"*THE ABCs OF MMRs AND DTPs: IS THERE AN ASSOCIATION BETWEEN VACCINATION AND AUTISM?*" BY ERIC LONDON:

A BIBLIOGRAPHIC ESSAY

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Prologue

The February 28, 1998 issue of the prestigious medical journal The Lancet contained a preliminary report on a study which occasioned much discussion and controversy: "*Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children*," by Andrew Wakefield and a team of British scientists.[1] The British group investigated, initially, a group of twelve children; by January 1998 Wakefield's team totaled fifty-one investigations of children with regressive autistic spectrum disorders and inflammatory bowel disease and, as of July 1998, had at least one year's worth of additional examinations scheduled as parents of similarly autistic children 'queued up.' Intriguingly, the scientists found measles virus proteins in the germinal centers of the children's intestinal lymphatic tissues, along with indications of classic inflammatory bowel disease. The children had previously been vaccinated with the MMR/MR vaccines or had experienced wild measles.

In a recent issue of NAARRATIVE, newsletter of the National Alliance for Autism Research, issue number 3 from fall 1998, appears an article entitled, "*The ABCs of MMRs and DTPs: Is There an Association Between Vaccination and Autism*?" According to the organization's web site description, NAAR is a national nonprofit, tax-exempt organization begun by parents in 1994, dedicated to finding the causes, effective preventive strategies, effective

treatment and, ultimately, the cure for the autism spectrum disorders (www.naar.org). As a member of NAAR's Scientific Advisory Board, Joseph T. Coyle, MD, commented, "*the pervasive developmental disorders have been neglected unduly but are now approachable by serious research efforts given the rapid advances in neuroscience* (www.naar.org : Mission)." NAAR's stated mission is to fund, promote and support biomedical research on autism. "NAAR aims," the site states, "to have an aggressive and far-reaching research program developed with the expert guidance of its prestigious Scientific Advisory Board."

Unfortunately, the "*ABCs*" article in NAARRATIVE newsletter treats pivotal issues using narrowly-selected details from an equally narrow range of resources, and which contains vague allusions, unfounded or false statements, and injudicious speculation. The issues raised in Dr. London's article are significant and should be considered at length by autistic persons (where possible) and their families, as well as the professionals, the pharmaceutical industry, and the units of government whose work and decisions profoundly effect them. This paper is a work-in-progress which is offered in the spirit of scholarly inquiry and critical thought, to assist those attempting to make decisions regarding the vaccination/autism issue. This paper or parts thereof may be reproduced for purposes of study or reference. Concerning endnotes, several reference numbers will appear out of sequence; initially, a traditional reference sequence was adopted--1, 2, etc., appearing consecutively; later in this paper's evolution, a random numbering approach was adopted. Endnote numbers should lead readers to the correct citations, regardless.

[Link to Eric London's article on NAAR's website (NAARRATIVE no. 3, Summer/Fall 1998) at http://babydoc.home.pipeline.com/naar/naar.htm., or www.naar.org : NAARRATIVE.]

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Fundamental issues

The cause(s) of autism

In "*The ABCs…*" London asserts that autism has been proven to be a simple (single-faceted) genetic disorder which begins before birth, and thus is not related in any way to vaccination, but cites only a few selected studies in supporting this idea. In formulating his argument he casts doubt on the validity of the memories and reasoning of parents who observe a period of initial normality in their children, before vaccination. The first major failure of reporting in this article lies in the lack of differentiation between typical, or "Kanner's," autism and atypical or regressive autism, and childhood disintegrative disorder—or any of the other pervasive developmental disorders. The next lies in the implication that only a single causative factor for autism exists—genetics. In fact, the only proven genetic causes of autism to date are well-defined syndromes like the Fragile X chromosome anomaly—which do not explain the autism of any of the children "*ABCs*" concerns. London's '[all] autism is genetic, not immunological' thesis is contradicted in important works from the autism literature, and even in some of the works he cites:

Diagnostic and Statistical Manual of Mental Disorders: DSM-IV, fourth edition, 1994, pages 66 and 69. DSM-IV specifies that "*Autistic Disorder* (defined as "early infantile autism…or Kanner's autism") *must be differentiated from other Pervasive Developmental Disorders… Autistic disorder differs from Childhood Disintegrative Disorder, which has a distinctive pattern of developmental regression following at least 2 years of normal development*. In Autistic Disorder, developmental abnormalities are usually noted within the first year of life," pages 66 and 69 [emphases mine]. [It is important to note that DSM-IV's definition of autistic disorder differs markedly from earlier definitions, and that autistic spectrum subtypes are still very much in the definition stage.]

Cohen and Volkmar, eds., Handbook of Autism and the Pervasive Developmental Disorders, 2nd edition, 1997: Chapter 18, MEDICAL CONDITIONS[:] **Infections**…**Immunological Association**[:] The claim has been made that a small but significant proportion of children develop autism as a result of pre-or post-natal infections—for example, with rubella, cytomegalovirus, herpes simplex, HIV, and so on…Interest in the immune system and autism arises from the various case reports in which infections (and possibly altered immune response) are associated with the development of autism or autistic features" (p. 398).

Margaret L. Bauman and Thomas Kemper's The Neurobiology of Autism (Johns Hopkins Press), 1994, is listed among London's references at the conclusion of "*ABCs*;" the work of Bauman and Anthony Bailey are also used by London as supports for the idea that autism results from a genetic, prenatal maldevelopment of the brain—yet the Introduction to Neurobiology, written by Isabelle Rapin, clearly states, "***missing from this book**, because of the dearth of prospective information**, is a focus on the many potential nongenetic etiologies of the autistic spectrum…*** **Another topic** of interest, because of its potential therapeutic implications, that is **not considered as such in this book is autistic regression.** *At least 40 percent of parents report that their infant or toddler, whose development may or may not have been entirely normal up to then, experienced a regression, usually insidious but occasionally abrupt*, in language, sociability, and play… Development resumes after a plateau…but, in most cases, never returns to its previous level… *Many speculations have been offered…to explain autistic regression: slow viral infection, autoimmune phenomenon,* lack or insufficiency of a growth factor *at a particular time in development... **Data need to be collected to investigate these speculations***" (pages 13-14)**.** Bauman and Kemper resume, in chapter 2, "The Genetics of Autism," with the following passages occurring on pages 30 to 31 of Neurobiology:

Environmental Factors[:] In examining the importance of genetic factors in the etiology of autism, evidence for environmental factors should be reviewed as well. Unfavorable pre-, peri-, and neonatal factors have been shown to occur more commonly in autistic individuals than normal controls….. **Modes of Inheritance[:]** Although the importance of hereditary factors in the etiology of idiopathic autism is well established, particularly genetic mechanisms have not yet been identified…*it is almost certain that autism is an etiologically heterogeneous* [multi-causal] *disorder*. It is known, for example, that autism can develop in association with etiologies as diverse as congenital rubella, tuberous sclerosis, and the fragile X anomaly, *as well as in the absence of any identifiable, co-occurring, etiologically defined condition* [emphases mine]. Among autistic individuals without identifiable, associated etiologic conditions, several points suggest that there may also be genetic heterogeneity. [See also the discussion of T. C. Binstock's research paper, "*Changing the autism paradigm: a critique of Kemper & Bauman's speculations regarding in-utero timing*," in **Factoid fallacies,** below.]

Journal of Autism and Developmental Disorders, vol. 28, no. 5. 1998 (entirely devoted to the genetics of autism; preface by Eric Fombonne): as one would surmise, the general tone of the separate contributions indicate the strong possibility of a genetic component in autism (though the writers rarely differentiate between subtypes), but this research is far from finished, and does not exclude other factors, as London implies. In P. Szatmari, et al., "*Genetics of Autism: Overview and New Directions*," the authors observe that the genetics of autism in its various manifestations is likely to involve the complex interaction of multiple genes, but even in this event "it is too early to say what type of complex genetic disease autism/PDD represents." A variety of factors may interact to produce these conditions (pages 355; 365)—possibly different mechanisms for each 'type' of autism (page 365). The authors venture so far as to say that "certain severe insults" during pregnancy and birth might "play a causative role in the development of PDD: "*For some children, genetic vulnerability may interact with insults on the developing nervous system to lead to autism*." Such insults might lead to PDD even in the absence of genetic factors—for instance, thalidomide exposure or congenital rubella (page 364). The authors do not examine the period of development after birth, but it is likely that the same reasoning could apply to the developing nervous system during the first three years of life—or even later.

Marian Sigman and Lisa Capps' Children With Autism: a Developmental Perspective (Harvard University Press), 1997 asks, in Chapter 8, **In Search of Core Deficits and Causes: "*What Are the Causes of Autism?*"** "*Autism is not a unitary disease with a single etiology,"* the authors state. "*It is a heterogeneous behavioral syndrome found in association with many etiologies…Complications in any part of… development…could cause damage that leads to autism*. *Infections affecting the central nervous system in early life could also have this effect* (pp. 171-2). Evidence for genetic factors is mounting, they note, but "*it is not clear, however, whether all forms of autism are genetically transmitted in the same way* (pp. 172-3)." Syndromes like the fragile X-chromosome anomaly and tuberous sclerosis are distinct from other pervasive developmental disorders. *"Autism accompanied by mental retardation is inherited differently than when not…*" Evidence suggests that multiple genes are involved (pp. 173-4).

C. Gillberg and M. Coleman, The Biology of the Autistic Syndromes—2nd edition, 1992. Chapter 8, GENETIC FACTORS[:] "It is currently believed that a genetic disease underlies 10 to 20 percent of all autism cases…" (page 96); "There is another autism subgroup in which the autistic symptomatology is linked to disease processes currently thought to be, in many instances, of a non-genetic (or at least 'non-inherited') character" (page 103). Chapter 18, INFECTIOUS DISEASES[:] **Postnatal infections[:] *…Can an infection after birth cause an autistic syndrome*?** "…In summary…infectious agents in the prenatal or postnatal period may be a factor in the development of autism. The most common mechanism appears to be a direct toxic effect on brain cells from the infection (encephalitis)… It is of interest that, in reports concerning both rubella and HSV infections, cases with late onset are found. *An infectious aetiology is a strong contender in the differential diagnosis of autistic symptoms…*" (pages 222-4).

Schopler and Mesibov, eds., Diagnosis and Assessment in Autism (1988). Chapter authors Schopler and Michael Rutter note on page 28, "*[Considering] the very fact that the clinical picture of autism can arise from diseases as diverse as congenital rubella, tuberous sclerosis, encephalopathy…cerebral lipoidosis, and neurofibromatosis…it remains quite uncertain whether the…cases with a known pathologic cause represent phenocopies of some other unitary disorder* with (an as yet undiscovered) single etiology…." Page 86 quotes Volkmar and Cohen, "*…it is clear that the preponderance of available evidence suggests the importance of multiple biologic factors acting through one or more mechanisms to produce the autistic syndrome.*" On pages 295-6, chapter authors Watson and Marcus note the importance of a medical assessment paralleling psychological and other testing: "it is important to recognize the possible medical factors associated with autism. A variety of biologic conditions have been documented including…certain viral infections, abnormalities in purine metabolism and intestinal absorption…" The authors refer readers to Coleman and Gillberg, quoted above, for comprehensive information on the medical aspects of autism.

Shirley Cohen, Targeting Autism, 1998, pages 139 and 140: "*There is increasing evidence of immune system abnormalities in autism. A substantial number of reports of research on this subject have appeared in medical journals since the 1980s, and most of these articles present data that appear to support the theory of a connection between immune system dysfunction and some cases of autism…study of immune system abnormalities in autism has attained the status of mainstream medical research*."

The Centers for Disease Control, [1998], "*Vaccines and Autism: Is There a Relationship*?" (http://www.cdc.gov/nip/vacsafe/vac_autism.htm): Notoriously pro-vaccine, the CDC nevertheless says, of autism, "Some prenatal factors included intrauterine *rubella*; tuberous sclerosis; chromosomal abnormalities, such as Down's Syndrome, as well as brain abnormalities, like hydrocephalus. …*postnatal conditions associated with autism* are untreated phenylketonuria, infantile spasms, and *herpes simplex encephalitis*. …Evidence that *genetics is an important, but not exclusive, cause of autism* includes a three to eight percent risk of recurrence in families with one affected child. …*An issue unresolved* [by a working group convened by the National Institutes of Health in 1995] *was the role of immune factors in autism spectrum disorders; it was suggested that studies to clarify the situation are needed*."

Uta Frith, Autism: Explaining the Enigma, 1989/1994, p. 79, "*The theory that psychotic illness can be due to immune dysfunction and/or viral infection has particular justification in the area of Autism. It has been shown …that a virus infection in a young child preceded the onset of typical symptoms of Autism, before which there was a period of apparently normal development…If the central nervous system becomes infected at a critical time, either before or after birth, Autism may result…Of special interest are certain types of virus called retrovirus, which totally integrate themselves in genetic material in the body cells…These can remain dormant for years but from time to time can be reactivated*."

Van Gent, et al. present a review of the literature in the emerging field of "psychoneuroimmunology" through 1997 in "*Autism and the Immune System*," Journal of Child Psychology and Psychiatry, vol. 38 no. 3, March 1997, pp. 337-349. "Over the last 30 years increasing evidence has been found for the existence of complex links between the immune system, the central nervous system and the endocrine system on the one hand, and psychological phenomena…on the other…: prenatal and *early childhood experiences could have prominent effects on the development of the responsiveness of the immune system, with far-reaching and long-lasting consequences for the immune capacity at a later age. Conversely, early derailments in the normal development of immune function, as, for instance, in the induction of autoimmunity in an early phase of the immunological developmental traject, could have important effects on the development of the nervous and endocrine systems…* [Regarding autism,] *two etiologically relevant immune hypotheses in particular have emerged: a viral and an autoimmune hypothesis, which are interrelated (p.345).* The basic neuroimmunologic premise of these hypotheses is that autoimmune and/or viral processes in some way affect the nervous system and alter central nervous system activity" (pages 337-8).

William Shaw, Biological Treatments for Autism and PDD, 1998. Shaw reports finding in children with autism and PDD none of the signs characteristic of known, inborn (genetic) conditions causing metabolic disorders (pages 31; 35-37; 68-9; 129). On pages 103-4, "**Role of immunizations in causing immune deficiencies**[:] "In several cases, electron microscopy has revealed live measles virus in the intestinal lining of children with autism, raising the possibility that the MMR may actually be responsible for some of the gastrointestinal abnormalities common in children with autism." Shaw also notes "some interesting parallels between autism and tetanus," citing Ellen Bolte's paper, "*Autism and Clostridium Tetani: An Hypothesis*" [Medical Hypotheses, vol. 51, 1998, pages 133-144] (Shaw page 22).

H. H. Fudenberg, NeuroImmuno Therapeutics Research Foundation, "*Classic Infantile Onset Autism is an Autoimmune Disease*," http://members.aol.com/nitrf/autism1.htm, accessed May 20, 1998:[2] In the process of describing transfer factor therapy studied in 40 autistic patients, Fudenberg notes, "The gene for classic autism has been localized to human chromosome-6, the site of human immune response genes and [is] linked to haplotypes containing the C4 null allele."

"*Role of Immunogenetics in the Diagnosis of Postvaccinal CNS Pathology*," Massimo Montinari, et al., Department of Pediatric Surgery, University of Bari, Italy, presented May 9, 1996 (text available http://www.healthy.net/library/articles/coulter/biochem.htm): after thirty children were found to have signs of central nervous system and genetic damage following vaccination, the authors remark, "*A study of the disease associated with genes of the HLA system has shown that this genetic complex can be responsible for a particular genetic susceptibility, predisposing to various diseases characterized predominantly by immune-system pathogenesis*… results indicate that autoimmune pathology is more frequent in countries where vaccination is more widespread….." [A fuller description of this study will be found in "**The attenuated virus--infectious or not?**" below.]

T. Binstock, Researcher in Developmental & Behavioral Neuroanatomy, Denver, Colorado, "*Familial does not mandate genetic[:]…Four categories of familial illness or disorder*." "Only one of [the first] three categories is purely genetic [--category A:]" A) familial occurrences indicating an actual gene-mutation that is hereditary; B) familial occurrences reflecting a genetically encoded susceptibility factor; C) familial illness via environmental factors. D) familial clustering of increased small intestinal permeability in families with Crohn's Disease. In category B, "the mutated gene is **not** the primary cause but is merely an inborn way that a person is more statistically likely, over time, to experience the primary cause. Genetically encoded immunodeficiencies are an example of 'susceptibility factor.' [With autism,] the most common genetic susceptibility factor is having a null allele of complement 4b" (referenced communication to Autism listserv originating at St. Johns, University, New York [autism@maelstrom.stjohns.edu], September 16, 1997, 08:39:13 -0700)**.**

G. Trottier et al., "*Etiology of infantile autism: a review of recent advances in genetic and neurobiological research*" (Journal of Psychiatry and Neuroscience, vol. 24, no. 2, March 1999, pp. 103-15): "The etiology of autism is complex, and in most cases the underlying pathologic mechanisms are unknown… Recent research has investigated…immunological factors. On the basis of family and twin studies, there appears to be a genetic basis for a wide 'autistic syndrome.' …Autoimmunity also may play a role; antibodies against myelin basic protein are often found in children with autism, who also have increased eosinophil and basophil response to IgE-mediated reactions. In summary, the prevailing view is that autism is caused by a pathophysiologic process arising from the interaction of an early environmental insult and a genetic predisposition."

A. M. Comi, et al., "*Familial clustering of autoimmune disorders and evaluation of medical risk factors in autism*" (Journal of Child Neurology, vol. 14, no. 6, June 1999, pp. 388-94): "Autism is an age-dependent neurologic disorder that is often associated with autoimmune disorders in the patients' relatives… The most common autoimmune disorders…[are] type 1 diabetes, adult rheumatoid arthritis, hypothyroidism, and systemic lupus erythematosus… An increased number of autoimmune disorders [in patients' families] suggests that in some families with autism, immune dysfunction could interact with various environmental factors to play a role in autism pathogenesis."

Barak, Y., et al., "*Autistic subjects with comorbid epilepsy: a possible association with viral infections*" (Child Psychiatry and Human Development, vol. 29, no. 3, Spring 1998, pp. 245-51): "This study evaluates the comorbidity of epilepsy as a variable supporting a viral hypothesis in Autism. Data covering a thirty-year period (1960-1989)…were collected… The annual birth pattern of subjects with comorbid epilepsy fit the seasonality of viral meningitis. These findings support the role of viral C.N.S. infections in the causality of this disorder."

"*Serological Association of Measles Virus and Human Herpesvirus-6 With Brain Autoantibodies in Autism*" (Clinical Immunology and Immunopathology vol. 89, number 1, October 1998, pp. 105-8): this study is the first to report an association between virus serology and brain autoantibody in autism; it supports the hypothesis that a virus-induced autoimmune response may play a causal role in autism.

The pattern of normality followed by regression, loss of development, or halted development and appearance of odd behavioral features was not fabricated by parents, as London implies; it is well represented in the autism literature. Probing the issue of genetics in autism, an important possibility goes unmentioned in London's "*ABCs*:" the genetic factor in regressive autisms and PDDs could well be a particular configuration of immune system components—in essence, vulnerabilities in the immune system such that affected children cannot adequately deal with the challenges posed by the current, frequently trivalent live-virus vaccines. Like Fudenberg, Van Gent, et al. observe that "*the immune response is regulated by genetic material located mainly on the sixth chromosome*." Failure of components of this system can lead to disruption of normal cell-mediated cellular immune responses. Virus infections may induce autoimmunity, and autoimmunity may result in increased susceptibility to infections and subsequent damage

to the CNS. Besides viral infections as a cause, it is possible that "a genetic predisposition to a relative deficiency of specific immune cells may be involved" (pp. 344-5).

The attenuated virus: infectious or not?

Oddly, rubella and other viral infections have been considered causal for autism if they occur before birth, or after birth if they occur 'naturally,' as in measles encephalitis, but not if they occur after vaccination. On page 16 of the NAARRATIVE issue in question, London claims that the viruses in vaccines are either dead or attenuated, and thus "can no longer cause disease." That live, attenuated viruses cannot cause disease is an astounding assertion given that, first, many polio cases have been caused by the oral polio vaccine—sometimes in persons caring for the vaccinee.[3] Jonas Salk, its inventor, once commented that the polio vaccine is the leading cause of polio today. An issue of Pediatrics(vol. 84, no. 5, November 1989, pp. 851-5) studies "*Anemia of a mild viral infection:* [using] *the measles vaccine as a model*." In this study, live attenuated measles virus was given to 93 infants in order to induce mild viral infections, in order to study hematologic changes (hemoglobin was shown to drop significantly in these infants). According toMolecular Virology, 1994, Chapter 3, **Vaccines and Immunotherapy**, "all live viruses are subject to a range of concerns… (p. 76)." Page 78 charts advantages and disadvantages of the different types of vaccines: live attenuated vaccine viruses can not only revert to virulence, but "may cause [a] mild form of disease;" or, due to the presence of viral genomes, "may be[come] pathogenic or oncogenic (cancer causing) in some [people's] systems."[4] Aside from these forms of infection, persistent (also called chronic, or 'slow') or latent infections may be engendered by viruses (MV, pp. 39-41). Viral "replication strategies may enable them to remain intracellular, either as latent or as persistent infections. Their replication, or their mere presence in the cell, may interfere with differentiated cell functions…*Viruses have the potential to cause a number of pathological changes, which may have profound and long-lasting clinical effects even after the virus has been eliminated from the body*" (Van Gent, pages 340-341). In Van Gent, p. 341, the authors warn that the immune system is particularly susceptible to tolerance induction when the cells of the immune system have not yet reached maturity. The condition of viral 'tolerance' is explained in Molecular Virology (David R. Harper, Bios Scientific Publishers, 1994), pages 39-40, and in The Immunology of the Immune System (Oxford University Press, 1977), pages 77-78.

Chronic viral infections can reactivate, moreover—sometimes years after initial infection. In Subacute Sclerosing Panencephalitis (SSPE), "virus replicates at low levels without producing infectious virus with altered production of viral proteins and an atypical immune response" (MV, p.39). But there is still another means by which a live virus vaccine can cause disease: since vaccine viruses are grown in animal or human cells, contaminating or endogenous ('produced from within') animal viruses can inhabit the vaccine and infect a vaccinated individual. Early batches of killed polio vaccine, for instance, were found to be contaminated with Simian Virus 40 (Molecular Virology, pp. 39-43; 75-6; 78-9), which has been linked with cancer years after infection. Other animal viruses—and viral proteins—have been known to contaminate vaccines, due to the use of particular animal cells or tissues in vaccine-making. Dr. John W. Martin has done extensive work in detecting such "stealth viruses" in the systems of vaccinees (texts of numerous papers and conference presentations are available at http://www.ccid.org). Contamination can also occur in a more limited setting, as with bacterial contamination during the manufacturing process. Accidents of other kinds can occur with vaccine manufacture, as with incomplete or incorrect inactivation or attenuation.

Viral infection--whether by vaccine viruses or those contacted in the environment--can cause or precipitate chronic disease in astonishing variety. Determined and skillful survivors, viruses can change form when "eradicated" by vaccines and reappear as new diseases, which seemingly unrelated to the original infection (at least, until viral proteins or other indications are discovered in researching these diseases). Live viruses from vaccines can bring about the death of vaccinees--either immediately after vaccination or years afterward--and can interact dangerously with antibodies already present in "second-generation" vaccinees (e.g., vaccinated children of fully-vaccinated parents). Due to their nature and methods of survival, in addition, vaccine viruses can change the very nature of their human hosts by altering genetic coding.

SELECTED STUDIES AND ARTICLES CONCERNING VACCINE VIRUSES

Acute infection by vaccine viruses

"*Mumps meningitis following measles, mumps, and rubella immunisation* [letter]" (The Lancet, vol. 2, July 8, 1989, p. 98; comments in vol. 2, August 12, 1989, pp. 394-5; vol. 2, September 16, 1989, p. 677): in the primary letter, mumps meningitis was reported in a three-year-old girl twenty-one days after measles, mumps, and rubella (MMR) immunization. The child exhibited lethargy, vomiting, headache, dry cough, fever, irritability, and meningeal irritation. There was no known exposure to the measles, mumps or rubella natural infections. No bacterial or other infections were found. In the August 12 issue of Lancet, a West German physician reported, also in a letter, a two-year-old boy with mumps meningitis twenty-one days following a different manufacturer's MMR vaccine. There was no exposure to natural mumps virus. The author of August 12 concludes, "*The incubation time for mumps is about 21 days. In some patients, time-lag between immunisation and manifestation of meningitis was very close to 3 weeks, without known previous mumps contacts. These facts strongly suggest that some patients may have had vaccine mumps meningitis, and not wild mumps infection.*" In the September 16 issue of Lancet, two British physicians report two 16-month-old boys with mumps meningitis admitted to the hospital 18 and 19 days following MMR immunization. Mumps virus was isolated from cerebrospinal fluid of both boys. One boy did not exhibit a rise in mumps antibodies in spite of vaccination and post-vaccinal meningitis. [Other vaccinal mumps meningitis citations: "Mumps meningitis, possibly vaccine related," Canada Disease Weekly Report, vol. 14-40, 1988, pp. 209-11; "A case of mumps meningitis: a post-immunization complication?" Canada Disease Weekly Report, vol. 13-35, 1987, pp. 155-6; "A case of mumps meningitis: a complication of vaccination?" Canadian Medical Association Journal, vol. 138, 1988, p. 135; "Vaccine-induced mumps-like disease," Development of Biological Standards, vol. 43, 1978, pp. 269-72; "Aseptic meningitis after vaccination against measles and mumps," Paediatric Infectious Diseases, vol. 8, 1989, pp. 302-8.]

"*Epidemics of aseptic meningitis due to enteroviruses following national immunization days in Bahrain*" (Annals of Tropical Paediatrics, vol. 18, no. 2, June 1998, pp. 101-9): Two successive epidemics of aseptic meningitis due to enteroviruses were observed after national immunization days against polio, comprising 286 and 169 cases, respectively, from July 1995-September 1996. Another report, "*Update of enterovirus infection in infants and children*" states, in a section titled "Viral meningitis," that natural polioviruses were an important cause of viral meningitis before vaccination (cases were called "nonparalytic poliomyelitis"). Now, "rare" cases of viral meningitis are attributed to the attenuated polioviruses in vaccines, in both vaccine recipients and their contacts (Pediatric Bulletin, http://home.coqui.net/myrna/virus.htm).

"*Disease caused by Haemophilus influenzae type b in the immediate period after homologous immunization: immunologic investigation*" (Pediatrics, vol. 85, number 4 part 2, April 1990, pp. 698-704): "One concern with the use of [current HIB vaccines] was the suggestion that the incidence of invasive disease caused by H influenzae type b in the immediate period after immunization might be increased; this idea was supported by evidence from several sources." In one case-controlled study, 4 children were hospitalized for invasive disease within 1 week of immunization; the rate of invasive disease was 6.4 times greater than the background rate in unvaccinated children.

"*Neurologic complications associated with oral poliovirus vaccine and genomic variability of the vaccine strains after multiplication in humans*," Acta Virologica, vol. 42, number 3, June 1998, pp. 187-94: The oral poliovirus vaccine (OPV) sometimes occasions paralytic poliomyelitis in vaccine recipients *and* their susceptible contacts. Molecular biology studies of polioviruses from these patients demonstrate genomic modifications known or suspected to increase neurovirulence. The same genomic modifications have been identified in strains isolated from non-symptomatic vaccinees. Other neurologic complications such as meningitis, encephalitis, convulsions, transverse myelitis and Guillain-Barre Syndrome have also been associated with this vaccine.

"*Paralytic poliomyelitis in a rural area of north India*" (National Medical Journal of India, vol. 10, no. 1, January-February 1997, pages 8-10): In a house-to-house survey conducted between 1990 and 1991, several cases of paralytic poliomyelitis were identified, 60 percent of which had had intramuscular injections preceeding paralysis, in treatment of minor fevers.

"*Poliomyelitis trends in Pondicherry, south India, 1989-91*" (Journal of Epidemiology and Community Health [London], vol. 51, no. 4, August 1997, pages 443-48): About 54 percent of children lamed as a result of poliomyelitis had received three doses of oral polio vaccine before the onset of paralysis.

"*Paralytic Poliomyelitis -- United States, 1980-1984*" (Morbidity Mortality Weekly Report, vo. 46, no. 4, January 31, 1997, pp. 79-83): The Advisory Committee on Immunization Practices (ACIP) observes that vaccine-associated paralytic poliomyelitis (VAPP) continues to occur; the risk of VAPP has not decreased. Of 125 cases associated with the vaccine, 46 cases occurred among *contacts* of vaccine recipients.

"*Comparative evaluation of immunization with live attenuated and inactivated polio vaccines*" (Annals of the New York Academy of Science, vol. 754, May 31, 1995, pp. 97-107): With both oral attenuated polio vaccine (OPV) and the enhanced potency inactivated polio vaccine (EP-IPV), revertant or non-revertant viral shedding occurred in body wastes for up to 60 days following vaccination. The authors concede, after saying the combined polio vaccines should be effective in establishing immunity in recipients, and should lower the rate of vaccine-associated paralytic poliomyelitis (VAPP) in recipients, that VAPP is expected to continue effecting susceptible contacts.

"*Poliomyelitis associated with type-2 poliovirus vaccine strain. Possible transmission from an immunised child to a non-immunised child*" (The Lancet, vol. 1, March 30, 1968, pp. 661-3): a sixteen-month-old boy hospitalized for high fever and paralysis had never received any poliovirus vaccine. From playing and sharing a bed with a cousin, he apparently had contracted paralytic poliomyelitis from the cousin, who had received type-2 oral poliovirus vaccine thirty-three days before. Virological and serological investigation revealed a vaccine-like strain of type-2 poliovirus. The patient's history revealed no particular susceptibility to infections.

L. J. Morse, et al., Journal of the American Medical Association, vol. 197, 1966, p. 1034: A case of paralytic poliomyelitis in an unvaccinated mother was reported, apparently acquired after exposure to her infant, who had received trivalent live, oral polio vaccine twenty-two days earlier.

"*Transmission of vaccine strain varicella-zoster virus from a healthy adult with vaccine-associated rash to susceptible household contacts*" (Journal of Infectious Disease, vol. 176, no. 4, October 1997, pp. 1072-5): Twelve days after receiving an investigational Oka strain live attenuated varicella vaccine, a 38-year-old healthy woman developed a rash consisting of 30 scattered lesions. Sixteen days later, her two children also developed a rash. Varicella-zoster DNA obtained from the skin lesions was determined to be the vaccine type. "This case documents transmission of varicella vaccine type virus from a healthy vaccinee to susceptible household contacts…ongoing studies will define the frequency of this transmission."

"*Live Virus Vaccines, High-Dose Steroids Don't Mix*" (Pediatric News, cited November 28, 1998, via@access1.net, 10:49 a.m.): Dr. Larry K. Pickering, a member of the American Academy of Pediatrics' "Red Book Committee," was quoted following a meeting at the University of South Dakota, saying children receiving more than 2 mg/kg per day of systemic glucocorticoids should not be given live virus vaccines, due to the risk of disseminated infection from the vaccines. Killed virus vaccines do not present the same risk. [Note: steroids such as prednisone partially suppress the immune system.]

"*Acute encephalopathy followed by permanent brain injury or death associated with further attenuated measles vaccines: a review of claims submitted to the National Vaccine Injury Compensation Program*," Pediatrics, vol. 101, no. 3, Part 1, March 1998; pages 383-387: This study details cases wherein 48 children, ages 10 to 49 months, who had been so affected. Eight children died, and the remainder had mental regression and retardation, chronic seizures, motor and sensory deficits, and movement disorders. "CONCLUSIONS: *This clustering suggests that a causal relationship between measles vaccine and encephalopathy may exist as a rare complication of measles immunization*." [Note regarding rarity: A huge number of vaccine reactions are never reported, and most of the thousands of vaccine injuries which are reported do not meet the current, very narrow VAERS/FDA criteria (a very few specific symptoms must occur within a very short timespan, in order for symtoms to be considered vaccine-related), and thus are not reported as vaccine-injury cases by government tabulators. Serious vaccine complications thus are said to be "rare" in quoted statistics. If independent research proves that the measles vaccine and PDD/autism are causally related, this kind of vaccine damage will inflate by thousands the cases of vaccine damage now on record. This tally, then, may be inflated further by the number of ADD/ADHD-diagnosed children with inflammatory bowel disorders, per the Georgetown University study cited in "Wakefield," below.]

"*Measles-Mumps-Rubella (MMR) Vaccine as a Potential Cause of Encephalitis (Brain Inflammation) in Children*," Harold E. Buttram, MD, Townsend Letters, December 1997 (available at http://www.mercola.com/issue5.htm). Childhood autism is the result of encephalitis affecting primarily the limbic system of the brain, located below the cerebral cortex. A relatively few number of cases are due to genetic causes, but officially the great majority are of unknown causes. It is now generally thought that the process of encephalitis, whether from wild viruses of live-virus vaccines, is associated with an interference with the myelination process brought about by the development of antibodies against myelin basic protein, a constituent of the myelin sheaths.

Chronic infection by vaccine viruses

"*Effect of subclinical infection on maintaining immunity against measles in vaccinated children in West Africa*" (The Lancet, vol. 353, January 9, 1999, pp. 98-102): Exposure to natural measles in 87 vaccinated children yielded 39 subclinical cases of measles. Antibody concentrations increased 45-fold and remained raised for at least six months [note: in such a recent study, opportunity has not yet presented itself to ascertain antibody levels one or two years after vaccination, etc.].

E. R. Bolte, "*Autism and clostridium tetani*" (Medical Hypotheses vol. 51, 1998, pp. 133-144): "This paper outlines the possibility of a subacute, chronic tetanus infection of the intestinal tract as the underlying cause for symptoms of autism observed in some individuals. A significant percentage of individuals with autism have a history of extensive antibiotic use. Oral antibiotics significantly disrupt protective intestinal microbiota, creating a favorable environment for colonization by opportunistic pathogens. Clostridium tetani is an ubiquitous anaerobic bacillus that produces a potent neurotoxin. Intestinal colonization by C. tetani, and subsequent neurotoxin release, have been demonstrated in laboratory animals… The vagus nerve is capable of transporting tetanus neurotoxin (TeNT) and provides a route of ascent form the intestinal tract to the CNS…once in the brain, TeNT disrupts the release of neurotransmitters by the proteolytic cleavage of ynaptobrevin… Lab animals injected in the brain with TeNT have exhibited many of these behaviors. Some children with autism have also shown a significant reduction in stereotyped behaviors when treated with antimicrobials effective against intestinal clostridia… A review of atypical tetanus cases, and strategies to test the validity of this paper's hypothesis, are included."

T. Zecca, D. Grafino, et al., University of Medicine and Dentistry, New Jersey and Children's Hospital of New Jersey, Newark, "*Elevated rubeola* [measles] *titers in autistic children linked to MMR vaccine*" (abstract submitted to the National Institutes of Health, 1997-8; text available at http://webpages.netlink.co.nz/~ias/mmraut1.htm): Rubeola (measles) titers were compared in autistic and normal children. Children diagnosed with autism revealed "a three fold increase" in their rubeola titers over expected normal range. "A Wilcoxon Kruskal Wallas test comparing 13 rubeola titers from normal children reveals a statistically significant P-value of 0.0050." The authors note that neurological sequelae following MMR are widely reported: "MMR therefore may play a role in the pathogenesis of Autism. *The elevated titers of anti-measles antibodies in Autistic children may signify a chronic activation of the immune system against this neurotropic virus*."

H. Trier and T. Ronne, "*Duration of immunity and occurrence of secondary vaccine failure following vaccination against measles, mumps and rubella*" (Ugeskr Laeger, vo. 154, no. 29, July 13, 1992, pp. 2008-13): While discussing loss of immunity after vaccination, the authors observe, "Subclinical infection is not uncommon after all three vaccines."

"*Characterisation of poxviruses from sporadic human infections*" (South African Medical Journal, vol. 72, no. 12, December 19, 1987, pp. 846-8): An orthopoxvirus was isolated from…a man in Natal who died in coma… Analysis of the viral DNA showed that it was a vaccinia virus, more closely related to the virus of South African smallpox vaccine than to other [natural] vaccinia viruses. DNA analysis also showed that an orthopoxvirus isolated from a sporadic case of severe pustular rash in Nigeria was a vaccinia virus closely related to the smallpox vaccine virus used there… [It was] suggested that some natural transmission of the virus had occurred…originat[ing] from the use of smallpox vaccine. *No similar cases have been detected since smallpox vaccination was discontinued*."

"*Vaccinia virus persistence in a child against the background of immune deficiency*" (J. Hyg. Epidemiol. Microbiol. Immunol., vol. 30, no. 2, 1986, pp. 177-83): " A young girl, vaccinated against smallpox 6 years before[,] suffered from a persistent vaccinia virus infection and a congenital skin disesase, i.e. epidermolysis bullosa. The virus was isolated from skin lesions at the vaccination site and remote sites and repeatedly from the blood… Examination of the child did not show any quantitative immune deficiency… The possible genesis of the virus persistence and the role of the virus in the clinical course of the disease are discussed." (A selected Medline [National Library of Medicine] "MESH" subject tracing for this report is "Smallpox Vaccine--adverse effects.")

O. Laitinen and A. Vaheri, University Central Hospital and Department of Virology, University of Helsinki, Finland, "*Very high measles and rubella virus antibody titres associated with hepatitis, systemic lupus erythematosus, and infectious mononucleosis*" (The Lancet, vol. 1, February 9, 1974, pp. 194-7): When patients with typical acute measles or rubella infections and their complications were excluded, certain groups of patients with very high antibody levels to measles and/or rubella viruses remained and were studied. These "patients showed very high measles or rubella antibodies although there had been no recent typical rubella or measles infection… Our data suggest that atypical viral infection plays an active role in the pathogenesis of at least some of the abovementioned conditions… these viruses may cause chronic infections with raised antibody levels…moreover, sera from some patients with other diseases…show very high levels of antibody against these two viruses… In multiple sclerosis raised levels of measles antibodies have been previously reported." Antibodies to many other viruses and to Mycoplasma and Toxoplasma were normal. The authors acknowledged the possibility that an abnormal immunological defense mechanism had lead to an abnormal virus infection—"e.g., carrier infection, reinfection, or incomplete measles or rubella virus infection—in some susceptible individuals…[;] however[,]…no *4-fold or greater* changes occurred in virus antibody levels in any patient with systemic lupus erythematosus, chronic active hepatitis, or infectious mononucleosis [as happens with autism, in which titers are often four to seven times that of normal]…" Evidence did not exclude chronic virus infection as an important factor in these diseases.

Reactivation of vaccine viruses "after the fact"

"*Measles, Mumps, Rubella Vaccine Induced Subacute Sclerosing Panencephalitis*," Journal of the Indian Medical Association, November 1997, vol. 95 no. 11, page 594: a particular case of SSPE is described in a *thirteen-year-old* girl who had been immunized against all childhood diseases; receiving the MMR vaccine at the age of *nine months*. The girl's intellectual functioning until development of illness had been very good. After illness developed, the child verbalized little and was socially inappropriate; her memory and thinking abilities were impaired. She grew progressively worse, and added myoclonic jerks of the upper limbs, with depressed deep tendon reflexes. The authors concluded that Subacute, Sclerosing Panencephalitis was engendered as a delayed adverse effect of measles vaccine. The authors note other cases of SSPE induced by the attenuated measles vaccine.

"*Measles Encephalomyelitis in a Patient With a History of Vaccination*," Acta Paediatrica Japonica, vol. 37, number 3, June 1995, pp. 374-376: A twelve-year-old girl vaccinated with a live attenuated measles vaccine developed an encephalomyelitis ten years post-vaccine. "The patient's definite history of measles vaccination, high titers of HI and IgG antibodies…indicated that this patient has an encephalomyelitis due to Secondary Vaccine Failure of measles. *It is suggested that measles virus can be a pathogen of encephalitis without symptoms indicative of ordinary measles in individuals who received live attenuated measles vaccines*."

"*Polymerase chain reaction detection of the hemagglutinin gene from an attenuated measles vaccine strain in the peripheral mononuclear cells of children with autoimmune hepatitis*," Archives of Virology volume 141, 1996, pages 877-884: "The measles virus is known to be persistent in patients with subacute sclerosing panencephalitis (SSPE) and measles inclusion body encephalitis (MIBE). Since the introduction of measles vaccines, vaccine-associated SSPE has increased in the USA. Therefore, we should pay attention to SSPE after inoculation with measles vaccine, despite the decrease in the incidence of [wild] measles."

Infection by vaccine contaminants

"*Children exposed to CJD infecton risk from vaccines*" (Antony Barnett, Public Affairs Editor, The Guardian/The Observer [Guardian Media Group], Sunday May 30, 1999; text

available http://www.guardianunlimited.co.uk/Archiv…le/0,4273,3870082,00.html?cantsetcookie=0): from 1989 to 1993 thousands of human vaccines for such diseases as tetanus and pertussis were knowingly approved for use by the Department of Health (UK), which may have been based on matter derived from BSE ("mad cow disease")-infected cattle. Before "mad cow disease" became a concern in 1988, vaccines were made with bovine serum without awareness of the possibility of contamination. Sir Richard Southwood, Professor of Zoology at Oxford University, stated that injection posed a far greater risk of CJD than eating foods made from infected cattle. When BSE is contracted by humans, it becomes Creutzfeld-Jacob Disorder, or CJD.

"*[Infectious diseases of animals and their prevention]*" (Bratisl. Lek. Listy., vol. 99, nos. 8-9, August-September 1998, pp. 465-73): "Infectious diseases of animals are the subject of continuous concern… Undoubtedly, microbes and parasites take part also in the development of malignant transformation of cells. The question of possible transfer of animal oncogenic [cancer-causing] microorganisms (retroviruses in particular) to humans remains open. The study points to the changes in the incidence of orthopoxviruses which occurred after 'eradication' [quote marks added] of human variola [smallpox] and the increasing importance of bartonelloses… We enter a period in which the resistance of animal organism begins to affect the transfer of genes encoding non-specific and specific protective [immunological] mechanisms of organisms."

"*The African polio vaccine-acquired immune deficiency syndrome connection*" (Medical Hypotheses, vol. 48, no. 5, May 1997, pp. 367-74): "Seroepidemiological, clinical and molecular findings suggest that the acquired immune deficiency syndrome virus **H**uman **I**mmunodeficiency **V**irus-1* was introduced into the human species at the the (late 1950s) and in the geographic area (Zaire) in which millions of Africans were vaccinated with attenuated poliomyelitis virus strains that were produced in kidney tissue obtained from monkeys. …it is reasonable to suspect that a then non-detectable monkey virus with human-1-like properties was unknowingly cocultured with the attenuated poliovirus and subsequently administered to the vaccinees. The possibility of such a polio vaccine-acquired immune deficiency syndrome connection is a reminder of the unpredictable danger of artifically crossing natural species-barriers in biomedical laboratories" [*bold text capitals added].

"*The origin of HIV-1, the AIDS virus*" (Medical Hypotheses, vol. 41, no. 4, October 1993, pp. 289-99): "a substantial case is presented that HIV-1 is a natural recombinant of Bovine Leukemia Virus (BLV) and Visna Virus. This natural recombinant may have been inadvertently transferred to humans through the Intensified Smallpox Eradication Program conducted in sub-Saharan Africa in the late 1960s and most of the 1970s."

"*Simian cytomegalovirus-related stealth virus isolated from the cerebrospinal fluid of a patient with bipolar psychosis and acute encephalopathy*" (Pathobiology , vo. 64, no. 2, 1996, pp. 64-6): a cytopathic 'stealth' virus was cultured from the cerebrospinal fluid of this patient, who developed a severe encephalopathy leading to a vegetative state. DNA sequencing of a polymerase chain reaction-amplified product from infected cultures revealed kinship to the African green monkey simian cytomegalovirus.

Immunosuppression and opportunistic infection

Abstract: also relevant to the London article, occasioned by concern over the MMR vaccine, the measles virus is noted for its ability to suppress the immune system, particularly cellular immunity. A person sustaining a chronic measles infection may therefore be increasingly subject to numerous other infections, whether viral, bacterial, or fungal [such abnormalities are well documented in autism].[5] Measles and rubella virus themselves, in addition, are associated with distinct central nervous system pathologies.[6]

"*Epidemiology of encephalitis in children. A prospective multicentre study*," European Journal of Pediatrics, vol. 156, number 7, July 1997, pp. 541-5: Investigators found 175 cases with acute encephalitis in children aged 1 month to 15 years during a two-year surveillance period in 1993-1994. Varicella zoster, respiratory and enteroviruses, Epstein-Barr virus, herpes simplex and rota viruses, and the new infections chlamydia pneumoniae and HHV-6 were found. While mumps, measles, and rubella virus associated encephalitis had been almost eliminated due to vaccination programs, these other viruses had increased in frequency and occurred in younger age groups. "**Conclusions**: The spectrum of encephalitis in children has changed due to vaccination programs. The incidence, however, appears to be about the same due to increasing frequency of other associated old and new microbes"—i.e.,

the number of cases of MMR-encephalitis eliminated have been replaced by an equal number of encephalitis cases from other microbes, previously not seen.

[Note: the following report does not address the cause of the systemic viral infection detailed; Epstein Barr is, however, capable of arising opportunistically when immune system responses are inhibited, as occurs in measles immunosuppression. Temporary or permanent neurological damage, or chronic disease, can result from such unhindered viral activity.] Ito, H., et al., "*Antineuronal antibodies in acute cerebellar ataxia following Epstein-Barr virus infection*," (Neurology, vol. 44, no. 8, August 1994, pp. 1506-7): A 29-year-old man developed acute cerebellar ataxia following Epstein-Barr infection. The ataxia gradually improved. The authors concluded that their findings in this case suggested a role for autoimmune mechanisms in the pathogenesis of acute cerebellar ataxia.

Pathogenesis: proliferation of disease

Persons—especially children—with Crohns disease, asthma, diabetes, ear infections and disorders; immunosuppression and secondary viral infections (EBV, CMV) have been found to harbor organisms, often those associated with vaccine-preventable diseases. Addressing bacterial infections, G. J. Domingue and H. B. Woody of Tulane University School of Medicine state, "A considerable body of experimental and clinical evidence supports the concept that difficult-to-culture and dormant bacteria are involved in latency of infection and that these persistent bacteria may be pathogenic… A series of experimental studies involving host-bacterium interactions illustrates the probability that most bacteria exposed to a deleterious host environment can assume a form quite different from that of a free-living bacterium… These organisms can survive and persist in a latent state within the host, and they can cause pathologic responses compatible with disease. A series of cases illustrating idiopathic conditions in which cryptic bacteria have been implicated…include nephritis, rheumatic fever, aphthous stomatitis, idiopathic hematuria, Crohn's disease, and mycobacterial infections…[;] nonculturable bacilli have been identified in patients with Whipple's disease and bacillary angiomatosis" ("*Bacterial persistence and expression of disease*," Clinical Microbiology Review, vol. 10, no. 2, April 1997, pp. 320-44).

"*Polymerase chain reaction detection of the hemagglutinin gene from an attenuated measles vaccine strain in the peripheral mononuclear cells of children with autoimmune hepatitis*," Archives of Virology, volume 141, 1996, pages 877-884: the authors observe, "Apparently, the attenuated vaccine is also capable of persisting, like sporadic wild strains, in certain immune diseases."

H. C. Huber, "*The pathogenesis of postvaccinal complications*" (Fortschr. Med., vol. 99, no. 11, March 19, 1981, pp. 380-1): "Paraspecific reactions to vaccines are--induction of autoimmune mechanisms, --immunosuppression,--induction of inflammation (e.g. "reactogenicity"). These undesirable side effects of vaccination are important factors in pathogenesis of postvaccinal complications."

Myocarditis

Diabetes

"*Hemophilus vaccine and increased IDDM, causal relationship likely*" (British Medical Journal, vol. 318, May 7, 1999); this letter conveys a re-interpretation by J. Bart Classen of data reported in "*Association between type 1 diabetes and Haemophilus influenzae type b vaccination: birth cohort study*" (British Medical Journal, vol. 318, May 1, 1999, pp. 1169-1172), which Classen Immunotherapies initiated and funded. "…the potential risk of the vaccine [in fact] exceeds the potential benefit." Classen discusses this data in the context of compensation for vaccine-damaged children with diabetes.

Coulter, Harris, Ph.D., Center for Empirical Medicine, Washington, D.C., "*Childhood vaccinations and juvenile-onset (type-1) diabetes: testimony before the Congress of the United States, House of Representatives…April 16, 1997*" (Committee on Appropriations, subcommittee on Labor, Health and Human Services, Education, and Related Agencies; text available at http://909shot.com/hcdiabetes.htm): the incidence of diabetes in the U. S. had increased 20 times since 1947. Healthcare costs are significant, both from the primary disease and from its complications such as cardiovascular disease, stroke, gangrene of the extremities, kidney failure, and blindness. A shortened lifespan is to be expected. Diabetes appears to be influenced by a genetic susceptibility, but environmental factors tend to lead to onset. Both pertussis toxin and rubella virus, ingredients of two mandated childhood vaccines, are capable of acting on the insulin-producing portions of the pancreas. "There is copious evidence[, also,] of a causal relationship between clinical mumps and subsequent development of diabetes [through pancreatitis]…many reports in the literature of Type-1 diabetes [report the condition] emerging after mumps vaccination." "There is no reason to make a distinction between…the disease process and…a vaccination… In both cases immune complexes are formed and persist in the host organism for lengthy periods. Immune complexes from a vaccination can attack the pancreas just as easily as if they were from contenital rubella syndrome. The actual mechanism of such an attack[, however,] is probably multifactorial[, …the most probable one being] the generation of an autoimmune state….." References are given to medical case reports describing the emergence of type-1 diabetes following vaccination.

Adverse Events Associated with Childhood Vaccines: Evidence Bearing on Causality (Washington, D.C.: National Academy of Sciences, Institute of Medicine, 1994): About the issue of vaccination and type-1 diabetes, the IOM Committee stated that "biologic plausibility data implicating the mumps virus in the pathogenesis of Type-1 diabetes include: 1) the association between viral infections, including mumps, and Type-1 diabetes in humans; 2) the detection of circulating autoantibodies against pancreatic antigens, particularly islet cells, during convalescence from mumps infection as well as early in the course of Type-1 diabetes; and 3) in vitro studies demonstrating that the wild-type mumps virus can infect human pancreatic beta cells" (p. 159).

Asthma

"*Measles virus infection synergizes with IL-4 in IgE class switching*" (Journal of Immunology, vol. 162, no. 3, February 1, 1999, pp. 1597-1602): "Increasing evidence suggests that viral infections are associated with the induction and exacerbation of asthma… These data provide the first indication of a potential mechanism for M[easles] V[irus] induced IgE up-regulation and suggest a model for a viral-induced exacerbation of IgE-mediated disorders such as asthma.

"*Is infant immunization a risk factor for childhood asthma or allergy?*" (Epidemiology, vol. 8, no. 6, November 1997): "Results of the Christchurch Health and Development Study, conducted by a team of New Zealand researchers, found a greater rate of asthma and allergy episodes among immunized children… The comparison produced similar results at ages five and 16, and the discrepancy does not appear to result from use of health services, ethnicity, socioeconomic status, or parental atopy or smoking."

"*Risk factors for invasive Haemophilus influenzae disease among children 2-16 years of age in the vaccine era, Switzerland, 1991-1993*" (International Journal of Epidemiology, vol. 25, no. 6, December 1996, pp. 1280-5): "Continued surveillance, and detailed investigation of direct and indirect effects of conjugated vaccines and risk factors…are important." 143 cases with invasive disease were selected, and vaccination status ascertained. "Cases more often than controls reported suffering from asthma and allergies… The observed association between asthma and epiglottitis is novel and deserves further investigation."

"*Very high measles and rubella virus antibody titres associated with hepatitis, systemic lupus erythematosus, and infectious mononucleosis*" (The Lancet, vol. 1, February 9, 1974, pp. 194-7). The authors note the existence of high viral titers in several diseases, including asthma: "[in] the other patients in whom only very high antibody levels to rubella…could be measured…bronchial asthma [was the only disease which was not rare, and for which] a possible viral role in their pathogenesis cannot be excluded."

Disorders of the ear

Blood disorders

"*Thrombocytopenic purpura as adverse reaction to recombinant hepatitis B vaccine*" (Archives of Disease in Childhood, vol. 78, no. 3, March 1998, pp. 273-4): Three cases of [auto]immune thrombocytopenic purpura after the first dose of recombinant hepatitis B vaccine occurred in infants under six months of age. There were no other possible causes; defect in platelet production was excluded in two children. Antiplatelet antibodies were present. The babies were treated with corticosteroids.

Hepatitis

"*Polymerase chain reaction detection of the hemagglutinin gene from an attenuated measles vaccine strain in the peripheral mononuclear cells of children with autoimmune hepatitis*," Archives of Virology volume 141, 1996, pages 877-884: Four pediatric and two adults patients with autoimmune hepatitis were tested and followed in this study. Twelve healthy children served as controls, who had either been infected with measles or vaccinated with an attenuated measles vaccine in the past. All controls were negative for measles virus except a recent (two week) vaccinee. Of the hepatitis patients, all were positive for measles virus—the children with *vaccine-strain* measles virus, and the adults with different strains. Conclusion: "our results demonstrated that children with autoimmune hepatitis can have persistence of the vaccine strain in vivo for many years after vaccination [abstract, page 877]." The authors state that the persistence of the measles virus might play some role in the pathology of autoimmune hepatitis, but further studies are needed to prove this hypothesis (page 883).

Also in "*Polymerase*," the authors observe that high levels of serum antibodies to measles virus have been reported in patients with autoimmune hepatitis (p. 877). References add systemic lupus erythematosus and infectious mononucleosis to the tally of autoimmune diseases with connections to measles (pages 883-4). [Note: high antibody titers of measles and rubella are also associated with autism.] Some provocative quotes, page 882: "Apparently, the attenuated vaccine is also capable of persisting, like sporadic wild strains, in certain immune diseases. The measles virus is known to be persistent in patients with subacute sclerosing panencephalitis (SSPE) and measles inclusion body encephalitis (MIBE). Since the introduction of measles vaccines, vaccine-associated SSPE has increased in the USA. Therefore, we should pay attention to SSPE after inoculation with measles vaccine, despite the decrease in the incidence of [wild] measles."

[Note: the following study did not broach the subject of vaccine involvement in diseases; rather it serves to point out the relationship of viral presences to disease.] …Department of Virology, University of Helsinki, Finland, "*Very high measles and rubella virus antibody titres associated with hepatitis, systemic lupus erythematosus, and infectious mononucleosis*" (The Lancet, vol. 1, February 9, 1974, pp. 194-7): In patients without preceding rubella or measles infection, "raised levels of viral antibodies were a constant finding in two repeated analyses" of hepatitis patients. The authors felt that "it is conceivable that rubella and/or measles infections or reinfections may cause acute hepatitis and persist in some individuals…such aberrant virus infection might be responsible for some clinical manifestations….." Chronic virus infection could not be excluded as an important factor in these diseases.

Inflammatory and autoimmune bowel disease

"*Paramyxovirus infections in childhood and subsequent inflammatory bowel disease*" (Gastroenterology, vol. 116, no. 4, April 1999, pp. 796-803): "Measles virus has been implicated in the etiology of both inflammatory bowel diseases (IBDs), Crohn's disease and ulcerative colitis… Mumps infection before age 2 years was a risk for ulcerative colitis… Measles and mumps infections in the same year of life were significantly associated with ulcerative colitis and Crohn's disease…but not with IDDM… Atypical paramyxovirus infections in childhood may be risk factors for later I[nflammatory] B[owel] D[isease.]" [Notes: measles-mumps-rubella vaccine is usually given around the age of 16 months. When vaccine viruses induce infection, the resulting illness is often atypical in character. A Reuter's Medical news release pertaining to this study, found at http://www.reutershealth.com/frame_about.html, cited mumps infection in the same year as monovalent measles vaccination appeared to increase the risk of later Crohn's disease.]

Lupus, multiple sclerosis and rheumatoid arthritis

Abstract: autoimmune diseases are becoming increasingly common. The majority seem to have viral associations.

"*Vaccine-induced autoimmunity*" (Journal of Autoimmunity, vol. 9, no. 6, December 1996, pp. 699-703): the authors summarize of case reports attributing autoimmune diseases and autoimmune phenomena to vaccines, and suggest possible mechanisms by which the two could be related. "The subject is complicated," they say, "by the fact that one vaccine may cause more than one autoimmune phenomenon, and a particular immune process may be caused by more than one vaccine. Furthermore, vaccines differ in their pathogenic influence on the immune system... The subject of the vaccine-autoimmunity relationship is still obscure; reports have been rare, [and] no laboratory experimentation on this topic has been undertaken....." (Oddly, the authors state that the benefits of vaccination outweigh the risks of disease, but given the authors' contentions that vaccines can cause one or more types of autoimmune disease, that reports are few and research non-existent, this statement is unsupported. Further, they conclude that "laborious clinical and laboratory studies should be initiated in order to evaluate the ...subject.")

C. M. Poser, Harvard Medical School, "*The pathogenesis of multiple sclerosis. Additional considerations*" (Journal of Neurological Science, vol. 115, April 1993, Supplement pp. S3-15): "Multiple sclerosis is acquired as a systemic "trait" by individuals who are genetically susceptible…It develops as the result of an antigenic challenge by a viral protein, either from a viral infection or a vaccination."

"*Multiple sclerosis and infectious childhood diseases*" (Neuroepidemiology, vol. 17, no. 3, 1998, pp. 154-60): multiple sclerosis patients studied had had measles, mumps, and varicella (chicken pox) infections at a later age than healthy controls. "These results are compatible with the hypothesis that the risk of developing multiple sclerosis may be associated with acquiring certain infectious childhood diseases at a later state in comparison to normal controls." [Early vaccination for these diseases, therefore, may predispose vaccinees to MS, as immunity from vaccinations frequently wanes in the years following early childhood vaccination (unlike immunity to natural infection). In the event of such a vaccine failure, natural infection may occur at a later age.]

"*Chronic arthritis after rubella vaccination*" (Clin. Infectious Disease, vol. 15, no. 2, August 1992, pp. 307-312. After reviewing a wide range of information sources, The Institute of Medicine, Washington, DC, found a causal relationship between rubella vaccination and chronic arthritis in adult women.

--for lupus, see <<cognitive disorders in systemic diseases,>> below--

Parasthesias/paralytic and muscular diseases

"*Drug Points: Transverse Myelitis After Measles, Mumps, and Rubella Vaccine*," BMJ [British Medical Journal], vol. 311 (7002), August 12, 1995, p. 422: a twenty-year-old man was vaccinated against rubella with the MMR vaccine. Five days later he developed fever, malaise, sore throat, and a transient, upper-body rash. Within the next two weeks, he developed an ascending paraesthesia. He was hospitalized on developing a rapidly progressive flaccid paraplegia. Serological tests showed a significant rise in rubella antibodies. Postvaccination transverse myelitis was diagnosed.

"*Poliovirus vaccine options*" (American Family Physician, vol. 59, no. 1, January 1, 1999, pp. 113-8, 125-6): "Of 142 confirmed cases of paralytic poliomyelitis reported in the United States from 1980-1996, 134 were classified as vaccine-associated paralytic poliomyelitis (VAPP). Persons with VAPP have a disabling illness….."

"*Demonstration of specific antineuronal nuclear antibodies in sera of patients with myasthenia gravis*" (Neurology, vol. 24, no. 7, July 1974, pp. 680-3).

Other disorders of the brain and nervous system

Vijendra K. Singh and others have found a significant association between autoimmune processes in autistic patients and viral presences--in particular, anti-myelin basic protein (anti-brain) antibodies, often in association with high antibody titers against specific microbes. In this regard, see also "*Demonstration of specific antineuronal nuclear antibodies,*" above, and the description of T. Zecca's report, "*Elevated rubeola* [measles] *titers in autistic children linked to MMR vaccine,*" above. Study of other cognitive, behavioral, and movement disorders has revealed immune system involvement.

<<seizure disorders>>

"*Autistic subjects with comorbid epilepsy: a possible association with viral infections*" (Child Psychiatry and Human Development, vo. 29, no. 3, Spring 1998, pp. 245-51): Data covering a 30-year period was examined in Israel. The annual birth pattern of 290 autistic subjects with comorbid epilepsy fit the seasonality of viral meningitis. "These findings support the role of viral C[entral] N[ervous] S[ystem] infections in the causality of this disorder."

"*Neurologic complications after vaccination against diphtheria, tetanus and whooping cough* (Cesk. Pediatr., vol. 47, no. 2, February 1992, pp. 122-4): Both in children free from neurological disease and in children with neurological disease the most frequent type of complications from DTP vaccination were "encephalopathies and febrile attacks as a consequence of metabolic and toxic changes following vaccination." Persisting neurological disorders were, in the majority, epileptic in character.

"*Vaccination against whooping-cough. Efficacy versus risks,*" The Lancet, vol. 1, January 29, 1977, pp. 234-7: "Adverse reactions and *neurotoxicity* following vaccination was strongly related to pertussis vaccine in 79 of 160 cases studied. A *shock* reaction and *cerebral disturbance* was seen, in most of these cases followed by convulsions, hyperkinesis, *and severe mental defect*. The authors conclude, "It seems likely that most adverse reactions are unreported and that many are overlooked…existing provisions, national and international, for epidemiological surveillance and evaluation are inadequate. The claim by official bodies that the risks of whooping-cough exceed those of vaccination is questionable, at least in the U.K."

O. Tonz and S. Bajc, "*Convulsions after whooping-cough vaccination*" (Schweiz. Med. Wochenschr., vol. 110, no. 51, December 20, 1980, pp. 1965-71): Convulsions or status epilepticus in 11 infants after pertussis vaccination are reported. In three of 11 cases, grand mal epilepsy persisted and two children developed infantile epileptic encephalopathy (Lennox Syndrome). "The following conclusions are drawn from these observations: 1) In view of the usually benign course of whooping cough today, current vaccination is hardly satisfactory. Improvement of the available vaccines is an urgent necessity… 2) Parents whould be better informed about the risks involved in pertussis vaccination. 3) Booster inoculations should be abandoned. 4) Health authorities should decide whether the current pertussis vaccination program should be abandoned. 5) Complications following vaccination should be registered….."

<<behavior and movement disorders >>

"*A controlled study of serum anti-locus ceruleus antibodies in REM sleep behavior disorder*" (Sleep, vol. 20, no. 5, May 1997, pp. 349-51): "The newly identified association of human nonnarcoleptic rapid eye movement (REM) sleep behavior disorder (RBD) with human leukocyte antigen (HLA) DQwl class II genes raises the possibility that RBD may arise from autoimmune mechanisms."

[The following reports are not vaccine-specific; rather they serve to underline one of the possible conditions resulting from altered permeability of, or damage to the intestine, as occurs in association with measles and other viruses. Note: strep-type bacteria are among those which can translocate from the gut; strep bacteria have been implicated in cases of Obsessive-Compulsive Disorder and Tourette Syndrome.] "*Bacterial translocation from the gastrointestinal tract*" (Trends in Microbiology, vol. 3, no. 4, April 1995, pp. 149-54): Viable indigenous bacteria from the gastrointestinal tract can migrate to other sites within the body, such as the mesenteric-lymph-node complex, liver, spleen, and bloodstream. Three mechanisms support bacterial translocation: intestinal bacterial

overgrowth, deficiencies in host immune defenses and increased permeability or damage to the intestinal mucosal barrier.

"*Case study: a new infection-triggered, autoimmune subtype of pediatric OCD and Tourette's syndrome*" (Journal of the American Academy of Child and Adolescent Psychiatry, vol. 34, no. 3, March 1995, pp. 307-11): the authors hypothesize that infections with group A beta-hemolytic streptococci, among other bacterial agents, may trigger autoimmune responses that cause or exacerbate some cases of childhood-onset obsessive-compulsive disorder (OCD) or tic disorders including Tourette's Syndrome. In this study, four boys aged 10 to 14 years presented with OCD or Tourette's Syndrome in the moderate to very severe range. Two had evidence of recent group A beta-hemolytic streptococci infections, and the others had histories of recent viral illnesses.

"*Speculations on antineuronal antibody-mediated neuropsychiatric disorders of childhood*" (Pediatrics, vol. 93, no. 2, February 1994, pp. 323-6): "Several converging lines of evidence suggest that some behavioral and neurological abnormalities of childhood may be mediated through antineuronal antibodies. These antineuronal antibodies appear to arise in response to group A [beta]-hemolytic streptococcal (GABHS) infections and to cross-react with cells within the central nervous system (CNS). Based on clinical observations of children with Sydenham's chorea, Tourette's syndrome (TS), and/or obsessive-compulsive disorder (OCD), we hypothesize that neuroimmunological dysfunction secondary to antineuronal antibodies may result in behavioral disturbances, such as anxiety, emotional lability, obsessive compulsive symptoms, hyperactivity, and sleep disturbances, and neurological abnormalities, such as motor and phonic tics, ballismus, chorea, and choreiform movements."

"*Antineuronal antibodies: tics and obsessive-compulsive symptoms*" (Journal of Developmental and Behavioral Pediatrics, vol. 15, no. 6, December 1994, pp. 421-5): 19 or 38 cases from an ongoing study of childhood neurodevelopmental disorders had existing or previously documented OCS [OCD] and attention-deficit hyperactivity disorder (ADHD), with or without concomitant tics. 19 controls had ADHD, but no tics or OCS. Evidence was found of basal ganglia involvement in OCS, and a generalized central nervous system response [to infection] was suggested.

"*Bipolar disorders, dystonia, and compulsion after dysfunction of the cerebellum, dentatorubrothalamic tract, and substantia nigra*" (Biological Psychiatry, vol. 40, no. 8, October 1996, pp. 726-30): the mechanism of the legions was not abstracted in this report; however, after focal cerebellar circuit lesions, these disorders presented in three of fifteen subjects.

"*Antineuronal antibodies in movement disorders*" (Pediatrics, vol. 92, no. 1, July 1993, pp. 39-43): 24 children with recent-onset movement disorders (Tourette Syndrome, motor and/or vocal tics, chorea, and choreiform movements) as well as ADHD, behavior disorders, or learning disabilities were studied. The authors concluded that their data strongly suggests an association between antecedent group A beta-streptococcal infection and serum antineuronal antibodies, which may, in turn, be linked to childhood movement disorders.

"*Antibodies to human caudate nucleus neurons in Huntington's chorea*" (Journal of Clinical Investigation, vol. 59, no. 5, May 1977, pp. 922-32): IgG antibodies against nervous system components were detected in patients afflicted with Huntington's and Parkinson's Diseases, as well as in asymptomatic spouses of patients. "These data may support an environmental or infectious factor somehow involved in the ultimate expression of HD."

[The following report is not vaccine-specific, but underlines a radical shift in thinking about cerebral palsy, and a variety of other neurological impairments, toward an infectious etiology.]

"*Infections may underlie cerebral palsy*" (Science News, vol. 154, no. 16, October 17, 1998, p. 244; available at http://www.sciencenews.org/sn_arc98/10_17_98/fob1.htm): "Most doctors have believed that cerebral palsy--a form of brain damage that impairs movements--results from a difficult birth… While asphyxia may indeed be a cause of cerbral palsy, a new study provides evidence that the brain damage might often arise from some other…assault on an unborn child. Molecular clues now lead to inflammatory infection as a possible culprit, says Karein B. Nelson, a pediatric neurologist at the National Institute of Neurological Disorders and Stroke in Bethesday, MD." A study was performed by Nelson and colleagues which compared blood from normal and CP infants: the team found that all the

stricken children harbored greater concentrations of substances indicating immune activation. In some of the children, indications of autoimmunity were seen as well. (Study citation: "*Neonatal cytokines and coagulation factors in children with cerebral palsy*," Annals of Neurology, vol. 44, October 1998, p. 665.)

"*Increased prevalence of antibrain antibodies in the sera from schizophrenic patients*" (Schizophrenia Research, vol. 14, no. 1, December 1994, pp. 15-22); "*Antibodies to brain tissue in sera of schizophrenic patients-preliminary findings*" (European Archives of Psychiatry and Clinical Neuroscience, vol. 242, no. 5, 1993, pp. 314-7): Antibrain antibodies have been found in the sera of schizophrenic patients, but not in normal controls. These seem to be directed against brain centers affected in schizophrenia. (More notes on schizophrenia are available under "**Factoid Fallacies**," below.)

<<***cognitive disorders in systemic diseases***>>

"*Characteristics of antineuronal antibodies in systemic lupus erythematosus patients with and without central nervous system involvement: the role of mycobacterial cross-reacting antigens*" (Israeli Journal of Medical Science, vol. 26, no. 7, July 1990, pp. 367-73): indirect immunofluorescence of human brain tissue sections revealed, in thirteen of sixteen patients, high antineuronal antibody titers. Competition assays showed that the binding of the antineuronal antibodies was blocked by mycobacterial glycolipids and bovine brain extracts. "This finding suggests an additional link between mycobacterial infection and SLE."

<<*cognitive disorders of the aged*>>

"*Neuroautoimmunity: pathogenic implications for Alzheimer's disease*" (Gerontology, vol. 43, no.s 1-2, 1997, pp. 79-94): "Immune factors such as cellular immunity, autoimmunity, and inflammation may play a pathogenic role in Alzheimer's disease… Antibrain antibodies may contribute through a cell-specific autoimmune assault leading to neurodegeneration of the A[lzheimer's] D[isease] type."

"*Immunoblot detection of antibodies to myelin basic protein in Alzheimer's disease patients*" (Neuroscience Letters, vol. 147, no. 1, November 23, 1992, pp. 25-8): 16 of 18 Alzheimer's disease patients, as opposed to 7 of 90 controls (healthy adults and elderlies, and adult and child patients with other diseases or disorders such as Parkinson's disease and Down syndrome proved positive for anti-MBP antibodies.

"*An immunological approach to dementia in the elderly*" (Age and Ageing, vol. 5, no. 3, August 1976, pp. 164-70): Immunofluorescence studies showed "an excess of antineuronal reactivity and a fall in antinuclear antibody in females with senile dementia."

<<*cognitive developmental disorders*>>

"*Serum autoantibodies to brain in Landau-Kleffner variant, autism, and other neurologic disorders*" (Journal of Pediatrics, vol. 134, no. 5, May 1999, pp. 607-613): "Etiologically unexplained disorders of language and social development have often been reported to improve in patients treated with immune-modulating regimens. Here we determined…children with L[andau] K[leffner] S[ydrome] V[ariant] and A[utistic] S[pectrum] D[isorder] have a greater frequency of serum antibodies to brain endothelial cells and to nuclei than children with non-neurologic illnesses or healthy children. The presence of these antibodies raises the possibility that autoimmunity plays a role in the pathogenesis of language and social developmental abnormalities in a subset of children with these disorders.

"*Serological Association of Measles Virus and Human Herpesvirus-6 With Brain Autoantibodies in Autism*." Clinical Immunology and Immunopathology, vol. 89, number 1, October 1998, pp. 105-8. This study is the first to report an association between virus serology and brain autoantibody in autism; it supports the hypothesis that a virus-induced autoimmune response may play a causal role in autism.

"*Positive Titers of Measles and Measles-Mumps-Rubella Antibody Are Related to Myelin Basic Protein Autoantibody in Autism*." Abstract of study prepared for the annual meeting of the American Association of

Immunologists (AAI) / Federation of American Societies for Experimental Biology (FASEB), San Francisco, April 1998. A significant number of autistic children exhibit positive titers of measles and MMR [measles-mumps-rubella] antibody, which in a vast majority of cases is associated with the presence of MBP [myelin basic protein, or brain] autoantibody. A measles- and/or MMR-triggered autoimmune response to myelin may play a pathogenesis role in autism.

"*Association of Anti-MBP and Anti-NAFP Antibodies With HHV-6 Antibodies in a Child With Autistic Regression*." Journal of Allergy and Clinical Immunology, vol. 101.1, S122, January 1998, part 2 (in section entitled, "*Program and Abstracts of Papers to Be Presented During Scientific Sessions [at the] 54th Annual Meeting, March 13-18, 1998*").

"*Circulating Autoantibodies to Neuronal and Glial Filament Proteins in Autism*." Pediatric Neurology, vol. 17, number 1, July 1997, pp. 88-90. A significant increase in incidence of anti-NAFP [neuron-axon-filament-protein] and anti-GFAP was seen in autistic subjects, but not in mentally retarded subjects. Clinically, these autoantibodies may be related to autoimmune pathology in autism.

"*Hyperserotoninemia and Serotonin Receptor Antibodies in Children With Autism but Not Mental Retardation*." Biological Psychiatry, vol. 41, number 6, March 15, 1997, pp. 753-5.

"*Elevated Serotonin Levels in Autism: Association With the Major Histocompatibility Complex*." Neuropsychobiology, vol. 34, number 2, 1996, pp. 72-5. Two of the most consistently observed biological findings in autism are increased serotonin levels in the blood and immunological abnormalities (including autoreactivity with tissues of the central nervous system). The major histocompatibiligy complex (MHC) regulates the immune system, and is associated with autoimmune disorders. In this study, a positive relationship was observed between elevated serotonin levels and the MHC types previously associated with autism.

"*Plasma Increase of Interleukin-12 and Interferon-gamma. Pathological Significance in Autism.*" Journal of Neuroimmunology, vol. 66, numbers 1-2, May 1996, pp. 143-5. Immune factors such as autoimmunity have been implicated in the genesis of autism, a neurodevelopmental disorder. Since autoimmune response involves immune activation, the plasma levels of interferon-alpha (IFN-alpha), IFN-gamma, interleukin-12 (IL-12), and IL-6 were measured, along with tumor necrosis factor (TNF-alpha) and soluble intercellular adhesion molecule-1 (sICAM-1). The levels of IL-12 and IFN-gamma were significantly higher in autistic patients than in controls (the remaining measures were not significantly different). It is suggested that IL-12 and IFN-gamma increases may indicate antigenic stimulation of Th-1 cells pathogenetically linked to autoimmunity in autism.

"*Immunogenetic Studies in Autism and Related Disorders*." Molecular Chemistry and Neuropathology, vol. 28, numbers 1-3, May-August 1996, pp. 77-81. The major histocompatibiligy complex comprises a number of genes that control the function and regulation of the immune system. One of these, the C4B gene, encodes a product that is involved in eliminating pathogens such as viruses and bacteria from the body. A deficient form of the C4B gene, termed the C4B null allele (no C4B protein produced) was previously seen to have an increased frequency in autism. In this study, this finding was confirmed, and this same condition was detected in related [neurodevelopmental] disorders as well. In addition, two alleles of the DR beta 1 gene also had significantly increased representation in autistic subjects.

"*Antibodies to Myelin Basic Protein in Children With Autistic Behavior*." Brain, Behavior and Immunity, vol. 7, number 1, March 1993, pp. 97-103. Approximately 58% of the sera of autistic children were found to be positive for anti-MBP [anti-brain antibodies]. This result was significantly different from that of the controls, among whom were children with normal health, idiopathic mental retardation, and Down syndrome. It is possible that anti-MBP antibodies are associated with the development of autistic behavior.

"*Possible Association of the Extended MHC Haplotype B44-SC30-DR4 With Autism.*" Immunogenetics, vol. 36, number 4, 1992, pp. 203-7. The complement C4B null allele appears to be associated with infantile autism. In this study, the incidence of B44-SC30-DR4 was increased by almost six-fold in the autistic subjects as compared with healthy controls. Moreover, the total number of extended haplotypes expressed on chromosomes of autistic subjects

was significantly increased as compared with those expressed on chromosomes of healthy subjects. Conclusion: a gene related to, or included in, the extended major histocompatibility complex may be associated with autism.

"*Increased Frequency of the Null Allele at the Complement C4b Locus in Autism*." Clinical Experiments in Immunology, vol. 83, number 3, March 1991, pp. 438-40. Associations between C4 deficiency and autoimmune disorders have been found over the past several years. In this study, autistic subjects and their mothers had significantly increased phenotypic frequencies of the C4B null allele, compared with controls. The siblings of the autistic subjects also had an increased frequency of the C4B null allele, but this was not significant. The fathers did not display this allele. All family members had normal frequencies of the C4A null allele, all normal C4A and C4B alleles and all BF and C2 alleles.

"*Changes of Soluble Interleukin-2, Interleukin-2 Receptor, T8 Antigen, and Interleukin-1 in the Serum of Autistic Children*." Clinical Immunology and Immunopathology, vol. 61, number 3, December 1991, pp. 448-455. Findings indirectly indicated that the activation of a subpopulation of T cells occurs in some children with autism, as opposed to healthy children or children with mental retardation (non-Down's syndrome).

"*Deficiency of Suppressor-inducer (CD4+CD45RA+) T Cells in Autism*." Immunological Investigations, vol. 19, number 3, June 1990, pp. 245-51. Autistic subjects as compared to a group of 35 healthy age-matched subjects had a significantly reduced number of lymphocytes, a decreased number of CD2+ T cells and reduced numbers of CD4+ and CD4+CD45RA+ lymphocytes. Results suggest that an alteration in the suppressor-inducer T-cell subset is associated with autism.

"*CD4+ Helper T Cell Depression in Autism*." Immunology Letters, vol. 25, number 4, September 1990, pp. 341-5. Autistic subjects had a significantly lower percentage and number of CD4+ cells, a lower number of T cells (CD2+ cells) and B cells (CD20+ cells), and a lower percentage and number of total lymphocytes than siblings and normal subjects. The level of blood values for female subjects appeared lower than those for males as compared to normal subjects of the same sex. Results suggest that a decrease in CD4+ cells is associated with autism.

--for cognitive disorders see also "*Vaccination against whooping-cough. Efficacy versus risks*," The Lancet, vol. 1, January 29, 1977, pp. 234-7, above, in <<*seizure disorders*>>.

"Survival of the fittest"—'morphing' of eradicated viral diseases into new diseases

Dr. Bruce P. Squires remarked, "it is ironic that as smallpox was finally being eradicated in the late 1970s, another virus [HIV] was being transmitted to unsuspecting hosts" (Canadian Medical Association Journal, vol. 149, 1993, p. 919). Dr. James H. Battershill comments on this in the January 15, 1994 issue of the Journal (vol. 150, no. 2, p. 128): "…I have often wondered whether there was a relation between the cessation of small-pox vaccination and the appearance of AIDS. A shared antigen, perhaps?" Whatever the case, viruses are extremely clever survivors, able to mutate into new disease forms when their original presentations are defeated by vaccines. In **Factoid Fallacies**, below, the section on "Disease eradication" deals with this theme in part.

"*Is RA27/3 rubella immunization a cause of chronic fatigue?*" (Medical Hypotheses, vol. 27, no. 3, November 1988, pp. 217-20): "Patients with chronic fatigue syndromes (primary fibrositis syndrome, major affective disorder, etc.) have elevated IgG serum antibodies to multiple common viruses. Only IgG rubella antibodies are positively correlated with the intensity of symptoms and have a height that is clearly significant compared to healthy controls…A new more potent strain of live rubella vaccine (strain RA27/3) was introduced in 1979. Within three years reports of patients with chronic fatigue began surfacing in the literature. Considering all this, the possible role of rubella immunization in the etiology of chronic fatigue syndromes deserves further study."

"*Second generation" -- layered -- antibodies*

A problem unforeseen in the development of vaccines for common diseases is the advent of high antibody counts in fully-vaccinated parents who, in time, have children who are also vaccinated. Similarly, the effects of repeated vaccination or vaccination of persons with high antibody levels warrants careful observation and study. In

"*Combination vaccines for childhood immunization: Recommendations*" (American Academy of Pediatrics/American Academy of Family Physicians), the safety and efficacy of administering combination (multivalent) vaccines to patients who already have immunity to one or more vaccine component (natural or via monovalent vaccine) is listed as a future research priority, indicating that the safety of this practice has not been proven. Even for persons not already immune to vaccine components, "the effects on immunogenicity and safety of simultaneous or repeated exposures to the same proteins used as antigens…and/or as carrier components" is at issue (pp. 10-12, Morbidity and Mortality Weekly Report, vol. 48, RR05, May 14, 1999, pp. 1-15; text available http://www.cdc.gov/epo/mmwr/preview/mmwrhtml/rr4805a1.htm).

"*Associations of prevaccination antibody levels with adverse reactions to pneumococcal and influenza vaccines*" (Vaccine, vol. 15, no. 10, July 1997, pp. 1133-7): The authors studied 85 elderly subjects vaccinated simultaneously with pneumococcal and influenza vaccines. Those subjects higher prevaccination antibody levels experienced a greater rise in body temperature post-vaccination. Injection site pain was more common with patients with higher pre-existing antibody counts in the case of the pneumococcal vaccine.

Death

SIDS (Sudden Infant Death Syndrome) has been associated with the diphtheria-pertussis-tetanus (DPT) vaccine. The new hepatitis B vaccine seems to present similar risks in use with infants. Cancer is an end result more and more frequently linked with viral contaminates of vaccines cultured in animal tissues. Other vaccine-precipitated diseases (featured above) can also result in death.[47]

"*Association says numbers show mandatory vaccinations not best for children*" (New York Times Syndicate news release, April 28, 1999): the Association of American Physicians and Surgeons found that children younger than 14 are three times more likely to be killed or seriously injured by the hepatitis B vaccines than they were to catch the disease, which is not spread by casual contact, but rather by injection, sex, or an infected mother.

Alteration of human genetic code

Viruses are able to infiltrate cells, inserting their genetic material into them. Indications have been found of changes to human genetic characteristics as a result of viral invasion.

"*Role of Immunogenetics in the Diagnosis of Postvaccinal CNS Pathology*," Department of Pediatric Surgery, University of Bari, Italy, presented May 9, 1996 (text available http://www.healthy.net/library/articles/coulter/biochem.htm):[7] initially, thirty young children were tested and followed who showed the first symptoms of CNS pathology with or immediately after vaccination with polio, DT, measles, DPT, anti-tuberculosis, or Hepatitis-B vaccines. Immediate reactions to the vaccines included convulsions, high fever, or diarrhea with or immediately after vaccination. Among the post-vaccinal symptoms were encephalopathies, food allergies, constipation, diarrhea, and other central nervous system pathology. Diagnoses applied to subjects after vaccination and before this study were epilepsy of various types; epileptigenic encephalopathy, autism, West Syndrome, and Angelman's Syndrome.

There were no genetic or metabolic anomalies revealed during testing which might have explained the CNS symptoms. The viral encephalopathies which presented with or following vaccination were not due to transplacental viral infection. EEGs after initial symptoms were negative in 92 percent. Following vaccination and CNS symptoms, serologic investigations for herpes viruses were positive in all cases for IgG. IgG for Epstein-Barr virus and cytomegalovirus were estimated to be positive in 73.8/71.4 percent respectively, herpes simplex in 47.6 percent, and varicella zoster in 21.4 percent of patients. 73.3 percent of subjects showed an increase in the HLA-A3 and HLA-DR7 antigens as compared with the Italian population at large.

The authors found and describe, in this paper, biochemical markers of vaccine damage (e.g., changes in inherited HLA type). They also point out that most vaccines contain thimerosal, a toxic substance associated with neurologic and gastrointestinal symptoms. The fact that post-vaccinal pathologies of the central nervous system are often not

thoroughly investigated occasioned this study. *Additional cases are under study to better define the possible association of HLA A3 and/or HLA DR7 with this CNS pathology following vaccination.*

"*New Genetic Study Points Way for Vaccine Reaction Research/Novel Genetic Clinical Marker Found in Blood of Gulfwar Vets*" (Press release, National Vaccine Information Center/PR Newswire, Washington, D.C., May 3, 1999, 5:48 p.m.; original source is Clinical and Diagnostic Laboratory Immunology, May 1999): A three year study funded and conducted by the Chronic Illness Research Foundation in collaboration with the University of Michigan School of Medicine found abnormal RNA in the blood of 50 percent of sick Gulf War veterans, indicating that chromosomal damage had occurred. This genetic material was not found in any of the healthy controls. Damage to chromosome 22q11.2 has been linked in other published studies to autoimmune diseases such as juvenile rheumatoid arthritis and other illnesses like multiple myeloma cancer. The discovery of RNA in the cell-free fractions of blood is an anomaly, as it is not normally present in serum. RNA can exist outside the cell only if it is protected, as RNA viruses can. Gulf War soldiers were given 17 different viral and bacterial vaccines, including experimental anthrax and botulinum toxoid vaccines. Experimental drugs were also given and [in veterans actually deployed to the Gulf] there were exposures to pesticides, low-level chemical warfare agents, low-level radiation, toxic combustion products, etc. The resultant symptoms are similar to those of vaccine-damaged children. Dr. Howard B. Urnovitz, microbiologist and Science Director of the Chronic Illness Research Foundation, interpreted findings to indicate that certain genotypes may be particularly at risk for sustaining chromosomal damage after exposure to toxic events; ways to identify and prescreen for individuals who may be at high risk for chromosomal damage should be found.

The Wakefield study---and others

Work at the Royal Free Hospital, London

The February 28, 1998 issue of the prestigious journal The Lancet contained the study which occasioned London's "*ABCs*" venture: "*Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children*," by Andrew Wakefield and a team of British scientists.[8] Dr. Wakefield, an internationally-known gastroenterologist, and colleagues had been searching for nearly a decade for the origins of Crohn's disease, a debilitating, increasingly common inflammatory bowel disorder. In "*Ileal-lymphoid-nodular-hyperplasia*," the British scientists investigated, initially, a group of twelve children ages three to ten; by January 1998 Wakefield's team totalled fifty-one investigations of children with regressive autistic spectrum disorders and inflammatory bowel disease and, as of July 1998, had at least one year's worth of additional examinations scheduled as parents of similarly autistic children queued up.[9]

The measles virus had long been a part of Wakefield, et al.'s investigations of Crohn's disease because of the virus' vascular effects.[10] The children in Wakefield et al.'s study met developmental milestones normally until the administration of the MMR or MR vaccine or until experiencing wild measles. At varying intervals following these events, the patients regressed noticeably, as susceptible people have been known to do in cases of complicated wild measles, and even with vaccines; eventually they were diagnosed with autism, PDD, disintegrative psychosis, or vaccinal encephalitis.[11] Known causes of childhood neurodegenerative disease were excluded through testing. There were no focal neurological abnormalities. MRI and EEG testing in these young sufferers of autistic enterocolitis"[12] was normal.[13] Ultimately, each was found to have classic signs of inflammatory bowel disease, with immune activation markers.[14] Measles virus proteins were discovered in the germinal centers of inflamed lymphoid tissue in the guts. Tissue samples proved negative for other viruses, parasites or other organisms.[15]

Wakefield, et al.'s conclusions in perspective

Before the Wakefield "early report" of February 1998, the measles virus and measles vaccination had been identified as a risk factor for Crohn's disease, and persistent (chronic) measles vaccine-strain virus infection had been found in children with autoimmune hepatitis.[16] That the intestinal and 'autistic' behavioral pathologies occurred together in these children might have happened by chance, but for the uniformity of the intestinal signs and the findings of intestinal dysfunction in previous studies of autistic children. Extensive metabolic dysfunction in autism had also been described previously, and in great detail. Disintegrative disorders had long been linked to measles and rubella infection ("*Ileal-lymphoid-nodular-hyperplasia*," February 1998; conference, Tulane University, July 1998). Researchers such as Fudenberg and Gupta and others had previously commented on similar developments in children in relation to vaccination.[17] Billed as an "Early Report" in the Lancet's terminology, "*Ileal-lymphoid-nodular-hyperplasia*" did not demonstrate a link between the MMR vaccine and "autistic enterocolitis," but underlined clearly the need for further research into this intestinal/developmental syndrome and its possible relation to the MMR vaccine. Wakefield, et al. offer a possible explanation for the syndrome's prevalence in certain children:

Published evidence is inadequate to show whether there is a change in incidence [of this syndrome] or a link with measles, mumps, and rubella vaccine… In the context of susceptibility to infection, a genetic association with autism, linked to a null allele of the complement C4B-gene located in the class III region of the major-histocompatibility complex, has been recorded by Warren and colleagues. C4B-gene products are crucial for the activation of the complement pathway and protection against infection: individuals inheriting one or two C4B null alleles may not handle certain viruses appropriately, possibly including attenuated strains.[18]

Investigative work continues in this area, as the British team of scientists look for molecular evidence of persistent measles virus infection in the intestinal tissues (conference). A first effort to accomplish this failed as chosen methods proved insufficiently sensitive.[19]

Vaccine-theory reactions and support

A firestorm of criticism met the Wakefield study. Though scrutinized pre-publication by *four* peer-reviewers, rather than the usual two, for inclusion in The Lancet, public health officials, their medical associates, and others denigrated the study and its author. Normally, commentaries on such Lancet publications are brief and are placed in subsequent issues of The Lancet, in the "letters" section, using the title of the article commented upon. In the case of "*Ileal-lymphoid-nodular-hyperplasia*," however, a rebuttal article by Robert Chen of the Centers for Disease Control was placed in the same issue, and given equal presence in the table of contents.[20] In the NARRATIVE article, "*The ABCs of MMRs and DTPs*," London begins his discussion of the Wakefield study 'on the wrong foot,' misquoting the name of its principal author. London cites a selectively few studies or opinions critical of Wakefield's work, ignoring several which are compellingly supportive, both from previous publications and publications which followed the February 28th Early Report—most notably, the replication of Wakefield's work with a group of children with ADD and ADHD and food allergy symptoms, at the International Center for Interdisciplinary Studies of Immunology and Department of Pediatrics, Georgetown University Medical Center, Washington, D.C. A reactionary study by a Finnish group is used by London in "*ABCs*," and has been used by others, as proof of no association between autism, inflammatory bowel disease and measles, though its methodology was in actuality poor, and its conclusions not applicable to the Wakefield findings. The Georgetown University study is reported in The Lancet as follows:

A J Wakefield and co-workers have identified a new relation between gastrointestinal disease and developmental disorders in children; it opens a new avenue for the study of the gastrointestinal tract and other diseases that may be immunologically mediated. Their findings of ileal-lymphoid-nodular hyperplasia and non-specific colitis gastrointestinal manifestations in connection with autistic-spectrum disorders is the first description of this relation, with strong data suggesting the anatomical and histological alteration of the gut in such disorders. Although these workers suggest possible mechanism(s) of increased permeability for exogenous molecules they do not offer any explanation for these gastrointestinal alterations. The endoscopic and histopathological findings of ileal-lymphoid-nodular hyperplasia and non-specific colitis have so far escaped explanation and have evaded pathogenetic definition.

In support of the findings of Wakefield et al are several behavioural and clinical features known to be related to the central nervous system (CNS), such as migraine, infantile colic, abdominal epilepsy, allergic-tension-fatigue syndrome, and attention-deficit-hyperactivity disorder, which have been related to food allergy, although the precise relation is still unclear. IgE-mediated food allergy is plainly not the only mechanism of tissue injury, and these specific disorders could involve other mechanisms.

A major investigative effort of our laboratories has been directed to the study of food allergy and the immunological involvement of the gut as a central focus for injury of other target organs (skin, lungs, and gastrointestinal tract). We have noted a striking appearance of ileal-lymphoid-nodular hyperplasia in patients with non-IgE-mediated food allergy who present with asthma, atopic dermatitis, and attention-deficit-hyperactivity disorder. We have also studied two patients with this hyperactive disorder who were allergic to various foods, and ***our findings*** *obtained by colonoscopy of their terminal ileum, shown in the figure****, match with those reported by Wakefield and co-workers.***

In our study, ileal-lymphoid-nodular hyperplasia is the hallmark lesion of the gastrointestinal tract, which allows entry of antigens across the inflamed mucosa of the bowel as a result of the reactive inflammatory response in the adjacent lymphoid tissue of Peyer's patches in patients with non-IgE-mediated food allergy. We propose that similar mechanism(s) may be involved in the pathogenesis of the CNS dysfunction in the patients described by Wakefield and co-workers.

Although Wakefield's study, which suggests a connection between the CNS and the gut in patients previously immunised with measles, mumps, and rubella vaccine, did not prove an association [Wakefield did call for further research which would be required to accomplish this], *it has stimulated further discussion and opened unanticipated lines of investigation concerning the role of ileal-lymphoid-nodular hyperplasia as a predictive marker of gastrointestinal inflammation responsible for immunologically mediated tissue injury in other target organs sites.*[21]

Wakefield's recommendations

London asserts, on page 15 of "*ABCs*," that Wakefield, et al. advocated use of existing vaccination schedules and products in the absence of further information about possible relationships between vaccine viruses and conditions such as autism. In "*Ileal-lymphoid-nodular-hyperplasia*," however, Andrew Wakefield, et al. actually suggest that, until research can be done to further clarify possible MMR problems in some children, that measles, mumps, and rubella vaccines be given *monovalently* (separately, during separate office visits, spaced widely), to avoid interactions that may encourage pathogenesis.

There is precedent for this line of thought. In 1989, a physician responds to reports of mumps encephalitis following MMR administration with a report of a case of mumps encephalitis after simultaneous mumps and adenovirus infections, noting "the possibility that children are more vulnerable to complications if they are exposed to more than one virus simultaneously" (The Lancet, vol. 2, August 12, 1989, pp. 394-5). In "*Adverse reactions associated with simultaneous administration of multiple vaccines to travelers*" (Journal of General Internal Medicine, vol. 9, no. 5, May 1994, pp. 255-60), the authors conclude, "Increasing the number of vaccines [and thus the number of viruses contacted simultaneously] is associated with increasing the rates of [both] local and systemic reactions." "*Mumps and Measles in [the] same year [is] linked to Later Inflammatory Bowel Disease*," stated a Reuters Medical News release dated April 6, 1999, and "*mumps infection in the same year as monovalent measles vaccination appeared to increase the risk of later Crohn's disease*."[22] The tender age at which children are vaccinated for—at present count—some 22 diseases may also contribute to the number of pathogenic reactions. In "*Age-dependent susceptibility in mumps-associated hydrocephalus: neuropathologic features and brain barriers*," Acta Neuropathologica, vol. 94, no. 3, September 1997, pp. 207-15, the authors state, "central nervous system susceptibility to viral infection is often age dependent for unclear reasons… Our results suggest that tight junctions in the early postnatal period are more immature and fragile than in the adult. We concluded that brain susceptibility in mumps virus-induced hydrocephalus is intimately related to the maturity of brain barriers." In "*Evaluation of humoral responsiveness in children*," Pediatric Infectious Disease Journal, vol. 11, no. 4, April 1992, pp. 304-10, the authors relate, "the wide age-dependent variation in antibody responses makes careful interpretation necessary in concluding that a pateitn falls outside the 'normal' pattern… Interpretation of antibody responses in children younger than 2 years of age is probably hazardous since manyu patients may have relative retardation in their normal development of polysaccharide antibody responsiveness."

Repeated challenges may also be a problem. In "*Over-immunization--an ever present problem*" (Australian Family Physician, vol. 5, no. 6, July 1976, pp. 734-55) the authors warn against a sense of false security [about immunization] and notes a tendency among the medical community to over-immunize. "Repeated antigenic challenge may cause hypersensitivity reactions which could harm the individual… The need to appreciate the dangers inherent in all immunization procedures in general, and in certain vaccines in particular, is emphasized [in the full-text article]." In "*Combination vaccines for childhood immunization: Recommendations*" (American Academy of Pediatrics/American Academy of Family Physicians), the Academies list as future research priorities the safety of administering combination vaccines to patients who might already have immunity (natural or vaccine-conveyed) to one or more component, as well as the safety of repeated exposures to the same proteins used as antigens and/or carrier components in existing and future conjugated vaccines is listed as a research priority--implying that the safety of this prevailing practice has not been satisfactorily proven. "Chemical incompatibility or immunologic interference when different antigens are combined into one vaccine could be difficult to overcome" (p. 8; pp. 10-12, Morbidity and Mortality Weekly Report, vol. 48, RR05, May 14, 1999, pp. 1-15; text available http://www.cdc.gov/epo/mmwr/preview/mmwrhtml/rr4805a1.htm).

Broader implications

Their association with a variety of diseases and conditions such as learning disabilities, hyperactivity, and conduct disorders seems to implicate viral activity, as discussed in Harris Coulter's Vaccination, Social Violence, and Criminality (Center for Empirical Medicine, 1990).

The "Thalidomide Connection"

Prior to birth, thalidomide embryopathy has been associated with autism ("*Etiology of infantile autism*," Journal of Psychiatry and Neuroscience, vol. 24, no. 2, March 1999, pp. 103-15). Environmental insults are thought to play a role in the etiology of autism after birth as well. Unknown to most consumers, toxic substances like thimerosal (mercury) and formalin (a formaldehyde derivative) are commonly used in vaccines.

"*Reactions to thimerosal in hepatitis B vaccines*" (Dermatologic Clinics, vol. 8, no. 1, January 1990, pp. 161-4): "Hypersensitivity to thimerosal in vaccines had been reported to induce persistent local reactions, urticarial and generalized exanthematic eruptions, and, in the case of the hepatitis B vaccine, urticaria with asthma. The authors describe two cases of extensive reactions…Although not all thimerosal-sensitive patients develop adverse reactions to vaccines containing this material, there is a potential risk, and the reactions can be very long lasting."

Vaccine necessity: manufactured epidemics

Historically the medical profession, and even more frequently the United States and foreign agencies responsible for disease prevention and control, have forecast epidemics or exaggerated the prevalence and threat of vaccine-preventable diseases many times, urging immediate, mandated vaccination of all persons, without actual epidemics or other evidence manifesting to justify these efforts. Invoking the memory of the worldwide influenza pandemic of 1918-19, government scientists and bureaucrats spread fear of a new pandemic of this "Swine flu," predicted for the late 1970s, in spite of the fact that "virologic science had no way to predict in advance the severity of a pandemic" (pp. 23 and 30,Pure Politics and Impure Science: The Swine Flu Affair, Johns Hopkins University Press, 1981). A vaccine was concocted, hastily, and a national immunization program was legislated into action. Not only did the pandemic fail to arrive, but many Swine Flu vaccinees contracted Guillain-Barre Syndrome, a body-wide, paralyzing autoimmune disorder. Some died from the Guillain-Barre, or died from the vaccine itself.

Investigative journalist Janine Roberts documents an urgent warning issued in the United Kingdom in November 1994, forecasting a measles epidemic which would infect between 100 and 200,000 children, of which some 50 would die--if children ages 5 to 15 were not immediately revaccinated for measles, mumps and rubella ("*The Fraudulent Measles Epidemic;*" posted as part of The Web Inquirer, September 29, 1995, http://www.gn.apc.org/inquirer/fraud.html; posted April 8, 1999, 12:13 a.m. on Vaccine Information and Awareness discussion list, via@access1.net). Roberts quotes Dr. Richard Nicholson, editor of the Bulletin of Medical Ethics, who studied

government reports and declared that there was no proof that such an epidemic was about to begin. The government's estimate of the number to be infected and killed, Nicholson stated, was based on "improper use of statistics and out of date death rates."

Interestingly, a British law firm investigating vaccine deaths and injuries notes a change in the way the diseases measles, mumps and rubella are regarded in medical reference texts, before and after vaccines for these became available--i.e., the diseases are first described as mild and transient, with low incidence of complications, then potentially serious, with considerable risk of complications, *after* the vaccine came into existence (Richard Barr and Kirsten Limb, "*Vaccines. Fact Sheet,*" section three, "*Background: Setting the illnesses in context*;" text available from the Society for the Autistically Handicapped, http://www.rmplc.co.uk/eduweb/sites/autism/index.html).

--see also **Factoid Fallacies**: …the severity of vaccine-preventable diseases..."*all of these illnesses are killers*," below--

Popular statistics (Vaccine efficacy, safety, and other figures)

The purvey of statistics is an art—and a science—which is fascinating and useful but highly manipulable. Statistics are easily concocted and are literally bought and sold in support of a wide variety of enterprises. Statistics on vaccine safety and efficacy have been published by the Centers for Disease Control which are favorable to the pharmaceutical industry and the government's efforts to promote immunization, but these have been widely challenged. As important as the statistics themselves are the methods, sources, and motives by which they are assembled. Government statistics which describe the number of incidents of death or serious injury by vaccines as "rare" are based on a set of criteria which exclude almost every case on record with VAERS, the government-established vaccine injury reporting facility--most often because the vaccine-caused death, injury, illness or disability occurred beyond a very narrow time period, such as seventy-two hours. Because of the lack of conclusive, long-term studies, many disabilities and diseases thought to be connected with viral activity are not included in the government's vaccine injuries compensation table, and as a result are not included in published statistics. Increasing the unreliability of these statistics is the unfortunate reality that most vaccine reactions are not reported; VAERS is a passive reporting system heavily dependent on physicians' and citizens' abilities to recognize, and willingness to report, vaccine reactions. Government statistics on vaccine efficacy and safety are listed and analyzed in Neil Z. Miller's *Vaccines: Are They Really Safe and Effective*? (Santa Fe: New Atlantean Press, 1992/1998).

To date, there has been no concerted national effort to determine the actual incidence of autism--one of the many disabilities/diseases that can be caused by immunization--since it was described by Kanner in the 1940s. The most frequently quoted incidence figure, based on large-scale surveys in the United States in England, is 4.5 in 10,000--not including other autism spectrum disorders.[23] Presently the CDC admits to an estimated autism incidence of 1 in 500 (contrasted with an earlier estimate of 1 in 10,000). The actual incidence is suspected by the autism community to be at least this high, perhaps higher than 1 in 200, and to be increasing exponentially [coincidentally or not, the number of mandated or recommended immunizations is also rapidly expanding].[24] In response to a recent inquiry, the Assessment, Evaluation and Support Unit of the California Department of Education's Special Education Division gave the following statement about increases in the numbers of special education students with autism:

"*…the only info I can provide on Autism is the number of students, ages 0-22, with IEPs receiving special education services in California. We first started collecting enrollments count on Autism in the 1992-93 school year. On April 1, 1993, there were 2,157 students (out of 540,472) reported* [to have autism]. *Five years later, on April 1, 1998, there were 8,084 students (out of 632,238) reported.*"[25]

The California Department of Developmental Services recently produced *A Report to the Legislature: Changes in the Population of Persons with Autism and Pervasive Developmental Disorders in California's Developmental Services System: 1987 through 1998* (text available at http://www.dds.ca.gov/autismreport.cfm). In 1987 there were 3,864 autistic children enrolled in the department's twenty-one regional programs; in 1998, 11,995 autistic children were counted--a 210% increase. Children with other disorders such as cerebral palsy and epilepsy increased at a rate consistent with the state's population growth (from 30-48%). The Illinois State Board of Education lists figures for the incidence of both autism *and*learning disabilities: autism cases on hand were 317 in 1991, and 2,305 in 1997. The number of children with learning disabilities was, in 1991, 111,326, and in 1997, 126, 065.[26]

While reviewing published statistics on vaccine safety and efficacy, a wide variety of statistics need to be compiled and considered in the same context--e.g., the overall number of children presently in special education, against the number in special schools or institutions, or at home due to disability, thirty to forty years ago, prior to widespread immunization; the number of children on public assistance (disability) rolls, and the present number of juvenile criminals and the types of crimes committed, presently and pre-immunization. Also highly relevant to this study are the incidences of pediatric illnesses such as asthma, leukemia, Crohn's Disease, disorders of the ear, autoimmune diabetes, etc., before and after the advent of vaccination. These should, of course, be weighed against population increases and other factors. These considerations are targeted in Harris Coulter's Vaccination, Social Violence, and Criminality (Center for Empirical Medicine, 1990).

Factoid fallacies

A number of short statements or rhetorical questions in the Eric London's "*ABCs of MMRs and DTPs*" should be reconsidered in the light of existing information:

(Page 14, London) "*…Schizophrenia* may *work the same way* [as autism]*: researchers have now found a great deal of evidence that schizophrenia happens in the womb.…* [no citations]"

In adjacent passages, London strongly implies that schizophrenia and autism are not only similar, but are purely genetic—not immunological or viral.

Autism was at one time called "childhood schizophrenia." It is now known to be a very different phenomenon. The Diagnostic and Statistical Manual of Mental Disorders, fourth edition, 1994, says, on page 66, "there is considerable evidence to suggest that the Pervasive Developmental Disorders are distinct from Schizophrena…" Michael Rutter and Eric Schopler observe, in Diagnosis and Assessment in Autism (1988), pages 16 and 17, "The likely discontinuity between autism and schizophrenia is strongly indicated by the bimodal distribution of age of onset…[it is] improbable that autism and schizophrenia constitute subvarieties of the same basic condition…in addition, autism and schizophrenia differ sharply in family history (a familial loading of schizophrenia is rare with autism), in phenomenology (delusions and hallucinations are rare in autism), in course (often episodic with periods of normality or near-normality in schizophrenia, but persistent in autism), and in the association with epileptic seizures (rare in schizophrenia but present in about a quarter of cases of autism)." On page 84, Volkmar and Cohen, also in Diagnosis and Assessment, 1988, elaborate on the differences between "childhood schizophrenia" and autism, referring to research. Gary Mesibov and colleagues reflect this same viewpoint in Autism: Understanding the Disorder, 1997: "today, with the accumulation of much evidence distinguishing them, autism and schizophrenia are viewed as completely separate disorders," giving a history of thought concerning these two illnesses and contrasting them further (pp. 42-3).

It is worth noting, however, certain similarities between schizophrenia and autism which go unremarked in London's article: Dr. Kalle Reichelt of the Pediatric Research Institute, Oslo, Norway, describes a body of research linking both schizophrenia and autism to faulty metabolism of gluten and casein-containing foods, permeable gut, and raised antibody levels against gluten and other food proteins.[32] Knight, et al., ask the riveting question, *"Can autoimmune mechanisms account for the genetic predisposition to schizophrenia*?"[33] Spivak, et al. note a diminished total haemolytic activity in schizophrenia not attributable to drug treatment, which might be related to the involvement of an autoimmune process in the pathophysiology of schizophrenia.[34] A study performed by Sirota, et al., found that anti-nuclear antibodies and anti-DNA autoantibodies were significantly more frequent in both schizophrenic patients and healthy relatives than in normal subjects, concluding, "*The data indicate that an autoimmune process may be involved in the etiology of a subset of patients with schizophrenia*."[35] A. E. Henneberg, working with various colleagues, found significant increases in the numbers of certain T-lymphocyte subpopulations in schizophrenic patients compared to controls. Antibodies to brain tissue were found in schizophrenic patients, but not in controls; the action of these was mainly directed against neurons in the frontal cortex and septal areas, which are regarded as important in the development of schizophrenia.[46]

In Immunologic Mechanisms in Neurologic and Psychiatric Disease, 1990, the authors examine psychiatric disorders from an neuroimmunological perspective—especially schizophrenia. On page 162 of Immunologic

Mechanisms it appears that, though the role of viral infection in the pathogenesis of schizophrenia has been studied, the findings did not (as of 1990) show substantial evidence linking a specific virus to schizophrenia [unlike in autism]—although "schizophreniform disturbances" have been noted in HIV infection (pp. 302-3). "*Recently, it has been suggested that retroviruses may be involved in the etiology of schizophrenia; however, this notion also lacks support* [i.e., sufficient research]." Instead, "numerous studies have considered the possibility that schizophrenia is owing to an autoimmune disorder that involves the brain (page 162)."[36] However, a slightly earlier Russian study found statistically significant elevation in the anti-smallpox antibody titers of 77 schizophrenics, compared with 44 normal controls. There was also "a certain increase" in antibody levels to measles virus. There was no difference between the schizophrenics and controls concerning two other viruses. A relationship between antibody production and the severity and course of the disease, as well as the age of the subjects, was noted ("*Antibodies to measles, smallpox, influenza and arenaviruses in schizophrenic patients*," Zh Nevropatol. Psikhiatr., vol. 86, no. 1, 1986, pp. 106-8).

Since Immunologic Mechanisms was published, the hypothesis that viruses or other infectious agents may cause schizophrenia or bipolar disorder has been revived. The Stanley Laboratory of the Johns Hopkins University School of Medicine devotes itself, as its overall goal, to the development of a training and research program devoted to the "elucidation of the role of infection and immunity in the etiology of schizophrenia and biopolar disorders http://www.med.jhu.edu/stanleylab/labinfo.html). "The scientific goals of the Stanley Laboratory are based on the premise that human neuropsychiatric diseases arise from the interaction of infectious agents, the immune response, and the genetic susceptibility of an individual. This approach assumes that complex human diseases represent multiple interactions between environmental and genetic constituents…*Working Hypothesis*: Environmental factors include infection and the immune response occurring during the prenatal or postnatal periods or later in life; genetic factors may include the determinants of the response to infection and the regulation of cytokines and other immune mediators" (http://www.med.jhu.edu/stanleylab/research/intro.html). The theoretical bases for this approach is outlined in "*Viruses as Etiologic Agents of Schizophrenia*" (Advances in Biological Psychiatry, vol. 18, nos. 1-12, 1997; http://www.med.jhu.edu/stanleylab/etiologi.html).

(Pages 1, 13-14, London) "*Margaret Bauman's and Anthony Bailey's brain autopsies both show many brain changes that must occur before birth; Joseph Piven's MRI studies indicate a prenatal defect in brain development…"* The foregoing passages are a part of London's argument that autism is genetic—occurring before birth—not immunological, and is not caused or triggered by immunization. But it is difficult to come to London's conclusion—that MRI studies indicate a prenatal defect in brain development in autistic children which is responsible for their autism—on actually reading Piven's work. In the Discussion section of Piven's study, the following passages appear (p. 738):

"In our study, cortical malformations were not confined to any particular lobe and were detected at the same rate in both hemispheres. The failure of *these lesions* to coincide topographically suggests that it is unlikely that they have a direct role in the pathogenesis of autism, but, rather, that *in a subgroup of autistic individuals*, they *are linked to the underlying mechanism in the disorder*…Both extrinsic and instrinsic factors, including fetal anoxia, maternal cytomegalovirus infection…have been implicated in the pathogenesis of cerebral cortical malformations. *Rakic, in addition, has hypothesized that cell-cell interactions resulting from viral infection, cell-mediated immune reactions, or defects in the recognition of specific proteins* essential for cell movement *may be responsible for abnormal neuronal migration*. **Immunologic abnormalities have been demonstrated in autism**…*these studies suggest that the relationship between cortical malformations and prenatal immunologic, neurochemical, and genetic abnormalities in autistic subjects warrants further exploration*. Our findings raise several questions regarding the validity of MRI in detecting cerebral cortical malformations….."[37]

That structural defects of the brain are implicated in autism seems to be true only of a small segment of the autistic population (recall the normal MRIs in Wakefield's study). As regards brain studies via autopsy, few autistic children—particularly regressively autistic children—have been autopsied, to date—far too few to come to any firm

conclusions, as London does in "*ABCs*." Children with regressive autism almost always have normal MRIs, or MRIs that are normal except for the kind of demyelination seen in diseases like multiple sclerosis, which is autoimmune in nature. EEGs, however, often show abnormalities. DSM-IV says, describing features of Autistic Disorder (page 68), "*Imaging studies may be abnormal in some cases, but no specific pattern has been clearly identified [in autistic disorder]. EEG abnormalities are common…..*" In "*Autism and the Immune System*," Journal of Child Psychology and Psychiatry, vol. 38 no. 3, 1997, the authors conclude their study and review of the literature by suggesting *that the brain abnormalities* that have been found in autism may be "but one consequence of abnormal brain development, which in turn *may be linked with disordered immune function* in a yet unknown way" (page 346). They stress the importance of further research on the immunological abnormalities in autism, and suggest some specific avenues for study.

Bauman and Kemper's belief that cerebellar abnormalities in autism are the result of an in-utero process is subject to a number of criticisms. T. C. Binstock's research paper, "*Changing the autism paradigm: a critique of Kemper & Bauman's speculations regarding in-utero timing*" observes that B/K's conclusions are contrary to other recent findings in many crucial respects. Part of the authors' rationale is based on work that is extremely dated (e.g., 1908). Binstock's critique offers three particular conclusions regarding the work of Bauman and Kemper: 1) not all autistic persons have cerebellar features identical to those described by B/K--thus, B/K's findings can only be taken to represent a subset of autistics, rather than all cases of autism (there may even have been an induced selection bias affecting B/K's observations, due to narrow criteria for selection of subjects); 2) neither lack of gliosis nor lack of retrograde cell loss in the interior olivary nucleus necessarily implies in-utero timing of altered cerebellar morphology. Binstock's final conclusion is that 3) ***some* cases of autism are in-utero with regard to timing of causality--not *all***.[38]

(Page 1, London) "*…there has been little if any scientific evidence to substantiate an association between vaccination and autism*."

The terminology used in discussing causation issues—such as "scientific;" "evidence;" "cases"—should be closely defined within the context of any such discussion; otherwise statements such as London's, above, are apt to seriously mislead the public. London offers the above passages as proof that there is no causal relationship between the vaccination and autism, but his statements actually underline a critical problem: in spite of the fact that most vaccine reactions are never reported, there are in fact many case reports, research articles, and even books spanning thirty years or more, written by highly-esteemed scientists, which report the numerous deaths, illnesses, and disablements associated with vaccine viruses but, paradoxically, these are not considered by the government or by much of the scientific community to be evidential.[27] Surveying the literature, the matter of "evidence" of relationships between disease conditions and microbes must be evaluated very carefully to determine the reliability of information presented, and the objectivity of its presenters.

At least three working definitions of the term "evidence" can be discerned in literature. In Clinical Aspects of Hearing (*Springer Handbook of Auditory Research*, vol. 7, Springer-Verlag, 1996; chapter 6, "The Role of Viral Infection in the Development of Otopathology," pp. 157-8), chapter author Nigel Woolf discusses the difficulty of proving viral causation for various otopathologies. The conditions to be satisfied in order to attribute a viral etiology to an auditory or vestibular pathology may include *Koch's postulates*, "as modified for infectious agents." These requirements include identification (isolation) of the virus from the location in question; a close association of the virus with a specific clinical syndrome; the ability to transfer the viral infection to an experimental animal model; and the capacity to recreate a homologous disease in the animal model following viral transfer. The latter are often impossible, as human viruses frequently will not transfer to experimental animal models (for many viruses, the different viral isotypes have been shown to be species-specific both in vivo and in vitro). However, the authors of Psychoneuroimmunology (Academic Press, 1991) point out that, while it is difficult to prove that a virus causes an illness, it is also "difficult to rule *out* viral causality because 'absence of proof is not absence of absence (pp.750-1).'" Working with bacteria, G. J. Domingue and H. B. Woody of Tulane University Medical School state, "A considerable body of experimental and clinical evidence supports the concept that difficult-to-culture and dormant bacteria are involved in latency of infection and that these persistent bacteria may be pathogenic…A series of cases illustrating idiopathic conditions in which cryptic bacteria have been implicated in the expression of disease is presented." The authors suggest that Koch's postulates, one mechanism for determining causation, may have to be

redefined in terms of molecular data when dormant and nonculturable organisms involved in latent or persistent infection are implicated as causative agents of "mysterious" diseases.[28]

There is little reliable "scientific" evidence in terms of formal, controlled studies chiefly because thorough, *independent* (non-industry or government-connected) studies examining the long-term influence of vaccinations on children have rarely been funded or conducted--particularly in the United States. Similarly, research has rarely been conducted in examination of the neurologically impaired, to ascertain the impact of vaccination. A recent post to an electronic mail newsgroup describes the problem in a nutshell:

The central defect in the numerous, recent mandatory vaccination requirements is that the same people who are profiting from these vaccines are also in control of the research on[,] and publication of[,] the dangers of the vaccines. The fox is pretending to guard the henhouse. ...foreign research has found problems with the Hepatitis B vaccine for children. Note that I said "foreign," because there is no independent *US medical research on this.*[29]

Incredibly, even industry safety studies on the MMR vaccine have not extended beyond three weeks following vaccination.

Oddly, viruses like rubella have long been considered causal for autism if encountered congenitally, or if encountered naturally in the environment after birth, but not if acquired through vaccination--even though scientific literature confirms that attenuated viruses CAN cause disease. Using case reports, research articles, and uncontrolled studies as platforms, medical scientists like Montinari, et al. (University of Bari, Italy, "*Role of Immunogenetics...*"), Wakefield, et al., and many others have, and continue to, urge further study on the impact of vaccines on a variety of medical conditions. Few have emerged, and reporting in the few published accounts of vaccine injury and death is frequently hindered by several factors. A study concerning bias, originating at the Institute of Vaccinology and Virology, Hamburg, Germany, was published in Acta Paediatrica Japonica in August 1991, volume 33, number 4, pages 421-7, titled, "*Bias in evaluating CNS complications following pertussis immunization*." An abstract of this paper briefly states that, in the evaluation of central nervous system complications following pertussis immunization, bias contributed to all of the following:

1. the infrequency of notifications of postimmunization adverse events

The author notes that the reporting and claims processes in Germany are simpler than in the United States, resulting in far more reported instances of adverse reactions in Germany than in the USA.

2. variable accuracy of publications by vaccine producers on the frequency of adverse reactions

Vaccine manufacturers in Germany do not hesitate, says the author, to publish statistics and narratives concerning complications from vaccination. German manufacturers reported far more reactions and deaths than comparable firms in the United Kingdom.

3. the lack of reporting or skewed reporting of comparison[s] of permanent brain damage after DPT AND DT immunization

Reactions to the DPT vaccine were significantly more numerous than those to the DT vaccine, pointing to problems with tolerance of the pertussis component in the DPT, but the evaluators of these cases put this down to bias, even though their case findings did not support this conclusion. When cross-examined in a court of law later, one of the evaluators, a former member of the Wellcome [pharmaceutical] Foundation, revealed that he had sent a draft of this evaluation to Wellcome [presumably for approval of its content]. Similarly, a judge dismissed many cases of "Reye-like syndromes" following DPT immunization, though they were well-founded.

4. the prevalence of pro-immunization sentiments

The author alludes to a "conspiracy of silence" among doctors reluctant to report serious events for which they were partially responsible; others may be so caught up in the prevention movement that they fail to realize that adverse events really occur. Still others may not report lest the reports fuel anti-vaccination debate. At the Fifth International Congress of Infectious Diseases, circa 1980, the author states, a clinic head from West Germany claimed never to have seen a neurological complication following pertussis immunization; later, after his retirement, his clinical staff recovered records of complications and stated that cases had usually been ignored with a "what *must* not be therefore *cannot* be" attitude (p. 424).

5. the background rate of convulsions and pertussis vaccine-associated seizures

The incidence of convulsions following vaccination are weighed against a calculated rate of "chance occurrences" of convulsions. The differences in the number of convulsions or serious disabilities following DPT as compared to DT vaccination in the Federal Republic of Germany were significant, though the judge in a key vaccine trial refused to acknowledge this, and ruled accordingly.

6. the reporting of, and the number of cases of immunization associated viral encephalitis

DPT vaccination may provoke encephalitis indirectly via a previously-existing herpes or coxsackie virus infection, either through over-stimulation or suppression of immune system components, or directly through a hyperacute allergic reaction; this connection, however, has been missed by many practitioners and insufficiently considered by others.

7. the inaccuracy of vaccine-injury statistics

"Normally one expects scientific statistics to be reliable," this paragraph begins. The most relied-upon expert in a particular trial presented statistical evidence to a judge which was skewed by incorrect grouping of data, and assessment of 50 patients as "normal" who had abnormal EEGs. This scientist claimed to have taken care to ensure…overestimation rather than under estimation of the risks of pertussis vaccine.

8. misdesign of the NCES guidelines for notification of adverse events following vaccination

Reporting of severe vaccine-related complications is hampered by an arbitrary temporal limit on seizure activity: if post-vaccination seizures, or seizures followed by coma, paralysis or other neurological signs occur within the first 3 days and last two hours or more, they are considered complications of vaccination. Seizures lasting less than two hours were discounted (many cases handled by the author lasted less than half an hour.

9. practitioners' personal opinions and concerns regarding vaccination and the arbitration process

The author notes that of 85 cases claiming postimmunization pertussis damage, he had dismissed 22 cases out of concern that he might be biased toward the plaintiffs. The authorities dismissed another 20 cases. That total of dismissals was equivalent to the number of cases the author felt were wrongly dismissed by others in this study.

The abstract of "*Bias in evaluating…*" tersely concludes, "A review of these points indicates an underestimation of CNS complications after pertussis immunization."

Dr. Harris Coulter, President of the Center for Empirical Medicine, Washington D. C., testified before the House of Representatives, United States Congress, in April 1997 saying, "The fact that the federal medical establishment - which would be the major source of funds for such an epidemiologic investigation - is itself highly committed to the childhood vaccination program, goes far to explain the absence of any official interest in this connection. This is a major disadvantage of all research on damage from the childhood vaccination program" ("*Childhood Vaccinations and Juvenile-Onset (Type-1) Diabetes*," April 16, 1997, page 2; text available at http://www.909shot.com/hcdiabetes.htm). In the United States, the federal government and the pharmaceutical industry have been united for years in the task of developing and mandating immunizations (witness

the Childhood Vaccine Industry Act). The combined influence of this government/drug industry alliance has thus far curtailed most efforts to probe the matter more fully. Put simply, the modus operandi of both drug industry and government, regarding vaccines, has been '*no studies, no evidence*.' Such conflicts of interest in medicine and scientific research heavily contributes to the "no evidence" problem, as regards vaccine-related injury and illness. It is a serious and much-addressed concern within the medical community regarding many other concerns as well (for further discussion, see **"Caveats"**).

Strongly affecting the lack of "evidence" is the fact that funding for independent immunological studies of autism, particularly those targeting the viral aspects of autism, is rarely forthcoming. Independent researchers in this area are aggressively discredited, combated through the use of flawed, concocted 'studies' such as the Finnish work mentioned above—or attention is directed away from studies and literature on the subject. Simultaneously massive "PR" campaigns for vaccines—more and more vaccines—accelerate, together with efforts to make immunization mandatory in all cases, and track via computer networks the medical records of children. (The mechanism of such dis-information campaigns, with their intermingling of government and special interests, can easily be understood in the context of tobacco industry events, or in the carefully-maneuvered relaxing of environmental restrictions to include the use of toxic "biosolids" in food production.[30]) In view of all the circumstances, statements such as "there is no scientific evidence" should not be taken to mean that there is no reason for considerable caution or change in methodology.

(Page 14, London) "**Vaccinations Save Lives**" (section subtitle) "…*Before we had immunization, many, many thousands of children died of now-preventable disease. Others suffered permanent injury and/or brain damage…*

"Vaccines save lives" is a frequently-heard claim which makes no reference to the number of lives lost or drastically changed by vaccines, and offers no concrete evidence. Proof of this statement is largely by prognostication--prediction of 'what would have happened if---.' Interestingly, the Pharmaceutical Advertising Advisory Board, Toronto, Canada, recently ordered Merck-Frosst Canada to withdraw advertising that claims that its new chickenpox vaccine saves lives. Said the Board, "There's no proof the claim made by Merck-Frosst Canada about the drug, Varivax, is true" (*CBC News Canada* news release, web-posted Friday, April 2, 1999; posted to the Australian Vaccination Network e-mail discussion list May 4, 1999, 09:41:51 +1000).

The import of numberless--and even 'numbered'--"statistics" such as London's passage above is markedly reduced by lack of specificity. Unanswered are questions arising, for example, from the above passage: "Over what time period did 'many, many thousands of children' die (three hundred years, or thirty)? Are these thousands drawn from worldwide scrutiny, or from a limited geographic area? What disease or diseases are being discussed, and what were the hygienic (and other) circumstances in the countries where this occurred? The dramatic quality of this paragraph and its lack of specifics are more akin to propaganda than to scientific reporting.

(Page 15 and 16, London) The myth of disease eradication is perpetuated in London's piece: "*a…known cause of autism…[rubella] has been virtually wiped out…if new parents faithfully vaccinate their young children against measles, measles…could go the way of smallpox and, soon, of polio. Why do so many people fear vaccines when they are one of the greatest success stories in medical history?*"

That rubella, smallpox, and even polio have been wiped out is a widely-cultivated myth. Global measles eradication has so far failed. Outbreaks of rubella, measles, and polio continue to occur because of the frequent failure of vaccines to establish and maintain complete immunity--in part due to viral mutation (material on this theme is explored below, in *'Vaccines are very effective…*'). Often, outbreaks of these diseases occur but are renamed by health officials under pressure to achieve disease-free status. In addition, when impeded by vaccination, natural viruses can mutate so far from their original form as to become recognized as different diseases. Vaccine viruses can cause the diseases they are created to prevent, possibly in a modified form, as earlier discussed; polio is the most flagrant offender in this regard (see ***The attenuated virus, infectious or not?*** above). Also, vaccine viruses can establish persistent infections and diseases seemingly unrelated to the original. Viral contaminants in vaccines, acquired through culturing in animal tissues, can cause diseases also unrelated to the diseases the vaccines are formulated to prevent--the most fearsome of which are AIDS and Ebola. At best, vaccination appears to be a substitution process in the many who are immunologically susceptible, involving the exchange of one disease for

another—frequently the substitution of a chronic, lifelong (perhaps eventually life-threatening) autoimmune disease for an acute but transitory viral infection.

smallpox

"*Is smallpox history?*" (The Lancet, vol. 353, no. 9164, May 8, 1999): "A pilgrim returned home to Yugoslavia from Mecca in February, 1972, with a fever… In the 4 weeks since the pilgrim first had his fever, 150 people were infected across the country. It took 4 weeks before doctors, nurses, and health authorities knew they were dealing with smallpox… 175 people contracted smallpox [thereafter] and 35 died… these events occurred in a well-vaccinated population."

"*Poxvirus dilemmas -- monkeypox, smallpox, and biologic terrorism*" (New England Journal of Medicine, vol. 339, no. 8, August 20, 1998): "More than 20 years have passed since the last case of smallpox was confirmed… Now, new dilemmas confront the world. Could recent outbreaks of human monkeypox in the Democratic Republic of the Congo [Zaire] represent the return of another form of smallpox?… The first case of human monkeypox was identified in 1970… The clinical picture of monkeypox resembles that of smallpox in Central Africa."

"*Is an old virus up to new tricks?*" (Science, vol. 277, July 18, 1997, pp. 312-3): "…an exotic infection …is alarming some public health experts: the largest outbreak ever seen in humans of a well-known virus called monkeypox. A first cousin of the once-dreaded smallpox, monkeypox causes nearly identical symptoms… '…for practical purposes, smallpox is back,' says virologist Peter Jahrling of the U.S. Army Medical Research Institute of Infectious Disease in Fort Detrick, Maryland… 'This could be worse than smallpox if it adapts to humans,' acknowledges virologist Bernard Moss of the National Institute of Allergy and Infectious Diseases (NIAID)…" Researchers were reluctant to recommend a new vaccination program--which would use smallpox vaccine--for the local population, because the vaccine can cause disease and death in persons with inadequate immune systems.

"*Lethal animal pox virus infection in an atopic patient simulating variola vera* [smallpox]" (Hautarzt., vol. 42, no. 5, May 1991, pp. 293-7): An 18-year-old patient acquired a cowpox-like virus infection clinically similar to smallpox from a domestic cat as carrier. In spite of intensive care, with…the last available vaccinia hyperimmunoglobulin, the patient died of pulmonary embolism…"

"*Variola* [smallpox] *or a severe case of varicella? A case of human variola due to monkeypox virus in a child from the Cameroon*" (Ann. Soc. Belg. Med. Trop., vol. 71, no. 2, June 1991, pp. 123-8): Human monkeypox was suspected on clinical grounds in a seven year old child in Cameroon. (A selected Medline [National Library of Medicine] "MESH" subject heading is "Smallpox--diagnosis.")

"*Human monkey pox: its clinico-epidemiological characteristics*" (Zh. Mikrobiol. Epidemiol. Immunobiol. vol. 6, June 1988, pp. 23-30): During the course of the smallpox eradication programme, a new eruptive disease clinically resembling smallpox was discovered in Zaire…the virus can be transmitted from man to man."

polio

(see especially the final case report in this section)

"*Health alert as virus paralyses six children*" (The Australian, June 29, 1999): A "rare" gastrointestinal virus referred to as enterovirus 71 paralyzed six young children in west Australia. Of these children, aged six to eighteen months, three are expected to be permanently disabled; for the others the outcome is uncertain. 14 cases of meningitis, also caused by enterovirus 71, were seen in addition. The virus is spread by fecal-oral transmission or by respiratory droplets, and can be contracted by parents changing their children's diapers. Steroid treatment appeared to reduce the extent of paralysis.

"*Global eradication of poliomyelitis and co-emergence of novel poliovirus strains*" (Epidemiology, vol. 7, no. 6, November 1996, p. 653 [Letters to the Editor]): Recent experience has vindicated an old theory that polioviruses,

harmless around 1900, mutated to strains of enhanced virulence. During the recent extensive usage of live polio-vaccines in endemic areas of Asia and Latin America, mass immunizations resulted in "phenotypic mixing or genetic reassortment" of different poliovirus strains. "Similar eventualities would be common in currently polio-free countries offering live poliovaccines. Such vaccines would also enhance the selection of virus mutants with increased virulence. National immunization days in endemic areas and live vaccination in disease-free areas could thus stimulate the emergence and selection for virulent strains by the end of this century."

"*Outbreak of pokomyelitis-like paralysis associated with enterovirus 71*" (Pediatric Infectious Diseases, vol. 8, no. 9, September 1989, pp. 611-6): In summer of 1987 five Philadelphian children experienced acute onset of flaccid paralysis of arms or legs. There were documented exposures to oral poliovirus vaccine and coxsackievirus B3 in some cases. Two children had residual paralysis with weakness and muscle wasting.

"*Enterovirus type 71 infections: a varied clinical pattern sometimes mimicking paralytic poliomyelitis*" (Review of Infectious Diseases, vol. 6, Supplement 2, May-June 1984, pp. S387-90): From 1969-1973 California suffered outbreaks of enterovirus 71 with meningitis/encephalitis emphasis; in 1975 Bulgaria suffered more than 705 cases, with 149 developing paralysis, and 44 deaths. Hungary experienced an epidemic in 1978. Infants and young children of polio vaccination age were the chief victims.

"*Epidemiological, clinical, and pathomorphological characteristics of epidemic poliomyelitis-like disease caused by enterovirus 71*" (Journal of Hygiene, Epidemiology, Microbiology, and Immunology, vol. 23, no. 3, 1979, pp. 284-95): In May through September 1975 an outbreak of epidemic disease "clinically and pathomorphologically simulating nearly all known forms of poliomyelitis" occurred predominantly among young infants in Bulgaria. Though the title of this piece lists enterovirus 71 as the culprit, the abstract describes the virus in these cases as "antigenically *related to*enterovirus 71." "The similarity to poliomyelitis and precariously rapid increase in the incidence led to the decision to urgently vaccinate the entire population with Sabin's live poliovirus vaccine." Soon afterward, the number of new cases began to decrese.The authors note that similar enterovirus epidemics were also suffered by Sweden in 1973 and Australia from 1972-1973, and Japan, from 1972-3.

haemophilus influenza type b

"*The emergence of Haemophilus influenzae types e and f as significant pathogens*" (Clinical Infectious Disease, vol. 21 no. 5, November 1995, pp. 1322-4): Before vaccination for Haemophilus influenzae type b, the other encapsulated serotypes of H. influenzae rarely caused invasive disease. [Now] the clinical features of non-type b H. influenzae meningitis are the same as those of type b. "We report these four cases to document an *in*crease in infection due to *non*-type b serotypes of H. influenzae, and we postulate that this change may result from the well-documented decrease in H. influenzae type b...that has occurred because of universal vaccination for H. influenzae type b."

"*Meningitis due to Haemophilus influenzae type f*" (Journal of Paediatrics and Child Health, vol. 34, no. 1, February 1998, pp. 95-6): "Despite the great success of Hib vaccines in reducing H. influenzae, cases of H. influenzae meningitis continue to occur, caused by less common encapsulated serotypes...infection due to non-b serotypes requires close monitoring."

"*Invasive disease due to Haemophilus influenzae serotype f: clinical and epidemiologic characteristics in the H. influenzae serotype b vaccine era*" (Clinical Infectious Disease, vol. 22, no. 6, June 1996, pp. 1069-76): "With the decline in the rate of infections caused by Haemophilus influenzae serotype b, H. influenzae serotype f (Hif) is becoming a relatively important cause of invasive disease. .the proportion of all invasive H. influenzae disease caused by Hif rose from 1% in 1989 to 17% in 1994…Continued surveillance is warranted to evaluate the trend toward the increasing incidence of Hif disease that was noted in this study."

measles-mumps-rubella

"*Epidemiology of encephalitis in children. A prospective multicentre study*," European Journal of Pediatrics, vol. 156, number 7, July 1997, pp. 541-5: Investigators found that, while mumps, measles, and rubella virus associated

encephalitis had been almost eliminated due to vaccination programs, encephalitis from other viruses had increased in frequency and occurred in younger age groups. "**Conclusions**: The spectrum of encephalitis in children has changed due to vaccination programs. The incidence, however, appears to be about the same due to increasing frequency of other associated old and new microbes"—i.e., the number of MMR-encephalitis cases eliminated have been replaced by an equal number of encephalitis cases from other microbes, previously not seen.

(London) That '*vaccines are very effective and almost always safe*' is a pervasive theme in London's "*ABCs*" piece.

The efficacy of common vaccines may be greatly exaggerated. In a 1998 study, it was stated that "investigator bias probably has overestimated the efficacy of most vaccines." Clinicians' compliance levels in monitoring illness in vaccine recipients varied widely in trial protocols. "Less compliant investigators were far more likely to report data making vaccines appear more effective against mild or moderate disease. Our data suggest that observer compliance (observer bias) can significantly inflate calculated vaccine efficacy…it is likely that all recently completed efficacy trials have been affected by this type of observer bias and all vaccines have considerably less efficacy against mild disease than published data suggest" (Pediatrics, vol. 102, no. 4, part 1, October 1998, pp. 909-912, reported as news release, "*Clinical Trials; Vaccine Efficacy Overestimated…*," posted December 2 to Vaccine Information and Awareness electronic mail discussion list[via@access1.net], 9:42 a.m.).

In reality, outbreaks of vaccine-preventable diseases occur in spite of vaccination. This is in part due to the fact that the immunity afforded by vaccines is typically not permanent, unlike that gained through disease, but may be due to other factors as well, such as poor bioengineering; unfavorable storage conditions; improper use, viral mutation, and the ability of the vaccine viruses themselves to cause infection in recipients or close contacts. The safety of vaccinations is challenged by case reports and studies such as the above, and by numerous research articles and books written by eminent scientists and medical researchers, as well as the common belief that most vaccine reactions are not reported. In "*Vaccination against whooping-cough. Efficacy versus risks*" The Lancet, vol. 1, January 29, 1977, pp. 234-7, for example, the authors conclude: "It seems likely that most adverse reactions are unreported and that many are overlooked…existing provisions, national and international, for epidemiological surveillance and evaluation are inadequate." Author H. U. Albonico expresses both doubt and concern about routine vaccination in "*Arguments against routine mumps vaccination*" (Soz Praventivmed, vol. 40, no. 2, pp. 116-23), "An unnecessary vaccination proves to be of insufficient efficacy, yet [is] associated with an unexpected high complication rate and bears thus the risk of discrediting also other immunizations in the general public… The [MMR mass-immunization] project remains epidemiologically highly vulnerable and thus hazardous…there are concerns about the longterm effects of manipulations of the natural balance between man and microorganisms." In "*The Immunization Campaign Against Measles, Mumps and Rubella…Medical Objections to a Continued MMR Immunization Campaign in Switzerland*" (Journal of Anthroposophic Medicine, spring 1992; text available at http://www.trufax.org/vaccine/mmr4.html): After three years of study, a medical working group representing 180 Swiss medical doctors specializing in general medicine, internal medicine and pediatrics pointed out that not only will a high vaccination rate in the U.S. fail to stop measles outbreaks, deaths, and complications completely, but that "*mandatory, mass vaccination with MMR vaccine is ineffective and dangerous*." Further, the group stated that natural recovery from childhood diseases such as measles plays a role in the maturation of the human immune system and helps the individual develop resistance to disease, including chronic diseases such as asthma and cancer." Continued mass vaccination of infants could destroy the natural resistence of populations to childhood diseases and create virgin populations at risk for future epidemics.

Efficacy is challenged additionally by the plentitude of reports on vaccine failure, exemplified below. Deaths, injuries, and vaccine-related disease in other countries, as well as in the United States, plus existing information on prevailing disease trends, the mechanics of viral activity and of the human immune system, need to be considered carefully (and less selectively) in evaluating the safety and efficacy of vaccines. [Readers should also see the section titled "*Wakefield's recommendations*," above.]

"*Reemergence of invasive haemophilus influenzae type b disease in a well-vaccinated population on remote Alaska*" (Journal of Infectious Diseases, vol. 179, no. 1, January 1999, pp. 101-106, reported via Vaccine Weekly, NewsEdge Corporation news release, February 12, 1999): In 1996, after administration of Hib conjugate vaccine (DTP whole-cell vaccine + Hib), cases of invasive Hib disease, as well as "silent" Hib infections, increased.

"*The incidence of rubella virus infections in Switzerland after the introduction of the MMR mass vaccination programme*" (European Journal of Epidemiology, vol. 11, no. 3, June 1995, pp. 305-10): In evaluating the impact of the MMR mass vaccination program begun in Switzerland in 1985, "we conclude that MMR mass vaccination has not interrupted the circulation of rubella virus in Switzerland, and that improvements in the implementation and surveillance of the MMR vaccination campaign are necessary in order to avoid [the] untoward effects of it."

"*Temporal trends in the population structure of bordetella pertussis during 1949-1996 in a highly vaccinated population* (Journal of Infectious Diseases, vol. 179, April 1999; 915-923. "Despite the introduction of large-scale pertussis vaccination in 1953 and high vaccination coverage, pertussis is still an endemic disease in The Netherlands, with epidemic outbreaks occurring every 3-5 years." One factor that might contribute to this is the ability of pertussis strains to adapt to vaccine-induced immunity, causing new strains of pertussis to re-emerge in this well-vaccinated population.

"*High incidence of breakthrough varicella observed in healthy Japanese children immunized with live attenuated varicella vaccine (Oka strain),*" Acta Paediatrica Japonica, vol. 39, no. 6, December 1997, pp. 663-8: the rate of varicella [chicken pox] occurrence among vaccinees was found to be much higher than rates reported previously by other authors. "Varicella vaccine seems to be effective in modifying the symptoms of varicella, but not potent enough in protecting from VZV infection."

"*The characteristics of poliovirus strains circulating in Ukraine in 1982-1994*" (Mikrobiol[ogie] Z. vol. 60, no. 2, March-April 1998, pp. 44-49 [article in Russian]): "The long-term use of the live poliomyelitis vaccine has not stopped circulation of virulent polioviruses."

"*Effect of subclinical infection on maintaining immunity against measles in vaccinated children in West Africa*" (The Lancet, vol. 353, January 9, 1999, pp. 98-102): Subclinical measles occurred in 45 percent of vaccinated children exposed to natural measles. "new epidemics, albeit milder in form, may occur in vaccinated areas[, a fact] which should be recognised in campaigns to eradicate measles." [Note: if sustained as chronic infections, subclinical measles infections can result in numerous other diseases.]

"*Is smallpox history?*" (The Lancet, vol. 353, no. 9164, May 8, 1999): "A pilgrim returned home to Yugoslavia from Mecca in February, 1972, with a fever… In the 4 weeks since the pilgrim first had his fever, 150 people were infected across the country. It took 4 weeks before doctors, nurses, and health authorities knew they were dealing with smallpox… 175 people contracted smallpox [thereafter] and 35 died… these events occurred in a well-vaccinated population."

"*Five cases of measles secondary vaccine failure with confirmed seroconversion after live measles vaccination*" (Scandinavian Journal of Infectious Disease vol. 29, no. 2, 1997, pp. 187-90): Two, five, seven and twelve years after vaccination with further attenuated live measles vaccine, three of five patients experienced modified measles infection, and the remaining two had typical measles. "This may be the first SVF case report that confirms the existence of completely waning immunity in recipients of the further attenuated live measles vaccines."

"*Clinical and epidemiological findings during a measles outbreak occurring in a population with a high vaccination coverage*" (Rev. Soc. Bras. Med. Trop., vol. 28, no. 4, Oct-Dec 1995, pp. 339-43): "The history of previous vaccination [in very early childhood] did not diminish the number of complications of the cases studied. The results of this work show changes in age distribution of measles leading to sizeable outbreaks among teenagers and young adults."

"*Measles serodiagnosis during an outbreak in a vaccinated community*" (Clin. Invest. Med., vol. 11, no. 4, August 1988, pp. 304-9): from a group of 30 measles-sufferers displaying IgM antibodies during the acute phase of illness, 17 had been vaccinated for measles. All 17 experienced measles again, showing IgM antibodies indicating acute infection. "A history of prior vaccination is not always associated with immunity nor with the presence of specific antibodies."

"*H[epatitis] B V[irus] prevalence is unchanged by hepatitis B [vaccine]*," report by Michael Belkin, statistician, based on nationwide sampling of the prevalence of hepatitis B by the Centers for Disease Control, 1988, to 1994, for comparison with figures from 1976 to 1980 [American Journal of Public Health, vol. 89, no. 14, 1999]: "There was an age-adjusted prevalence of 5.5% in the first study and 4.9% in the second; these differences are not statistically significant. The authors concluded that the widespread use of HBV vaccine in the 1980s has not had a major impact on the overall prevalence of this infection (communication posted on the Vaccine Information and Awareness (VIA) listserv [via@access1.net], March 11, 1999, 12:22 p.m.).

(London, p. 14) Addressing the severity of vaccine-preventable diseases, London asserts, "*These diseases from which we can now protect our children are no small thing…*all *of these illnesses are killers*."

In the main, diseases like varicella zoster (chicken pox), measles, pertussis, rotavirus diarrhea, etc., for which vaccines exist, are almost always temporary and/or mild in their duration and effects. Interestingly, a British law firm investigating vaccine deaths and injuries notes a change in the way measles, mumps and rubella are regarded in medical reference texts, before and after vaccines for these became available--i.e., the diseases are first described as mild and transient, with low incidence of complications, before MMR immunization was available, then as potentially serious, with considerable risk of complications, *after* the vaccine came into existence.[31] In numerous other texts, the often beneficial—even protective—effects of natural infection have been noted.

"*The epidemiology of pertussis in the Republic of Ireland*" (Communicable Disease Report[:] CDR Review, vol. 2, no. 3, February 28, 1992, pp. R31-3): Following adverse publicity in 1973, uptake of the vaccine fell to 30% in 1976. In recent years, it has leveled out at only 40-45%. Yet when large epidemics of pertussis occurred in 1985 and 1989, mortality from pertussis fell to almost negligible levels.

"*Severity of whooping cough in England before and after the decline in pertussis immunisation*" (Archives of Disease in Childhood, vol. 59, no. 2, February 1984, pp. 162-5): "Since the decline of pertussis immunisation, hospital admission and death rates from whooping cough have fallen unexpectedly… The severity of attacks and the complication rates in children [who *were*] admitted to hospital were virtually unchanged."

O. Tonz and S. Bajc, "*Convulsions after whooping-cough vaccination*" (Schweiz. Med. Wochenschr., vol. 110, no. 51, December 20, 1980, pp. 1965-71): Convulsions or status epilepticus in 11 infants after pertussis vaccination are reported. In three of 11 cases, grand mal epilepsy persisted and two children developed infantile epileptic encephalopathy (Lennox Syndrome). "The following conclusions are drawn from these observations: 1) In view of the usually benign course of whooping cough today, current vaccination is hardly satisfactory. Improvement of the available vaccines is an urgent necessity… 2) Parents should be better informed about the risks involved in pertussis vaccination. 3) Booster inoculations should be abandoned. 4) Health authorities should decide whether the current pertussis vaccination program should be abandoned. 5) Complications following vaccination should be registered….."

"*Whooping cough and pertussis vaccine: a comparison of risks and benefits in Britain during the period 1968-83*" (Development of Biological Standards, vol. 61, 1985, pp. 395-405): "Since 1975, acceptance of pertussis vaccine has fallen from over 70% to 50% or less in most parts of Britain. This permits evaluation of a continuing natural experiment in which the frequency and severity of whooping cough can be compared [with] those of adverse events following injections of pertussis vaccine… There is a significant correlation between vaccine-acceptance and hospital admission by district of residence… It is concluded that, in children living in non-deprived circumstances in Britain, the risk of pertussis vaccine during the period 1970-83 exceeded those of whooping cough. In some deprived sectors, the risks from whooping cough might have been marginally higher but there was no evidence that this was associated with any increase in deaths or permanent disabilities."

"*Vaccination against whooping-cough. Efficacy versus risks*" (The Lancet, vol. 1, January 29, 1977, pp. 234-7): Calculations based on the mortality of whooping-cough before 1957 predict accurately the subsequent decline and the present low mortality… Incidence [is] unaffected either by small-scale vaccination beginning about 1948 or by nationwide vaccination beginning in 1957… No protection is demonstrable in infants."

advantages of natural infection

"*Arguments Against routine mumps vaccination*" (Soz Praventivmed, vol. 40, no. 2, 1995, pp. 116-23): "As the maturation of the immune system follows learning principles, the question arises whether childhood diseases are not essential for the normal development of immuno-competence….."

"*The Immunization Campaign Against Measles, Mumps and Rubella…Medical Objections to a Continued MMR Immunization Campaign in Switzerland*" (Journal of Anthroposophic Medicine, spring 1992; text available at http://www.trufax.org/vaccine/mmr4.html): After three years of study, a medical working group representing 180 Swiss medical doctors specializing in general medicine, internal medicine and pediatrics stated that "natural recovery from childhood diseases such as measles plays a role in the maturation of the human immune system and helps the individual develop resistance to disease, including chronic diseases such as asthma and cancer." Continued mass vaccination of infants could destroy the natural resistence of populations to childhood diseases and create virgin populations at risk for future epidemics.

"*A case of myasthenia gravis with transient remission after influenza A virus infection*" (No To Hattatsu, vol. 31, no. 1, January 1999, pp. 70-4): researchers from the Department of Pediatrics, Nagaoka Central Hospital, Niigata, Japan found that a boy's intractable myasthenia gravis symptoms disappeared rapidly after an influenza A virus infection. "These results suggested that influenza A may improve the clinical signs of myasthenia gravis, as is the improved case with measles."

M. Lovik, "*Do infections reduce the development of allergy? Do measles reduce the risk of allergic disease?*" (Tidsskr Nor Laegeforen, vol. 117, no. 5, February 20, 1997, pp. 688-90): "Immunological theory indicates that [natural] infections can prevent allergy by directing the immune reaction against an antigen towards a Th1-type response, thus inhibiting an allergy-associated Th2-type response." A recent study from Guinea-Bissau demonstrates an apparently strong protective effect of [natural] measles infection on incidence of positive skin tests for reactions to common allergens. The author asks these medical and ethical questions: "do we have to choose between measles [infection] and an increase in allergic ciseases? And if so, who should decide--the individual or society?"

"*Relation between psoriasis and measles*" (Z. Hautkr., vol. 57, no. 6, March 15, 1982, pp. 439-40 [article in German]): Psoriatics with measles in the history had a light type of psoriasis and low titers of measles antibodies; psoriatics without measles in the history, however, had severe types of psoriasis, and the titers of measles antibodies were high."

(Page 18) "**The Autoimmune Question**: Although no one has found evidence that autism can be caused by an autoimmune disorder, researchers have not ruled it out…Could the MMR vaccine so overheat an already disordered immune system that it attacks…the brain? So far, given the **vast** amount of data…the answer is no" [emphasis mine].

Unfortunately, the "vast amount of data" is unspecified in the London piece, making it difficult to assess this claim. In medical literature there is compelling evidence, irregardless, of the involvement of autoimmunity in autism—possibly as a causal factor (see ***The attenuated virus: infectious or not?*** above, as well as passages discussing the work of Van Gent, et al., in ***Causes***).

(Pages 16/15, London) "*…the entire point of vaccination programs is to end the need for vaccinations!* "

"*The moral: we vaccinate children against disease in order to keep them alive and well*."

Few would argue that the immunization practices first arose out of concern for widespread suffering and death from disease. Once vaccines became a commodity, however, focus inevitably shifted to include other matters. The precedence of profit and reputation over health was amply enough demonstrated when, as a Swine influenza pandemic supposedly loomed in the late 1970s, U.S. government personnel initially resisted industry pressures for

federal government protection against liability for the pending Swine Flu vaccine. Political concerns surfaced during this period as key bureaucrats and scientists strove to appear concerned, decisive, swift, and justified in their actions:

"A public official might not only feel an obligation to protect the health of the people but might also suspect that a wrong decision on such an important issue might cost him his job. If he failed to act and an influenza pandemic did appear, might he not be indicted for negligence or stupidity? Far better to act positively, and run the lesser risk that if a pandemic failed to come, he could only be accused of wasting taxpayers' money…the influenza virologist, normally confined to his narrow circle of fellow specialists…must have felt a secret thrill of anticipation at seeing his subject in the forefront...the specialist in preventive medicine and public health would have been less than human not to feel a certain elation at the prospect of showing, in so significant a fashion, what disease control and epidemiology were capable of doing…(p. 33, Pure Politics, cited below)."

The different pressures converging, industry and government pushed through a national immunization program allotting financial protection to the pharmaceutical industry, and legislative backing to government scientists and bureaucrats. Though one school of thought had held that the hastily-concocted Swine Flu vaccine should not automatically be given, but should be stockpiled in case of a serious flu epidemic, the winning side pushed for immediate, country-wide vaccination. The result has been infamous ever since, as not only did the pandemic not appear, but many vaccinees contracted Guillain-Barre Syndrome, a body-wide autoimmune paralysis. Some of the vaccinees died from Guillain-Barre, and some from the vaccine itself (Arthur M. Silverstein, Pure Politics and Impure Science: The Swine Flu Affair, Johns Hopkins University Press, 1981).

In 1986 the federal government responded to further pressure from the pharmaceutical industry, which threatened to cease production of vaccines if it was not granted federal protection against liability suits. Responsibility for vaccine-related deaths and injuries was therewith globally assumed by the U.S. government through the Childhood Vaccine Injury Act. The pharmaceutical industry had already demonstrated a preoccupation with its financial well-being in the Swine Flu affair of the 1970s; through the 1986 Act, the government created for itself a flagrant conflict of interest wherein public monies are used to refute citizens' claims of damage by government-mandated vaccines.

According to investigative journalist Janine Roberts, [ethical] questions are now being raised about an urgent warning issued in the United Kingdom in November 1994, forecasting an epidemic which would infect between 100 and 200,000 children, of which some 50 would die--if children ages 5 to 15 were not immediately revaccinated for measles, mumps and rubella ("*The Fraudulent Measles Epidemic;*" posted as part of The Web Inquirer, September 29, 1995, http://www.gn.apc.org/inquirer/fraud.html; posted April 8, 1999, 12:13 a.m. on Vaccine Information and Awareness discussion list, via@access1.net). Roberts quotes Dr. Richard Nicholson, editor of the Bulletin of Medical Ethics, who studied government reports and declared that there was no proof that such an epidemic was about to begin--and even if this had happened, there had been no justification for the concomitant rubella immunization proposed. Faulty statistics were used in compiling the government's reasons for sudden mass immunization; further impropriety--and motivation--was discovered when the British government did not collect bids from a variety of vaccine vendors, as required by law. Instead, claiming extreme urgency, the government immediately contracted with SmithKline Beecham and Merieux UK, pharmaceutical firms which had earlier been forced to withdraw their MMR vaccines when the mumps element of the vaccine was shown to cause meningitis. This new contract reopened the market for their vaccines.

Caution regarding the lives of prospective vaccinees is a means by which good intent might be demonstrated; caution is not, however, built into the present immunization regimen in the United States and elsewhere. Prescreening appears to be avoided due to expense or loss or profit: a genetic weakness in autistic children and other persons, resulting in the inability to mount a complete immune response against viral and bacterial threats, has been identified (see "***Causes***" and Wakefield's recommendations, above)--yet there are no pre-screening procedures in place for distinguishing children with such a predisposition prior to immunization. An existing high antibody count can also be a risk factor for vaccine reactions and injury; as a recent British Medical Journal stated (cited September 29, 1995, "*The Reckless Vaccination Campaign*"). Antibody status should be checked before immunization, the authors stated, so that children do not incur risks through unnecessary vaccination. Currently, prior immune status is not checked in the United States or United Kingdom ("*The Fraudulent Measles Epidemic")*. In Britain, the Department of Health was shown to have saved on the costs of inoculation during its November 1994 measles and rubella vaccination campaign by eliminating such evaluations prior to immunization ("*The Gamble*," or "*The Reckless*

Vaccination Campaign," The Web Inquirer, http://www.gn.apc.org/inquirer/gamble.html). In addition, a family history of autoimmune disease is not considered a contraindication to vaccination, though links between microbes and autoimmune disorders have been established--including those represented in vaccines. As neither the genetic nor the immune screenings are difficult to obtain, a further motive such as financial and legal considerations must be considered part of the motive for continuing to vaccinate along present lines.

In "*Combination vaccines for childhood immunization: Recommendations*" (American Academy of Pediatrics/American Academy of Family Physicians), combination vaccines are preferred in order to fit the large number of available vaccines into a relatively few well-patient visits between ages 0-6--this helps to insure children will miss few if any shots, and reduce the cost for extra health-care visits. It also will facilitate adding more vaccines into the program (pp. 7-8). However, the Academies list as a future research priorities the safety of administering combination vaccines to patients who might already have natural or vaccine-conveyed immunity, as well as the safety, in any patient, of repeated exposures to the same proteins used as antigens and/or carrier components in existing and future conjugated vaccines --implying that the safety of this practice has not been satisfactorily proven (pp. 10-12, Morbidity and Mortality Weekly Report, vol. 48, RR05, May 14, 1999, pp. 1-15; text available http://www.cdc.gov/epo/mmwr/preview/mmwrhtml/rr4805a1.htm).

Since much of our current immunization program is geared to prevent diseases that are described as mild in medical texts predating the corresponding vaccines, and for which complications are extremely rare, and from the many vaccine-efficacy articles that emphasize loss of work time and dollars from the economy due to caring for the unvaccinated sick, as well as the above considerations, it can be inferred that the aims of vaccination programs are not as single-faceted as implied by London. The ever-increasing number of vaccines in various stages of conception, development and implementation, together with the non-essential nature and/or possible dangers of many of those vaccines, further demonstrates the existence of motives other than concern for public health. Ironically, many of those vaccines in various stages of conception and development at the present time are geared to combat illnesses or conditions which may be vaccine-related.

--see also **Caveats:** ***Conflicts of Interest***, below--

(Page 16, London) "*If you're not afraid of measles (and most parents are not) then there is no reason to be afraid of the measles vaccine.*"

Abstract: putting aside the emotional, rather than scientific, character of this argument, the statement itself is ill-founded: vaccine viruses are injected directly into the bloodstream, bypassing important mucosal immune system barriers in the digestive and respiratory tracts, giving the virus direct access to the body's systems, and robbing the body of the signals necessary to produce a fully developed immune response. Wild measles caught 'naturally' is filtered through these barriers. Further, "There is good evidence that mucosal immunity may be required for optimal protection against viruses that infect via such a route…" (Molecular Virology, 1994, p. 89). "*Combination vaccines for childhood immunization: Recommendations*" (American Academy of Pediatrics/American Academy of Family Physicians), mentioned above, notes that "research to develop and evaluate alternative means of antigen delivery by the mucosal, parenteral, and cutaneous routes" would allow new and existing vaccines to be administered more safely than with needles and syringes (p. 12).

Measles immunosuppression is an additional, serious consideration in weighing the benefits and risks of MMR vaccination, as vaccine recipients are rendered vulnerable to opportunistic infection. Existing vaccine safety studies are, moreover, inadequate to evaluate the long-term safety of the MMR in any context and, as well, most other vaccines. Safety testing of the MMR vaccine included only three weeks' surveillance.

(Page 18, London) "*I am often asked…if I have vaccinated my own children. The answer is yes…and if we were to have another child, we would vaccinate him or her as well.*"

Many doctors do not share this view. Medical surveys have repeatedly found that doctors and their families are the *least* vaccinated group in their communities (JAMA [Journal of the American Medical Association],1981 (vol. 245, no. 7, pp. 711-713; British Medical Journal, January 27, 1990). Reasons given for their own lack of consent to

vaccination include "I do not trust the vaccine" and "Vaccination is of no proven benefit." Similarly, Practical Parenting magazine, December 1998, found that *parents* who did not vaccinate were those who had done the most research before making that decision.

Caveats

Consider the source

This work is designed to be a compilation of quoted and paraphrased material from studies, articles, and books dealing with immunology and autism, plus other topics, in an effort to bring together the expertise of the original writers on the issue of autism and vaccination. In contrast, "*ABCs*" is written by NAAR co-founder Eric London—not an immunologist, virologist, or a geneticist; not a pediatric gastroenterologist, nor indeed a specialist in any area which would seem to present minimal qualifications for an authoritative, virtually unreferenced expression of facts and opinions on this issue. He is, in fact, a psychiatrist, and the father of a child with autism. London, interestingly, hails from the same institution—the University of Medicine and Dentistry, New Jersey—where work a group of excellent scientists with expertise relevant to London's article—including Dr. James Oleske and Dr. Tina Zecca, of that institution's Pediatric Allergy and Immunology division. Recently a cluster of children with autism in Brick County, New Jersey made national news.

The editor of London's "*ABCs*" article, Catherine Johnson, wrote a glowing review of Shirley Cohen's recent book, Targeting Autism, on page 9 of this same issue of NAARATIVE. Cohen points out the ever-increasing evidence of immune system abnormalities in autism (pages 139-140; actual passage quoted above, in "**Causes**"). NAARRATIVE is the house organ of the National Alliance for Autism Research; NAAR's website and newsletter issues refer not infrequently to grants from the National Institutes of Health for NAAR projects, and workshops co-sponsored by NAAR and government agencies such as the Centers for Disease Control.

Conflicts of interest

Another possible criticism of the London/*NAARRATIVE* article is that, typical of most research projects and articles nowadays, there is no financial disclosure regarding either Dr. London or NAAR. Carolyn Morelli, co-founder of Pennsylvania Parents for Vaccine Awareness, asserts that authors of medical literature should make full financial disclosure concerning all sources of income—including that from consulting, and income in the form of gifts or other favors from pharmaceutical companies such as Merck, Sharpe and Dohme, makers of the MMR vaccine. "The results of medical studies," Morelli says, in a Reuters news release dated this past July, "are likely to be tainted or flawed if they are funded by industry," for (in the words of Hurst Hannum in the British Medical Journal), "at a minimum, the recipient must be accountable for how grants are spent. At a maximum, the recipient must deliver a particular product that is acceptable to the donor." Morelli refers, most notably, to recent ethics articles from the Journal of the American Medical Association which note an alarming tendency toward conflict of interest in medical studies and treatises, citing in particular those associated with the tobacco and infant formula industries.[39]

Conflict of interest in the practice of medicine and in the research arena is now such a serious and widespread problem that numerous attempts to study and construct guidelines for physicians' acceptance of gifts from industry have been made. Congress recently put pressure on the American Medical Association to begin enforcing these. Drug manufacturers are aggressive marketers with top-dollar public relations machines, who are extremely active "educators" of medical school students and providers of funding and incentives to practicing physicians and scientists. As a matter of course these firms commonly promote the use of their products through liberal gifts of

product samples, together with other gifts and incentives, to physicians. Some conflict of interest studies cited by the New England Journal of Medicine and other publications are summarized in "Headlines: Follow the Money," Environment and Health Weekly, January 15, 1998.[40] In moving against mandatory hepatitis B vaccination of young children, the Association of American Physicians and Surgeons noted that children younger than 14 are three times more likely to be killed or seriously injured by the hepatitis B vaccines than they were to catch the disease, which is not spread by casual contact, but rather by injection, sex, or an infected mother. Kristine M. Severyn, a Ph.D. pharmacist and director of Ohio Parents for Vaccine Safety, collected letters from twenty-four individual physicians opposing the vaccine, together with a copy of a thank-you letter from the The American Academy of Pediatrics to Merck & Co., manufacturer of the hepatitis vaccine, for a $100,000 donation to the Academy, which had endorsed the vaccine ("*Association says numbers show mandatory vaccinations not best for children*" (New York Times Syndicate news release, April 28, 1999).

There is no disclosure of financial and business interests in London's article, "*The ABCs of MMRs and DPTs*," but one might reasonably assume that, as a psychiatrist, Dr. London dispenses the psychoactive drugs that have become mainstays in the treatment of mental disorders—antidepressants; anti-psychotics; substances like lithium for management of bipolar disorders; beta blockers and OCD inhibitors—in treating his patients, made by drug/vaccine manufacturers such as Merck, Sharpe and Dohme. There is no direct disclosure of the interrelationships between NAAR and the Centers for Disease Control or the National Institutes for Health, who work closely with the drug industry, but in NAAR literature one observes references to NIH funding of NAAR projects and a workshop sponsored jointly by NAAR and CDC (http://www.naar.org). The relationship between NAAR and the drug industry is evidenced in the Winter 1998 issue of NAARRATIVE, which displays front-page coverage of the awarding of a "NAAR/Bristol-Myers Squibb Research Fellowship in Autism and Neuropharmacology."

Scientific propaganda

Abstract: London's "*ABCs*" article is similar to other literature written and broadcast on medical topics, in that the use of communicative (rhetorical) devices designed not to inform, but to *persuade* are used. "Trade science" institutions affiliated with drug and other industries have proliferated in the past decades and have changed the course of environmental and health politics,[41] sporting titles eerily similar in sound to that of NAAR, "National Alliance for Autism Research."

The title of the "*ABCs*" article belies the importance and complexity of its central issue—immunizations and their ill-effects—which is of world-wide significance, now and in the future. Vague allusions like "*evidence indicates*," and "*geneticists believe*" (e.g., page 14, paragraph 1), dot the pages of London's article, appearing without specifics and references to back them up (there is a very short list of sources at the end, but most 'facts' are not tied to sources in a manner that can readily be checked). Numbers are airily summoned without specificity as to time, place, population and other qualifiers, as on page 14, paragraph 11, "*many, many thousands of children died of now preventable diseases*" (it is not specified over how many years, and how much geographical distance; also the ages of affected persons and disease specifics are excluded, along with details regarding other relevant circumstances). Paragraph 3 on page 14 is formed from purest conjecture:

"*Why do some children with autism seem normal until just after they have received their vaccinations? The answer to this question* COULD *be that many children with autism develop normally for 12 to 18 months, whether or not they were vaccinated. That* MAY *be the nature of the disorder in* SOME *of its expressions…A third-world child with autism who received no vaccinations of any kind* MIGHT *show exactly the same pattern.*

Having no other information, some readers of the London article might be inclined to dismiss the issue of vaccinations and autism.

The psychology of causal attribution

"*…it is well known that* OUR *memories are not accurate enough to support medical hypotheses one way or the other.* RESEARCHERS *have conducted studies in which they cross-checked* PARENTS' *accounts of their*

children's medical histories with the actual pediatric and/or hospital records—and often the parents have remembered things differently from the way they actually occurred. This is true of ALL *parents, on* ALL *subjects*….." **(London, p. 17)**

Specifics are unfortunately missing, in this statement, as *to whom* it is "well known" that memories are not accurate; which researchers have checked which parents' accounts; which studies were published describing this work; the details of those studies and, particularly, why such studies should invalidate *all* parents' *every* observation in *all*circumstances. Perhaps predictably—since he is a psychiatrist—London attributes to psychological factors parents' belief that vaccinations precipitated their children's developmental and medical conditions (in spite of the fact that such histories are given credence in works such as those in "***Cause*[s]**," above). A number of illnesses were pronounced "psychological" by physicians in the not-so-distant past—e.g., lupus erythematosus, Sjogren's Syndrome, and the Chronic Fatigue and Gulf War syndromes. London's thesis in this regard is challengeable also in its apparent assumption that parents would rather believe that their children's autism is the result of outside factors--e.g. vaccines--than their own genes. In actuality, most parents of autistic children reject out of hand the idea that the vaccinations commonly believed to be safe—that are represented as safe by the United States government and physicians around the country—could have harmed so many children, until laboratory reports and existing information put an end to denial. One of the strongest psychological drives of human beings is the need to cling to one's personal belief system or psychological "set"--which usually includes belief in one's government as an authoritative source of information and a protector of citizens. It is far less disturbing to believe that autistic children's conditions result from chance genetic encounters, problems at birth, or unknown factors—where no one is at fault—than it is to realize that industry concerns can and often do dominate those of government, in this case to mislead both parents and physicians. One mother—a physician herself—whose child regressed post-MMR wrote,

"Before…, my husband and I assumed that Mike's autism was completely genetic. It made the whole thing much easier to accept—it wasn't our fault…I did believe everything I was taught about vaccines—they were safe…[but] last December removed every trace of doubt from my mind when [a preeminent immunologist] reviewed Mike's labs with me and went over his history in great detail. *I did this to my child. I drove him to the pediatrician's office. I held him down while they gave him the MMR. The guilt is almost overwhelming at times…I ruined his life*."[42]

Parents vs. professionals?

London's skepticism toward parental conclusions is not shared by most professionals who work with autistic children. Rutter and Schopler, eminent in the field of autism, note the importance of obtaining detailed information from parents in formulating a comprehensive assessment of a child. Information (i.e., memories, as well as documented information) on the child's history is one of several types of information specifically sought from parents in the renowned TEACCH program (Diagnosis and Assessment in Autism, 1988, pages 294-5). This practice is standard in speech and occupational therapy, as well. Parental input is even mandated by federal law: in the special education arena, IDEA legislation made parents an essential element in the "Assessment Review and Dismissal" or "Individual Education Plan" committees which are charged with all aspects of a child's educational fate.

Regarding parental vaccine damage reports, even the medical community is not always prejudiced against parental input. In "*Measles, Mumps, Rubella Vaccine Induced Subacute Sclerosing Panencephalitis*" (Journal of the Indian Medical Association, November 1997), the authors comment, "The parents proved to be extremely reliable informants." Richard Moxon, Professor of Pediatrics at Oxford University, gave his support to parents pleading for improved research into vaccine side-effects, noting, "Over 200 [parents] had come forward…The reports I heard were plausible…" (Janine Roberts, "*A Horrendous Gap in Research*…, The Web Inquirer, October 7, 1995,http://www.gn.apc.org/inquirer/moxon.html). Even so, the parents of regressively autistic children with bowel and metabolic problems do not expect their memories to support medical hypotheses—only that these memories, together with the information and evidence at hand concerning immune system abnormalities in autism, provide the impetus for independent and thorough medical inquiry.

Autism research needed

Inquiry should focus not only on the immune abnormalities seen in autism, and the possible vaccine connection, but should also go, as shorter-term goals, toward development of a complete list of contraindications to vaccination (such as a family history of immune system abnormalities; high serum antibody counts to viruses represented in the prospective vaccines), screening procedures to eliminate persons susceptible to vaccine damage from the immunization program, and, in the long-term, toward development of safer types of vaccines. In the meantime, monovalent (single-virus) vaccines should be available for separate, carefully timed vaccination—particularly in the case of measles and rubella. This is particularly important in the case of the MMR: as noted by Wakefield, et al., in *"Ileal-lymphoid-nodular hyperplasia*," measles and rubella together may cause more damage than either alone.

Several scientists and authors have designated directions for further research regarding autism, genetics and immunology:

The Centers for Disease Control, [1998], "*Vaccines and Autism: Is There a Relationship*?" *http://www.cdc.gov/nip/vacsafe/vac_autism.htm*). *…An issue unresolved* by [a working group convened by the National Institutes of Health in 1995] *was the role of immune factors in autism spectrum disorders; it was suggested that studies to clarify the situation are needed*."

Van Gent, et al., "Autism and the Immune System," Journal of Child Psychology and Psychiatry, vol. 38 no. 3, 1997, pp. 337-349. The authors note that research in this area to date has been limited primarily to adults or mature subjects, rather than children. They, along with the authors of "*Autism: Towards an Integration of Clinical, Genetic, Neuropsychological, and Neurobiological Perspectives*,"[43] suggest specific directions for further research in the immunology of autism, and for linkage of these studies to studies on imaging, behavior, etc. Van Gent, cited above). Van Gent, et al. observe that "*the immune response is regulated by genetic material located mainly on the sixth chromosome.* The authors conclude their study and review of the literature by suggesting *that the brain abnormalities* that have been found in autism may be "but one consequence of abnormal brain development, which in turn *may be linked with disordered immune function* in a yet unknown way" (page 346). They stress the importance of further research on the immunological abnormalities in autism, and suggest some specific avenues for study.

"*Vaccine-induced autoimmunity*" (Journal of Autoimmunity, vol. 9, no. 6, December 1996, pp. 699-703): the authors summarize of case reports attributing autoimmune diseases and autoimmune phenomena to vaccines, and comment, "The subject of the vaccine-autoimmunity relationship is still obscure; reports have been rare, [and] no laboratory experimentation on this topic has been undertaken....." They conclude that "laborious clinical and laboratory studies should be initiated in order to evaluate the ...subject.")

Immunologic mechanisms in neurologic and psychiatric disease (Raven Press, 1990),[44] p. 1: "Our knowledge of disease pathogenesis in the case of schizophrenia *and the affective disorders* is too fragmentary to permit definitive assignment to an immuopathologic category. However, *the psychiatric disorder associated with systemic lupus erythematosus may serve as a paradigm of immunologically mediated psychiatric disease…..*"

Piven, J. et. al. (1990). Magnetic resonance imaging: evidence for a defect of cerebral cortical development in autism. American Journal of Psychiatry, vol. 147, number 6, pages 734-739, "*…the relationship between cortical malformations and prenatal* ***immunologic****, neurochemical, and genetic abnormalities in autistic subjects warrants further exploration*. Our findings raise several questions regarding the validity of MRI in detecting cerebral cortical malformations….. (p. 738)."

In Margaret L. Bauman and Thomas Kemper's The Neurobiology of Autism (Johns Hopkins Press), 1994, Introduction by Isabelle Rapin: "*At least 40 percent of parents report that their infant or toddler, whose development may or may not have been entirely normal up to then, experienced a regression*, usually insidious but occasionally abrupt, in language, sociability, and play… Development resumes after a plateau…but, in most cases, never returns to its previous level… *Many speculations have been offered…to explain autistic regression: slow viral infection,*

autoimmune phenomenon, lack or insufficiency of a growth factor *at a particular time in development*... ***Data need to be collected to investigate these speculations*** (pages 13-14)**.**"

"*Role of Immunogenetics in the Diagnosis of Postvaccinal CNS Pathology*," Department of Pediatric Surgery, University of Bari, Italy, presented May 9, 1996 (text available http://www.healthy.net/library/articles/coulter/biochem.htm--a fuller description of this study will be found in "**The attenuated virus--infectious or not?**" above): the fact that post-vaccinal pathologies of the central nervous system are often not thoroughly investigated occasioned this study. *Additional cases are under study to better define the possible association of HLA A3 and/or HLA DR7 with this CNS pathology following vaccination.* Initially, thirty children were found to have signs of central nervous system and genetic damage following vaccination. The authors remark, "*A study of the disease associated with genes of the HLA system has shown that this genetic complex can be responsible for a particular genetic susceptibility, predisposing to various diseases characterized predominantly by immune-system pathogenesis*… **Conclusion[:]** The results indicate that autoimmune pathology is more frequent in countries where vaccination is more widespread… ***With this study***, and with the individualization of alleles such as A3 and DR7, in the presence of viral DNA, ***it would be possible to define the subjects at risk of an autoimmune pathology from vaccination.***"

"*Cerebral palsy, autism, suboptimality…*Teresa C. Binstock, Researcher in Developmental and Behavioral Neuroanatomy, mused that further research of a purely genetic design might endanger children with autism: "If some autism-spectrum kids still have a subclinical infection within the CNS (as may be indicated by their gradual deterioration), then **waiting years for additional genetic research to be completed may enable further deterioration to occur**. **In contrast, if this subgroup of kids can acquire** aggressive diagnostics regarding possible subclinical CNS infections, and can then receive **the best treatments available for those infections…then further deterioration may be prevent[ed] or minimized** (post from Autism discussion list originating at St. Johns University, New York, April 30, 1999, 18:39:12 -0700).

NAAR representation and support

The word "National" corresponds to the "N" in the NAAR acronym. It is clear, in the case of the article this paper concerns, that only one viewpoint is represented—in essence the one held by the United States government and the pharmaceutical industry. The U.S. government linked itself with the drug industry in 1986, when it took responsibility for vaccine injury via the Childhood Vaccine Injury Act--thus placing itself in an obvious conflict of interest between public health and pharmaceutical industry concerns. One might speculate also that perhaps the word "national" might correspond to the organization's government-derived funding and associations. In any case, the term "Autism" appears to be used broadly by NAAR in its literature, without distinction as to subtypes: presumably, then, NAAR *purports* to represent the atypically autistic population, as well as sufferers of 'classic,' or "Kanner's" autism—but does not seem to be doing so in addressing immunological issues. Its commitment to quality research is certainly not reflected in "*ABCs*"--though "*ABCs*" might perhaps be seen as a expression of opinion. In the latter case, however, an opposing viewpoint was not permitted by NAAR: world-recognized autism expert Bernard Rimland of the Autism Research Institute requested, but was denied, the privilege of placing an article in the NAARRATIVE which would explore the same themes as London's "*ABCs*," and present alternative conclusions. 'Representation' in a high-quality research-devoted institution should necessarily extend not only to a variety of populations but a variety of ideas; unfortunately this is not yet the case with NAAR.

NAAR's web-prospectus (www.naar.org) indicates that its founders intended NAAR to become a nationwide alliance of "families, researchers and others concerned with autism," united in its efforts to fund and accelerate autism research. A number of families have withdrawn their memberships and support from the Autism Society of America because it has declined to take a firm stand on the need for further research into immunological and metabolic issues in autism.[45] Will NAAR suffer a similar fate because it fails to give such issues fair treatment and equal research support? The statements, in the Editor's Note and at the conclusion of the "*ABCs*" article—that NAAR will still [in spite of London's stance on the autism/immune dysfunction issue] support the "sound

epidemiological research that it would take to answer this question"—imply otherwise, but are, of course, yet to be substantiated.

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At eight years of age, Ruede's daughter Deanna has autism in tandem with inflammatory bowel disease and multiple chronic infections, including measles, rubella, diphtheria and tetanus, as indicated by excessively high antibody counts and corresponding metabolic patterns. She was fully vaccinated, experiencing none of the preventable childhood diseases except varicella zoster (chicken pox).

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LINKS

National Vaccine Information Center (NVIC) http://www.909shot.com/

Vaccine Information and Awareness (VIA) http://www.access1.net/via/

Parents Requesting Open Vaccine Education (PROVE--Texas) http://home.swbell.net/prove/

Vaccine or DPT injuries http://neuro-www.mgh.harvard.edu/forum/VaccineorDTPinjuriesMenu.html

Links to Immunology on the WWW (British Society for Immunology) http://www.sunderland.ac.uk/~hs0acu/link1.htm

Center for Complex Infectious Diseases (Rosemead, CA, USA) http://www.ccid.org/

Autism Autoimmunity Project http://www.gti.net/truegrit/

The Leading Edge Research Group Master Analysis of the Vaccination Paradigm http://www.trufax.org/vaccine/vacmaste.html

Autism Research Institute (San Diego, CA, USA) http://www.autism.com/ari/

Autism Research Unit (University of Sunderland, UK) http://osiris.sunderland.ac.uk/autism/index.html

Society for the Autistically Handicapped (SFTAH) http://www.rmplc.co.uk/eduweb/sites/autism/index.html