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INTELLECTUAL DISABILITIES AND THEIR COMORBIDITIES

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Intellectual disability refers to early-onset and long-term generalised impairments in intellectual functioning. These impairments are of a global nature (American Association on Mental Retardation 2002). They may comprise slowness in development with a significantly lower than average final level of intellectual functioning (retardation) or distortions of the developmental process – so-called 'qualitative impairments'. Often they comprise both together. These intellectual disabilities are associated with functional impairments in social, adaptive and other important life skills (Hatton 1998). They render the individual reliant on others, to a degree, in order to experience a reasonable quality of life free from avoidable secondary restrictions and exploitation. Standard diagnostic criteria of the *International Classification of Diseases* (ICD) of the World Health Organization (WHO 1992) and the *Diagnostic and Statistical Manual* (DSM) of the American Psychiatric Association (American Psychiatric Association 1994) stipulate the need for intellectual functioning as measured by standardised psychometric tests to be at least two standard deviations below the mean. Thus, for the usual model of a normal Gaussian distribution of intelligence within the general population, with a mean of 100 intellectual quotient (IQ) points and a standard deviation of 15 IQ points, individuals must score no greater than 70 in order to qualify for a label of ICD or DSM 'Mental Retardation', synonymous with the term 'intellectual disability' as used in this book. Hence, at any time, 2–3% of the population will have an IQ consistent with having intellectual disability. Moderate-to-profound intellectual disability (IQ less than 50) occurs in 3–4 per 1000 individuals (Abramowicz & Richardson 1975). However, only a proportion of those with intellectual disability will experience the functional impairments and associated social disabilities and disadvantages necessary to warrant the *clinical* label and to require professional support.

Terminology

Preference for the label 'intellectual disability' is more than cosmetic. First, it is gaining ever increasing international popularity as a term for this particular client group's difficulties and special needs. Indeed, the two leading European academic publications in this field, the *Journal of Intellectual Disability Research* and the *Journal of Applied Research in Intellectual Disabilities*, have for a time favoured this terminology. Second, the phrase avoids derogatory connotations associated with earlier labels, not only archaic ones such as idiot, imbecile and feeble-minded but more recent abbreviations and corruptions, for example

subnormal, retardate, cretin and spastic. Third, it avoids confusions between English usage of the term 'learning disability' (to mean generalised intellectual disability) and North American usage (to mean specific learning difficulties or specific developmental delays such as dyslexia, dyscalculia and dysgraphia). Fourth, audits and surveys of client group preferences confirm almost unanimous dislike for terms such as mental retardation, mental impairment and mental handicap. Service users argue that the term 'mental' leads too readily to confusion with serious psychiatric disorders such as schizophrenia and manic-depressive psychosis. Most individuals with intellectual disability do not suffer with such disorders although rates are certainly higher than in the general population for a number of biological, psychological and social reasons (Rutter et al. 1970, Emerson 2003, Ford et al. 2003). Furthermore, use of the term 'disability' shifts focus away from the underlying impairment which may be structural (such as brain damage through hypoxia, physical trauma or neurodevelopmental defect) or psychological (such as problem solving, executive function or information-processing limitations) but which is usually (but not always) immutable. It is the functional consequences of impairments which can be worked on in order to minimise disability and participation restriction and maximise potential.

Intellectual disability, in health terminology, can be mild (equivalent to a rough IQ estimate of 50–70), moderate (rough IQ estimate of 35–50), severe (rough IQ estimate of 20–35) or profound (rough IQ estimate of less than 20 IQ points). There is no particular preference for certain socio-economic, cultural or racial backgrounds.

Unhelpfully but importantly, educational terminology, at least in the United Kingdom, is based on a different classification termed 'learning difficulties'. In this scenario, moderate learning difficulties denotes a rough IQ estimate of 50–70, while severe learning difficulties denotes a rough IQ estimate of less than 50. The potential confusions are obvious, for example when a clinician describes mild intellectual disability only for this to be mistaken for low average intellectual functioning by educationalists. See Turk (1996), Turk et al. (2007) and Bernard et al. (2009) for further discussions of terminology and its implications.

Features associated with intellectual disability

Having intellectual disability is associated with a range of increased complications and needs. These include physical, psychiatric, social, linguistic, financial, economic, political and not least neurodevelopmental aspects (Turk 1997).

Physical comorbidities are more common in people with intellectual disability than in the general population. They may comprise characteristic appearances such as the facies in Down syndrome (Marder & Dennis 2001), the port wine facial stain indicative of intracranial cavernous haemangiomata in Sturge–Weber syndrome (Comi 2003), the elfin-like facial appearance in Williams syndrome (Tarjan et al. 2003), the jerky ataxic gait and open mouth in Angelman syndrome (Clayton-Smith & Laan 2003) and the longish, largish head with large chin, high-arched palate and simple protruding ears with postpubertal macro-orchidism, widespread ligamentous laxity and joint hypermobility frequently witnessed in fragile X syndrome (Hagerman 1996).

Sensory problems are also more frequent amongst people with intellectual disability than those of average intelligence, in particular visual and auditory difficulties (Turk &

Patton 2000). They are even more common and pronounced in some specific genetic syndromes which cause intellectual disability. Examples include the raised rate of conductive and sensorineural hearing loss, refractory visual errors and squint in Down syndrome, and the hyperacusis (auditory sensitivity) characteristic of Williams syndrome (van Borsel et al. 1997, Metcalfe 1999). Associated motor function impairments are common in conditions where intellectual disability co-occurs with cerebellar, brainstem and spinal cord anomalies. Examples include hydrocephalus, spina bifida, Joubert syndrome (congenital cerebellar vermis hypoplasia) (Merritt 2003) and Arnold–Chiari malformation (persistence of embryological cerebellar tonsils herniating through the foramen magnum) (Arnett 2003).

Social and linguistic comorbidities are common, as witnessed by the relationship between intellectual disability and autistic spectrum disorder. The rates of autism spectrum disorders in children with intellectual disability are substantially raised (Nordin & Gillberg 1996). Up to 50% of individuals with moderate to profound intellectual disability have an autistic spectrum disorder (Wing & Gould 1979) and the degree of intellectual disability is related to the likelihood of having an autistic spectrum disorder and severity of autistic features. Conversely, 70% of children on the autistic spectrum have a non-verbal IQ below 70 and 50% of children on the autistic spectrum have a non-verbal IQ below 50 while only about 5% of children on the autistic spectrum have an IQ above 100 (high functioning autism).

The general rate of *psychiatric disorder* in people with intellectual disability is raised, both in childhood (Rutter et al. 1970, Emerson 2003) and in adults. Epilepsy has a substantially greater prevalence in populations with intellectual disability (Jansen et al. 2004). Furthermore, the combination of epilepsy and intellectual disability produces substantially higher rates of psychopathology (Steffenburg et al. 1996, Espie et al. 2003). Cerebral palsy, too, is more common in people with intellectual disability (Arvio & Sillanpaa 2003). Again, having intellectual disability and cerebral palsy combined produces further elevated rates of psychiatric disorder as does the combination of having autism and epilepsy (Turk et al. 2009).

The key to a comprehensive understanding of comorbidities in the field of intellectual disability is an appreciation that intelligence defines only one of many dimensions of mental life and mental development. These dimensions can be viewed as modular – that is to say that although impairments in one frequently coincide with impairments in others, this is not always the case. Extreme examples include the catastrophic social and language impairments with associated personality difficulties, obsessions and rigidities coexisting with at least average intellectual ability in individuals with high-functioning autism and Asperger syndrome (Koning & Magill-Evans 2001, Soderstrom et al. 2002), and the shyness, social anxiety, attentional deficits, numeracy difficulties, visuospatial problems and executive function deficits witnessed in fragile X premutation carriers, even those with good general intellectual abilities (Aziz et al. 2003).

Thus intellectual disability is associated with multiple comorbidities spanning multiple domains of both physical and mental life and functioning. These comorbidities all have multiple possible causes and multiple inter-relationships and interactions with other developmental dimensions.

Reasons for psychiatric and psychological comorbidities

There is a range of reasons why psychological difficulties may be associated with intellectual disability. First, the co-occurring emotional or behavioural state, or 'challenging behaviour', may be consistent with the individual's general developmental level of abilities. For example, it would be unreasonable to expect a 10 year old with an IQ of approximately 50 to have social, linguistic and attentional skills that differ significantly from those of an average 5 year old. The fact that these attributes exist in an individual of substantially greater stature and strength may well in itself produce social if not clinical issues, and all the more so as the individual enters adolescence and puberty. However, understanding of the developmental 'normality' of such behavioural traits can help considerably in the creation of rational and realistic intervention and support strategies.

Secondly, the individual's presenting psychiatric, psychological and behavioural challenges may reflect emotional reactions to experiences common to us all, such as bereavement (Hollins & Esterhuyzen 1997), other losses, anticipatory anxieties, reactions to unexpected or overwhelming change in surroundings or routine, abuse and neglect (Turk & Brown 1993), being misunderstood by others, and other life events and daily hassles (Ghaziuddin 1988). In such situations care must be taken not to fall into the trap of diagnostic overshadowing, whereby all behavioural and other challenges are attributed to the individual having intellectual disability, with the therapeutic fatalism and nihilism inherent in such a perspective.

Thirdly, it is important to remember that an individual with generalised intellectual disability is still prone (indeed, more so than the general population) to specific developmental delays. These may be attentional, social, linguistic, obsessive-compulsive, sensory and motor. They are often multiple. At their most extreme, they can qualify the individual for further diagnoses such as attention-deficit–hyperactivity disorder (ADHD) (Ishii et al. 2003), autism spectrum disorder (Howlin 2000) or dyspraxia. Diagnosis of such associated specific developmental delays in the presence of generalised intellectual disability is certainly more complicated than is usually the case. However, it is all the more important given the potential for misunderstandings and misattributions regarding causality (for example, mistaking a neurodevelopmental delay for inept parenting, abuse or neglect, or personal malice), and the fact that often the difficulties are amenable to the same battery of evidence-based medical, psychological, educational and social interventions as apply to those without generalised intellectual disability.

Aetiologically, the common neuropsychiatric and neurodevelopmental disorders frequently witnessed in association with intellectual disability (i.e. ADHD and autism spectrum disorders) seem to be part of a common genetic predisposition whereby whatever the genetic basis of the intellectual disability, there are substantially increased rates of ADHD and autistic spectrum disorders, with the curious exceptions of Down syndrome and Prader–Willi syndrome, in particular the *unipaternal disomy* form (Åkefeldt & Gillberg 1999, Veltman et al. 2004). However, it is important to note that the rates of ADHD and autism spectrum disorder in individuals with trisomy 21 Down syndrome and Prader–Willi syndrome are substantially greater than in the general population. Nevertheless, they are lower than expected given the levels of intellectual

functioning of Down syndrome and Prader–Willi syndrome individuals (Green et al. 1989, Rasmussen et al. 2001).

Finally, such neurobehavioural disabilities and disorders may be specific to particular conditions, when they form part of the so-called *behavioural phenotype* (O'Brien 2002). A well-recognised example is the compulsive extreme self-injury in individuals with Lesch–Nyhan syndrome, an X-linked genetic disorder resulting in absence of the enzyme 5-hypoxanthine guanine phosphoribosyl transferase (5HGPRT) which is critical in the metabolism of urate (Robey et al. 2003). Another well-researched association is the voracious overeating (hyperphagia) and obesity in association with unpredictable tantrumming and skin picking found in individuals with Prader–Willi syndrome, due to anomalies on the long arm of chromosome 15 (Descheemaeker et al. 2002). These behaviours replace early floppiness, feeble feeding and failure to thrive, emphasising the importance of developmental trajectories in understanding the nature of neurodevelopmental comorbidities and behavioural phenotypes and their relationships to intellectual disability. A third well-documented illustration is the relentless cognitive deterioration with development of autistic tendencies, spinal curvature (scoliosis), respiratory compromise, overbreathing and midline 'hand wringing' stereotypies following an initial phase of apparently normal development in individuals with Rett syndrome (Colvin et al. 2003). This is associated with a micro-deletion towards the tip of the X chromosome's long arm, involving the MecP2 gene (Christodoulou & Weaving 2003). The *hemizygous* state (XY) is incompatible with life, such conceptions usually resulting in early miscarriage. The *heterozygous* state (XX), with one of the two X chromosomes bearing the Rett syndrome genetic anomaly, is usually compatible with life yet results in this devastating neurodevelopmental disability.

Down syndrome: an illustration of a specific aetiological cause of intellectual disability associated with a characteristic profile of comorbidities

Down syndrome is the most common cause of intellectual disability, occurring in approximately one in 650 livebirths (Selikowitz 1997). The vast majority of individuals have the trisomy 21 non-inherited variant, the main risk factor being increased maternal age. A small minority have a translocation variant which is inherited, emphasising the importance of chromosomal analysis of all newborn infants with Down syndrome, even when the clinical features and diagnosis are obvious, in order that familial genetic counselling can be undertaken where appropriate (Jyothy et al. 2002). The average level of intellectual functioning of people with Down syndrome is in the moderate-to-severe intellectual disability range (Carr 1988). However, the distribution remains statistically normal with 'outliers' at either end. The normal multifactorial (polygenic plus multiple environmental influences) distribution of intelligence has been 'shifted down' by the influence of a third chromosome 21. Thus, even in the population of people with Down syndrome, there are those who are 'brighter', functioning within the low average intelligence range, yet equally those with particularly special needs often compromising profound and multiple disabilities that lie cognitively at the other end of the intelligence distribution.

A further complication in Down syndrome is the occasional presence of mosaicism. In this situation, a proportion of the cells in the body and brain of an individual with Down

syndrome carry the normal complement of 46 chromosomes. In such instances the degree of physical and psychological affectedness is determined by the proportion of cells carrying the trisomy 21 anomaly. Greater proportions of such cells are associated with more severe levels of intellectual disability and more pronounced physical features.

There is little evidence for a particularly uneven cognitive profile in those with Down syndrome. However, adaptive behaviours and social skills, at least early on in development, may be relatively precocious, to the extent of belying the severity of intellectual disability (Dykens et al. 2002). Langdon Down's early anecdotal descriptions of a characteristic personality profile of friendliness, sociability, affection and love of music have to an extent been confirmed by more recent scientific studies, suggesting that there is indeed a characteristic personality and temperamental profile in people with Down syndrome (Gibbs & Thorpe 1983) even though the applicability of well-established personality and temperament scales to populations with intellectual disability may be in doubt (Gibbs et al. 1987).

The relatively low rates of autistic spectrum disorders and attention-deficit disorders in children with Down syndrome have already been alluded to. However, this can mean that when autistic disorders do coexist with Down syndrome, as they may in 10% of instances (as opposed to the much higher rate expected in a group with an average IQ in the moderate-to-profound intellectual disability range), then this important association is missed clinically (Kent et al. 1999).

A further concerning neurodevelopmental twist has been the confirmation of raised rates of clinical depression in adolescents and young adults with Down syndrome, even when level of intellectual functioning, chronological age, social environment, life events and daily hassles have been controlled for (Collacott et al. 1998). This, along with the now well-recognised association between Down syndrome and early-onset Alzheimer presenile dementia (Dodd et al. 2005), means that apparent cognitive decline, social and behavioural withdrawal, and diminishing interest in previously enjoyed activities and pastimes require particularly careful evaluation and appropriate supports.

Frequent clinical common final pathways

The example of Down syndrome illustrates the truism that any one cause of intellectual disability may be associated with a large number of phenomenological end-states – and any number of these may coexist in one individual. The opposite is also true, i.e. that any one phenomenological end-state may be associated with a large number of causes. Illustrative examples of this include ADHD, autistic spectrum disorders and self-injurious tendencies.

Traditionally, there have been considered to be two major determinants of the likelihood and severity of psychological and psychiatric disorders in individuals with intellectual disability, namely degree of intellectual impairment and the quality of the individual's social environment and upbringing (Rutter et al. 1970). These two variables are certainly critical in determining likelihood and severity of emotional and behavioural disturbance. However, to them we must now add aetiology of intellectual disability as a further cause, not just genetic but infective (e.g. congenital rubella (Carvill & Marston 2002)), toxic (e.g. fetal alcohol syndrome (Aronson et al. 1997, Lee et al. 2004)), iatrogenic (e.g. congenital

anticonvulsant exposure syndromes (Dean et al. 2002)) and possibly psychosocial as in the predicament of Romanian orphans starved of emotional and social stimulation at critical times in development (Chugani et al. 2001). Furthermore, there is a need to consider interactions, gene–gene (epistasis), gene–environment and even environment–environment, where one variable may ameliorate potentially adverse other variables (resilience factors) or may aggravate them (complicating or exacerbating factors).

Self-injury

There is no doubt that the likelihood and severity of self-injury in people with intellectual disability is largely determined by psychological and social issues and contingencies (Oliver 1995). However, it is equally true that the likelihood and severity of self-injurious tendencies increase as level of intellectual disability becomes more severe (McClintock et al. 2003). Additionally, self-injury may be the presenting feature in a number of specific genetically determined intellectual disability syndromes. In these instances, the nature or content of the self-injury is remarkably aetiology specific. Lesch–Nyhan syndrome is consistently associated with extreme compulsive and distressing gnawing and biting of knuckles to the extent of skin and underlying tendon erosion (Hall et al. 2001). Restraint is often welcomed by sufferers despite many practitioners' ethical aversion to its use. Examples of ingenious self-restraint and self-control strategies developed by sufferers to modulate their self-destructive compulsions have been reported (Oliver et al. 2003). In Cornelia de Lange syndrome, hyperactive tendencies and moderate-to-severe intellectual disability is associated with lip biting (Berney et al. 1999). In fragile X syndrome the self-injury usually takes the form of biting at the base of the thumb over the 'anatomical snuff-box' in response to anxiety or excitement (Symons et al. 2003). Prader–Willi syndrome predisposes to self-destructive overeating which commonly exacerbates and complicates the raised incidence of diabetes mellitus, as well as compulsive picking at already delicate and fragile skin (Descheemaeker et al. 2002). Smith–Magenis syndrome (Smith et al. 1998), attributable to a microdeletion on the short arm of chromosome 17, leads not only to intellectual disability but also to catastrophic sleep disturbance, gross inattentiveness and overactivity, autistic tendencies and extreme self-injury, usually in the forms of pulling out finger and toenails (onychotillomania) and inserting objects compulsively and repetitively into bodily orifices (polyembolokoilomania).

Reasons for this high specificity of type of self-injury in relation to genetic aetiology remain obscure and poorly understood. However, it seems clear that while likelihood, situationality and severity of self-injury are influenced substantially by psychosocial and in particular behavioural factors, the nature (and probably likelihood and severity) of self-injury is influenced considerably by neurodevelopmental aetiology.

The need for a conceptual shift

Historically there has been a trend for all people with intellectual disability to be considered as one homogeneous grouping, as suggested by early publications exploring the emotional and behavioural disturbances experienced by individuals with intellectual disability (e.g. Phillips & Williams 1975, Groden et al. 1982, Koller et al. 1983). We are now in a position

to commence a move from all-or-nothing, discrete, categorical, phenomenologically based classifications, such as 'mental retardation', towards multidimensional, aetiologically driven classifications of developmental disabilities which allow pinpointing of each individual in multidimensional space in terms of not just intellectual but also social, linguistic, attentional, sensory and motor functioning. To an extent, this movement has already commenced in terms of the multiaxial classificatory model's acknowledgement of the importance of not just psychiatric diagnosis but also coexisting specific developmental delays, level of general intellectual functioning, associated medical conditions, important psychosocial circumstances and experiences, and degree of functional impairment. However, a conceptual step further is required in order to acknowledge that aetiology of the individual's intellectual disability is important in determining not only severity of intellectual impairment but also profile of cognitive strengths and needs, associated developmental abilities and disabilities, developmental trajectories, and thus psychosocial associations. There is evidence that 'genes drive behaviour' (Scarr & McCartney 1983), that there are substantial genetic influences on temperament, personality and other psychological traits (Reif & Lesch 2003), and that these influences increase rather than decrease with age and experience (Plomin 1988).

Example of multidimensional aetiologically driven developmental and psychological profiles: fragile X syndrome

Fragile X syndrome is the most common identifiable cause of inherited intellectual disability, occurring in approximately 1 in 4000 livebirths (Hagerman & Cronister 1996). It has equal prevalence rates in all races and cultures worldwide. It is caused by an abnormal DNA expansion just above the tip of the X chromosome's long arm. This results in impaired production of the fragile X mental retardation 1 protein (FMRP) which is known to contain chemical sequences consistent with the roles of nucleus–cytoplasm transfer of messenger RNA and messenger RNA–ribosomal binding. It thus has important 'housekeeping' roles in terms of facilitating functioning of other protein-producing genes, presumably including at least some critical to neurodevelopment. There are a number of associated physical features (see Hagerman 1996, Turk & Patton 2000) but none of these is pathognomonic of the condition, all occur in other genetic disorders and indeed are seen in many members of the general population. However, a substantial number of research publications confirm the existence of a characteristic cognitive, psychological and behavioural phenotype with important developmental trajectories and clinical implications (Cornish et al. 2004).

Intellectual functioning is usually in the mild-to-moderate intellectual disability range. There is an uneven cognitive profile whereby relative linguistic strengths belie numeracy and visuospatial difficulties, even in those with average-range general intellectual abilities. Discrepancy between cognitive skills of those with fragile X syndrome and non-intellectually disabled peers increases as individuals approach adolescence (Fisch et al. 1996). This is attributable to specific difficulties with sequential (as opposed to simultaneous) information processing. Executive function difficulties have been noted in female carriers and male premutation carriers (Loesch et al. 2003). These manifest as concentration and attentional problems, difficulties in shifting from one topic to another, impaired problem-solving abilities, poor organisational skills and trouble with planning abilities.

Speech and language (Cornish et al. 2004, Taylor 2004) often have a humorous quality with up and down (litanic) swings of pitch, perseverations and repetitiveness, delayed echolalia and cluttering, a mixture of rapid and dysrhythmic speech components.

As many as 28% of young boys with fragile X syndrome have ICD-10 childhood autism. A larger proportion has a characteristic profile of multiple social and language autistic impairments in the presence of a friendly and sociable, albeit shy and socially avoidant personality (Turk & Graham 1997). There is an aversion to eye contact as exhibited in exaggerated avoidance postures during greeting (Garrett et al. 2004), self-injury in the form of hand biting in response to anxiety and excitement (Symons et al. 2003), delays in the development of imitative and symbolic play (Rogers et al. 2003) and a tendency toward stereotyped and repetitive behaviours, including hand flapping and insistence on routine (Turk & Graham 1997). These usually exist in the presence of good understanding of facial expression and theory of mind development consistent with general level of intellectual ability (Turk & Cornish 1998). Recent research findings suggest that rates of diagnosable autistic spectrum disorder increase substantially with age, although the exact reasons for this finding remain unresolved (Turk et al. 2003).

Attentional deficits are common, as are poor concentration, restlessness, fidgetiness, impulsiveness, distractibility and overactivity. Gross motor activity is not necessarily greater than expected for age and developmental abilities, but the other described features usually persist and often require medical and psychological intervention (Turk 1998).

Pilot work on young women with fragile X full mutations and premutations suggests that they too can show a wide range of affectedness, with autism spectrum disorders and ADHD being common even in the presence of reasonable intellectual functioning. Similarly, boys and men with fragile X premutations can experience important developmental difficulties socially, linguistically and attentionally (Aziz et al. 2003, Cornish et al. 2004). Also, the rate of diagnosable DSM-IV autistic disorder has been found to double in young adult men compared with preadolescent and early adolescent boys (Turk et al. 2003).

Conspicuousness by absence

It is of equal importance to note that specific aetiologies of intellectual disability need not predispose to developmental neuropsychiatric disturbances For example, Down syndrome is associated with surprisingly low rates of autistic spectrum and attention-deficit disorders given the general distribution of intelligence amongst individuals with the condition (Dykens et al. 2002). Individuals with Prader–Willi syndrome have similar if not lower rates of autistic spectrum and attention-deficit disorders, although they do have other psychological difficulties (Descheemaeker et al. 2002). FraX-E, a rare form of fragile X syndrome with its own discrete DNA expansion abnormality towards the X chromosome's tip, was initially thought to have a behavioural phenotype manifesting as a mild version of that seen in FraX-A, the 'common' form of fragile X syndrome (Barnicoat et al. 1997, Freeman & Turk, 2007). However, more recent studies suggest that individuals with FraX-E can have extremely variable levels of intellectual functioning ranging from average to severe/profound intellectual disability, and that the presence of autistic spectrum disorder can be equally variable, calling into question whether this particular condition has a behavioural phenotype at all (May et al. 2003).

Conclusion

In conclusion, it is evident that the presence of comorbid physical, psychiatric, psychological and behavioural disorders in individuals with intellectual disability is common, and more frequently witnessed than in those of more average intellectual ability. The occurrence of neurodevelopmental disorders (such as autism and ADHD) as comorbidities may even be the rule rather than the exception, particularly for those with moderate-to-profound intellectual disability. These comorbidities cover the entire biopsychosocial spectrum and can be attributed to the same causative agents as the intellectual impairments that they accompany. One practical consequence of this is that the educational needs of an individual with, say, severe intellectual disability and autism can only be met in a school environment expert in meeting the needs of students with both sets of neurodevelopmental challenges, not one or the other. In short, both intellectual disabilities and the comorbidities discussed in this chapter are common final clinical pathways with multiple possible causes and multiple possible combinations and permutations. These multiple causes and their combinations in any one individual contribute significantly to the nature of the developmental difficulties experienced and hence have substantial implications for intervention, as witnessed by publications focusing on the educational needs of students with particular genetic conditions (e.g. Lorenz 1998, Waters 1999, Dew-Hughes 2003). These multiple interactions of the biological, psychological and social are common and important diagnostically, therapeutically and amelioratively.

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