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Editorial

Autism is not a condition that I have studied and at times I have been concerned that this *Journal* may have published a disproportionate number of studies on a condition that is relatively rare and primarily determined by genetic factors, and for these reasons, perhaps of less relevance to most clinicians. Having read Pat Howlin's Practitioner Review I realise just how wrong I have been on both counts! Not only are autism, autism spectrum disorders, and Asperger syndrome far more common than I realised (the prevalence estimate of Wing for autism spectrum disorders of just under 1 per 1000 is quoted) but it is also clear that the recent research effort focused on these children has paid dividends in the development of approaches to treatment for the disorders. As Howlin emphasises, these advances have not lead to a cure for autism, as claimed by some proponents of therapies such as holding therapy and facilitated communication, and even those producing some change may require an unacceptable burden of commitment for families, for example the home-based behavioural programmes of Lovaas and colleagues. However, as she clearly indicates, we have learned how to guide the management of challenging behaviour and have methods both to enhance communication and to reduce obsessional behaviour in children with autism. A recurrent theme running through this Practitioner Review is the value of early intervention with these children. This, in turn, gives new impetus to both the research concerned with the basic deficits experienced by children with autism and the studies on the detection of early signs of the disorder by, amongst others, Baron-Cohen and colleagues. An example of the former is the paper in this issue by Leevers and Harris into the ability of the child with autism to conceptualise and image "impossible" entities, for example a man with two heads. This study showed that the imagination of children with autism is not as impaired as once thought. Care should be taken not to interpret poor performance on such tests as reflecting lack of creativity or imagination when a problem with understanding the nature of the task or with certain task demands, such as planning, may be responsible.

In her discussion of home-based interventions for children with autism Howlin was concerned not just with the impact of the behaviour of the child with autism on family members but with the impact of treatment on the family. The paper by Eisenberg et al. takes up the issue of the impact of children with learning disabilities on other family members, specifically siblings in this case. These siblings were not found to be at increased risk of psychological problems and this is not affected by whether the learning disabled child lives away from home or not. These children are comparable to their peers in families that do not have a child with learning disabilities on measures of self-esteem, psychological adjustment, and family environment. Indeed, many reported positive growth experiences. Although there does not appear to be a need for therapeutic services for this population, these siblings did express appropriate concerns about the future and services offering information and support would be helpful.

The impact of disability on the children themselves rather than on the family is addressed by the paper by Goodman. He undertook a longitudinal study of a representative sample of children with hemiplegic cerebral palsy. Around 70% of the children who met "caseness" for child psychiatric disorder still met those criteria 4 years later. Not only do children with cerebral palsy have a substantially increased risk of psychiatric disorder, but these problems are not transitory. In the school years, hyperactivity was particularly predictive of continuing psychiatric problems. Since the psychiatric difficulties of neurologically impaired children seem relatively unlikely to resolve spontaneously, there appears to be a strong case for treating these children's problems vigorously and at an early stage.

Again, as with autism, there is an endorsement of early intervention for children at risk as a result of neurological impairment. The same endorsement has been given for a wide variety of psychosocial problems in preschool children. The paper by Davis and Spurr addresses how services to preschool children and their families might be delivered effectively. In the context of child and family mental health needs far exceeding specialist resources, additional intervention or preventive methods need to be developed. Given that these problems are the daily concern of primary health care and community workers, an important strategy is to enable them to work more effectively by providing appropriate training and supervision. This paper explores these issues and provides preliminary evidence suggesting that community health care workers can intervene appropriately and effectively with families with young children.

The current issue includes a number of papers that address the natural history of disorders in childhood, for example that by Vitaro et al. on aggression and conduct problems. The study by Steinhausen and Winkler Metzke on substance abuse supports the observation that initiation into the use of alcohol and nicotine typically occurs during adolescence or even during preadolescence. This survey suggests that there is a close association between substance use by parents and adolescents. It is possible that exposure to substance use by parents acts as a model for their children and that such a model acts to increase the risk of later substance use by all children in the family. If so this would then be an example of a shared environmental influence.

A major contribution of behaviour genetic research has been the demonstration of the power of another facet of the environment to influence child psychopathology—

namely the nonshared environment: a finding replicated by Eley and colleagues in their study of depression in middle childhood. O'Connor et al. provide the latest set of findings from one of the most extensive studies concerned with the role of both genetic and environmental influences on behavioural development: the Nonshared Environment and Adolescent Development project. Genetically informative research designs, such as this one, are essential if we are to understand not just the types of genetic and environmental influence on the behavioural phenotype but, as with the present longitudinal study, the dynamic changes in symptomatology brought about by the continuities and changes in the pattern of genetic and environmental influence on behaviour. It is salutary to observe the extent to which change is mediated genetically. However, these results should not be interpreted as indicating that genetic factors are pervasive and immutable both in the origins of psychopathology and in its maintenance. Rather the findings that one third of the stability in depressive symptoms were accounted for by the nonshared environment suggests that effective action can be taken. These nonshared environmental influences are either experiences not shared with other brothers and sisters or are aspects of experience, possibly within the family, that do not have similar effects on all family members. This provides scope for intervention to relieve the stressful circumstances being experienced or to foster more adaptive coping by the child. The scope for change is also indicated by the finding that shared environmental influences, which may be more difficult to treat, did not contribute to the stability of depressive symptoms. Although there are indications for the appropriate forms of intervention for depression and antisocial behaviour within their findings, O'Connor and colleagues make the more basic point that high heritability either for behaviour or change in behaviour does not indicate that "these behaviours are immutable or would be refractory to intervention". The behaviour genetic analysis reflects the relative magnitude of influences usually experienced by the general population. Intervention, therapeutic or otherwise, may dramatically alter this pattern of relative genetic and environmental influence.

The remaining papers in this issue concern the nature of the deficit underlying aspects of hyperactivity and its origins. There are a number of possible theoretical accounts for the psychological core of ADHD. One of these has centred on a response inhibition deficit (Barkley, 1997). Central to this theoretical position has been the use of the "stop task" to measure the child's ability to inhibit inappropriate responding. The paper by Oosterlaan and colleagues provides a meta-analysis of the studies on this task and concludes that indeed children with ADHD do show a response inhibition deficit but that this may also be shown by children with conduct disorder. If Barkley's theory holds, it will provide a firm basis on which to plan interventions to compensate ADHD children for this fundamental deficit in inhibiting action.

There has been a growing recognition that the problems experienced by children with ADHD have a strong genetic component. The details of which biological systems might be mediating this genetic effect are still uncertain. Gaitens et al. have conducted a study on the possible involvement of the immunological system. They conclude that within their study there was no relationship between the degree of atopy shown by a child and the severity of ADHD symptomatology (or indeed any of the other scales derived from the Child Behavior Checklist). There is one important proviso that must be considered in relation to this study. As the authors recognise, they were only concerned with the role of degree of atopy in influencing behaviour within a population referred to an allergy clinic. It may still be that the presence of atopy itself is associated with an increased risk of ADHD. Further studies are needed to test this possibility and the possible role such an allergy-prone immunological system may play in mediating the impact of allergens, such as those in food, on hyperactivity.

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References

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