

Comorbidity is  
becoming an  
accepted feature of  
development



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Comorbidity is the co-occurrence of two different disorders in an individual at the same time. Sometimes multiple diagnoses have to be made to account for all the symptoms. Understanding the origins of comorbidities help us understand the causes of disorders but the underlying causal mechanisms for any developmental disorder are not thoroughly understood.

In Frith's three level framework the essential elements in developmental disorders are the biological, cognitive and behavioural levels that are all impacted upon by the environment. Separate disorders can be traced to the biological level and may have shared genetic causes, but distinct brain differences can be identified. Disorders interact at the cognitive and behavioural levels, but remain independent. In a child comorbid for ADHD and dyslexia, the ADHD may arise due to striatal-frontal dysfunction and the dyslexia due to magnocellular dysfunction. These separate biological problems could cause cognitive deficits, temporal processing deficit in dyslexia and problems with impulse control in ADHD. Deficits in temporal processing may cause problems with spoken and written language (also a behaviour seen in ADHD occurring along with attention impairment). So there is overlap at the behavioural level even though each disorder arises from separate cognitive and neural dysfunctions. Comorbidity can result in a more severe clinical problem as the attention impairments present in ADHD may exacerbate the problems of spoken and written language seen in dyslexia.

A problem in diagnosing developmental disorders is whether they are diagnosed categorically or dimensionally. Categorically, a single specific deficit should result in a specific disorder and vice versa. However,

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comorbidity is often explained by the interaction of multiple risk and protective factors occurring at the neural, cognitive or environmental level. These factors can alter the development of the neural systems controlling cognitive functions needed for normal development and so produce the symptoms of individual disorders. It was originally thought that dyslexia and reading disorders were primarily due to phonological deficits, however children with speech-sound disorder also had this deficit but didn't show signs of dyslexia. Pennington found children with RD often have processing speed difficulties but children with SSD don't. Thus despite children with SSD having a deficit in phoneme awareness, their intact processing speed acts as a protective factor against them developing a RD. Deficits in processing speed are also found in ADHD, helping to explain its high rate of comorbidity with dyslexia (Shanahan et al, 2006).

Comorbidity is more common than it "should" be based on prevalence of individual disorders in the population. The diagnostic symptoms of ADHD and ASD don't overlap, implying they're independent disorder, however Simonoff et al (2008) and Sinzig et al (2009) found 20-50% of children with ADHD met the criteria for ASD and 30-80% of children with ASD met the criteria for ADHD suggesting there's a categorical overlap, so disorders can't be explained by a single deficit model. If disorders are dimensional all 3 of Frith's levels influence the disorder but the degree of severity may differ based on the severity of impairment at each level and due to the influence of external factors. Developmental disorders are therefore seen to result from multiple deficits, the more impairments someone has the greater the severity of the disorder. Snowling (2008) found that the more cognitive

deficits a child had, the worse their literacy skills were. Frith's suggests the wide variability in disorders is due to strengths and weaknesses in other cognitive areas that can either enable compensation or aggravation of the disorder or it can be accounted for by the differences in severity of impairments at each level.

Gilger and Kaplan (2001) say comorbidity results from a single biological cause such as atypical brain development, causing distinct impairments. Here, a generalised underlying brain abnormality causes all developmental disorders. Where this abnormality affects coordination most it is termed DCD, where it effects reading most it is termed dyslexia. Here, disorders appear to depend on polygenic inheritance. Many different generalist genes are involved in the development and functioning of the brain. Genes that are implicated in developmental disorders are often the same as genes responsible for normal variations in development. For example, KIAA-0319 and ROBO1 are involved in general aspects of brain development such as growth and neural migration but are also implicated in dyslexia, indicating dyslexia is simply the lower end of a continuum of reading skill and not a discrete clinical entity (Plomin and Kovas, 2005). Landerl et al (2010) could only partly support this generalist genes theory and found that learning disorders are the result of an interaction between both general and disorder-specific factors.

At the neural level pleiotropy can occur. A single genetic risk factor can affect more than one neural system resulting in more than one phenotype.

Gilger et al (2007) twin study found concordance rates for ADHD and RD

were only slightly higher in MZ than DZ twins suggesting they are largely  
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genetically independent but there seemed to be a genetically controlled comorbid subtype when they co-occurred. Even though there seems to be limited overlap at the cognitive level, ADHD arising due to deficits in working memory, inhibition and aversion delays (Sonuga-Barke 2002) and RD arising primarily due to phonological deficits, Stevenson et al (2005) found a common gene ADRA2A is involved in the development of both disorders. So disorders are largely independent but genetics can account for comorbidity. Stevenson et al. (1993) and Light et al (1995) found shared genetic factors could account for 75% of the covariance between spelling and hyperactivity as well as reading ability and hyperactivity.

Research has shown disorders tend to co-occur and that they exist along a continuum of symptom severity but it is not clear whether the comorbid disorders are independent from each other or due to a single underlying cause (Landerl et al, 2010). Understanding the cognitive processes underlying behaviours, helps with diagnosis and treatment and the clarification of cognitive phenotypes of developmental disorders is essential for understanding their genetic and neural bases (Hulme and Snowling).

Developmental disorders are currently defined at the behavioural level and Frith (2001) and Morton (2004) suggest this is unsatisfactory. The same behaviour can result from more than one cause and the same cause could result in different behaviours. Behaviour can also change depending on circumstances but the underlying condition may still remain. It's important to assess cognitive ability so the behaviour can be interpreted in terms of the child's developmental level. Although children with a primary diagnosis of autism may also have intellectual disability, those with a primary diagnosis <https://assignbuster.com/comorbidity-is-becoming-an-accepted-feature-of-development/>

of ID may display false comorbidity with autism simply due to their cognitive delay. Assessing at the behavioural level alone would not be satisfactory as both disorders are associated with delays in symbolic play and verbal communication (Pennington). It can also lead to referral bias. If a child refuses to sit still and work they may be referred down an NHS pathway and diagnosed with ADHD, however it may be due to not being able absorb specific information and so an educational psychologist may diagnose them with RD

Behavioural assessments may not distinguish disordered from non disordered individuals. Children can use different methods for reading, so the behavioural level doesn't indicate how the task was done or if there are any underlying problems. Dyslexics can use compensatory mechanisms such as focused attention or mnemonic strategies to counter the problematic behaviour and mask it so it appears implicit and automatic like in non disordered individuals. If they were comorbid for ADHD they may have trouble sustaining focused attention and so behavioural manifestations would be more apparent. However it's important to assess at the cognitive level in case behavioural differences are not so apparent. Compensation strategies can be exposed if the dyslexic child is given multiple simultaneous tasks such as a written maths problem. In behavioural assessments the child may be thought to have dyscalculia when actually their focus on using compensatory reading strategies could result in a working memory overload causing impairment in performing additional tasks. If maths questions were presented numerically the supposed arithmetic problems would disappear.

Severity of impairments can exacerbate behavioural symptoms, so reducing the severity of impairments through remediation should result in behavioural improvements. Teaching phonological skills can help improve reading skills in RD. However if the RD is comorbid with ADHD, impairments may be more severe due to increased working memory deficits and problems with sustaining attention. Remediation of reading skills may not be successful until the problem with attention is addressed. Fabiano et al (2009) found the use of CBT therapies can help improve impairments in attention as can medication such as Ritalin (Stephen et al, 2003). Once the child can sustain attention they may be able to have remedial teaching for additional impairments such as phonological skills training.

Using dimensional instead of categorical statements of disorders helps provide descriptions of strengths and weakness that may help with educational management and remediation. Saying “Katie has dyslexia and ADHD” does not help state the cause of the problem. Saying “Katie has phonological and reading problems as well as difficulties in sustaining attention” helps lead to remedial recommendations. Katie needs help learning to read and may have extra difficulties here due to her problems with attention.

Intra category variability can be as large as inter category differences so pure phenotypes are extremely rare. Disorders don't seem to be independent as even if they share no diagnostic symptoms there's overlap at the cognitive or neural level suggesting assessment and remediation at the behavioural level is not enough. Individual differences in symptom severity differ due to the effects of different genes and neural systems combined with <https://assignbuster.com/comorbidity-is-becoming-an-accepted-feature-of-development/>

cognitive strengths and weaknesses as well as environmental effects. All individuals may have different risk factors, clinical courses, and responses to drug and behavioural treatments so proper assessment and understanding of each of these is important for refinements in assessment and remedial strategies.