CASER

Issue 17, May 2020

Child Assessment Service Epidemiology and Research Bulletin

Autism Spectrum Disorder: A Brief Review

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Conceptualization of Autism Spectrum Disorder (ASD)

From 1911 when the concept of autism was coined by the German psychiatrist Eugen Bleuler to describe a symptom of the most severe cases of schizophrenia,[1] the meaning of autism was gradually detached from hallucination and fantasy, to involve neurocognitive developmental factors including social and language impairments and a variety of compulsive, repetitive and stereotypic activities.[2,3] These reconceptualizations followed the expansion of an epidemiological approach in child psychiatry and developmental psychology, along with the growth in genetic and neuroscientific facts and findings.

In the 11th revision of the International Classification of Diseases[4] and Diagnostic and Statistical Manual of Mental Disorders Fifth Edition (DSM-5),[5] ASD is characterised as a neurodevelopmental disorder with persistent deficits in the ability to initiate and to sustain reciprocal social interaction and social communication, and by a range of restricted, repetitive, and inflexible patterns of behaviour and interests; where the onset of the disorder occurs during the developmental period but may not be fully manifested until later when social demands exceed limited capacities. Individuals along the spectrum may exhibit a full range of intellectual functioning and language abilities, and may have associated known medical, genetic or environmental factors or other neurodevelopmental, mental or behavioural disorders.

Neurogenetic and neurobiological mechanisms

Genetic factors play an important role in the aetiology of ASD, with increased rates in siblings and high concordance rates in twins with ASD.[6] Based on 13 twin studies,[7] correlation for monozygotic twins was 0.98 (95% Confidence Interval, 0.96-0.99), and dizygotic twins 0.67 (95% CI 0.61-0.72) when prevalence rate was set at 1%. More than a hundred known genetic disorders such as Fragile X syndrome and Rett syndrome have been associated with ASD, accounting for around 10% of ASD cases.[8] However, none of these genes represents more than 1% of the ASD cases. Technology enabling genome-wide throughput and single base pair resolution has increased identification of involved genetic variants. Rare variants associated with ASD are largely de novo, with copy number variation (CNVs) rare risk variants explaining around 5-10% of idiopathic ASD. Common variants which are relatively frequent in the population (e.g. in at least 5%), collectively contribute to ASD risk, but individually has small effect. Common single nucleotide polymorphism (SNP) variants have been reported to be significantly associated with ASD.[9] Interplay between rare and common variants on individuals contributes to many genetic forms of autism. The same risk variant may be associated with a specific co-morbidity profile, which often includes neurodevelopmental and psychiatric but also purely somatic phenotypes.[10]

Studies on the biological pathways of autism risk genes have converged mostly on proteins involved in chromatin remodeling (regulation of gene expression) and synaptic plasticity. It is hypothesised that abnormal synaptic plasticity and failure of neuronal / synaptic homeostasis could play key roles in susceptibility to autism.[11,12] Electrophysiological studies in individuals with autism have shown an imbalance between excitatory and inhibitory systems due to the lack of physiological inhibitory effect of gamma-Aminobutyric acid (GABA).[13] Reduced inhibition of the amygdala may be responsible for increase in long-term potentiation of excitatory synapses in subcortical circuits for face perception, leading to abnormal reaction to eye contact, avoidance of direct gaze and subsequent abnormal development of the social brain. Hyperactivation of the amygdala,[14] hypoactivation of inferior frontal gyrus or superior temporal sulcus[15] and under-connectivity between elements of the social brain[16,17] have been reported as abnormalities in the social brain of individuals with ASD.

Prevalence of ASD and its interpretation

Another aspect of ASD of keen prevailing interest to researchers and the public alike is the reported prevalence rates which appeared to have risen dramatically from the 1990s. Victor Lotter's first epidemiological study of autism posited a rate of 4.5 per 10,000 children,[18] followed by an apparent continuous rise in this figure in ensuing years. More recent studies include the Korean study which reported a prevalence of 2.64% in 2011[19] and the Center for Disease Control report in 2018 of 16.8 per 1,000 (one in 59) children aged 8 years.[20] Complex methodology in epidemiological surveys, different screening and diagnostic procedures, and different methods of gathering information where parents, teachers, clinicians and the ASD individuals themselves may or may not be involved, render prevalence differences among studies difficult to evaluate.

Meanwhile, large twin studies looking at autism related traits have shown steady prevalence rates.[21] A systematic review of epidemiological surveys of autistic disorder and pervasive developmental disorders was conducted worldwide which showed a median of prevalence estimates of ASD at 62/10,000.[22] Another review conducted on 61 surveys published since 2000 from 22 different counties and sample sizes ranging from 5,007 to 4.25 million, showed a wide range from 1.4/10,000 to 264/10,000 with substantial variation in confidence intervals' width. There was however some consistency in ASD prevalence found in the center of this distribution, with a median rate of 61.9/10,000 and a mean rate of 69.0/10,000 (interquartile range: 36.5–90.0/10,000).[23] With significant issues in case definition, case identification and case evaluation methods noted across studies, added to the lack of various information for comparison in different datasets, the power to detect time trends is limited. Changing diagnostic criteria over the past decades no doubt adds to complexity in evaluating observed data. The lack of boundary criteria for milder forms together with social influences including increased public awareness, clinician alertness to ASD symptoms and widened service availability, are believed to affect the rates of ASD observed and recorded.

Validity of ASD as a unitary concept

The validity of there being one version of ASD is challenged.[24] Debate includes whether a unitary ASD concept with a single dimension incorporating the different criteria domains should be reconceptualised, and viewed as co-occurrence of separate subdomains of impairment.[25] Problems also remain in DSM-ICD criteria based ASD research which has shown no early behaviours which could predict ASD diagnosis,[26] wide variations in developmental trajectories,[27,28] varied life outcomes[29-31] and no specific medical treatment.[32]

The current state of ASD research is at a turning point. There are complex combinations of multiple genetic and environmental factors to be resolved. Genetic architecture of individuals with ASD differ from one individual to another, with many individually varied "connections between brain and behavior".[33] Heterogeneity of risk factors, brain impairment and non-diagnostic symptoms in ASD individuals remain to be explained. As such, support for the neurobiological and construct validity

of ASD diagnostic criteria and ASD spectrum features has been increasingly called to question.[34] In 2008 the National Institutes of Mental Health (NIMH) proposed a Research Domain Criteria (RDoC) framework which aims to understand the mechanisms of complex behaviour and mental health disorders, including ASD, through neurobiological measures and their relationships to observed behaviour and context.[35-37] The objective is for neuroscience and behavioural science to provide a biologically valid, reformulated classification, instead of categorical diagnoses that may lead to samples that interfere with valid scientific analysis. While RDoC may not be a classification system in its own right in the foreseeable future, it is expected to coexist with DSM-ICD and provide scaffolding for large scale research programmes on ASD and other mental disorders.

Foundations of management

While nosology, research methodology and neurobiological studies continue, the current diagnostic and intervention measures remain in clinical practice. Inter-professional collaboration with a transdisciplinary approach is essential for effective management. Early identification and assessment, behavioural and educational training, social adjustment, as well as continual parental support are mainstay. Intervention programmes with scientific grounding and evidence of their effectiveness should focus on addressing core deficits of ASD including social communication, language, play skills, and behavioural issues, while medication is chiefly reserved for managing associated emotional problems such as anxiety and depression and for maladaptive behaviours. The full range of developmental needs of young children, teenagers and adults with ASD in coping with different transitions and life stages has to be borne in mind.

Early intensive and sustained intervention with the use of multiple treatment modalities carried out in natural settings, with active parental involvement, have proven to be effective. Complementary and alternative medicine (CAM) treatments, many with limited supporting scientific evidence, are often sought by parents of children with ASD.[38] Respectful discussion by the clinician with parents on their use and caution to potential adverse health impact are essential when addressing these options.

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CAS Epidemiological Data on Autism Spectrum Disorders from 2009-2018

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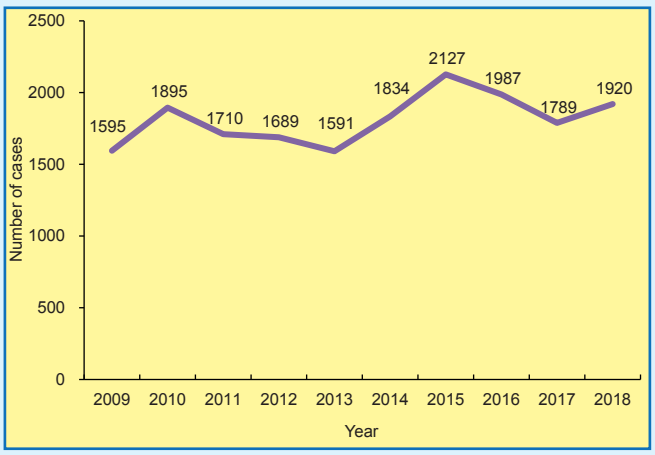
After the reported surge in the prevalence of Autism Spectrum Disorders (ASD) from worldwide ongoing surveillance and epidemiological studies from year 2000 to 2010, a plateau in ASD prevalence was apparently noted in recent years. Some longitudinal surveillance studies have shown a rising trend in ASD estimated prevalence. For 2014, the Centers for Disease Control and Prevention (CDC) of United States estimated that the overall prevalence of ASD was 16.8 per 1000 (one in 59) children aged 8 years,[1] while different rates were reported in various reviews. One systematic review for epidemiological data of ASD in the Global Burden of Disease Study 2010 (GBD 2010) by World Health Organization estimated an international prevalence of ASD of 7.6 per 1000 (one in 132).[2] A systematic literature review reported estimated prevalence of ASD of 26.6 per 10,000 (95% CI: 18.5, 34.6) in mainland China, Hong Kong and Taiwan, which was lower than estimates from western countries,[3] but substantial heterogeneity was identified between studies and strongly associated with the choice of screening instrument. Heterogeneity in methodology, study population, diagnostic criteria or case ascertainment was noted between epidemiological studies.[4-6] At Child Assessment Service (CAS), figures from 2009 to 2018 showed that the annual total number of newly diagnosed ASD ranged from 1591 to 2127 (Figure 1). The peak was in year of 2015. The observed numbers of cases over this period at CAS could be attributed to a variety of reasons, including increased public and clinician awareness, significant fluctuations in birth rate during this period in Hong Kong, rise in total number of referrals per year to CAS, service manpower situation and increase in community assessment service providers. The newly diagnosed ASD at CAS comprised of different subgroups, namely Autistic Disorder, Autism, Asperger's Disorder and Pervasive Developmental Disorder not otherwise specified (PDD-NOS). With introduction of revised diagnostic criteria of ASD in the

Diagnostic and Statistical Manual of Mental Disorders Fifth Edition (DSM-5), the distribution of different subgroups would change significantly, with Asperger's Disorder no longer being included under the umbrella term of ASD.

Figure 1. The number of ASD children newly diagnosed with ASD from year 2009 to 2018



Gender ratio

In the analysis, three subgroups of condition in the spectrum were included, namely Childhood autism (F84.0), Atypical autism (F84.1) and Asperger Syndrome (F84.5), using the ICD-10 classification. Among the 1920 children newly diagnosed with ASD in 2018, boys seemed to be more affected than girls (Table 1). It matched with data reported elsewhere. The male to female ratio of all ASD conditions taken together was 5.5 to 1 in children below 12 years of age. However, this ratio is different from the figure of 4 to 1 reported in the ADDM Network paper in 2018.[1]

Table 1. Number of children with ASD by gender, 2018

Subgroup of ASD	Male	Female
Childhood autism (F84.0)	658	126
Atypical autism (F84.1) and Asperger Syndrome (F84.5)	963	170
Total	1621	296

Note: Cases without valid gender information are excluded.

Age at diagnosis

Table 2. Number of children with ASD by age, 2018

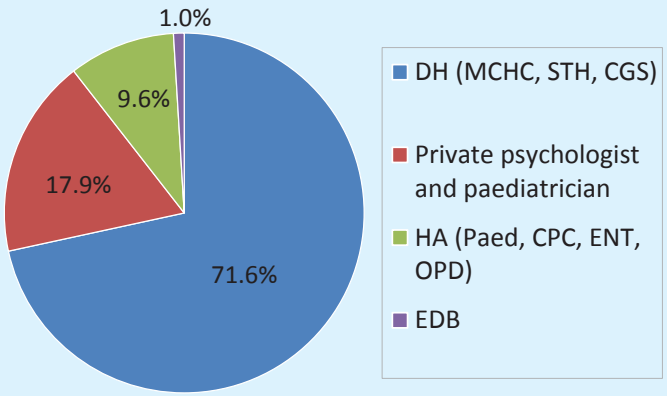
Age group	Childhood autism (F84.0)	Atypical autism (F84.1) & Asperger's Syndrome (F84.5)	Total
1 year old	7	1	8
2 years old	305	156	461
3 years old	296	344	640
4 years old	66	198	264
5 years old	54	177	231
6 years old	34	17	51
7 years old	12	41	53
8 years old	5	20	25
9 years old	1	8	9
10 years old	2	8	10
11 years old	1	5	6
12 years old	1	2	3

Note: Cases without valid date of birth information are excluded.

In 2018, the peak age of diagnosis of autism spectrum disorder was 3 years old amongst children of different subtypes of ASDs from 0 to 12 years. More than one third of children were diagnosed at 3 years of age (Table 2). For the subtype of Childhood autism (F84.0), the peak age of diagnosis can be as early as 2 years old. In the United States, the median age of earliest known ASD diagnosis was 52 months, as published in the ADDM Network paper.[1] The observed difference between Hong Kong and the United States could be explained by their difference in developmental surveillance programme and the referral system.

Sources of referral

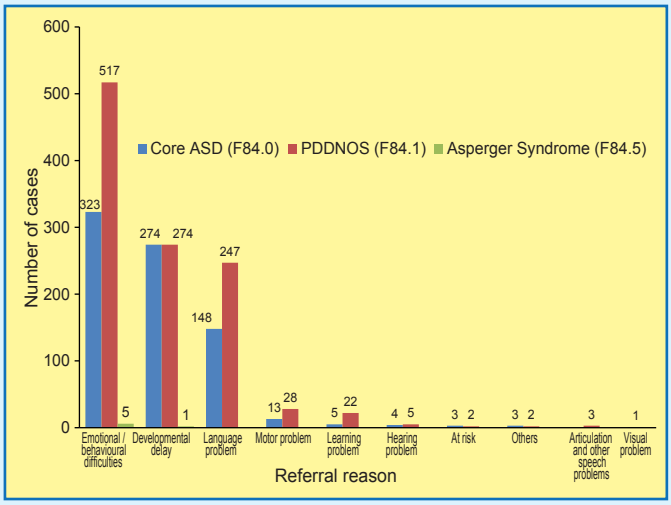
Figure 2. Number of children with ASD by source of referral, 2018



Child Assessment Service receives referral from specialists all over Hong Kong depending on the catchment areas of different centres. In 2018, majority of children (72%) subsequently diagnosed to have ASD was referred by Department of Health which included Maternal and Child Health Centre, Student Health Service and Clinical Genetic Service (Figure 2). The second major referral source was private Clinical and Educational Psychologists and private Paediatricians which make up another 18% of the total referrals. Children from specialists in Hospital Authority also make up around 10% of the total referrals. Hospital Authority specialists included Paediatricians, Otolaryngologists, Psychiatrists, Family Physicians and Ophthalmologists. Finally, there was a small portion of children referred from Educational Psychologists in Education Bureau.

Reasons of referral

Figure 3. Number of children with ASD by reason of referral, 2018



When reviewing the referral reasons for those children finally diagnosed with childhood autism (F84.0), Atypical autism (F84.1) and Asperger's Syndrome (F84.5) in 2018, emotional and behavioural difficulties was the major referral reason which arouse the concern from parents. It was more prominent for children with Atypical autism (F84.1) and Asperger's Syndrome (F84.5) (Figure 3). The second main referral reason was developmental delay noticed before 4.5 years old. Language problem at all ages and speech problem, was found to be the third common reason for referral. Other referral reasons included learning problem for those children over 4.5 years old, motor problem either gross or fine motor problem, hearing and vision concerns and those at risk groups (e.g. very low birth weight, birth asphyxia, etc). These referral reasons only made up of around 7% of the total referrals.

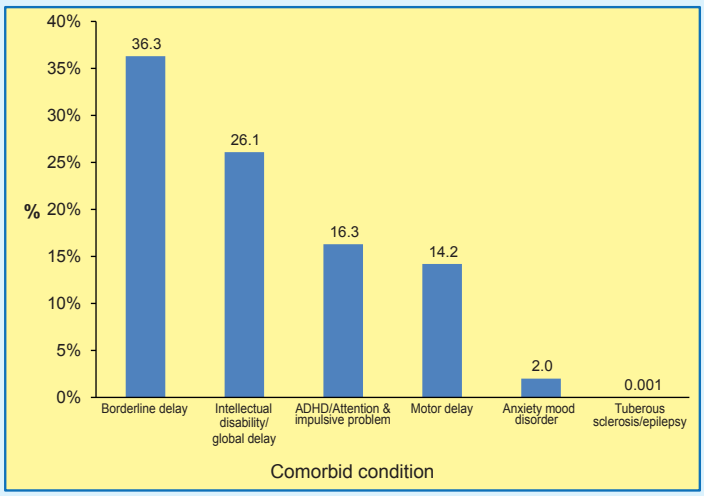
Comorbid conditions

Autism Spectrum Disorder has been well known for its frequent association with numerous neurodevelopmental, mental, physical and functional coexisting conditions, which often contribute to higher level of impairment and tend to cluster in the same individual. With the heterogeneity of study methodology, study samples and assessment methods, different studies yielded different rate of coexisting conditions. The most frequently reported comorbid conditions were: intellectual disability (ID) or developmental delay (prevalence of 15-65% for different samples), Attention Deficit Hyperactivity Disorder (ADHD) (28.2%), Anxiety Disorder (11.2%), academic learning difficulties (75% of individuals aged 9-18 years), speech and language delays (87% in 3-year-old children with ASD), sleeping problems (25-40%) or gastrointestinal symptoms (47%), tics and epilepsy (8-9%).[5] In the latest Autism and Developmental Disabilities Monitoring (ADDM) Network, which is an active, multisite surveillance system for ASD in United States,[7] in the 2010 survey year, over 95% of children with ASD had at least one co-occurring condition/symptoms, with higher prevalence in 8- than 4-year-olds. The different co-occurring condition/symptoms affected the age of ASD diagnosis, either impacting the severity of ASD symptoms causing earlier diagnosis or masking the core behaviours of ASD resulting in later identification of ASD.

As shown in Figure 4, borderline developmental delay (36.3%) and intellectual disability/global delay (26.1%) remained to be the most common comorbid conditions among children diagnosed with ASD in CAS in year 2018. In DSM-5 which dual diagnosis of ASD and ADHD was permitted, 16.3% of newly diagnosed ASD in CAS had comorbid attention problems/hyperactive problems or ADHD. Another common comorbid condition was motor delay, 14.2% of ASD had fine motor or gross motor delay. On the other hand, only 2% of ASD had comorbid with anxiety mood disorder which had much lower incidence when compared with worldwide data. In fact, the incidence of anxiety disorder/problems has

been relatively low but stable over the years in CAS, partly due to the internalising nature of the condition. In 2018, only 3 cases of ASD had comorbid epilepsy or tuberous sclerosis which was also lower than that reported in the literature.

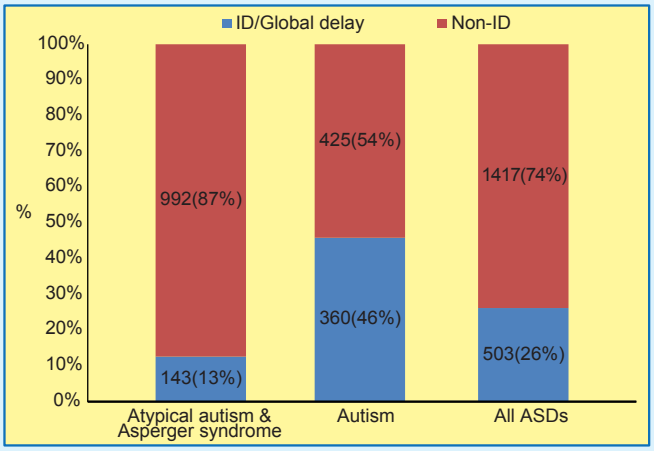
Figure 4. Comorbid conditions in children diagnosed with ASD in 2018



ASD and intellectual disability

One of the common and disabling comorbidities is intellectual disability or global developmental delay. Latest CDC prevalence study of children aged 8 years with ASD showed that 31% had intellectual disability (IQ <70), 25% were in the borderline range (IQ 71-85), and 44% had IQ >85.[1] Among the 1920 newly diagnosed ASD in the year 2018 in CAS, 26% of children had intellectual disability or global developmental delay, which was lower than that of CDC study. For the childhood autism cases, 46% of children had intellectual disability or global delay. It clearly showed that higher incidence of intellectual disability or global delay among children with more severe symptoms of ASD causing significant disease burden to the children and their families. For the atypical autism and Asperger syndrome cases, only 13% of children had intellectual disability or global delay (Figure 5).

Figure 5. Intellectual disability or global delay in ASD subgroups



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Diagnostic Considerations of Autism Spectrum Disorder in DSM-5

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General changes in the diagnosis of ASD in DSM-5

With the launching of Diagnostic and Statistical Manual of Mental Disorders Fifth Edition (DSM-5)[1] in 2013, the diagnosis "Autism Spectrum Disorder" (ASD) has been introduced to replace the prior category of "Pervasive Developmental Disorders" in the DSM-IV-TR. The various categorical diagnostic subtypes of Autism Spectrum Disorders, namely, Autistic Disorder, Asperger's Disorder, Childhood Disintegrative Disorder and Pervasive Developmental Disorder not otherwise specified (PDD-NOS), are now subsumed under the single umbrella diagnosis of ASD. Given its single-gene aetiology, a decision was made to remove Rett's Disorder from DSM-5, although an individual with this genetic condition would still receive an ASD diagnosis, probably with a specifier, if the diagnostic criteria for ASD are met.[2]

Over the years, researchers found that the former categorical Autism Spectrum Disorder groupings in DSM-IV-TR were not consistently applied across clinics and treatment centres, and the move from using previous clinical categorical diagnoses to unified dimensional descriptions of core features of deficits in social communication and cognitive flexibility was made to improve diagnostic accuracy and better reflect the state of knowledge about autism.[3]

In addition, there are several other important changes in the diagnostic criteria introduced in DSM-5. Firstly, the three areas of symptoms (i.e. social impairment, communication deficits, and repetitive/restricted behaviours) have been rearranged into two domains – deficits in reciprocal social communication and social interaction (criteria A), and restricted and repetitive patterns of behaviours, interests and activities (criteria B). Language impairment is no longer included in the diagnostic criteria, but is included as a specifier. DSM-5 had eliminated strict age criteria that required delays in social interaction and communication to be apparent before age 3 years. Instead, the onset of these symptoms in the "early developmental period" was enough to fulfil the criteria, but DSM-5 had also noted that the symptoms may not be fully recognised until social demands exceed their capacities, or they may be masked by compensatory strategies in later life. Besides, diagnostic criteria may be met when restricted, repetitive patterns of behaviours, interests, or activities were clearly present during childhood or at some time in the past, even if symptoms had subsided at the time of diagnosis. Moreover, the occurrence of potential sensory abnormalities was now incorporated as behavioural symptoms in Criteria B. Diagnostic reporting now includes specifiers to further describe the symptomatology or important features that are relevant to the management of the individual's disorder. Finally, in DSM-5, comorbidities with other neurodevelopmental, mental or behavioural disorders are recognised, thus, it is possible for an individual to receive dual diagnoses for ASD with another neurodevelopmental disorder (for examples, Attention Deficit/ Hyperactivity, and Anxiety Disorders) if diagnostic criteria of both conditions are met.

Neurobiological aetiology of ASD

In their review on issues related to the diagnostic classifications in ASD,[4] pointed out that despite past decades of effort in understanding the genetics or neurobiological underpinnings of ASD, findings on neurobiological causes for ASD remained diverse and lacking in specificity to behavioural dimensions of ASD.

Both epidemiological evidence from family and twin studies were found to be associated with ASD have

provided a strong genetic component in the aetiology of ASD.[5] Multiple genes and recurrent genomic imbalances that implicated autism spectrum condition have been identified, collectively accounting for 10-20% of ASD cases, while many of these have also been found to be casually implicated in other neurodevelopmental disorders and epilepsy.[6,7]

At a neuropathological level, abnormalities have been identified in the structures and cortical connectivity in limbic system, cerebellum, cerebral neocortex, and other brain structures suggestive of a common neuropathology despite the highly variable genetics and phenotypes associated with the disorders, yet further research is needed to identify features specific to ASD as compared to other neuropsychiatric diseases.[5] Given the lack of reliable biomarkers that could differentiate patients with autism, diagnosis of ASD must be made based on behavioural manifestations.[8]

Triadic to dyadic approach in diagnosis

In DSM-5, diagnostic domains were reduced from three to two, focusing on social communication and social interaction deficits (Criteria A), as well as restricted, repetitive patterns of behaviours, interests, and activities (Criteria B). Social communication and social interaction are combined into one category under DSM-5, in recognition that communication is necessarily social in nature.[9] For a diagnosis of ASD, individuals must display all the three kinds of social and communication deficits listed under criterion A, and they are pervasive and sustained, and must be manifested across multiple contexts.

Specifically, deficits in social-emotional reciprocity refer to impaired ability to engage with others and share thoughts and feelings, and may range from showing little initiation of social interaction and no sharing of emotions in young children, to communications that are often one-sided, functional (e.g. to request and label) and lacking in reciprocity, to difficulties in processing and responding to complex social cues in older children and adults. Deficits in nonverbal communicative behaviours are manifested by absent, reduced or atypical use of eye contact, gestures, facial expressions, body orientation, or speech intonation. While impairments maybe relatively subtle within individual modes, they should be noticeable in poor integration of nonverbal communicative behaviours and speech for social communication. Deficits in developing, maintaining, and understanding relationships maybe manifested by absent, reduced, or atypical social interest, rejection of others, passivity, or inappropriate social approach. Lacking of social and imaginative play in young children is now incorporated under this criterion. In older, or higher functioning individuals, they may have a desire to establish friendships, but the individuals may not have a complete or realistic understanding of what friendship entails. They might also present difficulties in judging what behaviour is considered appropriate in different situations, or understanding the different ways that language may be used to communicate thoughts, and feelings effectively.

Language delay is no longer considered to be a diagnostic criterion of ASD given that it is not specific to the disorder while the language abilities of children with ASD are found to be varied and highly correlated with other factors such as their intellectual functioning.[4]

RRBs and sensory processing problems

DSM-5 has included atypical sensory responsiveness (hyper- or hypo- reactivity to sensory input) or unusual interest in sensory aspects of the environment as one of the four possible elements of which two must be met under domain of restricted, repetitive patterns of behaviour, interests, activities (RRB).[9]

Although atypical responses to sensory stimuli were also reported in people with intellectual disability and other neurodevelopmental disorders,[10] these responses were more common in ASD group compared to the special educational need group. Atypical sensory behaviour was reported in 92% of ASD children while 67% of children with special educational need without ASD. Greater sensory dysfunction was associated with increased autism severity (specifically restricted and repetitive behaviours) and behaviour problems (specifically emotional sub-score) on teacher and parent's rating on

the Strengths and Difficulties Questionnaires.[11]

In validation study of proposed DSM-5 criteria for ASD by Frazier et al,[12] adding sensory sensitivities and unusual interests as a RRB criterion improved sensitivity (0.81 versus 0.78) without substantially altering specificity. Sensitivity to high-functioning ASD could be maximised by including sensory sensitivities or unusual sensory interests.

Bishop et al[13] suggested that restricted and repetitive behaviours (RRBs) could be subdivided into repetitive sensory motor and insistence on sameness behaviours. Wigham[14] reported that there was evidence for direct paths from sensory under-responsiveness to both repetitive motor behaviours and insistence on sameness, and from sensory over-responsiveness to insistence on sameness behaviours. According to Burns,[15] reports of sensory abnormalities characteristic of ASD fall into 3 primary domains: hypersensitivity, hyposensitivity, and sensory-seeking behaviours. Hypersensitivity is the exaggerated behavioural responses to sensory stimuli.[16] Neuro-physiological explanation includes dysfunction in the parasympathetic nervous system,[17] frontal lobes,[18] limbic system/hippocampus,[19] and cerebellum,[20] physiological hyperarousal (i.e. increased sympathetic activity, insufficient vagal tone) in various clinical groups, especially under stressful situation.[21] Hyposensitivity is a lack or insufficiency of response to sensory stimuli.[16] Neurological explanation of this in ASD involves aberrant functional connectivity[22] that may arise early in development and involve a variety of networks and structures (amygdala, dorsolateral prefrontal cortex, parietal lobe, cerebellum and superior temporal sulcus) associated with orienting to novelty, multisensory integration and/or disengagement of visual attention.[23] Sensory seeking behaviour involves unusual actions that intensify or reinforce a sensory experience.[24] Study found evidence that sensory-seeking behaviours are related to the RRBs characterised by ASD.[25] Researchers suggested that RRBs may serve as a method of managing poorly regulated arousal levels associated with sensory processing abnormalities: RRBs may serve to self soothe, avoid or reduce stimulation, or alternatively to create stimulation,[26] but the mechanisms underlying this are not yet fully understood. Self-stimulating behaviours are thought to provide sensory stimulation to the individual and are often automatically reinforced.[27]

Failla[28] in his study found diminished white matter integrity in a group of children with ASD in two tracts conveying somatosensory information. One tract linked the somatosensory ventroposterolateral nucleus of the thalamus with primary somatosensory cortex and primarily carried detailed information for discriminative touch. The other tract linked the posterior insula with the anterior insula and would be expected to convey information about the affective nature of somatosensory input. The findings contributed to the understanding of the neural basis of emotional responses to touch in autism.

Demopoulos[29] concluded that individuals with agenesis of the corpus callosum (AgCC) were at increased risk of being diagnosed with autism or experiencing dysfunction in associated symptom domains. He suggested that atypical sensory processing and corpus callosum abnormalities in autism are related. Caregiver surveys of individuals with AgCC indicated that they experienced reduced pain perception and increased rates of hearing and vision problems. In recent work assessing atypical sensory behaviour in adolescents and adults with AgCC, individuals with partial and complete agenesis appeared to have higher threshold for registering sensory information. Furthermore, they showed atypical auditory processing and olfactory/gustatory behaviours relative to the normative sample. The pairing of structural and functional neuroimaging with sensory perception and processing tasks informed our understanding of the contribution of the corpus callosum to atypical sensory experience in autism.

There was significant association between pupillary light reflexes constriction amplitude and a set of sensory behaviours in the ASD group. Smaller pupillary light reflexes constriction amplitude suggested lower parasympathetic modulation. This implied that some atypical sensory behaviours in children with ASD could be associated with decreased parasympathetic modulation.[30]

Further research is needed for better understanding of the neurophysiological explanations of the sensory processing abnormalities of ASD.

Use of specifiers

Beside the changes in triadic to dyadic approach in diagnosis, another valuable addition is the use of "Specifiers" to further describe the symptomatology, or to provide a descriptive subtyping of the population. List of "Specifiers" includes recording the severity of cardinal symptoms, the current language and intellectual ability and the presence of concurrent genetic, medical or environmental condition.

The severity levels of cardinal symptoms are recorded through classifying the amount of support that the autism condition required in each of the 2 cardinal symptoms, which ranged from one to three on a scale. The three levels are namely Level 3: Requiring very substantial support; Level 2: Requiring substantial support; and Level 1: Requiring support.

While language delay is no longer considered to be part of the core symptoms of ASD, the presence or absence of "language impairment" is used as a specifier, namely "with or without accompanying language impairment", considering its relevance to the diagnosis and intervention strategies of the disorder.[4] To use the specifier of "with accompanying language impairment", the current level of verbal functioning should be further assessed and described, which helps to further define the variable level of language disability in ASD.

Besides, descriptors on concurrent medical and neuropsychiatric condition, genetic correlates or environmental factors also allow further dissection of the autism condition into well-defined subgroups.

This system of specifiers enables the recognition of essential shared features of the autism spectrum condition while also attempting to individualise diagnosis through dimensional descriptors.[31] As such, a single categorical diagnosis will be complemented with a clearer symptom description and impairment measurement.

Dual diagnosis

Numerous coexisting paediatric and neurodevelopmental conditions are strongly associated with ASD, and often cause substantial impact on the adjustment and prognosis of the persons with ASD.[8] Comorbidities of intellectual disabilities, attention-deficit / hyperactivity disorder (ADHD) and anxiety are discussed in the following paragraphs.

ASD and ADHD

ASD and ADHD are common psychiatric comorbidities to each other. In the DSM-IV-TR, ADHD Criterion E prohibits clinicians from making an ADHD diagnosis in the context of Autism Spectrum Disorder. In the DSM-5, this exclusionary criterion has been removed and clinicians are now able to make an ADHD diagnosis in an individual with ASD. An expert review wrote by Antshel et al[32] suggested that a majority of children with ASD (31-95%) have significant symptoms of inattention and/or hyperactivity/impulsivity but not all children with ASD have these symptoms. ADHD-Inattentive type is somewhat more common in ASD than ADHD-Combined type. In a retrospective chart review, it was found that approximately 25% of children with ASD meet DSM-IV-TR symptom and impairment criteria for ADHD combined type, and approximately 35% meet DSM-IV-TR symptoms and impairment criteria for ADHD inattentive type.[33,34] A recent Swedish register-based cohort study for people born between 1987 to 2006 suggested that individuals with ASD were at a higher risk for ADHD compared with individuals without ASD (odds ratio (OR) = 22.33, 95% confidence interval (CI): 21.77-22.92).[35]

ASD and anxiety

A review of 40 studies on the prevalence, phenomenology, and treatment of anxiety in youth with autism and related conditions such as Asperger's disorder indicated that between 11% and 84% of children with ASD experience some degree of impairing anxiety.[36] A meta-analysis combining the findings from 31 studies involving 2,121 young people (aged<18 years) estimated that approximately 40% of youth with ASD present with either clinically significant symptoms of anxiety or at least one

comorbid anxiety disorder.[37] In the studies examining diagnosed anxiety disorders in ASD, deBruin et al[38] found that slightly more than 55% of the sample met criteria for at least one anxiety disorder[36] and Simonoff et al reported an overall anxiety disorder diagnosis rate of almost 42%.[39] Among children with ASD, a study found that 46% of a sample size of 1,429 children were at or above the clinically elevated range on the Child Behavior Checklist (CBCL) Anxiety Problems scale, compared with only 9% of typically developing (TD) siblings.[40]

Across studies, the most frequently reported anxiety disorders and symptoms in children with ASD are specific phobia (29.8%), obsessive compulsive disorder (17.4%) and social anxiety disorder (16.6%). Anxiety disorders are much more frequent in children with ASD when compared to typically developing children, and comparable to the levels of anxiety found in typically developing children presenting to clinics with an anxiety disorder. The types of anxiety most commonly found in children with ASD appear to differ slightly when compared to other clinical samples. For example, youth with ASD present with more social-evaluative, situational, and medical fears, but fewer fears of harm or injury when compared to non-ASD young people with Down's syndrome. The type of anxiety problem is likely influenced by cognitive ability youth with ASD. It was suggested that a somewhat lower IQ may be a risk factor for anxiety in general and for social anxiety disorder specifically, while higher IQ may increase the risk for obsessive-compulsive disorder and separation anxiety disorder in ASD youth.[37]

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ASD Intervention Treatment and Its Local Application

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Although autism is a neurobiological disorder, its pathophysiology remains obscure, and psychological and educational interventions are currently the primary treatments for addressing the core deficits in children with autism spectrum disorder (ASD). The universally accepted goal is to improve the overall functional status of the child and to help parents cope with the stress of raising a child with ASD. To be effective, treatment has to be intensive, systematic and structured, involving family members, individualised for each child and family, and carried out in natural settings/ scenarios in order to enhance possible generalisation. A comprehensive intervention programme should address deficits in the area of social communication, language, play skills, maladaptive function and maladaptive behaviour.[1] They should offer an ongoing parent education component in which parents can enhance the child's acquisition of skills and help transfer the newly acquired skills to home and community settings. According to the Scottish Intercollegiate Guidelines Network (SIGN) guidelines 145: Assessment, diagnosis and interventions for autism spectrum disorders, parent mediated intervention programmes should be considered for children and young people of all ages who are affected by ASD, as they may help families interact with their children, promote development and increase parental satisfaction, empowerment and mental health.[2]

An important point to note is that the needs of families with individual suffering from ASD will change over time. Clinicians should develop a long-term collaboration with these families and realise that service utilization could be tailored to their specific needs in different developmental stages. For young children, the issues of diagnosis and identification of treatment programmes often will be the most important. For school-age children, social and behavioural issues typically become more prominent. For adolescents, vocational / prevocational training and thoughtful planning for independence / self-sufficiency are important.

In Hong Kong, upon diagnosis preschool children with special needs would be enrolled in one of the preschool rehabilitation programmes such as Special Child Care Centre (SCCC), Intergrated Programme in Kindergarten-cum-Child Care Centre (ICCC), Early Education and Training Centre (EETC) and On-site Pre-school Rehabilitation Services (OPRS) under the subvention of Social Welfare Department. Children in these programmes are provided with different level of therapeutic support and intervention. Children with more delayed development and significant behavioural problems will usually receive more intensive support in SCCC. These children are supported by creating a very structured learning environment which includes organisation of the physical environment, predictable sequence of activities, visual schedules, routines with flexibility, structured work / activity systems, and visually structured activities as suggested by the approach of Treatment and Education of Autistic and related Communication Handicapped Children (TEACCH).[3] Besides a structured environment, young children who do not have speech yet can be helped through the use of alternative communication modalities or Picture Exchange Communication System (PECS).[4] It is a systematic process that begins with teaching a child to exchange a single symbol (pictures) for a preferred item. This exchange process is then developed to enable a child to discriminate between symbols and make more complex communication acts such as asking and answering questions, or commenting. Symbols can be combined to make simple sentences. Parents can be trained to use this programme at home but it is often delivered / managed by a speech therapist.

Depending on the functioning and specific needs of these young children, some parents might opt for individual training such as early intensive behavioural interventions or parent-mediated interventions. Applied Behavioral Analysis (ABA)[5] is generally intensive and highly individualised, with up to 40 hours per week of one-to-one direct teaching, initially using discrete trials to teach simple skills and progressing to more complex skills such as initiating verbal behaviour. In certain situations, a functional analysis of the target behaviour is performed, in which patterns of reinforcement are identified and then various behavioural techniques are used to promote a desired behavioural alternative. Other parent-mediated interventions included Developmental, Individual Difference, Relationship-based Model (DIR)[6,7] (also known as Floor Time), Relationship Development Intervention (RDI).[8] They focus on promoting development by encouraging children to interact with parents and others through play. Parents, working with a consultant trained in these approaches, are trained in techniques and strategies that make use of everyday activities to support the child's social and emotional development.

In recent years, there are more options of naturalistic communication therapies available in Hong Kong. The Hanen program[9] specialises in training caregivers to facilitate language development in children from birth to six years of age, in which "More Than Words" is an intensive training programme for parents of pre-school children with autism. The aim of the programme is for parents to learn how to use their child's everyday activities as the context for learning to communicate. The programme teaches a group of parents in eight interactive classes and three individual in-home videotaping and coaching sessions. Preschool Autism Communication Trial and Therapy (PACT)[10] is another evidence-based communication therapy for children who are pre-verbal or learning language. The intervention involves working with adults (parents and teachers) in specifically targeted dyadic interaction and communication to achieve spontaneous and reciprocal child communication and language. The use of the video feedback methods aims to enhance adults' skills in observing, responding and eliciting social communication in motivational activities and natural daily routines of children.

For school age children, follow up supports are provided by child psychiatry departments under the Hospital Authority, school-based services and Non-Government Organisations (NGOs). Children with ASD often have comorbid conditions such as Attention Deficit Hyperactivity Disorder (ADHD), Anxiety Disorder, and sleep disorders. Psychopharmacologic treatments for these comorbid conditions are offered by child psychiatry departments in addition to group training and psychotherapy if necessary.

In view of the increasing number of ASD students in mainstream schools, The Hong Kong Jockey Club Charities Trust has initiated a project entitled "JC A-Connect: Jockey Club Autism Support Network" (JC A-Connect) in 2015 to enhance support for these students and their families and schools. The Trust is collaborating with the Faculty of Social Sciences of The University of Hong Kong, the Hong Kong Education Bureau (EDB), and eight NGOs: Caritas-Hong Kong, Heep Hong Society, Hong Kong Sheng Kung Hui Welfare Council Limited, Hong Kong Young Women's Christian Association, New Life Psychiatric Rehabilitation Association, SAHK, The Salvation Army, and Tung Wah Group of Hospitals to provide holistic support for children with ASD attending mainstream schools, and their parents. The programme offers school-based support for students with ASD in over 300 primary and secondary schools, giving support to parents and families through 18 satellite centres, as well as organising public education programmes with an aim to raise public awareness and understanding about ASD.

To further enhance the ability of educators and professionals in supporting students with ASD, a resource package of group training suggestions for students with ASD features was also created under project JC A-Connect and distributed to primary and secondary schools, university libraries, and resource centres of EDB in early 2019. The student programmes consist of small-group training over 3 years, providing explicit instruction that addresses skill deficits associated with ASD. The areas of skill deficits include learning and self-management, basic communication skills, conversational skills, theory of mind, social thinking, friendly behaviours, peer interaction, emotional understanding and regulation, conflict resolution, self-advocacy, peer integration, secondary school adjustment etc. There will be at least 18 hours of training per year. Each training programme is tailor-made for the four to six participating students. This model of support emphasises addressing individual needs, the enhancement of critical skills using evidence-based methods, and the enhancement of whole-school support as well as home-school collaboration.

Some of these evidence-based methods may target on developing social and communication skills namely Social Thinking, Social-Communication, Emotional Regulation and Transactional Support (SCERTS) and Program for the Education and Enrichment of Relational Skills (PEERS). The Social Thinking[11] methodology was created to help people develop social competencies, to better connect with others, and to experience greater well-being. The treatment frameworks and strategies encourage individuals to focus their social attention, interpret the social context, and socially problem-solve to figure out how to respond. Social Thinking's concepts and strategies are designed for people with social learning challenges who have normal intelligence and language skills, regardless of diagnostic label (ASD, ADHD, social communication disorders, or no diagnosis at all). It is being adopted into the mainstream classroom to encourage explicit social-emotional learning for all students. SCERTS[12] model is used to teach children how to regulate their emotions and communicate with others. SCERTS concentrates on three key areas: social communication, emotional regulation, and transactional support (providing helpful aids to communication and learning). The model incorporates aspects of different well-established autism therapies, in an individualised programme designed by parents and the child's therapist. Program for the Education and Enrichment of Relational Skills (PEERS®)[13] is a 16-week evidence-based social skills intervention for motivated adolescents in middle school or high school who are interested in learning ways to help them make and keep friends. During each group session adolescents are taught important social skills and are given the opportunity to practise these skills in session during socialization activities (e.g. playing sports, board games, etc.). Parents are taught how to assist their teens in making and keeping friends by providing feedback through coaching during weekly socialization homework assignments.

In contrast, the support for young adults with ASD in Hong Kong is relatively under-developed. Most of the programmes are in pilot phase and are self-financed projects of NGOs. Up till now, only two pilot projects are under the subvention of the Social Welfare Department. They provide support to high functioning ASD via prevocational training, supportive counseling and case management. After assessing the young adults' abilities of independent living, vocational skills, problem solving skills,

peer relationship, social communication, career planning, community and family support, they offer individual or group training that tailor the needs of the individual and his / her family. These NGOs would also actively connect with companies and employers to provide internships and job opportunities for these young people. Training and consultation would be provided to employers and frontline rehabilitation staff to promote workplace integration of ASD individuals. Follow up service such as phone contact, talks and seminars would also be arranged after successful work placement. Resource centres are opened to offer useful vocational information. On the other hand, the Selective Placement Division of the Labour Department could provide free recruitment service to employers and free employment service to job seekers with disabilities, including those with ASD. In this endeavor, a practical guide for employing ASD individuals is recently published to increase public awareness and community's acceptance of young people with different abilities so as to create a caring and inclusive society.

The above mentioned intervention approaches and programmes include those with long histories and which have been widely researched, while others are more recently developed and gaining public attention. With different empirical support on their effectiveness, the levels of evidence indeed vary among the approaches and programmes. Due diligence has to be exercised in understanding the theoretical mechanism and effectiveness of different treatment programmes in clinical application.

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Developmental Trajectories of Theory-of-mind Ability in Children with Autism Spectrum Disorder and in Typically Developing Children

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Theory-of-mind (ToM) deficit is one of the major neuropsychological theories that explains the social-cognitive impairments of individuals with Autism Spectrum Disorder (ASD). According to the ToM hypothesis proposed by Baron-Cohen et al,[1] individuals with ASD are said to be suffering from a "mind-blindness" that underlies their social and communication impairments. They have primary deficits in understanding that people's behaviours can be interpreted on the basis of mental or psychological states associated with their desires, beliefs, or knowledge.[2]

In fact, theory-of-mind deficit is not only limited to individuals with ASD, but also common among various neurodevelopmental disorders such as children with Attention Deficit-Hyperactivity Disorder (ADHD), Developmental Language Disorder (DLD) (previously known as Specific Language Impairment), congenital hearing impairment and visual impairment, where innate abilities for perceiving and processing social information and learning of mental states are impeded.[3]

ToM ability is reported to be universally present among humans across cultures, following similar, age-related developmental trajectories. An individual attains and masters such skill that matures naturally and spontaneously with age, though there is a disparity of the precise age for its achievement.[4]

Moreover, ToM is a composite function which involves memory, joint attention, complex perceptual recognition (such as face and gaze processing), language, executive functions (such as tracking of intentions and goals and moral reasoning, inhibition control), emotion processing-recognition, empathy, and imitation. Despite individual differences in its maturity, there are multiple external, social and environmental factors affecting its development, such as an individual's upbringing, parental practices, social background (such as parental use and exposure to mental state vocabularies in daily conversations), training and education etc. to name just a few.[3] Hence, ToM development is dependent on the maturation of several brain systems and is shaped by social environment. In order to have better understanding of how children with ASD differ from typically developing children in social communication, the trajectory of theory-of-mind development in typically developing children is reviewed below.

Developmental trajectory of ToM ability

Research suggests that typically developing children attain ToM at roughly 3 to 4 years old through a progression of stages starting at around 18 months with awareness that their own mental states are distinct from those of others.

Early precursors of ToM skills as identified by researchers include the ability to use eye gaze, and to participate in joint attention, imitation and pretend play.[5-10]

An early sign of emerging ToM is the ability to engage in joint attention. This is a skill that is usually well developed at around 9-12 months of age.[7,11] Some researchers suggest that pretend play itself may be a precursor to ToM abilities as both require the skill of meta-representation.[7,10,12,13] Pretend play (pretense) develops and emerges in children from around 18 months to 2 years of age.[8,12] According to Leslie,[12] typically developing children demonstrate the ability to change the function of an object (substitution), attribute properties to an object that it does not have and refer to an absent object as if it is present (for example taking a banana as telephone).

The next stage in the development of ToM is an awareness of what another person can see: their visual perspective and knowledge.[14] This ability is usually present in 2-year-old children and becomes the basis of simple social games, such as hide-and-seek with an object. Around 3 years of age, other important indicators

of the precursors of ToM abilities are imaginative or make-believe play, where a doll or figure represents a person with thoughts and experiences, and the use of mental state terms in speech (with the correct use of words such as believe, think, know, feel, etc.).

The third developmental stage of ToM abilities involves the understanding of another person's desires and emotions, which helps to explain the behaviours and intentions of other people. For some children, the ability to understand basic desires occurs around the age of 2. At around the same age, children are able to identify basic facial expressions, such as happy, sad, angry, and scared. By the age of 4, they can understand whether another person is likely to express these feelings in everyday situations. Moreover, at 4 to 6 years of age, children display a great leap in social cognitive functioning and demonstrate a higher level of sophistication. Most children can attribute mistaken beliefs to themselves and to others, and so begin to show new and advanced forms of social interaction; these include performing tricks, jokes, and deception (e.g., telling lies).[14-17]

At school age (i.e., age 6 and above), a later stage of development, children's knowledge about mental representation is characterised by: (a) the role of preexisting biases and expectations in influencing both personal preferences, and how people interpret either ambiguous events or moral dilemmas of truth and rightness (i.e., prejudice, irony, mockery)[18] (b) subtle forms of social deception such as bluffs and white lies[16] and (c) mixed, multiple, and ambivalent emotions (e.g., embarrassment, jealousy).[19]

Theory of mind deficits among children with ASD

From the above review, it is not difficult to appreciate that for most children with ASD, their ToM development deviates from the typical developmental trajectory. Children with ASD show delays in imaginative play and are less likely to use mental state terms in their speech.[11] They also participate in significantly less instances of spontaneous pretend play than developmentally delayed or typically developing children of comparable mental age.[5,8]

There is also a large body of research evidence that suggests that individuals with ASD are impaired in their understanding of mental states and in their ability to recognise and attribute thoughts and feelings in order to make sense of how other people act.[1,16,20] In daily social communication, pragmatic difficulties are commonly encountered by children with ASD.[1,21,22] For example, they have difficulties with imaginary play, and in comprehending humor or figures of speech (e.g., irony). They are often noted to have difficulty in comprehending the underlying messages of a conversation, or have problems in perceiving the informational needs of others and maintaining topics of mutual interest in conversations.

Putting together what we have learnt about theory-of-mind development among typically developing children, and specific ToM deficits found among young children with ASD, it would be useful to examine those developmentally appropriate ToM skills in aiding early identification of at risk cases of ASD. In fact, difficulty in theory- of-mind skills among ASD individuals is not only evident at behavioural levels, but also at neural levels as supported by a number of neurobiological studies.[23-25] Further understanding of possible neural basis of theory-of-mind deficits specific to ASD individuals will enrich our understanding of the disorder per se. Finally, as theory-of-mind deficit is not unique among individuals of ASD but also found in other clinical groups, it will be equally important to study how other clinical groups (such as ADHD, DLD, and congenital sensory impairment, etc) perform on those early ToM skills in making differential diagnosis.

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Development of Hong Kong Battery of Theory of Mind Tasks for Children (AToM)

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In western literature, theory-of-mind tasks derived from the study of the social cognitive difficulties among children with ASD. Classic design of false belief tasks, such as the Sally & Anne Task and the Smartie Task were used in studying ToM ability among typically developing children and their ASD counterparts. Some researchers have reported that between 15% and 55% of children with ASD passed first-order belief ToM tasks[1,2] and some children with ASD passed the second-order ToM tasks.[3] Happé[4] developed a higher order false-belief task, the Advanced Strange Stories task, that requires a participant to utilise mind-reading abilities which use relatively more complicated story contexts taken from daily-life scenarios. It has been shown that typically developing children are able to pass these tasks following certain developmental milestones: at around 4 to 6 years of age for first-order false-belief tasks, and around the age of 10 to 12 for strange stories.[5] However, some children with ASD continue to fail this task long after they reach school age.

Wellman and Liu[6] looked into the conceptual changes of different aspects of ToM components, using the ToM Scale. They found a consistent progression of conceptual achievements that pace ToM understanding in typically developing children: diverse desires > diverse beliefs > knowledge access > false belief > hidden emotion. They argue that the ToM developmental order is not one of addition or substitution, but one of modification or mediation, following orderly conceptual progressions.

Liu et al[7] further conducted a meta-analysis of children's false-belief performance which provided the most comprehensive examination to date of theory-of-mind development in a population of non-Western children speaking non-Indo-European languages (i.e., Mandarin and Cantonese). The meta-analysis consisted of 196 Chinese conditions (127 from mainland China and 69 from Hong Kong), representing responses of more than 3,000 children, compared with 155 similar North American conditions (83 conditions from the United States and 72 conditions from Canada). The findings showed parallel developmental trajectories of false-belief understanding for children in China and North America coupled with significant differences in the timing of development across communities, where children's false-belief performance varied across different locales by as much as two or more years. It supports the importance of both universal trajectories and specific language, cultural and experiential factors in the development of theory of mind.

With the described trajectories for ToM development, a battery of ToM tasks is required for reflecting an individual's social cognitive profile. Moreover, the ability to pass laboratory-based theory of mind testing does not imply that the person is able to perform as well in daily life settings. Theory of Mind Task Battery (ToMTB), is a direct assessment of children's ToM ability developed and validated by Hutchins, & Prelock in 2010. It was normed on 96 children aged between 2 to 13 including children with ASD and typically developing children. It was co-normed with an assisted Theory-of-mind Inventory-2 (ToMI-2) that serves as a more comprehensive parent-informant measure of ToM.

In Hong Kong, empirical research on theory-of-mind development among Chinese children is limited. Most well-developed laboratory-based theory-of-mind tasks originated mainly from Western studies of the socio-cognitive aspect of children with ASD, yet most of these tasks do not have normative reference, which make inferences of the child's performance as compared with typically developing individual difficult and invalid. On top of that, the influence of cultural and language factors were reported to be closely related to ToM development by previous research. The development of culturally validated tasks is thus undeniably desirable. Moreover, it is believed that a battery of theory-of-mind tasks covering different aspects of theory-of-mind ability might better measure a child's overall social cognitive development than a single task alone. With the purpose to develop a comprehensive assessment of a child's theory-of-mind ability, a working group consisting of paediatricians, speech therapists and clinical psychologists from Child Assessment Service (CAS) together with scholars from the University of Hong Kong was formed in 2008.

The Hong Kong Assessment Scale of Children's Theory of Mind (HKAToM) is a locally developed, validated and standardised instrument for understanding theory -of-mind ability of Hong Kong children (aged between 4 to 12). It was designed for use by clinicians including developmental-behavioural paediatricians, child neurologists, clinical and educational psychologists, as well as speech therapists working for preschool and school age children.

The tool

Supported by a thorough review of the western ToM literature, seven subtests of the HKAToM were developed to measure different facets of theory-of-mind abilities. These include (i) Understanding of First-order false-belief, ii) Use of emotion vocabularies, (iii &iv) Understanding of perspectives and recognition of changed emotions, (v) Sabotage and deception, (vi) Use of figurative language, and (vii) Use of language in pragmatic situations. With construct validity analysis, the performance of an individual measured by the seven subtests could be explained by a single unitary factor. The total score on the seven subtests which yields a standard score and percentile rank can reflect the general theory-of-mind ability of a child among same-aged peers in the standardization sample. With a stratified random sampling method based on three variables, including school districts (Hong Kong Island, Kowloon and New Territories), age (7 age groups) and gender, 820 typically developing children aged between 4 years 11 months to 12 years 4 months old were selected from six primary schools, and 5 kindergartens/ nurseries (covering K2 to P.6) from the above three districts and included in the large-scale norming sample.

Results

Subject's performance on the seven subtests and the total score demonstrated significant growth across different age groups. Using the 75% acquisition criterion, children showed mastery in the understanding of first-order false belief (Section ii) at age 6. The next easiest task was (Section v) in which children at age 7 can understand sabotage and deception. At about age 8, most children could recognise change of emotion of a person in different contexts (Section iii & iv) and perform higher order mental state reasoning (Section vi). Ability to use appropriate language in pragmatic situations (Section vii) and emotion vocabularies (Section i) appeared to be relatively well-matured by age 9. Finally, understanding of different people's emotions or perspectives in the same scenario (Section ii) may be achieved until at age 10.

Conclusion

With the development of HKAToM, normative data for the developmental trajectory of theory-of-mind ability among Hong Kong children was developed. It sets the cornerstones for collecting useful information in better understanding the strengths and weaknesses in an individual's social cognitive profile, including those with neurodevelopmental disorders. It also paves the way for early identification of developmentally appropriate targets of intervention for those who demonstrate theory-of-mind deficit and ensuing functional difficulties.

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Recent Publication and Scientific Presentations

Publication

Leung C, Leung S, Lee F, Lo SK. Socioeconomic difference in development among preschool children. HK J Paediatr (new series) 2020;25:98-106.

Scientific Presentations

Sharing of the Child Assessment Service in Hong Kong on 16 April 2020 at School of Medical and Health Sciences, Tung Wah College by LIN Sik-ying, Lenzs.

Intellectual assessment and assessment of adaptive functioning of children with physical impairment and multiple disabilities on 18 December 2019 at Master of Social Science Program in Clinical Psychology, Department of Psychology, University of Hong Kong by CHEN Yuk-ki, Theresa.

Assessment and referral for non-Chinese speaking children with special education needs on 3 December 2019 at The Hong Kong Council of Social Service by Dr CHOW Chin-pang.

Assessment of speech and language abilities for school-aged children on 14 November 2019 at Department of Special Education and Counselling, The Education University of Hong Kong by CHAN Wai-ki, Amy.

Mathematics disability on 13 November 2019 at Master of Arts Program in Professional Educational Psychology, Faculty of Education, The Chinese University of Hong Kong and Master of Educational and Child Psychology, Hong Kong Polytechnic University by CHAN Mee-yin, Becky.

General approach to clinical assessment of children and assessment of behavioural, social and emotional aspects of children on 23 October 2019 at Department of Psychology, The University of Hong Kong by CHAN Mee-yin, Becky.

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