Title: Distinguishing Social Motivation from Autism Features in Children with Neurogenetic Syndromes

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Introduction: Elevated rates of challenging behaviors such as autism spectrum disorder symptoms (ASD) are common in individuals with neurogenetic syndromes (NGS), including Angelman (AS; 63%), Prader-Willi (PWS; 23%), and Williams (WS; 50%) syndromes. ASD prevalence in these populations is highly elevated compared to 2% in the general population and 20% in siblings of a child with ASD (Baio et al., 2018; Betancur & Coleman, 2013; Ozonoff et al., 2011). Specifically, several studies have suggested NGS subgroups exhibit intact or less impaired social motivation, however, few studies have empirically distinguished them from idiopathic (e.g. non-syndromic) ASD. As such, the present study tested whether informants describe children with NGS as displaying more typical social motivation relative to children with idiopathic ASD. Together, these findings aimed to clarify the social motivation profiles of NGS relative to ASD, as well as the within-group features that may moderate associations (e.g. NGS subgroup, functional impairment), preliminarily informing whether and how social motivation may inform differential diagnosis in NGS and other populations associated with ID.

Method: We administered the Social Responsiveness Scale 2nd Edition to caregivers of 91 children with NGS as part of an online, longitudinal study of early development and compared data to 45 children with ASD and similar cognitive skills drawn from the National Database for Autism Research (NDAR). We used nonparametric tests to test if (1) social motivation in NGS, and NGS subtypes, is less impaired compared to ASD with similar cognitive skills, and (2) social motivation is similar across ASD severity within NGS. We also conducted exploratory analyses to probe associations between social motivation and impairment, as measured by the Vineland Adaptive Behavior Scales 3rd Edition, as well as to test specificity of findings relative to other social constructs such as social communication, awareness, and cognition.

Results: Social motivation was less impaired across NGS subtypes and ASD symptom severity when compared to idiopathic ASD ($p$’s < .05 with Holm Bonferroni corrections). Further, social motivation was not associated with functional impairment within NGS ($p = -.20, p = .114$). In contrast to social motivation – which was similar across NGS regardless of ASD severity and NGS subtype – the remaining social features demonstrated unique patterns across both ASD severity and NGS subtype ($p$’s < .05 with Holm Bonferroni corrections).

Discussion: We generated three key findings: (1) social motivation was significantly less impaired in NGS irrespective of subtype and ASD symptom severity, (2) social motivation was not associated with impairment, and (3) social motivation exhibits a different profile than other social features in young children with NGS. Results preliminarily support previous reports that social motivation may operate as a relative strength for many children with NGS. Thus, tests of social motivation may not be particularly useful in differential diagnosis of ASD within NGS.

References:


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Page 1 of 1