VACCINATABLE DISEASES AND THEIR VACCINES

Dr Jayne L.M. Donegan MBBS DRCOG DFFP DCH MRCGP MFHom

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

CONTENTS

© Dr Jayne L.M. Donegan MBBS DRCOG DFFP DCH MRCGP MFHom
Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

INTRODUCTION

In the absence of any clear, open, objective and well designed studies on vaccination safety, the observations and recommendations in this report have been made by carefully sifting through what studies *are* published in refereed medical journals and other sources; in particular looking at the methods and the results of studies, rather than the conclusions which often do not reflect their findings. Analysing the data in this way raises serious questions about the safety and efficacy of vaccination. It is notable, however, that the authors of the papers from which I have quoted, almost without exception, conclude by urging vaccination or repeated doses of vaccinations.

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

DIPTHERIA

Diphtheria is a disease produced by the bacterium Corynebacterium diphtheriae. It is spread by droplets (coughs and sneezes) or infected bedding/clothes. The bacterium may be detected in people who have no symptoms but are 'carriers' and can pass it on to susceptible people. In people who become ill with the disease, as opposed to carriers, the severity depends upon the site of the primary lesion – those on the pharynx being more severe than those on the larynx, and least severe if on the nasal mucosa or skin. Although not always present, characteristically a grey membrane forms over the affected area made up of bacteria, dead skin and inflammatory products. It is firmly stuck down until it sloughs off three to four days later in mild cases or a week later in moderately severe cases. The neck glands become very swollen.

The bacterium may also produce an 'exotoxin' which can affect the heart, kidneys and nerves. Most deaths from diphtheria are due to obstruction of the airway by the membrane and the effects of the toxin on the heart or nervous system. Not all strains of the bacterium produce toxin.

Susceptibility to the complications of diphtheria generally depend upon the levels of antitoxin in the blood. However, second attacks of diphtheria are rare despite the fact that at least ten percent of those who have had the disease do not have measurable levels of antitoxin – so antibodies to the exotoxin (antitoxin) are not the only reason for immunity. Early treatment of diphtheria with antibiotics tends to render people susceptible to further attacks when the antibiotics are stopped (*1*).

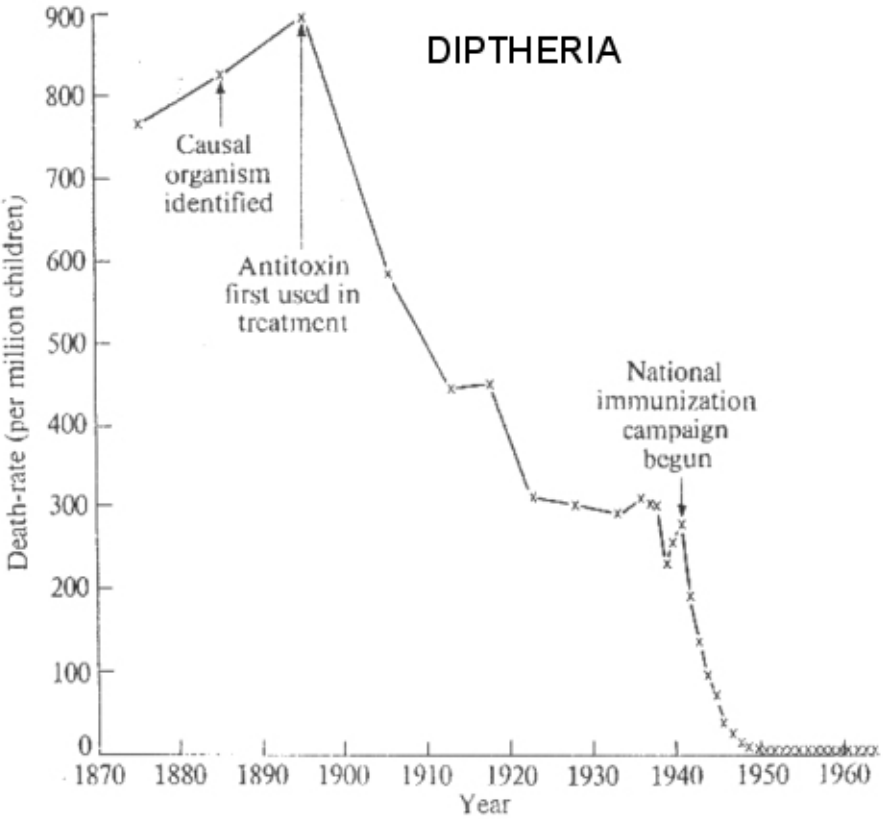


FIGURE 8.8. Diphtheria: death rates of children under 15: England and Wales. (2)

Diphtheria increased in prevalence and malignancy in the middle of the nineteenth century and declined before the introduction of the antitoxin. Antitoxin became available in the 1890s and reduced the case fatality rate so that mortality from diphtheria began to fall from that point, in a similar fashion to whooping

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

cough and measles. By the 1940s when a national immunisation campaign began, the death rate in children had dropped by two thirds and continued to drop (*2*).

In the USA, of the few cases reported, throat involvement is decreasing and symptomatic skin lesion make up a higher percentage, especially amongst indigenous peoples such as Native Americans and Pacific Islanders, where males living in crowded conditions with poor personal and community hygiene have the highest attack rate. Most cases are in adults, as in the former Soviet Union where most of the cases are in vaccinated adults, not unvaccinated children.

The fact there are so few cases of diphtheria reported in this country is more likely to be due to a trend towards decreased virulence of the organism and better resistance of the host – humans – because other diseases that have been vaccinated against have not disappeared in such a satisfactory fashion despite very high vaccination rates eg whooping cough, measles and mumps.

The Vaccine

Adsorbed Diphtheria vaccine for Adults contains Aluminium phosphate, Thiomersal (a mercury containing compound) and Formaldehyde as well as Diptheria Toxoid. A low dose formulation is recommended in children aged ten years and older because of the severity of the reaction to the regular dose in this age group compared with the reaction in babies.

Diphtheria is usually given with whooping cough and tetanus vaccine (which is supposed to make it produce more antibodies) so it is hard to separate out the side effects of the individual vaccine.

Listed side effects for the single, low dose, adult diphtheria vaccine *(Adsorbed Diphtheria Vaccine for Adults, Secretary of State for Health, Department of Health rev 1999)* are local pain at the injection site, redness and swelling. It also mentions that the thiomersal in the vaccine can cause kidney damage. Adults who are given the high dose children's vaccine can suffer severe systemic reactions. Long term effects are not known.

Vaccination Recommendation

The likelihood of contracting diphtheria in the United Kingdom is so low that I do not think any benefit is to be gained by vaccinating against it and any detrimental effects are, therefore, unacceptable.

References

(1) Harrison's Principles of Internal Medicine 11th Ed, McGraw-Hill Inc 1987
(2) The Role of Medicine, Thomas McKeown, Princeton University Press 1979:99 (Thomas McKeown was Professor of Social Medicine Emeritus at the Birmingham University and past chairman of the WHO advisory group on health research strategies)

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

PERTUSSIS (WHOOPING COUGH)

Whooping cough is a childhood illness caused by infection with the bacterium *Bordetella pertussis.* It is spread by droplets in coughs and sneezes. There is normally an incubation period of two weeks (when one is infectious without symptoms), a 'catarrhal' phase of two weeks, a paroxysmal or 'whooping' phase of two weeks and a recovery phase of two weeks. These may all vary in length and the more dangerous whooping phase may be absent altogether.

In the catarrhal phase there is mild fever, a runny nose and the start of a hacking cough that may keep the child awake at night. In the paroxysmal phase the cough comes. There may be repeated coughing without drawing breath while mucus and saliva stream from the nose and mouth. The child may vomit their last meal while coughing. Young children and babies may become cyanosed (blue) with bloodshot eyes. When the coughing has ended there is a long 'whoop' as the child breathes in. After a series of such episodes they may fall asleep, exhausted.

Looking after someone during this phase is particularly tiring and time consuming. They must be kept calm and quiet as excitement and exertion provoke the coughing attacks. During a spasm of coughing the child should be held in the recovery position to avoid the inhalation of vomit. Some small babies may require suction and oxygen after a spasm has ended. It is important to make sure that they get enough to drink – the best time to offer fluids is after an attack as it is less likely to be vomited.

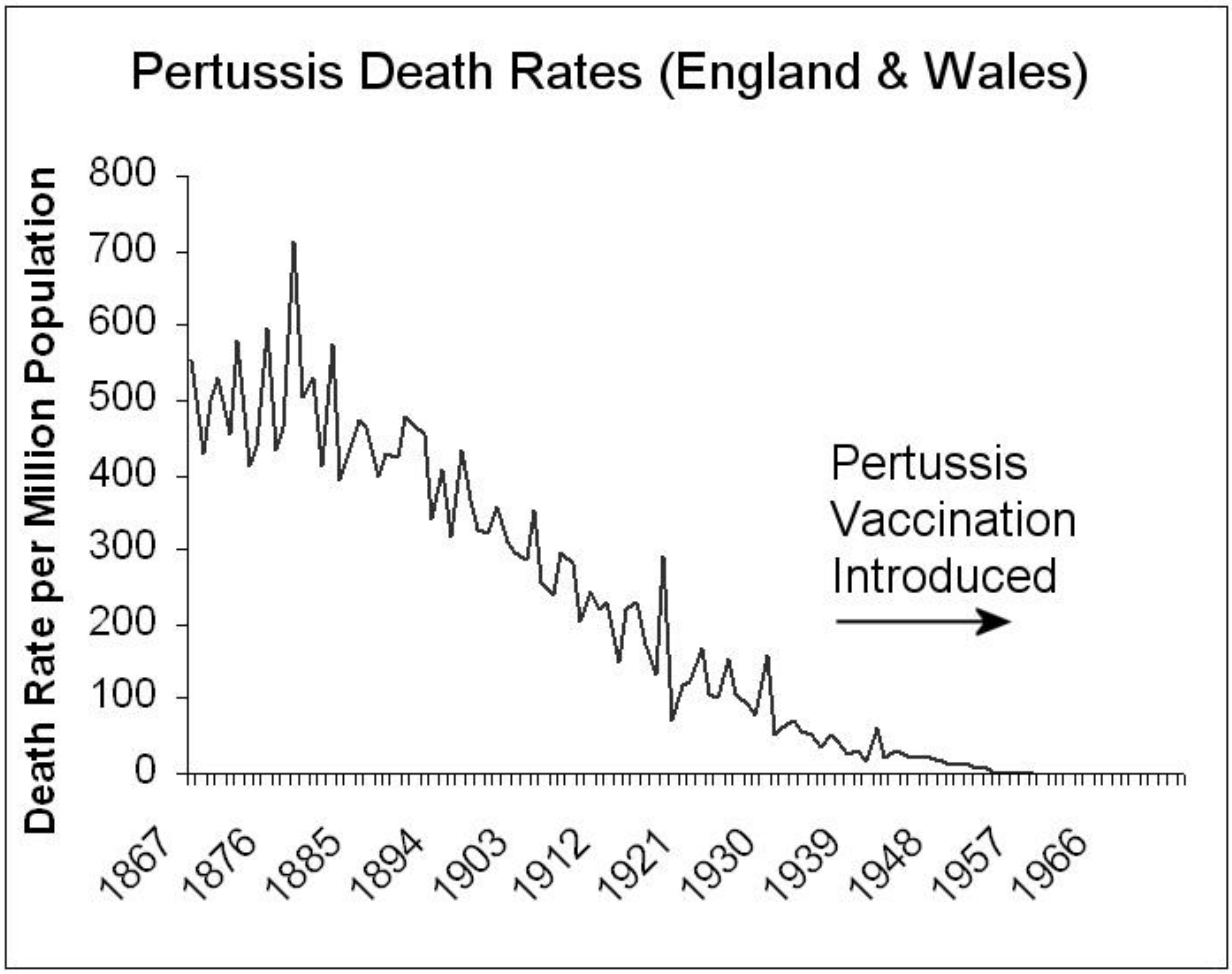
During the last fortnight the symptoms usually start to resolve. The whoops and the vomiting become less frequent so the child sleeps more at night and starts to regain weight. After recovery, a cough or cold during the following year may start off a series of whoops as will exposure to cigarette smoke. Although it is difficult to diagnose whooping cough in the first week because it is so like an ordinary cough or cold, the standard advice is that antibiotics given at this time will reduce the severity and duration of the illness. Giving them to siblings who have no symptoms is said to reduce spread to others.

Babies less than one year old usually have the most severe forms of the disease and it is in this age group that complications and death most often occur. There may be convulsions at the end of the coughing spasm and in rare cases these may cause a brain haemorrhage (bleed) which may produce temporary or permanent brain damage. Areas of the lung may collapse leading to bronchiectasis (dilated bronchioles filled with mucus) if re-expansion does not occur. Another complication of the disease is pneumonia, again, more common in babies and a major cause of death.

In the nineteenth century whooping cough was most definitely a killer disease. "Deaths from whooping cough remained at around 10 000 a year from 1847 until the 1900's and then **declined steeply as the health and care of children improved** and had reached less than 400 a year by 1950. Immunisation started in the 1950s, deaths continued to fall and notifications fell sharply." *(1)*

It is undoubtedly the case that whooping cough became a milder disease in this country over the course of the first half of the twentieth century. The death rate had fallen by over 99% before vaccination against pertussis was introduced in the 1950s *(fig1).* The introduction of the vaccine reduced the number of notified cases of whooping cough but peaks continued to occur every three to four years as they always had. Deaths continued their steady decline. This was most clearly seen in the 1970s and 80s when the vaccine coverage fell to less than 40% in 1976 because of health scares. In 1978 and 1982 there were over 65 000 notified cases of whooping cough but no concomitant rise in the number of deaths *(fig2)*. Between 30% and 70% of children in outbreaks are vaccinated *(2,3,4)*.

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk



(24)

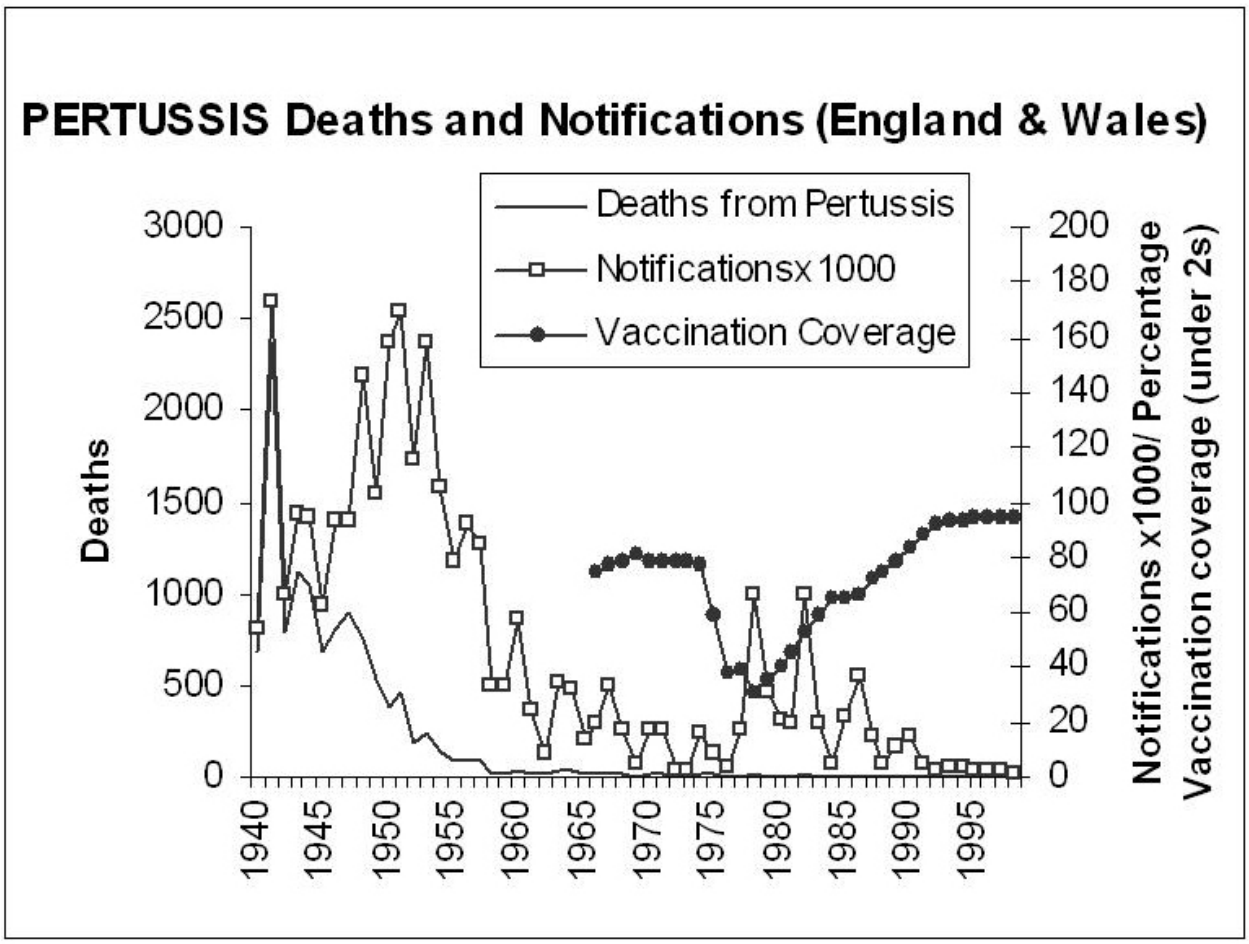
Is the pertussis vaccine useful in preventing pertussis disease? During infection with *Bordetella pertussis*, the inhaled organism sticks to the hairs lining the airways. It is then able to multiply and cause the inflammation, mucus, pus and ulceration that may block the narrow airways of young children and babies. During natural infection with pertussis IgG, IgM and IgA antibodies are produced. These IgA secretory antibodies are very important as they specifically stop the bacterium from sticking to the hairs and multiplying. Vaccination against pertussis does not produce this IgA antibody which is so important in protecting against further infection *(18)*.

The incidence of pertussis death and disease was falling well before the vaccine was introduced in the 1950s. In 1978 when United States passed laws requiring proof of vaccination before school entry to increase vaccination uptake there was a recognisable increase in the incidence of whooping cough in that country. This has continued to the extent that there are now five doses of pertussis vaccine in the US immunisation program *(19)*. In 1996 a study in California showed that 12% of adults with persistent cough had undiagnosed whooping cough *(20)*.

Because of continuing increases in pertussis notification in the UK, especially in young babies, an 'accelerated' schedule of vaccination was introduced (vaccination at 2m, 3m, 4m instead of the previous 3m, 5m,10m) to try to reduce the incidence of the disease.

Despite vaccination rates of 94% in under twos the incidence of pertussis has been increasing since 1995. Between 1995 and 1997, 10 of the 12 deaths from whooping cough were in babies under 2 months of age. As with a number of recent reports from the UK, USA and Australia, there seems to be a trend towards increasing numbers of deaths in very young children and a 'waning' of vaccine effectiveness in 1-4 year olds *(21)*.

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk



The increased incidence and deaths in young babies may also be due to poorer quality of placental antibodies being transferred from mothers who have themselves been vaccinated against whooping cough and which are not sufficient to protect their babies. One to four year olds appear to be contracting vaccine modified disease in increasing numbers. Vaccinated Adults are getting chronic coughs caused by pertussis despite having been vaccinated in childhood. The high prevalence of vaccination is also associated with a drift towards a higher incidence of disease caused by the 1,2 serotype which is more likely to be associated with complications and admission to hospital *(21)*. Despite this apparent failure of infant vaccination to protect the most vulnerable from the disease, the response to these problems is to add another pertussis vaccination to the programme and since October 2001 children have had pertussis added to their pre-school boosters.

The Vaccine

Pertussis vaccine may be whole cell or acellular. Whole cell is only available combined with other vaccines – diphtheria, tetanus +/- Hib and contains aluminium hydroxide and thiomersal (mercury containing compound).
Acellullar is available alone as a four component vaccine (*APV unlicensed in UK and only availabe for supply direct to the NHS childhood immunisation programme)* and contains aluminium hydroxide, aluminium phosphate and thiomersal. A combined product, Infanrix-Hib (*SmithKline and Beecham)*, which contains diphtheria, tetanus, three component acellular pertussis and Hib also has aluminium salts but not thiomersal.

Does the vaccine cause brain damage? A paper published in 1974 described neurological complications of pertussis vaccination *(5)*. This caused widespread panic among parents and some health

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

professionals. In order to investigate the matter, the National Childhood Encephalopathy Study was set up which looked at all 'serious neurological events' occurring in children aged two to thirty five months between 1976-79 and matched them with 'controls' who had not had such an event *(6)*. The study did not look at the number of children in the 'event' or 'control' group who had been vaccinated against pertussis compared with those who had not, but only at the numbers who had been vaccinated against pertussis **in the seven days before the neurological event**. This means that a child could have had a serious neurological reaction two or three weeks after pertussis vaccination and this would not have been included in the 'pertussis vaccine' figures. However, even with this timeframe, it was shown that those with severe neurological damage were 2.5 times more likely to have been vaccinated against pertussis in the seven preceding days than the 'controls'. The numbers were small but significant. A follow up study ten years later showed that those children who had since died or had neurological dysfunction were four times more likely to have been vaccinated against pertussis in the seven days preceding their original illness. As some of the neurological dysfunction was considered to be quite mild, a reanalysis was carried out which included only those children with more severe dysfunction and death. These children were 7.3 times more likely to have been vaccinated in those seven days *(7)*.
Fine and Chen pointed out that those being vaccinated against pertussis should be **less** likely to succumb to a neurological illness because those with fever, previous reaction to pertussis vaccination, family or personal history of epilepsy or pre-existing neurological impairment were generally advised not to be vaccinated. Taking this into account should make the association with pertussis vaccination stronger, which indeed it does *(8)*.

A similar case-control study in the United States found an association between pertussis vaccination and neurological damage *(9)*. The Institute of Medicine in America published the results of its study into the adverse consequences of pertussis and rubella vaccination in 1991 *(10)*. It found evidence consistent with a **causal** relation between DPT(Diphtheria, Pertussis, Tetanus) vaccination and acute encephalopathy, shock and 'an unusual shock-like state'. It found no evidence to accept **or reject** a causal relation between DPT vaccination and chronic neurological damage, Guillain-Barre syndrome, learning disabilities, attention-deficit disorder and peripheral neuropathy.
All these studies and reviews of them say that the risks of the vaccine are small and where the evidence is not regarded as sufficient to either accept or reject a causal association this is taken to mean that the vaccine is safe and that parents should be encouraged to carry on vaccinating their children *(11)*.

In 1994 Dr Michel Odent published a retrospective study which compared the incidence of asthma in 243 children who had been vaccinated against pertussis with 203 who had not. Vaccinated children were over five times more likely to suffer from asthma and twice as likely to have had ear infection than unvaccinated ones *(12)*. In 1997 another retrospective study of 1934 patients born between 1975 and 1984 from one general practice in Oxfordshire showed that children vaccinated against pertussis were 75% more likely to develop asthma, hay fever and eczema later in life *(13)*. A larger prospective (looking forward) study of 9444 children in Avon failed to show an association*(14)*.

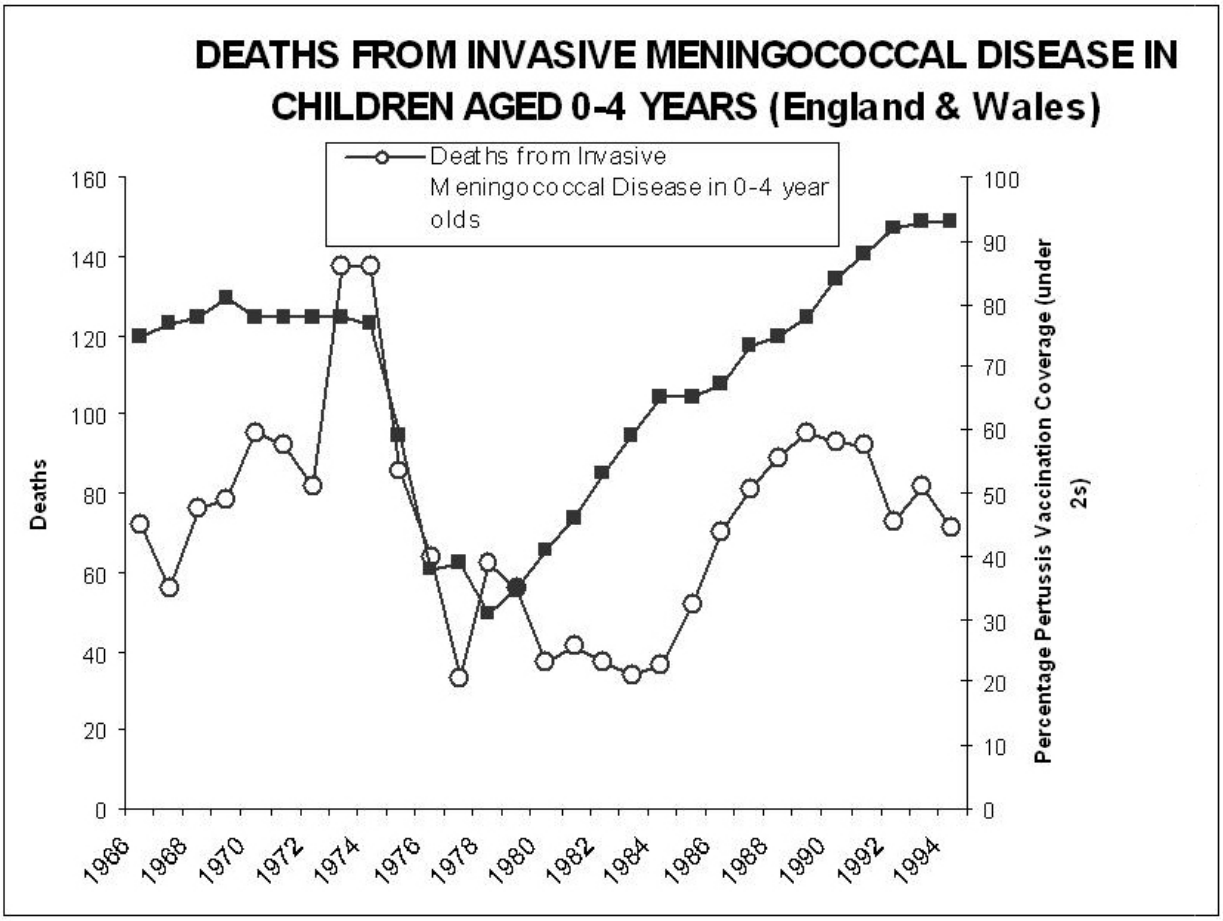
Questions have also been asked about the incidence of invasive bacterial infection in children who have recently been vaccinated against pertussis *(15)*. A 'natural experiment' took place in this country when the acceptance the vaccine fell dramatically in the mid 1970s to the mid 1980s and there was an accompanying fall in the number of deaths of children aged four years and less from invasive meningococcal disease. The numbers began to rise again as vaccine uptake increased *(fig3)*.

The Swedes abandoned the whole cell pertussis vaccine in 1979 because of worry about side effects and because of its perceived ineffectiveness as whooping cough swept through its population of whom the majority was fully vaccinated. The Japanese raised the vaccination age to two years in 1975 after a number of reports of severe reactions and deaths. This reduced the total number of deaths in infants younger than one year. In 1981 Japan introduced the acellular vaccine. This is said to be safer than the whole cell one.

A Swedish trial of one and two component acellular pertussis vaccines in 1986-87 compared vaccine to placebo. It concluded that side effects of the new vaccine were mild. The placebo was the 'vehicle', the liquid which 'carries' the vaccine. It contains thiomersal (a mercury containing compound), formalin and aluminium phosphate. The side effects of the new vaccine compared to this 'placebo' were indeed

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

minimal but, looking at the data, the incidence of floppiness, vomiting, inconsolable crying for more than one hour, fever and drowsiness that occurred after the 'vehicle' alone was substantial. The addition of the whooping cough component did not cause much more. It is certainly worrying that the 'vehicle' in which the vaccine is delivered seems to be so toxic. *(15a)*.



A report from Canada presented to the Infectious Diseases Society of America in Philadelphia in November 1999 suggested that there had been an 80% drop in admissions for seizures and a 75% decline in collapse within 72 hours of being vaccinated since the acellular vaccine had been introduced in that country *(16)*.

In the United Kingdom, acellular pertussis vaccine is not recommended for the primary vaccination program on the grounds of increased cost and reduced immunogenicity. However, only the three-component acellular vaccine is generally available in this country, not the five-component one which has been shown in Swedish trials to be as immunogenic as the whole cell *(17)*. The cellular vaccine is only recommended to be used in the pre-school booster because the side-effects of the whole cell pertussis vaccine in children over the age six months are so severe.

Pertussis vaccine is not usually given singly but side effects listed by the manufacturer for Acellular Pertussis Vaccine Adsorbed (APV) unlicensed in the UK and available for supply direct to the National Health Service Childhood Immunisation Programme 1997 list redness, heat, swelling and hardness at the site of the injection, fever, drowsiness, fretfulness and vomiting. Urticaria (itchy generalised skin rash) or other allergic reactions, persistent high pitched crying, convulsions, hypotonic –hyporesponsive (floppy-unconscious) episodes may occur. Neuropathies (nerve damage causing either muscle weakness or altered sensations) have been reported when also given with diphtheria and tetanus antigens (22). Whole cell pertussis vaccination is no longer supplied alone (as opposed to combined with DPT) as it is less immunogenic and causes more systemic reactions, especially fever (23).

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

Vaccination Recommendation

The major complications of whooping cough are most likely to occur in the first year of life. A baby whose mother had natural whooping cough is protected to some degree by transplacental immunity. Breast feeding prolongs this protection. If a child contracts the disease, they need to be nursed through it appropriately. I think that healthy children who are treated supportively rather than suppressively when they have childhood fevers are able to cope with having whooping cough. However, if parents are not happy with this course of action and wish to vaccinate, I think that they should only consider doing so with the acellular vaccine. It is currently unavailable as a single vaccine without the mercury containing additive thiomersal. The whole cell pertussis vaccine has such an enormously high incidence of side effects that I think it should never be used.

References

(3) The Health of Adult Britain: 1841-1994 Vols I,II Ed Charlton, J, Murphy M. London, The Stationary Office, ONS 1997: 15.3.5.
(4) Stewart GT. Re: "Whooping cough and whooping cough vaccine: the risks and benefits debate." Am J Epid 1984;119(1):135-9
(5) Ditchburn RK. Whooping cough after stopping immunisation. BMJ 1979;1:1601-1603
(6) Stewart GT. Vaccination against whooping cough. Efficacy versus risks. Lancet 1977;Jan29:234-7
(7) Kulenkampff M, Schwarztmann JS, Wilson J. Neurological complications of pertussis innoculation. Arch Dis Child 1974;49:46-9
(8) Alderslade R, Bellman MH, Rawson NSB, Ross EM, Miller DL. The national childhood encephalopathy study. In: Whooping cough. Reports from the Committee on Safety of Medicines and the Joint Committee on Vaccination and Immunisation. London: HMSO, 1981:79-169
(9) Miller DL, Madge M, Diamond J, Wadsworth J, Ross EM. Pertussis immunisation and serious acute neurological illnesses in children. BMJ 1993;1171-76
(10) Fine PEM, Chen RT. Confounding in studies of adverse reactions to vaccine. Am J Epidemiol 1992;136:121-135
(11) Gale JL, Thapa PB, Bobo JR, Wassilak SGF, Mendelman PM, Foy HM. Acute neurological illness and DPT: report of a case-control study in Washington and Oregon. In: Manclark CR, ed. Sixth international symposium on pertussis, abstracts. Bethesda, Maryland: Department of Health and Human Services, 1990:228-9. (DHSS publication No (FDA)90-1162.)
(12) Howson CP, Howe CJ, Fineberg HV, eds. Adverse effects of Pertussis and Rubella Vaccines. Division of Health Promotion and Disease Prevention, Institute of Medicine. Washington, DC: National Academy Press, 1991:65-124
(13) Bedford H, Elliman D. Childhood Immunisation a review for parents and carers. Health Education Authority 1998. London
(14) Odent MR, Culpin EE, Kimel T. Pertussis vaccination and asthma: is there a link? JAMA 1994;272:592-3
(15) Verrall M, Pertussis vaccine linked to atopy. Pulse 1st may 1999
(16) Henderson J, North K, Griffiths M, Harvey T, Golding J et al. Pertussis vaccination and wheezing illnesses in young children: a prospective cohort study. BMJ 1999;318:1173-6
(17) Storsaeter J, Olin P, Renemar B, Lagergard T, Norberg R, Romanus V, Tiru M. Mortality and morbidity from invasive bacterial infections during a clinical trial of acellular pertussis vaccine in Sweden. Paed Inf Dis J. 1988;7(99):637-45. (15a) Ad Hoc Group for the study of pertussis vaccines: clinical trial of two acellullar pertussis vaccines in Sweden: protective efficacy and adverse reactions as compared to placebo controls. Lancet 1984;1:122-126
(18) Sheifele DW, Halperin SA, Pless R, Delage G, Jadavji T, Vaudry W, et al. Marked reduction in febrile seizures and hypotonic-hyporesponsive episodes with acellular pertussis vaccines: results of Canada-wide surveillence. 1993-8 (abstract). Clin Infect Dis 1999;29:966
(19) Miller E. Overview of recent clinical trials of acellular pertussis vaccines. Biologicals 1999;27:79-86
(20) Harrison's Principles of Internal Medicine Ed Braunwald et al. 11th Ed McGraw Hill 1987. USA
(21) Gangarosa EJ, Galazka AM, Wolfe CR, Phillips LM, Gangarosa RE, Miller E, Chen RT. Impact of anti-vaccine movements on pertussis control: the untold story. The Lancet 1998;Jan31:356-61
(22) Minerva BMJ 1996;312:1620
(23) Van Buynder PG, Owen D, Vurdien JE, Andrews NJ, Matthews RC, Miller E. Bordetella pertussis surveillance in England and Wales 1995-7. Epidemiol Inf 1999;123:403-11
(24) Acellular Pertussis Vaccine Adsorbed (APV) unlicensed in the UK and available for supply direct to the National Health Service Childhood Immunisation Programme,1997
(25) Immunisation Against Infectious Diseases HMSO 1996).

Source of information for graphs: Deaths/Population1867-1900, Registrar General's Annual Returns, 1901-1994 Twentieth Century Mortality CDROM Office for National Statistics. Pertussis notification/vaccine coverage rates 1940-1998, Communicable Diseases Surveillance Centre, London NW9

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

TETANUS

Tetanus disease is caused the bacterium, *Clostridium tetani*. It is present in the gut of many farm animals and can live in spore form in soil and dust for many years. It is highly resistant to heat and drying. Colonisation by the organism itself does not produce symptoms. It is only when the organism produces toxin that harmful effects are caused. To produce toxin the organism needs to have anaerobic (no oxygen) conditions. This is why it is classically associated with wounds from rusty nails because it combines dirt with a deep penetrating wound that it is difficult for oxygen to reach. It is also why tetanus disease so rarely occurs despite undoubted frequent contamination of wounds. Other injuries that promote anaerobic infections are severe burns and severe crush injuries with the occurrence of dead, necrotic flesh. Tetanus infection has been reported after trivial or no injury (after tonsillitis, abortion, appendicitis).

Tetanus disease may develop five to 56 days after the injury, commonly 14. A shorter incubation period usually indicates more severe disease. The effects of tetanus disease may remain local to the site of the injury or become more generalised. People usually remain alert but may exHibit odd behaviour in the early stages. There may be irritability with muscle twitching and spasms, accompanied by a low grade fever. This can progress to 'lockjaw' and severe cases may have laryngeal spasm causing difficulty in swallowing saliva, spasm of the respiratory muscles necessitating artificial ventilation, and in some cases, death. Characteristically the symptoms worsen for three days, remain stable for the next 5-7 days and by two weeks may have disappeared all together. Most survivors recover completely in four weeks. All the effects of tetanus toxin appear to be self-limiting because those who recover from the disease have no residual defect *(1)*.

'Subacute tetanus' occurring in unimmunised patients has been described which has a good prognosis and a favourable outcome. The term 'subacute' is used rather than 'mild' because the presence of generalised muscle spasm is generally felt to imply at least 'moderate' tetanus. In these cases the good prognosis is maintained even with a short period of onset which with 'classic' tetanus is generally thought to predict a poor outcome *(2)*.

Complications contribute significantly to the likelihood of death or disability in tetanus disease. Some result from overly vigorous therapy and prolonged bed rest. It is important to keep the person in a low stimulation (dark, quiet and unemotional) environment and to guard against aspiration of secretions that cannot be swallowed properly. Tetanus antiserum/immunoglobulin does not neutralise toxin already fixed to tissue when it is given but its administration early in the diseases is associated with a reduced case fatality rate.

Neonatal tetanus is a severe disease that occurs in babies and is associated with poor hygiene in cutting the umbilical cord and care of the stump. It usually occurs within 10 days of birth and is characterised by difficulty sucking, grimacing, back arching and clenched fists *(1)*.

Tetanus disease is not regarded as being able to cause subsequent immunity but studies in isolated communities where people have not been vaccinated against tetanus (or anything else) have shown the presence of antibodies to tetanus toxin which increases with age. Immunity is thought to have been induced by ingestion of tetanus spores. These may germinate in the digestive tract and produce toxin which, depending upon the immune status of the individual, will give rise to either acute clinical or, more commonly, subclinical tetanus. Repeated infection of this type may induce both cell and antibody mediated immunity. It is thought that tetanus prone skin wounds may boost immunity but are unlikely to lead to primary immunity*(3,4)*

The lack of this gut based immunity may explain the occurrence of tetanus disease in fully immunised people with adequate levels of neutralising antibody *(5-7)* and the non occurrence of tetanus disease in unvaccinated individuals – such as everybody before vaccination was introduced, bearing in mind the ubiquity of the tetanus spores – in whom it is present.

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

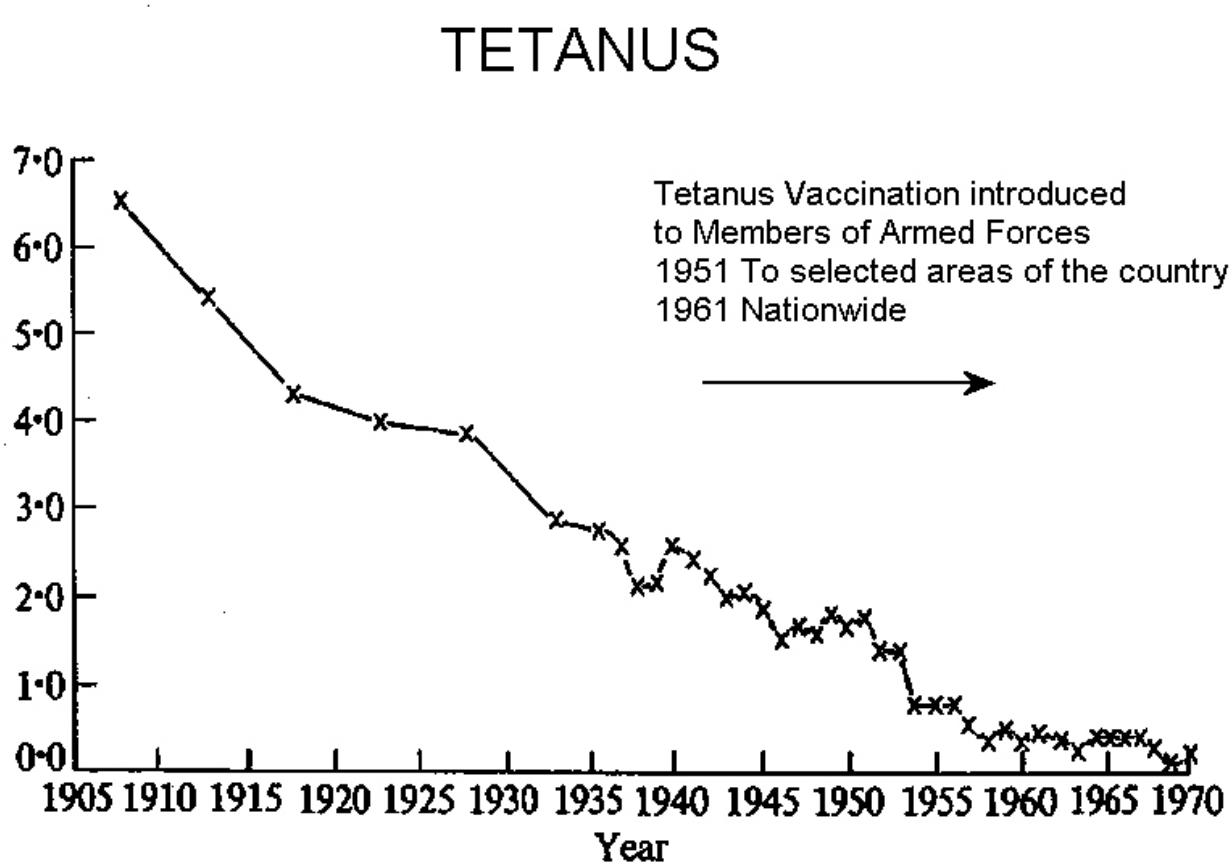


FIGURE 8.11. Tetanus: mean annual death rates: England and Wales.

(10)

The Vaccine

The vaccine is made from tetanus toxoid inactivated with formaldehyde and contains aluminium hydroxide and thiomersal (mercury containing compound)

It has been available since the Second World War and appears to have contributed substantially to reduced mortality from the disease. The considerable reduction in deaths before it was used are likely to be due to the availability of more effective treatment thus lowering the case fatality rate and other changes, such as the disappearance of the horse from the roads *(8)*

Listed side effects (*Adsorbed TETANUS Vaccine BP Pasteur Merieux MSD rev 1995)* are redness, swelling and tenderness or pain at the site of the injection, especially in adults. A persistent hard lump may be felt under the skin at the injection site especially if the injection was not very deep. Less commonly, there may be a raised temperature, headache, pallor and generally feeling unwell. These effects are expected to settle within 24-48 hours.

Other side effects are urticaria (itchy, generalised skin rash), angioneurotic oedema (swelling of the deeper layers of the skin especially the face and throat causing difficulty in breathing), acute anaphylactic shock (severe allergic reaction with respiratory and cardiovascular collapse).

Some people develop nerve damage causing either muscle weakness or altered sensations (*8*).

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

Vaccination of 11 healthy subjects with tetanus toxoid produced a lowering of the T-lymphocyte helper/suppressor ratio such as might be seen in patients with the acquired immunodeficiency syndrome (AIDS) *(9).*

Vaccination Recommendation

Prompt and adequate care of wounds is of major importance in preventing tetanus disease in vaccinated and unvaccinated people. They should be carefully cleaned, foreign bodies and devitalised tissue should be removed. Dirty wounds should not be sutured, to allow them to heal from the base up, reducing the likelihood of anaerobic conditions. Hydrogen peroxide 3% is very useful in this respect (H2O2). In the presence of pus or dead tissue it releases oxygen in high concentration. There is no evidence that the prescription of antibiotics influence the disease favourably (*1*). In a severe and tetanus prone wound, tetanus immunoglobulin may be given intramuscularly and intravenously in established cases of tetanus to produce immediately raised levels of antibodies.

In view of the above and the side effects associated with the tetanus vaccine, including the fact that it is only available in a vehicle containing thiomersal (a mercury compound), aluminium hydroxide and formaldehyde I would think that a reasonable alternative approach to the vaccine would be the promotion of a healthy immune system in the child combined with scrupulous wound toilet.

References

(26) Harrison's Principles of Internal Medicine 11th Ed, McGraw-Hill Inc 1987
(27) Ogunyemi AO, The clinical recognition of subacte tetanus, J Tropical Medicine & Hygiene 1986;89:131-135
(28) Veronesi R, et al, Naturally acquired antibodies to tetanus toxin in humans and animals from the Galapagos Islands, J. Infect. Dis.,1983;147 No2:308-311
(29) Matzkin H, Regev S, Naturally acquired immunity to tetanus toxin in an isolated community, Infection & Immunity 1985;48:267-268
(30) Luistro M, Iivanainen M, Tetanus of immunized children, Developmental Medicine and Child Neurology,1993;35:346-358
(31) Crone N, Reder AT, Severe tetanus in immunised patients with high anti-tetanus titres, Neurology, 1992;42:761-4
(32) Shimoni Z, Dobrousin A, Cohen J, Pitlik S, Tetanus in an immunised patient, BMJ,1999;319:1049
(33) The Role of Medicine, Thomas McKeown, Princeton University Press 1979:102-103
(34) Eibl MM, Mannhalter JW, Zlabinger G, Abnormal T-lymphocyte subpopulations in healthy subjects after tetanus booster immunization, New England Journal of Medicine 1984;310:198-9
(35) The Role of Medicine, Thomas McKeown, Princeton University Press 1979:103

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

POLIOMYELITIS

Polio occurs due to infection with poliovirus type 1-3. Polioviruses are enteroviruses and live in the intestine. Humans are the only known reservoir of the poliovirus which can be recovered from the stools of those with the disease and symptomless carriers as well as sewage and sometimes also from flies, water, milk and food. Transmission is mainly by ingestion of faecal material from a person with the disease or a carrier. Fly-borne spread has been postulated but unproven, as is the role of droplet (coughs and sneezes) infection.

Polio infection is usually asymptomatic. In those who have clinical symptoms they normally consist of a 'flu-like illness with or without 'summer diarrhoea'. In some cases there may be aseptic meningitis with full recovery. Actual paralysis, of which the majority recover, occur in 0.1-1% of cases*(1,2)*.

The virus enters the body through the gut or possibly the airways and after a one to three week incubation period may reach the nervous system via the blood stream. The risk of central nervous system involvement is increased during the incubation period by excessive physical exercise, intramuscular injection (especially of DPT) and tonsillectomy. Injections are associated with paralysis of the limb injected (provocation poliomyelitis) and tonsillectomy with bulbar involvement (paralysis of the muscles of breathing necessitating artificial ventilation) (*1*).

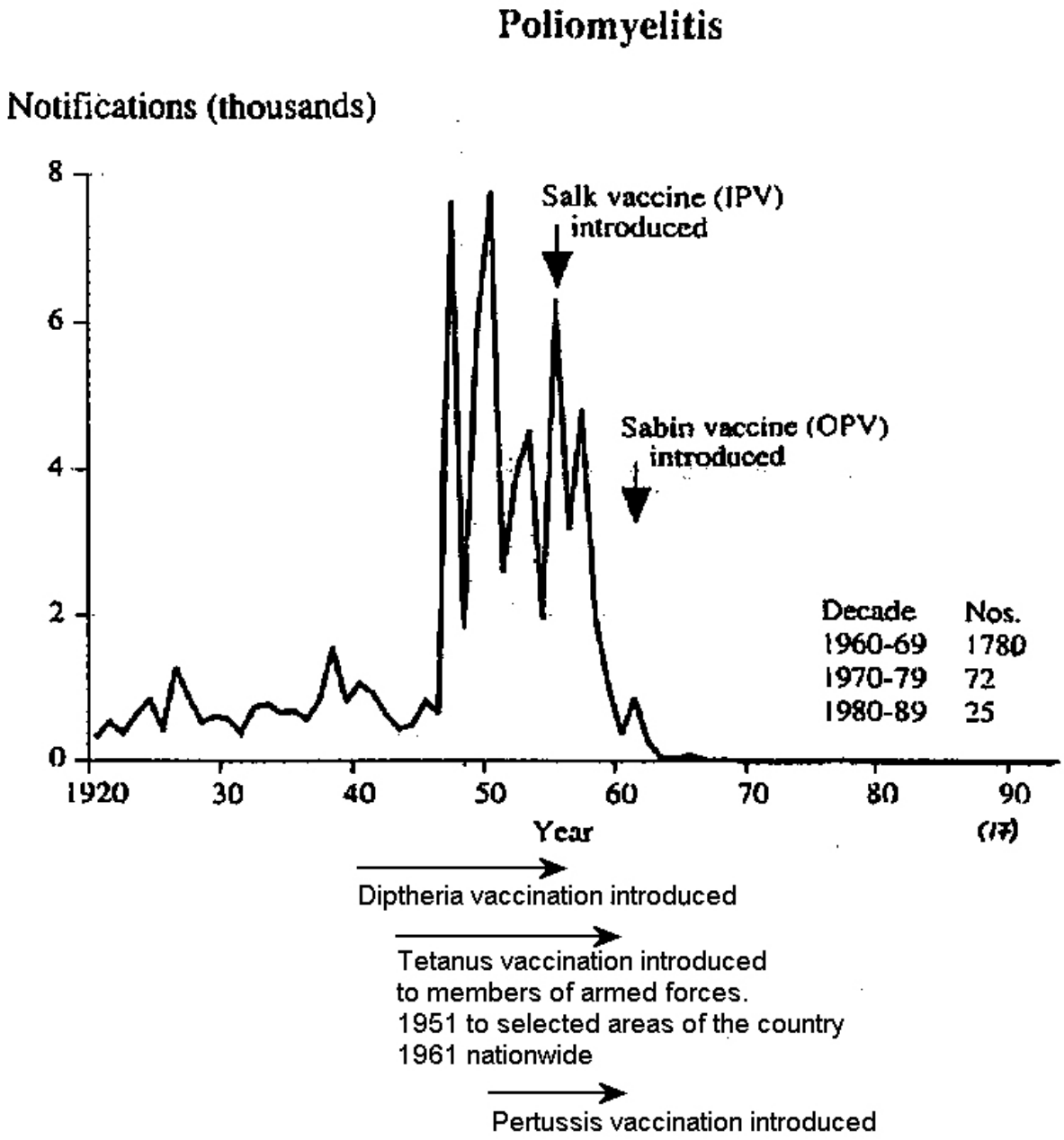
Poliomyelitis first emerged as a distinct disease entity in the late 19th, early 20th century, indeed it was not classified as a separate disease until 1911. This was about the same time as anti-toxin was introduced for the treatment of diphtheria (1895) and tetanus (First World War)*(3)*.

The incidence of the disease continued at moderate rates through the early twentieth century until the 1940s when enormous increases in the incidence of paralytic disease occurred which were thought to be due to excessive hygiene, meaning that children were not coming into contact with the virus and developing immunity to it at a young age. It also coincided with the widespread use of antibiotics which were initially all injected intramuscularly and massive vaccination campaigns against diphtheria (1940s onwards), tetanus (World War Two: armed forces personnel, 1951: selected districts UK, 1961: nation-wide coverage) and pertussis (1950-1957 and onwards). See graph

The introduction of the vaccine appears to have caused a great reduction in paralytic disease apart form large outbreaks of poliomyelitis in vaccinated children with many deaths which occurred when certain batches of vaccine from the Cutter laboratories (USA) were used. However the effect of the vaccine is not so clear as it at first appears. Before the introduction of the vaccine polio was diagnosed on clinical grounds and paralytic polio by the presence of paralysis. After the vaccine was introduced new stringent parameters were set for the diagnosis of polio including the presence of the virus in stool and serum samples and paralysis present for 60 days. This immediately removed most cases of paralytic polio from the figures as the vast majority of cases of paralysis have recovered by then. The requirement for laboratory diagnosis of polio also removed from the figures cases of polio caused by other enteroviruses like Coxsackie viruses as well as cases of aseptic meningitis, Guillain-Barre syndrome, transverse myelitis, encephalomyelitis, other cases of acute flaccid paralysis etc which would previously have diagnosed as polio. The change in the criteria for diagnosis of polio alone would have markedly reduced the number of cases reported (cf measles and Hib). This has occurred in every country to which the vaccine has been introduced.

Human milk can neutralise poliovirus and limitation of intestinal shedding of vaccine poliovirus by breast feeding has been shown*(4)*.

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk



The Vaccine

Polio vaccine is available in two formulations; an inactivated form (Salk, IPV) which is killed and injected and a live form (Sabin, OPV) which is given orally. As it is live it must not be given to anyone with untreated cancer or altered immunity, those receiving immunosuppressive or Xray therapy or high dose steroids. It must not be given within three weeks of another live vaccine but the recommendation is that it may be given at the same time. It should not be given to pregnant women. The oral polio vaccine is regarded as an ideal form of vaccine as it most closely mimics wild infection and induces a mucosal reaction as well as antibodies in the blood. In the UK OPV is used in the childhood immunisation programme.

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

The risk of a recipient of oral polio vaccine developing paralytic polio is estimated to be one in 520 000 first doses administers and one in 12.3 million subsequent doses (*5*). A study in England & Wales showed that recipients who became paralysed after the vaccine did so 11-28 days after receiving it (the ninth revision of the international classification for diseases, ICD-9, requires that paralysis occurs within 4-30 days after vaccination). Those who became paralysed after contact with a vaccinated person did so 25-52 days after vaccination (7)
Vaccine viruses excreted by recipients of oral polio vaccine may show evidence of mutation which in some cases produces increased neurovirilence. This is due to reversion towards the original characteristics of the wild virus *(5).*

In Britain with 94% coverage of oral polio vaccination no cases of wild polio were reported for 10 years (1984-94) *(8)* so all of the 28 reported cases of paralytic polio reported during that time period must have been vaccine related.

In the USA between 1980 and 1994, 133 people contracted paralytic polio. 125 of these cases (94%) were attributed to the oral vaccine such that the oral vaccine is no longer given as the first dose in that country*(9).*

As the World Health Organisation struggles to achieve its aim of worldwide eradication of polio it is notable that epidemics of paralytic poliomyelitis have occurred in highly vaccinated populations and, tragically, immediately after a polio vaccination campaign has occurred*(10,11,12)*. Indeed, as India struggles to meet the polio deadline, several cases have been reported of children contracting polio even after receiving up to a dozen doses of the vaccine*(13)*. In the Oman outbreak it was notable that the region with the highest attack rate (of paralytic polio), 117/100 000 had one of the highest (vaccine) coverage rates, whereas the region with the lowest coverage had a low attack rate, 6/100 000*(10)*. In a polio outbreak in Israel in 1988 in a highly vaccinated population. People vaccinated with inactivated polio vaccine (eIPV) served as reservoir for poliovirus multiplication and transmission to susceptible individuals. Such people have low intestinal mucosal immunity and excrete poliovirus for periods ranging from days to several weeks. The spread was also linked to the greater susceptibility of young adults previously vaccinated with OPV *(14).*

In 1961, inactivated polio vaccines (IPV) was found to contain live SV40 (simian vacuolating virus 40), a monkey virus that is more resistant that the poliovirus to chemical denaturation (inactivation)*(15)*. The live oral polio vaccine is grown on green monkey kidneys. Although batches of vaccine are tested for contaminants, those below a certain level are disregarded. Physical methods alone are used, not the more sophisticated genetic methods that have more recently become available, and it stands to reason that any method which leaves the vaccine virus alive is unlikely to have killed the other ones. An increased incidence of tumours of the nervous system has been reported in one study in children of mothers vaccinated during pregnancy. SV40 has now re-emerged as a potentially tumour causing virus and has been found in a variety of childhood neural tumours in a distribution that mirrors the range of tumours induced in animals who are injected with the virus. The virus has also been found in lymphomas (non-Hodgkin's> Hodgkin's). Not many tumours have so far been investigated for the presence of this virus so it is likely that more will emerge in time. SV40 has been found in people far to young to have been immunised with the documented contaminated vaccines so it is possible that it is still being transmitted through the vaccine or, having been incorporated in the human genome, vertical transmission is occurring from parents immunised with the contaminated vaccine to their children. It thus remains possible that a late adverse effect of the polio vaccination programme is emerging (*15).*

Listed side-effects of the vaccine are:

OPV *(Monodose OPV SmithKline Beecham 1995*) Paralysis has been reported very rarely in recipients or contacts. The patient information leaflet *(1994)* also mentions allergic reactions including a rash, tightness of the throat, shortness of breath or other unwanted effects.

IPV *(Imovax Polio, Pasteur Merieux Connaught 1998)* lists redness at the site of the injection and a moderate fever in a few cases. The US data sheet for *IPOL poliovirus vaccine inactivated, Pasteur*

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

Merieux Connaught 1992 also listed Guillain Barre syndrome as being temporally (occurring within the same time frame) as another inactivated poliovirus vaccine.

Vaccination Recommendation

Paralytic poliomyelitis is very rare in this country – at least paralysis that is reported to be due to polio. It is hard to know how much actual polio of the more usual sort occurs – asymptomatic or 'summer diarrhoea' as it is not looked for even though it is a notifiable disease. Indeed I was greeted with incredulity when I asked my local department of public health what tests I should be doing on children with summer diarrhoea to see if they had polio –"if they are not paralysed, why do you care?" they asked. I mentioned that it was because it is a notifiable disease but I was told not to bother. It is quite probable that there is a lot of mild polio because we are told that babies excreting vaccine virus into the community "provide an additional community benefit as contacts of recently immunised children may be protected through acquisition of the vaccine virus *(2)*."

May unvaccinated children go swimming?

Dr David Isaacs, Head of the Department of Immunology & Infectious Diseases, Sydney, Australia says, "In Britain there is a popular belief that babies should not be taken to their local swimming baths until they have had their first immunisation. It has no logic, given the scant protection provided by the first as opposed to the second and third immunisations and the high degree of sterility of most chlorinated pools. It is yet another myth about immunisation and we should not put restrictions on age for swimming in public pools *(16)*."

Due to the rarity of paralytic polio in the UK, USA and other such countries and the fact that almost 100% of cases that do occur are due to the vaccine I do not think that it benefits a child to put them at such a risk, particularly in view of the, as yet, unknown risk of the contaminants which are still being investigated.

References

(36) Patersons Sick Children Ed Lightwood, Brimblecombe & Bartrope, Balliere, Tindall & Cassell London 1971.
(37) Immunisation Against Infectious Diseases, HMSO London 1996
(38) McKeownT, The Role of Medicine, 1979, Princeton University Press.
(39) The Health of Adult Britain 1841-1994, Ed Charlton & Murphy, The Stationary Office ONS London, 1997p12
(40) Warren RJ, Lepow ML, Bartsch GE, Robbins FC. The relationship of maternal antibody, breast feeding and age to the susceptibility of newborn infants to infection with attenuated polioviruses. Pediatrics 1964;34:4-13
(41) Elliman D. Any Questions BMJ 1995;310:1044
(42) Joce R,Wood D, Brown D, Begg N Paralytic poliomyelitis in England and Wales, 1985-91 BMJ 1992;305:79-82
(43) Polio Vaccination has nearly 100% cover in Britain BMJ 1995;310:958
(44) US changes polio vaccination programme BMJ 1997;314:465
(45) Sutter RW Patriarca PA, Brogan S, Malankar PG, Outbreak of paralytic poliomyelitis in Oman: evidence of widespread transmission among fully vaccinated children, Lancet 1991;338:715-20.
(46) Fiore L,Genovese D, Diamanti E, Catone S et al, Antigenic and molecular characteristics of wild type 1 poliovirus causiing outbreaks of poliomyelitis in Albanina and neighboring countries in 1996 J Clin Microbiol 1998;36:1912-18
(47) van Niekerk ABW, Vties JB, Baard J, Schoub BD, Chezzi, Blackburn NK, Outbreak of paralytic poliomyelitis in namibia, Lancet 1994;344:661-4
(48) India struggles to meet polio deadline, BMJ 2000;321:403
(49) Slater PE, Orenstein WA, Morag A, Avni A, Handsher R et al, Poliomyelitis outbreak in Israel in 1988: a report with two commentaries, Lancet 1990;335:1192-8
(50) Stenton SC, Simian virus 40 and human malignancy BMJ1998;316:877
(51) Isaacs D Any questions BMJ 1992;305:290

The Health of Adult Britain 1841-1994, Ed Charlton J, Murphy M, London, The Stationary Office, ONS 1997:12. Legends added by JLMD

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

HAEMOPHILUS INFLUENZAE B

Haemophilus influenza B (Hib) is a bacterium that affects humans only. It occurs in capsulated and unencapsulated forms. Infection with the unencapsulated form may cause coughs, colds, sinusitis and ear infections. The encapsulated form has six types (A-F) and can cause invasive disease. Type B does so most commonly. In 'invasive' disease the organism may spread directly to inflame the skin of the face or cause epiglottitis. It may also invade the blood stream and cause more distant infection such as meningitis, arthritis or pneumonia. Invasive disease is most likely in children aged 3-48 months, the peak age being ten to eleven months. Boys are more commonly affected than girls and the highest seasonal incidence is between autumn and spring.

The symptoms of Hib meningitis are similar to those of other forms of meningitis in children. Hib meningitis occurs more commonly after colds, coughs and ear infections in young children and seizures are more common. The disease tends to have a slower onset than meningococcal meningitis and is less likely to cause a rash. It also tends to occur less often in epidemics.

The incidence of invasive disease caused by the encapsulated forms (A-F) of H. influenza have been rising since the 1950's which is also the time that mass vaccination was introduced and antibiotics started being prescribed so liberally.

90% of healthy individuals carry Haemophilus influenza in their noses, approximately 5% of these organisms will be type B. Antibodies form against the encapsulated forms either as a result of disease or of asymptomatic carriage in the nose or the pharynx. Antibodies may also be formed as a result of infections with other organisms that have similar capsules (eg *E. coli k100*, found in the gut)*(1)*

The Vaccine

The current vaccines are 'conjugate' vaccines. The original Hib vaccines were unconjugated and did not produce an immune response in children below the age of 18 months. The conjugate vaccines are made of a purified part of the capsule of Hib and are joined (conjugated) to another material with the aim of provoking an immune response at a younger age. The conjugate vaccines have been used since the mid 1980s and were licensed for use in two month old children in the USA in 1990. They were added to the UK Childhood Immunisation Programme in 1992. There are several varieties available depending on the material to which they are conjugated:

PRP-D Conjugated to diphtheria toxoid
PRP-T Conjugated to tetanus toxoid (*Pasteur Merieux ActHib and* ActHibDPT/ SmithKline Beecham Hiberix)
PRP-CRM Conjugated to Cross Reacting Mutant Diphtheria Toxoid (*Lederle Praxis CRM197 HibTITER*)
PRP-OMP Conjugated to Outer Membrane Protein of Group B Meningococcus
HbOC Conjugated to oligopolysaccharide

The Handbook of Immunisation against Infectious diseases (HMSO 1996) quotes several large field trials in Finland, the USA and the UK as evidence of its efficacy but the trials showing the highest efficacy were for the PRP-OMP and HbOC, neither of which are available in this country.

The trials of efficacy were over one to two years so none of the data were long term. In terms of side effects, the studies were on children given Hib, DPT and polio vaccine compared with those given only DPT and polio. This is not a very satisfactory control group bearing in mind the high incidence of adverse effects with the other vaccines.

Listed side effects (*ACT-Hib Pasteur Merieux 1998, Hib-TITER Lederle 1995)* are redness, swelling and tenderness or pain at the site of the injection, fever, headache, malaise, irritability, inconsolable and high pitched crying, rarely convulsions, erythema multiform (vivid red skin lesions caused by acute inflammatory infiltrate around blood vessels in the deeper layers of the skin) and transient cyanosis (blueness) of the lower limbs have been reported. The British National Formulary of 1996 mentioned loss

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

of appetite, vomiting, diarrhoea, rash, and urticaria (itchy generalised rash). Other reaction reported to the Vaccine Adverse vent Reporting System (VAERS) if the USA are Guillain-Barre Syndrome -a neurological disease which may eventually cause paralysis of the respiratory muscles requiring artificial ventilation, transverse myelitis – a paralytic disease mainly involving the legs and death (which may have been caused by the Hib vaccine or by other vaccines that were given at the same time). *(2,3)*

The vaccine is only against type B so it only affects infections caused by type B. It is possible that as infections by type B are suppressed there will be a drift towards infections produced by the other types – A and C to F instead.

When the vaccine was introduced it was not given to children over the age of four years or adults because they were regarded as already immune. However, once the vaccine was introduced children would be less likely to carry it in their nose and gain natural immunity. This would be expected to increase the likelihood of their contracting severe forms of the disease at a later age as has, in fact, been the case. After the introduction of the vaccine in 1992, cases of Hib disease were dramatically reduced, although some of this was thought to be due to significant underreporting of cases after introduction of the vaccine combined with more rigorous case definition with "consequent overestimation of the effectiveness of the immunisation programme"*(4).* However, after this initial decline, cases in children aged five to eleven months have been rising in England and Wales from 0.15 per 100,000 in 1998 to 0.76 in 2000 and in 2001, a similar rise was seen in one to two year olds; 0.45 in 1998 to 3.97 per 100,000 in 2001 (*figures presented by Dr J McVernon at the International Network of Paediatric Surveillance Units in York April 2002)*. It was thought to be due to possible problems with vaccine efficacy but is difficult to test because of lack of information on antibody levels in these children after their primary course *(5)*due to lack of long term studies.

In 1992 before the vaccine was introduced, carriage of Hib was found in 4% of 1,500 children tested, this had dropped to 0.7% by 1994 and in 1997 none of 500 children were carrying it (*unpublished results of Public Health Laboratory Service study)(6)*. The reduced carriage rates are leaving children without natural immunity to Hib and invasive disease is occurring in older children who would previously have been expected to be immune. Rather than giving serious thought to removing this vaccine from the schedule, a campaign to give a 'catch up' dose to all under four year olds took place in 2003 and a decision is being made as to whether or not to add HiB to the preschool booster. A booster dose given to four to five year olds is likely to push invasive disease into adulthood.

Vaccine Recommendation

Due to the risks associated with the vaccine and the disruption it causes to natural, long lasting immunity which could cause invasive disease at a later age I do not recommend the vaccine. I think that healthy children who are treated supportively rather than suppressively when they have childhood fevers are able to cope with encountering Hib.

References

(52) Harrison's Principles of Internal Medicine 11th Ed, McGraw-Hill Inc 1987
(53) Immunisation against Infectious Disease HMSO London 1996
(54) The Vaccine Guide, Randall Neustaedter, N Atlantic Books 1996
(55) Decrease in effectiveness of routine surveillance of H. influenza disease after introduction of the conjugate vaccine, Olowokure B,HawkerJ,Blair I,Spencer N,BMJ;2000;321:731-732
(56) Vaccine experts to debate need for booster Hib jab, Pulse, 15 April 2000
(57) Hib vaccine boosts herd immunity, Pulse, 27 May 2002

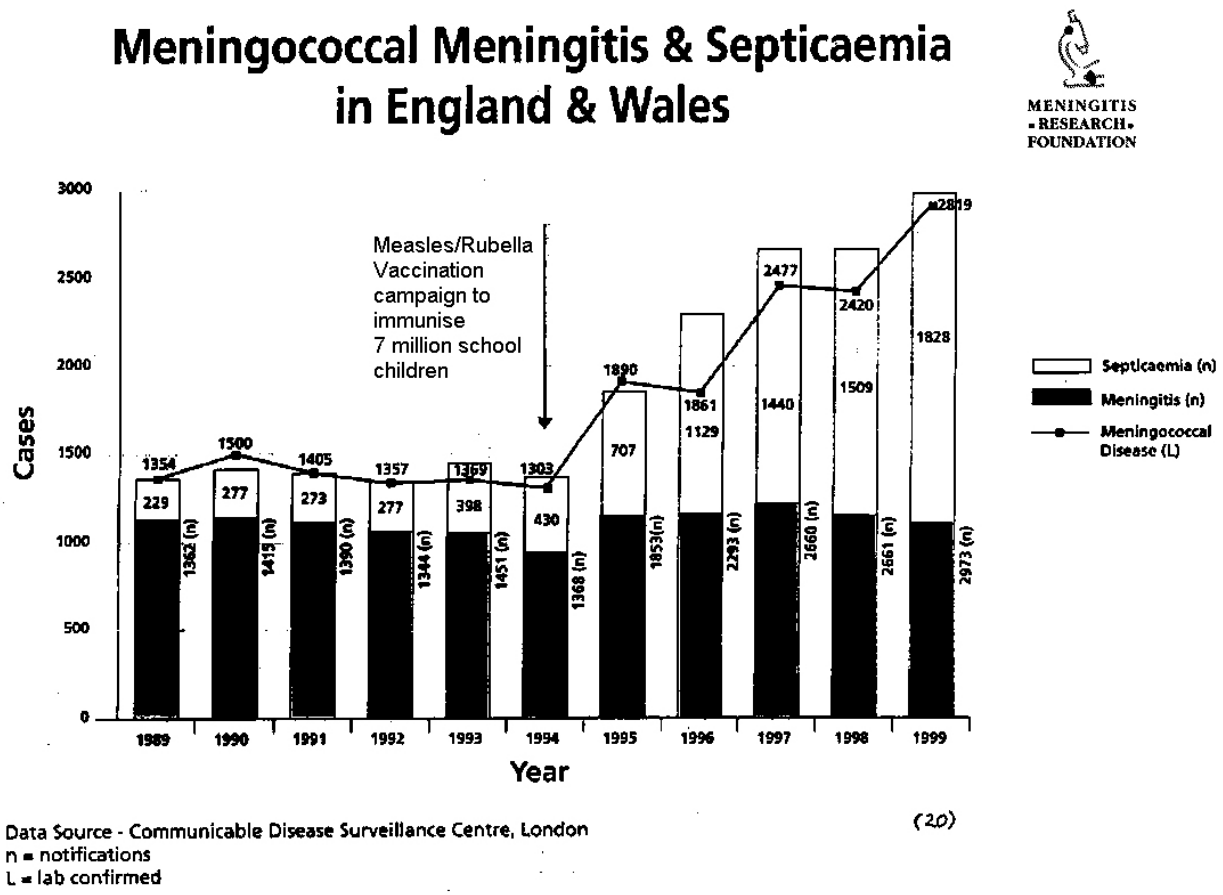
Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

MENINGOCOCCUS C

The meningococcus (otherwise known as *Neisseria meningitidis)* is a bacterium that lives in the nose and in the back of the throat of humans. It is spread by coughs and sneezes, not by clothing or bedding. At any one time, up to one in six people carry it in their nose passages without any particular symptoms. When occasional cases of meningococcal disease occur, this 'carrier' rate may rise to one in two people, in times of epidemic disease, all of us may carry it. Disease is not the same as 'carriage'. Most of thhose who 'carry' the meningococcus clear it on our own over a period of a few months, some people even carry it for years with no symptoms.

There are different varieties of meningococcus, known as groups, based on differences in the outer coating. They range from A – Z. Those known to cause disease in humans are groups A, B, C, W135 and Y. Once any of the meningococci have been 'carried', protective antibodies are developed to all of the groups. If one is unfortunate enough to get disease - when the meningococcus leaves the nose or throat and invades the blood stream (SEPTICAEMIA) or the brain or spinal cord (MENINGITIS) – then immunity is only gained to that particular group or strain (subdivision of a group).

The biggest factor determining whether the meningococcus in a person's nose is able to invade or not is the state of their immune system. Other things that make a lesser difference are the strain virulence, factors that spread the meningococcus from the throat and nose (eg a 'flu epidemic), and overcrowding associated with poverty*(1)*, young military recruits and university halls of residence *(2)*.



Most people who get disease do not get it from someone else with disease, they get it from an asymptomatic carrier *(3)*. This makes it very difficult to know whom to treat with antibiotics to stop passage of disease. In fact, unbridled use of antibiotics in schools and communities of those with disease may impair the body's ability to actually develop immunity to the meningococcus (similarly to diphtheria) as well as creating widespread resistance to antibiotics for those who really need them *(4)*.

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

The highest incidence of meningococcal disease is in children – boys – aged six months to one year – and in the winter. Most adults have protective antibodies. Earlier this century a large proportion of disease was caused by group A. From the 1960's most disease was caused by group B. In the late 1980s the percentage of cases of disease caused by group C rose from 30% to 40%. It caused more disease in older age groups, especially 15 to 24 year olds in whom the death rate is higher (15% of those with the disease compared to 5% in infants less than one year) and there are more cases of septicaemia (up to 70% in one series of deaths*(5)*)

Why would this be happening? Certainly being a university student is not the only factor. Dr Keith Neal of Nottingham University surveyed 75 universities over three years. He found that some reported no cases and some had a very high incidence, up to 40 cases per 100 000 students (the national rate for non-students of this age is 5 per 100,000 people). He thought that living in halls of residence as opposed to home could be a cause. But the risk is still very low, and most cases are still group B *(2)*.

Looking at possible reasons for a weakening of people's immune systems over the last ten years or so which would make them more susceptible to invasive disease it is certainly the case that children are having a much larger number of vaccines and at an earlier age than in the past. It may be that this is affecting their immune systems such that they are less able to cope with everyday pathogens. In the Measles Rubella Campaign in 1994 seven million five to sixteen year olds were vaccinated - some for the second and third time against measles. These are the people who would have going through university when the outbreaks of meningococcal C meningitis were occurring.

The Vaccine

The new vaccines against meningococcus C are 'killed vaccines' (the organism is not live) and have been developed using the same technology as that used to make the Haemophilus Influenza B (Hib) vaccine. A vaccine against meningococcus groups A and C and another one against groups A,C,Y and W135 have been available for years. They only produce antibodies for about three years and then only in those above 18 months of age.
It was reported in March 1997 that trials of two group C meningococcal vaccines had produced very good antibody responses *(6)*. In November 1998 Dr Elizabeth Miller, Head of Immunisation at the Communicable Diseases Surveillance Centre presented results of Government funded trials. She said, "a three dose schedule of the conjugate (new) vaccine at two, three and four months was *highly immunogenic* and had an *excellent safety profile*"*(7)*.

In the Oxford Vaccine Group study, 248 infants were vaccinated at two, three and four months while a **control** group received hepatitis B vaccine. At five months, 100% of infants had developed greater than 2mcg/ml of antibody. In a separate Government funded trial (no numbers mentioned, no follow up mentioned) 99% achieved putative protective antibodies. Public Health Laboratory researchers carried out a trial on 227 children aged 12 – 17 months to see whether one dose would be enough to provide *long term* protection for older children. One month later, 94% had putative protective levels *(8)*.

In July 1999 it was reported inthe British Medical that the new vaccine, unlike the existing one, provided long term protection. Dr Elizabeth Miller who had been co-ordinating the vaccine trials said that the vaccine had been given to 4500 children in the UK some of whom had been followed up for five years. Frank Dobson the Health Secretary said that the new immunisation program should start in October 1999 and expand as rapidly as manufacturers could supply vaccine *(10)*.

A letter in July 1999 from the Chief medical Officer to all doctors said that the vaccine was immunogenic in children from two years of age and appeared to induce immunological memory so that further boosting was likely not to be needed. The new vaccines were said to have been extensively tested by the manufacturers and the Public Health Service Laboratory Service and been found to have excellent immunogenicity and safety profiles in all ages*(11)*. It must be said that many of the follow ups were not very long, "some had been followed up for five years."

Immunogenicity means antibody levels. Antibody levels are not the same as immunity as was emphasised with the mumps vaccine in Switzerland in the 1990s*(see below)*. It is according to these levels that vaccines are regarded as providing immunity or not but it is only a surrogate measure for

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

immunity. That is why it is only said that the vaccines appear to induce immunity and that the levels are putatively protective.

Safety. The control group in one of the three trials of this vaccine was of children who were vaccinated with Hepatits B which is problematic as it is not without its own side-effects such that it has been removed from the schoolgirl vaccination programme in France due to an association with multiple sclerosis.

In 1997 the Department of Health was said to be resisting pressure to introduce blanket meningitis vaccinations for university students, "The problem is that several hundred thousand students would need to be vaccinated when the incidence of the disease is actually very small"*(14)*. In 1998, Southampton Local Medical Committee chairman Nigel Watson said that they advised against routine vaccination of 8000 new students as there was, "No clinical evidence to support it"*(15)*.

The vaccine was nonetheless introduced in November 1999 and there were unprecedented numbers of side effects reported. 15 million doses were distributed to be given to infants children and 15-17 year olds. By 29 August 2000 the Committee on the Safety of Medicines (CSM) had received 7,742 yellow card reports associated with meningitis C vaccine. This compares with 5750 reports received on the pertussis vaccine in the previous 37 years. More than one reaction was often reported on the same card and in this case there were a total of 16,527 reaction which gives a specific reaction of 11 per 10,000 doses distributed. Twelve deaths were reported to the CSM but it was decided that none of them were due to the vaccine *(16,17)*. One reason given for greater reporting of side effects was that nurses were allowed to fill in yellow cards for the first time and it is thought that they may be more open to reporting side effects than doctors.

Listed side effects (*Meningitec John Wyeth & Brother Ltd 1999)* are redness, swelling, tenderness, pain at the site of injection occasionally at least 3 cm and interfering with movement for 24 hours. In infants and toddlers: crying, irritability, drowsiness, impaired sleeping, anorexia, diarrhoea and vomiting were common but were thought to be due to concomitant administration of DPT vaccine. In adults, common side effects are headaches and myalgia (muscle pain).

By the summer of 2000 the CSM advised that further side effects should be added to the product information of the vaccine in relation to older children and teenagers:
Nausea, vomiting, rash, malaise, lymphadenopathy (swollen lymph glands), headache, myalgia and allergic reaction including allergic ones. Neck stiffness and photophobia have also been reported and convulsions a rate of one report per 100,00 doses*(17)*.

Reports of more headaches with sweling and redness at the injection site were reported for Menjugate produced by Chiron. Unlike Meningitec, this product contains aluminium hydroxide which may explain the difference.

The Meningococcus C vaccine only has any effect of disease caused by Meningococcus C. As is the case with the Hib vaccine, it is to be expected that as disease with one type declines, there will be a drift to more disease caused by others. This has certainly been seen with the polysaccharide meningococcal C vaccine. When used on US forces the incidence of meningococcal C disease was reduced two to three times but the total meningococcal acquisition rate was essentially the same regardless of vaccine status. Thus, the vaccinated recruits, although protected against group C organisms, acquired meningococci of other serogroups. In fact, the attack rate of group B meningococcal disease was higher among the vaccinated recruits *(18)*.

The introduction of meningococcus C vaccine has been hailed as an unqualified success by the Department of Health with reductions of up to 75% of expected disease. However, disease caused by meningococcus B has continued to rise in the England & Wales from 1400 cases in 1998/1999, mid year total to 1686 in 2000/2001 and 'other groups' rising from 103 to 186 respectively*(19)*.

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

Vaccine Recommendation

Crucial to a child's experience of meeting meningococcal organisms is the state of their immune system. Their immune system is supported, apart from by diet, fresh air, exercise and love, by supportive rather than suppressive treatment of childhood fevers. Fevers caused by infectious organisms, usually viruses, are how the child's immune system learns to respond appropriately to mild pathogens. This means that when potentially more serious ones come along, the immune system knows what to do. I think that this is a better protection against invasive meningococcal disease than vaccination.

References:

(1) Rees Jones I. et al. Social deprivation and bacterial meningitis in NE Thames region: three year study using small area statistics. British Medical Journal 1997;314:794-795
(2) Pulse, 29 Nov 1997, Vaccines would not stop disease, and Guardian Higher, 11 Nov 1997 p11, Menace in residence, quoting Dr K. Neal, University of Nottingham.
(3) Bjorn-Erik K. et al. Which contacts of patients with meningococcal disease carry a pathogenic strain of Neisseria meningitidis? A population based study. British medical Journal 1998;317:621-625
(4) Bjorn-Erik K. et al. Secondary prevention of meningococcal disease. British Medical Journal 1996;312:591-2
(5) Pulse, Aug 1999, Meningitis deaths mainly septicaemia, quoting Dr N. Ninis, Clinical researcher on Confidential Study of Meningococcal Disease, Royal College of Paediatrics.
(6) Pulse, 8 Mar 1997 p24, Reformulated meningitis B Vaccine trials, quoting Dr E. Miller Head of Immunisation, Communicable Diseases Surveillance Centre, Co-ordinator of the meningococcal group C vaccine trials.
(7) Pulse, 14 Nov 1998 p7, Meningitis C trials pave way for nationwide vaccination, quoting Dr E. Miller.
(8) Pulse,1 May 1999, New meningitis C vaccines by 2000.
(9) Pulse, 28 Aug 1999 p4, Unlicensed use of vaccine defended.
(10) Wise J. UK introduces new meningitis C vaccine. British Medical Journal 1999;319:278
(11) Introduction of immunisation against group C meningococcal infection. Dept of HealthPL/CMO/99/2, PL/CNO/99/4, PL/CPHO/99/1, 20 Jul 1999
(12) Schegel M. at al. Comparative efficacy of three mumps vaccines during disease outbreak in Switzerland: cohort study. British Medical Journal 1999;319:352-3
(13) Schonhofer P.. Letters. British Medical Journal1998;316:68
(14) Pulse, 29 Nov 1997, Student meningitis initiative resisted.
(15) Pulse, 22 Aug 1998 p8, GP's in meningitis vaccination fee row.
(16) Message from the chairman of the CSM and the chairman of the joint committee on vaccination and immunisation on meningitis C vaccines Sept 2000
(17) Safety of meningococcal group C conjugate vaccines, Current Problems in Pharmacovigilance Prepublication Copy.
(18) Artenstein MS, Gold R, Zimmerly J, Wyle FA, Schneider H, Harkins C, Prevention of meningococcal disease by group C polysaccharide vaccine, New England Journal of Medicine 1970;282:417-20
(19) Public Health Laboratory Service, Laboratory confirmed N.meningitidis England and Wales by Group 1989-2001 mid year totals www.phls.co.uk/seasonal/meningococcal/meningitis
(20) Meningitis Research Foundation, Bristol 1999

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

MEASLES

Measles disease is caused by a virus. One attack almost always confers permanent immunity. The incubation period is seven to 21 days and starts with a fever, the eyes and nose run and the face is puffy. There is a general feeling of being unwell. Koplik's spots appear inside the cheeks and towards the back of the mouth. This lasts for four to five days. The fever usually dips and then rises again with the appearance of the rash. In typical measles, the rash appears behind the ears, round the mouth and then spreads to the rest of the body. The throat is sore and there may be a bronchitic cough. There is usually photophobia (light hurts the eyes) and conjunctivitis causing sore eyes and a discharge. The neck glands are often swollen and sore. After about five days the rash starts to fade, the temperature drops and the symptoms all resolve. Treatment consists of general measures to keep the child comfortable and well hydrated (lots of drinks). Nursing in a darkened room is helpful if photophobia is troublesome. The most common complications are pneumonia, ear or mastoid infections and conjunctivitis *(1)*.

Most deaths due to measles follow complications such as pneumonia, croup and diarrhoea and are often associated with malnutrition *(2)*, particularly lack of vitamin A *(3,4)*. The Immunisation against Infectious diseases Handbook (1996) also states that, "complications are also more common and severe poorly nourished and chronically ill children *(5)*. This is why measles is such a killer in developing countries.

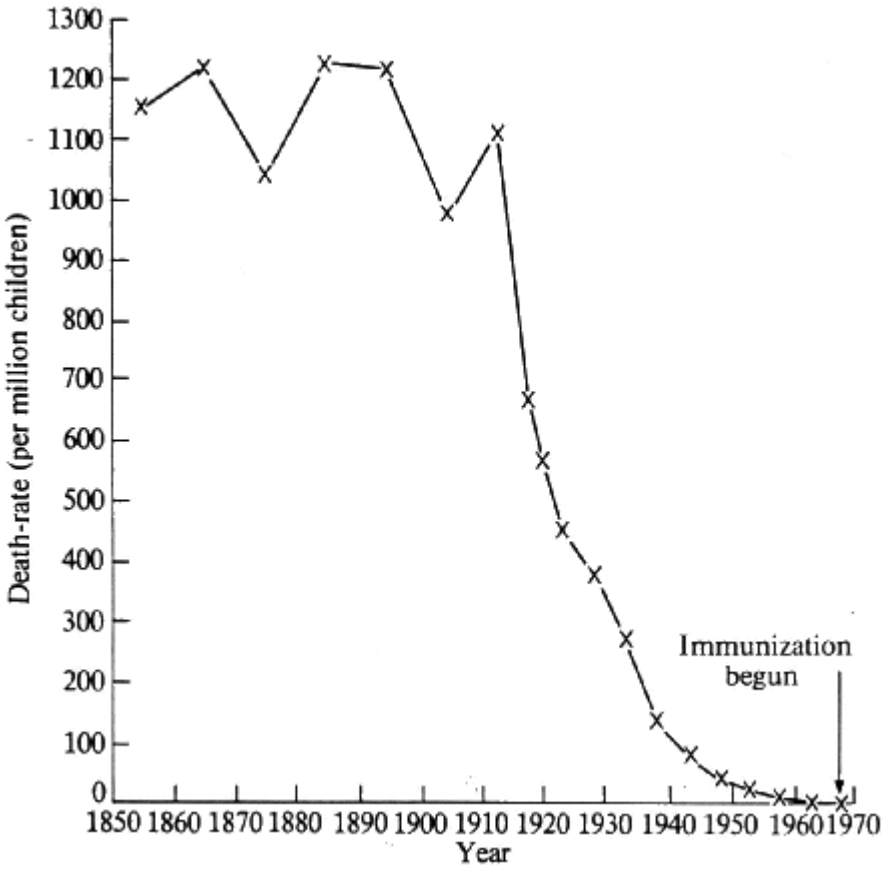
The Vaccine

The measles vaccine contains a live attenuated version of the measles virus. The MMR vaccine used in the UK contains either the Enders' Line of the Edmonston strain or the Schwartz strain. As it is a live vaccine it must not be given to anyone with untreated cancer or altered immunity, those receiving immunosuppressive or Xray therapy or high dose steroids. It must not be given within three weeks of another live vaccine but the recommendation is that it may be given at the same time. It is cultured on chick embryo so severe egg allergy is a contraindication. It contains neomycin and kanamycin and should not be given to those with allergies to these antibiotics. It is only currently licensed in the UK as a triple vaccine along with mumps and rubella vaccine *(5)*.

Measles disease may depress cell mediated immunity for up to three years *(6)*. The vaccine virus is attenuated but has similar characteristics to the wild virus so it would be expected to have the same characteristics. Indeed a high titre measles vaccine (*high dose Edmonston-Zagreb)* used in populations in Africa caused higher death rates in girls from other infectious diseases compared to boys or unvaccinated girls. To give a vaccine that has such an effect on the immune system at the same time and in the same needle as two other live viruses is, in my opinion, risky. (*6a*)

99% of the reduction in deaths due to measles in England & Wales occurred before the introduction of the measles vaccine in 1968 and has continued to fall since then.

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk



Measles outbreaks in unimmunised people tend to be mild in those who do not have underlying medical conditions. In developing countries, measles in 'secondary' cases (those infected by someone living in the house) are generally more severe than in primary cases (those infected by someone out of the house) but in well nourished individuals in the USA these cases are usually milder. In communities which generally do not immunise, the attack rate in infants less than one year of age is low because of protection by the superior maternal antibodies derived from natural infection as opposed to the vaccine *(7-9)*. Vaccinated people develop lower measles antibody titres than people contracting natural measles infection. This means that vaccinated mothers transfer smaller amounts of measles antibody across their placenta to their baby, consequently, measles antibody levels in these infants fall below protective levels at an earlier age than they do in the infants of mothers who have had natural infection *(10,10a)*.

Almost without exception, deaths occur in those with underlying medical conditions or poor nutrition or in those religious groups who refuse timely medical care when complications occur *(11,12)*. Those most at risk of complications from the disease are also those least likely to produce a good antibody response from being given the vaccine.

Vaccinating against measles in the USA since 1963 increased the attack rate in young children (less than 15 months) and pushed what was a childhood disease into being a disease of older school children. In some school outbreaks 74% of cases had had a single dose of measles vaccine. This prompted the recommendation of a second dose in 1989 which was adopted by virtually all states by 1992 *(10)*. In 1994 26% of all cases of measles were in people over the age of 20 years *(14)*. A study in high school children after a measles outbreak in a fully vaccinated school population showed that in those students with low levels of measles antibody, a booster dose of measles vaccine was not very successful in increasing the antibody levels. This lead to the observation that, "It appears possible that only recently have large cohorts of students reached an age when the decay of immunity induced only by vaccine and not augmented by natural exposure could reach levels sufficient for outbreaks to occur. Our results

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

indicate that a booster vaccine dose had little effect on this student population's measles antibody response *(15)*.

When a major upsurge of measles occurred in the USA in 1989 to 1990, pre-school populations made up a higher proportion of cases. A large percentage were children less than 16 months old *(10)*. In the UK where the measles vaccine was introduced five years later than in the USA, by 1994 cases of measles in babies under 15 months of age were also rising. In an outbreak in Northern Ireland those below the age of 15 months had the highest attack rate 9.02/1000 population, at least three and a half times the attack rate in 16 month to 18 year olds *(16)*.

Measles vaccination was started in the Republic of Ireland in 1985, MMR vaccination in 1988. In the 2000 outbreak figures for the Eastern Regional Health Authority (Co Dublin, Wicklow, Kildare) 40% of the cases were below the age of 15 months. There were three deaths reported: a two year old child with a severe underlying medical condition (tracheo-oesophageal fistula with severe respiratory problems who was already in intensive care when he caught the measles; a 12 month old child who was severely under nourished and suffering from failure to thrive; and an apparently healthy 12 month old *(17)*.

In 1994 seven million children aged five to 16 years were vaccinated against measles and rubella. At the same time a specific salivary test for the diagnosis of measles was introduced. After the campaign the incidence of measles was said to have been dramatically reduced but this would have been the case if only the new specific salivary test had been introduced as doctors are notoriously poor at diagnosing acute spotty rashes. In the recent Irish epidemic only 430 out of 1500 cases were actually confirmed as measles *(17).* Despite the purported success of this campaign a second dose (pre-school) of MMR was introduced in October 1996 to the UK schedule.

The Department of Health's Immunisation Handbook 1996 states that, "Before 1988, more than half the acute measles deaths occurred in previously healthy children who had not been immunised." *(5)* This is very misleading. Its source is a paper by C Miller, Senior Epidemiologist at the Public Health Laboratory Service, London which looked at deaths from measles in England & Wales 1970-83. There were 270 deaths of which 126 (47%) were in people with severe pre-existing conditions such as cerebral palsy, mental retardation, Down's syndrome, various congenital abnormalities, immunodeficiency, immunosuppression or lymphatic leukaemia. Social conditions were not examined and no attempt was made to establish vaccination history (!!). As in most outbreaks about half or more of those with measles have been vaccinated it is rather misleading to imply that all the cases of measles were unvaccinated, also, most people would agree that there is a gradient between such severe medical conditions and 'health' *(24)*

Side Effects Of The MMR Vaccine Listed In The Data Sheet

Listed Side Effects of MMR Vaccine (MMRII Aventis Pasteur MSD Ltd 2000, Priorix MMR vaccine live Smithkline Beeccham 1997) are quite detailed and I quote extensively:

Common
Burning/stinging at site of injection

Occasional
Fever, skin rash which may be generalised usually appearing day 5-12 after vaccination, redness and swelling at the site of the injection

Rare
Sore throat, general feeling of being unwell
Swollen salivary gland, vomiting, diarrhoea
Swollen lymph nodes, low platelet count, purple bruising (thrombocytopenic purpura)
Allergic reactions, urticaria, anaphylaxis
Arthralgia, arthritis, usually transient, myalgia (muscle pain. In children this occurs in 0-3%, in women: 12-20%, also more marked and of longer duration, symptoms may persist for months or, rarely, for years)

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

Febrile convulsions in children, afebril convulsions or seizures, headache, dizziness, paraesthesia (tingling sensation), polyneuritis, Guillain-Barre syndrome, ataxia. Encephalitis / encephalopathy has been reported one per every three million doses compared to one per two thousand reported cases of measles
Erythema multiforme
Optic neuritis (blurred vison due to a swollen optic nerve), retrobulbar neuritis, papillitis, retinitis, ocular palsies, middle ear infection, nerve deafness, conjunctivitis
Swollen testicles
Subacute sclerosing panencephalitis (SSPE). There have been reports of SSPE in children who did not have a history of natural measles but did receive the vaccine. Some of these cases may have been due to unrecognised measles in the first year of or possibly from the measles vaccination. Based on the estimated measles vaccine distribution in the USA the association of SSPE to measles vaccination is about one case per three million vaccine doses distributed. The association with natural measles is 6-22 per million cases *(I do not know their source for this estimation)*

A report in the British Medical Journal from the Communicable Disease Unit at the London School of Hygiene and Tropical Medicine (1996) *(18)* stated that after the1994 measles rubella campaign there were 530 severe reactions reported, one per 13200 vaccinations and higher than the one per million usually quoted. One report of SSPE occurred one month after vaccination. The child had a history of natural measles infection some years earlier. A report from a review by expert committees on serious adverse events associated with measles and rubella vaccine concluded that there was a causal relationship established for: Measles :

Anaphylaxis (1 in 30 000 to 1 per million doses)
Thrombocytopenia (1 in 30 000-40 000)
Death from the measles vaccine strain in immunocompromised children

They noted a theoretical risk of death from anaphylaxis or thrombocytopenia but no direct evidence.

The data were inadequate to accept or reject a causal relation with:

Encephalopathy or encephalitis
SSPE
Residual seizure disorder
Sensorineural deafness
Optic neuritis
Transverse myelitis
Guillain-Barre syndrome
Insulin dependent diabetes mellitus

For the rubella vaccine currently in use (RA 27/3) there was evidence for a causal relation with:

Acute arthritis or arthropathy

And was consistent with:

Chronic arthritis in women

A study in France concluded that there is sufficient evidence of a clear temporal relationship between MMR vaccination and the occurrence of thrombocytopenic purpura to make a causal relationship highly plausible *(19)*.

In the Avon longitudinal study 27 children were hospitalised for febrile convulsion after MMR vaccination compared with an expected background rate of 16. Cases peaked at two weeks after vaccination with 14 admissions compared with an expected four *(20)*.

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

I have specifically not mentioned autism in relation to the MMR vaccination. Much has been said on the matter and it is the subject of a large pending legal case. Many of the arguments as to whether autism has increased since the introduction of the MMR vaccine rely on sophisticated statistical re-analyses of studies that were not set up to look specifically at whether there is a link or not. Autism, however, was not described as a disease until the 1950s when major vaccination programmes were started and as these have become more universal, so has autism. If there is a link between MMR and autism it is certainly not the only vaccine which might be a culprit.

Vaccination recommendation

Bearing in mind that the death rate from measles had decreased by 99% before the introduction of the vaccine in the UK, that measles is generally a mild disease in a healthy child who is nursed appropriately and that vaccination just seems to put off having measles until a later age when it can be more serious, I think that the most sensible course of action is to try and get the disease. It is certainly circulating, that is why the booster has been introduced and there are likely to be more.

Having measles with a typical rash is associated with a lower incidence of the development of immunoreactive diseases, sebaceous skin diseases, diseases of bone, cartilage and certain tumours in adult life *(21)*, unlike the more atypical variety that more often occurs after vaccination.

In the Steiner community in Gloucester where an outbreak occurred in 1997-8 there were no severe cases and 62% of the respondents to a questionnaire reported a strengthening and maturing of their child both mentally and physically after measles infection. Dr Duffell from Gloucestershire health authority remarked that the mildness of the measles cases, similar to previous studies in the UK, “supports the notion that measles is not a severe illness in most children.” He points out that the cases were in, “fit, well nourished children from a community that advocates a healthy lifestyle.” *(22)*

I think sometimes that people have been so frightened by the medical profession’s descriptions of the worst complications of the disease, in an effort to encourage vaccination, that they have forgotten that it is generally a childhood illness that most children come through with flying colours.

If parents are prepared to nurse their children through this illness I would think that it is safe not to vaccinate the children and to allow them to have the natural disease. There are enough well documented adverse reactions to measles, mumps and rubella vaccines, alone or combined as to make them undesirable for the prevention of such generally benign disease.

References

(1) Patterson’s sick children 9th Ed, 1971 Balliere, Tindall & Cassell, London.
(2) Hussey G, Managing measles, BMJ 1997;314:316-7
(3) Glaziou PP, Mackerras DEM, Vitamin A supplementation in infectious diseases: a meta-analysis, BMJ 1993;306:366-70
(4) Daulaire MPD, Starbuck ES, Houston RM, Church MS et al, Childhood mortality after a high dose of vitamin A in a high risk population, BMJ 1992;304:207-10
(5) Immunisation against infectious diseases HMSO London 1996
(6) Shaheen SO, Aaby P, Hall AJ, Barker DJP et al, Cell mediated immunity after measles in Guinea-Bissau: historical cohort study, BMJ 1996; 313:969-74 (6a) Aaby P et al ‘Long-term survival after Edmonston-Zagreb measles vaccination in Guinea-Bissau: Increased female mortality rate’ The Journal of Pediatrics 1993;122:904-8.
(7) Sutter RW, Markowitz LE, Bennetch JM, Morris W et al, Measles among the Amish: a comparative study of measles severity in primary and secondary cases in households, J Infectious Diseases 1991;163:12-16
(8) Outbreak of measles in a religious group - Montreal, Quebec, Canada Communicable Disease Report 1995 ;1:1-5
(9) Lennon JL, Black FL, Maternally derived measles immunity in era of vaccine-protected mothers, J Pediatrics 1986;671-6
(10) Hutchins S, Markowitz L, Atkinson W, Swint E, Hadler S, Measles outbreaks in the United States 1987 through 1990 Padiatr Infect Dis J 1996;15:31-8
(11) (10a) Cutts FT, SteinglassR, Should measles be eradicated? BMJ 1998;316:765-7
(12) Novotny T, Jennings CE, Doran M, March RC et al, Measles outbreaks in religious groups exempt from immunization laws, Public Health Reports 1988;103:49-54
(13) Rodgers DV, Gindler JS, Atkinson WL, Markowitz LE, High attack rate and case fatality during a measles outbreak in groups with religious exemption to vaccination, Pediatric Infectious Disease Journal 1993;12:288-92
(14) Sloan DSG, Young adults susceptible to measles, BMJ 1996;310:1271
(15) Matson DO, Byington C, Canfield M, Albrecht P, Feigin RD, Investigation of a measles outbreak in a fully vaccinated school population including serum studies before and after revaccination, Pediatr Infect Dis J, 1993;12:292-9
(16) Tohani VK, Farrell B, Little importance placed on infants aged under 15 months, BMJ 1995;310:192

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

(17) Cronin M, Irish Department of Health, 2001 personal communication
(18) Cutts FT, Revaccination against measles and rubella, BMJ 1996;312:589-90l
(19) Jonville-Bera AP, Autret E, Galy-Eyraud C, Hessel L, Thrombocytopenic purpura after MMR vaccination: a retrospective survey by the French Regional Pharmacovigilance Centres and Pasteur-Merieux Serums et Vaccins, Pediatric Infectious Disease journal 1996;15:44-48
(20) MMR-linked febrile convulsions 'more common than expected' Pulse 27 Oct 2001
(21) Ronne T, Measles virus infection without rash in childhood is related to disease in adult life, Lancet 198:1-5
(22) Duffell E, Attitudes of parents towards measles and immunisation after a measles outbreak in an anthroposophical community, J Epidemiological Community Health 2001;55:685-6
(23) The Role of Medicine, Thomas McKeown, Princeton University Press 1979:105
(24) Miller CL, Deaths from measles in England and Wales, 1970-83, BMJ 1985;290:443-4

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

MUMPS

Mumps disease is caused by a virus. Humans are the only known natural host. Subclinical infections (with no obvious symptoms) are common, at least 25%. The peak age of incidence is five to nine years. One attack of clinical or subclinical mumps confers lasting immunity and second attacks are most unusual. *(1)*

The incubation period is an average of 18 days. The disease starts with pain and swelling in the region of one parotid gland (salivary gland in front of the ear) and fever. Neck glands and those under the tongue may be involved. After four to five days the glands on the other side may become involved as the swelling on the first side goes down. In more severe cases the child will be more ill with a high fever, dirty tongue and able only to drink fluids. In most patients the chief complaints refer to difficulty in eating, swallowing and talking. The disease usually resolves in 10 to 14 days and there is complete recovery as a rule. *(1,2)*

Treatment is supportive, bed rest when there is a fever and plenty of fluids which may be drunk through a straw if it hurts to move the jaw. *(2)*

The commonest complication is swelling of the testicles and this is more common after the age of puberty (20-25% of such cases). The swelling is generally unilateral but if it occurs bilaterally low sperm counts or sterility may ensue. There may be swelling of the ovaries in girls but it does not result in sterility *(1)*. In fact it is thought that having mumps with recognisable parotid swelling has a protective value against getting ovarian cancer in later years *(3)*. This is clearly desirable as ovarian malignancies generally have a very poor prognosis due to their being diagnosed late.

Mumps meningo-encephalitis is uncommon but is the most important complication seen in childhood. Headache, vomiting and sometimes convulsions are accompanied by neck stiffness and occasionally focal signs of paralysis. Mumps meningitis requires no specific treatment although lumbar puncture provides relief from the intense headache and the outlook is usually excellent *(2)*. Rarely, deafness can occur *(1)*.

In 1992 the MMR containing the Urabe strain of mumps virus was withdrawn in the UK because it caused mumps meningitis *(4)*. The vaccine manufacturers then sold this same vaccine to South America for their MMR vaccination campaign causing a predictable epidemic of mumps meningitis. When challenged as to why vaccine manufacturers would do such a thing if they had the best interests of children at heart, Dr Mike Watson, speaker for the UK Vaccine Manufacturers Group said that the mumps meningitis was, “only a bad headache and they all recovered.” *(5)* Yet the (small) risk of mumps meningitis associated with the disease is the main the reason that GPs pressure parents into having their children vaccinated against mumps.

The Vaccine

The vaccine is a live attenuated virus grown on chick embryo. The MMR vaccine used in the UK contains the Jeryl Lynn strain. The contraindications are the same as for live measles vaccine. It was added to the UK schedule in 1988 in the triple MMR vaccine.

By 2000, cases of mumps were steadily rising, increasing by 30% per year from 1999. In some place such as Leeds and Bradford there were increases of nine times and 30 times the incidence between 2000 and 2001 *(6)*. One third of those affected were aged over 15, just the time when boys are likely to be made infertile. In Northern Ireland 95% of confirmed cases were in nine to 19 years *(7)*. In Stockport the mumps virus identified from several cases was G6 genotype. The Jeryl Lynn mumps vaccine used in the UK has genotype A. The Public Health Laboratory Service advises that cross protection from the strains should be sufficient, but four of the confirmed cases in Stockport had received two doses of MMR. There is possibility that immunisation against mumps is causing a mutant strain to emerge with limited or no cross protection from the vaccine strain (cf pertussis) *(8)*.

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

As with measles the tendency is for mumps vaccination to push the disease incidence into higher age ranges which, in the case of mumps, is certainly more serious in terms of the side effects in boys. As yet there is no reported tendency for mumps to occur in very young children.

Data sheet listed side effects of the MMR vaccine are as above.

Vaccination Recommendation

Mumps is generally a mild illness. Those most at risk of complications are post pubertal males. In females there may be a distinct benefit in having clinical measles in terms of a protective effect against ovarian cancer. I do not recommend mumps vaccination as any benefit is minimal and any side-effects unacceptable.

References

(1) Harrison's Principles of Internal Medicine 11th Ed, McGraw-Hill Inc 1987
(2) Patterson's sick children 9th Ed, 1971 Balliere, Tindall & Cassell, London
(3) West RO, Epidemiologic study of malignancies of the ovaries, Cancer 1966;19:1001-7
(4) Immunisation against infectious diseases HMSO London 1996
(5) You & Yours, Radio IV, 11 Aug 2000
(6) 'Vaccinate' plea as mumps cases soar, Waites M, Yorkshire Post 30 Aug 2001
(7) Mumps outbreak shows need for MMR booster, Pulse 1 Dec 2001
(8) Baxter D, Prepare for resurgence of mumps infection, Pulse 2 Jun 2001:50-60

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

RUBELLA

Rubella was once most frequent among children aged five to nine years, but with the advent of immunisation programs often directed primarily at this group as well as pre-schoolers, a greater proportion of cases is now being reported among older people (15-24 years) *(1)*

Rubella is one of the mildest of the infectious fevers and its importance arises mainly from the 'rubella syndrome' whereby mothers who have had rubella during the first three months of pregnancy and less frequently during the second trimester are likely give birth to babies with multiple congenital abnormalities *(2)*.

The incubation period is ten to 21 days. It is apparent from serological studies that 25 to 50 per cent of infections are subclinical (no symptoms) or may only cause swollen lymph glands with no rash. The time from exposure to the appearance of a rash is 14 to 21 days. Children usually have no symptoms during this period, adults may feel unwell with cold-like symptoms. The rash appears on the forehead and face and then spreads down the trunk to the extremities. It is very easy to confuse the rubella rash with other spotty viral rashes of childhood. The rash generally lasts three days and may have disappeared in less than 24 hours. In adults, especially young women painful joints with swelling (arthralgia, arthritis) may occur. Very rarely there may be lowered platelets (thrombocytopenia) with purpura (purple coloured bruising) *(1,2)*.

Treatment is symptomatic and one attack confers immunity in 90 per cent of cases*(2)*.

The Vaccine

The rubella vaccine contains a live attenuated virus of the Wistar RA27/3 strain. It is grown on diploid cells of human origin. The contraindications are the same as for the measles and mumps vaccines except that egg allergy is not thought to be an issue. It contains neomycin and should not be given to those with allergies to this antibiotic. An absolute contraindication of rubella vaccine is pregnancy and women of childbearing age should avoid pregnancy for one month from the time of vaccination *(3)*.

Listed side effects of the vaccine *(Erevax Rubella Vaccine , Live PhEur (RA27/3 strain SmithKline Beecham 1998)* include getting a mild non-infectious case of rubella a few days after injection of the vaccine with rash, fever, swollen glands and swollen or painful joints with or without joint effusion. Extremely rarely, transient polyneuropathy (altered sensation, tingling or muscle weakness) or thrombocytopenic purpura (low platelets causing purple bruising). Such side effects tend to be more marked in adults than in children. symptoms when they do occur usually begin one to three weeks after vaccination and are normally transient. Some people have an acute anaphylactic reaction to the vaccine.

In the five years before the rubella vaccine was introduced in 1970 there were only 39 babies born with congenital rubella. In the ten years after 1970 there were 454 case. Even assuming that the 14 year old vaccinated in 1970 did not start to have babies until they were 24 (unlikely), in the ten years after 1980 there were still 333 affected babies. So the number of cases have gone up. It was only in the ten years after 1990 that the number of cases went down to 46 (44 live born, two stillborn) of whom the majority were born to mothers from abroad or who acquired the disease abroad. 12 of the affected babies were born in 1996. The mothers of eight of them were born and raised in the UK and would have been eligible for the schoolgirl immunisation programme (ie they had been vaccinated *(4)*. Indeed a video produced by SENSE (The National Deaf-Blind and Rubella Association) tells the tragic story of a child with multiple disabilities from rubella syndrome who was born to a mother who was vaccinated in the schoolgirl immunisation programme but became infected with rubella when she was pregnant because the immunity had worn off.

> *The experience in the USA is similar. "Since licensure of rubella vaccine in1969, policy in the United tates has emphasized immunization of pre-pubertal children beginning at 12-15 months of age. Although this approach has resulted in a 70 per cent reduction in rates of reported rubella, most of the reduction has occurred in children younger than 15 years of age, with little change in the attack rate for persons 15 years and older. Over 70 per cent of all reported cases now occur*

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

> *in this age group. The 10 to 25 per cent prevalence of susceptibility to rubella among adolescents and young adults has not changed since the introduction of rubella vaccines." (5)*

Vaccinating 12 to 15 month olds with rubella and again preschool almost guarantees that their antibodies to rubella will have worn off by the time they are likely to become pregnant. The hope that the additional dose of rubella in the preschool MMR will protect the only group of people who need protecting against rubella – pregnant women – is entirely speculative.

Vaccination Recommendation

Vaccination cannot be recommended to avoid the effects of rubella disease as they are so minor. Even though contracting the disease in adulthood can produce more symptoms – arthralgia/ arthritis, the same can be said for the vaccine which also produces more side-effects at that time, especially in young women.

To protect unborn babies from rubella syndrome in women who are not immune, it is possible to see that vaccination does not necessarily stop this occurring. In fact there are likely to be more cases occurring as vaccination is now given at a much younger age so it is likely to wear off sooner – even with two doses. It would be far preferable for girls to get rubella disease and have the better quality antibodies associated with natural infection so that in their own pregnancies they will not catch rubella for the first time. When they reach childbearing age they can have their rubella antibodies checked. If they do not have antibodies to rubella at that time, then rubella vaccination could be considered.

To argue that boys and girls should be vaccinated against rubella to stop susceptible women who have not had natural rubella infection from having a baby with rubella syndrome is not reasonable when there are side effects associated with the vaccine, the disease is more troublesome when older, babies with rubella syndrome still occur, and vaccination compromises the future immune status of girls when they have their own future pregnancies.

References

(1) Harrison's Principles of Internal Medicine 11th Ed, McGraw-Hill Inc 1987
(2) Patterson's sick children 9th Ed, 1971 Balliere, Tindall & Cassell, London.
(3) Immunisation against infectious diseases HMSO London 1996
(4) Tookey P, Peckham C, Miller E, Congenital rubella RCPCH BPSU 14th Annual Report 1999-2000 Sept 2000
(5) Polk, BF et al An outbreak of rubella among hospital personnel NEJM 1980;303:541-5

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

GENERAL CONTRAINDICATIONS TO VACCINES

Vaccines should not be given to people suffering from an acute illness, particularly with fever.

Vaccines should not be given to individuals who have a definite history of a severe local or general reaction to a preceding dose.

Local: an extensive area of redness and swelling which becomes hard and involves most of the front and side of the thigh or most of the circumference of the upper arm.
General: fever equal to or more than 39.5'C within 48 hours of the vaccine, anaphylaxis, bronchospasm, laryngeal oedema, generalised collapse, prolonged inconsolable or high pitched screaming for more than four hours, convulsions or encephalopathy occurring within 72 hours *(1)*.

Reference

(1) Immunisation against infectious diseases HMSO London 1996

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

HOW VACCINES WORK

Vaccines are intended to induce protective antibody formation by mimicing natural infection. However, the antibodies produced by vaccination (IgG) are only the final stage of a long series of protective measures by which a child deals with infections. When a child first meets an infectious agent they are protected by a skin barrier, unless it is breached. Organisms entering by the gut have to pass one molar hydrochloric acid in the stomach, those entering via the respiratory tract have to get past the mucus barrier. Having gone past these, the organism then has to contend with a secretory immunoglobulin, IgA, which is produced by the linings of the gut and airways (important in 'mucosal immunity'). Coated with IgA it then reaches the blood stream where is can stimulate an IgG response.

In the vaccination process, except in the case of oral polio vaccine, all these initial stages are missed and the attenuated organism or toxin is injected directly into the child, bypassing all the first stage defences and presenting itself 'naked' to the immune system, the very immature immune system. It is not surprising that problems occur. Only IgG antibodies are induced and mucosal immunity is not stimulated except in the case of oral polio vaccine.

Standard dietary advice for babies is that they should not be given unmodified milk products, nuts, citrus, wheat or salt before the age of six months because of the potential for inducing allergy and because the kidneys are immature. Yet when vaccinated they are injected with organisms of a different type and dose and via a different route to that which they are likely to meet naturally. Along with the organisms/toxoids are additives such as aluminium, formaldehyde and thiomersal (which contains).

Vaccination aims to produce antibodies, however, antibodies are not the same as immunity. This was emphasised with the mumps vaccine in Switzerland in the 1990s. Three mumps vaccines – *Rubini, Jeryl-Lyn* and *Urabe* (withdrawn in the UK in 1992 because it was caused mumps meningitis) all produced excellent antibody levels but those vaccinated with the *Rubini* strain had a higher attack rate than those not vaccinated at all *(1)*. Dr David Elliman, District Immunisation Coordinator for Merton, Sutton and Wandsworth health authority and Consultant in Community Child Health at St Georges Hospital London, says that it actually gave people mumps *(2)*.

References

(1) Schlegel M, Osterwalder JJ, Galeazzi RL, Vernazza PL, Comparative efficacy of three mumps vaccines during disease outbreak in eastern Switzerland: cohort study, BMJ 1999;319:352-3
(2) Elliman D, personal communication.

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

ADDITIVES

Ethyl mercury in thiomersal has been used in vaccines for 60 years. The 1999 product information for the adult diphtheria vaccine (*Secretary of State for Health, Department of Health*) states that it can cause kidney damage. In May 2002 pregnant women, babies and children under the age of 16 years were advised to stop eating shark, marlin and swordfish as a precautionary measure because high levels of mercury that have been found in these fish. The risk was said to be highest in babies in utero as mercury can damage the developing nervous system. In children possible effects on the developing nervous system might lead to impaired mental skills, such as attention and memory, and physical incoordination in childhood. The letter from the Deputy Chief medical Officer said that mercury in surface water was being changed by bacteria into the more toxic form of methyl mercury which was absorbed by the fish *(1)*.
Ethyl mercury is injected into babies in many of the childhood vaccines. Could this be one of the reasons for the marked prevalence of dyslexia among children today? Concerns about using such a toxic ingredient in vaccines were dismissed for years but after official US data raised the possibility of a link with developmental delay in 1999, the USA has moved to a thiomersal free vaccine schedule. By 2001, steps were eventually being take to remove it from vaccines in the UK (but only after all the old stocks have been used up) *(2)*.
Ingestion of aluminium is linked to Alzheimer's disease to the extent that many people have replaced their aluminium saucepans with stainless steel. People drinking aluminium contaminated drinking water in Camelford, Cornwall suffered considerable damage to cerebral function *(3).* Yet aluminium salts are injected into very young babies as part of the vaccination programme

Formaldehyde is used for preserving cadavers and is carcinogenic. It is present in diphtheria and tetanus vaccines.

References

(1) Message from Dr Sheila Adam, Deputy CMO, Dept of Health, CEM/CMO/2002/6 14 May 20002
(2) Vaccines and brain disorders probed, Pulse 30 June 2001
(3) Altman P, Cunningham J, Dhanesha U, Ballard M et al, Disturbance of cerebral function in people exposed to drinking water contaminates with aluminium sulphate; retrospective study of the Camelford water incident, BMJ 1999;319:807-11

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

FACTORS AFFECTING IMMUNITY

There are many factors other than vaccines and medicines which influence the acquisition and the outcome of infectious diseases. To build up a child's immune system they need a good diet, clean water, fresh air, exercise, sleep and love.

Adequate dietary or supplementary vitamin A has a major role in preventing morbidity and mortality in children in developing countries *(1)*.

A nutrition improvement project based on home garden production and nutrition information in preschool children in Vietnam reduced the incidence of respiratory infection including pneumonia and severe pneumonia, also diarrhoeal diseases *(2)*.

Children who had rheumatic fever in Bangladesh had lower serum albumin concentrations and body iron stores compared with those who did not *(3)*.

Children living in improved housing – fired mud bricks, tile roofing, concrete foundation and a pit latrine – in northern Malawi were less likely to have respiratory, gastrointestinal or malarial illnesses *(4)*.

These are similar to the improvements made by the Victorians in the nineteenth century in this country. Public health acts provided a basis for improved sanitation – clean water supplies and separate disposal of sewage (we are still using their drains now), new buildings' standards and slum clearance. Streets were widened, sewers were walled in, railways were built bringing fruit and vegetables into urban centres.

This led to a decline in deaths from scarlet fever, rheumatic fever (for which there are no vaccines), tuberculosis, whooping cough and measles. 99 per cent of the decline in mortality from whooping cough and measles was achieved before vaccination against them was introduced in the 1950s and 1968 respectively. Countries that do not use the BCG vaccine against tuberculosis such as the USA and Denmark have the same decline in deaths as those that do.

Critics of the 'better social conditions' theory of improved health point to the outbreaks of poliomyelitis in the 1950s and the recent resurgence of diphtheria in the former Soviet Union. Infantile paralysis, as polio was originally described, began to emerge as an important new disease in the 1920s. It often affected the limb which had been vaccinated against smallpox. Outbreaks in the 1950s were associated with the vaccination campaigns against diphtheria and whooping cough. The large numbers of cases of diphtheria in the former Soviet Union are in vaccinated adults, not in children who have not been vaccinated because of interrupted vaccine supplies. This is due in a large part to poorer social conditions, diet, job uncertainty and increased alcohol consumption since the break-up of Communism. When diseases are caused by adverse social conditions vaccines do not help.

Passive immunity passed by the mother to the fetus transplacentally is very important in protecting the child during its first year. Babies of vaccinated mothers do not receive such good antibodies. This is because antibodies from vaccination are of inferior quality and are not so long lasting as those acquired from natural infection (*see measles) (5)*. This is why there are increasing cases of whooping cough and measles in babies below the age of one year.

In Guinea-Bissau children aged 12-35 months who were not breast fed had a 3.5 times higher death rate than breast fed children and the beneficial effects of breast feeding were not restricted to infancy *(6)*.

References

(1) Glasziou PP, Mackerras DEM, Vitamin A supplementation in infectious diseases: a meta-analysis, BMJ 1992;306:366-7
(2) English RM, Badcock JC, Giay T, Ngu T, Waters AM, Bennett SA, Effect of nutrition improvement project on morbidity from infectious diseases in preschool children in Vietnam: comparison with control commune, BMJ 1997;315:112-5
(3) Zaman MM, Yoshiike N, Rouf MA, Haque S et al, Association of rheumatic fever with serum albumin concentration and body iron stores in Bangladeshi children: case control study, BMJ 1998;317:1287-8
(4) Wolff CG, Schroeder DG, Young MW, Effect of improved housing on illness in children under 5 years old in northern Malawi: a cross sectional study, BMJ 2001;322:1209-12.

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

(5) Lennon JL, Black FL, Maternally derived measles immunity in era of vaccine-protected mothers, J Pediatrics 1986;671-6 Hutchins S, Markowitz L, Atkinson W, Swint E, Hadler S, Measles outbreaks in the United States 1987 through 1990 Padiatr Infect Dis J 1996;15:31-8
(6) Molbak K, Grottschau A, Aaby P, Holjyng N et al, Prolongued breast feeding, diarrhoeal disease, and survival of children in Guinea-Bissau, BMJ 1994;308:1403

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

ARE CHILDHOOD INFECTIOUS DISEASES A GOOD THING?

Adequate exposure to microbes early in life is an important factor in the maturation of the immune system. As the human immune system has evolved under the selection pressure of infectious diseases it stands to reason that if they are removed, then problems may occur. Hence the old clinical adage, "autoimmunity is the price paid for eradicating infectious diseases." *(1)* There has been a noticeable rise in asthma, eczema and more severe autoimmune diseases in the last few decades.

A study in the Oxford region on the incidence of insulin dependent diabetes mellitus in children (1985-96) showed a rise in the incidence in under four year old children of 11 per cent per year *(2)*.
Italian recruits with antibodies for hepatitis A (regarded as a marker for exposure to 'dirt') had a lower incidence or atopic disease, asthma and eczema *(3)*.

A study in West Germany found a lower risk of developing asthma in children who had repeated viral infections, other than lower respiratory infections, early in life *(4)*.

References

(1) Wilson AG, Duff GW, Genetic traits in common diseases, BMJ 1995; 310:1482-3
(2) Garnder SG, Bingley PJ, Sawtell PA, Weeks S, Gale EAM, Rising incidence of insulin dependent diabetes in children aged under 5 years in the Oxford region: time trend analysis, BMJ 1997;315:713-7
(3) Matricardi PM, Rosmini F, Ferrigno L, Nisini MA st al, Cross sectional retrospective study of prevalence of atopy among Italian military students with antibodies against hepatits A virus, BMJ 1997;314:999-1003
(4) Illi S. von Mutius E, Lau S, Bergmann R et al, Early childhood infectious diseases and the development of asthma up to school age: a birth cohort study, BMJ 2001;322:390-5

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

TREATMENT OF CHILDHOOD INFECTIOUS DISEASES

It is very important that when children have their infectious disease that they are treated supportively and not suppressively. It has become widely recognised over the last few years that over prescribing of antibiotics leads to more chronic and repeated infections in children.

It is less widely recognised that high consumption of paracetamol is not desirable either. Recent studies have suggested that this may be linked to the development of asthma *(1)*.
Children who are ill need rest, fresh air, a moderate ambient temperature, loose clothes made of natural fibres, plenty of fluids and a light diet if hungry. I think they should not be sent back to school until 48 hours after their temperature has returned to normal.

Reference

(1) Toma T, High paracetamol intake may be linked with asthma, BMJ 2000;321:117

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk

VACCINE SAFETY

I quote extensively from an important paper published by the World health Organisation:

"As more and more infectious agents become targets for immunization programmes, the spectrum of adverse events linked to vaccines has been widening. Although some of these links are tenuous, relatively little is known about the immunopathogenesis of even the best characterized vaccine-associated adverse events (VAAEs). The range of possible use of active immunization is expanding...less virulent pathogens e.g. varicella (chicken pox), rotavirus in the developed world are also being targeted, and vaccine use is being justified in terms of societal and parental "costs," rather than in straightforward morbidity and mortality costs...In the developed world the pediatric immunization schedule is becoming crowded, with pressure to administer increasing numbers of antigens simultaneously in ever simpler forms...this trend while attractive in many ways brings hypothetical risks e.g. genetic restriction, narrowed shield of protection, and loss of randomness, which will need to be evaluated and monitored. The available epidemiological and laboratory tools to address the issues outlined above are somewhat limited. As immunological and genetic tools improve in the years ahead, it is likely that we shall be able to explain the immunopathogenesis of many VAAEs and perhaps even anticipate and avoid some of them. However, this will only happen if the human and financial resources needed for monitoring and studying vaccine safety stay in step with the accelerating pace of vaccine development. Failure to make such a commitment would put all immunization programmes at risk."

We know what needs to be done but will anyone do it?

Reference

(1) Ward BJ, Vaccine adverse events in the new millennium: is there reason for concern? Bulletin of the World Health Organisation 2000,78 2:205-15

Dr Jayne LM DONEGAN MBBS DRCOG DCH DFFP MRCGP MFHom 2004

Telephone/Fax 0044 (0)20 8632 1634
Email: jaynelmdonegan@yahoo.com Website: www.jayne-donegan.co.uk