A critique of the 16-page Australian pro-vaccination booklet entitled "The Science of Immunisation: Questions and Answers".*[1]

By Dr Viera Scheibner (PhD)

We can believe what we choose, we are answerable for what we choose to believe.
~Blessed John Henry Newman 1801-1890.

The science of immunisation?

Science simply means an organised system of knowledge. It does not mean infallibility, superiority, or eternal validity. and it is subject to changes and revisions. Many a crime has been committed in the name of science, starting with the medieval inquisition, through Nazi Germany's perversion of just about everything, including medicine, and now, continuing into the present day with harmful excesses of medicine.

Legalities of vaccination in Australia.

Australians must have their children immunised to receive certain payments?

Vaccination is non-mandatory in Australia. Parents have a constitutional and legal right to opt for natural immunity, which is achieved by contracting the natural infectious diseases of childhood. This is defined in the New tax system (Family Assistance) Act 1999 which stipulates that a child is considered immunised if it was administered a vaccine, developed natural immunity by contracting the alleged 'vaccine-preventable' diseases, or the parents have declared in writing a conscientious objection to the child being vaccinated.

Accordingly, GPs (General Practitioners) have a legal duty of care to sign the forms subsequent to a discussion in which they point out the benefits

[1] Published by the Australian Academy of Science Canberra November 2012; ISBN 978-0-85847-334-8. www.science.org.au/immunisation.html

and risks of vaccination, irrespective of their own views. A practitioner's refusal to sign the forms is a blatant disregard for what the law recognises to be a constitutional right of every person to refuse a medical procedure.

Needless to say, vaccination being a medical procedure, which admittedly carries the risk of serious reactions including death, should never be enforced or made compulsory. As shown elsewhere in this booklet, the Australian Immunisation Handbook 2009, 9th edition, (appendix 6) lists four full pages of observed serious reactions, including death. It also lists a great number of ingredients in vaccines, which include aluminium hydroxide, aluminium phosphate, borax, egg protein, formaldehyde, gelatin, gentamycin, kanamycin, monosodium glutamate, neomycin, phenol-xyethanol, polymixin, thiomersal and yeast, which by themselves are toxic, especially when injected (Ibid., appendix 4).

Former federal health minister Dr Michael Wooldridge wanted to mandate vaccination, but lawyers warned him about legal and constitutional obstacles. He therefore introduced the Family Assistance Act 1999, which misleadingly uses the term "conscientious objection" in reference to a non-mandatory medical procedure. This leads many financially struggling families, who are not informed about their legal options or known vaccination risks, to assume that vaccination is their only option.

This false impression was recently promulgated by most media in Australia who advertised the latest changes to the Family Tax system, and hence could leave vulnerable financially stressed families with the assumption that they now must vaccinate their children in order to get the new Family tax benefits.

One could reasonably expect that particularly those politicians who are lawyers by profession, which includes the Prime Minister Julia Gillard, should know that financially penalising parents for not taking up a non-mandatory medical procedure is illegal (not withstanding repulsive). It also discriminates against the poor. The rich, who do not depend on any family tax benefits, have an unencumbered freedom to exercise the constitutional and legal right not to vaccinate without any punitive measures. <u>One can also re-phrase it: being anti-vaccination is not illegal in Australia and hence cannot possibly carry any financial penalty.</u>

Moreover, Australia is among the few industrially-developed countries that have no legal avenue of compensating the victims of vaccine reactions and death.

The actions of the government are illegal. While Australian Constitution empowers the government to provide medical services, it goes no further, including any form of encouragement or worse,

compulsion. The legislation to "make sure" or even encourage "that children are immunised", is *ultra vires*.

Even if the government had such power, inseparable is the responsibility to provide full, honest, unbiased and accurate information on all its potential effects and to compensate all who have been injured by complying. Yet it has avoided, rejected, abrogated, and failed to embrace that responsibility.

"***Perceived power corrupts,***
absolute perceived power corrupts absolutely".[2]

The aim of the Australian Academy of Science (AAS) document is to clarify and summarise, for non-specialist readers, the current understanding of the "science of immunisation". The following six questions and answers are proposed.

- ◊ Question 1: What is immunisation?
- ◊ Question 2: What is a vaccine?
- ◊ Question 3: Who benefits from vaccines?
- ◊ Question 4: Are vaccines safe?
- ◊ Question 5: How are vaccines shown to be safe?
- ◊ Question 6: What does the future hold for vaccination?

Let's analyse the scientific factual quality and validity of this document's assertions.

In the following responses I provide documented facts, describing the reality of vaccines/vaccination, as published in reputable peer-reviewed medical journals.

[2] This quotation was from a letter from John Emerich Edward Dalberg Acton, first Baron Acton (1834-1902) to Bishop Mandell Creighton in 1887.

Question 1: What is immunisation?

The AAS document states that the purpose of vaccination is to prevent people from acquiring infectious diseases and to protect them against the associated short and long-term complications.

It also states that immunisation is the process whereby people are protected against an infection, while vaccination is the act of giving a vaccine to a person.

The documented effects of vaccines as shown by orthodox medical research:

♦ Sensitisation after vaccination.

At the turn of the twentieth century, medical researchers tested vaccines on themselves and other surgeons and medical students and established that vaccine injections result in the so-called negative phase of lowered bactericidal power of the blood, in other words, a measurable immune-suppression.[3]

Dr Parfentjev, an employee of Lederle Laboratories (one of many well-known vaccine manufacturers), reported that vaccination of mice with pertussis vaccine sensitised them, i.e. caused anaphylaxis (as opposed to prophylaxis), and increased their susceptibility to infection with several unrelated species of Gram negative bacteria and viruses.[4] Sensitisation (anaphylaxis) was achieved with injection of 15 billion cells of commercial pertussis vaccine. Compared with controls (normal, unvaccinated mice) the lethal dose of virus for sensitised mice was much smaller than for normal mice of the same age group. In other words, vaccination with pertussis vaccine increased the susceptibility of mice to lethal shock.

In another benchmark seminal work, Kind demonstrated that pertussis vaccines also sensitised the mice to the lethal effects of subsequent injections of pertussis vaccine as well as a variety of agents and conditions such as anaphylaxis, histamine, serotonin, and endotoxins, and certain proteins of "related" and "unrelated" organisms, such as Escherichia coli and Shigella dysenteriae.[5]

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[3] Wright AE. ON THE CHANGES EFFECTED BY ANTI-TYPHOID INOCULATION IN THE BACTERICIDAL POWER OF THE BLOOD; WITH REMARKS ON THE PROBABLE SIGNIFICANCE OF THESE CHANGES. Lancet, Volume 158, Issue 4072, Pages 715 - 723, 14 September 1901

[4] PARFENTJEV IA. Bacterial allergy increases susceptibility to influenza virus in mice. Proc Soc Exp Biol Med. 1955 Nov;90(2):373-5.

[5] KIND LS. Sensitivity of pertussis inoculated mice to endotoxin.. J Immunol. 1959 Jan;82(1):32-7.

Craighead reported that the same effects as observed in mice were also observed in children given inactivated microbial vaccines. He also wrote that during the past five years, significant advances were made in the understanding of the natural history of a number of common infections, among which he mentioned the apparent states of altered host reactivity consequent to vaccination.

> *Immunisation with inactivated vaccines could "sensitise" the recipients and result in an accentuated pattern of disease upon natural or experimental exposure.* [6]

Meaning, if a child did not react much to the first dose of vaccine, it may react seriously to subsequent doses.

Evidence for delayed hypersensitivity in recipients of "killed" vaccine is demonstrated by local skin reactions after the injection of live or inactivated microorganisms. The dermal response may also be caused by nonmicrobial constituents such as adjuvants and preservatives, which by themselves are highly toxic: aluminium and mercury compounds, formaldehyde, phenol, propylene glycol among others.

Modern immunological research regards vaccines as foreign antigens; indeed, vaccines represent superantigens, which are typified as multiple vaccines administered at the same session.

Earlier researchers have observed the many problems with antigenic stimulation by vaccines, such as the vaccine induced enhancement of viral infections which is known to occur with several vaccines.[7,8] This phenomenon was well described with the failed RSV (Respiratory Syncytial Virus) vaccines. However, as of 2009, scientists are still unsure of the exact mechanism.[9] As a result, vaccine development for lentivirus infections in general, and HIV/AIDS in particular, has been little successful.[10] Many trials of HIV vaccines, including the latest ones, confirmed this phenomenon: the trials had to be abandoned because a number of human volunteers contracted AIDS from the tested vaccines.

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[6] Craighead JE. Disease accentuation after immunisation with inactivated microbial vaccines. J Infect Dis. 1975 Jun;131(6):749-54.
[7] Ibid. Craighead.
[8] Huisman W, Martina BE, Rimmelzwaan GF, Gruters RA, Osterhaus AD. Vaccine-induced enhancement of viral infections. Vaccine. 2009 Jan 22;27(4):505-12. doi: 10.1016/j.vaccine.2008.10.087. Epub 2008 Nov 18.

[9] Varga SM. Fixing a failed vaccine. Nat Med. 2009 Jan;15(1):21-2. doi: 10.1038/nm0109-21.
[10] Ibid Huisman.

Equally unsuccessful are vaccines against bacterial infections such as whooping cough, diphtheria and Haemophilus influenzae as shown later in this critique.

Sabath documented antigen-induced transient hypersusceptibility to infections in mice and infants.[11] In mice they determined onset of infection and death due to influenza virus challenge at different times after antecedent monovalent influenza vaccine administration. In infants hospitalised for purulent meningitis there was a clustering of time intervals between routine vaccination and the onset of symptoms, proving the causal link.

Daum demonstrated a decline in serum antibody to the capsule of Haemophilus influenzae type b in the immediate postvaccination period in children.[12] They wrote that this increases the risk of invasive disease if it occurred during a period of asymptomatic colonization with H. influenzae type b, which, of course is a rule rather than an exception, because the bacterium is a ubiquitous commensal living on tonsils.

♦ **Effectiveness of vaccination.**

Outbreaks and epidemics of measles, whooping cough and poliomyelitis diseases in unvaccinated and fully vaccinated populations

The Amish are a religious community living across the USA that claim religious exemption to vaccination. Thus, the vast majority are not vaccinated.

They had not reported a single case of measles between 1970 and 1987.[13] At the same time, non-Amish highly-vaccinated communities still reported 2-3 year epidemics. Despite this obvious vaccination failure, pro-vaccinators claimed success with the measles vaccine.

In 1982, just when the US Secretary of State Joseph A. Califano Jr. planned to announce eradication of measles, the well-vaccinated non-Amish

[11] Sabath et al. Antigen-induced transient hypersusceptibility: a cause of sporadic fulminant infection in normals. 1989. Clin Research; 35(5): 617a

[12] Daum RS, Sood SK, Osterholm MT, Pramberg JC, Granoff PD et al.. Decline in serum antibody to the capsule of Haemophilus influenzae type b in the immediate postvaccinal period. J Pediatr. 1989 May;114(5):742-7.

[13] Sutter RW, Markowitz LE, Bennetch JM, Morris W, Zell ER, Preblud SR. Measles among the Amish: a comparative study of measles severity in primary and secondary cases in households. J Infect Dis. 1991 Jan;163(1):12-6.

populations started reporting huge outbreaks. The unvaccinated Amish did not have large epidemics of measles until much later, starting in early December 1987.

Outbreaks in the fully vaccinated American children continued with increasing frequency and severity. Without disclosing the vaccination status of children in measles epidemics, claiming victory over measles is just empty jabbering.

Moreover, vaccinated children started developing an especially vicious form of atypical measles. Fulginiti described the occurrence of atypical measles in children given formaldehyde-treated, aluminium precipitated measles vaccine, also referred to as "killed" measles.[14] He explained the problem as due to the altered immunological host response caused by vaccination.

Later on, when live-attenuated measles vaccine was introduced, the recipients starting developing atypical measles from it, as well.

Rauh and Schmidt described nine cases, which occurred in 1963 during a measles epidemic in Cincinnati.[15] The authors followed 386 children who had received three doses of killed measles virus vaccine in 1961. Of these 386 children, 125 had been exposed to measles and 54 developed it. The authors concluded that:

> *it is obvious that three injections of killed vaccine had not protected a large percentage of children against measles when exposed within a period of two-and a half years after immunization*.

Even when vaccination rates in the UK dropped in the 1990s and early 2000s, when confidence in the vaccine fell, measles deaths never exceeded 4 per year, and remain today at that level. Increasing laboratory-confirmed measles continue to occur in England and Wales even since vaccination rates have gone back to former levels. (Figure 1.)[16] This is evidence that measles vaccines at best interrupt transmission, but do not confer reliable immunity no matter how much of the herd is vaccinated.

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[14] Fulginiti VA, Eller JJ, Downie AW, Kempe CH. Altered reactivity to measles virus. Atypical measles in children previously immunized with inactivated measles virus vaccines. JAMA. 1967 Dec 18;202(12):1075-80.

[15] RAUH LW, SCHMIDT R. Measles immunization with killed virus vaccine. Am J Dis Child. 1965 Mar;109:232-7.

[16] Mellows-Facer A and Thompson G. Measles and MMR statistics - Commons Library Standard Note 17 February 2009 | Standard notes SN02581 http://www.parliament.uk/briefing-papers/SN02581 accessed 9 Feb 2013.

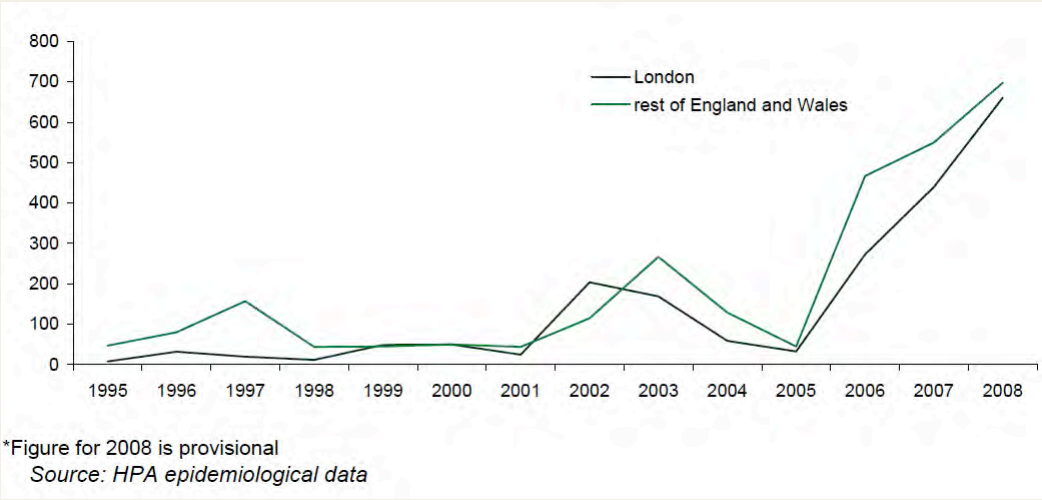

Figure 1: Laboratory confirmed measles cases, London and the rest of England and Wales, 1995-2008

Outbreaks of whooping cough (pertussis) in the vaccination era.

Right after the intense DPT vaccination that started in the mid 1970s, and right through the first decade of 2000, whooping cough outbreaks hit several US states, accompanied by similar outbreaks in all other countries that adopted intensive vaccination—including Australia.

In addition to pertussis (and measles) outbreaks in fully-vaccinated children, the outbreaks in the last thirty-odd years have been occurring increasingly in very young babies, born to mothers who were vaccinated when they were babies and as a result they lack transplacentally-transmitted immunity (TTI). Before the vaccine era, TTI protected babies and young children for up to two years against any infectious diseases of childhood.

Lennon and Black demonstrated that hemagglutinin-inhibiting and neutralizing antibody titers are lower in younger women who have been vaccinated than they are in older women.[17] The same applies to measles and pertussis.[18]

Breastfed infants of vaccinated mothers in the USA have nearly three times the risk of measles infection compared to those of naturally immune mothers, *even* in the era of vaccination when there is supposedly less measles virus in the environment.

[17] Lennon JL, Black FL. Maternally derived measles immunity in era of vaccine-protected mothers. J Pediatr. 1986 May;108(5 Pt 1):671-6.
[18] Mulholland K. Measles and pertussis in developing countries with good vaccine coverage. Lancet. 1995 Feb 4;345(8945):305-7.

> *Infants whose mothers were born after 1963 had a measles attack rate of 33%, compared to 12% for infants of older mothers.* ***Infants whose mothers were born after 1963 are more susceptible to measles than are infants of older mothers. An increasing proportion of infants born in the United States may be susceptible to measles****. . . the adjusted odds ratio for maternal year of birth (born after 1963) was 7.5 (95% confidence interval 1.8, 30.6).*[19]

This is most likely the result of lower levels of virus-specific immunity in the serum and milk in vaccinated mothers compared to naturally immune mothers. While the overall clinical case rate may have declined with measles vaccination, the most sensitive members of the herd are at an increased risk today- *because of vaccination*.

Hutchins et al. described pertussis epidemiology in the US. They wrote:

> *During the period 1980-1986, a total of 17,396 cases of pertussis were reported to CDC... The annual incidence of reported pertussis rose from 0.5 cases per 100,000 population to 1.7/100,000. Infants less than 12 months old had the highest average annual incidence... Children 1-4 years of age accounted for 25% of all cases but had an average annual incidence only 1/7th that of infants.* [20]

Figure 2[21] reveals a steady downward trend in the incidence and mortality from pertussis between 1922 and until about 1975-6; thereafter the downward trend in pertussis morbidity stopped and went sharply upwards, while pertussis mortality remained high but stationary. What could have caused such increase in the disease incidence seen in figure 3?

[19] Papania M. et al., Increased susceptibility to measles in infants in the United States, Pediatrics, November 1999, Vol. 1045, No. 5, e59, pp. 1-6.
[20] Hutchins SS, Cochi SL, Brink EW, Patriarca PA, Wassilak SG et al. Current epidemiology of pertussis in the United States. Tokai J Exp Clin Med. 1988;13 Suppl:103-9.
[21] Ibid.

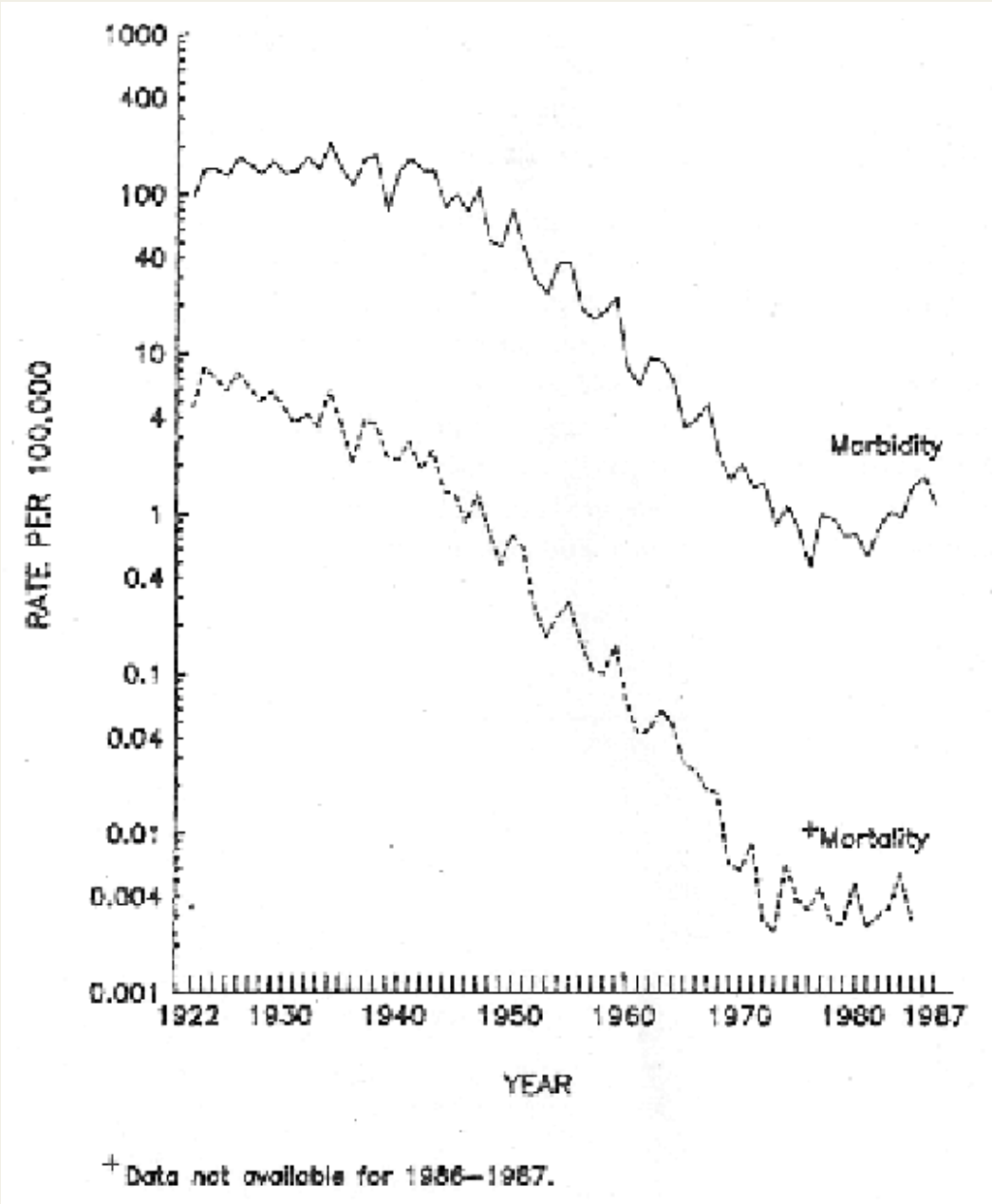

Figure 2: Pertussis, United States, 1922-1987

Hutchins et al. showed the reason for the increase, unwittingly, when they also wrote:

> *In 1978 a nationwide childhood immunization initiative was begun. Individual States passed legislation requiring proof of immunization for school entry at 5-6 years of age.*

The vaccination age started at 6-8 weeks (and not at 5-6 years), and large numbers of very young babies were vaccinated within a short period of time; hence the observed major increase of whooping cough in those babies straight after the first dose.

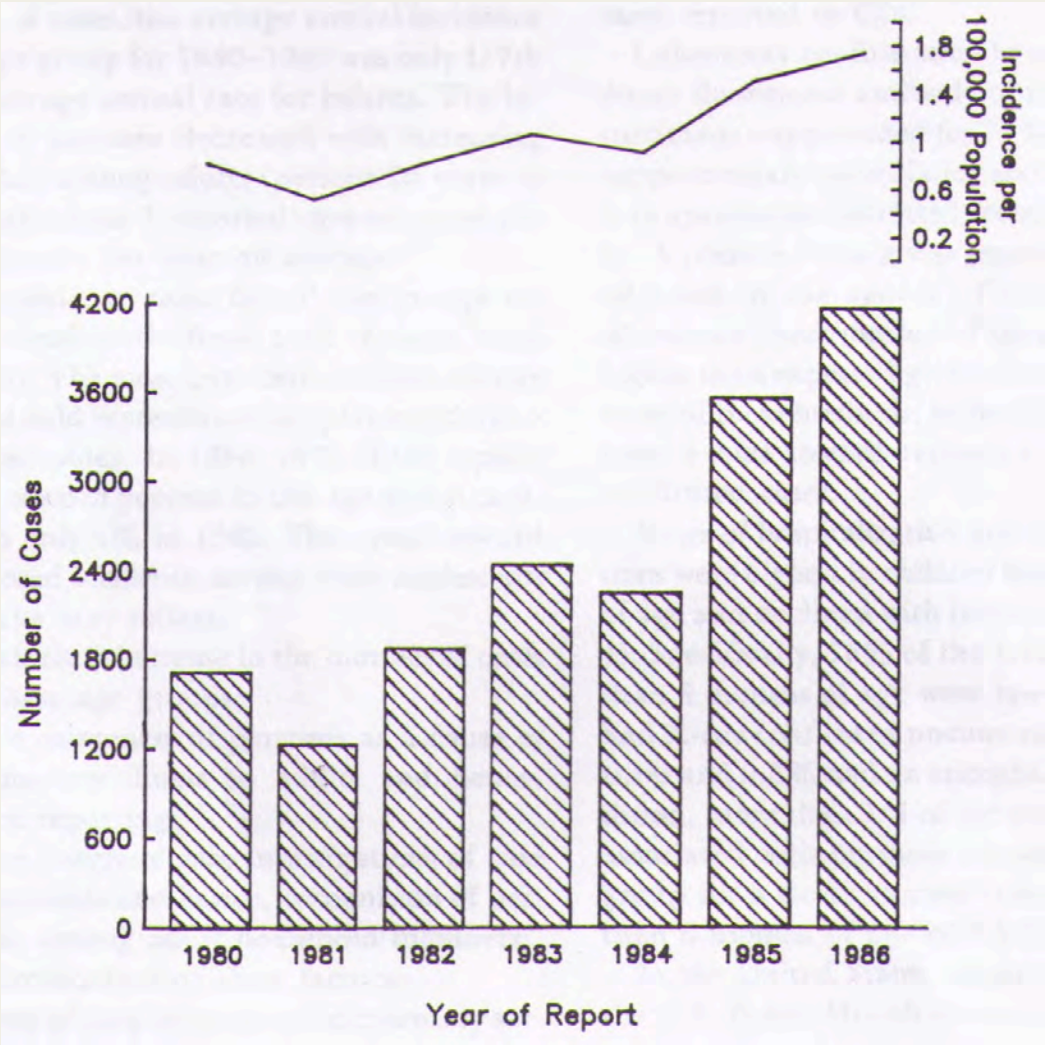

Figure 3: Number of Pertussis Cases Reported to MMWR and Incidence of Disease per 100,000 Population, United States, 1980-1986.

This also coincided with a sudden upsurge in cot deaths, of which the so-called Tennessee deaths were widely publicised. Bernier[22], Walker[23], and Griffin[24] all described a number of such tragedies. Their data showed a clear clustering of these deaths along the critical days as documented by data collection of babies breathing with Cotwatch breathing monitor.[25]

[22] Bernier RH, Frank JA Jr, Dondero TJ Jr, Turner P. Diphtheria-tetanus toxoids-pertussis vaccination and sudden infant deaths in Tennessee. J Pediatr. 1982 Sep;101(3):419-21.
[23] Walker AM, Jick H, Perera DR, Thompson RS, Knauss TA. Diphtheria-tetanus-pertussis immunization and sudden infant death syndrome. Am J Public Health. 1987 Aug;77(8):945-51.
[24] Griffin MR, Ray WA, Livengood JR, Schaffner W. Risk of sudden infant death syndrome after immunization with the diphtheria-tetanus-pertussis vaccine. N Engl J Med. 1988 Sep 8;319(10):618-23.
[25] Scheibner V. Dynamics of critical days as part of the non-specific stress syndrome discovered during monitoring with Cotwatch breathing monitor. 2004 J ACNEM; 23(3): 1-5.

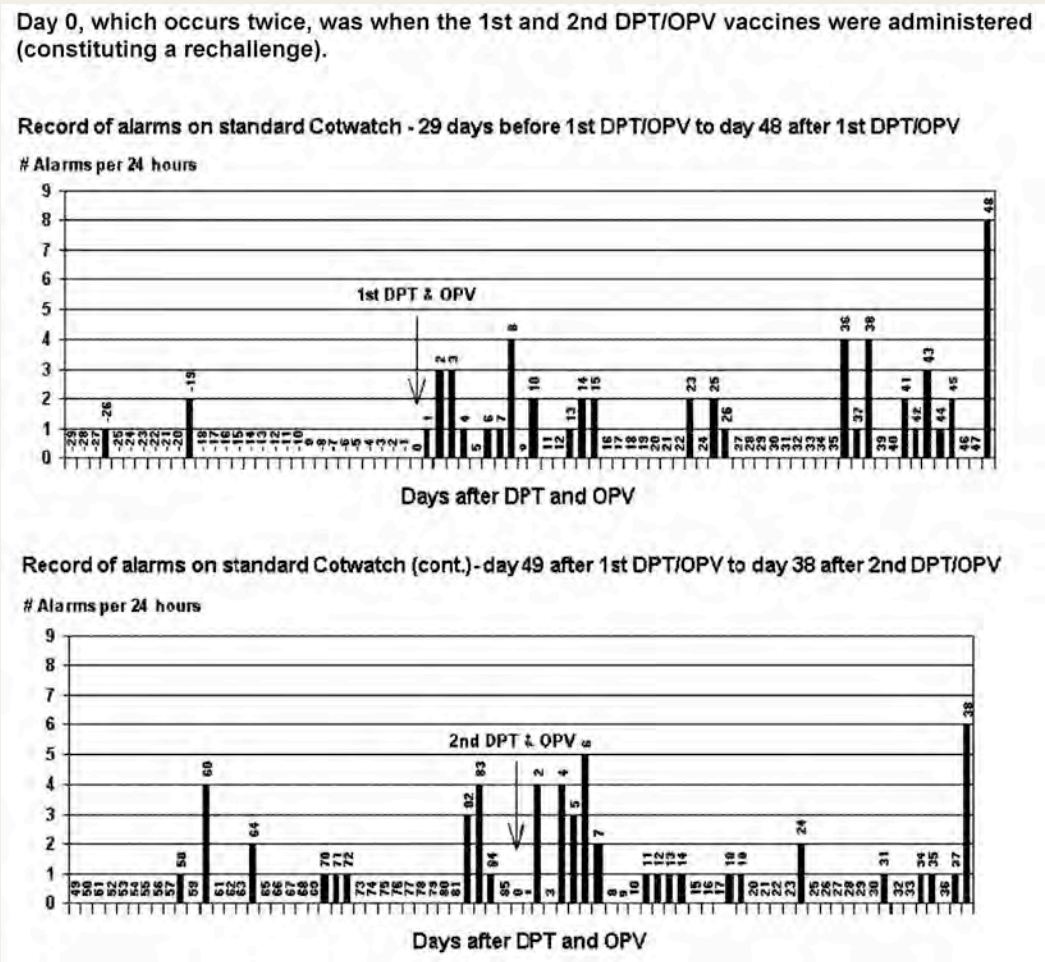

Figure 4: Record of alarms as recorded by the mother of a baby on the Cotwatch breathing monitor.

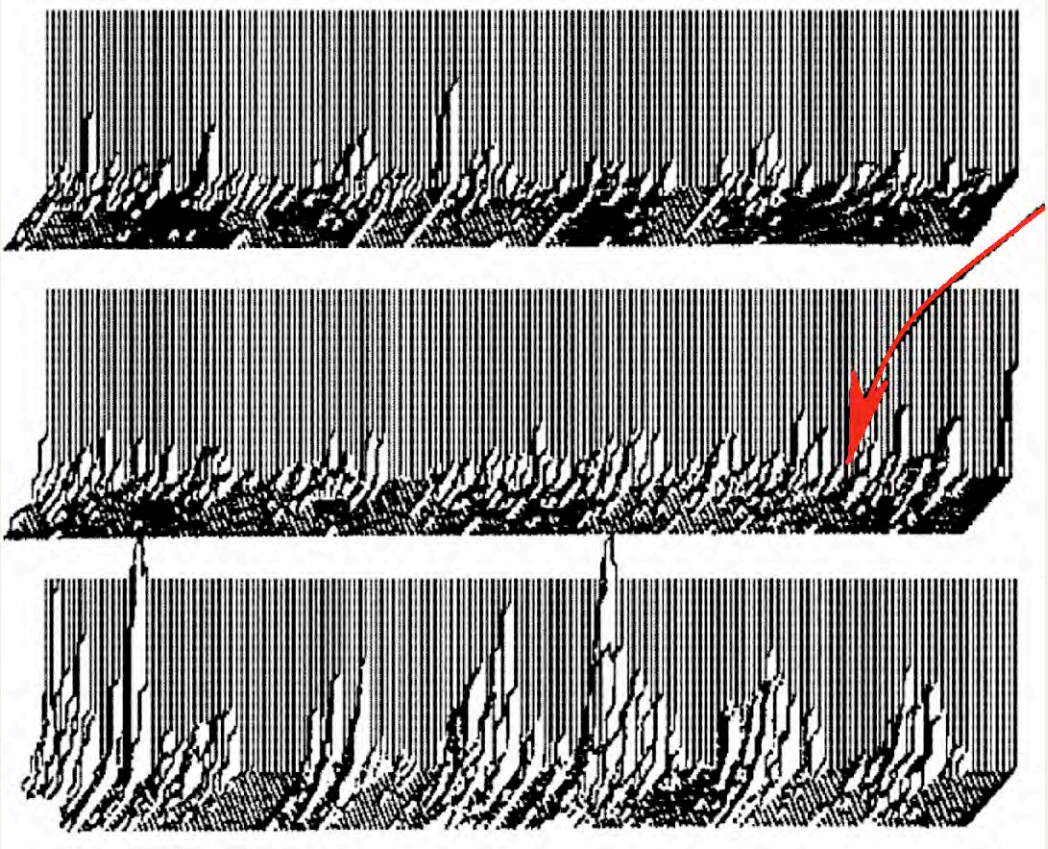

Figure 5: A "raw" record of breathing of baby one, as printed from the microprocessor Cotwatch breathing monitor. Every vertical line represents a histogram of events for one hour. Events from 6 to 15 seconds are mostly apneas (pauses in breathing), while the events above 15 seconds are mostly hypopneas (low volume breathing, which is only 5% of the volume of unstressed breathing). Hypopneas occur at critical hours in clusters of several shorter episodes within 10-15 minutes and are associated with exposure to a great variety of stressors.

The entire record represents 21 days of non-stop monitoring in sleep. The arrow indicates the day when the DPT vaccine was administered. A marked change in the pattern and duration of events in breathing occurred after the injection.

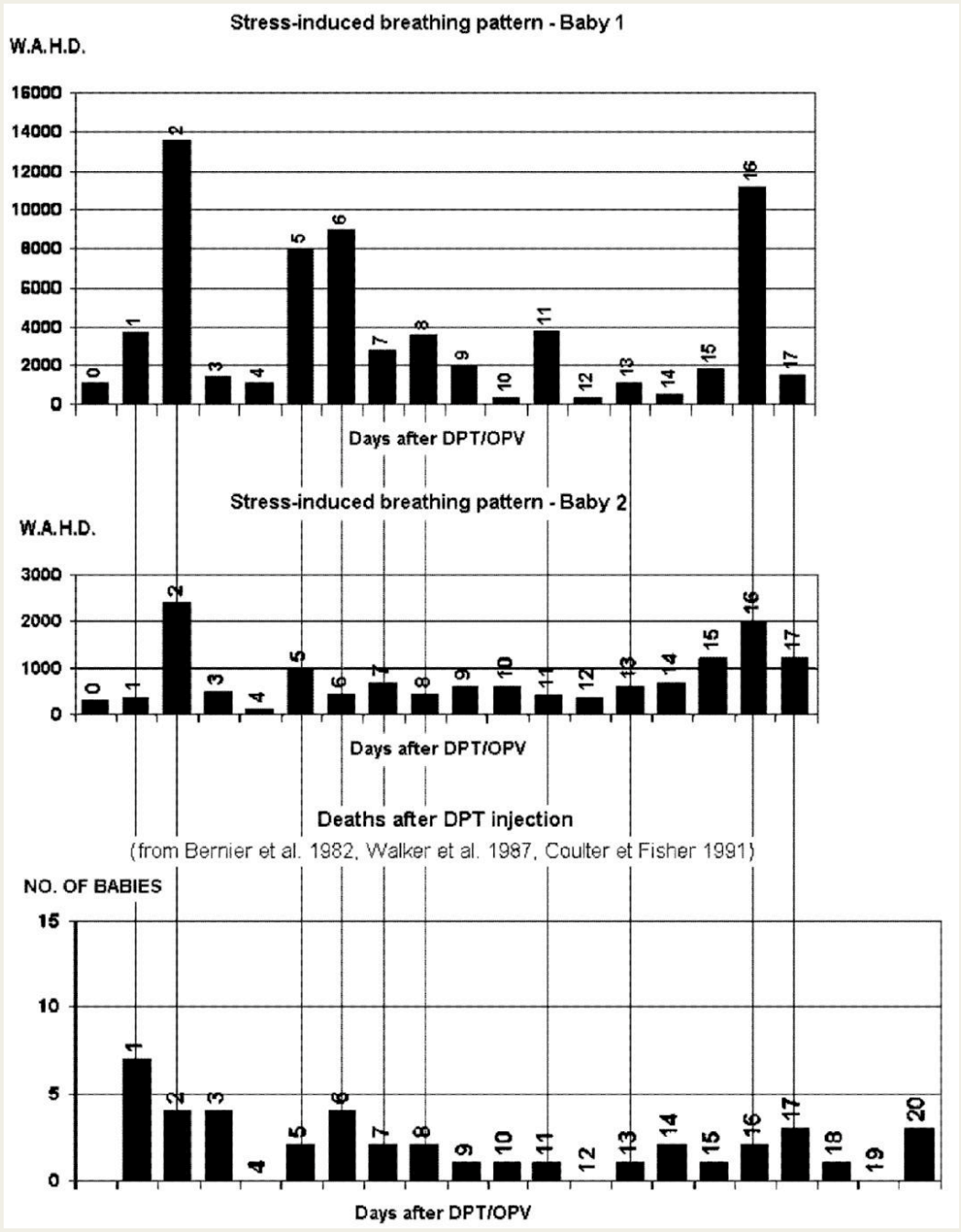

Figure 6: First and second charts: Record of events in breathing in two babies, as printed from the microprocessor Cotwatch breathing monitor.

- baby one had been given the third DPTP (diphtheria-pertussis-tetanus) and OPV (oral polio) vaccines and
- baby two had been give the first DPT and OPV vaccines.

Third chart: Actual deaths - 41 randomly listed in deaths in relation to when the last DPT vaccine had been administered.

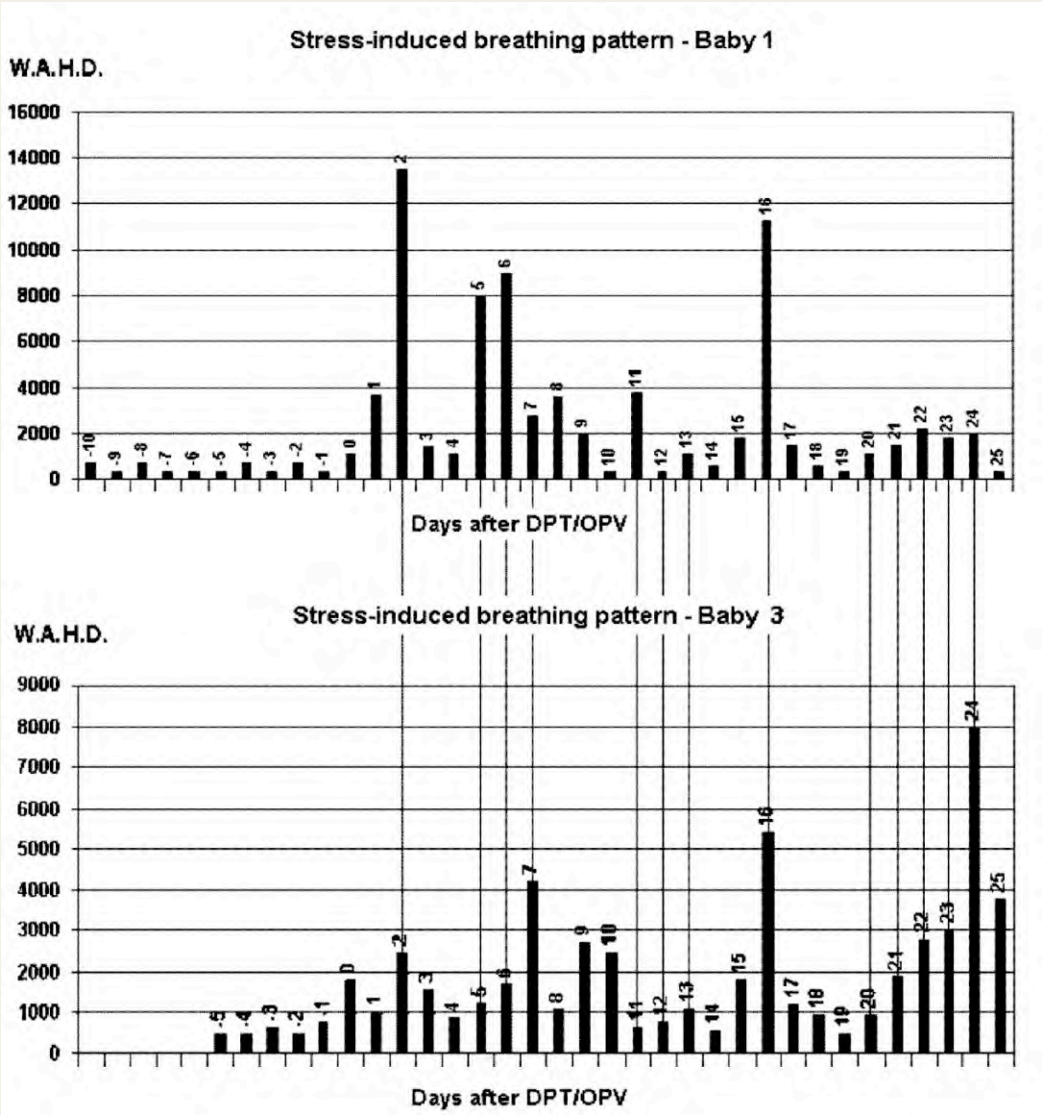

Figure 7: Record of events in breathing in two babies, as printed from the microprocessor Cotwatch breathing monitor

- baby one had been given the third DPT (diphtheria-pertussis-tetanus) and OPV (oral polio) vaccines
- baby three had been given the first DPT and OPV vaccines.

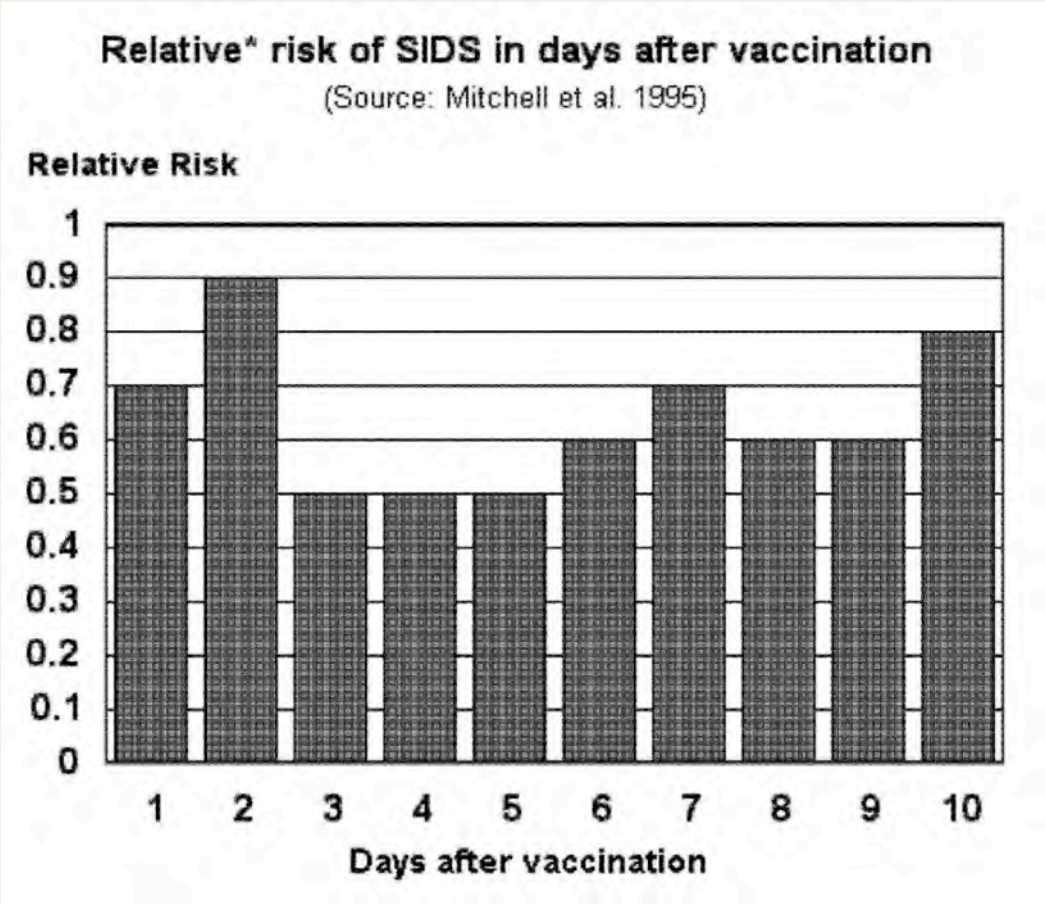

Figure 8: Relative* risk of SIDS in days after vaccination.

*Note: The risk is <u>not</u> relative to the risk of SIDS in unvaccinated babies. What is important to note here is the recognisable pattern of critical days.

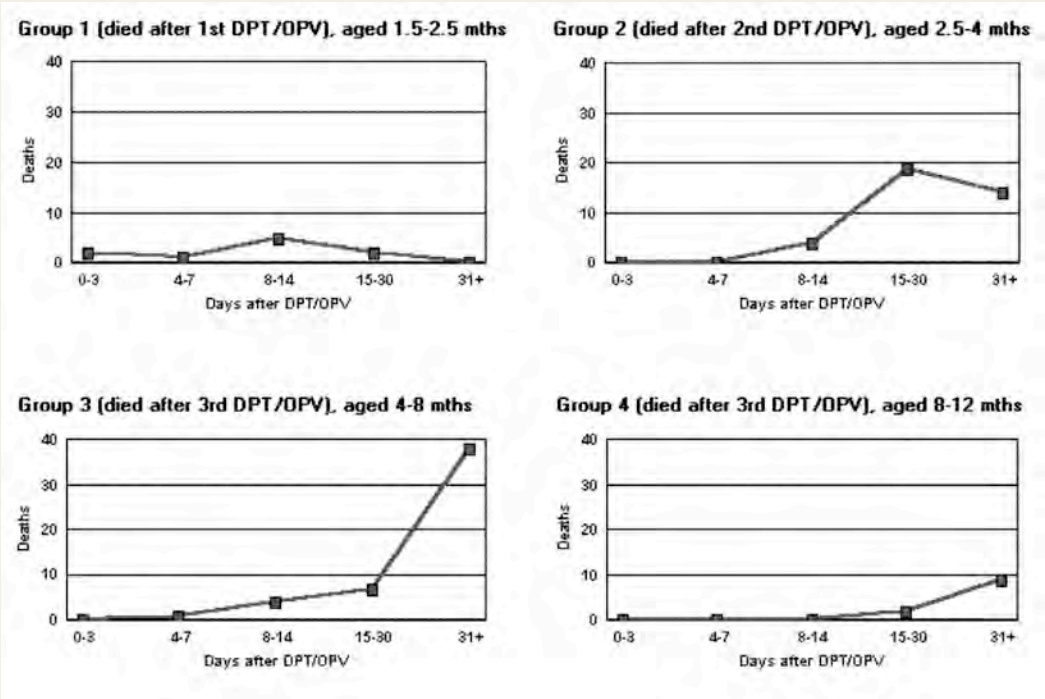

Figure 9: Links age, number of deaths and the time taken to die after vaccination (Source: Griffin et al. 1988)

Due to the 1975 UK television program reporting on brain damage linked to DPT vaccine, the vaccination compliance fell down to 30%, or even 10% in some areas, in the UK. This was followed by the longest inter-epidemic period with the lowest incidence of whooping cough on record.

Fine and Clarkson wrote:

> *Though overall pertussis incidence fell in England and Wales subsequent to the introduction of vaccination on a national scale in 1950s, pertussis epidemics have continued to occur regularly every 3-4 years. Since epidemic frequency is a function of the rate of influx of susceptibles, it is suprising that the interepidemic period did not decrease after the 1974 fall in vaccine uptake. One explanation for this paradox may be that pertussis vaccines are more effective in protecting against disease than in protecting against infection.*[26]

[26] Fine PE, Clarkson JA. The recurrence of whooping cough: possible implications for assessment of vaccine efficacy. Lancet. 1982 Mar 20;1(8273):666-9.

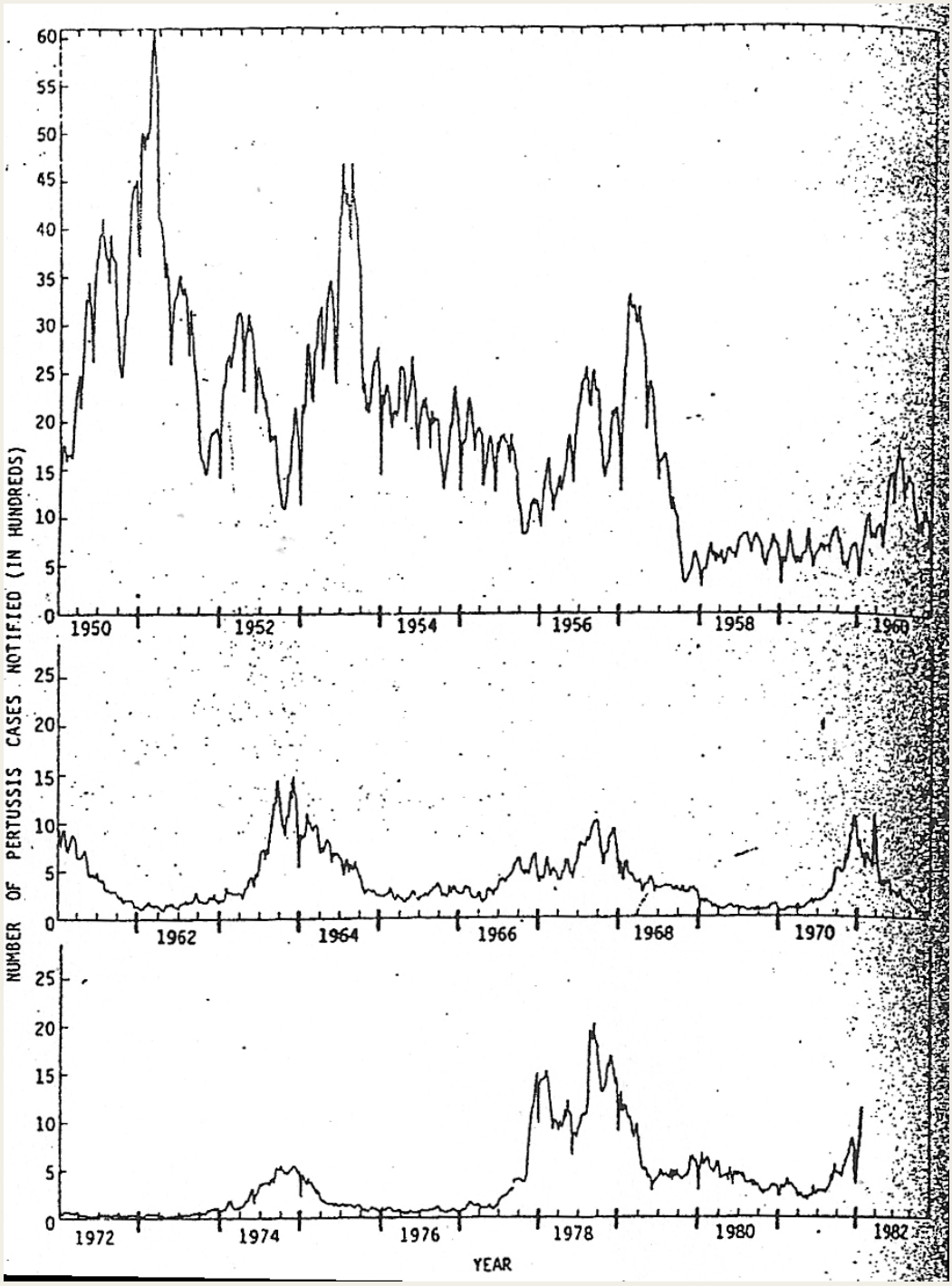

Figure 10: Weekly number of pertussis cases notified to Office of Population Censuses and surveys, from week 1 of 1950 to week 3 of 1982.

It is my opinion that the incidence of whooping cough fell worldwide in the mid 1970s due to natural dynamics, similar to those of measles, and not due to increasing levels of vaccination.

When vaccination stops, the incidence of the targeted disease returns back to normal dynamics. This explanation is supported by another observation in the UK and former West Germany. Miller and Farrington wrote:

> *In the West Germany unlike the UK, there are no national statistics on pertussis incidence, no national vaccination policy and no figures for vaccine uptake. . . vaccination rates are low and pertussis is prevalent, particularly in the 2-4 year age group, which is typical of a country with low vaccination uptake; similarly serotype 2 predominates. . . The age distribution was similar to that of cases reported in the UK during 1978 when vaccine uptake was at it's lowest with the highest proportion occurring in children aged 2-4 years.*[27]

Figure 11[28] is very instructive. The facts point strongly against the presumed benefits of vaccination.

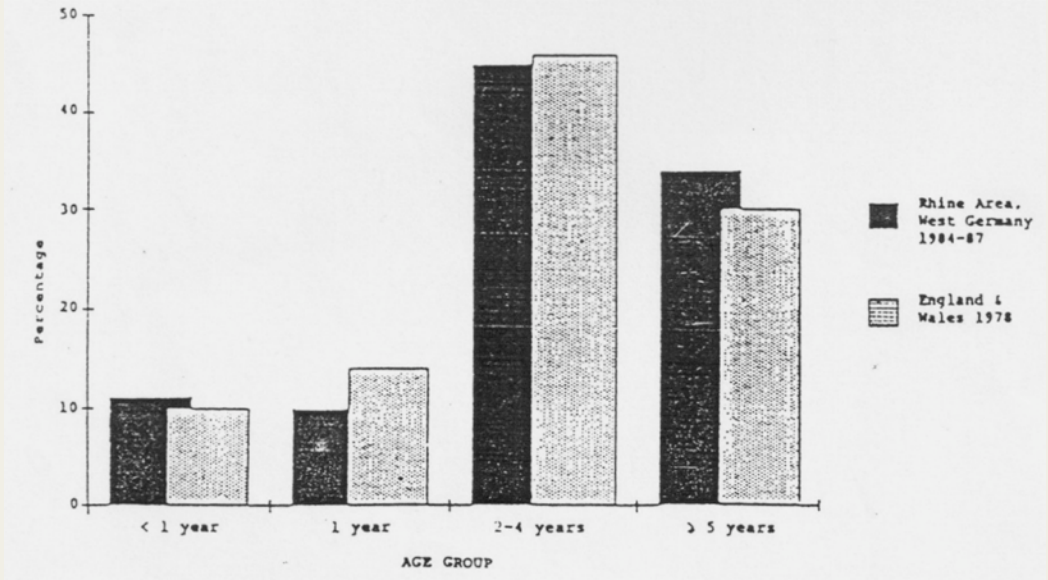

Figure 11: Age distribution of pertussis cases in West Germany and England & Wales.

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The dynamics of vaccine uptake as described above are also reflected in the dynamics of infant deaths after four weeks in England and Wales. According to Macfarlane:

> *The postneonatal mortality rate fell markedly in 1976, the year in which a sharp decline in perinatal deaths began. Between 1976 and 1979, however, neither the later neonatal nor the postneonatal mortality rate fell any further. Indeed, the postneonatal mortality rate*

[27] Miller E, Farrington CP. The current epidemiology of pertussis in the developed world: UK and West Germany. Tokai J Exp Clin Med. 1988;13 Suppl:97-101.
[28] Ibid.

increased slightly among babies born in 1977.[29] [when the vaccination compliance started climbing up.]

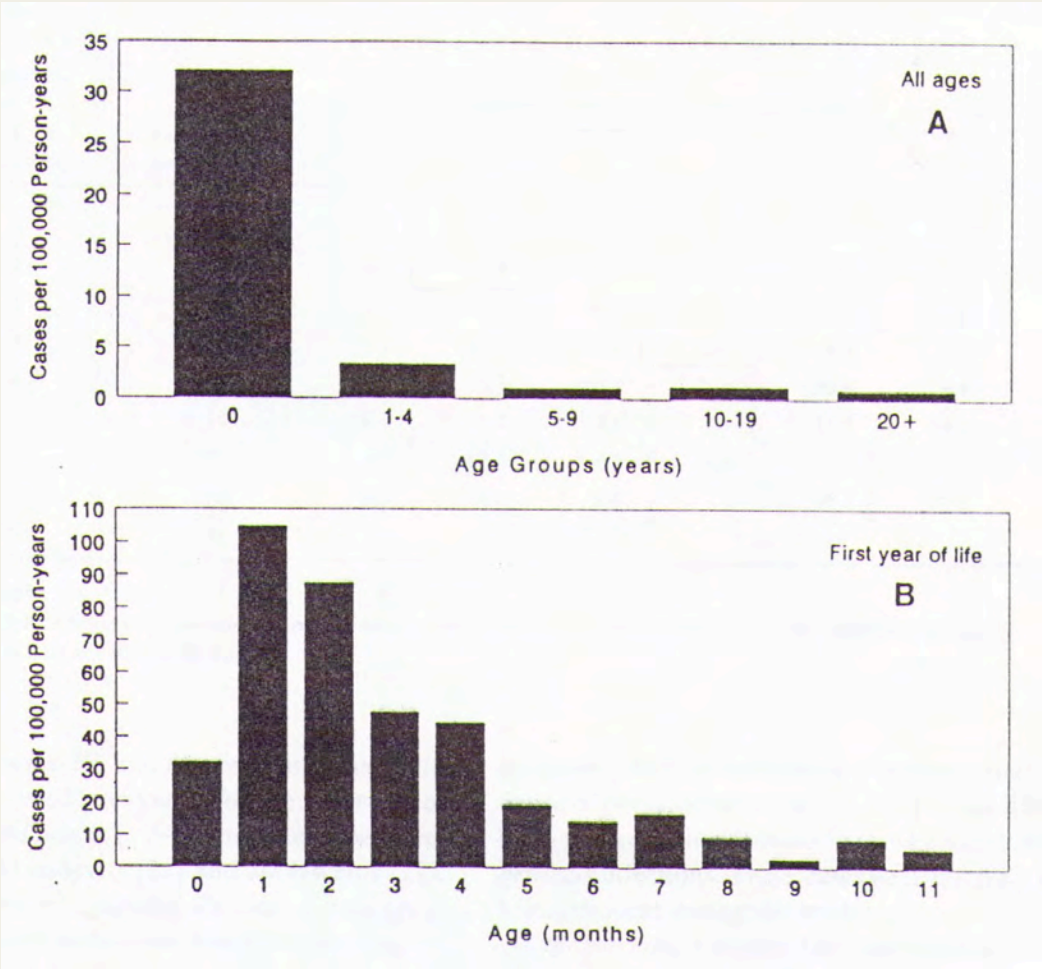

Figure 12: Age-specific incidence of bacteriologically confirmed pertussis. Massachusetts. 1981-1991. JID 1994:169 (June)

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In contrast, Marchant et al. described *inter alia* the age incidence of pertussis in Massachusetts in a ten year period 1981-1991[30] and demonstrated in figure 12 that the highest incidence of bacteriologically-confirmed pertussis was below the age of one; however, the breakdown in months showed the highest incidence was just after the first and second doses of DPT, with rapid decline afterwards. Equally revealing was the high incidence of pertussis below the vaccination age, in small babies (0 to

[29] MacFarlane A. Infant deaths after four weeks. Lancet. 1982 Oct 23;2(8304):929-30.
[30] Marchant CD, Loughlin AM, Lett SM, Todd CW, Wetterlow LH et al. Pertussis in Massachusetts, 1981-1991: incidence, serologic diagnosis, and vaccine effectiveness. J Infect Dis. 1994 Jun;169(6):1297-305.

6 weeks), this being due to the lack of TTI documentedly caused by the deleterious generational effect of vaccination.[31]

Sutter and Cochi studied pertussis hospitalisations and mortality in the United States between 1985 and 1988 and concluded that there was substantial under-reporting of pertussis in the US.[32] This of course would have inflated the perceived effectiveness of vaccination. They wrote that based on their indicators, the national health impact of pertussis is considerably higher than previously published reports suggested. Applying the age-specific hospitalisation rates, 187,867 to 515,930 cases of pertussis may have occurred during the study period, instead of only 14,057 cases reported to the CDC. They concluded that using different methods of estimation, approximately 121,340 cases of pertussis may have occurred during the study period, indicating 11.6% vaccine efficacy. Considering that the pre-vaccine era pertussis occurrence was in the order of 240,000 cases, vaccination has made no inroads into the pertussis incidence.

Williams et al, made a statement about infants who died:

> *Infants were less than six weeks of age and died from overwhelming cardiovascular respiratory compromise despite intensive care support. . .The excessive infant mortality from a preventable disease demonstrated the need for better pertussis immunity in the community and for erythromycin treatment of all suspected cases and family contacts.*[33]

But, their own data showed something completely different! All four babies were doing OK until they were admitted in hospitals and put on intravenous broad-spectrum antibiotics. The causal link to the administered antibiotics was clearly shown because the downhill slide followed closely the days when the offending antibiotics were administered.

Moreover, some of the mothers and siblings had whooping cough at the time of the infant's births, despite being fully vaccinated. One sibling's vaccination status was uncertain, but he was very likely vaccinated as part of the highly vaccinated generation.

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[31] Lavine JS, King AA, Bjørnstad ON. Natural immune boosting in pertussis dynamics and the potential for long-term vaccine failure. Proc Natl Acad Sci U S A. 2011 Apr 26;108(17):7259-64.
[32] Sutter RW, Cochi SL. Pertussis hospitalizations and mortality in the United States, 1985-1988. Evaluation of the completeness of national reporting. JAMA. 1992 Jan 15;267(3):386-91.
[33] Williams GD, Matthews NT, Choong RK, Ferson MJ. Infant pertussis deaths in New South Wales 1996-1997. Med J Aust. 1998 Mar 16;168(6):281-3.

This confirmed two phenomena:

- The increased incidence of whooping cough (and measles) in babies below the vaccination age reflects the lack of transplacentally-transmitted immunity in the era of vaccinated mothers as predicted by Lennon and Black.[34]
- A well documented phenomenon, that many cases (up to 65%) of infectious diseases develop straight after the first dose of the relevant vaccine in very young children.

Romanus et al. wrote that discontinuation of pertussis vaccination in 1979 in Sweden was followed by a low endemic level of pertussis for 3 years.[35] Thereafter the incidence gradually increased and there were two outbreaks in 1982-1983 and 1984-1986. In epidemic years, however, the incidence in infants and small children below the age of one year was very low (11%). The majority of cases (69%) occurred in older children up to 6 years, meaning: when Sweden stopped pertussis vaccination between 1979-1990, the disease incidence returned back to normal, with most cases occurring at the optimal age.

In contrast to this, when Sweden trialled the acellular pertussis vaccines for the second time (1990- 1995), as soon as the trial babies were vaccinated, there was a major outbreak of pertussis in those very young babies.[36] Since 82% of the entire live birth cohort participated in this trial, the pertussis epidemic reached noticeable proportions.

The acellular pertussis vaccine failed to make any inroads into pertussis incidence, as witnessed in Sweden: already during the [second] trials of that vaccine, the infant recipients contracted whooping cough which prompted discontinuation of the trial well before the planned date.[37] This is particularly instructive since during the eleven years without usage of pertussis vaccines (1979-1990) – babies under one year of age did not contract whooping cough and 90% of cases occurred in the ideal age group between 5-10 years.[38]

Despite high vaccination compliance, there remained a high persistent level of pertussis in regular 3.5 year epidemics. Vaccines made no inroads into incidence of pertussis as demonstrated in figure 13

[34] Ibid. Lennon and Black.
[35] Romanus V, Jonsell R, Bergquist SO. Pertussis in Sweden after the cessation of general immunization in 1979. Pediatr Infect Dis J. 1987 Apr;6(4):364-71.
[36] Olin P. Acellular pertussis vaccines--a question of efficacy. J Hosp Infect. 1995 Jun;30 Suppl:503-7.
[37] Ibid. Olin.
[38] Isacson et al. 1993. How common is whooping cough in a non-vaccinating country? Pediatr Infect Dis J; 12 (4): 284-288.

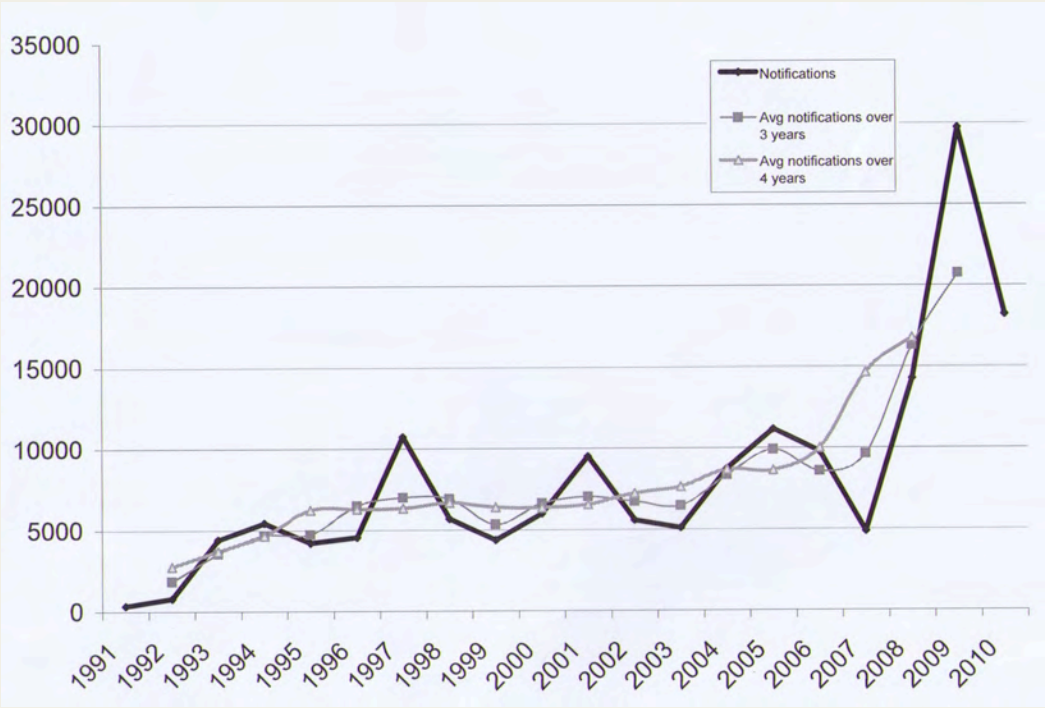

Figure 13: Pertussis notifications 1991-2009 (Australia).

♦The sordid history of Poliomyelitis vaccination

When the Salk injectable "formaldehyde killed" polio vaccine was tested on some 1.8 million American children in 1954-55, cases of paralysis in the vaccinated and some of their contacts started occurring within days.[39] The Cutter Laboratories were accused of distributing vaccines containing live polioviruses. Disasters with the Salk vaccines causing vaccine associated paralytic poliomyelitis (VAPP) seem to have been one of the main motivations behind development of an oral "live attenuated" Sabin vaccine, which was believed to simulate the natural infection. However, VAPP cases continued occurring with the Sabin vaccine.

I spent many hours locating and reading the older and more recent articles addressing the effectiveness, or otherwise, of combining IPV and OPV vaccines. I established that the results are not straightforward. Abraham reported that shedding of virulent poliovirus revertants, during immunization with oral poliovirus vaccines, after prior immunization with

[39] Peterson et al. VACCINATION-INDUCED POLIOMYELITIS IN IDAHO: PRELIMINARY REPORT OF EXPERIENCE WITH SALK POLIOMYELITIS VACCINE. *JAMA.* 1955;159(4):241-244.

inactivated polio vaccines, continued.[40] He also documented that prior immunization with EIPV (enhanced potency IPV) does not prevent faecal shedding of revertant polioviruses after subsequent exposure to OPV. [41]

Mensi and Pregliasco wrote:

> *In recent years great alarm has been generated by outbreaks of paralytic poliomyelitis in vaccinated populations…epidemics were observed in Finland in 1984, Senegal and Brazil in 1986, and Israel and Oman in 1988, all countries in which vaccination is widely deployed. Four epidemics were reported between 1991 and 1992. The first, in 1991, was in Bulgaria, which uses oral vaccination. Forty-three subjects developed paralytic type 1 polio; 88% of them belonged to a normal community and had not completed or even started a vaccination schedule. The second epidemic occurred in The Netherlands, where inactivated polio vaccine (IPV) is used, and involved 68 patients with type 3 poliovirus, members of the Amish…*[42] [In The Netherlands they are called members of orthodox religion and in fact use the polio vaccination (compliance between 40-50% and higher)].

Schaap et al. published a graph (figure 14) correlating the number of reported poliomyelitis cases with the vaccination rates in seven areas in The Netherlands.[43] Interestingly, the areas with the lowest compliance had the lowest number of cases and vice versa. The compliance ranged from 40-49% to 90-95%. In the 1992 epidemic, the first two cases occurred in a 14-year old boy and 23-year old male nurse, both vaccinated members of the orthodox religious group.

[40] Abraham R, Minor P, Dunn G, Modlin JF, Ogra PL. Shedding of virulent poliovirus revertants during immunization with oral poliovirus vaccine after prior immunization with inactivated polio vaccine.
J Infect Dis. 1993 Nov;168(5):1105-9.
[41] Carolina Mensi and Fabrizio Pregliasco. Poliomyelitis: Present Epidemiological Situation and Vaccination Problems. Clin Diagn Lab Immunol. 1998 May; 5(3): 278–280.
[42] Mensi C, Pregliasco F. Poliomyelitis: present epidemiological situation and vaccination problems. . Clin Diagn Lab Immunol. 1998 May;5(3):278-80.
[43] Schaap GJ, Bijkerk H, Coutinho RA, Kapsenberg JG, van Wezel AL. The spread of wild poliovirus in the well-vaccinated Netherlands in connection with the 1978 epidemic. Prog Med Virol. 1984;29:124–140.

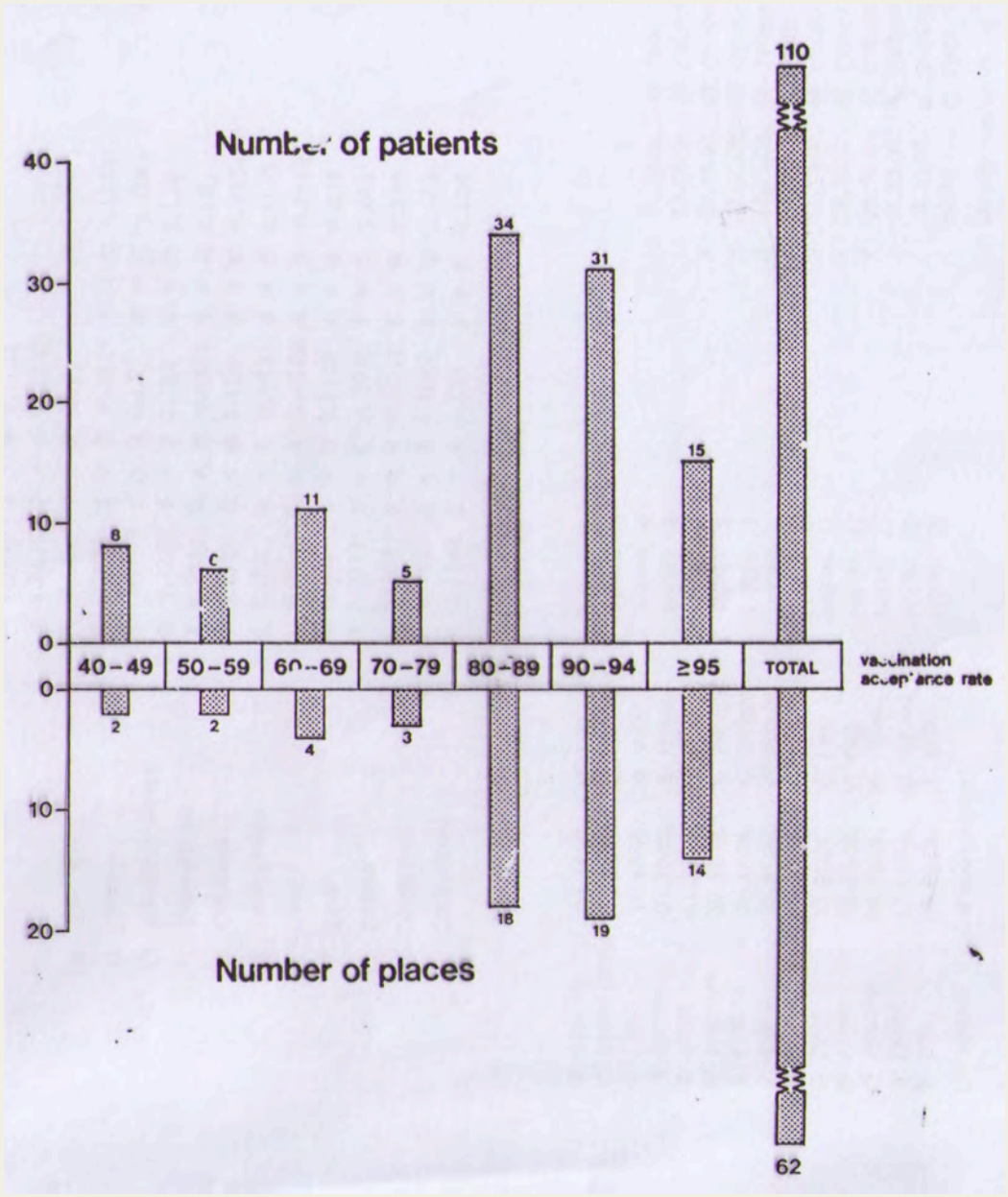

Figure 14: Number of places with polio cases, by average vaccination acceptance rate for birth cohorts 1971-1975 (Shaap/Bijkerk/Coutinho/Kapsenberg/van Wezel).

Sutter et al described an Oman outbreak as:

> *. . . evidence for widespread transmission among fully vaccinated children*.[44]

Incidence of paralytic disease was highest in children below 2 years:

> *. . . despite an immunisation programme that recently had raised coverage with 3 doses of oral poliovirus vaccine (OPV) among 12-*

[44] Sutter RW, Patriarca PA, Brogan S, Malankar PG, Pallansch MA et al. Evidence for widespread transmission among fully vaccinated children Lancet. 1991 Sep 21;338(8769):715-20.

> *months-old children from 67% to 87%... with transmission lasting for more than 12 months. Among the most disturbing features of this outbreak was that it occurred in the face of a model immunisation programme and that widespread transmission had occurred in a sparsely populated, predominantly rural setting.*[45]

One of the interesting reasons quoted was:

> *. . . rapid increases in vaccination coverage before the outbreak may have reduced or interrupted endemic circulation of indigenous strains, diminishing the contribution of natural infection to overall immunity levels in the general population.*[46]

The same reason was given by Biellik et al. in 1994 when they described the situation in Namibia. They wrote:

> *Endemic wild poliovirus circulation has continued uninterrupted in Angola and the two northern regions in Namibia across the well-travelled border since 1989, when cases were last reported. Although OPV3 cover age was fairly low in northern compared with southern Namibia, a higher proportion of northern children might have been protected, at least to type 1, by natural immunity, thus suppressing epidemics . . . the apparent interruption of* [natural] *poliovirus circulation* [by vaccination] *limited the acquisition of natural immunity.*[47]

Control of polio in the US shows the same phenomenon as the control of pertussis, namely downward trend, which stopped when individual states in the US mandated DPT and polio.

[45] Ibid.
[46] Ibid.
[47] Biellik RJ, Lobanov A, Heath K, Reichler M, Tjapepua V et al. Poliomyelitis in Namibia. Lancet. 1994 Dec 24-31;344(8939-8940):1776.

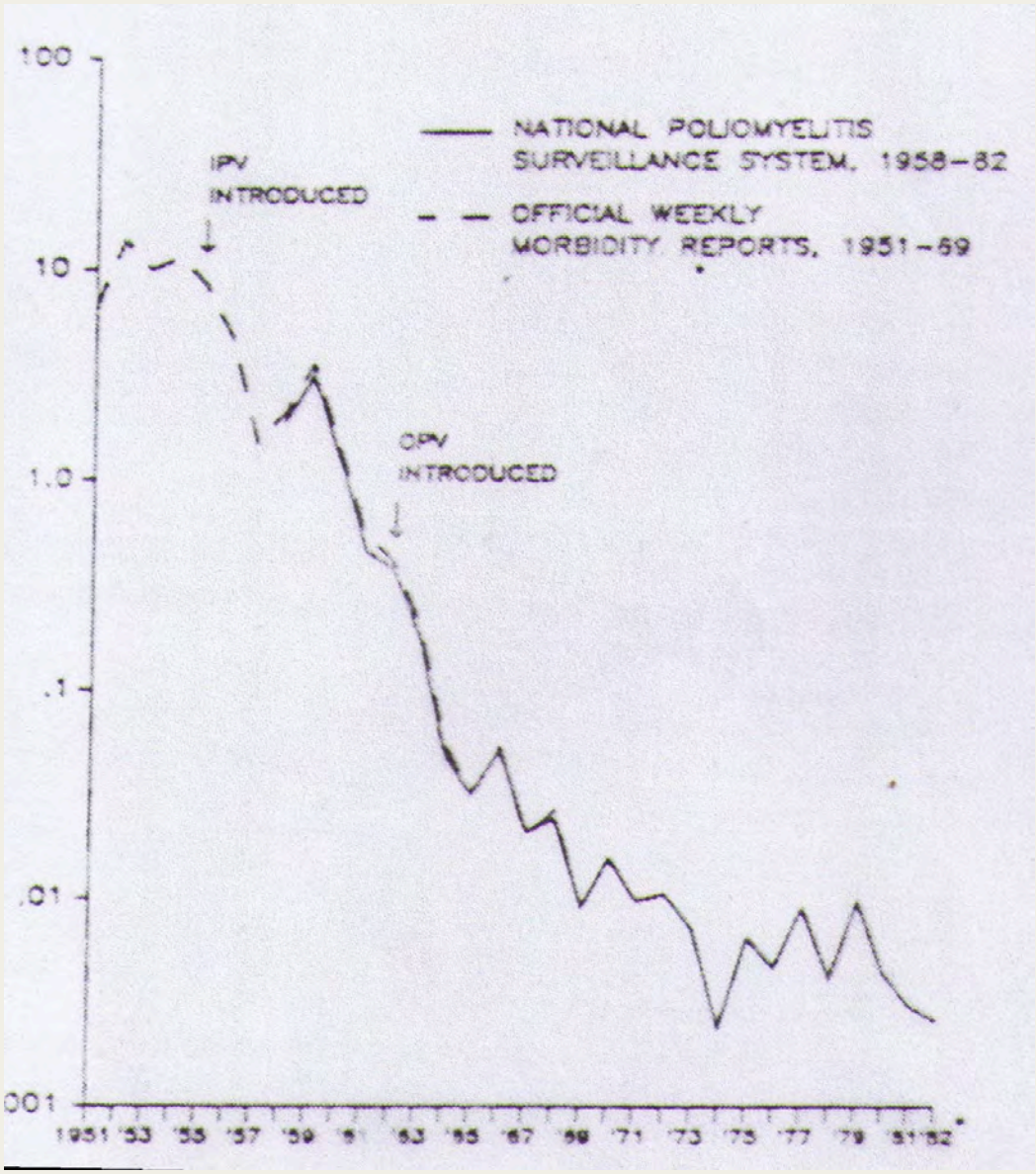

Figure 15: Annual reported paralytic poliomyelitis case rates, United States, 1951-1982 (Paralytic case rate/100,000 population.) The 1982 data are preliminary.

An interesting example of manipulation of data is polio "eradication" in the Americas. Figure 16[48] shows the effect of reclassification of poliomyelitis which allowed the ever increasing number of "notified" cases to morph into an ever decreasing number of "confirmed" cases.

[48] De Quadros CA, Andrus JK, Olivé JM, Da Silveira CM, Eikhof RM et al. Eradication of poliomyelitis: progress in the Americas. Pediatr Infect Dis J. 1991 Mar;10(3):222-9.

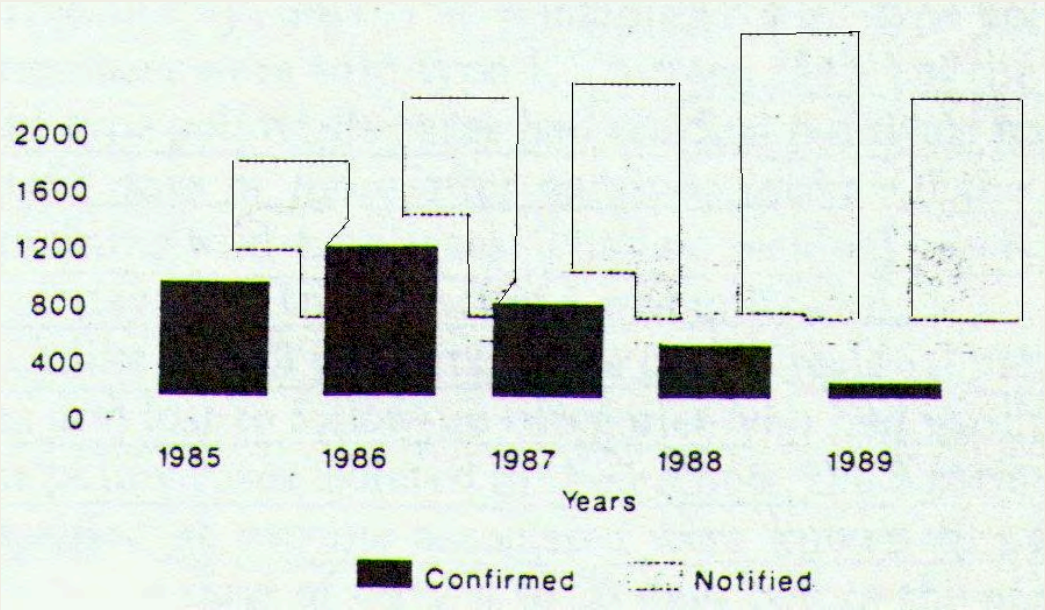

Figure 16: Polio cases notified and confirmed: The Americas. 1985-1989.

Dr HV Wyatt[49] quoted Hanlon et al. as stating:

> *…injections during an epidemic may provoke poliomyelitis in children already infected with poliovirus, [and] …provocation poliomyelitis occurs with injections of diphtheria/pertussis/tetanus vaccine, which, I am told, gives rise to unease among vaccinators. The risk of provocation poliomyelitis with the killed poliovaccine…occurred in the Cutter incident.*

During a poliomyelitis outbreak in Taiwan, Kim et al. reported that 65% of VAPP developed within 28 days of the first vaccine dose This report confirmed observations of others that two thirds of vaccine-targeted diseases occur after the first dose of relevant vaccines, including the polio vaccine,[50] and it also unwittingly confirmed the original and true definition of herd immunity that has nothing to do with vaccines: Epidemics occur during the accumulation of two thirds of susceptibles. Once natural immunity is 2/3 of susceptibles get the disease, the epidemic stops. Yet, the authors excluded (as unvaccinated) all paralytic cases (65% of all cases) from calculations of efficacy.

Ogra evaluated vaccination with live attenuated and inactivated poliovirus vaccines:

[49] Wyatt HV. Poliovaccination in the Gambia. Lancet. 1987 Jul 4;2(8549):43.

[50] Kim-Farley RJ, Rutherford G, Lichfield P, Hsu ST, Orenstein WA, Schonberger LB, Bart KJ, Lui KJ, Lin CC. Outbreak of paralytic poliomyelitis, Taiwan. Lancet. 1984 Dec 8;2(8415):1322–1324.

While the combination schedule employing EP-IPV followed by OPV should result in a decline of vaccine-associated (VAP) decease in OPV recipients, such immunization schedule may have little or no impact on the development of VAP in susceptible contacts. Furthermore, the logistics and the cost of combination schedule must be considered before current recommendations based on the use of OPV or EP-IPV alone are revised.[51]

Combined OPV and IPV recommendations

Continuing failures of polio eradication by OPV led to the proposals of using a combination of killed followed by oral polio vaccine delivery. However, such proposals are flawed and based on the ignorance of the documented past experience.

Simian Virus 40 contamination of polio vaccines

Perhaps the worst thing about polio vaccines is their continued contamination by monkey viruses of which SV 40 is the best researched one.

According to ample medical research evidence, polio vaccines of any kind cause VAPP. However, there are other major problems with the polio vaccine that justify scepticism about its benefits, one of which is the well-documented and continuous contamination by monkey viruses SV1-SV40.

Soon after the poliovirus mass vaccination programmes started in the US, a number of monkey viruses and amoebas were found in the vaccine seed brews. Hull, Milner et al.[52]and Hull, Johnston et al. (1955) encountered numerous filterable, transferable cytopathogenic agents other than polio virus in "normal" monkey renal cell cultures. Even though these agents completely destroyed culture tissues, and even caused serious diarrhoea in laboratory animals, all of which died, their possible pathogenesis in humans was ignored or glossed over. The central nervous system was particularly susceptible to the pathogenic properties of such viruses; the histopathological lesions observed in the intracerebrally inoculated monkeys revealed necrosis and complete destruction of the choroid plexus. Findings included generalised aseptic type meningitis. The isolated agent was called simian virus or SV and classified into 4 groups based on the cytopathogenic changes induced in monkey kidney cell cultures infected with these agents.

[51] Ogra PL. Comparative evaluation of immunization with live attenuated and inactivated poliovirus vaccines.Ann N Y Acad Sci. 1995 May 31;754:97-107.
[52] HULL RN, MINNER JR, SMITH JW. New viral agents recovered from tissue cultures of monkey kidney cells. 1956. Am J Hyg;63:204-215.

Hilleman and Sweet[53] reported on the "Vacuolating virus S.V. 40", which became the best researched among dozens of known monkey viruses. Gerber et al.[54] demonstrated that Sweet and Hilleman's method of inactivation of SV40 by 10 day treatment using 1: 4000 solution of formaldehyde was inadequate, since it took longer than 10 days to establish that the process was a subject to the asymptotic factor and hence incomplete. Fenner's research[55] has also established that even the inactivated portion of the viruses reverts back to the original virulence.

Dr Bernice Eddy documented the carcinogenic properties of these simian viruses: they caused tumours in hamsters injected with Rhesus monkey kidney cell extracts.[56] As established by many subsequent researchers, in humans SV40 causes characteristic brain tumours, bone sarcomas, mesotheliomas and an especially virulent form of melanoma cancer.

The stage was ready for a world-wide [admitted] contamination of hundreds of millions of children with an oncogenic monkey virus via polio vaccines. SV40 has been directly or indirectly implicated in an epidemic of great number of conditions and brain, lung, bone, renal and other tumours in all ages.[57,58,59,60,61]

Dr Stanley Kops is a modern day advocate for SV40 truth, and he wrote:

> *To date, the scientific literature and research examining SV40 and cancer-related diseases has been based upon an assumption that SV40 was not present in any poliovirus vaccines administered in the United States and was removed from the killed polio vaccines by 1963. The presumption has been that the regulation for live oral polio vaccine required that SV40 be removed from the seeds and monovalent pools ultimately produced in the manufacturing process…The confirmation*

[53] Sweet, B. H., and M. R. Hilleman. The vacuolating virus, SV40. Proc Soc Exp Biol Med. 1960 Nov;105:420-7.
[54] GERBER P, HOTTLE GA, GRUBBS RE. Inactivation of vacuolating virus (SV40) by formaldehyde. Proc Soc Exp Biol and Med; 108: 205-209.
[55] Fenner F. Reactivation of Animal Viruses. Br Med J. 1962 July 21; 2(5298): 135–142.
[56] EDDY BE, BORMAN GS, BERKELEY WH, YOUNG RD. Tumors induced in hamsters by injection of Rhesus monkey kidney cell extracts. 1961. Proc Soc Exp Biol and Med; 107; 191-7.
[57] Carbone M, Pass HI, Rizzo P, Marinetti M, Di Muzio M et al. Simian virus 40-like DNA sequences in human pleural mesothelioma. Oncogene. 1994 Jun;9(6):1781-90.
[58] Bergsagel DJ, Finegold MJ, Butel JS, Kupsky WJ, Garcea RL.DNA sequences similar to those of simian virus 40 in ependymomas and choroid plexus tumors of childhood. N Engl J Med. 1992 Apr 9;326(15):988-93.
[59] Carbone M, Rizzo P, Grimley PM, Procopio A, Mew DJ Simian virus-40 large-T antigen binds p53 in human mesotheliomas. Nat Med. 1997 Aug;3(8):908-12.
[60] Butel JS and Lednicky JA. Cell and molecular biology of simian virus 40: implications for human infections and disease.J Natl Cancer Inst. 1999 Jan 20;91(2):119-34.
[61] Weiner LP, Herndon RM, Narayan O, Johnson RT, Shah K Isolation of virus related to SV40 from patients with progressive multifocal leukoencephalopathy. 1972. NEJM;286(8):385-390.

of the removal by one manufacturer, Lederle, has been made public at an international symposium in January 1997, where its representatives stated that all Lederle's seeds had been tested and screened to assure that it was free from SV40 virus. However, in litigation involving the Lederle oral polio vaccine, the manufacturer's internal documents failed to reveal such removal in all its seeds. The absence of confirmatory testing of the seeds, as well as testimony for SV40 of a Lederle manager indicate that this claim cannot be fully substantiated...[62]

The scientific community should not be content with assurances to the contrary. The continuing occurrence of the above characteristic SV40 tumours in younger and especially quite recent generations of vaccinees should not be ignored or treated with indifference.

Contamination of polio vaccines by chimpanzee coryza virus, or RSV.

Another important consideration in attempts to eradicate poliomyelitis by vaccination is the contamination of polio vaccines by chimpanzee coryza virus, renamed respiratory syncytial virus (RSV).

In 1956, Morris et al. described monkey cytopathogenic agent that produced acute respiratory illness in chimpanzees at the Walter Reed Army Institute of Research and named it chimpanzee coryza virus (CCA). [63] In 1957, Chanock et al. wrote on the association of a new type of cytopathogic myxovirus with infantile croup.[64]

Chanock and Finberg reported on two isolations of similar agents from infants with severe lower respiratory illness (bronchopneumonia, bronchiolitis and laryngotracheobronchitis). The two viruses were indistinguishable from an agent associated with the outbreak of coryza in chimpanzees (CCA virus) studied by Morris in 1956.

A person working with the infected chimpanzees subsequently experienced respiratory infection with a rise in CCA antibodies during convalescence. They proposed a new name for this agent "respiratory syncytial virus" (RSV). RSV has spread via contaminated polio vaccines like wildfire all over the world and continues causing serious lower respiratory tract infections in infants.

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[62] Kops, SP. Oral polio vaccine and human cancer: a reassessment of SV40 as a contaminant based upon legal documents. Anticancer Res. 2000 Nov-Dec;20(6C):4745-9.

[63] Blount, R. E., Jr., J. A. Morris, and R. E. Savage. 1956. Recovery of cytopathogenic agent from chimpanzees with coryza. Proc. Soc. Exp. Biol. Med. 92**:**544-549.

[64] CHANOCK R, FINBERG L. Recovery from infants with respiratory illness of a virus related to chimpanzee coryza agent (CCA). II. Epidemiologic aspects of infection in infants and young children. Am J Hyg. 1957 Nov;66(3):291-300.

Beem et al. isolated the virus from inpatients and outpatients in the Bob Robert Memorial Hospital for Children (University of Chicago) during the winter of 1958-1959, in association with human acute respiratory illness.[65] The virus (named Randall) had an unusual cytopathic effect characterised by extensive syncytial areas and giant cells. Soon, 48 similar agents were isolated from 41 patients. There were antigenic similarities between RV and Long and Sue strains of CCA; it produced illness in humans (the age range 3 weeks to 35 years): acute respiratory diseases, croup, bronchiolitis, pneumonia and asthma ranging from mild coryza to fatal bronchiolitis. The isolation rate (46%) was particularly high among infants below six months.

In Australia, Lewis et al. isolated further viral specimens identical with CCA. [66]

Prior to July 1960, the influenza and parainfluenza viruses predominated in infant epidemic respiratory infections; in July 1961 the pattern changed abruptly with sudden increases in bronchiolitis and bronchitis, that were previously infrequent. 58% were under 12 months, and patients under 4 years predominated. Infants with bronchiolitis and severe bronchitis yielded RCA, not previously isolated. Deaths have occurred.

Rogers' 1959 observations on antibiotic ineffectiveness, and new serious additional problems fell on deaf ears. He wrote that life-threatening microbial infections continued to occur despite antibiotics, and that the previous microbial landscape also shifted by 1957-1958. There was streptococcal predominance from 1938-1940, and then an "impressive" increase in the number of life-threatening enterobacterial infections post antibiotic.

> *During the preantimicrobial era most infections were acquired before admission to hospital, while in the postantimicrobial era the vast majority of infections arose in hospital . . . Mycotic infections, especially with Candida albicans, became a major problem. Unusual serious generalised clostridial infections arose and antibiotics have not dramatically altered the risk of, or mortality resulting from, endogenous infections in sick, hospitalised patients.*[67]

[65] BEEM M, WRIGHT FH, HAMRE D, EGERER R, OEHME M. Association of the chimpanzee coryza agent with acute respiratory disease in children.
N Engl J Med. 1960 Sep 15;263:523-30.
[66] Lewis et al.. A syncytial virus associated with epidemic disease of the lower respiratory tract in infants and young children. 1961. Med J Australia: 932-933 and Forbes (1961. Ibid: 323-325).
[67] ROGERS DE. The changing pattern of life-threatening microbial disease. N Engl J Med. 1959 Oct 1;261:677-83.

Levy et al. wrote:

> *Respiratory syncytial virus (RSV) is the most prevalent cause of lower respiratory tract infections (LRTI) in infants and young children. Infections with RSV is a major health problem during early childhood and primary RSV infections occurs most often between the ages of 6 weeks and 2 years. Approximately one half of all infants become infected with RSV during the first year of life and nearly all infants by the end of their second year of life…in the US each year, approximately 100,000 children are hospitalised at an estimated cost of $300 million. More than half of those admitted for RSV bronchiolitis are between 1 and 3 months of age.* [68] [Clearly implicating vaccination.]

RSV vaccine developed in the late 1960s failed miserably. It is no mystery why there is no RSV vaccine recommended today. Fulginiti and others showed the vaccine was ineffective, and induced an exaggerated, altered clinical response… causing RSV illness requiring hospitalisations among vaccinees, and led to delayed dermal hypersensitivity.[69]

Simoes wrote:

> *Since it was identified as the agent that causes chimpanzee coryza in 1956, and after its subsequent isolation from children with pulmonary disease in Baltimore, USA, respiratory syncytial virus (RSV) had been described as the single most important virus causing acute respiratory-tract infections in children. The WHO estimates that of the 12.2. million annual deaths in children under 5 years, a third are due to acute infections of the lower respiratory tract. Streptococcus pneumoniae, Haemophilus influenzae, and RSV are the predominant pathogens… vaccinated children were not protected from subsequent RSV infection. Furthermore, RSV-naïve infants who received formalin-inactivated RSV vaccine, and who were naturally infected with RSV later, developed more severe disease in the lower respiratory tract than a control group immunized with a trivalent parainfluenza vaccine.*[70]

It should surprise nobody that data from ten developing countries—**with intense polio vaccination**, revealed that RSV was the most frequent cause of LRT infections (70% of all cases).

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[68] Levy BT, Graber MA. Respiratory syncytial virus infection in infants and young children. J Fam Pract. 1997 Dec;45(6):473-81.

[69] Fulginiti VA, Eller JJ, Sieber OF, Joyner JW, Minamitani M et al. Respiratory virus immunization. I. A field trial of two inactivated respiratory virus vaccines; an aqueous trivalent parainfluenza virus vaccine and an alum-precipitated respiratory syncytial virus vaccine.Am J Epidemiol. 1969 Apr;89(4):435-48.

[70] Simoes EA. Respiratory syncytial virus infection.Lancet. 1999 Sep 4;354(9181):847-52.

Polio vaccines are not only ineffective in preventing paralysis, they carry the risk of contamination with many harmful adventitious microorganisms, of which only some monkey viruses have been researched in more detail. Many other potentially dangerous microorganisms remain unaddressed.

Polio vaccination and brain-eating amoebas.

Contamination of monkey kidney tissue cultures (used in the production of polio vaccines) by live amoebas.

In 1996, while watching a TV news report on the death of two 5-year olds in Australia from brain-eating amoebae, I remembered a note in Hull et al.'s paper

> *Recently, an amoeba was isolated from monkey kidney tissue cultures and was identified as belonging to the genus Acanthamoeba. It grew readily in tissue cultures… It appeared to have the ability to infect and kill monkeys and mice following intracerebral and intraspinal inoculation.*[71]

Amoebas are unicellular protozoan microorganisms. According to Ma et al.[72], they are classified in the phyllum Sarcomastigophora and belong to Rhizopoda, equipped by propulsive pseudopodia and/or protoplasmic flow without production of pseudopodia. Acanthopodina, a suborder of Amoebida, form two families, Vahlkampfiidae and Acanthoamoebididae, with two genera Naegleria and Acanthamoeba respectively, with a number of species. Naegleria species form three life-stages, trophozoites, flagellates and cysts and Acanthamoeba species only two, trophozoites and cysts.

Jahnes et al.[73] isolated two strains of apparently the same amoeba which looked like round bodies, similar in appearance to cells manifesting changes induced by certain simian (monkey) viruses. On closer examination, they proved to be amoebic cysts. They varied in size, from 10 to 21 microns in diameter. In one experiment, the cysts were treated with 10% formalin, washed and inoculated into monkey kidney tissue culture tubes. The monkey kidney cells phagocytised the cysts. The trophozooites turned into cysts under refrigeration down to 4 degrees C. These were resistant even under –50 degrees C for months and survived in the pH

[71] Ibid Hull 1958.
[72] Ma P, Visvesvara GS, Martinez AJ, Theodore FH, Daggett PM et al. Naegleria and Acanthamoeba infections: review. Rev Infect Dis. 1990 May-Jun;12(3):490-513.
[73] JAHNES WG, FULLMER HM. Free living amoebae as contaminants in monkey kidney tissue culture. Proc Soc Exp Biol Med. 1957 Nov;96(2):484-8.

range 5.0-9.0. Their tissue cultures were not affected by streptomycin and penicillin.

Culbertson[74,75] confirmed that amoebas caused brain disease and death within days, in monkeys and mice.

The reports showed, that following inoculations, "extensive chorio-meningitis and destructive encephalomyelitis occurred" and killed monkeys in four to seven days and mice in three to four days. Intravenous injections of the amoebas resulted in perivascular granulomatous lesions. Intranasal inoculation in mice resulted in fatal infections in about four days. These mice exhibited ulceration of the frontal lobes of the brain. There were amoebas in the lungs, and they caused severe pneumonic amoeba reaction. Haemorrhage was a common feature. Sections of the kidney showed amoebas present in the glomerular capillaries.

Amoebas showed the ability to migrate through the tissues. The size of the inoculum did not matter: both small and large inoculums produced amoebic invasions. Intragastric inoculations were unsuccessful most probably because amoebic cysts were dissolved by bile.

Researchers, as a rule failed to address the seriousness of the introduction into children of Acanthamoeba via the polio vaccines, even though they were aware of their origin from monkey kidney tissue cultures used in the production of polio vaccines. However they noted that the most contaminated age group was babies below the age of crawling – between 2 and ten months.

Live amoebas were isolated from the air[76] in the UK, together with respiratory syncytial virus, and from the surfaces in hospital cubicles in which infants with acute bronchiolitis were being nursed. The amoebas were isolated at Booth Hall Children's Hospital in the cubicle occupied by a ten-week-old infant with acute bronchiolitis. First, only RSV was isolated and the child sent home, but later an unidentified cytopathic effect was noticed in the tissue cultures and was provisionally called "Ryan virus1"[77] by Pereira, and later also noted in a post-mortem bronchial swab of another seven-months old baby boy with RSV bronchiolitis.

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[74] CULBERTSON CG, SMITH JW, MINNER JR. Acanthamoeba: observations on animal pathogenicity. Science. 1958 Jun 27;127(3313):1506.

[75] CULBERTSON CG, SMITH JW, COHEN HK, MINNER JR. Experimental infection of mice and monkeys by Acanthamoeba. Am J Pathol. 1959 Jan-Feb;35(1):185-97.

[76] D. Kingston and D. C. Warhurst. Isolation Of Amoebae From The Air
J Med Microbiol February 1969 vol. 2 no. 1 27-36.

[77] Pereira MS, Marsden HB, Corbitt G, Tobin JO. Ryan virus: a possible new human pathogen. Br Med J. 1966 Jan 15;1(5480):130-2.

Pereira's paper describes the course of illness: Six days before admission, the baby developed a sore throat and ulcers in the mouth which later spread over the face; he was unwell, could not suck and developed loose stools. The day before admission, he developed a cough and started vomiting. He was drowsy and dyspnoeic, made jerky movements and died soon after admission. Necropsy showed some emphysema, petechiae, and small areas of congestion and alveolar haemorrhaging in the lungs, a fatty liver, prominent mesenteric nodes, and mucopus in the ears. Escherichia coli bacteria were cultured from his ears. Death was diagnosed as due to a respiratory infection associated with encephalomyelitis and hepatitis. Vaccination status was not disclosed, although considering the age, the baby could have received up to three doses of DPT and OPV vaccines.

Armstrong and Pereira identified the Ryan virus as Hartmanella castellanii.[78] They had no doubt that these amoebas came from the human respiratory tract. In Australia, Fowler and Carter[79] Carter[80], and Carter et al.[81] described a number of cases in children and adults.

Many cases all over the world occurred in children and adults, with and without histories of swimming in lakes and public swimming pools.[82]

Even if polio vaccines were effective in preventing polio paralysis, their potentially continued contamination by undesirable microorganisms (monkey viruses and amoebas) should encourage the abandonment of their use.

Well-meaning Rotarians should study the relevant medical research first, before engaging in global polio vaccination.

Question 2. What is in a vaccine?

The Australian Immunisation Handbook, 9th edition, appendix 4, lists all "components in vaccines used in the national immunisation program" [in

[78] J. A. Armstrong and M. S. Pereira. Identification of "Ryan Virus" as an amoeba of the genus Hartmannella. Br Med J. 1967 January 28; 1(5534): 212–214.
[79] M. Fowler and R. F. Carter.Acute Pyogenic Meningitis Probably Due to Acanthamoeba sp.: a Preliminary Report.Br Med J. 1965 September 25; 2(5464): 734-2, 740-742.
[80] Carter RF. Primary amoebic meningo-encephalitis: clinical, pathological and epidemiological features of six fatal cases. J Pathol Bacteriol. 1968 Jul;96(1):1–25.
[81] Carter RF, Cullity GJ, Ojeda VJ, Silberstein P, Willaert E. A fatal case of meningoencephalitis due to a free-living amoeba of uncertain identity--probably acanthamoeba sp.Pathology. 1981 Jan;13(1):51-68.
[82] Scheibner 1999. Brain-eating bugs: the vaccine connection. Nexus Magazine; (whale.to/vaccines/amoebas.html).

Australia]. It includes thiomersal (a mercury compound containing 49% mercury) in hepatitis B and several flu vaccines for adults and infants.

The list reveals the following ingredients in Australian vaccines: aluminium hydroxide and aluminium phosphate, borax, egg protein, formaldehyde, gelatin, gentamycin, kanamycin, monosodium glutamate (MSG), neomycin, phenol, phenoxyethanol, polymixin, and yeast. It is not difficult to see where all those allergies in vaccinated children come from.

Various oils have been used in vaccines in the past, including squalene, mineral oil, and peanut oil, in attempt to ramp up the local immune response at the injection site. The results were uniformly damaging. Peanut allergy epidemics appeared not long after the introduction of adjuvant 65-4, a peanut oil adjuvant used by Merck.[83] Today, these ingredients are still permitted in injectables, and don't have to be disclosed by vaccine manufacturers.[84] The adjuvant, as well as certain inactive ingredients must appear on the package label. General requirements for the package labelling can be found under the US Government Code of Federal Regulations package labeling standards "21 CFR 610.61". So basically if the oil is not in the adjuvant it doesn't have to be listed as adjuvant. FDA has allowed these substances to fall under the category of GRAS (generally regarded as safe) and the vaccine companies get to determine substances as such. The FDA has not reconsidered the GRAS substances since the 1980s as per a government report in 2010.[85] Peanuts contain naturally antigenic proteins. If they are compounded with aluminium the setup for allergy can be fierce.

Appendix 6 lists 4 pages called "Definitions of adverse events following immunisation". It includes death.

Both appendices are easily accessible on the Internet.

Question 3. Who benefits from vaccines?

From the previous pages, the reader can see that it is not the recipients who benefit from vaccines. Many vaccinees develop reactions, which differ only in their intensity and seriousness. Even if vaccines were consistently

[83] Smith JW, Fletcher WB, Peters M, Westwood M, Perkins FJ. Response to influenza vaccine in adjuvant 65-4.J Hyg (Lond). 1975 Apr;74(2):251-9.
[84] Emulsion compositions for polyfunctional active ingredients. What is claimed and desired to be secured by United States Letters Patent.
http://www.patentstorm.us/patents/6720001/claims.html accessed 8 Feb 2013.
[85] United States Government Accountability Office. Report to Congressional Requesters. Feb 2010.
http://www.gao.gov/new.items/d10246.pdf accessed 8 Feb 2013.

effective, which they obviously are not, is it a justifiable swap between having natural infectious diseases which are mostly mild and without permanent sequelae vs. developing organ damage and even death due to vaccines? Many vaccine recipients have obvious reactions, which may result in permanent and disabling sequelae.

Natural infectious diseases are mostly mild and easily manageable at home, provided they are not mismanaged by indiscriminate administration of antibiotics, painkillers, and anti-pyretics.

The only people that consistently benefit are the vaccine-producing drug companies and those who administer the vaccines.

Questions 4. and 5. Are vaccines safe? How are vaccines shown to be safe?

Vaccines are a well documented cause of several health disasters, including many cancers (linked to monkey viruses in vaccines), chronic ill health, organ problems as listed in PI, degenerative diseases of bone and cartilage, multiple behavioural and mental problems, dementia, suicidal depression, violence, criminality, the list is endless.

Stop vaccination and **in no time** the national medical bill will greatly diminish.

Vaccine danger has always been the rule, not the exception.

Even when Jenner in the early 1800s started inoculating individuals with his smallpox vaccine, people started developing smallpox from it. Huge epidemics occurred in the British Isles and only stopped in the areas where people revolted against vaccination and refused to be vaccinated. One such area was the city of Leicester. Far from protecting people against smallpox, Jenner's vaccine was causing smallpox.

The side effects and ineffectiveness of smallpox vaccination have been a major issue discussed in medical papers for a long time. In 1928, Dr Garrow showed that the fatality rate among vaccinated cases of smallpox in England and Wales between 1923 and 1926, in those over 15 years of

age, was five times higher than among the unvaccinated.[86] This stirred a lively discussion in the journal. Drs Stock and Wynne tried to explain this as waning vaccine immunity. They scolded Dr Garrow for:

> *broadcasting in the medical press an assertion …which he must be aware will be quoted, on his authority and without context, by the antivaccinist press. This kind of action can do nothing but handicap his colleagues who are engaged in combating the present epidemic of small-pox, with its serious burden on the public funds, the loss of wages involved, and the damage to industry quite apart from the detriment to public health…*[87]

Evidently, the main concern of vaccination proponents was to avoid embarrassing the medical profession by showing complete lack of concern about their blindness to the actual situation.

Dr Parry summarised the questions raised by Dr Garrow as follows:

1. *How is it that small-pox is five times as likely to be fatal in the vaccinated as in the unvaccinated?*
2. *How is it that, as the percentage of people vaccinated has steadily fallen from about 85 in 1887 to about 40 in 1925, the number of people attacked with variola has declined pari passu and the case mortality has progressively lessened? The years of least vaccination have been the years of least small-pox and the least mortality.*
3. *How is it that in some of our best vaccinated towns – for example Bombay and Calcutta – small-pox is rife, whilst in some of our worst vaccinated towns, such as Leicester, it is almost unknown?*
4. *How is it that something like 80 percent of the cases admitted into the Metropolitan Asylum Board small-pox hospitals have been vaccinated whilst only 20 percent have not been vaccinated?*
5. *How is it that in Germany, the best vaccinated country in the world, there are more deaths [from smallpox] in proportion to the population than in England – for example, in 1919, 28 deaths in England, 707 in Germany; in 1920, 30 deaths in England, 354 in Germany. In Germany, in 1919, there were 5,012 cases of small-pox with 6 deaths. What is the explanation?*
6. *Is it possible to explain the lessened incidence and fatality of small-pox on the same grounds as the lessened incidence and fatality of other infectious fevers – namely, as due to improved hygiene and administrative control?*[88]

[86] Garrow RP. Fatality rates of small-pox in the vaccinated and unvaccinated. Br Med J. 1928 January 14; 1(3497): 74.
[87] C. Killick Millard. FATALITY RATES OF SMALL-POX IN THE VACCINATED AND UNVACCINATED. Br Med J. 1928 January 21; 1(3498): 115–116.
[88] Ibid.

The "experts" commented that, "We think that Dr Parry in his desire for enlightenment, would have been wiser not to introduce assumptions of facts into the framework of his questions." A most interesting comment, given that Dr Parry was the one referring to the well-documented facts, not assumptions.

In fact, Dr Parry's letter was factual, logical and to the point and would still stand up to scrutiny today.

However, the comments of the other doctors reflected the same questionable reasoning still used by many contemporary proponents of vaccination. To this day, the provaccinators accuse those who oppose vaccination of expressing opinions, while the fact is that it is the other way around. It is those who oppose vaccination who study orthodox medical research literature and document their statements with published facts. I therefore wish to propose that the booklet "The science of immunisation" is a good example of such ignorance regarding the documented evidence incriminating vaccination.

Smallpox (variola) eradication campaign of the 1970s

It is obvious that harmful reactions and ineffectiveness of smallpox vaccine became an embarrassment to the medical establishment and was the main motivation behind the "smallpox eradication" campaign. Anywhere vaccination stopped, smallpox occurrence stopped as well.

While the "eradication" machinery was afoot in two still-remaining major African epidemic centres (the Sudan and Ethiopia), several patients in the 1970s with illness first indistinguishable from smallpox were discovered in allegedly smallpox-free forest areas. A virus isolated from some of the patients was later identified as monkeypox. This virus was isolated in 1958 during an outbreak of smallpox-like vesicular disease in captive monkeys in Copenhagen. Later, when the virus was investigated by laboratory methods, it was found indistinguishable from true variola virus. In spite of that, whenever smallpox occurred it was given new names such as monkeypox, buffalopox, camelpox or whitepox.[89]

Smallpox disease was pronounced officially eradicated on 8 May 1980. Small pockets of smallpox continued occurring, but greatly diminished in size and frequency—because the vaccine was not used anymore.

[89] Dumbell and Kapsenberg 1982. Laboratory investigation of two "whitepox" viruses and comparison with two variola strains from southern India. Bull WHO; 60 (3): 381-387.

The US tried to reintroduce smallpox vaccination in the early 2000s. This had to be discontinued because thirty six percent of recipients developed some form of significant illness[90], while some died from it. According to the VAERS database, nearly all deaths from smallpox vaccine since 2011 were purchased and administered by the military.

Leaving diseases to their own limitations and dynamics is a wise thing to do. Historical data confirms that the diseases will gradually extinguish themselves and all epidemics become self-limiting. Moreover, most people recover and develop solid immunity to diseases. The better the health of the population in general, the less noticeable and less serious are the symptoms.

Again, it is the good and valid orthodox medical research that has been dealing with serious and often deadly effects of all and any vaccines right from the beginning of intense vaccination.

Vaccines, such as whooping cough, have been used to deliberately induce encephalomyelitis in laboratory animals. When these unfortunate animals died from the administered vaccines or their active toxic ingredients, the researchers had no problem to causally link the administered vaccines or toxic ingredients with the observed deaths.

Behan and Lamarche wrote:

> *Acute necrotising haemorrhagic encephalopathy (acute haemorrhagic leukoencephalitis) is a demyelinating disease characterised by rapid dramatic onset, short clinical course and fatal outcome. The disease begins acutely several days to three weeks after apparent recovery from viral illness or immunisation. It can also occur in association with thrombocytopenic purpure or after immunizations, application of arsphenamine to the gums, or ingestions of certain [medical] drugs…Fever occurs in the majority of cases. Autopsy findings apart from those involving the central nervous system are nonspecific. They have included pulmonary oedema, bronchopneumonia, hepatic congestion, renal tubular necrosis, and reactive hyperplasia of spleen and lymph nodes. Grossly, the brain on removal appears congested, swollen and oedematous, the lesions affecting mainly one hemisphere. Microscopic examination confirms the presence of petechial haemorrhages of ball and ring type which may be large and confluent.*

[90] CDC. Adverse Reactions Following Smallpox Vaccination. http://www.bt.cdc.gov/agent/smallpox/vaccination/reactions-vacc-clinic.asp accessed 8 Feb 2013.

> *Immunological hypersensitivity has long been considered to be a factor in the etiology of this disease. The latent period, the failure of attempts to isolate a causative organism, the relationship to immunisations, the high incidence of atopy among the reported patients, and the similarity to the pathological findings to those of experimental allergic encephalomyelitis in monkeys all provide indirect [sic, this type of evidence is direct] evidence of an allergic pathogenesis.*[91]

Their studies in monkeys and some other animals showed that vaccination may produce a more virulent reaction, with necrosis of the tissues and neutrophilic, leukocytic infiltrates. This picture histologically resembles acute necrotising haemorrhagic encephalopathy. In highly susceptible rat strains and in nonhuman primates, the addition of killed pertussis vaccine (itself a powerful adjuvant) invariably produces a fulminating, hyperacute encephalopathy. The histological lesions in hyperacute experimental allergic encephalomyelitis suggest an Arthus reaction.

> *Six monkeys, vaccinated with neural antigens in CFA and pertussis vaccine, the course of the disease was different. On the seventh day, animals 7 and 8, vaccinated with whole spinal cord, developed fever of 40.5C and 42.2C. These two animals were ataxic from the onset of fever and on the morning of the tenth day both were severely ill, the first being totally blind with a dense left-sided paralysis while the second had dilated pupils and worsened ataxia. Monkeys 9 through 12 received myelin basic protein instead of the whole cord, and all had neurological signs on the tenth day, most beginning with the signs referable to the optic nerve. One of these animals suddenly became paraplegic, another had an acute cerebellar lesion, remaining on the cage floor and moving around in only one direction. This last animal had been normal in the morning and was found sick some three hours later. Monkey 20 hyperimmunized with multiple injections of homologous myelin basic protein in IFA failed to develop any clinical signs.*[92] [The authors did not indicate the length of observations of this monkey. It could have had a very much-delayed fatal reaction.]

None of the monkeys showed an immediate reaction to any of the antigens tested**.** All reactions were delayed.

These observations are important because in trials of vaccines, the observation period for reactions has usually been 48 hours**.**

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[91] Behan PO, Moore MJ, Lamarche JB. Acute necrotizing hemorrhagic encephalopathy. Postgrad Med. 1973 Oct;54(4):154-60.
[92] Ibid.

Graham et al. published a very important article in which they described characteristic pathological findings in septic shock resulting from the complement formation caused by a number of injuries, but most importantly injuries caused by a hyperacute form of postvaccinal encephalomyelitis. They wrote:

> *There was, however, plugging of capillary vessels with fibrin thrombi and some of the vessels were cuffed by inflammatory cells. Swelling of the brain with subarachnoid, subdural, and intraventricular haemorrhages was also found…" Russel (1955) had earlier shown that the vascular lesions in acute haemorrhahgic leucoencephalitis (AHLE) were similar histologically to those found in polyarteritis nodosa. She also demonstrated that the perivenous infiltrates found in AHLE were similar were similar to those in the parainfective and postvaccinal encephalomyelitis.*[93]

Lewis et al. developed a technique with which they detected CIC's [circulating immune complexes] in a 4-month-old child in whom a petechial and urticarial rash developed 5 hours after receiving DTP vaccine (diphtheria and tetanus toxoids and pertussis vaccine, absorbed). Subsequent antigenic analysis of the CICs showed them to be composed of vaccine-specific antigens.[94]

Munoz et al. described biological activities of crystalline pertussigen from Bordetella pertussis.[95]

[93] Graham DI, Behan PO, More IA. Brain damage complicating septic shock: acute haemorrhagic leucoencephalitis as a complication of the generalised Shwartzman reaction. J Neurol Neurosurg Psychiatry. 1979 Jan;42(1):19-28.
[94] Lewis K, Jordan SC, Cherry JD, Sakai RS, Le CT. Petechiae and urticaria after DTP vaccination: detection of circulating immune complexes containing vaccine-specific antigens.
J Pediatr. 1986 Dec;109(6):1009-12.
[95] Munoz JJ, Arai H, Bergman RK, Sadowski PL. Biological activities of crystalline pertussigen from Bordetella pertussis. Infect Immun. 1981 Sep;33(3):820-6.

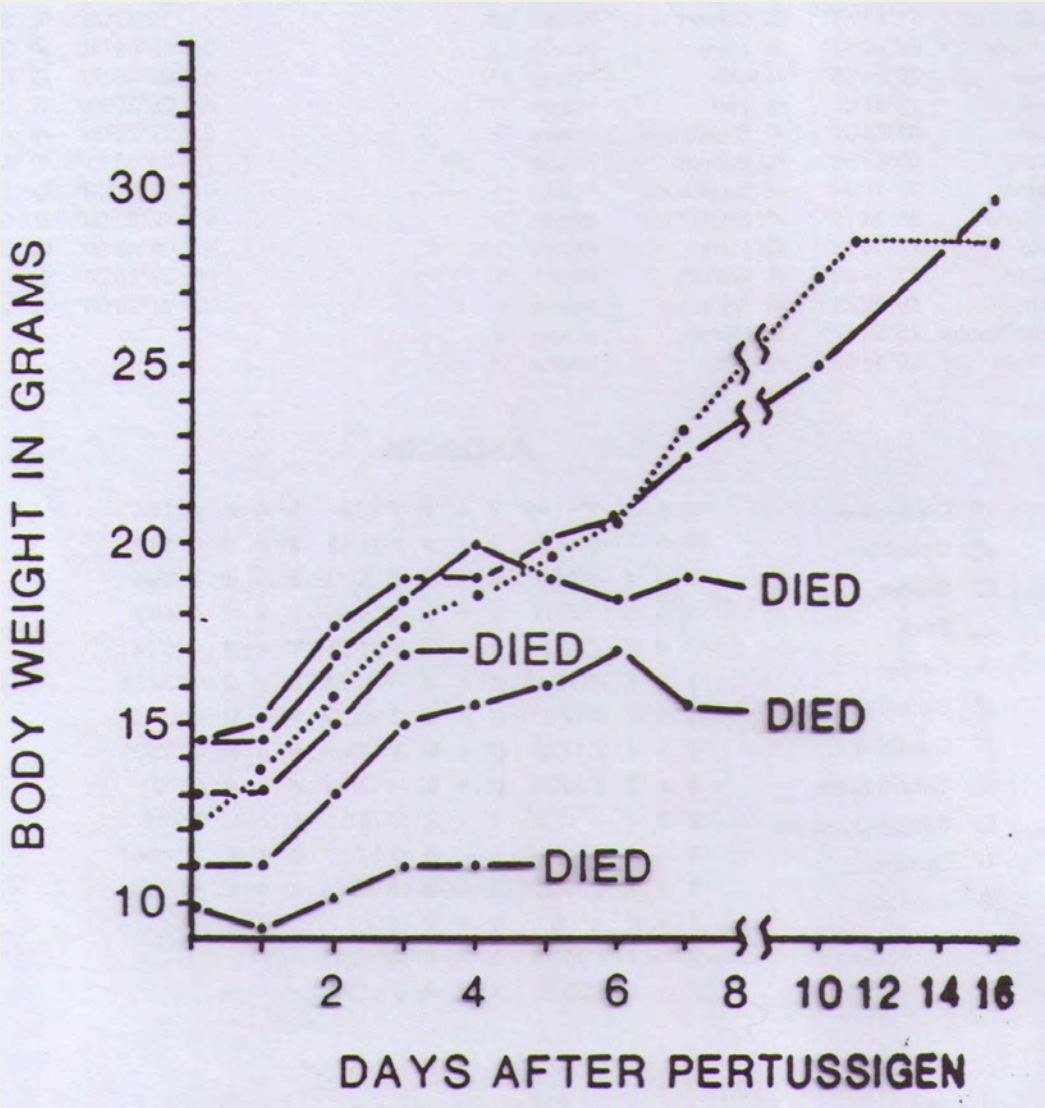

Figure 17: Body weights of five individual mice after i.p. inoculation of 1 µg of pertussigen (solid lines). For comparison, the dotted line shows the average weight gain of five mice that received diluent only. The weight gains of the individual mice in this control group were uniform and were very close to the average values shown in this figure.

Figure 17[96] shows day-by-day body weights of five individual mice after intraperitoneal inoculation of 1 microgram of pertussigen. The dotted line shows the average weight gain of five control mice that received the diluent. Four of the five inoculated mice died, interestingly but not surprisingly along the critical days as documented with the microprocessor-based Cotwatch breathing monitor.[97]

It is well known that babies who died of SIDS (read: sudden immunisation death syndrome) after vaccination, stopped putting on weight after each

[96] Ibid.
[97] Ibid.

vaccination and showed the same dynamics of stress response, proving the causal link to the administered vaccines.

This leads me to the infamous factitious shaken baby syndrome. . .

Shaken Baby Syndrome.

When some parents/caregivers vaccinate their babies, to simply show compliance or to avoid the usual hostile behaviour from such people (who as a rule intimidate them into vaccinating), they may get a rude awakening when they become a target of very serious accusations.

Babies can be seriously injured or die after vaccination, and their parents are too often accused of killing them, by allegedly shaking them to death. Those parents end up in prison for the crime that someone else committed.

It is absurd that the same doctors or other health workers who administered the lethal vaccines often become the accusers of the innocent parents.

When the frightened and puzzled caregivers arrive in hospitals with their very ill or dying infant, at a loss to understand just what happened to their precious baby, they will often and straight away be accused of shaken baby syndrome just on the basis of symptoms (major malaise, unconsciousness and fitting) and retinal haemorrhages, before any proper tests are done.

A group of South Australian pathologists and microbiologists looked at crossed linked fibrin degradation products (XLFDP) in SIDS infants and also in children who died from established causes (controls). Their findings, seen in figure 18[98], demonstrated that the SIDS infants had thousands fold higher levels of XLFDP compared with controls. I am puzzled why such tests are not done before the accusations of SBS are made.

[98] Goldwater PN, Williams V, Bourne AJ, Byard RW. Sudden infant death syndrome: a possible clue to causation. Med J Aust. 1990 Jul 2;153(1):59-60.

Age at death	Sex	Cause of death	XLFDP(mg/L)
Controls			
a) Death from trauma			
1 10 years	F	motor vehicle accident	32
2 3.5 years	F	motor vehicle accident	64
3 9.3 years	F	motor vehicle accident	≤32
4 7.8 years	F	motor vehicle accident	64
5 14 years	M	motor vehicle accident	≤32
6 2.7 years	M	motor vehicle accident	128
7 13.4 years	M	extradural haematoma	≤32
b) Death from non-bacterial medical causes			
8 5 months	M	cerebral palsy	128
9 6 months	F	Kawasaki disease	32
10 4 weeks	F	congenital heart disease, Trisomy 21	32
11 4 months	M	virus infection	64
12 3 months	M	congenital heart disease	64
13 14 months	M	congenital heart disease	32
SIDS			
1 6 weeks	F		1024
2 4 months	M		≥4096
3 7 months	F		4096
4 6 weeks	M		1024
5 1 month	M		2048
6 6 months	F		512
7 4 months	M		512
8 9 months	F		≥1024

Figure 18: TABLE: Cross-linked fibrin degradation products (XLFDP) in SIDS infants and in children who died from established causes (controls).

Perhaps, the most tragic thing is that the parents accused of murdering their infants are the compliant ones who unquestioningly trusted the medical personnel.

The requirement of the Australian law "presumed innocent until proven guilty" is out the window when these innocent, grieving parents are treated like criminals.

Everything they do or don't do is used as evidence of their guilt, particularly if they can't come up with what their accusers would consider a reasonable explanation of the observed symptoms.

Illogical, absurd, inexpert and unlawful reasoning and methods are applied by their accusers.

As an example I quote an article from the latest issue of the journal, Australian Doctor from 30 November 2012: 4 entitled "Parent's cry for help can be sign of abuse."

The article says that Dr Amanda Stephens presented what the article calls the "findings", at the annual scientific meeting of the Australasian College for Emergency Medicine. "The review of 68 'shaken babies' seen at the Children's Hospital at Westmead, Sydney, found most cases had no obvious prior warning flags indicating abuse. However, 12 infants had

received medical attention for signs of abuse long before the diagnosis was made".

"It was only when he [the kid] had seizures that they figured out he had been abused on a number of occasions."

If those doctors and Dr Stephens had checked the vaccination status of these greatly pained crying babies (which, as they as a rule don't), it would have transpired that they had one to three doses of multiple vaccines, besides hepatitis B at birth, administered before every episode of inconsolable crying and seizures. The lack of expertise on the part of the attending medical personnel is especially alarming because the babies may be given further doses of the offending vaccines. According to the article, "most infants were aged less than six months and came from both lower and middle class backgrounds".

The alarmingly inexpert nature of the article showed also in the cliché "Those who don't cruise don't bruise" implying that bruises never occur in small babies before crawling stage. Whomever came up with this absurd and inappropriate cliché obviously knows nothing about bruising caused by vitamin C deficiency and Henoch-Schonlein purpura, (anaphylactoid purpura), a leukocytoclastic vasculitis with characteristic bruising, and ecchymoses, as well as the acute haemorrhagic edema of infancy (AHEI)[99], which is linked by good medical research to vaccination, scurvy and the administration of antibiotics and other medical drugs.

This is reminiscent of the infamous cliché of another accuser of innocent parents, the UK proponent of Munchausen per proxy: "one death in the family is cot death, two death are suspicious, third death is murder."

Beal and Blundell wrote that in their experience in South Australia, the incidence of cot death in previous siblings was 10 times what was expected for the community. In second degree relatives it was five times and in third degree relatives four times that expected. [100]

According to Emery et al.,

> *The risk of cot death in a sibling is about one in 200.*[101]

[99] Kafaie P et al. Acute hemorrhagic edema of infancy: a case report. 2010. J Pakistani Assoc Dermatologists; 20: 172-175.

[100] Beal SM, Blundell HK. Recurrence incidence of sudden infant death syndrome. Arch Dis Child. 1988 Aug;63(8):924-30.

[101] Emery, Waite, Limerick, and Carpenter. A letter to the Editor. 1987. Arch Dis Child; 62: 99.

These reports are a far cry from the provaccinationist's claim of a one in 72 million chance, of having two children with SIDS in the same family.

Besides major injustice committed against innumerable of his victims, it cost the UK solicitor Sally Clark her freedom for four and half years, and ultimately her life. Her two boys died from vaccination; one four-five hours post and the other 23 days after the injections (this being one of the important critical days that are part of the dynamics of stress response after vaccination).

According to Neville Hodgkinson, as written in Spectator.co.uk, from the article entitled "What killed Sally Clark's child?":

> *Not many people know these facts because at Sally's trial the defence did not mention immunisation as a possible cause of death.*
>
> *Even paediatricians who gave testimony on Mrs Clark's behalf told defence lawyers that if vaccinations were mentioned as a possible cause of Harry's death, they would dispute it. Not wanting to confuse the jury, and with judges having a history of bowing to dominant medical opinion, the defence decided to stay silent on the issue.*
>
> *With hindsight, it is clear that this was a bad decision. Not just for Sally Clark, her husband, her surviving child, her family and friends, but because of the suppression of evidence of potentially vital importance to public health. Deaths and major injuries from vaccines are rare, but professionals take an ostrich like attitude towards those that do occur – and instead blame the parents – the scene could be set for disaster.*

Perhaps the most disconcerting statement in the above Australian Doctor article is the recommendation of Dr Stephens that "doctors refer suspected cases of abuse to a paediatric hospital under the guise of needing further testing, rather than confronting the parents. They [the doctors] could then call the hospital in confidence to flag the matter, she said."

This statement is unbelievably callous and it should send shivers down the spine of any decent person. The affected parents don't stand a chance.

I feel sorry for the largely ignorant parents who blindly trust the doctors and do not check the situation surrounding vaccination before agreeing to allow their babies to be injected.

Provaccinators will mercilessly accuse such parents, in the event of certain vaccine injuries, of the infamous factitious diagnosis named shaken baby

syndrome. Their children are taken away by the DoCS (Department of Child Services). Even if the parents win in the courts, the DoCS will not automatically return the snatched children back to their rightful parents.

Another stolen generation is being created by the tyrannical medical system.

A thought comes to my mind that such behaviour is reminiscent of Josef Goebbels who was Hitler's propaganda minister. After his last public address (before capitulation) to the cheering Nazis and fatally misled German people, Goebbels later cynically remarked to his entourage:

> *What an hour of idiocy. If I had told these people to jump from the fourth floor of the Columbus House they would have done it.*[102]

A similar contempt is shown by the vaccinator for the compliant parents whose babies were injured and died from vaccines.

Question 6. What does the future hold for vaccination?

There is no need for any vaccines, because there are more healthful means that strengthen immunity and easily manage all infections. One should not only be asking about the future, but also be inquiring about the past. Mortality from supposedly vaccine-preventable diseases was nearly gone in developed countries, before vaccines were instituted. There is no need to prevent the normal diseases of childhood. They are the ones that prime and mature the immune system, and represent developmental milestones even in a subclinical form.

Not only do vaccines not prevent disease, but in most instances, they propagate specific diseases which would have otherwise naturally disappeared from populations.

The mass use of vaccines resulted in many damaging consequences for the health of humanity, such as pandemic of diabetes especially in small children, pandemic of especially characteristic SV40 cancers, including mesothelioma, mistakenly considered an asbestos disease, the occurrence of "new" infectious diseases linked to mutated microorganisms, chronic fatigue and suicides, degenerative diseases of all organs, behavioural and learning problems, including autism. The product insert for Sanofi Pasteur Tripedia lists autism as one of the serious post marketing reactions. The package insert for Priorix GlaxoSmithKline Australia Pty Ltd MMR vaccine

[102] John Toland. *Adolf Hitler: The Definitive Biography*. Anchor; 1st edition (December 1, 1991). p. 1006 ISBN-13: 978-0385420532

lists *inter alia* colitis among the gastrointestinal adverse reactions. [103] The list is endless and includes mental illness. All this is aggravated by many toxic medications and especially antibiotics.

And last but not least: I suggest that the readers acquire Marcia Angell's (the former editor in chief of New England Journal of Medicine) book "The truth about the drug companies. How they deceive us and what to do about it" (Scribe Publications Pty Ltd. Melbourne).

Orthodox medicine has failed abysmally.

How many more vaccine tragedies are needed for vaccination to finally stop? Parents can stop it by refusing to expose their precious babies to this antiquated, scientifically invalid, toxic and useless medical procedure.

If vaccination is the best thing in medicine after sliced bread, then I shudder over the rest of their procedures.

Addendum

Scurvy – the main health scourge of humanity.

The provaccinationists never discuss scurvy in their educational materials. They will categorically deny the existence of the effects of vitamin C deficiency upon the sick and the vaccinated. Instead they prefer to pretend that scurvy was only a health problem in the Middle Ages.

Scurvy was known to Hippocrates (c. 460 BC- 370 BC) and to ancient Egyptians much earlier, and more recently during long sea and land voyages. The main life losses during the military campaigns of pre- and historic armies were not due to battle but due to scurvy deaths.

Bubonic plague (Black death) was complicated by typical scurvy haemorrhages, turning the sufferers' flesh black. Indeed, the original definition of scurvy was a haemorrhagic disease.

Scurvy also played a major role in smallpox, which practically disappeared with improved nutrition and inclusion of fruit and vegetables (especially saurkraut) into the staple diet.

While it is true that in centuries past, people were more susceptible to diseases and disease complications as a result of vitamin deficiencies, the fact remains that vitamin C is far more important even today than health authorities will admit.

[103] Monthly Index of Medical Specialties Annual 2009: 10-1359

Scurvy still persists even in developed countries especially in vaccinated children. This phenomenon is caused by the toxic effect of vaccines, which cause the body to rapidly consume vitamin C stores, resulting in acute scurvy.

Ratanachu-Ek et al. wrote that 93% of their patients between 1-4 years of age were fed over-cooked food with few vegetables and fruit. Supplementation with UHT (ultra high temperature) milk was found in 89%. Frequent manifestations were limping and inability to walk and pain in the lower limbs. Response to vitamin C treatment was dramatic.[104]

Gilman and Tanzer described haemorrhagic diatheses from scurvy resulting in subdural (and other) hematomas in infants back in 1932.[105]

Poyrazoglu et al. described acute hemorrhagic edema of infancy (AHEI) after infection, drug administration or immunization.[106]

Dr Archie Kalokerinos documented numerous cases of acute, scurvy after vaccination that was often reversible with injected vitamin C.[107] Unfortunately one in two vaccinated children died before the condition was discovered.

[104] Ratanachu-Ek et al. 2003. Scurvy in pediatric patients: A review of 28 cases. J Med Assoc Thai; 86, Suppl 3: S734-40.
[105] Gilman BB and Tanzer RC. SUBDURAL HEMATOMA IN INFANTILE SCURVY: REPORT OF CASE WITH REVIEW OF LITERATURE. September 17, 1932, Vol 99, No. 12. Pp. 989-991
[106] Poyrazoğlu et al. Acute hemorrhagic edema of infancy. Pediatr Int. 2003 Dec;45(6):697-700.
[107] Kalokerinos A. Every Second Child. Keats Pub (September 1981) **ISBN-13:** 978-0879832506