EINSTEIN Albert Einstein College of Medicine Montefiore

DECEMBER, 2020 VOL. 1 ISSUE 2

THE CNL

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

Editorial

On behalf of the entire team, we'd like to thank our readers for the warm response to our first issue of The CNL, which we released in June of this year. With 2020 coming to a close, we reflect on the unprecedented events of this year, and the impact they have had on our research, lab members, and participants. However, rather than focus on the challenges of this year, in this issue we highlight our successes, including adapting much of our research to online formats, and incorporating Spanish-translated materials (questionnaires and interviews) into our protocols to make our research more accessible. We also are elated that our lab published a total of 7 journal papers this year, and was awarded 2 grants. As we have always said, we are immensely grateful to our participants for making our research possible, and we'd like to thank all of our participants, colleagues, collaborators, and sponsors who have supported our research endeavors. We wish you all a happy and healthy holiday season and new year!

Stay safe and see you soon,

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

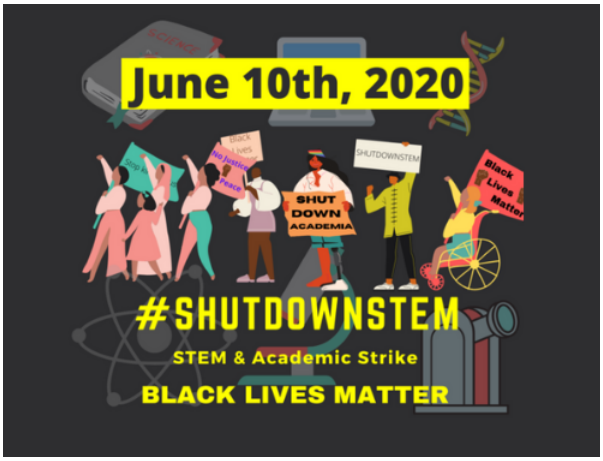
IN THIS ISSUE

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

CODE Update

In our first newsletter, we introduced the CODE Committee, which is a volunteer group of lab members dedicated to expanding our **C**ommunity **O**utreach and **D**iversity **E**fforts.

The CODE Committee was formed in response to the Black Lives Matter movement which encouraged us to reevaluate how we, an institution in The Bronx, serve the Black and other People of Color communities of The Bronx and surrounding areas. We aim for our efforts to increase these populations' general trust in science and their interest in pursuing careers in STEM. Moreover, we intend for our work to uphold principles of inclusivity for individuals of all races, ethnicities, gender identities, and sexual orientations.



CODE was formed in response to #ShutDownSTEM and #BlackLivesMatter movements.

Since its formation in June 2020, following #ShutDownSTEM, the CODE Committee has identified three initiatives in line with its core mission. They are as follows:

Material Monitoring: The Material Monitoring group regularly inspects the waiting area and testing spaces within the lab, in addition to online recruitment materials and participant surveys to ensure the physical environment and our online presence maintains an environment of inclusivity, An example of our most recent effort has been revising our enrollment form to include space for participants to include their gender identity and their preferred pronouns.

Code Camp: The Code Camp program was designed to introduce coding instruction and scientific data visualization skills to high school students in the South Bronx. Our team, in collaboration with community organizations within the South Bronx, is currently designing the first event, which will take place in the Summer of 2021, in collaboration with the Bronx Documentary Center.

Feedback Loop: The responsibilities of the Feedback Loop team involve actively seeking input from participants, parent advocacy groups. colleagues, and community members, and implementing their feedback into CNL/HCP operations. This group has added an "inclusivity suggestion box" into the CNL/HCP waiting room, to allow participants to anonymously share their thoughts with us.

Welcome Aboard!

The CNL welcomes two new lab members: Catherin Sancimino and Rinaldys Castillo!

Catherine Sancimino, Psy.D.

Catherine Sancimino, Psy.D., is a clinical psychologist with expertise in assessment of children with IDD. Dr. Sancimino joined the CNL/HCP to provide psychological testing for study participants. Her background includes providing evaluation and intervention in schools, outpatient psychiatric clinics, pediatric primary care, and research settings. She is excited to use her clinical skills to contribute to research at the CNL/HCP. Dr. Sancimino spends her free time practicing yoga, running marathons, cooking vegetables, and having fun with her husband and daughter.

Rinaldys Castillo, B.S.

Rinaldys is the Study Coordinator for our Autism Center of Excellence (ACE) project. We are one of six sites across the US in the ACE network, lead by UCLA, aiming to 1) increase the representation of Black participants in genetic research and 2) assess the barriers to diagnosis and treatment of Autism that their caregivers experience.

To this end, Rinaldys recruits, schedules, and interviews African, African American, Afro-Latinx, Afro-Caribbean, and Mixed-Race families with a child on the Autism spectrum. Additionally, he oversees the sample collection process and manages all medical records and datasets for this study.

Rinaldys graduated from Dickinson College in Carlisle, PA as a Biology major with an emphasis in biochemistry and molecular biology. He has previously worked at the Arthur Ashe Institute for Urban Health and the Max Planck Florida Institute for Neuroscience, and intends to pursue a career in translational/clinical research as a physician-scientist. Outside of the lab, Rinaldys can be found traveling, in a Latin ballroom or Afrobeats dance class, trying new cuisines, or at home binge-watching TV shows.

COVID-19 Update

How the CNL has responded to the COVID-19 pandemic

Since the COVID-19 shutdown in March 2020, we have been quick to adapt our procedures as needed to keep our research moving! For many of our projects, we have adapted our methods to allow for remote data collection (via online questionnaires and ZOOM interviews). For our projects that require in-person visits (e.g.: EEG recording), we have been back in business as of July 2020- though these visits look a little different! Our lab members now wear PPE, including scrubs, gloves, masks, and face shields, while working with participants, and are screened daily for COVID-19 symptoms and exposures. We've also modified our lab space with hand sanitizing stations, and provide face coverings for participants visiting us!

From left to right; Rinaldys, Alaina, and Douwe mask-and-scrub ready for participant visits!

Check out our "Welcome Back" video to see what you can expect during your visit to the CNL/HCP, and see how we are ensuring your safety during COVID-19.

Hand sanitizer station at the Lab

Project Spotlight

The MoBI Series: Mobile Brain-Body Imaging Research

Walking and Talking: Imaging the brain doing two things

One of the big questions about the brain is how it allows us to do several tasks at the same time. We are pretty good at navigating a busy mall while carrying on a conversation with a friend or daydreaming about a place we'd rather be. Yet, if we look carefully, there are costs to pay for doing two tasks at the same time. For example, we may slow down a bit if we start talking to a friend compared to walking without talking. Still, we are quite good at multitasking!

Our goal is to gain a better understanding of how the brain changes during multi-tasking in autism spectrum disorder, multiple sclerosis, and aging-related conditions such as mild cognitive impairment and dementia. Mobile Brain-body Imaging (MoBI) is a novel approach to acquire real-time measurements of active brain regions in concert with 3D body-tracking data to tie brain activity to gait with millisecond precision. MoBI represents a new frontier in the field of science and our group is one the first to apply this new approach in populations with mobility impairment.

Did you know?

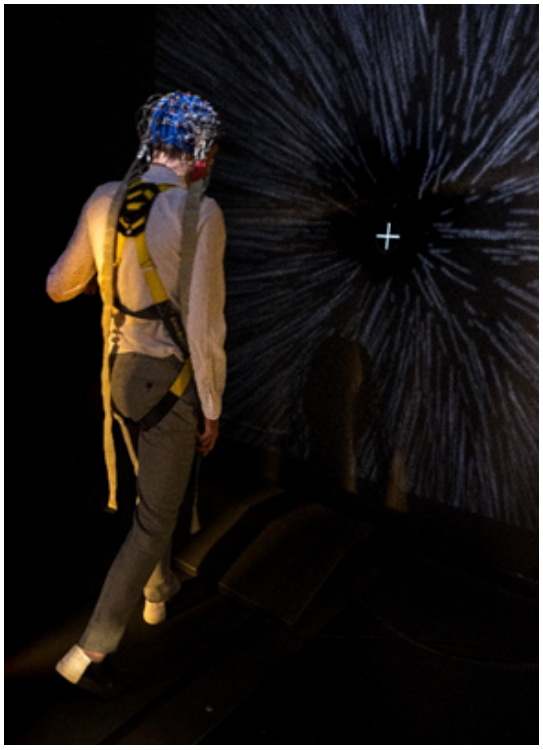
Aerobic exercise has been linked to improved cognition and brain function. Click here to read more.

Studying brain function during walking is important for many reasons. Walking is one of the most fundamental human activities. We know that staying active is important to delay and reduce the impact of aging on our health. The Go4Life initiative, a health education campaign by the National Institute on Aging, is driven by overwhelming evidence about the benefits of exercise to your health. With regard to children on the autism spectrum, motor coordination and gait abnormalities are among the earliest signs observed within the first two years of age. How motor deficits contribute to more complex behavior necessary for social and communicative development is a question we are trying to answer.

Project Spotlight

The MoBI Series: Mobile Brain-Body Imaging Research

Walking and Talking: Imaging the brain doing two things (cont.)



This picture shows the MoBI set up at our lab at Einstein. A volunteer is walking on a treadmill wearing a special cap to measure brain activity and a safety harness to secure against falls. The participant is immersed in a large scale star field moving outward. This creates the illusion of body forward movement through a virtual environment. We can control the visual input by introducing sudden shifts in the star field and study how well participants adapt to such visual perturbations that destabilize balance and gait. This can be thought of as a mobility stress test with varying degrees of stress to probe different situations.

For example, small amounts of stress may unmask subtle gait differences early in a patient's disease course that otherwise remain unseen. Large amounts of stress may reveal a patient's ability to function under high task difficulty. Brain resilience, the ability to cope with adversity, plays a big role in how an individual faces neurodevelopmental and neurological conditions.

Mobile Brain-body Imaging embraces the multidimensional nature of behavior. It jointly records human brain activity and 3D body tracking data while participants act and interact in a three-dimensional environment. It provides a new window into the brain and how it organizes our behavior to solve real-world tasks. This will lead to new knowledge about the brain and ways to help maintain and improve the health of children and older adults suffering from brain disorders.

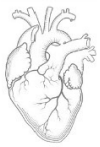
November was 22q11.2 deletion syndrome awareness month!

Click here for full version

Let's talk about 22q!!

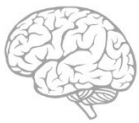
HOW ARE PEOPLE WITH 22q AFFECTED?

People with 22q are affected in different ways. Some have really mild signs and are never diagnosed. Others face life-threatening conditions. Here, we focus on three of the affected systems:



Most people with 22q have congenital heart disease. Those with more serious heart problems need surgery within their first year of life. Although much progress has been made, heart problems are still the main cause of death in this disease.

Most people with 22q have a weak immune system, which makes it much harder for the body to fight regular infections. Infections are more frequent and severe in 22q.



The brain of people with 22q is a little different. For example, language will develop later. This means that sometimes people with 22q might have a harder time in school and other daily activities. Fortunately, different strategies can be used to help them overcome these difficulties. People with 22q are more likely to be diagnosed with developmental and psychiatric disorders, such as ADHD and Autism. Anxiety is also very common in 22q.

Despite the challenges that people with 22q face, all without exception have incredible strengths and gifts and will achieve their potential with the right support!

November is 22q Awareness Month! Now that you heard about it, help us spread the word!

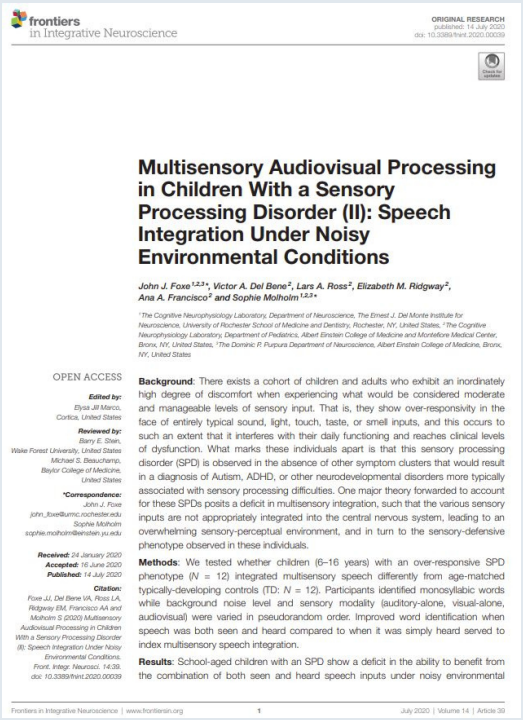
Awareness can save lives!

22q11.2 deletion syndrome

ANA ALVES FRANCISCO & DOUWE HORSTHUIS IN COLLABORATION WITH THE 22Q FAMILY FOUNDATION
NOVEMBER, 2020

Dutch, Portuguese, and Spanish versions coming soon!

Recent publications



frontiers in Integrative Neuroscience

ORIGINAL RESEARCH
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Multisensory Audiovisual Processing in Children With a Sensory Processing Disorder (II): Speech Integration Under Noisy Environmental Conditions

John J. Foxe[1,2,3], Victor A. Del Bene[2], Lars A. Ross[2], Elizabeth M. Ridgway[2], Ana A. Francisco[2] and Sophie Molholm[1,2,3]**

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Background: There exists a cohort of children and adults who exhibit an inordinately high degree of discomfort when experiencing what would be considered moderate and manageable levels of sensory input. That is, they show over-responsivity in the face of entirely typical sound, light, touch, taste, or smell inputs, and this occurs to such an extent that it interferes with their daily functioning and reaches clinical levels of dysfunction. What marks these individuals apart is that this sensory processing disorder (SPD) is observed in the absence of other symptom clusters that would result in a diagnosis of Autism, ADHD, or other neurodevelopmental disorders more typically associated with sensory processing difficulties. One major theory forwarded to account for these SPDs posits a deficit in multisensory integration, such that the various sensory inputs are not appropriately integrated into the central nervous system, leading to an overwhelming sensory-perceptual environment, and in turn to the sensory-defensive phenotype observed in these individuals.

Methods: We tested whether children (6–16 years) with an over-responsive SPD phenotype (N = 12) integrated multisensory speech differently from age-matched typically-developing controls (TD: N = 12). Participants identified monosyllabic words while background noise level and sensory modality (auditory-alone, visual-alone, audiovisual) were varied in pseudorandom order. Improved word identification when speech was both seen and heard compared to when it was simply heard served to index multisensory speech integration.

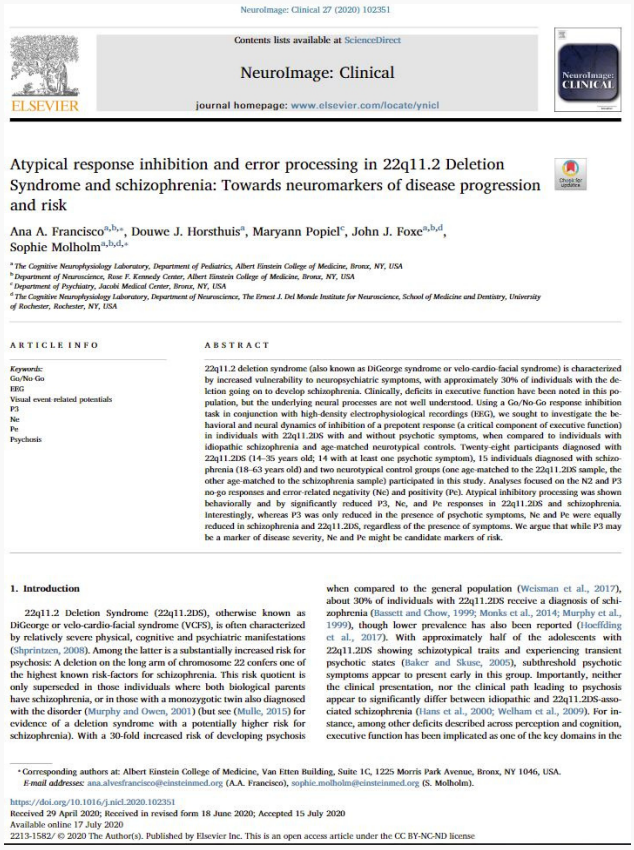
Results: School-aged children with an SPD show a deficit in the ability to benefit from the combination of both seen and heard speech inputs under noisy environmental

This work represents the ongoing close collaboration between the IDDRCs at the Rose F. Kennedy Center of The Albert Einstein College of Medicine and the University of Rochester's Del Monte Institute for Neuroscience.

Sensory Processing Disorder refers to a group of individuals who exhibit an inordinately high degree of discomfort when experiencing what would be considered moderate and manageable levels of sensory input – but who do not have any other diagnosis, such as a neurodevelopmental disorder or Autism.

It has long been speculated that SPD may arise because of poorer multisensory integration abilities. Here, we show that a group of children with SPD show a decrement in their ability to integrate heard and seen speech under noisy environmental conditions

We investigated response inhibition (ability to stop one's own behavior at the appropriate time, including stopping actions and thoughts) and error processing in a group of teenagers and adults with 22q11.2 deletion syndrome. Response inhibition is part of the set of skills we refer to as executive functions, fundamental to, for example, plan and execute goals, adapt to changes in the environment, etc. We asked our participants to press a button every time they saw a picture on the screen (most of the time) and to not press when the picture was repeated (only 15% of the time). This is a fast and difficult task: Imagine getting so used to doing something that you do it almost automatically and suddenly being asked to not do it! During all this, we were measuring the participants' brain responses to right and wrong responses using EEG. The participants with 22q11.2DS made more errors and their brain reacted differently to those errors, which may suggest that they not only have a harder time stopping responses, but may also experience some difficulties recovering from those errors. These difficulties impact personal and academic contexts, and should therefore be addressed. Strategies that may help include: Increased supervision, provide pre-corrects for desired behavior, rehearse skills before the potential difficult situation, teach to self-monitor, reinforce accurate self-monitoring.



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Atypical response inhibition and error processing in 22q11.2 Deletion Syndrome and schizophrenia: Towards neuromarkers of disease progression and risk

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ABSTRACT

22q11.2 deletion syndrome (also known as DiGeorge syndrome or velo-cardio-facial syndrome) is characterized by increased vulnerability to neuropsychiatric symptoms, with approximately 30% of individuals with the deletion going on to develop schizophrenia. Clinically, deficits in executive function have been noted in this population, but the underlying neural processes are not well understood. Using a Go/No-Go response inhibition task in conjunction with high-density electrophysiological recordings (EEG), we sought to investigate the behavioral and neural dynamics of inhibition of a prepotent response (a critical component of executive function) in individuals with 22q11.2DS with and without psychotic symptoms, when compared to individuals with idiopathic schizophrenia and age-matched neurotypical controls. Twenty-eight participants diagnosed with 22q11.2DS (14–35 years old; 14 with at least one psychotic symptom), 15 individuals diagnosed with schizophrenia (18–63 years old) and two neurotypical control groups (one age-matched to the 22q11.2DS sample, the other age-matched to the schizophrenia sample) participated in this study. Analyses focused on the N2 and P3 no-go responses and error-related negativity (Ne) and positivity (Pe). Atypical inhibitory processing was shown behaviorally and by significantly reduced P3, Ne, and Pe responses in 22q11.2DS and schizophrenia. Interestingly, whereas P3 was only reduced in the presence of psychotic symptoms, Ne and Pe were equally reduced in schizophrenia and 22q11.2DS, regardless of the presence of symptoms. We argue that while P3 may be a marker of disease severity, Ne and Pe might be candidate markers of risk.

1. Introduction

22q11.2 Deletion Syndrome (22q11.2DS), otherwise known as DiGeorge or velo-cardio-facial syndrome (VCFS), is often characterized by relatively severe physical, cognitive and psychiatric manifestations (Shprintzen, 2008). Among the latter is a substantially increased risk for psychosis: A deletion on the long arm of chromosome 22 confers one of the highest known risk-factors for schizophrenia. This risk quotient is only superseded in those individuals where both biological parents have schizophrenia, or in those with a monozygotic twin also diagnosed with the disorder (Murphy and Owen, 2001) (but see (Mulle, 2015) for evidence of a deletion syndrome with a potentially higher risk for schizophrenia). With a 30-fold increased risk of developing psychosis when compared to the general population (Weisman et al., 2017), about 30% of individuals with 22q11.2DS receive a diagnosis of schizophrenia (Bassett and Chow, 1999; Monks et al., 2014; Murphy et al., 1999), though lower prevalence has also been reported (Hoeffding et al., 2017). With approximately half of the adolescents with 22q11.2DS showing schizotypical traits and experiencing transient psychotic states (Baker and Skuse, 2005), subthreshold psychotic symptoms appear to present early in this group. Importantly, neither the clinical presentation, nor the clinical path leading to psychosis appear to significantly differ between idiopathic and 22q11.2DS-associated schizophrenia (Hans et al., 2000; Welham et al., 2009). For instance, among other deficits described across perception and cognition, executive function has been implicated as one of the key domains in the

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Recent publications

Timing of the Diagnosis of Autism in African American Children

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abstract

OBJECTIVES: African American (AA) children affected by autism spectrum disorder (ASD) experience delays in diagnosis and obstacles to service access, as well as a disproportionate burden of intellectual disability (ID) as documented in surveillance data recently published by the US Centers for Disease Control and Prevention. Our objective in this study was to analyze data from the largest-available repository of diagnostic and phenotypic information on AA children with ASD, and to explore the wide variation in outcome within the cohort as a function of sociodemographic risk and specific obstacles to service access for the purpose of informing a national approach to resolution of these disparities.

METHODS: Parents of 584 AA children with autism consecutively enrolled in the Autism Genetic Resource Exchange across 4 US data collection sites completed event history calendar interviews of the diagnostic odysseys for their children with ASD. These data were examined in relation to developmental outcomes of the children with autism and their unaffected siblings.

RESULTS: The average age of ASD diagnosis was 64.9 months (±49.6), on average 42.3 months (±45.1) after parents' first concerns about their children's development. The relationship between timing of diagnosis and ASD severity was complex, and ID comorbidity was not predicted in a straightforward manner by familial factors associated with cognitive variation in the general population.

CONCLUSIONS: These findings document significant opportunity to expedite diagnosis, the need to further understand causes of ID comorbidity, and the necessity to identify effective approaches to the resolution of disparities in severity-of-outcome for AA children with autism.

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*Contributed equally as co-first authors

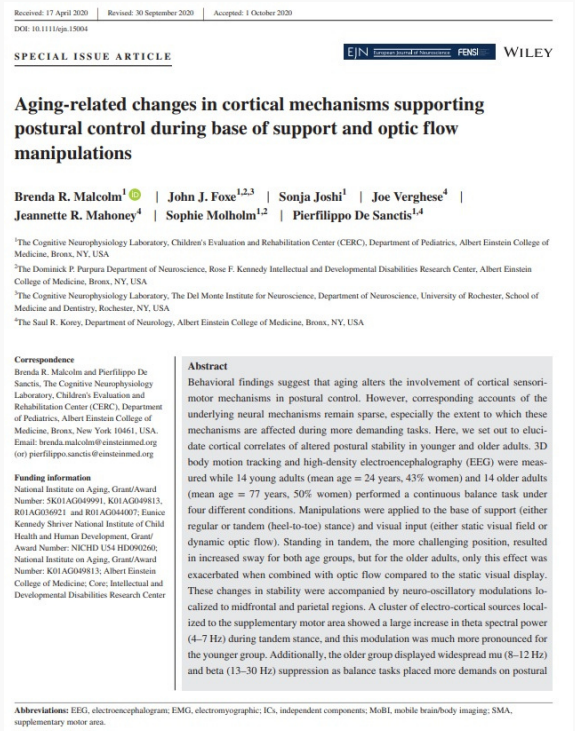
Dr Constantino drafted the initial manuscript, conceptualized and designed the study, secured funding, assumed the role of principal investigator for data collection site, and coauthored the Diagnostic Odyssey data collection instrument; Ms Abbacchi drafted the initial manuscript, coordinated and supervised data collection, and conducted analyses of data; Drs Saulnier and Klaiman drafted the initial manuscript and coordinated and supervised data collection; Dr Mandell drafted the initial manuscript and coauthored the Diagnostic Odyssey data collection instrument; Ms Zhang coordinated and supervised data collection and conducted analyses of data; Ms Hawks Conducted analyses of data; Dr Bates coordinated and supervised data collection; (Continued)

WHAT'S KNOWN ON THIS SUBJECT: African American (AA) children with autism experience racial disparities in timing of diagnosis and access to quality interventions. AA children experience twice the rate of comorbid intellectual disability and higher rates of misdiagnosis of autism compared with non-Hispanic white children.

WHAT THIS STUDY ADDS: These data reveal a 3-year time lag between parental recognition of developmental delay and autism diagnosis among AAs, and that excess intellectual disability burden cannot be explained by ascertainment bias or by traditional familial predictors of cognitive outcome.

To cite: Constantino JN, Abbacchi AM, Saulnier C, et al. Timing of the Diagnosis of Autism in African American Children. *Pediatrics*. 2020;146(3):e20193629

African American and Latino children experience delays in diagnosis of Autism Spectrum Disorder (ASD) in comparison to their Non-Hispanic White (NHW) peers. What's more, these children are more likely to experience comorbid intellectual disability (ID). Therefore, we, in collaboration with other universities across the US, investigated the drivers for these racial disparities by interviewing 584 African American and Afro-Latinx families about their journey to ASD diagnosis. We discovered that while the majority of our families had health insurance, on average, ASD diagnoses were made three years after parents reported their developmental concerns to a healthcare professional, and most children were diagnosed after the age of 4. We further documented that comorbidity of ASD with ID in our sample was not related to family income, prematurity, or the IQ of first-degree relatives, even though these factors have been associated with ID in the general population. Though this study did not identify definitive drivers of racial disparities within ASD diagnosis and ID comorbidity, it concludes that suspected sociodemographic and familial factors cannot fully account for this disparity.



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SPECIAL ISSUE ARTICLE

EJN European Journal of Neuroscience FENS WILEY

Aging-related changes in cortical mechanisms supporting postural control during base of support and optic flow manipulations

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Abstract

Behavioral findings suggest that aging alters the involvement of cortical sensorimotor mechanisms in postural control. However, corresponding accounts of the underlying neural mechanisms remain sparse, especially the extent to which these mechanisms are affected during more demanding tasks. Here, we set out to elucidate cortical correlates of altered postural stability in younger and older adults. 3D body motion tracking and high-density electroencephalography (EEG) were measured while 14 young adults (mean age = 24 years, 43% women) and 14 older adults (mean age = 77 years, 50% women) performed a continuous balance task under four different conditions. Manipulations were applied to the base of support (either regular or tandem (heel-to-toe) stance) and visual input (either static visual field or dynamic optic flow). Standing in tandem, the more challenging position, resulted in increased sway for both age groups, but for the older adults, only this effect was exacerbated when combined with optic flow compared to the static visual display. These changes in stability were accompanied by neuro-oscillatory modulations localized to midfrontal and parietal regions. A cluster of electro-cortical sources localized to the supplementary motor area showed a large increase in theta spectral power (4–7 Hz) during tandem stance, and this modulation was much more pronounced for the younger group. Additionally, the older group displayed widespread mu (8–12 Hz) and beta (13–30 Hz) suppression as balance tasks placed more demands on postural

Abbreviations: EEG, electroencephalogram; EMG, electromyographic; ICs, independent components; MoBI, mobile brain/body imaging; SMA, supplementary motor area.

The goal of this study was to investigate age-related changes in cortical functions that contribute to postural control. Young and older adults performed a series of challenging balance tasks, standing in either regular or tandem (heel-to-toe) stance, while viewing either a static visual image or a moving visual scene (optic flow). Three-dimensional body motion tracking and high-density EEG recorded body sway and neural oscillatory activity, respectively. We found that as the balance tasks became more demanding, the older group exhibited more instability compared to the young adults, and these increases in body sway were accompanied by modulations in neural activity localized to midfrontal and parietal brain regions. These findings may be useful in helping to identify early cortical correlates of balance impairments in otherwise healthy older adults.

Warm Wishes and Happy Holidays!

From all of us at the CNL/HCP, we wish you and your loved ones a very happy and healthy holiday season! Cheers to the new year!

Albert Einstein College of Medicine

The Children's Hospital at Montefiore

The Rose F. Kennedy Children's Evaluation & Rehabilitation Center

The Rose F. Kennedy IDDRC

Montefiore