Ethnicity and academic performance in the UK

White doctors and medical students do better but it is not clear why or what to do about it

MARK THOMAS/SPL

RESEARCH, p 584

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In the linked systematic review, Woolf and colleagues find that the ethnic origin of UK trained doctors and medical students is related to their academic performance at both undergraduate and postgraduate levels.[1]

Entry into medical schools is highly competitive, and most students who gain a place will have high grades, more than 60% will have parents who have professional backgrounds, and most will have come from selective secondary schools.[2 3] It seems counterintuitive that differences should arise in academic performance between these students from different ethnic groups who are born in the United Kingdom, especially when there is anonymous marking, "objective" clinical examinations, and machine marked tests.

A report commissioned by the Department for Education and Science on ethnicity and degree attainment in 2007 showed that black UK students and those from minority ethnic groups were less likely than white UK and Irish students to achieve good degrees (first class honours or upper second class honours).[3] The most recent figures showed that among those taking their first degree, 67% of white students achieved a good degree compared with 49% for black students and those from minority ethnic groups.[4] Much of the attainment gap can be explained by factors other than ethnicity—the 2007 analysis controlled for sex, previous attainment, disability, subject type, type of higher education institution, accommodation, and age—but it found that even after controlling for these factors, coming from a black and minority ethnic group still had a negative effect on degree attainment. Although medical degrees were excluded from the analysis because, in the UK, they are unclassified degrees, it is not surprising that the experience of black and ethnic minority students in medicine replicates that of those in other university disciplines. Woolf and colleagues have for the first time shown that medical schools cannot exempt themselves from the debate because the evidence they present suggests that at undergraduate and postgraduate level, assessments run by medical schools and royal colleges show that white students and qualified doctors do much better than their British non-white counterparts.

Since the introduction of the Race Relations Amendment Act 2000, universities have developed increasingly sophisticated means of collecting quantitative data about their students and can control for a range of factors when analysing data on degree attainment. This was a luxury not afforded to Woolf and colleagues, who relied on an extensive literature review to identify data sources from a vast range of smaller studies (22 reports covering 23 742 candidates). Previous research in this area has been criticised because results could not be generalised,[5] typically because they covered assessments in single examinations or a small cohort from a single year and were not UK based.[6] Although Woolf and colleagues' research is based primarily on data from medical schools in Nottingham and London, the numbers of students in their analysis allows them to draw valid conclusions. Not only did they carry out a rigorous meta-analysis but they also compared performance across a range of tests. Their results suggest that it is not only potential examiner biases or verbal communication skills that affect attainment. Problems with communication skills have often been suggested as a reason for differential attainment.[7-9] The authors could not properly control for previous attainment and socioeconomic status, but this does not make the findings less important—after all, the national data from a much more robust dataset on university attainment shows that even when these factors are controlled for, differences in attainment persist.

What should be done in the light of these findings? Although Woolf and colleagues' analysis points to a problem, it cannot explain why this problem occurs. It would be unwise to claim that discrimination is not a problem in medicine because racism in the medical profession has been well documented. It is apparent in admissions, in job applications, in how doctors are disciplined, and also in how they are paid. Woolf and colleagues' research makes an important contribution to this evidence base.[10-12] However, we should not just accept the situation because the solutions are complicated and contested.

It is unacceptable that ethnicity should be a factor determining the progress of students who enter medical school or qualified doctors who sit professional examinations. All medical schools and royal colleges should analyse their assessment results by ethnic group (they are already required by law to hold such data) and place their results in the public domain. Through this process we will identify examples of good practice that can be shared, and students and lecturers will be able to challenge their institutions to tackle the disparities that will inevitably be identified. Such complex problems are unlikely to have simple solutions—what happens in medical schools is a reflection of wider society.

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- Increasing diversity among clinicians (*BMJ* 2008;336:1082)
- Commentary: An "ethnic minority" medical student (*BMJ* 2008;337:a1240)

The solutions will be found through critically appraising assessment methods, curriculums, the way that we engage with students in an increasingly multicultural society, and the role models that we provide. A good precedent exists—at one time, few women entered medicine and fewer progressed in the profession, but current evidence shows that sex differences in achievement in university graduates have almost disappeared.[4]

1 Woolf K, Potts HWW, McManus IC. Ethnicity and academic performance in UK trained doctors and medical students: systematic review and meta-analysis. *BMJ* 2011;342:d901.
2 Dangerfield P. The demography of medical schools: a discussion paper. 2004. BMA.
3 Department for Education and Skills. *Widening participation in higher education*. 2003. www.bis.gov.uk/assets/biscore/corporate/migratedd/publications/e/ewparticipation.pdf.
4 Equality Challenge Unit. Equality in higher education—statistical report 2010. www.ecu.ac.uk/publications/equality-in-he-stats-10.
5 McManus I, Richards P. Prospective survey of performance of medical students during preclinical years. *BMJ* 1986;293:124-7.
6 Davidson RC, Lewis EL. Affirmative action and other special consideration admissions at the University of California, Davis, School of Medicine. *JAMA* 1997;278:1153-8.
7 Wass V, Hoogenboom R, Jones R, van der Vleuten C. Effect of ethnicity on performance in a final objective structured clinical examination: qualitative and quantitative study. *BMJ* 2003;326:800-3.
8 Beagan B. "Is this worth getting into a big fuss over?" Everyday racism in medical school. *Med Educ* 2003;37:852-60.
9 Esmail A, May C. Commentary: oral examinations—get them right or don't bother. *BMJ* 2000;320:375.
10 Esmail A, Everington S. Racial discrimination against doctors from ethnic minorities. *BMJ* 1993;306:691-2.
11 Esmail A, Everington S. Complaints may reflect racism. *BMJ* 1994;308:1374.
12 Esmail A, Abel P, Everington S. Discrimination in the discretionary points award scheme: comparison of white with non-white consultants and men with women. *BMJ* 2003;326:687-8.

Cystic fibrosis and survival in patients with advanced lung disease

rhDNase slows progression, and is strongly recommended in treatment guidelines

SIMON FRASER RVI/SPL

RESEARCH, p 586

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When Dorothy Andersen provided the first comprehensive description of cystic fibrosis in 1938,[1] survival was often measured in days and months. However, the introduction of penicillin resulted in children recovering from previously fatal infective respiratory exacerbations.[2] Subsequently, antibiotics given in combination with aggressive treatment of malnutrition resulted in some people with the condition living into adulthood.[3] Today, a myriad therapeutic strategies are directed at the infectious, inflammatory, and mucociliary defects in cystic fibrosis.[4] Combined with other non-pharmacological advances, children born today with cystic fibrosis are expected to live into their 50s.[5]

In the linked study, George and colleagues report on survival in a subgroup of adult patients with cystic fibrosis and advanced lung disease at the Royal Brompton Hospital, London, over 17 years (1990-2007).[6] Median survival improved by four years in those entering the cohort between 2002 and 2003 compared with those who entered between 1990 and 1991. Their data suggest that the widespread introduction of recombinant human deoxyribonuclease (rhDNase) at the centre in 1994 may be responsible for some of this improvement. If true, it would be the first treatment in the modern management era to be associated with an improvement in survival.

The authors found a 41% reduction (95% confidence interval 21% to 56%) in the adjusted hazard of death in patients with cystic fibrosis and advanced lung disease (defined as forced expiratory volume in one second (FEV_1) <30% predicted) prescribed rhDNase compared with those who were not prescribed rhDNase. Furthermore, increased use of rhDNase in 1994-5 was associated with a corresponding drop in the hazard ratio of death. However, can a strong causal relation between the use of rhDNase and survival be inferred from this observational study?

An association between rhDNase and a decreased hazard of death is biologically plausible. Treatment with rhDNase in patients with cystic fibrosis is associated with decreased pulmonary exacerbations,[7] and a recent systematic review found that rhDNase was well tolerated and improved lung function.[8] Pulmonary exacerbations and lung function are important predictors of survival.[9] Because patients with advanced lung disease have multiple exacerbations a year, long term administration of rhDNase could improve survival in patients with cystic fibrosis and advanced lung disease.

It is important to assess the study methods to determine whether the evidence is strong enough to alter current rhDNase practice patterns. The authors discuss many of the pertinent strengths and limitations in the conclusions, but several points deserve special consideration. Secondary outcomes examining factors associated with survival were decided a priori. This distinction is often not reported, but it is important because it decreases the probability that the association is spurious or overestimated.[10] Missing data and loss to follow-up are common in observational research, but this is not a problem in this study, so the risk of bias from informative censoring is low.[10] These methodological strengths lend credence to the conclusions.

However, one factor with the main Cox regression analysis is that, unlike another cystic fibrosis survival analysis,[11] the researchers did not adjust for year of entry into the cohort. Survival times would vary according to the year of entry because of differences in therapeutic options and general cystic fibrosis care. Therefore, the year of entry should be included in any survival analysis of a chronic disease that spans decades. For instance, when the authors divide the cohort into two time periods (1990-1995 *v* 1996-2003) and add this variable to the model in a sensitivity analysis, the association between rhDNase and survival is significantly attenuated. This suggests that at least part of the beneficial effect of rhDNase

might be the result of other unmeasured aspects of cystic fibrosis care that changed or became available during the course of the study.

A four year improvement in survival among patients with cystic fibrosis and advanced lung disease is an important finding for clinicians, patients, and care givers. The rhDNase results should be compared with those from other multicentre patient registries, and if they are replicated this will strengthen the argument for causality. In the meantime, the current study adds to the evidence supporting the use of rhDNase in patients with cystic fibrosis and advanced disease and is strongly recommended in treatment guidelines for cystic fibrosis.[12] Finally, the encouraging survival rates can be used as a source of motivation for patients to increase their adherence with the entire chronic treatment regimen as they await the next advance in cystic fibrosis care.

Although survival in patients with cystic fibrosis has improved greatly, we need to do better. At best, rhDNase slows the progression of lung disease. For the next dramatic leap in survival, interventions must stop the decline in lung function associated with the disease. Small molecule treatments or gene therapy (or both) that target the underlying defect show promise. The ultimate goal is for cystic fibrosis to be transformed into a lifelong condition that patients must live with but do not die from.

1 Andersen DH. Cystic fibrosis of the pancreas and its relation to celiac disease. *Am J Dis Child* 1938;56:344-99.
2 Di Sant'Agnese PA, Andersen DH. Celiac syndrome: IV. Chemotherapy in infections of the respiratory tract associated with cystic fibrosis of the pancreas; observations with penicillin and drugs of the sulphonamide group, with special reference to penicillin aerosol. *Am J Dis Child* 1946;72:17-61.
3 Shwachman H, Kulczycki LL. Long-term study of one hundred five patients with cystic fibrosis; studies made over a five- to fourteen-year period. *Am J Dis Child* 1958;96:6-15.
4 Davies JC, Alton EW, Bush A. Cystic fibrosis. *BMJ* 2007;335:1255-9.
5 Dodge JA, Lewis PA, Stanton M, Wilsher J. Cystic fibrosis mortality and survival in the UK: 1947-2003. *Eur Respir J* 2007;29:522-6.
6 George PM, Banya W, Pareek N, Bilton D, Cullinan P, Hodson ME, et al. Improved survival at low lung function in cystic fibrosis: cohort study from 1990 to 2007. *BMJ* 2011;342:d1008.
7 Fuchs HJ, Borowitz DS, Christiansen DH, Morris EM, Nash ML, Ramsey BW, et al. Effect of aerosolized recombinant human DNase on exacerbations of respiratory symptoms and on pulmonary function in patients with cystic fibrosis. The Pulmozyme Study Group. *N Engl J Med* 1994;331:637-42.
8 Jones AP, Wallis C. Dornase alfa for cystic fibrosis. *Cochrane Database Syst Rev* 2010;3:CD001127.
9 Liou TG, Adler FR, Fitzsimmons SC, Cahill BC, Hibbs JR, Marshall BC. Predictive 5-year survivorship model of cystic fibrosis. *Am J Epidemiol* 2001;153:345-52.
10 Vandenbroucke JP, von Elm E, Altman DG, Gotzsche PC, Mulrow CD, Pocock SJ, et al. Strengthening the Reporting of Observational Studies in Epidemiology (STROBE): explanation and elaboration. *PLoS Med* 2007;4:e297.
11 Dasenbrook EC, Checkley W, Merlo CA, Konstan MW, Lechtzin N, Boyle MP. Association between respiratory tract methicillin-resistant Staphylococcus aureus and survival in cystic fibrosis. *JAMA* 2010;303:2386-92.
12 Flume PA, O'Sullivan BP, Robinson KA, Goss CH, Mogayzel PJ Jr, Willey-Courand DB, et al. Cystic fibrosis pulmonary guidelines: chronic medications for maintenance of lung health. *Am J Respir Crit Care Med* 2007;176:957-69.

New drugs for hyponatraemia

Evidence is lacking that they are better than cheaper standard treatment

PRACTICE, p 594

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Hyponatraemia is the most common electrolyte abnormality, especially in patients in hospital,[1] and it is associated with substantial morbidity and mortality.[2] The causes of hyponatraemia are often difficult to ascertain and may be multifactorial. Treatment varies according to the cause, severity of symptoms, and speed of onset. Tolvaptan, a vasopressin 2 receptor antagonist, has recently been licensed in the United Kingdom for the treatment of hyponatraemia secondary to the syndrome of inappropriate antidiuretic hormone secretion.

Arginine vasopressin, also known as antidiuretic hormone, regulates the renal clearance of free water. Raised concentrations of arginine vasopressin cause the kidneys to retain water, and disorders of its release and regulation cause an imbalance in total body water. Vasopressin 2 receptors are found mainly in the collecting ducts of the kidneys, where they control free water reabsorption. These receptors have been targeted by non-peptide vasopressin 2 receptor antagonists, which promote the excretion of water without loss of electrolytes. These agents were originally developed for the treatment of hyponatraemia as a result of heart failure, decompensated cirrhosis, and the syndrome of inappropriate antidiuretic hormone secretion.

Tolvaptan was licensed after completion of SALT-1 and SALT-2 (Study of Ascending Levels of Tolvaptan in Hyponatremia 1 and 2).[3] In the United States, the Food and Drug Administration approved both oral tolvaptan and intravenous conivaptan for the management of hyponatraemia. SALT-1 randomised 205 patients to oral tolvaptan or placebo for 30 days, with a seven day follow-up. The primary end points were the change in the average daily area under the curve (AUC) for serum sodium concentration from baseline to day 4, and from baseline to day 30. Tolvaptan significantly increased serum sodium concentrations compared with placebo (an increase of 5.4 mmol/l *v* 1.0 mmol/l from a baseline of 128 mmol/l by day 4; 7.2 mmol/l *v* 2.3 mmol/l on day 30 after start of treatment). However, the groups did not differ significantly in self assessed health status—one of the secondary outcomes—except for one section of the combined analysis on mental health (mental component summary scores improved in the tolvaptan group; P=0.02). Adverse events—thirst, dry mouth, and weakness—were more common in the tolvaptan group. Patients with severe hyponatraemia (<120 mmol/l), who may have benefited most from the drug, were excluded. In addition, hyponatraemia had several causes, including chronic congestive cardiac failure, cirrhosis, and the syndrome of inappropriate antidiuretic hormone secretion; no subgroup analyses were carried out, making it difficult to interpret the data for the syndrome alone.

Could vasopressin 2 receptor antagonists be useful in patients with heart failure? Hyponatraemia often occurs in patients with severe heart failure, usually secondary to drugs such as diuretics and inhibitors of the renin-angiotensin system. In addition, several pathophysiological mechanisms

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may contribute to hyponatraemia. Total body water may exceed that of total body sodium, resulting in hyponatraemia. Alterations to the sympathetic nervous system and the renin-angiotensin-aldosterone system, in addition to impaired responses to various natriuretic peptides, all play a role in the development of hyponatraemia associated with heart failure. To date, there are no data on the effects of the vasopressin 2 receptor antagonists on mortality.[4] One randomised controlled trial found that tolvaptan given to people with congestive cardiac failure increased weight loss, reduced oedema, and helped correct sodium, so these drugs might be useful in severe cardiac failure.[5] However, unlike in the US, where tolvaptan is licensed for the treatment of hyponatraemia secondary to congestive cardiac failure, liver cirrhosis, and the syndrome of inappropriate antidiuretic hormone secretion, in the UK its licence does not extend to people with cardiac failure.

Rapid correction of hyponatraemia, particularly chronic hyponatraemia, may cause central pontine myelinolysis, and this may result in permanent neurological disability. Accurate clinical assessment of fluid balance is difficult but crucial in identifying extracellular fluid volume status in hyponatraemic patients.[6] Vasopressin 2 receptor antagonists may have detrimental effects in hypovolaemic hyponatraemia, because they can aggravate existing hypovolaemia as a result of increased clearance of free water.[7]

The role of vasopressin 2 receptor antagonists remains unclear. Should they be used in the acute setting in symptomatic hyponatraemia or for chronic hyponatraemia? Evidence for long term benefit is lacking, with few data on morbidity and mortality to justify the expense. For £74.68 (€84.04; $117.0) a day (per tablet), more evidence of benefit is needed. If tolvaptan simply allows these patients to drink an extra glass of water a day, the cost and the potential risk are not justified. Existing trials are relatively small and of short duration. SALT-1 and SALT-2 provided the evidence to license tolvaptan but the patients included were heterogeneous so it is difficult to draw conclusions and make comparisons with other populations.

Demeclocycline has been used for many years to treat chronic hyponatraemia. It is thought to act by inducing a form of nephrogenic diabetes insipidus, reducing urine concentration even in the context of high concentrations of arginine vasopressin.[8] However, the only data on its efficacy come from mostly small observational studies and case reports. It is cheap and relatively well tolerated, although it can cause nephrotoxicity, especially in the presence of coexisting cirrhosis; however, this is generally reversible.

Fluid restriction remains the main treatment for managing the syndrome of inappropriate antidiuretic hormone secretion,[9] with success depending on patient adherence. No trials or cost-benefit analyses have compared fluid restriction, demeclocycline, and vasopressin receptor antagonists so we have no evidence that these new drugs are any better than the much cheaper standard treatment.

1 Bagshaw SM, Townsend DR, McDermid CR. Disorders of sodium and water balance in hospitalized patients. *Can J Anaesth* 2009;56:151-67.
2 Ghali JK, Farah JO, Daifallah S, Zabalawi HA, Zmily HD. Conivaptan and its role in the treatment of hyponatremia. *Drug Design Dev Ther* 2009;3:253-68.
3 Schrier RW, Gross P, Gheorghiade M, Berl T, Verbalis JG, Czerwiec FS, et al. Tolvaptan, a selective oral vasopressin V2-receptor antagonist, for hyponatremia (SALT-1, SALT-2). *N Engl J Med* 2006;355:2099-112.
4 Schreier RW, Masoumi A, Elhassan E. Role of vasopressin and vasopressin receptor antagonists in type 1 cardiorenal syndrome. *Blood Purif* 2009;27:28-32.
5 Gheorghiade M, Niazi I, Ouyang J, Czerwiec F, Kambayashi J, Zampino M, et al. Vasopressin V2-receptor blockade with tolvaptan in patients with chronic heart failure: results from a double-blind, randomized trial. *Circulation* 2003;107:2690-6.
6 Chung HM, Kluge R, Schrier RW, Anderson RJ. Clinical assessment of extracellular fluid volume in hyponatremia. *Am J Med* 1987;83:905-8.
7 Verbalis JG. Vasopressin V2 receptor antagonists. *J Mol Endocrinol* 2002;29:1-9.
8 Dousa TP, Wilson DM. Effect of demethylchlorotetracycline on cellular action of antidiuretic hormone in vitro. *Kidney Int* 1974;5:279-84.
9 Baylis, P. The syndrome of inappropriate antidiuretic hormone secretion. *Int J Biochem Cell Biol* 2003;35:1495-9.

Using bibliometrics to define the quality of primary care research

A useful international benchmark, but should not be used to allocate resources

RESEARCH, p 588

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In the linked bibliometric analysis, Glanville and colleagues assess the value of primary care research from the United Kingdom and five comparator countries, as measured by references or citations in later research publications. The authors found that these measures consistently placed UK researchers among the best in the world.[1]

Although opinions differ as to how reliable bibliometrics are as a sole measure of the quality and impact of research,[2-4] they are increasingly used to assess peer reviewed research outputs in contexts such as local research performance indicators, grant applications, and academic appraisal processes. The Higher Education Funding Council for England's current recommendation is that, because bibliometrics are unlikely to be free from distortion,[5] the forthcoming Research Excellence Framework subpanels will be asked to decide whether they wish to use citation information to inform their review of research outputs. Some may well do so.

The decisions made by the framework will inform the distribution of public funds by the four UK higher education funding bodies and therefore influence the development of the evidence base, in one direction or another, that supports clinical decisions in the more than 300 million general practice consultations each year in the UK, which represent more than 95% of all NHS consultations.[6]

The role of research underpinning primary care is pivotal to a cost effective health service.[7] However, the ratio of academic to NHS consultants is only 1:225 for general practice (compared with 1:8 for medicine or public health, 1:15 for child health, and 1:18 for psychiatry), with many primary care or general practice departments disappearing into larger research groupings.[6] A key metric in the previous Research Assessment Exercise in 2008 was the proportion of outputs rated as three star (internationally excellent) and four star (world leading).

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Glanville and colleagues sought to benchmark UK primary care research against outputs from other resource rich countries as a way to inform thinking about where the Research Excellence Framework sets the bar for internationally competitive and excellent primary care research. On the basis of the six countries compared and the range of bibliometrics examined, UK primary care researchers who produce the most research are internationally competitive and UK primary care research provides value for money in terms of research funding. These findings are consistent with the discipline's transformation over the past two decades from producing often single investigator, single centre observational work to leading multicentre, multidisciplinary, and often international experimental studies.[6 8] The findings should provide a confidence boost for UK primary care researchers in the build-up to the Research Excellence Framework.

Although the global body of knowledge in this area is rapidly increasing—at least 180 journal articles were published in 2010 with "citation analysis" or "bibliometric analysis" in the title—the study is unique in benchmarking UK primary care research against that from other countries on the basis of citation analysis.

Some of the study's findings are borne out and updated by a simple analysis of publications in journals indexed in the "medicine—general and internal" category of the Web of Science databases, which include, but are not limited to, journals publishing primary care research. This confirms that the UK is indeed punching well above its weight in overall publication numbers, although average citations per paper are more modest (figs 1 and 2). This may be explained by Glanville and colleagues' observation that UK studies are less likely to cite research findings than are studies from the Netherlands and Canada, thereby leading to lower average citation counts.

How might primary care researchers respond to any increase in bibliometric based assessment of quality? Several respondents to the pre-Research Excellence Framework consultation observed that unintended behavioural effects of bibliometric evaluations are difficult to predict.[5] Citation clubs were often mentioned, as was the prospect of researchers pursuing research perceived as "Research Excellence Framework safe," which, in primary care terms, seems to be well cited large scale quantitative research published in high impact journals.

The citation advantage from open access publication is likely to result in more researchers publishing in open access journals (as opposed to non-indexed but possibly widely read journals) and via institutional repositories.[9] Researchers may also preferentially aim to publish in non-UK based journals and those journals indexed by the database(s) used to determine outputs for the purposes of the framework, with effects that are more difficult to predict. Paradoxically, researchers may also be under pressure to publish less to increase citation to publication ratios or change their research focus from areas that are less well cited.

If bibliometrics can be used to benchmark international quality at the country level, given their ease of measurement, and apparent objectivity, should citation metrics also form the basis of decisions on the allocation of funding to research groups within countries? Glanville and colleagues rightly do not propose this. Advances in primary care, in common with other disciplines, will depend also on much creative conceptual thinking and small scale exploratory work that might never be well cited but should nevertheless be nurtured. The framework's inclusion of case studies describing work undertaken over a much longer period will allow primary care research to show how small scale exploratory and development work has often led to large scale pragmatic evaluations that have answered crucial questions and transformed care.

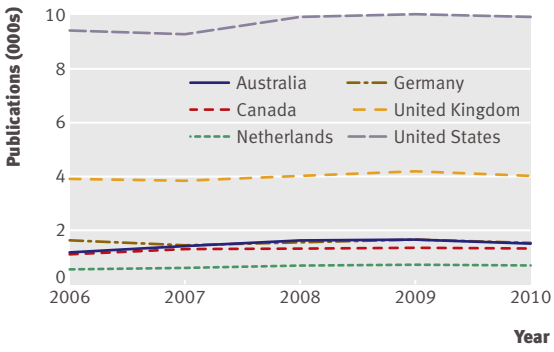

Fig 1 | Comparison across six countries of publications indexed by journals in the Web of Science "medicine—general and internal" category

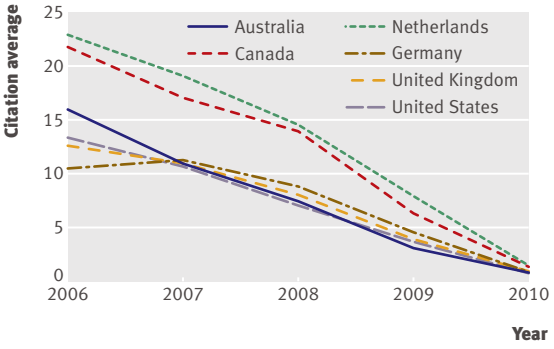

Fig 2 | Comparison across six countries of average number of citations per publication indexed by journals in the Web of Science "medicine—general and internal" category. Citations were measured up to the search date (24 January 2011) so older publications will have higher counts on average

1 Glanville J, Kendrick T, McNally R, Campbell J, Hobbs FDR. Research output on primary care in Australia, Canada, Germany, the Netherlands, the United Kingdom, and the United States: bibliometric analysis. *BMJ* 2011;342:d1028.
2 Abramo G, D'Angelo CA, Caprasecca A. Allocative efficiency in public research funding. Can bibliometrics help? *Res Policy* 2009;38:206-15.
3 Asknes DW, Rip A. Researcher's perceptions of citations. *Res Policy* 2009;38:895-905.
4 Frey BS, Rost K. Do rankings reflect research quality? *J Appl Econom* 2010;13:1-38.
5 Higher Education Funding Council for England. Responses to HEFCE 2007/34, the Research Excellence Framework consultation. 2008. www.hefce.ac.uk/pubs/consult/outcomes/ref.asp.
6 Academy of Medical Sciences. Research in general practice: bringing innovation into patient care. Workshop report. 2009. www.acmedsci.ac.uk/p101puid163.html.
7 Starfield B, Shi H, Mackinko J. Contribution of primary care to health systems and health. *Millbank Q* 2005;83:457-502.
8 Mant D, Del Mar C, Glasziou P, Knottnerus A, Wallace P, van Weel C. The state of primary care research. *Lancet* 2004;364:1004-6.
9 Swan A. The open access citation advantage. Studies and results to date. Technical report. University of Southampton, 2010. http://eprints.ecs.soton.ac.uk/18516/2/Citation_advantage_paper.pdf.

A new mental health strategy for England

Implementation is the next step

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Until 1999 mental health services in England enjoyed wide ranging freedoms to decide how to provide services, a form of "localism" that is now once again central to governmental thinking on public policy. The consequence was no overall pattern of service, variations in the standard of care, and dissatisfaction by service users and carers. This situation was transformed by the 1999 National Service Framework for Mental Health, which—typical of governmental policy at that time—set centrally agreed standards and required a particular model of care (including home treatment, assertive outreach, and early intervention teams) to be implemented consistently across England. Unusually for a national strategy, this framework was substantially put into practice,[1] largely through strong and financially incentivised performance management methods.

The coalition government has now published its new long term mental health strategy for England.[2] Its six main "shared objectives" are: more people will have good mental health; more people with mental health problems will recover; more people with mental health problems will have good physical health; more people will have a positive experience of care and support; fewer people will suffer avoidable harm; and fewer people will experience stigma and discrimination. Few people could quibble with these headline aspirations. The strategy "sets out our ambition to mainstream mental health, and establish parity of esteem between services for people with mental and physical health problems."[2]

The strategy includes several elements that will be welcomed by many people with mental illness and by the wider mental health sector. Firstly, the commitment to invest £400m (€470m; $650m) over the next four years to strengthen psychological treatments throughout the NHS in England is long overdue. The Improving Access to Psychological Treatments (IAPT) programme is extended to cover children and young people, older people and their carers, people with long term physical health problems, and those with severe mental illness.[3]

Secondly, the strategy explicitly states that the government will "commit to supporting and working actively with Time to Change and other partners on reducing stigma for people of all ages and backgrounds." This is timely because it is now well established that many people with mental illness experience profound forms of social exclusion and injustice.[4] It is also good to see a clear statement of support for the United Nations Convention on the Rights of Persons with Disabilities (which the United Kingdom has now ratified), given that violations of human rights can occur in psychiatric institutions.[5]

Thirdly, drawing on a review of the literature,[6] the focus on early stage interventions, not only for people with first episode psychotic disorders[7] but "across all ages" is a nudge to mental health practitioners to focus more on the earliest stages of prodromal or syndromic conditions, with the intention of improving the long term course and outcome.

Yet the strategy also has several shortcomings. It does not seriously consider the degree of neglect facing most people with mental illness. Although mental illnesses are surprisingly common, affecting about 20% of the population this year, only a quarter of mentally ill people across Europe receive any form of healthcare, compared with about 80% of people with diabetes.[8] Although the IAPT programme may have modestly increased the proportion treated, at the population level we still disregard the treatment needs of most people with mental illness, despite the recent well argued call to action by the World Health Organization.[9] More specifically, recent evidence indicates that people with psychotic, affective, personality, drug related, and alcohol related disorders die on average about 20 years earlier than their mentally well counterparts.[10] Although the strategy does refer to this problem of "diagnostic overshadowing" (the systematically worse physical healthcare given to people known also to have a mental illness) it provides no discernible practical plan to tackle it.

A further indication of the longstanding problems with mental health services is that people in black and ethnic minority groups often have a worse experience of care.[11] Although the strategy acknowledges this fact, again there is a lack of specificity about precisely what needs to be done and the evidence base, at a time when use of the Mental Health Act is increasing across England.

Strategies likely to succeed set out what actions will be taken, by whom, when, with which resources, and with which lines of reporting and accountability.[12] The early indications here are not auspicious, and the reader is given fair warning in the initial "reader box" document description, which states that the "action required" and "timing" are "N/A," and it is not clear whether this means not available or not applicable. Indeed, the only financial undertaking given in the whole strategy is that for the IAPT programme. Overall, this strategy details what is to be achieved, but not how. In my view, what is now needed at the national level is an implementation plan that sets out the details of exactly how these aims will be put into routine practice nationwide.

1 Glover G, Arts G, Babu KS. Crisis resolution/home treatment teams and psychiatric admission rates in England. *Br J Psychiatry* 2006;189:441-5.
2 Department of Health. No health without mental health. A cross-government mental health outcomes strategy for people of all ages. 2011. www.dh.gov.uk/en/Healthcare/Mentalhealth/MentalHealthStrategy/index.htm.
3 Clark DM, Layard R, Smithies R, Richards DA, Suckling R, Wright B. Improving access to psychological therapy: Initial evaluation of two UK demonstration sites. *Behav Res Ther* 2009;47:910-20.
4 Thornicroft G. Shunned: discrimination against people with mental illness. Oxford University Press, 2006.
5 ITHACA Study Group. The ITHACA toolkit for monitoring human rights and general health care in mental health and social care institutions. 2011. King's College London: www.ithacastudy.eu.
6 Knapp M, McDaid D, Parsonage M. Mental health promotion and prevention: the economic case. Department of Health. 2011. www2.lse.ac.uk/LSEHealthAndSocialCare/PSSRU/pdf/MHPP%20The%20Economic%20Case.pdf.
7 McCrone P, Craig TK, Power P, Garety PA. Cost-effectiveness of an early intervention service for people with psychosis. *Br J Psychiatry* 2010;196:377-82.
8 Ormel J, Petukhova M, Chatterji S, Guilar-Gaxiola S, Alonso J, Angermeyer MC, et al. Disability and treatment of specific mental and physical disorders across the world. *Br J Psychiatry* 2008;192:368-75.
9 WHO. Mental Health Gap Action Programme: scaling up care for mental, neurological and substance use disorders. 2008. www.who.int/mental_health/mhgap_final_english.pdf.
10 Tiihonen J, Lonnqvist J, Wahlbeck K, Klaukka T, Niskanen L, Tanskanen A, et al. 11-year follow-up of mortality in patients with schizophrenia: a population-based cohort study (FIN11 study). *Lancet* 2009;374:620-7.
11 Morgan C, Burns T, Fitzpatrick R, Pinfold V, Priebe S. Social exclusion and mental health: conceptual and methodological review. *Br J Psychiatry* 2007;191:477-83.
12 Thornicroft G, Tansella M. Better mental health care. Cambridge University Press, 2009.