1

Introduction

Philip Graham and Shirley Reynolds

The first edition of this book was published in 1998. It arose from a realisation that, despite the gradually increasing use of cognitive behaviour therapy (CBT) by child psychologists and psychiatrists, there was no systematic account of its use with children and families. The second edition appeared in 2005 at a time when it was becoming clear that the use of CBT with the young was unlikely to be a passing fashion but was here to stay (Graham, 2005). This third edition marks the establishment of CBT as the mode of therapy indisputably the best supported by scientific evidence for the majority of the conditions with which children and adolescents present to mental health services. The official national curriculum for the major British government NHS initiative 'Children and Young People's Improving Access to Psychological Therapies' (Department of Health, 2011) prescribes the use of CBT for anxiety and depressive states and encourages interventions with a CBT rationale for both conduct disorders and parent education. Further, as the international representation of the contributors to this book illustrates, CBT is flourishing worldwide, at least in the developed world. Strong innovative clinical and research activities in the field have come over recent years from the USA and Australasia as well as from many continental European countries.

The development of the CBT field over the past 15 years has drawn both on previously established traditions in psychology and psychiatry and on new trends in academic studies and clinical work. The need for a 'therapeutic alliance' in successfully engaging with children and their families, described by Boege and Ougrin in Chapter 5, resonates strongly with much earlier psychoanalytic ideas. When these authors refer to the 'relational bond between the therapist and the young person which binds the therapist and the young person together against the 'pain and vicissitudes of therapy'', one is inevitably reminded of the concept of transference though of course there are significant differences. In contrast, the approaches to the treatment of anxiety and phobic states described in Chapters 15 and 17 still retain a distinct element of behaviourism in their emphasis on desensitisation. Response prevention in the treatment of obsessive-compulsive disorders as described by Clark and Reynolds in Chapter 18 is similarly derived from behaviourist principles. Drawing again on earlier clinical approaches, the introduction of systemic ideas into a CBT formulation of clinical problems, as described by Dummett in Chapter 6, resonates with the family therapy tradition established in the 1970s. In all these cases, as the chapter authors describe, a cognitive component has been incorporated into the pre-existing approach to achieve greater effectiveness.

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But there is another tradition in psychology to which CBT has remained faithful in more than one way. Those engaged in the evaluation of therapy are still guided by the principle that it is through the falsification of hypotheses that knowledge advances (Popper, 1959). The randomised controlled trial (RCT) approach (described by Wolpert, Fugard and Deighton in Chapter 4) to evaluating a new intervention aims to *disprove* that it is superior to existing methods. If there is a failure to disprove this hypothesis then evidence exists that the new method offers a promising way forward. But this can only be a temporary advance in knowledge until the findings from another study show that even this newly gained knowledge needs to be revised or modified. Further, falsificationism permeates many CBT techniques. Socratic questioning, for example, requires clients to think critically of the beliefs they hold that may be maintaining their symptoms. Such questioning encouraging scepticism in the client is central to the CBT approach to delusions in schizophrenia as well as to the cognitive elements engaged in subverting the irrational elements of anxiety, depressive and eating disorders. Karl Popper's continuing influence in clinical child psychology and psychiatry has indeed been under-estimated (Graham, 2013).

Moving forward from traditional psychological theories, researchers have, over recent years, begun to investigate the neuroscientific and genetic basis for the changes brought about by CBT. For example, preliminary studies with adults have suggested that, when adults with obsessive-compulsive disorder were compared with controls, significant therapy-specific changes in normalised regional glucose metabolism were seen after brief, intensive CBT (Saxena *et al.*, 2009). Porto *et al.* (2009) have shown, again in adult patients with anxiety disorders, that CBT modified the neural circuits involved in the regulation of negative emotions. Neuroimaging studies have thus revealed that CBT can indeed change dysfunctions of the nervous system. Very little neuroscientific work of this nature has been carried out in children and adolescents. However in Chapter 15, Newall and her colleagues describe how, by examining the association between treatment response and the serotonin transporter gene promoter region (5HTTLPR), it may become possible to use genetic information regarding the allele configuration of anxious children as a tool to inform treatment choices. In a similar vein, in Chapter 2, Lau, Hilbert and Gregory describe how associations between the serotonin transporter gene variant and increased neural responses to the appraisal of fear in anxious and depressed adolescent patients have been identified. As they say, these findings will need to stand the test of replication in larger samples before they can be regarded as established, but the approach appears promising.

In more adventurous fashion, CBT researchers have been breaking hitherto untouched ground in applying new approaches and new technologies to the treatment of mental health problems in children and adolescents. These are outlined in Section 7 of this book. In Chapter 22, Turner and Krebs describe how more economic use of precious therapist time can be made by the use of 'low-intensity' methods of implementing CBT such as supervised self-help, running groups rather than relying on individual treatment, and delivering therapy by phone rather than face to face. In Chapter 23, Donovan, Spence and March describe how computer-based delivery of CBT has been applied in a variety of disorders, and in some cases shown to be of equal or superior efficacy than face to face methods. Given the increasing ease with which children and adolescents are outpacing their elders in the use of such technology, it is likely that such approaches will, in the future, have greater application in the young than in older clients.

The plethora of so-called third-wave CBT approaches is encroaching strongly on the adult field. These approaches consist of a loose affiliation of various CBT therapies

including: acceptance and commitment therapy (ACT), mindfulness-based cognitive therapy (MBCT) and dialectical behaviour therapy (DBT). In Chapter 24, Bögels, de Bruin and van der Oord describe preliminary work using mindfulness training applied, for example, to parents of children with autistic spectrum disorder and to adolescents with anxiety states. The cognitive limitations of younger children described by Stallard in Chapter 3 would suggest that these methods might have less applicability in the younger age groups. Even with this age group however, the evidence for the effectiveness of trauma-focused CBT as described by Trickey in Chapter 16, while requiring considerable competence in what might well be regarded as introspective mindfulness, is reasonably strong. The flexibility of CBT, even with young people who lack theory of mind (or mindfulness), is illustrated by the successful use of CBT with children who have autistic spectrum disorders as described by Wood and Schwartzman in Chapter 13. Much of this flexibility involves the use of parents in therapy with their child and, in Chapter 7, Creswell, Cartright-Hatton and Rodriguez describe a range of methods and techniques for delivering CBT to children through, and in collaboration with, their parents.

Now that CBT has acquired a dominant position among the so-called 'talking therapies' it has inevitably become a target for criticism. The label of 'reductionist' has been attached to it in hostile fashion. In fact, all constructions of the human mind inevitably and indeed appropriately reduce the complexity that governs our behaviour. The important principle was formulated by Einstein when he wrote 'everything should be as simple as it is, but not simpler'. The need to avoid over-simplicity is particularly important in relation to the use of questionnaires which, while having their uses for screening purposes, are no substitute for dialogue in assessment and therapy. It would indeed be regrettable if CBT formed part of the tick-box culture that pervades so much of our lives.

Interestingly, perhaps because of its dominant position in psychology, CBT has become a target attacked by novelists, psychologists' main competitors in the understanding of the vagaries of human behaviour. In 1923, D. H. Lawrence wrote that psychoanalysis was in danger of becoming a 'public danger'. James Joyce referred mockingly to Freud and Jung as the 'Viennese Tweedledee and the Swiss Tweedledum' (Gilbert, 1957). In 2011, no less a novelist than Ali Smith in her novel *There But For The . . .* depicts Jen, the most unsympathetic character in her book, shouting at another woman in tears at a dinner party that six sessions of CBT would 'sort her out' (p. 156). Jen 'shouts it like a mad person, and she shouts it over and over, until she has said it about six times'. In a laudatory review of a book by a contemporary psychoanalyst, Jacqueline Rose refers derisively to CBT. She praises Smith for having conveyed in her novel that 'there is something mad about a form of therapy whose vocabulary – get a grip, get CBT – possesses such frantic conviction' (Rose, 2011).

It would indeed be madness for CBT therapists to regard their approach as a panacea for all mental health problems. It is, at the moment, the intervention for mental health problems most strongly supported by the evidence. Further, in some forms of disorder, especially anxiety states and obsessive-compulsive disorder, the failure to use it might be regarded as a valid reason to sue for negligence. But the findings from the controlled trials make it clear that in most situations, while producing improvement, it by no means provides a cure, if indeed cure is an appropriate concept to use in relation to mental disorders. Further, some of the improvement obtained with the use of CBT is probably attributable to the non-specific effects of the therapy, especially of the therapist–client relationship (McQueen & Smith, 2012). It is for this reason that it is so important that

research activity, especially in the refinement of clinical methods and in scientific evaluation, is increased in future years. In the final chapter in this book we point to what seem to us to be the most promising directions such research might take.

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Section 1

Developmental cognitive theory and clinical practice

2

Anxiety and depression in young people: developmental considerations

Jennifer Y. F. Lau, Kevin Hilbert and Alice M. Gregory

Introduction

Anxiety and depression are common, disabling and costly (see Alonso *et al.*, 2004; Rice & Miller, 1998; Sobocki *et al.*, 2006). Anxiety disorders are characterised by persistent fears and worries, while depression involves chronic low mood and loss of pleasure, with effects on physical and cognitive function. Rising prevalence rates in recent cohorts (Kessler *et al.*, 2007) call for a better understanding of the root causes of anxiety and depression, and the development of preventive interventions to target these causes. Late childhood and adolescence may reflect an important focal point for studying the onset of anxiety and depression. Data show increases in the rates of anxiety and depression in the transition to, and across adolescence (Bartels *et al.*, 2011; Hankin *et al.*, 1998; Sumter *et al.*, 2009), indicating that more adolescents experience these problems, compared with children. Many adults with anxiety disorders also have their roots in youth (Beesdo *et al.*, 2009; Gregory *et al.*, 2007; Kessler *et al.*, 2005, 2007; Wittchen *et al.*, 1992; Wittchen & Fehm, 2003). Data from the National Comorbidity Survey indicate that up to 50% of adults with an anxiety disorder report onset before the age of 12 and 75% have onsets under the age of 21 years (Kessler *et al.*, 2005). These data suggest that those with a propensity to develop persistent, perhaps lifelong anxiety problems, are more likely to develop these early. There has therefore been a surge of interest in identifying risk factors for *individual differences* in the propensity to develop anxiety and depression in late childhood and adolescence. However, less is known about *developmental differences* in the propensity to develop these symptoms across life.

The main goal of this chapter is to review biological, cognitive and social changes that occur in late childhood and adolescence, which may explain the 'developmental sensitivity' for the onset of mood and anxiety problems. To do this, we consider factors that have typically been linked to *individual* differences in anxiety and depressive problems, and explore whether changes in these factors may account for *developmental* differences in risk. We aim to speculate on how new social stressors interact with 'genetic innovation' to produce changes in brain circuitry structure and function and how associated changes in emotion regulation abilities and social understanding may inform developmental sensitivity to anxiety and depression in the transition to, and across adolescence. Our proposed framework does not suggest that everyone will manifest symptoms during late childhood or adolescence. Nor do we suggest that anyone who shows an elevation in symptoms necessarily develops recurrent and persistent disorders. Instead, we simply ask why so many people who do suffer from lifelong problems of anxiety and depression typically show these signs early in life.

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Changes in the social world

Are environmental factors implicated in child and adolescent anxiety and depression?

One of the most consistent findings to emerge regarding the aetiology of anxiety and depression is the importance of environmental influences (Brown *et al.*, 1996, Kendler *et al.*, 2003), a finding that also occurs in children and adolescents (Grant *et al.*, 2004). One line of support comes from quantitative genetic studies, notably twin studies. Typically, while these can be used to estimate genetic contributions to behavioural outcomes, such as anxiety and depression (see Plomin *et al.*, 2008 for an introduction to twin studies and analyses), they can also quantify environmental contributions to the same outcomes. The degree to which not all behavioural similarity is explained by genetic factors supports the presence of 'common' environmental experiences (also known as the 'shared' environment); and the degree to which individuals differ on a behavioural outcome is assigned to individual-specific experiences (also known as the 'non-shared' environment). In short, as well as indicating the importance of 'nature', twin analyses also indicate shared and non-shared environmental influences on child and adolescent anxiety and depressive outcomes.

A notable limitation of twin studies however is that they do not identify the specific aspects of the environment that are involved. Fortunately, one can draw on a longstanding body of literature that has linked specific social factors to anxiety and depression in youth (Grant *et al.*, 2004), although much of this research has focused on research in children and young people living in Western societies. First, emotional problems have been linked to stressful life events. Such events can be proximal, precipitating the onset of symptoms, especially amongst those who have an existing predisposition. Alternatively, they can be distal, occurring in the form of early-life experiences, which have lasting effects on stress responding. These include early-life trauma and adverse events such as maltreatment and separation from parents (Brown *et al.*, 1993; Levitan *et al.*, 2003). Daily hassles and other chronic stressors have also been implicated (Eley & Stevenson, 2000). These include financial strains, usually reflected in parental socioeconomic status (Gilman *et al.*, 2003), as well as interpersonal strains. Such strains can occur in parent–child relationships, mediated via parenting style or behaviour. Indeed, controlling parenting (i.e. allowing children little autonomy) has been associated with childhood anxiety, with many studies demonstrating that mothers of anxious children allowed their children less autonomy than others (Eley *et al.*, 2010; Greco & Morris, 2002; Moore *et al.*, 2004). Negative interpersonal experiences also occur outside the home. Being rejected by peers or bullied have been associated with greater anxiety and depression (see Arseneault *et al.*, 2010; Hawker & Boulton, 2000 for reviews). While these influences may predict the onset of symptoms, often there are bi-directional associations, such that anxious and depressed adolescents may be more likely to avoid or withdraw from social situations and to attract more negative peer experiences (Gazelle & Ladd, 2003; Gazelle & Rudolph, 2004). Romantic dating, while less well-studied, has in some young people been associated with higher depression symptoms (Davila *et al.*, 2004) although other studies have shown it may act as a protective factor against emotional distress (La Greca & Harrison, 2005). Related to this, anxiety and depression have been associated with an absence of social support, which may serve to buffer against negative experiences (Barrera & Garrisonjones, 1992; Cauce *et al.*, 1992; Quamma & Greenberg, 1994).

Are there age-related changes in these environmental factors?

Again interesting insights on environmental change and continuity across age can be gleaned from more complex twin analyses. If twin analyses are performed on different-aged samples, age differences in the *size* of genetic and environmental influences across development can be tested. If twin analyses include longitudinal assessments of symptoms within a sample, this affords additional information on whether *sources* of genetic and environmental influence vary with age or remain stable. Using these methods, several twin studies now consistently indicate that shared environmental influences are more likely to play a role in anxiety and depression symptoms in young children than in adolescents (Lamb *et al.*, 2010; Rice *et al.*, 2002; Scourfield *et al.*, 2003; Thapar & McGuffin, 1994). Moreover, studies using longitudinal assessments report 'new' sources of non-shared environmental influences on depressive symptoms in adolescence (Lau & Eley, 2006; O'Connor *et al.*, 1998b).

To make sense of the findings on changes in environmental influences, one needs to consider data from studies that track changes in the wider social environment. Again the focus of most research in this area has been on children and young people growing up in Western societies. As noted above, stressful life events are a powerful predictor of the onset of symptoms. As children and adolescents mature, they may experience *more* stressful life events (Hoffman *et al.*, 1992) and daily hassles (Compas *et al.*, 1985), although not all of these reports have been replicated (Hoffmann & Cerbone, 1999). Alternatively, adolescents may experience other novel chronic stressors. For example, the transition to secondary school brings alterations in educational demands, peer groups and daily routines, and is often associated with a period of anticipatory anxiety and subsequent re-adjustment (Fenzel, 2000; Puskar & Rohay, 1999). During this period, adolescents compared with pre-transition children experience greater stress responses towards performance-related stressors (Stroud *et al.*, 2009).

Older children and adolescents in western societies also begin to spend more time with their peers than with their families (Larson *et al.*, 1996), with some suggestion that parent–child conflicts also increase (Sallinen *et al.*, 2007). In these societies romantic relationships develop for the first time, impacting on these existing social networks (Zimmer-Gembeck, 2002). These changes may serve to enhance the emotional salience of peer feedback. Indeed, adolescents exert a great deal of energy forming peer networks and soliciting peer approval (Steinberg & Morris, 2001). While having positive experiences with peers is generally beneficial, the need to gain peer approval can also result in negative outcomes. For example, some data point to more risky decisions being taken on a computer driving game, in the presence of peers (compared with playing alone) in adolescents, than in children and adults (Gardner & Steinberg, 2005). But some data also show that resistance to peers increases across adolescence (Steinberg & Monahan, 2007). As well as finding peers more rewarding, adolescents may also experience greater distress to negative peer experiences than adults. This has been demonstrated with the Cyberball paradigm (Williams *et al.*, 2000), in which participants play a computer-based ball-tossing game with two other 'players' that are in actuality computer generated. Under conditions of exclusion, the participant is rarely tossed the ball by either of the two co-players who toss to one another; under conditions of inclusion, the participant receives the ball roughly a third of the time. Recent studies exposing adolescent and adult participants to these conditions found that adolescents tended to report lower mood and more distress following ostracism relative to inclusion and baseline, than adults, who showed no difference across conditions (Sebastian

et al., 2010). Other studies have also demonstrated that adolescents undergoing the transition to secondary school may be more responsive to peer rejection than children prior to transition (Stroud *et al.*, 2009). Changes in the social world of young people could also bring with them increasingly complex exchanges, which require adolescents to acquire a more sophisticated understanding of the minds of others. For example, experimental studies in our laboratory show that adolescents deploy different strategies for establishing and maintaining reciprocity in their social relationships than adults (Belli *et al.*, 2012). In summary, certain chronic stressors known to predict mood and anxiety problems in young people may increase in the transition from late childhood to adolescence. These may include the experience of peer rejection and/or the experience of reduced peer acceptance, and perhaps the encountering of more ambiguous, perplexing social situations. These may enhance risks for developing mood and anxiety problems.

Genetic innovation across development

Are genetic factors implicated in child and adolescent anxiety and depression?

As described earlier, twin studies provide a powerful tool for estimating the heritability of mood and anxiety measures. Moderate genetic effects on early anxiety and depression have been reported (Lau & Eley, 2006; Lau *et al.*, 2007) but attempts to specify such genes have been largely unsuccessful despite advances in technology (such as the rapid advance of microarray technology, which can examine multiple genetic polymorphisms simultaneously, e.g. Gunderson *et al.*, 2005). While the short form of the serotonin transporter gene polymorphism has been implicated in adult mood and anxiety problems, findings in children and adolescents are more mixed. Some findings have been replicated, but not others. The pattern of replications and non-replications of results is not just limited to developmental samples, but across the field (e.g. for meta-analyses of associations between anxiety-related traits and the serotonin transporter gene polymorphism; see Munafó *et al.*, 2009; Schinka *et al.*, 2004) and it is becoming increasingly apparent that complex disorders such as anxiety and depression are influenced by multiple, interacting genetic and environmental influences of small effect size (see Plomin *et al.*, 2008). Thus, conclusions of specific genes in general and the few drawn from younger samples in particular await further verification in larger and more powerful studies, such as that offered by genome-wide association studies (Boomsma *et al.*, 2008; Flint *et al.*, 2012).

Researchers have begun to turn to the question of *how* genetic influences identified in twin studies take effect. Twin studies have shown how genetic risk factors may influence exposure towards particular pathological environments, and/or influence emotional responses towards these (Lau & Eley, 2008b). We have also begun to explore how genetic factors may contribute to negative cognitive styles linked to anxiety and depressive symptoms (Lau & Eley, 2008a). These data show that not only are cognitive styles heritable, but that the relevant genetic influences overlap with those contributing to depressive symptoms, suggesting there are shared genes. Finally, we have also found associations between the serotonin transporter gene variant and increased neural responses to the appraisal of fear in anxious and depressed adolescent patients (Lau *et al.*, 2009) but as with all candidate gene studies, these will need to stand the test of replication in larger samples.