ACSfN
2018 Annual Meeting
Little Rock, AR October 18th, 2018
Arkansas Chapter of the Society for Neuroscience

ANNUAL MEETING PROGRAM

Thursday, October 18, 2018

University of Arkansas for Medical Sciences
Little Rock, Arkansas

Business Meeting and Presentations
Jo Ellen Ford Auditorium, Room 1207
Donald W. Reynolds Institute on Aging

and

Poster Session
Foyer outside the Jo Ellen Ford Auditorium
Donald W. Reynolds Institute on Aging

Schedule

1:00 – 1:15 p.m.  Setup Posters
1:00 – 1:30 p.m.  Lunch Available
1:30 – 1:45 p.m.  Welcome and Opening Remarks, Elvis Cuevas, Ph.D.
1:45 – 2:45: p.m.  Keynote Seminar

"Photobiomodulation of the brain"
Analiz Rodriguez, M.D., Ph.D.
Neurosurgeon and assistant Professor, UAMS
College of Medicine, Department of Neurosurgery
Little Rock, Arkansas

2:45 – 2:50 p.m.  Brain Bee, BAW, Andrew James, PhD
2:50 – 3:00 p.m.  Coffee break/Networking
3:00 – 4:00 p.m.  Poster Session and Awards Committee meets with
Undergraduate/Graduate Student/Postdoctoral Fellow Presenters to
Evaluate Posters

4:00 – 4:30 p.m.  Business Meeting
1) Officers' Reports - Treasurer and Secretary
2) Election of Officers
3) Awards for Best Posters
4) Old Business
5) New Business

4:30 – 5:00 p.m.  Removal of Posters from Poster Boards
Officers of the Arkansas Chapter of the Society for Neuroscience 2018

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The wmN1 Enhancer Region is Required for expression of Human PLP1
Pankaj Patyal, Hamdan Hamdan, Nermian Kockara, Patricia Wight
University of Arkansas for Medical Sciences, Physiology and Biophysics, Little Rock, USA

The myelin proteolipid protein gene (PLP1) encodes the most abundant protein present in myelin from the central nervous system (CNS). Its expression must be tightly controlled as evidenced by mutations that alter PLP1 dosage; both overexpression (elevated PLP1 copy number) and lack thereof (PLP1 deletion) result in X-linked genetic disorders in man. However, not much is known about the mechanisms that govern expression of the human gene. To address this, transgenic mice were generated which utilize human PLP1 (hPLP1) sequences [proximal 6.2 kb of 5'-flanking DNA to the first 38 bp of exon 2] to drive expression of a lacZ reporter cassette. LoxP sites were incorporated around a 1.5 kb section of hPLP1 intron 1 since it contains sequence orthologous to the wmN1 region from mouse which, previously, was shown to augment expression of a minimally promoted transgene coincident with the active myelination period of CNS development. Frt sites were also incorporated in hPLP1 intron 1 in order to remove most of the (8,579 bp) intron. Eight transgenic lines were generated with the parental, 6.2hPLP(+)/Z/FL, transgene. All lines expressed the transgene appropriately in brain as evidenced by staining with X-gal in white matter regions and olfactory bulb. Immunostaining against cell type specific markers revealed that the 6.2hPLP(+)/Z/FL transgene is expressed in oligodendrocytes and oligodendrocyte precursor cells (OPCs), as well as in olfactory ensheathing cells (OECs) and neurons. Removal of the ‘wmN1’ region from 6.2hPLP(+)/Z/FL with a ubiquitously expressed Cre-driver caused a dramatic reduction in β-gal activity, throughout development, in the resulting 6.2hPLPAwmN1 subline. These results demonstrate for the first time that the wmN1 enhancer region: (i) is functional in hPLP1; (ii) works in collaboration with its native promoter, not just a basal heterologous promoter; (iii) is required for high levels of hPLP1 gene activity; (iv) has a broader effect, both spatially and temporally, than originally projected with mouse Plp1 (mPlp1).
Validation of a Spine Biomechanics Simulator
John T. Sherrill, MPH & Erin M. Mannen, PhD
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BACKGROUND & SIGNIFICANCE: The biomechanics of the spine are often studied with the use of a programmable mechanical testing machine to apply a continuous load to the specimen and an optical motion-capture system to measure vertebral motion. The Spine Biomechanics Simulator (SBS) in this experiment is a MTS servohydraulic test system capable of 6 degrees of freedom and application of pure moments. The Optotrak motion capture system has an accuracy of ±0.1mm or, under the conditions of this project, ±0.05°. The SBS is also capable of recording its own motion data. Quantifying the error in the displacement measured by the SBS will allow the machine to be used independently of the optical motion capture system and with greater confidence.1

GOAL: The goal of this project to compare the motion data collected by the biomechanics simulator against the accepted value collected by the motion-capture system.

HYPOTHESIS: The displacement data collected by the Spine Biomechanics Simulator will vary no more than 0.5° from the gold-standard motion capture data.

METHODS: The specimen used for testing is a synthetic functional spinal unit modeled in the likeness of L3 and L4 vertebrae (Sawbones, Pacific Research Labs, Vashon Island, WA, USA). The specimen is intended for biomechanical testing and has been shown to imitate human cadaver specimens.2-4 The automated test machine applied continuous load to the specimen as a constant rate to a maximum load. Axial rotation (AR), flexion/extension (FE), and lateral bending (LB) occurred at 1°/s to a maximum load of ±7.5Nm. Compression was applied at 0.5mm/s to a maximum of 600N and a minimum of 100N. The motion-capture system consisted of three non-collinear markers on optical motion-capture rigid body whose motion was tracked by three cameras (Optotrak Certus, Northern Digital Inc., Waterloo, ON, Canada).

RESULTS: The total ROM in FE showed only a difference between the collection systems (MTS=3.736°, OPT=3.726°). The root mean square error was found to be 0.247°. The max error was 0.384°. The total ROM in LB showed some difference between the collection systems (MTS = 6.343°, OPT = 5.564°). The root mean square error was found to be 0.363°. The max error was 0.629°. The total ROM in AR showed a small difference between the collection systems (MTS = 3.035°, OPT = 2.783°). The root mean square error was found to be 0.105°. The max error was 0.269°.

CONCLUSIONS & IMPLICATIONS: Translating the position data of the rigid body marker to the centroid of the gimbals may introduce some error. The method of attaching rigid body markers to the gimbals allowed for a small amount of motion which would allow error due to the vibrations of the machine. In cases where only global bending angle is of concern, the MTS system data is an acceptable approximation of the true ROM for FE and AR. More work needs to be done to reduce the error in LB to <0.5°. This will reduce data collection and analysis time. The custom made SBS can be used confidently in displacement control.


Figure 1: Load-displacement curve of a flexion/extension cycle.
Traumatic brain injury induces cell death and alters IGF signaling in human primary dopaminergic neuronal precursor cells

Division of Neurotoxicology, National Center for Toxicological Research, Jefferson, AR, USA. Traumatic brain injury (TBI) is caused when an external mechanical force induces damage to the brain, resulting in deformation throughout the brain tissue. Although one of the consequences of TBI is neuronal cell death, different molecular mechanisms are triggered as response to the initial trauma, including insulin-like growth factor-1 (IGF-1) signaling. Therefore, the aim of this study was to evaluate if neuronal levels of IGF-1 and related proteins are modified after TBI in vitro. Human dopaminergic neurons were submitted to mild (10% stretch) or severe (50% stretch) TBI in vitro using a commercially available system, subsequent analyses were performed 1 and 7 days after injury. LDH release increased only at 50% stretch 1 day post-injury, which values returned to control levels after 7 days. 50% stretch decreased while 10% stretch increased IGF-1 levels 1 day post injury; however, the levels of this hormone were decreased at both conditions after 7 days. IGF-2 decreased 1 day after injury, while increased after 7 days under both conditions. IGFBPs remained practically unchanged, except for IGFBP-2, which levels increased after 10% and 50% stretch both 1 and 7 days post injury. Insulin levels decreased 1 day post injury at both conditions, whereas 10% decreased and 50% increased its levels 7 days after injury. These data suggest that IGF signaling is altered by both mild and severe TBI and may trigger different mechanisms inside the neuron independent of cell death.
RNA-Seq analysis of spinal cord tissues from hPFN1\textsuperscript{G118V} transgenic mouse model of ALS.

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Amyotrophic lateral sclerosis (ALS) is a fatal neurodegenerative disease that leads to the loss of motor neurons. The molecular mechanisms of motor neuron degeneration are largely unknown and there are currently no effective therapies to treat this disease. We have conducted a whole transcriptome profiling using RNA-Seq technology and sequencing cDNA from the total RNA isolated from the spinal cords of mutant transgenic hPFN1\textsuperscript{G118V} mice and their wildtype transgenic hPFN1\textsuperscript{WT} controls. The aim of this study was to identify molecular changes in spinal cords of the hPFN1\textsuperscript{G118V} ALS mouse model at presymptomatic and end stages. To our knowledge, this is the first study to use next-generation RNA-Seq to measure differential gene expression in hPFN1\textsuperscript{G118V} mice at presymptomatic and end stages. The analysis of genes identified in this study revealed that end-stage hPFN1\textsuperscript{G118V} mice had 890 deferentially expressed genes (747 upregulated, 143 downregulated) when compared to presymptomatic hPFN1\textsuperscript{G118V} mice, and they had 836 deferentially expressed genes (742 upregulated, 94 downregulated) when compared to age-matched hPFN1\textsuperscript{WT} controls. Presymptomatic hPFN1\textsuperscript{G118V} mice were not significantly different from age-matched hPFN1\textsuperscript{WT} controls. We further analyzed the genes and their corresponding proteins form the database using ingenuity pathway analysis (IPA) and identified inflammatory pathways significantly activated in end-stage hPFN1\textsuperscript{G118V} samples, suggesting an excess of glial activation at end-stage disease. We have also analyzed the data for gene expression for cell type specificity (astrocytes, microglia, oligodendrocytes, neurons and motor neurons) and validated the RNA-Seq data by qRT-PCR using randomly selected 12 candidate genes. We will present our RNA-Seq data and graphical representations showing valuable and promising leads into the identification of molecules and pathways revealing the mechanism of neurodegeneration that could potentially serve as therapeutic targets for ALS.


Acknowledgments:
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Evaluation of the effects of sevoflurane on developing animal brain

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Our previous studies demonstrated that an 8-hour sevoflurane exposure at clinically-relevant
concentrations caused neuronal cell death in the developing nonhuman primate (NHP) central
nervous system. To understand the mechanisms by which sevoflurane exposure causes adverse
effects on the brain, microRNA (miRNA) profiling was performed on total RNAs extracted from
the frontal cortex, a susceptible brain area, using next-generation sequencing. 452 miRNAs
were identified in the frontal cortex. Eleven miRNAs were differentially expressed after
sevoflurane exposure, 6 of which were expressed at significantly lower levels than controls, and
the other 5 miRNAs were expressed significantly higher than controls. The differentially
expressed miRNAs (DEMs) were then loaded to the Ingenuity Pathway Analysis ( IPA) database
for pathway analysis. The IPA database identified 9 DEMs that target 5,049 mRNAs which
influence multiple cellular functions. Of note, the target genes were involved in neural networks,
inducing mitochondrial dysfunction and endoplasmic reticulum (ER) stress, causing apoptosis
and disturbance in fatty acid metabolism, etc. Among the identified networks, some confirmed
our previous observations and others extended our understanding of the effects sevoflurane on
the developing monkey brain. Moreover, the IPA database suggested sevoflurane exposure
induced autophagy in the brain, which is one of the mechanisms that ensure proper function of
neural cells and maintain cellular homeostasis. Using immunoblotting, miRNA-targeted
autophagy-related gene expression was verified at the translational level, providing evidence on
the regulatory roles of miRNA on gene expression, and highlighting the diverse effects of
sevoflurane exposure on the developing NHP brain.

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Neuroprotective effect of the acetyl-L-carnitine (ALC) in a chronic MPTP-induced Parkinson's disease mouse model

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Parkinson's disease (PD) is a progressive motor disease of unknown etiology. The clinical features of PD emerge due to selective degeneration of dopaminergic (DA) neurons in the substantia nigra pars compacta (SNC), which project to the caudate putamen (CPu) where they release DA. In the current study, we investigated whether acetyl-L-carnitine (ALC) could ameliorate the pathology seen in an \textit{in vivo} chronic 1-methyl-4-phenyl-1, 2, 3, 6-tetrahydropyridine (MPTP)-induced mouse model of PD. Four treatment groups were included: 1) CONTROL receiving probenecid (PROB; 250mg/kg) only, 2) MPTP \textsuperscript{P} [MPTP (25mg/kg) + PROB], 3) MPTP \textsuperscript{P} + ALC (100mg/kg), and 4) ALC alone. MPTP-induced losses in tyrosine hydroxylase (TH), a marker for DA neurons, and DA transporter (DAT) immunoreactivity in the ventral midbrain SNC and CPu were significantly reduced with ALC treatment. HPLC data further suggests that decreases in CPu dopamine produced by MPTP exposure were also attenuated by ALC treatment. In addition, microglial activation and astrocytic reactivity induced by MPTP were greatly reduced by ALC treatment, indicating protection against neuroinflammation. Similarly, glucose transporter-1 (Glut-1) that is expressed exclusively in brain endothelial cells and the tight junction proteins occludin (OCL) and zonula occludens-1 (ZO-1) are also protected from MPTP-induced down-regulation by ALC treatment. Together, the data from the present study suggest that in this \textit{in vivo} PD model, ALC treatment protects against MPTP-induced damage to endothelial cells and loss of DA neurons in the SNC and CPu, suggesting that ALC therapy may have the potential to slow or ameliorate the progression of PD pathology in a clinical setting.
Downregulation of 14-3-3 protein levels in an animal model of neurodegeneration and in human Alzheimer's brains
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The 14-3-3 proteins are among the most abundant proteins expressed in the brain, comprising about 1% of its total soluble proteins. They bind to specific phospho-serine and phosphothreonine-containing motifs found on a variety of signaling proteins (kinases and transcription factors, among others) to regulate a wide array of cellular processes including cell cycling, apoptosis, and autophagy. We have examined the expression of 14-3-3 and its different isoforms in frontal cortex of kainic acid-treated and control rats, as well as in frontal cortex of postmortem Alzheimer’s disease patients and age-matched control subjects. Among the different 14-3-3 isoforms in control rats, the relative abundance of expression is in the following order: 14-3-3-eta > tau > sigma > epsilon > gamma > alpha/beta > zeta/delta, whereas in human control samples the sequence of relative abundance is 14-3-3-eta > tau > sigma > epsilon > zeta/delta > alpha/beta, suggesting a conservative expression pattern of the seven 14-3-3 isoforms in mammalian forebrain. Twenty-four hours following a kainic acid injection (10 mg/kg i.p.), there was a significant decrease in the total protein levels of 14-3-3 as well as the isoforms eta, tau, epsilon and gamma, when compared to those in control animals. In Alzheimer’s disease samples, there was a significant decrease in total 14-3-3 levels and the eta and gamma isoforms and no difference in 14-3-3-tau levels between Alzheimer’s disease and control brains. Together, these results demonstrate an abundance of several 14-3-3 isoforms in the frontal cortex and that a downregulation of total 14-3-3 protein levels and specific 14-3-3 isoforms is associated with neurodegeneration. Given the known function of 14-3-3 proteins as inhibitors of apoptosis, the observed decreased levels of 14-3-3 proteins could be early molecular events leading to increased activities in cell death signaling and eventually neurodegeneration. Based on these results and available evidence from other neurodegenerative diseases and animal models, 14-3-3 proteins may serve as an early biomarker of neurodegeneration and a potential target of therapeutic intervention for neurodegeneration.
Supported by FDA/NCTR Protocols E0747701 and E0763101.
Evaluation of olfactory pathway neuropathology in a transgenic rat model of Alzheimer's disease
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Alzheimer’s disease (AD) is one of the most debilitating neurological diseases leading to impairments in cognitive, sensory and motor functions. Olfactory dysfunction in humans has been recognized as a potential biomarker for the early detection of AD. Olfaction is a process which initiates from a sensory neuron input to the olfactory bulb (OB), that is decoded in the piriform cortex followed by downstream stimulation of neurons in the hippocampus. An exploration of olfactory pathway alterations at different stages of disease progression, characterized by amyloid plaques (AP) deposition in the rat transgenic (Tg) AD model, holds the potential to describe a spatial-temporal pattern of neurodegeneration, related to early olfactory loss. Therefore, the aim of the present study was to characterize the neuropathology and protein alteration in the OB of AD Tg rats at different stages of disease progression (4-20 months). Histochemical staining with a styrylbenzene derivative, fluoro-styrylbenzene (FSB) was used to detect both AP and neurofibrillary tangles (NT). Fluoro-Jade C (FJC) was used to detect degenerating neurons. Western blot was used to detect proteins related with the amyloidogenic pathway including: APP (Amyloid-beta [Aβ] Precursor Protein), oligomeric Aβ, as well as pTAU (mainly component of NT), RAGE (receptor for advanced glycation end products, and LC3 (Light Chain 3). In the ADTg rats, moderate to intense AP deposition started around 6-8 months and progressively increased over time. Initially, non-fibrillar AP was detected at 6 months, that evolved to fibrillary AP after 8 months. Neurodegeneration observed as FJC positive neurons was observed after 8 months and progressed over time. No amyloid pathology or neurodegeneration were observed in control rats. Increased levels of APP and oligomeric Aβ started to be evident after 9 months in the AD Tg rat and increased over-time; compared to non-Tg rat. Levels of pTau were increased at all ages and gradually increased over-time. AD Tg rats showed increased levels of RAGE and LC3B starting at 15 months and increased over-time in AD Tg rat compared to non-Tg rat. Together, these data suggest that olfactory dysfunction worsen with disease progression in AD, which may correlate with early loss in olfaction. Further studies are necessary to fully characterize the amyloid pathology in the complete olfactory system including the entorhinal cortex and the hippocampus.

This study was supported by NCTR protocol E0763101.
Modeling of Parkinson’s disease related pathophysiology in primary human dopaminergic neurons: Role of Autophagy and Protein Aggregation
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The pathophysiology underlying the loss of dopaminergic (DA) neurons in Parkinson’s disease (PD) is still unclear. A major barrier to the development of effective therapies for PD is our limited understanding of the molecular and cellular events that lead to degeneration of the nigrostriatal DA system. 1-methyl-4-phenylpyridinium (MPP⁺) has been used as a reliable in vitro model of PD in dopaminergic neurons; however, the molecular mechanisms that lead to cell death with this model are not fully understood. Here, we characterized the expression of PD-related proteins and the incidence/rate of neuronal death after exposure of human DA neurons to MPP⁺ at different concentrations (0-10mM after 24h. From those data two concentration (1.0 and 2.5Mm) were chosen to test molecular markers of xxxx at 4, 8 and 24h. Viability assays and mitochondrial activity (LDH, XTT, Mitotracker, and Live/Dead staining), as well as protein expression of molecular markers of autophagy [lysosome-associated membrane protein 1 (LAMP-1), light chain 3 (LC3)], and dopaminergic status [tyrosine hydroxylase (TH)] were tested. The localization of α-synuclein (α-SYN) and parkin were also evaluated by immunocytofluorescence (ICF) at 1.0 and 2.5Mm of MPP⁺ after 24h. MPP⁺ significantly reduced cell viability, decreased mitochondrial activity, and reduced the number of live cells with a 24 h exposure. MPP⁺ at 1.0 and 2.5Mm induce expression of the LAMP-1. The ratio of LC3BII/LC3BI was also increased, whereas the expression of TH decreased. IF analysis revealed a decrease in parkin binding. Also α-SYN aggregation was cytoplasmic and nuclear. These data suggest that MPP⁺ could be affecting pathways related to autophagy, DA synthesis and protein aggregation.
Sex differences in olfactory dysfunction in a transgenic rat model of Alzheimer’s disease
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Olfactory dysfunction (OD) generally occurs at early stages in Alzheimer’s disease (AD), with a prevalence of approximately 90%. Accumulation of amyloid beta (Aβ) and hyper-phosphorylated tau (pTau) proteins has been observed in the olfactory bulb (OB) of AD patients. However, little is known about potential sex differences of OD in AD pathology even though AD is more prevalent in women. In the current study, those AD-related proteins in the OB were quantified in a rodent AD model. The OB of Tg AD rats (TgADr) from both sexes, along with their wild-type (WT) counterparts, were dissected and collected across different ages (9-24 months[M]). The protein expression of APP (Aβ Precursor Protein), mOC64 (Aβ1-42; conformation specific), as well as the receptor for advanced glycation end products (RAGE) and pTau and were evaluated using western blot (WB). Protein levels of APP, mOC64, and RAGE, in TgADr were significantly higher in females compared to males from 9-12M. Those levels were also significantly higher in TgADr females compared to same-sex WT controls from 9-12M. In TgADr males, APP levels were elevated from 13-17M, without any changes to mOC64 levels relative to same-sex WT controls. RAGE levels, however, were elevated from 9-12M, while pTau levels increased from 18-24M, relative to same-sex WT controls. These data suggest that: 1) OD appears earlier in the AD progression of females than males in TgADr, and 2) that a direct correlation exists between key proteins and degeneration in TgADr females. These data provide insight into gender-specific temporal expression of altered proteins in AD.
NanoTherapy for Parkinson’s disease: Nicotine-NanoCeria prevents neuronal damage and reverses electrophysiological function in Primary Human Dopaminergic Neurons.

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The pathophysiology of dopaminergic (DA) loss in Parkinson’s disease (PD) is still unclear. Lack of effective therapies maybe due to limitations in our understanding of the molecular and cellular events leading to degeneration of the nigrostriatal DA system. Here, we report that a combination of Nicotine and NanoCeria presents an outstanding case of prevention of PD progression, in a 1-methyl-4-phenylpyridinium (MPP+) induced in vitro model of PD in primary human dopaminergic neurons. A co-treatment of Nicotine and NanoCeria significantly inhibited MPP+ induced inhibition of parkin expression and prevented the aggregation of a-synuclein suggesting a regulation of protein degradation pathway. More importantly, in a functional electrophysiological analysis using Multi-Electrode Array, we report for the first time that this therapeutic combination of Nicotine and NanoCeria was not only able to prevent MPP+ induced loss of neuronal function but also was able to significantly reverse it towards normal. Our data might suggest an efficacious role of Nicotine-NanoCeria combination for the prevention of neuronal loss and restoration of neuronal function during PD progression. Additional in vivo work will be required to validate these significant findings.
Detection of Biomarkers of Neurological Disease Using Enhancing Raman Spectroscopic Techniques
Amber Moody$^{1,2}$ and Bhavya Sharma$^1$
$^1$University of Tennessee, Knoxville. $^2$National Center for Toxicological Research, 3900 NCTR Rd, Jefferson, AR.

The detection biomarkers of neurological disease requires the development of new sensing technologies that are rapid, label-free, and non-invasive, and detect multiple targets simultaneously. Many neurological diseases can be monitored through studying changes in neurotransmitter concentration. The majority of the existing detection methods require multiple steps to process complex biological samples or involve invasive measures such as drilling holes in the skull. Here, we present the use of Raman spectroscopy (RS) for the non-invasive, or minimally invasive detection of neurotransmitters through a rat skull. RS provides excellent chemical specificity with simple instrumentation and requires little to no sample processing. There is also no interference from water in RS as in several other techniques. Disadvantages include the inherently weak signal associated with RS along with the surface selectivity of the technique. Enhancement techniques such as surface enhanced Raman spectroscopy (SERS) greatly increase the Raman signal to allow detection of very low concentration analytes. Spatially offset Raman spectroscopy (SORS) allows the signal to be obtained from subsurface layers of turbid media by collecting the Raman scattered light at a location that is spatially offset from the incident illumination point. Combining SORS with SERS allows for an enhanced Raman signal to be obtained in deep layers of material in a technique termed SESORS. We have optimized detection conditions for the SERS detection of neurotransmitters in aqueous solutions followed by SESORS detection of neurotransmitters through a skull.
The role of semantic information in visual search: insights from eye movements

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Visual search tasks are used to investigate how visual attention enhances processing of selected items within the visual field, and can provide insight into neurodiversity in perceptual processing. Recent research has shown that semantic information can play a role in visual search for letters and digits, and can interfere with processing of perceptual information. For example, in the size congruity effect (SCE), participants view two numbers that have different physical and numerical sizes, and are asked to identify the physically (or numerically) larger (or smaller) number. Reaction times (RTs) are faster when the target’s physical and numerical size are congruent, implying that the processing of numerical (semantic) and physical (perceptual) size interact. Although this effect has been replicated in numerous studies, there is still debate about the stage of processing at which the interaction between the two types of information occurs.

Here, we investigated the stage at which semantic (numerical size) and perceptual (physical size) information interact in neurotypical individuals by tracking eye movements. Participants searched for a target digit among distractors (e.g. a 2 or a 3 surrounded by 8’s or 9’s) in circular displays containing 5, 7, or 9 digits. Targets were either congruent (e.g., a physically large 9 among physically small 2s and 3s) or incongruent (e.g., a physically large 2 among physically small 8s and 9s). We recorded participants’ eye movements and measured the time it took participants to initially fixate on the target, the duration of fixation on the target, and the total number of fixations. Participants took longer to initially fixate incongruent compared to congruent targets, suggesting that semantic and perceptual information may be processed together early rather than interacting at a later decision stage. Early interference between perceptual and semantic information could lead to longer processing time of individual items in the visual search display and therefore delayed initial fixation of incongruent targets. This pattern is somewhat surprising because of recent studies consistent with the idea that physical and numerical size are processed independently until the decision stage. Additional experiments will address whether fixation patterns remain consistent with the early interaction view when displays are modified to encourage participants to make eye movements on a greater proportion of trials. Future studies will address whether the stage of processing at which semantic and perceptual information interacts to influence the efficiency of visual search differs between neurotypical participants and individuals with neurodevelopmental conditions such as autism.
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