BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors.
Follow this format for each person. **DO NOT EXCEED FIVE PAGES.**

NAME: Sarah A Moore

eRA COMMONS USER NAME (credential, e.g., agency login): SAMOORE2204

POSITION TITLE: Associate professor, Neurology and Neurosurgery

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Auburn University (Auburn, AL)	DVM	May 2005	Veterinary Medicine
University of Missouri (Columbia, MO)	Internship	June 2006	Medicine and Surgery
North Carolina State University (Raleigh, NC)	Residency	July 2009	Neurology and Neurosurgery

A. **Personal Statement**

I am an associate professor in the department of Veterinary Clinical Sciences at OSU with a research focus in dog models spinal cord injury (SCI). As a board-certified veterinary neurologist and neurosurgeon by training, I am in the unique position to conduct translational research focusing on this area of great importance to human health. In my position, I currently manage 75-100 dogs with spontaneous SCI of varying causes each year. I view my clinical experiences and access to these veterinary patients as a tremendous opportunity to bridge the gap between laboratory models of SCI and human patients, using dogs as a spontaneous large animal model of SCI. I am experienced in behavioral assessment of dogs with SCI, including locomotor assessment and quantitative sensory testing (QST). My lab was the first to publish on QST in dogs with SCI and our techniques have been adopted successfully by many other laboratories. Our lab has also recently worked with Michele Basso to successfully complete the adaptation and validation of the Basso-Beattie-Bresnahan (BBB) locomotor scale for use in dogs with SCI. I am the current president and founding member of CANSORT-SCI, an international research consortium for clinical trials and translational neuroscience research in pet dogs with SCI.

B. Positions and Honors

Employment

September 2009-June 2015; Assistant Professor, Neurology and Neurosurgery, Department of Veterinary Clinical Sciences, The Ohio State University, Columbus, OH.

June 2015- Present; Associate Professor with tenure, Neurology and Neurosurgery, Department of Veterinary Clinical Sciences, The Ohio State University, Columbus, OH

Honors

ACVIM Neurology group, winning resident research abstract 2008
Graduated Magna Cum Laude, Auburn University 2005
Dr. Charles Knecht Neurology Award, Auburn University 2005
Auburn University College of Veterinary Medicine Dean's List, 2005
Auburn University College of Veterinary Medicine Dean's List, 2004
College of Veterinary Medicine Student Research Award, 2004
Dr. William Johnson Memorial Scholarship, Auburn University 2004

C. Contributions to Science

Establishing pet dogs as a translational clinical model of acute and chronic spinal cord injury (SCI).

Pet dogs develop spontaneously occurring SCI at rates more than double those of people **[Moore et al. J Small Anim Pract 2016, 57: 409-415]**. The major focus of my research has been on understanding the natural history of acute SCI in pet dogs caused by various etiologies- compressive/contusive, ischemic, and degenerative **[Lovett et al. Vet J 2014, 200 (2): 312-317; Moore et al. J Small Anim Pract 2016, 57: 409-415; Bartholomew et al. Vet Rec 2016, 10.1136/vr.103863]**- and on developing quantitative outcome measures of neurological recovery that can be used in the veterinary clinical setting to conduct translation research using dog models. Our laboratory has developed several novel mechanisms to quantify locomotor recovery **[Song et al. Vet J 2016, 210:61-67]** including the development and validation of a modification of the Basso-Beattie-Bresnahan locomotor rating scale for use in dogs with clinical SCI **[Song et al. J Neurosci Methods 2016, 268: 117-124]**. As a part of this work, I am also a founding member and current president of CANSORT-SCI, the canine SCI clinical trials consortium. This is a group composed of veterinary clinician-scientists, basic neuroscience researchers, physicians experienced in human SCI clinical trials, and members of industry. All have the shared goal of advancing translational research in SCI using canine clinical models via a multi-institutional, international approach. The group was conceived in November 2015 and since that time has developed an international canine SCI registry, has pending funding proposals for translational studies in dogs, and has a shared manuscript addressing translational value of dog models of SCI currently under review.

Developing a pre-clinical model of neuropathic pain using naturally occurring traumatic spinal cord injury (SCI) in pet dogs.

Neuropathic pain is an extremely common problem in people living with chronic SCI, affecting up to 90% of those patients**.** Viable animal models of neuropathic pain are currently lacking. I have explored the use of a pre-clinical model of naturally occurring SCI in pet dogs as a model of neuropathic pain. First, my laboratory developed and validated a technique for quantitative sensory testing (QST) that can be used in dogs with SCI at all phases of recovery. We published the technique and a preliminary comparison between normal dogs, those with SCI, and those with orthopedic injuries in 2013 **[Moore et al. The Veterinary Journal 2013, 197 (2):216-219]**. Next, we evaluated a large cohort of dogs with SCI during the acute phases of recovery to document how sensory thresholds (ST) change over time, and to establish intra-rater reliability of the technique. Test-retest reliability was high and ST changed over time mirroring locomotor recovery in dogs with SCI; however, longitudinal studies were needed to evaluate ST in the more chronic setting **[Song et al. Veterinary Journal 2016; 144-149]**. Currently we are conducting a longitudinal study evaluating ST in dogs with chronic SCI, as well as the effect of the neuromodulatory drug gabapentin on ST **[funded by The Gray Lady Foundation and OSU Canine research funds].** We are also exploring the ability to quantify hyperesthesia in other clinical models of neurologic disease in dogs and cats.

Defining ependymal cell responses to traumatic SCI in naturally occurring traumatic spinal cord injury (SCI) in pet dogs.

Spinal cord ependymal (SEL) cells are of recent interest to neuroscience researchers because of their ability to regenerate after CNS injury, and indications that they may function as endogenous neural stem cells to repair the injured spinal cord. In rodent models of SCI, cells of the SEL differentiate in to astrocytes, neurons, and oligodendroglia after injury and migrate to the lesion epicenter to assist with tissue repair. The ultimate goal would be to manipulate endogenous stem cell function of these cells to circumvent feasibility and ethical issues associated with the use of exogenous cell-based therapies. Pet dogs have a high incidence of naturally occurring SCI which can serve as a pre-clinical model to confirm and extend laboratory findings related to tissue regeneration and repair after SCI. We started by characterizing the tissues responses with in the SEL, and a specialized cluster of ependymal cells called the choroid plexus, after naturally occurring SCI in dogs. First, we investigated the choroid plexus as a potential effector of global inflammatory responses that may inhibit tissue repair **[Moore et al. Veterinary Immunology and Immunopathology 2012, 148 (3-4): 348-352.]** We found that the choroid plexus serves as an important source of pro-inflammatory cytokines such as TNF,

IL-1β, and heat shock proteins after SCI. Because of the proximity to the cerebrospinal fluid, these responses have the potential to exert global effects after injury and potential a diffuse pro-inflammatory state within the neuraxis. Additionally, this may serve to inhibit the neural stem cell niche within and around the spinal cord central canal and the spinal ependymal layer. Next we evaluated the morphology of the normal canine SEL and compared these findings to the SEL of dogs with SCI. We found strong similarities between the canine and human SEL, and observed increases in SEL cell numbers after injury indicating a possible proliferative response. Additionally, GFAP staining was increased in the SEL after injury, indicating that cells of the canine SEL take on a neural stem cell phenotype and may contribute to tissue repair and regeneration **[Moore et al. Veterinary Pathology 2015, 52 (6): 1108-1117.]** We are currently comparing SEL responses across several injury types, including traumatic, ischemic and neurodegenerative SCI models **[funded by The Gray Lady Foundation].** This will allow a better understanding of SEL responses to tissue injury and will highlight clinical disease models that will be likely to benefit from therapies that augment SEL proliferation and function.

D. Research Support (last 3 years)

Diagnosing and managing neuropathic pain in dogs with spinal cord injury.
Principal Investigator: Sarah Moore. Co-I Ronaldo da Costa, Laurie Cook, Austin Kerns.
Source: OSU Canine Research Funds, Period: 1/2016-12/2017. The major goal of this study is to provide a long term evaluation of the incidence of neuropathic pain in dogs with chronic spinal cord injury.

AAV9-mediated SOD1 down-regulation: Gene therapy for degenerative myelopathy.
Co-PIs: Brian Kaspar and Sarah Moore. Co-I Shibi Likhite, David Arnold.
Source: American Kennel Club Canine Health Foundation, Period 12/2015-11/2018. The major goal of this project is to evaluate the safety and feasibility of a gene therapy treatment in canine degenerative myelopathy, a naturally occurring model of familial ALS.

Diagnosing and managing neuropathic pain in dogs with spinal cord injury.
PI: Sarah Moore.
Source: The Gray Lady Foundation, Period 6/2015-5/2017. The major goal of this study is to assess inter-rater reliability of a technique for quantitative sensory testing (QST) in dogs and to assess the effects of gabapentin on QST in normal dogs.

Characterizing clinical and cellular responses in dogs with fibrocartilagenous embolism.
PI: Sarah Moore. Source: The Gray Lady Foundation, Period 5/2014-4/2016. The major goal of this project is to characterize the clinical and cellular responses within the spinal cord of dogs with FCE, a spontaneous model of ischemic myelopathy in humans.

Utility and repeatability of quantitative outcome measures to assess recovery after canine spinal cord injury.
Principle Investigator: Sarah Moore. Co-I: Ronaldo da Costa, Rachel Song, Michele Basso.
Source: Morris Animal Foundation, Period: 12/2013-11/2015. The major goal of this study is to validate several outcome measures for use in quantifying recovery after spinal cord injury in canine clinical trials.

Proteomic analysis of the cerebrospinal fluid of Great Danes with Cervical Spondylomyelopathy.
Principle investigator: Ronaldo da Costa. **Co-I Sarah Moore**, Matthew Allen, Kari Basso. Source: Gray Lady Foundation. Period: 9/2013-8/2014. The major goal of this project is to compare proteomic evaluation of CSF from dogs with Cervical Spondylomyelopathy to CSF from normal dogs.

Novel application of kinematic magnetic resonance imaging for evaluation of cervical spondylomyelopathy in dogs.
Principle Investigator: Ronaldo da Costa. Co-I Sarah Moore, Laurie Cook.
Source: Gray Lady Foundation. Period: 9/2013-8/2015. The goal of this project is to assess a novel positioning device for dynamic MRI studies in dogs with Cervical Spondylomyelopathy.

A simplified method of walking track analysis for dogs with spinal cord injury.
Principle Investigator: Sarah Moore. Co-I: Maureen Oldach.

Source: Merial. Period: 6/2013-9/2013. This grant provided salary support for a veterinary professional student to gain experience in research by working in my lab over the summer on a canine gait analysis project.

The role of hsp70, IL-1β and TNF-α responses in recovery after canine spinal cord injury. Principal Investigator: Sarah Moore. Co-I: Michael Oglesbee, Philip Popovich, Ronaldo da Costa, and Laurie Cook. Source: Clinical and Translational Sciences Award, Period: 2011-2013. The major goal of this grant is to examine the relationship between various inflammatory biomarkers in CSF and long term functional outcome in dogs with spinal cord injury.

Extracellular hsp70 response in canine Degenerative Myelopathy, a spontaneous model of familial Amyotrophic Lateral Sclerosis. Principle investigator: Sarah Moore. Co-I: Matthew Lovett, Michael Oglesbee, Joan Coates. Period: 10/2011-4/2013. The major goal of this project is to evaluate the types and sources of inflammatory biomarkers associated with canine degenerative myelopathy.

Sources of hsp70, IL-1β and TNF- in the central nervous system of dogs with acute spinal cord injury. Principle Investigator: Sarah Moore. Co-I: Michael Oglesbee. Source: The Gray Lady Foundation, Period: 2011-2013. The goal of this grant is to identify sources of various inflammatory biomarkers that have been identified in the CNS of dogs after acute spinal cord injury.