]

1. Hellman KM, Silton RL, Dillane KE, Polnazsek K, Harte SE, Tu FF. In preparation. Cortical Mechanisms of Sensory Amplification in Visceral Pain Sensitivity. *Under revision for Journal of Pain.*
2. Polnazsek MA, Hellman KM, Tu FF, Silton RL. The Association of Steady State Prefrontal Cortical Activity with Bladder Sensitivity in Dysmenorrhea. *In preparation.*

FUNDING

(a) Past:

1.Epilepsy Foundation of America. PI: KM Hellman. My role: PI. Title: "Computational Model of Dentate Gyrus." Total direct costs: $5,000. Project period: 6/1/99-9/1/99.

2.NIH MH064329-02. PI: KM Hellman. My role: PI. Title: "Memory Consolidation and Sleep". Total direct costs: $60,000. Annual salary recovery or effort: Graduate Student National Research Service Award. Project period: 9/1/02-8/31/03.

3. NIH DA022429. PI: P. Mason. Role: Co-Investigator. Title: "Opioid Analgesia in Awake Mice." Total direct costs: $416,989.

4. American Academy of Sleep Medicine. PI: KM Hellman. My role: PI. Title: "Neurophysiological Investigation of Pain Induced Arousal." Total direct costs: $60,000. Project period: 1/4/06 – 1/3/07.

5. NorthShore Research Career Development Award. PI: KM Hellman. My Role: PI. Title: "Modulation of Pain and Autonomic Function." Total direct costs: $225,000. Project period: 10/1/10 – 10/1/13.

6. NIH HD081709. PI: KM Hellman. My Role: PI. Title: "Neurophysical Diagnostics for Menstrual Pain." Total direct costs: $414,996. Project period: 8/1/14 – 7/31/17.

7. NIH DK100368-04S1. My role: Mentor. Title: "Minority Training Supplement." Total direct costs: $156,674. Project period: 4/1/17 – 3/31/19.

8. NIH DK100368. My role: Co-Investigator. Title: "Deciphering the Hormonal and Nociceptive Mechanisms Underlying Bladder Pain." Total direct costs: $2,408,178. Project period: 4/1/14 – 3/31/19.

9. NIH HD09150102. PI: KM Hellman. My role: PI. Title: "Noninvasive Imaging off Uterine Physiology to Improve Dysmenorrhea." Total direct costs: $429,000. Project period: 8/2/17 – 7/1/21.

10. PI: KM Hellman. My role: PI. Title: "Deciphering the Mechanisms of Visceral Pain with Novel Spinal Functional Imaging." Total direct costs: $40,000. Project period: 9/16/21 – 9/15/22

(b) Current:

1. NIH R01HD098193. My role: PI. Title: "Mechanistic Characterization of Uterine Pain (M-CUP) to Improve Diagnosis and Treatment for Dysmenorrhea". Total direct costs: $2,826.940. Project period: 9/6/19 – 8/31/25.

2. NIH HD096332. My role: Co-Investigator. Title: "Early Menstrual Pain Impact on Multisensory Hypersensitivity." Total direct costs: $3,351,390. Project period: 8/22/19 – 5/31/25.

3. NIH DK100368. My role: Co-Investigator. Title: "Cross Organ Mechanisms in Chronic Pelvic Pain." Total direct costs: $3,408,178. Project period: 8/1/23 – 7/31/28.

4. DOD CP220085. My role: Co-Investigator. Title: "Nonsteroidal Anti-Inflammatory Drug (NSAID) Response and Central Sensitization of Pain in Women with Dysmenorrhea." Total direct costs: $408,756.

5. Pfizer. PI: KM Hellman. My role: PI. Title: "Phenotyping of Idiopathic Pelvic Pain with Real-Time Uterine Imaging and Relugolix-Combination Therapy Treatment." Total direct costs: $200,000. Project period: 5/1/24 – 8/31/25.

6. NIH R01 HD116714. My role: PI. "Targeting Interindividual Variability in NSAID Responses to Mitigate Chronic Pelvic Pain Risk in Dysmenorrhea." Total direct costs: $3,100,000. Project Period: 9/17/2024 – 8/31/2029

HONORS, PRIZES, AND AWARDS

1994 Frank Academic Scholar

1995 University of Wisconsin Honor Society

1997 Neuroscience Training Program Award for Outstanding Research in Neurobiology
2000 National Institute of Health, National Research Service Award
2004 Elliot Stellar Scholar
2004 U.W. Madison Neuropsychology Travel Fellowship
2006 American Academy of Sleep Medicine Faculty Research Award
2012 Best abstract on pelvic pain, AAGL Global Congress
2012 Honorable Mention poster award, International Pelvic Pain Society
2013 Best abstract on pelvic pain, AAGL Global Congress
2013 Best poster award, International Pelvic Pain Society
2014 New investigator of the year, NorthShore University HealthSystem
2016 Spotlighted abstract, Society for Affective Science
2017 Best paper award, Chicago Gynecological Society
2018 Faculty of 1000 recommended paper: Nonsteroidal anti-inflammatory drug resistance in dysmenorrhea: epidemiology, causes, and treatment.
2019 Poster Award: International Association for the Study of Pain—Abdominal and Pelvic Pain SIG
2020 90th Percentile Score Publon Reviewer
2021 Expertscape: top 1% publication record on pelvic pain over the past decade
2022 Poster Award: International Association for the Study of Pain—Abdominal and Pelvic Pain SIG
2024 Poster Award: United States Association for the Study of Pain
2024 Abstract Award: Central Association of Obstetrics & Gynecology (top abstract of 75 on clinical research)

INVITED SPEAKING

2004 Plenary Lecture, University of Pennsylvania, Philadelphia, PA
2008 Research seminar, University of Chicago
2009 Research seminar, Rosalind Franklin University Medical School
2010 Research seminar, Medical College of Wisconsin
2011 Research seminar, University of North Carolina
2012 Plenary Lecture, International Pelvic Pain Society, Chicago, IL
2012 Research seminar, Southern Illinois University Medical School
2012 Research seminar, St. Francis Hospital System
2014 Research seminar, NorthShore Scientific Society
2014 Research seminar, University of Chicago, Department of Obstetrics and Gynecology
2014 Research seminar, AAGL, Las Vegas, NV
2016 Research seminar, NorthShore University HealthSystem, Evanston, IL
2016 Research seminar, AAGL, Washington DC
2017 Research seminar, Loyola University Chicago
2017 Research seminar, University of Michigan
2017 Research seminar, Department of Psychiatry, RUSH University Medical Center, Chicago, IL
2018 Plenary Lecture, International Pelvic Pain Society
2018 Symposium/Webinar, International Association for Study of Pain
2019 Research seminar, NorthShore University HealthSystem
2021 Research seminar, International Pelvic Pain Society
2022 Research seminar, NorthShore University HealthSystem
2022 Research seminar, Massachusetts Institute for Technology
2022 Symposium/Webinar, Annual Fibroid Summit
2022 Symposium, United States Association for the Study of Pain

2022 Plenary Lecture, United States Association for the Study of Pain
2022 Research Webinar, International Pelvic Pain Society
2023 Research Webinar, National Institute for Child and Human Development
2023 Plenary Lecture, Endometriosis Foundation of America
2023 Plenary Lecture, Open Endoscopy Forum
2023 Symposium, American Autonomic Society
2024 Plenary Lecture, International Association for the Study of Pain
2024 Grand Rounds University of Colorado, Dept of Ob/Gyn (UCHealth)

INVITED, ELECTED, OR APPOINTED EXTRAMURAL SERVICE

2009-2011 Councilor, Society for Neuroscience, Chicago Chapter
2016 Grant Reviewer, US-Israel Binational Science Foundation
2016 Grant Reviewer, Austrian Science Fund
Various ad hoc reviewer for American Journal of Obstetrics & Gynecology, American Journal of Physiology, Anesthesiology, Brain Research, Molecular Pain, Elife, Environmental Research, Journal of Neurophysiology, Journal of Neuroscience Methods, Journal of Pain, Journal of Visual Experiments, Neuroimage, Neuroscience, PAIN, Physiology & Behavior, PLoS ONE
2019 NIH Study Section: ZRG1 IFCN N 55: Discovery and Validation of Novel Safe Effective Pain Treatments
2020 NIH Study Section: Integrative and Clinical Endocrinology and Reproduction
2020 Reviewing Editor Pain Medicine
2021 Reviewing Editor Frontiers in Pain
2021 Medical Research Council Grant Reviewer: Advanced Pain Discovery Platform
NIH Study Section: ZRG1 IFCN-J(02) Neurobiology of Pain and Itch
NIH Study Section: ZRG1 IFCN-J(55) Discovery and Validation of Novel Safe Effective Pain Treatments
2022 NIH Study Section: ZRG1 IFCN-J(02) Neurobiology of Pain and Itch
NIH Study Section: ZRG1 IFCN-J(55) Discovery and Validation of Novel Safe Effective Pain Treatments, NIH Study Section: Integrative and Clinical Endocrinology and Reproduction
2024 NIH Study Section, ZRG1 EMS-S (Endocrine and Metabolic Systems)
2024 Elected Treasurer of Abdominal Pelvic Pain Special Interest Group, International Association for the Study of Pain
2024 UK Wellbeing of Women (Reckitt Grant Peer Reviewer)

PROFESSIONAL SOCIETIES

Elected or invited membership:
Society for Reproductive Investigation (formerly SGI)
American Academy of Sleep Medicine
Society for Neuroscience
Faculty of 1000

Other:
International Pelvic Pain Society
International Association for the Study of Pain

EDUCATION

The University of Wisconsin-Madison (B.A., B.S.):
None

The University of Pennsylvania (Ph.D.):

2001-2002 Teaching Assistant, Biology
2003-2004 Teaching Assistant, Neurobiology

Continuing medical education:

2010- Annual Lecture on Pelvic Pain as part of the NorthShore Obstetrics and Gynecology Departmental Grand Rounds

Courses at the University of Chicago):

2013- Cluster Group Leader, Summer Research Program, Pritzker School of Medicine
2015- Mentor for Evidence-Based Medicine Presentations, Resident Physician Program
2018- Neuroscience of Pain and Opioids, MS4 Elective, Pritzker School of Medicine

Research trainees:

(a) High school students and teachers

2009-2010 Multiple students and high school teachers (20+), University of Chicago, Department of Anesthesiology. Course on Neurobiology as part of an educational grant from the American Recovery and Reinvestment Act of 2009

2014- Stevenson High School, SPARK: Summer scientific Internship Program. 5 high school students performed academic internships in my laboratory. Among them, *Saaniya Farhan** and *Nathan Shlobin** are coauthors on publications for valuable contributions. Saaniya Farhan (Penn state-TJU), Nathan Shlobin (Northwestern), and Christy Kang (UIC) are now combined undergraduate-MD students.

(b) Undergraduate (B.A., B.S.)

2005-2006 Eric Ohlson, University of Arizona. Presently medical student, University of Arizona. Research Project on Pain.
2005-2006 Jane Russel. Research Project on Respiration
2006-2007 Aaron Lambert, Currently Post-doctoral Scientist at Harvard.
2008-2009 Nasya Mendoza-Elias, University of Chicago. Currently a Resident in Neurosurgery at Mayo Clinic. Research Project on Respiration.
2008-2009 Marco Mendez-Duarte, University of Chicago. Research Project on Respiration.
2009-2010 Shaun Teo, University of Chicago (Graduated with Research Honors). Currently a graduate student at Rockefeller University.
2009 Natalia Khosla, University of Chicago. Currently a medical student at University of Chicago.
2011 Van Sandwick. Currently a consultant at Trinity Partners. Research project on pain.
2011 Jack Carrow. Currently a Ph.D. candidate at Texas A&M. Research project on optics/pain.
2010-2012 Peter Yu. Currently a medical student at Ohio State University. Research project on uterine pain.
2011-2012 Allyson Westling. Currently a medical student at Tufts. Research project on uterine pain.
2012-2013 Alice Han. Currently a medical student at University of Illinois. Research project on uterine pain.
2011-2013 Chaya Segel. Currently a licensed speech pathologist. Research project on uterine pain.

2014- Nathan Shlobin. Currently in combined undergraduate/medical school program at Northwestern University. Research project on MRI of uterine pain.
2015-2017 Julia Kane. Currently a student in educational psychology. Research project on bladder pain.

(c) Medical (M.D.)
2007-2008 Scott Mendelson, University of Chicago. Currently in Neurology Fellowship Program, UCLA.
2013-2015 Nita Padavil, University of Chicago. Currently a Psychiatry Resident, Northwestern. Science & Discovery Track mentorship.
2014-2018 Rebecca Zuckerman, University of Chicago. Summer Research Program mentorship.
2015- Carrie Kuhn, University of Chicago. (Awarded Calvin Fentress Fellowship to study mechanisms of menstrual pain in Dr. Hellman's laboratory). Currently a Resident in Ob/Gyn at University of Chicago.
2018- Diana Kantarovich, Rosalind Franklin Medical Student.
2019- Cosy Sain, University of Chicago. Science & Discovery Track mentorship.
2022 Ola Kierzkowska, Rosalind Franklin Medical Student.

(d) Graduate (Ph.D.)
2015- Kelly Polnazsek, Loyola University of Chicago.
2017- Avisek Datta, University of Illinois Chicago. Research project on bladder pain sensitivity in dysmenorrhea.
2019- Genevieve Roth. Research project on parental influence on somatization.
2019- Hannah Hagy. Research project on parent influence on sleep and pain behavior.

(e) Postdoctoral
2012-2014 Adam Gafni-Kane MD, University of Chicago. Urogynecological Fellowship. Research project on interstitial cystitis.
2013-2015 Insiyyah Patanwala MD, Indiana University. Assistant Professor, research project on quantitative sensory testing in pelvic pain.
2013-2015 Sarah Conduit-Hulbert/Wilkinson MD, University of Chicago. Resident Physician, research project on hormonal transcriptional factors in pelvic pain.
2014-2016 Jennifer Rosenbaum MD, University of Chicago. Resident Physician.
2015- Diana X. Zhou MD, University of Chicago. Resident Physician, research project on MRI analysis of pelvic pain.
2016- Folabomi A. Oladosu Ph.D, University of Chicago. Postdoctoral Scholar, research project on mechanisms of NSAID resistance.
2017- Katharina Laus MD, University of Chicago. Resident Physician.
2018- Richard Cockrum MD, University of Chicago. Resident Physician, research project on doppler ultrasound.
2020-2022 Matt Kmiecik PhD. Research projects on EEG and multimodal hypersensitivity.
2023- Natalie Osborne PhD. Research projects on EEG and dysmenorrhea.

SERVICE
University of Pennsylvania
Committee membership:
2004 Curriculum Committee

Leadership:

2002 Journal Club Coordinator

Other:
2003 Minority Outreach

University of Chicago/NorthShore University HealthSystem
Committee membership:
2013 NorthShore IRB Committee on Informed Consent
2014- NorthShore Pilot Grant Review Committee
2015- NorthShore Institutional Animal Care and Use Committee
2018- NorthShore Research Institute Staff Onboarding Committee

Leadership:
2012 Symposium Organizer and Moderator on “Translational Optogenetics”

Other:
2012- Pain Journal Club (between 5-10 participants every Wednesday)

SCHOLARSHIP STATEMENT

Prefatory background on women's visceral pain research and overview of my research portfolio

Chronic visceral pelvic pain disorders, including conditions such as endometriosis-associated pelvic pain, irritable bowel syndrome, and bladder pain syndrome, affect approximately 25% of women, causing significant morbidity and imposing a financial burden of over 50 billion dollars in lost wages and healthcare expenditures in the U.S. Despite their prevalence, these conditions are potentially predictable and preventable. However, funding for women's health research lags significantly behind, with women's conditions receiving 2-3 times less funding than men. Dysmenorrhea (period pain), a highly prevalent condition affecting 50-80% of women and a leading risk factor for chronic pelvic pain, receives only 0.1% of all pain research funding despite its disproportionate impact on racial and ethnic minorities.

Background in Women's Visceral Pain Research: My research trajectory, initially fostered in Peggy Mason's laboratory, has been dedicated to unveiling the complex interplay of brainstem neurons in visceral pain modulation and opioid analgesia. This foundational work debunked traditional beliefs on pain modulation and opioid efficacy (See Hellman and Mason CV #11), propelling me towards developing an innovative mouse model to explore uterine pain. This model elucidated the roles of prostaglandin and Platelet Activating Factor (PAF) in visceral pain, challenging conventional wisdom and highlighting PAF as a promising target for NSAID-resistant dysmenorrhea. Importantly, this animal model provided the fundamental conceptual foundation for the mechanisms (inflammation, contractility, and ischemic pain) underlying menstrual pain that I subsequently tested in humans, as described in the following sections.

Bridging Basic Science with Clinical Research: In collaboration with Dr. Frank Tu, I have embarked on groundbreaking studies involving over 2,000 participants to dissect the underpinnings of dysmenorrhea and bladder pain syndrome. Our noninvasive approach, leveraging advanced MRI techniques and comprehensive sensory testing, has provided unprecedented insights into the physiological and nociceptive dimensions of menstrual pain. Our innovative method of studying visceral pain noninvasively by evaluating physiological factors alongside spontaneous pain assessment has unveiled insights into the contributions of uterine and non-uterine components to menstrual pain. This work positions our lab among the few dedicated to addressing dysmenorrhea, a major source of suffering and gender disparity globally. Early intervention in pain evolution is crucial for shifting away from outdated chronic pain management paradigms, such as opioid reliance, towards more effective and preventive strategies.

Identifying At-risk Populations for Chronic Pelvic Pain: A cornerstone of my scholarship is the development of predictive models to identify women at heightened risk for chronic pelvic pain, utilizing innovative noninvasive tests. This endeavor not only aims to refine clinical diagnoses but also to pave the way for preemptive treatment strategies, thereby mitigating the transition from episodic to chronic pain states. As we continue to decode the multifaceted narrative of dysmenorrhea and related conditions, our research underscores the urgent need for a paradigm shift in the management of visceral pain. By challenging entrenched doctrines and exploring uncharted territories, we are committed to illuminating the path towards efficacious, evidence-based preventative strategies that promise reduce risk for the development of chronic pelvic pain to billions of women worldwide.

Peer-reviewed work, published or accepted, since emergence from postdoctoral training:

1. SYSTEMATIC DEFINITION OF THE MECHANISMS RESPONSIBLE FOR MENSTRUAL CRAMPING PAIN: A hidden, personal, and painful reality of many women is severe, gut-wrenching menstrual cramps occurring every 3-5 minutes for several days each month. This misery is a leading cause of missed school and work. These cramps are hallmarks of dysmenorrhea and are not sufficiently relieved by nonsteroidal anti-inflammatories (NSAIDs) in roughly 15% of reproductive-age women (CV #26). Unfortunately, the field has been plagued by limited research that often led to failed drug trials. My research has often systematically dismantled outdated notions of dysmenorrhea. For example, it has been widely hypothesized that increased levels of oxytocin lead to uterine contractions underlying menstrual pain. Thus, others have used oxytocin antagonists to relieve menstrual pain unsuccessfully. We have confirmed that this hypothesis is flawed because women with menstrual pain have reduced levels of oxytocin (CV #24).

To improve our understanding of the mechanisms underlying menstrual, I have pioneered novel MRI and pain testing methodologies to elucidate the pathophysiological mechanisms; traditional invasive approaches, such as intrauterine catheters and tissue biopsy, obscure the understanding of pain mechanisms and offer limited clinical diagnostic value. A crucial gap in dysmenorrhea research I identified was the need for more exploration of the temporal dynamics between myometrial activity and pain perception. Therefore, my focus has been on the natural occurrence of cramps, central to the dysmenorrhea experience, and their temporal relationship to physiology. To investigate the causality of myometrial contractile-induced ischemia in cramping symptoms, I devised and validated a novel real-time method using fMRI to monitor spontaneous pain (CV #34). Participants signal severe menstrual cramp onset by squeezing a bulb, while concurrent pelvic MRI captures myometrial signal fluctuations, showing decreases either concurrent with or 30-70 seconds before pain reports. The spatial and temporal patterns of these signal changes led me to propose that cramping pain stems from a mix of uterine pressure and hemodynamic dysfunction (**Exp pub#1**). This approach is groundbreaking in correlating the physiology of an internal pelvic organ with spontaneous pain reports, offering a new avenue for diagnosing pelvic and other visceral pain disorders through fMRI.

Most critically, our advanced ultrasonographic and MRI studies on uterine perfusion and oxygenation challenge the prevailing hypothesis that menstrual pain results from prostaglandin-induced uterine ischemia (**Exp pub #2**). Our findings indicate that dysmenorrhea sufferers exhibit better perfusion and oxygenation than controls, with naproxen sodium—known for inhibiting prostaglandin synthesis and alleviating menstrual pain—decreasing uterine perfusion and oxygen availability in affected individuals. This paradoxical pharmacological effect contradicts the notion of episodic uterine perfusion impairment during menstrual cramps.These insights lead to a novel hypothesis: menstrual pain arises from uterine inflammation, with ischemia possibly playing a protective role. Uterine inflammation-mediated pain worsens cross-organ sensitization and visceral-motor-mediated pain. These mechanisms are supported by our studies linking abdominal muscle activity, menstrual cramping, and increased bowel and bladder sensitivity in women with dysmenorrhea (CV #12, #21 #35). This shift in understanding points to new directions for research and treatment strategies in dysmenorrhea and related conditions, particularly among those who do not respond to conventional medications (CV # 26)

2. DEVELOPMENT OF NOVEL TESTS TO IDENTIFY PATIENTS AT RISK FOR THE TRANSITION TO CHRONIC PELVIC PAIN. Since treatments for chronic pelvic pain have low efficacy, it is critical to develop prevention methods. A first step would be to identify at-risk women for prevention trials. Although we have established that menstrual pain is a leading risk factor for the development of

chronic pelvic pain (CV #13), it is not feasible to aggressively treat every patient with menstrual pain because menstrual pain is too prevalent (>50%). Thus, we have developed strategies to identify among women with dysmenorrhea those at increased risk for the development of chronic pelvic pain.

Dr. Tu and I validated a noninvasive bladder task that can identify participants with enhanced visceral sensitivity before the development of chronic pelvic pain. A critical first step to developing prevention methods would be identifying at-risk women for prevention trials. The prior use of invasive visceral tasks (such as colorectal distension) had greatly limited research because it deterred participation and was vulnerable to fear-related factors. Therefore, Dr. Tu developed a noninvasive task in which women are asked to promote diuresis by drinking water, followed by structured measurement of their level of bladder pain as their bladder fills, with serial monitoring of this process with ultrasonography (CV #12,15, 25). We have subsequently confirmed that this bladder test, even accounting for psychological factors, can predict those at increased risk of new-onset irritable bowel syndrome (CV #37).

Our recent studies that identify at-risk patients have focused on the neural mechanisms underlying this increased risk for pain related to the heightened relationship between stimuli and unpleasantness (**Exp pub #3).** Applying a controlled magnitude stimulus and evaluating its aversiveness is the basis for the clinical neurological exam and quantitative sensory testing (QST). QST has been thought to identify "central sensitization" -the leading hypothesis for developing chronic pain and has been used in thousands of pain studies. However, the prevailing hypotheses underlying "central sensitization" (where pain-related signals are amplified, resulting in hypersensitivity) fail to explain why the perception across sensory modalities not classically associated with pain, like vision or hearing, is intensified. Also, QST has not consistently helped predict future pain, particularly in larger cohorts. Instead, we hypothesized that a supraspinal mechanism could explain pain in highly prevalent conditions without an identifiable source, like irritable bowel syndrome or fibromyalgia, and why patients have increased sensitivity to sensory modalities both above and below the brainstem termed "multimodal hypersensitivity" (MMH). To fill this gap, we evaluated a multimodal QST panel that assessed mechanical pressure sensitivity of the body and internal pelvis, noninvasive bladder pain, descending inhibitory control, spinal "wind up" pain, and visual and auditory hypersensitivity. To better understand the trajectory of chronic pain development, MMH testing was performed on a cohort of women with moderate-severe dysmenorrhea (i.e., painful menstruation), bladder pain syndrome, chronic pain, and pain-free controls—the largest sample size of its kind to date. We confirmed the presence of MMH within this cohort. MMH was associated with worse self-reported menstrual pain, genitourinary symptoms, depression, anxiety, and health. Notably, MMH predicted worse pelvic pain outcomes up to four years later, even when adjusting for baseline pelvic pain. Our study provides crucial insight for future studies and clinical evaluation: your pain intensity is less relevant than your present hypersensitivity. If corroborated, our findings suggest that increased widespread hypersensitivity (MMH) should be evaluated and diagnosed to prevent the development of chronic pain—explicitly challenging statements by the American Academy of Pediatrics and the American Psychiatric Association that suggest sensory processing disorders are not clinically relevant. e

In the companion study, we identified potential neural mechanisms underlying MMH (Exp pub #4), thus providing some strategies for future treatment. Visual sensitivity was measured using a periodic pattern-reversal stimulus during EEG. Self-reported visual unpleasantness ratings were also recorded. Bladder pain sensitivity was evaluated with an experimental bladder-filling task associated with early clinical symptoms of chronic pelvic pain. Visual stimulation-induced unpleasantness was associated with bladder pain and evoked primary visual cortex excitation; however, the relationship between unpleasantness and cortical excitation was moderated by

bladder pain. Thus, future studies aimed at reversing the progression of MSH into chronic pain should prioritize targeting cortical mechanisms responsible for maladaptive sensory input integration.
A future enhanced mindfulness strategy combining simultaneous neurofeedback from our visual task itself could be used to reprogram the neural circuitry responsible for MMH.

3. REVIEW ARTICLES ON PELVIC PAIN AND PREVENTION OF CHRONIC PAIN

Utilizing insights into nociception, I have contributed expert analyses and evidence-based guidelines for neuropathic pelvic pain and comprehensive pain management strategies (documented in CV #10, #29, #33). Our review, addressing the challenges of treatment-resistant dysmenorrhea and recommended for gynecology board preparation (Exp pub #5), has become an essential tool for those developing new menstrual pain treatments, evidenced by its broad citation. Additionally, teaching medical students the critical evaluation of pain research has led to our collaborative creation of a guide on analyzing and reviewing pain information on Wikipedia (CV #27), enhancing the scholarly approach to navigating digital information sources.

(c) Work in progress or anticipated; work that has not undergone peer review and acceptance for publication

TABLE 1	**CONTRACTILE**			**NON-CONTRACTILE**
	C-Immediate	**C-Delayed**	**C- Mixed**	**NC**
Hypercontractility [HASTE]	↑↑↑	↑	↑↑	
Pain simultaneous with contraction	+++		+	
Pain delayed after contraction onset		+++	+	
Blood volume [1/ R2*]	↑	↑	↑	↑
Perfusion [ASL]	↓	↓↓	↓↓	
Transient ischemia [EPI-BOLD]	↓	↓↓↓	↓	
Lactate [PRESS]	↑	↑↑↑	↑	↑
Visceromotor EMG activity	+	+	+	
Pain pressure thresholds	↓	↓	↓	↓↓↓
Bladder sensitization	↑	↑	↑↑	↑↑↑
Hypothesized principal mechanism	**Myometrial Contractility**	**Perfusion, Ischemia**	**Contractility and Ischemia**	**Non-uterine**
This table shows the **expected** outcomes based on our preliminary data.				

A. Identifying the causes of menstrual pain to develop new treatments: from my current R01 and past 2 R21, I have collected MRI scans, doppler measurements, biospecimens, questionnaire data, and sensory testing results on over 200 women at multiple points during the menstrual cycle before and after analgesic medication. This comprehensive repository of data is being analyzed for sub-phenotypes of menstrual pain that could be used to develop new treatments. **Table 1** shows the predicted four phenotypes based on our preliminary data with MRI modalities in [brackets]. We expect women with dysmenorrhea will be broadly divided into two classes of uterine phenotypes: contractile (**C**) and non-contractile (**N**.C.), as defined with continuous HASTE scans during menses. Regardless of contractile phenotype, a common feature of women with menstrual pain of uterine origin is the presence of an increased 1/**R2*** signal compared to controls, indicative of decreased blood volume due to vasoconstriction.

Based on our preliminary data, we predict 3 sub-phenotypes will exist within the contractile group. One-third will report pain simultaneously with the onset of a contraction (**C-Immediate** phenotype), likely reflecting an underlying mechanism associated with myometrial contraction. Another third of these women will report pain 50+ seconds after contraction onset (**C-Delayed** phenotype), potentially due to myometrial ischemia. Transient changes in uterine oxygenation visible with EPI-BOLD (section C.4) and lactate (PRESS, section C.5) during menstrual pain indicate that transient anaerobic respiration is a significant factor. The remaining third will report both immediate and delayed pain throughout testing, suggesting 'mixed' contributions from uterine contractility and ischemia (**C-mixed** phenotype). Given that contractions have been observed in women with primary or secondary dysmenorrhea, we expect that the mechanisms responsible for cramping pain are independent of anatomical factors. The completion of our analyses will provide the best noninvasive evidence that both transient episodes of myometrial activity and reduced uterine oxygenation contribute to menstrual pain. Some women, however, may have central mechanisms contributing to their pain. Intriguingly, about 20% of women with severe menstrual cramping pain do not have uterine contractions or transient ischemia (**N**.C. phenotype). Overall, NC participants have greater widespread pain sensitivity and no abdominal visceromotor activity (see section B7), suggestive of a primarily central sensory processing disorder. Some NC participants self-reported significant bowel pain that was difficult to distinguish from uterine pain. Although not all women with the N.C. phenotype have secondary dysmenorrhea, they are more likely to have a history of endometriosis. The proposed analyses will also identify which factors (hypercontractility, uterine blood volume, perfusion, ischemia,

anaerobic respiration, visceromotor activity, and drug absorption/metabolism) contribute to NSAID-resistant pain. Armed with the results from this study, we can rapidly begin critical translational studies to test specific novel treatments for dysmenorrhea, particularly in refractory cases. Our consent process asks permission to re-contact participants for future research. For example, a follow-up study with sildenafil, a PDE5 inhibitor that increases uterine blood flow[56], might be warranted in participants with impaired uterine oxygenation observed on MRI. Participants with uterine myometrial activity linked to persistent menstrual pain could be followed up with studies examining the effects of drugs that reduce contractility, such as nifedipine.

B. Developing a trial to prevent the development of chronic pelvic pain. I have recently been awarded an NIH grant to prevent the development of chronic pelvic pain. My preliminary data shows that effective NSAID therapy for dysmenorrhea may reduce future CPP risk by normalizing local nerve activation, inflammatory molecules in the uterine lining, and painful hypersensitivity in adjacent pelvic organs. To distinguish the role of different sensory mechanisms in CPP progression, our interdisciplinary team proposes to use a collection of validated sensory tasks involving mechanical, thermal, electrical stimulation and bladder filling. This proposal also pairs high-density electroencephalography with sensory testing to identify brain differences related to abnormal pain interpretation. We plan to administer these tasks along with symptom questionnaires to understand why some women with menstrual pain respond better to NSAIDs and which factors predict diminished CPP risk following an optimized program to reduce menstrual pain. In Aim 1, we will conduct a one-year, randomized controlled trial (n=300) of optimized NSAID vs. placebo treatment for period pain in moderate-plus dysmenorrhea sufferers. We will determine whether active treatment, menstrual pain relief, and lower levels of uterine inflammatory molecules predict lower nonmenstrual pelvic pain (NMPP, a marker for future CPP risk) at one-year post-treatment. Aim 2, conducted within the Aim 1 study structure, will establish which specific sensory tests predict reductions in NMPP following treatment. EEG will pinpoint precise mechanisms for differences in these sensory responses. Collectively, this study of higher-risk women is designed to characterize how menstrual pain and bladder hypersensitivity interact to promote CPP and if these factors predict who benefits from early preventative treatment. The approach is novel for using a sample of women enriched for future pain risk (women with dysmenorrhea and bladder pain) and for conducting future pain-risk prediction with recognized factors: sensory testing profiles, adjacent organ pain involvement, and observed response to a targeted treatment. Structural equation modeling will formally define how these interact to cause CPP while controlling for other key pain risk factors, such as relative local inflammation and psychosocial profiles. These study results obtained by a diverse team of interdisciplinary collaborators are expected to markedly improve our understanding of pain risk heterogeneity for chronic pain prevention

#1

Reference: **Hellman KM**, *Kuhn CS*, Tu FF, *Dillane KE, Shlobin NA*, Senapati S, *Zhou X*, Li W, Prasad PV. CINE MRI During Spontaneous Cramps in Women with Menstrual Pain. (2018) American Journal of Obstetrics & Gynecology. 218(5):506.e1-506.e8 http://doi.org/10.1016/j.ajog.2018.01.035
Major finding: This study is unique in that it is the first demonstration of what occurs in an internal organ time-locked to the report of spontaneous pain. As a new noninvasive method it holds promise as a technique for improved radiological methods for identifying the cause of pelvic and abdominal pain.
Roles of authors: KMH obtained funding, developed analytic methods, performed analyses. KCS (medical student now resident) evaluated MRI signals, and helped write first draft of manuscript with KMH. PVP (senior faculty) developed MRI methods. KMH, KED (research assistant) and WL (radiologist) performed experiments. FFT (Co-I), NAS (medical student), DXZ (resident), S.S. (Co-I) examined MRI signals.

#2

Reference: Cockrum RH, Tu FF, Kierzkowska O, Leloudas N, Pottumarthi PV, **Hellman KM.** Ultrasound and MRI-based investigation of the role of perfusion and oxygen availability in menstrual pain. 2024 Jan 29:S0002-9378(24)00059-0. https://pubmed.ncbi.nlm.nih.gov/38295969/
Major finding: This finding refutes widely believed hypotheses underlying menstrual pain. Instead, we show that uterine pain is linked to inflammation rather than ischemia. Intriguingly, prostaglandins may have a protective vasoconstrictive ischemic effect in pain-free controls.
Roles of authors: KMH obtained funding, developed analytic methods, and performed analyses. RHC, OK (medical students) evaluated MRI signals and helped write the first draft of the manuscript with KMH. PVP (senior faculty) developed MRI sequences. KMH, NL (radiology technician) performed experiments. FFT (Co-I), examined MRI images and edited manuscripts

#3

Reference: Kmiecik MJ, Tu FF, Clauw DJ, **Hellman KM.** Multimodal hypersensitivity derived from quantitative sensory testing predicts pelvic pain outcome: an observational cohort study. Pain 2023 September 1;164(9):2070-2083. https://pubmed.ncbi.nlm.nih.gov/37226937/
Major finding: This study refutes the belief that sensory tests measure specific neurological mechanisms. Rather, sensory tests reflect sensitivity to unpleasantness, which, when carefully analyzed, can predict chronic pain up to four years later.
Roles of authors: KMH designed experiments, obtained funding and designed analyses with assistance from FFT and DJC (Co-I's). Directed MJK (postdoc) to analyze data. MJK and KMH wrote first draft together that was revised by DJC and FFT.

#4

Reference: Kmiecik MJ, Tu FF, Silton RL, Dillane KE, Roth GE, Harte SE, **Hellman KM**. Cortical Mechanisms of Visual Hypersensitivity in Women at Risk for Chronic Pelvic Pain. (2021) Pain. *Epub 2021 August 27.* https://doi.org/10.1101/2020.12.03.20242032
Major finding: This study elucidates the neural mechanisms underlying sensory unpleasantness. Notably, the same cortical neural mechanisms that underlie visual sensitivity also underlie visceral sensitivity.
Roles of authors: KMH designed experiments, obtained funding and designed analyses with assistance from FFT and directed MJK (postdoc) to analyze data. KED and GER performed experiments. MJK and KMH wrote first draft together that was revised by SEH and FFT.

#5

Reference: *Oladosu FA*, Tu FF, **Hellman KM**. Nonsteroidal antiinflammatory drug resistance in dysmenorrhea: epidemiology, causes, and treatment. (2017) American Journal o Obstetrics & Gynecology 218(4):390-400 http://doi.org/10.1016/j.ajog.2017.08.108
Major finding: This review article provides evidence that NSAID resistance mechanisms are not likely due to genetic mechanisms and are often unrelated to assumed anatomical contributing factors. Instead, other factors that have been poorly studied, such as absorption and alternative molecular pathways, contribute to menstrual pain.
Roles of authors: FAO (postdoc),FFT (Co-I), and KMH reviewed prior literature and wrote the manuscript together.

EDUCATION STATEMENT

The efficacy of my mentorship is evidenced by the numerous successful research endeavors led by my trainees. A significant portion of these projects has culminated in presentations at national scientific conferences and publications in reputable journals, and others are well on their way to being published. The substantial contribution of my trainees to scholarly work is highlighted by italicized names in our publication list and asterisks next to names on the trainee roster. My commitment to fostering diversity is reflected in the increased participation of ethnic/racial minorities and women in our programs.

Recognizing the importance of funding for sustained scholarly success, I have actively supported my trainees in securing necessary resources. Notable achievements include orchestrating a successful $40,000 grant application for Dr. Adam Gafni-Kane and facilitating Carrie Kuhn's project, which resulted in a Carl Fentress Fellowship award. Furthermore, I aided a postdoctoral scientist in acquiring a competitive NIH minority training supplement, thereby enhancing the research training experience and academic portfolios of students and postdoctoral trainees through independent scholarship in crucial scientific areas.

In addition, I direct educational activities at NorthShore by organizing a 2-3 hour journal club pertinent to pain research every Wednesday. Notably, these meetings have attracted the attention of Dr. Gerald Gebhart, a renowned expert on visceral pain and a regular participant. Other University of Chicago students and resident physicians mentored in nearby laboratories often attend and present these. Dr. Katharina Laus declared during her research rotation that this journal club was one of the best educational experiences during her entire OB/GYN residency. Beyond the educational value of the science being conveyed in these meetings, I have provided a safe and comfortable venue for other University of Chicago students and resident physicians to practice speaking about their research experience. This speaking experience is vital because it is one of the few venues where they can practice scientific speaking skills essential for the modern academic career.

I have also designed a new MS4 elective, Neuroscience of Pain and Opioids, that has sustained maximal enrollment, with most of the students scoring "strongly agreed" on every evaluated course metric of quality. I developed a website for a "*flipped classroom*," resulting in significant student dialogue (more than 50% of course time). Even offline, students were active, with an average of 8 blog entries of more than 6 lines per student. Over 4 weeks, I provided a guided tour through the primary opioid and pain literature. I taught how to critically evaluate scientific papers while simultaneously delivering pointed lectures on the scientific basis for opioids in modern pain medicine. We went through 13 of the most clinically relevant scientific articles, review articles, and policy statements on the mechanisms of pain and opioid analgesia/addiction. We also discussed case studies pertinent to selected articles. At the end of the course, students acquired a basic research foundation rooted in this critical scientific literature on pain, which should allow them to accelerate their research. Student questionnaires confirmed uptake, with half of the students scoring "strongly agreed" on every evaluated course metric.

Aligned with the National Pain Strategy 2017 Guidelines, my educational philosophy encompasses the four core competencies of understanding pain's multidimensionality, pain assessment, pain management, and the clinical context. My research and educational efforts are directly tied to these competencies, ensuring that all mentored trainees and seminar participants gain comprehensive exposure to these crucial aspects of pain management.

INSTITUTIONAL CITIZENSHIP STATEMENT

Past and current:

Since 2013, my primary contribution to academic citizenship has centered on mentoring undergraduate, graduate, and postdoctoral research projects within my laboratory and across various other laboratories. I have played an active role in the Pritzker Scholarship & Discovery and Summer Research Program, serving not only as a mentor but also as a cluster group leader and a paper judge. Despite the assignment of multiple group leaders to each section, the clinical commitments of others have frequently left me as the primary facilitator of our weekly sessions. Beyond these meetings, I have provided critical support in statistical analysis and manuscript preparation for the students. My encouragement has been instrumental in guiding many towards the continuation of their projects, culminating in publication. To date, I have reviewed and offered feedback on over 200 student papers within the Summer Research Program, a testament to its vital role in the transformative journey of medical students and the extensive faculty dedication it demands for success.

Additionally, I have independently led the MS4 elective "Neuroscience of Pain and Opioids." Although Dr. Dickerson is officially noted as a co-instructor, his attendance has been limited to just two sessions over the past six years. Through this course, I aim to address the pressing need for comprehensive medical education in the context of the ongoing societal challenges related to safe pain management.

I also contribute as a co-investigator on a University of Chicago faculty member, Dr. Sandra Laveaux's, ITM-funded study on the development of fibroids, utilizing quantitative MRI methods to foresee which patients will require intervention for rapidly enlarging fibroids. My expertise in using fMRI for uterine physiology research has been beneficial to this study, resulting in a co-authored publication and another paper currently under review.

Proposed and future:

I aim to continue and expand my contributions to the University of Chicago by upholding my commitment to exceptional mentorship through ongoing and new educational initiatives in pain management. My plan includes enhancing the current didactic activities and introducing broader pain education opportunities for students, resident physicians, and fellows.

In collaboration with Dr. Laveaux, a faculty member at the University of Chicago, we are preparing a new grant proposal aimed at the screening and prevention of fibroids. Our ITM proposal is designed to identify individuals at heightened risk for uterine fibroids (U.F.s), delve into the biological underpinnings of U.F. growth—with a focus on the role of inflammation—and investigate factors that may confer protection against U.F.s. A key component of this research is a pilot study employing a mobile ultrasound clinic to reach Black women, who are disproportionately impacted by U.F.s and often face barriers to accessing gynecological care. Our study goals include: 1) Investigating the inflammatory mechanisms contributing to fibroid growth by comparing pro-inflammatory markers among patients with varying U.F. growth rates, utilizing machine learning and ultrasonography for early detection, and 2) Assessing the impact of an anti-inflammatory diet on small fibroids through randomized interventions to monitor its effect on fibroid progression. This research aims to provide early, non-invasive intervention strategies, diminish the reliance on surgical treatments, and directly benefit the study's participants. By focusing on underserved populations, our work seeks to promote health equity, potentially yielding significant healthcare savings and enhancing the quality of life for countless women. Success in this preliminary study would pave the way for a larger, NIH-funded project to

advance our understanding and management of U.F.s, particularly among minority populations in Chicago.

Furthermore, I been awarded an NIH HEAL award for a R01 grant proposal with Dr. James Griffith, a long-term collaborator and co-author from the University of Chicago. Our proposal focuses on the prevention of pelvic pain, aligning with our mutual interest in pelvic pain research. Together, we aim to conduct a clinical trial on the use of NSAIDs and model various factors to determine the most beneficial patient-specific interventions.