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What is regressive autism and why does it occur? Is it the consequence of multi-systemic dysfunction affecting the elimination of heavy metals and the ability to regulate neural temperature?

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Abstract

There is a compelling argument that the occurrence of regressive autism is attributable to genetic and chromosomal abnormalities, arising from the overuse of vaccines, which subsequently affects the stability and function of the autonomic nervous system and physiological systems. That sense perception is linked to the autonomic nervous system and the function of the physiological systems enables us to examine the significance of autistic symptoms from a systemic perspective. Failure of the excretory system influences elimination of heavy metals and facilitates their accumulation and subsequent manifestation as neurotoxins: the long-term consequences of which would lead to neurodegeneration, cognitive and developmental problems. It may also influence regulation of neural hyperthermia. This article explores the issues and concludes that sensory dysfunction and systemic failure, manifested as autism, is the inevitable consequence arising from subtle DNA alteration and consequently from the overuse of vaccines.

Keywords: autism, physiological systems, autonomic nervous system

Introduction

That the occurrence of autism has risen steadily in the last decades is not in dispute. Prior to the 1930's and the introduction of vaccinations autism was unknown. By 1968 in the UK, when Polio and DPT vaccines were given at 6 and 7 months autism was very rare. In 1988, when Polio and DPT was given at 3 months, DPT at 5 months and MMR at c13 months autism rates were still low. In 1996, when Polio and DPT/HIB injections were given at 2, 3 and 4 months, followed by MMR at c13 months autism rates began rising rapidly. By 2006 the occurrence of autism had reached pandemic proportions. In the period shortly before the 1980's the occurrence of autism was estimated to be circa 3-5 per 10,000; the majority having autism from birth[1]. Since the introduction of multiple vaccines the prevalence of autism has increased to an

estimated 1 in 166 i.e. 60 per 10,000. Furthermore the trend is that of a continued increase. Some British teachers are claiming to see ASD in one in every 86 children[2]. This is supported by research which suggests that one in 100 British children may have some form of autism[3] and that ASDs are more prevalent than hitherto imagined[4] i.e. only severe cases of autism are recorded in the statistics. Such claims have been dismissed as mere speculation on the basis that there is not yet definitive proof of such claims however the perceived lack of evidence does not indicate that proof does not exist[5,6]. It may indicate that the understanding of the condition remains 'beyond the prevailing level of knowledge' (Table 1) [7].

By 1985 the incidence of regressive autism had equalled that from birth. By 1997 both types had increased although the regressive form was now >75% of the total occurrence. This suggests that an acquired condition was overtaking birth defects or purely genetic conditions. Autism affects four boys to every girl[10]. By contrast Autism appears not to occur in communities which do not use vaccines[11]. It occurs in immigrants from tropical climates who appear to have greater familial predisposition to autism[12] e.g. among Somali students in Minneapolis there was a rate of 1 in 28 (which compares with the local average of 1 in 56). This is more than five times the national rate of 1 in 150. Since the 1960's the number of vaccines given to a child before entering school has risen to c33. In children born to military families the occurrence of autism may now be as low as 1 in 67. In the vast majority of cases, the emergence of autistic indications appears to happen in children who had developed normally[10,13,14], and before three years[15,16]. The development of normal immune function appears to cease in the second year and is linked to the schedule of vaccines[17] and/or the MMR vaccine[18,19]. The consequences to society are estimated at c£2.4M in an autistic child's lifetime[20] which, if it continues to increase as many predict, will impose an unsustainable financial burden upon healthcare, education and social welfare systems.

The Systemic Nature of Physiology and Function

The body is a bio-dynamic, wholistic and systemic organism. It responds to sensory input which enables the autonomic nervous system thereby influencing behavior, the regulation of physiological systems, and function of the visceral organs (Fig. 1). The established association between visual perception, the autonomic nervous system, physiological systems, and biochemistry[21] raises issues which may be relevant to autism research.

- Different diseases are associated with differing colour perception[22] e.g. a yellow-blue deficit in diabetes[23], etc
- Different drugs are associated with altered color perception[24].
- Enzymes/Proteins are active in the visual spectrum[25,26].
- Suppressed immune function affects cognition[27]. In particular, t-cell deficiency (a common indicator of stress) is linked to cognitive dysfunction.

Any form of biochemical variation must therefore influence sense perception, sensory coordination and cognitive function. The existence of the physiological systems is not in doubt although there is not universal agreement on their structure. There is wide recognition that they regulate the function of organs (in each system), and that there are higher and lower levels for each system (homeostatic limits), however such systems remain an elusive and under-researched area of medicine. The Russian researcher

I.G.Grakov[28,29] has mathematically modelled the consequences of cognition upon the autonomic nervous system and physiological systems. This included identifying and mapping the nature and structure of the physiological systems (Table 2).

Physiological Systems

Sleeping, Breathing, Digestion, Excretion, Osmotic Pressure, Blood Pressure, Blood Cell Content, Blood Volume, Blood Glucose , Sexual Function, pH, Temperature, Posture and Locomotion. See Table 2.

Such an explanation is highly inclusive and complete by comparison to the currently accepted but exclusive and limited explanation. The essential functions of temperature, sleeping and pH are now included; excretion is not limited to urination; whilst blood cell content (and other related systems) comprise what has hitherto been regarded as the immune system. Absorption of nutrients is influenced by system function including (but not limited to) blood pressure, blood volume, blood cell content, pH, temperature, etc. Elimination of toxins is similarly influenced by the complexities of system function.

The brain manages the autonomic nervous system and the function of the physiological systems. In addition, the brain waves are in a dynamic relationship with molecular biochemistry illustrating how drugs can be used to influence the body's biochemistry in order to act upon the symptoms of disease and how brain wave technologies such as neurofeedback can be used to alter the brain waves, physiological systems, organs, cells and molecular bio-chemistry.

Such systems regulate the function of the body's biochemistry e.g. (1) Most enzymatic reactions in the body are temperature dependent and catalysed by Magnesium. (2) The body requires maintenance of pH within a narrow operating range, and also the supply of minerals and vitamins/cofactors, to catalyse protein-substrate reactions in the body. (3) Appropriate blood volume, blood pressure, blood cell content and pH are required to ensure optimal absorption of minerals, vitamins, fatty acids from the intestines.

It is increasingly accepted that the synchronised activity of groups of neurons[30] in functionally coherent structures (the physiological systems), which exist in the brain *and the body*, synchronise their electrical impulses[31]. This may be evident when noting the evoked visual potentials, indicative of neural synchronisation, which are atypical in autism[32] and which may be part of the processes influencing sense perception (figure 1), sense coordination, memory[33], learning, etc. If so, this indicates that sensory input through the neurovisual pathways is integrated into actions, behaviour and movement and that learning requires synchronised activity between the brain, sensory organs[34–36], and visceral organs. This is severely disrupted in the autistic[37]. Autism affects the function of all of the brain[38–40]. It is a neurobiologic, multi-systemic disorder i.e. affecting the function of every organ but not necessarily its structures[41]. It affects all aspects of the autonomic nervous system and hence influences all aspects of brain's function including that of neural networks involved in learning, memory, the function of the senses and the visceral organs.

The cerebellum, considered to be implicated in autistic spectrum disorders[42] comprises an estimated 50% of the brain's total processing capacity yet its role is not clear or understood[43]. It is involved in the accumulation of sensory data from the internal environment, including the organs in the body and those in the brain (including the sensory organs), thus distinguishing between sensory input from the external environment (a significant function of the cerebrum) and that of the biochemistry affecting the function of every organ (a significant function of the cerebellum), including the cerebellum. Such a role includes the processing, regulation and distribution of this data, through structures such as the Purkinje cells in the cerebellum which are attached by nervous structures to every part of the body. This includes the receipt of

biosignals involved in the processes of movement, coordination and balance. Impaired flow of data to the brain via the cerebellum (and brainstem) may lead to functional problems affecting the body's fine control of e.g. balance, coordination, etc. Movement and balance involve the coordinated function of all body systems and organs and are coordinated by (1) sensory feedback from the external and internal environments and (2) the allocation of energy resources to and from each organ. They are dependent upon the precise nature, and timing, of data about each organ being provided to and by the cerebrum *and* cerebellum. This illustrates how the brain determines behaviour and actions appropriate to developing situations. It illustrates how changes at the organ, cell or molecular level influence brain function and vice-versa.

There are indications of cerebellar dysfunction in autism[44]. Inhibited flow of data to the cerebellum may be followed by developmental decay, cerebellar dysfunction[45,46], and reduced size of brain-stem. This is equivalent to the ‘use it or lose it’ phenomena affecting muscle tone and function.

Without cognitive input the brain cannot and does not function. Disease and drugs create cognitive dysfunction, altered sense perception, in particular affects visual perception. Accordingly, vaccines must also influence sense perception and coordination. Vaccines have a long-term influence and hence may have a more pervasive influence upon sense perception.

Our cognitive function depends upon the extent and coordination of sense perception i.e. between the eyes, ears, nose, mouth and skin. Genetic and/or environmental influences affect sense perception, the degree of sensory coordination and ultimately our connectedness with the surrounding world. Visual function is linked to the primary mechanism (rods, cones and pigments) but is also influenced at the biochemical level – noted by how pathology and drugs alter color perception[22,47] and affect the magnocellular and parvocellular neurovisual pathways which alter color perception and visual contrast. This influences the stability and function of the autonomic nervous system[48] and alters the processes of memory fixation, concentration, and behavior[49].

Anyone contracting disease e.g. measles, mumps, rubella, tetanus, etc; experiences altered visual perception therefore a weakened strain of the disease e.g. in vaccines, must also influence visual perception/cognition. Chronic disease is also accompanied by significant cognitive dysfunction and influences the coordination and processing of sense signals by the brain. The greater the number of illnesses, drugs or vaccines[50] the greater the alteration to the body's biochemistry therefore the greater its influence upon sense function and the degree of sensory distortion. It influences the autonomic nervous system and physiological systems and hence the coordination and function of every organ – visceral and sensory. This is a significant feature of autism[51,52].

Almost all diseases are linked to cognitive and behavioral disorders. Conversely, behavioral traits are influenced by biochemistry e.g. testosterone, oestrogen, cortisol, oxytocin, adrenaline, etc. Oxytocin influences the formation of social bonds influencing social engagement and attachment - which are dysfunctional in the autistic child[53–57].

Autonomic nervous system dysfunction?

In general problems with the stability of the autonomic nervous system[21,58] can be expected to be manifest as follows:

- Loss of Sense perception and Sensory Coordination
- System dysfunction (e.g. influencing breathing, blood pressure, heart rate, etc)

- Behavioural dysfunction (including learning problems, information feedback)
- Problems with Diet and Elimination (of toxins and wastes)
- Impaired and/or Delayed Neural Development
- Atypical brain waves

These are prevalent in autism.

Evidence of Systemic Dysfunction in Autism

Multi-systemic dysfunction is associated with a wide range of physiological disorders e.g. diabetes and obesity[59], cancer, cardiovascular disorders, pre-eclampsia, dyslexia[60], depression, etc. It affects the central[39] and autonomic nervous system in autistic children[61]. Systemic dysfunction in Autism includes that of temperature, blood cell content and immune function[62], blood pressure[63,64], digestion, excretion, posture and locomotion, sleep[65–67], pH, breathing; respiration rates, lower skin temperature. Each influences metabolic rate[68]. Autonomic dysfunction has also been linked to problems with appetite, swallowing food, nausea, recurrent vomiting, and abdominal bloating; constipation or diarrhoea; dry eyes, dilated pupils; dry skin, flushed skin following a meal, abnormal sweating, and unexplained high fevers; sleep apnoea, insomnia; bed-wetting, difficulty urinating, difficulty potty-training; altered perception of pain, sensory defensiveness, poor socialisation skills, anxiety, phobias, tics, emotional instability; and light intolerance. That autistic seizures are often linked to neural blood flow[69–71] is supported by fact that medications used to raise or lower blood pressure can alter the occurrence of seizures and improve sleep in the autistic child.

Autism affects sensory processing and sensory coordination[72] which is manifest in various ways e.g. tactile perception[73], vision[74], hearing[75], and smell. Autistic children may also display synaesthesia in which sensations become confused with one another[76]. Sounds may be experienced as touch or as visual stimulation e.g. autistic children may cover their eyes when they hear a loud sound. That autistic children have such sensory synaesthesia and sensitivity may indicate that their brains have extreme problems with sensory processing, regulation and coordination[77,78,60].

Vaccines and Vaccine Side-effects

Background The introduction of modified live viruses as vaccines enable the virus to attach its genetic material into the cell which replicates i.e. the host cell continues to function whilst producing the viral protein. This stimulates the production of antibodies. Under normal circumstances exposure to a viral disease would be countered (in vivo) at various levels enabling the body to steadily increase its immune response. By contrast, the injection of vaccines directly into the blood system overpowers the normal immune response leading to its rapid depletion. It is now suspected that long-term persistence of viruses and other proteins may produce chronic disease i.e. instead of producing a genuine immunity the vaccines are altering the body's systemic and biochemical stability, suppressing the production of differing types of white blood cells and hence immune function. Furthermore the introduction of many vaccines (up to 30 in a typical vaccination schedule) introduces a large number of foreign proteins which may be sufficient to ensure that immune function never returns to baseline and/or that immune biochemistry is fundamentally altered[62]. Consequently there now exists a growing concern which links immunizations to the huge

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increase in recent decades of auto-immune diseases[79] e.g., rheumatoid arthritis[80,81], multiple sclerosis, lupus erythematosus, lymphoma, leukemia, autoimmune demyelinative optic neuritis, diabetes mellitus, etc.

Vaccinations influence the balance of viral scavengers[82,83]. They suppress the production of b-cells, t-cells, etc. The synergistic action of these cells impairs antibody formation and becomes less effective in phagocytosis. This influences recognition of viral pathogens, leads to the progressive failure of immune function and hence to the increased incidence of auto-immune disease which we note as allergies[84–86] and immunodeficiency[87].

Some vaccinations have a greater effect than others e.g. Hib vaccine, pertussis vaccine[88–90], measles vaccine[91], etc. Indeed some articles indicate that the use of such vaccines can reliably induce asthma[92] by moderating adrenergic function[93].

Modified live viruses alter the structure and function of DNA. Each virus is a large molecule therefore its spatial arrangement must be influenced by its biochemistry which influences cross-helical structures and linkages within the DNA helix. Accordingly it is inevitable that the steady accumulation of such foreign proteins arising from an intensive vaccine programme will reach the stage where it significantly weakens DNA, gene, and chromosome structure and function. The prevailing reaction conditions - the consequence of protein expression which has been influenced by previous vaccines - will also affect the introduction of each modified live virus. Each will depress immune function. The greater the number of viruses and foreign proteins (1) the greater the influence upon immune function and the time required for recovery from each vaccination; (2) the greater their influence upon DNA, gene and chromosome structure and function, the greater will be the risk of protein inhibition, system dysfunction, reproduction, etc.

The greater the amount of vaccines, introduction of foreign proteins and hence of alterations to the body's biochemistry the greater the risk that the body's immune function no longer recognizes or responds to existing vaccines or diseases[94] and/or that its immune response has been altered[95] and/or that sugar chains attached to an antibody alters its ability to bind to its receptors[96]. This may lead to mutated forms of disease[97–104] e.g. the reemergence of whooping cough[105], and a differentiated disease profile e.g. up to 30 per cent of individuals with a persistent cough are infected with B. pertussis[106]. Furthermore enhanced susceptibility to virus infection by vaccines is documented[107]. This could enable tougher strains to flourish[108].

Vaccines are not entirely safe. The currently used vaccines are merely less unsafe than previous vaccines[109,110] e.g.

- The Urabe strain of mumps vaccine in the MMR vaccine was replaced by the Jeryl Lynn mumps strain in response to reports from Japan linking the Urabe strain used, in the MMR vaccine, with high levels of meningoencephalitis.
- The Pluserix-MMR and Immramax-MMR vaccines were withdrawn because of reports of mild transient meningitis. The withdrawal of the smallpox vaccination led to a reduction in the incidence of TB.
- The Rubini vaccine continues to be used in some European territories although discredited[111].
- Leningrad-Zagreb strain is commonly used in developing countries, and may have superior efficacy when used during epidemics[112,113].
- Different strains of disease have different safety profiles[114]

- Different strengths of vaccine[115] carry risks which affect age groups or sexes differently.
- There are concerns over the use of whole-cell vaccines[116,117] although some argue that acellular vaccines are less effective[118].
- Sudden Infant Death Syndrome has been largely eradicated following withdrawal of the pertussis vaccine in Sweden and Japan.
- Side-effects arising from vaccination are associated with the onset of autoimmune disease[79,119], arthritis, diabetes mellitus, autoimmune demyelinative optic neuritis, etc.
- Sensory defects are a common side-effect of vaccines[120–122] e.g. sensori-neural hearing loss induced by the MMR vaccine.
- Drugs inhibit the effectiveness of vaccines (see 3.3.2). Systemic glucocorticoids (steroids) suppress the immune system and create risk of disseminated infection from live virus vaccines[123]. Vaccines may also be influenced by levels of immune function, dietary factors, and stress[124]. Many parents of autistic children and a number of medical experts believe the MMR vaccine is the culprit behind autism. In c15-20% of children it causes fever 7-12 days following immunization.

What are the risks from the diseases against which a vaccine is meant to protect?

Diphtheria, Polio, Tetanus, Meningitis, Pertussis Diptheria[125], Polio and Pertussis have largely been eradicated in the developed world although there may now be mutated forms of disease, a differentiated disease profile and/or an altered immune profile, which may be responsible for further outbreaks in vaccinated children and adults. Diphtheria is an upper respiratory tract infection characterized by sore throat and minor fever. It affects the central and peripheral nervous systems leading to deterioration of myelin sheaths, loss of motor control and sensation. Fatality rates are 5-10% although the rate of mortality may be higher for those under 5 years and over 40 years. It can be treated by antibiotics which prevent its transmission e.g. using erythromycin, procaine penicillin G, rifampin or clindamycin. Other minor complications including neck swelling, nausea, vomiting, listlessness, pallor, and a racing heart beat; lead to long term effects e.g low blood pressure, cardiac myopathy and peripheral neuropathy. Poliomyelitis is an infectious viral disease. Although c90% of polio infections are symptom-free, if the virus enters blood circulation this may lead to further complications. In c1% of cases, where the virus enters the central nervous system, it infects and/or destroys motor neurons thereby leading to muscle weakness and paralysis, usually involving the legs. Tetanus infection occurs through open wounds. It occurs commonly in hot, damp climates with soil rich in organic matter. It creates muscle spasms in the jaw, difficulty in swallowing, muscle stiffness and spasms throughout the body. The neonatal form of the disease is a significant public health problem in the developing and/or agricultural economies. There are about one million cases of tetanus reported each year, mainly in the developing world, causing an estimated 300,000 to 500,000 deaths. In the United States, there are about five deaths from tetanus each year. Tetanus is the only disease that is infectious but not contagious. Pertussis is a highly contagious disease. There are 10–90 million pertussis cases and about 600,000 deaths per year. Sixty percent of all cases occur in the developing world. In children it is characterized initially by mild respiratory infection symptoms before developing into the characteristic ‘whooping’ cough. Other complications may include encephalitis, pneumonia, and secondary bacterial infections. Naturally-acquired disease caused by Hib (H. influenza) appears only to occur in humans with low natural immunity[126]. In infants and young children, H. influenza type b may cause pneumonia, and acute bacterial meningitis. Both H. influenza and S.

pneumonia can be found in the upper respiratory system of humans i.e. both reside naturally in the body. Alterations in the immune response; attributed to poor nutrition, stress or transmission; enable their proliferation with potentially serious outcomes.

Measles, Mumps and Rubella Measles is largely a consequence of compromised immunity arising from poor diet and is linked to high levels of mortality[127] in the developing world. In developed countries, most children are immunized against measles by the age of 18 months, generally as part of the triple vaccine treating measles, mumps and rubella (children younger than 18 months usually retain measles antibodies (Immunoglobulins (Ig)) transmitted from the mother during pregnancy). The rate of mortality from measles is typically 0.3% however in the developing world this may be as high as 28%. The classical symptoms of measles are typically fever (up to 40C), cough, coryza and conjunctivitis. Complications include mild diarrhoea, pneumonia, encephalitis, SSPC, and corneal ulceration or scarring. They are usually more severe amongst adults. Permanent hearing loss or damage to vision is recognized complications of measles. Measles has been known to occur in children with congenital rubella syndrome, and has been implicated in the etiology of inflammatory bowel diseases (IBDs). The more common symptoms of mumps are parotitis, fever (typically 38.3C), headache and orchitis[128] Other symptoms of mumps include sore face and/or ears, and loss of voice. Known complications of mumps include infection of other organ systems, sterility in older men, mild forms of meningitis, encephalitis, sensorineural hearing loss, pancreatitis, inflammation of the ovaries, and risk of spontaneous abortion during pregnancy. Rubella is a mild disease which often passes unnoticed[129]. The primary reason for the introduction of a vaccine is to prevent infection during pregnancy. The common symptoms of rubella are the appearance of a rash on the face, trunk and limbs (after an incubation period of 14-21 days) which usually fades after several days. Other symptoms include fever (typically 38C), swollen glands (post cervical lymphadenopathy), joint pains, headache and conjunctivitis. Rubella is generally a mild disease, rare in infants or those over the age of 40. The older the person the more severe the symptoms e.g. some women experience arthritis type symptoms. Children exposed to rubella in the womb may show developmental delay, inhibited growth, hearing disabilities, diabetes, glaucoma, schizophrenia, etc. If infected during the first 12 week period of pregnancy this may lead to congenital rubella syndrome (CRS), which is manifest as a series of complications including spontaneous abortion and, in the neonate: cardiac, cerebral, ophthalmic and auditory side-effects. Known complications include prematurity, low birth weight, and neonatal thrombocytopenia, anemia and hepatitis. CRS is the main reason a vaccine for rubella was developed. It increases the risk of miscarriage or still birth in mothers who contract rubella shortly before or early in pregnancy. If the baby survives, it may have heart disorders, blindness, deafness, etc. CRS is manifest as sensorineural deafness, eye problems, heart disease. Other complications include low birth weight, mental retardation, problems with the spleen, liver and bone marrow, etc. Hepatitis B is difficult to catch and comes from blood or sexual contact with an infected carrier. Further, vaccine-derived immunity is thought to be short-lived. Hpv , an infection transmitted during sexual intercourse, clears naturally after several months/years. Mumps and Rubella may occur without the patient being aware that they have the disease.

Some diseases may confer natural immunity e.g. the mumps virus may confer a degree of immunity against ovarian cancer[130–133].

In summary, disease side-effects reflect the effect of the disease upon the body's functional systems i.e. upon temperature, digestion, excretion, etc. Typical viral fevers are circa 1-2C above the body's normal body temperature. Measles is particularly noteworthy because fever may reach 40C (or higher), some 3-4C above normal body temperature and just 1C below the point where proteins denature and at which brain death commences.

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What are the risks from the Vaccine? Typical vaccine side-effects There is evidence that BCG and measles vaccinations administered singly reduce child mortality[134] but that this is unrelated to the incidence of measles or measles deaths[135,136]. By contrast the pertussis vaccine is associated with a negative effect[137].

Dtap: Recorded common side-effects with the DtaP vaccine include *fever*, tiredness, poor appetite, vomiting and inflammation. Less common and more severe side-effects include distress (crying), seizures, lowered consciousness or *coma*, brain damage.

MMR: Recorded common side-effects with the MMR vaccine include *fever, swelling of the lymph glands*, tiredness, poor appetite, and *abhorrence of bright lights*. More severe problems include *low platelet count*, pain and stiffness in the joints/inflammation. Less common and more severe side-effects include distress (crying), seizures, *deafness*, lowered consciousness or *coma*, brain damage.

Tdap: Recorded common side-effects with the Tdap vaccine include pain, chills, *fever*, headache, tiredness, poor appetite, stomach ache, *vomiting, diarrhoea* and inflammation

The above listed vaccine side-effects are indicative of systemic instability affecting most physiological systems – temperature (chills and fever), excretion (inflammation of the lymph glands), blood cell content (low platelet count), excretion (diarrhoea), digestion (poor appetite, vomiting), sleep (coma), and metabolic rate (tiredness, lowered levels of consciousness). In addition there is evidence of altered sense perception, indicative of problems with the autonomic nervous system, which affects hearing, visual perception (abhorrence of bright lights), smell and touch.

Significant vaccine side-effects have been linked to swine flu vaccine (Guillain-Barre paralysis); in RSV vaccine[138]; in the measles, mumps and MMR vaccines[139]; hepatitis A and B vaccine[140]; tetanus vaccine; smallpox vaccine; polio vaccine; pertussis vaccine[141], etc. The incidence of vaccine side-effects may now be sufficiently great to question the claims that the risks from the disease exceed that of vaccines[109].

The MMR vaccine has been linked to autism, Crohn's disease, inflammatory bowel disease[142,143] and other serious chronic stomach problems[144], epilepsy, brain damage including meningitis[145,146], cerebral palsy, pancreatitis[147] and diabetes mellitus[148–150], encephalopathy, encephalitis[151,152], hearing and vision problems, arthritis, behavioural and learning problems, chronic fatigue syndrome, diabetes, Guillain-Barre syndrome, idiopathic thrombocytopaenic purpura, subacute sclerosing panencephalitis (SSPE), leukaemia, multiple sclerosis, and death.

There is evidence that in cases of immune deficiency that viruses continue to persist in the body[143,153–155]. The measles virus is known to persist in patients with subacute sclerosing panencephalitis (SSPE), measles inclusion body encephalitis (MIBE)[156] and multiple sclerosis[157]. Since the introduction of measles vaccines, vaccine-associated SSPE has increased in the USA. Furthermore patients with B or T-cell immunodeficiencies have cognitive side-effects[27] and are advised against vaccination due to the risk of severe and/or fatal infection (Merck). That viruses persist in the body and are linked to autoimmune disorders is a feature of rubella virus[158–160], anthrax vaccination[161], hepatitis B[162], etc. There is a reported increased risk of death with combined vaccination DPT and polio[134].

In summary, vaccine's side-effects reflect the vaccine's influence upon the body's functional systems i.e. upon temperature, digestion, excretion, blood cell content, etc.

The Cumulative Effect of Vaccines There is concern that the cumulative effect of vaccines upon the body's function has not been properly assessed[137]. Unvaccinated children appear to have less exposure to disease[84,85], delaying vaccination reduces exposure to disease[163], contracting the disease naturally leads to less disease in future[164], and that excessive vaccination is considered ineffective and dangerous[165].

Vaccine-vaccine and Vaccine-drug interactions In general, vaccines may be influenced by antibiotics[166], immunoglobulins, immunosuppressants, monoclonal antibodies, anticoagulants and corticosteroids. The interaction between a vaccine and a drug has been reported only with influenza vaccine and four drugs (aminopyrine, phenytoin sodium, theophylline, and warfarin sodium), and with BCG vaccine and theophylline. The clinical significance of vaccine-drug interactions is not fully determined[167]. There is further evidence of interactions involving most vaccines e.g. HPV Vaccine: (http://hpv.emedtv.com/hpv-vaccine/drug-interactions-with-the-hpv-vaccine.html); Shingles Vaccine: An Introduction: (http://senior-health.emedtv.com/shingles-vaccine/drug-interactions-with-the-shingles-vaccine.html); yellow fever vaccine; polio vaccine (neomycin, streptomycin, phenoxy ethanol, formaldehyde), rotavirus vaccine, etc.

Vaccines are not subject to double blind clinical trials despite the evidence of vaccine-drug interactions and perhaps also of vaccine-vaccine interactions.

Effectiveness of Vaccines/Vaccines are not 100% effective Whooping cough is becoming increasingly prevalent[168–170]. Although claimed to be 88 per cent effective among children of 7-18 months, during a nationwide epidemic of whooping cough in 1993, a group of researchers discovered that 82 per cent had completed their full complement of DPT vaccines[171]. Others have commented that the whooping cough vaccine is only to be 36% effective[109].

Many studies show that the measles vaccine isn't completely effective[172–175] and that a significant proportion of those infected in measles outbreaks (>60%) had been vaccinated. There is also a lack of consensus concerning the effectiveness of whole or acellular vaccines, each having their own side-effects and effectiveness[176] e.g. vaccine efficacy was estimated at 75.4% for an acellular 5 component vaccine, 42.4% for an acellular two component vaccine and 28% for a whole cell DTP vaccine[177]. The whole-cell vaccine was associated with different levels of side-effects including significantly higher rates of crying, cyanosis, fever, and local reactions than the other three vaccines.

There is evidence of declining vaccine immunity[178] illustrated by transmission of mumps[179], measles[180,181], rubella[182], polio[183], Hib[184,185], Hepatitis B[186,187], smallpox, diphtheria, varicella[188], whooping cough[189], etc.

Effect upon Learning One in 14 children i.e. up to half of all children starting school, have problems with speech, language and communication[190]. Is this significant bearing in mind[4] that the occurrence of autism may be more widely spread than has hitherto been considered possible i.e. that only the most severe and chronic cases of autism are recorded? Learning problems are a significant problem in autism[191]. It affects the body's processing of data from the external and internal environments. This affects, in the autistic, the ability of the autonomic nervous system to regulate organ function and hence influences their ability to make sense of the external world. The problem may be part of a spectrum of biochemical disorders[60] influencing all aspects of the learning process e.g. including memory, concentration, sense perception and sense coordination.

Biochemical Evidence

Biochemical Instability Indications of almost complete physiological instability are manifest in the autistic as a proliferation of biochemical deficiencies e.g. (1) Fatty acid deficiency[192]; (2) a distinctly different immune response[62] including reduced natural killer cell activity[193], decreased immunoglobulins and T cells and altered lymphocyte functions[194,195–197], (3) Vitamin D deficiency[198]. Vitamin D regulates the levels of glutathione which may explain the link between heavy metals and autism. Depleted levels of glutathione increase oxidative stress, suppress the detoxifying effect of liver enzymes e.g. P450, reduce the elimination of heavy metals, and increase the neurodegenerative effects of heavy metals. Mercury inhibits the enzyme methionine synthase which converts homocysteine into methionine. Accordingly, levels of cysteine, glutathione and metallothionine are low. This illustrates that the methionine pathway may be faulty in many with autism and supports earlier suggestions that redox imbalances[199–200] and detoxification are impaired. (4) Vitamin A deficiency[201–202] is a commonly observed symptom of measles. The severity of complications have been linked to the degree of Vitamin A deficiency; (5) Carnitine deficiency[203]; (6) increased norepinephrine levels and decreased dopamine-hydroxylase activity[204]; (7) demonstration of inter- and intra- species differences in serotonin binding sites by antibodies from an autistic child[205]; (8) the levels of gut flora[206]; (9) Enterocolitis in Children with Developmental Disorders[207]; (10) Adenosine Deaminase Activity Decreased in Autism[208,209]; (11) Small intestinal enteropathy with epithelial, IgG and complement deposition in children with regressive autism[210]; (12) Mitochondrial disorder[211]. Findings suggest that mitochondrial dysfunction, including abnormal enzyme function, mitochondrial structure, and mitochondrial DNA integrity, may be present in children with autism[212].

Other biochemical deficiencies/chromosomal abnormalities include:

Phosphoribosylpyrophosphate (PRPP) synthetase superactivity, Adenylosuccinate lyase deficiency, Histidinemia, Lesch-Nyhan disease, Fragile X syndrome, Rett Syndrome, Dihydropyrimidine dehydrogenase (DPD) deficiency, Tuberous sclerosis, Superactivity of pyrimidine 5'-nucleotidase (P5N), etc.

The use of Drugs Biochemical instability is a feature of autism. Accordingly, drugs are used to mitigate autistic symptoms e.g. (1) Lofexidine[213] has been shown to improve prefrontal cortical function in nonhuman primates. This is consistent with the view that the prefrontal cortex regulates executive/system function. (2) An open trial[214] suggested that methylphenidate use in autistic hyperactive children may ameliorate hyperactivity, and impulsivity in autistic children. (3) Neuroleptics e.g. haloperidol, are mildly effective in reducing hyperactivity, impulsivity, and inattention in children with autistic disorder[215]; clonidine is used in the treatment of tic disorders and ADHD[216]. Other drugs used include Tianeptine[217]; Galanthamine[218]; Immunoglobulins[219]; melatonin[220]; and beta-blockers[221].

The Cause of Autism The occurrence of autism is due to a significant genetic insult[222] but it is not considered to be an inheritable condition. How and when this occurs can be debated however, for a young child with a developing immune system, there are few factors which could be held responsible other than vaccines and/or the related and damaging effect of exposure to high levels of mercury. No other factor or explanation has been offered as a viable alternative explanation for the occurrence of regressive autism. The evidence indicates there is alteration to chromosome structure and/or function. It indicates the influence of external stressor(s) influencing mitochondrial structure and DNA, chromosomal instability and translocation, which ultimately influences protein expression. The combined effect influences system stability, organ function, the prevailing levels of biochemistry, sense perception, behavior, etc. It influences

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protein expression and the rate and completeness of subsequent protein-substrate reactions leading to lowered immune function, reduced absorption of nutrients, slowed metabolism, impaired development[262], etc; i.e. the body's biochemical processes do not proceed as they should.

Is this an indication of chromosomal damage?

Viruses are able to infiltrate cells, inserting their genetic material into them. As outlined earlier (see 4.1) there are biochemical markers of vaccine damage. That it affects four boys to every girl[10] illustrates that the condition is largely due to a defect with the X-chromosome and leads to consideration of the factors which could influence at the genetic/chromosomal level. In general, chromosomal damage is linked to radiation e.g. due to adverse nuclear events which leads ultimately to birth defects. The prevailing evidence appears to suggest the influence of e.g. proteolytic enzymes or temperature[223,224] which may alter chromosome structure. Little evidence has been offered for the 1 in 5 occurrence experienced by girls although this appears likely to be the consequence of a chromosomal stressor.

It is widely recognised that genetic predisposition and protein expression can be influenced by environment influences[7], and that genetic damage can be the result of exposure to radiation, however the evidence being offered appears to suggest a subtle form of genetic alteration - associated with the wider use of vaccines[17] - which may not necessarily be inherited but is responsible for altered system stability and function and consequently of altered biochemistry and function. There is evidence that system function is intact but dysfunctional i.e. that homeostasis is severely compromised. Such findings are supported by research into Gulf-War Syndrome (GWS) in which[225] untypical RNA was found in the blood of sick GW veterans. This illustrates that the viral encephalopathies originated from RNA-viruses and hence from vaccines. That immunosuppression, shown to be a factor in GWS[226] and autism, is associated with the concentrated use of vaccines[227] is further supported by the fact that French soldiers who were not vaccinated yet who served in the gulf war did not get GWS however American and British soldiers[228], irrespective of whether they served in Iraq or not, reported a significantly greater incidence of autistic-spectrum disorders and GWS.

The Effect of Heavy Metals

Heavy Metals and Mercury in particular, affects the function of the CNS and are extensively documented and associated with autism[229]. Amongst a variety of side-effects mercury decreases lymphocyte viability, and in the brain: dysfunction in the amygdala, hippocampus, basal ganglia, and cerebral cortex; destruction of neurons in the cerebellum; and brainstem abnormalities. Demyelination is evident in such conditions. The brain's electrical patterns are similarly abnormal.

The most significant contributors to the increased mercury burden are: Mercury in vaccines (e.g. DTP (at typically 25 micrograms of mercury per dose), Tetanus, Hepatitis B & (most) influenza vaccines), contamination of fish[230], wild/bush fires; and emissions from power stations[231] and industrial chimneys including incinerators, waste-burning cement works, crematoria, etc. The characteristics of autism and mercury poisoning are extremely similar which suggests that autism arises from mercury poisoning[232,233]. Children with autism have greater amounts of mercury and other heavy metals in their system[234]. For these children the exposure route is considered to be predominately via childhood vaccines, most of which contain thimerosal. Vaccinated children of circa 10-20 kgs are exposed to an adult overdose of mercury, over 62.5 micrograms of mercury within the first three months, which significantly increases a child's risk of developing some form of neuro-developmental disorder such as impaired development, speech and language, autism, stuttering and attention deficit disorder.

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Children living downstream of coal-fired power stations have a greater incidence of autistic spectrum disorders[231]. This indicates that the innate physiological processes, which the body uses to eliminate heavy metals, are being overcome by overexposure.

Mercury poisoning is an insidious process. In general the symptoms do not appear immediately upon exposure, although they may in especially sensitive individuals or in cases of excessive exposure. The initial preclinical stage is followed by the development of symptoms of mercury poisoning over a period which may last from weeks, months, and years[235–237]. Consequently, mercury given in vaccines to very young children would not be expected to lead to a recognizable disorder, except for subtle signs, before age 6-12 months, and might not emerge for several years[233].

In autistic children, the initial signs occur shortly after the first injections, and consist of abnormalities in motor behavior and in the sensory systems, particularly touch sensitivity, vision, and numbness in the mouth[15,238]. These signs are followed by parental reports of speech and hearing abnormalities appearing before the child's second birthday[10]. Finally, there is the development of autistic-like traits and a continuing regression or lack of development in subsequent years. These symptoms change[239] depending upon the circumstances surrounding each child.

Most autistic children have impaired liver detoxification. Many have low levels of metallothionine, conceivably the consequence of a deficiency of Zinc, which is indicative of a lowered capacity to chelate mercury and other heavy metals. Mercury is a powerful oxidant which depletes cellular antioxidants, especially glutathione. The P450 detoxifying enzymes of the liver rely heavily on adequate availability of glutathione. EthylMercury the active component in thimerosal causes apoptosis of the t-cells[240–242].

Although the withdrawal of mercury from vaccines has not resulted in an overall decline in the occurrence of autism this does not mean that the problem does not lie with thimerosal[243,263]. It may indicate that the problem is associated with the elimination of mercury[244] i.e. affecting function of the lymphatic system and excretion[245]. This is supported by noting evidence of urea cycle dysfunction. Problems with the urea cycle, conceivably the consequence of mercury poisoning, have been linked to autism. A child with ornithine transcarbamylase (OTC) deficiency is likely to be lacking in energy, have appetite problems, poorly-controlled breathing rate and/or body temperature, and slow development. Significantly, OTC deficiency is an X-linked recessive disorder (http://www.merck.com/mmpe/sec13/ch164/ch164a.html) one of a number of primary immunodeficiencies associated with vaccine use.

As in autism, onset of Hg toxicity symptoms is gradual in some cases, sudden in others[232,233]. In the case of poisoning, the first signs to emerge are abnormal sensation and motor disturbances. As exposure increases, these signs are followed by speech problems, and hearing deficits[246]. Upon removal of the mercury the symptoms tend to recede except in instances of severe poisoning, which may lead to death[232]. As in autism, epilepsy arising from Hg exposure is also associated with a poor prognosis[247]. Mercury acts upon the catecholamines and influences the function of the autonomic nervous system[245]. This affects cognitive performance[248], spatial vision[249], etc.

Other metals have been implicated in adverse neurodevelopmental outcomes in children e.g. lead and mercury[250,251], with exposure to cadmium, arsenic, antimony and chromium also a concern. Studies have found adverse effects of prenatal lead exposure on growth and development, but little research has examined an association with autism. Whilst Mercury is of concern, because of evidence for neurotoxic effects and the fact that it has become so prevalent in the wider environment[250], Aluminum also shares common mechanisms with mercury e.g. it interferes with cellular and metabolic processes in the nervous

system. Children given the recommended vaccinations are injected with nearly 5 mg of aluminum by the time they are just 1.5 years old, almost 6 times the safe level. Furthermore the nature of the Aluminium affects the prevailing blood levels and is also increasingly implicated, through their use as vaccine adjuvants, in autism[252].

Current Therapeutic Approaches used to Treat Autism

There is evidence that autism is a treatable disease and that some therapies can mitigate the effects of autism[253,254]. Although there is no recognised method of treatment, or of significant and/or proven outcomes, autistic children appear to respond to therapies which enhance the function of the breathing , to enhance oxygen levels[255], and excretory system e.g. by osteopathy[256]. Moreover a commonly observed side-effect with autistic children is that when a child has an elevated temperature, perhaps resulting from a fever, the autistic symptoms appear to recede and the child behaves normally[41]. Autistic children suffer from adverse sleep patterns. In the US autistic children are often treated by chelation therapy and biofeedback[257–259].

Dysfunction of the Excretory or lymphatic system leads to long-term exposure to mercury which under normal circumstances would have been rapidly eliminated from the body. This may also lead to higher neural temperatures which will inevitably influence brain function.

Further evidence of biochemical deficits[260] and of the benefit of biochemical based supplements e.g. vitamin B6 and magnesium; melatonin; methylcobalamin; vitamin A, C & D supplements; dimethylglycine (DMG) and trimethylglycine (TMG). DMG provides building blocks that are required for purine nucleotide synthesis. DMG comes from TMG when TMG methylates homocysteine. Significantly, absorption of Vitamin A Palmitate requires an intact gut mucosa at the appropriate pH and in the presence of bile for metabolism. Many autistic children have damaged mucosal surfaces therefore they have impaired capacity to absorb vitamin A[261].

That some children can become normal when their temperature increases above normal levels e.g. due to a viral infection,[41] may illustrate that the levels of the homeostatic mechanism affecting the physiological systems have been reset at what can be considered to be abnormal levels[47]. This may indicate that autism is treatable - perhaps to a greater degree than has hitherto been considered possible.

Discussion

The mass of scientific evidence compiled by researchers clearly indicates that the incidence of autism occurs following vaccination and is most closely associated with the schedule of vaccines culminating in the MMR vaccine. That vaccines suppress natural immune function is not in dispute e.g. those with naturally low levels of immune function (immigrants from tropical climates) show greater predisposition to autistic spectrum disorders.

The immediate effect arising from vaccination influences gene function and protein expression. This leads to lower levels of white blood cells including e.g. lymphocytes, immunoglobulins, t-cells, b-cells and/or neutrophils, and disturbs their synergistic action and hence their ability to memorize and respond to immune responses when challenged. This impairs the ability to kill pathogens thereby predisposing to further infections. The short and long-term outcome is to the neural mechanisms regulating system function affecting e.g. pH, the excretory system, temperature, and the elimination of toxins and heavy metals. This explains why the discontinuation of thimerosal in vaccines was followed by a steady increase in the incidence of autism and hence that researchers did not find a correlation between the incidence of

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autism and the use of thimerosal-containing vaccines[263]. This may also explain the effect of multiple vaccines, in particular the MMR vaccine, and the greater predisposition to autistic spectrum disorders in military families.

In most autistic children brain structures are initially unaffected but become steadily underdeveloped as a consequence of exposure to mercury and other heavy metals. This evolves into a neurodevelopmental problem leading to chromosomal abnormalities, affecting myelination, the subsequent degeneration of the cerebellum, etc.

The MMR triple vaccine may inhibit normal immune function which, directly or indirectly, ultimately leads to chromosomal and/or genetic damage and/or dysfunction. The occurrence of GWS in adults, a condition with many features which are common with autism, indicates the problem may be due to the number and/or intense schedule of vaccinations however this does not excuse the measles or MMR vaccine from suspicion. The combined vaccine raises body temperature whilst lowering immune and system function. This may make a mild measles vaccine more virulent which may increase fever to an abnormally high level. It suggests (1) single vaccines may pose less risk than triple vaccines; (2) some vaccines pose a greater risk than others e.g. pertussis and measles; and (3) the way in which vaccines are administered will be accompanied by different side-effects e.g. if pertussis is followed by measles or vice-versa, if BCG gives a beneficial effect to be followed by pertussis, if vaccines are given in combination, etc. Increased disease loading is the inevitable consequence of multiple vaccine or lots of single vaccines or triple vaccines e.g. of asthma, autoimmune disease, etc. It suggests that adherence to the vaccine schedule is the problem – too many vaccines, too quickly.

Vaccines cause an inflammatory response in some e.g. for those with an inadequately developed or artificially lowered immune system, for those genetically predisposed, or perhaps due to viral or bacterial infection. This creates genetic damage and/or dysfunction and hence influences the brain's ability to regulate the physiological systems, and especially to the lymphatic system and its ability to excrete mercury and heavy metals, would lead to long-term damage and problems processing sensory/cognitive input. This would inevitably affect the brain's ability to maintain a regulated temperature below that which affects brain damage (41° C). This inevitably influences the autonomic nervous system and the stability of all related physiological systems including temperature, blood pressure, blood cell content, blood glucose, digestion, excretion, sleeping, etc.

Further evidence of multi-level dysfunction is evident from unusual brain-wave stability, aberrant sleep patterns, loss of sense perception and coordination, mirror neuron dysfunction, lower pain thresholds, mental and physical deterioration, short periods of concentration, etc. That it is a problem of systemic dysfunction is further supported by noting how it can be treated using sensory therapies which may facilitate the re-establishment of some degree of physiological stability.

Where is the proof that vaccines are safe? The argument has never been that they are completely safe but that the consequences are less than having the disease. Now it is illustrated that the consequences of intensive vaccination schedules pose a greater risk than could ever have been imagined. This leads to the evolution of new viral strains, an unsurprising development when the environment to which it is exposed is being altered by new proteins, structural variants and altered DNA.

Vaccines are an essential component of preventative healthcare however it may be necessary to review the ways in which vaccines are used, administered and regulated[141,264] i.e.

- As drugs are tested in the clinical environment to assess their interaction with other drugs, the cumulative use of vaccines including that of multiple vaccines should be researched and shown, through double-blind placebo controlled clinical trials, to be free from any such interactions i.e. of one single vaccine with another single or multiple vaccine or drug. It has been considered unethical to select a control group of children which would otherwise not be vaccinated yet such is the levels of conscientious objectors in the industrialized world and through circumstances of impoverishment in the underdeveloped countries that such statistics must currently exist.
- Measures to assess the suitability of children for vaccination i.e. how to assess whether a child has a greater predisposition to an adverse vaccine reaction and the subsequent development of autism? [265]
- The time when vaccinations should be given and the time between vaccinations e.g. giving mumps and rubella vaccinations later in childhood.
- Are some vaccines necessary in the industrialized world e.g. mumps, rubella, Hib, Hpv, etc? With more than 200 other vaccines under development this must be an issue of review.

The risks from disease and vaccinations differ upon location. In the developed world, there is an estimated 0.1-0.3% risk of mortality from measles which compares with a 0.6% risk and rising (with some estimates at 1-2%) of autism. This excludes the cost of treating the wide range of side-effects which must clearly be attributed to the use of vaccines. The cost of treating vaccine-related side-effects may now be far greater than the diseases against which the vaccine(s) were designed to protect. Furthermore, in the developed world there is a highly developed social structure which is able to assist parents to deal with the condition. By comparison, what are the implications for an autistic child in the developing world where there is absence of resources to deal with the condition?

Statement of Interest

Graham and Elena Ewing (Dr) are Directors of Montague Healthcare a company devoted to the commercialisation of Virtual Scanning and hence to the diagnostic and therapeutic use of Virtual Scanning. They are co-authors of the book ‘Virtual Scanning – a new generation of healthcare – beyond biomedicine?’ ISBN 978-0-9556213-0-7 published by Montague Healthcare books.

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References

1. Rimland B. The autism increase: research is needed on the vaccine connection. Autism Research Review. 2000;14(1):3–6.

2. Barnard J, Broach S, Potter D, Prior A. Autism in Schools: Crisis or Challenge? National Autistic Society. 2002. http://www.autism.org.uk/content/1/c4/29/23/aawesn_ew02.pdf .

3. Baird G. Prevalence of disorders of the autism spectrum in a population cohort of children in South Thames: the Special Needs and Autism Project (SNAP) The Lancet. 2006;368(9531):210–5. [PubMed: 16844490]

4. Skuse DH, Mandy W, Steer C, Miller LL, Goodman R, Lawrence K, Emond A, Golding J. Social Communication Competence and Functional Adaptation in a General Population of Children: Preliminary Evidence for Sex-by-Verbal IQ Differential Risk. J.Amer Acad of Child and Adolescent Psychiatry. 2009;48(2):128–137. [PubMed: 19106766]

5. Altman DG, Bland JM. Absence of evidence is not evidence of absence. British Medical Journal. 1995;311:485. [PMCID: PMC2550545] [PubMed: 7647644]

6. O'Callaghan FJ. Autism—what is it and where does it come from? Q J Med. 2002;95:263–265. [PubMed: 11978896]

7. Lander E. http:// www. pbs.org/ wgbh/ nova/ genome/deco_lander.html .

8. Gottlieb S. US study shows 10-fold increase in autism over the past 20 years. British Medical Journal. 2003;326:71.

9. Hertz-Picciotto I, Delwiche L. The Rise in Autism and the Role of Age at Diagnosis. Epidemiology. 2009;20(1):84–90. [PMCID: PMC4113600] [PubMed: 19234401]

10. Gillberg C, Coleman M. The Biology of the Autistic Syndromes - 2nd Edition, page 90. Mac Keith Press. 1992

11. Olmsted D. http://www.nomercury.org/science/documents/Articles/UPI-The_Age_of_Autism-Mercury_and_the_Amish_5-21-05.pdf. http://www.whale.to/vaccine/olmsted.html. http://www.upi.com/Consumer_Health_Daily/Reports/2006/07/28/the_age_of_autism_amish_bill_introduced/3532/ http://pittsburgh.indymedia.org/news/2005/06/18948.php .

12. Barnevik-Olsson M, Gillberg C, Fernell E. Prevalence of autism in children born to Somali parents living in Sweden: a brief report. Dev Med Child Neurol. 2008;50(8):598–601. [PubMed: 18754897]

13. Bailey A, Phillips W, Rutter M. Autism: Towards an Integration of Clinical, Genetic, Neuro-psychological, and Neurobiological Perspectives. J.Child Psychol Psychiatry. 1996;37(1):89–126. [PubMed: 8655659]

14. Filipek P, Accardo P, Baranek G, Cook E, Dawson G, Gordon B, Gravel J, Johnson C, Kallen R, Levy S, Minshew N, Prizant B, Rapin I, Rogers S, Stone W, Teplin S, Tuchman R, Volkmar F. The Screening and Diagnosis of Autistic Spectrum Disorders. Journal of Autism and Developmental Disorders. 1999;29(6):439–484. [PubMed: 10638459]

15. Baranek G. Autism During Infancy: A Retrospective Video Analysis of Sensory-Motor and Social Behaviors and 9-12 Months of Age. Journal of Autism and Developmental Disorders. 1999;29(3):213–224. [PubMed: 10425584]

16. Lewine JD, Andrews R, Chez M, Patil A-A, Devinsky O, Smith M, Kanner A, Davis JT, Funke M, Jones G, Chong B, Provencal S, Weisend M, Lee RR, Orrison WW. Magnetoenchalography in Children with an Autistic Epileptiform Regression. J Pediatrics. 1999:405–418. [PubMed: 10469763]

17. Montinari M, Favoino B, Roberto A. Role of Immunogenetics in the Diagnosis of Postvaccinal CNS Pathology. Presented in Naples. Associazione per la Libera Universita Internazionale de Medicina Omeopatica "Samuel Hahnemann" (LUIMO) 1996 May 9; http://www.healthy.net/library/articles/coulter/biochem.htm .

18. Furlano RI, Anthony A, Day R, Brown A, McGarvey L, Thomson MA, Davies SE, Berelowitz M, Forbes A, Wakefield AJ, Walker-Smith JA, Murch SH. Colonic CD8 and gamma delta T-cell infiltration with epithelial damage in children with autism. J Pediatr. 2001;138(3):366–72. [PubMed: 11241044]

19. Kurita H. Infantile autism with speech loss before the age of thirty months. Journal of the American Academy of Child Psychiatry. 1985;24(2):191–196. [PubMed: 3989162]

20. Jarbrink K, Knapp M. The Economic Impact of Autism in Britain. Autism. 2001;5(1):7–22. [PubMed: 11708392]

21. Ewing GW, Ewing EN. Cognition, the Autonomic Nervous System and the Physiological Systems. J. Biogenic Amines. 2008;22(3):140–163.

22. Barbur JL. Trends in Cognitive Sciences Understanding colour: Normal and Defective Colour Vision edited by J.D. 2003;7(10):434–436. Understanding colour: Normal and Defective Colour Vision edited by J.D. Mollon, J. Pokorny and K. Knoblauch, ISBN 0-19-852530-3.

23. Muntoni S, Serra A, Mascia C, Songini M. Dyschromatopsia in diabetes mellitus and its relation to metabolic control. Diabetes Care. 1982;5(5):375–378. [PubMed: 7151653]

24. Lloyd MJ, Fraunfelder FW. Drug-induced optic neuropathies. Drugs Today. 2007;43(11):827. [PubMed: 18174968]

25. Martinek K, Berezin I. Artificial Light-Sensitive Enzymatic Systems as Chemical Amplifiers of Weak Light Signals. Photochemistry and Photobiology. 1979;29:637–649. [PubMed: 375252]

26. Azeemi STY, Raza SM, Yasinzai M. Colors as Catalysts in Enzymatic Reactions. J.Acupuncture and Meridian Studies. 2008;1(2):139–142. [PubMed: 20633466]

27. Kipnis J, Cohen H, Cardon M, Ziv Y, and Schwartz M. T cell deficiency leads to cognitive dysfunction: Implications for therapeutic vaccination for schizophrenia and other psychiatric conditions. Proc Natl Acad Sci U S A. 2004;101(21):8180–8185. [PMCID: PMC419577] [PubMed: 15141078]

28. Grakov I. Strannik Diagnostic and Treatment System; a Virtual Scanner for the Health Service. Minutes of Meeting No. 11 of the Presidium of the Siberian of the Academy of Medical Sciences of the USSR (AMN) 14, held in Novosibirsk. 1985 Dec 4;

29. Grakov I. Mimex, Sochi, Russia: 2002. Description of Virtual Scanning System for Operators. English translation available at: http://www.montague-diagnostics.co.uk/files/Grakov/Article7.pdf .

30. Cox RH, Shealy CN, Cady RK, Liss S. The Journal of Neurological and Orthopaedic Medicine and Surgery. 1996;17:32–34.

31. Bower B. Perception may dance to the beat of collective neuronal rhythms. Science News. 1998;153(8):120.

32. Milne E, Scope A, Pascalis O, Buckley D, Makeig S. Independent Component Analysis Reveals Atypical Electroencephalographic Activity during Visual Perception in Individuals with Autism. Biological Psychiatry. 2009;65(1):22–30. [PubMed: 18774554]

33. Varela F.J. http://findarticles.com/p/articles/mi_m1200/is_8_155/ai_54062666 .

34. Baron-Cohen S. Is There a Normal Phase of Synaesthesia in Development? Psyche. 1996;2(27)

35. Baron-Cohen S, Wyke M, Binnie C. Hearing words and seeing colours: an experimental investigation of a case of synaesthesia. Perception. 1987;16:761–67. [PubMed: 3454433]

36. Baron-Cohen S, Harrison J, Goldstein L, Wyke M. Coloured speech perception: Is synaesthesia what happens when modularity breaks down? Perception. 1993;22:419–426. [PubMed: 8378132]

37. Miltner WHR. Learning fosters synchronized neural activity. http://findarticles.com/p/articles/mi_m1200/is_8_155/ai_54062666 .

38. Hardan AY, Keshavan MS, Sucheeta S, Velumapalli M, Minshew NJ. An MRI study of minor physical anomalies in autism. J Autism Dev Disord. 2006;36:607–611. [PubMed: 16609827]

39. Minshew NJ, Williams DL. The New Neurobiology of Autism: Cortex, Connectivity, and Neuronal Organization. Arch Neurol. 2007;64(7):945–950. [PMCID: PMC2597785] [PubMed: 17620483]

40. Kennedy DP, Courchesne E. The intrinsic functional organization of the brain is altered in autism. Neuroimage. 2008;39(4):1877–85. [PubMed: 18083565]

41. Curran LK, Newschaffer CJ, Lee L-C, Crawford SO, Johnston MV, Zimmerman AW. Behaviors Associated With Fever in Children With Autism Spectrum Disorders. Pediatrics. 2007;120(6):e1386–e1392. [PubMed: 18055656]

42. Courchesne E. New evidence of cerebellar and brainstem hypoplasia in autistic infants, children, and adolescents: The MRI imaging study by Hashimoto and colleagues. Journal of Autism and Developmental Disorders. 1995;25:19–22. [PubMed: 7608031]

43. Bower JM, Parsons L. Rethinking the Lesser Brain. Scientific American. 2003;289:50–57. [PubMed: 12884538]

44. Ritvo ER, Freeman BJ, Scheibel AB, Duong T, Robinson H, Guthrie D, Ritvo A. Lower Purkinje cell counts in the cerebella of four autistic subjects: intitial findings of the UCLA-NSAC Autopsy Research Report, American Journal of Psychiatry. 1986;143:862–866. [PubMed: 3717426]

45. Hashimoto T, Tayama M, Miyazaki M, Sakurama N, Yoshimoto T, Murakawa K, Kuroda Y. Reduced brainstem size in children with autism. Brain & Development. 1992;14(2):94–97. [PubMed: 1621932]

46. Hashimoto T, Tayama M, Murakawa K, Yoshimoto T, Miyazaki M, Harada M, Kuroda Y. Development of the brainstem and cerebellum in autistic patients. Journal of Autism and Developmental Disorders. 1995;25(1):1–18. [PubMed: 7608030]

47. Ewing GW, Ewing EN. 'Virtual Scanning – a new generation of medical technology – beyond biomedicine?' ISBN 978-0-9556213-0-7 pub Montague Healthcare books.

48. Krakov SV. Colour Vision and the Autonomic Nervous System. Journal of the Optical Society of America. J. Opt. Soc. Am. 1941;31:335–337.

49. Cumberland P, Rahi J.S, Peckham CS. Impact of congenital colour vision on education and unintentional injuries: findings from the 1958 British Birth Cohort. British Medical Journal. 2004;329:1074–5. [PMCID: PMC526118] [PubMed: 15465847]

50. Van De Geijn EJ, Tukkie R, Van Philips LAM, Punt H. Bilateral optic neuritis with branch retinal artery occlusion associated with vaccination. Documenta Ophthalmologica. 1994;86(4):403–408. [PubMed: 7835178]

51. Sigman M, Ungerer JA, Mundy P, Sherman T. Cognition in Autistic Children.Handbook of Autism and Pervasive Developmental Disorders, John Wiley & Sons, Inc. 1987:103–130.

52. O'Neill M, Jones RSP. Sensory-Perceptual Abnormalities in Autism: A Case For More Research? Journal of Autism and Developmental Disorders. 1997;27(3):283–293. [PubMed: 9229259]

53. Takayanagi Y, Yoshida M, Bielsky IF, Ross HE, Kawamata M, Onaka T, Yanagisawa T, Kimura T, Matzuk MM, Young LJ, Nishimori K. Pervasive social deficits, but normal parturition, in oxytocin receptor-deficient mice. Proc Natl Acad Sci USA. 2005;102:16096–101. [PMCID: PMC1276060] [PubMed: 16249339]

54. Bartz JA, Hollander E. Oxytocin and experimental therapeutics in autism spectrum disorders. Prog Brain Res. 2008;170:451–62. [PubMed: 18655901]

55. Zak PJ, Stanton AA, Ahmadi A. Oxytocin increases generosity in humans. PLoS ONE. 2007;2(11):e1128. [PMCID: PMC2040517] [PubMed: 17987115]

56. Panksepp J. Commentary on the possible role of oxytocin in autism. Journal of Autism and Developmental Disorders. 1993;23(3):567–569. [PubMed: 8226589]

57. Porges SW. The Polyvagal Theory: phylogenetic contributions to social *behaviour*. Physiol Behav. 2003;79:503–513. [PubMed: 12954445]

58. Ewing GW, Parvez SH. 2009. Systemic Regulation of Metabolic Function. Approved for publication in Biogenic Amines. 2008;22(6):179–194.

59. Ewing GW, Ewing EN. 2009. NeuroRegulation of the Physiological Systems by the Autonomic Nervous System - their relationship to Insulin Resistance and Metabolic Syndrome.Approved for publication in. J. Biogenic Amines. 2008;22(4-5):99–130.

60. Ewing GW, Parvez SH. 2009. The Regulatory Significance of the Autonomic Nervous System and the Physiological Systems, and their relationship to Dyslexia. Approved for publication in Biogenic Amines. 2009 Jul

61. Ming X, Julu POO, Brimacombe M, Connor S, Daniels ML. Reduced cardiac parasympathetic activity in children with autism. Brain and Development. 2005;27(7):509–516. [PubMed: 16198209]

62. Ashwood P, Van de Water JA. A review of autism and the immune response. Clin Develop Immunology. 2004;11(2):165–174. [PMCID: PMC2270714] [PubMed: 15330453]

63. Zahn T.P, Rumsey J.M, Van Kammen D.P. Autonomic nervous system activity in autistic, schizophrenic, and normal men: Effects of stimulus significance. Journal of Abnormal Psychology. 1987;96(2):135–144. [PubMed: 3584662]

64. Hutt C, Forrest SJ, Richer J. Cardiac Arrhythmia and Behaviour in Autistic Children. Acta Psychiatrica Scandinavica. 2007;51(5):361–372. [PubMed: 1146592]

65. Stores G, Wiggs L. Abnormal sleeping patterns associated with autism: a brief review of research findings, assessment methods and treatment strategies. Autism. 1998;2(2):157–170.

66. Williams PG, Sears LL, Allard A-M. Sleep problems in children with autism. Journal of Sleep research. 2004;13(3):265–268. [PubMed: 15339262]

N Am J Med Sci
N Am J Med Sci

67. Malow BA. Sleep disorders, epilepsy, and autism. Mental Retardation and Developmental Disabilities Research Reviews. 2004;10(2):122–125. [PubMed: 15362168]

68. Chugani DC, Sundram BS, Behen M, Lee ML, Moore GJ. Evidence of altered energy metabolism in autistic children. Prog Neuropsychopharmacol Biol Psychiatry. 1999;23(4):635–41. [PubMed: 10390722]

69. Ohnishi T, Matsuda H, Hashimoto T, Kunihiro T, Nishikawa M, Uema T, Sasaki M. Abnormal blood flow in brain regions. Brain. 2000;123:1838–44. [PubMed: 10960047]

70. Starkstein SE, Vazquez S, Vrancic D D, Nanclares V, Manes F, Piven J, Plebst C. SPECT findings in mentally retarded autistic individuals. J Neuropsychiatry Clin Neurosci. 2000;12(3):370–5. [PubMed: 10956571]

71. Ryu YH, Lee JD, Yoon PH, Kim DI, Lee HB, Shin YJ. Perfusion impairments in infantile autism on technetium-99m ethyl cysteinate dimer brain single-photon emission tomography: comparison with findings on magnetic resonance imaging. Eur J. Nucl Med. 1999;26:253–259. [PubMed: 10079316]

72. Jones W, Carr K, Klin A. Absence of preferential looking to the eyes of approaching adults predicts level of social disability in 2-year-olds with autism. Archives of General Psychiatry. 2008;65(8):946–954. [PubMed: 18678799]

73. Cascio C, McGlone F, Folger S, Tannan V, Baranek G, Pelphrey KA, Essick G. Tactile Perception in Adults with Autism: a Multidimensional Psychophysical Study. Journal of Autism Development Disorders. 2008;38(1):127–137. [PMCID: PMC2185746] [PubMed: 17415630]

74. Rosenhall U, Johansson E, Gillberg C. Oculomotor findings in autistic children. Journal of Laryngology and Otology. 1988;102:435–439. [PubMed: 3397639]

75. Rosenhall U, Nordin V, Sandstrom M, Ahlsen G, Gillberg C. Autism and Hearing Loss. Journal of Autism and Developmental Disorders. 1999;29(5):349–358. [PubMed: 10587881]

76. Paulesu E, Harrison J, Baron-Cohen S, Watson JDG, Goldstein L, Heather J, Frackowiak RSJ, Frith CD. The physiology of coloured hearing A PET activation study of colour-word synaesthesia. Brain. 1995;118:661–676. [PubMed: 7600084]

77. Grice SJ, Spratling MW, Karmiloff-Smith A, Halit H, Csibra G, de Haan M, Johnson MH. Disordered visual processing and oscillatory brain activity in autism and Williams Syndrome. Neuroreport. 2001;12(12):2697–2700. [PubMed: 11522950]

78. Motomi T, Yoko K. Autistic adolescents' autonomic response to mental load. Japanese Journal of Child and Adolescent Psychiatry. 1999;40(4):319–328.

79. Cohen AD, Shoenfeld Y. Vaccine-induced Autoimmunity. Journal of Autoimmunity. 1996;9(6):699–703. [PubMed: 9115571]

80. Howson CP, Katz M, Johnston RB, Fineberg HV. Chronic arthritis after rubella vaccination. Clin. Infectious Disease. 1992;15(2):307–312. [PubMed: 1520764]

81. Howson CP, Fineberg HV. 1992 Adverse events following pertussis and rubella vaccines. JAMA. 1992;267(3):393–397. [PubMed: 1727962]

82. Rook GAW, Stanford JL. Give us this day our daily germs. Immunology Today. 1998;19:113–116. [PubMed: 9540269]

N Am J Med Sci
N Am J Med Sci

83. Taylor-Robinson AW. Multiple vaccination effects on atopy. Allergy. 1999;54:398–399. [PubMed: 10371102]

84. Odent M.R, Culpin E.E, Kimmel T. Pertussis vaccination and asthma: is there a link? JAMA. 1994;272:592–593. [PubMed: 8057511]

85. Kemp T, Pearce N, Fitzharris P, Crane J, Fergusson D, St George I, Wickens K, Beasley R. Is Infant Immunisation a risk factor for childhood asthma or allergy? Epidemiology. 1997;8(6):678–680. [PubMed: 9345669]

86. Hurwitz EL, Morgenstern H. Effects of diphtheria-tetanus-pertussis or tetanus vaccination on allergies and allergy-related respiratory symptoms among children and adolescents in the United States, Journal of Manipulative and Physiological Therapeutics. 2000;23(2):81–90. [PubMed: 10714532]

87. Patel NC, Hertel P, Estes M, Dela Morena M, Noroski L, Revell P, Hanson I, Paul M, Rosenblatt H, Abramson S. Vaccine-acquired rotavirus infection in two infants with severe combined immunodeficiency. American Academy of Allergy, Asthma & Immunology. 2009 Abstract L29.

88. Cherry JD, Brunell PA, Golden GS, Karzon DT. Report on the Task Force on Pertussis and Pertussis Immunization. Pediatrics. 1988;(81) Supplement –.

89. Terpstra GK, Raaijmakers JA, Kreukniet J. Comparison of vaccination of mice and rats with Haemophilus influenzae and Bordetta pertussis as models of atopy. Clin Exp Pharmacol Physiol. 1979;6(2):139–149. [PubMed: 311260]

90. Schreurs AJ, Nijkamp FP. Bronchial hyperreactivity to histamine induced by Haemophilus influenzae vaccination. Agents Actions. 1984;15(3-4):211–215. [PubMed: 6335351]

91. Update: Vaccine Side Effects, Adverse Reactions, Contraindications, and Precautions Recommendations of the Advisory Committee on Immunization Practices (ACIP) MMWR. 1996 Sep 06;45(RR-12):1–35. [PubMed: 8801442]

92. Vertes C, Gonczy S, Lendvay N, Debreczeni LA. A model for experimental asthma provocation in guinea-pigs immunized with Bordetella pertussis. Bull Eur Physiopathol Respir. 1987;23(Suppl 10):111s–113s. [PubMed: 2889487]

93. Schreurs AJ, Terpstra GK, Raaijmakers JA, Nijkamp FP. The effects of Haemophilus influenzae vaccination on anaphylactic mediator release and isoprenaline-induced inhibition of mediator release. Eur J. Pharmacol. 1980;62(4):261–8. [PubMed: 6154589]

94. Bradford Hill A, Knoweldon J. Inoculation and Poliomyelitis. British Medical Journal. 1950:1–6. [PMCID: PMC2038021] [PubMed: 15426789]

95. Imani F, Kehoe KE. Infection of Human B Lymphocytes with MMR Vaccine Induces IgE Class Switching. J. Clinical Immunology. 2001;100(3):355–361. [PubMed: 11513549]

96. Shields RL, Lai J, Keck R, O'Connell L.Y, Hong K, Meng YG, Weikert SHA, Presta LG. Lack of Fucose on Human IgG1 N-Linked Oligosaccharide Improves Binding to Human Fc RIII and Antibody-dependent Cellular Toxicity. J. Biol. Chem. 2002;277(30):26733–26740. [PubMed: 11986321]

97. Kaplan KM, Marder DC, Cochi SL, Preblud SR. Further evidence of the changing epidemiology of a childhood vaccine-preventable disease. Journal of the American Medical Association. 1988;260(10):1434–1438. [PubMed: 3404601]

N Am J Med Sci
N Am J Med Sci

98. Singh VK, Lin SX, Newell E, Nelson C. Abnormal measles-mumps-rubella antibodies and CNS autoimmunity in children with autism. Journal of Biomedical Science. 2002;9:359–364. [PubMed: 12145534]

99. Mooi FR, van Oirschot H, Heuvelman K, van der Heide HG, Gaastra W, Willems RJ. Olymorphism in the Bordella Pertussis virulence factors P.69/pertactin and pertussis toxin in the Netherlands:temporal trends and evidence for vaccine-driven evolution. Infect Immun. 1998;66:670–5. [PMCID: PMC107955] [PubMed: 9453625]

100. Gzyl A, Augustynowicz E, van Loo I, Slusarczyk J. Temporal nucleotide changes in pertactin and pertussis toxin genes in Bordetella pertussis strains isolated from clinical cases in Poland. Vaccine. 2001;20:299–303. [PubMed: 11672891]

101. Ribeiro GS, Reis JN, Cordeiro SM, Lima JB, Gouveia EL, Petersen M, Salgado K, Silva HR, Zanella RC, Almeida SC, Brandileone MC, Reis MG, Ko AI. Prevention of Haemophilus influenzae type b (Hib) meningitis and emergence of serotype replacement with type a strains after introduction of Hib immunization in Brazil. J.Infect Dis. 2003;187(1):109–16. [PubMed: 12508153]

102. Litt DJ, Neal SE, Fry NK. Changes in Genetic Diversity of the Bordetella pertussis Population in the United Kingdom between 1920 and 2006 Reflect Vaccination Coverage and Emergence of a Single Dominant Clonal Type. Journal of Clinical Microbiology. 2009;47(3):680–688. [PMCID: PMC2650949] [PubMed: 19158267]

103. Tsang RS, Sill ML, Skinner SJ, Law DK, Zhou J, Wylie J. Clin Infect Dis. 2007;44(12):1611–4. Characterization of invasive Haemophilus influenzae disease in Manitoba, Canada, 2000-2006: invasive disease due to non-type b strains. [PubMed: 17516405]

104. Nahm MH, Lin J, Finkelstein JA, Pelton SI. Increase in the Prevalence of the Newly Discovered Pneumococcal Serotype 6C in the Nasopharynx after Introduction of Pneumococcal Conjugate Vaccine The Journal of Infectious Diseases. 2009;199:320–325. [PMCID: PMC2743180] [PubMed: 19099489]

105. Guris D, Strebel PM, Bardenheier B, Brennan M, Tachdjian R, Finch E, Wharton M, Livengood JR. Changing epidemiology of pertussis in the United States: increasing reported incidence among adolescents and adults, 1990-1996. Clin Infect Dis. 1999;28:1230–1237. [PubMed: 10451158]

106. Cherry JD. Epidemiological, clinical, and laboratory aspects of pertussis in adults. Clin Infect Dis. 1999;28(Suppl 2):S112–7. [PubMed: 10447028]

107. Huisman W. Vaccine-induced enhancement of viral infections. Vaccine. 2009;27(4):505–512. [PubMed: 19022319]

108. Exley RM, Shaw J, Mowe E, Sun Y-H, West NP, Williamson M, Botto M, Smith H, Tang CM. Available carbon source influences the resistance of Neisseria meningitidis against complement. J Exp Med. 2005;201(10):1637–1645. 16. [PMCID: PMC2212924] [PubMed: 15897277]

109. Stewart GT. Vaccination against whooping-cough.Efficacy versus risks. The Lancet. 1977;1(8005):234–7. [PubMed: 64761]

110. Colville A, Pugh S, Miller E, Schmitt HJ, Just M, Neiss A. Withdrawal of a mumps vaccine. Eur J Pediatr. 1994;153(6):467–8. [PubMed: 8088305]

N Am J Med Sci
N Am J Med Sci

111. Schlegel M, Osterwalder JJ, Galeazzi RL, Vernazza PL. Comparative efficacy of three mumps vaccines during disease outbreak in eastern Switzerland: cohort study. British Medical Journal. 1999;319(7206):352. [PMCID: PMC32261] [PubMed: 10435956]

112. Peltola H, Kulkarni PS, Kapre SV, Paunio M, Jadhav SS, Dhere RM. Mumps outbreaks in Canada and the United States: Time for new thinking on mumps vaccines. Clin Infect Dis. 2007;45:459–66. [PubMed: 17638194]

113. Newsl EPI. Live attenuated measlesvaccine. 1980 Feb;2(1):6. [PubMed: 12314356]

114. Rima B.K, Earle J.A, Yeo R.P, Herlihy L, Baczko K, ter Meulen V, Carabaña J, Caballero M, Celma ML, Fernandez-Muñoz R. Temporal and geographical distribution of measles virus genotypes. J. Gen. Virol. 1995;76(5):1173–80. [PubMed: 7730801]

115. Garenne M, Leroy O, Beau J.P, Sene I. Child mortality after high-titre measles vaccines: prospective study in Senegal. The Lancet. 1991;338(8772):903–7. [PubMed: 1681265]

116. Trollfors B, Taranger J, Lagergard T, Lind L, Sundh V, Zackrisson G, Lowe CU, Blackwelder W, Robbins JB. A placebo-controlled trial of a pertussis-toxoid vaccine. N Engl J Med. 1995;333:1045–50. [PubMed: 7675047]

117. Marwick C. Acellular pertussis vaccine hailed for infants. JAMA. 1995;274:446–467. [PubMed: 7629938]

118. Miller E. Overview of recent clinical trials of acellular pertussis vaccines. Biologicals. 1999;27:79–86. [PubMed: 10600188]

119. Shoenfeld Y, Aron-Maor A. Vaccination and Autoimmunity - ‘vaccinosis’: A Dangerous Liaison? Journal of Autoimmunity. 2000;14(1):1–10. [PubMed: 10648110]

120. Kerrison J. Optic neuritis after anthrax vaccination. Ophthalmology. 2002;109(1):99–104. [PubMed: 11772587]

121. Asatryan A, Pool V, Chen RT, Kohl KS, Davis RL, Iskander JK. Live attenuated measles and mumps viral strain-containing vaccines and hearing loss: Vaccine Adverse Reporting System (VAERS), United States, 1990-2003. Vaccine. 2008;26(8):1166–1172. [PubMed: 18255204]

122. Roizen NJ. Nongenetic causes of hearing loss.Mental Retardation and Developmental Disabilities Research Reviews. 2003;9(2):120–127. [PubMed: 12784230]

123. Pickering LK. Pediatric News. 1998 Nov 28;

124. Marks AR. Physiological systems under pressure. J Clin Invest. 2008;118(2):411–412. [PMCID: PMC2214719] [PubMed: 18246190]

125. Atkinson W, Hamborsky J, McIntyre L, Wolfe S, editors. 10ed. Washington DC: Public Health Foundation; 2007. Epidemiology and Prevention of Vaccine-Preventable Diseases (The Pink Book) pp. 59–70.

126. Gupta N, Puliyel J. WHO study suggests low incidence of Hib in India is due to natural immunity. Indian J Med Res. 2009;129:205–207. [PubMed: 19293451]

127. Perry RT, Halsey NA. The Clinical Significance of Measles: A Review. The Journal of Infectious Diseases. 2004;189(S1):1547–1783.

128. Manson AL. Mumps orchitis. Urology. 1990;36(4):355–8. [PubMed: 2219620]

129. Siegel M, Fuerst HT, Guinee VF. Rubella epidemicity and embryopathy.Results of a long-term prospective study. Am. J. Dis. Child. 1971;121(6):469–73. [PubMed: 5581012]

130. West R. Epidemiologic study of malignancies of the ovaries. Cancer. 1966;19:1001–1007. [PubMed: 5939299]

131. Wynder E, Dodo H, Barber HR. Epidemiology of cancer of the ovary. Cancer. 1969;23:352. [PubMed: 5764976]

132. Newhouse M, Pearson RM, Fullerton JM, Boesen EA, Shannon HS. A case control study of carcinoma of the ovary. Brit J Prev Soc Med. 1977;31:148–53. [PMCID: PMC479015] [PubMed: 588853]

133. McGowan L, Parent L, Lednar W, Norris HJ. The woman at risk from developing ovarian cancer. Gynecol Oncol. 1979;7:325–344. [PubMed: 447120]

134. Kristensen I, Aaby P, Jensen H. Routine vaccinations and child survival: follow-up study in Guinea-Bissau, West Africa. British Medical Journal. 2000;321:1435–9. [PMCID: PMC27544] [PubMed: 11110734]

135. Aaby P, Samb B, Simondon F, Coll Seck AM, Knudsen K, Whittle H. Non-specific beneficial effect of measles immunization: analysis of mortality studies from developing countries. British Medical Journal. 1995;311:481–5. [PMCID: PMC2550544] [PubMed: 7647643]

136. Dalton C, Emerton D, Buckoke C, Finlay R, Engler T, Shann F, Aaby P. Unexpected beneficial effects of measles immunisation. British Medical Journal. 2000;320:938–938.

137. Odent MR. Long term effects of early vaccinations. Primal Health Research. 1994;2(1):6.

138. Polack FP. Why did RSV vaccine make kids sick? Nature Medicine. 2008 Dec 14;

139. Quast U, et al. Vaccine-induced mumps-like diseases. Developments in Biological Standardization. 1979;43:269–272. [PubMed: 520674]

140. Ronchi F, Cecchi P, Falcioni F, Marsciani A, Minak G, Muratori G, Tazzari PL, Beverini S. Thrombocytopenic purpura as adverse reaction to recombinant hepatitis B vaccine. Archives of Disease in Childhood. 1998;78(3):273–274. [PMCID: PMC1717498] [PubMed: 9613364]

141. Tonz O, Bajc S. Convulsions or status epilepticus in 11 infants after pertussis vaccination. Schweiz. Med. Wochenschr. 1980;51:1965–1971. [PubMed: 6792699]

142. Thompson P, et al. Is measles vaccination a risk factor for inflammatory bowel disease? The Lancet. 1995;345:1071–1074. [PubMed: 7715338]

143. Wakefield AJ, Murch SH, Anthony A, Linnell J, Casson DM, Malik M, Berelowitz M, Chillon AP, Thomson MA, Harvey P, Valentine A, Davies SE, Walker-Smith JA. ‘Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children‘ The Lancet. 1998;351:637–641. [PubMed: 9500320]

144. Erickson CA, Stigler KA, Corkins MR, Posey DJ, Fitzgerald JF, McDougle CJ. Gastrointestinal factors in autistic disorder: a critical review. J.Autism Dev Disord. 2005;35(6):713–727. [PubMed: 16267642]

145. Fujinaga T, Motegi Y, Tamura H, Kuroume T. A prefecture-wide survey of mumps meningitis associated with MMR vaccine. Paediatric Infectious Disease Journal (R) 1991 Mar [PubMed: 2041667]

146. Sawada H, Yano S, Oh Y, Togashi T. Transmission of Urabe mumps vaccine between siblings. The Lancet. 1993;342:371. [PubMed: 8101611]

147. Adler JB, Mazzotta SA, Barkin JS. Pancreatitis caused by measles, mumps, and rubella vaccine. Pancreas. 1991;6:489–490. [PubMed: 1876605]

148. Pawlowski B, Gries FA. Mumps vaccination and type-1 diabetes. Deutsche Medizinische Wochenschrift. 1991;116:635. [PubMed: 2015783]

149. Tuomilehto J, Rewers M, Reunanen A, Lounamaa P, Lounamaa R, Tuomilehto-Wolf E, Akerblom HK. Increasing trend in type 1 (insulin-dependent) diabetes mellitus in childhood in Finland.Analysis of age, calendar time and birth cohort effects during 1965 to 1984. Diabetologia. 1991;34(4):282–287. [PubMed: 2065863]

150. Tuomilehto J, Karvonen M, Pitkaniemi J, Virtala E, Kohtamaki K, Toivanen L, Tuomilehto-Wolf E. Record-high incidence of Type I (insulin-dependent) diabetes mellitus in Finnish children. The Finnish Childhood Type I Diabetes Registry Group. Diabetologia. 1999;42(6):655–660. [PubMed: 10382584]

151. Weibel RE, Caserta V, Benor DE, Evans G. 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. 1998;101(3) Part 1. [PubMed: 9481001]

152. Buttram HE. Measles-Mumps-Rubella (MMR) Vaccine as a Potential Cause of Encephalitis (Brain Inflammation) in Children.Townsend Letters. 1997 Dec

153. Laitinen O, Vaheri A. Very high measles and rubella virus antibody titers associated with hepatitis, systemic lupus erythematosus and infectious mononucleosis. The Lancet. 1974;(1):194–7. [PubMed: 4129878]

154. Zecca T, Grafino D. Elevated rubeola titers in autistic children linked to MMR vaccine, abstract submitted to the National Institutes of Health. 1997-8

155. Halsey Increased mortality after high titer measles vaccine. Paediatric Infectious Disease Journal (R) 1993 Jun

156. Bonthius D, Stanek N, Grose C. Subacute sclerosing panencephalitis, a measles complication, in an internationally adopted child. Emerg Infect Dis. 2000;6(4):377–81. [PMCID: PMC2640885] [PubMed: 10905971]

157. Salmi AA, Norrby E, Panelius M. Identification of different measles virus-specific antibodies in the serum and cerebrospinal fluid from patients with subacute sclerosing pancencephalitis and multiple sclerosis. Infect Immun. 1972;6(3):248–254. [PMCID: PMC422523] [PubMed: 4629257]

158. Chantler JK, Tingle AJ, Petty RE. New England Journal of Medicine. 313(18):1117–1123. [PubMed: 4047116]

159. Geier MR, Geier DA. A one year followup of chronic arthritis following rubella and hepatitis B vaccination based upon analysis of the Vaccine Adverse Events Reporting System (VAERS) database. Clin Exp Rheumatol. 2002;20(6):767–71. [PubMed: 12508767]

N Am J Med Sci
N Am J Med Sci

160. Bosma TJ, Etherington J, O'Shea S, Corbett K, Cottam F, Holt L, Banatvala JE, Best JM. Rubella Virus and Chronic Joint Disease: Is There an AssociationJ? J. Clin. Microbiol. 1998;36:3524–3526. [PMCID: PMC105233] [PubMed: 9817866]

161. Geier MR, Geier DA. Anthrax vaccination and joint related adverse reactions in light of biological warfare scenarios. Clin Exp Rheumatol. 2002;20(1):119. [PubMed: 12051402]

162. Geier MR, Geier DA. Arthritic reactions following hepatitis B vaccination: an analysis of the vaccine adverse events reporting system (VAERS) data from 1990 through 1994. Clin Exp Rheumatol. 2000;18(6):789–90. [PubMed: 11138356]

163. McDonald KL, Huq SI, Lix LM, Becker AB, Kozyrskyj AL. Delay in diphtheria, pertussis, tetanus vaccination is associated with a reduced risk of childhood asthma. J Allergy Clin Immunol. 2008;121(3):626–31. [PubMed: 18207561]

164. Ronne T. Measles virus infection without rash in chioldhood is related to disease in adult life. The Lancet. 1985;1(8419):1–5. [PubMed: 2856946]

165. Albonico H, Klein P, Grob C, en Pewsner D. The immunization campaign against measles, mumps and rubella -- coercion leading to a realm of uncertainty: medical objections to a continued MMR immunization campaign in Switzerland. JAMA. 1992;9(1)

166. Strebel PM, Aubert-Combiescu A, Ion-Nedelcu N, Biberi-Moroeanu S, Combiescu M, Sutter RW, Kew OM, Pallansch MA, Patriarca PA, Cochi SL. Paralytic poliomyelitis in Romania, 1984-1992.Evidence for a high risk of vaccine-associated disease and reintroduction of wild-virus infection. Am J Epidemiol. 1994;140(12):1111–24. [PubMed: 7998593]

167. D'Arcy PF. Vaccine-drug interactions. Drug Intelligence & Clinical Pharmacy. 1984;18(9):697–700. [PubMed: 6383754]

168. Wright SW, Decker MD, Edwards KM. Incidence of pertussis infection in healthcare workers. Infect Control Hosp Epidemiol. 1999;20:120–3. [PubMed: 10064216]

169. De Serres G, Bouliane N, Douville Fradet M, Duval B. Pertussis in Quebec: ongoing epidemic since the late 1980s. Can Commun Dis Rep. 1995;21:45–8. [PubMed: 7757050]

170. Andrews R, Herceq A, Roberts C. Pertussis notifications in Australia. Commun Dis Intell. 1997;21:145–8. [PubMed: 9188218]

171. Simon MW. Resurgence of Disease in a Highly Immunized Population of Children. N Engl J Med. 1994;331:16–21. [PubMed: 8202096]

172. Markowitz LE, Preblud SR, Orenstein WA, Rovira EZ, Adams NC, Hawkins CE, Hinman AR. Patterns of transmission in measles outbreaks in the United States, 1985-1986. N Engl J Med. 1989;320:75–81. [PubMed: 2911293]

173. Yeung LF, Lurie P, Dayan G, Eduardo E, Britz PH, Redd S.B, Papania MJ, Seward JF. A Limited Measles Outbreak in a Highly Vaccinated US Boarding School. Pediatrics. 2005;116:1287–1291. [PubMed: 16322148]

174. Egemen A, Tasdemir I, Eker L, Arcasoy M. Changing Epidemiology of Measles in Turkey: Need for Reassessment of Measles Vaccination Policy? J Trop Pediatr. 1996;42(5):299–301. [PubMed: 8936963]

N Am J Med Sci
N Am J Med Sci

175. Coetzee N, Hussey GD, Visser G, Barron P, Keen A. The 1992 measles epidemic in Cape Town – a changing epidemiological pattern. S Afr Med J. 1994;84(3):145–9. [PubMed: 7740350]

176. Schmitt HJ, Wirsing von Konig CH, Neiss A. Efficacy of acellular pertussis vaccine in early childhood after household exposure. JAMA. 1996;275:37–41. [PubMed: 8531284]

177. Gustafsson L, Hallander HO, Olin P, Reizenstein E, Storsaeter J. A controlled trial of a two-component acellular, a five-component acellular, and a whole-cell pertussis vaccine. N Engl J Med. 1996;334:349–55. [PubMed: 8538705]

178. Briss PA, Fehrs LJ, Parker LA, Wright PF, Sannella EC, Hutcheson RH, Schaffner W. Sustained transmission of mumps in a highly vaccinated population: assessment of vaccine failure and waning vaccine-induced immunity. Journal of Infectious Diseases. 1994;169:77–82. [PubMed: 8277201]

179. Ströhle A, Eggenberger K, Steiner CA, Matter L, Germann D. Mumps epidemic in vaccinated children in West Switzerland. Schweiz Med Wochenschr. 1997;127(26):1124–33. [PubMed: 9312835]

180. Tayil SE, El-Shazly MK, El-Amrawy SM, Ghouneim FM, Abou Khatwa SA, Masoud GM. Sero-epidemiological study of measles after 15 years of compulsory vaccination in Alexandria, Egypt. East Mediterr Health J. 1998;4(3):437–47.

181. Krause PJ, Cherry JD, Deseda-Tous J, Champion JG, Strassburg M, Sullivan C, Spencer MJ, Byson YJ, Welliver RC, Boyer KM. Epidemic measles in young adults.Clinical, epidemiologic, and serologic studies. Ann Intern Med. 1979;90(6):873–6. [PubMed: 443682]

182. Matter L, Bally F, Germann D, Schopfer K. The incidence of rubella virus infections in Switzerland after the introduction of the MMR Mass vaccination programme. European Journal of Epidemiology. 1995;11(3):305–10. [PubMed: 7493663]

183. Alexander LN, Seward JF, Santibanez TA, Pallansch MA, Kew OM, Prevots DR, Strebel PM, Cono J, Wharton M, Orenstein WA, Sutter RW. JAMA. 2004;292:1696–1701. Vaccine Policy Changes and Epidemiology of Poliomyelitis in the United States. [PubMed: 15479934]

184. Ramsay ME, McVernon J, Andrews NJ, Heath PT, Slack MP. Estimating Haemophilus influenzae type b vaccine effectiveness in England and Wales by use of the screening method. J Infect Dis. 2003;188(4):481–5. [PubMed: 12898433]

185. Sarangi J, Cartwright K, Stuart J, Brookes S, Morris R, Slack M. Invasive Haemophilus influenzae disease in adults. Epidemiol Infect. 2000;124(3):441–7. [PMCID: PMC2810930] [PubMed: 10982068]

186. McQuillan GM, Coleman PJ, Kruszon-Moran D, Moyer LA, Lambert SB, Margolis HS. Prevalence of hepatitis B virus infection in the United States: the National Health and Nutrition Examination Surveys, 1976 through 1994. Am J Public Health. 1999;89:14–18. [PMCID: PMC1508496] [PubMed: 9987458]

187. Sicot C. Le Concours Médical. Medico-Surgical. 1993;115(8)

188. Galil K, Fair E, Mountcastle N, Britz P, Seward J. Younger age at vaccination may increase risk of varicella vaccine failure. J Infect Dis. 2002;186(1):102–5. [PubMed: 12089668]

189. Tanaka M, Vitek CR, Pascual B, Bisgard KM, Tate JE, Murphy TV. Trends in Pertussis Among Infants in the United States, 1980-1999. JAMA. 2003;290:2968–2975. [PubMed: 14665658]

N Am J Med Sci
N Am J Med Sci

190. The Bercow Report - A Review of Services for Children and Young People (0-19) with Speech, Language and Communication Needs, pub 17-12-2008. Department for Education and Skills (DfES) ISBN 978-1-84775-211-6.

191. Kootz JP, Marinelli B, Cohen DJ. Sensory receptor sensitivity in autistic children. Journal of Autism and Developmental Disorders. 1982;12(2):185–193. [PubMed: 7174607]

192. Bell JG, MacKinlay EE, Dick JR, MacDonald DJ, Boyle RM, Glen AC. Essential fatty acids and phospholipase A2 in autistic spectrum disorders. Prostaglandins, Leukotrienes and Essential Fatty Acids. 2004;71(4):201–204. [PubMed: 15301788]

193. Warren RP, Margaretten NC, Foster A. Reduced Natural Killer Cell Activity in Autism. Journal of the American Academy of Child and Adolescent Psychiatry. 1987;26(3):333–335. [PubMed: 3597287]

194. Warren RP, Margaretten NC, Pace NC, Foster A. Immune Abnormalities in Patients with Autism. Journal of Autism and Developmental Disorders. 1986;16(2):189–197. [PubMed: 2941410]

195. Warren RP, Yonk LJ, Burger RA, Cole P, Odell JD, Warren WL, White E, Singh V.K. Deficiency of Suppressor-inducer (CD4+CD45RA+) T Cells in Autism. Immunological Investigations. 1990;19(3):245–251. [PubMed: 2142123]

196. Del Giuidce-Asch G, Hollander E. Altered immune function in autism. International Journal of Neuropsychiatric Medicine. 1997;2:61–68.

197. DeLong GR. Autism: new data suggest a new hypothesis. Neurology. 1999;52(5):911–916. [PubMed: 10102405]

198. Cannell JJ. Autism and Vitamin D. Medical Hypotheses. 2008;70(4):750–9. [PubMed: 17920208]

199. Coyle P, Philcox JC, Carey LC, Rofe AM. Metallothionine: the multipurpose protein. Cellular and Molecular Life Sciences. 2002;59(4):627–647. [PubMed: 12022471]

200. Maret W. Metallothionein redox biology in the cytoprotective and cytotoxic functions of zinc. Experimental Gerontology. 2008;43(5):363–369. [PubMed: 18171607]

201. West CE. Vitamin A and measles. Nutr Rev. 2000;58(2Pt 2):S46–54. [PubMed: 10748617]

202. Barclay AJ, Foster A, Sommer A. Vitamin A supplements and mortality related to measles: a randomised clinical trial. Br Med J (Clin Res Ed) 1987;294(6567):294–6. [PMCID: PMC1245303] [PubMed: 3101849]

203. Filipek PA, Juranek J, Nguyen MT, Cummings C, Gargus JJ. Relative carnitine deficiency in autism. J Autism Dev Disord. 2004;34(6):615–23. [PubMed: 15679182]

204. Lake CR, Ziegler MG, Murphy DL. Increased norepinephrine levels and decreased dopamine-hydroxylase activity in primary autism. Arch Gen Psychiatry. 1977;34:553–6. [PubMed: 558741]

205. Todd R, Ciaranello R. Demonstration of inter-and intraspecies differences in serotonin binding sites by antibodies from an autistic child. Proc Nat Acad Sci. 1985;82:612–616. [PMCID: PMC397091] [PubMed: 2578670]

206. Bingham M. Autism and the Human Gut Microflora. 2002 May

207. Wakefield AJ, Anthony A, Murch SH, Thomson M, Montgomery SM, Davies S, O'Leary JJ, Berelowitz M, Walker-Smith JA. Enterocolitis in Children with Developmental Disorders. Am.J.Gastroenterology. 2000;95(9):2285–2295. [PubMed: 11007230]

208. Stubbs G, Litt M, Lis E, Jackson R, Voth W, Lindberg A, Litt R. Adenosine Deaminase Activity Decreased in Autism. J.Am Acad Child Psych. 1982;21:71–74. [PubMed: 7096833]

209. Persico AM, Militerni R, Bravaccio C, Schneider C, Melmed R, Trillo S, Montecchi F, Palermo MT, Pascucci T, Puglisi-Allegra S, Reichelt KL, Conciatori M, Baldi A, Keller F. Adenosine Deaminase Alleles and Autistic Disorder. Am J Med Genetics. 2000;96:784–790. [PubMed: 11121182]

210. Torrente FP, Ashwood P, Day R, Machado N, Furlano RA, Anthony A, Davies S E, Wakefield AJ, Thomson MA, Walker-Smith JA, Murch SH. Small intestinal enteropathy with epithelial, IgG and complement deposition in children with regressive autism. Molec.Psych. 2002;7:375–382. [PubMed: 11986981]

211. Fillano JJ, Goldenthal MJ, Harker Rhodes C, Marin-Garcia J. Mitochondrial dysfunction in patients with hypotonia, epilepsy, autism, and developmental delay: HEADD syndrome. J Child Neurol. 2002;17(6):435–9. [PubMed: 12174964]

212. Oliveira G, Diogo L, Grazina M, Garcia P, Ataide A, Marques C, Miguel T, Borges L, Vicente AM, Oliveira CR. Mitochondrial dysfunction in autism spectrum disorders: a population-based study. Dev Med Child Neurol. 2005;47:185–9. [PubMed: 15739723]

213. Niederhofer H, Staffen W, Mair A. Lofexidine In Hyperactive And Impulsive Children With Autistic Disorder. Journal of the American Academy of Child & Adolescent Psychiatry. 2002;41(12):1396–1397. [PubMed: 12447025]

214. Birmaher B, Quintana H, Greenhill LL. Methylphenidate treatment of hyperactive autistic children. J Am Acad Child Adol Psychiatry. 1988;27:248–251. [PubMed: 3360732]

215. Perry R, Campbell M, Adams P, Lyneh N, Speneer EK. Long-term efficacy of haloperidol in autistic children: Continuous versus discontinuous drug administration. J Am Acad Child Adolesc Psychiatry. 1959;25:57–92. [PubMed: 2914841]

216. Cohen DJ, Young JG, Nathanson JA, Shaywitz BA. Clonidine in Tourette's syndrome. The Lancet. 1979;2:551–553. [PubMed: 89558]

217. Niederhofer H, Staffen W, Mair A. Tianeptine: a novel strategy of psychopharmacological treatment of children with autistic disorder. Human psychopharmacology. 2003;18(5):389–393. [PubMed: 12858327]

218. Niederhofer H, Staffen W, Mair A. Galantamine may be effective in treating autistic disorder. British Medical Journal. 2002;325:1422. [PMCID: PMC1124870] [PubMed: 12480867]

219. Niederhofer H, Staffen W, Mair A. Immunoglobulins as an Alternative Strategy of Psychopharmacological Treatment of Children with Autistic Disorder Neuropsychopharmacology. 2003;28:1014–1015. [PubMed: 12700706]

220. Niederhofer H, Staffen W, Mair A, Pittschieler K. Melatonin facilitates sleep in individuals with mental retardation and insomnia. J Autism Dev Disord. 2003;33(4):469–72. [PubMed: 12959427]

221. Ratey JJ, Bemporad J, Sorgi P, Bick P, Polakoff S, O'Driscoll G, Mikkelsen E. Brief report: Open trial effects of beta-blockers on speech and social behaviors in 8 autistic adults. Journal of Autism and Developmental Disorders. 1987;17(3):439–446. [PubMed: 3654495]

222. Beaudet AL. Autism: highly heritable but not inherited. Nature Medicine. 2007;13:534–536. [PubMed: 17479094]

223. Busto R, Dietrich WD, Globus MY, Valdes I, Scheinberg P, Ginsberg MD. Small differences in intraischemic brain temperature critically determine the extent of ischemic neuronal injury. J Cereb Blood Flow Metab. 1987;7:729–738. [PubMed: 3693428]

224. Torres AR. Is fever suppression involved in the etiology of autism and neurodevelopmental disorders? BMC Pediatr. 2003;3:9. [PMCID: PMC194752] [PubMed: 12952554]

225. Urnovitz HB, Tuite JJ, Higashida JM, Murphy WH. RNAs in the Sera of Persian Gulf War Veterans Have Segments Homologous to Chromosome 22q11.2. Clinical and Diagnostic Laboratory Immunology. 1999;6(3):330–335. [PMCID: PMC103718] [PubMed: 10225831]

226. Rook GAW, Zumla A. Gulf War Syndrome: is it due to a systemic shift in cytokine balance towards Th2 profile? The Lancet. 1997;349:1831–3. [PubMed: 9269228]

227. Meggs WJ. Multiple Chemical Sensitivities and the Immune System. Tox. Indust. Health. 1994;8:203–214. [PubMed: 1412486]

228. Hotopf M, David A, Hull L, Ismail K, Unwin C, Wessely S. Role of Vaccinations as risk factors for ill health in veterans of the Gulf War: cross sectional study. British Medical Journal. 2000;320:1363–7. [PMCID: PMC27378] [PubMed: 10818024]

229. DeSoto MC. Blood Levels of Mercury Are Related to Diagnosis of Autism: A Reanalysis of an Important Data Set. Journal of Child Neurology. 2007;22(11):1308–1311. [PubMed: 18006963]

230. Tokuomi H, Uchino M, Imamura S, Yamanaga H, Nakanishi R, Ideta T. Minamata disease (organic mercury poisoning): Neuroradiologic and electrophysiologic studies. Neurology. 1982;32:1369–1375. [PubMed: 6890643]

231. Palmer RF, Blanchard S, Stein Z, Mandell D, Miller C. Environmental mercury release, special education rates, and autism disorder: an ecological study of Texas. Health & Place. 2006;12(2):203–209. [PubMed: 16338635]

232. Amin-Zaki L, Majeed MA, Clarkson TW, Greenwood M.R. Methylmercury poisoning in Iraqi children: clinical observations over two years. British Medical Journal. 1978 Mar 1;:613–616. [PMCID: PMC1603391] [PubMed: 630256]

233. Joselow MW, Louria DB, Browder AA. Mercurialism: Environmental and occupational aspects. Annals of Internal Medicine. 1972;76:119–30. [PubMed: 4553740]

234. Wecker L, Miller S.B, Cochran SR, Dugger DL, Johnson WD. Trace Element Concentrations in Hair From Autistic Children. J. Ment Defic. Res. 1985;29:15–22. [PubMed: 4009700]

235. Adams CR, Ziegler DK, Lin JT. Mercury intoxication simulating amyotrophic lateral sclerosis. JAMA. 1983;250:642–643. [PubMed: 6864963]

236. Fagala GE, Wigg CL. Psychiatric manifestations of mercury poisoning. J Am Acad Child Adolesc Psychiatry. 1992;31(2):306–311. [PubMed: 1564033]

237. Kark RAP, Poskanzer DC, Bullock JD, Boylen G. Mercury poisoning and its treatment with n-acetyl-d, l-penicillamine. N Engl J Med. 1971;285:10–16. [PubMed: 5089366]

238. Teitelbaum P, Teitelbaum O, Nye J, Fryman J, Maurer RG. Movement Analysis in infancy may be useful for early diagnosis of autism. PNAS. 1998;95(23):13982–13987. [PMCID: PMC25000] [PubMed: 9811912]

239. Church C, Coplan J. The high functioning autistic experience: Birth to pre-teen years. J. Pediatric Health Care. 1995;9(22):29. [PubMed: 7745522]

240. Makani S, Gollapudi S, Yel L, Chiplunkar S, Gupta S. Biochemical and molecular basis of thimerosal-induced apoptosis in T-cells: a major role of mitochondrial pathway. Genes and Immunity. 2002;3:270–278. [PubMed: 12140745]

241. Shenker BJ, Datar S, Mansfield K, Shapiro IM. Induction of apoptosis in human T-cells by organomercuric compounds: a flow dytometric analysis. Toxicol Appl Pharmacol. 1997;143(2):397–406. [PubMed: 9144456]

242. Yonk LJ, Warren KP, Burger RA, Cote P, Odell JD, Warren WL, White E, Singh VK. CD4 helper –T cell depletion in autism. Immunology Letters. 1990;25:344–346. [PubMed: 1979061]

243. Parker SK, Schwartz B, Todd J, Pickering LK. Thimerosal-Containing Vaccines and Autistic Spectrum Disorder: A Critical Review of Published Original Data. Pediatrics. 2004;114(3):793–804. [PubMed: 15342856]

244. DeSoto MC, Hitlan RT. Relationship between mercury and autism - autistic children may be less efficient at eliminating mercury from the blood. J.Child Neurol. 2007;22:1308–11. [PubMed: 18006963]

245. Kabuto M. Chronic effects of methylmercury on the urinary excretion of catecholamines and their responses to hypoglycemic stress. Arch Toxical. 1991;65(2):164–167. [PubMed: 2059158]

246. Clarkson TW. Mercury: major issues in environmental health. Environ Health Perspect. 1992;100:31–38. [PMCID: PMC1519577] [PubMed: 8354179]

247. Brenner RP, Snyder RD. Late EEG finding and clinical status after organic mercury poisoning. Arch Neurol. 1980;37(5):282–284. [PubMed: 7387446]

248. Grandjean P, Weihe P, White RF, Debes F. Cognitive performance of children prenatally exposed to ‘safe’ levels of methylmercury. Environmental Research. 1998;77(2):165–172. [PubMed: 9600810]

249. Rice DC, Gilbert SG. Early chronic low-level methylmercury poisoning in monkeys impairs spatial vision. Science. 1982;216(4547):759–761. [PubMed: 7079739]

250. Counter SA, Buchanan LH. Mercury exposure in children: a review. Toxicology and Applied Pharmacology. 2004;198(2):209–230. [PubMed: 15236954]

251. Hrdina PD, Peters DA, Singhal RL. Effects of chronic exposure to cadmium, lead and mercury of brain biogenic amines in the rat. Research Communications in Chemistry, Pathology and Pharmacology. 1976;15(3):483–493. 1976. [PubMed: 996361]

252. Flarend RE, Hem SL. In vivo absorption of aluminum-containing vaccine adjuvants using 26Al. Vaccine. 1997;15:1314–1318. [PubMed: 9302736]

253. Rimland B. ‘Recovery from autism is possible’, cited in Autism Research Review International. 1994;8(2):3.

254. Rimland B, Baker S.M. Brief Report: Alternative Approaches to the Development of Effective Treatments for Autism. Journal of Autism and Developmental Disorders. 1996;26(2):237–241. [PubMed: 8744492]

255. Rossignol D, Rossignol L. Hyperbaric oxygen therapy may improve symptoms in autistic children. Medical Hypotheses. 2006;67(2):216–228. [PubMed: 16554123]

256. Lavine L. Osteopathic and Alternative Medicine Aspects of Autistic Spectrum Disorders. First International Autism Internet Conference, British Autism Society, London, England. 1999

257. Jarusiewicz B. Efficacy of neurofeedback for children in the autistic spectrum: A pilot study. Journal of Neurotherapy. 2002;6(4):39–49.

258. Kouijzer MEUJ, de Moor JMH, Gerrits BJL, Buitelaar JK, van Schie HT. Long-term effects of neurofeedback treatment in autism. Research in Autism Spectrum Disorders. 2009;3:496–501.

259. Sichel AG, Fehmi LG, Goldstein DM. Positive outcome with neurofeedback treatment of a case of mild autism. Journal of Neurotherapy. 1995;1(1):60–64.

260. Levy SE, Hyman SL. Novel Treatments for Autistic Spectrum Disorders Mental Retardation and Developmental Disabilities Research Reviews. 2005;11:131–142. [PubMed: 15977319]

261. Sommer A, Katz J, Tarwotjo I. Increased risk of respiratory disease and diarrhea in children with pre-existing vitamin A deficiency. Am J Clin Nutr. 1984;40:1090–5. [PubMed: 6496388]

262. Minshew NJ. ‘Brief Report: Brain Mechanisms in Autism: Functional and Structural Abnormalities’ Journal of Autism and Developmental Disorders. 1996;26(2):205–209. [PubMed: 8744486]

263. Madsen KM, Lauritsen MB, Pedersen CB, Thorsen P, Plesner A-M, Andersen PH, Mortensen PB. Thimerosal and the Occurrence of Autism: Negative Ecological Evidence From Danish Population-Based Data. Pediatrics. 2003;112(3):604–606. [PubMed: 12949291]

264. Wakefield AJ, Anthony A, Murch SH, Thomson M, Montgomery SM, Davies S, O’Leary JJ, Berelowitz M, Walker-Smith JA. Enterocolitis in Children with Developmental Disorders. Am.J.Gastroenterology. 2000;95(9):2285–2295. [PubMed: 11007230]

265. Thompson J. Vaccine antibodies are lower in very premature babies. Community Practitioner. 2002 Apr 1;

266. Review of Autism Research. London: Causes and Epidemiology, MRC; 2002. Medical Research Council.

Figures and Tables

Table 1

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Table 2

Typical Physiological Systems

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