Public Health England

West Yorkshire Health Protection Team Newsletter Feb 2015

TB: time for a strategy

Our TB control is failing. Bradford and Kirklees have some of the highest rates of tuberculosis (TB) in England, and this country as a whole has TB incidence rates four times that of the US. Now the Collaborative Tuberculosis Strategy for England sets out a stronger approach, and was launched regionally in Leeds on 24 Feb.

In 2013 there were 7290 TB cases reported in England, an overall incidence of 13.5 per 100,000 population, heavily concentrated in the big cities. Most occurs in people born in high incidence countries such as Pakistan, but these are not recent arrivals – they are settled migrants who have been here for years. So our services may have missed opportunities to prevent them. In Yorkshire & the Humber, 9% of our TB cases are children (twice the national average), and half are UK-born.

The strategy has ten aims:

1 Ensure access to services and early diagnosis
2 Provide universal access to high quality diagnostics
3 Improve treatment and care services
4 Ensure comprehensive contact tracing
5 Improve uptake of BCG vaccination
6 Reduce drug-resistant TB
7 Tackle TB in under-served populations
8 Implement new entrant latent TB screening
9 Strengthen surveillance and monitoring
10 Ensure an appropriate workforce for TB control

The principal step is to strengthen coordination by establishing formal TB control boards. TB control is fragmented at present. The care of individual TB cases and their contacts often crosses several local authority and NHS boundaries, and planning and commissioning is messy. Recently a local man with multi-resistant TB spent several months in an acute hospital isolation bed, simply because community care arrangements could not be made. Good practice in one locality is not replicated in the next-door borough, such as use of modern interferon-based screening tests. Areas that begrudge the extra cost of these go round in circles with slower methods that can't keep up with patient and contact movements.

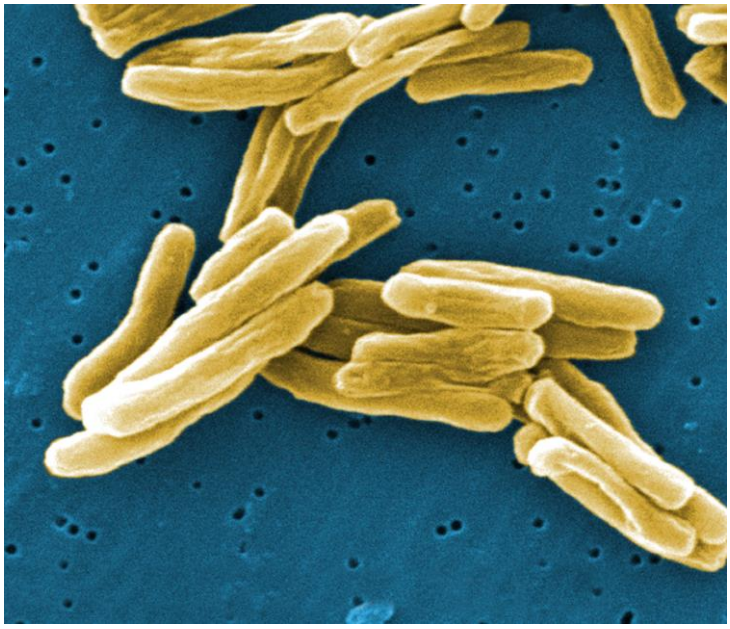
The new boards will cover the entire country not just the high-incidence areas – other areas have less TB but also lack specialist expertise and workforce. Boards will follow the same footprint as PHE Centres, thus there are nine in all with one for Yorkshire & the Humber.

Early tasks for the boards are to develop evidence-based model service specifications for TB services (both clinical and public health), to assess their local services against those specifications, and to develop plans to secure improvements.

Most of the interventions recommended by the strategy are likely to be cost-neutral, but three extra costs are foreseen:

- the TB control boards themselves need staffing
- testing for and treatment of latent TB in new entrants from high incidence countries, rather than waiting for clinical illness to present
- outreach services to under-served populations

Collaborative Tuberculosis Strategy for England 2015 to 2020



All that will add up to about £12.5M per year nationwide, but if the strategy is successful costs will fall from Year 5 and turn to net savings around Year 10.

There will also be stronger national support for local TB control arrangements. This includes an international dimension, such as quality-assured pre-entry screening for migrants. The former system of screening at UK airports has been disbanded, and migrants are required to prove they don't have TB as part of their visa application. There is also to be a national summit on TB and Hepatitis C on 3 March, chaired by the Minister for Public Health Jane Ellison.

The Director for each control board will be a crucial appointment; these posts will be advertised shortly.

WY wasted £191,284 of vaccines last year

In 2014 West Yorkshire GPs reported 77 incidents where the cold chain failed and vaccine had to be discarded. In 25 of these, the fridge wasn't working, causing wastage of over £105,000. Another £26,000 was wasted because fridges were turned off in error and another £17,500 lost by fridge doors being left open. Or fridges were slammed shut and the door bounced open again, or an obstruction prevented the door sealing. The total loss for England was £3.1 million. These figures don't include loss of Fluenz when stocks expired at the end of the year.

Cold chain incidents peak around September and October. This is the start of the flu campaign, so a likely factor is overstocked fridges. Much of the wastage could be avoided by:

- Annual service of the fridge
- Wire it direct into the mains, or failing that install covers to deter unplugging
- Ensuring sufficient fridge capacity
- Order in smaller amounts - Immform delivers weekly
- If you have a fridge problem, quarantine the vaccine but don't automatically discard it, take advice from the SI Team.

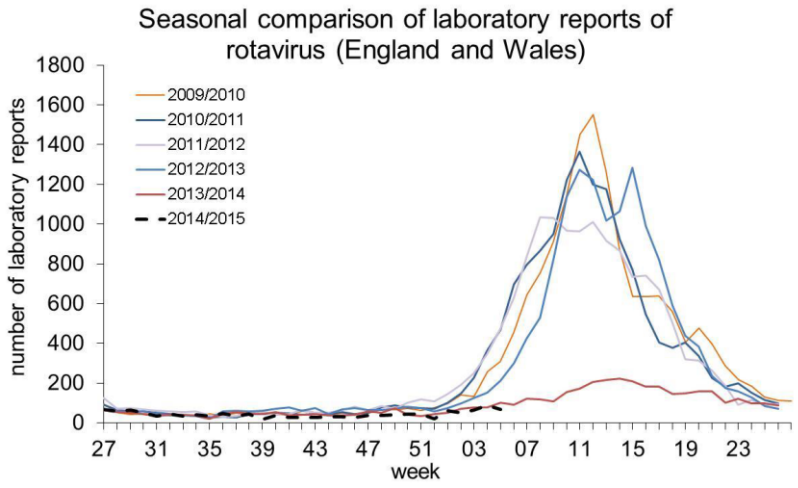
Flu

Flu in England was never high this winter and is now tailing off. It's mostly A(H3N2), and because this strain has drifted antigenically from last winter's version, our flu vaccine has been ineffective. Flu vaccine has an annual production cycle that's always playing catch-up, a year behind whatever the virus is doing. The other factor is that when there's not much flu, the unimmunised people don't catch it so the degree of protection from the vaccine looks poor. We'd much rather be in this situation than the opposite, with a highly protective vaccine but rip-roaring flu across the land. It's not a reason to become nihilistic about flu immunisation.

In China there's been a flurry of A(H7N9), but infection is bird-to-human, not human-to-human. Many patients had had contact with live poultry, and the Chinese poultry markets were busy in the run-up to Lunar New Year on Thurs 19 Feb (ending the Year of the Horse and bringing in the Year of the Sheep). Two cases recently occurred in Canada in people returning from China, but the great majority of the 486 cases seen in the last two years were in mainland China. The chief difference from A(H5N1) is that A(H7N9) seldom makes the birds ill, so they can be shedding the virus without anyone realising. PHE has confirmed that the risk to UK residents remains very low, but travellers to China should avoid live poultry and markets.

Rota prevented

Another spring, another rotavirus season we've prevented. Until 2013 this caused half of all gastroenteritis of under-5s, with some 130,000 GP consultations, 12,700 hospital admissions and 3 or 4 deaths each year in Britain. Infant immunisation began in May 2013, with good uptake, and in winter 2013/14 there wasn't a rota season; it's similarly flat-lined this winter.



We don't yet have definitive coverage figures for the vaccine, Rotarix by GlaxoSmithKline. But since it's oral, easily given along with the other routine immunisations at two months and three months age, coverage is believed to be over 95%. Indeed it is almost too easy, as when children come late to clinic there's a risk of the nurse going on auto-pilot and trying to catch up every vaccine. A first dose of Rotarix shouldn't be given from 15 weeks or a second from 24 weeks, because of the hypothetical risk of intussusception. But there have been no reports of intussusception associated with this programme, nor of other safety concerns.

Someone please invent something similar for norovirus, which continues its woeful ways.

Knee trouble?

knee trouble . . . ; it threw all the people into a fever. . . . Its first attack lasted for three days, after which the illness increased or diminished, according to the disposition of the individual. It was accompanied by pain in the joints, knees, and extremities, as well as inability to move, and often with swelling of the fingers.

This account of a Cairo outbreak of 1779 was long thought to describe Dengue, but more likely it was Chikungunya. This disease is spreading globally and recently has become established in the Caribbean. The virus is spread by *Aedes* mosquitoes, which feed by day. This mosquito vector is common in the Southern US so the virus could easily become prevalent there.

Source: "Chikungunya and Dengue: a case of mistaken identity?" DE Carey, J Hist Med July 1971: 243-262

Not just VTEC

As if we didn't have enough on our plate with VTEC – verotoxic E Coli, usually in the form of E O157, which causes bloody diarrhoea and haemolytic-uraemic syndrome. But this is just one of five or six families of pathogenic E Coli. The others aren't usually tested for so their symptoms will often be passed off as "diarrhoea, cause unknown", and we have limited information on how much is going around.

In January twelve people ate out in Leeds and seven of them developed gastroenteritis, as did another couple who ate at the same restaurant next evening. Standard investigations were negative and the place appeared hygienic, so we might have left it there. But someone had the inspired hunch that this could be one of the other E Coli families. Further testing was undertaken which revealed that 3 of those ill had ETEC; a food-handler there also had a mixed bag of ETEC, EAEC and EPEC. What is this miserable alphabet soup?

Three of the pathogenic families of E Coli infect animals as well as humans, so they can be acquired via the food chain. They are:

ETEC (enterotoxigenic): common in developing countries as a cause of diarrhoea in children and in travellers;

EPEC (enteropathogenic) causes shigella-like illness

and EHEC (enterohaemorrhagic) which includes the familiar VTEC

Two others are only seen in humans, so they would only be expected to spread case-to-case:
EIEC (enteroinvasive) – again, shigella-like
and EAEC (enteroaggregative).

The sixth family is DAEC – diffuse adherence E Coli – and is the least studied.

The letter "O" (not number 0) in designations such as E O157 refers to the O-antigen which is the outermost part of the bacterial membrane. E O157 happens to belong to the EHEC / VTEC group but the O-classification covers all gastrointestinal E Coli. These bacteria are also a common cause of urinary tract infections, but latch on in a different way and with a different classification. The taxonomy of E Coli is messy: it pre-dates phylogenetics and is ripe for overhaul. For instance the Shigellas arguably should be classed as E Colis.

Many E Coli are harmless, but because they are abundant in faeces and simple to test for, they are a standard marker for whether a food or water source has been contaminated.

Scarlet Fever

2014 saw an upsurge in scarlet fever. Now PHE is warning that we may be on the brink of a similar upsurge in 2015. Already Leeds and Kirklees are showing an increase in cases.

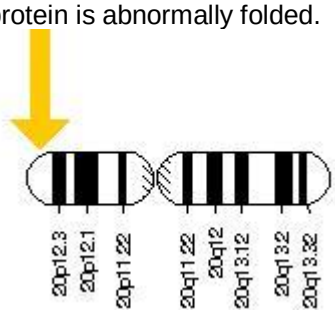
The scarlet fever season usually runs from about Christmas to Easter, with an epidemic every 4 years or so, the previous one being 2008. Subsequent years were quiet, with the whole of England seeing fewer than 200 notifications and 50 lab confirmations per week even at the height of the season. The next epidemic came in 2014, peaking in early March at over 1000 notifications per week and running on into mid-summer. We might now hope for things to go quiet again, but already 2015 figures for February exceed those for 2014.

Most parts of England are reporting double their 2014 rates, and three times higher in Cumbria and Lancs. Within our own region most cases have been in East Riding, Hambleton, Harrogate, Kirklees, Leeds and North Lincolnshire. 90% of them are children under 10, with an average age of four. Cases have shown the typical picture of sore throat, malaise, fever (often high), headache, nausea and vomiting. After 12 to 48 hours a fine red "sandpaper" rash spreads out from the chest and stomach. A white coating on the tongue peels after a few days, leaving a red swollen "strawberry" tongue. There are no reports of resistance to standard antibiotics such as Penicillin V.

One concern with the 2014 epidemic was that these non-invasive forms of Group A streptococcal infection might be the precursor to invasive forms: iGAS eg by septicaemia or necrotising soft tissue infection. That didn't happen last year, and so far hasn't manifested this year. Let's hope.

Where is the abnormal prion hiding?

One answer is, on the short (p) arm of chromosome 20 at locus 13. That's the gene PRNP which encodes prion protein, which does something in the brain that we haven't figured out yet. Some mutations in PRNP give rise to familial forms of CJD. However in variant CJD the gene is unchanged, yet the protein is abnormally folded.



The 129th amino acid in the protein may be either Methionine or Valine – and as we have paired genes, we may carry Met-Met, Met-Val or Val-Val. Cases of vCJD to date were almost always Met-Met, so will people with the other patterns develop vCJD in years to come? One in 2000 of us contain the abnormal prion through eating beef laden with BSE.

Our furthest neighbour

Perhaps its shining hour for public health was the Declaration of Alma Ata in 1978, with an ambitious vision of "Health for all by the Year 2000". What was then the Kazakh SSR is now the independent republic of Kazakhstan, but because of that history it remains part of WHO's European Region for health. Indeed, part of it lies west of the Ural River and is thereby in Europe. It's also in UEFA for football tournaments, but it's not in the European Broadcasting Union so we've been spared the Kazakh entry for the Eurovision Song Contest. How healthy is it?

A land of contrasts, as they say. The steppes of Kazakhstan reach all the way from the Caspian to the border with China, wheat-growing and very thinly populated. The cities, based on mineral industries, grew up in Soviet times: "Alma Ata" is nowadays called Almaty, and Astana is the capital.

Latest statistics from WHO are based on 2013:

	Kaz	UK
Population (millions)	16.4	63.1
% under 15	25.8%	17.5%
% over 60	10.2%	23.2%
Gross national income:		
$ per capita	20,570	35,760
Health expenditure		
$ per capita	608	3495
% of GDP	4.2%	9.4%
Life expectancy at birth	63m, 72f	79m, 83f
Fertility rate	2.5	1.9
TB incidence	139	13
Health workers per 10,000 population		
doctors	41	27
dentists	3.8	5.3
nurses & midwives	83	101

See www.who.int/countries for definitions and small print. For instance, only half of that limited Kazakh health expenditure came from public funds. Other payments made up the rest, so access to care was highly uneven.

Sharps: 4830 exposures, 9 seroconverted

From 2004 to 2013 there were 4830 sharps injuries and similar exposures of health workers to blood-borne viruses, according to PHE's report "Eye of the Needle". None of them acquired Hepatitis B or HIV. Nine seroconverted to Hepatitis C but seven of these were cleared by treatment.

What else happened in 1815?

It's hard luck on the events, personages and achievements of 1815 that the centenary memorial industry neglects them, because they're overshadowed by 1915 and trench warfare on the continent. There was little notice during the Great War of the centenary of Waterloo: Napoleon escaped from exile in Elba at the end of February 1815; by summer he was again defeated and returning to exile on St Helena. Louis XVIII returned to the French throne, and Marshal Ney was shot for changing sides once too often.

Franz Mesmer the founder of "animal magnetism" died in 1815, largely discredited. So too did Emma Hamilton, Nelson's mistress, in poverty and debt.

In April the massive eruption of Tambora in the East Indies caused global cooling and blighted the summer and the crops of 1816. In October a meteorite landed at Chassigny in Haute-Marne which is now believed to have come from Mars.

Sir Humphrey Davy brought out his Davy lamp for miners, and Levi Spear Parmly of New Orleans invented dental floss. The UK's Apothecaries Act 1815 required apprenticeship and qualifications for apothecaries, but the practice of medicine remained basically unregulated until the Medical Act of 1858.

Otto von Bismarck and Anthony Trollope were born in 1815. Also that year Anne Isabella Milbanke married Byron, and gave birth to Ada Lovelace, now regarded as the world's first computer programmer. Anne encouraged Ada's mathematics, seeing it as an antidote to inheriting Byron's wilder tendencies. But Ada attempted to use computing as a basis for gambling, and this went horribly wrong.

A synergistic birth in 1815 in Lincoln was George Boole, founder of logical algebra. True and false can be multiplied or added just like one and zero, and if they can be represented by mechanical settings or electrical flows, you've created a reasoning computer. "And", "or", "not" and "if" are the grammar of algorithms in natural language, eg in the clinical guidelines used by the WY HP Team. For instance our definition of a "probable" case of pneumococcal meningitis runs so:

Clinical syndrome of meningitis where pneumococcal infection is the most likely diagnosis, **or**
Lab-import of pneumococcus detected in CSF implies that syndrome.

Alas we were too late to help poor George. As Professor of Mathematics in Cork, in 1864 (aged 49) he got soaked in the rain but gave his lecture without changing. He developed a fever which his wife, lacking an evidence-based algorithm, treated by pouring more cold water over him. And so he died of pneumonia-cum-pleurisy.

This issue has been compiled with input from Ebere Okereke, Mary Law, Louise Coole, Judith Rushby and Graham Sutton. Our next issue is due out mid-May.