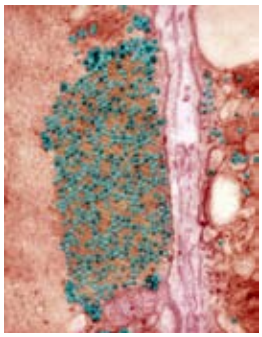
Enteroviruses and type 1 diabetes

The mechanism of the association is yet to be explained



COXSACKIE B3/AMI IMAGES/SPL

RESEARCH, p 421

Didier Hober professor of virology
didier.hober@chru-lille.fr
Famara Sane research assistant, Laboratory of Virology/EA3610, University Lille 2, Faculty of Medicine, CHRU Lille, 59120 Loos-lez-Lille, France

Competing interests: All authors have completed the Unified Competing Interest form at www.icmje.org/coi_disclosure.pdf (available on request from the corresponding author) and declare: no support from any organisation for the submitted work; no financial relationships with any organisations that might have an interest in the submitted work in the previous three years; no other relationships or activities that could appear to have influenced the submitted work.

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

Cite this as: *BMJ* 2010;341:c7072
doi: 10.1136/bmj.c7072

Type 1 diabetes is an autoimmune disease of the endocrine pancreas that results in impairment of insulin producing pancreatic β cells. Inflammation of the pancreas correlates with the onset of symptoms, and susceptibility to the disease is influenced by genetic factors.[1]

The incidence rate of type 1 diabetes has increased over the past 25 years at an annual rate of 3%, but this cannot be explained only by genetic modifications in the population. It has therefore been suggested that environmental factors—such as drugs; toxins; nutrients (for example, cows' milk); and viruses like rotaviruses, adenoviruses, retroviruses, reoviruses, cytomegalovirus, Epstein-Barr virus, mumps virus, or rubella virus—can play a role in the pathogenesis of the disease. Viruses of the enterovirus genus, which have an RNA genome, are the most likely candidates, especially serotypes like coxsackie B virus belonging to the human enterovirus B species.[2]

In the linked systematic review, Yeung and colleagues assessed the association between current enterovirus infection diagnosed by molecular testing and the development of autoimmunity or type 1 diabetes.[3]

The first report about the possible association between enteroviruses and type 1 diabetes was published in 1969 in the *BMJ* and was based on the detection of anti-enterovirus antibodies in patients' serum.[4] Better methods for detecting viruses have enabled further testing of the hypothesis. Enteroviruses have since been detected in blood, the gut, and the pancreas of patients with type 1 diabetes. Furthermore, there is an association between enterovirus infections and type 1 diabetes in genetically predisposed people,[5] and data from in vitro studies and animal models support the hypothesis of a role of enteroviruses in the diseases.

Yeung and colleagues, who report the first meta-analysis of molecular studies (based on detection of viral protein and RNA) find a strong association between enterovirus infection and type 1 diabetes (odds ratio 9.77, 95% confidence interval 5.50 to 17.35).[4] The results contrast with a previous meta-analysis of serological studies, which found no association.[6] The discrepancy might be because the serological studies examined only certain serotypes and the identification of current or recent infection was unclear.

However, Yeung and colleagues' review agrees with most retrospective and prospective epidemiological studies, which found higher rates of enterovirus infection in patients than in controls.[7] The possible role of enteroviruses is also supported by recent reports showing that enteroviruses, the patients' genotype, and their immune response are linked in the pathogenesis of type 1 diabetes.[8]

It is unclear whether enteroviruses are involved in all patients or just some. The lack of detection of enteroviruses in the blood or tissues of all patients with type 1 diabetes might be because the test is not sensitive enough. Furthermore, enteroviruses may be released only occasionally from sites such as the gut to reach the pancreas and mostly go undetected. More sensitive methods are therefore needed to investigate the presence of enteroviruses in patients, as well as longitudinal studies to assess the persistence of enteroviruses in patients.

Prospective studies suggest an association between enterovirus infections and the subsequent production of autoantibodies directed against pancreatic β cells that result in type 1 diabetes.[5] In addition, the detection of enteroviruses at the onset of disease suggests that these viruses, through persistence or consecutive infections, can play a role in the progression or acceleration of the disease. The role of viruses is complex, however, because they can also protect against the disease,[9] and animal studies suggest that the consequences of enteroviral infections might differ according to the patient's age.[10]

The link between enteroviruses and the pathogenesis of type 1 diabetes probably involves an interplay between viruses, pancreatic β cells, the innate and adaptive immune systems, and the genotype of the patient.[2] Further studies are needed to tease out the association of these factors and to establish the pathogenic mechanisms of enterovirus infections.

The association between enteroviruses and type 1 diabetes opens up the possibility of developing new preventive and therapeutic strategies to fight this disease.

1 Eizirik DL, Colli ML, Ortis F. The role of inflammation in insulitis and beta-cell loss in type 1 diabetes. *Nat Rev Endocrinol* 2005;5:219-26.
2 Hober D, Sauter P. Pathogenesis of type 1 diabetes mellitus: interplay between enterovirus and host. *Nat Rev Endocrinol* 2010;6:279-89.
3 Yeung W-CG, Rawlinson WD, Craig ME. Enterovirus infection and type 1 diabetes mellitus: systematic review and meta-analysis of observational molecular studies. *BMJ* 2011;342:d35.
4 Gamble DR, Kinsley MJ, Fitzgerald MG, Bolton R, Taylor KW. Viral antibodies in diabetes mellitus. *BMJ* 1969;3:627-30.
5 Jaïdane H, Sauter P, Sane F, Goffard A, Gharbi J, Hober D. Enteroviruses and type 1 diabetes: towards a better understanding of the relationship. *Rev Med Virol* 2010;20:1-16.
6 Green J, Casabonne D, Newton R. Coxsackie B virus serology and type 1 diabetes mellitus: a systematic review of published case-control studies. *Diabet Med* 2004;21:507-14.
7 Oikarinen S, Martiskainen M, Tauriainen S, Huhtala H, Ilonen J, Veijola R, et al. Enterovirus RNA in blood is linked to the development of type 1 diabetes. *Diabetes* 2010 October 13; doi:10.2337/db10-0186.
8 Von Herrath M. Diabetes: a virus-gene collaboration. *Nature* 2009;459:518-9.
9 Bach JF. Infections and autoimmune diseases. *J Autoimmun* 2005;25:74-80.
10 Tracy S, Drescher KM, Jackson JD, Kim K, Kono K. Enteroviruses, type 1 diabetes and hygiene: a complex relationship. *Rev Med Virol* 2010;20:106-16.

The north-south health divide

The NHS must do more than pick up the pieces

RESEARCH, p 423

Margaret Whitehead WH Duncan professor of public health, Department of Public Health and Policy, University of Liverpool, Liverpool L69 3GB, UK
mmw@liverpool.ac.uk
Tim Doran clinical research fellow, School of Health Sciences, University of Manchester, Manchester, UK

Competing interests: All authors have completed the Unified Competing Interest form at www.icmje.org/coi_disclosure.pdf (available on request from the corresponding author) and declare: no support from any organisation for the submitted work; no financial relationships with any organisations that might have an interest in the submitted work in the previous three years; no other relationships or activities that could appear to have influenced the submitted work.
Provenance and peer review: Commissioned; not externally peer reviewed.

Cite this as: *BMJ* 2011;342:d584
doi: 10.1136/bmj.d584

The north-south divide is one of England's most powerful and enduring myths: one country geographically, politically, and culturally separated into beautiful south and grim north. On one side rolling fields, dazzling sunshine, conspicuous wealth, conservatism, and flat beer; on the other coal, cloud, desolation, socialism, and froth.

In the linked observational study, Hacking and colleagues show that not only is the divide in premature mortality real, but that it has persisted and continued to widen over four decades and under five governments.[1] Since 1965, the toll of excess deaths has surpassed 1.5 million—the north is being decimated at the rate of a major city every decade.

Many explanations have been offered for this northern catastrophe. Hacking and colleagues discuss the contributions of genetics, lifestyle, and migration and find them modest. Explanations related to the differing social composition of the population in the north compared with the south also fall short, because southerners enjoy better health across the entire social spectrum.[2] Any damage inflicted by climate and geology is likely to be indirect: the cotton industry developed where the weather was damp, the coal industry where there were seams to exploit. These and other industries provided enough income to engender dependency, but too little to generate affluence. When the industries declined, entire communities withered with them.

The evidence suggests that the underlying causes of the divide are social and economic, so bridging it will require social and economic solutions. In 1997, the incoming New Labour government broke with its predecessor in acknowledging the existence of health inequalities and mounted a cross departmental campaign against them. The health divide continued to grow, however, and for people under 75 it is now at its widest for 40 years.[1] Having reached this nadir in the relatively favourable economic and political climate of the 2000s, future prospects look grim.

Analysis of the impact of the current recession shows that deprived communities in the northern city regions have borne the brunt. Unemployment has increased most in manufacturing areas, where it was already high, exacerbated by the disproportionate effects of the recession on housing led regeneration efforts in the north.[3] Government spending cuts will also hit hardest in the north, which will sustain greater proportional job losses in the public and private sectors and has a greater reliance on welfare benefits and public services that are being cut.[4] This increasingly intractable problem can only be solved by renewed regional development policies, which have repeatedly been stressed in the past but are insufficiently implemented.

Given its limited influence on the underlying causes, can the NHS do more than pick up the pieces? The Royal College of Physicians envisages an important role for clinicians as advocates, educators, and partners of public health specialists and local authorities.[5] These responsibilities will fall predominantly on general practitioners, both as providers of primary care and as commissioners. As providers, GPs have made substantial advances over the period of Hacking and colleagues' study. Inner city practices are no longer "the source of public danger" that some of them were in the 1950s,[6] and management of chronic conditions has improved markedly over the past decade.[7] The efforts of practices in response to quality improvement initiatives, particularly in deprived areas, have yielded more equitable care in terms of secondary prevention.[8] This has yet to be reflected, however, in outcomes.

It is also not clear whether the recent emphasis on secondary prevention through the Quality and Outcomes Framework (QOF) has distracted attention from primary prevention. In their new commissioning role,[9] GPs will be hampered by the loss of the ability to plan for whole populations in defined geographical areas as a result of the switch from primary care trusts to consortiums based on registered patients.[10] Effective collaboration with public health services and local authorities will be crucial, at a time when local authorities are under immense financial pressure. It will be imperative for GP consortiums to take on public health expertise to incorporate a population perspective into their commissioning processes. This applies to commissioning of services across the board, but especially in relation to tackling the social determinants of health and the health divide.[10]

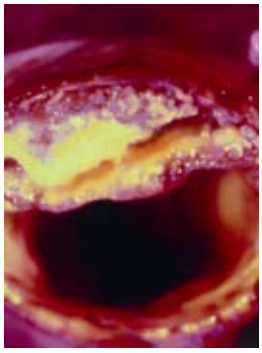
The activities of local consortiums will be constrained by the demands and allocations of the NHS Commissioning Board, which will have to balance its duty to tackle inequalities with those to improve quality and promote patient choice. A practical challenge for the board will be the distribution of GPs, which had become less equitable, year on year, since the early 1990s, and especially since the abolition of entry controls in 2002.[11] More fundamentally, the board will have to ensure the equitable allocation of NHS resources, taking account of the increased needs associated with deprivation around the country. This applies both to the formula for the allocation of commissioning funds to GP consortiums and to the transfer of ring fenced public health budgets to local authorities.[12]

Last, but not least, the board or the Department of Health must take a national overview of the cumulative effects of resource allocation to, and commissioning decisions made by, GP consortiums on equitable access to NHS services for different sections of the population living in different parts of the country. Otherwise, the result could be chaos and an even wider health divide.

1 Hacking JM, Muller S, Buchan IE. Trends in mortality between the English north-south divide from 1965 to 2008: comparative observational study. *BMJ* 2011;342:d508.
2 Doran T, Drever F, Whitehead M. Is there a north-south divide in social class inequalities in health in Great Britain? Cross-sectional study using data from the 2001 census. *BMJ* 2004;328:1043-5.
3 Dolphin T. *The impact of recession on northern city-regions*. Institute for Public Policy Research North, 2009. www.ippr.org.uk/ipprnorth/publicationsandreports/publication.asp?id=706.
4 Cox E, Schmuecker K. *Well north of fair: the implications of the spending review for the north of England*. Institute for Public Policy Research North, 2010. www.ippr.org.uk/ippmorth/publicationsandreports/publication.asp?id=782.
5 Royal College of Physicians. How doctors can close the gap: tackling the social determinants of health through culture change, advocacy and education. 2010. http://greenerhealthcare.org/events/2010/06/how-doctors-can-close-gap-tackling-social-determinants-health-through-culture-change.
6 Collings J. General practice in England today: a reconnaissance. *Lancet* 1950;i:555-85.
7 Campbell S, Reeves D, Kontopantelis E, Sibbald B, Roland M. Effects of pay for performance on the quality of primary care in England. *N Engl J Med* 2009;361:368-78.
8 Doran T, Fullwood C, Kontopantelis E, Reeves D. Effect of financial incentives on inequalities in the delivery of primary clinical care in England: analysis of clinical activity indicators for the quality and outcomes framework. *Lancet* 2008;372:728-36.
9 Department of Health. Equity and excellence: liberating the NHS. 2010. www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_117353.
10 Whitehead M, Hanratty B, Popay J. NHS reforms: untried remedies for misdiagnosed problems? *Lancet* 2010;376:1373-5.
11 Goddard M, Gravelle H, Hole A, Marini G. Where did all the GPs go? Increasing supply and geographical equity in England and Scotland. *J Health Serv Res Policy* 2010;15:28-35.
12 Department of Health. Healthy lives, healthy people: our strategy for public health in England. 2011. www.dh.gov.uk/en/Aboutus/Features/DH_123524.

C reactive protein and the risk of cardiovascular disease

Are clearly linked but a causal association is unlikely



ATHEROMA/BSIP VEM/SPL

RESEARCH, p 425

Bernard Keavney British Heart Foundation professor of cardiology, Institute of Human Genetics, Newcastle University, Newcastle upon Tyne NE1 3BZ, UK
b.d.keavney@ncl.ac.uk

Competing interests: BK has completed the Unified Competing Interest form at www.icmje.org/coi_disclosure.pdf (available on request from the corresponding author) and declares: no support from any organisation for the submitted work although his salary is paid by the British Heart Foundation; no financial relationships with any organisations that might have an interest in the submitted work in the previous three years; no other relationships or activities that could appear to have influenced the submitted work.

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

Cite this as: *BMJ* 2011;342:d144
doi: 10.1136/bmj.d144

Atherosclerosis is an inflammatory condition.[1] Observational studies have consistently shown that the concentrations of several systemic markers of inflammation measurable in blood are related to the risk of developing atherosclerosis. Among these associations, that between C reactive protein (CRP) and coronary heart disease has been most intensively studied.[2] In the linked study, the C Reactive Protein Coronary Heart Disease Genetics Collaboration combines the power of genetics and meta-analysis to assess whether higher concentrations of CRP cause coronary heart disease.[3]

High concentrations of CRP are seen in conditions such as bacterial sepsis, but the variation associated with the risk of coronary heart disease in epidemiological studies lies largely within the normal range for CRP. Statins lower CRP in addition to their effect on low density lipoprotein-cholesterol; the JUPITER trial showed that rosuvastatin substantially reduced cardiovascular events in apparently healthy people who did not have hyperlipidaemia (low density lipoprotein-cholesterol <3.4 mmol/L) but did have high concentrations of CRP (≥2.0 mg/L).[4] Since this study, the place for CRP in informing decisions about primary prevention with statins has been hotly debated.[5 6] However, a fundamental biological question—whether CRP itself predisposes to coronary heart disease, or whether it is an "innocent bystander"—has remained unanswered.

Because the processes leading to coronary heart disease have their origins in early life, the association between CRP and coronary heart disease seen in prospective studies could be explained by the presence of subclinical coronary heart disease (and thus, inflammation, which would result in higher CRP concentrations) in middle age, when such studies typically recruit apparently healthy participants. The possibility of such reverse causality makes it difficult to interpret the epidemiology.

The linked study uses a genetic approach known as mendelian randomisation, which is increasingly being used to assess controversial epidemiological associations.[7 8] The approach requires the presence of common genetic variations (typically, single nucleotide polymorphisms—substitutions of one of the four nucleotides in the DNA code by another at a known site in the genome) that are associated with plasma concentrations of the putative risk factor (here, CRP).

The authors typed single nucleotide polymorphisms in the gene for CRP that are known to be related to plasma CRP concentrations. Because genotype is allocated at conception, it cannot be affected by reverse causality. The study shows, in keeping with previous studies, an association between plasma CRP and risk of coronary heart disease (odds ratio 1.32 (95% confidence interval 1.19 to 1.47) for coronary heart disease per one standard deviation higher log plasma CRP) in more than 46 000 patients with coronary heart disease and more than 147 000 controls. It also confirms a substantial and significant association between genotype and plasma CRP. If the relation between CRP and coronary heart disease were causal, an association between genotype and risk of coronary heart disease would be expected, the size of which would be commensurate with the genotype-CRP and CRP-disease associations. However, no such association was seen. This suggests that CRP is unlikely to have even a small causal role in coronary heart disease, and that the observed associations between plasma CRP and coronary heart disease in prospective studies arise from reverse causality or residual confounding as a result of other causal factors (such as plasma lipids and body mass index) that are associated both with CRP concentrations and risk of coronary heart disease.

Previous studies have taken a similar approach to assessing the causality of the CRP-coronary disease association, and their results agree with those of the current study. For example, a meta-analysis in 2009 of primary genotyping data in 14 365 cases and 32 064 controls (with a literature based meta-analysis of a further 13 747 cases and 68 759 controls) found no association between single nucleotide polymorphisms that affect plasma concentrations of CRP and coronary heart disease, in the presence of significant single nucleotide polymorphism-plasma CRP and plasma CRP-coronary heart disease associations.[9] Because several

cohorts contributed data to both meta-analyses, the concordance is not surprising. However, the linked study had a 65% greater number of cases (the principal determinant of statistical power) than the previous meta-analysis, so if any small effect had been present, it would have had substantially more power to find it. The linked study also analysed individual participant data rather than relying mostly on summary data from the literature. This enabled additional analyses of genetic variants inherited together and appropriate correction for possible biases as a result of short term variations in plasma CRP within individuals.

The results of these mendelian randomisation experiments make even a small causal role for CRP in coronary heart disease unlikely. Similar results from mendelian randomisation studies, although using much smaller numbers of patients, argue against a causal role for CRP in type 2 diabetes, despite a well established epidemiological association between CRP concentrations and risk of diabetes.[10] However, such investigations do not contribute to the debate on the role of CRP in identifying patients who would benefit from treatment with statins to lower their risk of coronary heart disease.

So, where does that leave CRP? This marker is definitely associated with the risk of coronary heart disease, although it is unlikely to be causal. In future, clinical trials of recently discovered specific CRP antagonists or very low dose methotrexate that would reduce inflammation but not affect other risk factors for coronary heart disease may provide the final word in the debate.[11 12] However, it seems most likely that CRP is behaving as an integrative biomarker of the extent, and possibly the propensity to instability, of atheromatous plaques. In this way it can capture information about the risk of coronary heart disease that is complementary to levels of causative risk factors, such as low density lipoprotein-cholesterol. This holds out hope that other such biomarkers that will be useful in risk stratification may be discovered in the future.

1 Ross R. Atherosclerosis—an inflammatory disease. *N Engl J Med* 1999;340:115-26.
2 Kaptoge S, Di Angelantonio E, Lowe G, Pepys MB, Thompson SG, Collins R, et al. C-reactive protein concentration and risk of coronary heart disease, stroke, and mortality: an individual participant meta-analysis. *Lancet* 2010;375:132-40.
3 C Reactive Protein Coronary Heart Disease Genetics Collaboration (CCGC). Association between C reactive protein and coronary disease: mendelian randomisation analysis based on individual participant data. *BMJ* 2011;342:d548.
4 Ridker PM, Danielson E, Fonseca FA, Genest J, Gotto AM Jr, Kastelein JJ, et al. Rosuvastatin to prevent vascular events in men and women with elevated C-reactive protein. *N Engl J Med* 2008;359:2195-207.
5 Kaul S, Morrissey RP, Diamond GA. By Jove! What is a clinician to make of JUPITER? *Arch Intern Med* 2010;170:1073-7.
6 Ridker PM, Glynn RJ. The JUPITER Trial: responding to the critics. *Am J Cardiol* 2010;106:1351-6.
7 Keavney B. Commentary: Katan's remarkable foresight: genes and causality 18 years on. *Int J Epidemiol* 2004;33:11-4.
8 Keavney B, Danesh J, Parish S, Palmer A, Clark S, Youngman L, et al. Fibrinogen and coronary heart disease: test of causality by "Mendelian randomization." *Int J Epidemiol* 2006;35:935-43.
9 Elliott P, Chambers JC, Zhang W, Clarke R, Hopewell JC, Peden JF, et al. Genetic loci associated with C-reactive protein levels and risk of coronary heart disease. *JAMA* 2009;302:37-48.
10 Brunner EJ, Kivimaki M, Witte DR, Lawlor DA, Davey Smith G, Cooper JA, et al. Inflammation, insulin resistance, and diabetes—Mendelian randomization using CRP haplotypes points upstream. *PLoS Med* 2008;5:e155.
11 Pepys MB, Hirschfield GM, Tennent GA, Gallimore JR, Kahan MC, Bellotti V, et al. Targeting C-reactive protein for the treatment of cardiovascular disease. *Nature* 2006;440:1217-21.
12 Ridker PM. Testing the inflammatory hypothesis of atherothrombosis: scientific rationale for the cardiovascular inflammation reduction trial (CIRT). *J Thromb Haemost* 2009;7(suppl 1):332-9.

Helmets for skiers and snowboarders

Are protective, so better education and public awareness are now needed

Gerhard Ruedl postdoctoral researcher
gerhard.ruedl@uibk.ac.at
Martin Kopp professor
Martin Burtscher professor,
Department of Sport Science, University of Innsbruck, Innsbruck, Austria

Competing interests: All authors have completed the Unified Competing Interest form at www.icmje.org/coi_disclosure.pdf (available on request from the corresponding author) and declare: no support from any organisation for the submitted work; no financial relationships with any organisations that might have an interest in the submitted work in the previous three years; no other relationships or activities that could appear to have influenced the submitted work.

Provenance and peer review: Not commissioned; externally peer reviewed.

Cite this as: *BMJ* 2011;342:d857
doi: 10.1136/bmj.d857

In the winter of 2008-9, public awareness of the benefits of wearing ski helmets heightened after two celebrities were involved in fatal skiing injuries in Europe and North America.[1-3] In Austria, a German politician and a mother of four children collided on a ski slope on New Year's Day 2009. The politician, who was wearing a ski helmet, survived with a traumatic brain injury, whereas the woman, who was not wearing a helmet, died. In Canada, actress Natasha Richardson died after a traumatic head injury sustained while skiing without a helmet on a beginner slope in Quebec in March 2009.

During the weeks after the death of Natasha Richardson, visits to the emergency room at the Montreal Children's Hospital increased by 60%.[1] It was concluded that the media coverage had caused anxiety among parents, prompting those who might not otherwise have sought medical care to bring their children to the emergency room.[1] In addition, 15% of neurosurgeons in Germany, Switzerland, and Austria bought a helmet after the death of the German politician, possibly as a result of the increased media coverage.[2] The use of helmets increased in Austria from 44% in December 2008 to 57% in April 2009 in skiers and snowboarders, and it has become obligatory for children under 16 years in most Austrian provinces since the winter season 2009-10.[3] However, prospective studies evaluating the effects of this extended helmet use are still lacking.

What are the advantages of wearing a ski helmet? Head injuries account for 9-19% of all injuries reported by ski patrols and emergency departments.[3 4] Severe head injuries include traumatic brain injury, which is a leading cause of death among winter sports participants.[4] A recent meta-analysis showed that skiers and snowboarders with a helmet were significantly less likely than those without a helmet to have a head injury (odds ratio 0.65, 95% confidence interval 0.55 to 0.79). In children under 13 years the odds ratio was 0.41 (0.27 to 0.59).[4] A subsequent study found a similar effect across all age groups.[3] It has been suggested that head-neck-helmet biomechanics may increase the risk of cervical spine injury when wearing a helmet, especially in children, who have a greater head to body weight ratio.[4 5] However, recent studies have not confirmed this notion.[4 5] Thus, wearing a ski helmet

GEMSTONE IMAGES/GETTY

seems to make sense to prevent head injuries in all age groups.

Does wearing a ski helmet have disadvantages? According to the risk-compensation hypothesis, wearing a helmet may provide a false sense of security, resulting in riskier behaviour on the slopes.[6 7] However, one study of self reported behaviour found that although skiers and snowboarders who were risk takers skied faster than cautious people (53 *v* 45 km/h), the use of helmets was nearly equal in both groups (59.2% *v* 59.7%).[7] In addition, significantly more skilled skiers wore helmets (76.9% *v* 59.0%), and a similar proportion of those who did and did not wear helmets exhibited risk taking behaviour (29.8% *v* 30.2%).[7] Other studies have also shown that helmet use is higher in more skilled skiers than in less skilled ones.[6] So the use of a helmet is not necessarily associated with a higher level of risk taking but primarily with a higher level of skill.

Other arguments against helmets are that they impair hearing and limit the field of vision.[6 8] Only a few studies have assessed these aspects. A recent study showed that ski helmets could raise the hearing threshold of frequencies between 2 kHz and 8 kHz, which are characteristic of the hissing caused by a skier or snowboarder passing closely by or breaking behind.[8] However, sound was not attenuated at the frequencies characteristic of the human voice (<1 kHz), so that warning shouts should be heard.[8] In addition, the rules of the International Ski Federation (FIS) call for skiers to use their sight to avoid collisions. Collisions often lead to multiple trauma and are likely to involve the head. The victims of collisions are injured more often and more severely than those who cause the collision (93% *v* 25%), because the victim is usually hit unexpectedly and does not have time to react properly.[9]

One study found that most head injuries (74%) occurred when skiers hit their head on the snow,[10] 10% when they collided with other skiers, and 13% when they collided with fixed objects. These results suggest that protecting the head with a helmet must be beneficial.

In terms of the effect of helmets on field of vision, a randomised controlled pilot study found no differences in mean reaction time between people wearing a ski helmet or ski cap.[11] Ski goggles increased the reaction time, however, so may limit the field of vision.[11]

Evidence shows that ski helmets protect against head injury. Education about brain trauma can have a positive effect on attitudes towards wearing a helmet.[2] In addition to education and increased public awareness, helmet use could be increased and the incidence and severity of brain injuries decreased by the introduction of helmet loan schemes or routine inclusion of helmets in rental packages.[12] Future studies should evaluate strategies that focus on individual skiing behaviour. One example of such a study would be a randomised trial that compares the preventive effects of different educational (for example, web based) and behaviour change models. Public health physicians should take a leading role in research and in implementing measures for injury prevention.

1 Keays G, Pless IB. Impact of a celebrity death on children's injury-related emergency room visits. *Can J Public Health* 2010;101:115-8.
2 Jung CS, Zweckberger K, Schick U, Unterberg AW. Helmet use in winter sport activities—attitude and opinion of neurosurgeons and non-traumatic-brain-injury-educated persons. *Acta Neurochir* 2011;153:101-6.
3 Ruedl G, Sommersacher R, Woldrich T, Kopp M, Nachbauer W, Burtscher M. [Risk factors of head injuries on Austrian ski slopes]. *Dtsch Z Sportmed* 2010;61:97-102.
4 Russel K, Christie J, Hagel BE. The effects of helmets on the risk of head and neck injuries among skiers and snowboarders: a meta-analysis. *CMAJ* 2010;182:333-40.
5 Hagel BE, Russel K, Goulet C, Nettel-Aguirre A, Pless IB. Helmet use and risk of neck injury in skiers and snowboarders. *Am J Epidemiol* 2010;171:1334-43.
6 Sulheim S, Holme I, Ekeland A, Bahr R. Helmet use and risk of head injuries in alpine skiers and snowboarders. *JAMA* 2006;296:919-24.
7 Ruedl G, Pocecco E, Sommersacher R, Gatterer H, Kopp M, Nachbauer W, et al. Factors associated with self reported risk taking behaviour on ski slopes. *Br J Sports Med* 2010;44:204-6.
8 Tudor A, Ruzic L, Bencic I, Sestan B, Bonifacic M. Ski helmets could attenuate the sounds of danger. *Clin J Sport Med* 2010;20:173-8.
9 Burtscher M, Philadelphy M. Skiing collision accidents: frequency and types of injury. In: Mote CD, Ekeland A, eds. Skiing trauma and safety. Vol 10. ASTM International, 1996:73-6.
10 Greve MW, Young DJ, Goss AL, Degutis LC. Skiing and snowboarding head injuries in 2 areas of the United States. *Wilderness Environ Med* 2009;20:234-8.
11 Ruedl G, Herzog S, Schöpf S, Anewanter P, Geiger A, Burtscher M, et al. Do ski helmets affect reaction time to peripheral stimuli? *Wilderness Environ Med* [forthcoming.]
12 Levy AS, Hawkes AP, Rossie GV. Helmets for skiers and snowboarders: An injury prevention program. *Health Promot Pract* 2007;8:257-65.

Response on bmj.com

"The accumulated evidence about ski helmets is insufficient to allow one to state the benefits are clear, that skiers should be advised to wear them, or to imply public health doctors should be leading the charge."
Peter Ward, Central Gateshead Medical Group

To submit a rapid response, go to any article on bmj.com and select "Respond to this article"

Competition in the NHS in England

Debate about commissioning detracts from the radical extension of market principles in the Health and Social Care Bill

Chris Ham chief executive, King's Fund, London W1G 0AN, UK
c.ham@kingsfund.org.uk

Cite this as: *BMJ* 2011;342:d1035
doi: 10.1136/bmj.d1035

At the heart of the gargantuan Health and Social Care Bill published by the coalition government in January are provisions to establish a comprehensive system for the economic regulation of health and adult social care services. Part 3 of the bill is divided into eight chapters (far more than any other part of the bill), which encompass the role of Monitor as the economic regulator, plans for licensing of providers and setting prices, clauses on competition including the role of the Competition Commission and the Office of Fair Trading, and arrangements for insolvency and for a system of special administration to ensure continuity of designated services when providers fail. These provisions amount to the most ambitious attempt yet seen to apply a system of market regulation to the NHS.

Publication of the bill puts in perspective debates about the government's intentions that have dominated discussion since last May's election. Although many organisations have focused their attention on plans to give general practices a major role in commissioning

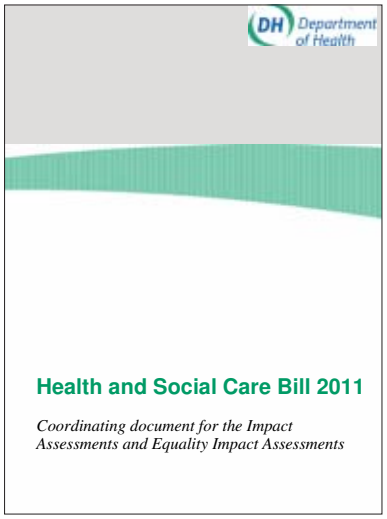
Competing interests: The author has completed the Unified Competing Interest form at www.icmje.org/coi_disclosure.pdf (available on request from the corresponding author) and declares: no support from any organisation for the submitted work; no financial relationships with any organisations that might have an interest in the submitted work in the previous three years; no other relationships or activities that could appear to have influenced the submitted work

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

bmj.com/nhsreforms

Access all *BMJ* articles about the proposed changes to the NHS in England at bmj.com/nhsreforms

health services and to require all NHS providers to become foundation trusts, these changes are of secondary importance compared with the radical extension of competition in healthcare. Building on the Thatcher government's internal market reforms in the 1990s and the Blair government's enthusiasm for choice and competition, David Cameron and his Health Secretary, Andrew Lansley, are going much further in putting in place the architecture they believe will enable the NHS to become truly world class.



The government's plans have been long in the making. As far back as July 2005, Andrew Lansley explained his philosophy of public service reform, invoking his experience as a civil servant working with Norman Tebbit on opening up the telecommunications sector to competition to set out seven principles to guide reform. These principles were to maximise competition, transfer risk to the private sector, ensure strong and independent regulation, set out standards and accountability clearly, specify universal service objectives and how they are to be funded, provide quality information for customers and maximise the number of providers, and ensure equitable access without sacrificing efficiency for equality.[1]

The provisions in the Health and Social Care Bill derive directly from these principles and underline the government's intention to draw on experience of privatising the utilities in taking forward the reform of the NHS. The question that arises is how applicable are these principles in view of the differences between healthcare and sectors like telecommunications? Also, will choice and competition help to transform the NHS and improve patient care or will they lead to increased fragmentation as Monitor exercises its duty "to promote competition where appropriate"?

On the positive side, the government has given much more thought to the mechanisms needed to make competition work in healthcare than its predecessors did. Whereas previous administrations have applied market principles in a piecemeal fashion and built up the architecture of competition incrementally, the coalition government has set out its proposals in detail from the outset. The government came into office with a clear plan and has moved rapidly to translate its ambitions into legislation; it has sought to learn from the failure of the Blair government by being bold in its approach to public service reform in its first term.[2]

On the negative side, the government's proposals run the risk of replacing the bureaucracy of performance management with the red tape of economic regulation. Monitor will need to employ large numbers of economists, lawyers, accountants, and managers to deal with competition issues, providers who fail, price setting, licensing providers, and other work. Add to this the need for Monitor to work hand in hand with the Competition Commission and the Office of Fair Trading on competition, the Care Quality Commission on regulation of quality, and the NHS Commissioning Board on price setting, and the complexities of the proposed regulatory arrangements become apparent.

The bigger question is whether competition in healthcare is the right route to take even if it has brought gains in other sectors. A recent review by Peter Smith, one of Britain's most respected health economists, for the Organisation for Economic Cooperation and Development found that evidence on the benefits of competition in healthcare is equivocal and underlined the challenges in applying market principles successfully. As the review concluded, "effective implementation of market-type mechanisms is . . . likely to require considerable managerial skills and impose substantial transaction costs, particularly in purchasing and regulatory institutions."[3]

The limits to markets have given rise to the argument that there should be increased collaboration between providers in many areas of care, including the provision of specialist cancer and cardiac services through networks and care for people with chronic diseases through integration of primary and secondary care.[4] Sensitive to the criticism that their plans may inhibit collaboration and increase fragmentation, ministers have gone on record as supporting integrated care where it will bring benefits.[5] This needs to be reflected in legislation to ensure that Monitor's duty to promote competition does not result in collaboration and integration being seen as anticompetitive.

Lessons from experience of applying market principles in the NHS since the 1990s also need to be heeded. In a highly visible public service like the NHS—the nearest thing we have to a national religion, according to Nigel Lawson[6]—it will always be difficult for politicians to distance themselves from controversial matters like reducing access to hospital services when providers fail to compete successfully, even if these decisions are taken by the regulator. In this respect, as in many others, healthcare is different from the former publicly owned utilities, underlining the political and technical challenges in adapting lessons from one sector to another.

1 Lansley A. Extract from "The future of health and public service regulation" speech. 2005. www.andrewlansley.co.uk/newsevent.php?newseventid=21.
2 Blair A. A journey. Hutchinson, 2010.
3 Smith P. Market mechanisms and the use of health care resources. In: Achieving better value for money in health care. Directorate for Employment, Labour and Social Affairs, Health Division, 2009.
4 Curry N, Ham C. Clinical and service integration: the route to improved outcomes. King's Fund, 2010. www.kingsfund.org.uk/publications/clinical_and_service.html.
5 Lansley A. Speech to the UCL partners/Monitor conference. 2011. www.gponline.com/News/article/1051076/video-lansley-responds-critics/.
6 Lawson N. The view from no.11: memoirs of a Tory radical by Nigel Lawson. Bantam Press, 1992.