EINSTEIN Albert Einstein College of Medicine Montefiore

JUNE, 2020 VOL. 1 ISSUE 1

THE CNL

The official newsletter of the Cognitive Neurophysiology Lab and the Human Clinical Phenotyping Core

Editorial

On behalf of the entire CNL team, we are delighted to present the first edition of THE CNL- the official newsletter of the Cognitive Neurophysiology Laboratory and the Human Clinical Phenotyping Core at Albert Einstein College of Medicine. As a lab, we continuously strive to connect with the wonderful communities that make our research possible. From our participation in local events to our engagement on social media, we work to bridge the worlds of science and community, which can sometimes feel so far apart. With this semi-annual newsletter, we intend to strengthen that bridge by sharing with you our interests, projects, and findings in a way that is both inviting and accessible. Each edition will highlight a subset of our work, beginning with this first edition, which focuses on our rare disease and autism spectrum disorder projects. To our participants near and far, we thank you, and hope this newsletter excites you about the studies you have contributed to and those that are yet to come. To our colleagues and members of the scientific community, we hope this encourages you to learn more about the neurodevelopmental and neuropsychiatric disorders we are investigating.

Stay safe and see you soon,

Alaina Berruti, Ana Francisco, Filip de Sanctis, & Sophie Molholm

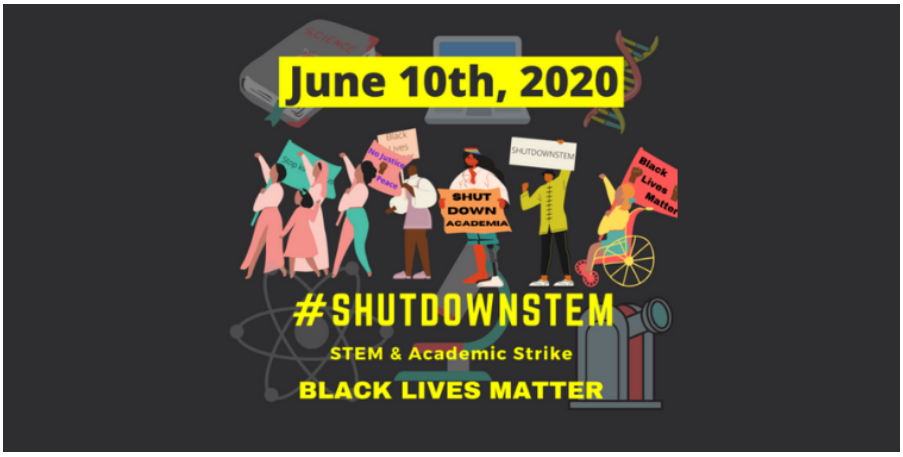
IN THIS ISSUE

To access the links inside, please see the online version of this newsletter: cognitiveneurolab.com/newsletters

The CNL stands with #BlackLivesMatter

BLACK LIVES MATTER

As a lab, we stand in solidarity with the Black community, alongside Einstein and Montefiore, in fighting against social and racial injustice. In reflecting on how we can use our resources to uplift the Black community, we have developed the **CODE** Committee. This committee will lead the CNL in expanding our scope of **C**ommunity **O**utreach and increasing our **D**iversity **E**fforts through various initiatives, which will be announced via social media and future newsletters. We are invested in the successful implementation of CODE initiatives, and welcome suggestions and collaborations from all participants, colleagues, and community members.



The CNL #ShutDownSTEM on Wednesday, June 10th, to reflect on how we, as a lab, can foster an environment of anti-racism.

The CNL, alongside the Einstein and Montefiore community, observed a moment of silence on Friday, June 5th, in memory of George Floyd and countless others.

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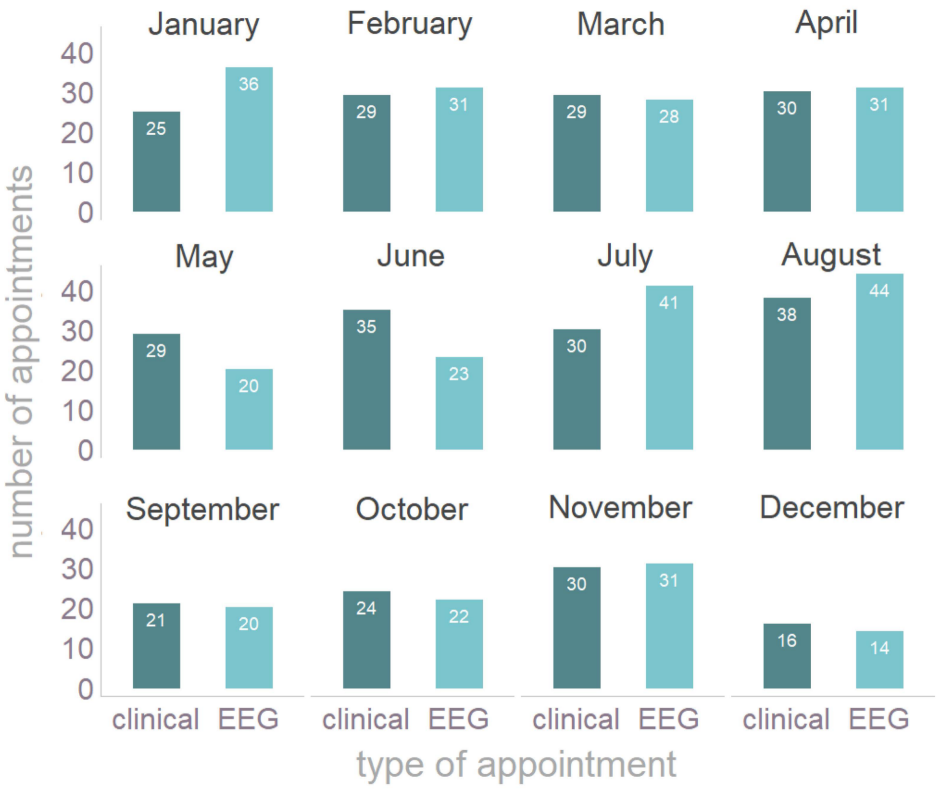
Our mission

The mission driving our research is to understand the neural processes underlying perception and cognition in the human brain, and to define how these differ in (and contribute to) neurodevelopmental and neuropsychiatric conditions. To this end, we use state-of-the-art brain imaging tools, such as electroencephalography (EEG) and magnetic resonance imaging (MRI), in concert with body/eye tracking, task guided behavior, standardized neuropsychological and clinical tests, and genetic information. We apply these tools to study attention, executive function, multisensory and sensorimotor integration, and basic sensory processing. We work with populations that span from childhood to older adults, and include genetic conditions such as 22q11.2 deletion syndrome and Rett syndrome, idiopathic neurodevelopmental and neuroconditions including autism and schizophrenia, and degenerative conditions such as multiple sclerosis. In our clinical work we collaborate with essential clinical partners at Montefiore, Jacobi, and University of Rochester Medical Center. Our ultimate goal is to increase basic understanding of brain function and, in turn, to use this knowledge for the development of markers of risk and disease progression and of optimized and targeted intervention programs in clinical conditions.

2019

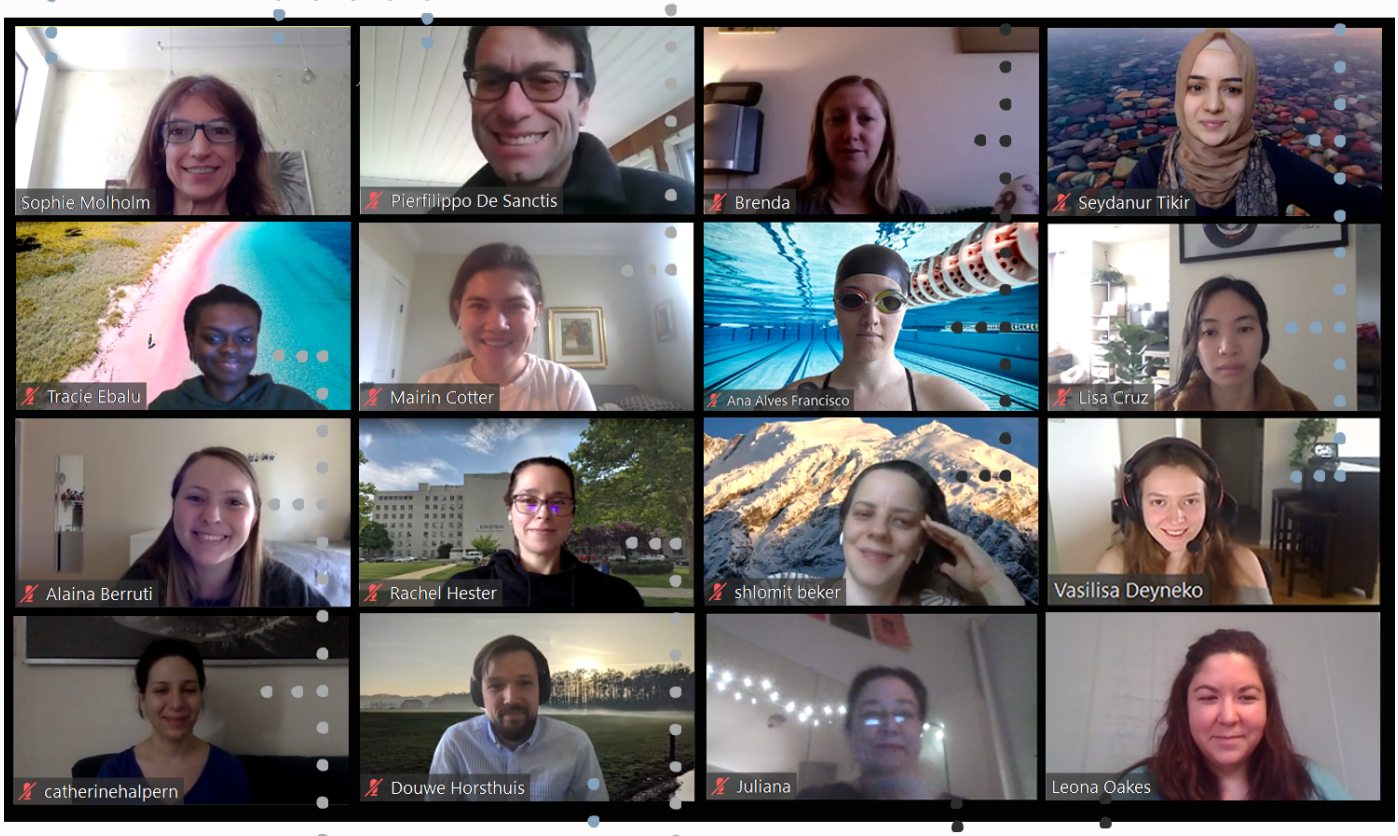
2019 was a great year! Check the numbers below!

Our work would not be possible without you and, therefore, we would like to extend our most sincere gratitude to the participants and their families for their interest, their involvement, and their time.



Humans of the CNL

Due to COVID-19 our lab members are working from home and communicating on ZOOM. Fortunately, we have been able to continue our research by conducting interviews and questionnaires with participants over the phone and online!



Not pictured: John Foxe (Founder & Primary Investigator), Emily Bates (Student Trainee)

Meet the group

In each newsletter, we will be introducing our select members of our lab! In this issue, you'll meet our coordinators, technicians, and administrative staff. Say hello to Alaina, Cat, Douwe, and Rachel!

CNL virtually takes the CNS

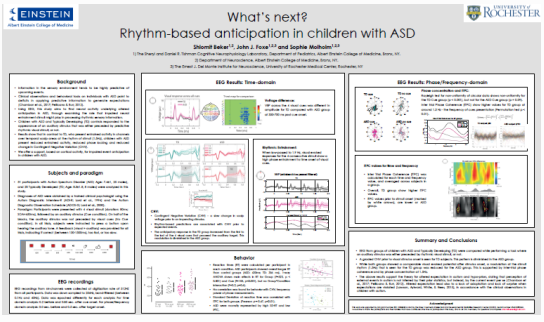
with a total of seven poster presentations!

The Cognitive Neuroscience Society (CNS) hosts an annual conference where its members gather to present their latest work. This year, the CNL had plans to travel to the Boston conference in March to present a total of seven posters, each showcasing different projects we've been working on. Due to the COVID-19 outbreak, the conference has since been postponed to May and moved online. Nevertheless, we are eager to present our work, safely at home.

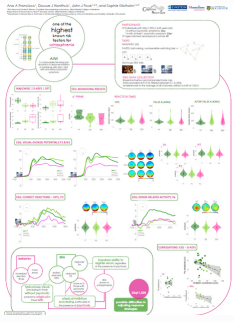
CNS2020 VIRTUAL

Check our posters below!
Click to enlarge and to see video presentations of some of the posters.

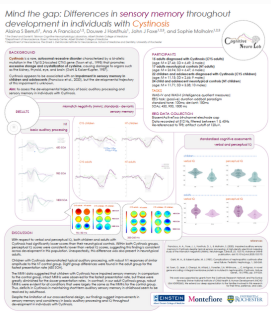
autism spectrum disorder



22q11.2DS



cystinosis



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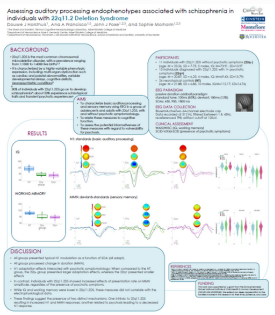
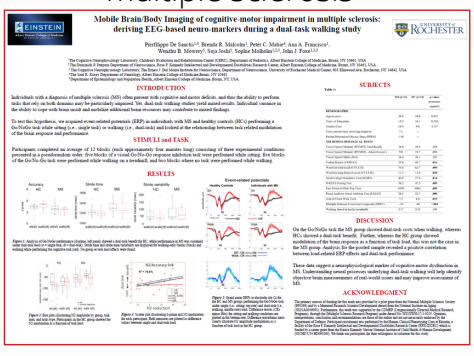
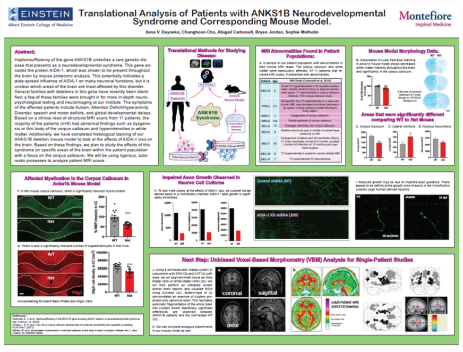


multiple sclerosis



ANKS1B

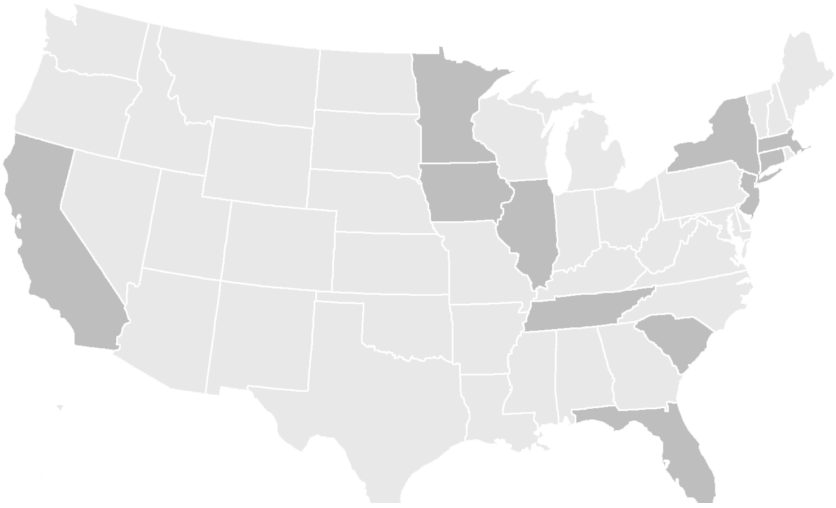


22q11.2DS

Celebrating rare disease research at the CNL

In honor of Rare Disease Day, we are showcasing our projects on cystinosis, rett syndrome, and 22q11.2 deletion syndrome.

Rare disease families across the US visited the CNL in 2019. We look forward to meeting more in 2020!



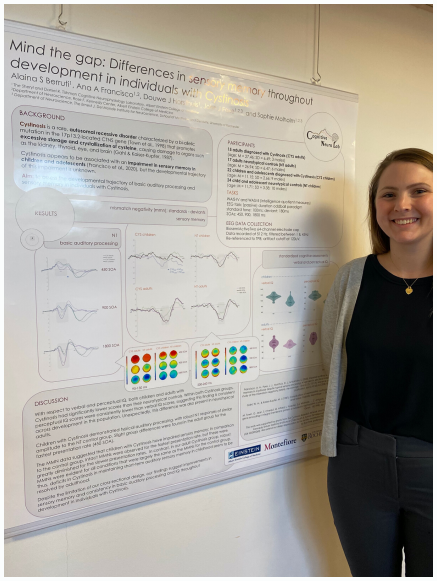
A rare disease or disorder is defined as a disease affecting up to

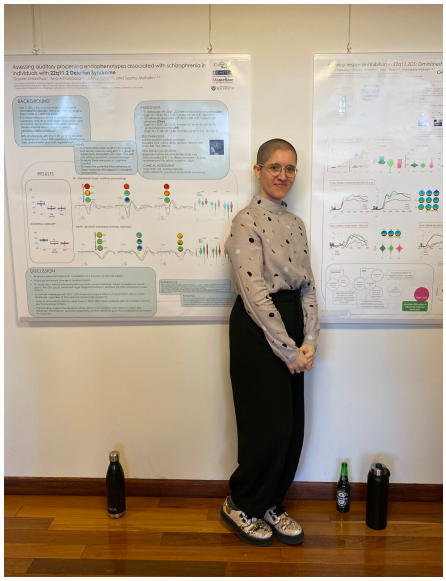
200,000 people in the U.S.

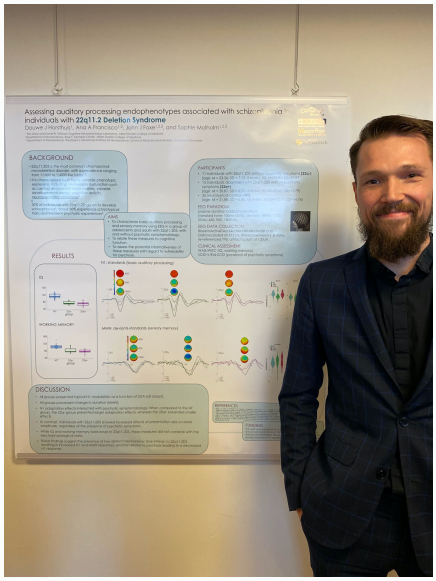
RARE DISEASES IMPACT **30 MILLION**

1 IN 10 AMERICANS

Celebrating Rare Disease Day 2020 at Einstein



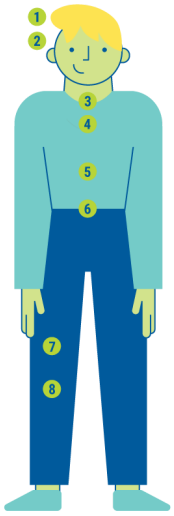




Celebrating rare disease research at the CNL

cystinosis

Cystinosis is a rare genetic disease affecting 1 in 100,000-200,000 live births in the United States. It is a lysosome storage disorder caused by a mutation in the CTNS gene on chromosome 17. This gene encodes a protein called cystinosin. Cystinosin transports an amino acid called cystine out of an intracellular compartment, the lysosome. If the cystinosin protein is absent or not fully functional, cystine accumulates in the lysosome and forms crystals, damaging the cells. Cystinosis affects different organs of the body!
Click here for a description of our research on cystinosis.



www.kidneyfund.org

1. BRAIN
   Visual or learning issues
2. EYES
   Sensitivity to light (photophobia), or blindness
3. THYROID
   Poorly working thyroid (hypothyroidism) leading to slowed growth
4. THROAT
   Trouble swallowing
5. PANCREAS
   Diabetes
6. KIDNEYS
   Fanconi syndrome (a type of kidney disease)
7. MUSCLES
   Muscle weakness and decreased muscle mass (myopathy)
8. BONES
   Softening or weakening of bones (rickets)

Earlier this year, Ana Francisco participated in the #GreatGivers campaign for the Cystinosis Research Network. Check it here!

A special thanks to Dr. Kaskel and to the Cystinosis Research Network for all their support!

CYSTINOSIS RESEARCH NETWORK

rett syndrome

Rett syndrome is a rare genetic neurological disorder affecting 1 in 10,000 female births. It is even rarer in boys. Rett is caused by mutations on the X chromosome on a gene called MECP2. It leads to mild/severe impairments, affecting nearly every aspect of the child's life: their ability to talk, eat, and even breathe. Rett is often diagnosed in children between 6 to 18 months as they begin to miss developmental milestones or lose abilities they had acquired.

The Children's Hospital at Montefiore

Click here for a description of our research on Rett. This work is done in collaboration with the Rett Syndrome Center at the Children's Hospital at Montefiore.

Celebrating rare disease research at the CNL

22q11.2 deletion syndrome

22q11.2 deletion syndrome (also referred to as velocardiofacialsyndrome (VCFS) or DiGeorge syndrome) is a disorder caused by a small missing piece of chromosome 22. Affecting 1 in 4,000 people worldwide, this syndrome is almost as common as Down syndrome. 22q11.2 deletion can result in about 200 mild to serious health and developmental issues in children, which include: growth delays, feeding problems, congenital heart disease, gastrointestinal difficulties, serious breathing concerns, cleft and craniofacial issues, calcium deficiencies, immune deficiencies, kidney problems, and skeletal anomalies. This list also includes the possibility of speech, developmental and cognitive delays, as well as ADHD, Autism, and anxiety and other psychiatric disorders.

Click here for a description of our research on 22q11.2 deletion syndrome.
This work is done in collaboration with the Montefiore-Einstein Regional Center for 22q11.2 deletion syndrome at the Children's Hospital at Montefiore.

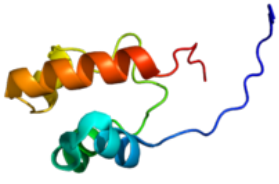
22Q TEXAS

We had an amazing time at the 22q Camp Texas! Thank you for having us!

For more on 22q11.2, check the resources of the 22q Family Foundation

the 22q FAMILY foundation

ANKS1B



The ANKS1B syndrome is caused by mutations in a gene with the same name. There is still a lot to be understood about this syndrome, but it appears to be very rare. In the families we were able to identify and test, ANKS1B is characterized by neurodevelopmental differences including Autism, ADHD, and speech and motor deficits. Our aim is to continue to characterize ANKS1B and understand how the syndrome affects individuals and families. We are using different methods to accomplish this goal, both in humans and in mouse and cell models.

In collaboration with Bryen Jordan's group, we published the first paper on this syndrome last year.

Project Spotlight

Autism Center of Excellence: Increasing representation of human diversity

Our goals

In this issue, we are spotlighting our on-going project with the Autism Center of Excellence. This project has two main goals, the first of which aims to understand the role of genetics in the development of Autism in those with African (African, African-American, Afro-Caribbean , Afro-Latino/a) heritage. The role of genetics in Autism has been a topic of interest for some time, but minority populations have been widely underrepresented in this research. Since we know various racial backgrounds have distinct genetic makeups, our project intends to fill the gaps in our knowledge of the genetic impact on Autism in these populations.

Secondly, our project aims to understand the barriers to diagnosis and treatment of Autism in these groups. Previous research has shown that minority children are, on average, diagnosed with Autism over a year later than children of Caucasian decent. Therefore, we are interested in the cultural, societal, and economic factors which contribute to this discrepency.

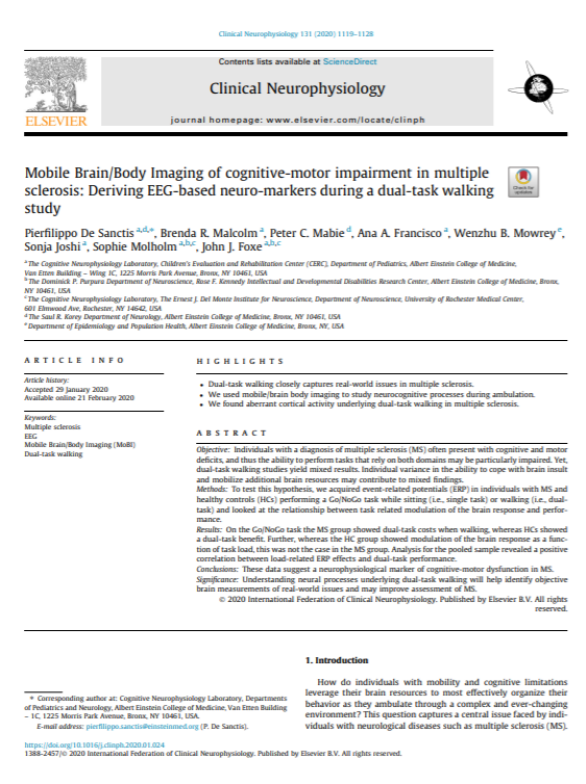
As we move towards developing targeted treatments and interventions for Autism, we need to ensure they will work for everyone. If you're interested in learning more about our project, please contact tracie.ebalu@einsteinmed.org.

Meet Tracie, the ACE study coordinator!

Tracie's responsibilities include recruitment and scheduling, administering clinical interviews, and data management. Tracie is interested in cognitive neurodevelopment in children and youth. In her spare time, Tracie enjoys solo-travelling, cooking, swimming, salsa dancing, and playing volleyball.

Einstein is one of six universities across the US collaborating on this exciting project.

Recent publications



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Contents lists available at ScienceDirect

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journal homepage: www.elsevier.com/locate/clinph

Mobile Brain/Body Imaging of cognitive-motor impairment in multiple sclerosis: Deriving EEG-based neuro-markers during a dual-task walking study

Pierfilippo De Sanctis [a,d,*], Brenda R. Malcolm [a], Peter C. Mabie [d], Ana A. Francisco [a], Wenzhu B. Mowrey [e], Sonja Joshi [a], Sophie Molholm [a,b,c], John J. Foxe [a,b,c]

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ARTICLE INFO

Article history:
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Keywords:
Multiple sclerosis
EEG
Mobile Brain/Body Imaging (MoBI)
Dual-task walking

HIGHLIGHTS

- Dual-task walking closely captures real-world issues in multiple sclerosis.
- We used mobile/brain body imaging to study neurocognitive processes during ambulation.
- We found aberrant cortical activity underlying dual-task walking in multiple sclerosis.

ABSTRACT

Objective: Individuals with a diagnosis of multiple sclerosis (MS) often present with cognitive and motor deficits, and thus the ability to perform tasks that rely on both domains may be particularly impaired. Yet, dual-task walking studies yield mixed results. Individual variance in the ability to cope with brain insult and mobilize additional brain resources may contribute to mixed findings.
Methods: To test this hypothesis, we acquired event-related potentials (ERP) in individuals with MS and healthy controls (HCs) performing a Go/NoGo task while sitting (i.e., single task) or walking (i.e., dual-task) and looked at the relationship between task related modulation of the brain response and performance.
Results: On the Go/NoGo task the MS group showed dual-task costs when walking, whereas HCs showed a dual-task benefit. Further, whereas the HC group showed modulation of the brain response as a function of task load, this was not the case in the MS group. Analysis for the pooled sample revealed a positive correlation between load-related ERP effects and dual-task performance.
Conclusions: These data suggest a neurophysiological marker of cognitive-motor dysfunction in MS.
Significance: Understanding neural processes underlying dual-task walking will help identify objective brain measurements of real-world issues and may improve assessment of MS.

1. Introduction

How do individuals with mobility and cognitive limitations leverage their brain resources to most effectively organize their behavior as they ambulate through a complex and ever-changing environment? This question captures a central issue faced by individuals with neurological diseases such as multiple sclerosis (MS).

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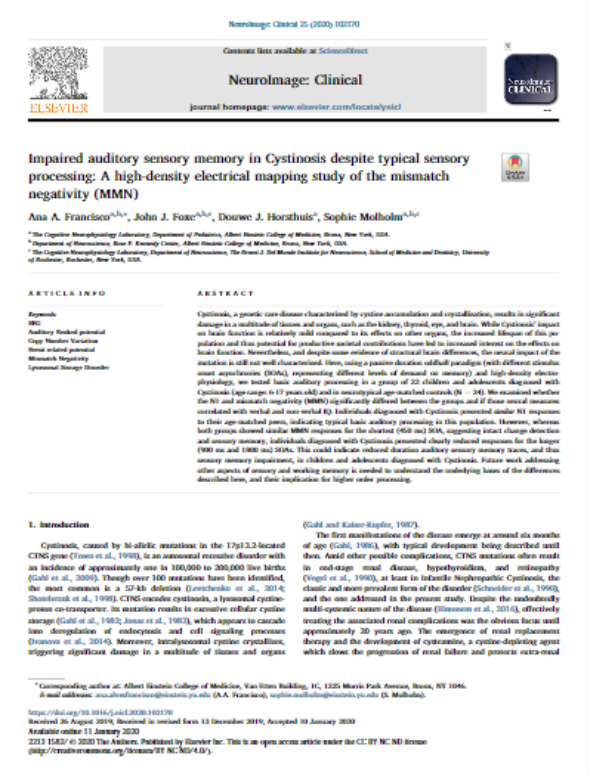
https://doi.org/10.1016/j.clinph.2020.01.024
1388-2457/

We measured brain activation using electroencephalography (EEG) in individuals with Multiple Sclerosis (MS) and healthy controls (HCs) performing a cognitive task while standing (single task) or walking (dual-task). We say that the brain processes multi-task information efficiently when the number of mistakes in the cognitive task while standing is equal or even less than the number of mistakes in the cognitive task while walking. In HCs, we found a relationship between changes in brain activation and better cognitive performance during dual-tasking, but there was no evidence of such relationship in MS. Our data could suggest that individuals with MS are less able to leverage brain resources in multi-task situations, but the comparison was somewhat unfair as our sample was composed of individuals with a diagnosis between 2 and 15 years' prior to study participation (we are currently enrolling individuals during early-stage MS!). Our studies have the potential to lead to improved diagnosis of cognitive and motor difficulties in MS and more effective interventions. With a better understanding of how the brain responds to and processes multi-task information, clinicians can begin to develop interventions that are based on known neurophysiological profiles.

We studied auditory processing and sensory memory in cystinosis. While basic auditory processing is fully functional, children and adolescents with cystinosis seem to have some difficulties with sensory memory. Sensory memory is the shortest type of memory and it helps keeping track of information in the millisecond rate. Because basic and complex aspects of brain processing are related and co-dependent (for example, a problem in a more basic process can hinder the processing at a more complex level), difficulties at this very basic level of memory could impact, for example, working memory—crucial to fulfill day-to-day tasks and to academic success. Remarkably, though, when we did this same task in adults with cystinosis (publication coming soon!!), their sensory memory appeared to be intact. This means that though some difficulties are present in younger individuals with cystinosis (which may have a negative impact in both school and home contexts and should be considered when developing education plans, for instance), they appear to be solved in adulthood.



NeuroImage: Clinical 25 (2020) 102170

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NeuroImage: Clinical

journal homepage: www.elsevier.com/locate/ynicl

Impaired auditory sensory memory in Cystinosis despite typical sensory processing: A high-density electrical mapping study of the mismatch negativity (MMN)

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ARTICLE INFO

Keywords:
HSC
Auditory Evoked potential
Copy Number Variation
Event related potential
Mismatch Negativity
Lysosomal Storage Disorder

ABSTRACT

Cystinosis, a genetic rare disease characterized by cystine accumulation and crystallization, results in significant damages in a multitude of tissues and organs, such as the kidney, thyroid, eye, and brain. While Cystinosis' impact on brain function is relatively mild compared to its effects on other organs, the increased lifespan of this population and thus potential for productive societal contributions have led to increased interest on the effects on brain function. Nevertheless, and despite some evidence of structural brain differences, the neural impact of the mutation is still not well characterized. Here, using a passive duration oddball paradigm (with different stimulus onset asynchronies (SOAs), representing different levels of demand on memory) and high-density electrophysiology, we tested basic auditory processing in a group of 22 children and adolescents diagnosed with Cystinosis (age range: 6-17 years old) and in neurotypical age-matched controls (N = 24). We examined whether the N1 and mismatch negativity (MMN) significantly differed between the groups and if those neural measures correlated with verbal and non-verbal IQ. Individuals diagnosed with Cystinosis presented similar N1 responses to their age-matched peers, indicating typical basic auditory processing in this population. However, whereas both groups showed similar MMN responses for the shortest (450 ms) SOA, suggesting intact change detection and sensory memory, individuals diagnosed with Cystinosis presented clearly reduced responses for the longer (900 ms and 1800 ms) SOAs. This could indicate reduced duration auditory sensory memory traces, and thus sensory memory impairment, in children and adolescents diagnosed with Cystinosis. Future work addressing other aspects of sensory and working memory is needed to understand the underlying bases of the differences described here, and their implication for higher order processing.

1. Introduction

Cystinosis, caused by bi-allelic mutations in the 17p13.2-located *CTNS* gene (Town et al., 1998), is an autosomal recessive disorder with an incidence of approximately one in 100,000 to 200,000 live births (Gahl et al., 2016). Though over 100 mutations have been identified, the most common is a 57-kb deletion (Levtchenko et al., 2014; Shotelersuk et al., 1998). CTNS encodes cystinosin, a lysosomal cystine-proton co-transporter. Its mutation results in excessive cellular cystine storage (Gahl et al., 1982; Jonas et al., 1982), which appears to cascade into dysregulation of endocytosis and cell signaling processes (Ivanova et al., 2014). Moreover, intralysosomal cystine crystallizes, triggering significant damage in a multitude of tissues and organs (Gahl and Kaiser-Kupfer, 1987).

The first manifestations of the disease emerge at around six months of age (Gahl, 1986), with typical development being described until then. Amid other possible complications, CTNS mutations often result in end-stage renal disease, hypothyroidism, and retinopathy (Vogel et al., 1990), at least in Infantile Nephropathic Cystinosis, the classic and more prevalent form of the disorder (Schneider et al., 1990), and the one addressed in the present study. Despite the undoubtedly multi-systemic nature of the disease (Elmonem et al., 2016), effectively treating the associated renal complications was the obvious focus until approximately 20 years ago. The emergence of renal replacement therapy and the development of cysteamine, a cystine-depleting agent which slows the progression of renal failure and protects extra-renal

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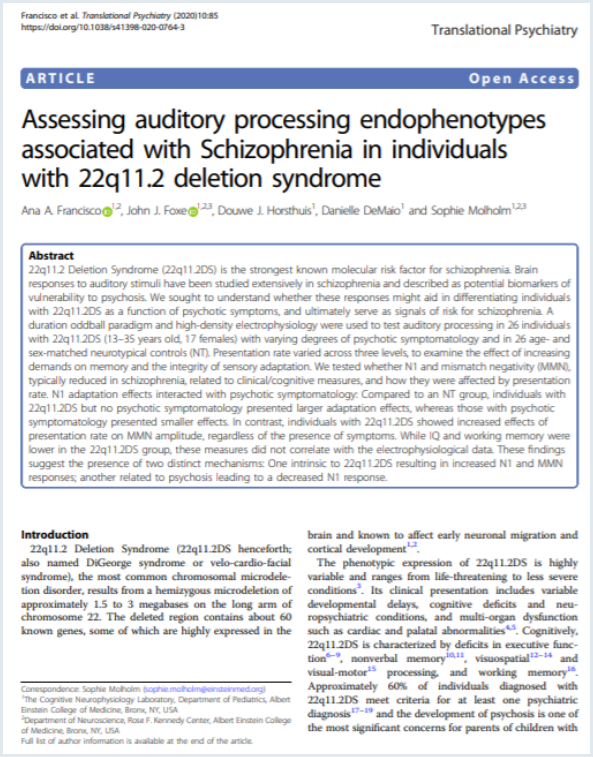
Recent publications



Francisco et al. *Translational Psychiatry* (2020)10:85
https://doi.org/10.1038/s41398-020-0764-3

Translational Psychiatry

ARTICLE Open Access

Assessing auditory processing endophenotypes associated with Schizophrenia in individuals with 22q11.2 deletion syndrome

Ana A. Francisco[1,2], John J. Foxe[1,2,3], Douwe J. Horsthuis[1], Danielle DeMaio[1] and Sophie Molholm[1,2,3]

Abstract

22q11.2 Deletion Syndrome (22q11.2DS) is the strongest known molecular risk factor for schizophrenia. Brain responses to auditory stimuli have been studied extensively in schizophrenia and described as potential biomarkers of vulnerability to psychosis. We sought to understand whether these responses might aid in differentiating individuals with 22q11.2DS as a function of psychotic symptoms, and ultimately serve as signals of risk for schizophrenia. A duration oddball paradigm and high-density electrophysiology were used to test auditory processing in 26 individuals with 22q11.2DS (13–35 years old, 17 females) with varying degrees of psychotic symptomatology and in 26 age- and sex-matched neurotypical controls (NT). Presentation rate varied across three levels, to examine the effect of increasing demands on memory and the integrity of sensory adaptation. We tested whether N1 and mismatch negativity (MMN), typically reduced in schizophrenia, related to clinical/cognitive measures, and how they were affected by presentation rate. N1 adaptation effects interacted with psychotic symptomatology: Compared to an NT group, individuals with 22q11.2DS but no psychotic symptomatology presented larger adaptation effects, whereas those with psychotic symptomatology presented smaller effects. In contrast, individuals with 22q11.2DS showed increased effects of presentation rate on MMN amplitude, regardless of the presence of symptoms. While IQ and working memory were lower in the 22q11.2DS group, these measures did not correlate with the electrophysiological data. These findings suggest the presence of two distinct mechanisms: One intrinsic to 22q11.2DS resulting in increased N1 and MMN responses; another related to psychosis leading to a decreased N1 response.

Introduction

22q11.2 Deletion Syndrome (22q11.2DS henceforth; also named DiGeorge syndrome or velo-cardio-facial syndrome), the most common chromosomal microdeletion disorder, results from a hemizygous microdeletion of approximately 1.5 to 3 megabases on the long arm of chromosome 22. The deleted region contains about 60 known genes, some of which are highly expressed in the brain and known to affect early neuronal migration and cortical development[1,2].

The phenotypic expression of 22q11.2DS is highly variable and ranges from life-threatening to less severe conditions[3]. Its clinical presentation includes variable developmental delays, cognitive deficits and neuropsychiatric conditions, and multi-organ dysfunction such as cardiac and palatal abnormalities[4,5]. Cognitively, 22q11.2DS is characterized by deficits in executive function[6–9], nonverbal memory[10,11], visuospatial[12–14] and visual-motor[15] processing, and working memory[16]. Approximately 60% of individuals diagnosed with 22q11.2DS meet criteria for at least one psychiatric diagnosis[17–19] and the development of psychosis is one of the most significant concerns for parents of children with

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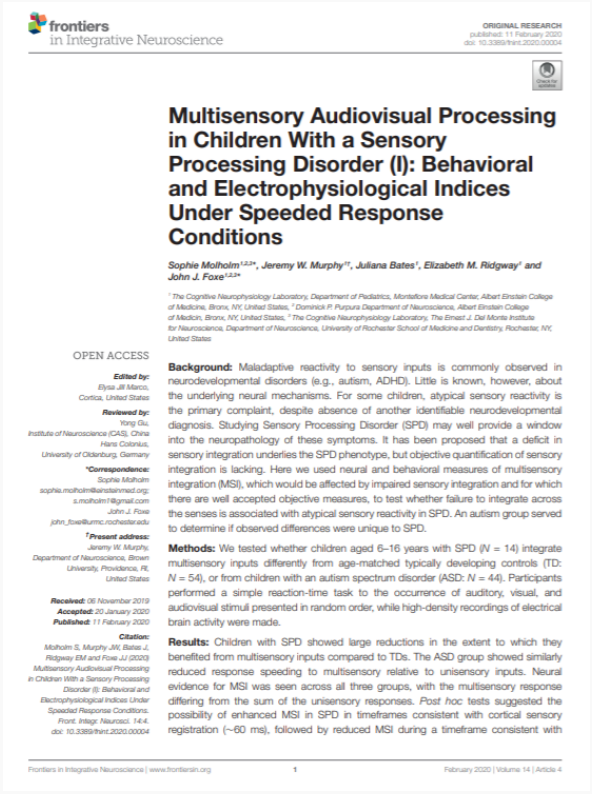
We used EEG, a non-invasive method which allows you to record the electrical activity of the brain, to understand basic auditory processing and sensory memory in people with 22q11.2DS. While basic auditory processing refers to the initial steps your brain takes to comprehend sound in your environment, sensory memory is the stage of memory that allows you to briefly remember these sounds. Our findings suggest that these two processes are overall functional in adolescents and adults with 22q11.2DS, at least in those individuals with less severe conditions. However, if there is any sign of psychosis, which is more prevalent in 22q11.2DS than in the general population, the auditory responses become reduced. We need larger groups of individuals with and without psychotic symptoms to confirm that these brain responses are indeed able to distinguish between those two groups, but these are important findings in trying to better understand the development of schizophrenia in 22q11.2DS.

Sensory Processing Disorder (SPD) is characterized by over or under responsiveness to sensory stimulation (sound, light, texture, etc). It is thought that this responsiveness is due to the nervous system's inability to regulate sensory input from the environment, but the exact mechanism of how this happens is still unknown. We sought to better understand how exactly sensory information is processed in individuals with SPD in comparison to typically developing individuals and individuals diagnosed with Autism, which is itself partly characterized by dysregulation of sensory processing. Here, we found that people with Autism and SPD process auditory (alone) and visual (alone) stimuli similarly to those that are typically developing. However, processing a joint audio-visual stimulus is a little different than processing a stimulus that is only auditory or only visual. In typically developing people, we see that audio-visual stimuli are processed much faster than stimuli that are only auditory or visual. That is, there seems to be an advantage to processing multi-sensory information over single-sensory information. Our study found evidence that people with Autism and SPD also benefit from processing multi-sensory information. However, this benefit is significantly reduced in comparison to that of their typically developing peers.



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ORIGINAL RESEARCH
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Multisensory Audiovisual Processing in Children With a Sensory Processing Disorder (I): Behavioral and Electrophysiological Indices Under Speeded Response Conditions

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Background: Maladaptive reactivity to sensory inputs is commonly observed in neurodevelopmental disorders (e.g., autism, ADHD). Little is known, however, about the underlying neural mechanisms. For some children, atypical sensory reactivity is the primary complaint, despite absence of another identifiable neurodevelopmental diagnosis. Studying Sensory Processing Disorder (SPD) may well provide a window into the neuropathology of these symptoms. It has been proposed that a deficit in sensory integration underlies the SPD phenotype, but objective quantification of sensory integration is lacking. Here we used neural and behavioral measures of multisensory integration (MSI), which would be affected by impaired sensory integration and for which there are well accepted objective measures, to test whether failure to integrate across the senses is associated with atypical sensory reactivity in SPD. An autism group served to determine if observed differences were unique to SPD.

Methods: We tested whether children aged 6–16 years with SPD ($N = 14$) integrate multisensory inputs differently from age-matched typically developing controls (TD: $N = 54$), or from children with an autism spectrum disorder (ASD: $N = 44$). Participants performed a simple reaction-time task to the occurrence of auditory, visual, and audiovisual stimuli presented in random order, while high-density recordings of electrical brain activity were made.

Results: Children with SPD showed large reductions in the extent to which they benefited from multisensory inputs compared to TDs. The ASD group showed similarly reduced response speeding to multisensory relative to unisensory inputs. Neural evidence for MSI was seen across all three groups, with the multisensory response differing from the sum of the unisensory responses. *Post hoc* tests suggested the possibility of enhanced MSI in SPD in timeframes consistent with cortical sensory registration (~60 ms), followed by reduced MSI during a timeframe consistent with

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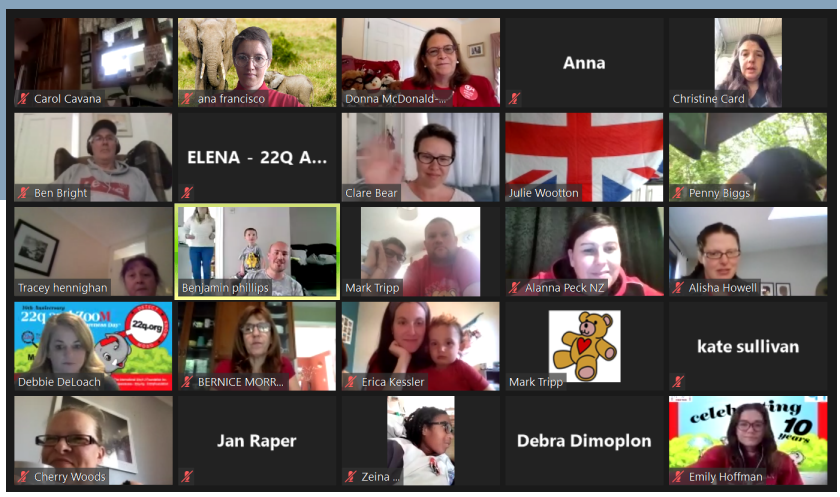
Online resources for children & caregivers navigating stress and changes from COVID-19.



This year, 22q at the Zoo was 22q and Zoom!

GoNoodle: free movement, yoga, and mindfullness videos for kids at home.

Remote education resource center: educational resources for all grade levels.

"Caring for Each Other" by Sesame Street: ebooks, coloring pages, games, and videos for children featuring their favorite Sesame Street characters.

Coronavirus social story: a book explaining the pandemic in simple language.

Child Mind Institute: A guide to supporting young adults during COVID-19.

Daily schedule maker: schedule templates for children at home.

Free & discounted services: a listing of entertainment, fitness, & mindfulness services.

Rare Diseases Clinical Research Network: Consider participating in research on how COVID-19 has impacted rare disease patients and their families.

#IStayHomeForRare: A campaign to support individuals living with rare diseases during this challenging time.

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