Psychological and Behavioral Characteristics of Chromosomal Anomalies and Congenital Contiguous Gene Syndrome

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Congenital anomalies exert significant impact on individuals and their families, with particularly negative effects on their quality of life. However, studies focusing on the psychological and behavioral characteristics of children with congenital anomalies are still limited, though this information is indispensable for the educational support of such children. In this paper, we reviewed articles dealing with psychological, behavioral and socio-emotional characteristics of children with congenital anomalies including Turner syndrome, Klinefelter (XXY) syndrome, XYY syndrome, Apert syndrome, Prader-Willi syndrome, Rubinstein-Taybi syndrome, Williams syndrome, Marfan syndrome, fetal alcohol syndrome, and congenital rubella syndrome. Educational support for these children is also discussed.

Keywords: Congenital malformation syndromes, chromosomal anomalies, congenital contiguous gene syndrome, characteristics, educational support

Congenital malformation syndromes, including chromosomal anomalies and congenital contiguous gene syndrome, can be defined as structural or functional anomalies which are present at birth, and are recognized by a number of common specific anomalies. These conditions can be caused by numerical or structural abnormalities of the chromosome (ex. Turner syndrome, Prader-Willi syndrome), genetic abnormalities (ex. Rubinstein-Taybi syndrome, Williams syndrome) or various risk factors that influence the developing embryo (ex. fetal alcohol syndrome, congenital rubella syndrome). Congenital malformation syndromes may cause long-term disabilities. On account of the significant impacts of these anomalies on individuals, families and societies, studies aiming to improve these conditions from a medical perspective are abundant.12) Yet the psychological and behavioral characteristics of children with these syndromes are not yet fully understood, though this knowledge is of crucial importance in evaluating these children and making plans for their education in school settings. The aim of this study is to elucidate the psychological and behavioral characteristics associated with congenital malformation syndromes that are frequently and/or occasionally encountered in school settings through a comprehensive review of the literature.

Chromosome Abnormalities

Turner (45X) syndrome

Turner syndrome, which is also called 45X syndrome, is characterized by short stature, webbed
neck, and cardiovascular abnormalities. Turner syndrome occurs when one normal X chromosome is present but the other sex chromosome is missing or structurally altered. This syndrome is relatively frequent, occurring in 1/2,500 females.\(^1\)

The average IQ of individuals with Turner syndrome is 90, though substantial research supports a discrepancy between their visuospatial abilities and verbal abilities: women with Turner syndrome often manifest reduced visuospatial ability. In 1991, a study investigated the cognitive ability and everyday functioning of women with Turner syndrome through comparison with a matched control group.\(^2\) Using WAIS-R, they found no significant intergroup difference in verbal IQ (VIQ); there were significant intergroup differences, however, in performance IQ (PIQ) and full scale IQ (FIQ). This result was confirmed in a larger sample of school-aged children.\(^3\) In addition, a recent research review indicated that girls with Turner syndrome may have difficulty with verbal tasks requiring significant elements of visuospatial or executive processing.\(^4\)

Some researchers have investigated the psychological characteristics associated with this syndrome. Increased rates of self-reported anxiety, depression, low self-esteem, and impaired social competence are reported in girls with Turner syndrome.\(^5\) In addition, facial recognition difficulty was repeatedly reported.\(^6\)\(^7\) These difficulties in facial processing might be associated with the visuospatial difficulties mentioned above.

**Klinefelter (XXY) syndrome**

Klinefelter syndrome, which is also called XXY syndrome, is characterized by long legs, hypogonadism, infertility and behavior problems. Men with this syndrome have a super-numerical X chromosome, creating the XXY chromosomal pattern.\(^8\) This syndrome, occurring in 1/700 males, is characterized by testicular dysfunction resulting in androgen deficiency and infertility.\(^9\) Therefore, testosterone replacement therapy is initiated at the age of 11 to 12 years.

FIQ range of individuals with Klinefelter syndrome is within normal, and their PIQ is higher than their VIQ.\(^10\)\(^11\) In 2003, a research group published the results of a comprehensive neuropsychological battery administered to 35 Klinefelter adolescents and adults. These individuals with Klinefelter syndrome scored significantly below controls in language skills, verbal processing speed, verbal and nonverbal executive abilities and motor dexterity.\(^12\)

Clinical evaluation including educational, intellectual and behavioral testing is recommended because of the variability in intellectual and behavioral development in this syndrome. These may contribute to a better understanding of the syndrome’s pathogenesis, leading to better prognosis and a higher likelihood of optimal social and educational development.\(^13\)

**XXY syndrome**

XXY syndrome is characterized by tall stature and aberrant behavior. Despite its incidence of 1/840 newborn males, this syndrome is seldom detected during childhood.\(^1\) Moreover, it is estimated that at least 85% of males with XYY are never diagnosed.\(^14\) Based on data collected from 80 individuals with XYY syndrome, it has been reported that their mean FIQ is 91, mean VIQ is 88 and mean PIQ is 95.\(^15\)

Behavior problems, especially distractibility, hyperactivity and tendency toward temper tantrums, are present during childhood and adolescence.\(^2\) In 2013, the problems related to cognition and behavior that are associated with XYY were summarized based on an investigation with a large number of samples. The problems thus identified were as follows: comorbid psychiatric disorder in 27/90 (35%), ADHD in 47/90 (52%), mild verbal and/or motor tics in 16/89 (18%) and autism spectrum disorders (ASD) in 26/90 (29%).\(^16\) Some researchers have also reported a risk for comorbid ASD with XYY syndrome.\(^15\)\(^16\)

**Prader-Willi syndrome**

Prader-Willi syndrome is characterized by obesity, low muscle tonus, diminished functional activity of the gonads, and intellectual disabilities. Approximately 70% of cases are caused by deletion of the long arm of chromosome 15 at q11-q13. Another 25% are caused by having two maternal copies and no paternal copy of 15q (maternal Uniparental Disomy; maternal UPD). The remaining 5% are caused by a mutation of an imprinting center or by a chromosomal translocation involving proximal 15q.\(^17\) The prevalence is said to be 1/10,000-25,000.\(^1\)
The range of their FIQ is estimated to be 60-70. Some researchers have described these individuals as having relatively good visuospatial abilities. For example, Dykens reported that children with Prader-Willi syndrome outperformed typically developing children on jigsaw puzzles; the children with Prader-Willi syndrome placed more than twice as many puzzle pieces as typically developing children matched by age and IQ.

The behavioral characteristics of this syndrome are relatively well-documented. In 2012, a Japanese research group examined the previous literature reporting behavioral phenotypes of Prader-Willi syndrome. Their review revealed that terms such as “argumentative,” “stubborn,” “tantrum,” “skin-picking,” “eating a lot,” and “compulsive” are commonly used to describe problems associated with Prader-Willi syndrome. Some researchers have reported behavioral and psychological differences between the two main subtypes, deletion and maternal UPD; these results are not consistent, however.

Genetic abnormalities Rubinstein-Taybi syndrome

Rubinstein-Taybi syndrome is a well-defined complex of congenital malformations, consisting of peculiar facies, broad thumbs and big toes, and mental retardation. The locus for this syndrome is at 16p13.3, a region that contains the gene for the human CREB binding protein (CBP), a nuclear protein participating as a co-activator in cyclic-AMP-regulated gene expression. The prevalence of this syndrome is 1/100,000 to 1/125,000. Individual IQ ranges between 30 and 79, with an average of 51.

Behaviors of individuals with Rubinstein-Taybi syndrome are characterized by short attention span and poor coordination. Sudden mood changes begin in early adulthood and seem to increase in frequency with age. It is also reported that they tend to have a happy disposition: after some initial shyness, they are friendly and eager to co-operate. Impulsivity and disruptive actions in adulthood are also observed among individuals with this syndrome. Almost all patients will be best stimulated if they attend special schools for children with learning disabilities.

Kabuki syndrome

Approximately 60% of Kabuki syndrome cases are caused by mutations in the mixed lineage leukemia 2 gene (MLL2) which encodes proteins involved in histone modification. Kabuki syndrome can be diagnosed based on five clinical manifestations: 1) peculiar facial features, characterized by eversion of the lower lateral eyelid, arched eye-brows with a sparse lateral third, depressed nasal tip and prominent ears, 2) skeletal abnormalities, 3) dermatoglyphic abnormalities (unusual fingerprints), 4) mental retardation and 5) short stature. The prevalence of this syndrome is estimated at 1/32,000 in Japan and at 1/86,000 in Australia and New Zealand.

Although mental retardation is considered a clinical manifestation of Kabuki syndrome, 12% of individuals have IQ equal to or greater than 80. Most individuals show mild or moderate mental retardation (mean IQ= 52), while severe mental retardation is uncommon. Case-studies have suggested that most individuals show better performance in VIQ than in PIQ (a difference of more than 10 points). Specifically, individuals with Kabuki syndrome manifest difficulties in visuospatial construction abilities, spatial memory and spatial reasoning. On the other hand, they have relatively strong ability to define words as well as good inductive and sequential reasoning abilities. Although verbal skills have been described as a strength in Kabuki syndrome, specific problems have been reported in phonological and morphosyntactic abilities, while problems in receptive language and expressive vocabulary seem to be less common.

From a socio-emotional perspective, Kabuki syndrome is said to be associated with poor eye-contact. However, Individuals with Kabuki syndrome are also said to be friendly, comfortable with strangers, cheerful and affectionate. Some behavioral problems are related to obsessive tendencies and/or anxiety problems. Several children with Kabuki syndrome have been reported to have musical aptitude; accordingly, music has been found to be an effective tool for teaching new skills and concepts.

Williams syndrome

Williams syndrome is a contiguous gene microdeletion syndrome, caused by a deletion in chromosome 7q11.23, a region that includes 25 to 30
The deletion usually includes the elastin gene (ELN) responsible for abnormalities of connective tissue (i.e. supra-valvular aortic stenosis, inguinal hernias, soft skin, etc.) and the LIMK1 gene which contributes to visuospatial construction impairment. The physical characteristics of this syndrome include elfin-like facies (prominent forehead, widely spaced eyes, upturned nose, underdeveloped mandible, incomplete or defective development of the teeth, patulous or wide lips), musculoskeletal dysmorphia and, cardiovascular and renal abnormalities. The incidence of this syndrome is estimated to be between 1/7,500 and 1/20,000.

The IQ range for Williams syndrome is 51-70. Studies using the Weschler tests to assess their intellectual ability did not find a significant difference between VIQ and PIQ. Studies using the Differential Ability Scales assessment (DAS), however, have reported significantly better performance in verbal and nonverbal reasoning and verbal short-term memory than in spatial ability (a difference of approximately 20 points). With regard to language development, infants with this syndrome show delay in canonical babble (repeated syllables in a timing relationship, i.e. “bababa”) and word production. Concrete vocabulary (receptive and expressive) is considered a relative strength compared to relational vocabulary (terms for spatial, temporal, quantitative, and dimensional concepts, conjunctions and disjunctions). Findings in functional neuroimaging suggest that difficulties in visuospatial abilities and relational vocabulary are linked to reduced gray matter in the region of the dorsal stream which processes motion, position and three-dimensional form perceptions. Characteristics of their conversational skills are: fluent speech including an excessive number of stereotyped phrases and idioms, over-familiarity, introduction of irrelevant personal experience, perseverative responding, inappropriate initiation of conversations and overdependence on context to interpret the message. Pragmatic language is also limited due to poor joint attention and less comprehension and production of gestures, leading to difficulties in maintaining the topic of conversation.

Individuals with Williams syndrome are described as gregarious, people-oriented, affectionate personality, tense and sensitive. Their social interactive behaviors are driven by a desire for social closeness. These individuals also show hypersensitivity to feelings of frustration, frequent temper outburst and lack of motivation to complete difficult tasks. Researchers have found that individuals with this syndrome attempt to distract or engage socially with the examiner in order to avoid difficult tasks. In contrast to individuals with Down syndrome, who exhibit lower cognitive and adaptive skills, individuals with Williams syndrome showed less persistent task-related behavior and mastery pleasure on moderately challenging tasks. It is worth mentioning that individuals with mutation or deletion of the ELN gene alone do not exhibit the cognitive or behavioral characteristics of Williams syndrome.

Regarding the educational support, some researchers have investigated visuospatial difficulties in writing Kanji (Chinese characters) in Japanese children with Williams syndrome. They found that color in addition to location enabled participants to succeed at visuospatial tasks. In teaching reading and writing, it has been reported that a systematic phonics approach yields better results in children with Williams syndrome than a whole-word or whole-language approach does.

Aarskog syndrome

Facio-digito-genital dysplasia or Aarskog syndrome is a genetic disorder linked to the X chromosome and caused by mutations in the FGD1 gene. This syndrome is clinically and genetically heterogeneous, but there are several distinctive features: namely, short stature, hypertelorism (abnormally increased distance between the eyes), shawl scrotum and brachydactyly (shortness of the fingers and toes). Additional features may include: short nose, interdigital webbing, joint hyperlaxity, and inguinal hernias. Although the phenotype is variable in males, females typically show only minor and mild clinical signs.

Levels of intelligence for this syndrome range from average to extremely low, but most individuals with Aarskog syndrome fall somewhere between low average and average with mild to moderate learning disabilities. Additionally, attention deficit hyperactivity disorder (ADHD)
and/or behavioral problems are common during childhood. Fryns describes Aarskog syndrome as having a “changing phenotype with age” and mentions that some physical and behavioral features are less marked after the age of 12 to 14 years; this has been corroborated by other studies in which participants showed age-related improvement of mental status.

The prognosis of Aarskog syndrome seems to grow more favorable as patient age increases. An appropriate educational approach during childhood is essential for an optimal long-term outcome. Researchers recommend cognitive training and structuring of daily life in order to reduce the intensity of disinhibited behaviors. They also recommend intervention in executive attentional processes because the syndrome may be linked to attentional problems.

Fragile X syndrome

Fragile X syndrome, also called Martin-Bell syndrome or Marker X syndrome, is an X-linked dominant neurodevelopmental disorder. This syndrome is the most commonly inherited form of mental retardation. The physical manifestations of Fragile X are a prominent chin, a long and narrow face, a bulbous nose, and abnormal size and shape of the ears. This syndrome is caused by an abnormal expansion of CGG trinucleotide repeats within the fragile X mental retardation 1: FMR1 gene located on the long arm of the X chromosome. The prevalence of this syndrome is 1/4,000 males and 1/8,000 females.

FIQ of individuals with Fragile X syndrome ranges between 30 and 55. Mild to severe intellectual disability is reported in males while variable intellectual disability (mild>severe) is reported in females. Cognitive features include declines in intellect, short-term memory and executive functioning. Furthermore, speech and language impairment including perseverative speech and echolalia are reported.

Behavioral features of ADHD, including hyperactivity, inattentiveness, distractibility, restlessness, and impulsivity, are present in 80% of individuals with this syndrome. Furthermore, these individuals tend to display aggressiveness, self-injurious behaviors, hypersensitivity, shyness, social avoidance, social anxiety, mood lability, gaze aversion, and autistic behaviors.

Sotos syndrome

Sotos syndrome, which is also called cerebral gigantism syndrome, is characterized physically by large hands and feet and poor coordination. Mutations and deletions of the nuclear receptor SET-domain-containing protein: NSD1 gene, located at chromosome 5q35 and coding for a histone methyltransferase implicated in transcriptional regulation, are responsible for more than 75% of cases. The prevalence of this syndrome is 1/10,000 to 1/50,000. Patients’ IQ ranges between 40 and 129 with a mean of 78.

The developmental delay, clumsiness, and uncoordination in Sotos children seems to be out of proportion to their congenital hypotonia; speech, math, and fine motor skills appear to be especially delayed.

Behavior is characterized by social relationship problems and anxiety. Children with this syndrome show more separation anxiety and tend to be more anxious in new situations. It is often reported by parents that children’s anxiety and emotional immaturity may contribute to their poor peer relationships. According to some researchers, most children with Sotos syndrome have no friends in their class or in their neighbourhood.

Early in childhood, programs including infant stimulation, occupational therapy, speech therapy, and adaptive physical education play a significant role in nurturing children with Sotos syndrome. After early intervention, some children have been able to participate in regular classrooms with support, while others have been enrolled in special classes with appropriate educational settings.

Apert syndrome

Apert syndrome is a type of craniosynostosis syndrome, characterized by distortions of the head and face (large skull but short from front to back, spaced eyes, etc.) associated with mid-face hypoplasia (the upper two-thirds of the face do not grow normally) and symmetric syndactyly (fusion of two or more digits) of the hands and feet and other systemic malformations. This syndrome is caused by one or two mutations in the human fibroblast
growth factor receptor 2 gene: FGFR2. The incidence of Apert syndrome is estimated to be between 1 in 80,000 and 15 in 1 million.

Studies of the Apert syndrome population have revealed a cognitive profile characterized by IQ ranging from extremely low to average, with a tendency toward better scores in PIQ than in VIQ. However, poor attention, arithmetic (solving sequential information) and memory skills have been described in 25 school-aged children with Apert syndrome. In addition, a reported case with average IQ showed poor performance in processing speed, attention, visual memory, executive function and fine motor. Despite the variety of brain malformations reported in Apert syndrome, we presume that, to some extent, their cognitive difficulties may be explained by their physical difficulties. Fine motor, processing speed, visual memory and visuospatial activities require coordination between eyes and hands. These parts of the body are especially poorly developed in Apert syndrome.

Regarding the socio-emotional profile, no severe maladjustment has been reported. However, children and adults with Apert syndrome are at increased risk of emotional problems and social withdrawal due to their facial deformation.

Marfan syndrome

Marfan syndrome is an autosomal dominant disorder characterized by arachnodactyly (long and slender fingers in comparison to the palm of the hand) with hyperextensibility, ectopia lentis (dislocation of the lens) and aortic dilation. This syndrome is caused by a mutation in the fibrillin gene: FBN1 located on chromosome 15q15-21.3.8, resulting in increased levels of the protein known as transforming growth factor beta, or TGF-β. This increase in TGF-β causes problems in connective tissues throughout the body. Approximately 1/5,000 individuals are affected, though this figure is considered an underestimation.

Some research has indicated that Marfan syndrome is associated with weak visuospatial abilities. In 1996, Paep and co-author evaluated the neuropsychological status of 13 adults with this syndrome and reported that they performed significantly worse than normal adults only on tests measuring sustained visual attention and visuoconstruction. Hofman and co-workers evaluated the cognitive ability of 30 children with this syndrome in 1988 and reported that their mean FIQ was 109.3; their PIQ score was lower than their verbal score, with two PIQ subtests, object assembly and coding, returning particularly low scores. Moreover, the severity of joint hypermobility was strongly correlated with verbal-performance discrepancy, indicating that the depressed PIQ score was due, in part, to motor incoordination. Hofman and co-workers also emphasized the necessity of careful evaluation of individuals with hand-wrist hypermobility, given the impact of this condition on writing ability, in the classroom.

Abnormalities caused by various risk factors

Congenital rubella syndrome

Congenital rubella syndrome is characterized by intrauterine growth restriction, microcephaly, meningoencephalitis, cataracts, retinopathy, hearing loss, cardiac defects, and hepatosplenomegaly. Maternal rubella during the initial eight weeks of pregnancy produces cataracts and congenital heart lesions, whereas infection during the first 16 weeks is associated with hearing loss. Due to the dual vaccination strategy currently employed against rubella in the United States (all infants 12 months to 15 months of age, and all women of child-bearing age), the estimated incidence of this syndrome is less than 2/100,000 live births in that country.

Using the Leiter International Scale, Macfarlane and co-workers evaluated 92 children with sufficient vision for testing and reported that the mean IQ for this group was 99.46. Concerning the behavioral effects of the syndrome, Chess investigated 243 children with congenital rubella and identified 10 of these children as autistic and another eight as showing a significant number of signs of autistic behavior. There are already a considerable number of studies describing an increased risk of autism among children with congenital rubella.

Fetal alcohol syndrome

Fetal alcohol syndrome is characterized by prenatal onset of growth deficiency, microcephalhy and short palpebral fissures. The incidence of this syndrome is estimated to range from 0.33/1,000 to
Matson and co-workers evaluated the cognitive ability of 34 children with this syndrome and reported that their mean FIQ was 74.4, with a mean VIQ of 75.3 and a mean PIQ of 77.9.

Thomas and co-workers assessed the social skills of children with this syndrome using the Vineland Adaptive Behavior Scales via interviews with their caregivers and reported that they were most impaired on the subdomain assessing interpersonal relationship skills.

References
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### Appendix: Characteristics of Each Syndrome

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<th>Syndrome</th>
<th>IQ/Cognitive Characteristics</th>
<th>Socio-Emotional Characteristics</th>
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<tr>
<td>Turner (45X) syndrome</td>
<td>Mean IQ: 90&lt;sup&gt;50&lt;/sup&gt;</td>
<td>Depression, anxiety, low self-esteem, facial recognition difficulty&lt;sup&gt;35&lt;/sup&gt;</td>
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<tr>
<td>Klinefelter syndrome</td>
<td>IQ:Normal&lt;sup&gt;50&lt;/sup&gt;</td>
<td>Distactibility, hyperactivity and impulsivity&lt;sup&gt;25&lt;/sup&gt;</td>
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<tr>
<td>XYY syndrome</td>
<td>Mean IQ: 91&lt;sup&gt;14&lt;/sup&gt;</td>
<td>Related to high percentage of comorbid psychiatric disorders&lt;sup&gt;15, 16&lt;/sup&gt;</td>
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<tr>
<td>Prader-Willi syndrome</td>
<td>IQ range: 60-70&lt;sup&gt;20&lt;/sup&gt;</td>
<td>Slightly better PIQ &gt; VIQ&lt;sup&gt;14&lt;/sup&gt;</td>
</tr>
<tr>
<td>Rubinstein-Taybi syndrome</td>
<td>IQ range: 30-79&lt;sup&gt;25&lt;/sup&gt;</td>
<td>G Good visuo-spatial skills&lt;sup&gt;18, 15&lt;/sup&gt;</td>
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<tr>
<td>Kabuki syndrome</td>
<td>Mean IQ: 52&lt;sup&gt;27&lt;/sup&gt;</td>
<td>PIQ &lt; VIQ&lt;sup&gt;28&lt;/sup&gt;</td>
</tr>
<tr>
<td>Williams syndrome</td>
<td>IQ range: 51-70&lt;sup&gt;35&lt;/sup&gt;</td>
<td>Short attention span, poor coordination&lt;sup&gt;21&lt;/sup&gt;</td>
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<td>Aarskog syndrome</td>
<td>Extremely low-normal&lt;sup&gt;40&lt;/sup&gt;</td>
<td>Happy disposition, friendly and co-operative&lt;sup&gt;23&lt;/sup&gt;</td>
</tr>
<tr>
<td>Fragile X syndrome</td>
<td>IQ range: 30-55&lt;sup&gt;55&lt;/sup&gt;</td>
<td>Good visuo-spatial skills&lt;sup&gt;25&lt;/sup&gt;</td>
</tr>
<tr>
<td>Sotos syndrome</td>
<td>IQ range: 40-129&lt;sup&gt;69&lt;/sup&gt;</td>
<td>Poor short-term memory, poor executive function, and echolalia&lt;sup&gt;30&lt;/sup&gt;</td>
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<tr>
<td>Apert syndrome</td>
<td>Extremely low-normal&lt;sup&gt;41&lt;/sup&gt;</td>
<td>Poor short-term memory, poor executive function, and echolalia&lt;sup&gt;30&lt;/sup&gt;</td>
</tr>
<tr>
<td>Marfan syndrome</td>
<td>Mean IQ: 109.3&lt;sup&gt;39&lt;/sup&gt;</td>
<td>Problems in speech, math, and fine motor skills&lt;sup&gt;50&lt;/sup&gt;</td>
</tr>
<tr>
<td>Congenital rubella syndrome</td>
<td>Mean IQ: 99.5&lt;sup&gt;72&lt;/sup&gt;</td>
<td>Hyperactivity, aggressive and self-injurious behavior&lt;sup&gt;31&lt;/sup&gt;, autistic behavior&lt;sup&gt;81&lt;/sup&gt;</td>
</tr>
<tr>
<td>Fetal alcohol syndrome</td>
<td>Mean IQ: 74.4&lt;sup&gt;78&lt;/sup&gt;</td>
<td>PIQ &lt; VIQ, motor incoordination&lt;sup&gt;78&lt;/sup&gt;</td>
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