

Winnipeg Group

Sunday, Nov. 11 AM – Poster Presentations

Day / Time	Prog #	Presentation Type	Location	Authors	1st Author 1st Affiliation	Title
Sunday, Nov. 11, 9:00 AM – 10:00 AM	96.6	Poster	XX-8	<u>K.J. Eriksen</u> ; S. Zeng; G.W. Glazner; J.D. Geiger*	Pharmacology and Therapeutics, St. Boniface Research Centre	INVOLVEMENT OF THE MITOCHONDRIAL PROTEIN BNIP3 IN GLUTAMATE-INDUCED NEUROTOXICITY

Sunday, Nov. 11 PM – Poster Presentations

Day / Time	Prog #	Presentation Type	Location	Authors	1st Author 1st Affiliation	Title
Sunday, Nov. 11, 4:00 PM – 5:00 PM	212.4	Poster	XX-60	<u>M. Xue</u> ; M.R. Del Bigio*	Human Anatomy, University of Manitoba	COMPARISON OF BRAIN CELL DEATH AND INFLAMMATION IN THREE MODELS OF INTRACEREBRAL HEMORRHAGE IN ADULT RAT

Monday, Nov. 12 AM – Poster Presentations

Day / Time	Prog #	Presentation Type	Location	Authors	1st Author 1st Affiliation	Title
Monday, Nov. 12, 9:00 AM – 10:00 AM	274.10	Poster	G-30	<u>A.C.W. Weeks</u> ^{1*} ; T.L. Ivancu ⁴ ; J.C. LeBoutillier ² ; R.J. Racine ³ ; T.L. Petit ²	Dept Psychol, Nipissing Univ	UNIQUE CHANGES IN SYNAPTIC MORPHOLOGY FOLLOWING TETANIZATION UNDER PHARMACOLOGICAL BLOCKADE
Monday, Nov. 12, 8:00 AM – 9:00 AM	297.5	Poster	PP-1	<u>K.P. Carlin</u> *; L.M. Jordan	Dept Physiol, Univ Manitoba	IDENTIFICATION OF SPINAL COMMISSURAL NEURONS FOR ELECTROPHYSIOLOGICAL ASSESSMENT IN A MOUSE SPINAL CORD SLICE PREPARATION.
Monday, Nov. 12, 10:00 AM – 11:00 AM	297.11	Poster	PP-7	<u>E. Zaporozhets</u> ; B.J. Schmidt*	Medicine and Physiology, University of Manitoba	PROPRIOSPINAL CONNECTIONS MEDIATE BULBOSPINAL ACTIVATION OF LOCOMOTOR-LIKE ACTIVITY IN THE <i>IN VITRO</i> NEONATAL RAT SPINAL CORD.
Monday, Nov. 12, 9:00 AM – 10:00 AM	306.6	Poster	SS-70	<u>S. Chakrabarty</u> *; J.N. Quevedo; K. Stecina; S. Gosgnach; D.A. McCrea	Physiology, University of Manitoba	VARIATIONS IN THE DELAY BETWEEN THE ONSET OF HIP AND ANKLE FLEXORS DURING FICTIVE LOCOMOTION.

Monday, Nov. 12, 10:00 AM – 11:00 AM	318.19	Poster	WW-32	<u>K.H. Park</u> ; D.F. Clayton; T. Ivanco*	Beckman Institute	CONTEXTUAL MODULATION OF THE ZENK GENE RESPONSE TO SOUND IN THE ZEBRA FINCH CAUDOMEDIAL NEOSTRIATUM (NCM)
Monday, Nov. 12, 9:00 AM – 10:00 AM	331.14	Poster	ZZ-49	<u>F.E. Parkinson</u> *; T. Othman; C.J.D. Sinclair	Dept Pharmacol, Univ Manitoba	RELEASE OF PURINES FROM RAT PRIMARY CORTICAL NEURONS AND ASTROCYTES DURING ATP DEPLETING CONDITIONS

Monday, Nov. 12 PM – Poster Presentations

Day / Time	Prog #	Presentation Type	Location	Authors	1st Author 1st Affiliation	Title
Monday, Nov. 12, 2:00 PM – 3:00 PM	402.6	Poster	GG-6	<u>J.S. Riddell</u> ^{1*} ; K. Stecina ² ; S. Gosgnach ² ; S. Chakrabarty ² ; S.J. Shefckyk ² ; D.A. McCrea ²	IBLS, University of Glasgow	SELECTIVE SUPPRESSION OF GROUP II SENSORY INPUT TO SPINAL INTERNEURONS DURING FICTIVE LOCOMOTION IN THE CAT
Monday, Nov. 12, 3:00 PM – 4:00 PM	402.7	Poster	GG-7	<u>S. Gosgnach</u> *; K. Stecina; S. Chakrabarty; D.A. McCrea	Physiol, Univ of Manitoba	PAIRED RECORDINGS REVEAL A DIFFERENTIAL DEPRESSION OF SYNAPTIC TRANSMISSION FROM PRIMARY AFFERENTS DURING FICTIVE LOCOMOTION AND SCRATCH.
Monday, Nov. 12, 4:00 PM – 5:00 PM	402.8	Poster	GG-8	<u>D.A. McCrea</u> *; J.N. Quevedo; K. Stecina; S. Gosgnach; S. Chakrabarty	Dept Physiology, Univ Manitoba Fac Med	GLYCINERGIC INHIBITION OF LOCOMOTOR-RELATED GROUP I DISYNAPTIC EXCITATION IN CAT HINDLIMB MOTONEURONS.

Tuesday, Nov. 13 AM – Poster Presentations

Day / Time	Prog #	Presentation Type	Location	Authors	1st Author 1st Affiliation	Title
Tuesday, Nov. 13, 9:00 AM – 10:00 AM	470.18	Poster	B-7	<u>J. de Melo</u> ; V. Spencer; X. Qiu; Q.P. Zhou; J.M. Sun; J. Thliveris; J. Davie; D.D. Eisenstat*	Manitoba Institute of Cell Biology, University of Manitoba	NUCLEAR SUBLOCALIZATION OF DLX-1 AND DLX-2 IN THE EMBRYONIC VERTEBRATE STRIATUM.

Tuesday, Nov. 13 PM – Poster Presentations

Day / Time	Prog #	Presentation Type	Location	Authors	1st Author 1st Affiliation	Title
Tuesday, Nov. 13, 3:00 PM – 4:00 PM	625.3	Poster	FF-11	<u>Y. Dai</u> *; L.M. Jordan	Dept of Physiology, University of Manitoba	A MODELING STUDY OF THE RELATIONSHIP BETWEEN THE REDUCTION OF THE AFTERHYPERPOLARIZATION AND HYPERPOLARIZATION OF VOLTAGE THRESHOLD IN CAT LUMBAR MOTONEURONS DURING FICTIVE LOCOMOTION
Tuesday, Nov. 13, 4:00 PM – 5:00 PM	625.4	Poster	FF-12	<u>S. Yakovenko</u> 1*; K. Stecina2; D.A. McCrea2; A. Prochazka1	Centre for Neuroscience, Univ Alberta	SPATIOTEMPORAL ACTIVATION OF CAT LUMBOSACRAL MOTONEURONS DURING MLR-INDUCED FICTIVE LOCOMOTION IN THE CAT
Tuesday, Nov. 13, 1:00 PM – 2:00 PM	625.5	Poster	GG-1	<u>S. Yakovenko</u> 1; D.A. McCrea2; A. Prochazka1*	Neuroscience, University of Alberta	PHASE AND CYCLE DURATIONS IN MLR-INDUCED FICTIVE CAT LOCOMOTION.
Tuesday, Nov. 13, 2:00 PM – 3:00 PM	626.14	Poster	HH-10	<u>W.E. Peterson</u> 1; L.M. Jordan1*; R.M. Brownstone2	Physiology, University of Manitoba	IMMUNOLESIONING OF IDENTIFIED MOTONEURON POOLS FOLLOWING INTRAMUSCULAR INJECTION OF THE IMMUNOTOXIN, 192-IGG-SAPORIN, IN NEONATAL RATS.

Wednesday, Nov. 14 AM – Poster Presentations

Day / Time	Prog #	Presentation Type	Location	Authors	1st Author 1st Affiliation	Title
Wednesday, Nov. 14, 8:00 AM – 9:00 AM	714.17	Poster	F-55	<u>B. Fedirchuk</u> *	Dept Physiol, Univ Manitoba	THE VOLTAGE THRESHOLD OF ACTION POTENTIALS IS HYPERPOLARIZED BY MONOAMINES IN SPINAL NEURONS OF THE NEONATAL RAT.
Wednesday, Nov. 14, 10:00 AM – 11:00 AM	773.3	Poster	ZZ-6	<u>J. Kong</u> *; S. Zeng; J.D. Geiger	Division of Neurovirology & Neurodegenerative Disorders, St. Boniface Hosp Res Ctr	PARAPTOTIC NEURONAL CELL DEATH IS MEDIATED BY THE MITOCHONDRIAL PROTEIN BNIP3

Wednesday, Nov. 14 PM – Poster Presentations

Day / Time	Prog #	Presentation Type	Location	Authors	1st Author 1st Affiliation	Title
Wednesday, Nov. 14, 2:00 PM – 3:00 PM	844.10	Poster	RR-41	<u>B.J. MacNeil</u> ; D.M. Nance*	Pathology, Univ of Manitoba	THE EFFECTS OF SPINAL CORD INJURY ON CUTANEOUS INFLAMMATION

Thursday, Nov. 15 AM – Poster Presentations

Day / Time	Prog #	Presentation Type	Location	Authors	1st Author 1st Affiliation	Title
Thursday, Nov. 15, 11:00 AM – 12:00 PM	911.12	Poster	E-11	<u>J.I. Nagy</u> ^{1*} ; X. Li ¹ ; L. Luo ¹ ; B.D. Lynn ¹ ; V.A. Ionescu ¹ ; D. Patel ² ; J. Rempel ¹ ; W.A. Staines ²	Dept Physiol Fac Med, Univ Manitoba	DISTIBUTION AND DIFFERENTIAL EXPRESSION OF CONNEXINS IN NEURONS AND GLIA

Program Number: 96.6

Day / time: Sunday, Nov. 11, 9:00 AM – 10:00 AM

Presentation Type: Poster

Presentation Location: Exhibit Hall XX–8

INVOLVEMENT OF THE MITOCHONDRIAL PROTEIN BNIP3 IN GLUTAMATE-INDUCED NEUROTOXICITY

K.J. Friesen; S. Zeng; G.W. Glazner; J.D. Geiger*

Pharmacology and Therapeutics, St. Boniface Research Centre, Winnipeg, MB, Canada

Neuronal cell death has been shown to occur by apoptotic, necrotic, and alternative cell death mechanisms. Recently, it was reported that BNIP3, a member of the Bcl-2 protein family without a functional BH3 domain, induces cell death in a necrotic-like manner and that this gene-regulated cell death pathway involves opening of the mitochondrial permeability transition pore without caspase activation and cytochrome c release. However, to date nothing is known about the role of BNIP3 in neuronal cell death. Here, we tested the hypothesis that BNIP3, when expressed in neurons, is capable of inducing necrotic (possibly paraptotic) cell death. Glutamate was applied to cultures of rat hippocampal neurons, expression of BNIP3 was detected using immunohistochemical techniques, and cell death was measured morphologically and by using trypan blue exclusion. BNIP3 expression following application of glutamate was both time- and glutamate concentration-dependent. Cell death was also found to be time- and concentration-dependent; increased numbers of trypan blue positive cells paralleled the increased number of cells immunopositive for BNIP3. Expression of BNIP3 was seen in those cells with morphological features associated with necrotic rather than apoptotic cell death. BNIP3 is involved in excitotoxic cell death and may be a mediator of paraptotic neuronal cell death.

Supported by: grants from CIHR and NS39184. KJF was supported by an NSERC fellowship

Program Number: 274.10

Day / time: Monday, Nov. 12, 9:00 AM – 10:00 AM

Presentation Type: Poster

Presentation Location: Exhibit Hall G–30

UNIQUE CHANGES IN SYNAPTIC MORPHOLOGY FOLLOWING TETANIZATION UNDER PHARMACOLOGICAL BLOCKADE

A.C.W. Weeks¹; T.L. Ivanco⁴; J.C. LeBoutillier²; R.J. Racine³; T.L. Petit²

1. Dept Psychol, Nipissing Univ, North Bay, ON, Canada

2. Dept Psychol, Prog in Neurosci, Univ of Toronto, Toronto, ON, Canada

3. Dept Psychol, McMaster Univ, Hamilton, ON, Canada

4. Univ of Manitoba, Winnipeg, MB, Canada

Long-term potentiation (LTP) in the hippocampus has been associated with changes in synaptic morphology. Whether these changes are LTP dependent or simply a result of electrophysiological stimulation has not yet been fully determined. The current research involved an examination of synaptic morphology in the rat hippocampus 24 hours after tetanization normally sufficient to induce LTP. Ketamine, a competitive NMDA antagonist, was injected before the tetanization to block the formation of LTP. Synapses were examined in the middle third of the molecular layer (MML) of the rat dentate gyrus following repeated high frequency tetanization of the perforant path. Ultrastructural quantification included the total number of synapses per neuron, synaptic curvature, the presence of synaptic perforations, and the maximum length of the synapses. Most of the changes observed previously in curvature and synaptic size (Weeks et al., 1999) following the induction of LTP do not persist under ketamine blockade. Ketamine was associated, however, with several novel non-LTP specific structural changes including an increase in synaptic length and an increase in the proportion of irregular shaped synapses. These results indicate that the use of ketamine as an NMDA antagonist may cause extraneous and/or compensatory changes in synaptic structure.

Supported by: Grants from the Natural Sciences and Engineering Research Council of Canada to T.L.P. and R.J.R.

Program Number: 212.4

Day / time: Sunday, Nov. 11, 4:00 PM – 5:00 PM

Presentation Type: Poster

Presentation Location: Exhibit Hall XX–60

COMPARISON OF BRAIN CELL DEATH AND INFLAMMATION IN THREE MODELS OF INTRACEREBRAL HEMORRHAGE IN ADULT RAT

M. Xue; M.R. Del Bigio*

1. Human Anatomy, 2. Pathology, University of Manitoba, Winnipeg, MB, Canada

Intracerebral hemorrhage (ICH) is associated with stroke and head trauma. Different experimental models are used, but it is unclear to what extent the tissue responses are comparable. The purpose of this study was to compare the responses to brain hemorrhage created by injections of autologous whole blood, collagenase digestion of blood vessels, and avulsion of cerebral blood vessels. Seventy adult rats were subjected to intracerebral hemorrhage. Rats were perfused fixed with 4% paraformaldehyde 1 hour to 28 days later. H&E, immunohistochemical, and TUNEL staining were used to allow quantification of pyknotic/eosinophilic neurons, neutrophils, CD8a immunoreactive lymphocytes, RCA-1 positive microglia/macrophages, and TUNEL positive cells at the edge of the hemorrhagic lesions. In all models eosinophilic neurons and TUNEL positive cells peaked at 2–3 days but were evident up to 28 days. In all models, neutrophils appeared briefly from 1–3 days but were substantially lower in the vascular avulsion model, perhaps owing to the devitalized nature of the tissue. Influx of CD8a immunoreactive leukocytes was later peaking at 3–7 days. The microglial/macrophage reaction occurred more gradually and peaked at 7 days in the avulsion and collagenase models although it peaked at 2–3 days in the autologous injection model. These results suggest that different models of intracerebral hemorrhage are associated with similar temporal patterns of cell death and inflammation. However, the relative magnitude of these changes varies somewhat.

Supported by: Heart and Stroke Foundation and Manitoba Health Research Council

Program Number: 297.5

Day / time: Monday, Nov. 12, 8:00 AM – 9:00 AM

Presentation Type: Poster

Presentation Location: Exhibit Hall PP–1

IDENTIFICATION OF SPINAL COMMISSURAL NEURONS FOR ELECTROPHYSIOLOGICAL ASSESSMENT IN A MOUSE SPINAL CORD SLICE PREPARATION.

K.P. Carlin*; L.M. Jordan

Dept Physiol, Univ Manitoba, Winnipeg, MB, Canada

Commissural cells (CC) extend an axon across the midline of the spinal cord and are thought to be part of the central pattern generator subserving interlimb coordination. In mammals, various CC are known to be rhythmically active during fictive locomotion including ventral spinocerebellar tract cells (Arshavsky et al 1978), lamina VII cholinergic cells (Huang et al 2000) and possibly cells in group II afferent pathways (Shefchik et al 1990). Presently, little is known about the intrinsic properties or the influence of various neurotransmitters on mammalian spinal CC. To identify ascending CC in a neonatal mouse spinal cord slice preparation for subsequent patch-clamp analysis, a rapidly transported fluorescent dextran amine was applied to one half of a rostrally hemisectioned in-vitro spinal cord (Eide et al 1999). After allowing time for transport, the lumbar cord was sliced and labeled cells located contralateral to the dye application were visually patch clamped. This method primarily labeled cell bodies in laminae VII, VIII, X of the slice. Labeled axons could be seen crossing in the ventral commissure whereupon they made a near 90 degree change in direction and traveled toward the medial aspect of the ventral funiculus. Electrophysiologically, both high and low voltage-activated calcium currents have been recorded in CC located in lamina VII. These cells do not appear to express dendritic voltage-activated calcium channels (Carlin et al 2000) as slow voltage ramps have failed to elicit hysteretic currents. These results demonstrate a viable method for targeting spinal commissural neurons for electrophysiological study.

Supported by: CIHR, NIH, MNI, MPI and WCB of Man.

Program Number: 297.11

Day / time: Monday, Nov. 12, 10:00 AM – 11:00 AM

Presentation Type: Poster

Presentation Location: Exhibit Hall PP-7

PROPRIOSPINAL CONNECTIONS MEDIATE BULBOSPINAL ACTIVATION OF LOCOMOTOR-LIKE ACTIVITY IN THE *IN VITRO* NEONATAL RAT SPINAL CORD.

E. Zaporozhets; B.J. Schmidt*

Medicine and Physiology, University of Manitoba, Winnipeg, MB, Canada

One of the major goals of spinal cord research is to determine how to re-establish descending motor control, such as the locomotor command signal, after spinal cord injury. This requires characterization of the involved bulbospinal systems. In this series, we examine whether brainstem activation of locomotor-like activity in the neonatal rat lumbar spinal cord is transmitted, at least in part, via descending propriospinal connections. Bilaterally intact brainstem-spinal cord preparations were isolated from rats (postnatal days 1 to 6) and maintained *in vitro*. The bath was partitioned at the cervico-medullary junction and at the mid-thoracic level, thereby producing three separate compartments. A combination of 5-HT, NMDA, and bicuculline was applied to the brainstem compartment to induce rhythmic motor activity in the lumbar cord, as monitored via suction electrodes applied to multiple lumbar ventral roots. Suppression of synaptic transmission in the middle (cervico-thoracic) compartment, using calcium-free bath solution, abolished lumbar rhythmic activity; low levels of tonic activity sometimes persisted. Enhancement of neuronal excitability in the cervico-thoracic compartment, by reducing the magnesium concentration, facilitated brainstem activation of lumbar rhythmic activity. These results suggest that propriospinal relays in the cervico-thoracic region are essential for transmission of the descending locomotor command. Whether propriospinal connections alone (i.e. in the absence of direct bulbospinal projections) are also sufficient is an important question, but remains to be explored.

Supported by: Canadian Institutes of Health Research

Program Number: 318.19

Day / time: Monday, Nov. 12, 10:00 AM – 11:00 AM

Presentation Type: Poster

Presentation Location: Exhibit Hall WW-32

CONTEXTUAL MODULATION OF THE ZENK GENE RESPONSE TO SOUND IN THE ZEBRA FINCH CAUDOMEDIAL NEOSTRIATUM (NCM)

K.H. Park; D.F. Clayton; T. Ivanco*

Beckman Institute, Urbana, IL, USA

The sound of birdsong activates ZENK gene expression in the avian NCM. Growing evidence suggests this response is modulated by exogenous behavioral factors such as social context and attention/arousal. In light of this, we set out to compare the patterns of ZENK induction in freely behaving versus restrained adult male zebra finches (n=48). Using *in situ* hybridization, relative responses were measured to four types of stimuli: a conspecific song (CON), flat-amplitude white noise (WN), a synthetic stimulus in which the amplitude of WN was modulated by the envelope of the CON stimulus (song-enveloped noise, SEN), and silence. Immediately prior to and during testing, each bird was subjected to one of three different conditions: 1) overnight isolation, freely moving in cage; 2) 1 hour isolation, freely moving in cage; 3) 1 hour isolation, restrained (to replicate the conditions of prior electrophysiological analyses). All birds were tested in the dark to minimize influence of uncontrolled visual factors. Data were quantified by cell counting and analyzed by two-way ANOVA. The results reveal a significant effect of restraint on the selectivity of the gene response: WN and SEN were as effective as CON in restrained birds, but were ineffective in freely moving birds after overnight isolation. To determine whether restrained birds are capable of discriminating CON and SEN, they were habituated to one and then tested for a ZENK response to the other. The stimuli were discriminated by this assay only when SEN preceded CON. These results have implications for the mechanisms of complex auditory processing in the avian NCM.

Supported by: NIH ROI-MH52086

Program Number: 306.6

Day / time: Monday, Nov. 12, 9:00 AM – 10:00 AM

Presentation Type: Poster

Presentation Location: Exhibit Hall SS-70

VARIATIONS IN THE DELAY BETWEEN THE ONSET OF HIP AND ANKLE FLEXORS DURING FICTIVE LOCOMOTION.

S. Chakrabarty*; J.N. Quevedo; K. Stecina; S. Gosgnach; D.A. McCrea

Physiology, University of Manitoba, Winnipeg, MB, Canada

Hip flexor (SART) activity precedes activity in ankle flexors (TA) during fictive locomotion by an average ~20ms (Quevedo et al, SFN 2000). Is this sequential activation a reflection of the intrinsic organization of the CPG or does it change as locomotion changes? Detailed analysis of the ENGs reveals variability in the onset delay of TA activity. In locomotor bouts with a frequency of 1 – 1.5Hz (normal) the start of activity in SART almost always preceded that in TA. However, in a few steps, the onset of activity in SART followed that in TA. This change in the sequence of flexor activity onset was more prevalent when the locomotion was irregular, or the frequency not constant. The variation in the onset delays is not associated with the frequency of locomotion, nor the burst duration of TA or SART activity. The onset delays for the normal locomotor bouts ranged from 9 to 31ms. The burst durations of TA and SART were similar in most of the bouts. The variability in the onset delay might be associated with varying levels of ENG or motoneuron activity. Paired intracellular motoneuron recordings will be done to determine whether variation in the onset delay between SART and TA activity reflect changes in the onset of motoneuron depolarization or changes in the approach to threshold. The variation in the onset of flexor activity suggests the existence of some degree of independence between CPG circuitry controlling hip and ankle flexors.

Supported by: CIHR

Program Number: 331.14

Day / time: Monday, Nov. 12, 9:00 AM – 10:00 AM

Presentation Type: Poster

Presentation Location: Exhibit Hall ZZ-49

RELEASE OF PURINES FROM RAT PRIMARY CORTICAL NEURONS AND ASTROCYTES DURING ATP DEPLETING CONDITIONS

F.E. Parkinson*; T. Othman; C.J.D. Sinclair

Dept Pharmacol, Univ Manitoba, Winnipeg, MB, Canada

In brain, the levels of adenosine increase up to 100-fold during cerebral ischemia; however, the role of specific cell types, enzymatic pathways and membrane transport processes in regulating intra- and extracellular concentrations of adenosine is poorly understood. We isolated and cultured rat cortical neurons and astrocytes. After 6 – 13 (neurons) or 12 – 21 (astrocytes) days in culture, cells were incubated with [3H]adenine for 30 min to radiolabel intracellular ATP. Cells were then treated with glucose-free buffer, hypoxia/hypoglycemia, 500 uM iodoacetate or 100 uM NaCN for 1 hour to stimulate metabolism of ATP and release of purines. The nucleoside transport inhibitor dipyridamole (10 uM), the adenosine kinase inhibitor iodotubercidin (1 uM), the adenosine deaminase inhibitor EHNA (1 uM), and the purine nucleoside phosphorylase inhibitor BCX-34 (10 uM) were used to examine the contribution of specific enzymes and transporters in the metabolism and release of purines. Our results indicate that (1) neurons were more sensitive to hypoxia/hypoglycemia and to iodoacetate than astrocytes, (2) astrocytes released more AMP and adenosine than neurons, (3) dipyridamole blocked adenosine release from neurons but not from astrocytes, and (4) EHNA had a greater effect on adenosine release than iodotubercidin. These data suggest that, in our experimental conditions, adenosine was formed by an intracellular pathway in neurons and then released via a nucleoside transporter. In contrast, AMP release and extracellular metabolism to adenosine appeared to predominate in astrocytes.

Supported by: CIHR, MHRC, NSERC

Program Number: 402.6

Day / time: Monday, Nov. 12, 2:00 PM – 3:00 PM

Presentation Type: Poster

Presentation Location: Exhibit Hall GG-6

SELECTIVE SUPPRESSION OF GROUP II SENSORY INPUT TO SPINAL INTERNEURONS DURING FICTIVE LOCOMOTION IN THE CAT

J.S. Riddell¹*; K. Stecina²; S. Gosgnach²; S. Chakrabarty²; S.J. Shefcheky²; D.A. McCrea²

¹ I.BLS, University of Glasgow, Glasgow, United Kingdom

² Department of Physiology, University of Manitoba, Winnipeg, MB, Canada

At rest activation of muscle spindle group II afferents evokes flexion reflexes. However, during fictive locomotion group II reflexes are reorganized into those that promote either flexion or extension. Interneurons interposed in group II reflex pathways are located throughout the lumbosacral enlargement of the cat spinal cord. One recently described population in the lower-lumbar segments are strongly excited by group II muscle afferents and co-excited by cutaneous afferents. About half of these neurons have an ascending projection to the midlumbar segments (Riddell & Hadian, 2000, *J.Physiol.* 522, 109–123). We have now compared the effectiveness with which group II afferents excite these lower-lumbar interneurons in adult decerebrate cats at rest and during MLR evoked fictive locomotion. During locomotion group II excitation of these interneurons is completely suppressed while at the same time, cutaneous input to them is largely unaffected. Previously, we found evidence of a preferential, presynaptic, depression of monosynaptic group II field potentials during fictive locomotion (Perreault et al. 1999, *J. Physiol.* 521, 691–703). Together, these observations suggest that the locomotor CPG can reduce transmission from selected primary afferents to specific group II interneuron populations during locomotion. Selective presynaptic inhibition of primary afferents may be an important mechanism for the reorganization of group II reflex pathways during locomotion. Supported by: CIHR and NIH

Program Number: 402.8

Day / time: Monday, Nov. 12, 4:00 PM – 5:00 PM

Presentation Type: Poster

Presentation Location: Exhibit Hall GG-8

GLYCINERGIC INHIBITION OF LOCOMOTOR-RELATED GROUP I DISYNAPTIC EXCITATION IN CAT HINDLIMB MOTONEURONS.

D.A. McCrea*; J.N. Quevedo; K. Stecina; S. Gosgnach; S. Chakrabarty

Dept Physiology, Univ Manitoba Fac Med, Winnipeg, MB, Canada

During locomotion sensory feedback from group I afferents evokes profoundly different reflexes than during the quiescent state. Extensor muscle spindle (Ia) and tendon organ (Ib) afferents evoke inhibition of hindlimb motoneurons at rest (non-reciprocal inhibition), while during fictive locomotion there is a reflex reversal and a disynaptic excitation emerges. The present studies concern the mechanism responsible for suppression of group I disynaptic excitation in the absence of locomotion. Results were obtained using intracellular recordings from hindlimb motoneurons in decerebrate adult cats. PSPs produced by stimulation of group I afferents were observed before and after intravenous administration of strychnine 50ug/kg. As expected, strychnine administration suppressed both reciprocal and non-reciprocal group I inhibition. The noteworthy observation, however, was the appearance of distinct, disynaptic group I EPSPs in both flexor and extensor motoneurons following strychnine administration. Locomotor-dependent group I excitation is believed to be mediated by a class of previously unknown and now partially identified spinal interneurons with monosynaptic input from group I muscle afferents projecting directly to motoneurons. If the EPSPs found after strychnine administration are mediated by the same interneurons, then our results suggest that there is a tonic, glycinergic inhibition of excitatory group I interneurons during rest. The ability to evoke disynaptic excitation in the absence of locomotion should greatly simplify further studies on the organization and characteristics of group I excitatory interneurons. Supported by: CIHR

Program Number: 402.7

Day / time: Monday, Nov. 12, 3:00 PM – 4:00 PM

Presentation Type: Poster

Presentation Location: Exhibit Hall GG-7

PAIRED RECORDINGS REVEAL A DIFFERENTIAL DEPRESSION OF SYNAPTIC TRANSMISSION FROM PRIMARY AFFERENTS DURING FICTIVE LOCOMOTION AND SCRATCH.

S. Gosgnach*; K. Stecina; S. Chakrabarty; D.A. McCrea

Physiol, Univ of Manitoba, Winnipeg, MB, Canada

The reduction in monosynaptic EPSPs in motoneurons and extracellular field potentials reveals a depression of synaptic transmission from hindlimb afferents during both fictive locomotion and scratch. This depression is likely presynaptic, occurs tonically during, and persists for minutes following cessation of rhythmic motor activity. We now report on the depression of monosynaptic transmission from peripheral nerves recorded in two locations during brainstem-evoked fictive locomotion or scratch (topical curare application on the cervical cord) in decerebrate adult cats. Simultaneous measurements were made of either 2 extracellular field potentials recorded in different spinal locations or of monosynaptic EPSPs recorded in 2 motoneurons. Field potential and EPSP amplitudes were compared for each pair of recordings (location-dependent depression) and also during fictive locomotion and fictive scratch (behavior-dependent depression). On average, there is a depression of synaptic transmission from group I muscle afferents of about 30% during locomotion and 35% during scratch. The paired recordings show that the depression of synaptic transmission from a particular nerve is not uniform. For example, in some cases monosynaptic Ia EPSPs were reduced substantially in one motoneuron and unchanged in another. Similarly, the extent of field potential reduction was often dissimilar for fields recorded in the same laminae and separated by as little as 4 mm. We suggest that the presynaptic inhibition produced by the CPG is evoked focally and to different extents in circumscribed spinal regions. Supported by: CIHR

Program Number: 470.18

Day / time: Tuesday, Nov. 13, 9:00 AM – 10:00 AM

Presentation Type: Poster

Presentation Location: Exhibit Hall B-7

NUCLEAR SUBLOCALIZATION OF DLX-1 AND DLX-2 IN THE EMBRYONIC VERTEBRATE STRIATUM.

J. de Melo; V. Spencer; X. Qiu; Q.P. Zhou; J.M. Sun; J. Thliveris; J. Davie; D.D. Eisenstat*

Manitoba Institute of Cell Biology, University of Manitoba, Winnipeg, MB, Canada

Multiple transcription factors are expressed in the developing striatum, including two Distal-less (Dlx) homeobox genes, Dlx-1 and Dlx-2. In the ventricular and subventricular zones, many embryonic striatal neurons co-express Dlx-1 and Dlx-2. Mice with null mutations of Dlx-1, Dlx-2 and both Dlx-1 and Dlx-2 have been generated. However, little is known about the direct gene targets of the DLX homeoproteins themselves. Cisplatin (CDDP) is a chemotherapeutic agent whose reactive intermediates form DNA crosslinks (XL). Unlike formaldehyde, CDDP enhances protein-DNA complex retrieval, especially for nuclear matrix (NM) bound proteins. Embryonic (E13.5) striatum was XL with either 1% or 4% paraformaldehyde (PFA) or CDDP. 1% PFA yielded more DLX-1 and DLX-2 than 4% PFA. This XL was time-dependent since DLX-1 but not DLX-2 was detected at 1 hour. CDDP XL established that both DLX-1&2 were bound to the NM in situ. Sequential nuclear, non-NM and NM preparations of E13.5 striatum demonstrated that DLX-1 is localized to both the NM and non-NM, whereas DLX-2 is localized only to the NM. Green fluorescence protein (GFP) constructs expressing DLX-1 or DLX-2 were transfected into NIH3T3 fibroblasts and compared to DLX (+) E13.5 striatal neurons. Both gave a speckled nuclear immunofluorescence pattern. Hence, DLX-1 and DLX-2 are expressed in different subnuclear compartments. Identification of specific DLX binding partners within these compartments will further elucidate the mechanisms whereby these closely related transcription factors play different yet overlapping roles in striatal development. Supported by: CancerCare Manitoba

Program Number:

625.3

Day / time: Tuesday, Nov. 13, 3:00 PM – 4:00 PM**Presentation Type:** Poster**Presentation Location:** Exhibit Hall FF-11**A MODELING STUDY OF THE RELATIONSHIP BETWEEN THE REDUCTION OF THE AFTERHYPERPOLARIZATION AND HYPERPOLARIZATION OF VOLTAGE THRESHOLD IN CAT LUMBAR MOTONEURONS DURING FICTIVE LOCOMOTION**Y. Dai*; L.M. Jordan*Dept of Physiology, University of Manitoba, Winnipeg, MB, Canada*

Cat lumbar motoneurons displayed a reduction of the afterhyperpolarization (AHP) and a hyperpolarization of voltage threshold (V_{th}) during fictive locomotion. It was suggested that the reduced AHP could be due to a reduced AHP conductance (g_{K_AHP}). Modulation of sodium conductance (g_{Na}) was shown in a modeling study to be the most likely basis for the hyperpolarization of V_{th} . Here, we investigate the question of whether there is interplay between these two conductances during fictive locomotion and what could be their mixed effect on AHP.

A single cell model with five-compartment was used in this study. The model included 8 active conductances in the somatic compartment, 4 in the proximal dendrite, 2 each in the axon and initial segment, and none in the distal dendrite. The V_{th} was used as reference to measure the AHP amplitude.

Simulation results show that 1) modulation of AHP by g_{K_AHP} and V_{th} by g_{Na} was produced by two mechanisms which were not related to each other; however, increasing g_{Na} and/or reducing g_{K_AHP} could result in AHP reduction; 2) reduction of the AHP is dependent on the method of analysis. A constant current injection produced a larger AHP reduction than a reduced current injection, which was used to maintain a constant firing frequency, when the AHP was reduced by increasing g_{Na} or reducing g_{K_AHP} ; 3) With constant current injection more than 40% reduction of the AHP could result from V_{th} hyperpolarization when V_{th} was hyperpolarized with an amount comparable to the experimental observation.

Supported by: Manitoba Neurotrauma Initiative and the Canadian Institute of Health Research

Program Number: 625.4**Day / time:** Tuesday, Nov. 13, 4:00 PM – 5:00 PM**Presentation Type:** Poster**Presentation Location:** Exhibit Hall FF-12**SPATIOTEMPORAL ACTIVATION OF CAT LUMBOSACRAL MOTONEURONS DURING MLR-INDUCED FICTIVE LOCOMOTION IN THE CAT**S. Yakovenko 1*; K. Stecina 2; D.A. McCrea 2; A. Prochazka 1*1. Centre for Neuroscience, Univ Alberta, Edmonton, AB, Canada**2. Physiology, University of Manitoba, Winnipeg, MB, Canada*

In a previous study we modeled the migration of motoneuron (MN) activity in the spinal cord during cat locomotion. The spatio-temporal activity was represented as a 3-D movie using EMG patterns of hindlimb muscles to scale the number of "lit-up" MNs in a digitized anatomical model of the spinal cord (Yakovenko et al., 2000, J. Physiol. 525:42P). We have now generated a similar movie for fictive locomotion. Activity profiles of 22 MN pools were constructed from neurograms recorded from muscle nerves during stable episodes of fictive locomotion elicited in 13 decerebrated, curarized cats by MLR stimulation. The amplitude of a given neurogram profile determined the number of active or "lit-up" MNs in a MN pool at a given moment. By displaying active MNs in 100 sequential frames and representing one complete fictive locomotor cycle (700 ms), a movie of the ensemble MN activity was generated. As in the intact cat step cycle, there was a clear rostro-caudal oscillation of MN activity correlated with extensor and flexor phases of the cycle, but the duration of the flexor phase was proportionally much greater and the migration of MN activity from flexion to extension was significantly different because of a transitional zone of activity corresponding to an early onset of knee extensor nerve activity. Furthermore, there was reduced activity of MN pools of the bi-functional muscles BFP, Smp, ST and FDL, which retract the limb at swing onset in the normal step cycle. The absence of proprioceptive feedback in fictive locomotion may account for some of these differences.

Supported by: CIHR

Program Number: 626.14**Day / time:** Tuesday, Nov. 13, 2:00 PM – 3:00 PM**Presentation Type:** Poster**Presentation Location:** Exhibit Hall HH-10**IMMUNOLESIONING OF IDENTIFIED MOTONEURON POOLS FOLLOWING INTRAMUSCULAR INJECTION OF THE IMMUNOTOXIN, 192-IGG-SAPORIN, IN NEONATAL RATS.**W.E. Peterson 1; L.M. Jordan 1*; R.M. Brownstone 2*1. Physiology, University of Manitoba, Winnipeg, MB, Canada**2. Surgery, Dalhousie University, Halifax, Canada, NS, Canada*

Studies have shown that the immunotoxin, 192-IgG-Saporin, can selectively lesion p75-positive cholinergic neurons of the basal forebrain in adult rats. Here we demonstrate the novel use of 192-IgG-Saporin to induce MN loss following intramuscular (I.M.) injection in neonatal rats.

Two days following I.M. injection of 192-IgG-Cy3, neonatal rats (but not adult rats or neonatal mice) had Cy3-labelled MNs. This suggests that the 192-IgG antibody and its conjugates can be internalised by receptor-mediated endocytosis and retrogradely transported to spinal motor neurons.

To induce MN loss, the left hind limb musculature of anaesthetised Sprague-Dawley rats were exposed, and several muscles injected with 0.5 μ g of 192-IgG-Saporin

(Chemicon). Right hind-limb muscles were injected with DiI. Animals were sacrificed 25 days later. Ten μ m coronal sections were obtained using a cryostat and Nissl stained.

The neonatal rats showed signs of a locomotor deficit 2.5 weeks post injection with 192-IgG-Saporin, which increased slightly in severity over the next week and a half. Nissl stained coronal sections of the lumbar region showed an obvious MN deficit on the 192-IgG-Saporin treated side compared to control side. The injected muscles were also severely atrophic, a not unexpected finding given that they too express p75 receptors. We conclude that 192-IgG-Saporin can be used to lesion MN pools when IM injected in neonatal rats. This model may prove useful for testing cell replacement therapies for the treatment of MN diseases like amyotrophic lateral sclerosis (ALS).

Supported by: CIHR

Program Number: 625.5**Day / time:** Tuesday, Nov. 13, 1:00 PM – 2:00 PM**Presentation Type:** Poster**Presentation Location:** Exhibit Hall GG-1**PHASE AND CYCLE DURATIONS IN MLR-INDUCED FICTIVE CAT LOCOMOTION.**S. Yakovenko 1; D.A. McCrea 2; A. Prochazka 1**1. Neuroscience, University of Alberta, Edmonton, AB, Canada**2. Physiology, University of Manitoba, Winnipeg, MB, Canada*

Previous studies have shown that the durations of extensor phases (T_e) of the intact cat step cycle vary more with cycle period (T_c) than those of flexor phases (T_f). We now report that this is reversed in MLR-induced fictive locomotion. Neurograms (ENGs) were recorded from 15 nerves of hindlimb muscles during stable episodic fictive motor activity elicited by electrical stimulation of the mesencephalic locomotor region in 10 curarized, decerebrated cats. Cyclical ENG bursts were analyzed using a thresholding method to identify onsets and cessations of activity. Regression analysis of ENGs of individual nerves showed that T_e and T_f increased linearly with T_c , but unlike in the intact cat, the T_f line of best fit had a steeper slope (Fig.1: T_f slope 0.75, T_e slope 0.16). ENGs of the bi-functional muscles FDL and PBSt were mainly coactive with those of pure extensors such as LGS, which differs markedly from intact gait, where FDL and PBSt have prominent flexor EMG bursts. We analyzed FDL and PBSt ENGs separately and found they had slopes similar to pure extensors (Fig.1: T_b slope 0.06). Overall the results are consistent with the idea that during unperturbed locomotion movement-related afferent feedback, which is absent in this preparation, plays an important role in locomotor pattern generation.

Supported by: CIHR

Program Number:

714.17

Day / time: Wednesday, Nov. 14, 8:00 AM – 9:00 AM

Presentation Type: Poster

Presentation Location: Exhibit Hall F-55

THE VOLTAGE THRESHOLD OF ACTION POTENTIALS IS HYPERPOLARIZED BY MONOAMINES IN SPINAL NEURONS OF THE NEONATAL RAT.

B. Fedirchuk*

Dept Physiol, Univ Manitoba, Winnipeg, MB, Canada

During fictive locomotion in the cat, motoneurons become more excitable because of a lowering of the voltage threshold for action potentials (V_{th}) (i.e. V_{th} becomes more negative or 'hyperpolarized'; Krawitz et al. 2001, *J Physiol* 532, 271–281). The mechanism(s) underlying this rapid, state-dependent facilitation of neuronal firing is not known. To test the possibility that monoamines (MAs) may hyperpolarize V_{th} , experiments were conducted on the isolated spinal cords of neonatal (P0–P5) rats. Whole-cell patch clamp recordings were obtained from ventral horn neurons. The V_{th} was assessed during current clamp recordings by firing the cell with depolarizing current injections, or in voltage clamp by applying depolarizing voltage steps and determining the threshold voltage step required to elicit a fast inward sodium current. V_{th} values were determined prior to and during bath application of MAs, and all recordings were made in the absence of locomotor activity. Bath applied 5-Ht ($12\mu\text{M}$) caused a hyperpolarization of V_{th} (2 to 8 mV) in the majority of cells tested. The hyperpolarization was maximal within minutes, recovered to control levels with washout of the 5-Ht, and could be repeated by re-application of 5-Ht following washout. Bath applied NA ($12\mu\text{M}$) and ACh ($120\mu\text{M}$) were also able to hyperpolarize V_{th} , to an extent comparable to that for 5-Ht. These results show a novel effect exerted by MAs: their ability to regulate neuronal V_{th} . Exogenously applied MAs can hyperpolarize V_{th} in the absence of locomotion to an extent comparable to that seen during fictive locomotion in the cat. Endogenously released MAs may underlie the facilitated neuronal firing seen during motor activities.

Program Number: 844.10

Day / time: Wednesday, Nov. 14, 2:00 PM – 3:00 PM

Presentation Type: Poster

Presentation Location: Exhibit Hall RR-41

THE EFFECTS OF SPINAL CORD INJURY ON CUTANEOUS INFLAMMATION

B.J. MacNeil; D.M. Nance*

Pathology, Univ of Manitoba, Winnipeg, MB, Canada

These studies tested if disruption of normal innervation by spinal cord injury (SCI) would alter cutaneous inflammatory responses. Sprague Dawley rats received a SCI at the T1–T2 level and were maintained for 4 weeks after which turpentine was injected intradermally (id) on the dorsal trunk below the neurological level of the SCI. Control animals displayed a robust inflammatory response by 2 hours post-injection. In contrast, SCI animals showed minimal inflammation until 12–24 hours after injection. In a second experiment animals received id turpentine injections above and below the level of the SCI and the in vivo cytokine mRNA response assessed 2 hours later. In control animals, within group comparisons indicated that interleukin (IL)-1 beta, tumor necrosis factor-alpha (TNF), IL-6 and monocyte chemoattractant protein mRNA production was not different between skin sites from above and below the sham SCI level. However, SCI rats had a significant decrease in all cytokine mRNA levels below the SCI. Preliminary data indicate a similar SCI effect following id injection of killed *Staphylococcus Aureus* bacteria measured 6 hours after injection in that the level of TNF-alpha is reduced in SCI rats below the level of the SCI. These data indicate that SCI animals have a reduced inflammatory response below, but not above, the level of a SCI. The distinct anatomical pattern of cytokine responses which correspond to the SCI suggests that neural factors rather than systemic or circulating factors account for these changes. These studies reveal that humans with a SCI may be more susceptible to cutaneous pathogens over skin sites which are effected by the SCI.

Supported by: Paralyzed Veterans of America

Program Number: 773.3

Day / time: Wednesday, Nov. 14, 10:00 AM – 11:00 AM

Presentation Type: Poster

Presentation Location: Exhibit Hall ZZ-6

PARAPTOTIC NEURONAL CELL DEATH IS MEDIATED BY THE MITOCHONDRIAL PROTEIN BNIP3

J. Kong*; S. Zeng; J.D. Geiger

Division of Neurovirology & Neurodegenerative Disorders, St. Boniface Hosp Res Ctr, Winnipeg, MB, Canada

Neuronal cell death has been shown to occur by apoptotic, necrotic and alternative cell death mechanisms. Recently, it was reported that the gene BNIP3, a member of the Bcl-2 family without a functional BH3 domain, induces cell death in a necrotic-like manner and that this gene-regulated cell death pathway involves opening of the mitochondrial permeability transition pore without caspase activation and cytochrome c release; BNIP3-mediated cell death could be induced by hypoxic challenge in non-neuronal cells. Here we test the hypothesis that BNIP3 may be a gene that when activated in brain is capable of inducing necrotic (possibly paraptotic) cell death. In rat brain, BNIP3 staining was not detectable by immunohistochemistry. However, BNIP3 expression was increased dramatically in models of brain injury and excitotoxicity. After blunt trauma to brain or intra-striatal injection of kainic acid, BNIP3 immunopositive cells were observed adjacent to the sites of injury and the BNIP3 positive cells showed DNA fragmentation as demonstrated by TUNEL and Hoechst 33342 staining. Western blot analysis showed a 30 kDa band from kainic acid injected brain; positive immunoblot was blocked by a BNIP3-GST recombinant protein. Similar results were observed in a chronic seizure model using i.p. injections of kainic acid. In situ hybridization results using a riboprobe for BNIP3 mRNA revealed that intra-striatal injections of kainic acid increased levels of BNIP3 mRNA in sites of brain injury. These results suggest that BNIP3 may be a new target for neuronal rescue strategies.

Supported by: grants from CIHR and NS39184

Program Number: 911.12

Day / time: Thursday, Nov. 15, 11:00 AM – 12:00 PM

Presentation Type: Poster

Presentation Location: Exhibit Hall E-11

DISTRIBUTION AND DIFFERENTIAL EXPRESSION OF CONNEXINS IN NEURONS AND GLIA

J.L. Nagy 1*; X. Li 1; L. Luo 1; B.D. Lynn 1; V.A. Ionescu 1; D. Patel 2; J. Rempel 1; W.A. Staines 2

1. Dept Physiol Fac Med, Univ Manitoba, Winnipeg, MB, Canada

2. Cell Mol Medicine, Univ Ottawa, Ottawa, ON, Canada

Numerous members of the connexin (Cx, the subunit of gap junctions) family of proteins are expressed in the adult mammalian CNS. Definitive information on their relative cellular and subcellular localization is required to clarify their potential functional roles in the glial syncytium on one hand or in the gap junction-mediated electrical coupling between neurons on the other. We have addressed these issues using antibodies against Cx26, Cx29, Cx30, Cx32, Cx36, Cx43 and Cx47 in conjunction with western blotting and immunohistochemistry. Our results have revealed connexin (Cx26, Cx30, Cx32 and Cx43) expression patterns among astrocytes and oligodendrocytes which display distinct regional and cellular differences in level of expression and connexin subtype, indicative of selective regulation of connexin expression in glia which differ according to brain region. Further, our results indicate that neuronal Cx36 as well as recently identified Cx47, which is also reported to be expressed by neurons, are widely distributed in brain, including regions where electrical synapses between neurons have not been previously reported or suspected. The data suggest that mammalian CNS contains an abundance of neuronal gap junctions that provide the structural basis for widespread electrical and/or metabolic integration between neurons in mammalian CNS. Our observations on the regional and cellular densities of neuronal connexins provide clear directions for further studies by illuminating CNS areas on which to focus functional investigations.

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