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Research Article

Social Cognition in Adolescents With Gender Dysphoria and Congenital Adrenal Hyperplasia: A Preliminary Investigation of Biological vs. Experiential Gender Effects

Yulug-Tas B et al. Social Cognition at the Crossroads of Gender Biology and Gender Experience

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What is already known on this topic?
It is well-established that prenatal androgens affect social cognition skills. Additionally, individuals with gender dysphoria often exhibit deficits in social cognition, with accompanying psychiatric comorbidities influencing these impairments.

What this study adds?
A key strength of this study lies in its approach to social cognition skills, examining them from both perceptual and cognitive perspectives while incorporating a neurodevelopmental framework. The homogeneity of the participant groups represents a positive aspect; however, the cross-sectional design and small sample size impose limitations on the generalizability of the findings.

Abstract
Objective: This study aims to explore hormonal and neurodevelopmental influences on social cognition among individuals with Gender Dysphoria (GD), Congenital Adrenal Hyperplasia (CAH), and typically developing (TD) controls.
Method: Participants included 34 GD, 29 CAH, and 35 TD individuals. Social cognition was assessed using the Faces Test (FT), Reading the Mind in the Eyes Test (RMET), and Unexpected Outcomes Test (UOT). Psychiatric comorbidities were evaluated via the Kiddie-Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS-PL), depressive symptoms using the Children's Depression Inventory (CDI), autistic traits with the Autism Spectrum Screening Questionnaire (ASSQ), and ADHD symptoms through the ADHD Rating Scale.
Results: Psychiatric diagnoses were significantly more prevalent in the GD group, with Major Depressive Disorder (64.7%) and ADHD (50%) being the most common ($p<0.001$). TD participants showed moderately better performance on RMET ($p=0.003$) and UOT ($p<0.001$) compared to GD and CAH, while CAH individuals scored lower on FT ($p=0.046$). Regression analyses revealed depressive symptoms ($B=-0.105$, $p=0.004$) and CAH status ($B=-2.221$, $p=0.003$) predicted RMET scores, while GD ($B=-3.232$, $p=0.022$) and CAH ($B=-7.974$, $p<0.001$) predicted lower UOT performance. FT regressions were nonsignificant.
Conclusions: Findings highlight the interplay of hormonal and psychosocial factors in social cognition, emphasizing the need for nuanced, context-sensitive approaches to supporting social functioning and well-being in gender-diverse youth.
Keywords: Androgen exposure; congenital adrenal hyperplasia; gender dysphoria; neurodevelopmental disorders; social cognition; theory of mind

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INTRODUCTION

'Sex' is a phenotype influenced by chromosomes, reproductive anatomy, and sex steroids (1). Sex differentiation occurs during early intrauterine stages and concludes in the first days of life (2). Gender identity is the personal acceptance of one's gender, independent of genetic or societal influences (3), while gender dysphoria (GD) refers to the incongruence between assigned sex at birth and experienced gender (4). The development of GD has been linked to genetic (5), hormonal (6), and neurobiological factors (7). Although not directly associated with congenital adrenal hyperplasia (CAH)—a condition involving prenatal androgen exposure (8)—testosterone exposure is linked to reduced theory of mind skills, such as understanding emotions and empathy (9), which are also impaired in individuals with GD.
Gender identity is a core aspect of personal and social identity (10), while social cognition involves mental processes related to social interactions (11). Social cognitive skills are measured by theory of mind, which includes social cognition (interpreting others' behavior) and social perception (awareness of others' mental states through observable information) (12). Impairments in these skills are characteristic of autism (13) and other conditions such as anorexia nervosa (14) and ADHD (15).
Testosterone affects emotions, behaviors, and cognition, with high levels influencing social interactions (16). A 2017 study found a negative relationship between prenatal testosterone levels and social interaction skills (17). Despite not having hormonal imbalances, individuals with GD show more significant social-cognitive differences compared to the general population (18), leading to social rejection, bullying, and discrimination (19). A 2011 study found that 72% of individuals with GD, with no psychiatric co-occuring disorders, experienced suicidal

thoughts (20). The prevalence of autism is higher in those with GD, with 6.4% of children, 7.8% of adolescents, and 5.5% of adults diagnosed with GD also having autism (21). The reasons for this co-occurrence remain unclear, with theories such as the hyper-masculinized brain theory and social exclusion being discussed (22).

Thus, considering the complex interaction of neurodiversity, gender and hormonal influences, this study aims to explore how different hormonal and neurodevelopmental pathways might intersect with social cognition outcomes in adolescence. Specifically, we compared three groups: (1) adolescents assigned female at birth diagnosed with GD, presumed to have developed under typical hormonal conditions but experiencing significant gender-related psychosocial stress; (2) adolescents with CAH, exposed to elevated prenatal androgens but without gender dysphoria; and (3) adolescents assigned female at birth, developing under typical hormonal conditions without gender dysphoria. Rather than viewing these groups as strict representations of "biological" versus "experiential" gender factors, we approached them as differing developmental contexts in which social cognition may be shaped. Our primary aim was to examine group-level differences in theory of mind performance and autistic traits, and to explore whether any observed differences could be explained by affective, neurodevelopmental, or other variables such as depressive symptoms, attention, and autistic features.

METHODS

Participants and study design

The cross-sectional study was conducted in collaboration with the Departments of Child and Adolescent Psychiatry and Pediatric Endocrinology at Ege University, following ethical standards outlined in the Declaration of Helsinki and approved by the Ege University Clinical Research Ethics Committee (decision no: 22-12T/56). All children and their parents were informed about the study, and written informed consent—or verbal assent when written consent was not feasible—was obtained.

Participants, aged 11-18, were selected as this age range is optimal for diagnosis and scale reliability. All participants were assigned female at birth. Exclusion criteria included developmental delay, neurological conditions, and intellectual disabilities.

Gender Dysphoria Group

Participants were recruited from the clinical registry of individuals who had previously presented to the child and adolescent psychiatry clinic and disclosed gender-related distress. From this registry, 49 eligible individuals were invited to participate. During the intake interview, six no longer met DSM-5 criteria for GD and were excluded. An additional nine participants identified as nonbinary were excluded due to the study's focus on androgen exposure and male gender identity (i.e., assigned female at birth with current male identification). GD diagnoses were confirmed through structured clinical interviews conducted by a certified child and adolescent psychiatrist and confirmed by a senior child and adolescent psychiatrist with over 30 years of clinical experience and extensive expertise in gender-related care and neurodevelopmental disorders. All participants had normal hormonal profiles, confirmed via Pediatric Endocrinology evaluation.

Congenital Adrenal Hyperplasia Group

The comparison group consisted of 29 individuals with CAH who were recruited through Pediatric Endocrinology outpatient clinics and had no reported GD. Individuals with a diagnosis of autism were excluded from both the GD and CAH groups to avoid confounding effects when assessing autistic traits.

Typically Developing Comparison Group

A third comparison group included 35 females, assigned female at birth, with age-appropriate secondary sex characteristics, no endocrine abnormalities, and no medical or neurodevelopmental conditions. Participants were recruited through advertisements posted throughout Ege University and via snowball sampling. All applicants underwent endocrinological screening to confirm the absence of hormonal abnormalities, and the absence of psychiatric disorders was verified through the K-SADS-PL clinical interview.

Procedure

All participants and their parents were interviewed in person, and sociodemographic data, including ages and educational levels, were collected. Comorbid psychiatric disorders were assessed using the Kiddie-Schedule for Affective Disorders and Schizophrenia (K-SADS-PL) by a certified child and adolescent psychiatrist. IQ was informally assessed through clinical interviews, developmental milestone reviews, and academic performance (e.g., school grades), offering a general understanding of cognitive abilities.

The Global Assessment Scale (GAS) was used to evaluate the impact of comorbid psychiatric symptoms on functionality. Social cognition skills were assessed using the Faces Test (FT), Reading the Mind in the Eyes Test (RMET), and the Unexpected Outcomes Test (UOT). Additionally, participants completed the Children's Depression Inventory (CDI), while parents filled out the Autism Spectrum Screening Questionnaire (ASSQ) and the ADHD Rating Scale.

Measures

Sociodemographic Data Form (SDF): The SDF was developed to gather information about the sociodemographic characteristics of the groups. It includes variables such as the age and educational levels of the participants and their parents.

Kiddie-Schedule for Affective Disorders and Schizophrenia for School-Age Children - Present and Lifetime Version (K-SADS-PL): This form is used to evaluate lifetime comorbid psychopathologies in children. If diagnostic symptoms are identified during the initial interview, an additional evaluation checklist is administered. The presence and severity of positive findings are determined based on the clinician's, family's, and participant's input. The standardization of the form has been conducted for Turkish children (23).

Global Assessment Scale (GAS): The GAS evaluates variables such as the level of illness, social and occupational functioning, and coping mechanisms for adverse conditions, providing a measure of the individual's overall well-being and functionality. Developed by the creators of the K-SADS, the scale assigns a score between 0-100 based on the clinician's assessment, as described in the literature. A higher score indicates better overall well-being and functionality (24).

Faces Test (FT): The Faces Test, developed by Ekman in 1972, evaluates social perception skills by measuring participants' ability to recognize emotions. The test involves showing participants 60 photographs depicting six basic facial expressions (happiness, sadness, anger, disgust, surprise, fear). Participants are asked to identify the emotion represented in each photograph, one at a time, without time restrictions. Responses are scored on a 0-1 scale and compared with a control group (25). The Turkish version of the FT has been validated and standardized (26).

Reading the Mind in the Eyes Test (RMET): RMET aims to measure the ability of the participant to recognize emotions and infer the meaning beyond them by observing the eyes in a photograph (27). Initially developed by Baron-Cohen and colleagues in 1997, it was revised and improved in 2001 to assess the social perception component of social cognition as identifying the emotional state of another person, solely from the eyes, without the additional cues provided by the rest of the face, presents a significant challenge. It consists of 28 photographs showing only the eye region, each accompanied by four response options. The goal is to observe subtle aspects of an individual's theory of mind. The Turkish version has been validated and standardized (28).

Unexpected Outcomes Test (UOT): The UOT is designed to assess participants' social cognitive theory of mind abilities by presenting 12 short scenarios. In each scenario, participants are asked to infer what the individuals depicted might be feeling or thinking based on the context (29). The responses to the questions are scored between 0 and 2, with a total score of 24. If a participant answers incorrectly three consecutive times,

the test is terminated. Research has demonstrated that the UOT correlates well with other measures of social cognition skills (30). The Turkish version has been validated and standardized (31).

Children's Depression Inventory (CDI): The CDI is a self-assessment scale developed to evaluate depressive symptoms in childhood. It is intended for children aged 6-17 and is based on the Beck Depression Inventory. The scale has been standardized for Turkish children (32).

Autism Spectrum Screening Questionnaire (ASSQ): The ASSQ consists of 27 statements and is used as a screening tool to identify and initiate early intervention for Autism Spectrum Disorder (ASD). A parent-report form initially designed to screen for Asperger's Syndrome in school-age children, it was later renamed under the broader category of autism and recognized as a reliable screening tool in assessing autistic traits (33). Köse and colleagues have established the validity and reliability of the scale in the Turkish population with a cut-off of 16 or higher being considered highly indicative of ASD in distinguishing clinical groups from controls (34).

ADHD Rating Scale: This scale comprises 41 items across four subdomains, designed to assess ADHD and its two frequent comorbidities, Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD) in alignment with its description and criteria outlined in the DSM-IV. Responses are filled out by the caregivers and scored on a scale from 0 to 3. The standardization of the scale for Turkish participants was conducted by Ercan and colleagues (35).

Statistical Analysis

Statistical analyses were conducted using IBM SPSS version 25.0. The normality of the data was assessed using the Shapiro-Wilk test, histograms, and Skewness-Kurtosis coefficients. Differences in CDI, ASSQ, ADHD scales, RMET, FT, and UOT scores among the GD, CAH, and TD groups were evaluated using the Kruskal-Wallis test. Post-hoc pairwise comparisons between groups were performed with Dunn's test. Nominal data were compared using the Pearson chi-square test. Bonferroni correction was applied for all cases of multiple comparisons. Pairwise comparisons were conducted only for variables with statistically significant group differences in the omnibus test ($p < .05$). No post-hoc tests were performed for non-significant results. The relationships between the dependent variables (RMET, FT, and UOT total scores) and predictors such as gender dysphoria, congenital adrenal hyperplasia diagnosis, and other social cognition factors that differed between groups were analyzed through linear regression. All statistical tests were two-tailed, with significance set at $p < .05$.

RESULTS

The mean age of the participants were 14.98±2.05.There were no statistically significant differences between participants' ages and the ages of their parents ($p>.05$). A difference in maternal education was observed ($p=.001$). In the pairwise comparisons with Bonferroni correction, it was found that this discrepancy was due to the higher observed prevalence of tertiary education ($p=.012$) and lower observed prevalence of primary education than expected ($p=.001$) among TD mothers. The observed difference in paternal education ($p=.030$) revealed no statistically significant differences between the groups after the Bonferroni correction (Table 1).

The psychiatric diagnoses in the GD group were significantly higher compared to the CAH group. ($p<0.001$). Major Depressive Disorder was diagnosed in 64.7% of the GD group, followed by ADHD (50%). All psychiatric diagnoses are summarized in Table 2, with generalized anxiety disorder, social anxiety disorder, and separation anxiety disorder identified through the K-SADS-PL being grouped under the category of "Anxiety Disorders".

The GD group received the lowest total score on the GAS, while the controls received the highest score ($p<0.001$). The CDI total score ($p<0.001$) and the Inattention Subscale of the ADHD Scale ($p=.004$) were significantly higher in the GD group compared to the other groups ($p<0.001$). The GD group also had higher scores in the ASSQ compared to TD ($p=.003$). The psychiatric diagnoses, psychiatric symptomatology and related scales are summarized in Table 3.

Among the social cognition tests, significant differences were observed in the RMET scores ($p= .003$). Pairwise comparisons showed that the TD group had higher scores compared to both the GD and CAH groups, with no significant difference between GD and CAH. The FT test also showed a significant difference ($p = .046$), with the CAH group scoring lower than the TD group (Table S1). However, there were no significant differences between groups on the six FT subscales, which correspond to the basic emotions of surprise, happiness, anger, sadness, disgust, and fear. Additionally, there were highly significant differences in the UOT test among the groups ($p < .001$), with both GD and CAH groups scoring lower than the TD group, but no significant difference between GD and CAH (Table 4).

Three separate linear multiple regression models were analyzed to control for variables that differed between groups and might explain the variations in social cognition test results for RMET, FT, and UOT. The GD and CAH groups were dummy-coded, and ASSQ, CDI, and Inattention scores were included in all three models to account for autistic traits, depressive symptoms, and inattention, which could potentially impact social cognition. The first model for RMET yielded significant results with $F(5,92)=4.441$, $p=0.001$, $R^2=0.194$, and an Adjusted R^2 of 0.151. CDI scores ($B=-.105$, $p=.004$, CI=-.175 to -.034) and CAH diagnosis ($B=-2.221$, $p=.003$, CI=-3.669 to -.773) were identified as significant negative predictors of RMET scores. In contrast, the second model for the Faces Test was not significant, with $F(5,92)=1.694$, $p=.144$, $R^2=0.084$, and an Adjusted R^2 of 0.035. The third model for UOT was significant with $F(5,92)=8.144$, $p<0.001$, $R^2=0.307$, and an Adjusted R^2 of 0.269. For UOT, being in the CD group ($B=-3.232$, $p=.022$, CI=-5.988 to -.476) and the CAH group ($B=-7.974$, $p<.001$, CI=-10.542 to -5.405) were significant negative predictors. The significant results for these two models are summarized in Table 5.

Although the CAH (androgen-exposed) group had significantly lower scores on the Faces Test in group comparisons, regression analysis did not yield a significant overall model. While androgen exposure emerged as a statistically significant individual predictor ($B = -3.096$, $p = .030$), this should be interpreted cautiously, as the model itself did not reach significance, ($F(5, 92) = 1.694$, $p = .144$) summarized in Table S1.

DISCUSSION

Our study revealed significant findings, suggesting a potential impact of hormonal and experiential variations on social cognition. First, individuals in the CAH group demonstrated significant social cognition deficits on the RMET and UOT tests, suggesting a potential biological influence of prenatal androgen exposure. Although a statistically significant group difference was found in the total score of the FT, this result should be interpreted with caution, as none of the emotion-specific subscales showed significant group differences, and FT performance was not predicted by any group or clinical variables in regression analyses.

Taken together, these findings suggest that individuals with CAH may experience difficulties in both affective and cognitive aspects theory of mind, specifically in interpreting and predicting social outcomes and attributing complex emotional states to others, while basic emotion recognition abilities remain intact.

Second, the GD group exhibited deficits in social perception and cognitive theory of mind abilities "as evidenced by their lower scores in UOT and RMET compared to the TD group. However, the key finding and a potential explanatory link is that depressive symptoms emerged as a significant predictor of RMET performance. Although GD status was not itself a predictor, the GD group had elevated depressive symptoms, suggesting that their lower RMET scores may be attributable to affective distress rather than gender identity alone. Notably, the TD group consistently outperformed both the CAH and GD groups across all social cognition measures, highlighting the complex interplay of hormonal, neurodevelopmental, and societal influences on social cognition.

Neurodevelopmental Perspective

This study aimed to compare two neurodevelopmentally diverse groups—those with experiential or hormonal variations in gender norms, namely the GD and CAH groups, who are known to face challenges in social cognition—with a comparison group exhibiting typical neuroendocrine development. Neurodevelopment is strongly shaped by endocrine status in conditions such as CAH, however, it also follows its own trajectory in individuals without endocrine differences, as seen in the GD group. As such, we also sought to interpret the factors influencing social cognition abilities, approaching the social cognition construct from both a social perception and a social cognitive ability perspective, while also identifying neurodevelopmental parameters that could potentially influence these skills, such as neurodevelopmental disorders, traits, and comorbid psychiatric conditions.

To that end, the three groups were first compared regarding their psychiatric disorders, as well as ADHD and autistic traits. The GD group had significantly more psychiatric disorders, such as major depressive disorder (MDD) and ADHD-inattention, which is in line with the extant literature (36). The GD group also exhibited more autistic traits in group comparisons.

The rationale for assessing neurodevelopmental disorders, traits, and psychiatric conditions was straightforward, as challenges in social cognition skills are often difficult to separate and interpret independently from other executive functions (37). This approach facilitated the disentanglement of which observed differences are more likely driven by hormonal biology and which reflect broader neurodevelopmental or psychiatric processes. For instance, the ASSQ, which was utilized in our study to identify autistic traits, does not directly or indirectly measure social cognition but does capture social difficulties in autism. However, the social challenges identified by this scale may arise from a range of factors, with theory of mind deficits being only one possible contributor. As such, the ASSQ was included in the regression models to control for trait-level neurodevelopmental variation. Beyond autistic traits alone, psychiatric comorbidities such as major depressive disorder and anxiety disorders also affect social cognition. A review suggests that individuals with depression tend to exhibit a mood-congruent bias toward social stimuli, meaning they interpret cues in a way that aligns with their depressive mood, and face difficulties in the cognitive aspects of theory of mind (38). Additionally, anxiety and depressive symptoms are reported to influence the relationship between autistic symptomatology and social cognition and adjustment in children diagnosed with ASD, further emphasizing the role of comorbidities in social functioning (39).

Biological Influences: The Role of Androgens

Building on the neurodevelopmental framework, the deficits observed in the CAH group provide insights into how prenatal androgen exposure impacts social cognition. Secondary analyses comparing social cognition across groups revealed that the typically developing comparison group outperformed both the CAH and GD groups on the RMET and UOT tests, and only the CAH group on the FT test. These findings align with existing literature (40) and are particularly interesting given the role of androgens in emotional processing and aggression (41).

While these results suggest significant differences between groups, further investigation into psychiatric comorbidities, such as depression and inattention identified during preliminary comparisons—was necessary. In regression analyses controlling for inattention, depression, and autistic traits, membership in the CAH group emerged as a significant predictor. This may reflect the biological effects of androgens on social cognition and emotional processing. However, the lack of formal IQ testing in this study means that the observed differences could also be related to the cognitive challenges associated with CAH, highlighting the need for further research using standardized IQ assessments or other cognitive measures.

An alternative explanation for CAH as a predictor in the RMET and UOT is the direct effect of androgens on social cognition (42). Research has shown that individuals with higher fetal testosterone levels score lower on the RMET, which tests theory of mind (43). This test assesses social perception skills, crucial for understanding and adapting to others' perspectives (44). As emotion recognition and social perception are closely related (45), our findings further support the role of hormonal influences in shaping social cognition.

These findings highlight the hormonal pathway into neurodevelopmental outcomes and that endocrine processes may influence social cognitive development.

The GD "Experience"

The biological effects of androgens on social cognition provide one perspective, but the GD group's results highlight how affective, neurodevelopmental, and experiential factors may also influence social perception and theory of mind abilities. Notably, while the GD group scored significantly lower than the TD group on the RMET in group comparisons, GD status did not emerge as a significant predictor in the regression model. Instead, CDI scores were a negative predictor of RMET performance in the model. This suggests that the lower social cognition scores in the GD group may be more influenced by depressive symptoms than by autistic traits or gender dysphoria itself, emphasizing that the issue is more about "dysphoria" than "gender" (46). An alternative perspective highlights studies suggesting that performance on the RMET is negatively associated with autistic traits and may be indirectly related to the severity of gender dysphoria. (47). However, it is particularly important to note that this study did not account for depressive symptoms. In contrast, our findings indicate that when depression is controlled for, the association between autistic traits and RMET performance becomes non-significant, offering an alternative interpretation of the underlying mechanisms. The RMET does not solely assess theory of mind abilities; it also involves broader psychological processes such as cognitive speed, attention, motivation, and facial expression processing. This aligns with previous studies reporting that, even in the absence of pronounced deficits on the RMET, individuals with depression often exhibit overall impairments in social cognition (48). Furthermore, there is evidence suggesting that depressive symptoms negatively affect social cognitive performance (49).

This distinction is crucial for a group already facing marginalization, as difficulties in social cognition may further hinder social integration and peer relationships (50). Addressing depressive symptoms in adolescents with GD may reduce social anxiety, making it easier for them to connect with typically developing peers (51).

From a developmental standpoint, gender identity and social cognition evolve in parallel throughout childhood and adolescence. A central feature of theory of mind is the ability to distinguish between external appearance and internal reality which is also relevant for navigating identity and societal expectations (52). While none of the participants met the diagnostic criteria for autism, their ASSQ scores suggested a greater presence of autistic traits. Prior research has investigated whether features such as intense interests or perspective-taking difficulties, often seen in autism, may interact with the development of gender dysphoria (53). Indeed, autistic traits have been found to be overrepresented in individuals with GD, pointing to a complex, but not necessarily causal, relationship between gender diversity, neurodivergence, and social cognition (21).

However, our regression analyses suggest that these traits alone do not account for the observed differences in social cognition. Specifically, GD status was a significant predictor of UOT performance even when depressive symptoms, inattention, and autistic traits were controlled for. This distinguishes UOT from RMET, where depressive symptoms explained the variance. Given that the UOT taps into pragmatic theory of mind, such as inferring how beliefs guide future actions, RMET primarily measures social perceptual abilities in the affective ToM domain and FT targets emotion recognition; the altered response in UOT may reflect a unique cognitive-emotional adaptation shaped by lived experience in gender-diverse individuals.

In contrast to the CAH group, where social cognition differences may reflect hormonal factors, the GD group's performance pattern may have been influenced by the psychosocial context. The finding that both RMET and UOT were predicted by CAH reinforces the role of biological

variables, while the UOT specific association with GD may suggest an experiential component, as both neurodivergence traits and depressive symptoms were accounted for in the regression model. Although the present cross-sectional study did not measure interactions with social norms directly, and as such can not infer causality, the results raise important questions about how long-term navigation of societal expectations might shape social cognition in ways that are not captured by trait-level neurodevelopmental metrics (54).

The contrast with CAH is of particular importance, as endocrine status appears to modify developmental trajectories in CAH, whereas in GD, divergent pathways seem to arise primarily through psychosocial and neurodevelopmental mechanisms in the absence of hormonal variation. Collectively, these findings highlight that social cognition is shaped through both biological and experiential influences, which may converge or remain distinct depending on the context.

For example, individuals with GD may process social scenarios differently, albeit not incorrectly, based on their personal histories and identity development. This could explain altered performance on UOT, which involves anticipating others' behavior in ambiguous contexts. The interpretation of this altered performance in UOT, hence, might be only that, an alteration, rather than a deficit. As such, these results highlight the limitations of conventional social cognition tasks, which often rely on majority-norm assumptions and may not fully capture the breadth of human diversity.Labeling social cognition responses as "right" or "wrong" may reflect biases rooted in societal norms, marginalizing minority populations like those with GD or autism (55).

This perspective aligns with emerging neurodiversity frameworks, which argue that cognitive divergence, including that seen in GD, autism and many others, should not be pathologized but understood as reflecting different, equally valid ways of engaging with the world. Autistic individuals, for example, often interpret the world in ways that diverge from conventional norms (56). Similarly, binary thinking embedded in societal norms may not align with the experiences of those with GD as they may interpret and respond to social situations in ways shaped by their unique experiences with gender identity and social marginalization.

Rather than expecting neurodiverse individuals to adjust to rigid standards of "correct" social cognition, it may be more constructive for neurotypical society to develop greater cognitive flexibility. This shift can foster more inclusive environments that reduce stigma, promote understanding, and support diverse modes of social engagement. By framing differences in social cognition as contextually shaped rather than inherently impaired, we move toward a more compassionate and accurate understanding of human variation.

STRENGTHS AND LIMITATIONS

A key strength of this study lies in its approach to social cognition skills, examining them from both perceptual and cognitive perspectives while incorporating a neurodevelopmental framework. The homogeneity of the participant groups represents a positive aspect; however, the cross-sectional design and small sample size impose limitations on the generalizability of the findings.

The present study refers to androgen exposure but does not measure the degree or duration of such exposure. While categorical differences were observed, the precise extent of these differences remains undetermined. Additionally, the sample was limited to individuals assigned female at birth, and no comparison group assigned male at birth was included. This limits the ability to generalize findings across sexes with different physiological androgen exposure. Moreover, the absence of a control group with a male gender identity but without gender dysphoria prevents disentangling the effects of gender identity from those of dysphoria itself. Consequently, the observed effects cannot be attributed solely to androgen exposure or dysphoria, although the findings offer meaningful insights into social cognition in individuals with XX chromosomes who experience atypical gender development.

Furthermore, while the GD and CAH groups were recruited from clinical settings, the TD comparison group was drawn from the general population through school and community advertisements. This difference in recruitment sources may introduce sampling bias, particularly in terms of psychiatric morbidity or access to care. Although structured psychiatric interviews (K-SADS-PL) were used to ensure diagnostic consistency, the context of referral remains a potential confounder.

Social cognition is heavily influenced by cognitive abilities, including intelligence. The lack of a formal IQ assessment represents a significant limitation of this study, particularly when considering the potential impact of steroids on cognitive development, especially in the CAH group (57). Although clinical evaluations of intelligence were conducted, they were not formalized. Given the established role of intelligence as a predictor of social cognition, larger-scale studies incorporating formal IQ measurements and integrating these into explanatory models are necessary.

As such, this study does not offer a definitive explanation or modeling of social cognition but rather a springboard for larger-scale studies and future research that integrate hormonal and neurodevelopmental factors in this field.

CONCLUSION

This study highlights the distinct yet interconnected roles of biological and societal influences on social cognition. By addressing both the biological challenges faced by CAH individuals and the societal pressures experienced by GD individuals, tailored interventions can foster improved social integration and well-being for these populations.

Social cognition skills are far more complex than they may seem, influenced by multifactorial interactions. In our study, prenatal androgen exposure represented by the CAH group emerged as the primary factor disrupting social cognition skills. The presence of social deficits in the CAH group, despite the absence of autistic traits, suggests a biological influence. In contrast, in the GD group, social perception skills may be influenced by accompanying conditions such as depression and neurodevelopmental traits (e.g., ADHD comorbidity), while cognitive abilities - social or otherwise- appear to be shaped by their unique way of perceiving and experiencing the world.

Individuals with CAH are typically monitored by a multidisciplinary treatment team from early developmental stages, which may contribute to their lower rates of psychiatric comorbidities. In contrast, GD individuals often seek mental health services later in adolescence, after attempting to navigate and adapt to a world that struggles to accept differences. This delayed access to support can result in higher exposure to psychiatric diagnoses.

Therefore, it is crucial to adopt not only a diagnostic but also a transdiagnostic approach when addressing these cases. These findings underscore the need for mental health interventions that address both biological and societal influences on social cognition, with tailored approaches for CAH and GD populations that emphasize early support and societal acceptance.

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Table 1. Sociodemographic data of the participants with GD, CAH, and TD

	GD (n=34)	CAH (n=29)	TD (n=35)	H/ $\chi 2$	p
Age	15.59±1.54	16.34±2.66	14.91±1.77	4.677	.096
Maternal age	45.97±6.13	42.72±6.45	44.23±5.81	4.012	.135
Maternal education Primary Secondary Tertiary	 15 (%44.1) 6 (%17.6) 13 (%38.2)	 15 (%51.7) 4 (%13.8) 10 (%34.5)	 2 (%5.7) 7 (%20) 26 (%74.3)	19.199	**.001**
Paternal age	50.24±6.55	46.03±7.23	48.06±6.20	4.540	.103
Paternal education Primary Secondary Tertiary	 15 (%44.1) 5 (%14.7) 14 (%41.2)	 9 (%31) 5 (%17.2) 15 (%51.7)	 4 (%11.4) 12 (%34.3) 19 (%54.3)	10.649	**.030**

GD = Gender Dysphoria, CAH = Congenital Adrenal Hyperplasia, TD = Typically Developing

Table 2. Psychiatric diagnoses and psychiatric symptomatology across GD, CAH and TD groups.

	GD (n=34)	CAH (n=29)	TD (n=35)	H/ $\chi 2$	p
Psychiatric Disorder (n, %)				10.708*	**.001**
No	3 (8.8)	13 (44.8)	35 (100)		
Yes	31 (91.2)	16 (55.2)	0 (0)		
ADHD					
No	17 (50)	2 (86.2)	35 (100)		
Yes	17 (50)	4 (13.8)	0 (0)		
MDD					
No	12 (35.3)	25 (86.2)	35 (100)		
Yes	22 (64.7)	4 (13.8)	0 (0)		
OCD					
No	34 (100)	28 (96.6)	35 (100)		
Yes	0 (0)	1 (3.4)	0 (0)		
Anxiety Disorder					
No	27 (79.4)	21 (72.4)	35 (100)		
Yes	7 (20.6)	8 (27.6)	0 (0)		

BPD					
No	33 (97.1)	29 (100)	35 (100)		
Yes	1 (2.9)	0 (0)	0 (0)		

*TD group was excluded from the analysis as the inclusion criteria for the TD group was the absence of any psychiatric disorders
GD = Gender Dysphoria, CAH = Congenital Adrenal Hyperplasia, TD = Typically Developing
ADHD = Attention-Deficit/Hyperactivity Disorder, MDD = Major Depressive Disorder, OCD = Obsessive Compulsive Disorder, BPD = Bipolar Disorder

Table 3. Psychiatric symptomatology across GD, CAH and TD groups.

	GD (n=34)	CAH (n=29)	TD (n=35)	H/ $\chi 2$	p	p^1	p^2	p^3	
GAS	82.79 ± 7.804	91.72 ± 4.487	97.43 ± 3.509	57.577	**<.001**	**.001**	**<.001**	**.001**	**3>2>1**
CDI	23.44± 10.16	15.9 ± 8.78	14.89 ± 6.65	15.565	**<.001**	**.004**	**.001**	**1.000**	**1>2, 1>3**
ASSQ	12.5 ± 9.67	8.21 ± 6.38	5.69 ± 5.06	11.669	**.003**	.282	**.002**	**.335**	**1>3**
ADHD Scale Score									
Inattention	8.82 ± 6.96	4.07 ± 4.61	4.37 ± 3.94	11.075	**.004**	**.006**	**.029**	**1.00**	**1>2, 1>3**
Hyperactivity	5.36 ± 6.1	2.66 ± 3.65	3.17 ± 3.0	3.113	.211				
Opposition-Defiance	6.41±5.84	3.48±3.6	4.54±4.02	4.068	.131				
Conduct Problems	2.09±5.96	.38±.98	.29±.71	4.884	.087				

1:GD, 2:CAH,3:TD
GD = Gender Dysphoria, CAH = Congenital Adrenal Hyperplasia, TD = Typically Developing, ADHD=Attention Deficit Hyperactivity Disorder
GAS = Global Assessment Scale, CDI = Children's Depression Inventory, ASSQ = Autism Spectrum Screening Questionnaire
p1 GD-CAH pairwise comparison
p2 GD-TD pairwise comparison
p3 CAH-TD pairwise comparison

Table 4. Differences in social cognition (affective and cognitive theory of mind) in GD, CAH and TD groups

	GD (n=34)	CAH (n=29)	TD (n=35)	H	p	p^1	p^2	p^3	
RMET	19,91 ± 3,127	19,38 ± 3,234	21,69 ± 2,564	11.520	**.003**	1.000	**.036**	**.004**	**1<3, 2<3**
FT Total Score	44,29 ± 5,562	42,48 ± 6,18	45,69 ± 4,801	6.151	**.046**	.403	.942	**.040**	2<3
Surprise	14,62 ± 3,402	13,66 ± 4,194	15,86 ± 2,658	5.418	.067				
Happiness	9,21 ± 1,225	9,28 ± 0,882	9,46 ± 0,886	.370	.831				
Anger	6,94 ± 1,347	6,55 ± 1,723	6,83 ± 1,272	.929	.628				
Sadness	6,5 ± 1,619	6,31 ± 1,815	7 ± 1,534	3.532	.171				

Disgust	6,65 ± 1,368	6,28 ± 1,533	6,31 ± 1,183	1.451	.484				
Fear	0,15 ± 0,359	0,1 ± 0,31	0,17 ± 0,453	.324	.850				
UOT	19,50 ± 4,98	15,52 ± 7,37	23,46 ± 1,482	45.916	**<.001**	.084	**<.001**	**<.001**	**1<3, 2<3**

1:GD, 2:CAH,3:TD
GD = Gender Dysphoria, CAH = Congenital Adrenal Hyperplasia, TD = Typically Developing
RMET = Reading the Mind in the Eyes Test, FT = Faces Test, UOT = Unexpected Outcomes Test
p1 GD-CAH pairwise comparison
p2 GD-TD pairwise comparison
p3 CAH-TD pairwise comparison

Table 5. Two separate models of Multiple Linear Regression Analysis for Reading the Mind Through the Eyes (RMET) and the Unexpected Outcomes Test (UOT).

	B	**Std. Error**	**Beta**	***t***	***p***	**95% CI**	
						LL	***UL***
Model-1 (RMET)							
GD	-0.758	0.782	-0.117	-0.968	.335	-2.312	0.796
CAH	-2.221	0.729	-0.328	-3.046	**.003**	-3.669	-0.773
CDI	-0.105	0.036	-0.316	-2.935	**.004**	-0.175	-0.034
ASSQ	0.004	0.046	0.01	0.089	.929	-0.087	0.095
Inattention	-0.034	0.062	-0.062	-0.544	.588	-0.157	0.089
Model-3 (UOT)							
GD	-3.232	1.388	-0.26	-2.329	**.022**	-5.988	-0.476
CAH	-7.974	1.293	-0.616	-6.166	**<.001**	-10.542	-5.405
CDI	-0.046	0.063	-0.073	-0.735	.464	-0.172	0.079
ASSQ	0.02	0.081	0.026	0.241	.81	-0.142	0.181
Inattention	-0.104	0.11	-0.1	-0.945	.347	-0.322	0.114

GD = Gender Dysphoria, CAH = Congenital Adrenal Hyperplasia, CDI = Children's Depression Inventory, ASSQ = Autism Spectrum Screening Questionnaire
* p <.05; ** p <.01; ***p <.001
Model 1 (RMET): F(5,92)=4.441, p=0.001, R^2=0.194, Adjusted R^2=0.151
Model 3 (UOT): F(5,92)=8.144, p<0.001, R^2=0.307, Adjusted R^2=0.269

TABLE S1. Regression Analysis for Faces Test (FT)

	B	**Std. Error**	**Beta**	***t***	***p***	**95% CI**	
						LL	***UL***
(Constant)	46.634	1.37	-	34.037	0.0	43.913	49.355
CDI	-2	68	-4	-32	974	-138	134
ASSQ	-58	88	-81	-656	513	-232	117
Inattention	-134	119	-138	-1.128	262	-371	102
GD	-0.38	1.503	-32	-253	801	-3.365	2.606
CAH	-3.096	1.401	-254	-2.21	**0.03**	-5.878	-314

GD = Gender Dysphoria, CAH = Congenital Adrenal Hyperplasia, CDI = Children's Depression Inventory, ASSQ = Autism Spectrum Screening Questionnaire

Model 2 (FT): $F(5,92)=1.694$, $p=.144$, $R^2=0.084$, Adjusted $R^2=0.035$*