(WAIS-IV DSB, DSS), visuospatial functioning (JLO), language (VNT), memory (RAVLT Delayed Recall, WMS-IV Logical Memory II), and executive function (TMTB, Stroop Color-Word). Separate moderation analyses were conducted with depression as the predictor and *APOE4* or *BDNFMet* status as the moderator using the SPSS PROCESS macro v4.0. Age was a covariate for models with processing speed, memory, language, and executive function as outcome variables.
Results: Participants were largely male (93%) and White (75%). Ten percent met criteria for depression, 26% were *APOE4* carriers, and 32% were *BDNFMet* carriers. The overall model examining depression, *APOE4*, and memory was significant ($p < .01$, $R^2 = .14$). Depression was associated with lower memory performance ($p < .05$), however, *APOE4* was not a significant moderator ($p > .05$). Similarly, the overall model examining depression, *APOE4*, and language was also significant ($p < .05$, $R^2 = .10$). While the direct effects of depression and *APOE4* on language were nonsignificant ($p > .05$), there was a significant two-way interaction between *APOE4* and depression ($p = .03$). The overall model with depression, *BDNFMet*, and memory was significant ($p < .001$, $R^2 = .18$). While neither depression nor *BDNFMet* had significant direct effects on memory ($p > .05$), a two-way interaction emerged between depression and *BDNFMet* ($p = .05$). Simple slopes analyses were used to further investigate significant interactions. Depression, *APOE4*, and *BDNFMet* did not significantly impact attention, processing speed, working memory, visuospatial functioning, or executive function, and no significant interactions were noted among variables. *BDNFMet* had no direct impact on language.
Conclusions: *APOE4* and *BDNFMet* were found to differentially moderate the relationship between depression and cognition. Specifically, *APOE4* carriers with depression had worse language performance compared to those who were healthy, depressed, or *APOE4* carriers. *BDNFMet* carriers with depression performed worse on measures of memory compared to those who were healthy, depressed, or *BDNFMet* carriers. The treatment of depression in *APOE4* and *BDNFMet* carriers may reduce associated cognitive impairments. Limitations and future implications are also discussed.

Categories: Genetics/Genetic Disorders
Keyword 1: depression
Keyword 2: cognitive functioning
Keyword 3: genetics
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55 The Neurocognitive Profile of a Child with Rubinstein-Taybi Syndrome (RSTS-Type 2)

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Objective: Rubinstein-Taybi Syndrome (RSTS) is a rare multiple congenital autosomal dominant disorder, with an incidence of roughly 1/125,000 live births (Milani et al., 2015). RSTS is characterized by several typical somatic characteristics and developmental disabilities. Common neurological findings in patients with RSTS include mild or moderate intellectual impairment and delays in gross motor development (Taupiac et al., 2021; Hamilton et al., 2016). Additional characteristics observed among individuals with RSTS include hyperactivity, abnormalities in expressive language, inattention, motor difficulties, noise intolerance, maladaptive behaviors, and fewer modes of communication (Waite et al., 2015). Due to the condition's rarity, very few studies have investigated the cognitive profiles of RSTS patients with clinical features of EP300 (Type 2; Morel et al., 2018) in effected youth. This case study represents the first reported comprehensive neuropsychological description to our knowledge of an individual with this condition.
Participants and Methods: Participant: The participant is an 8-year, 4-month-old young girl referred for neuropsychological evaluation. LX was diagnosed with failure to thrive due to her small size, although she met all developmental milestones on time. LX was diagnosed with RSTS, Type 2 through genetic testing and blood work following concerns about small stature, microcephaly discovered on an MRI, and feeding difficulties. A 4kb deletion of 22q13.2 which contains exon 2 of EP300 was identified. Method: Medical and school records review, a clinical interview with LX and her family, neuropsychological assessment, and parent- and teacher-report questionnaires were used to

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assess functioning. Raw scores were standardized for comparison against age- and (where appropriate) gender-matched peers. **Results:** In addition to her medical diagnoses of RSTS, Type 2, and her prior diagnosis of ADHD – Combined Subtype, LX also met diagnostic criteria for Specific Learning Disability with Impairment in Reading and a Mixed Receptive-Expressive Language Disorder. LX also met criteria for Encephalopathy as characterized by weakness in visual perception, visuospatial judgment and reasoning, and working memory. LX demonstrated adaptive functioning weaknesses in domains such as self-direction, and self-care, while communication skills were reported to be average. Overall, LX's current levels of general cognitive ability and adaptive functioning were consistent with Borderline Intellectual Functioning; however, the diagnosis was deferred at present. Parents and teachers reported difficulties with peer relationships, hyperactivity, and aggression, consistent with known features of this condition. Strengths were noted in verbal and nonverbal reasoning, spelling, math calculation, verbal and visual memory, and improvement in attention with medication, which all fell within the broadly average range of functioning. **Conclusions:** LX's presentation and pattern of neuropsychological findings are consistent with the current conceptualization of development in RSTS, Type 2, but reflect a more nuanced clinical picture. In particular, although general cognitive ability was borderline overall, deficits were largely circumscribed to spatial reasoning, with broadly average verbal and nonverbal reasoning abilities. This case highlights the importance of comprehensive neuropsychological testing of patients with RSTS. Reporting of general cognitive ability scores alone may obscure underlying patterns of relative strengths and weaknesses that have important ramifications for both targeted interventions and for a more positive prognosis related to functioning in academic, home, and community environments.

Categories: Genetics/Genetic Disorders
Keyword 1: neuropsychological assessment
Keyword 2: cognitive functioning
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56 Genetic Models for Long-Term Neurocognitive Outcomes in Pediatric Medulloblastoma and Traumatic Brain Injury

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Objective: Children who suffer from brain insults (i.e., traumatic brain injury (TBI), chemotherapy and radiation treatment for brain tumors) are susceptible to late-emerging cognitive sequelae. Even with similar neurological risk variables, variability in long-term cognitive outcomes remains an area of investigation for researchers of acquired brain injury. Given the potential for genetic factors to influence response to chemoradiation, researchers have examined associations between germline, inherited, single nucleotide polymorphisms (SNPs), and neurocognitive outcomes for cancer survivors. Children who sustain an uncomplicated mild TBI generally recover without long-term neuropsychological consequences. However, TBI survivors have overlapping mechanism categories with cancer survivors through secondary injury variables that can be influenced by genomic variation (e.g., oxidative stress and neuroinflammation). Furthermore, the study of genomic vulnerability is limited in heterogenous groups of pediatric TBI survivors. This study aims to identify associations between genotype and long-term neurocognitive outcomes for acquired brain injury survivors by utilizing machine learning to uncover pathophysiological similarities and differences between groups.
Participants and Methods: Fourteen brain tumor survivors, 139 traumatic brain injury survivors, and 63 healthy, age-matched controls completed the Letter N-back task to obtain performances on core neurocognitive skills (attention, working memory, and processing speed). Ten targeted genotypes were examined across five pathophysiological pathways (neurotransmission, oxidative stress, neuroinflammation, plasticity, growth and repair, and folate metabolism). Data were trained and tested utilizing three regression machine learning models. Mean estimated error and R^2 were generated for each neurocognitive

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