Disinvestment in health care
A shared vocabulary, language, and narrative of change are needed

Four linked BMJ articles consider disinvestment in health care. They suggest several ways in which disinvestment can be promoted, including better evidence based clinical decision making; better alignment of health services between primary and secondary care providers; better integration of the health system with the social care system and community care; new technology; a culture of collaboration rather than competition; a better managed system of skill development; changes in working practice; the empowerment of patients; reductions in administrative costs; and greater dialogue to promote knowledge and understanding, so that policy options can be better discussed and agreed between relevant stakeholders. All of these are valuable insights into possible actions to promote change. However, they tend to underestimate the factors that promote resistance to the kinds of change a strategy of disinvestment is bound to cause.

Sociological and psychological research indicates why disinvestment is likely to meet resistance. Sociologists emphasise the contested problem of managerial control and how institutions tend to change only when a major shift in the nature of control occurs and a new management model is generally accepted. Psychologists emphasise resistance to change as a cognitive and emotional response at the individual and group level. We cling to what we know. Indeed, it seems natural to resist change, and it would be unexpected if major change was enthusiastically embraced. Studies of resistance to change imply that for change to happen managers need to be skilled in aligning individuals, groups, and stakeholders in terms of promoting more ways of framing contentious matters and they must tackle the problems of irrational thought processes. In the complex environment of health care, managing change requires skilful management at several levels, including leadership, culture (organisational and local), teams, and technology. In public sector change generally, we need to do better at engaging the front line in policy making. Studies of healthcare disinvestment identify five key challenges in managing disinvestment: lack of resources to support policy development; lack of agreement about comparative cost effectiveness within and between disciplines; political, clinical, and social resistance to removing an existing technology or practice; disputes over evidence; and lack of research into disinvestment as a policy option and practice. Discussions in this area have tended to focus on economic outcomes, but in these debates—for example, in relation to decisions by the National Institute for Health and Clinical Excellence about clinical effectiveness—we lack “national champions” of what needs to be “displaced” to fund new treatments. As a result, the debate has tended to remain fragmented, with little shared agreement on what needs to be done.

We lack a shared common language, a vocabulary, and a narrative of change for discussing the subject. Without this, an integrated policy of disinvestment will be difficult to introduce. Indeed, the very term “disinvestment” is problematic because for many it suggests reduced investment and divestment. It runs the risk of being associated only with cost reduction strategies, rather than with a coordinated policy of maximising the returns of investment in health care. We shouldn’t be looking only to cut things but to ensure that funding is focused on healthcare interventions and technologies that optimise health outcomes, individually and collectively.

To set a positive agenda of disinvestment, we need to convince healthcare professionals and the users of services that it can be an important means of freeing up resources, thereby improving the efficiency and the quality of health care. This will require a coordinated dialogue between healthcare managers and healthcare professionals to determine how a systematic, policy based approach to disinvestment is managed.

Body fat and increased risk of cirrhosis
Better diagnosis and treatment of non-alcoholic fatty liver disease are needed

Overweight (defined for European populations as a body mass index (BMI) of between 25 and <30) and obesity (defined as BMI ≥30) have become a considerable threat to public health worldwide. The prevalence of overweight and obesity continues to rise in most countries—for example, the proportion of over 16 year olds classified as obese in England rose from 15% in 1991 to 24% in 2007, and this has resulted in increased use of healthcare resources to treat the consequences of obesity. Established consequences of obesity include cardiovascular disease, diabetes, musculoskeletal problems, gallstones, and various cancers. In the linked studies, Liu and colleagues and Hart and colleagues assess the link between BMI, alcohol intake, and liver disease.

Using data from a cohort of 1.2 million women within the Million Women Study, who had no liver disease or cancer at baseline, Liu and colleagues found that the relative risk of liver cirrhosis increased by 28% for every 5 unit increase in BMI above 22.5 in each stratum of alcohol consumption. Women were recruited between 1996 and 2001 at a mean age of 56 years from NHS breast screening centres in the United Kingdom, and they self reported height and weight (with measured data available for a subgroup). Data on hospital admissions and deaths were collected from record linkage after a mean of 6.2 years. Over this period, 1811 women had a first hospital admission or died from cirrhosis and the absolute risk of cirrhosis was about one per 1000 women over five years.

Compared with the risk of cardiovascular events in middle aged people, an absolute risk of one case per 1000 people over five years seems low. However, this absolute risk still represents a substantial burden of illness for the patients concerned and for the health service. A key question is what proportion of cirrhosis can be attributed to modifiable risk factors? In tackling this question, the investigators concluded that during the study period 42% of all hospital admissions or deaths from liver cirrhosis could be attributed to alcohol consumption and 17% were caused by excess body weight.

Interestingly, the combination of obesity and an alcohol consumption of 150 g (about 18 units) or more each week was associated with a marked increased risk of cirrhosis (about fivefold) compared with that seen in obese women who drank less than 70 g of alcohol a week. The important question of whether alcohol and excess body fat have a simple additive adverse effect on the liver, or whether these two factors exert a synergistic effect to produce liver disease, is dealt with in the second of the two linked papers. Hart and colleagues analysed data from the Midspan prospective cohort studies of 9559 men in Scotland and investigated the relation between baseline BMI and self reported alcohol consumption at baseline on liver related morbidity and mortality after a median of 29 years (maximum 42 years). Both BMI and alcohol consumption were strongly and independently associated with mortality from liver disease, as shown by Liu and colleagues. Hart and colleagues also show that being overweight or obese and drinking 15 units of alcohol or more each week has a synergistic effect, which amplifies the insult to the liver and greatly increases the risk of liver related morbidity and mortality.

The association between increased BMI and cirrhosis can be explained by the biochemical and physiological consequences of obesity on the liver. Twenty years ago researchers began to appreciate the importance of insulin resistance in relation to cardiometabolic risk in what was first termed “syndrome X,” but is now known as the metabolic syndrome—a cluster of risk factors associated with central obesity. The presence of “ectopic” fat is thought to represent a key component of the metabolic syndrome with the accumulation of ectopic fat in the liver resulting in non-alcoholic fatty liver disease. This disease represents a spectrum of associated liver conditions, from simple steatosis to end stage cirrhosis and hepatocellular carcinoma. In as many as 40% of people with non-alcoholic fatty liver disease, the condition progresses over time to non-alcoholic steatohepatitis, with the development of additional hepatic inflammation. Non-alcoholic steatohepatitis may progress to advanced fibrosis and cirrhosis and carries an increased risk of hepatocellular carcinoma. Progressive fibrotic scarring results in a loss of hepatic parenchymal cells and thereby loss of fat laden liver cells, removing the clue that non-alcoholic fatty liver disease originally caused the liver fibrosis and cirrhosis.

BMI is a good proxy measure of central obesity and visceral body fat in epidemiological studies. Increased BMI in both the Million Women Study and the Midspan studies probably reflects the presence of central obesity and non-alcoholic fatty liver disease at the time of recruitment to the study.

What does this association mean for clinicians? Establishing a diagnosis of non-alcoholic fatty liver disease is difficult. Simple liver function tests, such as the measurement of serum alanine transaminase (ALT), have poor sensitivity and specificity as diagnostic tests. In addition, although liver ultrasound can detect liver fat, it cannot detect inflammation or early fibrosis. Non-alcoholic steatohepatitis can be identified only by liver biopsy—no other validated tests are available. Simpler, inexpensive, less invasive tests are needed to identify people with hepatic steatosis who are at risk of inflammation and progressive liver disease.

Risk factors for progression of non-alcoholic fatty liver disease include increasing age, smoking, and obesity. The role of moderate alcohol consumption is unclear because by definition non-alcoholic fatty liver disease should be diagnosed only in people who drink less than 10 g of alcohol a day. It is hoped that simple algorithms based on these risk factors and blood tests can be developed to identify people with non-alcoholic steatohepatitis so that liver biopsy will no longer be necessary. The causes of non-alcoholic steatohepatitis are complex and many unanswered questions remain, not least what causes progression of liver disease from simple steatosis to non-alcoholic steatohepatitis. Future research must
focus on developing an approach to diagnosing and treating non-alcoholic fatty liver disease. In the meantime, the old adage of “prevention is better than cure” remains pertinent to dealing with the problem of non-alcoholic fatty liver disease.11

The increasing prevalence of obesity and alcohol consumption over time, together with the increasing prevalence of hepatitis C, are contributing to the increasing incidence and prevalence of liver disease. Data from the Scottish health survey for 2003 (SHS 2003) show that the prevalence of obesity in women over 50 years was 32% compared with 18% for participants in the Million Women Study (between 1996 and 2001).12 Among men in SHS 2003, the prevalence of obesity was 24%, compared with 6% for Midspan participants (between 1965 and 1973). Even more worrying, the proportion of people who are obese and consume excess amounts of alcohol has increased over time. The proportion of men who were both obese and reported drinking more than 15 units of alcohol a week was 9.4% in SHS 2003, compared with 1.7% in Midspan. Reducing alcohol consumption and obesity are, at present, our only weapons against non-viral liver disease. The progression of non-alcoholic fatty liver disease to end stage liver disease can now be added to the list of the undesirable consequences of modern lifestyles.

Vitamin A supplements and survival in children

New evidence points to a differential effect in girls and boys

In the linked randomised controlled trial, Benn and colleagues assess the effect of giving high dose vitamin A supplements to low birthweight neonates in Guinea-Bissau. Overall, they found no effect on infant mortality, although the effect differed by sex; boys tended to benefit but a significantly harmful effect was seen on girls’ survival.13

In 1983 it was reported that young Indonesian children with mild xerophthalmia, Bittor’s spots, and night blindness—the clinical symptoms of vitamin A deficiency—had a higher risk of death.14 A subsequent randomised controlled trial of vitamin A supplementation showed an impressive benefit on all cause mortality.15 This prompted further large scale trials elsewhere in Asia and Africa that confirmed the effect. A meta-analysis indicated that this cheap and simple intervention reduces child mortality by 30% in countries with evidence of at least marginal vitamin A deficiency.16 The World Health Organization subsequently recommended a protocol for universal vitamin A supplementation of children aged six to 60 months, and this has been adopted as government policy in more than 60 countries worldwide.

If vitamin A supplementation is so effective at saving lives in later infancy and childhood, why not give supplements to newborn babies? Infant mortality is greatest in the first six months of life so it would be reasonable to expect an even greater benefit. The initial strategy was to supplement mothers postpartum in the hope that the benefit would be transferred through breast milk, but subsequent evidence questioned the efficacy of this approach. Safety concerns also surfaced after reports of acute bulging fontanelle after dosing, and it took some years and a WHO multicentre trial of vitamin A, administered when the neonate is given the diphtheria-pertussis-tetanus vaccine at 2, 3, and 4 months for these anxieties to subside.17 This paved the way for trials of neonatal vitamin A supplementation. Six such trials with reasonable power to detect effects on mortality were conducted. In 2008, WHO commissioned a meta-analysis of these trials and convened an expert advisory group to consider whether neonatal vitamin A supplementation should be adopted as policy. The meta-analysis found no survival benefit, but significant heterogeneity existed, with evidence of benefit from Asian trials and evidence of no effect (or harm) in two African trials.18 One of the African trials that showed a trend towards increased mortality after neonatal vitamin A supplementation was conducted by Benn and colleagues in Guinea-Bissau.19

Such a result was obviously unpalatable to the international vitamin A community, which had saved many thousands of lives through their advocacy and implementation of universal vitamin A supplementation. The finger was pointed at the fact that the Guinea-Bissau trial had intentionally excluded low birthweight babies. Surely the trial would have shown benefit if these most vulnerable neonates had been included? Sadly not. The current study by Benn and colleagues assesses neonatal vitamin A supplementation in 1717 low birthweight babies (<2500 g) born at the national hospital.1 In a by two by factorial trial, babies were randomised to receive 25 000 IU vitamin
A or placebo, and early BCG or the usual later BCG. The timing of BCG had no detectable effect on mortality, and—contrary to the authors’ initial expectation—they found no interaction between vitamin A and early BCG. The results for the two BCG arms were therefore combined in the analysis of the effect of supplementation.

The overall mortality rate ratio (MRR) for neonatal vitamin A supplementation versus placebo assessed up to 12 months was 1.08 (95% confidence interval 0.79 to 1.47), but a significant interaction with sex was seen. The MRR was 0.74 (0.45 to 1.22) in boys and 1.62 (0.94 to 2.15) in girls. These results were similar to the previous trial in Guinea-Bissau in normal birthweight babies. The authors now present a combined analysis which yields the following MRRs: overall 1.08 (0.87 to 1.33), boys 0.80 (0.58 to 1.10), and girls 1.41 (1.04 to 1.90), with a significant interaction (P<0.01) between neonatal vitamin A supplementation and sex.

The post hoc combination of two trials in the absence of a formal systematic review and meta-analysis is normally discouraged and must always be interpreted with caution. However, these two trials showed remarkable homogeneity, and they independently showed a tendency towards a harmful effect of vitamin A at birth that seemed to be confined to girls. In this case a combined analysis can be defended on the grounds that the two trials represent complementary arms covering the full birthweight spectrum and sex.

The authors have a reputation for challenging dogma in relation to vaccine and micronutrient supplementation policies. Through retrospective analysis of numerous datasets (their own and those of others) they have shown repeated examples of how vaccines, micronutrients, and exposure to infections can strongly affect all cause mortality in regions with a high burden of infection. In relation to the present context, Benn and colleagues have previously reported that vitamin A and DTP (possibly all killed vaccines) can have malign effects in girls that may be potentiated when the two are combined. Sceptics have argued that their evidence has been based on “unintended experiments,” and that the trends are often non-significant in isolation. The emergence of supporting evidence from prospective randomised controlled trials now cannot be ignored.

Sex differential effects, presumably relating to immune modulation, are certainly plausible. Sex differences in susceptibility to certain infections and in responses to vaccination have been recognised for decades.

Where do we go from here? With the support of the Bill and Melinda Gates Foundation, WHO has commissioned three large trials in an attempt to resolve the possibility that neonatal vitamin A supplementation may be beneficial in Asia but not in Africa. As commissioned, these trials are only required to assess mortality up to six months of age. Benn and colleagues rightly argue that failure to monitor mortality until 12 months would be a costly missed opportunity and may be dangerously misleading. If, as hypothesised, the potentially negative effects of early vitamin A supplementation in girls are potentiated by DTP, then a longer follow-up is essential. In Benn and colleagues’ combined analysis, the detrimental effect of neonatal vitamin A supplementation in girls had a MRR of 1.21 from birth to three months but 1.70 (1.08 to 2.67) between four and 12 months. Although the new trials are not individually powered to assess sex differential effects, they could help to resolve this controversy once and for all.

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Medical implications of the Taser

Serious harm is rare, but incident reporting needs to be improved

Several tactical options are available to police officers facing potentially aggressive or violent people or those with acute behavioural disturbance. The less lethal options include restraint, batons, incapacitant sprays, impact rounds, and conducted energy devices such as Tasers. Although none is risk free, Tasers have attracted particular controversy, with Amnesty International identifying more than 300 deaths associated with their use in the United States. However, association is not causation, and other factors complicate the interpretation of fatal outcomes.

The dominant conducted energy device used in police forces worldwide is the Taser X26. This device generates five second trains of electrical pulses that are delivered to the body either by two propelled barbs (which embed in clothing or skin and remain connected to the handset by conductive wire) or by direct contact of the handset’s electrodes (drive-stun mode). In the United Kingdom, propelled barbs are used by police in 90% of incidents in which such a device is discharged. Anecdotal evidence indicates that the threat of discharge alone may be an effective deterrent.

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In drive-stun mode, the principal action of the discharge is to induce pain (designed to gain the subject’s compliance). When the barbs are propelled, greater electrode separation facilitates the induction of involuntary (and painful) contraction of skeletal muscle mediated by stimulation of sensorimotor nerves.2

The medical consequences of these discharges include barb injuries, localised discharge burns, and injury from falls or from the intense muscle contraction.2 Eye and brain injuries from barb penetration have been documented.2 4 Tonic-clonic seizure after discharge of a conducted energy device to the head has been described.1 Pneumothorax after pleural barb penetration has been reported.6 Six fatal head injuries may have resulted from falls induced by these devices.7 Discharge of a conducted energy device does not induce clinically relevant changes in heart rate, blood pressure, or respiratory related parameters in healthy subjects.7

Whether the current from one of these devices can directly affect the heart is controversial. Atrial fibrillation in an apparently previously healthy child has been associated with exposure to discharge.9 Although induction of ventricular fibrillation is considered improbable,8 the safety margin for precipitating ectopic beats may be smaller because of a lower threshold for their initiation by an extrinsic electrical stimulus.9 Limited data in volunteers subjected to barb application of device discharge across the upper right to lower left anterior chest found no evidence for cardiac capture.10 However, no human studies have investigated discharge applied directly over the heart. In a review of outcomes after use of a Taser against 1 201 people in the US, two deaths were noted, neither of which was attributed to the device.11 This implies that the risk of induction of ventricular fibrillation, if one exists, is low.

The device is likely to be used on people who are physiologically and emotionally stressed, factors that may predispose to an increased risk in susceptible individuals (for example, those intoxicated with drugs or those who have cardiovascular disease).

The term excited delirium has been used to describe the bizarre behaviour, often related to the use of cocaine, exhibited by many of those who have died after use of a conducted energy device.1 However, excited delirium is not unique to the use of these devices and is associated with deaths after use of other forms of force by the police.12

The medical assessment of people subjected to discharge from a Taser must be guided, in part, by the range of potential outcomes described above. The assessment must include full past and current medical history, medication use, drug and psychiatric history. Electrocardiography may be indicated, particularly where intoxication with drugs, alcohol, or volatile substances exists or where subjects have cardiac disease or dysrhythmias. Pregnant women should be referred for obstetric review.

A history should be sought from police officers present when the device was deployed, so that the examination can be tailored to evaluate injuries at sites of barb penetration or contact with the stun electrodes, and at other sites where injury may have been sustained (such as the head, spine, or eyes). Acute shortness of breath associated with barb penetration of the chest should be evaluated by radiography.

Barbs may be removed by application of firm in-line traction, unless they are embedded in regions at risk of potential serious injury (such as eyes and the skull), in which case specialist intervention should be sought. Further investigation is unlikely to be needed if the subject is stable and has no illness, condition, or injury beyond the superficial lesions caused by the barbs or stun electrodes.

The UK government has established a standing committee of independent clinicians which provides evidence based information to advise ministers on the medical implications of less lethal weapons used by the police and the army. The committee has advised on technologies such as water cannons, impact rounds, and Taser, and their opinion feeds into user training, deployment policy, and post-incident medical management. Unless dealing with technologies that have sensitive national security implications, the committee’s advice is accessible publicly (for example, www.westmercia.police.uk/assets/_files/documents/sep_09/wmp__1252486507_ACPolicy_and_Operational_U.pdf).

The UK Scientific Advisory Committee on the Medical Implications of Less-Lethal Weapons (SACMILL) monitors operational use, medical reports, and media reports for emerging problems. When necessary, the committee will recommend changes to operational use or training to minimise the risk of adverse medical outcomes.

Reports in the medical literature of serious injuries associated with the deployment of Tasers are few, despite several hundred thousand estimated uses of the device.2 Lesser injuries may be under-reported. It is crucial, therefore, that governments and law enforcement organisations, assisted by healthcare professionals, establish mechanisms to improve understanding of the medical consequences surrounding the use of conducted energy devices such as Tasers and other less lethal technologies. The systematic capture of medically relevant data from operational incidents is a vital step in this process.
**Management of severe infections in rural Africa and Asia**

The winner of the Research Paper of the Year category in the 2010 BMJ Group Awards was a paper that described the use of pre-referral rectal artesunate (a rapidly effective antimalarial) in patients with severe malaria. This remarkable study conducted in Bangladesh, Ghana, and Tanzania randomised 17,826 patients with suspected malaria in rural areas to rectal artesunate or placebo before referral to a health facility. Although mortality did not differ between groups, the composite of death and disability was significantly reduced in people who received artesunate, in an analysis that was restricted to those with confirmed malaria. The effect was largely limited to participants who were delayed for more than six hours before arriving at a health facility, in whom the risk ratio was 0.49 (95% confidence interval 0.32 to 0.77). The findings suggest that in patients with severe malaria in whom treatment is likely to be delayed, pre-referral treatment with rectal artesunate could reduce the risk of mortality and disability.

The study was an outstanding logistical feat and clearly highlighted that treatment of severe malaria in remote areas can reduce morbidity and mortality. The paper has generated much debate since publication about its methods and implications for practice, and it has raised ethical concerns. It identified important differences between Asia and Africa, and between children and adults. Three challenging clinical questions arising from the study do not currently have clear answers.

Firstly, can the approach of giving rectal antimalarials in remote areas where malaria is endemic be operationalised? Is it currently unclear if giving rectal artesunate (or equivalent effective antimalarials) can be made to work outside a trial setting, but the findings of this study suggest that we should try. As with many interventions, deprived communities in rural areas some distance from formal health care have the most to gain but are also those most difficult to reach. It would be inappropriate to set up a parallel system only to deliver rectal antimalarial, which should be delivered through existing community based systems providing care. It cannot be assumed that improving care in the community will lead to reduced mortality at a population level, and this would have to be tested. The cost of delivering rectal antimalarials for pre-referral use must be weighed against the cost of improving speed of access to good quality health care.

Secondly, how can we enhance the rapid referral of severely sick children and adults to health facilities? The need for rapid referral extends beyond malaria. Delivering rectal artesunate is not a substitute for formal care, but a way to reduce the effect of delayed treatment in cases of severe malaria, and the sooner correct treatment is started the lower the risk of death. The study adds to the overwhelming evidence that delays in treating severe infections kill, and it highlights differences between Asia and Africa. Barriers to rapid referral include inadequate information and cultural beliefs about illness, as well as factors such as transport, distance, direct and indirect costs, relationships within households and between carers and professionals, and patients’ or carers’ impressions of quality of care at health facilities. The successful management of illness in the community is usually dependent on functioning health systems. Tackling any one barrier to referral in isolation will therefore have limited impact without changes to the health system as a whole.

Thirdly, what should we do for patients who have severe febrile disease but do not have malaria? How best to manage severe non-malarial illness is complex. In many areas of Africa and Asia where malaria is endemic, it is common to treat most severe febrile illnesses as if they were malaria. Malaria is, however, only one severe infection that may cause mortality. In some areas of Africa and Asia the proportion of severe illness attributable to malaria is decreasing, and the relative importance of non-malarial causes of illness is therefore increasing. Alternative causes vary by geographical area, age, and HIV prevalence, but bacterial infections make up a substantial proportion of treatable causes in many settings. One approach is to improve diagnosis; the World Health Organization now recommends parasitological testing before antimalarial treatment, and this is a major shift in case management. Rapid diagnostic testing to guide treatment is gaining momentum in remote areas, although introducing tests does not in itself immediately solve the problem of misdiagnosis because clinicians’ prescribing behaviour is complex and tests are only one element of this. An alternative response is to use syndromic treatment, which combines an antibiotic with an antimalarial in probable severe infection, but the impact of this approach has yet to be tested, and it is not clear which antibiotic is best.

The award winning study clearly shows that substantial delays in obtaining treatment have serious effects on seriously ill patients in Africa and Asia even when effective antimalarial drugs are available. The failure of healthcare systems to deliver effective treatment early contributes substantially to avoidable mortality and morbidity. Fixing this system failure will not be easy or quick, but it must be a priority.