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Date 13th October, 2015

Dear Sir/Madam,

Re: Social Services Legislation Amendment (No Jab, No Pay) Bill 2015

WHO AM I?

I'm Jesse Sleeman, a 66-year-old father of five adult children, and grandfather to five young children. I have an MA in Psychology from Sydney University, majoring in Social Psychology and Clinical Psychology. I also trained in the UK in Medical Herbalism. Over the past 40 years I've taught medical philosophies and western herbal medicine at 12 college of natural therapies across Australia.

My submission is based on my educational background, my experience as a herbalist and as a parent who never had one of his children vaccinated, and also as an author of many articles and a book entitled, *Cry for Health, Vol I, Health the Casualty of Modern Times*.

Part 1

Part 1 of my submission is based on an aspect of social psychology and political history.

When Australian Prime Minister Tony Abbott announced his 'no jab, no pay' policy, and Labor Party leader Bill Shorten declared his support for this draconian legislation, they were propelling Australia into medical fascism. Philosophical and religious exemptions to vaccinations will be quashed, and families who choose not to vaccinate their children will lose 70 per cent of child welfare payments.

It's a path the Nazis began when they jackbooted their way across Europe 70 years ago, vaccinating all children with diphtheria vaccine, and conducting other medical experiments at concentration camp hellholes. Aside from war casualties, diphtheria had been the leading cause of death and disease in Europe during World War II.

After the war, the Allies vowed that such experiments should never again happen. The freedom to choose your or your children's medical treatment was enshrined first in the Nuremberg Code, and later in the Declaration of Helsinki (1964). Though Australia has no bill of rights, it has an historical convention that citizens have the right to choose their medical treatment. Freedom of choice is the very foundation of traditional medicine.

The Australian Charter of Healthcare Rights: A Guide for Healthcare Providers, published by the Australian Commission on Safety and Quality in Health Care, created by Health Ministers in 2006, states: "Participation by patients in their health care…includes informing patients and consumers of their right to refuse or withdraw consent at any time…".

So, the bipartisan legislative proposal will not only breach the Nuremberg Code, the Declaration of Helsinki, and the historical conventions about medical choice, but it will also breach the UN Convention on the Rights of the Child, in this instance Articles 5, 18, 26, and 27. This is to be expected of both major political parties since they've been breaching this UN Convention (Article 22) by sending refugee children to prison hellholes (euphemistically called "detention centres").

And both parties are ignoring a legal precedent established by the High Court (Rogers vs Whitaker, 1992), that medical choice about any medical intervention (and a vaccination is a medical intervention) is meaningless unless the likelihood of adverse events, however long the odds of that happening may be, is explained to the patient.

This is also yet another episode in the 5000-year saga of male domination, suppression and violation of the rights of women and children, so well explored by Riane Eisler in *The Chalice and the Blade.*

The looming uproar is not about the pros and cons of vaccines. It's about choice; our rights— the freedom to choose our medical treatment.

Part II

Part 2 of my submission is an excerpt from my book *Cry for Health*. The information in this chapter is based on 17 years of research. This is Chapter 5, and has 740 references appended after the chapter. From reading this Chapter, you'll get an idea of the arguments and ideas upon which parents decide to refuse vaccinations for their children. It looks at the safety, efficacy and history of many failed vaccines, and argues that vaccinations are the very antithesis of being the greatest public health achievements of the 20th century, as Big Pharma and Big Mediine claim.

It's a along chapter…52 pages…but it is all "evidence-based".

To Vaccinate or Not to Vaccinate

The hype

To vaccinate or not to vaccinate? That is the question every parent must face—unless, of course, you live in a country where the government makes the decision for you. And behind that question are a host of others. Is vaccination safe? Is every one of the 13-odd vaccines, which the government recommends every child should get several times before he or she turns two, really safe? Is there a link between certain vaccines and autism or other disorders? Do vaccinations really work? What if I don't have my baby vaccinated, what will happen?

The medical hype, of course, is that immunisation is one of the most important health interventions of the 20th century; that it has eliminated smallpox infection worldwide, driven polio from most western countries and made formerly common infections like diphtheria, whooping cough, and measles rare occurrences.

So, who do you turn to, who do you trust? The vaccine manufacturers and their scientists, the public health department with its pro-vaccination campaigners, and your doctor who administers the jab? Or do you trust those who warn against vaccinations, claiming that vaccinations are neither safe nor efficacious?

In every country except Australia, doctors have no legal obligation whatsoever to tell you all the issues about any medical procedure. Their advice is based solely on 'medical opinion', and the doctor paternalistically decides what is in the patient's best interests. But in Australia, thanks to a High Court ruling in 1992 (Rogers v. Whitaker), Australian doctors have a legal duty of care to warn patients of all the side effects of all procedures whether the patient asks or not.[1] The problem is, most doctors only know about the pros of vaccinations and have no idea about the cons.

Since your baby's future health is at stake, it's certainly an emotive issue. Indeed the last thing you need in making your decision is to have fear added to the mix. But alas, that's exactly what governments and many doctors do. Hence informed choice flies out the window, the result being that those of us who have children invariably succumb to fear-mongering tactics.

Parents who do refuse to have their children vaccinated are typically condemned as being irresponsible. The professional inquisitors are well armed: 'Do you know how dangerous diphtheria and tetanus and measles and mumps, and all the others diseases for which modern medicine has created a vaccine, are …?' 'Do you appreciate how miserable your children will be when they contract whooping cough …?' 'Why would you put your children at risk when a simple shot or two, or thirty (which is roughly the number they'll get before they start school) will protect them from nasty childhood illnesses?'

If such rhetoric fails to convince the parent, there's always the stab to the parent's social conscience: 'Do you know that by refusing to vaccinate your child you're putting other people at risk as well?'

What your doctor doesn't tell you, is that unlike every other drug, not a single vaccine has ever been tested for either its efficacy or its safety in a double-blind, placebo-controlled field trial. No one has dared to compare the health of vaccinated and non-vaccinated children because such a trial is construed as unethical. But in a logical absurdity worthy of *Alice in Wonderland*, it is considered ethical to test vaccines on mass populations of children and adults.

Nor does your doctor tell you that many doctors do not follow their own advice: many refuse to have their own children vaccinated; or won't follow the nation's vaccination guidelines by delaying the administration of certain vaccines until their own child is aged two or more, or by picking and choosing which vaccines their own children will and won't get.

In a rare instance of doctors disclosing their own biases and indeed their own double standards, a Swiss survey sent to 2,000 doctors—roughly half of whom, tellingly, didn't reply—revealed that 15 per cent refused to allow their own children to have certain or all vaccines.[2] Five per cent wouldn't allow their children to have the Hib vaccine (against Haemophilus influenzae type b) or the MMR vaccine (against measles, mumps and rubella). And 10 per cent refused to give, or delayed giving, their children the DPT vaccine (against diphtheria, pertussis (whooping cough), and tetanus).

Similarly, a US survey of paediatricians revealed that 12 per cent of those who were parents hadn't vaccinated their own eligible children against chickenpox,[3] and 4 per cent had refused permission for an immunisation for their own children younger than 11.[4] And a Canadian survey showed that 41 per cent of nurses didn't fully agree with medical claims that vaccines were safe and effective.[5] In fact, 40 per cent of nurses thought that such practices as homoeopathy, good eating habits and a healthy lifestyle can eliminate the need for vaccination. Even the former prime minister of Britain, Tony Blair, whose government was waging a campaign to have all children vaccinated against measles, mumps and rubella, refused to disclose whether or not he had had his young son Leo inoculated with the MMR vaccine.

Chances are your doctor isn't even adhering to the medical profession's vaccination guidelines for health workers. Various reports have shown that half of British doctors in one survey, and less than a quarter of US doctors in another survey, were vaccinated against hepatitis B;[6,7] that only 20 per cent of susceptible physicians were vaccinated against rubella;[8,9] that only one of 11 obstetricians and gynaecologists at the Los Angeles County–University of Southern California Medical Center was vaccinated against rubella;[9] and that only a quarter of the trainee doctors in a US hospital emergency department, and a third of all emergency health workers in four hospitals in Canada, were vaccinated against influenza.[10,11]

Even when doctors are bombarded by campaigns to get themselves vaccinated many seem reticent to comply. A study at a Texas training hospital, where only 32 per cent of doctors and medical students had ever been vaccinated against influenza, found that despite sending out a memorandum, followed by a personal letter, and then a telephone call, and then providing free vaccinations at medical

conferences, only 62 per cent of the medical staff decided to be vaccinated.[12]

Which all goes to show that many members of the medical profession, and undoubtedly many politicians, are not convinced by their own propaganda.

Of course, we don't know which aspect of the propaganda each of them didn't buy, for the flagstaff from which the vaccination flag flutters is held aloft by three tangled mainstays: one, if not for vaccinations we'd still be ravaged by deadly diseases that plagued past generations of people; two, each vaccine that is targeted at a specific germ will prevent the vaccine's recipient getting the disease that germ causes—in other words, vaccines work; and three, vaccines are safe.

If not for vaccines ...

For 200 years we have been sold the story that vaccines saved generations of people from such diseases as smallpox, diphtheria, whooping cough, polio, tuberculosis, tetanus and, in recent years, from measles, mumps, German measles (rubella), influenza and hepatitis B. But did they?

Let's investigate the record of the first vaccine to be used, the one that has been hailed as the drug that defeated smallpox, first from Europe and now from the world.

The first vaccine to fail

If Edward Jenner's 'great discovery'—borrowed from a Gloucestershire dairymaid's superstition that the pus from a cowpox infection protected milkmaids from smallpox—was so revolutionary, then why did the prevalence and severity of the disease in Britain increase in the years following the introduction of the vaccine in 1796?

Previously, the worst smallpox epidemic Britain had experienced throughout the 18th century was in 1793. London in particular bore the brunt, with two and a half per cent of the population affected, but only one-half of one percent of these people died.[13] A century before this, the eminent British physician, Dr Thomas Sydenham had noted, '… provided no mischief be done either by physician or nurse, it [smallpox] is the most safe and slight of all diseases.'[14]

But mischief was done. After years of widespread vaccinations, an epidemic in 1837–1840 claimed 42,000 lives.[15] Determined to halt further outbreaks, the authorities in 1853 made vaccinations compulsory for children.

Alas, 17 years later, with over 90 per cent of its population vaccinated, Britain experienced its biggest smallpox epidemic in two centuries. In 1871, during the second year of the epidemic, which had then claimed 10,000 lives, the esteemed medical journal *The Lancet* ran an editorial warning that smallpox had reached plague proportions and that the smallpox vaccine seemed ineffectual in halting it. Of the 9,392 smallpox patients in London hospitals, it noted, 6,854 (or 73 per cent) had been vaccinated.[16] And the journal had calculated that 122,000 vaccinated people throughout the realm had contracted the disease. When the 1870–1872 epidemic finally subsided, it had claimed 44,000 lives.[15]

Until the turn of the century, when public sanitation measures, implemented through various

Public Sanitation and Public Health Acts, were completed, smallpox continued to plague the squalid, overcrowded sections of Britain's cities even though well over 90 per cent of their populations had been vaccinated. As one hospital physician, Dr Walter R. Hadwen, chronicled of his experiences: 'The vaccinated and re-vaccinated hospital officials fell before the disease side by side with the vaccinated and the re-vaccinated inhabitants.'[17]

In the city of Sheffield—'the best vaccinated town in the kingdom', Hadwen wrote—where 98 per cent of the population were vaccinated, an eighth of the population contracted the disease.[17] Where did that epidemic of 'beggars disease' spring from? A populated area covered with cesspits. And did the vaccine protect them? No. It was useless.

Yet in Leicester, a city where the population had refused vaccinations because of the high death rate in the 1870–72 epidemic and had instead opted for the tried and tested practice of quarantining those with smallpox, less than one person per year had died from the disease.[18]

But the lie that the smallpox vaccine protected people continued to spread like a contagion through the medical profession. It continued despite damning evidence being presented to the Royal Commission on [smallpox] Vaccinations by Alfred Russel Wallace, the famous British naturalist and co-discoverer (with Charles Darwin) of the theory of natural selection. He had presented statistical evidence showing that in the 1891–93 epidemic, for instance, Leicester, with 0.007 per cent of its population vaccinated, had 19 cases of smallpox, and one death, while Warrington, with 99.2 per cent of its population vaccinated, had 123 cases of the disease and 11 deaths.[19]

Wallace had also revealed that the death rate in the 1871–1872 smallpox epidemic in Prussia had been double that of Britain. And yet 95.7 per cent of the Prussian population had been vaccinated. Similarly, in Sweden, the death rate during the 1874–1876 epidemic had been double that of Britain during its worst epidemic 100 years earlier. And Sweden prided itself on the fact that the whole population was vaccinated.

The fraudulent claims for the vaccine's success were also identified by other academics. In Italy, for instance, Professor Carlos Ruata had noted that even though 98.5 per cent of his nation's population were officially declared to have been vaccinated, it had suffered over 48,000 smallpox deaths in the epidemic of 1887–1889.[20] As Ruata reasoned, why did more men than women die in the epidemic? After all, every man had to undertake army service at the age of 20, and every one of them had to be vaccinated twice a year.[21]

Notwithstanding the warnings from Wallace and Ruata, and from such distinguished epidemiologists as Charles Creighton and William Farr, and from Edward M. Crookshank, Professor of Bacteriology and Comparative Pathology at King's College, London, the lie continued. This led Wallace to later suspect that looming behind the fraud were vested interests.[22]

Something, however, did come out of the citizenry's revolt against compulsory vaccinations, something that would continue down the years: the British Parliament passed legislation that incorporated a 'conscience clause' for parents who refused to have their children vaccinated. Sound familiar?

Thus, by 1919, Britain had become one of the least vaccinated countries in the developed world. And yet that year it had only 28 deaths from smallpox.[23] Tellingly, the best vaccinated country in the world, Germany, had 707 deaths from smallpox that year.

And in that very year the Philippines was being ravaged by its worst smallpox epidemic ever. Throughout the 19th century it had also been plagued by smallpox epidemics. According to reports from

the Philippines Health Service, about 10 per cent of those who contracted the disease died.[24] Then in 1898, immediately after defeating the former Spanish rulers, the US began to impose its own rule and set about cleaning up the place and vaccinating the whole population. Initially, the prevalence of smallpox declined although, as a portent of things to come, the death rate for those unfortunates who did contract smallpox soared by between 25 and 50 per cent.

Then in 1917, during a lull in smallpox outbreaks, the US army had another go. In the first round it forced a third of the Philippine population to have the vaccine, yet again. Immediately afterwards, 47,000 people developed smallpox, one third of whom died.[24,25] But true to form, the US army didn't flinch. It then forced vaccinations on the remaining two-thirds of the nation's 10 million population. A further 65,000 people contracted the disease, two-thirds of whom died. In total 60,855 Filipinos died in the worst smallpox epidemic that nation ever had.

Japan's experience was much the same. It too had suffered from smallpox epidemics, and in 1872 had begun a compulsory vaccination programme. But the epidemics kept coming. So in 1885 the government passed a law ensuring that everyone was vaccinated again, and would thenceforth be re-vaccinated every seven years. But the epidemics kept coming. In the following seven years 156,000 people contracted smallpox, of whom 25 per cent died.[26,27] And that was despite the fact that two-thirds of the population had been re-vaccinated.

Obviously dismayed by the vaccine's failure, the Japanese government then passed a law ensuring that everyone was to be re-vaccinated every five years. But the epidemics kept on coming, and with renewed severity. Every year between 1889 and 1908, an average of 8,500 people developed smallpox, 28 per cent of whom died.[26,27] In that 20-year period, a total of 172,000 people contracted the disease, and 48,000 of them died.

In 1908, 'when the Empire should have been reaping the best fruits of its rigorous vaccination laws', to quote John Pitcairn, then President of the Anti-Vaccination League of America, 'the smallpox vaccination cases numbered 18,000—a number not exceeded since 1897—and the deaths were nearly 6,000, or over 32 per cent.'[28]

And yet Australia, which was the least smallpox-vaccinated country in the world, had on average only one smallpox death a year throughout its history.[13] Smallpox eventually dwindled from underdeveloped countries not because of the vaccine, which had been given to less than 10 per cent of the people in those countries, but because of improved sanitation and nutrition.[29–33]

But has smallpox really been eradicated, as the WHO officially proclaimed it had on 10 May 1980? Some researchers suspect that it hasn't. Many other pox viruses exist in nature, and when humans catch such diseases as white pox, camelpox or monkeypox, the clinical signs and symptoms are indistinguishable from those of smallpox. Nor, until the recent advent of gene sequencing, could the pox viruses be distinguished.

Throughout the 1980s, however, there were outbreaks of 'monkeypox' in Cameroon, Ivory Coast, Liberia, Nigeria, Sierra Leone, and, as recently as 1996, in Zaire. Some medical researchers suspect that monkeypox may simply be smallpox renamed.[23,34,35]

As for the first recipients of Jenner's experimental vaccine, his elder son Edward, who had been given swine pox at the age of 18 months and then cowpox when he was 9-years-of-age, was afterwards never well again and died of tuberculosis at the age of 21.[36] James Phipps, the 8-year-old lad who was the first to receive Jenner's cowpox vaccine, and had been given at least 20 cowpox vaccinations during his short life, died at the age of 20, of tuberculosis. If only Jenner had known that cows as well as humans

harbour TB.

The second vaccine to fail

The story of diphtheria is not dissimilar to that of smallpox. It too had a notorious history of spreading like wildfire through crowded and impoverished communities. Infants and children in particular bore the brunt of it, estimates being that as many as 70 per cent of those who contracted the disease were under the age of 15.[37] Of course, if they survived they had life-long immunity to the disease. Diphtheria was often deadly, however, because the toxin the germ produces causes swelling of the throat and tonsils, and can thus block airways. Not surprisingly, it was known in some cultures as 'the strangler'.

During the 39 epidemics that swept through squalid European communities between 1557 and 1803, for instance, as many as 80 per cent of those who contracted the disease died.[29] And in the years following the Napoleonic Wars in the early 1800s, as many as 25 per cent of people who came down with the disease died.

During the late 19th century, however, well before the advent of mass vaccination campaigns of the 1940s and '50s, deaths from diphtheria began to rapidly decline. In Toronto, for instance, the death rate of 132 per 100,000 people during the 10-year period of 1886–1895 had halved a decade later, and it continued to halve as each decade went by.[29] When the diphtheria toxoid arrived on the scene in 1924, deaths from diphtheria had declined by over 85 per cent. And by 1940, just before the mass vaccination campaigns for children began, the death rate had declined a further 12 per cent. The United States experienced the same 97 per cent reduction in diphtheria deaths between 1900 and 1940.[38]

In England and Wales, where statistics on deaths had been collated since the mid-1800s, the childhood death rate from diphtheria had declined from 932 deaths per 100,000 children in the 20-year period of 1861–1880, to 293 per 100,000 in the 1921–1940 period.[39]

Why did the death rate drop by only 68 per cent when the US and Canada had much greater declines during this period? Poor nutrition was undoubtedly one factor. But there was another factor, and here's the clue. In 1897, three years after British medical authorities had begun inoculating communities with the anti-diphtheria horse serum, which would have contained horse-protein antigens, deaths from diphtheria began to skyrocket. Indeed the death rates in the ensuing epidemic that lasted until 1910 were 75 per cent higher than they had been 19 years earlier.[40]

In London alone, according to a report by the city's health authorities, 89,445 people contracted the disease during this 15-year epidemic, and 10,837 died.[40] But a closer inspection clearly identifies the culprit: of all the people who contracted the disease 75,310 had been vaccinated, and 10,095, or 13.4 per cent of them had died of the disease; however, of the 13,135 people who hadn't been vaccinated only 742, or 5.65 per cent had died. This means that vaccinated people who contracted the disease—and the vaccine was supposed to protect them—had an approximate 240 per cent greater risk of dying of diphtheria than unvaccinated people.

By the mid-1920s British health authorities had seen the error of their ways and switched to using the new diphtheria toxoid developed by researchers at the Pasteur Institute in Paris. It was the first vaccine to contain the germicidal and carcinogenic chemical formaldehyde; a chemical that would certainly damage the immune system. The vaccine would later be hailed as a godsend in the war on diphtheria. But alas, in 1938 and 1939 yet another diphtheria epidemic struck Britain, killing 32 in every

100,000 children.[41] Steely in their war-time resolve, the British authorities began mass vaccination campaigns in early 1941. After an initial hiccough in 1941 and early 1942, when an epidemic resulted in a considerable increase in diphtheria deaths, the disease began to peter out.

Throughout the war years and up until 1948, health authorities estimated that only 60 to 65 per cent of children had been vaccinated.[41] Nevertheless, despite the fact that the death toll amongst both vaccinated and unvaccinated children in England and Wales declined—in unvaccinated children from 3,000 in 1940, to 551 in 1945, and to 63 by 1949—health authorities proclaimed the diphtheria toxoid a total success. As Lily Loat, then Secretary of The National Anti-Vaccination League of Great Britain, would document, 'Three and half million uninoculated children were as free from diphtheria as five and a half million inoculated children.'

British health authorities ignored the fact that even though diphtheria deaths in England and Wales had declined by 89 per cent since the 1860s, deaths from other childhood infectious diseases had declined even further: whooping cough by 91 per cent, measles by 94 per cent, and scarlet fever, by 99.7 per cent.[41] And all without vaccinations—the whooping vaccine would not arrive on the scene until 1951, the measles vaccine until 1964, and the scarlet fever vaccine … well, there never was a vaccine for that. And they ignored the impact of the social health reforms, particularly the 1944 Education Act which ensured free school milk and subsidised meals for all children at state schools. At the very least, vitamin A from milk and vitamin C from meals would have provided a boost to children's immune systems.

On the Continent, however, the diphtheria vaccine received no kudos whatsoever, particularly from the Germans. After losing the First World War the nation had been ravaged by reparations imposed by the victors, and had then suffered a further blow when the Great Depression struck in 1929. Not surprisingly, poverty and poor nutrition were rife.[42] And as we know, war and its aftermath—social disruption and overcrowding, poor sanitation and hygiene, poverty and poor nutrition—provide the ideal breeding ground for diphtheria bacteria.

Naturally enough, in the 15 years leading up to the Second World War, epidemics did increase in Germany.[41,43] Health authorities countered the onslaught by inoculating people with the new toxoid wherever an outbreak occurred. And in lock-step with that biochemical war on diphtheria, epidemics increased and the death toll rose. Echoing Dr Hadwen's words about the smallpox epidemics in the previous century, Dr T. Crowley, Medical Superintendent of the Wath Wood Hospital and Medical Officer of Health for Wath-on-Dearne in South Yorkshire would write of the whole affair: 'Where they have done the most immunising they are getting the most diphtheria.'[44]

By 1937, as storm clouds gathered over Europe, deaths from diphtheria began to soar. In 1938, on the eve of hostilities, 150,000 Germans (212 in every 100,000) contracted the disease and about 2,500 of them (3.8 in every 100,000) died.[43]

Meanwhile, in France that year—the year French authorities made the vaccine compulsory for children—41 in every 100,000 people contracted the disease.[37] In the Netherlands, which rarely used the vaccine, the rate was 14 in every 100,000. And in Norway and Sweden, neither of which had deployed the vaccine,[41] the incidence of the disease that year was, respectively, 6.3 per 100,000 and 1.6 per 100,000.[37] In other words, the incidence in unvaccinated Norway was 3,300 per cent lower than in vaccinated Germany, and 13,200 per cent lower for unvaccinated Sweden. Indeed, Sweden suffered no deaths at all that year.[45]

Then war broke out. Soon afterwards, the German Reich began to enforce its decree that all children must be inoculated—for adults it was voluntary.[41] And in early 1941, German occupying forces

began to enforce the same compulsory ordinance on the children of France, Belgium, Denmark, the Netherlands and Norway.

Did the vaccine protect people? Not one jot. Within a year, the number of diphtheria cases in Germany was 3,000 per cent higher than it had been in the pre-war (1928–1938) years; in France it was nearly 160 per cent higher.[37,46] And as the contagion swept into Belgium and Denmark, and a swathe of other northern European nations, significant increases were being recorded there too.

But the worst affected were the Netherlands and Norway. Though the incidence rates in both nations were slightly less than in Germany at that time, when compared with the levels these countries had had in 1939, they had nevertheless risen: in the Netherlands by 1,500 per cent, and in Norway by 11,700 per cent.[37]

When the epidemic reached its peak in 1943, Germany was still encumbered with roughly the same number of diphtheria cases as it had the previous year: approximately 240,000.[37] France had had a further 150 per cent increase. Denmark and Belgium, and the neutral countries of Sweden and Switzerland had recorded slightly smaller rises. But the populations of the Netherlands and Norway were reeling. Both had had nearly a 300 per cent increase on the previous year's incidence. The sheer scale of the epidemic is readily apparent when we compare their 1943 rates with those in 1939: for the Netherlands 4,400 per cent higher, and for Norway a staggering 32,000 per cent increase.[37]

Soon after the war, the newly-created United Nations estimated that, in 1943 alone, one million people throughout northern Europe, excluding the USSR, had contracted the disease, and 50,000 of them had died.[47]

The epidemic continued unabated through 1944. But by 1945 it was beginning to subside. Why? Were Allied forces inoculating populations as they advanced on the foe, liberating people from the scourge of diphtheria as well as from Nazism? Not at all. US military authorities evidently had little faith in the vaccine for they'd refused to inoculate their own healthy young troops with the toxoid after discovering that it caused moderate to serious reactions in 'an appreciable number' of adolescents and young adults.[48] No, the reason for the epidemic subsiding was that those who had survived the epidemics had acquired lifelong immunity to the disease.

By war's end, possibly as many as two million Europeans had contracted the disease, and as many as 100,000 had died. Aside from war casualties it had been the leading cause of death and disease.[47]

Diphtheria, however, is only one of the consequences of war. Many other infectious diseases also sweep through worn-torn populations. Little wonder that in 12 of the war-torn countries the number of cases of cerebrospinal meningitis, poliomyelitis, typhoid, dysentery, and scarlet fever had doubled.[47]

Pro-vaccination campaigners still argue that the epidemic remained unchecked because of inadequate vaccination coverage. But the evidence belies these claims. Certainly the compulsory ordinance to inoculate children was obeyed in spite of the bombing. In Berlin in 1942, for instance, 70 per cent of the children aged 3 to 5 had been inoculated, as had 85 per cent of the children aged 6 to 13.[41] And in 1943, about half the children of Berlin had received two injections, a much higher number than in London that year.

Even in America, where vaccinations are mandatory for school children, there had been a resurgence of diphtheria in several cities from the late 1960s until 1975. Medical authorities had of course blamed the outbreak on poor vaccination rates. But in Chicago, for instance, of the 16 children who had contracted the disease, 4 had been 'fully immunised', 5 others had received one or more doses of the

toxoid, and 2 of them had shown evidence of 'full immunity'.[49] And where did the victims live? In poor, overcrowded communities.

Failures of the vaccine have in recent years also occurred in Sweden, Germany, Portugal, China and Thailand, even though some of these nations have maintained high childhood vaccination rates for at least 30 years.[50] And where did they occur? In poor, disadvantaged groups living in crowded conditions.[37]

As a British adviser to the World Health Organisation had acknowledged in 1975, 'The degree of protection achieved with toxoid immunisation is often less than satisfactory.'[51] The document revealed that the US Communicable Disease Centre [the Centers for Disease Control] regularly admitted that about 7–10 per cent of reported cases of diphtheria in the US occurred in individuals whose medical records indicated that they had been 'completely immunised'. And as recently as 1999, a US researcher identified that both the diphtheria and the whooping cough toxoids produced only about 70 per cent 'immunity' in an individual, and as little as 50 per cent 'immunity' in the community at large.[52]

Clearly the diphtheria vaccine was having an abysmal track record, even by the medical profession's criterion that 'immunity' means having a certain level of antibodies to a particular germ. Public health officials were undoubtedly dismayed for they'd been preaching that the diphtheria toxoid, along with the vaccines for measles and mumps and whooping cough, and German measles, was a godsend. Something clearly had to be done to head off public concern.

And indeed it was. Public health officials began to preach that a community would only be protected against an infectious disease if a sizeable percentage of them had been vaccinated. 'Herd protection' (the medical profession prefer to call it 'herd immunity') entered the vernacular. Unbeknownst to most people, however, the term had first been used by cattle ranchers in the US to describe the way a herd would naturally fight off an infectious disease and thenceforth be immune to the disease. A young American researcher, Arthur Hedrich, had coined the term to describe the way children in a community would catch measles, fight it off, and then acquire lifelong immunity to the disease.[53] But public health officials had bastardised the term to mean protection by vaccination.

Lack of 'herd protection' would soon be blamed on the epidemic that erupted in Russia and the Newly Independent States soon after the collapse of the Soviet empire. Stretching from Latvia to the Ukraine in the west, and from Georgia to Kazakhstan in the south, and with Russia at the hub, the region, in 1990, was an epidemic waiting to happen. Millions of people were struggling with a partial breakdown of public infrastructure, as well as sporadic civil wars and social upheavals arising from mass movements of people. Public sanitation and personal hygiene were less than ideal, overcrowding and poor nutrition were commonplace, and people were highly stressed. In a nutshell, their health was suffering.

Sure enough, diphtheria did erupt, first in Moscow and St Petersburg, and then it rapidly spread into neighbouring lands. From 1990 until the epidemic subsided in 1997, 157,000 people had contracted the disease and over 5,000 had died.[54]

But unlike diphtheria epidemics in the pre-vaccination era, when about 70 per cent of cases were in children under the age of 15, this one primarily affected adults. Indeed, the figures had reversed: 70 per cent of cases occurred in people over the age of 15.[37] In fact, 76 per cent of the deaths occurred in adults, not children. Another new trend was also emerging: a large proportion of patients, both young and older, experienced complications from the disease, and those most affected were aged 40 to 49.

And who were the people least affected? The people over 50. Only 2.8 in every 100,000 was affected.[37] And the reason? Most had acquired lifelong immunity to the disease when they were children:

when airborne diphtheria germs had wafted onto their skin and into their nostrils; and their bodies, perhaps without symptoms, had generated a natural defence against the very source of the toxins, the germ; their bodies would 'remember' that germ for the rest of their lives, and rapidly slaughter it should it waft in again.

Of course, the World Health Organisation and the US Centers for Disease Control readily identified the culprit: lack of 'herd protection'.[37,54] And they clung to that story despite the fact that from 1958 until the early 1980s there had been universal childhood 'immunisation' throughout the Soviet Union.[54] Although childhood vaccination rates had declined thereafter, nonetheless they were sufficient at the time of the outbreak to 'protect' about 60 per cent of the young herd'. This is despite the fact that 60 per cent herd protection against diphtheria is considered by western scientists to be sufficient to well nigh protect the whole 'herd';[55] despite the fact that the same percentage of 'herd protection' occurred in many European nations where diphtheria was virtually non-existent;[50,56] and despite the fact that the highest rate of incidence was amongst adolescents aged 15 to 17, a group who were certain to have received their full quota of vaccines, not to mention a veritable cocktail of the aluminium and mercury compounds, and formaldehyde, that accompany them.

No doubt the great purveyors of vaccines had been troubled by events. So they upped the ante and declared that 'herd protection' could only be guaranteed if 95 per cent of the 'herd' had been vaccinated. And that applies for all diseases for which a vaccine exists. For some nations, this means compelling all children to be vaccinated.

Undoubtedly the greatest absurdity of the propaganda on 'herd protection' is this: if 70 per cent of the victims were adults, not children, then how would vaccinating children protect their older siblings, let alone their mothers and fathers? After all, they'd already been vaccinated, and they were already 'protected'. Or were they?

Such outbreaks would suggest that vaccinations induce tolerance, rather than genuine immunity, to a disease. When an epidemic does occur, those people who have not been constantly topped up with boosters, are the most likely to succumb. And who do you suspect are the silent carriers of the disease? Here's a hint. In the epidemic that swept through Russia and the Newly Independent States, it was mainly mothers, not fathers, who came down with diphtheria.[57]

Many of them caught it from their vaccinated children, just as much of the adult population of Europe during the Second World War had caught it from their vaccinated children.

And as for the healthy, young, unvaccinated American GIs who had gone off to Europe to help fight the Nazi foe, only 2,500 out of as many as 3 million of them contracted the disease, and of those only 71 died.[48] Why was the rate, at 5.4 per 100,000 troops, so low? They were healthy, and as many as 80 per cent of those who had come from urban areas in the US had acquired lifelong immunity to the disease.[58]

The emperor's new clothes and the medical courtiers

Perhaps it began with the delusion that a vaccine had eradicated diphtheria. Perhaps it was the post-war

hubris that technology could defeat all foes. Perhaps it was simply a noble desire to protect children. Whatever the reason, soon after World War II the germ of an idea began spreading like a contagion through western governments and drug companies: why not create vaccines to protect all children from all infectious diseases? Soon their 'experts', the medical courtiers, would jump on the bandwagon and begin singing the praises of the emperor's new clothes.

Tetanus

As we've seen, the diphtheria vaccine had been the first off the rank—and the courtiers proclaimed that that had been a total success—followed soon thereafter by the tetanus vaccine. Tetanus is not, of course, a contagious disease, but it can arise from penetrating wounds. Though the bacteria are present in human and animals faeces, it is only when the spores penetrate oxygen-deprived wounded tissues and begin to exude a strychnine-like toxin that they wreak havoc on our nervous systems: 'lockjaw' sums up the horror of it.

Wounded soldiers have always been prime candidates for contracting tetanus, particularly if their wounds are contaminated by debris from well manured soils, such as those of the Western Front in World War I.[59] For instance, for every 1,000 soldiers wounded on the fields of Spain and Portugal during the Peninsula War of 1808 to 1814, there had been 12.5 cases of tetanus; in the Crimean War, 2 cases.[60] The same rate of incidence had occurred during the American Civil War, but in that war tetanus had been particularly deadly: 89 per cent of those soldiers who contracted it had died from the disease.[61] Indeed, during that war, germs had claimed many a soldier's life: two-thirds of the 600,000 men who died during the war were killed by epidemics of infectious diseases that swept through the Union and Confederate armies.[62]

As the practice of resolving conflicts through war continued through the remainder of the 19th century, the prevalence of tetanus amongst wounded soldiers continued much as it had always been: amongst every 1,000 troops wounded on the well manured fields of Europe during the Franco-Prussian War in the early 1870s, there had been 3.5 cases of tetanus, and 90 per cent of those had died from it.[63] And yet on the barren African soils during the Boer War at the dawning of the new century, there had been only 0.28 cases in every 1,000 wounded soldiers.

During the 1890s, a tetanus antitoxin horse serum had been developed for passive 'immunisation' in the treatment of wounds. This vaccine had been widely used during the First World War, and was later touted as a godsend to wounded soldiers. Deaths from tetanus among wounded soldiers on the Allied side certainly did decline, from about 8 cases per 1,000 in 1914, to 1.4 per 1,000 during the following four years.[64] Hence, of the 520,000 wounded American soldiers, only 70 developed tetanus.[65] But amongst the wounded British soldiers, 2,595 had developed tetanus, 95 per cent of cases occurring on the Western Front.[63] And yet despite the anti-tetanus serum, 54 per cent of them had died of tetanus.

But what medical courtiers fail to tell us is that, as in every war, great strides had been made in surgical techniques and the treatment of wounds. During the Great War the occurrence of Clostridial infections (tetanus from *Clostridium tetani*, and gas gangrene from *Clostridium perfringens*) had been linked to the inadequate debridement of wounds (cleaning away damaged tissues and debris), and to early wound closure.[59] At the Inter-Allied Surgical Conference in Paris in 1917, delegates had established a new policy for the management of wounds: namely, debridement and delaying the suturing of large wounds. In other words, the best treatment for preventing tetanus is to thoroughly cleanse any wound and expose it to the air for at least four days, if not longer—which makes total sense since oxygen-hating Clostridial bacteria die when exposed to oxygen.

Moreover, from 1915, improved methods of transportation had facilitated the evacuation of wounded soldiers from the battlefields to hospitals so that their wounds could be properly treated.[63]

Hence, not only did tetanus decline, but so did gas gangrene: the latter from about 100 to 120 cases in every 1,000 wounded soldiers in 1914–1915, to about 10 cases per 1,000 wounded soldiers in 1918[63,66] —that's a drop of more than 90 per cent, and without a vaccine being used.

In addition, surgeons had begun using Dakin's hypochlorite solution for flushing wounds.[59] Hydrogen peroxide had also been extensively used from the early years of the 20th century till the 1950s for the same purpose. Such oxidising agents alone would have killed the oxygen-hating tetanus spores. Applying honey to wounds, a traditional folkloric practice in many parts of the world, particularly in Russia and China—and employed by Russian troops during the Great War—has the same effect. When diluted with bodily fluids in a wound, honey releases hydrogen peroxide.[67]

Nonetheless, the medical courtiers claimed that tetanus had been conquered by the tetanus antitoxin serum—they had no proof, just an empty claim. When World War II broke out, the lessons gleaned from the Great War about wound treatment and rapid evacuation of wounded soldiers to hospital facilities were readily put into practice. Doctors realised that the sooner a wound was debrided—and certainly within six hours—the better the chances of avoiding gas gangrene and tetanus. And of course, every soldier was inoculated with a newly-developed vaccine to produce 'active immunisation' in order to prevent tetanus developing in the first place.

Again, the great medical courtiers claimed that the reason why only 12 American soldiers out of 2,734,819 hospital admissions had contracted tetanus was because of the vaccine.[68] Five of those died;[69] which surely begs the question: if the vaccine was so successful, why did any soldier develop tetanus, let alone die from it? Another question also begs answering: why in the British Army was the death rate amongst vaccinated men not significantly lower than in unvaccinated men?[60]

As for gas gangrene, even the new wonder-drugs, pencillin and the sulphonamides, had failed to lower the incidence of deaths from it: the death rate was no different to the rate in the First World War, before the advent of antibiotics.[63] As British medical historian Dr Geoffrey Noon had noted, 'A quotation from a standard surgical textbook published after the Second World War, is apposite: "Gas gangrene, more than any other complication of wound infection, serves to point the moral that no ancillary methods will make amends for neglect of meticulous primary surgery." '[70] The same could be said for tetanus prevention.

Meanwhile, in the unvaccinated civilian population of Britain, the death rate from tetanus declined amongst males by 66 per cent between 1938 and 1957.[71] And in America, the death rate amongst unvaccinated young children dropped by 85 per cent between 1900 and 1940, and a further 8 per cent by 1950.[72] Which just goes to prove that cleanliness, not a hypodermic needle filled with a biochemical cocktail, is next to godliness.

Nevertheless, despite the overwhelming evidence that has been accruing since the 1940s in every vaccinated nation—at least 26 research papers have been published in peer-reviewed journals attesting to the fact that people who are fully vaccinated against tetanus can get still get the disease,[73–99] even when their antibody levels are 16 times the amount considered to be 'protective'[96]—the courtiers continue with their mantra that the emperor's new clothes are resplendent. And that a booster will make them more so.

As for mothers passing on to their children their own 'immunity', the World Health Organisation admits that, 'The number of reports from Asia and Africa describing the failure of tetanus toxoid to prevent neonatal tetanus in infants of immunised women has increased recently.'[100]

Now to put all this into perspective, from 40 to 50 (0.15 per million) Americans get tetanus every year, and 7 to 10 die from it.[101] And about 260 Americans are struck by lightning each year, and roughly 93 of them die from that.[102] Which means that you have roughly a six-times greater chance of being struck by lightning than of contracting tetanus, and about a 10-times greater chance of dying from being hit by lightning than from contracting tetanus.

To put this into the context of modern diseases, autism—which has been linked to the mercury compound, thiomersal (thimerosal in the US), in vaccines[103–110]—affects 1 in 150 children.[111] Which means that children have nearly a 45,000-times greater chance of becoming autistic than of contracting tetanus.

And given that Alzheimer's disease—which has been overwhelmingly linked to aluminium,[112–149] a toxic metal that occurs in many vaccines, including the tetanus toxoid—affects 1 in 8 Americans over the age of 65,[150] we can conclude that elderly Americans have at least a 830,000-times greater chance of getting Alzheimer's disease than tetanus.

Whooping cough

From the 1950s onwards, the tetanus toxoid for children would be accompanied by the biochemical cocktail of the vaccines against diphtheria and whooping cough: the DPT cocktail. And by the late 1960s, a vaccine to conquer measles mumps and rubella, all combined for convenience into one biochemical cocktail, the MMR vaccine, would be added to the list.

Admittedly, small outbreaks of whooping cough during the 1950s and '60s did blemish the emperor's finery, but the loyal courtiers blamed these on parents who had either failed to keep their children's inoculations up-to-date, or had refused point blank to have their children 'immunised'.

Then in the early 1970s something happened, something that government courtiers had not expected. And it wasn't the return of whooping cough epidemics, though that certainly precipitated the affair. It was the occasion of a Scotsman by the name of Gordon Stewart daring to break ranks and declare that the emperor has no clothes.

Stewart, then Emeritus Professor of Public Health at the University of Glasgow, had undertaken some epidemiological studies which revealed that during the 1975 whooping cough epidemic in Glasgow, nearly a third of the notified cases were fully vaccinated.[151] And where did he find the greatest prevalence of the disease? In the poorer districts of the city. 'The decline in recent years,' he reasoned, 'could be attributable to improvement in these conditions at least as much as to immunisation. [Hence] There is no epidemiological justification for continuing mass vaccination … '.

Soon afterward, another Scotsman, Dr Robert Ditchburn from Shetland, would also break ranks with the medical courtiers. Ditchburn had good reason to observe the emperor up close. He had been a rural family doctor for years and had vaccinated 94 per cent of the children in the district. Only in 1974 did he stop using the whooping cough vaccine. Thus, when an epidemic swept through the island at the end of 1977, he could witness and document the effects of the disease on vaccinated and unvaccinated children.

And what an abysmal failure the vaccine was: 45 per cent of 144 children under the age of 16 developed whooping cough; 86 per cent of 3.5 to 5-year-olds who contracted the disease were fully vaccinated, as were 42 per cent of cases among the 6- to 10-year-olds and 50 per cent of cases among the 11- to 15 year-olds.[151,152] And as you would expect, of the 35 children born after he'd ceased giving the

whooping cough vaccine, 46 per cent were naturally infected.

Overall, 54 per cent of all 'fully immunised' children had contracted the disease, as had 56 per cent of all unvaccinated children.[153] 'Heads I win, tails you lose,' sums up the charade. The vaccine was clearly a failure; the emperor was indeed naked. In his epitaph to the vaccine Ditchburn concluded: 'My findings … do not support the routine use of pertussis immunisation in rural Shetland today.'

When the 1977–1979 epidemic finally subsided in Britain, 35 per cent of reported cases had occurred in fully vaccinated children aged between 1 and 5. But of the under-5s who had not been vaccinated, only 5 per cent had been recorded as having contracted the disease.[154] As Stewart noted, children who lived in deprived communities and those who had been vaccinated were the worst affected, many having to be hospitalised. Recall that Dr Hadwen had made the same observation about smallpox cases a century before.[17]

By 1983, 50 per cent of British parents were refusing to have their children inoculated with the vaccine. They, too, could see that the emperor was naked. Many had been swayed by the epitaph Professor Stewart wrote for the vaccine: 'In the United Kingdom and in many other countries, whooping cough (and measles) are no longer important as causes of death or severe illness except in a small minority of infants who are usually otherwise disadvantaged. In these circumstances, I cannot see how it is justifiable to promote mass vaccination of children everywhere against diseases which are generally mild, which confer lasting immunity, and which most children escape or overcome easily without being vaccinated.'[154]

But most of all, what clinched the decision for parents was Professor Stewart's revelation that in some children the vaccine had caused screaming attacks and convulsions, for others it had caused irreversible brain damage, and a few it had killed.[155,156] And his warning that for non-deprived children 'the risk of pertussis vaccine during the 1970–83 period exceeded those of whooping cough.'[157]

Britain's medical courtiers of course blamed the epidemic, which affected 200,000 people, on poor vaccination rates. But Britain was not the only nation to experience the failure of the vaccine. All nations, including those like the United States and the Netherlands with childhood vaccination rates as high as 96 per cent, experienced waves of epidemics beginning in the 1970s and their recurrence, on average, every four years to this very day.[158–176]

In the United States, for instance, between 1980 and 1986, 17,400 people contracted whooping cough, and 25 per cent of them were children aged from 1 to 4.[161] And United States health authorities prided themselves on the fact that 94 per cent of children were fully vaccinated by the time they started school.

Similarly in Australia, between 2003 and 2005, there were 25,000 reported cases of whooping cough, 5,200 of which were in children under the age of 15.[162] And Australian medical authorities proclaim that 86 per cent of children are fully vaccinated. A closer inspection of the Australian statistics reveals that 70 per cent of the 1- to 4-year-olds who contracted the disease were fully vaccinated, that 12 per cent were partially vaccinated, and that 61 per cent of the 4- to 9-year-old children were also fully vaccinated and 15 per cent were partially vaccinated. In other words, two-thirds of the children under the age of 9 who contracted the disease were either fully or partially vaccinated.

And there's plenty more evidence to prove the bleeding obvious: that the emperor is truly stark naked. In the 1993-94 epidemics that struck the US cities of Chicago and Cincinnati, for instance, 65 per cent of cases occurred in children under five-years-of-age.[161,164,165] According to the US Centers for Disease Control, 84 per cent of these children were fully or partially vaccinated. And in Cincinnati, nearly

a third of children with whooping cough were hospitalised.

The hollow promise of using needle-dispensed white man's magic to eradicate whooping cough through 'herd protection', let alone halt outbreaks, in developing countries would also become evident. For example, in Cape Town, South Africa, a city where 95 per cent of children were fully vaccinated when a whooping cough epidemic struck in 1988–89, 33 per cent of pre-primary school children were affected.[166] And, as had happened in Cincinnati and Britain, a high number of vaccinated children had to be admitted to hospital.

In the West African nation of Gambia, there was an outbreak of measles and whooping cough during the early 1990s, even though childhood vaccination rates for both diseases were high.[167]

Swedish authorities were so disenchanted with their vaccine—after learning that 84 per cent of a sample of 620 children who had contracted the disease during epidemics in the mid-1970s were fully vaccinated—that they abandoned its use in 1979.[168]

Curiously, before the vaccination era, whooping cough—as with diphtheria, and measles and mumps, and all the other diseases for which a vaccine has been created—was remarkably rare in adults and rarely did it occur twice in the same person.[175,177] But now it is commonplace. Why would this be? Because not only does the whooping cough vaccine not confer lifelong immunity to the disease, but also, as Russian researchers had discovered during the diphtheria outbreaks in the early 1990s, because vaccinated children are the silent carriers of the disease.

This was exactly what Israeli researchers suspected when they investigated an outbreak of whooping cough in two day-care centres in 2000 and found that not only did 55 per cent of the 5- to 6-year old children, all of whom had received four shots of the DPT vaccine, have whooping cough, so did many of their siblings and parents.[176] The same had been apparent during a whooping cough outbreak in an isolated community in the Gascoyne region of Western Australia in late 1999, even though 96 per cent of children were fully vaccinated.[163]

Measles

Not surprisingly, the vaccines to conquer measles, mumps and rubella (German measles), combined for convenience into a single cocktail (MMR), have fared little better. Up until the 1960s, when the measles vaccine arrived on the scene, most children had contracted measles, mumps and rubella, and thereafter had lifelong immunity to these diseases.[178–183] Many parents had deliberately exposed their children to these endemic diseases—at 'measles parties', for example— knowing that contracting these diseases later in life could cause medical miseries; for example, meningitis, mastitis and inflammation of the testes from mumps. And when girls grew up and became mothers, they would pass on to their babies antibodies to the diseases to protect them during the first crucial year of life.

Moreover, most doctors knew how to treat these diseases. In a case of measles, for instance, the infected child was to be kept in a darkened room, out of draughts, and kept warm to ensure the measles rash became pronounced. They knew that should the rash disappear, the infection could turn inwards and affect the ears, eyes, lungs, heart, or brain. If that did happen, one technique employed was to make the child wear a vest that had been soaked in salty water. The skin would become irritated and the rash would soon reappear. So long as no mischief was done—for instance, by quelling the child's fever with such antipyretic drugs as aspirin or paracetamol—then measles would run a benign course.[184] Of course, as with other diseases, impoverishment and malnourishment, by weakening a child's ability to fight off these diseases, could prove fatal.

By the 1950s, well before the programme to inject the measles vaccine into children's arms began, deaths from measles had plummeted; in Britain, for instance, by 99.4 per cent compared with the death rates in 1900,[185] and in the US by 99.7 per cent.[186,187] Nevertheless, governments and drug companies remained hell-bent on eradicating these diseases. Not once did they stop to consider that measles alone might serve some function in human health, that it might kick-start a child's immune system, that it might be a blessing or, as the physicians of India once believed, a visitation of the goddess.

Certainly in the 10 years following the advent of the measles vaccine in 1964, the prevalence of measles did decline in the United States, from about 400,000 cases each year during the 1950s to between 22,000 and 75,000 during the late 1960s and early 1970s.[187] Following each outbreak, scouts from state public health departments and the emperor's palace in Atlanta, Georgia, later to be known as the US Centers for Disease Control and Prevention, would be despatched to ascertain the reason for the vaccine's failure to protect.

In Jacksonville, Florida, for example, 28 kindergarten children had come down with the disease, even though 25 of them were vaccinated.[188] To make matters worse, there was little difference in the severity of the disease between the vaccinated and unvaccinated children.

Outbreaks continued during the 1970s, belying claims that measles was a 'vaccine-preventable disease'.[189–198] Even in schools where over 99 per cent of the children had been vaccinated, outbreaks still occurred; for example, in two schools in Erie County, New York in the spring of 1978.[198] Equally worrisome for the palace courtiers was the fact that from 84 to 94 per cent of the vaccinated children who contracted measles had sufficient antibodies to the disease to make them 'immune'. At least that was the courtiers' assumption. Moreover, there were negligible declines in the death rate from measles;[199] for example, 130 children had to be hospitalised and 6 of them died during the epidemic that swept through St Louis, Missouri, in 1971 to 1972, despite vaccination rates in school children being as high as 89 per cent.[194] Nevertheless, courtiers at the palace kept up the facade by using dubious statistical projections,[200] to spin the tale that 60 million doses of the vaccine in its first 10 years had saved 2,400 children's lives.[201]

Even though the United States had set the goal of eradicating measles by October 1982—indeed the palace courtiers could gloat that measles cases had reached an all time low of 1,497 cases in 1983[202]—outbreaks amongst vaccinated children continued to rise during the 1980s: for instance, 46 cases in Warren County, Pennsylvania in 1981/82, within two weeks of all children being vaccinated;[203] 16 cases among high school students in Illinois in 1983/84, all of whom were vaccinated.[204] There were also 1,806 cases among high school students in Corpus Christi, Texas, 99 per cent of whom were vaccinated, and of whom more than 95 per cent had antibodies to the measles virus;[205] 118 cases on a Blackfeet reservation in Montana, 82 per cent of whom were vaccinated; and 23 cases among school children in Browning, Montana, 98.7 per cent of whom were vaccinated.[206]

In fact, between 1985 and 1986 there had been over 253 measles outbreaks amongst the most vaccinated people on earth: 26 per cent of cases had occurred in pre-school age children, 14 per cent of whom were vaccinated; and 67 per cent of cases had occurred in 5- to 19-year-old school children, 60 per cent of whom were vaccinated.[207] During 1987 and 1988, measles outbreaks continued to occur amongst school age children, 98 per cent of whom were vaccinated,[208–210] averaging about 3,000 cases each year.[211]

But 1989 was the year measles epidemics erupted like a rash across the United States.[212] Within the first 26 weeks there had been 7,335 reported cases,[213] and by year's end the tally was 18,193 in 815 outbreaks.[211,214] Forty states were affected, and in particular the cities of Houston, Los Angeles and

Chicago. According to the US Centers for Disease Control and Prevention, more than half of the cases occurred among appropriately vaccinated children aged 5 to 19; 30 per cent of cases occurred in children under 5; and the remainder of cases occurred in older adults who may or may not have been vaccinated.[213]

One instance of the vaccine's abysmal failure is that at a high school in Illinois where 68 measles cases occurred, yet 99.7 per cent of students at the school were vaccinated.[215]

All told, between 1989 and 1991 there had been 55,467 reported cases of measles, 11,251 hospital admissions, and 166 suspected measles-related deaths.[216] The total number of measles cases may well have been much higher, however, according to researchers who investigated the outbreaks in Wisconsin and Texas, because unlike unvaccinated children, 97 per cent of whom develop the typical measles rash, only about 15 per cent of vaccinated children who contract measles get the rash;[217,218] this is known as 'atypical measles'. Indeed, this had been identified in those who had been inoculated with the earlier killed-measles-virus vaccine, as well as the later live-attenuated-virus vaccine.[219] In other words, cases of measles are typically under-reported. Hence the vaccine's efficacy is overestimated.

Proof of under-reporting came in 1991, when researchers investigating a measles outbreak in New York City discovered that only 45 per cent of the 1,487 cases admitted to New York hospitals had been reported to public health authorities.[220]

The palace courtiers did at least acknowledge that measles was far harder to stamp out than they had previously thought.[213] Two researchers from the Mayo Clinic in Minnesota at least had the nous to conclude: 'The apparent paradox is that as measles immunisation rates rise to high levels in a population, measles becomes a disease of immunised persons. Because of the failure rate of the vaccine and the unique transmissibility of the measles virus, the currently available measles vaccine, used in a single-dose strategy, is unlikely to completely eliminate measles.'[221]

Realising that parents were unlikely to remain convinced about the splendour of the emperor's new clothes, the courtiers took a new tack: implement laws requiring all children to be inoculated twice with the failed MMR vaccine;[222] encourage health-care providers 'to take advantage of every opportunity' to vaccinate susceptible adolescents and adults; 'encourage persons in religious groups who do not seek health care to accept vaccination'; during an epidemic to make sure everyone at risk is inoculated; and, of course, continue to panic the population by stressing the 'seriousness of measles illness'.[223]

Since 1991 the number of reported cases of measles in the United States has certainly declined radically. When outbreaks do occur, they are often blamed on alien imports, on people who have come from or travelled through countries that have not caught America's obsession with defeating viruses through mandatory inoculations.

An outbreak in Indiana in 1991, for instance, was blamed on three measles-virus-carrying, albeit previously vaccinated New Zealand athletes.[223] State public health officials had gone to town to prevent an outbreak: the three athletes were quarantined for four days, 1,300 possible contacts were inoculated, and surveillance teams had been set up to record twice-daily reports from international athletic delegations. Come hell or high water, the efficacy of the double-dose MMR vaccine would not be put to the test. Nor would any imports tarnish America's reputation of having achieved 'herd protection'.

But it had already failed the test. Four healthcare workers at the Children's Hospital of Philadelphia had contracted measles from infected children in 1991.[224] Three of them had done the right thing and had had at least two doses of the measles vaccine, and all of them had levels of antibodies that courtiers would consider to indicate that they were 'immune' to the disease.

And the vaccine continues to fail the test to this very day. Reports from the Centers for Disease Control continue to identify cases of measles in people who have had two or even three doses of the MMR vaccine.[225–231]

The lengths to which courtiers will go when a viral foe threatens to expose the emperor's flanks is evident in their response to a measles outbreak in seven schools in Anchorage, Alaska in September 1998.[232] This had also been blamed on a foreign importation; in this instance, on a four-year-old visitor from Japan. Thirty-three people, mainly students, contracted the disease, reputedly from the child, even though 29 of them had had at least one inoculation against the disease, and one certainly had had two inoculations.

In response to the outbreak, Alaska's Department of Health and Social Services, issued an emergency decree that by mid-November 1998, all Anchorage school children must have received two doses of the vaccine. The order was subsequently expanded to require all students in Alaska to have two doses of the measles vaccine by early January 1999. The irony is that for the nation that purports to be 'the land of the free and the home of the brave', the decree smacked of the order issued by the German Reich 58 years earlier to inoculate its children against diphtheria.

Nonetheless, in March 2000, the United States proudly proclaimed that measles had been eradicated from the nation.[233] Thereafter, every case of measles, every outbreak, would be blamed on foreign importations.

But in every nation, whether richer or poorer, the measles vaccine from the outset has proved to be an abysmal failure.[234–277] Regardless of the percentage of the population that has been vaccinated—even when it is as high as 98, 99, or 100 per cent of the population,[237,240,248] and regardless of whether one or two shots of the vaccine are administered—outbreaks still continue to this very day. As two Canadian researchers concluded, 'Neither prior vaccination nor detectable secondary immune response [having specific antibodies to the measles virus] ensures protective immunity.'[239]

A classic case, not only of the failure of the vaccine to protect, but also of its dangers, occurred in Gambia.[250,251] In 1967, following a mass vaccination campaign in which 97 per cent of the population had been forced to be inoculated, the World Health Organisation proclaimed that measles had been eradicated from the nation. But in 1972 an epidemic erupted. And medical complications from the disease, and deaths, were significantly higher than they had ever been before.

As of 2004, the medical courtiers of over 50 per cent of the world's countries have managed to achieve childhood vaccination rates of more than 90 per cent.[278] But in 20 per cent of the world's nations, including Britain, France, Italy, Norway, China, New Zealand and South Africa, the courtiers have been battling to convince all parents that the emperor is clothed: vaccination rates are between 80 and 90 per cent. And doctors in the poorer nations aren't too convinced about white-man's magic either. Thus the idea of eradicating measles from the planet is pie in the sky.

Mumps

Perhaps the greatest joke the courtiers have spun is that mumps is a 'vaccine-preventable disease'. Introduced in the late 1960s and routinely used as the second 'M' of the MMR vaccine from the late 1970s, the vaccine started off with an apparently impressive record: in the first 18 years of its use the incidence of mumps in the United States declined by 98 per cent.

Foreshadowing subsequent epidemics, however, researchers had warned the courtiers that anti-

mumps antibody levels in inoculated individuals were far lower than in those who had acquired natural immunity to the disease.[279]

Then in 1985 the lull broke. By the following year there were 7,790 reported cases in the United States. And in 1987, 12,848 cases had been recorded in 44 states.[280] The incidence had risen nearly five-fold in all age groups, and in the 15- to 19-year-olds there was over an eight-fold increase.

Outbreaks continued through the 1980s in highly vaccinated populations[281]—in Douglas County, Kansas, for instance, where 97.6 of the cases were fully vaccinated[282]—and on into the 1990s amongst school populations where 98 per cent of cases were fully vaccinated.[283–286]

The vaccine's appalling track record continued into the 21st century.[287–292]

Even though 97 per cent of American elementary school children, and 98 per cent of middle school children, had been twice vaccinated against mumps, as had 84 per cent of 18- to 24-year-olds, an outbreak occurred in 2006, affecting 6,584 people in 11 states. Those most affected were the 18- to 24-year-olds, followed by the 5- to 17-year-olds. In Iowa alone, where the epidemic began, 94 per cent of the population of children and adolescents were vaccinated. The idea of 'herd immunity' had clearly been trashed.

Parents in Canada, Europe and Asia would have been as equally dismayed as their American counterparts at the vaccine's failure.[293–324] From the 1990s up until today, there have been mumps epidemics in highly vaccinated populations of people who never had a chance to acquire lifelong natural immunity to the disease. Affecting all age groups, but particularly adolescents and young adults, epidemics swept through Switzerland in 1993 (when the nation recorded its highest levels since records began in 1984,[295] and despite people having high levels of antibodies),[296] through Portugal in 1996–1997 (with 30,000 cases),[302] through Spain in 1997–1998 (with 6,915 cases, 93 per cent of whom were fully vaccinated),[300] through England and Wales in 2004–2005 (with 56,390 cases),[313] and through Poland in 2006 (with 15,115 cases).[322]

Two nations, the Czech Republic and Moldova, certainly had the opportunity to test the theory of 'herd immunity', since compulsory vaccinations had ensured that at least 95 per cent of their young populations had been vaccinated. But the Czech Republic was struck by a series of mumps epidemics, first in 1995–1996, affecting 11,680 people, then in 2003–2004, and again in 2005–2006.[318] And in Moldova in 2007–2008, there were 14,438 cases, the vast majority of whom had been inoculated with two doses of the vaccine.[319]

Rubella

As for rubella, it is a mild infectious disease causing a low-grade fever and typically a two-day rash. Before the vaccination era most children contracted the disease and hence acquired long-term immunity. Exposure to the freely circulating virus during epidemics would simply top up their immunity. In fact, the disease is so mild that 20 to 50 per cent of children get no symptoms at all, and thus the presence of a rubella infection can only be identified through blood tests.[325,326]

The only reason the rubella vaccine was introduced was to prevent women getting the virus while pregnant. In 1941, an Australian ophthalmologist, Dr Norman Gregg, had made the link between women having rubella while pregnant and subsequently giving birth to babies with a congenital syndrome of cataracts, deafness, and heart disorders. Mind you, catching many other diseases during pregnancy, particularly HIV, toxoplasmosis and cytomegalovirus infections, but also measles, mumps, chickenpox

and even influenza, can cause congenital abnormalities in foetuses[327]—which is a good reason for girls to catch and acquire lifelong immunity to typical childhood infectious diseases, and for women to be healthy and have strong immune systems before they conceive.

In 1972, just as the rubella vaccine was being added to the medical armamentarium, the World Health Organisation estimated that as many as 10 to 15 per cent of women who contracted rubella during the first three months of pregnancy would give birth to a deformed child.[328] (As an indication of how the medical courtiers whip up public fear, nowadays they claim that the syndrome is likely to affect 80 per cent of babies if the mother is infected in the first eight weeks of pregnancy.[329]) But instead of finding out why 85 to 90 per cent of pregnant women who contracted rubella in the first 12 weeks of pregnancy did not give birth to deformed babies, rather than recognising that a diet containing high amounts of vitamin A and C could help prevent or reduce the severity of infectious diseases,[330] the thrust of medical prevention became focused on the new vaccine.

In the beginning, two different national approaches were adopted in the war on rubella. The United States, obsessed with 'herd immunity', decided on the potentially dangerous ethical concept in medical care of vaccinating one segment of society (young boys, as well as young girls), to prevent disease in a second segment of society (pregnant women) which in turn would prevent the disease in a third segment (foetuses). Canada, forever under the influence of its powerful neighbour, followed suit. Needless to say, the ethics of this approach, where boys gained no benefit whatsoever from a medical procedure inflicted on them, stirred up considerable controversy.[331–333] The same controversy would later emerge with the advent of the vaccine to tackle the human papilloma virus.[334,335] Other nations were less obsessed with the concept of 'herd immunity' and initially decided on vaccinating adolescent girls only. But during the 1980s and '90s, these nations adopted the American way of mass vacccination of children by adding the rubella vaccine to the measles and mumps vaccines (the MMR vaccine).

From the outset it was clear that the 'immunity' conferred by the attenuated virus in the rubella vaccine paled by comparison with the natural immunity gained through infection with the wild virus. Researchers had found that three to five years after being vaccinated, some children's antibody levels were undetectable, and that cell-mediated immunity was far stronger, and more persistent and stable in those who had acquired immunity from getting the disease.[336-338]

In 1979–80, eight years after Britain had begun vaccinating all 13-year-old school girls, researchers in Glasgow found that 11.5 per cent of females aged 13 to 21 had insufficient antibodies to protect them against rubella.[339] If ever there was proof that the rubella vaccine was a failure was the fact that 10 per cent of males in the same age group were also found to be susceptible to the virus … and they weren't even vaccinated. Ninety per cent of them had acquired natural immunity from being exposed to the freely circulating, wild rubella virus. The fact that there was no difference between vaccinated females and unvaccinated males prompted the researchers to comment: 'The rubella vaccination programme has clearly failed to reduce the number of susceptible women in this practice.'

Waning immunity in vaccinated adolescent females had also been identified by researchers in Canada, the United States, the Netherlands, Denmark, Switzerland and Finland.[340–354] Ten per cent of army recruits tested in a study in Canada, for instance, and 15 per cent of sixth-graders in a US study, had insufficient antibodies to protect them against rubella.[343,347] And according to a recent study in Finland, vaccine-induced 'immunity' dwindles so dramatically that by the age of 15, girls could well become infected with the very disease the vaccine was supposed to protect them against, especially when they're pregnant.[354] Indeed researchers in Switzerland suspected that the high percentage of Swiss women with antibodies, 15 years after they had been vaccinated, could be explained by them being reinfected by the wild virus, with no credit whatsoever being afforded to the rubella vaccine.[353]

So it should come as little surprise that rubella infections do occur in vaccinated individuals. One of the first researchers to discover the abysmal failure rate of the rubella vaccine was Australian virologist, Dr Beverley Allan. In 1972 she had monitored a group of male army recruits who were vaccinated, and who had produced antibodies to rubella, immediately prior to being sent to an army camp that typically had an annual outbreak of rubella. Four months later, when the outbreak did occur, 80 per cent of the vaccinated males contracted the disease.[355]

American researchers had also identified an 80 per cent failure rate of the vaccine during an epidemic in the late 1960s.[356] Studies have shown that during epidemics many vaccinated individuals do become infected with the rubella virus,[357–369] but from 50 to 80 per cent of them produce no clinical symptoms at all. In other words, only through blood tests can the infection be identified. According to Canadian researchers the actual number of rubella cases may well be 50 per cent higher that those reported;[370] hence, the claim that the incidence of rubella dropped dramatically after the advent of the vaccine is simply hype.

Moreover, far more vaccinated individuals become reinfected with the rubella virus, even after a second vaccination, than do people who have acquired natural immunity.[356,364,371] Because of this, and because of the failed immunity amongst vaccinated individuals and the continuing epidemics, the extents of which have clearly been under-reported, several researchers called into question the very concept of 'herd immunity' to the rubella virus.[331–333,372–375] Indeed, the very idea of vaccinating children had been predicated on the assumption that they were the major source of infection for pregnant women. But since the incidence of women giving birth to babies with congenital rubella syndrome is no greater in women who have several children compared with those with only one, this had been shown to be a flawed assumption.[376]

If the idea of vaccinating children was to prevent them infecting pregnant women, then how has it fared? If we are to believe the US Centers for Disease Control and Prevention, it has been a total success. In the year 2000 the emperor's palace in Atlanta, Georgia, announced that the United States was on the verge of eliminating both rubella and congenital rubella syndrome, and in 2004 it proclaimed that the goal had been achieved.[377,378]

Certainly many studies have shown that vaccinated women have developed subclinical rubella;[379–390] and that the foetuses of such women do become infected with the virus.[391–403] So what do many pregnant women do when they realise they've been infected with the virus? They opt for an abortion.

When a rubella epidemic struck the island of Oahu in Hawaii in 1977, for instance, 429 people, mainly women aged 20 to 24, were affected.[397] Twelve of them were pregnant, and 11 opted for an abortion; one, who contracted rubella in the second trimester of her pregnancy, decided to continue with the pregnancy, and she gave birth to a normal healthy baby.

Now it just so happens that neither the United States, nor Canada, nor most other nations monitor the number of pregnancies terminated because of rubella. Canadian authorities do at least acknowledge that the incidence of chronic rubella syndrome is vastly under-reported, and that the less severe cases of chronic rubella syndrome are not even being diagnosed, much less reported.[398–400] Undoubtedly therapeutic abortions do distort national statistics on the incidence of congenital rubella syndrome, and thereby create the illusion that the emperor's new clothes are immaculate.

Britain, Japan, and Israel, however, do record the number of pregnancies terminated because of exposure to, or infection with, the rubella virus. When a rubella epidemic struck Israel in 1972, for

instance, Israel's Ministry of Health recorded as many as one-and-a-half times more abortions than in the previous three years—about 20 per cent of the abortions were due to a history of exposure to rubella in the first months of pregnancy.[401]

Similarly, Britain's Office of Population Censuses and Surveys identified that the number of abortions due to exposure to, or infection with, rubella between 1976 and 1978 was over 13 times higher than the number of babies born with congenital rubella syndrome.[402] In 1978 alone there were 830 abortions because pregnant women had contracted rubella.[403]

And when Japan was struck by a rubella epidemic in 1987—as it had done so every five years since 1976 despite the introduction of the rubella vaccine in 1977—Japanese authorities reported that 100 children were born with congenital rubella syndrome. But 2,500 women had decided to terminate their pregnancies.[405] Which means that the incidence of congenital rubella syndrome could be up to 25 times higher than the medical courtiers report.

In fact, in the late 1970s the Chief Medical Officer at the Department of Health and Social Security in Britain, Henry Yellowlees, contended that the incidence of congenital rubella syndrome (which at that time in Britain was assumed to be about 400 cases each year) was probably twice as high as recorded figures, even excluding abortions.[406]

Certainly the number of abortions due to rubella has dramatically declined in Britain in recent years.[407] But the incidence of congenital rubella syndrome is less easy to ascertain, the reason being that the syndrome can include delayed effects, including cataracts, autism and Type I diabetes.[408] Tellingly, the incidence of both autism and Type I diabetes in young children has skyrocketed in recent years, as have cases of atopic cataracts.[409–413] But because most young children are inoculated with the MMR vaccine and have vaccine-induced antibodies by the age of 15 months, no one can determine whether these, let alone deafness, are the result of congenital rubella syndrome or not.

To compound the mess, by vaccinating children the vaccinators shifted the incidence of rubella to older age groups, thereby creating a potential time bomb amongst the very people the vaccine had been purported to protect.[414] Thus, in the late 1960s, before the advent of the vaccine, only 23 per cent of rubella cases in the United States occurred in people over 15.[415] But by 1975, the figure was 62 per cent;[415] by 1990, 81 per cent;[416] and by 1997, 85 per cent.[417]

Further compounding the debacle was evidence from computer modelling that unless 60 per cent of the population was vaccinated with the MMR vaccine, then congenital rubella syndrome would increase.[418] To tackle this, the courtiers are now recommending that not only should all women of child-rearing age be vaccinated, but so too should menfolk. Hence, between 2007 and 2008, Brazil vaccinated 70 million males and females aged 20 to 39, Argentina vaccinated 6.5 million men aged 16 to 39, and Chile did the same to 1.3 million Chilean men aged 19-29.[419] And from Australia comes the recommendation that all men between 17 and 44 should be vaccinated with the vaccine.[420]

The absurdity of man-made 'immunity' is highlighted by an outbreak of rubella amongst an Amish community in the United States in 1991. Known for shunning modern lifestyles and maintaining their traditional healthy ways, the Amish have always refused to be vaccinated. During the epidemic, 20 per cent of those children affected had no symptoms save the typical rash, and not a single pregnant woman contracted rubella.[421] The reason is not hard to find. Their immunity, 17 years after an earlier epidemic, had protected them and their unborn babies, just as the 1991 epidemic would protect the next generation and *their* unborn babies.

But the final word on the MMR vaccine goes to doctors at the prestigious Cochrane

Collaboration, a respected international organisation that conducts and publishes systematic reviews into the effectiveness of medical treatments. While acknowledging the decline in rubella worldwide, the Collaboration had identified 131 scientific articles on the efficacy of the MMR vaccine, and had reviewed 31 of them. Their conclusion was that not a single field study had identified the efficacy of the MMR vaccine.[422] Which is not surprising given that another study had noted that all vaccines have considerably less efficacy against mild disease than the published data would suggest; the reason being that highly biased observers will rate vaccines as being twice as effective as would less biased physicians.[423]

The polio scam

The disease that strikes the greatest fear into parents is polio. Images of withered limbs and iron lungs will stir many a parent to rush off and have their young child inoculated against the disease. But history reveals yet another sorry saga, one that is the undoubtedly the greatest vaccine scam of all.

Little is known about the disease before the 19th century, though some medical historians contend that the disease had afflicted ancient Egyptians, evident, so they claimed, in the withered limbs of people depicted on stelae. The first mention of acute paralysis in infants, as a disease entity, was by Michael Underwood, a London physician, in 1789.[424,425] The disease, he wrote, is characterised by 'debility of the lower extremities which gradually become more infirm, and after a few weeks are unable to support the body.' This syndrome he blamed on 'teething', 'foul bowels', or 'a fever'. No mention was made of the disease being contagious, nor did it pose a serious problem because it was so rare. Indeed, Underwood wrote that he had never seen a child die from the condition.

The culprit: poisons or germs?

As if a sign of the industrial revolutionary times, the first outbreak of infantile paralysis ever to be recorded happened in the early 1830s on Saint Helena, the island in the Atlantic Ocean, off Angola where Napoleon Bonaparte had been exiled.[426] Then, in 1835, an outbreak, affecting four young children, occurred in the English hamlet of Worksop, in Nottinghamshire.[427] The United States recorded its first outbreak, affecting 8 to 10 children, in Louisiana in 1841.[428] Norway was next in 1868 with 14 cases in Oslo, 5 of whom died; and Sweden had its first outbreak in the northern town of Umea in 1881, affecting 20 children. Sweden also had the world's first full-scale epidemic in 1887 in Stockholm, affecting 44 children, 3 of whom died; ominously, several of the cases were school age children rather than the typically affected under-fours.[429] France's first outbreak occurred in the village of Sainte-Foy-l'Argentière, near the city of Lyon, in 1885, affecting 13 young children.[430]

In the final decade of the 19th century the disease became more menacing. America's first epidemic struck Rutland County, Vermont in 1894, affecting 132 people, a quarter of whom were over the age of six: 18 people died and over 40 were left permanently paralysed in one or more limbs.[428,430–432] During the summer of 1907 'the crippler', as it later came to be called, struck many thousands of children, as well as adults, throughout the US's north-eastern states. New York City alone had over 2,700 cases. 'The crippler', and the ensuing public panic, returned in the summer of 1908, and it did so every summer thereafter for the next half-century, paralysing or killing nearly 400,000 Americans in total. The same was happening to populations throughout the industrialised world. But, tellingly, not in

underdeveloped countries.

Was it contagious? And, more importantly, what was causing it? In 1840 a German orthopaedist, Jakob von Heine, had suspected that the disease was contagious. Other researchers, however, thought not. Dr Charles Caverly, who had investigated the epidemic that struck Massachusetts in 1908, believed the culprit to be a toxin, not a micro-organism, since there was no evidence of contagion.[432] Nevertheless, during the 1890s, Swedish paediatrician, Oskar Medin also came to suspect that the disease was contagious. Hence, for a long time infantile paralysis, which was beginning to strike down not only infants, but also children and some adults, was known as 'Heine-Medin disease'. Later, it was renamed 'poliomyelitis' after researchers in the 1840s had identified that the grey matter (*polios*, Greek) of the anterior horns of the spinal cord's motor neurons (*myela*, Greek) were inflamed (*itis*, Greek).

But it was Medin's student, Ivar Wickman who believed he had found confirmation of the contagious nature of the disease: its greatest prevalence occurred amongst children living near busy river ways and main roads; places, he reasoned, where travellers with mild symptoms of the disease could spread it to others.[429] And, of course, the only thing that is contagious is germs. Wickman had also noted that dogs, too, were being paralysed. The problem is, dogs do not get polio: only humans do.

Nevertheless, the time was ripe for searching for an infectious agent. After all, Louis Pasteur had only recently promulgated his germ theory of disease. So researchers began searching in earnest for the germy culprit. In 1908, two Austrian scientists, Karl Landsteiner and Erwin Popper, thought they had found it. They had extracted diseased tissues from the spinal cord of a nine-year-old boy who had died from infantile paralysis, minced up the tissues, filtered them to remove bacteria, made a suspension in water, and had then injected the noxious mix directly into the abdominal cavities of two rhesus monkeys. The diseased foreign proteins and toxins, human DNA, cellular debris and possibly a host of prions, micoplasmas and viruses made the two monkeys severely ill; one died and the other was left paralysed in the legs. After dissecting the monkeys' brains, the researchers found that the damage to their central nervous systems was similar to that found in victims of acute infantile paralysis.

Tellingly, when the monkeys were made to drink the vile brew they did not become paralysed; nor did those that acquired the disease after the abdominal injection pass the disease on to other monkeys.[433] As British-born human rights campaigner and investigative journalist Janine Roberts noted in her report on the polio vaccine scam, published in the *Ecologist* in May 2004, this evidence alone debunked the claims that polio was highly contagious. Nevertheless, the World Health Organisation still credits these two researchers with discovering the poliovirus.

Inspired by the crude experiment of the Austrian researchers, two US researchers, Simon Flexner and Paul Lewis of the Rockefeller Institute for Medical Research, conducted a similar experiment.[429] They injected a similarly noxious brew derived from the diseased spinal cord of a victim into the spinal cord of a rhesus monkey, allowed it to wreak its damage, then extracted some fluid from the monkey's inflamed spinal cord and injected this into another monkey's spinal cord, and so on through a series of monkeys. Again, unsurprisingly, all monkeys were left paralysed. But through twisted logic, Flexner and Lewis concluded that the culprit was an unidentified virus. It was not until the mid-1930s that electron microscopy enabled scientists to identify viruses, those packets of genetic material surrounded by a protein coat that are at least 50 times smaller than bacteria.

And so began the search for a vaccine to defeat the assumed viral foe. Not once did government health scientists stop to consider that toxins, as Dr Charles Caverly had suspected, may have been the culprit, that paralysis can be caused by various chemical nerve poisons. As Ralph Scobey, a New York poliomyelitis researcher, had written—in a statement prepared for the US House of Representatives

Select Committee to Investigate the Use of Chemicals in Food Products, 1950-1952—there are over 170 diseases that cause polio-like signs and symptoms. Amongst them are those resulting from such nutritional deficiencies as beriberi (vitamin B1, or thiamine deficiency), pellagra (vitamin B3, or niacin deficiency), and scurvy (vitamin C, or ascorbic acid deficiency), as well as those from chemical poisoning.[434]

The Dutch physician Herman Boerhaave, for example, had in 1765 noted that if people inhaled the fumes of mercury they are 'rendered paralytic'.[435] Similarly, in 1924 the English scientist John Cooke had identified that inhaling the fumes of lead, arsenic or mercury, or drinking solutions of these poisons, often causes paralysis.[436] Further evidence of a link between infantile paralysis and toxins arrived in 1879 when French neurologist Alfred Vulpian found that lead poisoning in dogs caused not only paralysis of their extensor muscles, but also caused the same spinal cord lesions found in human victims of polio.[437] Indeed, Vulpian considered that what he had found was in fact poliomyelitis.

In 1881, the Russian researcher Popow had found that ingestion of arsenic could cause acute poliomyelitis.[438] And the Australian Dr Altman had noted that just prior to the first polio epidemic in Australia in 1897—in Port Lincoln, South Australia, which affected 18 children, none of whom died—phosphorus had been widely used in the district to kill an infestation of rabbits.[439] Indeed, an epidemic of paralysis had occurred in the spring of 1930 in Ohio, Kentucky, Alabama and Mississippi after people had drunk a commercial extract of Jamaican ginger contaminated with triorthocresyl phosphate.[440,441]

Many other reports have documented symptoms of polio in people who have been exposed to lead, arsenic, cyanide, and carbon monoxide.[442–446] And, as is typical of the so-called polio incubation period of seven to 10 days after the onset of fever, headaches, and vomiting, the symptoms of flaccid paralysis often occurred several days after exposure to the various chemical poisons. This had happened in Western Samoa in 1936, after people from 38 villages had been injected with an arsenical medication to treat an outbreak of the tropical skin and bone disease, yaws.[447]

But not once did government researchers consider this evidence. They ignored the fact that arsenic had been extensively used throughout the 19th century in paints and adhesive envelopes, in medicated soaps, in Fowler's solution—a remedy for numerous medical conditions including malaria and asthma—and as a fungicide in wallpaper. It is therefore not surprising that samples of Napoleon Bonaparte's hair, tested recently by Italian researchers, were found to contain levels of arsenic 100 times higher than would be found in people today.[448] And, in particular, the polio researchers ignored the fact that the mechanised spraying of an arsenic-based pesticide called Paris Green had been extensively used in all industrialised countries since 1868—the year of the first polio outbreak in Norway—to stop codling moth infestations destroying apple crops.

Nor did anyone connect the dots between the time when farmers began using the more deadly lead arsenate pesticide in Massachusetts in 1892 and the polio epidemic in the neighbouring state of Vermont two years later.[433,449] Nor between the three cotton mills in western Massachusetts that were extracting cottonseed oil, undoubtedly through the use of carbon tetrachloride, and the epidemic amongst people living in the river valleys downstream from the processing plants.[450]

As New York researcher Jim West has noted, 1907 was the year when the United States began high-volume production of carbon tetrachloride for use as a fumigant, herbicide, insecticide and cleaning solvent.[451–453] It was surely no coincidence that in the following year, small-scale polio epidemics erupted in Massachusetts. And, tellingly, 1915 was the year when high volume production of the neurotoxin chloral benzene began at two large chemical factories at Niagara Falls in upstate New York. The following year marked the first of America's major polio epidemics. This one swept through the north-

eastern states, affecting 27,363 people and killing 7,179. In New York City alone, nearly 9,000 people were afflicted.

Government health authorities did not recognise the absurdity of claiming that a virus was causing the epidemics of human paralysis, when horses and chickens and pigs and dogs were also becoming paralysed.[454] After all, the medical 'experts' claimed that poliomyelitis was a peculiarly human affliction.

As more and more industrial poisons continued to be sprayed onto crops, flushed into rivers, and released into the air, the numbers of people affected by the epidemics of polio in the industrialised world kept rising.

Then in 1945, DDT and hexachlorobenzene (HCB) entered the arsenal in the war on germs. DDT was readily sprayed on everything, from crops and fabrics, people and dairy cows, and other animals, to whole cities, to kill off mosquitoes, flies and every other pest. Adding to the insanity was the belief that polio was spread by flies; hence DDT was considered the saviour, not the culprit. By 1954, 3.1 billion pounds of persistent pesticides had entered the human environment, which is the equivalent of a large cup of endocrine-disrupting, nerve-poisoning chemicals for every person then alive.[455]

In tandem with what American endocrinologist Morton Biskind had called 'the most intensive campaign of mass poisoning in known human history',[456] the incidence of polio skyrocketed. Biskind was one of the heroes of this saga for he, together with Ralph Scobey, had tried to alert the scientific community and the US House of Representatives to the human health dangers of pesticides and their link to polio.[457,458]

In the nine years leading up to the release of DDT, from 1937 until 1945, the US had had just under 87,000 polio cases.[459] That's an average of 9,600 a year. But in the nine years between the release of DDT in 1945 and 1954, the United States had suffered nearly 300,000 cases of polio. That's over 33,000 cases a year. The greatest epidemic of all occurred in 1952, when 57,897 people contracted polio, over 21,000 of whom had some degree of paralysis. Thus, once DDT entered the human environment, polio cases had skyrocketed by 345 per cent.

Adding to the damning evidence was the fact that during World War II Allied troops were in the habit of dousing themselves and their camps with DDT to exterminate lice, mosquitoes, bedbugs, cockroaches and fleas. And, of course, the incidence of polio among US troops abroad was far higher than among those at home.[460,461] As the United States Army's Surgeon General had reported: 'It became apparent during the years 1941–45 that men of military age, born and brought up in the United States during the 1920s and 1930s, were more susceptible to poliomyelitis than their fathers had been in 1917–18. This was unexpected.'[462] Also unexpected was the high death rate from polio.

Moreover, there had been unexpected polio epidemics amongst US, British and New Zealand troops in the hot climate of the Middle East.[463,464] And yet the population at large had been spared, just as the Philippine population had been spared the polio epidemic that had occurred among American troops stationed in Manila in 1936.[465]

Besides DDT there had been another load of toxins that was being added to children's bodies. These were the chemicals in vaccines: residues of formaldehyde used in killing the accompanying germ; the preservative thiomersal (a mercury-based compound); and aluminium sulphate (alum), used for stimulating an antibody response to the germ. Any toxicologist would have predicted problems arising from injecting these known neurotoxins into children's blood streams. But evidently, the medical courtiers either hadn't known or weren't interested.

Polio arising from vaccination soon came to be called 'provocation polio', and it has been well documented. In an article in the *Archives of Disease in Childhood*, in March 1950, a British doctor, JK Martin, gave details of 17 cases in which poliomyelitis followed within 28 days of children being inoculated: 8 cases followed inoculation with the alum-precipitated diphtheria toxoid; 5 had had the combined diphtheria and whooping cough vaccine; 2 had injections against diphtheria with a fluid toxoid alone; and 1 had had a whooping cough inoculation alone.[466] Meanwhile, two physicians, Bertram McCloskey in Australia and Dennis Geffen in Britain, had also identified 'provocation polio' in some children and found that paralysis was far more likely to have started in the limb in which the injection had been given.[467,468]

Alarmed by these findings, the British Ministry of Health arranged for two medical statisticians to investigate the risk between polio and vaccines. After examining all case histories of the under-fives who had been afflicted during the polio epidemic of 1949, they identified 410 cases where there was evidence of provocation polio.[469] 'We must conclude,' they wrote in the *British Medical Journal* of 1 July 1950, 'that in the 1949 epidemic of poliomyelitis in this country cases of paralysis were occurring which were associated with inoculation procedures carried out within the month preceding the recorded date of onset of the illness.'

As the editor of the *British Medical Journal* wrote: 'It may be that children with general malaise of incipient poliomyelitis are not taken to the clinic for inoculation, but it seems more likely that the effect of injection is to produce paralytic symptoms in a patient who might otherwise have exhibited few if any signs and symptoms of poliomyelitis infection.'[470] But as Lily Loat, Secretary of the National Anti-Vaccination League in Britain, had commented: 'Whether the inoculation caused the paralysis or whether it made the limb more susceptible to the poison of infantile paralysis hardly mattered if the inoculation was to blame.'[41]

There was further evidence that cast doubt on the germ theory of polio. Given that exposure to a micro-organism produces immunity, then why did some monkeys and humans get a second attack of poliomyelitis?[471–473] The viral theory of poliomyelitis became totally unhinged when researcher John Toomey, who chaired a committee of the American Academy of Pediatrics to investigate childhood acute diseases, confirmed earlier research that no matter how intimately laboratory monkeys were exposed to polio-infected monkeys, polio is not contagious.[474] Indeed, Toomey had doubted that the disease induced by injecting diseased human tissues into the brains and abdomens of laboratory monkeys was the same as human polio.

Casting further doubt on the need for a vaccine had been the findings of Frederick Klenner, an American physician who had pioneered the use of massive doses of vitamin C therapy for treating disease. He had discovered that by injecting 25 to 30 grams of vitamin C each day into adult polio sufferers, they overcame polio, becoming well within three days, and with no paralysis whatsoever.[475]

Furthermore, another American physician, Benjamin Sandler, who had investigated the link between diet and polio, had found that sugar leaches calcium from bones, muscles and nerves, and that the weakened nerves were readily attacked by polioviruses.[476] Tellingly, countries with the highest per capita sugar consumption had had the greatest incidence of polio. And when do children tend to eat sugary foods, such as sweets, ice cream and soft drinks? During the summer months, which was exactly the season of polio epidemics. Confirmation of Sandler's theory that sugar was another culprit for polio epidemics came in 1949. In the spring of that year, Sandler had aired his ideas on radio in North Carolina, and afterwards many people had shunned such foods, the result being that whereas there were 2,498 polio cases in North Carolina in 1948, there were only 229 cases in the summer of 1949.[477]

Despite all this evidence, and despite the fact that even during an epidemic 95 per cent of people who contract the designated virus get no symptoms whatsoever—which surely begs the questions: Why are 5 per cent vulnerable? Why do less than two percent of cases result in flaccid paralysis? And for that matter, why do only half of these result in permanent paralysis?[478]—the virus hunters remained obsessed with creating a vaccine. They even claimed that polio epidemics were the result of excessive cleanliness, of children no longer being exposed to the wild virus and thus not acquiring natural immunity. After all, US army doctors during World War II had found widespread immunity to the suspected poliovirus amongst people in the Middle East, Asia and Africa, and no evidence of infantile paralysis.[433] In Turkey, infantile paralysis was even known as 'the American disease'. If it were true that cleanliness was to blame for the polio epidemics, then why hadn't the courtiers claimed the same for smallpox, cholera, diphtheria and so on?

The quest for the saviour

Stirring the virus hunters into action had been public panic, engendered by the media and by the National Foundation for Infantile Research, together with the presidential push to conquer the disease. Franklin Delano Roosevelt (FDR) himself had been crippled by the disease in 1921, at the age of 39, while holidaying with his wife and children at the family retreat on Campobello, a small Canadian island across the bay of Fundy from Maine. Three days before the first appearance of signs of the disease that would leave him crippled in both legs, he had fallen from his yacht into the icy, and polluted, waters of the Bay of Fundy; and the day before, he had swum in these polluted waters. As Jim West has noted, there were many industries in the area—paint, clothing and hardware manufacturers, breweries, tanneries, ship builders, and oil refineries—many of which were undoubtedly dumping organochlorine wastes, as well as lead, arsenic and mercury into the bay.[479]

When FDR later became president, he inspired Americans to help conquer this foe, just as he had inspired them during the war years to conquer their human foes. Fund-raising began with the President's Birthday Balls. But the most successful campaign was that of the National Foundation for Infantile Paralysis, which he had founded in 1937. And though the Foundation promptly decided that there was no cure for the disease, its campaign rapidly became a media event to garner small coin contributions from the American public; hence it later came to be called the 'March of Dimes'.

Years later, Herbert Ratner, a public health official who would become a fierce critic of the polio immunisation programme, revealed that in order to keep the statistical incidence of polio elevated, and hence engender public panic and the concomitant flow of funds for research, the Foundation paid physicians $25 for each reported diagnosis of paralytic polio.[480]

Scientists and scientific institutions everywhere rapidly began jumping on the polio bandwagon. Fierce competition to find a vaccine had already tarnished such endeavours. Two US researchers, Drs Maurice Brodie from New York City and John Kolmer from Philadelphia, independently had created vaccines and tested them on nearly 20,000 children whose parents had 'volunteered' them for trials during 1934 and 1935. The result had been disastrous: many children contracted polio, 12 were paralysed and at least 3 died. But in the frenzy to find a vaccine, people soon forgot that disaster.

To create a polio vaccine researchers needed plenty of polioviruses. The problem was that the three strains of the poliovirus, discovered during the 1930s, were not always present in the diseased spinal cord of victims. As investigative journalist Janine Roberts has remarked, 'This should have stopped the vaccine trials dead.'[481] But it didn't, for two researchers had found a ready source of polioviruses in the excrement of paralysed children. They also discovered what later came to be called the Coxsackie virus,

which also causes acute paralysis (but that inconvenient discovery was ignored in the stampede to create a polio vaccine).[482]

Another team of researchers, John Enders, Thomas Weller and Frederick Robbins, had found a way to grow these viruses, initially on the minced up tissues of aborted human foetuses, and later on the kidneys of monkeys, and for that feat they later received the Nobel Prize.[483] Years later, such cultures were discovered to harbour other virulent viruses, including the simian virus 40 (SV40), the simian cytomegalovirus (SCMV), and the simian immunodeficiency virus (SIV).

Thus, in the development of a polio vaccine, tens of thousands of monkeys were sacrificed in American laboratories. Jonas Salk, whose killed-virus vaccine was first off the rank, confessed to killing at least 17,000 rhesus monkeys in his research at the University of Pittsburgh.[433] Indeed he and his rival, Albert Sabin, had estimated that from a single monkey's kidney they could culture sufficient viruses to produce 6,000 doses; at three doses per child, that would mean roughly 47,000 monkeys would need to be sacrificed to vaccinate America's children.

After generating the living viruses on slices of monkey kidneys, Salk and his colleague, Julius Youngner (who incidentally received no kudos whatsoever for his part in the development of the vaccine), had killed them off with diluted formaldehyde and heat. At least, that's what they thought. The Salk vaccine was now ready for testing.

In 1954, in a trial of unprecedented size and scope, 440,000 second-grade school children in 44 American states, two Canadian provinces, and in the city of Helsinki, Finland, were injected with the vaccine. To ensure scientific rigour, epidemiologist Thomas Francis Jr., the official in charge of the trials, made sure that 210,000 first- and third-grade children were injected with a placebo; and that over a million children were observed as controls.

Salk had stipulated that children who received only one of the three injections were to be classified as 'not-inoculated'.[484] Ominously, that meant that adverse reactions to the initial dose would not be recorded.

Other worrisome signs were also emerging. In 1953, US researcher Albert Mitzer had warned that steeping the viruses in formaldehyde for nine days, as Salk had done, was insufficient time to kill all viruses.[485] After testing Salk's technique, the esteemed Swedish virologist Sven Gard had concluded that at least 12 weeks were needed. Even more alarming were the findings of Bernice Eddy, a researcher at the Laboratory of Biologics Control, a group at the US National Institutes of Health in Bethesda, Maryland.[486] She had been in charge of assessing the safety of Salk's vaccine and had discovered that monkeys, into whose brains and muscles she had injected Salk's vaccine, not only became paralysed, but also had live polioviruses in their spinal cords. Nevertheless, in the stampede to produce a vaccine, all this was ignored.

The National Foundation for Infantile Paralysis, which had paid for Salk's research and the mass trial, was so confident of the vaccine, it had ordered enough vaccine to inoculate nine million children.[487]

And so, on 12 April 1955, before an audience of 500 scientists and doctors and 150 reporters assembled in a makeshift newsroom in the Rackham Auditorium at the University of Michigan in Ann Arbor, and before 54,000 doctors watching the proceedings on closed-circuit television in theatres across the United States and Canada, and to people listening to radio broadcasts across America and, via *The Voice of America*, throughout the world, Dr Francis, announced that the Salk vaccine had proven to be totally 'safe, effective and potent'.

The news was greeted with jubilation. Across the nation church bells rang, air-raid sirens screamed, court-room proceedings were adjourned, and people stood for a minute's silence. Powerful symbolism was also afoot, for it was 10 years to the day since FDR, the most famous polio victim of all, had died; and it was 94 years to the day since the first salvoes had been fired against another enemy to announce the start of another war, the American Civil War.

Later that afternoon, William Workman, director of the Laboratory of Biologics Control convened a meeting of specialists to determine whether licences should be granted to five pharmaceutical companies for the manufacture of the vaccine. Despite the fact that the advisory committee had to assess 2,000 pages of information about the vaccine trial and the manufacturing procedures, it had agreed within two-and-a-half hours that the vaccine should be licensed. Today it takes at least one year to license a vaccine.[488] And William Workman, Bernice Eddy's boss, never said a word about her discovery that three of the six batches of vaccine, manufactured by Cutter Laboratories and submitted to her laboratory for assessment, had paralysed laboratory monkeys.[486]

Soon afterwards in Washington, DC, Oveta Culp Hobby, the US Secretary of Health, Education and Welfare, signed licences for the manufacture of the vaccine. Less than four hours later, Parke-Davis & Co, one of five drug companies under contract to the Foundation, made its first shipment of the vaccine.[487] Four days later, first- and second-graders in San Diego, California, became the first recipients of the vaccine.

The first shots in the war on polio

The jubilation was short-lived, however. Thirteen days after the war on polio had begun, after about a million children had been inoculated, the first casualties were announced: six cases of paralytic polio turned up in children who had received the vaccine manufactured by the Cutter Laboratories in Berkeley, California. The vaccines from Wyeth, Parke-Davis and Eli Lilly, three of the five manufacturers, were also implicated.[489] But it was the Cutter Laboratories that took the bad rap.

In the following days, health authorities discovered that of the 120,000 children who were inoculated with Cutter's vaccine, 40,000 had come down with mild symptoms of poliomyelitis, 51 were permanently paralysed—tellingly, in each case the paralysis began in the inoculated limb—and five had died.[488] Cutter's vaccine had also started an epidemic: 113 people in the inoculated children's families and communities were paralysed, and 5 had died. In his book, *The Cutter Incident, 50 Years Later*, paediatric immunologist Paul Offit, summed up the whole affair as '… one of the worst pharmaceutical disasters in U.S. history.'

The consensus was that the poliovirus had survived the formaldehyde treatment at the Cutter Laboratory, just as Albert Mitzer and Sven Gard had warned, and as Bernice Eddy had foreseen when she warned a friend: 'There's going to be a disaster. I know it.'[486]

The Cutter vaccine was immediately withdrawn, and on 7 May, the US Surgeon General called a halt to the vaccination programme, having been told by polio experts, to his surprise, that there were a variety of technical problems in the manufacture of the Salk vaccine, and that there could be no guarantees that the vaccine would be totally safe. But three weeks later, the vaccination programme was reinstated, thanks to the push by Jonas Salk and Thomas Francis to introduce 'improved' manufacturing procedures.

By late August that year, 150 million doses of the vaccine had been administered: half of the population under 40 had been injected with one dose, and one third had received three doses of the

vaccine.

Was the vaccine an immediate success? No, it was an abysmal failure. Four months after the resumption of the programme, Boston recorded 2,000 cases of infantile paralysis, a seven-fold increase compared with the same time the previous year, and 130,000 children had been vaccinated.[490] The incidence of paralytic polio in Rhode Island and Wisconsin was five times higher than it had been at the same time the previous year, in Vermont three times higher, in Connecticut twice as high, and six other north-western states recorded incidences at least 50 per cent higher.

Not only was the Salk vaccine failing to protect children, it was even contributing to epidemics. Alarmed by the outbreaks, various state health authorities called a halt to their vaccination programmes. Newark, New Jersey, abandoned its programme in June 1955; Idaho followed suit on 1 July 1955 after a polio outbreak hospitalised 79 children and killed seven; and Utah did the same on 12 July.[490]

The vaccine's reputation fared little better during 1956. According to the *New York Times*, the national incidence of infantile paralysis during the first four months of the year had increased by 12 per cent over the rates during the same period in 1955.[491] Alarmed by the vaccine's track record in the United States, all European countries except Denmark called a halt to their vaccination programmes, just as Canada had done in July 1955. And by January 1957, 17 US states had followed suit.

A rose by any other name

Children certainly weren't being protected. But the medical courtiers were determined to at least protect the US President, as well as Jonas Salk, the vaccine manufacturers, and themselves, from the humiliation of the vaccine being revealed as an outright failure. After all, the nation's reputation was at stake. Something had to be done.

And indeed it was. Taking a perverse interpretation of Shakespeare's famous lines—'What's in a name? that which we call a rose by any other name would smell as sweet'[492]—the courtiers began to rename polio to statistically create the illusion that polio was declining. The results of this charade would eventually go down in history as one of the most successful disappearing acts of all time.

First, they abandoned the World Health Organisation's definition of polio—the presence in a person of paralytic signs for 24 hours—and in its place adopted Salk's definition of polio, a definition he had used during his mass trial: the presence in a person of paralytic signs for at least 60 days. Because up to 98 per cent of people would recover within 60 days:[478] 'this nifty but dishonest administrative move,' wrote Australian research scientist Dr Viera Scheibner, 'excluded more than 90 per cent of polio cases from the definition of polio.'[493] That was undoubtedly an underestimate.

Second, all cases of polio that occurred within 30 days of inoculation were to be reclassified as 'pre-existing', just as Salk had done during the mass trial of his vaccine.[433]

By 1958, thanks to advances in microbiology, the courtiers alighted on a third ploy: shuffle as many cases of polio as possible into the categories of other diseases. Thus, where the victim suffered from inflammation of the membranes that protect the brain and spinal nerve cells and cause muscular weakness and pain—a syndrome that was previously diagnosed as 'non-paralytic poliomyelitis'—the disease was to be reclassified as 'aseptic meningitis' or 'viral meningitis'.[494]

And the fourth trick was to initially classify all cases of paralytic polio as 'acute flaccid paralysis'; and if no poliovirus could be found in the excrement of the patient, then the disease was definitely not to

be classified as polio. It could be classified as Coxsackie virus infection, or as ECHO virus infection, both of which are clinically indistinguishable from paralytic poliomyelitis.[495] Or it could be classified as Guillain-Barré Syndrome, which some physicians suspect Franklin Delano Roosevelt had contracted.[496] Or it could be called myalgic encephalomyelitis (ME), a syndrome that first surfaced in 1954, and would later be named chronic fatigue syndrome.[497,498]

There was a final ploy the courtiers had hit upon to reduce the statistical incidence of polio. They redefined a 'polio epidemic': prior to 1955 it was 6 cases per 100,000 people, but after 1955 it was defined as 35 cases per 100,000.[499]

Unsurprisingly, the official statistics would show that the incidence of non-paralytic 'polio' had declined by 95 per cent between 1955 and 1960; and that paralytic polio had declined by 85 per cent.[459]

The person who had blown the whistle on the statistical manipulation was biostatistician Bernard Greenberg. A former Chairman of the Committee on Evaluation and Standards for the American Public Health Association, he had informed colleagues at the 120th Annual Meeting of the Illinois Medical Society in May, 1960: 'My primary concern, my only concern, is the very misleading way that most of this data has been handled from a statistical point of view.'[500] And, further: 'A scientific examination of the data, and the manner in which the data were manipulated, will reveal that the true effectiveness of the present Salk vaccine is unknown and greatly overrated.'

The living virus lurking in the Salk vaccine

Worse was to come. Bernice Eddy, the researcher at the National Institutes of Health (NIH), who had already found problems with Salk's vaccine, had been inspired by the work of fellow researcher Sarah Stewart. The latter had discovered that viruses could cause cancer. That was the last thing their superiors at the NIH wanted to hear. So, without her boss's authorisation, Eddy went back to her laboratory to make further tests on the Salk vaccine. After injecting the monkey kidney tissues, upon which the polioviruses had been cultured, into 23 hamsters, she was horrified to discover that 20 of them grew large cancerous tumours.[501] By 1960, she knew that a virus, soon isolated by Ben Sweet and Maurice Hilleman (two scientists at the Merck research laboratories in Philadelphia), and dubbed SV40 because it was the 40th simian virus to be discovered, was the culprit, and that it had been far more resistant to being killed by formaldehyde than the poliovirus. She also discovered that it was capable of infecting recipients of the vaccine.[502,503]

Needless to say, Eddy's findings caused consternation amongst the medical courtiers. They certainly had cause for concern. Estimates were that from 10 to 30 per cent of Salk's vaccine had been contaminated with SV40, and they knew that about 98 million American children and adults—about 60 per cent of the American population—had been injected with the vaccine.[504] Several studies at the time had shown that SV40 caused brain tumours in experimental animals, and could cause cancer in human tissues.[505–508]

That meant that in the seven-year period from 1954 until 1961, as many as 30 million Americans, and up to 100 million people worldwide, were injected with a ticking time bomb from rhesus monkeys. As of 1968, the US had exported close to half a billion doses of the Salk vaccine. Britain alone imported 10 million doses.[501]

But according to Herbert Ratner, Director of Public Health in Oak Park, Illinois, editor of the *Bulletin of the American Association of Public Health Physicians*, and the man who chaired the meeting at which Greenberg had revealed the courtiers' statistical sleight of hand, 'The National Foundation for Infantile

Paralysis and the US Public Health Service, who were recovering from previous troubles, were well aware how upset parents would be to discover that Salk anti-polio vaccinators, like a hoard of hungry mosquitoes, had descended on their children with African monkey viruses.'[501] Accordingly, government courtiers 'did what they could to suppress and minimise the discovery.' Even the National Cancer Institute soft-pedalled on the revelations to avoid publicity on the matter.

Although two of the four vaccine manufacturers, Merck and Parke-Davis, immediately recalled their polio vaccines, the courtiers at the US National Institutes of Health, as well as those in the departments of health in Britain and Canada, were more intent on saving reputations than children, and refused to recall the rest of the supply, fearing the public backlash would jeopardise the vaccination campaign.[509] Thus they knowingly allowed even more millions of people to be dosed with a cancer-causing virus. Tellingly, the US Public Health Service concealed that secret for 40 years.[510]

After a flurry of tests, the courtiers quickly concluded that there were no health risks from the Salk vaccine. The first public disclosure that the vaccine was contaminated with a monkey virus, and that Merck had withdrawn its vaccine, was buried on page 33 of the *New York Times* on 26 July 1961. When asked to comment, the US Public Health Service proclaimed that there was no evidence that the virus was dangerous.[511]

As for Bernice Eddy, because she had breached the official wall of silence by discussing her findings with other scientists, she, like many other whistle-blowers, was persecuted and demoted.

Out of Africa

The mood amongst physicians was rapidly turning sour. One writer in the 25 February 1961 edition of the *Journal of the American Medical Association*, aptly summed up the consensus: 'It is now generally recognised that much of the Salk vaccine used in the U.S. has been worthless.'[512] Such sentiments spurred the American Medical Association to exert pressure on the Federal Government to abandon the Salk vaccine and replace it with the Sabin vaccine. It was cheaper, it had been tested on tens of millions of children in Latvia, Estonia and Kazakhstan—albeit contaminated with SV40—and because it was taken by mouth on sugar cubes or as a liquid preparation, not injected, it was easier to administer. Because the vaccine contained a live but weakened poliovirus, the courtiers also believed it would more readily confer 'immunity' on recipients than the Salk vaccine. Data on the Salk vaccine showed that antibody levels two to four years after inoculation were so low that repeated, if not frequent, booster shots would be required to retain 'immunity.'[513] Moreover, Sabin's attenuated virus would remain in the intestinal tract of the recipient for at least six to eight weeks after the time of vaccination, and thus it would readily infect other members of the community—admittedly without their consent—but that was of no concern to the courtiers. At least it would ensure widespread 'immunity' to the weakened virus.

The courtiers were also confident in the safety of the oral vaccine, since researchers at the National Cancer Institute, in evaluating the carcinogenicity of SV40, had quickly concluded in 1963 that there were more dangers from Salk's injectable vaccine than from Sabin's oral vaccine.[514] Seven years after the horse had bolted, researchers conducted a follow-up study and found no evidence of deaths in newborn recipients of the Sabin vaccine.[515]

By 1963, the Sabin vaccine was off the starting blocks, with instructions to the three remaining manufacturers, Parke-Davis, Wyeth-Lederle, and Pfizer, to ensure that no SV40 was present in the vaccine. Merck had called a permanent halt to producing polio vaccine; in its letter to the US Surgeon General, it cited technical difficulties in removing all simian virus contaminants, 'which may be difficult if

not impossible to detect at the present stage of technology.'[516]

That letter alone should have been sufficient warning to health officials that to continue with the vaccination programme was foolhardy. But policy was being made on the run, and the warning fell on deaf ears. To ensure no SV40 was present in any vaccine, regulations had been introduced in March 1961, stipulating that manufacturers were to test kidney cell cultures for at least 14 days to detect the presence of the virus. But according to scientists at the Division of Biologics Standards at the NIH, where Bernice Eddy had worked, at least five weeks were needed to detect SV40.[517] Curiously, the regulations did not stipulate that old seed stocks were to be discarded and started afresh, even though they undoubtedly carried SV40-contaminated poliovirus hybrids (recombinant mutants). In other words, the testing requirements to protect children from a cancer-causing virus were crude and unreliable, and had been based on poor science.

The US National Institutes of Health had also recommended to Sabin, and to Salk, that they switch from using the kidneys of rhesus monkeys to those of African green monkeys, which supposedly were not infected with SV40.

The change to using kidneys of African green monkeys was made quickly and quietly to ensure that the public would remain clueless to the fact that tens of millions of people had been infected with a cancer-causing virus. But, in the frenzy, no one had bothered to evaluate whether African green monkeys also carried viruses that could infect humans. That would later prove to have been a disastrous mistake.

What the courtiers also failed to see was that the live-virus vaccine was potentially more dangerous than Salk's vaccine. As was discovered later, the weakened viruses could revert to virulent strains—and hence give recipients paralytic polio; and through the shedding of the virus in their stools, those inoculated with it could spread it to other members of the community.[518] Ominously, the people who could least afford to be infected, either directly through inoculation, or indirectly by catching poliovirus from the recipient of the vaccine, were those with weak immune systems.

Concern about the Sabin vaccine harbouring unidentified and virulent viruses from African green monkeys spurred one of the two remaining Sabin vaccine manufacturers, Lederle, to request that studies be done on the vaccine. Together with the Bureau of Biologics, Lederle scientists found the simian cytomegalovirus (SCMV) in the kidneys of all 11 monkeys tested.[519] Yet again, the courtiers decided to suppress the information, as Australian-born virologist, John Martin, who had worked at the Bureau of Biologics during the 1970s, discovered. And so the story of SV40, like the virus itself and the 39 other simian viruses, remained hidden for another 20 years.

The Sabin vaccine's fall from grace

By the 1970s, the total number of paralytic polio cases had dropped to less than 25 per year, according to the US Centers for Disease Control and Prevention (CDC).[520] But in 1976, Jonas Salk, Sabin's arch-rival, testified before a Senate subcommittee that Albert Sabin's oral vaccine was the 'principal if not the sole cause' of all reported cases of paralytic polio in the United States since 1961. 'To avoid occurrence of such cases,' he warned, 'it would be necessary to discontinue the routine use of the live-polio vaccine.'[521] Over the years, each had accused the other of causing polio. And they were both right!

The CDC did at least acknowledge the dangers posed by the vaccine when it reported that all of the 21 cases that occurred during 1982–1983 were caused by the vaccine.[520]

There were hiccoughs with the vaccine overseas too. In Oman, for example, an epidemic of paralytic polio erupted six months after authorities had completed vaccinating 98 per cent of children. The highest incidence was in children under two-years-of-age, 87 per cent of whom had received three

doses of the Sabin vaccine.[522]

In Romania, between 1984 and 1992, the risk of vaccine-associated paralytic polio was 14-times higher than in the United States, and 17 times higher than in other countries. Researchers were at a loss to explain why.[523] Moreover, doctors who examined Romanian children found that the chance of contracting paralytic polio was directly related to the number of antibiotic injections they had received: a single injection within one month of having received the Sabin vaccine raised the risk eight times; two injections, 27 times; 10 or more, 182 times.[524] This was nothing other than provocation polio.

Indeed, over 80 published studies on populations in 16 countries on all continents had shown that vaccine-associated paralytic polio was rife.[525]

Sabin's vaccine eventually fell from grace in 1994 when CDC researchers confirmed that the main cause of polio in the US was the vaccine.[526] Their recommendation was that the United States should switch back to the injectable, killed-poliovirus vaccine. But because production would take several years to get up and running, and because of heavy lobbying by the sole oral vaccine manufacturer, Lederle, the CDC would ordain an interim period when two shots of the Salk vaccine would be administered followed by two doses of the Sabin vaccine. Thus, in 2000 the Sabin vaccine became a mere footnote in the saga of medical history, though the effects of its Green monkey viral contaminants would continue.

Polio's great disappearing act

Certainly the medical courtiers had managed to pull off polio's great disappearing act. Thanks to the 'rose by any other name' ploy, Los Angeles County health authorities would note in 1967 how the statistics on non-paralytic polio had been swapped for those of viral meningitis. According to Christopher Kent, writer and later President of the Council on Chiropractic Practice, the report for 1967 stated: 'All cases [of polio] now reported as meningitis.'[527]

		Viral meningitis	Poliomyelitis
July 1955	50	273	
July 1961	161	65	
July 1963	151	31	
September 1966	256	5	

Nationally, non-paralytic polio's decline and demise, and viral meningitis's birth and reciprocal growth, are readily apparent in the following table presented by Gary Krasner, Director of the Coalition for Informed Choice, New York City:[528]

		Viral meningitis	Poliomyelitis
1951–1960	0	70,083	
1961–1980	102,999	589	

1983–1992	117,366	0

In 1997, the US Centers for Disease Control and Prevention did admit that 30,000 to 50,000 Americans contracted viral meningitis each year.[529] 'That's where all those 30,000–50,000 cases of polio disappeared after the introduction of mass vaccination,' declared Australian scientist Viera Scheibner.[493]

The aftermath

In 1954, a polio-like syndrome had surfaced, initially called 'myalgic encephalomyelitis' (ME) by British scientists. But because of pressure by their American counterparts, the name was changed by the 1980s to 'chronic fatigue syndrome' (CFS).[497,498] As British researcher Betty Dowsett would note, by emphasising a 'fatigue definition' of the syndrome, and the associated psychiatric inference in the term, the American courtiers managed to distract attention from serious research into the viral aspects and clinical features of the syndrome.[530] After all, some researchers had noted that the syndrome was indistinguishable from non-paralytic polio, once known as 'abortive polio', or 'atypical polio', and, in particular, the chronic phase known as 'post-polio syndrome'.[531–534] It had simply been rebadged.

More to the point, there were suspicions that ME/CFS was caused by any combination of the 69 strains of enteroviruses that are genetically related to the three strains of poliovirus. These include the Coxsackie viruses and ECHO viruses. At least 69 epidemics of the syndrome have been recorded since 1934.[534] But, as Dr Richard Bruno, American clinical psychophysiologist, expert in post-polio sequelae, and Director of the Post-Polio Institute, International Centre for Post-Polio Education and Research and Fatigue Management Programs at New Jersey's Englewood Hospital and Medical Center, explains, '… something unexpected, frightening and unrecognised happened after the polio vaccine was distributed: the number of cases of CFS/ME went through the roof.'[535] The reason?

'It appears that the vaccine that eliminated polio had an unintended consequence,' Bruno continues.[535] 'The elimination of the three types of poliovirus left a vacuum that had to be filled. Just as a flock of dominant and aggressive blue jays blocks less aggressive robins from roosting in your back yard, poliovirus are the bluejays of enteroviruses, the viruses that live and grow in your intestines. When poliovirus "blue jays" disappeared from your intestines thanks to the vaccine, other enteroviruses "robins" took over the poliovirus' old intestinal breeding ground and filled the vacuum. With the polioviruses gone other enteroviruses were able to multiply, spill into the bloodstream and enter the spinal cord and brain.' In other words, by changing our intestinal microflora, the Salk vaccine, and particularly the oral Sabin vaccine, indirectly caused ME/CFS.[536]

Given that about one million Americans suffer from ME/CFS, as do about 200,000 Britons, 90,000 Canadians, 65,000 Australians and 12,000 New Zealanders, and that a quarter of them are severely incapacitated, we can begin to understand the enormous consequences of the polio vaccination campaign.

But one researcher believes that ME/CFS, as well as many other diseases of epidemic proportions, including autism and attention-deficit-hyperactivity disorder in children, as well as cancer, fibromyalgia, and a host of neurological disorders, are the direct result of Sabin's polio vaccine. Dr John Martin, the Australian virologist who had worked at the Bureau of Biologics during the 1970s, and then went on to become Professor of Pathology at the University of Southern California School of Medicine, and to establish the privately-funded Center for Complex Infectious Diseases in Rosemead, California, had taken a keen interest in the simian cytomegalovirus (SCMV). His interest had been whetted after discovering that SCMV was a green monkey contaminant in the Sabin polio vaccine, and that the medical

courtiers had suppressed that information, not only from the public, but also from the scientific community. Using a recently developed, sophisticated analytic technique called the polymerase chain reaction (PCR) that enables scientists to identify gene sequences, he had identified DNA fragments of SCMV in many people suffering from ME/CFS.[537,538] Normally our immune system reacts to a viral infection with an inflammatory response, but curiously the viruses he extracted and cultured from brain biopsies, the cerebrospinal fluid, and the blood of people with ME/CFS, had somehow managed to evade that immune response.[539,540]

Equally disturbing was his discovery that fragments of these immune-eluding viruses readily combined with fragments of bacterial DNA (which he dubbed viteria), with DNA fragments of other viruses, and with DNA fragments of our own genes, potentially including our own oncogenes (cancer-causing genes). And they can capture, amplify and mutate cellular genes without our immune system being any the wiser. Thus he coined the term 'stealth viruses'.[541]

To prove the point, Martin injected cats with stealth viruses taken from people suffering from ME/CFS. The cats ended up with encephalopathy (brain dysfunction).[542] And when an epidemic of encephalopathy struck people in the Mohave Valley, Arizona, in 1996, they too showed the tell-tale signs of a stealth virus.[543] Further proof of the epidemic's stealth-viral origin emerged in the case of a young boy from the Valley who was so severely affected that doctors expected him to die within months. He recovered after being prescribed an antiviral drug.[544]

In essence, the polio vaccine has been responsible for creating new and potentially virulent life forms called 'stealth viruses', which, as Martin has portrayed them, are 'nature's biological weapons programme'. Except that they were spawned in human laboratories.

Unsurprisingly, the courtiers were furious, for Martin had dared to reveal that the emperor's naked body was not only appallingly grubby, but also a festering mess. Despite repeatedly urging the CDC to investigate the issues, his efforts have come to nought. His proposals for funding to investigate the risks, submitted in 1978, and again in 1995, were rejected, and several abstracts submitted to CDC meetings were also rejected. He has even been denied the right to test patients for stealth viruses. The courtiers' message is clear: if you break the code of silence and impugn the excellence of the emperor's new clothes, then you will be ostracised, if not pilloried.

And whatever became of SV40? Well, if the courtiers had heeded the warnings from researchers in the 1960s—that, when injected into hamsters, SV40 caused ependymomas, a rare form of brain cancer; that it caused chromosomal aberrations in human kidney cell cultures; and that, when injected into human cells, it produced tumours—then they may have foreseen that sooner or later some of the recipients of the Salk vaccine, or their children, would end up with various cancers.[505–508]

If they had but trawled through the details of all epidemiological studies on the impact of SV40, they would have found, as did a group of Canadian and Italian biostatisticians,[545] that according to an Australian study, children who had been inoculated with the Salk vaccine had a 40 per cent greater risk of developing cancer within 10 years of the shots than those who hadn't been inoculated, and for children over the age of one, the risk was 69 per cent higher.[546] According to the Connecticut Tumor Registry, children born between 1956 and 1962 had twice the risk of central nervous system tumours compared with other children, and that 66 per cent of medulloblastomas, a variety of brain cancer, contained SV40.[547] According to a group of researchers at the Boston Medical Center, not only was there an increased incidence of ependymomas in children born between 1956 and 1962, but the children of mothers inoculated during these years had two-and-a-half times the risk of getting cancer; and if their mothers had been inoculated during the first four months of their pregnancy, then the risk was 13 times

greater than expected, and if inoculated in the first three months of pregnancy, then the risk of cancer was more than 15 times greater.[548]

The courtiers' game of make-believe should have been over in 1988, when a team of US physicians and scientists, using the newly developed polymerase chain reaction, found that half the children they examined who had choroid plexus papilloma (a form of brain cancer), and all but one of the 11 children with ependymomas, had the DNA-footprints of SV40 in their tumours.[549] Several years later, the same researchers found that 14 of 17 children with choroid plexus papillomas and ependymomas had the SV40 footprints.[550]

Then in 1993 more bad news arrived. Italian researchers had found that when SV40 was injected into hamsters it caused mesothelioma, a lung cancer normally associated with exposure to asbestos.[551] One of the researchers, molecular pathologist Michele Carbone, now working at Chicago's Loyola University Medical Center, followed his hunch and he and his colleagues, using the polymerase chain reaction, examined the lung tissues of patients with mesothelioma. What they found was astounding, particularly since the disease was blamed entirely on asbestos: 60 per cent of cases had the tell-tale DNA footprints of SV40.[552]

Was SV40 the reason why mesothelioma first emerged during the 1950s? And was that the reason why cases of mesothelioma in Turkey, which didn't start its polio vaccination programme until the 1970s, and in Finland, which never used SV40-contaminated vaccine, showed no evidence of SV40?[553,554] Given that 80 per cent of cases of mesothelioma in the western world develop in individuals who have been exposed to excessive levels of asbestos, and yet only a fraction of people thus exposed will go on to develop mesothelioma, is SV40 the hidden factor, a co-carcinogen?[555] Carbone and his fellow research scientists certainly believe so.[556–559]

Two years later, Carbone and his colleagues would detect SV40 in 60 per cent of osteosarcomas, a type of bone cancer.[560]

By 1996, dozens of scientists had reported finding SV40 in a wide variety of brain and bone cancers, in mesothelioma, and in non-Hodgkin's lymphoma. But the courtiers were sceptical. Between 1997 and 2003, more than 25 peer-reviewed studies were published on the link between SV40 and mesothelioma, and 16 other studies had been published on the connection between SV40 and brain and bone cancers, non-Hodgkin's lymphoma, and other cancers, including kidney tumours.[510,556–572] And by 2003, the link between SV40 and these cancers had been identified in 18 developed countries. But the courtiers remained sceptical, at least publicly.

On the issue of brain cancers, for instance, researchers from Italy, France, and China had found SV40 footprints in a high percentage of all types of brain cancers. The Italian team headed by Professor Mauro Tognon, at the University of Ferrara's School of Medicine, found that SV40 was present in 83 per cent of choroid plexus papillomas, 75 per cent of ependymomas, 47 per cent of astrocytomas, and 37 per cent of glioblastomas.[567,568] The French team at the International Agency for Research on Cancer, in Lyons, had found SV40 in all brain tumour types, including 56 per cent of ependymomas in children, 38 per cent of choroid plexus papillomas, and 29 per cent of medulloblastomas.[569] And a Chinese team at the Department of Neurosurgery at Xijing Hospital in Xi'an had found SV40 in each of the common brain tumours, including all cases of ependymomas and choroid plexus papillomas, 90 per cent of pituitary adenomas, 73 per cent of astrocytomas, 50 per cent of glioblastomas, and 33 per cent of medulloblastomas.[570] Ominously, the Italian team had discovered that SV40 was present in 8 per cent of normal brain tissue.[568]

SV40 had even been detected in 45 per cent of sperm specimens and 23 per cent of blood samples collected from healthy adults.[571,572]

Revelations

One question arising from all this is, how does SV40 cause cancer? According to research scientists, the answer is that the simian virus 40 acts like a hit-and-run driver, wreaking havoc as it goes.[573] Its power lies in one of its proteins, which binds to and inactivates our body's own tumour-suppressing proteins, particularly a protein known as 'the guardian of the genome,' or less poetically 'p53'.[574] This is probably the reason why cancers that contain SV40 are less likely to be responsive to chemotherapy and radiation therapy.[575] To make matters worse, SV40 also causes cellular aberrations in immune system cells, as is readily apparent in its link to lymphomas.

But the big question is, how did SV40 end up in children who were born long after Salk's SV40-contaminated vaccine was removed from vaccination programmes? Many researchers suspect that the virus can be passed from a mother to her unborn child, that it can be sexually transmitted, and that it can be transmitted through blood transfusions.[568,572,576,577] Undoubtedly a person's health also plays a huge part, because researchers at Baylor College of Medicine in Houston, Texas, noted that six per cent of hospitalised children, many of whom were immune-compromised, had antibodies to SV40.[576]

Mysteriously, American researchers had noted that some mothers of children who had brain tumours containing SV40 had no evidence themselves of antibodies to the virus.[578,579] A case in point is that of Alexander Horwin, a young American boy who contracted a malignant brain tumour, medulloblastoma, eight months after having received the oral polio vaccine.[575] He died five months later, four months shy of his third birthday. Neither his parents, nor his placenta, carried any sign of SV40; but according to four independent laboratories, SV40 footprints were apparent in his tumour. Believing that their son's cancer was caused by the polio vaccine, Michael and Raphaele Horwin, from a beachside suburb in Los Angeles, would go on to establish the SV40 Foundation, to help other parents of children with cancer, and to alert the public and the US Congress to the issues surrounding SV40 and the polio vaccine.

But that mystery may have been solved, not by a scientist, but by a lawyer. Over the years, Philadelphia-based lawyer Stanley Kops had represented allegedly vaccine-damaged plaintiffs in litigation against the US Government and vaccine manufacturers. He had read and heard a lot about manufacturing procedures. And he wasn't impressed. He was particularly irked by statements made by representatives of Lederle, the leading US manufacturer of oral vaccines since 1963, and the sole manufacturer from 1978 until 2000; the company prided itself on the fact that it had distributed 650 million doses of the vaccine in the United States since 1963. At an international conference in early January, 1997, entitled 'Simian Virus 40: A Possible Human Polyoma Virus Workshop', organised by the US Department of Health and Human Services, and attended by officials from the FDA and the CDC, as well as scientists from around the world, a representative of Lederle had assured the assembly that Lederle's oral polio vaccine was SV40-free, and that their testing had proved it so.[580] Kops knew it wasn't true.

So, three years later, after reading through numerous scientific articles on SV40, Kops blew the whistle amongst the science community, and in the process undoubtedly set a precedent for being the only lawyer to have an article published in a peer-reviewed medical journal.[581] He warned readers of the November/December 2000 edition of *Anticancer Research* that all scientific literature and research on SV40 up until that time had been based on the assumption that SV40 had been removed from all oral polio vaccines since 1963. But he knew that the evidence suggested otherwise. Even though manufacturers

were required by law from 25 March 1961, to ensure there were no extraneous microbial contaminants at each stage of the manufacturing process, documents subpoenaed from Lederle during litigation involving its oral polio vaccine failed to reveal that the original Sabin SV40-contaminated poliovirus seeds had been removed from manufacturing procedures, or that procedures for neutralising and testing the vaccine lots cultured from these seeds had been carried out. Tellingly, neither Lederle nor the FDA could produce documented evidence that all the poliovirus seeds were tested and that they all passed the mandated standards.[582]

Kops didn't pull his punches when he testified three years later, in September 2003, before the US House of Representatives Subcommittee on Human Rights and Wellness of the Committee on Government Reform, on the subject of 'The SV40 Virus: Has Tainted Polio Vaccine Caused an Increase in Cancer?'. Coincidentally, the topic was the result of efforts by Michael and Raphaele Horwin to persuade the Chairman of the Committee, Congressman Dan Burton, to investigate the relationship between SV40 and public health.

In his damning testimony Kops reeled off a litany of allegations against Lederle:[583] that it knew that 10 per cent of the green monkeys it used were infected with SV40; that from 1963 until 1980 it cultured the master seeds of two of the three strains of poliovirus on rhesus monkey kidneys, not those of green monkeys; that it knew that 50 to 60 per cent of the rhesus monkeys it used were infected with SV40; that from the 1980 until 2000 it cultured vaccines on monkeys previously used in experiments; and that it had continually failed to follow the mandated requirements for testing its vaccine.

A year earlier, in July 2002, Kops had presented the same issues in a presentation at an Institute of Medicine (IOM) meeting on 'SV40 Contamination of Polio Vaccine and Cancer' in Washington, DC. The Institute had been so concerned that the regulatory agency, the FDA, had made no response to Kop's allegations that its report in October of that year stated: 'The committee urges that the FDA or other agencies address these claims to try to resolve the uncertainty regarding the possibility of exposure to SV40 after 1963.'[584]

And the response of the FDA to the IOM's request? Deathly silence.

But Lederle was not the only drug company to have made an SV40-contaminated polio vaccine. An international team of scientists had undertaken the task of testing the quality of 13 oral vaccines manufactured around the world. Alarmingly, they found that a major eastern European manufacturer had made an SV40-contaminated vaccine from the early 1960s until about 1978, and had distributed it not only to eastern Europe and the USSR, but also to Africa and Asia.[585]

The big question arising from this is, could the simian virus 40 be transmitted via SV40-contaminated oral polio vaccine? The answer had arrived in 1962, when two American researchers discovered that SV40, as with the poliovirus, could be found in the faeces of recipients of the oral vaccine for up to five weeks after taking the vaccine.[586] The faecal-oral route could account for any contagion.

Meanwhile, Herbert Ratner, the public health official who had been a fierce critic of the polio vaccination programme, re-entered the scene. He had retrieved some old vials of Salk vaccine from 1955 that he had kept in his refrigerator, and he presented them to Michele Carbone and his fellow scientists to run some tests. What they found was alarming. There was not just one strain of SV40 virus, but two: the second was much slower growing and hence would have escaped detection by the mandated protocols.[587] They would have needed at least 21 days culture, not 14 days as was the mandated safety protocol, to have been detected. And what was even more alarming was that the original flawed testing protocols were the only safety tests required right up until 2000. The more sophisticated PCR tests had never been made a requirement.

Notwithstanding all this evidence, the courtiers of the US Public Health Service, from its regional castles to the grand palace in Atlanta, Georgia, could not, and would not, accept any of it. They quibbled about the reliability of the PCR tests, and, in particular, they refused to accept any connection between SV40 and cancer. However, they did at least accept that SV40 came from contaminated polio vaccines.

Thus in 2004, the National Cancer Institute, citing two studies that had shown that antibodies to SV40 were no higher in people with non-Hodgkin's lymphoma than in controls, stated: 'Studies investigating the possible connection between SV40 and human cancer have been inconclusive.'[588]

To state otherwise would have meant admitting to a hideous error. And worse, at least for the courtiers, it would have undermined the whole vaccination programme that purported to prevent disease, not cause it. And so they fell back on the old political ploy of casting doubt about an inconvenient truth. It had been used before, and it would be used again later.

The Legacy

Imagine this: You're in charge of assessing the safety of medicines for children. You're presented with a list of recipes that the manufacturer claims will prevent a variety of infectious diseases when injected into children's bodies. You find that each one contains a veritable cocktail of chemicals, so you check the material safety data sheets on each. Unfortunately, there is virtually no information about the long-term, low-dose toxicity of any ingredient. But material safety data sheets and other toxicological data do list the acute, higher-dose toxicity of each: formalehyde, mercury and aluminium compounds (although thiomersal, an ethyl mercury preservative, was supposedly removed from most vaccines, it is still present in both the infant and adult influenza vaccines),[589] sulphate and phosphate compounds, phenol, 2-phenoxyethanol, borax, glutaraldehyde, polysorbate 20/80, polyethylene 9–10 nonyl phenol, sorbitol, aspartame, beta-propiolactone, benzethonium chloride, monosodium glutamate (MSG), and so on. In fact, there are about 40 chemical compounds you need to check off.

You find that many of the ingredients, at least in high doses, are neurotoxic, mutagenic or carcinogenic; some are toxic to the gastrointestinal system, the respiratory system, the reproductive system, the cardiovascular system, the blood and the liver; and others interfere with a child's development. Unfortunately, you have no information whatsoever about the synergistic effects of all these chemicals together, about how they interact.

Next, you assess the safety of injecting into a child the various antifungal and antibiotic agents, antioxidants, amino acids and other culture nutrients. Add to this the foreign proteins and DNA, and possibly micoplasmas and prions, from a host of viruses, bacteria, and animals upon which the medicines have been cultured: organ tissues and blood from monkeys, cow hearts, calf serum, chicken embryos and eggs, duck eggs, pig blood, sheep blood, horse blood, dog kidneys, rabbit brains, and aborted human foetuses; large foreign proteins from egg albumen, casein (milk protein); and gelatin from calves, cattle skins, de-mineralised cattle bones, or pork skin.

The manufacturer has run some short-term tests on various animals, as well as on healthy human adult volunteers. Curiously, the manufacturer hasn't bothered to evaluate whether or not the proposed vaccine has the potential to cause cancer, genetic mutations or infertility (see Chapter 3, 'Clinical tests'). The limited tests reveal that there were no adverse effects; and that antibodies to the infectious germs

were produced.

Now it's decision time. What do you do, particularly knowing that children as young as two months of age will be injected with them (in fact, for Hepatitis B shots, at birth)? And that a child will receive over 60 doses of combinations of these substances. Do you approve them, as our medical courtiers have done?

Or do you refuse, knowing that the US Vaccine Adverse Events Reporting System (VAERS), part of an agency established in 1986 to compensate American victims of vaccines, receives approximately 11,000–12,000 reports of adverse reactions to vaccines every year, of which about 15 per cent prove to be serious—meaning they require hospitalisation, are life-threatening, lead to permanent disability or are fatal—and that from 100 to 200 people die.[590] And that's only the tip of the iceberg, because the CDC and the FDA estimate that only 1 to 10 per cent of reactions are reported.[591,592]

And if you did approve the vaccines, how do you think you'd feel when you realised they were being linked to some of the following diseases and syndromes?: convulsions and seizures; sudden infant death syndrome (SIDS or cot death); developmental problems, attention deficit/hyperactivity disorder, and social violence; epilepsy, paralysis, cerebral palsy, mental retardation, autism and other neurological disorders; allergies, asthma, and anaphylaxis; leukaemia, lymphoma, brain tumours, and other cancers; Crohn's disease, multiple sclerosis, Guillain-Barré syndrome, amyotrophic lateral sclerosis (Lou Gerig's disease), systemic lupus erythematosus, Type I diabetes, and other autoimmune diseases.[23,589,593–596] And that's just to name a few.

How would you feel knowing you contributed to the 'approved vaccines'? You'd probably go into denial, which is a normal human defence mechanism to shocking news. So is anger. Perhaps you'd muddy the waters by setting up experiments to prove you were right all along. Maybe you'd even disparage or demote those who dared to break ranks. So imagine how doctors must feel, knowing that they may have done far more harm than good, particularly to their youngest patients.

On top of that, how would you deal with the knowledge that a child with a compromised immune system should never be vaccinated? And yet, according to the Medical Advisory Committee of the Immune Deficiency Foundation, in Towson, Maryland, most immune deficiencies cannot be diagnosed until a child is one year old.[597] The insanity of vaccinating children before they are one is obvious.

The charade, the subterfuge and the propaganda

The courtiers, of course, went far further than denial, or denigrating and demoting whistle-blowers and renegade scientists. They used every trick in the book. In summing up the charade, the subterfuge and the propaganda employed, Paul King and Gary Goldman, two American research scientists writing in *Medical Veritas*, a journal that seeks the truth in medical science, said it perfectly: 'The propaganda dispensed by Public health care and vaccine apologists is, at best, a weak attempt to rationalize the healthcare establishment's positions using all the tools of doublespeak or, as George Orwell called it in his book *1984*, "newspeak", to:

(a) mislead, (b) distort reality, (c) pretend to communicate, (d) make the bad seem good, (e) avoid and/or shift responsibility, (f) make the negative appear positive,

(g) create a false verbal map of the world, and (h) create dissonance between reality and what their narrative said or did not say.'[595]

And these researchers continue, detailing the subterfuge employed: 'Such propaganda often relies on half-truths and/or superficially logical, but foundationally flawed, phrasing. However, this propaganda is fundamentally flawed and based on pseudo-science or non-reviewable statistical studies of medical records, where, contrary to ethical science, the study design, data selection/rejection criteria, exact approach used to evaluate the data, and/or the original data itself are kept confidential making independent evaluation/verification of the published findings impossible.'[595]

But cancer registries don't lie. In the United States, for instance, the incidence of childhood cancer has continued to rise since the 1930s, as it has in other developed countries.[5965–606] Between 1935 and 1979, in the state of Connecticut, for instance, there was a three-fold increase in the incidence of cancer in the under-five-year-olds.[598] Other age groups fared little better. Particularly significant in the under-fives was a rise in the incidence of neuroblastoma, a tumour of the central nervous system. And neuroblastomas are linked to SV40 from polio vaccines.[607]

Meanwhile, in the state of Minnesota, cancer incidence in under-15-year-olds increased by 1 per cent each year from 1970 to 1989.[603] The greatest increase was in tumours of the central nervous system, which increased by 2.7 per cent each year. During the same time period, the national incidence of osteosarcoma (bone cancer), astrocytoma (a form of brain cancer), and rhabdomyosarcoma (soft tissue cancer) in the under-15s increased by 2 per cent per annum.[601] These cancers have also been linked to SV40 from polio vaccines.[560,567,568,608] The greatest rise was in children under three. And for children in their first year of life, the greatest increase was in cases of neuroblastoma and retinoblastoma (cancer of the eye's retina), both of which are linked to SV40.[607,609]

Tellingly, the abrupt rise in childhood leukaemia, cancers of the central nervous system, and infant neuroblastomas, began in the mid-1980s, soon after the MMR vaccine had been added to the medical armamentarium.[605,606]

In Australia—a nation that has played deputy dog to the United States in many things since World War II, including using the same FDA-approved medicines—the national incidence of childhood cancer during the 1980s was 34 per cent higher than in the United Kingdom.[610]

The medical courtiers have no answers to what has caused the rise in childhood cancer, but they contend that it's definitely not vaccines.

The incidence of autism shows the same rising trend. First identified in the 1940s, it has continued to rise ever since the advent of mass childhood vaccination programmes. During the 1960s, it was estimated that 1 in every 2,000 children had the condition.[611,612] By the mid-1970s it was 1 in 250 children.[613,614] Today, autism affects 1 in every 150 US children, and 1 in 160 Australian children.[615,616] If the less severe forms of autism, such as Asperger's Syndrome, are included in what is now called the Autistic Spectrum, then about 1 in 100 children are affected.

The medical courtiers have no answers to what causes autism. But they claim that it's definitely not vaccines.

Whistle-blowers

Despite the courtiers' propaganda, many people with autistic children, as well as many members of the medical profession, are unconvinced.[589,595,596,617] A case in point is that of Dr Jon Poling, a neurologist from Athens in the US state of Georgia, whose daughter Hannah developed regressive autism soon after receiving a series of vaccines in 2002 at the age of 18 months. Medical professionals eventually conceded

that vaccinations had caused Hannah's regressive autism. As a result, the US Department of Health and Human Services eventually paid compensation to the family. The message from the US Government was clear: for the first time it had conceded that children can develop regressive autism following vaccinations.

As Dr Jon Poling and his wife Terry wrote in the *New York Times* in April 2008, in response to misstatements made in a previous letter to the newspaper by Paul A. Offit, Chief of Infectious Diseases at the Children's Hospital of Philadelphia, Professor of Pediatrics at the University of Pennsylvania School of Medicine, and chief US propagandist for vaccinations:[619] 'Our daughter, Hannah, developed normally until receiving nine vaccines at once. She immediately developed a fever and encephalopathy, deteriorating into what was diagnosed, based on the Diagnostic and Statistical Manual of Mental Disorders, or D.S.M. IV, as autism.'[596]

In response to Offit's claim that a pre-existing medical condition contributed to Hannah Poling's autism[618]—and definitely not vaccines—the Polings continued: 'Dr Offit's assertion that "even five vaccines at once would not place an unusually high burden on a child's immune system" is theory and risky practice for a toddler's developing brain. No one knows if Hannah's mitochondrial dysfunction existed before receiving vaccines. Dr Offit's claim that Hannah had "already weakened cells" is unfounded.'[596]

Ever the vaccine propagandist, Dr Offit repeated his inaccurate statements in a May 2008 edition of *The New England Journal of Medicine*.[619] To alert medical professionals to the issues, and to correct Offit publicly for the second time, Dr Poling wrote a letter to the same journal, published three months later.[620]

Not once have the courtiers at the CDC, FDA, NIH, the American Medical Association, or the American Academy of Pediatrics thought to compare the data on the prevalence of autism, let alone any other medical condition, in vaccinated and non-vaccinated children.

Given that the American Academy of Pediatrics admits that 1 in 6 American children are diagnosed with a developmental and/or behavioural problem, it may well be that more than 20 per cent of American children have some vaccine-induced deficit.[621] No wonder the courtiers would prefer to muddy the waters than investigate the issues.

But evidence for harm is emerging. A recent Canadian study, for example, examined the prevalence of asthma in more than 11,500 children in Manitoba.[622] Those children whose first shot of the DPT vaccine had been delayed by at least two months had half the risk of developing asthma than those who had their first shot at the recommended two-months-of-age. And delaying all three DPT shots by more than two months reduced the risk of having life-long asthma by 60 per cent.

Conversely, some vaccines administered two months after a child is born seem to be more dangerous than if given at birth. J. Barthelow Classen, a former researcher at the US National Institutes of Health, and founder and CEO of Classen Immunotherapies in Baltimore, Maryland, found that vaccinating a baby with Hepatitis B shots at birth—supposedly for newborn 'prostitutes and intravenous drug users'—is far less likely to result in insulin-dependent diabetes than starting at least three or more weeks after birth.[623–625] And the Haemophilus influenza type b (Hib) vaccine seems to do the exactly the same.[626] Of course, the more shots a child receives, the greater the risk of developing juvenile diabetes.

Thus, when medical courtiers in New Zealand undertook an intensive campaign in 1988 to inoculate children at six weeks of age with the Hepatitis B vaccine, the incidence of Type I diabetes increased by 60 per cent during the following three years.[627] And Finland's incidence of juvenile diabetes increased 147 per cent in the under-five-year-olds after three new vaccines were introduced during the

1970s; and, during the 1980s, when the MMR and Hib vaccines were introduced, the incidence in the 5- to 9-year-olds increased by 40 per cent.[623]

Since 1985, sharp rises of early onset diabetes have also been recorded in the under-fives in both the United States and Britain—the incidence in Britain soaring by 11 per cent each year between 1985 and 1995.[628,629] And the rising incidence corresponded with the advent of the Hib and HBV (Hepatitis B virus) vaccines.

The courtiers had of course added another line to former US Secretary of Defense, Donald Rumsfeld's immortal lines on knowing and not knowing. Theirs is: 'There are some things we know we don't want to know.' But some physicians did get to know what the courtiers preferred they didn't know.

During the late 1960s, the impact of vaccines, and particularly the DPT vaccine, on Aboriginal health had prompted two prominent members of Australia's medical profession to break ranks. At the time, Archie Kalokerinos had just become Medical Superintendent of Collarenebri Hospital, in rural New South Wales, 840 kilometers (500 miles) north-west of Sydney. He was a firm supporter of vaccines. But he was appalled to find an extraordinarily high rate of infant mortality among Aboriginal babies.[630] He soon came to realise that many Aboriginal infants became ill after receiving routine vaccines. Some became extremely ill, and many died. In some Aboriginal communities every second baby died after being vaccinated. Hence the title of his book, *Every Second Child*.[631]

He noted that illness or death was more likely to occur in infants who were ill at the time of receiving a vaccine, or in infants who had recently been ill, or in those who were incubating an infection (and there is no way that physicians can clinically detect disease in its incubation period). But by giving babies an intramuscular or intravenous injection of a high dose of vitamin C, he could reverse the reaction and lives would be saved.

This wonderful news about preventing sudden infant death syndrome, or SIDS, was greeted with extreme hostility by Australia's medical courtiers. But as Kalokerinos explains, 'This forced me to look into the question of vaccination further, and the further I looked into it the more shocked I became. I found that the whole vaccine business was indeed a gigantic hoax. Most doctors are convinced that they are useful, but if you look at the proper statistics and study the instance of these diseases you will realise that this is not so.'[632]

Then in 1969 the Australasian College of Biomedical Science appointed Melbourne pathologist Glen Dettman to head a research team to investigate the claims made by Archie Kalokerinos, that children were dying because of subclinical scurvy (vitamin C deficiency) brought on by impoverished diets and infections, and by the added burden of vaccines. Indeed, viral infections alone have been shown to reduce vitamin C levels by 50 per cent.[633] Vaccines, Kalokerinos reasoned, were simply an affront to their already weakened immune systems.

Dettman was soon convinced of the veracity of Kalokerinos's findings, and joined forces with him, publishing articles on the myth that vaccines conquered various diseases, and trying to convince the courtiers of the dangers of vaccinating sick children.[25,634–637] But their efforts were all in vain, for the medical courtiers clung to the opposite belief: that sick children must be vaccinated to protect them.

Nevertheless, through their efforts the Aboriginal infant mortality rate in Kalokerino's health district dropped to zero.

Then, as chance would have it, a politician, one of Kalokerinos's ex-patients, asked him to investigate why the Aboriginal infant death rate in the Northern Territory had suddenly doubled in 1975,

and was set to double again by the following year. There was no obvious explanation. Then Kalokerinos was stuck by an epiphany: to improve Aboriginal health, the authorities had stepped up their vaccination campaign. In other words they were vaccinating sick children. He went off to investigate. What he witnessed horrified him. Health authorities had stormed into Aboriginal communities, rounding up children even though their mothers may have refused permission for their children to be vaccinated, even chasing fleeing children on foot or in Land Rovers and then forcibly vaccinating them. A few weeks later, the vaccination merry-go-round would begin again as the children were forcibly given booster shots. As Kalokerinos remarked, 'It is a wonder that any kid survived really, not that the death rate had just doubled. It is a wonder that anyone survived.'[632]

Years later, Kalokerinos would state about his experiences: 'Deliberate attempts have been made to allow (Aboriginal) infants under my care to die. The real authorities don't want these infants to live. The real intention on the part of the authorities is genocide.'[638]

Kalokerinos's appraisal of international organisations that promote vaccinations is no less savage: 'My final conclusion after forty years or more in this business is that the unofficial policy of the World Health Organisation and the unofficial policy of "Save the Children's Fund" and almost all those organisations is one of murder and genocide. They want to make it appear as if they are saving these kids, but in actual fact they don't. I am talking of those at the very top. Beneath that level is another level of doctors and health workers, like myself, who don't really understand what they are doing. But I cannot see any other possible explanation, it is murder and it is genocide.'[632]

Because of the overwhelming evidence for harm, many doctors who dare to think have broken ranks. Almost a century ago, Dr Walter R. Hadwen had done just that. He was the physician who had witnessed the devastating effects of the smallpox vaccine upon Britain's population during the late 19th century; the man who had persuaded the townsfolk of Gloucester not to get vaccinated against smallpox during the epidemic of 1895–1896, who watched as the whole child population, only 4 per cent of which was vaccinated, passed through the epidemic unscathed; who recorded that of the 2,000 people who did contract smallpox, two-thirds of them had indeed been vaccinated; and who had identified the culprit—leaking sewers near drinking water pipes.[639]

After winning a legal case of alleged manslaughter brought by the medical courtiers, and before a packed audience in Queen's Hall, London, on Friday 6 February 1925 Hadwen declared: 'I once believed in Jenner; I once believed in Pasteur. I believed in vaccination. I believed in vivisection. But I changed my views as the result of hard thinking.'[640]

The importance of infections

If doctors had done the hard thinking, then they might have come to understand that viral and bacterial infections, even parasitic infections, in childhood are essential for human health.

For example, in *Immunology Today* in an article entitled 'Give us this day our daily germs', two British medical microbiologists, Professor Graham Rook and Laura Brunet, have suggested that our use of vaccinations and antibiotics, our fear of germs, and our obsession with hygiene, are depriving our children of the very germs that play a role in the correct maturation of their immune systems.[641]

In other words, we are born with the hardware of an immune system that is primed to deal constantly with environmental germs, but its correct functioning depends on the information fed into its software after birth. But because the fine cytokine balance and fine-tuning of T-cell regulation have never been kick-started by persistent exposure to viruses, bacteria and parasites, our immune systems have

never learned to switch off. This 'hygiene hypothesis of atopy' (allergy), the two medical microbiologists contend, may explain the soaring prevalence of asthma, allergies and autoimmune diseases throughout the developed world.

Though the importance of acute illnesses for human health, and their treatment, will be dealt with in Volume 2, suffice it to say here that there is overwhelming evidence that children from farming communities, where they are exposed to farmyard germs; from Rudolf Steiner schools, where treatment with vaccines, antibiotics, and antipyretics (drugs that reduce fever) is discouraged; from large families, and those from small families who go to playschool during their first year of life, and hence are exposed to numerous germs from other children; that all these children have far less eczema, hayfever and asthma, than other children.[642–645]

And there is clear evidence that girls who catch mumps early in childhood are far less likely to develop ovarian cancer later in life.[646–649]

Measles alone, according to many studies, prevents or reduces the tendency to develop skin allergies and sensitivity to house dust mites, hayfever and asthma, malaria in tropical climes, autoimmune diseases, various degenerative diseases of the bones, juvenile rheumatoid arthritis (Still's disease), various tumours, psoriasis, Parkinson's disease, and even epilepsy.[650–666]

Children exposed to the tuberculosis bacillus or Hepatitis A virus earlier in their lives also have less asthma and allergies than other children.[667,668] The same applies to those who are infected with parasitic worms (helminths: tape worm, fluke worms and nematodes)[669] British researchers suspect that hookworms, caught only through walking barefoot on contaminated soil, may help people overcome Crohn's disease (an intestinal autoimmune disease).[670] Indeed, Japanese researchers have found that intractable epileptic seizures disappear within two weeks after a child has had not only measles but also mumps, rotavirus colitis, or exanthema subitum (roseola rash).[671]

In other words, being healthy, but nevertheless being exposed to unhygienic living conditions early in life, is evidently a recipe for health. And the earlier the exposure to germs, the better.

Moreover, catching a cold, or the flu, or some other viral infections, may prevent cancer and even destroy cancer cells.[672–6]

According to a group of researchers at the University of Newcastle, in New South Wales, Australia, a wild-type, common cold-producing virus, Coxsackievirus A21, has powerful anti-tumour activity against malignant melanomas, multiple myeloma, prostate cancer and breast cancer.[672–676] And the ECHO virus (an intestinal infection that although potentially life threatening in babies, usually produces minimal symptoms in adults) apparently targets and destroys human ovarian cancer cells.[677]

Groups of American and Canadian researchers have recently found that infection with reovirus (which may elicit no symptoms at all) also destroys cancer cells, in particular breast cancer and prostate cancer cells.[678–681] Scottish and American researchers found that the same applies to the herpes simplex virus.[682,683] And according to researchers in the US and Japan, the flu virus apparently triggers our immune systems to target and kill off cancerous cells;[684–691] as does the human respiratory syncytial virus, and the human cytomegalavirus.[692,693]

Hidden footnotes to the vaccination saga

This investigation of vaccines would not be complete without mention of two diseases, the origins of which still lurk in the shadows: AIDS and the Gulf War Syndrome.

AIDS

Seven years and a day after the WHO proclaimed that it had wiped smallpox from the face of the Earth, *The Times* of London ran a front page story stating that the epidemic of AIDS sweeping through Central Africa may have been triggered by the WHO's final assault on the smallpox virus.[694] Using a 'modified version' of the vaccine, the WHO had inoculated nearly 100 million people in seven Central Africa states, as well as in Brazil. During the 13-year campaign, which ended in 1980, 14,000 Haitians working for the UN in Zaire had also been inoculated. And in which countries did the AIDS epidemic suddenly appear, as if out of nowhere, in about 1978? In the very countries the WHO had just vaccinated.

As the WHO adviser who tipped off *The Times* stated: 'I thought it was just a coincidence until we studied the latest findings about the reactions which can be caused by Vaccinia [the cowpox virus]. Now I believe the smallpox vaccine theory is the explanation for the explosion of AIDS.'

Furthermore, *The Times* reported that according to the Walter Reed Army Medical Center in Washington, DC, a previously healthy 19-year-old US Army recruit, who had received the vaccine during routine 'immunisations' against possible biological warfare agents, had also contracted AIDS, and soon thereafter had died.

But the American media remained stony silent, proving that politics, not newsworthiness, was at play.

To allay fears and dumb-down the public, the medical courtiers had contrived the following racist fantasy: HIV/AIDS had been transmitted through a bite from a jungle-dwelling African green monkey to some person in Central Africa.[695] That person's body had transformed the green monkey simian immunodeficiency virus (SIV)—which incidentally was also present in the polio vaccine—into human immunodeficiency virus (HIV), and then that person had transmitted the new virus through sexual intercourse (homosexual or heterosexual) to another person, and so on. And because African males are a promiscuous lot, the infection had rapidly spread through rural communities into city dwellers, and then exploded into such plague proportions that to date 15 million Africans have died of AIDS and about 25 million are infected with HIV.[696]

Some courtiers had come up with variations on the mode of transmission. They included such racist hypotheses as contracting the disease through sex with a green monkey; parents allowing their children to play with dead monkeys as toys, and hence becoming infected; or adults inoculating themselves with monkey blood around their pubic region and thighs to induce sexual fervour.[697,698]

During the 1990s, the courtiers decided to change the hypothesised animal vector from a green monkey to a chimpanzee. But in their scurry to announce such fantasies, the courtiers never thought to investigate whether AIDS was rife amongst the very people who hunt monkeys and chimpanzees, the Pygmies. In fact, AIDS is notable for its absence amongst these people.[699] Moreover, the virus thought to be the culprit, SIV, was found to have no effect whatsoever on green monkeys, nor did it genetically resemble HIV.[700,701]

The courtiers couldn't even agree on when AIDS first arose. Some thought it was during the 1930s, some claimed it emerged during the late 1950s, and Jonas Salk contended that it arose in Africa 900 years ago—which begs the question: given that African peoples have always travelled the world, and that at least 100 million had been forcibly transported as slaves to the New World, why wasn't the rest of the world affected with AIDS long ago?

The courtiers have an even more fantastical explanation for how AIDS suddenly erupted throughout gay communities in the United States. The story goes like this: Some of the 14,000 Haitian men who had been working in Africa when the disease erupted there had carried the virus back to their home country, and then transmitted it to American homosexual and bisexual men who frequented the island for sex. The gay American holiday makers had, in turn, taken the virus back to the United States. And because they, too, are a promiscuous lot, had rapidly spread it amongst gay communities.

Come hell or high water, the courtiers could not afford to have the public discover that AIDS in the US had been caused by a vaccine trial approved by the National Institutes of Health. The trial of a new Hepatitis B vaccine had been conducted by Professor Wolf Szmuness, an epidemiologist from the Columbia University School of Public Health and New York City's Blood Center. In 1978, he had selected over 1,000 healthy, young, highly promiscuous, homosexual and bisexual men from New York City, and had inoculated them with the vaccine. A year later he had vaccinated another 3,400 gay men from New York City. Then in 1980, he selected similar cohorts of thousands of gay men from the cities of San Francisco, Los Angeles, Chicago, St Louis, and Denver and inoculated them.

The first hint that something was amiss had become apparent by March 1981. Amongst young gay men in New York City there had been at least eight cases of a rare form of cancer, Kaposi's sarcoma, and it was far more aggressive than was typical.[702] About the same time, researchers from the CDC noted that cases of both Kaposi's sarcoma and a rare lung infection, *Pneumocystis carinii* pneumonia, were coming to light amongst the gay communities of New York and California.[703,704] What researchers were looking at were the first cases of people with AIDS.

CDC investigators noted that 6 of the first 10 cases of AIDS in San Francisco were part of the 6,800 gay men who participated in Szmuness's Hepatitis B trial.[705] Taking a representative sample of the trial cohort, they found that HIV infection had risen from just over 4 per cent of the group during the period 1978 to 1980, to 67 per cent in 1984, and 73 per cent by 1985. Clearly most had been infected by the hepatitis vaccine. Of the 31 members of the representative sample, 10 had developed AIDS or AIDS-related conditions by 1981. And of the whole cohort of human experimental guinea pigs, 166 of them had AIDS by 1984. By 1985, the number had increased to 262.

As for the 4,400 gay men who participated in Szmuness's Hepatitis B trial in New York City, five years later 48 per cent of them were infected with HIV.[706]

Meanwhile, other CDC investigators had noted outbreaks of AIDS in the same cities in which Szmuness trialled the Hepatitis B vaccine. In fact, the CDC acknowledged that those cities accounted for 80 per cent of all AIDS cases in the United States.[707] In New York City and San Francisco alone, the number of AIDS cases per million was 10 times higher than that for the entire nation.

This marked the beginning of America's AIDS epidemic. Seventeen years after the trials in New York and San Francisco, 57 per cent of the gay men who had received the Hepatitis B shots were dead: 95 per cent of them from AIDS.[708]

Contrary to the courtiers' racist propaganda that AIDS came out of Africa,[709] it is clear that AIDS emerged from American laboratories that manufactured the lethal Hepatitis B vaccine, and from

laboratories that supplied the smallpox vaccine to the WHO Smallpox Eradication Programme in Central Africa.

Was HIV deliberately or accidentally made? Many Black Americans believe it's the former. In a survey conducted in 1990, 35 per cent of Black American church members believed AIDS is a form of genocide; 1 in 10 believed HIV was deliberately created to infect black people, and an additional 2 out of 10 thought it might be so.[710]

Black Americans certainly had good reason to suspect sinister motives on the part of the US Government. Since the 1930s, the US Government and its agencies had conducted many experiments on its unsuspecting citizens. For example, the US Public Health Service had conducted a 40-year experiment on poor, illiterate, syphilis-infected, black sharecroppers in Tuskagee, Alabama.[711] Government researchers had withheld medication for, and knowledge about, the men's medical condition, all in the name of investigating how syphilis impacted on the men and their families.

Black Americans may have also noted that the prevalence of AIDS in the United States, which during the 1980s was a disease primarily of white, gay men, had by 2008 become a disease primarily of minority groups, the virus's prevalence being particularly high in women and children of Black, Hispanic , and Native American ethnicity.[710] According to two British investigative journalists, these ethnic groups have twice the rate of HIV infections compared with American Whites, and account for 80 per cent of children and 90 per cent of infants with AIDs.[712]

Moreover, the number of sub-Saharan Africans living with HIV/AIDS accounts for two-thirds of all cases in the world today.[713] As a New York Department of Health official had forewarned the *New York Times* in April 1992, 'AIDs in future generations may be primarily a disease of black people.'[710]

Some researchers suspect sinister motives lie behind the creation of the virus. In their books, *The Extremely Unfortunate Skull Valley Incident*, and *AIDS: The Crime Beyond Belief*, Donald W. Scott—editor of *The Journal of Degenerative Diseases*, co-founder of the Common Cause Medical Research Foundation in Ontario, Canada, and Adjunct Professor at the Institute for Molecular Medicine at Huntington Beach, California—and his son William have documented many of the government dealings that led up to the release of the HIV.[714,715] Similarly, British investigative journalists Robert Harris and Jeremy Paxman have documented in their book, *A Higher Form of Killing*, the international story of the development of chemical and biological warfare, and its ties to medical research; and have looked particularly at the US Army's objective of developing ethnic weapons.[712] Alan Cantwell, a New York dermatologist who took a particular interest in the origins of HIV, has written several books on the subject.[716–719] Investigative journalist Harry V. Martin has also revealed evidence that the AIDS virus was tested on expendable people.[720] And Leonard G. Horowitz, an American public health authority, explains in his book, *Emerging Viruses: AIDS and Ebola*, how a small group of virologists working for major military-medical contractors, and under the auspices of the US National Cancer Institute and the WHO, had for years conducted dangerous experiments with viruses that ravage the human immune system.[721]

As long ago as 1966, scientists as esteemed as Australia's Frank Macfarlane Burnet had warned molecular biologists against tampering with life, that there were limits beyond which there were grave dangers.[722] But no one took any notice of such warnings, the reason being that politics, money and egos were at play. Author and editor Jonathon Vankin summed up the situation perfectly: 'Public comprehension of science is scant, depending entirely on third-party interpreters, "experts" who have agendas of their own. Not only is general scientific knowledge therefore minimal, more importantly few people understand how science works. We think we're getting objective truth when what we're seeing is a political acerbically personal process involving billions of dollars, reputations and egos, and belief systems

that censor large slices of fact and theory.'[723]

Thus politics, money and egos, together with a belief system that life is a toy to be played with, was the reason why Dr Donald MacArthur, US Deputy Director of Defense Research and Engineering, requested in 1968 that a US Congress Appropriations Committee fund a research programme into engineering new micro-organisms … and the funds were granted.[724] It is also why a committee at the WHO recommended the following: 'An attempt should be made to ascertain whether viruses can in fact exert selective effects on immune function, e.g. by depressing 7S versus 19S antibody, or by affecting T cell function as opposed to B cell function. The possibility should also be looked into that the immune response to the virus may itself be impaired if the infecting virus damages more or less selectively the cells responding to the viral antigens.'[725] In other words, the World Health Organisation recommended that an AIDS-causing virus be created and tested.

By playing God, the science of molecular biology was certainly toying with potentially catastrophic dangers. Thus, during the 1980s, when scientists began to examine the genetic structure of HIV, they discovered that it was nothing like a monkey virus. Both Robert Strecker, a Californian pathologist, and J. Grote, an AIDS researcher in London stated that the virus appeared to be a recombinant virus, a cross between a sheep and a bovine virus called *bovine visna* virus.[726,727] In a letter to the *Journal of the Royal Society of Medicine*, Dr Grote wrote that the bovine visna virus was a known contaminant of foetal calf serum.[727] Foetal calf serum just happens to be one of the cell cultures used in the manufacture of vaccines. So perhaps the virus was accidentally created through contaminated Hepatitis B vaccines and smallpox vaccines.

Incidentally, Robert Strecker tried to alert the medical profession to his findings, but not one medical journal would publish his evidence. Undeterred, he created a video, *The Strecker Memorandum*, to at least alert the public.[726]

Whether deliberate or accidental, the courtiers could never admit that HIV had been spread by vaccines. To do so would have been to hammer the greatest nail into the coffin of vaccination campaigns.

The Gulf War Syndrome

Nor could the courtiers admit that Gulf War syndrome was caused by an experimental vaccine. In his book, *Vaccine A: The Covert Government Experiment That's Killing Our Soldiers*, investigative journalist Gary Matsumoto unearthed shocking evidence that the US Department of Defense and the British Ministry of Defence approved an experimental vaccine to protect armed forces personnel against anthrax.[728] The justification for using the vaccine was that Saddam Hussein might deploy chemical weapons against coalition forces after the UN decision had been made to drive Iraqi forces out of Kuwait, which Iraq had invaded and annexed on August 2, 1990.

Time was of the essence, and the licensed vaccines took up to eight months to produce 'immunity' to anthrax.[729] The defence establishments in the United States and Britain needed a vaccine that worked quickly. So US military doctors added an unlicensed adjuvant (a chemical that stimulates the body to produce antibodies) to the vaccine. This adjuvant was squalene, an oil-based substance found in olive oil. When squalene is injected into the bloodstream it had been shown in peer-reviewed scientific literature to be capable of causing incurable, if not fatal, diseases.[730]

By the time the United Nations sanctioned a coalition of military forces from 34 nations to drive Hussein's troops out of Kuwait, 41 per cent of US combat soldiers and 57–75 per cent of UK combat soldiers had been inoculated against anthrax, though not all with the experimental vaccine.[731]

About a year after hostilities ended, reports began to emerge about a strange malady that afflicted many military veterans from America, Britain, Australia and Canada.[732] Symptoms included chronic fatigue, dizziness and headaches, loss of balance, short-term memory loss, muscle and joint pains, shortness of breath, skin rashes, diarrhoea, dyspepsia and indigestion, weight loss, hair loss, sore gums, fibromyalgia, terminal tumours, and a host of autoimmune diseases including multiple sclerosis, systemic lupus erythematosus, amyotrophic lateral sclerosis (Lou Gerig's disease), Guillain-Barré syndrome, inflammatory arthritis, endocarditis, thyroiditis, polyarteritis nodosa, and collagen vascular disease.

The victorious coalition forces suffered only 190 combat deaths and, sadly, 379 deaths by accidents or 'friendly' fire; the US suffered only 148 combat deaths, though a quarter of these were by 'friendly' fire.[733] But however successful the armed conflict had been in its strategic aims and limited loss of life, the aftermath was horrendous. Of the 700,000 American servicemen and women who participated in the war, over a third of them (183,000) have been declared permanently disabled by the US Department of Veteran Affairs.[731]

Moreover, according to a US survey, Gulf War veterans are at least twice as likely to have children with birth defects compared to veterans who were never deployed to the Gulf, and who therefore never received the anthrax vaccine.[734] Of course, the courtiers could never accept that a vaccine caused such human misery. They blamed the syndrome on denatured uranium, on a US Army engineering battalion inadvertently releasing a plume of nerve agent from an Iraqi chemical munitions stockpile at Khamisiyah, on the extensive use of pesticides by English-speaking troops, and even on the stresses of war.[735]

The courtiers were clearly clutching at straws, for they'd overlooked the fact that many army personnel who had never departed their home shores were also suffering from the syndrome. Moreover, soldiers from most countries in the anti-Iraq coalition did not suffer from the malady, nor did Arab soldiers or civilians on either side of the conflict, nor did any journalist, 'embedded' or not, get sick.[732]

The generals and officials at the US Department of Defense even hid from Congress and public scrutiny the fact that by continuing to use the vaccine, long after the First Gulf War, over 20,000 military personnel between 1998 and 2000 had been hospitalised immediately after receiving the squalene-containing anthrax vaccine.[736] According to *The Newport Daily News* in Virginia, military officials misled Congress and the public by claiming that fewer than 100 people had been hospitalised or become seriously ill after receiving the shot; and they failed to report three cases of amyotrophic lateral sclerosis.[736]

Though researchers at Tulane University had repeatedly identified antibodies to squalene in military personnel who were suffering from symptoms of Gulf War syndrome,[737] and even though those same researchers had later found that 47 per cent of military recipients inoculated after 1997 with the anthrax vaccine had antibodies to squalene—squalene is still present in the anthrax vaccine, as well as many influenza vaccines—and that recipients of other vaccines had no antibodies whatsoever to squalene,[738] the US Department of Defense, in 2009, proudly published a study that claimed it had found no relationship between squalene antibodies and symptoms. The Defense Department researchers concluded: 'We found no association between squalene antibody and chronic multisystem disease. The etiology of Gulf War syndrome remains unknown, but should not include squalene antibody status.'[739] This is an abstruse way of saying, 'Let's drown the subject.'

Perhaps the wisest words, and most prescient, on the dangers of vaccines had been uttered by Nicholas Wade, a British-born science reporter, who is now an author of several books, and writes for the 'Science Times' section of the *New York Times*. In an article entitled 'The boat that never rocks', published

in *Science* magazine in 1972, he wrote: 'There can be few graver opportunities for man-made disaster than the mass immunization campaigns that are now routine in many countries.'[740]

The 740 references hereunder go over 53 pages…

REFERENCES TO CHAPTER 5

CHAPTER 5—To Vaccinate or Not to Vaccinate

1. *Rogers v. Whitaker*, the High Court of Australia 1992, 175 CLR 479; 19th November, 1992.

2. Posfay-Barbe KM, Heininger U, Aebi C, Desgrandchamps D, Vaudaux B, and Siegrist C-A. "How do physicians immunize their own children? Differences between pediatricians and nonpediatricians." *Pediatrics*, 2005; 116 (5): 623–633.

3. Katz-Sidlow RJ, and Sidlow R. "A look at the pediatrician as parent: experiences with the introduction of Varicella vaccine." *Clinical Pediatrics*, 2003; 42 (7): 635–640.

4. Diekema DS, and the Committee of Immunization of Children. "Responding to parental refusals of immunization of children." *Pediatrics*, 2005; 115 (5): 1428–1431.

5. Dionne M, Boulianne N, Duval B, Lavoie F, Laflamme N, Carsley J, Valiquette L, Gagnon S, Rochette L, and De Serres G. "Lack of conviction about vaccination in certain Quebec vaccinators." *Canadian Journal of Public Health*, 2000; 92 (2): 100–104.

6. Kinnersley P. "Attitudes of general practitioners towards their vaccination against hepatitis B." *British Medical Journal*, 1990; 300 (6719): 238.

7. Gerety RJ. "Hepatitis B transmission between dental and medical workers and patients." *Annals of Internal Medicine*, 1981; 95 (2): 229–231.

8. Preblud SR, and Hinman AR. "Rubella vaccination of hospital employees." *Journal of the American Medical Association*, 1981; 245 (7): 736–737.

9. Orenstein WA, Heseltine PN, LeGagnoux SJ and Portnoy B. "Rubella vaccine and susceptible hospital employees. Poor physician participation." *Journal of the American Medical Association*, 1981; 245 (7): 711–713.

10. Bishburg E, Shah M, and Mathis AS. "Influenza vaccination among residents in a teaching hospital." *Infection Control and Hospital Epidemiology*, 2008; 29: 89–91.

11. Saluja I, Theakston KD, and Kaczorowski J. "Influenza vaccination rate among emergency department

personnel: a survey of four teaching hospitals." *Canadian Journal of Emergency Medical Care*, 2005; 7 (1): 17–21.

12. Ohrt CK, and McKinney WP. "Achieving compliance with influenza immunization of medical house staff and students. A randomized controlled trial." *Journal of the American Medical Association*, 1992; 267 (10): 1377–1386.

13. Dole L. *The Blood Poisoners*, Gateway Book Company, Croydon, Surrey, UK, 1965; chapter 2.

14. Kenneth R, and Wear A, *The Medical Revolution of the Seventeenth Century*, Cambridge University Press, Cambridge, UK, 1989; p. 178. Citing K. Dewhurt, *Dr Thomas Sydenham (1624–1689): His Life and Original Writings*, University of California Press, Berkeley, California, USA, 1966; p. 163.

15. Wohl AS. *Endangered Lives: Public Health in Victorian Britain*, JM Dent and Sons Ltd, London, UK, 1983; p. 133.

16. Editorial: *The Lancet*, July 15, 1871.

17. Hadwen, W.S. "Sanitation v. vaccination–the origin of smallpox." *Truth*, January 17, 1923.

18. Wallace WR. *The Wonderful Century: It's Successes and Its Failures*, Swan Sonnenschein and Co, London, UK, 1898; In chapter entitled "Vaccination a Delusion."

19. Wallace WR. *To Members of Parliament and Others: Forty-five Years of Registration Statistics, Proving Vaccination to be Both Useless and Dangerous (S374: 1885)*. Reprinted in Wallace WR, ibid reference 18.

20. Ruata C. "Vaccination in Italy." *New York Medical Journal*, July 22, 1899. Cited in Dr JW Hodge, *The Vaccination Superstition*, Niagara Falls, NY, USA, 1901 (Read before the Western New York Homeopathic Medical Society in Buffalo) April 11, 1902; pp. 6–7.

21. Briggs JT. *Leicester: Sanitation versus Vaccination*. Anti-Vaccination League, London, UK, 1912; chapter 103.

22. Slotten RA. *The Heretic in Darwin's Court: The Life of Alfred Russel Wallace*, Columbia University Press, New York, USA, 2004; pp. 422–436.

23. Scheibner V. *Vaccination: 100 Years of Orthodox Research shows that Vaccines Represent a Medical Assault on the Immune System*, published by Viera Scheibner, Blackheath, NSW, Australia, 1993; pp. 205–223.

24. Report of the Philippines Health Service by Dr V. de Jesus, 1920. Cited in Koch WF. *Survival Factor in Neoplastic and Viral Diseases, An Introduction to Carbonyl and Free Radical Therapy*, Natural Immunity Series, 1961; chapter 2.

    [Online, accessed 7th September, 2009]. URL:http://www.williamkoch.com

25. Kalokerinos A, and Dettman G. "Viral vaccines vital or vulnerable." *The Australasian Nurses Journal*, 1980; 9 (9): 27–32.

26. "Smallpox in vaccinated and re-vaccinated Japan." *The Vaccination Inquirer*, May 1, 1908; 30: 28. "Smallpox in Japan", ibid., July 1, 1908; 30: 62. And "The argument from Japan." ibid., October 1, 1909, 31: 141–143.

27. Hodge JW. "The failure of vaccination to protect from smallpox in re-vaccinated Japan." *The Twentieth Century Magazine*, 1910; 2 (12): 508–522.

28. Pitcairn J. "The fallacy of vaccination." *Ladies Home Journal*, May, 1910.

29. McCormick WJ. "The changing incidence and mortality of infectious disease in relation to changed trends in nutrition." *Medical Record*, Medical Journal and Record Publishing Company Inc, September, 1947. Reprinted by the Lee Foundation for Nutritional Research, Milwaukee, Wisconsin, USA; reprint no. 5a.

30. Dubos R. *Mirage of Health: Utopias, Progress and Biological Change*, Rutgers University Press, Brunswick, New Jersey, USA, 1959.

31. Illich I. *Limits to Medicine: Medical Nemesis–The Expropriation of Health*, Marion Boyars, London, UK, 1976.

32. McKeown T. *The Role of Medicine: Dream, Mirage or Nemesis?* Nuffield Provincial Hospital Trust, London, UK, 1976.

33. McKinlay JB, and McKinlay SM. "The questionable contribution of medical measures to the decline of mortality in the United States in the twentieth century." *Milbank Memorial Fund Quarterly*, Summer, 1977; 55 (3): 405–428.

34. Obomsawin R. *Universal Immunization: Medical Miracle or Masterful Mirage*, Health Action Network Society, Burnaby, British Columbia, Canada, 1996; Section 1: "WHO Smallpox Eradication Success Re-Considered."

35. Baratosy P. *Vaccination: It's Your Informed Choice*, published by Peter Baratosy, Adelaide, South Australia, Australia, 2004; pp. 12–13.

36. McBean E. *The Poisoned Needle: Suppressed Facts about Vaccination*. Health Research Books, Pomeroy, California, USA, 1959; Chapter IV: "The history of vaccinations".

37. Galazka A, and Tomaszunas-Blaszczyk J. "Why do adults contract diphtheria." *Eurosurveillance*, 1997; 2 (8): 60–63.

38. Winslow CEA. "Who killed Cock Robin?" *American Journal of Public Health*, 1944; 34: 658–659.

39. McBean E. Ibid reference 36, Chapter II: "Smallpox Declined Before Vaccination was Enforced."

40. Biggs JT. *Leicester: Sanitation versus Vaccination*, Part 13, Chapter 107: "London Evidence Against Anti-Toxin", National Anti-Vaccination League, London, UK, 1912.

41. Loat L. *The Truth about Vaccination and Immunization*. Health For All Publishing Company, London, England, UK, 1951; Part II.

42. Kater MH. *Doctors under Hitler*, University of North Carolina Press, Chapel Hill, North Carolina, USA, 2000; pp. 40–41.

43. Stowman K. "Diphtheria rebounds." *Epidemiological Information Bulletin*, UN Refugee and Relief Agency, Health Division, Washington, DC, USA, 15th February, 1945; 1: 157–168.

44. Crowley, T. Letter published in the *South Yorkshire Times*, March 11th, 1938. In: Beddow-Bayly M. *The "Schick" Inoculation for Immunisation Against Diphtheria: An Exposure of its Dangers and Fallacies*. Second Edition, The National Anti-Vaccination League, London, UK, 1939. Section 5: "Shick Immunisation," No. 18.

45. Dole L. *The Blood Poisoners*, Gateway Book Company, Croydon, Surrey, UK; 1965.

46. Editorial: "Diphtheria incidence and trends in relation to artificial immunization, with some comparative data for scarlet fever." *Public Health Reports*, 15th February, 1946; 61 (7): 203–237.

47. Stowan K. "The epidemic outlook in Europe." *Epidemiological Information Bulletin*, UN Refugee and Relief Agency, Health Division, Washington, DC, USA, February 15, 1945; 1: 101–111.

48. Liebow AA, and Bumstead JH. "Cutaneous and other aspects of diphtheria." In: Lieutenant General LD Heaton, Colonel JB Coates Jr., and WP Havens Jr. (editors), *Internal Medicine in World War II*, Volume II, Infectious Diseases; Office of the Surgeon General, Department of the Army, Washington, DC, USA, 1963; pp. 275–279.

49. Mendelsohn R. "The truth about immunization." *The People's Doctor*, April, 1978; p. 1.

50. Galazka AM, and Robertson SE. "Diphtheria: patterns in the developing world and the industrialized world." *European Journal of Epidemiology*, 1995; 11 (1): 107–117.

51. Edsall G. *Some Unsolved Problems in Diphtheria*, World Health Organisation, WHO/BAG/75.2, July 4, 1975.

52. Schneerson R. "Similarities between the pathogenesis of and immunity to diphtheria and pertussis: the complex nature of serum antitoxin-induced immunity to these two diseases." *Clinical Infectious Diseases*, 1999; 28: 136–139.

53. Hedrich A. *Epidemic Studies: The Monthly Variation of Measles Susceptibles in Baltimore, Maryland from 1901 to 1928*. Thesis, John Hopkins University, Baltimore, Maryland, USA, 1933.

54. Vitek CR, and Wharton M. "Diphtheria in the former Soviet Union: reemergence of a pandemic epidemic." *Emerging Infectious Diseases*, 1998; 4 (4): 539–550.

55. Robbins JB, Schneerson R, Trolifors B. "Pertussis in developed countries." *The Lancet*, 2002; 360 (9334): 657–658.

56. Editorial: "Diphtheria in the 1990s–do we have all the answers?" *Eurosurveillance*, 1997; 2 (8): Article 1.

57. Vitek CR, Brisgalov SP, Bragina VY, Zhilyakov AM, Bisgard KM, Brennan M, Kravtsova ON, Lushniak BD, Lyerla R, Markina SS, and Strebel PM. "Epidemiology of epidemic diphtheria in three regions, Russia, 1994–1996." *European Journal of Epidemiology*, 1999; 15 (1): 75–83.

58. Holt LE Jr., and McIntosh R. *Molt's Diseases of Infancy and Childhood*, 11th edition, D. Appleton-Century, New York, USA, 1940; p. 1083.

59. Pruitt BA, and Pruitt JH. "History of trauma care." In: ME Moore, DV Feliciano, and KL Mattox (editors). *Trauma*, Edition 5, McGraw-Hill Professional, New York, USA, 2003, pp. 13–16.

60. Boyd J. "Tetanus in Two World Wars." In: "Section of Epidemiology and Preventive Medicine", *Proceedings of the Royal Society of Medicine*, 1958; 52 (2): 109–110.

61. Murray CK, Hinkle MK, and Yun HC. "History of infections associated with combat-related injuries." *The Journal of Trauma*, 2008; 64 (3): S221–S231.

62. Bollet AJ. "The major infectious epidemic diseases of Civil War soldiers." *Infectious Disease Clinics of North America*, 2004; 18 (2): 293–309.

63. Noon G. "The treatment of casualties in the Great War." In: P Griffith (editor), *British Fighting Methods in the Great War*. Routledge, New York, USA, 1998; p. 95–97.

64. Furste WA. "A golden opportunity." *The Journal of Trauma*, 1998; 44 (6): 1110–1112.

65. Adams EB, Laurence DR, Smith JWG. *Tetanus*. Blackwell Scientific, Oxford, UK, 1969; p. 745.

66. MacLennan JD. "Anaerobic infections of war wounds in the Middle East." *The Lancet*, 1943; 2: 63–66, 94–99, 123–126.

67. Wahdan H. "Causes of the antimicrobial activity of honey." *Infection*, 1998; 26 (1): 26–31.

68. Long AP, and Sartwell PE, "Tetanus in the U.S. Army in World War II. *Bulletin of the US Army Medical Department*, 1947; 7: 371–385.

69. Glen F. "Tetanus–a preventable disease: including an experience with civilian casualties in the Battle for Manila (1945)." *Annals of Surgery*, 1946; 124 (6): 1030–1040.

70. Noon D. Ibid reference 63, p. 96.

71. Conybeare ET. "Tetanus in the civilian population of England and Wales." In: "Section of Epidemiology and Preventive Medicine", *Proceedings of the Royal Society of Medicine*, 1958; 52 (2): 112–114.

72. Fraser DW. "Tetanus in the United States, 1900–1969: analysis by cohorts." *American Journal of Epidemiology*, 1972; 96 (4): 306–312.

73. Boyd JSK. "Tetanus in the African and European theatres of war, 1939–1945." *The Lancet*, 1946; 1: 113–119.

74. Hall WW. "The U.S. Navy's war record with tetanus toxoid." *Annals of Internal Medicine*, 1948; 28 (2): 298–308.

75. Boyer JK, Corre-Hurst L, Sapin-Jalouste H, and Tissier M. "Le tetanos en milieu urbain: conditions d'apparition-deduction a prophylactiques." [In French], [Tetanus in urban conditions: conditions under which it appears and preventive measures.] *La Presse Medicale*, 1953; 61 (34): 701–703.

76. Hedrick EC. "Tetanus: two cases in immunized persons." *California Medicine*, 1953; 79 (1): 49–50.

77. Long AP. "Immunization to tetanus." *Industrial Medicine and Surgery*, 1954; 23 (6): 275–277.

78. Moss GW, Waters GG, and Brown MH. "The efficacy of tetanus toxoid." *Canadian Journal of Public Health*, 1955; 46 (4): 142–147.

79. Christensen NA, And Thurber DL. "Clinical experience with tetanus: 91 cases." *Proceedings of the Staff Meetings, Mayo Clinic*; 1957; 32 (7): 146–158.

80. Edsall G. "Specific prophylaxis of tetanus." *Journal of the American Medical Association*, 1959; 171: 417–427.

81. Peterson HI. "A case of tetanus in spite of active toxoid prophylaxis." *Acta Chirurgia Scandinavica*, 1965; 129: 235–237.

82. National Communicable Disease Center. *Tetanus Surveillance Report No. 1*, Atlanta, Georgia, USA, Bureau of Disease Prevention and Environmental Control, United States Public Health Service, February 1, 1968.

83. Peebles TC, Levine L, Eldred MC, and Edsall G. "Tetanus-toxoid emergency boosters: a reappraisal." *The New England Journal of Medicine*, 1969; 280 (11): 575–581.

84. Goulon M, Girard O, Grosbuis S, Desormeau JP, and Capponi MF. "Les anticorps antitetaniques: titrage avant seroanatoxinotherapie chez 64 tetanique." [In French] ["Antitetanus antibodies: assay before anatoxinotherapy in 64 tetanus patients."] *La Nouvelle Presse Medicale*, 1972; 1 (45): 3049–3050.

85. Spittle BJ. Pollock M. and O'Donnell TV. "Tetanus occurring in spite of active immunisation." *New Zealand Medical Journal*, 1973; 77 (491): 250–251.

86. Berger SA, Cherubin CE, Nelson S, and Levine L. "Tetanus despite preexisting antitetanus antibody." *Journal of the American Medical Association*, 1978; 240 (8): 769–770.

87. Dittman S. *Atypische Verlaeute nach Schutzimpfungen*. Johan Ambrosius Barth, Leipzig, Germany, 1981; p. 156.

88. Baptist EC. "Tetanus in a partially immunized infant with burns." *Pediatric Infectious Disease*, 1984; 3 (5): 487–488.

89. Vieira BI, Dunne JW, and Summers, Q. "Cephalic tetanus in an immunized patient." *Medical Journal of Australia*, 1986; 145: 156–157.

90. Passen EL, and Andersen B. "Clinical tetanus despite a protective level of toxin-neutralising antibody." *Journal of the American Medical Association*, 1988; 255 (9): 1171–1173.

91. Crone NE, and Reder AT. "Severe tetanus in immunized patients with high anti-tetanus titers." *Neurology*, 1992; 42: 761–764.

92. Luisto M, and Iivanainen M. "Tetanus of immunized children." *Developmental Medicine and Child Neurology*, 1993; 35 (4): 351–355.

93. Pryor T, Onarecker C, and Coniglione T. "Elevated abitoxin titers in a man with generalized tetanus." *The Journal of Family Practice*, 1997; 44: 299–303.

94. Shimoni Z, Dobrousin A, Cohen J, and Pilik S. "Tetanus in an immunised patient." *British Medical Journal*, 1999; 319: 1049.

95. Vinson DR. "Immunisation does not rule out tetanus." *British Medical Journal*, Letters, 2000; 320: 383.

96. Abramanian FM. "Fatal tetanus in a drug abuser with "protective" anti- tetanus antibodies." *The Journal of Emergency Medicine*, 2000, 18 (2): 189–193.

97. Atabek ME, and Pirgon O. "Tetanus in a fully immunized child." *Journal of Emergency Medicine*, 2005; 29 (3): 345–346.

98. Maselle SY, Matre R, Mbise R, and Hofstad T. "Neonatal tetanus despite protective serum antitoxin concentration." *FEMS Microbiology Letters*, 2006; 76 (3): 171–176.

99. Koenig K, Ringe H, Dorner BG, Diers A, Uhlenberg B, Mueller D, Varnholt V, and Gaedicke G. "Atypical tetanus in a completely immunized 14-year-old boy." *Pediatrics*, 2007; 120 (5): 1355–1358.

100. Galazka AM. *The Immunological Basis of Immunization Series Module 3: Tetanus*. World Health Organisation, Geneva, Switzerland, 1993 (WHO/EPI/Gen 93.13); p. 6.

101. Pascual FB. McGinley EL, Zanardi LR, Cortese MM, and Murphy TV. "Tetanus Surveillance: United States, 1998–2000." *Morbidity and Mortality Weekly Report*, 3003; 52 (SS03): 1–8.

102. Lopez RE and Holle RL. "Demographics of lightning casualties." *Seminars in Neurology*, 1995; 15: 286–295.

103. Coulter HL, and Fisher BL. *A Shot in the Dark*, Harcourt Brace Jovanovich, New York, 1985.

104. Fisher BL. *Vaccines, Autism and Chronic Inflammation*, National Vaccination Information Center, Vienna, Virginia, USA, 2008.

105. Bernard S, Enayati A, Redwood L, Roger H, and Binstock T. "Autism: a novel form of mercury poisoning." *Medical Hypotheses*, 2001; 56 (4): 462–471.

106. Colman E. "Mercury in infants given vaccines containing thiomersal." *The Lancet*, Correspondence, 2003; 361 (9358): 699.

107. Hornig M, Chian D, and Lipkin WI. "Neurotoxic effects of postnatal thimerosal are mouse strain dependent." *Molecular Psychiatry*, 2004; 9: 833–845.

108. Westphal GA, Asgari S, Schutz TG, Buenger J, Mueller M, and Hallier E. "Thimerosal induces micronuclei in the cytochalasin B block micronucleus test with human lymphocytes." *Archives of Toxicology*, 2003 77 (1): 50–55.

109. Geier MR, and Geier DA. "Neurodevelopmental disorders after thimerosal-containing vaccines: a brief communication." *Experimental Biological Medicine*, 2003 228 (6): 660–664.

110. DeLong G. "Can vaccines trigger autism?" *Social Science Research Network*, 2008; Abstract no. 1207850.

111. Centers for Disease Control and Prevention. "Prevalence of autism spectrum disorders–autism and developmental disabilities monitoring network, six cities, United States, 2000." *Morbidity and Mortality Weekly Report*, 2007; 56 (SS-1): 1–40.

112. Walton, J. "Absorption of aluminium and its effects on brain cells." In: *Aluminium: Report of an International Meeting, 20–21 April 1995, Brisbane*. National Environmental Health Health Monographs: Metal Series No. 1; P Imray, MR Moore, PW Callan, and W Lock (editors); Department of Human Services, Australia: pp. 22–34.

113. Walton J, Hams G, and Wilcox D. "Bioavailability of aluminium from drinking water: co-exposure with foods and beverages." *Research Report No. 83*, Melbourne, Victoria, Australia; Urban Water Research Association of Australia; 1994. Cited in Walton, ibid reference 112.

114. Perl DP. "Relationship of aluminum to Alzheimer's disease." *Environmental Health Perspectives*, 1985; 63: 149–153.

115. Alfrey AC, LeGendre GR, and Kaehny WD. "The dialysis encephalopathy syndrome: possible aluminum intoxication." *The New England Journal of Medicine*, 1976; 294: 184–188.

116. Klatzo I, Wisniewski H, and Streicher E. "Experimental production of neurofibrillary degeneration: I Light microscopic observations." *Journal of Neuropathology and Experimental Neurology*, 1965; 24: 187–199.

117. Terry RD and Pena C. "Experimental production of neurofibrillary degeneration. 2. Electron microscopy,

phosphatase histochemistry and electron probe analysis." *Journal of Neuropathology and Experimental Neurology*, 1965; 24: 200–210.

118. Crapper DR, Krishnan SS, and Dalton AJ. "Brain aluminum distribution in Alzheimer's disease and experimental neurofibrillary degeneration." *Science*, 1973; 180 (4083): 511–513.

119. Uemura E. "Intranuclear aluminum accumulation in chronic animals with experimental neurofibrillary changes." *Experimental Neurology*, 1984; 85 (1): 10–18.

120. Hollosi M. "Stable intrachain and intrachain complexes of neurofilament peptides: a putative link between aluminium and Alzheimer disease." *Proceedings of the National Academy of Sciences*, 1994; 91: 4902–4906.

121. Bilkei-Gorzo A. "Neurotoxic effect of enteral aluminum." *Food and Chemical Toxicology*, 1993; 31 (5): 357–361.

122. Crapper DR, Krishnan SS, and Quirrkat S. "Aluminum, neurofibrillary degeneration and Alzheimer's disease." *Brain*, 1976; 99: 67–80.

123. Perl DP, and Brody AR. "Alzheimer's disease: x-ray spectrometric evidence of aluminum accumulation in neurofibrillary tangle-bearing neurons." *Science*, 1980; 208: 297–299.

124. Perl, DP. "Aluminum and Alzheimer's disease: intraneuronal x-ray studies." *Banbury Report 15: Biological Aspects of Alzheimer's Disease*. Cold Spring Harbor Laboratory, Cold Spring Harbor, NY, USA, 1983; pp. 425–431. Cited in Perl DP, ibid reference 114.

125. Walton J, Tuniz C, Fink D, Jacobsen G, and Wilcox D. "Uptake of trace elements of aluminum into the brain from drinking water." *Neurotoxicology*, 1995; 16 (1): 187–190.

126. Becaria A, Campbell A, and Bondy SC. "Alumium as a toxicant." *Toxicology and Industrial Health*, 2002; 18 (7): 309–320.

127. Clauberg, M. and Joshi, JG. "Regulation of serine protease activity by aluminum: implications for Alzheimer disease. "*Proceedings of the National Academy of Sciences,* 1993; 90: 1009–1012.

128. Evans P. "Free radicals in brain metabolism and pathology." *British Medical Bulletin*, 1993; 49: 577–587.

129. Wisniewski HM, Sturman JA, Shek JW, "Aluminum chloride induced neurofibrillary changes in the developing rabbit, a chronic animal model." *Annals of Neurology*, 1980; 8 (5): 479–490.

130. Varner JA, Jensen KF, Horvath W, and Isaacson RL. "Chronic administration of aluminum-fluoride and sodium-fluoride to rats in drinking water: alterations in neuronal and cerebrovascular integrity." *Brain Research*, 1998; 784: 284–298.

131. Vogt T. "Water quality and health: study of a possible relationship between aluminum in drinking water and dementia." *Sosiale og Okonomiske Studier* (Central Bureau of Statistics), Oslo, Norway, 1986; 61: 1–99.

132. Flaten TP. "Geographic associations between aluminium in drinking water and death rates with demential (including Alzheimer's disease), Parkinson's disease and amylotrophic lateral sclerosis in Norway." *Environmental Geochemistry and Health*, 1990; 12: 152–167.

133. Flaten TP. "Aluminum as a risk factor in Alzheimer's disease, with emphasis on drinking water." *Brain Research Bulletin*, 2001; 55 (2): 187–196.

134. McLachlan DRC, Bergeron C, Smith JE, Boomer D, and Rifat SL. "Risk for neuropathologically confirmed Alzheimer's disease and residual aluminum in muncipal drinking water employing weighted residential histories." *Neurology*, 1996; 46 (2): 401–405.

135. Martyn CN, Barker DJ, Osmond C, Harris EC, Edwardson JA, and Lacey RF. "Geographic relation between Alzheimer's disease and aluminum in drinking water." *The Lancet*, 1989; 1 (8629): 59–62.

136. Rondeau V, Commenges D, Jacqmin-Gadda H, and Dartigues JF. "Relation between aluminum in concentrations in drinking water and Alzheimer's disease: an 8-year follow-up study." *American Journal of Epidemiology*, 2000; 152 (1): 59–66.

137. Jacqmin H, Commenges D, Letenneur L, Barberger-Gateau P, and Dartigues J-F. "Components of drinking water and the risk of cognitive impairment in the elderly." *American Journal of Epidemiology*, 1994; 139 (1): 48–57.

138. Rondeau V, Jacqmin-Gadda H, Commenges D, and Dartigues J-F. "Aluminum in drinking water and cognitive decline in elderly subjects: the Paquid Cohort." *American Journal of Epidemiology*, 2001; 154 (3): 288–290.

139. Jacqmin-Gadda H, Commenges D, Letenneur L, and Dartigues JF. "Silica and aluminum in drinking water and cognitive impairment in the elderly." *Epidemiology*, 1996; 7 (3): 281–285.

140. Gillette-Guyonnet S, Andrieu S, and Vellas B. "The potential influence of silica present in drinking water on Alzheimer's disease and associated disorders." *The Journal of Nutrition, Health and Aging*, 2007: 11 (2): 119–123.

141. Gajdusek DC, and Salazar AM. "Amylotrophic lateral sclerosis and Parkinsonism syndromes in high incidence among the Auyu and Jakai people of West New Guinea." *Neurology*, 1982; 32: 107–126. Cited in Perl DP, ibid reference 114.

142. Gajdusek DC. "Foci of motor neuron disease in high incidence in isolated populations of East Asia and the Western Pacific." In: *Human Motor Neuron Diseases, Advances in Neurology*, LP Rowland (editor), Raven Press, New York, USA, 1982; volume 36, pp. 363–394. Cited in Perl DP, ibid reference 114.

143. Sharaki H, and Yase H. "Amylotrophic lateral sclerosis in Japan." In: *Handbookd of Clinical Neurology*, PJ Vinken and GW Bruyn (editors), North Holland, Amsterdam, The Netherlands, 1975; volume 22, pp. 353–419.

144. Crapper McLachlarf DR, McLachlan CD, Krishnan B, Krishan SS, Dalton AJ and Steele JC. "Aluminium and

calcium in soil and food from Guam, Palau and Jamaica: implications for amylotrophic lateral sclerosis and Parkinsonism-dementia syndromes of Guam." *Environmental Geochemistry and Health*, 1989; 112 (2): 45–53.

145. Perl DP and Good PF. "The association of aluminum, Alzheimer's disease, and neurofibrillary tangles." *Journal of Neural Transmission*, Supplementum, 1987; 24: 205–211.

146. Lindegarde B. "Aluminum and Alzheimer's disease." *The Lancet*, 1989; 267–268.

147. Yase Y. "Pathogenesis of amylotrophic lateral sclerosis." *The Lancet*, 1972; 2: 292–296.

148. Yase Y. "Environmental contribution to the ALS process." In: Serratrice Gea (editor), *Neuromuscular Diseases*, Raven Press, New York, USA, 1984, pp. 335–339.

149. McLachlan D. "Intramuscular desferioxamine in patients with Alzheimer's disease." *The Lancet*, 1991; 337: 1304.

150. US Alzheimer's Association. "2009 Alzheimer's Disease Facts and Figures." *Alzheimer's and Dementia*, 2009; 5 (3).

151. Bassili WR, and Stewart GT. "Epidemiological evaluation of immunisation and other factors in the control of whooping cough." *The Lancet*, 1976; 1 (7957): 471–474.

152. Ditchburn RK. "Whooping cough after stopping pertussis immunisation." *British Medical Journal*, 1979; 2 (6183), (Correspondence): 207–208.

153. Ditchburn RK. "Whooping cough after stopping pertussis immunisation." *British Medical Journal*, 1979; 1 (6178): 1601–1603.

154. Stewart GT. "Whooping cough in relation to other childhood infections in 1977–9 in the United Kingdom." *Journal of Epidemiology and Community Health*, 1981; 35 (2): 139–145.

155. Stewart GT. "Vaccination against whooping cough: efficacy versus risks." *The Lancet*, 1977; 1 (8005): 234–237.

156. Stewart GT. "Toxicity of pertussis vaccine: frequency and probability of reactions." *Journal of Epidemiology and Community Health*, 1979; 33 (2): 150–156.

157. Stewart GT. "Whooping cough and pertussis vaccine: a comparison of risks and benefits in Britain during the period 1968–83." *Developments in Biological Standardization*, 1985; 61: 395–405.

158. Shelton T. "Dutch whooping cough epidemic puzzles scientists." *British Medical Journal*, 1998; 316 (7125): 91–94.

159. de Melker HE, Schellekens JFP, Neepelenbroek, SE, Mooi FR, Rumke HC, and Conyn-van Spaendonck MAE. "Reemergence of pertussis in the highly accinated population of the Netherlands: observations on

surveillance data." *Emerging Infectious Diseases*, 2000; 6 (7): 348–355.

160. Hutchins SS, Cochi SL, Brink EW, Patriarca PA, Wassilak SG, Rovira EZ, and Hinman AR. "Current epidemiology of pertussis in the United States." *The Tokai Journal of Clinical Medicine*, 1988; 13 (Supplement): 103–109.

161. Centers for Disease Control and Prevention. "Resurgence of pertussis–United States, 1993." *Morbidity and Mortality Weekly Report*, 1993; 42 (49): 945–968.

162. National Centre for Immunization Research and Surveillance of Vaccine Preventable Diseases, and the Australian Institute of Health and Welfare, Australian Government of Health and Aging. "Vaccine Preventable Disease and Vaccine Coverage in Australia, 2003 to 2005." In: *Communicable Diseases Intelligence*, 2007; 31, Supplement: 49–54.

163. Cordova SP, Gilles MT, and Beers MY. "The outbreak that had to happen: Bordetella pertussis in north-west Western Australia in 1999." *Communicable Diseases Intelligence*, 2000; 24 (12): 375–379.

164. Kenyon TA, Izurieta H, Shulman ST, Rosenfeld E, Miller M, Daum R, Strebel PM. "Large outbreak of pertussis among young children in Chicago, 1993: investigation of potential contributing factors and estimation of vaccine effectiveness." *Pediatric Infectious Diseases*, 1996; 15 (8): 655–661.

165. Christie C, Marx ML, Marchant CD, and Reising SF. "The 1993 epidemic of pertussis in Cincinnati: resurgence of disease in a highly immunized population of children." *The New England Journal of Medicine*, July 1994; 331 (1): 16–21.

166. Strebel P, Hussey G, Metcalf C, Hanslo D, and Simpson J. "An outbreak of whooping cough in a highly vaccinated community." *Journal of Tropical Pediatrics*, 1991; 37 (2): 71–76.

167. Mulholland K. "Measles and pertussis in developing countries with good vaccine coverage." *The Lancet*, 1995; 345 (8945): 305–307.

168. Trollfors B, and Rabo E. "Whooping cough in adults." *British Medical Journal (Clinical Research Edition)*, 1981; 283 (6293): 696–697.

169. Mink CA, Sirota NM, and Nugent S. "Outbreak of pertussis in a fully immunized adolescent and adult population." *Archives of Pediatrics and Adolescent Medicine*, 1994; 148 (2): 153–157.

170. Crowcroft NS, Andrews N, Rooney C, Brisson M, and Miller E. "Deaths from pertussis are underestimated in England." *Archives of Disease in Childhood*, 2002; 86 336–338.

171. Matthias RG "Whooping cough in spite of immunization." *Canadian Journal of Public Health*, 1978; 69 (2): 130–132.

172. Bennett NM. "Whooping cough in Melbourne." *Medical Journal of Australia*, 1973; 2 (10): 481–487.

173. Blakely T, Mansoor O, and Baker M. "The 1996 pertussis epidemic in New Zealand: descriptive epidemiology." *New Zealand Medical Journal*, 1999; 112 (1081): 30–33.

174. Van Loo IH, and Mooi FR. "Changes in the Dutch Bordetella pertussis population in the first 20 years after the introduction of whole-cell vaccines." *Microbiology*, 2002; 148 (7): 2011–2018.

175. Mannerstedt G. "Pertussis in adults." *Journal of Pediatrics*, 1934; 5 (5): 596–600.

176. Srugo I, Benilevi D, Madeb R, Shapiro S, Shohat T, Somekh E, Rimmar Y, Gershtein V, Gershtein R, Marva E, and Lahat N. "Pertussis infection in fully vaccinated children in day-care centers, Israel." *Emerging Infectious Diseases*, 2000; 6 (5): 526–529.

177. Frost WH, Frobisher M, Van Volkenburgh VA, and Levin ML. "Diphtheria in Baltimore: a comparative study of morbidity, carrier prevalence and antitoxin immunity in 1921–1924 and 1933–1936." *American Journal of Hygiene*, 1936, 24: 568–586.

178. Pannum PL. *Observations Made During the Epidemic of Measles on the Faroe Islands in the Year 1846*. Delta Omega Society, New York, USA, 1940.

179. Christensen PE, Schmidt H, Bang HO, Andersen V, Jordal B, and Jensen O. "An epidemic of measles in Southern Greenland, 1951: measles in virgin soil, I." *Acta Medica Scandinavica*, 1953; 144: 313.

180. Arroyo M, Alia JM, Mateos ML, Carrasco JL, Ballesteros F, and Lardinois R. "Natural immunity to measles, rubella and mumps among Spanish children in the pre-vaccination era." *International Journal of Epidemiology*, 1986; 15 ((1): 95–100.

181. Fields B, Knipe DM, Howley PM, and Griffin DE. *Field's Virology*. Lippincott Williams and Wilkins, Hagerstown, Maryland, USA, 2006; p. 1538.

182. Horstmann DM, Schuedersberg A, Emmons JE, Evans BK, Randolph MF, and Andiman WA. "Persistence of vaccine-induced immune responses to rubella: comparison with natural infection." *Review of Infectious Diseases*, 1985; 7 (Supplement 1): 80–85.

183. Buxbaum S, Doerr HW, and Allwinn R. "Epidemiological analysis of immunity against vaccine-preventable diseases" rubella, measles, mumps and chickenpox." *Deutsche Medizinische Wochenschrift* [In German], 2001; 126 (46): 1289–1293.

184. Horwitz G, Grunfeld K, Lysgaard-Hansen B, and Kjeldsen K. "The epidemiology and natural history of measles in Denmark. " *American Journal of Epidemiology*, 1974; 100 (2): 136–149.

185. Office of National Statistics.*Twentieth Century Mortality*, CD-Rom, Her Magesty's Stationery Office (HMSO), London, UK, 2001.

186. Veeder BS. "The morbidity and mortality of pertussis and measles with particular reference to age." In:

American Association for Study and Prevention of Infant Mortality: Transactions of the Seventh Annual Meeting, Milwaukee, October 19–21, 1916. Franklin Printing Company, Baltimore, Maryland, USA, 1917; p. 98.

187. Halsey NA, Modlin JF, and Jabbour JT; US Centers for Disease Control and Prevention. *Measles Suveillance Report No. 11, 1977–1981*. September 1982; 6–89.

188. Baratta RO, Ginter MC, Price MA, Walker JW, Skinner RG, Prather EC, and David JK. "Measles (rubeola) in previously immunized children." *Pediatrics*, 1970; 46 (3): 397–402.

189. Wyll SA, and Witte JJ. "Measles in previously vaccinated children: an epidemiological study." *Journal of the American Medical Association*, 1971; 216 (8): 1306–1310.

190. Lerman SJ, and Gold E. "Measles in children previously vaccinated against measles." *Journal of the American Medical Association*, 1971; 216 (8): 1311–1314.

191. Currier RW, Hardy GE Jr., and Conrad JL. "Measles in previously vaccinated children: evaluation of an outbreak." *American Journal of Diseases of Children*, 1972; 124 (6): 854–857.

192. Linnemann CC Jr., Rotte TC, Schiff GM, and Youtsey JL. "A seroepidemiologic study of a measles epidemic in a highly immunized population." *American Journal of Epidemiology*, 1972; 95 (3): 238–246.

193. Schluederberg A, Lamm SH, Landrigan PJ, and Black FL. "Measles immunity in children vaccinated before one year of age." *American Journal of Epidemiology*, 1973; 97 (6): 402–409.

194. Cherry JD, Feigin RD, Lobes LA Jr., Hinthorn DR, Shackelford PG, Shirley RH, Lins RD and Choi SC. "Urban measles in the vaccine era: a clinical, epidemiologic, and serologic study." *Journal of Pediatrics*, 1972; 81 (2): 217–230.

195. Weiner LB, Corwin RM, Nieburg PI, and Feldman HA. "A measles outbreak among adolescents." *The Journal of Pediatrics*, 1977; 90 (1): 17–20.

196. Shasby DM, Shope TC, Downs H, Herrmann KL, and Polkowski J. "Epidemic measles in a highly vaccinated population." *The New England Journal of Medicine*, 1987; 296 (11): 585–589.

197. Krause PJ, Cherry JD, Deseda-Tous J, Champion JG, Strassburg M, Sullivan C, Spencer MJ, Bryson YJ, Welliver RC, and Boyer KM. "Epidemic measles in young adults." *Annals of Internal Medicine*, 1979; 90 (6): 873–876.

198. Judelsohn RG, Fleissner ML, O'Mara DJ. "School-based measles outbreaks: correlation of age at immunization with risk of disease." *American Journal of Public Health*, 1980; 70 (11): 1162–1165.

199. McKinlay JB, and McKinlay SM. "The questionable contribution of medical measures to the decline of mortality in the United States in the Twentieth Century." *The Millbank Memorial Fund Quarterly: Health and Society*, 1977; 55 (3): 405–428.

200. McKinlay JB, and McKinlay SM. Ibid reference 199; pp. 421–425.

201. Wittke JJ, and Axnick NW, "The benefits from ten years of measles immunization in the United States." *Public Health Reports*, 1975; 90 (3): 205–207.

202. Centers for Disease Control and Prevention. "Recommendations of the Immunization Practices Advisory Committee Measles Prevention: Supplementary Statement." *Morbidity and Mortality Weekly Report*, January 13, 1989; 38 (1): 11–14.

203. Wassilak SGF, Orenstein WA, Strickland PL, Butler CA, and Bart KJ. "Continuing measles transmission in students despite a school-based outbreak control program." *American Journal of Epidemiology*, 1985; 122 (2): 208–217.

204. Centres for Disease Control and Prevention. "Measles outbreak among vaccinated high school students: Illinois." *Morbidity and Mortality Weekly Report*, 1984; 33 (24): 349–351.

205. Gustafson TL, Lievens AW, Brunell PA, Moellenberg RG, Buttery CM, and Sehulster LM. "Measles outbreak in a fully immunized secondary school population." *The New England Journal of Medicine*, 1987; 316 (13): 771–774.

206. Davis RM, Whitman ED, Orenstein WA, Preblud SR, Markowitz LE, and Hinman AR. "A persistent outbreak of measles despite appropriate prevention and control measures." *American Journal of Epidemiology*, 1987; 126 (3): 438–439.

207. Markowitz LE, Preblud SR, Orenstein WA, Rovira EZ, Adams NC, Hawkins CE, and Hinman AR. "Patterns of transmission in measles outbreaks in the United States, 1985–1986." *The New England Journal of Medicine*, 320 (2): 75–81.

208. Nkowane BM, Bart SW, Orenstein WA, and Baltier M. "Measles outbreak in a vaccinated school population: epidemiology, chains of transmission and the role of vaccine failures." *American Journal of Public Health*, 1987; 77 (4): 434–438.

209. Rullan JV, Pozo F, Gamble WB Jr., Jackson K, and Parker RL." Measles in a highly vaccinated South Carolina school population." In: *Proceedings of the 36th Annual Conference of the Epidemic Intelligence Service, April 6–10, 1987,* US Centers for Disease Control and Prevention, Atlanta, Georgia, USA, 1987; p. 24.

210. Hersh BS, Markowitz LE, Hoffman RE, Hoff DR, Doran MJ, Fleishman JC, Preblud SR, and Orenstein WA. "A measles outbreak at a college with a prematriculation immunization requirement." *American Journal of Public Health*, 1991; 81 (3): 360–364.

211. Hutchins S, Markowitz L, Atkinson W, Swint E, Hadler S. "Measles outbreaks in the United States, 1987 through 1990." *The Pediatric Infectious Disease Journal*, 1996; 15 (1): 31–38.

212. Brunell PA. "Measles one more time." *Pediatrics*, 1990; 86 (3): 474–478.

213. Centers for Disease Control and Prevention. "Measles: United States, first 26 weeks, 1989." *Morbidity and*

Mortality Weekly Report, 1989; 38 (50): 863–866, 871–872.

214. Centers for Disease Control and Prevention. "Health objectives for the nation public health burden of vaccine-preventable diseases among adults: standards for adult immunization practice." *Morbidity and Mortality Weekly Report*, 1990; 39 (41): 725–729.

215. Chen RT, Goldbaum GM, Wassilak SB, Markowitz LE, and Orenstein WA. "An explosive point-source measles outbreak in a highly vaccinated population. Modes of transmission and risk factors for disease." *American Journal of Epidemiology*, 1989; 129 (1): 173–182.

216. Centers for Disease Control and Prevention. "Public-sector vaccination efforts in response to the resurgence of measles among preschool-aged children: United States, 1989–1991." *Morbidity and Mortality Weekly Report*, 1992; 41 (29): 522–525.

217. Edmonson MB, Addiss DG, McPherson JT, Berg JL, Circo SR, and Davis JP. "Mild measles and secondary vaccine failure during a sustained outbreak in a highly vaccinated population." *Journal of the American Medical Association*, 1990; 263 (18): 2467–2471.

218. Matson DO, Byington C, Canfield M, Albrecht P, Feigin RD. "Investigation of a measles outbreak in a fully vaccinated school population including serum studies before and after revaccination." *The Pediatric Infectious Disease Journal*, 1993; 12 (4): 292–299.

219. St Geme JW Jr., George BL, Bush BM. "Exaggerated natural measles following attenuated virus immunization." *Pediatrics*, 1976; 57 (1): 148–149.

220. Davis SF, Strebel PM, Atkinson WL, Markowitz LE, Sutter RW, Scanlon KS, Friedman S, and Hadler SC. "Reporting efficiency during a measles outbreak in New York City, 1991." *American Journal of Public Health*, 1993; 83 (7): 1011–1015.

221. Poland GA, and Jacobson RM. "Failure to reach the goal of measles elimination: apparent paradox of measles infections in immunized persons." *Archives of Internal Medicine*, 1994; 154 (16): 1815–1820.

222. Centers for Disease Control and Prevention. "Rubella and congenital rubella syndrome: United States, January 1, 1991–May 7, 1994." *Morbidity and Mortality Weekly Report*, 1994; 43 (21): 397–401.

223. Centers for Disease Control and Prevention. "Measles at an international gymnastics competition: Indiana, 1991." *Morbidity and Mortality Weekly Report*, 1992; 41 (07): 109–111.

224. Ammari LK, Bell LM, and Hodinka RL. "Secondary measles vaccine failure in healthcare workers exposed to infected patients." *Infection Control and Hospital Epidemiology*, 1993; 14 (2): 81–86.

225. Centers for Disease Control and Prevention. "Measles–United States, 1996, and the interruption of indigenous transmission." *Morbidity and Mortality Weekly Report*, 1997; 46 (11): 242–246.

226. Centers for Disease Control and Prevention. "Measles: United States, 1997." *Morbidity and Mortality Weekly*

Report, 1998; 47 (14): 273–276.

227. Centers for Disease Control and Prevention. "Measles: United States, 1999." *Morbidity and Mortality Weekly Report*, 2000; 49 (25): 557–560.

228. Centers for Disease Control and Prevention. "Import-associated measles outbreak: Indiana, May–June 2005." *Morbidity and Mortality Weekly Report*, 2005; 54 (42): 1073–1075.

229. Centers for Disease Control and Prevention. "Preventable measles among U.S. residents, 2001–2004." *Morbidity and Mortality Weekly Report*, 2005; 54 (33): 817–820.

230. Centers for Disease Control and Prevention. "Measles: United States, 2005." *Morbidity and Mortality Weekly Report*, 2006; 55 (50): 1348–1351.

231. Centers for Disease Control and Prevention. "Update: Measles—United States, January–July, 2008." *Morbidity and Mortality Weekly Report*, 2008; 57 (33): 893–896.

232. Centers for Disease Control and Prevention. "Transmission of measles among a highly vaccinated school population–Anchorage, Alaska, 1998." *Morbidity and Mortality Weekly Report*, 1999; 47 (51, 52): 1109–1111.

233. Katz SL, and Hinman AR. "Summary and conclusions: measles elimination meeting, 16–17 March." *Journal of Infectious Disease*, 2004; 189 (Supplement 1): 43–47.

234. Rawls WE, Rawls ML, and Chernesky MA. "Analysis of a measles epidemic: possible role of vaccine failures." *Canadian Medical Association Journal;* 1975; 113 (10): 941–944.

235. Yuan L. "Measles outbreak in 31 schools: risk factors for vaccine failure and evaluation of a selective revaccination strategy." *Canadian Medical Association Journal*; 1994; 150 (7): 1093–1098.

236. Sekla L, Stackiw W, Eibisch G, Johnson I. "An evaluation of measles serodiagnosis during an outbreak in a vaccinated community." *Clinical and Investigative Medicine*, 1988; 11 (4): 304–309.

237. Boulianne N, De Serres G, Duval B, Joly JR, Meyer F, Dery P, Alary M, Le Henaff D, and Theriault N. "Major measles epidemic in the region of Quebec despite a 99% vaccine coverage." *Canadian Journal of Public Health*, 1991; 82 (3): 189–190.

238. Centers for Disease Control and Prevention. "Measles: Quebec." *Morbidity and Mortality Weekly Report*, 1989; 38 (18): 329–330.

239. Ozanne G, and D'Halewyn M-A. "Secondary immune response in a vaccinated population during a large measles epidemic." *Journal of Clinical Microbiology*, 1992; 30 (7): 1778–1782.

240. Hull HF, Montes JM, Hays PC, and Lucero RL. "Risk factors for measles vaccine failure among immunized students." *Pediatrics*, 1985; 76 (4): 518–523.

241. de Oliveira SA, Soares WN, Dalston MO, de Almeida MT, and Costa AJ. "Clinical and epidemiological findings during a measles outbreak occurring in a population with a high vaccination coverage." [In Portuguese]. *Revista da Sociedade Brasilera de Medicina Tropical*, 1995; 28 (4): 339–343.

242. Miller CL. "Deaths from measles in England and Wales, 1970–83." *British Medical Journal*, (Clinical Research Edition), 1985; 290 (6466): 443–444.

243. Warin JF, Harker P, and Mayon-White RT. "Measles in vaccinated children in Oxford." *The Lancet*, 1972; 2 (7781): 810–812.

244. Hicks NR. "Misplaced loss of confidence in measles vaccination: an investigation in a primary school." *Journal of the Royal College of General Practitioners*, 1989; 39: 151–152.

245. Carter H, and Gorman D. "Measles outbreak in Fife: which MMR policy?" *Public Health*, 1993; 107 (1): 25–30.

246. Hidaka Y, Aoki T, Akeda H, Miyazaki C, and Ueda K. "Serological and clinical characteristics of measles vaccine failure in Japan." *Scandinavian Journal of Infectious Diseases*, 1994; 26 (6): 725–730.

247. Kawamoto A, Honda T, Ishida K, Ozeki T, Hayashibara H, Shiraki K, and Hino S. "Two independent outbreaks of measles in partially vaccinated junior high schools in Tottori, Japan." *Archives of Virology*, 1995; 140 (2): 349–354.

248. Hirose M, Hidaka Y, Miyazaki C, Ueda K, Yoshikawa H. "Five cases of measles secondary vaccine failure with confirmed seroconversion after live measles vaccination." *Scandinavian Journal of Infectious Diseases*, 1997; 29 (2): 187–190.

249. Ong G, Hoon HB, Ong A, Chua LT, Kai CS, and Tai GK. "A 24-year review on the epidemiology and control of measles in Singapore, 1981–2004." *Southeast Asian Journal of Tropical Medicine and Public Health*, 2006; 37 (1): 96–101.

250. Albonico H-U. "Arguments against routine mumps immunization." [In German]. *Sozial und Praventivmedizin*, 1995; 40 (2): 116–123.

251. Williams P, and Hull H. "Status of measles in The Gambia, 1981." *Reviews of Infectious Diseases*, 1983; 5 (3): 391–394.

252. Commey JO, and Richardson JE. "Measles in Ghana: 1973–1982." *Annals of Tropical Paediatrics*, 1984; 4 (3): 189–194.

253. Kenya PR. "Measles and mathematics: control or eradication." *East African Medical Journal*, 1990; 67 (12): 856–863.

254. Dabis F, Sow A, Waldman RJ, Bikakouri P, Senga J, Madzou G, and Jones TS. "The epidemiology of measles in a partially vaccinated population in an African city: implications for immunizing programs." *American*

Journal of Epidemiology, 1988; 127 (1): 171–178.

255. Whittle HC, Aaby P, Samb B, Jensen H, Bennett J, Simondon R. "Effect of subclinical infection on maintaining immunity against measles in vaccinated children in West Africa." *The Lancet*, 1999; 353 (9147): 98–102.

256. Adu FD, Ikusika A, and Omotade O. "Measles outbreak in Ibadan: serological and virological identification of affected children in selected hospitals." *The Journal of Infection*, 1997; 35 (3): 241–245.

257. Lagunju IA, Orimadegun AE, Oyedemi DG. "Measles in Ibadan: a continuous scourge." *African Journal of Medicine and Medical Sciences*, 2005; 34 (4): 383–387.

258. Ekanem EE. "A 10-year review of morbidity from childhood preventable diseases in Nigeria." *Journal of Tropical Pediatrics*, 1988; 34: 325.

259. Coetzee N, Hussey GD, Visser G, Barron P, and Keen A. "The 1992 measles epidemic in Cape Town: a changing epidemiological pattern." *South African Medical Journal*, 1994; 84 (3): 145–149.

260. Murray M, and Rasmussen Z. "Measles outbreak in a northern Pakistani village: epidemiology and vaccine effectiveness." *American Journal of Epidemiology*, 2000; 151 (8): 811–819.

261. Puvimanasinghe JP, Arambepola CK, Abeysinghe NM, Rajapaksa LC, and Kulatilaka TA. "Measles outbreak in Sri Lanka, 1999–2000." *The Journal of Infectious Diseases*, 187 (Supplement 1): 241–245.

262. Atrasheuskaya AV, Kulak MV, Neverov AA, Rubin S, Ignatyev GM. "Measles cases in highly vaccinated population of Novosibirsk, Russia, 2000–2005." *Vaccine*, 2008; 26 (17): 2111–2118.

263. Paunio M, Peltola H, Valle M, Davidkin I, Virtanen M, and Heinonen OP. "Explosive school-based measles outbreak." *American Journal of Epidemiology*, 1998; 148 (11): 1103–1110.

264. Paunio M, Hedman K, Davidkin I, Valle M, Heinonen OP, Leinikki P, Salmi A, and Peltola H. "Secondary measles vaccine failures identified by measurement of IgG avidity: high occurrence among teenagers vaccinated at a young age." *Epidemiology and Infection*, 2000; 124 (2): 263–271.

265. Nagy G, Kosa S, Takatsy S, and Koller M. "The use of IgM tests for analysis of the causes of measles vaccine failures: experience gained in a epidemic in Hungary in 1980-1981." *Journal of Medical Virology*, 1984; 13 (1): 93–103.

266. Agocs MM, Markowitz LE, Straub I, and Domok I. "The 1988–1989 measles epidemic in Hungary: assessment of vaccine failure." *International Journal of Epidemiology*, 1992; 21 (5): 1007–1013.

267. Centers for Disease Control and Prevention. "Measles outbreak: Romania, 1997." *Morbidity and Mortality Weekly Report*, 1997; 46 (49): 1159–1163.

268. Hellenbrand W, Siedler A, Tischer A, Meyer C, Reiter S, Rasch G, Teichmann D, Santibanez S, Altmann D,

Claus H, and Kramer M. "Progress towards measles elimination in Germany." *The Journal of Infectious Diseases*, 2003; 187 (Supplement 1): 208–216.

269. Pedersen IR, Mordhorst CH, Glikmann G, and Von Magnus H. "Subclinical measles infection in vaccinated seropositive individuals in arctic Greenland." *Vaccine*, 1989; 7 (4): 345–348.

270. Herceg A, Passaris I, and Mead C. "An outbreak of measles in a highly immunised population: immunisation status and vaccine efficacy." *Australian Journal of Public Health*, 1994; 18 (3): 249–252.

271. McDonnell LF, Jorm LR, Patel MS. "Measles outbreak in Western Sydney. Vaccine failure or failure to vaccinated?" *Medical Journal of Australia*, 1995; 162 (9): 471–475.

272. Cullen RM, and Walker WJ. "Measles epidemics 1949–91: the impact of mass immunisation in New Zealand." *New Zealand Medical Journal*, 1996; 109 (1032): 400–402.

273. Hardy IR, Lennon DR, and Mitchell EA. "Measles epidemic in Auckland 1984–85." *New Zealand Medical Journal*, 1987; 100 (823): 273–275.

274. Harrison GP, and Durham GA. "The 1991 measles epidemic: how effective is the vaccine?" *New Zealand Medical Journal*, 1992; 105 (938): 280–282.

275. Ceyhan M, Kanra G, Vargel S, Isikcelik Y. "The evaluation of vaccination against measles at nine months of age (report of an epidemic)." *Turkish Journal of Pediatrics*, 1992; 34 (3): 127–133.

276. Yu X, Wang S, Guan J, Mahemuti P, Gou A, Liu Q, Jin X, and Ghildyal R. "Analysis of the cause of increased measles incidence in Xinjiang, China in 2004." *The Pediatric Infectious Disease Journal*, 2007; 26 (6): 513–518.

277. Muscat M, Bang H, and Glismann S. "Measles is still a cause for concern in Europe." *Eurosurveillance*, 2008; 13 (4): Article 18837.

278. Moss WJ, and Griffin DE. "Global measles elimination." *Nature Reviews Microbiology*, 2006; 4: 900–908.

279. Weibel RE, Sokes J Jr, Buynak EB, Whitman JE Jr, and Hilleman MR. "Live, attenuated mumps-virus vaccine: 3. Clinical and serological aspects in a field situation." *The New England Journal of Medicine*, 1967; 276 (5): 245–251.

280. Centers for Disease Control and Prevention. "Current trends mumps: United States, 1985–1988. *Morbidity and Mortality Weekly Report*, 1989; 38 (7): 101–1-5.

281. Wharton M, Cochi SL, Hutcheson RH, Bistowish JM, and Shaffner W. "A large outbreak of mumps in the postvaccine era." *Journal of Infectious Diseases*, 1988; 158 (6)" 1253–1260.

282. Hersh BS, Fine PE, Kent WK, Cochi SL, Kahn LH, Zell ER, Hays PL, and Wood CL. "Mumps outbreak in a highly vaccinated population." *Journal of Pediatrics*, 1991; 119 (2): 187–193.

283. Centers for Disease Control and Prevention. "Brief report: update: mumps activity—United States, January 1–October 7, 2006." *Morbidity and Mortality Weekly Report*, 2006; 55 (42): 1152–1153.

284. Briss PA, Fehrs LJ, Parker RA, Wright PF, Sannella EC, Hutcheson RH, and Schaffner W. "Sustained transmission of mumps in a highly vaccinated population: assessment of primary vaccine failure and waning vaccine-induced immunity." *Journal of Infectious Diseases*, 1994; 169 (1): 77–82.

285. Cheek JE, Baron R, Atlas H, Wilson DL, and Crider RD Jr. "Mumps outbreak in a highly vaccinated school population. Evidence for large-scale vaccination failure." *Archives of Pediatrics and Adolescent Medicine*, 1995; 149 (7): 774–778.

286. Anderson LJ, and Seward JF. "Mumps epidemiology and immunity: the anatomy of a modern epidemic." *The Pediatric Infectious Disease Journal*, 2008; 27 (10): 75–79.

287. Schaffzin JK, Pollock L, Schulte C, Henry K, Dayan G, Blog D, and Smith P. "Effectiveness of previous mumps vaccination during a summer camp outbreak." *Pediatrics*, 2007; 120 (4): 862–868.

288. Centers for Disease Control and Prevention. "Update: multistate outbreak of mumps: United States, January 1–May 2, 2006. *Morbidity and Mortality Weekly Report*, 2006; 55 (20); 559–563.

289. Centers for Disease Control and Prevention. "Brief report: update: mumps activity—United States, January 1–October 7, 2006." *Morbidity and Mortality Weekly Report*, 2006; 55 (42): 1152–1153.

290. Bitsko RH, Cortese MM, Dayan GH, Rota PA, Lowe L, Iversen SC, and Bellini WJ. "Detectiion of RNA of mumps virus during an outbreak in a population with a high level of measles, mumps, and rubella vaccine coverage." *Journal of Clinical Microbiology*, 2008; 46 (3): 1101–1103.

291. Kancheria VS, and Hanson IC. "Mumps resurgence in the United States." *Journal of Allergy and Clinical Immunology*, 2006; 118 (4): 938–941.

292. Dayan GH, Quinlisk MP, Parker AA, Barskey AE, Harris Ml, Schwarz JM, Hunt K, Finley CG, Leschinsky DP, O'Keefe Al, Clayton J, Kightlinger LK, Dietie EG, Berg J, Kenyon CL, Goldstein ST, Stokely SK, Redd SB, Rota PA, Rota J, Bi D, Roush SW, Bridges CB, Santibanez TA, Parashar U, Bellini WJ, and Seward JF. "Recent resurgence of mumps in the United States." *The New England Journal of Medicine*, 2008; 358 (15): 1580–1589.

293. Public Health Agency of Canada. "Outbreak of mumps, Montreal, October 1998 to March 1999–with a particular focus on a school." *Canada Communicable Disease Report*, 2000; 25-08.

294. Watson-Creed G, Saunders A, Scott J, Lowe L, Pettipas J, and Hatchette TF. "Two successive outbreaks of mumps in Nova Scotia among vaccinated adolescents and young adults." *Canadian Medical Association Journal*, 2006; 175 (5): 483–488.

295. Matter HC, Cloetta J, and Zimmermann H; Sentinella Arbeitsgemeinschaft. "Measles, mumps, and rubella: monitoring in Switzerland through a sentinel network, 1986–94." *Journal of Epidemiology and Community Health*, 1995; 49 (Supplement 1): 4–8.

296. Shlegel M, Osterwalder JJ, Galeazzi RL, and Vernazza PL. "Comparative efficacy of three mumps vaccines during disease outbreak in eastern Switzerland: cohort study." *British Medical Journal*, 1999; 319 (7206): 352–353.

297. Germann D, Stroehle A, Eggenberger K, Steiner CA, and Matter L. "An outbreak of mumps in a population partially vaccinated with the Rubini strain." *Scandinavian Journal of Infectious Diseases*, 1996; 28 (3): 235–238.

298. Richard Jl, Zwahlen M, Feuz M , and Matter HC; Swiss Sentinel Surveillance Network. "Comparison of the effectiveness of two mumps vaccines during an outbreak in Switzerland in 1999 and 2000: a case-cohort study." *European Journal of Epidemiology*, 2003; 18 (6): 569–577.

299. Gabutti G, Rota MC, Salmaso S, Bruzzone BM, Bella A, and Crovari P; Serological Study Group. "Epidemiology of measles, mumps and rubella in Italy." *Epidemiology and Infection*, 2002; 129 (3): 543–550.

300. Pons C, Pelayo T, Pachon I, Galmes A, Gonzalez L, Sanchez C, and Martinez F. "Two outbreaks of mumps in children vaccinated with the Rubini strain in Spain indicate low vaccine efficacy." *Eurosurveillance*, 2000; 5 (7): Article 14.

301. Cardenosa N, Dominguez A, Camps N, Martinez A, Torner N, Navas E, and Salleras L. "Non-preventable mumps outbreaks in school children in Catalonia." *Scandinavian Journal of Infectious Diseases*, 2006; 38 (8): 671–674.

302. Goncalves G, de Araujo A, and Monteiro Cardoso ML. "Outbreak of mumps associated with poor vaccine efficacy–Oporto, Portugal, 1996." *Eurosurveillance*, 1998; 3 (12): Article 2.

303. Dias JA, Cordeiro M, Afzal MA, Freitas MG, Morgado MR, Silva JL, Nunes LM, Lima MG, and Avilez F. "Mumps epidemic in Portugal despite high vaccine coverage: preliminary report." *Eurosurveillance*, 1996; 1 (4): Article 2.

304. Kumar V. "Measles outbreak in Gibraltar, August–October 2008: a preliminary report." *Eurosurveillance*, 2008; 13 (45): Article 19034.

305. Parent du Chatelet I , Floret D, Antona D, and Levy-Bruhl D. "Measles resurgence in France in 2008, a preliminary report." *Eurosurveillance*, 2009; 14 (6): Article 19118.

306. Vandermeulen C, Roelants M, Vermoere M, Roseeuw K, Goubau P, and Hoppenbrouwers K. "Outbreak of mumps in a vaccinated child population: a question of vaccine failure." *Vaccine*, 2004; 22 (21–22): 2713–2716.

307. Kaaijk P, van der Zeijst BA, Boog MC, and Hoitink CW. "Increased mumps incidence in the Netherlands: review on the possible role of vaccine strain and genotype." *Eurosurveillance*, 2008; 13 (26): Article 18914.

308. Wehner H, Morris R, Logan M, Hunt D, Jin L, Stuart J, and Cartwright K. "A secondary school outbreak of mumps following the childhood immunization programme in England and Wales." *Epidemiology and Infection*, 2000; 124: 131–136.

309. Reaney EA, Tohani VK, Devine MJ, Smithson RD, and Smyth B. "Mumps outbreak among young people in Northern Ireland." *Communicable Disease and Public Health*, 2001: 4 (4): 311–315.

310. Donaghy M, Cameron JC, and Friedrichs V. "Increasing incidence of mumps in Scotland: options for reducing transmission." *Journal of Clinical Virology*, 2006; 35 (2): 121–129.

311. van den Bosch CA, Cohen B, Walters T, and Jin L. "Mumps outbreak confined to a religious community." *Eurosurveillance*, 2000; 5 ((5): Article 15.

312. Pugh RN, Akinosi B, Pooransingh S, Kumar J, Grant S, Livesley E, Linnane J, and Ramaiah S. "An outbreak of mumps in the metropolitan area of Walsall, UK." *International Journal of Infectious Disease*, 2002; 6 (4): 283–187.

313. Centers for Disease Control and Prevention. "Mumps epidemic—United Kingdom, 2004–2005." *Morbidity and Mortality Weekly Report*, 2006; 55 (7): 173–175.

314. Cohen C, White JM, Savage EJ, Glynn JR, Choi Y, Andrews N, Brown D, and Ramsay ME. "Vaccine effectiveness estimates, 2004–2005 mumps outbreak, England." *Emerging Infectious Diseases*, 2007; 13 (1): 12–17.

315. Roberts C, Porter-Jones B, Crocker J, and Hart J. "Mumps outbreak on the Island of Anglesey, North Wales, December 2008–January 2009." *Eurosurveillance*, 2009; 14 (5): Article 19109.

316. Gee S, O'Flanagan D, Fitzgerald M, and Cotter S. "Mumps in Ireland, 2004–2008." *Eurosurveillance*, 2008; 13 (18): Article 18857.

317. Whyte D, O'Dea F, McDonnell C, O'Connell NH, Callinan S, Brosnan E, Powell J, Monahan R, Fitzgerald R, Mannix M, Greally T, Dee A, and O'Sullivan P. "Mumps epidemiology in the mid-west of Ireland 2004–2008: increasing disease burden in the university/college setting." *Eurosurveillance*, 2009; 14 (16): Article 19182.

318. Kubinyiova M, Benes C, Prikazsky V, Roubalova K, and Castkova J. "Mumps vaccination in the Czech Republic." *Eurosurveillance*, 2008; 13 (27): Article 18920.

319. Bernard H, Schwarz NG, Melnic A, Bucov V, Caterinciuc N, Pebody RG, Mulders M, Aidyralieva C, and Hahne S. "Mumps outbreak ongoing since October 2007 in the Republic of Moldova." *Eurosurveillance*, 2008; 13 (13): Article 8070.

320. Atrasheuskaya AV, Blatun EM, Kulak MV, Atrasheuskaya A, Karpov IA, Rubin S, and Ignatyev GM. "Investigation of mumps vaccine failures in Minsk, Belarus, 2001–2003. *Vaccine*, 2007; 25 (24): 4651–4658.

321. Pogorzelska M, Oldak E, Sulik A. "Mumps: still actual epidemiological problem in Poland." *Przeglad Epidemiologiczny* [In Polish], 2005; 59 (4): 841–849.

322. Stefanoff P, and Rogalska J. "Mumps in Poland in 2006." *Przeglad Epidemiologiczny* [In Polish], 2008; 62 (2): 225–228.

323. Oda K, Kato H, Konishi A. "The outbreak of mumps in a small island in Japan." *Acta Paediatrica Japonica* (Overseas Edition), 1996; 38 (3): 224–228.

324. Lee JY, Na BK, Kim JH, Lee JS, Park JW, Shin GC, Cho HW, Lee HD, Gou UY, Yang Bk , Kim J, Kang C, and Kim WJ. "Regional outbreak of mumps due to genotype H in Korea in 1999." *Journal of Medical Virology*, 2004; 73 (1): 85–90.

325. Centers for Disease Control and Prevention. "Rubella Outbreak: Arkansas, 1999." *Morbidity and Mortality Weekly Report*, 2001; 50 (50): 1137–1139.

326. Gendrel D. "Measles and rubella." [In French]. *La Revue du Praticien*, 1997; 47 (13): 1434–1437.

327. Gilbert GL. *Infectious Disease in Pregnancy and the Newborn Infant*, Informa Health Care/Taylor and Francis Group, London, UK, 1991.

328. Krohn EF. "Epidemiological aspects of rubella in Europe." *International Journal of Epidemiology*, 1972; 1 (3): 267–270.

329. Chantler J, Wolinsky JS and Tingle A. "Rubella virus." In: Knipe DM, and Howley PM (editors), *Fields Virology*, 4th Edition, Lippincott Williams and Wilkins, Philadelphia, Pennsylvannia, USA; 2001: pp. 963–990.

330. Hughes DA, Darlington LG, and Bendich A (editors). *Diet and Human Immune Function*, Humana Press Inc, Totowa, New Jersey, USA; 2004.

331. Joncas JH. "Rubella immunization." *Canadian Medical Association Journal*, 1974; 111 (9): 907–911.

332. Fulginiti VA. "Controversies in current immunization policy and practices: one physician's viewpoint." *Current Problems in Pediatrics*, 1976; 6 (6): 3–25.

333. Welch JP. "Prevention of congenital rubella." *Canadian Medical Association Journal*, 1977; 117 (2): 151–156.

334. Colgrove J. "The ethics and politics of compulsory HPV vaccination." *The New England Journal of Medicine*, 2006; 355: 2389–2391.

335. Colgrove J. "Compulsory HPV vaccination." *The New England Journal of Medicine*, Correspondence, 2007; 356: 1074–1075.

336. Horstmann DM. "Controlling rubella: problems and perspectives." *Annals of Internal Medicine*, 1975; 83 (3): 412–417.

337. Honeyman MC, Forrest JM, and Dorman DC. "Cell-mediated immune response following natural rubella and rubella vaccination." *Clinical and Experimental Immunology*, 1975; 17 (4): 665–671.

338. Horstmann DM, Schluederberg A, Emmons JE, Evans BK, Randolph MF, and Andiman WA. "Persistence of vaccine-induced immune responses to rubella: comparison with natural infection." *Reviews of Infectious Diseases*, 1985; 7 (Supplement 1): 80–85.

339. Gilmore D, Robinson ET, Gilmour WH, and Urquhart GE. "Effect of rubella vaccination programme in schools on rubella immunity in a general practice population." *British Medical Journal* (Clinical Research Edition), 1982; 284 (6316): 628–630.

340. Stoffman JM, and Wolfish MG. "The susceptibility of adolescent girls to rubella." *Clinical Pediatrics*, 1976; 15 (7): 625–626.

341. Chappell JA, and Taylor MA. "Implications of rubella susceptibility in young adults." *American Journal of Public Health*, 1979; 69 (3): 279–281.

342. Tingle AJ, Chantler JK, Kettyls GD, Larke RP, and Schulzer M. "Failed rubella immunization in adults: association with immunologic and virological abnormalities." *Journal of Infectious Diseases*, 1985; 151 (2): 330–336.

343. Duclos P, Tepper ML, Weber J, and Marusyk RG. "Seroprevalence of measles-and rubella-specific antibodies among military recruits, Canada, 1991." *Canadian Journal of Public Health*, 1994; 85 (4): 278–281.

344. Evans PC and Reisinger KS. "Rubella susceptibility in Navajo women." *Journal of Reproductive Medicine*, 1975; 14 (2): 84–85.

345. Freis PC, Sussman EK, and Shearin R. "Sero-immunity screening of adolescent girls in a military population to determine susceptibility to the rubella virus." *Military Medicine*, 1976; 141: 684–685.

346. McKusick MJ. "Screening for rubella on a university campus." *Post Graduate Medicine*, 1976; 59: 202–205.

347. Lawless MR, Abramson JS, Harlan JE, and Kelsey DS. "Rubella susceptibility in sixth-graders: effectiveness of current immunization practice." *Pediatrics*, 1980; 65 (6): 1086–1089.

348. Schiff GM, Young BC, Stefanovic GM, Stamler EF, Knowlton DR, Grundy BJ, and Dorsett PH. "Challenge with rubella virus after loss of detectable vaccine-induced antibody." *Reviews of Infectious Diseases*, 1985; 7 (Supplement 1): 157–163.

349. Crowder M, Higgins HL, and Frost JJ. "Rubella susceptibility in young women of rural east Texas: 1980 and 1985." *Texas Medicine*, 1987; 83 (5): 43–47.

350. Johnson CE, Kumar ML, Whitwell JK, Staehle BO, Rome LP, Dinakar C, Hurni W, and Nalin DR. "Antibody persistence after primary measles-mumps-rubella vaccine and response to a second dose given at four to six vs. eleven to thirteen years." *The Pediatric Infectious Disease Journal*, 1996; 15 (8): 687–692.

351. van der Heijden OG, Conyn-van Spaendonck MA, Plantinga AD, and Kretzschmar ME. "A model-based evaluation of the national immunization programme against rubella infection and congenital rubella

syndrome in The Netherlands." *Epidemiology and Infection*, 1998; 121 (3): 653–671.

352. Trier H, and Ronne T. "Duration of immunity and occurrence of secondary vaccine failure following vaccination against measles, mumps and rubella." [In Danish]. *Ugeskrift for Laeger*, 1992;154 (29): 2008–2013.

353. Just M, Just V, Berger R, Burkhardt F, and Schilt U. "Duration of immunity after rubella vaccination: a long-term study in Switzerland." *Reviews of Infectious Diseases*, 1985; 7 (Supplement 1): 91–94.

354. Davidkin I, Peltola H, Leinikki P, and Valle M. "Duration of rubella immunity induced by two-dose measles, mumps and rubella (MMR) vaccination. A 15-year follow-up in Finland." *Vaccine*, 2000; 18 (27): 3106–3112.

355. Allan B. "Rubella immunisation." *Australian Journal of Medical Technology*, 1973; 4: 26–27.

356. Horstmann DM, Liebhaber H, Le Bouvier GL, Rosenberg DA, and Halstead SB. "Rubella: reinfection of vaccinated and naturally immune persons exposed in an epidemic." *The New England Journal of Medicine*, 1970; 283 (15): 771–778.

357. Horstmann DM, Pajot TG, and Liebhaber H. "Epidemiology of rubella. Subclinical infection and occurrence of reinfection." *American Journal of Diseases in Children*, 1969; 118 (1): 133–136.

358. Wilkins J, Leedom JM, and Portnoy B, and Salvatore MA. "Reinfection with rubella virus despite live vaccine induced immunity. Trials of HPV-77 and HPV-80 live rubella virus vaccines and subsequent artificial and natural challenge studies." *American Journal of Disease in Children*, 1969; 118 (2): 275–294.

359. Abrutyn E, Hermann KL, Karchmer AW, Friedman JP, Page E, and Witte JJ. "Rubella vaccine comparative study. Nine-month follow up and serologic response to natural challenge." *American Journal of Disease in Children*, 1970; 120 (2): 129–133.

360. Chang TW, DesRosiers S, and Weinstein L. " Clinical and serologic studies of an outbreak of rubella in a vaccinated population." *The New England Journal of Medicine*, 1970; 283 (5): 246–248.

361. Forrest JM, Menser MA, Honeyman MC, Stout M, and Murphy AM. "Clinical rubella eleven months after vaccination." *The Lancet*, 1972; 300 (7774): 399–400.

362. Rachelefsky GS, and Herrmann KL. "Congenital rubella surveillance following epidemic rubella in a partially vaccinated community." *Journal of Pediatrics*, 1974; 84 (4): 474–478.

363. Kono R. "Rubella vaccination." *Bulletin of Pan American Organization*, 1976; 10 (3): 198–201.

364. Baba K, Yabuuchi H, Okuni H, Harima R, Minekawa Y, Taniuchi M, Otsuka T, Takahashi M, and Okuno Y. "Rubella epidemic in an institution: protective value of live rubella vaccine and serologicalbehavior of vaccinated, revaccinated and naturally immune groups." *Biken Journal*, 1978; 21 (1): 25–31.

365. Schiff GM, Rauh JL, Young B, Trimble S, Rotte T, and Schiff BE. "Rubella-vaccinated students. Follow-up in a public school system." *Journal of the American Medical Association*, 1978; 240 (24): 2635–2637.

366. Weibel, Buynak EB, McLean AA, and Hilleman MR. "Persistence of antibody after administration of monovalent and combined live attenuated measles, mumps, and rubella virus vaccines." *Pediatrics*, 1978; 61 (1): 5–11.

367. Weibel RE, Buynak EB, McLean AA, Roehm RR, and Hilleman MR. "Follow-up surveillance for antibody in human subjects following live attenuated measles, mumps and rubella virus vaccines." *Proceedings of the Society for Experimental Biology and Medicine*, 1979; 162 (2): 328–332.

368. Polk BF, White JA, De Girolami PC, and Modlin JF. "An outbreak of rubella among hospital personnel." *The New England Journal of Medicine*, 1980; 303 (10): 541–545.

369. Balfour HH Jr., Groth KE, Edelman CK, Amren DP, Best JM, and Banatvala JE. "Rubella viraemia and antibody responses after rubella vaccination and reimmunisation." *The Lancet*, 1981; 317 (8229): 1078–1080.

370. Arbuckle TE, and Sherman GJ. "Is rubella syndrome a vanishing disease?" *Chronic Diseases in Canada*, Public Health Agency of Canada, 1992; 13 (21): 24–28.

371. Banatvala JE. "Rubella vaccines." In: AP Waterson (editor), *Recent Advances in Clinical Virology*, Churchill Livingstone, Edinburgh, 1977; pp. 171–190.

372. Lehane DE, Newburg NR, and Beam WE Jr. "Evaluation of rubella herd immunity during an epidemic." *Journal of the American Medical Association*, 1970; 213: 2236.

373. Klock LE, and Rachelefsky GS. "Failure of rubella herd immunity during an epidemic." *The New England Journal of Medicine*, 1973; 288 (2): 69–72.

374. Bart KJ, Orenstein WA, Preblud SR, and Hinman AR. "Universal immunization to interrupt rubella." *Review of Infectious Diseases*, 1985; 7 (Supplement 1): 177–184.

375. Weinstein L, and Chang TW. "Prevention of rubella." *Pediatrics*, 1975; 55 (1): 5–6.

376. Schoenbaum SC, Biano S, and Mack T. "The role of maternal parity. Epidemiology of congenital rubella syndrome." *Journal of the American Medical Association*, 1975; 2333 (2): 151–155.

377. Centers for Disease Control and Prevention. "Measles, rubella, and congenital rubella syndrome: United States and Mexico, 1997–1999." *Morbidity and Mortality Weekly Report*, 2000; 49 (46); 1048–1050, 1059.

378. Centers for Disease Control and Prevention. "Achievements in public health: elimination of rubella and congenital rubella syndrome–United States." *Morbidity and Mortality Weekly Report*, 2005; 54 (11): 279–282.

379. Strannegard O, Holm SE, Hermodsson S, Norrby R, and Lycke E. "Case of apparent reinfection with rubella." *The Lancet* (Letter), 1970; 1 (7640): 240–241.

380. Boue A, Nicolas A, and Montagnon B. "Reinfection with rubella in pregnant women." *The Lancet*, 1971; 1

(7712): 1251–1253.

381. Chin J. Ebbin AJ, Wilson MG, and Lennette EH. "Avoidance of rubella immunization of women during or shortly before pregnancy." *Journal of the American Medical Association*, 1971; 215 (4): 632–634.

382. Joncas J. "Immunization and related problems." [In French]. *L'Union Medicale du Canada*, 1971; 100 (6): 1124–1130.

383. Bott LM, and Eizenberg DH. "Congenital rubella after successful vaccination." *Medical Journal of Australia*, 1982; 1 (12): 514–515.

384. Forsgren M, and Soren L. "Subclinical rubella infection in vaccinated women with rubella-specific IgM response during pregnancy and transmission of virus to the fetus." *Scandinavian Journal of Infectious Diseases*, 1985; 17 (4): 337–341.

385. Morgan-Capner P, Hodgson J, Hambling MH, Dulake C, Coleman TJ, Boswell PA, Watkins RP, Booth J, Stern H, Best JM, and Banatvala JE. "Detection of rubella-specific IgM in subclinical rubella reinfection in pregnancy." *The Lancet*, 1985; 1 (8423): 244–246.

386. Kantoch M, and Imbs D. "Postinfection and postvaccination antirubella immunity." *Acta Virologica*, 1986; 30 (5): 381–389.

387. Miller CL, Miller E, and Waight PA. "Rubella susceptibility and the continuing risk of infection in pregnancy." *British Medical Journal* (Clinical Research Edition), 1987; 294: 1277–1278.

388. Robinson J, Lemay M, and Vaudry W. "Congenital rubella after anticipated maternal immunity: two cases and a review of the literature." *The Pediatric Disease Journal*, 1994; 13 (9): 812–815.

389. Aboudy Y, Fogel A, Barnea B, Mendelson E, Yosef L, Frank T, and Shalev E. "Subclinical rubella infection during pregnancy followed by transmission of virus to fetus." *Journal of Infection*, 1997; 34 (3): 273–276.

390. Barfield W, Gardner R, Lett S, and Johnsen C. "Congenital rubella reinfection in a mother with anti-cardiolipin and anti-platelet antibodies." *The Pediatric Infectious Disease Journal*, 1997; 16 (2): 247–249.

391. Bott LM, Eizenberg DH. "Congenital rubella after successful vaccination." *Medical Journal of Australia*, 1982; 1 (12): 514–515.

392. Enders G, Calm A, and Schaub J. "Rubella embryopathy after previous maternal rubella vaccination." *Infection*, 1984; 12 (2): 96–98.

393. Saule H, Enders G, Zeller J, and Bernsau U. "Congenital rubella infection after previous immunity of the mother." *European Journal of Pediatrics*, 1988; 147 (2): 195–196.

394. Das BD, Lakhani P, Kurtz JB, Hunter N, Watson BE, Cartwright KA, Caul EO, and Roome AP. "Congenital rubella after previous maternal immunity." *Archives of Diseases in Children*, 1990; 65 (5): 545–546.

395. Miller E, Waight PA, Vurdien JE, White JM, Jones G, Miller BHR, Tookey PA, and Peckham CS. "Rubella surveillance to December 1990: A joint report from the PHLS and National Congenital Rubella Surveillance Programme." *Communicable Disease Review*, 1991; 1 (4): 33–37.

396. Miron D, On A. "Congenital rubella syndrome after maternal immunization." *Harefuah*, 1992; 122 (5): 291–293.

397. Serdula MK, Marks JS, Herrmann KL, Orenstein WA, Hall AD, and Bomgaars MR. "Therapeutic abortions following rubella infection in pregnancy: the potential impact on the incidence of congenital rubella syndrome." *American Journal of Public Health*, 1984; 74 (11): 1249–1251.

398. Public Health Agency of Canada. "Survey of congenital rubella syndrome, Montreal, Laval, and Monteregie, Quebec, 1985–1991." *Canada Communicable Disease Report*, 1996; 22 (5): F1–F2.

399. Public Health Agency of Canada. "Surveillance of congenital rubella syndrome and other rubella-associated adverse pregnancy outcomes." *Canada Communicable Disease Report*, 1996; 22 (5): F3–F4.

400. De Serres G, Dery P, Lebel M, et al. "Epidemiologie de la rubeole congenitale au Quebec de 1965 a 1994." [In French]. Presented at the V e Colloque quebecois sur les maladies infectieuses, Rimouski, Quebec, Canada, May, 1994. Cited in Public Health Agency, ibid reference 399.

401. Swartz TA, Praiss I, Isacson M, Nishmi M, Ben-Porath E, and Hornstein L. "Early results of an extensive rubella epidemic." *International Journal of Epidemiology*, 1975; 4 (4): 331–335.

402. Office of Population Censuses and Surveys, London. "Rubella-associated terminations of pregnancy." *OPCS Monitor*, February 5, 1980. Cited in Serdula et al, ibid reference 397.

403. Tobin JO, Sheppard S, Smithells RW, Milton A, Noah N, and Reid D. "Rubella in the United Kingdom, 1970–1983." *Reviews of Infectious Diseases*, 1985; 7 (Supplement 1): 47–52.

404. Kadoya R, Ueda K, Miyazaki C, Hidaka Y, and Tokugawa K. "Incidence of congenital rubella syndrome and influence of the rubella vaccination programme for schoolgirls in Japan, 1981–1989." *American Journal of Epidemiology*, 1998; 148 (3): 263–268.

405. Katow, S. "Rubella virus genome diagnosis during pregnancy and mechanism of congenital rubella." *Intervirology*, 1998; 41: 163–169.

406. Yellowlees H. "Incidence of congenital rubella." *British Medical Journal*, Correspondence, 1978; 2 (6151): 1569.

407. Miller E, Waight PA, Vurdien JE, Jones G, Tookey PA, and Peckham CS. "Rubella surveillance to December 1992: second joint report from the PHLS and National Congenital Rubella Surveillance Programme." *Communicable Disease Report*, 1993; 3 (3): 35–39.

408. Plotkin SA, and Orenstein WA. *Vaccines*. Third Edition. WB Saunders Company, Philadelphia,

Pennsylvania, USA, 1999; p. 410.

409. Lotter, V. "Epidemiology of autistic conditions in young children." *Social Psychiatry and Psychiatric Epidemiology*, 2004; 1 (3): 124–137.

410. Gardner SG, Bingley PJ, Sawtell PA, Weeks S and Gale EAM. "Rising incidence of insulin dependent diabetes in children aged under 5 years in the Oxford region: time trend analysis." *British Journal of Medicine*, 1997; 315 (7110): 713–717.

411. Bingley PJ, and Gale EAM "Rising incidence of IDDM in Europe." *Diabetes Care*, 1989; 12 (4): 289–295.

412. Diabetes Epidemiology Research International Group. Secular trends in incidence of childhood IDDM in 10 countries." *Diabetes*, 1990; 39 (7): 858–864.

413. Rahi JS, Dezateux C, and the British Congenital Cataract Interest Group. "Congenital and infantile cataract in the United Kingdom: underlying or associated factors." *Investigative Ophthalmology and Visual Science*, 2000; 41 (8): 2108–2114.

414. Cherry JD. "The 'new' epidemiology of measles and rubella." *Hospital Practice*, 1980; 15 (7): 49–57.

415. Centers for Disease Control and Prevention. "Current trends rubella and congenital rubella syndrome: United States, 1985–1988." *Morbidity and Mortality Weekly Report*, 1989; 38 (11): 173–178.

416. Centers for Disease Control and Prevention. "Current trends increase in rubella and congenital rubella syndrome: United States, 1988–1990." *Morbidity and Mortality Weekly Report*, 1991; 40 (6): 93–99.

417. Centers for Disease Control and Prevention. "Rubella and congenital rubella syndrome: United States, 1994–1997." *Morbidity and Mortality Weekly Report*, 1997; 46 (16): 350–354.

418. Vynncky E, Gay NJ, and Cutts FT. "The predicted impact of private sector MMR vaccination on the burden of congenital rubella syndrome." *Vaccine*, 2003; 21 (21–22): 2708–2719.

419. Centers for Disease Control and Prevention. "Progress toward elimination of rubella and congenital rubella syndrome: the Americas, 2003–2008." *Morbidity and Mortality Weekly Report*, 2008; 57 (43): 1176–1179.

420. Kelly H, Worth L, Karapanagiotidis T, and Riddell M. "Interruption of rubella virus transmission in Australia may require vaccination of adult males: evidence from a Victorian sero-survey." *Communicable Diseases Intelligence*, 2004; 28 (1): 69–73.

421. Briss PA, Fehrs LJ, Hutcheson RH, and Shaeffer W. "Rubella among the Amish: resurgent disease in a highly susceptible community." *The Pediatric Disease Journal*, 1992; 11 (11): 955–959.

422. Demicheli V, Jefferson T, Rivetti A, and Price D. "Vaccines for measles, mumps and rubella in children." *Cochrane Database of Systemic Reviews*, 2005; Issue 4, Article no: CD004407.

423. Cherry JD, Heininger U, Stehr K, and Christenson P. "The effect of investigator compliance (observer bias) on calculated efficacy in a pertussis vaccine trial." *Pediatrics*, 1998; 102 (4): 909–912.

424. Underwood M. *Treatise on the Diseases of Children: With General Directions for the Management of Infants from the Birth*, Published by T. Dobson, Philadelphia, Pennsylvania, USA, 1793. American Antiquarian Society database, first series 26291.

425. Paul JR. "Historical and geographical aspects of the epidemiology of poliomyelitis." *Yale Journal of Biology and Medicine*, 1954; 27 (2): 101–113.

426. Bell C. *The Nervous System of the Human Body As Explained in a Series of Papers, Read Before the Royal Society of London*, 3rd edition, London, UK, 1836, p. 434.

427. Badham J. *London Medical Gazette*, 1836; 17: 215.

428. Trevelyan B, Smallman-Raynor M, and Cliff AD. "The spatial dynamics of poliomyelitis in the United States: from epidemic emergence to vaccine-induced retreat, 1910–1971." *Annals of the Association of American Geographers*, 2005; 95 (2): 269–293.

429. Peters ST. *Epidemic: The Battle Against Polio*, Marshall Cavendish Corporation, Tarrytown, NY, USA, 2005; pp. 13–21.

430. Massachusetts State Board of Health. *Infantile Paralysis in Massachusetts, 1907–1912*, Wright and Potter Publishers, 1914. Published by Project Gutenberg Internet Library Archives, San Francisco, California, USA.

[Online, accessed 7th September, 2009]. URL:http://www.archive.org/details.infantile paraly00healgoog

431. Painter CF. "Infantile paralysis. An epidemic of thirty-eight cases." *The Journal of Bone and Joint Surgery*, 1902; s1–15: 414–433.

432. Caverly CS. *Infantile Paralysis in Vermont, 1894–1922*. Published and distributed by the Vermont State Department of Public Health, Burlington, Vermont, USA, 1924.

433. Roberts J. "Polio: the virus and the vaccine." *Ecologist*, May 2004, pp. 36–52.

434. Scobey RR. "The poison cause of poliomyelitis and obstructions to its investigation." *Archive of Pediatrics*, 1952; 69 (4): 172–193.

435. Van Sweeten G. *Aphorisms of Dr Herman Boerhaave*, Volume 10, 1765. Cited in Scobey, ibid reference 434.

436. Cooke J. *Treatise of Nervous Diseases*, 1824. Cited in Scobey, ibid reference 434.

437. Vulpian A. Quoted by Lovett RW, *Boston Medical and Surgical Journal*, 1908; 159: 131–138. Cited in Scobey, ibid reference 434.

438. Popow. Quoted by Mills CK, *Boston Medical and Surgical Journal*, 1883; 108: 248–250. Cited in Scobey, ibid reference 434.

439. Altman. *Australasian Medical Gazette*, July 3, 1897, p. 173. Quoted by LE Holt, and FH Bartlett. "Epidemiology of acute poliomyelitis: a study of thirty-five epidemics." *American Journal of the Medical Sciences*, 1908; 135 (5): 647–661. Cited in Scobey, ibid reference 434.

440. Vonderabe AR. *Archives of Neurology and Psychiatry*, 1931; 25: 29–43. Cited in Scobey, ibid reference 434.

441. Gringer RR. *Archives of Neurology and Psychiatry*, 1931; 25: 649–653. Cited in Scobey, ibid reference 434.

442. Onuff (1900). Quoted by RW Lovett, *Boston Medical and Surgical Journal*, 1908; 159: 131–138. Cited in Scobey, ibid reference 434.

443. Philippe and Gauthard (1903). Quoted by RW Lovett, *Boston Medical and Surgical Journal*, 1908; 159: 131–138. Cited in Scobey, ibid reference 434.

444. Obrastoff (1902). Quoted by RW Lovett, *Boston Medical and Surgical Journal*, 1908; 159: 131–138. Cited in Scobey, ibid reference 434.

445. Collins J, and Marchland HS. "Disease of primary motor neurones causing clinical picture of acute anterior poliomyelitis: result of poisoning by cyanide of potassium; clinical and experimental contribution to toxic effects of cyanide of potassium." *Journal of Nervous and Mental Disease*, 1908; 35: 417–426. Cited in Scobey, ibid reference 434.

446. Edsall DE. *Osler's System of Medicine*, Volume 1, 1907; p. 144. Cited in Scobey, ibid reference 434.

447. Lambert SM. "A yaws campaign and an epidemic of poliomyelitis in Western Samoa." *Journal of Tropical Medicine and Hygiene*, 1936; 39: 41–46. Cited in Scobey, ibid reference 434.

448. Fiorini E. "Physics of rare events: insights on Napoleon death." *Nuclear Physics B–Proceedings Supplements*, 2009; 188: 365–370.

449. Scobey RR. "Is Human Poliomyelitis Caused by an Exogenous Virus." *Archives of Pediatrics*, 1954; 71 (4): 111–123. Commentary by Jim West.

[Online, accessed 7th September, 2009]. URL:http://www.harpub.co.cc/scobexog.htm

450. Emerson HC. "An epidemic of infantile paralysis in Western Massachusetts in 1908." *Boston Medical and Surgical Journal*, 1909; 151: 115–119. Commentary by Jim West.

[Online, accessed 7th July, 2009]. URL:http://www.harpub.co.cc/mass1908_2articles.htm

451. West J. *Images of Poliomyelitis: Polio in the United States*, Graph Timeline, US 1870–1998.

[Online, accessed 7th September, 2009]. URL:http://www.harpub.co.cc/pol_all.htm

452. West J. *Images of Poliomyelitis: Pesticide Introduction and Poliomyelitis*.

[Online, accessed 7th September, 2009]. URL:http://www.harpub.co.cc/pestintr.htm

453. Scobey RR. "Is human poliomyelitis caused by an exogenous virus?" *Archives of Pediatrics*, 1954; 71 (4): 111–123. Commentary by Jim West.

[Online, accessed 7th September, 2009]. URL:http://www.harpub.co.cc/scobexog.htm

454. Caverly (1894), Dana (1894), Free (1908). Cited in Lovett RW, "The occurrence of infantile paralysis in Massachusetts in 1908." Reported for the Massachusetts State Board of Health." *Boston Medical and Surgical Journal*, 1908; 159: 131–138.

[Online, accessed 7th July, 2009]. URL:http://www.harpub.co.cc/mass1908_2articles.htm

455. West J. "Pesticides and Polio."*Images of Poliomyelitis: A Critique of Scientific Literature*.

[Online, accessed 7th September, 2009]. URL:http://www.harpub.co.cc/overview.htm

456. Biskind MS. "Public health aspects of the new insecticides." *American Journal of Digestive Disease*, 1953; 20 (11): 331–341.

457. Biskind MS, and Bieber I. "DDT poisoning: a new syndrome with neuropsychiatric manifestations." *American Journal of Psychotherapy*, 1949; 3 (2): 261–270.

458. Biskind MS. "Statement on clinical intoxication from DDT and other new insecticides." Presented before a Select Committee to Investigate the Use of Chemicals in Food Products, US House of Representatives, December 12, 1950. *Journal of Insurance Medicine*, May, 1951.

459. Post Polio Health International. *Incidence Rates of Poliomyelitis in US*.

[Online, accessed 7th September, 2009]. URL:http://www.post-polio.org/ir-usa.html

460. Sabin AB. "The epidemiology of poliomyelitis: problems at home and among the armed forces abroad." *Journal of the American Medical Association*, 1947; 134 (9): 749–756.

461. Paul JR. "Poliomyelitis attack rates in American troops, 1940–1948." *The American Journal of Hygiene*, 1949; 50 (1): 57–62.

462. Paul JR. "Neurotropic Virus Diseases: Poliomyelitis." In: Lieutenant General LD Heaton, Colonel JB Coates Jr., and WP Havens Jr. (editors), *Internal Medicine in World War II*, Volume II, Infectious Diseases; Office of the Surgeon General, Department of the Army, Washington, DC, USA, 1963; pp. 91–99.

463. Paul JR, Havens WP Jr, and Van Rooyen CE. "Poliomyelitis in British and American troops in the Middle East; isolation of the virus from human faeces." *British Medical Journal*, 1944; 1 (4355): 841–843.

464. Caughey JE, and Porteous WM. "An epidemic of poliomyelitis occurring among troops in the Middle East." *Medical Journal of Australia*, 1946; 1: 5–10.

465. Hillman CC. "Poliomyelitis in the Philippine Islands." *Military Surgeon*, 1936; 79: 48–58.

466. Martin JK. "Local paralysis in children after injections." *Archives of Diseases of Childhood*, 1950; 25: 1–14.

467. McCloskey BP. "The relation of prophylactic inoculations to the onset of poliomyelitis." *The Lancet*, 1950; 255 (6606): 659–663.

468. Geffen DH. "The incidence of paralysis occurring in London children within four weeks after immunization." *Medical Officer*, 1950; 83: 137–140.

469. Bradford Hill A, Knowelden J. "Inoculation and poliomyelitis: a statistical investigation in England and Wales." *British Medical Journal*, 1950; 2 (4669): 1–6.

470. Editorial: "Inoculation and poliomyelitis." *The British Medical Journal*, 1950; 2 (4669): 28–29.

471. Toomey JA. "Second attacks of poliomyelitis." *American Journal of Disease in Children*, 1938; 56 95): 969–974.

472. Bridge EM, Clarke GH, and Abbe D. "Clinical immunity in poliomyelitis." *American Journal of Disease in Children*, 1946; 72 (5): 501–509.

473. Fox MJ, Madden WJ, and Kohn SE. "Recurrent poliomyelitis." *American Journal of Disease in Children*, 1948; 75 (3): 395–400.

474. Toomey JA. "Intranasal or gastrointestinal portal of entry in poliomyelitis." *Science*, 1935; 82 (2122): 200–201.

475. Klenner FR. "The treatment of poliomyelitis and other virus diseases with vitamin C." *Southern Medicine and Surgery*, 1949; 111 (7): 209–214.

476. Sandler BP. *Diet Prevents Polio*, The Lee Foundation for Nutritional Research, Milwaukee, Wisconsin, USA, 1951.

[Online, accessed 7th September, 2009]. URL:http://www.whale.to/v/sandler.html

477. Sandler, ibid reference 476, p. 43 and p. 146.

478. *Physicians' Desk Reference* (PDR), 55th edition. Medical Economics, Montvale, New Jersey, USA, 2001, p. 778.

479. West J. *Images of Poliomyelitis*. "Franklin D. Roosevelt at Campobello."

[Online, accessed 7th September, 2009]. URL:http://www.harpub.co.cc/fdr.htm)

480. Interview with J Sonnabend. "AIDS Inc", *Conspiracy Nation*, 1988; 7: (33): Part 4.

[Online, accessed 7th September, 2009]. URL:http://www.textfiles.com/conspiracy/CN/cn07–33.txt

481. Roberts J. *Fear of the Invisible: An Investigative Journey into a Reckless and Contaminated Medical Industry*, Impact Investigative Media Productions, Bristol, UK, 2008, p. 53.

482. Dalldorf G, and Sickles GM. "An unidentified, filterable agent isolated from the feces of children with paralysis." *Science*, 1948; 108 (2794): 61–62.

483. Enders JF, Weller TH, and Robbins FC. "Cultivation of the Lansing strain of poliomyelitis in cultures of various human embryonic tissues." *Science*, 1949; 109: 85–87.

484. Beddow Bayly M. *The Story of the Salk Anti-Poliomyelitis Vaccine*, "Statistical errors," 1956.

[Online, accessed 7th September, 2009]. URL:http://www.whale.to/vaccine/bayly.html

485. Offit PA. *The Cutter Incident, 50 Years Later: How America's First Polio Vaccine Led to the Growing Vaccine Crisis*, Yale University Press, New Haven, Connecticut, USA, 2005, pp. 42–44.

486. Offit, ibid reference 485, pp. 62–63.

487. Engel L. "The Salk vaccine: what caused the mess?" *Harpers Magazine*, August 1955, pp. 27–33.

488. Offit, ibid reference 485, pp. 61–62.

489. Offit PA. "The Cutter incident, 50 years later." *The New England Journal of Medicine*, 2005; 352 (14): 1411–1412.

490. Krasner G, and Mesh B. "The Salk Vaccine and the 'Disappearance' of Paralytic Polio: Is Paralysis a Viral Disease?" *In: Polio Awareness Day: Medical Myths Die Hard*, Part 1, 22nd September, 2006.

[Online, accessed 7th September, 2009]. URL:http://www.whale.to/a/krasner1.html

491. *New York Times*, May 11, 1956 Cited by Schippell TM. "The Salk monkey kidney juice." In: McBean, ibid reference 36, Chapter 10, "The hidden dangers in polio vaccine."

492. Shakespeare W. *Romeo and Juliet*, Act 2, Scene 2, Lines 46 and 47, 1594.

493. Scheibner V. *Hearings on Hepatitis B Vaccine*, "Letter to Miss Pinkerton," 16 June, 1999.

[Online, accessed 7th September, 2009]. URL:http://www.whale.to/vaccines/scheibner8.html)

493. James W. *Immunization, the Reality Behind the Myth*, 2nd edition, Bergin and Garvey (imprint of Greenwood Publishing Group), Westport, Connecticut, USA, 1995, p. 36.

495. Menkes JH. *Textbook of Child Neurology*, 5th edition, Williams and Wilkins, Baltimore, Maryland, USA, 1995, p. 420.

496. Goldman AS, Schmalstieg EJ, Freeman DH Jr, Goldman DA, and Schmalstieg FC Jr. "What was the cause of Franklin Delano Roosevelt's paralytic illness?" *Journal of Medical Biography*, 2003; 11 (4): 232–240.

497. Lyle WH. "An outbreak of a disease believed to have been caused by ECHO 9 virus." *Annals of Internal Medicine*, 1959; 51: 248–269.

498. Ramsay AM, Aberd MD, and O'Sullivan E. "Encephalomyelitis simulating poliomyelitis." *The Lancet*, 1956; 267 (6926): 761–764.

499. James W, ibid reference 493, p. 36. Citing the Los Angeles County Health Index, *Morbidity and Mortality*, "Reportable Diseases", 1967.

500. Rodale JI (editor-in-chief). *The Encyclopedia of Common Diseases*, Rodale Press, Emmaus, Pennsylvania, USA, 1962. Citing: *Illinois Medical Journal*, 1960; August and September editions. Reprinted in: *Child and Family,* 1980; 19: 195–213, 259–280.

501. Ratner H. *Nature, the Physician, and the Family: Selected Writings of,Herbert Ratner, MD*, MT Baggot (editor), AuthorHouse, Bloomington, Indiana, USA, 1962, pp. 259–264.

502. Sweet BH, and Hilleman MR. "The vacuolating virus, SV40." Proceedings of the Society for Experimental Biology and Medicine, 1960; 105: 420–427.

503. Eddy BE. "Tumors produced in hamsters by SV40." *Federal Proceedings*, 1962; 21: 930–935.

504. Immunization Safety Review Committee, Institute of Medicine of the National Academies. *Immunization Safety Review: SV 40 Contamination of Polio Vaccine and Cancer*, Stratton K, Alamario DA, and McCormick MC (editors), The National Academies Press, Washington, DC, USA; "Executive Summary", p. 5.

505. Kirschstein RL, and Gerber P. "Ependymomas produced after intracerebral inoculation of S40 into new-born hamsters." *Nature*, 1962; 195: 299–300.

506. Schein HM, and Enders JF. "Transformation induced by simian virus 40 in human renal cell cultures, I. Morphology and growth characteristics." *Proceedings of the National Academies of Sciences*, USA, 1962; 48 (7): 1164–1172.

507. Koprowski H, Ponten J, Jensen F, Ravdin RG, Moorhead P, and Saksela E. "Transformation of cultures of human tissue infected with simian virus 40." *Acta-Unio Internationalis Contra Cancorum*, 1963; 19: 362–367.

508. Jensen F, Koprowski H, Pagano JS, Ponten J, and Ravdin RG. "Autologous and homologous implantation of human cells transformed in vitro by simian virus 40." *Journal of the National Cancer Institute*, 1964; 32 (4): 917–937.

509. Horwini ME. "Simian virus 40 (SV40): a cancer causing monkey virus from FDA-approved vaccines." *Albany Law Journal of Science and Technology*, 2003; 13 (3).

510. Bookchin D, and Schumacher J. *The Virus and the Vaccine: The True Story of a Cancer-Producing Monkey Virus, Contaminated Polio Vaccine, and the Millions of Americans Exposed*, St Martin's Press, New York, NY, USA, 2004.

511. Carlsen W. "Rogue virus in the vaccine: early polio vaccine harbored virus now feared to cause cancer in humans." *San Francisco Chronicle*, July, 2001.

512. "Questions and answers." *Journal of the American Medical Association*, 1961; 175 (8): 735–740.

513. American Academy of Pediatrics. *Report of Committee on Infectious Diseases*, American Academy of Pediatrics, Evanston, Illinois, USA, 1966, 47.

514. Fraumeni JF Jr, Ederer F, and Miller RW. "An evaluation of the carcinogenicity of the simian virus 40 in man." *Journal of the American Medical Association*, 1963; 185 (9): 713–718.

515. Fraumeni JF Jr. Stark CR, Gold E, an Lepow ML. "Simian virus 40 in polio vaccine follow-up of newborn recipients." *Science*, 1970; 167 (914): 59–63.

516. Letter from John T. Conner of Merck & Co. to Dr Leroy Burney, Surgeon General of the United States, dated 12/16/60; Plaintiff's Exhibit No. 54. In: *Sabin Polio Litigation, MDL 780, U.S.D.C. MD, Baltimore, Maryland*. Cited in Kops SP. "Oral polio vaccine and human cancer: a reassessment of SV40 as a contaminant based upon legal documents." *Anticancer Research*, 2000; 20 (6C): 4745–4749.

517. Meyer HM, Hopps HE, Rogers NG, Brooks BE, Berneheim BC, Jones WP, Nisalak A, and Douglas RD. "Studies on simian virus 40." *Journal of Immunology*, 1962; 88: 796–806.

518. Abraham R, Minor P, Dunn G, Modlin JF, and Ogra PL. "Shedding of virulent poliovirus revertants during immunization with oral poliovirus vaccine after prior immunization with inactivated polio vaccine." *The Journal of Infectious Diseases*, 1993; 168 (5): 1105–1109.

519. Lederle's *Cytomegalovirus Contingency Plan*, 4 August, 1972. Cited by Martin JW, Center for Complex Infectious Diseases, Rosemead, California, USA, *Detection of Stealth Viruses in Human Tissues*. Presented at a meeting entitled "Consultation on detection of simian megaloviruses in human tissues," National Institute of Allergy and Infectious Diseases, Rockville, Maryland, USA, July 1, 1996.

    [Online, accessed 7th September, 2009]. URL:http://www.ccig.org/

520. Centers for Disease Control and Prevention. "Current trends paralytic poliomyelitis: United States, 1982 and 1983." *Morbidity and Mortality Weekly Report*, Nov 16, 1984; 33(45): 635.

521. Boffey PM. "Polio: Salk challenges safety of Sabin's live-virus vaccine." *Science*, 1977; 196 (4285): 35–36.

522. Sutter RW, Patriarca PA, Cochi SL, Pattansch MA, Kew OM, Hall DB, Brogan S, Malankar PG, Al-Ghassany AAK, Suleiman AJM, El-Bualy MS, Bass AG, and Alexander JP. "Outbreak of paralytic poliomyelitis in Oman: evidence for widespread transmission among fully vaccinated children." *The Lancet*, 1991; 338 (8769): 715–720.

523. Strebel PM, Aubert-Combiescu A, Ion-Nedelcu N, Biberi-Moroeanu S, Combiescu M, Sutter RW, Kew OM, Pallansch MA, Patriarca PA, and Cochi SL. "Paralytic poliomyelitis in Romania, 1984–1992." *American Journal of Epidemiology*, 1994; 140 (12): 1111–1124.

524. Strebel PM, Ion-Nedelcu N, Baughman AL, Sutter RW, and Cochi SL. "Intramuscular injections within 30 days of immunization with oral poliomyelitis vaccine: a risk factor for vaccine-associated paralytic poliomyelitis." *The New England Journal of Medicine*, 1995, 332 (8): 500–506.

525. Whale Vaccination Information Website. *Citations of Vaccine-associated Polio*.

[Online, accessed 7th September, 2009]. URL:http://www.whale.to/vaccine/polio6.html

526. Strebel PM, Sutter RW, Cochi SL, Biellik RJ, Brink EW, Kew OM, Pallansch MA, Orenstein WA, and Hinman AR. "Epidemiology of poliomyelitis in the United States one decade after the last reported case of indigenous wild virus-associated disease." *Clinical Infectious Disease*, 1992; 14 (2): 568–579.

527. Los Angeles County Health Index: *Morbidity and Mortality, Reportable Diseases, 1967*. Cited in: Kent C. "Drugs, bugs, and shots in the dark." *Health Freedom News*, Monrovia, California, January 1983, p. 26. Quoted by: James W, *Immunization, The Reality Behind the Myth*, Bergen and Garvey Publishers Inc, South Hadley, Massachusetts, USA, 1988, p. 28.

528. Krasner G. *Polio Awareness Day: Medical Myths Die Hard*, September 19, 2006.

[Online, accessed 7th September, 2009].

URL:http://www.whale.to/vaccine/Polio%20Awareness%20Day%20%20Medical%20Myths%20Die%20Hard.pdf

529. Centers for Disease Control and Prevention. "Outbreak of aseptic meningitis: Whiteside County, Illinois, 1995." *Morbidity and Mortality Weekly Report*, March 14, 1997; 46 (10): 221–224.

530. Dowsett EG, Ramsay AM, McCartney RA, and Bell EJ. "Myalgic encephalomyelitis: a powerful enteroviral infection?" *Postgraduate Medical Journal*, 1990; 66 (777): 526–530.

531. Dowsett EG. *The Late Effects of ME: Can They Be Distinguished from the Post-Polio Syndrome?* Presentation to the All Party Group of MPs on ME/PPS on 31st January, 2001. Published online by the Lincolnshire Post-Polio Library, February, 2001.

[Online, accessed 7th September, 2009].

URL:http://www.ott.zynet.co.uk/polio/lincolnshire/library/dowsett/lateeffectsme.html

532. Bruno RL, Frick NM, Creange S, Zimmerman JR, and Lewis T. "Polioencephalitis and the brain fatigue

generator model of post-viral fatigue syndromes." *Journal of Chronic Fatigue Syndrome*, 1996; 2 (2/3): 5–27.

533. Bruno RL, Creange SJ, Frick NM. "Parallels between post-polio fatigue and chronic fatigue syndrome: a common pathophysiology?" *The American Journal of Medicine*, 1998; 105 (3A): 66S–73S.

534. Hyde B (editor). *M.E. Epidemics: The Clinical and Scientific Basis of Myalgic Encephalomyelitis/Chronic Fatigue Syndrome*, Nightingale Research Foundation, Ottawa, Ontario, Canada, 1992.

535. Bruno RL. *Dr Bruno's Response to People Who Write About "Poliovirus Causing CFS*, June 12, 2002.

[Online, accessed 7th September, 2009].

URL:http://www. ippso-world.org/ppsinfo/articles/bruno/response.html

536. Douglas WC. "Chronic fatigue syndrome: the hidden polio epidemic." *Second Opinion Newsletter*, 1996; 6 (8): 1–6.

537. Martin WJ. "Detection of viral related sequences in CFS patients using the polymerase chain reaction." In: Byron M Hyde (editor),*The Clinical and Scientific Basis of Myalgic Encephalomyelitis/Chronic Fatigue Syndrome*, Nightingale Research Foundation Press, Ottawa, Ontario, Canada, 1992, pp. 278–283.

538. Martin WJ. "Viral infection in CFS patients." In: Byron M Hyde (editor)*The Clinical and Scientific Basis of Myalgic Encephalomyelitis/Chronic Fatigue Syndrome*, , Nightingale Research Foundation Press, Ottawa, Ontario, Canada, 1992, pp. 325–327.

539. Martin WJ. Zeng LC, Ahmed K, and Roy M. "Cytomegalovirus-related sequences in an atypical cytopathic virus repeatedly isolated from a patient with the chronic fatigue syndrome." *American Journal of Pathology*, 1994; 145: 441–452.

540. Martin WJ, Ahmed KN, Zeng LC, Olsen J-S, Seward JG, and Seehrai JS. "African green monkey origin of atypical cytopathic 'stealth virus' isolated from a patient with chronic fatigue syndrome." *Clinical and Diagnostic Virology*, 1995; 4 (1): 93–103.

541. Martin WJ. "Stealth adaptation of an African green monkey simian cytomegalovirus." *Experimental and Molecular Pathology*, 1999; 66 (1): 3–7.

542. Martin WJ, and Glass RT. "Acute encephalopathy induced in cats with a stealth virus isolated from a patient with chronic fatigue syndrome." *Pathobiology*, 1995; 63 (3): 115–118.

543. Martin WJ, and Anderson D. "Stealth virus epidemic in the Mohave Valley: initial report of viral isolation." *Pathobiology*, 1997; 65 (1): 51–56.

544. Martin WJ, and Anderson D. "Stealth virus epidemic in the Mohave Valley: severe vacuolating encephalopathy in a child presenting with a behavioral disorder." *Experimental and Molecular Pathology*, 1999; 66 (1): 19–30.

545. Dang-Tan T, Mahmud SM, Puntoni R, and Franco EL. "Polio vaccines, simian virus 40, and human cancer: the epidemiologic evidence for a causal association." *Oncogene*, 2004; 23: 6535–6540.

546. Innis MD. "Oncogenesis and poliomyelitis vaccine." *Nature*, 1968; 219 (5157): 972–973.

547. Farwell JR, Dohrmann GJ, Marrett LD, and Meigs JW. "Effect of SV40 virus-contaminated polio vaccine on the incidence and type of CNS neoplasms in children: a population-based study." *Transactions of the American Neurological Association*, 1979; 104: 261–264.

548. Heinonen OP, Shapiro S, Monson RR, Hartz SC, Rosenberg L, and Slone D. "Immunization during pregnancy against poliomyelitis and influenza in relation to malignancy." *International Journal of Epidemiology*, 1973; 2 (3): 229–235.

549. Bergsagel DJ, Finegold MJ, Butel JS, Kupsky WJ, and Garcea RL. "DNA sequences similar to those of simian virus 40 in ependymomas and choroid plexus tumors of childhood." *The New England Journal of Medicine*, 1992; 326 (15): 988–993.

550. Lednicky JA, Garcea RL, Bergsagel DJ, and Butel JS. "Natural simian virus 40 strains are present in human choroid plexus and ependymoma tumors." *Virology*, 1995; 212 (2): 710–717.

551. Cicala C, Pompetti F, and Carbone M. "SV40 induces mesotheliomas in hamsters" *American Journal of Pathology*, 1993; 142 (5): 1524–1533.

552. Carbone M, Pass HI, Rizzo P, Marinetti M, Di Muzio M, Mew DJ, Levine AS, and Procopio A. "Simian virus 4-like DNA sequences in human pleural mesothelioma." *Oncogene*, 1994; 9 (6): 1781–1790.

553. De Rienzo A, Tor M, Sterman DH, Aksoy F, Albelda SM, and Testa JR. "Detection of SV40 DNA sequences in malignant mesothelioma specimens from the United States, but not from Turkey." *Journal of Cellular Biochemistry*, 2002; 84 (3): 455–459.

554. Hirvonen A, Mattson K, Karjalainen A, Ollkainen T, Tammilehto L, Hovi T, Vainio H, Pass HI, Di Resta I, Carbone M, and Linnainmaa K. "Simian virus 40 (SV40)-like DNA sequences not detectable in Finnish mesothelioma patients not exposed to SV40-contaminated polio vaccines." *Molecular Carcinogenesis*, 1999; 26 (2): 93–99.

555. Carbone M, Kratzke RA, and Testa JR. "The pathogenesis of mesothelioma." *Seminars in Oncology*, 2002; 29 (1): 2–17.

556. Bright RK, Kimchi ET, Shearer MH, Kennedy RC, and Pass HL. "SV40 Tag-specific cytotoxic T lymphocytes generated from the peripheral blood of malignant pleural mesothelioma patients." *Cancer Immunology, Immunotherapy*, 2002; 50 (12): 682–690.

557. Foddis R, De Rienzo A, Broccoli D, Bocchetta M, Stekala E, Rizzo P, Tosolini A, Grobelny JV, Jhanwar SC, Pass HI, Testa JR, and Carbone M. "SV 40 infection induces telomerase activity in human mesothelial cells." *Oncogene*, 2002; 21 (9): 1434–1442.

558. Carbone M, Kratzke RA, and Testa JR. "The pathogenesis of mesothelioma." *Seminars in Oncology*, 2002; 29 (1): 2–17.

559. Gazdar AF, and Carbone M. "Molecular pathogenesis of malignant mesothelioma and its relationship to simian virus 40." *Clinical Lung Cancer*, 2003; 5 (3): 177–181.

560. Carbone M, Rizzo P, Procopio A, Giuliano M, Pass HI, Gebhardt MC, Mangham C, Hansen M, Malkin DF, Bushart B, Pompetti F, Picci P, Levine AS, Bersagel JD, and Garcea RL. "SV40 sequences in human bone tumors." *Oncogene*, 1996; 13 (3): 527–535.

561. Testa JR, Carbone M, Hirvonen A, Khalili K, Krynska B, Linnainmaa K, Pooley FD, Rizzo P, Rusch V, and Xiao GH. "A multi-institutional study confirms the presence and expression of simian virus 40 in human malignant mesotheliomas." *Cancer Research*, 1998; 58 (2): 4505–4506.

562. Vilchez RA, Lednicky JA, Halvorson SJ, White ZS, Kozinetz CA, and Butel JS. "Detection of polyomavirus simian virus 40 tumor antigen DNA in AIDS-related systemic non-Hodgkin lymphoma." *Journal of Acquired Immune Deficiency Syndrome*, 2002; 29 (2): 109–116.

563. Vilchez RA, and Butel JS. "SV40 in bone cancers and non-Hodgkin's lymphoma." *Oncogene*, 2003; 22 (33): 5164–5172.

564. Martini F, Lazzarin L, Iaccheri L, Vignocchi B, Finocchiaro G, Magnani I, Serra M, Scotlandi K, Barbanti-Brodano G, and Tognon M. "Different simian virus 40 genome regions and sequences homologous with SV40 large T antigen in DNA of human brain and bone tumours and of leukocytes from blood donors." *Cancer*, 2002; 94 (4): 1037–1048.

565. Vilchez RA, and Butel JS. "Simian virus 40 and its association with human lymphomas." *Current Oncology Reports*, 2003; 5 (5): 372–379.

566. Butel JS, Arrington AS, Wong C, Lednicky JA, and Finegold MJ. "Molecular evidence of simian virus 40 infections in children." *Journal of Infectious Diseases*, 1999; 180 (3): 884–887.

567. Martini F, De Mattei M, Iaccheri L, Lazzarin L, Barbanti-Brodano G, Tognon M, and Gerosa M. "Human brain tumors and simian virus 40." *Journal of the National Cancer Institute*, 1995; 87 (17): 1331.

568. Martini F, Lazzarin L, Iaccheri L, Corallini A, Gerosa M, Trabanelli C, Calza N, Barbanti-Brodano G, and Tognon M. "Simian virus 40 footprints in normal human tissues, brain and bone tumors of different histotypes." *Developments in Biological Standardization*, 1998; 94: 55–66.

569. Huang H, Reis R, Yonekawa Y, Lopes JM, Kleihues P, and Ohgaki H. "Identification in human brain tumors of DNA sequences specific for SV40 large T antigen. *Brain Pathology*, 1999; 9 (1): 33–42.

570. Zhen HN, Zhang X, Bu XY, Zhang ZW, Huang WJ, Zhang P, Liang JW, and Wang XL. "Expression of the simian virus 40 large tumor antigen (Tag) and formation of Tag-p53 and Tag-pRb complexes in human brain tumors." *Cancer*, 1999; 86 (10): 2124–2132.

571. Martini F, Iaccheri L, Lazzarin L, Carinci P, Corallini A, Gerosa M, Iuzzolino P, Barbanti-Brodano G, and Tognon M. "SV40 early region and large T antigen in human brain tumors, peripheral blood cells, and sperm fluids from healthy individuals." *Cancer Research*, 1996; 56: 4820–4825.

572. Martini F, Iaccheri L, Lazzarin L, Carinci P, Corallini A, Gerosa M, Iuzzolino P, Barbanti-Brodano G, and Tognon M. "SV40 early region and large T antigen in human brain tumors, peripheral blood cells, and sperm fluids from healthy individuals." *Cancer Research*, 1997; 57 (15): 3319–3320.

573. Barbanti-Brodano G, Sabbioni S, Martini F, Negrini M, Corallini A, and Tognon M. "Simian virus 40 infection in humans and association with human diseases: results and hypotheses." *Virology*, 2004; 318 (1): 1–9.

574. Zhen H, Zhang X, Zhang Z, Fei Z, He X, Liang J, Huang W, Liu X, and Zhang P. "Simian virus 40 large tumor antigen forms specific complexes with p53 and pRb in human brain tumors." *Chinese Medical Journal* [In English], 2001; 114 (4): 382–386.

575. Horwin M. *Simian Virus 40 (SV40) from FDA-Approved Polio Vaccine*, SV40 Foundation.

[Online, accessed 7th September, 2009]. URL:http://www.sv40foundation.org

576. Butel JS, Jafar S, Wond C, Arrington AS, Opekun AR, Finegold MJ and Adam E. "Evidence of SV40 infections in hospitalized children." *Human Pathology*, 1999; 30 (12): 1496–1502.

577. Butel JS, and Lednicky JA. "Response re: Cell and molecular biology of simian virus 40: implications for human infections and disease." *Journal of the National Cancer Institute*, 1999; 91 (13): 1166–1167.

578. Rosa FW, Sever JL, and Madden DL. "Absence of antibody response to simian virus 40 after inoculation with killed poliovirus vaccine of mothers of offspring with neurologic tumors." *The New England Journal of Medicine*, 1988; 318 (22): 1469.

579. Rosa FW, Sever JL, and Madden DL.. "Response to: Neurologic tumors in offspring after inoculation of mothers with killed poliomyelitis vaccine." *The New England Journal of Medicine*, 1988; 319: 1226.

580. Brock B. *Paper Presented at the Workshop on Simian Virus 40 (SV40): A Possible Human Papillomavirus Workshop*, January 1997. At: Kops SP, SV40Cancer.com.

[Online, accessed 7th September, 2009]. URL:http://www.sv40cancer.com/paperled.asp

581. Kops SP. "Oral polio vaccine and human cancer: a reassessment of SV40 as a contaminant based upon legal documents." *Anticancer Research*, 2000; 20 (6C): 4745–4749. Also at: Kops SP, SV40Cancer.com.

[Online, accessed 7th September, 2009]. URL:http://www.sv40cancer.com/pap1.asp

582. Kops SP. *The Paper Trail: Correspondence with FDA, 1–6*. At: Kops SP, SV40Cancer.com

[Online, accessed 7th September, 2009]. URL:http://www.sv40cancer.com/cor1.asp

583. Kops SP. *Oral Presentation to the Subcommittee on Human Rights and Wellness of the Committee on Government Reform: "The SV40 Virus: Has Tainted Polio Vaccine Caused an Increase in Cancer?"* September 10, 2003.

[Online, accessed 7th September, 2009]. URL:http://www.sv40cancer.com/oralpres.asp

584. Board on Health Promotion and Disease Prevention, Institute of Medicine. *Immunization Safety Review: SV40 Contamination of Polio Vaccine and Cancer*, The National Academies Press, Washington, DC, USA, 2002.

585. Cutrone R, Lednicky J, Dunn G, Rizzo P, Bocchetta M, Chumakov K, Minor P, and Carbone M. "Some oral poliovirus vaccines were contaminated with infectious SV40 after 1961." *Cancer Research*, 2005; 65: 10273–10279.

586. Melnick JL, and Stinebaugh S. "Excretion of vacuolating SV40 (papovavirus group) after ingestion as a contaminant of oral polio vaccine." *Proceedings of the Society for Experimental and Biological Medicine*, 1962; 109: 965–968.

587. Rizzo P, Di Resta I, Powers A, Ratner H, and Carbone M. "Unique strains of SV40 in commercial poliovaccines from 1955 not readily identifiable with current testing for SV40 infection." *Cancer Research*, 1999; 59: 6103–6108.

588. National Cancer Institute, US National Institutes of Health. *Studies Find No Evidence that SV40 is Related to Human Cancer*, 23 August, 2004.

[Online, accessed 7th September, 2009]. URL:http://www.cancer.gov/newscenter/pressreleases/SV40.

589. Wakefield AJ, Blaxhill M, Haley B, Ryland A, Hollenbeck D, Johnson J, Moody J, and Stott C. "Response to Dr. Ari Brown and the Immunization Action Coalition." *Medical Veritas*, 2009; 6: 1907–1924.

590. US Food and Drug Administration, Vaccine Adverse Event Reporting System (VAERS), *News and Events: Statement of Susan S Ellenberg before the Subcommittee on Criminal Justice, Drug Policy and Human Resources House Committee on Government Reform*, May 18, 1999.

[Online, accessed 7th September, 2009]. URL:http://www.fda.gov/NewsEvents/Testimony/ucm115058

591. Rosenthani S, and Chen R. "The reporting sensitivities of two passive surveillance systems for vaccine adverse events." *American Journal of Public Health*, 1995; 85: 1706–1709.

592. Braun MM. *Vaccine Adverse Event Reporting System (VAERS): Usefulness and Limitations*, John Hopkins Bloomberg School of Public Health, Baltimore, Maryland, USA, 2006.

[Online, accessed 7th September, 2009]. URL;http://www.vaccinesafety.edu/VAERS/htm

593. Whale.to Website: *Medical Citations (Vaccination)*.

[Online, accessed 7th September, 2009]. URL:http://www.whale.to/vaccine/citations.html

594. Diodati C, Horowitz L, Scheibner V, and West E. "Vaccination: Part II–The Risks They Pose to Your Health." *Consumer Health* (Consumer Health Organization of Canada, Toronto, Ontario, Canada), 1999; volume 22,

issue 5.

[Online, accessed 7th September, 2009].

URL:http://www.consumerhealth.org/articles/display.cfm?ID=19990705003020

595. King PG, And Goldman GS. "Key realities about autism, vaccines, vaccine-injury compensation, Thimerosal, and autism-related research." *Medical Veritas*, 2008; 5: 1610–1644.

596. Poling J, and Poling T. "Vaccines, autism and our daughter Hannah." *New York Times*, April 5, 2008.

597. Medical Advisory Committee of the Immune Deficiency Foundation. *The Clinical Presentation of the Primary Immunodeficiency Diseases : A Primer for Physicians*, Towson, Maryland, USA, 1992.

598. Van Hoff J, Schymura MJ, and Curnen MG. "Trends in the incidence of childhood and adolescent cancer in Connecticut, 1935–1979." *Medical and Pediatric Oncology*, 1988; 16 (2): 78–87.

599. Gurney JG, Davis S, Severson RK, Fang JY, Ross JA, and Robison LL. "Trends in cancer incidence among children in the U.S." *Cancer*, 1996; 78 (3): 532–541.

600. Bleyer WA. "What can be learned about childhood cancer from *Cancer Statistics Review*, 1973–1988." *Cancer*, 1993; 71 (10): 3229–3236.

601. Gurney JG, Davis S, Severson RK, Fang JY, Ross JA, and Robison LL. "Trends in cancer incidence among children in the U.S." *Cancer*, 1996; 78 (3): 532–541.

602. Gurney JG, Davis S, Severson RK, and Robison LL. "The influence of subsequent neoplasms on incidence trends in childhood cancer." *Cancer Epidemiology, Biomarkers and Prevention*, 1994; 3 (4): 349–351.

603. Bunin GR, Feuer EJ, Witman PA, and Meadows AT. "Increasing incidence of childhood cancer: report of 20 years experience from the greater Delaware Valley Pediatric Tumor Registry." *Paediatric and Perinatal Epidemiology*, 1996; 10 (3): 319–338.

604. Swenson AR, and Bushhouse SA. "Childhood cancer incidence and trends in Minnesota, 1988–1994." *Minnesota Medicine*, 1998; 81 (12): 27–32.

605. Linet MS, Ries LA, Smith MA, Tarone RE, and Devesa SS. "Cancer surveillance series: recent trends in childhood cancer incidence and mortality in the United States." *Journal of the National Cancer Institute*, 1999; 91 (12): 1051–1058.

606. Mangano JJ. "A rise in the incidence of childhood cancer in the United States." *International Journal of Health Services*, 1999; 29 (2): 393–408.

607. Moll UM, LaQuaglia M, Benard J, and Riou G. "Wild-type p53 undergoes cytoplasmic sequestration in undifferentiated neuroblastomas but not in differentiated tumors." *Proceedings of the National Academy of*

Sciences, USA, 1995; 92 (10): 4407–4411.

608. Teitz T, Chang JC, Kitamura M, Yen TSB, and Kan YW. "Rhabdomyosarcoma arising in transgenic mice harboring the beta-globin locus control region fused with simian virus 40 large T antigen gene. *Proceedings of the National Academy of Sciences, USA*, 1993; 90 (7): 2910–2914.

609. DeCaprio JA, Ludlow JW, Figge J, Shew JY, Huang CM, Lee WH, Marsilio JY, Paucho E, and Livingston DM. "SV40 large tumor antigen forms a specific complex with the product of the retinoblastoma susceptibility gene." *Cell*, 1988; 54 (2): 275–283.

610. McWhirter WR, Dobson C, and Ring I. "Childhood cancer incidence in Australia, 1982–1991." *International Journal of Cancer*, 1998; 65 (1): 34–38.

611. Lotter V. "Epidemiology of autistic conditions in young children." *Social Psychiatry and Psychiatric Epidemiology*, 1967; 1 (4): 163–173.

612. Brask BH. "A prevalence investigation of childhood psychoses." Paper presented at the 16th Scandinavian Congress of Psychiatry, 1970. In: *Nordic Symposium on the Care of Psychotic Children,* Trykningssentral, Oslo, Norway, *Barnepsykiatrist Forening*, 1972: 145-153.

613. Fombonne E. "Epidemiology of autistic disorder and other pervasive developmental disorders." *Journal of Clinical Psychiatry*, 2005; 66 (Supplement 10): 3–8.

614. Fombonne E. "Epidemiological surveys of autism and other pervasive developmental disorders: an update." *Journal of Autism and Developmental Disorders*, 2003; 33 (4): 365–382.

615. Centers for Disease Control and Prevention. "Prevalence of autism spectrum disorders: Autism and Developmental Disabilities Monitoring Network, six sites, United States, 2000." *Morbidity and Mortality Weekly Report*, 2007; 56 (SS01): 1–11.

616. MacDermott S, Williams K, Ridley G, Glasson E, and Wray J. *The Prevalence of Autism in Australia: Can it be Established from Existing Data?* A report prepared for Australian Advisory Board on Autism Spectrum Disorders, Forestville, NSW, Australia, 2006.

617. Yasbak EF, Lang-Radosh KL. "Adverse outcomes associated with postpartum rubella or MMR vaccine." *Medical Sentinel*, 2001; 6 (3): 95–99.

618. Offit PA. "Inoculated against facts." *New York Times*, March 31, 2008.

619. Offit PA. "Vaccines and autism revisited—the Hannah Poling case." *The New England Journal of Medicine*, 2008; 358: 2089–2091.

620. Poling J. "Vaccines and autism revisited." Letters, *The New England Journal of Medicine*, 2008; 359: 655–656.

621. American Academy of Pediatricians. *A.L.A.R.M. Autism (Fact Sheet)*, January 2004.

[Online, accessed 7th September, 2009]. URL:http://www.aap.org/healthtopics/autism.cfm

622. McDonald KL, Huq SI, Lix LM, Becker AB, and Kozyrskyj AL. "Delay in diphtheria, pertussis, tetanus vaccine is associated with a reduced risk of childhood asthma." *The Journal of Allergy and Clinical Immunology*, 2008; 121 (3): 626–631.

623. Classen DC, and Classen JB. "The timing of pediatric immunization and the risk of insulin-dependent diabetes." *Infectious Diseases in Clinical Practice*, 1997; 6 (7): 449–454.

624. Classen DC, and Classen JB. "Immunizations in the first month of life may explain the decline in incidence of IDDM in the Netherlands." *Autoimmunity*, 1999; 31: 43–45.

625. Classen JB, and Classen DC. "Public should be told that vaccines may have long term adverse effects." Letters, *British Medical Journal*, 1999; 318 (7177): 193.

626. Classen JB, and Classen DC. "Association between type 1 diabetes and Hib vaccine." Letters, *British Medical Journal*, 1999; 319 (7217): 1133.

627. Classen JB. "Diabetes epidemic follows hepatitis B immunization program." *New Zealand Medical Journal*, 1996; 109 (1022): 195.

628. Dokheel TM. "An epidemic of childhood diabetes in the United States? Evidence from Allegheny County, Pennsylvania." *Diabetes Care*, 1993; 16 (12): 1606–1611.

629. Gardner S, Bingley PJ, Sawtell PA, Weeks S, and Gale EA. "Incidence of insulin dependent diabetes is rising sharply in children under 5." *British Medical Journal*, 1997; 315 (7110): 713–716.

630. Kalokerinos A. *Medical Pioneer of the 20th Century: Dr. Archie Kalokerinos: An Autobiography*. Biological Therapies Publishing, Melbourne, Victoria, Australia, 2000.

631. Kalokerinos A. *Every Second Child*, Nelson, Melbourne, Victoria, Australia, 1974.

632. Kalokerinos A. Interview with Dr Kris Gaublomme, Genk, Belgium: *International Vaccine Newsletter*, June 1955.

[Online, accessed 7th September, 2009]. URL:http://www.whale.to/v/kalokerinos.html

633. Hume R, and Weyers E. "Changes in the leucocyte ascorbic acid concentration during the common cold." *Scottish Medical Journal,* 1973; 18: 3.

634. Kalokerinos A, and Dettman G. "Sudden death in infancy syndrome in Western Australia." *Medical Journal of Australia*, 1976; 2 (1): 31–32.

635. Dettman G. "Factor 'X', subclinical scurvy and S.I.D.S. Historical. Part 1." *The Australasian Nurses Journal*, 1978; 7 (7): 2–5.

636. Kalokerinos A, and Dettman G. "Aboriginal health: the gentle art of deception." *The Australasian Nurses Journal*, 1980; 10 (1): 14–15.

637. Kalokerinos A, and Dettman G. "Rubella immunisation: a tangle of absurdities and some comments." *The Australasian Nurses Journal*, 1981; 10 (11): 3–6.

638. Kalokerinos A. *The Vaccination Dilemma*, (video), 1992. Cited by HV Martin/Free-America, "Was the AIDS virus tested on expendable people?" *North American Investigative Journal*, 1995.

[Online, accessed 7th September, 2009]. URL:http://dmc.members.sonic.net/sentinel/naij4.html

639. Hadwen WR. "The fraud of vaccination." *The Truth*, January 3, 1923.

640. Hadwen WR. *Dare Doctors Think?* Verbatim Report: Great Meeting Held in Queen's Hall, London, Friday February 6, 1925. Published by The British Union for the Abolition of Vivisection, London, 1925.

641. Rook GA, and Stanford JL. "Given us this day our daily germs." *Immunology Today*, 1998; 19 (3): 113–116.

642. Reidler J, Braun-Fahrlander C, Eder W, Schreuer M, Waser M, Maisch S, Carr D, Schieri R, Nowak D, von Mutius E. (The ALEX Study Team, Salzburg, Austria). "Exposure to farming in early life and development of asthma and allergy: a cross-sectional survey." *The Lancet*, 2001; 358 (9288): 1129–1133.

643. Braun-Fahrlander C, Gassner M, Grize L, Neu U, Sennhauser FH, Varonier HS, Vuille JC, and Wuthrich B. (SCARPOL: Swiss Study on Childhood Allergy and Respiratory Symptoms with Respect to Air Pollution). "Prevalence of hay fever and allergic sensitization in farmers' children and their peers living in the same rural community." *Clinical and Experimental Allergy*, 1999; 29 (1): 28–34.

644. Strachan DP. "Family size, infection and atopy: the first decade of the 'hygiene hypothesis'." *Thorax*, 2000; 55 (Supplement 1): S2–S10.

645. Kramer U, Heinrich J, Wist M, and Wichmann HE. "Age of entry to day nursery and allergy in later childhood." *The Lancet*, 1999; 353 (9151): 450–454.

646. Cramer DW, Welch WR, Cassells S, and Scully RE. "Mumps, menarche, menopause, and ovarian cancer." *American Journal of Obstetrics and Gynecology*, 1983; 147 (1): 1–6.

647. West RO. "Epidemiologic study of malignancies of the ovaries." *Cancer*, 1966; 19 (7): 1001–1007.

648. Menczer J, Modan M, Ranon L, and Golan A. "Possible role of mumps virus in the etiology of ovarian cancer." *Cancer*, 1979; 43 (4): 1375–1379.

649. Yazdanbakhsh M, Kremsner PG, and van Ree R. "Allergy, parasites, and the hygiene hypothesis." *Science*, 2002; 298 (5567): 490–494.

650. Ronne T. "Measles virus infection without rash in childhood is related to disease in later life." *The Lancet*, 1985; 1 (8419): 1–5.

651. Kucukosmanoglu E, Cetinkaya F, Akcay F, Pekun F. "Frequency of allergic diseases following measles." [In Spanish]. *Allergologia et Immunopathologia*, 2006; 34 (4): 146–149.

652. Shaheen SO, Aaby P, Hall AJ, Barker DJ, Heyes CB, Shiell AW, and Goudiaby A. "Measles and atopy in Guinea-Bissau." *The Lancet*, 1996; 347 (9018): 1792–1796.

653. Aim JS, Swartz J, Lilja G, Scheynius A, Pershagen G. "Atopy in children of families with an anthroposophical lifestyle." *The Lancet*, 1999; 353 (9163): 1485–1488.

654. Flotsrup H, Swartz J, Bergstrom A, Aim JS, Scheynius A, van Hage M, Waser M, Braun-Fahrlander C, Schram-Bijkerk D, Huber M, Zutavern A, von Mutius E, Ublagger E, Riedler J, Michaels KB, Pershagen B; (the Parsifal Study Group). "Allergic disease and sensitization in Steiner school children." *Journal of Allergy and Clinical Immunology*, 2006; 117 (1): 59–66.

655. Rosenlund H, Bergstrom A, Aim JS, Swartz J, Scheynius A, van Hage M, Johansen K, Brunekreef B, von Mutius E, Ege MJ, Riedler J, Braun-Fahriander C, Waser M, Pershagen B; (Parsifal Study Group). "Allergic disease and atopic sensitization in children in relation to measles vaccination and measles infection." *Pediatrics*, 2009; 123 (3): 771–778.

656. Rooth IB, and Bjorkman A. "Suppression of Plasmodium falciparum infections during concomitant measles or influenza but not during pertussis." *American Tropical Medicine and Hygiene*, 1992; 47 (5): 675–681.

657. Lepore L, Agosti E, Pennesi M, Barbi E, De Manzini A. "Long-term remission induced by measles infection and followed by immunosuppressive therapy in a case of refractory juvenile rheumatoid arthritis." *La Pediatria e Medical Chirurgia*, [In Italian] 1988; 10 (2): 191–193.

658. Simpanen E, van Essen R, and Isomaki H. "Remission of juvenile rheumatoid arthritis (Still's disease) after measles." *The Lancet*, 1977; 2 (8045): 987–988.

659. Yoshioka K, Miyata H, and Maki S. "Transient remission of juvenile rheumatoid arthritis after measles." *Acta Pediatrica Scandinavica*, 1981; 70 (3): 419–420.

660. Urbach J, Schirr D, and Abramov A. "Prolonged remission of juvenile rheumatoid arthritis (Still's disease) following measles." *Acta Paediatrica Scandinavica*, 1983; 72 (6): 917–918.

661. Bonjean M, and Prime A. "Suspensive effect of measles in psoriasic erythroderma of 12 years' duration." [In French], *Lyon Medical*, 1969; 222 (40): 839.

662. Fomkin KF. "Cure of psoriasis after co-existing measles." [In Russian],*Vestnik Dermatologi i Venerologii*, 1961; 35: 66-68.

663. Lintas N. "Case of psoriasis cured after recurrent measles." [In Italian], *Minerva Dermatologica*, 1959; 24 (4):

296–297.

664. Thiers H, Normand J, Fayolle J. "Suspensive effect of measles on chronic psoriasis in children: 2 cases." [In French], *Lyon Medical*, 1969; 222 (40): 839–840.

665. Sasco AJ, and Paffenbarger RS Jr. "Measles infection and Parkinson's disease." *American Journal of Epidemiology*, 1985; 122 (6): 1017–1031.

666. Yamamoto H , Yamano T, Niijima S, Kohyama J, and Yamanouchi H.n"Spontaneous improvement of intractable epileptic seizures following acute viral infections." *Brain and Development*, 2004; 26 (6): 377–379.

667. Cookson WO, and Moffatt MF. "Asthma: an epidemic in the absence of infection?" *Science*, 1997; 275 (5296): 41–42.

668. Matricardi PM, Rosmini F, Ferrigno L, Nisini R, Rapicetta M, Chionne P, Stroffolini T, Pasquini P, and D'Amelio R. "Cross sectional retrospective study of prevalence of atopy among Italian military students with antibodies against hepatitis A virus." *British Medical Journal*, 1997; 314 (7086): 999.

669. Yazdanbakhsh M, Kremsner PG, and van Ree R. "Allergy, parasites, and the hygiene hypothesis." *Science*, 2002; 298 (5567): 490–494.

670. Fortun P. "Scientists team up with an unlikely ally—hookworms." *News and Reviews*, University of Nottingham, UK, August 4, 2006.

[Online accessed 7th September, 2009].
URL:http://research.nottingham.ac.uk/Newsreviews/newsDisplay.aspx?id=270

671. Yamamoto H, Yamano T, Niijima S, Kohyama J, and Yamanouchi H. "Spontaneous improvement of intractable epileptic seizures following acute viral infections." *Brain and Development*, 2004; 26 (6): 377–379.

672. Shafren DR, Au GG, Nguyen T, Newcombe NG, Haley ES, Beagley L, Johansson ES, Hersey P, and Barry RD. "Systemic therapy of malignant human melanoma tumors by a common cold-producing enterovirus, Coxsackievirus A21." *Clinical Cancer Research*, 2004; 10 (Part 1): 53–60.

673. Au GG, Lindberg AM, Barry RD, and Shafren DR. "Oncolysis of vascular malignant human melanoma tumors by Coxsackievirus A21." *International Journal of Oncology*, 2005; 26 (6): 1471–1476.

674. Au GG, Lincz LF, Enno A, Shafren DR. "Oncolytic Coxsackievirus A21 as a novel therapy for multiple myeloma." *British Journal of Haematology*, 2007; 137 (2): 133–141.

675. Berry LJ, Au GG, Barry RD, and Shafren DR. "Potent oncolytic activity of human enteroviruses against human prostate cancer." *Prostate*, 2008; 68 (6): 577–587.

676. Skelding KA, Barry RD, and Shafren DR. "Systemic targeting of metastatic human breast tumor xenografts by Coxsackievirus A21." *Breast Cancer Research and Treatment*, 2009; 113 (1): 21–30.

677. Shafren DR, Sylvester D, Johansson ES, Campbell IG, and Barry RD. "Oncolysis of human ovarian cancers by echovirus type 1." *International Journal of Cancer*, 2005; 115 (2): 320–328.

678. Clarke P, Meintzer SM, Gibson S, Widmann C, Garrington TP, Johnson GL, and Tyler KL. "Reovirus-induced apoptosis is mediated by TRAIL." *Journal of Virology*, 2000; 74 (17): 8135–8139.

679. Hirasawa K, Nishikawa SG, Norman KL, Coffey MC, Thompson BG, Voon CS, Waisman DM, and Lee PWK. "Systemic reovirus therapy of metastatic cancer in immune-competent mice." *Cancer Research*, 2003; 63 (2): 348–353.

680. Thirukkumaran CM, and Morris DG. "Oncolytic viral therapy using reovirus." *Methods in Molecular Biology*, 2009; 542: 607–634.

681. Thirukkumaran CM, Nodwell MJ, Hirasawa K, Shi Z-Q, Diaz R, Luider J, Johnston RN, Forsyth PA, Magliocco AM, Lee P, Nishikawa S, Donnelly B, Coffey M, Trpkov K, Fonseca K, Spurreff J, and Morris DG. "Oncolytic viral therapy for prostate cancer: efficacy of reovirus as a biological therapeutic." *Cancer Research*, 2010; 70 (6): 2435–2444.

682. MacKie RM, Stewart B, Brown SM. "Intralesional injection of herpes simplex virus 1716 in metastatic melanoma." *The Lancet*, 2001; 357 (9255): 525–526.

683. Varghese S, and Rabkin S. "Oncolytic herpes simplex virus vectors for cancer virotherapy." *Cancer Gene Therapy*, 2002; 9 (12): 967–978.

684. Steiner PE, and Loosli CG. "The effect of human influenza virus (Type A) on the incidence of lung tumors in mice." *Cancer Research*, 1950; 10: 385–392.

685. Zamai L, Ahmad M, Bennett IM, Azzoni L, Alnemri ES, and Perussia B. "Natural killer (NK) cell-mediated cytotoxicity: differential use of TRAIL and Fas ligand by immature and mature primary human NK cells." *Journal of Experimental Medicine*, 1998; 188 (12): 2375–2380.

686. Griffith TS, Wiley SR, Kubin MZ, Sedger LM, Maliszewski CR, and Fanger NA. "Monocyte-mediated tumoricidal activity via tumor necrosis factor- related cytokine, TRAIL. *Journal of Experimental Medicine*, 1999; 189 (8): 1343–1354.

687. Kayagaki N, Yamaguchi N, Nakayama M, Eto H, Okumura K, and Yagita H. "Type I interferons (IFNs) regulate tumor necrosis factor-related apoptosis-inducing ligand (TRAIL) expression on human T cells: a novel mechanism for the antitumor effects of type I IFNs." *Journal of Experimental Medicine*, 1999; 189 (9): 1451–1460.

688. Kayagaki N, Yamaguchi N, Nakayama M, Takeda K, Akiba H, Tsutsui H, Okamura H, Nakanishi K, Okumura K, and Yagita H. "Expression and function of TNF-related apoptosis-inducing ligand on murine activated NK cells." *Journal of Immunology*, 1999; 163 (4): 1906–1913.

689. Takeda K, Hayakawa Y, Smyth MJ, Kayagaki N, Yamaguchi N, Kakuta S, Iwakura Y, Yagita H, and

Okumura K. "Involvement of tumor necrosis factor-related apoptosis-inducing ligand in surveillance of tumor metastasis by liver natural killer cells." *Nature Medicine*, 2001; 7 (1): 94–100.

690. Ishikawa E, Nakazawa M, Yoshinari M, and Minami M. "Role of tumor necrosis factor-related apoptosis-inducing ligand in immune response to influenza virus infection in mice." Journal of Virology, 2005; 79 (12): 7658–7663.

691. Brincks EL, Kucaba TA, Legge KL, and Griffith TS. "Influenza-induced expression of functional tumor necrosis factor-related apoptosis-inducing ligand (TRAIL) on human peripheral blood mononuclear cells." *Human Immunology*, 2008; 69 (10): 634–646.

692. Kotelkin A, Prikhod'ko EA, Cohen JI, Collins PL, and Bukreyev A. "Respiratory syncytial virus infection sensitizes cells to apoptosis mediated by tumor necrosis factor-related apoptosis-inducing ligand." *Journal of Virology*, 2003; 77 (17): 9156–9172.

693. Sedger LM, Shows DM, Blanton RA, Peschon JJ, Goodwin RG, Cosman D, and Wiley SR. "IFN-gamma mediates a novel antiviral activity through dynamic modulation of TRAIL and TRAIL receptor expression." *Journal of Immunology*, 1999; 163 (2): 920–926.

694. Wright P. "Smallpox vaccine triggered AIDs Virus: the World Health Organisation masterminded the 13-year vaccination campaign." *London Times*, May 11, 1987.

695. Gallo RC. *Virus Hunting: AIDS, Cancer, and the Human Retrovirus: A Story of Scientific Discovery*, New Republic/Basic Books, New York, NY, USA, 1991.

696. Wyllie A. "Two worlds, one hope." *The Scotsman*, December 1, 2007.

697. Green J, and Miller D. *AIDS: The Story of a Disease*, Grafton Books, London, UK, 1986, p. 66.

698. Noireau F. "HIV transmission from monkey to man." *The Lancet*, 1987; 1 (8548): 1498–1499.

699. Konotey-Ahulu FID. "Group specific component and HIV infection." *The Lancet*, 1987; 1 (8544): 1267–1269.

700. Mulder C. "Human virus not from monkeys." *Nature*, 1988; 333: 396.

701. Fukasawa M, Miura T, Hasegawa A, Morikawa S, Tsujimoto H, Miki K, Kitamura T, and Hayami M. "Sequence of simian immunodeficiency virus from African green monkey, a new member of HIV/SIV group." *Nature*, 1988; 333: 457–461.

702. Hymes KB, Cheung T, Greene JB, Prose NS, Marcus A, Ballard H, William DC, and Laubenstein LJ. "Kaposi's sarcoma in homosexual men: a report of eight cases." *The Lancet*, 1981; 2 (8247): 598–600.

703. Centers for Disease Control and Prevention. "Kaposi's sarcoma and pneumocystis pneumonia among homosexual men: New York City and California." *Morbidity and Mortality Weekly Report*, 1981; 30 (25): 305–

308.

704. Centers for Disease Control and Prevention. "Pneumocystis pneumonia: Los Angeles." *Morbidity and Mortality Weekly Report*, 1981; 30 (21): 1–3.

705. Centers for Disease Control and Prevention. "Current trends update: acquired immunodeficiency syndrome in the San Francisco Cohort Study, 1978–1985." *Morbidity and Mortality Weekly Report*, 1985; 34 (38): 573–575.

706. Stevens CE, Taylor PE, Zang EA, Morrison JM, Harley EJ, Rodriguez de Cordoba S, Bacino C, Ting RC, Bodner AJ, Sarngadharan MG, Gallo RC, and Rubinstein P. "Human T-cell lymphotropic virus type III infection in a cohort of homosexual men in New York City." *Journal of the American Medical Association*, 1986; 255 (16): 2167–2172.

707. Centers for Disease Control and Prevention. "Current trends update on acquired immune deficiency syndrome (AIDS): United States." *Morbidity and Mortality Weekly Report*, 1982; 31 (37): 507–508, 513–514.

708. Koblin BA, van Benthem BHB, Buchbinder SP, Ren L, Vittinghoff E, Stevens CE, Coutinho RA, and van Griensven JP. "Long-term survival after infection with human immunodeficiency virus type 1 (HIV-1) among homosexual men in hepatitis B vaccine trial cohorts in Amsterdam, New York City, and San Francisco, 1978–1995." *American Journal of Epidemiology*, 1999; 150 (10): 1026–1030.

709. Harrison-Chirimuuta R, and Chirimuuta R. *AIDS and Africa: A Case of Racism vs Science?*

[Online, accessed 7th September, 2009]. URL:http://www. virusmyth.com/aids/hiv/rcafrica.htm

710. "The AIDS 'plot' against blacks." *The New York Times*, May 12, 1992.

711. Jones JH. *Bad Blood: The Tuskagee Syphilis Experiment*, Free Press, New York, NY, USA, 1981.

712. Harris R, and Paxman J. *A Higher Form of Killing: The Secret History of Chemical and Biological Warfare*, Random House, London, UK, 1982. p. 265.

713. "Living with AIDS" *National Geographic*, 2005.

[Online, accessed 7th September, 2009].

URL:http://ngm.nationalgeographic.com/ngm/0509/feature 4/map.html

714. Scott DW, and Scott WLC. *AIDS: The Crime Beyond Belief*, Trafford Publishing, Bloomington, Indiana, USA, 2008.

715. Scott DW, and Scott WLC. *The Extremely Unfortunate Skull Valley Incident: Chronic Fatigue Syndrome, Acquired Immunodeficiency Syndrome, Gulf War Syndrome, and American Warfare*, The Chelmsford Publishers, Sudbury, Ontario, Canada, 1997.

716. Cantwell A Jr. *AIDS: The Mystery and the Solution*, Aries Rising, Los Angeles, California, USA, 1984.

717. Cantwell A Jr. *AIDS and the Doctors of Death*, Aries Rising, Los Angeles, California, USA, 1988.

718. Cantwell A Jr. *The Cancer Miracle: The Hidden Killer in Cancer, AIDS and Other Autoimmune Diseases*, Aries Rising, Los Angeles, California, USA, 1990.

719. Cantwell A Jr. *Queer Blood: The Secret AIDS Genocide Plot*, Aries Rising Press, Los Angeles, California, USA, 1993.

720. Martin HV. *Was the AIDS Virus Tested on Expendable People?* FreeAmerica, 1985.

[Online, accessed 7th September, 2009]. URL:http://www.whale.to/b/martin.html

721. Horowitz LG. *Emerging Viruses: AIDS and Ebola: Nature, Accident or Intentional?* Tetrahedron, Sandpoint, Idaho, USA, 1996.

722. Burnet FM. "Men or molecules? a tilt at molecular biology." *The Lancet*, 1966; 1 (7427): 37–39.

723. Vankin J. *Conspiracies, Cover-ups and Crimes: Political Manipulation and Mind Control in America*, Paragon House, New York, NY, USA,1996. Cited in Cantwell, ibid reference 719, p. 60.

724. US Congress Appropriations Committee Hearing. *Research, Development, Testing, and Evaluation of Synthetic Biological Agents, Appropriations for 1970, Hearings Before a Subcommittee on Appropriations, House of Representatives, Ninety-First Congress, First Session*. US Government Printing Service. Reposted on the Internet by PhilFam Committee, 22 June, 1998. Transcript of July 1, 1969 of Dr Donald MacArthur, Deputy Director of Defence, Research and Engineering. pp. 129–130.

[Online, accessed 7th September, 2009]. URL:http://panindigan.tripod.com/aidsdodhear.html

725. Allison AC, Beveridge WIB, Cockburn WC, East J, Goodman HC, Koprowski H, Lambert PH, van Loghemn JJ, Miescher PA, Mimms CA,,Notkins AI, and Torrigiani G. "Memoranda: Virus-associated immunopathology: animal models and implications for human disease." *Bulletin of the World Health Organisation*, 1972; 47 (2): 257–274.

726. Strecker RB. *The Strecker Memorandum*, a video from The Strecker Group, Eagle Rock, California, USA, 1983.

727. Grote J. "Bovine visna virus and the origin of HIV." Letter, *Journal of the Royal Society of Medicine*, 1988; 81 (10): 620.

728. Matsumoto G. *Vaccine A: The Covert Government Experiment That's Killing Our Soldiers and Why GIs Are Only the First Victims*, Basic Books, Cambridge, Massachusetts, USA, 2004.

729. UK Ministry of Defence. *Background to the Use of Medical Countermeasures to Protect British Forces during the Gulf War (Operation Grandby)*, Section 42, p.8. Cited in Matsumoto G, ibid reference 728, p. 84.

730. Matsumoto G, ibid reference 728, pp. 46–55.

731. Research Advisory Committee on Gulf War Veterans' Illnesses. *Gulf War Illness and the Health of Gulf War Veterans: Scientific Findings and Recommendations*, Washington, DC, USA, November 2008.

732. Fukuda K, Nisenbaum R, Stewart G, Thompson WW, Robin K, Washko RM, Noah DL, Barrett DH, Randall B, Herwaldt BL, Mawle AC, and Reeves WC. "Chronic multisymptom illness affecting Air Force veterans of the Gulf War." *Journal of the American Medical Association*, 1988; 280 (11): 981–988. Cited in Matsumoto G, ibid reference 728, p. 119 and p. 149.

733. CNN.com. *In Depth Specials: Gulf War Facts*.

[Online, accessed 7th September, 2009].

URL:http://webarchive.org/web.20070510125644/http://edition.cnn.com/SPECIALS/2001...

734. Kang H, Magee C, Mahan C, Lee K, Jackson L, and Matanoski G. "Pregnancy outcomes among U.S. Gulf War veterans: a population-based survey of 30,000 veterans." *Annals of Epidemiology*, 2001; 11 (7): 504–511.

735. Matsumoto G, ibid reference 728, pp. 146–155.

736. *The Newport Daily News*, Virginia, USA. "Effects of anthrax vaccine downplayed." Tuesday, December 20, 2005.

[Online, accessed 7th September, 2009]. URL:http://www.gulfwarvets.com/vaccine_downplayed.htm

737. Matsumoto G, ibid reference 728, pp. 155–165.

738. Asa PB, Wilson RB, Garry RF. "Antibodies to squalene in recipients of anthrax vaccine." *Experimental and Molecular Pathology*, 2002; 73: 19–27.

739. Phillips CJ, Matyas GR, Hansen CJ, Alving CR, Smith TC, and Ryan MAK. "Antibodies to squalene in US Navy Persian Gulf War veterans with chronic multisystem illness." *Vaccine*, 2009; 27 (29): 3921–3926.

740. Wade N. "Division of Biologics Standards: The boat that never rocked." *Science*, 1972; 175 (4027): 1225–1230.

Part III

Part 3 is from my book *Cry for Health*, Chapter 3, a section on Drugs, and clinical tests. This part identifies that there are no clinical trials on vaccinations. None! This is yet another reason to reject the "no jab, no pay" proposed legislation.

I would also suggest that the Senators who are reviewing these submissions visit the Database of Adverse Event Notifications (DAEN) run by the Therapeutic Goods Administration. There you will find that between January 2000 and May 2015, a period of 15 years, Infanrix, a combined vaccine, caused 8 reported deaths (the figure on the website is 7. It's an error!!). The MMR vaccine manufactured by Merck, during the same 15-year period, reportedly killed 6 (the incorrect figure of 2 is published, which leads me to think that the TGA deliberately diminish the figures for deaths). And Boostrix, a combination of DPT and polio vaccines, killed 1. Finally, Fluavax, manufactured by CSL, killed 14.

Which means that many people have done their homework and know that vaccines are medical interventions that do indeed result in some deaths, contrary to the propaganda of the medical profession and the government. And the bias of the media compounds the problem: Rupert Murdoch's family are heavily involved. His late mother was involved with the Murdoch Institute of Research at the Childrens Royal Hospital in Mebourne and worked in collaboration with CSL/Merck. And James Murdoch, Rupert's son, was a Director of London-based GlaxoSmithKine. Not surprisingly, NewsCorp is very biased against parents who refuse to allow their children to receive vaccines. And those parents cop a huge amount of abuse from some schools, GPs and the media in general. I, as a herbal practitioner for many years, would counsel such parents and allow them to make their own minds free of abuse. Sadly, the proposed legislation thrusts the Federal Government into the realm of abuser.

To reinforce your perception of dangers that vaccines do pose for recipients, I would also recommend that you check out the Vaccine Adverse Events Reporting System (VAERS) of the USA where vaccination damages are paid out by the US Government and no one can sue any vaccine manufacturer (through an act of Congress in the mid-1980s).

The following is from Ch 3, of *Cry for Health*. Do note in this Part III, that not a single vaccine has been evaluated for its potential to cause cancer, mutagenicity (gene mutation) or infertility. And now realise that today, one in 3 women will get some form of cancer, and half of all men will get some form of cancer in their lifetimes!!!

Clinical tests

Which brings us to the most contentious issue of all: clinical testing. When a new drug is developed, it is initially tested on animals. Why? Are all creatures really the same? Can we assume that because rabbits relish the fly agaric toadstool then we can readily eat it?[11] And, because koalas thrive on eucalyptus leaves, we can do the same? Of course not. For us the toadstool is a deadly poison. And should we eat more

than one or two eucalyptus leaves our kidneys become irritated, and if we were to eat higher amounts we'd die from respiratory failure.

Similarly, whereas cats have no problems eating food contaminated with botulin toxin, we're more than likely to suffer an agonising death.[11] Morphine will sedate us as it does many animals. But as veterinary surgeons know, it sends cats into a frenzy. Again, penicillin kills many bacteria, but if given to guinea pigs it will kill them too. And arsenic kills us, but is harmless to guinea pigs, chickens and monkeys. We could go on providing example after example, but the simple fact is that one creature's food is another creature's poison.

A sad example of the absurdity of testing drugs on animals is the drug thalidomide. Scientists had subjected a host of animals, including rats, mice, rabbits, hamsters, ferrets, armadillos, pigs, dogs, cats, and primates to the drug.[12] Only occasionally did it cause malformed foetuses. On the basis of those animal tests, government regulatory bodies approved the drug for use by pregnant women who had morning sickness. As we now know, it was a human disaster.

We can conclude, as do many medical academics, that assessing a drug's safety on the basis of animal tests is not only unreliable and misleading, indeed meaningless, it is also scientific fraud.[11,13–23] As Dick Smithells—Professor of Paediatrics and Child Health at Leeds University, UK, during the 1980s, and a former member of the UK Committee on the Safety of Medicines, and the man who discovered that lack of dietary folic acid in pregnancy was linked to spina bifida—was driven to comment: 'The extensive animal reproduction studies to which all new drugs are now subjected are more in the nature of a public relations exercise than a serious contribution to drug safety.' He continued, 'The illogicality of the situation is demonstrated by the continued use of well-established drugs which are known to be teratogenic [leading to foetal abnormalities] in some mammalian species (e.g. aspirin, penicillin/streptomycin, cortisone). Conversely a new drug comes through its animal reproductive studies with flying colours may nevertheless be teratogenic in man.'[13]

Moreover, subjecting animals to the misery of laboratory testing also calls into question the assumption that our society is ethical, compassionate and intelligent.[22–24] Animal tests have nothing whatsoever to do with ensuring the safety of drugs. The real reason for animal testing is the legislative requirements that protect official regulatory bodies and corporations from legal liability should a drug later be found to be harmful.[11,15,20]

As for human tests, only about 2,000–3,000 subjects at best are tested in typical clinical trials.[25,26] Some researchers claim that at least 16,000 subjects should be tested to determine whether there is likely to be an adverse reaction in one out of 10,000 patients.[7] Moreover, people with complicated medical histories or medication regimes are often excluded from the trials, and most trials also exclude children, the elderly, and pregnant or breast-feeding women.[27] The upshot is that the human subjects taking part in tests do not represent those who will use a drug after its approval.

Marcia Angell, a former editor of *The New England Journal of Medicine*, is scathing in her criticism of pharmaceutical companies, particularly on the topic of how they conduct clinical trials in America.[28–30] As the author of *The Truth About Drug Companies: How They Deceive Us and What to Do About It*, she has documented how pharmaceutical companies design tests to guarantee favourable results for their drugs, in terms of both safety and efficacy. Any negative findings are simply not published. She has even speculated that 'perhaps most' clinical trials in the US are considered by medical critics to be 'excuses to pay doctors to put patients on a company's already-approved drug.'[31]

Equally reprehensible is the trend for drug companies to use poor countries as testing grounds for unapproved drugs, drugs that will eventually be prescribed in the wealthy West for such First World

medical conditions as obesity, high blood pressure, and raised cholesterol levels.[32–34] Angell estimates that close to half of all clinical trials today are conducted in the Third World.[32] The reason? It's cheaper, and in many respects easier and faster to conduct them there. This ploy enables drug companies to circumvent ethical committees, avoid the necessity of obtaining 'informed' consent from subjects, and bypass other drug-testing protocols stipulated by regulatory authorities in the developed world. Even more enticing to drug companies is the fact that in poor regions of the world, they can more readily get away with distorting research to make their drugs look safer and more effective than they really are.

Vaccines are not even subject to the standard double-blind, placebo-controlled tests required for every other drug. A placebo is a biologically inert substance; ingesting a sugar pill, or being injected with a sterile saline solution is to be given a placebo. But to compare an experimental group that is injected with the new vaccine with a control group that is injected with either another vaccine or with the experimental vaccine from which the biological antigens have been removed is to guarantee that harm from the experimental vaccine will never be identified.

It is little wonder that investigators at the prestigious Cochrane Collaboration, a global organisation that provides independent systematic reviews of research into various healthcare interventions, should report that for many vaccines the design of experiments and the reporting of safety outcomes was 'largely inadequate', 'scarce and incomplete', and indicated 'reporting bias' and 'lack of standardisation'; and for some vaccines tested, that 'no studies reported on adverse reactions.'[35–38]

Indeed there are no true placebo groups in any vaccine trial because the experimental vaccine is tested on one group of previously vaccinated children and compared with another group of previously vaccinated children. This is despite research indicating that vaccinations have been implicated in brain damage, meningitis, encephalitis, autism, Guillain-Barré syndrome, attention deficit/ hyperactivity disorder (ADHD), asthma, arthritis, and multiple sclerosis (see Chapter 5).

And, according to the American drug manual, the *Physician's Desk Reference*, not a single vaccine has been evaluated for its potential to cause cancer, mutagenicity (gene mutation) or infertility.[39]

Despite the controversy surrounding a possible link between autism and vaccines, and the fact that the prevalence of autism spectrum disorder in America's 8-year-old children increased by 57 per cent between 2002 and 2006 (from 1 in 150 to 1 in 110),[40] and that the estimated US national prevalence amongst 3- to 17-year-old children is now 1 in 91,[41] not a single official study has ever been conducted to compare the prevalence of autism in vaccinated versus unvaccinated children. The reason? Medical officialdom blames autism on people's genes, together with 'some environmental factor', and vaccines, it claims, are certainly not to blame. Hence the standard reply is that there are too few unvaccinated children in the US to use as a comparision.

But given that a study by the US Centers for Disease Control and Prevention found that in 2004 about 17,000 American children aged between 19- and 35-months-of-age were unvaccinated,[42] it would mean that close to 200,000 American children up to the age of 17 years would be unvaccinated; and they would provide the perfect comparison group.

One person who did dare to investigate the issue was investigative journalist Dan Olmstead, a former senior editor with United Press International in Washington, DC. Between January 2005 and July 2007 he wrote 113 reports, collectively entitled 'The Age of Autism', about his findings. And where better to find unvaccinated children than among Amish communities in which the vast majority of parents obtain exemptions on religious grounds from otherwise mandatory vaccinations.

His starting point was among the Amish community in Lancaster County, Pennsylvania. From talking to medical practitioners, public health officials and residents, and from the e-mails he received thanks to

his nationwide syndicated articles, he learned that autism was almost non-existent among Amish children; in a population of 22,000 there should have been dozens of autistic children in Lancaster County, based on the CDC's then-estimated prevalence nationwide of 1 in 166.[43–46] But Olmstead had heard of only three, possibly four cases, and tellingly at least two of them had been vaccinated.

Olmstead then turned to Ohio, the state with America's largest Amish population. He learned from Dr Heng Wang, medical director, physician and researcher at the Das Deutsch Center Clinic for Special Needs Children, in Middlefield, Ohio, that the prevalence of autism in the Amish community in that area of north-eastern Ohio, was 1 in 15,000, literally—there was only one child, a boy, with autism in a population of 15,000, and ominously he had received routine immunisations.[47] Olmstead unearthed similar patterns from his investigations among the Amish populations of Kentucky and Indiana.

Chicago was Olmstead's next port of call. There, many of the city's parents took advantage of Illinois' relatively permissive immunisation policy to avoid having their children vaccinated. Perhaps not surprisingly, the incidence of autism in Illinois in 2005 was 1 in 263, 37 per cent lower than the prevalence in the rest of the nation.[48] Olmstead learned from Dr Mayer Eisenstein, medical director of Homefirst Medical Services in Chicago—a large practice that provides a service to 30,000 to 35,000 children, many of whom have never been vaccinated—that he was aware of only one case of autism (and one case of asthma) amongst any unvaccinated children.

In essence, Olmstead had discovered that among America's Amish population of approximately 100,000 people, there were fewer than 10 cases of autism. [49] No, it wasn't a scientific study, but it did unearth something that regulatory watchdogs refused to investigate … and still refuse to investigate to this very day.[50,51] It's an issue that would never have arisen in the first place if there had been rigorous clinical trials on vaccines.

But there is one organisation that did investigate the issue, and it wasn't a government agency. Rescue Generation, a public advocacy organisation founded by parents of children with such neurological disorders as ADHD and autism, commissioned a telephone survey in 2006 to study the prevalence of neurological disorders among 17,600 4- to 17-year-old children in nine counties of California and Oregon; 991 of the children were described as never having been vaccinated.[52]

Just as many parents with neurologically damaged children had suspected, the survey did indeed find a strong correlation between neurological disorders and vaccinations. Vaccinated boys aged 4 to 17 years, were two-and-a-half times (155 per cent) more likely to have neurological disorders compared with unvaccinated boys of the same age. The risk of having ADHD was 224 per cent higher in vaccinated boys, and the risk of having autism was 61 per cent higher. But the 11- to 17-year-old boys fared far worse. They were 317 per cent more likely to have ADHD than their unvaccinated counterparts, and 112 per cent more likely to have autism.

That's certainly something many American parents might have wanted to have known before they had had their children vaccinated, given that 1 in 91 American children today have autism,[41] and that 1 in 13 have ADHD;[53] and that for 16-year-old boys the figure for ADHD is 1 in 7.[53]

REFERENCES

11. Editorial. "Drug industry situation uncertain but hopeful." *Journal of the American Medical Association*, 1964; 187 (11): 35. Quoting a speech made by James D Gallagher, director of Medical Research at Lederle Laboratories, at a conference presented by the American College of Neuropsychopharmacology in

Washington, DC, on January 16, 1964.

12. Schardein JL. *Drugs as Teratogens*, CRC Press, Cleveland, Ohio, USA, 1976.

13. Smithells RW. "Drug teratogenicity." In: WHW Inman (editor), *Monitoring for Drug Safety*, MTP Press, Lancaster, England, UK, 1986; pp. 383–390.

14. Bross I. "Animals in cancer research: a multi-million dollar fraud", *Fundamental and Applied Toxicology*, 6 November, 1982.

15. Hawkins DF (editor). *Drugs and Pregnancy: Human Teratogenesis and Related Problems*, D.F. Hawkins (editor), Churchill Livingstone, New York, NY, USA, 1983.

16. Lorke D. "A new approach to acute toxicity testing." *Archives of Toxicology*, 1983; 54: 275–287.

17. McLachlan JA, Pratt RM, and Markert CL. *Developmental Toxicology: Mechanisms and Risks (The Banbury Report)*, Cold Spring Harbor Laboratory, Cold Spring Harbor, NY, USA, 1987; p. 313.

18. Sharpe R. *The Cruel Deception: The Use of Animals in Medical Research*, Thorsons Publishing Group, Wellingborough, Northamptonshire, UK, 1988.

19. Croce P. *Vivisection or Science–A Choice to Make*, CIVIS (International Centre of Scientific Information on Vivisection) Publications, Lugano, Switzerland, 1991.

20. Reusch H. *Naked Empress or the Great Medical Fraud*, CIVIS, Zurich, Switzerland, 1982.

21. Brent RL. "Utilization of animal studies to determine the effects and human risks of environmental toxicants (drugs, chemicals, and physical agents)." *Pediatrics*, 2004; 113 (4): 984–995.

22. Bailey J, Knight A, and Balcombe J. "The future of teratology research is in vitro." *Biogenic Amines*, 2005; 19 (2): 97–146.

23. Knight A, Bailey J, and Balcombe J. "Animal experiments harm human health." *American Chronicle* [Online newspaper], 8 October 2005.

    [Online, accessed 15th July, 2009].

    URL:http://www.americanchronicle.com/articles/viewArticle.asp?artcleID=2819

24. Balcombe J, Barnard N, and Sandusky C. "Laboratory routines cause animal stress." *Contemporary Topics in Laboratory Animal Science*, 2004; 43 (6): 42–51.

25. Yusuf S, Collins R, and Peto R. "Why do we need some large and simple randomized trials?" *Statistical Medicine*, 1984; 3: 409–420.

26. Altman DG. "Size of clincal trials." *British Medical Journal* (Clinical Research Edition), 1983; 286 (6381): 1842–1843.

27. Van Spall HGC, Toren A, Kiss A, and Fowler RA. "Eligibility criteria of randomized controlled trials published in high-impact general medical journals." *Journal of the American Medical Association*, 2007; 297 (11): 1233–1240.

28. Relman AS, and Angell M. "America's other drug problem: how the drug industry distorts medicines and politics." *The New Republic*, December 16, 2002, pp. 27–41.

29. Angell M. *The Truth about the Drug Companies: How They Deceive Us and What to Do About It*, Random House, New York, NY, USA, 2004.

30. Angell M. "Drug companies and doctors: a story of corruption." *The New York Review of Books*, January 15, 2009; 56 (1): 8–12.

31. Angell M. Ibid reference 29, p. 30.

32. Angell M. "The body hunters." *The New York Review of Books*, October 6, 2005; 52 (15): 23–25.

33. Angell M. "The ethics of clinical research in the third world." *The New England Journal of Medicine*, 1997; 337 (12): 847–849.

34. Shah S. *The Body Hunters: Testing New Drugs on the World's Poor*, The New Press, New York, NY, USA, 2006.

35. Demicheli V, Jefferson T, Rivetti A, and Price D. "Vaccines for measles, mumps and rubella in children." *Cochrane Database of Systematic Reviews*, 2005; 4 (CD004407).

36. Jefferson T, Rivetti A. Harnden AR. Di Pietrantonj C, and Demicheli V. "Vaccines for preventing influenza in healthy children." *Cochrane Database of Systematic Reviews*, 2008; 2 (CD004879).

37. Macartney K, and McIntyre P. "Vaccines for post-exposure prophylaxis against varicella (chicken pox) in children and adults." *Cochrane Database of Systematic Reviews*, 2008; 3 (CD001833).

38. Soares-Weiser K, Goldberg E, Tamimi G, Leibovici L, and Pitan F. "Rotavirus vaccine for preventing diarrhoea." *Cochrane Database of Systematic Reviews*, 2004; 1 (CD002848).

39. *Physicians' Desk Prescriber*, 61st Edition, Thomson, Montvale, New Jersey, USA, 2007; pp. 819, 1405, 1426, 1452, 1457, 1478, 1550, 1596, 1902, 1915, 1930, 1986, 2007, 2020, 2032, 2037, 2065, 2073, 2076, 2098, 2102, 2111, 2947, 2951, 2956, 3427, and 3467.

40. US Centers for Disease Control and Prevention. "Prevalence of autism spectrum disorders; Autism and Developmental Disabilities Monitoring Network, United States, 2006." *Morbidity and Mortality Weekly Report*, December 18, 2009; 58 (SS10): 1–20.

41. Kogan MD, Blumberg SJ, Schieve LA, Boyle CA, Perrin JM, Ghandour RM, Singh GK, Strickland BB, Trevathan E, and Van Dyck PC. "Prevalence of parent-reported diagnosis of autism spectrum disorder among children in the US, 2007." *Pediatrics*, 2009; 124 (9): 1395–1404.

42. Smith PJ, Chu SY, and Barker LE. "Children who have received no vaccines: who are they and where do they live?" *Pediatrics*, 2004; 114 (1): 187–195.

43. Olmsted D. "The age of autism: the Amish anomaly." United Press International, April 18, 2005.

    [Online, accessed 11th January, 2010].

    URL:http://www.upi.com/Science_News/2005/04/19/The-Age-of-Autism-The-Amish-anomaly/UPI-95661113911795/

44. Olmsted D. "The age of autism: Julia." United Press International, April 19, 2005.

    [Online, accessed 11th January, 2010].

    URL:http://www.upi.com/Science_News/2005/04/19/The-Age-of-Autism-Julia/UPI-55491113918060/

45. Olmstead D. "The age of autism: absence of evidence." United Press International, May 9, 2005.

    [Online, accessed 11th January, 2010].

    URL:http://www.upi.com/Science_News/2005/05/09/The-Age-of-Autism-Absence-of-evidence/UPI-50051115667435/

46. Olmstead D. "The age of autism: witness." United Press International, May 10, 2005.

    [Online, accessed 11th January, 2010].

    URL:http://www.upi.com/Science_News/2005/05/10/The-Age-of-Autism-Witness/UPI-36431115734474/

47. Olmstead D. "The age of autism: one in 15,000 Amish." United Press International, June 8, 2005.

    [Online, accessed 11th January, 2010].

    URL:http://www.upi.com/Science_News/2005/06/08/The-Age-of-Autism-One-in-15000-Amish/UPI-74721118251747/

48. Olmstead D. "The age of autism: 'a pretty big secret". United Press International, December 7, 2005. [Online, accessed 11th January, 2010].

    URL:http://www.upi.com/Health_News/2005/12/07/The-Age-of-Autism-A-pretty-big-secret/UPI-68291133982531/

49. Olmstead D. "The age of autism: Amish ways." United Press International, June 6, 2005.

    [Online, accessed 11th January, 2010].

URL:http://www.upi.com/Science_News/2005/06/The-Age-of-Autism-Amish-ways/UPI-80061118082873/

50. Olmsted D. "The age of autism: 'Amish bill' introduced." United Press International, July 28, 2006.

[Online, accessed 11th January, 2010].

URL:http://www.upi.com/Health_News/2006/07/28/The-Age-of-Autism-Amish-bill-introduced/UPI-35321154110819/

51. Olmstead D. "The age of autism: the last word." United Press International, July 18, 2007.

[Online, accessed 11th January, 2010].

URL:http://www.upi.com/Health_News/2007/07/18/The-Age-of-Autism-The-last-word/UPI-57611184777255/

52. Generation Rescue/SurveyUSA. *Cal-Oregon Vaccinated vs. Unvaccinated Survey*, Generation Rescue, Sherman Oaks, California, USA, and SurveyUSA, Verona, New Jersey, USA. Press release: Portland, Oregon, September 25, 2007.

[Online, accessed 11th January, 2010]. URL:http://www.GenerationRescue.org/survey.html

53. US Centers for Disease Control and Prevention. "Mental health in the United States: prevalence of diagnosis and medication treatment for attention-deficity/hyperactivity disorder; United States, 2003." *Morbidity and Mortality Weekly Report*, September, 2005; 54 (34): 842–847.

Part IV

Part 4 is from Chapter 4 of *Cry for Health*, and deals with the real reason for the decline in infectious diseases…not vaccinations, but improvements in sanitation and hygiene.

Why the death rate declined

Why did these diseases decline? Scholars believe that higher living standards, especially improved nutrition, enhanced people's resistance to infections.[11-15]

The quality, variety and amounts of foods available had increased following improved trade and the introduction of a new method of crop rotation. The latter had replaced the earlier method of three-crop rotation and a year of lying fallow, with a four-year crop rotation that advocated the successive planting of root vegetables, legumes, barley and wheat.

The squalor, filth and poverty of the densely populated cities of Europe and North America, however, continued to provide the ideal breeding ground for such diseases as cholera and tuberculosis, as well as for childhood infections. Not until the late 1800s, after the introduction of public sanitation methods for sewage disposal, drainage, water supply, food storage and building ventilation, did the

epidemics of cholera cease, the incidence of tuberculosis decline, and infant mortality rates decrease sharply.[3,14]

The introduction of hygienic birthing and surgical practices, as well as practices introduced by New Zealand's trailblazer in neonatal care, Dr Truby King, drastically reduced infant mortality rates during the first half of the 20th century.

According to the British Association for the Advancement of Science, childhood diseases decreased 90 per cent between 1850 and 1940, well before mandatory vaccination programmes.[16] Infectious diseases, including measles, diphtheria and whooping cough, continued to decline up until the 1950s, after which they levelled out and remain much the same today.[9] Neither antibiotics, which were developed in the 1940s, nor vaccination programmes, which reached only a small percentage of the population, can take credit for the reduction in communicable diseases.[17] Even the polio epidemics, which peaked in Britain in 1950, had already declined by 82 per cent when the Salk vaccine was introduced in 1956.[18]

Nor has modern medicine been a major factor in the decline in disease and death rates in Third World countries in recent years. According to a recent World Health Organisation report, the decline has been the result of improved standards of sanitation, hygiene and diet.[19] Quite simply, infectious diseases thrive where there is overcrowding, filth, poverty, wars and famine. Improve the social, economic, nutritional and sanitary conditions and such diseases rapidly decline.

REFERENCES

11. Dubos R. *Mirage of Health: Utopias, Progress and Biological Change*, Harper & Row, New York, NY, USA, 1959, pp. 88-89.

12. Illich I. *Limits to Medicine: Medical Nemesis, the Expropriation of Health*, Marion Boyars, London, UK, 1976, pp. 23–30.

13. McKeown T. *The Role of Medicine: Dream, Mirage or Nemesis*, Princeton University Press, Princeton, New Jersey, USA, 1979, pp. 29–44.

14. Mc Keown T. *The Modern Rise of Population*, Edward Arnold, London, UK, 1976.

15. McKeown T, Brown RG, and Record RG. "An Interpretation of the Modern Rise of Population in Europe." *Population Studies*, 1972; 26 (3): 345–82.

16. RR Porter. "The contribution of the biological and medical sciences for human welfare." Presidential Address to the British Association for the Advancement of Science, Swansea Meeting, 1971 (London, UK: The Association, 1972) pp. 95–97.

17. Pollard JH. "Morbidity and Longevity." In: Ross JA (editor), *International Encyclopedia of Population, Vol 2*, The Free Press, New York, NY, USA, 1982, pp. 452–459.

18. Coulter HL. *The Medical Assault on the American Brain: Vaccination, Social Violence, and Criminality*, North

Atlantic Books, Berkeley, California, USA, 990, pp. 83 and 101.

19. Rutstein SO. "Factors associated with trends in infant and child mortality in developing countries during the 1990s." *Bulletin of the World Health Organization*, 2000; 78 (10): 1256–1270.

Yours sincerely,

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