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Co-Occurrence of Developmental Disorders: Children Who Share Symptoms of Autism, Dyslexia and Attention Deficit Hyperactivity Disorder

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<http://dx.doi.org/10.5772/54159>

1. Introduction

Children with autism spectrum disorders (ASD) have a higher risk of suffering from several other conditions. In this chapter I review the extent to which autistic individuals can also experience a range of other difficulties, but my focus will be on the common neurodevelopmental disorders. The most common of these include dyslexia, attention deficit hyperactivity disorder (ADHD), dyspraxia, specific language impairment, and dyscalculia. There is considerable symptom overlap in particular between ADHD and dyslexia, and like autism both are described as developmental disorders by psychiatric classification systems (American Psychiatric Association, 2000; World Health Organization., 1992). Overlapping conditions are termed 'comorbidity' by medical practitioners. Co-morbidity may reflect the greater difficulties experienced by children with a combination of deficits. Sometimes it is apparent that many children with a developmental disorder could be classified in several ways. Here I will firstly examine the research evidence that examines how often symptoms of dyslexia and ADHD occur in the population of autistic children, and second, review the various theories that have tried to explain why such co-occurring difficulties are so common.

'Comorbidity', a term used in medical literature to mean a dual diagnosis, or multiple diagnoses, can reflect an inability to supply a single diagnosis that accounts for all symptoms. Children with ASD have been shown to have higher rates of epilepsy, with 30% of cases having epilepsy comorbid (Danielsson, Gillberg, Billstedt, Gillberg, & Olsson, 2005). Other conditions that are commonly co-morbid with ASD include hearing impairment (Kielinen, Rantala, Timonen, Linna, & Moilanen, 2004) mental health and behavioural problems (Bradley, Summers, Wood, & Bryson, 2004), including anxiety, and depression (Evans, Canavera,

Kleinpeter, Maccubbin, & Taga, 2005). It has also been shown that parents of autistic children are twice as likely themselves to have suffered from psychiatric illness than parents of non-autistic children (Daniels et al., 2008).

Most of these problems are distinct from those examined in this chapter: the common developmental disorders of childhood which are also found to co-occur with autism, particularly ADHD and dyslexia.

Before reviewing the evidence that suggests many children share difficulties symptomatic of these conditions, and the theories of why this may be, I will briefly describe how dyslexia and ADHD manifest themselves.

2. Dyslexia

Dyslexia is conceptualized by both educational bodies and the psychiatric classification systems as a learning difficulty that primarily affects the skills involved in accurate and fluent word reading and spelling. Characteristic features of dyslexia are difficulties in phonological awareness, verbal memory and verbal processing speed. Dyslexia is developmental delay in literacy and generally slow and inaccurate reading and spelling. The definition of dyslexia has changed over time, and such changes have often been based on the research identifying a range of associated difficulties that occur with dyslexia. Estimates of the prevalence of dyslexia have been complicated because dyslexia cut-offs are contested (Coltheart & Jackson, 1998) and dyslexia manifests itself differently in various languages according to levels of phonic regularity (Miles, 2004). Research over the last 40 years has focused on phonological skills. These are the reading and de-coding skills used when breaking down language into its component sounds and reassembling the parts in order to read or to spell a word.

Like autism, dyslexic difficulties are considered to exist in a continuum throughout the general population (Fawcett, 2012). There is much interest in the association of cognitive ability with changing symptom profiles and diagnosis. The definition of dyslexia is in flux, and has been recently redefined by many national bodies, for example in the UK, the British Psychological Society, focusing on literacy learning at the 'word level' without attainment discrepancy:

Dyslexia is evident when accurate and fluent word reading and/or spelling develops very incompletely or with great difficulty (British Psychological Society, 1999)

This definition implies that the problem is severe and persistent despite appropriate learning opportunities. This UK definition differs from the ICD-10 diagnosis of developmental dyslexia or 'Specific Reading Disorder', which requires a discrepancy between actual reading ability and the reading ability predicted by a child's IQ. So an intellectual disability, (generally considered IQ below 70) can co-occur with the British Psychological Society definition of dyslexia. This new definition includes the so called 'garden variety' dyslexic chil-

dren who have difficulties with reading and spelling as well as other generalized intellectual disabilities. The implications of including this group as dyslexic mean that more children with an intellectual disability would also be classified as 'dyslexic'. As ASD includes a large group with intellectual disability the extension is likely to increase the number of children who may be classified as having both conditions. This is important as the clinical and education label may determine the interventions a particular child receives.

In addition to these characteristics, dyslexic children may experience visual and auditory processing difficulties, similar to hyper or hypo sensitivity often associated with ASD. Like the 'islets of ability' seen in many children with ASD, some dyslexic children may also have strengths in particular areas, such as design, logic, and creative skills.

3. ADHD

ADHD is known as 'Hyperkinetic Disorder' in ICD-10; there are three subtypes of ADHD according to the DSM. In the first, a child will primarily have problems with attention which may manifest as an inability to remain 'on task' for long periods, lack of response to instruction or distractibility. In the second sub-type, symptoms of hyperactivity and impulsivity dominate, which is characterized by wriggling, squirming, being unable to sit still, interrupting and finding it difficult to wait. Children may also be climbing in inappropriate situations and always on the move when free to do so. The third sub-type is simply the co-existence of both attention problems and hyperactivity, with each behavior occurring infrequently alone and symptoms starting before seven years of age.

According to ICD-10, eventually, assessment instruments should develop to the point where it is possible to take a quantitative cut-off score to assess ADHD. Like dyslexia and autism, the symptoms are behavioural in nature, and are part of a continuously distributed pattern that extends into the population at large.

The persistence of ADHD symptoms is not so marked as for autism. Around 70 to 50 percent of those individuals diagnosed in childhood do not continue to have symptoms into adulthood (Elia, Ambrosini, & Rapoport, 1999). There is evidence suggesting to some extent symptoms of ADHD are expressed in reaction to home (Mulligan et al. 2011) and other environmental contexts. Individuals with ADHD also tend to develop coping mechanisms to compensate for some or all of their impairments. ADHD is diagnosed more often in boys with the reported ratio varying from 2:1 to 4:1 (Dulcan, 1997; Kessler et al., 2005) though some studies suggest this may be partially due to referral bias where teachers are more likely to refer boys than girls (Sciutto, Nolfi, & Bluhm, 2004). Treatments for ADHD involve a combination of medication, usually methylphenidates which are well established in improving symptoms of inattention, and behavioral intervention in education and at home. The issue of girls being overlooked on identification is a common thread for research in dyslexia, ADHD and autism. Our own results suggest there is some evidence to back up the claim that boys with ASD symptoms are given the diagnosis more frequently than girls with

equivalent ASD symptoms (Russell, Steer, & Golding, 2011). This may be because the disorders tend to be conceptualized as 'male' leading to referral bias.

Because ASD, Dyslexia and ADHD are all behaviorally defined, so 'symptoms' are behaviours. All three conditions are conceived as particular behaviours along a spectrum, where traits have a continuous distribution and extend into the general (non-disordered) population. An arbitrary cut off point determines who is considered to be within the various categories and who is not. The clinician giving a diagnosis will be responsible for judging where this cut off may come, guided by diagnostic criteria and standards within disciplines as well as perceived implications: the benefits versus any possible risks of assigning a diagnosis. This is perhaps best established for autism: Constantino and Todd (2003) measured autistic traits in a large community sample, and found no jump in the threshold of autistic behaviours between 'normal' individuals and those with an autism spectrum diagnosis, rather they found a continuous distribution. These findings concurred with those in a Scandinavian study (Posserud, Lunder-vold, & Gillberg, 2006). One of our own studies has likewise shown that autistic traits do extend into the 'subclinical' population (Figure 1). As with dyslexia and ADHD, there is not a sharp line separating severity in those with a diagnosis from less severe traits in those without (London, 2007). In both dyslexia, ADHD and the autism spectrum, some children have more severe difficulties than others, and the symptoms extend into the population of children (and adults) as a whole. For dyslexia, there are many people who may have mild dyslexic difficulties but perhaps might not qualify as 'dyslexic'. For autism spectrum disorders, many people without an autism diagnosis do have autistic-type behaviours but the severity and frequency of those behavioural symptoms is less severe than in those deemed to qualify for a diagnosis.

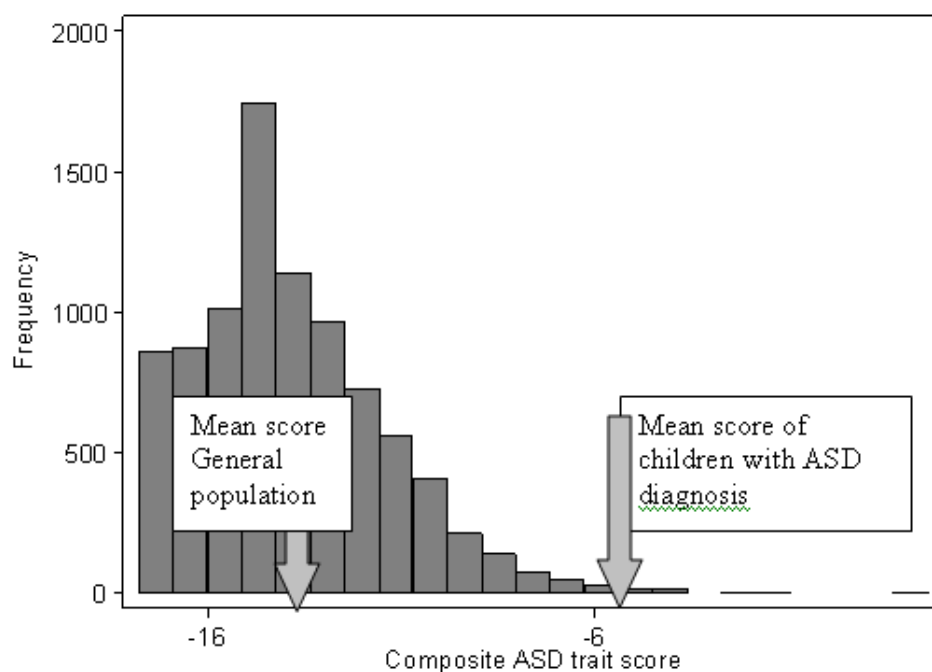


Figure 1. The distribution of an ASD composite trait in the general population from Russell et al.(2012)

The imposition of a cut off between normality and abnormality is therefore 'an arbitrary but convenient way of converting a dimension into a category' as Goodman and Scott (1997, p. 23) point out.

4. Evidence of symptom overlap – ASD and ADHD

Various studies have looked for ADHD or ADHD symptoms in samples of children with autism or ASD. Rates of ADHD have ranged from 28% to 78% of these samples (Ronald, Edelson, Asherson, & Saudino, 2010). Studies that look at ADHD symptoms have reported even higher numbers: for example, Sturm, Fernell, & Gillberg, (2004) looked at a sample of around 100 high functioning children with ASD and found 95% had attention problems, 75% had motor difficulties, 86% had problems with regulation of activity level, and 50% had impulsiveness. About three-quarters had symptoms compatible with mild or severe ADHD, or had deficits in attention, motor control, and perception, indicating a considerable overlap between these disorders and high-functioning ASD in children.

In an large analysis of nine hundred forty-six twins, Reiersen and colleagues (2008) assigned DSM-IV ADHD diagnoses, and measured autistic traits using the Social Responsiveness Scale. The study showed that there are clinically significant elevations of autistic traits in children meeting diagnostic criteria for ADHD. These findings confirm results in earlier studies (Clark, Feehan, Tinline, & Vostanis, 1999). Santosh and Mijoovic (2004) which found children with ADHD had elevated levels of impairment in all three autistic symptom domains, namely social deficits, communication and stereotyped behaviors. Clark *et al* found 65-80% of parents of children with ADHD reported difficulties in social interaction (particularly in empathy and peer relationships) and in communication (particularly in imagination, and maintaining conversation). So the presence of autistic traits in children with ADHD appears common (Ronald et al., 2010).

In an analysis conducted with Lauren Rodgers at the Peninsula Medical School in the UK using data from the Millennium Cohort Study, a cohort of around 19,000 children who were all born between 2000 and 2002, we noted 44 children had a dual diagnosis of both ASD and ADHD (proportion of total population 0.3%) by age seven. The prevalence of children with identified ADHD in the ASD sample was 17%. Conversely, the prevalence of children with ASD in the ADHD sample was higher at 27%. Both figures indicate substantial overlap between these conditions.

Various European research groups have examined co-morbid disorders in adults with diagnosed ASD. An international team lead by Hofvander studied a group of 122 adults with normal IQ from specialist clinics in three European cities: Gothenburg, Paris and Malmö (Hofvander et al., 2009). Here the overwhelming majority had symptoms of ASD. Nonverbal communication problems were also very common, described in 89% of all their subjects. In this study over half the participants, (52%) were diagnosed with co-morbid ADHD. Interestingly, participants diagnosed with pervasive developmental disorder. 'Not Otherwise Specified' (PDD-NOS) diagnosis had significantly more symptoms of inattention and

hyperactivity/impulsivity compared to subjects diagnosed with Asperger's syndrome. However, the prevalence of the categorical diagnosis of ADHD did not differ significantly between the groups, nor were gender differences apparent. Although the study presents clear evidence of many cases where patients display symptoms of both ADHD and ASD, the clinical setting may have led to selection bias as patients with complex needs may be more likely to seek help.

Because behaviours associated with both conditions lie on a spectrum extending into the normal range, some studies have found a range of frequency and severity of symptoms. In Mulligan et al.'s (2009) study, for example, 75 of children with ADHD had severe autism traits, and over half showed sub-clinical autism symptoms. Kadesjö and colleagues (Kadesjö, Gillberg, & Hagberg, 1999), looked at comorbidity of ADHD in Swedish school-age children and found only 1% of children meeting the threshold for ADHD had comorbid Aspergers Syndrome (AS). The estimates of co-morbidity of ADHD symptoms with ASD symptoms vary widely because of differing methods of case ascertainment. An additional problem is that the estimate of the prevalence of ASD itself has increased so much in western countries, making ASD itself a 'moving target' (Figure 2).

Patricia Howlin (2000) reviewed the estimated rates of co-existing psychiatric disorders in subjects with high functioning ASD and found these estimates varied from 9% to 89% - very substantial differences. However it is possible to generalise; thirty years of research have confirmed that attention deficits and hyperactivity are relatively common in children and adults with ASD even if the exact extent of overlap is dependent on methodology and ascertainment (Hofvander et al., 2009, Sturm, Fernell, & Gillberg, 2004).

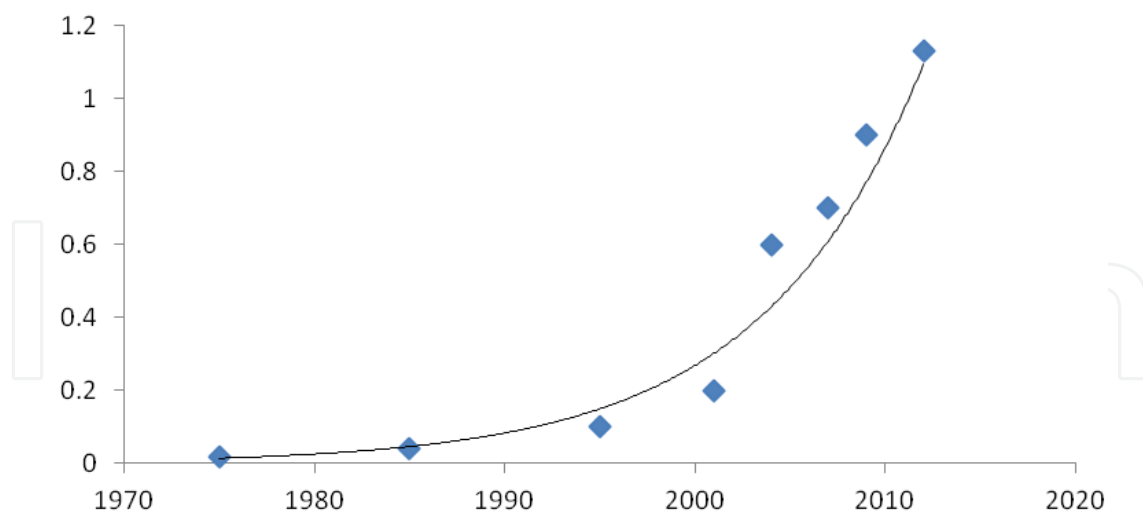


Figure 2. The rising prevalence of autism spectrum disorders over 50 years. (Data from 'Autism Speaks' and CDC, USA)

Recent trends have made categorical diagnosis an integral part of everyday clinical and research practice (Sonuga-Barke & Halperin, 2010). Christopher Gillberg (2010) points out that clinicians have become focused on dichotomous categories of disorder and that clinics have become increasingly specialized and overlook difficulties not within their immediate juris-

diction. Gillberg has argued that co-existence of disorders is the rule rather than the exception in child psychiatry and developmental medicine. He has coined the acronym ESSENCE (referring to Early Symptomatic Syndromes Eliciting

Neurodevelopmental Clinical Examinations). This describes cases where a combination of symptoms including inattention, hyperactivity, social and reading difficulties are observed. Major problems in at least one ESSENCE domain before age 5 years often signal major problems in the same or overlapping domains years later.

To summarize, although ADHD and ASD are separate and recognizable, there is good evidence that these conditions co-occur, constituting an amalgam of problems.

5. Comorbidity between dyslexia and ASD

There is only a small literature on the overlap in symptomology between autism spectrum disorders with those of dyslexia. Officially, as for ADHD, ASD is an exclusionary criterion for diagnosis of dyslexia and vice versa, but ASD also shows overlap with dyslexia in both cognitive and behavioural features (Reiersen & Todd, 2008, Simonoff et al., 2008). A proportion of children share symptoms between dyslexia, ADHD and ASD.

The number of children that do share symptoms of ASD and dyslexia is likely to be small (Wright, Conlon, Wright, & Dyck, 2011). The frequency of reading disorder in combination with disorder of written expression (i.e. dyslexia) was around 14% in a sample of adults with Asperger's Syndrome (AS) so according to this result around one in seven individuals with AS will have co-occurring dyslexia (Hofvander et al., 2009). However the proportion of individuals with dyslexia who have co-occurring AS is likely to be low as Asperger's Syndrome is much a rarer condition than dyslexia.

A common problem for children with dyslexia is misinterpretation of spoken language, which can also manifest itself in comprehension. This produces further overlap with pragmatic language impairment (PLI) which itself is virtually indistinguishable from communication difficulties associated with high functioning autism. Pragmatic language difficulties may involve literal interpretation so 'run on the spot' would have a child looking for a big black spot to run on, for example. Children with PLI will often fail to interpret the core meaning or saliency of events. This causes a penchant for routine and 'sameness' (also seen in autism and Asperger's Syndrome) as PLI children struggle to generalize and take hold of the meaning of novel situations. Obvious and concrete instructions are clearly understood and carried out, whereas simple but non-literal expressions such as jokes, sarcasm and general social chatting are difficult and may be misinterpreted. PLI may therefore impact on the social abilities of the child who has difficulty interpreting jokes. Current thinking is that PLI is not a problem rooted in language skills but one of social communication and information processing. Griffiths (2007) identified difficulties of this type in dyslexic students, showed they were impaired in making inferences from a story and choosing the right punch-line for a joke. This of course can have implications for written language and examinations under stress, as well as for a range of social interactions.

It is not just that ASD is co-morbid with dyslexia and ADHD. Other studies have noted high comorbidity with other developmental disorders. Dyspraxia and dyscalculia and conditions with shared symptoms such as specific language impairment are frequently comorbid with autism. Also dyslexia and ADHD themselves co-occur Willcutt and colleagues (Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005) showed that 40% of a sample of twins with either dyslexia or ADHD was co-morbid for the other disorder. Reading difficulties were measured with both rating scale and an objective task in a study by Cheung et al. (2012) and correlations were observed among ADHD, reading difficulties and IQ. Over half, (53%-72%) of the overlapping familial influences between ADHD and reading difficulties were not shared with IQ. In a school based study Kadesjö and colleagues found 40% of children with ADHD showed reading problems and 29% writing problems (2005).

Overall, the literature suggests, there is good evidence to suggest that some children do suffer from symptoms of both dyslexia and ASD, although this is not so well established, and does not occur so frequently as co-morbidity between ADHD and ASD.

6. Reasons for co-occurrence of ASD with other developmental disorders

Several theories have been put forward to explain the shared symptoms of the various developmental conditions – in other words why specific learning and language and social disorders are not specific. It is likely that all the explanations below play a part in co-occurrence; the causality of co-morbidity is most probably due to a complex web of interacting factors.

7. Genetic explanations

One of the most persuasive explanations is that a genetic predisposition may lead to abnormal neurological development, which in turn may manifest in various different aberrant behaviors and developmental delays. As autism, ADHD and dyslexia and other developmental conditions are all highly heritable, so they all have a large genetic component, the theory seems plausible. The same genetic anomaly may lead to several disorders or psychiatric conditions. In other words one genotype may lead to several (related) phenotypes. This is known as 'pleiotropy'. Researchers have suggested that co-occurrence of autism and ADHD (and other developmental disorders) may reflect such common genetic causes (Reiersen et al, 2008). In this model, the origins of both sets of difficulties are due to common genetic anomalies that predispose children to delayed or atypical neurological development. Certainly, specific genetic anomalies have been associated with a range of psychopathologies in adulthood. However, the genetic picture is complex and exact pathways are not established. It is estimated there are more than a thousand gene variations which could disrupt brain development enough to result in social delays (Sanders et al., 2012).

Such a genetic predisposition is almost certainly complex and multi factorial. So far, over 100 candidate genes have been associated with ASD, most of which encode proteins in-

volved in neural development, but exact mutations within the candidate genes have yet to be identified (Freitag, 2007). Furthermore, different individuals may have mutations in different sets of genes and most of the discovered gene variations are likely to have a low penetrance, thus not all carriers will develop the disorder. There may be interactions among mutations in several genes, e.g. between regulatory genes and coding regions, or between the environment and mutated genes, altering their expression. The effect of a mutation or deletion can depend on processes relating to gene expression and regulation as well as the subsequent effects on the expression of other genes.

The advent of genomics and the emphasis placed on this has led to much research to identify genetic predispositions to ASD. The field of psychiatry as a whole has been 'geneticised' according to some social theorists. This refers to the potential reclassification of psychiatric conditions in the light of findings from molecular biology. For example, a particular sub-category of DSM-IV schizophrenia has been linked to a substitution of a single base in the sequence of DNA of a particular gene localised to a precise place on a particular chromosome, leading to a substitution of one amino-acid for another in an enzyme involved in neurotransmission. Hedgecoe (2001) provides a discussion of the geneticisation of schizophrenia. The debate as to whether the old psychiatric systems of classification should be overhauled in the light of new genomic knowledge which illuminates genetic aetiologies is ongoing (Ericson & Doyle, 2003).

8. Gene-environment interactions

A second theory is that an environmental insult or a stressful event in the life of the fetus or in a young child's life, may trigger a genetic predisposition to be expressed. Thus this constitutes a gene-environmental interaction theory. An example might be the high testosterone levels in the womb that have been observed in some studies. Baron-Cohen's Cambridge group, for example, has carried out work that has suggested high levels of fetal testosterone may be linked to the development of autistic traits (Ingudomnukul, Baron-Cohen, Wheelwright, & Knickmeyer, 2007). According to the gene-environment explanation, the elevated testosterone might lead to the differential expression of genes controlling the neurological development of the child. Another example that has been quite widely publicized concerns Omega 3 fatty acids. These have been implicated by Richardson (2006), who has argued that attention-deficit/hyperactivity disorder, dyslexia, developmental coordination disorder (dyspraxia) and conditions on the autism spectrum may all share common origins triggered by problems with phospholipid (fatty acid) metabolism. However this is just one genetic / environmental explanation for co-occurrence that vies with several others, and the available evidence is subject to interpretation.

In the majority of cases, the gene-environment hypothesis seems highly plausible. It may be that autism and co-occurring developmental conditions may all be caused by a genetic predisposition which is triggered by an early environmental influence (Trottier, Srivastava, & Walker, 1999).

Many environmental factors have been implicated in ASD but the effect of each is poorly established. After the well publicized paper that linked autism to the MMR vaccination, research has repeatedly refuted a link between the MMR jab and ASD (Rutter, 2005). Deykin and MacMahon (1979) found increased risk due to exposure to, and clinical illness from, common viral illnesses in the first 18 months of life. In this study, mumps, chickenpox, fever of unknown origin, and ear infections were all significantly associated with ASD risk. Epidemiological studies have shown there is a higher rate of adverse prenatal and postnatal events in children with ASD than in the general population (Zwaigenbaum et al., 2002). Newschaffer and colleague's (2007) review named associated obstetric conditions that included low birth weight, gestation duration, and caesarean section. It is possible that such an underlying cause partially could explain both autism and the associated conditions (Kolevzon, Gross, & Reichenberg, 2007). There is evidence to suggest adverse prenatal and perinatal events are also associated with ADHD and cognitive development. Some studies have suggested that the risk of autism may be increased with advancing maternal age (Bolton et al., 1997). Paternal age too has frequently (but not always) associated with autism. There are more mutations in the gametes of older men, and this higher rate of mutation in the genetic material from the paternal side may explain the higher levels of neurodevelopmental disabilities in their offspring. An alternative explanation is that fathers who themselves have autistic traits are less likely to have children young. Using anticonvulsants during pregnancy also appears to increase the risk of ASD (Moore et al., 2000). These drugs are used to combat epilepsy which is commonly often comorbid with ASD. Parental occupational exposure to chemicals during the preconception period has also been higher in ASD families than controls in some studies (Felicetti, 1981).

Environmental risk factors have received widespread media coverage within the last few years, perhaps because of the strong degree of public concern (Russell & Kelly, 2011). In most health and disease categories, a secondary function of diagnosis is to group together people who have a common aetiology. However, the specific effects of genetic factors and environmental risk factors that might play a part in abnormal neural development are largely unresolved. Goodman and Scott (1997) stress that current understanding of aetiology for childhood developmental conditions will probably look ridiculously simplistic or misguided in years to come. Despite, or perhaps because of, the uncertainty, there is an underlying concern among people involved with children who are diagnosed with developmental conditions that environmental influences may be partially to blame for rising incidence. Novel prenatal and perinatal medical practices, changing diet, shifting family structures and childhood social activities have all been the subject of lay theories to explain rising prevalence not just of ASD, but developmental disorders in childhood more generally, including ADHD and dyslexia (Russell & Kelly, 2011).

9. The influence of childcare and the child's environment

A third possibility is that environmental factors alone may be enough to trigger not just autistic behaviors, but also other maladaptive behaviors such as inattention. Autistic behaviors

were observed in a study of abandoned Romanian children, conducted by Michael Rutter and colleagues (1999). As well as cases with known genetic causes, in some cases, underlying social factors may predispose autistic symptoms. In this study, Rutter and colleagues noted a very high instance of autism (6%) in the Romanian baby cohort, which they put down to poor early care. These children exhibited typical symptoms of autism at four years old, but unlike cases of autism without maltreatment, symptoms by age 6 were much milder. This case is an illustration of how children who share severe autistic symptoms at young ages may have differing developmental trajectories. In this study, the symptoms of autism may have been triggered primarily by the early neglect, rather than by a genetic predisposition, for if a genetic predisposition was involved it would affect 6% or more of the babies, a very high proportion.

It is not just aetiological environmental factors that seem to lead to increased risks of displaying autistic behaviours. Aetiological causes can be distinguished from proximate determinates which occur at the same time as symptoms, for example, social situations or fluorescent lights may exacerbate the expression of ASD symptoms. There are also those influences in the environment that are sometimes referred to in psychiatry as maintenance factors, including stigmatisation and labelling. Although their influence in perpetuating ASD and other developmental disorders is unclear, an influence in maintaining symptomatic behaviours of autism and co-morbid conditions can not be discounted. Biological causes and behavioural outcomes are mediated by experiential and environmental factors.

10. Cognitive causes and developmental consequences

The competing psychological theories that have been put forward concerning the psychological mechanisms of ASD include weak central coherence theory, deficits in executive function and the extreme male brain theory, all were reviewed by Happé in 1994.

The extreme male brain theory as developed by Baron-Cohen (2002) suggests that autistic individuals can systematize—that is, they can develop internal rules of operation—but are less effective at empathizing and handling events that are unexpected or social. The theory was developed from the earlier ‘theory of mind’ (Baron-Cohen, Leslie, & Frith, 1985). This suggested that autistic people lack the ability to understand other people’s mental states, put themselves in another person’s place or imagine what they might be thinking or experiencing. This lack of mentalising is discussed by Frith and Happé in their discussion of dyslexia, autism and downstream effects of specific impairments (1998). The ‘theory of mind’ lines up with the ‘mirror neuron theory of autism’ (Iacoboni & Dapretto, 2006) which was based on the discovery that the macaque monkey brain contained ‘mirror neurons’ that fired not only when the animal is in action, but also when it observes others carrying out the same actions.

An alternative psychological theory for autism is provided by Frith whose ‘weak central coherence’ theory (Frith, 2003; Happé & Frith, 2006) describes the ability to place information in a context in order to give it meaning. Most people pull together numerous stimuli to form

a coherent picture of the world, allowing them to see the 'bigger picture'. In central coherence theory, the failure to appreciate the whole accounts for the piecemeal way in which people with ASD acquire knowledge. People with ASD may also show relative strengths in some areas, known as 'islets of ability'; and this accounts for savant skills. Related to central coherence is the theory that autistic behaviours are due to interference in executive function (Hill, 2004). Executive functions coordinate the flow of information processing in the brain and are the mechanisms of transferring attention from one thing to another flexibly and easily. They allow people to plan strategically, solve problems and set objectives. Their absence means autistic people show an inability to plan and attain overarching goals. This manifests as easily distractible behaviour and reliance on routines. Such psychological theories of ASD are useful models but have also been subject to criticism. Bailey and Parr (2003) describe such theories of psychological mechanisms as 'narrow cognitive conceptualisations' (p. 27), because they cannot accommodate the presence of sub-clinical autistic traits in the general population.

These theories seem very distinct from some psychological theories that explain dyslexic type and attention and hyperactive difficulties. The exception to this is that, deficits in executive function have been suggested as causal for ADHD, as they affect both cognitive and motivational systems (Willcutt et al., 2005). Frith and Happé (1998) focusing on dyslexia and autism, argue that psychological mechanisms could act as 'gateways' to impairment in other domains. These downstream developmental effects have not yet been fully considered, they suggest. Although they focus on autism and dyslexia, ADHD and other developmental disorders could easily be included in their model. As they point out, both dyslexia and autism have genetic origins, an anatomical basis and extremely variable behavioral manifestations. Their idea is that in addition to the genetic and anatomical origins, an additional developmental pathway may contribute to later difficulties. They argue that specific impairments seen in dyslexia or autism (such as dyslexic phonological or autistic mentalising difficulties) may have a 'gatekeeping' function and subsequently lead to difficulties in other areas. Thus impairments in domain-specific functions may have wide ranging developmental effects.

The idea put simply is that during development, one behavior exacerbates problems in other domains. It is perhaps easier to understand given a few concrete examples. Frith and Happé suggest that the core autistic difficulty of social engagement may lead to missed opportunities for learning, including learning vocabulary. This may effect language acquisition and in turn the development of language based skills evident in dyslexia. An easier pathway to understand might be via gatekeeping function of inattention. If a child is inattentive (a core symptom of ADHD) then the likelihood is they may struggle to focus on learning to read. Hence difficulties symptomatic of dyslexia may be expected. Conversely perhaps reading difficulties are primary, in which case inattention might come from frustration and inability to deal with task demands. This direction of causality seems likely in the sub-group of ADHD children whose problems only appear at school, and who are more likely than other groups to show reading problems according to Taylor (2011). Furthermore, an inattentive child may find it difficult to socialize normally, and may have difficulties following instruction. This may lead to the impairment in social skills symptomatic of autism.

In a similar way, it is possible to theorize that each domain of behavioural impairment in the triad for autism might lead to another. In a review of evidence for single genetic or cognitive causes for autism, Happé, Ronald, and Plomin (2006) note that twin studies suggest combinations of largely non-overlapping genes act on each area of impairment. Their own study found only modest correlations between the three domains of behavioural traits in the triad (namely deficits in social skills and communication and stereotyped behaviour or restricted interests). In the general population, correlations ranged from 0.1- 0.4 for the relationship of each domain to the other. This evidence shows that the three types of autistic traits may be clustered or linked or co-inherited, but with a weak association. These low correlations could be attributed to developmental pathways factors as well as genetic links. Such residual downstream developmental effects are easy to conceptualise. If a young boy is very asocial for example, then his communication skills will not be practised with peers, so he is unlikely to develop as quickly in measures of communication as a more sociable child. The weak correlation between repetitive behaviours is harder to explain. Speculation is possible: repetitive behaviours have been shown to have both self-stimulatory as well as calming functions (Turner, 1999). Repetitive behaviours can therefore be interpreted as responses to unwanted stimuli, e.g. social stimuli with which autistic people have difficulty. Williams (1994) has given a first person account of use of repetitive behaviours to ameliorate the stress of social situations. Conversely, the need for stimulatory repetitive behaviours, concentrating on drawing lines or circles for example, may interfere with social opportunities. Weak associations do not confirm or deny genetic co-inheritance. Developmental pathways where one type of behaviour leads to another may also provide a partial explanation.

In a different but related developmental scenario, Cheslack-Postava and Jordan –Young (2012) suggest that a child's upbringing is highly gendered, and proposed a gendered embodiment model for autism. They cite numerous studies illustrating that the nature of parenting in particular depends on the gender of the child. This they use to describe a gendered theory of development of autism, although the model could also explain the large predominance of boys with other developmental disorders. Cooper (2001) suggests boys are socialized to encourage competition and activity thus a conflict between passivity required at western schools and masculine identity is generated. Some behaviours associated with ADHD when used excessively in school environments, climbing trees for example, are encouraged more often in boys than girls. Cheslack-Postava and Jordan –Young suggest such gendered social processes interact with biology to promote certain 'disordered' behaviours. This they call the 'pervasive developmental environment'.

As well as downstream developmental models, some theorists have suggested one cognitive deficit may underlie several symptomatic behaviours. Although the cognitive/psychological theories of dyslexia and autism seem quite distinct, some research does suggest children with both ADHD and dyslexic difficulties show a distinctive deficit in rapid naming speed, so it may be that processing speed underlies the link (Bental & Tirosh, 2007).

A second example is provided by executive function which is impaired in both autism and ADHD (Willcutt et al, 2005). According to some models, an underlying impairment in executive function prevents children from coordinating information processing in the brain, and

prevents the transfer attention from one thing to another. It is easy to understand how this absence may translate into symptoms of either autism, due to inability to plan with strategic overarching vision, and hence reliance on routines, or as inattention and distractibility symptomatic of ADHD. Executive functions are neuropsychological processes needed to sustain problem-solving toward a goal. Executive functions allow a resolution of conflict when two responses are simultaneously called for by stimuli. In the laboratory, the Stroop task is an example. The conflicting combination of a word like *red* written in green ink creates conflict when the task is to say the color of the ink (green), due to the overlearned reading response that automatically elicits the response based on the meaning of the word (red). Executive function allows for the inhibition of the overlearned response and the execution of a response that is more appropriate given the context. Research has confirmed the involvement of deficits in executive functions that are essential for effective self-regulation in people with ADHD. The mental processes most often listed as being part of the notion of executive function are quite diverse so there is no standardized definition. They include: inhibition, resistance to distraction, self-awareness, working memory, emotional self-control, and even self-motivation. Bramham and colleagues (2009) found that both adults with ASD and ADHD had impaired executive function, although they did have distinctive profiles. Nyden and colleagues found that children with Asperger's Syndrome and dyslexia did not differ in tests of executive function: they could not establish any test of executive function that captured the differences in these disorders (1999).

Russell Barkley (2012) conceptualizes executive control as the methods of self-regulation. He writes entertainingly on how a person might use executive functions to resist the temptation to buy a tempting pastry from a shop:

...avert your eyes from the counter, walk to a different section of the shop away from the tempting goodies, engage yourself in mental conversation about why you need to not buy those products, and even visualize an image of the new slenderer version of yourself you expect to achieve in the near future. All of these are self-directed actions you are using to try and alter the likelihood of giving into temptation and therefore increase your chances of meeting your goal of weight loss this month. This situation calls upon a number of distinct yet interacting mental abilities to successfully negotiate the situation. You have to be aware that a dilemma has arisen when you walked into the shop (self-awareness), you have to restrain your urge to order the pastry to go with the coffee you have ordered (inhibition), you redirected your attention away from the tempting objects (executive attention or attentional management), you spoke to yourself using your mind's voice (verbal self-instruction or working memory), and you visualized an image of your goal and what you would look like when you successfully attain it (nonverbal working memory, or visual imagery). You may also have found yourself thinking about various other ways you could have coped effectively with these

temptations (problem-solving), and may have even used words of encouragement toward yourself to enhance the likelihood that you would follow your plan (self-motivation).

Barkley explains that these and other mental activities are usually included in the understanding of human self-regulation, and it is difficulties in these areas (which are processes in executive function) that may lead to ADHD. Children with ADHD are distractible and self-regulation, the ability to override incoming stimuli, to see the bigger picture and lack the ability to see the consequences of their future actions. Children with ASD have difficulties transferring attention from one thing to another because they also lack overview (and implications of their actions in the future).

Gooch, Snowling and Hulme (2011) note that deficits in time perception (the ability to judge the length of time intervals) have been found in children with both dyslexia and ADHD. These researchers found children with comorbid dyslexia and attention problems performed poorly on measures of executive function as well as on phonological tasks. However, their results were interpreted as the effect of independent underlying cognitive causes. Although deficits in duration discrimination were associated with both dyslexia and attention problems, they concluded the results supported the claim that the two disorders are products of different cognitive defects originating from shared genes with pleiotropic effects.

Developmental models explain comorbidity of developmental disorders by shared cognitive deficits, either as 'gateways' as in Frith and Happés (1998) model, where one difficulty leads to another later in life, or as underlying shared deficits, for example impaired executive function causing both autism and ADHD. The alternative model suggests that cognitive difficulties associated with each disorder are distinct, but multiple cognitive deficits arise from similar genetic/environmental origins. All these theories have some empirical support.

11. Diagnostic substitution and the influence of society and culture

When symptoms of two or more conditions are shared, whatever the psychological mechanisms (whether or not there are shared underlying cognitive deficits, and /or genetic and neurological differences) then the area of functioning that is highlighted as a problem may depend on which tests are administered. In our recent research we followed a six year old child who was assessed by three educational psychologists and one multidisciplinary team, each blind to the findings of the others. One concluded that the child had dyspraxia, two that the child had dyslexic difficulties, and a third that borderline AS was likely. We interpreted these differences in the use of diagnostic labels as dependent on settings that varied during assessments, and assessment methods that exposed different types of behaviour (Russell, Norwich, & Gwernan-Jones, 2012). This work suggests that which diagnosis is assigned depends to some extent on social and cultural factors as well as actual symptoms. If a child has symptoms of several disorders, then one context or test may draw out symptoms associated with one disorder, whereas another setting may expose symptoms of another.

Thus for co-occurring symptoms it is difficult to differentiate between disorders and the likelihood that a co-morbid disorder will be missed is increased. This emphasizes the need for assessment in multiple settings and reassessment over time.

One of the most compelling cross cultural descriptions of how autism is regarded across various cultures was the book *Unstrange Minds*. Written by the anthropologist Roy Grinker (2008), Grinker explains how the category of ASD is contingent on the culture through which it is expressed- the condition is associated with differing levels of stigma in different cultures. In the US, several studies have also shown that clinicians may diagnose ASD when resources are targeted at the diagnosis, whereas previously, under other circumstances, they may have diagnosed another category of childhood disorder. Paul Shattuck has written about the extent to which increases in the administrative prevalence of autism have been associated with corresponding decreases in the use of other diagnostic categories, mental retardation and learning disabilities (2006). This process of 'diagnostic substitution' he argues, may partially explain the rise in prevalence in autism in the US.

Our own work suggests that since the 1980s, the recorded prevalence of both ASD and ADHD in the UK has increased dramatically. We examined data from both the Millennium Cohort Study, (the large cohort of around 19,000 children who have been followed from their birth through to seven years old and beyond), and another cohort, called the British Cohort Study, where children were born thirty years previously. Both cohorts were representative of the UK as a whole, and medical reports of both ASD and ADHD were given when children were age seven for in 2007-9 and ten in 1980. The results from 2007 contrasted with the 1980 sample at age 10. Only 11 children in the 1970 British Cohort Study were reported as having ADHD in their medical exam, giving an estimated prevalence of 0.083%. The autism diagnosis was rarely used with just 3 children assigned the label; 0.023% of children. A number of other child psychiatric diagnoses were available and many of these were diagnosed during the medical exams. Details of these alternative labels are given in Table 1.

1980 Diagnosis (ICD 9 codes)	N of children	Percentage of total examined %
Autism (299.0/1/8/9)	3	0.023
ADHD (314.00/01, 314.9)	11	0.083
Disturbance in emotions (313)	7	0.053
Delays in development: Reading (315.0)	13	0.098
Delays in learning & development (315.2/8/9/5)	81	0.614
Delays in language (315.3)	62 (1 autism co-morbid)	0.462
Impulse control (312.3/9)	1	0.007
Mild mental retardation (317)	34 (1 ADHD co-morbid)	0.258
Other specified delays in development (318)	22 (1 ADHD co-morbid)	0.166

1980 Diagnosis (ICD 9 codes)	N of children	Percentage of total examined %
Unspecified delays in development (319)	25 (1 ADHD co-morbid)	0.379
Total	259	1.961

Table 1. Named conditions using ICD-9 categories for 10 year old children in 1980 (n=13201).

Among the 14,043 children in the 2007 cohort, 209 (1.49%) were reported to have ASD, and 180 (1.28%) were reported having been given an ADHD diagnosis by a clinician (unweighted figures). There was disproportional stratification in the Millennium Cohort, meaning that all analyses were weighted to account for the clustering and over-inclusion of participants from disadvantaged areas. After weighting, 1.7 % of children were reported as having an ASD (95% CI, 1.4-1.99). 1.3% of these were boys, and 0.25% girls, giving boy girl ratio of approx 5:1 for ASD. Surprisingly, the figure for ADHD was lower. After weighting, 1.4% of the population were reported as having ADHD (95% CI, 1.2-1.7). Of these, 2.3% were boys and 0.25% girls, giving a gender ratio of approximately of 1 girl to every 4 boys with ADHD.

One interpretation of the historical shift is that diagnostic substitution has occurred: children with similar symptoms in 1980 may have been more likely to receive generalised labels of 'delays in learning & development' than ASD or ADHD. So changing diagnostic practice, cultural factors and context may do much to explain both co-morbidity and rising prevalence. The steep rise in children assigned these diagnoses cannot be totally explained by the substitution mechanism- twice as many children were given either ASD or ADHD diagnoses in 2009 as the total number diagnosed with any type of developmental disorder in 1980.

Context also has a big part to play in the identification of difficulties, in terms of what is considered to be 'disordered'. Social constructionists have also pointed out that the conceptualization of difficulties associated with both dyslexia and ASD as 'disorders' is itself a product of social and cultural standards, and of course the definition of each disorder has changed over time. This has prompted calls for the term autism spectrum 'conditions' to replace autism spectrum 'disorders' (2009). Our own analysis of the Millennium Cohort has shown a strong association between ADHD and poverty, reflecting findings from US studies which have also found differing levels of ADHD amongst various ethnic groups- Hispanic children were more likely to be identified with ADHD in a study by Akinbami et al. (2011). It is unclear whether this is entirely due to greater awareness and access to health care in some groups, differential reporting about the same level of difficulties between ethnic groups or whether children in different groups have truly varying symptom levels (Boyle et al., 2011). A study by Cuccaro et al. (1996) showed the nature of diagnosis of developmental disorders varied according to the socio-economic status of the child's family; autism was more likely to be identified in children of higher income families, although no biases of SES were found for identification with ADHD. Cooper (2001) points out that the behaviour symptomatic of ADHD becomes problematic where high value is placed on ability to remain sedentary and sustain attention on tasks, in other words, in schools. Hulme and Snowling (2009) describe how differences of this nature must therefore be thought of as *both* biological *and* as a product of the social and environmental world.

12. Conclusion

Two conclusions can be drawn. First, co-morbidities between developmental disorders are common, and second, the causes of these overlapping difficulties are likely to be complex, multifactorial and interacting. Firstly, the high overlap between symptoms of different developmental disorders has been identified in a number of studies and there is an international consensus on this overlap. Studies from Canada, the UK, USA and Scandinavia all show how hard it is provide an unequivocal diagnosis, leading to the quote from Kaplan and her colleagues (2001) *in developmental disorders co-morbidity is the rule, not the exception*. This was informed by the group's work studying a population-based sample of 179 children receiving special support in Calgary: If the children met the dyslexia criteria, there was a 51.6% chance of having another disorder. If the children met the ADHD criteria there was an 80.4% chance of having another disorder. They criticize the term 'comorbidity', as it implies unsubstantiated presumption of independent aetiologies. The authors argue that discrete categories do not exist in real life.

Secondly, in considering the reasons for co-morbidities, a complex bio-psycho-social model is required that leads to symptoms that may result in diagnosis. The nature of the diagnosis itself may depend on social context as well as an individual child's behaviour. A hint of this complexity is achieved in Figure 3, which is a schematic diagram of various potential causal pathways. It is plausible that the same underlying genetic or neurological mechanisms may underlie co-occurrence of dyslexia, ADHD and ASD. The reverse pathways are not at first so obvious. But recent advances in systems biology have shown that the environment of the cell affects gene expression and protein synthesis at molecular levels. Thus environmental influences can alter 'core' biology: for example Mack and Mack (1992) describe how tweaking rats' whiskers changes gene expression in the sensory cortex. In systems theory, genetic influences are conceptualised more like a set of piano keys on which notes may be played or not played, played slowly or quickly, and there is enormous variation in the music produced even with the same basic set of keys. So the cellular environment can affect genetic expression. A simplified model underlying much behaviour genetics research envisages a direct linear relationship between individual genes and behaviours. The reality is likely to be far more complex with gene networks and multiple environmental factors impacting brain development and function, which in turn will influence behaviour (Hamer, 2002). Karmiloff-Smith (2007) emphasizes how learning and experience effects gene expression in humans. Such scholars demonstrate that the social can affect the biological as well as the more intuitive path of genetic origin leading to neurological development leading to aberrant behaviour. Diagnosis itself may influence behaviour too, through differential treatment and interventions. Thus the pervasive developmental environment is composed of many related factors, environmental stresses, and genetic predispositions, and the social contexts all of which may interact to produce developmental outcomes that themselves may contribute to predicting ongoing child development.

Snowling (2012) suggests a new dimensional classification of disorder, where deficits in different components of learning are seen as additive, impacting on the potential for remedia-

tion, rather than classing children into dichotomous 'disorder' categories. Taylor (2011) notes that for many children, it is better to think of changes in cognitive style, learning and motivation rather than symptoms. Both conclude that it is important to examine children for evidence of co-occurring disorders, and not simply continue to examine the areas which we expect to be impaired according to categorization. The practical application of assessing children for a range of difficulties is that children will be best helped not by any all encompassing diagnosis, but by individual analysis of their strengths and weaknesses. Future research may be wise to focus on the individual profiles of children across a broad range of areas, looking at the unique strengths, as well as the weaknesses of the individual children, so that parents and educators may adapt their support accordingly, regardless of the diagnostic label a child receives.

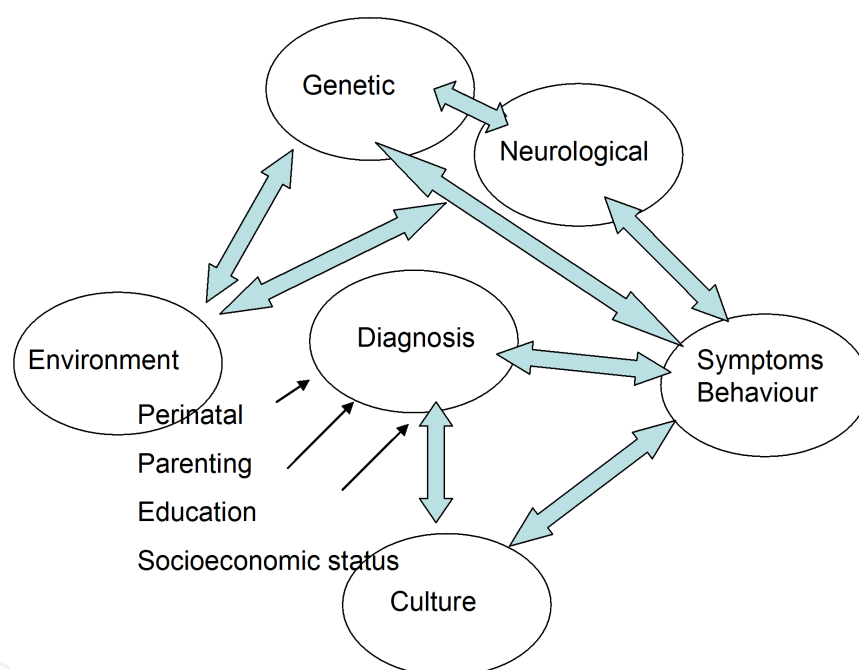


Figure 3. Schematic of interacting causal mechanisms for co-morbidity.

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