Contamination of polio vaccines by monkey viruses and other microorganisms
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Abstract

Polio viruses for the production of polio vaccines have been cultured on monkey kidney tissue and cell lines. These are naturally infected by monkey (simian) viruses and other microorganisms and agents (protozoans, amoebas). They cause, and/or are a co-factor, in a great variety of cancers and other diseases. The methods used to "clean-up" the animal tissue and/or microorganisms of these undesirable contaminants are incomplete and reversible. The inactivation with formaldehyde is a subject to asymptotic factor, which means that within a number of hours (usually 40) most of the microorganisms are inactivated (about 75%), but afterwards there is a viable residue of live organisms capable of multiplying and spreading indefinitely. When such inadequately treated vaccines are introduced into the vaccine recipients, even those organisms that were inactivated, may and do, revert back to the original virulence. Most of them are carcinogenic and cytopathogenic (tissue destructive) and cause or co-cause a variety of cancers and other diseases, practically all those modern ills of children and humanity.

Introduction

Not only the polio virus vaccines were causing paralysis, already during the first trials, but already in 1954-58 researchers knew that all brews of polio vaccines were contaminated by a number of new viral and other (protozoan – amoebas: for more detail see Scheibner (1) agents which originated from tissue cultures of monkey kidney cells. These monkey kidney cells were the medium on which all polio vaccines have been cultured. Rustigian *et al.* (2) established that monkeys carry a great number of viruses, called SV1 to SV40. Of these, SV40 was simply the most researched one. Other viruses were B virus, foamy agent, paramyxoviruses (respiratory syncytial, a measles-like, virus), herpes-like viruses (cytomegalovirus), adenovirus, haemadsorption viruses, LCM virus, arboviruses, amoebas and a great variety of miscellaneous viral and non-viral agents.

Hull *et al* (3) wrote that the increased use of the technique of cell cultivation for isolation, maintenance and study of viruses, has resulted in the discovery of many hitherto unknown cytopathogenic agents, such as amoebas belonging to the genus Acanthamoeba. They grew readily in tissue cultures and could be titred to very low dilutions. They appeared to have the ability to infect and kill monkeys and mice following intracerebral and intraspinal inoculation.

In screening human's stool samples or rectal swabs for all poliomyelitis viruses, Melnick and Stinebaugh (4) isolated several "orphan viruses" which do not type with polio virus antiserums, and, from the same source, Sabin obtained five viruses referred to as HE1 and HE2. The new respiratory infection (RI) series of viruses was isolated in tissue cultures by Chanock et al. (5) and Chanock and Finberg (6) from the throat washings of chimpanzees with coryza and soon after from babies (vaccinated with such contaminated vaccines) with respiratory disease. Their contemporaries, Rustigian *et al* (2), reported a recovery of two cytopathogenic agents from cultures of monkey kidney cells. Thus, a number of viral agents are present in tissues and excreta of man and lower animals, which defy detection by methods other than tissue culture.

During the production and testing of poliomyelitis vaccines, hundreds of thousands of monkey kidney cultures prepared from thousands of wild monkeys were observed by Hull *et al* (3) who encountered numerous filterable, transferable, cytopathogenic agents other than polio virus. They were introduced into the cultures with the monkey kidney tissue. Even though several of these agents totally destroyed cultures tissues and even caused serious diarrhoea in laboratory animals, all of which died, their pathogenesis in humans was not further researched. Central nervous system was particularly

susceptible to the pathogenic properties of such viruses; the histopathological lesions observed in the intracerebrally inoculated monkeys revealed necrosis and complete destruction of the choroid plexus, plus caused generalised aseptic (non-bacterial) type of meningitis.

The isolated agents were referred to as Simian viruses (SV). As it turned out, the SV40 was the one best researched. Even though there was no yet known and published research about the harmful effects to humans of any of these SV viruses, the authors concluded that they were probably harmless. The future developments proved them woefully wrong.

Sweet *and* Hilleman (7) further elaborated on SV40 as the vacuolating virus. They wrote that viruses are commonly carried by monkeys and may appear as contaminants in cell cultures of their tissue, especially the kidney. They listed B virus, foamy agent, measles-like virus, haemadsorption viruses, LCM viruses, arboviruses, and a wide variety of miscellaneous viral agents.

During the preceding two years Sweet *and* Hilleman's (7) research has repeatedly encountered a new simian virus of rhesus and cynomolgus monkey kidney origin. The virus was considered unique because it did not cause a cytopathic effect in the rhesus or cynomolgus kidney cultures from which it was derived. Instead, it grew and caused marked cytopathic change in cell cultures of heterologous species. It was named the vacuolating virus because of the prominent cytoplasmic vucuolation seen in affected cell cultures. Dr. Hull suggested that this agent be given the "official" designation of SV40. This also raised the important question of the existence of other such viruses. More than 20 strains of vacuolating virus have been recovered. All strains of Dr. Albert Sabin's live attenuated poliomyelitis vaccines contained strains of vacuolating viruses. The essentially ubiquitous occurrence of vacuolating virus in the various virus seed stocks and vaccines suggested a high infection rate among normal rhesus and cynomolgus monkey cell cultures; of 10 lots of rhesus kidney, 7 yielded vacuolating virus, even though these cultures showed no sign of cytopathic changes. [Here, I have to make a parenthesis: when such authors wrote that their cultures showed no signs of cytopathic changes, they did not disclose that it only meant “within the time of their observation”. The time of observation in many tests is arbitrarily chosen as 7, 10 or 14 days etc. which invariably means that they missed all delayed reactions; it explains why there is such an epidemic of typical cancers and other major problems caused by monkey (or other animal) viruses in adults who were vaccinated with the contaminated vaccines as school children or babies.]

The vacuolating agent was readily filtered through bacterial sterilising filters. It was relatively heat stable compared with most viral agents.

The occurrence of vacuolating virus in human and monkey sera.

The discovery of the vacuolating virus in monkey kidney cell cultures, but not detected by the current procedures used at the time, raised the question of whether other as yet undetected viruses might also await discovery. As shown in their report, Sweet and Hilleman (7) demonstrated that all types of Sabin’s live polio virus vaccines, fed to millions of persons of all ages, but especially the infants and young children, were contaminated by vacuolating virus.

All nine persons studied by the above authors, given two doses of adenovirus vaccine prepared in rhesus and cynomolgus monkey kidney culture, developed antibody against the vacuolating virus. Similarly, seven of thirteen persons given two doses of Salk poliomyelitis vaccine developed antibody against the vacuolating virus. These results showed the high frequency of vacuolating virus in monkey renal cell cultures and also the apparent high potency of vacuolating virus antigen in these vaccine preparations. None of five persons developed antibody against the vacuolating virus when fed Sabin live polio vaccine on six occasions. The authors wrote that this suggests lack of infectiousness of the

vacuolating virus when administered by the oral route. However, the infectiousness in man when administered by the respiratory route was high.

I reiterate that, decades later, we now have an increasing epidemic of SV40 cancers in the developed countries that used these vaccines for mass vaccination programs. It is not surprising that the highest incidence of such tumours is in such developed countries as Sweden which has exclusively used the injectable Salk type vaccines. The oncogenic properties of this virus have now been well established. Olin and Giesecke (8) claimed that potential exposure to SV40 in polio vaccines used in Sweden during 1957 had no impact on cancer incidence rates 1960 to 1993. In 1957, when national polio vaccination was started in Sweden, potentially SV40 contaminated vaccine was given to approximately 700,000 individuals, mainly preschool and school children born 1946 to1953. From 1958, only Swedish polio vaccine was used, initially produced by culture on kidney cells from Javanese macaques. Testing for SV40 started in 1961, when also vaccine batches produced earlier were tested retrospectively, and during the 30 years that monkey kidneys were used for production of the Swedish inactivated polio vaccine, allegedly no SV40 contamination was detected according to the manufacturer (R. Salenstedt, personal communication).

Olin and Giesecke (8) claimed to have explored cancer incidence rates in the cohorts exposed to potentially contaminated polio vaccines in Sweden, based on the Swedish Cancer Registry of annual cancer rates in 5-year-age groups for the years 1960-1993. Cancer incidence cohorts maximally exposed were followed during this period, and the incidence when these cohorts reached a specific age was followed during this period, and it was compared to the incidence when unexposed cohorts reached the same age. The results of such research were as follows. "For osteosarcoma and brain ependymoma, the overall age standardised incidence rates were essentially unchanged between 1960 and 1993, and age specific rates were similar in the exposed and unexposed male and female cohorts. During the same period the overall age standardised incidence rates in males of brain cancers increased from 9.0 to 13.1 and of pleural mesotheliomas from 0.2 to 2.1 per 100,000. None of these increased rates was associated with the exposed cohorts". In their own interpretation, Olin and Giesecke (8) wrote "The use of potentially SV40 contaminated inactivated polio vaccines in Sweden had not been shown to be associated with increased cancer incidence. However, the exposed cohorts have not yet reached the age of increased risk of brain cancer or mesothelioma".

Another, more viable, explanation is possible, namely that, just as polio vaccines produced in other countries, the Swedish-produced vaccines continued to be contaminated by monkey viruses, including SV40. The following established facts support my conclusion.

When the oncogenic properties of SV40 became public knowledge, the manufacturers were asked to clean up their act. They have been doing it by subjecting the brew of the polio vaccine to 14-day treatment with 1:4000 solution of formaldehyde. However, as demonstrated by Gerber *et al* (9**),** this process is not entirely effective. It is subject to the asymptotic factor: within about 40 hours, most of SV40 is inactivated, but then there remains a residual viable live viral fraction remaining throughout the entire 14 day period of the treatment, and, indeed, indefinitely. As Gerber *et al* (9) demonstrated, Sweet *and* Hilleman (7) observed their essay tests for a period of only ten days. However, there is a marked delay in the appearance of cytopathic effect caused by the formaldehyde treated SV40. In most instances initial CPE did not appear until the 11th day and the final infectivity titers were reached on the 13th and 14th day. Another factor which could influence the detection of SV40 in formalinised vaccines is the degree of sensitivity of essay techniques.

The conclusions of Gerber *et al* (9) were interesting. Even though large groups of the population in the USA and abroad must have been infected with varying amounts of SV40 during the mass vaccination with formalinised poliomyelitis and adenovirus vaccines, they concluded that clinical studies on selected groups of vaccinees suggested that no harmful effect can be attributed to vacuolating virus

within the limits of the observation period. How about outside the observation period? They have not considered it, but we now know better.

Eddy *et al* (10) demonstrated that viruses contaminating monkey kidney tissue cultures cause tumours in younger than one to three day-old hamsters. The earliest tumour was detected 99 days after injection of the cell extract but most of the tumours developed after 7 to 9 months. Transplants from the first tumour injected into 12 hamsters resulted in formation of tumours in 10 of them. These tumours appeared within 13 to 41 days and all 10 hamsters died by the 73rd day. Some hamsters developed tumours in their lungs and others in their kidneys. They appeared to be undifferentiated carcinomas in subcutaneous tissue, kidneys and lungs. Newborn hamsters did not develop tumours after injection with hamster-tumour extracts. However, there was no indication about the length of the follow up time.

Hull *et al* (11) described additional agents both from cultures of monkey-kidney cells and directly from tissues and excreta of monkeys, such as amoeba belonging to the genus Acanthamoeba.

In another experiment (Kravetz *et al* (12)), sixteen of 27 individuals inoculated with a mixture of RS virus and ten thousand units of SV40 experienced respiratory disease. All volunteers developed neutralising antibody against the SV40 component. None of the eight subjects given the dual material mixed with SV40 antiserum developed SV40 antibody during the month following inoculation. Interestingly, from throat swabs of two of the test subjects, SV40 virus was recovered on the seventh day and from another, on the eleventh day, on the critical days as established by my own research. The recent experience with the epidemic of SV40-caused brain and other cancers proved that the short-term follow up studies were totally insufficient.

Starting in 1962 literally hundreds of studies have been published demonstrating the oncogenic effects of SV40 in humans.

The evidence of oncogenic effects of SV40 in animals and humans

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Shah *and* Nathanson (13) wrote a review article on human exposure to SV40; they wrote that the time period during which SV40 contamination of virus pools occurred was between 1954 and 1963, after which date all vaccines on the market were "probably free of SV40". Vaccines studied at the Division of Biologics Standard after June 30 1961 were required to be free of SV40, but vaccine lots previously cleared (even though they were contaminated) were not withdrawn from the market. That means that contaminated vaccines continued to be given to babies. Other then polio vaccines also contained SV40, such as adenovirus vaccines given subcutaneously primarily to young males in army camps, formalin-inactivated polio vaccines, given subcutaneously to millions of people including experimental lots of live polio vaccines given orally.

In some other countries, notably the USSR and eastern block, mass vaccination with oral vaccines during 1959 and subsequently, have exposed millions of people to orally administered SV40. In conclusion, conservatively, between 10% and 30% of such vaccines had live SV40 in them.

Baguley and Glasgow (14) wrote “From 1956 to 1966, the incidence of subacute sclerosing panencephalitis (S.S.P.E.) in the northern half of the North Island of New Zealand was approximately one hundred times greater than might be expected. No case was seen before 1956, and none has been seen since 1969…Mass vaccination of primary-school children with Salk vaccine was begun in 1956. The vaccine used is likely to have contained live SV40 virus. Killed measles virus is another possible contaminant. It is believed that the administration of Salk vaccine in New Zealand was related to the

appearance of S.S.P.E. in the community. The idea that an unusual reaction to measles infection is the sole cause of S.S.P.E. is not consistent with the observations in New Zealand."

Weiner et al. (15) reported on isolation of virus related to simian-virus 40 (SV40) from brains of two patients with progressive multifocal leukoencephalopathy, a human demyelinating disease. Virus was grown in cell cultures of primary African green-monkey kidney inoculated with cultures of cells derived from patients' brains. Electron microscopy showed virus-like particles resembling papovaviruses in the brain of one patient and in monkey-kidney cell cultures infected with both virus isolates. Immunologic studies using fluorescent and neutralizing antibodies indicate a close relationship between the isolated viruses and SV40.

Dohrmann *et al* (16) analysed histologically verified cases of ependymoma and ependimoblastoma (malignant ependymoma) occurring in children in Connecticut from 1935 to 1973. An increase in the incidence of ependymomas was noted since the mid 1950s. The mean ages of the diagnosis of ependymomas and ependymoblastomas were 5.6 and 5.0 years, respectively. [The ages correspond with the ages at administration of the contaminated vaccines.]

Mortimer *et al* (17) published a long-term follow-up of 1073 children who were born between 1960 and 1962 and who, as new-born infants, received monovalent oral polio virus vaccines or, inactivated polio virus vaccine that contained SV40. They wrote that "A total of 15 studied subjects died, all of identified causes (chiefly birth defects or trauma) and no child died of cancer. A 15 year old girl had a mixed tumour of the salivary gland with histolopathological features of a low degree of malignancy." They concluded that no excess risk of cancer was observed in persons who were exposed as newborns to SV40 polio vaccine and followed for 17 to 19 years.

However, others reported the presence of SV40 like-antigens in tumours of patients and demonstrated that inactivated poliomyelitis vaccine and influenza vaccine given during pregnancy is associated with tumours in offspring.

Overbaugh *et al.* (18) monitored changes in the simian immunodeficiency virus (SIV) envelope (*env*) gene in two macaques which developed AIDS after inoculation with a molecular clone of SIV. As the animals progressed to AIDS, a selection occurred for viruses with variation in two discrete regions (V1 and V4) but not for viruses with changes in the region of SIV *env* that corresponds to the immunodominant, V3 loop of human immunodeficiency virus. Variability within the human immunodeficiency virus (HIV) genome begins with changes introduced by the highly error-prone HIV reverse transcriptase.

The contamination of polio vaccines with SIV, as the original cause of AIDS epidemic, is a subject of another assay (Scheibner in prep.).

SV40 and mesotheliomas (so called asbestos disease)

Cicala *et al.* (19) published an article titled "SV40 induces mesothelioma in hamsters". They wrote that in the course of studies to elucidate relative contribution of simian virus 40 (SV40) large T and small t proteins during oncogenesis, they observed the appearance of pericardial and pleural tumours in 100% of Syrian hamsters injected in the pleural space with wild SV40. When SV40 was injected via the intracardiac or intraperitoneal routes, more than 50% of hamsters developed mesothelial tumours. Macroscopic, microscopic, ultramicroscopic, and histochemical characteristics identified these neoplasms and derived cell lines as mesotheliomas and mesothelioma-derived cell lines. The absence [sic; it should be 'the presence'] of mesotheliomas in hamsters injected with SV40 small t deletion mutants indicates that the small t protein plays an important role in the development of SV40-induced

mesotheliomas. They considered their findings the first definitive published report of virus-induced mesotheliomas in mammals.

They also wrote that DNA tumour viruses, including SV40, have not been reported to induce mesotheliomas, however, "fetal mice inoculated in utero with polyoma virus developed proliferative lesions of the mesothelium. Although these lesions sometimes covered the entire serosa, they did not become malignant and later regressed completely". Another problem with experimental mesothelioma models is the long latency, the relatively low number of animals developing mesotheliomas, and the appearance in addition to mesotheliomas, of other tumours, such as liposarcomas and rhabdomyosarcomas.

Wild type SV40 is highly oncogenic particularly in hamsters and when the newborn hamsters are particularly susceptible, usually developing fibrosarcomas at the injection site following subcutaneous injection of SV40. When newborn hamsters are injected SV40 intracerebrally, they develop ependymomas. When SV40 is injected intravenously into weanling hamsters, a circumstance in which many cell types are exposed to high concentrations of the virus, lymphocytic leukaemia, lymphoma, soft tissue sarcoma, and/or osteosarcoma occur.

They also wrote that recent discovery of an association of the large T antigen of SV40 with both the retinoblastoma (RB) and p53 cellular gene products and the clear correlation of defects in these same cellular proteins (RB and p53) with a number of human tumours strongly suggest that the transformation of cells by SV40 may have direct relevance to our understanding about the molecular events that lead to cancer in man.

Under the subheading "Macroscopic characteristics of SV40-induced tumors", Cicala et al. (19) reported on the results of their study. They wrote, "Approximately 3 months after injection, hamsters injected ic (intracardially) or ipl (intrapleurally) with wt (wild type) SV40 suddenly developed dyspnea, appeared lethargic, and became extremely ill. Upon necroscopy, these animals revealed extensive tumor formation within the thorax. These tumors formed a continuous layer over the pleural and the pericardial surface, encasing the lungs and often the heart and obliterating the pleural and pericardial cavities. Multiple gray or white ill-defined nodules were seen in a diffusely thickened pleura. Pleural effusion was always present. The tumors did not penetrate deeply into the lung or heart tissue; however, they spread extensively over the chest wall and the diaphragm, which were often infiltrated. No distant metastases were observed. Some of the hamsters injected ipt (intraperitoneally) with wt SV40 developed lesions similar to those described above but in the peritioneal space. These primary peritoneal tumors spread widely over the serosal surfaces, including that of the intestine, and were easily distinguished from secondary tumors, which spread from the pleura through the diaphragm and invaded the peritoneal cavity. The macroscopic characteristics of all these tumors – pleural, pericardial, and peritoneal – were reminiscent of mesotheliomas".

"All of the 43 hamsters injected with wt SV40 (wt 776 and wt 830) developed tumors. Thirty of these tumors had macroscopic characteristics of diffuse malignant mesotheliomas…mesotheliomas developed 3 to 6 months after virus injection, and hamsters with mesotheliomas did not develop additional tumors. Lymphomas (10 hamsters), osteosarcomas (four), and myxoma (one) were observed in those hamsters that did not develop mesotheliomas."

And "All of the hamsters injected ic with SV40 small t mutants dl (deletion mutants) 883 and dl 2006 developed multiple, soft, white/grey encapsulated tumors of about 2 mm to 3 cm in diameter, apparently originating from the abdominal lymph nodes. Characterisation of these tumors indicated that they were either true histiocytic lymphomas or B-cell lymphomas. Only one mesothelioma was observed in hamsters injected with SV40 small t mutants…These animals, injected with dl 2006,

developed both a mesothelioma and a lymphoma. None of the hamsters injected with media alone (control group) developed any type of tumor."

Microscopic characteristics of SV40-induced mesotheliomas. "Histologically, mesotheliomas revealed various morphologies. These were independent of the type of virus injected or route of virus injection. In most tumors, malignant cells formed papillary structures, the small papillae consisting of a core of delicate connective tissue covered with closely packed cuboidal; cells…In other tumors, or in different areas of the same tumor, neoplastic cells formed tubules or glandlike spaces (pseudoacini…). Some mesotheliomas were instead highly cellular formed by interwoven bundles of spindle cells, often in mitosis, with ovoid basophilic nuclei, and eosinophilic cytoplasm…Thus, histologically these lesions were reminiscent either of the epithelial mesotheliomas…or of the spindle cell (sarcomatoid) mesotheliomas…found in humans. However, most of the SV40 induced mesotheliomas were of the mixed type. In these mixed type mesotheliomas, both spindle-cell (sarcomatoid) areas and epithelial areas were observed. In one mesothelioma, areas of osseous and cartilaginous metaplasia were encountered."

The authors further established cultures from 17 different mesotheliomas. Mesotheliomas-derived cell lines grew in tissue culture adherent to the plastic dishes; these showed polygonal shape with large nuclei and abundant cytoplasm.

Macroscopic and microscopic characteristics of the pleural, pericardial, and peritoneal tumours observed in these hamsters were all of mesothelial origin. However, malignant mesotheliomas had to be carefully distinguished from metastatic carcinoma, particularly so in the case of pulmonary adenocarcinoma. The authors used histochemical staining and also found electronmicroscopic examination useful in differentiating mesotheliomas from other tumours. All these methods of testing suggested the mesothelial origin of the tumors. The original tumour, as well as the tumour and the derived cells had similar ultramicroscopic characteristics.

Mesotheliomas occurred only in hamsters injected with SV40 viruses: thus there was little doubt that they were virally induced. To verify this hypothesis, the authors isolated high molecular weight cellular DNA from five mesotheliomas and three mesothelioma-derived cell lines and performed Southern blot analyses on all of them using different restriction enzymes: the SV40 genome was integrated into the cellular hamster DNA. 100% of the cells from each of the seven mesotheliomas were induced by the SV40 that had been injected into the hamsters. Furthermore, the absence of spontaneous mesotheliomas in control animals injected with media alone and the rarity of mesotheliomas in hamsters injected with SV40 small t mutant (one animal developed mesothelioma out of 34 hamsters injected) further suggested that the SV40 plays a causal role in the induction of these mesotheliomas and that the small t antigen of SV40 was required in this process.

Cicala *et al.* (19) quoted Gerber and Kirschstein (1962) who reported that SV40 induced ependymomas in hamsters. Their contemporaries, Bergsagel *et al* (20) reported DNA sequences similar to those of simian virus 40 in ependymomas and choroid plexus tumours of children.

Carbone et al. (21) investigated simian virus 40-like DNA sequences in human pleural mesothelioma. They wrote that mesotheliomas are pleural, pericardial, or peritoneal neoplasms frequently associated with asbestos exposure, and it is estimated that over the next twenty years up to 80,000 new cases are expected in the USA alone. However, they also wrote that they found simian virus 40-like DNA sequences in 29 of 48 mesotheliomas studied (60%) and demonstrated simian virus large T-antigen expression in 13 of 16 specimens. The matching lung samples did not contain simian virus 40-like sequences; however, they contained asbestos. They considered their findings the first demonstration of a physical link between DNA virus-like sequences in human mesothelioma. They suggested that a simian virus 40-like virus may act independently or as a co-carcinogen with asbestos. It originates

from the serosal lining of the pleural, peritoneal, or pericardial cavities. Survival from diagnosis is usually less than one year and none of the currently available therapeutic approaches have been shown to alter the natural history of this disease.

They also wrote that the incidence of malignant mesothelioma in Europe and in the United States until the 1940s was extremely low. Most pathologists at that time believed that "mesotheliomas did not exist at all, but that neoplasms involved the pleura always originated from other primary tumours, usually from "microscopic nodules of cortical broncho-pulmonary carcinomas".

They quoted Mark and Yokoi (22**)** who analysed retrospectively the autopsy files of The Massachusetts General Hospital. In 95 years they recorded approximately 100 pleural mesotheliomas at autopsy, all of which occurred in the most recent 45 years. Importantly, they found no evidence for background mesotheliomas before the commercial use of asbestos and suggested that mesothelioma might be a "new disease". The incidence of mesotheliomas continued to rise and they represent a serious threat to public health with approximately 2000-3000 cases per year in the United States. [The last 45 years means since 1949-1950 which is important due to the subsequent findings.] However, there were even more important findings, especially that the prevalence of mesotheliomas in people with prolonged heavy exposure to asbestos is 2% to 10%, and the latency period between the initial exposure to manifestation of disease is usually 20 to 50 years. The authors asked why it was that only a relatively small proportion of people exposed to asbestos develop mesotheliomas, or why approximately 20% of patients with mesothelioma lack a history of asbestos exposure. Although asbestos and cigarette smoke acted in a multiplicative fashion to increase the risk of lung cancer, cigarette smoke itself was not associated with the development of mesotheliomas. Even though asbestos was the only agent thought to be a known agent associated with the development of mesotheliomas and appears to have different effects on different cell types, i.e. as a complete carcinogen on mesothelial cells, it still acted more as a co-factor combined with cigarette smoke on bronchial epithelial cells. Additional as-yet-unknown factors as co-carcinogens with asbestos were missing in the induction of mesotheliomas.

In tissue cultures, asbestos fibers can cause mutagenic events, including DNA strand breaks and deletion mutations, through the production of hydroxyl radicals and superoxide anions, and thus alter chromosome morphology and ploidy by mechanically interfering with their segregation during mitosis.

Furthermore, macrophages will produce DNA-damaging oxyradicals following phagocytosis of asbestos fibers, and elaborate lymphokines which may depress the host immune response. Finally, asbestos fibers can mediate transformation of monkey cells by exogenous plasmid DNA, and similarly facilitate transformation of mouse cells by simian virus 40 (SV40), as demonstrated by Dubes (1993). SV40 is a DNA tumour virus that induces tumours in rodents (Topp et al. 1987**)** and immortalises human mesothelial cells in vitro (Ke et al. 1989) [all three papers quoted by Carbone et al. 21].

Carbone et al. (23) wrote that they recently reported that wild type SV40 will induce tumours which are morphologically, immune- and electron microscopically identical to mesotheliomas in 100% of intrapleurally SV40 injected hamsters and in 60% of those injected intracardially or intraperitoneally, as also demonstrated by Cicala et al. (19). Although SV40 is a monkey virus that has not been shown to naturally infect humans, only human cells are truly semipermissive, and supporting in tissue cultures, both cell transformation and SV40 replication. Except for a report demonstrating SV40 in one metastatic melanoma, SV40 has not been considered to be oncogenic for humans. Recently, however, the notion that SV40 is not oncogenic in humans has been challenged by the study of Bergsagel et al. (20) who demonstrated the expression of SV40-like sequences in human ependymomas and choroid plexus tumours. This substantiated a few reports about the presence and expression of SV40 in human brain tumours and was consistent with the discovery 30 years earlier

that SV40 could produce ependymomas and choroid plexus tumours in hamsters (Gerber and Kirschtein 1962). Carbone et al. (23) asked another question whether SV40 virus represents together with asbestos a co-carcinogen in human mesothelioma. The further research answered this question in an affirmative.

Among the results of their study was a finding that of the 36 patients exposed to asbestos, 22 had SV40-like sequences in their tumours; while, although having asbestos fibers in their lungs, the 2 benign FTP patients did not have viral sequences in their tumours.

In their discussion, Carbone et al. (23) wrote that their data indicate that SV40-like sequences may be present in as many as 60% of human pleural mesotheliomas, the sequence-positive tumours tested for viral expression contain Tag, that the majority of patients with these SV40-like sequences have an associated asbestos history, and, with two exceptions, samples 17 and 21, serum Tag antibodies correlate with the presence of SV40-like sequences and of Tag expression, evidencing systemic SV40 exposure. They concluded that these findings raise questions regarding the established notions of the pathogenesis of mesothelioma and its relationship to asbestos.

Carbone et al. (23) also wrote that their initial study which demonstrated that simian virus 40 (SV40) induced mesotheliomas in hamsters and that 60% of human mesotheliomas contain and express SV40 sequences, are now confirmed by others, including Griffith et al. ("Simian virus 40: a possible human polyoma virus presented at NIH workshop (27-28 January 1997) in Bethesda. They reiterated that mesothelioma is an aggressive malignancy resistant to therapy, originating from serosal lining of the pleural, pericardial and peritoneal cavities. "The incidence of mesothelioma continues to increase worldwide because of exposure to crocidolite asbestos. However, at least 20% of mesotheliomas in the United States are not associated with asbestos exposure, and only a minority of people exposed to high concentration of asbestos develop mesotheliomas. Thus other carcinogens may induce mesothelioma in individuals not exposed to high concentrations of asbestos, and/or may render particular individuals more susceptible to the carcinogenic effects of asbestos". They found that SV40 Tag retains its ability to bind and to inactivate p53, a cellular protein that, when normally expressed, plays an important role in suppressing tumor growth and inducing sensitivity to therapy. They also found that SV40 Tag interferes with the normal expression of the tumour suppressor gene p53 in human mesotheliomas. They concluded that their findings indicate the possibility that SV40 contributes to the development of human mesotheliomas which influence should further be investigated.

Wiman and Klein (24**)** summarised the studies of DNA tumor viruses such as SV40 as having provided critical clues about regulation of the cell cycle and malignant transformation of mammalian cells. They referred to Ludlow (1993) that virally encoded large T antigen (Tag) of SV40 promotes transformation of cultured human and rodent cells and induces tumours when expressed in transgenic mice. The complexing of Tag with two important tumor suppressor proteins, p53 and pRB, and with other members of pRb pocket protein family disrupts normal cell cycle control and presumably antagonises p53-induced apoptosis.

According to Vousden (25) who studied interactions of human papillomavirus transforming proteins with the products of tumour suppressor genes, the oncogenic subtypes of the human papilloma viruses (HPV) also encode oncoproteins that interfere with the control of cell growth and survival in a similar fashion, although HPV uses two different proteins for this purpose, E7 that binds members of the pRb family and E6 that binds p53. Oncogenic HPV subtypes are associated with about 90 percent of cases of cervical cancer, which constitutes some 10 percent of all human tumours. Recently, PCR has permitted the detection of SV40-like DNA sequences in human tumours including osteosarcomas, ependymomas, gliomas and mesotheliomas, raising the possibility that SV40 or a related virus is involved in their etiology. Then he added that "Yet the mere presence of viral DNA does not prove

that the virus is a causative agent; it may just travel along as an inactive passenger". This is an interesting reasoning; one can also re-phrase it that it would be important to actually find out what is the virus doing there and that its presence could be the first indication that it plays some, and even important, role in HPV carcinogenicity. It is a specialty of some medical research that it often negates the causal link but fails to explain the role, if any, of such important indications. Why would it just be "travelling along as an inactive passenger"? Where is the evidence for that assertion?

Waheed et al. (26) provided an answer to this problem by defining the relevance of SV40 T/t antigen expression in established human mesothelioma cell lines deficient for p16INK4a as well as ARF expression. SV40 early region sequences were readily detected in genomic DNA isolated from pleural mesothelioma lines, even though levels of SV40 T/t antigen expression were highly variable. An adenoviral vector expressing an antisense transcript to SV40 early region inhibited T antigen expression and mediated significant growth inhibition and apoptosis in T-antigen-positive mesothelioma cells and SV40-transformed COS-7 cells. Abrogation of T/t antigen expression coincided with enhanced p21/WAF-1 expression, suggesting that restoration of p53-mediated pathways may have contributed to the growth inhibition and apoptosis induced by the antisense construct. These effects were not observed after similar treatment of mesotheliomas or lung cancer cells containing no SV40 DNA sequences. Collectively, these data suggest that SV40 oncoproteins contribute to the malignant phenotype of pleural mesotheliomas and indicate that interventions designed to abrogate their expression may be efficacious in the treatment of individuals with these neoplasms.

They concluded that data presented in their study suggest that SV40 oncoproteins contribute to the malignant phenotype of human pleural mesotheliomas cells and imply that strategies designed to inhibit their expression may be efficacious in the treatment of individuals with MPMs (and possibly osteosarcomas or brain tumours) that harbour SV40. Conceivably 9p allelic deletions and expression of SV40 oncoproteins are complementary, rather than redundant, events that simultaneously inactivate the Rb and p53 tumour suppressor pathways during malignant transformation in pleural mesothelioma. The relevance of SV40 oncoproteins in the pathogenesis of malignant pleural mesotheliomas should not be underestimated, irrespective of their levels of expression.

An extremely important finding of unique strains of SV40 in commercial polio virus vaccines from 1955, not readily identifiable with current testing for SV40 infection, was published by Rizzo et al. (27).

They wrote that SV40 was first identified as a contaminant of polio vaccine used from 1955 until 1963. "Recently, SV40 was detected in several human tumors. The virus identified in human tumors often contained only one 72-bp enhancer in the regulatory region, in contrast to the SV40 originally isolated from poliovaccines, which contained two 72-bp enhancers. The origin of virus with one 72-bp enhancer in humans was unknown, because it was thought that these viruses were not present in poliovaccines. It was thought also that all poliovaccine vials produced from 1955 until 1963 had been discarded, thus the possibility that one 72-bp virion which contaminated those vials could not be tested. We unexpectedly obtained what appear to be the last available vials of poliovaccine produced in 1955. In these vials, we detected and sequenced SV40 containing only one 72-bp enhancer in the regulatory region. The tissue culture cytoplastic test currently used in the United States to screen oral poliovaccines was designed to detect rapidly proliferating SV40 virions containing two 72-bp enhancers. We found that this test is not sensitive enough to detect low amounts of the slow-replicating SV40 virions, containing one72-bp enhancer. This virus was easily detected in the same cells by immunostaining and PCR. Twelve current vials of poliovaccines tested uniformly negative for SV40, suggesting that the precaution of preparing poliovaccines from kidneys obtained from monkeys bred in isolated colonies prevented SV40 contamination. Our data demonstrate that humans were exposed to SV40 viruses with both one 72-bp and two 72-bp enhancers SV40 through

contaminated vaccines. Our data also suggest that instead of cytopathic tests, immunohistochemical, and/or molecular studies should be used to screen poliovaccines for SV40 to completely eliminate the risk of occasional contamination."

[The usual disclaimer followed, that "The development and distribution of poliovaccines that began in the early 1950s have virtually eliminated paralytic poliomyelitis and have been of unquestionable benefit to the entire human race." How untruthful this statement is, considering the administrative machinations and, in effect, confusion caused by the US health authorities by re-defining the disease poliomyelitis and introducing what can best be described as desk-top statistics not supported by a meaningful research.]

Then Rizzo et al. (27**)** continued that questions concerning the safety of the polio vaccines were raised in 1959 when Bernice Eddy found that hamsters injected with polio vaccine preparations developed sarcomas and suggested that a virus was contaminating polio vaccines.

Sweet and Hilleman (7) reported that a monkey virus called SV40 contaminated both Salk and Sabin polio vaccines and caused the tumours observed by Eddy in hamsters.

The facts behind this unqualified disaster are that polio vaccines were produced in kidney cell cultures derived from rhesus, green, and patas monkeys (Butel and Lednicky (28)). Rhesus monkeys are natural hosts of SV40, and, in captivity, related species caged with infected animals, including the cynomolgus macaque and African green monkey, are also easily infected. SV40 infection appears harmless in immunocompetent hosts, indicating that monkeys carrying the virus showed no obvious signs of illness and thus were not excluded for use in vaccine production. What use is all that medical technology when it fails to detect and disclose the reality?

I'd like to make a parenthesis here: the above described dangerous limited thinking is characteristic of medical researchers who are unable to realise that: l. Monkey organs contain monkey viruses, and 2. Just because the researchers do not see something it does not mean that it does not exist. Absence of evidence is not the same as evidence of absence. They continue to think this way with disastrous results for the consumers of their products, reflected in post-marketing tragedies.

Millions of people, particularly children, were exposed to SV40 contaminated vaccines. The authors, as well as other pro-vaccinators, then asserted that such contamination only occurred in vaccines produced from 1955 to 1963. [Adenovaccines 3 and 7, distributed to a limited extent among the military and civilian personnel between 1961 and 1965, were also shown to be contaminated with SV40 viruses.]

The published facts show otherwise. Gerber et al. (9) reported on the problems with inactivation of vacuolating virus SV40 by formaldehyde. They referred to Sweet *and* Hilleman (7) who then just reported a discovery of a new virus of rhesus and cynomolgus monkey origin. They asserted that unlike other simian viruses this agent failed to cause detectable cytopathogenic changes in cell cultures of rhesus and cynomolgus monkey kidneys where it multiplied to relatively high titers. However, it did cause cytopathic effects in African Green monkey, Cercopithecus aethiops, characterised by cytoplasmic vacuolations which coalesce with eventual destruction of the cells. For this reason the virus has been named vacuolating virus and has been designated SV40 by Hull.

In their report, Gerber et al. (9) described studies on formaldehyde inactivation of SV40 and its detection in some lots of poliomyelitis and adenovirus vaccines. They were critical of Sweet and Hilleman's method of inactivating SV40 by subjecting it to formaldehyde treatment for only ten days, and based on a very small, indeed inadequate, virus titre. Referring to their Figure 1, they wrote "The results in this study indicate that the course of treatment of SV40 with 1:4000 formaldehyde was

characterized 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. The slope of the linear portion of the curve approximately paralleled the one described by Sweet *and* Hilleman (7). Their failure to detect viable virus after 46 hours of the inactivation is undoubtedly due to the fact that the titer of the virus suspension at the start was 2.25 logs lower than the one used in this study. Therefore the infectivity of the samples taken after 46 hours of inactivation fell below detectable levels when 0.2 ml volumes were used for assay tests. This has been indicated by the hypothetical projection (dotted line Figure 1) of their curve. Furthermore, Sweet and Hilleman observed their assay tests for a period of only 10 days. In our experience there was a marked delay in the appearance of cytopathic effects (CPE) caused by formaldehyde treated SV40. In most instances initial CPE did not appear until the 11th day and the final infectivity titers were reached on the 13th and 14th day. This observation is similar to the findings by Bottiger et al. (5) with formaldehyde-treated poliovirus. Hull's (6,7) data on rate of formaldehyde inactivation of the simian viruses known at that time revealed that all of the these agents were inactivated more rapidly than poliovirus, with the exception of SV12 and SV27 which were destroyed at a rate similar to that for poliovirus". [Needless to say, these findings, again, provide further evidence for the validity of my discovery of the critical days established and documented during monitoring with Cotwatch breathing monitor (Scheibner (29)]. They concluded that "In retrospect, therefore, one can assume that large groups of population in this country and abroad must have been injected with varying amounts of SV40 during the course of immunization with formalinized poliomyelitis and adenovirus vaccines". [Considering the year of the publication of Gerber et al.'s article – 1961 – one can, perhaps, forgive them for their naïve and overoptimistic suggestion that no harmful effect can be attributed to vacuolating virus within the limits of the observation period. It is unforgivable today]. They also cautioned that, even though "Furthermore, oral or intranasal (10) administration of SV40 in man results in viral multiplication without causing illness", at the same time they also wrote "While short term studies of the pathogenicity of SV40 in laboratory animals have been reported as negative (1), the long term effect in newborn animals remains to be determined." The history proved them both wrong and right in the pandemic of delayed onset of a variety of characteristic SV40 cancers.

Fenner (30) reported on the phenomenon of reactivation of animals viruses. He wrote "It is still a common practice among medical men to speak of "killed" and "live" viral vaccines, and the everyday meanings of the terms are clear enough. But, as I shall demonstrate, virologists now recognize a variety of situations in which "killed" virus may multiply and produce new infectious virus. They have therefore discarded the term "killed" and adopted the word "inactivated" to replace it. Even "inactivated' however, is used in a restricted sense; it refers to the loss of viral infectivity – that is, to the inability of virus particles to multiply and produce new infectious virus in susceptible cells, when these cells each receive only single particles of the inactivated preparation, and no other virus particles or derivates of."

There were three main objectives in the inactivation of viral infectivity:

1. The necessity to sterilize objects contaminated with viruses;
2. The understanding of the mode of action of antibodies and other naturally occurring components of biological systems which could play a part in recovery from viral infections or protection against them; and
3. The preparation of vaccines composed of non-infectious but antigenically potent viral material.

Fenner (30) continued "More recently, it was discovered that inactivation was sometimes reversible – that is, inactivated virus could be rendered infectious again. This was first recognized with virus inactivated by treatment with antibody. Apart from reversal of neutralization by simple dilution virus "neutralized" by antibody could be rendered infectious again by treatment at low pH by ultrasonic vibration, by papain digestion, and by treatment with fluorocarbon. Other examples of extracellular

inactivation and reactivation are the reversal of the toxic effects of mercuric chloride by hydrogen sulphide, and the reactivation of formalin-treated phage T3 by incubation with asparagines".

Of equally great interest, from the point of view of the analysis of viral structure and function, was the discovery by Luria (1947) [quoted by Fenner 30] that bacterial viruses inactivated by ultraviolet (UV) irradiation could undergo reactivation. Ionising and non-ionising radiation and radiomimetic chemicals were shown to inactivate the infectivity of viruses primarily by damaging their genetic materials (although they all affect other components of the virion also).

Sometimes, the genetic damage could be directly repaired, as in photoreaction of UV damages, but usually reactivation involved the intracellular participation of genetic material from more than one virion. Reactivation of UV irradiated phage by multiple infection of susceptible bacteria has been termed multiplicity reactivation; the rescue of markers from irradiated phage of simultaneous infection of the same cell with UV damaged phage and an active unlike phage was called cross-reactivation or marker-rescue.

Fenner (30) continued that although a great deal of information has been accumulated on the inactivation of animal viruses…little attention has been given to their intracellular reactivation. This stems principally from the lack of sufficiently precise quantitative methods and to some extent from the lack of suitable genetically marked viruses. Fenner (30) quoted the examples of multiplicity reactivation of UV-irradiated influenza virus and irradiated vaccinia virus, undergoing both multiplicity and cross-reactivation. Others have demonstrated that some rabbits inoculated with a mixture of heat-inactivated myxoma virus and active fibroma virus died of myxomatosis.

Perhaps the most telling is an article by Kops (31) published in a journal called Anticancer Research, titled "Oral polio vaccine and human cancer: a reassessment of SV40 as a contaminant based upon legal documents". He wrote *"To date, the scientific literature and research examining SV40 and cancer-related diseases has been based upon an assumption that SV40 was not present in any poliovirus vaccine administered in the United States and was removed from the killed polio vaccine by 1963. The basis for this presumption has been that the regulations for live oral polio vaccine required that SV40 be removed from the seeds and monovalent pools ultimately produced in the manufacturing process. The Division of Biologic Standards permitted an additional two tissue culture passages--from three to five--in order to allow manufacturers the ability to remove this contaminant from the oral poliovirus vaccines then awaiting licensure. The confirmation of the removal by one drug manufacturer, Lederle, has been made public at an international symposium in January 1997, where its representatives stated that all of Lederle's seeds had been tested and screened to assure that it was free from SV40 virus. However, in litigation involving the Lederle oral polio vaccine, the manufacturer's internal documents failed to reveal such removal in all of the seeds. The absence of confirmatory testing of the seeds, as well as testimony of a Lederle manager, indicate that this claim of removal of SV40 and the testing for SV40 in all the seeds cannot be fully substantiated. These legal documents and testimony indicate that the scientific community should not be content with prior assumptions that SV40 could not have been in the oral polio vaccine. Only further investigation by outside scientific and independent researchers who can review the test results claimed in the January 1997 meeting and who can conduct their own independent evaluations by testing all the seeds and individual mono-valent pools will assure that SV40 has not been present in commercially sold oral poliovirus vaccine manufactured by Lederle."*

Kops' (31) further comments are equally important and should send shivers down the spine of every pro-vaccinator and especially the parents of babies who are the ultimate recipients of these dangerous, and still contaminated, vaccines.

At the conference held in January 1997 at which the NIH, the FDA and the CDC met in Bethesda, Maryland joined by scientists from around the world, Dr MR Hilleman of the Merck Institute reviewed the historic record of when SV40 was discovered as a polio vaccine contaminant and what was done to assure its removal differentiating between oral and killed polio vaccine products (7). That meeting was also reviewed by Carbone et al. (21).

As mentioned above, Olin and Giesecke (8) tried to demonstrate no impact of potential exposure to SV40 in polio vaccines used in Sweden during 1957 on cancer incidence rates from 1960 to 1993. They wrote that in 1957, when national polio vaccination was started in Sweden, potentially contaminated vaccine was given to approximately 700,000 individuals, mainly preschool and school children born 1946 to 1953. From 1958, a Swedish inactivated polio vaccine was exclusively used, which has been claimed to be free of SV40. Let's pause here a bit: Firstly, those original polio vaccines were definitely contaminated by SV40, not just potentially contaminated. Secondly, one should ponder about their careful choice of words about the Swedish-produced (post-1958) polio vaccine; they wrote "which has been claimed to be free of SV40". There is a huge difference between writing "which has been free" and "which has been claimed to be free". Thirdly, under the subheading "Findings" Olin and Giesecke (8) wrote "For osteosarcoma and brain ependymoma overall age standardised incidence rates were essentially unchanged between 1960 and 1993, and age specific rates were similar in the exposed and unexposed male and female cohorts". "Moreover, during the same period overall age standardised incidence rates in males of brain cancers increased from 9.0 to 13.1 and in pleural mesotheliomas from 0.2 to 2.1 per 100,000". Olin and Giesecke (8) emphasised that "None of these increased rates was associated with the exposed cohorts". They interpreted this surprising (and no doubt uncomfortable) finding as follows: "The use of potentially SV40 contaminated inactivated polio vaccines in Sweden has not been shown to be associated with increased cancer incidence. However, the exposed cohorts have not yet reached the age of increased risk of brain cancer or mesotheliomas." Their Table 1 and Table 2
and Figures 1 and 2 show something different, namely the ever increasing rates of two categories (brain cancer and ependymoma) and basically steady occurrence of osteosarcoma and pleural mesotheliomas in age standardised cancer incidence rates, and ever increasing rates of pleural mesotheliomas particularly in males (age standardised incidence) and ever increasing rates of mesotheliomas (particularly in males) by age groups. The meaning of the increasing rate in age groups up to some 80 years shows the danger of infection (transfer) by SV40 to parents and grandparents of the vaccinated children who were handling the nappies of their vaccinated children and grandchildren. The correct overall meaning of the situation in Sweden as described by Olin and Giesecke (9) is that the Swedish produced **inactivated** polio vaccine continued to be contaminated by SV40. The keyword here is **inactivated**. As demonstrated above, inactivation of SV40 with formaldehyde (or any other means) is incomplete and reversible.

I personally met a Swedish mother who approached me after one of my talks about dangers and ineffectiveness of vaccination in Sweden: her child born in 1997 developed a typical SV40 brain tumour which was laboratory proven. Needless to say, the child was given the Swedish inactivated polio vaccine. She confirmed that Swedish hospitals were full of children with cancers of her child's age. If I rephrase it: Sweden has the highest incidence of typical SV40 cancers in the world.

Strickler et al. (32) published an article on contamination of polio virus vaccines with simian virus 40 (1955-1963) in the US, and the subsequent cancer rates. They concluded that "After more than 30 years of follow-up, exposure to SV40 contaminated poliovirus vaccine was not associated with significantly increased rates of ependymomas and other brain cancers, osteosarcomas, or mesotheliomas in the United States". They illustrated the results of their "research" in Figure 1. To read this figure, one must first be well-aware of its construct: firstly, Strickler et al. (32) plotted the incidence of four cancer categories: A. Ependymoma; B. Brain Cancers; C. Osteosarcoma; and D. Mesothelioma. Except for osteosarcoma (which exhibits the age specific incidence with a maximum

at about 18 years), all categories show clear increases in the incidence. So, does it mean that after polio virus vaccines were allegedly devoid of SV40 the incidence of these categories of tumours increased exponentially and so it was caused by something else than SV40? Not quite. The real meaning of this situation is that whether the polio virus vaccines were alleged or considered SV40-contaminated or not, the incidence of these typical SV40 tumours increased as the age of the population of children given the contaminated vaccines was increasing. The reader is advised to look at the legend: the data on the horizontal line of the graphs indicate the age of the recipients of the contaminated vaccines at the time of the Strickler et al's research. The figure also looks at three cohorts by the alleged exposure to SV40. The interrupted line is the cohort of the recipients born 1947-1952 (exposed as school children); thin line (exposed as infants); and the full line shows the recipients allegedly unexposed. The true meaning of the graph is that it shows the age reached by the four groups at the time of their research; not only the incidence of the SV40 cancers continued to rise, but the so-called unexposed group was only about 27 years old, the exposed as infants group was about 34 years old and the exposed group up to 40 years old. Another meaning of the figure is in showing the delayed onset of these cancers. Far from showing that, as the authors concluded, SV40 was not implicated as the cause of these cancers, their own figure is incriminating the vaccines which continue to be contaminated. Indeed, the so-called "exposed as infants" and "unexposed" groups developed these cancers at about 2-3 and around 10 years of age respectively, indicating that it took about that time for the inactivated viruses to revert back to the original virulence and also multiply sufficiently to cause these cancers. It also reflects changes in the production of polio virus vaccines and the fact that they stopped using wild monkeys and instead used kidneys of the monkeys grown in laboratories with a lower, but still significant and sufficient, viral (SV40) load. And, of course, it also reflects that the method of inactivation carries all the inadequacies as described by Gerber et al. (9) and Fenner (30). Perhaps I should also comment on the use of the word "significant" by Strickler et al. (32). The onset of mesotheliomas is most delayed of all cancers. This could indicate the contributing role of the asbestos exposure. Correctly read, Figure 1 shows that all increases were sustained and significant and the polio vaccine continue to be contaminated by SV40.

Chimpanzee coryza agent (renamed as respiratory syncytial virus RSV) and acute respiratory disease in children

Chanock (33) wrote on the association of a new type of cytopathogenic myxovirus with infantile croup. The viruses produced an unusual "sponge-like" cytopathogenic change in monkey kidney tissue culture, isolated from the pharyngeal swabs of 2 of 12 infants with croup. The infants from whom the agents were isolated and 3 additional patients developed significant increases in neutralising or haemoagglutination-inhibition and complement fixing of all 3 varieties of antibodies during convalescence.

Morris et al. (34) described the recovery of a cytopathogenic agent that produced acute respiratory illness in chimpanzees and possibly in human beings. During October 1955, a respiratory illness characterised by coughing, sneezing, and mucopurulent nasal discharge occurred in a colony of 20 "normal" chimpanzees at the Walter Reed Army Institute if Research. They named it the chimpanzee coryza agent. Chanock (33) wrote on the association of a new type of cytopathogenic myxovirus with infantile croup. The viruses produced an unusual "sponge-like" cytopathogenic change in monkey kidney tissue culture, isolated from the pharyngeal swabs of 2 of 12 infants with croup. The infants from whom the agents were isolated and 3 additional patients developed significant increases in neutralising or haemoagglutination-inhibition and complement fixing of all 3 varieties of antibodies during convalescence.

Morris *et al* (34*)* experimented on human volunteers; they injected them with two viruses: respiratory syncytial (RS) virus prepared for use in monkey kidney cells which were found to be contaminated with SV40, as witnessed by the recovery of RSV and SV40 from the throat swabs. All 8 volunteers

remained free of signs and symptoms of illness in the month following inoculation. Two of eight subjects developed respiratory disease.

Chanock et al. (5) and Chanock and Finberg (6) reported on two isolations of a similar agent from infants with severe lower respiratory illness (bronchopneumonia, bronchiolitis and laryngotracheobronchitis). They also found serological evidence of infection in a number of additional children from whom they could not isolate the virus. This infection was shown to occur in a significant portion of outpatients with respiratory infections. The two viruses were indistinguishable from an agent associated with an outbreak of coryza in chimpanzees (CCA virus) studied by Morris et al. (34). A person working with the infected chimpanzees subsequently experienced respiratory infection and, although virus isolation attempts were unsuccessful, a rise in antibody for CCA virus was observed during convalescence. They mused that there was a suggestion that CCA was a virus of human origin which produced an outbreak of mild respiratory illness when introduced into a susceptible population of chimpanzees. They proposed a name for this agent “respiratory syncytial virus”.

Beem et al. (36) isolated the virus from inpatients and outpatients seen in the Bobs Roberts Memorial Hospital for Children of the University of Chicago during the winter of 1958-1959, and provided additional evidence that this agent is associated with acute respiratory illness in humans. They named it Randall virus. It had an unusual cytopathic effect on H Ep-2 cells characterised by the formation of extensive syncytial areas and giant cells. Over the course of the next five months, 48 similar agents were isolated from 41 patients. Complement-fixation tests and crossneutralisation tests using serums from rabbits made hyperimmune to Randal virus and the Long and Sue strains of chimpanzee coryza virus showed antigenic similarities between all three viruses. The youngest patient from whom the Randall virus was isolated was 3 week and the oldest 35 years. The clinical diagnoses included acute respiratory disease, croup, bronchiolitis, pneumonia and asthma. Randal virus, antigenically similar to chimpanzee coryza virus produces infection and illness in humans. It could be demonstrated in respiratory-tract secretions of children with acute respiratory disease but not from such material obtained from a control group without respiratory illness. The occurrence was not linked to any other microorganisms, such Myxovirus parainfluenza, types 2 and 3, of beta-hemolytic streptococci, Diplodoccus pneumoniae and H. influenzae. The illnesses of patients from whom the virus was isolated ranged from mild coryza symptoms to fatal bronchiolitis. The rate of isolation was particularly high among infants less than six months of age (46%), compared with older patients (16 %). Beem et al. (36) hypothesised that the difference in age distribution was due to a smaller likelihood of isolating virus from infected older patients. In general, the clinical illness of infants was more severe and protracted. The reader now knows better.

In Australia, Lewis et al. (37) and Forbes et al. (38) isolated further viral specimens which corresponded in tissue-culture behavior and clinical association to respiratory syncytial virus. They wrote that for several years, prior to July 1960, the influenza and parainfluenza virus groups were the predominant causes of epidemic respiratory infections. In July 1960, the pattern changed abruptly, with a sudden increase in number of infants admitted with bronchiolitis and bronchitis and a concomitant increase in infants with pneumonia. These were not paralleled by an increase in croup or by a similar increase in pneumonia in older age groups. Prior to this, the diagnosis of bronchiolitis was infrequent. Two hundred and forty-four children under the age of four years were admitted to hospital with the diagnosis of bronchiolitis, bronchitis or pneumonia during this epidemic. Fifty eight percent were less than 12 months old, and patients under the age of four years dominated the group of respiratory infections at that time. The major clinical feature of the group was the large proportion of infants with “wheezing” or “bronchospasm” associated with other evidence of pulmonary infection. This epidemic pattern was repeated in April 1961, when 237 patients under the age of four years were admitted, of whom 55% were aged less than one year. It was shown by Dr Ferris and his colleagues, that while the croup in 1960 was due predominantly to parainfluenza virus infection, and that in 1961

to parainfluenza and influenza type B viruses, the infants with brochiolitis and severe bronchitis yielded a respiratory syncytial agent not previously isolated. The majority of infants had a history of two to three days' respiratory illness, often coryzal in onset. In milder cases, the diagnosis of bronchitis was used, but there were many more severe infection, with dyspnoea with expiratory wheezing (different from that seen in croup), tachycardia in all cases, with inspiratory rhonchi and rales. All this indicated inflammatory narrowing rather than muscular spasms of bronchioles. There was a disparity between seriousness of symptoms and temperature, which was either normal or only slightly elevated. There was also a relative absence of x-ray findings. Some cases had a paroxysmal cough. Two deaths occurred during each epidemic. According to Kravetz et al. (12), during their study it was found that the simian kidney vacuolating virus SV40 was present in the RS virus inoculums and caused inapparent infection in some volunteers. [It would be interesting to find out how many later on developed any characteristic tumours caused by SV40 virus. Unfortunately, we shall never know].

Rogers (39) wrote an essay on the changing pattern of life-threatening microbial disease. He wrote that the hope that antimicrobial drugs would abolish infections as a cause of death has not been adequately realised. Microbial infections continue to pose life-threatening problems and antibiotic drugs do not offer susceptible human beings significant protection from certain types of microbial disease. During the course of intrahospital survey it became apparent that the specific microbial causes of serious disease in hospital practice and the actual contribution of infection to deaths in hospitalised patients in 1959 were somewhat different from those suggested in the current literature. In this paper, Rogers (39) analysed the incidence and nature of infections contributing to deaths on the medical service of The New York Hospital, contrasting the period 1957-1958 with the period 1938-1940. The clinical records, laboratory data ad autopsy protocols of 200 consecutive medical-service patients subjected to post-mortem examination between 1938 and 1940 and an equal number of those dying in 1957 and 1958 were analysed in detail. While pneumococcal infection played a major role in the earlier pre-antibiotic times. Pneumococcal infection played a minor role in the antimicrobial era. Streptococcal infection was directly responsible for the deaths of 14 patients during 1938-1940. Significant antecedent disease was present in all 14 patients. In contrast, streptococcal infection did not play a part in any of 200 deaths occurring during 1957-1958. The same trend was observed with tuberculous and staphylococcal infections.

An inactivated RS virus vaccine has been developed and tested in children. Kapikian et al. (40), Fulginiti et al. (41), and Chin et al. (42) reported on the results of field trials. They all concluded that the vaccine not only failed to offer protection, but also induced an exaggerated, altered clinical response to naturally occurring RS virus infection in the younger vaccinees, as nine (69%) of 13 vaccinated and only 4 (9%) of nonvaccinated Harrison Cottage residents 6-23 months of age developed pneumonia. The paradoxical effect of vaccination suggests that serum antibody may play an active role in the pathogenesis of RS virus disease (Kapikian et al. (40)). According to Fulginiti et al. (41), three injections of an aquaeous trivalent parainfluenza vaccine failed to provide significant protection against natural disease caused by the parainfluenza viruses, while with exposure to natural infection, an unexpected increase in the incidence of RS virus illness requiring hospitalisation among vaccines was observed as compared to control groups. This difference was most significant in the 6-11 month old group (13.7% were hospitalised with RSV illness as compared to only 0.86% of an age-matched unvaccinated control group. They ascribed this to delayed hypersensitivity caused by the vaccine.

Chin et al. (42) reported very high attack rates of parainfluenza virus, types 1 and 3 and RS virus during the study period of infants and children in both vaccine groups. A protective effect was not demonstrated for either vaccine. Infants who received the RS virus vaccine and who subsequently became infected with RS virus tended to have a more severe clinical illness than infants who did not receive the vaccine.

Additionally, Forsyth (43) described a development of delayed dermal hypersensitivity in Guinea Pigs vaccinated with inactivated RSV vaccines. As with alum containing DPT vaccine (in contrast to aequeous vaccines), animals vaccinated with bovine kidney parainfluenza vaccine developed delayed dermal hypersensitivity when skin tested with bovine kidney RS vaccine. Partially purified RS antigen A and B did not elicit delayed dermal hypersensitivity.

Going back to Rogers (39): there was an impressive increase in the number of enterobacterial infections of life-threatening concern observed during the latter period of the study (1957-1958). Only 9 patients dying during 1938-1940 had important enterobacterial infections. In contrast, 22 patients with infections due to gram-negative bacilli were observed during the post-antimicrobial period, and indeed such infections represented the most common microbial cause of death on the medical service during this period. There was also a shift in the place where infection was acquired. During the pre-antimicrobial era, most infections were acquired before admission to hospital, while in the post-antimicrobial era the vast majority of infections arose in hospital.

Mycotic infections, were also more frequent in patients hospitalised in 1957-1958, compared with the post-antimicrobial era; infections produced by strains of Candida albicans becoming a major problem. Unusual generalised clostridial infections arose as serious illness. In contrast to this, apparently, deaths due to infections produced by pneumococci, streptococci and tubercle bacilli diminished in the post-antimicrobial era.

In summary, Rogers (39) concluded that his figures support the impression that antimicrobials have not dramatically altered the risk of, or mortality resulting from, endogenous infections arising in sick, hospitalised patients. Microbial disease remains a significant medical problem. What is puzzling to me is the total disregard for these findings by medical system and the continued mass use, and the development of many more, antibiotics since 1959 to this day by the drug companies.

Right through the seventies, eighties, ninetieth and to this day, a great number of papers have been published dealing with the pandemic of bronchiolitis caused by respiratory syncytial virus in infants and young children. Some started in the present, without mentioning the original virus name chimpanzee coryza virus, others started from the original published research and mentioned chimpanzee coryza virus. They all agreed, though, that RSV became the most prevalent cause of bronchiolitis in very young children, with many cases serious and even fatal. All represent jigsaw pieces in a tell-tale story. One of such publications is that by Levy et al. (44) who wrote, “Respiratory syncytial virus (RSV) is the most common cause of lower respiratory tract infection (LRTI) in infants and young children. Infection with RSV is a major health problem during early childhood and primary RSV infection occurs most often between the ages of 6 weeks and 2 years. Approximately one half of all infants become infected with RSV during the first year of life and nearly all by the end of their second life.” This is very strongly indicative of vaccines being the source of RSV infection; in the US, the first vaccine is given at 6 weeks. They also wrote that in the US each year, approximately 100,000 children are hospitalised at an estimated cost of $300 million. More than half of those admitted for RSV bronchiolitis are between 1 and 3 months of age. A real tell-tale story incriminating vaccination.

Prober and Sullender (45) wrote that respiratory syncytial virus, “first isolated in 1955 [sic] from adolescent monkeys with severe coryza illness, was initially called chimpanzee coryza agent. Soon thereafter, the virus was isolated from children with bronchiolitis and pneumonia and was recognized as the most common cause of annual winter epidemics of lower respiratory tract infections throughout the world. 2,3 Because the virus had a proclivity for the respiratory tract and formed multinucleated giant cell syncytia in tissue cultures, it was renamed respiratory syncytial virus in 1957.” They also wrote that RSV is an enveloped RNA virus in the family Paramyxoviridae. There are two major antigenic groups of RSV, designated A and B.

Shann et al. (46) wrote that acute respiratory tract infections kill 4 million children every year in developing countries.

Simoes (47) wrote that "since it was identified as the agent that causes chimpanzee coryza in 1956,1 and after its subsequent isolation from children with pulmonary disease in Baltimore, USA, 2 respiratory syncytial virus (RSV) has been described as the single most important virus causing acute respiratory-tract infections in children The WHO estimates that of the 12.2 million annual deaths in children under 5 years, a third are due to acute infections of the lower respiratory tract. Streptococcus pneumoniae, Haemophilus influenzae, and RSV are the predominant pathogens. Then he, surprisingly, wrote that A formalin-inactivated RSV vaccine, tested in the 1960s, 4 was immunogenic, with high-rates of seroconversion. Despite this immunogenicity, vaccinated children were not protected from subsequent RSV infection. Furthermore, RSV-naïve infants who received formalin-inactivated RSV vaccine, and who were naturally infected with RSV later, developed more severe disease in the lower respiratory tract than a control group immunized with a trivalent parainfluenza vaccine. He philosophised that although the age distribution of RSV infection in children in developing countries is similar to that in developed countries, older children are more severely affected in developing countries, perhaps reflecting the (unidentified) crowding, indoor smoke pollution, and malnutrition may play a part in the development of a more severe disease. The WHO-sponsored field studies were done in Indonesia, Ethiopia, Guinea Bissau, Mozambique, Nigeria, and South Africa. Data from ten developing countries (Argentina, Colombia, Guatemala, Kenya, Nigeria, Pakistan, Papua New Guinea, the Philippines, Thailand, and Uruguay, showed that the most frequent cause of LRT infection was RSV (70% of all cases). Need less to say, these are also the countries with intense polio vaccination. It is no secret that the source of RSV is the contaminated polio vaccines in both developing and developed countries.

Bent et al. (48) performed a meta-analysis of randomised controlled trials to estimate the effectiveness of antibiotics in the treatment of acute bronchiolitis. They concluded that their meta-analysis suggests a small benefit from the use of the antibiotic erythromycin, doxycycline, or trimethoprim/sulfamethoxazole in the treatment of acute bronchitis in otherwise healthy patients. "As this small benefit must be weighed against the risk of side effects and the societal cost of increased antibiotics resistance, we believe that the use of antibiotics is not justified in these patients."

Nystad et al. (49) wrote that children who attend day care have an increased risk of asthma with early infections as a mediator risk. They have also detected a dose-response relationship between type of day care and infection. Their study was also supported by a study among Finnish children, which revealed that care in day care centres was a determinant of acute respiratory infections in children under 2 years of age. Family day care did not essentially increase the risk. What they did not mention is that children attending day care centers are more likely to be fully vaccinated.

Waris (50) wrote that RSV, a member of the Paramyxoviridae family, genus Pneumovirus, is considered the major pathogen causing severe lower respiratory tract infections among infants and children. Yearly epidemics of RSV have been occurring world wide with seasonal regularity. In the Scandinavian countries a major outbreak alternates with a minor one every second year, a phenomenon, apparently, not reported elsewhere. In at least some parts of the United States, an alternation in timing but not in the size of the epidemics are evident. It has been suggested that the variations on the epidemiological peaks are caused by interference between RSV, parainfluenza viruses, and influenza virus. Time-resolved fluoroimmunoessay with monoclonal antibodies distinguishing between respiratory syncytial virus (RSV) group A and B strains were used to analyze their prevalence in Finland during 1981-1990 among 3285 patients with laboratory diagnosis of RS, most of them hospitalized. The group typing of antigens in 680 RSV-positive nasopharyngeal aspirates showed a regular alternation of group prevalence, following the cycle occurrence of the

virus. Group A predominated in 73%-90% of specimens from 1981-1982, 1985-1986, and 1989-1990, whereas group b predominated in 70%-100% of specimens from 1983-1984 and 1987-1988. This indicated that children more than 6 months of age during the first infection were more resistant to severe reinfection with the homologous than with the heterologous group virus. The study shows that group antigenic variation of RSV has a significant effect on the epidemiology of the virus.

Singleton et al. (51) characterised the epidemiology of Alaska native children hospitalised for RSV infections. They reviewed records of hospitalisations during the winter season of 1991 to 1992 and 1992 to 1993 at a hospital in Anchorage and a rural hospital in the Yukon Kuskokwim Delta (YKD) region of southwestern Alaska. The median age of hospitalisation for RSV infection was 2 months of age for YKD residents and 4.5 months for Anchorage residents. Sixteen percent of the hospitalised YKD children were less than 1 month of age, whereas the same was true for only 3% of the Anchorage children. Eight percent of the YKD patients required mechanical ventilation, whereas none of the Anchorage patients required ventilation. The median hospital stay was 4.8 days for YKD patients and 3.2 days of Anchorage patients. The extremely high hospitalisation rate especially among very young infants in the rural YKD region points to a need for early preventative efforts.

Among the reasons mentioned by the authors for this difference, was overcrowding (with 3-4 children sleeping in the same bed), lack of flush toilets and town water, indoor smoking and younger age at infection (under one month of age). In my opinion, this facilitated early RSV infection from older siblings already infected with RSV from their vaccines. Very few, if any, of the native children are unvaccinated due to lack of knowledge of vaccine dangers and poor educational level.

Translated into absolute figurers, we are talking about thousands of children affected by RSV LRTIs. The authors list 91,000 hospitalisations with 2000 death annually in the US, in the peak age between 2 and 5 months. This is the vaccination age.

Hadi et al. (52) studied the effect of vitamin A supplementation on growth with varied results. They analysed data from a randomized, double-marked, placebo-controlled trial to examine the role of infections and diarrhea in modifying the growth response to vitamin A supplementation. A single high dose of vitamin A or placebo was given every 4 months to 1405 children aged 6-48 months, and 4430 child treatment cycles were used in their analysis. Their results showed that vitamin A supplementation improved the linear growth of children who have a low intake of vitamin A but this effect was muted by respiratory infections.

Shay et al. (53) studied bronchiolitis-associated mortality and estimates of Respiratory Syncytial virus-associated deaths among US children under 5 years of age. From 1980 through 1996, the RSV proportion of LRD hospitalisations increased from 22% to 47%. They revised the original estimate of 4500 deaths due to RSVI down to 519 RSV-associated deaths annually during the study period.

Langley et al. (54) evaluated temporal trends in hospitalisation for bronchiolitis found among Canadian children for 1980-2000. The rate of hospitalisations increased in all provinces over the 2 decades for all age groups but was highest in those aged less than 6 months. Because a concurrent increase in other respiratory diagnostic codes was not seen, it is unlikely that physician practice variation could explain this consistent trend over almost 2 decades, which may indicate a change in disease prevalence or severity.

Hoebee et al. (55) examined the association of variants of genes encoding interleukin (IL)-4 and the IL-4 receptor alpha chain (IL-4R alpha) with respiratory syncytial virus (RSV) bronchiolitis in hospitalised infants. Polymorphisms in IL-4 (C-590T) and IL-4R alpha (I50V and Q551R) were genotyped by restriction fragment-length polymorphism analysis. Control subjects included parents of the hospitalised children (for the transmission/disequilibrium test), and a random population sample

(for the case-control study). Results were also analysed in a combination of these two tests, using Fisher's method. The IL-4 590T allele was found more frequently among children hospitalised with RSV than expected in the case-control and combination tests. Higher frequencies of both the IL-4 590T allele and the IL-4 R alpha R551 allele were found in children who were more than 6 months old when hospitalised for RSV infection compared with the control group or with the younger than 6 months olds. These results indicate that gain-of-function variants of T helper type 2 cytokine genes may play a role in increasing the severity of RSV disease, which appears more pronounced after the first half-year of life.

Such results are not surprising considering that by 6 months of age most infants are given three doses of multiple vaccines which substantially suppress and damage their immune system.

Seven viruses isolated from the vervet monkey

Malherbe and Harwin (56) described seven new viruses isolated from the vervet monkey, Cercopithecus aethiops pyerythrus, the animal most commonly used in South Africa for the laboratory isolation and maintenance of strains of poliomyelitis virus, and for the production of poliomyelitis vaccine. They refer to the introduction by Enders, Weller and Robbins of cultures of non-nervous tissue for the growth of this virus, various tissues from the vervet monkey have been used, the most suitable tissue for the large-scale cultures, and also the most sensitive for the isolation of poliomyelitis and a number of other human viruses, being renal epithelium prepared according to the trypsinisation technique of Youngner (57). They also wrote that it is known that viruses may accompany tissues cultivated in vitro, manifesting themselves after variable periods in the cultures. The first of the group of agents now classified as adenoviruses was recognised through the occurrence of spontaneous degeneration in cultures of human adenoids).

The subject of their paper was to deal; with a group of seven SA (probably meaning South African) which have been encountered since 1954 in the laboratories of South African Poliomyelitis Research Foundation. Six of these viruses were isolated from uninoculated cultures of renal tissue from the vervet monkey, and one from the faeces.

SA1 produces syncytia with aggregates of nuclei of normal appearance, and is probably identical with the MK virus of Rustigian, Johnson and Reinhart (2).

Cytomegalovirus infections

Cytomegalovirus is described as a ubiquitous herpes virus with capacity to cause a diversity of clinical symptoms (Golden et al. (58). Since effective control of CMV requires intact cell-mediated immunity, the populations at the highest risk for invasive CMV disease include neonates, allograft recipients, and individuals infected with HIV type 1 and elderly.

Hsiung (59) published an essay on latent virus infections in primate tissues with special reference to simian virus. He listed the following latent viruses located in tissues of primates as well as nonprimates. DNA viruses: papova virus group; adenovirus group; herpesvirus group and poxvirus. RNA viruses included picornavirus group; reovirus group; myxovirus group; and pseudomyxovirus group. Under the heading "Possible origin of virus infection in kidney tissues" he wrote that a variety of viruses have been isolated from the kidney tissues of apparently healthy animals, while it yes not known how often and to what extent the kidney is infected. They tried to find out by intranasally infecting monkeys with polio virus type 1. Virus was recovered from the urine 30 min later. The titres of polio virus recovered in the serum fell rapidly during the first 7 hours of sampling. The titres in the urine also fell rapidly and in unison with the titres of the blood samples. Although polio virus is

not one of the viruses involved in latent infections discussed in his paper, he wrote that these studies indicated that viremia occurred in the inoculated monkeys and also in an uninoculated cage mate. Viruria followed immediately. Others (Meyer et al. (60)) showed that intranasal inoculation of SV40 into monkeys resulted in the multiplication of this virus in the nasopharynx and digestive tract. SV40 was recovered from cell cultures derived from kidney tissues of these monkeys 4 to 5 months after initial infection. Ashkenazi and Melnick (61) showed that SV40 could be recovered in the urine of all monkeys inoculated into the kidneys. SV40 persisted in the kidney tissue 6 to 8 weeks after virus administration, regardless of the route of infection. The recovery of SV40 from the kidney tissues succeeded only when the tissue cells were cultivated for a prolonged period of time and not from test culture inoculated with the minced kidney tissue suspensions.

To show the unpredictable nature of research, I will quote some of the comments of Hsiung (59). He wrote that human embryonic kidney (HEK) cell cultures were available commercially and were being used by many laboratories for the propagation of a variety of viruses. During a 3-year period, November 1964 to December 1967, 124 lots of human kidney cell cultures were examined, and seven viruses (6%) were isolated. These included two measles viruses, two foamy viruses, one reovirus, one adenovirus, and one myxovirus. One measles virus isolation was made from a lot of human kidney cell culture from a 3-year old child who had been exposed to measles and had been given 0.6 ml of gamma globulin at the time of exposure. The child died 2 weeks later. The presence of measles virus in this child's kidney cell culture would not have been recognized if the kidney cell cultures would not have been kept for 33 days after planting. This example illustrates the standing problem in research of not keeping the cultures used for whatever reason for a sufficient period of time. A lot of viruses (and other organisms) and their cytopathic effects simply escape detection.

Herpesvirus group viruses were isolated in monkeys of the Old World species represented by herpes B or Herpesvirus simiae produced a naturally occurring mild infection. However, most human cases of B virus infection have been fatal. Hull and Nash (62) showed that 10% of newly caught rhesus monkeys had antibodies to the B virus, and the percentage rose to 60% to 70% when the monkeys were confined in "gang-cages". Other studied indicate that 100% of the experimental monkeys showed B virus antibody rise when the monkeys were housed together over a period of 6 weeks. Vaccine cultured on their kidney were contaminated by all these viruses and then injected into millions of infants all over the world. It is a real horror story.

Just how much longer are we going to let the vaccine producers get away with destroying the human immune system and even the genetic code, and literally get away with murder?

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