

BJPpsych

The British Journal of Psychiatry

Youth mental health: appropriate service response to emerging evidence

Edited by Swaran P. Singh and Max Birchwood

Editorials

- s1 Mental health services for young people: matching the service to the need**
M. Birchwood and S. P. Singh
- s3 Prevention, innovation and implementation science in mental health: the next wave of reform**
Patrick McGorry

Papers

- s18 Preventing depression and promoting resilience: feasibility study of a school-based cognitive-behavioural intervention**
Paul Stallard and Rhiannon Buck
- s36 Transfers and transitions between child and adult mental health services**
Moli Paul, Tamsin Ford, Tami Kramer, Zobia Islam, Kath Harley and Swaran P. Singh

Special articles

- s5 Adult mental health disorders and their age at onset**
P. B. Jones
- s11 Clinical staging in severe mental disorder: evidence from neurocognition and neuroimaging**
Ashleigh Lin, Renate L. E. P. Reniers and Stephen J. Wood
- s24 Prevention and early intervention for borderline personality disorder: current status and recent evidence**
Andrew M. Chanen and Louise McCutcheon
- s30 Designing youth mental health services for the 21st century: examples from Australia, Ireland and the UK**
Patrick McGorry, Tony Bates and Max Birchwood
- s41 The divide between child and adult mental health services: points for debate**
Clare Lamb and Margaret Murphy

BJPsych

The British Journal of Psychiatry

ISSN 0007-1250

We gratefully acknowledge Birmingham and Solihull Mental Health Trust, Research and Innovation Department and the Birmingham and Black Country Collaboration for Leadership in Applied Healthcare Research and Care (CLAHRC) for their support for the National Youth Mental Health Conference on which this supplement is based.

Youth mental health: appropriate service response to emerging evidence

Edited by Swaran P. Singh and Max Birchwood

<http://bjp.rcpsych.org>

Subscriptions

Non-members of the College should contact the Publications Subscription Department, Maney Publishing, Suite 1C, Joseph's Well, Hanover Walk, Leeds LS3 1AB, UK (tel. 0113 386 8168; fax. 0113 386 8178; email: subscriptions@maney.co.uk). For subscriptions in North America please contact: Maney Publishing (USA) Inc., Two Penn Center, Suite 200, Philadelphia, PA 19102, USA (tel. 215 854 6402; fax. 215 569 0216; email: northamerica@maneypublishing.com). Annual subscription rates for 2013 (12 issues post free) are as follows:

	INSTITUTIONS	INDIVIDUALS
Europe (& UK)	£349	£287
US & Canada	\$649	\$457
Elsewhere	£407	£318

Full airmail is
£38/US\$66 extra.
Single copies of the Journal are £26, \$45
(post free).

Subscription to the printed Journal includes unlimited free access to the online Journal

Online-only subscription rates: individuals £186 (US\$297); institutions £287 (US\$458)

British Psychiatry Online (3-journal package)

Full access to the *British Journal of Psychiatry*, *The Psychiatrist* and *Advances in Psychiatric Treatment*, online-only subscription rates: individuals £306 (US\$548); institutions £365 (US\$657). View the journals at <http://www.rcpsych.org>

Queries from College members about missing or faulty copies should be addressed to the Membership Office, The Royal College of Psychiatrists, 17 Belgrave Square, London SW1X 8PG, UK.

Payment should be made out to the British Journal of Psychiatry.

Back issues

Back issues may be purchased from Maney Publishing, Customer Service, Suite 1C, Joseph's Well, Hanover Walk, Leeds LS3 1AB, UK. Tel. +44(0)113 386 8168; Fax +44 (0)113 386 8178; email: subscriptions@maney.co.uk.

Advertising

Correspondence and copy should be addressed to Stephen H. P. Mell, Advertising Manager, PTM Publishers Ltd, Pandora House, 41-45 Lind Road, Sutton, Surrey SM1 4PP, UK (tel. 020 8642 0162; fax 020 8661 5879).

US Mailing Information

The *British Journal of Psychiatry* USPS 536-870 is published monthly by the Royal College of Psychiatrists, 17 Belgrave Square, London SW1X 8PG, UK. The 2012 US annual subscription price is 607USD. Airfreight and mailing in the USA by Agent named Air Business, c/o Worldnet Shipping USA Inc., 156-15 146th Avenue, 2nd Floor, Jamaica, NY 11434, USA. Periodicals postage paid at Jamaica, NY 11431.

US POSTMASTER: Send address changes to Royal College of Psychiatrists, c/o Worldnet Shipping USA Inc., 156-15 146th Avenue, 2nd Floor, Jamaica, NY 11434, USA. Subscription records are maintained at Royal College of Psychiatrists, 17 Belgrave Square, London SW1X 8PG, UK.

TMThe paper used in this publication meets the minimum requirements of the American National Standard for Information Sciences – Permanence of Paper for Printed Library Materials, ANSI Z39.48-1984.

Typeset by Tavistock Digital Data, Tavistock.

Printed in Great Britain by Henry Ling Limited, The Dorset Press, 23 High East Street, Dorchester, Dorset DT1 1HD.

Founded by J. C. Bucknill in 1853 as the *Asylum Journal* and known as the *Journal of Mental Science* from 1858 to 1963.

©2013 The Royal College of Psychiatrists. Unless so stated, material in the *British Journal of Psychiatry* does not necessarily reflect the views of the Editor or the Royal College of Psychiatrists. The publishers are not responsible for any error of omission or fact.

Instructions to authors

Full instructions to authors are given at <http://bjp.rcpsych.org/misc/fora.xhtml>.

Information about the College's publications is available at <http://www.rcpsych.ac.uk>. The *British Journal of Psychiatry* is published monthly by the Royal College of Psychiatrists (a charity registered in England and Wales (228636) and in Scotland (SC038369). The *BJP* publishes original work in all fields of psychiatry. Manuscripts for publication should be submitted online at <http://submit-bjp.rcpsych.org>.

All papers in this journal are peer-reviewed. No person is permitted to take any role in the peer-review of a paper in which they have an interest, defined as follows: fees or grants from, employment by, consultancy for, shared ownership in, or any close relationship with, an organisation whose interests, financial or otherwise, may be affected by the publication of the paper.

Contributors

Tony Bates, Headstrong – The National Centre for Youth Mental Health, Dublin, Ireland

Max Birchwood, School of Psychology, University of Birmingham, and YouthSpace, Birmingham and Solihull Mental Health Foundation Trust, Birmingham

Rhiannon Buck, Department for Health, University of Bath, Bath, UK

Andrew M. Chanen, Orygen Youth Health Research Centre, Centre for Youth Mental Health, University of Melbourne, and Orygen Youth Health Clinical Program, Northwestern Mental Health, Melbourne, Australia

Tamsin Ford, Peninsula Medical School, University of Exeter, Exeter, UK

Kath Harley, South London and Maudsley National Health Service Foundation Trust, London, UK

Zoebia Islam, University of Warwick, Warwick Medical School, Coventry, UK

P. B. Jones, Department of Psychiatry, University of Cambridge, CAMEO Early Intervention Services, Cambridgeshire and Peterborough Foundation National Health Service Trust, Herchel Smith Building for Brain and Mind Science, Cambridge CB2 0SX, UK

Tami Kramer, Imperial College London, St Mary's Campus, London

Clare Lamb, North Wales Adolescent Service, Conwy, UK

Ashleigh Lin, School of Psychology, University of Birmingham, UK

Louise McCutcheon, Orygen Youth Health Research Centre, Centre for Youth Mental Health, University of Melbourne, and Orygen Youth Health Clinical Program, Northwestern Mental Health, Melbourne, Australia

Patrick McGorry, Orygen Youth Health Research Centre, University of Melbourne, Locked Bag 10, Parkville, Melbourne, VIC 3052, Australia

Margaret Murphy, Child and Adolescent Mental Health Service, Brookside Clinic, Cambridge, UK

Moli Paul, University of Warwick, Warwick Medical School, Coventry, UK

Renate L. E. P. Reniers, School of Psychology, University of Birmingham, UK

Swaran P. Singh, Division of Mental Health and Wellbeing, Warwick Medical School, University of Warwick, Coventry, and YouthSpace, Birmingham and Solihull Mental Health Foundation Trust, Birmingham, UK

Paul Stallard, Child and Adolescent Mental Health Research Group, Department for Health, University of Bath, Bath, UK

Stephen J. Wood, School of Psychology, University of Birmingham, UK, Melbourne Neuropsychiatry Centre, Department of Psychiatry, University of Melbourne and Melbourne Health, Melbourne, Australia

Editorial

Mental health services for young people:
matching the service to the need

Max Birchwood and Swaran P. Singh

**Summary**

It is now known that the onset of severe and recurring mental health problems begins for the most part before the age of 25: this provides a clear focus for preventive strategies and public mental health that are a feature of many health policy frameworks. The present distinction between child and adolescent mental health services and adult services at 16 or 18 does not fit easily with these data and the now well-documented problems of transition suggest that a fundamental review of services for young people is

overdue. This supplement provides an overview of the epidemiological, conceptual and service structures for young people with emergent and existing mental health problems, and asks the question, 'How should we design services for young people to promote prevention and service engagement, and to improve outcomes?'

Declaration of interest

None.

Max Birchwood (right) is Clinical Director of Youthspace, Birmingham and Solihull Mental Health Foundation Trust, and Professor of Youth Mental Health, University of Birmingham. Swaran Singh (left) is Head of Division, Mental Health and Wellbeing, Warwick Medical School, and Honorary Consultant Psychiatrist, Birmingham and Solihull Mental Health Foundation Trust.

'The children now love luxury; they have bad manners, contempt for authority; they show disrespect for elders and love chatter in place of exercise. Children are now tyrants, not the servants of their households. They no longer rise when elders enter the room. They contradict their parents, chatter before company, gobble up dainties at the table, cross their legs, and tyrannize their teachers. You would agree with me? Yes.'

(Attributed to Socrates, probably from *Plato's Republic*, book 4)

This famous (possibly apocryphal) quotation tells us how adults have always found it difficult to understand the young, even though they have themselves not long since been members of the breed. Adolescence is a turbulent period of flux with a complex interplay between biological, psychological and social changes as young people navigate their way to adulthood, embark on career pathways, assume adult roles and responsibilities and develop sexual relationships. Parent–child conflict can develop and intensify as adolescents struggle for independence while still requiring support. In this supplement we learn of the paradox that although adolescence and early adulthood are a period of maximum physical health,¹ it is a period with a steep rise in mental health problems.² Prospective studies suggest that more than half of young people by the age of 21 years will have experienced one or more psychiatric disorders,^{3,4} with many starting in childhood. Although society is concerned with the healthcare needs of an ageing population, we also have overwhelming evidence that the foundation of lifelong mental health begins in childhood, particularly in adolescence – a time when emerging mental health problems are underrecognised and certainly undertreated.¹ Recent large epidemiological studies have demonstrated that over three-quarters of serious mental health problems begin before the age of 25 years, with those that start later being delayed presentations or secondary conditions.^{2,5}

In this supplement Lin *et al* and McGorry argue that many of these adolescent disorders are rather undifferentiated, poly-symptomatic presentations that are capable of progressing to more traditional differentiated types with the passage of time and ongoing biosocial process;^{1,6} they call it the 'staging' model, taken from staging familiar in oncology. Lin *et al* outline the concept

and the supporting evidence,⁶ and argue that this provides a framework for the study of developmental psychopathology and – crucially – for prevention and public health, currently high on the UK policy agenda.⁷

Current evidence strongly suggests that mental health problems presenting in adolescence increase the risk of disorder occurring in adulthood,² hence providing a conceptual basis for early intervention, including a focus on high-risk groups. Stallard & Buck and Chanen & McCutcheon present two examples: Stallard & Buck describe a successful pilot investigation to prevent depression in adolescents through a school-based intervention focusing on those at risk,⁸ and Chanen & McCutcheon report on an early intervention approach for emerging borderline personality disorder in adolescents.⁹ This work builds on existing work developing early intervention in relation to eating disorders and the psychoses.¹

Matching service to need

How best should we provide mental health support to our young people? For many years the international care model has distinguished the child and adolescent mental health service (CAMHS) pathway for those aged up to 18 years (or 16 years in some settings) from adult mental health services (AMHS). The developmental dimension described above broadly supports such a distinction, particularly if opportunities for prevention are realised. Singh *et al*, however, reported that the transition from CAMHS to AMHS is problematic for many adolescents, with a large proportion dropping through a care gap between the two services and losing much-needed continuity of care.¹⁰ Adolescents with a serious mental illness such as psychosis or bipolar affective disorder under CAMHS care do get referred to adult care, especially if in receipt of medication or admitted to hospital. However, young people with conditions such as attention-deficit hyperactivity disorder (ADHD), autism spectrum disorders, mild intellectual disability, emotional and neurotic disorders and emerging personality disorder are either not referred to adult care or if referred are not accepted. Those who do make the journey across services feel unprepared for the transition and the abrupt cultural shift from a child-centred developmental approach to the adult care model. It is perhaps for this reason that many disengage from adult services. For the majority, transition is poorly planned, poorly executed and poorly experienced. Singh *et al* reported that many felt overburdened and others felt

abandoned by services.^{10,11} Clearly this lamentable state of affairs needs to be corrected.

The question then arises whether the problems with the CAMHS–AMHS distinction at age 16 or 18 years can be remedied, or whether we should consider it as fundamentally flawed and a structural impediment to care and treatment. Jones describes a steep rise in age incidence at this time,² and McGorry *et al* argue therefore that ‘the current system is weakest where it needs to be strongest’.¹ McGorry *et al* challenge us to consider whether, if we were to design services now, we would propose the present structure or argue instead that a care pathway from age 12 years to 25 years best fits epidemiological data and clinical need.¹² According to McGorry *et al* this would fit with international definitions of youth, and incidentally in the UK would align with local authority definitions.¹²

Lamb & Murphy present a considered analysis of the current position and options from a CAMHS perspective.¹³ They argue that separate commissioning frameworks for CAMHS and AMHS potentiate discontinuities and are inimical to good care and effective use of resources. They raise critical questions about the future structure of services for young people and consider a number of potential options for service redesign. McGorry *et al* describe alternative service models from the different settings of Australia, Ireland and England.¹² Neither the status quo nor these alternative models have clear evidence of efficacy; McGorry *et al* argue that the issue here is to agree on the criteria that need to be followed in designing such services, for example that they are aligned to evidence on epidemiology and age at onset and meet opportunities for prevention.¹² These two papers, by Lamb & Murphy and McGorry *et al*, together lay out the critical issues in reforming mental health services for our young people.^{12,13}

Concluding remarks

In the UK a zeitgeist has emerged in government policy encouraging more systematic attention to public mental health and prevention,⁷ one that the Royal College of Psychiatrists has strongly endorsed.¹⁴ A consistent theme of the papers in this supplement is that we can realise this aspiration by a fundamental review and reform of mental health services for young people so as to give them (and us) the best opportunity to prevent lifelong recurrence. We hope that this supplement will trigger a much-needed debate about the future of services for our young people so that, unlike Socrates, we will no longer look upon them as a lost cause.

Funding

M.B. was part-funded by the National Institute for Health Research through the Collaborations for Leadership in Applied Health Research and Care for Birmingham and the Black Country.

Acknowledgements

We thank the Collaborations for Leadership in Applied Health Research and Care for Birmingham and the Black Country for their support of the Youth Mental Health Conference, Birmingham, 2010, on which this supplement is based.

Max Birchwood, DSc, School of Psychology, University of Birmingham, and YouthSpace, Birmingham and Solihull Mental Health Foundation Trust, Birmingham; **Swaran P. Singh**, MD, Division of Mental Health and Wellbeing, Warwick Medical School, University of Warwick, Coventry, and YouthSpace, Birmingham and Solihull Mental Health Foundation Trust, Birmingham, UK

Correspondence: Professor Swaran P. Singh, Division of Mental Health and Wellbeing, Warwick Medical School, University of Warwick, Coventry CV4 7AL, UK. Email: s.p.singh@warwick.ac.uk

References

- McGorry P. Prevention, innovation and implementation science in mental health: the next wave of reform. *Br J Psychiatry* 2013; **202** (suppl 54): s3–4.
- Jones PB. Adult mental health disorders and age at onset. *Br J Psychiatry*, 2013; **202** (suppl 54): s5–10.
- Costello EJ, Angold A, Burns BJ, Stangl DK, Tweed DL, Erkanli A, et al. The Great Smoky Mountains Study of Youth: goals, designs, methods, and the prevalence of DSM-III-R disorders. *Arch Gen Psychiatry* 1996; **53**: 1129–36.
- Silva PA. The Dunedin multidisciplinary health and developmental study: a 15 year longitudinal study. *Paediatr Perinat Epidemiol* 1990; **4**: 96–127.
- Kessler RC, Chiu WT, Demler O, Merikangas KR, Walters EE. Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Arch Gen Psychiatry* 2005; **62**: 617–27.
- Lin A, Reniers RLEP, Wood SJ. Clinical staging in severe mental disorder: evidence from neurocognition and neuroimaging. *Br J Psychiatry* 2013; **202** (suppl 54): s11–7.
- Department of Health. *No Health Without Mental Health*. Department of Health, 2011.
- Stallard P, Buck R. Preventing depression and promoting resilience: feasibility study of a school-based cognitive-behavioural intervention. *Br J Psychiatry* 2013; **202** (suppl 54): s18–23.
- Chanen AM, McCutcheon L. Prevention and early intervention for borderline personality disorder: current status and recent evidence. *Br J Psychiatry* 2013; **202** (suppl 54): s24–9.
- Singh SP, Paul M, Ford T, Kramer T, Weaver T, McLaren S, et al. Process, outcome and experience of transition from child to adult mental healthcare: multiperspective study. *Br J Psychiatry* 2010; **197**: 305–12.
- Paul M, Ford T, Kramer T, Islam Z, Harley K, Singh SP. Transfers and transitions between child and adult mental health services. *Br J Psychiatry* 2013; **202** (suppl 54): s36–40.
- McGorry P, Bates T, Birchwood M. Designing youth mental health services for the 21st century: examples from Australia, Ireland and the UK. *Br J Psychiatry* 2013; **202** (suppl 54): s30–5.
- Lamb C, Murphy M. The divide between child and adult mental health services: points for debate. *Br J Psychiatry* 2013; **202** (suppl 54): s41–4.
- Royal College of Psychiatrists. *No Health without Public Mental Health: The Case for Action* (Position statement PS4/2010). Royal College of Psychiatrists, 2010.

Editorial

Prevention, innovation and implementation science in mental health: the next wave of reform

Patrick McGorry

**Summary**

Although the corrosive effect of mental ill health on human health and happiness has long been recognised, it is only relatively recently that mental illness has been acknowledged as one of the major threats to economic productivity worldwide. This is because the major mental disorders most commonly have their onset during adolescence and early adulthood, and therefore have a disproportionate impact on the most productive decades of life. With the costs associated with mental ill health estimated to double over the next two decades, a greater emphasis

on prevention and early intervention has become even more imperative. Although prevention largely remains aspirational for many reasons, early intervention is well within our current reach and offers the potential to significantly reduce the impact of mental ill health on our health, happiness and prosperity in the immediate future.

Declaration of interest

None.

Professor Patrick McGorry's major interest is in youth mental health, particularly early psychosis, and in designing effective healthcare services for young people with emerging mental disorders.

In a report drawing on three different forms of economic analysis, the World Economic Forum recently identified mental ill health as being at least equal to cardiovascular disease as the principal threat to gross domestic product (GDP) over the next two decades.¹ *The Lancet's* campaign for global mental health and the international Grand Challenges in Mental Health initiative on research priorities also underline the growing recognition that mental ill health is the awakening giant of health and social reform.^{2,3} Although the costs of mental ill health are otherwise set to double over the next 20 years, billions could be saved through a greater emphasis on prevention and early intervention. The key reason that mental ill health is as potently corrosive of economic growth as it is of human happiness and potential is that 'mental disorders are the chronic diseases of the young'.⁴ The distribution of such disorders within the life cycle is the mirror image of physical illness. Seventy-five per cent of mental disorders emerge before the age of 25 years, about a quarter before the age of 12 years and the rest in a steady surge of premature mortality, morbidity and comorbidity through the emerging adult years.⁵ There is evidence that the mental health of young people is worsening, prompting the evocative assertion that young people are the 'miners' canaries' of society.⁶ The rates of diagnosable mental disorders during the period of transition between childhood and adulthood can reach as high as 50%, with multiple or recurrent disorders present in more than half of cases. Neither is this to be dismissed as teenage angst or growing pains, since there are real and lasting consequences for earning potential and educational and social outcomes at age 30 years.⁷ There have been major changes in the developmental experience of the transition to adulthood in recent decades, which have some positive aspects but may also carry a hidden yet increasing cost.^{8,9} In summary, because mental disorders have their origins early in life, surge disproportionately during the stage of transition from childhood to adulthood and cast a long shadow through the decades of peak social and economic productivity,¹⁰ they now represent the most urgent threat and potentially the greatest opportunity for

prevention and control among the non-communicable diseases, a landscape otherwise arguably characterised by diminishing returns in the developed world at least.

Prevention is always better than cure, but for many reasons it remains aspirational in mental health. The feasibility of universal prevention with whole populations has been seriously questioned on the basis of power and the low malleability of risk factors.¹¹ Poverty, social and economic inequality and trauma, abuse and neglect make smoking and diet seem simple targets. Yet it may not be mission impossible. The natural experiment reported in the Great Smoky Mountains study, for example, showed that income supplementation for American Indian families reduced the prevalence of psychiatric disorders across adolescence.¹² Selective prevention whereby risk factors are targeted within high-risk subgroups is perhaps more within reach and is more researchable. One example is interventions triggered by screening for postnatal depression. When we move on to the firmer ground of indicated prevention, the spectrum begins conceptually and practically to merge with early intervention and treatment. The definition of indicated prevention allows subthreshold clinical features to be viewed as risk factors for fully fledged disorder. The identification of clinically significant (and functionally impairing) yet subthreshold disorder represents the frontier of research and service reform in mental healthcare and has challenged psychiatry to face the controversies and measure up to the standards of the rest of healthcare. But is it really 'prevention'? To label it so has misled some into believing that those involved are asymptomatic and currently 'not ill'. However, in psychiatry, a person's need for care demonstrably precedes the threshold for meeting full criteria for diagnosis, at least in terms of our current categorical diagnostic systems. Despite warnings about the medicalisation of human distress by critics appropriately concerned that diagnosis in some health systems (especially in the USA) means prescription of medication, there is a much greater risk of denying effective help and support to large numbers of people, many of whose lives will be at risk as a result of significant and sustained morbidity. Many more will lead thwarted lives, with poor mental health contributing to the erosion of their life chances. Indicated prevention, closely followed by early detection of full-threshold disorder, is theoretically the next best option after universal and selective prevention, and is

practically much more achievable, with Cuijpers suggesting ways it can be sharpened and enhanced.¹¹ We have since developed a heuristic diagnostic framework, the clinical staging model, to guide further research and reform along this frontier and either side of it.^{13,14} This model is attracting increasing support,¹⁵ although it must transcend the current diagnostic silos in terms of treatment and biomarker research.

Innovation is a vital ingredient and a pressing need if we are to shift the focus from the palliative legacy of traditional mental healthcare to a proactive effort to limit the corrosive havoc that mental disorders can wreak on the lives of those on the threshold of productive life and beyond. We have only partially relinquished the deterministic concepts of 19th-century psychiatry which continue to influence the energy as well as the topography of mental healthcare. Innovation is like an orchid, exquisitely sensitive to context and environment,¹⁶ and we need to understand the innovation cycle as it applies in other fields. Innovation involves new thinking, new models, new treatments – all of which we desperately need. Innovators and early adopters need to be nurtured as we seek progress in mental healthcare.

Even if there were to be no new treatment advance in the next 20 years, we could still substantially reduce what Andrews¹⁷ describes as the ‘avertable burden of disease’ by increasing the scale and coverage of mental healthcare and re-engineering the timing and culture of the provision of services. The related concepts of implementation science and ‘scaling up’ of innovations,¹⁸ especially of service models, are particularly relevant to this supplement. Evidence-based medicine (and its forerunner the Cochrane Collaboration) has been a valuable safeguard against ‘great and desperate cures’,¹⁹ particularly in psychiatry; however, it can also be misused to obstruct the diffusion of genuine advances. As I have argued elsewhere, Cochrane loses relevance when it is applied beyond the level of individual treatment to cover health services research.²⁰ Evidence-based healthcare, a cousin of evidence-based medicine, simply cannot be a prisoner of Cochrane. The orchid of innovation needs a range of nutrients to grow, and although evidence is certainly one of these, a genuine need for change, champions, context and new resources are others. Many flowers will germinate and flourish in a particular setting, yet few will disseminate to other fields in a systematic or franchised manner. The scaling-up literature,^{21,22} again a body of knowledge that cuts across many fields of endeavour, bears witness to the key elements that are required for success.

This supplement makes the case for a transformational reform of mental healthcare based on the principles of early intervention, and a priority focus on the developmental period of greatest need and capacity to benefit from investment: the period of emerging adulthood. This by no means argues against investments earlier or later in life, which are also essential. Heartened by the highly successful evidence-informed scaling up of early intervention in psychosis across many hundreds of locations and numerous national health systems since the mid-1990s, and the newly emergent youth mental health models of the past 5 years, a number of leaders, policy makers and service developers are working to create an international momentum to address the mental health needs of young people and their families. There are already rapidly emerging examples of these modern stigma-free cultures of care designed and operated with young people themselves, and these are described in this supplement.²³ The arguments for this type of transformational reform are resonating strongly with the community and with policy makers while attracting predictable resistance from middle management and conservative elements within professional groups. These examples of 21st-century clinical infrastructure will also facilitate some of

the population-based and universal programmes that may link with mental health awareness and promotion activities and with new internet-based technologies. If these new mind-sets and reforms spread widely we might be able to reduce the impact of mental ill health on our health, happiness and prosperity over the next two decades.

Patrick McGorry, MD, PhD, FRCP, FRANZCP, Orygen Youth Health Research Centre, University of Melbourne, Locked Bag 10, Parkville, Melbourne, VIC 3052, Australia. Email: pmcgorry@unimelb.edu.au

References

- Bloom DE, Cafiero ET, Jane-Llopis E, Abrahams-Gessel S, Bloom LR, Fathima S, et al. *The Global Economic Burden of Non-Communicable Disease*. World Economic Forum, 2011.
- Lancet Global Mental Health Group. Scale up services for mental disorders: a call for action. *Lancet* 2007; **370**: 1241–52.
- Collins PY, Patel V, Joestl SS, March D, Insel TR, Daar AS, et al. Grand challenges in global mental health. *Nature* 2011; **475**: 27–30.
- Insel TR, Fenton WS. Psychiatric epidemiology: it's not just about counting anymore. *Arch Gen Psychiatry* 2005; **62**: 590–2.
- McGorry PD, Purcell R, Goldstone S, Amminger GP. Age of onset and timing of treatment for mental and substance use disorders: implications for preventive intervention strategies and models of care. *Curr Opin Psychiatry* 2011; **24**: 301–6.
- Eckersley RM. *Never Better – Or Getting Worse? The Health and Wellbeing of Young Australians*. Australia21, 2008.
- Gibb SJ, Fergusson DM, Horwood LJ. Burden of psychiatric disorder in young adulthood and life outcomes at age 30. *Br J Psychiatry* 2010; **197**: 122–7.
- Arnett JJ. *Emerging Adulthood. The Winding Road from the Late Teens Through the Twenties*. Oxford University Press, 2004.
- Rutter M, Smith DJ. *Psychosocial Diseases in Young People: Time Trends and Their Causes*. Wiley, 1995.
- Goodman A, Joyce R, Smith JP. The long shadow cast by childhood physical and mental problems on adult life. *Proc Natl Acad Sci USA* 2011; **108**: 6032–7.
- Cuijpers P. Examining the effects of prevention programs on the incidence of new cases of mental disorders: the lack of statistical power. *Am J Psychiatry* 2003; **160**: 1385–91.
- Costello EJ, Erkanli A, Copeland W, Angold A. Association of family income supplements in adolescence with development of psychiatric and substance use disorders in adulthood among an American Indian population. *JAMA* 2010; **303**: 1954–60.
- McGorry PD. Issues for DSM-V: clinical staging: a heuristic pathway to valid nosology and safer, more effective treatment in psychiatry. *Am J Psychiatry* 2007; **164**: 859–60.
- McGorry PD, Hickie IB, Yung AR, Pantelis C, Jackson HJ. Clinical staging of psychiatric disorders: a heuristic framework for choosing earlier, safer and more effective interventions. *Aust N Z J Psychiatry* 2006; **40**: 616–22.
- Insel TR. Rethinking schizophrenia. *Nature* 2010; **468**: 187–93.
- Brooks H, Pilgrim D, Rogers A. Innovation in mental health services: what are the key components of success? *Implement Sci* 2011; **6**: 120.
- Andrews G, Issakidis C, Sanderson K, Corry J, Lapsley H. Utilising survey data to inform public policy: comparison of the cost-effectiveness of treatment of ten mental disorders. *Br J Psychiatry* 2004; **184**: 526–33.
- Tansella M, Thornicroft G. Implementation science: understanding the translation of evidence into practice. *Br J Psychiatry* 2009; **195**: 283–5.
- Valenstein E. *Great and Desperate Cures: The Rise and Decline of Psycho-surgery and Other Radical Treatments for Mental Illness*. Basic Books, 1986.
- McGorry P. At issue: Cochrane, early intervention, and mental health reform: analysis, paralysis, or evidence-informed progress? *Schizophr Bull* 2012; **38**: 221–4.
- Cooley L, Kohl R. *Scaling Up – From Vision to Large-Scale Change: A Management Approach for Practitioners*. Management Systems International, 2006.
- Bradach J. Going to scale: the challenge of replicating social programs. *Stanford Soc Innovat Rev* 2003; spring: 19–25.
- McGorry P, Bates T, Birchwood M. Designing youth mental health services for the 21st century: examples from Australia, Ireland and the UK. *Br J Psychiatry* 2013; **202** (suppl 54): s30–5.

Special article

Adult mental health disorders and their age at onset

P. B. Jones

Summary

The study of age at onset of mental health disorders is technically and conceptually difficult. It is important to consider these age distributions in order to understand causes and mechanisms of illness and to intervene at an appropriate juncture for primary and secondary prevention. This article reviews some of the approaches to studying age at onset, sets out the evidence to support the assertion that adult mental disorders begin in adolescence, and finds that perhaps half of all adult mental health disorders have begun by the teenage years. The paper then discusses whether this

fits what is known about the developmental neurobiology of the brain and introduces the implications for mental health services.

Declaration of interest

P.B.J. nominated his department to receive a fee for providing scientific advice to Roche. He receives honoraria for contributing to the biannual British Association of Psychopharmacology schizophrenia master class series.

Adult mental health disorders begin by adolescence. This seems a simple enough statement that suggests we cannot understand mental illness affecting adults unless we appreciate what goes on during the preceding period of life. If true, childhood and adolescence, divided by the biological gateway of puberty, would be crucial epochs in terms of understanding causes and mechanisms of adult illness. This would frame attempts at early intervention and both primary and secondary prevention. Following the theme of this supplement, this paper focuses on the continuities between the adolescent and early adult stages of the life course, arguing that these are better seen as a single developmental epoch. However, the seeds of many conditions manifest in this period are sown in even earlier life, such that some reference is also made to childhood.

In some conditions such as psychotic illness, intervention during incipient illness or its early stages may prevent a lifetime of disability for a proportion of those affected.¹ Onset of the full syndrome at a very young age is often associated with a severe course,² but – intriguingly – a good response to treatment.³ In other conditions such as dementia, very early onset may suggest a different disease entity and so direct management accordingly. From the perspectives of surveys, epidemiological research and health service planning, it is also essential to understand the distribution of different disorders over the life course. This underpins precise estimates of projected morbidity and appropriate policy development. For example, most people with chronic psychotic disorders in a mental health service are middle-aged, but our understanding of the onset distribution of these conditions means preventive and early intervention services must target a much earlier age group, as described below.

Defining and measuring age at onset

This life-course view means that identifying the age at onset of mental disorders is important, but it is difficult.⁴ The classic psychiatric disorders of adult life often begin with non-specific symptoms. As studies of the evolution of symptoms become more detailed, it is clear that early psychopathologic changes can be transient and dynamic before crystallising into a syndrome fitting some operational category such as the American Psychiatric Association's DSM. Only in retrospect can a decision be taken

as to whether the early features were in fact part of the eventual picture. In epidemiological terms this leads to enormous scope for error or bias in the recall or memory of events;⁵ some elements may be misattributed to illness and others simply forgotten. From a clinical point of view it is well known that the better the history, the earlier the age at onset can be dated. Furthermore, just as periods of low mood and occasional elation are part of normal life, it seems likely that some psychological experiences such as voice hallucination, hitherto considered psychopathological, are relatively common during certain developmental periods; it is their persistence or coexistence with other features that marks them as significant. This is leading to conceptual difficulties with clinical concepts such as the prodrome that are at best meaningless or confusing when used at the population level, and may be damaging if they lead to interventions that have adverse benefit-to-harm profiles quite different from the clinical situation.

Retrospective accounts of age at onset for some disorders are more useful than others, and perfect for none. Despite the problems alluded to above, it is a reasonable (and useful) assumption that a large proportion of people with psychotic illness eventually receive treatment, especially given that in the definitions of some types of psychosis such as schizophrenia, disability and loss of function are included in their definition. Thus, a survey using retrospective accounts of age at onset for schizophrenia or a broader group of non-affective psychosis yields useful information. It also avoids contamination by the data for the vast majority of young people who may experience psychotic phenomena without ever being ill. However, this is at the cost of excluding the few people with incipient illness but not yet in contact with services and, for many years, of masking researchers to the extent of psychotic phenomena as part of normal experience; for many decades epidemiologists regarded such phenomena simply as false positives. The retrospective approach is also hampered by the long duration of untreated psychosis in many with non-affective psychosis who eventually make service contact, but the alternative approach – using a community survey for incident illness – would be hugely inefficient given the relative rarity of cases and the fact that the illness itself means that affected people would be less likely to take part.⁶ It is important to recognise that the retrospective approach in clinical samples yields a self-fulfilling estimate of the age at onset of psychotic disorders

that are seen and treated in clinical services, not a broader phenotype that does not meet this threshold. That said, the method – especially when combined with population registers of treated mental disorder – can lead to definitive results.⁶

Using retrospective accounts of age at onset from cohorts of treated individuals is not useful for less disabling mental disorders because most are never treated. In some areas such as cardiovascular disease and cancer it is effective to follow up large cohorts of individuals and track these outcomes which are relatively easy to measure.⁷ This is much more difficult for common mental disorders where the true incidence and age at onset need intensive study, but has been achieved for psychotic illness using population-based birth cohort studies where follow-up is achieved either through regular interviews and surveys,⁸ or through population registers.⁹ Such studies are rare and usually problematic in some way as they were initiated for reasons other than mental health disorders. For these reasons, retrospective reports from carefully designed cross-sectional community surveys provide most evidence on age at onset for most mental health disorders.

Examples of each of these methodological approaches to age at onset are outlined below, taking different exemplar disorders. This is followed by a discussion as to whether the findings on age at onset in mental health disorders fit with what we know about brain development. One final comment about the definition of age at onset arises from modern accounts of many long-term conditions in the framework of life course models. Here, a model of cardiovascular disease in late adulthood might encompass genetic inheritance, the epigenetic modification of the genome by fetal environment and childhood nutrition, the importance of social context in the beginnings of cigarette smoking at school, followed by further genetic and behavioural aspects in the establishment of nicotine dependence and sedentary lifestyle. The example could go on but illustrates the gradual accretion of interacting components to a self-perpetuating cascade of causation. In the domain of mental health disorders the seeds of schizophrenia may be sown in early life,⁸ and early motor developmental abnormalities may betray important information as to the underlying neurobiology of psychotic symptoms in a later phase of development.¹⁰ However, one would be hard pushed to say that later motor milestones were the beginning of schizophrenia. Thus, in this article, the onset of a disorder is defined as the onset of features that form part of the disorder and that are contiguous with its first expression.

Clinical studies and clinical epidemiology

Schizophrenia

Schizophrenia is an archetype among the psychotic disorders for which retrospective accounts reconstructing the evolution of illness in treated clinical samples yield strong results; it is also a disorder with a developmental component. The same arguments also pertain to a broader spectrum of these illnesses seen in clinical practice.¹¹ The idea that schizophrenia might be developmental in origin is not new.¹² It was evident in early clinical descriptions of the syndrome,^{13,14} rediscovered as a concept towards the end of the 20th century,¹⁵ and received empirical support from epidemiological life course studies shortly afterwards.⁸ However, much of this evidence comes from the early appearance of neurobehavioural and cognitive characteristics that may betray abnormal function relevant to the incipient mental disorder, rather than the clinical entity we call schizophrenia. Large-scale, population-based studies focusing on the onset of schizophrenia itself showed that it appeared characteristically during the

post-pubertal epoch. Schizophrenia occurs in childhood but is vanishingly rare before puberty, after which its incidence increases rapidly until the mid-20s before declining over the following decades. This is best illustrated by the seminal studies of Häfner *et al* in Mannheim,^{16,17} who ascertained all incident cases of schizophrenia occurring in their catchment area and reconstructed the characteristics of their onset in minute detail (Fig. 1). The earliest signs of any mental disturbance occurred before puberty, but psychotic symptoms and index admissions for the full syndrome were confined to the decade and half after puberty. The data for women (Fig. 1(b)) indicate a continued developmental phenomenon into the fifth and sixth decades, where a second, smaller peak in incidence has been linked to changes in oestrogen levels associated with the menopause. These findings have been repeatedly replicated,¹¹ and represent some of the most robust findings in psychiatric epidemiology.

Depression and anxiety

Depression and anxiety, unlike schizophrenia, are well-known entities before puberty.¹⁸ Follow-up studies of these disorders in children and adolescents in clinical settings indicate a high degree of homotypic continuity into adult life, meaning that the same clinical syndrome is found in both earlier and later life,^{19–22} this being the case also for conduct disorders, which in addition showed a broader range of adverse outcomes (i.e. heterotypic

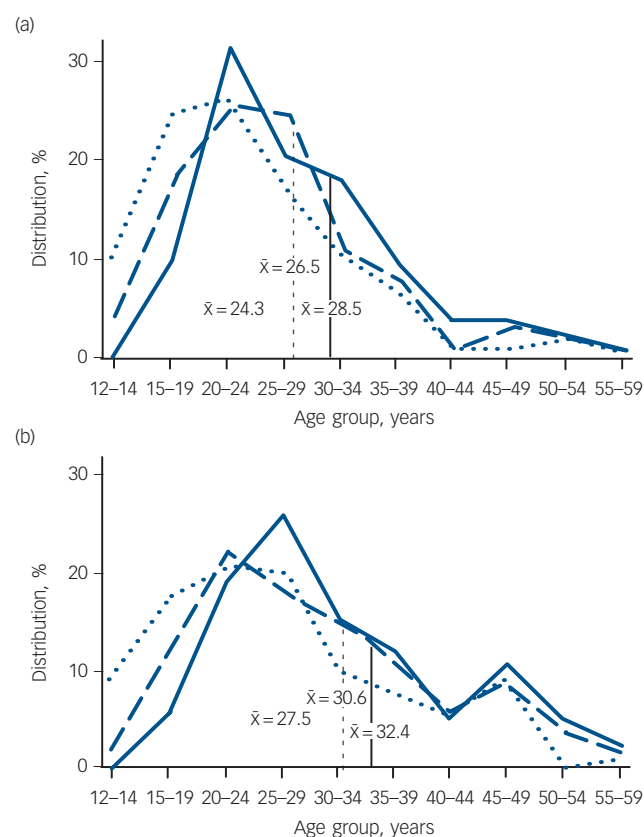


Fig. 1 Distribution of age at onset (as percentage of the whole sample, with being mean age in years) of schizophrenia (broad definition, ICD-9 codes 295, 297, 298.3, 298.4). (a) Males: earliest sign of mental disturbance, $n = 117$ (dotted line); first psychotic symptom, $n = 125$ (dashed line); index admission, $n = 133$ (solid line). (b) Females: earliest sign of mental disturbance, $n = 131$ (dotted line); first psychotic symptom, $n = 139$ (dashed line); index admission, $n = 143$ (solid line). From Häfner *et al.*^{16,17}

continuity) in addition to homotypic adult antisocial personality traits.²³ Thus, for the proportion of children and adults who went on to develop the adult syndrome, their adult mental health disorders can be said to have begun in childhood and adolescence.

Population-based approaches

Children who present to clinical services with psychiatric illness may not be at all typical of those in the general population, where even specific operational diagnoses are common at around one in ten of the population under 16 years old; few of these ever cross the threshold into clinical care. For instance, the British Child and Adolescent Mental Health Survey of 1999 surveyed 10 438 children, triangulating information from the children themselves and from their parents and teachers; around 9.5% had at least one current DSM-IV diagnosis.²⁴ Following such large samples into adult life is a considerable undertaking but has been done on several occasions in the context of birth cohorts. In these studies large samples of people defined by geography and birth date or period are followed, often for many years, simply because they represent the general population. Several birth cohorts have contributed a great deal to our knowledge about risk factors and long-term outcomes of psychiatric illness.²⁵ Here, we look in more detail at two studies: one from Britain and one from New Zealand. Both studies have been influential in elucidating the link between adult disorders and their adolescent onset.

The British 1946 birth cohort

Sometimes referred to as the grandfather of birth cohorts, the British 1946 birth cohort is a stratified, random sample of 5362 people born in one week in March 1946, first assessed 6 weeks after birth,²⁶ and regularly thereafter, irrespective of their life experiences and masked to their outcomes; these individuals continue to be followed as many contemplate the milestone of retirement and all that is brought by later middle age. Colman *et al* used ratings made by teachers when the survey members were aged 13 and 15 years (in 1959 and 1962 respectively) that included items akin to those seen in internalising disorders such as depression and anxiety.²⁷ Survey participants scoring in the top 6% of the population distribution were defined as cases of 'common mental disorder' (CMD); 277 were in this group at either age, whereas 46 met case definition at both 13 years and 15 years of age and were defined as 'persistent CMD' cases; interestingly, these observations were made before it was believed that these disorders occurred in children and well before current classifications of mental illness such as DSM and the World Health Organization's (WHO's) ICD. The remaining survey members ($n = 3002$) were classified as healthy, falling into neither group.

The presence of common mental disorder in the teenage years had a profound association with mental health in subsequent surveys of this cohort stretching out into middle life when assessments were made at ages 36, 43 and 53 years. Those in the CMD group were over two-and-a-half times more likely to be rated as having common mental disorder at age 36 years compared with the mentally healthy teenagers, and one-and-a-half times more likely to be ill at age 43 years; the effect was no longer present at age 53 years. For those with persistent CMD as teenagers the outlook was bleaker: they were over six times more likely to be ill with a common mental disorder at ages 36 and 43 years, with the effect persisting at age 53 years, when they were at fourfold risk. The ripples of episodic common mental disorder had faded by middle age but persisted after the age of 50 years for those who had experienced persistent depression and anxiety in their early teenage years; their poor mental health could be traced back to adolescence, perhaps even earlier.

The Dunedin Longitudinal study

Another example of the birth cohort approach comes from New Zealand and the Dunedin Multidisciplinary Health and Development Study.²⁸ This is based on a sample of 1037 children born between April 1972 and March 1973 in Dunedin and first assessed at age 3 years. Now in the era of operational criteria and standardised diagnostic measures, the participants were assessed at ages 11, 13 and 15 years with the Diagnostic Interview Schedule for Children, and then at ages 18, 21 and 26 years with the Diagnostic Interview Schedule;²⁹ these measures allowed DSM-IV diagnoses to be generated.³⁰ Cohort members receiving treatment and those using intensive mental healthcare were also identified.

The prevalence in the past year of any mental disorder at age 26 years was high at 48%, with men and women being equally affected. Over 80% of those with a mental health disorder at this age also had a prior diagnosis of any mental illness since the age of 11 years; overall, 74% had received a diagnosis before 18 years of age and 50% before 15 years of age. The percentage of prior diagnoses increased according to the severity of disorder by age 26 years in terms of treatment received. Of those with a disorder seeking treatment, 77% were categorised as meeting diagnostic criteria before 18 years of age and 58% before age 15 years. For those using intensive mental health services, 80% received a diagnosis before 18 years of age and 60% before age 15 years. There was a pattern of homotypic continuity, especially for conduct disorder and adult antisocial personality disorder, but there were also heterotypic associations; adult anxiety and schizophreniform disorders were preceded by a wide range of adolescent diagnoses. For all adult disorders, 25–60% of cases had a history of conduct and/or oppositional defiant disorder. The authors concluded that the majority of mental health disorders manifest at age 26 years should be considered as extensions of adolescent disorders, just as suggested by Colman *et al* for common mental disorders in the British 1946 birth cohort.²⁷ The eradication of adolescent disorders, particularly conduct disorders, would have a profound effect on reducing subsequent morbidity.

Cross-sectional surveys

These two birth cohorts give clear evidence that a considerable proportion of mental health disorders are already manifest in adolescence; these studies have many methodological advantages in their prospective approach but are limited by their periodic assessments during the life course, and the relatively small proportion of whole life that they have yet covered. A major advance in our understanding of age at onset distributions has come with developments in the methodology that can be used with cross-sectional mental health surveys. The National Comorbidity Survey Replication (NCS-R) built on a succession of major epidemiological surveys in the USA, particularly the Epidemiological Catchment Area study and the National Comorbidity Survey, and was a major part of a global initiative, the WHO World Mental Health Survey Initiative.^{29,31–34}

The NCS-R used the WHO Composite International Diagnostic Interview (WHO-CIDI),³⁵ a fully structured lay-administered interview, to generate DSM-IV diagnoses from data collected in a household survey of 9282 respondents (70.9% response rate). Relative to prior studies, methodological innovations included an expanded set of diagnoses; in-depth clinical validation of field research diagnoses based on clinician-administered Structured Clinical Interview for DSM-IV re-interviews;³⁶ dimensional self-ratings on clinically anchored scales;^{37,38} inclusion of subthreshold diagnostic syndromes; assessment of disability and impairment; the use of disease burden metrics

linked to marker physical disorders; and assessments of service use, treatment barriers and adequacy of treatment. The study was not designed for rare disorders such as schizophrenia and autism which lead to considerable non-response, but generated remarkable information on the lifetime prevalence of many conditions.

A number of methodological enhancements allowed this survey to obtain helpful estimates of age at onset, despite its retrospective method which is often associated with error.⁵ Questions were designed to avoid implausible responses by using a time-line approach,³¹ and by breaking the life course into blocks such as pre-school, high school, university and employment, so that respondents could date both important symptoms (e.g. first panic attack) and full syndromes at least to a certain epoch of their lives.³⁹ The ages by which 25%, 50% and 75% of respondents first became affected are shown for a number of syndrome groups in Table 1 together with their overall lifetime prevalence, standardised to age 75 years.

Overall, there are four headlines from the NCS-R relevant to this review. First, half (51%) of Americans will experience a DSM-IV mental health disorder during their lifetime, with some evidence that this proportion may be increasing with younger cohorts. Second, half of these disorders (as shown by the 50th percentile or median) were incident by age 14 years. The median onset tended to be earlier for anxiety disorders (age 11 years), some of which begin and end in childhood, and for impulse control disorders (age 11 years) than for substance use disorders (age 20 years) and mood disorders (age 30 years). Third, age at onset was concentrated in a narrow age range for most disorders: interquartile ranges (the number of years of age between the 25th and 75th percentiles of the age-at-onset distributions) were only 8 years (age 7–15 years) for impulse control disorders, 9 years (age 18–27 years) for substance use disorders and 15 years (age 6–21 years) for anxiety disorders, compared with 25 years (age 18–43 years) for mood disorders. Half of all mental health disorders over the life course occurred first between the ages of 7 years and 24 years.

Analysis of service contact data from the NCS-R leads to the fourth major finding and presents a dismal tale of failure, delay and lost opportunity.⁴⁰ The vast majority of people with lifetime disorders eventually made contact with health services, although more commonly for mood disorders (88–94%) than for anxiety (27–95%), impulse control (34–52%) or substance use (53–77%) disorders. Delay among those who eventually made contact ranged from 6 years to 8 years for mood disorders and from 9 years to 23 years for anxiety disorders. Failure to make initial treatment contact at all and delay among those who did eventually make treatment contact were both associated with early age at onset, being older at the time of the survey, and a number of sociodemographic characteristics including being

male, married, poorly educated and from a Black and minority ethnic group.

From neurobiology to service provision

One further question is whether what is happening to the brain during the adolescent period mirrors this tide of incident mental health disorder where, excluding the neurodegenerative disorders of later life, half of all onsets have occurred by the mid-teens.

The brain is constantly changing throughout life; even reading this article may consolidate some aspect of neural circuitry and leave a memory trace. That said, there are several epochs over the life course where there is radical transformation of neural architecture and function. One of these epochs spans the traditional legal, educational and service boundary of 17–18 years of age. In fact, this artificial transition falls right in the middle of a crucial phase of brain development that begins shortly before puberty and ends during the third decade of life. This questions the whole concept of adolescence (based on sexual maturity) being distinct from adulthood (based on a concept of attainment of mental and physical capacity).

Development of the neonatal brain from its ectodermal origins is a dramatic achievement of nature. Itself complex and depending on a variety of processes such as the establishment of connectivity and programmed cell death, followed by a rapid development of basic cabling through myelination in the first year, this epoch ushers in some classical neurodevelopmental disorders and learning delays; but this is only part of the story. It takes three decades to grow a mature human brain, during which time there is much further development. The period from mid-childhood through puberty to the mid-20s is another phase of profound change, particularly in the balance of connectivity between brain areas.⁴¹ It is during this period that the risk of adult mental health disorders becomes apparent; it may be the characteristics of normal development that lead to this risk, rather than any particular abnormality in this process.⁴² Indeed, aspects of syndromes such as impulse control disorders are so common during adolescence that it is questionable as to whether they should be considered abnormal. This second, more leisurely phase of development has only recently begun to be understood. It is probably heralded by prepubertal maturational events in the adrenal glands known as adrenarche that take place around 6–8 years of age.⁴³ Adrenarche involves the production of two steroid compounds, dehydroepiandrosterone (DHEA) and its metabolite dehydroepiandrosterone sulfate (DHEAS), which are abundant within the brain. Longitudinal studies of brain structure using repeated magnetic resonance imaging scans in healthy children have demonstrated considerable pre-adolescent increases in cortical grey matter, peaking some 2–3 years after adrenarche and then declining in an inverted U-shaped manner.^{44,45} Increases

Table 1 Ages at onset for five categories of mental health disorder

	Projected lifetime risk, ^a % (s.e.)	Age at which % of projected lifetime risk attained, years ^b		
		25%	50% (median)	75%
Anxiety disorders	31.5 (1.1)	6	11	21
Mood disorders	28.0 (0.8)	18	30	43
Impulse control disorders	25.4 (1.1)	7	11	15
Substance use disorders	16.3 (0.6)	18	20	27
Any disorder	50.8 (1.2)	7	14	24

a. Proportion of whole population that will have experienced disorder by age 75 years.
b. Data for standardised age-at-onset distributions of DSM-IV diagnoses derived from the World Health Organization Composite International Diagnostic Interview with projected lifetime risk at age 75 years; adapted from Kessler *et al* where data on specific diagnoses and details of the sample are available.³¹

in white matter occur over a longer period into the third decade. There is a general pattern of childhood peaks of grey matter followed by adolescent declines, functional and structural increases in connectivity and integrative processing;⁴⁶ the balance between limbic and subcortical and frontal lobe functions extends well into young adulthood.⁴⁶ The higher-order association cortices mature after lower-order somatosensory and visual cortices, and phylogenetically older brain areas mature earlier than those more recently evolved such as the frontal cortex. The control of these changes, particularly the initial childhood growth in grey matter, is likely to be dependent on androgens, with regional differences and contrasts between girls and boys being mediated by differences in androgen receptor density and function.⁴⁷ Cortical developments are more dependent on environmental influences and transactions. Although the brains of children and young adults may have similar 'small world' organisation at the global level, they differ significantly in hierarchical organisation and interregional connectivity. These large-scale changes underpin the transition from dependent child to independent adult able to draw on sophisticated learning from reward and punishment, and effective executive and emotional control.^{48–50}

Casey *et al* provide a model of these key brain structural and neurocognitive phenotypes developing at different rates.⁴² They highlight the relevance of normal development in understanding emergence of psychopathology in the developmental epoch from puberty to the late 20s.⁴⁵ A 'neural maturation gap' during adolescence exists between earlier maturation of limbic and subcortical reward processing networks dependent on the neurosteroid growth spurt, and later consolidation of neocortical cognitive and emotional control networks dependent on environmental experience. This maturation gap may present a window of vulnerability during which these two different systems are not yet fully coordinated.

Health service implications

It seems that from the perspective of brain development it should come as no surprise that most adult mental health disorders have their roots in adolescence. We may ask why they persist thereafter, and what can be done about them. Mechanisms of persistence are beyond the scope of this article but the robust findings from modern epidemiology and their consilience with the emerging neurobiology of adolescence should be a clarion call for intervention involving both primary and secondary prevention.⁵⁰ Many incident disorders will resolve but it is clear that many do persist, and result in lifelong disability and cost to society and the individual. Not every anxiety disorder or depression in the childhood or teenage years will lead to adult disorder. However, the findings suggest that appropriate interventions should be widely available to speed initial treatment contact and reduce the burdens and hazards of untreated mental disorder.⁴⁰ It is straightforward to propose that early signs of recurrence or persistence of adolescent mental health disorder should lead to a concerted therapeutic effort, probably coordinated by primary care, and there is evidence to suggest that – in psychotic disorders at least – such early specialist intervention is cost-effective.⁵¹ However, the appropriate response to the earliest signs of disorder in large numbers of young people is less clear, and the risk to benefit ratio of specialist care will be totally different in the wider subclinical or primary care population from that in the services in which many interventions are developed. Traditional mental health services have evolved without the knowledge that psychopathology observes no transition between adolescence and early adulthood. From the perspective of brain development, services

may require re-engineering to provide an appropriately seamless and developmentally sensitive approach to individuals on the two-decade journey from puberty to adulthood. These questions are explored elsewhere in this supplement and require considerable and sophisticated research in order to guide appropriate care at the clinical and public health levels.

Establishing age at onset of mental health disorders is methodologically difficult. A range of epidemiological approaches provide convergent evidence not only that adult mental health disorders are common in the population, but that most of them have their onset by adolescence. This period of life after puberty also sees a range of important organisational developments in the brain that last until the late 20s and usher in adulthood as might be defined from a developmental rather than a social or legal point of view. These biological changes are characterised by a temporary uncoupling of higher-order, more recently evolved cortical functions from the limbic and subcortical systems that mature earlier. This maturational gap is normal, but appears to leave the adolescent vulnerable to a range of experiences and responses, some of which form the components of mental disorders and may persist as such. These findings provide a persuasive argument to reappraise our formulations of mental health disorder into a new developmental psychiatry embracing a modern life-course approach that focuses on the second and third decades of life. Such a view also triggers the need for a new research agenda to guide these new ventures, the therapeutic interventions they may wield and the service structures through which these are delivered to young people.

P. B. Jones, PhD, Department of Psychiatry, University of Cambridge, CAMEO Early Intervention Services, Cambridgeshire and Peterborough Foundation National Health Service Trust, Herchel Smith Building for Brain and Mind Science, Cambridge CB2 0SX, UK. Email: pbj21@cam.ac.uk

Funding

P.B.J. acknowledges research support from the Wellcome Trust and the National Institute for Health Research (NIHR; programme grant RP-PG-0606-1335). The work forms part of the NIHR Collaboration for Leadership in Applied Health Research and Care for Cambridgeshire & Peterborough.

Acknowledgements

The author thanks Mrs Anne Robinson and Dr Golam Khandaker for their help with referencing.

References

- McGorry P, Johansen JO, Lewis S, Birchwood M, Malla A, Nordentoft M, et al. Early intervention in psychosis: keeping faith with evidence-based health care. *Psychol Med* 2010; **40**: 399–404.
- Nicolson R, Lenane M, Hamburger SD, Fernandez T, Bedwell J, Rapoport JL. Lessons from childhood-onset schizophrenia. *Brain Res Brain Res Rev* 2000; **31**: 147–56.
- Alvarez-Jimenez M, Gleeson JF, Henry LP, Harrigan SM, Harris MG, Amminger GP, et al. Prediction of a single psychotic episode: a 7.5-year, prospective study in first-episode psychosis. *Schizophr Res* 2011; **125**: 236–46.
- Kessler RC, Amminger GP, Aguilar-Gaxiola S, Alonso J, Lee S, Ustun TB. Age of onset of mental disorders: a review of recent literature. *Curr Opin Psychiatry* 2007; **20**: 359–64.
- Simon GE, VonKorff M. Recall of psychiatric history in cross-sectional surveys: implications for epidemiologic research. *Epidemiol Rev* 1995; **17**: 221–7.
- Perala J, Suvisaari J, Saarni SI, Kuoppasalmi K, Isometsa E, Pirkola S, et al. Lifetime prevalence of psychotic and bipolar I disorders in a general population. *Arch Gen Psychiatry* 2007; **64**: 19–28.
- Yan LL, Liu K, Daviglius ML, Colangelo LA, Kiefe CI, Sidney S, et al. Education, 15-year risk factor progression, and coronary artery calcium in young adulthood and early middle age: the Coronary Artery Risk Development in Young Adults study. *JAMA* 2006; **295**: 1793–800.

- 8 Jones P, Rodgers B, Murray R, Marmot M. Child development risk factors for adult schizophrenia in the British 1946 birth cohort. *Lancet* 1994; **344**: 1398–402.
- 9 Isohanni M, Makikyro T, Moring J, Rasanen P, Hakko H, Partanen U, et al. A comparison of clinical and research DSM-III-R diagnoses of schizophrenia in a Finnish national birth cohort. Clinical and research diagnoses of schizophrenia. *Soc Psychiatry Psychiatr Epidemiol* 1997; **32**: 303–8.
- 10 Ridler K, Veijola JM, Tanskanen P, Miettunen J, Chitnis X, Suckling J, et al. Fronto-cerebellar systems are associated with infant motor and adult executive functions in healthy adults but not in schizophrenia. *Proc Natl Acad Sci USA* 2006; **103**: 15651–6.
- 11 Kirkbride JB, Fearon P, Morgan C, Dazzan P, Morgan K, Tarrant J, et al. Heterogeneity in incidence rates of schizophrenia and other psychotic syndromes: findings from the 3-center AeSOP study. *Arch Gen Psychiatry* 2006; **63**: 250–8.
- 12 Crichton-Browne J. The psychical diseases of early life. *J Ment Sci* 1860; **6**: 284–320.
- 13 Bleuler E. *Dementia Praecox oder Gruppe der Schizophrenien [Dementia Praecox or The Group of Schizophrenias]*. Deuticke, 1911.
- 14 Kraepelin E. Dementia praecox and paraphrenia. In *Textbook of Psychiatry*, 8th edn (ed. G Robertson, transl. RM Barclay): vol 3, part 2. Livingstone, 1919.
- 15 Murray RM, Lewis SW. Is schizophrenia a neurodevelopmental disorder? *BMJ* 1987; **295**: 681–2.
- 16 Häfner H, Maurer K, Löffler W, Fatkenheuer B, van der Heiden W, Riecher-Rossler A, et al. The epidemiology of early schizophrenia. Influence of age and gender on onset and early course. *Br J Psychiatry* 1994; **164** (suppl 23): 29–38.
- 17 Häfner H, Behrens S, de Vry J, Gattaz WF, Löffler W, Maurer K, et al. Warum erkranken Frauen später an Schizophrenie? *Nervenheilkunde* 1991; **10**: 154–63.
- 18 Goodyer I. *The Depressed Child and Adolescent*, 2nd edn. Cambridge University Press, 2001.
- 19 Fombonne E, Wostear G, Cooper V, Harrington R, Rutter M. The Maudsley long-term follow-up of child and adolescent depression: 1. Psychiatric outcomes in adulthood. *Br J Psychiatry* 2001; **179**: 210–7.
- 20 Harrington R, Bredenkaamp D, Groothues C, Rutter M, Fudge H, Pickles A. Adult outcomes of childhood and adolescent depression. III. Links with suicidal behaviours. *J Child Psychol Psychiatry* 1994; **35**: 1309–19.
- 21 Harrington R, Fudge H, Rutter M, Pickles A, Hill J. Adult outcomes of childhood and adolescent depression. I. Psychiatric status. *Arch Gen Psychiatry* 1990; **47**: 465–73.
- 22 Harrington R, Fudge H, Rutter M, Pickles A, Hill J. Adult outcomes of childhood and adolescent depression: II. Links with antisocial disorders. *J Am Acad Child Adolesc Psychiatry* 1991; **30**: 434–9.
- 23 Fombonne E, Wostear G, Cooper V, Harrington R, Rutter M. The Maudsley long-term follow-up of child and adolescent depression: 2. Suicidality, criminality and social dysfunction in adulthood. *Br J Psychiatry* 2001; **179**: 218–23.
- 24 Ford T, Goodman R, Meltzer H. The British Child and Adolescent Mental Health Survey 1999: the prevalence of DSM-IV disorders. *J Am Acad Child Adolesc Psychiatry* 2003; **42**: 1203–11.
- 25 Mann A. The evolving face of psychiatric epidemiology. *Br J Psychiatry* 1997; **171**: 314–8.
- 26 Wadsworth MEJ. *The Imprint of Time: Childhood History and Adult Life*. Clarendon, 1991.
- 27 Colman I, Wadsworth ME, Croudace TJ, Jones PB. Forty-year psychiatric outcomes following assessment for internalizing disorder in adolescence. *Am J Psychiatry* 2007; **164**: 126–33.
- 28 Silva PA, Stanton WR. *From Child to Adult: The Dunedin Multidisciplinary Health and Development Study*. Oxford University Press, 1996.
- 29 Robins LN, Regier DA. *Psychiatric Disorders in America: The Epidemiologic Catchment Area Study*. Free Press, 1991.
- 30 Kim-Cohen J, Caspi A, Moffitt TE, Harrington H, Milne BJ, Poulton R. Prior juvenile diagnoses in adults with mental disorder: developmental follow-back of a prospective-longitudinal cohort. *Arch Gen Psychiatry* 2003; **60**: 709–17.
- 31 Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Arch Gen Psychiatry* 2005; **62**: 593–602.
- 32 Kessler RC, Chiu WT, Demler O, Merikangas KR, Walters EE. Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Arch Gen Psychiatry* 2005; **62**: 617–27.
- 33 Kessler RC, McGonagle KA, Zhao S, Nelson CB, Hughes M, Eshleman S, et al. Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States. Results from the National Comorbidity Survey. *Arch Gen Psychiatry* 1994; **51**: 8–19.
- 34 Kessler RC, Angermeyer M, Anthony JC, de Graaf R, Demyttenaere K, Gasquet I, et al. Lifetime prevalence and age-of-onset distributions of mental disorders in the World Health Organization's World Mental Health Survey Initiative. *World Psychiatry* 2007; **6**: 168–76.
- 35 Wittchen HU. Reliability and validity studies of the WHO-Composite International Diagnostic Interview (CIDI): a critical review. *J Psychiatr Res* 1994; **28**: 57–84.
- 36 Spitzer RL, Williams JB, Gibbon M, First MB. The Structured Clinical Interview for DSM-III-R (SCID). I: History, rationale, and description. *Arch Gen Psychiatry* 1992; **49**: 624–9.
- 37 Hamilton M. A rating scale for depression. *J Neurol Neurosurg Psychiatry* 1960; **23**: 56–62.
- 38 Rush AJ, Trivedi MH, Ibrahim HM, Carmody TJ, Arnow B, Klein DN, et al. The 16-Item Quick Inventory of Depressive Symptomatology (QIDS), clinician rating (QIDS-C), and self-report (QIDS-SR): a psychometric evaluation in patients with chronic major depression. *Biol Psychiatry* 2003; **54**: 573–83.
- 39 Knauper B, Cannell CF, Schwarz N, Bruce ML, Kessler RC. Improving the accuracy of major depression age of onset reports in the US National Comorbidity Survey. *Int J Methods Psychiatr Res* 1999; **8**: 39–48.
- 40 Wang PS, Berglund P, Olfson M, Pincus HA, Wells KB, Kessler RC. Failure and delay in initial treatment contact after first onset of mental disorders in the National Comorbidity Survey Replication. *Arch Gen Psychiatry* 2005; **62**: 603–13.
- 41 Blakemore SJ, Burnett S, Dahl RE. The role of puberty in the developing adolescent brain. *Hum Brain Mapp* 2010; **31**: 926–33.
- 42 Casey BJ, Duhoux S, Malter Cohen M. Adolescence: what do transmission, transition, and translation have to do with it? *Neuron* 2010; **67**: 749–60.
- 43 Auchus RJ. The physiology and biochemistry of adrenarche. *Endocr Dev* 2011; **20**: 20–7.
- 44 Giedd JN, Blumenthal J, Jeffries NO, Castellanos FX, Liu H, Zijdenbos A, et al. Brain development during childhood and adolescence: a longitudinal MRI study. *Nat Neurosci* 1999; **2**: 861–3.
- 45 Paus T, Zijdenbos A, Worsley K, Collins DL, Blumenthal J, Giedd JN, et al. Structural maturation of neural pathways in children and adolescents: in vivo study. *Science* 1999; **283**: 1908–11.
- 46 Gogtay N, Giedd JN, Lusk L, Hayashi KM, Greenstein D, Vaituzis AC, et al. Dynamic mapping of human cortical development during childhood through early adulthood. *Proc Natl Acad Sci USA* 2004; **101**: 8174–9.
- 47 Raznahan A, Lee Y, Stidd R, Long R, Greenstein D, Clasen L, et al. Longitudinally mapping the influence of sex and androgen signaling on the dynamics of human cortical maturation in adolescence. *Proc Natl Acad Sci USA* 2010; **107**: 16988–93.
- 48 Galvan A, Hare TA, Parra CE, Penn J, Voss H, Glover G, et al. Earlier development of the accumbens relative to orbitofrontal cortex might underlie risk-taking behavior in adolescents. *J Neurosci* 2006; **26**: 6885–92.
- 49 Supekar K, Musen M, Menon V. Development of large-scale functional brain networks in children. *PLoS Biol* 2009; **7**: e1000157.
- 50 Insel TR, Fenton WS. Psychiatric epidemiology: it's not just about counting anymore. *Arch Gen Psychiatry* 2005; **62**: 590–2.
- 51 McCrone P, Craig TKJ, Power P, Garety PA. Cost-effectiveness of an early intervention service for people with psychosis. *Br J Psychiatry* 2010; **196**: 377–82.

Special article

Clinical staging in severe mental disorder:
evidence from neurocognition and neuroimagingAshleigh Lin,[†] Renate L. E. P. Reniers[†] and Stephen J. Wood**Summary**

A new approach to understanding severe mental illnesses such as schizophrenia and affective disorders is to adopt a clinical staging model. Such a model defines the extent of the illness such that earlier and milder phenomena are distinguished from later, more impairing features. Part of the appeal of such a model is that it should have cross-diagnostic applications, but to date there has been no attempt to examine imaging or neurocognitive evidence for staging in this way. We review these two domains of study with particular focus on major depression and

bipolar affective disorder. Although there is some support for the staging model in affective disorders, conclusions are limited by the large variability in the clinical samples studied, especially with regard to the presence of psychotic symptoms. We suggest that future research needs to take a transdiagnostic and longitudinal approach.

Declaration of interest

None.

Clinical staging is a practical tool that has demonstrated utility in general medicine. It defines the extent of progression of disease at a particular point in time, and where a person's condition currently lies along a continuum of the course of illness.^{1,2} For example, stages of certain cancers are distinguished by the extent of local invasion of tumour, lymphatic involvement and metastatic spread. Thus, early and milder clinical phenomena are differentiated from later stages that have evidence of illness extension, progression and chronicity. From a practical perspective, clinical staging enables the clinician to select treatments relevant to the stage, with less invasive interventions being more effective in earlier stages than when delivered later in the illness course.¹ Again, the cancer analogy is useful here: minor surgery and local radiotherapy may be appropriate for early stages of breast cancer, whereas in later stages this would not be sufficient and more radical treatment such as mastectomy and chemotherapy might be indicated.

It has been proposed that the concept of a staging model can be applied to psychiatry.¹ In particular we put forward the hypothesis that severe mental disorders, such as schizophrenia, bipolar affective disorder and severe depression, develop from initial non-specific symptoms and syndromes (i.e. a pluripotential state) and from a background of specific and non-specific risk factors such as genes or early environment. From the initial non-specific clinical picture, worsening of symptoms and acquisition of new symptoms occur, together with progressive neurobiological changes and related neurobehavioural deficits, until clearly recognisable mental disorder appears. Further progression of symptoms and neurobiological abnormalities may occur after 'threshold' diagnosis. Thus, the natural history of major mental illness is postulated to consist of transition from being asymptomatic and not seeking help, through a stage of undifferentiated general symptoms such as mild anxiety, depressive and somatic symptoms, followed by the worsening of existing symptoms and acquisition of new ones (e.g. psychotic-like experiences, substance use) which may be associated with behavioural and functional decline. Further progression of illness may still occur, with development of chronic symptoms, relapses and ongoing impairment. Although the staging approach has

much intellectual appeal, it is still a heuristic concept with extensive research work required to develop stage markers. Previously we have examined the evidence for clinical staging in schizophrenia, with a particular focus on neuroimaging and treatment data.² Here we extend this investigation to neurocognitive findings and to affective disorders.

Neurocognition

Neurocognitive impairments are a feature of severe mental illness, but it is unclear whether these impairments support a clinical staging model. In schizophrenia, cognitive impairment is large and documented across a range of cognitive domains, most notably verbal learning and memory, performance and full-scale IQ scores, sustained attention and cognitive flexibility.³ Similarly, moderate impairments in a number of domains are documented in bipolar affective disorder, the largest occurring in verbal learning and memory and in executive function;^{4–7} these are evident during euthymia and amplified when symptoms are experienced.^{8,9} Individuals with major depressive disorder also show neurocognitive impairment, although the affected domains are unclear; impairments have most consistently been demonstrated in verbal learning and memory, attention and executive function,^{10–12} although to a lesser extent than in schizophrenia and bipolar disorder.

If neurocognition is to be a reliable indicator of clinical stage, then variability in performance should indicate illness severity, chronicity and progression. In schizophrenia the relationship between chronicity and impairment is not straightforward. The magnitude of neurocognitive impairment in the first psychotic episode¹³ is equivalent to that of samples with established illness,³ suggesting that there is no further decline in neurocognitive ability after the onset of frank psychotic symptoms. This is supported by a lack of longitudinal evidence of progressive deterioration over illness course.¹⁴ A subgroup of these individuals who develop 'deficit' schizophrenia,¹⁵ characterised by a chronic illness course, prominent negative symptoms, poor functional outcome and significantly reduced cognitive performance,^{16,17} might show progressive impairment. However, it seems more likely that deficits are longstanding rather than associated with transition between clinical stages; cognitive deficit early in the illness course

[†]These authors contributed equally to the work.

is predictive of poor functional outcome and negative symptoms many years later.^{18,19} In contrast, data from samples of people with bipolar disorder show evidence of a relationship between multiple episodes (both manic and depressive) and poorer neurocognitive performance, particularly for verbal learning and memory and for executive function.^{4,5} In these individuals longer illness duration is also associated with greater impairment, although not as robustly as number of affective episodes.^{7,8} Similarly, meta-analytic evidence from individuals with major depression suggests that symptom severity is significantly associated with neurocognitive impairment in episodic memory, executive function and processing speed, but these associations explain less than 10% of the variance in performance.²⁰ Other cross-sectional evidence for a relationship between other indices of severity (duration of illness, number of episodes and length of episodes) and cognitive deficits is variable and conclusions are difficult to draw.¹²

Evidence regarding the longitudinal course of neurocognitive impairment in affective disorders is limited by the lack of longitudinal studies.^{11,21} The longest follow-up of individuals with bipolar disorder showed that cognitive impairment persisted but did not deteriorate over a 3-year period.²² Longitudinal studies of neurocognition in major depression are rarely longer than 6 months,¹¹ making it impossible to ascertain how impairment progresses over the illness. Our understanding of the course of neurocognitive impairment in affective disorders is further complicated by the fact that state-related reductions in cognitive performance may persist over the short term, leading to the misclassification of such impairment as trait-related.^{5,11}

Another problem in interpreting the neurocognitive performance of individuals with affective disorders is the effect of confounding factors, which are often not controlled for in analyses.^{11,21} These include the impact of medication, illness subtype, age, comorbid disorders and substance use, all of which may influence cognitive performance at the time of testing. In particular, a history of psychosis is rarely reported or controlled for, yet almost all of the published research has shown that current or past psychotic symptoms are associated with greater and more broad cognitive impairments in bipolar disorder and major depression.^{23–27} Individuals with affective disorders without psychotic features have been shown to perform at a level equivalent to healthy controls or show only minimal impairment.^{28–30} The profile of individuals with non-psychotic depression is more consistent with frontostriatal dysfunction (i.e. reduced performance was most evident in attention and executive function), as opposed to the frontotemporal dysfunction associated with schizophrenia.²⁷ In line with this, meta-analytic evidence demonstrates that the largest decrements in samples with affective psychosis are in psychomotor speed, sustained attention, verbal learning and memory and semantic fluency, similar to those observed in schizophrenia.^{3,31} Altogether, this suggests that psychotic rather than affective pathology is driving impairments; teasing out the effects of psychosis and other confounds is vital to our understanding of the pattern of neurocognitive impairment in affective disorder in regard to clinical staging.

The utility of neurocognition in clinical staging is enhanced if alterations in cognitive performance are evident early in the illness. Research has shown that individuals who later develop schizophrenia demonstrate poor academic performance and intellectual ability in childhood and adolescence.^{32–37} Furthermore, it is now accepted that individuals at ultra-high risk of psychosis also perform worse than healthy controls across a range of neurocognitive domains. Within this group, those who make the transition to frank psychosis show greater impairment than those who do not develop psychosis, primarily in the verbal

domain. The most often cited reductions include lower general vocabulary or verbal IQ score,^{38–41} verbal learning and memory,^{38,39,42–44} verbal fluency^{39,43,45} and slower processing speed.^{39,40,46} It remains unclear whether a decrement in cognition occurs from the prodromal stage to the first-episode stage of illness. Some cross-sectional studies have demonstrated that the magnitude of impairment in the ultra-high risk group who later develop psychosis is comparable to first-episode populations, at least in overall ability,^{38,44} verbal IQ score⁴¹ and verbal memory.^{38,41} Only a few studies have followed ultra-high risk samples over the period of transition to psychosis, and these found little or no progressive impairment in neurocognitive ability.^{47–49} (Further information available from the authors.)

Potential early neurocognitive impairment in bipolar disorder is less well understood. There is evidence of lowered performance in unaffected relatives of patients, particularly in verbal learning and memory and some executive functions,^{7,50} suggesting that cognitive reductions may be trait-related. However, findings to date show neurocognition to be an unreliable indicator of future bipolar disorder. Some studies have identified intellectual and language delays and lowered visual spatial reasoning and set-shifting in young people who later developed the illness,^{51–53} but others found no reduction relative to healthy controls.^{32,33,37,54} Once again, conclusions from these studies are limited by confounding psychotic symptoms; most studies did not differentiate the outcomes of bipolar disorder with and without psychosis.

High-risk clinics do not exist for bipolar disorder in the same way that they do for schizophrenia, making it difficult to characterise neurocognitive ability immediately prior to illness onset. Only one study has assessed cognitive performance in the prodromal period of bipolar disorder by investigating the onset of this disorder in patients at ultra-high risk of psychosis.⁵⁵ The authors found no difference in premorbid IQ, current IQ or global ability between individuals who developed bipolar disorder and those who did not develop either bipolar disorder or schizophrenia. It is similarly unclear which impairments might be present immediately after the onset of bipolar disorder, especially since a first episode of mania could have been preceded by a period of depression. Albus *et al* found that individuals with a first episode of mania without psychosis demonstrated cognitive performance that was equivalent to healthy controls.²⁸ Two other studies of first-episode mania have shown impairment in verbal fluency, perceptual-motor ability, set-shifting and psychomotor speed/attention;^{56,57} however, neither study reported the prevalence of psychotic symptoms in the samples.

The evidence also suggests that early neurocognition is not useful as an indicator of later major depression. Studies of offspring of women with depression have identified a specific reduction in verbal ability⁵⁸ or no decrement at all,^{59,60} relative to healthy controls. Interestingly, in offspring of women with depression there was an association between current depressive symptoms and performance on some indices of executive function and processing speed,⁶⁰ suggesting that reductions in these domains might be related to current symptoms or occur as part of the disease process rather than being markers of vulnerability. On the other hand, evidence from a large population study demonstrated that children who later developed major depression showed poorer performance on tasks of psychomotor speed and attention at age 13 years but no reduction in other domains,³³ nor deviations in motor or language development.³² Considering the evidence to date it is unclear whether cognition is impaired before the diagnosis of depressive disorder.

It is important to note that not all individuals with these severe mental illnesses show cognitive impairment. A quarter to a third of individuals with schizophrenia demonstrate 'normal'

neurocognitive performance within the average range.^{30,61} Furthermore, it has been estimated that only 28% of people with major depression,^{30,62} and 38–41% of those with bipolar disorder,^{62,63} have neurocognitive impairment. It is possible that people with psychotic and affective disorders who present with neurocognitive impairments represent a different underlying disease process. Support for the concept of different disease processes comes from demographic and clinical differences between those with impaired and intact neurocognitive performance in schizophrenia and bipolar disorder.^{61,63} Clinical staging offers the potential to overcome artificial diagnostic boundaries by incorporating cognitive performance into stage definitions, although its usefulness is dependent on how well it can be used to predict illness progression and treatment response.

In summary, although there is some evidence that neurocognitive impairment might fit within a clinical staging framework, there are too many confounds at present for it to be incorporated into the model. Critically, these conclusions are limited by the large variability of clinical populations with affective disorders, particularly where data from individuals with and without psychotic symptoms are combined. Further, the lack of longitudinal data examining progression over time in affective disorders and comparing individuals with early- v. late-stage disorder reduces our ability to draw strong inferences.

Neuroimaging

In addition to neurocognitive functioning, a model of clinical staging should differentiate the neurobiological correlates of the disorder's distinct stages. Neurobiological changes associated with mental disorders do not necessarily develop in parallel with behavioural symptoms or correlate with behaviour,⁶⁴ highlighting the importance of investigating both. We recently reviewed the imaging literature for psychotic disorders and showed that whereas some neurobiological changes are already present before the illness onset, others arise as it progresses and tend to be more pronounced with severity of illness.^{2,65} Similar differential patterns of biomarkers have been suggested for early v. late stages of major depression and bipolar disorder,^{66–69} suggesting it might also be possible to find neuroimaging markers of specific illness stages in affective disorders.

Enhanced vulnerability to psychosis is associated with grey-matter volume reductions in prefrontal, limbic and temporo-parietal regions of the brain,⁷⁰ whereas those who later develop first-episode psychosis have more specific reductions in the inferior frontal, superior temporal and parietal regions.^{70,71} Not surprisingly, therefore, individuals with schizophrenia commonly present with reductions in grey matter in the frontotemporal regions.^{72,73} Consistent with the predictions of clinical staging, these changes become more extensive through first-episode and chronic illness.^{74–77} A similar staging pattern has been observed for cortical thickness,⁷⁸ and for structural abnormalities affecting white matter.⁷⁹

Progressive brain changes and increased pathological signs related to severity of illness have also been observed in affective disorders. In major depression reduced thickness of the posterior cingulate cortex has been observed in people with non-remitted disorder compared with those in remission, and decreased perfusion in frontal regions and the anterior cingulate cortex has been shown in the non-remission group compared with healthy controls.⁸⁰ There are cautious suggestions that reductions in cerebral and cerebellar grey matter volume,⁸¹ as well as basal ganglia volume,⁸² are related to severity of illness. Furthermore, basal ganglia volume reductions have been linked to illness

duration and the number of prior depressive episodes.⁸³ With increased duration of illness, individuals with major depression have shown reduced glutamate and increased choline concentrations in ventromedial prefrontal regions,⁸⁴ and (more inconsistently) a reduction in hippocampal volume.^{85–88} In bipolar disorder the number of episodes of illness has been related to enlargement of the lateral ventricles,^{89,90} and decreased cerebellar vermal volume.⁹¹ Compared with healthy individuals, grey matter density of the hippocampus, fusiform gyrus and cerebellum of individuals with bipolar disorder has been shown to reduce at an accelerated rate.⁹²

Many structural abnormalities such as ventricular enlargement have been repeatedly associated with both schizophrenia and affective disorders, albeit with greater enlargements in schizophrenia.^{5,8,88,93,94} Such non-specific changes perhaps reflect the presence of psychotic symptoms in the affective disorder group,⁸ and/or similarities relating to clinical stage. Diagnostic differences do exist, however. Smaller hippocampal and amygdala volumes have been observed in individuals with schizophrenia compared with bipolar disorder.⁹⁵ Further distinctions on the basis of grey matter deficits have been made,⁹⁶ and functional differences in medial frontal and visual cortex, as well as differential disruptions in white matter tracts associated with the occipital and frontal lobes,⁹⁷ have been shown. Whereas volumetric reductions in brain tissue, in particular temporal lobe grey matter, are more consistently found in schizophrenia than in bipolar disorder, white matter hyperintensities are more common in affective disorders.⁸ Individuals with bipolar disorder additionally show enlargement of basal ganglia and amygdala, whereas those with major depressive disorder are characterised by volume reduction in these regions as well as in the hippocampus.^{88,98,99} Affective disorders are furthermore distinguished by increased corpus callosum cross-sectional area in major depression compared with bipolar disorder.⁸⁸ These neurobiological differences may provide useful diagnostic markers in relation to the different stages of the individual disorders.

The reports of white matter pathology indicate that severe mental illness may not simply be a result of structural lesions to the brain,⁵ but rather of abnormal connectivity between regions.¹⁰⁰ As early as 1998 Friston suggested that schizophrenia was caused by dysfunctional interaction in the dynamics of associated brain regions rather than by dysfunctional specialisation within a region.¹⁰¹ Certainly in psychotic disorders, stage of illness affects structural and functional networks differently.² Consistent across stages of illness are findings of reduced (or in some cases increased) connectivity in frontal lobe and frontotemporal interactions, but as illness progresses these patterns become more widespread across the brain and are observed with higher frequencies.¹⁰⁰ Progression between stages could represent weakened strength of connections or even a total loss of connections in a network with a consequential imbalance between local and global connections. Indeed, connection patterns could show alterations, resulting in a loss of function such as working memory impairment,¹⁰² or phenomena such as positive symptoms.¹⁰³ Evidence for dysconnectivity in grey and white matter across all stages of this disorder, and even before onset of illness, is building.^{64,100,104–106} Suggestions of dysconnectivity in affective disorders are also rapidly emerging,^{5,107–109} with distinct patterns for schizophrenia and bipolar disorder being identified.¹¹⁰ Factors such as genetics, insults during brain development and neurotransmitter imbalance are thought to influence the process of dysconnectivity.¹¹¹

For neurobiological changes to consolidate their position in a model of clinical staging, changes caused by the illness need to be distinguished from epiphenomena. Factors such as life stress and

substance use have been related to progression in severity of illness,^{2,112–114} and individual differences in dysconnectivity have been shown to relate to individual differences in symptom presentation.¹¹¹ Antidepressant medication has been found to decrease resting-state functional connectivity,¹¹⁵ and the effects of medication on brain structure, volume and functioning also require further investigation.^{2,70,100,113,116,117} In addition, the influence of adolescent development,^{2,90,100,113,118,119} gender,¹⁰⁰ and comorbidity,^{2,86,120,121} should be considered. Accounting for these factors provides important challenges for the immediate future.

Current models of clinical staging do not make explicit whether an individual can move down a stage, i.e. whether a remission of symptoms is equivalent to moving from stage 2 to stage 1. However, certain functions can (at least partially) be recovered.^{122,123} Furthermore, brain volume abnormalities seem to be potentially reversible (in first-episode psychosis),¹²⁴ or at least to lessen with continued development in childhood-onset schizophrenia.¹²⁵ This suggests regression in severity of illness to some extent, which should be reflected in the staging model.

Neurobiological evidence for staging in severe mental illness is still limited. Different methods adopted by the various studies make it difficult to compare findings and stress the need for future research to adopt a transdiagnostic perspective. Studies investigating disorders with overlapping features will not only be able to highlight shared neurobiological features but may provide evidence for distinct neurobiological markers important for treatment and prognosis. As adolescence is the critical period for onset of severe mental illness, studies should focus on brain networks that develop during this period. Furthermore, multi-modal longitudinal studies will be crucial in monitoring transition between stages and associated neurobiological changes.²

Future research

Clinical staging is a promising model for improving our understanding of the way in which severe mental illnesses develop and helping clinicians choose the most appropriate treatment. Both neurocognition and neuroimaging evidence provide tentative support for the application of a staging model to schizophrenia and affective disorders. The paradox here is that we are using the current diagnostic categories to investigate the validity of a model that explicitly attempts to negate the current categorical system. Future work needs to take a transdiagnostic and longitudinal view, covering both neurocognition and neuroimaging in order to overcome current issues.

Ashleigh Lin, PhD, **Renate L. E. P. Reniers**, PhD, School of Psychology, University of Birmingham, UK; **Stephen J. Wood**, PhD, School of Psychology, University of Birmingham, UK, Melbourne Neuropsychiatry Centre, Department of Psychiatry, University of Melbourne and Melbourne Health, Melbourne, Australia

Correspondence: Professor Stephen Wood, School of Psychology, University of Birmingham, Edgbaston B15 2TT, UK. Email: s.j.wood@bham.ac.uk

References

- McGorry PD, Hickie IB, Yung AR, Pantelis C, Jackson HJ. Clinical staging of psychiatric disorders: a heuristic framework for choosing earlier, safer and more effective interventions. *Aust N Z J Psychiatry* 2006; **40**: 616–22.
- Wood SJ, Yung AR, McGorry PD, Pantelis C. Neuroimaging and treatment evidence for clinical staging in psychotic disorders: from the at-risk mental state to chronic schizophrenia. *Biol Psychiatry* 2011; **70**: 619–25.
- Heinrichs RW, Zakzanis KK. Neurocognitive deficits in schizophrenia: a quantitative review of the evidence. *Neuropsychology* 1998; **12**: 426–45.
- Quraishi S, Frangou S. Neuropsychology of bipolar disorder: a review. *J Affect Disord* 2002; **72**: 209–26.
- Savitz J, Solms M, Ramesar R. Neuropsychological dysfunction in bipolar affective disorder: a critical opinion. *Bipolar Disord* 2005; **7**: 216–35.
- Robinson O, Sahakian B. Recurrence in major depressive disorder: a neurocognitive perspective. *Psychol Med* 2008; **38**: 315–8.
- Bora E, Yucel M, Pantelis C. Cognitive endophenotypes of bipolar disorder: a meta-analysis of neuropsychological deficits in euthymic patients and their first-degree relatives. *J Affect Disord* 2009; **113**: 1–20.
- Bearden CE, Hoffman KM, Cannon TD. The neuropsychology and neuroanatomy of bipolar affective disorder: a critical review. *Bipolar Disord* 2001; **3**: 106–50.
- Kurtz MM, Gerraty RT. A meta-analytic investigation of neurocognitive deficits in bipolar illness: profile and effects of clinical state. *Neuropsychology* 2009; **23**: 551–62.
- Zakzanis KK, Leach L, Kaplan E. On the nature and pattern of neurocognitive function in major depressive disorder. *Neuropsychiatry Neuropsychol Behav Neurol* 1998; **11**: 111–9.
- Douglas KM, Porter RJ. Longitudinal assessment of neuropsychological function in major depression. *Aust N Z J Psychiatry* 2009; **43**: 1105–17.
- McClintock SM, Husain MM, Greer TL, Cullum CM. Association between depression severity and neurocognitive function in major depressive disorder: a review and synthesis. *Neuropsychology* 2010; **24**: 9–34.
- Meshulam-Gately RI, Giuliano AJ, Goff D, Faraone SV, Seidman LJ. Neurocognition in first-episode schizophrenia: a meta-analytic review. *Neuropsychology* 2009; **23**: 315–36.
- Szöke A, Trandafir A, Dupont M-E, Méary A, Schürhoff F, Leboyer M. Longitudinal studies of cognition in schizophrenia: meta-analysis. *Br J Psychiatry* 2008; **192**: 248–57.
- Carpenter WT, Heinrichs DW, Wagman AMI. Deficit and nondeficit forms of schizophrenia: the concept. *Am J Psychiatry* 1988; **145**: 578–83.
- Fenton WS, McGlashan TH. Antecedents, symptom progression, and long-term outcome of deficit syndrome schizophrenia. *Am J Psychiatry* 1994; **151**: 351–6.
- Cohen AS, Saperstein AM, Gold JM, Kirkpatrick B, Carpenter WT, Buchanan RW. Neuropsychology of the deficit syndrome: new data and meta-analysis of findings to date. *Schizophr Bull* 2007; **33**: 1201–12.
- Milev P, Ho BC, Arndt S, Andreasen NC. Predictive values of neurocognition and negative symptoms on functional outcome in schizophrenia: a longitudinal first-episode study with 7-year follow-up. *Am J Psychiatry* 2005; **162**: 495–506.
- Lin A, Wood SJ, Nelson B, Brewer WJ, Spiliotacopoulos D, Bruxner A, et al. Neurocognitive predictors of functional outcome two to 13 years after identification as ultra-high risk for psychosis. *Schizophr Res* 2011; **132**: 1–7.
- McDermott LM, Ebmeier KP. A meta-analysis of depression severity and cognitive function. *J Affect Disord* 2009; **119**: 1–8.
- Lewandowski K, Cohen B, Ongur D. Evolution of neuropsychological dysfunction during the course of schizophrenia and bipolar disorder. *Psychol Med* 2011; **41**: 225–41.
- Balanza-Martinez V, Tabares-Seisdedos R, Selva-Vera G, Martinez-Aran A, Torrent C, Salazar-Fraile J, et al. Persistent cognitive dysfunctions in bipolar I disorder and schizophrenic patients: a 3-year follow-up study. *Psychother Psychosom* 2005; **74**: 113–9.
- Martinez-Aran A, Vieta E, Reinares M, Colom F, Torrent C, Sanchez-Moreno J, et al. Cognitive function across manic or hypomanic, depressed, and euthymic states in bipolar disorder. *Am J Psychiatry* 2004; **161**: 262–70.
- Torrent C, Martínez-Aran A, Daban C, Sánchez-Moreno J, Comes M, Goikolea JM, et al. Cognitive impairment in bipolar II disorder. *Br J Psychiatry* 2006; **189**: 254–9.
- Glahn DC, Bearden CE, Barguil M, Barrett J, Reichenberg A, Bowden CL, et al. The neurocognitive signature of psychotic bipolar disorder. *Biol Psychiatry* 2007; **62**: 910–6.
- Fleming SK, Blasey C, Schatzberg AF. Neuropsychological correlates of psychotic features in major depressive disorders: a review and meta-analysis. *J Psychiatr Res* 2004; **38**: 27–35.
- Hill SK, Keshavan MS, Thase ME, Sweeney JA. Neuropsychological dysfunction in antipsychotic-naïve first-episode unipolar psychotic depression. *Am J Psychiatry* 2004; **161**: 996–1003.
- Albus M, Hubmann W, Wahlheim C, Sobizack N, Franz U, Mohr F. Contrasts in neuropsychological test profile between patients with first episode schizophrenia and first episode affective disorders. *Acta Psychiatr Scand* 1996; **94**: 87–93.
- Simonsen C, Sundet K, Vaskinn A, Birkenaes AB, Engh JA, Færden A, et al. Neurocognitive dysfunction in bipolar and schizophrenia spectrum disorders

- depends on history of psychosis rather than diagnostic group. *Schizophr Bull* 2011; **37**: 73–83.
- 30 Rund BR, Sundet K, Asbjørnsen A, Egeland J, Landro NI, Lund A, et al. Neuropsychological test profiles in schizophrenia and non-psychotic depression. *Acta Psychiatr Scand* 2006; **113**: 350–9.
 - 31 Bora E, Yucel M, Pantelis C. Cognitive functioning in schizophrenia, schizoaffective disorder and affective psychoses: meta-analytic study. *Br J Psychiatry* 2009; **195**: 475–82.
 - 32 Cannon M, Caspi A, Moffitt TE, Harrington H, Taylor A, Murray RM, et al. Evidence for early-childhood, pan-developmental impairment specific to schizophreniform disorder: results from a longitudinal birth cohort. *Arch Gen Psychiatry* 2002; **59**: 449–56.
 - 33 Cannon M, Moffitt TE, Caspi A, Murray RM, Harrington H, Poulton R. Neuropsychological performance at the age of 13 years and adult schizophreniform disorder. Prospective birth cohort study. *Br J Psychiatry* 2006; **189**: 463–4.
 - 34 David AS, Malmberg A, Brandt L, Allebeck P, Lewis G. IQ and risk for schizophrenia: a population-based cohort study. *Psychol Med* 1997; **27**: 1311–23.
 - 35 Davidson M, Reichenberg A, Rabinowitz J, Weiser M, Kaplan Z, Mark M. Behavioral and intellectual markers for schizophrenia in apparently healthy male adolescents. *Am J Psychiatry* 1999; **156**: 1328–35.
 - 36 MacCabe JH, Lambe MP, Cnattingius S, Torrang A, Bjork C, Sham PC, et al. Scholastic achievement at age 16 and risk of schizophrenia and other psychoses: a national cohort study. *Psychol Med* 2008; **38**: 1133–40.
 - 37 Reichenberg A, Weiser M, Rabinowitz J, Caspi A, Schmeidler J, Mark M, et al. A population-based cohort study of premorbid intellectual, language, and behavioral functioning in patients with schizophrenia, schizoaffective disorder, and nonpsychotic bipolar disorder. *Am J Psychiatry* 2002; **159**: 2027–35.
 - 38 Eastvold AD, Heaton RK, Cadenhead KS. Neurocognitive deficits in the (putative) prodrome and first episode of psychosis. *Schizophr Res* 2007; **93**: 266–77.
 - 39 Pukrop R, Ruhrmann S, Schultze-Lutter F, Bechdolf A, Brockhaus-Dumke A, Klosterkötter J. Neurocognitive indicators for a conversion to psychosis: comparison of patients in a potentially initial prodromal state who did or did not convert to a psychosis. *Schizophr Res* 2007; **92**: 116–25.
 - 40 Seidman LJ, Giuliano AJ, Meyer EC, Addington J, Cadenhead KS, Cannon TD, et al. Neuropsychology of the prodrome to psychosis in the NAPLS Consortium: relationship to family history and conversion to psychosis. *Arch Gen Psychiatry* 2010; **67**: 578–88.
 - 41 Woodberry KA, Seidman LJ, Giuliano AJ, Verdi MB, Cook WL, McFarlane WR. Neuropsychological profiles in individuals at clinical high risk for psychosis: relationship to psychosis and intelligence. *Schizophr Res* 2010; **123**: 188–98.
 - 42 Brewer W, Francey S, Wood S, Jackson H, Pantelis C, Phillips L, et al. Memory impairments identified in people at ultra-high risk for psychosis who later develop first-episode psychosis. *Am J Psychiatry* 2005; **162**: 71–8.
 - 43 Kim HS, Shin NY, Jang JH, Kim E, Shim G, Park HY, et al. Social cognition and neurocognition as predictors of conversion to psychosis in individuals at ultra high risk. *Schizophr Res* 2011; **130**: 170–5.
 - 44 Lencz T, Smith CW, McLaughlin D, Auther A, Nakayama E, Hovey L, et al. Generalized and specific neurocognitive deficits in prodromal schizophrenia. *Biol Psychiatry* 2006; **59**: 863–71.
 - 45 Becker HE, Nieman DH, Dingemans PM, van de Fliert JR, De Haan L, Linszen DH. Verbal fluency as a possible predictor for psychosis. *Eur Psychiatry* 2010; **25**: 105–10.
 - 46 Riecher-Rossler A, Pflueger MO, Aston J, Borgwardt SJ, Brewer WJ, Gschwandtner U, et al. Efficacy of using cognitive status in predicting psychosis: a 7-year follow-up. *Biol Psychiatry* 2009; **66**: 1023–30.
 - 47 Wood SJ, Brewer WJ, Koutsouradis P, Phillips LJ, Francey SM, Proffitt TM, et al. Cognitive decline following psychosis onset. Data from the PACE clinic. *Br J Psychiatry* 2007; **191**: s52–7.
 - 48 Hawkins KA, Keefe R, Christensen B, Addington J, Woods SW, Callahan J, et al. Neuropsychological course in the prodrome and first episode of psychosis: findings from the PRIME North American Double Blind Treatment Study. *Schizophr Res* 2008; **105**: 1–9.
 - 49 Becker HE, Nieman DH, Wiltink S, Dingemans PM, van de Fliert JR, Velthorst E, et al. Neurocognitive functioning before and after the first psychotic episode: does psychosis result in cognitive deterioration? *Psychol Med* 2010; **40**: 1599–606.
 - 50 Balanza-Martínez V, Rubio C, Selva-Vera G, Martínez-Aran A, Sánchez-Moreno J, Salazar-Fraile J, et al. Neurocognitive endophenotypes (endophenocognities) from studies of relatives of bipolar disorder subjects: a systematic review. *Neurosci Biobehav Rev* 2008; **32**: 1426–38.
 - 51 Meyer SE, Carlson GA, Wiggs EA, Martínez PE, Ronsaville DS, Klimes-Dougan B, et al. A prospective study of the association among impaired executive functioning, childhood attentional problems, and the development of bipolar disorder. *Dev Psychopathol* 2004; **16**: 461–76.
 - 52 Sigurdsson E, Fombonne E, Sayal K, Checkley S. Neurodevelopmental antecedents of early-onset bipolar affective disorder. *Br J Psychiatry* 1999; **174**: 121–7.
 - 53 Tiihonen J, Haukka J, Henriksson M, Cannon M, Kieseppä T, Laaksonen I, et al. Premorbid intellectual functioning in bipolar disorder and schizophrenia: results from a cohort study of male conscripts. *Am J Psychiatry* 2005; **162**: 1904–10.
 - 54 Zammit S, Allebeck P, David AS, Dalman C, Hemmingsson T, Lundberg I, et al. A longitudinal study of premorbid IQ score and risk of developing schizophrenia, bipolar disorder, severe depression, and other nonaffective psychoses. *Arch Gen Psychiatry* 2004; **61**: 354–60.
 - 55 Olvet DM, Stearns WH, McLaughlin D, Auther AM, Correll CU, Cornblatt BA. Comparing clinical and neurocognitive features of the schizophrenia prodrome to the bipolar prodrome. *Schizophr Res* 2010; **123**: 59–63.
 - 56 Nehra R, Chakrabarti S, Pradhan BK, Khehra N. Comparison of cognitive functions between first- and multi-episode bipolar affective disorders. *J Affect Disord* 2006; **93**: 185–92.
 - 57 Gruber SA, Rosso IM, Yurgelun-Todd D. Neuropsychological performance predicts clinical recovery in bipolar patients. *J Affect Disord* 2008; **105**: 253–60.
 - 58 Seidman LJ, Giuliano AJ, Smith CW, Stone WS, Glatt SJ, Meyer E, et al. Neuropsychological functioning in adolescents and young adults at genetic risk for schizophrenia and affective psychoses: results from the Harvard and Hillside Adolescent High Risk Studies. *Schizophr Bull* 2006; **32**: 507–24.
 - 59 Klimes-Dougan B, Ronsaville D, Wiggs EA, Martínez PE. Neuropsychological functioning in adolescent children of mothers with a history of bipolar or major depressive disorders. *Biol Psychiatry* 2006; **60**: 957–65.
 - 60 Micco JA, Henin A, Biederman J, Rosenbaum JF, Petty C, Rindlaub LA, et al. Executive functioning in offspring at risk for depression and anxiety. *Depress Anxiety* 2009; **26**: 780–90.
 - 61 Palmer B, Heaton R, Paulsen JS, Kuck J, Braff D, Harris MJ. Is it possible to be schizophrenic yet neuropsychologically normal? *Neuropsychology* 1997; **11**: 437–46.
 - 62 Iverson GL, Brooks BL, Langenecker SA, Young AH. Identifying a cognitive impairment subgroup in adults with mood disorders. *J Affect Disord* 2011; **132**: 360–7.
 - 63 Martino DJ, Strejilevich SA, Scapola M, Igoa A, Marengo E, Ais ED, et al. Heterogeneity in cognitive functioning among patients with bipolar disorder. *J Affect Disord* 2008; **109**: 149–56.
 - 64 Fornito A, Yoon J, Zalesky A, Bullmore ET, Carter CS. General and specific functional connectivity disturbances in first-episode schizophrenia during cognitive control performance. *Biol Psychiatry* 2011; **70**: 64–72.
 - 65 Oertel-Knochel V, Bittner RA, Knochel C, Prvulovic D, Hampel H. Discovery and development of integrative biological markers for schizophrenia. *Progr Neurobiol* 2011; **95**: 686–702.
 - 66 Hetrick S, Parker A, Hickie I, Purcell R, Yung A, McGorry P. Early identification and intervention in depressive disorders: towards a clinical staging model. *Psychother Psychosom* 2008; **77**: 263–70.
 - 67 Kapczynski F, Dias VV, Kauer-Sant'Anna M, Brietzke E, Vazquez GH, Vieta E, et al. The potential use of biomarkers as an adjunctive tool for staging bipolar disorder. *Progr Neuropsychopharmacol Biol Psychiatry* 2009; **33**: 1366–71.
 - 68 Kauer-Sant'Anna M, Kapczynski F, Andreazza AC, Bond DJ, Lam RW, Young LT, et al. Brain-derived neurotrophic factor and inflammatory markers in patients with early- vs. late-stage bipolar disorder. *Int J Neuropsychopharmacol* 2009; **12**: 447–58.
 - 69 Fernandes BS, Gama CS, Maria Cereser K, Yatham LN, Fries GR, Colpo G, et al. Brain-derived neurotrophic factor as a state-marker of mood episodes in bipolar disorders: a systematic review and meta-regression analysis. *J Psychiatr Res* 2011; **45**: 995–1004.
 - 70 Fusar-Poli P, Borgwardt S, Crescini A, Deste G, Kempton MJ, Lawrie S, et al. Neuroanatomy of vulnerability to psychosis: a voxel-based meta-analysis. *Neurosci Biobehav Rev* 2011; **35**: 1175–85.
 - 71 Dazzan P, Soulsby B, Mechelli A, Wood SJ, Velakoulis D, Phillips LJ, et al. Volumetric abnormalities predating the onset of schizophrenia and affective psychoses: an MRI study in subjects at ultrahigh risk of psychosis. *Schizophr Bull* 2012; **38**: 1083–91.
 - 72 Fornito A, Yucel M, Patti J, Wood SJ, Pantelis C. Mapping grey matter reductions in schizophrenia: an anatomical likelihood estimation analysis of voxel-based morphometry studies. *Schizophr Res* 2009; **108**: 104–13.
 - 73 Ellison-Wright I, Glahn DC, Laird AR, Thelen SM, Bullmore E. The anatomy of first-episode and chronic schizophrenia: an anatomical likelihood estimation meta-analysis. *Am J Psychiatry* 2008; **165**: 1015–23.

- 74 Chan RC, Di X, McAlonan GM, Gong QY. Brain anatomical abnormalities in high-risk individuals, first-episode, and chronic schizophrenia: an activation likelihood estimation meta-analysis of illness progression. *Schizophr Bull* 2011; **37**: 177–88.
- 75 Premkumar P, Kumari V, Corr PJ, Sharma T. Frontal lobe volumes in schizophrenia: effects of stage and duration of illness. *J Psychiatr Res* 2006; **40**: 627–37.
- 76 Mitelman SA, Nikiforova YK, Canfield EL, Hazlett EA, Brickman AM, Shihabuddin L, et al. A longitudinal study of the corpus callosum in chronic schizophrenia. *Schizophr Res* 2009; **114**: 144–53.
- 77 Velakoulis D, Wood SJ, Wong MT, McGorry PD, Yung A, Phillips L, et al. Hippocampal and amygdala volumes according to psychosis stage and diagnosis: a magnetic resonance imaging study of chronic schizophrenia, first-episode psychosis, and ultra-high-risk individuals. *Arch Gen Psychiatry* 2006; **63**: 139–49.
- 78 Jung WH, Kim JS, Jang JH, Choi JS, Jung MH, Park JY, et al. Cortical thickness reduction in individuals at ultra-high-risk for psychosis. *Schizophr Bull* 2011; **37**: 839–49.
- 79 Olabi B, Ellison-Wright I, McIntosh AM, Wood SJ, Bullmore E, Lawrie SM. Are there progressive brain changes in schizophrenia? A meta-analysis of structural magnetic resonance imaging studies. *Biol Psychiatry* 2011; **70**: 88–96.
- 80 Jarnum H, Eskildsen SF, Steffensen EG, Lundbye-Christensen S, Simonsen CW, Thomsen IS, et al. Longitudinal MRI study of cortical thickness, perfusion, and metabolite levels in major depressive disorder. *Acta Psychiatr Scand* 2011; **124**: 435–46.
- 81 Pillay SS, Yurgelun-Todd DA, Bonello CM, Lafer B, Fava M, Renshaw PF. A quantitative magnetic resonance imaging study of cerebral and cerebellar gray matter volume in primary unipolar major depression: relationship to treatment response and clinical severity. *Biol Psychiatry* 1997; **42**: 79–84.
- 82 Pillay SS, Renshaw PF, Bonello CM, Lafer BC, Fava M, Yurgelun-Todd D. A quantitative magnetic resonance imaging study of caudate and lenticular nucleus gray matter volume in primary unipolar major depression: relationship to treatment response and clinical severity. *Psychiatr Res* 1998; **84**: 61–74.
- 83 Lacerda AL, Nicoletti MA, Brambilla P, Sassi RB, Mallinger AG, Frank E, et al. Anatomical MRI study of basal ganglia in major depressive disorder. *Psychiatr Res* 2003; **124**: 129–40.
- 84 Portella MJ, de Diego-Adelino J, Gomez-Anson B, Morgan-Ferrando R, Vives Y, Puigdemont D, et al. Ventromedial prefrontal spectroscopic abnormalities over the course of depression: a comparison among first episode, remitted recurrent and chronic patients. *J Psychiatr Res* 2011; **45**: 427–34.
- 85 Sheline YI, Wang PW, Gado MH, Csernansky JG, Vannier MW. Hippocampal atrophy in recurrent major depression. *Proc Natl Acad Sci USA* 1996; **93**: 3908–13.
- 86 Sheline YI. 3D MRI studies of neuroanatomic changes in unipolar major depression: the role of stress and medical comorbidity. *Biol Psychiatry* 2000; **48**: 791–800.
- 87 MacQueen GM, Campbell S, McEwen BS, Macdonald K, Amano S, Joffe RT, et al. Course of illness, hippocampal function, and hippocampal volume in major depression. *Proc Natl Acad Sci USA* 2003; **100**: 1387–92.
- 88 Kempton MJ, Salvador Z, Munafo MR, Geddes JR, Simmons A, Frangou S, et al. Structural neuroimaging studies in major depressive disorder: meta-analysis and comparison with bipolar disorder. *Arch Gen Psychiatry* 2011; **68**: 675–90.
- 89 Strakowski SM, DelBello MP, Zimmerman ME, Getz GE, Mills NP, Ret J, et al. Ventricular and periventricular structural volumes in first- versus multiple-episode bipolar disorder. *Am J Psychiatry* 2002; **159**: 1841–7.
- 90 Brambilla P, Harenski K, Nicoletti M, Mallinger AG, Frank E, Kupfer DJ, et al. MRI study of posterior fossa structures and brain ventricles in bipolar patients. *J Psychiatr Res* 2001; **35**: 313–22.
- 91 DelBello MP, Strakowski SM, Zimmerman ME, Hawkins JM, Sax KW. MRI analysis of the cerebellum in bipolar disorder: a pilot study. *Neuropsychopharmacology* 1999; **21**: 63–8.
- 92 Moorhead TW, McKirdy J, Sussmann JE, Hall J, Lawrie SM, Johnstone EC, et al. Progressive gray matter loss in patients with bipolar disorder. *Biol Psychiatry* 2007; **62**: 894–900.
- 93 Arnone D, Cavanagh J, Gerber D, Lawrie SM, Ebmeier KP, McIntosh AM. Magnetic resonance imaging studies in bipolar disorder and schizophrenia: meta-analysis. *Br J Psychiatry* 2009; **195**: 194–201.
- 94 Elkins H, Friedman L, Wise A, Meltzer HY. Meta-analyses of studies of ventricular enlargement and cortical sulcal prominence in mood disorders. Comparisons with controls or patients with schizophrenia. *Arch Gen Psychiatry* 1995; **52**: 735–46.
- 95 Altshuler LL, Bartzokis G, Grieder T, Curran J, Jimenez T, Leight K, et al. An MRI study of temporal lobe structures in men with bipolar disorder or schizophrenia. *Biol Psychiatry* 2000; **48**: 147–62.
- 96 McDonald C, Bullmore E, Sham P, Chitnis X, Suckling J, MacCabe J, et al. Regional volume deviations of brain structure in schizophrenia and psychotic bipolar disorder. Computational morphometry study. *Br J Psychiatry* 2005; **186**: 369–77.
- 97 Sui J, Pearlson G, Caprihan A, Adali T, Kiehl KA, Liu J, et al. Discriminating schizophrenia and bipolar disorder by fusing fMRI and DTI in a multimodal CCA+ joint ICA model. *Neuroimage* 2011; **57**: 839–55.
- 98 Strakowski SM, Adler CM, DelBello MP. Volumetric MRI studies of mood disorders: do they distinguish unipolar and bipolar disorder? *Bipolar Disord* 2002; **4**: 80–8.
- 99 Videbech P, Ravnkilde B. Hippocampal volume and depression: a meta-analysis of MRI studies. *Am J Psychiatry* 2004; **161**: 1957–66.
- 100 Pettersson-Yeo W, Allen P, Benetti S, McGuire P, Mechelli A. Dysconnectivity in schizophrenia: where are we now? *Neurosci Biobehav Rev* 2011; **35**: 1110–24.
- 101 Friston KJ. The disconnection hypothesis. *Schizophr Res* 1998; **30**: 115–25.
- 102 Kang SS, Sponheim SR, Chafee MV, Macdonald AW. Disrupted functional connectivity for controlled visual processing as a basis for impaired spatial working memory in schizophrenia. *Neuropsychologia* 2011; **49**: 2836–47.
- 103 Rotarska-Jagiela A, van de Ven V, Oertel-Knochel V, Uhlhaas PJ, Vogeley K, Linden DE. Resting-state functional network correlates of psychotic symptoms in schizophrenia. *Schizophr Res* 2010; **117**: 21–30.
- 104 Patel S, Mahon K, Wellington R, Zhang J, Chaplin W, Szeszko PR. A meta-analysis of diffusion tensor imaging studies of the corpus callosum in schizophrenia. *Schizophr Res* 2011; **129**: 149–55.
- 105 Whitfield-Gabrieli S, Thermenos HW, Milanovic S, Tsuang MT, Faraone SV, McCarley RW, et al. Hyperactivity and hyperconnectivity of the default network in schizophrenia and in first-degree relatives of persons with schizophrenia. *Proc Natl Acad Sci USA* 2009; **106**: 1279–84.
- 106 Woodward ND, Rogers B, Heckers S. Functional resting-state networks are differentially affected in schizophrenia. *Schizophr Res* 2011; **130**: 86–93.
- 107 Hasler G, Northoff G. Discovering imaging endophenotypes for major depression. *Molec Psychiatry* 2011; **16**: 604–19.
- 108 Hamilton JP, Furman DJ, Chang C, Thomason ME, Dennis E, Gotlib IH. Default-mode and task-positive network activity in major depressive disorder: implications for adaptive and maladaptive rumination. *Biol Psychiatry* 2011; **70**: 327–33.
- 109 Zhang J, Wang J, Wu Q, Kuang W, Huang X, He Y, et al. Disrupted brain connectivity networks in drug-naïve, first-episode major depressive disorder. *Biol Psychiatry* 2011; **70**: 334–42.
- 110 Ongur D, Lundy M, Greenhouse I, Shinn AK, Menon V, Cohen BM, et al. Default mode network abnormalities in bipolar disorder and schizophrenia. *Psychiatry* 2010; **183**: 59–68.
- 111 Cole MW, Anticevic A, Repovs G, Barch D. Variable global dysconnectivity and individual differences in schizophrenia. *Biol Psychiatry* 2011; **70**: 43–50.
- 112 Kapczynski F, Dias VV, Kauer-Sant'Anna M, Frey BN, Grassi-Oliveira R, Colom F, et al. Clinical implications of a staging model for bipolar disorders. *Exp Rev Neurotherap* 2009; **9**: 957–66.
- 113 Savitz J, Drevets WC. Bipolar and major depressive disorder: neuroimaging the developmental-degenerative divide. *Neurosci Biobehav Rev* 2009; **33**: 699–771.
- 114 Foti DJ, Kotov R, Guey LT, Bromet EJ. Cannabis use and the course of schizophrenia: 10-year follow-up after first hospitalization. *Am J Psychiatry* 2010; **167**: 987–93.
- 115 McCabe C, Mishor Z. Antidepressant medications reduce subcortical-cortical resting-state functional connectivity in healthy volunteers. *Neuroimage* 2011; **57**: 1317–23.
- 116 Ho BC, Andreasen NC, Ziebell S, Pierson R, Magnotta V. Long-term antipsychotic treatment and brain volumes: a longitudinal study of first-episode schizophrenia. *Arch Gen Psychiatry* 2011; **68**: 128–37.
- 117 Phillips ML, Travis MJ, Fagioli A, Kupfer DJ. Medication effects in neuroimaging studies of bipolar disorder. *Am J Psychiatry* 2008; **165**: 313–20.
- 118 Yatham LN, Kapczynski F, Andreazza AC, Trevor Young L, Lam RW, Kauer-Sant'anna M. Accelerated age-related decrease in brain-derived neurotrophic factor levels in bipolar disorder. *Int J Neuropsychopharmacol* 2009; **12**: 137–9.

- 119 Chakos MH, Lieberman JA, Bilder RM, Borenstein M, Lerner G, Bogerts B, et al. Increase in caudate nuclei volumes of first-episode schizophrenic patients taking antipsychotic drugs. *Am J Psychiatry* 1994; **151**: 1430–6.
- 120 Owen MJ, O'Donovan MC, Thapar A, Craddock N. Neurodevelopmental hypothesis of schizophrenia. *Br J Psychiatry* 2011; **198**: 173–5.
- 121 Altamura AC, Serati M, Albano A, Paoli RA, Glick ID, Dell'Osso B. An epidemiologic and clinical overview of medical and psychopathological comorbidities in major psychoses. *Eur Arch Psychiatry Clin Neurosci* 2011; **261**: 489–507.
- 122 Payne BR, Lomber SG. Reconstructing functional systems after lesions of cerebral cortex. *Nat Rev Neurosci* 2001; **2**: 911–9.
- 123 Castren E, Rantamaki T. The role of BDNF and its receptors in depression and antidepressant drug action: reactivation of developmental plasticity. *Dev Neurobiol* 2010; **70**: 289–97.
- 124 Schaufelberger MS, Lappin JM, Duran FL, Rosa PG, Uchida RR, Santos LC, et al. Lack of progression of brain abnormalities in first-episode psychosis: a longitudinal magnetic resonance imaging study. *Psychol Med* 2011; **41**: 1677–89.
- 125 Gogtay N. Cortical brain development in schizophrenia: insights from neuroimaging studies in childhood-onset schizophrenia. *Schizophr Bull* 2008; **34**: 30–6.

Preventing depression and promoting resilience: feasibility study of a school-based cognitive–behavioural intervention

Paul Stallard and Rhiannon Buck

Background

The limited reach and effectiveness of psychological treatments for adolescent depression have fuelled interest in alternative approaches designed to promote resilience. Schools offer a convenient location for the widespread delivery of depression prevention programmes, although little research has evaluated the feasibility of delivering interventions in this setting.

Aims

To investigate the feasibility of delivering and evaluating a universal school-based depression prevention programme for children aged 12–16 years.

Method

A three-arm pilot study was conducted in one UK secondary school ($n = 834$).

Results

Interventions had good reach (96%), with high rates of consent (89%) and reasonable retention (78%). The majority of intervention sessions were delivered as intended, with 85% of students attending seven or more sessions. The programme was acceptable to students and teachers, with the specific content of the active intervention being rated differently from the control programmes.

Conclusions

Delivering and undertaking methodologically robust evaluations of universal school-based depression programmes is feasible.

Declaration of interest

None.

The prevalence of depressive disorders in the UK in children (under 13 years old) and adolescents (aged 13–18 years) has been estimated at 2.8% and 5.6% respectively.¹ These disorders have a significant adverse impact on school, social and family functioning and increase the risk of suicide and substance misuse in young adulthood.^{1–4} Depressive disorders persist over time and there is continuity between adolescent depression and depressive disorders in young adults.⁵ Relapse is common, with up to 70% of adolescents with depression experiencing a recurrent depressive episode within 5 years.^{6,7} Randomised controlled trials have demonstrated that effective psychological interventions are available for the treatment of depression in adolescents, at least in the short term.^{8–10} Although this is encouraging, the majority of adolescents with depression remain unidentified and untreated.^{11,12} The limited reach and effectiveness of current treatment programmes have led researchers to investigate whether depressive disorders can be prevented through the widespread provision of prevention programmes.

Prevention programmes tend to be conceptualised by their intended focus, either universal (provided to whole populations regardless of risk status) or targeted (e.g. provided to those at increased risk of developing depression). Universal programmes tend to be less stigmatising and have good reach, whereas targeted approaches tend to produce larger treatment effects and from a public health perspective may represent a better use of limited resources.¹³ For adolescents, schools provide a natural and convenient location for the delivery of mental health prevention programmes. Recognition of the potential role of schools in promoting mental health has been emphasised in recent UK government initiatives such as Targeted Mental Health in Schools and Social and Emotional Aspects of Learning.^{14,15}

Although schools offer a potentially convenient way of accessing large numbers of young people, the effects of mental health programmes delivered in such settings have not always been

positive.^{16–18} Variations between studies have been investigated in systematic reviews which have highlighted a number of issues. First, in terms of delivery, targeted depression prevention programmes tend to produce larger post-treatment effects than universal programmes.^{19,20} However, practically targeted programmes may prove more difficult to provide, since individual students need to be identified and additional arrangements made within the school to deliver the intervention. This might be difficult for busy secondary schools with limited space, which typically organise and plan timetables around year groups and classes rather than individual students. Second, sufficient time needs to be made available to deliver depression prevention programmes, which usually require 8–16 sessions.^{20,21} Finding sufficient dedicated time within an already full timetable can be a practical problem that might prohibit their use in schools. Third, the majority of depression prevention programmes are based on cognitive–behavioural therapy (CBT) and tend to be more effective when delivered by mental health practitioners rather than trained school staff.²⁰ Although programmes are more likely to be sustainable if delivered by educational staff, teachers might not necessarily feel sufficiently skilled or knowledgeable about CBT or comfortable talking about mental health issues. However, if programmes are externally provided then school and classroom staff need to be supportive of their delivery. Fourth, undertaking robust research evaluations of prevention programmes in schools is complicated and many existing studies suffer from significant methodological weakness.^{13,19,22} In order for results to be meaningful, school-based studies need to achieve good recruitment and retention rates, and assessments need to be acceptable and easily completed. Finally, the identification of appropriate comparison groups is an important issue for school trials.²² Comparison groups need to be appropriate and acceptable to the school. In addition they need to be matched for any possible non-specific elements such as increased attention

and assessment completion, and assessed to ensure that the content of the programmes delivered to the intervention and comparison groups are sufficiently different.

The aim of this study was to examine the feasibility and acceptability of delivering and evaluating a depression prevention programme for adolescents within the UK educational context.

Method

Promoting Mental Health in Schools through Education (PROMISE) is a randomised controlled trial evaluating the effectiveness of a school-based depression programme for young people aged 12–16 years.²³ To maximise fit with schools and minimise timetabling problems the programme was universally provided to whole classes of young people. However, the focus of the evaluation of the effectiveness of the programme was on students who had persistent and elevated levels of depressive symptoms, defined as a score of 5 or over on the Short Mood and Feelings Questionnaire (SMFQ),²⁴ completed on two occasions 2 weeks apart.

Ethical approval and consent

The study was approved by the University of Bath ethical committee with consent/assent involving three stages. First, interested schools were required to opt into the study. Second, parents or carers of all students in years 8–11 (aged 12–16 years) on the school roll were sent a project information sheet and invited to return an opt-out form if they did not wish their child to complete the project assessments. Finally, young people were required to sign a consent form before completing assessment questionnaires.

Recruitment

Information about the project was sent to 66 non-denominational comprehensive secondary schools in Bath and north-east Somerset, Bristol, Wiltshire, Nottingham and Nottinghamshire. Nine schools were recruited, one for the pilot study and eight for the main trial.

Intervention

The PROMISE study is a randomised controlled trial with the following three trial arms.

Resourceful Adolescent Programme

The Resourceful Adolescent Programme (RAP) is a depression prevention programme based on CBT designed to be delivered to whole classes of young people aged 12–15 years. It has been subjected to evaluations in Australia, New Zealand and Mauritius.^{25–28} The original 11-session programme was adapted for use in the UK educational system and consisted of 9 sessions facilitating the development of skills in six main areas. First, the participants were encouraged to identify and recognise their personal strengths and the importance of maintaining good self-esteem and positive mood. The second area focused on cognitions and encouraged adolescents to identify, check and challenge unhelpful cognitions and to replace them with more balanced, enabling and helpful ways of thinking. Emotional management was the third area, facilitating emotional recognition and the development of emotional management strategies. The fourth focused on the development of problem-solving skills and the fifth on identifying support networks to draw upon to help with problems. The final section was concerned with keeping the peace

and how to use these skills to resolve interpersonal problems and to promote harmony. The programme involved a mix of large-group discussion, role play and small-group exercises, and each young person was given a workbook summarising key issues and messages.

The sessions were led by two facilitators working alongside the class teacher. Facilitators had at least an undergraduate university degree in a relevant discipline and all had experience of working with young people. All received initial training in the cognitive model of depression and RAP and attended ongoing supervision sessions.

Attention control group

As part of the national curriculum schools provide personal, social and health education (PSHE). The curriculum covers a range of topics relating to citizenship and psychological well-being, including drug and sexual education, human rights, diversity, difference and discrimination. The class teacher leads the sessions and in this trial was supported by two facilitators. The group was matched for time (9 sessions) and adult contact with the RAP group.

Usual PSHE

Young people participated in the usual PSHE sessions provided by the school (i.e. treatment as usual). The sessions were provided solely by the teachers.

Primary outcome

The primary outcome was change in symptoms of low mood at 12 months as assessed with the SMFQ, a 13-item measure of symptoms of low mood and depression.²⁴

Other measures

In order to assess whether the contents of RAP and usual PSHE were different, lesson facilitators were asked independently to assess the content of each session on a 5-point scale ranging from 'not at all' (0) to 'a lot' (4). At the conclusion of the programme, feedback concerning RAP was obtained from students by means of individual semi-structured interviews and from teachers through focus group discussion.

Results

The feasibility study was conducted in one mixed-gender, non-denominational secondary school. The students' educational attainment, eligibility for free school meals and absence rates were comparable to the national average (Table 1); however, fewer children were identified with special educational needs or from minority ethnic backgrounds.

Recruitment and retention

In terms of eligibility, 801 (96%) students on the school roll were attending school and were therefore able to participate in the study (Fig. 1). The consent process appeared acceptable, with dual parent and young person consent to complete the assessment measures being obtained for 713 (89%) students. Both screening and baseline assessments were completed by 624 (88%) of those who consented, of whom 552 (88%) completed the 6-month assessment and 489 (78%) completed the final 12-month assessment. Twelve-month retention rates in years 8 (91%), 9 (90%) and 10 (83%) were good, but there was a particular problem with year 11 (45%); these students had completed their

Table 1 Pilot school demographic summary		
	Proportion of pupils, %	
	Pilot school	National rate ^a
Pupils with SEN statements or supported on School Action Plus	5.4	9.3
Pupils at end of Key Stage 4 achieving level 2 English and maths	57	54
Pupils achieving 5 or more A*–C grade GCSEs including English and maths	57	50
Pupils eligible for free school meals	8.5	7.3
Overall pupil absence rate	6.9	7.3
Persistent absence rate	5.3	5.0
Ethnicity: BME	9	18
BME, Black and minority ethnic; GCSE, General Certificate of Secondary Education; SEN, special educational needs. a. Rates for England and Wales.		

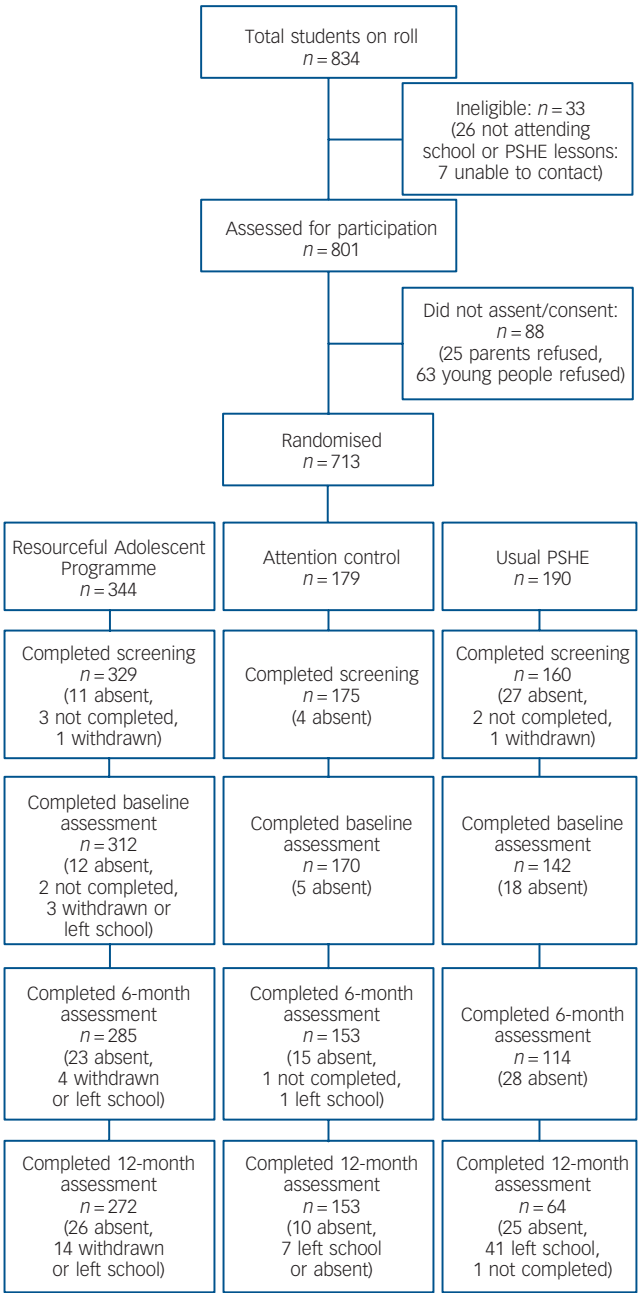


Fig. 1 Study profile. PSHE, personal, social and health education.

General Certificate of Secondary Education (GCSE) examinations and many had left school to transfer to other colleges or start work.

Outcome measures

Item completion on the SMFQ (the primary outcome measure) is summarised in Table 2 and highlights that completion was easy, with few missing data. Students were categorised as being at risk of developing a depressive disorder if they had scores of 5 or more on the SMFQ at both screening and baseline assessments (i.e. continuity of symptoms). A total of 191 (31%) students who completed the SMFQ on both occasions were classified as being at high risk. Of these, 138 (72%) were reassessed at 12 months.

Symptom change

The study was not powered to assess between-group differences on the primary outcome measure (SMFQ). Descriptive statistics are therefore presented in Table 3 for students at risk of depression in each trial arm at each assessment point. There was a decrease in SMFQ scores in all groups from screening and baseline assessment to 12 months.

Intervention delivery

The intervention was provided to students in years 8 and 10. All 9 RAP sessions were delivered to 15 classes, with the remaining class receiving 8 sessions. A total of 137 RAP sessions (95%) were delivered as intended by two facilitators, with the other 7 sessions being led by one. Seven sessions were unexpectedly cancelled owing to adverse weather (n=2), early school closure (n=1), bank holidays (n=1), examinations (n=1), a school project day (n=1) and PSHE being cancelled (n=1).

Session attendance

Of the 409 eligible children in years 8 and 10, only 9 (2%) failed to attend any RAP session. Of these children 5 were no longer at school, either having been expelled (n=2) or moving school (n=3) before the sessions started. Approximately half (n=188) attended all nine sessions, with 357 (87%) attending seven or more sessions.

Table 2 Short Mood and Feelings Questionnaire completion rates			
	RAP (n = 344)	Attention control (n = 179)	Usual PSHE (n = 190)
Screening			
All items complete	326	171	15
One or more missing	2	4	3
Students absent	16	4	30
Baseline			
All items complete	311	172	169
One or more missing	7	1	0
Students absent	26	6	21
6 months			
All items complete	301	158	141
One or more missing	5	0	0
Students absent	38	21	49
12 months			
All items complete	291	157	75
One or more missing	1	3	0
Students absent	52	19	115
PSHE, personal, social and health education; RAP, Resourceful Adolescent Programme.			

Overlap between RAP and usual PSHE

Assessment of the contents of RAP and usual PSHE by lesson facilitators on a 5-point scale showed significant between-group differences on most variables (Table 4). The RAP facilitators rated the coverage of self-esteem, emotional awareness and positive thinking significantly higher compared with the PSHE facilitators, who gave highest ratings to the coverage of topics traditionally covered in this subject (bullying, smoking, drugs, alcohol, sex education, ethical issues, diversity, religion and citizenship). There was no significant difference between the groups in the specific focus on depression, although RAP facilitators rated the direct focus on mental health more highly.

Acceptability of RAP

Student feedback

Individual semi-structured interviews were undertaken with nine year 8 and ten year 10 students who took part in RAP. Overall feedback was supportive, with students liking the programme content, positive focus and the way in which the individual sessions built upon each other. A year 10 student commented:

'I'm sort of a negative person but it made me realise what maybe I need to improve things.'

Another reported:

'I'm quite negative so it's made me think about maybe sort of changing how I think.'

Similar comments were reported by year 8 students, for example:

'It made people think a bit more about how they could help themselves when they've been sad.'

The accompanying workbook was liked by most younger students. For example:

'The layout was good and the design fantastic.' (year 8 student)

Some older students thought that it was pitched at a younger level:

'I think it might have been a little childish because of some of the animations.' (year 10 student)

Some students expressed a preference for more activities, role plays and discussions:

'Like we did the role playing stuff to get everybody involved and contributing.' (year 8 student)

The video clips were seen as outdated and unclear:

'They were helpful but just a bit old.' (year 10 student)

The sessions that students found most helpful were those focusing on problem-solving, emotional recognition, the connection between thoughts and feelings, thought checking and relaxation. Those that focused on identifying and changing unhelpful thoughts were seen as repetitive and the support network session was considered by some to be too long:

'I thought it was a good message but they shouldn't have taken a whole lesson to do it.' (year 8 student)

Teacher feedback

A focus group was undertaken with the eight teachers whose classes received RAP. Initially teachers were concerned about addressing mental health in a group but by the end of the programme felt reassured:

	SMFQ score, mean (s.d.)			
	Screening	Baseline	6 months	12 months
RAP group	11.89 (5.31) (<i>n</i> = 93)	11.00 (4.96) (<i>n</i> = 93)	9.86 (6.46) (<i>n</i> = 86)	9.03 (7.03) (<i>n</i> = 78)
Attention control group	11.88 (5.77) (<i>n</i> = 48)	12.22 (6.26) (<i>n</i> = 48)	12.13 (6.26) (<i>n</i> = 39)	10.32 (6.39) (<i>n</i> = 40)
Usual PSHE group	10.40 (4.48) (<i>n</i> = 50)	10.66 (4.89) (<i>n</i> = 50)	8.24 (4.79) (<i>n</i> = 34)	9.05 (6.03) (<i>n</i> = 20)
PSHE, personal, social and health education; RAP, Resourceful Adolescent Programme.				

	RAP ^a		Attention control ^a		Difference in mean scores (95% CI)
	<i>n</i>	Mean (s.d.)	<i>n</i>	Mean (s.d.)	
How much were interpersonal relationships covered in this lesson?	279	2.83 (1.18)	128	2.02 (1.45)	0.81 (0.54 to 1.07)
How much was bullying covered in this lesson?	278	0.38 (0.66)	128	0.97 (1.38)	−0.59 (−0.79 to −0.39)
How much was self-esteem covered in this lesson?	279	1.59 (1.37)	128	0.88 (1.11)	0.71 (0.45 to 0.99)
How much were feelings/emotions covered in this lesson?	279	3.22 (0.95)	128	1.20 (1.21)	2.02 (1.80 to 2.24)
How much was smoking covered in this lesson?	279	0.12 (0.38)	128	0.81 (1.42)	−0.69 (−0.87 to −0.51)
How much were drugs covered in this lesson?	279	0.19 (0.51)	128	1.40 (1.82)	−1.21 (−1.44 to −0.97)
How much was alcohol covered in this lesson?	279	0.25 (0.52)	128	0.91 (1.40)	−0.66 (−0.85 to −0.48)
How much were sex and/or contraception covered in this lesson?	279	0.18 (0.49)	128	0.91 (1.61)	−0.73 (−0.93 to −0.52)
How much were ethical issues covered in this lesson?	279	0.31 (0.84)	128	0.54 (1.15)	−0.23 (−0.43 to −0.28)
How much were green issues covered in this lesson?	279	0.01 (0.12)	128	0.00 (0.00)	0.01 (−0.01 to 0.03)
How much were diversity and ethnicity covered in this lesson?	278	0.18 (0.54)	128	0.88 (1.58)	−0.70 (−0.90 to −0.48)
How much was religion covered in this lesson?	278	0.07 (0.25)	128	0.72 (1.39)	−0.65 (−0.82 to −0.48)
How much was problem-solving covered in this lesson?	278	2.67 (1.22)	128	1.65 (1.17)	1.02 (0.77 to 1.28)
How much was thinking in positive ways covered in this lesson?	278	2.92 (1.07)	172	0.98 (0.96)	1.94 (1.73 to 2.17)
How much was citizenship covered in this lesson?	279	0.16 (0.48)	128	1.17 (1.27)	−1.01 (−1.18 to −0.84)
How much was depression covered in this lesson?	279	0.83 (0.98)	128	0.72 (1.08)	0.11 (−0.10 to 0.33)
Overall, how engaged were students with this session?	279	3.06 (0.86)	128	2.30 (1.22)	0.76 (0.54 to 0.97)
How much did this session directly focus upon mental health issues?	279	2.27 (0.99)	128	0.69 (0.89)	1.58 (1.38 to 1.78)
a. Scored on a 5-point scale (0, not at all; 4, a lot).					

'I thought it was brilliant to be honest. I really enjoyed it. I mean I must admit I – we – sort of had a bit of conversation a few months back. I had a few concerns really. Probably from the lack of my understanding perhaps more than anything.'

The teachers were positive about the programme facilitators leading the sessions and the way in which assessment were conducted. They felt the concepts in the programme were memorable for themselves as well as for the students:

'I don't know about you but I find myself going home thinking this is self-talk, I'm falling into a negative thinking trap [laughter]. You do find yourself saying "I'm snowballing", but you know they really latched onto those key words.'

'I thought my year eights weren't engaged at all – I've got some interesting characters – and then the last session that I had they did a recap of the whole thing and someone in that group could remember every single part [of the programme].'

It was felt that the benefits of the programme might not necessarily be obvious immediately, but that the skills students acquired could be useful as and when they encountered problems in their lives:

'I think a lot of what's in here actually the students wouldn't have been conscious of absorbing it until they need it.'

Teachers liked the content of the programme but at times felt it was pitched more towards the younger students (year 8) and may not have stretched the most able students:

'I think for some of our brighter students [the workbook] would almost be slightly patronising.'

Teachers also raised concerns about the ability of less able students to engage with RAP:

'Although they remembered some of the concepts, the lessons seemed very similar to them and actually they weren't able to separate [the concepts] in their mind because they weren't some of the more able students. They weren't able to separate, you know, the different kind of techniques they were being given.'

Disruptive student behaviour was also a major issue, particularly if students became disengaged (e.g. with some of the older video clips) and where there was a large amount of group discussion involved for students who were not used to learning in this way. In classes where disruptive behaviour was a problem, moving between small-group and whole-class activities was difficult to manage. The ability of facilitators to manage student behaviour came to light as a salient issue during this feasibility study. The additional support from external staff was viewed as essential, particularly with regard to working with large classes and being able to manage the small group activities:

'If I'm being honest about whether this would work as it stands as a programme, without the support that we've had it wouldn't. I don't think it would be possible in a class of twenty-five plus to run the kind of discussions that we've needed to run the programme.'

In terms of delivery the teachers felt that the sessions were sometimes repetitive, and they had many ideas about how sessions could be more interactive and engaging, such as making the graphics in the workbooks more age-appropriate, updating some of the materials (particularly the video clips), and using more practical tasks in addition to the discussions.

Discussion

Schools offer a convenient location for the widespread dissemination of mental health prevention programmes for children and adolescents. However, although schools provide a natural focus for prevention, little attention has been paid to the feasibility of delivering such interventions within educational settings and whether methodologically robust evaluations are possible within this context. This feasibility study has demonstrated that the delivery and evaluation of a school-based depression prevention programme is practical within the UK educational context. In the school studied, 96% of students on roll were actually attending school and able to access and potentially benefit from the interventions. The complete nine-session Resourceful Adolescent Programme was delivered to all but one class with 95% of sessions

being delivered as intended by two trained facilitators. Of the students who received RAP, almost 90% attended seven or more of the nine sessions. However, in this pilot study RAP was delivered to only two of the school year groups and it is unclear whether delivery and attendance rates would be similar for the other year groups. Indeed, delivering to year 11 students might be particularly problematic as the main focus for these students is on preparing for their GCSEs. Nonetheless, these results are encouraging and suggest that the majority of students in secondary school would be able to access and receive sufficient dosage from mental health prevention programmes.

Comparison groups

Providing appropriate comparison groups against which active interventions can be assessed in schools is challenging. Schools need to ensure that they deliver the national curriculum and inevitably there will be some overlap in content with more focused mental health programmes. Facilitator ratings completed at the end of each session revealed no difference between the RAP and enhanced groups in the specific focus on depression, although there were significant between-group differences in other aspects of content. The RAP is based on CBT, with facilitators rating the emphasis on emotional awareness and positive thinking significantly more highly than those facilitating the usual PSHE group. This suggests that the content of RAP and PSHE are sufficiently different and that PSHE as provided by the school is an acceptable comparator against which focused mental health prevention programmes can be compared.

Methodology

In terms of research methodology the consent process was both practical and acceptable, with consent to complete study assessments approaching 90%. Retention rates declined over time, although 78% were retained at the 12-month follow-up. Retention rates of year 11 students were the lowest (45%) as many had left school. Alternative ways of contacting older students, such as mobile telephones, email and putting the assessments online, will be considered to maximise retention in the main trial. In relation to assessment measures, there were few missing data on the primary outcome measure, suggesting that it is acceptable to students. The criteria for classifying students as 'at risk' in terms of severity and persistence of symptoms resulted in approximately 30% of students being identified. This is higher than predicted (20%) but nonetheless appears an acceptable alternative to undertaking diagnostic assessments to identify students with elevated and persistent symptoms of low mood.

Future research

The session contents and exercises will be modified in the light of the qualitative feedback to ensure that the materials are engaging, appealing and relevant to all age groups. Greater emphasis also needs to be placed on working in a school environment with whole classes and on working alongside teachers during training and supervision of facilitators.

To conclude, these results support the premise that universal depression prevention programmes delivered in schools have the potential to reach the majority of students. Delivery by external health personnel is feasible and the intervention was viewed as acceptable by students and teaching staff. There were some concerns about the developmental pitch of the materials and a particular problem in retaining year 11 students. This study has achieved good recruitment, reasonable retention and usual PSHE appear sufficiently different to RAP provide an appropriate comparator.

Further research is now required to determine the effectiveness of depression prevention programmes delivered in schools.

Paul Stallard, PhD, **Rhiannon Buck**, PhD, Department for Health, University of Bath, UK

Correspondence: Professor P. Stallard, Child and Adolescent Mental Health Research Group, 22–23 Eastwood, Department for Health, University of Bath, Bath BA2 7AY, UK. Email: p.stallard@bath.ac.uk

Funding

This study was funded by the National Institute of Health Research Health Technology Assessment (06/37/04). The views and opinions expressed therein are those of the authors and do not necessarily reflect those of the HTA programme, NIHR, NHS or the Department of Health.

References

- Costello EJ, Erkanil A, Angold A. Is there an epidemic of child or adolescent depression? *J Child Psychol Psychiatry* 2006; **47**: 1263–71.
- Birmaher B, Ryan ND, Williamson DE, Brent DA, Kaufman J, Dahl RE, et al. Childhood and adolescent depression: a review of the past 10 years. Part 1. *J Am Acad Child Adolesc Psychiatry* 1996; **35**: 1427–39.
- Lewinsohn PM, Rhode P, Seeley JR. Major depressive disorder in older adolescents: prevalence, risk factors and clinical implications. *Clin Psychol Rev* 1998; **18**: 765–94.
- Gould MS, Greenberg T, Velting DM, Shaffer D. Youth suicide risk and preventative interventions: a review of the past 10 years. *J Am Acad Child Adolesc Psychiatry* 2003; **42**: 386–405.
- Kim-Cohen J, Caspi A, Moffitt TE, Harrington H, Milne BJ, Poulton R. Prior juvenile diagnoses in adults with mental disorder developmental follow-back of a prospective-longitudinal cohort. *Arch Gen Psychiatry* 2003; **60**: 709–17.
- Kovacs M. Presentation and course of major depressive disorder during childhood and later years of the life span. *J Am Acad Child Adolesc Psychiatry* 1996; **35**: 705–15.
- Lewinsohn PM, Rhode P, Seeley JR, Klein DN, Gotlib IH. Natural course of adolescent major depressive disorder in a community sample: predictors of recurrence in young adults. *Am J Psychiatry* 2000; **157**: 1584–91.
- Goodyer I, Dubicka B, Wilkinson P, Kelvin R, Roberts C, Byford S, et al. Selective serotonin reuptake inhibitors (SSRIs) and routine specialist care with and without cognitive behaviour therapy in adolescents with major depression: randomised controlled trial. *BMJ* 2007; **335**: 142.
- March J, Silva S, Curry J, Wells K, Fairbank J, Burns B, et al. The Treatment for Adolescents with Depression Study (TADS): outcomes over 1 year of naturalistic follow-up. *Am J Psychiatry* 2009; **166**: 1141–9.
- Brent D, Emslie G, Clarke G, Wagner KD, Asarnow JR, Keller M, et al. Switching to another SSRI or to venlafaxine with or without cognitive behaviour therapy for adolescents with SSRI-resistant depression. The TORDIA randomised controlled trial. *JAMA* 2008; **299**: 901–13.
- Coyle JT, Pine DS, Charney DS, Lewis L, Nemeroff CB, Carlson GA, et al. Bottom of Form Depression and Bipolar Support Alliance consensus statement on the unmet needs in diagnosis and treatment of mood disorders in children and adolescents. *J Am Acad Child Adolesc Psychiatry* 2003; **42**: 1494–503.
- Ford T, Hamilton H, Meltzer H, Goodman R. Predictors of service use for mental health problems amongst British school children. *Child Adolesc Ment Health* 2008; **13**: 32–40.
- Giesen F, Searle A, Sawyer M. Identifying and implementing prevention programmes for childhood mental health problems. *J Paediatr Child Health* 2007; **43**: 785–9.
- Department for Children, Schools and Families. *Targeted Mental Health in Schools Project: Using the Evidence to Inform Your Approach: A Practical Guide for Head Teachers and Commissioners*. DCSF, 2008.
- Department for Education and Skills. *Primary National Strategy. Excellence and Enjoyment: Social and Emotional Aspects of Learning*. DfES, 2005.
- Humphrey N, Lendrum A, Wigelsworth M. *Social and Emotional Aspects of Learning (SEAL) Programme in Secondary Schools: National Evaluation*. DFE-RR049. Department for Education, 2010.
- Sawyer MG, Pfeiffer S, Spence SH, Bond L, Graetz B, Kay D, et al. School-based prevention of depression: a randomised controlled study of the beyondblue schools research initiative. *J Child Psychol Psychiatry* 2010; **51**: 199–209.
- Challen A, Noden P, West A, Machin S. *UK Resilience Programme Evaluation: Interim Report*. RR094. Department for Children, Schools and Families, 2009.
- Horowitz JL, Garber J. The prevention of depressive symptoms in children and adolescents: a meta-analytic review. *J Consult Clin Psychol* 2006; **74**: 401–15.
- Calear AL, Christensen H. Systematic review of school-based prevention and early intervention programs for depression. *J Adolesc* 2010; **33**: 429–38.
- Challen A, Noden P, West A, Machin S. *UK Resilience Programme Evaluation. Final Report*. Department for Education, 2011.
- Spence SH, Shortt AL. Research review: can we justify the widespread dissemination of universal, school based interventions for the prevention of depression among children and adolescents? *J Child Psychol Psychiatry* 2007; **48**: 526–42.
- Stallard P, Montgomery AA, Araya R, Anderson R, Lewis G, Sayal K, et al. Protocol for a randomised controlled trial of a school based cognitive behaviour therapy (CBT) intervention to prevent depression in high risk adolescents (PROMISE). *Trials* 2010; **11**: 114.
- Angold A, Costello E, Messer S, Pickles A, Winder F, Silver D. Development of a short questionnaire for use in epidemiological studies of depression in children and adolescents. *Int J Methods Psychiatr Res* 1995; **5**: 237–49.
- Shochet IM, Dadds MR, Holland D, Whitefield K, Harnett PH, Osgarby SM. The efficacy of a universal school-based program to prevent adolescent depression. *J Clin Child Psychol* 2001; **30**: 303–15.
- Shochet I, Ham D. Universal school-based approaches to preventing adolescent depression: past findings and future directions of the Resourceful Adolescent Program. *Int J Ment Health Promotion* 2004; **6**: 17–25.
- Merry S, McDowell H, Wild CJ, Bir J, Cunliffe R. A randomized placebo-controlled trial of a school-based depression prevention program. *J Am Acad Child Adolesc Psychiatry* 2004; **43**: 538–47.
- Rivet-Duval E, Heriot S, Hunt C. Preventing adolescent depression in Mauritius: a universal school-based program. *Child Adolesc Ment Health* 2011; **16**: 86–91.

Special article

Prevention and early intervention for borderline personality disorder: current status and recent evidence

Andrew M. Chanen and Louise McCutcheon

Summary

Borderline personality disorder (BPD) is a leading candidate for developing empirically based prevention and early intervention programmes because it is common in clinical practice, it is among the most functionally disabling of all mental disorders, it is often associated with help-seeking, and it has been shown to respond to intervention, even in those with established disorder. Moreover, it can be reliably diagnosed in its early stages and it demarcates a group with high levels of current and future morbidity and mortality.

Data also suggest considerable flexibility and malleability of BPD traits in youth, making this a key developmental period during which to intervene. Novel indicated prevention and early intervention programmes have shown that BPD in young people responds to intervention. Further work is required to develop appropriate universal and selective preventive interventions.

Declaration of interest

None.

Borderline personality disorder (BPD) is a severe mental disorder that is characterised by a pervasive pattern of impulsivity, emotional instability, interpersonal dysfunction and disturbed self-image.¹ It affects 0.7–2.7% of the general adult population,^{2,3} 9.3–22.5% of people receiving psychiatric out-patient treatment, and in some settings over 40% of in-patients.⁴ The outcome of this disorder in adulthood is now reliably characterised by attenuation of diagnostic criteria over time, but with severe and continuing functional disability across a broad range of domains that is comparable to or greater than that associated with many mental state disorders.^{5,6} People with the disorder also have continuing high rates of health service use,^{7,8} and a suicide rate of around 8%.⁹ Although effective interventions exist for adults,^{10–12} the overall outcomes from such interventions are modest and their availability is limited.

Disorder in young people

Despite long-standing general agreement that personality disorders have their roots in childhood and adolescence,¹³ diagnosing these disorders prior to age 18 years has been more controversial than diagnosing them in adults;¹⁴ however, this is no longer justified.¹⁵ Borderline personality disorder is increasingly seen as a lifespan developmental disorder,¹⁶ which is just as reliable and valid in adolescence as it is in adulthood,^{17,18} is not reducible to Axis I diagnoses,¹⁹ and can be identified in day-to-day clinical practice.²⁰ When the diagnosis is applied, BPD occurs in approximately 3% of community-dwelling teenagers and young people.^{21,22} Indeed, BPD might be better considered as a disorder of younger people, with a rise in prevalence from puberty and a steady decline with each decade from young adulthood.^{23–25} Limited data suggest that BPD occurs in up to 22% of adolescents and young adults receiving out-patient treatment.^{20,26} Borderline personality disorder (or its dimensional representations) in young people demarcates a group with high morbidity and a particularly poor outcome. This disorder uniquely and independently predicts current psychopathology, general functioning, peer relationships, self-care and family and relationship functioning.¹⁹ It also uniquely predicts poor outcomes up to two decades into the future, such as a future BPD diagnosis, increased risk of Axis I

disorders (especially substance use and mood disorders), interpersonal problems, distress and reduced quality of life.^{27–29}

Prevention and early intervention

The above data suggest that BPD is a leading candidate for developing empirically based prevention and early intervention programmes because it is common in clinical practice, it is among the most functionally disabling of all mental disorders, it is often associated with help-seeking (cf. schizotypal or antisocial personality disorder),³⁰ and it has been shown to respond to intervention even in those with established disorder. Moreover, BPD can be reliably diagnosed in its early stages and it demarcates a group with high levels of current and future morbidity and mortality. Data also suggest considerable flexibility and malleability of BPD traits in youth,³¹ making this a key developmental period during which to intervene, and adolescent BPD features have been shown to respond to intervention.^{32,33}

Aims of prevention and early intervention

Prevention and early intervention for BPD should primarily aim to alter the life-course trajectory of young people with borderline personality pathology by attenuating or averting associated adverse outcomes and promoting more adaptive developmental pathways. It should not be narrowly focused on the diagnostic features of BPD, as these naturally attenuate over time. Antisocial personality disorder provides a useful model for such purposes. There is a remarkable amount of information about childhood-onset and adolescent-onset conduct disorder and the developmental pathways leading to antisocial personality disorder, along with associated outcomes such as substance misuse, mental disorders and poor physical health.³⁴ These data logically give rise to potential 'universal', 'selective' and 'indicated' preventive interventions,³⁵ along with early intervention for the established phenotype.^{36,37} Although the time course and form of early manifestations of BPD are likely to differ from those of antisocial personality disorder, the two disorders have substantial phenotypic overlap, and similar objectives might be realised for BPD

through identifying appropriate risk factors and antecedents for intervention.

Risk factors

Findings from prospective longitudinal studies of community samples and studies of young people with borderline pathology suggest a variety of genetic, neurobiological, psychopathological and environmental risk factors for BPD. A fundamental drawback of these data is that their specificity for the disorder appears to be limited.

Heritability estimates for BPD (or its dimensional representations) are around 40%.^{38–40} No specific gene has been identified as causative of BPD and findings from molecular genetic studies have been difficult to replicate.⁴¹ Evidence supports both gene–environment interaction and correlation in the development of BPD.⁴² This means that individuals with a ‘sensitive’ genotype are at greater risk of the disorder in the presence of a predisposing environment. Furthermore, the genes that influence BPD features also increase the likelihood of being exposed to certain adverse life events. So-called comorbidity of BPD with other disorders might also be due to gene–environment correlation: for example, the genetic and environmental correlations between adult BPD traits and adult attention-deficit hyperactivity disorder (ADHD) symptoms support a shared aetiology.⁴⁰

Frontolimbic network abnormalities have been associated with many of the features of BPD in adults,¹ but it is unclear whether these findings are a cause, an effect or an epiphenomenon of the disorder.⁴³ In order to be implicated in the aetiology such abnormalities should be present early in the course of the disorder. Thus far, three structural neuroimaging studies have compared adolescents with BPD with healthy control participants.^{43–45} They found reduced orbitofrontal cortex and anterior cingulate cortex volumes,^{43–46} but not reduced hippocampal or amygdala volumes (which have been widely reported in adult BPD).⁴⁷ One study also included a non-BPD clinical comparison group and found similar changes in this group (compared with healthy control participants),⁴⁴ suggesting that the findings might not be specific to BPD. Although hypothalamic–pituitary–adrenocortical (HPA) axis findings in BPD have been contradictory,⁴⁸ individuals with this disorder have an attenuated cortisol response to acute stress compared with a healthy control group,⁴⁹ and this has also been found in adolescents who self-harm.⁵⁰ More numerous parasuicidal behaviours in adolescents with first-presentation BPD were associated with increased pituitary volumes,⁵¹ suggesting greater activation of the HPA axis. Therefore, it is possible that prolonged activation of the HPA axis in individuals with chronic stress might induce HPA axis hypo-responsiveness, providing a possible developmental pathway to BPD. Adolescent patients with BPD show stronger orienting to negative emotional stimuli than healthy comparison participants but this does not appear to be specific to the disorder.⁵² Adolescent borderline pathology has also been linked to an inability to disengage attention from negative facial expressions during attentional maintenance when in a negative mood.⁵³ However, one study of young people (15–24 years old) with BPD showed no evidence of heightened sensitivity to emotional facial expressions, compared with a community control group.⁵⁴ Young people with borderline pathology have been found to have an impaired capacity to differentiate and integrate the perspective of the self with the perspectives of others (social perspective coordination),⁵⁵ along with impairments in theory of mind (mentalising) due to overinterpretive mental state reasoning, rather than the reduction or loss of theory of mind capacity *per se*.⁵⁶ They have also been found to have a preference

for immediate gratification and a tendency to discount longer-term rewards, which might be related to trait impulsivity.⁵⁷

Taken together, these findings provide interesting clues but no clear and consistent target for intervention. Prospective, longitudinal data are more consistent in demonstrating that a range of childhood and parental demographic characteristics, adverse childhood experiences, early relational difficulties, parental problems and forms of maladaptive parenting are risk factors for adolescent and adult BPD.

Although there is a strong association between BPD and adverse childhood experiences,⁵⁸ the precise aetiological role of childhood adversity remains unclear because putative risk factors such as childhood abuse, adverse familial environment and a family history of psychopathology are highly intercorrelated.⁵⁹ One study has demonstrated that the association between life events and BPD features can be explained by shared genetic influences, causal effects and an interaction between genes and environment, depending on the type of life event.⁴² Specific data on prospectively assessed risk factors for BPD are still insufficient, with the Children in the Community (CIC) study being the only study to have published prospective risk factors over multiple waves from childhood through to adulthood.²⁷ A series of CIC publications report childhood abuse or neglect, childhood and parental demographic characteristics, maladaptive parenting and maladaptive school experiences as risk factors for adolescent and adult personality disorder.^{27,60} Prospective longitudinal data have found that childhood physical abuse, sexual abuse and neglect,⁶¹ along with low socioeconomic status of the family of origin,⁶² are independently associated with elevated features of BPD up to two decades later. Also, maternal inconsistency in child-rearing in the presence of high maternal overinvolvement,⁶³ maladaptive parenting behaviour present during the child-rearing years,⁶⁴ early separations of offspring from their mothers before age 5 years,⁶⁵ and early relational experiences including attachment disorganisation and maltreatment, maternal hostility and boundary dissolution, family disruption related to father's presence and family life stress,⁶⁶ all predict elevations in BPD features 2–30 years later.

Precursor signs and symptoms

Prospective longitudinal data indicate that certain temperamental characteristics and early-onset mental state or behavioural problems that are analogous to characteristics of BPD are precursors to the emergence of the BPD phenotype but do not predict its onset with certainty. These include ADHD, oppositional defiant disorder, conduct disorder, substance use, depression and self-harm, along with the actual features of BPD. However, it is technically imprecise to refer to many of these phenomena as ‘risk factors’,⁶⁷ as these same phenomena are later used to define personality disorder. Eaton *et al* refer to the signs and symptoms from a diagnostic cluster that precede a disorder but do not predict its onset with certainty as ‘precursor signs and symptoms’.⁶⁸

Maternal reports of childhood temperament are related to BPD in adolescence or adulthood, up to 30 years later.^{65,66} Substance use disorders during adolescence – particularly alcohol use disorders – also specifically predict young adult BPD,^{69,70} and there are strong prospective data that disturbances in attention, emotional regulation and behaviour, especially the disruptive behaviour disorders (conduct disorder, oppositional defiant disorder, ADHD) in childhood or adolescence are independent predictors of young adult BPD.^{66,71,72} Moreover, in one study

the rate of growth in ADHD scores from age 10 to 13 years and the rate of growth in oppositional defiant disorder scores from age 8 to 10 years uniquely predicted higher BPD symptoms at age 14 years, suggesting that for adolescent BPD symptoms, difficulties with emotion regulation and relationships might precede problems with impulse control.⁷¹

Self-harm is a core feature of BPD,¹ and retrospective reports from adults with the disorder indicate childhood onset of self-harm in more than 30% and adolescent onset in another 30%.⁷³ However, it is surprisingly underresearched as a potential precursor to BPD. Although self-harm is relatively common among adolescents and young adults,⁷⁴ and is associated with a range of clinical syndromes, there is evidence that repetitive self-harm (which is less frequent) might differ from occasional self-harm.⁷⁵ Borderline personality disorder can be diagnosed in the majority of adolescent girls receiving in-patient treatment for self-harm,⁷⁶ and the likelihood of meeting the diagnosis of BPD is greater in adolescents endorsing both self-harm and suicide attempts compared with individuals reporting self-harm or suicide attempts alone.⁷⁷ Also, the number of BPD criteria met is predictive of whether or not an adolescent has engaged in self-harm or attempted suicide.⁷⁸

There is now clear evidence that dimensional representations of BPD features have similar stability in adolescence and adulthood.¹⁷ Evidence is emerging that the underlying dimensions of these features (conceptualised as impulsivity, negative affectivity and interpersonal aggression) are also stable in children.^{79,80} Only the CIC has specifically measured childhood or adolescent features of personality disorder as a predictor of later personality disorder over multiple assessments from childhood to adulthood.²⁷ Personality disorder symptoms in childhood or adolescence were the strongest long-term predictors, over and above disruptive behaviour disorders and depressive symptoms, of later DSM-IV cluster A, B or C disorders.^{27,81–83} Overall, the CIC data support a normative increase in BPD traits after puberty, perhaps bringing the problems associated with this disorder to clinical attention. As this wanes in early adulthood, partly due to maturational or socialisation processes,²⁷ a group is revealed that is increasingly deviant compared with their peers,⁸⁴ and perhaps conforms more to the 'adult' BPD phenotype. This suggests that young people displaying BPD features are a major group from which the adult phenotype arises. In short, signs and symptoms might appear from childhood through to adolescence that resemble aspects of the BPD phenotype and presage its later appearance in adolescence or emerging adulthood. Certain early temperamental and personality features, internalising and externalising psychopathology and specific BPD criteria are all candidate precursor signs and symptoms. However, more work needs to be done to gain a better understanding of the part these factors play in the developmental pathways to BPD and to increase their specificity for the disorder.

What form should intervention take?

Stand-alone universal (whole population) prevention of BPD is not currently feasible because the condition is not sufficiently prevalent to justify whole-population approaches, and it is unclear what form of intervention would be appropriate. Similarly, selective prevention (targeting those with risk factors for BPD) is currently impractical because many of the risk factors (particularly environmental factors) more commonly lead to, or are associated with, outcomes other than BPD (multifinality).⁸⁵ This should not diminish the importance of intervention for some risk factors (e.g. child abuse and neglect) as primary objectives

because they are undesirable, immoral or unlawful. However, many factors such as poverty require major social and political change and are unlikely to have a major impact on BPD prevention in the near future. Also, it is difficult to design studies with adequate statistical power to demonstrate the efficacy or effectiveness of universal and selective prevention.⁸⁶ Some of these problems would be overcome if current universal and selective programmes (e.g. parent training programmes) were to measure multiple syndromes as outcomes, and the above data constitute a strong case for including BPD as one of these syndromes.

The data reviewed above suggest that 'indicated prevention' is currently the 'best bet' for prevention of BPD.¹⁷ This targets individuals displaying precursor (early) signs and symptoms of the disorder. Although the BPD phenotype is not clearly identifiable in children, its underlying dimensions can be measured, appear to be relatively stable and could be directly targeted. Moreover, typical child and adolescent psychopathology (e.g. disruptive behaviour disorders, self-harm, substance use and depressive disorders) might additionally be regarded as targets for indicated prevention of BPD, rather than separate domains of psychopathology that might then be renamed in adulthood. Two programmes, described below, have been developed that directly target subsyndromal borderline pathology in adolescents,^{32,33,87} while concurrently targeting syndromal BPD.

Early detection and intervention

Early detection and intervention for BPD are now justified and practical in adolescence and emerging adulthood,^{15,20} and novel early intervention programmes have been developed and researched in Australia and The Netherlands.^{32,33,87} Such programmes should be differentiated from conventional BPD treatment programmes that are applied to individuals who have established, complex and severe disorder but happen to be less than 18 years old. Intervention for this latter group should now be considered part of routine clinical practice in adolescent mental health.¹⁵

Principles of early intervention

The Australian Helping Young People Early (HYPE) and Dutch Emotion Regulation Training (ERT) treatment programmes have several features in common.^{33,87} They have broad inclusion criteria, with limited exclusions for co-occurring psychopathology (which is common in BPD). They view BPD dimensionally, combining subsyndromal (indicated prevention) and syndromal (early intervention) disorder. Borderline and other personality disorders are carefully diagnosed, often supported by semi-structured interview. Both HYPE and ERT are time-limited, being 16–24 and 17 sessions long respectively. Both have adapted interventions designed for adults with BPD to make them developmentally suitable. The HYPE programme uses cognitive–analytic therapy,⁸⁸ whereas ERT uses Systems Training for Emotional Predictability and Problem Solving (STEPPS).⁸⁹ The major difference between these programmes is that ERT is delivered in a group format as an adjunct to treatment as usual, whereas HYPE employs a comprehensive, team-based, integrated intervention. In a randomised controlled trial, ERT plus treatment as usual was not substantially different from treatment as usual alone.³³ In contrast, a quasi-experimental comparison of the HYPE intervention and treatment as usual found that HYPE achieved faster rates of improvement in internalising and externalising psychopathology and lower levels of psychopathology at 2-year follow-up.⁹⁰ This suggests that some or all of the

elements of a team-based, integrated intervention might be important for early intervention, and matches clinical experience working with this population. In HYPE, these elements include:

- (a) assertive, 'psychologically informed' case management integrated with the delivery of individual psychotherapy;
- (b) active engagement of families or carers, with psychoeducation and time-limited family intervention, using the same model as individual psychotherapy;
- (c) general psychiatric care by the same team, with specific assessment and treatment of co-occurring psychiatric syndromes (comorbidity), including the use of pharmacotherapy, where indicated for such syndromes;
- (d) capacity for outreach care in the community, with flexible timing and location of intervention;
- (e) crisis team and in-patient care, with a clear model of brief and goal-directed in-patient care;
- (f) access to a psychosocial recovery programme (shared with other programmes at Orygen Youth Health);
- (g) individual and group supervision of staff;
- (h) a quality assurance programme.

Remaining barriers and potential risks

Despite evidence of sufficient reliability and validity for the BPD diagnosis in young people, stigma is a lingering barrier to its early diagnosis in day-to-day clinical practice. Borderline personality disorder is highly stigmatised among professionals,⁹¹ and it is also associated with patient 'self-stigma'.⁹² This fuels the perception that the diagnosis is 'controversial',¹⁴ and experience suggests that many clinicians will deliberately avoid using the diagnosis in young people with the aim of 'protecting' individuals from harsh and/or discriminatory practices. Although concerns about stigma are genuine and the response is well intentioned, this practice runs the risk of perpetuating negative stereotypes, reducing the prospect of applying specific beneficial interventions for the problems associated with BPD and increasing the likelihood of inappropriate diagnoses and interventions and iatrogenic harm (such as polypharmacy). The National Institute for Health and Clinical Excellence (NICE) guideline for BPD supports the diagnosis of this disorder in adolescents,¹⁵ and the forthcoming revisions of the ICD and DSM classification systems are both proposing to remove age-related caveats on the diagnosis of personality disorders.^{93,94} Moreover, the ICD (and possibly the DSM) will include the identification of subthreshold personality pathology. These innovations foster not only the early diagnosis of BPD but also the identification of subthreshold disorder, supporting the aims of indicated prevention and early intervention. However, this will bring into the clinical realm young people (and adults) who might once have been considered 'colourful', and potential benefits are accompanied by potential risks associated with 'medicalising' common problems – risks that are not confined to the field of personality disorders.

Future perspectives

Borderline personality disorder should now be seen as a lifespan developmental disorder with substantial ramifications across subsequent decades. Consequently, intervention at any stage should aim to alter the life-course trajectory of the disorder, not just its diagnostic features. There is now sufficient evidence to support diagnosing and treating BPD when it first appears as

routine clinical practice. This has already been adopted by the NICE guideline for BPD and it is likely to be supported by the ICD-11 and DSM-5. There are also data showing that targeting subsyndromal borderline pathology through indicated prevention is a promising approach and that the benefits of intervention appear to outweigh the risks. However, this approach requires further development and evaluation over longer periods in order to ensure that there are no significant 'downstream' adverse effects.

Indicated prevention and early intervention also offer a unique platform for investigating BPD earlier in its developmental course, where duration of illness factors that complicate the psychopathology and neurobiology of the disorder can be minimised. This might make more sense of the confusing array of biological and psychopathological research findings for the disorder. In the future a more detailed understanding of risk factors, precursors, pathways and mechanisms for the development of BPD might enable the development of universal or selective preventive approaches, but these are likely to require the joint effort of research groups aiming to prevent the range of major mental disorders. 'Clinical staging' for BPD,⁹⁵ which is analogous to disease staging in general medicine, offers a potential integrating framework for selecting appropriate interventions and predicting outcome. A key implication of such an approach is that treatment needs will differ by phase or stage of disorder, with the possibility that interventions might be more benign and/or effective in earlier phases.

Andrew M. Chanen, MBBS, MPM, PhD, FRANZCP, **Louise McCutcheon**, DPsych, MAPS, Orygen Youth Health Research Centre, Centre for Youth Mental Health, University of Melbourne, and Orygen Youth Health Clinical Program, Northwestern Mental Health, Melbourne, Australia

Correspondence: Associate Professor Andrew M. Chanen, Orygen Youth Health Research Centre, Locked Bag 10, Parkville, Victoria 3052, Australia. Email: achanen@unimelb.edu.au

Funding

The Orygen Youth Health Research Centre is funded by an unrestricted philanthropic grant from the Colonial Foundation, Melbourne, Australia.

References

- 1 Leichsenring F, Leibing E, Kruse J, New AS, Leweke F. Borderline personality disorder. *Lancet* 2011; **377**: 74–84.
- 2 Coid J, Yang M, Tyrer P, Roberts A, Ullrich S. Prevalence and correlates of personality disorder in Great Britain. *Br J Psychiatry* 2006; **188**: 423–31.
- 3 Trull TJ, Jahng S, Tomko RL, Wood PK, Sher KJ. Revised NESARC Personality disorder diagnoses: gender, prevalence, and comorbidity with substance dependence disorders. *J Pers Disord* 2010; **24**: 412–26.
- 4 Zimmerman M, Chelminski I, Young D. The frequency of personality disorders in psychiatric patients. *Psychiatr Clin North Am* 2008; **31**: 405–20, vi.
- 5 Gunderson JG, Stout RL, McGlashan TH, Shea MT, Morey LC, Grilo CM, et al. Ten-year course of borderline personality disorder: psychopathology and function from the collaborative longitudinal personality disorders study. *Arch Gen Psychiatry* 2011; **68**: 827–37.
- 6 Zanarini MC, Frankenburg FR, Reich DB, Fitzmaurice G. The 10-year course of psychosocial functioning among patients with borderline personality disorder and axis II comparison subjects. *Acta Psychiatr Scand* 2010; **122**: 103–9.
- 7 Horz S, Zanarini MC, Frankenburg FR, Reich DB, Fitzmaurice G. Ten-year use of mental health services by patients with borderline personality disorder and with other axis II disorders. *Psychiatr Serv* 2010; **61**: 612–6.
- 8 Sansone RA, Farukhi S, Wiederman MW. Utilization of primary care physicians in borderline personality. *Gen Hosp Psychiatry* 2011; **33**: 343–6.
- 9 Pompili M, Girardi P, Ruberto A, Tatarelli R. Suicide in borderline personality disorder: a meta-analysis. *Nord J Psychiatry* 2005; **59**: 319–24.
- 10 Bateman A, Fonagy P. Randomized controlled trial of outpatient mentalization-based treatment versus structured clinical management for borderline personality disorder. *Am J Psychiatry* 2009; **166**: 1355–64.

- 11 Linehan MM, Comtois KA, Murray AM, Brown MZ, Gallop RJ, Heard HL, et al. Two-year randomized controlled trial and follow-up of dialectical behavior therapy vs therapy by experts for suicidal behaviors and borderline personality disorder. *Arch Gen Psychiatry* 2006; **63**: 757–66.
- 12 Giesen-Bloo J, van Dyck R, Spinhoven P, van Tilburg W, Dirksen C, van Asselt T, et al. Outpatient psychotherapy for borderline personality disorder: randomized trial of schema-focused therapy vs transference-focused psychotherapy. *Arch Gen Psychiatry* 2006; **63**: 649–58.
- 13 American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders (3rd edn) (DSM-III)*. APA, 1980.
- 14 Chanan AM, McCutcheon LK. Personality disorder in adolescence: the diagnosis that dare not speak its name. *Personal Ment Health* 2008; **2**: 35–41.
- 15 National Collaborating Centre for Mental Health. *Borderline Personality Disorder: Treatment and Management* (Clinical Guideline CG78). National Institute for Health and Clinical Excellence, 2009.
- 16 Tackett JL, Balsis S, Oltmanns TF, Krueger RF. A unifying perspective on personality pathology across the life span: developmental considerations for the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders. *Dev Psychopathol* 2009; **21**: 687–713.
- 17 Chanan AM, Jovev M, McCutcheon L, Jackson HJ, McGorry PD. Borderline personality disorder in young people and the prospects for prevention and early intervention. *Curr Psychiatry Rev* 2008; **4**: 48–57.
- 18 Miller AL, Muehlenkamp JJ, Jacobson CM. Fact or fiction: diagnosing borderline personality disorder in adolescents. *Clin Psychol Rev* 2008; **28**: 969–81.
- 19 Chanan AM, Jovev M, Jackson HJ. Adaptive functioning and psychiatric symptoms in adolescents with borderline personality disorder. *J Clin Psychiatry* 2007; **68**: 297–306.
- 20 Chanan AM, Jovev M, Djaja D, McDougall E, Yuen HP, Rawlings D, et al. Screening for borderline personality disorder in outpatient youth. *J Pers Disord* 2008; **22**: 353–64.
- 21 Bernstein DP, Cohen P, Velez CN, Schwab-Stone M, Siever LJ, Shinsato L. Prevalence and stability of the DSM-III-R personality disorders in a community-based survey of adolescents. *Am J Psychiatry* 1993; **150**: 1237–43.
- 22 Moran P, Coffey C, Mann A, Carlin JB, Patton GC. Personality and substance use disorders in young adults. *Br J Psychiatry* 2006; **188**: 374–9.
- 23 Ullrich S, Coid J. The age distribution of self-reported personality disorder traits in a household population. *J Pers Disord* 2009; **23**: 187–200.
- 24 Johnson JG, Cohen P, Kasen S, Skodol AE, Hamagami F, Brook JS. Age-related change in personality disorder trait levels between early adolescence and adulthood: a community-based longitudinal investigation. *Acta Psychiatr Scand* 2000; **102**: 265–75.
- 25 Samuels J, Eaton WW, Bienvenu III OJ, Brown CH, Costa Jr PT, Nestadt G. Prevalence and correlates of personality disorders in a community sample. *Br J Psychiatry* 2002; **180**: 536–42.
- 26 Chanan AM, Jackson HJ, McGorry PD, Allott KA, Clarkson V, Yuen HP. Two-year stability of personality disorder in older adolescent outpatients. *J Pers Disord* 2004; **18**: 526–41.
- 27 Cohen P, Crawford TN, Johnson JG, Kasen S. The children in the community study of developmental course of personality disorder. *J Pers Disord* 2005; **19**: 466–86.
- 28 Crawford TN, Cohen P, First MB, Skodol AE, Johnson JG, Kasen S. Comorbid Axis I and Axis II disorders in early adolescence: prognosis 20 years later. *Arch Gen Psychiatry* 2008; **65**: 641–8.
- 29 Winograd G, Cohen P, Chen H. Adolescent borderline symptoms in the community: prognosis for functioning over 20 years. *J Child Psychol Psychiatry* 2008; **49**: 933–41.
- 30 Tyrer P, Mithard S, Methuen C, Ranger M. Treatment rejecting and treatment seeking personality disorders: Type R and Type S. *J Pers Disord* 2003; **17**: 263–8.
- 31 Lenzenweger MF, Castro DD. Predicting change in borderline personality: using neurobehavioral systems indicators within an individual growth curve framework. *Dev Psychopathol* 2005; **17**: 1207–37.
- 32 Chanan AM, Jackson HJ, McCutcheon LK, Jovev M, Dudgeon P, Yuen HP, et al. Early intervention for adolescents with borderline personality disorder using cognitive analytic therapy: randomised controlled trial. *Br J Psychiatry* 2008; **193**: 477–84.
- 33 Schuppert H, Giesen-Bloo J, van Gemert T, Wiersema H, Minderaa R, Emmelkamp P, et al. Effectiveness of an emotion regulation group training for adolescents – a randomized controlled pilot study. *Clin Psychol Psychother* 2009; **16**: 467–78.
- 34 Moffitt TE, Arseneault L, Jaffee SR, Kim-Cohen J, Koenen KC, Odgers CL, et al. Research review: DSM-V conduct disorder: research needs for an evidence base. *J Child Psychol Psychiatry* 2008; **49**: 3–33.
- 35 Mrazek PJ, Haggerty RJ. *Reducing Risks For Mental Disorders: Frontiers For Preventive Intervention Research*. National Academy Press, 1994.
- 36 Woolfenden SR, Williams K, Peat JK. Family and parenting interventions for conduct disorder and delinquency: a meta-analysis of randomised controlled trials. *Arch Dis Child* 2002; **86**: 251–6.
- 37 Weisz JR, Hawley KM, Doss AJ. Empirically tested psychotherapies for youth internalizing and externalizing problems and disorders. *Child Adolesc Psychiatr Clin N Am* 2004; **13**: 729–815, v–vi.
- 38 Distel MA, Trull TJ, Derom CA, Thiery EW, Grimmer MA, Martin NG, et al. Heritability of borderline personality disorder features is similar across three countries. *Psychol Med* 2008; **38**: 1219–29.
- 39 Kendler KS, Aggen SH, Czajkowski N, Roysamb E, Tambs K, Torgersen S, et al. The structure of genetic and environmental risk factors for DSM-IV personality disorders: a multivariate twin study. *Arch Gen Psychiatry* 2008; **65**: 1438–46.
- 40 Distel MA, Carlier A, Middeldorp CM, Derom CA, Lubke GH, Boomsma DI. Borderline personality traits and adult attention-deficit hyperactivity disorder symptoms: a genetic analysis of comorbidity. *Am J Med Genet* 2011; **156B**: 817–21.
- 41 Chanan AM, Kaess M. Developmental pathways toward borderline personality disorder. *Curr Psychiatry Rep* 2012; **14**: 45–53.
- 42 Distel MA, Middeldorp CM, Trull TJ, Derom CA, Willemsen G, Boomsma DI. Life events and borderline personality features: the influence of gene–environment interaction and gene–environment correlation. *Psychol Med* 2011; **41**: 849–60.
- 43 Chanan AM, Velakoulis D, Carison K, Gaunson K, Wood SJ, Yücel M, et al. Orbitofrontal, amygdala and hippocampal volumes in teenagers with first-presentation borderline personality disorder. *Psychiatry Res* 2008; **163**: 116–25.
- 44 Brunner R, Henze R, Parzer P, Kramer J, Feigl N, Lutz K, et al. Reduced prefrontal and orbitofrontal gray matter in female adolescents with borderline personality disorder: is it disorder specific? *Neuroimage* 2010; **49**: 114–20.
- 45 Goodman M, Hazlett EA, Avedon JB, Siever DR, Chu KW, New AS. Anterior cingulate volume reduction in adolescents with borderline personality disorder and co-morbid major depression. *J Psychiatr Res* 2011; **45**: 803–7.
- 46 Whittle S, Chanan AM, Fornito A, McGorry PD, Pantelis C, Yücel M. Anterior cingulate volume in adolescents with first-presentation borderline personality disorder. *Psychiatry Res* 2009; **172**: 155–60.
- 47 Nunes PM, Wenzel A, Borges KT, Porto CR, Caminha RM, de Oliveira IR. Volumes of the hippocampus and amygdala in patients with borderline personality disorder: a meta-analysis. *J Pers Disord* 2009; **23**: 333–45.
- 48 Zimmerman DJ, Choi-Kain LW. The hypothalamic-pituitary-adrenal axis in borderline personality disorder: a review. *Harv Rev Psychiatry* 2009; **17**: 167–83.
- 49 Nater UM, Bohus M, Abbruzzese E, Ditzel B, Gaab J, Kleindienst N, et al. Increased psychological and attenuated cortisol and alpha-amylase responses to acute psychosocial stress in female patients with borderline personality disorder. *Psychoneuroendocrinology* 2010; **35**: 1565–72.
- 50 Kaess M, Hille M, Parzer P, Maser-Gluth C, Resch F, Brunner R. Alterations in the neuroendocrinological stress response to acute psychosocial stress in adolescents engaging in nonsuicidal self-injury. *Psychoneuroendocrinology* 2012; **37**: 157–61.
- 51 Jovev M, Garner B, Phillips L, Velakoulis D, Wood SJ, Jackson HJ, et al. An MRI study of pituitary volume and parasuicidal behavior in teenagers with first-presentation borderline personality disorder. *Psychiatry Res* 2008; **163**: 273–7.
- 52 Von Ceumern-Lindenstjerna IA, Brunner R, Parzer P, Mundt C, Fiedler P, Resch F. Initial orienting to emotional faces in female adolescents with borderline personality disorder. *Psychopathology* 2010; **43**: 79–87.
- 53 Von Ceumern-Lindenstjerna IA, Brunner R, Parzer P, Mundt C, Fiedler P, Resch F. Attentional bias in later stages of emotional information processing in female adolescents with borderline personality disorder. *Psychopathology* 2010; **43**: 25–32.
- 54 Jovev M, Chanan AM, Green M, Cotton SM, Proffitt T, Coltheart M, et al. Emotional sensitivity in youth with borderline personality pathology. *Psychiatry Res* 2011; **187**: 234–40.
- 55 Jennings T, Hulbert C, Jackson H, Chanan A. Social perspective coordination in youth with borderline personality pathology. *J Pers Disord* 2012; **26**: 128–42.
- 56 Sharp C, Pane H, Ha C, Venta A, Patel AB, Sturek J, et al. Theory of mind and emotion regulation difficulties in adolescents with borderline traits. *J Am Acad Child Adolesc Psychiatry* 2011; **50**: 563–73.

- 57 Lawrence KA, Allen JS, Chanen AM. Impulsivity in borderline personality disorder: reward-based decision-making and its relationship to emotional distress. *J Pers Disord* 2010; **24**: 786–99.
- 58 Afifi TO, Mather A, Boman J, Fleisher W, Enns MW, Macmillan H, et al. Childhood adversity and personality disorders: results from a nationally representative population-based study. *J Psychiatr Res* 2011; **45**: 814–22.
- 59 Bradley R, Jenei J, Westen D. Etiology of borderline personality disorder: disentangling the contributions of intercorrelated antecedents. *J Nerv Ment Dis* 2005; **193**: 24–31.
- 60 Cohen P. Child development and personality disorder. *Psychiatr Clin North Am* 2008; **31**: 477–93.
- 61 Johnson JG, Cohen P, Brown J, Smailes E, Bernstein DP. Childhood maltreatment increases risk for personality disorders during early adulthood. *Arch Gen Psychiatry* 1999; **56**: 600–6.
- 62 Cohen P, Chen H, Gordon K, Johnson J, Brook J, Kasen S. Socioeconomic background and the developmental course of schizotypal and borderline personality disorder symptoms. *Dev Psychopathol* 2008; **20**: 633–50.
- 63 Bezirgianian S, Cohen P, Brook JS. The impact of mother-child interaction on the development of borderline personality disorder. *Am J Psychiatry* 1993; **150**: 1836–42.
- 64 Johnson JG, Cohen P, Chen H, Kasen S, Brook JS. Parenting behaviors associated with risk for offspring personality disorder during adulthood. *Arch Gen Psychiatry* 2006; **63**: 579–87.
- 65 Crawford TN, Cohen PR, Chen H, Anglin DM, Ehrensaft M. Early maternal separation and the trajectory of borderline personality disorder symptoms. *Dev Psychopathol* 2009; **21**: 1013–30.
- 66 Carlson EA, Egeland B, Sroufe LA. A prospective investigation of the development of borderline personality symptoms. *Dev Psychopathol* 2009; **21**: 1311–34.
- 67 Kraemer HC, Kazdin AE, Offord DR, Kessler RC, Jensen PS, Kupfer DJ. Coming to terms with the terms of risk. *Arch Gen Psychiatry* 1997; **54**: 337–43.
- 68 Eaton WW, Badawi M, Melton B. Prodromes and precursors: epidemiologic data for primary prevention of disorders with slow onset. *Am J Psychiatry* 1995; **152**: 967–72.
- 69 Rohde P, Lewinsohn PM, Kahler CW, Seeley JR, Brown RA. Natural course of alcohol use disorders from adolescence to young adulthood. *J Am Acad Child Adolesc Psychiatry* 2001; **40**: 83–90.
- 70 Thatcher DL, Cornelius JR, Clark DB. Adolescent alcohol use disorders predict adult borderline personality. *Addict Behav* 2005; **30**: 1709–24.
- 71 Stepp SD, Burke JD, Hipwell AE, Loeber R. Trajectories of attention deficit hyperactivity disorder and oppositional defiant disorder symptoms as precursors of borderline personality disorder symptoms in adolescent girls. *J Abnorm Child Psychol* 2012; **40**: 7–20.
- 72 Burke JD, Stepp SD. Adolescent disruptive behavior and borderline personality disorder symptoms in young adult men. *J Abnorm Child Psychol* 2012; **40**: 35–44.
- 73 Zanarini MC, Frankenburg FR, Ridolfi ME, Jager-Hyman S, Hennen J, Gunderson JG. Reported childhood onset of self-mutilation among borderline patients. *J Pers Disord* 2006; **20**: 9–15.
- 74 Nock MK. Self-injury. *Annu Rev Clin Psychol* 2010; **6**: 339–63.
- 75 Brunner R, Parzer P, Haffner J, Steen R, Roos J, Klett M, et al. Prevalence and psychological correlates of occasional and repetitive deliberate self-harm in adolescents. *Arch Pediatr Adolesc Med* 2007; **161**: 641–9.
- 76 Nock MK, Joiner JTE, Gordon KH, Lloyd-Richardson E, Prinstein MJ. Non-suicidal self-injury among adolescents: diagnostic correlates and relation to suicide attempts. *Psychiatry Res* 2006; **144**: 65–72.
- 77 Muehlenkamp JJ, Ertelt TW, Miller AL, Claes L. Borderline personality symptoms differentiate non-suicidal and suicidal self-injury in ethnically diverse adolescent outpatients. *J Child Psychol Psychiatry* 2011; **52**: 148–55.
- 78 Jacobson CM, Muehlenkamp JJ, Miller AL, Turner JB. Psychiatric impairment among adolescents engaging in different types of deliberate self-harm. *J Clin Child Adolesc Psychol* 2008; **37**: 363–75.
- 79 Stepp SD, Pilkonis PA, Hipwell AE, Loeber R, Stouthamer-Loeber M. Stability of borderline personality disorder features in girls. *J Personal Disord* 2010; **24**: 460–72.
- 80 Crick NR, Murray-Close D, Woods K. Borderline personality features in childhood: a short-term longitudinal study. *Dev Psychopathol* 2005; **17**: 1051–70.
- 81 Cohen P. Childhood risks for young adult symptoms of personality disorder: method and substance. *Multivar Behav Res* 1996; **31**: 121–48.
- 82 Bernstein DP, Cohen P, Skodol A, Bezirgianian S, Brook J. Childhood antecedents of adolescent personality disorders. *Am J Psychiatry* 1996; **153**: 907–13.
- 83 Kasen S, Cohen P, Skodol AE, Johnson JG, Brook JS. Influence of child and adolescent psychiatric disorders on young adult personality disorder. *Am J Psychiatry* 1999; **156**: 1529–35.
- 84 Crawford TN, Cohen P, Johnson JG, Kasen S, First MB, Gordon K, et al. Self-reported personality disorder in the Children in the Community Sample: convergent and prospective validity in late adolescence and adulthood. *J Pers Disord* 2005; **19**: 30–52.
- 85 Cicchetti D, Toth SL. The past achievements and future promises of developmental psychopathology: the coming of age of a discipline. *J Child Psychol Psychiatry* 2009; **50**: 16–25.
- 86 Cuijpers P. Examining the effects of prevention programs on the incidence of new cases of mental disorders: the lack of statistical power. *Am J Psychiatry* 2003; **160**: 1385–91.
- 87 Chanen AM, McCutcheon L, Germano D, Nistico H, Jackson HJ, McGorry PD. The HYPE Clinic: an early intervention service for borderline personality disorder. *J Psychiatr Pract* 2009; **15**: 163–72.
- 88 Ryle A, Kerr IB. *Introducing Cognitive Analytic Therapy*. Wiley, 2002.
- 89 Blum N, St John D, Pfohl B, Stuart S, McCormick B, Allen J, et al. Systems Training for Emotional Predictability and Problem Solving (STEPPS) for outpatients with borderline personality disorder: a randomized controlled trial and 1-year follow-up. *Am J Psychiatry* 2008; **165**: 468–78.
- 90 Chanen AM, Jackson HJ, McCutcheon L, Dudgeon P, Jovev M, Yuen HP, et al. Early intervention for adolescents with borderline personality disorder: a quasi-experimental comparison with treatment as usual. *Aust N Z J Psychiatry* 2009; **43**: 397–408.
- 91 Aviram RB, Brodsky BS, Stanley B. Borderline personality disorder, stigma, and treatment implications. *Harv Rev Psychiatry* 2006; **14**: 249–56.
- 92 Rusch N, Holzer A, Hermann C, Schramm E, Jacob GA, Bohus M, et al. Self-stigma in women with borderline personality disorder and women with social phobia. *J Nerv Ment Dis* 2006; **194**: 766–73.
- 93 Skodol AE, Bender DS, Morey LC, Clark LA, Oldham JM, Alarcon RD, et al. Personality disorder types proposed for DSM-5. *J Pers Disord* 2011; **25**: 136–69.
- 94 Tyrer P, Crawford M, Mulder R, Blashfield R, Farnam A, Fossati A, et al. The rationale for the reclassification of personality disorder in the 11th revision of the International Classification of Diseases (ICD-11). *Personal Ment Health* 2011; **5**: 246–59.
- 95 McGorry PD. Risk syndromes, clinical staging and DSM V: new diagnostic infrastructure for early intervention in psychiatry. *Schizophr Res* 2010; **120**: 49–53.

Special article

Designing youth mental health services for the 21st century: examples from Australia, Ireland and the UK

Patrick McGorry, Tony Bates and Max Birchwood

Summary

Despite the evidence showing that young people aged 12–25 years have the highest incidence and prevalence of mental illness across the lifespan, and bear a disproportionate share of the burden of disease associated with mental disorder, their access to mental health services is the poorest of all age groups. A major factor contributing to this poor access is the current design of our mental healthcare system, which is manifestly inadequate for the unique developmental and cultural needs of our young people. If we are to reduce the impact of mental disorder on this most vulnerable population

group, transformational change and service redesign is necessary. Here, we present three recent and rapidly evolving service structures from Australia, Ireland and the UK that have each worked within their respective healthcare contexts to reorient existing services to provide youth-specific, evidence-based mental healthcare that is both accessible and acceptable to young people.

Declaration of interest

None.

National mental health policies in the new century will increasingly seek to explore preventive strategies and better reflect the pattern of mental ill health across the lifespan. This pattern is essentially the mirror image of that seen in physical illness, with the peak age at onset and need for initial care for mental disorders occurring in adolescence and early adulthood.^{1,2} Australian National Mental Health Survey data have revealed that young people not only have the highest incidence and prevalence of mental illness across the lifespan, they also manifest the worst service access of any age group, with only 21.8% of Australians between 16 and 24 years of age with a diagnosable mental disorder accessing professional help.³ Alarming, only 13% of young men with a mental disorder accessed mental healthcare.³ Recent data from New Zealand and the USA reveal rates of 50% incidence between ages 12 and 25 years and 40% 12-month prevalence between ages 13 and 18 years.^{4,5} Much of this mental ill health is persistent and causes serious functional impairment which has lasting impacts. Hence, although it might be tempting to dismiss this phenomenon as 'overdiagnosis', the facts do not support this. An equally important influence on service culture and structure has been the changing experience of the developmental transition from childhood to adulthood in the 21st century.⁶ Emerging adulthood is now a more prolonged and unstable developmental stage with novel aspects, yet with increased risks of mental ill health.⁶ The world has changed dramatically in recent decades and young people are not only in the vanguard of these changes, but are also bearing the burden associated with them.⁷

Our existing services are manifestly not providing access or appropriate care. Both child and adolescent mental health services (CAMHS) and adult mental health services (AMHS) provide delayed and heavily restricted access to services for a small subgroup of people with severe and complex disorder, whose developmental and family needs are not met in a holistic manner across the age range, with the artificial boundary at age 18 years a major barrier. The challenge we face is therefore a matter of scale, scope, culture and expertise. Redesign and transformational change are needed. Although the status quo is a tenacious opponent, we should also recognise that our service models have shallow roots. Current AMHS are recent successors to the purely bed-based 19th-century asylum system, and clinically continue

to adopt a similar focus in most jurisdictions. The CAMHS system is essentially a recent extension of a quite different tradition, the child guidance model. Admirably, it has sought to fill a huge gap and extend coverage through adolescence to the lower reaches of the adult system. Despite the best will in the world, this mimicry of the paediatric/adult divide of general healthcare has not been successful, as the work of Singh *et al* has illustrated in the UK.⁸ There is not only a clash of history and culture, but practically speaking a yawning gap into which many young people and their families are falling every day around the world. The current system is weakest where it needs to be strongest. This is not a situation where incremental change is likely to work, because the fundamentals of any new system need to be right. Although the existing service structures are fundamentally flawed, the challenge of designing stigma-free services with a better match to the emerging needs of young people should not be underestimated. We describe here three recently evolving services from Australia, Ireland and the UK that have attempted, in their different healthcare contexts, to redefine service structures for young people up to 25 years old in the light of this emerging evidence.

Innovation in youth mental health

It is possible to describe a set of key features, principles and targets for the redesign of services to better meet the needs of young people; these may be summarised as follows:

- (a) youth participation at all levels, essential to create youth-friendly, stigma-free cultures of care;
- (b) a holistic, preventive and optimistic stance with sequential/stepwise care governed by risk/benefit and shared decision-making principles;
- (c) early intervention, social inclusion and vocational outcomes as core targets;
- (d) care reflecting both the epidemiology of mental ill health in young people and the new developmental culture of emerging adulthood in the early 21st century;
- (e) elimination of discontinuities at peak periods of need for care and developmental transition;

- (f) positive and seamless linkages with services for younger children and older adults.

The service models described here all seek to address some or all of these issues. It is important to note that each of these descriptions can be assigned to one of two tiers: an enhanced primary care level, which has extensions into many community domains; and a specialist youth mental health level, which enables acute, complex and potentially more severe and enduring forms of mental ill health to be responded to in a timely and developmentally appropriate manner.

Australia

Australia is experiencing a new wave of mental health reform within which transformational change in youth mental health is one of the key growth points. Reform is occurring at both the primary and specialist care levels within the complexities of the federal/state governmental system. The Royal Australian and New Zealand College of Psychiatrists has established a special interest group in youth mental health to create new professional interest, knowledge and skills in this emergent field and to help meet workforce needs within the expanding service system described below.

Headspace

Headspace, the National Youth Mental Health Foundation (www.headspace.org.au/), is an enhanced primary care model for youth mental healthcare in Australia. It was established by the Australian federal government in 2006, with the mission to promote and support early intervention for young people aged 12–25 years with mental ill health.⁹ The major part of its mandate was to establish youth-friendly, highly accessible centres that target young people's core health needs by providing a multidisciplinary enhanced primary care structure or 'one-stop shop', with close links to locally available specialist services and schools and many other community-based organisations. These centres are not designed to substitute for existing primary care services, but rather to complement them by encouraging young people to access an enhanced form of primary care as early as possible. The provision of a youth-friendly environment is vital as this is rarely available in standard primary care or the specialist mental health systems, and provides a soft entry point that is more appealing and effective in attracting distressed or struggling young people into the service without labelling or prematurely medicalising the problem.

Consulting for physical health problems is an important part of the Headspace mandate. This is because the physical health services provide a stigma-free access point to the scheme for young people, as well as continuity of care for their mental health problems. It also goes some way towards addressing the comorbidity of physical and mental health problems. The evaluation study of Headspace revealed that the physical health services were popular with the young people who used the service,¹⁰ and that 62% of the young people surveyed as part of the in-depth evaluation process of the study reported improved physical health since using the service. Furthermore, both clinicians and young people reported that it was extremely useful to have medical and counselling services co-located as this not only encouraged young people to seek help, but also increased the likelihood that they would follow the medical advice that they were given. Young people said they would be more likely to take advice from Headspace clinicians than from other doctors, and that they felt more confident about advice when it came from a number of different, trusted practitioners – from both a general practitioner (GP) and a psychologist, for example.

The model is a universal one, congruent with Australia's universal system of healthcare; however, many Headspace centres

have been located in regions with relatively poor access to standard mental health services, particularly in regional and rural parts of Australia and outer metropolitan regions with high need and poor access. The overarching aims of each centre are to promote and support early intervention for mental and substance use disorders through four core service streams: mental health, drug and alcohol services, primary care (general health) and vocational/educational assistance. To achieve this type of integrated care, each Headspace centre is led by a key agency (typically, but not always, a primary healthcare divisional structure that coordinates primary care in that region) on behalf of a local consortium of organisations who take responsibility for the coordination and delivery of the four core streams within a 'one-stop shop' or single location. This approach is designed to facilitate the coming together of existing local services that are already working well within the region, to create a new, highly visible portal and platform for the care of young people. In addition to the provision of services within the four core streams, each Headspace centre also delivers local community awareness campaigns to enhance young people's help-seeking behaviour, the capacity of families and local service providers to identify emerging mental health concerns early and to strengthen referral pathways into the service. Headspace has also developed internet-based programmes to provide online support and interventions and school-based intervention programmes across the country.

Four Headspace sites have strong links with specialised youth mental health services; Campbellfield and central Sydney with the Brain and Mind Research Institute (BMRI) in Sydney, and western and northern Melbourne with Orygen Youth Health in Melbourne. Because Orygen Youth Health and the BMRI are major mental health research institutes, these links will provide an unparalleled opportunity for the conduct of clinical research to improve the evidence base for the utilisation of treatments specific to the stage and severity of emerging mental disorders in young people, as well as the trialling of methods that foster efficient take-up of evidence-based treatments and models of care into clinical practice.

Headspace has been evaluated and the results, although preliminary, were very positive. Ninety-three per cent of young people were satisfied with the care they received, the engagement of young men was just as successful as that of young women (a major change) and access has been provided so far to over 50 000 young Australians through the 30 centres that have been operating in the first wave of this national programme.¹⁰ These sites are uniformly stigma-free and strongly supported by their local communities. The bulk of the young people using Headspace services are experiencing moderate levels of mental ill health; however, at most of the sites there is also a substantial subset of young people with more complex, severe and enduring problems who currently are unable to gain access to the traditional child and adult mental health systems.¹¹ Within the context of a 10-year reform programme, the federal government has allocated an additional AUD\$197 million of funding to strengthen the capacity of the existing sites and to increase the number of Headspace sites to 90 nationally by 2015. The programme has bipartisan political support, and we are now witnessing for the first time communities across Australia lobbying and competing for a Headspace service to be established locally.

Orygen Youth Health

Headspace addresses early intervention, particularly for common mental health problems, but a second tier or back-up system is necessary for young people with complex presentations or more

severe conditions, who typically require intensive, specialised treatment and a longer tenure of care. Orygen Youth Health (<http://oyh.org.au>) was established in 2002, having evolved during the middle to late 1990s from the Early Psychosis Prevention and Intervention Centre (EPPIC) model to deliver specialised early intervention to a broader range of diagnostic groups;^{12,13} it is Australia's largest youth-specific mental health organisation and comprises an integrated research and clinical programme.¹² Its clinical programme provides a range of community-based and acute services for over 700 young people per annum aged 15–25 years living within a catchment area of approximately 1 million people in north-western metropolitan Melbourne.

Orygen Youth Health focuses on early intervention for psychosis, mood disorders and borderline personality disorder in young people, acknowledging the complexities of service provision in an age group where comorbidity is the norm, and that linkages with other mental health and general support agencies are essential in ensuring quality service provision. The 'front end' of its clinical programme is the youth access team, a 24h, 7 days per week triage, assessment and crisis response service, which also provides community- and home-based services to those who require more intensive treatment than can be offered by their case manager alone. Once accepted into the service, a young person is managed by the continuing care team, which is structured around four specialised clinics: EPPIC, for young people who are experiencing a first episode of psychosis (including type 1 bipolar disorder); the Personal Assessment and Crisis Evaluation (PACE) clinic, which accepts young people who are assessed as being at ultra-high risk of developing a psychotic disorder; the Youth Mood Clinic, for young people experiencing a range of non-psychotic disorders, predominantly major depression and type 2 bipolar disorder; and Helping Young People Early (HYPE), for young people with emerging borderline personality disorder. These clinics each offer a 2-year period of care and provide a full range of specialised interventions, including case management, individual support and therapy, and consultation–liaison, and work closely with Orygen's psycho-social recovery programme to support the young person's social and vocational recovery and return to optimal functioning as soon as possible. Particularly critical in this context are vocational interventions and groups that focus on assisting clients with school, study and work goals and functioning. Orygen Youth Health also has a 16-bed in-patient unit specifically for young people which focuses on acute care, emphasising brief admission in order to prepare the young person for community support provided by the youth access team or case manager.

National scaling-up of the EPPIC model

Early intervention for psychosis, largely focused on young people, commenced with the original EPPIC model in 1992 in Melbourne.¹³ Although early intervention models have been developed and scaled up in hundreds of locations internationally during the past two decades, Australia had until now largely failed to carry out this reform systematically. In the context of the new national reforms, and backed by AUD\$222 million of federal funding with matching state government funding, from early 2012, a national system of 16 high-fidelity early psychosis services will be developed across the nation, a national partnership between federal and state governments which by 2015 will see many more Australian communities at last gaining access to one of the most evidence-based and popularly supported reforms in mental healthcare. Focused on those aged 15–24 years and linked where possible to the expanding Headspace network, these early psychosis services will provide much-needed back-up to many

of the young people in Headspace who need a more specialised service with a youth-friendly culture. This reform will also build a national base for potential future extension of the early intervention strategies to non-psychotic disorders in young people along the lines of the Orygen model described earlier.

Ireland

High rates of suicide and self-harm have created a sense of alarm in Ireland and a deep concern about the mental health and well-being of Irish young people. Prevalence studies have confirmed high rates of mental health problems among young people,^{14–16} and an escalating drumbeat of media stories about suicide, antisocial behaviour, school failure and substance misuse has reinforced the perception of a generation in crisis. It was in this context that Headstrong, the National Centre for Youth Mental Health, was founded as an autonomous Irish charitable organisation with the intent of promoting change through a public–private partnership. This occurred in the context of a national desire to see widespread mental health reform occur as captured in the national mental health policy framework *A Vision for Change*.¹⁷ Pathways to care for young people were non-existent or dysfunctional; there was no coherent continuum of support, providers tended to operate within silos and did not communicate or collaborate, and narrow funding streams and territoriality resulted in rigidity in the way people thought about and responded to young people, while young people felt they had no voice.

Jigsaw

The Jigsaw model of service delivery was Headstrong's response to the challenge of transforming the way young people in Ireland access support and changing the way Ireland thinks about young people. The model is based on certain key ideas and assumptions. The existing community-based system of specialist mental health services was believed to be inadequate; however, simply adding more positions, services and programmes would not necessarily improve the current system. Headstrong felt that systemic and cultural transformation was needed. To achieve this, young people needed to be actively engaged in the design, implementation and review of programmes to ensure that these programmes would be accessible and non-stigmatising for young people, and that partnerships among services engaged in promoting positive youth mental health would be fostered.

Guided by the phrase 'somewhere to turn to, someone to talk to',¹⁸ the Jigsaw model aimed to strengthen a community's capacity to support its young people. Headstrong engaged strongly and consistently with a number of communities across the country to gauge and enhance the level of commitment to tackle the challenge of youth mental health. This meant providing avenues for the voices of local young people to be heard, engaging all relevant stakeholders, including key statutory, community and voluntary agencies (e.g. CAMHS, AMHS, primary care, youth sector services, education and local development groups), rigorous planning processes, and training and community awareness activities. This was a successful strategy that generated strong local support for the next step that would require the re-engineering of services, new access points and establishment of new partnerships. As in Australia, high-level political support has been crucial, bipartisan and strong, and both the President and the Taoiseach have been directly involved in these reforms.

Jigsaw demonstration sites

Five Irish communities were selected as Jigsaw demonstration sites: Counties Galway, Kerry and Meath, and the towns of

Roscommon and Ballymun, the last a disadvantaged neighbourhood of north Dublin. The intention was for these sites to implement transformation strategies with fidelity, serve as learning communities for ongoing development, validate the Jigsaw model and become centres of excellence for the remaining communities in Ireland. As of November 2011 a total of 2079 young people had been seen on an individual basis by the three fully operational Jigsaw sites: Galway, Ballymun and Kerry. The data demonstrate a wide diversity of access pathways, most commonly self- and parent referral. Many referrals came from secondary schools, social work services, youth programmes, adult mental health, general practitioners and peers. The majority of Jigsaw support recipients were in the 15–18 year age range, but the programme has also engaged a significant number of emerging adults in the 19–25 year age range.

The most common presenting issues for young people are anger, stress, tension, low self-worth, family problems and alcohol use. The resultant goal plans cover a wide range of areas, but the most common focus is on emotional, cognitive and behavioural self-regulation, as well as substance use, learning and family issues. To date, interventions related to peer relationships, help-seeking, daily living skills, physical health and emotional regulation have the highest rates of goal attainment. In contrast, lower levels of goal attainment are seen in areas such as housing, employment, problem-solving and conflict management. Approximately 5–10% of engaged young people have needs requiring higher-level mental health specialty services.

Despite the tight fiscal environment in Ireland, funding has been committed for the expansion of the number of sites to 12, and the Health Service Executive is becoming more strongly involved in the reform process. An Irish special interest group in youth mental health has been in operation for over 2 years and has held one highly successful national youth mental health conference.

England

Birmingham is the UK's second largest city with a population of 1.2 million; it is often characterised as the 'youngest city in Europe', with a population slanted towards youth and ethnic diversity including large Black, Muslim and Sikh communities. The population is served by two mental health services: Birmingham and Solihull Mental Health Foundation Trust (BSMHFT), serving those aged 16 years and above, and the CAMHS, which is sited in the Birmingham Children's Hospital. The adult mental health service in BSMHFT acted as the crucible for the UK's National Framework for Mental Health, pioneering integrated community services including early intervention in psychosis, home treatment/crisis resolution and assertive outreach teams.¹⁹

Youthspace

The BSMHFT created a youth services programme, Youthspace, to catalyse the development of youth-sensitive service provision to improve youth access and health outcomes. Youthspace emerged following extensive consultation with young people and qualitative research about the experience of existing youth-focused care.²⁰ The seeds of long-term social disability and exclusion among people with recurring mental health problems begin in adolescence (Jones, this supplement);²¹ improving life chances for young people is currently a political imperative in the UK, particularly for those who by age 25 years are not in employment, education or training. There are many non-health youth agencies in the UK working with socially marginalised young people who have considerable experience of this task; pre-eminent among these is the Prince's Trust (<http://princes-trust.org.uk>), which

provides numerous projects across the country to improve education, skill training and entrepreneurship for young people up to the age of 25 years. Youthspace has developed a strategic partnership with the Prince's Trust to jointly deliver mental health services to young people under 26 years old in Birmingham, placing social inclusion and employment at its heart.

Improving youth access. Access to services for the 16–25 year age group is being consolidated into two pathways. First, the adult community mental health teams are developing a youth access pathway – the youth access teams – which is being rolled out across Birmingham. These teams provide assessment and formulation to the referring GP within 1 week of referral; the default evidence-based intervention is brief cognitive-behavioural therapy, and any medication needs are delivered by the GP following advice from the team's consultant psychiatrist. Young people are seen in low-stigma channels of the young person's choice, including primary care or Prince's Trust facilities. In addition to symptomatic treatment, cases are screened for risk of emerging psychosis, bipolar disorder and eating and personality disorders within a staging framework (Lin *et al*, this supplement).²²

Improving transitions from CAMHS is the responsibility of a subteam that includes child psychiatrists and psychologists operating under an agreed transitions policy. This team is also responsible for managing the admissions of young people aged 16 or 17 years to non-adult units.

Intensive care streams. Those requiring further intensive interventions have access to the following specialised streams:

- (a) early intervention in psychosis – five early intervention teams provide care from 14 years upwards (in conjunction with CAMHS up to age 16 years), a CAMHS-trained care coordinator manages all patients 14- to 16-years old in conjunction with the CAMHS teams;
- (b) attention-deficit hyperactivity disorder (ADHD) – an ADHD service provides care to all young people as appropriate, transitioning from CAMHS;
- (c) eating disorders – specialised in-patient and community services;
- (d) forensic – YouthFIRST is a specialist community and in-patient forensic mental health service for young people at risk of offending or repeat offending.

Public youth mental health. In line with the UK mental health policy to promote prevention, early intervention and public well-being,²³ Youthspace operates across Birmingham providing mental health awareness and interventions to promote resilience in young people through school-based interventions, together with targeted intervention with groups at high risk of lifelong mental health difficulty, particularly those in local authority care or young offenders.²⁴ Internet and social media technologies are used to maximise reach to young people in the city. At the heart of this is the website www.youthspace.me, which has been designed by young people and gives advice, education and individualised assessment. Those accessing care are given personalised access to the website, which has online cognitive-behavioural therapy built in. A Facebook page and Twitter feed are available.

Evaluation. Youthspace has been subjected to a UK Health Innovation and Education Cluster (service innovation) evaluation comparing the programme's results with access and outcomes for young patients seen previously within CMHTs. Further research

into level of engagement/drop-out from services, time to assessment and clinical outcomes is in progress.

Discussion

The services described here have been built around a recognition of the major weakness of the health system for young people with mental ill health, consequent major unmet need, and a shared commitment to improve the accessibility, scale and cultural/developmental appropriateness of mental health services to young people and families, and to reduce the need for harmful transitions at critical points in the young person's development. Jigsaw in Ireland is a public-private funded initiative providing additional early intervention support to young people and largely operates by coordinating existing provisions. Within mainstream healthcare, Headspace in Australia also addresses early intervention, particularly for common mental disorders, and is increasingly a fundamental building block of the primary care system and a new portal of access, information and multi-disciplinary holistic care for young people. In Melbourne it is linked to a 'back-up' specialist system (Orygen Youth Health) for young people with complex presentations or more severe conditions, who typically require intensive, specialised treatment and a longer tenure of care. With the scaling-up of early psychosis services in Australia similar back-up will become increasingly available. If enhanced primary care youth health services were to develop in the UK then the early intervention in psychosis services could be accessed in this fashion by the subset of young people with psychosis who need them. Indeed, this would greatly aid the early detection of psychosis in young people, which is typically subject to long delays even after access to CAMHS and adult services. Youthspace in Birmingham occupies the other end of the continuum, where improved youth access and care are being undertaken through redesign of existing secondary healthcare provision and hence is similar to Orygen in focus. In Melbourne the integration of wider youth access via Headspace and specialised support through a dedicated youth mental health service provides the most complete picture of what a comprehensive service might look like in the future.

We believe that for transformational change to be successful, models like these need to be created, perfected and then scaled up within the context of national mental health policy frameworks that recognise the needs of people experiencing mental ill health across the lifespan, and that the provision of mental healthcare, notwithstanding the principle of integration with physical healthcare, must be correctly engineered, weighted and sequenced. The International Youth Mental Health Association has been established with leadership from seven countries to promote these objectives. One international youth mental health conference has been held in Melbourne in 2010, with a second to occur in March 2013 in Ireland.

Whenever new service frameworks appear they attract healthy debate and an appropriate demand for these alternatives to 'prove themselves'. This has been the story of early intervention in psychosis services;²⁵ however, it has hardly been a level playing field. It is also incumbent on those supporting the status quo to do the same, yet this demand is seldom met. The service reconfigurations described here challenge us to ask whether the existing systems remain the best solution to the changing landscape of need and evidence in relation to the mental healthcare of young people in the 21st century. These systems have not been designed from first principles, but have evolved from different origins under a range of influences. With inertia and self-interest as powerful allies the status quo is hard to change.

It tends to privilege the needs of professionals and managers over those needing the service; hence its defenders are drawn primarily from the ranks of the former. The innovations described here seek to give voice to the latter, and we hope that service reform for young people continues to be informed by evidence, user preference and an increasing focus on preventive strategies. We recognise that the alternative models described here also have their weaknesses; in particular they include their own transition points. However, we contend that the evidence reviewed in this supplement convinces us that if we were to set about designing mental health services now we would not include a transition point at age 16–18 years; indeed, this is the point likely to do most harm. We believe that services for people up to 25 years old should be conceived as preventive in nature, interfacing with public mental health initiatives on the one hand and offering interventions that promote resilience as well as symptom reduction on the other. The aim of youth services should therefore be to reduce the need for transition into adult services. This reframing of the role of services, we believe, can galvanise the research and service commissioning agenda and decisively move services from symptom reduction and containment to prevention and social inclusion. We look forward to the debate.

Patrick McGorry, MD, PhD, FRCP, FRANZCP, Orygen Youth Health Research Centre, University of Melbourne, Australia; **Tony Bates**, PhD, Headstrong – The National Centre for Youth Mental Health, Dublin, Ireland; **Max Birchwood**, PhD, DSC, YouthSpace Programme, Birmingham and Solihull Mental Health and School of Psychology, University of Birmingham, UK

Correspondence: Professor Patrick McGorry, Director of Clinical Services, Orygen Youth Health Research Centre, University of Melbourne, Locked Bag 10, Parkville, Melbourne, Victoria 3052, Australia. Email: pmcgorry@unimelb.edu.au

Funding

P.McG. is supported by the Colonial Foundation, the Australian National Health and Medical Research Council, and the Australian and Victorian governments. T.B. is supported by the One Foundation and the Health Services Executive of the Government of Ireland. M.B. is part-funded by the National Institute for Health Research Birmingham and Black Country Collaboration for Leadership in Applied Health Research and Care.

References

- 1 Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Arch Gen Psychiatry* 2005; **62**: 593–602.
- 2 McGorry PD, Purcell R, Goldstone S, Amminger GP. Age of onset and timing of treatment for mental and substance use disorders: implications for preventive intervention strategies and models of care. *Curr Opin Psychiatry* 2011; **24**: 301–6.
- 3 Australian Institute of Health and Welfare. *Young Australians: Their Health and Well-Being 2007*. AIHW, 2007.
- 4 Gibb SJ, Fergusson DM, Horwood LJ. Burden of psychiatric disorder in young adulthood and life outcomes at age 30. *Br J Psychiatry* 2010; **197**: 122–7.
- 5 Kessler RC, Avenevoli S, Costello EJ, Georgiades K, Green JG, Gruber MJ, et al. Prevalence, persistence, and sociodemographic correlates of DSM-IV disorders in the National Comorbidity Survey Replication Adolescent Supplement. *Arch Gen Psychiatry* 2012; **69**: 372–80.
- 6 Arnett JJ. *Emerging Adulthood: The Winding Road from the Late Teens Through the Twenties*. Oxford University Press, 2004.
- 7 Eckersley RM. *Never Better – or Getting Worse? The Health and Wellbeing of Young Australians*. Australia21, 2008.
- 8 Singh SP, Paul M, Ford T, Kramer T, Weaver T, McLaren S, et al. Process, outcome and experience of transition from child to adult mental healthcare: multiperspective study. *Br J Psychiatry* 2010; **197**: 305–12.
- 9 McGorry PD, Tanti C, Stokes R, Hickie IB, Carnell K, Littlefield LK, et al. Headspace: Australia's National Youth Mental Health Foundation – where young minds come first. *Med J Australia* 2007; **187** (suppl 7): s68–70.
- 10 Muir K, Powell A, Patulny R, Flaxman S, McDermott S, Oprea I, et al. *Independent Evaluation of headspace: the National Youth Mental Health*

Foundation. Social Policy Research Centre, University of New South Wales, 2009.

- 11 Scott EM, Hermens DF, Glozier N, Naismith SL, Guastella AJ, Hickie IB. Targeted primary care-based mental health services for young Australians. *Med J Aust* 2012; **196**: 136–40.
- 12 Purcell R, Goldstone S, Moran J, Albiston D, Edwards J, Pennell K, et al. Towards a 21st century approach to youth mental health care: some Australian initiatives. *Int J Ment Health* 2011; **40**: 72–88.
- 13 McGorry PD, Edwards J, Mihalopoulos C, Harrigan SM, Jackson HJ. EPPIC: an evolving system of early detection and optimal management. *Schizophr Bull* 1996; **22**: 305–26.
- 14 Lynch F, Mills C, Daly I, Fitzpatrick C. Challenging times: a study to detect Irish adolescents at risk of psychiatric disorders and suicidal ideation. *J Adolesc* 2004; **27**: 441–51.
- 15 Lynch F, Mills C, Daly I, Fitzpatrick C. Challenging times: prevalence of psychiatric disorders and suicidal behaviours in Irish adolescents. *J Adolesc* 2006; **29**: 555–73.
- 16 Martin M, Carr A, Burke L, Carroll L, Byrne S. *The Clonmel Project: Mental Health Service Needs of Children and Adolescents in the South East of Ireland*. Health Service Executive Clonmel, 2006.
- 17 Expert Group on Mental Health Policy. *A Vision for Change: Report of the Expert Group on Mental Health Policy*. Government of Ireland, 2006.
- 18 Bates T, Illback R, Scanlan F, Carroll L. *Somewhere To Turn To, Someone To Talk To*. Headstrong, National Centre for Youth Mental Health, 2009.
- 19 Joseph R, Birchwood M. The national policy reforms for mental health services and the story of early intervention services in the United Kingdom. *J Psychiatry Neurosci* 2005; **30**: 362–5.
- 20 Lester H, Birchwood M, Bryan S, England E, Rogers H, Sirvastava N. Development and implementation of early intervention services for young people with psychosis: case study. *Br J Psychiatry* 2009; **194**: 446–50.
- 21 Jones PB. Adult mental health disorders and age at onset. *Br J Psychiatry* 2013; **202** (suppl 54): s5–10.
- 22 Lin A, Reniers RLEP, Wood SJ. Clinical staging in severe mental disorder: evidence from neurocognition and neuroimaging. *Br J Psychiatry* 2013; **202** (suppl 54): s11–7.
- 23 Department of Health Division of Mental Health. *No Health Without Mental Health: A Cross-Government Mental Health Outcomes Strategy for People of All Ages*. UK Department of Health, 2011.
- 24 Department of Health Division of Mental Health. *Confident Communities, Brighter Futures: A Framework for Developing Well-being*. UK Department of Health, 2010.
- 25 McGorry P, Johanessen JO, Lewis S, Birchwood M, Malla A, Nordentoft M, et al. Early intervention in psychosis: keeping faith with evidence-based health care. *Psychol Med* 2010; **40**: 399–404.

Transfers and transitions between child and adult mental health services

Moli Paul, Tamsin Ford, Tami Kramer, Zobia Islam, Kath Harley and Swaran P. Singh

Background

Transfer of care from one healthcare provider to another is often understood as a suboptimal version of the process of transition.

Aims

To separate and evaluate concepts of transfer and transition between child and adolescent mental health services (CAMHS) and adult mental health services (AMHS).

Method

In a retrospective case-note survey of young people reaching the upper age boundary at six English CAMHS, optimal transition was evaluated using four criteria: continuity of care, parallel care, a transition planning meeting and information transfer.

Results

Of 154 cases, 76 transferred to AMHS. Failure to transfer resulted mainly from non-referral by CAMHS ($n=12$) and refusal by service users ($n=12$) rather than refusal by AMHS ($n=7$). Four cases met all criteria for optimal transition, 13 met none; continuity of care ($n=63$) was met most often.

Conclusions

Transfer was common but good transition rare. Reasons for failure to transfer differ from barriers to transition. Transfer should be investigated alongside transition in research and service development.

Declaration of interest

None.

Healthcare transition has been described as ‘a purposeful, planned process that addresses the medical, psychosocial and educational/vocational needs of adolescents and young adults with chronic physical and medical conditions as they move from child-centered to adult-oriented healthcare systems.’¹ It may be one of a number of developmental transitions that young people face as they move through adolescence into adulthood. Healthcare transition planning and management are key elements in the organisation and delivery of health services.^{2–4} Transition is particularly important between child and adolescent mental health services (CAMHS) and adult mental health services (AMHS),³ because failure results in service delivery being weak when the needs of young people are most pressing, as illustrated by other papers in this supplement.^{5–7} Transfer is often discussed as a suboptimal version of transition⁴ but, in our hypothesis, it is distinct from transition and should be investigated alongside transition. Transfer is the termination of care by a children’s healthcare provider and its re-establishment with an adult provider,⁸ i.e. more of an event or transaction between services. Transition is a process requiring therapeutic intent, which may be expressed by the young person’s preparation for transition, a period of handover or joint care, transition planning meetings (involving the young person and carer, and key CAMHS and AMHS professionals) and transfer of case notes or information summaries. Transition ultimately results in established engagement of the young person with adult services and therefore includes vital aspects of continuity of care.⁹

The TRACK study was a multistage, multicentre and multimethod study of adolescents’ transitions between CAMHS and AMHS, undertaken in the English National Health Service (NHS). It included an audit of the policies and procedures relating to transition,¹⁰ a case-note survey and organisational analysis,¹¹ and a qualitative study of the views of service users, carers and mental health professionals on the process of transition.¹² This paper analyses data from TRACK’s case-note survey of adolescents’ progression through CAMHS/AMHS boundaries in order to separate the concepts of transfer and transition and to evaluate each process.

Method

Full details of the study method have been published.¹³ This retrospective case-note survey was undertaken in six mental health trusts (NHS provider organisations): three in Greater London and three in the West Midlands. The West Midlands trusts merged during the study. Together these trusts deliver specialist mental health services, free at the point of delivery, to a socio-demographically diverse population of 8.1 million in urban and rural areas. All specialist (secondary care) CAMHS teams referring to local AMHS were included. Highly specialist tertiary services, e.g. condition-specific services with a national catchment area (such as specialist national eating disorder services), were excluded because of atypical populations served and logistical problems created by their interface with AMHS spread nationally. To identify a cohort of adolescents reaching the age boundary for transition to AMHS in the preceding year (from September 2003 in London, from January 2006 in the West Midlands) we intended to use service databases and cross-reference with CAMHS clinician-generated lists.

Separate data extraction tools, available online,¹³ were used for cases transferred to AMHS (CAMHS and AMHS notes) and those not transferred (CAMHS notes only). Data on sociodemographic, clinical, transition pathway and outcome variables were extracted. Two researchers independently extracted data from five transferred cases from a different trust to test interrater reliability. Comparing 491 non-text variables, the error rate was less than 2%. The data extraction tools collected information on presenting problems rather than diagnoses, because non-psychiatrists within multidisciplinary CAMHS do not always use diagnoses in their practice. Subsequently, presenting problems were independently assigned to seven diagnostic groups by three CAMHS psychiatrists (M.P., T.F. and T.K.). When required, discussion facilitated consensus. Comorbidity was defined as the presence of more than one diagnostic category. The diagnostic groups were serious and enduring mental disorders (including schizophrenia, psychotic disorders, bipolar affective disorder, depression with psychosis);

emotional/neurotic disorders (including anxiety, non-psychotic depression, post-traumatic stress disorder, obsessive-compulsive disorder); eating disorders (anorexia nervosa, bulimia nervosa, atypical eating disorder); conduct disorders (including other behavioural disorders); neurodevelopmental disorders (including pervasive developmental disorders, attention-deficit hyperactivity disorder, intellectual disabilities); substance use disorders (alcohol and/or drug misuse); and emerging personality disorder.

There were four criteria for optimal transition: continuity of care (either engaged with AMHS 3 months after transition or appropriately discharged); a period of parallel care or joint working between CAMHS and AMHS; at least one transition planning meeting, involving the adolescent, with or without a carer, and key AMHS and CAMHS professionals, prior to the handover of care; and information transfer (any of the following transferred from CAMHS to AMHS: referral letter, summary of CAMHS contact, CAMHS notes, a contemporary risk assessment). These criteria were developed from literature on continuity of care and CAMHS transition protocols.^{10,14}

The term 'cases' is used as only case notes were consulted. The term 'transferred' is applied to cases referred to and seen by AMHS, and the completed process of data extraction from case notes is referred to as 'tracking'. Wandsworth Research Ethics Committee approved the study.

Results

Databases did not exist at two sites and were poorly functional in relation to case ascertainment at four sites. Collating clinician-generated lists was a protracted process and, given the budget and time frame of the study, data extraction was completed on 155 of 186 cases identified in total. There was some variation between sites in relation to proportions of cases for which tracking was completed: 27/27 for London site 1, 50/60 for London site 2, 36/44 for London site 3; 5/5 for West Midlands site 1, 6/8 for West

Midlands site 2, 31/31 for West Midlands site 3. One case was excluded from subsequent analysis as transition was to a neurology service.

The sample consisted of 78 (51%) males and 76 females, with a mean age of 18.1 years (s.d. = 0.8) at the time of data collection. The majority ethnic group was White (31%), followed by Black (23%). No ethnicity was recorded in 27% of cases. The majority of individuals (76%) spoke English as their first language. Most adolescents lived with their parents (71%) and were in either education or employment (60%). Most presenting problems at the time of transition fell into the diagnostic category of emotional/neurotic disorders (51%, $n=78$), followed by neurodevelopmental disorders (25%, $n=38$), serious and enduring mental disorders (22%, $n=34$), substance misuse (9%, $n=14$), conduct disorders (4%, $n=6$), eating disorders (4%, $n=6$) and emerging personality disorder (3%, $n=4$). In five cases (3%) the presenting problem was not recorded. Almost a fifth had comorbidity (19%, $n=29$).

Transfer

Of the 154 cases that crossed the transition boundary (i.e. the age boundary between CAMHS and AMHS), 131 (85%) were thought suitable by CAMHS clinicians for transfer to adult services, 102 (66%) were referred and 90 (58%) were accepted by AMHS (Fig. 1). In 76 (49%) cases, at least one appointment at AMHS was attended, i.e. transfer was achieved. The reasons for non-referral to AMHS were refusal by the adolescent or parents/carers; CAMHS clinicians thinking AMHS would not accept the referral or had no appropriate service; or CAMHS still planning to refer to AMHS. Adult mental health services refused to accept 7 (5%) and a decision by AMHS was pending in 5 (4%) cases.

Table 1 summarises what happened to the 90 cases referred to and accepted by AMHS. Seven (8%) had no appointment arranged, for reasons including non-response to AMHS attempts to arrange the appointment (3%, $n=3$), the adolescent's

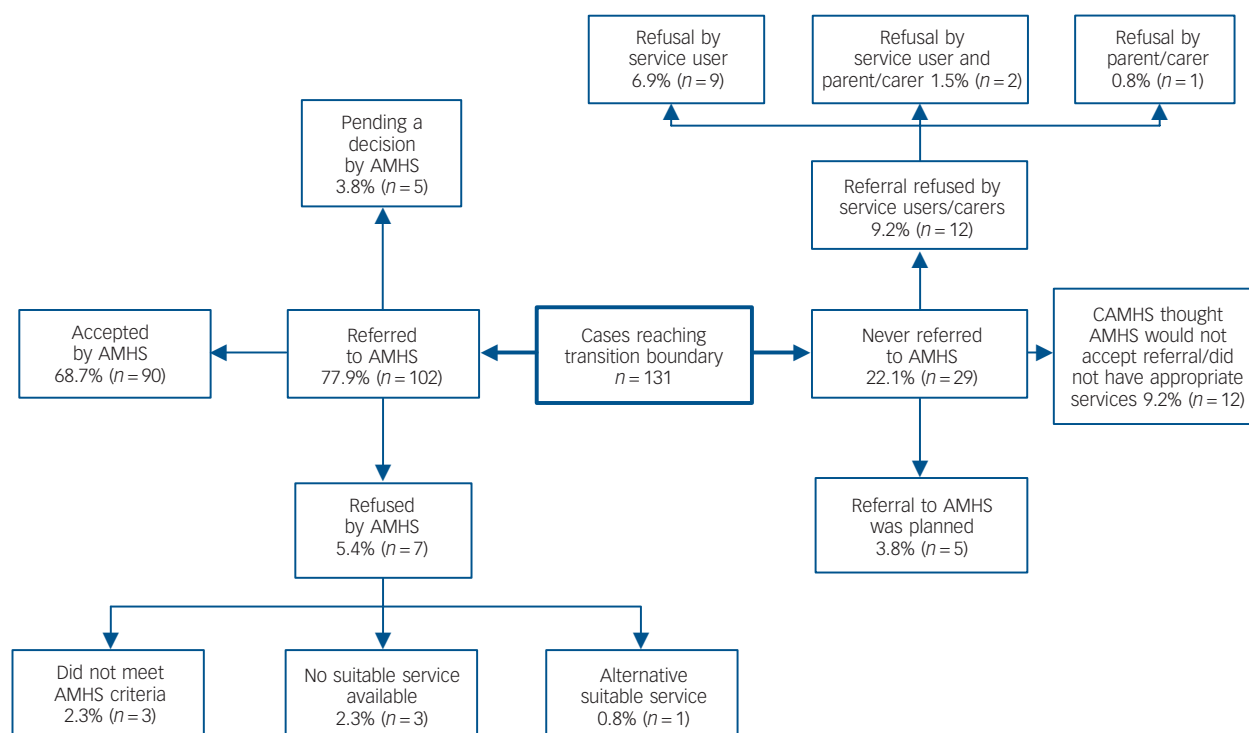


Fig. 1 Crossing the transition boundary between child and adolescent mental health services (CAMHS) and adult mental health services (AMHS) with ongoing clinical need.

Table 1 What happened to cases accepted by adult mental health services

	Cases accepted by AMHS (<i>n</i> = 90) <i>n</i> (%)	Cases offered appointments by AMHS (<i>n</i> = 83)		
		Initial assessment appointments attended <i>n</i> (%)	Subsequently offered assessment appointments attended, <i>n</i> (%)	Any assessment appointments attended <i>n</i> (%)
Appointment arranged	83 (92)	63 (76)	13 (16)	76 (92)
Appointment not arranged	7 (8)	NA	NA	NA
Subsequent contact				
Attended follow-ups	58 (64)	50 (60)	8 (10)	58 (70)
Discharged	15 (17)	12 (15)	3 (4)	15 (18)
Disengagement followed by a return to care under mental health legislation	1 (1)	1 (1)		1 (1)
Discharge followed by a return to care under mental health legislation	1 (1)		1 (1)	1 (1)
Lost to follow-up	1 (1)		1 (1)	1 (1)
Failure to attend appointments offered	7 (8)			

AMHS, adult mental health services; NA, not applicable.

disengagement with CAMHS (1%, *n* = 1) and administrative failure (3%, *n* = 3). Of 83 initial AMHS appointments offered, 63 (76%) were attended. In 20 (24%) of the 90 cases accepted by AMHS the person did not attend the first appointment offered. Of these, 16 were offered second appointments and 4 discharged. Only a quarter of the 16 second appointments were attended. The remaining 12 were offered a third appointment, 9 of which were attended. No fourth appointment was offered. The adult service succeeded in seeing 13 of 20 cases (65%) where the initial appointment was not attended. Transfer was therefore achieved in 92% of cases offered appointments by AMHS, 84% of cases accepted by AMHS, 74% of cases referred by CAMHS and 58% of cases crossing the transition boundary with ongoing clinical need. By the end of the study, of the 131 cases crossing the transition boundary with ongoing clinical need, 60 (46%) remained open to follow-up by AMHS and 15 (12%) had been discharged following attendance at AMHS.

Transition

Among the 90 cases accepted by AMHS, the element of optimal transition most often met was continuity of care (70%, *n* = 63), followed by having had at least one transition planning meeting (40%, *n* = 36), good information transfer (27%, *n* = 24) and a period of parallel care (24%, *n* = 22). No criterion for optimal transition was experienced in 13 (14%) cases, only one criterion in 33 (37%), two criteria in 24 (27%), three in 16 (18%) and all four criteria in 4 (4%) cases. In the four cases that met criteria for optimal transition, two of the individuals were male, three were 18 years old at the time of transfer and one was 19, and all four were from ethnic minority backgrounds. Three had a diagnosis of a serious and enduring mental disorder and had been admitted to hospital at some point while attending CAMHS (two under mental health legislation). All four were on medication at the time of transition and came from London (two each from London trusts 2 and 3).

Discussion

In our sample more than five-sixths of young people leaving CAMHS were thought to have ongoing clinical need. Transfer of care was common but good transitional care was rare. Many individuals transferred successfully to AMHS without experiencing good transitional care. Transfer failed more often because of young people's refusal to accept referral to adult

services, and CAMHS clinicians' failure to refer, than AMHS refusing to accept referrals or discharging young people who did not attend the first appointment offered; the latter two reasons have been assumed to be the main reasons in the past.^{15,16} The majority of transferred adolescents experienced continuity of care but other elements of good transition such as transition planning meetings, periods of joint care and good transfer of information were far less frequent. Transfer does not, therefore, guarantee successful transition. Moreover, poor transition, in the context of successful transfer, does not necessarily bode ill: TRACK's case-note survey and qualitative study findings indicate that despite suboptimal transitions most young people stayed engaged with AMHS and reported improvement in their mental health.^{11,12} Small sample sizes indicate that further investigation of the relevance of key elements of good transition to better outcomes is necessary. The requirement for a better-quality, more generalisable evidence base is reiterated by recent reviews of the broader paediatric to adult transitions literature.^{17,18}

Transfer

On the whole transfers were found to be successful in this study, although a small minority of those referred were not accepted by AMHS. An Australian study of a CAMHS with different referral criteria, however, found that many adolescents were not accepted by a youth mental health service (for 15–24 year olds).¹⁹ One of the two main reasons for a quarter of the TRACK cases with ongoing clinical need failing to be transferred was CAMHS clinicians thinking that AMHS would reject the referral or would not have appropriate services. We cannot say whether these assumptions, based on experience or knowledge of AMHS referral criteria, were correct. We can say that the majority of those referred to AMHS in this sample were accepted and, by the end of the study, almost three-quarters of transferred cases remained open to AMHS, sometimes following multiple offers of an 'initial' appointment. Overall, more cases were 'not referred' than 'not accepted'. Making referrals based on need, regardless of assumptions about whether adult services will accept them, may highlight the types of adult service that are inadequate or non-existent. This should aid the development of appropriate services for young adults.

A second reason for failure to transfer despite ongoing need was the young person's refusal to be referred to AMHS. Future research, policy and service development should separate cases of young people who fail to transfer for other reasons from cases

of those who (or whose parents) choose not to engage with adult services even though professionals think this would be of benefit. Some young people or parents/carers might prefer private or voluntary sector care. Lack of transfer to AMHS is therefore not necessarily equivalent to needs remaining unmet. We do not know, however, what proportions find alternative suitable care, and we need to remain concerned about those who fail to transfer to AMHS.

Poor adherence to health supervision around the time of transition is also noted in relation to young people with chronic physical health problems such as diabetes.²⁰ Disengagement with services may result from developmental characteristics such as emerging autonomy and distrust of authority figures, changing family relationships and heightened influence by peers alongside young people's need to acquire many life skills,²¹ or concerns about confidentiality, knowledge about and accessibility of the service proposed.²²

Equitable, accessible, acceptable, appropriate and effective youth-friendly services, as promoted by the World Health Organization (WHO),²³ or separate youth services, as suggested by user-oriented research,²⁴ might help with acceptance of referral to adult services. There remains debate, however, about whether separate youth mental health services are preferable.^{25,26} There may be specific reasons why young people and carers are reluctant to be seen at AMHS, such as stigma²⁷ and lack of insight into some mental health problems.²² Also, some young people do not seek help for mental health problems, are not referred to CAMHS,²⁸ or disengage with the service; adult services may therefore need to engage adolescents who never engaged with CAMHS. Push and pull factors remain important beyond the transition boundary between CAMHS and AMHS – for instance, the proportion of young people receiving public mental health services in the USA declines from the age of 17 years, and only a quarter of those aged 18–20 years who want services receive them.²¹

Transition

Our study provides evidence of extremely poor quality of transition: less than 5% of transfers fulfilled all four criteria of optimal transition. This is despite the use of tight definitions of transition and continuity of care, the latter compatible with the wider literature.^{9,14} A broader definition of transitional care for young people with serious mental health conditions was used in a study from the USA of transitional services providing options such as supported housing, vocational support, preparation for independent living and the availability of dual diagnosis services, e.g. mental health and substance misuse services.²⁹ It found that a quarter of child and a half of adult State mental health systems offered no transitional service of this broad type. Most options were available in less than 20% of states.²⁹

The paediatric evidence base has produced guidance on how to 'do' transition well, for instance, by focusing on the 'four Ps' of people (the young person, parents or caregivers, a transition coordinator and keyworker, interested adult services, primary care, multidisciplinary/multi-agency networks and professional training), process (written transition policy, transition programmes, and evaluation and audit), paper (informational resources and administrative support) and place (youth-friendly spaces).³⁰ It highlights paediatric and adult service staff needs for training and attitudinal changes towards each other's services, while young people need to be trained and empowered to become effective partners in their own transition.³¹ Addressing transitions in education and employment, adolescent health (fertility and sexual health), ethical and legal issues (consent, competence and

autonomy) and societal factors (health inequalities and urban/rural differences) at the time of transition between paediatric and adult services may also be key.³² This might indicate the need for generic adolescent health services rather than condition-specific (e.g. psychosis-specific) or youth mental health services.

This analysis of the TRACK data indicates that transition and transfer are related, but different, processes. Differences have implications for services, which are seeking to improve experiences and outcomes for individuals crossing the paediatric/adult service interface, and for researchers, in relation to the methodology of future transitions research. Transfer can be of use even if transition has been poor;¹¹ transition processes and policies may be followed impeccably yet still result in failure to transfer (e.g. if the patient chooses not to go to adult services for some reason). There may be barriers to transfer that are quite different from barriers to transition. Services and researchers need to address both aspects.

Limitations of the study

There were significant problems in case ascertainment. Databases either did not exist or could not provide accurate and suitable information. This reflects the poor level of data collection (at the time) in the NHS. Information technology has a low priority within the NHS, which is funded from a public service budget, with no need to bill for individual contacts, as in private practice or insurance-dependent services. Some clinicians felt too busy to provide relevant information, some could not be contacted because of high staff turnover, and some could not accurately recall appropriate cases because of high case-loads. Some case notes were difficult to locate or access. Clinicians might be most likely to recall cases where transition had been problematic. Even if every unascertained case had an ideal transition, we still document a worrying number of poor experiences. Also, case notes may not accurately reflect the quality and content of services delivered. The population studied was large and diverse, making findings generalisable to other services in the UK. The main conclusion of this paper, that future service development and research need to explicitly address barriers and facilitators of both transfer and transition, should be of interest to those working in different service structures internationally.

Moli Paul, MRCPsych, PhD, University of Warwick, Warwick Medical School, Coventry; **Tamsin Ford**, MRCPsych PhD, Peninsula Medical School, University of Exeter; **Tami Kramer**, MRCPsych, Imperial College London, St Mary's Campus, London; **Zoebia Islam**, PhD, University of Warwick, Warwick Medical School, Coventry; **Kath Harley**, DClinPsy, South London and Maudsley National Health Service Foundation Trust, London; **Swaran P. Singh**, FRCPsych, DM, University of Warwick, Warwick Medical School, Coventry, UK

Correspondence: Dr Moli Paul, Room B153, The Medical School Building, Gibbet Hill Campus, University of Warwick, Coventry CV4 7AL, UK. Email: moli.paul@warwick.ac.uk

Funding

The TRACK study was funded by the National Institute for Health Research Health Services and Delivery Research programme (www.sdo.nihr.ac.uk).

References

- 1 Blum RW, Garell D, Hodgman CH, Jorissen TW, Okinow NA, Orr DP, et al. Transition from child-centered to adult health-care systems for adolescents with chronic conditions. A position paper of the Society for Adolescent Medicine. *J Adolesc Health* 1993; **14**: 570–6.
- 2 Winters NC, Pumariga A. Practice parameter on child and adolescent mental health care in community systems of care. *J Am Acad Child Adolesc Psychiatry* 2007; **46**: 284–99.

- 3 Department of Health. *New Horizons: A Shared Vision for Mental Health*. Department of Health 2009.
- 4 Kennedy I. *Getting It Right for Children and Young People – Overcoming Cultural Barriers in the NHS So As To Meet Their Needs*. Department of Health, 2010.
- 5 Birchwood M, Singh SP. Mental health services for young people: matching the service to the need. *Br J Psychiatry* 2013; **202** (suppl 54): s1–2.
- 6 Lin A, Reniers RLEP, Wood S. Clinical staging in severe mental disorder: evidence from neurocognition and neuroimaging. *Br J Psychiatry* 2013; **202** (suppl 54): s11–7.
- 7 Jones PB. Adult mental health disorders and age at onset. *Br J Psychiatry* 2013; **202** (suppl 54): s5–10.
- 8 McDonagh JE, Kelly DA. Transiting care of the pediatric recipient to adult care givers. *Ped Clin North Am* 2003; **50**: 1561–83.
- 9 Forbes A, While A, Ullman R, Lewis S, Mathes L, Griffiths P. *A Multi-Method Review to Identify Components of Practice Which May Promote Continuity in the Transition from Child to Adult Care for Young People with Chronic Illness or Disability. Report for the National Coordinating Centre for Service Delivery and Organisation Research and Development (NCCSDO)*. NCCSDO, 2001.
- 10 Singh SP, Paul M, Ford T, Kramer T, Weaver T. Transitions of care from child and adolescent mental health services to adult mental health services (TRACK study): a study of protocols in Greater London. *BMC Health Serv Res* 2008; **8**: 1–7.
- 11 Singh SP, Paul M, Ford T, Kramer T, Weaver T, McLaren S, et al. Process, outcome and experience of transition from child to adult mental healthcare: multiperspective study. *Br J Psychiatry* 2010; **197**: 305–12.
- 12 Hovish K, Weaver T, Islam Z, Paul M, Singh SP. Transition experiences of mental health service users, carers and professionals: the TRACK study. *Psychiatr Rehab J* 2012; **11**: 68.
- 13 Singh S, Paul M, Islam Z, Weaver T, Kramer T, McLaren S, et al. *Transition from CAMHS to Adult Mental Health Services (TRACK): A Study of Service Organisation, Policies, Process and User and Carer Perspectives. Report for the National Institute for Health Research Service Delivery and Organisation programme*. NCCSDO, 2010.
- 14 Freeman G, Crawford M, Weaver T, Low J, de Jonge E. *Promoting Continuity of Care for People With Severe Mental Illness Whose Needs Span Primary, Secondary and Social Care. A Multi-Method Investigation of Relevant Mechanisms and Contexts*. National Coordinating Centre for NHS Service Delivery and Organisation, 2002.
- 15 Davis M, Sondheimer DL. State child mental health efforts to support youth in transition to adulthood. *J Behav Health Serv Res* 2005; **32**: 27–42.
- 16 Singh SP. Transition of care from child to adult mental health services: the great divide. *Curr Opin Psychiatry* 2009; **22**: 386–90.
- 17 Crowley R, Wolfe I, Lock K, McKee M. Improving the transition between paediatric and adult healthcare: a systematic review. *Arch Dis Child* 2011; **96**: 548–53.
- 18 Watson R, Parr JR, Joyce C, May C, Le Couteur AS. Models of transitional care for young people with complex health needs: a scoping review. *Child Care Health Dev* 2011; **37**: 780–91.
- 19 Cosgrave EM, Yung AR, Killackey EJ, Buckby JA, Godfrey KA, Stanford CA, et al. Met and unmet need in youth mental health. *J Ment Health* 2008; **17**: 618–28.
- 20 Pacaud D, Yale J, Stephure D, Trussell R, Davies H. Problems in transition from pediatric care to adult care for individuals with diabetes. *Can J Diabetes* 2005; **29**: 13–8.
- 21 Davis M. Addressing the needs of youth in transition to adulthood. *Adm Policy Ment Health* 2003; **30**: 495–509.
- 22 Zwaanswijk M, van der Ende J, Verhaak PFM, Bensing JM, Verhulst FC. Factors associated with adolescent mental health service need and utilization. *J Am Acad Child Adolesc Psychiatry* 2003; **42**: 692–700.
- 23 World Health Organization. *Adolescent Friendly Health Services: An Agenda for Change*. WHO, 2002.
- 24 YoungMinds. *A Call to Action: Commissioning Mental Health Services for 16–25 Year-olds*. YoungMinds, 2006.
- 25 McGorry PD. Is early intervention in the major psychiatric disorders justified? Yes. *BMJ* 2008; **337**: a695.
- 26 Pelosi A. Is early intervention in the major psychiatric disorders justified? No. *BMJ* 2008; **337**: a710.
- 27 Jorm AF, Wright A. Influences on young people's stigmatising attitudes towards peers with mental disorders: national survey of young Australians and their parents. *Br J Psychiatry* 2008; **192**: 144–9.
- 28 Verhulst FC, van der Ende J. Factors associated with child mental health service use in the community. *J Am Acad Child Adolesc Psychiatry* 1997; **36**: 901–9.
- 29 Davis M, Geller JL, Hunt B. Within-state availability of transition-to-adulthood services for youths with serious mental health conditions. *Psychiatr Serv* 2006; **57**: 1594–9.
- 30 McDonagh JE. Transition of care: how should we do it? *Paediatr Child Health* 2007; **17**: 480–4.
- 31 Viner RM. Transition of care from paediatric to adult services: one part of improved health services for adolescents. *Arch Dis Child* 2008; **93**: 160–3.
- 32 Royal College of Physicians of Edinburgh Transition Steering Group. *Think Transition: Developing the Essential Link Between Paediatric and Adult Care*. Royal College of Physicians of Edinburgh, 2008.

Special article

The divide between child and adult mental health services: points for debate

Clare Lamb and Margaret Murphy

Summary

This discussion paper outlines our personal views for debate on some of the complexities inherent in the crucial task of improving mental health services for young people in the UK.

Declaration of interest

None.

The issue of how young people move from child and adolescent mental health services (CAMHS) to adult mental health services (AMHS) has been the subject of a number of policy documents and studies over the past 20 years.^{1–11} Yet despite this work and some positive changes, recent research demonstrates that problems persist.^{8,11–13} Lin *et al*, Jones and Chanan & McCutcheon in this supplement have described the developmental context and nature of mental disorders in late adolescence and early adulthood;^{14–16} here we focus more narrowly on the nature and context of the services themselves.

The debate about the so-called 'CAMHS–AMHS divide' is generally focused on reported difficulties in transition from CAMHS to secondary care AMHS.^{7–10} For the purposes of argument this is where we start our discussion, but in terms of considering potential solutions we shall not confine our thoughts to these traditional service structures. Difficulties in transition from one service to another are not confined to mental health services,¹⁷ nor to the adolescent–young adult transition. In considering the CAMHS–AMHS divide and potential solutions it is important to understand the nature of the services on either side of that divide. There are differences in the remits of the two types of service that have contributed to differences in theory and practice, including differences in eligibility thresholds for referral and in the level and style of intervention.^{3,7}

The CAMHS population

Child and adolescent mental health services in the UK are grouped into:

- (a) universal services, such as general practitioners (GPs), health visitors and school nurses;
- (b) targeted services, such as Mental Health in Schools projects and mental health services for children who are 'looked after' by the local authority;
- (c) specialist services such as specialist multidisciplinary mental health teams.

These different layers of service are commissioned and provided by government departments of health, education and social care. Funding streams and organisation are subject to a degree of local variation. Children and young people with mental disorders may be provided for at any of the three levels. Many young people and their families may be receiving services from several agencies simultaneously; in addition, the adults in the household may have mental or physical health problems.

Specialist CAMHS provides care for children and young people with established mental disorders. However, the profile of disorders treated differs from that for secondary care AMHS.

This is in part a consequence of the pattern and nature of mental disorder in children and young people. Children's mental health services provide for children and young people with a wide range of disorders, including mental illnesses such as depression, anxiety, eating disorders, obsessive–compulsive disorder and psychosis, as well as autism spectrum disorders, intellectual disabilities, attention-deficit hyperactivity disorder (ADHD) and conduct disorder. In addition, specialist and targeted services provide for children and young people in difficult circumstances that put them at risk of mental disorder, for example those in the care system, young people involved with the criminal justice system, and children and young people who have experienced abuse and neglect. Services also provide interventions for children with high levels of impairment related to mental health difficulties, but who might not reach diagnostic criteria for mental disorder.

The remit of secondary care AMHS is narrower in the main, focusing on adults with more severe mental illness. The majority of adults with mental disorders are cared for within primary care. These differences in remit occur alongside a range of other differences between child and adult services. There are differences in training between professionals working in the two services. Specialist and targeted CAMHS and AMHS are often provided by different organisations. Current commissioning models generally place CAMHS and AMHS commissioning within different frameworks and structures. This has the potential for AMHS and CAMHS commissioning strategies and care pathways to develop separately. At government policy level CAMHS and AMHS have previously been planned separately. For example, in England, Scotland and Wales there are separate national service frameworks and national plans involving different government departments.^{18–22} Unlike adult policy, CAMHS health policy links to the Department of Education.

The consequence of these service differences is that young people in receipt of a service from CAMHS may find that on reaching adulthood their condition and presentation has not changed but secondary care AMHS are not configured to provide for them. If there is no alternative service available in primary care or the voluntary sector, young people and their families are left to cope alone.

The transitions cohort

Many young people will experience some form of transition from CAMHS and there are a number of possible transitional trajectories:

- (a) young people who have a severe mental illness and who are accepted by secondary AMHS;⁸

- (b) young people who have received a service from specialist CAMHS and are likely to have ongoing problems but who are not accepted by AMHS or not referred because it is believed they would not be accepted;⁸ this group often includes young people with autism spectrum disorders, ADHD and emergent personality disorder and some young people with depression, anxiety disorders, obsessive-compulsive disorder and eating disorders;^{11–13}
- (c) young people experiencing a high degree of impairment in the context of mental health problems that may not clearly fulfil criteria for a particular disorder; they have received a service from either specialist or targeted CAMHS, but might not be referred to or accepted by secondary care AMHS because they do not have a severe mental illness;^{7,8}
- (d) young people who have mental health problems but whose needs have been met outside the healthcare system, in settings such as special schools and pupil referral units, and/or by social care;
- (e) young people who would not require transition if CAMHS worked with them for longer.

In a study of transition processes to AMHS, Singh *et al* reported that over 80% of cases were considered suitable for transfer by CAMHS, but a third were not referred.⁸ The adult services accepted 93% of all referrals but 25% of cases accepted by AMHS were discharged without being seen. Even among those who crossed the gap few experienced 'optimal transition', defined as at least one transition planning meeting, a period of joint working between CAMHS and AMHS, good information on transfer and being engaged with AMHS 3 months following transfer.⁸

The problem of transition of young people who meet the criteria of current adult services can and should be solved by improved working between current service providers. However, the problem of how to improve the experiences of young people with transition trajectories that do not meet current eligibility criteria for AMHS is challenging for our current service structures. Tackling these problems requires action on the part of those commissioning and planning services as well as those providing services.¹² The key issue for young people who do not meet eligibility criteria for AMHS is that no service currently exists to meet their needs. The improvement of mental health services for these young people requires extension of adult services to offer interventions for young adults with developmental problems, high-risk behaviours associated with emerging personality disorders and those with severe anxiety and affective disorders. This requires new resources whatever the service design. It also requires the creation of more collaborative links with primary care and other agencies.²³

What young people and their families tell us

Studies show that young people, their families and carers want their views to be taken seriously and to participate actively in the process of transition.^{24–26} Studies have elicited the views of young people and of parents and carers about what they want from services.^{4,27–30} Young people value good information, consistent support from a keyworker, and flexible, non-stigmatising community-based services appropriate for their age group.

'Young people have a lot of problems and it is easier for them to walk into one place where they sort everything out. I wouldn't want to keep explaining my situation over and over again. It's just too upsetting.'²⁹

'Before I moved here, I didn't go to anyone for help . . . when I did try to go to someone for help, they would turn me away, so I ended up drinking, cutting myself,

finding myself in arguments. But since I've been here my keyworker, she's brilliant, I love her to bits and I could go to her about anything.'³¹

Young people and their parents describe the change in service philosophy between child and adult services confusing, especially in relation to the role and involvement of families. Professionals can experience difficulties in meeting the expectations of young people and families at transition. These problems are not unique to mental health services as they also occur in physical medicine.¹⁷ Parents have expressed concern about the lack of services for specific groups of young people, including those with ADHD, autism spectrum disorders and emerging personality disorders.¹³

'The lady at CAMHS kept everyone together, but everything was lost completely through the transition phase. She had meetings with the adult teams and got absolutely nowhere. As soon as it stopped, as soon as she was out of the picture, everything went to pieces.' (Parent of a young adult)¹²

'I am the carer of a now adult son . . . with Asperger's syndrome . . . My son's transition into adulthood was a nightmare both for him and for the rest of the family. There was no transition from CAMHS to adult mental health services and there was no clinician in my local health trust with any expertise regarding Asperger's syndrome . . . no service exists where I live, so I just struggle on supporting him, looking for more appropriate help.'¹³

'It's sometimes said from the adult services that CAMHS transfers are quite difficult because they've been pampered by CAMHS services . . . maybe the care coordinators in the adult teams feel a wee bit inadequate by comparison . . . definitely CAMHS transfers that come to us are really disappointed by what we can offer and can get really upset initially because they feel their needs aren't being met.' (AMHS keyworker)⁸

In the past decade, in order to combat the gaps in service and inequity of provision, there has been considerable impetus in the development of innovative services across the UK that promote greater working between CAMHS and AMHS. However, many practice developments and service models for improving transitions are at an early stage of development and there are few robust, effective studies currently available.^{11,13}

In England the Department of Health and the Department for Education sponsored the National Mental Health Development Unit and National CAMHS Support Service to work in partnership with the Social Care Institute for Excellence on a project to provide a series of resources to improve transitions for young people with mental health problems.¹² The project identified a series of case examples of services in the UK for older adolescents and young adults. These included clinical liaison/link posts, disorder-specific services (e.g. early intervention in psychosis) and in-reach to primary care. At an international level there has been considerable interest in developing youth mental health services, often spanning an age range of 16–25 years. Examples include Orygen Youth Health in Australia, Headstrong in Ireland, and in England Youthspace in Birmingham and the City & Hackney CAMHS Extended Service in London (see McGorry *et al*, this supplement).³² In some areas non-statutory youth services lead multi-agency resourced and managed young people's centres which bridge the traditional transition age gap.³² These services, such as 'The Zone' in Plymouth (www.thezoneplymouth.co.uk), operate flexible opening, drop-in facilities which link to statutory mental health services in a variety of ways. All these services have required significant new investment.

Current context

There is some room for optimism in that the new English mental health strategy and parallel developments in the other UK jurisdictions recognise the importance of effective intervention early in life and have focused attention on the transitions issue. However, this should be balanced against the recognition that the UK is in a period of financial austerity when the level of resources available to specialist CAMHS and AMHS will become

an even greater challenge, with planned public sector savings, particularly in social care and education in many areas.³⁴ The potential consequences of lower investment in AMHS and CAMHS could include adult secondary care mental health and adult social care services needing to raise thresholds consequent to decreased resources; voluntary sector providers, who provide many aspects of youth services, experiencing instability as funding becomes more difficult; specialist CAMHS experiencing reductions in resource, which could lead to reduced capacity and increased thresholds; and reductions in the capacity and provision of local authority services for children and young people through youth service cuts. In addition, young people are particularly affected by the current economic recession. It is known that there are strong links between mental well-being and employment and it is of great concern that the highest unemployment rates in the UK are experienced by those aged 16–24 years.³⁵ The lack of opportunities for work and the consequent increased stress that many young people face leave them at greater risk of developing mental health problems and pose further difficulties for those recovering from pre-existing mental disorders.

Ways forward

It is clear from the available research that significant improvements are required in the implementation of high-quality policy and practice around transition from CAMHS to AMHS.¹¹ In the case of young people who meet current eligibility criteria for AMHS, this can be achieved through providers working together more effectively, and involving young people and their families in improving transition processes.⁸ However, if we are to provide services for young people who currently have no service to progress to, a broadening of eligibility criteria and reduction of threshold of referral for young people to adult services are required. It is important to consider whether broadening the eligibility for adult-led mental health services for young adults would result in a reduction in services for the very young. It might be possible to achieve improved access and a broader range of interventions for young adults through working more closely with the Youth Information Advice Counselling and Support services,²⁹ and with GP-led primary care services such as the Young People's Clinic in Herne Hill, South London.²³

Alternatively, should we consider separate youth services that provide a broad range of evidence-based interventions up to the age of 25 years? If this is the case, then what should be the lower age range for a youth service? Some argue for 12 years, the age soon after transfer to secondary school in the UK. Others argue for 16 years, the age at which compulsory education ends, at which a young person can give consent to medical interventions and can consent to sexual relations. There is also an argument for a model of youth service within AMHS that commences at 18 years, when a young person is no longer viewed as a child under the Children Act 2004 and is considered an adult by the UK legal system. It is important to consider the developmental age of an individual young person in addition to chronological age and the need for flexibility across any age boundary. Flexibility across age boundaries requires agreement between commissioners and effective collaboration and good working relationships between professionals. Attendance at local regular joint meetings and joint training events by CAMHS and AMHS professionals have been shown to improve working relationships and create opportunities for collaborative work.

We must be sure to improve the accessibility and range of mental health interventions in a youth-friendly context while

avoiding difficulties inherent to the creation of new boundaries. New age boundaries could act as barriers and create the need for new transitions that do not match those of other agencies working to meet the needs of young people, including education and social care. This could lead to fragmentation of important multi-agency links. In solving one set of problems we must ensure we do not create others, and be alert to the risk of unintended consequences.

We need to develop services for our young people but be mindful not to do this at the expense of universal early intervention and prevention with the very young. Any solution regarding service models or service re-design is unlikely to be a 'one size fits all' solution, both at individual and service level. Different solutions will fit different local situations. What is not in doubt is that clinicians and commissioners of both AMHS and CAMHS need to work together. Resources are tight and each will need to support the other at different times in order to achieve the best services for young people. It is worth acknowledging that for clinicians working with adolescents and young adults there are more similarities than there are differences, and we can achieve great things for our young people by working closely together and with the respective other agencies that engage with this population. However, in all circumstances if we are to improve transitions and provide interventions to meet the needs of those not currently eligible for adult services, new resources and different commissioning structures will be required. Professionals from both CAMHS and AMHS must work collaboratively with each other, with primary care and with commissioners to find new ways to achieve the services our young people need.

Clare Lamb, MB BS, FRCPsych, North Wales Adolescent Service, Conwy; **Margaret Murphy**, MB ChB, MRCPsych, Child and Adolescent Mental Health Service, Brookside Clinic, Cambridge, UK

Correspondence: Dr Clare Lamb, North Wales Adolescent Service, Abergele, Conwy LL22 8DP, UK. Email: Clare.Lamb@wales.nhs.uk

References

- 1 Kay H. *Bright Futures*. Mental Health Foundation, 1999.
- 2 Select Committee on Health. Transitions between child/adolescent and adult services. In *Fourth Report. Provision of NHS Mental Health Services*. TSO (The Stationery Office), 2000.
- 3 Reder P, McClure M, Jolley A. *Family Matters: Interfaces Between Child and Adult Mental Health Services*. Routledge, 2000.
- 4 Smith K, Leon L. *Turned Upside Down: Developing Community Based Crisis Services for 16–25 Year Olds Experiencing Mental Health Crisis*. Mental Health Foundation, 2001.
- 5 Street C. *Whose Crisis? Meeting the Needs of Children and Young People With Serious Mental Health Problems*. YoungMinds, 2000.
- 6 Richards M, Vostanis P. Interprofessional perspectives on transitional mental health services for young people aged 16–19 years. *J Interprof Care* 2004; **18**: 115–28.
- 7 Lamb C, Hall D, Kelvin R, Van Beinum M. *Working at the CAMHS/Adult Interface: Good Practice Guidance for the Provision of Psychiatric Services to Adolescents/Young Adults*. Royal College of Psychiatrists, 2008.
- 8 Singh SP, Paul M, Ford T, Kramer T, Weaver T, McLaren S, et al. Process, outcome and experience of transition from child to adult mental healthcare: multiperspective study. *Br J Psychiatry* 2010; **197**: 305–12.
- 9 McGorry PD, Purcell R, Hickie IB, Jorm AF. Investing in youth mental health is a best buy. *Med J Aust* 2007; **187**: s5–7.
- 10 McGorry P. The specialist youth mental health model: strengthening the weakest link in the public mental health system. *Med J Aust* 2007; **187** (suppl 7): s53–6.
- 11 Brodie I, Goldman R, Clapton J. *Mental Health Service Transitions for Young People*. Research Briefing 37. Social Care Institute for Excellence, 2011.

- 12 Appleton S, Pugh K. *Planning Mental Health Services for Young Adults – Improving Transition: A Resource for Health and Social Care Commissioners*. National Mental Health Development Unit, 2011.
- 13 Meier R. *Developing Services to Improve the Quality of Life of Young People with Neurodevelopmental Disorders, Emotional/Neurotic Disorders and Emerging Personality Disorder* (Occasional Paper OP77). Royal College of Psychiatrists, 2011.
- 14 Lin A, Reniers RLEP, Wood SJ. Clinical staging in severe mental disorder: evidence from neurocognition and neuroimaging. *Br J Psychiatry* 2013; **202** (suppl 54): s11–7.
- 15 Jones PB. Adult mental health disorders and age at onset. *Br J Psychiatry* 2013; **202** (suppl 54): s5–10.
- 16 Chanen AM, McCutcheon L. Prevention and early intervention for borderline personality disorder: current status and recent evidence. *Br J Psychiatry* 2013; **202** (suppl 54): s24–9.
- 17 Viner R. Transition from paediatric to adult care. Bridging the gaps or passing the buck? *Arch Dis Child* 1999; **81**: 271–5.
- 18 Department for Education and Skills, Department of Health. *National Service Framework for Children, Young People and Maternity Services*. Department of Health, 2004.
- 19 Department of Health. *National Service Framework for Mental Health: Modern Standards and Service Models*. Department of Health, 1999.
- 20 National Assembly for Wales. *Adult Mental Health Services: A National Framework for Wales*. National Assembly for Wales, 2002.
- 21 National Assembly for Wales. *National Service Framework for Children, Young People and Maternity Services in Wales*. National Assembly for Wales, 2006.
- 22 Children and Young People's Mental Health. *A Framework for Promotion, Prevention and Care*. Scottish Executive, 2006.
- 23 Appleton S, Pugh K. Practice example 6. Herne Hill Group Practice, Lambeth. In *Planning Mental Health Services for Young Adults – Improving Transition: A Resource for Health and Social Care Commissioners*: 29. National Mental Health Development Unit, 2011.
- 24 Munoz-Solomando A, Townley M, Williams R. Improving transitions for young people who move from CAMHS to mental health services for adults: lessons from research and young people's and practitioners' experiences. *Curr Opin Psychiatry* 2010; **23**: 311–7.
- 25 Pugh K, Meier R. *Stressed Out and Struggling Project Report 1: Service-Mapping*. YoungMinds, 2006.
- 26 Street C, Stapelkamp C, Taylor E, Malek M, Kurtz Z. *Minority Voices: Research into Access and Acceptability of Services for the Mental Health of Young People from Black and Minority Ethnic Groups*. YoungMinds, 2005.
- 27 YoungMinds. *Emerging Practice Examples of Mental Health Services for 16–25 Year Olds*. YoungMinds, 2006.
- 28 Garcia I, Vasilou C, Penketh K. *Listen Up! Person-Centred Approaches to Help Young People Experiencing Mental Health and Emotional Problems*. Mental Health Foundation, 2007.
- 29 Lee S, Kenrick J. *A Proven Early Intervention Model: The Evidence for the Effectiveness of Youth Information Advice Counselling and Support Services (YIACS)*. Youth Access, 2010.
- 30 Marshall M, Rathbone J. Early intervention for psychosis. *Cochrane Database Syst Rev* 2006; **4**: CD004718.
- 31 Vasilou C. *Making the Link Between Mental Health and Youth Homelessness: A Pan-London Study*. Mental Health Foundation, 2006.
- 32 McGorry P, Bates T, Birchwood M. Designing youth mental health services for the 21st century: examples from Australia, Ireland and the UK. *Br J Psychiatry* 2013; **202** (suppl 54): s30–5.
- 33 Pugh K, McHugh AMF. *Stressed Out and Struggling: Two Steps Forward and One Step Back*. YoungMinds, 2006.
- 34 YoungMinds. *Briefing on Cuts to Children and Young People's Mental Health Services*. YoungMinds, 2011.
- 35 Office for National Statistics. *Labour Market Statistics*. ONS, 2011 (<http://www.labour.market@ons.gov.uk>).