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EDITOR

99th Annual Meeting

April 12-13, 2007

Minot, North Dakota

EDITOR'S NOTES

HISTORY

The *Proceedings of the North Dakota Academy of Science* (NDAS) was first published in 1948, with Volume I reporting the business and scientific papers presented for the 40th annual meeting, May 2-3, 1947. Through Volume XXI**,** the single yearly issue of the *Proceedings* included both abstracts and full papers. Commencing with Volume XXII, the *Proceedings* was published in two parts: A, published prior to the annual meeting, contained an abstract of each paper to be presented at the meeting, and B, published later, contained full papers by some of the presenters.

In 1979 (Vol. 33) the *Proceedings* changed to an 8½ x 11-inch format. Produced from camera-ready copy submitted by authors, it was distributed at the annual meeting. Commencing with Vol. 51 submissions were on computer disk; the *Proceedings* was then assembled with desktop publishing software. This approach allows the Editor to format papers so as to assure the *Proceedings* a consistent look. This method also produces an electronic copy of the *Proceedings;* the Secretary-Treasurer has the capability to generate electronic copies of past issues.

VOLUME 61 ORGANIZATION

In 2003 the NDAS council voted to accept all abstracts scheduled for presentation at the Annual Meeting. Thus, communications in volumes 58 to present haven't undergone a "typical" peer review. Rather, they provide an accurate reflection of the material presented before the NDAS membership at the Annual Meeting. The presentations in this year's *Proceedings* are presented in three major sections. The first contains the undergraduate communications presented as part of the A. Rodger Denison Student Research Competition. The second section comprises the graduate Denison Competition papers, and the final section comprises professional communications presented by faculty members of the Academy. Readers may locate communications by looking within the major sections of these *Proceedings (see table of contents)* or by referring to the author index on page 91

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SYMPOSIA COMMUNICATIONS

Commencing with the 88th Annual Meeting [Vol. 50], Symposia presenters at annual meetings have had opportunity to contribute full-length articles or multiple-page contributions, thus providing much greater depth and coverage than that ordinarily possible. Speakers have presented educationally-oriented lectures and workshop discussions, and have still provided rigorous, more technical professional papers to the *Proceedings.*

COLLEGIATE AND PROFESSIONAL COMMUNICATIONS

Each Collegiate and Professional presentation at the annual meeting is represented by a Communication. Designed as more than a typical abstract but less than a full paper. Communications report results and conclusions, and permit sharing of important data and conclusions. Crucially, they provide for timeliness and ease of production.

CONSTITUTION AND BYLAWS

This issue of the Proceedings also contains the Constitution and Bylaws of the Academy, a list of officers and committee members. We're working on maintaining a list of dues-paying members of the Academy (we'd appreciate your help in building and adding to this list with names of new and prospective members), a listing of past presidents of the Academy, and an index of presenters and paper authors. Copies of the financial statement and the unapproved minutes from last year's annual business meeting will be available at the meeting as appendices A & B, respectively.

IN APPRECIATION

The Academy wishes to acknowledge current and emeritus members of the Academy who continue to support the mission of the North Dakota Academy of Science Research Foundation through their special gifts. A listing of these supporters will accompany the Financial Report. The Academy also wishes to express its thanks to the presenters of papers at the Annual meeting, the session chairs, as well as all who have helped in organizing spaces and places, soliciting manuscripts, and compiling of this year's communications. The President of the Academy also wishes to sincerely thank Dr. Peter Minorsky who served as honored guest speaker at this year's meeting.

Christopher Keller	Siegfried Detke
President	Secretary-Treasurer, *Proceedings* Editor

TABLE OF CONTENTS

UNDERGRADUATE COMMUNICATIONS

IN THE

A ROGER DENISON COMPETITION

SCHEDULE OF PRESENTATIONS

Undergraduate talks will be in the "media center" one the ground floor of the Gordon B. Olson Library – session will be chaired by A. Rodger Denison Competition judges

MORNING SESSION

7:30 Registration desk open

8:20 Greetings from President Christopher Keller

8:40 MICROWAVE-ASSISTED SYNTHESIS OF N-VANILLYLFORMAMIDE. Mikhail M. Bobylev[1)] and Brent D. Keller*

9:00 PLANT COMMUNITY DYNAMICS IN ONE METER PLOTS SURROUNDING THE WESTERN PRAIRIE FRINGED ORCHID (PLATANTHERA PRAECLARA) OVER A THREE-YEAR PERIOD. Amanda Bryson* and Bonnie Alexander

9:20 EFFECT OF CHLOROPHENOXY HERBICIDE ON DEVELOPMENT AND GROWTH OF *DROSOPHILA MELANOGASTER* DURING MULTI GENERATIONAL EXPOSURE. Heidi M. Gienger*, Bridget M. Blunck, Hilde E. van Gijssel

9:40 ALPHA-2 ADRENERGIC RECEPTOR INHIBITION OF HIPPOCAMPAL CA3 NETWORK ACTIVITY: AGONIST STRUCTURE-ACTIVITY RELATIONSHIPS. Brianna L. Goldenstein*, Ke Xu, Kristan M. Green, Jacquline A. Pribula, Jasmine J. O'Brien, Kylie L. Davis, Sarah J. Boese, Jessica A. Lichter, Brian W. Nelson, Melissa N. Austreim, James E. Porter, Van A. Doze

10:00 AN INITIAL STUDY OF THE INFLUENCE OF MAGNESIUM ION ON THE FLUORESCENCE LIFETIME OF NADH BOUND TO ALDH2[†]. Jeffrey R. Hovde*[‡], Thomas P. Gonnella[‡], Matthew J. Picklo[§], and Khwaja G. Hossain[‡]

10: 20 BREAK

10:40 EFFECT OF 2,4-DICHLOROPHENOXYACETRIC ACID ON CELL PROLIFERATION OF HUMAN LUNG FIBROBLASTS. Erica L. Jorde*, Bridget M. Blunck and Hilde E. van Gijssel,

11:00 ALPHA-2A ADRENERGIC RECEPTOR ACTIVATION INHIBITS EPILEPTIFORM BURST DISCHARGES IN THE RAT HIPPOCAMPAL CA3 REGION. Brian W. Nelson*, Kylie L. Davis, Ke Xu, Jasmine J. O'Brien, Jacquline A. Pribula, Sarah J. Boese, Jessica A. Lichter, Brianna L. Goldenstein, Chris W.D. Jurgens, Melissa N. Austreim, James E. Porter, Van A. Doze

11:20 DNA CLEAVAGE IN VITRO BY DNA TOPOISOMERASE II IN THE *AF4* GENE TRANSLOCATION BREAKPOINT REGION. Ashley L. Olander *, Cheryl A. Lepp, Heidi J. Super

11:40 ENDOCRINE DISRUPTING EFFECTS OF ATRAZINE ON THE *DROSOPHILA MELANOGASTER.* Tiffany J. Ost* and Andre W. DeLorme

12:00 LUNCH (served in the Olson Library Media Center)

AFTERNOON SESSION

1:00 LIFE HISTORY OF THE TIGER SALAMANDER, *AMBYSTOMA TIGRINUM*, IN NORTHWEST NORTH DAKOTA. Michael Poitra[1], Kenneth C. Cabarle[1], Dwight K. Blackhawk[2], F. Drew Henry[1], Judd Entzel[1] and Christopher K. Beachy[1]

1:20 TRANSIENT RECEPTOR POTENTIAL CANNONICAL -1 IS ASSOCIATED WITH LIPID RAFT DOMAINS OF SUBMANDIBULAR GLAND CELLS. Kristina Rauser, Biswaranjan Pani, Brij B. Singh

1:40 DEVELOPING HYBRID PEPPERS: *CAPSICUM ANNUUM* L. 'HUNGARIAN HOT WAX' X *CAPSICUM ANNUUM* L. 'EARLIEST ACE SWEET' FOR ZONE 3B HARDINESS. Jennifer R. Robinette*, Deborah M. DeMarey and Frank Kutkla

2:00 DISRUPTION OF BEAN ROOT CELL MEMBRANE POTENTIALS BY HYDROQUINONE. Joshua E. Seil*, Morgan L. Grundstad, Christopher P. Keller

2:20 TESTING THE EFFECTS OF ATRAZINE ON THE FLAT-HEADED MAYFLY (*Stenacron sp.)* Rachel M. Stack* and Andre W. DeLorme

2:40 BREAK

3:00 THE EFFECT OF LYCOPENE ON THE EXPRESSION OF CONNEXIN MRNAS AND PROTEINS. Samuel Sticka*, Josh Seekins, Tracy Greff, Erin Rice, Jill McLain, William Wolf, and Lynn Burgess

3:20 POPULATION ANALYSIS OF UNIONID BIVALVES IN THE SHEYENNE RIVER. Trevor J. Tompkins* and Andre W. DeLorme

3:40 BURROWING IN THE CORAL COBRA (*ASPIDELAPS LUBRICUS).* BobbiRae Wickum, Brock Thuen, Alexandra Deufel

4:00 CHARACTERIZATION OF ADRENERGIC RECEPTOR SUBTYPES IN RAT HIPPOCAMPAL NEURONS USING REAL-TIME SINGLE CELL RT-PCR. Ke Xu*, Chris W.D. Jurgens, Kristin L. Hillman, James E. Porter, Van A. Doze

4:30 Business meeting (open to all members) – location to be determined

EVENING

6:00 Banquet will be at the Taube Museum of Art, 2 Main St in downtown Minot. Cash bar open at 5.

PLANT COMMUNITY DYNAMICS IN ONE METER PLOTS SURROUNDING THE WESTERN PRAIRIE FRINGED ORCHID (*PLATANTHERA PRAECLARA*) OVER A THREE-YEAR PERIOD

Amanda Bryson* and Bonnie Alexander

Department of Biology, Valley City State University, Valley City

The western prairie fringed orchid (*Platanthera praeclara*) is North Dakota's only federally listed threatened plant. This plant inhabits wetland swales in the Sheyenne National Grassland (SNG) in southeastern North Dakota. The SNG is managed by the United States Forest Service who has been given the charge by the United States Fish and Wildlife Service to protect and maximize the reproduction and growth of this threatened orchid on federal lands. The SNG is grazed (cattle), mowed, and burned for management on a regular basis. Due to the need to protect and preserve this remnant orchid population, the identification of viable orchid habitat in the SNG is a high priority.

Managers and researchers have described western prairie fringed orchid habitat using major vegetation types observed in association with the orchid. Wolken (1) actually correlated specific vegetation with orchid location in an effort to use it as an indicator of available orchid habitat. Observations by Alexander (personal communication) indicated that vegetation in close association with the orchid appears to change from year to year. This study was undertaken in an effort to examine the vegetation surrounding flowering orchids in order to identify orchid habitat and/or to rule out ways in which it cannot be identified. Objectives were to: 1) identify and characterize the plant community immediately surrounding flowering orchids over a 3-year period; 2) identify and characterize changes in this plant community between years; 3) answer the question of whether orchid habitat can be accurately identified by using the immediate surrounding plant community.

We looked at: species diversity, changes in perennial vs. annual makeup, change in upland vs. wetland species, and shifts in amount of exposed ground in 1-Meter plots surrounding flowering orchids. Of the 254 orchid locations observed in 2002 only 35 flowering orchids returned above ground in 2003 and zero returned in 2004. New flowering orchids were observed in lower elevations in the wetland swales in 2003 and at even lower elevations in 2004. Orchid locations from 2002 showed a shift from 83 to 100 percent perennial plants suggesting the presence of the orchids in a more stable plant community in 2004. The percentage of bare ground in plots which contained orchids in 2002 was reevaluated in 2004 even though orchids were no longer present in any of the plots. That data showed an increase from 3.9% bare ground in 2002 to 10.6% in 2004. This shift verified the observation that the 2002 orchid locations had significantly dried out by 2004 destroying 6.7% of the vegetation and probably caused the elimination of all above-ground flowering orchids. When comparing flowering orchid sites from 2002 to those of 2004 a similar more exaggerated trend was observed. A change from 3.9% bare ground in 2002 to 26.3% in 2004. Observations noted that this change illustrated not a drying out of the habitat but the presence of new flowering orchids in newly created habitat. Standing water was observed in 2002 where flowering orchids were found growing in 2004.

Further research will include the mapping (Arcview desktop software) of the orchid population's movements over the three year period. An analysis of test well water levels in the habitat will also be examined and correlated with flowering orchid position in the habitat each year.

1) Wolken PM (1995) Habitat and life history of the western prairie fringed orchid (*Platanthera praeclara*), MS thesis. University of Wyoming, Laramie; 93 p.

EFFECT OF CHLOROPHENOXY HERBICIDE ON DEVELOPMENT AND GROWTH OF *DROSOPHILA MELANOGASTER* DURING MULTI GENERATIONAL EXPOSURE.

Heidi M. Gienger*, Bridget M. Blunck, Hilde E. van Gijssel

Toxicology Laboratory, Science Department, Valley City State University, Valley City ND, 58072

Recent studies have shown an increase in both respiratory and circulatory birth defects in wheat growing counties in the U.S. The chance of birth defects increases even further when babies (especially boys) are conceived during the application season of herbicides. It has been suggested that exposure to chlorophenoxy herbicides is responsible for the increase in birth defects, but as of yet the mechanism is unknown. Chlorophenoxy herbicides such as 2,4-Dichlorophenoxyacetic acid (2,4-D), and 2-Methyl-4-chlorophenoxyacetic acid (MCPA), have been used since the 1940's, and multiple generations have been exposed. Therefore, we hypothesize that the risk of birth defects increases due to exposure of multiple generations. The *Drosophila melanogaster* is used as a model organism to study the effect of multigenerational exposure, because of its short generation time (2 weeks) and genetic homology with humans. The goal of our experiment is to determine if multi-generational exposure to chlorophenoxy herbicides changes the growth and development of the *D. melanogaster*.

Drosophila embryos were collected after a pre-lay period to ensure embryos of equal age. Embryos were added to a vial containing food with chlorophenoxy herbicides in the following concentrations (1µM and 3mM 2,4-D or MCPA and control). Development was observed every 24 hrs for a period of 16 days. Concurrently after the *Drosophila* adults were used to collect embryos, adults (75 females and 50 males) were put into bottles with food containing the same concentrations as above to raise enough flies to produces the next generations. After 7 days the parents were removed and stored in freezer for future experiments. After 14 days we collected the new generation and proceeded as described above. Development and growth were followed for 4 generations.

Currently, data is available for the 1st and 2nd generation. Results show a clear delay in development in subsequent generations of flies exposed to 3mM 2,4-D and MCPA during their lifetime. In control bottles the new generation can be collected after 15 days. In the bottles with 3 mM MCPA and 2,4-D very few adults were present on days 15. It also reduced the fertility of the females and fewer adults appeared in each bottle and fewer embryos could be collected. These effects were not present in flies exposed to 1 µM of 2,4-D and MCPA. No obvious delay in development was visible and fertility does not seem to be affected. Exposure to 2,4-D and MCPA increase the number of males being produced. The male/female ratio increased from 0.72 (control) to 1.42 (MCPA 1µM, $p < 0.05$) in the 2nd generation. This increase was not visible after the exposure of the first generation. An increase in the male/female ratio was also seen after exposure to 2,4-D but this was not significant.

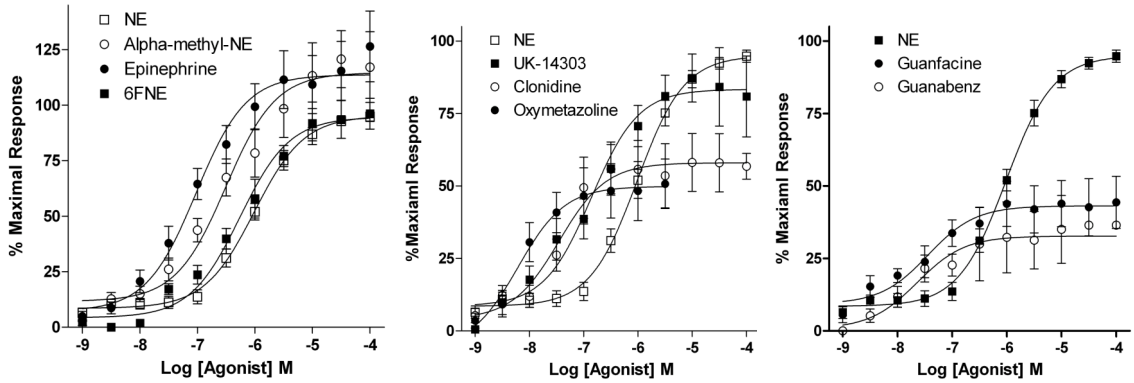
Our data shows that long term multigenerational exposure affects the development of the *D. melanogaster*. It also showed that the effects increased in every generation. Further studies are needed to determine which pathways and genes that are involved. A gene-array experiment is planned after the forth generation to identify likely candidates. Because metamorphosis is hormonally regulated these results support the hypothesis that chlorophenoxy herbicides exhibit endocrine disrupting effect.

ALPHA-2 ADRENERGIC RECEPTOR INHIBITION OF HIPPOCAMPAL CA3 NETWORK ACTIVITY: AGONIST STRUCTURE-ACTIVITY RELATIONSHIPS

Brianna L. Goldenstein*, Ke Xu, Kristan M. Green, Jacquline A. Pribula, Jasmine J. O'Brien, Kylie L. Davis, Sarah J. Boese, Jessica A. Lichter, Brian W. Nelson, Melissa N. Austreim, James E. Porter, Van A. Doze

Department of Pharmacology, Physiology & Therapeutics, School of Medicine & Health Sciences, University of North Dakota, Grand Forks, ND 58203

Alpha-2 adrenergic receptors (ARs) are important regulators of many physiological processes (1). Although many AR ligands have been characterized on peripheral ARs, little is known about their actions in central nervous system. This study is focused on generating functional agonist data for alpha-2 AR which mediates the antiepileptic effects of the endogenous neurotransmitter norepinephrine (NE) in the hippocampus (2). We hypothesized that among the different types of agonists tested, the performance of catecholamines on alpha ARs will be higher than that of the synthetic compounds of the imidazoline and guanidine chemical classes because most of the catecholamines tested were endogenous or derivatives of the endogenous neurotransmitters in the brain. Using extracellular field potential recordings in the hippocampal cornu ammonis 3 (CA3) region of rat brain slices, agonists of the various chemical classes described above were studied, and their antiepileptiform actions recorded and analyzed. The variations of the potency and efficacy of the chemicals are taken into consideration along with the parameters of their structural differences. Agonist classifications (full *vs.* partial) were based on their relative efficacy to the endogenous neurotransmitter norepinephrine, with having a >80% relative efficacy considered being a full agonist. The results indicated that the potency rank order of catecholamines was epinephrine > 6-fluoronorepinephrine = alpha-methyl-norepinephrine > norepinephrine, with all of them being full agonists (see figures below). In contrast, among the imidazoline and guanidine agonists (rank order potency was dexmedetomidine > oxymetazoline > clonidine > UK-14303 and guanabenz > guanfacine, respectively, see figures below), only UK-14303 was a full agonist. These results confirm our initial hypothesis that the catecholamines would perform better than imidazolines and guanidines, and that the catecholamine compounds collectively were more efficacious. One interesting finding of our results is that although the imidazolines and guanidines were partial agonists, their potency are significantly higher than that of the catecholamines. This makes intuitive sense because they are man-made ligands that were initially developed based on their binding affinities. Since the differences of the structures between catecholamines were trivial while the structural differences among each class of agonist were significant, conclusions can be drawn that structural attributes of the catecholamines contribute to their efficacy and potency at inhibiting hippocampal CA3 epileptiform activity.



1) Pupo AS and Minneman KP (2001) CNS Spectr. 6, 656.
2) Giorgi FS, Pizzanelli C, Biagioni F, Murri L and Fornai F (2004) Neurosci. Biobehav. Rev. 28,507.

Acknowledgments: Supported in part by North Dakota EPSCoR through NSF grant EPS-0447679 (VAD), NSF CAREER award 0347259 (VAD), and NIH COBRE program grant 5P20RR017699 (VAD, JEP).

AN INITIAL STUDY OF THE INFLUENCE OF MAGNESIUM ION ON THE FLUORESCENCE LIFETIME OF NADH BOUND TO ALDH2[†]

Jeffrey R. Hovde*[‡], Thomas P. Gonnella[‡], Matthew J. Picklo[§], and Khwaja G. Hossain[‡]

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Within many mammalian systems aldehyde dehydrogenases (ALDH's) convert toxic aldehydes into more manageable carboxylic acids. Nicotinamide adenine dinucleotide (NAD^+) is an essential coenzyme for this enzymatic process to occur. Through the course of a five step reaction scheme[1], NAD^+ is converted to its reduced form (NADH) and released. The release of NADH from the ALDH is necessary for further turnover of the enzyme. The addition of magnesium ions to these ALDH systems modulates activity, in some cases increasing activity (ALDH2) and in others, like ALDH1, decreasing activity. The specific role of the magnesium ions the reaction scheme of the different ALDH's is in the process of being resolved.[2]

Our research is focused on using the intrinsic fluorescence of NADH to provide fundamental information regarding enzyme-cofactor interactions. By applying time resolved fluorescence spectroscopy we have been able to distinguish between free NADH in solution (ϑ = 0.4 ns) and NADH bound to recombinant rat ALDH2 (ϑ = 6.5 ns) in the presence of magnesium ions. With such as dramatic difference in fluorescence lifetimes, we plan to determine the extent to which the fluorescence lifetime and dissociation constant for NADH is affected by changes in the structure of the enzyme (through site-directed mutagenesis) and the presence/absence of Mg^{2+} ions. Other experiments will utilize aldo-keto reductases that utilize NADPH as a cofactor.

[†] This research was supported by the NIH grants P20 RR016741 (INBRE) and P20 RR17699-01 (COBRE) from the NCRR.

[‡] Division of Science and Mathematics, Mayville State University

[§] Department of Pharmacology, Physiology, and Therapeutics, University of North Dakota

[1] Hammen, P. K.; Allali-Hassani, A.; Hallenga, K.; Hurley, T.D.; Weiner, H. *Biochemistry* **2002**, *41*, 7156.

[2] Ho, K. K.; Hurley, T.D.; Weiner, H. *Biochemistry* **2006**, *45*, 9445.

EFFECT OF 2,4-DICHLOROPHENOXYACETRIC ACID ON CELL PROLIFERATION OF HUMAN LUNG FIBROBLASTS.

Erica L. Jorde*, Bridget M. Blunck and Hilde E. van Gijssel

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An increased number of birth defects have been found in areas of high wheat production and are believed to be caused by the chlorophenoxy herbicide 2,4-Dichlorophenoxyacetic acid (2,4-D) (1,2). 2,4-D is classified as an endocrine disrupter, which means it can affect hormones levels and influence metabolism(1). Residues of 2,4-D are detectable in urine and semen samples of men who apply the herbicide, due to inhalation. The goal of our experiment is to study the effect of 2,4-D on proliferation of human lung fibroblasts and determine the effect of 2,4-D on molecular pathways involved in proliferation.

Human lung fibroblasts were exposed to 1µM, 3µM, 10µM, 30µM, and 100µM of 2,4-D for 24, 48 and 72 hrs. Three controls were used: no cells, no ethanol or solvent solution, and only ethanol or solvent solution. The experiment was repeated 3 times. Cells were seeded on day -1 and left to attach and settle for 24 hrs. 2,4-D was added at day 0. After 24 hr (day 1), 48 hr (day 2) or 72 hr (day 3), 70µL of MTT reagent was added to each well and plates were returned to the incubator. After 4 hr, 700µL of MTT detergent was added to the wells and the plates were placed in a dark area overnight. Proliferation was measured by UV/VIS spectrometer at a wavelength of 570nm. Proliferation was normalized to the rate of proliferation in samples exposed to solvent solution only; proliferation in these wells was set to be 100%.

The first experiments showed that proliferation was affected by ethanol used as a solvent for 2,4-D. Therefore for subsequent experiments, a solvent solutions was used which contained less ethanol (3%) and 1% Tween-80 in water. Cell proliferation was not affected after 24 hr of exposure of 2,4-D. After 48 and 72 hr of 2,4-D exposure, proliferation in cells exposed to 1 and 3 µM 2,4-D increased to a maximum of 130% compared to proliferation of cells exposed to solvent solution only. This effect was more pronounced in cells exposed for 48 hr to 2,4-D compared to cells exposed for 72 hr. Proliferation in cells decreased to 97% in cells exposed to the higher concentrations.

Our experiments shows that 2,4-D has an effect on proliferation. The effect was most pronounced after longer exposures. To our surprise lower concentrations seem to stimulate growth where as higher concentrations block proliferation. The decrease in proliferation is most likely not due to cell death because no floating cells or cell parts could be seen in the medium. More experiments need to be done to exclude cell death. The dual effect of 2,4-D on proliferation (increased proliferation at lower concentrations and decreased proliferation at high concentrations) is a phenomenon seen more often with hormones. The mechanism is not yet fully understood. The results, however, support our hypothesis that 2,4-D is an endocrine disruptor and may be capable of disrupting lung development during embryonic development.

References:

1) 2,4-D. (2004). *chemicalWATCH Factsheet*. Retrieved February 26, 2007, from http://www.beyondpesticides.org/pesticides/factsheets/24D_Jul04.pdf

2) Schreinemachers, D. M. (2003). Environ Health Perspect, 111, 1259.

ALPHA-2A ADRENERGIC RECEPTOR ACTIVATION INHIBITS EPILEPTIFORM BURST DISCHARGES IN THE RAT HIPPOCAMPAL CA3 REGION

Brian W. Nelson*, Kylie L. Davis, Ke Xu, Jasmine J. O'Brien, Jacquline A. Pribula, Sarah J. Boese, Jessica A. Lichter, Brianna L. Goldenstein, Chris W.D. Jurgens, Melissa N. Austreim, James E. Porter, Van A. Doze

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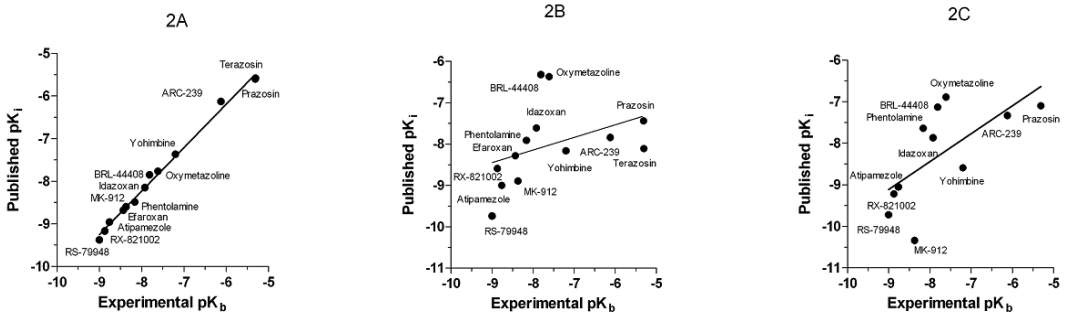
Norepinephrine has potent antiepileptic properties, the pharmacology of which is unclear. Under conditions where GABAergic inhibition is blocked, norepinephrine (NE) has been shown to reduce hippocampal CA3 epileptiform activity through α_2 adrenergic receptor (AR) activation on pyramidal cells (1). Characteristic of the CA3 region is a dense recurrent network among the excitatory pyramidal neurons (2), which is believed crucial for performing these cognitive functions (3). However, the immense connectivity of the CA3 axon collaterals makes this region extremely vulnerable to overexcitation (4). Therefore, it is crucial to understand the pharmacology of NE inhibition of CA3 network activity in order to gain insight about its role in controlling the network activities. In this study, we addressed this problem by investigating which α_2AR subtype(s) mediate the antiepileptic effect of NE. Our preliminary data of α_{2}AR mRNA expression characterization using real time reverse-transcriptase polymerase chain reaction (RT-PCR) on the hippocampal CA3 region have shown that α_{2A}AR was the predominate subtype, also some α_{2C}AR population was present but no α_{2B}AR genomic expression was detected. This led us to hypothesize that α_{2A}AR may be the subtype which primarily mediates the antiepileptic effect of NE in rat hippocampus. However, mRNA expression does not necessarily dictate the presence of ARs in the cells. To test this hypothesis, hippocampal CA3 epileptiform activity was examined using field potential recordings in brain slices. First, the selective αAR agonist 6-fluoronorepinephrine (2) caused a reduction of CA3 epileptiform activity, as measured by decreased frequency of spontaneous epileptiform bursts. In the presence of βAR blockade, concentration-response curves for AR agonists suggest that α_{2}ARs mediate this response, which reaffirms our initial assumption. Finally, equilibrium dissociation constants (K_b) of selective αAR antagonists were functionally determined to confirm the specific α_{2}AR subtype mediating CA3 epileptiform activity inhibition. Apparent K_b values calculated for RS-79948 (1.0 nM), RX-821002 (1.3 nM), atipamezole (1.7 nM), efaroxan (3.7 nM), MK-912 (4.8 nM), phentolamine (6.9 nM), idazoxan (12 nM), BRL-44408 (15 nM), oxymetazoline (25 nM), yohimbine (63 nM), ARC-239 (76 nM), prazosin (4900 nM) and terazosin (5000 nM) correlated best with affinities previously published for the α_{2A}AR subtype (r = 1.00, slope = 1.01, see figure below), while the correlation with the α_{2B} (r = 0.39, slope = 0.32) and α_{2c} (r = 0.66, slope = 0.67) AR subtypes were not significant. These results indicate that, under conditions of impaired GABAergic inhibition, activation of α_{2A}ARs is primarily responsible for the antiepileptic actions of NE in the hippocampal CA3 region, which confirms our initial hypothesis.



1) Jurgens CWD, Boese SJ, King JD, Pyle SJ, Porter JE and Doze VA (2005) Epilepsy Res. 66, 117.
2) Li XG, Somogyi P, Ylinen A and Buzsaki G (1994) J. Comp. Neurol. 339, 181.
3) Traub RD, Wong RK, Miles R and Michelson H (1991) J. Neurophysiol. 66, 635.
4) Schwartzkroin PA (1986) *in* Advances in Neurology, vol 44, Basic Mechanisms of the Epilepsies (Delgado-Escueta AV ed), New York: Raven Press, pp. 991.

Acknowledgments: Supported in part by North Dakota EPSCoR through NSF grant EPS-0447679 (VAD), NSF CAREER award 0347259 (VAD), and NIH grant 5P20RR017699 from the COBRE program (VAD).

DNA CLEAVAGE IN VITRO BY DNA TOPOISOMERASE II IN THE *AF4* GENE TRANSLOCATION BREAKPOINT REGION

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Objective: The mixed-lineage leukemia gene, (*MLL*), fuses to one of >50 different translocation partner genes in several subtypes of de novo human acute leukemia. All breaks in *MLL* occur within the same 8 kilobase (kb) region, (bcr). The most prevalent *MLL* translocation partner is *AF4*. *MLL* fusions are also observed chemotherapy-related, secondary leukemias. These *MLL* fusions often follow treatment with drugs which interfere with DNA topoisomerase II (topo II) activity and implicate topo II in the translocation mechanism, in vivo.

In vitro studies show the *MLL* 8 kb translocation bcr is cleaved in a restricted region in the presence of common topo II inhibiting drugs. A second gene, *AF9* which is a common *MLL* partner gene in secondary leukemias also shows sensitivity to cleavage by topo II in the translocation break region. Although the *AF4* gene is the most common translocation partner gene of *MLL*, the *MLL-AF4* fusion is rare in secondary leukemias and this gene has not been tested for cleavage in the presence of topo II inhibitors.

In the present study we have tested the *AF4* bcr for sensitivity to cleavage by topoisomerase inhibitors. Evidence of cleavage in *AF4* would support a common mechanism in *MLL* translocations in de novo leukemias and therapy-related, secondary leukemias, involving cleavage by topo II. Lack of cleavage may suggest a unique translocation mechanism for de novo leukemias, which does not involve topo II.

Methods: A cell-free in vitro assay was used to test for cleavage of the *AF4* bcr with a known topo II inhibitor, etoposide. Plasmid DNA from the cloned *AF4* bcr, the *MLL* bcr, and a control plasmid (pRYG) was incubated at 37 degrees C with etoposide in the presence of purified human topoisomerase II. Cleavage products were recovered after SDS/Proteinase K treatment. Cleavage was assessed by agarose gel electrophoresis.

Results: Cleavage was noted in the control pRYG plasmid which has a previously described single strong topo II cleavage site. Etoposide cleaved both the *MLL* bcr plasmid and the *AF4* bcr plasmid at a single site as both plasmids showed linear forms after incubation with etoposide and topo II. The level of cleavage was etoposide dose-dependent. Cleavage was not seen in the absence of etoposide or topo II or with DMSO solvent.

Conclusions: The *AF4* bcr we have cloned appears to have a single topo II cleavage site. Since *MLL-AF4* fusions are the most common type of *MLL* fusion, but rarely occur in treatment-associated secondary leukemias, we propose that topo II may function aberrantly to induce breaks in the *AF4* gene which initiate rearrangement with *MLL* in leukemia de novo. Experiments are underway to map the site of cleavage in the *AF4* bcr clone.

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ENDOCRINE DISRUPTING EFFECTS OF ATRAZINE ON THE *DROSOPHILA MELANOGASTER*

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Introduction

Atrazine has been one of the most widely used herbicide in the United States and also has been the most frequently detected pesticide in ground and surface water. While originally considered to be nontoxic, recent work has shown atrazine to cause endocrine disruption in a variety of animals (1). We would like to examine whether atrazine has an endocrine disrupting effect that would affect the normal rate of insect development. The *Drosophila melanogaster* (fruit fly) is an organism with a well documented and short life cycle that is conducive for testing in a controlled environment. On a molecular level, the fruit fly is well known and thoroughly documented. For these reasons, the fruit fly was used to determine whether or not atrazine had any effect.

Methods

Two experimental trials were done and for both we used five experimental concentrations of atrazine and three controls. Atrazine was dissolved in acetone and brought to a desired concentration before placing it in the vial where it would mix in with a certain amount of food. Given that acetone was used to dissolve the atrazine, acetone was used in the controls as well. The atrazine concentrations used for the first experiment were 0.1, 1.0, 3.0, 10, and 30mM. In the second experiment the concentrations were kept within a narrower range of 0.1, 0.5, 1.0, 2.0, and 3.0mM. At the start of the experiment, 30 to 70 embryos were placed in each vial. The vials were checked daily to observe any growth or development from embryos to larvae, larvae to pupae, and pupae to adult. Once emergence to adulthood was reached, the date was recorded, the fly would be immediately removed followed by sex determination, and would then be placed into another vial containing 70% alcohol for later evaluation.

Results

In both trials, an evident delay, in comparison with the controls was seen in the 1.0mM concentration. A delay of three days, compared to that of the controls, was seen in both pupae formation and adult emergence in the 1.0mM. As for concentrations of 2.0 and 3.0mM, larvae were seen, but never reached the pupae stage of development and in the 10 and 30mM the embryo stage was never surpassed. The developmental times for the concentration of 0.1mM matched the controls. When examining the male to female ratios, there may be dominance in male adult emergence over female. In the controls the male to female ratios were 1: 1.11 and 1: 1.62. That ratio was 1.06:1 in the 0.1mM concentration, 1.42:1 in the 0.5mM concentration, and 2.11:1 in the 1.0mM concentration.

Discussion

The endocrine system works by dictating growth and development with the use of hormones. Development in fruit flies from one life stage to the next is strictly regulated by hormones. With this in mind, it is quite possible that the effect that atrazine has shown on the fruit fly, a definite delay, could be caused by interference with the endocrine system. An interesting phenomenon with endocrine disruptors is that they can sometimes have a greater influence at lower concentrations. One example is the leopard frog where there was an effect even at the low concentrations of 0.1 ppb (1). This is comparable to nanomolar concentration. For future testing, we will examine lower concentrations of atrazine to see if we see an endocrine disrupting effect.

References

1. Hayes T, Haston K, Tsui M, Hoang A, Haeffele C, Vonk A. (2003) Atrazine-Induced Hermaphroditism at 0.1 ppb in American Leopard Frogs (*Rana pipiens*): Laboratory and Field Evidence. Environ Health Perspect 111:568-575.

LIFE HISTORY OF THE TIGER SALAMANDER, *AMBYSTOMA TIGRINUM*, IN NORTHWEST NORTH DAKOTA

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The tiger salamander (*Ambystoma tigrinum*) complex is widespread across the continental United States, north into Canada, and south into Mexico. Three groups exist: the California form (now identified as *A. californiense*), an eastern North American form (*A. t. tigrinum*) that is essentially found east of the Mississippi River, and a west/central group that is made of up the rest of the members including the Mexican assemblage (1, 2). The eastern group has a life history that includes an obligate metamorphosis, the Mexican assemblage contains species that are obligately paedomorphic (i.e., fail to metamorphose and become reproductively mature while retaining larval morphology), and the west/central group has populations that can vary from metamorphic to facultatively paedomorphic. While a large number of life history observations have been published (3), there is not a thorough life history study that reports most of the essential life history parameters for a single population.

In order to establish the life history of the tiger salamander in northwest North Dakota, we established four study populations. Two of the sites were equipped with drift fences. In addition to sampling every migrating salamander at drift-fenced sites, we also conducted survey collections from all four sites, using a variety of techniques. Our 2006 data included approximately 650 captures. All captures were measured for mass, length (SVL), morphological status (larval or transformed), maturation status (if possible), and sexed (if possible). Animals collected from one site were returned to the lab, killed, preserved and dissected. This allowed for large and sequential samples whereby the above data were positively confirmed (Fig. 1).

Like the rest of the west/central forms, tiger salamanders in North Dakota grow extremely rapidly. Maturation can occur as larvae or after metamorphosis, and the frequency of paedomorphosis is associated with the permanence (i.e., depth) of the pond. In addition, paedomorphic larvae retain the ability to metamorphose. Males more frequently become reproductively mature as larvae than do females. Maturation appears to be possible in the second summer of life (for larvae) or later (for larvae and metamorphs). Metamorphosed juveniles also seem to return to the ponds for reasons other than courtship.

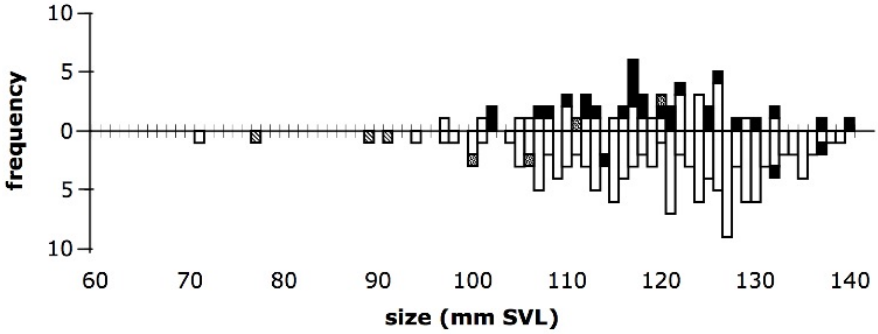

Fig. 1. – Size-frequency histogram of Ambystoma tigrinum collected in early June 2006 from Swalls Lake, Ward County, North Dakota. Bars above x-axis are males, bars below are females. Open bars are juvenile larvae, black bars are mature larvae, hatched bars are metamorphosed juveniles, shaded bars are metamorphosed adults.

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Sources:

1) Shaffer, H.B., and M. McKnight. 1996. Evolution 50:417-433.
2) Irschack, D.J., and H.B. Shaffer. 1997. Herpetologica 58:30-49
3) Petranka, J. 1998. Salamanders of the United States and Canada. Smithsonian Institution Press, Washington, DC, USA

TRANSIENT RECEPTOR POTENTIAL CANNONICAL -1 IS ASSOCIATED WITH LIPID RAFT DOMAINS OF SUBMANDIBULAR GLAND CELLS

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Calcium is a ubiquitous signaling molecule that regulates a spectrum of cellular events ranging from cell proliferation to cell death. In order for calcium to participate in cellular signal transduction processes, it has to be brought into the cell form the extracellular environment. Recently, mammalian homologues of drosophila Transient Receptor Potential (TRP) proteins have been suggested as critical plasma membrane calcium channels. Transient Receptor Potential Cannonical-1 (TRPC1) is the founding member of TRPC sub-family, is known to be expressed ubiquitously and is a prime a store operated plasma membrane calcium channel that regulates calcium entry into the cell. Plasma membrane TRPC1 becomes active upon depletion of endoplasmic reticulum (ER) calcium stores. In spite of the universal expression pattern of TRPC1, its precise localization on plasma membrane and mechanism(s) of activation has not been elaborately defined. Here, using a Human submandibular gland (HSG) cell culture model and mouse salivary gland tissue, we demonstrate the association of TRPC1 with distinct compartments of the plasma membrane called lipid rafts. Lipid rafts are heterogeneous, small (10-200nm) membrane microdomains that are enriched cholesterol and serve as a signaling platform for a multitude of cellular processes. Lipid rafts from HSG cells and mouse salivary gland tissues, were isolated using discontinuous sucrose density gradient ultracentrifugation method. Lipid rafts were characterized by determining the cholesterol content of the different fractions isolated from the density gradient ultracentrifugation process and the proteins were analyzed by western blotting. We identify TRPC1 association with lipid raft domains of HSG cell line and mouse salivary gland tissue. We also demonstrate that the activation of store operated calcium entry (SOCE) via TRPC1 in these cells is dependant on the integrity of lipid raft microdomains. Upon disruption of these domains with cholesterol sequestering drugs like methyl-beta-cyclodextrin and Fillipin-III, a significant reduction in SOCE was observed. Hence, we hypothesize that activation of SOCE attributed to TRPC1 in submandibular gland cells is dependant on its lipid raft localization.

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DEVELOPING HYBRID PEPPERS: *CAPSICUM ANNUUM* L. 'HUNGARIAN HOT WAX' X *CAPSICUM ANNUUM* L. 'EARLIEST ACE SWEET' FOR ZONE 3B HARDINESS

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Southwestern North Dakota and eastern Montana is a region that has a short growing season with severe temperature fluctuation seasonally as well as daily. The growing degree days are limited by this regional location. Peppers require 3 to 4 months of frost-free weather to produce good yields. Typically the last frost-free date in this region is approximately the 15th of May. A killing frost may occur as early as late August or early September. Based on sales from surrounding business and nurseries, people prefer the California Wonder pepper in this region due to the regions' environmental conditions. California Wonder pepper plant has a maturation period of 69 to 80 days.

The purpose of this research is to develop a hybrid pepper plant that has an earlier maturation period, increased hardiness within the local region, and produce reasonable yield than the peppers currently grown in garden settings within the North Dakota and Montana region.

A Hungarian Hot Wax pepper plant and an Earliest Ace Sweet pepper plant were crossed by sex reversal pollination, meaning the pollen from the male Hungarian Hot Wax was put onto the stigma of the female Earliest Ace Sweet and the pollen from the male Earliest Ace Sweet was put onto the stigma of the female Hungarian Hot Wax. This sex reversal pollination of the two plants was done to see if the resulting hybrid plant would demonstrate characteristics of both species, be hardy in zone 3b (North Dakota and eastern Montana), and produce a pepper of culinary quality. The Hungarian Hot Wax pepper plant grows 36 to 48 inches in height and produces fruit that turns yellow at 60 maturation days and red at 85 maturation days. The Earliest Ace Sweet pepper plant grows 24 to 30 inches in height and produces fruit that turns green at 50 maturation days and red at 65 maturation days.

Seeds from the F1 hybrid cross were started in a greenhouse, transplanted at seedling stage, and at 12 weeks they were distributed to volunteers in the surrounding community to be planted in backyard gardens. According to standard practice, pepper seeds need to be planted 8 to 12 weeks indoors before being transplanted into a garden. The greenhouse setting provided the conditions necessary for successful germination of the seeds and establishes seedlings up to the 12 week growth stage.

Based on results, this hybrid cross grew to maturity, produced fruit, and seed. A majority of the plants grew tall and spindly which resembled the characteristics of the Hungarian Hot Wax pepper plant. Most of the fruit produced were banana shaped and very hot which are also characteristics of the Hungarian Hot Wax pepper plant. Few of the peppers demonstrated characteristics of the Earliest Ace Sweet pepper plant in that the fruit had three to four lobes, were shorter in length, and displayed a box-like shape. The plants showed hardy traits because even though they were infested with aphids and whitefly, they continually produced new growth and still produced fruit at maturation.

Continued research has revealed that the F1 generation plants not only produced fruit and seed from both the sex reversal pollinated plants, but that the seeds are viable. Future research will include distribution of the F2 generation plants into the surrounding community gardens to determine if the plants will grow to maturity, produce fruit, and F3 generation seed.

DISRUPTION OF BEAN ROOT CELL MEMBRANE POTENTIALS BY HYDROQUINONE

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Hydroquinone (HQ) is a phenolic compound synthesized by certain plants. Secreted into the soil, HQ elicits phytoxic responses in some other plants. For example, leafy spurge (*Euphorbia esula*) exposed experimentally to millimolar concentrations of HQ had reduced water uptake by leaves, resulting in closed stomata and decreased photosynthesis (1). The mechanism, however, by which HQ disrupts water uptake is not understood. Our hypothesis is that HQ disrupts mineral nutrition and, as a consequence, water uptake by osmosis in the roots. Poor water status in the roots subsequently starves the leaves of water. Disruption of root mineral nutrition can be monitored by recording the membrane potential of root cells. Previously Mazurek et al. (2) found that the common bean, *Phaseolus vulgaris*, is highly sensitive to HQ with concentrations of as little as 10 µM HQ significantly inhibiting plant growth. Preliminary results suggested an effect of HQ on bean root cell membrane potentials (3). The current project attempted to confirm the earlier results and to establish the sensitivity of bean root cell membrane potentials to HQ across a range of concentrations.

Bean root tips, approximately 8-10 days old, were incubated in 0.1 mM KCl, 1 mM CaCl2, and 1 M Mes/BTP pH 6.0 for a minimum of 1 hour. After mounting the root in a perfusion chamber, the roots were exposed to the same medium. The root was than pierced with a microelectrode filled with 1 M KCl to monitor the cell membrane potential. Potentials were monitored by an electrometer (Electro 770; World Precision Instruments) and recorded by a chart recorder (Linseis, L200E). After a stable membrane potential recording was established of at least -100 mV for ten minutes, the root was exposed to HQ solutions of various concentrations for a minimum of 60 minutes or until failure of the recording.

Membrane potentials responded to HQ exposure by an initial hyperpolarization (0-6 mV), followed by a larger depolarization (0-65 mV). The root cell membrane response to HQ was dose responsive, with the degree of depolarization larger when exposed to higher concentrations of HQ. Even at very low concentrations (micromolar), membrane potentials showed significant sensitivity. This dose-response indicates that depolarization of bean root cell membrane potentials by HQ represents an overall reduced chemiosmotic gradient in the root cells, which in turn should result in reduced osmosis and water uptake by the plant. Reducing the amount of water uptake by the root cells will result in reduced amounts of water transport to leaves, closure of stomata, and an overall decrease in photosynthesis and plant injury.

1) Barkosky RR, Butler JL, Einhellig FA *J. Chem. Ecol*. 25 (7): 1611-1621, 1999.
2) Mazurek SA, Barkosky RR, Keller CP Proc. North Dakota Acad. Sci. 58: 41, 2004.
3) Mazurek SA, Grundstad ML, Seil JE, Barkosky RR, Keller CP Proc. North Dakota Acad. Sci. 59: 21, 2005.

TESTING THE EFFECTS OF ATRAZINE ON THE FLAT-HEADED MAYFLY (*Stenacron sp.)*

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INTRODUCTION: Atrazine, (2-chloro-4-(ethylamine)-6-(isopropylamine)-s-triazine), is the most widely used corn herbicide in the U.S. according to the Environmental Protection Agency. Atrazine works by binding to plastoquinone-binding protein in photosystem II, inhibiting electron transport and eventually killing the plant. It is thought to be an endocrine disrupter in a variety of animals; this is especially true for aquatic organisms because most herbicides end up in rivers and streams. Our lab is investigating the possible endocrine disrupting effects on aquatic insects. We chose the flat-headed mayfly because as a larva it lives in the water and when it turns into an adult it emerges out of the water. Being that it is an aquatic species, they are more sensitive to toxins in the water because of their gills being a point of absorption. The mayfly has incomplete metamorphosis, meaning that it does not have a pupae stage and it also can have from forty to forty-five molts before it turns into an adult. The molts are controlled by hormones, hormones which we believe the atrazine will have an effect on. Another reason we chose the mayfly for these experiments is the fact that it is one of the most abundant aquatic species and is a good representative of the North Dakota rivers and streams.

MATERIALS AND METHODS: Experiments were run using three to six ten-gallon fish tanks, each tank contained ten liters of water and an assortment of rocks and sticks. The water used to fill the tanks was river water taken from the same site that we collected the insects. The rocks and sticks were collected from the river and reused in each experiment. For every set up there was a tank with just water in it, a tank with the dissolving agent used and in the third and fourth tanks there were concentrations of atrazine. The dissolving agents used were methanol in the first four experiments and acetone in the fifth experiment. We used an artificial light set up giving the mayflies sixteen hours of light and eight hours of dark. Tanks were checked daily and newly emerged adults were removed, sexed and measured. Enzyme-Linked ImmunoSorbent Assay (ELISA) was run twice a week to determine the concentrations of atrazine in each of the tanks. Water quality measurements of temperature, pH, percent dissolved oxygen, specific conductivity, turbidity, and dissolved oxygen were taken once a week with a Eureka multiprobe. Algae were taken from the tanks when the experiment was over and mounted on slides for later analysis.

RESULTS: There was a definite delay in the emergence time in both the five parts per billion (ppb) and the twenty ppb tanks compared to the control. For example, after two months in the most recent experiment, twenty out of twenty-five adults had emerged from the control tank, eight had emerged from the five ppb tank, and only four had emerged from the twenty ppb tank. The tanks with just water and the water and acetone were both very close to each other in emergence times. In all of the experiments run, there is a general decrease in the amount of atrazine present. For example, after about four months the twenty ppb tank was down to about ten ppb, and the five ppb tank was down to around one ppb. The water quality tests stayed steady throughout the experiments. In the first four experiments when methanol was used there seemed to be a delay in the methanol tanks also, so the methanol may have had an effect on the mayfly emergence as well. The male to female ratio in the control tank was 1:1.75, in the five ppb tank is was 1:3.5, and in the twenty ppb tank it was 1:2. The average length of adult in the control tank was 10.287mm with a standard deviation of 0.808. The average length of adult in the five ppb tank was 10.880mm with a standard deviation of 0.801. The average length of adult in the twenty ppb tank was 9.522mm with a standard deviation of 0.634.

DISCUSSION: Our experiments show that we can successfully raise mayfly larva in ten gallon tanks. In many of the experiments, the survival rate was eighty to one hundred percent. In this last experiment we saw a distinct delay, which is what we hypothesized would happen. We need to do more experiments with acetone as a solvent because is seemed to not have any type of effect on the results of the emergence of the adults. We also need to look further into the amount of food available in each tank or the lack of food. It is possible atrazine could be depleting algae populations in the tanks. The fact that there might not have been enough food in the atrazine tanks could have had an effect on the emergence time of the adults.

THE EFFECT OF LYCOPENE ON THE EXPRESSION OF CONNEXIN MRNAS AND PROTEINS

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Epidemiological studies have shown that dietary levels of lycopene and consumption of its sources is inversely related to the incidence of prostate, bladder, pancreas, stomach, and lung cancers. Our hypothesis is that lycopene's anticarcinogenic properties are due to its ability to enhance Gap Junctional Communication (GJC) between adjacent cells by increasing the available connexin proteins. It has been shown that normal cells use GJC to suppress adjacent cancer cells from proliferating by maintaining intercellular signaling.

The following human cell lines: DU-145, prostate adenocarcinoma; A549, lung carcinoma; HS-578T, breast carcinoma; and IMR-90, lung fibroblast; were seeded and grown to confluency. Then media with lycopene and 1% acetone was added at a dose of 10^{-10} M to 10^{-5} M. Cells were incubated for 48 hours and RNAs or proteins were isolated from each flask. Reverse transcriptase was used to obtain cDNA from the mRNA and then connexin expression was quantified using QPCR. Standard PCR was used to determine which connexin mRNAs were expressed prior to the use of QPCR. Total protein concentration of the cell lysate was determined by BCA and by using Indirect ELISA, the protein concentration of each connexin and dosage was determined.

Our QPCR data showed a dose dependent increase in connexin mRNA expression for: 31.9, 32, and 43 for DU-145; and 31.9 for HS-578T. No dose dependent change in mRNA expression was observed for the following connexins: 40 for DU-145; 32 and 43 for A549; 30, 32, 43, and 45 for HS-578T; and 32, 43 and 45 for IMR-90.

ELISA has confirmed dose dependent increases in connexin 26 in DU-145; connexin 26, 40, and 45 in A549; connexin 32, 40, 43, and 45 in HS-578T; and connexin 45 in IMR-90. No dose dependent change in protein levels was observed for the following connexins: 32, 40, 43, and 45 for DU-145; 32 and 43 for A549; 26 and 43 for HS-578T; 26 and 40 for IMR-90. A dose dependent decrease in protein levels was observed in connexins 32 and 43 for IMR-90.

Our data supports our hypothesis that lycopene enhances GJC in some cell lines. It also appears that it suppresses GJC in nonepithelial cells. These effects appear to occur at the physiological dose. Our research is ongoing.

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POPULATION ANALYSIS OF UNIONID BIVALVES IN THE SHEYENNE RIVER

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INTRODUCTION – Populations of freshwater mussels have been declining tremendously in North America over the past 30 years. Currently 55% of the taxa in this group are recognized as extinct, endangered, or threatened, compared to 7% of the continents birds and mammals (1). This decline in population is largely due to loss of suitable habitat. This is significant because mussels act as filter feeders and are important parts of aquatic ecosystems. As for North Dakota, there have been few studies done on the status of freshwater mussels with the last study being done in 1993 (2). This study showed that the most abundant species of mussels found in the Sheyenne River were the Threeridge, Fatmucket, Wabash Pigtoe, and Giant Floater. Other species found included: Cylindrical Papershell, Mapleleaf, Pocketbook, Black Sandshell, Creek Heelsplitter, Pink Heelsplitter, and White Heelsplitter. The authors of this study noted specific concern for the Threeridge, Wabash Pigtoe, and Mapleleaf due to declines in their population from the previous study and concerns about their ability for long-term maintenance of a viable population. The goal of our study is to sample part of the Sheyenne River and compare our findings to this previous study.

METHODS – Sampling was done in one location along the river below the Katherine Dam. The collection of mussels was done by sampling three 1 m^2 quadrates. All mussels, living and dead, in each quadrate were collected by hand and placed in a bucket to be sorted by species. Collection by hand was possible because the river was shallow enough to reach the bottom with an arms length. Once the mussels were sorted into species groups, measurements to the nearest mm were taken for length, height, and thickness of the shell. After measurements were taken, all the living mussels were returned to the river. The dead shells collected in each section were brought back to the lab and similar measurements were also taken on them.

RESULTS - With our study we found the following species: Threeridge, White Heelsplitter, Fatmucket, Wabash Pigtoe, Black Sandshell, Giant Floater, Pocketbook, and Pink Heelsplitter. The most abundant of these species that we found were the Threeridge (found in densities of 21.3/m^2), Fatmucket (24/m^2), and Wabash Pigtoe (21.6/m^2). We had a sample site density of 97.3 mussels/m^2 which is extremely high when compared to the previous studies 7.2/m^2. Our length measurements showed that, as with the previous study there were few young mussels collected. One point of interest when comparing the number of live Threeridge shells found with the number of dead shells found was that although the Threeridge was one of the most abundant species found with 64 total, there were only 7 dead ones found. A similar trend was found with the Black Sandshell which we found 24 total living but none dead. The opposite was found with the Giant Floaters which there were 28 living mussels found and 66 total dead.

DISCUSSION - Our study was similar to the 1993 study in that the most abundant species found in both studies were the Threeridge, Fatmucket, and Wabash Pigtoe. The concerns that were felt about the Threeridge and the Wabash Pigtoe appear to be unjustified as their populations seem to be prosperous. The Mapleleaf was also a species of concern and it should be noted that none of these were found. Our findings on the Black Sandshell are significant because the 1993 study only found .08/m^2 compared to our 8/m^2. This shows a significant increase in their population. Overall, the mussel populations in this section of the Sheyenne seem to be flourishing when compared to the data from the last study. Some notable absences from our study were the Mapleleaf, Cylindrical Papershell, and Creek Heelsplitter but this could be because of the lack of sites sampled.

REFERENCES

1. Master, L. 1990. The imperiled status of North American aquatic animals. Biodiversity Network News 3:1-2, 7-8.

2. Jensen, William F. et al. Distribution, Relative Abundance, and Species Diversity of Freshwater Mussels in the Sheyenne and Red Rivers of Eastern North Dakota. North Dakota Fish and Game Department, Div. Rpt. 42. 20 pp. August, 2001

BURROWING IN THE CORAL COBRA (*ASPIDELAPS LUBRICUS)*

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Introduction:

This study investigates the mechanics of burrowing in coral cobras (*Aspidelaps lubricus)*. *Aspidelaps lubricus* is found in southern Africa in a semi-arid desert climate. It is a small snake that emerges usually at night to forage. Their dramatic coloration of orange to coral red with black bands and a yellowish belly makes them easy to distinguish. Recent research has found that *Aspidelaps scutatus* (shield nose cobra), a close relative of *A. lubricus*, has a kinetic skull with a mobile shovel-like snout and burrows using muscles generally used for prey capture and transport. In contrast, most other elongated vertebrates whose burrowing mechanics have been studied, Uropeltids, Amphisbeanians, and Caecilians, all share the characteristics of a compact skull and specialized skull shapes for penetrating and pushing aside substrate.

Methods and Materials:

Burrowing trials were conducted using a ten gallon tank filled with about 10 cm of substrate (washed sand). We conducted numerous trials with two specimens, a male and a female, using both dry and moist sand as our substrate. Before placing *Aspidelaps lubricus* in our controlled environment we determined the soil humidity. We recorded 22 trials, each lasting around thirty minutes or more, with written and visual data using a Sony camcorder. We attempted burrowing with the male three times in dry sand and twice with the female in dry sand. We noticed a lot of poking around in the dry sand, but it was too weak to hold a tunnel. We then switched our substrate to moist sand. We did five trials with each snake in the moist sand. We also attempted a different method in hopes of provoking burrowing. We placed a wooden platform beneath the soil line and enclosed a mouse. We tried this approach three times with the male and four times with the female. A dissection of an *Aspidelaps lubricus* was also conducted to compare its anatomy with *A. scutatus*.

Results:

In the first two or three trials the cobras did not burrow or even attempt to burrow into the substrate. After the cobras became more familiar with the tank and substrate we began to see occasional burrowing activity. Burrowing attempts were rare, but when they happened we noticed a few key mechanics that *A. lubricus* used for penetrating and moving soil. These include a scooping action using the anterior 1/3 of the body, side to side motion of the head, and also a forward pushing motion. Trials that included prey elicited more frequent attempts at burrowing.

The anatomy of *A. lubricus* differs from that of *A. scutatus* in some aspects. *A. lubricus* has the typical kinetic skull of most snakes, but without the enlarged premaxilla seen in *A. scutatus*. We found that a medial slip of the retractor pterygoidii muscle, which in *A. scutatus* attaches to the large rostral scale, in *A. lubricus* is attached to the anterior medial portion of the maxilla. This medial slip is entirely absent in other cobras.

Conclusions:

Aspidelaps lubricus has a kinetic skull like other snakes and its relative *A. scutatus*, not a solid skull like other elongate vertebrates whose burrowing mechanics have been studied. There was no evidence of the use of the rostral scale for burrowing, which was different from *A. scutatus. Aspidelaps lubricus*'s muscle does not insert on the rostral scale or premaxilla, it inserts on the anterior medial portion of the maxilla, meaning that it can not swivel its snout like *A. scutatus*.

Aspidelaps lubricus does not seem to want to burrow nearly as readily as *A. scutatus*, but definitely shows some desire to shelter. The snakes are usually hidden under newspaper in their tanks. They may burrow more readily in a substrate that is comparable to their natural habitat or use the burrows made by other animals (sheltering).

In hopes of solving the *A. lubricus* burrowing mystery we will try a few new ideas. We noticed more success in moist substrates so we hope to try a few different substrates such as potting soil or wet beach sand. We noticed a scooping pattern when a burrow was already started so we may try a substrate that will hold a good tunnel. In the next attempt we will also place the platform which encloses the mouse at a larger depth below the surface. In our first attempts it seemed too easy for the snakes to retrieve the prey and no real burrowing occurred.

CHARACTERIZATION OF ADRENERGIC RECEPTOR SUBTYPES IN RAT HIPPOCAMPAL NEURONS USING REAL-TIME SINGLE CELL RT-PCR

Ke Xu*, Chris W.D. Jurgens, Kristin L. Hillman, James E. Porter, Van A. Doze

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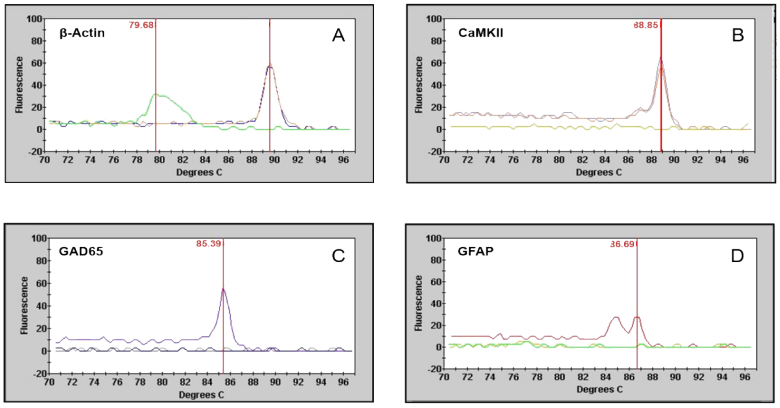
Adrenergic receptors (ARs) are important regulators of many physiological processes. The activation of αARs in the rat hippocampus reduces epileptiform burst activity in the CA3 region while βAR stimulation enhances burst frequency (1). Currently, three β, three α_1 and three α_2AR subtypes exist with distinct genomic sequences. In this study, we characterized the localization of each AR subtype in the pyramidal cells and interneurons of the rat hippocampus using real time single cell RT-PCR, which allows the detection of multiple transcripts within a single cell sample. The selection of genomic contamination-free cellular samples was based on the expression of house-keeping gene β-actin and the lack of genomic marker STS ET3. Cell origin was confirmed by the presence of mRNA transcripts for: calcium/calmodulin-dependent protein kinase-II (CamK-II) for excitatory pyramidal cells, glutamate decarboxylase (GAD65) for inhibitory interneurons and glial fibrillary acidic protein (GFAP) for astrocytic cells (see Figure). We hypothesize that each cell type will have differential expression of various ARs in different regions of the rat hippocampus. If so, this could help explain why the endogenous neurotransmitter norepinephrine (NE), which activates all AR subtypes, enhances long-term potentiation (2) and consequently learning and memory (3), while still having potent epileptic effects (1). Because the localization of different types of ARs among different cell types across the hippocampus allows NE to activate multiple pathways at the same time, NE is able to have multifaceted actions in the hippocampus. Preliminary results indicated that the pyramidal cells in the hippocampal CA3 region expressed mainly α_{2A} and α_{2C}ARs, as gene-specific amplification demonstrated that of the 25 samples tested, 12 cells expressed α_{2A}AR transcript, with three of the 12 cells additionally expressing mRNA for α_{2C}AR subtype, and no cells possessing α_{2B}AR mRNA. Pyramidal cells in the CA1 region predominately expressed β_2 and β_1ARs, with all 17 samples tested expressing β_2AR mRNA transcript and 4 samples additionally expressing β_1AR transcripts. In contrast, hippocampal CA1 interneurons in stratum oriens expressed α_{1A}AR transcript in six out of ten cells tested, with three of them also expressing α_{1B}AR mRNA, and an additional three interneurons expressing α_{1A}AR mRNA only. Similarly, the interneurons from the CA3 region also only expressed α_{1A}AR and/or α_{1B}AR transcripts in five out ten samples. Finally, interneurons in stratum lacunosum-moleculare of the hippocampal CA1 region appear to express α_{2A}AR and/or α_{2C}AR mRNA only. These results support our hypothesis that the molecular foundation of ARs expressed in the rat hippocampus is highly differentiated among cell types and regions of the hippocampus. These findings should help narrow future functional studies on the physiological effects of these ARs in the hippocampus.



1) Jurgens CWD, Boese SJ, King JD, Pyle SJ, Porter JE and Doze VA (2005) Epilepsy Res. 66, 117.
2) Hopkins WF and Johnston D (1988) *J. Neurophysio.l* 59, 667.
3) Murchison CF, Zhang XY, Zhang WP, Ouyang M, Lee A and Thomas SA (2004) Cell 117, 131.

Acknowledgments: Supported in part by North Dakota EPSCoR through NSF grant EPS-0447679 (VAD), NSF CAREER award 0347259 (VAD), and NIH grant 5P20RR017699 from the COBRE program (VAD, JEP).

MICROWAVE-ASSISTED SYNTHESIS OF N-VANILLYLFORMAMIDE

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The Leuckart reaction is a fast and convenient method of synthesis of various formamides and amines and has been successfully used for the synthesis of many biologically active compounds, including agrochemicals and pharmaceuticals. Surprisingly, it has never been used for the synthesis of vanillylamine (I) from vanillin (II) via the intermediate N-vanillylformamide (III). In the literature, the intermediate III is described only once, when it was obtained via the formylation of I, not by the reductive amination of II. In this work, we report a successful synthesis of III from II via a microwave assisted Leuckart reaction. This method provides a new synthetic pathway to I, which is a valuable intermediate in the synthesis of a number of biologically-active compounds. This method can also be used for the synthesis of other hydroxy-substituted benzylamines. The project is supported by NIH grant P20 RR016741 from the NCRR.

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GRADUATE COMMUNICATIONS

IN THE

A ROGER DENISON COMPETITION

SCHEDULE OF PRESENTATIONS

Graduate talks will be in "Study 2" room on the ground floor of the Gordon B. Olson Library – session will be chaired by A. Rodger Denison Competition judges

MORNING SESSION

7:30 Registration desk open

8:20 Greetings from President Christopher Keller

8:40 AN ACTIVITY-DEPENDENT CHANGE IN CILIARY NEUROTROPHIC FACTOR EXPRESSION IN THE RAT MAGNOCELLULAR NEUROSECRETORY SYSTEM. Jason Askvig* and John Watt

9:00 ROLE OF THE C3 TRANSIENT RECEPTOR POTENTIAL CHANNEL IN RAT SUPRA OPTIC NUCLEUS. Sunitha Bollimuntha*, John A Watt, Brij B Singh

9:20 ADSORPTION DYNAMICS OF ALKANES ON CARBON NANOTUBES: A MOLECULAR BEAM SCATTERING STUDY. S. Funk*, U. Burghaus

9:40 N-/ISO-BUTANE ON ANATASE (001) THIN FILM: A SURFACE CHEMISTRY STUDY. J. Goering*, E. Kadossov, U. Burghaus

10:00 BREAK

10:20 ADRENERGIC RECEPTOR MEDIATED SUPPRESSION OF SEIZURES: α_2 ADRENERGIC RECEPTORS INHIBIT EPILEPTIFORM ACTIVITY VIA A SYNAPSE SPECIFIC MECHANISM. Chris WD Jurgens* and Van A Doze

10:40 α_{1A} ADRENERGIC RECEPTOR REGULATION OF SEIZURES & NEURODEGENERATION. Jessica A. Lichter[a]*, Christopher A. Knudson[b], Chris W.D. Jurgens[a], Patrick A. Carr[b], Dianne M. Perez[c], Van A. Doze[a]

11:00 RECRUITING AMERICAN INDIAN PARTICIPANTS FOR A GENETIC EPIDEMIOLOGIC STUDY. Melanie A. Nadeau*, Lyle G. Best

11:20 A NEW ASSAY FOR THE QUANTIFICATION OF BIOFILM. Preeti Sule, Nathan J Carr, Birgit M Prüß

12:00 LUNCH (served in the Olson Library Media Center)

AFTERNOON SESSION

1:00 NON – FLAGELLAR PHENOTYPES OF *YERSINIA ENTEROCOLITICA* FLAGELLAR MUTANTS. Megan K. Townsend*, Penelope S. Gibbs, Birgit M. Pruess

1:20 VASOACTIVE INTESTINAL PEPTIDE RECEPTOR – 1 CONTRIBUTES TO THE PROLIFERATIVE CAPACITY OF T CELLS BY REGULATING THE PHOSPHORYLATION PATTERN OF THE ANTI-TUMOR FACTOR IKAROS. Travis Van der Steen *, Lohit Garg, Keith Benton, Steven Meinhardt and Glenn Dorsam

4:30 Business meeting (open to all members) – location to be determined

EVENING

6:00 Banquet will be at the Taube Museum of Art, 2 Main St in downtown Minot. Cash bar open at 5.

AN ACTIVITY-DEPENDENT CHANGE IN CILIARY NEUROTROPHIC FACTOR EXPRESSION IN THE RAT MAGNOCELLULAR NEUROSECRETORY SYSTEM

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Ciliary neurotrophic factor (CNTF) is a member of the interleukin-6 (IL-6) family of pleiotropic cytokines, which also includes oncostatin M, leukemia inhibitory factor, and cardiotrophin-1. CNTF acts as a survival factor for a variety of neuronal cell types, including sensory, motor, cerebral, hippocampal and hypothalamic magnocellular neurons (MCN). In the magnocellular neurosecretory system (MNS), CNTF is exclusively expressed in astrocytes of the supraoptic nucleus (SON), which are predominately located in the ventral glial limitans (VGL), and perivascular cells of the neurohypophysis. While the CNTF protein and mRNA levels have been demonstrated to be upregulated in response to injury *in vivo* (1) and CNTF is widely known to be a neuronal survival factor *in vitro*, there is a caveat: CNTF lacks a secretory signal sequence and has yet to be demonstrated to be secreted from uninjured cells *in vivo*.

Utilizing immunocytochemical analysis on the proportional area of the SON positive for CNTF-immunoreactivity (CNTF-ir), we previously demonstrated that unilateral lesion of the hypothalamo-neurohypophyseal tract will result in a statistically significant increase in CNTF-ir in the axotomized SON over a 30-day post lesion recovery period (1). Concurrently, the intact contralateral SON, which undergoes a robust axonal sprouting response in conjunction with heightened neurosecretory activity (2), also showed a statistically significant, but comparatively smaller, increase in CNTF-ir proportional area over a period of 3 to 10 days post lesion. However, it remains to be determined if the observed increase in CNTF-ir in the contralateral SON is related to the sprouting response per se or more generally to the increased metabolic and neurosecretory activity associated with the post-lesion response. Therefore, the purpose of the current study is to test the hypothesis that, "CNTF expression is upregulated by physiological stimulation of neurosecretory activity in the rat SON".

Male Sprague-Dawley rats (n=8; Charles River) were placed on 2% salt water for 10 days that will result in hypernatremia via heightened neurosecretory activity from the MCNs. Coronal cryosections of the hypothalamus were stained for CNTF (1:100; R&D Systems) and then analyzed (Image J) for the optical density of CNTF-ir within the SON. Analysis demonstrated that **the proportional area of the SON immunoreactive for CNTF significantly *decreased* (p=0.014, student's t-test) in hypernatremic SON, compared to age-matched normatremic SON.**

Quantitative analysis demonstrates that there is a significant decrease in CNTF protein expression in response to hypernatremia; however, it is still uncertain what leads to this decrease in protein expression and future studies will be performed to help answer this question. RT-PCR analysis will be utilized to determine if there is a change in hypernatremic CNTF mRNA levels compared to normatremic mRNA levels; however, it should be noted that a change in an mRNA pool may not have a direct effect on protein levels. A second possible explanation for the decrease in CNTF-ir is that in response to physiological activation the SON undergoes a morphological change that decreases the area the VGL occupies and the orientation of the astrocytic processes (3); therefore, immuno-electron microscopy will be employed to elucidate if CNTF expression decreases as a result of the morphological change that occurs in the SON.

In summary, CNTF expression has previously been demonstrated to change only in response to injury; yet, our research has demonstrated that CNTF expression can be changed in an activity-dependent fashion independent of cellular injury.

References:

1. Watt, JA, Bone, S, Pressler, M, Cranston, HJ, Paden, CM (2006) Ciliary neurotrophic factor is expressed in the magnocellular neurosecretory system of the rat in vivo: evidence for injury- and activity-induced upregulation. *Exp Neurol.* **197:** 206-214.
2. Watt, JA, Paden, CM (1991) Compensatory sprouting of uninjured magnocellular neurosecretory axons in the rat neural lobe following unilateral hypothalamic lesion. *Exp Neurol.* **111:** 9-24.
3. Bobak, JB and Salm, AK (1996) Plasticity of astrocytes of the ventral glial limitans subjacent to the supraoptic nucleus. *J Comp Neurol.* **376:** 188-197.

ROLE OF THE C3 TRANSIENT RECEPTOR POTENTIAL CHANNEL IN RAT SUPRA OPTIC NUCLEUS

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The C3 Transient Receptor Potential Canonical (TRPC3) channel is a receptor mediated plasma membrane calcium channel, which is activated by the G Protein Coupled Receptor (GPCR) signaling mediated depletion of endoplasmic reticulum (ER) calcium stores. Calcium plays an important role in vesicular exocytosis and in turn in neurosecretion. To understand the role of TRPC3 calcium channel in vesicular fusion we have examined TRPC3 localization and function in Vasopressin (VP) and oxytocin (OT) producing neurons of the hypothalamic Supra optic Nucleus (SON). The neurosecretory vesicles harboring VP and OT are present all over the neuronal axis with the axonal endings terminating in the neural lobe (NL) of the pituitary gland. The hormones are released into blood circulation through vesicular exocytosis. Dual-immunohistochemical staining of TRPC3 with VP and OT in rat brain sections of SON and NL has shown that TRPC3 colocalizes with VP and OT containing neurosecretory vesicles. The VP and OT neurosecretory vesicles also colocalized with VAMP2 suggesting the co-expression of VAMP2 and TRPC3 on the vesicles. VP release assay by ELISA showed increased VP release upon activation of TRPC3 with OAG. Calcium influx studies on the axon terminals of the NL also confirmed the expression and function of TRPC3. These results suggest that TRPC3 through its interaction with VAMP2 might play a role in neurosecretion.

ADSORPTION DYNAMICS OF ALKANES ON CARBON NANOTUBES: A MOLECULAR BEAM SCATTERING STUDY

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Molecular beam scattering techniques were used to investigate the adsorption dynamics of alkanes on single wall carbon nanotubes (SWCNTs) supported on silica (1). The kinetics of n/iso-butane adsorption on both open-end (o-CNTs) and closed-end nanotubes (c-CNTs) was studied with thermal desorption spectroscopy (TDS). Scanning electron microscopy (SEM), X-ray photoelectron spectroscopy (XPS), and energy-dispersive X-ray spectroscopy (EDX) were employed at Columbia University, from whom we obtained the samples, to inspect the sample morphology and cleanliness both before and after measurements (Fig. 1).

Flashing the sample of c-CNTs above 450 K removed functionalities that block the tube ends, resulting in a transformation to o-CNTs. Evident from TDS data, o-CNTs allow internal adsorption sites to be populated which is correlated with an increase in the initial adsorption probabilities as compared with c-CNTs, consistent with the enhancement in the surface area by opening the tube ends. The thermal desorption spectroscopy curves for annealed CNTs (o-CNTs) consist of four structures which can be assigned to adsorption on interior, groove, and exterior sites as well as condensation of the alkanes, as observed before for longer chain alkanes. Precursor-mediated (Kisliuk-like) adsorption dynamics were observed for the o-CNTs, whereas the c-CNTs show more Langmuirian-like (direct) adsorption dynamics.

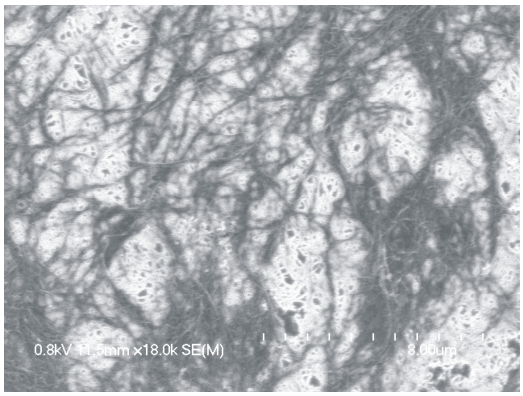

Fig. 1 SEM image on carbon nanotubes deposited on a silica support

To distinguish carbon nanotube effects from support (silica) effects, SiO_2/Si(001) has thoroughly been characterized. Data for n-nonane, n-hexane, n/isobutene, ethane, CO_2, CO, O_2, and H/H_2 was collected. Carbon nanotube binding to silica, however, is relatively weak, and flashing the samples above 500 K distinctly reduced the amount of nanotubes on the support.

Acknowledgements We are grateful for the support from B. White, S. O'Brien, N.J. Turro (Columbia University, New York) throughout the project. Financial support by the DoE Office of Science under Award DE-FG02-06ER46292 (ND state grant), and ND NSF-EPSCoR IIP seed (EPS-047679) is acknowledged.

References

(1) Adsorption dynamics of alkanes on single wall carbon nanotubes: a molecular beam scattering study
S. Funk, U. Burghaus, Brian White, Stephen O'Brien, Nicholas J. Turro, submitted

N-/ISO-BUTANE ON ANATASE (001) THIN FILM: A SURFACE CHEMISTRY STUDY

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An anatase (001) TiO_2 thin film, grown on a $SrTiO_3$ (001) substrate was probed with ultra-high vacuum (UHV) techniques: thermal desorption spectroscopy (TDS) and beam scattering. The sample was obtained from the Pacific Northwest national laboratories (PNNL). This system was chosen in reference to a study done on TiO_2 nanotubes containing both anatase and rutile crystallites (1). The (001) TiO_2 thin film can be considered the planar analog to the nanotube. Furthermore, anatase is considered to be the catalytically more active polymorph of TiO_2.

The thin film was characterized by x-ray diffraction (XRD) at PNNL prior to the UHV experiments to confirm that the deposited layer was, in fact, anatase (001). Throughout the study, the state of the film was monitored by Auger electron spectroscopy (AES) to confirm that it was both clean and intact, i.e., no substrate AES peaks have been observed.

TDS experiments for n/isobutane showed distinct mono-, bi, and multilayer features (Fig 1). AES showed that the surface remained clean during these measurements, hence molecular adsorption was concluded. In contrast, a bond activation of the alkanes would lead to the detection on carbon AES lines. TDS measurements of n-nonane showed multiple peaks at higher exposures, and subsequent AES revealed significant carbon on the surface. This is evidence of bond activation, i.e., C-C bond scission. Argon sputtering and oxygen annealing cycles were necessary to restore the anatase film. Binding energies were calculated from the TDS peak positions.

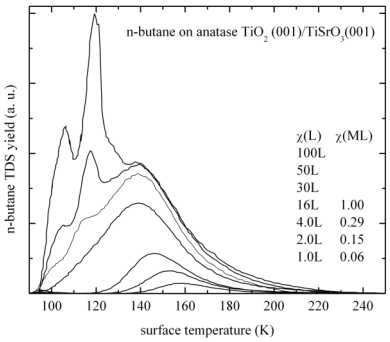

Fig. 1 TDS of n-butane on anatase (001) show distinct mono-, bi-, and multilayer desorption features.

Molecular beam scattering data yielded decreasing adsorption probability with increasing impact energy for both n- and iso-butane. Adsorption probability was systematically less for the more spherical iso-butane. This suggests that iso-butane collisions with the surface are more elastic and hence, the energy transfer to the surface is less efficient than n-butane.

Both n- and iso-butane showed coverage-independent behavior at lower impact energies (precursor-mediated adsorption) and increasingly adsorbate-assisted behavior at higher impact energies.

1. A. A. Chambers, C. W. Wang, S. Thevuthasan, I. Droubay, D. E. McCready, A. S. Lea, V. Shutthanandan, C. F. Windisch. (2002) Thin Solid Films 418, 197

ADRENERGIC RECEPTOR MEDIATED SUPPRESSION OF SEIZURES: α_2 ADRENERGIC RECEPTORS INHIBIT EPILEPTIFORM ACTIVITY VIA A SYNAPSE SPECIFIC MECHANISM

Chris WD Jurgens* and Van A Doze

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Introduction. The brain adrenergic system is a diverse projection of norepinephrine releasing neurons which regulate many cognitive functions including attention and arousal. Several lines of evidence have shown an important role for this system in the control of epilepsy. Recent studies have shown that correct manipulation of the adrenergic system may not only be effective against currently intractable epilepsies, but more importantly, may abolish seizures without deleterious side effects on learning and memory, as is experienced with many current antiepileptic therapies. This makes exploitation of adrenergic mechanisms a prime target for new antiepileptic treatments. However, because of the complexity of the adrenergic signaling system, research into the exact mechanism of the adrenergic system's effects on seizures had been hindered. Fortunately, new advances in technology and selective pharmacological agents have made it possible to begin to define the mechanisms involved in the adrenergic system's antiepileptic capabilities.

Integral in learning and memory processes, hippocampal CA3 pyramidal cells and are a common focus in temporal lobe epilepsy. Our lab has identified an α_{2A} adrenergic receptor (AR) mediated response on hippocampal CA3 pyramidal neurons which robustly inhibits epileptiform activity. Present studies have localized this response to the CA3 pyramidal cells themselves, showing no reduction of effect in the presence of pharmacological and surgical isolation. We have examined neurotransmission evoked from the major connections of the CA3 pyramidal neurons to further localize this particular adrenergic effect.

Methods. Using whole-cell recordings in rat hippocampal slices, we examined the effect of α_2 AR stimulation on excitatory post-synaptic currents (EPSCs) evoked from the major CA3 synaptic inputs (mossy fiber and CA3-CA3 recurrent), as well as from the CA3 inputs onto CA1 pyramidal neurons.

Results. EPSCs evoked from the mossy fiber-CA3 pathway and the CA3-CA1 pathway showed no reduction in response to αAR stimulation with epinephrine in the presence of 10μM timolol. In contrast, EPSCs evoked from the CA3-CA3 recurrent synapses were inhibited under the same conditions.

Conclusions. Preliminary evidence suggests that only CA3-CA3 recurrent synaptic strength is inhibited by α_2AR stimulation, while the excitatory drive to and from the CA3 cells is not inhibited. Overexcitation of the CA3 recurrent synapses is thought to be essential in temporal lobe seizures. Since this α_2AR response is specific to the CA3 recurrent synapses, this may explain how the adrenergic system is antiepileptic while at the same time not affecting other areas of cognitive function such as learning or memory.

Acknowledgments: Supported by NIH HL61438 (DMP), ND EPSCoR through NSF grant EPS-0447679 (VAD), NSF CAREER award 0347259 (VAD), and NIH COBRE program grant 5P20RR017699 (VAD).

α_{1A} ADRENERGIC RECEPTOR REGULATION OF SEIZURES & NEURODEGENERATION

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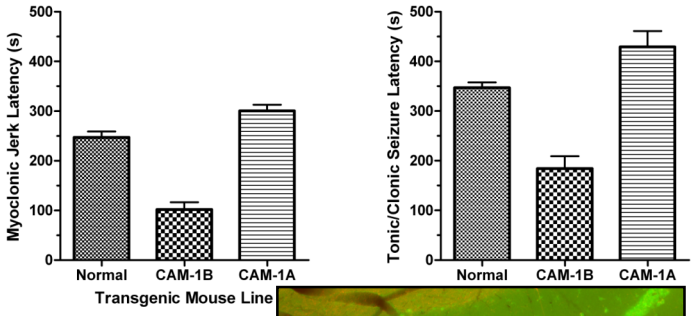
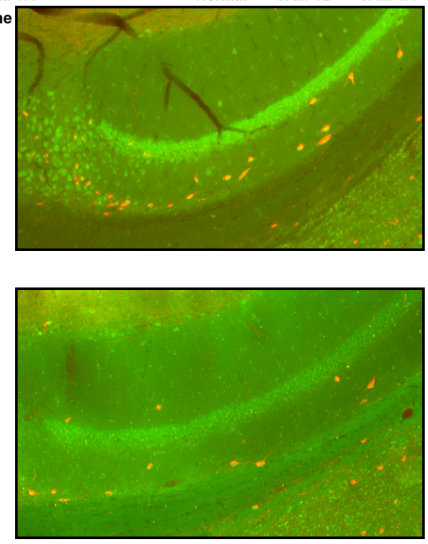
The endogenous neurotransmitter, norepinephrine (NE), has demonstrated antiepileptic effects (1, 2) and is also implicated in neurogenesis (3); however, the specific pharmacology of these actions of NE has not been clearly established. Identification of the receptor signaling mechanism by which NE exerts these effects may have significant pharmacological and therapeutic relevance. To address this, the epileptogenic agent, flurothyl, was administered *in vivo* to transgenic mice overexpressing α_{1A} (CAM-1A) or α_{1B}ARs (CAM-1B). The latency period preceding seizure progression to the exhibition of the myoclonic jerk, the first definitive sign of hyperexcitability; and tonic/clonic seizure, the first profound generalized seizure, were recorded. As illustrated in this figure, transgenic mice overexpressing α_{1A}ARs showed a significant increase in latency periods (compared to age-matched control animals) preceding the initial myoclonic jerk and the progression to the first generalized seizure or tonic/clonic seizure. In contrast, mice overexpressing α_{1B}ARs showed a marked decrease in latency and increased seizure progression to both the myoclonic jerk and to the first generalized seizure. These findings suggest that α_{1A}AR stimulation is antiepileptic, and activation of α_{1B}ARs is proepileptic.



Immunohistochemistry was then used to visualize fluorescently labeled interneurons in the hippocampal CA1 region of transgenic mice overexpressing α_{1A} or α_{1B}ARs. As shown in these images, α_{1A}AR overexpression was associated with increase numbers of interneurons [top: α_{1A}AR (FITC-green) immunofluorescence on interneurons labeled with somatostatin-14 (CY3-red)]. In contrast, decreased interneuron numbers were observed in mice overexpressing α_{1B}ARs [bottom: α_{1B}AR (FITC-green) immuno- fluorescence on interneurons labeled with somatostatin-14 (CY3-red)]. Control mice exhibited interneuron counts intermediate between the overexpressed α_{1A} and α_{1B}AR mice (data not shown).



The implications of these finding are potentially very significant as they link the α_{1A}AR to possible, antiepileptic, neuroprotective and neurogenic qualities. Further insight into the mechanism by which these processes occur may lead to pharmacological advancements in the treatment of epilepsy and other neurodegenerative diseases.

1) Giorgi FS, Pizzanelli C, Biagioni F, Murri L and Fornai F (2004) Neurosci. Biobehav. Rev. 28, 507.
2) Weinshenker D and Szot P (2002) Pharmacol. Ther. 94, 213.
3) Hiramoto T, Ihara Y and Watanabe Y (2006) Neurosci. Lett. 408, 25.

Acknowledgments: Supported by NIH HL61438 (DMP), ND EPSCoR through NSF grant EPS-0447679 (VAD), NSF CAREER award 0347259 (VAD), and NIH COBRE program grant 5P20RR017699 (VAD).

RECRUITING AMERICAN INDIAN PARTICIPANTS FOR A GENETIC EPIDEMIOLOGIC STUDY

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Due to previous negative experiences, some American Indian (AI) communities are distrustful of biomedical research in general and genetic research in particular. The Turtle Mountain Community College was awarded an NIH grant to study possible genetic influences on pre-eclampsia, to encourage tribal college students to consider a biomedical career and to develop the local research infrastructure. Pre-eclampsia is a serious medical condition among both Caucasian and AI populations. The prevalence in AI populations of genetic polymorphisms that may increase the risk of pre-eclampsia is unknown. In addition, the influence of these polymorphisms, given the AI genetic background, may differ from majority populations.

This analysis is based on recruitment of cases, controls, and prospective study participants. Approval for the study has been given by relevant Institutional Review Boards. Cases/controls are identified using the electronic records system at the local Indian Health Service hospital. Potential participants were given a brief description of the project and if agreeable, met for formal informed consent. Much effort has been made to publicize the project to the community.

To date, 76 cases, 90 controls, and 42 cohort participants have been recruited. During the past 28 months, 393 individuals have been identified and contact information was adequate for 309 (78.6%), 246 were given basic information, 212 have had full consent interviews, and of these 1.4% opted not to participate. Travel was required to recruit 75.3% of retrospective participants.

When recruiting potential participants in Indian Country, traveling to their homes appears more effective and efficient. Advertising efforts have been questionable in their effectiveness. Having local individuals as recruiters seems to be a very important component of the process.

A NEW ASSAY FOR THE QUANTIFICATION OF BIOFILM

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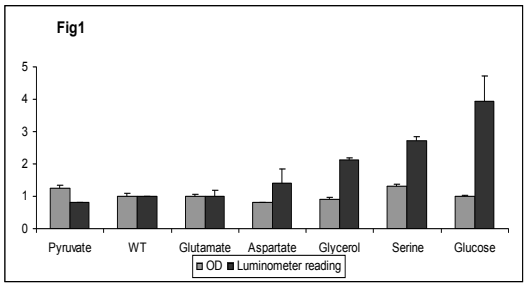
Biofilm can be defined as community of bacterial cells on solid surface or at the solid-liquid interface. The formation of biofilm follows a set pattern over a period of time. The reversible attachment is followed by irreversible attachment and eventually maturation of the biofilm is observed (Toole et al, 2000), however at a later stage some cells might escape from the biofilm set up, contributing to what is called the dispersal stage. The cells in the biofilm set up are known to exhibit antibiotic resistance (Stewart et al, 2001.), and have eventually been found to be of great importance in various fields of science. The vitality of such a bacterial community hence cannot be overlooked. The bacterial biofilm formation till date has been estimated using the crystal violet stain, the technique is qualitative and largely inconsistent as a quantitative measure. In the light of these difficulties we tried to develop a new quantitative technique for biofilm estimation that is reliable and less time consuming.

The Bactiter-Glo® system works by estimating the number of ATP molecules produced by bacterial cultures. The produced ATP is converted to a light signal by the enzyme system and depending on the intensity of the emitted light the number of bacterial cells present in the biofilm environment can be estimated. Using the system it was found that a 2 fold increase in the bioluminescence signal always corresponded to a two fold increase in the number of bacterial cells.

The system was tested by measuring the effect of various substrates on the biofilm formation. These substrates were mostly metabolic intermediates and were hypothesized to affect the acetyl-CoA levels in the cell. Increased acetyl-CoA resulted in an increased level of acetyl phosphate. Increased level of acetyl phosphate affected the expression of numerous genes that are involved in the biofilm formation (Wolfe et al 2003).

The substrates used in the experiment were serine, pyruvate, aspartate, glycerol, glucose and glutamate. The supplemented media was inoculated with overnight broth (37^{o}C) at a 1:100 dilution. 50 µl of each culture was dispensed in 16 wells of the 96 well micro-titer plate. 16 wells of inoculated, unsupplemented media were used as a control. The plate was incubated at 37^{o}C for 36 hours. After incubation, the wells were rinsed with Phosphate Buffer Saline to get rid of excess media and any other debris. Quantitative estimation of the biofilm was carried out using the Bactiter-Glo® assay. In a separate set of experiments cultures with the six substrates were grown (at 37^{o}C) and growth curves were plotted over 48 hours. The experiments were carried out in duplicates and the values from each set of experiment were averaged.



The experiments revealed that the presence of glucose increased biofilm formation by almost 4 fold (Fig1). Serine was also found to increase biofilm formation by a factor of 2.9. Amongst the other substrates glycerol caused a 2 fold increase, whereas aspartate, glutamate and pyruvate did not have any significant quantitative effect on the biofilm formation.

The overall bacterial growth and the biofilm formation followed completely different patterns.
This indicates that the effect of the metabolites on biofilm formation is not merely caused by alterations of the growth rate.

Reference

Toole, OG, Kaplan, BH, Kolter, R. (2000) Annual Rev Microbiol., 54,49
Wolfe, A, Chang, ED, Walker, DJ, Seitz-Partridge, EJ, et al. (2003) Molecular Biology., 48,977

NON – FLAGELLAR PHENOTYPES OF *YERSINIA ENTEROCOLITICA* FLAGELLAR MUTANTS

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Yersinia enterocolitica is a gram-negative rod-shaped enteropathogen closely related to *Escherichia coli*. It is capable of producing flagella. The flagellar hierarchy in *Y. enterocolitica* is thought to be closely related to that of *E. coli* with the expression divided between three classes of genes (class I-III). In both species the FlhD/FlhC complex constitutes the class I master regulator of the system. FliA is part of class II and is the sigma factor of the flagellar system. In *Y. enterocolitica* FlhD/FlhC is a regulator of many non-flagellar genes, including many which are involved in central metabolism (1). Similarly, it has been shown that FliA has a negative affect on the expression of several plasmid-encoded virulence factors (2). Based on this complex gene regulation it is hypothesized that *flhD* and *fliA* mutants will have pleiotropic phenotypes.

Phenotype microarrays were previously used to study central metabolic phenotypes of *Y. enterocolitica* 8081c and its isogenic *flhD* and *fliA* mutants (3). Both mutant strains showed similar growth profiles, displaying higher growth on purines, intermediates of the urea cycle and of the tricarboxylic acid cycle. The wild-type demonstrated better growth on pyrimidines, L-histidine and on sugars degraded by early stages of glycolysis. Several dipeptides also resulted in differences in growth patterns between the *flhD* and *fliA* mutants and the wild type *Yersinia*. Genes associated with the phenotypic differences in central metabolism were chosen for further examination using real-time PCR. The expression ratios of wild-type/mutant were normalized with three housekeeping genes and calculated as $2^{-(\Delta Tc\ norm)}$. Gene expression correlated with the metabolic phenotype.

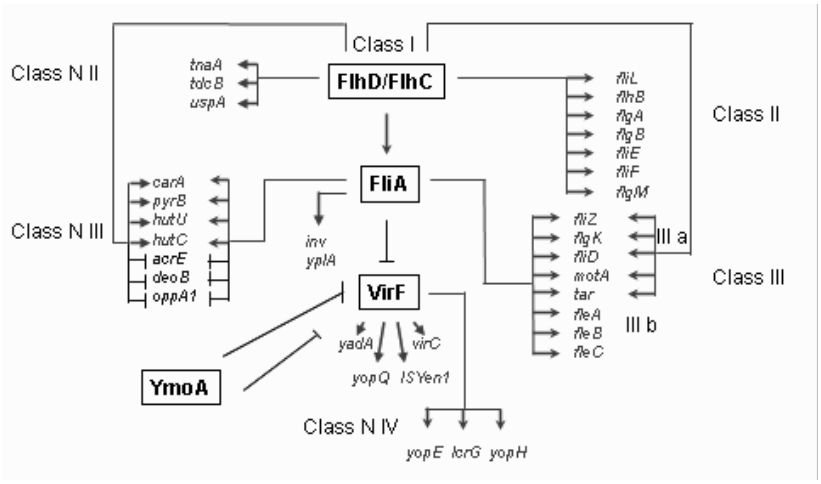

Fig. 1: Outline of Gene Regulation by FlhD/FlhC and FliA

Virulence phenotypes were studied using the chicken embryo lethality assay that was previously described for *E. coli* (4), and has been modified for use with *Y. enterocolitica*. Twelve day old chicken embryos were inoculated into the allantoic cavity with *Y. enterocolitica* strains 8081v, 8081c, 8081v *flhD*, or 8081v *fliA*. Embryos were monitored for viability over five days and lethality was calculated by percent. The *fliA* and 8081v strains showed high levels of lethality (75-80%). The *flhD* mutant and the 8081c strains caused lower lethality at 49% and 34%, respectively. Statistical analysis included logistic regression and the Duncan's Multiple Range test.

In contrast with the metabolic phenotypes, in which the mutants shared a phenotypic profile, the virulence phenotypes of the *flhD* and *fliA* mutants differ. The inability to chemotax to the nutrients may be the cause of some of the metabolic phenotypes. As chemotaxis genes are included in the genes regulated by both FlhD/FlhC and FliA this could explain the shared metabolic activities. The difference in virulence levels between the *flhD* and *fliA* mutants indicates that FlhD/FlhC controls genes important to virulence which are unregulated by FliA. These virulence affecting genes are potentially part of the flagellar type III secretion system. The lack of this system in the *flhD* but not the *fliA* mutants would prevent the export of proteins that may contribute to virulence.

1) Kapatral, V. *et al*, 2004. Microbiol. 150:2289.
2) Horne, S.M., and B.M. Prüβ. 2006. Arch. Microbiol. 185:115.
3) Iyer, J. *et al*. 2006. Proceedings for The North Dakota Academy of Science. Vol. 60 In Press
4) Gibbs, P.S. *et al*. 2003. Avian Dis. 47:370.

VASOACTIVE INTESTINAL PEPTIDE RECEPTOR – 1 CONTRIBUTES TO THE PROLIFERATIVE CAPACITY OF T CELLS BY REGULATING THE PHOSPHORYLATION PATTERN OF THE ANTI-TUMOR FACTOR IKAROS.

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Acute and chronic leukemias are well-documented worldwide killers. Since 2000, nearly 200,000 human cases have been diagnosed in the United States with a mortality rate greater than 60%. Nearly half of these patients will have deletions in the DNA-binding domain of their Ikaros (IK) gene. Furthermore, $IK^{+/-}$ mice that have a deletion in their DNA-binding domain develop leukemia with 100% penetrance. IK, a tumor-suppressor transcription factor, is selectively expressed in the hemo-lymphoid compartment and is a master regulator for lymphopoiesis. Furthermore, IK is differentially phosphorylated in a cell cycle dependent manner through several phosphorylation kinase consensus binding domains within its primary sequence, including protein kinase A (PKA). As T cells cycle from G_1 to G_2/M phase, the phosphorylation pattern of IK changes and modulates its DNA-binding affinity. We have previously reported that Ikaros DNA-binding isoforms nearly silence the expression of a lymphocyte, cell-cycle-modulating, G-protein coupled receptor (GPCR), termed vasoactive intestinal peptide receptor – 1 (VPACR-1). This receptor is highly expressed in the immune compartment and impedes T cell proliferation through a Gs/cAMP/PKA dependent pathway. The leukemogenesis activities of Ikaros may be due to the dysregulation of VPACR-1 induced PKA signaling that results in an Ikaros phosphorylation pattern switch from a non-proliferative to a proliferative state. There is, therefore, a *critical need* to understand how VPACR-1 signaling through PKA maintains Ikaros in a non-proliferative phosphorylation state in naïve T cells. To this end, we will employ the use of HUT-78 cells, a cutaneous T lymphocyte that expresses high levels of VPACR-1. Addition of exogenous concentrations of the vasoactive intestinal peptide, the natural ligand for VPACR-1, to HUT-78 T cells will be the primary experimental procedure to test whether the Ikaros phosphorylation pattern changes. HUT-78 cell lysates will be collected, Ikaros protein immunoprecipitated, and two-dimensional electrophoresis gels performed. Differential phosphorylation patterns will be verified by nano-flow mass spectrophometry. We expect this research to allow us to gain valuable new insight into the molecular mechanisms of Ikaros-mediated leukemogenesis potentially controlled by the signal transduction of a not well understood GPCR gene, VPACR-1. This research will be funded by a NIH/NIDDK KO1 5K01PK64828-2 grant, Center of Protease Research and The Core Biology Facility at NDSU.

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PROFESSIONAL COMMUNICATIONS

SCHEDULE OF PRESENTATIONS

Graduate talks will be in "Study 2" room on the ground floor of the Gordon B. Olson Library – session will be chaired by A. Rodger Denison Competition judges

MORNING SESSION

7:30 Registration desk open

8:20 Greetings from President Christopher Keller

12:00 LUNCH (served in the Olson Library Media Center)

AFTERNOON SESSION

1:40 THE EFFECT OF ATRAZINE ON THYROID HORMONE INDUCTION OF METAMORPHOSIS IN THE AXOLOTL, *AMBYSTOMA MEXICANUM.* Christopher K. Beachy, Kenneth C. Cabarle, Charles Crites, Leah Crites, and Pam Clarkson

2:00 ANALYSIS OF GONADAL DEVELOPMENT IN ATRAZINE EXPOSED AMBYSTOMA MEXICANUM, THE AXOLOTL. Kenneth C. Cabarle [1], Dwight K Blackhawk[2], and Christopher K Beachy[1]

2:20 NOVEL DETERMINATION OF PHYTATE BY ION CHROMATOGRAPHY IN WILD RICE AND DIET COMPOSITES. G.M. Dahlen,* C. A. Zito and W.K. Canfield

2:40 RESURRECTING FORGOTTEN FOSSIL MUSSELS OF BRITISH COLUMBIA. Joseph H. Hartman*

3:00 BREAK

3:20 FACTOR V LEIDEN GENETIC VARIANT IN AN AMERICAN INDIAN POPULATION. Melanie Nadeau[1], Sheri T. Dorsam2, Jacob Davis[1], Levi Gourneau[1], Jordan Vallie[1], Lyle G. Best[1]*

3:40 ANALYSIS OF PROTEIN BINDING IN THE *MLL* TRANSLOCATION BREAKPOINT REGION Heidi J. Super, Alysa L. Anderson and Aileen Aldrich

4:30 Business meeting (open to all members) – location to be determined

EVENING

6:00 Banquet will be at the Taube Museum of Art, 2 Main St in downtown Minot. Cash bar open at 5.

THE EFFECT OF ATRAZINE ON THYROID HORMONE INDUCTION OF METAMORPHOSIS IN THE AXOLOTL, *AMBYSTOMA MEXICANUM*

Christopher K. Beachy, Kenneth C. Cabarle, Charles Crites, Leah Crites, and Pam Clarkson

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The thyroid hormones (TH) T_3 and T_4 initiate metamorphosis in larval amphibians. The endocrine axis-regulation of TH secretion is controlled upstream at several upstream physiological checkpoints (e.g., the pituitary secretes thyroid stimulating hormone, the hypothalamus secretes corticotropin-releasing hormone that induce secretion by the pituitary and interrenals). A growing body of evidence suggests that the heavily-used herbicide Atrazine can disrupt normal endocrine function, and any compound that has this capability is referred to as an endocrine disruptor (or ED). For example, there is experimental evidence that atrazine exposure during the larval stage results in male metamorphosing frogs (Xenopus laevis) with low testosterone levels and hermaphroditic gonads (1). We tested the hypothesis that atrazine can influence the endocrine-regulated control of metamorphic development by growing larval salamanders (*Ambystoma mexicanum*) at three chronic dosages of Atrazine, inducing metamorphosis by treatment with three levels of T_4 (thyroxine), and recording metamorphic development.

Embryos of *A. mexicanum* were obtained from the *Ambystoma* Genetic Stock Center (Lexington, KY) and placed in a 40 L aquarium in reverse-osmosis water. Upon hatching, larvae were individually placed in plastic boxes with RO water and were randomly assigned to one of 36 experimental treatments (Fig. 1).

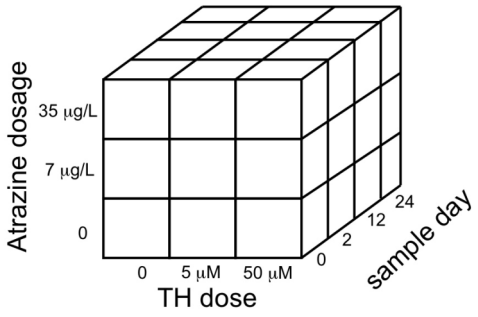

Fig. 1. -- Schematic representation of matrix of treatments. Structure was a 3 X 3 X 4 full-factorial randomized block design. Number of replicates per treatment was seven for a total 252 salamanders.

Larval salamanders were fed an ad libitum diet of freshly hatched brine shrimp nauplii and blackworms. Water was exchanged every other day, and atrazine treatments were initiated upon hatching. After seven months of growth, TH treatments were initiated. Salamanders were sampled at four time points after initiation of TH. Salamanders were killed by immersion in MS-222, and preserved in formalin and stored in ethanol. At a later time, animals were measured for size (mass and length [SVL]) and scored for metamorphic development by measuring length of gill and tail fin. These two morphological features are an objective measurable assay of metamorphic development.

Larvae treated with TH exhibited expected metamorphic progress. All larvae at high TH metamorphosed by day 24 of TH treatment. Metamorphic development was unaffected by atrazine treatment. This supports the suggestion that action of atrazine is limited to the region of endocrine control that influence gonadal development.

This research was supported by NIH Grant Number P20 RR016741 from the INBRE Program of the National Center of Research Resources.

Sources:

1) Hayes, T.B., A. Collins, M. Lee, M. Mendoza, N. Noriega, A.A. Stuart, and A. Vonk. 2002. Proc. Nat. Acad. Sci. USA 99:5476-5480.

ANALYSIS OF GONADAL DEVELOPMENT IN ATRAZINE EXPOSED AMBYSTOMA MEXICANUM, THE AXOLOTL.

Kenneth C. Cabarle [1], Dwight K Blackhawk[2], and Christopher K Beachy[1]

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Minot State University, Minot, ND 58707, USA, [2] Department of Science, Fort Berthold Community College,
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Environmental endocrine disruptors (ED's) present a toxicological stressor in aquatic environments. Analysis of the effects of applied pesticides and fertilizers are two of the ED inputs that may be leading to declines in amphibian populations (Lannoo, 2005). Currently the Amphibian Growth Project has been exploring the effects of the pesticide atrazine on the model salamander *Ambystoma mexicanum,* the Axolotl (Putta et al., 2004). We hypothesized that Atrazine exposure may cause developmental and morphological anomalies in the treated Axolotls. Individual specimens from three separate experiments were assessed for morphological, and developmental differences (n = 530). Preserved specimens were assessed for gross exterior morphology, measured for SVL, gonads were dissected from individuals, weighed to the nearest .001 grams, and images of gonads were captured with a digital camera.

Gonad gross morphology was assessed visually using a dissecting microscope with a maximum magnification of 630X. Specimens were scored as males or females based on a scale modified from Ryan and Semlitsch, 1998. Digital photographs were manipulated in Photoshop CS2 and then analyzed with Image J software. Gonads were hand digitized and measured for total area and perimeter. Multivariate statistical evaluation has shown an effect of atrazine on gonads ($p > .05$). It appears that the effect is different between the sexes with atrazine having a stronger effect in relation to the development of ovaries.

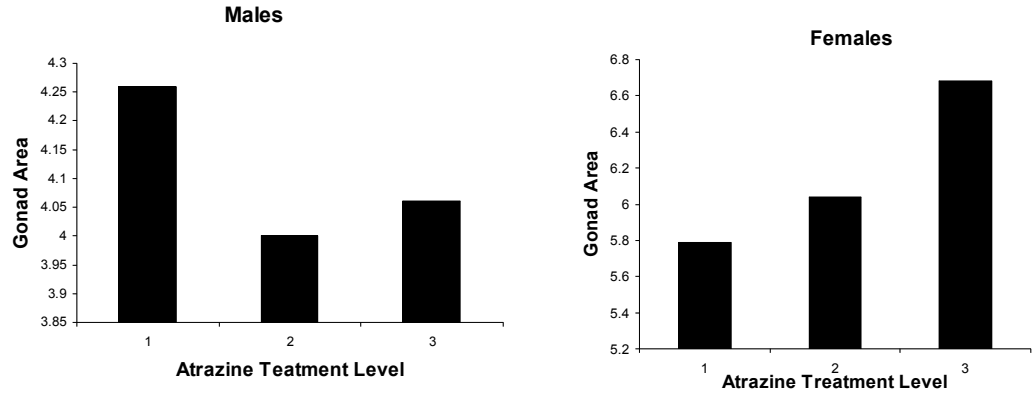

Figure 1: Bar graphs plotting atrazine level (1=0μg/L, 2=7μg/L, and 3=35μg/L) effects by sex on axolotl gonad area.

This research was supported by NIH Grant Number P20 RR016741 from the INBRE Program of the National Center of Research Resources.

Sources:

1) Lannoo, M. (ed.). 2005. Amphibian Declines: The Conservation Status of United States Species. University of California Press, Berkeley. 1094 pp.
2) Putta, S., and 14 others. 2004. From biomedicine to natural history research: expressed sequence tag resources for ambystomatid salamanders. BMC Genomics 5:54.
3) Ryan, T. J. & Semlitsch, R. D. (1998). Intraspecific heterochrony and life history evolution: decoupling somatic and sexual development in a facultatively paedomorphic salamander. Proceedings of the National Academy of Sciences of the United States of America 95, 5643–5648.

NOVEL DETERMINATION OF PHYTATE BY ION CHROMATOGRAPHY IN WILD RICE AND DIET COMPOSITES

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USDA, ARS, Grand Forks Human Nutrition Research Center
Grand Forks, N.D. 58202

We have developed an ion chromatography (IC) assay using ultraviolet (UV) detection following post-column derivatization with ferric nitrate to determine phytate [inositol hexakis phosphate (iP6)] (1) (2) in wild rice samples and other diet composites. Samples were ground to a fine homogeneous powder, dried under vacuum, extracted with an acid (0.5 N hydrochloric) for 17 hours under constant agitation and centrifuged. Recoveries for phytate in wild rice extracts ranged from 96%-102%.

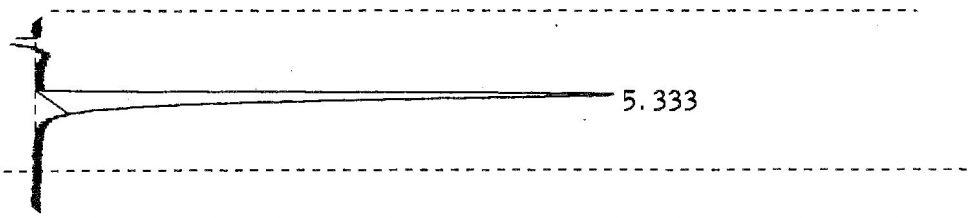

Figure 1. Chromatogram showing resolution of 4 ug phytate (5.33 min.).

To evaluate sensitivity, IC post-column derivatization was compared with the Association of Analytical Chemists (AOAC) "Official Method 986.11" results. Detection limits for phytate were 400ng for IC and 20ug AOAC respectively.

Diet composites were analyzed by both methodologies and produced similar results with IC method yielding slightly higher results [Table 1.]

Table 1. Phytate concentrations for diet composites by both methods.

Diet	Phytate IC			Phytate AOAC		
	mg/g	STD	%CV	mg/g	STD	%CV
Composite 1.	1.00	0.070	7.10	0.786	0.020	2.49
Composite 2.	1.71	0.156	9.09	1.42	0.040	2.83
Composite 3.	1.06	0.134	12.73	0.732	0.028	3.86
Composite 4.	1.85	0.088	4.73	1.434	0.039	2.74

AOAC method 986.11 is a tested method for phytate analysis. However, the method is laborious and does not have the sensitivity necessary for smaller samples that may require phytate quantification. Post-column derivatization has a 50-fold increase in sensitivity and requires only one mobile phase (isocratic) operation for phytate separation.

1. Phillippy, B., Bland J.M., and Evens, T.J., (2003) J. Agric. Food Chem., 51, 350-353.

2. Harland, B.F., Williams, S.S, and Oberleas, D., (2004) J. Food Composition and Analysis 17, 227-233.

RESURRECTING FORGOTTEN FOSSIL MUSSELS OF BRITISH COLUMBIA

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Introduction. There are numerous continental molluscan taxa that are known only from their first publication. A new species is described, sometimes from substandard material, and it may be many years before it sees further reference outside of a compilation of names or citations. These taxa may go on, however, to be used in summaries of biotic diversity when little else is known of their chronostratigraphic range, sedimentary content, or morphological variability. This contribution examines two essentially forgotten taxa and provides them with modern taxonomic, geographic, and geologic context in the hope that additional specimens will be found and studied, permitting the taxa to be used more meaningfully.

Historical Context. *Unio hubbardii* Gabb 1869 (1) and *Unio nanaimoensis* Whiteaves 1901 (2) were both described from Cretaceous strata in the vicinity of coal mines on the islands of western British Columbia, Canada. Both holotype specimens were illustrated as line drawings, the latter being rather generalized in nature. Both were later assigned by Russell (3) to *Elliptio* on the basis of shape, with comparisons to specific modern *Elliptio* taxa. The description of *U. hubbardii* was based on a single articulated specimen referred to the Upper Cretaceous Chico Group at the Nanaimo Coal mine (JHH Locality L6762). This location was refuted by Whiteaves (2, 4), who otherwise reported *U. hubbardii* in some abundance at Hooper's Creek or King's Tunnel, near Cowgitz' Coal Mine on Graham Island (Queen Charlotte Islands) (L3243). One addition specimen was figured by Whiteaves (4, p. 67) from "Carbonaceous Shales" from L3243. Whiteaves (2, p. 178) described *U. nanaimoensis* from a single articulated specimen "in shale at the top of No. 6 Pit, Wellington Colliery, Nanaimo" (L3053), Vancouver Island.

Interpretations/Documentation. Subsequent spellings of *Unio hubbardii* have, for whatever reason, typically omitted the second "*i*." A description later provided by Whiteaves (4), indicates *U. hubbardii*, can likely be assigned to *Plesielliptio* on the basis of beak sculpture. The reassignment of *Unio nanaimoensis* to *Plesielliptio* would be logical based on Russell's (3) first revision description, but *Rhabdotophorus* is equally likely without knowledge of the umbo and postumbonal sculpture. The holotype specimen (GSC 5889) is presently unavailable. Whiteaves' (4) figured hypotype (GSC 4923) of *P. hubbardii* (holotype unknown) was later questionably recorrelated with the Lower Cretaceous Haida Formation by the Geological Survey of Canada (catalog data). The age and stratigraphic assignment of *P. hubbardii* is uncertain. Haggart (5, 6) revised this age based on associated palynomorphs and placed *P. hubbardii* in lower Eocene to lower Oligocene strata informally named the Slatechuck Formation (7). The Cowgitz mine works are located at 53.22000E N latitude and 132.26500E W longitude, on the eastern flank of Mount Seymour, east of Long Inlet, on the southern Graham Island. *P.? nanaimoensis* can be correlated with the lower Campanian Extension Formation (Nanaimo Group), and is associated with the Wellington coal bed (8). The shaft/pit locality is now wooded and in a semi-developed area on the outskirts of Nanaimo. An approximated location based on mine map and observed pit depressions is 49.190312E N latitude and 124.010522EW longitude. The locality is between Maxey Road on the southwest and Highway 19 on the northeast, and north of the T-junction of Durnin Road.

Conclusions. This brief report significantly updates the location, age, and stratigraphy of rare occurrences of mussels specifically unrelated to Western Interior mussel diversification. These two British Columbia species have been out of circulation, while geologists have continued to study the slivers of mollusk-bearing continental strata. Hopefully, with this knowledge, additional specimens can be forthcoming to provide a more insight into the evolution of freshwater mussels on terrain elements in this part of North America.

1. Gabb, W.M. (1869) Geological Survey of California, Palaeontology, v. II, p. 1-299, plus pls.
2. Whiteaves, J.F. (1901) The Ottawa Naturalist, v. 14, no. 10, p. 177-179.
3. Russell, L.S. (1934) The Canadian Field-Naturalist: v. 48, no. 1, p. 1-4.
4. Whiteaves, J.F (1876) Geological Survey of Canada, Mesozoic Fossils, v. 1, pt. 1, p. 65-68; (1884), pt 3, p. 230.
5. Haggart, J.W. (1990) Geological Survey of Canada Paper 90-10, p. 253-277.
6. Lewis, P.D., and Ross, J.V. (1990) Geological Survey of Canada Paper 90-10, p. 31-50.
7. Haggart, J.W. (2004) Geological Survey of Canada, Open File 4681, 1 sheet, scale 1:250,000.
8. Clapp, C.H. (1912) Geological Survey of Canada, Map 54A, 1 sheet.

FACTOR V LEIDEN GENETIC VARIANT IN AN AMERICAN INDIAN POPULATION

Melanie Nadeau[1], Sheri T. Dorsam2, Jacob Davis[1], Levi Gourneau[1], Jordan Vallie[1], Lyle G. Best[1]*

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The clinical condition characterized by an increased propensity to pathologic clot formation is termed "thrombophilia". In 1994 Bertina(1) and Greengard(2) independently reported that a genetic variant of the human Factor V gene, known as Factor V Leiden (FVL), causes activated protein C resistance and is often found in patients with thrombophilia. An FVL allelic frequency of about 2% was initially reported in the Dutch population(1). Multiple populations have now been screened for FVL and while it continues to be found in relatively high prevalence in Caucasian populations and to a lesser extent in Asia Minor, it remains rare in other areas of the world.(3) Analysis of haplotypes associated with FVL indicate that this variant may have arisen from a single founder(4) and despite a 40-80 fold increased risk of pathologic clot formation among FVL homozygotes(5), in the past it may have conferred a selective advantage on heterozygotes due to reduced blood loss during trauma or childbirth(6).

Factor V Leiden has been examined as a risk factor for other pathologic conditions that may be less directly related to increased clot formation. One of these is the obstetric complication known as pre-eclampsia (PE). The classic signs of PE include hypertension, nephropathy and in severe cases, seizures and consumptive coagulopathy. While the etiology of PE is unknown, one theory is that placental ischemia causes the release of various inflammatory and vaso-active substances that create these secondary effects. In 1996 investigators began to study the possible role of thromophilia and FVL (among other genetic variants) in the development of this placental ischemia.(7,8) Meta-analyses have found significant pooled odds ratios (OR 1.81 95% CI 1.14-2.87) for pre-eclampsia in those with one or more FVL alleles(9); but some other studies have not found significant associations.(10)

As part of an effort to enhance biomedical research capabilities at a tribal college, we investigated the possible etiologic role of FVL among PE patients in this American Indian community. A case-control study has enrolled 76 cases and 90 control participants to date. Genotyping of cases was given priority and has identified 5 heterozygous and no homozygous individuals with FVL variants thus far. Control genotypes (15) have not included any FVL alleles as yet. Pooling both case (which are probably not representative of the population) and control genotypes would give an allele frequency of 2.6% (95% CI 0.4% to 5.2%). The literature contains 4 references to FVL variants among American Indians or related populations. Only 3 FVL heterozygous individuals were found among a total of 1014 Oji-Cree and Pima Indian participants in Ontario, Canada and southwestern United States, respectively.(11,12) Two other studies among a total of 167 Greenland Inuit and American Indians residing in California found no FVL alleles.(13,14)

Completed genotyping of the remaining controls (including the planned 62 controls yet to be enrolled) will allow sufficient power to detect an odds ratio of PE (given FVL) as low as 5.3.

Supported by NIH grant P20 RR016741 from the NCRR.

[1] Bertina RM, Koeleman BPC, Koster T, Rosendaal FR, Dirven RJ, deRonde H, van der Velden PA, Reitsma PH. (1994) Nature.,369,64.
[2] Greengard JS, Sun X, Xu X, Fernandez JA, Griffin JH, Evatt B. (1994) Lancet., 343,1361.
[3] Rees DC, Cox M, Clegg JB. (1995) Lancet.,346,1133.
[4] Cox MJ, Rees DC, Martinson JJ, Clegg JB. (1996) Br J Haematol.,92,1022.
[5] De Stefano V, Rossi E, Paciaroni K, Leone G. (2002) Haematologica.,87,1095.
[6] Donahue BS, Gailani D, Higgins MS, Drinkwater DC, George AL Jr. (2003) Circulation.,107,1003.
[7] Dizon-Townson DS, Nelson LM, Easton K, Ward K. (1996) Am J Obstet Gynecol.,175,902.
[8] Grandone E, Margaglione M, Colaizzo D, Cappucci G, Paladini D, Martinelli P, Montanaro S, Pavone G, Di Minno G. (1997) Thromb Haemost.,77,1052.
[9] Lin J, August P. (2005) Obstet Gynecol.,105,182.
[10] GOPEC Consortium. (2005) Am J Hum Genet.,77,127.
[11] Hegele RA, Harris SB, Cao H, Hanley AJ, Zinman B. (1998) Diabetes Care.,21,1203.
[12] Kohler HP, Boothby M, McCormack L, Knowler WC, Grant PJ. (1997) Thromb Haemost.,78,961.
[13] Gregg JP, Yamane AJ, Grody WW. (1997) Am J Med Genet.,73,334.
[14] deMaat MPM, Kluft C, Jespersen J. (1996) Lancet.,347, 58.

ANALYSIS OF PROTEIN BINDING IN THE *MLL* TRANSLOCATION BREAKPOINT REGION

Heidi J. Super, Alysa L. Anderson and Aileen Aldrich

Department of Biology, Minot State University, Minot ND 58707

Objective: The *MLL* gene is remarkable for its involvement in translocations with greater than 50 unique loci in several different subtypes of human acute leukemia. In addition, the region of *MLL* that breaks and joins to other genes in leukemogenic translocations is remarkably conserved at just over 8 kilobases (kb) in a central region of the gene. This 8 kb region is referred to as the *MLL* breakpoint cluster region (bcr).

Much sequence and structure analysis has been done to characterize the *MLL* bcr to elucidate the mechanism of *MLL* translocation. Various studies have shown evidence for potential recombination mediating features, including a DNA topoisomerase II cleavage site, a DNase I hypersensitivity site and recombination signal sequences for V(D)J gene segment joining. In addition, the region is rich in repetitive DNA with several Alu repeats which could facilitate homologous recombination with loci throughout the genome.

However, little is known about the protein-binding characteristics of this region. We have begun analyzing protein binding in the *MLL* bcr using electrophoretic mobility shift assay (EMSA). Detection of differential protein binding potential within the bcr may provide insight into the regions of the *MLL* bcr which are necessary for recombination with other loci. More important, eventual identification of specific *MLL* bcr binding proteins would provide essential clues to the mechanism of aberrant recombination involving *MLL*.

Methods: Nuclear extracts from hematopoietic and non-hematopoietic cells are incubated with labeled double stranded oligonucleotides from 50-200 bp in length in EMSA binding buffer. Binding of proteins to specific *MLL* bcr oligonucleotides is indicated by retarded migration in 6% polyacrylamide gels relative to oligonucleotide migration in the absence of proteins. Specific binding is shown by competition with unlabeled oligonucleotides.

Results: Binding of nuclear protein(s) has been detected in the extreme 5' end of the 8 kb *MLL* bcr. The binding is sequence specific as shown by competition with unlabeled oligonucleotide of the same sequence. Binding was seen with nuclear extracts from two hematopoietic cell lines, REH and Jurkat, but not with a purified control protein (OCT2 transcription factor). The degree of retardation of migration indicates a possible protein complex binding the 5' oligonucleotide. Other regions of the *MLL* bcr, including the extreme 3' boundary of the *MLL* bcr and a region described as an in vivo DNA topoisomerase II cleavage site were did not show retarded migration in EMSA with REH or Jurkat nuclear extracts.

Conclusions: We have identified one apparent region of protein binding within the *MLL* bcr. The region marks the extreme 5' boundary of the bcr and may be relevant in the formation of *MLL* translocations. Experiments are underway to narrow the 200-bp region of binding by using small overlapping oligonucleotides from the region and to test binding with nuclear extracts from non-hematopoietic cell lines. Ultimately protein binding will be compared throughout the *MLL* bcr with the final goal of identifying potential recombination-mediating nuclear proteins.

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CONSTITUTION of the NORTH DAKOTA ACADEMY OF SCIENCE

Founded 1908, Official State Academy 1958

ARTICLE I - *Name and Purpose*

Section 1. This association shall be called the NORTH DAKOTA ACADEMY OF SCIENCE.

Section 2. The purpose of this association shall be to promote and conduct scientific research and to diffuse scientific knowledge.

ARTICLE II - *Membership*

Membership in the Academy shall be composed of persons who share the stated purpose of the Academy and who are active or interested in some field of scientific endeavor.

ARTICLE III - *Council*

The officers of the Academy shall be a President, a President-Elect, and a Secretary-Treasurer. The Council, consisting of the officers, the retiring President, and three elected Councilors, shall be responsible for the fulfillment of the scientific and business obligations of the Academy.

ARTICLE V - *Dissolution and Limits of Action*

Section 1. In the event of dissolution of the Academy, any remaining assets shall be distributed to organizations organized and operated exclusively for education and scientific purposes as shall at the time qualify as exempt organizations under Section 501(c) (3) of the Internal Revenue Code of 1954.

Section 2. No substantial part of the activities of the Academy shall be the carrying on of propaganda, or otherwise attempting to influence legislation, and the Academy shall not participate in or intervene in, any political campaign on behalf of any candidate for public office.

Section 3. No part of any net earnings shall inure to the benefit of, or be distributable to, Academy members or officers, or other private persons, except that the Academy may authorize the payment of reasonable compensation for services rendered.

ARTICLE VI - *Amendments*

Section 1. This Constitution may be amended at any annual Business Meeting of the Academy by a two-thirds vote. Proposed amendments shall be submitted in writing to the Secretary-Treasurer who shall send them to the members at least two weeks before the meeting at which such amendments are to be considered.

Section 2. Bylaws may be adopted or repealed at any regular business meeting by a two-thirds vote.

BYLAWS

BYLAW 1. *Meetings*

Section 1. *Scientific Meetings*. The Academy shall hold at least one annual scientific meeting each year at a time and place determined by the Council. Other scientific meetings, regional, state, or local, may be held at times and places determined by the Council. The Council shall establish regulations governing the presentation of papers at Academy sessions. Such regulations shall be made available to members at least three months before any meeting at which they are to apply.

Section 2. *Business Meetings*. A Business Meeting of the membership shall be scheduled at the regular, annual scientific meeting of the Academy. Ten percent of the active members shall constitute a quorum at the annual

business meeting.

Section 3. *Special Meetings*. Special meetings shall be called by the President upon the request of ten percent of the active members and require twenty percent of the active members for a quorum. Notice of the time and place of such meetings shall be sent to all members of the Academy at least four weeks in advance of the meeting. Only matters specified in the call can be transacted at a special meeting.

Section 4. *Procedure*. Parliamentary procedures to be followed in all business meetings shall be those specified in "Standard Code of Parliamentary Procedure" by Alice F. Sturgis.

BYLAW 2. *Financial*

Section 1. *Dues and Assessments*. The annual dues and assessments may be changed from time to time by the Council, subject to approval by a two-thirds vote of the members at an annual Business Meeting. The student member dues shall be one-third (to nearest dollar) of the regular member dues. These dues are payable 1 December of each year.

Section 2. *Supporting Members*. Council shall maintain a program to encourage members to voluntarily contribute funds over and above the regular dues and assessments for the support of activities of the Society.

Section 3. *Sustaining Members*. Any association, corporation, institution, or individual desiring to support the Society with funds or services valued at $50 or greater may be invited by the President or designee to become a Sustaining Associate.

Section 4. *Audit and Reports*. The Nominating Committee shall appoint on a yearly basis one member who is not a member of Council to conduct at least one internal audit per year. The Secretary-Treasurer shall report on the financial affairs of the Society, including the results of an annual audit, as may be requested by the Council.

BYLAW 3. *Membership*

Section 1. *Membership Categories*. Classes of membership shall include the following: (a) Regular, (b) Student, (c) Emeritus, (d) Honorary, (e) Supporting, (f) Sustaining, and (g) Lifetime Members.

Section 2. *Eligibility and Procedure for Membership*. Candidates for membership, except Sustaining Member, may be proposed by any regular or emeritus member of the Academy by submitting the candidate's name to the chairman of the Membership Committee.

(a) *Regular Members*. Any person who is active or interested in some field of scientific endeavor shall be eligible for regular membership. A majority vote of Council shall elect to regular membership.

(b) *Student Members*. Any student who is an undergraduate or graduate student in some field of science shall be eligible for student membership. A majority vote of Council shall elect to regular membership.

(c) *Emeritus Members*. Any member in good standing upon formal retirement is eligible for emeritus membership. A majority vote of Council shall elect to emeritus membership.

(d) *Honorary Member*s. The Academy may recognize, by awarding honorary membership, any person (nonmember or member) who has in any way made an outstanding contribution to science. It shall be the responsibility of the Membership Committee to be aware of individuals whom it would be fitting for the Academy to honor in this fashion. A two-thirds vote of members attending the annual business meeting shall elect to honorary membership.

(e) *Supporting Members*. Regular or student members may voluntarily contribute funds over and above the regular dues and assessments for the support of activities of the Society.

(f) *Sustaining Associates*. Any association, corporation, institution, or individual desiring to support the Society with funds or services valued at $50 or greater may be invited by the President or designee to become a Sustaining Associate.

(g) *Lifetime Members*. Any regular member in current good standing for at least one year may become a Lifetime Member by paying an assessment equal to 18 times the current annual dues in one lump sum or in two equal payments over the current and following year.

Section 3. *Privileges of Membership*.

(a) Voting at the annual business meeting is permitted of regular and emeritus members.

(b) Members of all categories may attend business meetings of the Academy.

(c) The Secretary-Treasurer and members of Council must be regular members in good standing.

(d) Regular, student, and emeritus members may submit abstracts or communications for scientific meetings of the Academy.

(e) Emeritus and Honorary Members shall be exempt from payment of dues.

(f) A Sustaining Member is provided a display area at the annual scientific meeting of five linear feet per $50 donation up to a maximum of 20 linear feet.

(g) Every member in good standing shall receive a printed copy or an electronic copy (if available and of equal or lesser cost than the printed copy) of the annual *Proceedings of the North Dakota Academy of Science*, the form to be determined by the member.

(h) Special offices such as Historian may be created by the unanimous vote of the regular members at the annual Business Meeting.

(i) All student research participants shall receive a properly inscribed certificate.

Section 4. *Forfeiture of Membership*.

(a) *Nonpayment of dues*. Members shall be dropped from the active list on 31 November following the nonpayment of dues during the membership year commencing the previous 1 December. A member may return to the active list by paying the current year dues.

(b) *Expulsion for Cause*. Membership may be terminated for conduct injurious to the Academy or contrary to the best interests of the Academy. The accused member shall be given an opportunity for a hearing before the Council. If a majority of the Council votes to expel the member, the action must be ratified by at least two-thirds of the members present at the next annual business meeting of the Academy. An expelled member shall forfeit all paid dues and assessments.

BYLAW 4. *Duties and Responsibilities of the Council and Council Members*

Section 1. *Council*. The Council shall meet, at the call of the President, at least twice a year. The Council shall:

(a) be the governing board of the Academy, responsible only to the membership.

(b) arrange for programs, approve committee appointments, be responsible for the fiscal affairs of the Academy, and transact such business as necessary and desirable for function and growth of the Academy.

(c) determine the location of the annual meeting three years in advance.

(d) annually appoint an Academy representative to the National Association of Academies of Science and to Section X (General) of the American Association for the Advancement of Science.

(e) shall appoint and may compensate a Secretary-Treasurer.

(f) shall appoint and may compensate an Editor of the PROCEEDINGS and other publications.

(g) shall be empowered to charge a publication fee of authors on a per page basis.

(h) shall control all activities of the Academy including grant applications.

Section 2. *President*. The President shall preside at meetings of the Council and over the annual business meeting of the Academy at the close of the regular term of office. The President shall vote only to break a tie. Unless otherwise specified, the President shall, with the approval of the Council, appoint members to serve on Standing Committees and *ad hoc* Committees, designate the chair of each Committee, and appoint representatives to other organizations. The President serves as Coordinator of the Local Arrangements Committee for the annual meeting that occurs at the end of the President's term.

Section 3. *President-Elect*. The President-elect shall be considered a vice president and shall serve as such in the absence of the President.

Section 4. *Past-President*. The retiring President shall serve as Past-President and chair of the Nominating Committee. The Past President shall serve ex officio on those committees designated by the President and shall serve in the absence of the President and President-elect.

Section 5. *Secretary-Treasurer*. The Secretary-Treasurer shall:

(1) Assist Council in carrying on the functions of the Academy including the receipt and disbursement of funds under the direction of Council.

(2) Manage the Academy Offices under Council's general supervision.
(3) Serve as Managing Editor of the *Proceedings of the North Dakota Academy of Science*.
(4) Prepare a summary of the most recent audit and a report of the Academy's current financial status. This information shall be shared with the membership at the annual business meeting and published in the PROCEEDINGS following the business meeting.
(5) Perform all other duties of the Secretary-Treasurer listed in the Bylaws.
(6) Serve as archivist and be responsible for all official records, archives, and historic material which shall be in reposit with the Secretary-Treasurer.

BYLAW 5. *Appointment, Nomination and Election of Members of Council*

Section 1. *Eligibility for Office*. All candidates for election or appointment to the Council must be regular members in good standing. Nominees for President-elect must be members who reside within easy commuting distance of the site of the annual meeting selected by the Council that occurs when the President-elect serves as President.

Section 2. *Nomination Procedures*. The Nominating Committee shall be responsible for all nominations to elective office, shall determine the eligibility of nominees, shall ascertain that nominees are willing to stand for office, and shall be required to advance to the Secretary-Treasurer at least two names for each open position as needed. Academy members shall have been encouraged to suggest nominees to the committee prior to the Committee submitting its report.

Section 3. *Election Procedures*. Election shall be by secret mail ballot. The Secretary-Treasurer shall prepare a printed ballot that bears all names submitted by the Nominating Committee, that contains a brief biography of each candidate, and that has space for write-in candidates for each office. This ballot is to be mailed to all members no later than 1 November. Each member wishing to vote must return the marked ballot in a sealed signed envelope to the Secretary-Treasurer postmarked not more than thirty days after the ballots were mailed out to members. The President shall appoint tellers, who shall count the ballots that have been received by the Secretary-Treasurer and the tellers shall present the results in writing to the President. A plurality of the votes cast shall be necessary to elect and in the case of a tie vote, the President shall cast the deciding vote. The results of the election shall be announced at the annual Business Meeting.

Section 4. *Term of Office*. A President-Elect shall be elected annually by the membership and the following years shall succeed automatically to President and Past President to constitute a three-year nonrenewable term. Three Councilors shall be elected by the membership to three-year, non-renewable terms on a rotating basis. All elected Council members shall take office at the end of the next annual Business Meeting following election and shall continue until relieved by their successors. Council is empowered to appoint and compensate a Secretary-Treasurer to successive three-year terms that commence with the beginning of the fiscal year.

Section 5. *Removal from office or position.* If for any reason any elected member of Council is unable to fulfill his/her duties, the Council member may be removed from office by two-thirds vote of Council. If for any reason the Secretary-Treasurer is unable to fulfill his/her duties, the Secretary-Treasurer may be relieved of all duties by a majority vote of Council.

Section 6. *Interim vacancies*. Should a vacancy occur in the Presidency, the Council by a majority vote shall appoint a member of the Academy able to coordinate the next annual meeting to fill the unexpired term. A retiring interim President shall succeed automatically to Past President. Should a vacancy occur in the Presidency-elect, the Council shall reassess and change the location of the coinciding annual meeting as necessary and then call for a special election by mail ballot. An interim vacancy in the Past-Presidency shall be filled by the most recently retired Past-President able to fill the duties of the Past-President. Persons appointed to fill the unexpired term of Secretary-Treasurer are expected to remain in the position for a minimum of three years. A vacancy in the office of Councilor shall be filled by a majority vote of Council until the following election at which time the interim Councilor may stand for a full three year nonrenewable term.

BYLAW 6. *Committees*

Section 1. *Standing Committees*. Standing committees shall include but not be limited to, the following: Editorial,

Education, Denison Award, Necrology, Nominating, Resolution, Membership, and Audit Committees. The President shall appoint members of committees other than the Nominating and Audit Committees.

Section 2. *Editorial Committee*. The Editorial Committee shall consist of three regular members appointed to three year terms. The duties are explained in BYLAW 7 (Publications).

Section 3. *Education Committee*. The Education Committee shall consist of five regular members and two high school teachers appointed to five year terms. The Education Committee shall work with high school students and teachers in the state, in visitation programs, Science Talent Search programs, and other programs to stimulate an interest in science by the youth of the state. It shall operate the Junior Academy of Science program and administer the AAAS high school research program.

Section 4. *Denison Awards Committee*. The Denison Awards Committee shall consist of six regular members appointed to three year terms. The Denison Awards Committee shall have as its prime duty the judging of student research and paper competitions, both undergraduate and graduate, and any other similar competitions. The committee shall also maintain the criteria to be used in the judging and selection of papers, such criteria to be circulated to prospective competitors.

Section 5. *Necrology Committee*. The Necrology Committee shall consist of three regular members appointed to three year terms. The Necrology Committee shall report to the annual meeting on those deceased during the preceding year. Obituaries may be included in the minutes of the annual meeting and/or published in the Proceedings.

Section 6. *Nominating Committee*. The Nominating Committee shall consist of the five most recent past-presidents. The major duties of the Nominating Committee are listed in BYLAW 5 (*Appointment, Nomination and Election of Members of Council*). The Nominating Committee will also administer the selection process, develop a separate funding source for a monetary award, and develop, for Executive
Committee approval, the criteria for the North Dakota Academy of Science Achievement Award.

Section 7. *Resolution Committee*. The Resolution Committee shall consist of three regular members appointed to three year terms. The Resolution Committee shall prepare such resolutions of recognition and thanks as appropriate for the annual meeting. Further, the Committee shall receive suggested resolutions for the membership and transmit such resolutions and the Committee recommendation to the membership.

Section 8. *Membership Committee*. T he Membership Committee shall consist of unlimited numbers of regular members appointed annually.

Section 9. *Audit Committee*. The Nominating Committee shall appoint on a yearly basis one member who is not a member of Council to conduct at least one internal audit per year.

Section 10. *State Science Advisory Committee*. The State Science Advisory Committee (SSAC) shall consist of five regular or emeritus members appointed to four year terms. The SSAC shall serve to direct questions of a scientific nature to the appropriate expert as requested, shall inform regional granting agencies and state and national science policymakers of its expertise and availability and shall counsel those agencies and persons upon their request. The SSAC shall adhere in particular to the guidelines described in Article V, Section 2 of the Constitution.

Section 11. *Ad hoc Committees*. The President may appoint such additional committees as may be needed to carry out the functions of the Academy. Ad hoc committees serve only during the tenure of the president who appointed them. Reports of ad hoc committees shall be presented to Council or to the annual meeting.

BYLAW 7. *Publications*

Section 1. *Editorial Committee*. Three regular members are appointed to the Editorial Committee for renewable three year terms. The Editorial Committee shall develop and recommend the Academy publication program and policies to the Council. It will assist the Editors of each official publication in reviewing manuscripts for those publications that include the *Proceedings*. Chairs of symposia will review manuscripts written for relevant

symposia.

Section 2. *Managing Editor*. The Secretary-Treasurer shall serve as the

Section 3. *Editor*. Editors shall serve three year terms. The Editors shall edit all official publications of the Academy including the *Proceedings*.

BYLAW 8. *Memorial Fund*

The Council of the Academy shall establish a J. Donald Henderson Memorial Fund and administer this fund so that the proceeds will be used to promote science in North Dakota.

BYLAW 9. *Fiscal Year*

The fiscal year of the North Dakota Academy of Science, for the purpose of financial business, shall be 1 January to 31 December.

BYLAW 10. *Achievement Award*

The Academy establishes the North Dakota Academy of Science Achievement Award to be given periodically to an Academy member in recognition of excellence in one or more of the following:
a. Nationally recognized scientific research.
b. Science education.
c. Service to the Academy in advancing its goals.
The Nominating Committee will administer the selection process, will develop a separate funding source for a monetary award, and will develop, for Council approval, the criteria for the award.

BYLAW 11. *Research Foundation*

The **North Dakota Science Research Foundation** is established as an operating arm of the Academy. The purposes of the Foundation are:
(1) to receive funds from grants, gifts, bequests, and contributions from organizations and individuals, and (2) to use the income solely for the making of grants in support of scientific research in the State of North Dakota. Not less than 50% of the eligible monies received shall be placed in an endowment from which only the accrued interest shall be granted.

The foundation shall be responsible for soliciting the funds for the purposes described. The Foundation funds shall be in the custody of the Secretary-Treasurer of the Academy and shall be separately accounted for annually. The Foundation Board of Directors shall be comprised of five members of the Academy, representing different disciplines. Members shall be appointed by the President of staggered five year terms. The chairperson of the Board shall be appointed annually by the President. The Board shall be responsible for developing operating procedures, guidelines for proposals, evaluation criteria, granting policies, monitoring procedures, and reporting requirements, all of which shall be submitted to the Executive Committee for ratification before implementation.

The Foundation shall present a written and oral report to the membership of the Academy at each annual meeting, and the Secretary-Treasurer shall present an accompanying financial report.

BYLAW 12. *Affiliations*

The Academy may affiliate itself with other organizations which have purposes consistent with the purposes of the Academy. Such affiliations must be approved by the Council and by a majority of those attending a regularly scheduled business meeting of the membership.

BYLAW 13. *Indemnification*

Section 1. Every member of the Council or employee of the North Dakota Academy of Science shall be indemnified by the Academy against all expenses and liabilities, including counsel fees, reasonably incurred or imposed upon him/her in connection with any proceedings to which he or she may be made part, or in which he or

she may become involved, by reason of being or having been a member of the Council, or employee at the time such expenses are incurred, except in such cases wherein the member of the Council or employee is adjudged guilty of willful misfeasance or malfeasance in the performance of his or her duties. Provided, however, that in the event of a settlement of the indemnification herein shall apply only when the Council approves such settlement and reimbursement as being for the best interests of the Academy. The foregoing right of indemnification shall be in addition to and not exclusive of all other rights to which such members of the Council or employee may be entitled.

MINUTES (UNAPPROVED) OF THE NORTH DAKOTA ACADEMY OF SCIENCE ANNUAL BUSINESS MEETING

Valley State University, Valley City, North Dakota, April 27, 2006, 12:00 pm

(To be distributed on April 12, 2007 and published in Volume 62 (2008))

AGENDA/NOTES

Notes for the 99th Annual Business meeting

The first order of business was to approve the minutes of the previous business meeting from the April 2006 annual meeting in Valley City, North Dakota.

The minutes: were | were not approved
as printed in the Proceedings | as amended.

A brief financial report was presented by Secretary-Treasurer Detke. At this time, the Academy is financially sound. Secretary-Treasurer Detke noted, however, that a large fraction of the membership failed to pay dues for 2007 despite repeated reminders by email. In order to cover the fixed costs of the annual meeting and conducting Academy business throughout the calendar year and because inflation has continued since the dues were last raised in 2004, Secretary-Treasurer Detke suggested that the academy increase the Professional dues by $5, the student dues by $2 dollars and life dues by $50. A discussion of this: did | did not ensue and a motion was proposed and seconded to raise the dues by this amount and was approved | not approved by voice vote. The increased dues will become effective in 2008.

Secretary-Treasurer Detke noted that the membership role was incomplete and that the names of many of the newer members that joined since 2004 were lost from our database. He urged those in attendance to contact their Academy colleagues who joined in the years 2005-2006 to make themselves known to the Secretary-Treasurer so that their names may be placed on our roster.

Douglas Munski's tenure as counselor ended in 2008. (Person to be name) has volunteered and has been elected without opposition by voice vote.

(Person to be name) has volunteered to be President in 2009. He | She was elected without opposition by voice vote.

Meeting statistics: _____ Registered attendees ( ) professional, ( ) student
_____ Guests

We also had ( ) professional talks and ( ) Denison papers presented, of which ( ) were graduate and ( ) were undergraduate.

A. Rodger Dennison Award winners:

Graduate category:

Undergraduate category:

A. Rodger Dennison Award runner ups:

Graduate category:

Undergraduate category:

Christopher Keller (Minot State University) officially ended his duties as President by introducing Van Doze (University of North Dakota). President Doze discussed preliminary plans for the Academy's 100th Annual Meeting, over which he will preside in Grand Forks on April ( ) and ( ), 2008.

Executive Committee Membership

President
Past-President
President-Elect
Secretary-Treasurer
Councilors (three-year terms)

President
Christopher Keller
Department of Biology
Minot State University
Minot, ND
(701)858-3067
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President-Elect
Van Doze
Department of Pharmacology
Physiology & Therapeutics
University of North Dakota
Grand Forks, ND 58203
(701)777-6222
vdoze@medicine.nodak.edu

Past-President
Andre DeLorme
206 Rhoades Science Center
Valley City State University
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Secretary-Treasurer
Siegfried Detke (2005-2008)
Department of Biochemistry & Molecular Biology
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Councilors
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Birgit M Pruess (2005-2008)
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COMMITTEES OF THE NORTH DAKOTA ACADEMY OF SCIENCE

Executive Committee
Editorial Committee*
Education Committee*
Denison Awards Committee*
Necrology Committee*
Nominating Committee
Resolution Committee*
Membership Committee*
North Dakota Research Foundation Board of Directors*

* indicates available openings

PAST PRESIDENTS AND THE LOCATIONS OF THE ANNUAL MEETING

OF THE NORTH DAKOTA ACADEMY OF SCIENCE

1909	M A Brannon	Grand Forks
1910	M A Brannon	Fargo
1911	C B Waldron	Grand Forks
1912	L B McMullen	Fargo
1913	Louis VanEs	Grand Forks
1914	A G Leonard	Fargo
1915	W B Bell	Grand Forks
1916	Lura Perrine	Fargo
1917	A H Taylor	Grand Forks
1918	R C Doneghue	Fargo
1919	H E French	Grand Forks
1920	J W Ince	Fargo
1921	L R Waldron	Grand Forks
1922	Daniel Freeman	Fargo
1923	Norma Preifer	Grand Forks
1924	O A Stevens	Fargo
1925	David R Jenkins	Grand Forks
1926	E S Reynolds	Fargo
1927	Karl H Fussler	Grand Forks
1928	H L Walster	Fargo
1929	G A Talbert	Grand Forks
1930	R M Dolve	Fargo
1931	H E Simpson	Grand Forks
1932	A D Wheedon	Fargo
1933	G C Wheeler	Grand Forks
1934	C I Nelson	Fargo
1935	E A Baird	Grand Forks
1936	LR Waldron	Fargo
1937	J L Hundley	Grand Forks
1938	P J Olson	Fargo
1939	ED Coon	Grand Forks
1940	J R Dice	Fargo
1941	F C Foley	Grand Forks
1942	F W Christensen	Fargo
1943	Neal Weber	Grand Forks
1944	E A Helgeson	Fargo
1945	W H Moran	Grand Forks
1946	J A Longwell	Fargo
1947	A M Cooley	Grand Forks
1948	R H Harris	Fargo
1949	R B Winner	Grand Forks
1950	R E Dunbar	Fargo
1951	A K Saiki	Grand Forks
1952	Glenn Smith	Fargo
1953	Wilson Laird	Grand Forks
1954	C O Clagett	Fargo
1955	G A Abbott	Grand Forks
1956	H B Hart	Jamestown
1957	W E Comatzer	Grand Forks
1958	W C Whitman	Fargo
1959	Arthur W Koth	Minot
1960	H J Klosterman	Fargo
1961	Vera Facey	Grand Forks
1962	J F Cassel	Fargo
1963	C A Wardner	Grand Forks
1964	Fred H Sands	Fargo
1965	P B Kannowski	Grand Forks
1966	Paul C Sandal	Fargo
1967	F D Holland, Jr	Grand Forks
1968	W E Dinusson	Fargo
1969	Paul D Leiby	Minot
1970	Roland G Severson	Grand Forks
1971	Robert L Burgess	Fargo
1972	John C Thompson	Dickinson
1973	John R Reid	Grand Forks
1974	Richard L Kiesling	Fargo
1975	Arthur W DaFoe	Valley City
1976	Donald R Scoby	Fargo
1977	Om P Madhok	Minot
1978	James A Stewart	Grand Forks
1979	Jerome M Knoblich	Aberdeen, SD
1980	Duane O Erickson	Fargo
1981	Robert G Todd	Dickinson
1982	Eric N Clausen	Bismark
1983	Virgil I Stenberg	Grand Forks
1984	Gary Clambey	Fargo
1985	Michael Thompson	Minot
1986	Elliot Shubert	Grand Forks
1987	William Barker	Fargo
1988	Bonnie Heidel	Bismark
1989	Forrest Nielsen	Grand Forks
1990	David Davis	Fargo
1991	Clark Markell	Minot
1992	John Brauner	Grand Forks
1993	John Brauner	Jamestown
1994	Glen Statler	Fargo
1995	Carolyn Godfread	Bismark
1996	Eileen Starr	Valley City
1997	Curtiss Hunt	Grand Forks
1998	Allen Kihm	Minot
1999	Joseph Hartman	Grand Forks
2000	Mark Sheridan	Moorhead, MN
2001	Ron Jyring	Bismark
2002	Jody Rada	Grand Forks
2003	Richard Barkosky	Minot
2004	Anna Grazul-Bilska	Fargo
2005	Holly Brown-Borg	Grand Forks
2006	Andre Delorme	Valley City
2007	Chris Keller	Minot

A

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1/1/2007 - 3/27/2007

ASSETS	
Operating Accounts	
Checking	$5,951.67
Trust Accounts	
Scholarship (Savings)	$11,365.22
Scholarship (Stocks)	$65,549.38
Research Foundation (Savings)	$4,457.06
Total	$87,323.33
DUES	
Reinstatements	$25.00
Current year	$1,230.00
Sponsor/Patron	$0.00
Total	$1,255.00
INSTITUTIONAL SUPPORT	
NDUS TOTAL	$0.00
ANNUAL MEETING	
Registration fees	$2,910.00
AWARDS PROG RAM	
Scholarship Dividends	$0.00
NDAS Research Foundation	$0.00
Total	$0.00
PUBLICATION SALES	$83.00
MISCELLANEOUS INCOME	
Interest	$ 30.93
Dividend Income (Reinvested)	$288.61
Total	$319.54
MEMBERSHIP	2007 estimates
Emeritus	Not available
Students	38
Professional	35
Delinquent	70+
Withdrew	3
ANNUAL MEETING	
Speakers Expenses	$0.00
Meals/Refreshments	$0.00
Printing	$0.00
General Expenses	$0.00
Total	$0.00
AWARD PROGRAMS	
ND Science/Engineering Fair	$50.00
Denison	$0.00
Total	$50.00

PUBLICATION	
Proceedings	$0.00
Total	$0.00
OFFICE EXPENSES	
Postage	$0.00
Post Office Box Rental	$39.00
Duplication	$59.59
Supplies	$0.00
Other	$0.00
Bank Fees	$0.00
Total	$98.59
MISCELLANEOUS	
Fidelity Bond	$100.00
ND annual Report	$70.00
Total	$170.00
SCIENCE RESEARCH FOUNDATION	
CASH INCOME	$0.00
Donations from Members	$0.00
Allocations from Dues	$0.00
Interest Accrued	$0.00
Sponsors/Patrons	$0.00
Total	$0.00
CASH EXPENSE	
Grants	$0.00
Total	$0.00
SCHOLARSHIP FUND	
CASH INCOME	$0.00
Sempra Energy (Dividend)	$210.25
Alliant Energy (Dividend)	$78.36
Total	$288.61
ASSETS	
Sempra Energy (purchased as ENOVA	
Number of shares 250 (1983)	979.586
Price 18.50	$56.14
Value $4,625.00	$55,082.13
IEC/Alliant Energy (purchased as IES Industries	
Number of shares 120 (1990)	246.811
Price 31.63	$42.41
Value $3,795.60	$10,467.25
Total Investment Value	$65,549.38

Agenda for Business meeting
Thursday April 12, 2007 4:30

1) Approval of the minutes for the 2006 NDAS Annual Meeting in Valley City

2) Old business

3) Election of President-elect

4) Election of Councilor(s)

5) Changes in By-Laws

A) Proposed change #1 in BYLAW 2. (*Financial)*

Create new section which will be referred to as section 1. The other sections will be renumbered accordingly.

"The fiscal year shall run concurrently with the calendar year from January 1 to December 31."

I suggest this because Section 1 now states "These dues are payable 1 December of each year." It is not clear if the dues are to be paid for the current year by the previous December or December of the current fiscal year.

B) Proposed change #2 in BYLAW 2. (Financial) Section 1.

Delete "~~These dues are payable 1 December of each year."~~ and replace it by "These dues are payable by January 31 for the current fiscal year or by the Annual North Dakota Academy of Science Meeting for those registering for the meeting". We need the dues early to pay for conducting NDAS business but many like to write one check for dues and registration for the annual meeting. This should accommodate them.

C) Proposed change #3 in BYLAW 2. (Financial) Section 1.

Delete ~~"The student member dues shall be one-third (to nearest dollar) of the regular member dues."~~ We want to keep dues manageable but this locks us in to a ratio that limits us as to what we can request. We either have to keep student dues very low or increase the dues for the professional category much more to compensate.

D) Proposed change in dues to become effective January 1, 2008.

BYLAW 2. (*Financial)* Section 1. *Dues and Assessments*. "The annual dues and assessments may be changed from time to time by the Council, subject to approval by a two-thirds vote of the members at an annual Business Meeting".

The Council has discussed this and has come to the consensus that our Dues are too low. The last time dues were increased was in 2004 and inflation has taken its toll since then. It was suggested that Student's dues be increased from $10 to $12 and Professional from $25 to $30 dollars.

6) Other new business

7) Adjourn