Diabetes: Causes, Natural Control, and the Mercury/Vaccine Factor

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1.1 Introduction

Diabetes (hyperglycemia) incidence is increasing drastically. For individuals born in 2000, the lifetime risk of diabetes in the U.S. is 33% and over 16 million in the U.S. currently have diabetes (502d, 580, 598), while over 54 million have insulin resistance or pre-diabetes (1). Childhood diabetes is increasing even more rapidly than adult diabetes (82, 493, 502, 598, 1). Over the last 20 years the percent of diabetes cases below 20 years old has increased from 2% to over 30%, and there was a 70% increase in cases under 40 years of age between 1990 and 1998 (502). Studies in the U.S. and Europe have found toxic metals and vaccinations to be factors in the increased diabetes cases (502, 369, 35, 100, 490-492) along with poor dietary habits (1). A greatly increased incidence of juvenile diabetes has been correlated to specific vaccination sequences and to the number of vaccines given (11, 502). Autoimmune diseases with a predominant Th1 cytokine component involvement in some cases include insulin-dependent diabetes mellitus (12). Incidence of insulin resistance which a factor in many chronic conditions has also increased significantly. Diabetes and pre-diabetes are conditions rather than diseases, and usually can be prevented or controlled - see Section V.

There are 2 basic types of diabetes, Type I (autoimmune) diabetes and Type II (insulin resistance). In type I diabetes the body's immune system attacks its own insulin producing tissue, the beta cells of the pancreas (580). Type II diabetes involves metabolic failure related to poor diet, obesity, environmental factors, and genetic susceptibility (1). Insulin resistance is a primary factor in type 2 diabetes and results when the body cannot properly use insulin, which is secreted by the pancreas to move glucose from the blood into the cells that need it. The pancreas then produces extra insulin in a futile effort to compensate, leading to higher insulin and glucose levels in the blood along with a deficiency of glucose in the cells that need sugar to function properly. Those with either type I or type II diabetes have been found to be more likely to have other chronic conditions including heart disease, strokes, kidney disease, Alzheimer's, eye conditions and blindness (580, 581)

1.2 Causes of Diabetes and the mercury/toxic metal connection

Type 2 diabetes and pre-diabetes is an epidemic among young Americans and greatly increases the incidence of heart attack, blindness, stoke, infertility, and early death. Brain inflammation has been found to be a major factor in pre-diabetes, diabetes, and the closely related metabolic syndrome which includes diabetes or pre-diabetes, high blood pressure, and high cholesterol (598, 1, 581b, etc.). Causes of oxidative stress and lipid peroxidative related brain inflammation that have been documented include vaccines, mercury, aluminum, excitotoxins such as MSG, aspartame, food additives, and overconsumption of high-fructose corn sweetener, starches, sweets, and other omega 6 oils (13, 424, 489, 494, 496, 596-599, 1, etc.). These cause high glutamate levels in the brain and oxidative damage-resulting in inflammation of the brain and immune system, as well as damage to brain microglia cells and the mitochondrial DNA, fatigue, high triglycerides, metabolic syndrome, etc. These have been found to be factors in most chronic neurological diseases. Inflammation induced by vaccine adjuvants like aluminum and mercury or by excitotoxins like MSG has been found to play a significant role in insulin resistance (type-2 diabetes) and in high levels of LDL cholesterol (597, 598, etc.). There is also evidence that the diet drink sweetener aspartame can cause or increase the effects of diabetes and hypoglycemia (450, 498). Higher levels of vit D have been found to be associated with lower levels of insulin resistance and appears to be protective (580). Theoflavins from black or green tea and curcumin have been found to be significantly effective in protection against inflammation and inflammatory conditions (580).

Reduced levels of magnesium and zinc are related to metabolic syndrome, insulin resistance, and brain inflammation, and are protective against these conditions (599, 43). Mercury and cadmium inhibiting magnesium and zinc levels as well as inhibiting glucose transfer are other mechanisms by which mercury and toxic metals are factors in metabolic syndrome and insulin resistance/diabetes (43, 198, 338, 597). Iron overload has also been found to be a cause of insulin resistance/type 2 diabetes (595).

Mercury exposure levels from vaccinations have likewise shown major increases during this period and have been found to be a major factor in the increase of other conditions like autism and ADHD (8). The most common mechanism of causality has been found to be blockage by mercury and other toxic metals of the enzymatic processes related to digestion of milk casein and wheat gluten, resulting in dumping of morphine like substances in the blood of the majority of children with these conditions (9).

A direct mechanism involving mercury's inhibition of cellular enzymatic processes by binding with the hydroxyl radical (SH) in amino acids appears to be a major part of the connection to allergic/immune reactive conditions (15-23, 98, 4). For example mercury has been found to strongly inhibit the activity of xanthine oxidase (16) and dipeptyl peptidase (DPP IV) which are required in the digestion of the wheat protein gluten or the milk protein casein (4, 15, 17, 19, 20, 24-26, 98, 499) - the same protein that is cluster differentiation antigen 26 (CD26) which helps T lymphocyte activation. CD26 or DPPIV is a cell surface glycoprotein that is very susceptible to inactivation by mercury binding to its cysteinyl domain. Mercury and other toxic metals also inhibit binding of opioid receptor agonists to opioid receptors, while magnesium stimulates binding to opioid receptors (15).

Beta-casomorphine-7 is a morphine like compound that results in neural disfunction (24, 25, 4), as well as being a direct histamine releaser in humans and inducing skin reactions (14, 21, 25c).

A similar mechanism related to mercury and toxic metals blocking the enzymatic processes related to digestion of gluten and casein have also been shown to be a significant factor in causality of children's type I diabetes. Early introduction of feeding cow's milk to infants rather breastfeeding has been shown to be a significant risk factor in type I diabetes incidence (4, 5).

Most infants in recent years have been found to be exposed to high levels of mercury and toxic

metals through vaccines, mother's amalgam dental fillings, and other sources (8), with toxic metals documented to block the enzymatic processes needed to digest casein and gluten. Studies have found high levels of Ig A antigen specific antibodies for casein, lactalbumin and beta-lactoglobulin and IgG and IgM for casein in cow's milk (9, 4, etc.). In vitro the bioactive peptide beta-casomorphin 7 (BCM-7) is yielded by the successive gastrointestinal proteolytic digestion of bovine beta-casein variants A1 and B, but this was not seen in variant A2 (4, 498). In hydrolysed milk with variant A1 of beta-casein, BCM-7 level is 4-fold higher than in A2 milk. Consumption of the beta-casein A1 variant had a strong correlation to diabetes incidence (r = +0.726). Even more pronounced was the relation between beta-casein (A1+B) consumption and diabetes (r = +0.982).(4a) These latter two cow caseins yield a bioactive peptide beta-casomorphin-7 after in vitro digestion with intestinal enzymes whereas the common A2 variant or the corresponding human or goat caseins do not. BCM-7 has also been found to be a factor in autism and schizophrenia. Mercury and toxic metals have been found to disable the enzymatic process required to digest casein (9).

A1 is the most frequent in Holstein-Friesian (0.310-0.660), Ayrshire (0.432-0.720) and Red (0.710) cattle (4b, 498). In contrast, a high frequency of A2 is observed in Guernsey (0.880-0.970) and Jersey (0.490-0.721) cattle as well as Asian and African cattle. BCM-7 may play a role in the aetiology of many human diseases including diabetes, neurological, immune, and cardiovascular (498). Epidemiological evidence from New Zealand claims that consumption of beta-casein A1 is associated with higher national mortality rates from ischaemic heart disease. It seems that the populations that consume milk containing high levels of beta-casein A2 have a lower incidence of cardiovascular disease and type 1 diabetes. BCM-7 has also been suggested as a possible cause of sudden infant death syndrome. In addition, neurological disorders, such as autism and schizophrenia, apprear to be associated with milk consumption and a higher level of BCM-7 (4b)

Lactose Intolerance

Lactose (milk sugar), which is a major component of milk, is a disaccharide sugar made up of the simple sugars glucose and galactose (3). Lactase is an enzyme which facilitates digestion of lactose. Over 50% of non-Caucasians are lactose intolerant, to a significant degree and about 20% of Caucasians. Infants are most lactose tolerant but lactase activity declines dramatically over time so that by adulthood to about 5 to 10% of the level of infants. Only a relatively small percentage of people retain enough lactase activity to absorb significant amounts of lactose throughout their adult life (3). Lactose intolerance results in undigested lactose in the intestines which often causes gas, bloating, abdominal discomfort, and proliferation of bacteria in the intestines. In addition to inhibiting the enzymes such as peptidases required to digest milk casein and whey, chronic mercury exposure in animals has also been found to inhibit lactase and glucose-6-phosphatase needed to digest lactose and other polysaccharides (32). Thus chronic exposure to mercury and toxic metals also increases lactose intolerance and digestion problems of carbohydrates in general. Digestive problems have been found to *commonly be improved*[1] by reducing chronic mercury and toxic metal exposures.

Lactose intolerance can also be alleviated to some degree by supplemental enzymes, using fermented milk products such as yogurt or kefir, or using only small amounts of milk products spread throughout the day (3).

Rat study findings suggest that mercury-induced changes in RT6+ T lymphocytes appear to be related to the development of renal autoimmune insulin-dependent diabetes mellitus disease in genetically predisposed BN rats (6). Treatment of a strain of rats that are more prone to diabetes than other strains with mercury chloride results in significantly higher incidences of autoimmunity to thyrogobulin and laminin (30). Mercury has been documented to cause autoimmune conditions like thyroiditis, MS, Lupus, eczema, etc. and people who replace amalgam fillings commonly recover or significantly improve from these conditions (369b).

[1]**Internet:** "http://www.flcv.com/hgrecovp.html".

Another study found that the age or timing of vaccinations containing mercury thimerosal affects the incidence of diabetes in rats (31). Vanadium, uranium, and other toxic metals have also been found to damage pancreatic islet cells and be a factor in diabetes (490).

Until mercury thimerosal was removed from most vaccines, vaccines were the largest source of mercury in most infants and young children (8a) and exposures far more than Government health guidelines were common. Dental amalgam has been documented to be the largest source of mercury in most who have several amalgam mercury fillings (500) with high typical exposures far more than Gov't health guidelines. Dental amalgam has been documented to be the largest source of mercury in most mothers with several amalgam fillings and the largest prenatal mercury exposure source to the fetus, as well as significant source of mercury exposure in infants (501).

Mercury causes release of inflammatory cytokines such as Tumor Necrosis Factor-alpha (TNFa) and Interleukin-4 which are documented to be factors in the chronic inflammatory conditions, cell death, and damage to the immune system. (47, 65, 101) TNFa (tumor necrosis factor-alpha) is a cytokine that controls a wide range of immune cell response in mammals, including cell death (apoptosis). Mercury has been shown to induce TNFa, deplete glutathione, and increase glutamate, dopamine, and calcium related toxicity, causing inflammatory effects and cellular apoptosis in immune cells (47). Cell signaling mechanisms like sphingolipids are part of the control mechanism for the TNFa apoptosis mechanism (101). Glutathione is an amino acid that is a normal cellular mechanism for controlling apoptosis. When glutathione is depleted, reactive oxidative species increased, and CNS and cell signaling mechanisms are disrupted by toxic exposures such as mercury, neuronal cell apoptosis results and cellular damage. Oxidative stress including methylmercury-induced oxidative stress causes pancreatic beta-cell apoptosis (programmed cell death) and dysfunction (491, 492, 493, 499). Thimerosal has similar toxicity to methyl mercury (8a) and dental amalgam is a significant source of methyl-mercury in those with amalgam fillings (500).

Both immune cell type Th1 and Th2 cytokine responses are involved in autoimmunity (425c). Mercury has been found to affect both Th1 and Th2 cytokines causing an increase in inflammatory Th2 cytokines (152, 181, 285, 404b) as well as in Th1 cytokines in some circumstances (6). In the pancreas, the cells responsible for insulin production can be damaged or destroyed by the chronic high levels of cytokines, with the potential of inducing type I or II diabetes - even in otherwise healthy individuals with no other risk factors for diabetes (502). Mercury inhibits production of insulin and is a factor in diabetes and hypoglycemia, with significant reductions in insulin need after replacement of amalgam filings and normalizing of blood sugar (35, 502).

Mercury exposure causes an approximately 1.8-fold increase in glucose transport. This glucose transport corresponds with an increase in GLUT 1 glucose transporters. Phosphorylation of p38 kinase increased with mercury exposure. Activation of p38 and an increase in glucose transport corresponding to an increase in GLUT 1 are indicative the induction of a stress response, which can contribute to the induction of insulin resistance in adipocytes. While the magnitude of the action of mercury is modest, its effects were sustained over many days of exposure and impacted subsequent insulin-mediated glucose transport. Pre-treatment with HgCl2 decreased insulin-mediated glucose transport 1.3-fold suggesting that exposure to mercury may contribute to pathologies associated with glucose homeostasis (10).

Studies have also found evidence of a connection between low levels of zinc and childhood-onset diabetes (72). Zinc is an antagonist to toxic metals like cadmium and mercury, and adequate levels are required to balance the adverse effects of these toxic metals on cellular calcium and other enzymatic processes (28, 74). Mercury reduces the bloods ability to transport essential nutrients such as calcium, magnesium, and zinc; and has an adverse effect on cellular levels of these essential minerals, as well as blocking the function of magnesium and zinc in cellular enzymatic functions (43, 96, 198, 338). Part of the toxic effects of mercury, cadmium, lead, etc. are through their replacing essential minerals such as zinc at their sites in enzymes, disabling the necessary enzymatic processes. Other connections

between mercury and type 1 diabetes have also been demonstrated. Mercury inhibits production of insulin and is a factor in diabetes and hypoglycemia, with significant reductions in insulin need after replacement of amalgam filings and normalizing of blood sugar (35).

Dr. J. Bart Classen discovered it would be possible to study the effect of Hemophilus B immunization on the incidence of IDDM using data from a large clinical trial in Finland. Dr. J. Bart Classen and D.C. Classen initiated and funded a collaboration with Dr. Tuomilehto in Finland. All children born in Finland between October 1st, 1985 and August 31st, 1987, approximately 116,000 were randomized to receive 4 doses of the HiB vaccine (PPR-D, Connaught) starting at 3 months of life or one dose starting at 24 months of life. Classen and Classen calculated the incidence of IDDM in both groups through age 10 and in an group which did not receive the HiB vaccine, a cohort which included all 128, 500 children born in Finland in the 24 months prior to the Hemophilus vaccine study. Immediately following the completion of these two arms all children born in Finland over a two year period were randomized to receive 3 doses of the old PPR-D HiB vaccine or 3 doses of a newerHbOC HiB vaccine. The data supports published findings that the immunization starting after 2 months of life is associated with an increased incidence of IDDM. Rises in diabetes have been seen in the UK and USA following the introduction of the hemophilus vaccine (99). Other studies have found that any vaccine that contains mercury or other substances that can induce autoimmunity can cause type I or II diabetes and that vaccinations are a common cause of autoimmunity (100).

1.3 Insulin Resistance increases incidence of neurological conditions including Alzheimer's

Higher insulin and glucose levels in the blood and deficiency of glucose in brain cells that need it has been found to lead to neurological problems such as Alzheimer's (580, 581). Diabetes also impacts memory by increasing the risk blood vessels will become obstructed, restricting blood flow to the brain.

High blood glucose levels also impact cognition through formation of sugar-related toxins called advanced glycation end products (AGEs). AGEs have been found to be a factor in aging, diabetes, and Alzheimer's. Glycotoxins are formed when sugars interact with proteins and lipids, damaging the structure of proteins and membranes, rendering them less able to carry out their many vital processes. (581). Studies have shown that AGEs are a key factor in cross-linking of harmful beta-amyloid plaques in the brain that are implicated in Alzheimer's. As previously documented mercury and aluminum exposure increase insulin resistance and amalgam replacement and detoxification reduce insulin resistance.

1.4 Effect of Persistent Organic Pollutants (POPs)

Effects of low-level exposure to some persistent organic pollutants (POPs) was surveyed in a large population (NHANES) survey (497). There was a strong dose-response relations between serum concentrations of six selected POPs and the prevalence of diabetes. Diabetes prevalence was strongly positively associated with lipid-adjusted serum concentrations of all six POPs. When the participants were classified according to the sum of category numbers of the six POPs, adjusted odds ratios were strongly significant for trend. The association was consistent in stratified analyses and stronger in younger participants, Mexican Americans, and obese individuals. Systemic detoxification has been found to be effective in reducing the levels of toxic metals and POPs and to lessen related adverse health effects. There are many alternatives for accomplishing this that have been demonstrated to have effectiveness.

1.5 Effective Treatment or Control of Diabetes

Authorities referenced here suggest the following measures to prevent diabetes and insulin resistance: lower toxic exposure levels, control weight and lose weight if overweight, reduce body fat percentage where excessive, regular exercise, good diet using glycemic index or glycemic load information, take appropriate supplements in addition to a good multivitamin/multimineral supplement (see below), monitor blood glucose level.

For those with several amalgam dental fillings and diabetes, amalgam replacement and detoxification commonly results in significant improvement (499, 598, etc.). Reduced consumption of excitoxins like MSG and aspartame and of high fructose corn syrup and other omega 6 oils along with increased consumption of magnesium and omega 3 oils such as virgin olive oil and coconut oil have been found to reduce brain inflammation and improve inflammatory conditions like insulin resistance, diabetes, depression, etc. (598, 599, 580, etc.). Drugs such as diuretics, blood pressure medications, steroids for asthma, arthritis, or allergies, prozac, and birth control pills that can increase insulin resistance and blood sugar (1). Drugs used to treat diabetes such as Avandia and Actos have been found to cause significant increases in cardiovascular disease and heart attacks, as well as increased risk of hip fracture (581b, FDA, etc.)

Other factors that have been found to be significantly associated with cardiovascular disease and diabetes include daily consumption of soda drinks, diet drinks, fried foods, or a "Western Diet" high in fried foods, refined grains, fast foods, soda, etc. and low in fruits and vegetables (590, 598, 580, 1). These diet patterns all have been found to be significantly associated with metabolic syndrome, a cluster of cardiovascular disease and diabetes risk factors including elevated waist circumference, high blood pressure, elevated triglycerides, low levels of high-density lipoprotein (HDL or "good") cholesterol, clogged arteries, and high fasting glucose levels. The presence of three or more of the factors increases a person's risk of developing diabetes and cardiovascular disease. Avoidance of these dietary causes of insulin resistance and metabolic syndrome along with **exercise**such as walking, have been found to be highly effective at preventing or controlling these conditions (1, 598, etc.) Some carbohydrates elevate blood glucose almost instantly – others much more slowly. The *Glycemic Index* (GI) is a measure of how quickly carbohydrates elevate glucose. A lower number indicates a slower glucose rise – and better glucose control. Monitoring and avoiding high glycemic foods along with exercise have been found to be effective in controlling insulin resistance (1c).

Oily fish, greens (chlorophyll) and Fenugreek are too foods that have been documented to be effective in helping to control insulin resistance (1a). Other foods supportive of controlling blood sugar include foods with high fiber content, whole grain bread, brown rice, dried beans and peas, fresh vegetables and fruit (1a). Glycemic Load (GL) takes into account not only the quality of carbohydrate but also quantity and its effects.

Several studies have documented that lipoic acid (an antioxidant and chelator) resulted in improvement in the majority of diabetes cases it was used for, by improving glucose metabolism, increasing insulin sensitivity, and reducing nerve damage (including in diabetic neuropathy)(502e) Lipoic acid is also a mercury chelator, commonly used for that purpose.

Several supplements have been found to be significantly effective in promoting healthy blood glucose metabolism. These include Gymnema sylvestre, bitter melon, N-acetyl-cysteine (NAC), Vitamin C and E, Goat's Rue, Cinnamon, Quercetin, Vanadyl sulfate, Bitter melon, PolyGycopleX (PGX) (581, 1). Nutrients that have been found effective at blocking AGEs include: Carnosine, Vitamin B1 and B6, NAC, Guava, chromium, Pycnogenol, B7, magnesium, and Yerbamate (581).

Several types of organ damage can be caused by diabetes/insulin resistance. Diabetic neuropathies are one such. Supplements documented to help with such neuropathies include lipoic acid, Vit B6 and B12, Folic Acid, Fish oil, and Huckleberry leaf tea (1a). Diabetes can also be a factor in vision problems such as cataracts and diabetic retinopathy. Supplements found to help with these conditions

include Taurine, bilberry, lycopene (1a). CAN-C drops (N-acytyl-Carnosine) have been found to be effective at reducing or reversing damage cataracts or other vision problems (582). Acetyl-L carnitine (ALC) reduces effects and pain of diabetic neuropathy (581b).

Supplements found to help with diabetic related kidney damage include Taurine, melatonin, and cranberry proanthocyanidins (1a). Supplements found to help with diabetic related cardiovascular conditions include L-Arginine, L-Carnitine, and Hawthorne which can be found in hawthorne tea or supplements (1a).

Chromium has been found to aid insulin transportation into cells. Cinnamon has been found to lower blood sugar levels. Lipoic acid has been found to improve glucose balance for people with type II diabetes. CoQ10 has cardiovascular benefits and prevents high blood pressure in people with blood glucose control problems. (581b)

1.6 Diabetes Tests

Fasting Glucose Test (optimal: 76-81 mg/dL, normal: 82-85 mg/dL prediabetic: 86-125, diabetic: ¿126 mg/dL**), Glucose test insulin level:** (optimal: =¡ 7mcU/ml, prediabetic: 8-25 mcU/ml, diabetic: ¿25 mcU/ml); **Fasting Glucose Tolerance Test with glucose and insulin levels:** (normal: glucose ¡ 140 mg/dL & insulin level ¡55 mcU/ml, prediabetic: blood glucose level of 140 to 159 mg/dL or insulin level of 56-90 mg/dl, dangerous: glucose¿ 160 mg/dL or insulin ¿90 mcU/ml); **Hemoglobin A1C (HbAiC)** (measures damage to blood proteins by free radicals created) (normal: 4.5-4.9%, prediabetic: 5-6.9%, Diabetic: ¿ 7%)

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Dr. J. Bart Classen discovered it would be possible to study the effect of Hemophilus B immunization on the incidence of IDDM using data from a large clinical trial in Finland. Dr. J. Bart Classen and D.C. Classen initiated and funded a collaboration with Dr. Tuomilehto in Finland. All children born in Finland between October 1st, 1985 and August 31st, 1987, approximately 116, 000 were randomized to receive 4 doses of the HiB vaccine (PPR-D, Connaught) starting at 3 months of life or one dose starting at 24 months of life. Classen and Classen calculated the incidence of IDDM in both groups through age 10 and in an group which did not receive the HiB vaccine, a cohort which included all 128, 500 children born in Finland in the 24 months prior to the Hemophilus vaccine study. Immediately following the completion of these two arms all children born in Finland over a two year period were randomized to receive 3 doses of the old PPR-D HiB vaccine or 3 doses of a newerHbOC HiB vaccine. The data supports published findings that the immunization starting after 2 months of life is associated with an increased incidence of IDDM. Rises in diabetes have been seen in the UK and USA following the introduction of the hemophilus vaccine (see HIB)

Dr. J. Bart Classen, **Hemophilus Vaccine Study in Finland Proves a Causal Relationship Between Vaccines and Diabetes, Autoimmunity 35:247-253, 2002, http://www.vaccines.net/hen**
Congressional Testimony by Dr. J. Bart Classen regarding the link between vaccines and **diabetes** in the military. *http://www.whale.to/vaccines/classen3.html*[44]

Dr. J. Bart Classen, Hemophilus Meningitis Vaccine Proven to Cause **Diabetes** in Clinical Trial of Over 100, 000 Children, Autoimmunity, Vol 39, July 2004

The study followed over 100, 000 children which had been randomized in a large clinical trial to receive 1 or 4 doses of the hemophilus vaccine and over 100, 000 unvaccinated children. After 7 years the group receiving 4 doses of the vaccine had a statistically significant 26% elevated rate of diabetes, or an extra 54 cases/100, 000 children, compared to children who did not receive the vaccine. By contrast immunization against hemophilus is expected to prevent only 7 deaths and 7 to 26 cases of permanent disability per 100, 000 children immunized. The study showed that almost all of the extra cases of diabetes caused by the vaccine occurred between 3-4 years after vaccination. Furthermore the paper provides new data proving the vaccine causes diabetes in mice and reviews data from 3 smaller human studies, which all had similar results to the current study, but were too small to reach statistical significance. "Our results conclusively prove there is a causal relationship between immunization schedules and diabetes. We believe immunization schedules can be made safer", stated Dr. Bart Classen. Classen JB. The diabetes epidemic and the hepatitis B vaccines. NZ Med J. 1996 Sep 27; 109 (1030):366.

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