Behavioural phenotypes: The changing role of the neurodevelopmental psychiatrist; genetic simplification only serves to illuminate complexity.

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The authors reflect on the importance of understanding behavioural phenotypes based on the first author's six month fellowship in dual training in Child Psychiatry and Paediatrics, in the Developmental Neuropsychiatry Team in the Department of Psychological Medicine at the Children's Hospital at Westmead.

The team at the Children's Hospital at Westmead provides a tertiary/quaternary service for psychiatric disturbance in the developmental context of Autism and/or Intellectual Disability. The team provides opinions to paediatricians and child psychiatrists who continue to case manage the referral, as a measure of their special interest, with priority given to the paediatricians of the Children's Hospital at Westmead.

Behavioural problems first need assessment and treatment by disability services, now funded through the National Disability Insurance Scheme (NDIS), which now means that behavioural support services are generally not provided until an annual review of the NDIS package has recognised the need for intensive and multidisciplinary support or a review is requested mid-plan. Conversely, there are many cases that have failed to improve despite input from behaviour support, psychology, speech therapy and occupational therapy. In those with more severe disturbance, a psychiatric assessment is required, and often these cases also need child mental health skills in family and relationship assessment. This is a vexed area, as while there is clinical consensus on the prevalence of multiple psychiatric diagnoses in these cases, the lack of communication skills in the young person can lead to diagnostic overshadowing, attributing disturbance to the developmental disability, and difficulties in reliability of diagnosis. However, one hears of many cases with clear psychiatric disorder being refused a psychiatric service, which is unethical, immoral and illegal under the Disabilities Inclusion Act, 2014 (https://www.adhc.nsw.gov.au/about_us/legislation_agreements_partnerships/nsw_disability_inclusion_act) and Disability Discrimination Act, 1992 (http://www.pwd.org.au/student-section/key-pieces-of-legislation.html#dda1992) .

Although sometimes the behaviours relate to emerging mental health issues, sometimes they are related to physical health issues, and need close collaboration,

and even to be led by a general paediatric, neurology or other subspecialty paediatric service. At times, problems arise related to a long standing behavioural pattern getting worse with onset of puberty and growth spurts. Regardless of the cause, these issues have enormous impact on the parents and family well-being. Often we need to support parents to navigate the NDIS and sometimes even the empathic appreciation of the adverse predicament can have a huge effect on families' well-being and capacity for survival. In the presence of longstanding severe disturbance, families really value even moderate improvements, as it can make continuing to care for their child a bit longer possible, e.g. in helping sleep disturbance or reducing the severity and frequency of violent behaviours.

Paediatric training and exposure to developmental paediatrics confirmed an interest in Autism and intellectual disability for the first author; Dr Singhal. However this term's experience has determined her future subspecialty career pathway in developmental neuropsychiatry. During the term, we came across a series of cases with behavioural phenotypes, some were textbook descriptions and some were extremely rare with their own unique challenges. This increased Dr Singhal's curiosity in the concept of behavioural phenotypes and describing the experience. The definition of behavioural phenotypes can be described as "behaviour, including cognitive processes and social interaction style that is consistently associated with, and specific to, a syndrome which has a chromosomal or genetic aetiology" (Skuse, 2000). Such syndrome-based patterns of behaviour provide insight to the biological underpinning of the human behaviour. How the genes influence protein production, proteins influence the neurophysiology, and neurophysiology influences the behaviour in a trickle down sort of a chain reaction. This sequence is made more complex by the growth of epigenetics, ie. attempts to understand what turns a gene on and off in the course of development and the environmental contributors to epigenetics. As a result, study of these minority populations of behavioural phenotypes takes one to the cutting edge of neuropsychiatry and the gene revolution.

In a 6 month period we saw around 25 cases with different behavioural phenotypes. With the rising awareness of the severity of psychiatric disorder in young people with neurodevelopmental disorder, any of these can present to a mainstream child and adolescent psychiatry service. Conversely, the speed of development in genetics leads to identifying new genetic disorders and new behavioural phenotypes at such a quick rate that it is a common event to be referred a new disorder that we haven't previously heard of.

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While some conditions, such as Fragile X, Prader Willi, tuberose sclerosis or complex epilepsy, may have the benefit of specialist syndrome-based clinics, often supported by a parent group, such a notion for all behavioural phenotypes in the resource-poor public mental health system is fanciful. However, equity of access means that those with ASD/ID need 3-4 times greater access than a mainstream population. Accordingly, any child or adolescent mental health clinician may need to be prepared to consult to the mental health problems of a well-established or a newly recognised behavioural phenotype. No psychiatrist can keep up to date with this rate of change, and a search on Google before seeing a new behavioural phenotype can quickly arm you with a global case series of health and mental health vulnerabilities. Although such special knowledge may make a clinician feel disarmed, especially if the family come bearing several of the latest scientific articles, as psychiatrists, we bring a range of skills to formulate and make diagnostic sense of severe mental health scenarios.

In this article we aim to provide some clinical insights, partly from newer scientific reports from developmental neuropsychiatry and partly based on some case examples. Of the 25 behavioural phenotype cases we managed over the six months, some are comparatively common ones that all mental health clinicians should be aware of, such as:

- Fragile X (CGG repeats in X Chromosome, most common inherited cause of ID) (www.fragilex.org.au)
- Down syndrome (trisomy 21), (a case presented with developmental decline secondary to Catatonia due to Major Depression (JMHCAIDD, 2017, 8 (1): 4-10),
- Noonan's syndrome ('male Turner's Syndrome') with autosomal dominant genetic defect (mostly causing mutations in the Ras/mitogen activated protein kinase),
- Velocardiofacial sydrome (22q11.2 deletion) associated with marked increased rate of psychosis in adolescence/adulthood predicted by cognitive decline; also increased ASD, anxiety and ADHD in childhood, (JMHCAIDD, 2014, 5(2): 4-7)

“All these conditions have raised rates of anxiety and ADHD”

- Williams syndrome (deletion on chromosome 7q), cocktail party chatter, sound sensitivity and visual-spatial problems, and provides models of different types of anxiety eg thunder storms vs fear of heights.
- Fetal Alcohol Spectrum Disorder, complex uneven developmental problems with major self-regulation problems and ODD and major depression in adolescence. (School-link Newsletter 2012. 3(2): 2-5.)

All these conditions have raised rates of anxiety and ADHD. Amongst those that are less common syndromes and scenarios were:

- Kleefstra syndrome (intellectual disability, limited or absent speech, hypotonia, microcephaly, characteristic facies and childhood obesity),
- Sanfilippo syndrome or Mucopolysacharidosis type III, a lysosomal storage disease that may present with severe hyperactivity in the context of a slow developmental fatal decline
- 18p deletion (intellectual disability, behavioural problems and distinctive facial features) (see Euan’s story in JMHCAIDD, 2017, 8 (2))
- 16p11.2 deletion (may have mild ID, language delay, ASD and motor impairments)
- Merosin deficient muscular dystrophy (a severe congenital muscular dystrophy; can lead to problems with contractures, sleeping and breathing and feeding)
- Landau-Kleffner syndrome (rare complex epilepsy, progressive loss of speech, ADHD, often with severe intellectual disability); could night time epilepsy in a teen that is difficult to assess with EEG be causing sleep disturbance that generates other major psychiatric disturbance?
- Kluver-Bucy syndrome (neuropsychiatric rather that genetic): compulsive eating, hypersexuality, hyperorality, visual agnosia and docility from bilateral lesions of medial temporal lobe and amygdala
- New behavioural challenges in post stereotactic surgery for complex epilepsy, with resultant lobectomy, hemispherectomy, corpus callosectomy mostly in context pre-surgical complex developmental disorders.

Below we present some brief case summaries with learning points.

A case that confirms that describing complex mental health co-morbidity has a value.

A 13 year old apparently compliant boy presented with mild-moderate learning difficulty, autistic rigidity, behavioural issues and major attentional problems. He was a day dreamer (not due to absence seizure). He appeared to be lacking motivation, yet showed significant planning skills in his antisocial behaviour eg stealing from his grandmother, significant violence to her and outrageous lying eg late for school because ‘his aunt had died’. He also had some subtle unusual facial and physical features. He lived with his grandmother, who was his main stable carer. We diagnosed and treated his ADHD with a range of medications, but he continued to be antisocial despite a good care environment. His mother had a similar history of school problems and strange asocial and impulsive behaviours, for example she still disappears for six months at a time without warning. We also treated his mother for her ADHD to try and help her and improve her influence on him. We organised a genetic microarray and he was found to have a 16p11.2 microdeletion. The gene testing helped clinicians and custodial grandparent to have an explanation of the features of his presentation. The genetic testing and behavioural phenotyping provided an explanation on “why he was the way he was.” Knowing the genetic cause of his presentation had a huge positive impact on family and they “did not need to feel guilty or feel they were doing something wrong”. Further, the behavioural phenotype was characteristic of the above mixture of learning problems, ADHD, and ASD features and oppositional behaviour. This affirmed that the multidimensional psychiatric diagnosis was an accurate description. Nonetheless he continued to cause problems and some sexual inappropriate behaviour led to his expulsion from school. His uncle subsequently provided him with a positive relationship, daytime supervision and sheltered employment. His grandmother benefitted from toughening up on her behavioural management skills with good effect, reducing the need for some of his medications. This was a good example of the positive impact of a behavioural phenotype, adding meaning and explanation to behaviour that didn’t fit the socio-cultural environment.

A rare complexity:

FLVCR1 gene mutation is autosomal recessive, one of five reported cases in the world literature. The presenting six year old girl had severe intellectual disability, was still learning to sit up, unable to feed self or otherwise fend for self, growth retardation (10kgm); retinitis pigmentosa and blindness; peripheral neuropathy and

inability to feel pain; sensory ataxia from degeneration of posterior columns of spinal cord; scoliosis, camptodactyly, achalasia and gastrointestinal dysmotility, needing supplementary feeding by a gastric peg, and sensitivity to most foods causing frequent diarrhoea. She was referred because of self-injury of biting her fingers, leading to loss of her teeth, largely managed with arm splints. She also had sound sensitivity leading to waking at night. She was managed by five teams: general paediatrics, neurology, gastroenterology, brain rehabilitation and palliative care. She is loved by remarkable parents who give so much attention. She communicates needs with blowing raspberries, responds to sensory input with cuddles and music, and has a vocabulary of over 100 words and can make choices. Psychotropic medication may still have a role in sleep management (clonidine) and could contribute to reducing self-injury (naltrexone).

A rare clinical predicament led to a new approach to medical decision making:

A nine year old girl with PCDH 19 mutation, a rare (approximately 150 cases identified but with many different identified mutations and deletions of this gene on X chromosome at q22.1) congenital complex epileptic condition in girls with developmental delay and behavioural difficulties including autism, aggression, stripping and self-injury. The patient was severely intellectually retarded, with limited language, mainly echolalia. She needed multiple admissions for uncontrolled complex seizures/status. She had tried multiple antiepileptic medications singly and in combinations (including newer treatments such as ganaloxone, rufinamide and cannabinoids) and psychotropic medications (including stimulants, major tranquilisers, mood stabilisers and melatonin) to help with her intense agitation and sleep disturbance. Her mother was very involved in her care and has been to the bi-annual syndrome specific conferences in the USA. The mother wished to withdraw the girl from all anticonvulsants, because they appeared ineffective and she was concerned they made her behaviour worse. Her neurologist was concerned there was an increased risk of sudden death in epilepsy on no anticonvulsants. This difference of opinion in a clinically unusual situation led to a multidisciplinary review, led by the department of medical ethics. This enabled the consideration of opposing legal concerns of informed consent versus clinical responsibility to act in the best interests of the child. Balancing the issues of quality of life for the child, versus the risk of no anticonvulsant led to an agreement to treat seizures only when needed with Midazolam and has been associated over time with an improvement in the frequency and severity of the seizures but not the behaviour disturbance.

Both hormones and pharmacology have a psychological benefit in Prader Willi Syndrome (PWS)?

A 15 year boy with PWS , mild intellectual disability, ADHD and anxiety was having difficulty in mainstream education, and was troubled by explosiveness in school and school refusal. He also had social disinhibition and social immaturity, rigidity and perseveration. A change of school routine or teacher could make him so distressed that he would leave his class and could not be persuaded to return. His diet was managed actively with restricted access to food, not just for him but also his younger brother who had Sotos' syndrome. He was helped by moving to a support class, where he was able to show greater relative competence and confidence. He wasn't helped by two different SSRIs, clonidine made him sleepy, both risperidone and quetiapine were helpful but led to problems with weight gain. He got significant benefit from a small dose of propranolol of 10mg, but higher doses made him feel dizzy. His weight and appetite has been most significantly been helped by growth hormone injections but now he has stopped growing in height, he is now only entitled to growth hormone on a private script of $800/month. He is due to start testosterone injections for bone health and density.

Could this complex case be Smith Magenis syndrome?

A 13 year boy presented with early onset of severe violence in context of brain damage of prematurity, bor-

derline intellectual disability, ASD and difficult to treat complex partial epilepsy. He had a psychiatric inpatient admission for acute suicidality and violence at 10 years of age, but because of his age he was 'not entitled' to psychiatric service follow up! He presented with problems of anger, mood regulation, and auditory and visual hallucinations which appeared to be mood related. He would get quite paranoid and misinterpret social situations. He also got flashbacks of traumatic experiences. He had a preoccupation with violence, despite his parents' efforts to modify his media access. His mother has Aspergers and a strong maternal family history of bipolar disorder and actual suicide. There was no contact with father who had history of ADHD, gambling and substance abuse. His behavioural phenotype, facial appearance and his self-injury of pulling off his toe nails to eat them raised a question of Smith-Magenis syndrome. Initial gene probe was negative, but we are checking for less frequent gene subtypes. His management has stabilised with excellent parenting skills and behaviour management from a school for emotional disturbance. NDIS has funded occupational therapy, emotion-based social learning, socialisation through 'Jedi training camp' and learning the piano. Medication for ADHD, anxiety, sleep disorder and mood instability included: mirtazapine at night, carbamazepine, clonidine day and night, risperidone and PRN quetiapine. Working with cases of Smith Magenis syndrome emphasises the importance of behavioural management plus active multimodal management approaches to create the art of possible.

A child psychiatrist/mental health clinician should be familiar with the most common behavioural phenotypes, but here are some of valuable specific characteristic clinical features that have been described by researchers in recent years.

- 15% of people with Down syndrome have autism, which makes them a challenge to care for.
- As young people with Fragile X become older, their developmental trajectory slows and they have an increased risk of being diagnosed with autism; carriers can have Tremor/Ataxia syndrome with memory problems.
- People with Prader Willi syndrome have problems of shifting attention which leads to increased aggression, and the need for careful warning processes for change of routine.
- Any neurodevelopmental disorder increases your risk of catatonia.
- Young people with Smith Magenis syndrome have increased rates of autism and autistic spectrum features, but also have a strong attachment to their primary carer and separation anxiety contributing to greater violence to parents than other adults.
- Self-injury in people with Cornelia de Lange's syndrome is usually caused by the inability to express pain, e.g. from gastro-oesophageal reflux, dental/sinus pain. (A frequent challenge in those with non-verbal Autism).
- There is an awareness of high rates of ADHD in all people with neurodevelopmental disorders, but Developmental Coordination Disorder has less attention paid to it, but can add increased functional disability and dependency especially for independence skills.
- Angelman's syndrome (Happy Puppet Syndrome) includes unusual laughter which is exaggerated by the environment, and seeking of attention and affection especially in the presence of adults. Genetic disposition provides survival advantage to illicit care and can be modified by change in environment.
- In PKU (Phenylketonuria) early intervention with a phenyl alanine diet prevents intellectual disability, but later childhood/teens levels of phenylalanine can contribute to features of ADHD. Phenylalanine in the diet of a mother with PKU can also cause problems for a genetically normal fetus.
- Behavioural Phenotypes illustrates different types of self-injury with different genetic disorders, such as eating lips in Lesch Nyhan syndrome (related to receptor hypersensitivity); Cornelia De-Lange syndrome (pain related to gastro-oesophageal reflux); Smith Magenis syndrome (putting objects in orifices) or tearing off toe nails. One still has to look for psychiatric co-morbidity, for example unrecognized anxiety or depression e.g. in Fragile X.

"A child psychiatrist/mental health clinician should be familiar with the most common behavioural phenotypes"

Will the genetic revolution make psychiatric disorder redundant?

These scenarios and cases provide diagnostic, management, medicolegal and ethical learning points. They illustrate some of the complex co-morbidities and aetiologies. New genetics syndromes present new or unique clinical situations, some of which require novel approaches. In the context of such novelty psychiatrists/mental health clinicians bring a range of integrative skills to diagnosis and formulation that are helpful and insightful. This includes medical/psychiatric

knowledge, family, parental and relationship assessment skills and attention to co-morbidity, plus psychopharmacological experience. These cases, while benefitting from genetic research, still depend on traditional approaches to child and family assessment. Behavioural Phenotypes have become an established part of child neuropsychiatry but recent developments provide more detailed understanding of the clinical aetiologies, often for different symptoms in the same genetic disorder and appreciating greater individual differences.

Some argue that the new Diagnostic and Statistical Manual (DSM), although increasingly reliable, lacks biological validity. Some are quick to say that the DSM will soon be redundant, to be replaced by a 'disruptive technology' research approach of the Research Domain of Criteria (RDoC) a project from the US National Institute of Mental Health, based on genetic/biometric data (https://www.nimh.nih.gov/research-priorities/rdoc/index.shtml).

In 2013, NIMH director, Thomas Insel, published a blog post critical of the DSM methodology and highlighting the improvement offered by the RDoC project. The RDoC approach is based on the biology as well as the symptoms and not be constrained by the current DSM categories. "Mental disorders are biological disorders involving brain circuits that implicate specific domains of cognition, emotion, or behaviour. Each level of analysis needs to be understood across a dimension of function. Mapping the cognitive, circuit, and genetic aspects of mental disorders will yield new and better targets for treatment." RDoC dimensional **psychological constructs (or concepts),** relevant to human behaviour and mental disorders, are measured using multiple methodologies and as studied within the essential contexts of developmental trajectories and environmental influences. Constructs are in turn grouped into higher-level **domains of human behaviour and functioning** that reflect contemporary knowledge about major systems of emotion, cognition, motivation, and social behaviour. The major RDoC research **domains/constructs**:

- **Negative Valence Systems**

Fear, Anxiety, Loss, Frustrated, Non-reward

- **Positive Valence Systems**

Reward learning, Reward valuation, Habits

- **Cognitive Systems**

Attention, Perception, Declarative Memory, Working Memory, Cognitive control

- **Systems for Social Processes**

Attachment formation, Social Communication, Perception of self, Perception of others

- **Arousal/Modulatory Systems**

Arousal, Circadian rhythm, Sleep and wakefulness

The domains are tentative: “It is important to emphasize that these particular domains and constructs are simply starting points that are not definitive or set in concrete. Methods used to investigate and understand constructs (termed “**units of analysis**”) can include molecules, genes, cells, neuro-circuits and behaviours, self-reports and paradigms.” In these terms schizophrenia can be seen as an information processing disorder, prioritising the importance of motivational, cognitive and social dysfunction. So far, there is no indication whether these approaches will be helpful to anyone.

Remember the limitations to genetic explanations

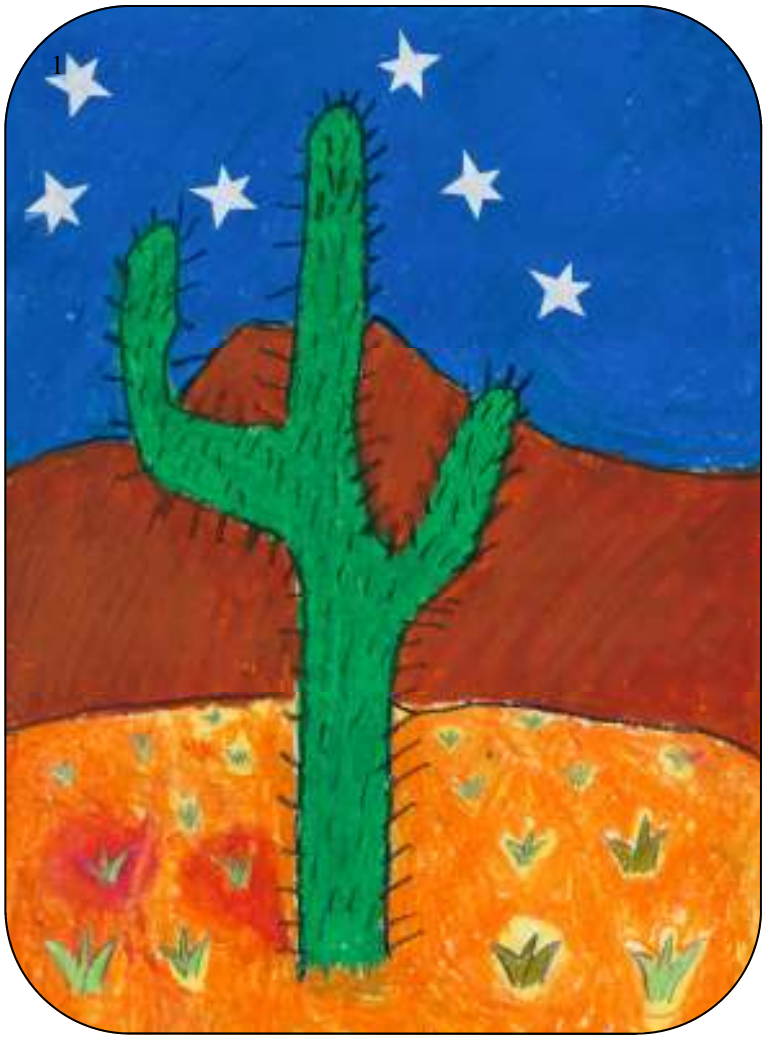
6-Pyruvoyltetrahydropterin Synthase *(6-PTS) Deficiency* is one rare behavioural phenotype series managed at the Children’s Hospital. *This* autosomal recessive disorder causes malignant hyperphenylalaninemia due to tetrahydrobiopterin deficiency. This enzyme is necessary for the creation of Serotonin, Dopamine and Noradrenaline, which are arguably the 3 neurotransmitters involved in all psychiatric disorders. Commonly reported symptoms are truncal and appendiclar hypertonia, bradykinesia, cogwheel rigidity, generalised dystonia, and marked diurnal fluctuation. They become confined to bed, immobile and unable to communicate. Other reported clinical features include difficulty in swallowing, oculogyric crises somnolence, irritability, hyperthermia,
and seizures. Chorea, athetosis, hypersalivation and sudden death have also been reported. Treatment involves substitution with neurotransmitter precursors (levodopa, 5-hydroxytryptophan), monoamine oxidase inhibitors, and tetrahydrobiopterin. Response to treatment is remarkable, with patients resuming normal intellect and daily competences, although long-term and functional outcome is variable and remains uncertain.

One case from the series was referred to the Developmental Psychiatry Team, because of the violent behaviour in the context of moderate intellectual disability. Lengthy assessment and treatment concluded that the behavioural phenotype was not the cause of the violence but that is was due to the deficits of behaviour management, style of parenting and marital problems. Accordingly, even in context of an overwhelming biological factor, environmental factors can be the primary determinant of psychiatric disorder.

Could we be throwing the baby out with the bathwater? Is this rush of enthusiasm for genetic biases and explanations of ‘life’s rich tapestry’ instead of the patience needed to build an understanding of the family context, leading paediatricians to be less inclined to consider family relationships, and parental mental health in their comprehensive clinical assessments? Even worse, does this biological theoretical practice bias, at the risk of neglect of the social context, lead in some cases to excessive medicalisation of dysfunction or even factitious disorder and potentially medical child abuse?

Conclusion

The cases presented confirm the value of the current diagnostic system. Indeed our capacities to be helpful confirms its predictive validity. The tried and tested model of case series of behavioural phenotypes illustrates the importance of applying clinical descriptive research. Currently, child psychiatry services are overwhelmed, with a shortage of, and inadequate training and recruiting of, psychiatrists. At the same time, political emphasis is on client-led adult recovery services. These changes indicate a downgrading of the importance of science and medicine in Mental Health. The study of psychiatric disorder has been a revolution of understanding and helping people with such disorders and enabling them to return to the mainstream of society. In particular, people are seldom institutionalised due to the advances of psychiatric treatment. Scientific methods include the progressive delineation of subtypes and validity of psychiatric disorder over time,



> “Both paediatrics and child mental health clinicians need to continue to collaborate”

just as in any medical specialty. There is an essential need for further research into these disorders which can be seen as central importance to the future of human flourishment and quality of life. Disruptive research attempts should not be the excuse to lower its priority in health. This article confirms that genetics provide important additional information to the human predicament. However, such science progressively moulds our clinical understanding, rather substituting it.

We would suggest that there needs to be a greater appreciation of the validity and importance of neuropsychiatric disorder and its importance to helping our patients. Take for example Tuberose Sclerosis which is a common genetic disorder appreciated by all paediatricians. On top of the physical health problems, Tuberose Sclerosis Neuropsychiatric Disorder occurs in 90%, most of which are not identified by routine clinical consultations. Petrus DeVries and colleagues (www.tscinternational.org/) have identified, using the ‘TAND checklist’ (TS associated Neuropsychiatric Disorder), that these neuropsychiatric disorders are only assessed or treated in 20%. The ‘TAND checklist’ developmental assessment include assessment of key developmental milestones, and common, key psychological symptoms e.g. anxiety, depressed moods, mood swings, aggressive outbursts, tantrums, self-injury, language problems, repetitive behaviours, rigidity/inflexibility, over/hyperactivity, concentration problems, restlessness, impulsivity, problems of eating or sleeping. The common neuropsychiatric disorders found are: ASD (25-50%), ADHD (30-50%), Anxiety Disorder (30-60%), Depressive Disorder, Obsessive Disorder and Psychotic Disorder.

This routine assessment of young people with Tuberose Sclerosis assesses for intellectual level and learning problems in reading, writing, spelling and mathematics. It identifies neuropsychological problems in memory, attention, dual tasking, visuo-spatial skills, executive skills in planning, organising, flexible thinking, and orientation for time, place and person. These dimensions should be considered in the context of the level of family stress and relationships. They argue that the ‘TAND checklist’ should be part of routine management for every case.

Such a multidimensional neuropsychiatric approach is a model that is applicable for any brain-based or genetic behavioural phenotype. Accordingly, both paediatrics and child mental health clinicians need to continue to collaborate, follow the growth of skills and knowledge in neuropsychiatric disorder, and work to make clinically valid multidimensional approaches become mainstream paediatrics.

Additional reading on behavioural phenotypes from the CHW School-Link Newsletter and the Journal for the mental health of children and adolescents with intellectual and developmental disabilities.

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