## Annexure A

+1 416-963-4962

Select Page

# Suitable Students

## Is the Arrowsmith Program suitable for you or your child?

Students entering Arrowsmith School have ordinarily been experiencing a range of problems including

- reading
- writing
- mathematics
- comprehension
- logical reasoning
- visual memory
- auditory memory
- dyslexia
- non-verbal learning
- auditory processing
- attention

We suggest that parents or students review the list of **Learning Dysfunctions Addressed** which contains a list of the learning problems that our program addresses and their common features.

## The typical student at Arrowsmith School

- is of average or above average intelligence
- has a combination of the learning dysfunctions that are described in the **Descriptions of Learning Dysfunctions** on our web site and in our **brochure** does not have severe intellectual, cognitive, emotional or behavioural disorders that would significantly affect his or her ability to participate in the Arrowsmith Program.
- does not have acquired brain injury or an autism spectrum disorder
- is of elementary, secondary or post-secondary school age

These are guidelines only. There are many students who fall within these guidelines; others who may require further consideration and still others for whom we feel this program cannot provide meaningful benefit.

For example, we do not accept children with severe autism but we have had success with high functioning students with Asperger Syndrome. Some professionals consider Asperger Syndrome to be the same as or similar to high functioning autism, others associate it with non-verbal learning disorder. There is no hard and fast rule and we will consider their appropriateness for our program in consultation with the student's parents.

These considerations apply equally to students enrolling at one of the schools that offer our program.

If you are not familiar with the work of Arrowsmith School, we invite you to view our Introductory **VIDEO**. This fifteen-minute introduction provides an overview of our program and we hope it will answer many of your questions about what we do and the suitability of our program for your child or for yourself.

If it appears you or your child may be a suitable candidate for the Arrowsmith Program, please contact us by email at **reception@arrowsmithschool.org** or by telephone to Daina Luszczek, Arrowsmith School Secretary, at 416 963-4962 and she will direct you to the appropriate person.

## Questionnaire

Complete the Arrowsmith Program® Cognitive Profile Questionnaire.


4 x 3 =


12
1
2
3
4
5
6
7
8
9
10
11
?


Arrowsmith SchoolsArrowsmith Program

Toronto Campus
Peterborough Campus
Programs
Recognize the Signs
Information Sessions
FAQs
Parent Log-in

Learning Dysfunctions Addressed
Implementation Guidelines
Participating Schools
Prospective Schools
FAQs

## Background

- Barbara Arrowsmith Young
- Neuroplasticity
- Research
- Methodology
- Trademarks
- Advocacy
- Implementation Guidelines

## About

- Assessments
- Cognitive Questionnaire
- Employment
- Media Room
- Events
- Dare to Dream Fundraiser

## Contact

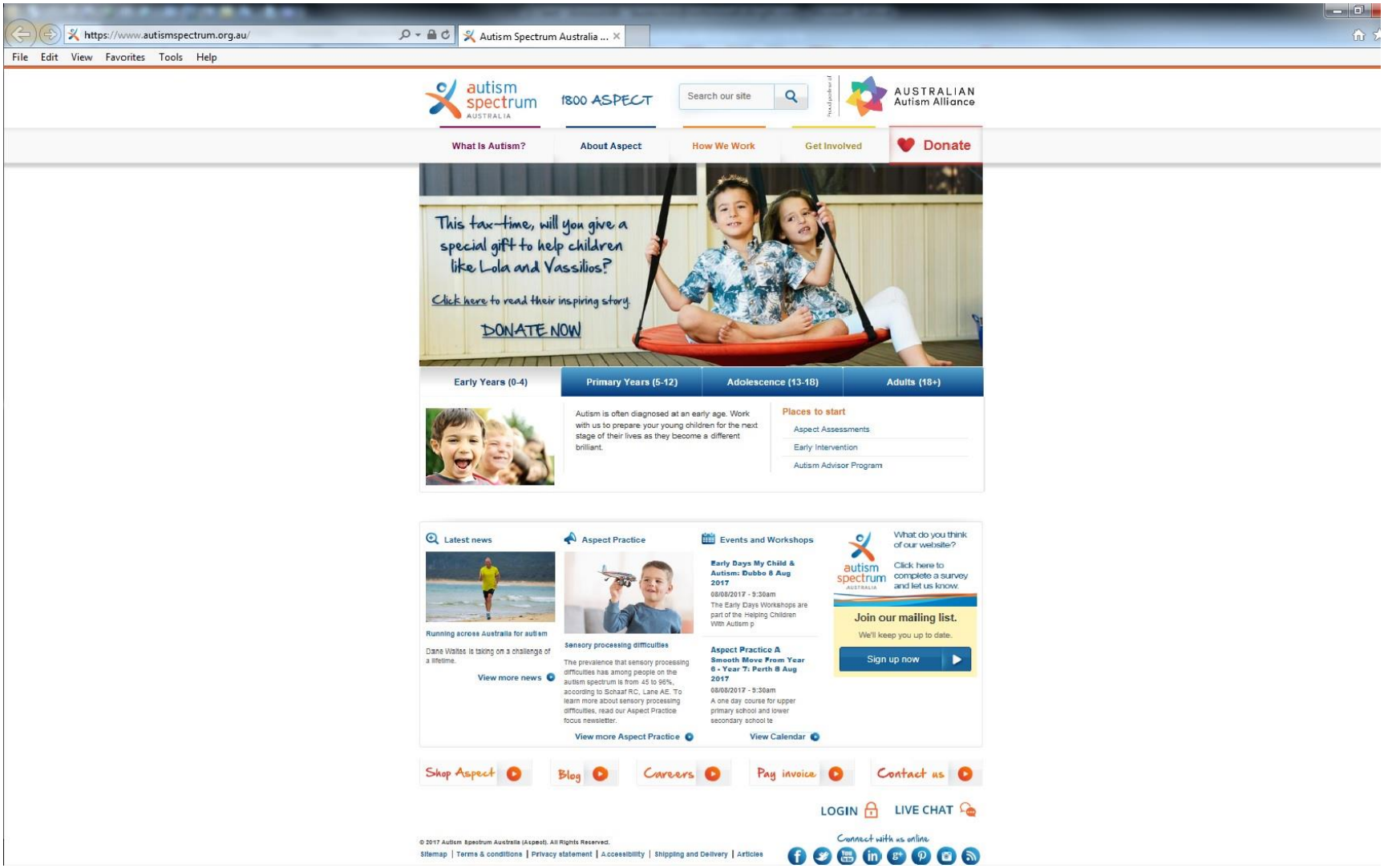
- Arrowsmith Program
- Toronto School
- Peterborough School
- Assessments

Search

Privacy Policy

Site Map

## Annexure B


https://www.autismspectrum.org.au/
Autism Spectrum Australia ...
File Edit View Favorites Tools Help
autism spectrum AUSTRALIA
1800 ASPECT
Search our site
AUSTRALIAN Autism Alliance
What Is Autism?
About Aspect
How We Work
Get Involved
Donate
This tax-time, will you give a special gift to help children like Lola and Vassilios?
Click here to read their inspiring story.
DONATE NOW
Early Years (0-4)
Primary Years (5-12)
Adolescence (13-18)
Adults (18+)
Autism is often diagnosed at an early age. Work with us to prepare your young children for the next stage of their lives as they become a different brilliant.
Places to start
Aspect Assessments
Early Intervention
Autism Advisor Program
Latest news
Running across Australia for autism
Dane Waites is taking on a challenge of a lifetime.
View more news
Aspect Practice
Sensory processing difficulties
The prevalence that sensory processing difficulties has among people on the autism spectrum is from 45 to 96%, according to Schaaf RC, Lane AE. To learn more about sensory processing difficulties, read our Aspect Practice focus newsletter.
View more Aspect Practice
Events and Workshops
Early Days My Child & Autism: Dubbo 8 Aug 2017
08/08/2017 - 9:30am
The Early Days Workshops are part of the Helping Children With Autism p
Aspect Practice A Smooth Move From Year 6 - Year 7: Perth 8 Aug 2017
08/08/2017 - 9:30am
A one day course for upper primary school and lower secondary school te
View Calendar
What do you think of our website?
Click here to complete a survey and let us know.
Join our mailing list.
We'll keep you up to date.
Sign up now
Shop Aspect
Blog
Careers
Pay invoice
Contact us
LOGIN
LIVE CHAT
© 2017 Autism Spectrum Australia (Aspect). All Rights Reserved.
Sitemap | Terms & conditions | Privacy statement | Accessibility | Shipping and Delivery | Articles
Connect with us online

## Annexure C

Catholic Education Commission NSW

**21 April 2017**

# Memorandum

**To:** Contact Officer,
Legislative Council Inquiry into Students with a Disability or Special Needs in NSW
(gpscno3@parliament.nsw.gov.au)

**From:** Ian Baker, Director – Education Policy and Programs

**Subject:** **CECNSW "Questions on Notice" arising from the Public Hearing held Monday 27 March 2017 from 3pm**

On Monday 27 March 2017, CECNSW together with AISNSW attended a session of the Students with Disability or Special Needs Inquiry's Public Hearings. Commission attendees were:

- Ian Baker, CECNSW
- Geraldine Gray, CECNSW
- Tony Fitzgerald, Mater Dei Special School Camden

As highlighted in the transcript text provided to CECNSW following the hearing, the hearing resulted in a set of 'Questions on Notice' being identified for CECNSW response by COB Friday 21 April 2017.

Of the questions for further advice:

1) Two related to Commonwealth Royal Commission into Institutional Responses to Child Sexual Abuse proceedings: these questions are addressed, refer attachment 1.

2) One related to how the Inquiry might progress its objectives, refer attachment 2.

3) Three related to the 'Arrowsmith Program': these questions have been addressed by Sydney Catholic schools, refer Attachment 3. The Inquiry is advised that only one of the eleven NSW Diocesan School Authorities is involved with this program, being the Archdiocese of Sydney.

In addition, one question related to the 2016 Productivity Commission Report on Government Services in respect of childcare, schooling and training. The CEC secretariat has reviewed this Report. The relevant section of the Productivity Commission report is available at http://www.pc.gov.au/research/ongoing/report-on-government-services/2016/childcare-education-and-training/school-education/rogs-2016-volumeb-chapter4.pdf

Relevant Report data includes:

p4.9 says

*Students with disability are educated in both mainstream and special schools. Nationally in 2014, the proportion of students with disability for all schools was 5.3 per cent, and significantly higher in government schools (6.1 per cent) than in non-government schools (3.9 per cent) (figure 4.2).*

P4.74 supports the CECNSW statement that datasets differ

*Nationally consistent data on students with disability is under development for future reporting*.

…so does P4.77

*Students included in the annual system reports to the Department of Education and Training. The definitions of students with disabilities are based on individual State and Territory criteria, so data are not comparable across jurisdictions*.

Table 4A.31 shows how many funded students with a disability each sector (19,073 in non-govt schools, out of 67,665) and proportion (6.4% in govt schools, 4.7% in non-govt schools)

Table4A.33 shows proportion of SWDs increasing in non-government schools (from 3.9% in 2010 to 4.7% by 2014).

With respect to this data, CECNSW notes that:

(a) It is dated, being 2014 data (Cited in a 2016 report)

(b) The Productivity Commission itself recognises (p 4.74) that relevant datasets differ (Mr Baker's point at the hearing) and that "data (sets) are not comparable across jurisdictions" (p 4.77)

(c) Both CECNSW and the Productivity Commission are in agreement that jurisdictional and sector comparisons of SWD enrolment data and trends will only be possible once the Australian Department of Education and Training 'Nationally Consistent Collection of Data on School Students with Disability (NCCD)' has been fully implemented (p 4.74)

With respect Royal Commission into Institutional Responses to Child Sexual Abuse matters, the following response points are made:

(i) The responses set out in Attachment 1 derive from one NSW Diocesan School System but are illustrative of state-wide response processes.

(ii) All NSW Catholic schools are compliant with the School Registration provisions of section 47 (g) of the NSW Education Act 1990 being:

> *(g) a safe and supportive environment is provided for students by means that include:*
>
> *(i) school policies and procedures that make provision for the welfare of students, and*
>
> *(ii) persons who are employed at the school being employed in accordance with Part 2 of the Child Protection (Working with Children) Act 2012 , and*
>
> *(iii) school policies and procedures that ensure compliance with relevant notification requirements imposed in relation to persons employed at the school by Part 3A of the Ombudsman Act 1974 and the Child Protection (Working with Children) Act 2012 , and*
>
> *(iv) maintaining a student enrolment and attendance register,*

(iii) CECNSW is currently working with the Office of NSW Children's Guardian consultation for the finalisation of a set of NSW 'Principles for Child Safe Organisations', in response to Royal Commission criteria for child-safe institutions. https://www.kidsguardian.nsw.gov.au/child-safe-organisations In this context, CECNSW supports the independent oversight of child protection planning by schools, noting the current involvement of NESA, the NSW Ombudsman and the Office of the NSW Children's Guardian.

Also CECNSW wishes to remind the Inquiry that NSW Catholic Schools are currently fully engaged with the NSW Ombudsman's Inquiry into 'Evidence – based behaviour management in government and non-government schools' (Refer CECNSW submission to the current Legislative Inquiry at section 5c)

Finally, with respect to question 2 above, "how the Inquiry might progress its objectives", the Commission restates the advice set out in section 7 of its submission that:

i. *The conduct and purpose of the separate State (SWD) and National (NCCD) data collections needs to be clarified by the Australian Education Council, refer section 3.*

ii. *The non-government schools sectors need to be included in the membership of all relevant NSW NDIS planning committees, refer section 3.3.*

iii. *Student identification data suggests that disability services access is still an equity issue for rural schools, refer section 4.4.*

iv. *The National NCCD collection is not yet reliable or robust enough to be used as a funding tool at any level of decision making, refer section 4.*

v. *The existing parent complaint processes established and oversighted by NESA should be better advised to parents by NESA, refer section 5.*

vi. *Existing NSW Supervisor subsidy grant eligibility should be reformed so that it is based on assessed student need only, rather than on both student need and the type of school a student attends, which are the present criteria, refer section 2 - "Recommendation 4".*

vii. *CECNSW is of the view that additional and particular Guidelines for all NSW schools are required that will enable both schools and parents to understand the processes that must be followed and the factors to be taken into account when determining what reasonable adjustments are required to manage behaviour and whether these adjustments are reasonable or unreasonable. These proposed NSW Guidelines should assist all parties to appreciate that a school is not required to implement unreasonable adjustments, see "Disability Standards for Education 2004, Guidance Notes" at 4.2 – "making reasonable adjustments" and "no requirement to make unreasonable adjustments", refer section 6.*

Should the Inquiry wish to further explore these matters please contact me by email ian.baker@cecnsw.catholic.edu.au

**Truth Justice Healing Council**

**Diocese of Broken Bay - examination of the Church's response to child sexual abuse**

## STRUCTURES, POLICIES AND PROCEDURES

| **Safeguarding against Child Sexual Abuse: Structures, policies and procedures** - *(Number 8 on TJHC Information Template)* | | | |
|---|---|---|---|
| **What structures, policies and procedures are in place to safeguard against child sexual abuse in your area?** | | | |
| **Detail information about the structure, policy, procedure or practice in place in your area** | **Category** (structure, policy, procedure, practice, form etc) | **Are there any structures, policies or procedures currently being developed?** | **Are there any planned?** |
| **1. Dedicated CSO Child Protection Team employed with specialist child protection practitioners -** The Catholic Schools has a dedicated child protection team consisting of a Child Protection Team Leader, two Child Protection Officer's and a Child Protection Administrative Assistant servicing the 44 systemic catholic schools in the Diocese of Broken Bay. The practitioners all have significant child protection experience spanning the past 30 years. The team is responsible for all areas of child protection in schools including supporting Principals in managing significant risk of harm and wellbeing concerns of students, responding to allegations against staff, managing the Working with Children Check, delivering child protection professional development to all staff, producing all child protection policies and procedures, undertaking audits under the School Review and Development process, contr buting to submissions for the Royal Commission, networking and offering advice and support to other practitioners and agencies in the child protection sector. | Practice | N/A | Consideration of appointing a member of staff as a "Child Safe champion" at each school to work alongside the child protection team in ensuring each school site has a safe and supportive environment for students. |
| **2. Child Protection Policies regarding Risk of Significant Harm, Allegations Against Employees and the Working With Children Check -** The CSO has three dedicated child protection policies in place. The "Managing Risk of Significant Harm and Wellbeing Concerns" policy outlines the processes for responding to students at risk of significant harm under the *Children and Young Persons (Care & Protection) Act 1998.* The "Addressing Allegations of Inappropriate Behaviour By Staff" policy outlines our obligations under Part 3A of the *Ombudsman Act 1974.* The "Working with Children Check" policy documents the requirements under the *Child Protection (Working with Children) Act 2012* . There are also other related CSO policies in place such as the DSS Pastoral Care Policy, DBB Pastoral Care and Wellbeing Framework and DSS Social Media Policy which contr bute to safeguarding children against sexual abuse. In 2007 a comprehensive Child Protection Resource Manual was launched for Diocesan Systemic Schools. The manual was designed to provide practical guidance to assist all school staff in fulfilling their child protection responsibilities. The manual sets out the procedures to follow for making reports to the child protection statutory authority and gives clear guidance as to how allegations of inappropriate conduct involving a staff member are to be handled. | Policy | A Child Protection "weebly" is currently being developed to enhance the availability of all child protection related forms, processes, and policy documentation for staff. It will be designed to be a one-stop shop for school staff to access relevant material in relation to meeting their child protection obligations. This will replace the current Child Protection Resource Manual for Diocesan Systemic Schools which is now outdated. | N/A |

| | | | |
|---|---|---|---|
| **3. Child Protection protocols and procedures in relation to Risk of Significant Harm, Allegations Against Employees and the Working With Children Check -** The CSO has a large variety of protocols and procedures in place to address all three compliance areas of child protection.<br><br>Managing Risk of Significant Harm - The CP team provides a specialist service to schools so that Principals, School Counsellors or other school staff can consult with a Child Protection Officer to receive advice on whether a student needs to be reported to the Department of Family & Community Services (FACS) or if the threshold for risk of significant harm (ROSH) is not met, support can be provided in relation to managing the wellbeing concerns of a child or young person. Data and records are kept on all consultations and reports made to FACS via reporting forms and the CP Team ROSH database. A case management service is also provided to schools when managing serious or complex child protection cases (such as student on student abuse) whereby a child protection officer or member of the Pastoral Care & Wellbeing Team is allocated to the school to attend parent meetings, liaise with FACS/NSW Police and refer to specialist services. Chapter 16A information exchange provisions are also in place where necessary with other prescribed bodies.<br><br>Working with Children Check (WWCC) - The CP team oversees the implementation of the Working with Children Check for the whole of the diocesan systemic school system and Catholic Schools Office. "Guidelines for Schools for the Working with Children Check" were developed in 2013 following the introduction of the new Working with Children Check scheme. These guidelines assist schools and CSO staff in determining who needs a WWCC and when they need to have a WWCC completed. Specific procedures are in place at each school in relation to managing volunteers and contractors and all exempt volunteers and contractors who do not require a WWCC are required to complete a Declaration to confirm their suitability to be engaged in child-related work. Verifications of all WWCC's are completed centrally by the CSO Human Resources Team and a spreadsheet and database records are maintained of cleared and barred persons. Classification of all CSO positions occurred in 2016 and certain positions were approved to be "deemed" child related work by the Office of the Children's Guardian.<br><br>Allegations Against Employees - The CP Team is responsible for handling all allegations of reportable conduct against employees. Schools are required to report all matters directly to the Child Protection team. When an allegation amounts to a reportable conduct concern the CP Team is responsible for conducting the workplace investigation. For exemption matters, Principals are provided with support to complete the investigation with oversight from the CP Team. Specific Head of Agency protocols are in place for managing allegations against employees including not in jurisdiction matters, exemption matters and reportable conduct matters. Standard templates, forms, flowcharts, support documents and checklists are in place including risk assessment forms, investigation plans, jurisdiction determination form, investigation report, template letters and guidesheets. All matters are recorded on the Head of Agency Child Protection database, including "intelligence" matters which may include historical reports of sexual abuse. | Protocols and procedures | N/A | In recognition of the limited involvement that the CSO has in place for allowing meaningful contributions from children and young people in the development of practices that affect them, further programs (in conjunction with the Pastoral Care & Wellbeing Team) are to be developed that allow for the voice of the student to be heard more prevalently. |
| **4. Training for employees and professional development for CP practitioners -** the Child Protection team has a detailed professional development strategy in place to train all school and CSO staff. The model, also aligned with the School Review and Development Program allows for schools who are on external review (approximately 8 per year) to receive two, 1.5 hour face to face sessions regarding "Promoting Child Safety - Managing Risk of Significant Harm Concerns" and "Promoting Child Safety - Allegations Against Employees". Schools who are on self review (approximately 7 per year) receive a 45 minute face to face upskilling session on a child protection related topic relevant to their school. Upskilling sessions include, Dealing with problematic sexualised behaviour at schools, Addressing self-harm/mental health issues of students, Setting professional boundaries with students, Calmer Classrooms – engaging children affected by trauma, How to handle disclosures of a child protection nature, Practical case studies/scenarios & the Child Protection Helpline, Neglect – a closer look at neglect as a category of abuse, Physical abuse – a closer look at physical harm as a category of abuse, Sexual abuse – a closer look at sexual harm as a category of abuse, Psychological harm – a closer look at psychological harm as a category of abuse, Sexting – looking at the problem of sexting and young people. The remaining schools (29 schools) complete two online modules of SALT compliance training in relation to Child Protection. Essentially this equates to all 44 schools in the Diocese completing some form of child protection training annually. In addition orientation sessions held for all new school staff to the Diocese are held at the commencement of each school year in regards to an overview of child protection responsibilities in relation to managing risk of significant harm and allegations against employees. Child protection presentations are also provided to Catholic Schools Office "field" staff and leadership on an as needs basis.<br><br>Child Protection Practitioners are also actively engaged in annual professional development opportunities addressing the complex area of child sexual abuse and creating child safe organisations including attending conferences, symposiums and presentations by imminent international experts such as Monica Applewhite and Ian Elliott and prominent Australian presenters such as Gerard Webster. | Practice | N/A | A more comprehensive professional development approach for volunteers in schools is required. |
| **5. Recruitment and screening processes -** The Catholic Schools Office have in place comprehensive recruitment practices to assist the CSO being a child safe organisation. Child related job advertisements pronounce the requirement for preferred applicants to be subject to child protection screening. Application forms for all prospective employees ask specific questions in relation to any history of applicants being subject to child protection investigations. Requirements in relation to the recruitment and selection of school staff are outlined in the "Recruitment and Selection of Teaching and General Employees" Guidelines for Principals. All staff in child-related work require a Working with Children Check and before appointment specific child protection questions must be asked of referees (former employer) before proceeding with any offer of work. Any issues highlighted in the recruitment process are referred to the Child Protection team for further assessment. | Policy and procedures | N/A | A more thorough and streamlined induction process for casuals, volunteers and other new staff is in the process of being considered in conjunction with Human Resource Services at the Catholic Schools Office. |

| | | | |
|---|---|---|---|
| **6. School Review and Development program -** The purpose of the School Review and Development program is to provide support to diocesan systemic schools to ensure compliance with regulatory requirements and to provide the framework for self-evaluation and school development under the NSW Education Act 1990 which is administered by BOSTES.<br><br>Schools involved in either a Tier 2 self-review or an external panel review are reviewed by the CSO Child Protection Officer in a formal way. The CSO Child Protection Officer will:<br>- check that the school's Staff Handbook contains sections outlining procedures for:<br>- handling allegations against staff of inappropriate behaviour towards children<br>- mandatory reporting students at risk of significant harm<br>- employment screening requirements for paid staff, external service providers and volunteers<br>- check that the Child Protection Resource Manual (including the Professional Guidelines for School Staff in their Relationships with Students) and all Child Protection polices are accessible in hard or soft copy to all staff<br>- check that the Parent Handbook contains a section informing parents of the school's child protection responsibilities<br>- ensure that all schools have available, in their reception areas, a pamphlet for parents explaining to the community what the school does in terms of meeting legal child protection responsibilities<br>- ensure that staff induction manuals/packages outline child protection procedures and refer to key documents such the Child Protection Resource Manual and the Professional Guidelines for School Staff in their Relationships with Students.<br>- sight any school based CP PL records<br>- check that the School Principal maintains confidential student records involving risk of significant harm or wellbeing concerns in a lockable, restricted access filing cabinet<br>- check that appropriate Working With Children Check documents are collected at the school for all external service providers and volunteers and that their details are recorded in a database<br>- check samples of teacher personnel files for evidence of Working With Children Check clearance and referee checking. | Procedure | N/A | N/A |
| **7. Compliance reporting and data analysis -** The CSO Child Protection team prepares annual compliance reports for the CSO Director's Group providing statistics and analysis relating to allegations against employees, reports relating to risk of significant harm and wellbeing concerns and professional learning. These reports help to identify anomalies in expected reporting from schools, potential gaps in knowledge and understanding of child protection requirements, as well as other emerging trends/issues which may need to be addressed. | Practice | N/A | N/A |
| **8. Professional Networking -** The CSO Child Protection practitioners are actively involved in regular professional networking meetings with external stakeholders including the NSW Ombudsman Office, NSW Office of the Children's Guardian, Department of Family & Community Services, NSW Police Child Abuse Squad, Catholic Education Commission of NSW, Catholic Commission for Employment Relations, NSW Department of Education and other non-government agencies. The networking allows for collaborative relationships to be built and contr butions made to specific affairs that affect practice in the greater child protection sector (for example input into the review of the FACS Mandatory Reporter's Guideline and the NSW Ombudsman's Defining Reportable Conduct definitions). The Child Protection Team Leader is the current Chair of the NSW Catholic Systemic Schools Child Protection Practitioners Group. The group was established by the Conference of Diocesan Directors of Education NSW & ACT to provide advice on policy and operational matters pertaining to child protection and enable networking and support for Catholic Education Offices and Catholic Schools Offices child protection officers. | Practice | N/A | N/A |
| **9. Internal communication and relationship building -** Continual communication and relationship building occurs with schools, CSO staff and other diocesan agencies, in relation to policy, practice and developments regarding child protection. This is facilitated through regular submissions in the weekly school newsletter to Principals and CSO staff and via face to face presentations at network meetings with Principals, Assistant Principals, Senior Admin Officers, Directors Group, Pastoral Care & Wellbeing staff, and Chancery Safeguarding, etc. The constant communication and development of relationships allows for trust to be built between the schools/CSO staff/Diocesan agencies and the Child Protection Team and ensures that child protection is always at the forefront of stakeholders minds, resulting in an enhanced child safe culture within the organisation. | Practice | N/A | N/A |

| | | | |
|---|---|---|---|
| **10. Responding to historical disclosures/claims of child sexual assault -** Matters are either reported directly to the Catholic Schools Office or via contact with a school. Matters are immediately referred to the CSO Child Protection Team and Office of the Director for review. Consultation occurs in the first instance with the Chancery (Office for Chancery Services, Safeguarding) for advice and direction. Depending on the circumstances, case management will either be immediately handed over to the Office for Chancery Services, Safeguarding unit or the CSO Child Protection Team will have initial case management with oversight from Safeguarding, Office for Chancery Services. In circumstances where the CSO Child Protection Team carry the case management role, initial contact is made with the victim and where possible a meeting arranged to see what supports can be extended to the victim in terms of; police reporting obligations (noting no blind reports can be made); explaining the Towards Healing process including options for financial claims; exploring what pastoral support is required such as a formal apology from the Church; advising of rights in relation to legal support available; offering specialist counselling referrals and contact numbers for victim support groups; informing the victim of any investigation which may be required; and hearing the details of the abuse if the victim feels ready to disclose the information. | Procedure | N/A | N/A |
| **11. Referrals to specialist services -** Where there is an identified need, for example when a student requires specialist treatment for problematic sexualised behaviour due to peer on peer abuse, referrals are facilitated to prominent private practitioners who specialise in this area. Schools work with the family to ensure that support is appropriate and the needs of the student engaging in such behaviour is addressed. Victims of sexual abuse are able to access the school counselling service for generalised counselling or can also be referred to specialist services either through the Department of Family & Community Services and/or JIRT or directly through the CSO Child Protection Team or Pastoral Care & Wellbeing Team. | Practice | N/A | N/A |
| **12. Curriculum -** Broken Bay diocesan schools follow the NSW Board of Studies syllabuses. In particular the content addressing child safety and protective behaviours is part of the K-6 Personal Development, Health Education and Physical Education (PDHPE) and 7-10 PDHPE syllabuses which all schools in NSW are required to implement. In Years 11 & 12 the PDHPE syllabus is an elective course. The aim of child protection education programs is to assist students in developing skills in recognising and responding to unsafe situations, seeking assistance, establishing and maintaining relationships and strengthening attitudes and values related to equality, respect and responsibility. Schools explore the concept of recognising abuse in many ways such as physical, verbal, emotional and sexual abuse within the contexts of personal safety, resilience, bullying, cyber safety, violence, protective strategies, power in relationships and sexual harassment. In Years K-6 the CSO Broken Bay has developed sample units in PDHPE (based on the NSW syllabus) to support schools to implement the PDHPE syllabus which includes child safety programs in each stage of learning. In addition, the NSW Catholic Education Commission has produced "Towards Wholeness" K-6 and Towards Wholeness 7-10 which provide a Catholic perspective on the NSW PDHPE syllabuses. Schools may also utilise the services of external providers such as Interrelate or national curriculum available through the Office of the e-Safety Commission on "sexting" for example, to enhance the curriculum offered in child safety. | Policy | N/A | It is intended that a review will occur of curriculum's or programs currently available nationally in the area of "protective behaviour" to determine a future approach on how this specific curriculum should be delivered in Broken Bay systemic schools in the long term. |
| **13. Code of conduct -** In 2008, the Catholic Schools Office developed guidelines entitled "Professional Guidelines for School Staff in their Relationships with Students" for both teaching and support staff to assist in defining professional boundaries and acceptable practices in relation to interactions with students. These guidelines are still in place and actively promoted as the code of conduct that all school staff must adhere to. The guidelines provide information on appropriate and inappropriate behaviour in key areas of professional practice including communication with students, physical contact with students, student management and care and maintaining professional boundaries. These guidelines are typically distributed to all school staff at the beginning of every school year, provided as a resource when training is delivered, and in addition hard copies of the Guidelines must be kept in an accessible location at every school site (eg in the staff room). School staff are also expected to read and sign off that the Guidelines are understood when they undertake SALT online compliance training. | Guidelines | In 2013, the Catholic Systemic Schools Child Protection Practitioners Group under the auspices of the Conference of Diocesan Directors of Education NSW and ACT (CDD) released a new code of conduct entitled "Guidelines for Professional Conduct in the Protection of Children and Young People". The Guidelines promote a common understanding of expected behaviour for all people working in or providing services in Catholic Systemic Schools. A review of the existing "Professional Guidelines for School Staff in their Relationships with Students" document and the newly created "Guidelines for Professional Conduct in the Protection of Children and Young People" is being undertaken with a view to updating the existing Guidelines where necessary. | N/A |

| **Safeguarding Measures since the Royal Commission commenced** - *(Number 9 on the TJHC Information Template)* | | | |
|---|---|---|---|
| **What Safeguarding Measures have you introduced into your area of responsibility since the Royal Commission (January 2013) in relation to professional standards?** | | | |
| **Detail information about the measure introduced** | **Date measure introduced** | **Are there any measures currently being development?** | **Are there any planned?** |

| | | | |
|---|---|---|---|
| **Improved Professional Development strategy** - By 2013 mandatory professional development for all school employees was delivered on a 5 yearly basis (aligned with the School Review & Development program) which consisted of two, 1.5 hour face to face sessions regarding "Promoting Child Safety - Managing Risk of Significant Harm Concerns" and "Promoting Child Safety - Allegations Against Employees". These sessions were in addition to Orientation sessions held for all new school staff to the Diocese which consisted of an overview of child protection responsibilities in relation to managing risk of significant harm and allegations against employees held at the commencement of each school year. In 2015 a new professional development strategy was launched in recognition of the need to increase training to all school employees regarding their child protection responsibilities. The new model, also aligned with the School Review and Development Program allows for schools who are on external review (approximately 8 per year) to receive two, 1.5 hour face to face sessions regarding "Promoting Child Safety - Managing Risk of Significant Harm Concerns" and "Promoting Child Safety - Allegations Against Employees". Schools who are on self review (approximately 7 per year) receive a 45 minute face to face upskilling session on a child protection related topic relevant to their school. Upskilling sessions include, Dealing with problematic sexualised behaviour at schools, Addressing self-harm/mental health issues of students, Setting professional boundaries with students, Calmer Classrooms – engaging children affected by trauma, How to handle disclosures of a child protection nature, Practical case studies/scenarios & the Child Protection Helpline, Neglect – a closer look at neglect as a category of abuse, Physical abuse – a closer look at physical harm as a category of abuse, Sexual abuse – a closer look at sexual harm as a category of abuse, Psychological harm – a closer look at psychological harm as a category of abuse, Sexting – looking at the problem of sexting and young people. The remaining schools (29 schools) complete two online modules of SALT compliance training in relation to Child Protection. Essentially this equates to all 44 schools in the Diocese completing some form of child protection training annually. In addition orientation sessions held for all new school staff to the Diocese are held at the commencement of each school year in regards to an overview of child protection responsibilities in relation to managing risk of significant harm and allegations against employees. Child protection presentations are also provided to Catholic Schools Office "field" staff and leadership on an as needs basis. | Jan-15 | N/A | Rolling out of SALT online compliance training to all CSO staff deemed in child-related work. |
| **Increased Child Protection personnel** - Since the inception of the Broken Bay diocese, the number of personnel within the child protection team at the Catholic Schools has fluctuated up and down. In 2013 there was the equivalent of one full-time Child Protection Officer and one full-time Child Protection Administrative Assistant. By January 2014 there was recognition of the need for additional personnel to meet the growing demands of child protection work generated by the 44 systemic schools within the diocese. This resulted in an additional 0.4 Child Protection Officer position being created. By June 2014, this additional position was increased to 0.6 (temporary). In May 2016, another additional 0.6 Child Protection Officer (temporary) position was created to again meet the ongoing work load. Therefore the current configuration of the Catholic Schools Office Child Protection Team includes the following personnel: 1 x 1.0 Child Protection Team Leader, 2 x 0.6 Child Protection Officers and 1 x 1.0 Child Protection Administrative Assistant, to service the 44 catholic systemic schools in the Diocese of Broken Bay. | Jan-14 | N/A | N/A |
| **Cross analysis of data** - The child protection team produces annual compliance reports for the Directors Group which breaks down data in relation to reported allegation matters and consultations in regards to risk of significant harm concerns at each school. This data is then cross-analysed to identify any school who is not making contact with the CSO Child Protection Team in either area. Schools who have zero or low reporting rates are targeted for further professional development and communication from leadership to ensure they are meeting their child protection legislative obligations. | Jun-13 | N/A | N/A |
| **Review of all CSO Child Protection Cases** - At the commencement of the Royal Commission, the diocese engaged an independent reviewer to examine all child protection files to ensure that all matters within the scope of the Royal Commission had been handled appropriately and to identify any outstanding issues which needed to be addressed, for example flagging any cases where police reports should be made but hadn't. As a result of the review any outstanding action identified was since completed. | Jan-13 | N/A | N/A |
| **Document search** - At the commencement of the Royal Commission a memo was sent to all schools by the Director of Schools which required all schools to complete an extensive search of all files held at each school site. The purpose of the document search was to identify any possible past child protection matters which may have not been dealt with properly or dealt with without reference to the Catholic Schools Office. Any relevant documents were required to be sent to the Catholic Schools Office for review and action. | Jan-13 | N/A | N/A |

| | | | |
|---|---|---|---|
| **Post Investigation Governance** - The Child Protection Team in consultation with the Office of the Director created a new process to ensure that recommendations which are identified within reportable conduct matters are followed through with. The process involves a report being generated for the Director of Schools on a bi-annual basis. The report lists all recommendations made by the Child Protection Team/Investigator in any allegation matter (including not in jurisdiction, exemption and reportable allegations) and assigns responsibility for action to be undertaken. Evidence of the recommended actions having been implemented by the school or CSO must be added to the investigation file and signed off by the Director of Schools to ensure compliance. | Jun-14 | N/A | N/A |
| **CSO website** - In 2014 the new Diocese of Broken Bay Community of Catholic Schools Office website was launched. The new website publishes specific information in relation to child protection and also publishes all relevant child protection policies. | 2014 | N/A | N/A |
| **Screening of exempt people** - As part of the overall child safe approach for schools, all schools are required to ensure that exempt individuals and contractors defined under the Child Protection (Working with Children) Act 2012, complete a declaration confirming they are not a prohibited person. This is used as an extra measure to ensure suitable people volunteering or working in schools are safe. | 2013 | N/A | N/A |
| **Truth Justice and Healing Council** - Throughout the Royal Commission, the Catholic Schools Office has actively contributed (along with the rest of the Broken Bay diocese) to and responded to requests by the Truth Justice Healing Council following invitations by the Royal Commission for input and feedback in relation to practices in place or ideas for improved process in relation to being a child safe institution. | 2013 | N/A | N/A |
| **Activity in the Royal Commission** - Since the commencement of the Royal Commission, the Catholic Schools Office has consistently reviewed activity in the Royal Commission by having standing agenda items at relevant meetings including the NSW Catholic Systemic Schools Child Protection Practitioner Group and the Diocese of Broken Bay Child Protection Head of Agency meetings with a view to ensuring all structures, policies, procedures and practices in place to safeguard children against child sexual abuse in schools are up to date and relevant. | 2013 | N/A | N/A |
| **Professional Development** - Child Protection Practitioners participated in relevant professional development opportunities addressing the complex area of child sexual abuse and creating child safe organisations including attending conferences, symposiums and presentations by imminent international experts such as Monica Applewhite and Ian Elliott and prominent Australian presenters such as Gerard Webster. | 2013 | N/A | N/A |
| **NSW Catholic Systemic Schools Child Protection Practitioner Group** - As a member of this group, the Broken Bay CSO Child Protection Practitioners contr buted to special projects initiated by the Catholic Systemic Schools Child Protection Practitioner Group. The first project involved the development of an audit tool designed to allow the different Church agencies to audit their compliance with appropriate safeguarding and child protection approaches to safety. The second project involves the development of Safeguarding Leadership training designed to be delivered to the leadership of the Catholic Schools Office and Catholic Systemic Schools outlining detailed content relating to child protection. | 2015 | The roll out of both the Safeguarding Audit tool and Safeguarding Leadership training is due to occur later in 2016. | N/A |
| **Updated policies & procedures** - The existing child protection policy was replaced with three stand alone child protection policies including the Diocesan Systemic Schools Addressing Allegations of Inappropriate Behaviour by Staff, Managing Risk of Significant Harm & Wel being Concerns and Working with Children Check. Procedures relating to the Selection and Recruitment of Teachers was also reviewed and questions regarding child protection requirements were tightened including the need for applicants to disclose information relating to previous reportable conduct matters and referee questions requiring disclosure of information relating to any risk posed by applicants needing to be revealed. | Nov-14 | N/A | N/A |
| **What Safeguarding Measures have you introduced into your area of responsibility since the Royal Commission (January 2013) in relation to pastoral care of victims?** | | | |
| **Detail information about the measure introduced** | **Date measure introduced** | **Are there any measures currently being developed?** | **Are there any planned?** |
| Continued to initiate appropriate referrals to specialist counselling support services for sexual abuse victims or those engaging in problematic sexualised behaviour where the school counselling service was not appropriate or the victim involved was now an adult (usually at the expense of the Catholic Schools Office). | 2013 | N/A | N/A |

| | | | |
|---|---|---|---|
| Participation in "Royal Commission" information meetings which allowed employees and the wider Diocesan community to openly discuss their feelings in regards to child sexual abuse in the Catholic Church. | 2014 | N/A | N/A |
| Provided feedback regarding the pre-release of "A priest in the family" film (sponsored by the Diocese of Broken Bay) which deals with secondary victims of child sexual abuse so that the launch of the film was sensitive to the needs of all stakeholders. | 2014 | N/A | N/A |
| Expanded the celebration of National Child Protection Week in September each year by ensuring all schools acknowledge and participate in at least one small activity to raise awareness of child abuse. | 2013 | N/A | In 2015 schools were encouraged to participate in the national online survey for students between 10 – 18 years which aimed to better understand how children and young people conceptualise safety and how they think institutions should, and do, respond to their safety needs. The survey was conducted by the Institute of Child Protection Studies at the Australian Catholic University and commissioned by the Royal Commission into Institutional Responses to Child Sexual Abuse.<br><br>In 2016, there are plans to be involved in a joint project with the Department of Family & Community Services on the Central Coast who are organising an art exhibition for school children regarding "At the Heart of the Matter - Keeping Kids Safe" |

## GOVERNANCE AND PERSONNEL MANAGEMENT

| **Non-ordained lay people (especially women)** - *(Number 11 on TJHC Information Template)* | | | | |
|---|---|---|---|---|
| **To what extent are lay people, particularly women, actively involved in governance and management activities in the diocese?** | **What roles do these lay people have within the diocese?** | **When did the role/s commence?** | **Are there any developments in progress in this area?** | **Are there any planned?** |
| The CSO and schools have approximately 50:50 representation of men and women in leadership roles. | School Principals, Heads of Service (CSO), Assistant Director, School Board members. | This distribution has been consistent for at least the past 5 years. | N/A | N/A |
| | | | | |
| | | | | |
| | | | | |

| **Culture** |
|---|
| **Do you think that the culture of your area of responsibility has changed, since the start of the Royal Commission (January 2013) ?** |
| The Catholic Schools Office takes the responsibility to protect children seriously. All staff are expected to promote child safety by having a clear understanding of their legal child protection responsibilities and act in accordance with those responsibilities. Since the commencement of the Royal Commission there has been an increased awareness of the importance of these responsibilities and a genuine desire from leadership to learn from the failures in the past so that past mistakes are not repeated. |
| |
| **If so, why? And how has it changed?** |
| The commitment at both the Catholic Schools Office level and school level to this approach has been enhanced since the commencement of the Royal Commission and as a result the culture has moved to one where child protection is embedded in everything we do. This is evidenced by the new and updated structures, policies, procedures and practices which have been put in place since the Royal Commission was announced. |
| |

## LITIGATION, SETTLEMENTS AND POLICIES FOR DEALING WITH PERPETRATORS - CHILD SEXUAL ABUSE

| **Approach to claims for child sexual abuse** - *(Number 12 on TJHC Information Template)* | | |
|---|---|---|
| **Describe the approach you take to claims for child sexual abuse** | **Are there any developments in progress in this area?** | **Are there any planned?** |
| Matters are either reported directly to the Catholic Schools Office or via contact with a school. Matters are immediately referred to the CSO Child Protection Team and Office of the Director for review. Consultation occurs in the first instance with the Chancery (Office for Chancery Services, Safeguarding) for advice and direction. Depending on the circumstances, case management will either be immediately handed over to the Office for Chancery Services, Safeguarding unit or the CSO Child Protection Team will have initial case management with oversight from Safeguarding, Office for Chancery Services. In circumstances where the CSO Child Protection Team carry the case management role, initial contact is made with the victim and where possible a meeting arranged to see what supports can be extended to the victim in terms of; police reporting obligations (noting no blind reports can be made); explaining the Towards Healing process including options for financial claims; exploring what pastoral support is required such as a formal apology from the Church; advising of rights in relation to legal support available; offering specialist counselling referrals and contact numbers for victim support groups; informing the victim of any investigation which may be required; and hearing the details of the abuse if the victim feels ready to disclose the information. | N/A | N/A |
| | | |
| | | |
| | | |

# LITIGATION, SETTLEMENTS AND POLICIES FOR DEALING WITH PERPETRATORS - CHILD SEXUAL ABUSE

| **Child Sexual Abuse - Perpetrators - How does the Diocese of Broken Bay manage these persons** - *(Number 15 on TJHC Information Template)* | | | | |
|---|---|---|---|---|
| **Category of person** | **What is your policy?** | **How are these persons managed?** | **Are there any developments in progress in this area?** | **Are there any planned?** |
| **Convicted** | Persons convicted of a child sexual assault offence would be subject to the Diocesan Systemic Schools Addressing Allegations of Inappropriate Behaviour by Staff policy (November 2014) and the Working with Children Check policy (November 2014). | If a person becomes convicted of a child sexual assault offence, the person would be subject to a reportable conduct investigation as per the NSW Ombudsman scheme and would either have their WWCC cancelled as per the Child Protection (Working with Children) Act 2012 or be subject to a bar on their WWCC and as a result would be immediately removed from undertaking any child related work. | N/A | N/A |
| | | | | |
| **Substantiated Claim civil/internal** | Current employees are subject to the Diocesan Systemic Schools Addressing Allegations of Inappropriate Behaviour by Staff policy (November 2014). | Following the outcome of a reportable conduct investigation as per the NSW Ombudsman scheme, a person who has had a substantiated finding of Sexual Misconduct or Sexual Offence, has their name reported to the NSW Office of the Children's Guardian. If the result of the notification is that the person is given a bar or interim bar on their WWCC, the person is immediately removed from child related work. However other disciplinary action may have already occurred prior to the decision by the NSW Office of the Children's Guardian depending on the seriousness of the matter. For instance the person may have already had their employment terminated or have been moved out of child related work at the initiative of the Catholic Schools Office. | N/A | **N/A** |
| | | | | |
| **WWCC Bar** | Current employees are subject to the Diocesan Systemic Schools Addressing Allegations of Inappropriate Behaviour by Staff policy (November 2014) and the Working with Children Check policy (November 2014). | An employee subject to a bar on their WWCC is immediately removed from child related work. The options available to the Catholic Schools Office are: dismiss the worker, suspend the worker from child related work pending the outcome of an appeal, or transfer them to non-child related work at the Catholic Schools Office. | N/A | N/A |
| | | | | |
| **Assessed as high risk** | Current employees are subject to the Diocesan Systemic Schools Addressing Allegations of Inappropriate Behaviour By Staff policy (November 2014). | If an employee has been subject to a risk assessment at the end of a reportable conduct investigation and they have been assessed as being a high risk, a number of provisions can be put in place to manage the ongoing risk if the person remains in a child related role. These options can include increased supervision and monitoring, being placed on a performance management plan, restriction or removal of certain duties, training or specialist support. | N/A | N/A |
| | | | | |

| | | | | |
|---|---|---|---|---|
| **Substantiated perpetrator - Child on Child abuse** | Managing Risk of Significant Harm and Wellbeing Concerns policy (November 2014). | Students who exhibit sexually abusive behaviour are reported to the NSW Department of Family & Community Services (FACS) as a child or young person who is at risk of significant harm. The Catholic Schools Office Child Protection Team and Pastoral Care & Wellbeing Team case manage the matter in conjunction with the statutory authority (FACS or JIRT - Joint Investigation Response Team) and/or NSW Police Child Abuse Squad (CAS). Risk management strategies are put in place at the school level (for example, restrictions on the playground or moving students to different classes) and referrals to specialist counselling services are initiated where necessary to support both the victim and student engaging in the sexually abusive behaviour. On some occasions it is necessary to ask the student who has engaged in sexually abusive behaviour to leave the school. | N/A | N/A |
| | | | | |

# CECNSW Supplementary Questions: "Going Forward" (Attachment 2)

**1. Background**

In order to respond to questions posed during our hearing we would first like to remind the committee of the statistics reported in our CECNSW submission to the Inquiry. That is, attention is drawn to:

- Sections 1.2. 1.3 and 1.4. NSW Catholic Congregational and Special school enrolments.
- Section 1.8. Detailed figures and definitions for enrolment of students with Disability identified by the AGDET School's census (State criteria and NCCD criteria)
- Section 4.2 Data arising from State and Commonwealth collections

In response to the request; "as to what needs to be done to improve the disability area going forward?": CECNSW reminds the Inquiry of the following summary recommendations as noted in our submission….

**2. Understanding Data Collections**

a. The conduct and purpose of the separate State Students with Disability (SWD) and National Comparable Collection of Data (NCCD) collections needs to be clarified by the Australian Education Council, refer section 3.

As in 2010 the requirement of the Australian Government Department of Education and Training (AGDET) for the recognition of a NSW student as eligible for funding as a student with a disability continues to be based on the requirements of the New South Wales Department of Education (http://www.schools.nsw.edu.au/studentsupport/programs/disability.php

Since 2014 schools now also collect data to show how they meet adjustment requirements under the Disability Discrimination Act (DDA) and Disability Standards for Education (DSE) through the National Comparable Collection of Data ( NCCD)

b. NCCD implementation is an Australian Education Act (2013) and Regulation requirement. The NCCD applies the disability definition found in the Disability Discrimination Act 1992, which is a broad definition of disability, and can include students with mild medical impairments, as well as learning and impulsive behavioural difficulties. On average only one third of students identified by the application of NCCD criteria are also identified applying State SWD criteria.

The NCCD process requires both an understanding of DDA requirements and an analysis of each student's adjustments for learning.

The following NCCD student information is collected for each child/student:

i. The level of adjustment provided;

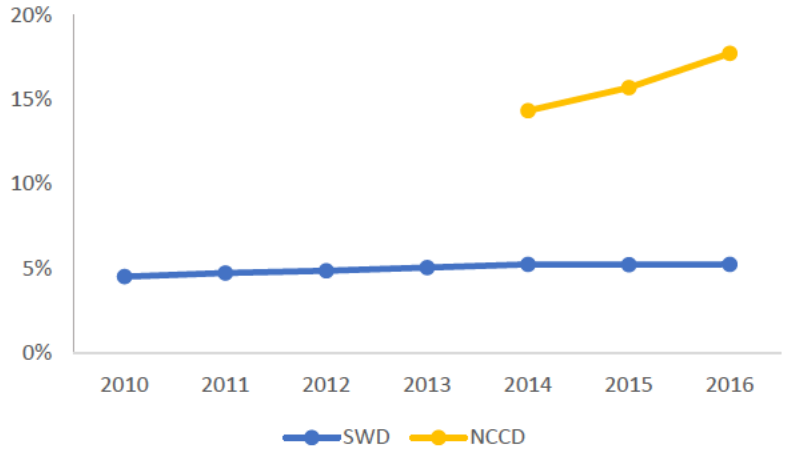
ii. Number of students receiving each level of adjustment to participate in education on the same basis as other students; and

iii. The student's type(s) of disability

Since 2010, the proportion of students identified as SWD has shown a small increase but remains relatively static. The change from 4.5% of total enrolments in 2010, to 5.2% in 2016 is a modest increase of less than 1% in six years.

In contrast, in the year from 2015 (the first year of full participation) to 2016, students identified under the NCCD definition have increased from 14.3% to 17.7%. This 3.4% increase is triple that of the SWD growth.

*Figure 1: Growth in NCCD students vs SWD students as a percentage of total enrolments*




c. The above-cited data suggests that the minimum proportion of the school age population requiring disability support is 5% of all students.

CECNSW also further notes that the National NCCD collection is not yet reliable or robust enough to be used as a funding tool at any level of decision making.

**3. Promoting Further Reform**

When considering how the enquiry might progress its objectives, CECNSW highlights the following advice:

i. The non-government schools sectors are not currently included in any NSW planning committees in regard to NDIS implementation and depend on briefings from the NSW Department of Education for updates and briefings. The non- government sector appreciates the support of the NSW Department of Education but believes that non-government schools need to be included in the membership of all relevant NSW NDIS planning committees.

ii. CECNSW is of the view that additional and particular Guidelines for all NSW schools are required that will enable both schools and parents to understand the processes that must be followed and the factors to be taken into account when determining what reasonable adjustments are required to manage behaviour and whether these adjustments are reasonable or unreasonable. These proposed NSW Guidelines should assist all parties to appreciate that a school is not required to implement unreasonable adjustments, see "Disability Standards for Education 2004, Guidance Notes" at 4.2 – "making reasonable adjustments" and "no requirement to make unreasonable adjustments", refer section 6 of the CECNSW submission.

iii. CECNSW restates that the existing parent complaint processes established and oversighted by NSW Education Standards Authority (NESA) should be better advised to parents by NESA.

iv. Existing NSW Supervisor subsidy grant eligibility should be reformed so that it is based on assessed student need only, rather than on both student need and the type of school a student attends. The existing NSW Supervisor Subsidy Grant is still only available to students with Autism or a moderate to profound intellectual disability who are enrolled in a recognised special school. This Grant should be applied to all students, regardless of their school of attendance, who require substantial and extensive adjustments as identified through the NCCD process.

v. CECNSW would support the formation of a cross sector special education group to advise school communities with respect to the use of evidence-based research in both initial teacher education disability support courses and related professional learning for teachers and Principals, including both elearning training and face-to-face professional development.

# CECNSW Supplementary Questions: Arrowsmith Program (Attachment 3)

**PORTFOLIO COMMITTEE NO 3 - EDUCATION**

**INQUIRY INTO EDUCATION FOR STUDENTS WITH DISABILITY OR SPECIAL NEEDS IN NEW SOUTH WALES SCHOOLS**

**HEARING: MONDAY 27 MARCH 2017 Answers to be returned to the Committee secretariat by Friday 21 April 2017**

**Supplementary questions for the Catholic Education Commission NSW**

*1. How many Catholic Education Commission NSW schools run, participate in or support the provision of the Arrowsmith Program?*

Sydney Catholic Schools operates as a systemic school authority within the Archdiocese of Sydney. Sydney is the only NSW diocese currently offering the Arrowsmith Program for students with complex learning disorders.

Arrowsmith classes operate on the following Sydney Catholic Schools sites:

- One Secondary Arrowsmith class at Casimir Catholic College, Marrickville
- Two Primary classes - one class at Holy Innocents' Catholic Primary School Croydon, the other class at Sacred Heart Catholic Primary School, Mosman
- A part-time, after school hours Arrowsmith Program is offered at the Eileen O'Connor Catholic College, Lewisham. Students attend this program for four hours a week. These students are all full-time students enrolled at various Sydney Catholic Schools, Independent Schools or NSW Department of Education Schools.

Unlike the Canadian implementation model, students in the classes at Marrickville, Croydon and Mosman are enrolled in the mainstream school on the respective school site. They participate in Arrowsmith classes for part of the day and then join their age cohort to engage in curriculum that complies with New South Wales Educational and Standards Authority (NESA) requirements.

No State Literacy and Numeracy Strategy funds are used to implement the Arrowsmith program within Sydney Catholic Schools. Program costs are met by families paying a course fee to Arrowsmith in Canada, in addition to the payment of Sydney Catholic Schools' school tuition fees.

*2. How many dioceses run, participate in or support the provision of the Arrowsmith Program?*

Sydney is currently the only NSW diocese to offer the Arrowsmith Program.

*3. How many students in Catholic Education Commission NSW schools participate in the Arrowsmith Program?*

A total of 58 students participate in the Arrowsmith Program during school hours on mainstream school sites, and a further 17 students participate in a part-time after hours program at Lewisham.

Recognising that acceptance of enrolment into Arrowsmith classes within Sydney Catholic Schools, can occur at any time throughout the school year, as at 13/4/2017 the number of students participating in the Arrowsmith Program at each school site is as follows:

- Casimir Catholic College, Marrickville – 17 students
- Holy Innocents' Catholic Primary School, Croydon – 18 students
- Sacred Heart Catholic Primary School, Mosman – 23 students
- Eileen O'Connor Catholic College, Lewisham (part-time after hours program) - 17 students

*4. What steps have been taken by the Catholic Education Commission or any dioceses to assess the effectiveness of the Arrowsmith Program?*

At the conclusion of the first (pilot) year of offering the Arrowsmith Program in Sydney Catholic Schools, an external evaluation was conducted. This was facilitated by an Australian Catholic University academic. The results of the evaluation indicated that the program was having a beneficial effect on students: educationally, personally and socially.

Specifically, the evaluator noted that the Arrowsmith Program, had made a positive educational and social difference to the lives of students who had previously not experienced success in academic tasks and had difficulties in other aspects of their functioning. The evaluator also commented that parents of the students enrolled in this inaugural Sydney Catholic Schools Arrowsmith class, had investigated and trialled many intervention programs for their children. These interventions in many cases, represented significant costs to families and were ultimately unsuccessful.

In the pilot program evaluation, feedback from parents was positive, with reports of clear improvements in their children's basic day to day life skills and personal development. Growth in self confidence and sense of independence was also experienced by the students and observed by teachers, school counsellors and parents.

On the basis of these findings, Sydney Catholic Schools has continued to provide the Arrowsmith Program for students who meet the program's selection criteria. It has expanded the program to include two Primary classes and a part-time after hours class. Over the period 2013-2017, operational adjustments to the implementation model, have been made in light of feedback from key stakeholders.

Sydney Catholic Schools has a commitment to continuous improvement of programs offered to support students with diverse learning needs. Therefore regular monitoring of students' growth is an important part of students' engagement with the Arrowsmith Program.

Student performance data related to mastery of the Arrowsmith cognitive exercises is collected and regularly remitted to Arrowsmith Canada for analysis and feedback. Additionally, achievement and growth in students' mainstream classes is assessed and tracked by way of students participation in the National Assessment Program and other assessments.

From 2017, a variety of diagnostic instruments will be integrated into the assessment of Arrowsmith students in order to gauge the impact the program is having on students' academic attainment, perseverance capacity and overall wellbeing.

Other aspects of the program will be formally reviewed as required.

*5. What advice has been provided by the Catholic Education Commission to dioceses or schools regarding the Arrowsmith Program?*

CECNSW has not been involved with Arrowsmith Program participation or advice.

## Annexure D

# ARROWSMITH PROGRAM RESEARCH UPDATE (August 2017)

Peer-review Publication Focus (Learning Disabilities and Brain Injury Research)

## Learning Disabilities (LD) and Arrowsmith Program Research - (Six Research Studies Underway)

**University of British Columbia**: Faculty of Medicine

1) Dr. Lara Boyd et al (commenced 2014)

- Year 1 data – positive for Arrowsmith Program, but needed to increase control group sample sizes, so had to wait another year to find subjects for control groups.
- Currently collecting 2-year data point. This will be completed by the end of June 2017.
- Control groups – close to 20 in each group – a) Children without LD b) public school students with LD in traditional special education programs
- 55 Arrowsmith students in experimental group (from Vancouver and Redmond)
- One paper has been sent in for peer review – but just on base-line data – not outcome data
- Submission to journals in late fall 2017 or early winter 2018

2) Dr. Kimberly Schonert-Reichl and Dr. Lara Boyd (Data collection starts September 2017)

- Title of research: Neuroplasticity and Social Emotional Learning – Early Human Learning Partnership – UBC
- To study social emotional outcomes of those doing the Arrowsmith Program in comparison to control groups – children in public school programs.
- Analyze Social Emotional Learning in different educational contexts for children with LD – so Eaton Arrowsmith School, Public School, etc.

3) Dr. Rachel Weber and Dr. Lara Boyd – From Faculty of Education and Faculty of Medicine, UBC (commenced October 2016)

- Arrowsmith Program and impact on working memory
- Have recruited students from Eaton Arrowsmith School
- Working Memory is highly correlated to academic success – one of the key cognitive capacities that have this correlation

4) Dr. Lara Boyd and Dr. Rachel Weber (commenced July 2017)

- Researching LD students participating in Eaton Arrowsmith School's summer Cognitive Intensive Program
- Studying the 6-week summer intensive Symbol Relations Program
- fMRI, Executive Functioning, behavioural measures (achievement and cognitive)
- Looking at both children and young adults in this research
- Vancouver, Eaton Arrowsmith School participating

**Southern Illinois University**

5) Dr. Greg Rose - Director, Center for Integrated Research in Cognitive & Neural Sciences (commenced 2014)

- Researching 40 LD students at Brehm School, those in Arrowsmith Program and those receiving Brehm standard special education program.
- First year data showing that Arrowsmith Program students are showing improvements in various aspects of brain functioning including a network called – Executive Control Network.
- Increasing sample size this year to help with N and publications of data.
- Seeing similar changes in the Executive Control Network and other brain regions as Dr. Boyd.

6) Dr. Greg Rose - (commenced July 2017)

- Researching LD students participating in Arrowsmith School Toronto's and Peterborough's summer Cognitive Intensive Program
- Studying the 6-week summer intensive Symbol Relations Program
- fMRI resting state measures
- 22 participants
- Looking at both children and young adults in this research

## Brain Injury and Arrowsmith Program Research

University of British Columbia, Faculty of Medicine

*Changes in brain-behavior relationships following a 3-month pilot cognitive intervention program for adults with traumatic brain injury* (S. Porter, I.J. Torres, W. Panenka, Z. Rajwani, D. Fawcett, A. Hyder, N. Virji-Babul)

- Published August 4, 2017 http://www.heliyon.com/article/e00373?via=sd&cc=y%3D&
- Research on Traumatic Brain Injury and the Arrowsmith Program – 3 month pilot
- 10 adult participants with mild to severe brain injury and 11 age and gender matched healthy controls.

The 3 month pilot study found a statistically significant increase in the composite cognitive score in the TBI participants and a statistically significant decrease in functional connectivity in the right inferior frontal gyrus. In addition, there was evidence of changes in the brain-behavior relationships following the Arrowsmith intervention. The results from this pilot study provide preliminary evidence for functional network reorganization that parallels cognitive improvements after cognitive rehabilitation in individuals with chronic TBI.

## Annexure E

**Independent Scientific Evidence: Arrowsmith Program**

The Arrowsmith Program has implemented independent evidence-based research over three decades. This research has included various levels of research design including both independent research and peer-reviewed research in progress and recently completed. Three types of educational research have been applied to analyzing the Arrowsmith Program including descriptive research (e.g., case study and survey), associational (e.g., correlational and causal-comparative), and intervention research (which investigates the impact of an intervention on individuals or groups). This evidence-based research has used a variety of research designs and data collection methods including case study, intervention group/control group studies, interview, neuroimaging and behavioural assessment and self-reporting.

The use of various research designs and methods has support from one of Australia's and New Zealand's most prominent academics, Dr. John Hattie, Professor of Education and Director of the Melbourne Education Research Institute at the University of Melbourne. In a paper published in 2005, Dr. Hattie stated, "A current fad radiating out from the United States is the notion of evidence-based decision-making – and this term has been hijacked to mean a very narrow form of evidence. Liberty and Miller (2003), for example, consider 'evidence-based' relates to meeting peer-review standards, and including evidence directly impacting on children's learning…This cuts out so much of today's literature…But an extra condition has been added, that of the type of research design to collect data: preferably random assignment to various groups…While this may be exemplary, it is not the only design of merit. Moreover, in classrooms, teachers still need to base their evidence on data from their students and from their teaching, and rarely does random assignment occur. It is this form of teacher-available data that is of interest to my forms of accountability." (*What is the nature of evidence that makes a difference to learning?* conference paper for 2005 Australian Council for Educational Research conference: Using Data To Support Learning, p. 12, ACEReSearch). http://research.acer.edu.au/cgi/viewcontent.cgi?article=1008&context=research_conference_2005

Random assignment of subjects (RCT or Randomized Control Trials or "gold standard") for an experimental design are very problematic to implement in education (as noted by Dr. John Hattie). For example, it is problematic for researchers to implement as some parents, teachers or administrators do not want to exclude students from receiving treatment. Because of these issues, in 2010, the U.S. Department of Education broadened its definition of "gold standard" research to include nonexperimental designs. As well, many academics and researchers are realizing the problems associated with trying to conduct "gold standard" research and have published on this issue.

Professor Dylan Williams wrote an article titled, *Randomized Control Trials in Education Research*, (June 2014, R.Ed Research in Education, p.3, University of Brighton). https://www.brighton.ac.uk/_pdf/research/education/red%20publications/red_vol6_no_1-5.pdf)

In the article, Dr. Williams points out the problems in implementing and interpreting randomized control trials. After outlining the various problems with randomized control trials, he states, "None of the foregoing is intended to suggest that randomized control trials are a bad idea. Rather the discussion highlights that if we rely only on such experimental designs, we end up not being able to say very much, and even when we do conduct such experiments, they benefit from research designs that include complementary approaches to inquiry."

The Arrowsmith Program has utilized and continues to utilize multiple research designs and methods to achieve understanding of cognitive intervention on children.

Research has been conducted by independent researchers not involved in the authorship or ownership of the Arrowsmith Program. These are highly respected academic independent researchers from Faculties of Medicine, Psychology, Psychiatry, Education, Neuroscience, Physics. Some of those involved are:

Dr. Lara Boyd, University of British Columbia, Professor, Faculty of Medicine, Canadian Research Chair (Tier II)

Dr. Greg Rose, Southern Illinois University, Professor, Faculty of Medicine, Director of Centre for Integrative Research in Cognitive and Neural Sciences

Dr. Kimberly Schonert-Reichl, University of British Columbia, Professor and Applied Developmental Psychologist, Department of Educational and Counselling Psychology and Special Education. Director of the Human Early Learning Partnership in the School of Population and Public Health, Faculty of Medicine.

Dr. Rachel Weber, University of British Columbia, Assistant Professor, Faculty of Education, Educational and Counselling Psychology and Special Education

Dr. William Lancee, University of Toronto, Head of Research, Mount Sinai, Department of Psychiatry

Dr. Naznin Virji-Babul, University of British Columbia, Director, Faculty of Medicine, Perception-Action Laboratory

Dr. William Panenka, University of British Columbia, Assistant Professor, Faculty of Medicine, Department of Psychiatry, Neuropsychiatry.

Dr. Ivan Torres, University of British Columbia, Clinical Professor, Faculty of Medicine, Department of Psychiatry.

Dr. James B. Hale, Nanyang Technological University, Singapore, Professor of Educational Neuroscience, Centre for Research and Development in Learning (CRADLE)

Dr. Alex MacKay, University of British Columbia, Professor, Physics, Departments of Radiology, Physics and Astronomy – Research area: Nuclear Magnetic Resonance/Magnetic Resonance Imaging

Dr. Todd C. Handy, University of British Columbia, Professor, Department of Psychology, Cognitive Neuroscience, neuroimaging

The Arrowsmith Program continues to be committed to conducing independent evidence-based research that can be peer-reviewed and published in academic journals. Research is often incremental, with increasing levels of rigour, culminating in peer-reviewed, replicated, published research.

Recent data analysis outlined the cognitive and behavioural impact of the Arrowsmith Program for individuals with traumatic brain injury: (S. Porter, I.J. Torres, W. Panenka, Z. Rajwani, D. Fawcett, A. Hyder, N. Virji-Babul, *Changes in brain-behavior relationships following a 3-month pilot cognitive intervention program for adults with traumatic brain injury*, August 4 2017, Heliyon, http://www.heliyon.com/article/e00373?via=sd&cc=y%3D& ). This peer-reviewed published research provided insight into increasing the exposure of complexity to further advance cognitive and behavioural gains for those with traumatic brain injuries.

The research currently being conducted by Dr. Lara Boyd and colleagues is targeted for 4 to 5 peer-reviewed publications during 2018 using data collected from the Arrowsmith Program study. This study includes control groups in the analysis. Dr. Boyd has recently completed year 2 data collection, and will be publishing peer-reviewed journal articles from this data set. According to the preliminary findings on year one data presented by Dr. Lara Boyd at Columbia University in June 2016 (Cognitive Remediation in Psychiatry Conference, New York) results have been positive and benefits shown for students in the Arrowsmith Program on academic, cognitive and imaging measures, including changes in the executive control network involved in high-level cognitive processing required to pay attention, exert executive control, plan, problem-solve, make decisions and for working memory. It should be noted that the Arrowsmith Program has given Dr. Boyd complete autonomy to analyze and publish the data. Dr. Greg Rose and his team have also been given complete autonomy and are also heading towards peer-

reviewed publications on his data from the Brehm School's implementation of the Arrowsmith Program compared to control groups.

It is interesting to note that Dr. John Hattie's research considered the major source of variance in student's achievement. In his 2003 paper titled, *Teachers Make a Difference, What is the research evidence?* (conference paper for 2003 Australian Council for Educational Research conference: Building Teacher Quality: What does the research tell us?, ACEReSearch) he noted that the second most important source of variance after *feedback* was a *student's prior cognitive ability*.
http://research.acer.edu.au/cgi/viewcontent.cgi?article=1003&context=research_conference_2003

It is promising that the evidence-based research into the Arrowsmith Program is highlighting that it is possible to improve the cognitive capacities in children with learning difficulties.

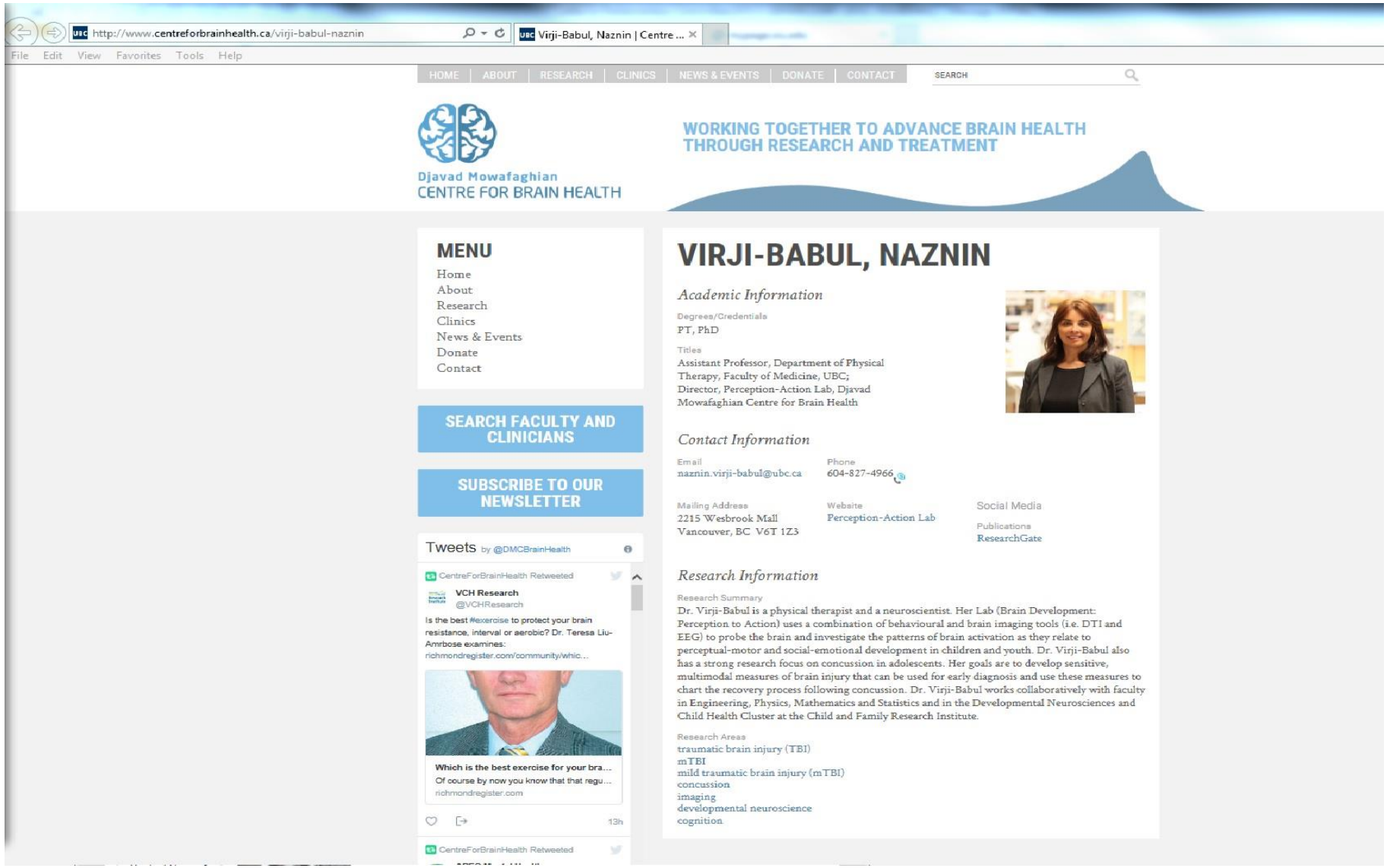
Sincerely,

Howard Eaton, Ed.M.

Director of Research, Arrowsmith Program

August 2017

## Annexure F


http://www.centreforbrainhealth.ca/virji-babul-naznin
Virji-Babul, Naznin | Centre ...
File Edit View Favorites Tools Help
HOME | ABOUT | RESEARCH | CLINICS | NEWS & EVENTS | DONATE | CONTACT
SEARCH
Djavad Mowafaghian
CENTRE FOR BRAIN HEALTH
WORKING TOGETHER TO ADVANCE BRAIN HEALTH THROUGH RESEARCH AND TREATMENT
MENU
Home
About
Research
Clinics
News & Events
Donate
Contact
SEARCH FACULTY AND CLINICIANS
SUBSCRIBE TO OUR NEWSLETTER
Tweets by @DMCBrainHealth
CentreForBrainHealth Retweeted
VCH Research
@VCHResearch
Is the best #exercise to protect your brain resistance, interval or aerobic? Dr. Teresa Liu-Ambrose examines:
richmondregister.com/community/whic...
Which is the best exercise for your bra...
Of course by now you know that that regu...
richmondregister.com
13h
CentreForBrainHealth Retweeted
VIRJI-BABUL, NAZNIN
Academic Information
Degrees/Credentials
PT, PhD
Titles
Assistant Professor, Department of Physical Therapy, Faculty of Medicine, UBC;
Director, Perception-Action Lab, Djavad Mowafaghian Centre for Brain Health
Contact Information
Email
naznin.virji-babul@ubc.ca
Phone
604-827-4966
Mailing Address
2215 Wesbrook Mall
Vancouver, BC V6T 1Z3
Website
Perception-Action Lab
Social Media
Publications
ResearchGate
Research Information
Research Summary
Dr. Virji-Babul is a physical therapist and a neuroscientist. Her Lab (Brain Development: Perception to Action) uses a combination of behavioural and brain imaging tools (i.e. DTI and EEG) to probe the brain and investigate the patterns of brain activation as they relate to perceptual-motor and social-emotional development in children and youth. Dr. Virji-Babul also has a strong research focus on concussion in adolescents. Her goals are to develop sensitive, multimodal measures of brain injury that can be used for early diagnosis and use these measures to chart the recovery process following concussion. Dr. Virji-Babul works collaboratively with faculty in Engineering, Physics, Mathematics and Statistics and in the Developmental Neurosciences and Child Health Cluster at the Child and Family Research Institute.
Research Areas
traumatic brain injury (TBI)
mTBI
mild traumatic brain injury (mTBI)
concussion
imaging
developmental neuroscience
cognition

## Annexure G

Heliyon

*Received:*
17 June 2017
*Revised:*
22 June 2017
*Accepted:*
27 July 2017

Cite as: S. Porter, I.J. Torres, W. Panenka, Z. Rajwani, D. Fawcett, A. Hyder, N. Virji-Babul. Changes in brain-behavior relationships following a 3-month pilot cognitive intervention program for adults with traumatic brain injury.
Heliyon 3 (2017) e00373.
doi: 10.1016/j.heliyon.2017.e00373

# Changes in brain-behavior relationships following a 3-month pilot cognitive intervention program for adults with traumatic brain injury

**S. Porter [a], I.J. Torres [b], W. Panenka [b,c], Z. Rajwani [d], D. Fawcett [b], A. Hyder [f], N. Virji-Babul [d,e,*]**

[a] *Graduate Program in Rehabilitation Sciences, University of British Columbia, Canada*

[b] *Department of Psychiatry, University of British Columbia*

[c] *British Columbia Provincial Neuropsychiatry Program*

[d] *Djavad Mowafaghian Centre for Brain Health, University of British Columbia*

[e] *Department of Physical Therapy, University of British Columbia, Canada*

[f] *Graduate Program in Neuroscience, University of British Columbia*

* Corresponding author at: Dept. of Physical Therapy, University of British Columbia, 212–2177 Westbrook Mall, Vancouver, British Columbia, V6T 1Z3, Canada.

E-mail address: Naznin.virji-babul@ubc.ca (N. Virji-Babul).

## Abstract

Facilitating functional recovery following brain injury is a key goal of neurorehabilitation. Direct, objective measures of changes in the brain are critical to understanding how and when meaningful changes occur, however, assessing neuroplasticity using brain based results remains a significant challenge. Little is known about the underlying changes in functional brain networks that correlate with cognitive outcomes in traumatic brain injury (TBI). The purpose of this pilot study was to assess the feasibility of an intensive three month cognitive intervention program in individuals with chronic TBI and to evaluate the effects of this intervention on brain-behavioral relationships. We used tools from graph

http://dx.doi.org/10.1016/j.heliyon.2017.e00373

theory to evaluate changes in global and local brain network features prior to and following cognitive intervention. Network metrics were calculated from resting state electroencephalographic (EEG) recordings from 10 adult participants with mild to severe brain injury and 11 age and gender matched healthy controls. Local graph metrics showed hyper-connectivity in the right inferior frontal gyrus and hypo-connectivity in the left inferior frontal gyrus in the TBI group at baseline in comparison with the control group. Following the intervention, there was a statistically significant increase in the composite cognitive score in the TBI participants and a statistically significant decrease in functional connectivity in the right inferior frontal gyrus. In addition, there was evidence of changes in the brain-behavior relationships following intervention. The results from this pilot study provide preliminary evidence for functional network reorganization that parallels cognitive improvements after cognitive rehabilitation in individuals with chronic TBI.

Keywords: Neurology, Evidence-based medicine, Rehabilitation

## 1. Introduction

Traumatic brain injury (TBI) is a serious and complex public health issue. In the USA alone, there are over 1.6 million TBI's reported every year (Langlois et al., 2006) with many more unreported. A complex and debilitating neurological disorder, TBI is defined as an alteration in brain function, or other evidence of brain pathology, caused by an external force (Menon et al., 2010). Classified by severity, TBI ranges from mild to severe, based on the length of time of loss of consciousness, post-injury amnesia, Glasgow Coma Score (Teasdale and Jennett, 1974) and trauma related findings on neuroimaging (Malec et al., 2007). The hallmark of TBI is diffuse axonal changes combined with continually evolving secondary changes. This feature combined with the heterogeneity of external factors such as location of injury, severity of injury, previous history and individual response to brain injury, can result in unique and dramatic changes in brain structure and brain function at many levels ranging from microscopic tears in white matter to global changes in functional brain networks. Headaches, dizziness, sleep disturbances, and fatigue often occurs immediately post injury, and may be severe. Attention, memory, processing speed and executive functions (i.e. working memory, cognitive flexibility, etc.) are also compromised early (Rabinowitz and Levin, 2014) and recover inconsistently (Broglio and Puetz, 2008; McAllister et al., 2006). A significant proportion of individuals with TBI continue to suffer from persistent cognitive and behavioural complaints and disability (Kraus et al., 2005; McAllister et al., 2006), resulting in prolonged or lifelong disability and dependence on the health care system. It is estimated that 2% of the population live with permanent disability related to TBI, with an estimated economic impact of


http://dx.doi.org/10.1016/j.heliyon.2017.e00373

$60 Billion/year in the USA(Finkelstein et al., 2006). Developing effective rehabilitation strategies for this population is therefore of great importance.

At a behavioural level there is considerable evidence that targeted cognitive interventions can improve attention, executive function, and memory (Caeyenberghs et al., 2016; Jolles et al., 2013; Kundu et al., 2013). In addition, cognitive rehabilitation is reportedly effective in helping patients learn and apply compensatory mechanisms for remaining deficits (Cicerone et al., 2011). Improvements in specific tasks related to executive function and memory have also been shown to generalize to functional improvement in everyday activities (Kennedy et al., 2008). Given that cognition is dependent on the activity of widely distributed functional brain networks (Bressler and Menon, 2010), evaluating the characteristics of the underlying networks in response to behavioral change stimulated by cognitive intervention can provide important insights into the underlying neuroplastic process.

A powerful and elegant method of evaluating the characteristics of brain networks is through the use of graph theory. In this framework, the brain is viewed as a network with interactions and communication occurring over multiple levels between local and distant areas (Rubinov and Sporns, 2010). One approach to understanding the nature of these interactions is via functional connectivity. Functional connectivity refers to the statistical interdependencies between physiological time series recorded from the brain (Friston, 2011). Graph theory has emerged as a promising tool in recent years for characterizing brain connectivity at both global and regional levels (Bullmore and Sporns, 2009; Rubinov and Sporns, 2010). Graph theory allows for the quantitative analysis of network organization, characterizing the brain as a set of networks, with each network being composed of distinct brain regions or "nodes". The various nodes are functionally connected via edges. The relationship between nodes and edges provides information about the organization and efficiency of the network. Networks with an ordered structure have a high clustering coefficient (a measure that depicts the connectedness of immediate neighbors around individual vertices), long characteristic path length (an index reflecting the overall integration of the network), low global efficiency (defined as the average inverse shortest path) and low density or cost (the fraction of present connections to possible connections) (Wu et al., 2012). In contrast, randomly organized networks are characterized by a low clustering coefficient, short characteristic path lengths, and a high global efficiency and density.

The healthy brain has been shown to be a combination of ordered networks with a certain fraction of randomly rewired links which results in "small-world" networks, with tightly connected neighborhoods, short characteristic path lengths and a high local efficiency (Wu et al., 2012). These small-world networks balance


http://dx.doi.org/10.1016/j.heliyon.2017.e00373

between local specialization and global integration, which are optimal for information processing (Watts and Strogatz, 1998).

Graph theoretical approaches have recently been applied to understand the effect of brain injury from a network perspective. In adults with TBI there appears to be a shift towards sub-optimal network organization (Caeyenberghs et al., 2012; Nakamura et al., 2009; Pandit et al., 2013). We have previously shown that mild TBI in adolescents does not alter resting state global network efficiency but does cause change in the local networks within the prefrontal cortex (Virji-Babul et al., 2014). This provides evidence for local changes in the frontal regions of the brain that are likely to affect the efficient processing of cognitive functions following mTBI. Arnemann et al. (2014) applied graph theory to predict individual responses to cognitive training in individuals with brain injury (Arnemann et al., 2015). They found that modularity (a measure of the density of links within a community relative to ones between communities) was able to predict improvements in attention and executive function following cognitive training. This suggests that functional brain imaging and graph theory has the potential to provide valuable information for understanding the mechanisms that influence recovery from TBI.

The purpose of this study was to pilot measures using graph theory analysis to evaluate the feasibility of these measures in capturing change following intervention. The specific objectives of this study were: (1) To establish the baseline global and local functional connectivity in individuals with TBI and evaluate the association with neuropsychological functioning, (2) To evaluate the changes in global and local functional connectivity in individuals with TBI following a 3-month cognitive intervention program and (3) To determine if network reorganization as measured by functional connectivity is associated with changes in cognitive recovery following the 3-month cognitive intervention program.

## 2. Methods

### 2.1. Participants

Participants with TBI were recruited from brain injury associations across the Greater Vancouver (British Columbia, Canada) area. All TBI participants were chronic with injuries occurring a minimum of 1 year prior to the start of the intervention. All healthy controls were recruited from the lower mainland of Vancouver in close proximity to the university. All controls were screened to ensure that they had no history of head trauma, neuropsychiatric disorders, substance abuse or any other neurological conditions. The participants with TBI were first interviewed to determine eligibility and to evaluate the severity of TBI. Participants were excluded if they were involved in litigation, had a history of current or recent substance abuse or if they had other severe medical conditions


http://dx.doi.org/10.1016/j.heliyon.2017.e00373

affecting brain function. They were also excluded if they had a diagnosis of psychiatric illness based on self report or when assessed with the Mini-international neuropsychiatric interview (MINI) (Sheehan et al., 1998). TBI severity was classified mild or moderate/severe, as per ACRM criteria (Mild Traumatic Brain Injury Committee, Head Injury Interdisciplinary Special Interest Group, American Congress of Rehabilitation Medicine. 1993). using retrospective self report of the duration of loss of consciousness and length of post-traumatic amnesia.

The experiments described in this study were approved by the Human Ethics Review Board at the University of British Columbia. All participants provided written consent according to the guidelines set forth by the Clinical Research Ethics Board at the University of British Columbia and this study complies with all regulations.

**Table 1.** Demographic and clinical profile of participants with TBI and Controls. M = male; F = female; TBI = Traumatic Brain Injury; MVA = Motor Vehicle Accident.

| Participant ID | Sex | Age | Years of education | Time since injury (years) | Etiology | Mechanism of injury | Severity |
|---|---|---|---|---|---|---|---|
| S001 | M | 51 | 14 | 22 | TBI | MVA | Severe |
| S002 | F | 50 | 18 | 29 | TBI | MVA | Severe |
| S003 | M | 36 | 16 | 24 | TBI | Multiple Concussion | Mild |
| S004 | M | 41 | 12 | 8 | TBI | MVA | Severe |
| S005 | M | 18 | 13 | 2 | TBI | Multiple Concussion | Mild |
| S007 | F | 48 | 20 | 4 | TBI | MVA | Mild |
| S008 | M | 26 | 13 | 6 | TBI | Fall | Severe |
| S009 | F | 35 | 13 | 8 | TBI | MVA | Severe |
| S011 | F | 38 | 18 | 7 | TBI | MVA | Severe |
| S012 | F | 50 | 17 | 7 | TBI | MVA | Mild |
| C001 | F | 53 | 20 | NA | Healthy | NA | NA |
| C002 | M | 36 | 20 | NA | Healthy | NA | NA |
| C003 | M | 30 | 18 | NA | Healthy | NA | NA |
| C004 | M | 52 | 20 | NA | Healthy | NA | NA |
| C005 | M | 21 | 16 | NA | Healthy | NA | NA |
| C006 | M | 38 | 14 | NA | Healthy | NA | NA |
| C008 | F | 50 | 14 | NA | Healthy | NA | NA |
| C009 | M | 24 | 16 | NA | Healthy | NA | NA |
| C010 | F | 27 | 12 | NA | Healthy | NA | NA |
| C011 | M | 45 | 14 | NA | Healthy | NA | NA |
| C012 | M | 21 | 16 | NA | Healthy | NA | NA |


http://dx.doi.org/10.1016/j.heliyon.2017.e00373

## 2.2. Demographic characteristics

Table 1 shows the demographic features and clinical features for all the participants in this study. The mean age of the TBI participants was 39.6 years (18, 54), with a mean of 15.3 years of formal education. The gender distribution was 50% male. Time since initial injury for the TBI group ranged from 2 to 29 years with an average of 11.7 years. The healthy controls had a mean age of 37.4 years (21, 53) with a mean of 16.6 years of formal education and a gender distribution of 73% male. There were no significant differences in age [$t(19) = 0.429$, $p = 0.673$, $d = 0.19$] or education [$t(19) = -1.055$, $p = 0.305$, $d = -0.46$] between groups. All participants were right handed. All participants with TBI were able to complete the 3-month program.

## 2.3. Neuropsychological testing

All subjects were administered several neuropsychological tests assessing processing speed, memory, and executive function, as deficits in these cognitive skills have been documented across a range of TBI (Podell et al., 2010). The specific tests used were as follows:

1. Rey Auditory Verbal Learning Test (RAVLT)(Lezak and Lezak, 2004): On this verbal learning and memory test, the primary variables employed were total recall during learning trials and delayed free recall.

2. Trailmaking Test Parts A and B (Reitan and Wolfson, 1993): The Trails A test measures visual attention and processing speed, and Trails B assesses more complex attentional shifting. The time to completion for each task was employed.

3. Verbal Fluency (Lezak and Lezak, 2004): On the phonemic verbal fluency task, subjects were asked to generate as many words beginning with the letters F, A, S. The main outcome was number of correct words produced.

For all tasks, demographics corrected z-scores were used as the primary measures. For the RAVLT and Verbal fluency tasks, alternate versions of the tests were used during follow-up testing (see below) to minimize practice effects. A global composite score was derived by averaging z-scores for the 5 primary measures described above.

## 2.4. EEG recording and analysis

EEG was recorded using a 64-channel HydroCel Geodesic SensorNet (EGI, Eugene, OR). The EEG cap was placed on each participant's head and 5 minutes of resting state data was recorded with their eyes closed. EEG was recorded and amplified using Net Amps 300 amplifier, at a sampling rate of 250 Hz. Scalp electrode impedances were generally under 50 kΩ. The signal was referenced to


http://dx.doi.org/10.1016/j.heliyon.2017.e00373

the vertex (Cz) and filtered from 4 to 40 Hz. A notch filtered at 60 Hz was applied. The EEG signals were analyzed offline using Brain Electrical Source Analysis (BESA) (MEGIS Software GmbH). An automated artifact scan was performed for extracting motion and excessive eye movement artifacts. BESA brain source montage was used to convert the EEG activity obtained from all the 64 scalp channels into predicted contributions of a set of 15 different brain source activity. The advantage of using a brain source montage is that the volume conduction effects are reduced in comparison with the surface electrodes and provides a better model of the underlying brain source activity.

## 2.5. Graph theoretical analysis

Based on the learned connectivity networks, graph theoretical analysis was used to extract the structural features from learned networks (Bullmore and Sporns, 2009). Traditional graph theoretical measures were used to characterize the network features in terms of density, global efficiency, clustering coefficient, and modularity. Density is defined as the fraction of present connections to all possible connections. Global efficiency describes the communication ability of the entire graph (Latora and Marchiori, 2001), and is defined as the average of the inverse shortest path. Clustering coefficient describes the degree to which nodes in a graph tend to cluster together. Modularity of the network is used to measure how well the network can be divided into the sub-modules (Newman and Girvan, 2004). A higher value of modularity demonstrates that the graph is better divided with tighter connections within modules. We used the Brain Connectivity Toolbox (Rubinov and Sporns, 2010) running Matlab (Natick, MA) to perform the graph theoretical analysis.

## 2.6. Construction of connectivity matrix

In this paper, we constructed the brain functional connectivity networks using the preselected EEG signals and an error-rate controlled network learning algorithm. Based on the learned connectivity networks, the graph measures were further calculated to extract the functional network features. EEG signals were interpolated at 27 locations (FP2, FPZ, FP1, F10, F8, F4, FZ, F3, F7, F9, A2, T8, C4, CZ, C3, T7, A1, P10, P8, P4, PZ, P3, P7, P9, O2, OA, O1) on the scalp using BESA's Virtual Standard 10 10 Average montage. EEG time series from these 27 locations were used to construct the brain connectivity networks with each channel representing one brain region in the network. The connectivity network graphs were then computed for each individual subject and for each emotional expression using false discovery rate controlled PC (PCFDR) algorithm, which is a statistical model that tests the conditional dependence/independence between any two regions based on all other brain regions (Li and Wang, 2009).


http://dx.doi.org/10.1016/j.heliyon.2017.e00373

We used partial correlation to evaluate the conditional independence, which estimates the directed interactions between any two brain regions after removing the effects of all other brain areas. The PC algorithm starts from a complete graph and tests for conditional independence in an efficient way. The PCFDR algorithm is designed to control the false discovery rate (FDR), which evaluates the proportion between the connections that are falsely detected to all those detected, below a specified predefined level. Compared to the traditional Type-1 and Type-2 error rates, FDR has more conservative error rate criteria for modeling brain connectivity due to its direct relation to the uncertainty of the networks of interest. The PCFDR algorithm and pseudo-code are described in details in (Li and Wang, 2009). F FDR threshold was set at the 5% level.

## 2.7. Baseline and Post-intervention testing

Prior to the start of the program all participants took part in a comprehensive baseline assessment, which included resting state EEG and neuropsychological testing. All testing was completed at the University of British Columbia. The neuropsychological testing and clinical scales took approximately 90 120 min to complete. All participants took part in the EEG testing. Only 6 TBI patients completed the initial neuropsychological testing at the first time point. 4 additional TBI patients received their baseline neuropsychological testing at the 3-month time point and thus we elected to exclude the additional 4 patients from analyses involving cognitive data.

Following the 3-month intervention program EEG was conducted on all 10 TBI patients. Symptoms of generalized anxiety, depression, and general psychological distress were assessed using the Generalized Anxiety Disorder – 7 (GAD-7; (Spitzer et al., 2006), Patient-Health Questionnaire (PHQ-9; (Kroenke et al., 2001), and Brief Symptom Inventory – 18 (BSI-18; (Derogatis and Melisaratos, 1983)).

## 2.8. Cognitive intervention

The Arrowsmith Program is a suite of cognitive exercises. The goal of these exercises is to improve cognitive functioning across a broad domain, for example, executive functions, reasoning and memory. Each individual's program was based on an assessment of that individual's learning profile to identify his or her specific areas of difficulty. Each individual had his or her specific schedule of tasks and exercises to be completed during the course of a day. The exercises for each individual student were uniquely based on their individually identified learning profiles. These include written, visual, auditory and computer exercises. Each cognitive program had a series of intensive and graduated tasks. Performance criteria of automaticity, consistency and accuracy are built in at all levels and an individual was required to meet these criteria before mastering to the next level of


http://dx.doi.org/10.1016/j.heliyon.2017.e00373

complexity. Goals were set daily, weekly and monthly and each month the individual's attained levels in each program was entered into a database and analyzed to see if progress met benchmark expectations. In order to maximize evaluation of transfer effects, the neuropsychological battery that was used to assess outcome was assembled independently of the cognitive exercise tasks that were used within the cognitive intervention program.

The cognitive intervention program took place four days a week for 4 − 5 hours a day for a 3-month period. Each day was composed of 6 8 blocks of different activities depending on individual's specific areas of weakness.

## 2.9. Analysis

Demographic data including age, gender and years of education were evaluated for between group differences with t-tests. T-tests were also performed to test differences in the EEG measures and the neuropsychological measures between groups at baseline. Two a priori EEG clusters of interest were selected for analysis based on our previous work examining changes in functional connectivity in adolescents with mTBI (Virji-Babul et al., 2014). EEG clusters of interest included F7 (L) inferior frontal gyrus (IFG) and F10 (R) inferior frontal gyrus. Paired t-tests were conducted to compare baseline and post-intervention scores in the TBI group. Pearson correlations were conducted to evaluate specific correlations between graph theory metrics and the individual and global composite neuropsychological scores.

# 3. Results

## 3.1. Differences between groups at baseline

Table 2 shows the mean scores (SD) on the global composite score based on the mean of all test scores. As expected, at baseline the TBI group performed worse on the overall composite score although the difference did not reach statistical significance as a likely result of diminished power. Consistent with this, the

**Table 2.** Global composite scores for the controls, TBI participants at baseline and TBI participants at 3 months post intervention.

| Cognititon Composite (Z score) | | N | Mean | Std. Deviation | Significance (2 tailed) |
|---|---|---|---|---|---|
| Control vs. TBI | Control | 12 | .1166 | .62026 | .102 |
| | TBI | 6 | .5823 | 1.10851 | |
| Baseline vs. 3 month | Baseline | 6 | .5823 | 1.1085 | .000 |
| | 3 Month | 6 | .0103 | 1.3002 | |


http://dx.doi.org/10.1016/j.heliyon.2017.e00373

Cohen's d effect size for the difference was .78, which indicates a moderate to large difference.

At baseline there were no significant differences between groups for any of the four graph metrics associated with global connectivity: density ($t(17) = 1.03$, $p = 0.32$, $d = 0.47$), global efficiency ($t(17) = -0.278$, $p = 0.78$, $d = -0.13$), modularity ($t(17) = 1.05$, $p = 0.31$, $d = 0.48$), clustering coefficient, $t(17) = 1.612$, $p = 0.125$, $d = 0.74$.

In contrast with the global metrics we found significant differences in local metrics at the F10 and F7 electrode clusters corresponding to the right and left inferior frontal gyrus, respectively between groups (Fig. 1). Specifically, the F10 hub value was significantly higher compared to the healthy control group, $t(17) = 2.24$, $p = 0.039$, $d = 1.01$. A number of graph metrics for F7, showed significant decreases in the TBI group in comparison with the healthy control group. These included F7 degree, $t(17) = -2.28$, $p = 0.036$, $d = -1.05$, F7 betweenness, $t(17) = -2.41$, $p = 0.028$, $d = -1.1$ and F7 hub value, $t(17) = -2.28$, $p = 0.035$, $d = -1.05$. These results suggest that network connections are denser in the right inferior frontal regions and less dense in the left inferior frontal regions in the TBI group.

## 3.2. Cognitive training related changes

Table 2 also shows the scores following the three-month cognitive intervention in the participants that completed testing at baseline and post-intervention. A paired-

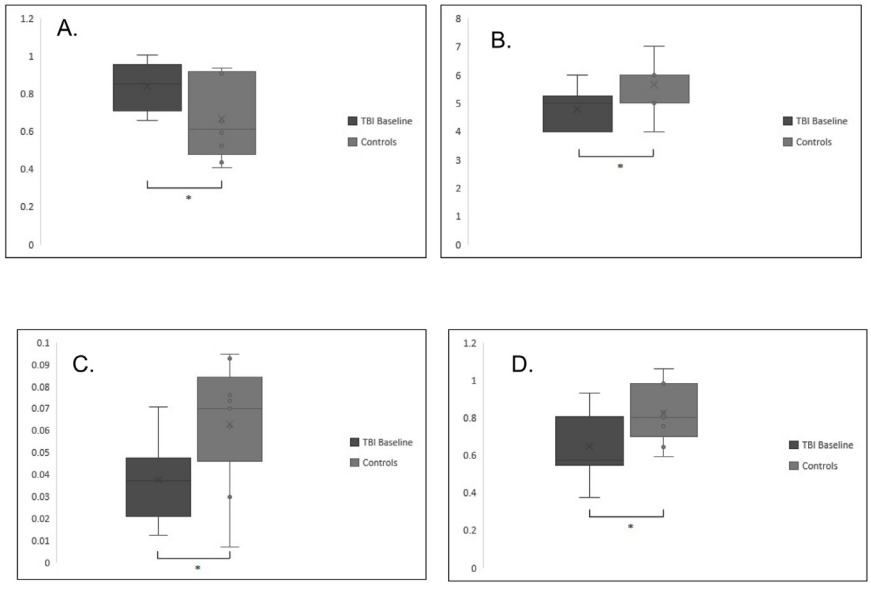



**Fig. 1.** Baseline functional connectivity measures showing differences between TBI Baseline and controls in local connectivity measures. (A. F10 Hub Value; B. F7 Degree; C. F7 Betweenness; D. F7Hub Value).


http://dx.doi.org/10.1016/j.heliyon.2017.e00373

test showed that there was a statistically significant increase in the composite score between baseline and post-intervention in the TBI group. In addition, there were no significant changes in generalized anxiety, $t(5) = 0.08$, $p = 0.94$, depressive symptoms, $t(5) = 0.86$, $p = 0.43$, or general psychological distress, $t(5) = 0.58$, $p = 0.59$.

In terms of functional connectivity, no changes were seen in the global connectivity measures. For the local connectivity measures, we noted changes in specific graph theory metrics at the F10 and F7 electrode clusters. Fig. 2 shows that at F10, degree, [$t(9) = 3.35$, $p = 0.008$] (Fig. 2A), betweenness [$t(9) = 3.52$, $p = 0.007$] (Fig. 2B), and hub value [$t(9) = 3.53$, $p = 0.006$] (Fig. 2C) showed a statistically significant decrease following the intervention. At F7, measures increased, however none were statistically significant: degree, $t(9) = -0.896$, $p = 0.394$, betweenness, $t(9) = -1.05$, $p = 0.323$ and hub value, $t(9) = -1.01$, $p = 0.338$.

Fig. 3 shows the relationship between global density and the overall composite score. Note that there is a positive correlation in the controls with higher scores associated with higher global density (P=.63). In the TBI group there was a negative correlation between these variables at baseline (P = -.39). Following intervention, there was a trend towards a positive slope (P=.28). Fig. 4 shows the relationship between the RAVLT and global density (4A) and the RAVLT and F10 degree (4B). Both correlations show a change from a negative correlations at baseline (P = -.38; P = -.33) to a positive correlation at 3 months post intervention (P=.69; P=.77). No other correlations were found to be significant.

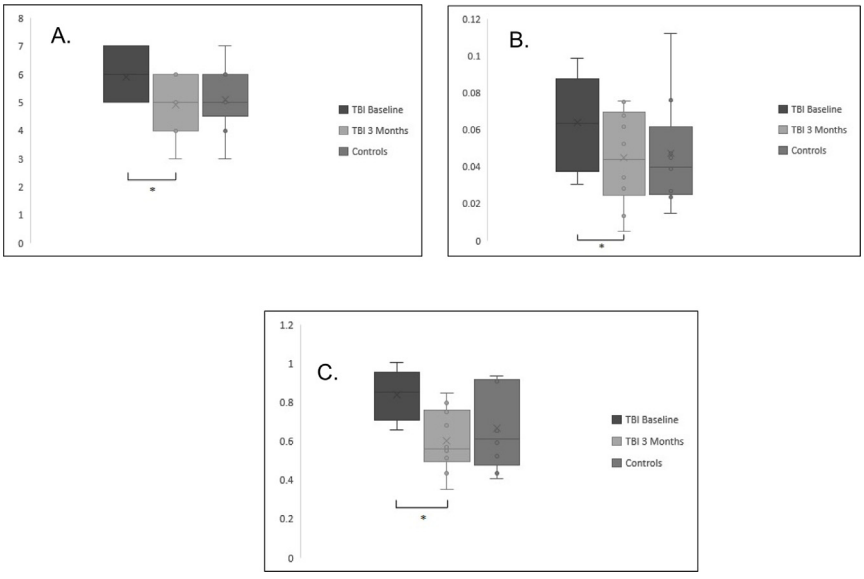



**Fig. 2.** Post intervention connectivity measures for TBI Baseline, TBI 3 Months Post Intervention compared to Controls, showing significant changes after 3 months in F10 Degree (A.), F10 Betweenness (B.) and F10 F10Hub Value (C.).


http://dx.doi.org/10.1016/j.heliyon.2017.e00373

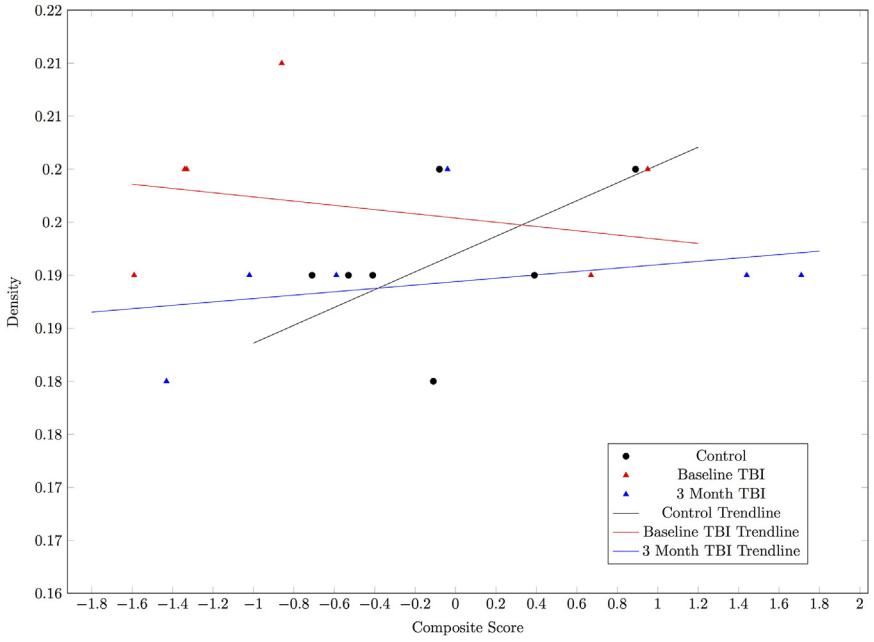



**Fig. 3.** Scatter plot showing relationship between Global Density and Global composite score in controls, TBI participants at baseline and TBI participants at 3 months post intervention.

## 4. Discussion

The purpose of this study was to pilot measures using graph theory analysis to evaluate the feasibility of these measures to capture change following a 3 month program of cognitive intervention and to determine if these changes correlated with measures of cognition. The data from our pilot study provides preliminary evidence for improved performance in cognition and corresponding changes in local functional network connectivity in bilateral frontal regions following a 3 month cognitive intervention program in a small sample of adults with chronic TBI.

Graph theoretical analysis at baseline revealed that there were no significant differences in global measures of functional connectivity between groups suggesting that in this cohort, there was no change in resting state global network connectivity, although there was evidence of disruption in the relationship between global metrics and measures of cognition. We did find significant changes in local networks in this group. Specifically, we observed increases in graph metrics in F10 and decreases in F7. F10 corresponds to the (R) inferior frontal gyrus (IFG) and F7 to the (L) IFG (Koessler et al., 2009). The IFG is a key region involved in three processes of cognitive control: working memory, task switching and inhibitory control (Sundermann and Pfleiderer, 2012). The increase in hub value of IFG on the right and decrease on the left may seem paradoxical, but it suggests a disruption of network organization that is centered around the frontal regions. The hub value


http://dx.doi.org/10.1016/j.heliyon.2017.e00373

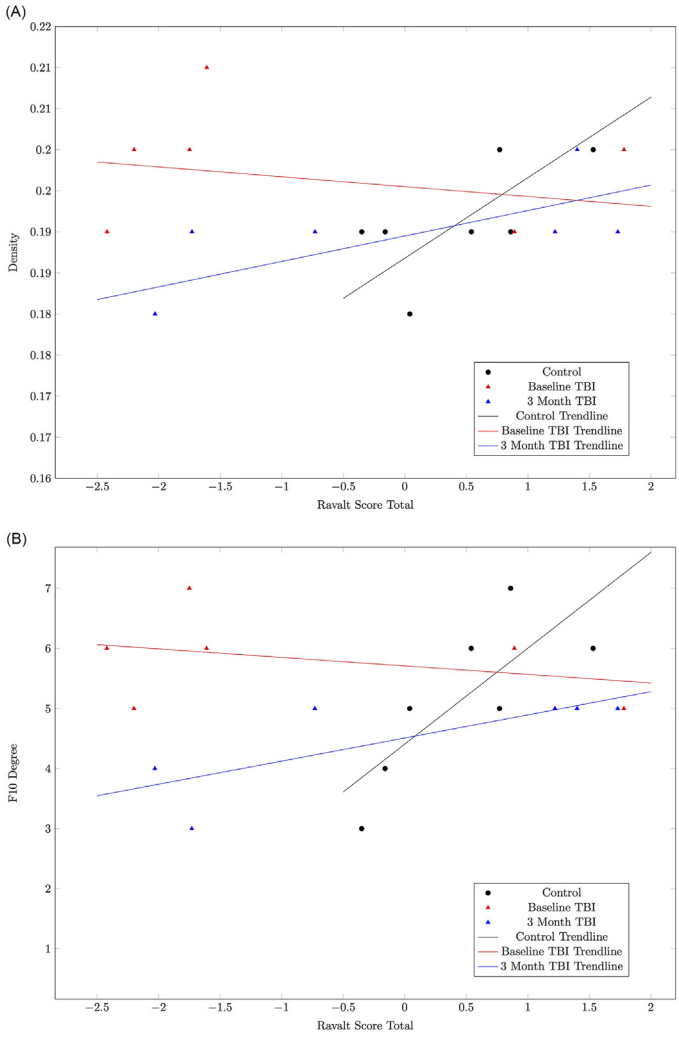



**Fig. 4.** (A). Scatter plot showing the relationship between RAVALT and Density in controls, TBI participants at baseline and TBI participants at 3 months post intervention. (B). Scatter plot showing the relationship between RAVALT and F10 Degree in controls, TBI participants at baseline and TBI participants at 3 months post intervention.

of one node is related to the structure of its neighbors, where a high value means that there are other key nodes that are connected with that hub. The increased value of the hub on the right indicates hyper-connectivity and the decrease on the left suggests hypo-connectivity. Overall, the consequences of disruption in the physical network due to TBI are reflected in disruptions in functional connectivity. These types of disruptions in functional connectivity have previously been reported in the literature. Hillary et al. (Hillary et al., 2014) found increased functional connectivity specifically in the brain regions that are most highly connected or "rich hubs". We have also previously found increased functional connectivity in adolescents with mTBI in frontal regions (Borich et al., 2014). Palacios and colleagues recently reported increased functional connectivity within the frontal


http://dx.doi.org/10.1016/j.heliyon.2017.e00373

lobe in patients with chronic TBI. In contrast, Pandit et al., reported decreased functional connectivity in patients with chronic TBI (Pandit et al., 2013).

Although initial hypotheses in the literature related to increased connectivity have focused on compensatory strategies as a mechanism to account for loss of structural connectivity, more recently, Hilary et al. (Hillary et al., 2015) have shown that hyper-connectivity is a common finding across a range of neurological conditions including TBI, multiple sclerosis, mild cognitive impairment and Alzheimer's disease. They hypothesize that increased connectivity may allow the brain to continue to meet task demands in the face of network disruption. Importantly, they suggest that this increased connectivity comes at the cost of slowed processing speed and cognitive fatigue. Our data shows that increased connectivity in the frontal regions is in fact correlated with lower cognitive scores suggesting that greater resources are being used that may lead to lower information processing efficiency.

Although hyper-connectivity is a common finding across a range of neurological disorders, from a network perspective it is unlikely that this is the only response to structural changes in the brain. Across all brain networks it is more likely that there is a combination of hyper and hypo connectivity that reflects the changes in different brain networks (Hillary et al., 2015). Indeed, our data supports this by showing that hyper-connectivity is not uniform across brain regions following TBI. Furthermore, the pattern of connectivity that we observed in each hemisphere provides data for the hypothesis that detailed analysis of network organization across the whole brain is necessary to understand and map the patterns of brain connectivity that underlie the cognitive deficits in individuals with TBI.

## 4.1. Training related changes in cognition

Within several important constraints of the present study (i.e. small sample size and a lack of a TBI control group who did not receive cognitive intervention which is necessary to rule out practice effects for cognitive improvements), the analysis of cognitive performance from baseline to post-treatment in patients suggested trends in improvements in overall cognition. Importantly, these improvements were observed in a sample of chronic patients. There are few published studies that have examined the effectiveness of cognitive intervention in patients who are in the chronic stages of recovery(Cook et al., 2014).

In a recent re-analysis of meta-analytic studies assessing the effects of cognitive rehabilitation on acquired brain injury, it was concluded that patients with traumatic brain injuries were more likely to receive benefit from cognitive retraining targeting attention based tasks (Cicerone et al., 2011). Because the cognitive measures included in the neuropsychological battery within this study


http://dx.doi.org/10.1016/j.heliyon.2017.e00373

were highly attention-based, this likely maximized the likelihood of observing cognitive gains as a result of the cognitive intervention.

## 4.2. Training related changes in functional connectivity

Interestingly, our data show that cognitive intervention lead to changes in the organization of brain networks such that connectivity within hub areas are reorganized, and this occurred with a parallel improvement in cognition. In particular, we found a small shift in the correlation between overall Global density and the global composite score in the TBI group which was negatively correlated at baseline and moved to a slight positive trend following the intervention period. We also observed shifts in correlations between auditory verbal learning and global connectivity as well as measures related to brain regions in the right inferior frontal gyrus. These changes suggest a subtle reorganization between brain and behavior relationships that were disrupted at baseline.

Our results provide preliminary evidence that participating in an intensive cognitive intervention program was associated with neuroplastic changes in adults with chronic TBI that occurred in parallel with improvements in cognition. Overall, we observed a shift from a baseline pattern of network organization that may be characterized by neural inefficiency and decreased cognition to a reorganization that reflected improved efficiency with possible improvements in fluid cognition. Importantly this data suggests that brain network organization is capable of reorganization even in chronic patients with intense intervention. Further work with a larger sample is clearly needed to understand the nuances of how brain organization impacts on cognitive ability and performance.

# Declarations

## Author contribution statement

Shaun Porter: Performed the experiments; Analyzed and interpreted the data; Wrote the paper.

Ivan Torres: Conceived and designed the experiments; Analyzed and interpreted the data; Wrote the paper.

William Panenka: Conceived and designed the experiments.

Zahra Rajwani: Performed the experiments; Analyzed and interpreted the data.

Delrae Fawcett, Amna Hyder: Analyzed and interpreted the data.

Naznin Virji-Babul: Conceived and designed the experiments; Performed the experiments; Analyzed and interpreted the data; Wrote the paper.


http://dx.doi.org/10.1016/j.heliyon.2017.e00373

## Funding statement

This work was supported by Mitacs-Accelerate Graduate Research Internship Program in collaboration with the Eaton Educational Group.

## Competing interest statement

The authors declare no conflict of interest.

## Additional information

No additional information is available for this paper.

## Acknowledgements

We would like to thank all the participants who took part in this study.

## References

Arnemann, K.L., Chen, A.J., Novakovic-Agopian, T., Gratton, C., Nomura, E.M., D'Esposito, M., 2015. Functional brain network modularity predicts response to cognitive training after brain injury. Neurology 84 (15), 1568 1574.

Borich, M., Babul, A.N., Huang, P.H., Boyd, L., Virji-Babul, N., 2014. Alterations in resting state brain networks in concussed adolescent athletes. J. Neurotrauma.

Bressler, S.L., Menon, V., 2010. Large-scale brain networks in cognition: emerging methods and principles. Trends Cogn. Sci. 14 (6), 277 290.

Broglio, S.P., Puetz, T.W., 2008. The effect of sport concussion on neurocognitive function, self-report symptoms and postural control: a meta-analysis. Sports Med. 38 (1), 53 67.

Bullmore, E., Sporns, O., 2009. Complex brain networks: graph theoretical analysis of structural and functional systems. Nat. Rev. Neurosci. 10 (3), 186 198.

Caeyenberghs, K., Leemans, A., Heitger, M.H., Leunissen, I., Dhollander, T., Sunaert, S., Swinnen, S.P., 2012. Graph analysis of functional brain networks for cognitive control of action in traumatic brain injury. Brain 135 (Pt 4), 1293 1307.

Caeyenberghs, K., Metzler-Baddeley, C., Foley, S., Jones, D.K., 2016. Dynamics of the Human Structural Connectome Underlying Working Memory Training. J. Neurosci. 36 (14), 4056 4066.

Cicerone, K.D., Langenbahn, D.M., Braden, C., Malec, J.F., Kalmar, K., Fraas, M., Ashman, T., 2011. Evidence-based cognitive rehabilitation: updated review of the literature from 2003 through 2008. Arch. Phys. Med. Rehabil. 92 (4), 519 530.

 http://dx.doi.org/10.1016/j.heliyon.2017.e00373

Cook, L.G., Chapman, S.B., Elliott, A.C., Evenson, N.N., Vinton, K., 2014. Cognitive gains from gist reasoning training in adolescents with chronic-stage traumatic brain injury. Front Neurol. 5, 87.

Derogatis, L.R., Melisaratos, N., 1983. The Brief Symptom Inventory: an introductory report. Psychol. Med. 13 (3), 595 605.

Finkelstein, E.A., Phaedra, S.C., Ted, R.M., 2006. Incidence and economic burden of injuries in the United States. Oxford University Press.

Friston, K.J., 2011. Functional and effective connectivity: a review. Brain Connect. 1 (1), 13 36.

Hillary, F.G., Rajtmajer, S.M., Roman, C.A., Medaglia, J.D., Slocomb-Dluzen, J. E., Calhoun, V.D., Wylie, G.R., 2014. The rich get richer: brain injury elicits hyperconnectivity in core subnetworks. PLoS One 9 (8) e104021.

Hillary, F.G., Roman, C.A., Venkatesan, U., Rajtmajer, S.M., Bajo, R., Castellanos, N.D., 2015. Hyperconnectivity is a fundamental response to neurological disruption. Neuropsychology 29 (1), 59 75.

Jolles, D.D., van Buchem, M.A., Crone, E.A., Rombouts, S.A., 2013. Functional brain connectivity at rest changes after working memory training. Hum. Brain Mapp. 34 (2), 396 406.

Kennedy, M.R.T., Coelho, C., Turkstra, L., Ylvisaker, M., Sohlberg, M.M., Yorkston, K., Kan, P.F., 2008. Intervention for executive functions after traumatic brain injury: A systematic review, meta-analysis and clinical recommendations. Neuropsychol. Rehabil. 18 (3), 257 299.

Koessler, L., Maillard, L., Benhadid, A., Vignal, J.P., Felblinger, J., Vespignani, H., Braun, M., 2009. Automated cortical projection of EEG sensors: anatomical correlation via the international 10-10 system. Neuroimage 46 (1), 64 72.

Kraus, J., Schaffer, K., Ayers, K., Stenehjem, J., Shen, H., Afifi, A.A., 2005. Physical complaints, medical service use, and social and employment changes following mild traumatic brain injury: a 6-month longitudinal study. J. Head Trauma Rehabil. 20 (3), 239 256.

Kroenke, K., Spitzer, R.L., Williams, J.B., 2001. The PHQ-9: validity of a brief depression severity measure. J. Gen. Intern. Med. 16 (9), 606 613.

Kundu, B., Sutterer, D.W., Emrich, S.M., Postle, B.R., 2013. Strengthened effective connectivity underlies transfer of working memory training to tests of short-term memory and attention. J. Neurosci. 33 (20), 8705 8715.

 http://dx.doi.org/10.1016/j.heliyon.2017.e00373

Langlois, J.A., Rutland-Brown, W., Wald, M.M., 2006. The epidemiology and impact of traumatic brain injury: a brief overview. J. Head Trauma Rehabil. 21 (5), 375 378.

Latora, V., Marchiori, M., 2001. Efficient behavior of small-world networks. Phys. Rev. Lett. 87 (19), 198701.

Lezak, M.D., Lezak, M.D., 2004. Neuropsychological assessment, Oxford. Oxford University Press, New York.

Li, J., Wang, Z.J., 2009. Controlling the false discovery rate of the association/causality structure learned with the PC algorithm. J. Mach. Learn. Res. 10 (2), 475 514.

Malec, J.F., Brown, A.W., Leibson, C.L., Flaada, J.T., Mandrekar, J.N., Diehl, N. N., Perkins, P.K., 2007. The mayo classification system for traumatic brain injury severity. J. Neurotrauma 24 (9), 1417 1424.

McAllister, T.W., Flashman, L.A., McDonald, B.C., Saykin, A.J., 2006. Mechanisms of working memory dysfunction after mild and moderate TBI: evidence from functional MRI and neurogenetics. J. Neurotrauma 23 (10), 1450 1467.

Menon, D.K., Schwab, K., Wright, D.W., Maas, A.I., Demographics, Clinical Assessment Working Group of the, International, . . . Psychological, Health, 2010. Position statement: definition of traumatic brain injury. Arch Phys Med Rehabil 91 (11), 1637 1640.

Nakamura, T., Hillary, F.G., Biswal, B.B., 2009. Resting network plasticity following brain injury. PLoS One 4 (12) e8220.

Newman, M.E., Girvan, M., 2004. Finding and evaluating community structure in networks. Phys. Rev. E Stat. Nonlin. Soft. Matter Phys. 69 (2 Pt 2), 026113.

Pandit, A.S., Expert, P., Lambiotte, R., Bonnelle, V., Leech, R., Turkheimer, F.E., Sharp, D.J., 2013. Traumatic brain injury impairs small-world topology. Neurology 80 (20), 1826 1833.

Podell, K., Gifford, K., Bougakov, D., Goldberg, E., 2010. Neuropsychological assessment in traumatic brain injury. Psychiatr. Clin. North Am. 33 (4), 855 876.

Rabinowitz, A.R., Levin, H.S., 2014. Cognitive sequelae of traumatic brain injury. Psychiatr. Clin. North Am. 37 (1), 1 11.

Reitan, Ralph M., Wolfson, Deborah., 1993. The Halstead-Reitan neuropsychological test battery: theory and clinical interpretation. Neuropsychology Press, S. Tucson, Arizona.

 http://dx.doi.org/10.1016/j.heliyon.2017.e00373

Rubinov, M., Sporns, O., 2010. Complex network measures of brain connectivity: uses and interpretations. Neuroimage 52 (3), 1059 1069.

Sheehan, D.V., Lecrubier, Y., Sheehan, K.H., Amorim, P., Janavs, J., Weiller, E., Dunbar, GC, 1998. The Mini-International Neuropsychiatric Interview (M. I. N. I.): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. J. Clin. Psychiatry 59 (Suppl 20), 22 33 quiz 34-57.

Spitzer, R.L., Kroenke, K., Williams, J.B., Lowe, B., 2006. A brief measure for assessing generalized anxiety disorder: the GAD-7. Arch. Intern. Med. 166 (10), 1092 1097.

Sundermann, B., Pfleiderer, B., 2012. Functional connectivity profile of the human inferior frontal junction: involvement in a cognitive control network. BMC Neurosci. 13, 119.

Teasdale, G., Jennett, B., 1974. Assessment of coma and impaired consciousness. A practical scale. Lancet 2 (7872), 81 84.

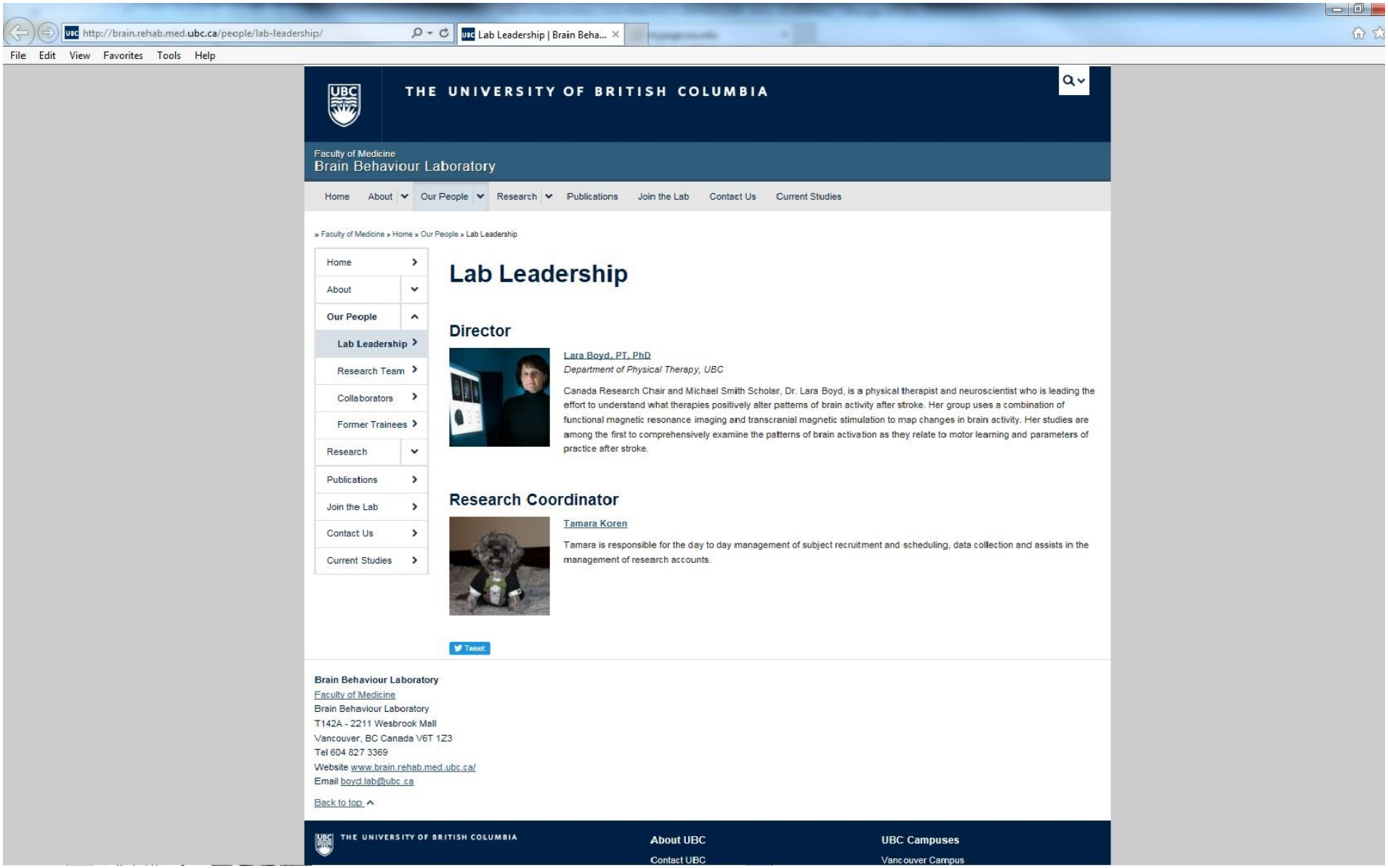
Virji-Babul, N., Hilderman, C.G., Makan, N., Liu, A., Smith-Forrester, J., Franks, C., Wang, Z.J., 2014. Changes in functional brain networks following sports-related concussion in adolescents. J. Neurotrauma 31 (23), 1914 1919.

Watts, D.J., Strogatz, S.H., 1998. Collective dynamics of ‘small-world' networks. Nature 393 (6684), 440 442.

Wu, J., Zhang, J., Liu, C., Liu, D., Ding, X., Zhou, C., 2012. Graph theoretical analysis of EEG functional connectivity during music perception. Brain Res. 1483, 71 81.


http://dx.doi.org/10.1016/j.heliyon.2017.e00373

## Annexure H


http://brain.rehab.med.ubc.ca/people/lab-leadership/
Lab Leadership | Brain Beha...
File Edit View Favorites Tools Help
THE UNIVERSITY OF BRITISH COLUMBIA
Faculty of Medicine
Brain Behaviour Laboratory
Home About Our People Research Publications Join the Lab Contact Us Current Studies
» Faculty of Medicine » Home » Our People » Lab Leadership
Home
About
Our People
Lab Leadership
Research Team
Collaborators
Former Trainees
Research
Publications
Join the Lab
Contact Us
Current Studies
Lab Leadership
Director
Lara Boyd, PT, PhD
Department of Physical Therapy, UBC
Canada Research Chair and Michael Smith Scholar, Dr. Lara Boyd, is a physical therapist and neuroscientist who is leading the effort to understand what therapies positively alter patterns of brain activity after stroke. Her group uses a combination of functional magnetic resonance imaging and transcranial magnetic stimulation to map changes in brain activity. Her studies are among the first to comprehensively examine the patterns of brain activation as they relate to motor learning and parameters of practice after stroke.
Research Coordinator
Tamara Koren
Tamara is responsible for the day to day management of subject recruitment and scheduling, data collection and assists in the management of research accounts.
Tweet
Brain Behaviour Laboratory
Faculty of Medicine
Brain Behaviour Laboratory
T142A - 2211 Wesbrook Mall
Vancouver, BC Canada V6T 1Z3
Tel 604 827 3369
Website www.brain.rehab.med.ubc.ca/
Email boyd.lab@ubc.ca
Back to top
THE UNIVERSITY OF BRITISH COLUMBIA
About UBC
Contact UBC
UBC Campuses
Vancouver Campus

## Annexure I

a place of mind
THE UNIVERSITY OF BRITISH COLUMBIA

# ARROWSMITH BRAIN IMAGING STUDY: END OF YEAR 1 UPDATE AND FUTURE PLANS

A proposal prepared by Lara Boyd of the UBC Faculty of Medicine | April 11, 2016

## OVERVIEW

Learning disabilities affect one in 10 Canadians, compromising their ability to acquire the fundamental skills of reading, writing, and mathematics. The challenges associated with learning disabilities may extend beyond school to other domains such as social skills, seriously hindering a child's ability to reach their full potential in life.

Recent advances in neuroimaging and neuroscience have led to an enormous growth in the understanding of the brain's capability to change, which is termed neuroplasticity. Given this new understanding of the dynamic nature of the brain, there is now acute interest in learning to exploit it to refine learning-based interventions to optimize outcomes and enhance function. One such learning-based intervention is the Arrowsmith Educational Program, which was designed to identify and strengthen deficient cognitive functions that are thought to be a source of specific learning disabilities in children. Enhancing our understanding of what brain functions and structures are affected by interventions such as the Arrowsmith program is critical to determining their effectiveness and refining their utility.

The University of British Columbia (UBC) partnered with the Vancouver-based Eaton Arrowsmith School to advance our mutual interest in exploring the scientific validity of neuroplasticity in youth. The research team is led by Dr. **Lara Boyd**, PT, PhD, Professor of Physical Therapy and Canada Research Chair in the Neurobiology of Motor Learning. The study represents an essential first step in expanding our understanding of how neuroplastic change may be operationalized in children with learning disabilities and what types of change are stimulated by the Arrowsmith program. The seed funding provided by the Eaton Foundation was successfully leveraged to secure additional funding for a much larger pilot study, which will provide the preliminary data needed to design a large-scale assessment of the Arrowsmith program's impact on brain plasticity. The published findings are also expected to catalyze future work in this area, which may lead to the development of novel educational interventions that optimally stimulate learning in children.

With additional funding, the team will increase the size of the control group, add testing of social and emotional development associated with the intervention, fund a full time posti  doctoral fellow and continue to follow children in the Arrowsmith group and matched controls through their second year of the program. This type of longeri  term follow up and analysis is critical as changes in the brains and behaviour of these children will undoubtedly continue to occur over the second year. More children are interested in participating in the study, but funding must be secured to allow them to enroll. The results of the expanded pilot study are expected to come out in Spring 2017.

## EXPERIMENTAL OVERVIEW

No brain imaging data that characterizes the impact of the Arrowsmith program has been published to date, and this is the first brain imaging study to explore the impact of the Arrowsmith program. The aims of the pilot study are:

1) To provide information regarding the feasibility of brain imaging in this population, and
2) To generate preliminary data to power and motivate future studies of the Arrowsmith program with larger scope.

The first aim has already been accomplished; brain imaging is feasible in this population. The second aim will be accomplished once the larger control groups are recruited and tested.

To begin to assess how the Arrowsmith Program may alter the brain, we employing a multiple time point design to capture neuroplastic change associated with training in children with a learning disability. Each child enrolled will already have a diagnosis of learning disability. Educational psychologists confirm the diagnosis and administer a battery of educational and cognitive tests. Multiple forms of imaging take place, including structural (T1 anatomy, myelin water imaging) and functional (resting and taski based functional MRI).

Initially, we set out to test 20 children: 10 from the Arrowsmith program and 10 matched typically developing controls. By leveraging the seed funding provided by the Eaton Foundation, we have nearly tripled the number of children enrolled in the study. At this time (Fall 2015), 56 participants have been enrolled. Given the variability in how children learn and develop, the ability to enroll a larger sample has significantly increased our power to detect differences. This is a tremendously exciting development. We are now planning to study a total of 90 children from three groups: 1) children with learning disabilities who are enrolled in the Arrowsmith program, 2) children with learning disabilities who are enrolled in other educational programs, and 3) typically developing children who are matched for age and sex.

So far, we have tested (or are in the midst of testing) 32 children from the Arrowsmith program. These children and their families have come from Eaton Arrowsmith schools in Vancouver, White Rock, Victoria, and Seattle/Redmond, Washington. We have also 14 children with learning disabilities who are not in the Arrowsmith program and 10 children who are typically developing and do not have a learning disability. Our focus moving forward is to locate and enroll more children in the two control groups.

## INTERVENTION

Children in the Arrowsmith program continue with their regular education, which includes the engagement of the student in six 40-minute blocks of Arrowsmith cognitive exercises, five days per week. Children in the control group are necessary to control for the effects of development and educational exposure. Control participants will be matched for age, gender, handedness, years of education, and type of learning disability. A typically developing control group was added to the study to

factor in the effects of development on the measures. Recruitment of the controls is off by one year from that of the Arrowsmith group by design; this allows for careful matching across the three groups.

For each group, three brain imaging time points took place, separated by one year. Cognitive testing was completed at the beginning of the study (in the fall) and again at the end of the school year (in June), as a delay is required between many of the tests being employed. During the study, all children completed their normal coursework as assigned. Each child will be scanned three times (at the beginning, middle, and end of the school year).

## KEY FINDINGS TO DATE

At this point, the research team has performed interim analyses on the first 40 study participants (28 from Arrowsmith, 15 learning disabled controls, and 7 typically developing controls). In our examination of baseline data across our whole sample, we have discovered a strong relationship between myelination in the corpus callosum and reading ability (Fig. 1). This finding is important as it supports other work that suggested that callosal integrity is important for word attack. Our data extend these past findings and demonstrate a specific neurobiological substrate. Understanding the brain-behaviour relationships that are key to academic achievement during development illustrates biomarkers that may be shifted with interventions and can also serve as therapeutic targets.

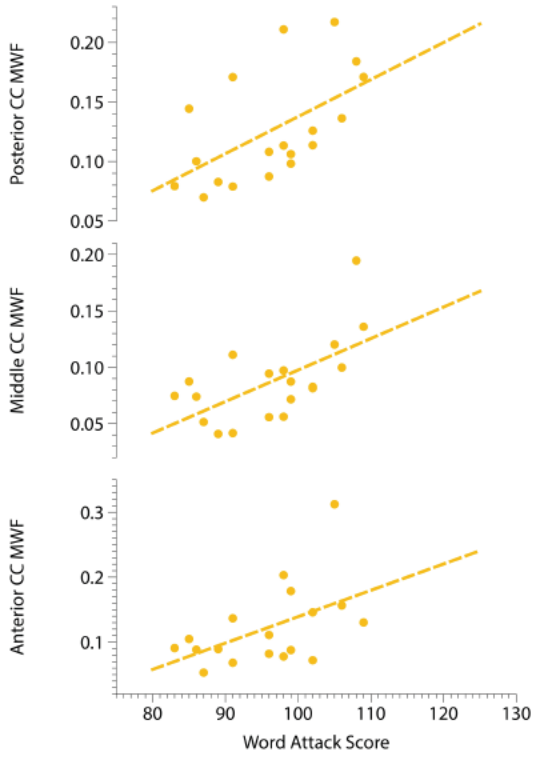



Fig.1. Relationship between corpus callosum myelin water fraction and word attack score.

We have also considered how the size of certain brain regions may relate to educational ability. This approach relies on the ability to understand the morphometrics of the brain. Here we discovered that a larger right insular cortex is evident in children with higher math ability (above a normalized score of 100), as measured by the Woodcock-Johnson III test (Fig. 2). This finding is very interesting in light of our functional MRI findings (see below) that indicate the insula is activated during one of the signature Arrowsmith cognitive exercises, the clocks task. Although these data are preliminary, they suggest that improvement on clocks may alter insular structure and function, and that perhaps these alterations in brain may promote changes in math ability.

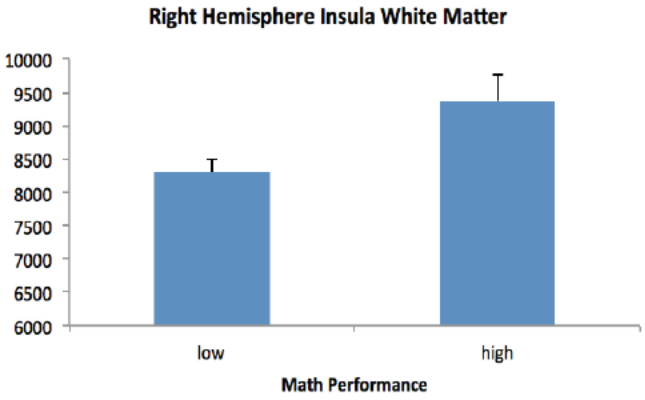



Fig. 2. The right insula is larger in children with higher math performance scores (as measured by the Woodcock-Johnson III).

We also conducted analyses of brain function during the clock task. In this work, we note that a unique network is activated in association with increased proficiency with this task (Fig. 3). Notably, the children with learning disability in the Arrowsmith group demonstrate significant activity in the insular cortex while performing the task when time points 1 and 2 are contrasted.

Finally, we have considered the effect of the program on resting state brain activation. Interestingly, we note very low amounts of activity in the prefrontal cortex at time point 1 in children with learning disability (Fig 4A). However, after just three months in the Arrowsmith program, this activity has shifted such that a small yet notable prefrontal region of activity is emerging (Fig 4B). This is much more similar to normal and suggests that the Arrowsmith program may be helping to facilitate a more normal resting state pattern of brain activity. A full analysis of all data will be required to confirm this finding, yet our early data are highly encouraging.

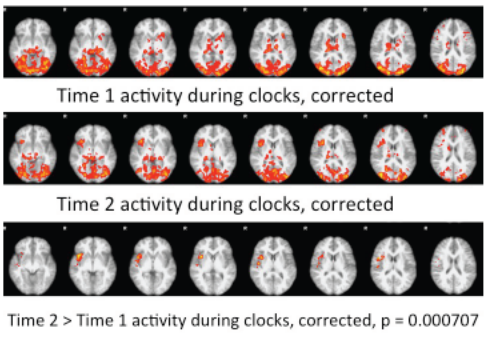



Fig. 3: Functional MRI data during clocks task at time points 1 and 2. The unique network is shown in the lowest panel.

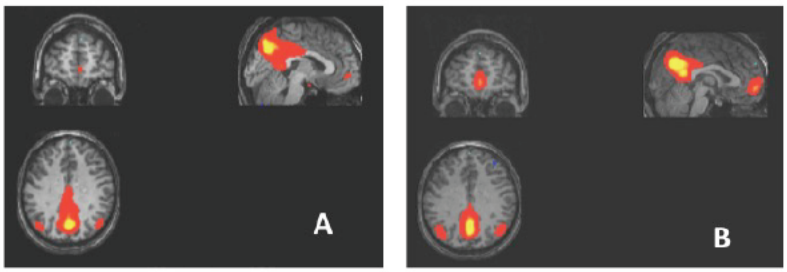



Fig. 4: Resting state functional MRI data from time points 1 and 2. Note the emergence of a frontal network at time point 2.

We have already been able to present and publish in abstract form data from time point 1 that characterizes the relationships between brain function and educational ability (Lakhani et al. (2015) Organization for Human Brain Mapping). These results show that myelination increases with age and confirms past work in this field. The presentation of this work catalyzed a new method where structural and segmented brain data could be merged to generate highly specific cortical maps. After working to optimize this approach for the past three months, we recently completed a full re-analysis of our data. We are presently interpreting these results. It is important to note that this work catalyzed both a unique approach to understanding how learning disability affects the brain and also a new method that we are applying in other work considering stroke and diabetes. As such, it has had a large impact on the neuroimaging field in general.

## FUTURE PLANS

Additional funding will enable us to continue recruiting children to the study until the goal of 90 participants in the intervention group and two control groups is achieved, as well as to continue following the children in the intervention group through their second year of the Arrowsmith program. The additional study participants will be invaluable in generating enough power to detect significant

differences among the children, and these data will be crucial in determining the long-term impact of the Arrowsmith program on children who have received this training.

A new aim in our research plan is to build a more complete picture of how the Arrowsmith program impacts social and emotional health in children. To accomplish this goal, we are now collaborating with Dr. Kim Schonert-Reichl, an expert in indexing social and emotional health in children (Schonert-Reichl, K.A. et al. *Developmental Psychology*, 2015, 51:52-66). Dr. Schonert-Reichl was originally a teacher, has a PhD in Education and directs the Human Early Learning Partnership at UBC. She and Dr. Boyd are collaborating to consider the question of how varied educational interventions impact social and emotional health, as well as patterns of neuroplastic change. This collaboration arose in response to the subjective observation of large amounts of change in the affect of the children who are participating in the Arrowsmith program. We suspect that new successes associated with this program are impacting the overall emotional well-being of the children who are enrolled in the Arrowsmith intervention. If so, this would be a critical discovery.

School is the first place where many children are exposed to a larger social world beyond the family, and it is during school years that children gradually acquire the social skills necessary to succeed in a complex world. Growing up without essential social and emotional skills such as compassion and empathy can lead to lifelong mental illness. By 2020, it is estimated that mental illness will represent one of the leading health care cost in the world. Further, children with learning disabilities are at higher risk for developing mental illness as adults, and suffer from greater rates of depression and anxiety as compared to their peers. Thus, a program that facilitates social and emotional health in children with learning disabilities has to potential to mitigate the development of serious mental illness and profoundly impact lives.

The addition of Dr. Schonert-Reichl's testing will not significantly increase either the testing burden on children or the costs associated with the study. Her battery of tests is computerized, can be completed in 1-hour sessions, and includes child self-report of: empathy, perspective taking, optimism, self-control, anxiety, social responsibility, and depressive symptoms (Schonert-Reichl et al., 2015). Our plan is to add this to our existing battery of testing in an effort to consider how the social and emotional health of children who are in the Arrowsmith program compares to both children with learning disabilities in other programs and typically developing children.

**For more information, please contact:**

a place of mind
UBC

**Fatima Hassam**, Associate Director of Development
UBC Faculty of Medicine
317 - 2194 Health Sciences Mall
Vancouver, BC V6T 1Z3
Phone: 604-822-8079 / Cell: 604-218-0508
Email: fatima.hassam@ubc.ca

## Annexure J

a place of mind
THE UNIVERSITY OF BRITISH COLUMBIA

**Faculty of Medicine** Vancouver Campus
Department of Physical Therapy
212 - 2177 Wesbrook Mall
Vancouver, BC Canada V6T 1Z3

Phone 604 822 8225
Fax 604 822 1870
physical.therapy@ubc.ca
www.physicaltherapy.med.ubc.ca

August 7, 2017

To Whom it May Concern,

This letter is to confirm that we are currently studying the impact of participation in the Arrowsmith Program on children with learning disabilities. This work is taking place at the University of British Columbia (UBC) in Vancouver, Canada. At this time two studies are taking place. The first is being led by Dr. Lara Boyd and is an investigation of the impact of the Arrowsmith program on brain structure and function (measured using magnetic resonance imaging [MRI]), and academic and cognitive achievement (measured using the Woodcock-Johnson III). This is a year long study with MRI, and academic and cognitive testing taking place at the beginning of the school year in September, and again at the end of the school year in June. To assure adequate numbers of participants this study has been running for 3.5 years. We recently completed data collection (June 2017) and have assessed 65 children who are between the ages of 9 and 17. Currently data analyses are underway and we anticipate that the results will be submitted for peer-review in an academic journal in the Fall of 2017. In addition, a smaller cohort was followed for a second year (N=20). These data are also being analyzed at this time.

In addition, a second study that seeks to understand the impact of the Arrowsmith program on social and emotional learning will launch in August 2017. Dr. Kimberly Schonert-Reichl at UBC is leading this study. This work will enroll a total of 250 children who will be recruited from the Arrowsmith program, as well as local public and private schools. It will also span 1-year and will consider how the classroom and school environment impacts the development of social and emotional skills. We will also consider the impact of school environment on stress hormone regulation (i.e., cortisol) and perform brain imaging in a sub-group of students (N=40). Data collection for this study will conclude in June 2018; results should be ready for peer-review and publication by fall of 2018.

Sincerely,

Lara A. Boyd, PhD, PT
Professor, Director, Brain Behaviour Lab
Health Research Advisor to the Vice-President Research, Department of Physical Therapy & Centre for Brain Health
Phone 604-822-7197
lara.boyd@ubc.ca
www.brain.rehab.med.ubc.ca

Kimberly A. Schonert-Reichl, Ph.D.
Professor, Director, Human Early Learning Partnership
School of Population and Public Health, Department of Educational & Counselling Psychology, and Special Education
Phone 604 822-2215
kimberly.schonert-reichl@ubc.ca
http://ecps.educ.ubc.ca/|
http://educ.ubc.ca/research/ksr/index.html

## Annexure K

CURRICULUM VITAE

# Gregory M. Rose, Ph.D.

**Center for Integrative Research in Cognition and Neural Science**
**Southern Illinois University Carbondale**
**Lindegren Hall, Room 308**
**600 Agriculture Drive**
**Carbondale, IL 62901**

## SCIENTIFIC INTERESTS

- Neurobiology of Learning and Memory
- Behavioral Models for Neurological and Psychiatric Disorders
- Drug Discovery for Cognitive Enhancement

## DEGREES

| | |
|---|---|
| **B.S.**, 1975, Cum Laude | University of California, Irvine; Major: Biological Sciences |
| **Ph.D.**, 1980 | University of California, Irvine; Major: Psychology |
| **Title of Dissertation** | **Physiological Analysis of the Hippocampus During Behavior** |
| **Certificate**, 1983 | Emily Griffith Opportunity School, Denver, CO: Commercial Baking and Pastry Making |

## EDUCATION

Undergraduate

| | |
|---|---|
| October 1971 - January 1972 | University of California, Berkeley; Major: Chemistry |
| January 1972 - June 1975 | University of California, Irvine; Major: Biological Sciences |

Graduate

| | |
|---|---|
| October 1975 - April 1977 | Developmental and Cell Biology (School of Biological Sciences), University of California, Irvine |
| April 1977 - September 1980 | Psychology (School of Social Sciences), U.C. Irvine<br>Advisor: Gary S. Lynch |

## PROFESSIONAL MEMBERSHIPS

- International Behavioral Neuroscience Society
- International Brain Research Organization
- Sigma Xi

♦ Society for Neuroscience

**POSITIONS HELD**

| | |
|---|---|
| January 2009 – present | **Director**<br>Center for Integrative Research in Cognition and Neural Science<br>Southern Illinois University, Carbondale, IL<br>**Professor**<br>Departments of Anatomy and Physiology (as of 1/1/2010), SIU School of Medicine, Carbondale, IL |
| July 2007 – present | **Principal**<br>Cognition Consultants, Guilford, CT |
| November 2004 – July 2007 | **Director**<br>Neuroscience Discovery Biology<br>Bristol-Myers Squibb Co., Wallingford, CT |
| May 2004 – November 2004 | **Vice President**<br>Functional Neuroscience<br>Memory Pharmaceuticals Corp., Montvale, NJ |
| January 2002 – May 2004 | **Director**<br>Functional Neuroscience<br>Memory Pharmaceuticals Corp., Montvale, NJ |
| September 1999 – January 2002 | **Associate Director**<br>Head of Behavioral Pharmacology and<br>Head of Neurophysiology<br>Memory Pharmaceuticals Corp., Montvale, NJ |
| October 1997 – September 1999 | **Principal Scientist**<br>Neuroscience Drug Discovery<br>Bristol-Myers Squibb Co., Wallingford, CT |
| July 1989 – October 1997 | **Associate Professor**<br>Department of Pharmacology, University of Colorado Health Sciences Center (UCHSC), Denver, CO |
| April 1989 – October 1997 | **Associate Research Career Scientist**<br>Veterans Affairs Medical Center, Denver, CO |
| July 1984 -- July 1989 | **Assistant Professor**<br>Department of Pharmacology, UCHSC, Denver, CO |
| November 1981 -- April 1989 | **Research Biologist**<br>Medical Research Service, Veterans Affairs Medical Center, Denver, CO |
| August 1989 -- October 1989 | **Visiting Scientist**<br>Douglas Hospital Research Center, McGill University, Montreal, Canada<br>Sponsor: Alain P. Gratton |

May 1985 -- July 1985 **Visiting Scientist**
Neurosurgery Research Clinic, University Hospital, Zürich, Switzerland
Sponsor: Helmut L. Haas

April 1981 -- July 1984 **Regular Fellow**
Department of Pharmacology, UCHSC, Denver, CO
Mentor: Barry J. Hoffer

September 1980 -- April 1981 **Postdoctoral Fellow**
Friedrich Miescher Laboratory, Max-Planck-Institute, Tübingen, Germany
Mentor: Wilfried E. Seifert

## GRANT SUPPORT

Current

July 2009 – July 2010 **Principal Investigator, SIU Center for Alzheimer's Disease and Related Disorders Basic Research Grant**
*Cognitive Consequences of Metabolic Alterations in 3xTg Mice*
The goal of this work is to characterize age-related cognitive deficits in a mouse model of Alzheimer's disease and investigate the relationship of peripheral and central metabolic markers to behavioral performance.

September 2009 – September 2010 **Principal Investigator, SIU Interdisciplinary Research Seed Grant**
*Fatty Acid Profiling in a Model of Alzheimer's Disease*
The goal of this study is to evaluate age-related changes in brain and plasma long-chain polyunsaturated fatty acid levels in cognitively characterized 3xTg mice.

Past

April 1984 – October 1997
Renewed in 1986, 1989, 1994, 1997 **Principal Investigator, VA Merit Review Grant**
*Cellular Neurobiology of Age-Induced Changes in Hippocampal Function*
Major goals of this work were to characterize age-related changes in the rat hippocampus, examine for correlations between changes in this system and spatial learning deficits, and test for beneficial effects of subchronic neurotrophin (e.g., NGF) administration.

April 1989 – October 1997
Renewed in 1994 **Principal Investigator, Project 3, National Institutes of Mental Health (NIMH) Program Project**
*Genetics and Neurobiology of Schizophrenia*
The goal of this portion of a program project (R. Freedman, Program Director) was to develop rodent auditory gating models to determine the neurobiological basis of sensory processing deficits seen in mania and schizophrenia, with a particular focus on the involvement of nicotinic cholinergic receptors.

April 1989 – October 1997
Renewed in 1994 **Associate Research Career Scientist Award**
Veterans Affairs Medical Center
This award, given to high-performing Ph.D. scientists within the VA Research Service, provided salary support.

September 1991 -- October 1997
Renewed in 1997

**Principal Investigator, National Institute on Aging (NIA) R01**
*Cholinergic Circuits and Hippocampal Function in Aging*
Major goals of this grant were to characterize age-related changes in hippocampal cholinergic (particularly nicotinic) receptors and evaluate their relationship to learning deficits. The grant has also supported the development of a model of cytochrome oxidase inhibition in order to study the effects of selective bioenergetic defects upon learning and memory.

October 1995 – October 1997

**Co-Investigator, National Institute on Aging (NIA) R01**
*Glucocorticoids, Stress and Hippocampal Aging*
The goals of this project (M. Meaney, P.I.) were to determine whether subpopulations of adult rats that are hyper-responsive to stress are more likely to suffer age-relate cognitive impairments, and to examine chronic antidepressant treatment as a prophylactic measure. My subcontract provided funds to examine hippocampal plasticity in these animals.

January 1994 -- January 1996

**Principal Investigator, Amgen Basic Research Grant**
*Growth Factors and Aging*
This project evaluated the effects of several neurotropins on behavioral and synaptic plasticity measures in aged rats.

September 1988 -- September 1994
Renewed in 1991

**Principal Investigator, National Science Foundation (NSF) R01**
*Hippocampal Plasticity Induced by Patterned Stimulation*
The goals of this work were to understand whether intrinsic components of hippocampal physiology would support lont-term potentiation and to examine the effects of stress and stress hormones on synaptic plasticity and behavior.

September 1980 -- April 1981

**Stipendium: Max-Planck-Gesellschaft**
*Electrophysiological Studies of the Hippocampus*
The goals of this work were to characterize inhibitory pathways in rat hippocampal slices.

January 1976 -- October 1976

**Stipendium: Max-Planck-Institute for Psychiatry**
*Action of Adenosine in the Hippocampal Formation*
This funding supported neuroanatomical studies of the transneuronal transport of adenosine and electrophysiological studies of its effects on hippocampal neurons.

**FELLOWSHIPS (STUDENT/POSTDOCTORAL SUPPORT)**

April 1990 -- April 1993 — **NSF Reseach Enrichment for Undergraduates Supplement**
Recipient: A.G. Humphreys

September 1991 -- September 1993 — **NIMH National Research Service Award (Postdoctoral)**
Recipient: K. E. Stevens

April 1993 -- April 1994 — **NSF Reseach Enrichment for Undergraduates Supplement**
Recipient: J.C. Lehman

October 1993 -- October 1998 — **NIMH National Research Service Award (Predoctoral M.D./Ph.D.)**

Recipient: T.M. Rampy

April 1996 -- April 1998 **NIMH Minority Research Fellowship (Postdoctoral)**
Recipient: R.G. Johnson

July 2009 – July 2010 **SIU REACH Award (Undergraduate)**
Recipient: K.P. Osborn

## EDITORIAL AND REVIEW ACTIVITIES

Associate Editor **PharmExperts.com**, 2008-2009

Field Editor **Psychobiology,** 1986 to 1996
**CNS Neuroscience & Therapeutics** (formerly **CNS Drug Reviews)** , 2001 to present

Reviewer **Behavioral Neuroscience**
**Brain Research**
**European Journal of Neuroscience**
**Hippocampus**
**Journal of Neuroscience**
**Neurobiology of Aging**
**Neuroscience**
**Proceedings of the National Academy of Sciences U.S.A.**
**Science**

Grant Reviewer National Science Foundation
National Institutes of Mental Health
National Institute on Aging
Veterans Administration
Medical Research Council of Canada
Wellcome Trust
Alzheimer's Association

Study Sections and other Review Panels **Drug Discovery in Alzheimer's Disease**
ad hoc review committee for National Institutes on Aging, July 1991
**Neurology B**: substitute member, September 1991
**Program Project Review for NIA:** February, 1994
February, April and November, 1996
March and April, 1997
February, 1998
June, 2003
**NIA SEP--Alzheimer's Disease Drug Development:** January 2008-present
**NIA Grand Opportunity Grants:** September 2009
**NIA SEP—IND Toxicology for Drugs to Treat Alzheimer's Disease and Other Aging-Related Diseases:** December 2009

## CLASSROOM TEACHING EXPERIENCE

Participation in Courses

2009 **SIUC Physiology 570q: Foundations of Memory**
Co-directed with Peter Patrylo, Ph.D.

This weekly class for graduate students and upper level undergraduates is a mixture of lectures and journal article reviews. Students spend 1 hour/week in the classroom.

1984 to 1997 — I have taught graduate, medical, dental and pharmacy students on a regular basis, giving individual 1 or 2 hour lectures or minitutorials (up to 6 lectures over a 3 week period). Subject matter included neuroanatomy, neurophysiology, neuropharmacology and substrates of behavior. I also lecture on drug abuse and on memory-related topics. Further details can be provided upon request.

Course Direction

1986 to 1990 — **IDPT 610, Pharmacology and Anatomy of the Central Nervous System**
Co-directed with Thomas E. Finger, Ph.D. This class, presented to graduate and M.D./Ph.D students, gave an extensive overview of CNS anatomy, basic pharmacology, and the localization of classical neurotransmitters and neuropeptides. Students spent 11 hours/week in lecture and laboratory activities.

## GRADUATE STUDENTS

Thesis Students

| | |
|---|---|
| Kevin C.H. Pang | **Physiological and Behavioral Actions of Norepinephrine in the Hippocampus**; Ph.D. granted in July 1986 |
| David A. Engstrom | **Cholinergic Modulation of Hippocampal Function;** Ph.D. granted in May 1991 |
| Thomas M. Rampy | **Glucocorticoid Modulation of Rat Brain Protaglandin H2 Synthase;** Ph.D. granted in July, 1996; M.D. granted in 1998 |

Laboratory Rotations

| | |
|---|---|
| Gary L. Conboy | **Determination of Age-Related Alterations in Catecholaminergic Neurotransmission Using *in vivo* Electrochemistry**, June 1984 |
| Marilyn N. Friedemann | **Presynaptic Effects of Cocaine in Rat Neostriatum: An *in vivo* Electrochemical Study**, November 1986 |
| David A. Engstrom | **Effects of Cholinergic Drugs on Long-Term Potentiation**, March 1987 |
| Margaret J. Velardo | **Cholinergic Modulation of Primed Burst Potentiation**, December 1990 |
| Thomas M. Rampy | **Effects of Corticosterone on Hippocampal Plasticity**, June 1991 |
| C. Jason Frazier | **Effects of Cholinergic Deafferentation upon Hippocampal Responsiveness to Nicotine**, June 1992 |
| Christina Bäckman | **NGF and Anti-Transferrin Receptor Antibody Conjugate: Behavioral Effects on Aged Rats**, June 1994 |

Comprehensive Examination Committees

- Michelle Mynlieff, Department of Pharmacology, September 1985
- Karen D. Parfitt, Department of Pharmacology, October 1986
- Kari S. Johnson, Department of Pharmacology, April 1987
- Craig Van Horne, Medical Scientist Training Program, (Pharmacology), August 1988
- Gina M. Nelson, Neuroscience Training Program (Cellular and Structural Biology), June 1989
- Lie-Huey Lin, Neuroscience Training Program (Pharmacology), December 1989
- John L. Hudson, Neuroscience Training Program (Pharmacology), January 1991
- Christine Miller, Neuroscience Training Program (Pharmacology), April 1991

- Ronald Klein, Department of Pharmacology, July 1991
- Karen Miller, Department of Pharmacology, June 1992
- Ronald Lickteig, Department of Pharmacology, June 1993
- Maria Curtis, Department of Cellular and Structural Biology, May 1994
- Tammy Kay Booker, Department of Pharmacology, August 1994
- Jennifer Bloomquist, Dept. of Anatomy and Neurobiology, CSU, Fort Collins, CO, December 1995

Thesis Advisory Committees

- Donald M. Burnett, Department of Pharmacology, UCHSC
- Craig Van Horne, Medical Scientist Training Program (Pharmacology), UCHSC
- John L. Hudson, Medical Scientist Training Program (Pharmacology), UCHSC
- Christine Miller, Neuroscience Training Program (Chairman), UCHSC
- Karen Miller, Department of Pharmacology, UCHSC
- Yvonne Rollins, Medical Scientist Training Program (Pharmacology; Chairman), UCHSC
- Jeremy Ebersole, Department of Physiology, SIUC
- Lauren Macklin, Department of Physiology, SIUC
- Daniel Kanak, Department of Physiology, SIUC
- Cole Vonder Haar, Department of Psychology, SIUC

Thesis Examination Committees

- Lise R. Thomas, Department of Pharmacology, UCHSC, August 1989
- Donald M. Burnett, Department of Pharmacology, UCHSC, September 1989
- Gail Perez (Masters), Department of Psychology, University of Colorado at Boulder, August 1990
- Craig Van Horne, Medical Scientist Training Program (Pharmacology), UCHSC, September 1990
- John L. Hudson, Medical Scientist Training Program (Pharmacology), UCHSC, March 1993
- Gerald H. Clayton, Neuroscience Training Program, UCHSC, October 1993
- Paul C. Chaulk (Masters), Dept. of Psychology, Memorial University of New Foundland, September 1994
- Christine Miller, Neuroscience Training Program (Chairman), June, 1995
- Karen Miller, Department of Pharmacology, May, 1996
- Yvonne Rollins, Medical Scientist Training Program (Pharmacology; Chairman), June, 1996
- Maria Curtis, Department of Cellular and Structural Biology, February, 1998
- Robert Brown, Department of Psychology, Memorial University of New Foundland, May 2004
- Andea Quigley, Department of Psychology, Southern Illinois University Carbondale, October, 2009

Major Seminars Mentored

| | |
|---|---|
| David A. Engstrom | **Involvement of Cholinergic Neurotransmission in Mammalian Memory Processes;** April 1987 |
| Marilyn N. Friedemann | **Role of the NMDA Receptors in Synaptic Plasticity;** May 1988 |
| Brandi Soldo | **Noradrenergic Modulation of Plasticity in the Visual Cortex and Hippocampus;** June 1990 |
| Thomas M. Rampy | **Beta-Amyloid, Excitotoxicity and Calbindin-D28k;** December 1992 |
| C. Jason Frazier | **NI-35 and NI-250: A Potential Mechanism for Signal Amplification *in vivo***; April 1993 |

Postdoctoral Trainees

- Kevin C.H. Pang, Ph.D. 1986 - 1988
- David M. Diamond, Ph.D. 1986 - 1990

- Karen E. Stevens, Ph.D. 1989 - 1993
- M. Catherine Bennett, Ph.D. 1987 - 1993
- Kevin B. Austin, Ph.D. 1993 - 1997
- Robert G. Johnson, Ph.D. 1994 - 1997
- Karin Coleman-Mesches, Ph.D. 1996 - 1997

## UNIVERSITY SERVICE and ADMINISTRATIVE ACTIVITIES

| | |
|---|---|
| July 2009 – present | **Member, Conflict of Interest Committee**<br>Southern Illinois University School of Medicine |
| October 1994 – October 1997 | **Member, Hospital Space Utilization Committee**<br>Veterans Affairs Medical Center, Denver, CO |
| February 1994 – October 1997 | **Member (Chairman from April, 1997) UCHSC Standing Committee on Space**<br>University of Colorado Health Sciences Center, Denver, CO |
| July 1992 -- October 1997 | **Member, Graduate Training Committee, Neuroscience Training Program**<br>University of Colorado Health Sciences Center, Denver, CO |
| June 1988 -- October 1997 | **Member (Chairman since October, 1991), Research Service Space and Resources Committee**, Veterans Affairs Medical Center, Denver, CO |
| February -- December 1994 | **Member, Search Committee for Associate Chief of Staff for Research and Development**, Veterans Affairs Medical Center, Denver, CO |
| July 1986 -- July 1994 | **Member, Neuroscience Training Program Graduate Admissions Committee** |
| June -- September 1990 | **Director, Neuroscience Summer Workshop for Minority Undergraduates**<br>University of Colorado Health Sciences Center, Denver, CO |
| October 1987 -- October 1991 | **Member, Research Service Research and Development Committee**<br>Veterans Affairs Medical Center, Denver, CO |
| March -- October 1991 | **Member, Graduate School 5-Year Planning Committee**<br>University of Colorado Health Sciences Center, Denver, CO |
| May 1986 -- July 1991 | **Member, Neuroscience Training Program Curriculum Committee**<br>University of Colorado Health Sciences Center, Denver, CO |
| May 1986 -- January 1989 | **Director for Graduate Education, Neuroscience Training Program**, University of Colorado Health Sciences Center, Denver, CO |
| April 1984 -- October 1991 | **Member, Research Service Automated Data Processing Committee**<br>Veterans Affairs Medical Center, Denver, CO |

## INVITED LECTURES

| | |
|---|---|
| September 1976 | University of Vermont, Burlington, VT |
| April 1977 | City of Hope National Medical Center, Duarte, CA |
| November 1980 | Max-Planck-Institute for Psychiatry, Munich, Germany |
| November 1980 | Nencki Institute of Experimental Biology, Warsaw, Poland |
| January 1981 | Zoological Institute, Munich University, Munich, Germany |
| February 1981 | Max-Planck-Institute for Virus Research, Tüebingen, Germany |
| June 1985 | Biological Research Laboratories, Ciba-Geigy Ltd., Basel, Switzerland |
| July 1985 | University of Düsseldorf, Düsseldorf, Germany |
| July 1985 | Max-Planck-Institute for Psychiatry, Munich, Germany |
| March 1986 | Center for the Neurobiology of Learning & Memory, U. of California, Irvine, CA |
| May 1986 | Thomas Jefferson University, Philadelphia, PA |
| September 1987 | University of Colorado, Boulder, CO |
| November 1988 | Concordia University, Montreal, Quebec, Canada |

| | |
|---|---|
| October 1989 | Trent University, Peterborough, Ontario, Canada |
| October 1989 | McGill University, Montreal, Quebec, Canada |
| February 1990 | Wellesley College, Boston, MA |
| February 1990 | Boston College, Boston, MA |
| February 1990 | Children's Hospital, Harvard University, Boston, MA |
| May 1990 | University of Virginia, Charlottesville, VA |
| August 1990 | University of California, Riverside, CA |
| September 1990 | Oberlin College, Oberlin, OH |
| April 1993 | Amgen Neuroscience Development Group, Thousand Oaks, CA |
| September 1993 | University of Colorado, Boulder, CO |
| September 1993 | Colorado State University, Fort Collins, CO |
| May 1994 | Geriatric Research, Education and Clinical Care Center (GRECC), VAMC, Seattle, WA |
| May 1994 | Carleton College, Northfield, MN |
| May 1995 | University of Florida, Gainesville, FL |
| January 1996 | NPS Pharmaceuticals, Salt Lake City, UT |
| January 1996 | University of Texas, Austin, TX |
| February 1997 | Trent University, Peterborough, Ontario, Canada |
| April 1997 | Center for the Neurobiology of Learning & Memory, U. of California, Irvine, CA |
| May 1997 | Bristol-Myers Squibb, Wallingford, CT |
| June, 1999 | Memory Pharmaceuticals, New York, NY |
| May, 2000 | Rockefeller University, New York, NY |
| May, 2001 | Nathan Kline Institute, Orange, NY |
| February, 2002 | St. Peter's College, Jersey City, NJ (Vickers Lecture) |
| August, 2004 | H. Lundbeck A/S, Copenhagen-Valby, Denmark |
| July, 2007 | Cortex Pharmaceuticals, Irvine, CA |
| September, 2007 | CHDI, Inc., Los Angeles, CA |
| November, 2007 | Merck & Co., Boston, MA |
| May, 2007 | Southern Illinois University, Carbondale, IL |
| December 2009 | Southern Illinois University, Carbondale, IL |

**INVITED LECTURES AT CONFERENCES AND SYMPOSIA**

| | |
|---|---|
| March 1981 | **Behavioral Correlates of Hippocampal Dentate Granule Cells**<br>Neural Mechanisms of Learning and Memory III, Institute of Neurology, University College London, London, England |
| September 1981 | **Physiological and Behavioral Characteristics of Dentate Granule Cells**<br>Molecular, Cellular and Behavioral Neurobiology of the Hippocampus, Schloss Ringberg am Tegernsee, West Germany |
| January 1982 | **Adenosine -- Electrophysiological, Biochemical and Anatomical Significance in the Central and Peripheral Nervous Systems**<br>Fifteenth Annual Conference on Brain Research, Steamboat Springs, CO |
| January 1987 | **Is Hippocampal Unit Activity During Learning Related to Sensory or Behavioral Events?**<br>Eleventh Annual Conference on Learning and Memory, Park City, UT |
| August 1987 | **Primed Burst Potentiation: Lasting Synaptic Plasticity Invoked by Physiologically Patterned Stimulation**<br>IBRO Satellite Meeting, Pecs, Hungary |
| September 1991 | **Animal Models of Sensory Gating**<br>Centers for Neuroscience and Schizophrenia Conference, Yale University, New Haven, CT |
| April 1992 | **Differential Effect of Aging on Hippocampal Responsiveness to Muscarine and Nicotine**<br>Third Biennial Spring Hippocampal Research Conference, Grand Cayman, British West Indies |
| April 1992 | **LTP, Learning, and Synaptic Plasticity** |

Third Biennial Spring Hippocampal Research Conference, Grand Cayman, British West Indies

November 1994 **Hippocampal Plasticity and Age-Related Memory Loss**
New Directions in the Neurobiology of Aging (sponsored by National Institute on Aging), Ft. Lauderdale, FL

November 1997 **Behavioral, Electrophysiological and Biochemical Consequences of Chronic Cytochrome Oxidase Inhibition**
Cytochrome Oxidase in energy Metabolism and Alzheimer's Disease, Society for Neuroscience, New Orleans, LA

October 2000 **Strategic Issues in the Discovery of Cognitive Enhancers**
Cognition Enhancers, Anti-Alzheimer and Neuroprotective Drugs, Yale University, New Haven, CT

October 2000 **Strategies for Characterizing and Treating Cognitive Deficits Induced by Stress and Sleep Deprivation**
DARPA Continuous Performance (24/7) Workshop, Washington, D.C.

July 2001 **Drug Discovery for Continuous Assisted Performance**
DARPA Continuous Assisted Performance Teaming Workshop, Las Vegas, NV

April 2003 **Drug Discovery for Cognitive Enhancement**
Division for Behavioral Pharmacology Symposium: Learning and Cognition—Behavioral, Pharmacological and Molecular Relationships, ASPET, San Diego, CA

February 2004 **Developing Treatments for Age-Related Memory Loss**
Co-chair (w/ Diana Woodruff-Pak) of session on Pharmacological Treatment of Age-Related Memory Loss—Myth or Reality, Winter Conference on Neuroplasticity, Castries, St. Lucia

August 2004 **Developing Treatments for Age-Related Memory Loss**
XVIIIth International Symposium on Medicinal Chemistry, Copenhagen, Denmark

February 2006 **PDE4 Inhibitors for Cognitive Enhancement**
39th annual Winter Conference on Brain Research, Steamboat Springs, CO

October 2007 **Comments From a Pharmaceutical Perspective**
Cognitive Aging Summit (sponsored by NIA and the McKnight Foundation), Washington, D.C.

October 2009 **Behavioral Models for the Development of Effective Cognitive Enhancers**
Second Alzheimer's Disease Translational Research Investigators' Meeting (sponsored by NIA), Bethesda, MD

November 2009 **Can a Mouse get Alzheimer's Disease?**
Alzheimer's Awareness Family Caregiver Conference, Springfield, IL

## PATENT APPLICATIONS

Fedorov, N. B., T.H. Michael, G.Y. Oksman, A.R. Chow and G.M. Rose. Perfusion chamber for recording evoked and spontaneous electrical activity from submerged acute brain slices. United States Patent Application No. 20050131463; June 2005.

Trippodi-Murphy, C., G.M. Rose and A. Unterbeck. Compositions and methods of treatment using L-type calcium channel blockers and cholinesterase inhibitors. United States Patent Application No. 20050153953; July 2005.

## PUBLICATIONS

Papers in Refereed Journals

1. Koopowitz, H., D. Silver and **G. Rose** (1975) Neuronal plasticity and recovery of function in a polyclad flatworm. *Nature* 256:737-738.

2. Lynch, G., C. Gall, **G. Rose** and C. Cotman (1976) Changes in the distribution of the dentate gyrus associational system following unilateral or bilateral entorhinal lesions in the adult rat. *Brain Res.* 110:57-71.
3. Koopowitz, H., D. Silver and **G. Rose** (1976) Primitive nervous systems. Control and recovery of feeding behavior in the polyclad flatworm, *Notoplana acticola*. *Biol. Bull.* 150:411-425.
4. **Rose, G.**, G. Lynch and C.W. Cotman (1976) Hypertrophy and redistribution of astrocytes in the deafferented dentate gyrus. *Brain Res. Bull.* 1:87-92.
5. Landfield, P.W., **G. Rose**, L. Sandles, T.C. Wohlstander and G. Lynch (1977) Patterns of astroglial hypertrophy and neuronal degeneration in the hippocampus of aged, memory-deficient rats. *J. Gerontol.* 32:3-12.
6. **Rose, G.** and P. Schubert (1977) Release and transfer of [$^3$H]adenosine derivatives in the cholinergic septal system. *Brain Res.* 121:353-357.
7. Schubert, P., **G. Rose**, K. Lee, G. Lynch and G.W. Kreutzberg (1977) Axonal release and transfer of nucleoside derivates in the entorhinal-hippocampal system: an autoradiographic study. *Brain Res.* 134:347-352.
8. Gerbrandt, L.K., **G. Rose**, R.L. Wheeler and G. Lynch (1978) Distribution of the perforant path following selective elimination of granule cells. *Exp. Neurol.* 62:122-132.
9. Gall, C., **G. Rose** and G. Lynch (1979) Proliferative and migratory activity of glial cells in the partially deafferented hippocampus. *J. Comp. Neurol.* 183:539-550.
10. Deadwyler, S.A., J. Biela, **G. Rose**, M. West and G. Lynch (1979) A microdrive for use with glass or metal microelectrodes in recording from freely-moving rats. *Electroenceph. Clin. Neurophysiol.* 47:752-754.
11. Erzurumlu, R.S., **G. Rose**, G.S. Lynch and H.P. Killackey (1981) Selective uptake and anterograde transport of horseradish peroxidase by hippocampal granule cells. *Neuroscience* 6:897-902.
12. Haas, H.L. and **G. Rose** (1981) Is disinhibition a component of hippocampal long-term potentiation? *J. Physiol.* 318:41P.
13. Haas, H.L. and **G. Rose** (1982) Long-term potentiation of excitatory synaptic transmission in the rat hippocampus: the role of inhibitory processes. *J. Physiol.* 329:541-552.
14. **Rose, G.**, D. Diamond and G.S. Lynch (1983) Dentate granule cells in the rat hippocampal formation have the behavioral characteristics of theta neurons. *Brain Res.* 266:29-37.
15. **Rose, G.** (1983) A temperature controller for *in vitro* recording chambers. *Brain Res. Bulliten* 10:713-714.
16. **Rose, G.**, K. Pang, M. Palmer and R. Freedman (1984) Differential effects of phencyclidine upon hippocampal complex-spike and theta neurons. *Neurosci. Lett.* 45:141-146.
17. Haas, H.L. and **G. Rose** (1984) The role of inhibitory mechanisms in hippocampal long term potentiation. *Neurosci. Lett.* 47:301-306.
18. Björklund, H., M. Eriksdotter-Nilsson, D. Dahl, **G. Rose**, B. Hoffer and L. Olson (1985) Image analysis of GFA-positive astrocytes from adolescence to senescence. *Exp. Brain Res.* 58:163-170.
19. **Rose, G.**, G. Gerhardt, I. Strömberg, L. Olson and B. Hoffer (1985) Monoamine release from dopamine-depleted rat caudate nucleus reinnervated by substantia nigra transplants: an *in vivo* electrochemical study. *Brain Res.* 341:92-100.
20. Gerhardt, G., **G. Rose**, I. Strömberg, G. Conboy, L. Olson, G. Jonsson and B. Hoffer (1985) Dopaminergic neurotoxicity of 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) in the mouse: an *in vivo* electrochemical study. *J. Pharmacol. Exp. Therap.* 235:259-265.
21. **Rose, G.** (1986) Full directory. *Profiles* 3:9-10.
22. **Rose, G.M.**, H.-R. Olpe and H.L. Haas (1986) Testing of prototype antiepileptics in hippocampal slices. *Naunyn-Schmiedeberg's Arch. Pharmacol.* 332:89-92.
23. **Rose, G.M.**, G.A. Gerhardt, G.L. Conboy and B.J. Hoffer (1986) Age-related alterations in monoamine release from rat striatum: an *in vivo* electrochemical study. *Neurobiol. Aging.* 7:77-82.
24. Gerhardt, G.A., **G. Rose** and B. Hoffer (1986) Release of monoamines from striatum of rat and mouse evoked by local application of potassium: evaluation of a new *in vivo* electrochemical technique. *J. Neurochem.* 46:842-850.
25. Adler, L.E., **G. Rose** and R. Freedman (1986) Neurophysiological studies of sensory gating in rats: effects of amphetamine, phencyclidine, and haloperidol. *Biol. Psychiatry* 21:787-798.
26. **Rose, G.M.** and T.V. Dunwiddie (1986) Induction of hippocampal long-term potentiation using physiologically patterned stimulation. *Neurosci. Lett.* 69:244-248.
27. Olson, L., E.-O. Backlund, G. Gerhardt, B. Hoffer, O. Lindvall, **G. Rose**, A. Seiger and I. Strömberg (1986) Nigral and adrenal grafts in parkinsonism: recent basic and clinical studies. *Adv. Neurol.* 45:85-94.

28. Gerhardt, G.A., K. Pang and **G.M. Rose** (1987) *In vivo* electrochemical demonstration of the presynaptic actions of PCP in rat caudate nucleus. *J. Pharmacol. Exp. Therap.* 241:714-721.
29. McGivern, R.F., **G. Rose**, C. Berka, A.N. Clancy, C.A. Sandman and B.E. Beckwith (1987) Neonatal exposure to a high level of $ACTH_{4-10}$ impairs adult learning performance. *Pharmacol. Biochem. Behav.* 27:133-142.
30. Gerhardt, G.A., **G.M. Rose** and B.J. Hoffer (1987) *In vivo* electrochemical demonstration of potassium-evoked monoamine release from rat cerebellum. *Brain Res.* 413:327-335.
31. Hoffer, B.J., G.A. Gerhardt, **G.M. Rose**, I. Strömberg and L. Olson (1987) Voltammetric analysis of nigral graft function. *Ann. N.Y. Acad. Sci.* 495:510-527.
32. Haas, H.L and **G.M. Rose** (1987) Noradrenalin blocks potassium conductance in rat dentate granule cells *in vitro*. *Neurosci. Lett.* 78:171-174.
33. Pang, K. and **G.M. Rose** (1987) Differential effects of norepinephrine on hippocampal complex-spike and θ-neurons. *Brain Res.* 425:146-158.
34. Freedman, R., L.E. Adler, G.A. Gerhardt, M. Waldo, N. Baker, **G.M. Rose**, C. Drebing, H. Nagamoto, P. Bickford-Wimer and R. Franks (1987) Neurobiological studies of sensory gating in schizophrenia. *Schizophr. Bulliten* 13:669-678.
35. Hoffer, B.J., **G. Rose**, K. Parfitt, R. Freedman and P.C. Bickford-Wimer (1988) Age-related changes in cerebellar noradrenergic function. *Ann. N.Y. Acad. Sci.* 515:269-286.
36. Bickford-Wimer, P.C., J.A. Miller, R. Freedman and **G.M. Rose** (1988) Age-related reduction in responses of rat hippocampal neurons to locally applied monoamines. *Neurobiol. Aging* 9:173-179.
37. Adler, L.E., K. Pang, G. Gerhardt and **G.M. Rose** (1988) Modulation of the gating of auditory evoked potentials by norepinephrine: pharmacological evidence obtained using a selective neurotoxin. *Biol. Psychiatry* 24:179-190.
38. Ng, Cheng Ton, J.M., G.A. Gerhardt, M. Friedemann, A.M. Etgen, **G.M. Rose,** N.S. Sharpless and E.L. Gardner (1988) The effects of $\delta^9$-tetrahydrocannabinol on potassium-evoked release of dopamine in the rat caudate nucleus: an *in vivo* electrochemical and *in vivo* microdialysis study. *Brain Res.* 451:59-68.
39. Diamond, D.M., T.V. Dunwiddie and **G.M. Rose** (1988) Characteristics of hippocampal primed burst potentiation *in vitro* and in the awake rat. *J. Neurosci.* 8:4079-4088.
40. Gerhardt, G.A., A. Gratton and **G.M. Rose** (1988) *In vivo* electrochemical studies of the effects of cocaine on dopamine nerve terminals in rat neostriatum. *Physiol. Bohemoslovaca* 37:249-257.
41. Diamond, D.M., M.C. Bennett, D.A. Engstrom, M. Fleshner and **G.M. Rose** (1989) Adrenalectomy reduces the threshold for hippocampal primed burst potentiation in the anesthetized rat. *Brain Res.* 492:356-360.
42. **Rose, G.M.** and K.C.H. Pang (1989) Differential effect of norepinephrine upon granule cells and interneurons in the dentate gyrus. *Brain Res.* 488:353-356.
43. Gerhardt, G.A., M. Friedemann, M.S. Brodie, T.W. Vickroy, A.P. Gratton, B.J. Hoffer and **G.M. Rose** (1989) The effects of cholecystokinin (CCK-8) on dopamine-containing nerve terminals in the caudate nucleus and nucleus accumbens of the anesthetized rat: an *in vivo* electrochemical study. *Brain Res.* 499:157-163.
44. Pang, K. and **G.M. Rose** (1989) Differential effects of methionine$^5$-enkephalin on hippocampal pyramidal cells and interneurons. *Neuropharmacology* 28:1175-1181.
45. Bickford-Wimer P.C., H. Nagamoto, R. Johnson, L.E. Adler, M. Egan, **G.M. Rose** and R. Freedman (1990) Auditory sensory gating in hippocampal neurons: a model system in the rat. *Biol. Psychiatry* 27:183-192.
46. Engstrom, D.A., M.C. Bennett, K.E. Stevens, R.L. Wilson, D.M. Diamond, M. Fleshner and **G.M. Rose** (1990) Modulation of hippocampal primed burst potentiation by anesthesia. *Brain Res.* 521:148-152.
47. Diamond, D.M., M.C. Bennett, K.E. Stevens, R.L. Wilson and **G.M. Rose** (1990) Exposure to a novel environment interferes with the induction of hippocampal primed burst potentiation in the behaving rat. *Psychobiology* 18:273-281.
48. Stevens, K.E., L.L. Fuller and **G.M. Rose** (1991) Dopaminergic and noradrenergic modulation of amphetamine-induced changes in auditory gating. *Brain Res.* 555:91-98.
49. Bickford-Wimer, P., K. Pang, **G.M. Rose** and G.A. Gerhardt (1991) Electrically-evoked release of norepinephrine in the rat cerebellum: an *in vivo* electrochemical and electrophysiological study. *Brain Res.* 558:305-311.

50. Bennett, M.C., D.M. Diamond, M. Fleshner and **G.M. Rose** (1991) Serum corticosterone level predicts the magnitude of hippocampal primed burst potentiation and depression in urethane-anesthetized rats. *Psychobiology* 19:301-307.
51. Bennett, M.C. and **G.M. Rose** (1992) Chronic sodium azide treatment impairs learning of the Morris water maze task. *Behav. Neural Biol.* 58:72-75.
52. Diamond, D.M., M.C. Bennett, M. Fleshner and **G.M. Rose** (1992) Inverted-U relationship between the level of peripheral corticosterone and the magnitude of hippocampal primed burst potentiation. *Hippocampus* 2:421-430.
53. Moore, C.I., M.D. Browning and **G.M Rose** (1993) Hippocampal plasticity induced by primed burst, but not long-term potentiation, stimulation is impaired in area CA1 of aged Fischer 344 rats. *Hippocampus* 3:57-66.
54. Stevens, K.E., J. Meltzer and **G.M. Rose** (1993) Disruption of sensory gating by the $\alpha 2$ selective noradrenergic antagonist yohimbine. *Biol. Psychiatry* 33:130-132.
55. Engstrom, D.A., P. Bickford, R. de la Garza, D. Young and **G.M. Rose** (1993) Increased responsiveness of hippocampal pyramidal neurons to nicotine in aged, learning-impaired rats. *Neurobiol. Aging* 14:259-266.
56. Diamond, D.M., M. Fleshner and **G.M. Rose** (1994) Stress repeatedly blocks hippocampal primed burst potentiation in behaving rats. *Behav. Brain Res.* 62:1-9.
57. Diamond, D.M. and **G.M. Rose** (1994) Does associative LTP underlie classical conditioning? *Psychobiology*, 22:263-269.
58. Freedman, R., Adler, L.E., Bickford, P., Byerley, W., Coon, H., Cullum, C.M., Griffith, J.M., Harris, J.G., Leonard, S., Miller, C., Myles-Worsley, M., Nagamoto, H., **Rose, G.** and Waldo, M. (1994) Schizophrenia and nicotinic receptors. *Harvard Rev. Psychiatry* 2:179-192.
59. Bodnoff, S. R., A. G. Humphreys, J. C. Lehman, D. M. Diamond, **G. M. Rose** and M. J. Meaney (1995) The effects of chronic corticosterone treatment on spatial learning, synaptic plasticity and hippocampal neuropathology in young and mid-aged rats. *J. Neurosci.* 15:61-69.
60. Diamond, D.M. and **G.M. Rose** (1995) Stress impairs LTP and hippocampal-dependent memory. *Ann. N.Y. Acad. Sci.* 746:411-414.
61. Stevens, K.E., J. Meltzer and **G.M Rose** (1995) Nicotinic cholinergic normalization of amphetamine-induced loss of auditory gating in freely moving rats. *Psychopharmacol.* 119:163-170.
62. Bennett, M.C., D.E. Fordyce, **G.M. Rose** and J.M. Wehner (1995) Chronic sodium azide treatment decreases bound protein kinase C (PKC) activity in the rat hippocampus. *Neurobiol. Learn. Memory* 64:187-190.
63. Miller, C.L., P.C. Bickford, A.K. Wiser and **G.M. Rose** (1995) Long-term potentiation influences auditory processing in the rat hippocampus. *J. Neurosci.* 15:5820-5830.
64. Smith, C. and **G.M. Rose** (1995) Evidence for a paradoxical sleep window for place learning in the Morris water maze, *Physiol. Behav*.59:93-97.
65. Cada, A., F. Gonzalez-Lima, **G.M. Rose** and M.C. Bennett (1995) Regional brain effects of sodium azide treatment on cytochrome oxidase activity: a quantitative histochemical study. *Metabolic Brain Disease* 10:303-320.
66. Diamond, D.M., B.J Branch, M. Fleshner and **G.M. Rose** (1995) Effects of dehydroepiandrosterone sulfate and stress on hippocampal electrophysiological plasticity. *Ann. N.Y. Acad. Sci.* 774:304-307.
67. Bennett, M.C., G. Mlady, M. Fleshner and **G.M. Rose** (1996) Synergy between chronic corticosterone and sodium azide treatments in producing a spatial learning deficit and inhibiting cytochrome oxidase activity. *Proc. Natl. Acad. Sci.* 93:1330-1334.
68. Stevens, K.E., J. Luthman, E. Lindqvist and **G.M. Rose** (1996) The effects of neonatal 6-hydroxydopamine treatment on auditory gating in the rat., *Pharmacol. Biochem. Behav.* 53:817-823.
69. Granholm, A.-C., M. Curtis, D.M. Diamond, B.J. Branch, K. Heman and **G.M. Rose** (1996) Development of an intact blood-brain barrier in brain tissue transplants is dependent on the site of transplantation. *Cell Transplantation* 5:305-314.
70. Bennett, M.C., G.W. Mlady, Y. Hwa-Lin and **G.M. Rose** (1996) Chronic sodium azide infusion induces selective and stable inhibition of cytochrome oxidase. *J. Neurochem.* 66:2606-2611.
71. Taglialatela, G., M. Gegg, J.R. Perez-Polo, L.R. Williams and **G.M. Rose** (1996) Evidence for DNA fragmentation in the CNS of aged Fischer-344 rats. *Neuroreport* 7:977-980.
72. Diamond, D.M., M. Fleshner, N. Ingersoll and **G.M. Rose** (1996) Psychological stress impairs spatial working memory: relevance to electrophysiological studies of hippocampal function. *Behav. Neurosci.* 110:661-672.

73. Stevens, K.E., R. Freedman, A.C. Collins, M. Hall, S. Leonard, M.J. Marks, D. Dill and **G.M. Rose** (1996) Inhibition of auditory evoked response among inbred mouse strains correlates with hippocampal α-bungarotoxin binding sites. *Neuropsychopharmacology* 15:152-162.
74. C.J. Frazier, Y. Rollins, M.E. Hall, D. Young and **G.M. Rose** (1996) Cholinergic deafferentation enhances rat hippocampal pyramidal neuron responsiveness to locally applied nicotine. *Brain Res.* 727:217-220.
75. Bäckman, C., **G.M. Rose**, B.J. Hoffer, M. Henry, R. Bartus, P. Friden and A-Ch. Granholm (1996) Systemic administration of a nerve growth factor conjugate reverses age-related cognitive dysfunction and prevents cholinergic neuron atrophy, *J. Neurosci.* 16:5437-5442.
76. Bickford, P.C., C.E. Adams, S.J. Boyson, P. Curella, G.A. Gerhardt, C. Heron, G.O. Ivy, A.M.L.Y. Lin, M.P. Murphy, K. Poth, D.R. Wallace, D.A. Young, N.R. Zahniser and **G.M. Rose** (1997) Long-term treatment of male F344 rats with deprenyl: assessment of effects on survival, behavior and brain function. *Neurobiol. Aging* 18:309-318.
77. K.E. Stevens, H. Nagamoto, R.G. Johnson, C.E. Adams and **G.M. Rose** (1997) Kainic acid lesions as a model of schizophrenia: changes in auditory evoked potential gating in the unanesthetized rat, *Neuroscience* 82:701-708.
78. Bäckman, C., **G.M. Rose**, R. Bartus, B.J. Hoffer, E. Mufson and A-Ch. Granholm (1997) Carrier mediated delivery of NGF: alterations in basal forebrain neurons in aged rats revealed using antibodies against low and high affinity NGF receptors. *J. Comp. Neurol.* 387:1-11.
79. K.E. Stevens, R.G. Johnson and **G.M. Rose** (1997) Rats reared in social isolation show schizophrenia-like changes in auditory gating. *Pharmacol. Biochem. Behav.* 58:1031-1036.
80. Smith, C. and **G.M. Rose** (1997) Post-training paradoxical sleep in increased after spatial learning in the Morris water maze. *Behav. Neurosci.* 6:1197-1204.
81. Austin, K.B. and **G.M. Rose** (1997) A new technology for quantifying behavioral activation in the rodent. *IEEE Transactions.*
82. Austin, K.B. and **G.M. Rose** (1997) Automated behavior recognition using continuous-wave Doppler radar and neural networks. *IEEE Transactions.*
83. Smith, C.T., J.M. Conway and **G.M. Rose** (1998) Brief paradoxical sleep deprivation impairs reference, but not working, memory in the radial arm maze task. *Neurobiol. Learning Memory* 69:211-217.
84. Johnson, R.G., K.E. Stevens and **G.M. Rose** (1998) $5HT_2$ receptors modulate auditory filtering in the rat. *J. Pharmacol. Exp. Therap.* 285: 643-650.
85. Young, D.A., A.G. Humphreys, D.A. Engstrom and **G.M. Rose** (1998) Parallel nature of hippocampal synaptic plasticity. *Synapse* 30:112-115.
86. Granholm, A.-C., D. Albeck, C. Bäckman, M. Curtis, T. Ebendal, P. Friden, M. Henry, B. Hoffer, J. Kordower, **G.M. Rose**, S. Söderström and R.T. Bartus (1998) A non-invasive system for delivering neural growth facotrs across the blood-brain barrier: a review. *Reviews in the Neurosciences* 9:31-55.
87. Moxon, K.A., P.C. Bickford, K.B. Austin, G.A. Gerhardt, **G.M. Rose**, D.J. Woodward and L.E. Adler (1999) Multiple single units and population responses during inhibitory gating of hippocampal auditory response in freely-moving rats. *Brain Res.* 825: 75-85.
88. **Rose, G.M.** (1999) Behavioral effects of neurotrophic factor supplementation in aging. *Age* 22:1-7.
89. Eid, C.N., Jr. and **G.M. Rose** (1999) Cognition enhancement strategies by ion channel modulation of neurotransmission. *Current Pharmaceutical Design* 5:345-361.
90. Mesches, M.M., M. Fleshner, K.L. Heman, **G.M. Rose** and D.M. Diamond (1999) Exposing rats to a predator blocks primed burst potentiation in the hippocampus *in vitro*. *J. Neurosci.* 19: RC18 (1-5).
91. Albeck, D.S., C. Bäckman, **G.M. Rose**, L. Veng, P. Friden and A-Ch.E. Granholm (1999) Acute application of NGF increases the firing rate of aged rat basal forebrain neurons. *Eur. J. Neurosci.* 11:2291-2304.
92. Guillou, J.-L., **G.M. Rose** and D.M.F. Cooper (1999) Differential activation of adenylyl cyclases by spatial and procedural learning. *J. Neurosci.* 19:6183-90.
93. Diamond, D.M., C.R. Park, K.L. Heman and **G.M. Rose** (1999) Exposure to a predator impairs working memory in the radial arm water maze. *Hippocampus* 9:542-552.
94. Diamond, D.M., M. Fleshner and **G.M. Rose** (1999) The enhancement of hippocampal primed burst potentiation by dehydroepiandrosterone sulfate (DHEAS) is blocked by psychological stress. *Stress* 3:107-21.

95. Hellier, J.L., P.R. Patrylo, P. Dou, M. Nett, **G.M. Rose** and F.E. Dudek (1999) Assessment of inhibition and epileptiform activity in the septal dentate gyrus of freely-behaving rats during the first week after kainate treatment. *J. Neurosci.* 19:10053-10064.
96. Smith, C. and **G.M. Rose** (2000) Evaluating the relationship between REM and memory consolidation: a need for scholarship and hypothesis testing. *Brain Behav. Sci.* 23: 1007-1008.
97. **Rose, G.M.** (2000) Strategic issues in the discovery of cognitive enhancers. *CNS Drug Reviews* 6, Suppl. 1:3.
98. Gould, T.J., W.B. Rowe, K.L. Heman, M.H. Mesches, D.A. Young, **G.M. Rose** and P.C. Bickford (2002) Effects of hippocampal lesions on patterned motor learning in the rat. *Brain Res. Bulliten* 58:581-586.
99. Tombaugh, G.C., W.B. Rowe, A. R. Chow, T.H. Michael and **G.M. Rose** (2002) Theta-frequency synaptic potentiation in CA1 *in vitro* distinguishes cognitively impaired from unimpaired aged Fischer 344 rats. *J. Neurosci.* 22:9932-9940.
100. Albeck, D.S., M.M. Mesches, S. Juthberg, M.D. Browning, P.C. Bickford, **G.M. Rose** and A.-C. Granholm (2003) Exogenous NGF restores endogenous NGF distribution in the brain of the cognitively impaired aged rat. *Brain Res.* 967:306-310.
101. Rowe, W.B., J.-P. O'Donnell, D. Pearson, **G.M. Rose**, M.J. Meaney and R. Quirion (2003) Long-term effects of BIBN-99, a selective muscarinic M2 receptor antagonist, on improving spatial memory performance in aged cognitively-impaired rats. *Behav. Brain Res.* 145:171-178.
102. Veng, L.M., A.-C. Granholm and **G.M. Rose** (2003) Age-related sex differences in spatial learning and basal forebrain cholinergic neurons in F344 rats. *Physiol. Behav.* 80:27-36.
103. Papke, R.L., J.K. Porter-Papke and **G.M. Rose** (2004) Activity of $\alpha$7-selective agonists at nicotinic and serotonin 5HT3 receptors. *Bioorg. Med. Chem. Lett.* 18:1849-1853
104. Tombaugh, G.C., W.B. Rowe and **G.M. Rose** (2005) The slow afterhyperpolarization in hippocampal CA1 neurons covaries with spatial learning ability in aged Fischer 344 rats. *J. Neurosci.* 25: 2609-2616.
105. Zhang, H.-T., Y. Huang, N.U. Suvarna, C. Deng, A.M. Crissman, A.T. Hopper, M. De Vivo, **G.M. Rose** and J.M. O'Donnell (2005) Effects of novel PDE4 inhibitors MEM1018 and MEM1091 on memory in the radial-arm maze and inhibitory avoidance tests in rats. *Psychopharmacol.* 179:613-619.
106. **Rose, G.M.**, A. Hopper, M. De Vivo and A. Tehim (2005) Phosphodiesterase inhibitors for cognitive enhancement. *Current Pharmaceutical Design* 11:3329-3334.
107. Oksman, G.Y., X. Li, **G.Rose**, V.G. Skrebitsky and N.B. Fedorov (2005) Increase in slow afterhyperpolarization led to learning delay in DBA mice. *Bull. Exp. Biol. Med.* 140:274-277.
108. Zhang, H.-T., Y. Zhao, Y. Huang, C. Deng, A.T. Hopper, M. De Vivo, **G.M. Rose** and J.M. O'Donnell (2006) Antidepressant-like effects of PDE4 inhibitors mediated by the high-affinity rolipram binding state (HARBS) of the phosphodiesterase-4 enzyme (PDE4) in rats. *Psychopharmacol.* 186:209-17.
109. Stevens, K.E., H.C. O'Neill, **G.M. Rose** and J. Luthman (2006) The 5-$HT_{1A}$ receptor active compounds (*R*)-8-OH-DPAT and (S)-UH-301 modulate auditory evoked EEG responses in rats. *Amino Acids*, 31:365-375.
110. Lindner, M.D., J.B. Hogan, R.G. Krause, F. Machet, C. Bourin, D.B. Hodges, Jr., J.A. Corsa, D.M. Barten, J.H. Toyn, D.A. Stock, **G.M. Rose** and V.K. Gribkoff (2006) Soluble A□ and cognitive function in aged F-344 rats and Tg2576 mice. *Behav. Brain Res.* 173:62-75.
111. Lindner MD, J.B. Hogan, D.B. Hodges, Jr, A.F. Orie, P. Chen, J.A. Corsa, J.E. Leet, K.W. Gillman, **G.M. Rose**, K.M. Jones and V.K. Gribkoff (2006) Donepezil primarily attenuates scopolamine-induced deficits in psychomotor function, with moderate effects on simple conditioning and attention, and small effects on working memory and spatial mapping. *Psychopharmacol.* 188:629-40.
112. **Rose, G.M.**, V. Ong and D.S. Woodruff-Pak (2007) Efficacy of MEM 1003, a novel calcium channel blocker, in delay and trace eyeblink conditioning in older rabbits. *Neurobiol. Aging* 28:766-773.
113. Rowe, W.B., E.M. Blalock, K.-C. Chen, I. Kadish, D. Wang, J.E. Barrett, O. Thibault, N.M. Porter, **G.M. Rose** and P.W. Landfield (2007) Hippocampal expression analyses reveal selective association of immediate-early, neuroenergetic, and myelinogenic pathways with cognitive impairment in aged rats. *J. Neurosci.* 27:3098-3110.

Invited Book Chapters

1. Lynch, G., **G.M. Rose**, C. Gall and C. Cotman (1975) The response of the dentate gyrus to partial deafferentation. In: *Golgi Centennial Symposium. Proceedings*, M. Santini (Ed.), Raven Press, New York, pp. 305-317.
2. Lynch, G., **G. Rose** and C. Gall (1978) Anatomical and functional aspects of the septo-hippocampal projections. In: *Functions of the Septo-Hippocampal System*, Ciba Foundation Symposium 58, Elsevier, Amsterdam, pp. 5-24.
3. **Rose, G.** (1983) Physiological and behavioral characteristics of dentate granule cells. In: *Neurobiology of the Hippocampus*, W. Seifert (Ed.), Academic Press, New York, pp. 449-472.
4. Hoffer, B., **G. Rose**, G. Gerhardt, I. Strömberg and L. Olson (1985) Demonstration of monoamine release from transplant-reinnervated caudate nucleus by *in vivo* electrochemical detection. In: *Neural Grafting in the Mammalian CNS*, A. Björklund and U. Stenevi (Eds.), Elsevier Science Publishers B.V., London, pp. 437-447.
5. **Rose, G.** and K. Pang (1985) Pharmacological differentiation of hippocampal neurons. In: *The Electrical Activity of the Archicortex*, G. Buzsaki and C.H. Vanderwolf, (Eds.), Publishing House of the Hungarian Academy of Sciences, Budapest, Hungary, pp. 191-204.
6. Jonsson, G., I. Mefford, L. Olson, I. Strömberg, E. Sundström, B. Hoffer, G. Conboy, R. Freedman, G. Gerhardt, S. Johnson and **G. Rose** (1986) Electrophysiological and neurochemical correlates of the neurotoxic effects of MPTP on central catecholamine neurons in the mouse. In: *MPTP: A Neurotoxin Producing a Parkinsonian Syndrome*, S.P. Markey, N. Castagnoli, Jr., A.J. Trevor and I.J. Kopin (Eds.), Academic Press, Orlando, Florida, pp. 275-288.
7. **Rose, G.M.**, D.M. Diamond, K. Pang and T.V. Dunwiddie (1988) Primed burst potentiation: lasting synaptic plasticity invoked by physiologically patterned stimulation. In: *Synaptic Plasticity in the Hippocampus*, H.L. Haas and G. Buzsaki (Eds.), Springer-Verlag, Berlin, pp. 96-98.
8. Gerhardt, G.A., **G.M. Rose**, A.P. Gratton, I. Strömberg, L. Olson and B.J. Hoffer (1988) *In situ* analysis of substantia nigra graft function: an electrochemical study. In: *Progress in Catecholamine Research Part B: Central Aspects*, Alan R. Liss Inc., New York, pp. 125-132.
9. Vickroy, T.W., B.R. Bianchi, J.F. Kerwin, Jr., H. Kopecka, M.S. Brodie, G.A. Gerhardt, A.P. Gratton, **G.M. Rose**, B.J. Hoffer and A.M. Nadzan (1989) Cholecystokinin-enhanced dopamine release from rat nucleus accumbens *in vitro* and *in vivo*: implications for the role of type A CCK receptors. In: *The Neuropeptide Cholecystokinin (CCK). Anatomy and Biochemistry, Receptors, Pharmacology and Physiology*, J.L. Hughes (Ed.), Ellis Horwood Ltd., New York, pp. 175-180.
10. Hoffer, B., P. Bickford-Wimer, M. Eriksdotter-Nilsson, A.-C. Granholm, M. Palmer, G. Gerhardt, L. Olson, A. Seiger and **G. Rose** (1989) Age-related changes in rat hippocampal noradrenergic transmission: insights from *in oculo* transplants. In: *Neuronal Grafting and Alzheimer's Disease*, F. Gage, A. Privat and Y. Christen (Eds.), Springer-Verlag, Berlin, pp. 130-140.
11. Diamond, D.M., M.C. Bennett, M. Fleshner and **G.M. Rose** (1991) Modulation of hippocampal primed burst potentiation by stress and corticosterone. In: *Peripheral Signaling in the Brain*, R.C.A. Fredrickson, J.L. McGaugh and D.L. Felton (Eds.), Hogrefe & Huber, Toronto, pp. 503-507.
12. **Rose, G.M.** and D.A. Engstrom (1992) Differential effect of aging on hippocampal pyramidal cell responses to muscarine and nicotine. In: *The Treatment of Dementias: A New Generation of Progress*, E.M. Meyer, J.W. Simpkins, J. Yamamoto and F.C. Crews (Eds.), Plenum Press, New York, pp. 57-73.
13. Bennett, M.C., D.M. Diamond, W.D. Parker, S.L. Stryker and **G.M. Rose** (1992) Inhibition of cytochrome oxidase impairs learning and hippocampal plasticity: a novel animal model of Alzheimer's disease. In: *Treatment of Dementias: A New Generation of Progress,* E.M. Meyer, J.W. Simpkins, J. Yamamoto and F.T. Crews (Eds.), Plenum Press, New York, pp. 485-501.
14. Meaney, M.J., S.R. Bodnoff, D. O'Donnell, W. Rowe, A. Sarrieau. **G.M. Rose**, J. Poirer and J.R. Seckl (1993) Glucocorticoids as regulators of neuron survival and repair in the aged brain. In: *Restorative Neurology*, *Vol. 6*, A.C. Cuello (Ed.), Elsevier, Amsterdam, pp. 267-289.
15. Freedman, R., S. Leonard, L. Adler, P. Bickford, W. Byerley, H. Coon, C. Miller, V. Luntz-Leybman, M. Myles-Worsley, H. Nagamoto, **G. Rose**, K. Stevens and M. Waldo (1995) Nicotinic receptors and the pathophysiology of schizophrenia. In: *Advances in Pharmacological Sciences: Effects of Nicotine on Biological Systems II*, P.B.S. Clark, M. Quik, F. Adlkofer and K. Thurau (Eds.), Birkhauser-Verlag, Basel, pp. 307-312.
16. **Rose, G.M.**, K.L. Heman, G.W. Pypali and D.A. Turner (1998) Electrophysiological assessment of the aged hippocampus in vivo and in vitro. In: *Methods in Aging Research*, B.P. Yu (Ed.), New York, CRC Press, pp. 116-143.

17. Bennett, M.C. and **G.M. Rose** (1998) Behavioral, electrophysiological and biochemical consequences of chronic cytochrome oxidase inhibition in rats. In: *Cytochrome Oxidase in Neuronal Metabolism and Alzheimer's Disease*, F. Gonzalez-Lima (Ed.), New York, Plenum Press, pp.217-232.
18. **Rose, G.M.** and D.M. Diamond (2001) What studies in old rats tell us about the role of LTP in learning and memory. In: *Neuronal Mechanisms of Memory Formation*, C. Hölscher (Ed.), London, Cambridge University Press, pp. 346-361.
19. Diamond, D.M., C.R. Park, M.J. Puls and **G.M. Rose** (2001) Differential effects of stress on hippocampal and amygdaloid LTP: insights into the neurobiology of traumatic memories. In: *Neuronal Mechanisms of Memory Formation*, C. Hölscher (Ed.), London, Cambridge University Press, pp. 379-403.
20. **Rose, G.M.** (2003) Cognitive enhancers. In: *Learning and Memory*, J.H. Byrne (Ed.), New York, Macmillan, pp. 84-86.
21. **Rose, G.M.** (2003) Drugs and memory. In: *Learning and Memory*, J.H. Byrne (Ed.), New York, Macmillan, pp. 118-120.
22. **Rose, G.M.** (2003) Pharmacological treatment of memory deficits. In: *Learning and Memory*, J.H. Byrne (Ed.), New York, Macmillan, pp. 520-523.

**ABSTRACTS**

1. Deadwyler, S.A., G. Lynch, E.J. Stanford, **G. Rose** and C.W. Cotman (1974) *In vitro* studies of the deafferented hippocampus. Plasticity I. *Fourth Annual Meeting of the Society for Neuroscience*, San Diego, California.
2. Schubert, P., **G. Rose**, K. Lee and G. Lynch (1976) Axonal release of nucleoside derivatives and transfer to postsynaptic neurons. *J Cell Biol* 70:299a.
3. Gall, C., R.K. Jones, **G. Rose** and G. Lynch (1977) Glial proliferative response in the deafferented dentate gyrus. *Anat Rec* 187:583.
4. Lynch, G. and **G. Rose** (1977) Anatomical and functional aspects of the septo-hippocampal projections. *Ciba Foundation Symposium*, London, England.
5. **Rose, G.**. D. Diamond and G. Lynch (1981) Physiological and behavioral characteristics of dentate granule cells. *Soc Neurosci Abstr* 7:845.
6. Hoffer, B., S. Wood, W. Freed, R. Wyatt and **G. Rose** (1983) Morphology of substantia nigra neurons transplanted into the lateral ventricle of the rat. *Soc Neurosci Abstr* 9:849.
7. **Rose, G**., K. Pang and T. Dunwiddie (1983) Effects of systemic morphine upon hippocampal electrophysiology in the rat. *Soc Neurosci Abstr* 9:1129.
8. Hoffer, B.J., G. Gerhardt, **G. Rose**, M.R. Palmer, A. Sieger, R.N. Adams and L. Olson (1984) Transmission in catecholamine cell grafts *in oculo* and *in situ*: demonstration by *in vivo* electrochemical detection. *Symposium on Transplantation in the Mammalian CNS*, Lund, Sweden.
9. Eriksdotter-Nilsson, M., H. Björklund, D. Dahl, **G. Rose** and L. Olson (1984) Computer image analysis of rat GFA-positive astrocytes from adolescence to senescence. *Soc Neurosci Abstr* 10:428.
10. Gerhardt, G., **G. Rose**, I. Strömberg, L. Olson and B. Hoffer (1984) Demonstration of monoamine release from transplant-reinnervated caudate nucleus by *in vivo* electrochemical detection. *Soc Neurosci Abstr* 10:182.
11. Johnson, S., G. Gerhardt, **G. Rose**, G. Conboy, G. Jonsson, L. Olson and B. Hoffer (1984) A rodent model of parkinsonism: dopamine denervation of the mouse striatum following administration of NMPTP. *Soc Neurosci Abstr* 10:413.
12. Pang, K. and **G. Rose** (1984) Differential effects of met-enkephalin on hippocampal CA1 complex-spike and theta cells. *Soc Neurosci Abstr* 10:1109.
13. **Rose, G.**, K. Pang and R. Freedman (1984) Pharmacological differentiation of dentate granule cells and hippocampal theta neurons. *Soc Neurosci Abstr* 10:951.
14. Adler, L.E. and **G. Rose** (1985) Neurophysiologic studies of sensory gating in rats: effects of amphetamine, phencyclidine and haloperidol. *Society of Biological Psychiatry*, Annual Meeting.
15. Adler, L.E., **G.M. Rose** and R. Freedman (1985) Sensory gating in rats: parallels with psychosis. *American Psychiatric Association*, 138th Annual Meeting.

16. Gerhardt, G. and **G. Rose** (1985) Presynaptic action of phencyclidine (PCP) in the rat striatum defined using *in vivo* electrochemical methods. *Soc Neurosci Abstr* 11:1205.
17. Pang, K., A. Chavez and **G. Rose** (1985) Effects of central depletion of norepinephrine or serotonin upon memory tasks using the radial maze. *Soc Neurosci Abstr* 11:534.
18. **Rose, G**., G. Gerhardt, G. Conboy and B. Hoffer (1985) Age-related alterations in monoamine release from rat striatum: an *in vivo* electrochemical study. *Soc Neurosci Abstr* 11:729.
19. Mahalik, T.J., **G. Rose**, G. Gerhardt, I. Strömberg, L. Olson, B. Hoffer and T. Finger (1985) Characterization of fetal substantial nigra grafts to catecholamine depleted striatum: immunocytochemistry and *in vivo* electrochemistry. *International Symposium on Neural Regeneration*, sponsored by Veterans Administration/Paralysed Veterans of America.
20. Hoffer, B.J., G.A. Gerhardt, **G.M. Rose**, I. Strömberg and L. Olson (1986) Voltammetric analysis of nigral graft function. *New York Academy of Sciences.*
21. Mahalik, T.J., **G.M. Rose** and T.E. Finger (1986) Neurons in damaged and aged rat brain contain serum proteins: a light and electron microscopic study. *Seventh Annual Neurosymposium*, *Rocky Mountain Region Neuroscience Group.*
22. **Rose, G.M.**, P.C. Bickford and R. Freedman (1986) Diminished effects of norepinephrine and serotonin upon hippocampal pyramidal neurons recorded from senescent Fischer 344 rats. *J Am Aging Assoc* 9:113-114.
23. Waldo, M.C., L. Adler, N. Baker, **G. Rose**, R. Franks, K. Pang, C. Drebing, G. Gerhardt, P. Bickford and R. Freedman (1986) Noradrenergic and dopaminergic modulation of auditory evoked potentials in schizophrenia and mania. *Psychophysiology Society*, 14th Annual Meeting.
24. Adler, L.E., **G.M. Rose**, K. Pang and R. Freedman (1986) Neurophysiological studies of sensory gating in rats: effects of amphetamine, haloperidol and DSP4. *Soc Neurosci Abstr* 12:1145.
25. Bickford, P.C., **G.M. Rose**, A. Seiger and R. Freedman (1986) Diminished effects of norepinephrine and serotonin upon hippocampal pyramidal neurons recorded from senescent Fischer 344 rats. *Soc Neurosci Abstr* 12:274.
26. Gerhardt, G.A., **G.M. Rose** and B.J. Hoffer (1986) Potassium-evoked monoamine release from rat cerebellum: measurement by *in vivo* electrochemistry. *Soc Neurosci Abstr* 12:136.
27. Pang, K. and **G.M. Rose** (1986) Differential effects of norepinephrine on hippocampal neurons. *Soc Neurosci Abstr* 12:1391.
28. **Rose, G.M.** and T.V. Dunwiddie (1986) Induction of hippocampal long-term potentiation using physiologically patterned stimulation. *Soc Neurosci Abstr* 12:1377.
29. Freedman, R., L. Adler, N. Baker, **G. Rose**, M. Waldo, R. Franks, K. Pang, C. Drebing, G. Gerhardt and P. Bickford (1986) Noradrenergic and dopaminergic modulation of auditory evoked potentials in schizophrenia and mania. *American College of Neuropsychopharmacology*, 1986 Annual Meeting.
30. Freedman, R., L.E. Adler, M. Waldo, **G. Rose**, N. Baker and H. Nagamoto (1987) Auditory evoked potential studies of schizophrenia. *American Psychiatric Association*, 140th Annual Meeting.
31. Bickford-Wimer, P.C., J. Miller, R. Freedman and **G.M. Rose** (1987) Age-related reduction in responses of rat hippocampal neurons to locally applied monoamines. *Boulder Gerontology Interest Group Conference.*
32. Adler, L.E., G. Gerhardt, K. Pang and **G.M. Rose** (1987) Modulation of auditory gating by norepinephrine. *Society of Biological Psychiatry*, Annual Meeting, p.214.
33. Diamond, D. and **G.M. Rose** (1987) Induction of hippocampal long term potentiation in the awake rat using physiologically patterned stimulation. *Eighth Annual Neurosymposium, Rocky Mountain Region Neuroscience Group.*
34. Gerhardt, G.A. and **G.M. Rose** (1987) *In vivo* electrochemical studies of catecholamine reuptake in rat neostriatum. *IBRO Satellite Meeting: Neurone and Glia Microenvironment*, Prague, Czechoslovakia.
35. **Rose, G.M**., D.M. Diamond, K. Pang and T.V. Dunwiddie (1987) Primed burst potentiation: lasting synaptic plasticity invoked by physiologically patterned stimulation. *IBRO Satellite Meeting: Synaptic Plasticity in the Hippocampus,* Pecs, Hungary, p. 22.
36. Diamond, D.M. and **G.M. Rose** (1987) Induction of hippocampal long term potentiation in the awake rat using physiologically patterned stimulation. *Soc Neurosci Abstr* 13:975.
37. Pang, K., P. Bickford-Wimer, **G.M. Rose** and G.A. Gerhardt (1987) Electrically-evoked release of norepinephrine in the rat cerebellum: an *in vivo* electrochemical study. *Soc Neurosci Abstr* 13:909.
38. Wilson, R.L., K. Pang and **G.M. Rose** (1987) Characteristics of crossed and multisynaptic pathways from the entorhinal cortex to the contralateral dentate gyrus in unanesthetized rats. *Soc Neurosci Abstr* 13:975.

39. Diamond, D.M., M.C. Bennett and **G.M. Rose** (1988) Novelty stress inhibits the induction of PB potentiation in the hippocampus of awake rats. *Soc Neurosci Abstr* 14:477.
40. Gerhardt, G.A., M.S. Brodie, A.P. Gratton, T.W. Vickroy, **G.M. Rose** and B.J. Hoffer (1988) *In vivo* electrochemical studies of the effects of cholecystokinin on evoked overflow of dopamine (DA) in the caudate nucleus and nucleus accumbens of the anesthetized rat. *Soc Neurosci Abstr* 14:145.
41. Nagamoto, H.T., P.C. Bickford-Wimer, L.E. Adler, M. Egan, R.G. Johnson, R. Freedman and **G.M. Rose** (1988) Neurophysiological studies of sensory gating in rat hippocampus: effects of amphetamine and haloperidol. *Soc Neurosci Abstr* 14:967.
42. Vickroy, T.W., B.R. Bianchi, J.F. Kerwin, H. Kopecka, M.S. Brodie, G.A. Gerhardt, A.P. Gratton, **G.M. Rose**, B.J. Hoffer and A.M. Nadzan (1988) Mechanistic and pharmacological characterization of cholecystokinin-enhanced [$^3$H]dopamine efflux from rat nucleus accumbens *in vitro*: results with novel cholecystokinin antagonists indicate a role for peripheral-type receptors. *Cambridge Meeting on CCK.*
43. Freedman, R., L.E. Adler, P. Bickford-Wimer, C. Drebing, G. Gerhardt, A.-C. Granholm, B.J. Hoffer, H. Nagamoto, L. Olson, **G.M. Rose** and M. Waldo (1989) Candidate neuronal mechanisms for abnormal sensory gating in schizophrenia. *Schizophr Res* 2:82.
44. Diamond, D.M., M.C. Bennett, M. Fleshner and **G.M. Rose** (1989) Behavioral and hormonal modulation of primed burst potentiation. *Sixth Annual International Symposium on Hormonal Control of Peripheral Signaling in the Brain*, University of California, Irvine, October 16-20.
45. Bennett, M.C., D.M. Diamond, M. Fleshner and **G.M. Rose** (1989) Modulation of hippocampal primed burst potentiation by adrenalectomy and corticosterone. *Soc Neurosci Abstr* 15:1102.
46. Engstrom, D.A. and **G.M. Rose** (1989) Influence of cholinergic neurotransmission on hippocampal synaptic plasticity. *Soc Neurosci Abstr* 15:85.
47. Luthman, J., M.N. Friedemann, P. Bickford-Wimer, **G. Rose**, L. Olson, B.J. Hoffer and G.A. Gerhardt (1989) *In vivo* electrochemical and electrophysiological studies of the caudate nucleus of neonatal 6-OHDA-lesioned rats. *Soc Neurosci Abstr* 15:687.
48. Diamond, D.M., M. Fleshner and **G.M. Rose** (1990) Environmental manipulations can repeatedly influence hippocampal PB potentiation in the behaving rat. *Soc Neurosci Abstr* 16:652.
49. Engstrom, D.A. and **G.M. Rose** (1990) Nicotine differentially affects the spontaneous firing of hippocampal pyramidal cells and interneurons. *Soc Neurosci Abstr* 16:207.
50. Nagamoto, H.T., K.E. Stevens, L.L. Fuller, S. Bernal, R. Johnson and **G.M. Rose** (1990) Effects of intraventricular kainic acid on sensory gating of the rat N40 auditory evoked potential. *Soc Neurosci Abstr* 16:1351.
51. **Rose, G.M.** and A. Gratton (1990) Eating behavior is accompanied by dopamine release in rat medial striatum. *Soc Neurosci Abstr* 16:437.
52. Stevens, K.E., L.L. Fuller and **G.M. Rose** (1990) Dopamine D1, but not D2, receptor blockade reversed amphetamine-induced changes in auditory gating. *Soc Neurosci Abstr* 16:1351.
53. Gerhardt, C. Drebing, R. Johnson, M. Johnson, L. Hoffer, E.M. Cawthra, A.C. Madison and R. Freedman (1991) Ten years of studies on P50 sensory gating: a review and considerations for future work. *Schizophr Res*
54. Bennett, M.C., D.M. Diamond, W.D. Parker, S.L. Stryker and **G.M. Rose** (1991) Inhibition of cytochrome oxidase impairs learning and hippocampal plasticity: a novel animal model of Alzheimer's disease. *Second Suncoast Workshop on the Neurobiology of Aging.*
55. **Rose, G.M.** and D.A. Engstrom (1991) Differential changes in the sensitivity of hippocampal CA1 pyramidal neurons to locally applied muscarine and nicotine in aged, learning-impaired rats. *Second Suncoast Workshop on the Neurobiology of Aging.*
56. **Rose, G.M.** and D.A. Engstrom (1991) Hippocampal nicotinic cholinergic supersensitivity in aged, learning impaired rats. *IBRO World Congress of Neuroscience Abstracts* 3:102.
57. Stevens, K.E., J. Meltzer and **G.M. Rose** (1991) Nicotine modulation of amphetamine-induced changes in gating of auditory evoked potentials in the rat. *IBRO World Congress of Neuroscience Abstracts* 3:209.
58. Bennett, M.C. and **G.M. Rose** (1991) Cytochrome oxidase inhibition by sodium azide infusion impairs learning in the Morris water maze. *Age* 14:142-143.
59. **Rose, G.M.**, D.A. Engstrom, A.G. Humphreys and P.C. Bickford-Wimer (1991) Differential effect of aging upon hippocampal pyramidal cell responsiveness to muscarine and nicotine. *Age* 14:137.

60. Bennett, M.C. and **G.M. Rose** (1991) Cytochrome oxidase inhibition by sodium azide infusion impairs learning in the Morris water maze. *Soc Neurosci Abstr* 17:697.
61. Diamond, D.M., M.C. Bennett, J. Meltzer, M. Fleshner and **G.M. Rose** (1991) Inverted-U relationship between corticosterone and hippocampal primed burst potentiation in urethane-treated rats. *Soc Neurosci Abstr* 17:387.
62. Engstrom, D.A. and **G.M. Rose** (1991) The effect of locally applied cholinergic drugs on primed burst potentiation. *Soc Neurosci Abstr* 17:387.
63. Moore, C.I., **G.M. Rose** and M.D. Browning (1991) Long term potentiation induced by primed burst potentiation is reduced in hippocampal CA1 of aged, learning impaired rats. *Soc Neurosci Abstr* 17:1329**.**
64. **Rose, G.M.** and A. Humphreys (1991) Septal lesions impair hippocampal primed burst potentiation and LTP. *Soc Neurosci Abstr* 17:387.
65. Stevens, K.E., J. Meltzer, S.L. Stryker and **G.M. Rose** (1991) Alpha$_2$ adrenergic blockade with yohimbine disrupts auditory sensory gating in rats. *Soc Neurosci Abstr* 17:1454.
66. Luthman, J., K. Stevens, E. Lindqvist and **G. Rose** (1992) Neonatal 6-hydroxydopamine lesions in rats as a dopamine-dependent schizophrenia model. *Behav Pharmacol* 3(Suppl. 1):69.
67. Bodnoff, S.R., A. Humphreys, **G.M. Rose**, and M.J. Meaney (1992) Effects of long-term corticosterone treatment on spatial learning, synaptic plasticity and neuron loss in mid-aged rats. *Proceedings of the XXIII Congress of the International Society of Psychoneuroendocrinology*, p. 129.
68. Diamond, D., B. Branch, C. Bennett, M. Fleshner and **G.M. Rose** (1992) Dexamethasone mimics the inhibitory effects of elevated levels of corticosterone on hippocampal primed burst potentiation. *Proceedings of the XXIII Congress of the International Society of Psychoneuroendocrinology*, p. 140.
69. Bennett, M.C., D. Fordyce, D.M. Diamond, **G.M. Rose** and J. Wehner (1992) Chronic cytochrome oxidase inhibition by sodium azide infusion alters hippocampal protein kinase C activity. *Age* 15:131.
70. Bickford, P.C., C. Heron, **G.M. Rose**, A. M.-Y. Lin, M. Friedemann and G.A. Gerhardt (1992) Effects of long-term deprenyl administration on behavior and noradrenergic neurotransmission in aged rats. *Age* 15:131.
71. Diamond, D., B. Branch, M. Fleshner and **G. Rose** (1992) Pharmacology manipulations of the inverted-U relationship between hippocampal primed burst potentiation and corticosterone. *Fifth Conference on the Neurobiology of Learning and Memory*, Irvine, CA., 5:20.
72. **Rose, G.M.**, A.K. Wiser and C. Smith (1992) A paradoxical sleep window for place learning in the Morris water maze. *Fifth Conference on the Neurobiology of Learning and Memory*, Irvine, CA., 5:53.
73. Bennett, M.C., L. Waltrip, D.M. Diamond, **G.M. Rose** and J. Wehner (1992) Chronic cytochrome oxidase inhibition by sodium azide infusion alters hippocampal protein kinase C activity. *Soc Neurosci Abstr* 18:1245.
74. Bickford, P.C., C. Heron, **G.M. Rose**, N. Miniclier, K. Poth, A.M.-Y. Lin, M. Friedemann and G.A. Gerhardt (1992) The effect of long term deprenyl administration on behavior and brain function in aged rats. *Soc Neurosci Abstr* 18:904.
75. Bodnoff, S.R., A. Humphreys, **G.M. Rose** and M.J. Meaney (1992) Effects of corticosterone and stress on spatial learning, hippocampal plasticity and neuron loss. *Soc Neurosci Abstr* 18:534.
76. Diamond, D.M., B. Branch, M.C. Bennett, M. Fleshner and **G.M. Rose** (1992) Dexamethasone blocks the induction of hippocampal primed burst potentiation. *Soc Neurosci Abstr* 18:1348.
77. Moore, C.I., B.A. Duff, E.M. Dudek, T.V. Dunwiddie, **G.M. Rose** and M. Browning (1992) A multi-electrode array capable of delivering tetanic stimulation to an entire hippocampal CA1 mini-slice. *Soc Neurosci Abstr* 18:1499.
78. **Rose, G.M.** and C. Smith (1992) A paradoxical sleep window for place learning in the Morris water maze. *Soc Neurosci Abstr* 18:1226.
79. Stevens, K.E., K.R. Harris, J.C. Lehman, S.L. Stryker and **G.M. Rose** (1992) Dopamine autoreceptor antagonist AJ76 disrupts auditory sensory gating in rats. *Soc Neurosci Abstr* 18:1514.
80. Velardo, M.J., **G.M. Rose**, E.S. Deneris, M.J. Marks and S.W. Rogers (1992) Glutamate receptor expression changes in rat hippocampus after entorhinal cortex lesion. *Soc Neurosci Abstr* 18:90.
81. Wiser, A.K. and **G.M. Rose** (1992) Electrochemical detection of monoamines in the hippocampus. *Soc Neurosci Abstr* 18:325.
82. **Rose, G.M.** (1993) The case for hippocampal nicotinic cholinergic receptor involvement in age-related memory dysfunction. *Fourteenth Annual Neurosymposium*, *Rocky Mountain Region Neuroscience Group.*
83. Reite, M., N. Berton, J. Delmore, J. Stocker, P. Teale, J. Wang and **G. Rose** (1993) Auditory evoked potentials and fields in macaque monkey. *Electroenceph Clin Neurophysiol* 86(5):75P.

84. Bennett, M.C., J.C. Lehman, G.W. Mlady and **G.M. Rose** (1993) Synergy between corticosterone and sodium azide in producing a place learning deficit in the Morris water maze. *Soc Neurosci Abstr* 19:188.
85. Branch, B.J., **G.M. Rose**, K.R. Harris and D.M. Diamond (1993) A method for simultaneously recording the CA1 population spike and dendritic EPSP in the intact hippocampus. *Soc Neurosci Abstr* 19:909.
86. Diamond, D.M. and **G.M. Rose** (1993) Psychological stress interferes with working, but not reference, spatial memory. *Soc Neurosci Abstr* 19:366.
87. Eckles, K.E., J.C. Lehman, **G.M. Rose** and M.D. Browning (1993) The deficit in isoproterenol stimulation of synapsin phosphorylation in aged animals is not due to an increase in the basal phosphorylation state of synapsin. *Soc Neurosci Abstr* 19:388.
88. Frazier, C.J. and **G.M. Rose** (1993) The effect of cholinergic deafferentation on hippocampal neuron responsiveness to nicotine. *Soc Neurosci Abstr* 19:1132.
89. Harris, K.R., K.E. Stevens, J.C. Lehman and **G.M. Rose** (1993) Spontaneously non-gating rats: a model of the auditory gating deficit in schizophrenia. *Soc Neurosci Abstr* 19:837.
90. Miller, C.L., **G.M. Rose**, A.K. Wiser, R. Freedman and P. Bickford (1993) Long-term potentiation reduces auditory gating in the CA3 region of the hippocampus. *Soc Neurosci Abstr* 19:909.
91. Rampy, T.M., F.A. Fitzpatrick and **G.M. Rose** (1993) Chronic dexamethasone treatment alters arachidonic acid-stimulated production of *ex vivo* prostaglandin $E_2$ production in selected brain regions. *Soc Neurosci Abstr* 19:1189.
92. Rollins, Y.D., K.E. Stevens, K.R. Harris, M.E. Hall, **G.M. Rose** and S. Leonard (1993) Reduction in auditory gating following intracerebroventricular application of $\alpha$-bungarotoxin binding site ligands and $\alpha$7 antisense oligonucleotides. *Soc Neurosci Abstr* 19:837.
93. **Rose, G.M.**, B.J. Branch, A.G. Humphreys, C.I. Moore and D.M. Diamond (1993) The population spike is a valid measure of hippocampal long-term potentiation. *Soc Neurosci Abstr* 19:909.
94. Stevens, K.E., R. Freedman, M. Hall, S. Leonard and **G.M. Rose** (1993) Hippocampal CA3 $\alpha$-bungarotoxin binding is correlated with auditory gating ability in inbred mouse strains. *Soc Neurosci Abstr* 19:465.
95. Wiser, A.K. and **G.M. Rose** (1993) Primed burst potentiation in the dentate gyrus. *Soc Neurosci Abstr* 19:909.
96. **Rose, G.M.** (1993) Searching for the causes of, and treatments for, the learning and memory problems of old age. *Symposium: The Future of VA Mental Health Research, Department of Veterans Affairs and National Foundation for Brain Research,* November 8-9, 1993.
97. Stevens, K.E., R. Freedman and **G.M. Rose** (1993) Mouse strains with differing numbers of hippocampal $\alpha$-bungarotoxin binding sites display varying ability to gate to paired auditory stimuli. *Schizophr Res*, 9:168.
98. Stevens, K.E., K.R. Harris, J.C. Lehman and **G.M. Rose** (1993) Nicotinic cholinergic receptor modulation of sensory gating in rats. *Int Behav Neurosci Soc (IBNS) Abstr* 2:38.
99. Smith, C., J. Conway and **G. Rose** (1993) Evidence for a N-methyl-D-aspartate dependent window for memory of a spatial learning task. *Sleep Res* 22:345
100. Smith, C., J. Conway and **G. Rose** (1993) Evidence of a paradoxical sleep window for spatial memory. *Sleep Res* 22:346.
101. Bickford, P., K. Hershman, **G. Rose**, J. Lehman, P. Friden and T. Ebendal (1994) Effects of chronic alcohol on learning, memory and cholinergic systems in aged F344 rats. *Research Society on Alcoholism.*
102. Bickford, P., K. Hershman, T. Fay, **G. Rose**, J. Lehman, T. Ebendal and P. Friden (1994) Effects of chronic alcohol on cholinergic systems in aged F344 rats. *The Seventh Congress of the International Society for Biomedical Research on Alcoholism.*
103. Rollins, Y.D., K.E. Stevens, K.L. Heman, K.R. Harris, M.E. Hall, **G.M. Rose** and S. Leonard (1994) Reduction in auditory gating with alpha-bungarotoxin binding site antagonists and $\alpha$7 antisense oligonucleotides. *Neuropsychopharmacol.* 10:268S.
104. Stevens, K.E., K.R. Harris, K.L. Heman and **G.M. Rose** (1994) Dopamine D2 antagonist AJ76: low dose disrupts auditory gating, high dose normalizes amphetamine-induced loss of gating. *Int Behav Neurosci Soc (IBNS) Abstr*, 3:87.
105. Bennett, M.C. and **G.M. Rose** (1994) Behavioral, electrophysiological and biochemical effects of chronic cytochrome oxidase inhibition in the rat. *First International Conference on Oxidative Stress and Aging Abstracts,* p. 50.
106. Bennett, M.C., G.W. Mlady, J.C. Lehman and **G.M. Rose** (1994) Alzheimer-like pathology produced by sodium azide-induced cytochrome oxidase inhibition: potentiation by glucocorticoid. *Fourth International Conference on Alzheimer's Disease and Related Disorders, Neurobiol. Aging* 15:S15.

107. Stevens, K.E., R. Freedman, M.J. Marks, A.C. Collins, S. Leonard, M. Hall, and **G.M. Rose** (1994) Inbred mouse strains with differing numbers of hippocampal alpha-bungarotoxin binding sites show correlated ability to gate evoked responses to paired auditory stimuli. *International Symposium, Cholinergic Synapse: Structure, Function and Regulation* P8.14.
108. Johnson, B.W., **G. Rose**, P. Teale and M. Reite (1994) Suppression and recovery of auditory evoked potentials with reversible lesions of the medial geniculate body. *Soc Neurosci Abstr* 20:321.
109. Cada, A., F. Gonzalez-Lima, **G.M. Rose** and M.C. Bennett (1994) Regional cerebral effects of sodium azide treatment on cytochrome oxidase activity: a quantitative histochemical study. *Soc Neurosci Abstr* 20:574.
110. Rollins, Y.D., M.Hall, K.L. Heman, P.Dobelis, J.P. Walrond, **G.M. Rose** and S. Leonard (1994) Effect of methyllycaconitine and related alkaloids, in vitro and in vivo, at the α-bungarotoxin binding site in rodent brain. *Soc Neurosci Abstr* 20:1133.
111. Diamond, D.M., B.J. Branch, **G.M. Rose**, and G.Tocco (1994) Stress effects on memory and AMPA receptors are abolished by adrenalectomy. *Soc Neurosci Abstr* 20:1215.
112. Patrylo, P.R., **G.M. Rose**, K.L Heman and F.E. Dudek (1994) Altered electrical activity in the dentate gyrus of freely-behaving rats after kainate-induced status epilepticus. *Soc Neurosci Abstr* 20:1455.
113. Patrylo, P.R., **G.M. Rose**, K.L Heman and F.E. Dudek (1994) Electrical activity in the dentate gyrus of freely-behaving rats during and after kainate-induced status epilepticus. *Epilepsia* 35 (Suppl 8): 39.
114. Rollins, Y.D., K.E. Stevens, K. Heman, **G.M. Rose** and S. Leonard (1995) α7 nicotinic receptor antagonists in the awake rat simulate the auditory gating deficit found in schizophrenia. *Schiz Res* 15:183
115. Granholm, A.-C, C. Bäckman, M. Curtis and **G.M. Rose** (1995) Development of a blood-brain barrier in intracranial and intraocular septal forebrain transplants. *Amer. Soc. For Neural Transplantation.*
116. Stevens, K.E., J. Luthman and **G.M. Rose** (1995) (-)UH-301, a 5-HT1A receptor antagonist, normalizes amphetamine-induced loss of auditory gating in freely moving rats. *Schiz Res* 15:186.
117. Bäckman, C., A.-C. Granholm, R. Bartus, M. Henry, B. Hoffer and **G.M. Rose** (1995) Anti-transferrin receptor antibody-NGF conjugate improves spatial memory and ameliorates cholinergic neuron atrophy in aged rats. *Soc Neurosci Abstr* 21:548.
118. Johnson, R.G., K.E. Stevens and **G.M. Rose** (1995) Modulation of auditory gating by 5HT-2 receptors. *Soc Neurosci Abstr* 21:745.
119. Frazier, C.J., **G.M. Rose** and T.V. Dunwiddie (1995) Direct and disinhibitory effects of nicotine in the hippocampal slice. *Soc Neurosci Abstr* 21:1569.
120. Diamond, D.M., B.J Branch, M. Fleshner and **G.M. Rose** (1995) Enhancement of hippocampal primed burst potentiation by dehydroepiandrosterone sulfate (DHEAS). *Soc Neurosci Abstr* 21:1686.
121. **Rose, G.M.**, D.E. Fordyce and J.M. Wehner (1996) Hippocampal protein kinase C activtion correlates with spatial learning in aged male Fischer 344 rats. *International Behavioral Neuroscience Society* 5:53.
122. Johnson, R.G., K.E. Stevens, L.M. Veng, R.G. Wiley and **G.M. Rose** (1996) Selective loss of medial septal cholinergic neurons does not affect brain surface or hippocampal auditory gating in unanesthetized rats. *International Behavioral Neuroscience Society* 5:58.
123. Stevens, K.E., R.G. Johnson and **G.M. Rose** (1996) Rats reared in social isolation show schizophrenia-like alterations in auditory gating. *International Behavioral Neuroscience Society* 5:69.
124. Mesches, M.H., C.J. Frazier, **G.M. Rose,** and D.M. Diamond (1996) Effects of DHEAS on hippocampal electrophysiology *in vitro*. *Soc Neurosci Abstr* 22:141.
125. Bloomquist, J.L., P.R. Patrylo, P.Dou, **G.M. Rose** and F.E. Dudek (1996) Partial recovery of perforant-path-evoked hypterexcitability in the dentate gyrus of freely-behaving rats during the first week after kainate treatment. *Soc Neurosci Abstr* 22:187.
126. Frazier, C.J., W.R. Proctor, **G.M. Rose** and T.V. Dunwiddie (1996) Identification of a novel current mediated by α7 nicotinic receptors in rat hippocampal stratum radiatum interneurons, but not in CA1 pyramidal cells. *Soc Neurosci Abstr* 22:334.
127. Stevens, K.E., R.G. Johnson, J. Luthman and **G.M. Rose** (1996) Equivalent effects of stimulation of blockade of 5-HT1A receptors upon amphetamine-induced loss of auditory gating. *Soc Neurosci Abstr* 22:608.
128. Johnson, R.G., K.E. Stevens and **G.M. Rose** (1996) DOI, a 5-HT2 agonist, normalizes auditory gating in isolation-reared rats. *Soc Neurosci Abstr* 22:608.

129. Albeck, D., **G.M. Rose**, L. Veng, R. Bartus and A.-C. Granholm (1996) Electrophysiological effects of OX-26NGF on medial septal neurons. *Soc Neurosci Abstr* 22:752.
130. **Rose, G.M.**, K.L. Heman and L.R. Williams (1996) Chronic nerve growth factor administration enhances hippocampal primed burst potentiation in aged, but not young, male Fischer 344 rats. *Soc Neurosci Abstr* 22:752.
131. Flach, K.A., R. Johnson, **G.M. Rose**, G.A. Gerhardt, L.E. Adler and P. Bickford (1996) Correlation between single-unit activity and the auditory evoked potential response to paired click stimuli in the rat. *Soc Neurosci Abstr* 22:900.
132. Rollins, Y.D., C. Breese, C. Adams, C. Drebing, **G.M. Rose** and S. Leonard (1996) Cellular localization of $\alpha$-bungarotoxin binding and $\alpha$7 mRNA in the hippocampus related to auditory gating in the awake behaving rat. *Soc Neurosci Abstr* 22:1272.
133. Love, J., R. Puch, D. Tremblay, J.W. Rudy, **G. Rose**, D. Diamond, R.S. Mazzeo, L.R. Watkins and M. Fleshner (1996) Dehydroepiandrosterone (DHEA) and dehydroepiandrosterone sulfate (DHEA-S) in the rat: converging evidence of anti-glucocorticoid properties. *Soc Neurosci Abstr* 22:2061.
134. Patrylo, P.R., J.L. Bloomquist, P. Dou, **G.M. Rose** and F.E. Dudek (1996) Spontaneous prolonged filed-potentials in the dentate gyrus of freely-behaving rats 1 week after kainate treatment. *Epilepsia* 37:66.
135. **Rose, G.M.**, K.L. Heman and L.R. Williams (1997) Chronic administration of nerve growth factor enhances primed burst potentiation, but not LTP in aged male Fischer 344 rats. *International Behavioral Neuroscience Society* 6:31.
136. Johnson, R.G., K.E. Stevens and **G.M. Rose** (1997) $5HT_{1A}$ and $5HT_2$ receptor modulation of auditory gating in unanesthetized rats. *International Behavioral Neuroscience Society* 6:46.
137. Rowe, W.B., **G.M. Rose**, K Kitaichi, J. Richard, H. Doods, M.J. Meaney and R. Quirion (1997) Long-term effects of BIBN-99, a muscarinic M2 antagonist, in reversing cognitive deficits in aged memory-impaired rats. *Soc Neurosci Abstr* 23:214.
138. L.M. Veng, A-Ch. Granholm, D.M. Diamond and **G.M. Rose** (1997) Age and gender differences in size of p75-NGF receptor positive neurons in the medial septum of Fischer 344 rats. *Soc Neurosci Abstr* 23:348.
139. Johnson, R.G., K.E. Stevens, R.G. Wiley and **G.M. Rose** (1997) Cholinergic deafferentation does not affect brain surface or hippocampal auditory gating. *Soc Neurosci Abstr* 23:668.
140. Austin, K.B. and **G.M. Rose** (1997) Is auditory gating a form of habituation? *Soc Neurosci Abstr* 23:1583.
141. Rowe, W., S. Sharma, J. Yau, J.R. Seckl, R. Quirion, **G.M. Rose**, and M.J. Meaney (1998) Hypothalamic-pituitary-adrenal axis hyperactivity in mid-aged rats predicts cognitive deficits in later life which are attuated by chronic antidepressant treatment. *Soc Neurosci Abstr* 24:1376.
142. Mesches, M.H., K.L Heman, M. Fleshner, **G.M. Rose** and D.M. Diamond (1998) Inescapable exposure to a predator blocks PB potentiation, but not LTP, *in vitro*. *Soc Neurosci Abstr* 24:1420.
143. Austin, K.B. and **G.M. Rose** (1998) Parametric modeling of brain responses to paired stimuli. *Soc Neurosci Abstr* 24:1878.
144. Dworetzky, S.J., J.T. Trojnacki, M. Goldstein, C. Boissard, C.D. Weaver, **G. Rose** and V.K. Gribkoff (1998) Cloning and expression of mouse KCNQ2: a nervous-system specific voltage-gated potassium channel. *Soc Neurosci Abstr* 24:2032.
145. Hellier, J.L, P.R. Patrylo, P. Deu, M. Nett, **G.M. Rose** and F.E. Dudek (1999) Assessment of hippocampal inhibition and epileptiform activity in freely-behaving rats during the first week after kainate treatment. *Soc Neurosci Abstr* 25:1111.
146. Rose, W.B. and **G.M. Rose** (1999) Differential learning in Tg2576 mice: role of sex, phenotype and task complexity. *Soc Neurosci Abstr* 25:54.
147. **Rose, G.M.**, K.L. Heman, D.M. Barten and W.B. Rowe (1999) Deficits in Y-maze alternation and contextual fear conditioning in aged male Tg2576 mice. *Soc Neurosci Abstr* 25:55.
148. Hellier, J.L., P.R. Patrylo, P. Dou, **G.M. Rose**, K.J. Staley and F.E. Dudek (2000) Analysis of interictal-to-ictal transition in the dentate gyrus of freely behaving rats with kainate-induced epilepsy. *Soc Neurosci Abstr* 26:225.
149. Bickford, P.C., T.J. Gould, A. Pollock, C.A. Rabiner, KL. Heman, W.B. Rowe and **G.M. Rose** (2000) Hippocampal lesions in F344 rats disrupt retention but not acquisition of a motor learning task. *Soc Neurosci Abstr* 26:467.
150. Tombaugh, G.C., W.B. Rowe, A. Chow and **G.M. Rose** (2000) LTP induced in CA1 by theta-frequency stimulation in vitro distinguishes learning-impaired from unimpaired aged rats. *Soc Neurosci Abstr* 26:530.

151. Brucato, F.H., W.B. Rowe and **G.M. Rose** (2000) Identification of age-related deficits in novel object recognition memory in rats. *Soc Neurosci Abstr* 26:531.
152. Trippodi, C., R.M. Gordon and **G.M Rose** (2000) Characterization of novel object recognition in young and aged C57BL/6 mice. *Soc Neurosci Abstr* 26:531.
153. **Rose, G.M.** (2000) Strategic issues in the discovery of cognitive enhancers. *CNS Drug Rev* 6, Suppl 1:3.
154. Kogan, J.H. and **G.M. Rose** (2001) Trace fearing conditioning deficits in aged mice. *Soc Neurosci Abstr* 27:86.13.
155. Trippodi, C. and **G.M. Rose** (2002) Evaluation of MEM 1414 in novel object recognition memory. Program No. 80.4. *2002 Abstract Viewer/Itinerary Planner*. Washington, DC: Society for Neuroscience, Online.
156. Tombaugh, G.C., W.B. Rowe and **G.M. Rose** (2002) The slow after-hyperpolarization (sAHP) in CA1 neurons correlates with spatial learning ability in aged F344 rats. Program No. 80.5. *2002 Abstract Viewer/Itinerary Planner*. Washington, DC: Society for Neuroscience, Online.
157. Zhang, H.-T., Y. Huang, N.U. Suvarna, A.M. Crissman, A.T. Hopper, M. De Vivo, **G.M. Rose** and J.M. O'Donnell (2002) Effects of novel PDE4 inhibitors MEM 1018 and MEM 1091 in models of memory and antidepressant sensitivity. Program No. 684.12. *2002 Abstract Viewer/Itinerary Planner*. Washington, DC: Society for Neuroscience, Online.
158. Trippodi, C. and **G.M. Rose** (2003) Object recognition and object location memory in young and aged Sprague Dawley rats. Program No. 625.9. *2003 Abstract Viewer/Itinerary Planner*. Washington, DC: Society for Neuroscience, Online.
159. Callahan, P.M., K.L. McNicholas, C. Ilch, W.B. Rowe, F.H. Brucato, J.H. Kogan and **G.M. Rose** (2003) Characterization of nicotinic α7 receptor agonists in animal models of cognition. Program No. 625.8. *2003 Abstract Viewer/Itinerary Planner*. Washington, DC: Society for Neuroscience, Online.
160. Brucato, F.H., W.B. Rowe, C.P. Ilch and **G.M. Rose** (2003) Context fear and Morris water maze performance identifies similar populations of memory impaired or unimpaired aged F344 rats. Program No. 625.7. *2003 Abstract Viewer/Itinerary Planner*. Washington, DC: Society for Neuroscience, Online.
161. Rowe, W.B., C. Ilch, K. Solomon and **G.M. Rose** (2003) Enduring effect of the nicotinic α7 agonist, AR-R-17779, on spatial learning ability in aged F344 rats. Program No. 625.6. *2003 Abstract Viewer/Itinerary Planner*. Washington, DC: Society for Neuroscience, Online.
162. Tombaugh, G.C., W.B. Rowe, and **G.M. Rose** (2003) Calcium spikes are enhanced selectively in aged F344 rats with performance deficits in the Morris water maze. Program No. 625.5. *2003 Abstract Viewer/Itinerary Planner*. Washington, DC: Society for Neuroscience, Online.
163. Brucato, F.H., J.H. Kogan, K.L. McNicholas, N.B. Fedorov, **G.M. Rose**, M. Klitenick, D. Liu, P. Poon, and D. Brunner (2004) Pre-training lesions of the dorsal hippocampus impair both spatial and non-spatial memory in mice. Program No. 550.11. *2004 Abstract Viewer/Itinerary Planner*. San Diego, CA: Society for Neuroscience, Online.
164. Ilnicka M.J., G. Shyy, J. Allard, Z. Zhang, L. Markovtsova, G.A. Peltz, T.J. Novak, C. Trippodi, D. Wang, A.R. Chow, **G.M. Rose**, B. Bugaj-Gaweda, M. De Vivo, and C.S. Freedland (2004) Transcriptional changes associated with novel object recognition training. Program No. 769.3. *2004 Abstract Viewer/Itinerary Planner*. Washington, DC: Society for Neuroscience, Online.
165. Trippodi, C. and **G.M. Rose** (2004) Temporal order discrimination in young and aged Sprague-Dawley rats. Program No. 898.3. *2004 Abstract Viewer/Itinerary Planner*. Washington, DC: Society for Neuroscience, Online.
166. Hogan, J.B., D.B. Hodges, A.F. Orie, J.A. Corsa, K.W. Gillman, J.E. Starrett, **G.M. Rose**, V.K. Gribkoff, J.E. Leet and M.D. Lindner (2005) Donepezil attenuates scopolamine-induced deficits in fear conditioning and psychomotor speed but not working memory or spatial mapping. Program No. 651.9. *2005 Abstract Viewer/Itinerary Planner*. Washington, DC: Society for Neuroscience, Online.
167. Heman, K.L., **G.M. Rose** and S. Lelas. (2005) Effects of combination of haloperidol and 5-HT receptor subtype-selective antagonists on conditioned avoidance responding and catalepsy in rats. Program No. 914.6. *2005 Abstract Viewer/Itinerary Planner*. Washington, DC: Society for Neuroscience, Online.
168. Blalock, E.M., W. B. Rowe, K. Chen, I. Kadish, D. Wang, J. E. Barrett, N. M. Porter, **G. M. Rose** and P. W. Landfield (2006) Alterations in mylelinogenic and plasticity pathways discriminate between cognitively impaired and unimpaired aged animals. Program No. 169.6. *2006 Abstract Viewer/Itinerary Planner*. Atlanta, GA: Society for Neuroscience, Online.

169. Trippodi-Murphy, C., **G. M. Rose** and D. F. Fiorino (2006) The effect of MEM 1414 on visual, spatial, and temporal order memory in aged Sprague-Dawley rats. Program No. 273.18. *2006 Abstract Viewer/Itinerary Planner*. Atlanta, GA: Society for Neuroscience, Online.

170. Kanak, D.J., A. L. Willingham, **G. M. Rose** and P. R. Patrylo (2009) Aging-dependent alterations in network excitability of hippocampal region CA3 during 5-Hz stimulation of the mossy fibers in hippocampal slices from aged F344 rats. Program No. 331.22. *2009 Abstract Viewer/Itinerary Planner*. Chicago, IL: Society for Neuroscience, Online.

171. Macklin, L.N., Y. Cai, D. J. Kanak, J. Ebersole, A. E. Corbin, A. Womack, D. Wendt, S. Oddo, F. M. Laferla, X.-X. Yan, G. J. Brewer, R. Struble, **G. M. Rose**, A. D. Strader and P. R. Patrylo (2009) Impaired glucose tolerance precedes neuroanatomical identification of A and hyperphosphorylated tau accumulation in the 3xTg model of Alzheimer's disease. Program No. 528.26. *2009 Abstract Viewer/Itinerary Planner*. Chicago, IL: Society for Neuroscience, Online.

172. Ebersole, J.M., D. J. Kanak, A. E. Corbin, T. Eid, G. M. Rose and P. R. Patrylo (2009) 13C isotopic incorporation studies reveal alteration in glutamate-glutamine cycling the the hippocampus of aged Fischer 344 rats. Program No. 535.10. *2009 Abstract Viewer/Itinerary Planner*. Chicago, IL: Society for Neuroscience, Online.

SIU School of Medicine

August 7, 2017

To whom it may concern,

I am a neuroscientist with a Ph.D. in Psychology obtained in 1980 from the University of California at Irvine. I currently hold the rank of professor in the Departments of Anatomy and Physiology at the Southern Illinois University School of Medicine and was, until recently, Director of the Center for Integrative Research in Cognitive and Neural Sciences at Southern Illinois University. My group is examining the effects of the Arrowsmith program on academic, neuropsychological and neuroimaging measures. The basic study design compares the progress of students with complex learning disabilities who are enrolled at the Brehm Preparatory School in Carbondale, Illinois, and who are receiving either the standard Brehm curriculum or the Brehm curriculum supplemented with the Arrowsmith program. Baseline neuropsychological and neuroimaging measures are also being obtained from age-matched typically developing students recruited from the local community.

At present, the effects of Arrowsmith are being evaluated over a period of approximately one year. Preliminary results indicate that students in the Arrowsmith program show significant improvement in some measures of executive function [notably, the Delis-Kaplan Executive Function System (D-KEFS) Tower task] and working memory. Imaging work indicates a significant improvement in resting state connectivity within the default mode network, particularly the connections between medial prefrontal areas and the precuneus/posterior cingulate cortex and hippocampus/ parahippocampal regions. The relationship of the changes in neuropsychological and imaging measures to academic performance are being evaluated using Measures of Academic Performance (MAP) and Woodcock-Johnson (WJ III) assessments.

Definitive results from this very involved study will likely take two more years before a sufficient sample size is reached and the analysis in complete. A more focused study that examines the relationship between the Arrowsmith Symbol Relations task with alterations in resting state network function is currently underway using participants in the Arrowsmith summer Cognitive Intensive Program in Toronto, Canada. In this study, progress in mastering the Symbol Relations task over a 6-week period will be correlated with potential alterations in resting state neuroimaging measures. This study provides an opportunity to directly examine whether a key element in the Arrowsmith program results in measurable alterations in brain functioning thought to underlie broader improvements in academic progress.

Please do not hesitate to contact me if you would like further information or clarification of any of my comments.

Sincerely,

Gregory M. Rose, Ph.D.
Professor, Departments of Anatomy & Physiology

Department of Anatomy
Southern Illinois University School of Medicine | Life Science III 2086 - Mail Code 6523
1135 Lincoln Drive | Carbondale, Illinois 62901
(618) 453-1563 | Fax: (618) 453-1527 | www.siumed.edu/anatomy