EDITORIALS

Mental illness in deployed soldiers

Is more likely as traumatic exposures increase, and this is often related to length of deployment



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More than 29 armed conflicts involving 25 countries are now occurring around the globe.¹ For people in the United Kingdom and United States the situation in Iraq and Afghanistan is a constant reminder of the cost of war. The price that soldiers, sailors, airmen, marines, and their families pay is always considerable.

In this week's *BMJ*, Rona and colleagues assess the effect of the frequency and duration of deployment on the mental health of 5547 randomly chosen military personnel with experience in deployment.² They found that people who were deployed for more than 12 months in the past three years were more likely to have mental health problems (odds ratio for post-traumatic stress disorder 1.55, 95% confidence interval 1.07 to 2.32), although exposure to combat partly accounted for these associations. Post-traumatic stress disorder was more likely when a mismatch occurred between the expected and actual lengths of deployment.

The study could help identify those at high risk of long term disability and guide policy.³ Deployment is a strange term. Few people would suggest that deployment itself is a substantial cause of psychiatric disorder or distress. Many nations deploy soldiers around the globe. The US and UK have deployed soldiers for decades to overseas assignments, both with and without their families, and without substantially increased risk of post-traumatic stress disorder.⁴ However, it is the nature of the deployment experience—the "toxic" exposures—including traumatic events, loss of attachments, and psychological and physical demands that increase the risk of mental illness.

Another example of how the nature of the deployment affects the risk of mental illness is seen in US military veteran prisoners of war repatriated at the end of the Vietnam war. Duration of solitary confinement and weight loss were the most robust independent predictors of poor psychiatric outcome because they were strongly related to various "toxic" exposures.⁵ If the length of deployment corresponds with the amount of combat trauma and related experiences, it can be a strong predictor of the risk of mental illness. But this is not always the case, as deployments vary greatly in the frequency, intensity, and type of exposures encountered. In real time, wars change in days, weeks, and months, and so may the exposures that comprise a deployment. The length of deployment is just one measure of these factors-remembering this is important for healthcare planning as well as for protecting forces in war.

The incidence of mental illness is usually only measured after soldiers return from deployment, often well after the trauma. The challenge is to assess the risk of mental illness in real time. This would enable risk to be assessed, so that soldiers identified to be at high risk or those diagnosed with mental illness could be treated at the battlefront. We must, therefore, move towards measuring relevant exposures in real time. Exposure to traumatic events and loss of coping and social support must be assessed in real time by commanders to protect the health of their personnel. Decisions about how long soldiers should be deployed must take into account how stressful the combat is likely to be. In addition, decisions on length of deployment must consider the stress of rotation home and return (for example, the transition from "battle mind" to "home front mind" and back to "battle mind") and the ability of soldiers to sustain skills and mental and physical strength while home.

Perhaps most importantly, Rona and colleagues have shown that the Iraq war is not without its costsboth to the health of those deployed and eventually to the healthcare system-and that these same costs are related to duration of exposure. To date, the US army surgeon general has set up four mental health advisory teams to assess the mental health of deployed US soldiers via anonymous surveys. In 2006, the fourth team collected data from surveys and qualitative interviews from more than 1300 soldiers and nearly 450 marines.⁶ The report noted that the length of deployment and uncertainty about the date of return home were the top two concerns of soldiers. Morale among soldiers deployed several times was lower than that among those deployed for the first time. Similarly, soldiers deployed several times to Iraq were more likely to fulfil criteria for acute stress, post-traumatic stress disorder, depression, or any mental disorder than those who were deployed once. Soldiers deployed several times were 1.6 times more likely to screen positive for post-traumatic stress disorder than those who were deployed once, 1.2 times more likely to screen positive for anxiety, and 1.7 times more likely to have depression. Importantly, no specific cut off for duration of deployment eliminated risk. Soldiers deployed for longer than six months were also between 1.5 and 1.6 times more likely to screen positive for acute stress than those deployed for less than six months.

War develops as a result of seemingly unavoidable circumstances emerging within a specific social context. Similarly, the ability to adapt to normal life after war is shaped by the specific social circumstances and contexts of the conflict.⁷ Rona and colleagues' finding that unmet expectations for a shorter deployment are associated with post-traumatic stress disorder shows how our hopes and beliefs about the future, a part of our changing social context, affect health and disease.

For the practitioner and the health planner, soldiers with the longest deployments will be among those most likely to need care, both at the battlefront and after their return home. Length of deployment is but one measure, not the most direct, of the exposures and risks when they return home. Providing continuity of care across time and space is a challenge for providers and health systems.

- 1 Plowshares. Armed Conflicts Report 2007. Summary. 2007. www. ploughshares.ca/libraries/ACRText/Summary2006.pdf.
- 2 Rona RJ, Fear NT, Hull L, Greenberg N, Earnshaw M, Hotopf M, et al. Mental health consequences of overstretch in the UK armed forces: first phase of a cohort study. *BMJ* 2007 doi:10.1136/ bmj.39274.585752.BE
- 3 Committee on Veterans' Compensation for Posttraumatic Stress Disorder of Institute of Medicine. *PTSD compensation and military service*. Washington DC: National Academies Press, 2007.
- 4 Benedek D, Ursano RJ, Holloway HC. Military and disaster psychiatry. In: Saddock H, Kaplan BJ, eds. *Comprehensive textbook of psychiatry*. 8th ed. New York: Lippincott Williams and Williams, 2004: 2426-35.
- 5 Ursano R.J, Benedek DM. The prisoner of war: long-term health outcomes. Lancet 2003;362(suppl):s22-3.
- 6 Mental Health Advisory Team IV. Operation Iraqi freedom. 2007. Final report. www.armymedicine.army.mil/news/mhat/mhat_iv/mhativ.cfm.
- 7 Engel CC. Post-war syndromes: illustrating the impact of the social psyche on notions of risk, responsibility, reason and remedy. *J Am Acad Psychoanal Dyn Psychiatry* 2004:32:311-34.

The Wanless review Slow progress on public health may need more health spending

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This article was posted on bmj.com on 17 September 2007: http://bmj. com/cgi/doi/10.1136/ bmj.39338.501447.80 The review of National Health Service (NHS) funding and performance since 2002 published this week, which has been led by Derek Wanless, has something for everyone.¹² The government's supporters will focus on progress made in appointing extra staff, modernising buildings, buying new equipment, cutting waiting lists and waiting times, and improving priority areas of service provision such as cancer and cardiac care.

The government's critics will emphasise the failure to improve productivity and the high cost of the new contracts for general practitioners, consultants, and other staff. Independent observers will note that progress on reform of the NHS and on the wider public health agenda falls well short of the most optimistic "fully engaged" scenario set out in the original Wanless reports.^{3 4} The implication of this shortfall is that government may need to increase planned spending on the NHS to enable it to meet future demands.

While the review provides a comprehensive and even handed assessment of NHS reform, two factors need to be borne in mind in drawing conclusions. The first concerns the lack of reliable information to assess progress in some key areas.

Most importantly, incomplete data on the range and quality of services delivered for the increase in resources made available to the NHS make it hard to accurately assess changes in productivity since 2002. This is important because estimates of future resource requirements are particularly sensitive to improvements in productivity.

While work is in hand to fill gaps in data and to develop a measure of productivity that reflects quality of care and the full range of services provided,⁵ the extent to which extra spending has improved performance is uncertain, as the review emphasises. Arguments that further major increases in funding are needed should therefore be treated with caution.

The second factor relates to the scale of the challenges involved in the NHS reform programme. Transformational changes of this kind rarely proceed in a linear fashion, so that performance often deteriorates before it improves.

The review says nothing about this problem and offers a "before and after" assessment of progress, rather than a more nuanced account. Failure to analyse the rhythm and pace of change means that it is not clear whether reform is on a rising or declining trajectory. If the progress noted is accelerating, then the review's verdict on the state of the NHS today is more positive than it would appear.

Looking to the future, Wanless argues that the policy direction taken by the government is right, notwithstanding the disruptive effects of organisational change. In making this point, the review emphasises that the most notable improvements have been driven centrally through national service frameworks, guidance from the National Institute for Health and Clinical Excellence, and government targets.

It is all the more surprising therefore, that the report lends qualified support to patient choice, provider competition, and commissioning as drivers of change in the next stage of reform. These policies have been implemented too recently to have been evaluated properly, and it is not clear that they will be more effective than other approaches in bringing about change.

To take just one example, much hinges on general practitioners and primary care trusts becoming "world class commissioners,"⁶ yet evidence from other countries shows how difficult it is to commission health care effectively.⁷ On this matter, the authors' aversion to further lurches in policy direction has outweighed a more considered assessment of the evidence.

The review is on sounder ground in its criticisms of the policy making process. Specifically, it notes that the pressure to produce quick results has led to some policies and initiatives being introduced without adequate preparation. It also argues that the government has failed to take full account of how the various elements of its reforms fit together. There are strong echoes here of the Cabinet Office's capability review of the Department of Health and its criticisms of the quality of policy making in government.⁸

However, the review sounds the loudest warning bells on public health. Despite continuing progress in increasing life expectancy and reducing infant mortality, considerable threats for the future are identified in widening health inequalities and increasing rates of obesity.

The review reiterates the need for a comprehensive framework for public health and criticises the raiding of public health budgets to help tackle the financial deficits that emerged in the NHS in 2005. During a week in which the new secretary of state for health chose public health as the subject of his first major speech, it may be that the review's message will be heeded this time round.

If there is a surprise about the review, it lies less in its analysis and recommendations, and more in the muted reaction of politicians. At least for now, there seems to be broad political consensus on the future funding of the NHS and the policies that need to be put in place to deliver further improvement. The pessimistic view is that this reflects the poverty of thinking in the political class. A more generous interpretation is that it stems from the challenges involved in turning around a major organisation like the NHS and the realisation that no quick fixes are on offer. The opportunity this creates is for the NHS to build further momentum for improvement, relatively sheltered from the shifting winds of political debate.

- Wanless D, Appleby J, Harrison A, Patel D. Our future health secured? London: King's Fund, 2007. www.kingsfund.org.uk/publications/ kings_fund_publications/our_future.html.
- 2 Kmietowicz Z. Record investment in NHS fails to boost productivity. BMJ 2007;335:529. (15 September.)
- 3 Wanless D. Securing our future health: taking a long term view. Final report. London: HM Treasury, 2002. www.hm-treasury.gov. uk/media/A/F/letter_to_chex.pdf.
- 4 Wanless D. Securing good health for the whole population. Final report. London: HM Treasury, 2004. www.hm-treasury.gov.uk/ consultations_and_legislation/wanless/consult_wanless04_final. cfm.
- 5 Cabinet Office. Capability review of the Department of Health. 2007. www.civilservice.gov.uk/reform/capability_reviews/publications/ pdf/Capability_Review_DfH.pdf.
- 6 Mooney H. Commissioning's stakeholders design the world class future. *Health Serv*/ 2007;117:5.
- 7 Ham C. Commissioning in the English NHS: the case for integration. London: Nuffield Trust, 2007. http://nuffieldtrust.nvisage.uk.com/ uploadedFiles/Grants/commisioning%20series%20report%20draft %20(3).pdf.
- 8 Office for National Statistics. Public service productivity: health. *Economic Trends* 628. London: ONS, 2006.

Screening for familial hypercholesterolaemia

Insufficient evidence exists to support universal screening

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In this week's *BMJ*, Wald and colleagues propose a universal screening strategy for familial hypercholesterolaemia.¹ They suggest that serum cholesterol should be measured in children aged 1-9 years during routine visits to primary care, and that those with abnormal total cholesterol (greater than 95th centile) should have genetic tests or clinical investigations to confirm the diagnosis. A population cascade screening programme could then identify the parents of children who screen positive for the disorder.

This proposal is based on their meta-analysis of screening for familial hypercholesterolaemia. This study showed that measuring serum cholesterol in children age 1-9 can detect 88%, 94%, and 96% of cases, with false positive rates of 0.1%, 0.5%, and 1%, respectively.¹ Their proposal is based on the ability of the test to detect the disorder with a reasonably high detection rate and a relatively low false positive rate. The authors present no new evidence for the long term health benefits or potential harms of identifying and treating children with familial hypercholesterolaemia.

Much attention has been paid to screening for lipid disorders at a young age because half of children with high concentrations of total cholesterol and low density lipoprotein will continue to have raised lipids in adolescence and early adulthood, and early identification and treatment in certain populations of adults

can prevent coronary heart disease.² A screening programme could identify three groups of children with abnormal cholesterol concentrations-children with monogenic dyslipidaemias, such as familial hypercholesterolaemia; those with undiagnosed secondary causes of dyslipidaemia (diabetes, hypothyroidism, etc); and those with multifactorial dyslipidaemias (polygenetic or related to risk factors, such as obesity). The group most likely to benefit from screening, earlier identification, and treatment would be children with familial hypercholesterolaemias. In these children, treatment with statins and bile acid resins improves lipid profiles and intermediate outcomes.² In children with abnormal lipids but without familial hypercholesterolaemia (multifactorial dyslipidaemias), evidence shows that medical or behavioural interventions do not improve lipid levels.²

The US National Cholesterol Education Panel and American Academy of Pediatrics recommend screening in children with a positive family history of hypercholesterolaemia or those with risk factors.³ This has been problematic because of a high false negative rate in detecting high serum cholesterol, ranging from 17% to 90%, as a result of variable definitions of positive family history and differing thresholds of abnormal cholesterol. Taking a family history is also associated with a high false positive rate, with 25-55% of children and adolescents qualifying for serum cholesterol screening on the basis of family history alone.²

The screening strategy proposed by Wald and colleagues seeks to identify only those children with familial hypercholesterolaemia by requiring specific criteria for a clinical diagnosis: total or low density lipoprotein cholesterol above a given value, raised serum cholesterol in a first degree relative, and a family history of tendon xanthomata. As reported by Wald and colleagues, detection rates are relatively high, but even with a relatively low false positive rate, a universal screening programme without genetic confirmation will identify a large number of children who do not have the disorder. However, a programme that incorporates genetic confirmation of the diagnosis is likely to be expensive.

A third strategy, which is more appropriately called case finding rather than true screening, is cascade screening, where the family members of all patients with known familial hypercholesterolaemia undergo clinical diagnosis or genetic testing. This strategy is endorsed by the UK National Screening Committee and supported by the 2000 health technology assessment report, which concludes that such a case finding strategy in relatives of patients with familial hypercholesterolaemia followed by a clinical or genetic diagnosis would be most cost effective.⁴

Unfortunately, we have no direct evidence on the adverse effects of any of the above screening strategies. No data are available on the safety of long term treatment with drugs started in childhood or adolescence. Although lipid concentrations in children with familial hypercholesterolaemia can be improved with treatment,² we have no evidence of a long term benefit on health. If a benefit exists, the difference between this benefit and that associated with detecting and treating the disorder in adults would need to be examined, and there is currently no evidence to do this.

Finally, while the false positive rate may be low, those children who are found to have raised cholesterol but who do not have familial hypercholesterolaemia or are false positives may be treated unnecessarily. Treatment in children with non-familial hypercholesterolaemia has not been shown to improve health outcomes in children or adults,² and again the long term safety of lipid lowering agents in young children has not been determined.

On the basis of current evidence, the most cost effective approach to identifying people with familial hypercholesterolaemia is case finding in the family of those known to have the disorder. There is insufficient evidence to support universal screening with either serum cholesterol followed by clinical or genetic confirmation, or family history taking followed by serum testing.⁵

- Wald DS, Bestwick JP, Wald NJ. Child-parent screening for familial hypercholesterolaemia: screening strategy based on a metaanalysis. BMJ 2007 doi: 10.1136/bmj.39300.616076.55.
- 2 Haney E, Huffman L, Bougatsos C, Freeman M, Steiner R, Nelson H. Screening and treatment for lipid disorders in children and adolescents: systematic evidence review for the US Preventive Services Task Force. *Pediatrics* 2007;120:e180-214.
- 3 National Cholesterol Education Program. Highlights of the report of the expert panel on blood cholesterol levels in children and adolescents. *Pediatrics* 1992;89:495-501.
- 4 Marks D, Wonderling D, Thorogood M, Lambert H, Humphries SE, Neil HAW. Screening for hypercholesterolaemia versus case finding for familial hypercholesterolaemia: a systematic review and costeffectiveness analysis. *Health Technol Assess* 2000;4(29).
- 5 US Preventive Services Task Force. Screening for lipid disorders in children: US Preventive Services Task Force recommendation statement. *Pediatrics* 2007;120:e215-9.

Increasing exclusive breast feeding

Interventions are effective but must be tailored to the specific setting

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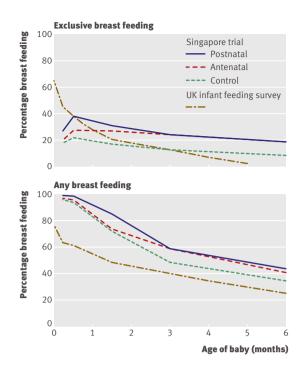
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In this week's *BMJ*, a randomised controlled trial by Su and colleagues compares the effect of two different strategies on the rate of exclusive breast feeding in 450 healthy pregnant women in a tertiary hospital in Singapore.1 The World Health Organization (WHO) recommends that, wherever possible, infants are exclusively breast fed for the first six months after birth²; during this period they should receive breast milk only, and no other liquids (except drugs) or solids. In developing countries, where the risk of infection is high and facilities for adequate sterilisation are scarce, breast feeding protects against infant mortality, particularly mortality related to infection.³ Rates of breast feeding are high in such countries, but rates of exclusive breast feeding are lower as a result of certain cultural practices, such as delaying the initiation of breast feeding and giving prelacteal feeds.4 However, starting breastfeeding on the first day after birth protects against neonatal mortality.⁴ Exclusive and predominant breast feeding compared

with partial breast feeding or no breast feeding protect against mortality in the first half of infancy.⁵

In more developed countries, where infection and inadequate sterilisation pose less of a problem, the health benefits of exclusive breast feeding persist. A cluster randomised trial of promoting breast feeding in Belarus resulted in significantly more exclusive breast feeding and significantly less diarrhoeal disease in the intervention clusters compared with the control clusters.⁶ In recent observational studies from Spain⁷ and the United Kingdom,⁸ exclusive breast feeding protected against hospital admission for infection in infancy. In the UK, rates of mothers starting breast feeding have increased from 62% in 1990 to 76% in 2005, but rates of sustained exclusive breast feeding remain low.⁹

The trial by Su and colleagues includes an antenatal and postnatal intervention to promote exclusive breast feeding and compares these interventions with routine hospital care.¹ The first group of women were shown a



Rates of breast feeding in the Singapore trial¹ and the UK Infant Feeding Survey 2005.⁹ The denominator is all women who breast fed in the UK trial and women who intended to breast feed in the Singapore trial (estimated at about 95%). The definition of exclusive breast feeding was stricter in the UK trial (exclusive since birth) than in the Singapore trial

16 minute video about breast feeding in the antenatal period. Women in the second group had two 30 minute sessions with a lactation consultant, one within the first three days after birth before discharge from hospital, and another during their first routine postnatal visit one to two weeks after delivery. Both strategies doubled the rate of exclusive breast feeding at most time points between two weeks and six months compared with usual care. About 64% of women in the trial had only primary education or less, 40% were primiparous, and 21% had a caesarean section.

Among the strengths of the trial are the evaluation of two separate hospital based interventions and a primary outcome of exclusive breast feeding. The initial increase in the rate of exclusive breast feeding in both groups suggests that exclusive breast feeding was defined as being "exclusive within a defined period" (usually the previous 24 hours). This definition will tend to overestimate the rate of exclusive breast feeding since birth, although such overestimation is unlikely to differ between the trial arms.

How generalisable are these findings to other settings? A systematic review of breastfeeding interventions reported conflicting evidence on the effectiveness of antenatal education and postnatal support for breast feeding.¹¹ Many breastfeeding support strategies are effective in particular settings only. When the breastfeeding practices observed in the control arm of the Singapore trial are compared with the 2005 UK infant feeding survey,⁹ some striking contrasts are apparent (figure). In Singapore, mixed feeding is common and, therefore, breastfeeding rates are relatively high, but rates of exclusive breast feeding are low. Here, the challenge will be to increase the duration and exclusivity of breast feeding, as was shown in the intervention arms. In the UK, breastfeeding rates are lower than in Singapore, but the rate of exclusive breast feeding is higher, at least in the first few months.

The postnatal intervention in the trial by Su and colleagues included a visit by a lactation consultant within the first three days after birth before discharge. It would be difficult to implement this intervention in the current UK setting, as many women are discharged within 24 hours of delivery. A recent UK study found that delivering in "baby friendly" accredited maternity units was not associated with a longer duration of breast feeding.¹² In contrast, in the Belarus trial, where the mean length of stay after birth was successful.

Further research should focus on evaluating the cost effectiveness of these hospital based interventions in Singapore and similar settings. In the UK, the National Institute for Health and Clinical Excellence is currently developing public health guidance on maternal and child nutrition (expected to be available at www.nice.org.uk in February 2008). The guidance will include recommendations aimed at promoting breast feeding, particularly in low income households. The next step will be to implement and evaluate the cost effectiveness of this guidance.

- Su L-L, Chong Y-S, Chan Y-H, Chan Y-S, Fok D, Tun K-T, et al. Antenatal education and postnatal support strategies for improving rates of exclusive breast feeding: randomised controlled trial. *BM*/ 2007 doi: 10.1136/bmj.39279.656343.55.
- 2 World Health Organization. Global strategy for infant and young child feeding. 2003. www.who.int/nutrition/publications/gs_infant_ feeding_text_eng.pdf.
- 3 WHO collaborative study team on the role of breastfeeding on the prevention of infant mortality. Effect of breastfeeding on infant and child mortality due to infectious diseases in less developed countries: a pooled analysis. *Lancet* 2000,355:451-5.
- 4 Edmond K, Zandoh C, Quigley MA, Amenga Etego S, Tawaih C, Owusu Agyei S, et al. Delayed breastfeeding initiation increases risk of neonatal mortality. *Pediatrics* 2006,117:e380-6.
- 5 Bahl R, Frost C, Kirkwood BR, Edmond K, Martines J, Bhandari N, et al. Infant feeding patterns and risks of death and hospitalization in the first half of infancy: multicentre cohort study. *Bull World Health Organ* 2005,83:418-26.
- 6 Kramer MS, Chalmers B, Hodnett ED, Sevkovskaya Z, Dzikovich I, Shapiro S, et al. Promotion of breastfeeding intervention trial (PROBIT): a randomised trial in the Republic of Belarus. JAMA 2001;285:413-20.
- 7 Paricio Talayero JM, Lizan-Garcia M, Otero Puime A, Benlloch Muncharaz MJ, Beseler Soto B, Sanchez-Palomares, et al. Full breastfeeding and hospitalization as a result of infections in the first year of life. *Pediatrics* 2006,118:92-9.
- 8 Quigley MA, Kelly YJ, Sacker A. Breastfeeding and hospitalization for diarrheal and respiratory infection in the UK millennium cohort study. *Pediatrics* 2007,119:e837-42.
- 9 Bolling K, Grant C, Hamlyn B, Thornton A. Infant feeding survey 2005. Information Centre, 2007. http://tinyurl.com/26l4ev.
- 10 Aarts C, Kylberg E, Hornell A, Hofvander Y, Gebre-Medhin M, Greiner T. How exclusive is exclusive breastfeeding? A comparison of data since birth with current status data. *Int J Epidemiol* 2000,29:1041-6.
- 11 Britton C, McCormick FM, Renfrew MJ, Wade A, King SE. Support for breastfeeding mothers. *Cochrane Database Syst Rev* 2007;(1): CD001141.
- 12 Bartington S, Griffiths LJ, Tate AR, Dezateux C; Millennium Cohort Study Child Health Group. Are breastfeeding rates higher among mothers delivering in baby friendly accredited maternity units in the UK? *Int J Epidemiol* 2006,35:1178-86.

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Chikungunya in Italy Globalisation is to blame, not climate change

An epidemic of chikungunya virus has recently occurred in Italy, involving more than 190 cases.¹ The concern is that climate change will bring mosquito borne tropical diseases to Northern Europe, but is this outbreak really the result of global warming? Although such an epidemic is new in Europe, it is probably caused by globalisation rather than climate change. Increased amounts of long distance tourism, travel, and trade mean that organisms that live in and on people or goods have more opportunity to be transported across continents.

Chikungunya is an epidemic disease with many similarities to dengue—it causes fever that lasts four to seven days, sometimes with a rash. It is often accompanied by intense arthralgia.² Most infections cause noticeable disease, but haemorrhagic symptoms and other life threatening manifestations are rare.

The virus can be carried by several species of mosquito, but the vector in Italy and in recent epidemics elsewhere is Aedes albopictus. Its common names are Asian tiger in English and zanzara tigre in Italian. Its biology is similar to that of its cousin, Ae aegypti. Both evolved to breed in natural containers such as tree holes and plant axils, but now they have adapted to life with humans. They are abundant in many modern tropical and subtropical cities, and they exploit many kinds of containers made by humans. Unlike other mosquitoes their eggs can withstand desiccation, which allows them to travel around the world in a variety of containers. The international ship borne trade in used tyres has played a major role in such spread because tyres make good breeding sites and hold water no matter which way up they are stored.

Decades ago, *Ae aegypti* travelled in this way from its ancestral home of coastal East Africa to every corner of the tropical world, becoming the main dengue vector in most of the world's tropical cities. Much later, its Asian cousin *Ae albopictus* began the same process of migration, radiating throughout the Western Pacific and the Indian Ocean Islands, reaching Brazil in 1986 and Nigeria in 1991. It first arrived in the southern United States in 1983 and is now present in 26 states. It arrived in Italy in 1990, and gradually spread to scattered foci all over the country.³ The arrival of the zanzara tigre tends to be noticed because it bites during the day.

Meanwhile, an unprecedented series of chikungunya epidemics has been spreading throughout the Indian Ocean.² These epidemics are often intense and in the past few years have involved millions of cases in Comoros, Madagascar, India, and the East African coast. Travellers have also been affected, and hundreds of imported cases have been reported from Europe (including Italy) and the US. In 2006, there were 133 imported cases in the United Kingdom and 774 in France. In the Italian outbreak, the index case reportedly travelled from India, and a recent analysis in Italy⁴ pointed to tourism as the main reason for travel in imported cases—11 of the 17 infected patients were tourists. The Italian climate has always been suitable for *Ae albopictus* to flourish. The winters in its home range of Japan and Korea are colder than Italian winters; in these conditions the adults die out and the species survives the winter in the egg form. In Italy the adult forms may be able to live through the winter.⁵ If so, this could have important epidemiological consequences, because it might allow the virus to survive the winter inside mosquitoes and to reappear in spring.

What could have been done to prevent the recent outbreak, and what can be done to prevent further outbreaks in future? It is hard to see how Italian scientists could have done more to alert local health authorities to the risks arising from the invasion of the vector,³ but perhaps more could have been done to prevent its establishment and spread.

The options for prevention are limited as no vaccine exists. Better surveillance is needed, if only to ensure that cases are given appropriate attention and care, but surveillance alone is unlikely to curb transmission. Human cases of chikungunya and dengue are viraemic and infective to mosquitoes early on in the disease course, so that prompt isolation of cases may not prevent onward transmission. In malaria, by contrast, humans are infectious to mosquitoes later in the course of the disease, and local cases in Europe are likely to be diagnosed and treated before the infection reaches this stage. That is probably one reason why the thousands of imported cases of malaria that enter Europe each year have not triggered local malaria epidemics, despite the presence of suitable vectors.

Control of transmission can probably be achieved only by measures directed against the vectors. We cannot stop people going to endemic areas, but education about the risks and methods of personal protection may help. Control of vector populations in Italy will certainly be more difficult and expensive now than it would have been in 1991 before the mosquito had spread over the whole country. The only effective long term approach is to suppress and eliminate the breeding sites, which is difficult to do thoroughly because there are many sites, which are usually small and scattered. Nevertheless, the longer we delay the harder it will be.

Finally, as well as focusing on vector control in Italy, European health authorities could consider whether European Union support for vector control efforts in areas where chikungunya is endemic might also benefit European citizens at home.

- Watson R. Chikungunya fever is transmitted locally in Europe for first time. BMJ 2007;335:532-3.
- 2 Pialoux G, Gauzere BA, Jaureguiberry S, Strobel M. Chikungunya, an
- epidemic arbovirosis. *Lancet Infect Dis* 2007;7:319-27.Romi R. Aedes albopictus in Italy: an underestimated health problem in
- Italian. Ann Ist Super Sanita 2001;37:241-7.
 Beltrame A, Angheben A, Bisoffi Z, Monteiro G, Marocco S, Calleri G, et al. Imported chikungunya infection, Italy letter. Emerg Infect Dis 2007
- www.cdc.gov/EID/content/13/8/1264.htm.
 Romi R, Severini F, Toma L. Cold acclimation and overwintering of female Aedes albopictus in Roma. *JAm Mosq Control Assoc* 2006;22:149-51.