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Annual Research Review: Optimal outcomes of child and adolescent mental illness

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Abstract

Background—'Optimal outcomes' of child and adolescent psychiatric disorders may mean the best possible outcome, or the best considering a child's history. Most research into the outcomes of child and adolescent psychiatric disorder concentrates on the likelihood of adult illness and disability given an earlier history of psychopathology.

Methods—In this article we review the research literature (based on a literature search using PubMed, RePORT and Google Advanced Scholar databases) on optimal outcomes for young people with a history of anxiety, depression, attention-deficit/hyperactivity disorder, conduct disorder, oppositional defiant disorder, or substance use disorders in childhood or adolescence. We consider three types of risks that these children may run later in development: future episodes of the same disorder, future episodes of a different disorder, and functional impairment. The impact of treatment or preventative interventions on early adult functioning is briefly reviewed.

Results—We found that very few studies enabled us to answer our questions with certainty, but that in general about half of adults with a psychiatric history were disorder-free and functioning quite well in their 20s or 30s. However, their chance of functioning well was less than that of adults without a psychiatric history, even in the absence of a current disorder.

Conclusions—Among adults who had a psychiatric disorder as a child or adolescent, about half can be expected to be disorder-free as young adults, and of these about half will be free of significant difficulties in the areas of work, health, relationships, and crime. Optimal outcomes are predicted by a mixture of personal characteristics and environmental supports.

Keywords

Adulthood; adolescence; prediction; epidemiology; outcome

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Introduction

> When I have promised my patients help or improvement … I have often been faced by this objection: 'Why, you tell me yourself that my illness is probably connected with my circumstances and the events of my life, [and that] you cannot alter these in any way. How do you propose to help me, then?' And I have been able to make this reply: '…much will be gained if we succeed in transforming your hysterical misery into common unhappiness. [Thus] with a mental life that has been restored to health you will be better armed against that unhappiness.'
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> Breuer & Freud, *Studies on Hysteria*, 1893–*1895 (1955, p. 305)*

Recently, the phrase 'optimal outcomes' has been invoked in psychiatry, particularly in relation to autism spectrum disorders (ASD), as a goal of treatment. 'Optimal' has several linked meanings, including (1) the best that could be envisaged for the individual in the best of all circumstances; (2) the best that can be expected across the population given the available resources; (3) the best that could be desired for a given individual, taking into account his/her history or circumstances. Wiktionary encapsulates all three aspects in its definition of optimal as: 'The best, most favourable or desirable, especially under some restriction.'

In this review we explore evidence on optimal outcomes in other, more common child psychiatric disorders. As a background, we begin by outlining some general issues that bear on the assessment of later outcomes: (1) the expected time-course of the disorder; (2) the likelihood of recovery from the disorder itself; (3) the probability of freedom from associated problems.

Onset and time course

It is now clear that most child psychiatric disorders follow relatively distinct patterns of onset and course (see e.g. Angold & Egger, 2007). Early-onset forms of psychopathology long regarded as 'life-long conditions' include ASDs. Recently, however, several researchers and clinicians have argued that 'a significant minority of children with well-documented ASD have recovered' (Helt et al., 2008) with or without treatment, although others, such as contributors to the 5th edition of the Diagnostic and Statistical Manual of the American Psychiatric Association (DSM-5; American Psychiatric Association [APA] 2013), are less convinced. Separation Anxiety Disorder, by contrast, is an example of a group of disorders described in DSM-5 as first diagnosed in childhood but rarely persisting into adulthood (DSM-5, p.192). A third group (e.g. Major Depressive Disorder) are described as episodic, and either restricted to a single episode or recurring. Finally, there are those, such as Substance Use Disorders [SUDs] and some forms of antisocial behaviour, that tend to peak in adolescence and then show a marked decline. Clearly, expectations about outcomes will vary with these variations in expected time course. Table 1 provides a summary of what DSM-5, the currently accepted taxonomy, says about the expected time course of the disorders included in this review (ICD does not discuss course and outcome). For many diagnoses the long-term prognosis for recovery is far from optimistic. This is particularly the case for most anxiety disorders; for example, DSM-5 warns that in cases of Panic Disorder

or Generalized Anxiety Disorder ‘rates of full remission are very low’ (DSM-5, p.223). And whereas DSM-IV (APA, 1994) stated confidently that in ADHD, symptoms attenuate during late adolescence and adulthood in most individuals (DSM-IV, p.82), a much worse prognosis is given in DSM-5: ‘A substantial proportion of children with ADHD remain relatively impaired into adulthood’ (DSM-5, p.62).

Expected recovery from a psychiatric disorder

Questions about how to define optimal outcomes apply across this range of expected time-courses. Is the optimal outcome the absence of a diagnosis - and if so, how often and after how long can it be expected? Should treatment aim at producing an ‘optimal’ child, functioning at least at the population norm for developmental stage? Or should we have different expectations for children with different backgrounds and psychiatric histories? The timing of outcome assessments will also be important. Outcomes may be measured immediately after treatment or at particular stages of development, and timing of assessment may clearly affect whether the individual is found to have achieved an optimal outcome. In this review article we aimed to take a longer-term view, and focus on outcomes measured beyond childhood and adolescence; in practice, this generally means the 20s or 30s, since much of the currently available literature confines itself to this interpretation and this time period.

Expected freedom from associated problems

Built into most countries’ societal and fiscal assumptions about children and adolescents – including those with histories of psychiatric disorder and many developmental disabilities – is that they will become independent adults somewhere between ages 18 and 25. Publicly-funded support for education and health care often tapers or comes to an end at that stage, as do child social services; if support is still needed it will have to be applied for all over again, often from a different agency. Apparently our societal expectation is that by the developmental stage defined as ‘adulthood’ all of us, no matter our earlier problems, will have achieved ‘skills at a level and with a quality that *reaches the trajectory of typical development in most or all areas*’ (Degenhardt et al., 2013); that is, enough to function independently. But even if children recover from a mental illness in the strict sense of no longer meeting full diagnostic criteria, they may be left more vulnerable to future episodes, or to sub-diagnostic symptom clusters, or to other psychiatric disorders, or to psychosocial or medical problems that make it harder for them to function as independent adults. For some, the experience may strengthen them in some or all areas of functioning. Assessments of optimal outcomes need to include evidence on these other aspects of functioning as well as on disorder *per se*.

Literature available

Our search strategy has used the usual sources (PubMed, Google Advanced Scholar, NIH RePORT, etc.), informed by our experience with longitudinal research in child and adolescent psychopathology. In the event, only a handful of studies met our needs (reviewed in Rutter, Kim-Cohen and Maughan (2006); see also Clark, Rodgers, Caldwell, Power and Stansfeld (2007); Fergusson, Boden and Horwood (2007); Naicker, Galambos, Zeng,

Senthilselvan and Colman (2013); Pine, Cohen, Gurley, Brook and Ma (1998); Rohde, Lewinsohn, Kahler, Seeley and Brown (2001); Steinhausen (2006); Kim-Cohen, Caspi, Moffitt, Harrington, Milne and Poulton (2003); and other papers from the same research groups). In principle, studies of adults could provide valuable sources of information here; in practice, however, most countries lack the earlier data needed to tell what proportion of newly-fledged adults had psychiatric disorders earlier in their lives, how they functioned then and how they are functioning as adults. And although retrospective and multiple-cohort cross-sectional studies can provide information on adult risk from child exposures at the *group* level (e.g., as odds ratios), longitudinal studies are needed to calculate *which individuals* recover and why. Accordingly, wherever possible this review concentrates on findings from prospective, longitudinal research. In addition, to reflect expectable outcomes for the majority of young people with early mental health difficulties, we focus so far as possible on findings from studies of representative, community-based samples of children and adolescents.

We focus on four disorders/disorder groupings, chosen to represent disorders with differing expected time courses: depressive and anxiety disorders; SUDs; disruptive and antisocial disorders; and ADHD. In each case, we explore evidence in relation to five main questions:

1. Does the outcome of a given diagnosis (Dx1) include increased risk of the same diagnosis in the future?
2. Does the outcome of a given diagnosis (Dx1) include increased risk of a different diagnosis (Dx2) in the future?
3. Does the outcome of Dx1 include increased risk of poor health and functioning?
4. What treatments or preventative programs work to improve outcomes of Dx1?
5. What factors predict optimal outcome after Dx1?

Depressive and anxiety disorders

We review depression and anxiety disorders together as ‘emotional disorders’, because taxonomic and genetic studies have suggested that the mood and anxiety disorders can be collapsed together into an overarching class of emotional disorders (Watson, 2005) (although this did not happen in DSM-5). In fact the overlap between the two diagnostic groups is mainly confined to depression and Generalized Anxiety Disorder (GAD) (Copeland, Angold, Shanahan, & Costello, 2014), and many longitudinal studies do not distinguish among the anxiety disorders. Emotional disorders are clinically perceived as episodic, although treatment may extend long after the end of the severest phase, sometimes with the goal of preventing recurrent episodes (Baldessarini et al., 2012).

Does the outcome of emotional disorders include increased risk of the same diagnosis in the future?

It is now clear from several longitudinal studies that children who have early episodes of emotional disorders are at increased risk into adulthood (Shanahan, Copeland, Costello, & Angold, 2011). Despite different estimates of point prevalence across studies, the conclusion that half of youth with depression will have an adult episode of a depressive or anxiety

disorder is well-substantiated (e.g., Rohde, Lewinsohn, Klein, Seeley, & Gau, 2013; Wilson, DiRago, & Iacono, 2014; Copeland, Shanahan, Costello, & Angold, 2009; Naicker et al, 2013; McLaughlin, Green, Gruber, Sampson, Zaslavsky, & Kessler, 2010; Keenan-Miller, Hammen, & Brennan, 2007; Patton et al., 2014). For example, in the Oregon Adolescent Depression Study (OADS) (Rohde et al., 2013), childhood depression doubled the risk of adolescent depression, child or adolescent depression doubled the risk of depression in early adulthood, and depression at any time up to the early 20s almost tripled the risk of adult depression (43% vs.16%).

Looking at the data from the viewpoint of optimal outcomes, more than half of those with adolescent depression in OADS had no further episodes in early adulthood, and only those with multiple adolescent episodes were at risk (Lewinsohn, Rohde, Seeley, Klein, & Gotlib, 2000). It is also worth noting that the Woodlawn longitudinal study of a predominantly urban African American sample found that childhood depression did *not* increase the likelihood of adult depression in either sex (Green, Fothergill, Robertson, Zebrak, Banda, & Ensminger, 2013.) The Great Smoky Mountains Study (GSMS), a longitudinal study of rural youth, also found that, after adjusting for comorbidities, adolescent depression did not predict adult depression (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003). However, in other data sets adolescent depression has been found to carry an elevated risk of adult depression irrespective of comorbidity (Fombonne, Wostear, Cooper, Harrington, & Rutter, 2001). Very few studies provide information on anxiety disorders separately (Bögels, Knappe, & Clark, 2013; Benjamin, Harrison, Settipani, Brodman, & Kendall, 2013) but they clearly have different trajectories (Copeland et al, 2014).

Retrospective studies using adult samples find that over half of those with a lifetime history of depression or anxiety had the diagnosis in the past 12 months (Kessler et al., 1994; Kessler, Berglund, Demler, Jin, Merikangas, & Walters, 2005; Kessler, Chiu, Demler, & Walters, 2005). This suggests that the optimal psychiatric outcome – no diagnosis – is achieved in fewer than half of adults at best.

Does the outcome of emotional disorders include increased risk of a different diagnosis in the future?

Although early emotional disorders have a less-than-promising prognosis, it is worth noting that this may have to do with comorbidities with other disorders and symptoms. For example, depression is much less likely to have a poor outcome if it is not comorbid with Oppositional Defiant Disorder (ODD) (Costello et al., 2003). Recent studies suggest that aspects of the new DSM-5 Disruptive Mood Dysregulation Disorder (DMDD) are linked to depression via irritability, which is a symptom of both depression and ODD (Brotman et al., 2006; Stringaris, Zavos, Leibenluft, Maughan, & Eley, 2012). There is an unresolved debate about whether adolescent depression predicts adult SUDs (the ‘self-medication’ hypothesis (Kuo, Gardner, Kendler, & Prescott, 2006), or whether, on the contrary, substance use is depressogenic (Fergusson, Boden, & Horwood, 2009). Naicker and colleagues, using the Canadian National Population Health Survey (Naicker et al., 2013), found that adolescent depression predicted not only adult depression but also alcohol abuse, smoking, and migraines. In a two-year follow-up of the National Epidemiologic Survey on Alcohol and

Related Conditions (NESARC), early adult depression predicted adult alcohol use disorders (Dawson, Grant, Stinson, Chou, Huang, & Ruan, 2005). Longitudinal studies of youth have quite consistently shown that early depression predicts later anxiety disorders, in particular GAD (Costello, Mustillo, Keeler, & Angold, 2004; Benjamin et al., 2013), and prediction from anxiety disorders to later depression is also reported (Costello et al. 2004).

Does the outcome of emotional disorders include increased risk of poor health and functioning?

The ability to function as an independent adult is a developmental goal of adolescence. Adult competencies include accepting responsibility for oneself, the capacity to care for others, including children, independent decision-making, and financial independence (Arnett, 2000). In a Swedish sample (Jonsson, Bohman, von Knorring, Olson, Patton, & von Knorring, 2011) adolescent depressed females were more likely, as adults, to be divorced, single parents, to have miscarried, experienced intimate partner violence, or to have had a sexually transmitted disease. In the Brisbane birth cohort study (Keenan-Miller et al, 2007), controlling for adult depression, early adolescent depression continued to be associated with poorer interviewer-rated health, poorer self-perceived general health, higher health care utilization and increased work impairment due to physical health, although not with limitations to physical functioning or the presence of chronic medical conditions. In GSMS four areas of adult functioning were defined as contributing to ‘optimal outcomes’ in adulthood: (1) health; (2) education and income (SES); (3) social relationships; and (4) criminality or self-injurious behavior (Copeland et al., 2014). In all four areas participants with early depression and/or anxiety were significantly worse off than those with no psychiatric history, and were the most likely of any diagnostic group to perform poorly as adults. However, this analysis did not control for adolescent comorbidity (e.g., depression with SUD).

What treatments or preventive interventions reduce risk of onset, recurrence, and adult disability from emotional disorders?

There are few studies that follow depressed or anxious children into adulthood following treatment, and there is a very real risk of publication bias (failure to publish non-significant successes). Harrington et al.’s follow-up of children treated at the Maudsley Hospital in the 1960s showed that, compared with non-depressed psychiatric patients, the depressed group was more likely to have one or more episodes of adult depression: by age 30 fewer than half were unaffected, compared with more than 70% of psychiatric controls (Harrington, Fudge, Rutter, Pickles, & Hill, 1990). By definition, all the cases had received treatment, but this study preceded the use of psychopharmacological treatments for depression in children. Kovacs’s early studies of children referred for treatment found that ‘the presence or absence of treatment had no observable impact’ (Kovacs, 1985, p. 387). A recent example of a treatment study that included the use of SSRIs is the multi-site Treatment for Adolescents with Depression Study (SOFTADS), which found that 96.4% of participants had recovered by 42 months. However, only half of these (53.3%) were still free of depression throughout the 3-year follow-up period (Curry et al., 2011). In a review of the effectiveness of cognitive-behavioral treatment for anxiety disorders, Saavedra and colleagues (Saavedra,

Morgan-Lopez & Kurtines, 2010) found that 8 to 13 years after various types of cognitive behaviour therapy (CBT), all groups showed improvement in both anxiety and depression symptoms. However, there was no untreated control group. The authors also emphasize the importance of *successful* treatment: treated youth who did not improve significantly showed higher rates of panic disorder, alcohol dependence, and drug abuse in adulthood than successfully-treated youth (Saavedra et al., 2010). What is most remarkable in reviewing studies of the adult outcome of early affective disorders is, in fact, the minimal attention paid to effects of treatment; it is rarely included even as a potential mediator in long term studies.

What factors predict optimal outcomes after emotional disorders?

Evidence from cross-generational, twin, and family studies makes it clear that depressed children without a family history of depression stand a better chance of a full recovery than do those with a family history (Weissman et al., 1999). A question that arises is the relative contribution of genes and environment to this pattern. Longitudinal studies of twins have shown that the genetic contribution to twins' similarity in their depression history diminishes across early adulthood, and reaches a plateau by middle age (Jones, 2013). This suggests that the similarity seen in child and adolescent twins is partly environmental, and fades as twins create their own environments in adulthood. Using adults' retrospective recall of the way their parents treated them, parental coldness was the strongest environmental predictor of adult depression (Jones, 2013).

The OADS (Lewinsohn et al., 2000) used a prospective design in a community sample of adolescents to examine variables before and after first onset MDD as predictors of recurrent MDD over a 12-year follow-up (Rohde et al., 2013). Parental history of MDD and lifetime history of minor depression prior to MDD onset significantly predicted recurrent MDD. Following MDD onset, a higher number of major life events significantly predicted recurrence. In the same study, low levels of excessive emotional reliance, a low proportion of family members with recurrent major depressive disorder, low levels of antisocial and borderline personality disorder symptoms, and a positive attributional style (males only) independently predicted which formerly depressed adolescents would remain free of future psychopathology. Female gender, multiple major depressive disorder episodes in adolescence, a higher proportion of family members with recurrent major depressive disorder, elevated borderline personality disorder symptoms, and conflict with parents (females only) independently predicted recurrent major depressive disorder. Comorbid anxiety and substance use disorders in adolescence and elevated antisocial personality disorder symptoms independently distinguished adolescents who developed recurrent major depressive disorder comorbid with non-mood disorder from those who developed pure major depressive disorder.

The question is how these early stressors affect adult functioning in a way that increases risk for recurrent anxiety or depression (Morales & Guerra, 2006). Pathways suggested include cumulative or additive strain, rather than the specific effect of individual stressors. Cumulative strain operates perhaps by preventing the development of necessary adult skills, such as self-regulation (Morales & Guerra, 2006).

Findings from the Christchurch Health and Development Study in New Zealand showed that family bonding and parent child relations were modestly protective against adult depression, even after controlling for earlier depression and other environmental factors (Raudino, Fergusson, & Horwood, 2013). Family conflict, rather than childhood depression, was the strongest predictor of adult depression in the mainly-minority Woodlawn study (Green et al, 2013).

Summary: Depression and anxiety

The optimal outcome, recovery from depression and anxiety disorder, can be expected in about half of child and adolescent cases found in community samples and followed through into their 20s. Even in the event of no further episodes, however, there appears to be a 50% risk of residual damage in the form of poor adult functioning. Interestingly, longitudinal studies of clinical or high-risk samples (Harrington et al., 1990; Weissman et al., 1999) also report that about half of all patients have had no recurrence of depression by early adulthood. The reason for this similarity may be that so few depressed youth get treatment that there is little difference between treated and epidemiologic samples. Whereas a family history of emotional disorders and the presence of comorbid difficulties are associated with increased risks of recurrence, a positive emotional style, family bonding and good parent-child relationships may be protective against the likelihood of adult depression. Exposure to cumulative strains may compromise the development of compensatory coping skills.

Attention-Deficit/Hyperactivity Disorder

Does the outcome of ADHD include increased risk of the same diagnosis in the future?

We turn next to a disorder with a very different expected time-course: attention deficit-hyperactivity disorder (ADHD). ADHD is now widely viewed as a neurodevelopmental disorder that onsets early in childhood and carries potentially long-term consequences. The clinical presentation evolves across development, with symptoms of hyperactivity becoming less prominent with age and those of inattention more so (Cherkasova, Sulla, Dalena, Pondé & Hechtman, 2013). In addition, from middle childhood onwards evidence from follow-up studies points to an age-dependent decline in rates of disorder and impairing symptoms that continues into the early adult years (Faraone, Biederman & Mick, 2006). Although ADHD often represents a long-term, impairing condition, for some young people it clearly does remit.

At this stage, specifying the extent of that remission is more difficult, in part because most current evidence is based on referred samples, and in part because – in the absence until recently of agreed criteria for defining ADHD in adulthood - investigators have taken somewhat different approaches to characterizing persistence/remission. The implications of these variations can be marked; in their meta-analysis, for example, Faraone et al., (2006) reported that while only around 15% of childhood cases met full (DSM-IV) criteria for disorder by age 25, a further 40–60% continued to face impairments associated with residual symptoms. The few follow-ups of girls with ADHD (see e.g. Biederman, Petty, O'Connor, Hyder & Faraone, 2012a; Hinshaw et al, 2012) paint a broadly similar picture.

The longest-term follow-up reported to date extends findings to mid-life. Klein et al (2012) followed a sample of 6–12 year old boys referred for treatment in the 1970s, with difficulties consistent with DSM-IV ADHD-combined type. Re-interviewed at age 41, 22% met full DSM-IV criteria for ADHD and a further 10% met reduced symptom criteria. The limited data on non-referred samples are broadly consistent with this picture. Moyá, Stringaris, Asherson, Sandberg and Taylor (2014), for example, found that 22.5% of pervasively hyperactive 6–7 year-old boys identified from a community sample met criteria for ADHD at age 27, while Lara et al. (2009) estimated a 50% persistence rate (varying markedly between countries) from retrospective reports of childhood ADHD gathered in the World Health Organization World Mental Health Surveys. Although a substantial minority of children with ADHD are likely to experience impairing symptoms well into adult life, for some – currently estimated by a number of commentators at around one in three - the disorder does appear to remit with age.

Does the outcome of ADHD include increased risk of a different diagnosis in the future?

In childhood, ADHD shows strong overlaps with a range of other difficulties including lower IQ, specific learning difficulties (see e.g. Sexton, Gelhorn, Bell, & Classi, 2012), ASDs, and other psychiatric and behavioural disorders (Thapar, Cooper, Eyre & Langley, 2013). Ideally, assessments of longer-term outcomes need to take account of these associated difficulties in evaluating the outlook for ADHD *per se*.

Prospective data from community as well as referred samples provide evidence on associated disorders later in development. Copeland et al. (2013) report findings from a combined analysis of data from the Christchurch, GSMS and Dunedin Multidisciplinary Health and Development Study longitudinal cohorts. Between childhood and adolescence (data from Dunedin and GSMS only), the only significant prediction from childhood ADHD, after controlling or all other disorders, was to adolescent ODD. This echoes numerous other reports that ADHD is a key risk factor for disruptive behaviour in childhood; that it is associated with the persistence of conduct/antisocial difficulties in childhood/adolescence; and that it continues to be associated with new onsets of disruptive behaviour problems in the teens (see, e.g., Langley et al, 2010). Between adolescence and early adulthood (20s and early 30s) Copeland et al. (2013) found that associations with later anxiety disorders became non-significant after adjustment for other comorbidities, but a doubling of risk for SUDs remained robust. No increased risks for ASPD were evident in the GSMS (Copeland et al, 2009) or Dunedin (Kim-Cohen et al, 2003) samples, and unadjusted associations in the Christchurch sample (Fergusson, Boden, & Horwood, 2010) became non-significant when controlled for comorbid adolescent disorders. With the exception of substance use, these findings in community samples thus suggest that child and adolescent ADHD is not associated with markedly increased risks of other disorders early in adult life.

Though details vary somewhat, follow-ups of referred samples of males lead to broadly similar conclusions. Biederman, Petty, Woodworth, Lomedico, Hyder, & Faraone (2012b), for example, found that although *life-time* rates of antisocial, anxiety, mood and substance disorders were elevated in their ADHD probands at age 27, with the exception of anxiety disorders and smoking, *interval* (past five year) rates were not. The authors speculated that

comorbid disorders may emerge early in development but then remit, or that stimulant treatment may help mitigate their development. Klein et al. (2012), in their mid-life follow-up, also concluded that the period of increased risk for onset of new disorders was limited to adolescence. Follow-ups of female samples with ADHD paint a less optimistic picture (see, e.g., Hinshaw et al., 2012), with increased risks of CD/ODD, depression, anxiety, suicidality and self-harm in early in adulthood; we must await longer-term follow-ups to know how far these risks persist to older ages.

Does ADHD increase risk of future poor health and functioning?

Evidence from both referred and community samples is unequivocal in showing that as a group, children with ADHD are at increased risk for difficulties in a range of aspects of adolescent and adult health and social functioning (see Chersakova et al. [2013] for a review). Some of the most consistent associations are with poor school achievements/ educational outcomes. In the National Comorbidity Survey-Adolescent supplement, adolescents with ADHD were at much increased risk of low average grades (Kessler et al, 2014), while in the Christchurch study 54% left school without qualifications and only 7% (by contrast with 39% in the non-ADHD portion of the sample) had achieved a university degree by age 25 (Fergusson et al, 2010). In both studies additional analyses suggested that these poor educational outcomes were primarily attributable to ADHD and not to associated difficulties.

Many follow-up studies also report increased risks of crime. In community samples at least, however, these seem primarily attributable to co-occurring conduct problems and aggression (see e.g. Fergusson et al, 2010; Pingault, Coté, Lacourse, Galera, Vitaro & Tremblay, 2013). In the Christchurch sample increased risks of teen pregnancy/fatherhood and inter-partner violence in the early 20s were also reduced to non-significance by controls for confounders. In referred samples there is evidence that young people with ADHD may take longer to achieve independence from parents than their non-ADHD peers, and that elevated levels of family conflict may persist to adulthood (Biederman et al, 2012b); difficulties of this kind may be less marked in community samples (Moyá et al, 2014).

Higher rates of driving violations, accidents and injuries are widely reported, and (in males at least) potentially mitigated by the effects of medication (Chang, Lichtenstein, D'Onofrio, Sjölander & Larsson, 2014). Nigg (2013) has recently summarized evidence on a range of adverse health outcomes; risks for obesity are attracting particular attention here, with prospective evidence that ADHD may constitute an independent risk (Khalife et al, 2013).

What ADHD treatments work? What preventions work?

Current guidelines (see, e.g., NICE, 2008) recommend multimodal treatments for school-age children with moderate to severe ADHD, combining use of stimulant medications with parent training/education programmes and/or CBT or social skills training for young people themselves. In the short term, pharmacological treatments can be extremely effective, with reported effect sizes of .7 and higher (Faraone & Buitelaar, 2010). Not all parents are comfortable with using medications, however, and compliance is often poor, especially among adolescents. In addition, long-term effects remain uncertain. In the NIMH

Collaborative Multisite Multimodal Treatment Study of Children With Attention-Deficit/ Hyperactivity Disorder (MTA, Molina et al, 2009), although symptom improvements after the intensive 14-month intervention were largely maintained, follow-ups in adolescence showed no differential effects according to treatment type (medication, behavioural, or combined), and the ADHD samples remained considerably impaired by comparison with community controls on a wide range of indicators.

Sonuga-Barke and colleagues (2013) undertook meta-analyses of findings on non-pharmacological interventions including dietary treatments, cognitive training, neurofeedback and behavioural programmes. Focusing specifically on effects on ADHD symptoms they found small but significant reductions associated with supplementation with free fatty acids, and indications that excluding artificial food colour may be beneficial for children with food sensitivities. Psychological treatments showed significant effects on 'most proximal' outcome assessments (typically, ratings by parents), but these dropped to non-significance (and in some instances to zero) in analyses based on blind or probably blind assessments of outcomes. Although parent-training and other behavioural interventions are helpful in managing the disruptive behaviours that often accompany ADHD, they have not yet shown evidence of efficacy in relation to ADHD symptoms *per se*.

Recognizing the challenges of achieving sustained improvements once ADHD symptoms are established, investigators are increasingly exploring the possibility of early interventions. Halperin, Bedard and Curchack-Lichtin (2012), for example, highlight the potential of both physical exercise and environmental enrichment in facilitating brain development in preschoolers, and Sonuga-Barke and Halperin (2010) review a range of promising approaches to cognitive enhancement in young children. Because co-occurring learning difficulties almost certainly contribute to some poor later outcomes, strategies directly targeting these conditions also seem likely to be valuable (Sexton, Gelhorn, Bell, and Classi, 2012). And finally, for those young people whose symptoms do persist beyond adolescence, there are increasing calls for improved transitions from child to adult mental health services, and the provision of continued treatment to optimize outcomes for individuals with ADHD in adult life (Asherson, Manor & Huss, 2014).

What factors predict optimal outcome in ADHD?

Current evidence points to a variety of factors that are associated with, and may be implicated in, variations in ADHD outcomes over time. The severity of initial symptoms, along with the presence of comorbid disorders in childhood, are widely supported, and seem associated with both the persistence of ADHD and risks of associated impairments (Chersakova et al., 2013). Disruptive disorders may be especially important here given their strong independent associations with adverse social consequences, which, as we outline in later sections, can markedly constrain later life-chances.

Beyond the behavioural domain there is much interest in the roles of cortical development and neuropsychological functioning in the course of ADHD. Evidence from longitudinal structural imaging studies has shown, for example, that ADHD is characterized by a delay in structural brain maturation, suggesting that structural and functional normalization of the

cortex may underlie symptomatic remission (Shaw, Gogtay & Rapoport, 2010). Consistent with this view, evidence is emerging that variations in adult symptom severity are associated with variations in cortical trajectories (Shaw, Malek, Watson, Greenstein, de Rossi, & Sharp, 2013). Findings on the role of executive functions (Halperin & Schulz, 2006) are less consistent, but a number of studies have noted that higher IQ is associated with more positive treatment response and later outcomes. Pathways for these effects are unclear, but may include a compensatory role for higher IQ, supporting the development of more positive coping strategies.

Childhood ADHD is highly heritable, and twin studies have identified strong genetic influences on persistence from childhood to adolescence (see Franke et al, 2012); comparable evidence is not yet available on the transition to adulthood. In one follow-up study family history emerged as a predictor of persistence from childhood to adolescence, but not from adolescence to early adult life, suggesting that familiality may be associated with later moves to remission (see Chersakova et al, 2013).

Finally, we note that psychosocial factors (whether in the form of positive supports or adverse exposures) have received relatively little attention as predictors of persistence/remission in ADHD. In their retrospective study of adults, Lara et al (2009) found that persisting ADHD was not associated with childhood adversities such as neglect or maltreatment; higher childhood SES was, however, linked with better initial treatment response and long-term outcomes in the MTA (Molina et al, 2009). In childhood, ADHD is often marked by problems in peer relationships. Mrug et al. (2012) found that peer rejection made independent contributions to later assessments of poor global functioning, but that having a reciprocal friend did not offset these effects. There is little evidence at this stage on the extent to which a supportive psychosocial environment later in development can help mitigate the impact of symptoms in adult life.

Summary: ADHD

Follow-up findings have been critical in demonstrating that ADHD can and does persist to adulthood in some young people, with associated impacts on social functioning. Available treatments can be highly effective in the short term, but longer-term impact remains uncertain; as a result, there is a clear need for continuing service provision for those whose symptoms persist. At the same time, follow-up findings equally clearly demonstrate remission in a significant minority of cases, and levels of social functioning that may not deviate too markedly from the norm provided they are not complicated by poor educational attainments or more general behaviour problems. Evidence is emerging that symptomatic remission may be associated with aspects of cortical development, and that higher IQ may support the development of coping strategies; at this stage, the role of psychosocial supports is less clear. Further study of these differing long-term trajectories are likely to hold important lessons for aetiology as well as course.

Disruptive and Antisocial Disorders

Does the outcome of disruptive disorders include increased risk of the same diagnosis in the future?

The long-term outlook for childhood disruptive and antisocial behaviours has been studied for many years. Robins' classic follow-back of clinic attenders was published almost 50 years ago now (Robins, 1966), and her replication of those findings just over a decade later (Robins, 1978). In four cohorts of young men growing up in different historical eras, in different parts of the US, and from different ethnic backgrounds, a consistent picture emerged. Looking backwards from adulthood, severe adult antisociality virtually required a history of childhood conduct problems. Looking forwards from childhood, however, the conclusion was quite different; even in clinical samples, the majority of those with antisocial difficulties in childhood did *not* go on to be severely antisocial later in life.

In general, subsequent studies of diagnostically defined samples have reached similar conclusions: only a minority of children and adolescents with disruptive behaviour disorders go on to show severe antisocial tendencies later in life. Strictly, of course, it is not possible to say how far young people with disruptive disorders are at risk of 'the same' disorder in adulthood, because Conduct Disorder (CD) and ODD are not typically assessed beyond the late teens; instead, studies have focused on Antisocial Personality Disorder (ASPD) or indicators of violence/aggression or crime. In referred samples of boys, persistence from child or adolescent CD to later ASPD is below 50% (Burke, Waldman & Lahey, 2010); in community samples, child and adolescent CD are still significant risk factors for ASPD, but the proportions of young people affected in early adulthood are much lower (see, e.g., Copeland et al., 2009). Evidence on continuities from ODD is more limited; where it has been explored separately, however, findings suggest that associations between ODD and ASPD often run via CD. In GSMS neither childhood nor adolescent ODD was associated with any increased risk of ASPD in early adulthood (Copeland et al, 2009). In the Christchurch study associations were detectable, but only14% of 14–16 year-olds with ODD went on to meet criteria for ASPD between ages 18 and 25 (Fergusson, Boden & Horwood, 2010).

Other approaches to conceptualizing and operationalizing childhood behaviour problems point to similar conclusions. Fergusson, Horwood and Ridder (2005), for example, used a dimensional measure of teacher and parent-rated conduct problems at ages 7–9 years to predict outcomes at ages 21–25. Severity of early conduct problems showed a dose-response relationship with poor later outcomes, but even among those in the top 5% of the conduct problem distribution under 20% met criteria for ASPD in early adulthood, and just under a third had been arrested for/convicted of a crime.

Studies based on Moffitt's (1993) developmental taxonomy highlight other issues. First, a 'childhood limited' sub-group has emerged, where childhood behavior problems remit before or during adolescence (see, e.g., Barker & Maughan, 2009; Odgers et al, 2008); in population samples this pattern appears to apply to a majority of those with early onset difficulties. Second, in follow-ups to adulthood, 'adolescent limited' sub-groups have shown a somewhat less benign outcome than initially assumed. Third, while individuals on 'life

course persistent' (LCP) trajectories do indeed have markedly elevated rates of continuing antisocial behaviour in adulthood, even in these highly antisocial sub-groups significant proportions do eventually desist. In the Dunedin cohort, for example, Odgers et al. (2008) found that among LCP men (some 8–10% of the study males), just under a third had a conviction for a violent offence between ages 26 and 32, and just over half (59%) had some involvement in self- or other-reported violence. Outcomes for women on the LCP pathway were more adverse. Although girls are much less likely than boys to show severe antisocial behaviour in childhood, for the small group who do, the long-term outlook may be poor.

Does the outcome of disruptive disorders include increased risk of a different diagnosis in the future?

Looking beyond the antisocial domain, studies highlight increased risks of a spectrum of other disorders, internalizing as well as externalizing, for young people with conduct problems in childhood; indeed, follow-back analyses in the Dunedin cohort showed that child and adolescent CD/ODD formed part of the developmental history of *all* the early adult disorders assessed (Kim-Cohen et al., 2003).

Increased risks of later substance use are among the best-established outcomes of CD. Young people with CD start using both legal and illegal substances earlier than their peers (Hopfer et al., 2013), putting them at higher risk of developing SUDs. As we discuss in more detail below, substance use and substance disorders are relatively common in mid- to late adolescence, but decline thereafter; as a result, estimates of the increased risk for young people with conduct problems may vary with the age at which outcomes are assessed. By age 32, however, comparisons between men on the LCP and low antisocial trajectories in the Dunedin sample showed clearly elevated rates of alcohol dependence (21% vs 8%), cannabis dependence (20% vs 5%) and other illegal drug dependence (22% vs 1%). Although most antisocial young people are unlikely to face ongoing drug or alcohol problems in adulthood, a sub-group of those with the most severe and persisting difficulties does appear to be at considerably increased risk.

Heterotypic continuities between CD/ODD and later internalizing disorders are also consistently reported. For example, in the Christchurch study (Fergusson et al., 2010) any elevated level of childhood conduct problems above the 50% percentile was associated with an increased risk of anxiety/depression at ages 21–25. As noted earlier, other evidence suggests that ODD-related behaviours, and in particular irritability, may be among the key risk markers here.

Do disruptive disorders increase risk of future poor health and functioning?

Alongside 'disorder' outcomes, follow-up studies have consistently documented a litany of adverse health and social sequelae of early antisocial behaviours. Fergusson et al. (2005), for example, found dose-response relationships between severity of childhood conduct problems and poor educational and occupational achievements; lower earnings; problems in sexual and partner relationships; early parenthood; and, in subsequent follow-ups, problems in parenting. Associations with educational and occupational outcomes were largely

attributable to confounding from IQ and attentional difficulties; all other associations with early conduct problems remained robust.

Subsequent studies have shown that poor pychosocial and mental health outcomes persist at least to mid-life (Colman et al, 2009), and may indeed cumulate over time. Physical health constitutes a further area of vulnerability. Odgers et al. (2008) documented higher rates of poor health-related behaviours and chronic disease markers in LCP (and to an extent, adolescent onset) groups in the Dunedin cohort in early 30s, and long-term follow-ups of both representative (Maughan, Stafford, Shah & Kuh, 2014) and high risk samples (see e.g. Laub & Vaillant, 2003) have identified increased risks of premature death among antisocial youth.

What treatments work? What preventions work?

> On the basis of experimental research and systematic reviews, we now know that many types of interventions are effective, including nurse home visiting, cognitive-behavioral skills training, parent management training, preschool intellectual enrichment programs, multi-systemic therapy, mentoring, hot spots policing, and drug courts. We also know that Scared Straight and convictions of juveniles are damaging and that boot camps, more intensive supervision, and many correctional treatments are ineffective.
>
> Farrington (2013, p 502)

As this summary suggests, there is now extensive evidence base on the range of interventions that can work (and some that clearly do not) in the prevention and treatment of childhood antisocial behaviour. Many programmes are designed to prevent the emergence/escalation of behaviour problems early in development, and show large effect sizes at that stage (Comer, Chow, Chan, Cooper-Vance & Wilson, 2013). At older ages, multi-systemic family therapy also has strong evidential support (Henggeler, 2011). Follow-ups of early childhood interventions are now beginning to report positive findings in the late teens, with indications of better outcomes for children who were initially most severely affected (see e.g. Bierman et al, 2011; Scott, Briskman & O'Connor, 2014). Reaching to adulthood, a meta-analysis of effects of a range of early prevention programmes (Dekovic, Slagt, Asscher, Boendermaker, Eichelsheim & Prinzie, 2011) concluded that they '…can help put children on a more positive developmental trajectory, but there is still no convincing evidence that they can prevent adult crime' (p 542). As the authors note, however, it may be unrealistic to expect a single intervention early in development to redirect long-term trajectories. Instead, approaches more akin to a 'dental model' may be required, with on-going check-ups and booster sessions needed to capitalize on early gains and support families through the new challenges that arise as young people mature.

What factors predict optimal outcomes in disruptive and antisocial disorders?

Current evidence points to at least two distinct periods when 'recovery' from disruptive/antisocial behaviours is most likely to occur: in middle childhood (when the 'childhood limited' (CL) groups identified in trajectory-based studies emerge), and in late adolescence/early adulthood, when many young people desist from antisocial behaviour and crime.

Perhaps surprisingly, relatively little is known about predictors of recovery in 'childhood limited' behaviour problems. Barker and Maughan (2009) compared CL and Early Onset Persistent (EOP) groups on prenatal and early childhood risks; in addition to showing higher temperamental activity levels, children in the EOP class had been exposed to higher rates of prenatal anxiety, poor relationships between parents, and harsh parenting. There are also pointers that integration in the school context may aid reductions in behaviour problems in some young children.

Declines in antisocial and delinquent behaviours in the teens/early twenties form part of the more general pattern of desistance from offending reflected in the age-crime curve (Farrington, 1986). Several different processes are likely to be involved here, some involving maturation of factors such as impulse control and moral reasoning, and other depending on involvement in adult roles that both constrain opportunities for offending and enhance the value of a more prosocial stance (Mulvey et al, 2004). Longitudinal twin studies report genetic continuity in general antisocial phenotypes from late childhood to early adulthood, along with new genetic (and environmental) influences in adolescence (Wichers, Gardner, Maes, Lichtenstein, Larsson, & Kendler, K.S. (2013)). Byrd, Loeber and Pardini (2012) found that elevated levels of conduct symptoms and interpersonal callousness predicted delinquency that persisted to adulthood, while Stouthamer-Loeber, Wei, Loeber & Masten (2004) found that desistance from serious delinquency was most likely for young men with good job skills, positive earlier relationships with peers, awareness of the risks of offending, and active engagement in either education or employment. Among adjudicated offenders Sweeten, Piquero and Steinberg (2013) found changes in antisocial peer affiliations among the strongest correlates of changing involvement in crime, while Laub and Sampson (2003) highlighted the role of adult 'turning point' experiences, including social attachments to work, and supportive marital relationships, in promoting desistance from crime. Antisocial young people may vary in the extent to which they have access to, or can benefit from, later positive experiences of this kind; indeed, a variety of evidence now highlights the extent to which the cumulating consequences of early disruptive behaviours function to select antisocial young people into risk-prone environments and limit opportunities for involvement in more positive relationships and roles. As a result, avoiding 'snares' of this kind may be among the key pathways to optimizing outcomes in the antisocial domain.

Summary: Disruptive and Antisocial Disorders

A small proportion of children with disruptive behaviour problems go on to show severe antisocial difficulties well into adult life. For the majority, however, the expectable course seems to be one of recovery, at least from overt antisocial difficulties, either during childhood or the early adult years. The consequences of early antisocial behaviours for increased risk of other psychiatric disorders, poor social functioning and compromised physical health (often compounded by less than optimal social contexts) may be less easy to leave behind. A range of different interventions have proved effective in the short term, but may require boosters/continuing reinforcement to support longer-term benefits. Maturational factors, attachment to adult social bonds and avoidance of the adverse downstream

consequences of early disruptiveness are among the best known correlates of more positive later outcomes.

Substance Use Disorders

Does the outcome of SUDs include increased risk of the same diagnosis in the future?

There is a large literature on SUDs across the transition to adulthood (Stone, Becker, Huber, & Catalano, 2012), but a lot of it relies on cross-sectional surveys. This makes it hard to tell whether the picture is one of persistence or of different individuals with episodes at different ages. Data from the Monitoring The Future study, a series of surveys of high-school students, indicate that problem levels of drug and alcohol use, including daily use, binge drinking, and daily drunkenness, are highest during young adulthood (Johnston, O'Malley, Bachman, & Schulenberg, 2012), but leave open the question of how many of these young adults with SUD are the same young people as were abusing substances as adolescents. The Dunedin longitudinal study (Meier et al., 2013), looking at alcohol dependence up to age 32, found that most cases were limited to the period between 18 and 21: of 216 cases of alcohol dependence at age 18–21, 132 had recovered by age 26 and a further 50 by age 32, leaving only 34 persistent cases. Thus, 84% of adolescents with alcohol dependence were recovered by adulthood. The National Comorbidity Study Replication, using adults' retrospective lifetime recall, found that only 26% of adults with a lifetime SUD diagnosis had one during the 12 months preceding the interview (Kessler, Berglund, et al., 2005; Kessler, Chiu, et al., 2005). Conversely, the large majority of those with an adolescent SUD did not have one as an adult. In a 2-year follow-up of a Hispanic adolescent sample in Chicago (Reardon & Buka, 2002) the onset rate (new cases) between 15 and 17 was 13.7% for alcohol abuse or dependence, and for marijuana 9.5%. Between 18 and 20, onset rates were lower (8.3%, 3.6%), but persistence much more common (38.4%, 36.5%). Thus half to two-thirds of those with SUD at 15/17 were drug free at 17/20. In a longitudinal study of Canadian youth (Vida et al., 2009) most participants had fewer days of heavy drinking at 25 than at 19, and only one group – heavy drinkers with conduct disorder at 19 – drank significantly more than the No Diagnosis cluster. In the case of illicit drug use (mainly cannabis) it was the youth with both drug abuse and depression who continued to have higher rates of frequent drug use than the No Diagnosis cluster. However, it is notable that 40% of this group were not using illicit drugs at age 25.

The consensus of both prospective and retrospective community and clinical studies seems to be that adolescent SUD leaves a significant, but not very large, risk of adult SUD above the general population level, with odds ratios of around 1.3 (McLaughlin et al., 2010; Copeland et al., 2009).

Does the outcome of SUDs include increased risk of a different diagnosis in the future?

Some prospective studies have found that adolescent alcohol use disorder (AUD) predicts heightened risk of adult depression and anxiety disorders (Fröjd, Ranta, Kaltiala-Heino, & Marttunen, 2011; Harrington et al., 1990). Adolescent cannabis and tobacco use have been shown to predict adult depression (Georgiades & Boyle, 2007). In a prospective study of Australian adolescents through age 29, weekly use in adolescence predicted anxiety

disorders at 29, but the association disappeared if adjusted for adolescent anxiety or depression (Degenhardt et al., 2013).

Some studies allow us to examine the timing of onsets of different diagnoses among co-occurring cases. In the OADS, depression preceded AUD in 57% of cases, AUD preceded depression in 41%, and the 2 were concurrent in 2% (Briere, Rohde, Seeley, Klein, Lewinsohn, 2014). In the NESARC follow-up (Flórez-Salamanca, Secades-Villa, Budney, García-Rodríguez, Wang, & Blanco, 2013), remitted cannabis abusers were more likely to relapse by young adulthood if they developed conduct disorder or depression after the adolescent interview. In the same study, onset of prescription opioid addiction at Wave 2 was predicted by other mental disorders (i.e., mood, personality disorders and substance use disorders).

However, some studies show a protective effect of psychiatric disorders, especially emotional disorders, on the development of SUD. For example, a 10-year follow-up of the Munich community sample (Goodwin, Perkonigg, Höfler, & Wittchen, 2013) found that, in contrast to nicotine users with an increasing or persistent trajectory, those with decreasing use were *more* likely to have a lifetime distress disorder at baseline, in particular a depressive disorder. A pre-existing panic disorder or somatoform disorder predicted a *lower* risk of adult nicotine addiction.

Do SUDs increase risk of future poor health and functioning?

Using a nationally representative cross-sectional survey of college students from a sample of 119 public and private colleges, Wechsler, Lee, Kuo, and Lee (2000) found that frequent binge drinkers were over eight times more likely to get hurt or injured than non-binge drinkers, 17 more times more likely to have missed classes, seven times more likely to have engaged in unplanned sexual activity, and 8 times more likely to have gotten into trouble with campus or local police (Wechsler et al., 2000). In the Christchurch study, adolescent alcohol misuse predicted adult breakdown in relationships (Boden, Fergusson & Horwood, 2013). A Canadian study that followed adolescents from 19 to age 25 (Vida et al., 2009) found that while the general level of functioning improved or remained high across the sample, that of the adolescent drug abusers was consistently lower. The adolescent drug abusers were less likely than those with no adolescent diagnoses to have completed high school, and more likely to have children and to have been arrested. The Ontario Child Health Study also found that tobacco use in adolescence and continued use in adulthood was associated with increased risk for poorer functioning across multiple domains, including physical health and life satisfaction, personal income, and years of education (Georgiades & Boyle, 2007). Gambling problems also predicted an increased 3-year incidence of nicotine dependence in women and alcohol dependence in men (Pilver, Libby, Hoff, & Potenza, 2013). Cannabis use in adolescence, and continued use in adulthood, was associated with fewer adverse outcomes than tobacco, and the magnitude of the effects was weaker (Georgiades & Boyle, 2007). In the Dunedin study (Meier et al., 2012) persistent cannabis use was associated with neuropsychological decline broadly across domains of functioning, even after controlling for years of education. Informants also reported noticing more cognitive problems for persistent cannabis users. Impairment was concentrated among

adolescent-onset cannabis users, with more persistent use associated with greater decline. Further, cessation of cannabis use did not fully restore neuropsychological functioning.

In the GSMS adolescent SUD increased risk of poor health and functioning in young adulthood relative to those with no adolescent psychiatric disorder, but not as powerfully as did anxiety (Copeland et al., 2014) or depression (Copeland, unpublished).

What treatments or preventive programs for SUDs work?

Of 45 randomized controlled trials of treatments for alcohol problems reviewed by Healey et al., only 7 included subjects under 18 (Healey, Rahman, Faizal, & Kinderman, 2013). The review found evidence that treatment is superior to no treatment, but there was insufficient evidence to compare the effectiveness of treatment types. The exception to this is that outpatient family therapy appears superior to other forms of outpatient treatment. A meta-analysis (Tanner-Smith, Wilson, & Lipsey, 2012) of the effectiveness of different types of outpatient psychotherapy for adolescent SUD showed effect sizes of 0.2(0.0 – 0.5) to 0.6 (0.3 – 1.0) (small to good), with family and group psychotherapy the most effective. However, we should note that the effect size for no treatment was 0.4 (0.3 – 0.6). Only for family therapy was the effect size significantly better than the no treatment effect size.

Pharmacotherapies for treatment of addiction to alcohol (e.g, disulfiram, naltrexone, acamprosate), opiates (eg, methadone and buprenorphine), and nicotine (bupropion, nicotine replacement) may or may not be appropriate for adolescents. In the absence of empirical evidence documenting efficacy of pharmacological treatments for adolescents, caution is warranted even in use of treatments for which evidence supports use in adults (Stevens, Verdejo-García, Goudriaan, Roeyers, Dom, & Vanderplasschen, 2014).

One review concluded that family-based therapy is ‘not only a viable treatment alternative for the treatment of drug abuse, but [is] now consistently recognized among the most effective approaches for treating both adults and adolescents with drug problems’ (Rowe, 2012). However, the nationally-representative NESARC study found that across the lifetime only a third (37.9%) of adults reporting drug dependence, and only 8.1% of those reporting abuse, had ever been treated or even sought treatment (Compton, Thomas, Stinson, & Grant, 2007). This means that optimal outcomes that are dependent on treatment are still a long way from being realistically achievable.

What factors predict optimal outcome after SUD?

Only a few studies tell us anything about predictors of good outcomes. In a 9th-grade school sample from Australia (Victoria) followed into early adulthood (Toumbourou, Evans-Whipp, Smith, Hemphill, Herrenkohl & Catalano, 2014), AUDs were less common in those who were involved in religious groups, had completed high school, or were in an intimate relationship, and more likely in those with adolescent alcohol use, or who were living with friends, or in smaller towns or rural areas. Variables most consistently related to successful outcome of treatment are treatment completion, low pre- treatment substance use, and peer/parent social support/nonuse of substances (Williams & Chang, 2000).

On the negative side, there is a great deal of evidence that parental drug use increases adolescent risk (Chassin, Flora, & King, 2004), and probably adult risk too (Chassin et al., 2004; Toumbourou et al, 2014). In the second wave of the NESARC study, conduct disorder or a major depressive disorder in the interval after Wave 1 increased the risk of cannabis use disorder relapse by wave 2 (Flórez-Salamanca et al., 2013). Conduct disorder was also found to predict adolescent SUD in GSMS (Sung, Erkanli, Angold, & Costello, 2004), but a more detailed longitudinal analysis showed that this association was restricted to early adolescence; by age 15–16, when drinking became the norm, CD no longer increased the likelihood of SUD (Sung, Erkanli and Costello, 2014).

Data from NESARC showed that individuals with alcohol or nicotine dependence who also had a history of abuse or physical neglect were more likely to have a chronic, unremitting course of dependence (Florez-Salamanca et al., 2013). In the Ontario study (Vida et al., 2009), youth with a substance abuse disorder at age 19 were less likely to have graduated from high school, and between ages 19 and 25 were more likely to have married, to have children, to have a job (rather than being in higher education), and to have been arrested. Persistent alcohol use after adolescence in the Dunedin longitudinal study was predicted by an inability to reduce drinking and by continued use despite problems by age 18 (Meier et al., 2013). This was in contrast to those who began to have alcohol problems for the first time as adults, who were almost indistinguishable from other cohort members as children or adolescents.

Summary: SUD

Most of the evidence provided here comes from a very limited range of studies, and most of these in turn focus on predictors of bad rather than optimal outcomes. The consensus seems to be that many, perhaps three out of four, adolescent substance abusers grow up to perform fairly normally as adults, although their risk of harmful outcomes is certainly increased compared with those with no SUD history. High school completion and supportive psychosocial environments are among the few currently known predictors of more positive outcomes; while some treatment studies have shown an effect, so few young addicts receive treatment that treatment effectiveness at the population level is unknown.

Discussion

> Since spontaneous recovery is frequent, it is to be hoped that careful study of those circumstances surrounding recovery might suggest strategies of intervention with the 40%–50% who do not recover spontaneously
>
> Robins (1978, p 620)

We set out to review longitudinal findings on outcomes of child and adolescent anxiety disorders, depression, ADHD, conduct disorder, and substance use disorders, to answer the questions: what is the best expectable adult outcome of these disorders? What is the most likely adult outcome? Does treatment in childhood increase the chance of an optimal outcome? And what have follow-up studies taught us about other factors associated with recovery?

The first conclusion from the longitudinal epidemiologic findings we reviewed is remarkably similar to Lee Robins's conclusion more than 30 years ago: around half of all children with one of these diagnoses will be free of their initial childhood difficulties by early adulthood, and many may be within the normal range in the areas of health, educational and career achievements, social functioning and avoidance of criminal or dangerous behavior. Here, however, we must enter one important caveat. Most reports from follow-up studies (including those we have contributed to ourselves) have focused on indicators of *problem functioning* later in development rather than on *adult competencies* (Arnett, 2000), and have detailed them separately for different outcome domains. As a result it is often difficult to know how far, for example, the majority of adverse outcomes are concentrated in the same sub-set of childhood sufferers, or what proportion of children with a disorder have indeed reached a level that could be regarded as an 'optimal outcome', whatever one's definition of that slippery term. Increasingly, longitudinal studies are including indicators of positive competencies and adaptive skills in their follow-up assessments, along with measures of participants' own subjective evaluations of their well-being and satisfaction in life. We look forward with interest to the light these very different types of assessments will cast on our views of the outcomes of childhood disorders, and the new questions they will inevitably prompt.

Our second conclusion is that most types of child psychiatric disorders lower a young person's chances of a normal life as an adult. The likelihood of a recurrence of the same disorder, or the onset of a different one, results in an adult prevalence rate higher than that seen in adults with no psychiatric history. In addition, evidence is beginning to accumulate that even in those with no young-adult diagnosis, a disorder in childhood or adolescence increases the risk of poor 'real world' outcomes in the areas of educational and work achievement, troubles with the law, social isolation and suicidality, and physical frailty and ill-health.

Third, this review of the literature on adult outcomes raises the question of the distinction between 'what is the *average* optimal outcome of a given diagnosis?' and 'what are *this individual's* chances of achieving an optimal outcome?' Is every child with early psychopathology likely to score lower on a hypothetical scale of optimality than every child with no such history, or do some children with a psychiatric history grow up indistinguishable from their peers, while others are seriously disabled? We do not yet know the answers to these questions, and there is a serious lack of data to answer them. For clinicians and policy-makers alike, however, it is clearly important to know whether we should reduce expectations for nearly all young adults with a history of mental illness, or pay attention to identifying the few who are particularly vulnerable to persisting difficulties.

Fourth, it is almost impossible to draw any general conclusions from this literature on the personal or environmental characteristics that help to support optimal outcomes into adulthood. Personal characteristics the literature singles out as helpful include a positive emotional style (depression), impulse control and moral reasoning (conduct disorder), a higher than average IQ (ADHD) and completing high school (SUD). Supportive home and school environments seem to encourage optimal outcomes after a range of disorders. But it is still far from clear how far optimal outcomes are possible if the disorder takes an

untreated course, and how far they are the result of strategies to manage the disorder, to avoid its negative downstream consequences (which, as we have seen, often include selection into environments that perpetuate exposure to risk), or to compensate for ensuing functional disabilities. Knowledge of the processes that contribute to more positive outcomes is yet more limited. Our findings suggest (as Lee Robins also noted many years ago) that a focus on the mechanisms underlying 'positive discontinuity' of this kind may offer rich rewards.

Our final conclusion is that there is a need for further research on optimal outcomes both within specific diagnostic groups and across child psychopathology as a whole. The shape of the prevalence curve varies across diagnoses, but we do not know much about the reasons for this, although it could surely give us hints about aetiology as well as likely course. On the other hand this review points to some strong similarities across diagnoses, especially in the areas of adult deficits that seem to be common to most or all, and also possibly in the qualities that encourage resilience.

Why are our conclusions more optimistic than those of DSM-5, reviewed in Table 1? An obvious reason is that DSM-5 is largely the work of clinicians who see treatment-seeking patients. This group may have more severe symptoms, and are likely to be comorbid (Costello & Janiszewski, 1990). DSM-5 repeatedly warns of worse outcomes in untreated cases (e.g., agoraphobia, social anxiety disorder), and more than half of the children with psychiatric disorders identified in epidemiologic studies have received no specialty mental health care (Burns et al., 1995; Burns, Costello, Erkanli, Tweed, Farmer, & Angold, 1997; Merikangas, He, Brody, Fisher, Bourdon & Koretz, 2010; Merikangas et al., 2011). This means that clinicians may well have a less hopeful view of optimal outcomes than do epidemiologists.

The reader may have noticed that, in the face of our training in developmental psychopathology and of a literature that concentrates so fiercely on the negative sequelae of childhood difficulties, we have found it hard to stay focused on 'optimal outcomes'. We hope that research on what society can do to encourage good outcomes will continue to emerge and expand.

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Key Points

- The concept of optimal outcomes has been extended from autism spectrum disorder to outcomes of other disorders of childhood and adolescence (depression, anxiety, ADHD, conduct disorder, opposition defiant disorder, or substance use disorder).
- About half of young adults who had one or more of these disorders in childhood or adolescence will have no diagnosis in their 20s or early 30s.
- Even if these individuals have no adult disorder, they are more likely than those with no early diagnosis to be in the most impaired groups on measures of adult health, education and income, social and family functioning, and avoidance of crime or risk-taking.
- The range of personal qualities and environmental supports early in life that promote optimal outcomes in adulthood vary across diagnoses.

Table 1

DSM-5 statements about the course of disorders covered in this review*.

Diagnostic group	Onset and duration
Conduct disorder	'Remits by adulthood' in the majority (p.473)
ADHD	'difficulties with restlessness, inattention, poor planning, and impulsivity persist. A substantial proportion of children with ADHD remain relatively impaired into adulthood' (p.62).
Major depression	Although course is described as varying from person to person, 'the course…within individuals does not generally change with aging' (p.166).
Dysthymia	'by definition, a chronic course' (p.170).
GAD	'Rates of full remission are very low' (p.223)
Panic disorder	'Rates of full remission are very low' (p.223)
Separation anxiety disorder	Rarely persists into adulthood (p.192)
Social Anxiety Disorder	Remits over time, although the course may last for several years in about 60% of untreated sufferers (p. 205).
Specific phobias	'Likely to wax and wane' but those that persist into adulthood are unlikely to remit (p.200).
Agoraphobia	Rarely has onset before adolescence, but complete remission occurs in fewer than 10% of untreated cases (p.219).
Alcohol Use Disorder	'Erroneously perceived as an intractable condition' (p.493). Marked fall in cases by mid 20s
Cannabis Use Disorder	Adolescent onset, Marked fall in cases by mid 20s
Tobacco Use Disorder	Initiation rare after 21. More than half succeed in stopping, usually after age 30 (p.573)
Stimulant Use Disorder (Cocaine, Amphetamines)	Initiation occurs from teens onward; heaviest use in 30s (p.565)
Opioid Use Disorder	Often begins in early 20s and continues many years, but 20%–30% achieve abstinence especially after 40 (p.543).

* Page numbers refer to APA, 2013 (DSM-5)