Slow walking speed in elderly people

Is associated with vascular mortality, but may also predict future frailty

ANNE W KRAUSE/CORBIS

RESEARCH, p 1187

Rowan H Harwood consultant geriatrician, Nottingham University Hospitals NHS Trust, Queen's Medical Centre, Nottingham NG7 2UH
rowan.harwood@nuh.nhs.uk
Simon P Conroy geriatrician, University of Leicester School of Medicine, Clinical Sciences Building, Leicester Royal Infirmary, Leicester LE2 7LX
spc3@le.ac.uk

Competing interests: None declared.

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

Cite this as: *BMJ* 2009;339:b4236
doi: 10.1136/bmj.b4236

People who walk faster are less likely to die than slow walkers, especially from vascular disease. In the linked prospective cohort study, Dumurgier and colleagues show that this is true for walking speed measured over 6 m in 3208 relatively fit older people, over five years of follow-up.[1]

Walking speed is determined by physical features such as age, sex, and height, by the presence or absence of diseases, and by physical fitness.

Evidence that exercise is beneficial for health goes back over 50 years, and includes activity during sport and work as well as fitness training. Benefits are primarily in mortality and morbidity from vascular disease, and are seen in middle aged and older people, and those with and without established cardiovascular disease, diabetes, hypertension, or obesity.[2 3]

Diseases that slow down walking include musculoskeletal, cardiac, respiratory, neurological, and psychiatric disorders. Some of these are associated with excess mortality from vascular disease by virtue of being vascular diseases themselves or via common causal factors, such as smoking. If angina or heart failure cause slow walking, the association between slow walking and vascular mortality is through reverse causation. In epidemiological studies this can be discounted to some extent by excluding people with diagnosed vascular disease at baseline from the analysis, or by excluding events that occur in the first 5-10 years of follow-up, which suggests that the condition was not present at baseline but developed subsequently.

Measures of physical performance are more strongly associated with adverse outcomes than some classic risk factors.[3 4] This means that they are more important causally or that they can be measured more precisely. Most studies measure physical activity by using questionnaires. Lack of standardisation and problems with recall or classification of activities present a major challenge for research and weaken the observed association between activity and outcomes. Physical performance is strongly and causally associated with activity and can be measured more precisely. Exercise tolerance on a treadmill, peak oxygen consumption, self reported and measured gait speed, and pulse rate have all been associated with vascular disease in this way. Walking speed may therefore simply be a good way of measuring habitual activity.

Walking speed has been suggested as a new "vital sign" or screening test for people at risk of vascular disease.[5] In practice, this approach has two problems. An epidemiologist is more interested in whether walking speed per se is related to outcomes than the effects of confounding and reverse causal factors, and so tries hard to exclude their effects. However, to clinicians, slow walking is slow walking whatever the cause. Moreover, as with classic risk factors, walking speed in isolation is of limited use in defining the likelihood of disease in individual patients. For predicting all cause mortality, the sensitivity and specificity of the lowest third of gait velocity in Dumurgier and colleagues' study are modest (0.5 and 0.67, respectively).

Preventing vascular disease is important, especially for older people who are well. But as old age progresses, there is a trade off between preventing disease and the burden of polypharmacy, and the priority shifts from preventing mortality to predicting and forestalling transitions to disability.

Walking speed has also been investigated as part of a syndrome of frailty, defined as a state of vulnerability to deterioration in the face of stressors. Frailty is common in older people (about 20% of those over 65) and is associated with falls, disability, and admission to hospital and care homes.[6 7] Previously, approaches to defining frailty have included counting diagnoses, disabilities, or vulnerability factors (such as cognitive impairment, poor vision, or a history of falls).[8] However, recently a physiological model has been proposed that comprises three or more of the following criteria: unintentional weight loss, self reported exhaustion, weakness (reduced grip strength), slow walking speed, and low physical activity (albeit with a much lower threshold for slow walking than that used by Dumurgier and colleagues—0.65 m/s compared with 1.35-1.5 m/s).[9] Frailty by this definition may represent the effects of underlying processes of ageing. It predicts adverse outcomes,[7] but being a physiological definition it also suggests interventions that could slow or reverse the process.

Researchers assessing frailty also refer to a "pre-frail" state, where not all of the features of the syndrome exist, or where the severity of features is below the threshold.[9 10] If interventions can modify frailty or attenuate its association with poor outcomes, they are likely to be valuable, not just in preventing vascular disease. Interventions may include lifestyle changes such as exercise or diet, but in the future they may include drugs that are anabolic or that modify age associated cellular and metabolic dysfunction.

Showing that these interventions improve outcomes for frail or pre-frail older people has been difficult.[10] One notable exception is falls prevention,[11] where exer-

cise based treatments reduce clinically important end points. Evidence that this extends to other outcomes (such as death and institutionalisation) is suggestive, but weak.[12]

1 Dumurgier J, Elbaz A, Ducimetière P, Tavernier B, Alpérovitch A, Tzourio C. Slow walking speed and cardiovascular death in well functioning older adults: prospective cohort study. *BMJ* 2009;339:b4460.
2 Blair SN, Morris JN. Healthy hearts and the universal benefits of being physically active: physical activity and health. *Ann Epidemiol* 2009;19:253-6.
3 Nocon M, Hiemann T, Muller-Riemenschneider F, Thalau F, Roll S, Willich SN. Association of physical activity with all-cause and cardiovascular mortality: a systematic review and meta-analysis. *Eur J Cardiovasc Prev Rehabil* 2008;15:239-46.
4 Myers J, Prakash M, Froelicher V, Dat D, Partington S, Atwood JE. Exercise capacity and mortality among men referred for exercise testing *N Engl J Med* 2002;346:793-801.
5 Hall WJ. Update in geriatrics. *Ann Intern Med* 2006;145:538-43.
6 Rothman MD, Leo-Summers L, Gill TM. Prognostic significance of potential frailty criteria. *J Am Geriatr Soc* 2008;56:2211-6.
7 Ensrud KE, Ewing SK, Taylor BC, Fink HA, Stone KL, Cauley JA, et al; for the Study of Osteoporotic Fractures Research Group. Frailty and risk of falls, fracture, and mortality in older women: the study of osteoporotic fractures. *J Gerontol Ser A Biol Sci Med Sci* 2007:62:M744-51.
8 Rockwood K, Mitnitski A. Frailty in relation to the accumulation of deficits. *J Gerontol Ser A Biol Sci Med Sci* 2007;62:722-7.
9 Fried LP, Tangen CM, Walston J, Newman AB, Hirsch C, Gottdiener J, et al. Frailty in older adults: evidence for a phenotype. *J Gerontol A Biol Sci Med Sci* 2001;56:M146-56.
10 Faber MJ, Bosscher RJ, Chin A Paw MJ, van Wieringen PC. Effects of exercise programs on falls and mobility in frail and pre-frail older adults: A multicenter randomized controlled trial. *Arch Phys Med Rehabil* 2006;87:885-96.
11 Gillespie LD, Gillespie WJ, Robertson MC, Lamb SE, Cumming RG, Rowe BH. Interventions for preventing falls in older people. *Cochrane Database Syst Rev* 2003;(4):CD00340.
12 Skelton D, Dinan S, Campbell M, Rutherford O. Tailored group exercise (falls management exercise—FaME) reduces falls in community dwelling older frequent fallers. *Age Ageing* 2005;34:636-9.

Greater equality and better health

Benefits are largest among the poor, but extend to nearly everyone

RESEARCH, p 1178

Kate E Pickett professor of epidemiology, Department of Health Sciences, University of York, Heslington, York YO10 5DD
kp6@york.ac.uk
Richard G Wilkinson professor emeritus of social epidemiology, Division of Epidemiology and Public Health, University of Nottingham Medical School, Nottingham NG7 2UH

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

Cite this as: *BMJ* 2009;339:b4320
doi: 10.1136/bmj.b4320

In 1996, when discussing studies of income inequality and health, the editor of the *BMJ* wrote, "The big idea is that what matters in determining mortality and health in a society is less the overall wealth of that society and more how evenly wealth is distributed. The more equally wealth is distributed the better the health of that society."[1]

Since then, the "big idea" has been widely tested. More than 200 peer reviewed studies of the association have been published. Income inequality has been variously associated with lower life expectancy, higher rates of infant and child mortality, shorter height, poor self reported health, low birth weight, AIDS, depression, mental illness, and obesity. In the linked meta-analysis, Kondo and colleagues further assess the association between income inequality and health.[2]

Perhaps because of the deep political implications of a causal relation between better health of the population and narrower differences between incomes, interpretations of the evidence have come to different conclusions.[3-6] The controversial question is not whether more equal societies really do have better health, but why they do and whether it is an effect of inequality itself. We know that lower incomes and poorer conditions are related to worse health, but can the association between smaller differences in incomes and better population health be explained on that basis alone, or does inequality have broader psychosocial effects on population health?

RICHARD BAKER/CORBIS

Two rival explanations for the link between inequality and health have been proposed—"compositional" and "contextual" explanations. Compositional explanations suggest that more unequal societies have worse health simply because they have more poor people. Redistribution of income from rich to poor would be expected to improve average health if the poor spent the extra money on things that benefit health, such as better food and warmer housing, whereas reducing luxury expenditure among the rich had little effect on their health. Such explanations also imply that the relation between inequality and population health has little to do with inequality itself—for example, with social comparisons or hierarchy—but is merely a result of how individual incomes affect health.

In contrast, contextual explanations are more closely related to the idea that inequality is somehow divisive and socially corrosive. What we now know about the importance to health of psychosocial factors—including social status, friendship, social capital, and sense of control—makes contextual explanations increasingly plausible.[7]

Compositional effects can be separated from contextual effects by using multilevel statistical models, in which the societal association between inequality and health is adjusted for the health effects of the

individual incomes of the population. Kondo and colleagues report a meta-analysis of multilevel studies including about 60 million people.[2] They find that even after taking account of the effects of individual socioeconomic characteristics, health is better in more equal societies.

Because we are dealing with whole populations, the attributable risk of the contextual effects is large, even though the effect size is fairly small. For every 0.05 increase in the Gini coefficient of income inequality, mortality increased 7.8%, with an estimated 1.5 million excess deaths each year in 30 countries in the Organisation for Economic Cooperation and Development. But policy makers should not be misled. The distinction between compositional and contextual effects is merely a distinction between causal pathways. If income differences were made smaller, health would improve in two ways—from increasing the relative incomes of the poor, and from the wider contextual benefits of greater equality.

Multilevel modelling is likely to produce conservative estimates of the size of the contextual effects of inequality on health. The idea that individual socioeconomic characteristics affect health purely through material pathways, regardless of the wider social context, has become less plausible over the years. As Marmot and others have argued,[8] an important part of why individual income is related to health probably reflects its role as a marker of social status. Similarly, the association between education and health probably reflects the importance not only of knowledge, but of the individual's educational status relative to other people.[9] Rather than a simple dichotomy between material effects of individual income unaffected by context, and context dependent psychosocial influences, psychosocial processes are probably important in both spheres.

When the empirical evidence of the effects of inequality was confined to health, it was reasonable to think that we should not assume that inequality had any psychosocial effects before we eliminated other possible explanations. But since then the evidence base has grown. It is now clear that unequal societies have an increased prevalence of a host of social problems, including violence, bullying, teenage births, higher rates of imprisonment, low educational performance, reduced social mobility, low levels of trust, and longer working hours.[10] Insofar as these are behavioural outcomes, they provide strong evidence that psychosocial processes are associated with inequality.

The benefits of greater equality tend to be largest among the poor but seem to extend to almost everyone.[10] A more equal society might improve most people's quality of life. Rather than merely paying lip service to creating a "classless society," it is a task for politicians and policy experts to repair our "broken society" by undoing the widening of inequalities that has taken place since the 1970s.

1 The big idea [Editor's Choice]. *BMJ* 1996;312. (20 April.)
2 Kondo N, Sembajwe G, Kawachi I, van Dam R, Subramanian SV, Yamagata Z. Income inequality, mortality and self rated health: A meta-analysis of multilevel studies. *BMJ* 2009;339:b4471.
3 Deaton A. Health, inequality, and economic development. *J Econ Lit* 2003;41:113-58.
4 Lynch J, Smith GD, Harper S, Hillemeier M, Ross N, Kaplan GA, et al. Is income inequality a determinant of population health? Part 1. A systematic review. *Milbank Q* 2004;82:5-99.
5 Subramanian SV, Kawachi I. Income inequality and health: what have we learned so far? *Epidemiol Rev* 2004;26:78-91.
6 Wilkinson RG, Pickett KE. Income inequality and population health: a review and explanation of the evidence. *Soc Sci Med* 2006;62:1768-84.
7 Berkman LF, Glass T, Brissette I, Seeman TE. From social integration to health: Durkheim in the new millennium. *Soc Sci Med* 2000;51:843-57.
8 Marmot M. Status syndrome: how your social standing directly affects your health and life expectancy. Bloomsbury, 2004.
9 Chandola T, Clarke P, Morris JN, Blane D. Pathways between education and health: a causal modelling approach. *J R Stat Soc Ser A* 2006;169:337-59.
10 Wilkinson R, Pickett K. The spirit level: why more equal societies almost always do better. Allen Lane, 2009.

Control of hepatitis B and C worldwide

Treatment is costly with limited effectiveness: prevention is essential

Rakesh Aggarwal additional professor
aggarwal.ra@gmail.com
Amit Goel senior resident,
Department of Gastroenterology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow 226014, India

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

Cite this as: *BMJ* 2009;339:b4431
doi: 10.1136/bmj.b4431

Hepatitis B virus (HBV) and hepatitis C virus (HCV) are hepatotropic viruses that share several characteristics, including transmission through percutaneous routes, such as transfusion of blood products and contaminated injections. The viruses often cause persistent infection with serious long term sequelae such as liver cirrhosis and hepatocellular cancer.

HBV infection is highly endemic (the prevalence of hepatitis B surface antigen exceeds 8% in the general population) in sub-Saharan Africa, Southeast Asia, and Western Pacific regions, and HCV infection is highly prevalent in Egypt, affecting 15-20% of the population. Globally, nearly 340 million and 170 million people are thought to be chronically infected with HBV and HCV, respectively,[1 2] far exceeding the 33 million people infected with HIV.

Chronic HBV and HCV infection are responsible for nearly 57% of cases of liver cirrhosis (HBV 30%, HCV 27%) and 78% of hepatocellular cancers (HBV 53%, HCV 25%) worldwide and cause nearly one million deaths each year.[3] Furthermore, these infections often affect young people, impairing their quality of life and productivity. Safe vaccines with more than 95% protective efficacy against HBV have been available for more than two decades. In 1991, the World Health Organization recommended that hepatitis B vaccine should be included in national infant immunisation programmes. By 2007, 171 of the 193 member states had complied, with 65% of the annual

birth cohort receiving the recommended three doses.[4] As a result, carrier rates of hepatitis B surface antigen in children plummeted from 9.8% to 0.7% in Taiwan.[5] Furthermore, the incidence of hepatocellular cancer in vaccinated Taiwanese children aged 6-9, 10-14, and 15-19 years decreased from 0.51, 0.60, and 0.52 per 100 000 person years, respectively, to 0.15, 0.19, and 0.16 per 100 000 person years—a decrease of 69%.[6]

These encouraging results recently led the WHO Western Pacific region, home to half the global HBV carrier pool, to aim for lower than a 2% seroprevalence of hepatitis B surface antigen in children aged 5 years by 2012.[7] It was thought this could be achieved by improving coverage of the first dose given within 24 hours of birth. The progress to date indicates that this goal is within reach.

Unfortunately, no vaccine is available for HCV. It may be difficult to develop one, because of the high mutation rate and substantial heterogeneity of the HCV genome, and poor understanding of immunological phenomena underlying viral clearance by the host. Lack of a small animal model and, until recently, of a reliable virus culture system have posed extra problems. Several strategies have been tried,[8] but we are still far from a clinical vaccine.

Vaccines cannot help people who are already infected. Such patients need treatments that can either clear the infecting virus or control its multiplication, thereby preventing long term complications. Major advances in the treatment of HBV and HCV infection have occurred in the past two decades.

Treatment options for chronic hepatitis B can be divided broadly into injectable immunomodulators—interferon and pegylated interferon—and oral nucleos(t)ide analogues.[1] Advantages of interferon based regimens include their defined duration of administration (four to 12 months) and their lasting response, albeit in a subset of patients. However, they are costly, contraindicated in patients with advanced cirrhosis, and often have to be discontinued because of serious adverse events.

Nucleos(t)ide analogues are safe but have lower response rates, an uncertain duration of treatment (which can be life long), and a higher risk of recurrence on stopping treatment. Low cost nucleos(t)ide analogues are also associated with frequent development of drug resistant viral mutants, which decreases their clinical effectiveness. Newer ones have a lower risk of drug resistance but cost more. Combination chemotherapy reduces the risk of development of drug resistant mutants but is associated with the appearance of multidrug resistant mutants and increased cost. Thus, despite a bevy of drugs being available, results remain suboptimal.

ELISA HABERER/CORBIS

The only currently approved treatment for chronic hepatitis C is a combination of interferon or pegylated interferon and ribavirin. This treatment has overall sustained virological response rates of 54-56%, which vary with viral genotype, host factors, and stage of liver disease. Besides high cost and interferon related adverse events, ribavirin induced haemolysis is another problem. Several novel compounds including interferon congeners, ribavirin analogues, inhibitors of viral NS5B polymerase and NS3-4A protease, monoclonal or polyclonal antibodies against HCV envelope proteins, antisense nucleotides, ribozymes, and small interfering RNAs are being developed.[9]

Unfortunately, most treatment trials for both HBV and HBC have used short to intermediate term laboratory end points, rather than long term ones that are clinically relevant. Also, current treatment protocols are beyond the reach of patients in resource poor countries that have the largest burden of disease. Thus, drug treatment is unlikely to have a major effect on the global burden of these diseases in the near future.

We thus need to ramp up preventive efforts. Hepatitis B immunisation can prevent up to 84% of global HBV related deaths.[10] To achieve this we need to ensure that hepatitis B immunisation is introduced in countries that have failed to do so and improve the coverage of the dose given at birth.

Unsafe injections are estimated to cause nearly 21.7 million and two million new HBV and HCV infections, respectively, each year.[11] These may be prevented by educating healthcare workers to avoid unnecessary injections, which account for up to 70% of injections in some regions, and the reuse of syringes, which is estimated to occur in 40% of injections worldwide.[11]

In 2001-2 nearly 40% of countries lacked universal pretransfusion testing of blood, and 47.5% of blood units transfused in underdeveloped countries had not been screened for HCV.[12] Although the situation may have improved since then, promotion and strict implementation of such screening should pay rich dividends.

1 Lok ASF, McMahon BJ. AASLD practice guidelines. Chronic hepatitis B: update 2009. 2009. www.aasld.org/practiceguidelines/Documents/Bookmarked%20Practice%20Guidelines/Chronic_Hep_B_Update_2009%208_24_2009.pdf.
2 WHO. Hepatitis C: global prevalence (update). *Wkly Epidemiol Rec* 1999;74:425-7.
3 Perz JF, Armstrong GL, Farrington LA, Hutin YJF, Bell BP. The contributions of hepatitis B virus and hepatitis C virus infections to cirrhosis and primary liver cancer worldwide. *J Hepatol* 2006;45:529-38.
4 WHO. WHO vaccine-preventable diseases: monitoring system—2008 global summary. 2008. http://whqlibdoc.who.int/hq/2008/WHO_IVB_2008_eng.pdf.
5 Chan C-Y, Lee S-D, Lo K-J. Legend of hepatitis B vaccination: the Taiwan experience. *J Gastroenterol Hepatol* 2004;19:121-6.
6 Chang M-H, You S-L, Chen C-J, Liu C-J, Lee C-M, Lin S-M, et al; the Taiwan Hepatoma Study Group. Decreased incidence of hepatocellular carcinoma in hepatitis B vaccinees: a 20-year follow-up study. *J Natl Cancer Inst* 2009;101:1348-55.
7 Rani M, Yang B, Nesbit R. Hepatitis B control by 2012 in the WHO Western Pacific region: rationale and implications. *Bull World Health Organ* 2009;87:707-13.
8 Stoll-Keller F, Barth H, Fafi-Kremer S, Zeisel MB, Baumert TF. Development of hepatitis C virus vaccines: challenges and progress. *Expert Rev Vaccines* 2009;8:333-45.
9 Webster DP, Klenerman P, Collier J, Jeffery KJM. Development of novel treatments for hepatitis C. *Lancet Infect Dis* 2009;9:108-17.
10 Goldstein ST, Zhou F, Hadler SC, Bell BP, Mast EE, Margolis H. A mathematical model to estimate global hepatitis B disease burden and vaccination impact. *Int J Epidemiol* 2005;34:1329-39.
11 WHO. Injection safety. 2006. www.who.int/mediacentre/factsheets/fs231/en/print.html.
12 WHO. WHO global database on blood safety: report 2001-2002. 2004. www.who.int/bloodsafety/GDBS_Report_2001-2002.pdf.

Population, gender, and climate change

Improving access to family planning services and promoting sexual equality are the priority

JACOB SILBERBERG/PANOS

NEWS, p 1165

Karen Hardee vice president for research, Population Action International, 1300 19th Street, NW, Ste 200, Washington, DC 20036, USA
KHardee@popact.org

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

Cite this as: *BMJ* 2009;339:b4703
doi: 10.1136/bmj.b4703

The 2009 state of the world population report from the United Nations Population Fund (UNFPA) tackles what UN Secretary General Ban Ki-moon has termed the greatest challenge facing humanity.[1 2] The report, "*Facing a Changing World: Women, Population and Climate*," is published just weeks before representatives of the world's nations arrive in Copenhagen to forge a new climate treaty. The treaty will hopefully include agreements to reduce emissions globally and equitably, and ways of adapting and building resilience in countries most vulnerable to the effects of the changing climate. The UNFPA report should convince negotiators to pay more attention to women's crucial role in mitigating and adapting to climate change and to ensure that population factors stay on the table—within the rights based framework of the programme of action of the 1994 International Conference on Population and Development.

Women and men are affected and respond differently to the challenges of climate change. The UNFPA report highlights the vulnerability and disadvantaged position of women as the global temperature rises—women fare worse than men as a result of chronic food and water insecurity as well as natural disasters. In Malawi, Mozoe Gondwe, a farmer, says she can no longer predict when the rains will come. "I grew up in the area and I know how the system is changing."[1] Within the next 40 years, average temperatures in Malawi could rise by at least 1°C, with a substantial fall in agricultural yields. In addition, Malawi's population is estimated to triple almost, from 15 million to 42 million by 2050.

At the same time, the report shows that women, as the world's primary farmers, tend to be better stewards of the earth than men. Women are also the most sustainable consumers,[3 4] and their participation is crucial to navigate climate change successfully. The presence of women's organisations in low income countries may help protect forests against destruction.[5] In India, a collective of 5000 women in 75 villages in Andhra Pradesh is working on chemical free, non-irrigated, organic agriculture as a response to global warming.[1] Examples such as this are found across the world from Bolivia to Nepal to Kiribati. Wangari Maathai of Kenya won the Nobel Peace Prize for her work through the Green Belt Movement she founded in Kenya to rejuvenate the environment by planting trees. Monique Barbut is executive director of the Global Environment Facility, which among other things, administers funding for programmes on climate adaptation.

How many people the earth has to sustain is also important, as well as where they live, what they consume, what types of household they live in, and their age patterns. The UNFPA report states that if the world's population remained at 300 million as it was 1000 years ago, rather than the 6.8 billion it is today, "greenhouse gases would not be accumulating so hazardously."[1] Similarly, whether the world's population reaches the United Nations Population Division's "low variant projection" of 8.0 billion in 2050 rather than the high estimate of 10.5 billion will make a difference to carbon emissions, and our ability to cope with climate changes. One study suggests that achieving the low variant projection for 2050, rather than the medium growth scenario of around nine billion people, could result in one to two billion fewer tons of carbon emissions.[6]

Of the 41 least developed countries that have prepared National Adaptation Programmes of Action (NAPA), 37 note that population pressure is exacerbating the effects of climate change.[7] Yet few of these programmes include family planning and reproductive health as priorities, partly because the global architecture for tackling climate change favours technological solutions over actions to strengthen human capital. In NAPA countries with relevant data, around 20% of women say they have enough children and want to stop or wait to have their next child, yet are not using contraception. Around the world, more than 200 million women have an unmet need for family planning. In the United States, half of all pregnancies are unplanned.

Strengthening rights based voluntary family planning programmes along with education and livelihood programmes, and integrating them with projects designed to tackle food and water insecurity, is a "no lose" strategy for Mozoe, Malawi, and the world. Such integrated approaches will help meet the expressed needs of individuals to plan their families, slow population growth, alleviate pressure on limited food and water resources, and help reduce carbon emissions. Investments in voluntary family planning and girls' education are cost effective ways to reduce carbon emissions, compared with other strategies including nuclear energy and wind energy.[8]

In the lead-up to Copenhagen, the UNFPA report highlights the need for governments to expand access to voluntary family planning and reproductive health, and to achieve equality between the sexes. Taking these steps and ensuring that women have a strong voice in all aspects of the climate discussion—as scientists, advocates, and policy makers—will contribute to the global challenge of reducing carbon emissions and helping people adapt to climate change.

1 United Nations Population Fund. Facing a changing world: women, population and climate. State of the world population report 2009. UNFPA, 2009.
2 Climate change world's "greatest challenge": UN chief. *World News* 10 August 2009. http://article.wn.com/view/2009/08/10/Climate_change_worlds_greatest_challenge_UN_chief/.

3 Organization for Economic Cooperation and Development. Promoting sustainable consumption: good practices in OECD countries. 2008. www.bcsdportugal.org/files/1890.pdf.
4 Organization for Economic Cooperation and Development. Environmental policy and household behaviour: evidence in the areas of energy, food, transport, waste and water. 2009.
5 Shandra JM, Shandra C, London B. Women, non-governmental organizations, and deforestation: a cross-national study. *Popul Environ* 2008;30:48-72.
6 O'Neill B. 2009. Population and change and population growth. In Mazur L. *A pivotal moment: population, justice and the environmental challenge*. Island Press, 2009.
7 Mutunga C, Hardee K. Population and reproductive health in National Adaptation Programmes of Action (NAPAs) for climate change. PAI Working Paper WP09-4. Population Action International, 2009. www.populationaction.org/Publications/Working_Papers/August_2009_Climate/WP09-04_NAPA.pdf.
8 Wheeler D, Hammer D. The economics of population policy for carbon emissions reduction. Working Paper. Center for Global Development (in press).

Capping earnings from private patients in NHS foundation trusts

Higher earnings must not compromise patient care and use of public money

John Appleby chief economist, King's Fund, London W1G 0AN
j.appleby@kingsfund.org.uk

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

Cite this as: *BMJ* 2009;339:b4698
doi: 10.1136/bmj.b4698

The boundaries between public and private care in the NHS have never been clearly demarcated—not even in theory, let alone in practice. Recently the Department of Health announced that it will review the cap on foundation trusts' earnings from private patients.[1] As with the recent review of top-up payments,[2] the review is the latest example of re-emerging tensions arising from historic fudging of a messy and complicated subject.

The department wants an alternative way of regulating private income that protects the interests of NHS patients and prevents taxpayers subsidising private care, but that also deals with anomalies in the current capping system, which have led to perceived unfairness between NHS hospitals and a potential restriction on innovation and trade.

The private patient income (PPI) cap was set out in section 44 of the National Health Service Act 2006 and was designed to allay fears that, in the words of the latest review consultation, "[there was] a potential risk that freedom from ministerial powers of direction could give NHS foundation trusts an incentive to expand their private patient activity to the detriment of their principle purpose to provide NHS goods and services." As implied, the cap applies only to foundation trusts. The 2006 act also states that the PPI cap is fixed at the level earned by foundation trusts in 2002-3.

Although the total amount of income earned by NHS trusts from private patients is relatively small—a survey of 40 foundation trusts reported earnings of around £100m (€112m; $166m),[3] which suggests a total of between £300m and £400m as trusts vary widely. Many earn little or nothing from private work, but for some it is a vital source of revenue. The Royal Marsden, for example, has a PPI cap fixed at nearly 31% of its total patient income; last year the trust earned more than £43m from private patients—29% of its total income (this excludes payments to consultants).[4]

One answer to the variation in ability to earn private income could be to abolish the cap but have an enforceable and demonstrable requirement on all NHS trusts that their private activities must not interfere with their public duties to NHS patients and the taxpayer. With the Care Quality Commission rating the Royal Marsden as one of the best trusts in the country—it has been rated as "excellent" for both quality and finances for four years running[5]—it could be argued that even high levels of private income are not necessarily detrimental to NHS patients and taxpayers.

On the other hand, abolishing the cap may be seen by private healthcare providers as a threat to their business and their ability to attract NHS consultants to carry out sessions in private facilities.

Some critics of the PPI cap have pointed out that the definition of private patient income extends beyond the direct income received from such patients and covers other commercial activities, such as joint ventures where the trust has no overall control of the project, services to charities, and support to government programmes. As it happens, this extension of the definition of private work was decided by the foundation trust regulator, Monitor, in their own review of the PPI cap after a challenge from the health union Unison (which is still ongoing).[6 7] The Foundation Trust Network argues that this wider definition of private income could stifle innovation, as well as beneficial ventures and collaborations.[8]

The Department of Health has already decided to raise the PPI cap for mental health foundation trusts from zero to 1.5% of total patient income, and, as public funding starts to tighten in the coming years, further relaxations on the ability of the NHS to earn money from private sources are likely. NHS patients and taxpayers will not want this to result in a trade off against their care and the efficient use of public money.

1 Department of Health. Call for evidence—review of restrictions on private patient income: the NHS foundation trust private patient income cap. 2009. www.dh.gov.uk/prod_consum_dh/groups/dh_digitalassets/@dh/@en/documents/digitalasset/dh_107951.pdf.
2 Richards M. Usage of cancer drugs approved by NICE: report of review undertaken by the national cancer director. Department of Health, 2006.
3 Gainsbury S. Survey finds foundation trusts' hidden private income. *Health Serv J* 2009; www.hsj.co.uk/survey-finds-foundation-trusts-hidden-private-income/1735548.article.
4 Royal Marsden NHS Foundation Trust. 2008/9 annual report: our patients, our people, our passion. 2009. www.royalmarsden.nhs.uk/NR/rdonlyres/30B10669-2F6A-4799-8869-9173E8816422/0/AnnualReport0809.pdf.
5 Care Quality Commission. Care Quality Commission publishes NHS performance ratings. 2009 www.cqc.org.uk/newsandevents/newsstories.cfm?cit_id=35456&FAArea1=customWidgets.content_view_1&usecache=false.
6 Gainsbury S. Court hears private patient cap arguments. *Health Serv J* 2009; www.hsj.co.uk/news/legal/court-hears-private-patient-cap-arguments/5008116.article.
7 Monitor. Monitor announces result of consultation on interpretation and application of the private patient income cap. 2008. www.monitor-nhsft.gov.uk/home/news-and-events/media-centre/press-release-archive/2008/monitor-announces-result-consultation-i.
8 Slipman S. Increasing private patient work at NHS foundation trust hospitals would benefit us all. Joe Public Blog. *The Guardian* 2009. www.guardian.co.uk/society/joepublic/2009/nov/09/nhs-sue-slimpan-foundation-trust-network.