

UManitobaSFN2005

Saturday, Nov. 12 (PM) – Poster Presentations

Day / Time	Prog #	Presentation Type	Location	Authors	1st Author 1st Affiliation	Title
Saturday, Nov. 12, 4:00 PM – 5:00 PM	28.4	Poster	B64	<u>Z.Wang</u> ^{1*} ; N.J.Gardiner ³ ; C.Luke ³ ; A.Gott ³ ; P.Fernyhough ^{1,2}	Div. of Neurodegenerative Disorders, St. Boniface General Hospital Research Ctr.	EXPRESSION AND REGULATION OF HEXOKINASE ISOFORMS IN THE ADULT PERIPHERAL NERVOUS SYSTEM
Saturday, Nov. 12, 2:00 PM – 3:00 PM	28.10	Poster	B70	<u>C.W.Tweed</u> ^{1,2*} ; G.W.Glazner ^{2,3} ; P.Fernyhough ^{2,3}	Faculty of Life Sciences, Univ. of Manchester	ROLE OF TUMOR NECROSIS FACTOR—INDUCED ACTIVATION OF NUCLEAR FACTOR-B IN AXON REGENERATION IN AXOTOMIZED ADULT SENSORY NEURONS
Saturday, Nov. 12, 2:00 PM – 3:00 PM	37.6	Poster	E24	<u>C.E.Flores</u> ^{1*} ; X.Li ² ; J.I.Nagy ² ; A.Pereda ¹	Neuroscience, Albert Einstein Col. of Medicine	CONNEXIN35 (CX35) AND ZONULA OCCLUDENS-1 (ZO-1) INTERACT AT ELECTRICAL SYNAPSES IN GOLDFISH BRAIN
Saturday, Nov. 12, 3:00 PM – 4:00 PM	55.7	Poster	X10	<u>B.L.Shay*</u> ; S.Mehta	Dept Physical Therapy, Univ. of Manitoba	KINEMATIC ANALYSIS WITH LEG LENGTH DISCREPANCIES (LLD) OF THE LOWER LIMB IN HUMAN SUBJECTS

Sunday, Nov. 13 (PM) – Poster Presentations

Day / Time	Prog #	Presentation Type	Location	Authors	1st Author 1st Affiliation	Title
Sunday, Nov. 13, 4:00 PM – 5:00 PM	252.16	Poster	C3	<u>J.De Melo</u> ^{1*} ; Q.Zhou ² ; S.Cheng ² ; D.D.Eisenstat ^{1,2}	Dept Human Anatomy & Cell Sci, Univ. of Manitoba	DLX1 AND DLX2 REGULATION OF THE NEUROTROPHIN RECEPTOR TRK-B AND SURVIVAL OF LATE-BORN RETINAL GANGLION CELLS IN THE DEVELOPING MOUSE RETINA
Sunday, Nov. 13, 2:00 PM – 3:00 PM	337.10	Poster	VV50	<u>O.H.Khan</u> ^{1,2} ; M.Del Bigio ^{1,2*}	Pathology, Univ. of Manitoba	BEHAVIORAL AND NEUROPATHOLOGICAL CHANGES AFTER ADMINISTRATION OF NIMODIPINE, MAGNESIUM CHLORIDE, AND MAGNESIUM SULPHATE IN KAOLIN-INDUCED NEONATAL RAT HYDROCEPHALUS

Monday, Nov. 14 (AM) – Poster Presentations

Day / Time	Prog #	Presentation Type	Location	Authors	1st Author 1st Affiliation	Title
Monday, Nov. 14, 10:00 AM – 11:00 AM	377.3	Poster	D40	<u>Y.Dai</u> ^{1*} ; R.M.Brownstone ² ; L.M.Jordan ¹	Dept Physiol, Univ. of Manitoba	CHARACTERIZATION OF HYPERPOLARIZATION-ACTIVATED INWARD CURRENT IN LOCOMOTOR ACTIVITY RELATED NEURONS IN C-FOS--EGFP MICE
Monday, Nov. 14, 10:00 AM –	426.3	Poster	NN25	<u>P.Fernyhough</u> ^{1,2*} ; A.Verkhatsky ³ ; T.Huang ³	Div. of Neurodegenerative Disorders, St.	INSULIN-LIKE GROWTH FACTOR 1 ENHANCES MITOCHONDRIAL INNER MEMBRANE POTENTIAL AND

11:00 AM					Boniface Hospital Research Ctr.	INCREASES THE REDOX STATE OF THE MITOCHONDRIAL NAD(P)H POOL IN CATECHOLAMINERGIC CAD NEURONS
Monday, Nov. 14, 8:00 AM – 9:00 AM	456.5	Poster	VV90	<u>J.Lawrence</u> ^{1*} ; P.F.Gardiner ¹ ; K.R.Gardiner ¹ ; K.L.Malisza ^{1,2}	Dept Physiol, Univ. of Manitoba	COMPARISON OF NEURAL ACTIVITY IN THE RAT SPINAL CORD USING FUNCTIONAL MAGNETIC RESONANCE IMAGING (FMRI) AND FIELD POTENTIALS DURING NOXIOUS ELECTRICAL STIMULATION OF THE HIND PAW
Monday, Nov. 14, 10:00 AM – 11:00 AM	456.19	Poster	WW8	<u>J.Kornelsen</u> ^{1*} ; P.Stroman ²	Physiol., Univ. of Manitoba	INVESTIGATION OF NEURONAL ACTIVITY OF THE INJURED SPINAL CORD DURING LOWER LIMB MOVEMENT WITH SPINAL FMRI

Monday, Nov. 14 (PM) – Platform Presentations

Day / Time	Prog #	Presentation Type	Location	Authors	1st Author 1st Affiliation	Title
Monday, Nov. 14, 3:00 PM – 3:15 PM	477.9	Slide	Washington Convention Center – Room 206	<u>T.N.Le</u> ^{1*} ; D.D.Eisenstat ^{2,3}	Dept Biochem & Med Genetics, Univ. of Manitoba	INHIBITION OF SEMAPHORIN SIGNALING BY DLX HOMEBOX GENES PROMOTES TANGENTIAL MIGRATION OF INTERNEURONS TO THE NEOCORTEX

Monday, Nov. 14 (PM) – Poster Presentations

Day / Time	Prog #	Presentation Type	Location	Authors	1st Author 1st Affiliation	Title
Monday, Nov. 14, 2:00 PM – 3:00 PM	516.6	Poster	BB5	<u>J.Liu</u> ^{1*} ; J.R.McVagh ¹ ; P.B.Hedlund ² ; L.M.Jordan ¹	Physiol., The Univ. of Manitoba	5-HT-INDUCED LOCOMOTOR-LIKE ACTIVITY IS DEFECTIVE IN 5-HT7 RECEPTOR KNOCK-OUT MICE
Monday, Nov. 14, 4:00 PM – 5:00 PM	516.8	Poster	BB7	<u>L.M.Jordan</u> *; C.G.Gibbs	Dept Physiology, Univ. of Manitoba	MOTONEURONS THROUGHOUT THE LUMBAR SPINAL CORD OF THE NEONATAL RAT POSSESS 5-HT_{2A} RECEPTORS
Monday, Nov. 14, 4:00 PM – 5:00 PM	554.12	Poster	SS92	<u>K.L.Olson</u> ^{1*} ; J.S.Schapansky ² ; R.Van Der Ploeg ² ; G.W.Glazner ^{1,2}	Pharm. & Therap., Univ. of Manitoba	THE ROLE OF HYPERGLYCEMIA/HYPERINSULINEMIA ON NEURONAL SURVIVAL IN TGCRND8 MICE

Tuesday, Nov. 15 (AM) – Poster Presentations

Day / Time	Prog #	Presentation Type	Location	Authors	1st Author 1st Affiliation	Title
Tuesday, Nov. 15, 8:00 AM – 9:00 AM	630.1	Poster	Z7	<u>I.A.Rybak</u> ^{1*} ; D.A.McCrea ²	Sch. Biomed. Eng., Drexel Univ.	COMPUTATIONAL MODELING OF THE MAMMALIAN LOCOMOTOR CPG

Tuesday, Nov. 15 (PM) – Platform Presentations

Day / Time	Prog #	Presentation Type	Location	Authors	1st Author 1st Affiliation	Title
Tuesday, Nov. 15, 5:30 PM – 6:30 PM	N/A	Workshop	Washington Convention Center – Room 208			MEMBERS' BUSINESS MEETING

Tuesday, Nov. 15 (PM) – Poster Presentations

Day / Time	Prog #	Presentation Type	Location	Authors	1st Author 1st Affiliation	Title
Tuesday, Nov. 15, 4:00 PM – 5:00 PM	731.8	Poster	N3	<u>X.Li</u> *; C.Ciolofan; C.Olson; J.I.Nagy	Department of Physiology, Univ. of Manitoba	THE Y-BOX TRANSCRIPTION FACTOR 3 MSY3/ZONAB CO-LOCALIZES WITH CONNEXIN36 IN MOUSE RETINA AND IS TARGETED TO THE PROMOTER OF THE CONNEXIN36 GENE
Tuesday, Nov. 15, 1:00 PM – 2:00 PM	750.9	Poster	DD3	<u>D.C.Button</u> *; K.R.Gardiner; T.Marqueste; P.F.Gardiner	Physiol., Univ. of Manitoba	FREQUENCY--CURRENT RELATIONSHIPS IN ANESTHETIZED AND DECEREBRATED RAT HINDLIMB MOTONEURONS <i>IN SITU</i>
Tuesday, Nov. 15, 2:00 PM – 3:00 PM	788.18	Poster	TT62	<u>Z.Zhang</u> ; X.Ma; X.Yang; S.Zhang; J.Kong*	Human Anatomy and Cell Science, Univ. of Manitoba	A BNIP3-ACTIVATED NEURONAL CELL DEATH PATHWAY IN STROKE

Wednesday, Nov. 16 (AM) – Poster Presentations

Day / Time	Prog #	Presentation Type	Location	Authors	1st Author 1st Affiliation	Title
Wednesday, Nov. 16, 11:00 AM – 12:00 PM	848.12	Poster	L6	<u>S.Connor</u> ¹ ; P.T.J.Williams ^{1,3} ; B.Armstrong ⁴ ; T.L.Ivanco ² ; A.C.W.Weeks ^{1*}	Psychology, Nipissing Univ.	CHANGES IN SYNAPTIC MORPHOLOGY ARE ASSOCIATED WITH CORTICAL LONG-TERM POTENTIATION IN THE ANESTHETIZED RAT
Wednesday, Nov. 16, 10:00 AM – 11:00 AM	865.3	Poster	AA4	<u>S.Rossignol</u> ^{1*} ; J.Provencher ¹ ; L.M.Jordan ²	Physiology, Univ. Montreal Fac. Med.	EFFECTS OF INTRATHECAL CHOLINERGIC DRUGS ON HINDLIMB LOCOMOTION IN THE FIRST 2 WEEKS AFTER A COMPLETE SPINALIZATION IN CATS
Wednesday, Nov. 16, 9:00 AM – 10:00 AM	900.6	Poster	SS2	<u>K.Hartle</u> ^{1,2,3} ; T.L.Ivanco ^{1,2,3*}	Psychology, Univ. of Manitoba	MILD ISCHEMIA PRODUCES INFARCT BUT NOT REORGANIZATION

Wednesday, Nov. 16 (PM) – Poster Presentations

Day / Time	Prog #	Presentation Type	Location	Authors	1st Author 1st Affiliation	Title
Wednesday, Nov. 16, 1:00 PM – 2:00 PM	964.13	Poster	N6	<u>C.O.Olson</u> *; J.I.Nagy	Physiology, Univ. of Manitoba	CX36 REGIONAL AND DEVELOPMENTAL EXPRESSION IN MAMMALIAN BRAIN AND SPINAL CORD
Wednesday, Nov. 16, 3:00 PM – 4:00	965.19	Poster	P8	<u>N.Kamasawa</u> ¹ ; K.G.V.Davidson ¹ ; C.Furman ² ;	Biomedical Sciences, Colorado	CONNEXIN36-CONTAINING GAP JUNCTIONS IN OFF AND ON SUBLAMINAE OF THE INNER

PM

J.A.Sampson¹;
T.Yasumura¹;
J.I.Nagy³;
J.E.Rash^{1*}

State Univ.

**PLEXIFORM LAYER OF ADULT
RODENT RETINA**



Program Number: 28.4

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

Presentation Type: Poster

Presentation Location: Washington Convention Center – Hall A–C, Board # B64

EXPRESSION AND REGULATION OF HEXOKINASE ISOFORMS IN THE ADULT PERIPHERAL NERVOUS SYSTEM

Z.Wang^{1*}; N.J.Gardiner³; C.Luke³; A.Gott³; P.Fernyhough^{1,2}

1. Div. of Neurodegenerative Disorders, St. Boniface General Hospital Research Ctr., Winnipeg, MB, Canada^{2. Div. of Pharmacology and Therapeutics, Univ. of Manitoba, Winnipeg, MB, Canada^{3. Faculty of Life Sciences, Univ. of Manchester, Manchester, United Kingdom}}

Impairment of neuronal metabolism has been proposed as a key event in various neurodegenerative diseases. The enzyme, hexokinase, catalyzes the first rate-limiting step of glycolysis and regulates the conversion of glucose into glucose-6-phosphate. This initial phosphorylation of glucose is critical for efficient energy production within cells. Hexokinase is expressed at high levels in the CNS, however, the expression levels, isoform profile and regulation of activity of hexokinase is unknown in the PNS. Therefore, the aim of this study was to determine in the PNS the expression profile of hexokinase, role of growth factors in regulation of activity and effect of neurodegenerative disease on hexokinase function. Western blotting and immunofluorescent staining was used to determine the expression of the hexokinase isoforms. Hexokinase I was highly expressed in lumbar dorsal root ganglia (DRG) neurons and in sciatic nerve. There was evidence of low expression of hexokinase IV (glucokinase) in DRG. In cultures of adult sensory neurons treatment with insulin significantly raised hexokinase activity by 30% through a phosphoinositide 3-kinase (PI 3-kinase) dependent pathway. The effect of diabetes on hexokinase function was studied. DRG were isolated from 3wk streptozotocin-diabetic rats (model of type 1 diabetes) and hexokinase activity levels were reduced (p<0.05) by 25% compared with age matched controls (protein levels of hexokinase I were unaltered). The results show that hexokinase I is highly expressed in small-medium DRG neurons, is under control of insulin and, in an animal model of diabetic neuropathy, there is evidence of reduced hexokinase function. The latter finding could be a crucial contributor to energy failure in axons in diabetes and may trigger axon denervation in skin.

Support Contributed By: JDRF, Diabetes UK and CIHR

Citation:

Z. Wang, N.J. Gardiner, C. Luke, A. Gott, P. Fernyhough. EXPRESSION AND REGULATION OF HEXOKINASE ISOFORMS IN THE ADULT PERIPHERAL NERVOUS SYSTEM Program No. 28.4. 2005 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: 37.6

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

Presentation Type: Poster

Presentation Location: Washington Convention Center – Hall A–C, Board # E24

CONNEXIN35 (CX35) AND ZONULA OCCLUDENS-1 (ZO-1) INTERACT AT ELECTRICAL SYNAPSES IN GOLDFISH BRAIN

C.E.Flores^{1*}; X.Li²; J.I.Nagy²; A.Pereda¹

1. Neuroscience, Albert Einstein Col. of Medicine, Bronx, NY, USA^{2. Physiology, Univ. of Manitoba, Winnipeg, MB, Canada}

While protein-protein interactions are involved in the regulation of chemical synapses, little is known about their role in regulating gap junction-mediated electrical synapses. Auditory afferents terminating as “Club Endings” on the goldfish Mauthner cells are anatomically and physiologically identifiable “mixed” (electrical and chemical)

synaptic terminals that constitute a valuable model for the study of vertebrate electrical synapses, as it is possible to correlate physiological properties with structural and biochemical composition of individual synapses. Electrical synapses at these terminals are mediated by Cx35, the fish ortholog of the mammalian neuronal gap junction protein Cx36. Recent studies have revealed the interaction of Cx36 with ZO-1 in mouse brain, a scaffold protein of the MAGUK family. Given the high sequence homology between Cx36 and Cx35, we asked whether this association with ZO-1 also occurs in goldfish brain. In double-immunolabeling experiments, confocal microscopy revealed that Cx35 co-localizes with ZO-1 not only at Club Endings but also in other inputs to this and other neurons, suggesting that this association is common in goldfish brain. Biochemical experiments showed that Cx35 and ZO-1 co-immunoprecipitate from goldfish brain homogenates. Furthermore, as with Cx36, affinity precipitation and peptide competition experiments showed that this interaction is specific to PDZ-1 domain of ZO-1 and the carboxyl-terminal domain of Cx35. While the function of connexin35/ZO-1 association is not yet known, their interaction through conserved regions of both Cx35 and Cx36 carboxy-terminus, suggest that such a function could constitute a widespread property, relevant to all Cx35/Cx36-mediated electrical synapses. Finally, connexin35/ZO-1 association at Club Endings provides the opportunity to explore possible functional roles in regulating electrical transmission at these highly plastic junctions.

Support Contributed By: NIH (DC03186 to A.P.) and CIHR (to J.N.)

Citation:

C.E. Flores, X. Li, J.I. Nagy, A. Pereda. CONNEXIN35 (CX35) AND ZONULA OCCLUDENS-1 (ZO-1) INTERACT AT ELECTRICAL SYNAPSES IN GOLDFISH BRAIN Program No. 37.6. 2005 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: 28.10

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

Presentation Type: Poster

ROLE OF TUMOR NECROSIS FACTOR- α -INDUCED ACTIVATION OF NUCLEAR FACTOR- κ B IN AXON REGENERATION IN AXOTOMIZED ADULT

SENSORY NEURONS

C.W.Tweed^{1,2*}; G.W.Glazner^{2,3}; P.Fernyhough^{2,3}

1. Faculty of Life Sciences, Univ. of Manchester, Manchester, United Kingdom^{2. Div. of Neurodegenerative Disorders, St. Boniface Hospital Research Ctr., Winnipeg, MB, Canada}^{3. Dept Pharmacology and Therapeutics, Univ. of Manitoba, Winnipeg, MB, Canada}

Peripheral nerve damage induces tumor necrosis factor- α (TNF- α) expression at the site of

nerve damage and within the lumbar dorsal root ganglia (DRG). This axotomy-related up-regulation of TNF- α causes hyperalgesia, maintains sensory neuron survival through

activation of nuclear factor- κ B (NF- κ B), and plays a role in Wallerian degeneration in peripheral nerve. The aim of this study was to determine if TNF- α signaling through NF- κ B

directly regulates axon regeneration from axotomized adult rat sensory neurons. Dissociated adult DRG neurons cultured for 24h in defined serum-free medium, an in vitro model of axotomy, expressed (40pg/mg total protein) and secreted (92pg/ml supernatant) TNF- α .

Addition of 50ng/ml exogenous TNF- α to adult DRG neuron cultures caused a 2-fold increase in activation of NF- κ B within 15min, as measured by Western blot probing for

phosphorylated-I κ B α (an indicator of the activation state of NF- κ B). The activated

NF- κ B dimer can exist as a combination of various subunits, including p50, p65 and c-Rel. At a site of crush of the sciatic nerve, immunofluorescent staining for p50, p65 and c-Rel increased

within axons and Schwann cells by 12h post-injury. Treatment of cultured DRG neurons with 5 μ M κ B decoy DNA, an inhibitor of NF- κ B nuclear translocation, decreased neurite

outgrowth by 50% (p<0.05) at 1, 2 and 3 days in culture. Therefore, NF- κ B appears to play a role in collateral sprouting and axon regrowth perhaps through activation by local production of

TNF- α , induced in response to injury.

Support Contributed By: BBSRC, UK and CIHR, Canada

Citation:

C.W. Tweed, G.W. Glazner, P. Fernyhough. ROLE OF TUMOR NECROSIS FACTOR- α -INDUCED ACTIVATION OF NUCLEAR FACTOR- κ B IN AXON

REGENERATION IN AXOTOMIZED ADULT SENSORY NEURONS Program No. 28.10. 2005 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: 55.7

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

Presentation Type: Poster

Presentation Location: Washington Convention Center – Hall A–C, Board # X10

KINEMATIC ANALYSIS WITH LEG LENGTH DISCREPANCIES (LLD) OF THE LOWER LIMB IN HUMAN SUBJECTS

B.L.Shay^{*}; S.Mehta

Dept Physical Therapy, Univ. of Manitoba, Winnipeg, MB, Canada

Clinically it has been observed that patients seeking treatment for disorders of the musculoskeletal system often have a limb length discrepancy (LLD). Generally, LLD of greater than 2 cm is corrected with a shoe raise, as it is thought that LLD of this magnitude would significantly change the biomechanics involved in ambulatory activities of daily living. It is also thought that LLD contributes to greater risk for developing disabling spinal disorders and other related degenerative changes in the lower limbs. There is little research to date on the effects of the magnitude of LLD and corresponding changes in kinematics. Objectives of this study are to compare joint kinematics of the lower limb including pelvis, hip and knee using a human model of LLD. Healthy, young subjects (aged 20–40) with no existing LLD are recruited for the study. The VICON motion analysis system is used to capture lower body kinematic data during level walking. LLD is induced by providing a cork shoe raise attached with Velcro to one limb. Each participant is analyzed in 4 conditions: no raise, 1 cm, 2 cm and 3 cm raises. Joint angles are compared using the VICON software. Significant alterations are noted in hip abduction/adduction and pelvic obliquity in the 3cm LLD condition. Thus, limb length discrepancies greater than 2 cm cause significant biomechanical and kinematic alterations during level walking. To avoid abnormal movement patterns contributing to stress and strain on the hip, pelvis and corresponding spinal joints, LLD should be minimized with a shoe raise.

Support Contributed By: CFI 3209

Citation:

B.L. Shay, S. Mehta. KINEMATIC ANALYSIS WITH LEG LENGTH DISCREPANCIES (LLD) OF THE LOWER LIMB IN HUMAN SUBJECTS Program No. 55.7. 2005 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: 252.16

Day / time: Sunday, Nov. 13, 4:00 PM – 5:00 PM

Presentation Type: Poster

Presentation Location: Washington Convention Center – Hall A–C, Board # C3

DLX1 AND DLX2 REGULATION OF THE NEUROTROPHIN RECEPTOR TRK–B AND SURVIVAL OF LATE–BORN RETINAL GANGLION CELLS IN THE DEVELOPING MOUSE RETINA

J.De Melo^{1*}; Q.Zhou²; S.Cheng²; D.D.Eisenstat^{1,2}

1. Dept Human Anatomy & Cell Sci, Univ. of Manitoba, Winnipeg, MB, Canada. 2. Dept Pediatrics & Child Health, Univ. of Manitoba, Winnipeg, MB, Canada

Dlx homeobox genes play important roles during development of the vertebrate forebrain and retina. Members of the Dlx gene family are expressed in retinal ganglion cells (RGC), amacrine and horizontal cells of the developing and postnatal retina. Expression begins at E12.5 and is maintained until late embryogenesis for Dlx1, while Dlx2 expression extends to adulthood. The Dlx1/Dlx2 null retina demonstrates a reduced ganglion cell layer (GCL), with loss of late–born differentiated RGCs due to increased apoptosis and optic nerve thinning. Proliferation indices are unaffected in the mutant retinas. Signaling through the neurotrophin receptor trkB is proposed to regulate the dynamics of RGC apoptosis throughout retinal development. The onset of trkB expression is E12.5 and extends postnatally through to the adult. Initial expression is found in the vitreal/central neuroretina, then becomes limited to the GCL by E18.5. TrkB is co–expressed with RGC markers such as Brn3b and Isl–1. There is co–expression of trkB with Dlx1 and Dlx2. In the Dlx1/Dlx2 null retina, trkB expression is reduced in the GCL as early as E13.5. We have explored trkB as a candidate transcriptional target of Dlx genes. Using a modified chromatin immunoprecipitation (ChIP) assay of embryonic retina, DLX2 binds to specific regions of the trkB promoter in situ. In vitro confirmation of binding and the functional consequences of DLX binding to regions of the trkB promoter signify the potential importance of trkB regulation by Dlx transcription factors. RGC differentiation and survival require the coordinated expression of transcription factors, such as Math5, Brn3b, Dlx1 and Dlx2. Signaling mediated by the neurotrophin receptor trkB may contribute to survival of late–born RGCs whose terminal–differentiation is regulated by Dlx gene function.

Support Contributed By: CIHR (JdM), Manitoba Health Research Council (SC) and the Foundation Fighting Blindness – Canada (DDE)

Citation:

J. De Melo, Q. Zhou, S. Cheng, D.D. Eisenstat. DLX1 AND DLX2 REGULATION OF THE NEUROTROPHIN RECEPTOR TRK–B AND SURVIVAL OF LATE–BORN RETINAL GANGLION CELLS IN THE DEVELOPING MOUSE RETINA Program No. 252.16. 2005 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: 377.3

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

Presentation Type: Poster

Presentation Location: Washington Convention Center – Hall A–C, Board # D40

CHARACTERIZATION OF HYPERPOLARIZATION–ACTIVATED INWARD CURRENT IN LOCOMOTOR ACTIVITY RELATED NEURONS IN C–FOS–EGFP MICE

Y.Dai^{1*}; R.M.Brownstone²; L.M.Jordan¹

1. Dept Physiol, Univ. of Manitoba, Winnipeg, MB, Canada. 2. Surgery, Anat & Neurobiol, Dalhousie Univ., Halifax, NB, Canada

Hyperpolarization–activated inward current (I_h) is a nonselective cation current widely distributed in many cell types. I_h has been shown to be involved in the production of bursting during various forms of rhythmic activity, and can be enhanced by 5–HT in many types of neurons. Since I_h is activated at subthreshold voltage, it could play a role in initiating or modulating rhythmic activity such as locomotion. Study of I_h in the neurons related to rhythmic generation is important for us to understand the mechanism generating locomotion. However, little is known about the properties of I_h in spinal neurons involved in the locomotion. Using CFos–EGFP transgenic mice (P6–P12) we are able to target the spinal interneurons activated by locomotion. Following a locomotor task, whole–cell patch clamp recordings were obtained in spinal cord slices (200–250 μm) from EGFP+ neurons distributed in lamina VII, VIII and X. In voltage clamp mode, I_h was activated by voltage step commands (3 sec) –50 to –130 mV from holding potentials of –40 to –50 mV and measured as the difference between the initial current and the steady–state inward current. I_h was expressed in 55% of EGFP neurons (21/38). The voltage of activation of I_h was about –69 ± 11 mV and the maximum conductance (G_{h_max}) was –0.47 ± 0.53 nS, which was calculated by Ohm

law using the value for the largest I_h (usually the last command step). Bath application of 5–HT (15–20 μM) usually induced a

persistent inward current and enhanced the I_h by depolarizing its activation voltage by 4.5 ± 7 mV with a small increase in G_{h_max}

(11.5%, n=8). I_h was blocked by ZD7288 (15–50 μM) or Cesium (1–2 mM) with or without 5–HT. Some cells (n=5), however, had an

opposite response to 5–HT, which was shown as a small decrease in G_{h_max} accompanied by a reduction of the current at the holding potential with no shift in activation voltage. The mechanisms mediating the modulation of I_h by 5–HT are being investigated.

Citation:

Y. Dai, R.M. Brownstone, L.M. Jordan. CHARACTERIZATION OF HYPERPOLARIZATION–ACTIVATED INWARD CURRENT IN LOCOMOTOR ACTIVITY RELATED NEURONS IN C–FOS–EGFP MICE Program No. 377.3. 2005 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: 337.10

Day / time: Sunday, Nov. 13, 2:00 PM – 3:00 PM

Presentation Type: Poster

Presentation Location: Washington Convention Center – Hall A–C, Board # VV50

BEHAVIORAL AND NEUROPATHOLOGICAL CHANGES AFTER ADMINISTRATION OF NIMODIPINE, MAGNESIUM CHLORIDE, AND MAGNESIUM SULPHATE IN KAOLIN–INDUCED NEONATAL RAT HYDROCEPHALUS

O.H.Khan^{1,2*}; M.Del Bigio^{1,2*}

1. Pathology, Univ. of Manitoba, Winnipeg, MB, Canada. 2. Manitoba Inst. of Child Health, Winnipeg, MB, Canada

O. Khan, M.R. Del Bigio* Department of Pathology, University of Manitoba

Hydrocephalus is a common neurological condition characterized by obstruction of cerebrospinal fluid (CSF) flow leading to enlargement of CSF–containing ventricular cavities in the brain. The use of pharmacological drugs to reduce brain damage in immature (5–7 weeks)

hydrocephalic rats have been investigated in our lab with various outcomes (Del Bigio 2002). Magnesium sulphate and nimodipine have shown promising results with improvements in behavioral outcomes and reduced damage in the white matter (Del Bigio 2001, Khan 2003). However, shunting delay is more likely to be a clinical problem in very premature infants. Hence our desire to study the effects of these agents in neonatal rats with brains more comparable to those of 24–26 week humans. Rats were injected with kaolin on postnatal day 1 and magnetic resonance imaging (MRI) was performed on day 7 to assess ventricle size. On postnatal day 7 osmotic mini pumps with MgSO₄, MgCl₂ (150 mg/kg/day), or nimodipine (15 or 30 mg/kg/day) were loaded and placed subcutaneously. Behavior tests for developmental delay were conducted at various time–points. Rotorod and Morris Water Maze were performed on day 20. MRI was conducted on day 21 for final ventricle size assessment. Pups were euthanized on day 21 and brains were subjected to histopathological and biochemical analyses. Nimodipine was of no benefit. Pups receiving MgCl₂ were also of no benefit; however, the hyperosmolarity of the drug caused scab formation and possibly complicated results. MgSO₄ experiments are currently being assessed. *Support Contributed By: University of Manitoba and Manitoba Institute of Child Health*

Citation:

O.H. Khan, M. Del Bigio. BEHAVIORAL AND NEUROPATHOLOGICAL CHANGES AFTER ADMINISTRATION OF NIMODIPINE, MAGNESIUM CHLORIDE, AND MAGNESIUM SULPHATE IN KAOLIN–INDUCED NEONATAL RAT HYDROCEPHALUS Program No. 337.10. 2005 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: 426.3

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

Presentation Type: Poster

Presentation Location: Washington Convention Center – Hall A–C, Board # NN25

INSULIN–LIKE GROWTH FACTOR 1 ENHANCES MITOCHONDRIAL INNER MEMBRANE POTENTIAL AND INCREASES THE REDOX STATE OF THE MITOCHONDRIAL NAD(P)H POOL IN CATECHOLAMINERGIC CAD NEURONS

P.Fernyhough^{1,2*}; A.Verkhatsky³; T.Huang³

1. Div. of Neurodegenerative Disorders, St. Boniface Hospital Research Ctr., Winnipeg, MB, Canada. 2. Dept of Pharmacology and Therapeutics, Univ. of Manitoba, Winnipeg, MB, Canada. 3. Faculty of Life Sciences, Univ. of Manchester, Manchester, United Kingdom

Impaired neuronal metabolism has been proposed as underlying numerous neurodegenerative processes in the central nervous system (CNS). The mechanism of loss of dopaminergic neurons of the substantia nigra in Parkinson's disease is unknown and the target of intense investigation.

We studied the role of insulin–like growth factor 1 (IGF–1) and neurotrophin–3 (NT–3) in control of energy metabolism in a differentiated catecholaminergic CNS clonal cell line, CAD cells. This mouse cell line exhibits some phenotypic features of dopaminergic neurons of the substantia nigra. Real time fluorescence video microscopy was utilized to analyze mitochondrial inner membrane potential (Δψ_m) and the redox state of the NAD(P)H pool of the mitochondria. IGF–1 or NT–3 treatment for 24 hr increased Δψ_m by 35%–70% dependent on the imaging method employed (p<0.005). Inhibition of phosphoinositide 3–kinase (PI 3–kinase) by 10 μM LY294002 completely prevented the IGF–1–dependent increase in Δψ_m.

Interestingly, acute treatment of IGF–1 elevated the redox state of the mitochondrial NAD(P)H pool within seconds of application, suggesting an elevation in the rate of glycolysis and tricarboxylic acid (TCA) cycle upon IGF–1 treatment. This study demonstrates that IGF–1 utilizes the PI 3–kinase pathway to control energy metabolism in differentiated catecholaminergic CAD neurons. The results suggest that dopaminergic loss of function and eventual cell loss in the substantia nigra in Parkinson's disease could be ameliorated by IGF–1– and/or NT–3–based therapies.

Citation:

P. Fernyhough, A. Verkhatsky, T. Huang. INSULIN–LIKE GROWTH FACTOR 1 ENHANCES MITOCHONDRIAL INNER MEMBRANE POTENTIAL AND INCREASES THE REDOX STATE OF THE MITOCHONDRIAL NAD(P)H POOL IN CATECHOLAMINERGIC CAD NEURONS Program No. 426.3. 2005 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: 456.5

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

Presentation Type: Poster

Presentation Location: Washington Convention Center – Hall A–C, Board # VV90

COMPARISON OF NEURAL ACTIVITY IN THE RAT SPINAL CORD USING FUNCTIONAL MAGNETIC RESONANCE IMAGING (fMRI) AND FIELD POTENTIALS DURING NOXIOUS ELECTRICAL STIMULATION OF THE HIND PAW

J.Lawrence^{1*}; P.F.Gardiner¹; K.R.Gardiner¹; K.L.Malisza^{1,2}

1. Dept Physiol, Univ. of Manitoba, Winnipeg, MB, Canada^{2. MR Research and Development, National Research Council–Inst. for Biodiagnostics, Winnipeg, MB, Canada}

Functional MRI is a non-invasive technique that indirectly detects areas of neuronal activity by observing local changes in blood oxygen. Until recently fMRI has been used only for brain imaging, however, it has since been applied to the spinal cord (spinal fMRI). The relationship between areas of fMRI activity and areas of neuronal activity must be verified by comparison with gold standards in order for spinal fMRI to become more widely accepted. In the present study we compare areas of fMRI activity with areas of extracellular field potentials during noxious electrical stimulation of the hind paw in rats. Halothane-anesthetized rats were placed supine on a quadrature surface coil tuned and matched to 300 MHz. Six, 2 mm thick slices were centred on the vertebra and intervertebral discs between T12 and L1. Three functional imaging experiments on each animal were performed using electrical stimulation (~ 7 mA) delivered by two silver needle electrodes inserted subcutaneously in the dorsal surface of the right hind paw. In the same rats, electrophysiology studies were then performed in which the third to fifth lumbar spinal cord segments were exposed and extracellular recordings were taken in the gray matter on both sides using a 2–Mg tungsten electrode. Recordings were taken during rest and stimulation every 150 μm to a depth of 1800 μm, at several sites spanning these segments. The greatest amount of fMRI activity was observed at the T13 vertebra. Peak negative extracellular potentials were also observed at this level corresponding to the L4 spinal cord segment. The comparison of areas of field potentials with areas of fMRI activity confirm that spinal fMRI can be used to detected areas of neuronal activity in the spinal cord.

Citation:
J. Lawrence, P.F. Gardiner, K.R. Gardiner, K.L. Malisza. COMPARISON OF NEURAL ACTIVITY IN THE RAT SPINAL CORD USING FUNCTIONAL MAGNETIC RESONANCE IMAGING (fMRI) AND FIELD POTENTIALS DURING NOXIOUS ELECTRICAL STIMULATION OF THE HIND PAW Program No. 456.5. 2005 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: 477.9

Day / time: Monday, Nov. 14, 3:00 PM – 3:15 PM

Presentation Type: Slide

Presentation Location: Washington Convention Center – Room 206

INHIBITION OF SEMAPHORIN SIGNALING BY DLX HOMEBOX GENES PROMOTES TANGENTIAL MIGRATION OF INTERNEURONS TO THE NEOCORTEX

T.N.Le^{1*}; D.D.Eisenstat^{2,3}

1. Dept Biochem & Med Genetics, Univ. of Manitoba, Winnipeg, MB, Canada^{2. Dept Pediatrics & Child Health, 3. Dept Human Anatomy & Cell Science, Univ. of Manitoba, Winnipeg, MB, Canada}

Dlx genes are expressed in the ganglionic eminences (GE) of the developing forebrain. Dlx1/Dlx2 double knockout mice die at birth with abnormal cortical development, including loss of tangential migration of GABAergic inhibitory interneurons to the neocortex. We have applied ChIP (chromatin immunoprecipitation) to identify transcriptional targets of DLX homeoproteins derived in vivo from embryonic day 13.5 (E13.5) GE. Following cross-linking to enrich for protein–DNA complexes, nucleoproteins were incubated with DLX antibodies and genomic DNA (gDNA) fragment pools, including putative DLX transcriptional targets, were further characterized. PCR for the Neuropilin–2 promoter (NRP–2) showed that both DLX1 and DLX2 bind to this regulatory region in situ; neither binds to the Neuropilin–1 promoter. Electromobility shift assays confirmed direct binding of DLX1 and DLX2 to the NRP–2 promoter in vitro. Reporter assays demonstrated that both DLX1 and DLX2 repress NRP–2 expression, confirming the functional significance of DLX binding to this promoter region and consistent with increased and aberrant expression of NRP–2 in the Dlx1/Dlx2 null mouse. Hence, DLX homeoproteins may function as transcriptional repressors in vivo. We have generated a Dlx1/Dlx2/Npn–2 triple knockout mouse by crossing Dlx1/Dlx2 heterozygotes with Nrp–2 null mutants. The neocortex of the triple knockout compared to the Dlx1/Dlx2 null demonstrates a partial restoration of GABA and calbindin–expressing interneurons in the neocortex by P0 (p<0.05). NRP–2 is a receptor for semaphorin axonal guidance ligands. ChIP provides evidence for NRP–2 as a direct Dlx homeodomain target from embryonic forebrain in situ. These findings improve our understanding of Dlx function in cortical development in vivo, especially regulation of tangential interneuron migration to neocortex.

Support Contributed By: NSERC (TNL) and the Foundation Fighting Blindness – Canada (DDE)

Citation:

T.N. Le, D.D. Eisenstat. INHIBITION OF SEMAPHORIN SIGNALING BY DLX HOMEBOX GENES PROMOTES TANGENTIAL MIGRATION OF INTERNEURONS TO THE NEOCORTEX Program No. 477.9. 2005 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: 456.19

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

Presentation Type: Poster

Presentation Location: Washington Convention Center – Hall A–C, Board # WW8

INVESTIGATION OF NEURONAL ACTIVITY OF THE INJURED SPINAL CORD DURING LOWER LIMB MOVEMENT WITH SPINAL FMRI

J.Kornelsen^{1*}; P.Stroman²

1. Physiol., Univ. of Manitoba, Winnipeg, MB, Canada^{2. Centre for Neuroscience Studies, Queens Univ., Kingston, ON, Canada}

The patterns of neuronal activity in response to passive and active motor tasks in spinal cord injured (SCI) volunteers are compared to those of healthy volunteers, with the aim of assessing the condition of the cord caudal to an injury site. Using functional magnetic resonance imaging (fMRI) of the spinal cord, images were obtained in the sagittal orientation while subjects participated in either a passive or active movement task. During the active task, the volunteer alternately flexed and extended the ankles to produce a pedaling movement on a MR-compatible pedaling device. During the passive task, the examiner moved the pedaling device to induce the ankle flexion/extension movements with no assistance or resistance from the volunteer. 12 SCI volunteers participated, and participated in active and/or passive activities according to their abilities. Two sets of data were acquired with each activity in order to determine reproducibility. Previous results obtained with healthy volunteers demonstrated neuronal activity in the lumbar spinal cord in the dorsal horn of the gray matter during passive movement, and in the dorsal, ventral and intermediate area of the gray matter during active movement. Results obtained with SCI volunteers are similar, and show neuronal activity in dorsal gray matter during passive movement, and activity in the ventral gray matter as well as apparent reflex activity in the intermediate area during active movement. Results suggest that spinal cord condition can be assessed using spinal fMRI with sagittal imaging sufficient to reveal areas of neuronal activity in the cord caudal to an injury, with motor, sensory and reflex activity. This information is expected to aid with the assessment of spinal cord injury and subsequent rehabilitation. Significantly, this information can be obtained in a non-invasive manner in standard clinical MRI systems, and is beyond that which self report techniques are able to provide.

Support Contributed By: Canadian Institutes of Health Research

Citation:

J. Kornelsen, P. Stroman. INVESTIGATION OF NEURONAL ACTIVITY OF THE INJURED SPINAL CORD DURING LOWER LIMB MOVEMENT WITH SPINAL FMRI Program No. 456.19. 2005 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: 516.6

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

Presentation Type: Poster

Presentation Location: Washington Convention Center – Hall A–C, Board # BB5

5-HT-INDUCED LOCOMOTOR-LIKE ACTIVITY IS DEFECTIVE IN 5-HT7 RECEPTOR KNOCK-OUT MICE

J.Liu^{1*}; J.R.McVagh¹; P.B.Hedlund²; L.M.Jordan¹

1. Physiol., The Univ. of Manitoba, Winnipeg, MB, Canada^{2. Molec. Biol., The Scripps Research Inst., La Jolla, CA, USA}

It has been well established that the application of 5-HT elicits locomotor-like activity in both postnatal rat and mouse *in vitro* preparations. Several lines of evidence have shown that both 5-HT_{2A} and 5-HT₇ receptors in the spinal cord are involved in the control of locomotor-like activity. Hedlund et al (PNAS 100:1375–1380, 2003) generated a mouse strain with a targeted disruption of the 5-HT₇ receptor gene (5-HT₇^{–/–} mice). Here, we have used this mouse strain to test the hypothesis that 5-HT induces locomotor-like activity in the isolated neonatal mouse spinal cord by acting at the 5-HT₇ receptor. Experiments were performed on post-natal day 1–4 animals. The spinal cord (below T1) was isolated, pinned ventral side up, and recordings were made from the L2 and L5 ventral roots bilaterally. 5-HT (20–50 μM) was applied into the recording chamber for evoking locomotor-like activity. Spinal cords from 5-HT₇^{+/+} animals

displayed the typical locomotor-like response to 5-HT, with alternation between ipsilateral flexor (L2) and extensor (L5) root rhythmic discharges. This locomotor-like activity was slowed and then blocked by SB269970, a specific 5-HT₇ receptor antagonist. 5-HT₇^{–/–} animals did not consistently respond to 5-HT with rhythmic activity. Increases in tonic activity in the four ventral roots were observed in some cases. In others, random synchronous burst discharges in two or four ventral roots occurred. In other cases, rhythmic activity in the ventral roots occurred without consistent locomotor-like patterns. Such rhythmic activity in the 5-HT₇^{–/–} animals was not affected by the 5-HT₇ antagonist SB269970, but could be abolished by the 5-HT_{2A} antagonists ketanserin and spiperone. We conclude that locomotor-like activity produced by 5-HT applied to the isolated neonatal mouse spinal cord requires functional 5-HT₇ receptors. 5-HT may produce rhythmic activity in 5-HT₇^{–/–} mice by acting at 5-HT_{2A} receptors.

Support Contributed By: CHR and MHRC

Citation:

J. Liu, J.R. McVagh, P.B. Hedlund, L.M. Jordan. 5-HT-INDUCED LOCOMOTOR-LIKE ACTIVITY IS DEFECTIVE IN 5-HT7 RECEPTOR KNOCK-OUT MICE Program No. 516.6. 2005 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: 516.8

Day / time: Monday, Nov. 14, 4:00 PM – 5:00 PM

Presentation Type: Poster

Presentation Location: Washington Convention Center – Hall A–C, Board # BB7

MOTONEURONS THROUGHOUT THE LUMBAR SPINAL CORD OF THE NEONATAL RAT POSSESS 5–HT_{2A} RECEPTORS

L.M.Jordan*; C.G.Gibbs

Dept Physiology, Univ. of Manitoba, Winnipeg, MB, Canada

Previous studies have shown that 5–HT induces locomotor–like activity in the isolated spinal cord of the neonatal rat, in part via 5–HT_{2A} receptors. However, we (Liu and Jordan, J. Neurophysiol. doi:10.1152/jn.00136.2005) found that 5–HT_{2A} antagonists applied directly to the L2 segment did not alter L2 discharges, whereas applications over the L5 segment blocked locomotion. Here we test the hypothesis that the differential affects of these antagonists on the discharge of L2 and L5 motoneurons may be due to a differential distribution of 5–HT_{2A} receptors on motoneurons in different segments of the lumbar spinal cord. We used intraperitoneal injections of Fluorogold (FG) to label all the motoneurons in the lumbar cord of PND 5 neonatal rats and determined the distribution of 5–HT_{2A} receptors using immunolabeling. We observed extensive 5–HT_{2A} labeling on motoneuron cell bodies and dendrites in all segments examined (L1 – L5). Not all FG positive cells were labeled, and distinct clusters of retrogradely labeled motoneurons in L2 and L5 had no 5–HT_{2A} labeling. Most groups of motoneurons were densely labeled at all levels of the lumbar cord, however, and there was no obvious difference between the 5–HT_{2A} receptor labeling of presumed limb motoneurons in the L2 and L5 segments. Autonomic motoneurons in the intermediolateral cell column do not possess 5–HT_{2A} receptors, and retrogradely labeled motoneurons near the central canal were also negative for the 5–HT_{2A} receptor. Using this approach, it was not possible to identify any non–motoneurons in areas of the ventral horn outside the boundaries of lamina IX that were positively labeled for the 5–HT_{2A} receptor. Labeling in laminae VII and VIII was very sparse in comparison to lamina IX. There were bundles of densely labeled dendrites extending from medial lamina IX into the area beneath and around the central canal. Labeled neurons were also observed in the superficial dorsal horn. We conclude that at PND 5 most limb motoneurons in the lumbar cord, regardless of their rostro–caudal position, are rich in 5–HT_{2A} receptors.

Support Contributed By: NIH, CIHR

Citation:

L.M. Jordan, C.G. Gibbs. MOTONEURONS THROUGHOUT THE LUMBAR SPINAL CORD OF THE NEONATAL RAT POSSESS 5–HT_{2A} RECEPTORS Program No. 516.8. 2005 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: 630.1

Day / time: Tuesday, Nov. 15, 8:00 AM – 9:00 AM

Presentation Type: Poster

Presentation Location: Washington Convention Center – Hall A–C, Board # Z7

COMPUTATIONAL MODELING OF THE MAMMALIAN LOCOMOTOR CPG

I.A.Rybak1*; D.A.McCrea2

1. Sch. Biomed. Eng., Drexel Univ., Philadelphia, PA, USA2. Spinal Cord Res. Center, Univ. of Manitoba, Winnipeg, MB, Canada

In mammals, the basic locomotor pattern of alternating flexor and extensor activities is produced by spinal central pattern generator (CPG) that can operate in the absence of rhythmic input from higher centers and peripheral feedback. We have developed a model of the cat spinal rhythm generator (RG) that includes a homogenous population of directly coupled excitatory neurons that is split into two "half–centers" inhibiting each other via inhibitory interneuron populations. The endogenous rhythmic properties of single neurons (modeled in the Hodgkin–Huxley style) are based on the persistent sodium and h channels. The RG model can generate a realistic range of locomotor cycle periods. Increases in excitatory drive to the half–centers decrease the cycle period. The durations of the flexor and extensor phases replicate the wide ranges seen in real locomotion and can be adjusted by regulating intrinsic neuronal properties, mutual inhibition and tonic excitatory drives to the each of half–centers. In accord with in vitro observations in rats, blockade of synaptic inhibition in the model destroys the locomotor pattern and results in synchronized oscillations of flexor and extensor activities. This RG model has been incorporated into our recent model of spinal circuitry in which the CPG is comprised of separate RG and pattern formation networks (Rybak et al. 2004). The two–level CPG allows for independent control of locomotor phase timing and the degree of motoneuron recruitment. This flexibility allows the model to reproduce a number of experimental observations obtained during brainstem–evoked fictive locomotion in the cat. These include (a) realistic motoneuron firing patterns, (b) spontaneous deletions of rhythmic activity during locomotion, and (c) perturbations of motoneuron timing and recruitment produced by sensory feedback from hindlimb muscle and cutaneous afferents. The model also provides a basis for functional identification of spinal interneurons involved in generation and control of the locomotor pattern.

Support Contributed By: CIHR and NIH

Citation:

I.A. Rybak, D.A. McCrea. COMPUTATIONAL MODELING OF THE MAMMALIAN LOCOMOTOR CPG Program No. 630.1. 2005 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: 554.12

Day / time: Monday, Nov. 14, 4:00 PM – 5:00 PM

Presentation Type: Poster

Presentation Location: Washington Convention Center – Hall

A–C, Board # SS92

THE ROLE OF

HYPERGLYCEMIA/HYPERINSULINEMIA ON

NEURONAL SURVIVAL IN TGCRND8 MICE

K.L.Olson1*; J.S.Schapansky2; R.Van Der Ploeg2;

G.W.Glazner1,2

1. Pharm. & Therap., Univ. of Manitoba, Winnipeg, MB,

Canada2. St. Boniface Hospital Research Ctr., Winnipeg, MB,

Canada

Alzheimer's disease (AD) is a progressive form of dementia

characterized by decline in cognition and behavioural alterations. Brain pathology of AD is distinguished by the accumulation of amyloid beta (A β) plaques.

TgCRND8 mice encode for the double mutant human form of APP695 (KM670/671NL + V717F), generate A β plaques, and

possess cognitive deficits as seen in humans with AD.

Evidence has shown that people with type II diabetes may be at increased risk for developing AD. Type II diabetes is characterized by hyperglycemia & early hyperinsulinemia (due to insulin receptor insensitivity), followed by hypoinsulinemia in later stages. Studies suggest neuronal glucose metabolism is controlled by insulin, and, disruptions in insulin signaling may contribute to pathophysiological aberrations seen in AD. Further, cellular clearance of A β may be modulated in part by

insulin. We examined cell survival in type II diabetic

conditions in TgCRND8 mice.

Embryonic day 16/17 primary cortical neurons were harvested

from APP (+/–) mice and wild–type (WT) littermates (–/–) and

cultured according to established protocol. Cells were allowed

to grow in neurobasal for 7 days. Different concentrations of

insulin & glucose were added to culture media. Using digital

photography & phase–contrast microscopy, baseline survival

photos were taken before treatments & subsequent photos were

taken every 24 hours for 14 days.

Hyperglycemic conditions elicited a 40% increase in survival 6

days after treatment in APP cultures. WT cultures mirrored

“normal control survival” trends with no significant death

observed regardless of culture condition. 5 days after treatment,

high insulin & high glucose added together to APP cultures

showed a 20–35% increase in survival when compared to low

insulin & high glucose conditions. No significant increase in

survival was observed in WT cultures under these conditions.

Support Contributed By: The Alzheimer's Society of Manitoba,

and the McCrorie–West Family Fellowship

Citation:

K.L. Olson, J.S. Schapansky, R. Van Der Ploeg, G.W. Glazner.

THE ROLE OF HYPERGLYCEMIA/HYPERINSULINEMIA

ON NEURONAL SURVIVAL IN TGCRND8 MICE Program

No. 554.12. 2005 Abstract Viewer/Itinerary Planner.

Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: N/A

Day / time: Tuesday, Nov. 15, 5:30 PM – 6:30 PM

Presentation Type: Workshop

Presentation Location: Washington Convention Center –

Room 208

MEMBERS' BUSINESS MEETING

Information will be posted on the SfN Web site as it becomes available.

CONTACT: Melinda Colton

Executive Department

Society for Neuroscience

11 Dupont Circle, NW

Suite 500

Washington, DC 20036

Phone: (202) 462–6688

Email: melinda@sfn.org

www.sfn.org

Citation:

. MEMBERS' BUSINESS MEETING Program No. N/A. 2005

Abstract Viewer/Itinerary Planner. Washington, DC: Society

for Neuroscience, 2005. Online.

Program Number: 731.8

Day / time: Tuesday, Nov. 15, 4:00 PM – 5:00 PM

Presentation Type: Poster

Presentation Location: Washington Convention Center – Hall A–C, Board # N3

THE Y–BOX TRANSCRIPTION FACTOR 3 MSY3/ZONAB CO–LOCALIZES WITH CONNEXIN36 IN MOUSE RETINA AND IS TARGETED TO THE PROMOTER OF THE CONNEXIN36 GENE

X.Li*; C.Ciolofan; C.Olson; J.I.Nagy

Department of Physiology, Univ. of Manitoba, Winnipeg, MB, Canada

It has been previously reported that the canine Y–box transcription factor ZONAB binds to the SH3 domain of ZO–1, a membrane–associated guanylate kinase (MAGuK) family member. ZONAB was shown to interact with the ErbB2 promoter sequence containing an inverted CCAAT box and to regulate ErbB2 promoter activity. We have previously reported that neuronal connexin36 (Cx36) binds to the first PDZ domain of ZO–1. Since the Cx36 promoter sequence contains an inverted CCAAT box, we investigated whether the mouse orthologue of ZONAB, Y–box transcription factor 3 (MsY3), has the ability to bind also to the Cx36 promoter. We isolated a 2.8 kb mouse Cx36 promoter by PCR, and constructed Cx36promoter–pEGFP–1 and Cx36promoter–pGL3 vectors. After transient transfection of these plasmids separately into the PC12 cells, which express Cx36 and ZO–1, and show co–localization of these proteins, GFP expression was found in these cells. Reporter gene assay showed that Cx36promoter–pGL3 had strong promoter activity in PC12 cells. Using a newly characterized MsY3 antibody, we demonstrated MsY3 expression in mouse retina, and double immunofluorescence showed ZO–1/Cx36 and ZO–1/MsY3 co–localization. We used chromatin immunoprecipitation (ChIP) to identify a direct transcriptional target of MsY3 from mouse retina *in vivo*. One percent paraformaldehyde was used to cross–link protein–DNA complexes, and anti–MsY3 antibody was used to capture immunoenriched MsY3 genomic DNA transcriptional targets. After PCR with primers that encompassed the Cx36 promoter sequence containing inverted CCAAT, we found the Cx36 promoter to be one target for MsY3. Electrophoretic mobility shift assays (EMSA) showed that the Cx36 promoter containing the inverted CCAAT sequence has binding activity with a protein migrating at 30–40 kDa. Additional work including gel supershift, inverted CCAT box mutations, co–transfection experiments are in progress to investigate the role of MsY3 in the regulation of Cx36 promoter activity. This work was supported by grants from CIHR to J.I.N.

Citation:

X. Li, C. Ciolofan, C. Olson, J.I. Nagy, THE Y–BOX TRANSCRIPTION FACTOR 3 MSY3/ZONAB CO–LOCALIZES WITH CONNEXIN36 IN MOUSE RETINA AND IS TARGETED TO THE PROMOTER OF THE CONNEXIN36 GENE Program No. 731.8. 2005 *Abstract Viewer/Itinerary Planner*. Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: 788.18

Day / time: Tuesday, Nov. 15, 2:00 PM – 3:00 PM

Presentation Type: Poster

Presentation Location: Washington Convention Center – Hall A–C, Board # TT62

A BNIP3–ACTIVATED NEURONAL CELL DEATH PATHWAY IN STROKE

Z.Zhang; X.Ma; X.Yang; S.Zhang; J.Kong*

Human Anatomy and Cell Science, Univ. of Manitoba, Winnipeg, MB, Canada

Neuronal cell death in stroke occurs in apoptosis, necrosis and atypical apoptosis pathways. BNIP3, a member of the Bcl–2 family, has been shown to induce cell death through an atypical apoptosis mechanism that involves mitochondrial dysfunction but is independent of caspase activity. The BNIP3 gene contains a functional HIF–1–responsive element (HRE) and is potentially activated in hypoxia and other oxidative stress conditions. Here we show that BNIP3 is upregulated in ischemic rat brains and in cultured primary neurons exposed to hypoxia. Inhibition of BNIP3 expression by RNAi protected neurons from hypoxia–induced cell death. Our results also show that expression of BNIP3 in neurons resulted in mitochondrial release and nuclear translocation of endonuclease G. Nuclear translocation of EndoG was found in neurons exposed to hypoxia, and knockdown of BNIP3 expression by RNAi prevented hypoxia–induced EndoG translocation and neuronal cell death. Taken together, our results demonstrate that at least one form of neuronal cell death in stroke is activated by BNIP3 and mediated by EndoG. This novel cell death pathway may also play a role in neuronal cell death in other neurodegenerative diseases.

The study was supported by Canadian Institutes of Health Research and Heart and Stroke Foundation of Canada.

Citation:

Z. Zhang, X. Ma, X. Yang, S. Zhang, J. Kong, A BNIP3–ACTIVATED NEURONAL CELL DEATH PATHWAY IN STROKE Program No. 788.18. 2005 *Abstract Viewer/Itinerary Planner*. Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: 750.9

Day / time: Tuesday, Nov. 15, 1:00 PM – 2:00 PM

Presentation Type: Poster

Presentation Location: Washington Convention Center – Hall A–C, Board # DD3

FREQUENCY–CURRENT RELATIONSHIPS IN ANESTHETIZED AND DECEREBRATED RAT HINDLIMB α MOTONEURONS IN SITU

D.C.Button*; K.R.Gardiner; T.Marqueste; P.F.Gardiner

Physiol., Univ. of Manitoba, Winnipeg, MB, Canada

F–I relationships using 5–s up–down ramp current injections (RCI) have been determined previously in cat hindlimb α –Mns “in situ” and rat tail α –Mns “in vitro.” Such f–I

relationships have frequently demonstrated counter–clockwise hysteresis (higher frequencies on the descending vs. the ascending ramp), which may reflect the activation of voltage–dependent persistent inward currents (PICs) which underlie “bistable”

behaviour. The purpose of this study was to describe the f–I relationships of hindlimb α –Mns in anesthetized and decerebrated rats “in–situ.” Experiments were carried out on Sprague–Dawley rats (250 – 350 g) subjected to anaesthesia (ketamine/xylazine (KX))

or a precollicular decerebration (PD). Mns of the sciatic nerve were impaled via sharp glass microelectrodes and the f–I relationship was measured in response to 5–s RCI. The results show that in response to 5s RCIs, anaesthetized and decerebrated rat hindlimb α –Mns

could be categorized into 1 of 4 f–I relationship types: 1) linear, 2) adapting, 3) linear + sustained and 4) late acceleration, as reported by Bennett et al. (2001). Types 3 and 4 demonstrated lower threshold currents and lower firing frequencies (FF) at spike de–recruitment (SDR) compared to spike recruitment (SR). Type 1 and 2 Mns f–I relationships showed a linear relationship with overlying FFs and currents at SR and SDR, and a clockwise hysteresis, respectively. Mns of the same input resistance required 70% more current at SR when KX was used as anesthetic (14nA) than in PD preparations (6nA). The addition of pentobarbital to a KX cell demonstrating f–I type 3 properties caused it to gradually (over 1 hr) show f–I type 1 and 2 properties. Some α –Mns displayed

self–sustaining firing (SSF) during “ramp and hold” maneuvers. Although KX cells were

less excitable during 5s RCI at SR compared to PD, the SSF behaviour was not influenced, thus suggesting that use of this anesthetic in our future planned experiments to monitor changes in PICs following altered activity may be warranted.

Support Contributed By: CIHR and NSERC.

Citation:

D.C. Button, K.R. Gardiner, T. Marqueste, P.F. Gardiner. FREQUENCY–CURRENT RELATIONSHIPS IN ANESTHETIZED AND DECEREBRATED RAT HINDLIMB α

MOTONEURONS IN SITU Program No. 750.9. 2005 *Abstract Viewer/Itinerary Planner*. Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: 848.12

Day / time: Wednesday, Nov. 16, 11:00 AM – 12:00 PM

Presentation Type: Poster

Presentation Location: Washington Convention Center – Hall A–C, Board # L6

CHANGES IN SYNAPTIC MORPHOLOGY ARE ASSOCIATED WITH

CORTICAL LONG–TERM POTENTIATION IN THE ANESTHETIZED RAT

S.Connor1; P.T.J.Williams1,3; B.Armstrong4; T.L.Ivanco2; A.C.W.Weeks1*

1. Psychology, Nipissing Univ., North Bay, ON, Canada. 2. Psychology, Univ. of Manitoba, Winnipeg, MB, Canada. 3. Psychology and Neuroscience, Univ. of Lethbridge, Lethbridge, AB, Canada. 4. Psychology, Univ. of Toronto, Toronto, ON, Canada

Long–term potentiation (LTP) has been described in the sensorimotor cortex of freely moving rats. Changes in dendritic morphology and dendritic spine density have also been found in this region following the induction of LTP. The current research examined changes in synaptic number and ultrastructure associated with these dendritic changes. Acute LTP was induced over a 1 h period and the animals were sacrificed 2 h after the initial stimulation of the LTP group. Synapses were quantified by determining the total number of synapses per neuron, the number of excitatory and inhibitory contacts, number of synapses with different curvature subtypes, number of perforated synapses, and maximal synaptic length. This analysis revealed several changes in synaptic morphology of excitatory synapses but no overall increase in the number of synapses per neuron. Specifically, the induction of LTP was associated with an increased number of excitatory perforated and concave shaped synapses. No significant changes in synaptic length were observed. Further, increased numbers of perforated concave synapses were found to be significantly correlated with the degree of potentiation in LTP animals. These and previous results suggest similar synaptic changes in both the cortex and hippocampus during the early phases of LTP maintenance and distinct synaptic changes during later phases of LTP maintenance.

Support Contributed By: NSERC Canada to PTJW (USRA), TLI (grant), and ACWW (grant)

Citation:

S. Connor, P.T.J. Williams, B. Armstrong, T.L. Ivanco, A.C.W. Weeks. CHANGES IN SYNAPTIC MORPHOLOGY ARE ASSOCIATED WITH CORTICAL LONG–TERM POTENTIATION IN THE ANESTHETIZED RAT Program No. 848.12. 2005 *Abstract Viewer/Itinerary Planner*. Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: 865.3

Day / time: Wednesday, Nov. 16, 10:00 AM – 11:00 AM

Presentation Type: Poster

Presentation Location: Washington Convention Center – Hall A–C, Board # AA4

EFFECTS OF INTRATHECAL CHOLINERGIC DRUGS ON HINDLIMB LOCOMOTION IN THE FIRST 2 WEEKS AFTER A COMPLETE SPINALIZATION IN CATS

S.Rossignol^{1*}; J.Provencher¹; L.M.Jordan²

1. Physiology, Univ. Montreal Fac. Med., Montreal, PQ, Canada *2. Physiology, Univ. of Manitoba, Winnipeg, MB, Canada*

In cats, locomotion of the hindlimbs can be evoked by alpha-2 noradrenergic agonists (clonidine) soon after spinalisation at T13. Neither serotonergic nor glutamatergic drugs can trigger locomotion in that period. Cholinergic agonists can induce fictive locomotion in decerebrate cats. Given that cholinergic interneurons are present in the feline lumbar spinal cord, we studied the effects of cholinergic drugs on locomotor capabilities in the first two weeks after spinalisation. Two adult cats (4.9 and 5.3 Kg) were implanted chronically, under aseptic conditions, with electromyographic (EMG) electrodes in both hindlimbs and an intrathecal (i.t.) cannula. EMG and synchronized video recordings were obtained to document the normal locomotor pattern. Two weeks later, the spinal cord was sectioned at T13 and i.t. injections (bolus of 100 μ l) of the various drugs were made during the first two post-spinalisation weeks, a

period during which the cats either do not walk at all or perform limited stepping movements. Edrophonium (a cholinesterase inhibitor, 0.5–2 mM) given 5 days after spinalisation did not induce locomotion, neither did Acetylcholine (1 mM) given on the same day. However, clonidine (3.8 mM) given a few minutes later induced a well organized locomotor pattern indicating that the cats were capable of expressing locomotion. Carbachol (1 mM), a cholinergic agonist, increased the spinal excitability so that perineal stimulation led to prolonged hyperflexion of the limbs but only very occasional steps. The most surprising result, in both cats, was that the muscarinic antagonist atropine clearly enhanced the quality, the vigour and the speed range of spinal locomotion at day 12, at a time when only some irregular stepping had appeared. It is concluded that the cholinergic system may play a role in the recovery of locomotion after a spinal section and that muscarinic (and perhaps nicotinic) blockade might enhance spinal locomotion. *Support Contributed By: CIHR and CR Chair on Spinal Cord*

Citation:

S. Rossignol, J. Provencher, L.M. Jordan. EFFECTS OF INTRATHECAL CHOLINERGIC DRUGS ON HINDLIMB LOCOMOTION IN THE FIRST 2 WEEKS AFTER A COMPLETE SPINALIZATION IN CATS Program No. 865.3. 2005 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: 964.13

Day / time: Wednesday, Nov. 16, 1:00 PM – 2:00 PM

Presentation Type: Poster

Presentation Location: Washington Convention Center – Hall A–C, Board # N6

CX36 REGIONAL AND DEVELOPMENTAL EXPRESSION IN MAMMALIAN BRAIN AND SPINAL CORD

C.O.Olson^{*}; J.I.Nagy

Physiology, Univ. of Manitoba, Winnipeg, MB, Canada

Gap junctions composed of connexin36 (Cx36) mediate electrical coupling between neurons in mammalian CNS via the formation of electrical synapses. The global occurrence of electrical synapses in mammalian CNS as reflected by the distribution of Cx36 was investigated in mouse and rat CNS by immunofluorescence microscopy using several affinity-purified monoclonal and polyclonal anti-Cx36 antibodies generated against different epitopes in Cx36. The absence of Cx36 immunolabeling in Cx36 knockout mice was used to confirm antibody specificity. We also examined the chemical neurotransmitter phenotype of neurons expressing Cx36. Further, we determined the regional and developmental extent of Cx36/ZO-1 interaction in mammalian CNS. Cx36 was detected throughout adult brain and various rostro-caudal levels of spinal cord. Immunolabeling of Cx36 revealed patterns of isolated puncta occurring at different densities in various CNS regions. In addition, puncta localized to neuronal proximal dendrites and somata were observed in the lateral vestibular nucleus and in spinal cord motor nuclei. Higher levels of Cx36 were detected throughout the CNS at early postnatal ages, but patterns of immunolabeling were similar to that found in adult CNS. In all age groups studied, a proportion of Cx36 immunopositive puncta were found to be co-localized with immunolabeling of vesicular glutamate transporter 2. By confocal microscopy and co-immunoprecipitation analysis, ZO-1 was found in association with Cx36 at neuronal gap junctions in adult spinal cord as well as many CNS regions during development, thus extending previous observations of Cx36/ZO-1 interaction in adult brain. Our results demonstrate the widespread occurrence of electrical synapses in adult and developing mammalian CNS as inferred by Cx36 immunolabeling. The association of Cx36 with glutamatergic synapses in brain and spinal cord provides support for the prevalence of mixed synapses in mammalian CNS. *Support Contributed By: CIHR grants to JIN and MHRC Studentship to CO*

Citation:

C.O. Olson, J.I. Nagy. CX36 REGIONAL AND DEVELOPMENTAL EXPRESSION IN MAMMALIAN BRAIN AND SPINAL CORD Program No. 964.13. 2005 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: 900.6

Day / time: Wednesday, Nov. 16, 9:00 AM – 10:00 AM

Presentation Type: Poster

Presentation Location: Washington Convention Center – Hall A–C, Board # SS2

MILD ISCHEMIA PRODUCES INFARCT BUT NOT REORGANIZATION

K.Hartle^{1,2,3}; T.L.Ivanco^{1,2,3*}

1. Psychology, 2. Centre on Aging, 3. MICH, Univ. of Manitoba, Winnipeg, MB, Canada

Transient ischemic attacks do not appear to induce a severe deficit and very little is understood about the physiological consequences of this type of insult. Since the nature of the episode is temporary and symptoms have often disappeared by the time the individual is seen by a physician, efforts must be made to understand what the impact of these attacks has on the brain. Disappearance of the overt symptoms does not mean that the brain has escaped without damage. The purpose of this study was to investigate the consequences a mild ischemic episode has on the brain. Rats underwent a photochemically-induced stroke within the right motor cortex. To induce damage, animals were anesthetized, and placed in a stereotaxic frame where the skull was exposed. A fiber optic bundle with an aperture of 1.5 mm was positioned over the primary motor cortex. The animal then received an intravenous injection of Rose Bengal in physiological saline over 1–2 minutes. For 20 minutes, a high wattage, cold light source was used to illuminate the brain through an intact skull. Control animals underwent the same procedure, except the light was not turned on. Twenty days after surgery brains were removed and processed using the Golgi-Cox method. Neurons of layer II and layer V from the damaged hemisphere and the homotopic region on the contralateral hemispheres were drawn with a microscope equipped with a special drawing tube. Sholl analysis was used on the cell drawings from both the damaged and intact hemispheres. Results showed there were no differences in dendritic length in layer II cells between experimental and control animals. There was a trend indicating that layer V cells had less dendritic length in experimental animals, but this was not statistically significant. These findings are important for increasing our understanding of how the brain responds to a mild stroke and why profound behavioral deficits are not always present with this type of injury. *Support Contributed By: CFI, MIF, NSERC, University of Manitoba, The UofM Centre on Aging, Carsen Group, and MicroBrightField, Inc*

Citation:

K. Hartle, T.L. Ivanco. MILD ISCHEMIA PRODUCES INFARCT BUT NOT REORGANIZATION Program No. 900.6. 2005 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2005. Online.

Program Number: 965.19

Day / time: Wednesday, Nov. 16, 3:00 PM – 4:00 PM

Presentation Type: Poster

Presentation Location: Washington Convention Center – Hall A–C, Board # P8

CONNEXIN36-CONTAINING GAP JUNCTIONS IN OFF AND ON SUBLAMINAE OF THE INNER PLEXIFORM LAYER OF ADULT RODENT RETINA

N.Kamasawa¹; K.G.V.Davidson¹; C.Furman²; J.A.Sampson¹; T.Yasumura¹; J.I.Nagy³;

J.E.Rash^{1*}

1. Biomedical Sciences, Colorado State Univ., Fort Collins, CO, USA *2. Anatomy and Neurobiology, Southern Illinois Univ., Carbondale, IL, USA* *3. Physiology, Univ. of Manitoba, Winnipeg, MB, Canada*

Gap junctions are essential in initial processing of visual information in the retina. Confocal immunofluorescence microscopy and freeze-fracture replica immunogold were used to identify Cx36 containing gap junctions in adult rodent retina under various physiological and experimental conditions. In inner plexiform layer (IPL) of retina, Cx36 was found in >1400 gap junctions in both OFF and ON sublamina. “Plaque” gap junctions predominated, while “string” and “ribbon” gap junctions were mostly restricted to the OFF sublamina. Plaque junctions were subdivided into “regular” and “irregular” subtypes, consisting of crystalline hexagonal arrays vs. irregular clusters, with packing densities (Pd) of connexons (number per unit area) were Pd=1 and Pd=1.7 respectively. String gap junctions consisted of curvilinear strands that were one (occasionally two) connexons wide and up to 150 connexons long. Ribbon gap junctions consisted of multiple strands of curvilinear rows that were two or three connexons wide. To compare the “area of influence” of each type of gap junction, we calculated its “dispersion coefficient” (Dc). Gap junctions were delineated using the smallest ellipse that would completely enclose all connexons, number of connexons was counted, and the area of the ellipse was measured. The density of connexons in “regular plaque” gap junctions was ca 15,000/ μ m² and was defined as minimally dispersed, yielding a “dispersion coefficient” of Dc = 1. Dc varied almost 30 fold, ranging from Dc = 1 (regular plaque gap junction) to Dc = 30 (string gap junction). These data suggest either reversible dynamic stages (“plasticity”) of gap junctions during different physiological activities and/or conditions of anesthesia, or the existence of different but stable morphologies of gap junctions in the ON vs. OFF sublaminae. *Support Contributed By: NIH and CIHR*

Citation:

N. Kamasawa, K.G.V. Davidson, C. Furman, J.A. Sampson, T. Yasumura, J.I. Nagy, J.E. Rash. CONNEXIN36-CONTAINING GAP JUNCTIONS IN OFF AND ON SUBLAMINAE OF THE INNER PLEXIFORM LAYER OF ADULT RODENT RETINA Program No. 965.19. 2005 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2005. Online.