Selective serotonin reuptake inhibitors and congenital malformations

The small risk of harm must be balanced against risk of suboptimal or no treatment

DONNA DAY/GETTY IMAGES

RESEARCH, p 735

Christina Chambers
associate professor, Division of Dysmorphology and Teratology, Departments of Pediatrics and Family and Preventive Medicine, School of Medicine, University of California San Diego, 9500 Gilman Drive, MC 0828, La Jolla, CA 92093-0828, USA
chchambers@ucsd.edu

Competing interests: CC has received grant funding from pharmaceutical companies including Amgen, Abbott, Bristol Myers Squibb, Sanofi-Pasteur, Teva, Sandoz, Kali, Barr, and Apotex, some of which manufacture or distribute selective serotonin reuptake inhibitors.

Provenance and peer review: Commissioned; not externally peer reviewed.

Cite this as: *BMJ* 2009;339:b3525
doi: 10.1136/bmj.b3525

Major depressive disorder in women is most common during their childbearing years, and about 13% of women in the United States have taken an antidepressant drug during pregnancy.[1 2] In the past 20 years, selective serotonin reuptake inhibitors (SSRIs) have become a mainstay of treatment in women with major depressive disorder; however, concerns persist about safety for the developing fetus. This is counterbalanced by equally compelling concerns about the consequences of undertreatment for mother and child.[3]

In the linked population based cohort study from Denmark, Pedersen and colleagues confirm a previously reported doubling of risk for septal heart defects after early exposure in pregnancy to SSRIs (odds ratio 1.99, 95% confidence interval 1.12 to 3.53).[4] However, in contrast to previous studies, redemptions of prescriptions for citalopram and sertraline, but not paroxetine or fluoxetine, were significantly associated with this group of heart defects.[5-8] Furthermore, unlike two previous large case-control studies conducted in the US, no association was noted with anencephaly, omphalocele, craniosynostosis, or right ventricular outflow tract defects.[7 8]

Lack of consistency across these studies with respect to specific malformations and specific drugs makes it difficult to translate the findings into clinical practice. One of the fundamental principles of teratology is that teratogenic exposures induce specific patterns of malformation, and not an increase in the incidence of every defect. In other words, if some or all SSRIs are teratogenic, we would expect to see similar findings for specific drug exposures and specific defects in all studies.

One explanation for this inconsistency, assuming that SSRIs do cause specific birth defects, is differences in study designs. For example, although Pedersen and colleagues linked records for 496 881 singleton live born infants, they identified only 1370 mothers who redeemed multiple prescriptions for an SSRI in the perinatal period. Therefore, the study may have been insufficiently powered to detect the previously suggested twofold to threefold increased risk for anencephaly, omphalocele, craniosynostosis, or right ventricular outflow tract defects, all of which occur at least an order of magnitude less frequently than septal defects.

Alternatively, these findings could be spurious and attributable in observational studies to unmeasured or inadequately controlled confounding factors, such as maternal obesity, alcohol, tobacco, or periconceptional use of folic acid supplements; confounding by the mother's underlying condition; or detection bias, in which mothers being treated for major depressive disorders are more likely to seek out or receive more comprehensive prenatal and postnatal testing of their children.

How does Pedersen and colleagues' study contribute to clinicians' and patients' decisions about the use of SSRIs in pregnancy, and how should this be weighed against the risks of non-treatment? The answer remains as before—if an increased risk for major congenital malformations does exist, this study and others suggest that the absolute risk for the individual pregnant woman is very low. Furthermore, each of the more commonly used drugs in this class has been implicated in at least one study, so it is difficult to conclude that one SSRI is "safer" than another.

We need information from larger studies of specific SSRIs, with study designs that control for maternal disease type and severity, comorbidities, and other exposures. In addition, studies of basic science might elucidate the mechanisms involved in inducing specific birth defects to support the biological plausibility of a causal association.

In August 2009, the American College of Obstetrics and Gynecology released a joint statement with the American Psychiatric Association on treatment recommendations for depression during pregnancy.[9] Briefly, the recommendations state that women with major depressive disorder who are contemplating pregnancy or who are currently pregnant can start or continue taking their drugs. Women who prefer to avoid or discontinue drugs may benefit from psychotherapy, although this will depend on their psychiatric history. Women should be informed about the possible risks and benefits of their treatment choices, and ongoing consultation between the patient's obstetrician and psychiatrist is needed during pregnancy, to determine and carry out the most appropriate and acceptable treatment plan.

Most drugs taken by pregnant women have not been well studied, or studied at all with respect to safety of the fetus.[10] Although research about SSRIs and pregnancy outcomes is plentiful, it does not necessarily provide definitive answers for clinical practice. Clinicians and patients need to balance the small risks associated with SSRIs against those associated with undertreatment or no treatment.

1 Kessler RC, Berglund P, Demler O, Jin R, Koretz D, Merikangas KR, et al. The epidemiology of major depressive disorder: results from the National Comorbidity Survey Replication (NCS-R). *JAMA* 2003;289:3095-105.
2 Cooper WO, Pont ME, Ray WA. Increasing use of antidepressants in pregnancy. *Am J Obstet Gynecol* 2007;196:544e1.
3 Deave T, Heron J, Evans J, Emond A. The impact of maternal depression in pregnancy on early childhood development. *BJOG* 2008;115:1043-51.
4 Selective serotonin reuptake inhibitors in pregnancy and congenital malformations: population based cohort study. *BMJ* 2009;339:b3569.
5 Kallen B, Olausson PO. Maternal use of selective serotonin re-uptake inhibitors in early pregnancy and infant congenital malformations. *Birth Defects Res Part A Clin Mol Teratol* 2007;79:301-8.
6 Diav-Citrin O, Shechtman S, Weinbaum D, Wajnberg R, Avgil M, Di Gianantonio E, et al. Paroxetine and fluoxetine in pregnancy: a prospective, multicentre, controlled, observational study. *Br J Clin Pharmacol* 2008;66:695-705.
7 Louik C, Lin AE, Werler MM, Hernandez-Diaz S, Mitchell AA. First-trimester use of selective serotonin-reuptake inhibitors and the risk of birth defects. *N Engl J Med* 2007;356:2675-83.
8 Alwan S, Reefhuis J, Rasmussen SA, Olney RS, Friedman JM, National Birth Defects Prevention Study. Use of selective serotonin-reuptake inhibitors in pregnancy and the risk of birth defects. *N Engl J Med* 2007;356:2684-92.
9 Yonkers KA, Wisner KL, Stewart DE, Oberlander TF, Dell DL, Stotland N, et al. The management of depression during pregnancy: a report from the American Psychiatric Association and the American College of Obstetricians and Gynecologists. *Gen Hosp Psychiatry* 2009;31:403-13.
10 Lo WY, Friedman JM. Teratogenicity of recently introduced medications in human pregnancy. *Obstet Gynecol* 2002;100:465-73.

Thigh circumference and risk of heart disease and premature death

The strength of the association needs further research

ALPHOTO/SPL

RESEARCH, p 736

Ian A Scott director of internal medicine and clinical epidemiology, Princess Alexandra Hospital, Ipswich Road, Brisbane, QLD 4102, Australia
ian_scott@health.qld.gov.au

Competing interests: None declared.
Provenance and peer review: Commissioned; not externally peer reviewed.

Cite this as: *BMJ* 2009;339:b3302
doi: 10.1136/bmj.b3302

Several anthropomorphic indices have been devised to help clinicians predict cardiovascular risk, including body mass index, waist circumference, hip circumference, and waist-hip ratio. Because none has clearly been shown to be superior,[1] investigators continue to look for better measures, and in the linked study Heitmann and Frederiksen propose a new one—thigh circumference.[2]

In a cohort of 1436 men and 1380 women aged 35-65 years participating in the Danish MONICA (monitoring trends in and determinants of cardiovascular disease) project, the authors examined the association between thigh circumference and the incidence of cardiovascular disease and coronary artery disease at 10 years and total mortality at 12.5 years. They fitted four separate proportional hazard regression models to the data for either sex to examine the association between thigh circumference, measured in centimetres directly below the gluteal fold of the right thigh, and hazard ratio of disease and death. The model adjusted for smoking, education, physical activity, menopause (in women), body fat percentage, height, body mass index, waist circumference, alcohol intake, systolic blood pressure, and concentrations of total cholesterol and triglycerides. They used centiles of thigh circumference and made the 50th centile (equal to 55 cm) the base reference (hazard ratio of 1). In most of the models they found an almost linear increase in risk as thigh circumference dropped below 55 cm, which was independent of age or sex; above that circumference, risk decreased but showed no consistent relation. They concluded that below a threshold of about 60 cm the risk of developing heart disease, or dying prematurely, was greatly increased.

The results raise several questions. Is this association real and independent, or a spurious or chance finding? The statistical modelling used in this study was rigorous—it removed the effects of known conventional risk factors and minimised (but did not, and cannot, totally eliminate) residual confounding. Unfortunately, with regard to coronary heart disease for both sexes and cardiovascular disease for women, the hazard ratios became non-significant as more variables were added to the models, which may not have happened if the sample had been larger.

Is this association biologically plausible? It would seem logical that having bigger thighs would be a reflection of greater adiposity, and that this would increase the risk of heart disease. However, the authors cite studies suggesting that too little muscle or subcutaneous fat (or both) in the lower limbs may predispose to adverse glucose and lipid metabolism. Has the association been replicated in other studies? They cite a single study of patients with chronic obstructive pulmonary disease in whom the mid-thigh muscle cross-sectional area was a better predictor of mortality than body mass index. Interestingly, other studies have shown that larger hip circumference (which might be a proxy for thigh circumference) significantly reduces the risk of incident diabetes and coronary heart disease.[3]

Will this association help clinicians predict risk in individual patients more accurately than they already do using readily accessible and validated risk calculators? The answer is—we do not know. To improve individual risk estimates beyond those that we can derive now, the hazard ratios would need to be much larger than those seen in this study. The highest risk equalled hazard ratios between 2.0 and 2.5, and these applied to only 2.5% of all patients—those with thigh circumferences between 46.0 cm and 46.5 cm. A risk predictor is potentially useful if people who go on to have an event have a higher predicted risk on the basis of the new predictor than those who do not have an event. The probability of seeing such an increased risk in every patient who demonstrates the risk predictor is called the c-statistic, and the higher it is (c-statistic ≥0.7), the more useful the predictor. To achieve such values, the strength of association between the risk predictor and the risk of disease needs to be high, close to an unadjusted hazard ratio of 10 or more.[4] Given that the high-

est hazard ratio associated with models that were not fully adjusted in this study was 4.65, it seems unlikely that thigh circumference will be clinically useful.

If a risk prediction model that incorporates thigh circumference in addition to other known risk factors is to be incorporated into usual practice, we need to ensure several things—firstly, that the new model discriminates better (has a higher c-statistic) than existing models; secondly, that it is well calibrated—that the predicted and observed risk estimates for each stratum of risk are similar; and thirdly, that using the new model will lead to an appropriate change in intended management in more patients now correctly reclassified as having higher or lower risk than would be the case using existing risk prediction models.

More research is needed to see whether measuring the thigh circumference with a tape measure adds anything more to our clinical management than eliciting risk factors from the history, examining the cardiovascular system, and measuring serum lipids. Randomised trials are needed to test whether interventions that increase thigh muscle mass through increased physical activity—in addition to or separate from current primary prevention strategies—decrease cardiovascular risk more than current practice. If this approach is shown to be effective, the public health implications would be intriguing.

1 Canoy D. Distribution of body fat and risk of coronary heart disease in men and women. *Curr Opin Cardiol* 2008;23:591-8.
2 Heitmann BL, Frederiksen P. Thigh circumference and risk of heart disease and premature death: cohort study. *BMJ* 2009;339:b3292.
3 Parker ED, Pereira MA, Stevens J, Folsom AR. Association of hip circumference with incident diabetes and coronary heart disease: the Atherosclerosis Risk in Communities study. *Am J Epidemiol* 2009;169:837-47.
4 Pepe MS, Janes H, Longton G, Leisenring W, Newcomb P. Limitations of the odds ratio in gauging the performance of a diagnostic, prognostic, or screening marker. *Am J Epidemiol* 2004;159:882-90.

Can financial incentives improve health equity?

Evidence shows that they might, if targeted appropriately

ANALYSIS, p 725

Adam Oliver RCUK senior academic fellow, LSE Health, London School of Economics, London WC2A 2AE
a.j.oliver@lse.ac.uk

Competing interests: AO is a codirector of CSI Health (Centre for the Study of Incentives in Health), but he is not ideologically for or against the use of incentives. CSI Health is funded by a strategic award in biomedical ethics from the Wellcome Trust (grant no 086031/Z/08/Z).
Provenance and peer review: Commissioned; not externally peer reviewed.

Cite this as: *BMJ* 2009;339:b3847
doi: 10.1136/bmj.b3847

Recently, much interest has been shown in how financial incentives can increase health enhancing behaviours.[1-3] Two centres are studying the subject—the Centre for the Study of Incentives in Health (a joint initiative between King's College, Queen Mary, and the London School of Economics; www.kcl.ac.uk/schools/biohealth/research/csincentiveshealth/) and the Center for Health Incentives at the University of Pennsylvania (www.med.upenn.edu/ldichi/). By encouraging healthier behaviours, it is hoped that incentives will help to contain healthcare costs and improve health. If the incentives motivate people in higher socioeconomic groups more than those in lower socioeconomic groups, however, they could exacerbate health inequalities. In the linked analysis article, Schmidt and colleagues highlight this as a potential problem in Germany, where a sickness fund rewards people for engaging in preventive activities and for minimising use of health care, which might encourage the less well off to forgo needed health care.[1]

These are legitimate concerns, but we should not conclude that all incentives harm health equity. Studies across a range of interventions have shown that people within lower socioeconomic groups do sometimes respond significantly to incentives. Most of these studies were conducted in the United States, but their findings should be applicable to other countries.

For example, vouchers redeemable for fruit juice significantly increased concentrations of β carotene in pregnant women on low incomes.[4] This finding concurs with the Organisation for Economic Co-operation and Development's recent recommendation that cash payments or food vouchers should be offered to materially deprived pregnant women to boost the take-up of antenatal services.[5] Early visits to childhood health centres and uptake of vaccinations have been increased by financial incentives in Mexico, Nicaragua, Colombia, and Jamaica.[2] A $10 (£6; €6.8) incentive significantly increased the uptake of mammograms in women on low incomes aged 40-64 years.[6] Financial incentives have also improved participation of intravenous drug users in a hepatitis B vaccination programme and a tuberculosis treatment programme.[7 8] Several other examples of the positive effects of financial incentives have been published.[9 10]

These studies show that in some areas of health care modest financial incentives can substantially affect the behaviours of the relatively poor. Healthcare incentives do not always have a positive effect, however, and evidence of a positive sustained effect on more complex lifestyle behaviours, such as smoking or weight loss, is lacking.[3]

Some of the studies may have volunteer bias—volunteers may be particularly motivated to change their behaviour—and few studies provide adequate information on costs, let alone value for money. Moreover, the studies do not test the differential effect of incentives on the relatively poor versus the better off. Because less wealthy people do respond to incentives, health inequalities could be reduced if incentives were targeted at them.

Targeting certain groups is controversial because it can breed resentment in the untargeted population. This can undermine solidarity, a key feature of European healthcare systems. Also, should the target be set at the family level (for example, families whose income is below a certain amount) or the geographical level (poor communities)? Because pockets of wealth often exist in poor communities, targeting at the family level seems the most sensible choice. Targeted interventions may be the best option in the current global financial climate because they are less expensive than those aimed at the population.

A child receiving an oral vaccine in Mexico

KEITH DANNEMILLER/ALAMY

Evidence indicates that appropriately targeted incentives could reduce inequalities in health outcomes. Ongoing assessment of their affordability, effectiveness, cost effectiveness, and unintended consequences is needed. Irrespective of the effectiveness of incentives, some people will argue that they do not tackle the root cause of poverty, and that money and health behaviours are incommensurate goods.[11] Like all tools, financial incentives may have unfortunate consequences unless handled with care, but it seems premature and irresponsible to exclude them completely from the policymaking kitbag.

1 Schmidt H, Gerber A, Stock S. What can we learn from German health incentive schemes? *BMJ* 2009;339:b3504.
2 Forde I, Zeuner D. Financial incentives to promote social mobility. *BMJ* 2009;339:b3219.
3 Jochelson K. *Paying the patient: improving health using financial incentives*. King's Fund, 2007. www.wpro.who.int/NR/rdonlyres/BCC2F5C3-B685-4FF1-AE77-39C52F4ED247/0/payingthepatient.pdf.
4 Burr ML, Trembeth J, Jones KB, Geen J, Lynch LA, Roberts ZE. The effects of dietary advice and vouchers on the intake of fruit and fruit juice by pregnant women in a deprived area: a controlled trial. *Public Health Nutr* 2007;10:559-65.
5 Organisation for Economic Co-operation and Development. *Doing better for children*. 2009. www.oecd.org/document/12/0,3343,en_2649_34819_43545036_1_1_1_37419,00.html.
6 Slater JS, Henly GA, Ha CN, Malone ME, Nyman JA, Diaz S, et al. Effect of direct mail as a population-based strategy to increase mammography use among low-income underinsured women ages 40 to 64 years. *Cancer Epidemiol Biomarkers Prev* 2005;14:2346-52.
7 Seal KH, Kral AH, Lorvick J, McNees A, Gee L, Edlin BR. A randomized controlled trial of monetary incentives versus outreach to enhance adherence to the hepatitis B vaccine series among injection drug users. *Drug Alcohol Depend* 2003;71:127-31.
8 Malotte CK, Hollingshead JR, Larro M. Incentives versus outreach workers for latent tuberculosis treatment in drug users. *Am J Prev Med* 2001;20:103-7.
9 Post E, Cruz M, Harman J. Incentive payments for attendance at appointments for depression among low-income African Americans. *Psychiatr Serv* 2006;57:414-6.
10 Haukoos JS, Witt MD, Coil CJ, Lewis RJ. The effect of financial incentives on adherence with outpatient human immunodeficiency virus testing referrals from the emergency department. *Acad Emerg Med* 2005;12:617-21.
11 Szmukler G. Financial incentives for patients in the treatment of psychosis. *J Med Ethics* 2009;35:224-8.

Screening for intracranial aneurysms in ADPKD

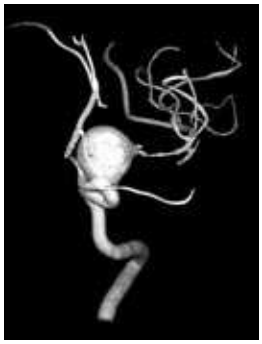
A more accurate risk assignment model is needed

Albert C M Ong professor of renal medicine, Academic Nephrology Unit, Faculty of Medicine, Dentistry and Health, University of Sheffield, Beech Hill Road, Sheffield S10 2RX
a.ong@sheffield.ac.uk

Competing interests: None declared.
Provenance and peer review: Not commissioned, externally peer reviewed.
Cite this as: *BMJ* 2009;339:b3763
doi: 10.1136/bmj.b3763

Autosomal dominant polycystic kidney disease (ADPKD) is one of the most common monogenic human diseases, with an incidence of 1 in 1000. Asymptomatic aneurysms can be detected in 6% of patients with ADPKD without a family history, but in up to 16% of patients with a family history.[1] This compares with an estimated prevalence of 1-2% in the general population. Intracranial aneurysm rupture is a rare but devastating complication of AKPKD that occurs on average 10 years younger than sporadic intracranial aneurysms. The youngest reported case was a 13 week old infant, and in one study 10% of patients were younger than 21 years.[2] Intracranial aneurysm rupture is associated with a death rate of up to 65%. Treatment of a ruptured intracranial aneurysm by either neurosurgical clipping or endovascular treatment also carries an unacceptably high mortality rate of 8-10% and morbidity (disability or dependency) rate of 16-21%.[3]

The risk of rupture of asymptomatic intracranial aneurysms occurring in the general population is primarily determined by size, location, and a history of rupture.[4] For instance, the rate of rupture for intracranial aneurysms less than 10 mm in diameter


NEIL BORDEN/SPL

is estimated at 1 in 2000 patient years (0.05% per year), but is 10 times higher when a previous rupture has been documented.[5] Nevertheless, the natural history of intracranial aneurysm in ADPKD has been uncertain, and it has been reported that aneurysm formation and rupture may cluster in families.[6] Given this uncertainty, which patients should be screened for asymptomatic intracranial aneurysms? And can we begin to assign a lifetime risk of rupture for each ADPKD patient?

Recent studies have shed light on the natural history of asymptomatic intracranial aneurysms in ADPKD. In one study, 21 patients without a history of rupture (66.7% with a positive family history) with small (3.5 mm diameter) intracranial aneurysms detected by presymptomatic screening were followed-up for a median of seven years.[7] No ruptures were detected. Only one patient (who had a negative family history) had an increase in aneurysm size and another (who had a positive family history) developed a second aneurysm. Another prospective study included 20 patients with and without a previous rupture.[8] Over 15 years, only 11% of asymptomatic patients initially detected by screening developed a new aneurysm, however, 36% presenting with a previous rupture developed new ones. In a third study, 76 patients with an initial negative scan had a repeat scan after a median follow-up of 9.7 years.[9] Of these, 42% had a positive family history of intracranial aneurysm rupture. New ruptures were detected in two patients—one with, and the other without a positive family history.

Although these studies are small with a selection bias, the overall conclusion is that the prognosis for asymptomatic intracranial aneurysms in ADPKD is excellent. The only factor that seemed to correlate with the development of new ruptures was a history of rupture. The studies also imply that aneurysms that are destined to rupture could have a different natural history to those that are not going to. This is consistent with observations that 50% of intracranial aneurysms in ADPKD that rupture are small—that is, less than 10 mm.[1]

Can genetics help us? The two genes mutated in ADPKD, PKD-1 (85%) and PKD-2 (15%), have been identified, and intracranial aneurysms occur in patients with both genes. The most extensive study to date indicates that different ADPKD mutations may confer different risk for a vascular phenotype.[10] Although no clear relationship between genotype and phenotype was found for PKD-2, PKD-1 patients with intracranial aneurysms were significantly more likely to carry germline mutations in the 5′ half of the gene compared with those without intracranial aneurysms. People with a stronger family history—that is, more than two first degree relatives who are affected—were significantly more likely to carry mutations more 5′ than those with no family history. Many patients with 3′ mutations, however, also had a rupture. This means that mutation testing is insufficiently precise to assign risk.

Where does this leave us in terms of clinical practice? Patients who have had a previous rupture seem to be at higher risk and need lifelong screening. For asymptomatic patients, the presence of a strong family history of rupture (at least two first degree relatives) remains the best predictor of rupture, and a case can be made to screen this group.[4]

Recently, an attempt has been made to quantify the probability of intracranial aneurysm rupture for individual patients using a Bayesian random effects model, which is based on the frequency of rupture in each pedigree in the context of family size.[6 11] This needs to be tested in larger populations and refined by including relevant risk factors such as previous rupture, the specific ADPKD mutation, and factors important in sporadic intracranial aneurysm rupture—for example, hypertension, smoking, and alcohol consumption.[4] Nevertheless, it is a start to assign individual risk more precisely, and hence inform the decision to screen. Overall, patients and doctors should be reassured by evidence from these studies, which does not support routine screening in all patients with ADPKD. Until a more accurate risk assignment model is available, doctors need to explain the balance of benefits and harms to patients, and discuss the best course of action.[12]

1 Pirson Y, Chauveau D, Torres V. Management of cerebral aneurysms in autosomal dominant polycystic kidney disease. *J Am Soc Nephrol* 2002;13:269-76.
2 Chauveau D, Pirson Y, Verellen-Dumoulin C, Macnicol A, Gonzalo A, Grunfeld JP. Intracranial aneurysms in autosomal dominant polycystic kidney disease. *Kidney Int* 1994;45(4):1140-6.
3 Molyneux A, Kerr R, Stratton I, Sandercock P, Clarke M, Shrimpton J, et al. International Subarachnoid Aneurysm Trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: a randomised trial. *Lancet* 2002;360:1267-74.
4 Rinkel GJ. Intracranial aneurysm screening: indications and advice for practice. *Lancet Neurol* 2005;4:122-8.
5 Investigators ISoUIA. Unruptured intracranial aneurysms--risk of rupture and risks of surgical intervention. *N Engl J Med* 1998;339:1725-33.
6 Belz MM, Hughes RL, Kaehny WD, Johnson AM, Fick-Brosnahan GM, Earnest MP, et al. Familial clustering of ruptured intracranial aneurysms in autosomal dominant polycystic kidney disease. *Am J Kidney Dis* 2001;38:770-6.
7 Gibbs GF, Huston J 3rd, Qian Q, Kubly V, Harris PC, Brown RD Jr, et al. Follow-up of intracranial aneurysms in autosomal-dominant polycystic kidney disease. *Kidney Int* 2004;65:1621-7.
8 Belz MM, Fick-Brosnahan GM, Hughes RL, Rubinstein D, Chapman AB, Johnson AM, et al. Recurrence of intracranial aneurysms in autosomal-dominant polycystic kidney disease. *Kidney Int* 2003;63:1824-30.
9 Schrier RW, Belz MM, Johnson AM, Kaehny WD, Hughes RL, Rubinstein D, et al. Repeat imaging for intracranial aneurysms in patients with autosomal dominant polycystic kidney disease with initially negative studies: a prospective ten-year follow-up. *J Am Soc Nephrol* 2004;15:1023-8.
10 Rossetti S, Chauveau D, Kubly V, Slezak JM, Saggar-Malik AK, Pei Y, et al. Association of mutation position in polycystic kidney disease 1 (PKD1) gene and development of a vascular phenotype. *Lancet* 2003;361:2196-201.
11 Ring T, Spiegelhalter D. Risk of intracranial aneurysm bleeding in autosomal-dominant polycystic kidney disease. *Kidney Int* 2007;72:1400-2.
12 Caplan LR. Should intracranial aneurysms be treated before they rupture? *N Engl J Med* 1998;339:1774-5.

The future of social care in England

Should be a national service that aims to reduce variability in access and availability

Christina R Victor professor of gerontology and health services research, School of Health and Social Care, University of Reading, Reading RG6 1HY
c.r.victor@reading.ac.uk

Competing interests: None declared.
Provenance and peer review: Commissioned; not externally peer reviewed.

Cite this as: *BMJ* 2009;339:b3384
doi: 10.1136/bmj.b3384

The green paper on the future of social care for adults was published on 14 July 2009.[1] The purpose of *Shaping the Future of Care Together* is to debate the options for the future funding of social care and support for two key groups of vulnerable adults: older people and younger people with disabilities. In both groups the numbers in need of care are substantial and predicted to increase.[1] About 2.45 million older people have some social care needs,[2] and 1.2 million receive some publicly provided social care services.[3]

At the heart of the debate is the distinction, enshrined in the initial structure of the post war welfare state, between healthcare needs and social care needs and how these should be met. Health needs are the province of the NHS, funded mainly from general taxation within the broad structure of a national system that is committed to equitable quality and access to care. Social care needs were conceptualised as separate. Local authorities administer the system and organise the nature and extent of social care in response to local needs, and they are authorised to charge whatever is deemed appropriate.[4-6]

Analysis of different forms of welfare provision internationally shows that medical care is seen to merit special consideration, with care provided free, or with users having to pay only a modest contribution.[7] Even in the United States, where health care may be construed as closest to consumer goods, some aspects of the system show the special status of medical care, such as the Medicare programme for older people. This contrasts with social care, which has no special status, and where the types of tasks involved—such as personal care, preparation of meals, or care of the house—are part of normal daily life and services that we might be expected to pay for ourselves. Indeed, social care is often represented as an area of personal responsibility, in contrast to the more collective responsibility ascribed to medical care. However, these tasks are important in enabling older people to continue to live at home independently.[8]

It has always been presumed that people should contribute towards their social care, in the United Kingdom and other countries, although the size of the contribution may vary. Thus there is an increasing distinction between the provision of health care and social care based not just on how needs are defined and met, but also on how they are paid for. An example of this tension is the funding of long term care.[9] Social care is means tested in England, and anyone with assets worth more than £23 500 (€27 300; $38 500) who needs to go into a care home receives no help from the state. Thus, older people and their families can face huge bills for care. The cost of a care home is about £24 000 a year and that of a nursing home is £35 000.[10] Older people (and their families) spend around £5.9bn on social care, a figure that

PAUL DOYLE/ALAMY

matches state funding.[10] Under the current system, some people have to pay as much as £200 000 for social care, whereas others receive it free.[1]

In response to what is termed the "postcode" social care lottery, the green paper proposes a national social care service to tackle this variability in service provision and charging. At the heart of this are the proposals for the future funding of social care and three national policy options, two of which explicitly include some state support for social care. Specifically excluded are both fully funded state care (too expensive) and making people entirely responsible for their social care arrangements (too many people would be unable to afford such care). In two of the proposals the state would contribute a quarter to a third of social care costs, with people taking out insurance at a cost of £20 000-£25 000 to cover the remaining costs, or paying themselves, with help for those on low income. The third option is compulsory insurance against the need for social care.

These are important proposals that will shape the future of the provision of social care for future generations of vulnerable adults. They mark a shift away from the inherent variability of the social care system towards a nationally based social care system, relating to England in the first instance. This is an important debate that is currently open to public consultation until 13 November (http://www.dh.gov.uk/en/Consultations/Liveconsultations/DH_102339).

1 Burnham A. *Shaping the future of care together.* Department of Health. 2009. www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_102338 .
2 Wanless D. *Securing good care for older people. Taking a long term view.* 2006. www.kingsfund.org.uk/publications/kings_fund_publications/securing_good.html.
3 NHS Information Centre. *Community care statistics.* Leeds: NHSIC, 2009.
4 Victor CR. *The social context of ageing.* London: Routledge, 2005.
5 Means R, Smith R. *From poor law to community care.* 2nd ed. Bristol: Policy Press, 1998.
6 Means R, Richards S, Smith R. *Community care: policy and practice.* 4th ed. Basingstoke: Palgrave-Macmillan, 2008.
7 Twigg J. *Bathing: the body and community care.* London: Routledge, 2000.
8 Phillips J. *Care.* Cambridge: Polity Press, 2007.
9 Sutherland S (chairman). *With respect to old age: long term care—rights and responsibilities. A report by the Royal Commission on Long Term Care.* London: Stationery Office, 1999.
10 Laing and Buisson PLC. *Care of elderly people market survey.* London: Laing and Buisson, 2008.