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IN THIS ISSUE

This issue contains two reviews, one on recent advances in genetic findings on attention deficit hyperactivity disorder (ADHD) and one on the cognitive neuropsychology of depression in the elderly. Other sets of papers continue these themes, examining various aspects of ADHD and depression in older people.

Genetic findings on ADHD

In the first review paper, Thapar *et al*. (pp. 1681–1692) review advances in genetic findings on ADHD. Building on family and twin studies that have established the importance of genetic influences on the aetiology and persistence of ADHD, a small number of gene variants (e.g. the 48-bp variable number tandem repeat variant in the dopamine D4 gene) have been consistently linked to ADHD in recent molecular genetics studies. The authors note key directions for future research, including the potential importance of studies investigating gene × gene and gene × environment interactions.

Cognitive neuropsychology of depression in the elderly

In the second review, Herrmann *et al*. (pp. 1693–1702) present findings from a systematic review of studies comparing cognitive neuropsychology in older people with early- and later-onset depression and healthy controls. The authors found that, compared with those with early-onset depression and controls, those with later-onset depression tend to show greater reductions in processing speed and executive function. Compared with controls, both depressed groups show reduced function in most domains, the exception being mental state.

Attention deficit hyperactivity disorder

Four papers examine further aspects of ADHD. In the first, Andreou *et al*. (pp. 1703–1715) investigated reaction time (RT) performance in ADHD in a sample of 144 subjects with ADHD, 125 siblings and 60 controls. In line with previous research, the authors found that those with ADHD had slower and more variable RTs. They further found that ADHD was associated with improvement in RT in fast-incentive conditions. In addition, the authors estimated that around 60–70% of the phenotypic correlation was due to shared familial influences, assuming such effects result from genetic effects.

Lampe *et al*. (pp. 1717–1729) compared various motor and cognitive inhibitory functions and working memory between subjects with ADHD [$n=22$ ADHD only, 20 ADHD and borderline personality disorder (BPD)], subjects with BPD ($n=21$) and healthy controls ($n=20$). The authors found that ADHD subjects performed worse on individual tasks, had longer RTs, and higher intra-individual variance in attentional tasks. The BPD group did not differ from controls on these assessments, but did overlap with the ADHD group on some behavioural problems. The authors conclude that, while there may be overlaps in behavioural problems in ADHD and BPD, these are not underpinned by common cognitive deficits.

Monuteaux *et al*. (pp. 1731–1741) investigated the predictors, clinical characteristics, and outcomes of conduct disorder (CD) in a sample of girls aged 6–18 years with ADHD ($n=140$) and without ADHD ($n=122$), who were followed for 5 years. They found that ADHD at baseline was associated with lifetime CD (hazard ratio 5·8). In those with ADHD, childhood onset of CD was associated with parental anti-social personality disorder, and adult onset was associated with family conflict. Lifetime CD was associated with academic, psychiatric and sexual behaviour problems in those with ADHD at follow-up.

Faraone *et al*. (pp. 1743–1752) examined the relationship between pharmacotherapy for ADHD and subsequent substance use in a sample of 206 adults with ADHD, assessed retrospectively. In comparisons between subjects grouped according to lifetime history of treatment for ADHD

(no treatment, past treatment, current and past treatment), the authors found no differences in the prevalence of substance use and abuse, however measured. The authors conclude that these findings support the hypothesis that pharmacotherapy for ADHD does not cause subsequent substance use disorders.

Depression in older people

Six papers examine aspects of depression in older people. In the first, Jessen *et al.* (pp. 1753–1762) investigated variations in, and discriminators between, patterns of subjective memory impairment (SMI) in a sample of 2389 unimpaired subjects aged 75–89 years. The authors identified three clusters of SMI: (1) no memory complaints; (2) general memory complaints; and (3) general and task-specific memory complaints. In Classification and Regression Tree analyses, the number of depressive symptoms distinguished clusters 1 and 2 from 3 at the first level. In those with few depressive symptoms, delayed recall distinguished cluster 1 from 2 and 3.

Taylor *et al.* (pp. 1763–1773) investigated orbitofrontal cortex (OFC) volume, and associations with *5HTTLPR* genotype, apolipoprotein E (*APOE*) genotype and hyperintense lesion volume, in a sample of 226 depressed and 144 non-depressed subjects aged 60 years or above. The authors found that depressed subjects had smaller OFC volumes. Further, rated white-matter lesion severity was negatively associated with OFC volume. No association between *5HTTLPR* genotype and OFC volume was found. In the non-depressed group, those who were *APOE* ε4 allele-positive had larger OFC volumes.

Almeida *et al.* (pp. 1775–1786) in a sample of 5438 men aged 70 years or over, examined whether the association between C-reactive protein (a non-specific marker of inflammation) and depression in later life is due to poor physical health. The authors replicated the basic association between C-reactive protein and depression. However, once other factors, including measures of physical morbidity, were taken into account, this association was no longer significant. The authors conclude that C-reactive protein is unlikely to be a major factor in the onset and maintenance of depression in older men.

McDougall *et al.* (pp. 1787–1795) report findings from the MRC Cognitive Function and Ageing Study on the prevalence of depression in older people aged 65 years and over. In a two-stage design, with an initial screen ($n=13\,004$) and subsequent assessment of a random subsample ($n=2640$), the authors found a prevalence of depression of 9%, increasing to 10% when those with dementia were included. The prevalence was higher in women (10%) than men (7%) and was associated with functional disability, co-morbid medical disorder and social deprivation. The prevalence was lower in older age groups.

Spek *et al.* (pp. 1797–1806) report findings from a randomized controlled trial of internet-based cognitive behavioural therapy (CBT) for subthreshold depression in people aged 50 years and over. Participants were randomized to one of three groups: (1) internet-based treatment ($n=102$); (2) group CBT ($n=99$); and (3) waiting-list control ($n=100$). Compared with the waiting-list control group, both intervention groups showed significantly greater improvements on the Beck Depression Inventory at 10 weeks. There were no differences in outcome between the two treatment groups. The authors conclude that an internet-based intervention may be as effective as more commonly used group CBT for subthreshold depression in older people.

In the final paper, King *et al.* (pp. 1807–1815) investigated the relationship between various forms of religious involvement and depressive symptoms in a sample of 709 primary care attenders aged 65 years or older, who were assessed at baseline and 1-year follow-up. The authors found a number of complex relationships. For example, at baseline, there was a U-shaped association between depressive symptoms and organized religious activity, such that those with high or low levels of involvement scored higher on depressive symptoms than those with moderate levels of involvement. A similar association was found over time for private religious involvement. The authors conclude that the relationships between religious involvement and depression in older primary care attenders are complex and depend on the form of religious activity being considered.

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