# VACCINATION

## 100 Years of Orthodox Research shows that Vaccines Represent a Medical Assault on the Immune System

Viera SCHEIBNER Ph.D.

This book is dedicated to those babies and their parents
who suffered from vaccination

*Boldness has genius, power and magic in it* — Goethe

# ACKNOWLEDGEMENTS

This project has made me acutely aware that EVERY CHILD IS MY CHILD. That I have to share my knowledge on the subject of childhood vaccination with all parents, to show them the dangers and ineffectiveness of vaccines.

The Cotwatch breathing monitor, developed by Leif Karlsson, sounded the alarm both literally and figuratively to warn of the stress, often fatal, that vaccines caused in babies. Tragically, none of those who should have seen the connection, who should have understood, was prepared to admit the clinical significance of the Cotwatch alarms, so babies kept on dying. Although it was not our original intention, Leif and I had to undertake basic research into the patterns of babies' breathing using Cotwatch, and later a micro-processor controlled Cotwatch breathing monitor.

The resistance we encountered in pursuing this research, in having its significance recognised, became the best and most effective goad to us to continue.

So I wish to thank all those pædiatricians and other health professionals who resisted or opposed our endeavours, who listened to us but did not seem to hear and who would not speak out against the silent killer of babies.

There were a few who felt we were on the right track; they hoped we would win without their help, so I thank them too.

And at the last, but not the least, I thank with true sincerity all those who silently or openly helped us, who kept us going, often financially, allowing us to continue this project against all odds.

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# FOREWORD

"I do not believe in Modern Medicine. I am a Medical Heretic ... I haven't always been a Medical Heretic; I once believed in Modern Medicine."

*Dr Robert Mendelsohn*: **Confessions of a Medical Heretic**

Fifteen years ago, when I graduated from medical school, I would have been surprised to be writing a foreword to a book against vaccination. I was a conservative orthodox doctor just off the medical school production line. I have changed greatly. My ideas have changed. Like Dr Mendelsohn I have also become a medical heretic. The more I read and study, the more I believe that medicine is heading in the wrong direction.

I am not denying that modern medicine has done some marvelous things – acute emergencies and trauma management are unparalleled, but that is where its usefulness ends. Modern medicine is trying to control nature. This is totally wrong – we should be working with nature.

Medicine today is controlled partly by Government bureaucracy but mostly by the influence of the pharmaceutical multi-national corporations. No wonder that any move away from the status quo is being resisted.

This book has come at a critical time in the history of medicine. A time of change. The population is now better educated, more informed and making their own decisions about their own health. This change, this revolution, is not from the top but from the base, that is, the people, and it is slowly filtering upwards.

People are no longer blindly accepting what is told to them by doctors. More are asking questions, more are gaining information, more and more are turning towards natural therapies (working with nature) because of disillusionment with orthodoxy.

Vaccination, until recently, was a one-sided story: "Vaccinate or die" seemed to be the catch-cry of the orthodoxy. Some began to question this.

There was a gut feeling amongst many that vaccination was not as safe or as effective as claimed.

This book is the culmination of years of research. Dr Viera Scheibner has hunted through thousands of articles, read between the lines, reviewed the raw data – all of this information published in orthodox medical journals – and has found facts that piece together to form a terrifying picture. What was thought to be safe – isn't.

This book has the references to back the case against vaccination.

Any parent who is concerned about the safety and effectiveness of vaccination, and who is concerned about the welfare of their children, should read this book. It is ultimately the parents of the children who should decide whether to vaccinate or not. This decision must be made only when the parents are fully informed.

After reading this book, I feel that more and more people will decide against vaccination.

Peter Baratosy, M.B.,B.S.

# AUTHOR'S FOREWORD

On 12th October 1985 my life changed profoundly. On that day I met Leif Karlsson, a biomedical electronics engineer specialising in patient monitoring systems. After only a few hours' acquaintance, on learning of his professional specialty, I asked him to develop a breathing monitor for babies.

He said yes.

One year and one day later, the first Cotwatch went to the first parents wishing to monitor their newborn baby's breathing. We had decided to rent the first 150 units and to keep in close contact with the parents who used them. Soon, some twenty units were out there working and some time later parents started ringing us to report that the Cotwatch was sounding alarms.

A few questions soon established that alarms occurred at certain hours while babies were deeply asleep. Clusters of five to seven short alarms sounded within about a 15 minute period. These occurred after the baby had been exposed to stress, or happened a day or two before the child went down with a common cold or cut its first tooth. An important fact about the vast majority of these alarms was that the babies had not actually stopped breathing, but, rather, were breathing very shallowly. In most cases no intervention was needed to interrupt the type of breathing that triggered the alarms as the babies spontaneously resumed normal (deeper) breathing.

All new parents who monitored with Cotwatch were given a questionnaire on which to record all alarms for two weeks. By chance, 28 of the monitored babies were 'near-miss' (babies who stopped breathing, were found in time and successfully resuscitated). A further 22 newborn babies were monitored by choice of the parents.

Records by parents of near-miss babies showed substantially higher numbers of alarms compared with the number of alarms reported for newborn babies. We realised that the alarms were an important indicator of stress level in the babies.

We concluded that when babies are under stress (whether due to insult or while cutting teeth or incubating illness), their breathing changes to what we named the stress-induced breathing pattern and they experience episodes of low-volume breathing in clusters at critical hours while asleep .

We could not find a paediatrician who would undertake independent research to elucidate and further develop our ideas, based on the initial observations with Cotwatch, so we decided to do the necessary data collection and research ourselves.

It was a long and rocky road to travel. One task was development of a microprocessor-based breathing monitor, to ensure that the data collection was completely objective and scientific. It took over six months' full-time work before we were able to produce computer records of babies' breathing.

Without endeavouring to do so specifically, we recorded the breathing of babies before and after they were vaccinated. The pattern of breathing that emerged over the days and weeks was extremely interesting and highly significant. It showed that babies' breathing was affected in a certain characteristic manner and over a long period of time following diphtheria-pertussis-tetanus (DPT) injections.

At this time, (1988), we did not know that the merits of vaccination were being hotly debated and we did not know that a lot of evidence on its dangers and ineffectiveness had been published in very reputable medical journals. We saw only that DPT vaccinations caused babies a lot of stress, reflected in sometimes major flareups of stress-induced breathing over a period of at least 45 to 60 days following the injections. The dynamics of these flareups showed a remarkable uniformity: even though the amplitude of the flareups differed, the days on which they occurred following injection were the same.

Paediatricians to whom we showed our first records pointed to the arrow indicating day zero (when DPT injection was administered), and commented without hesitation: 'This is the cause". Then, pointing to the summaries of stress-induced breathing patterns over

several days, each unfailingly followed with 'This is the effect". They, of course, knew that the day zero arrow indicated the DPT injection.

We also learned from parents who monitored a subsequent child after a cot death that most commonly the previous child had died after DPT injection. We realised that a great number of cot deaths followed DPT injections and we felt we had to address the issue. However, when we approached a few paediatricians with this observation and conclusion, we realised that we had touched a very sensitive and contentious issue. Once again, we were forced to start our own search for the truth.

Several years later I had collected just about every publication written on the subject of the effectiveness and dangers of vaccines. Supported by data from our continuing research with the Cotwatch breathing monitor, I decided to write a concise and brief summary of my literature search, reviewing the many thousands of pages of scientific journals and other publications I had studied.

I did not find it difficult to conclude that there is no evidence whatsoever that vaccines of any kind — but especially those against childhood diseases — are effective in preventing the infectious diseases they are supposed to prevent. Further, adverse effects are amply documented and are far more significant to public health than any adverse effects of infectious diseases.

Immunisations, including those practised on babies, not only did not prevent any infectious diseases, they caused more suffering and more deaths than has any other human activity in the entire history of medical intervention. It will be decades before the mopping-up after the disasters caused by childhood vaccination will be completed. All vaccination should cease forthwith and all victims of their side-effects should be appropriately compensated.

Dr Viera Scheibner
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17. 5. 1993.

# INTRODUCTION

If you raised the subject of immunisation with any medical doctor they would probably tell you that vaccination is the most effective intervention of modern medicine which prevented more suffering and saved more lives than any other medical procedure.

They would also tell you that the demise of epidemic diseases like small pox or polio is one of the success stories of mass vaccination programmes. However, this claim is totally unsubstantiated.

The documented truth is that the incidence of and mortality from any infectious diseases which used to decimate populations of Europe only some one hundred years ago declined by up to 90% before any vaccine has ever been used in mass proportions. Also, diseases, like bubonic plague or scarlet fever disappeared without any vaccination programmes at all. The mortality from the dreaded diphtheria declined decades before *Corynebacterium diphtheriae* had even been discovered and isolated.

Immunization against diphtheria was introduced in 1932-35 and on a mass scale in 1940, by which time the annual death rate was negligible (less than 300 deaths per million). It is amply documented in medical literature that this mass vaccination was followed by unprecedented diphtheria epidemics – in fully vaccinated subjects.

The 1940s saw also the introduction of mass vaccination against tetanus and whooping cough which in many countries, including Australia, lead to outbreaks of the so-called provocation poliomyelitis.

In 1950 Dr McCloskey published evidence that there indeed was an association between administration of pertussis and/or pertussis-diphtheria toxoid and provocation poliomyelitis within one to ninety days after the injections. The majority of paralyses occurred in the inoculated limb. Leake in England reported cases of poliomyelitis closely following pertussis vaccination administered within days before the onset of the symptoms. This is the same famous polio epidemic of 1949-50 which is used to push parents into

vaccinating their children especially against polio. The provocation poliomyelitis is a well-known phenomenon which may follow administration of any vaccine, but especially DPT and polio. It is officially admitted that all cases of polio in the US, since the introduction of the vaccine, are caused by the vaccine. The same has been seen in Australia and other countries like England. So the occurrence of the same phenomenon all around the world would be asking too much of coincidence.

The truth about polio and smallpox vaccines is that they are heavily contaminated with animal viruses, being produced on monkey kidneys and calves respectively. This gave us AIDS which started in central eastern Africa in those states where the WHO conducted the eradication campaign against smallpox and polio. The batches of vaccines used here were heavily contaminated with both SV 40 and SIV (Simian Immuno-deficiency Virus) and bovine retrovirus, another AIDS-related virus. One syringe was used on 40 to 60 people and contributed to the spread of AIDS to hundreds of thousands of innocent unsuspecting people. It is beyond coincidence that the present raging epidemic of AIDS is affecting mostly those states where the polio/smallpox eradication campaign was conducted.

It should not come as a surprise that a new syndrome of immune incompetence or immuno-suppression developed in babies too. High incidence of child leukæmia and cancer has been linked to vaccines by many authors who attributed this to inappropriate antigenic stimulation provided by vaccines and to the presence of contaminating SV40 virus. Respiratory syncytial virus, or more befittingly, the chimpanzee coryza virus, causes lingering upper and especially lower respiratory tract diseases in babies. These are only the viruses which were discovered and are now, perhaps, looked for. What about the myriad of other, unknown animal viruses lurking in the vaccines?

It has also been documented that vaccine against tuberculosis had no impact whatsoever on the incidence of the disease, which is essentially a disease of malnutrition and overcrowding.

The best evidence of ineffectiveness of vaccines comes from two facts: firstly, such deadly diseases as bubonic plague disappeared without any immunisation programmes, simply because of better sanitation and nutrition and uncrowded life styles and secondly, the countries which do not vaccinate against certain diseases, like pertussis, report amelioration of the disease and the incidence which compares favourably with the incidence of whooping cough in those countries which claim an almost complete pertussis vaccination cover. Hamburg in Germany has enjoyed freedom from vaccination push since 1962 without the incidence of infectious disease exceeding the incidence in countries that claim more than 90% vaccination compliance.

In 1975 Japan raised the minimum vaccination age to two years; this was followed by the virtual disappearance of cot death and infantile convulsions. Since the eighties, after allowing vaccination of 3-month and older babies, the incidence of cot death in Japan has increased. Sweden stopped vaccinating against whooping cough in 1979, due to ineffectiveness of the whole-cell vaccine and adverse effects which far exceeded the adverse effects of the whooping cough illness. After trialling two Japanese acellular pertussis vaccines, Sweden rejected these also, and for the same reasons.

While studying thousands of pages written on vaccines I have not found a single paper which would demonstrate that in epidemic situations only unvaccinated children contracted the diseases. Even during vaccine trials many children contracted the diseases against which they were vaccinated, often within a few days. Although the initial target of all vaccination programmes was to eradicate the infectious diseases like whooping cough, polio and measles, when it became all too painfully clear that it is an unrealistic goal, the proponents of vaccination started telling parents and the public that at least the vaccines alleviate the disease.

Not even this is true. Not only diseases like whooping cough can affect seriously both vaccinated and unvaccinated children (based on hospital admissions), but there is a new disease – atypical measles –

which is an especially vicious form of measles only affecting vaccinated children and with a considerable mortality rate.

After studying the extensive literature demonstrating ineffectiveness of vaccines and their dangers, I concluded that the call for suspension of all vaccination programmes is now inevitable.

Instead of relying on a "magic bullet" (one injection solves it all), the orthodoxy should start learning the dynamics and importance of infectious diseases and effective treatment. It is absurd to set out to eradicate infectious diseases which play an important role in the maturation of the immune systems of our children.

It has been documented in medical literature that people who contracted cancer and other chronic degenerative diseases in later years have remarkably few infectious diseases of childhood to report. A proper development of rash during such infectious diseases as measles is apparently important for the prevention of cancer and other serious diseases in later life.

The sordid story of vaccination programmes reveals the enormous gaps in the knowledge base of the orthodox medical establishment, especially a profound lack of knowledge of the dynamics of health and disease and functioning of the human body. It is this same medical industry which enjoys the protection of the institutions of the State in most industrially developed countries.

The attention of medical professionals and the State should turn to such scientific medical systems as homeopathy which is not only based on sound knowledge of human physiology but also on a profoundly scientific knowledge of the healing processes and the testing of thousands of specific remedies.

It should concern us all that scientific healing systems like homeopathy or naturopathy enjoy a substantially higher rate of success and a substantially lower rate of side-effects from their remedies than do those of allopathic medicine. The cost effectiveness of these, today still called 'alternative', medical systems is another good reason for the State to look seriously into them as viable alternatives to play an important part in the national health system.

## The vaccination hypothesis — How are the vaccines supposed to work?

According to orthodox medicine, the purpose of vaccination is to eradicate infectious diseases. These diseases are considered bad and a nuisance rather than the way nature primes and challenges the immature immune systems of our children. "Measles is misery" scream the posters in doctors' surgeries and try to tell you that one injection will do away with the problem. They don't tell you that measles and other infectious diseases of childhood have an important role to play.

A group of Swiss medical doctors formed an action group which questions the MMR (measles, mumps, rubella) vaccination of children. In their 18 page document they write that in a famous paediatric clinic in Basel until recently (1969) they used to induce measles in children with serious renal diseases (nephrotic syndrome) as means of healing or at least improving substantially the condition.

Auto-immune diseases like asthma, lupus erythematosus or excema also either disappeared or greatly improved after the child contracted and overcame measles.

They also questioned the wisdom of relentlessly trying to suppress natural expressions like fever instead of recognising its importance in the natural healing process. Also, infectious diseases represent important developmental milestones in children. Perhaps the most important of all good reasons to accept the infectious diseases of childhood is a well-documented fact that the immune system must be primed and challenged in young individuals if it is to function properly and protect the individual against the far worse auto-immune diseases of later life, such as cancer.

All medical systems — except orthodox or allopathic medicine— look at the human (and animal) body as a whole and interconnected system. Homeopathy understands disease as a need of the body to rid itself of toxins and it does so in an orderly and meaningful fashion. Although homeopathic science looks at individual symptoms for guidelines in understanding the diseases and selecting a remedy, it does not attempt to suppress the symptoms, rather initially

accentuates the symptoms to enhance the natural healing efforts and mechanisms of the body.

Hering's 'law' holds that as a disease passes from an acute to a chronic form the symptoms move from the surface of the body to the interior, from the lower part of the body to the upper and from the less vital organs to the more vital ones. Under correct (homeopathic) treatment this movement is reversed and the symptoms move from the more vital organs to the less vital, from the upper part of the body to the lower, and from the interior to the skin. This is also true for the movement of symptoms in acute disease. In cases of the so-called fixed miasmic diseases, like measles, the rash first appears on the forehead and moves onto the trunk and extremities. In contrast, the rash of atypical measles in vaccinated children first appears on the extremities, moves to the trunk and attacks the lungs and other internal organs.

Vaccination, by introducing viruses directly into the blood stream, far from preventing diseases, actually pushes the disease into a chronic form and deeper into the body where it then attacks vital organs. The results of suppressing measles and other infectious diseases in this manner are cancer and other auto-immune and chronic diseases.

Medical assessment of alleged effectiveness and efficacy of vaccines centres around the production of antibodies. Modern immunologists studying the biologic significance of the secretory gamma A immunoglobulins hold that immunity is classically concerned with resistance to infection. This is based on the well-known fact that individuals who recover from an infectious disease almost never succumb to the same disease again. Today we know that the functions of the immune system are more diverse and include not only defense but also homeostasis and surveillance.

In the vertebrates (which group includes humans as we too have a backbone), a diverse cell system has developed — the lympho-reticular system which is distributed throughout the body and lines the lymphatic and vascular systems. Its cells occur within the thymus, lymph nodes and spleen, forming an internal secretory

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system and an external secretory system in those body tracts exposed to the exterior — the respiratory, gastrointestinal and genitourinary systems.

The tissues of the lympho-reticular system contain a variety of cells performing separate functions, either directly or through producing a variety of antibodies. These are activated by a variety of influences recognised as foreign by the host. The internal secretory system produces serum immunoglobulins. Of these, the gamma G immunoglobulins are a major, indeed a predominant, part. The external secretory system produces a specific group of antibodies — the secretory gamma A immunoglobulins. The precise function of the secretory component is not understood.

A study evaluating the relative roles of serum and nasal antibody in protecting against parainfluenzæ type 1 infection showed that the nasal antibody played a very important part — much higher than the serum antibodies. This casts doubt on the importance of the serum antibodies produced following vaccination, and hence doubt on the conferring of immunity. Indeed, we see this proven when vaccinated children contract the diseases against which they have been vaccinated.

Vaccinated children commonly exhibit a deranged immunological response by developing atypical measles, mumps and possibly many other atypical manifestations of the diseases targeted by vaccines. It is far better, then, to allow the natural processes to proceed without harmful interference.

# 1

# HEPATITIS B VACCINES: Babies are accessible

It is becoming more and more obvious that, instead of healing people, the orthodox medical system is creating more and more diseases — all of which seem to be deadly. Since the development of an almost fanatical preoccupation with 'prevention' of infectious diseases by vaccination, all of these diseases, even chicken pox, 'flu and measles, have become deadly diseases.

**'If you don't vaccinate your child, it will die!'** is often uttered at bewildered parents.

Recently, hepatitis B became a declared deadly disease which is poised to attack all newborn babies, or is going to attack them after they all become intravenous drug users, prostitutes, practicing male homosexuals or hæmodialysis patients. Some time ago, the system was going to screen all mothers to determine if they were carriers of the 'deadly' virus. Then attention shifted to 'ethnic' mothers.

All of a sudden there is now no screening, no selectivity — somehow every mother in Australia is now to be regarded as a carrier and all newborn babies are to be injected with a vaccine containing the deadly virus. And deadly virus it is.

As is true of all foreign antigens, hepatitis B vaccine affects the newborn liver, which may become dysfunctional for 14 days or more after the injection. When the first DPT and polio vaccines are injected, the antigenic insult is too much for a number of babies, but with a convenient and ever-present waste basket called cot death handy to explain the mortality, these deaths are not a problem. Not for the system, anyway.

Just for the unsuspecting parents and families of these unfortunate infants.

Who, then, are in the hepatitis B high risk bracket?

Data from medical literature indicate that the groups at highest risk of contracting hepatitis B are: prostitutes, sexually active homosexual men, intravenous drug users, institutionalised children and adults, some health workers, the military and people on hæmodialysis. Other people have only a minute chance of coming into contact with and contracting hep B.

The average health worker's chance of contracting hep B is very small and does not warrant general vaccination. However, even if it were true that doctors were among the high risk group, they would be the first to reject the vaccine as they rejected rubella vaccine — a fact well documented in the medical literature.

Dienstag and Ryan (1982) did a sero-epidemiological survey of 624 health workers and found that frequency of hepatitis B serologic markers increased as a function of contact with blood, previous hepatitis history, years of occupation and age, but not as a function of contact with patients. The authors concluded that in the setting of continuous, low intensity exposure to hepatitis B, health workers may become naturally immunised with hepatitis B surface antigen rather than infected with hepatitis B.

If hepatitis B were such a general problem as is often painted by proponents of vaccination, then the incidence of hepatitis B would be enormous. We all know that this is simply not true. With these facts to hand, one has seriously to question the motivation behind hep B vaccination promotions, especially since they are based on scare tactics. Viral hepatitis is associated with at least three and probably four different viruses, of which hepatitis A (infectious hepatitis) and hepatitis B (serum hepatitis) are the most important. There are also non-A and non-B hepatitis associated with different viruses.

Hepatitis infection, as with any other infection, represents a problem for blood transfusion services and also for individuals, since in a small number of cases the infection may progress to chronic active hepatitis which may progress to cancer.

In some areas in Africa, Asia and the Pacific as many as 20% of the population may be carriers, but in Europe, North America and Australia, the situation is very different — carriers represent only 0.1% or less of the population. However, there are major differences in hep B infection in different ethnic groups and the incidence of hep B in a variety of different nationalities cannot be generalised. As Yodfat *et al.* (1982) demonstrated, Indians and Pacific Islanders living in Israel are among the groups with the lowest incidence of hep B and hep A.

Blood, saliva, seminal fluid and breast milk have all been implicated in the spread of the viruses, but infectivity seems especially related to blood, whether by transfusion of blood and plasma derivatives, use of inadequately sterilised needles, syringes and instruments, sexual contact or insect transmission.

According to Anonymous (1980), there are several types of hep B vaccines. Some are derived from the plasma of persistent carriers of hep B antigens. These of course carry the risk of contamination by undesirable host components, like the human immuno-deficiency virus (HIV) associated with AIDS. Vaccines are also prepared from some of the constituent polypeptides of hepatitis B surface antigen.

Another possible source of hepatitis B surface antigen is those cell lines derived from human hepato-cellular carcinomas (cancerous tumours), provided that both production and quality can be more precisely controlled. Synthesised vaccines contain immunogens: amino-acid sequences thought responsible for antigenic activity and have an "obvious advantage compared with current microbial vaccines, which often contain much irrelevant antigenic material contaminating the essential immunogen and provoking side effects" (Anonymous 1980).

Jacobson *et al.* (1984) discussed the potential contamination of plasma-derived hepatitis B vaccines. According to these authors, plasma-derived vaccines consist of purified particles of hepatitis B surface antigen. Many of the plasma donors are chronic carriers of the surface antigen, specifically those who are homosexual men, a group at risk of acquired immuno-deficiency syndrome (AIDS).

*"Because epidemiological observations suggest that AIDS is likely to be transmitted by a blood-borne virus, theoretically, an AIDS agent could contaminate the plasma pool from which hepatitis B vaccine is produced."*

Because of this concern, many persons with increased risk of hepatitis B viral infection have been reluctant to receive the vaccine. The authors further discussed the possibility of contamination of plasma-derived hep B vaccines. They admitted that commercial lots of hep B vaccine available in 1984 were prepared from plasma collected in 1981, when AIDS was first recognised as a distinct clinical entity and public health problem. The vaccine lots used in their study were prepared from plasma collected from 1977 to 1980 (the clinical disorders recognised retrospectively as cases of AIDS were observed in the US as early as 1979). The authors admitted that the donors of the plasma used in preparing the study vaccines could have been in the incubation stage of AIDS and could have provided the AIDS virus contamination.

Shaw *et al.* (1988) described adverse effects occurring after inoculation with the new plasma-derived hepatitis B vaccine. At the time when 850,000 persons were injected with the vaccine a total of 41 spontaneous reports were received: 5 cases of convulsions, 10 cases of Bell's palsy, 9 cases of Guillain-Barré syndrome, 5 cases of lumbar reticulopathy, 3 cases of brachial plexus neuropathy, 5 cases of ocular neuropathy and 4 cases of transverse myelitis. Half of these reactions occurred after the first of the required three doses. There were no reported deaths. The authors were quite aware that, due to under-reporting, these cases did not represent all occurrences of adverse reaction. Despite this, however, the authors concluded that the preventative benefits of the vaccine in persons of high risk for hepatitis B would unequivocally outweigh the risk of any neurologically adverse event.

Even the recombinant hepatitis B vaccines cause serious side effects. According to the Australian Adverse Drug Reactions (ADRAC) Bulletin, August 1990, some of the 203 reports of adverse reactions to hepatitis B recombinant vaccine listed neurological and

psychological effects. Of the 203 reported cases, 28 patients were subjected to re-challenge and their symptoms recurred.

The most common reactions to hep B vaccine reported to ADRAC in 1990 were: rash and/or itch (65 persons); pain, e.g. muscle, joint or abdominal (44); headache (32); fever (31); nausea, vomiting or malaise (29); injection site reaction (23); fatigue/asthenia (23); dizziness (21). The most serious were two reports of optic neuritis and one report of Guillain-Barré syndrome. One recent report described vertigo and diplopia with evidence (MRI scan) of demyelination persisting for over 8 months. Similar reports were associated with the use of plasma-derived vaccine.

The same ADRAC Bulletin wrote that the Committee was concerned with the possibility of adverse neurological reactions occurring after the administration of hep B vaccine. Indeed they wrote that "ADRAC would appreciate receiving a blue card from any of our readers who have observed demyelinating adverse events in association with all forms of vaccination."

Herroelen *et al.* (1991) described two cases of central nervous system demyelination after administration of recombinant hepatitis B vaccine.

Reitschel and Adams (1991) reported hypersensitivity to thiomersal, a mercury compound (sodium ethylmercurythiosalicylate) used as a preservative in the hepatitis B and the whooping cough, diphtheria and tetanus vaccines. It can be a component in eardrops and ophthalmic solutions, such as those used for soaking contact lenses. It is also a well-known sensitiser. Thiomersal in hepatitis B vaccines can cause severe cutaneous reactions. Forstrom *et al.* (1980) reported hypersensitivity to thiomersal (merthiolate) in other vaccines. This hypersensitivity can, of course, be delayed.

Cox and Forsyth (1988) stated that most young individuals in the UK are likely to have iatrogenic exposure to thiomersal via all vaccines for diphtheria, tetanus and pertussis. According to these authors, individual reactions to thiomersal demonstrated a need for vaccines with an alternative preservative.

Zuckerman (1975) warned that a variable amount of protein and perhaps carbohydrate may be complexed with hepatitis B viral protein in quantities apparently greater than those in most recognised viruses. These host proteins may include various pre-existing structures of the liver cell and may thus induce undesirable immunological reactions.

Smith, Kline and French pharmaceutical company was severely reprimanded for their advertising campaign in 1989. Instead of retracting their untrue and misleading statements about the dangers of hepatitis B and the need for vaccination of the majority of the population, they left the Australian Pharmaceutical Manufacturer's Association (Melbourne Herald 30 October 1989).

The Australian Dr Weekly (ADW) quoted a doctor in Brisbane saying that many people were being vaccinated for the wrong reasons, like pensioners hapily married for 40 years, while carriers were generally people from well-documented ethnic groups, intravenous drug users or the sexually promiscuous. But he also said that routine child vaccination was planned in Australia when cost of the vaccine is reduced.

So, the cost seems a more important factor than the imagined need or danger, if it can wait until the vaccine is produced in such large numbers that its cost will enable its mass use.

Another article in ADW in 1989 said that the recently identified hepatitis viruses C and D were regarded as more serious than the common A and B forms.

Again, these viruses occur most commonly in intravenous drug users, homosexuals and prostitutes. Also, the article said that hepatitis C and D are around but the incidence of these forms appears to be very low at this stage and *"doctors should look for it without expecting to find it"*.

So, doctors are aware that hepatitis A, B, C and D are rare diseases, affecting quite specific high risk groups of adults, yet infants will soon be vaccinated against one of them as if they were just about to become intravenous drug users, homosexuals, prostitutes or hæmodialysis patients.

To understand this totally unreasonable attitute it will help the reader of this book to hear the statement made by Dr George Peter, Chairman of the American Academy of Pediatrics (AAP) at the National Pediatric Infectious Disease Seminar on 12 June 1992 in Washington D.C. (NVIC Newsletter, August 1992). He gave the following reasons for recommending hepatitis B vaccine to all infants:

1. Hepatitis B remains a public health problem which sometimes occurs outside of high risk groups;
2. High risk groups have not accepted vaccination or have been difficult to reach;
3. Children are accessible;
4. Cost of vaccinating infants is less than vaccinating adults, since a smaller dose is required.

Committee on Infectious Diseases (Anonymous 1992) stated that universal screening of pregnant women for HBsAg (hepatitis B surface antigen) with active and passive immununisation of their infants, will prevent only about 6,000 of the more than 200,000 HBV infections that occur in the United States each year. Despite these telltale figures, the Committee recommended general mandatory infant immunisation, although the full effects of this strategy will not be known for 25 or more years.

*"Universal immunisation of adolescents alone has the advantage of a more immediate effect, but this strategy is problematic. Adolescents of the highest risk would most likely be the least compliant, and asking adolescents to participate in a three dose immunisation series over a 6-month period is likely to result in high drop out rates."*

One could justifiably ask: is the system afraid to mandate vaccination for people who can talk and describe adverse reactions to the vaccines? Most probably yes. Babies cannot talk.

The most alarming statement is *'children are accessible'*. Does this mean that infants are fair game for the medical system? If this is true, it would violate the rights of totally helpless human beings. Parents must take warning from this, because they have to defend

their babies. The right of choice, at very least, is being taken aw from parents by the mandating of vaccination in those countri which boast they are free.

The same Committee which recommended general vaccination all newborn infants admitted that the full duration of protectio induced by 3 HBV vaccines has not been determined. Although th rationale behind infant vaccination is to prevent hepatitis B infectio and disease in adulthood, it is quite likely that it will be a waste effort, since revacination will be needed after five to nine years.

Another worrying factor in hep B immunisation is the lack of knowledge of neonatal immunological response. As Chin-Yun Lee *et al.* (1983) stated: *"We are unaware of analogous phenomena with other infectious diseases. Further understanding of the immune system at the beginning of life is needed."*

The system is happily mandating vaccination of children but is surprisingly lenient to health workers: only 40% of health-care workers, who are considered at risk because of transmission through blood, have been vaccinated (New York Times, 3 March 1991).

Marwick (1991) discussed the introduction of hep B vaccine into the current standard pædiatric vaccines. This means that American infants (probably followed by Australian babies) will receive 15 injections of various vaccines within the first 6 months of life. This is hyperimmunisation *par excellence*.

The most immediate effect of the recently introduced and forcefully performed hepatitis B vaccination of newborn babies will soon be an increased rate of illness and cot death, which is already high in Australia and the United States. Liver dysfunction caused by vaccine injections (with hep B injection at birth being no exception), will hardly fall back to normal; the first DPT and polio vaccines will provide yet another major insult to underdeveloped immune systems. Many babies will not be able to handle this type of stress.

Perhaps the increase in cot deaths will alert parents and possibly some medical professionals to the facts of life. Naturally, there are also the long-term adverse effects of such hyperimmunisation, including diabetes, asthma, leukæmia, cancer and chronic ill health.

All this is done despite lack of knowledge of the duration of immunity (if any). The J Amer Med Ass Medical News (1 June 1984) stated that the spokesman for the manufacturer says *"the cost of the vaccine itself is about $100 for the three doses, and immunity probably lasts about five years, although that is not certain."*

They also quoted Dr S. Handler and colleagues, including members of the CDC Multicenter HBV Vaccine Study Group, who observed more than 500 multicentre trial participants for up to 40 months. *"Their findings suggest that antibody levels to HBV vaccine decline substantially within three years of vaccination and that risk of infection increases as antibody levels decline. However ... HBV infection occurring in these circumstances is mild, and there is no evidence of atypical infection."*

Here we go again, as with whooping cough, measles etc. The vaccine does not protect for life, but it is claimed that at least when you get the disease it will be mild, despite documented evidence that this is simply not true (see Chapter 2 on DPT and Chapter 3 on measles).

The well-vaccinated US is experiencing epidemics of infectious diseases in fully vaccinated children. No amount of denial and underreporting will cover up the one most obvious fact, namely, that vaccines do not work.

Freed *et al.* (1993: Pediatrics; **91 (4)**: 699-702) wrote that: *"Despite immunization programs targeting high-risk groups, the incidence of hepatitis B has risen 37% over the last decade with 300 000* (sic) *new infections and 5 000 related deaths now occurring annually in the United States ..."*

*"This increase led the Advisory Committee on Immunization Practices (ACIP) of the Centers for Diseases Control (CDC) to re-evaluate its hepatitis B control strategy and on November 22, 1991, to recommend universal immunization of infants against hepatitis B virus."*

*"778 pediatricians in North Carolina were surveyed by mail two to 34 months after publication of the new CDC recommendations.*

*Only 32% of those questioned believed that hepatitis B vaccination was warranted in their practices."*

*"Most pediatricians simply were not convinced that the hepatitis B immunization is needed by their patients, regardless of other factors."*

They also quoted anecdotal reports which *"... have raised concern over the* 'pincushion effect' *of multiple injections on children"*

Following the Hippocratic dictum *"First, do no harm"*, it would certainly be prudent to refrain from general hepatitis B vaccination of newborn infants. Knowledge of newborn immune systems is inadequate; the duration of immunity, if any, conferred by the vaccine is totally unresearched and there is a wealth of information on neurological side-effects following vaccination, even with the genetically-engineered product.

## References


1. Dienstag, J.L., and Ryan, D.M., 1982.
Occupational exposure to hepatitis B virus in hospital personnel: infection or immunization?
Am J Epidemiol; **115(1)**: 26-39.
2. Yodfat, Y., London, W.T., Whitford, P., and Wax, Y., 1982.
A seroepidemiological study of hepatitis B in a rural area of Israel.
Am J Epidemiol; **116 (3)**: 456-462.
3. Anonymous, 1980.
Hepatitis B vaccines.
Br Med J; 26 Jan (**6209**): 203-205.
4. Jacobson, I.M., Dienstag, J.KL., Zachoval, R., Hanrahan, B.A., Watkins, E., and Rubin, R.H., 1984.
Lack of effect of hepatitis B vaccine of T-cell phenotypes.
N Engl Med; **311 (16)**: 1030-1032.
5. Shaw, F.E., Graham, D.J., Guess, H.A., Milstien, J.B., Johnson, J.M., Schatz, G.C., Hadler, S.C., Kuritsky, J.N., Hiner, E.E., Bregman, D.J., and Maynard, J.E., 1988.
Postmarketing surveillance for neurologic adverse events reported after hepatitis B vaccination.
Am J Epidemiol; **127 (2)**: 337-352.
6. Herroelen, L., Keyser, J. de, Ebinger, G., 1991.
Central nervous system demyelination after immunisation with recombinant hepatitis B vaccine.
Lancet; **338**: 1174-1175.
7. Reitchel, R.L., and Adams, R.M., 1990.
Reactions to thiomersal in hepatitis B vaccines.
Dermatol Clin; **8**: 161-164.
8. Forstrom, L., Hannuksela, M., Kousa, M., and Lehmuskallio, E., 1980.
Merthiolate hypersensitivity and vaccination.
Contact Dermatitis; **6**: 241-245.
10. Cox, N.H., and Forsyth, A., 1988.
Thiomersal allergy and vaccination reactions.
Contact Dermatitis; **18**: 229-233.
11. Zuckerman, A.J., 1975.
Hepatitis vaccine: a note of caution.
Nature; **255**: 104-105.
12. Noel, I., Galloway, A., and Ive, F.A., 1991.
Hypersensitivity to thiomersal in hepatitis B vaccine.
Lancet; **338**: 705.
13. Anonymous, 1992.
Universal hepatitis B immunization. Committee on Infectious Diseases.
Pediatrics; **89 (4)**: 795-800.

14. Marwick, C., 1991.
Hepatitis B vaccine appears headed for pediatric immunization schedule.
J Amer Med Ass; **265 (12)**: 1502.
15. Chin-Yun Lee, G., Lu-Yu Hwang, Beasley, R.P., Shen-Hyi Chen,
and Tzu-Yao Lee *et al.*, 1983.
Immunogenicity of hepatitis B virus vaccine in healthy Chinese neonates.
J Infec Dis; **148 (3)**: 526-529.

# 2

# DPT VACCINE: a cot death connection

Diphtheria, pertussis, tetanus (DPT) vaccine, also known as 'triple antigen', is administered to babies as young as six to eight weeks in a number of industrially developed countries like the United States, Canada, France and Australia. However, many countries omit the whooping cough (pertussis: 'P') component for two reasons: its ineffectiveness in preventing whooping cough and its adverse effects. The pertussis vaccine has been demonstrated to be encephalitogenic (causing encephalitis, inflammation of the brain) in laboratory animals and babies. It is generally considered more reactogenic than the tetanus and diphtheria components.

In Australia, by the time they reach six months of age, babies are supposed to have received three DPT injections together with polio vaccine (either orally or injected). Also, all babies born in hospitals are given hepatitis B vaccine injections within 24 hours of birth.

In the US, a support group of parents whose children were adversely affected by vaccines, especially by DPT, was formed under the name Dissatisfied Parents Together ('DPT'). They opened a National Vaccine Information Center (NVIC). In their NVIC Newsletter, the reason for mandating hepatitis B vaccination at birth was summarised by Dr George Peter, the Chairman of the American Academy of Pediatrics' Committee on Infectious Diseases, as follows:

*1. Hepatitis B remains a public health problem which sometimes occurs outside of high-risk groups; 2. High-risk groups have not accepted vaccination or have been difficult to reach; 3. Children are accessible; 4. Cost of vaccinating infants is less than vaccinating adults, since a smaller dose is required.*

Although all vaccines are controversial, due to their inability prevent infectious diseases and due to a causal link to mild or serio local and systemic reactions, DPT is undoubtedly the mo controversial of them all. This is partly because of the very hig incidence of adverse reactions and partly because most doctors an health authorities vehemently deny any causal link between th vaccine and most of these reactions.

Yet the bulk of information on pertussis vaccine dangers an ineffectiveness has been published in medical journals. Mos importantly, DPT vaccine with its pertussis component has bee linked to cot death.

## Whooping Cough (Pertussis): a Hundred-Day Disease

Pertussis or whooping cough used to be a very serious disease especially for young children and infants below the age of one year. The reason was not only the exhausting paroxysmal coughing but also its duration — the Chinese and Japanese call it the hundred-day disease. In the 1940s, treatment consisted of the administration of sulphonamide and convalescent or hyperimmune human or rabbit serum globulin.

It is less well-known and less publicised that a change of air was widely used as a quite effective treatment. One of the causative organisms, *Bordetella pertussis*, is remarkably sensitive to changes in composition and temperature of air and to changes in altitude. However, it is quite understandable that many saw a need for effective prophylaxis, and doctors and researchers looked for an effective vaccine.

## Whooping Cough Vaccine Trials

In England, the Whooping-Cough Immunisation Committee of the Medical Research Council assessed the prophylactic value of pertussis vaccines used in the 1940s. From 1942 to 1944, controlled trials were conducted in Oxford City in children attending welfare clinics and day nurseries, and also in Oxfordshire, Berkshire and Buckinghamshire in children attending residential nurseries.

McFarlan *et al.* (1945) published results of these trials. Vaccines were ineffective because no significant difference was observed in the incidence or severity of whooping cough between the vaccinated and unvaccinated children. In the Oxford City trial, 12.5% or 327 vaccinated and 14.1% or 305 unvaccinated children developed pertussis. In those residential nurseries in which whooping cough developed, 55% of 33 vaccinated and 63% of 30 unvaccinated children developed the disease.

Others reported similar unfavourable results. Results of some American trials of pertussis vaccine were initially so variable that in 1931 the Council on Pharmacy and Chemistry of the American Medical Association recommended withdrawal of pertussis vaccine from the New and Non-Official Remedies.

The first hopeful results from large-scale vaccination campaigns were observed in two epidemics in the Faroe Islands. A plain vaccine, prepared from freshly isolated strains of *Bordetella pertussis* was used.

In the first epidemic, vaccination was begun during the outbreak and was found to have no effect on the incidence of whooping cough, although it was reported that it had reduced the severity of the disease. Madsen (1933) wrote that as a prophylactic measure, the value of the vaccine was *"not worth mentioning"*.

In the second Faroe Island epidemic, which followed shortly after the first, vaccination was completed just before the outbreak. Of the 1,832 vaccinated children, 458 (25%) did not contract pertussis compared with eight (less than 2%) among the 446 unvaccinated children. In both epidemics six patients of the 3,926 vaccinated died and 26 among the 1,073 unvaccinated cases died. So the vaccine seemed to provide some degree of protection; however, the numbers of vaccinated and unvaccinated are so different that any comparison is scientifically invalid.

Bell (1941, 1948) conducted two well-documented trials with two vaccines: alum-precipitated pertussis vaccine alone and alum-precipitated pertussis vaccine and diphtheria toxoid combined . The vaccines were given in two inoculations at four-week intervals. The

pertussis incidence in unvaccinated children was three to four times greater than in the vaccinated. Kendrick (1942, 1943) also reported encouraging results with an alum-precipitated vaccine given in three doses.

Based on these results, the Committee in England decided to repeat the trials with different vaccines. The trials were disappointing mainly because of the war and lack of staff to do a proper follow-up.

Subsequent trials were conducted in five areas in England. Parents of 8,927 children agreed to participate. Some 4,500 children were in the vaccinated group and the rest in the so-called "unvaccinated group". The "unvaccinated" children were injected with an anti-catarrhal vaccine which contained preparations of *Staphylococcus aureus, Streptococcus pneumoniæ, Corynebacterium hofmanni* and *Neisseria catarrhalis* and was similar in turbidity to the plain pertussis vaccines.

This is one of the first trials of vaccines in which the so-called controls were not a true control group.

One in five children vaccinated with the whooping cough vaccine or with the anti-catarrhal vaccine were monitored for up to 24 to 72 hours after vaccination. Only reactions to the pertussis vaccine were briefly described; no data on local or systemic reactions were given for children injected with the anti-catarrhal vaccine.

After home exposure, the rate of pertussis incidence was 18.2% in vaccinated and 87.3% in the anti-catarrhal groups. This difference in attack rates cannot be attributed solely to a protective effect of the pertussis vaccines because the so-called unvaccinated group who served as a 'control' were in fact given the anti-catarrhal vaccine.

Just like the pertussis vaccine, this anti-catarrhal vaccine contained a number of foreign proteins (antigens) and had the ability to lower the resistance of the recipients. For this reason alone, the above trial cannot be considered valid.

This evaluation of the above trial of pertussis vaccines is particularly feasible since Wilson *et al.* (1965) demonstrated that in epidemic situations pertussis vaccines were ineffective in preventing the spread of an epidemic.

## Ineffectiveness and Adverse Reactions to DPT or Pertussis ( P) Vaccines

Provenzano *et al.* (1959) reported on four children in one family vaccinated after accidental exposure to pertussis during summer camp. All had been given three injections of DPT (the two older children also received booster injections after exposure). The two younger ones developed severe whooping cough. *"This observation persuaded the pediatrician to introduce the five initial injections of three plain vaccines and two DPT as a prophylactic measure in a number of his patients"* without investigating other possible reasons for the two older vaccinated children not contracting whooping cough after this particular exposure.

Also, Provenzano (1959) wrote that *"... severe cases of pertussis developed in the two younger children, R and D, but not in B and A"*. It may mean that mild cases of pertussis developed in the two older children. However, it is well-known that even totally unvaccinated children do not contract whooping cough during an epidemic.

Lambert (1965) reported on an outbreak of whooping cough in Michigan and concluded that the direct relationship between increased pertussis incidence in vaccinated persons and increased interval since the last injection of pertussis vaccine was the most significant finding. The gradual loss of protection for vaccinated persons was apparent in all age groups and was independent of the number of injections received or the age at which the primary course was initiated.

Immediately after the earliest use of the various pertussis vaccines, reports of adverse reactions to these vaccines started filling pages in medical journals. Madsen (1933) published his report on the death of two newborn infants in Denmark. The physicians in charge decided to vaccinate both babies because there was a case of pertussis in each family. One child was given 0.1 cc of whooping cough vaccine immediately after birth. This caused no symptoms, so the child was given 0.15 cc of the vaccine four days later. Half an hour after this injection, the child had convulsions, cyanosis and hiccup, and died within a few minutes.

The second death occurred two years later. A baby born five we prematurely, weighing only 2.250 kg, was given 0.1 cc of whoop cough vaccine eight days after birth. Three days later, 0.2 cc of vaccine was injected. The baby died two hours later. Postmort failed to establish a definite cause of death.

Despite the fact that numerous newborn babies were vaccinat without any ill effect, vaccination of children younger than o month was not further recommended. Madsen (1933) wrote th many of his colleagues told him that they had seen *"a considerab malaise following the vaccination"*.

One of the most important reports of this kind was the study b Byers and Moll (1948). All 15 originally normal children reacte violently within 72 hours of pertussis vaccine injections. Two o these babies died, nine suffered irreparable damage, three had no been followed-up for a sufficient time and only one had apparently recovered completely. This report disturbed doctors because, until this time, pertussis vaccine had been considered innocuous. Strangely enough, American doctors continued to insist that the benefits of pertussis vaccine outweighed the dangers.

Anderson and Morris (1950) described severe neurological complications in a two and a half year old boy who developed convulsions thirty six hours after a combined diphtheria-pertussis vaccine. Skull X-rays eight months later showed dilatation of the left lateral ventricle and diminished electrical activity of the left cerebral hemisphere. The child remained mentally retarded and partially paralysed.

The authors also briefly reviewed other publications reporting similar problems after whooping cough and combined pertussis-diphtheria vaccines. They also concluded that if there is any untoward reaction to the first injection, or previous neurological symptoms, vaccination should be abandoned.

Low (1955) reported a case of a little girl who was born normal in every respect. About 12 hours after an injection of alum-precipitated DPT vaccine she had a brief convulsion. A month later she was given the second DPT injection; 12 hours later she had severe convulsions

and died 12 hours after the onset of the first symptoms. The autopsy showed major brain damage.

Electroencephalograms were recorded before and after pertussis immunisations in 83 cases, so that each child served as their own control. All children except three were found to be normal before and after the injections. Two of the three affected had abnormally slow leads after the injections. The third one had abnormal encephalograms before and after the injections; there was a family history of convulsions in this case.

Baird and Borofsky (1957) reviewed their experience with infantile myoclonic seizures, in view of observations by others that a number of patients with infantile myoclonic seizures had histories suggesting normal development prior to their immunisation with diphtheria, pertussis and tetanus vaccines. The authors found association with cerebral palsy and marked visual problems. They listed DPT immunisation among the ætiological factors for development of infantile myoclonic seizures.

Another important paper dealing with neurological complications and pertussis inoculation was published by Kulenkampff, Schwartzman and Wilson (1974). They reviewed 36 cases and stated that clustering of complications in the first 24 hours after inoculation suggested a causal rather than a coincidental relationship. They recommended that pertussis vaccine should not be given to patients with a history of fits in first-degree relatives, or to those who have had a reaction to previous inoculations, who have had recent intercurrent infection or those with presumed neurological deficit.

Miller and Stanton (1959) presented a review of the literature on neurological complications of prophylactic inoculations and serum administration, and a report of 12 further recent cases. The authors wrote that although neurological sequelæ of inoculations are described in textbooks of neurology, they receive only scant attention and are often entirely ignored in some large works on infectious diseases and preventive medicine. It is generally implied that the parenteral introduction (by injection into the blood stream) of a variety of biological products is a procedure devoid of risks. The

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Importantly, Miller and Stanton (1959) recognised that the nervous system has a limited repertoire of clinical as well as pathological responses. This means that reactions to a variety of vaccines may be essentially the same.

Noah (1976) discussed the attack rate of whooping cough in fully immunised and partly-immunised children. Although there was a lower incidence of whooping cough in fully immunised children compared with those partly immunised, the fact remains that the incidence in both groups was quite high. If the pertussis vaccine were effective, no immunised child should have contracted the disease.

Miller and Fletcher (1976) reported on the severity of notified whooping cough. Some 8,000 cases were reported during an outbreak. The age of victims ranged from less than five months to less than five years. Of the hospital admissions, 39 were fully vaccinated, 41 partially vaccinated, 616 not vaccinated, and for 79, the vaccination status was unknown.

Of the home cases, 2,901 were fully vaccinated, 590 partially vaccinated, 1,808 not vaccinated at all, and 2,028 not known.

These figures hardly indicate that the vaccine was effective.

In 1960 Justus Strom, a Swedish medical doctor, published an article in the British Medical Journal in which he stated that, in Sweden, not only did the incidence of neurological complications after pertussis appear to be not as high as the complications after vaccination, but the disease itself had become much milder and did not justify mass vaccination.

Continuing research into *Bordetella pertussis*'s biological activities revealed a number of disconcerting properties. Munoz and Bergman (1966) demonstrated that *Bordetella pertussis* vaccine, or soluble preparations of the histamine sensitising factor, produced histamine sensitisation in mice lasting 21 days at a high level, less at 42 days and was still evident 84 days post injection. They also established that sensitisation was dose-dependent.

A Scottish combined study (1970) demonstrated that *Bordetella pertussis* was isolated in only about one half of pertussis patients; previous immunisation appeared to reduce the chance of isolating *B. pertussis*. This was confirmed by many other studies.

Connor (1970) published evidence for an ætiological role of adenovirus infection in pertussis syndrome. Eleven of 13 infants and children with the clinical diagnosis of whooping cough syndrome were excreting adenovirus types 1, 2, 3 or 5 from the respiratory, intestinal and genito-urinary tracts.

In all these cases, cultures for *Bordetella pertussis* and *B. parapertussis* were negative and the majority had no evidence of pertussis infection as determined by rises in anti-pertussis agglutinins.

Indeed, Lautrop (1971) compared parapertussis and pertussis as infectious entities on the basis of 1,800 isolates of *B. parapertussis* and about 41,000 isolates of *B. pertussis* in Denmark between 1946 and 1970. He established that both infections present as epidemics every fourth year, with a two-year shift between the pertussis and parapertussis peaks.

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Linnemann and Perry (1977) stated that eight of 37 (22%) *Bordetella* organisms isolated from patients in Cincinnati were *B. parapertussis*. He concluded that *B. parapertussis* infections may be more common in the US than generally recognised.

Balagtas *et al.* (1971) performed a controlled study of the efficacy of pertussis immune globulin in treatment of whooping cough. Children with whooping cough were given 2.5 cc of pertussis immune globulin in the first week of paroxysmal cough. Both cases and controls received ampicillin for 10 days. There was no difference in recovery rate between the two groups. Neither the frequency of paroxysms, vomiting or pulmonary complications nor the rate of suctioning required were different in the case and control groups.

Melchior (1975) claimed that when the vaccination schedule in Denmark changed from five, six and fifteen months to five weeks, nine weeks and ten months, there was no corresponding shift in the incidence of convulsions.

However, his own graph clearly shows a shift to earlier ages and to ten months. Melchior (1975) did not realise that most of the vaccine reactions, including convulsions, are delayed and that not all babies were vaccinated at exactly five and nine weeks, so the shift was gradational, reflecting the delay in reactions and individual vaccination schedules.

In a landmark article, Bassili and Stewart (1976) demonstrated that nearly one-third of notified cases of whooping cough were fully vaccinated.

In Glasgow (and probably in the UK as a whole), the persistence of whooping cough in many areas was more strongly correlated with adverse socio-economic conditions than with lack of immunisation. Their figures 1 and 2 clearly show that there was a considerable decline in incidence of whooping cough long before mass immunisation started.

The authors pointed out that the downward trend of whooping cough incidence actually slowed down after 1960. There was also no evidence of reduction in severity of disease nor in complications in immunised cases admitted to hospital.

## England and Whooping Cough vaccination

The National Childhood Encephalopathy Study (NCES) was carried out in England, Wales and Scotland from 1 July 1976 to 30 June 1979. During the study period, 1,182 children were hospitalised with acute severe neurological illness. The findings were quite important. One of them was that DPT vaccination had occurred significantly more frequently within 72 hours and the first seven days after vaccine injections in the children with neurological illness than in the controls at the same time periods. A similar analysis for DT also showed an increase in relative risk of neurological illness in affected children compared with controls. The estimated risk of serious neurological disorder within seven days of DPT immunisation in previously normal children was estimated to be one in 110,000 immunisations and the estimated rate of permanent brain damage one year later was estimated to be one in 310,000 immunisations.

At the same time as admitting a significant association between serious neurological illness and pertussis vaccine, the authors[(Miller *et al.*, (1981)] made statements confusing the whole issue. They said that although there had been reports of neurological illness after pertussis immunisation since 1933, *"... none has been based on established epidemiological methods using relevant controls. The inconclusive nature of this evidence has resulted in much debate in the media, particularly in Britain, and left doctors and parents anxious and confused about the safety of the vaccine."*

It is not true that the nature of the evidence like that published by Madsen (1933) is inconclusive. Case histories of babies dying and/or being damaged shortly after administration of the vaccine were clear-cut and straightforward. The case histories simply don't suit the proponents of vaccination, who devised scientifically invalid case-control studies where both cases and controls are vaccinated with either the same or two different vaccines. So, there is no true control group for comparison. Just because these case-control studies became a norm does not make them valid.

The Committee maintained that decline in immunisatio compliance resulted in the largest epidemics of pertussis in 20 year However, as Fine and Clarkson (1982) documented, th inter-epidemic period did not decrease after the 1974 fall in vaccin uptake.

The raw data on the age distribution of notified infants at onset o symptoms reveal the authors'erroneous reporting on the results o the NCES study.

Firstly, the age distribution of neurological illness shows a sudden increase in the number of infants suffering side-effects at two months of age. Nine times as many babies, who were originally completely normal or had only slight neurological damage, had the onset of adverse reactions after the first DPT when compared with one- and 12- month olds.

Secondly, from two and three months of age, there is a sudden and substantial increase in the number of cases until six months of age. This again gives immunisation away. Before reaching seven months the majority of babies usually receive some or all of the first, second and third DPT injections. By 17 months they have all received their booster, with a tail-end at 18 and 19 months. After this age there is a substantial drop-off in the numbers of cases (only one sixth of the cases at 20 months compared with younger ages).

Afterwards, the number of cases remained at a very low level. The vaccination times are thus quite clearly reflected in the authors' tabulation. This analysis is further supported by the statement of the authors that there was no noticeable clustering of cases. Vaccination is performed all year round with little seasonal fluctuation.

The arbitrary cut-off point at seven days after vaccination for accepting the cases into the study was quite unjustified in view of the well-known fact of delayed reactions to a variety of vaccines, DPT being no exception. But, of course, this lowered the numbers of vaccine-related reactions quite substantially.

Ehrengut (1978) expressed doubt whether the Joint Committee on Vaccination and Immunisation adequately analysed the problem of adverse reactions, especially of cerebral nature. He wrote that the

diminished acceptance rate of pertussis vaccination was a particular concern of the Committee. While 70-80% of British children were immunised against pertussis in 1970-71, the rate in 1978 was only 39%. The Committee predicted that the next pertussis epidemic would probably be more severe than the one of 1974-75. However,

*"... they do not explain why, in 1970-71, there were more than 33,000 cases of pertussis with 41 fatal cases among the very well immunised British child population ... whereas in 1974/75 with a declining rate of vaccination, a pertussis epidemic caused only 25,000 cases with 25 fatalities."*

Ehrengut's (1978) figure 1 demonstrates that despite the lack of pertussis vaccination in Hamburg since 1962, and an increase in live births, admissions for pertussis fell from 3.7 to 0.8 per thousand cases and the proportion of complications has also fallen.

Social reasons for the drop in hospital admissions can be excluded because, in the same period, the number of mumps cases increased six-fold.

He was also critical of the Committee's attitude to pertussis vaccine central nervous system complications:

*"Cases of convulsions and encephalopathy following the use of triple vaccine ... must be examined to see if they differ in any clinical respect from those which occur naturally, apart from a purely temporal relationship."*

Then the Committee contradicted this by stating that H.G. Miller and J.B. Stanton had observed that neurological complications after pertussis vaccination *"differed from those of other agents"*. The Joint Committee greatly overrated the probability of a non-vaccine ætiology of complications.

*"Even if a virus were isolated from a patient with CNS complications developing within 72 hours of vaccination, vaccination could not be excluded as an additional factor."*

Ehrengut was also critical of the Committee's decision to wait for the results of prospective studies, despite the soundly based knowledge and voluminous evidence from several cases of vaccination encephalopathy reported by Byers and Moll (1948).

Equally regrettable was the treatment of 32 cases encephalopathy and 142 convulsions after diphtheria-tetanus pertussis (triple) vaccine injections as reported to the Committee fo the Safety of Medicine between 1964 and 1978. John Wilson neurologist at the Hospital for Sick Children in London, whos documentation of CNS complications after pertussis vaccination i well-known, was not asked for his expert opinion.

Even if only 32 cases of encephalopathy occurred during 1964-1976, when some seven million children in England and Wales were vaccinated, then the rate would be one in 220,000 complete immunisations. The Committee estimated only one case in 310,000 immunised children, i.e. a rate of approximately one twelfth of cases experienced in West Germany and one sixth of the rate in East Germany. The official figure of encephalopathies in West Germany was 1 in 30,000 vaccines and not one in 100,000 as misquoted by the Committee. Since doctors in most European countries are not legally required to notify pertussis vaccine-associated complications, these figures are far from complete. Only a minority of cases are reported to the authorities and this underreporting is not likely to improve.

The Public Health Laboratory Service (PHLS) of the Communicable Disease Surveillance Centre study (1982) published interesting data on age distribution of whooping cough before and during mass vaccination programmes and also after the compliance to vaccinate fell below 30%.

The death and case fatality ratios declined tenfold in the mid-1950s (long before the vaccine was introduced in 1957). The age distribution before 1957 was such that about 10% occurred in age group 0-1 year and two thirds in age group 1-4 years. However, in 1970-75, the rate of whooping cough cases in 0-1 year group exceeded that in 1-4 years group (70%). This was the time of the highest uptake of the vaccine. The case fatality in this age group was 20 times that in the other age groups. When the uptake of the vaccine fell dramatically in 1975, this was followed by a reversal to normal age distribution of whooping cough, i.e. the incidence in the 0-1 year age group fell dramatically compared with the incidence in the 1-4

years age group. Quite obviously, pertussis vaccine was spreading the disease to the very infants supposed to be protected by it.

In 1977, an epidemic of whooping cough occurred in a rural practice in Shetland, containing 144 children under 16. Before 1 July 1974 all children were immunised against pertussis, but after that date immunisation stopped. The incidence of infection was similar in those who had and who had not been immunised, and in those born before and after 1974. Ditchburn (1979) described the epidemic in detail. The first child affected by the pertussis epidemic was a 15-year-old girl who was fully immunised as a child. The first eight of the case children were fully immunised. Overall, whooping cough occurred in 46 (49%) of 93 immunised children and in 18 (44%) of 41 unimmunised children.

Ditchburn (1979) considered it interesting that the outbreak started among the older children, the immunisation rate of whom was 94%. If the immunisation had been effective, this high rate should have produced herd immunity sufficient to have prevented the epidemic. It did not and almost half of the children under 16 years and some adults were affected. The illness was relatively mild. No child suffered permanent damage and there were no hospital admissions, though the disease was unpleasant and prolonged. One child who started having convulsions on the night of his second triple antigen injection in 1969, who required anti-epileptic treatment until 1976, developed whooping cough in the 1977 outbreak.

Ditchburn concluded that there was no evidence to support the routine immunisation against pertussis in rural Shetland.

The same epidemic of whooping cough in seven areas of rural Shetland was also described by MacGregor (1979). He divided 233 children in the one to four years of age group into fully vaccinated, partially vaccinated and not vaccinated.

Although 6% and 15% of fully and partially vaccinated children (21% in total) contracted whooping cough as opposed to 18% of not vaccinated, he concluded that *"... in the vulnerable group of 1 to 4 year olds, pertussis vaccination has continued to confer substantial protection against whooping cough."*

These differences in evaluation of the Shetland epidemics are quite clearly due to bias. Published evaluation by authors who collected the raw data themselves (like Ditchburn 1979) show unequivocally that vaccination had no effect on the incidence or mortality of pertussis in Great Britain.

Stewart (1977) brings further evidence to show that both pertussis morbidity and mortality showed very much a declining trend, long before mass immunisation was practised in Great Britain, and no protection by vaccination can be demonstrated. Despite evidence to the contrary, most experts and official bodies claimed that the risks of whooping cough exceeded those of vaccination. They seem to have accepted and implemented vaccination. However, there are a number of well-documented cases of serious adverse cerebral reactions and the majority of these go unreported. Publications and literature from manufacturers tend to discount reactions and do not mention the possibility of death or permanent brain damage.

Stewart (1977) was convinced that adverse reactions are more common and more serious than is generally acknowledged. He wrote that examination of national data and survey of the present position in Glasgow reinforces the published views that present schedules of vaccination with *B. pertussis* are ineffective and that epidemiological monitoring of efficacy and adverse reactions is incomplete. Stewart then analysed 69 cases of whooping cough in a family study, of whom 47 (68%) had been fully vaccinated. In a school study, 59 cases of whooping cough were identified from absence records in ten primary schools. The majority of cases were in children who had received three injections of DPT vaccine. He also established that notifications in this group by general practitioners to the Health Department were significantly fewer than the notifications of whooping cough in children who were unvaccinated or incompletely vaccinated. On the subject of permanent brain damage, Stewart (1977) reasoned that if the risk is one in 20,000 then at least 30 children will suffer permanent brain damage each year. This far exceeds the risk of death or permanent brain damage from whooping cough disease or even, in some parts of the country, the chance of contracting it.

Reports of increased epidemics shortly after a fall in vaccination compliance are quite untrue and, at best, exaggerated. As mentioned above, important information relevant to this was published by Fine and Clarkson (1982). The authors wrote that, although overall pertussis incidence fell in England and Wales after the introduction of mass vaccination in the fifties, whooping cough epidemics continued to occur regularly every three to four years. And indeed, surprisingly for those who believe that epidemic frequency is a function of the number of susceptible persons, the intra- epidemic period did not decrease after the 1974/75 fall in vaccine uptake.

Stewart (1979) criticised the handling of the pertussis epidemic of 1977-78 by MacGregor (1979) and Jenkinson (1978). These authors confined their "experience" to the age group 1-4 years. Since their overall attack rates were similar (vaccinated 21% and unvaccinated 19%), but attack rates in unvaccinated very different (27% and 79%) there must be differences other than vaccine protection between these selected samples.

In contrast, Ditchburn (1979) showed an overall attack of 55% in age group 1-4 and 48% in all age groups.

Stewart ascertained that, as Fisher showed in 1935, data extracted from less than the whole of the data are misleading if the data relate to mutually exclusive events.

Both Jenkins and MacGregor (*op.cit.*) assumed that prior probabilities of being infected were equal in vaccinated and unvaccinated, while their data provided no evidence of it. While the epidemic of 1977-78 started in one area of Shetland in mostly vaccinated schoolgirls aged 8-16 years, in another area 43% of whooping cough "victims" were vaccinated.

The occurrence of whooping cough in vaccinated children can legitimately be seen as evidence that protection is incomplete, since exposure has obviously occurred. The non-occurrence in vaccinated children indicates protection only when there is proof of exposure and, at the same time, occurrence of whooping cough in unvaccinated children. As stated in Stewart's 1980 paper, about 35% of reported cases were in children who had received three doses of pertussis

vaccine. About 95% of unvaccinated children in the age group 0— years either escaped infection or were not notified as havi whooping cough.

The American Journal of Epidemiology published criticism b G.T. Stewart (1984) of an article by Miller, Alderslade, and Ros (1982), and especially of their claim that "epidemics" of whoopin cough which occurred in Britain in 1977-79 and 1981-82 were cause by the drop in vaccination. Stewart wrote that data used by Miller *e al.* (1982) came from passive surveillance by optional notifications while such notifications can be fallacious and unrepresentative. In the west of Scotland, about 50% of notifications of whooping cough (and measles) came from about 12% of practitioners; 37% of notifications from 2% of practitioners. There is also a tendency during an outbreak to report coughs in unvaccinated children as whooping cough and to under-report coughs in vaccinated children. This was true in other parts of Britain.

Also, the earlier stages of the "epidemics" in 1977-79 and 1981-82 were accompanied by, and increasingly explained by, infections associated with organisms other than *B. pertussis*. In 1982, 201 cases were admitted to the only infectious hospital in Glasgow, of which only 147 were confirmed clinically as whooping cough, and only 86 microbiologically. About 30% of cases were in fully vaccinated children. In fact, the "epidemics" of 1977-79 and 1981-82 were the expected cyclical recurrence of whooping cough every 44 months. In some areas it is quite certain that the cyclical recurrence began in older children, many of whom were fully vaccinated.

Stewart also wrote that there would be no controversy if whooping cough were as severe as is claimed, or if the vaccine were safer. Assumption that the risk-benefit analysis favours vaccination creates the danger, not only of accepting an hypothesis which is not tested, but also, because the assumption is already adopted as policy, of creating conditions whereby it cannot be tested.

Natural conditions for such testing, however, occurred when after 1975 the vaccination compliance fell substantially. This time, the hypothesis has been tested: a shortening of the inter-epidemic period

to less than 44 months with an increase in hospital admissions, complications and deaths required to confirm the hypothesis has not occurred. Quite to the contrary: the inter-epidemic period of 1974-1978 was the longest on record and the death rate as well as number of hospital admissions in 1978-79 were both very low.

Stewart was also critical of the estimate of toxicity (acute encephalopathy) published by Miller *et al.* (1982), especially since they included only those children who were admitted to hospital after a convulsion lasting 30 minutes or longer, occurring within 30 days after vaccination, who were still in hospital 15 days later. The unknown, but considerable number of children who experienced lesser reactions, or who were not hospitalised as acute cases, were excluded.

Other similar retrospective studies estimated the frequency of severe mental and physical defects as 1 in 25,000 to 1 in 60,000, and of severe immediate neurotoxic reactions not followed up or not reported as 1 in 750.

If whooping cough is represented as a dangerous infectious disease, vaccination against pertussis are also dangerous. With the trend towards much milder forms of disease and fewer reactions, damage from vaccines could easily exceed corresponding risk of infection or damage from the disease. Stewart summarised that

*"Unfortunately, and largely based on data provided only in Britain and the United States, the world is now committed via the World Health Organisation to a policy of vaccinating all infants without any active surveillance of the current frequency and risk of whooping cough, without any plans for evaluating the results and, seemingly, without any thought of compensating the uncounted victims of unnecessary vaccination. Obviously, there are many parents and doctors—in Britain, West Germany, Sweden, and elsewhere — who prefer, under these circumstances to 'take chances with nature'."*

The British Medical Journal (1982) published an article on whooping cough surveillance prepared by the Communicable Disease Surveillance Centre. Whooping cough became notifiable in

England and Wales in 1940. After a peak in 1941 and 1948 - 1953, (when the vaccine first became available), notifications fell by more than two thirds between 1957 and 1961, when pertussis vaccine was in general (mass) use. Afterwards, the decline in notifications slowed down with outbreaks every three to four years, the smallest in 1974-75 when the uptake of pertussis vaccine fell in many areas down to 30% and even 10%. Notifications began to rise again in 1977 and large outbreaks followed in 1978-79 and 1981-82, coinciding with the rapidly increasing acceptance of the vaccine.

Even more interesting are data on the age distribution of whooping cough. Around 1947, about 10% of whooping cough occurred in children less than one year of age. However, by 1970-75, the previously highest rates in one to four year olds were exceeded by the rate in babies under one year of age. This was undoubtedly caused by the increasing administration of pertussis vaccine in this age group. The vaccine, instead of preventing babies below one year of age from contracting whooping cough, shifted the majority of cases into this age group as a consequence of sensitisation by the three injections of the vaccine within the first year of life. The same trend was observed in death rates in this age group, which increased from about two thirds in 1945-51 to about 70% since then.

Before the introduction of routine immunisation, most *Bordetella pertussis* organisms belonged to types 1,2 and 1,2,3. The original vaccines contained antigen types 1 and 2. Antigen 3 was included in the vaccines in the late 1960s, but between 1970 and 1979 types 1,3 remained the most common circulating type, accounting for more than 80% of nearly 6,000 isolates examined. Since 1980 this proportion has declined to 62% of nearly 2,000 isolates and types 1,3 and 1,2,3 have increased.

In Australia, Bennett (1973) analysed the incidence of whooping cough in Melbourne and concluded that in children admitted to hospital, vaccination against pertussis does not modify the disease if the total duration of patient's disease is used as an index of severity. He also stated that the decline in notifications of whooping cough is not reflected in the number of cases admitted in hospital.

He advocated a more accurate monitoring of the incidence in the next decade for proper evaluation of the value of vaccination. The main factor that reduced mortality from the disease appeared to be the use of chemotherapeutic agents (mainly antibiotics) to prevent and treat pneumonia.

Goldacre and Harris (1981) on their figure displayed oscillations in number of statutory notifications and hospital admissions for whooping cough in each quarter of 1974-79. The interesting thing about this figure is that it clearly shows in mid-1975, when vaccination compliance fell dramatically, the incidence of whooping cough and hospital admissions also started falling and kept falling consistently for two years. There was an increase in both incidence and hospital admissions in 1976, followed by another fall at the beginning of 1977, then a gradual increase in incidence and admissions again over a period of two years.

If pertussis vaccines were effective, the fall in vaccination compliance in mid-1975 should have been followed by a huge epidemic. All raw data over that period presented by a number of authors show exactly the opposite: a clear fall in the incidence of whooping cough.

Pollock *et al.* (1984) also reported that *"Since the decline of pertussis immunisation, hospital admission and death rates from whooping cough have fallen unexpectedly."* The same was experienced in Sweden after vaccination against whooping cough was discontinued in 1979.

### Sweden Rejects Whooping Cough Vaccines

The history of whooping cough incidence in Sweden is of special interest since Sweden stopped vaccinating with the whole cell vaccine in 1979. Trollfors and Rabo (1981) wrote that during the 1970s, despite general immunisation, whopping cough returned to Sweden after more than 10 years' absence. The disease became endemic. In 1978 5,140 bacteriologically verified cases of whooping cough were reported to the National Bacteriological Laboratory in Stockholm. Investigation of a subsample showed that of 620 cases

of one to six year old children with pertussis, 521 (84%) had received three injections of pertussis vaccine.

Similarly, another investigation of 84,015 preschool children, born between 1974-78 in various regions of Sweden, showed that they had been given three injections of pertussis vaccine. Vaccination with this obviously ineffective vaccine was stopped in 1979.

Taranger (1982) in a letter to the Editor of the Lancet referred to Peltola and Michelsson's article in the Lancet (1982) who quoted a Swedish paper from 1970 when stating that "pertussis is most life-threatening in infants who are less than 6 months old". Taranger wrote that in Sweden today not even for infants of that age is pertussis a serious disease. The clinical course of pertussis has become milder, although the incidence rates approached those of the pre-vaccine era, and no child has died of pertussis since 1970.

Discontinuation of pertussis vaccination was followed by three years of low endemic levels of whooping cough. Thereafter the incidence gradually increased and there were two outbreaks in 1983 and 1985. Romanus *et al.* (1987) summarised the situation in Sweden after cessation of pertussis immunisation. He wrote that outbreaks in Sweden in 1977 to 1978 appeared despite high immunisation coverage (more than 80%). The next outbreak in 1982 and 1983 occurred in a population in which the 3 or 4 youngest age groups were totally unimmunised. The magnitude of the epidemics is difficult to assess because pædiatricians and doctors specialising in infectious diseases are not obliged to report cases of pertussis. Although the reported figures of pertussis cases are much lower than the actual figures, they nevertheless reflect the general trends. Children born in 1978 had a lower pertussis incidence than those born in 1977, 1979 and 1980.

It is not possible to compare the seriousness of pertussis in immunised and unimmunised children born in the 1970s, because information on the pertussis patients hospitalised in the 1970s is not available. There are no data to follow-up the long-term records of sequelæ. However, according to Romanus *et al.* (1987), it is on record that all patients were apparently healthy on leaving the

hospitals and at the next visit. Trollfors (1984) stated that the pertussis mortality was generally currently very low in the industrialised countries and there was no difference in severity and incidence of whooping cough between countries with high, low and zero immunisation rates.

In 1982-83 a trial of the efficacy of a plain whole-cell pertussis vaccine was conducted in Sweden. 525 infants aged two months, born on even numbered days, received three doses of vaccine one month apart, and 615 infants born on odd numbered days of similar age served as controls. During the 28 months of follow-up there were 55 cases of pertussis in children between the ages 6 to 23 months. The attack rate in those vaccinated was 1.5% (8/525) and in the unvaccinated 7.6% (47/615).

All eight cases among the vaccinated were confirmed by laboratory results and in the unvaccinated the proportion was 32 out of 47. Bacteriologically 5 out of 8 and 21 out of 47 were confirmed in the two groups. The estimated efficacy of the vaccine based on laboratory results was 71%; the overall efficacy, including the unconfirmed clinical cases, was estimated at 80%. Considering the low numbers of participating infants, these results are inconclusive.

**Swedish trial of Japanese acellular pertussis vaccines**

In 1986-87 Sweden trialled two Japanese acellular pertussis vaccines. Both were developed by the Japanese National Institute of Health, (JNIH), and produced by the Kanonji Institute, Osaka University. One of these vaccines (JNIH-6) was a two-component vaccine containing formaldehyde-detoxified lymphocytosis-promoting factor and filamentous hæmagglutinin, used in Japan since 1981 for children two years and older. JNIH-7 was an experimental monocomponent vaccine, manufactured for this trial.

3,801 children six to eleven months old were divided into two groups of approximately 1,400 each and given one of the two acellular vaccines. 954 babies were given placebo and served as a control group. The placebo (vaccine solvent) contained formalin (tissue fixative), thiomersal (a preservative containing mercury) and

aluminium phosphate in phosphate-buffered saline in a final preparation of 0.15 mg of aluminium.

The immediate side-effects were considered mild. Small local reactions occurred more in the vaccinated than in the placebo groups, especially after the second dose of the two-component vaccine. However, the following systemic reactions were recorded in the first 24 hours after vaccination only.

| | JNIH-6 | JNIH-7 | "placebo" |
|---|---|---|---|
| Hypotonia (dose 1): | 0.5%; | 0.5% | 0.6% |
| (dose 2): | 0.1%; | 0.2% | 0.1% |
| Twitching/spasm | | | |
| (dose 1): | 0.3% | 0.5% | 0.4% |
| (dose 2): | 0.1% | 0.5% | 0.0% |
| Anorexia (dose 1): | 7.1% | 6.2% | 6.1% |
| (dose 2): | 6.1% | 5.8% | 7.5% |
| Vomiting (dose 1): | 5.6% | 4.1% | 5.0% |
| (dose 2): | 4.7% | 3.2% | 4.1% |
| Persistent crying | | | |
| (dose 1): | 1.1% | 2.1% | 0.8% |
| (dose 2): | 1.4% | 1.4% | 1.1% |
| Persistent crying 1hr or more | | | |
| (dose 1): | 0.3% | 0.7% | 0.8% |
| (dose 2): | 0.4% | 0.4% | 0.4% |
| Fever 38 deg C at 3 or 6 hr. after dose | | | |
| (dose 1): | 6.1% | 6.7% | 4.0% |
| (dose 2): | 4.9% | 6.0% | 5.2% |
| Drowsiness, no fever | | | |
| (dose 1): | 7.0% | 6.8% | 6.0% |
| (dose 2): | 6.6% | 6.7% | 6.7% |
| Pallor (dose 1): | 1.3% | 0.8% | 1.3% |
| (dose 2): | 0.5% | 0.4% | 0.6% |

The following local reactions were recorded after the first and second trial doses:

| | | JNIH-6 | JNIH-7 | "placebo" |
|---|---|---|---|---|
| 25 hr. questionnaire: local redness, swelling and/or tenderness | | | | |
| | (dose 1): | 10.2% | 10.4% | 7.8% |
| | (dose 2): | 17.8% | 12.7% | 9.1% |
| 24 hr. examination: redness 10mm | | | | |
| | (dose 1): | 0.2% | 0.4% | 0.1% |
| | (dose 2): | 8.6% | 3.4% | 0.7% |
| swelling | (dose 1): | 0.4% | 0.6% | 0.1% |
| | (dose 2): | 5.3% | 2.6% | 0.9% |
| tenderness | (dose 1): | 1.5% | 1.8% | 1.2% |
| | (dose 2): | 5.1% | 2.9% | 2.7% |
| 14-day questionnaire: nodule on day 14 | | | | |
| | (dose 1): | 9.2% | 5.1% | 1.7% |
| | (dose 2): | 11.1% | 4.9% | 2.6% |
| Local reaction, one or more, within 14 days | | | | |
| | (dose 1): | 15.4% | 11.7% | 2.6% |
| | (dose 2): | 28.9% | 19.0% | 14.5% |
| Examination at dose 2/ post-vaccination blood sampling: remaining nodule | | | | |
| | (dose 1): | 1.9% | 0.9% | 0.4% |
| | (dose 2): | 0.3% | 0.1% | 0.0% |

One must bear in mind that the "placebo" was a highly noxious substance (see above) and not just a (sugar or lactose) placebo in the true sense.

The rate of adverse reaction was quite high and cannot by any means be described as mild or as "no serious systemic reactions". Hypotonia, twitching, vomiting, persistent crying and drowsiness usually signify brain swelling or encephalitis.

During the fifteen-month follow-up, starting from 30 days after the second dose, a number of cases of whooping cough occurred in all three groups. The cases of whooping cough were divided into four

categories: 1. Culture confirmed; 2. serologically confirmed; epidemiologically linked, and 4. clinical only.

The authors emphasised that both vaccines "protected" th recipients against the bacteriologically confirmed pertussi However, it has been published previously that in vaccinate individuals it is generally difficult bacteriologically to isolate an confirm *Bordetella pertussis*. This observation is not a reflection o measure of protection. There has been no effort to explain wha protected hundreds of babies in the "placebo" group who did no contract whooping cough.

Importantly, no correlation was found between post-vaccination serum concentrations of antibodies and subsequent protection against whooping cough. Health authorities concluded that the biological mechanisms for protection by pertussis vaccine remain unknown and the role of cellular immunity and secretory antibodies in parenteral administration of pertussis vaccines needs further study.

**Pertussis vaccines and Systemic Bacterial Infections**

Perhaps the most interesting observation during this trial was the incidence of systemic bacterial infections. Eleven babies in the two vaccines group contracted invasive bacterial infections associated with *Hæmophilus influenzæ*, *Meningococcus*, *Streptococcus pneumoniæ*, *Streptococcus mitis*, *Klebsiella pneumoniæ* and *Staphylococcus aureus*.

Four babies died (one additional child died of neuroblastoma). Five babies in the "placebo" (diluent) group contracted systemic bacterial infections associated with *Streptococcus pneumoniæ*, none with *Hæmophilus*, and none died.

This demonstrates that the vaccine diluent is not an innocuous substance even on its own, but, when combined with the bacterial and viral antigenic components, represents quite a noxious cocktail. The writers [Storsaeter *et al.* (1988)] did not admit to any causal connection between pertussis vaccine injections and the above systemic infections. However, they stated that they could not refute the causal relationship either.

The trial of the two Japanese acellular pertussis vaccines ended with Professor Hans Wigzell, head of the National Bacteriology Laboratory in Stockholm, issuing the following statement:

*"The National Bacteriological Laboratory now withdraws the application for licencing of a Japanese pertussis vaccine after consultations with the Division of Drugs, Board of Health and Welfare. The vaccine was studied in a large clinical trial of acellular pertussis vaccines which was finished in the autumn of 1987. The Division of Drugs judges that the efficacy of the vaccine may be lower than that of whole-cell vaccines. The uncertainty about a possible association with deaths due to serious bacterial infections, which occurred among vaccinated children, has also contributed to the recommendation made by the Division of Drugs of comparative trials between acellular pertussis vaccines and well-known whole-cell vaccines".*
(Anonymous, 1989).

Is there or is there not a causal relationship between pertussis (and other) vaccines and the high incidence of invasive bacterial infections including meningitis in babies in industrially developed countries, with special emphasis on *Hæmophilus influenzæ* type b?

Michaels (1971) reported an increase in *Hæmophilus influenzæ* meningitis admissions at the Pittsburg Children's Hospital, Pennsylvania. No evidence was found that it was due to increased referrals to this hospital, improved laboratory methods or change in age distribution. While there has been an approximately 50% rise in total admissions during the past 25 years, there was a more than 400% increase in admissions due to *Hæmophilus influenzæ* meningitis during the same period. Specifically, there has apparently been no recent change in the age distribution of influenzal meningitis in the Pittsburgh area and no disproportionate increases in older children or newborn babies.

The increase in *H. influenzæ* meningitis was only recorded in babies 3 months and up to one year of age. Below that age, the incidence of meningitis remained unchanged.

Cartwright *et al.* (1986) wrote that since 1940, when there were over 12,000 notifications in England and Wales, meningococcal disease has been much less common. Following the last peak in 1974 (1,296 notifications), the annual number of notifications declined steadily until 1984 when only 401 cases were notified. In 1985, 549 cases were notified and the rise has continued into the first quarter of 1986.

Since 1970, group B strains have predominated to an increasing degree and now (1986) account for about 60% of infections. Smith *et al.* (1991) dealt with meningococcal disease in Norway (1935-1990). They demonstrated a number of things. Firstly, there was an overall increase since 1942 of 400% in meningococcal disease but only in children 3 months and older. The rate of occurrence in babies below this age remained the same as it was in 1942. Secondly, between 1942 and 1965 the incidence fell gradually and substantially to an almost pre-1942 level and then started climbing after 1965.

These three articles — Michaels (1971), Cartwright *et al.* (1986) and Smith *et al.* (1991) — provide very important clues for understanding and explaining the increased incidence of meningococcal diseases in babies between three months and one year of age. In all three instances one can correlate this increase with the introduction in the 1940s of mass vaccination with pertussis and with later oscillations in the public acceptance of pertussis vaccine and compliance to vaccinate.

Perhaps the best example is the known major fluctuations in vaccine acceptance, especially in England. Up until 1974 the public acceptance of pertussis vaccine was very high — around 85%. However, after public disclosure of encephalitogenic effects of the pertussis vaccine, the compliance fell to as low as 10% in most of Great Britain. This was quite clearly accompanied by a substantial fall in incidence of meningococcal meningitis. The same is valid for the other two examples. The renewed push for mass vaccination in all industrially developed countries, with the exception of Italy, Hamburg and Sweden, was accompanied by a substantial rise in the incidence of invasive bacterial meningitis.

The age distribution of the increases also provides a telltale story: the increase has been experienced only in babies of vaccination age — from 3 months upward. The Swedish trial provided important clues also in that the vast majority of invasive bacterial diseases occurred after the second dose of the vaccines. This is again very compatible with the observed and documented peak occurrence of invasive bacterial infections between 6 and 11 months of age, clearly showing the same thing — most babies will have had their second DPT shots by six months or later, accompanied by accentuation of susceptibility to infections after repeated injections of foreign antigens such as DPT vaccine [Craighead (1979)].

After the Japanese acellular pertussis vaccine fiasco in Sweden, Kimura and Kuno-Sakai (1988) sprang to the defence of the Japanese acellular vaccines. In their figure showing age-specific mortality rates for sepsis and meningitis in Japan since 1971, despite saying something else, they show that there indeed was a substantial and consistent fall in the incidence of these diseases in 0 to 1 year olds with the decreasing compliance to vaccinate. Since 1975, when the vaccination age was moved to 2 years, the incidence of these diseases in 2-year-olds clearly and sharply increased with the increasing acceptance of pertussis vaccine, especially after 1983.

Perhaps the best direct evidence of the causal relationship between administration of pertussis vaccine and the occurrence of invasive infections comes from the case of a 33-year old nurse [Boulton-Jones *et al.*(1974)]. In December 1969 she presented with an influenza-like illness associated with fever and red indurated lesions on her right leg. In March 1970 she was admitted to hospital with cellulitis of the right leg. She was treated with antibiotics and discharged, only to be re-admitted in June 1970 with a further crop of indurated red lesions around the right knee. She had fever and was found to have renal failure due to a severe proliferative glomerulonephritis with hypo-complementæmia and cryoglobulinæmia.

The problem continued until, in March 1972, she had two generalised seizures. A search of her bedside locker revealed phials of DPT vaccine and used needles and syringes. She admitted to

injecting herself with 2ml of DPT vaccine every two months over the previous four years. This case is quite instrumental in showing a range of adverse reactions to DPT vaccine, including cellulitis, one of the assortment of invasive infections occurring in babies at the age when the DPT vaccine is usually administered.

Previously, Bishop *et al.* (1966) described a case of a male prisoner who volunteered to produce hyperimmune serum and was repeatedly injected with DPT vaccine. Within weeks (after the eighth injection) he developed fever, arthropathy, lymphadenopathy and proliferative glomerulonephritis and died of renal failure three months later.

Repeated injections of antigens often lead to hyperimmunisation associated with the above-mentioned complaints and also invasive infections [infective endocarditis: Amsel *et al.* (1986)]. The authors described a 3 month old child who developed myocarditis several hours after diphtheria, tetanus and pertussis vaccination. The child had been normal and playful until 12 hours after administration of the second DPT vaccine, when it became irritable and experienced mild respiratory difficulties. X-ray examination showed generalised cardiac enlargement. The infant remained severely distressed for 48 hours, after which time a moderate gradual improvement was noticed. The cardiac enlargement showed progressive resolution, as well as the electrocardiogram, and heart rate dropped smoothly. At seven months of age the baby was given oral polio vaccine without adverse reaction. DPT b oosters were not administered.

The authors believed that the above reaction was associated with DPT vaccine, the pertussis or diphtheria component being the possible provocation of the cardiac damage.

In contrast to experimental animals, the dosage causing considerable problems in humans was much smaller.

This represents a serious warning that repeated injections of DPT are a well-defined causal agent precipitating a host of health problems in small babies. The demonstrated chronological large-scale increases in the incidence of invasive bacterial infections, especially those associated with ubiquitous commensals like *Hæmophilus influenzæ*, and following directly the pattern of

acceptance of the pertussis vaccine, are very convincing evidence that there indeed is a causal relationship.

The incidence of invasive bacterial infections, especially those associated with *Hæmophilus influenzæ*, is 52-87.5 per hundred thousand in white Australian babies [Clemens and Gilbert (1990)] and up to 991 per hundred thousand in Central Australian Aboriginal babies [Hanna (1990)].

The high incidence of invasive bacterial infections, and especially the high mortality rate (10% or more) and the 25% or more incidence of serious residual CNS complications, are sufficient reason to follow the Swedish example and discontinue the use of pertussis vaccine. The demonstrably doubtful benefits of the vaccine purported to prevent whooping cough, coupled with other, well-documented, side effects, should justify such prudent action.

When Japan moved the vaccination age to two years, the entity of cot death in that country disappeared [Cherry *et al.* (1988)], while the amount of adverse reactions in 2-year-olds remained the same or increased. At the present time, Japan has the lowest infant mortality in the world, followed by Sweden. In contrast to this, the US infant mortality is so high that it puts that country in the 20th place.

The enforced mandatory vaccination of all babies starting at the tender age of two to three months is the direct cause of this unsatisfactory situation — especially since it can be demonstrated that cot death is the single biggest cause of infant death. It has also been demonstrated that DPT injections are a major factor causing cot death [Scheibner (1991), Karlsson and Scheibnerova (1991)].

The Swedish trial sent-shock waves through the pædiatric community, especially in the United States. Pædiatricians were especially concerned about the high incidence (with 4 deaths) of invasive bacterial infections in the recipients of the two Japanese acellular vaccines.

Davidson *et al.* (1991) carried out a two-part study in Alaskan native children to evaluate the potential risk of invasive bacterial disease and the occurrence of minor illnesses after immunisation with diphtheria and tetanus toxoids and whole-cell pertussis vaccine.

They concluded that despite high rates of invasive bacterial disease and nearly complete DTP immunisation status in this population, no consistent relationship could be demonstrated between DTP immunisation and susceptibility to infectious diseases.

However, this is not true. Based on their raw data, the authors unwittingly provided evidence that there indeed was a causal relationship between DPT injections and the occurrence of invasive bacterial infections. 1. The highest incidence of invasive disease occurred after the 3rd DTP. This is compatible with the well-documented process of sensitisation to infectious diseases by repeated injections of foreign antigens.

2. The authors set an arbitrary 30-day limit for including the onset of invasive bacterial disease. In fact, the maximum incidence of invasive diseases was in the 31-60 day period.

3. The authors hypothesised that a cause-and-effect relationship would manifest itself in a shortened interval between vaccine injection and onset of the disease for the cases. This hypothesis is nothing more than an assumption. The real time relationship was 31-60 days.

4. The concept that both cases and controls are given DPT injections is wrong. The true controls should be unimmunised. The Swedish trial and other published information brought evidence indicating a causal relationship between pertussis vaccine administration and the occurrence of invasive bacterial infections.

It is quite clear that not all babies injected with DPT would contract invasive bacterial disease within 30 or even 31-60 days of vaccination. This does not mean that a certain number would not. Comparing two groups of immunised children is meaningless.

Another article endeavouring to show no causal relationship between DPT immunisation and invasive bacterial infections is that by Black *et al.* (1991). The authors claimed that they found no relationship between injections of childhood immunisations and incidence of invasive bacterial infections. Between January 1986 and December 1988, 223 cases of invasive bacterial disease were identified by the Kaiser Permanente Medical Care Program Regional

Microbiology Laboratory. Of these cases, 144 were associated with *Pneumococcus*, 58 with *Hæmophilus influenzæ*, seven with *Escherichia coli*, five with *Meningococcus* and nine with other organisms. Most of these diseases occurred between five and fifteen months of age. Interestingly, Black *et al.* (1991) also show that most cases occurred between 31 and 60 days after DPT and oral polio vaccine (OPV) vaccination. Also, they demonstrated that children in day-care were both more likely to be vaccinated and at a higher risk of contracting invasive bacterial infections. This was the second most important indicator of the cause-and-effect relationship. The last sentence in this paper is worth quoting:

> *"it is possible that the apparent protective effect is related to good health practices that are only partially reflected in well-child care visits"*.

The third paper trying to dispell any concerns about the cause-and-effect relationship between childhood immunisations and invasive bacterial infections is that by Griffin *et al.* (1992).

The authors studied a cohort of 64,591 children immunised through Tennessee county health clinics who had a total of 158 episodes of invasive bacterial infections after a DPT immunisation. Eight cases of invasive diseases occurred within 7 days following DPT injections. Seven infections occurred in the 8-14 day interval, and twenty in the 15-28 day interval following DPT injections. Instead of providing reassurance that DPT vaccine is not associated with a large increased risk of serious bacterial infections, the obvious clustering of cases provided evidence that there indeed is an increased risk of invasive bacterial infections shortly after DPT injections.

Just how shocked the American pædiatric community was by the fiasco of the Swedish trial of Japanese acellular vaccines is demonstrated by the fact that the American Academy of Pediatrics, in their article published in Pediatrics (1992), wrote that *"A licensure application has been made for B-type vaccine based in part on the results of this Swedish Trial"* and did not add that the same application for licensure of one of the acellular Japanese vaccines

was withdrawn. Neither did they admit that Sweden has not resumed pertussis vaccination after the Swedish trial. Instead, the American Academy of Pediatrics recommended that all American infants be immunised with five doses of pertussis vaccine beginning at two months of age. The initial series of three doses should be given with the whole-cell preparation. The new acellular vaccine should be used only for the fourth and fifth doses for children older than 15 months and younger than 7 years.

**Japanese experience of whooping cough vaccination**

Japanese experience in pertussis epidemiology and vaccination in the past thirty years was described by Kanai (1980). As in other industrially developed countries, whooping cough in Japan has undergone major changes. Pertussis incidence declined to such a low level that in 1974 only some 393 cases were reported with no deaths.

From 1947 to 1950, while Japan was recovering from the war and housing and sanitation were in poor condition, the incidence of whooping cough was quite high (more than 100 cases per 100,000). In 1950 nation-wide pertussis vaccination was mandated by the US occupational forces. Mortality from whooping cough fell sharply at about the same time, most probably due to the introduction of antibiotics.

Over the next 20 years socio-economic conditions improved vastly and pertussis incidence declined substantially. There were only sporadic outbreaks. A surveillance of one of these outbreaks showed that out of 252 cases (only 25 were reported to the authorities) 123 were confirmed bacteriologically. Of these 123 cases, 55 were vaccinated and 65 unvaccinated. In eight cases the vaccination status was not sure. This example shows that vaccination was totally ineffective.

After two cases of death following whooping cough vaccination in 1974 and 1975 [Sato *et al.* (1984)], doctors in the Okayama Prefecture boycotted the vaccine. According to Noble *et al.* (1987) there were 57 reported severe reactions with 37 deaths between 1970 and 1974. So the two deaths in 1974-75 were just the last straw which

resulted in the raising of the minimum vaccination age to two years. In 1975, 72 cases of whooping cough were reported in the Okayama Prefecture and 300 in the surrounding prefectures.

Sato *et al.* (1984) estimated that by the end of 1979 the total whooping cough notifications in Japan would have probably reached 10,000. In 1981 Japan introduced a series of acellular pertussis vaccines which were supposed to be less reactogenic. Sato *et al.* (1984) reported that the pertussis component vaccine was less reactogenic than the whole cell (pertussis dead-cell) vaccine.

However, Aoyama *et al.* (1986), when describing the results of a trial with 115 children, ranging in age from 3 to 23 months, showed that local adverse reactions started about 7 days after the first DPT vaccination and 48 hours after the second, third and booster DPT injections.

The incidence of local reactions tended to be higher after the second, third and booster shots than after the first for all three lots of acellular DPT. Also, a very high percentage (up to 48.8%) of children had some local reactions like palpable induration, redness more than 1 cm in diameter and redness more than 5 cm in diameter.

Practically every child, independent of age, had some form of local reaction. The authors concluded that there was no difference in local and systemic reactions between the three acellular vaccines (lot A, lot B and lot C). However, their table III shows clearly that the three listed local reactions (palpable induration, redness more than 1 cm and redness more than 5 cm) affected 9.1%, 22.1% and 1.9% of children after lot A; 37.6%, 30.6% and 3.5% after lot B and 24.8%, 48.8% and 3.3% of recipients after lot C.

Noble *et al.* (1987) also concluded that the incidence of more serious local reactions and high temperatures (both without sequelæ) may be more common after vaccination with acellular vaccines. There was also only a slight decrease, if any, in the incidence of serious reactions with sequelæ in children over two years of age. The authors hoped that some questions regarding product-specific and age-specific efficacy may be answered by the ongoing field trials of Japanese acellular pertussis vaccines begun in 1986 in Sweden.

Aoyama *et al.* (1988), when writing about type-specific efficacy of acellular pertussis vaccine, mentioned that the acellular vaccine was quickly introduced into widespread use before characterisation of pertussis antigens contained in the vaccine was completely known. At the time of its introduction, the only requirement of efficacy for Japanese acellular pertussis vaccine was its potency, determined by intracerebral mouse protection test.

The survey of 442 household patients with pertussis identified 440 children and 987 adults who represented household contacts. Thirteen primary cases of children and 21 primary cases of adults were excluded. The secondary attacks in this study were 88% in unimmunised children aged 0 to 1 year and 61.3% in those aged 2 to 8 years. In fully immunised children aged 2 to 8 years, the attack rate was 10.8% in accellular vaccine, 13.5% for whole-cell vaccine and 14.3% for both. The attack rates were much higher in partially immunised children.

However, in the 442 household contacts surveyed, 40 patients with primary cases, 21 with co-primary cases and 77 with secondary cases were adults. Ten transient carrier adults were identified in whom *B. pertussis* was isolated. Five of them had only a mild cough and the remainder were asymptomatic. Thus adults represent an important source of pertussis infection and the authors suggest possible vaccination of adults with the acellular vaccine — after a full assessment of safety and duration of immunity before introduction of booster injections. This statement is rather interesting considering that acellular vaccines were introduced for use in children in Japan before their safety and efficacy was fully ascertained.

Kimura and Sakai (1990) summarised the development in pertussis vaccination in Japan. In 1973-74, 20 out of 50 children who contracted pertussis received between one and four doses of the whole-cell vaccine, whereas in the 1982-84 study, only 16 (4%) of 116 children with pertussis had received between one and four doses of the acellular vaccine. Compared with the Swedish trial of two Japanese acellular vaccines, these studies show much higher efficacy. In the Swedish trial, one vaccine (JNIH-6) was found 69% effective;

the second (JNIH-7) only 54%. However, in the Swedish trial 2,800 children were given these vaccines, compared with only a few hundred studied in the above-mentioned Japanese follow-ups.

The most important lesson from the Japanese experience is that when the vaccination age was moved to two years, the entity of cot death disappeared [Cherry *et al.* (1988)].

### DPT Vaccination Mandatory in the United States

DPT vaccination is mandatory in the United States. While a great number of papers on the vaccination problems were being published in US medical journals, mass vaccination has been widely practised and enforced, and doctors adopted a general attitude of rejecting any connection between the observed adverse reactions (convulsions, hypotonic-hyporesponsive episodes), and especially of deaths, and the administered vaccines. This effort to minimise or disregard a causal relationship between vaccine injections and untoward reactions was probably precipitated by fear of upsetting the mandatory use of vaccines and fear of accepting responsibility.

Among the best evidence of the dangers of pertussis vaccine is the paper presented by Hutchins *et al.* (1988). The incidence and mortality from whooping cough fell steadily between 1922 and 1975. In 1978 a nationwide immunisation initiative was begun; legislation was passed requiring proof of immunisation for school entry at five to six years of age. Almost immediately the overall incidence of pertussis in the United States trebled, especially in the age group below six months. This age group also experienced the highest mortality from whooping cough.

In 1992 Pichichero *et al.* published an evaluation of immunogenicity of and adverse reactions to a two-component acellular pertussis vaccine when given as a primary immunisation series at 2, 4 and 6 months of age. The authors concluded that this acellular vaccine produced greater immunogenicity and fewer adverse effects than the currently licensed whole-cell vaccine.

However, one has only to look at the number of withdrawals, and the reasons for withdrawal of babies from the trial, to see that this

optimistic statement is incorrect: 31 of the 380 children withdrew from the study; 13 of 285 withdrew from the acellular vaccine group 6 refused the follow-up phlebotomy; 6 moved from the study area and 1 parent declined continued participation due to the infant's *"excessive irritability"* after the first dose. Eighteen babies out of 95 withdrew from the whole-cell vaccine group: 7 had reactions sufficiently serious for the parents and/or investigator to discontinue participation in the study; 3 had true high-pitched crying; 2 refused the follow-up phlebotomy; 2 experienced hypotonic/hyporesponsive episodes following the second vaccination; 3 moved away and 1 child developed culture-confirmed pertussis after the first injection. Also, a high incidence of drowsiness and irritability were reported in recipients of both vaccines and a higher than expected rate of unusual "high-pitched" crying was noted.

The authors' comment *"Given the prolonged disruption in the infants' routine schedule for enrolment, vaccination and post-vaccination observation, as well as for venipuncture, it is not surprising that many of the children were tired and irritable"* seems quite out of place considering the well-known and much published findings of Selye (1978) that overproduction of mineralocorticoids CODs due to exposure to non-specific stress syndrome causes brain lesions accompanied by drowsiness and irritability. The total oblivion of doctors to encephalitogenic effects of vaccines administered to young babies is quite incredible, to say the least.

The proponents of vaccination have another difficulty, namely difficulty in seeing the most obvious: pertussis vaccines are ineffective in preventing whoopping cough. A number of papers published over the years either try to camouflage this fact or claim that the vaccine made whooping cough milder. A recent example of the former is an article by Etkind *et al.* (1992) on pertussis outbreak in groups claiming religious exemptions to vaccination. The title of the paper and the abstract leave nothing to the imagination: the case is presented as a clear-cut example of an outbreak of whooping cough in unvaccinated children. But under the heading "Results" the authors wrote: *"The immunisation status of patients and the*

*diagnostic testing performed in each outbreak are summarized in tables 1 and 2, respectively".*

Table 1 says that in outbreak 1, of the total number of 25 cases, 5 patients were fully immunised, 2 under-immunised and 10 were patients claiming exemption; 8 patients were over 18 years of age. In outbreak 2, of the total 26 patients, 3 were fully immunised, 1 under-immunised, 17 claimed exemption; 5 patients were over 18. In outbreak 3, of the total 16 patients, 1 was immunised, none under-immunised and 13 claimed exemption; 2 were older than 18. In outbreak 4, of the total 46 patients 7 were fully immunised, 5 underimmunised and 32 claiming exemption. 2 were older than 18.

Nobody would claim that unimmunised children would not contract whooping cough during an epidemic or an outbreak. The unimmunised children represented 49% of all school children, and, behold, in outbreak 1, 41% of cases were immunised. Since not all unvaccinated cases contracted whooping cough in these outbreaks, the authors should first explain what protected those unimmunised children who did not contract whooping cough.

Since the title and the abstract of the paper does not disclose the reality of this outbreak of whooping cough, namely, that 25% to 49% of cases were in fact immunised, the authors should explain what they were trying to achieve by hiding this important fact from the readers.

Pertussis vaccine quite obviously did not provide any protection against contracting whooping cough to its recipients, so the authors'claim that pertussis can be prevented by immunisation is not only untrue and incorrect, but also totally absurd. Had all children been vaccinated in this particular case, then the report would have had to deal with an outbreak of pertussis in a fully vaccinated population, as did many other published reports.

Indeed, Halperin *et al.* (1987) reported on the persistence of pertussis in an immunised population in Nova Scotia. During the 28 months of enhanced surveillance, 526 cases of pertussis were identified (74/100,000 population). Most (91%) patients had received at least three doses of pertussis vaccine.

By supplementing culture techniques with immunofluorescent staining and serologic methods, they increased the laboratory confirmation from 17% to 65%, suggesting that strict clinical criteria accurately reflect the incidence. The authors concluded that pertussis remains a significant health problem in Nova Scotia despite nearly universal vaccination.

Sutter and Cochi (1992) studied pertussis hospitalisation and mortality in the United States between 1985 and 1988, and concluded that there is a substantial under-reporting of pertussis. Based on these indicators, the national health impact of pertussis is considerably higher than previously published reports have suggested. Applying the age-specific hospitalisation rates, 187,867 to 515,930 cases of pertussis may have occurred during the study period; instead only 14,057 cases were reported to the CDC. The reported efficacy was found to be only 2.7% to 7.5%. Using different methods of estimation, approximately 121,340 cases of pertussis may have occurred during the study period, indicating11.6% efficacy.

Integrating two different projections derived from US and non-US sources, an estimated 30,000 or as many as 125,000 cases of pertussis may have occurred annually in the United States. These projections suggest that pertussis has remained an important public health problem in the United States, and certainly one of much greater magnitude than published data on reported cases alone would indicate. Considering that mandatory vaccination would assure vaccination of up to 95% of all babies, the efficacy, if any, of pertussis vaccine, is very low indeed.

The authors concluded that their study, together with recent information documenting silent transmission of *Bordetella pertussis* in families, and increasing recognition of the contribution of adults with asymptomatic infection or mild disease in transmitting the disease, suggests that control of pertussis had not yet been achieved in the United States. They continued by saying that to achieve improved control and ultimate eradication of pertussis, a vaccine that not only prevents clinical disease but also prevents infection may be required. Until such vaccine is developed high vaccination coverage

with the existing pertussis vaccine and prophylaxis of persons in contact with patients will continue to be the most effective means to further reduce the health impact of pertussis. Blindness to the truth, perhaps?

Proponents of vaccination are so enmeshed in their belief in the efficacy of vaccines that they appear totally oblivious to evidence to the contrary. They are also totally blind to the observed fact that most unvaccinated children do not contract the disease — even after exposure. Perhaps the most instructive example of this is the Swedish trial of two Japanse acellular vaccines. After the trial was completed several published papers discussed the efficacy of the vaccines. However, nobody commented on one very obvious fact: the vast majority of children who were given the "placebo" (vaccine diluent) did not contract whooping cough during the follow-up period of some19 months. They should have asked that one all-important question: just what exactly protected these children? Because the same factor must also be protecting immunised children from contracting whooping cough, and not the vaccine.

**Do DPT Vaccines Cause Serious Side-effects?**

The pertussis component of the triple antigen or DPT is often blamed for all adverse reactions to this vaccine.

Cody *et al.* (1981) compared the incidence of adverse reactions to DPT and DT and concluded that the rate of reactions to DT was much less than to DPT. However, the number of babies in the DT group was too small to provide meaningful figures. As the previous example shows, with comparable and large numbers in both groups, the rate of adverse reactions is very similar after both vaccines.

The Workshop on Neurologic Complications of Pertussis and Pertussis Vaccination [Menkes and Kinsbourne (1990)] concluded

1. Vaccines are not standardised between manufacturers;
2. for a given manufacturer, vaccines are not standard from one batch to the next;
3. unless the vaccine is properly prepared and refrigerated, its potency and reactivity varies with shelf-life.

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1. Vaccines are not standardised between manufacturers;
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3. unless the vaccine is properly prepared and refrigerated, its potency and reactivity varies with shelf-life.

Since neurological damage is not specific to pertussis vaccination, its temporal relationship to vaccination is critically important for determining its cause.

It was the consensus of the workshop that there was no inherent difficulty in assigning cause and effect to the vaccine and subsequent permanent neurologic damage. The workshop concluded there was sufficient experimental data to implicate both endotoxin and pertussis toxin in adverse neurologic reactions to pertussis vaccine.

Also, it was the consensus of neurologists that, although the majority of seizures following pertussis vaccination are associated with fever, they could not be described as febrile convulsions because they are not necessarily benign.

Astonishingly, in the face of abundant evidence to the contrary, the participants concluded *"that there was no demonstrated association between DPT vaccination and SIDS, because sudden death after pertussis vaccination is too rare to be detectable in the context of presently available series."*

There are seven to ten thousand cases of sudden infant death per year in the US alone. With DPT vaccination mandatory there can be no doubt the majority of these deaths are attributable to the vaccine.

Cherry (1990) editorialising in *Journal of the American Medical Association*, clearly labelled vaccine encephalopathy a 'myth', accusing the American Academy of Pediatrics *"... and other well-meaning physicians ..."* of joining forces with parents groups and lawyers as well. The result of this is the National Vaccine Injury Program.

*"... this, unfortunately, is the new national tragedy, because it has legitimized ideas as to causation that were made by the special interest groups and by physicians not trained in epidemiologic science... The published Vaccine Injury Table is now used in conjunction with the published vaccine contra-indications in what I believe is increased litigation against physicians.*

*"We need to end this national nonsense ... we need vaccine information relating to risks and benefits that represent true*

*scientific evidence (such as reviewed in this report) and not the wishes of special interest groups. ... Last, new vaccines are needed, not to prevent non-existent problems such as 'pertussis vaccine encephalopathy' but to decrease the many disquieting reactions, such as high fever, persistent uncontrollable crying, and hypotonic hyporesponsive state, that do occur with presently available wholesale pertussis vaccines."*

Cherry (1990) was answered when, in 1992, the Institute of Medicine finally admitted that:

*"... the evidence is consistent with a causal relation between DPT vaccine and acute encephalopathy (defined in the controlled studies reviewed as encephalopathy, encephalitis or encephalomyelitis) and shock and 'unusual shock-like state' ..."*

*" ... the evidence indicates a causal relation between DPT vaccine and anaphylaxis, between the pertussis component of DPT vaccine and protracted, inconsolable crying ...".*

## Animal Experiments and Adverse Reactions to Pertussis Vaccine in Babies

Levine *et al.* (1966), Levine and Sowinski (1973) and others wrote that pertussis vaccine has been employed as an immunologic adjuvant in a number of different experimental systems. The adjuvant property of pertussis vaccine for production of experimental allergic encephalomyelitis (EAE) has been demonstrated in many different ways. Pertussis vaccine administered peritoneally to mice or rats enhanced the encephalitogenic effect of central nervous system antigen administered separately by a different route several days later.

*Bordetella pertussis* organisms, like mycobacteria, enhanced development of EAE when incorporated with oil and CNS antigen in water-in-oil emulsion. When pertussis vaccine has been used as an adjuvant for production of EAE by admixture with aqueous CNS suspension, the disease was not only intensified and accelerated, but also changed its histologic character [many polymorphonuclear

leukocytes, much swelling (edema) and fibrin in the perivascular exudates] and could be described as "hyperacute".

Clinical signs of EAE were limp tail, weakness, ataxia, paralysis, urinary retention and loss of weight. In an initial experiment, groups of five rats were given CNS antigen intraperitoneally, without any pertussis vaccine, or with pertussis vaccine injected intradermally in a remote site (back of the neck).

They developed ordinary EAE after an average latent period of 10 days, had mild clinical signs, no mortality and no fibrin in the perivascular lesions. A mixture of 0.05 ml pertussis vaccine concentrate (10 billion organisms) with 200 mg cord tissue was injected into the peritoneum (i.p.) of five other rats. Eight days later all were paralysed or dead. Many vessels contained fibrin-rich perivascular exudates, typical of the hyperacute form of EAE.

When guinea pig (spinal) cord homogenate was mixed with progressive twofold dilutions of pertussis vaccine concentrate and various dilutions tested, all but one rat had the onset 6 to 8 days after inoculation, with rapid progression to paralysis. However, only the three highest doses produced hyperacute EAE.

The ability of pertussis vaccine to convert EAE to the hyperacute form was eliminated by heating the vaccine to 80 deg C for 30 minutes, but was unaffected by 56 deg C for 30 minutes. It was only slightly decreased by adding acetone, centrifuging and resuspending in saline. Saline extract of pertussis cells was prepared and treated with formaldehyde. This treatment caused a marked reduction in histamine sensitising potency. It also reduced the adjuvant effect. None of the rats treated with this extract and CNS homogenate became paralysed, while four or five rats, treated with the original saline extract and CNS homogenate, did become paralysed.

Cavanagh *et al.* (1981) described three cases of children with common viral infections and severe neurological illness, in whom there was a temporal relationship with either a natural infection with *B. pertussis* or with diphtheria, tetanus, pertussis (DPT) vaccine and oral polio vaccine (OPV).

A 16-month-old girl, who had developed normally, became irritable and febrile twelve hours after her third injection of DPT and OPV. She had shown no reaction following her DPT injections at 4 and 10 months of age (except for lingering upper respiratory infection since 6 months of age and measles at 14 months). Over the next 24 hours she became drowsy and vomited; four days later she had a generalised convulsion. She became severely retarded and blind.

Munoz *et al.* (1981) studied various biological activities of crystalline pertussigen and found that, in mice, as little as 0.5 nanograms (ng*) of pertussigen induced hypersensitivity to histamine in 50% of the mice, 8-40 ng induced leucocytosis, 2 ng increased production of insulin, 0.1 ng increased production of immunoglobulin (Ig) E and Ig $G_1$ antibodies to hen egg albumin, 9.5 ng increased susceptibility to anaphylactic shock, and 0.5 ng increased the vascular permeability of striated muscle. In Lewis rats 20 ng of pertussigen produced hyperacute allergic encephalomyelitis (EAE).

(*One nanogram is one thousandth part of one millionth part of a gram: $10^{-9}$g).

Pertussigen given intraperitoneally (i.p.) was toxic to mice at a dose of 546 ng. Typically, the deaths were delayed, as has been reported for preparations of late-appearing toxic factor. After treatment of pertussigen with glutaraldehyde this toxicity and some of pertussigen's other activities were reduced. Mice injected with 1,700 ng of detoxified pertussigen were protected against intracerebral challenge with $3x10^4$ viable *Bordetella pertussis* cells. However, at the otherwise well-tolerated 100-200 ng doses the preparations failed to protect mice.

When as little as 0.5 ng of pertussigen was given intravenously (i.v.) to mice, the increased susceptibility of the animals to histamine could be detected 84 days later. When 5 micrograms of pertussigen was administered, most mice did not gain weight and the mice started dying by day 5. The last mouse died on day 8. The authors do not point this out, but even the surviving mouse had a crisis on days 3 and 5 when the weight gain levelled off before recovering.

A 1 microgram dose of one preparation killed four of five mice. They first gained weight from days 2 to 5 and then remained at nearly constant weight until they died. The biological properties of crystalline pertussigen indicated their similarity to leucocytosis-promoting factor, islet-activating protein, late-appearing toxic factor and mouse-protective antigen of *B. pertussis*.

Pertussigen induced an increased production of insulin by the beta cells of the pancreas of mice at a dose of 2 ng. Indeed, Hewlett *et al.* (1983) demonstrated that administration of whole-cell killed pertussis vaccine caused hyperinsulinæmia and enhanced secretion of insulin in response to a variety of secretagogues in rats and mice. They discovered two separate effects of the bacteria on glucose and insulin levels in mice. Firstly, a heat-stable component caused a brief hyperinsulinæmia which is measurable by 1 hr, maximum at 8 hr and ends at 48 hr. Secondly, the heat-labile component of *B. pertussis* organism induces a sustained (more than 14 days) dose-dependent hyperinsulinæmia which reaches a maximum at 5 to 7 days and has no associated hypoglycæmia. The authors concluded that these findings may be important in the well-recognised reactogenicity of pertussis vaccine in humans.

Very similar effects were observed and recorded by Hannik and Cohen (1979) in human infants given pertussis vaccine. Who is to say that many doses of the vaccine are not toxic enough to produce EAE and lethal enough for individual babies to cause cot death? Certainly the time-pattern of death, showing clear clustering around critical days, is more than enough evidence that this indeed is true (Scheibner 1991, Karlsson and Scheibnerova 1991).

Iwasa *et al.* (1985) wrote that administration of pertussis vaccine causes a variety of untoward reactions in children. Encephalopathy and neurological disturbance, severe brain damage and even death, have brought on vaccination boycotts.

In their paper they reported that intracerebral injection of pertussis vaccine induced brain swelling in mice. And indeed similar brain-swelling in infants after administration of pertussis vaccine is described repeatedly in the literature.

Jacob and Manning (1979) described a case of a 7 month old baby who, 15 hours after the third DPT injection, experienced elevated temperature (38.9 deg C). Nine hours later the child's parents, a nurse and a pædiatrician, noticed that the anterior fontanelle was bulging and the child became irritable. Later, in the hospital, it was established that the child had increased intracranial pressure as measured by lumbar puncture. The child started recovering about 40 hours after the offending injection.

No amount of denial will cover up thousands of pages of papers reporting adverse reactions, including encephalitis and deaths, reported in medical journals over the decades of mass vaccination. It would be straining credulity too much to assume that thousands and thousands of cases of brain damage and tens of thousands of deaths following DPT vaccination were all coincidental.

## DPT VACCINATION - A LINK TO COT DEATHS

Deaths of infants after whooping cough vaccination were described as early as 1933, when Madsen published an article reporting on death of 2 infants within hours of being injected with the vaccine.

Werne and Garrow (1946) described deaths of identical twins within hours after their second shots. Doctors then recommended that vaccines should not be administered to newborn babies.

People have short memories.

On 9 March 1979, the Tennessee Department of Health reported to the Center for Disease Control that four sudden and unexplained deaths occurred since November 1978, in infants who had been vaccinated during the 24-hour period prior to death. All four deaths were classified as sudden infant death syndrome (SIDS), and all had received their first vaccination of diphtheria-tetanus toxoids-pertussis (DPT) vaccine and oral polio vaccine [Bernier *et al.* (1982)]. Altogether, 52 cot deaths and/or *"deaths resulting from unknown causes"* were recorded in Tennessee between August 1977 and March 1978, and from August 1978 to 15 March 1979.

Bernier *et al.* (1982) concluded that the evidence was adequate to indicate *"an unusual temporal association between DPT vaccination with lot A and SIDS"*. However, they also stated that *"Whether or not this temporal association reflects a causal relationship remains undetermined; we found no evidence to support such a causal relationship."*

William C. Torch presented a paper at the Thirty-Fourth Annual Meeting of the American Academy of Neurology in 1982, referring to the Tennessee deaths and to over 200 randomly reported SIDS cases. In his published abstract, Torch (1982) wrote that preliminary data on the first 70 cases studied showed that two thirds had been immunised prior to death. On the average, DPT one, two and three were administered at 2, 4, and 6 months of age. In the DPT SIDS group, 6.5% died within 12 hours of DPT injection, 13% within 24 hours, 26% within 3 days, and 37%, 61%, and 70% within 1, 2, and 3 weeks, respectively. There was a significant clustering of deaths within the first two to three weeks of DPT injections. He also wrote that deaths frequencies peaked at 2 months of age in the non-DPT group, and had a biphasic peak occurrence at 2 and 4 months in the DPT group.

Cot deaths occurred maximally in the autumn/winter season in the non-DPT group, but was nonseasonal in the DPT group. Deaths occurred mostly in sleep in healthy allergy-free infants after a brief period of irritability, crying, lethargy, upper respiratory tract symptoms and sleep disturbance. Pathology findings were typical of cot deaths: petechiæ (spot-like bleeding) of lung, pleura, pericardium and thymus; vascular conjestion, pulmonary and brain oedema (swelling), and pneumonitis.

His conclusion, quite correctly, was that

*"DPT vaccination may be a generally unrecognized major cause of sudden infant and early childhood death, and that the risks of immunization may outweigh its potential benefits. A need for re-evaluation and possible modification of current vaccination procedure is indicated by this study."*

According to Coulter and Fisher (1991), Torch was heavily criticised for his study and conclusions. Subsequently published "studies" of further cot deaths after vaccination concluded that there was no evidence of a causal link between DPT injections and cot deaths [Walker *et al.* (1987); Hoffman *et al.* (1987), and Griffin *et al.* (1988)].

Stewart (1979), when re-analysing eight of the Tennessee cot deaths after vaccination, stated that *"These incidents do not establish that DPT killed infants, but they show beyond doubt a highly significant, non-random clustering of an excess of undiagnosed sudden infant deaths following vaccination."* Finally, after the Tennessee deaths, in March 1979 the US Surgeon General intervened and the company which produced the vaccine withdrew all unused doses of batch 64201 of the vaccine.

Geraghty (1984), in his letter to the editor of Journal of Pediatrics, criticised Bernier *et al.*'s (1982) and Hoffman *et al.*'s (1982) treatment of the Tennessee cot deaths after DPT injections. He was especially critical of their conclusion that they found no evidence in support of reducing efforts to immunise infants with DPT. In Geraghty's opinion, this conclusion served to soften the concerns among clinicians that may have been raised by Bernier *et al.*'s (1982) earlier statement that the evidence seemed adequate to indicate an unusual temporal association between DPT vaccination with lot A and SIDS.

Hoffman *et al.* (1982) stated at the May 1982 meeting of the American Pediatric Society that, recently, two investigators had suggested DPT might be a generally unrecognised cause of sudden infant deaths. These references are to the work of Torch (1982) and Baraff *et al.* (1983).

Geraghty (1984) did not consider the matter of the link between DPT and cot deaths closed and supported the continued inclusion in the product insert of information regarding the clinical observation of the link without imputing causality.

Coulter and Fisher (1991) write that in 1984, Wyeth Laboratories' insert stated: *"The occurrence of sudden infant death syndrome*

*(SIDS) has been reported following administration of DPT. The significance of these reports is unclear. It should be kept in mind that the three primary immunizing doses of DPT are usually administered to infants between the ages of two and six months of age, with the peak incidence at age 2 to 4 months*". Also: *"In 1986, Connaught's insert stated,* 'SIDS has occurred in infants following administration of DPT', *but went on to state that one study showed that there was no causal connection."*

Baraff *et al.* (1983) investigated 145 SIDS victims who died in Los Angeles County between 1 January 1979 and 23 August 1980: 53 of these babies had received a DPT immunisation, 27 within 28 days of death. Six deaths occurred within 24 hours and 17 occurred within 1 week of DPT immunisation. These sudden infant deaths were "*significantly more than expected were there no association between DPT immunization and SIDS*". Baraff *et al.* (1983) also stated *"An additional 46 infants had a physician/clinic visit without DPT immunization prior to death. Forty of these infants died within 28 days of this visit, seven on the third day and 22 within the first week following the visit. These deaths were also significantly more than expected. These data suggest a temporal association between DPT immunization, physician visits without DPT immunization and SIDS."*

It may, perhaps, sound a bit confusing, that there was a significant association between sudden infant deaths, DPT injections and physician/clinic visits without DPT injections, but there is more to it than meets the eye. According to our results [Karlsson and Scheibnerova (1991), Scheibner (1991)] typical cot deaths are deaths due to exposure to non-specific stress syndrome, provoked by any injury, insult or noxious substance including upper respiratory tract infections, overtiredness or vaccine injections.

Baraff *et al.*'s clinical observations and data are a correct reflection and confirmation of our independent findings, and vice-versa. The seeming and widely perpetuated dilemma "*is there or is there not a causal relationship between DPT injections and cot death*" has, quite

adequately and indeed without a shadow of doubt, been resolved by the Japanese experience with cot death.

According to Cherry *et al.* (1988), *"the category of* 'sudden death' *is also instructive in that the entity disappeared following both whole-cell and acellular vaccines, when immunization was delayed until a child was 24 months of age."*

Speaking of a **temporal** relationship between DPT injections and cot death, as opposed to a **causal** relationship, is totally inappropriate semantics. It would be just too much of a coincidence that tens of thousands of babies die after DPT injections, yet none of these deaths would be caused by such a highly noxious substance as DPT vaccine. This is especially instructive since the vast majority of these babies often were healthy bouncing infants just hours before they were injected with DPT and died.

Nor is it just the pertussis component that acts as a highly noxious substance causing cot deaths. When Pollock *et al.* (1984) studied symptoms after routine primary immunisation of 6,004 infants with DPT vaccine and 4,024 infants with DT, some very interesting facts became quite obvious. First of all, in both groups, reactions of any kind increased with each subsequent dose. Differences in rate of reactions between vaccines were minor except for the third dose of plain DPT, when the rate of reactions was almost three times as high as reactions after the first and second dose. Reactions of any kind were much higher after the plain DPT.

There were 7 cases of cot death within 6 weeks of vaccination in the DPT group (at 4, 20, and 37 days) and 4 in the DT group (at 2, 5, 37 and 40 days). The child who died 20 days after DPT (a second dose of adsorbed vaccine) had been unwell since first vaccination with attacks of high-pitched crying 3 or 4 times a day for 10 days, followed by a period of sleepiness. She had a recurrence of high-pitched crying on the 16th day, 4 days before death.

Three children died before the first follow-up and the other three had no post- vaccination symptoms of any note; 3 other deaths occurred in DT- immunised cases within 6 weeks of immunisation (two from respiratory infection).

Three of the 7 cases died within the first week after vaccination. In one case, of a child who died 3 weeks after immunisation with DPT, the symptoms which preceded its death (diagnosed as cot death) were present since immunisation.

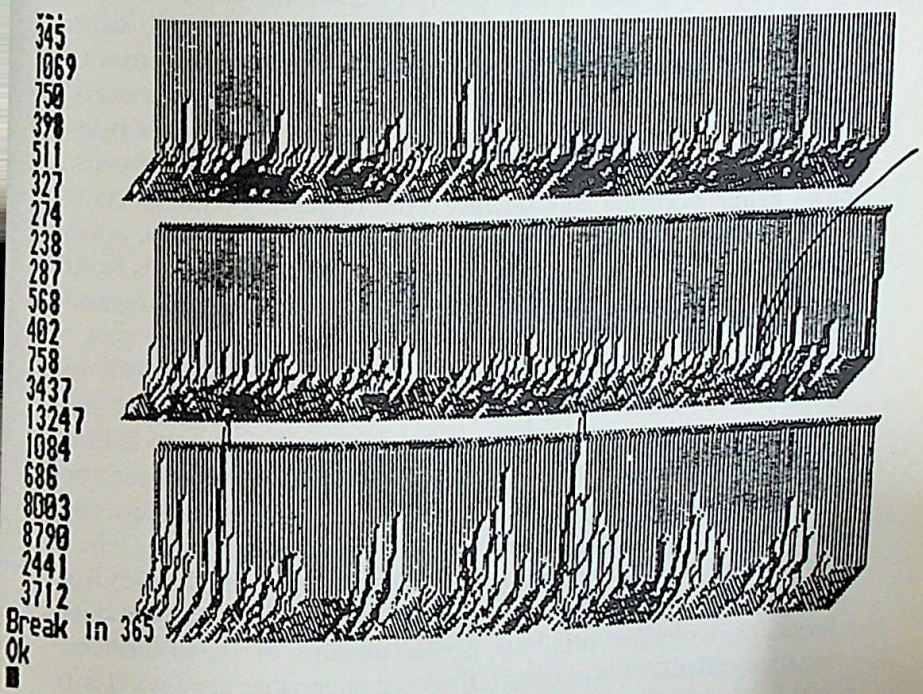
These clinical observations are straightforward and clearly show a causal relationship between vaccine injections (DPT or DT) and cot death. However, there is now truly scientific and objective evidence of this causal relationship: computer printouts of the records of breathing of babies after vaccination as recorded with the microprocessor Cotwatch breathing monitor (figures 1-4).

**DESCRIPTION OF THE RECORDS OF BREATHING IN BABIES AFTER VACCINATION (FIGURES 1-4)**

**FIGURE 1:** *(Facing page).* A 'raw' record of breathing of baby one. Every vertical line represents a histogram of events for one hour. Events from 6 to 15 seconds are mostly apneas (pauses in breathing), while the events above 15 seconds are mostly hypopneas (low volume breathing). Hypopneas represent low volume breathing (only 5% of the volume of unstressed breathing). They occur at critical hours and in clusters of several shorter episodes within 10-15 minutes and are associated with exposure to a great variety of stressors.

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

[For a more extensive discussion and explanation of the method and results of the micro-processor controlled Cotwatch breathing monitor, refer to Chapter 10].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1069
750
398
511
327
274
238
287
568
402
758
3437
13247
1084
686
8083
8790
2441
3712
Break in 365
Ok

**FIGURE 1**

**FIGURE 2:** *(Facing page)* This graph correlates the longitudinal record of breathing of two babies after DPT injections with the records of 41 babies by day-by-day interval from DPT injection to death. It also compares the pattern of flareups of stressed breathing after DPT injections with the dynamics of adreno-cortical activity in animals exposed to non-specific stress syndrome, as illustrated by Selye (1978), and with the pattern of non-specific clinical symptoms in babies who succumbed to cot death and the control babies, 21 days before death and before interview, respectively [after Watson *et al.* (1981)].

Baby one was given his third DPT injection, baby two his first. Note that the flareups of stress-induced breathing follow the same pattern in both babies, even though the amplitude of the flareups was different. The day-by-day distribution of deaths of 41 babies closely follows the dynamics of the flareups of stressed breathing of baby one and baby two after the administration of DPT. In other words, the 41 deaths occurred significantly more frequently on those days when flareups of stressed breathing after DPT injections were experienced. Information on the 41 day-by-day deaths comes from papers by Bernier *et al.* (1982), Walker *et al.* (1987), and Coulter and Fisher (1991).

The lowermost graph on figure 2 provides additional data to support our conclusion that cot death is death due to exposure to the non-specific stress syndrome. The full line represents the case babies who died of cot death. The dotted line are babies who suffered the same non-specific symptoms as the case babies, 21 days before the interview, but recovered. When we draw lines across the days when there was an increase in percentages of babies displaying non-specific symptoms, we can see a perfect correlation with our data based on computer printouts of the flareups of stress-induced breathing after DPT injections. Of special importance are days 2, 5,6 and 8, 11, 13- 16 and 18-21. Day 16 quite obviously represents a point of crisis. After day 16, those babies who died, got worse (the group experienced a marked increase in percentages of babies with non-specific symptoms) when compared with those who recovered.

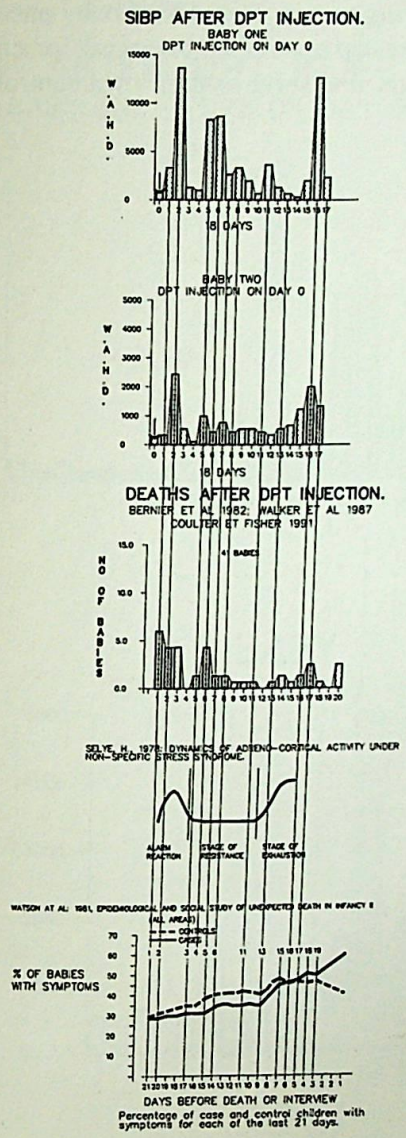
SIBP AFTER DPT INJECTION.
BABY ONE
DPT INJECTION ON DAY 0
W.A.H.D.
15000
10000
5000
0
18 DAYS
BABY TWO
DPT INJECTION ON DAY 0
W.A.H.D.
5000
4000
3000
2000
1000
0
18 DAYS
DEATHS AFTER DPT INJECTION.
BERNIER ET AL 1982; WALKER ET AL 1987
COULTER ET FISHER 1991
NO OF BABIES
15.0
10.0
5.0
0.0
4 BABIES
SELYE, H. 1976. DYNAMICS OF ADRENO-CORTICAL ACTIVITY UNDER NON-SPECIFIC STRESS SYNDROME.
ALARM REACTION
STAGE OF RESISTANCE
STAGE OF EXHAUSTION
WATSON ET AL. 1981. EPIDEMIOLOGICAL AND SOCIAL STUDY OF UNEXPECTED DEATH IN INFANCY II
(ALL AREAS)
CONTROLS
CASES
% OF BABIES WITH SYMPTOMS
70
60
50
40
30
20
10
DAYS BEFORE DEATH OR INTERVIEW
Percentage of case and control children with symptoms for each of the last 21 days.

**FIGURE 2**

**FIGURE 3.** *(Facing page).* Records of baby one and baby three show that both babies had unstressed breathing for a number of days before DPT injections, and serve as their own controls.

FIGURE 3

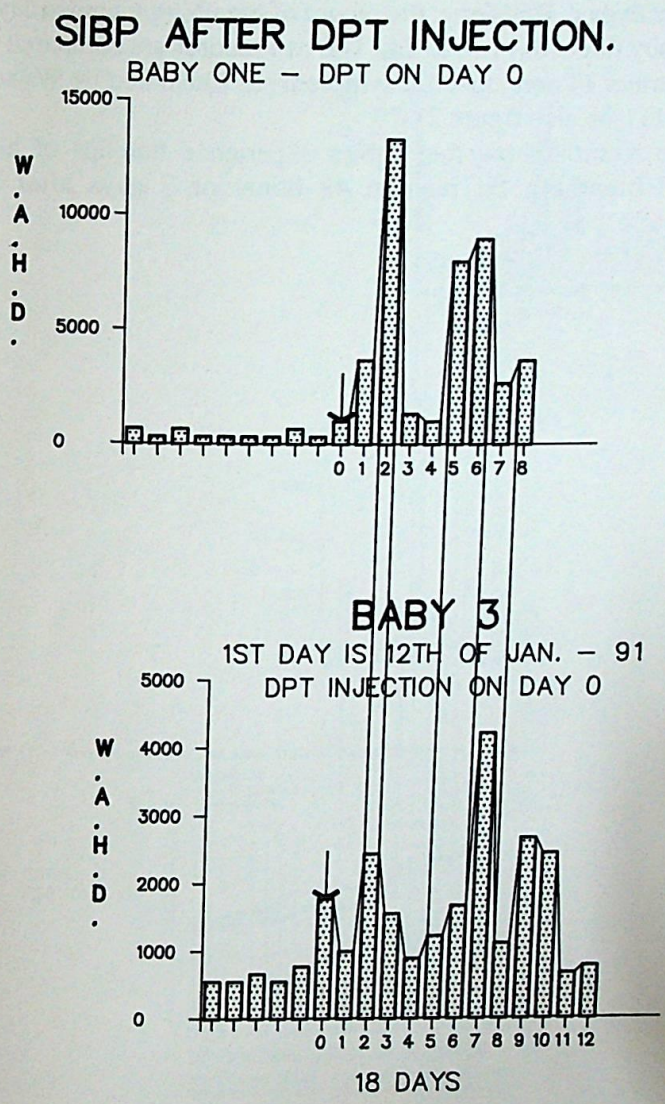


**FIGURE 3**

**FIGURE 4.** *(Facing page).* Records of breathing beyond the seventeenth day. Baby one: the record of breathing includes days 10 to 25; baby three: days 16 to 24. The two records are compared with the dynamics of non- specific symptoms as illustrated by Watson *et al.* (1981) (see also figure 2).

These records show that babies experience flareups of highly stressed breathing far beyond 48 hours or 7 days after DPT vaccination.

FIGURE 4

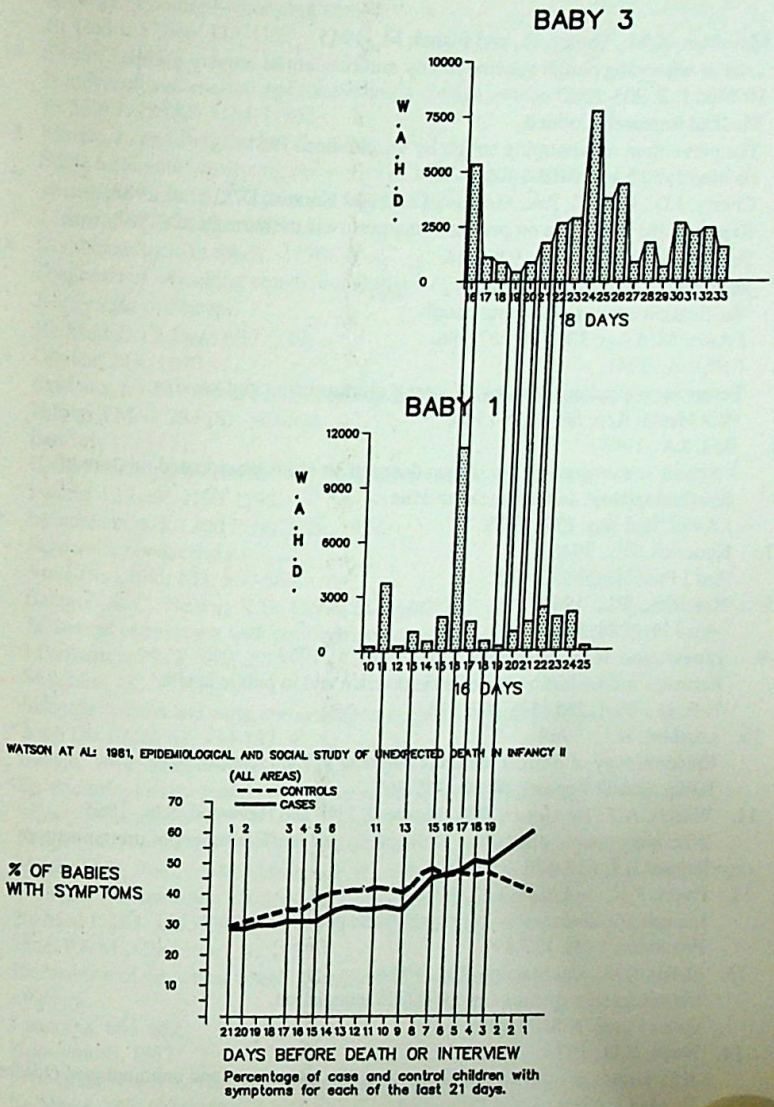


**FIGURE 4.**

## References


1. McFarlan, A.M., Topley, E., and Fisher, M., 1945.
   Trial of whooping cough vaccine in city and residential nursery groups.
   Br Med J; 2: 205-208.
2. Medical Research Council.
   The prevention of whooping cough by vaccination. 1951.
   Br Med J; 30 June: 1463-1471.
3. Cherry, J.D., Brunell, P.A., Golden,. G.S., and Karzon, D.T., *et al.* 1988.
   Report of the task force on pertussis and pertussis immunisation - 1988.
   Pediatrics - **Supplement**: 939-984.
4. Madsen, T., 1933.
   Vaccination against whooping cough.
   J Amer Med Ass; **101 (3)**: 187-188.
5. Bell, J.A., 1941.
   Pertussis prophylaxis with two doses of alum-precipitated vaccine.
   Publ Health Rep; **56**: 1535-1546.
6. Bell, J.A., 1948.
   Pertussis immunization: use of two doses of an alum-precipitated mixture of diphtheria toxoid and pertussis vaccine
   J Amer Med Ass; **137**: 1276.
7. Kendrick, P.L., 1942.
   Am J Publ Health; **32**: 615.
8. Kendrick, P.L., 1943.
   Am J Hyg; **38**: 193.
9. Provenzano, R.W., Wetterlow, L.H., Ipsen, J., 1959.
   Pertussis immunisation in pediatric practice and in public health.
   N Engl J Med; **261 (10)**: 473-478.
10. Lambert, H.L., 1965.
    Epidemiology of a small pertussis outbreak in Kent County, Michigan.
    Public Health Reports; **80 (4)**: 365-369.
11. Wilson, A.T., Henderson, I.R., Moore, E.J.H., and Heywood, S.N., 1965.
    Whooping cough: difficulties in diagnosis and ineffectiveness of immunisation.
    Br med J; 2: 623-626.
12. Byers, R.K., and Moll, F.C., 1948.
    Encephalopathies following prophylactic pertussis vaccine.
    Pediatrics; 1 (4): 437-457.
13. Miller, H.G., and Stantion, J.B., 1954.
    Neurological sequelæ of prophylactic inoculation.
    Quart J med, N.S. 23; (89): 1-27.
14. Noah, N.D., 1976.
    Attack rates of notified whooping cough in immunised and unimmunised children.
    Br Med J; 17Jan: 128-129.

15. Miller, C.L., and Fletcher, W.B., 1976.
Severity of notified whooping cough.
Br Med J; 17 Jan: 117-119.
16. Strom, J., 1960.
Is universal vaccination against pertussis always justified?
Br Med J; 22 Oct: 1184-1186.
17. Munoz, J., and Bergman, R.K., 1966.
Some histamine sensitizing properties of soluble preparations of the histamine sensitizing factor (HSF) from *Bordetella pertussis*.
J Immunol; **97 (1)**: 120-125.
18. Combined Scottish Study. 1970.
Diagnosis of whooping cough: comparison of serological tests with isolation of *Bordetella pertussis*.
Br Med J: 12 Dec: 637-639.
19. Connor, J.D., 1970.
Evidence for an etiologic role of adenovirus infection in pertussis syndrome.
N Engl J Med; **283 (8)**: 390-394.
20. Lautrop, H., 1971.
Epidemics of parapertussis. 20 years' observations in Denmark.
Lancet, 12 June: 1195-1198.
21. Linnemann, C.C., and Perry, E.B., 1977.
*Bordetella parapertussis*.
Am J Dis Child; **131**: 560-563.
22. Balagtas, R.C., Nelson, K.E., Levin, S., and Gotoff, S.P., 1971.
Treatment of pertussis with pertussis immune globulin.
J Pediatrics; **79 (2)**: 203-208.
23. Melchior, J.C.,1977.
Infantile spasms and early immunization against whooping cough.
Arch Dis Child; **52**: 134-137.
24. Bassili, W.R., and Stewart, G.T., 1976.
Epidemiological evaluation of immunisation and other factors in the control of whooping cough.
Lancet; 28 Febr: 471-474.
25. Miller, D.L., Ross, E.M., Aldserslade, R., Bellman, M.H., and Rawson, N.S.B., 1981.
Pertussis immunisation and serious neurological illness in children.
Br Med J; **282**: 1595-1599.
26. Fine, P.E.M., Clarkson, J.A., 1982.
Recurrence of whooping cough: possible implications for assessment of vaccine efficacy.
Lancet; **1**: 666-669.
27. Anonymous, 1982.
Whooping cough surveillance (PHLS Comunicable Disease Surveillance Centre).
Br Med J, **285**: 1583-1584.

## References


1. McFarlan, A.M., Topley, E., and Fisher, M., 1945.
   Trial of whooping cough vaccine in city and residential nursery groups.
   Br Med J; 2: 205-208.
2. Medical Research Council.
   The prevention of whooping cough by vaccination. 1951.
   Br Med J; 30 June: 1463-1471.
3. Cherry, J.D., Brunell, P.A., Golden,. G.S., and Karzon, D.T., *et al.* 1988.
   Report of the task force on pertussis and pertussis immunisation - 1988.
   Pediatrics - **Supplement**: 939-984.
4. Madsen, T., 1933.
   Vaccination against whooping cough.
   J Amer Med Ass; **101 (3)**: 187-188.
5. Bell, J.A., 1941.
   Pertussis prophylaxis with two doses of alum-precipitated vaccine.
   Publ Health Rep; **56**: 1535-1546.
6. Bell, J.A., 1948.
   Pertussis immunization: use of two doses of an alum-precipitated mixture of diphtheria toxoid and pertussis vaccine
   J Amer Med Ass; **137**: 1276.
7. Kendrick, P.L., 1942.
   Am J Publ Health; **32**: 615.
8. Kendrick, P.L., 1943.
   Am J Hyg; **38**: 193.
9. Provenzano, R.W., Wetterlow, L.H., Ipsen, J., 1959.
   Pertussis immunisation in pediatric practice and in public health.
   N Engl J Med; **261 (10)**: 473-478.
10. Lambert, H.L., 1965.
    Epidemiology of a small pertussis outbreak in Kent County, Michigan.
    Public Health Reports; **80 (4)**: 365-369.
11. Wilson, A.T., Henderson, I.R., Moore, E.J.H., and Heywood, S.N., 1965.
    Whooping cough: difficulties in diagnosis and ineffectiveness of immunisation.
    Br med J; **2**: 623-626.
12. Byers, R.K., and Moll, F.C., 1948.
    Encephalopathies following prophylactic pertussis vaccine.
    Pediatrics; **1 (4)**: 437-457.
13. Miller, H.G., and Stantion, J.B., 1954.
    Neurological sequelæ of prophylactic inoculation.
    Quart J med, N.S. 23; (89): 1-27.
14. Noah, N.D., 1976.
    Attack rates of notified whooping cough in immunised and unimmunised children.
    Br Med J; 17Jan: 128-129.

15. Miller, C.L., and Fletcher, W.B., 1976.
Severity of notified whooping cough.
Br Med J; 17 Jan: 117-119.
16. Strom, J., 1960.
Is universal vaccination against pertussis always justified?
Br Med J; 22 Oct: 1184-1186.
17. Munoz, J., and Bergman, R.K., 1966.
Some histamine sensitizing properties of soluble preparations of the histamine sensitizing factor (HSF) from *Bordetella pertussis*.
J Immunol; **97 (1)**: 120-125.
18. Combined Scottish Study. 1970.
Diagnosis of whooping cough: comparison of serological tests with isolation of *Bordetella pertussis*.
Br Med J: 12 Dec: 637-639.
19. Connor, J.D., 1970.
Evidence for an etiologic role of adenovirus infection in pertussis syndrome.
N Engl J Med; **283 (8)**: 390-394.
20. Lautrop, H., 1971.
Epidemics of parapertussis. 20 years' observations in Denmark.
Lancet, 12 June: 1195-1198.
21. Linnemann, C.C., and Perry, E.B., 1977.
*Bordetella parapertussis*.
Am J Dis Child; **131**: 560-563.
22. Balagtas, R.C., Nelson, K.E., Levin, S., and Gotoff, S.P., 1971.
Treatment of pertussis with pertussis immune globulin.
J Pediatrics; **79 (2)**: 203-208.
23. Melchior, J.C.,1977.
Infantile spasms and early immunization against whooping cough.
Arch Dis Child; **52**: 134-137.
24. Bassili, W.R., and Stewart, G.T., 1976.
Epidemiological evaluation of immunisation and other factors in the control of whooping cough.
Lancet; 28 Febr: 471-474.
25. Miller, D.L., Ross, E.M., Aldserslade, R., Bellman, M.H., and Rawson, N.S.B., 1981.
Pertussis immunisation and serious neurological illness in children.
Br Med J; **282**: 1595-1599.
26. Fine, P.E.M., Clarkson, J.A., 1982.
Recurrence of whooping cough: possible implications for assessment of vaccine efficacy.
Lancet; **1**: 666-669.
27. Anonymous, 1982.
Whooping cough surveillance (PHLS Comunicable Disease Surveillance Centre).
Br Med J, **285**: 1583-1584.

28. Ehrengut, W., 1978.
Whooping cough vaccination. Comment on Report from Joint Committee on Vacination and Immunisation.
Lancet; 18 Feb: 370-371.
29. Ditchburn,. R.K., 1979.
Whooping cough after stopping pertussis immunisation.
Br Med J; 1: 1601-1603.
30. MacGregor, J.D., 1979.
Whooping-cough vaccination: a recent Shetland experience.
Br Med J: 1: 1154.
31. Stewart, G.T., 1979.
Whooping cough in Shetland.
Br Med J; 19 May: 1352.
32. Stewart, G.T., 1980.
Whooping cough in the United Kingdom 1977-8.
Br Med J; 9 Aug: 451-452.
33. Stewart, G.T., 1977.
Vaccination against whooping-cough. Efficacy versus risks.
Lancet; 29 Jan: 234-237.
34. Stewart, G.T., 1984.
Re: "Whooping cough and whooping cough vaccine: the risks and benefits debate".
Am J Epidemiology; 119(1): 135-139.
35. Bennett, N. McK., 1973.
Whooping cough in Melbourne.
Med J Aust; 8 Sept: 481-487.
36. Goldacre, M.J., and Harris, I., 1981.
Hospital admissions for whooping cough in the Oxford Region, 1974-1979.
Br Med J: 282: 106-107.
37. Anonymous, 1982.
Efficacy of pertussis vaccination in England. Report from the PHLS Epidemiological Research Laboratory and 21 area health authorities.
Br Med J; 285: 357- 359.
38. Trollfors, B., and Rabo, E., 1981.
Whooping cough in adults.
Br Med J; 283: 696-697.
39. Taranger, J., 1982.
Mild clinical course of pertussis in Swedish infants today.
Lancet; 12 June: 1360.
40. Romanus, V., Jonsell, R., and Bergquist, S.O., 1987.
Pertussis in Sweden after the cessation of general immunisation in 1979.
Ped Inf Dis J; 6: 364-371.
41. Trollfors, B., 1984.
*Bordetella pertussis* whole cell vaccines. Efficacy and toxicity.
Acta Pædiatr Scand: 73: 417- 425.

42. Storsaeter, J., Olin, P., Renemar, B., Lagergard, T., Norberg, R., Romanus, V., and Tiru, M., 1988.
Mortality and morbidity from invasive bacterial infections during a clinical trial of acellular pertussis vaccines in Sweden.
Ped Inf Dis J;**7 (9)**: 637-645.

43. Michaels, R.H., 1971.
Increase in influenzal meningitis.
N Engl J Med; **285 (12)**: 666-667.

44. Cartwright, K.A.V., Stuart, J.M., and Noah, N.D., 1986.
An outbreak of meningococcal disease in Gloucestershire.
Lancet; **2**: 558-561.

45. Smith, E.W.S., and Haynes, R.E., 1972.
Changing incidence of *Hæmophilus influenzæ* meningitis.
Pediatrics; **50 (5)**: 723- 727.

46. Anonymous, 1989.
Licence application for pertussis vaccine withdrawn in Sweden.
Lancet; 14 Jan: 114.

47. Craighead, J.E., 1975.
Report of a workshop: disease accentuation after immunisation with inactivated microbial vaccines.
J Infect Dis; **1312 (6)**: 749-754.

48. Kimura, M., and Kuno-Sakai, H., 1988.
Acellular pertussis vaccines and fatal infections.
Lancet; 16 April: 881-882.

49. Boulton-Jones, J.M., Sissons, J.G.P., Naish, P.F., Evans, D.J., and Peters, D.K., 1974.
Self-induced Glomerulonephritis.
Br Med J; 10 Aug: 387-390.

50. Bishop, W.B., Carlton, R.F., and Sanders, L.L. et al. 1966.
Diffuse vasculitis and death after hyperimmunisation with pertussis vaccine.
N Engl J Med; **274**: 616.

51. Amsel, S.G., Hanucoglu, A., Fried, D., Wolyvovics, M., 1986.
Myocarditis after triple immunisations.
Arch Dis Child; **61**: 403-405.

52. Karlsson, L., and Scheibnerova, V., 1991.
Association between non-specific stress syndrome, DPT injections and cot death.
Pre-print of a paper delivered to the 2nd Immunisation Conference,
Canberra, Australia, 27-29 May 1991.

53. Scheibner, V., 1992.
Evidence of the association between non-specific stress syndrome, DPT injections and cot deaths.
Proc 2nd National Immunisation Conference,
Canberra, Australia, 27-29 May 1991: 90-91.

54. Davidson, M., Letson, G.W., Ward, J.I., Ball, A., Bulkow, L., Christensen, P., and Cherry, J.D., 1991.
DPT immunisation and susceptibility to infectious diseases.
Am J Dis Child; 145: 750-754.
55. Black, S.B., Cherry, J.D., Shinefield, H.R., Fireman, B., Christensen, P., and Lampen, D., 1991.
Apparent decreased risk of invasive bacterial disease after heterologous childhood immunisation.
Am J Dis Child; 145: 746-754.
56. Griffin, M., Taylor, J.A., 1992.
No increased risk for invasive bacterial infections found following diphtheria-tetanus-pertussis immunisation.
Pediatrics; 89 (4): 640-642.
57. American Academy of Pediatrics, 1992.
Acellular pertussis vaccines: recommendations for use as the fourth and fifth dose.
Pediatrics; 90 (1): 121-123.
58. Kanai, K., 1980.
Japan's experience in pertussis epidemiology and vaccination in the past 30 years.
Jpn J Med Sci Biol; 33: 107-143.
59. Sato, Y., Kimura, M., and Fukumi, H., 1984.
Development of a pertussis component vaccine in Japan.
Lancet; 1: 122-126.
60. Noble, G.R., Bernier, R.H., Esber, E.C., *et al.*, 1987.
Acellular and whole-cell pertussis vaccines in Japan: report of a visit by US scientists.
J Amer Med Ass; 257: 1351-1356.
61. Aoyama, T., Hagiwara, S., Murase, Y., Kato, T., Iwata, T., 1986.
Adverse reactions and an antibody response to acellular pertussis vaccine.
J Pediatr; 109: 925-930.
62. Aoyama, T., Murase, Y., Gonda, T., and Iwata, T., 1988.
Type-specific efficacy of acellular pertussis vacine.
Am J Dis Child; 142: 40-42.
63. Kimura, M., and Kuno-Sakai, H., 1988.
Acellular pertussis vaccines and fatal infections.
Lancet, 16 April: 881-882.
64. Hoskins, T.W., Cochi, S.L., Brink, E.W., Patriarca, P.A., Wassilak, S.G.F., Bovina, P.A., and Hinman, A.R., 1988.
Current epidemiology of pertussis in the United States.
Tohoku J Exp Clin Med; 13(Suppl): 103-109.
65. Pichichero, M.E., Francis, A.B., Blatter, M.M., Reisinger, K.S., Green, J.L., Marsocci, S.M., Disney, F.A., 1992.
Acellular pertussis vaccination of 2-month-old infants in the United States.
Pediatrics; 89 (5): 882-887.

65. Selye, H., 1978.
The Stress of Life.
McGraw-Hill Publ. Co.
67. National Vaccine Information Center (NVIC) Newsletter 1992.
68. Etkind, P., Lett, S.M., MacDonald, P.D., Silva, E., and Peppe, J., 1992.
Pertussis outbreak in groups claiming religious exemptions to vaccination.
Am J Dis Child; **146**: 173- 176.
69. Halperin, S.A., Bortolusi, R., MacLean, D., and Chisholm, N., 1989.
Persistence of pertussis in an immunised population:
results of the Nova Scotia enhanced pertussis surveillance program.
J Pediatrics; **1125**: 686-693.
70. Sutter, R.W., and Cochi, S.L., 1992.
Pertussis hositalizations and mortality in the United States, 1985-1988.
J Amer Med Ass; **267** (3): 386-390.
71. Pollock, T.M., Miller, E., Mortimer, J.Y., Smith, G., 1984.
Symptoms after primary immunisation with DPT and DT vaccine.
Lancet; 21 July: 146-149.
72. Cody, C.L., Baraff, L.J., Cherry, J.D., Marcy, S.M., Manclark, C.R., 1981.
Nature and rates of adverse reactions associated with DPT and DT immunizatio
infants and children.
Pediatrics; **5**: 650.
73. Anderson, I.M. and Morris, D., 1950.
Encephalopathy after combined diphtheria-pertussis inoculation.
Lancet; 25 March: 537-539
74. Low, N.L., 1955.
Electroencephaolgraphic studies following pertussis immunizations.
J Pediatrics; **47**: 35-39.
75. Baird, H.W., and Borofsky, L.G. 1957.
Infantile myoclonic seizures.
J.Pediatrics; **50**: 332-339.
76. Kulenkampff, M., Schwartzman, J.S., and Wilson, J., 1974.
Neurological complications of pertussis inoculation.
Arch Dis Child; **49**: 46-49.
77. Connor, J.D., 1970.
Evidence for an ætiological role of adenoviral infection in pertussis syndrome.
N Engl J Med; 20 Aug; 390-394.
78. Pollock, T.M., Miller, E., and Lobb, J., 1984.
Severity of whooping cough in England before and after the decline in pertussis
immunisation.
Arch Dis Child; **59**: 162-165.
79. Howson, C.P., and Fineberg, H.V., 1992
The ricochet of magic bullets: Summary of the Institute of Medicine Report,
*Adverse effects of pertussis and rubella vaccines*.
Pediatrics; **89(2)**:318-324

80. Cherry, J.D., 1990
'Pertussis vaccine encephalopathy': it is time to recognise it as the myth that it is.
J Amer Med Ass; **263(12)**:1679-1680.

## Animal Experiments And Pertussis Vaccine

1. Levine, S., Wenk, E.J., Devlin, H.B., Pieroni, R.E., and Levine, L., 1966.
Hyperacute allergic encephalomyelitis: adjuvant effect of pertussis vaccine and extracts.
J Immunol; **97 (3)**: 363-368.
2. Levine, S., and Sowinski, R., 1973.
Hyperacute allergic encephalomyelitis.
Am J Pathol; **73 (1)**: 247-258.
3. Munoz, J.J., Arai, H., Bwergman, R.K., and Sadowski, P.I., 1981.
Biological activities of crystalline pertussigen from *Bordetella pertussis*.
Infec and Immunity; **33 (3)**: 820-826.
4. Hewlett, E.L., Roberts, C.O., Wolff, J., and Manclark, C.R., 1983.
Biphasic effect of pertussis vaccine on serum insulin of mice.
Infecand Immun; **41 (1)**: 137-144.
5. Hannik, C.A., and Cohen, H., 1978.
Changes in plasma insulin concentration and temperature of infants after pertussis vaccination: 297-299.
In C.R. Manclark and J.C. Hill (ed.). International Symposium on Pertussis.
U.S. Dept of Health, Education and Welfare, Washington, D.C.
6. Iwasa, ., Ishida, S., Akama, K., 1985.
Swelling of the brain in mice caused by pertussis vaccine — its quantitative determination and the responsible factors in the vaccine.
Japan J Med Sci Biol; **38**: 53-65.

## DPT-Cotdeath Link References

1. Madsen, T., 1933.
Vaccination against whooping cough.
J Amer Med Ass; **101(3)**: 187-188.
2. Werne, J., & Garrow, I., 1946.
Fatal anaphylactic shock occurrence in identical twins following second injection of diphtheria toxoid and pertussis antigen.
J Amer Med Ass; **131(9)**: 730-735.
3. Bernier, R.H., Frank, J.A., Dondero, T.J., and Nolan, T.F., 1982.
Diphtheria-tetanus toxoids-pertussis vaccination and sudden infant deaths in Tennessee.
J Pediatrics; **101(5)**: 419-421.

4. Torch, W.C., 1982.
Diphtheria-pertussis-tetanus (DPT) immunization:
a potential cause of the sudden infant death syndrome (SIDS).
Neurology; **32(4)**: A169 (abstract).
5. Coulter, H.L., & Fisher, B.L., 1991.
A shot in the dark.
Avery Publishing Group Inc. N.Y.: 246 pp.
6. Walker, A.M., Jick, H., Perera, D.R., Thompson, R.S., and Knauss, T.A., 1987.
Diphtheria-tetanus-pertussis immunization and sudden infant death syndrome.
Am J Pub Health; **77**: 945- 951.
7. Hoffman, H.J., Hunter, J.C., and Hasselmeyer, E.G., 1982.
SIDS and DPT.
Seventeenth Immunization Conference Proceedings, CDC (May) 79-88.
Washington DC.
8. Griffin, M.R., Wayne, A.R., Livengood, J.R., and Schaffner, W., 1988.
Risk of sudden infant death syndrome after immunization with the
diphtheria-tetanus-pertussis vaccine.
New Engl J Med; **319(10)**: 618-623.
9. Stewart, G.T., 1979.
Death of infants after triple vaccine.
Lancet; 18 August: 354-355.
10. Geraghty, K.C., 1984.
DPT immunization and SIDS.
J Pediatrics; **105(1)**: 169-170.
11. Baraff, L., Ablon, W., and Weiss, P., 1983.
Possible temporal association between diphtheria-tetanus toxoid-pertussis
vaccination in sudden infant death syndrome.
Pediatr Infect Dis; 2:7.
12. Karlsson L.G., & Scheibnerova, V. 1991.
Evidence of the association between non-specific stress syndrome, DPT injections
and cot death. Pre-print of a talk delivered to the 2nd National Immunisation
Conference in Canberra, May 1991: 1-9.
13. Scheibner, V., 1991.
Evidence of the association between non- specific stress syndrome, DPT injections
and cot death.
Proceedings of the 2nd National Immunisation Conference, Canberra May 1991:
90-91.
14. Cherry, J.D., Brunell, P.A., Golden, G.S., and Karzon, D.T., 1988.
Report of the task force on pertussis and pertussis immunization.
Pediatrics (**Suppl**): 939-984.
15. Pollock, T.M., Miller, E., Mortimer, J.Y., and Smith, G., 1984.
Symptoms after primary immunisation with DPT and DT vaccine.
Lancet; 21 July: 146-149.

# 3

# MEASLES — Visitation by A Goddess

According to Langmuir (1962), measles is a *"self-limiting infection of short duration, moderate severity, and low fatality, which has maintained a remarkably stable biological balance over centuries"*. People in India consider measles the visitation of a goddess.

Measles is a truly universal disease present on all continents. The period from exposure to the first symptoms (incubation) is 10 to 11 days. It may be shortened if the virus is introduced intravenously or subcutaneously. The symptomatic stage of measles has two distinct periods. The first is the prodromal period, lasting 2 to 4 days, and characterised by fever, feeling of general malaise, rhinitis, conjunctivitis and tracheobronchitis. Yellowish spots appear in the mouth and soft palate (Koplik spots). During this stage, the measles virus is present in the nasopharyngeal secretions, in the tissues, internal organs and the lymphoid tissues.

The characteristic feature of measles infection is the development of multinucleate giant cells. There are two different types of these giant cells: the epithelial giant cells, which appear in the respiratory epithelium, and the reticuloendothelial giant cells, which occur in the lymphatic tissues, including the appendix, tonsils, lymph nodes, thymus, Peyer's patches and the spleen for up to five days before appearance of the rash.

In the second stage of measles, the characteristic rash typically occurs on or about the fourteenth day after exposure. It first appears on the face, neck and upper trunk. Over a period of about three days it spreads to the lower trunk and extremities. Rash then begins to fade and disappears after becoming brownish. As the rash appears,

the systemic symptoms persist and may even become worse for a short period of time.

The improvement starts within 2 to 3 days. The temperature falls and recovery is quite rapid. Koplik's spots may still be present during the first or second days of rash, but disappear after this time. The giant cells begin to degenerate and disappear shortly after the rash becomes evident. Also, as the rash appears, so do the antibodies in the blood. The duration of the acute phase is about 7 days.

## Measles Herd Immunity?

Measles is a prevalent disease, and practically all children get it before they reach puberty. It also has a characteristic epidemiology which was a subject of interest to A.W. Hedrich who, in 1933, published a study on the epidemiological patterns of measles in Baltimore from 1900 to 1931. He concluded that when 68% of the children less than 15 years of age were immune to measles, epidemics did not occur. This is the basis of the concept of herd immunity.

Cherry (1980) wrote that "*... today we would regard that proportion of immunes to susceptibles as too low, but we still retain the basic concept — that there exists a threshhold of herd immunity that will prevent epidemics.*"

We know that today, in the US, with 98% immunisation status due to enforced vaccination, epidemics of measles still occur at three to four year intervals, unabated and uninfluenced by vaccination.

Epidemiologists have a hard time explaining this recurrence, yet it is really quite easy to see why. First of all, Hedrich (1933) talked about natural immunity, achieved by contracting measles. The fact that, despite 98% vaccination compliance, epidemics of measles still occur means that vaccination against measles is totally ineffective.

Measles occurs irrespective of and despite vaccination. It is governed by the same rules of natural immunity (there is no other true immmunity) which is achieved only by contracting measles, as in Hedrich's time. The major difference between then and now is that, due to vaccination, we now have **atypical measles**, an especially vicious form of measles resisting treatment, and the so-called "mild

measles" with under-developed rash, which exposes children in later life to dangers of chronic diseases, including cancer.

A large group of Swiss doctors formed a working committee questioning the Swiss Health Department's policy of mass vaccination against MMR (measles, mumps and rubella). They wrote in their proclamation of 1989 that up to 1969, at the Basel University Pædiatric Clinic, artificial infection with the measles was used to treat nephrotic syndrome.

Many practitioners know that cancer patients have a particularly small number of infectious diseases of childhood to report in their medical history. Ronne (1985) found evidence of a relationship between lack of rash in measles and increased incidence of degenerative and autoimmune diseases. It is also well-known that measles is an important developmental milestone in the life and maturing processes in children. Why would anybody want to stop or delay the maturation processes of children and of their immune systems?

## Measles Vaccines — Is There a Need for Them? Are They Effective and Safe

Miller wrote in 1964 that one of the major sources of doubt about the need for vaccination is that measles is a mild disease with rare serious complications and negligible fatality in normal children. About half the recorded deaths occur in persons with serious chronic disease or disability. Nevertheless the development of measles vaccines went ahead. By 1965, several vaccines had been introduced for the prevention of measles. They included

1. Live attenuated measles virus vaccine,
2. Further attenuated live vaccine, and
3. Formalin inactivated measles virus vaccine.

## Atypical Measles

Soon after measles vaccine was first administered, a new and serious problem arose: vaccinated children were contracting what became known in the medical literature as atypical measles.

Rauh and Schmidt (1965) described 9 cases which occurred in 1963 in Cincinnati during an epidemic of measles. The authors followed 386 children who had received three doses of killed measles virus vaccine in 1961. Of these 386, 125 had been exposed to measles and 54 of them developed the disease. *"It is obvious that three injections of killed vaccine had not protected a large percentage of children against measles when exposed within a period of two-and-a-half years after immunization ..."* wrote the authors. Many of these children were so ill with high fever and pneumonia that they had to be hospitalised.

Fulginiti *et al.* (1967) described the occurrence of atypical measles in ten children who had received inactivated (killed) measles virus vaccine five to six years earlier. Petechial rash appeared first on feet and palms and then moved towards the body. Nine children developed pneumonia which resisted all treatment. Serious reactions also occurred in children originally injected with killed measles virus, and then re-vaccinated with live measles virus [Scott and Bonanno, (1967)]. Further authors not only described more cases of atypical measles occurring in vaccinated children, but also outbreaks of measles in fully vaccinated populations.

Despite these worrying occurrences, other authors published articles on the benefits due to measles vaccinations [Axnick *et al.* (1968)]. Against all the evidence, measles vaccines continued to be described as effective and safe by some, yet at the same time, the medical literature was teeming with reports of the ineffectiveness of measles vaccines and of serious local and systemic reactions to them [Buser (1967); Lennon (1967); Nader *et al.* (1968)].

Barratta *et al.* (1970) investigated an outbreak of measles in Florida from December 1968 to February 1969 and found there was little difference in the incidence of measles in vaccinated and unvaccinated children. However, while 43% of unimmunised children developed rash, only 12% of those vaccinated developed proper rash.

In 1971, Conrad *et al.* published an article in the American Journal of Public Health in which they analysed what had actually happened

in the past 4 years. They conceded that measles was on the increase and that *"eradication, if possible, now seems far in the future"*. Despite their own findings, they still believed that it was not the vaccine failure, but a failure to vaccinate more than 72% of all children; they recommended intensifying the vaccination programme.

Reports of vaccine failure and atypical measles in vaccinated children continued. Improper storage of vaccines and too early age at vaccination were blamed for this [Plotkin (1973): Editorial].

Cherry *et al.* (1972) described an epidemic of measles in St Louis City and County during 1970 and 1971, during which 130 children were hospitalised and six died. The attack rate was much higher in vaccinated than unvaccinated children. A number of vaccinated children developed atypical measles. In this case, vaccine failure was admitted as the major contributor to this epidemic. Cherry *et al.* (1973) studied 103 children with measles vaccine failure. Seventy six had clinically typical measles, 15 had mild modified measles and 12 had an illness resembling the atypical measles syndrome.

Linnemann *et al.* (1973) demonstrated that measles vaccines were not provoking a proper immunologic response in vaccinated children. Measles IgM antibodies were found in the convalescent sera from five of seven children with a primary measles infection, but in only one of seven previously vaccinated children with clinical measles, and in none of seven previously vaccinated children who were injected with the attenuated measles virus vaccine.

Reports of vaccine damage continued to be published. Landrigan and Witte (1973) described some 80 cases of neurologic disorders, starting within 30 days after measles virus vaccination. Because 45 of these children experienced the onset of neurological disorders between 6 and 15 days after measles vaccination, the authors suggested that this clustering indicated a causal link between vaccine administration and the observed adverse effects. Indeed, their figure 1 shows a characteristic clustering of all adverse reactions around the critical days in the same pattern as they were recorded with the microprocessor breathing monitor in babies after DPT and poli

vaccination [Karlsson and Scheibnerova (1991)]. Landrigan and Witte (1973) and Cheng (1973) also reported cases of subacute sclerosing panencephalitis (SSPE) after measles virus vaccination.

Roden (1974) reported the incidence of 19/10,000 cases of convulsions after measles vaccine injections and warned that estimates of vaccine-associated convulsions vary with the source of information.

Meanwhile the push for measles vaccination continued; the authors were for the most part oblivious to the clinical facts of ineffectiveness and the dangers of measles vaccines. Witte and Axnick (1975) claimed humanistic and economic benefits resulting from alleged prevention of infectious diseases by vaccination. They blamed the increasing incidence of measles in the seventies on the diminishing vaccination efforts. They estimated that vaccination would result in some $1.3 billion in economic savings and considered the benefits derived from immunisation as impressive. However, they did not explain why measles epidemics continued to occur consistently in fully vaccinated children.

Since the introduction of measles vaccine, reports of measles outbreaks in adults have appeared in medical journals. Rand *et al.*(1975) wrote that not only was the number of reported cases of measles six times higher in the first half of 1975 compared with 1974, but more and more adults were contracting measles. So, again, while some were claiming victory, others were bringing evidence to the contrary.

Not even booster vaccination of previously vaccinated children made any difference although some writers, against all the evidence to the contrary, were still claiming that vaccines were effective in preventing measles. While measles vaccines were effective in elevating measles-neutralising antibody in a number of children (although not in all), this had already demonstrated to be irrelevant in preventing the disease [Bellanti *et al.* (1971)]. Quite without any evidence to support his statement, Cherry (1980) claimed that after more than 16 years of routine measles immunisation *"we have done well in controlling ... measles"*.

In October 1978 the Secretary of the Department of Health, Education and Welfare, Joseph A. Califano Jnr, announced *"We are launching an effort that seeks to free the United States from measles by 1st of October 1981"* [Hinman (1978)].

Reuman (1979) warned about an increasing number of adolescents contracting measles. While in the pre-vaccine era 90% of all measles patients were 5-9 years old, in the post-vaccine era 55% to 64% were older than 10 years. The average age of patients during the measles outbreak in the UCLA was 20-24 years and 91% of the students were found to have measles-specific antibodies. According to Reuman (1979), the history of natural measles, or measles vaccination, correlated poorly with what was generally considered serologic evidence of immunity. Re-vaccination of these young adults was associated with high rates of major side effects, with about 17% reporting significant fever, eye pain and the need for bed rest. Improper storage of measles vaccines and only about 67% rate of compliance were blamed for this failure, instead of an acknowledgement of the more plausible and real reason: the inability of the measles vaccines to prevent measles.

Predictably, the programme to eradicate measles in the US by 1st October 1981 fell flat on its face. After 1981, instead of achieving eradication of measles, the US was hit repeatedly by major epidemics of measles, mostly in fully vaccinated communities. Atypical measles persisted as a *"continuing problem"* [Nichols (1979)]. The age of those contracting measles continued to climb well above 10 years and was associated with serious illnesses. Adults, and babies below the age of 2 years, often only a few months old, were now contracting measles.

One of the quoted reasons for these outbreaks was use of the *"ineffective formalin-inactivated ('killed') measles vaccine, which was administered to 600,000 to 900,000 individuals from 1963 to 1967"* [Morbidity Mortality Weekly Reports (MMWR) 4 October 1984].

Another MMWR report (June 1984) reported on a measles outbreak among high school students, all of whom were vaccinated

on or after the age of one, in accordance with Illinois Law. The outbreak subsided spontaneously. Another outbreak of measles occurred in junior high schools in Hobbs, New Mexico, where 98% of students were vaccinated against measles shortly before the outbreak began (MMWR 1 February 1985).

In the measles epidemic of 1984-85 in Auckland, New Zealand, [Hardy *et al.* (1986)], 34% of all measles cases were vaccinated, 9% were unsure and 67% were unvaccinated. However, the largest number of cases were in children one year old or less, that is, below the age at which they would be vaccinated. In the rest of the cases, the incidence in the vaccinated and unvaccinated was the same.

Gustafson *et al.* (1987) described an outbreak of measles in a secondary school population in which more than 99% had records of vaccination with live measles vaccine. MMWR (2 September 1988) dealt with 76 measles outbreaks in the United States. Most of the cases were primary vaccine failures.

These examples demonstrate that, while measles vaccination was compulsory, it was done with vaccines which had always been found to be ineffective during outbreaks of measles.

During some outbreaks, re-vaccination with the same vaccines was recommended, and often enforced, even while papers were being published demonstrating that re-vaccination was ineffective. Worse still, despite published facts about obvious examples of vaccine failure, Frank *et al.*(1985) published a paper in which they claimed that "*Great success has been achieved in controlling measles in the United States with a greater than 90% reduction in incidence rate from the prevaccine era.*" They quoted an incidence of 1,500 to 3,000 reported cases per year and stated that "*The major reason for failure to achieve elimination appears to be the fact that some persons for whom vaccine is indicated have not been vaccinated ...While vaccine failures and importations play a role in transmission, sustained transmission in a totally vaccinated community has not been demonstrated.*"

Black *et al.* (1984) summarised data on the problem of ineffectiveness of re-vaccination published by several authors, who

demonstrated that antibody titre in re-immunised children may fall after several months to very low levels, and that children vaccinated twice may still experience clinically recognisable measles, although in a much milder form. Black *et al.* (1984) concluded that *"This state in which a child is immunologically sensitized, but not immune to infection, we shall call* **'inadequate immunity'**."

This observation highlighted another looming problem, namely, that generations of children with this *"inadequate immunity"* would grow into adults with no placental immunity to pass on to their children, who would then contract measles at an age when babies are normally protected by maternal antibody.

This was indeed confirmed by another study [Lennon and Black (1986)] which demonstrated that *"hæmagglutinin-inhibiting and neutralizing antibody titers are lower in women young enough to have been immunised by vaccination than in older women"*.

Perhaps the most unfortunate thing about the idea of eliminating infectious diseases by vaccination is that indeed there is no need to do so. As pointed out by the group of Swiss doctors opposing the US-inspired policy of mass vaccination against measles, mumps and rubella in Switzerland, *"We have lost the common sense and the wisdom that used to prevail in the approach to childhood diseases. Too often, instead of reinforcing the organism's defences, fever and symptoms are relentlessly suppressed. This is not always without consequences ..."*.

They quoted measles as an example of a childhood disease with fever and eruptions affecting the organism as a whole. When the process of general inflammation is not correctly handled, the illness may subsequently affect the ears (otitis), the lungs (pneumonia) or the central nervous system, giving rise to the feared complication: encephalitis. They also pointed out the benefits and cure potential of childhood infectious diseases.

An important message from history which, unfortunately, has not lost its basis in medical practice but has lost its impact, was published by Hillary Koprowski (1962). This author wrote that a very clear statement was made in 1712 of how not to treat measles.

In a letter to the Duchess Sofie, mother of the future George I of England, Princess Elizabeth Charlotte (Liselotte) von Pfalz, Duchess of Orleans and widow of the younger brother of Louis XIV, wrote:

*"Our misfortune continues. The doctors have made the same mistake treating the little Dauphin as they did ministering to his mother, the Dauphiness. When the child was quite red from the rash and perspired profusely, they (the doctors) performed phlebotomy and administered strong emetics; the child died during these operations. Everybody knows that the doctors caused the death of the Dauphin, since his little brother who had the same sickness was hidden away from the 9 physicians who were busy with his older brother, by the young maids, who have given him a little wine with biscuits.*

*Yesterday, while the child had high fever, they wanted also to perform phlebotomy but his two governesses were firmly opposed to the idea and instead kept the child warm. This one also would have certainly died if the doctors had had their way. I do not understand why they don't learn by experience. Had they no heart, when they saw the Dauphiness die after phlebotomy and emetics, not to dispose of her child?"*

Koprowski summarised the historical message *"Avoid physicians and thou wilt be cured"*. However, again, it is quite obvious that the historical message has not sunk in as far as the treatment of measles and vaccination practices are concerned. The relentless suppression of fever in children with measles is still widely practised.

With the advent of mass vaccination programmes, little attention was paid to effective treatment of measles. However, an important paper was published by Barclay *et al.* (1987) about vitamin A supplements and mortality related to measles. In the Mvumi Hospital in central Tanzania, 180 children admitted with measles were randomly allocated to receive routine treatment alone or with additional large doses of vitamin A (200, 000 IU orally immediately on admission and again the next day). Of the 88 children given vitamin A, 6 died; of the 92 controls (children who were not given vitamin A supplement) 12 died. This difference in mortality was most

obvious for children aged under 2 years. In this age group, one child out of 46 receiving vitamin A died while in the control group 7 out of 42 died. The authors maintained that vitamin A is essential for proper performance of epithelial tissues.

When vitamin A is deficient, mucosal epithelium becomes squamous and the turnover of cells decreases. The measles virus infects and damages epithelial tissues throughout the body; serum concentrations of vitamin A, even in well nourished children, may decrease to less than the levels observed in malnourished children. Thus, during measles, children with marginal liver stores of vitamin A may develop an acute vitamin A deficiency, resulting in eye damage and possibly increased deaths from respiratory diseases and diarrhoea.

Sommer *et al.* (1983) demonstrated an increased mortality in children with mild vitamin A deficiency. Sommer *et al.* (1984) documented an increased risk of respiratory disease and diarrhoea in children with pre-existing mild vitamin A deficiency. Measles, coupled with acute vitamin A deficiency, poses an important risk of xerophthalmia in many developing countries.

Frieden *et al.* (1992) measured vitamin A levels in 89 children younger than 2 years with measles, and in a reference group, in New York City, NY. Vitamin A levels in children with measles ranged from 0.42 to 3.0 micromol/L; 22% were low. Children with low levels were more likely to have fever of 40 deg C or higher, to have fever for 7 days or more and to be hospitalised. They also had lower measles-specific antibody levels. The authors suggested that clinicians may wish to consider vitamin A therapy for children younger than 2 years with severe measles. They also felt that more studies of vitamin A should be done in measles and other infectious diseases, and in vaccine efficacy trials.

Weiss (1992) reported on the WHO's suspension of an experimental high-titre Edmondson-Zagreb measles vaccine. Children in some third-world countries seemed well-protected from measles but had an increased risk of dying from a variety of other diseases in the years following administration of this vaccine.

The researchers were baffled. However, it is quite clear that contracting and overcoming measles primes and matures the immune system and increases immunity to a host of other diseases.

Children in the third world countries need improved vitamin A and general nutritional status, not vaccines.

These publications call clearly for the way out of the sordid and embarrassing situation caused by the wholesale failure of measles vaccination. Despite his belief that he has new clothes, the king is in fact naked. The time has come to see the most obvious, namely that, like other vaccines, measles vaccines simply do not work. The time has come for the orthodox medical system finally to learn the basic facts about infectious diseases of childhood and, like the Swiss physicians, start respecting nature and recognise infectious diseases for the value they bring to children.

In April 1993, the Ministry of Health and Welfare in Japan decided to discontinue the use of measles, mumps and rubella vaccine [Sawada *et al.* 1993; Lancet; **342** (7 August): 371]. This decision was prompted by published reports of vaccinated children and their (unvaccinated) contacts contracting mumps from the MMR vaccine, and reports of one in 1044 vaccinees developing encephalitis.

## References


1. Langmuir, A.D., 1962.
Medical importance of measles.
International Conference on measles immunization, Nov 7-9, Bethesda.
Am J Dis Child; **103**: 54-56.
2. Albonico, H., Klein, P., Grob, C., and Pewsner, D., 1990.
Vaccination campaign against measles, mumps, and rubella. A constraining project for a dubious future? Working group of doctors for selective MMR vaccination.
18 pp. Unpublished.
3. Ronne, T., 1985.
Measles virus infection without rash in childhood is related to disease in adult life.
Lancet, 5 Jan: 1-5.
4. Miller, D.L., 1964.
Frequency of complications of measles, 1963. Report of a National Inquiry by the Public Health Laboratory Service in Collaboration with the Society of Medical Officers of Health.
Br Med J; **2**: 75-78.
5. Rauh, L.W., and Schmidt, R., 1965.
Measles immunization with killed virus vaccine.
Am J Dis Child; **109**: 232-237.
6. Fulginiti, V.A., Eller, J.J., Downie, A.W., and Kempe, C.H., 1967.
Altered reactivity to measles virus. Atypical measles in children previously immunized with inactivated measles virus vaccines.
J Am Med Ass; **202 (12)**: 1075-1080.
7. Scott, T.F., Bonanno, D.E., 1967.
Reactions to live-measles-virus vaccine in children previously inoculated with killed-virus vaccine.
N Engl J Med; **277 (5)**: 248-251.
8. Buser, F., 1967.
Side reaction to measles vaccination suggesting the Arthus phenomenon.
N Engl J Med; **277 (5)**: 250- 251.
9. Lennon, R.G., and Isaacson, P., 1967.
Delayed dermal hypersensitivity following killed measles vaccine.
J Pediatrics; **71 (4)**: 525-529.
10. Nader, P.R., Horwitz, M.S., and Rousseau, J., 1968.
Atypical exanthema following exposure to natural measles.
Eleven cases of children previously inoculated with killed vaccine.
J Pediatrics; **72 (1)**: 22-28.
11. Barratta, R.O., Ginter, M.C., Price, M.A., Walker, J.W., Skinner, R.G., Prather, E.C., and David, J.K., 1970.
Measles (Rubeola) in previously immunized children.
Pediatrics; **46 (3)**: 397-402.

12. Conrad, J.L., Wallace, R., and Witte, J.J., 1971.
The epidemiologic rationale for the failure to eradicate measles in the Unites States.
Am J Public Health; **61 (11)**: 2304-2310.
13. Plotkin, S.A., 1973.
Failures of protection by measles vaccine.
J Pediatrics; **82**: 908-911.
14. Cherry, J,D., Feigin, R.D., Lobes, L.A. Jr, Hinthorn, D.R., Schackelford, P.G., Shirley, R.H., Lins, R.D., and Choi, S.C., 1972.
Urban measles in the vaccine era: a clinical, epidemiologic and serologic study.
J. Pediatrics; **81**: 217-230.
15. Cherry, J.D., Feigin, R.D., Schackelford, P.G., Hinthorn, D.R., and Schmidt, R., 1973.
A clinical and serologic study of 103 children with measles vaccine failure.
J Pediatrics; **82**: 802-808.
16. Linnemann, C.C., Hegg, M.E., Rotte, T.C., *et al.*, 1973.
Measles IgM response during re-infection of previously vaccinated children.
J Pediatrics; **82**: 798-801.
17. Landrigan, P.J., and Witte, J.J., 1973.
Neurologic disorders following live measles-virus vaccination.
J Am Med Ass; **223 (13)**: 1459-1462.
18. Karlsson, L., and Scheibnerova, V., 1991.
Association between non-specific stress syndrome, DPT injections and cot death.
Pre-print of a paper delivered to the 2nd Immunisation Conference, May 27-29, Canberra 1991.
19. Roden, A.T., 1974.
Fits following immunization.
Proc. Roy Soc Med; **67**: 24.
20. Witte, J.J., and Axnick, N.W., 1975.
The benefits from 10 years of measles immunization in the United States.
Public Health Report, 90 (3): 205-207.
21. Rand, K.H., Emmons, R.W., and Merigan, T.C., 1976.
Measles in adults: An unforeseen consequence of immunization?
J Am Med Ass; **235**: 1028-1031.
22. Bellanti, J.A., 1971.
Biologic significance of the secretory gamma A immunoglobulins.
E. Mead Johnson Award Address.
Pediatrics; **48 (5)**: 715-729.
23. Cherry, J.D., 1980.
The "new" epidemiology of measles and rubella.
Hospital Practice; July 1980: 49-57.
24. Reuman, P.D., 1979.
Measles: ready for eradication?
Ann Int Med; **90 (6)**: 978-980.
25. Hinman, A.R., 1979.
The oportunity and obligation to eliminate measles from the United States.
J Am Med Ass; **242 (11)**: 1157-1162.

26. Nichols, E.M., 1979.
Atypical measles: a continuing problem.
Am J Public Health; **69 (2)**: 160-162.
27. Hardy, I.R.B., Lennon, D.R., and Mitchell, E.A., 1987.
Measles epidemic in Auckland 1984-85.
NZ Med J; 13 May: 273- 275.
28. Gustafson, T.L., Lievens, A.W., Brunell, P.A., Moellenberg, R.G., Christopher, B.S., Buttery, M.G., and Sehulster, L.M., 1987.
Measles outbreak in a fully immunized secondary-school population.
N Engl J Med; **316 (13)**: 771-774.
29. Frank, J.A., Orenstein, W.A., Bart, K.J, Bart, S.W., El-Tantawy, N., Davis, R.M., Hinman, A.R., 1985.
Major impediments to measles elimination.
Am J Dis Child; **139**: 881-888.
30. Black, F.L., Berman, L.L., Reichelt, C.A., de Pinheiro, P., Da Rosa, A.T., Figueira, F., Gonzales, E.S., 1984.
Inadequate immunity to measles in children vaccinated at an early age: effect of revaccination.
Bull WHO; **62 (92)**: 315-319.
31. Lennon, J.L., and Black, F.L., 1986.
Maternally derived measles immunity in era of vaccine-protected mothers.
J Pediatrics; **108 (1)**: 671-676.
32. Koprowski, H., 1962.
The role of hyperergy in measles encephalitis.
Am J Dis Child; **103**: 103-108.
33. Barclay, A.J.G., Foster, A., and Sommer, A., 1987.
Vitamin A supplements and mortality related to measles: a randomised clinical trial.
Br Med J; **294**: 294-296.
34. Sommer, A., Tarwotjo, E., Djunaedi, F., 1986.
Impact of Vitamin A Supplementation on childhood mortality: A randomised controlled community trial.
Lancet; **1**:1169-1173
35. Sommer, A., Katz, J., and Tarwotjo, E., 1984.
Increased risk of respiratory disease and diarhea in children with pre-existing mild vitamin A deficiency
Am J Clin Nutr; **40**: 1090-1095.
36. Frieden, T.R., Sowell, A.L., Hening, K.J., Huff, D.L., and Gunn, R.A., 1992.
Vitamin A levels and severity of measles. New York City.
Am J Dis Child; **146**: 182-186.
37. Anonymous, 1992.
Two MMR vaccines withdrawn.
Lancet; **340**: 722.
38. Weiss, R., 1992.
Measles battle loses potent weapon
Science; 258 (23 October): 546-547.

# 4

# MUMPS: is there really any need to prevent it?

Mumps is a common childhood disease which is benign in the vast majority of cases. It is desirable that mumps be contracted in early childhood because, when contracted in adulthood, the disease may cause meningitis and/or damage to the testes, ovaries, auditory nerves or pancreas. However, and equally importantly, women are less likely to contract ovarian cancer if they have had mumps during childhood (West 1966).

### Vaccine ineffective and dangerous

The push for general vaccination (which is of dubious value to the recipients) overshadowed and suppressed the wisdom of understanding the enormous value infectious diseases of childhood bring to the recipients. As the group of Swiss medical doctors opposing mass vaccination against measles mumps and rubella say in their memorandum:

*"We have lost the common sense and the wisdom that used to prevail in the approach to childhood diseases. Too often, instead of reinforcing the organism's defences, fever and symptoms are relentlessly suppressed. This is not always without consequences over the development of the disease."*

[Albonico *et al.* (1990)].

The history of mumps vaccination is a repetition of the history of vaccination against other childhood diseases like measles, rubella or whooping cough. For decade after decade medical journals have been publishing information on side-effects and ineffectiveness of vaccines to prevent the diseases, side-by-side with papers claiming an illusory victory in the battle against these diseases.

Authors writing on mumps meningitis or meningoencephalitis agree that they are usually remarkably benign conditions. When they occur, they usually appear within a few days of parotid enlargement and recovery follows without complications in three to four days. In some cases meningeal symptoms may precede parotitis by as much as ten days.

Major neurological complications are much less common and there may be a considerable delay in their appearance (up to 23 days following infection). Clinical features of encephalitis include drowsiness, irritability when disturbed, dizziness, convulsions, headache, psychoses, ataxia and hemiplegia (Russel and Donald, 1958 and others). However, the most common clinical pattern of mumps is parotitis (enlargement of the parotid salivary gland, or occasionally the testes).

As with other infectious diseases of childhood, the temptation to prevent mumps by vaccination was irresistible. The first vaccines were developed and tested in the 1950s. Henle *et al.* (1959) reported on testing of commercially produced mumps vaccines derived from infected chick embryos; the mumps virus was inactivated with formalin. They concluded that "*... single dose of vaccine may be insufficient to produce immunity in a significant proportion of susceptible individuals for a reasonable period of time.*''

Their study set out to evaluate the effect of repeated doses on the incidence and height of neutralising antibody and to determine the optimum spacing of injections. Because they expected only a transient immunity due to vaccine administration, they used young children who "*...would still have ample opportunity to acquire permanent immunity due to natural infection prior to adolescence.*"

The vaccine, produced by the American Cyanamid Company was tested in three institutions for orphans and retarded children. The ages of children were one to five years in two institutions and five to eight years in the third.

It is interesting to note that in one of the institutions there had been no case of mumps noted during the three years preceding the testing of the vaccine, but the first case of mumps occurred three months

into the experiment. The cases kept occurring for several months during the experiment. Those children who were given one vaccine injection contracted mumps at a slightly higher rate than unvaccinated (31.8% and 30.2% respectively) while the incidence of clinical mumps was 4.2-7.7% in children receiving two or more doses of the vaccine. Thus multiple doses of vaccine prevented five out of six children from displaying clinical disease for periods of at least six months.

On the other hand, the rate of total infections (clinical and subclinical) was high and of similar order — 86%-91% — in all groups. One has to question the wisdom of suppressing the clinical symptoms of mumps by vaccine administration. The negative and undesirable long-term consequences of this phenomenon in measles has been documented by Ronne (1985) who demonstrated that those individuals who did not develop a proper clinical measles with rash were more likely to contract cancer and degenerative diseases of cartilage and bone than those who did develop proper measles rash.

Weibel *et al.* (1967) tested live, attenuated mumps-virus vaccine on a group of children with high socio-economic background. A proportion of the children who received mumps vaccine and controls also received one of the respiratory vaccines (formalin-killed respiratory syncytial, parainfluenza 1 and 2 and 3 viruses and *M. pneumoniæ*). Again, as in the previous study, mumps outbreak occurred during this experiment. A number of children contracted mumps within sixteen days of vaccine injections. These cases were excluded from the calculations on the assumption that *"in such children mumps usually developed as a result of the natural exposure ...and the occurrence of mumps as a result of vaccination was ruled out."* However, considering the similar precedent in the previous experiment, there was a strong possibility that this mumps outbreak also could have been caused by vaccine injections.

In a subsequent paper, Hilleman *et al.* (1967) tabulated cases of clinical mumps in this outbreak (figure 1). It is of utmost interest that several cases (the authors did not specify exactly how many) of mumps occurred within 16 days of the beginning of the study and

the outbreak was consistent for several months, with a major culmination at four and a half months into the study. The fact that the authors manipulated the circumstances of this outbreak by "discarding" the initial cases from calculations made their evaluation of this experiment questionable. In their conclusions the authors wrote that, in contrast to measles vaccine which can cause fever and rash in a number of vaccinees, the mumps vaccine does not cause clinically apparent infection. This statement, however, is invalidated by the occurrence of several cases of clinical mumps within 16 days of vaccine injections.

Similar manipulation of data evaluating mumps vaccine efficacy occurred during another field evaluation of Jeryl Lynn level B strain of live virus mumps vaccine described by Sugg *et al.* (1968). 36 cases of clinical mumps occurred among the vaccinated children, 28 of which occurred between 1 and 14 days after vaccine injections. Only 3 cases occurred between 15 and 30 days and 5 cases between 31 and 180 days. In contrast to this, of the 20 cases of clinical mumps occurring among the unvaccinated (placebo group), only 4 occurred between days 1-14, 3 between 15 to 30 and 13 between 31 and 180 days.

It would be too much of a coincidence if, in both trials, a large number of children contracted mumps within 14 days after injections, allegedly quite independently of the effect of the vaccines. Rather, in my opinion, these documented experiences show that injecting live mumps virus shortens the incubation time (as has been documented with other viral vaccines) and that these cases are indeed vaccine-related. This explanation is made even more plausible when one considers the observed fact that in the placebo group the same low number of cases occurred between 1-14 days and 15-30 days. Three to four times as many cases occurred between 31 and 180 days after the placebo injection, which would indicate a normal incubation time associated with natural infection.

If, as the above authors assumed, all 28 cases of mumps within 1 to 14 days were cases of children incubating mumps by coincidence from the day they were injected with mumps vaccine, there should

have been increased numbers of cases of clinical mumps in the placebo group as well. This was not the case.

Another interesting finding was that 65% of those tested for antibody titers before vaccination showed immunity against mumps.

Gunby (1980) reported on the occurrence of atypical mumps in previously vaccinated children during an outbreak of mumps. Symptoms included fever, appetite loss, nausea, malaise and a 24-hour erythematous papular rash which was generalised except for the head, neck, palms and soles.

A University of Arkansas College of Medicine paediatrician, Dr Yamauchi, investigated five cases of this puzzling disease and concluded that it was atypical mumps. All five of these children were immunised against mumps long before their symptoms developed. His conclusion was based on the following facts:

1. Although mumps virus was not recovered from any of the five patients, all of them had fourfold or greater changes (from acute to convalescent) in mumps virus neutralising antibody titer.
2. No other potentially causative viruses were isolated from the patients despite extensive serological studies.
3. The illness occurred while *"wild natural mumps virus was circulating among unimmunised schoolmates of the five patients"*.
4. The affected children had been immunised against mumps five to seven years earlier, but at different times and places and with vaccines from different sources.
5. Vaccine failure seems unlikely since all five had antibody titer levels suggesting previous exposure to mumps virus antigen and none of the lots from which their vaccines were taken had been incriminated as faulty.

All five patients received Jeryl Lynn live attenuated mumps virus vaccine which is believed to produce protective antibody levels in 95% to 98% of recipients. Despite all this, an expert panel organised by the FDA has confirmed the safety and effectiveness of the vaccine and endorsed its vigorous use in childhood immunisation programs

in the United States. Dr Yamauchi concluded that previously immunised children may have altered responses to the wild mumps virus and a new spectrum of clinical symptoms may be appearing in these children.

Fiumara and Etkind (1982) described mumps outbreak in Westwood, Massachusetts in 1981. 33 cases occurred altogether, of which 29 were vaccinated; 2 were unvaccinated and1 did not know.

The authors claimed that the efficacy of the monovalent vaccine was 130%. However, another look at their figures gives quite a different picture. The total number of students in the affected classes (9-12) was 1043. Of these, 455 were immunised with monovalent vaccine and 438 with MMR. One hundred and thirty were unimmunised. The likelihood of contracting mumps in these two groups was the same — 2.6% (2 out of 130 unvaccinated and 29 out of 893 vaccinated). Vaccination did not protect at all even though 97.41% of those vaccinated did not contract the disease since 97.4% of the unvaccinated did not contract mumps in this outbreak either.

This outbreak demonstrates very clearly that there are factors other than the presence of antibody which prevent any person from contracting mumps at any given time.

MMWR (27 July 1983) reported on an outbreak of 63 cases of mumps in six schools (Atlantic County, New Jersey). These 63 cases represented a 40% increase over the previous year's mumps cases. Before 1978, mumps vaccination was not required for school entry in New Jersey. However, since 1978 mumps vaccination was required for school entry for children seven years of age or younger. The enquiry found that vaccination compliance was 95% and that poor compliance with the law was not the reason for this outbreak.

Just as mumps itself can be associated with meningitis, so too may the mumps vaccine. Natural and vaccine strains of mumps viruses are neurotropic and meningotropic.

Gray and Burns (1989) confirmed that it is the vaccine strain itself and not the wild virus that causes vaccine-related mumps meningitis. The virus isolated from the cerebrospinal fluid 21 days after vaccination was identical to the vaccine strain (Urabe Am 9).

Bottiger (1987) reported 19 cases of serious neurologic sequelæ probably or possibly associated with the Jeryl Lynn mumps strain in Sweden (1982 to 1984) of which 18 recovered completely.

Ehrengut (1989) described 27 cases of vaccine-related neurological complications in Germany with Jerryl Lynn and Urabe Am 9 strains.

Von Muhlendahl (1989) commented on 8 cases of Urabe Am 9 vaccine-associated meningitis from Canada and 1 case from Germany and pointed out that the incubation time of this vaccine-associated mengititis was 21 days. In one of the patients echoviruses were cultured from the lymph nodes and the child died on the 10th day after vaccination. He thought that parents need not be told about the risk before deciding on vaccination.

Forsey *et al.* (1992) have been collecting mumps viruses from cases of parotitis and meningitis after MMR vaccination. They confirmed that mumps vaccine viruses (Urabe and Jerryl Lynn) were isolated from cases of post-vaccinal meningitis.

Arday *et al.* (1989) analysed the US Army's experience with mumps hospitalisations for the years 1980 to 1986. The rates of mumps declined from 3.85/100,000 active duty soldiers per year in 1980 to 1.28 in 1985. However, the rate of mumps in 1986 was 6.65/100,000; three quarters of cases occurred in soldiers with three years or less of service. Reported complications were mild.

A cost- benefit analysis of vaccinating all soldiers showed that the cost of hospitalisation of mumps cases ($61,525) was less by a factor of 4.7 than the cost of annual vaccination ($286,789). Mumps incidence would have to reach 15/100,000 before savings on avoided hospitalisations would equal the cost of annual vaccination. Only vaccinating susceptible individuals could show benefit.

Champagne and Thomas (1988) reported on a case of mumps meningitis in a 14-year old girl with no previous history of MMR vaccination after an injection of Trivirix vaccine.

Mumps virus was identified but it was allegedly not possible to differentiate whether this was a wild mumps or vaccine virus strain.

Falk *et al.* (1989) studied the epidemiology of mumps in southern Alberta (Canada) over the years 1980-1982. The authors concluded their investigations by stating that mumps is a generally mild disease, tending to occur in early life, with no documented mortality and very little hospitalisation in Canada; less than 20% of people enter adult life with susceptibility to it. A very high rate of mumps is inapparent clinically and many infections with only respiratory symptoms were accompanied by the development of immunity to mumps. (Hippocrates linked respiratory symptoms to mumps-like illness). The need to immunise against mumps was not clear. However, in spite of the mildness of the disease, a program of immunisation of all children with measles-mumps-rubella vaccine was considered cost effective.

Gray and Burns (1989) described a case of mumps meningitis in a 3 year old girl. The child received full diphtheria, tetanus and whooping cough immunisation with oral poliomyelitis and, 21 days before her illness began, she had received MMR (Pluserix) vaccine. Her temperature rose to 38 degC, she was drowsy, irritable and had a mild meningeal irritation. She was treated with paracetamol (?!?) and recovered after eight days. The authors warned that general practitioners and paediatricians should be aware of the (rare) occurrence of mumps (vaccine) meningitis.

Nalin (1992) wrote that

*"no enhancement of immunogenicity or protection has been demonstrated to be associated with Urabe strain vaccine's increased reactogenicity ... Indeed, several studies indicate that Urabe meningitis may be a marker for a generally heightened reactogenicity of the Urabe vaccine, including high rate of vaccine-associated mumps, lymphadenopathy, fever, restlessness and vomiting."*

The reason for this may be that the Urabe strain is underattenuated. However, it is perhaps of some relevance that Nalin *(op.cit.)* was representing a drug company producing one mumps vaccine, while the Urabe strain was marketed by a different company.

An interesting article was published in 1988 by Cochi *et al.* on perspectives on the relative resurgence of mumps in the United States. The authors wrote that the live virus mumps vaccine was licensed in 1967 but it took a decade before the vaccine was endorsed as a routine vaccination and acceptance was only gradual. Despite this low acceptance, the incidence of mumps declined. Since 1986 the US had experienced a resurgence of mumps, characterised by an increasing number of middle and high school students contracting the disease.

Despite Cochi *et al.*'s statements to the contrary, mumps has been documented to have occurred in vaccinated as well as unvaccinated persons. They concluded that the relative resurgence of mumps in the United States was due to a failure to vaccinate all susceptible persons.

This conclusion is not supported by published information on the incidence of mumps and rate of immunisation, which was very high in the discussed period [even according to Cochi *et al.* (1988) 80 million doses have been administered].

Chaiken *et al.* (1987) quite independently showed the same thing, namely that while the compliance to vaccinate was low, the incidence of mumps was low. As the compliance increased, mainly due to enforced vaccination, quite substantial outbreaks of mumps started occurring. An outbreak in 1983 in the Egg Harbor Township school district in Atlantic City represented a 40% increase over the previous year's total mumps cases among schoolchildren in the state.

Despite the fact that many cases occurred in vaccinated children (indeed in the sixth grade 12 out of 13 were vaccinated) and despite concluding that poor compliance with the school vaccination law did not lead to this outbreak, a massive vaccination effort was started during this outbreak.

Only one of the 11 unvaccinated children did not contract clinical mumps. The attack rate in vaccinated and unvaccinated was 7% and 9% respectively.

Sullivan *et al.* (1985) unwittingly provided evidence for the same phenomenon: an upsurge of mumps outbreaks after mumps vaccine

became a requirement for school children. While the incidence of mumps was low, only two states required mumps vaccination in 1976, while in 1983 30 states required mumps vaccination as a condition of school entry.

Another interesting example of the misconceptions about the effect and effectiveness of mumps (measles, mumps and rubella) vaccination is the paper published by Peltola *et al.* (1986). The authors claimed that a drastic fall in the incidence of mumps has occurred as a direct result of the implementation of the immunisation programme to eliminate measles, mumps and rubella from Finland which started in November1982.

Their figure 1 shows the incidence of measles, mumps and rubella cases as reported to the National Board of Health between January 1980 and April 1985. This figure shows that the incidence of mumps was falling steadily from 1980 for three years before general vaccination started, and the fall continued at the same pace after general vaccination started. There is no evidence in this graph to support the authors' assumption of the alleged effect of mumps vaccination. The same applies to measles and rubella. As far as rubella was concerned there was a sharp rise in incidence in early 1985, reflecting the recent epidemic of rubella in Scandinavia generally. This represents further evidence to support the conclusion that vaccination did not affect the incidence and general downward trend in the incidence of rubella; quite to the contrary, one can claim that intensified vaccination pushed the incidence upward against the natural downward trend pre-existing since at least 1980.

From January to July 1987 an epidemic of mumps occurred in Chicago. A total of 106 cases occurred in persons older than 20 years. This epidemic culminated with a major outbreak of mumps (119 cases) at the Chicago futures stock exchange [Kaplan *et al.* (1988)]. Three of these adults had documented evidence of immunisation; ages ranged from 17 to 70 years. This epidemic is also interesting in that it occurred after an intensified push for mumps vaccination.

It is significant that the 1967-1977 decade with very little mumps vaccination activity was a decade of very low mumps incidence. This

clearly implicates the widespread use of the vaccine in the documented increase of the incidence of mumps in the United States in the eighties.

How hell-bent on vaccination, irrespective of side-effects, the vaccinators are, is demonstrated by a small notice in the Lancet (2 January 1993). Due to meningitis associated with measles-mumps-rubella vaccine, two combined measles-mumps-rubella vaccines containing the Urabe mumps virus strain were withdrawn from use in the UK. However, the company producing these vaccines announced that it will continue to produce and supply vaccines that contain Urabe mumps strain, so that existing immunisation programmes in areas where no alternative mumps vaccine is available, need not be suspended.

## References


1. West, R.O., 1966.
Epidemiologic studies of malignancies of the ovaries.
Cancer, July: 1001-1007.
2. Russel, R.R., and Donald, J.C., 1958.
The neurological complications of mumps.
Br Med J; 5 July : 27-30.
3. Albonico, H., Klein, O., Grob, C., and Pewsner, D., 1990.
Vaccination campaign against measles, mumps, and rubella.
A constraining project for a dubious future?
Proclamation of the Doctors' Working Group for selective MMR vaccination.
Unpublished.
4. Henle, W., Crawford, M.N., Henle, G., Faz Tabio, H., Deinhardt, F., Chabau, A.G., and Olshin, I.J., 1959.
Studies on the prevention of mumps. VII. Evaluation of dosage schedule for inactivated mumps vaccine.
J Imunology; **83**: 17-28.
5. Ronne, T., 1985.
Measles virus infection without rash in childhood is related to disease in adult life.
Lancet, 5 January: 1-5.
6. Weibel, R.E., Stokes, J., Buynak, E.B., Whitman, J.E., and Hilleman, M.R., 1967.
Live, attenuated mumps-virus vaccine.
N Engl J Med, **276 (5)**: 245-251.
7. Sugg, W.C., Finger, J.A., Levine, R.H., and Pagano, J.S., 1968.
Field evaluation of live virus mumps vaccine.
J Pediatr; **72 (4)**: 461-466.
8. Hilleman, M.R., Weibel, R.E., Buynak, E.B., Stokes, J., and Whitman, J.E., 1967.
Live, attenuated mumps-virus vaccine.
4. Protective efficacy as measured in a field evaluation.
N Engl J Med; **276 (5)**: 252-258.
9. Gunby, P., 1980.
"Atypical" mumps may occur after immunization.
J Am Med Ass; **243 (23)**: 2374-2375.
10. Fiumara, N.J., and Etkind, P.H., 1982.
Mumps outbreak in Westwood, Massachusetts — 1981.
Epidemiologic Notes and Reports.
MMWR; **33 (29)**: 421-430.
11. Fiumara, N.J., and Etkind, P.H., 1982.
Mumps outbreak — New Jersey.
MMWR; **33 (29)**: 421-430.
12. Gray J.A., and Burns, S.M., 1989.
Mumps meningitis following measles, mumps and rubella immunisation.
Lancet; **2**: 98.

13. Gray J.A., and Burns, S.M., 1989.
Mumps vaccine meningitis.
Lancet, **2**: 927.
14. Bottiger, M., Christenson, B., Romanus, V., Taranger, J., and Strandell, A., 1987.
Swedish experience of two dose vaccination programme aiming at eliminating measles, mumps and rubella.
Br Med J; **295**: 264-267.
15. Ehrengut, W., 1989.
Mumps vaccine and meningitis.
Lancet; **2**: 751.
16. Von Muhlendahl, K.E., 1989.
Mumps meningitis following measles, mumps and rubella immunisations.
Lancet, 12 August: 394.
17. Anonymous, 1989.
Mumps meningitis and MMR vaccination.
Lancet; 28 October: 1015-1020.
18. Thomas, E., 1988.
A case of mumps meningitis: A complication of vaccination?
Can Med Assoc J; **138** (15 Jan): 135.
19. Forsey, T., Bentley M.K., and Minor, P.D., 1992.
Mumps vaccines and meningitis.
Lancet; **340**: 980.
20. Arday, D.R., Kanjarpane, D.D., and Kelley, P.W., 1989.
Mumps in the US Army 1980-86: Should recruits be immunized?
Am J Dis Child, **79 (4)**: 471-474.
21. Champagne S., and Thomas, E., 1988.
A case of mumps meningitis: a post-immunization complication?
Can Dis Weekly Rep; **13-35**: 155-156.
22. Falk, W.A., Buchan, K., Dow, M., Garson, J.Z., Hill, E., Nosal, M., Tarrant, M., Westbury, R.C., and White, F.M.N., 1989.
The epidemiology of mumps in southern Alberta.
Am J Epidemiol; **130 (4)**: 736-749.
23. Nalin, D. R., 1992.
Evaluating mumps vaccines.
Lancet; 1 Feb: 305.
24. Cochi, S.L., Preblud, S.R., Orenstein, W.A., 1988.
Perspectives on the relative resurgence of mumps in the United States.
Am J Dis Child; **142**, May: 499-507.
25. Chaiken, B.P., Williams, N.M., Preblud, S.R., Parkin, W., and Altman, R., 1987.
J Am Med Ass; **257 (18)**: 2455-2458.
26. Sullivan, K.M., Halpin, T.J., Kim-Farley, R., and Marks, J.S., 1985.
Mumps disease and its health impact: an outbreak-based report.
Pediatrics; **76 (4)**: 533-536.

27. Peltola, H., Kurki, T., Virtanen, M., Nissinen, M., Karanko, V., Hukkanen, V., Penttinen, K., and Heinonen, O.P., 1986.
Rapid effect on endemic measles, mumps, and rubella of nationwide vaccination programme in Finland.
Lancet, 18 Jan: 137-139.

28. Kaplan, K.M., Marder, D.C., Cochi, S.L., and Preblud, S.R., 1988.
Mumps in the Workplace. Further evidence of the changing epidemiology of a childhood vaccine-preventable disease.
J Am Med Assoc; **260(10)**: 1434-1438.

29. Anonymous, 1993.
Urabe stays on.
Lancet; **341**: 46.

# 5

# RUBELLA: another useless and dangerous exercise

Rubella is an innocuous infectious disease. The vast majority of people contract rubella and develop a life-long effective immunity to it. Side-effects are extremely rare. The most serious side effects occur when a woman contracts rubella in the first trimester of pregnancy because the rubella virus may cause deformities in the fetus.

In the US rubella vaccine was licensed in 1969. However, its effectiveness and safety remain questionable.

As Cherry (1980) wrote, despite distribution of over 83 million doses of rubella vaccine since 1969, there were periodic upswings in incidence. There was also a shift in the age groups susceptible to rubella. *"Essentially, we have controlled the disease in persons 14 years of age or younger but have given it a free hand in those 15 or older."* He continued *"Of course, the point of rubella immunization is not prevention of rubella but preventing of the congenital rubella syndrome. Since 1969 and 1970, when the CDC's National Congenital Rubella Registry listed 78 and 90 cases respectively, the number of cases has declined and apparently has remained relatively stable at about 30 to 40 per year (data for the last three years or so must be regarded as incomplete because of a common lag in recognition of the syndrome)."*

Then he continued: *"This decline in congenital rubella is curious because number of infections in women of childbearing age has remained the same. It is perhaps artifactual and explained by the fall in the fertility rate in the United States and also the more frequent use of therapeutic abortion. Anyway, it is clear that the apparent stability in the control of congenital rubella is precarious."*

Cherry (1980) said several important things in one breath:

- that rubella vaccination was ineffective in eradicating rubella disease (on p.56 Cherry says that re-infection has been noted in patients with vaccine-induced or natural antibody),
- that vaccination pushed the age of contracting rubella upwards into groups where rubella is undesirable, and
- although the purpose of rubella vaccination was prevention of congenital rubella syndrome, this was really being controlled by some other factor — namely, elective abortion.

So why continue vaccinating against this normally innocuous disease, especially considering the well-documented side-effects of vaccination with available vaccines?

In children, skin rash and lymphadenopathy following rubella vaccination have been described, as well as transient arthritis [Cooper *et al.* (1969)] and pain syndrome in the wrists, hands and knees accompanied by a crouch [Kilroy *et al.* (1970)].

Gilmartin *et al.* (1972) described 36 children with myeloradiculoneuritis syndrome following a mass rubella vaccine program. The reaction was seen with equal frequency in children given the HPV-77 DK12, HPV-77 DE5, and Cendehill vaccine. The highest incidence of reaction was seen in preschool children and appeared up to six weeks after vaccination. In many children the joint pain was recurrent over a long period of time. Abnormal nerve conduction velocity was a consistent laboratory finding.

Spruance *et al.* (1972) described recurrent joint symptoms in children six to eight months old after they received the HPV- 77 dog kidney rubella vaccine. The symptoms appeared two to seven weeks after vaccination and there were recurrent attacks at one to three month intervals lasting one to seven days.

The question of effectiveness of rubella vaccine soon became a hot issue just as with the other vaccines: in epidemics of rubella a large percentage of victims were vaccinated [Rauh *et al.*(1972)]. Nevertheless, like other authors dealing with vaccine problems, these authors recommended intensified rubella vaccination.

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Klock & Rachelefsky (1973) described a rubella epidemic (over 1000 cases) between January and May 1971 in Casper, Wyoming. This occurred nine months after a rubella vaccination program in which 83% of elementary-school children and 52% of pre-school children were vaccinated. The concept that a highly immune group of prepubertal children will prevent the spread of rubella in the rest of the community was shown by this epidemic to be not valid.

Rachelefsky & Herrmann (1974) reported on congenital rubella syndrome following the above epidemic of rubella in a partially vaccinated community. Twentyfour women contracted serologically documented prenatal rubella infection — seven clinical, 17 inapparent. No infant was born with congenital defects. There was no increase in stillbirths or abortions. The authors concluded that rubella vaccine-induced immunity, although not adequate to prevent the epidemic, probably reduced the occurrence of fetal infection and congenital malformations.

This conclusion is highly hypothetical in that it does not consider the published observation that congenital rubella syndrome does not always occur after exposure to rubella virus in pregnant women and the authors did not consider the small number of cases they studied.

Modlin *et al.* (1975) reviewed a five year experience with rubella vaccine in the United States. On the one hand they claimed a decline in reported rubella and congenital rubella syndrome since1969, concurrent with widespread use of rubella vaccine, and claimed that the vaccine provided durable protection. On the other hand, they admitted to re-infection after rubella vaccination. They also wrote that there is a small but significant incidence of adverse reactions and a potential risk to women who are vaccinated during pregnancy. They also claimed that rubella vaccines as they are *"currently applied"*, are safe and effective.

The word "reported" is of course very significant in understanding reports like this. The general and chronic under-reporting of infectious diseases after the introduction of vaccines of any kind in the US is quite well-known and often criticised [New York Times, The Doctor's World, 7/10/90)].

In that sense Modlin *et al.* (1975) are quite factual. However, the small number of cases due to under-reporting is not and cannot possibly be a reflection of the reality. Also, outbreaks of rubella did not occur only in unvaccinated populations as Modlin *et al.* (1970) claim. Horstman *et al.* (1970), Abrutyn *et al.* (1970), Chang *et al.* (1970) and many others reported on outbreaks of rubella in highly vaccinated populations.

In Australia, a very important study on rubella vaccine was performed and published by Dr Beverly Allan. In 1973 she published her report on two trials of the Cendevax rubella vaccine in army recruits selected because of their lack of immunity as determined by blood test.

The men produced antibodies to rubella after given Cendevax (an attenuated rubella virus). They were then sent to a camp which usually had an annual outbreak of rubella. Three to four months after vaccination, 80% of the men became ill with rubella. A further trial, performed shortly after the army trial, on institutionalised retarded people, resulted in a similar failure of vaccine to protect against rubella disease.

As Kalokerinos and Dettman (1978) report, Sir Henry Yellowlees released a press statement in the London Daily Telegraph (26 February, 1976) and a letter to all doctors saying that despite high vaccination figures, there has been no detectable reduction in the number of babies born with congenital rubella defects.

Forrest and Menser (1977) reported a case of rubella vaccine failure to prevent congenital rubella syndrome. They also warned that about 5% of vaccinees do not sero-convert. Duration of vaccine-induced immunity is not known. Clinical re-infection in vaccinees may occur. A second strain of rubella vaccine— RA 27/3 — appears more effective in preventing re-infection.

In the eighties, a number of published papers appeared which reported on persistent rubella infection, rubella-associated arthritis and other side effects. Chantler & Ford (1982) wrote that acute polyarthritis is a frequent manifestation of both natural infection with rubella virus and immunisation with attenuated rubella virus.

They mentioned one case of arthritis after natural infection and six cases after immunisation with HPV 77 DE5 vaccine persisting for up to six years.

Herrmann *et al.* (1982) studied persistence of antibodies ten years after rubella vaccination with three different vaccines in some 5,153 children on the islands of Kauai and Hawaii. Their figure shows that within four years the level of antibodies halved, compared with their level just after the injections. There was a slight fall between four and seven years, at which level there was a sharp rise in all three groups. This apparent rise in antibodies corresponded temporally with a documented outbreak of rubella on the island of Oahu in June-August 1977.

Herrmann *et al.* (1982) maintained that the outbreak did not affect the islands of Kauai and Hawaii following the Oahu outbreak. However, their table 5 shows that of 110 vaccinees with no HI (hæmagglutinin-inhibition titre) response to rubella vaccination, there were 48 cases of rubella illness which were considered vaccine failures. The authors considered this trial a success although, after ten years, only 741 of the 5,153 original vaccines were enrolled in the study; the rest were lost to follow-up.

In 1969, live attenuated rubella virus vaccine was first licensed in Canada. The justification for rubella vaccination was seen as preventing congenital rubella syndrome (CRS). Spika and Clogg (1983) discussed several aspects of rubella incidence and rubella vaccination, the most interesting, perhaps, being the question of the logistics of assuring compliance with vaccination.

Interestingly enough, the biggest problem appeared to be non-compliance and lack of cooperation from physicians. Orenstein *et al.* (1981) wrote that several reasons were given for refusal to be vaccinated. Fear of unforeseen vaccine reactions, prompted by the Guillain-Barré syndrome seen in influenza vaccine, was the reason given most often by house officers. Coercion by threat of dismissal was considered impracticable, and possibly illegal, so the only alternative seemed to be mandatory immunisation. This is very disconcerting indeed, especially considering that there are many

unanswered questions regarding the duration of vaccine-induced immunity (if any) and the ability of the virus to spread in a community (like the military training camps) considered relatively immune.

Joncas (1983) emphasised that the purpose of rubella vaccination is prevention of the congenital rubella syndrome, because rubella is otherwise a benign disease that does not justify prevention by vaccination. The author compared two vaccination strategies: the British (immunising only a group at risk: prepubertal girls and women of childbearing age), and the United States strategy of vaccinating preferentially, but not exclusively, all girls at age one.

Although this campaign was claimed to be successful, nevertheless there was an outbreak of rubella in California in young adults, the group at greatest risk of transmitting rubella to unborn children. California reported 13 confirmed or probable cases and four possible cases of CRS, as well as six cases of congenital rubella following such an outbreak in 1979.

It is quite important that the author stated that *"If a nationwide increase in the incidence of CRS and rubella in the high-risk group is indeed confirmed for 1981 and 1982, then the greater effort made in the United States from 1979 to 1982 to immunise women of childbearing age may have greatly contributed to it."* He also wrote that *"Since natural rubella infection is almost always benign and confers better immunity than the vaccine, but without additional risks, rubella vaccination is not justified in young children."*

In the United States outbreaks of rubella still occur in adolescents and young adults, despite compulsory immunisation of young children. *"Other factors that are often not considered in evaluating decreases in the incidence of CRS are the number of therapeutic abortions performed when rubella is confirmed in pregnant women... In one instance in which therapeutic abortions were taken into account the result was a 10% increase in the incidence of CRS and the real rate was probably much higher."*

Very importantly, Joncas (1983) wrote that *"Members of national advisory committees on immunization practices in the United States (where compulsory measures are already in effect) and in Canada*

*(where they are being contemplated) should remember that even the most desirable goal never justifies means that violate essential rights, particularly when complications of vaccination can occur. In the United States, compulsory measures should be even less acceptable since, in contrast to Sweden, no compensation program has been established for the rare complications of vaccination. Education, not coercion, is the key to successful immunization programs."*

Then the author warned about the risk of complications of rubella vaccination such as arthritis, usually transient and mild. However, persistent and recurrent arthritis with evidence of virus in the joints following rubella vaccination has been documented.

The rubella virus has even been recovered from peripheral blood leukocytes 2 years after vaccination. The risk of degenerative or immunopathologic long-term complications, such as rheumatoid arthritis, was a cause for concern since attenuated (usually mutant) vaccine strains may persist more readily.

Very importantly, the author stated that although immunity from the RA 27/3 vaccine is better than that of earlier vaccines, it is still probably inferior to that provided by natural infection. He recommended concentrating on the group at risk — women of childbearing age.

The National Advisory Committee on Immunization in Canada made the following recommendations (1983): *"Rubella vaccine should be given routinely to all children of both sexes at 12 months of age or as soon thereafter as possible, preferably in combination with measles and mumps vaccines... A clinical history of rubella is not a reliable indicator of immunity."*

Further: *"Rubella vaccine should be given to all female adolescents and women of childbearing age unless they have either laboratory evidence of detectable antibody or documentary evidence of having received vaccine ... testing need not be undertaken where testing is likely to interfere with acceptance or delivery of vaccine. There are no known adverse effects following administration of vaccine to immune women."*

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Rubella vaccine was contraindicated in pregnant women.

One has to pause a little here and think: the general tone of these recommendations sounds just like the political proclamations made in countries with totalitarian régimes. The medical system in the US and other "free, western" countries is dictating and mandating their procedures (which are principally money-earning exercises) to millions of people and their babies. Unbelievably, the vast majority of people seem quite willing and without protestation to submit their children to vaccination despite the fact the vaccination procedures, especially their long-term effects, aren't even properly followed-up.

The phrase *"there are no known adverse effects following administration of vaccine to immune women"* means only ignorance. There could be unknown adverse long-term effects like those from contaminated polio vaccines. Yet, people seem to be taking these empty and dangerous statements as a true reassurance and guarantee of safety.

The Australian policy of rubella vaccination was summarised by Burgess (1990). The rubella vaccine was licensed in the late sixties; pregnancy was mostly considered a contraindication to its use. However, the author stated that *"To date, there is no evidence that rubella vaccine (of any strain) administered in pregnancy could cause defects suggestive of congenital rubella syndrome in live born infants."* She supported her statement with several instances of published studies. Enders (1985) studied 365 women (including 95 known to be susceptible) who continued to term after vaccination with Cendehill vaccine. IgM antibodies were detected in two infants and virus was recovered from one of 34 *"products of conception"*.

In a British study [Sheppard *et al.* (1986)] 21 live babies were born to 54 women: one had a cardiac murmur. According to Burgess (1990), theoretical risk from the vaccine used in Australia was 4.9%. Because risk to the fetus could not be ruled out entirely, *"pregnancy should remain a contraindication to vaccination and precautions should be taken to avoid vaccinating pregnant women."*

Menser *et al.* (1984) reported on the impact of rubella vaccination in Australia. After 13 years of rubella vaccination there has been a notable increase in the proportion of rubella sero-positive pregnant

women. However, it is also true that 86% of a Melbourne ante-natal clinic's patients were sero-positive without any rubella vaccination. Rubella vaccination of 12-14 year old girls started in 1971. The study also showed that 61% of girls are immune to rubella before vaccination and 76% of men are immune at 18-23 years.

A study of 144 children 2-8 years old showed that 30% were immune. 247 vaccinees were sero-positive 5-8 years after vaccination, while 24% of unvaccinated males of the same age were still sero-negative. However, without previous screening and testing and relating these datat to outbreaks of natural rubella, these figures are really meaningless. This is especially important since congenital rubella was not notifiable until 1983. The only records of importance are records of rubella deafness. Since 1977 there was a striking fall in the incidence of deafness due to congenital rubella. Of course, this fall could be attributed to termination of affected pregnancies. Despite these doubts, the authors concluded that the vaccination program played a major role in this fall by preventing infections in pregnancy.

The report on employee screening programs in Arizona hospitals by Sacks *et al.* (1983) is very interesting. It showed there was only limited interest or response on the part of employees, lack of staff to carry out the program of screening and immunisation, and vaccine reactions. The majority of hospitals had no policy or action with regard to susceptible physicians. Low immunisation rates were most distressing: 53% for all susceptible persons, 22% for physicians and 9% for obstetricians despite their exposure to pregnant patients. Physicians, who were often not salaried, had no restrictions placed on their work areas or association with the hospital if they remained susceptible. The majority of screening hospitals did not take measures to make immunisation (or rather immunity) mandatory regardless of the category of their employees. 63% of hospitals addressed the issue by keeping lists of susceptible persons.

Badenoch (1984) discussed the US policy aimed at interrupting transmission of rubella virus among young children and thus reducing the possibility of exposure of susceptible pregnant women.

However, the lack of fall in incidence of rubella during pregnancy and the absence of a significant drop in incidence of congenital rubella in the seventies showed that these measures were insufficient.

In contrast to this, in the United Kingdom selective immunisation of schoolgirls and susceptible women was adopted. One of the reasons for this policy was uncertainty about duration of vaccine immunity. The continuing circulation of wild virus in young unvaccinated children would be lost with infant immunisation. It would also require a level of compulsion unacceptable in Britain.

Tingle *et al.* (1985) investigated the mechanisms of vaccine failure in 13 adults in Canada. Seven had single immunisations and six had multiple immunisations against rubella with evaluation between six months and eight years. There was a significant medical history: chronic inflammatory joint syndrome (3 persons); recurrent enlargement of the parotid salivary gland (1); common variable hypogammaglobulinæmia (1); chronic lymphocytic thyroiditis (1). In one person, a syndrome of polyarticular arthritis began 2-3 weeks after administration of the RA 27/3 vaccine and has continued with recurrent flareups over the three year follow-up study.

A significant medical history was recorded on one person (psoriasis) in a 20 member group with wild rubella virus infection and in one (lymphosialadenitis) of 37 members of a successful rubella immunisation group. Persistence of rubella virus in peripheral blood mononuclear cells was detected in three persons at 21, 31, and 24 months after immunisation. Rubella virus antigens were detected in mononuclear cell cultures from another individual.

These findings provide evidence of altered or abnormal host immunologic reactivity to vaccine-introduced rubella virus as the principal mechanism of failed vaccine in this group. Most of the failed immunisation group studied had high levels of antibody to rubella virus. Persistent rubella infection in peripheral blood mononuclear cells provided additional evidence for this conclusion. The authors concluded that these findings showed lack of understanding of the nature and significance of altered immunologic responses caused by the current rubella vaccine programmes.

Das *et al.* (1990) described two cases of congenital rubella after previous presumed maternal immunity, one in a vaccinated mother. The authors concluded that the quality of antibody produced in women in whom sero-conversion has occurred after vaccination may be inadequate for total protection compared with that induced by natural infection. They quoted Harcourt *et al.* (1980) who found significant differences in rubella-specific IgG, IgA and IgM responses on subsequent challenge with rubella vaccine between volunteers who were vaccinated and those who acquired natural immunity.

Liebermann (1991) established that rubella virus, both natural and vaccine introduced, has been found to play a major role in chronic fatigue syndrome.

In August 1991, the Institute of Medicine released a report on Adverse effects of pertussis and rubella vaccines. The evidence indicated causal relationship between RA 27/3 rubella vaccine and acute arthritis in 13% to 15% of adult women. However, *"the evidence did not provide for reliable estimates of excess risk of* 'chronic arthritis' *following RA 27/3 vaccine."*

Despite these and many other reports indicating quite serious problems caused by rubella vaccines, the push for rubella vaccination continues. NHMRC (Dr Weekly 1991) reported on the occurrence of congenital rubella syndrome in babies of two mothers who were vaccinated as teenagers. Despite this obvious evidence of vaccine failures, further vaccine is recommended, instead of accepting that MMR vaccination simply does not work.

## References


1. Cherry, J.D., 1980.
The "new" epidemiology of measles and rubella.
Hospital Practice, July 1989: 49-57.
2. Cooper, L.Z., Ziring, P.R., Weiss, H.J., *et al.*, 1969.
Transient arthritis after rubella vaccination.
Am J Dis Child; **118**: 218-225.
3. Kilroy, A.W., Schaffner, W., Fleet, W.F., Lebkowitz, Jr, L.B., Karzon, D.T., and Fenichel, G.M., 1970.
Two syndromes following rubella immunization.
J Am Med Ass; **214 (13)**: 2287- 2292.
4. Gilmartin, R.C., Jabbour, J.T., and Duenas, D.A., 1972.
Rubella vaccine myeloradiculoneuritis.
J Pediatrics; **80 (3)**: 406-412.
5. Spruance, S.L., Klock, L.E., Bailey, J.R., and Smith, C.B., 1972.
Recurrent joint symptoms in children vaccinated with HPV- 77DK12 rubella vaccine.
J Pediatrics, **80 (3)**: 413-417.
6. Rauh, J.L., Schiff, G.M., and Johnson, L.B., 1972.
Rubella surveillance and vaccination at adolescence.
Am J Dis Child; **124**: 27-28.
7. Klock, L.E., and Rachelefsky, G.S., 1973.
Failure of rubella herd immunity during an epidemic.
N Engl J Med; **288 (2)**: 69-72.
8. Rachelefsky, G.S., and Herrmann, K.L., 1974.
Congenital rubella surveillance following epidemic rubella in a partially vaccinated community.
J Pediatrics; **84 (4)**: 474-478.
9. Modlin, J.F., Brandling-Bennett, D.A., Witte, J.J., Campbell, C.C., and Meyers, J.D., 1975.
A review of five years' experience with rubella vaccine in the United States.
Pediatrics; **55 (1)**: 20-29.
10. Horstmann, D.M., Liebhaber, H., LeBovier, G.L., *et al.*, 1970.
Rubella: reinfection of vaccinated and naturally immune persons exposed in an epidemic.
N Engl J Med; **283**:771.
11. Abrutyn, E., Herrmann, K.L.K., Karchmer, A.W., *et al.*, 1970.
Rubella-vaccine comparative study: nine-months follow-up and serologic response to natural challenge.
Am J Dis Child, **120**: 129.
12. Chang, T., DesRosiers, S., and Weinstein, L., 1970.
Clinical and serological studies of an outbreak of rubella in a vaccinated population.
N Engl J Med; **283**:246.

13. Allan, B., 1973.
Rubella immunisation
Aust J Med Tech. **4**: 26-27.
14. Kalokerinos, A., and Dettman, G., 1978.
Does rubella vaccine protect?
Australasian Nurses J, May 1978: 1-4.
15. Forrest, J.M., and Menser, M.A., 1977.
Failure of rubella vaccination to prevent congenital rubella.
Med J Aust; **1**:77.
16. Preblud, S.R., Herrmann, K.L., Orenstein, W.A., and Hinman, A.R., 1981.
Congenital rubella affecting an infant whose mother had rubella antibodies before conception.
Br Med J; **282:** 1235.
16. Chantler J.K., and Ford, D.K., 1982.
Persistent rubella infection and rubella-associated arthritis.
Lancet, 12 June: 1323-1325
17. Herrmann, K.L., Halstead, S.B., and Wiebenga, N.H., 1982.
Rubella antibody persistence after immunization.
J Am Med Ass; **247 (2)**: 193-196.
18. Spika, J.S., and Clogg, D.K., 1983.
Rubella vaccination: a course becomes clear.
Can Med Ass J; **129**: 15 July: 106-110.
19. Orenstein, W.A., Heseltine, P.N.R., LeGagnoux, S.J., and Portnoy, B., 1981.
Rubella vaccine and susceptible hospital employees. Poor physician participation. J Am Med Ass; **245 (7)**: 711-713.
20. Joncas, J., 1983.
Preventing the congenital rubella syndrome by vaccinating women at risk.
Can Med Ass J; **129**: 15 July: 110-112.
21. Editorial, 1983.
Rubella vaccine: recommendations for use.
Can Med Ass J; **129**: 15 July: 105-106.
22. Burgess, M. A., 1990.
Rubella vaccination just before or during pregnancy.
Med J Aust; **152**, May 21: 507-508.
23. Enders, G., 1985.
Rubella antibody titers in vaccinated and nonvaccinated women and results of vaccination during pregnancy.
Rev Inf Dis; **7 (suppl. 1)**: S103-S107.
24. Sheppard, S., Smithells, R.W., Dickson, A., Holzel, H., 1986.
Rubella vaccination and pregnancy: preliminary report of a national survey.
Br Med J; **292**: 727.
25. Menser, M.A., Hudson, J.R., Murphy, A.M., and Cossart, Y.E., 1984.
Impact of rubella vaccination in Australia.
Lancet, 12 May, 1059-1061.

26. Sacks, J.J., Olson, B., Soter, J., and Clark, C., 1983.
Employee rubella screening program.
J Am Med Ass; **249**: 2675- 2678.
27. Badenoch, J., 1984.
Rubella immunisation: whose baby?
Br Med J; **288**: 564-565.
28. Tingle, A.J., Chantler, J.K., Kettyls, G.D., Bryce Larke, R.P. and Schulzer, M., 1985.
Failed rubella immunization in adults: association with immunologic and virological abnormalities.
J Infect Dis; **151** (2): 330-336.
29. Das, B.D., Lakhani, P., Kurtz, J.B., Hunter, N., Wartson, B.E., Cartwright, K.A.V., Caul, E.O., and Rome, A.P.C.H., 1990.
Congenital rubella after previous maternal immunity.
Arch Dis Child; **65**: 545-546.
30. Liebermann, A.D., 1991.
The role of the rubella virus in the chronic fatigue syndrome.
Clinical Ecology; **7** (3): 51-54.
31. Anonymous, 1991.
NHMRC contemplates rubella program change.
The Australian Dr Weekly, 25/10/1992.
32. Howson, C.P., and Fineberg, H.V., 1992.
Adverse events following pertussis and rubella vaccines.
Summary of a report of the Institute of Medicine.
J Am Med Ass; **267** (3): 392-396.

# 6

# FACTS ABOUT *HÆMOPHILUS INFLUENZÆ* B VACCINES

**What every doctor and parent needs to know about the vaccine against a generally harmless bacterium which normally lives in harmony with a healthy individual.**

Invasive infections associated with *Hæmophilus influenzæ* b bacteria (Hib) are serious systemic diseases in young children occurring increasingly in a number of developed countries like the United States, Finland, Norway, England and Australia. Hib is associated with most cases of meningitis, epiglottitis, cellulitis, septic arthritis, osteomyelitis, pericarditis and pneumonia. The incidence of meningitis peaks between 6 and 11 months of age; that of epiglottitis at 2.5 years. About 75% of all forms of Hib occur in children less than 18 months of age and about 25% in children more than 24 months of age. The mortality rate is around 5% and neurological sequelæ occur in 25-35% of those who survive.

A vaccine (Hæmophilus b polysaccharide, polyribosylribitol phosphate or "PRP") was licensed in April 1985 in the US. The interesting thing about this license is that it was not based on clinical studies in the US, but on the "Finnish trial". The US pre-licensing efficacy data came from a randomised clinical trial in Mecklenburg County, NC [Parke *et al.*(1977)] that involved 16,000 children 2 months to 5 years of age.

This study failed to demonstrate significant protective efficacy (only 69%) of this vaccine and also of group C *Neissereia meningitidis* vaccine.

In the Finnish trial of PRP, described by Peltola *et al.* (1977), 48,977 children three months to five years of age were injected with the vaccine, while an equal number of children were given group A meningococcal vaccine and served as controls. The vaccine was found 90% effective, but not in children 3-17 months old.

More importantly, only 1,000 children out of almost 100,000 were followed up for reactions and 499 for serum antibody levels. The authors claimed that there were no serious side effects; however, one child had an anaphylactic reaction and was given adrenalin. Considering that only 1000 children were followed up this is not a low incidence.

It is not surprising that in a follow-up study, one year after the FDA licensed the vaccine, Milstien *et al.* (1987) demonstrated a much higher rate of adverse reactions compared with the Finnish study. They studied 152 recorded cases, excluding those of vaccine failure and what they called concurrent infection [for more detail see Daum *et al.* (1989)] and found several adverse reactions not previously recognised, including convulsions, allergic reactions such as anaphylaxis and serum sickness-like reactions and vomiting.

Because of lack of efficacy and lack of immune response to the PRP vaccine, in December 1987 a new "improved" vaccine (PRP-D conjugate) was licensed by the FDA. This decision was again based on another Finnish trial [Eskola *et al.*(1987)] in which some 30,000 children were injected with PRP-D vaccine. Again, the follow-up study of antibody response was performed on only 99 children, which cannot be considered an adequately representative sample of 30,000 children. In a control group some 30,000 children received 3 DPT and one polio vaccines in contrast to the case group which received 3 DPT, polio and PRP-D vaccines.

The efficacy of the PRP-D vaccine was considered 83%. There were 20 potentially serious adverse reactions, including 1 three month old baby with convulsions 12 hours after injection and 1 baby was non-responsive 3 hours after injection.

In a group of 99 babies, increased irritability was recorded in 41% of the DPT and PRP-D group and 23% after DPT-polio injection.

This represents a quite high incidence of adverse reactions. After nine months there were three cases of invasive Hib infection among those who received 3 doses of PRP-D, with 20 cases among the controls (3 doses of DPTand Polio).

It is very likely that if there had been a true control group of unvaccinated infants, there would have been no cases of invasive Hib infections in unvaccinated babies.

The post-licensing US studies [Black *et al.* (1988), Shapiro *et al.* (1988), Harrison *et al.* (1988)], indicated only 45% to 88% efficacy of the PRP-D vaccines. Black *et al.* (1988) estimated the efficacy at 69% in a "matched case-control" analysis. 35 cases of Hib disease occurred (4 within one week) after injection. Indeed, other studies demonstrated not only a total lack of efficacy [Osterholm *et al.* (1988) Ward *et al.* (1990)] but also the existence of a "window" of a week or two following administration of the vaccine during which the babies and rats were more susceptible to invasive Hib infections [Sood *et al.* (1988), Daum *et al.* (1989), Hiner and Frasch (1988)].

Granoff *et al.* (1986) analysed 228 reports of invasive disease due to Hib in vaccinated children submitted to the FDA administration between May 1985 and September 1987. Over 90% of these children were more than 24 months of age, when the vaccine is supposed to be somewhat effective A high proportion of cases were reported to have occurred within the first two months after vaccination, with 10 cases occurring within 72 hours of vaccine injections. Vaccination did not alter the expected frequencies of the different Hib diseases.

Postvaccination susceptibility to invasive Hib disease was also confirmed by Sood *et al.* (1988) in rats. The authors passively immunised infant rat pups with an immune globulin preparation, vaccinated them with a wide range of doses of *H. influenzæ* type b capsular polysaccharide vaccine and challenged them with *H. influenzæ* type b intraperitoneally. Bacteriæmia occurred in 89% of pups compared with an incidence of 17% in protected unvaccinated pups. In protected, vaccinated pups the rate of bacteriæmia resembled that in unprotected unvaccinated control pups and did not vary with the dose of vaccine administered.

They confirmed the findings of Wright (1901) who established the existence of a "negative" phase lasting a few weeks to several months after vaccination with typhoid vaccines .

Daum *et al.* (1989) confirmed that a decrease in serum antibody in the three weeks after administration occurs in most children and adults injected with any of the two vaccines. The lowest point occurred on days 1 to 3.

Another study [Granoff *et al.* (1986)] deals with 55 cases of invasive Hib diseases occurring in children at least three weeks after vaccination. Meningitis developed in 39 children of whom 3 died and 6 had neurologic after-effects. The level of antibody to Hib in convalescent-phase serum from 31 of the vaccinated children who had Hib disease was significantly lower than that in the serum from 25 patients of similar age (range 17 to 47 months) with the disease who had never received the Hib vaccine.

All studies of efficacy of Hib vaccines admit that the vaccine is ineffective in children younger than 18 months.

Norway conceded that the protective effect of another vaccine, outer membrane meningococcal b, was insufficient to justify a public vaccination programme [Bjune *et al.* (1991)].

Ward *et al.* (1990) trialled PRP-D vaccine in 2102 Alaskan native children and found there was no "significant" protective efficacy with the vaccine. There was a lower incidence of Hib infections in babies not participating in the study and the "vaccine" group, very similar to the rate in the "placebo" group. There were more cases of aseptic meningitis and sudden infant death in the "vaccine" group than among the "controls" (the authors did not tabulate or give exact figures).

These results must of necessity raise questions about the relevance of the original Finnish studies, especially since the same (negative) results were obtained when the same vaccine batch was given to Finnish babies and infants in Albany, New York [Ward *et al.* (1988)].

Decker *et al.* (1992) performed a double-blind, randomised trial to compare the immunogenicity and reactogenicity of four conjugate Hib vaccines by injecting them into 2, 4 and 6 months old infants.

The four vaccines differed markedly in their ability to stimulate the production of antibodies (PRP, PRP-D, PRP-CRM {PRP conjugated with cross-reacting mutant diphtheria protein} and PRP-OMP).

Of these vaccines only PRP-OMP (polyribosylribitol phosphate conjugated with outer membrane protein of *Neisseria meningitidis*) produced what the authors considered a clinically pertinent elevation in antibody level after one or two injections in 75% of infants. However, even with this vaccine, the third injection did not elicit further significant elevation of antibody levels.

Nevertheless, the PRP-OMP vaccine was licensed for routine administration at 2, 4 and 12 months of age.

The levels of antibodies reported by these authors were lower than those reported in other studies. Decker *et al.* (1992) found this intriguing, but could not offer any explanation.

Perhaps the most soul-destroying experiment with *Hæmophilus influenzæ* b vaccine is the one conducted on some 5,000 Navajo Indian babies from July 1988 to August 1990, described in a paper by Santosham *et al.* (1991). 2,588 Navajo infants were given the Hib vaccine together with DPT and OPV (oral polio vaccine); 2,602 Navajo babies were given DPT and OPV with placebo (2 mg of lactose) injection. The first dose was given at 42 to 90 days of age and the second at 70 to 146 days. The mean follow-up time was 269 days in the "vaccine" group and 267 in the "placebo" group.

An independent monitoring committee was appointed to advise the investigators when to stop the study, either for reasons of safety or because the efficacy of the vaccine had been established. The committee met on 2nd August 1990 after 23 definite cases of *H. influenzæ* infection had occurred. It advised stopping the trial because of the difference in distribution of cases of *H. influenzæ* disease between the "vaccine" and "placebo" groups. On two previous occasions the committee broke the study codes assigned to participants who died.

The trial was terminated after 4,161 (80%) of participants received their second dose of "vaccine" (2,056) or "placebo" (2,105). 249 children of the "vaccine" and 250 of the "placebo" groups had

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received their first dose and were still eligible to receive their second dose when the trial was terminated.

**Incidence of invasive diseases:**

| "**vaccine**" group (2,056 babies) | "**placebo**" group (2,105 babies) |
|---|---|
| 1 case of Hib disease | 22 cases of Hib disease |
| 15 cases aseptic meningitis | 13 cases aseptic meningitis |
| 19 cases invasive pneumococcal disease | 13 cases invasive pneumococcal disease |
| **Total number: 35** | **Total number: 48** |
| Rate per 1,000: 17 | Rate per 1,000: 22.8 |
| 9 cases of seizures (4.38/1,000) | 7 cases of seizures (3.33/1,000) |
| 8 deaths (3.89/1,000) | 8 deaths (3.80/1,000) |
| 104 hospitalisations | 106 hospitalisations |
| 8 viral infections | 28 viral infections |
| 34 cases of conjunctivitis | 54 cases of conjunctivitis |

**Local reactions**

| | "**vaccine**" group | "**placebo**" group |
|---|---|---|
| reddness less than 2.54 cm | 48 babies | 18 babies |
| reddness more than 2.54 cm | 91 babies | 10 babies |

In summary, babies given DPT plus oral polio vaccines and Hib vaccine (the "vaccine" group) experienced 35 cases of invasive infections (rate of 17/1,000) while babies given DPT plus oral polio vaccines with placebo (the "placebo" group) experienced 48 cases (rate of 22.8/1,000).

Whether these invasive infections were associated with *Hæmophilus influenzæ* B or other bacterium is really irrelevant; the clinical effect was the same. I use the word "associated" rather than "caused", because it is quite clear that the reason for these babies having contracted the invasive disease is that their immune system was suppressed by administration of the vaccine which enabled a variety of this ubiquitous commensal bacteria to become virulent.

The "vaccine" group also had a virtually identical rate of deaths and convulsions as the "placebo" group and a slightly higher number of seizures than did the placebo group. The authors simply excluded these from the trial calculations, although the death rate was twice as high as the official US cot death rate. Both groups had the same rate of hospitalisation due to viral and/or other infections. The "vaccine" group had substantially more local reactions.

The above is one of the best examples of unacceptable handling of vaccine trials. Perhaps the worst aspect is the analysis of the 16 deaths: the committee considered them unrelated to the vaccine injections. The authors did not even tabulate the deaths to reveal the time interval between vaccine injections and these deaths.

Why have developed countries experienced such an increase of invasive infections in the past 40 years? According to Smith and Haynes (1972) a 399% increase in the incidence of invasive Hib infections was recorded from 1942-50 through 1951-59 to 1960-68. Similar trends were presented by Bjune *et al.* (1991). The best demonstrable common factor in this period is a documented push for mass vaccination. This explanation is especially plausible since the number of cases has not increased in babies below three months of age since 1942! [Bjune *et al.* (1991) and Smith and Haynes (1972)]. This clearly implicates DPT injections in the increase of Hib diseases.

An important insight into this question undoubtedly comes from the so-called Swedish trial of two Japanese acellular whooping cough (pertussis) vaccines [Storsaeter *et al.* (1988)]. Sweden discontinued pertussis vaccination in 1979 because of reports which demonstrated that the vaccines were neither effective nor safe.

According to Strom (1960,1967) the rate of side effects from the vaccine far exceeded the rate of side effects from the disease. As Strom pointed out, it was not because Sweden was experiencing more side effects, but because she had a better recording system.

Nevertheless, in 1986/87 Sweden trialled two Japanese acellular vaccines (compared with the so-called cellular pertussis vaccine, the acellular version is supposed to have fewer reactions) [Storsaeter *et al.* (1988) and Ad Hoc Group for study of pertussis vaccine (1988)].

The trial was abandoned without any recommendation to resume pertussis vaccination in Sweden because of insufficient efficacy of the two vaccines to prevent whooping cough and because of concern about an unacceptably high incidence of invasive infections in the vaccine recipients [Hinman and Orenstein (1990)]. Of 2,800 infants, 11 contracted invasive diseases and 4 died instead of the theoretically predicted one. No child died from invasive infections in the control group — over 900 babies who were injected with placebo.

Sweden withdrew the application for licensure of the acellular vaccine [Anonymous (1989)]:

*"The National Bacteriological Laboratory now withdraws the application for licensing of a Japanese pertussis vaccine after consultations with the Division of Drugs, Board of Health and Welfare. The vaccine was studied in a large clinical trial of acellular pertussis vaccines which was finished in the autumn of 1987. The Division of Drugs judges that the efficacy of the vaccine may be lower than that of whole-cell vaccines. The uncertainty about a possible association with deaths due to serious bacterial infections, which occurred among vaccinated children, has also contributed to the recommendation made by the Division of Drugs of comparative trials between acellular pertussis vaccines and well-known whole-cell vaccines".*

The invasive diseases peak in children between 6 and 11 months of age, which is after two or three DPT vaccines are normally given. All vaccines, including DPT, cause infections of increased severity. As early as 1901 Wright described the appearance after typhoid vaccine injections of a "negative" phase which lasted from three to five weeks or up to several months. During this "negative" phase the bactericidal power of the blood was significantly decreased. Craighead (1975) reported on disease accentuation after immunisation with inactivated viral or bacterial vaccines.

Japanese authors Kimura and Kuno-Sakai (1988) sprang to the defence of Japanese acellular pertussis vaccines. In their only graph they showed the incidence of invasive infections in various age groups (in 1975 the vaccination age in Japan was raised to two years).

The lower part of their figure shows a clear decline in the incidence of meningitis in 0 and 1 year olds after 1975, while the incidence of the same skyrocketed in 2 and 3 year olds, clearly reflecting the consequences of the shift in vaccination age to two years, coupled with an increased vaccination compliance. It is a well-known fact that Japanese parents' interest in vaccinating their babies (coupled with Japanese doctors' boycott of pertussis vaccine) fell dramatically after a spate of vaccine-related deaths through the seventies and early eighties [Kanai (1980)].

A recent article by Griffin, Taylor, Dougherty and Ray (1992) set out to demonstrate that there is no increase in risk of invasive bacterial infections following diphtheria-tetanus-pertussis vaccination. What they did not realise is that their raw data actually confirm the causal relationship between the two: there was clear clustering of cases of invasive bacterial infections (including those associated with *Hæmophilus influenzæ* b) within the first 7 days, between 8 and 14 days and 15 through 28 days, clearly following the critical days so prominently featuring in all other studies of vaccine reactions. The incidence peaked at 6 through 11 months of age, thus clearly reflecting the observed incidence in all countries carrying out mass DPT vaccination.

Nevertheless the authors, as have many others, concluded that their data provide reassurance that use of DPT vaccine is not followed by a large increase in risk of serious bacterial infections. Of 64,591 children followed up, 158 had invasive bacterial infection. According to the authors this represents a rate of 394 infections per 100,000 person years. This rate is higher only in Australian Aboriginal babies — 991 per 100,000 cases.

Recently, several reports appeared in the literature claiming that mass use of Hib vaccines was accompanied by a rapid fall and indeed disappearance of Hib meningitis in Finland [Jonsdottir *et al.* (1992)], Iceland [Booy *et al.* (1992)], England [Poolman *et al.* (1986)] and the United States [Peltola (1993)]. In Finland in 1986-87, primary vaccination consisted of three doses of PRP-D at 3, 4 and 6 months, but was offered to only 50% of infants. In 1988-89 two doses of

PRP-D or HbOC were given at 4 and 6 months to all infants. Since 1990, PRP-T alone has been given at ages 4 and 6 months. Since 1986 the primary immunisation has been followed by a booster dose at 14-18 months. The Hib vaccines were always given at separate sites at the same time as the routine immunisations: DPT vaccine at 3 or 4 months, with inactivated polio vaccine (IPV) at 6 months, and with measles-mumps-rubella vaccine at 14-18 months. Over 90% of babies were vaccinated.

The number of cases of Hib meningitis among children aged 0-4 years rose by 130% between 1950-54 and 1980-84 (the total number of children rose only by 24% between 1950-1980). The incidence of Hib meningitis among children 0-4 years old rose by 270% between 1946-50 and 1976-80. After the peak years 1985 and 1986 (27 and 30 cases respectively) the number of cases declined steadily: 22 cases in 1987, 9 in 1988, 4 in 1989 and 3 in 1990.

In 1991 no Hib meningitis was diagnosed in any of the three hospitals. The authors do not quote any cases of meningitis associated with bacteria other than Hib (*Neisseria meningitidis, Pneumococcus* etc.) or other clinical forms of invasive infections.

In contrast to the Finnish study, a similar study done in Iceland stated that, although since 1989 no case of Hib meningitis has been confirmed in Iceland, 2 children had Hib bacteræmia (beside 2 newborn babies); one in January 1990 aged 4 years (unvaccinated, meaning 3 DPT and polio, no Hib vaccines) and one in December 1991 (a 9 month old child who had received 3 doses of DPT and polio and PRP-D). Throat swabs of 178 children in day-care centres were tested in February and May, 1992: none carried Hib.

In England (Oxford region) at least 90% of 29,600 children who were offered Hib vaccine have been immunised in four districts. In the control districts (where only the routine DPT and P were administered) on past experience eleven cases of invasive Hib infections would have been expected. 12 were observed, 8 of which were meningitis. In districts where Hib vaccine was administered, 11 cases would have been expected among vaccinees, *"but none occurred. There were two cases, both in children who were not given*

*Hib vaccine*." However, the authors do not explain why the fall in incidence also affected unimmunised children. Based on the above estimates of an expected 11 cases of invasive Hib infections, a greater number of cases than the two experienced were expected in unimmunised children [Booy *et al.* (1992)].

All these authors credited Hib vaccines with the disappearance of Hib meningitis. However, some important clues to this observed phenomenon come from two publications. One is by Santosham *et al.* (1991) who described the results of a trial of Hib OMP vaccine on Navajo Indians in which only 1 case of Hib meningitis occurred in the "vaccine" groups and 22 in the "placebo group. However, other invasive infections associated with other bacteria occurred in both groups [35 in the "vaccine" group and 45 in the "placebo" group (for more details see above)].

The second publication possibly shedding light on this question is by Poolman *et al.* (1986) describing the shift in meningococcal serogroup b disease in north-west Europe. Examination of the trends of meningococcal infection in Norway, the Netherlands, Iceland, Faroe Islands, Denmark, England and Wales has shown that *Neisseria meningitidis* B:2b:P1.2w and B:2a:P1.2 phenotypes were associated with peak infection in the Netherlands in 1966, in Iceland in 1976-77, and in England and Wales in 1973-75.

These strains were present in all six countries in the decade 1970-80 but their prevalence is now practically negligible. Instead, the prevalence of the B:15:P1.16 phenotype has risen. In the Faroe Islands and Norway this prevalence has been followed by rises in the incidence of meningococcal disease. The same may be happening in England and Wales and probably in Finland, Iceland and elsewhere and has quite likely nothing to do with Hib vaccination.

*Hæmophilus influenzæ* type b strains of outer membrane sub-types 1 and 1c are associated with different types of invasive disease. Takala *et al.* (1987) found that of 275 consecutive Hib strains isolated from children with invasive disease in Finland in 1985-86, 74% were of the common European outer membrane protein (OMP) subtype 1 and 22% were of OMP subtype 1c, which is usually rare.

Strains of subtype 1c were associated with more meningitis and less epiglottitis than were strains of subtype 1. Also, children with disease associated with strains of subtype 1c were younger than those with diseases associated with strains of subtype 1.

Has the problem of invasive infections of *Hæmophilus influenzæ* been resolved by mass vaccination? As Peltola (1993) says in her article published in the Lancet: not at all. The number of cases of invasive infections (including meningitis) has not diminished, but instead of being associated with capsular Hib, they are now associated with non-capsular Hib in vaccinated children.

Even more interesting is the article by Michaels and Ali (1993) in which they demonstrate a striking decline in the incidence of *Hæmophilus mengitidis* type b cannot be attributed entirely to immunisation. In fact, their figure 1 shows a peak incidence in 1976-77 which was followed by a steady and rapid decline which saw hospital admissions drop to less than half of the peak level **before** the vaccine was even licensed. The precipitous decline continued between 1985 (the year the vaccines were licensed) and 1990, especially in the age group which was not given the vaccine at all.

These observations indicate the futility of attempting to stop various *Hæmophilus influenzæ* diseases with vaccines based on different strains of the bacterium.

The documented contamination of vaccines by animal viruses is a chapter of its own. The recent article in the March 1992 issue of The Lancet warns about the contaminants in the polio vaccine which are linked to AIDS, leukæmia and cancer [Kyle (1992)].

It has been well documented that injections of foreign proteins, including those in vaccines, do not immunise, rather they sensitise. Instead of protecting against infectious diseases, they increase the recipient's susceptibility to infectious diseases. Moreover, vaccines modify the immunologic response and cause a great variety of autoimmune diseases. This is important to recognise, since many researchers consider infections occurring even shortly after vaccination to be coincidental (that is, completely unrelated to the injection of the foreign antigens).

The time has come to reveal this to the public, and especially to parents of small children. In their efforts to do their absolute best for their children, parents are emotionally vulnerable and an easy target for those selling vaccines.

An extensive study of medical literature reveals that there is no evidence whatsoever of the ability of vaccines to prevent any diseases. To the contrary, there is a great wealth of evidence (direct and indirect) that they cause serious side-effects.

The time has come to press for the removal of PRP and PRP-D (and other Hib) vaccines from use. All are demonstrably ineffective in preventing invasive infections. Australia should seriously consider following the Swedish example and stop vaccinating against pertussis. The whole-cell vaccine was rejected by Sweden in 1979 because of concerns about lack of efficacy to prevent whooping cough and because of serious side-effects including those of a cerebral nature [Storsaeter *et al.* (1988) and Strom (1960)]. The acellular pertussis vaccine was rejected for similar reasons [Anonymous (1989)].

One of the most worrying aspects of the effect of vaccines is that they accentuate susceptibility to a variety of infections, including the invasive types. The Swedish governmental bodies equivalent to our Health Department recognised this quite clearly and acted upon it. The less than reasonable US attitude to continue mandatory vaccination with ineffective and dangerous vaccines in the face of all the evidence [Mortimer (1988)] should not deter others from the correct decisions.

Ultimately, this is an issue dealing with infants' lives and health.

## References.


1. Parke, J.C., Schneerson, R., Robbins, J.B., *et al.*, 1977.
Interim report of a controlled field trial of immunization with capsular polysaccharides of *Hæmophilus* type b and group C *Neisseria meningitidis* in Mecklenburg County, North Carolina.
J Infect Dis; **136**: S51-S56.
2. Peltola, H., Kayhty, H., Sivonen, A., and P. Helena Makela, 1977.
*Hæmophilus influenzæ* type b capsular polysaccharide vaccine in children: a double-blind field study of 100,000 vaccinees 3 months and 5 years of age in Finland.
Pediatrics; **60**; 730-737.
3. Milstien, J.B., Gross, T.P. and Kuritsky, J.N., 1987.
Adverse reactions reported following receipt of *Hæmophilus influenzæ* type b vaccine: an analysis after 1 year of marketing.
Pediatrics; **80(2)**: 270-274.
4. Eskola, J., Peltola, H., Takala, A. K., *et al.* 1987.
Efficacy of *Hæmophilus influenzæ* type b polysaccharide - Diphtheria - toxoid conjugate vaccine in infancy.
N Engl J Med; **317 (12)**: 717-722.
5. Black, S.B., Shinefield, H.R., Hiat, R.A., Fireman, B.H., 1988.
Efficacy of *Hæmophilus influenzæ* type b capsular polysaccharide vaccine.
Ped Inf Dis; **7**: 149-156.
6. Shapiro, E.D., Murphy, T.V., Wald, E.R. *et al.*, 1988.
The protective efficacy of *Hæmophilus* b polysaccharide vaccine.
J Am Med Ass; **260**: 1419-1422.
7. Harrison, L.H., Broome, C.V., Hightower, A.W., *et al.*, 1988.
A day care-based study of the efficacy of *Hæmophilus* b polysaccharide vaccine.
J Am Med Ass; **260**: 1413-1418.
8. Harrison, L.H., Broome, C.V., Hightower, A.W., 1989.
*Hæmophilus influenzæ* type b polysaccharide vaccine: an efficacy study.
Pediatrics; **84(2)**: 255-261.
9. Osterholm, M.T., Rambeck, J.H., White K.E., *et al.*, 1988.
Lack of efficacy of *Hæmophilus* b polysaccharide vaccine in Minnesota.
J Am Med Ass; **260 (10)**: 1423-1428.
10. Ward, J., Brenneman, G., Letson, G.W., and Hayward, W.L., 1990.
Efficacy of a *Hæmophilus influenzæ* type b conjugate vaccine in Alaska native infants.
N Engl J Med; **323(2)**: 1393-1401.
11. Sood, S.K., Schreiber, J.R., Siber, G.R., *et al.*, 1988.
Postvaccination susceptibility to invasive *Hæmophilus influenzæ* type b disease in infant rats.
J Pediatr.; **113**: 814-819.

12. Daum, R.S., Sood, S.K., Osterholm, M.T. *et al.*, 1989.
Decline in serum antibody to the capsule of *Hæmophilus influenzæ* type b in the immediate postimmunization period.
J Pediatr.; **114**: 742-747.
13. Hiner, E.E., and Frasch, C.E., 1988.
Spectrum of disease due to *Hæmophilus influenzæ* type b occurring in vaccinated children.
J Infect Dis; **158(2)**: 343-348.
14. Granoff, D.M., Schakelford, P.G., Suarez, B.K., *et al.* 1986.
*Hæmophilus influenzæ* type b disease in children vaccinated with type b polysaccharide vaccine.
N Engl J Med; **315**: 1584-1590.
15. Bjune, G., Holby, E.A., Gronnesby, J.K., *et al.*, 1991.
Effect of outer membrane vesicle vaccine against group b meningococcal disease in Norway.
Lancet; **338 (8775)**: 1093-1096.
16. Smith E.W.S., and Haynes, R.E., 1972.
Changing incidence of *Hæmophilus influenzæ* meningitis.
Pediatrics; **50(5)**: 723-727.
17. Decker, M.D., Edwards, Kathryn M., Bradley, R., and Palmer, P. 1992.
Comparative trial in infants of four conjugate *Hæmophilus influenzæ* b vaccines.
J Pediatrics; **120**: 184-9.
18. Ward, J. I., Brenneman, G., Lepow, M., Lum, M., Burkhart, K., and Chiu, C.Y., 1988.
*Hæmophilus influenzæ* type b anticapsular antibody responses to PRP-pertussis and PRP-D vaccines in Alaska Native infants.
J Infect Dis; **158**: 719-723.
19. Santosham, M., Wolfe, M., Reid, R., *et al.*, 1991.
The efficacy in Navajo Infants of a conjugate vaccine consisting of *Hæmophilus influenzæ* type b polysaccharide and *Neissereia meningitidis* outer-membrane protein complex.
N Engl J Med; **324(25)**: 1767-1772.
20. Storsaeter, J., Olin, P., Renemar, B., *et al.*, 1988.
Mortality and Morbidity from invasive bacterial infections during a clinical trial of acellular pertussis vaccine in Sweden.
Pediatr Infect Dis J; **7(9)**: 637-645.
21. Strom, J., 1960.
Is universal vaccination against pertussis always justified?
Brit Med J; 22 October: 1184-1186.
22. Strom, J., 1967.
Further experience of reactions, especially of a cerebral nature, in conjunction with triple vaccination: a study based on vaccination in Sweden 1959-65.
Br Med J; 11 Nov.: 320-323.

23. Ad Hoc Group for the study of pertussis vaccines. 1988.
Placebo-controlled trial of two acellular pertussis vaccines in Sweden — protective efficacy and adverse effects.
Lancet; 30 April: 955-960.

24. Hinman A.R., and Orenstein, W.A., 1990.
Immunization practices in developed countries.
Lancet; **335**: 707- 710.

25. Anonymous, 1989.
License application for pertussis vaccine withdrawn in Sweden.
Lancet; 14 Jan: 114.

26. Wright, A.E., 1901.
On the changes effected by antityphoid inoculation in the bactericidal power of the blood; with remarks on the probable significance of these changes.
Lancet 14 Sept, 1901: 715-723.

27. Craighead, J.E., 1975.
Report of a workshop: disease accentuation after immunization with inactivated microbial vaccines.
J Infect Dis; **1312(6)**: 749-754.

28. Kimura, M. and Kuno-Sakai, H., 1988.
Acellular pertussis vaccines and fatal infections.
Lancet, 16 April: 881-882.

29. Kanai, K., 1980.
Japan's experience in pertussis epidemiology and vaccination in the past thirty years.
Japan J Med Sci Biol; **33**: 107-143.

30. Griffin, M., Taylor, J.A., Daugherty, J. R., and Ray, W.A. 1992.
No increased risk for invasive bacterial infections found following diphtheria-tetanus-pertussis immunization.
Pediatrics; **89 (4)**: 640-642.

31. Hanna, J. 1990
The epidemiology and prevention of *Hæmophilus influenzæ* infections in Australian Aboriginal children.
Med J Aust;**152**: 234 -240

32. Kyle, W.S., 1992.
Simian retroviruses, poliovaccine and origin of AIDS.
Lancet; **339**: 600-601.

33. Mortimer, E.A., 1988
Efficacy of *Hæmophilus* b polysaccharide vaccine: an enigma.
J Am Med Ass; **260 (10)**: 1454-1455.

34. Peltola, H., Kilpi, T., and Anttila, M., 1992.
Rapid disappearance of *Hæmophilus influenzæ* type b meningitis after routine childhood immunisation with conjugate vaccines.
Lancet; **340**: 592-594.

35. Jonsdottir, K.E., Steingrimsson, O., and Olafsson, O., 1992.
Immunisation of infants in Iceland against *Hæmophilus influenzæ* type b.
Lancet; **340**: 252-253.

36. Booy, R., Moxon, E.R., MacFarlane, J.A., Mayon-White, R.T., and Slack, M.P.E., 1992;
Efficacy of *Hæmophilus influenzæ* type b conjugate vaccine in Oxford region.
Lancet; **340**: 847.
37. Poolman, J.R.T., Jonsdottir, K., Jones, D.M., Lind, I., Froholm, L.O., and Zanen, H.C., 1986.
Meningococcal serotypes and serogroup B disease in north-west Europe.
Lancet, 6 Sep: 555-557.
38. Takala, A.K., Eskola, J., Bol, P., Alphen, L., van Palmgren, J., and Makela, P.H., 1987.
*Hæmophilus influenzæ* type b strains of outer membrane subtypes 1 and 1c cause different types of invasive disease.
Lancet; 19 Sept: 647-650.
39. Peltola, H., 1993.
*H. influenzæ* in the post-vaccination era.
Lancet; **341**: 864-865.
40. Michaels, R.H. and Ali, O., 1993
A decline in *Hæmophilus influenzae* type B meningitis.
J. Pediatrics; **122**: 407-409.

# 7

# POLIO VACCINES: leukæmia, cancer, simian retroviruses and AIDS

Everybody remembers, or has heard of, the 1949-50 epidemic of polio in Australia and in many other countries. It is commonly quoted to scare parents into vaccinating their children. Even those parents who decide not to vaccinate against whooping cough, diphtheria, tetanus and measles usually make a concession and vaccinate against polio. After all, who would want their child exposed to the risk of paralysis?

However, only very few know that that famous 1950s polio epidemic has a quite different, and much more sinister, background.

In 1950, Dr McCloskey published an article in The Lancet "The relation of prophylactic inoculations to the onset of poliomyelitis."

He wrote in the introduction that *"Early in the epidemic, attention was directed to a few patients who had been given an injection of pertussis vaccine, or of a mixture of diphtheria toxoid and pertussis vaccine, shortly before the onset of their symptoms.*

*"The parents of these children were naturally inclined to blame the inoculations for the development of the disease, though their medical attendants either dismissed the probability of any causal relationship, or else considered the effect to be due to a radiculitis caused by the vaccine... Considerable evidence, however, will be presented to show that such an association has existed in this epidemic."*

The British Medical Journal 1950 (1 July) published an article by Hill and Knowelden on Inoculation and Poliomyelitis. The authors wrote, among other things, that during the progress of the diphtheria

immunisation campaign, begun in 1942, there have been occasional and sporadic cases of paralysis reported following the injection of an antigen: *"This paralysis has sometimes been limited to the limb in which the injection was made; sometimes it had involved other limbs as well... In most cases a diagnosis of poliomyelitis was made."*

The same issue of the British Medical Journal published another article, by MacCallum, on clinical poliomyelitis in association with peripheral inoculation of prophylactics. The author wrote that: *"Stools have been obtained less than 21 days after the onset of the illness in a number of children included in some of the recent observations, and the poliomyelitis virus has been isolated by monkey inoculation from each of the five children whose stools have so far been tested. The rhesus monkeys used all developed a disease similar to experimental poliomyelitis, and lesions pathognomonic of the disease were found in the brain and (spinal) cord at necropsy."*

The affected children were given smallpox, diphtheria-pertussis and/or tetanus injections prior to the onset of poliomyelitis.

The same journal (BMJ 29 July) published an article by Banks and Beale (1950) titled "Poliomyelitis and immunisation against whooping-cough and diphtheria" reporting on a number of paralytic polio cases which occurred in 1947, 1948 and 1949, following pertussis and diphtheria inoculations, with a preponderance of upper-arm paralysis (most inoculations in London being done in the arm, rather than in the leg). The interval between the last injection and the onset of paralysis in the majority of cases was between nine and 14 days.

Leake (1950), in his letter to the Editor of the Journal of the American Medical Association, summarised the published data confirming the occurrence of paralysis after a variety of vaccine injections including pertussis, diphtheria and typhoid-paratyphoid.

Perhaps not surprisingly, an American study of similar associations between injections of vaccines and paralytic polio by Bell (1950) [quoted in Leake(1950)] discounted any suggestion of similar association between diphtheria and pertussis inoculations. However, the author nevertheless cautioned against administration

of any inoculations during the polio seasons, as well as against strenuous physical activity, and unaccustomed sunning and chilling.

At least one American study, by Anderson and Skaar (1950), concluded that *"In poliomyelitis patients who have received some antigen during the month prior to onset there is a high degree of correlation between site of paralysis and site of injection. Such cases tend to show a different distribution of paralysis and a more severe paralysis than do comparable children immunised two to six months previously, immunised in previous years or never immunised."*

Martin (1950) collected data on 17 cases of paralysis after diphtheria vaccine injections in which paralysis occurred in one limb within 28 days of the injection. In almost all cases the diagnosis of poliomyelitis was made. It is very unlikely that the relationship would have been totally coincidental, especially considering the great number of papers reporting the same observations.

Geffen (1950) reported the occurrence of poliomyelitis in recently inoculated children in the Metropolitan Borough of St Pancras and London. Paralysis tended to occur in the injected limb.

Wyatt (1981) dealt with the repeated observations of provocation poliomyelitis after a number of procedures.

He reviewed data from literature starting with Hans Kern's report published in 1914 on the occurrence of poliomyelitis in institutionalised children in Germany. Of 22 children suffering congenital syphilis, five developed polio after being treated with Neosalvarsan, Salvarsan and other drugs.

The period from injections of Neosalvarsan to illness was from five to 21 days, and from six to 31, and two to 14 days, after an injection of Hg-salicyl-Kur in individual cases of paralysis.

A further seven cases were reported in Germany after similar treatment between 1921 and 1926. Some authors reported provocation polio after injections of alum-precipitated diphtheria and pertussis vaccine.

Many other similar cases were reported. Likewise, many authors reported polio after tonsillectomies.

**Increased risk of contracting polio following tonsillectomy**

Paffenbarger (1957) presented convincing evidence for a statistical association between previous tonsillectomy and increased risk of contracting poliomyelitis, specifically the bulbar form. Although the timing of onset of symptoms varies, the association is most prominent within one month after surgery.

The reported observations were based on: 1. an epidemic of type 1 poliomyelitis in Olmsted County, Minnesota, in 1952, when 215 cases developed among 49,000 residents, an attack rate of 438 per 100,000; 2. the reported poliomyelitis outbreak in metropolitan Washington DC, during an endemic year, 1954, when 155 paralytic cases, representing infections with all 3 virus types occured among nearly 1,500,000 residents; 3. 113 cases from the St Louis encephalitic epidemic in Hildago County, Texas, in 1954, when over 500 cases developed among 160,000 residents, an attack rate exceeding 310 per 100,000.

Over half of those poliomyelitis patients whose illness began with bulbar symptoms had undergone tonsil removal, as compared with remarkably uniform proportions approximately fourth in the control groups which did not undergo tonsilectomy.

Lambert (1936) described a campaign to treat yaws in Samoa with neoarsphenamine administered by two or more injections. In 1932 some 36,000 persons were treated this way. The first case of paralysis occurred a week after the second yaws injection. All patients first seen had paralysis in the lower limbs and all had double buttock injections. Paralysis occurred in all 37 villages where the yaws campaign was conducted. Although Lambert had a very convincing account of provocation poliomyelitis, he rejected the connection despite all natives claiming the connection.

Wyatt (1981) also commented on a connection between small pox vaccination and paralysis. These observations were ignored because there was no obvious connection between the two and because a *"fuss over these observations would have disturbed the smallpox vaccination program."* The same author commented on the Brodie-Park and Kolmer polio vaccine accidents in 1935. He wrote

that *"It has always been assumed that the cases of poliomyelitis associated with the Kolmer and Brodie-Park vaccines were caused by virus incoculation as in the Cutter incident of 1955."*

In another paper Wyatt *et al.* (1992) examined the effect of prior injections on the pattern and severity of paralytic poliomyelitis by a retrospective analysis of cases recorded in an outpatient clinic in South India. Of 262 children with acute polio, 176 had received unnecessary injections less than 48 hours before paralysis and 12 had received diphtheria-pertussis-tetanus provocative injections. There was a considerable association between the injected arm and localisation of paralysis. After injections there was greater likelihood of death or lack of recovery of muscle strength.

In a more recent paper, Wyatt (1993) called for campaign to banish all unnecessary injections.

### Polio — the Disease and Immunity to It

The above reports of polio, undoubtedly triggered by injections of a variety of antigens and other foreign substances, are of great interest, especially after years of experience with mass immunisations. First of all, they give credence to opinions expressed several decades ago by such authors as Jungeblut and Engle(1932), who believed that polio may, after all,be of endogenous origin.

These authors realised that the occurrence of infection is obviously dependent upon a delicate balance between the virulence of the invading micro-organism and the receptivity of the host. They wrote that with *"high virulence of the ætiologic agent, differences in individual predisposition of the host will frequently be obscured; a low innate susceptibility to a disease, on the other hand, may virtually obliterate the infectious character of its incitant. A rational analysis of resistance or susceptibility must, therefore, allocate in each instance proportionally a greater or lesser importance to specific immunologic and nonspecific physiologic factors.*

*The lack of natural resistance frequently is synchronised with fluctuations in the regulatory influence of non-specific physiologic elements, such as age, sex, heredity, diet and endocrine activities."*

Polio represents an extreme example of an infectious disease in which lack of susceptibility to the disease is so widespread that only under particular environmental circumstances does it ever assume epidemic proportions. According to these authors, support for their conclusions comes from innumerable instances of epidemics, for instance the epidemic in Brooklyn in which, in 469 cases of 500, it was impossible to establish contact with a previous case (*op. cit.*).

Jungeblut and Engle (1932) recognised a strong seasonality of polio incidence. They also stressed that because clinical cases of polio are numerically far too few they cannot account for the universal natural immunty of the adult population.

However, Paul *et al.* (1932) emphasised the importance of minor illnesses occurring during an epidemic ("Heine-Medin" disease of uncharacteristic sporadic colds or diarrhoeas occurring in summer).

The formation of antibodies against certain infectious diseases, and some non-infectious agents, is essentially an expression of an endogenous, hereditarily fixed maturation process, rather than the result of exogenous influences, which reaches its climax around puberty. Paul *et al.* (1932) talked about serologic ripening and the age-dependent development of agglutinins. They reasoned that a gradually increasing number of negative reactions to the Schick and Dick tests through adolescence occurs also with dysentery toxins. Under extreme climatic conditions, such as occur in the tropics or subtropics and in arctic regions, negative reactions to the Schick and Dick tests may develop with puberty while clinical diphtheria and scarlet fever occur only very sporadically.

The viricidal power of blood develops with maturity both in monkeys and man, despite a low incidence of clinical polio (the 'hundred monkeys' syndrome?).

Jungeblut and Engle (1932) also discussed the differences in polio-virus neutralising power of serum of different blood groups from normal human adults. One half of group A sera were capable of neutralisation when tested in the usual quantities while 80% of group B sera possessed this property. Group O sera behaved similarly to group A, with one half neutralising. Thus the genetic factor was

infinitely more important than any hypothetical contact factor in influencing neutralisation capacity of a person's serum.

A similar relationship was ascertained between blood group and neutralising power in serum of convalescent patients. Group B sera neutralise at many times higher dilution than do group A sera.

In persons of blood group A, a constitutional inability to form diphtheria antitoxin after recovery from that disease has been described by Nowak (1931) [quoted by Jungeblut and Engle (1932)].

Jungeblut and Engle (1932) quoted the work of Draper (1932) who methodically observed an endocrine deficiency in children stricken with poliomyelitis. It is known that after puberty the incidence of polio declines earlier and more abruptly in females than in males. Pregnant women show remarkable resistance to polio; babies receive certain circulating hormones, charactertistic of adult age, with the maternal blood . However, Anderson et al., (1952) stated that poliomyelitis has frequently been recorded during pregnancy, indicating increased susceptibility to recognisable infection.

A premenstrual drop in bactericidal power of blood has been described by Geller (1930) [quoted by Jungeblut and Engle (1932)].

Previously, Aycock (1926) acknowledged an apparent discrepancy between the theory of spread through direct person-to-person contact and observations in the field and epidemiologic incidence of scant direct contact. There is a great number of comparatively mild forms of polio which escape recognition, and a relatively large proportion of healthy carriers. Taken together, these are largely responsible for the spread of the virus.

Support for the endogenous origin of polio comes also from such examples as the incidence of three paralytic cases in a camp of 60 boys or simultaneous occurrence of cases within the same families.

Hudson *et al.* (1936) discussed at some length the factors of resistance and immunity in experimental poliomyelitis. They wrote that efforts to induce immunity in humans have been based on *"classic principles effective in other infectious states, and insufficient attention has been paid to the basic difficulties and to factors of the host's resistance to this particular virus."*

They concluded that the upper respiratory tract is the portal of entry of the polio virus and at the same time a certain degree of resistance is manifested by the nasopharyngeal mucosa. The intestinal mucosa was an effective barrier to infection by virus administered directly into isolated intestinal loops.

The spleen played a definite role in inducing resistance to the virus. Neutralising antibodies were formed in monkeys "vaccinated" with certain preparations, but their presence was not an indication of effective protection of the animal to intranasal virus. Natural or artificially induced menstruation and physiologic maturation did not lead to a demonstrable viricidal property of the blood.

They also wrote that, after entering the olfactory nasal tract, the virus migrates intracellularly through the central nervous system to the "loci of predilection" in the cord, and sensitises the nervous tissues in some way so that it is resistant to re-exposure to the virus. The neutralising antibodies in the natural conditions are thus an indication of specific sensitisation by extra-neural stimulation after nerve cell migration of the virus. Antibodies induced artificially are not necessarily a measure of nerve tissue resistance.

The authors warned that artificial immunisation of humans, either active or passive, should take into account the distinctness of the central nervous system in the pathogenesis of polio and the significance of certain factors of resistance imposed as a barrier to the virus between the vascular and central nervous systems.

Sub-lethal doses were made fatally infective through damage to the cerebral cortex by starch injections. This phenomenon may provide a theoretical explanation for the occurrence of poliomyelitis after injections as discused above.

### Epidemiology of poliomyelitis

The epidemiology of poliomyelitis is equally as interesting as the questions of immunity to it. Nathanson and Martin (1979) considered many of the salient epidemiological features of polio enigmatic. Apparently, polio appeared as an epidemic disease some 100 years ago in northern Europe and the United States. It is basically a disease

of infancy, and practically all individuals experience infection at some time during their lives. There was a steady decrease in the incidence of polio while the age distribution rose dramatically in both areas. While there was an upward trend in age, there was no upward trend in incidence. After decades of constant incidence there was only a dramatic upsurge of incidence in 1945-1954. This may be partly accounted for by the reporting of non-paralytic cases.

However, there is a more plausible explanation, namely intensified vaccination with a variety of vaccines leading to provocation poliomyelitis as documented above.

Vaccination trials with poliovirus vaccines, based on monkey neural tissues, were carried out in the 1930s. The trials were condemned because of marked side effects and fatal cases associated with administration of these vaccines. However, the work resumed some 20 years later and resulted in the production of the Salk polyvalent vaccine. A large trial with the Salk vaccine was organised by the poliomyelitis vaccine evaluation center of the University of Michigan, Ann Arbor, Michigan (Francis *et al.* 1955).

During the trial, one syringe was used for five children. Concerns about the possibility of transmitting infectious diseases or serum hepatitis because of this led several areas to organise their own clinics using individual syringes for each child. Some doctors objected to the fact that the vaccines contained dead monkey kidney tissue. However, nobody did anything about it.

In this trial, over 1,000,000 children were injected with three doses of the Salk vaccine between 1 May and 1 December 1954. One hundred and twenty nine cases of "presumed" poliomyelitis occurred during the trial period, including three weeks after the third injection: 90 cases in the vaccinated group and 39 cases in the placebo controls. However, because the 90 cases occurred during the vaccination period between May 1 and two weeks after the third injection, they were excluded from calculations.

Of 749,236 placebo control children, 428 developed poliomyelitis; of 1,080,680 vaccinated children, 585 developed polio. The corrected figures are 428 plus 39 (467) and 585 plus 90 (675).

The authors of the report stated that there were 57/100,000 cases of polio among the vaccinated and 54/100,000 among the unvaccinated. So the rate of polio was slightly higher in the vaccinated than in the unvaccinated. When the complete figures are calculated (including the 90 which were excluded) the rate of cases of polio in the vaccinated children is even higher.

Based on their own figures, the trial showed a total failure of the Salk polio vaccine to protect against poliomyelitis.

Despite this, in April 1955 six pharmaceutical companies obtained licences for production of inactivated poliovirus vaccines. Within 14 days of the release of the vaccine a large number of babies (94), their parents (126) and other contacts (40) were reported to have contracted paralytic polio from the vaccine (the "Cutter incident") encompassing a period from 25 April to the end of June 1955 and described in detail by Nathanson and Langmuir (1963a, b c).

Vaccination was halted for two weeks but resumed again in May. Polio epidemics continued occurring in the United States despite a high proportion of the population being vaccinated. During a 1959 epidemic in Massachusetts, 77.5% of the paralytic cases had received three or more doses of inactivated vaccine. Similar disappointing results were reported from many other countries where the Salk vaccine was used on a large scale: Czechoslovakia, Hungary and Israel. The quality of the vaccines had been very uneven and generally low. Not only were the vaccines ineffective, they continued causing paralytic poliomyelitis at the time when there were no epidemics with the wild virus.

### Contamination of Polio vaccines by Animal Tissue

A new menace appeared when polio vaccines proved to be contaminated with a great number of animal (simian) retroviruses, called SV1 to SV40. Of these, SV40 was the most researched. Rustigian *et al.* (1955) established that monkeys carry a great number of viruses such as B virus, foamy agent, measles-like virus, hæm-adsorption viruses, LCM virus, arboviruses and a great variety of miscellaneous viral agents.

Hull and Minner (1955) and Hull *et al.* (1958) extensively studied viruses found in normal monkey kidney cell cultures and called them simian viruses or SV. They were classified into four groups based on the kind of cytopathic changes induced in monkey kidney cell cultures infected with these viral agents. Twenty eight of these viruses were grouped into serological types and an additional 24 unidentified viruses were recorded.

Malherbe and Harwin (1957) distinguished seven different types among the simian agents or SA viruses recovered from vervet kidney.

According to Sweet and Hilleman (1960) a new simian virus has been encountered repeatedly. This virus was unique among simian viruses because it did not cause detectable cytopathic effects in the rhesus or cynomolgus kidney cell culture from which it was derived, but instead grew and caused marked cytopathic changes in cell cultures of other species. The virus was recognised by McClelland in the course of safety testing of vaccines and was subsequently called vacuolating SV40 virus.

At about the same time another virus was isolated from laboratory chimpanzees with coryza and was called respiratory syncytial (RS) virus (Morris *et al.* 1956). Soon it was established that a number of these viruses caused cold-like symptoms in adult human volunteers. Needless to say, they formed prominent contaminants in polio vaccines and were soon detected in children, causing respiratory tract infections in babies and small infants vaccinated with polio vaccines.

Parrot *et al.* (1961) published results of serologic studies over a 34-month period of children with bronchiolitis, pneumonia and minor respiratory diseases. Their data confirmed an ætiologic relationship between RS virus and respiratory tract illness, particularly *"relatively severe lower respiratory tract illness in children"*.

Chanock *et al.* (1961) recovered respiratory syncytial virus from 57% of young infants with bronchiolitis or pneumonia during a 5-month period. The virus was also recovered from older infants and children with pneumonia or bronchiolitis and from a significant proportion (12%) of young patients with a milder febrile respiratory

disease. The outbreaks lasted from three to five months. They established that RS virus represents a major pathogen during early life. Soon, these animal viruses spread and were recovered from cases of common cold in human adults [Hamparian *et al.* (1961)].

Melnick and Stinebaugh (1962) and many others confirmed that SV40 vacuolating virus was a "viable contaminant" of killed and live virus vaccines grown on rhesus and cynomolgus kidney cultures. They wrote that it appears that *"the virus has been injected as a live contaminant of formalinised polio vaccines and adeno-vaccines into hundreds of thousands, if not millions, of persons, and especially babies, and that it has been fed in active form together with live poliovaccine to groups equally as large"*.

The authors however emphasised that in spite of the large numbers of persons injected or fed the virus, not *"a single human illness has been attributed to this agent."*

That belief can no longer be sustained.

### Human Illness Associated with Simian Virus

ZuRhein and Chou (1965) found numerous spherical randomly distributed virus particles in tissues infected with any number of papova viruses (for more detail see below) including SV40 in human cerebral demyelinating disease. Demyelination resulted from the cytocidal effect of the virus on oligodendroglia.

Weiner *et al.* (1972) reported on isolation of virus related to SV40 from patients with human demyelinating disease: progressive multifocal leukoencephalopathy. Shah *et al.* (1972) tested four groups of donors for the presence of SV40 reacting antibodies. Vaccines have been required since 1961 to be produced free of SV40. However, SV40 neutralising antibodies were found in some tested babies born after 1964. The authors did not even consider that this may have shown that the vaccines are very much not free of the S40 virus – they were looking for sources of the infection in humans other than the vaccines.

Baguley and Glasgow (1973) reported on the incidence of subacute sclerosing panencephalitis (SSPE) which occurred from

1956 to 1966 in the northern half of the North Island of New Zealand. It was 100 times greater than expected. Mass vaccination of primary-school children with Salk vaccine from 1956 onwards was blamed for this major outbreak because the vaccine used *"is likely to have contained live SV40 virus"*. The authors concluded that *"the administration of Salk vaccine in New Zealand was related to the appearance of S.S.P.E."* and that *"The idea that an unusual reaction to measles infection is the sole cause of S.S.P.E. is not consistent with the observation in New Zealand"*.

In 1962 Eddy *et al.* identified oncogenic substance in rhesus monkey kidney cell culture as the simian SV40 virus. Both Eddy *et al.* (1962) and Girardi *et al.* (1961) confirmed that SV40 virus causes tumours in hamsters inoculated in the neonatal period with vacuolating virus SV40.

Rabson *et al.* (1962) demonstrated that oncogenicity of SV40 was not restricted to hamsters. Fraumeni *et al.* (1963) reported on the oncogenicity of SV40 in humans introduced into the body with injections of inactivated Salk poliomyelitis vaccine. They quote several authors who clearly demonstrated alterations of human tissue cultures infected with SV40.

Shein and Enders (1962) observed a reproducible "epitheloid transformation" characterised by abnormal growth pattern, greatly accelerated growth rate and chromosomal aberrations in human kidney tissue cultures. Koprowski *et al.* (1962) recorded similar transformation during SV40 infection of organ cultures of adult human skin and buccal musosa. Rabson *et al.* (1962) produced a strain of rapidly multiplying pleomorphic cells in human thyroid tissue *in vitro* with SV40 which grew rapidly, with large amount of virus persisting and demonstrated intranuclear virus in 5% of these cells. Eddy *et al.* (1962) demonstrated multiplication of SV40 in continuous line cultures of human cancer cells.

Melnick (1962) invented the term 'Papova' viruses for a group that includes "pa(pilloma)", "po(lyoma)" and "va(cuolating)" (SV40) viruses. These are all de-oxyribonucleic acid (DNA) viruses. Other similarities include a slow growth cycle with multiplication inside

the cell nucleus, an ability to cause chronic and latent infections in their host and a capacity to induce tumours in their natural and/or host species.

Although the authors played down these findings, they nonetheless quoted Innis (1968) who established that from 1955 to 1959 the leukæmia mortality rate per 100,000 rose from 3.5 to 3.8 for children 5-9 years old and from 2.2 to 2.5 for children 10-14 years old. The leukæmia mortality for those states using vaccine containing SV40 was generally greater than that for the states whose vaccine was free of SV40. Their figure 2, nevertheless, shows a major increase in deaths from cancers other than leukæmia in children under 12 months of age and 5-9 years of age, and an increase in leukæmia deaths in under one year, 5-9 years of age and in the 1-14 yearsage groups.

In 1973 Shah *et al.* confirmed a high prevalence of antibodies to BK virus, an SV40-related papova virus, in residents of Maryland. The prevalence of antibodies increased from 50% to 100% between the ages of three to 10-11 years and then declined to 67% in the age group 35 years or older. Shah *et al.* (1972) also confirmed that SV40 neutralising antibodies were found in 3.2% of tested sera (9 children) of Maryland children born during or after 1964. This was established despite the fact that vaccines were required to be free of SV40 since 1961. Indeed, in 1992 (Kyle) published in The Lancet an article proposing a link between contaminated polio vaccines in treatment by homosexual men of genital herpes, popular in the late seventies, and the simultaneous outbreak of AIDS in American homosexual men. Reverse transcriptase analyses of released vaccine have shown positive for such simian viruses up to 1985 and the author urges that a critical look should now be taken at all such vaccines and "*... the results should be made public*".

**Contaminated Poliovirus Vaccines and the Causation of AIDS**

By far the most competent and veritable summary of the dangers of the oral polio vaccine and the origin of AIDS is that published by Louis Pascal. He is the epitome of the independent scholar, a person without formal affiliation to a university or a research institution.

His paper, ***"What happens when science goes bad. The corruption of science and the origin of AIDS. A study of spontaneous generation"*** was finally published by the University of Wollongong as a Working Paper (No.9) in 1991, after a number of research journals rejected or simply ignored his submissions.

In his paper, Pascal demonstrated that AIDS originated in the Belgian Congo as a direct result of mass oral polio vaccination while the vaccine was conatminated with a simian immunodeficiency virus (SIV). The special strains of polio virus that had been carefully bred to lack neurovirulence, but that would nevertheless induce immunity, retained the ability to revert to their former neurovirulence by passage from one person to another. Also, and very importantly, the manufacturing procedure almost guaranteed contamination with foreign viruses which could not be killed without also killing the polio virus and ruining the vaccine.

In late 1957, in the eastern part of the Belgian Congo, and especially in the early part of 1958 in Ruanda-Urundi, the world's first mass vaccination campaign using live polio vaccine was conducted. A few months later, the same batch of the vaccine was used in Leopoldville, the capital of the Belgian Congo, 900 miles (almost 1500 km)west of the sites of the first vaccination campaign.

Almost immediately, contaminating viruses began emerging (in addition to the SV1-40 group referred to above). Indeed, Pascal quoted Hilary Koprowski, who manufactured the very batch of vaccine used in these two African campaigns, writing that "If indeed somebody were to poke his nose into the live virus vaccine, he might find a non-polio virus in all the preparations currently available."

He also added that this presented no real problem because people were exposed to many viruses every day in their food.

Pascal correctly pointed out that Koprowski was wrong on 3 counts.

Firstly, if vaccines could not be made free of contamination, they should be abandoned or the contaminants pronounced harmless. Koprowski was hardly a disinterested party, because in the first instance he would have had to renounce years of work.

Secondly, the contaminating viruses were monkey viruses and humans are not daily exposed to monkey viruses. Also the fact that these contaminated vaccines were fed to infants less than 30 days old (or even just 48 hours old), increases the danger, since their immature immune systems presented much less of an obstacle to a foreign virus than a mature immune system would have. (Infant animals are generally used in experiments for just this reason).

Thirdly, new viruses starting in "virgin" populations without prior immunological experience are often particularly virulent and contagious.

In 1985 the first of the simian immunodeficiency viruses was identified in rhesus monkeys, one of the three main species whose tissues were used to culture the polio vaccine. Since then, the SIVs have been found in the two other species — cynomolgus and African Green monkey — as well. All of these SIVs are closely related to each other and are the closest-known relatives of the HIV. Pascal quite correctly highlights *(op. cit.)* that the conclusion was immediately drawn that transfer from a monkey to a human of one of these viruses had caused AIDS. *"And how was it transmitted? Why, through a monkey bite, of course!"*

Pascal also raised ethical and moral questions in asking why neither the scientific community nor the reporters raised these questions much earlier to prevent the spread of the SIVs through vaccines.

Instead, both the World Health Organisation and the health authorities in the so-called developed countries are still pushing, and indeed intensifying, a general mass vaccination of human infants.

Billi Goldberg (1993) ascertained that in the late 1950s and early 1960s, poliovirus vaccines grown on diploid lines from human embryonic tissues (attenuated and killed) were acceptable only when grown in primary monkey kidney *"since such tissue was presumed to have no malignant properties."* A human diploid line was inoculated with the CHAT strain type 1 poliovirus cultured and attenuated in monkey kidney, a source of simian viral and retroviral contamination. Between August 1958 and April 1960, more than

75,000 children uder five years in Leopoldville (Belgian Congo) were given the vaccines prepared at the Wistar Institute. The vaccine was squirted into the back of the child's throat. As shown in other experiments [Morris et al. (1961)], *"Subclinical viral infection may be initiated by spraying of contaminated vaccines into the nasopharyngeal cavity if the vaccine enters the respiratory system."* And *"Retroviral infection of recipients of nebulised contaminated vaccine remains a possibility."*

Myers et al. (1992) asked whether as a "starting point for enquiry, HIV might simply be SIV adapting to a human host." "The notion is less far-fetched in 1992 than it was merely a few years ago" concluded Goldberg (1992).

Salk (1955) discussed considerations in the preparation and use of poliomyelitis virus vaccine. The motivation to write this article came from the "Cutter incident"(see below). In April 1955 the polio vaccine preparations caused " ... *subsequent development of paralytic poliomyelitis. 146 cases of paralytic polio developed in vaccinated children and their contacts within a short period of time. This demanded a very intensive re-examination of the theoretical and practical implications of vaccine preparation, testing, and use ...*

*"It has been realised always that the preparation of a safe poliomyelitis vaccine would, at the beginning at least, require adherence to detail such as is not demanded for the preparation of any other immunising agent.*

*"It was recognised, too, that there must be incorporated into the vaccine preparation process itself a test for safety, which we refer to as the 'margin of safety', of such degree that the most sensitive tests upon a sample of each successive batch would be negative for living virus unless something unexpected had occurred, unknowingly, in the process of manufacture, or in subsequent handling, or in testing; it would follow that such would not be expected to occur but rarely."*

And, also:

*"The relative infrequency of severe paralysis under natural circumstances requires, above all else, that the vaccine must be free, insofar as it is possible to create such a preparation, of the capacity*

*to induce the disease that it is intended to prevent; nor should a vaccine for poliomyelitis cause such side effects as would make its use undesirable. If either the direct effects associated with its use, or incidental side-effects to any constituent, are of such nature as to make one prefer the chance of escaping the paralytic disease, one would not have a practically useful immunising agent."*

And further

*" ... The objective in the preparation of a poliomyelitis vaccine cannot include the knowing or wilfull acceptance of a risk that is tangible, or measurable to any degree. Any risk that is involved, so long as it is recognised, must be corrected, whatever may be its cause."*

Every point in this proclamation has been violated and there is no guarantee that it does not continue to be violated. Children receiving any of the polio vaccines continue contracting paralysis from the vaccine. The wide-spread incidence of chronic ill health, an endless stream of respiratory infections, usually resisting all treatment, occurring in small children and the continuing high incidence of child leukæmia and cancer are themselves the evidence that not all is healthy in the kingdom of vaccines.

The continued contamination of polio vaccines with animal viruses is of special concern. Goffe *et al.* (1961) pointed out that the vacuolating virus (SV40) which was resistant to formaldehyde occurred potentially in all polio vaccines, since until 1961 none of them was checked for this virus. It was also only one of many viruses and other agents lurking *"hitherto undetected"* in the monkey kidney tissue culture preparations. Despite these warnings, the authors considered oral administration of polio vaccines safer since they thought the alimentary tract may serve as a selective screen.

Gerber *et al.* (1961) reported on inactivation of vacuolating virus (SV40) by formaldehyde. Their studies showed there was a residual viable fraction of SV40 throughout the entire 14 days of observation. SV40 also showed a remarkable thermostability as evidenced by full retention of infectivity during heating without formaldehyde at 37 degC for a period of 14 days.

Randomly selected samples of poliomyelitis and adenovirus vaccines were tested for the presence of viable SV40: four of eight polio vaccines and three out of three adenovirus vaccines produced characteristic cytopathic changes in all subculture tubes. An isolate from each positive vaccine was identified by serum neutralisation tests.

The repeat tests with different lots of monkey kidney gave similar results. All subcultures derived from positive vaccines showed characteristic cytopathic changes between the seventh to the tenth day. They also found that the course of treatment of SV40 with 1:4,000 formaldehyde was characterised by a biphasic reaction. The major portion of the viral population was inactivated progressively at a slightly slower rate than poliovirus. The second phase of the curve indicated the persistence of a residual fraction which resisted inactivation.

When tested for more than ten days there was a marked delay in the appearance of cytopathic effects (CPE) caused by formaldehyde-treated SV40. In most cases CPE did not appear until the 11th day and the final infectivity titers were reached on the 13th and 14th days. The amount of viable SV40 depended on the concentration of this agent before formaldehyde treatment. The degree of contamination of the seed virus inoculum with SV40, the total incubation period of the kidney cell cultures during vaccine production and specific variations in the manufacturing process may also influence the final concentrations.

Sweet and Hilleman (1960) confirmed that, following injection of two doses of polio or adenovirus vaccines, a high percentage of persons developed relatively high antibody titers to SV40.

Results of studies by Shah (1973), Weiner *et al.* (1972), Baguley and Glasgow (1973) and others show that millions of children were indeed infected with SV40 during the course of vaccination with formalinised polio and adenovirus vaccines, with tragic long-term consequences. Baron *et al.* (1961) reported on more sensitive methods developed to test the commercial vaccines. They were able to detect viable virus in one out of every 23 commercial vaccines.

## Polio vaccination, leukæmia and cancer

Girardi *et al.* (1973) confirmed that the oncogenic effect of SV40 was especially pronounced in younger individuals. Innis (1965) wrote in his letter to the editor of The Lancet that

*"Since antigenic stimulation is known to cause hyperplasia of mammalian lymphoreticular tissue and since such hyperplasia may, in some strains of mice, proceed to malignant neoplasia, the possibility that a similar mechanism could operate in children submitted to the repeated antigenic stimulation inherent in immunisation seemed worth investigating."*

He looked at immunisation status of 59 (out of 65) patients with leukæmia and compared it with that of patients admitted for different illnesses and concluded that

*"The difference between the state of immunisation of leukæmic and non-leukæmic children may therefore be regarded as significant ... and the logical conclusion is that human lymphoreticular tissue, like that of some strains of mice, is possibly provoked to, or conditioned for, neoplasia by antigenic stimulation."*

He considered his study a pilot survey indicating a need for further investigation. As an interim measure, he recommended a protection against individual disorders, when indicated, instead of immunisation with triple antigen since "fewer clones would then be provoked to (unnecessary) proliferative activity."

Answering the criticism of his conclusion by Lancaster and Clements (1965) Innis stated:

*"Retrospective surveys are not the best means of determining antecedent events, but if mine was valueless, it should be simple to disprove an association between antigenic stimulation (including immunisation) and leukæmia."*

In another letter to the editor of The Lancet, Innis continued

*"...if the premises on which the deductions are based have been tested and found to be statistically significant, as have those used here, then any valid conclusion from these premises is inevitably true to fact; antigens, including those used for immunisation, are therefore leukæmogenic and carcinogenic in inviduals with the*

*requisite number of inherited and/or acquired mutant genes in a generative somatic cell."*

Dr Scheuer-Karpin (1965) confirmed that antigenic stimulation, such as that provided by chronic and/or recurring inflammatory disease, appeared to be higher in the histories of patients with leukæmia than in the histories of patients with other disorders. The inflammatory diseases included chronic tuberculosis, recurrent discharging inflammation of the ear, nose and throat and urinary tract; chronic bronchitis with bronchial asthma and chronic osteomyelitis.

There was also a higher incidence of eczema and autoimmune disorders in the cases than in the controls. The well-known causal relationship between administration of vaccines and accentuation and susceptibility to a great variety of chronic infections certainly supports Innis's (*op. cit.*) warnings.

## Flies Spread Polio Viruses

An interesting article on possible spread of polioviruses was published by Riordan *et al.* (1961). The authors described an experiment in which the oral polio vaccine was administered during a non-epidemic period (February 1958) to a group of Yaqui Indians living in the village of Guadalupe, Arizona.

The intention was to infect a small number of children with attenuated type 1 poliovirus vaccine and to follow the spread of the inapparent infection within the community by testing for the presence of poliovirus in rectal swabs from vaccinees and their contacts; in specimens of fæces from the various privies and the flies trapped in the village.

Six days after the administration of the oral vaccine, seven fly traps were set within the study area and five outside its periphery, but still within the village. The collections were made in the afternoons for about 70 days. Poliovirus types 1 and 3, ECHO 2, and Coxsackie B 5 viruses were isolated consistently from flies trapped both inside and outside the study area. Isolations increased during warm weather.

Type 3 poliovirus was isolated from virtually all the traps at one time or another, so it must have been widely disseminated throughout

the village. Type 1 poliovirus was isolated more during the latter part of February and March than at other periods. Many of the strains were tested on monkeys and shown virulent, intermediate and attenuated (from the vaccine). The number of vaccine viruses was small because the number of vaccinated children was small.

In this particular environment it was not uncommon to see food at mealtimes covered with flies so that they could act as disseminators of the enteroviruses.

During the summer of 1958 an outbreak of poliomyelitis occurred among the Blackfeet Indians residing in Glacier County, Montana. 19 cases occurred, of which 18 lived in the reservation. This represented an incidence of 171 cases per 100,000 in a population of 11,100 (Glacier County) while the rate for Indians on the reservation was 640 per 100,000 among the 2,810 residents. The rate of the entire state was 10 per 100,000.

Since 1947 only seven cases of poliomyelitis had been reported to the Montana State Board of Health, so it was highly unusual that so many cases occurred in a population which, due to poor sanitary conditions, usually enjoyed solid immunity to poliomyelitis.

Poliovirus 1 was recovered from 50 per cent of 16 children under the age of 1 and from 69 per cent of children between one and four years. It was not detected in persons 29 and older. Of 19 patients, nine were unvaccinated (mostly adults– four), while one was vaccinated three times, two twice and seven once.

Flies were extremely prevalent, commonly crawling on garbage and fæces scattered outside the homes. The authors demonstrated that vaccination did not prevent infection or produced an intestinal immunity, since over 50% of the children vaccinated three or more times were found to be excreting type 1 poliovirus. There was no evidence of a common source of infection. 90% of individuals tested had neutralising antibodies against all three poliovirus types, irrespective of vaccination status. Considering the above-mentioned circumstances, it is quite clear that vaccination was responsible for this epidemic in the Indian community.

Ashkenazi and Melnick (1962) described an induced latent infection of monkeys with vacuolating SV40 papova virus. They wrote that like other papova viruses, SV40 can establish latent infections. The virus has infected and transformed human cells, even producing chromosome changes in them. In hamsters, huge fibrosarcomas, larger than the host individual, are produced.

The authors induced a latent infection by a variety of routes in African primates with vacuolating SV40 virus. Virus was recovered from the urine of all four baboons inoculated parenterally, but not from three baboons infected by intranasal or oral administration of the virus. Kidney biopsies were obtained three to eight months after infection, when virus was no longer recoverable from the urine. The latent infection developed and the virus was recovered in four out of five previously positive monkeys in a second biopsy six to eight months after infection.

Black and Rowe (1962) described changes in morphology and growth characteristics by SV40 in human tissue such as buccal mucosa, skin and human embryonic kidney cultures. During the experiment, by the 18th to 20th day, a small number of multinucleated cells 2- 4 times the size of normal cells were seen in the inoculated tubes. During the first 30 days after inoculation, slow "non-specific" degeneration of all cellular types occurred. By the 34th day cellular proliferation and progressive increase in acidification was observed in the virus-inoculated tubes. During the next 30 days these areas spread over most of the glass which was covered by tissue culture medium. In places, the cell sheet was several layers thick.

From about the 50th to 55th day after inoculation, parts of the sheet underwent necrosis followed by re-growth of epithelial tissue. This resulted in patches of epithelial re-growth alternating with areas of fibroblastic tissue growing in a disorganised, criss-cross fashion.

When these altered cells were transplanted into hamsters 4-6 weeks old they produced local tumours in 3-4 weeks. Qualitative as well as quantitative chromosomal changes were also noted in response to SV40 infection.

## Polio Vaccine Viruses May Revert to Neurovirulence

Friedman *et al.* (1962) discussed the recent findings that there was an increase in neurovirulence for monkeys after human intestinal passage of the Sabin type 3 attentuated poliovirus.

They studied the possibility of similar changes with Sabin type 1 virus which is important in the epidemiology of polyomyelitis. They observed that the simian neurovirulence characteristic of the vaccine virus strain was unstable, tending to increase after human gastrointestinal passage. These changes occurred at any time after administration of the vaccine and were not correlated with the duration of gastrointestinal infection by the vaccine virus.

They gave newborn infants about 100 times the dose of vaccine virus that is usually given to older individuals. It is of importance to know that low neurovirulence for the monkey is the most important characteristic for any live poliovirus vaccine being considered safe for humans. The stability of this low virulence is also very important. However, many studies indicated that there was a random instability of the genetic characteristics of the virus. Some increase in simian neurovirulence of another agent, an attenuated encephalitis virus vaccine strain, has been also established and the present studies confirmed the same for the attentuated polio vaccine virus. However, the authors recommended caution in interpreting these findings.

What about the babies? Nobody seems to care.

It is not accidental that a patient who died in Michigan in 1963 within 16 days of Salk vaccine inoculation was found in autopsy to have died of disseminated myelitis. Another man who died after receiving type 3 oral polio vaccine had a disseminated encephalomyeltis.

Sabin (1963) contended that whether or not the virus was recovered from the central nervous system or stool of these victims did not indicate that the polio virus was responsible for their illness. Well, the timing does. But the coincidence has become a favoured speculation in the cover-up of the causal relationship between administration of polio virus vaccines and diseases of the central nervous system.

*"Highly qualified physicians with extensive experience in this field"* (Sabin, 1963) were only too happy to invoke coincidence and highly speculative explanations to divert attention away from the offending vaccine viruses. It is asking too much of coincidence to explain the incidence, time and again, all over the world, of the same illnesses and deaths clustering around certain (critical) days after vaccine administration. Perhaps the ultimate of the attempts at explaining this is the following statement of Sabin (*op. cit.*):

*"The fact that in recent years 20% to 30% of the reported paralytic cases had received three or more doses of Salk vaccine does not mean that the expected protection is only 70% to 80%, but is in large measure a reflection of the proportion of the population that has had this number of doses of Salk vaccine."*

Apparently, he did not even consider that this result could mean the total ineffectiveness of the vaccine.

Polio vaccine has been documented to spread polio to unvaccinated children and adults. Patterson and Bell (1963) reported on two cases of poliomyelitis in a nursery school in Glasgow. The first case occurred in a four year old child five months after receiving his second dose of inactivated vaccine. On the same day as this patient, another, unvaccinated, child became ill in the same nursery school. Poliovirus 1 was cultured in the fæces of both children. Special Advisory Committee on Oral Poliovaccines to the Surgeon General of the Public Health Service published a report on vaccine-associated cases. 87 such cases were reported in non-epidemic areas since the introduction of oral vaccines. The onset of illness fell between four and 30 days, with the majority occurring within eight to 21 days. This is again a perfect example of clustering around critical days. Continued polio vaccination was recommended.

Henderson *et al.* (1964) reported on 123 cases of paralytic poliomyelitis occurring less than 30 days after administration of polio vaccines.

They all talk about a very small number of cases of vaccine-associated paralytic disease. They all assume that the vaccine was

effective in preventing poliomyelitis, while in fact it was bringing paralytic polio into areas previously free of the disease for many years before mass vaccination took place. The reported cases were taken as the only cases occurring despite the general knowledge of and admission of the notoriously slack reporting system. Statistics have been based on these reported cases and no extrapolation was done of the kind recently done for the incidence of whooping cough in the United States based on the hospital admissions (Sutter and Cochi, 1992).

Larsen (1965) commented on the protective effect of Salk vaccine during an epidemic of polio in British Columbia. 1959 and 1960 were clearly epidemic years despite the widespread use of Salk vaccine during the preceding four years. The apparent decrease in the efficacy of the vaccine during 1960, especially in six to eleven year olds caused some concern. Nevertheless the author defended the vaccine by saying that, in 1959-1969, patients with paralytic polio who had had at least three doses of Salk vaccine appeared to be less severely affected that those who had had no vaccine or only one or two doses.

What about the unvaccinated people who did not contract polio? Nobody ever calculated the amount of protection enjoyed by the unvaccinated who did not contract the disease. If they did, they would find out that there must be other factors involved in the spread of infectious diseases including the polio.

Stolley *et al.* (1968) reported on another case of paralytic polio in an unvaccinated 16 month-old-child who was in close contact with his cousin who had received oral-type 2 vaccine 33 days before the onset of the disease.

Hopkins *et al.* (1969) analysed 103 cases of paralytic poliomyelitis reported in the USA in 1966 and 40 case in 1967. In 1965-67, eight cases of paralytic polio were reported in recent recipients of oral vaccine and 16 cases in contacts with the recipients.

The fact that only about 25% to 50% were vaccinated does not indicate vaccine efficacy. A vaccinated child should not contract polio. Many unvaccinated contracted the illness from the vaccinated.

The fact that not all unvaccinated people (in fact, the majority) do not contract the disease indicates there are other factors acting selectively in causing children to contract or escape the illness — not the vaccines.

Schonberger *et al.* (1976) reported on vaccine-associated poliomyelitis in the United States, in both vaccinees and their contacts. The interval between immunisation and the onset of polio clustered between seven and 21 days. There was an increased number of reports of the incidence of polio in contacts of vaccinees. These cases clustered between 20 and 29 days after the contacts were vaccinated. After 1964 the adult vaccination programme was curtailed and attention was centered on vaccinating infants. A substantial underreporting of cases continued.

Nightingale (1977) expressed concern about the decreasing rate of polio vaccination, mainly because of concern about side effects and polio-caused cases of paralytic polio. The swine-flu fiasco was another warning sign making American parents weary of polio vaccination.

Allegedly, by 1974, the vaccination compliance fell to 45%. Despite documented (and admitted) cases of vaccine-caused paralytic polio cases, the author recommended an increased push for polio vaccination.

It was certainly interesting that she did not document in any way that low compliance increased the polio incidence.

In 1979 Nathanson and Martin (1979) published an analysis of the epidemiology of poliomyelitis. They admitted that despite intensive study over a century, many basic points of the epidemiology of polio infection remain obscure and unknown. Among the most interesting findings is a significant upward trend in age, but no upward trend in incidence, between 1910 and 1954 in northern Europe and the US before mass vaccination programmes started.

Another important feature is a dramatic upsurge in incidence of polio between 1945 and 1954 for which the authors offer no explanation. In my opinion, this upsurge in polio incidence was in direct response to intensified pertussis, diphtheria and tetanus

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vaccination followed by well-documented occurrences of provocation poliomyelitis (see above).

The rise in age is, in my opinion, a direct reflection of improved sanitation and general hygiene coupled with increased prevalence of breast feeding which effectively protected infants below the age of one from contracting subclinical poliomyelitis, thus creating a large group of children above the age when children are susceptible to poliomyelitis. This conclusion is based on the known distribution of polio in underdeveloped countries, in which, due to poor sanitation and hygiene, babies are exposed to subclinical polio and achieve natural immunity early in life.

This may possibly be the reason behind the observed problems with polio vaccination in underdeveloped countries (Domok *et al.* 1974) characterised by a low "take rate" due to high neutralisation rate of the vaccine viruses. It takes five doses of OPV to prompt babies in underdeveloped countries to seroconvert and start shedding the vaccine virus (Anonymous, BMJ 1976). In my opinion, this happens only after the natural neutralisation capacity is totally depleted by repeated challenges with the vaccine.

Bottiger *et al.* (1979) described a case of paralytic polio in Sweden in 1977. The authors saw this as a good opportunity to study the spread of virus in a cluster of unvaccinated and vaccinated individuals. 64 of the 130 schoolchildren in a private kindergarten were unvaccinated and 66 vaccinated.

The investigation found that three adults and three children were excreting the virus. The mother of the index child was vaccinated and her two children were not. The father spent three months in the Netherlands and returned to Sweden two weeks before one of the children became ill. The mother and the two children had chills and diarrhoea in January.

A great variety of people lived nearby and had a lively social contact. Seven out of 20 preschool children belonged to a religious sect and were unvaccinated (that means 13 were vaccinated). All vaccinated individuals received a killed polio virus by injection. In the village where the patients lived there were frequent exchanges of

students and teachers from all over Europe and viruses could have been introduced this way. The studies of virus in sewage indicated that the virus was circulating widely in the community and was not restricted to the excretors detected in this study. Several members of the sect were vaccinated with live polio virus when abroad.

The authors concluded that, because the isolated viruses were not the vaccine type, they were probably not derived from the vaccines. However, others demonstrated that polio viruses change in passage through the gastrointestinal tract (see above) so this conclusion is irrelevant. It is noteworthy that the authors were constantly emphasising the number of unvaccinated childen, although the majority were in fact vaccinated. Also, they highlighted that seven of the tested vaccinated were not excreting the virus, while 14 of 20 unvaccinated were.

In my opinion it was much more interesting that six of the 20 tested unvaccinated children were not excreting the virus. I believe that the polio virus was probably either imported from The Netherlands by the father who returned two weeks before one of his unvaccinated children became ill, or by the German students who stayed with the family until Christmas. The fact that the minority of people were not vaccinated, while the majority were, is not pointing the finger at the unvaccinated as the source of this outbreak.

Schaap et al., (1984) described the 1978 polio outbreak in The Netherlands. The authors maintained that the outbreak was confined to the religious group refusing vaccination. However, they also stated that only 65,000 our of some 500,000 members of the the religious group had not been vaccinated. Also, there were a further 400,000 unvaccinated children not belonging to the religious group and who were not affected by the epidemic at all. Their figure 4 shows that populations with 100% vaccination rates had the highest numbers of polio cases during the outbreaks of polio between 1971 and 1975.

In the seventies and eighties, a number of reports appeared in medical literature dealing with the syndrome of poliomyelitis- like illness associated with acute bronchial asthma (Hopkin's syndrome).

Manson and Thong (1980) described three patients who suffered this syndrome. All patients had varying degrees of nonspecific immune deficiency although polio virus was not isolated in the acute stage. All patients had previously been fully immunised against poliomyelitis with Sabin vaccine in infancy. Immunodeficiency was suggested by several authors.

Asthmatics as a group have a reduced immunological response when compared with non-asthmatics. Patients with selective IgA deficiency have an increased predilection to bronchial asthma and allergies. All children developed paralysis a number of days after a severe asthma attack. In none of the children were polio virus titres examined at the time of the acute illness.

The authors considered the ætiology of "this devastating and mysterious syndrome" unknown, but admitted that "all recorded cases of (this) syndrome had previously been fully immunised against poliomyelitis". The published records of the association between polio vaccination and paralytic poliomyelitis led the authors to investigate the immunological status of these children. The immunological abnormalities of these children indicated some impairement of cell-mediated immunity — low T-cell numbers — which had been seen by one of the authors in association with paralytic poliomyelitis. The authors proposed that these children had an underlying minor immunodeficiency. Further immunosuppression occurred at the time of acute asthma attack as a result of stress, intercurrent infection, or corticosteroid therapy.

The authors also speculated that,during the host's decreased resistance, either an opportunistic virus or latent virus residing within the body (chronic infection that may follow vaccination) may invade the anterior horn cells and cause paralysis. There can be a latent period of several months. A parallel observation was described with Herpes simplex reactivation due to experimentally induced immunosuppression.

If this were true then it would be impossible to find the virus in conventional sites such as the alimentary tract. This would explain why polio virus could not be detected in these children. The authors

hinted that this was a pre-existing underlying immunosuppression. I personally feel that the immunosuppresion was caused by vaccination.

It is very likely that these children not only received the full course of polio vaccine but also DPT, which was most probably an important ætiological factor in these children's developing asthma as well. The suppression of acute asthma attack by corticosteroids resulted in paralysis.

The phenomenon of provocation illness after suppression of other symptoms is well-known. Williams *et al.*(1984) described a case of a patient who was sufferring from lichen planus which regressed with topical and systemic corticosteroids. One month later generalised nodular skin lesions erupted and spread rapidly, His lungs developed reticulonodular shadowing and symptoms of adult T-cell leukæmia were diagnosed. The case was even more interesting because the patient was showing antibodies against "human T-cell leukæmia virus" (HTLV). The authors acknowledged the connection between animal leukæmia and retroviruses.

1984 saw ever-increasing reports of immunosuppressive disorders in Africa and the beginning of the AIDS epidemic. The connection between AIDS epidemic and smallpox and polio vaccination has now been well established. It is quite feasible that in this case, the immunosuppression and latent infection by animal retroviruses (the contaminants of vaccines) manifested itself as lichen planus skin eruptions. Suppression of these skin eruptions (like the suppression of asthma attack in the previous three patients, leading to paralysis) led to full-blown symptoms of leukæmia.

Different viruses, different "illness", but the principle is the same: suppression of symptoms resulting in a provocation illness.

The WHO consultative group discussed the relation between acute persisting spinal paralysis and poliomyelitis vaccine over a period of ten years (1982). Over those ten years there have been consistent reports of cases thought causally related to the viruses in the vaccine, especially poliovirus type 3 in vaccine recipients, and type 2 in contacts. The authors conceded that neurovirulence tests of safety

do not prove or guarantee the innocuity of the vaccine. This called for an effective surveillance system.

The number of reported cases varied widely in different countries, in my opinion reflecting the varying quality of reporting systems. So the rate of these cases when given as 0.14 per million doses per year only reflects the rate of reporting, not the actual incidence. 698 cases altogether were reported, in which 253 has virus isolated; in 28 the diagnosis was on clinical grounds only. 92 patients were investigated for immunodeficiency; 12 were immunodeficient, and five died. Out of 70 contact cases 43 reported no polio immunisation history.

The grouping of cases due to types 2 and 3 within a short interval after immunisation and the isolation of vaccine-like strains of the virus from the central nervous system was taken as evidence of a causal relationship between vaccine administration and paralytic poliomyelitis.

Sabin (1982) discussed the vaccine control of poliomyelitis in the 1980s. He wrote that in the US, the average estimated number of non-persisting and persisting cases of paralytic polio was 135 per million total population per year during the pre-vaccine period 1951-1955. He compared this incidence with the reported cases of 26 per million total population in 1956-1960 (when only inactivated polio virus vaccine was used) and four per 100 million in 1973-1978 when only OPV was used.

However, these were only the reported cases and the system is known to be notorious for underreporting of infectious diseases. One has to consider that vaccine-related cases of polio can occur after a delay of several weeks or even months and years (see Manson and Thong 1980). The majority of these cases would not be considered associated with the polio vaccines.

Cerebral palsy cases probably are equally insidious vaccine-related neurological problems. All countries with intensive or mandatory vaccination of infants experienced a 400% increase in the incidence of cerebral palsy in the last fifty years, not diagnosed at birth.

Between April 1982 and June 1983, four children aged three to 24 months developed neurological abnormalities found to be compatible with vaccine-related poliovirus infection (Gaebler *et al.*. 1986). A vaccine-like strain of polio virus was isolated from all patients, and each developed symptoms persisting for at least six months. Three children had left leg paralysis and one had developmental regression, spasticity and progressive fatal cerebral atrophy. Two children had abnormal immune function (hypogammaglobulinæmia, combined immunodeficiency) and two had polio 3 infection.

The incidence of observed vaccine polio virus-related infection in Indiana recipients of oral polio vaccine was significantly greater than predicted. In each of these cases the referring doctors failed to associate the neurologic abnormality with administration of OPV. Referring diagnoses included trauma in two, spinal cord tumor, and failure to thrive.

The first child developed his symptoms 22 days after the initial dose of DPT and OPV. The second child developed his symptoms two weeks after a fourth DPT and OPV. The third child received his first DPT and OPV at four months and his second at six months. At seven and a half months he became listless, stopped verbal activity and had decreased use of his left arm and a leg. At eight months he was evaluated as suffering " ... failure to thrive, and slow development" and a diagnosis of cerebral palsy of undetermined origin was made. Poliovirus 2 was recovered from stool and throat. At 19 months CT showed profound bilateral cerebral atrophy; neurologic and respiratory function progressively worsened and the child died at 21 months. The parents refused permission for postmortem examination.

The fourth baby developed flaccid leg paralysis four weeks after the first DPT and OPV. Poliovirus type 3 was isolated from stool. The condition persisted for 18 months.

Despite reports like this one, the vaccinators keep administering multiple doses of polio vaccine. This is most pronounced in tropical countries where the vaccinators admit that vaccine does not work.

Up to five or more doses are administered to babies in India (Krishnan *et al.*. (1982)). Does anybody collect information on possible (often delayed) reactions to this practice?

Grist (1983) reported on safety of poliomyelitis vaccines and wrote *"During the past 20 years, live oral poliomyelitis vaccines have proved to be highly effective and very safe." And "... occasional cases of paralysis after the use of live vaccine, whether coincidental or attributable, have long been recognised, and in 1969 the World Health Organisation arranged a collaborative study to obtain definite information about such risks and how they may be reduced, if found to be real."*

Perhaps Freudian slips like *"The third country experienced a large fall in the number of vaccine associated cases after a change from monovalent to trivalent oral vaccine"* tell us more about the real incidence of vaccine-associated paralytic polio. If there were only sporadic cases of vaccine-associated cases then the fall could not be described as "large". And further, could the use of vaccine type 1 mean that vaccine-associated cases were no longer associated with the vaccine, since the wild virus infection is mostly associated with type 1?

In contrast, the fourth country reported a persistently high incidence of vaccine related type 2 and 3 cases until 1979, when only type 1 vaccine was given; reversion to the previous pattern followed return to the use of trivalent vaccine. Despite passing standard tests, the vaccine used in that country "seemed to differ in some way from that used elsewhere." Maybe the only difference was that this country efficiently reported cases of vaccine-associated cases.

Krishnan *et al.* (1983) reported that "The immunogenic efficacy of inactivated (Salk) poliovirus vaccine (IPV) was evaluated in infants in India, in view of the high frequency of vaccine failure after immunisation with oral (Sabin) poliovirus vaccine (OPV)".

On the basis of seroconversion rate (not the incidence of poliomyelitis) the authors concluded that *"the immunogenic efficacy of IPV was found satisfactory."*

Kim-Farley *et al.* (1984) reported on poliomyelitis in the USA and claimed a virtual elimination of disease caused by wild virus. In their summary they claim that the average number of paralytic poliomyelitis cases per year in the US has fallen from 16,000 just before vaccine was introduced in the 1950s to only 12 in 1979-83. However, in the paper they talk about reported cases of vaccine-associated polio and serious side-effects being reported following administration of the currently available inactivated poliovaccine. These authors either ignored cases published in medical literature, or, the published cases have never been reported to the CDC.

Cruickshank *et al.* (1984) described two cases of vaccine-associated paralytic polio linked in time and place. In case 1, a developmentally normal six weeks old girl was given her first DPT and OPV. 26 days later she developed fever and fell ill. She stopped eating and became floppy, unable to lift her head and was reluctant to move her legs. Poliovirus type 3 was isolated from fæces. In case 2 a developmentally normal little girl was given her first DPT and OPV at three months. 14 days later she became ill, feverish and irritable and partly paralysed. Polioviruses 2 and 3 were isolated from fæces. Despite these serious symptoms, both babies were given further doses of inactivated polio vaccine. One should ask what for, wasn't the polio disease from the vaccine enough to acquire natural immunity, even though the price was so high? Obsession with vaccination leads to these absurd actions by some doctors.

Kim-Farley *et al.* (1984) reported on the outbreak of paralytic poliomyelitis in Taiwan. Taiwan had been free of poliomyelitis outbreaks since 1975, but from 29 May to 2 October 1982, 1,031 cases of type 1 paralytic polio were reported to the Taiwan health authorities. Before the outbreak, approximately 80% of infants had received at least two doses of trivalent oral poliovaccine (OPV) by their first birthday.

Vaccinations received 28 days before onset of illness were not counted because they were considered as given during incubation time after exposure. However, not all cases should have been

considered of this kind. There was no evidence to prove or disprove this assumption. At least in some cases a causal link between vaccination and onset of polio should have been considered. Anyway, 35% of the victims were definitely vaccinated and in only 86% of polio patients was the vaccination status known. This is quite unbelievable, since the entire epidemic could have been vaccine-linked. It is well-documented that unvaccinated children can contract paralytic polio from vaccinated individuals. Sharing of water from municipal or non-municipal sources and sharing of toilet with at least one other family was another important factor in the incidence of poliomyelitis.

On one hand the authors wrote that this outbreak

*"...shows that major epidemics can occur in places that have been practically free of poliomyelitis for many years and that have high overall community vaccination levels"*

and on the other they stated that

*"failure to vaccinate rather than vaccine failure was the most important risk factor for paralytic poliomyelitis."*

They did not explain why 19% of victims received one dose, 8% two doses and 8% three doses of vaccine and yet contracted paralytic form of polio. Moreover, the rate of vaccination of the victims was only estimated. They also did not explain why the majority of unvaccinated did not contract the disease.

Anonymous (1984) commented on this epidemic and wrote that

*"In the event, it was established that the vaccine was very effective but that an epidemic can occur in a subpopulation that is poorly immunised ... The evidence from Taiwan seems to indicate that the benefits of OPV are largely confined to the immunised and that the herd benefit is unlikely to be much superior to that afforded by killed poliovaccine of adequate potency."*

This representation of the Taiwan outbreak is quite untrue, especially since 35% of the victims were vaccinated (see above).

Finland introduced injectable polio vaccine in 1957. Six injections are given at 5-6 and 20-24 months and 6-7, 11-13 and 16-17 years. In mid-October 1984 a six-year-old boy was hospitalised with serous

meningitis [Hovi *et al.*, (1986)]. He apparently recovered fully, but was found to be excreting poliovirus type 3. His mother and 4-year-old sister and many of his friends were found a few weeks later to be excreting poliovirus type 3. At about the same time, a 17-year old paralysed boy and a 2-year-old acutely febrile girl were found to be excreting type 3 poliovirus. None of these persons were in direct contact. Altogether, 9 patients with paralytic polio were identified in December 1984 and January 1985. The authors wrote that, except for the three adult patients, all the others had been fully vaccinated. Several of them had received a booster dose in the year before the onset of their illness.

The entire population of Finland was offered the oral polio vaccine between 9 February and 15 March, 1985. Although 45 vaccinees had neurological symptoms, the authors concluded that there was no proven case of OPV-induced paralytic disease in Finland in 1985. One suspected case was of a 33-year-old woman previously vaccinated with 3 doses of IPV. An asymmetrical lower motorneurone disease, not unlike poliomyelitis, started to develop 2 weeks after OPV administration. Vaccine-like poliovirus 2 was isolated from her stools. During this epidemic there was also a significant increase in incidence of Guillain-Barré syndrome temporally associated with the OPV campaign [Kinnunen *et al.* (1989)]. Kinnunen *et al.* (1989) concluded that live-attenuated polio viruses may, like other infectious viruses, sometimes trigger GBS.

When one considers that only 9 people contracted polio, most of whom (4) recovered and only one died (under general anæsthetic) the damage from the vaccine far exceeded the damage from contracting poliomyelitis.

Polio vaccine is usually hailed as the safest vaccine of them all. However, medical papers abound with examples of serious damage caused by polio vaccine. Chonmaitree and Lucia (1986) described a case of a six-week old baby girl who experienced a near-miss cot death one day after she received her first dose of DPT and oral polio virus vaccine. A vaccine-like polio virus type 2 was isolated from the CSF. The child ended up with severe neurologic abnormalities.

Rasch, Wells and Fowlkes (1986) reported a case of a five-month-old baby boy who was infected with vaccine-like strain of poliovirus received by two family members one and two months previously. The little boy suffered serious neurological damage. From the age of two months, the child had failed to thrive and showed obvious signs of immuno-suppression. Although the authors do not say it, it is reasonable to suppose it was caused by the child's routine DPT vaccination; the polio vaccine may well have been the last straw.

Slater *et al.* (1990) reported on poliomyelitis outbreak in Israel in 1988. 15 cases of paralytic polio associated with type 1 occurred between July and October 1988. Nine of the victims had previously been immunised with at least three doses of oral poliovacines. Two victims received 1 dose and one two doses of OPV. Four victims were unvaccinated.

It is quite obvious that vaccination was totally ineffective in preventing paralytic polio outbreak. However, it was concluded that intensified vaccination was the answer.

The authors of the article were divided in their interpretation of the cause of this outbreak. One group claimed that wild poliovirus was transmitted to susceptible people because of low gut immunity (recipients of IPV) and the other claimed that it was the exposure to epidemic strain of wild virus from sewage that caused the outbreak and recommended an intensified vaccination programme combining IPV and OPV.

One must ask two valid questions: how can a vaccine containing different strains of poliovirus protect against a different epidemic strain? And how many vaccinations would protect if three vaccine doses did not?

The authors omitted one valid comment: vaccination simply does not work.

After reading the published evidence to the contrary, the reader may justly be surprised that The Lancet 1990 published an article by Beale — *Polio vaccines: time for a change in immunisation policy?*

which among others claimed that while OPV and IPV were initially prone to contamination by simian visuses, one of which, SV 40, was resistant to inactivation with formalin, there seemed to be no evidence that human infection with SV40 caused disease.

Until 1985 no one knew that about 70% of African Green monkeys, the species still used to produce the polio vaccine, had Simian Immunodeficiency Virus (SIV), very similar to the Human Immunodeficiency Virus (HIV). The virus, which occurs naturally in these monkeys, does not cause disease in this species but kills Rhesus macaques, which were used in American laboratories to produce polio vaccine until the early sixties. Macaques used in American laboratories died of SIV infection which they contracted from Green monkeys in captivity. [See also below: Pascal (1991)].

In the 1970s in the United States, homosexual men were exposed to both SIV and SV40 through unauthorised use of polio vaccine administered at weekly intervals in a desperate attempt to treat genital herpes (Kyle 1992).

After admitting that all three vaccine strains show some tendency to revert to neurovirulence (type 3 back mutation at nucleotide 472 usually occurs within a few days of vaccine administration) and occasionally cause paralytic poliomyelitis, three policy options were presented, none of which was to stop polio vaccination in American infants.

After 35 years of immunisation which did not eradicate polio even in temperate climates (problems of efficacy of polio vaccines in tropical countries being very obvious) the first option was to persist with OPV for infant immunisation as the basis of a global eradication program. The second option was to adopt IPV in place of OPV. The author quoted the example of Sweden and Netherlands as an example that successful eradication was possible. The third option was to use IPV and OPV sequentially. Table II listed five possible schedules between the ages of two months and six years of IPV and OPV.

An outbreak of paralytic polio in Holland (17th September to 9th October, 1992) is one of many good examples of a biased handling

of the data from the incidence of infectious diseases in vaccinated individuals by proponents of vaccination.

On 3rd October The Lancet (1992:841) published a short report by Marjanke Spanjer on the "*Netherlands: poliomyelitis epidemic*'. As of September 30th, there were notifications of paralytic poliomyelitis in two patients, both members of a religious sect which rejects vaccination and keep largely to themselves. Twenty percent of the students at the Christian secondary school attended by the two victims were carrying the virus. *"The Dutch authorities are convinced that some hundreds of persons have been infected"*. However, MMWR (16th October, 1992) reported that there were five cases of paralytic polio in all, two of which, both members of the above religious sect, were in fact vaccinated. Two of the victims, allegedly without a record of polio vaccination, were 39 and 33 years old. These two, and a six-year-old boy, were not members of the religious sect. All victims had polio virus type 3 isolated. This usually indicates the polio vaccine virus. It is quite clear that, if it were the religious group that caused the above outbreak, it was so because both victim members were vaccinated and excreting the polio virus type 3. Also, five cases widely dispersed can hardly be described as an epidemic even though a number of the victims' contacts were excreting the virus.

Epidemics of polio in countries like Israel and elsewhere affecting individuals given three or more doses clearly demonstrates that no number of doses confer immunity effective during epidemics. After all, that is the reason for vaccinating -- to prevent individuals from catching the disease during an epidemic.

## References


1. McCloskey, B.P., 1950.
   The relation of prophylactic inoculations to the onset of poliomyelitis.
   Lancet; 18 April: 659-663.
2. Hill, B.A., and Knowelden, J., 1950.
   Inoculation and poliomylitis.
   A statistical investigation in England and Wales in 1949.
   Br Med J; 1 July: 1-6.
3. MacCallum, F.O., 1950.
   Clinical poliomyelitis in association with peripheral inoculation of prophylactics.
   Br Med J; 1 July: 6-7.
4. Banks, H.S., and Beale, A.J., 1950.
   Poliomyelitis and immunisation against whooping-cough and diphtheria.
   Br Med J; 29 July: 251-252.
5. Leake, J.P., 1950.
   Anterior poliomyelitis and pertussis and diphtheria immunization.
   Br Med J; **144 (3)**: 259-260.
6. Bell, J., 1950.
   In: Leake, J.P., 1950
   Br Med J; **144(3)**: 260
7. Anderson, G.W., and Skaar, A.E., 1951.
   Poliomyelitis occurring after antigen injections.
   Pediatrics; **7 (6)**: 741-759.
8. Martin, J.K., 1950.
   Local paralysis in children after injections.
   Arch Dis Child; **25**: 1.
9. Geffen, D.H., 1950.
   Incidence of paralysis occurring in London children within four weeks after immunisation.
   Med Offr; **83**: 137.
10. Wyatt, H.V., 1981.
    Provocation poliomyelitis: neglected clinical observations from 1914 to 1950.
    Bull Hist Med; **55**: 543-557.
11. Paffenbarger, R.S., Jr., 1957.
    The effect of prior tonsillectomy on incidence and clinical type in acute poliomyelitis.
    Am J Hyg; **66**: 131-150.
12. Lambert, S.M., 1936.
    A yaws campaign and an epidemic of poliomyelitis in Western Samoa.
    J Trop Med Hyg; **389**: 41-46.
13. Wyatt, H.V., Mahadevan, S., and Srinivasan, S., 1992.
    Unnecessary injections and paralytic poliomyelitis in India.
    Trans Roy Soc Trop Med Hyg; **86**: 546-549.

14. Wyatt, H.V., 1993.
Provocation paralysis.
Lancet; **341**: 61- 62.
15. Jungeblut, C.W., and Engle E.T., 1932.
Resistance to poliomyelitis. The relative importance of physiologic and immunologic factors.
J Am Med Ass; **99 (25)**: 2091-2097.
16. Paul, J.R., Salinger, R., and Trask, J.D., 1932.
"Abortive" poliomyelitis.
J Am Med Ass; 98: 2262.
17. Anderson, G.W., Anderson, G., Skaar, A., and Sandler, F., 1957.
Poliomyelitis in pregnancy
Am J Hyg; **55**: 127-139.
18. Aycock, W.L., 1926.
The epidemiology of poliomyelitis. With reference to its mode of spread.
J Am Med Ass; **87 (2)**: 75-79.
19. Hudson, N.P., Lennette, E.H., and Gordon, F.B., 1936.
Factors of resistance in experimental poliomyelitis.
J Am Med Ass, **106 (24)**: 2037-2042.
20. Nathanson, N., and Martin, J.R., 1979.
The epidemiology of poliomyelitis: enigmas surrounding its appearance, epidemicity and disappearance.
Am J Epidemiology; **110(6)**: 672-692.
21. Francis, T., Korns, R.F., Voight, R.B., Boisen, M., Hemphill, F.M., Napier, J.A., and Tolchinski, A., 1955.
Evaluation of the 1954 poliomyelitis vaccine trials.
From the poliomyelitis vaccine evaluation center
University of Michigan, Ann Arbor, Michigan. 12 April, 1955: 50pp.
22. Nathanson, N., and Langmuir, A.D., 1963a.
The Cutter Incident. Poliomyelitis following formaldehyde-inactivated poliovirus vaccination in the United States during the spring of 1955. I. Background.
Am J Hyg; **78**: 16-28.
23. Nathanson, N., Langmuir, A.D., 1963b.
*Ibid.* II. Relationship of poliomyelitis to Cutter vaccine.
Am J Hyg; **78**: 29-60.
24. Nathanson, N., Langmuir, A.D., 1963c.
**Ibid.** III. Comparison of the clinical character of vaccinated and contact cases occurring after use of high rate lots of Cutter vaccine.
Am J Hyg; **78**:61- 81.
25. Rustigian, R., Johnston, P., and Reihart, H., (1955).
Infection of monkey kidney tissue cultures with virus-like agents.
Proc Soc Exp Biol Med; **88**: 8-16.

26. Hull, R.N., Minner, J.R., and Smith, J.W., 1956.
New viral agents recovered from tissue cultures of monkey kidney cells.
I. Origin and properties of cytopathogen agents S.V.1, S.V.2, S.V. 3, S.V. 4, S.V. 5, S.V. 6, S.V.11, S.V. 12, and S.V. 15.
Am J Hyg; **63**: 204-215.
27. Hull,R., Minner, J.R., and Mascoli, C., 1958.
New viral agents recovered from tissue cultures of monkey kidney cells.
III. Recovery of additional agents both from cultures of monkey tissues and directly from tissues and excreta.
Am J Hyg; **68**: 31-44.
28. Malherbe, H., and Harwin, R., 1957.
Seven viruses isolated from the vervet monkey.
Br J Exp Pathol; **38 (5)**: 539-541.
29. Sweet, B.H., and Hilleman, M.R., 1060.
The vacuolating virus SV40.
Proc Soc Exp Biol Med; **105**: 420-427.
30. Morris, J.A., Blount, R.E., Jr. and Savage, R.E., 1956.
Recovery of cytopathogenic agent from Chimpanzees with coryza. (22538).
Proc Soc Exp Biol Med; **92**: 544-549.
31. Parrott, R.H., Vargoslo, A.J., Hyun Wha Kim, Cumming, C., Turner, H., Huebner, R.J., Chanock, R.M., 1961
II. Serologic studies over a 34-month period in children bronchiolitis, pneumonia and minor repiratory diseases.
J Am Med Ass; **176(8)**: 653-657
32. Chanock, R.M., Wha Kim, H., Vargosko, A.J., Deleva, A., Johnson, K.M., Cumming, C., and Parrott, R.H., 1961.
Respiratory syncytial virus.
J Am Med Ass; **176 (8)**: 647-653.
33. Hamparian, V.V., Ketler, A., and Hilleman, M.R., 1961.
Recovery of new viruses (Coryza virus) from cases of common cold in human adults.
Proc Soc Exp Med Biol; **108**: 444-453.
34. Melnick, J.L., and Stinebaugh, S., 1962.
Excretion of vacuolating SV-40 virus (papova virus group) after ingestion of oral polio vaccines.
Proc Soc Exp Biol Med; **109**: 965-968.
35. ZuRhein, G.M., 1965.
Particles resembling papova viruses in human cerebral demyelinating disease.
Science: 9 February: 1477-1479.
36. Weiner, L.P., Herndon, R.M., Narayan, P., Johnson, R.T., Shah, K., Rubinstein, L.J., Preziosi, T.J., and Conley, F.K., 1972.
Isolation of virus related to SV40 from patients with progressive multifocal leukoencephalopathy.
N Engl J Med; **286(8)**: 385-389.

37. Shah, K.V., McCrumb, F.R.Jr., Dabiel, R.W., and Ozer, H.L., 1972.
Serologic evidence for a simian-virus-40-like infection in man.
J Nat Cancer Inst: **48**: 557-561.
38. Baguley D.M., and Glasgow, G.L., 1973.
Sabacute sclerosing panencephalitis and Salk vaccine.
Lancet; 6 October: 763-765.
39. Eddy, B.E., Borman, G.S., Berkeley, W.H., and Young, R.D., 1961.
Tumors induced in hamsters by injection of Rhesus monkey kidney cell.
Proc Soc Exp Biol Med; **107**: 191.
40. Eddy, B.E., Bormna, G.S., Grubs, G.E. and Young, R.D., 1962.
Identification of the oncogenic substance in Rhesus monkey kidney cell cultures as simian virus SV40.
Virology; **17**:65-75.
41. Girardi, A.J., Sweet, B.H., Slotnick, V.B., and Hilleman, M.R., 1962.
Proc Soc Exp Biol Med; **109**: 649-660.
42. Rabson, A.S., *et al.*, 1962.
Papillary ependymomas produced in *Rattus (Mastomys) natalensis* inoculated with vacuolating virus (SV 40).
J Nat Cancer Inst; **29**: 765-787.
43. Fraumeni, J.F., Ederer, F., and Miller, R.W., 1963.
An evaluation of the carcinogenicity of Simian virus 40 in man.
J Am Med Ass;**185 (9)**: 713-718.
44. Shein, H.M., and Enders, J.F., 1962.
Transformation induced by Simian virus 40 in human renal cell cultures.
I. Morphology and growth characteristics.
Proc Nat Acad Sci USA; **48**: 1164- 1172.
45. Koprowski, H., *et al.*, 1962.
Transformation of cultures of human tissue infected with simian virus SV40.
J Cell Comp Physiol; **59**: 281-292.
46. Melnick,J.L., 1962.
Papova virus group.
Science; **135**: 1128- 1130.
47. Shah, K.V., Daniel, R.W., and Warszawski, R.M., 1973.
High prevalence of antibodies to BK virus, an SV40 - related papova virus, in residents of Maryland.
J Infect Dis; **128 (6)**: 784-787.
48. Kyle, W.S., 1992.
Simian retroviruses, poliovaccine, and origin of AIDS.
Lancet; **339**: 600-601.
49. Goldberg, B., 1992.
Origin of AIDS.
Lancet; **339 (20 June)**: 1548

50. Morris, J.A., Johnson, K., Aulisio, C., and Chanock, R., 1961.
Clinical and serologic responses in volunteers givne vacuolating virus (SV40) by respiratory route.
Proc Soc Exp Biol Med; 108: 56-59.
51. Myers, G., MacInnes, K., and Korber, B., 1992.
The emergence of simian/human immunodeficiency viruses.
AIDS Res Hum Retrovir; **9**: 373-386.
52. Salk, J.E., 1955.
Considerations in the preparation and use of poliomyelitis virus vaccine.
J Am Med Ass; **158**: 1239-1248.
53. Goffe, A.P., Hale, J., and Gardner, P.S., 1961.
Poliomyelitis vaccines.
Lancet; 18 March: 612.
54. Gerber, P., Hottle, G.A., and Grubbs, R.E., 1961.
Inactivation of vacuolating virus (SV40) by formaldehyde. (26892).
Proc Soc Exp Biol Med; **108**: 205-209.
55. Baron, S., Kirschstein, R.J., Hoosier van, G.L., Jr., Abinanti, F.R., and Hottle, G.A., 1961.
The safety test for poliomyelitis vaccine.
II. Concentrates of poliomyelitis vaccine for the monkey test.
Am J Hyg; **74**: 220-224.
56. Girardi, A.J., Sweet, B.H., and Hilleman, M.R., 1963.
Factors influencing tumor induction in hamsters by vacuolating virus, SV40. (28133).
Proc Soc Exp Biol Med; **112**: 662-667.
57. Innis, M.D., 1965.
Immunisation and childhood leukæmia.
Lancet; 13 March: 605-606.
58. Lancaster, H.O., and Clements, F.W., 1965.
Immunisation and childhood leukæmia.
Lancet, 20 March: 654-655.
59. Innis, M.D., 1965.
Immunisation and childhood leukæmia.
Lancet; 17 April: 867.
60. Scheuer-Karpin, R., 1965.
Immunisation and childhood leukæmia.
Lancet, 5 June: 1224-1225.
61. Innis, M.D., 1968.
Oncogenesis and poliomyelitis vaccine
Nature; **219**: 972-973.
62. Elves, M.W., Roath, S., and Israels, M.C.G., 1963.
The response of lymphocytes to antigen challenge in vitro
Lancet, 13 April: 807-808.

63. Riordan, J.T., Paul, J.R., Yoshika, I., and Horstmann, D.M., 1961.
The detection of poliovirus and other enteric viruses in flies.
Am J Hyg; **74**: 123-136.

64. Ashkenazi, A., and Melnick, J.L., 1962.
Induced latent infection of monkeys with vacuolating SV40 papova virus.
Virus in kidneys and urine. (27794).
Proc Soc Exp Biol Med; **111**: 367- 372.

65. Black, P.H., and Rowe, W.P., 1962.
Transformation of Hamster kidney monolayers by vacuolating virus, SV40.
Virology; **19**: 107-109.

66. Friedman, R.M., Kirschstein, R.L., Borman, G.S., and Robbins, F., 1962.
Characteristics of Sabin type 1 poliovirus after gastrointestinal passage in newborn infants. I. Monkey neurovirulence and temperature marker findings.
Am J Hyg; **76**: 137-143.

67. Sabin, A.B., 1963.
Is there an exceedingly small risk associated with oral poliovirus vaccine?
J Am Med Ass; **183 (4)**: 268-271.

68. Patterson, W.J., and Bell, E.J., 1963.
Poliomyelitis in a nursery school in Glasgow.
Br Med J; 15 June: 1574-1576.

69. The Special Advisory Committee on Oral Poliomyelitis Vaccines, 1964.
Oral poliomyelitis vaccines.
J Am Med Ass; **190 (1)**: 49-51.

70. Geffen, T.J., and Spicer, C.C., 1960.
Poliomyelitis in the vaccinated England and Wales, 1958.
Lancet; 9 July: 87-89.

71. Henderson, D.A., Witte, J.J., Morris, L., and Langmuir, A.D., 1964.
Paralytic disease associated with oral polio vaccines.
J Am Med Ass; **190 (1)**: 41-48.

72. Sutter, R.W.,and Cochi S.L., 1992.
Pertussis hospitalizations and mortality in the United States, 1985-1988.
J Am Med Ass; **267(3)**: 386-391.

73. Larsen, A.A., 1965a.
The protective effect of Salk vaccine during an epidemic of poliomyelitis, British Columbia, 1959- 1960.
Can J Publ Health; **56 (2)**: 47-50.

74. Larsen, A.A., 1965b.
Poliovirus antibodies in British Columbia School Children.
Can J Publ Health; **56 (2)**: 51-53.

75. Stolley, P.D., Joseph, J.M., Allen, J.C., Deane, G., and Janney, J.H., 1968.
Poliomyelitis associated with type-2 poliovirus vaccine strain.
Lancet; 30 March: 661-663.

76. Hopkins, C.C., Dismukes, W.E., Glick, T.H., and Waren, R.J., 1969.
Surveillance of paralytic poliomyelitis in the United States.
J Am Med Ass, **210 (4)**: 694-700.

77. Schonberger, L.B., McGowan, J.E., Jr., and Gregg, M.B., 1976.
Vaccine associated poliomyelitis in the United States, 1961-1972.
Am J Epidemiol; **104 (2)**: 202-211.
78. Nightingale, E.O., 1977.
Recommendations for a national policy on poliomyelitis vaccination.
N Engl J Med; **297**: 249- 253.
79. Nathanson, N., and Martin, J.R., 1979.
The epidemiology of poliomyelitis: enigmas surrounding its appearance, epidemicity, and disappearance.
N Engl J Med; **110 (6)**: 672-692.
80. Domok, I., Balayan, M.S., Fayinka, O.A., Skrtic, N., Soneji, A.D., and Harland, P.S.E.G., 1974.
Factors affecting the efficacy of live poliovirus vaccine in warm climates.
Bull WHO; **51**: 333- 347.
81. Anonymous, 1976.
Antibody response of infants in tropics to five doses of oral polio vaccine.
Br Med J: 3 April: 812.
82. Bottiger, M., Mellin, P., Romanus, V., Soderstrom, H., Wesslesn, T., and von Zeipel,G., 1979.
Epidemiological events surrounding a paralytic case of poliomyelitis in Sweden.
Bull WHO; **57 (1)**: 99-103.
83. Schaap, G.J.P., Bijerk, H., Coutinho, R.A., Kapsenberg, J.G., and Wesel, A.L., von, 1984.
The spread of wild polioviruses in the well-vaccinated Netherlands in connection with the 1978 epidemic.
Progr Med Virol; **29**: 124 - 140.
84. Manson, J.I., and Thong, Y.H., 1980.
Immunological abnormalities in the syndrome of poliomyelitis-like illness associated with acute bronchial asthma (Hopkins's syndrome).
Arch Dis Child; **55**: 26-32.
85. Williams, C.K.O., Alabi, G.O., Junaid, T.A., Saxinger, C., Gallo, R.C., Blayney, D.W., Blattner, W.A., Greaves, M.F., 1984.
Human T cell leukæmia virus associated lymphoproliferative disease: report of two cases in Nigeria.
Br Med J; 1495-1496.
86. WHO Consultative Group.
The relation between acute persisting spinal paralysis and poliomyelitis vaccine — results of a ten-year enquiry.
Bull WHO; **60**: 231-242.
87. Sabin, A.B., 1982.
Vaccine control of poliomyelitis in the 1980s.
Yale J Biol Med; **55**: 383-389.

88. Gaebler, J.WE., Kleiman, M.B., Morris, M.M., French, L.V., Chastain, G., Barrett, C., and Griffin, C., 1986.
Neurologic complications in oral polio vaccine recipients.
J Pediatrics; **108**: 878-881.

89. Krishnan, R., Jadhav, M., Selvakumar, R., John, J. T., 1982.
Immune response of infants in tropics to injectible polio vaccine.
Br Med J; 16 Jan: 164.

90. Grist, N.R., 1983.
Safety of poliomyelitis vaccines.
Br Med J; **286**: 917.

91. Krishnan, R., Jadhav, M., and John, J. T., 1983.
Efficacy of inactivated poliovirus vaccine in India.
Bull WHO; **61 (4)**: 689-692.

92. Kim-Farley, R.J., Schonberger, L.B., Nkowane, B.M., Kew, O.M., Bart, K. J., Orenstein, W.A,., Hinman, A.R., Hatch, M.H., and Kaplan, J.E., 1984. Poliomyelitis in the USA: virtual elimination of disease caused by wild virus. Lancet; 8 Dec: 1315-1317.

93. Cruickshank, J.G., L'Orme, R., Haas, L., Gill, N.O., Roebuck, M.O., Magrath, D.I., and Chamberlain, R., 1984.
Two cases of vaccine-associated paralytic poliomyelitis linked in time and place.
Lancet; 6 Oct: 804-805.

94. Kim-Farley, R.J., Litchfield, P., Orenstein, W.A., Bart, K.J., Rutherford, G., Shu-Tao Hsu, Schonberger, L.B., Kung-Jong Lui and Chau-Ching Lin, 1984.
Outbreak of paralytic poliomyelitis, Taiwan.
Lancet; 8 Dec: 1322-1324.

95. Anonymous, 1984.
Polio reconsidered.
Lancet; 8 Dec: 1309- 1310.

96. Chonmaitree, T., and Lucia., H., 1986.
Presence of vaccine-strain poliovirus in cerebrospinal fluid of patient with near-miss sudden infant death syndrome.
Amer J Dis Child; **140**: 1212-1213.

97. Rasch, D.K., Wells, O., and Fowlkes, J., 1986.
Fatal disseminated infection due to poliovirus type 2 vaccine.
Amer J Dis Child; **140**: 1211-1212.

98. Hovi, T., Huovinlainen, A., Kuronen, T., Poyry, T., Salama, N., Cantell, K., Kinnunen, E., Lapinleimu, K., Roivainen, M., and Stenvik, M., 1986.
Outbreak of paralytic poliomyelitis in Finland: widespread circulation of antigenetically altered poliovirus type 3 in a vaccinated population.
Lancet; 21 June: 1427-1432

99. Kinnunen, E., Farkkila, M., Hove, T., Juntunen, J., and Weckstrom, P., 1989.
Incidence of Guillain-Barré syndrome during a nationwide oral poliovirus campaign.
Neurology; 39: 1034-1036.

100. Slater, P.E., Orenstein, W.A., Morag, A., Avni, A., Handsher, R., Green, M.S., Costin, C., Yarrow, A., Rishpon, S., Havkin, O., Ben-Zvi, T., Kew, O.M., Rey, M., Epstein, I., Schwartz, T.A., and Melnick, J.L., 1990.
Poliomyelitis outbreak in Israel in 1988: a report with two commentaries.
Lancet; 19 May: 1192- 1198.

101. Beale, A.J., 1990.
Polio vaccines: time for a change in immunisation policy?
Lancet; **335**: 839-842.

88. Gaebler, J.WE., Kleiman, M.B., Morris, M.M., French, L.V., Chastain, G., Barrett, C., and Griffin, C., 1986.
Neurologic complications in oral polio vaccine recipients.
J Pediatrics; **108**: 878-881.

89. Krishnan, R., Jadhav, M., Selvakumar, R., John, J. T., 1982.
Immune response of infants in tropics to injectible polio vaccine.
Br Med J; 16 Jan: 164.

90. Grist, N.R., 1983.
Safety of poliomyelitis vaccines.
Br Med J; **286**: 917.

91. Krishnan, R., Jadhav, M., and John, J. T., 1983.
Efficacy of inactivated poliovirus vaccine in India.
Bull WHO; **61 (4)**: 689-692.

92. Kim-Farley, R.J., Schonberger, L.B., Nkowane, B.M., Kew, O.M., Bart, K. J., Orenstein, W.A,., Hinman, A.R., Hatch, M.H., and Kaplan, J.E., 1984. Poliomyelitis in the USA: virtual elimination of disease caused by wild virus. Lancet; 8 Dec: 1315-1317.

93. Cruickshank, J.G., L'Orme, R., Haas, L., Gill, N.O., Roebuck, M.O., Magrath, D.I., and Chamberlain, R., 1984.
Two cases of vaccine-associated paralytic poliomyelitis linked in time and place.
Lancet; 6 Oct: 804-805.

94. Kim-Farley, R.J., Litchfield, P., Orenstein, W.A., Bart, K.J., Rutherford, G., Shu-Tao Hsu, Schonberger, L.B., Kung-Jong Lui and Chau-Ching Lin, 1984.
Outbreak of paralytic poliomyelitis, Taiwan.
Lancet; 8 Dec: 1322-1324.

95. Anonymous, 1984.
Polio reconsidered.
Lancet; 8 Dec: 1309- 1310.

96. Chonmaitree, T., and Lucia., H., 1986.
Presence of vaccine-strain poliovirus in cerebrospinal fluid of patient with near-miss sudden infant death syndrome.
Amer J Dis Child; **140**: 1212-1213.

97. Rasch, D.K., Wells, O., and Fowlkes, J., 1986.
Fatal disseminated infection due to poliovirus type 2 vaccine.
Amer J Dis Child; **140**: 1211-1212.

98. Hovi, T., Huovinlainen, A., Kuronen, T., Poyry, T., Salama, N., Cantell, K., Kinnunen, E., Lapinleimu, K., Roivainen, M., and Stenvik, M., 1986.
Outbreak of paralytic poliomyelitis in Finland: widespread circulation of antigenetically altered poliovirus type 3 in a vaccinated population.
Lancet; 21 June: 1427-1432

99. Kinnunen, E., Farkkila, M., Hove, T., Juntunen, J., and Weckstrom, P., 1989.
Incidence of Guillain-Barré syndrome during a nationwide oral poliovirus campaign.
Neurology; 39: 1034-1036.

100. Slater, P.E., Orenstein, W.A., Morag, A., Avni, A., Handsher, R., Green, M.S., Costin, C., Yarrow, A., Rishpon, S., Havkin, O., Ben-Zvi, T., Kew, O.M., Rey, M., Epstein, I., Schwartz, T.A., and Melnick, J.L., 1990.
Poliomyelitis outbreak in Israel in 1988: a report with two commentaries.
Lancet; 19 May: 1192- 1198.

101. Beale, A.J., 1990.
Polio vaccines: time for a change in immunisation policy?
Lancet; 335: 839-842.

# 8

# INFLUENZA VACCINES: Guillain-Barré, Legionnaires' — Is it really worth it?

No vaccines have been proven effective and safe, and the 'flu vaccine is no exception. Smith (1974), in his Lancet article on *"Vaccination and the control of influenza"*, described the effect of influenza vaccination on Post Office staff. He compared absences due to sickness in over 50,000 employees, to whom an injection of influenza vaccine was offered each winter, with absences in a similar group of employees not offered any vaccination. He concluded that the *"annual offer of an injection of influenza vaccine in a large industry has not resulted in a significant reduction in sickness"*.

Dr McCarthy, medical director of the Commonwealth Serum Laboratories in 1976, was quoted in The Age (on 18.4.1976) as not having a 'flu shot in ten years.

*"I regard myself as a healthy middle-aged man and I think that I can withstand a bout of 'flu if I get it."*

The same article quoted Dr John Forbes of Fairfield Hospital in Melbourne who said studies of immunity imparted by 'flu vaccines showed effectiveness levels down as low as 30%.

Ever since the 'flu vaccine was introduced, published reports of ineffectiveness and adverse reactions including deaths were filling the medical journals.

Curphey (1947), in an article *"Fatal allergic reaction due to influenza vaccine"*, described a case of a three and a half year old child who developed abdominal pain, chills, vomiting and convulsions four hours after the injection of influenza A and B vaccine. Besides very high temperature, this child developed

cyanosis and pronounced bleeding at the site of the injection (indicating the presence of unclotting blood, one of the symptoms of non-specific stress syndrome). Seven hours after the onset of the initial symptoms, the child showed signs of shock with collapse of the veins and overheating (hyperthermia). The child became more cyanotic and died despite being put on a respirator. The presence of fluid (unclotting) blood was confirmed at autopsy.

Major Warren (1956) described a case of encephalopathy due to influenza vaccine in a 19-year-old man. He had been entirely well until the day of admission when, in the morning, he was injected with influenza virus vaccine. At 2.30 pm he had a sudden onset of profuse rhinorrhea and wheezing. Half an hour later he became feverish, had shaking chills, soreness behind his eyes and aches in his head, back and arms. He became confused and semiconscious and experienced muscle weakness and hyporeflexia on his right side. The patient recovered slowly over a three week period. It was revealed later that one year previously he had had severe malaise for two days following influenza vaccination. However, he did not have allergies of any kind and had no history of intolerance to eggs ('flu vaccine virus is cultured on hen eggs).

Rosenberg (1970) wrote that post-vaccinal encephalomyelitis, a recognised complication of several vaccines including smallpox, rabies and typhoid, has been reported as a result of influenza vaccine. Then he described a case of a patient in whom meningo-encephalitis developed 12 to 14 days after inoculum with a purified influenza vaccine.

Wells (1971) warned that vaccination against influenza may be complicated by a neurological illness. He presented two cases together with a summary of seven others.

Archives of Neurology published a letter to the editor by Cherington (1977), reporting on locked-in syndrome after swine 'flu inoculation. The author wrote that swine 'flu vaccine was implicated as a possible cause of the Guillain-Barré syndrome.

Encephalomyelitis following bivalent 'flu vaccine was reported in a patient in 1972. The author recently saw a patient with probable

brain stem encephalitis 18 days after receiving swine 'flu vaccine. The clinical findings included the locked-in syndrome and downward gaze which persisted for one month. The patient remained mute.

Weintraub (1977) described a case of paralytic brachial neuritis after swine 'flu vaccine. Paralytic brachial neuritis is a well- defined clinical entity with a typical pattern of signs and symptoms. It may occur after viral infections, use of foreign sera or inoculation. The author maintained that irrespective of the vaccine used, reactions occur. Brachial neuritis is seen frequently and its clinical manifestations may differ slightly, depending on which nerves are involved.

Some authors (Smith, Bellanti and Chanock, 1967) warned that the level of virus neutralising antibody in nasal secretions was a better index of host resistance than was the level of serum antibody.

Despite all these warnings, trials with both killed and live influenza vaccines continued and 'flu vaccines were gradually introduced on the market. It has also been established that children had higher levels of adverse reactions to the 'flu vaccine injections than adults. Hennessy (1969) complained about the antigenic contents of certain influenza virus vaccines. Perkins (1969) discussed problems of bacterial contamination of eggs on which 'flu vaccines are cultured. He stated that, although the Rous inhibitory factor (RIF) viruses (causing cancer in poultry) in eggs were inactivated, there was no data on the effect of having RIF virus nucleic acids in the vaccine.

> "If the price and availability of RIF-free eggs were equal to those of RIF-contaminated eggs, then there would certainly be a greater effort to change to use of RIF-free eggs".

The greatest gaps in the knowledge according to this author were related to the mechanisms of immunity. Perkins also emphasised that although batches of vaccines tested in the United Kingdom were free of pyrogens (substances producing fever) when tested in laboratory animals, they caused as many reactions as the whole-virus vaccine, when given to humans.

Interesting results were obtained and described by Eickhoff and Meiklejohn, (1969). These authors tested a bivalent and polyvalent

Hong Kong influenza vaccines on approximately 1,200 students each on 30 and 31 October 1968. Confirmed cases of Hong Kong 'flu among the vaccinated students began to occur within a week of administration of the vaccines, reaching a peak three weeks later. However, the authors excluded the cases that occurred within a week from the analysis and included only cases that occurred after the 16th of November. This manouvre achieved the impression that the incidence of 'flu in the control group was greater than the incidence in the two vaccine groups. When all cases were included in the analysis there was no difference in the incidence of 'flu in the three tested groups.

Hjordis, Cooney, McMahan, and Grayston (1969) demonstrated in field trials of Hong Kong strain influenza vaccine that the vaccine was ineffective in preventing disease and much more reactogenic than anticipated. There was no true control group since the "control" group was given vaccine against influenza B. This vaccine was shown to produce unacceptable reactions in children and was equally as ineffective in preventing disease as the A2/Hong Kong-strain vaccine.

Besides individual cases of reactions, there were also some quite spectacular outbreaks of illness and deaths caused by the 'flu vaccine, the most famous being the swine 'flu vaccine debacle and the so-called Legionnaires' disease (14-17).

In February 1976 a human infection with a swine influenza virus occurred in five recruits at Fort Dix, a military camp in New Jersey. The antigenic composition of the virus was interpreted as a major antigenic shift and doctors expected a pandemic to occur imminently.

An extensive mass vaccination program was planned; on March 13 the Director of the Center for Disease Control (CDC) presented to the Assistant Secretary for Health the recommendations of the Advisory Committee on Immunisation Practices (ACIP) on a proposed total nationwide vaccination programme. President Ford signed into law the bill authorising $135 million for a comprehensive influenza vaccination programme. From June to September 1976, a complex public-private vaccination delivery system was set up,

reaching its full potential on and after October 1. Many private doctors refused to participate in the program because of the liability implications. By the end of November, various manufacturers had produced some 150 million doses of the vaccine.

Right from the beginning this plan had one major flaw: the new strain showed no capability of epidemic spread. Nevertheless, some 40 million adult people were vaccinated. There followed within four months the occurrence of hundreds of cases of so-called Guillain-Barré paralysis in vaccinees, with tens of deaths. Some 4,000 law suits were lodged seeking compensation for the damage sustained. Some three billion dollars were paid out in compensation.

On 16 December 1976, the Public Health Service elected to place a moratorium on all influenza vaccines pending reassessment of vaccine risks. The moratorium was lifted on 9 February 1977 for all groups at "highest risk of fatal disease from infection" with the then currently prevalent influenza A and B vaccines.

People have short memories. Despite the above tragedy, vaccination against influenza continued and in January 1978-March 1981, during and after the 1978-1979 influenza vaccination campaign, 575 cases of Guillain-Barré Syndrome (GBS) were reported by participating neurologists in the national GBS surveillance system. The incidence was highest in the fifty to seventy four year old age group, with a lesser peak in fifteen to thirty five year olds. Victims experienced respiratory and gastrointestinal illness before the symptoms of ascending paralysis appeared. 67% of the total number of vaccinees reported receiving an A/New Jersey (swine) influenza vaccine in 1976 before being revaccinated two years later.

This means that 67% of Guillain-Barré Syndrome victims experienced the well-documented phenomenon of vaccine sensitisation. They were affected by subsequent injection of the 'flu vaccine two years after previously receiving the initial dose of the swine 'flu vaccine.

In other words, these two and a half thousand victims represented an aftermath of the swine 'flu debacle. However, the authors writing

about the incident (Hurwitz, Schonberger, Nelson, and Holman, 1981; Kaplan, Schonberger, Hurwitz, and Katona, 1983) did not recognise this and did not correlate it with the well-documented phenomenon of vaccine-sensitisation as described in 1901 by Dr Wright and others.

It is quite reasonable to say that the influenza epidemic, which followed unabated in 1979-80 despite the above mass "immunisation" programmes, not only was NOT stopped but was most probably precipitated by weakening and sensitising large numbers of vaccinees to the very illness the 'flu vaccines were purporting to prevent.

Many deaths, described as 'excess deaths' in the elderly and the very young were in fact very important evidence supporting this conclusion. Mass influenza immunisation programmes (just as for measles, mumps and rubella), instead of protecting, actually predispose many people to the disease and make them susceptible through the process of sensitisation to serious and, indeed, fatal outcomes.

In July 1976 a pneumonia-like epidemic occurred among members of the American Legion who had attended a meeting in Philadelphia (Friedman 1978).

There were an estimated 180 cases with 29 deaths. This was only a tip of the proverbial iceberg. Many other sporadic outbreaks occurred all over the United States. In England and Scotland (Bartlett 1979; Macrae et al. 1979) some 84 cases with 18 deaths were reported between January 1976 and September 1978. During 1980-82 some 1300 cases of illness compatible with the Legionnaires' diseases were studied in Spain (Otero et al. 1983). In the autumn of 1992 substantial reports of the incidence of the Legionnaires' disease with many deaths were reported in Australia, especially in Sydney. It was certainly not coincidental that these outbreaks followed a vigorous television advertising campaign encouranging people to take the 'flu shots.

Outbreaks of such deadly diseases as the Legionnaires' disease, synchronous with intensified 'flu vaccine campaigns, are a warning

to all of us that something very wrong is happening as a direct consequence of 'flu vaccine injections. To see the connection, one has only to scrutinise sporadic seasonal outbreaks of Legionnaires' disease in the countries that offer — by quite forceful advertising, especially in autumn and winter — a variety of 'flu vaccines.

Legionnaires' disease, generally believed to be associated with the *Legionella pneumophila* microorganism, is characterised by fever, cough and pneumonia, headache, nausea, vomiting and diarrhoea. Many patients typically become dizzy and disoriented, with loss of short-term memory and hallucinations and become cyanotic, suffer breathing difficulties, develop tremors of the limbs and renal failure.

It is not coincidental that very similar pneumonic (including lung abscesses) and gastrointestinal manifestations were observed in major outbreaks of influenza B and of influenza A to a lesser extent.

If the 'flu vaccine status of the victims of Legionnaires' disease were checked, it is very likely that many, if not all of them, were given a variety of 'flu vaccines shortly before becoming ill. During the latest outbreak of the Legionnaires' disease in Sydney, family members of many victims who died claimed that their relatives were given 'flu injection shortly before they became violently ill (Daily Telegraph Mirror, September 1992).

Despite all these and more reports on ineffectiveness and danger of 'flu vaccines, there are still major efforts to push the vaccines Ohrt and McKinney (1992) discussed the "optimal method" to increase influenza immunisation rates of medical house staff and students. Despite recommendation by CDC that physicians who treat high-risk patients take 'flu vaccines, the reported immunisation rates have remained low, ranging from 2% to 36%.

The main and most quoted reason for the low uptake of influenza vaccine by medical staff was fear of adverse reactions. The majority of persons were also concerned about secondary febrile illness, which might have been due "to frequent reporting of this side effect by patients". This, I hope, speaks for itself.

Perhaps even more dagerous are talks about introducing statutory requirements for vaccination of adults. MMWR (1992; 41 (4): 773-

775) in the editorial note to an article discussing activities to increase influenza vaccination levels in the Unites States state that vaccination programmes for adults have been difficult to implement for at least four reasons, one of which is that *"although statutory requirements exist for vaccination of children, few such requirements exist for adults"*.

The American medical system obviously has not learned from the swine 'flu fiasco.

Hudson (1979) compared the outbreaks of Legionnaires' disease with the bubonic plague in mediæval Europe. There is more similarity between the Black Death epidemics and the Legionnaires' disease than Hudson ever intended to admit or allowed himself to see. The first and most important similarity is the mode of transmission: a flea bite in the Black Death transmission and an injection "bite" in the 'flu (and other) vaccines.

In the 12th century, communities were reduced by the plague to one half or even less, mainly because of poor nutrition and especially vitamin C status, coupled with crowded and unhygienic living conditions. All this resulted in poor and vulnerable immune systems.

The modern plague — "vaccination"— is destroying immune systems in the industrially developed countries. Potent poisons and foreign antigens are being daily introduced by injection (equivalent to a flea bite) into the bloodstream of tiny babies. These are followed by potent immunosuppressive chemicals called, quite ironically, 'medications'.

The next target are the elderly, who are urged by the largely uninformed public media, to take their 'flu shots every year.

The most tragic consequence of vaccination madness, of course, is AIDS. Although all efforts were made to keep hidden the connection between AIDS and smallpox / polio vaccinations, the secret can no longer be kept away from the public.

Coupled with a number of other diseases of immune derangement, the growing epidemic of AIDS may justifiably be attributed to the ignorance, hubris and lack of scientific basis of orthodox medicine.

It is slowly but surely accelerating and within 30 years may reach the "efficiency" of the Black Death in decimating humanity, except that it will be on an even much larger scale than the plagues that occurred close to 700 years ago.

History is certainly repeating itself; humanity has not really become wiser or more scientific for all its technical progress.

## References


1. Smith, J.W.G., 1974.
   Vaccination in the control of influenza. Interim report to the Director of the Public Health Laboratory Service on a collaborative study with the Post Office.
   Lancet; 10 Aug: 330-333.
2. Curphey, T.J., 1947.
   Fatal allergic reaction due to influenza vaccine.
   J Amer Med Ass; **133 (15)**: 1062-1064.
3. Warren, W.R. (Major), 1956.
   Encephalopathy due to influenza vaccine.
   Arch Intern Med: 803-805.
4. Smith, C.B., Bellanti, J. A., Chanock, R. M., 1967.
   Immunoglobulins in serum and nasal secretions following infection with type 1 Parainfluenza virus and injection of inactivated vaccines.
   J Immunol; **99 (1)**: 133-141.
5. Mostow, S.R., Schoenbaum, S.C., Dowdle, W.R., Coleman, M.T., Kaye, H.S, 1969.
   Studies with inactivated influenza vaccines purified by zonal centrifugation.
   Bull WHO; **41**: 525-530.
6. Hennessy, A.V., 1969.
   Problems in determination of the antigenic content of influenza vaccines.
   Bull WHO; **41**: 563-566.
7. Perkins, F.RT., 1969.
   Control of influenza by vaccine, with special reference to experience in the United Kingdom.
   Bull WHO; **41**: 554-555.
8. Eickhoff, T.C., and Meiklejohn, G., 1969.
   Protection against Hong Kong influenza by adjuvant vaccine.
   Bull WHO; **41**: 562-563.
9. Hjordis, M.F., Cooney, M., McMahan, R., and Grayston, T., 1969.
   A field trial of Hong Kong-strain influenza vaccine in Seattle schoolchildren.
   Bull WHO; **41**: 564-566.
10. Schonberger, L.G., Brefgman, D.J., Sullivan-Bolyai, J.Z., Keenlyside, R.A., Ziegler, D.W., Retailliau, H.F., Eddins, D.L, and Bryan, J.A., 1979.
    Guillain-Barré syndrome following vaccination in the national influenza immunization program.
    Am J Epidemiology, **110 (2)**: 105-123.
11. Langmuir, A.D., 1979.
    Guillain-Barré syndrome:the swine influenza virus vaccine incident in the United States of America,1976-77: preliminary communication.
    J Roy Soc Med; **72**, Sept: 660-669.

12. Cherington, M., 1977.
Locked-in syndrome after "Swine flu" inoculation.
Arch Neurol; **34**, April: 258.
13. Weintraub, M.I., 1977.
Paralytic brachial neuritis after swine flu vaccination.
Arch Neurol; **34**, Aug: 518.
14. Hurwitz, E.S., Schonberger, L.B., Nelson, D.B., and Holman, R.C., 1981.
Guillain-Barré syndrome and the 1978-79 influenza vaccine.
N Engl J Med; **304**: 1557-1561.
15. Kaplan, J.E., Schonberger, L.B., Hurwitz, E.S., and Katona, P., 1983.
Guillain-Barré syndrome in the United States, 1978- 1981:
Additional observation from the national surveillance system.
Neurology; **33**, May: 633-637.
16. Anonymous, 1984.
Influenza and the Guillain-Barré syndrome.
Lancet, 13 Oct: 850-851.
17. Wright, A.E.,1901.
On the changes effected by antityphoid inoculation in the bactericidal power of the blood; with remarks on the probable siginificance of these changes.
Lancet, 14 Sept: 715-723.
18. Otero, M.R., Anda, P., Fernandes, M.V., Casal, J., and Najera, R., 1983.
Legionnaires' disease in Spain.
Lancet; 2 April: 759.
19. Ohrt, C.K., and McKinney, W.P., 1992.
Achieving compliance with influenza immunization of medical staff and students.
J Amer Med Ass, **267(10)**: 1377-1380.
20. Rosenberg, G.A., 1970.
Meningoencephalitis following an influenza vaccination.
N Engl J Med; **283 (22)**: 1209.
21. Wells, C.E.C., 1971.
A neurological note on vaccination against influenza.
Br Med J; **3**: 755-756.
22. Friedman, H.M., 1978.
Legionnaires' Disease in Non- Legionnaires.
Ann Int Med; **88**: 294-302.
23. Hudson, R.P., 1979.
Lessons from Legionnaires' Disease.
Ann Int Med; **90**: 704-707.

# 9

# SMALLPOX: Is it really eradicated?

Even though orthodox medicine admits that no vaccines are as effective as they would wish them to be, and eradication of any disease is not always realistic, there is one apparent exception: smallpox. Orthodox medicine claims that it eradicated smallpox by vaccination.

In 1967 the World Health Organisation (WHO) initiated a world-wide eradication campaign against smallpox. In that year some 131,000 cases of smallpox were reported to WHO from 42 countries. It was understood this number might represent only 5% of total cases.

The smallpox eradication campaign was carefully orchestrated by WHO; a Global Commission for the Certification of Smallpox Eradication was established by the Director-General of WHO to appraise progress towards global eradication of the disease.

In 1970 only 21 of the original 42 countries reported having any cases of smallpox; by the late seventies and early eighties, smallpox was considered all but eradicated and many countries dropped the requirement of smallpox vaccination certificates for travellers.

Arita and Breman (1979) reported on evaluation of WHO's smallpox vaccination policy. They wrote that the World Health Organisation's campaign to eradicate smallpox had reached a turning-point.

*"Interhuman transmission of smallpox, which continued for more than 3000 years, appears to have come to an end on 26 October 1977, when the world's last known case developed his rash in Merca, Somalia."*

The disease was pronounced officially eradicated on 8 May 1980.

It is rather interesting that according to Arita and Gromyko (1982), an important benefit of global eradication of smallpox was the

*"recommendation of the World Assembly that smallpox vaccination should be discontinued in all countries. By March 1982, 150 of the 158 WHO Member States had officially terminated their smallpox vaccination programmes."*

The authors then continued:

*"despite these considerations, some reports of complications caused by smallpox vaccination have been published recently ... In some countries vaccination of recruits to the armed services has continued; these recruits will occasionally transmit vaccinia infection to unvaccinated persons, and inevitably, some of the complications will be fatal."*

It is certainly of special importance that the main benefit of smallpox eradication was discontinuation of vaccination.

Side effects and ineffectiveness of smallpox vaccination have been the main smallpox issue discussed in medical papers for a long time. In 1928 The British Medical Journal (14 January) published an article written by Dr R.P. Garrow showing that the fatality rate among the vaccinated cases of smallpox in England and Wales in 1923 and 1926, in those over 15 years of age, was higher than among the unvaccinated.

This stirred a lively written discussion, published on 21 January 1928.

Among the most interesting comments are those of Percy Stocks, who considered waning immunity the reason for increased mortality among those over 15 years old. He also reasoned that, when comparing the expected deaths from all causes in various age groups with those from smallpox, there was no evidence of any significant difference either way between the mortality rates in vaccinated and unvaccinated. This was supposed to imply that, although dying within 2 months of contracting smallpox, victims could have died of other causes. Fred E. Wynne tried to explain the five-fold higher smallpox mortality rate in the over 15 year old vaccinees during 1923

and 1926 smallpox epidemics by waning immunity. He also issued a scalding of R.P. Garrow for broadcasting in the medical press an assertion

*"which he must be aware will be quoted, on his authority and without context, by the antivaccinist press. This kind of action can do nothing but handicap his colleagues who are engaged in combating the present epidemic of small-pox, with its serious burden on the public funds, the loss of wages involved, and the damage to industry, quite apart from the detriment to public health, which in my recent experience is becoming more serious as the infection is passed through the human medium."*

C. Killick Millard reasoned that, although the mortality rate in those vaccinated is five times that of the unvaccinated, the case mortality is so trifling in either group that it at once arouses suspicion of a *"... catch somewhere. The 'catch' is that, under the term 'small-pox', we are including two varieties of the disease so utterly different as regards their mortality that as, for statistical purposes, they are two distinct diseases, and it is most misleading to include them together under the same heading ...there should be little practical difficulty in keeping the statistics for the two varieties separate, because I doubt if there has been a single outbreak of small-pox, say in the past ten years, where there was any real doubt as to which variety of small-pox was being dealt with."*

Then he continued that when the figures are sorted out into (1) variola major and (2) variola minor, *"we find that we have under (1) an insignificant minority of, say, under one hundred cases of variola major with most of the 13 deaths, and an overwhelming majority of nearly 10,000 cases of variola minor with practically no deaths.*

*The few deaths that have been attributed to variola minor are usually due to some intercurrent complication, and, if these be deducted, we find that variola minor is for practical purposes, a non-fatal disease in vaccinated and unvaccinated persons alike."*

Then Millard wrote this quite unbelievable statement:

*"As regards the cases and deaths in the small variola major group, it so happens that the few isolated outbreaks which have occurred in*

*the years in question have been among adults rather than among children. But adults in most parts of the country are still, on the whole, a vaccinated class; therefore it is not very surprising that many of these cases have been in vaccinated persons.*"

L.A. Parry was, according to his own words, educated to believe that smallpox was a disease which was contracted by unvaccinated persons and was with them a terrible and fatal malady. In the vaccinated, a case was a rare event of disease which was trifling and of no importance. Parry summarised the questions raised by Dr Garrow as follows:

1. How is it that small-pox is five times as likely to be fatal in the vaccinated as in unvaccinated.
2. How is it that, as the percentage of people vaccinated has steadily fallen (from about 85 in 1870 to about 40 in 1925), the number of people attacked with variola has declined *pari passu* and the case mortality has progressively lessened? The years of least vaccination have been the years of least small-pox and of least mortality.
3. How is it that in some of our best vaccinated towns — for example Bombay and Calcutta — small-pox is rife, whilst in some of our worst vaccinated towns, such as Leicester, it is almost unknown?
4. How is it that something like 80 per cent of the cases admitted into the Metropolitan Asylum Board small-pox hospitals have been vaccinated whilst only 20 percent have not been vaccinated?
5. How is it that in Germany, the best vaccinated country in the world, there are more deaths in proportion to the population than in England - for example, in 1919, 28 deaths in England, 707 in Germany; in 1920, 30 deaths in England, 354 in Germany. In Germany, in 1919 there were 5,012 cases of small-pox with 707 deaths; in England in 1925 there were 5,363 cases of small-pox with 6 deaths. What is the explanation?
6. It is possible to explain the lessened incidence and fatality of small-pox on the same grounds as the lessened incidence and

fatality of other infectious fevers - namely, as due to improved hygiene and administrative control?

He finished his letter with these words:

*"There are just a few points in connection with the subject which are puzzling me, and to which I want answers. I am in doubt, and I want to know the truth. Will some of the experts help me?"*

The "experts" represented by the journal commented:

"*We think that Dr Parry, in his desire for enlightenment, would have been wiser not to introduce assumptions of fact into the framework of his questions*".

This discussion aired a number of very important issues which are still relevant to smallpox (and other) vaccination issues.

The editorial comment accused Dr Parry of introducing assumptions of fact into his questions, while he was referring to well-known facts. Indeed, his was the only letter which was factual, logical and to the point which would still stand up to scrutiny today. However, the contributions of the other authors reflected the same questionable reasoning still used today by some proponents of vaccination.

In their fervor to defend vaccination practice, proponents of vaccination made a number of 'Freudian slips'. Perhaps the best of them is the statement of Millard (*op.cit.*) to the effect that, since most adults are vaccinated, a number of cases of variola major will inevitably occur in vaccinated subjects.

Isn't the smallpox vaccine supposed to protect against contracting the disease, especially variola major? And if it does not, isn't it a failure of the vaccine to protect?

Let us now look at some more published information on side-effects of smallpox vaccination. Spillane and Wells (1964), in their voluminuous account of post- vaccinal encephalomyelitis following smallpox vaccination, stated that this group of serious adverse reactions to smallpox vaccine has only been recognised after 70 years of compulsory vaccination in England. In the sixties the high incidence of smallpox encephalomyelitis was recorded in Hol England and Germany.

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In one small town in Holland the incidence was sometimes 1 in 63 people vaccinated (Dixon 1962). In 1942 there were 3 outbreaks of smallpox in Scotland, beginning in Glasgow in May, in Methilhill, Fife in August, and in Edinburgh in October. Mass vaccination at the time of each of the three Scottish outbreaks was followed by varying numbers of reported cases of vaccination encephalitis.

In 1951-1958 there were 60 cases of encephalitis in Great Britain, 51 following primary vaccination and 36 in infants. Of the 25 deaths, 21 occurred in infants. At this time the reporting system was not working very well, so the real number of cases will probably never be known.

In the reported immunisations the phenomenon of sensitisation was documented because of accelerated and hyperacute reaction to subsequent inoculations. The neurological reactions ranged from encephalitis to epilepsy, polyneuritis and multiple sclerosis.

The time of onset of encephalitic symptoms ranged from 24 hours to several days, with clustering around the same critical days as observed with other vaccinations. Time and again, the evidence proving the phenomenon of sensitisation appeared in publications by various authors. De Vries (1960), Dixon (1962) and Dick (1962) have suggested that the mortality rate from vaccination was greater in those who were primarily vaccinated in infancy and then revaccinated in later years. The onset of encephalitis was often sudden, explosive and characterised by convulsions or status epilepticus. Hemiplegias and aphasia were common but spinal involvement did not seem to occur. The cerebrospinal fluid was often quite normal, but under increased pressure. The electro-encephalogram showed asymmetrical slow waves, often with focal abnormality. Recovery has not always been complete. Cerebral oedema and vascular lesions occurred often.

The authors noted the lack of detailed information on neurological reactions to Jennerian vaccination. There was no clear directive about notification of cases of post-vaccinal encephalitis and any other complications involving the central nervous system. However, the authors felt that better reporting may throw light on the causation of

"spontaneous" diseases such as multiple sclerosis and acute disseminated encephalomyelitis.

Indeed, Miller *et al.* (1967) described 9 patients who developed (5 cases) or experienced exacerbation of (4 cases) multiple sclerosis after primary or secondary vaccinations, mostly against smallpox, in England and Poland. The onset of symptoms was recorded as at 7, 12, 48 or ' a few' hours or 7-14 or 'a few' days. The authors also quoted a number of other authors who described similar cases after vaccination against smallpox, typhoid, paratyphoid, tetanus, poliomyelitis and tuberculosis (BCG) and after injections of anti-diphtheric serum and gammaglobulins.

These authors described the onset of symptoms of multiple sclerosis within hours or days (2-14) or 3 to 12 weeks. However, the interval between BCG or rabies vaccination and the onset of multiple sclerosis was sometimes measured in years. It was also noteworthy that local reactions at the site of injection were often inconspicuous in people who later developed multiple sclerosis.

Lane *et al.* (1969) described complications of smallpox vaccination based on 572 cases in the United States. They quoted Neff *et al.* (1967) and others who found that complications occurred most frequently in children under one year of age. There were nine deaths; four were caused by postvaccination encephalitis, four associated with vaccinia necrosum, and one by eczema vaccinatum. Morbidity and mortality rates were highest in infants, with 112 complications, and five deaths, eczema was more severe for contacts than for vaccinees. They stated that many cases of complications went unreported and that their estimates must be considered minimal. The United States Public Health Service and other groups subsequently recommended that primary vaccination in the United States be deferred to the second year of life.

A few years after smallpox was declared eradicated by WHO, smallpox vaccine was used by physicians in an attempt to treat recurrent genital herpes simplex virus — with disastrous results. Injecting smallpox vaccine not only did not heal the original condition, it caused development of recurrent herpes lesions at the

vaccination site. Mintz (1982) reported on one such case of the inappropriate use of smallpox vaccine.

Belief in the effectiveness of smallpox vaccination was quite ingrained, despite published or generally-known cases of adverse reactions, including deaths, and the observed ineffectiveness to prevent the disease. The incidence of smallpox diminished thanks to better sanitation and generally better nutrition and vitamin status, but proponents of vaccination kept claiming credit for this situation.

At a conference in Kampala in 1971, D.A. Henderson stated that only two major epidemic centres remained in Africa, the Sudan and Ethiopia, and there was *"every likelihood that these areas will soon be cleared."*

Then a remarkable story started unfolding. According to Anonymous (1973), in 1970 several patients with illnesses at first indistinguishable from smallpox were discovered in allegedly smallpox-free forest areas of West and Central Africa. Between October 1970 and May 1971 a virus was isolated from some patients in West Africa which was later identified as monkeypox.

This virus was not new; it was isolated in 1958 by von Magnus *et al.* (1959) during an outbreak of smallpox-like vesicular disease in captive monkeys in Copenhagen. Foster (1959) described the first six cases in humans (five in children) of this disease in West Africa (four in Liberia, one in Sierra Leone and one in Nigeria). None of the six was considered vaccinated against smallpox. Three of the patients were severely ill, but all recovered. Three of the cases occurred in an area which had been free of smallpox for at least a year, though 38% of local residents did not have a history of scarring from smallpox or vaccination. Two of the patients with monkeypox had occasionally consumed freshly-killed monkeys and others were observed playing with the viscera of freshly-killed monkeys.

Serological studies on monkey sera failed to produce any convincing evidence of poxvirus infection in monkeys. One additional and severe case with an eruption more typical of smallpox was found in a rural forest area of east Nigeria.

Further virological studies in the Congo found evidence of monkey pox viruses in one clinically normal cynomolgus monkey and one normal chimpanzee, but failed to locate any significant source of monkeypox virus infection. *"If it wasn't for the smallpox surveillance program, the true identity of these apparently rare cases might never have come to light."*

The monkeypox story continued. Gispen *et al.* (1976) reported on monkeypox-specific antibodies in human and simian sera on the Ivory Coast and Nigeria. These serological results provided evidence for a monkeypox reservoir in wild monkeys. The virus was indistinguishable by laboratory methods from variola virus.

Arita and Henderson (1976) discussed monkeypox and whitepox viruses in West and Central Africa.

Smallpox eradication has been defined by the WHO Expert Committee on Smallpox Eradication as *"... elimination of clinical illness caused by variola virus."* The Committe added: *" ... since there is no human carrier state of epidemiological importance and no recognised animal reservoir of the disease, the absence of clinically apparent cases in man may be assumed to signify the absence of an naturally occurring smallpox."*

The authors continued: *"Evidence of the absence of animal reservoir has been strengthened over the last ten years by the fact that greatly intensified epidemiological surveillance has not documented a single outbreak of smallpox in a smallpox-free area except when introduced by man from a known smallpox-infected area."*

Between 1966 and 1980 there were more and more cases of monkeypox reported in the tropical rain forest area in Zaire, Liberia and Sierra Leone. Of the twenty patients, two were vaccinated against smallpox, and four died. So, smallpox vaccination did not protect and the disease was quite severe in that 20% of its victims died. In Nigeria, of the twelve unvaccinated family contacts only one became infected.

During the epidemiological search, 94 children with facial scarring caused by the disease during the past three years were

located. All except two showed smallpox vaccination scars. It was concluded that facial scarring was caused by chickenpox (!?!) Monkeypox virus was isolated in 13 cases; in the remaining seven cases, the poxvirus was detected by electron microscopic examination or by the presence of poxvirus antibody in sera. In patients 7 and 8 (Nigeria) and 9 (Ivory Coast), sera taken five years after the onset of disease showed monkeypox specific antibody.

In 1975 a special survey was conducted in Sierra Leone, Liberia, the Ivory Coast and Nigeria almost five years after smallpox cases last occurred in these countries, in order to detect any additional monkeypox cases. These studies failed to detect any additional cases, despite the fact that only 40-70% of the village population had been vaccinated.

Serological surveys conducted in areas of West and Central Africa revealed the presence of antibodies to smallpox in a number of animals. It was not possible to distinguish the recovered four variola-like poxvirus isolates by currently available laboratory tests. All produced small, whitish pocks on the chorio-allantois of chick embryos, "*similar to those produced by variola virus.*" [Arita and Henderson (1976), p. 350]. Despite this, they were named "whitepox viruses". All four whitepox viruses have been isolated from monkey or rodent specimens collected in Equateur Province (Zaire) where the eight cases of monkeypox in humans have been found.

It is certainly very interesting that there were many cases of smallpox before 1970, while after that date all suspected cases were "caused" by monkeypox or other viruses.

However, an important study was published in 1963 (Bedson *et al.*) which clearly differentiated 23 strains of variola virus in various parts of Tanganyika during 1961, 1962 and 1963. Their behaviour under laboratory tests suggested the existence of a third variety of variola virus, beside the known variola major and variola minor (alastrim), although the differences were not clear-cut.

Clinically, the illness they were associated with was indistinguishable from the mild form of smallpox (variola minor or alastrim).

In the seventies, several published reports dealt with smallpox-like viruses found in animals. Baxby (1972) conducted laboratory studies on three camelpox virus strains isolated in Iran which showed themselves to be members of the variola/vaccinia subgroup of poxviruses, extremely closely related to variola virus.

Baxby (*op. cit.*) considered this very important for the eradication program of the WHO which *"... can only be successful in the absence of a non-human reservoir for smallpox virus."* He wrote that there are several poxviruses affecting both humans and animals, of which monkeypox virus, although clearly distinguishable from variola virus by simple laboratory tests, can cause clinical smallpox in humans. The second group is represented by the so-called "white" poxviruses, found in healthy monkeys, which in laboratory tests are indistinguishable from variola virus.

The author also stated that this camelpox virus was indistinguishable from the standard *"international reference strain of variola major virus"* and certain strains of variola, isolated from east Africa (Tanzania). All test viruses neutralised rabbit and rhesus monkey antisera to virtually the same extent, indicating a very close serological affinity between all three viruses. The author concluded that only further laboratory testing and field work could determine whether camelpox represents a non-human reservoir for variola virus.

In 1979, Marennikova published important evidence that rodents may be poxvirus carriers. Poxvirus antibodies were detected in the kidneys and/or lungs of rodents in Europe and Africa. Also, some poxviruses, isolated from Turkmenistan rodents or white rats near Moscow, appeared very close to cowpox viruses, while those from Zaire rodents were identical to variola-like (whitepox) viruses found earlier in monkeys in Zaire.

Mounting evidence of the existence of an animal reservoir of variola-like viruses became embarrassing in light of the fact that the smallpox eradication campaign was undertaken only because of alleged good epidemiological evidence that there was no non-human (animal) reservoir of variola virus.

Dumbell and Kapsenberg (1982) seemed to resolve the problem by demonstrating that all the so-called "whitepox" viruses were isolated in laboratories that also handled variola viruses, and were the result of cross-contamination from cultures inoculated with the variola virus from Indian (Vellore) specimens.

Simultaneously, however, Bedson (1982) found that all five "whitepox" viruses examined behaved like smallpox viruses in the new (enzyme) test and consequently they must be considered genuine variola viruses.

In the meantime, smallpox outbreaks continue. The Australian Dr Weekly (17.7.1992) reported that smallpox killed 130 people in a remote area of southern Laos. Most victims were children younger than 14 years. Perhaps the most interesting statement in this report is that health education and sanitation are still unknown to these people.

Further recent oubreaks continue in Somalia and other parts of Africa and India.

Smallpox has not been eradicated. It has a potent animal reservoir. We don't even have to go into complicated discussions about monkeypox. The very smallpox vaccine itself is based on the cowpox virus.

## Bubonic Plague Model of Vaccination Irrelevance

Perhaps the best example of the irrelevance of vaccination in the dynamics of disease is the bubonic plague. Bubonic plague has a perfect, large and ubiquitous animal reservoir — rats and fleas. Because of this, one would imagine it nearly impossible to eradicate this disease, yet we are all witnesses to the disappearance of plague to same degree as smallpox.

Only sporadic oubreaks still occur: Morbidity and Mortality Weekly Report (31 August, 1984) described a case of bubonic plague with secondary plague pneumonia in a 35-year-old veterinarian in Claremont (California), only 10 miles from the area where a humam case of plague was identified in 1979. No secondary cases occurred, despite 61 persons having had face-to-face contact with this person. Only one cat, which had an illness with symptoms compatible with

those usually seen in pneumonic plague, was identified. The cat died. One cat was infected by this animal, was treated with antibiotics and survived. However, several dead rodents were found in the area in which the case cat lived. Several dogs, cats and coyotes were bled from that area and found to have antibodies to *Yersinia pestis*.

Since 1959 four veterinarians and one veterinary assistant have had confirmed plague infections; one of the veterinarians died. There have been reports of plague in dogs and cats. Since 1975, 32 of 188 human plague patients have had plague pneumonia. Despite high rates of exposure, no secondary cases were detected.

Since 1978, additional plague infections have been diagnosed, especially in California. The most interesting moral from these reports is that, despite a complete and total absence of vaccination, even during the outbreaks, bubonic plague is self limiting and does not blow up into epidemics such as were experienced in past centuries. Obviously there are potent factors which prevent these epidemics from happening.

The same rationale applies to smallpox. There is no evidence to support the claim that vaccination had any impact on the virtual disappearance of smallpox from all developed and most underdeveloped countries, despite the well-documented fact that there is an efficient animal reservoir. Changing lifestyles, including better sanitation and changes in dietary habits, should be credited with this situation, not vaccination. Vaccination programmes reached only a small percentage of persons; when they did, they were demonstrably ineffective in preventing the disease as documented above. Blind belief in the vaccination myth only prevented us from learning much earlier the real dynamics of smallpox prevention and treatment, as well as that of other diseases.

Today, the smallpox vaccination saga is history. However, routine childhood vaccination against smallpox was not abandoned without some last attempts by vaccinators to argue the "case" for continuing vaccination of babies. When Lane and Millar (1969) published their article demonstrating that *"The benefits of routine childhood smallpox vaccination no longer outweigh its risks, and consideration*

*should be given to its discontinuation"*, Krugman and Katz (1969) maintained that *"Any proposal to curtail the smallpox vaccination program should be subjected to critical evaluation in the light of this historical perspective."*

The "historical perspective" was that, in spite of the availability of an "effective" vaccine, smallpox continued to be a critical problem in the United States during the early part of the twentieth century. Only a few lines later, the same authors wrote that during the 15 years the United States has been free of smallpox, importations from Europe have resulted in 723 cases, with 111 fatalities.

Because the risk of postvaccinal encephalitis appears to increase with advancing age after the first year of life *"... Cessation of routine vaccination of children will expose them to the increased hazards of the procedure in later years, because of military service, hospital service or travel abroad."*

American Journal of Epidemiology in 1971 published three articles: the case for continuing "routine" childhood smallpox vaccination, the case for abolishing routine childhood smallpox vaccination and possible alternatives to routine smallpox vaccination in the United States.

Samuel Katz (1971) introduced his case for continuing "Routine" childhood smallpox vaccination by writing:

*"The title of this paper was an assigned one and contains one word to which may be attributed some significant portion of our current problem and concerns. No immunisation procedure should be* 'routine', *least of all one which is intended to protect against a disease absent from our nation for more than 20 years.* 'Routine' *evokes too many mental images (nightmares) of smallpox vaccine applied indiscriminately to the upper arms of children who may then return to homes where other family members have exfoliative dermatitis; or the vaccination of individuals who have a disease-caused, or medication-induced, deficiency in cell-mediated immunity. Unless proper enquiry is made to divulge such clear contraindications to vaccine, the rates of eczema vaccinatum and vaccinia necrosum will not be reduced.*

'Routine' *also implies an unthinking adherence to patterns which may have become fixed, and even antiquated, without the flexibility imparted by continuing surveillance and judicious evaluation."*

Katz then continued by praising Kempe, Neff, Lane, Millar and others who warned about uncritical retention of old routines for smallpox vaccination. As an example he mentioned that smallpox vaccination *"is now deferred until the second year of life"*. However, this change was long in coming, partly due to reluctance to change.

Katz pointed out that infants are by far not the risk group for contracting smallpox. In fact, hospital- associated personnel are at highest risk of contracting smallpox as secondary cases. However, infants and school children, by virtue of being seen more by medical doctors, were uniquely available for immunisation. Katz added that availability cannot, by itself, be a justification today for vaccination.

How correct and topical were these wise words — by a provaccinator! And how topical these words still are!

John Neff presented a case for abolishing routine childhood smallpox vaccination in the United States. In his commissioned paper, Neff (1971) stated that smallpox vaccination in the United States was associated with a significant morbidity and mortality. He continued by saying that the wrong age group was overvaccinated; absolute protection against smallpox lasted no more than three years after vaccination and, after 25 years, there seemed to be very little protection left. Recent large hospital surveys showed that 65% of adults had not been vaccinated in 15 years and it would seem that compulsory vaccination of children may not have the desired result of producing an immune adult population in a high risk group. Instead, it produced many complications without producing herd immunity in an appropriate adult population group.

Neff also stated that the extraordinary freedom from smallpox in the United States was not the direct result of the policy of routine childhood immunisation, but the result of the diminishing incidence of smallpox worldwide, that travellers have been well-immunised and, more importantly, come from those socio-economic classes in which smallpox is becoming increasingly rare.

This last statement is especially important since, in the so-called developed countries, the quality of life, state of nutrition and general living conditions (including housing) improved so much that a vast majority of people now belong to what previously would have been classified as the well-to-do socio-economic class.

Well, now we all know what happened when smallpox vaccination was discontinued: exactly nothing. No epidemics of smallpox followed, not even due to potential importations via a vastly intensified movement of the travelling public.

Equally, there will be no epidemics of these diseases when routine diphtheria and tetanus vaccination of infants is discontued. When whooping cough vaccination is stopped, as it was in Sweden, Italy and the former West Germany, the incidence of pertussis will diminish or will continue at the same rate as at present, but with some important differences. The age distribution will return to normal. Only about 10% of whooping cough cases will occur in babies below the age of one and the rest in those children several years old who can take the disease well. The tragic toll of vaccine-induced diseases will stop abruptly and the bizarre modern entity of cot death, as we know it now, will disappear (as it vanished from Japan after 1975).

## References


1. Arita, I., and Gromyko, A., 1982.
   Surveillance of orthopoxvirus infections, and associated research, in the period after smallpox eradication.
   Bull WHO; **60** (3): 367-375.
2. Arita, I., 1979.
   Virological evidence for the success of the smallpox eradication programme.
   Nature (London); **279**: 293- 298.
3. Garrow, R.P., 1928.
   Fatality rates of small-pox in the vaccinated and unvaccinated.
   Br Med J; 14 Jan: 74.
4. Stocks, P., 1928.
   Fatality rates of small-pox in the vaccinated and unvaccinated.
   Br Med J; 21 Jan: 115.
5. Wynne, F.E., 1928.
   Fatality rates of small-pox in the vaccinated and unvaccinated.
   Br Med J; 21 Jan: 115.
6. Millard, C.K., 1928
   Fatality rates of small-pox in the vaccinated and unvaccinated.
   Br Med J; 21 Jan: 115-116.
7. Parry, L.A., 1928.
   Fatality rates of small-pox in the vaccinated and unvaccinated.
   Br Med J; 21 Jan: 116.
8. Spillane, J.D., and Wells, E.C., 1964
   The neuorology of Jennerian vaccination - a clinical account of the neurological complications which ocurred during the smallpox epidemic in South Wales in 1962.
   Brain; **87**: 1-44.
9. Dixon, C.W., 1962.
   Smallpox, London.
   J & A. Churchill Ltd.
10. Dick, G.W.A., 1962.
    Scientific Proceedings; Symposium on virus diseases. Thirteenth Annual Meeting of the British Medical Association, Belfast.
    Br Med J; 2: 319.
11. de Vries, E., 1959.
    Postvaccinal perivenous encephalitis.
    Amsterdam, Elsevier Publ Co.
12. Miller, H., Cendrowski, W., and Shapira, K., 1967.
    Multiple sclerosis and vaccination.
    Br Med J; 22 April: 210-213.
13. Lane, M.J., 1969.
    Complications of smallpox vaccination, 1968.
    N Engl J Med; **281** (22): 1201-1208.

14. Neff, J.M., Levine, R.H., Lane, J.M., *et al.*, 1967.
Complications of smallpox vaccination, United States, 1963.
II. Results obtained by four statewide surveys.
Pediatrics; **39**: 916-923.
15. Mintz, L., 1982.
Recurrent Herpes simplex infection at a smallpox vaccination site.
J Amer Med Ass; **247 (19)**: 2704-2705.
16. Arita, I., and Breman, J,G., 1979.
Evaluation of smallpox vaccination policy.
Bull WHO; **57 (1)**: 1-9.
17. Anonymous, 1974.
Smallpox target zero?
The Lancet; 23 February: 295-296.
18. von Magnus, P., Andersen, I. K., Petersen, K.B., and Birch-Andersen, A., 1959.
A pox-like disease in cynomolgus monkeys.
Acta Pathologica et Microbiologica Scandinavica; **46 (2)**: 156-176.
19. Gispen, R., Brand-Saathof, B., and Hekker, A.C., 1976.
Monkeypox-specific antibodies in human and simian sera from the Ivory Coast and Nigeria.
Bull WHO, **53**: 355-360.
20. Anonymous, 1973.
Monkeypox.
Br Med J; 6 Jan: 3-4.
21. Bedson, H.S., Dumbell, K.R., and Thomas, W.R.G., 1963.
Variola in Tanganyika.
Lancet; 23 Sept: 1085-1088.
22. Arita, I., and Henderson, D.A., 1976.
Monkeypox and whitepox viruses in West and Central Africa.
Bull WHO; **53**: 347-353.
23. Baxby, D., 1972.
Smallpox-like viruses from camels in Iran.
Lancet; 18 Nov: 1063-1065.
24. Marennikova, S.S., 1979.
Field and experimental studies of poxvirus infection in rodents.
Bull WHO; **57 (3)**: 461-464.
25. Dumbell, K.R., and Kapsenberg, J.G., 1982
Laboratory investigation of two "whitepox" viruses and comparison with two variola strains from southern India
Bull WHO; **60 (3)**: 381-387.
26. Bedson, H.S., 1982.
Enzyme studies for the characterization of some orthopoxvirus isolates.
Bull WHO; **60 (3)**: 377-380.
27. Lane, M.J., and Millar, J.D., 1969.
Routine childhood vaccination against smallpox reconsidered.
N Engl J Med; **281 (22)**: 1220-1224.

28. Krugman, S., and Katz, S.L., 1969.
Smallpox vaccination. Editorial.
N Engl J Med; **281 (22)**: 1241-1242.
29. Katz, S.L., 1971.
The case for continuing "routine" childhood smallpox vaccination in the United States.
Am J Epidemiol; **93(4)**: 241-243.
30. Neff, J.M., 1971.
The case for abolishing routine childhood smallpox vaccination in the United States.
Am J Epidemiol; **93(4)**: 245-247.
31. Benenson, A.S., 1971.
Possible alternatives to routine smallpox vaccination in the United States.
Am J Epidemiol; **93(4)**: 248-252.

# 10

# NON-SPECIFIC STRESS SYNDROME and VACCINATION

## A Tribute to Hans Selye

Ever since any vaccines were administered to humans, those who administered them were quite aware of reactions to vaccine injections, ranging from mild local reactions through to serious systemic reactions with permanent residual disability and death. Temporal association of vaccine injections and reactions was quite accepted as an indication of causal relationship.

Medical doctors were also quite aware that the reactions, especially of systemic nature, were often delayed. However, this has apparently changed since vaccination of infants became mandatory in the United States and/or a subject of national policies of "prevention" of infectious diseases; these policies, of course, made vaccine profits become quite substantial.

Infants cannot talk, and, because symptoms of illness in small babies are often quite subtle, they too often escape attention. This is made worse by the reality of the natural non-specific dynamics of symptomatology of response to noxious substances and injuries and insults of any kind in mammals, including humans.

In 1936 Dr Hans Selye published his first paper **"A Syndrome Produced by Diverse Nocuous Agents"**. This was the first presentation of the *"syndrome of response to injury as such"*.

This means that living organisms have a general non-specific reaction-pattern, a general defence mechanism with which they meet damage caused by a great variety of potential disease-producers.

Selye spent his whole life researching and defining the non-specific stress syndrome, or General Adaptation Syndrome and its pathology and symptomatology. The pathology of the Non-Specific Stress Syndrome includes enlarged adrenal cortex (as a result of increased adreno-cortical activity), intense atrophy of the thymus, the spleen and all lymphatic structures, signs of petechial bleeding into the lungs, thymus, pericardium and other internal organs, and intrathoracic cavity, ulceration of the lining of the stomach and duodenum, disappearance of eosinophil cells from circulating blood, a number of chemical alterations in the constitution of the body fluids and tissues, changes in the viscosity and clotting properties of the blood (the presence of unclotting, liquid, blood) and signs of derangements in body temperature control (overheating or underheating).

The clinical symptoms of the Nonspecific Stress Syndrome include general feeling of malaise, nausea, coated tongue, reflux, otitis media, upper respiratory tract infections, runny nose, sticky eyes, clamminess, deranged (elevated or below normal) body temperature, rash, tenderness of the liver and spleen, diffuse pains and aches in the joints, gastro-intestinal disturbance, with loss of appetite and weight, diarrhoea and/or constipation.

Selye (1937) recognized three stages in the Non-Specific Stress Syndrome:

1. The alarm stage, when the body is acutely affected and mobilises all its defences and the corticoid activity rises sharply;
2. The stage of resistance, when the body is at a maximum capacity to resist the insult and
3. The stage of exhaustion when all defences have been exhausted and the organism may succumb. One can also consider the stage of exhaustion a point of crisis which is mostly followed by recovery.

Generally, an organism cannot maintain a continuous state of alarm. If the organism is confronted with an insult so damaging that continuous exposure to it is incompatible with life, then death follows within hours or days.

Microprocessor Cotwatch records of breathing of adults suffering chronic fatigue syndrome show that, if the insult is long-lasting and the body cannot rid itself of it, a stage of repeating alarm periods lasting 4-7 days, with some relaxation of the stress in between, will develop; this explains why the organisms may suffer chronic fatigue.

In the alarm stage, the cells of the adrenal cortex discharge the hormone-containing granules into the blood stream. The density of blood increases. There is a loss of weight. During the stage of resistance, the adrenal cortex accumulates reserves of secretory granules. The blood is diluted and the body weight returns to normal. Under continued effect of an insult, the acquired adaptation is eventually lost. The symptoms of exhaustion are in many respects similar to those of the alarm stage and the organism may succumb. However, the stage of exhaustion may also represents another effort to mobilise defences and mostly appears as a point of crisis after which most organisms recover. A stress affected organisms may, however, die in any stage of the Non-Specific Stress Syndrome.

Selye's original research centered on endocrine activity. Exposure to stress affects hormonal activity. This is especially well-manifested in individuals' reactions to vaccine injections. The inability to adapt to an insult caused by vaccines leads to quite classic signs of illness as described repeatedly by Selye. Under certain conditions an excess production of the hormone called mineralocorticoid desoxycorticosteron - DOC - causes brain lesions. When this is coupled with vascular lesions, also characterictic of the Non-specific Stress Syndrome, it may lead to the destruction of large parts of the brain. Behaviourally, these destructive changes manifest themselves in irritability and destructive aggression in affected individuals. Insomnia or excessive somnolence are another effect of hormonal imbalance due to an insult. This may be associated with feeling of depression with suicidal tendecies. Other effects are

manifested in hightened perception and dissociation of the ego and the "id".

Certain breakdown products of adrenaline can cause hallucinations and delirium as a result of high fever or burns. Further, DOC-like hormones can cause spells of periodic paralysis in genetically predisposed individuals. Very similar paralysis occurrs in persons in whom an adrenal tumour produces an excess of the DOC-like hormone: aldosterone. In monkeys, given excess of DOC, this type of paralysis was accompanied by intense epilepsy-like attacks or convulsions. The condition in these animals was alleviated by withdrawal of salt and aggravated by increased intake of the same. Other experiments produced evidence that large amounts of DOC produced a kidney disease in rats very similar to human nephrosclerosis. Sexual derangements were produced in animals exposed to prolonged stress. During stress, the sex glands shrink and become less active in proportion to the enlargement and increased activity of the adrenals. This can be explained by a shift in pituitary hormone production; the pituitary has to produce so much of adrenocorticotrophic hormone (ACTH) to maintain life that it must cut down on the production of other hormones. A good example is a slow-down or complete cessation of growth in young animals during stress. Lactating female animals and humans may stop producing milk during stress. In men both the sexual urge and sperm production are diminished.

The gastrointestinal tract is especially sensitive to general stress. Signs of irritation and upset of the digestive organs occur in any type of stress. Loss of appetite is one of the first symptoms of just being ill. This may by accompanied by vomiting, diarrhoea or constipation. Gastric or duodenal ulcers can develop virtually overnight. Ulcerative colitis is known to develop following a prolonged frustration. Many of the metabolic diseases are largely diseases of adaptation. Excessive weight loss or weight gain belong in this category. Diabetes is another classic example. Diabetes is not always due to an insufficiency of insulin production. It is often caused by an excessive production of such adaptive hormones as

ACTH, somatotrophic hormone (STH, a growth hormone) and various anti-inflammatory hormones. Hypoglycæmia is often associated with chronic fatigue. Glucagon plays an important role in this process. It is an anti-insulin substance of the pancreas - a "stress hormone". A sudden increase in blood glucagon concentration has been demonstrated both in animals and in man under the influence of a number of stressors.

Paul Lemonde demonstrated that during the alarm reaction there is a marked liver (hepatic) insufficiency. This is of a particular importance because corticoids are normally metabolised and destroyed in the liver. The importance of the liver in the resistance to a great variery of toxic substances has become particularly obvious with the discovery of catatoxic steroids. These substances induce a variety of detoxifying enzymes capable of inactivating many damaging substances which circulate in the blood. My comment: Vitamin C, as a general detoxifier, must inevitably play an important role in helping the body to handle stress. That would explain why vitamin C reserves are depleted during stress and any illness. It would also explain the immediate beneficial effect of vitamin C in any illness and even as a curative agent in cancer.

The most nonspecific breakdown in resistance to stress is shock. A person affected by intense insult, injury or poisoning, may develop a syndrome characterised by a substantial and life threatening fall in blood pressure, by temperature fall way below normal and the affected person may die without any sign of specific injury to any one of the vital organs. This makes shock an eminently nonspecific stress condition. Autopsy usually reveals the characteristic triad of the alarm reaction: adrenocortical enlargement, thymicolymphatic atrophy and bleeding erosions into the cardio-respiratory and gastrointestinal tracts. We are looking here at the breakdown of body defences in general, rather than at the specific action of any one particular pathogen. Injections of foreign protein may cause a severe reaction - allergic anaphylaxis or hypersensitivity. The body reacts to these foreign substances by launching an intense inflammatory response. Rheumatic and rheumatoid diseases in man belong in the

same category. This is demonstrated by the beneficial effect of administration of anti-inflammatory hormones on these diseases. Focal infections (inflammations) can lead to generalised diseases like arthritis, asthma, allergies etc.

Let's now have a look how reactions to vaccine injections reflect the reality of the Non-specific Stress Syndrome as recorded by the microprocessor breathing monitor.

## Microprocessor records of the Non-specific Stress Syndrome in Breathing

Our involvement in the study of vaccine reactions in babies is based on studies of breathing of babies with a sensor-pad, battery operated true breathing monitor - Cotwatch. The sensor- pad is placed under the mattress on a firm base; monitoring is completely non-invasive and non-inhibiting since nothing is attached to the baby's body or its garments. The electronics process all movements picked up by the sensor and recognises breathing, heart-beat and other movements. Only breathing delays provoke the alarm. Because of the 100% separation of heart-beat and breathing, Cotwatch gives meaningful information on breathing. The use of Cotwatch revealed the existence of the Stress-induced Breathing Pattern associated with the Non-specific Stress Syndrome.

Initially this discovery was based on the alarms reported and recorded by parents using Cotwatch to monitor their babies' breathing. The alarms occurred characteristically in clusters of several shorter alarms within about 15 minutes and at critical hours and either preceded the onset of symptoms of illness (a common cold, any upper respiratory tract infection, otitis media, teething or followed any insults ranging from fatigue, exposure to cigarette smoking, handling by visitors to vaccine injections.

There was an internal consistency between what we called warning alarms and the nature of the insult. When the child was incubating an illness, the alarms preceded the onset of the symptoms, while when the child was exposed to fatigue or vaccinated, the alarms followed. As a rule, a child incubating a common cold had a cluster or two of

warning alarms a night or two before the onset of symptoms, while a child had five to ten clusters of warning alarms per night for two weeks before it went down with full- blown symptoms of pneumonia.

All babies had long periods of time without any alarms at all.

To make the data collection on babies' breathing more objective, Leif Karlsson developed a microprocessor-based breathing monitor, which records and stores information on breathing and reproduces it in the form of computer printouts.

There are a number of ways to present the microprocessor recorded information on breathing. The program can be modified according to the requirements of the researcher. In our studies two basic presentations were used: the so-called "raw" record of breathing consisting of a series of histograms hour by hour and turned at 120 degrees along the "y" axis and with "x" axis tilted 30 degrees (figure 1). The second presentation includes 24 hour summaries of weighted apnea-hypopnea density in vertical columns (figures 2-4). The basic information is processed so that only events (apneas and hypopneas) of 6 to 20 sec duration are displayed. The events from 6 to 15 seconds are mostly apneas, while events over 15 seconds are mostly hypopneas. The events are weighted logarithmically so that even a few events of longer duration are displayed.

The alarms with Cotwatch indicate the existence of what we called the Stress-induced Breathing Pattern which is a low volume breathing (only abouty 5% of the volume of non-stressed breathing) and associated with stress. It occurs characteristically in clusters within about 15 minutes at a time, in sleep and at critical hours.

The microprocessor and standard Cotwatch are encased in the same type of box and the sensor pad is also the same. The difference is in the electronics of the units.The microprocessor unit can be used in parallel with the standard unit. It stores all information on breathing and processes it so that events (apneas and hypopneas) are assembled in the form of histograms. After downloading, the unit is reset, and the data collection runs for a minimum of three weeks or longer depending on the minimum time unit selection. This system enables a non- stop monitoring indefinitely.

Both units are fully battery operated. Longitudinal records of breathing of babies after vaccination revealed a number of facts. First of all, these records revealed a pattern of flareups of stressed breathing following quite clearly the general pattern of critical days. Figure 2 shows records of breathing of 2 babies after the third and first DPT injections over 18 days. It also compares these with the pattern of day-by-day distribution of deaths of 41 babies who died after DPT injections as published by Coulter and Fisher (1991), Walker et al. (1987), and Bernier, et al. (1982). The fourth graph down the line is a schematic illustration of the three stages of Selye's Non-specific Stress Syndrome based on the dynamics of adreno-cortical activity in individuals exposed to non-specific stress syndrome.

First of all, records of breathing of the two babies after DPT injections reveals the existence of the three stages of non-specific stress syndrome: the alarm stage, the stage of resistance and the stage of exhaustion, or, in these cases, rather the point of crisis. Records of breathing of the two babies were placed in parallel with each other because they show that the amplitude of the flareups of stressed breathing differs depending on the intensity of the insult in relation to the resistance of the baby. The amplitude of the flareups indeed differed markedly. The maximum weighted apnea-hypopnea density (WAHD) values in baby two (first DPT) was about 2500, while the maximum WAHD value in baby one (third DPT injection) was around 14 000. However, the days on which the flareups of stressed breathing were recorded were generally remarkably similar. The maximum flareups were recorded on day 2, then around day 5 and the 16th day was quite clearly a point of crisis after which in both babies the flareups of stressed breathing subsided and the babies started recovering. However, the three phases of the non-specific stress syndrome may repeat themselves virtually indefinitely; this is seen quite clearly in the chronic fatigue syndrome.

The maximum level of stress in breathing after DPT injections or other insults varies greatly from child to child. Some babies have delayed reactions. While initially (during the first two weeks) there may only be slight increases in the stress levels in breathing, later on,

starting on the 15-16th or 20-25th days, the level of stress in breathing starts climbing up, reaching three or four fold levels when compared with the stress levels in breathing during the first two weeks.

The distribution of 41 babies who died after administration of DPT clearly follows the dynamics of stressed breathing in the two babies. This indicates that DPT and death of these 41 babies were causally related. Quite clearly, more babies died on days when babies experience flareups of stressed breathing after DPT injections. These days are critical days and their number in any individual child depends on the intensity of the insult and the individual's reaction to the insult. It is a well-established fact that injections of any antigen sensitise the individual so that further injections of the same antigen elicit more response (Wright (1901), Goodall (1918), Darcy (1966)). Our microprocessor records are quite consistent with this well-documented observation.Further support for our data comes from the work by Watson et al. (1981). These authors looked at the symptoms displayed by a number of babies three weeks before they died of cot death (case babies) and controls, which suffered the same, non-specific symptoms, but recovered. The full line represents case babies, the dotted line controls. Again, it is quite clear that day 16 represented a point of crisis, after which the case babies got worse and died on day 21 after the onset of symptoms, while the controls got better and recovered. It is also quite instructive that the overall dynamics of the distribution of observed and recorded symptoms followed the dynamics highly comparable with our records. The fact that the case babies displayed fewer symptoms was taken by Watson *et al.* (*op. cit.*) as reflecting the quality of both parental and medical care, resulting in the symptoms being played down.

**Vaccine reactions and the Non-specific Stress Syndrome**

So far all vaccine recipients whose breathing was recorded with the microprocessor Cotwatch showed reactions to vaccine injections. There are individual differences in the amplitude of flareups of stressed breathing, which, however, occur on much the same, critical, days.

Clustering of either the onset of adverse reactions or deaths around the days when any baby has flareups of stressed breathing is well-documented in medical literature dealing with adverse reactions or deaths occurring after vaccine injections.

Landrigan & Witts (1973) presented very interesting data on the day-by-day onset of neurological disorders following the live measles virus vaccination. Their figure 1 shows quite clear clustering of the onset of either febrile convulsions or other disorders around days 3, 7-10, 13, 15, 18 and 25. It is quite clear that the live virus measles vaccine elicited a response quite characteristic of the Non-Specific Stress Syndrome. Pollock et al. (1984) list 7 cases of cot death within 6 weeks after vaccination. The three deaths in the DPT group happened on the fourth, 20th and 37th days and the other four in the DT group at the second, fifth, 37 and 40th days. The child who died 20 days after the second adsorbed DPT had been unwell since vaccination with attacks of high pitched crying 3 or 4 times a day for 10 days, followed by a period of excessive sleepiness. The high-pitched crying recurred 4 days (on the sixteenth day) before the baby's death on the 20th day. Three of those who died did not have any visible post-vaccination symptoms of note. Two babies in the DT group died from repiratory infection. One child had a convulsion on the third day after DPT injection and one baby on the fifth day after the DT injection. Myoclonic epilepsy developed in one child about one months after the first DPT dose and in two babies in the DT group; in one 8 days after and in the other 6 weeks after the injection. The 3rd child had a convulsion with transitory hemiplegia 5 weeks after the injection. Many cases of screaming, limpness, pallor, glazed eyes etc. were reported by these authors within 24 hours after both DPT and DT vaccine administration.

Hirtz et al. (1983) deal with 40 convulsions suffered by 39 babies after a variety of vaccine injections. They demonstrate clustering of these reactions on days one, two, 3 to 6, and 7 to 10. Most babies who died after vaccine injections show pathology compatible with the pathology of the Non-Specific Stress Syndrome. Petechiated lung, thymus, pericardium and intrathoracic cavity are among the

most common autopsy findings. The presence of liquid, unclottting blood and signs of derangements of body temperature control are often puzzling the cot death researchers [Goldwater et al. (1990)]; Goldwater 1992, Denborough et al 1982).

As a matter of fact, all publications dealing with either death or onset of adverse reactions to a great variety of vaccines bring evidence of clustering of these around the days when we recorded flareups of stressed breathing in babies after vaccine injections. There is not a shadow of doubts that vaccines are killing and maiming babies.

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## References


1. Selye, H., 1936.
   A syndrome produced by diverse nocuous agents.
   Nature, July 4, **138**: 32.
2. Selye, H., 1937.
   Studies on adaptation.
   Endocrinology; **21 (2)**: 169-188.
3. Selye, H., 1978.
   The Stress of Life.
   McGraw-Hill Book Co.
4. Coulter, H.L., & Fisher, B.L., 1991.
   A shot in the dark.
   Avery Publishing Group Inc.
5. Walker, A.M., Hershel, J., Perera, R.S., Thompson, R.S., and Knauss, T.A., 1987.
   Diphtheria-Tetanus-Pertussis immunisation and sudden infant death syndrome.
   Amer J Publ Health; **77 (89)**: 945-951.
6. Bernier, R.H., Frank, J.A., and Dondero, T.J., 1982.
   Diphtheria-tetanus toxoids-pertussis vaccination and sudden infant deaths in Tennessee.
   J Pediatrics; **101 (3)**: 939-984.
7. Hoffman, H.J., Hunter, J.C. and Hasselmeyer, R.N., 1982.
   SIDS and DPT.
   Proc. 17th Immunol. Conf. May 18-19, Atlanta: 79-88.
8. Griffin, M.R., Ray, W.A., Livengood, J.R., and Schaffner, W., 1988.
   Risk of sudden infant death syndrome after immunization with the diphtheria-tetanus-pertussis vaccine.
   N Engl J Med; **319**: 618-623.
9. Wright, A.E., 1901.
   On the changes effected by antityphoid inoculation in the bactericidal power of the blood; with remarks on the probable significance of these changes.
   The Lancet; Sept 14: 715-723.
10. Darcy, D.A., 1966.
    Enhanced response of an "acute phase" serum protein to repeated tissue damage in the rat.
    Br J Pathol; 480-487.
11. Watson, E., Gardner, A.S., and Carpenter, R.G., 1981.
    An epidemiological and sociological study of unexpected deaths in infancy in nine areas of Southern England.
    Med Sci Law; **21 (2)**: 89-98.
12. Landrigan, P.J., and Witte, J.J., 1973.
    Neurologic disorders following live measles-virus vaccination.
    JAMA; **223 (13)**: 1459- 1462.

13. Pollock, T.M., Miller, E., Mortimer, J.Y., and Smith, G., 1984.
Symptoms after primary immunisation with DPT and with DT vaccine.
The Lancet, July 21: 146-149.
14. Hirtz, D.G., Nelson, K.B., and Ellenberg, J.H., 1983.
Seizures following childhood immunizations.
J Pediatrics; **102 (12)**: 14-18.
15. Goldwater, P.N., Williams, V., Bourne, A.J., Byard, R.W., 1990.
Sudden infant death syndrome: a possible clue to causation.
Med J Aust; **153**: 59-60.
16. Goldwater, P.N., 1992.
Reappraisal of the SIDS enigma: an epidemiological ands clinico-pathological approach.
J Paediatr Child Health; **28**: Suppl 1 S 21-25.
17. Denborough, M.A., Galloway, G.J., and Hopkinson, K.C., 1982.
Malignant hyperpyrexia and sudden infant death.
The Lancet; 13 Nov: 1068-1072.
18. Stanton, A.N., 1984.
Overheating and cot death.
The Lancet; 24 Nov: 1199-1201.
19. Dunne, K.P., and Matthews, T.G., 1988.
Hypothermia and sudden infant death syndrome.
Arch Dis Child, **63**: 438-440.

# 11

# ADVERSE EFFECTS OF VACCINES VALIDATE HOMEOPATHY

Extensive study of medical literature, dealing with side effects of vaccines, revealed some interesting and unexpected facts which prompted me to look up the basic concepts of homeopathy. The results of this study left me with no other option but to conclude that the observed and documented adverse effects of vaccines unwittingly provide evidence for the validity of basic concepts of homeopathy.

Homeopathy is a system of pharmacological medicine. Homeopathic doctrine is a set of rules for administering precise medicines, prescribed for each patient individually, to make him or her well.

The principles of homeopathy have been tested in practice for some 180 years and their scientific validity has been conclusively demonstrated.

Homeopathic doctrine has eight elements [Coulter (1980)]:

1. The Reactivity of the Organism to External Stimuli; the disease is an expression of the adaptive effort of the whole organism.
2. The Biphasic Action of Medicines.
3. The Provings.
4. Ultrasensitivity of the Organisms to the Similar Medicine.
5. The Infinitessimal Dose.
6. The Single Remedy.
7. The Law of Similars.
8. Hering's Law of Appearance of Symptoms and of chronic disease.

**1. The Reactivity of organisms to the External Stimuli.**

Disease is part of the adaptive effort of the whole organism. The symptomatic changes have priority over structural and pathological changes.

All living organisms respond to environmental stimuli. Life is a continuous adjustment and adaptation of the internal relations of the organism to the external ones. Living beings maintain a dynamic equilibrium, in harmony with their changing environment, by automatically adjusting through their endocrine and nervous systems. Healthy organisms adapt promptly and adequately.

Hans Selye (1978) called this process a non-specific stress syndrome or General Adaptation Syndrome. It is a universal, general response to any stimulus, insult or injury or noxious substance of any kind, and, according to Selye's experiments, is largely independent of the nervous system. It is innate to all body tissues, to every cell. Sometimes the balancing act does not succeed outright and the organism becomes sick. It will send out signals of disease — symptoms. Symptoms represent the totality of the patient's response to a given insult.

Homeopathy considers all diseases general and does not speak of local diseases or local treatment. Selye's non-specific stress syndrome, or General Adaptation Syndrome (GAS), is the most appropriate modern expression of the reality behind this principle of homeopathy. Selye recognised that in the GAS all organs are affected and are participating in the body's reaction and balancing act. In contrast, the bulk of allopathy is mostly unaware of this and speaks of target organs (stomach, lungs, bowel, etc.) instead of recognising the general (systemic) nature of disease as homeopathy and Selye have done.

According to the teachings of homeopathy, the natural process and the stages of patient's illness must be respected by the doctor, otherwise there is risk of turning acute conditions chronic. Skin rashes in particular, by developing external local diseases, are nature's own way of healing the internal disease which threatens the vital organs .

Natural infectious diseases of childhood are an essential natural process for achieving the balance in a fast-growing organism. An attempt to suppress these diseases by vaccines, hyperimmune serum globulins, antibiotics and antipyretics causes much more serious illnesses, like asthma, chronic bronchitis, eczemas, allergies and even cancer, by pushing the diseases and imbalances deep into the body where they attack the internal organs. The enormous increase in the incidence of these diseases, and the appearance of a great variety of immunodeficiencies plaguing humanity, and especially children, since the introduction of mass vaccination programmes, is well documented in medical literature. The list includes childhood common variable immunodeficiency, late-onset immunoglobulin deficiency, idiopathic "acquired" agamma-globulinemia, acute haemolytic anæmia related to diphtheria-tetanus vaccination, chronic fatigue syndrome, eosinophilic leukaemoid reaction, multiple sclerosis, cancer, tuberculosis, T-cell leukaemia, lymphadenopathy, systemic lupus erythematosus, AIDS — it seems almost endless.

However, some of these diseases may have another ætiological factor, namely contamination of vaccines by animal viruses, in particular retroviruses, which are notorious in their possible link to AIDS, leukaemia and cancer (Kyle 1992).

Ronne (1985) documented an association between a negative history of measles, exposure in early life (possibly injections of immune serum globulin after exposure) without developing proper rash, and development of immunoreactive diseases: sebaceous skin diseases, degenerative diseases of bone and cartilage, and certain tumours. He suggested that the presence of measles virus specific antibodies at the time of acute infection interferes with development of specific cytolytic reactions and enables the intracellular measles virus to survive the acute infection.

In my opinion this mechanism is also valid for measles vaccines, which stimulate production of measles specific antibodies; however, without the full expression of the general inflammatory process, proper natural immunity is not developed. This interferes with natural processes and exposes the recipient of the measles vaccine to

later, and much more serious, autoimmune diseases. The well-documented enormous increase in childhood cancer is a serious reminder that we must reconsider the safety of medications administered by orthodox medicine, and especially the efficacy and safety of vaccines used in mass immunisation of our children.

The appropriate homeopathic remedies commonly bring out many symptoms previously suppressed — skin eruptions suppressed by previous application of various ointments; catarrhal complaints, suppressed by antibiotics, may be followed by rheumatic problems. The return and discharge of the original symptoms is then the best sign the patient is being really cured.

**2. The Biphasic Action of Medicines.**

Hahnemann, the founder of homeopathy, established that a drug administered to a sick or healthy person gives rise to two successive symptom patterns. The first is called the primary symptoms and represents the immediate effect of the drug on the organism. The second, secondary symptoms, is the reaction of the organism to the immediate drug effect. The secondary symptoms are more or less the opposite of the primary symptoms.

The relationship of the primary and secondary symptoms is to some extent a function of the dose size. If a large dose is given to a patient, the primary symptoms are too violent, while the secondary reaction is also disordered. If a small dose is given, the primary effect is minimal and is soon succeeded by the secondary (disappearance of the patient's symptoms). Hahnemann also observed that if the primary symptoms of the medication, when administered to a healthy person, are identical with the symptoms of the sick person, the secondary symptoms of the medicine would act to remove the patient's symptoms and thus restore him to health.

The allopathic medicine expresses the Hahnemann's discovery as the so-called "Arndt-Schulz Law": every drug has a stimulating effect in small doses, while large doses inhibit and very large doses kill. Karl Koetschau in the early twenties further refined this law as the "type effect hypothesis". According to this hypothesis, there are

3 typical effects of medicinal drugs, depending upon a dose:1. with small doses a stimulant effect. 2. With moderate doses, an effect is first stimulating, but then depressive, with the patient eventually returning to normal 3. With large doses a very brief stimulating effect is followed by a severely depressive effect and may lead to death. With certain substances the reverse effect occurs (the stimulant effect will be depressive).

Joseph Wilder (the early fifties) proposed a reformulation of the Arndt-Schulz and Koetschau Laws. His Law of Initial Values is as follows:Not only the intensity, but also the direction of a response of a body function to any agent, depend to a large extent on the initial level of that function at the start of the experiment. The higher this "initial level" the smaller is the response to function-raising and the greater is the response to function- depressing agents. At more extreme initial levels there is a progressive tendency to "no response" and to "paradoxic reactions", i.e. reversal of the usual direction of the response.

Wright (1901) described the effect of various dosages of the typhoid vaccine on the bactericidal properties of the blood. The typhoid inoculations were performed on surgeons-on-probation and the doses of primary inoculations were in almost every case greatly reduced. In certain instances, a negative phase of diminished bactericidal power of the blood was recorded in the period immediately following inoculation. It happened especially in patients who reacted to the vaccine more violently than others.

The bactericidal power of the blood declined from a comparatively high average before inoculation to a point below which it could not be quantitatively estimated. The negative phase continued in these cases for at least three weeks after inoculation. Before the fifth week a very marked positive phase of increased bactericidal power has established itself. When such a small dose of typhoid vaccine was used which did not provoke a marked constitutional reaction, a positive phase of increased resistance could have occurred in some persons without formation of a negative phase as early as 24 hours after inoculation. Administration of large doses, provoking a

constitutional reaction, was considered inadvisable. Wright (op.cit.) also observed that there appears to be a definite limit beyond which the bactericidal power of the blood cannot be increased by inoculation with sterilised cultures of the typhoid bacilli, usually corresponding to the level before inoculation. When Wright injected a larger dose to himself, the pronounced negative phase was not followed by a definite positive phase. Only after 2 months, his blood regained temporarily the same bactericidal power as was measured a week before inoculation. He concluded that the occurrence or non-occurrence of a negative phase of diminished bactericidal power, subsequent to inoculation, and its duration, is determined by the quantity of the vaccine and by the "resisting powers" of the inoculated persons.

### 3. The Provings.

Hahnemann tested (proved) hundreds of remedies on himself. The philosophy behind this rule is that a healthy person, given a remedy, will develop certain symptoms. The remedy will support a healing process of a sick person sufferring the same symptoms. Medical doctors should test all remedies on themselves. Tests on animals are much less valuable because animals can't tell us how they feel. One of the best exercises of this kind would be, for the doctors administering vaccines to inject themselves with the same vaccines as routinely administered to babies (and in doses in a proper proportion to body weight). They would soon know how babies feel and what effect the vaccines have on them. Wright (1901) certainly tested the typhoid vaccines on himself.

### 4. Ultrasensitivity of the Organism to the Similar Medicine.

Hahnemann established that the patient suffering an illness is ultrasensitive to the medicine which causes his symptoms in a healthy person. Koch (1891) rediscovered hypersensivity when he demonstrated that normal animals tolerated large amounts of tuberculin, while tuberculous animals reacted violently even to very

small doses, some dying within hours. This phenomenon occurs when a person is injected with a poison, vaccine, foreign protein or practically any foreign substance. Instead of becoming immune to the substance, the person will be sensitised to it. This rule is well documented by the observed fact that children react more violently to subsequent vaccine injections. Indeed, vaccinated people and children suffer the so-called hyperimmunisation after repeated injections of various vaccines or immunoglobulins. Hyperimmunisation can be fatal (Bishop et al. 1966).

In 1960 Cox reported that animals, immunised with several low potency inactivated viral or rickettsial vaccines, responded to challenge with an infection of increased severity. Grayson et al (1961) noted an accentuated pattern of disease in previously vaccinated persons who were experimentally given inoculations of a live trachoma agent in the eye. Quite regularly, over the last thirty years, several groups of investigators reported the occurrence of an atypical pattern of naturally acquired measles (or after vaccine injections subsequent to the initial vaccination) often several years after the administration of an inactivated or live measles virus to children.

Smith (1967) described a febrile illness accompanied by pneumonia in experimentally infected recipients of a killed *Mycoplasma pneumoniæ* vaccine, and who failed to produce detectable antibody. Others (Kapikian et al. 1969, Fulginiti et al. 1969 and many others) reported an unusually severe respiratory disease in infants and young children, developing natural infections with the measles or syncytial respiratory viruses, after vaccination with formaldehyde-inactivated vaccines. These observations demonstrated that injections of viruses, whether attentuated or live, often sensitise the recipient, and result in an accentuated pattern of disease upon natural exposure or revaccination.

The sensitisation process applies to all foreign proteins or noxious substances injected into the blood stream. The Medical Journal of Australia (1959) published an article by Anonymous on repeated snake bites. The article quotes Parrish and Pollard (1959) who

concluded that there was no evidence of acquired immunity in persons after repeated snake bites. Instead, a number of persons bitten repeatedly developed allergy (hypersensitivity) to the snake venom.

Osterholm et al (1988) evaluated the efficacy of *Hæmophilus influenzæ* b polysaccharide vaccine in children in Minnesota and concluded that vaccination with Hib vaccine had no effect in preventing Hib type disease. In another publication (Daum et al 1989), it was demonstrated, that there was an increased incidence of invasive Hib disease within a week or so in children vaccinated with the vaccine.

Despite discontinuing pertussis vaccination in 1979, Sweden trialed two Japanese acellular vaccines in 1986-87 (Storsaeter et al 1987). The trial was concluded without recommendation to introduce pertussis vaccination "...because of concern about the possibility of an increased rate of death from invasive disease cause by encapsulated bacteria in vaccine recipients; efficacy based on the definition used to diagnose pertussis during the trial, was also lower than expected." (Hinman & Orenstein 1990). Indeed, the Swedish health authorities withdrew the application for licensure of the Japanese acellular pertussis vaccine (Anonymous 1989).

During the trial, out of 2847 children given either of the two vaccines, eleven developed invasive infections and 4 died; this was unacceptably high, compared with the estimated incidence of one (Ad Hoc Group for the Study of Pertussis Vaccine, 1988).

This is hardly surprising knowing that the highest incidence of invasive infections in children occurs between 2 to 6 months of age; this is the age when babies are routinely given the first 2 to 3 DPT injections. The Swedish trial clearly demonstrated that the Pertussis vaccine indeed sensitises to invasive bacterial infections, especially the ubiquitous *Hæmophilus influenzæ* b and other commensals. The seriously lowered resistance due to DPT injections is a direct cause which facilitates these serious and life threatening infections. The well-known ineffectiveness of the pertussis vaccine (also confirmed by this and other Swedish trials) is a serious warning that DPT

vaccination should be discontinued. Pertussis became a mild disease (Taranger 1982), and the incidence of pertussis in Sweden and other countries, not vaccinating against pertussis, is the same as in the countries that continue vaccinating. After Japan increased the vaccination age to two years, the cot death entity has disappeared, but the rate of adverse reactions to the vaccine in two year olds remained the same (Cherry et al. 1988).

Hypersensitivity reactions to tetanus, rubella and other vaccines are also well documented (Benn & Alstead 1931, Blumstein & Kreithen 1966, Cunningham 1940, Holliday & Bauer 1983).

Serum reaction or serum sickness is a reaction of body to foreign protein. The dosage of serum has a noticeable influence on the incidence and severity of reactions. Previous injections sensitise the recipient so that the subsequent injections cause reactions in more individuals and their severity in the same individuals is increased (Goodall 1918a, b, and many others).

Serum reactions follow certain time dynamics. The first signs of a reaction can be delayed but, interestingly, always occur on what can be called critical days. Vaccine not only cause reactions similar to serum sickness, but also the dynamics of their appearance follows the same pattern. Records of breathing of babies with a microprocessor breathing monitor (Karlsson & Scheibnerova 1991), before and after DPT injections, revealed the existence of the Stress-induced Breathing Pattern, consisting of episodes of low volume breathing. The SIBP is characterised by flareups of low volume breathing in clusters of several shorter episodes within about 15 minutes at certain (critical) hours and on certain (critical) days. These are the same days as the critical days observed in serum reactions. The Hippocratic writings present a doctrine of the critical days. The seven day period of critical days is more potent than others. Next is the four-day period (half of the seven day period). The seven-day periods are estimated as follows: the first two weeks are days 1 through 7 and 8 through 14. The week three starts on day 14, so that the 3 week period ends on day 20. That's why day 40 and not

42 is critical, similarly the 60th and 80th, and not the 63rd or 84th days [Coulter (1975)].

The clear evidence of the validity of critical days in our records of breathing after vaccination or other insults, adds special importance to the temporal association between DPT injections and cot death, seen in computer printouts of records of breathing of babies after vaccination. These are an important evidence of the causal relationship between injections of vaccines and adverse reactions, including deaths (Karlsson & Scheibnerova 1991). Just as reactions to serum can be delayed and can occur from one to 60 (or more) days after administration, so do reactions to vaccines, including deaths. There is absolutely no justification for only following the onset of possible adverse reactions (including deaths) after vaccine injections for 48 hours or a couple of weeks. The onset of adverse reactions can be delayed in the same fashion as has been documented in serum reactions, and adverse reactions to vaccines, in adults.

### 5. The Minimum or Infinitessimal Dose.

Hahnemann discovered a means of "separating the structural content of a chemical form from its associated chemical mass". The homeopathic high dilutions form part of the area of research dealing with the effect of physical field phenomena on solvents. Water crystallises in a particular pattern depending on barometric pressure. This pattern reproduces itself when the ice is melted and refrozen at a lower pressure. This is another important evidence for a highly scientific basis of homeopathy.

If we want to immunise at all, we should use homeopathic vaccines, nosodes, which are high potency remedies produced by scientific ultradilution of vaccines. However, even then, vaccination should remain the choice of the individual or parents of small children.

### 6. The Single Remedy.

Hahnemann never administered more than one remedy at a time. He reasoned that it would be impossible to predict the effect of several drugs administered simultaneously. Clemens et al (1992) demonstrated that "Concurrent administration of PRP-T vaccine with DPT either in the same syringe or at different sites, interfered with antipertussis responses to a primary series of immunizations". They were not certain of the clinical significance of this antagonism, but they cautioned against adding new vacines to the existing immunisation regimens.

### 7. The Law of Similars.

Hahnemann observed that substances, causing certain illness in large doses, will, in ultradiluted form, enhance the curative process in a sick person, affected by the same substance, and/or sufferring the same symptoms, as the substance causes in a healthy person. Certainly, the idea of vaccination is close to the homeopathic law of similars. However, the homeopathic remedies used in homeopathic immunisation (nosodes) differ very much from the allopathic vaccines in that:

1. Their size is much smaller (ultradilution);
2. Only one nosode is administered at a time (Golden, 1990).

If we want to vaccinate at all, we should consider using judiciously the homeopathic nosodes in selected cases, where the prevention of a disease is preferable to allowing the child to contract the natural disease.

The dangers of allopathic vaccines are well documented in medical literature. Side effects of vaccines are very similar to the adverse effects of the individual infectious illnesses. Mumps or measles meningitis are triggered by mumps and measles vaccines as amply documented in medical literature (Landrigan and Witte, 1973; Gray and Burns, 1989a, b; Anonymous, 1992).

**8. Hering's Law of the Movement of Symptoms.**

According to this law, when the disease passes from an acute to a chronic form, the symptoms move from the surface of the body to the interior, from the lower part of the body to the upper part, and from less vital organs to the more vital ones. This is also true, in part, about the movement of symptoms in an acute disease. Under correct homeopathic treatment, and when the disease progresses in a natural and beneficial way, the symptoms move from the more vital organs to the less vital, from the upper part of the body to the lower, from the interior to the skin and from the interior to the extremities.

In typical measles, rash appears on the forehead, trunk and then moves to extremities. The pattern of atypical measles is characterised by high fever, pneumonia with pleural effusions, and an atypical distribution (hands and feet) of a severe haemorrhagic rash and greatly increased mortality.

It is quite clear that atypical measles brings evidence of the validity of the essence of Hering's Law. The measles vaccines (both live and attenuated) not only sensitise the vaccinated children to the measles virus, they push the measles virus deep into the body, where it attacks the vital organs, like the lungs. Atypical mumps in vaccinated children has also been described (Gunby, 1980).

Perhaps the time has come to accept homeopathy for what it is: the most modern and scientific medical system. The orthodoxy at present is not only lacking a philosophy of illness and health, but the drugs are administered haphazardly and without proper scientific understanding of their effects.

## References.


1. Coulter, H.L., 1980.
   Homeopathic Science and Modern Medicine.
   North Atlantic Books, Berkeley, California.
2. Selye, H., 1978.
   The Stress of Life.
   McGraw-Hill Book Co. Toronto. 515pp.
3. Kyle, W.S., 1992.
   Simian retroviruses, poliovaccine, and origin of AIDS.
   Lancet; 339: 600-601.
4. Ronne, T., 1985.
   Measles virus infection without rash in childhood is related to disease in adult life.
   Lancet; Sept. 5: 1-5.
5. Wright, A.E., 1901.
   On the changes effected by antityphoid inoculation in the bacterial power of the blood; with remarks on the probable significance of these changes.
   Lancet, Sept. 14: 715-723.
6. Koch, R., 1891.
   Forsetzung der Mittheilungen ueber ein Heilmittel gegen Tuberculose.
   Deutsche Medizinische Wochenschrift **17**: 101-102.
7. Bishop, W.B., Carlton, R.F., and Sanders, L.L., 1966.
   Diffuse vasculitis and death after hyperimmunization with pertussis vaccine.
   New Engl J Med; **274 (11)**: 616-619.
8. Cox, H.R., 1960.
   The present status of polio vaccines.
   Ill Med J; **118**: 160-168.
9. Grayston, J.T., Wang, S.P., Lin, H.M., Tai, F.H., Khaw, O.K., and Woolridge, R.L., 1961.
   Trachoma vaccine studies in volunteer students of the National Defence Medical Center. II. Response to challenge eye inoculation of egg grown trachoma virus.
   Chinese Med J; **8**: 312-318.
10. Smith, E.W.S., and Haynes, R.E., 1972.
    Changing incidence of *Hæmophilus influenzæ* meningitis.
    Pediatrics; **50 (5)**: 723- 727.
11. Kapikian, A.Z., Mitchell, R.H., Chanock, R.M., Shvedoff, R.A., and Stewart, C.E., 1969.
    An epidemiologic study of altered clinical reactivity to respiratory syncytial (RS) virus infection in children previously vaccinated with an inactivated RS virus vaccine.
    Am J Epidemiol; **89**: 422-434.
12. Fulginiti, V.A., Arthur, J.H., 1969.
    Altered reactivity to measles virus antigens in recipients of killed measles virus vaccine.
    J Pediatr; **75**: 609-616.

13. Anonymous, 1959.
Repeated snake bite.
Med J Aust; Oct. 31: 653.
14. Parrish, H.M., and Pollard, C.B., 1959.
Effect of repeated poisonous snake bites in man.
Am J Med Sci; **237**: 277-286.
15. Osterholm, M.T., Rambeck, J.H., White, K.E., et al., 1988.
Lack of efficacy of Haemophilus b polysaccharide vaccine in Minnesota.
J Amer Med Ass; **260 (10)**: 1423-1428.
16. Daum, R.S., Sood, S.K., Osterholm, M.T., et al., 1989.
Decline in serum antibody to the capsule of *Hæmophilus influenzæ* type b in the immediate post-immunization period.
J Pediatrics; **114**: 742-747.
17. Storsaeter, J., Olin, P., Renemar, B., et al., 1988.
Mortality and morbidity from invasive bacterial infections during a clinical trial of acellular pertussis vaccine in Sweden.
Ped Inf Dis J; **7 (9)**: 637-645.
18. Hinman, A.R., and Orenstein, W.A., 1990.
Immunization practices in developed countries.
Lancet; **335**: 707-710.
19. Ad Hoc group for the study of pertussis vaccine.
Placebo-controlled trial of two acellular pertussis vaccines in Sweden - protective efficacy and adverse effects.
Lancet 1988; April 30: 955-960.
20. Anonymous, 1989.
Licence application for pertussis vaccine withdrawn in Sweden.
Lancet; 14 January : 114.
21. Cherry, J.D., Brunell, P.A., Golden, G.S., and Karzon, D.T., 1988.
Report of the Task Force on Pertussis and Pertussis Immunization - 1988.
Pediatrics (suppl.): 938-984.
22. Benn, E.C., and Alstead, S., 1931.
Diphtheritic hemiplegia.
Lancet; 21 Nov: 1131-1133.
23. Cunningham, A.A., 1940.
Anaphylaxis after injection of tetanus toxoid.
Br Med J; Oct 19: 522-523.
24. Blumstein, G.I., and Kreithen, H., 1966.
Peripheral neuropathy following tetanus toxoid administration.
J Amer Med Ass; **198**: 1030-1031.
25. Holliday, P.L., and Bauer,R.B., 1983.
Polyradiculoneuritis secondary to immunization with Tetanus and Diphtheria toxoids.
Arch Neurol; **40**: 56-57.

26. Goodall, E.W., 1918.
Serum sickness.
Lancet; 2 March: 323- 327.
Conclusion in *ibid*. 9 March: 361-370.
27. Coulter, 1981.
The Controlled Clinical Trial.
Center for Empirical Medicine. Washington, D.C.
28. Karlsson, L., and Scheibnerova, V., 1991.
Association between non-specific stress syndrome, DPT injections and cot death.
Pre-print of a paper presented to the 2nd National Immunisation Conference, Canberra 1991: 1-9.
29. Scheibner, V., 1991.
Association between non-specific stress syndrome, DPT injections and cot death.
Proc. 2nd National Immunisation Conference, Camberra, 27-29 May 1991:90-91.
30. Clemens, J.D ., Ferreccio, C., Levine M.M., Horwitz, I., Rao, M.R., Edwards, K.M. and Fritzell, B., 1992.
Impact of *Hæmophilus influenzæ* type b Polysaccharide-Tetanus Protein conjugate vaccine on responses to concurrently administered Diphtheria-Tetanus-Pertussis Vaccine.
J Amer Med Ass, 1992; **267 (5)**: 673-678.
31. Landrigan, P.J., and Witte, J.J., 1973.
Neurologic disorders following live measles vaccination.
J Amer Med Ass; **223 (13)**: 1459-1462.
32. Gray, J.A., and Burns, S.M., 1989.
Mumps meningitis following measles, mumps, and rubella immunisation.
Lancet; 2: 98.

# 12

# SUMMARY

Extensive study of medical papers dealing with vaccination published over the past one hundred years reveals a number of facts which can be summarised as follows:

**1. Vaccines are highly noxious substances made up of bacterial or viral components in a solvent.**

The solvent is saline or water solution containing tissue fixatives (formaldehyde, aluminium phosphate, aluminium hydroxide) and preservative (thiomersal, a mercury compound). The solvent solution alone is often used as a "placebo" in vaccine trials. When injected it causes local reactions (redness, swelling at injection site) or even systemic reactions (fever, vomiting). Allergy to thiomersal has been described.

The bacterial and viral content of vaccines are the main antigenic component. Bacteria and viruses, which are supposed to provoke immunity against a particular disease, can be live, killed or attenuated (usually by formaldehyde treatment). However, even killed micro-organisms still contain foreign nucleic acids, DNA and RNA, which continue to have a deleterious effect on the recipient. It has been documented that attenuated bacteria and viruses can become virulent through passage in humans.

Having known foreign substances injected into the blood stream is bad enough, but vaccines present additional serious danger: contamination by tens or hundreds of unknown animal or human bacteria and viruses which are inevitably present in the animal (or human) tissues used for culturing the vaccine microorganisms.

Contamination of polio vaccines with animal viruses living in the monkey kidneys used to produce those vaccines has been extensively

studied and the results published. The most intensive research of this subject was conducted mainly in the fifties and sixties.

The most important of these contaminating animal viruses is a group of some 40 simian retroviruses, of which SV40 has been extensively studied and found oncogenic (cancer producing) in hamsters, mice and other animals (small wild rodents) and in humans. The enormous upsurge in incidence of leukæmia and cancer in children since the introduction and mass use of vaccines is also well-documented in the medical literature. In addition to this, antigenic over-stimulation by foreign antigens in vaccines also leads to cancer as amply documented in animal studies and in humans.

The presence of animal retroviruses was known soon after the mass polio vaccination started. The reason given by the FDA for not taking drastic action was that there was no published material on harmful effects of these viruses to humans. However, even when the harmful (oncogenic) effect of these viruses did become known, vaccines continued to be administered, despite containing these animal viruses. Treatment with formaldehyde proved to be ineffective to a great extent — a 14 day treatment by formaldehyde, although destroying the bulk of the microorganisms, tapers off and leaves some viruses intact.

The second group of animal viruses , documented to contaminate polio vaccines, are simian immunodefficiency viruses (SIV). An elegant analysis of the batch of polio vaccines and the timing and geography of the polio eradication campaign in certain African countries by an independent scholar, Louis Pascal, provided evidence for a causal link between administration of these contaminated polio vaccines and the origin of AIDS.

The repeated injection of polio vaccine at weekly intervals in an attempt to treat genital herpes in American homosexuals is now blamed for the start of the epidemic of AIDS in the United States. Polio and smallpox vaccination with vaccines contaminated by SV40 and bovine retrovirus is blamed for starting the epidemic of AIDS in Africa. This affected most those countries targeted for the so-called smallpox eradication programme by the World Health Organisation.

**2. Vaccines are ineffective in preventing infectious diseases.**

Again, medical literature brings evidence upon evidence that vaccinated children contract the very diseases against which they are vaccinated at the same as or at a higher rate than unvaccinated children. The percentage of fully vaccinated "victims" simply reflects the vaccination compliance.

Proponents of vaccination often claim that if the vaccines do not prevent children from catching the diseases, at least they make the diseases less severe. Now there is ample evidence that for instance whooping cough became a mild disease in those countries that do not vaccinate against whooping cough — Sweden, the former West Germany (especially Hamburg and Stuttgart) and Italy. On the other hand, there is growing evidence that measles in vaccinated children can often be a much more serious illness than in the unvaccinated. Children vaccinated by either live or killed measles vaccine may develop atypical measles, an especially vicious form of measles with pulmonary involvement and atypical rash, serious side effects and high death rate.

Vaccines, like any other noxious substances, do not immunise when injected into the blood stream, rather they sensitise. This was known more than 100 years ago and was well described in medical journals like The Lancet. The upsurge in allergies in children in the past 50 years is the result of repeated injections of the foreign antigens in vaccines. An exaggerated reaction to measles virus or subsequent measles virus vaccine is just another example of sensitisation by vaccination.

Asthma and other autoimmune diseases are another widespread side effect of vaccination. Antigens contained in the vaccines cause lingering upper and lower respiratory tract infections, such as otitis media and bronchitis, leading to the development of wheezing and asthma. Increase in incidence and mortality due to these diseases is also well documented in medical papers.

**3. Vaccines may cause serious local and systemic side effects, of which the most worrying is neurological damage.**

When adults experience local or systemic reactions from vaccines, there is no problem accepting these as causally connected with the vaccines. The fact of delayed reactions is well-documented and accepted in adults. However, when it comes to babies who also have delayed reactions to vaccines, when a convulsion, fit or encephalitis starts later than 72 hours after vaccine injections, it is considered 'coincidental'. Time and time again doctors do not hesitate to say that "it would have happened anyway". No reason for the onset of these events could be more potent than an injection of highly noxious substances, such as are in vaccines. The reasons for this most unfortunate interpretation are that vaccines are considered innocuous and totally safe and that babies cannot speak. A baby screaming in pain is usually ignored or handled almost with contempt.

Quite obvious signs of encephalitis like drowsiness, excessive somnolence, irritability, screaming with pain, and/or uncontrollable movements and fits, are described as events on their own as if they were not clinical signs of encephalitis or encephalopathy. When adults after vaccination experience the same clinical signs, they are readily and without hesitation described as signs of encephalitis or encephalopathy.

This general habit of considering side effects of vaccine injections coincidental is of special concern, whether it is the onset of adverse reactions like convulsions, fits and serious brain damage or illness against which the person was vaccinated. There is now an enormous pile-up of "coincidental" adverse reactions or onset of illness within 14 or more days of vaccination all over the world amounting to tens of thousands of cases. Clustering of the onset of adverse reactions or of illness around the same - critical - days are published time and again. In fact all papers dealing with side effects of vaccination or onset of illness and especially deaths after vaccination brings data on clustering. Most authors, however, do not understand the importance of clustering. Instead of talking of coincidence, it would be much more reasonable to see clustering for what it is: the evidence a causal

relationship. Again, nothing can be more intruding than an injection of a highly noxious and infectious substance like vaccines.

**4. There is no need to artificially immunise our children and ourselves.**

The body has proper, natural mechanisms to create immunity to diseases. The diseases themselves are the priming and challenging mechanisms of the maturation process leading to the competence of the immune system. It has been demonstrated time and again, that infectious diseases of childhood are very beneficial for children to catch. They function to even out differences in rates of development of different body systems and so perform a sort of balancing act in a fast-growing organism. They also represent important milestones in the overall development of children. The general inflammatory process is important in the dynamics of maturation of the immune response to diseases. I feel embarrassed to hear and read of orthodox medicine's futile efforts to stop children from getting childhood diseases — it is a sign of ignorance and a naive approach. The fanaticism fed by ignorance, irrational fear of illness and greed are the moving forces behind the ritual of vaccination. Vaccine injections represent an enormous and unjustified insult to a young child.

**5. Normal portals of entry for infectious agents — mouth and nose — are instrumental in the natural immunological process.**

Vaccines, being injected into the blood stream, bypass these normal passages. Immunologists repeatedly warn that unless vaccination exactly emulates the natural immunity process, life-long immunity cannot and will not be achieved.

Moreover, introducing vaccines into the circulation gives them direct access to the major immune organs and tissues without any obvious way of getting rid of them.

Various elements of vaccines can stay in the body for long periods of time, some of them permanently, often by incorporating themselves into the genetic material of the host's cells. This provokes

constant effort to expel these foreign substances leading to a systematic weakening of the immune system. Constant antigenic stimulation of the immune system leads to cancer and leukæmia and a host of other autoimmune diseases.

Instead of providing protection against acute infectious diseases, vaccination drives the disease deeper into the body and leads to chronic infestation by the pathogenic agent. Subacute sclerosing panencephalitis is one of many examples of this slow process. Warts, herpes, shingles and AIDS are other examples.

**6. Proponents of vaccination claim victory over diseases.**

Despite all this being published in innumerable medical papers, those pushing for mass vaccination seem totally to ignore the equally well-documented fact that all infectious diseases, including those against which they vaccinate, have been on the decline for decades, before any vaccine was even developed. Better living conditions, better nutrition and uncrowded living, and above all, better sanitation and clean water are the only factors that should be credited with the fall in incidence, mortality and especially severity of infectious diseases.

The best evidence for the validity of this is that many diseases, like bubonic plague, scarlet fever and tuberculosis which used to cause many deaths, have disappeared without mass vaccination programmes. Even smallpox receded substantially, although not entirely, despite the very low percentage of people vaccinated. Indeed, those countries with the highest vaccination rates experienced time and again the biggest smallpox epidemics with very high mortality. None of these disease has the ability to spread these days, even though bubonic plague occurs in small outbreaks even in the United States and tested domestic and semi-feral animals show antibody to *Yersinia pestis*.

Bubonic plague, tuberculosis and smallpox have potent animal reservoirs. Nevertheless their importance has totally disappeared; the only factors relevant to this being improved nutrition and better living conditions. Even influenza epidemics do not eventuate any

more. In my opinion, the reason for this is that some 40%-50% of people (at least in the developed countries) take regular supplements of vitamins, and especially of vitamin C.

Flu vaccines not only do not prevent influenza outbreaks, they introduced serious and deadly reactions like the Guillain-Barré syndrome and the Legionnaires' disease.

Many data indicate that the existing downward trend at the time of the introduction of many vaccines like whooping cough was actually slowed down after mass vaccination started. Based on hospital admissions, it was estimated recently that some 125,000 cases of whooping cough per year occur in the United States despite mandatory vaccination. This incidence matches the reported cases of whooping cough in the countries that abandoned whooping cough vaccination.

### 7. Proponents of vaccination often misrepresent results of trials and tests of vaccines.

Babies that died during the trial are as a rule excluded from evaluation. A convenient 'waste basket' label called cot death is readily available. Serious side effects, especially of a neurologic nature, are considered 'coincidental'. Infections which are directly caused by vaccine injections, due to their ability to lower the resistance of the host, are also described as 'coincidental'. Many diseases have been renamed and reclassified to conceal the true nature of vaccine-caused disease: instead of paralytic poliomyelitis we have cerebral palsy, smallpox has become monkey pox or white pox.

Researchers often do not quite understand their own raw data. Therefore it is very important to study in detail the basic data on which many trials of vaccine effectiveness or safety are based. Conclusions contradicting the data are often expressed and published. Absurd reasoning in medical papers is not rare. As if the only conclusions allowed are that despite all the demonstrated ineffectiveness and dangers of various vaccines, vaccination must go

on. The pain and deaths of babies seem quite irrelevant. As if the death of a small baby was a lesser death.

**8. Fear and intimidation are powerful weapons.**

These are the weapons of mental torture applied to make parents submit their babies to this barbaric, unscientific and totally unnecessary procedure. However, it is the parents who have all the power over vaccination in their hands. Therefore, it is important and indeed vital for the parents of small babies to educate themselves about everything pertaining to the well-being of their children. Since they are left with the problems caused by vaccines, they also have to decide whether they are going to vaccinate their children or not. Parents have to realize that illness is good for the health and development of children and they have to allocate the time and patience to allow the infectious diseases to take their natural course.

Above all, parents have a moral and legal obligation to ensure health and good prospect of a full life for their children. Their loyalty should at all times be with their children and not with their peers or the medical system.

Medical doctors also have to see the writing on the wall and decide to protect and truly serve their patients. The medical system in the developed countries has become totalitarian and highly politicised. It lacks the philosophy of healing and true knowledge of the physiology of the human body. It is little more than a big business which is mandating their procedures and enforcing their medications. Who would not wish to have a product or service that everybody has to buy by law?

**9. Vaccination is the single biggest cause of cot death**

Thousands of babies die every year of cot death in the so-called developed countries. The age at which most (80%) cot deaths happen is between two and six months. Doctors usually say that this is coincidental with vaccinations. However, most studies of cot deaths and infantile convulsions do not include information on vaccination. Those few publications which listed cot death babies by the time

interval from the injections to death provided the evidence of the causal association between DPT (and polio) injections and cot death even though, they, quite absurdly, concluded that there was no causal relationships between the two. The simple scientific truth is that there was a significant and clear clustering of these deaths around the critical days as recordced by the microprocessor Cotwatch and as even accepted in a few medical papers dealing with side effects of vaccines.

These critical days are the days on which any baby has flareups of stress induced breathing after vaccine injections.

The post-mortem findings of cot death babies are quite characteristic of the non-specific stress syndrome as defined by Hans Selye. It is sad that most medical doctors in the developed countries never heard of Selye's work. It is also sad that Hans Selye did not live long enough to see the reflection of the dynamics of his non-specific stress syndrome in breathing of babies as recorded with microprocesssor Cotwatch.

When Japan moved the vaccination age to 2 years, the entity of cot death, following DPT injections, disappeared in that country. Japan zoomed to the lowest incidence of infant mortality in the world.

**10. Vaccination is one of the biggest "magic bullets".**

Vaccination is the epitome of ignorance and the unscientific approach to illness. Yet, modern medicine considers itself scientific. Nothing could be further from the truth. The only scientific medical systems are those which are based on detailed and meticulous observation and knowledge of the human body and which cater to individuality. One does not need expensive and complex diagnostic tools to be able to diagnose the patient's condition and choose the right remedy.

Without the knowledge of dose-related effects of remedies and without testing these on themselves, the orthodox medical doctors will continue causing more harm than good.

Hippocrates is all but forgotten. Homeopathy, based on the intimate knowledge of the human body and the dynamics of h

and illness, is the medicine of the twenty-first century. Although developed by Dr Hahnemann some two hundred years ago, it is based on the most modern understanding of the dose effect of remedies and the most modern physics of solutions. It is also based on an intimate knowledge of the dynamics of health and illness.