

# **The Contribution of Soy Consumption to three Current Major World Epidemic Diseases**

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A thesis submitted in fulfillment  
of the requirements for the degree of

Master of Medical Sciences

The University of Adelaide, Australia

2012

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**This thesis contains a publication**

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This thesis includes three parts, as during the progress of work, results concerning each of these three diseases were prepared for conference presentations, and for publication. The first of those publications has already appeared and constitutes Part I of this thesis, with Parts II and III being prepared for print.

Some Conference presentations resulting from this work:

2010. July            Channel 7 interview for national TV coverage on the issue of obesity
- 2010 August:        Post-graduate poster presentation University of Adelaide.
- 2010:August:        Award for presentation on world-wide problem of obesity.
- 2010 November:    Conference presentation Australasian Society for Human Biology,  
Auckland, NZ.
- 2011 April:           American Association of Physical Anthropologists Annual Meeting,  
Minneapolis, MI peer reviewed abstract published, as well as poster  
presentation
- 2011 September    Post-graduate poster presentation on child leukaemia, University of  
Adelaide
- 2012 August:        Post-graduate conference and poster presentation on Alzheimer's  
Dementia, University of Adelaide

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The adjoined paper on “Soy contribution to Obesity” has already been published.

For the manuscript of the second paper see pages XXXX

## Preface

With the current worldwide health problems of an epidemic of obesity identified by the WHO in 1997 as a disease running parallel with other major diseases that are causing untold suffering both amongst the very young with childhood blood cancer leukaemia and an increasing epidemic amongst the elderly with Alzheimer's Dementia disease. The problem for many authorities is both cause and possible solutions. Most explanations as to why obesity occurs remain unsatisfactory. While medical science has improved the survival rates for childhood leukaemia cancer, it so far, has not provided sufficient explanation, as to why, the high incidence of child leukaemia continues to occur despite medical advances. The burden of Alzheimer's disease is also reaching epidemic proportions across the Western world as well as other countries, such that, the spiralling economic cost of care alone is now calculated in the billions, and which, if it continues may possibly cripple the, economy of some countries. The question remains, why, in this modern day and age with all the advances of science and understanding of medical science these three apparently disparate areas of human affliction continue to increase across many populations. Currently there are no plausible answers forthcoming for the proliferation of these diseases. In relation to obesity, many still regard it merely a matter of eating too much and exercising too little. For child leukaemia especially those born leukaemic, it is tenuous to correlate the disease with lifestyle factors. For those who develop Alzheimer's Disease, lifestyle over a lifetime could certainly have a part to play, but what, when other individuals with similar lifestyles do not develop the disease?

The simplest common factor explaining the onset of these three afflictions is diet. The question arises as to what kinds of dietary changes are now in place or what type of food changes are being invoked and consumed, in sufficient quantities, and that are perhaps markedly different from previous generations. The food supply of the majority of humans in the 21<sup>st</sup> century contains large amounts of industrially processed products. These new, and abundant, dietary components may contain, and have, unexpected consequences and effects contributing to obesity, child leukaemia and Alzheimer's disease. The one foremost food ingredient besides sugar, that seems to dominate the food supply is soybean. Industrially processed soybean products are now ubiquitous in the food supply of many nations. Unlike traditional Asian soy derived foods, western style industrially processed soy products (soybean oil, soy flour, soy milk, soy lecithin, tofu and an extensive array of soy utilizing processed packaged foods), are not subject to fermentation. Fermentation reduces anti-nutrient and phytohormonal contents of soy although not completely. It is well recognized that modern soy products block absorption of essential minerals (e.g. iodine) and that they contain large amounts of (e.g. genistein). Although soy is promoted as a source of cheap protein, that is supposed to improve human diets, its anti-nutrient and xenoestrogen component content, may in actual fact, produce more detrimental effects for the human body, than the so far perceived and commercially promoted "benefits" derived from increased consumption of readily and cheaply available soy protein.

The capacity of soy to not only lower iodine intake besides other essential minerals and nutrients, also, introduces into the human body, a number of extraneous estrogen like substances which may slow down metabolism sufficiently and promote female pattern of



fat deposition. Hence, their hypothesized link to obesity in the first instance. Genistein is also known to be a topoisomerase type II poison, and could well be a contributing factor to interference of cellular development, causing DNA disruption and creating chromosomal aberrations in the rapidly developing embryo, during the vulnerable prenatal stages, especially, where the mother is ingesting soy products as part of her diet. And similarly therefore, the hypothesis linking soy to childhood leukaemia. Furthermore, genistein, despite some short term studies not only disrupts thyroid function, with its interaction with the hippocampus, pituitary and thyroid axis may also possibly be a slow, contributing factor, to the early stages of memory loss as well as impaired motor coordination as frequently observed in Alzheimer's symptoms.

The meta-analysis of soy consumption and these three major diseases with prevalence across all countries for which WHO information is available, presented in this thesis, is the first in the literature tabulating an empirical result supporting the hypothesis that industrially processed soy products may contribute to the worldwide increase of overweight and obesity, childhood leukaemia and Alzheimer's disease. Of special interest are the results showing greater prevalence of obesity and poor, but heavily soybean exposed, Latin American countries than in wealthy, but using little soy, countries of the European Union. The results also show, that the two nations with the highest consumption of soy per capita, are also the two nations with the highest incidence of child leukaemia, and also the highest incidence of Alzheimer's disease (China and USA) besides their problems with an epidemic of obesity.

These empirical findings indicate the need for the implementation of public health measures, to counter the increasing obesity and Alzheimer's epidemics, the continued high incidence of childhood leukaemia despite improved medical survival rates. At policy level it may have to be decided to weigh up the economic-political benefits as opposed to the economic drain due to the escalating high cost and maintenance of medical care for all these diseases. On a broader scale, this thesis also argues that the food industry needs to be more thoroughly scrutinised to prevent its profit seeking business model and/or behaviour from creating worldwide public health problems.

**Part I.**

**Obesity and Soy**

See attached pdf for copy of published article.

Roccisano D., Henneberg M, Soy Contribution to Obesity *JNS* Feb 2012 DOI:  
10.4236/FNS202011.

# Soy Consumption and Obesity

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Received October 27<sup>th</sup>, 2011; revised December 14<sup>th</sup>, 2011; accepted December 22<sup>nd</sup>, 2011

## ABSTRACT

Obesity is now present worldwide, including China, India and developing countries. It now seems no longer acceptable to argue that obesity can simply be explained in terms of caloric consumption only using simple concept of energy in and energy out. There may be specific causes of altered metabolism that produce nutritional imbalances. Individual variation in response to food intake may also be considered. Specific substances in the food chain can influence metabolism towards an increase in fat deposits. Xenoestrogens have been suggested to have such an influence. Soy contains phytoestrogens plus phytates, protease inhibitors and other anti-nutrients which block or compromise the body's uptake of essential vitamins and minerals. This may contribute to nutritional anomalies. We analyzed data from WHO and FAO for 167 countries. These contained percentage of obese individuals ( $BMI > 30 \text{ kg/m}^2$ ), GDP, caloric consumption per capita, and sugar and soy consumption per capita. Regressions and partial correlations were used. Soy consumption correlates significantly with levels of obesity, irrespective of GDP and caloric intake. For instance, poor Latin America with soy consumption of 28.9 kg/person/year has more obesity (18.4%) than better off European Union (14.1%) consuming 16.1 kg/person/year of soy. Soy consumption seems to contribute approximately 10% - 21% to the worldwide variation in obesity, depending on the method of statistical analysis. The ubiquitous presence of unfermented soy products in mass produced foods seems to be an important contributor to the obesity epidemic.

**Keywords:** Soy; Obesity; Anti-Nutrients; Diet; Metabolism; Phytoestrogens; Phytates

## 1. Introduction

The World Health Organization (WHO) recognizes obesity as a disease of global epidemic proportions [1]. Contrary to western perceptions, now even the Chinese have an obesity problem [2]. Caloric imbalance (over-eating and a lack of exercise) that has been for many decades, widely acknowledged as the cause of obesity, is now regarded as an inadequate explanation; sleep debt, endocrine disruptors, pharmaceutical iatrogenesis [3] as well as parental influences, economic and genetic predispositions are suggested [4,5]. Body frame size differences were recently postulated as contributive to individual variation in obesity [6]. This opens up the perception that some other specific causes may be contributing to the epidemic of obesity.

Soy is ubiquitously used for mass-produced foods, both for humans and for farm animals, especially industrialized farm animals and feed lots. According to Soyatech, "world soybean production has increased by over 500% in the last 40 years," [[www.soyatech.com/soyfacts.htm](http://www.soyatech.com/soyfacts.htm)]. Soy is a vegetable that has become a "politically correct food," and is promoted as a source of abundant health for the 21<sup>st</sup> century by the soyfood processing industry. Part

of this claim is that the cheap protein from soy is equivalent or better for humans than that from animal sources. The soy industry generates an extensive array of new products affecting most of the global processed food supply with new processed foods promoted daily. Soy use takes many forms, either directly, in the form of soy flour and soy oil throughout the baking, bread-making, cooking and supermarket supply industries, or extensive in the restaurant, hotel, cafeteria and multiple fast food industry. Soy-rich processed food items include soy milk, soy-cheese, soy-yogurts, textured vegetable protein and a range of imitation meat products (often combined with other possible excess weight promoting co-factors). Directly or indirectly soy products, either with soybean extracts, soybean oil or both are heavily used in the large scale snack food industry. Soy lecithin is also used widely in the worldwide confectionery industry and the pharmaceutical industry, not only in the production of capsules, but also in their medical contents. Soy oil is hydrogenated to create many medical formulations (eg. compounded in commonly used beta blockers). Soy is also consumed by humans indirectly via feed through industrialized farm animals, particularly those bred for

human consumption. (For example, soy compounded pellets for caged animals which encourage the fattening process for market, including chickens and fish farms.) A commonly used argument for the benefits of soy, has been its supposedly extensive use in Asian cultures for thousands of years. However, most traditional Asian soy use has relied on methods such as fermentation to produce flavouring agents, soups and garnishes, rather than being used as the main dietary staple except in the historical past, in times of destitution. Historically, Asian cultures in the main, have always preferred real meat products (especially pork and seafood) to artificial imitation meat products. With regards to soy, western methods of soy processing and consumption predominately use unfermented soybeans. Such processes are and were, quite different from traditional Asian fermented soy products, thus altering contents and quantities of soy products consumed. The fact that Western methods of processing and consumption have now been exported back to, and readily adopted by Asian cultures, have made the modern western technologically based soy products available to be consumed worldwide.

Here, we advance a hypothesis that there is a correlation between soy consumption and the rise in obesity because of an unprecedented rise in consumption and ingestion of xenoestrogens and antinutrients contained in unfermented soy products.

Foods from soy can alter normal metabolic processes in a way that may lead to excess weight gain. Soy contains a number of phytates, trypsin inhibitors as well as phytoestrogens and topoisomerase II toxins, in larger quantities than most other vegetables. The xenoestrogens in soy behave like human estrogens and encourage fat accumulation in most mammals including humans. Thus, an argument could be made that the consumption of soy food products may contribute to obesity. The causes of the observed relationship may possibly belong to two categories: soy phytoestrogens and soy's ability to inhibit absorption of essential nutrients. Phytoestrogens, in particular Genistein and similar substances contained in soy, interfere with metabolism in many ways [7,8], and may induce increased fat accumulation and distribution over the body depending on its intake levels [9]. The antinutrients in soy such as trypsin inhibitors and phytates [10,11] block the absorption of minerals such as iodine, zinc, iron, magnesium, copper and chromium, and many others, [12-14] and may thus require increased food consumption to compensate for the restricted intake of these minerals manifested by continual snacking and consuming larger quantities of food. Such foods often involve refined carbohydrates and flavour enhancers which also contribute to obesity in making soy palatable.

The effects of soy on the thyroid have been well documented [14,15]. Thyroid activity compromised by

consumption of iodine inhibitors, as found in soy [16-19] leads to reduced metabolic activity which may translate into lethargy with reduced inclination to be involved in physical activities thereby encouraging further weight gain. Furthermore, low thyroxine levels have a negative domino effect on the liver, which generates more fatty acids and cholesterol encouraging further weight gains [17-19].

Soy may also exert an inhibitive influence on the trace element chromium [20] reducing the amount of available chromium, which in turn, also reduces the effectiveness of insulin [21-23]. Chromium is essential to improving glucose tolerance in mammals [23-25]. Western diets are typically low in chromium and if the microscopic levels are reduced even further by consumption of processed foods containing the anti-nutrients abundant in soy that block chromium, it is possible, that it will in turn also contribute to increased fatness and thus obesity [23-25] quite aside from problems of insulin resistance and related consequences for any associated diabetes morbidities. The chain of events that soy seems to influence gets longer with each type of newly developed processed food. An argument could also be made that many foods consumed today that have the appearance similar to what we consumed 30 - 50 years ago, are substantially different in both nutrient and solid content that have been partly or totally replaced by soy in its many forms. Hence, the importance of the necessity of correct labeling and properly detailed nutrition panels.

## Materials and Methods

WHO data on the prevalence of obesity, FAO data on soy consumption per country and UN information on GDP and on caloric consumption per country were used to explore relationships between soy consumption and obesity. Information published by the WHO by country on the percentage of persons with body mass index (BMI) greater than 30 kg/m<sup>2</sup> was used (<http://who.int/research/en>). The gross domestic product (GDP in US\$) per capita and the caloric consumption per capita were obtained from United Nations sources (<http://unstats.un.org/unsd/snaama/>). Data from the Food and Agriculture Organization (FAO) generated information on soy consumption by country (<http://faostat.fao.org/>) which was converted to per capita by dividing it over population of each country. Each country was treated as an individual in the analysis. Data were analysed statistically using Microsoft Excel. Linear and curvilinear bivariate regressions, and comparisons between means (t-test, unpaired, two tailed) were used. All countries for which information was available were analysed. Some countries were included in special groupings sharing specific characteristics like language or geo-political status. For particular analyses,

the number of countries included may have differed somewhat because all information was not uniformly available for all countries, as they may not have supplied data to relevant agencies (e.g. there were no data on soy consumption in Canada available from the FAO source). The groupings were Westernised English Speaking which included Australia, Bahamas, Barbados, Canada, Jamaica, Malta, New Zealand, South Africa, United States of America, European Union, including also countries of the European Economic Area and Switzerland, Austria, Belgium, Bulgaria, Czech Republic, Denmark, Estonia, Finland, France, Germany, Greece, Hungary, Italy, Iceland, Ireland, Latvia, Lithuania, Luxembourg, Malta, Netherlands, Norway, Poland, Portugal, Romania, Slovakia, Slovenia, Spain, Sweden, Switzerland, United Kingdom, and Latin America: Argentina, Bahamas, Barbados, Belize, Bolivia, Brazil, Chile, Costa Rica, Colombia, Cuba, Dominican Republic, Ecuador, El Salvador, Guatemala, Guyana, Haiti, Honduras, Jamaica, Mexico, Nicaragua, Panama, Paraguay, Peru, Trinidad and Tobago, Uruguay, Venezuela. Countries not included in those special groupings were also analysed when the entire world was considered.

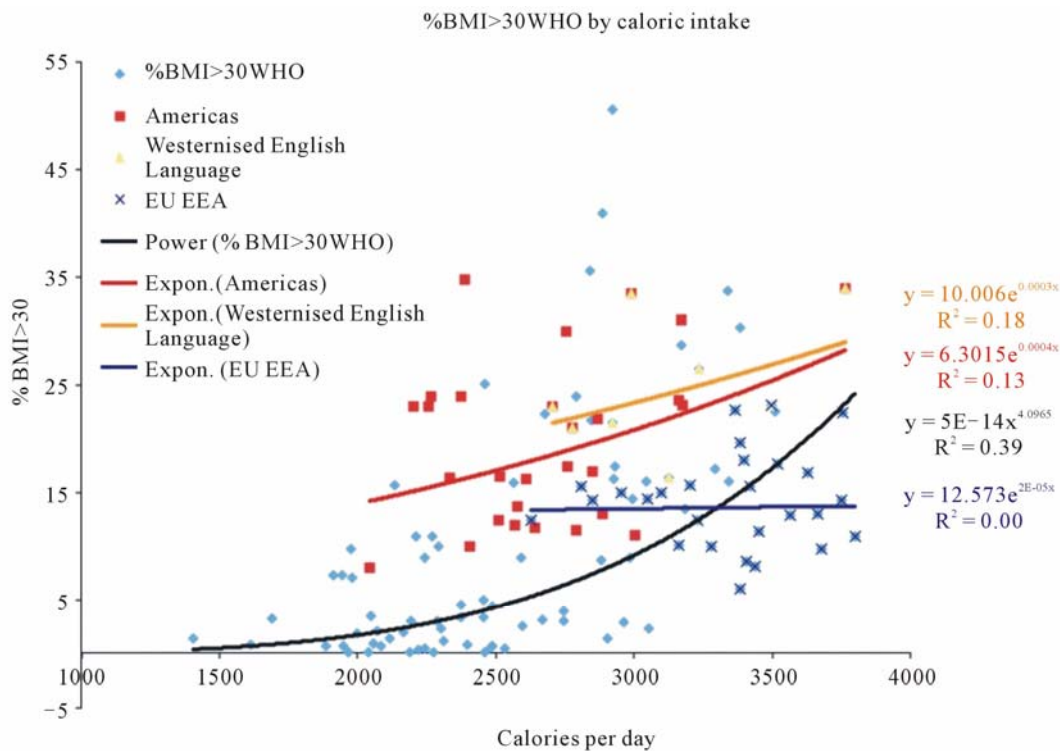
**2. Results**

The analysis shows that although the prevalence of obesity (% of people with BMI > 30 kg/m<sup>2</sup>), in general, increases with increased caloric intake and GDP, the rela-

tionship is different for different groups of countries (**Figure 1**). Especially, the prevalence of obesity is higher in the group of Westernised English-speaking countries (e.g. USA, Canada, Australia, NZ) and countries located in both Americas. The same is true for the relationship between the prevalence of obesity and GDP (**Figure 2**). Most notably, higher levels of obesity are observed at different levels of GDP and caloric intake in westernised English-speaking countries and in Latin America that have similar high soy consumption levels. In contrast, European Union countries, despite having, on average, higher GDP and caloric intake than westernised English-speaking countries, have only half the levels of obesity. This is directly attributable to consuming only half the amount of soy (**Table 1**, last column). The differences between prevalence of obesity and soy consumption in EU and other two groups of countries are significant (unpaired t-test, p < 0.05). BMI EU vs Westernised English speaking countries t = 2.77, BMI EU vs Latin America t = 2.43. Soy EU vs Westernised English speaking countries t = 1.96, Soy EU vs Latin America t = 3.98. Worldwide, the prevalence of obesity is exponentially correlated with soy consumption standardised on caloric intake (**Figure 3**).

**3. Discussion**

We have observed significant relationships between soy consumption and the prevalence of obesity. Despite the



**Figure 1. Relationship between prevalence of obesity and caloric intake, in groups of countries.**

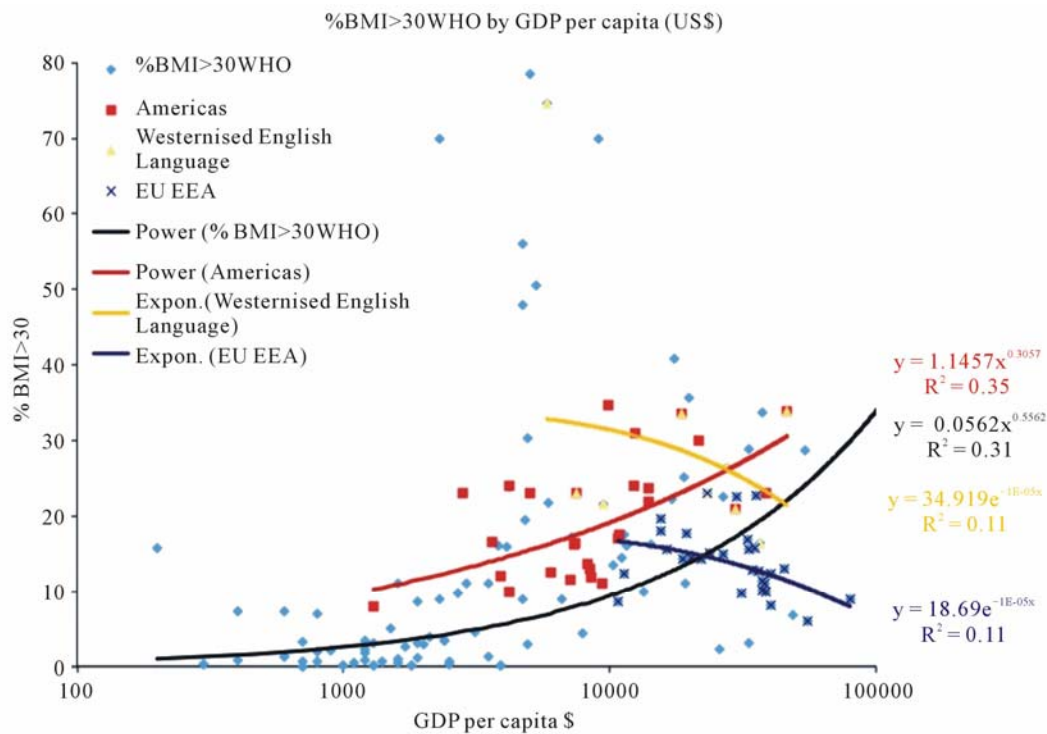


Figure 2. Relationship between prevalence of obesity and GDP in, groups of countries.

Table 1. Comparison of prevalence of obesity and soy consumption in variously grouped countries.

Country grouping	Contributing factors				
	N	%BMI > 30	GDP/capita	Caloric intake	Soy per capita
World total	142	15.6	14,372	2688	17.17
Westernised English speaking	9	30.4	22,755	3127	31.56
European union*	29	14.1	31,120	3347	16.07
Latin America	20	18.4	8,135	2640	28.92

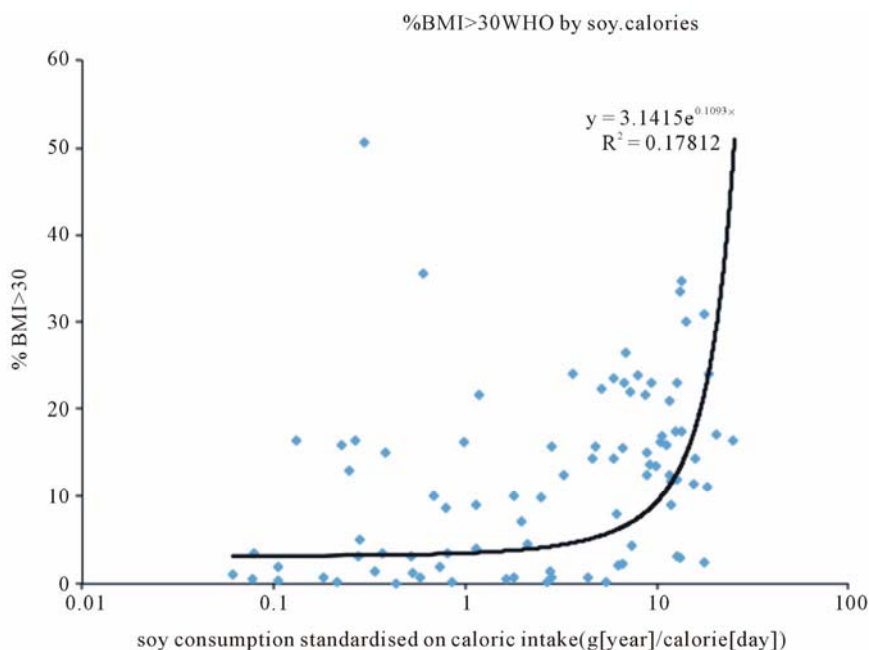
\*Includes EEA countries and Switzerland.

use of fairly crude measures of soy consumption and obesity derived from collective statistics, we have a range of interesting and statistically relevant results. Statistics reported by various countries may have various inaccuracies, in the case of larger countries averaging over the entire economy and the entire population may be questionable. BMI itself is an approximate measure of fatness and its reporting by country depends on local surveys of only a fraction of each population. (eg. Australian Bureau of Statistics surveys some 10,000 adults out of just over 20 million inhabitants)[26]. This explains scatter of the individual datum points.

The recognition of the worldwide problem of obesity by the WHO has put the issue fairly and squarely on all governments agenda as an item that needs special funding consideration and deliberation from many quarters,

besides biophysical wellbeing. Perhaps people in environments which experience frequent famine may see it as a positive [27], whereas in most countries or places, many obese people are not happy with their lot and in consequence develop a range of psychological problems and defenses, that also compromise their health placing a further burden on themselves and the people who care for them, as well as most publicly subsidized medical systems [28].

Much nutritional advice is still based on fallacious concepts promulgated from the 1950's, 60's and 70's without taking full advantage of the data, from long term studies that included and spanned those decades. The Frammingham Heart Study was one of the major studies that has now arrived at entirely different conclusions, advanced, by the misleading saturated fat and cholesterol



**Figure 3. Relationship between prevalence of obesity and soy consumption standardized on caloric intake by country.**

interpretations, aggressively promoted by Ancel Keyes which he derived from the hand-picked “Seven Nation Study” in the 1950’s. For example, the Frammingham Heart study that extended from the late 1940’s to the present day has enabled us to see that one of the real culprits behind heart disease and being over-weight, was not so much the natural saturated fatty acids and cholesterol, but the artificial trans fats (artificially saturated fats) much preferred by the cooking and baking industry using polyunsaturated oils and margarines. Margarines were preferred because trans fats gave long shelf life properties to their products [29,30]. The trans fats and margarines were produced from predominantly polyunsaturated vegetable oils, that were hydrogenated, of which the main one that was cheap to use, was soybean oil. In the last decade or so, with the gradual realization of the health benefits of the natural monounsaturated, and saturated fats new margarines have been developed. Some nutritionists and/or manufacturers, have pushed the strange idea, for example that by combining margarine with a small percentage of olive oil, makes the margarine a better product. It certainly is a more marketable product, but lest it escape us, all it means, is that any efficacy inherent in the olive oil is compromised by the artificiality of the hydrogenated polyunsaturated vegetable oil, which usually means soybean oil. With marketing duplicity, any label that hides behind a vegetable oil termed description where the manufacturer does not spell out the types of vegetable oils that are used, in any product under consideration, skepticism should prevail and possibly conclude that the oil used will most likely be the cheaper soybean oil. This could mean that soy consumption may be even higher

than the figures show.

Although soy consumption shows statistical coincidence with the prevalence of obesity, the awareness of numerous other factors that are involved in increasing fatness and obesity [3,10] such as caloric imbalance, genetic predisposition and metabolic syndrome still need to be taken into account. The fact that soy products often require other factors for palatability that are also implicated in the obesity equation [31] should not escape us. Nonetheless the statistically significant relationship that has been observed, between soy and obesity, strongly indicates and thereby requires that it should be scrutinised with much more specific attention than has so far been cursorily given.

Perhaps the way to combat obesity that seems to have escaped many analysts’ attention, would be to not only consider programs of multiple interventions, but to also take more seriously a far more careful analysis and research, of the composition of foods, *that were not part of our evolutionary history*, especially those modern foods containing soy that are promoted and consumed in the greatest amounts, which are now so widespread throughout the food supply.

#### 4. Acknowledgements

Financial support to MH was provided by the Wood Jones Bequest to the University of Adelaide.

DR reviewed the literature, formulated the hypothesis relating soy to obesity and participated in interpreting results and writing the text, MH did the data analyses and participated in interpreting results and writing the text.



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## **Part II.**

# **The Contribution of Soy Consumption to Childhood Leukaemia**

### **ABSTRACT**

Of the cancers in children, Leukaemia is the most frequent. Leukaemia data indicate that of childhood cancer which is about 30%, with the most frequent type being acute lymphocytic leukaemia at about 80%. Despite the numerous advances in modern medical science and advancing cancer research with better modes of treatment and improved survival rates, the fact remains, that the causes of child leukaemia are still largely unknown and the incidence is not decreasing.

Most research on the causes of leukaemia seems to refer to the possibility of chemicals in the environment or radiation of some sort, or viruses, or genetic factors (importance of family histories). We would make the suggestion from both the literature and statistics that the ingestion of food items related to unfermented soybean products, whereby the anti-thyroid, anti-nutrient, and endocrine disruption properties of soy may have in sensitive individuals a deleterious effect on their immune system. Such an effect, may in consequence compromise the child's body defences against many diseases particularly cancer. This is especially so amongst the very young, as well as amongst the very old. It is conceivable that despite the heavily promoted processed soy products, the strong

anti-nutrient properties in soy may in actual fact in the end outweigh the supposed benefits of cheap vegetable protein. The anti-nutrient effects on humans and mammals which may not be immediately observable in the short term are more likely to be revealed and quantitatively determined over the long term which may require performing studies that take into account quantitative observations over successive generations. To this end, we may need to consider animal models, over successive generations. It is not expected to be a short term study of a few weeks or months.

Leukaemias being cancers of the blood, most certainly involve the body's immune system. We need to observe as to what may be specifically prolific contributing factors in both the environment and in particular, the food supply. From our previous publication (Roccisano and Henneberg2012) on 'Soy and Obesity', we consider adding the strong possibility that despite all appearances from the positive spin and promotion for soy consumption, the anti-nutrient properties found in soy, may, in addition to obesity, also be contributing factors to the heavy incidence of leukaemia amongst children whose immune systems are not fully developed.

Leukaemia data on mortality were obtained from WHO information in 2008 and FAO information on soy consumption from 2005. Statistical analysis of average soy consumption, per capita, per country, where, numbers in childhood (0-14 years) mortality are observed, and where, such mortality occurred, show that greater soy consumption is related to leukaemia mortality. Specifically there is a significant Spearman correlation of soy consumption with child leukaemia mortality( $r=0.265$ ,  $n=66$ ).

## Introduction

By 1980, leukaemia contributed to the highest level of childhood cancer mortality in China's population (Xiao et al.1988). It has taken America 30 years to attain the dubious honour of becoming equally placed with China in having the highest levels of childhood leukaemia in their populations. Over the same time, both nations also developed other parallel problems of almost equal footing, in obesity, Alzheimer's and other diseases. The increasing incidence of cancer affects most developed and developing nations, showing no respect for political boundaries (Murray & Lopez 1997). Some have estimated that the number of cases of cancer will reach at least 20 million by 2020 (Mistry, et al. 2011), with cancer deaths increasing to 12 million (Siegel et al. 2012) and the incidence evenly shared between both developed and developing countries (Cancer Trends Progress report). Not only is the increase in diverse cancers a devastating burden for sections of the communities in developed countries, which have advanced treatment facilities, but it is an even greater burden for developing countries (Hanna et al 2010), which have very few resources they can draw upon to deal with what some describe as a global pandemic of cancer (*Lancet* 2011) that is destroying possibly as many lives as conflicts or wars. The balance of new cases with increased mortality in developing countries is steadily overtaking that in developed countries, owing to lack of adequate treatment in developing countries. Cancer seems to be predominantly a modern disease, possibly inflicted inadvertently by humans on humans by upsetting both the environment and exposure to unnatural substances that were not part of our evolutionary history. The Lancet Oncology Commission (2011) has prepared a large volume of work on the problem of "*delivering affordable cancer care in high-income*

*countries*”, acknowledging “*that cancer is a leading cause of death and morbidity throughout the world*” (p. 93). The magnitude of the problem is made global because cancer cuts across political lines and economic budgets, cultural and sociological issues, and health care systems and different worldviews, as well as, strategies for dealing with it. At times there are so many conflicting voices that it seems that part of the problem is to primarily discern a sensible path of light through the forest. Not every voice makes sense, but the Lancet Oncology Commission deserves recognition for its valiant efforts in clearing a path through the tangled confusion.

Using data from the cancer progress report from 2006, the U.S. National Cancer Institute stated that the United States spends about \$4.5 billion on leukaemia treatment in each fiscal year and more recently in the 2009 fiscal year spent \$220.6 million on leukaemia research. The heavy toll of cancer in general and leukaemia in particular is not restricted to the United States, but is of increasing concern for many countries. In the early 2000s, Australian childhood leukaemia accounted for more than a third of childhood cancers. The Australian Institute of Health and Welfare (AIHW) also released figures showing that leukaemia was the most expensive cancer to treat, with lifetime treatment costs of \$51,000 per case in Australia a decade ago (Murray and Lopez 1997). And although there is an increased survival rate, the cost has not gone down, but has become even more expensive. As well as the very emotional and psycho-social burden of not knowing how, their child became afflicted, families also take on most of the high cost of treatment, which by 2005 had increased about \$58,000 per child. We would expect that with Australia’s advanced medical knowledge and technical development it would be highly unusual for many other

countries (even developed countries) to achieve the same survival rates in Australia at less expense.

In 1996 the Australian Government, through the AIHW, recognised and made cancer, a National Health Priority Area. This response was necessitated due to “concerns over the rising incidence of many cancers, and the burden of mortality and morbidity due to cancer (leukaemia), was placing on the Australian population”... The statistics also revealed “a small but significant rise in the incidence of cancer among Australian children”. And compared with other cancers, for all other ages, leukaemia was far more prevalent in the 0–14 age group (Mellstedt 2006). The increasing incidence rates were obtained from the National Cancer Statistics Clearing House. The data obtained indicated that, by 2001, leukaemia was the most prevalent cancer for children in the 0–14 age group, and more specifically, with the highest incidence occurring for children aged 0–4 years.

This phenomenon is disconcerting insofar as most leukaemia cancers are usually expected increase in incidence with increasing age as, historically, after puberty cancers only start to rise gradually in incidence, in adults, after 45 years of age. The data from the United States National Cancer Review via the SEER (*Surveillance, Epidemiology and End Results*) Cancer Statistics Review shows that the risk of contracting acute lymphoblastic leukaemia (ALL) is greatest in the first five years of life, reflecting the Australian experience, and “an increase in occurrence isn't seen again until later in life” About 3500 children aged 0–7 were expected to contract ALL in 2010 in the United States, according the American Cancer Society.

The incidence rates are projected to increase slightly not only for leukaemia but for a number of other cancers as well. Although the percentages indicate only small increases, for children the statistics have not levelled out or dropped away and the incidence statistics are still increasing, despite the advances in cancer research.

On the question of whether or not childhood leukaemia can be prevented, the American Cancer Society, along with other similar organisation, bluntly admits on its website that although adults can minimize obvious known risk factors and adopt healthier diets and fitness activities, it is very difficult to invoke such changes for the prevention of the majority of cancers in children at present. Put in simple terms, young children have not really had a chance to develop a lifestyle that warrants modification to changing to attain a better health or cancer preventative outcome. Most children (0–7) with leukaemia have no known risk factors, other than perhaps, to what their parents subject them, or what they encounter in their immediate environment, so there is no sure way to prevent their leukaemias from developing. In Australia as at 2009, however, improved methods of treatment gave some hope, with mortality rates dropping slightly with an average increase in chances of survival of 0.6% per year.

Despite these very encouraging improvements in childhood leukaemia, the costs are still quite prohibitive in two major aspects. First, the obvious cost already alluded to is a large economic drain. Second, there is the personal human cost, which is often quickly glossed over, in an effort to display a positive survival rate, no matter how small. The issue essentially revolves around the question: With all the advances and successful treatment of



such cancers, why is it that for some cancers the incidence is maintained and, in some instances, keeps increasing?

## **Possible causes**

Cancer seems to be predominantly a modern disease possibly inadvertently inflicted by humans on humans, both by upsetting the environment, and by exposure to unnatural substances that were not part of our evolutionary history. The disconcerting observation is the unfortunate increasing affliction of infants and very young children with leukaemia, particularly ALL, when infants have not even had time to develop a lifestyle that could in any way be a contributing cause of their illness. Despite the negative results so far in the search for the cause, or causes of leukaemia, as leukaemia is responsible for greater mortality in children than other forms of cancer, the search for answers is both a scientific and moral necessity. To this end, there have been many suggestions and theories as to the causes for leukaemia. For many decades most authorities have concluded that there is no known cause of childhood leukaemia and therefore no rational defence or intelligible strategy to prevent it from occurring among infants. Despite the many advances in medical research and the better medical care that has greatly improved child survival rates, the incidence of childhood leukaemia and related morbidities continues to rise. The search for a cause of leukaemia, whether one or a combination that acts as the trigger, needs to continue so that we may one day be able to prevent childhood leukaemia from occurring and recurring. The following discussion outlines possibilities raised in the literature, with a focus on childhood vulnerability.

The incidence of cancers and leukaemia occurred for many people and was highlighted by the work of the Polish Nobel prize winner Madame Curie, whose life was eventually cut short by her exposure to radioactive materials (AIHW 2001). The connection was made by people who worked with hazardous materials and associated elements that generate ionizing radiation even (Murray R, et al 1959) before the cellular disrupting process was understood. The possibility of the medical use of X-rays and CT scans disrupting cellular development while the child is growing in the uterus is no longer under any dispute (SEER 2010; AIHW 2004, 2005), and nursing mothers are discouraged from breastfeeding their infant if they have had a CT-scan, until the dye has passed out of their bodies (Brenner D 2002). Unborn infants may also be subject to X-rays if the mothers do not know that they are pregnant. In addition, therapies involving organ transplants, the use of immunosuppressant drugs or standard chemotherapies, and radiation therapies also greatly increase the chances of later development of cancers and leukaemia. In short, any therapy that affects the mother's immune system and consequently that of her child, will greatly increase the chances of developing of any type of cancer, including leukaemia (Cancer Trends Progress Report). Although the link with radiation exposure, fallout (especially in the wake of nuclear accidents) or even naturally occurring ionisation in the environment and so on is unquestioned, a number of other possibilities have come under scrutiny. Children with Down's syndrome and Klinefelter syndrome, seem to also have an increased disposition to developing leukaemia.

Over the last decade more attention has been directed to the possibility that chronic inflammation provides an environment in which cancers in general and leukaemias in

particular can develop, presumably because of viruses attacking the immune system. If the mother has had an illness during pregnancy, the treatment protocol alongside the virus itself could also affect the developing foetus (Belson M, et al. 2007; McNally and Parker 2006; Wakeford R, 2004).

Environmental pollutants that have an excess of xenoestrogens and other oestrogen mimickers, such as pesticides, herbicides and defoliants( Ma X, et al. ), are known to have endocrine and/or hormonal disruptive behaviour(Metayer C Buffler PA, 2008). Children of Vietnam Veterans (AIHW, 2001) who were either directly or indirectly exposed to agent orange or similar chemicals may fall into this category. If this connection with leukaemia is valid, then it indicates that the father is just as important (certainly not irrelevant) as the mother in the leukemogenesis process .Damage to the spermatogenesis process in any way will also compromise the early stages of rapid cell development of the foetus. For example, it could be reasoned, that if the father has had radiation exposure or X-ray treatment of the hip region shortly before conception occurs, the integrity of the sperm and genetic material may be compromised.Other aspects of the father's occupation which may involve exposure to known cancer causing agents including farm chemicals and herbicides(Perez-Saldivar et al 2008) Another consideration is the interference caused of many domestic chemicals that can disrupt the endocrine and immune system of the bodies of both potential parents. These are contained in a large range of household products, including hairsprays, shampoos, deodorants, dry cleaning fluids, and food containers, which also have an abundance of estrogen mimickers (Fucic 2012 et al.). Parents who work in the paint or plastics industry (Perez-Guzman et al 2008)or being involved in industries that are classed as hazardous

occupations are more likely have children who develop some form of leukaemia, especially if the one or both of the parents' reproductive systems are affected (Colt & Blair 1998).

Others have put forward the scenario of picking up an infection that leads to child leukaemia in a day care facility. Picking up an infection(Chan LC, et al.2002)may initiate a compromised immune system paving the way for cancer to either begin, or if it is already there, to spread. To extend the childcare possibility a step further is the interesting possibility that such cancers may be connected to socioeconomic status, especially for children who have been fed inadequate nutrition through either ignorance or poverty. (Borugian et al. 2005)

Another scenario is the possibility of symbiotic groups of identifiable cancer cells proliferating in a young vulnerable body well supplied by the hosts blood supply. (Robinson DH 2005, Wainwright M 2000). Cancers can display rapid growth in the context of a weakened immune system that allows the development and growth of a symbiotic combination between the host mammalian cells and a resident or invading plant bacterium.

All the above possibilities are just that. They are the diverse range of possibilities that we can consider, that if successfully tackled and eliminated individually as disparate and unconnected contributors to child leukaemia, still leaves the incidence increase of leukaemia.

Clear connections do not appear to be readily obvious for any accepted definite link to a general or specific cause of leukaemia, especially ALL in children. If there is a consensus, it seems that it could be anything, which is tantamount to declaring that we still don't really know. Any search of the growing citation Medline or Pubmed list of *150,000 plus* citations

on leukaemia indicates that the search for acceptable answers is ongoing, no matter what combinations of search items are used. At present we seem to be grasping at any straw. There is no clearly defined known general cause of child leukaemia and therefore no known rational defence strategy to prevent its occurrence. The previous work on soy contribution to obesity shows that the human food supply, needs to be revisited as a potential source for the cause and closely monitored for foods that have endocrine, hormone and immune disruptive properties (Roccisano and Henneberg 2012), because it is at this level that the developing foetus will draw its nutrients and derive its growth and future immune system development. An abnormally depleted or disrupted nutrient environment may provide a plausible explanation for the development of infant leukaemia.

Many children who are both short- and long-term survivors of leukaemia manifest associated debilities arising from the treatment protocol, such as disrupted bone metabolism in the form of regular bone fractures and loss of normal gait and muscle control. To this we can add the further complication of sterility and increased likelihood of becoming obese. In reverse, a study at the Saban Research Institute of Childrens Hospital Los Angeles purportedly shows that "Diet induced Obesity Accelerates Leukemia", whereby being obese generates the possibility of an increased chance of developing leukaemia. (Yun et al. 2010) The authors found that there was an increased risk of ALL in older mice, which is consistent with the SEER numbers of increasing incidence with the older age group. Although here we are not looking at age or ageing as a factor, the incidence of leukaemia in the older age group is not irrelevant. We could concur with the conclusion arrived at by Mittleman with Yun et al (2010) *in that "The data indicates that some unknown causative*

*factor may set off the leukaemia process*". Dr Mittleman observed that the causative factor is "**some hormone or other factor** or perhaps the fat itself, that signals for the start of the *leukaemia process*." (flagged in *Science Daily* Sept 10, 2010). The work of Roccisano and Henneberg (2011, flagged in the Australian media in July 2010, shows correlations between the now universally consumed soybean in its myriad forms pointing to the soybean's anti-nutrient and hormone disruptive content as a possible obesity catalyst. The soybean is ubiquitously placed and heavily manifest in nearly all manner of industrially processed food, which is frequently and often *unavoidably* consumed throughout the global food supply.

This is in partway consistent with our previous results and discussion, which indicated that the anti-nutrient content and phytate inhibitors, protease inhibitors (Liener IE, 1994) and other hormone and endocrine disruptors in soy seem to correlate with the contribution of soy to obesity (R&H.2011), simply because such compounds in soy deny the body's needs to maintain an adequate access to essential nutrients. In the first instance this creates an inflammatory response, thereby building an excess quantity of fat cells and encouraging the obesity syndrome. Second, those same anti-nutrients, inhibitors and hormone disruptors create an environment that compromises the body's immune system, laying the path open to chronic inflammatory responses that can be associated with cancers or some form of leukomgenic development, and possibly many other morbidities, cancerous or otherwise. The obesity–leukaemia connection/association has also been found with a study that indicated that obese women tend to have a higher leukaemia incidence (Ross J, et al 2004). That connection, has been made, and

obviously has corroborative implications with leukaemia and obesity for the Yun-Mittleman leukemia (2010) research study.

## **Hypothesis**

Industrially produced and processed food from soybean may contain chemicals that alter or interfere with normal metabolic and hormonal processes, thereby disrupting the endocrine and immune systems of the human body in vulnerable individuals, especially prenatal foetus and postnatal infant and young children. The pervasive use of soy throughout the food supply cannot be ignored, as soy is also a strong goitrogen and contains many anti-nutrients in stronger quantities than other vegetables; these are not so readily removed by normal cooking processes. In short, the general consumption of industrially produced soybean food products may contribute to the increasing incidence of childhood cancers in general and the blood cancer leukaemia in particular.

## **Materials and methods**

Some have claimed that early intake of soy protects against breast cancer (see, for instance, Messina (2009)). We decided to look at the consequences of such a recommendation, if applied in practice, to the soy-consuming societies. In such instances “early intake” could be interpreted as a child being fed via a number of means such as soy infant formula, soy influenced baby foods and any number of soy-processed other food items available to children and lactating mothers from the supermarket shelves. This would

be quite aside from indirect ingestion of soy anti-nutrients from maternal milk if the mother is also consuming soy products.

Information was obtained from WHO on mortality from leukaemia per country. Soy consumption was obtained from the FAO, and GDP per capita by country from the UN. The WHO differentiates between reliable information, reasonably estimated information and extrapolations based on data from other countries. Only reliable and reasonably estimated information was used here. Data on soy consumption were converted to kg/year/person using population sizes of respective countries. Owing to skewed distributions of mortality data, Spearman rank correlations were used to study the connection between mortality and other variables.

## Results

Mortality from leukaemia in countries providing reliable information and those with reasonable estimates was correlated with GDP: in age group 0–14 this correlation was negative;  $\rho=0.5$ . In age groups 15–59 and 60+ correlation was positive:  $\rho=0.25$  and  $0.23$  respectively. This indicates better health care in wealthier countries. Soy consumption correlated significantly with GDP,  $\rho=0.43$ . After controlling for GDP, mortality from leukaemia correlated significantly with soy consumption in all (N=89 countries that had reliably estimated data on mortality from leukaemia:  $\rho=0.25$  (p, 0.05), as shown in Table 1 below. There was no significant correlation in other age groups.



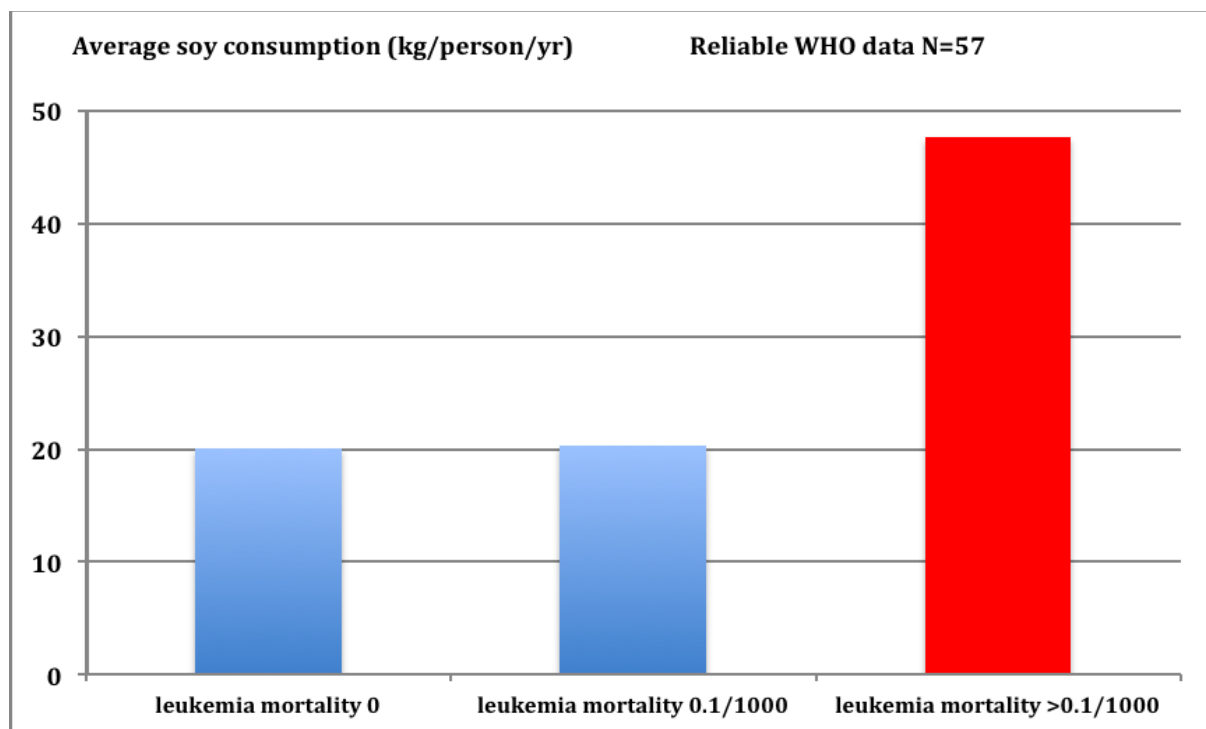
**Table 1. Childhood leukemia mortality**

<b>Childhood leukemia mortality</b>	<b>N</b>	<b>average</b>	<b>sd</b>	<b>N</b>	<b>average</b>	<b>sd</b>
Zero and 0.1/1000	52	20.0	15.9	69	19.6	16.2
More than 0.1/1000	5	47.6	31.8	20	24.4	26.6

Investigating further correlation between soy consumption and leukaemia mortality in the 0–14 age group, we found a significant direct correlation between mortality and soy consumption:  $\rho=0.31$  ( $p<0.05$ ) in countries providing reliable mortality information ( $N=57$ ). This relationship became stronger after controlling for GDP effects:  $\rho=0.42$  ( $p<0.01$ ).

Soy consumption was on average lower in countries with no mortality from leukaemia in the 0–14 age group or low mortality for (0.1 per 1000) than in countries with higher levels of childhood leukaemia mortality. The difference between average soy consumption in countries of no or low mortality and those having higher mortality was significant at 0.05 level (one tailed  $t= 1.92$  Wilcoxon [Mann-Whitney] U test  $z=1.64$ ) in a sample of 57 countries with reliable information ( $n_1= 52$ ,  $n_2=5$ ). It persisted when countries with estimated information were added to the sample, increasing N to 89, but was not formally significant (fig. 1 and fig. 2).

**Figure 1. Average soy consumption (kg/person/year) in countries with no childhood leukaemia mortality, and those where children died of leukaemia**



### **The inadequacy of soy as a food source**

Not only do the phytates in soy present a problem in blocking the body's access to essential trace minerals as in the aforementioned depletion of iron, iodine, calcium and zinc, but there also is a mineral competition for absorption between zinc and calcium, which affects the health status of both mother and child. The delicate balance that enables good health can easily be disturbed by over-fortification of one element over another. This again supports the contention/hypothesis that the phytate binding and mineral absorption disrupting properties of soybean foods, can contribute to the leukomogenic process. Many soy milks are calcium fortified, not so much because of manufacturers' concern for our better health, but more so because of the need to compensate for the calcium-binding

phytates, and the metabolic competition between calcium and zinc — if the body's zinc reserves (which can be also caused by the phytate binders in soy) are below par, zinc uptake will take precedence. Because the phytic acid in soy readily acts as a binder to calcium, soy can only be a source of calcium under artificial conditions of extra calcium fortification.

Heaney et al. (2000) still concluded that despite calcium fortification, *“calcium-fortified ‘soy milk’ was not as good a calcium delivery mode compared to cow milk”*. In a recent Australian study (Cheung ALTF 2010) there was the clear confirmation that although soy milk does not match the calcium profile of cow's milk. The attempt to bring soy milk up to comparison successfully with cow's milk requires a heat treatment (which can often denature many proteins rendering them useless) with a proprietary calcium phosphate supplement. In a later study, Cheung stipulates that the attempt to use fermented calcium enriched soy milk did not have any more encouraging results for hoped for, improved calcium absorption (Cheung ALTF, 2011).

We can no longer ignore the repeated findings from the research data at the micro cellular level, that phytate binders block the body's ability to absorb essential mineral nutrients such as Ca, Fe, Mg, I, Cr and Zn (Hurrell RF, et al. 1992), (Cook et al. 1981), (Zhao XF, et al. 2003). An extra problem occurs, in that on the one hand soy inhibits many essential microelements while at the same time enhances the absorption of undesirable heavy metal micro antinutrients such as cadmium (Eklund & Oskarsson, 1999), mercury, aluminium and the like. From another perspective phytates could also be seen to be beneficial in that the removal of excess iron as well as overloads of other micro mineral nutrient that are occasionally problematic. However to expect phytates to restrict cancer development has

been met with disappointment.(Takaba K et al. 1997). Furthermore, there have been attempts to fortify wheat flour with iron(Fe) to resolve a deficiency in iron in many nations (Hurrell et al 2010). Here again is an interesting situation in that wheat flour has to be fortified with iron because of the generally understood problem of iron deficiency in many populations, (78 countries were assessed in the Hurrell et al. 2010 study).However, when the contents of most wheat products including bread and most bakery products are examined, it will be observed that the majority have some form of soy product in them, by which it could be seen, as completely negating the costs and process of fortifying the wheat flour with iron, in the first place, effectively nullifying the iron fortification by the phytate binders in soy. The need for iron fortification is obviously necessary from a simple requirement as part of the blood formation process –without the ‘haem’ there is no haematogenesis , with very obvious consequences for cancers of the blood. In short another avenue and reason to seriously consider excluding soy and soy products from the maternal diet , especially for vulnerable individuals. Furthermore, some regarded (Stabler and Allen 2004, Allen LH, 2009) the worldwide problem of vitaminB<sub>12</sub> deficiency as co-related especially for mothers breast feeding their infants.Lack of B<sub>12</sub> in both mother and child is a high risk problem for both, in that the child in a vegetarian or vegan situation may readily develop common B<sub>12</sub> illnesses including growth defects as well as anaemia or pernicious aenemia which on occasion have presented and misdiagnosed as leukaemia and treating a nonleukaemic individual with devastating consequences associated with cancer therapies as leukaemia,(Lennard A, 1990) depending on the severity of the B<sub>12</sub> depletion. The necessity of a proper complement of available nutrient supply including vitamins from the B group as well as C and E, along with niacin and folate, are

necessary for optimal DNA integrity, of which the majority of these essential micronutrients is required for normal foetal growth should not escape our attention, especially in avoiding DNA and chromosomal disruptions, and damage in the process of foetal development (Ames BN 1999). Furthermore, Ho E, et al. (2003) indicate that oxidative damage to the DNA also occurs when there is a deficiency in zinc. The phytate binders and protease inhibitors, by their actions, in restricting the very nutrients necessary for proper cell and DNA development and replication, paving the way for damage to occur in the DNA. Further evidence of soybean's disruption refers to the trypsin and protease inhibitors in soy, which inhibit the body's ability to process essential proteins, thereby again disrupting at the cellular level, the use of the proper building blocks for DNA and chromosomal integrity, as well as interfering with the building of a normal blood profile

As each cell replicates, forming new cells, the DNA must be copied, and under normal conditions the process works well. However, when mistakes are made, either naturally or "induced", there are enzymes in place to repair and maintain the standard integrity of the DNA. These nuclear enzymes have the job of repairing transient breaks in the DNA and/or other disruptive breaks in the DNA strand, or even cutting and splicing the DNA strand when it gets far too twisted up, and/or is subject to any other unnatural changes in the topology of the normal developing molecular structural process. This is particularly significant during rapid cell division and growth in early foetal development. The topoisomerase enzymes can even assist to the extent of allowing double helices to pass through each other during the repair process, thereby preventing any chromosomal aberrations from occurring.

Hengstler et al. (2002) reported “an almost 10-fold higher risk of infant AML leukaemia for mothers consuming relatively high levels of topoisomerase II-poison containing foods”. Such foods include coffee, wine, tea, and some fruits and vegetables but predominantly soy and soy products, which have very high levels of topoisomerase II toxins, as well as other aforementioned anti-nutrient cofactors such as phytate binders and trypsin inhibitors and high levels of isoflavones, genistein and other endocrine disruptors. Identification of bioflavonoids which exist in some foods in the maternal diet may create in some instances nonreversible chromosome translocations causing DNA damage (Strick R, et al.2000) and eventual aberrations if not cell deaths. These are implicated in the generation of a number of illnesses including a number of cancers in general besides the possibility of adding the particular case of leukaemia to the list. (Hengstler et al.2002) These topoisomerase poisons and inhibitors are readily found in soy and soy products. Unfermented industrialised soy foods have very high levels of topoisomerase II toxins, as well as other anti-nutrient cofactors such as phytate binders and trypsin inhibitors and high levels of isoflavones, genistein and other endocrine disruptors, which are implicated in the generation of a number of illnesses as well as a number of cancers, possibly including leukaemia (Hengstler et al. 2002) conclude that:

*If there is even a partial connection between dietary exposure to topoisomerase-II poisons and infant leukaemia... care should be taken to reduce exposure to such critical foods as well as other foods that containing topoisomerase type II toxins during pregnancy (p. 8)*

## Genistein

The highly active topoisomerase type II poison, genistein, readily induces DNA breakages even in colon cancer cells within 60 minutes of application (Salti et al. 2000). Others have confirmed the fact that genistein readily mediates double strand DNA breakages in mammals (Markovits et al. 1989).

The importance of the maternal diet in relation to soy consumption may also require experimental studies over successive generations, which can be readily facilitated by appropriate animal studies. Unfortunately, many studies that have gone into print in support of the general consumption of soybean are disappointingly almost *always* of a short-term nature!. However Hilakivi-Clarke L et al. (1999) traced the influence of soy diet consumption in subsequent generations, finding a continual occurrence of breast cancer in offspring of subsequent generations. The indication is that higher than normal estrogen levels at inappropriate times can increase the chances of developing breast cancer. Although topoisomerase type II poison, genistein, which may have far reaching consequences in formation breast cancer, in successive generations of rats. It stands to reason, that similar effects down the line can in effect be attributed to the generation of cancer of the blood. Have these consequences been ignored or glossed over for short term economic gain from the highly marketable soybean products? To find in animal models that mutations or cancerous growths in successive generations or offspring can be attributed to soy products is disturbing. Proper DNA integrity is crucial to mammalian development and the risk of mutation is highly influential to this integrity. It probably only needs a few errant steps in the early DNA replication process , even without the destructive influence of the

genistein topoisomerase toxins, for the mutation to do damage throughout the body. As leukaemia is a blood cancer it will spread in similar ways to other afflictions by the action of the blood as a medium as the blood travels and circulates throughout the body. The decatenationary repairing activity of mammalian topoisomerase II is inhibited by genistein, which also inhibits tyrosine kinase, (Peterson G 1995), (Yakisich ED et al 1999) and , which could be a 'seed factor' that may affect learning as well as memory and dementia later in life(O'Dell et al 1991)(see also Part III). At the same time genistein can induce cleavages independent of intercalatory requirements (Markovits et al. 1989; Yamashita et al. 1990; Mayr 1992). Testing genistein with different animal models (inc hamsters etc) also results in strand breakages in the DNA (Kulling & Metzler 1997) The inhibition of tyrosine kinase by genistein, resulting in mistakes in chromosome structure, has also been observed in insects and not restricted to mammals (Nicklaus et al. 1993).

### **Phytoestrogen properties of soy and disruption to DNA replication**

In broader terms, soy also disrupts the hormonal balance in mammals and humans by the phytoestrogen content mimicking the normal oestrogen molecules as well as clogging up the normal oestrogen receptor sites. The bioflavonoid compound genistein, as found in soybeans and other vegetable sources of topoisomerase II toxins (although not as strong as soy) can, by subjecting the maternal womb nutrient environment to exposure even at low doses in the diet, can be considered as hindering the proper development of the foetus, resulting in the possible occurrence the conditions that may initiate foetal/infant leukaemia. The anti-nutrients in soy can therefore increase the disruption to DNA replication, especially if the topoisomerase type II enzymes are restricted from doing their DNA repair jobs,



making the unborn child very vulnerable to the leukaemic cancer process. This is especially so for pre-natal and post-natal children, as the infant's immunity is dependent on the mother's immune system even for a time after birth through the nutrition profile found in mother's milk, which no amount of imitation chemistry can replace (Heaney et al. 2000). Maternal pre-birth diets can not be ignored to have significant influence on the incidence of leukaemia in newborn and children in the 0–4 age group. The continued use of soy foods in the maternal diet also would seem to be most unwise (Hilakivi-Clarke et al. May 1999), (Hilakivi-Clarke L, deAssis S 2006), (Hilakivi-Clarke et al. 1999 Sept – Oct 1999) in the light of the negative effects that soy anti-nutrients also generate in the reproductive system of the mammalian animal model (Anderson et al. 1997). The controversy surrounding the soy formula composition will continue.

The prescription use of the female hormone diethylstilbesterol (DES) to prevent miscarriages was terminated (in the US) in the 1970s because of the strong association observed with the high occurrence women prescribed the hormone having daughters who subsequently developed vaginal cancer. The sons also, did not escape abnormalities generated by the extra oestrogen through their developmental/foetal stage. These cancers as with many others are never picked up or identified with human short studies. The soy industry has also never produced or released any long term studies espousing its products. An animal model leukaemia research at the Rockefeller Institute revealed, in utilising the Rockefeller Institute Leukemia Strain (RIL), that while female rats had a higher incidence of leukaemia with RIL than normal male rats, once the males were castrated they attained almost the same levels of leukaemia as the females. Removal of the ovaries, however, did

not change the statistics for the females. Exposing normal, intact males to DES however, generated the statistics numbers exhibited by female controls (Murphy & Sturm, 1949). Indeed, the authors concluded that the result was caused by the interference of the extraneous DES female hormone (upsetting a proper balance) with the male hormone. The action of these hormonal compounds, whether they are present, nonexistent or in excess, have far-reaching consequences. Newbold et al. (2001) found that with genistein, being far more freely dispersed throughout the food supply, it was likely to be an emphatically stronger cancer-producing agent than DES (as if DES wasn't bad enough) (Newbold et al. 2001) Clearly there is a strong need for further experimental assessment to both evaluate and establish appropriate markers, as there seem to be more factors to the effects due to genistein.

Contrary to the above experimental work is the view that early intake of soy may be protective against breast cancer (Messina & Hilakivi-Clarke 2009), (Lamartiniere CA, 2000). Such comments in favour of early introduction of genistein can only be made, if ignoring data, which indicates that when genistein is removed from the diet, tumours are observed to regress (Allred et al. 2001). Contrary to support for dietary genistein by Messina and Lamartiniere (2000), it was observed by Ju et al. (2002) who reported that including genistein in the diet overwhelms the breast cancer inhibitory effect of tamoxifen on MCF-7 cancerous growth, clearly an unacceptable consequence of continued soy consumption. Messina et al. also ignored or failed to take into account the results reported by Newbold et al (2002) that the data show the very opposite, in that, introducing genistein early in an infant's development, there is an increased later chance of also developing cancer of the

uterus(Newbold R et al. 2002) in the same way that DES after many years had to be banned for humans, (even though it was still used in industrial farming as a growth hormone). Many years after it was banned for humans the EEC also banned meat and meat products from US, to safeguard their populations from unregulated hormone usage in the US meat and livestock industry.It took 40 years,culminating in the 1970's to prove the carcinogenicity of DES before it was banned for humans , and yet the US. meat and livestock industry still use it regardless in its export trade.

To introduce genistein early, quite aside from the above indicated possibility of generating breast, vaginal and uterine cancer developments in subsequent offspring, also neglects to account for the data that indicates that genistein has also been found to inhibit and disrupts the nuclear enzyme topoisomerase type II action, specifically in feotal DNA repair, causing strand breaks in the DNA molecule to remain unrepaired, thereby indicating the strong possibility of leukaemia development (Abe T. 1999), (Hengstler et al. 2002). It should only require these few instances of the reported cancer causing properties of genistein to reconsider the continued consumption of soy and soy products, especially in the unfermented form. The few mixed results seized upon by soy advocates as being beneficial for breast cancer prevention in recommendations by Messina et al.(2009)are again typical of the soy industry rushing ahead making claims without waiting for the science to fully outwork a proper understanding association of soy and cancer in this context.(Trock BJ,Hilakivi-Clarke L, Clarke R, 2006).To promote soy as beneficial in preventing breast cancer by early ingestion does not follow the principle of least harm as well as running the risk of repeating the disastrous polyunsaturated/ saturated fat/cholesterol nutrition mistake made 70 years ago

In a recent controversial media release, the New South Wales Department of Public Health warned that any members of a family that had a family member with cancer should avoid soy consumption. Perhaps such warnings should also include those with family histories that include leukaemia as well as hormone driven cancers.

We can no longer ignore the repeated findings of the various research analysis at the micro cellular level showing that phytate binders block the body's ability to absorb essential mineral nutrients such as Ca, Fe, Mg, I, Cr, Zn Not only that, but soy also disrupts the hormonal balance in mammals and humans via the phytoestrogen content, mimicking the normal oestrogen molecules as well as clogging up the normal estrogen receptor sites. Other compounds in soy, such as the topoisomerase-II poisons, have been linked with immune disruption again at the cellular level, interfering with the DNA replication process as well as inducing chromosomal aberrations (If the reproductive system of either parent is compromised, it is not unreasonable to consider that the product of that reproduction would also have an increased chance of being likewise adversely affected.

Perhaps the same argument should be applied to the father's diet, which should also be free from soy consumption(as well as alcohol and tobacco) prior to conception, with the mother avoiding soy products for the duration of her pregnancy and until the child is weaned, including the use of soy formula for children 0–4years. From the list of causes that havev gained credence in that the paternal influence can also have an effect on the unborn child, for the same reasons that indicate the parent working in a toxic environment can have similar negative influences, depending both on diet and/or compromising work environment. For adult cells and immune system should have stabilised, so are less

susceptible to cellular damage, but the developing foetus in a depleted nutrient environment is in no such position to either accommodate a nutrient depleted environment as well as avoiding any cellular damage which the mature immune system of an adult can tolerate. This situation can be considered even more crucial if the foetal nutrient environment is both depleted of essential nutrients but also contaminated with a toxic mix from both the diet that may include soy topoisomerase poisons (genistein) etc. etc., as well as toxins from smoking and alcohol.

It has been clearly shown from other contexts, that the expectant mother should not only be counselled to avoid alcohol, smoking and consumption of foods that contain heavy metals (particularly some seafoods). It stands to reason there can be many other consumable items that the expectant mother should be taking into account. From our perspective these would include soy; noting that the chemicals and alkaloids in tobacco and other items can have some estrogenic compounds that behave similar to soy. A responsible prospective parent should consider avoiding a range of processed foods with such compounds for at least the duration of the pregnancy if not only for a period both before and after birth for the mother, as well as some time before conception, for the father, (as he would/should be counselled if his occupation involves toxic chemicals) thereby lessening the chance that these compounds will compromise the immune system of the unborn child. Perhaps, in the case of soy, experiments may have to be designed to determine, what sort of time is necessary for such compounds to clear the mother's body before conception should be considered, especially in today's modern day polluted environment. Furthermore in consideration of the father, with the current (controversial) worldwide reduction in

sperm quality, the father's contribution prior to anticipated conception will also have to be taken into account.

We do realise and acknowledge the multiple effects that many external processes from the environment can have across all ages. For children in the 0–4 age group, the results reveal that the continued promotion of the ingestion of soy and soy products should be reconsidered and treated in the same way that alcohol and smoking have also finally been acknowledged as (even indirectly via the maternal diet intake) generating many childhood morbidities. Soy with its hormone, endocrine-disrupting and phytate nutrient blocking properties, may eventually be acknowledged as directly or indirectly contributing to childhood leukaemia and some other cancers, as well as foetal morbidity.

### **Vitamin B<sub>12</sub>**

Although it is now widely acknowledged that it is more important to control high levels of homocysteine than cholesterol, for heart disease (McKully,1999 ). Homocysteine has come under increasing scrutiny. High levels of homocysteine may also be correlated with breast cancer and ALL. In this context of soy consumption, the B<sub>12</sub> analog in soy, consumption of which, not only contributes to anaemia, but by occupying the B<sub>12</sub> receptor site may indeed actually generate a far worse problem in pernicious anaemia. Because B<sub>12</sub> is a functional building block crucial for the synthesis of DNA, having an excess amount of the wrong type in place may also set the context for further aberrant chromosomal development. Many vegans whose whole source of protein comes from soy products often have to receive their B<sub>12</sub> via injection, since the B<sub>12</sub> from soy is non-bioavailable for humans and possibly worse than useless for the developing foetus.

This may also be exacerbated by the topoisomerase-II poisons alluded to earlier. These aspects of soy's destructive activity in the past have for better or worse been kept low key and often separate, or have been treated as isolated and possibly irrelevant and have thus far been considered somewhat innocuous and of no consequence to human health. However, when different research results are put together, a different picture begins to emerge and a very damning and inescapable profile builds, that eventually it will be realized that soy is not the going to be the solution to the world's future protein source; but is rather generating ,a serious burden on the medical responsibilities of all governments. The financial burden is becoming so unsustainable that even developed countries are having a hard time managing the proliferation of cancers for themselves, both financially and socially. This problem is becoming globally apparent.

### **Soy implicated in leukaemia**

The analysis of the data reveals to us that soy is significantly related to increased mortality in children from leukaemia. The complications generated by soy anti-nutrients, are becoming more evident in an increasing number of diseases and associated morbidities.

Clearly from reviewing the research at the cellular and micro scale, as well as from a global perspective, at the macro scale, of the incidence from the analysis coming from many countries. It is now, not very difficult to observe, that the results indicate that feeding soy to immature immune systems as found in young children is not very wise and should not continue to be recommended for children or infants with immature immune systems. Feeding children must be considered from two basic perspectives. Indirectly with the nutrient supply via the maternal diet, and directly through actual ingestion of unfermented

soy influenced processed foodstuff, including soy infant formulas, unlabelled baby foods and the like. The technology exists to remove the phytates and trypsin inhibitors etc, but the manufacturers are more interested in profits rather than public health.

Even if the results were not as significant as they are, it is clear that the principle of least harm has not been applied, but has been set aside in the interests of economic advantage to both government and corporate agribusiness, and perhaps with a complicit medical establishment. Until responsible governments see both converging pictures, the advantage of cheap food will vaporise in the face of consequences of burgeoning medical costs and complications, in an increasing array of diseases from minor allergies, to a number of serious afflictions, including cancers and leukaemias, evident in the medical cost to population health and economic welfare. The increasing and crippling costs to each nation cannot be sustained indefinitely.

There is a very clear indication, that there is a large scope for further experimental research on the carcinogenic properties of soy and processed soy products, which must be carried out independently of the vested interests of the soy industry.

### **Soy's inadequacy as a food staple**

It is a wonder that soy has been so heavily endorsed as a food staple, as it needs to be boosted and/or supplemented with so many extra nutrients.

Extra iodine is needed to counteract the soy's negative effect on the thyroid (Divi et al. 1997). The promotion of calcium-fortified soy milk presented and promoted as a means of preventing osteoporosis in women may indeed be functional as was demonstrated in the



5-year Edinburgh study, but what was not highlighted were the side-effects of increased breast cancer in women and prostate cancer in men.

### **Vitamin D deficiency**

From some of the work of Imataka et al (2004) we consider another clue as to the emerging child leukaemia picture in that an infant nursed with soybean milk developed rickets due a vitamin D deficiency among other items due to soybean milk. We would be concerned as other research has revealed that a vitamin D deficiency precedes and accompanies the development of many cancers (Helou MA,et al. 2008). The statistics points out that each child diagnosed with some form of malignancy should immediately go on a supplementation with vitamin D<sub>3</sub> as part of the healing process. A sufficiency of vitamin D is very important for the protection of the foetal immune system (Reichrath and Querings.2005). Again fortification with vitamin D of processed soy influenced foods by the industry presenting extra vitamin D has often been questionable, firstly since soy has very little vitamin D, that it definitely needs fortification with vitamin D. Secondly, the cheaper, less active form of vitamin D has been used in the past, that is vitamin D<sub>2</sub>, instead of the more active D<sub>3</sub>. Industry of most types is all about short cuts and maximising profits and for a while many foods and supplements would only list nonspecific vitamin D until the community was better informed and demanded better quality and standards that vitamin D<sub>3</sub> became as common place in the community mindset as has concerns about 'omega3s'.

### **Vitamin B<sub>12</sub> inadequacy**

Although soy has vitamin B<sub>12</sub>, it is an analogue that the body cannot utilise efficiently; by occupying the B<sub>12</sub> receptor site, the B<sub>12</sub> from soy is more likely to hinder proper B<sub>12</sub>

metabolism, thereby paving the way for pernicious anaemia which sometimes has been misdiagnosed as leukaemia among other multiple morbidities.

### **Childhood leukaemia**

Soy, with its hormone, endocrine disrupting and phytate nutrient blocking also needs to be acknowledged as being directly or at the minimum of being indirectly involved as being responsible to contributing to childhood leukaemia and some other cancers, besides other morbidity's on foetal development.

Each of these effects influenced by the antinutrients in soy, by themselves, may have been considered, in the past, as being very small, and by themselves perhaps almost unnoticeable, and easily dismissed because individually they are/were perceived as being of little consequence. However, even if they are small effects, when combined and added together the overall effect can become a foundation context for generating the leukomogenic process. Furthermore the resulting accumulation of these effects on a micro scale when combined with what is observed with the results on a macro scale form a picture that is reflected in the continuing incidence of child leukemia that is becoming more plausibly influenced due to the consumption of processed foodstuffs influenced by the anti-nutrient nature and content of soy. The fact that other possible instigators of leukaemia have been identified only reinforces how vulnerable the foetus is, as well as a young infant at a time of rapid growth when normal development is subject to the biologically disrupting chemical influences as found in soy bean products.

As a supposedly superior foodstuff, soy has frequently to be strangely fortified in many ways, in order to be promoted as a healthy food item. Many mistakes have been made

in the fortification process, as in a recent soybean milk recall in NSW Dec 2009( Crawford BA et al. 2010), many people (40+) were hospitalised from drinking a soymilk that had been iodine fortified 1000 times more than necessary to overcompensate the iodine-blocking properties inherent in soymilk. Not surprisingly, some of the worst cases involved pregnant women suffering miscarriages after drinking the soymilk. That unfortunate incident is now the subject of a legal class action, in NSW, in which, over 500 Australians have become part of a lawsuit in the class action.

### **Antinutrients in soy, Chelators of vital essential nutrients and elements**

The phytates in soy are very strong chelators of many vital essential nutrients and elements. The soy chelating process is very likely involved in depriving the developing infant of those essential nutrients necessary under normal circumstances for the healthy child to thrive. One would expect that if you subject a robust healthy child, as a young toddler, to a nutrient-depleted food supply, it will eventually become sick. How much more so for the unborn child? In normal circumstances it would be expected that such actions may well be construed as child abuse. Unfortunately the foetus is not in any position to articulate its disquiet, let alone with an immature immune system to adequately defend itself. The antinutrients in soy may not necessarily do the nutritional damage directly, and may not be immediately obvious, but may instead create an environment, by depleting essential nutrients and minerals, thereby compromising, the development of the normal immune system of the foetus. This can only mean that the immune system does not develop properly and, thus weakened, enables the cancerous leukaemia to develop, either in the uterus, or not long after. As soy consumption increases around the globe, the incidence of

child leukaemia is likely to continue to increase and spread, despite better methods of treatment and improved survival rates. Improved survival is welcome. However, because of the side effects collateral damage experienced while going through the leukaemia cancer rehabilitation process, such children will often experience other morbidities, afflictions and other cancers that are frequently associated with the treatment protocol(s).

While promoted as a health food and cheap source of protein in many countries, it has been found that as far back as the 1930s (McCarrison R, 1933), (Wulgus HR et al. 1935) (Sharpless GR et al.1939) soy has also many detrimental chemicals and factors that compromise mammalian immunity and health particularly with issues related to iodine and goitrogenicity. Some of these compounds and enzymes have repeatedly been implicated in a number of illnesses besides cancer.

It is a wonder that soy has been so heavily endorsed as a food staple, because nearly every industrially soy produced food item requires to be boosted and/or supplemented with many extra nutrients. To claim that many processed soy food products are natural sources of nutrients is an outright marketing deception. The anti-nutrient properties of soy seem to be inherent in the soybean profile regardless of its source, organic or not.

We are increasingly becoming aware of the heavy pollution in the modern world both from deleterious chemicals in the environment, from industry and impatient farming practices, as well as questionable food products, and even food containers that can contaminate stored foods as well as chemicals that are applied physically, including creams, lotions and sprays-many of which have both known and unknown toxins and excessive amounts of xenoestrogens and even metallo-xenoestrogens. In the light of such pollutants it

would/should be considered highly advisable that throughout the gestation phase in the increasing polluted modern environment, the mother should not only avoid smoking and drinking both before and during gestation but attempt the more difficult task in trying to eliminate or cut back on and avoid as much soy consumption as possible. Because soy is almost everywhere in the food supply, it is has now become almost impossible to avoid, extra effort should be made to detoxify the maternal environment, as well as excluding soy products where possible, from the foods ingested. If a 'magic bullet' eventually does appear, it will be well received. However in the meantime, the only plausible solution to child leukaemia may very well be a preventative measure in avoiding soy consumption by both mother and child and perhaps for a time before anticipated conception the father.

## **Part III. A possible cause of Alzheimer's dementia — industrial soy products**

### **ABSTRACT**

Alzheimer data indicate that at present, about one new case of this form of dementia is identified in the USA every 68 seconds and that by 2050, the incidence, will be about every 33 seconds, with projections from the Alzheimer Association (USA) indicating 25% of the United States will experience Alzheimer's disease by 2031. Despite the numerous advances in medical science and neurological research, the causes of Alzheimer's are still unknown and the incidence is not decreasing or levelling out.

Most research on the causes of Alzheimer's indicates the possible roles of viruses, obesity, physical inactivity, diabetes, psychological depression, high blood pressure, frequent inflammation, environmental or domestic chemicals and toxins, or inescapable genetic factors. Alzheimer's, being degeneration of parts of the neural pathways in the brain, may indeed involve neuro-toxic compounds that can bypass the blood-brain barrier. So it is necessary to examine what is prolific in the environment and, in particular, the food supply. One of the many suggestions in the literature is the ingestion of food items derived from and related to unfermented soybean products; the anti-thyroid, anti-nutrient, and endocrine disruption properties of soy can have a deleterious effect in many individuals. We obtained mortality data on Alzheimer's from WHO publications 2008 and 2011 and used

earlier FAO information on soy consumption from 2005, as its effects may need some time to manifest in the form of Alzheimer's. Altogether we matched mortality and soy consumption data for 90 countries. Analysis of data per country shows a relationship between average soy consumption per capita per country, and mortality from AD in that country (linear correlation  $r=0.39$ ,  $p<0.001$ ). In countries with soy consumption in the lowest tertile (below 4 kg/person/yr) the mortality from Alzheimer's is on average 0.57 persons per 100,000 per year, while in the middle tertile of soy consumption (4 to 20 kg/person/yr) the Alzheimer's mortality is 4.57/100,000, and in the upper tertile (21-85 kg/person/year) the average mortality is 7.62/100,000. On simple count, among 17 countries of the lowest tertile that have data on Alzheimer's mortality, 15 countries (i.e. 88%) have Alzheimer's mortality below 1 per 100,000 and two countries over 1/100,000, while in the remaining two tertiles of more abundant soy consumption only 39 countries (i.e. 53%) have Alzheimer's mortality less than 1/100,000 and 34 have higher Alzheimer's mortality. This difference is significant at  $p<0.01$  (Chi-squared=7.74, 1 df). For a country, the odds ratio of having high Alzheimer's mortality while consuming more soy is  $OR=6.54$ . These results are clear and significant, even although we used statistics compiled from varied government sources that may have differing accuracy. Among the many theories and different factors that may be involved in dementias, soy consumption seems to be a significant contributor to AD, and from the data cannot be excluded as a possible contributing cause.

## Introduction

Throughout history and in all cultures without exception, the elderly were the repositories of knowledge and wisdom that was passed on from parent to child, or grandchild. This has been corroborated by anthropological studies in each culture. Literature, art and sculptures frequently depict the archetype of the wise elderly who were often independent and sometimes chose to live independently as hermits, whom members of the associated society would seek out and consult for answers to difficult questions and problems. The Greek philosophers and thinkers who have given us most of our culture today were not young people, they were elderly, having acquired the wisdom from ancient times and were esteemed amongst the wise in retaining and transmitting the secrets of their ancestors as well as being knowledgeable in the ways of the world. Fortunately for us, some of their writings have been preserved and have been transmitted to us today, through the classics or other recorded forms. Western science and culture draws heavily on Judeo-Greek- Roman and Christian philosophies and social writings, usually by persons who lived long enough, in complete charge of their mental faculties, to enjoy the benefits of a 'ripe old age'. Such fully competent matriarchs and patriarchs are now rare in modern and Western societies, but are quite common in non-industrialised societies.

In Western societies the elderly are packed off to nursing homes, with many losing their minds and heavily drugged to stop their mindless and aimless wandering. Those who live alone or who are without family become a burden on the state. Some have become completely dependent on others for daily survival. The frightening experience to lose control



of one's faculties, often not recognising members of their own family or even where they reside, can become a serious burden for both patient and carer. Lack of understanding can disrupt and destroy families. The archetype of the wise and benevolent earth mother or the gentle and knowledgeable wise old grandfather, is now under attack. In the Western world, in unprecedented numbers, the elderly are losing their memories and their physical capabilities. Having lost their independence, they often sink into serious depression and lose the will to live. Statistics from many Western societies indicate that Alzheimers disease (AD) is the most frequent form of disability for the elderly. Families and societies who still revere and respect their elderly grandparents are becoming fewer with each passing day.

According to the peak body for dementia and AD in Australia, the data indicate that *"dementia is already the single greatest cause of disability in older Australians (>65)"*, as well as:

*if dementia were a country it would have the economic numbers that would be equivalent of GDP to a country ranked at 18<sup>th</sup> in the world with a budget much larger than the commercial activities of either the companies of Wal-Mart (US\$414billion) or Exxon Mobil (US\$311billion). ... "[Unless there is a] significant medical break-through, the Australian costs are projected by 2060 to outstrip any other health condition reaching spending levels in the order of \$83 billion, with total estimated 2010 worldwide costs for dementia having already reached US\$604billion.*

Unfortunately dementia in its various forms is not only increasing in the older age group, but there is also an alarming incidence of the disease in the 30 to 50-year-olds. There are already many documented cases where younger people in their 30s and 40s have become victims of this disease. This has created a new concern. Such patients often have

young children and heavy financial commitments that can destroy most families, potentially placing an extra burden on the state and social services.

Aside from medical care, *“the cost of replacing family carers with paid carers is estimated at \$5.5 billion per annum” (Alzheimer Australia)*

Dementia was never an expected consequence of aging. Why, in the 21st century, with improved understanding of healthy living and advanced medical knowledge, should it become a disease of epidemic proportions such that it is projected to become within two decades *“the third greatest cost- burden of health and residential aged-care.”(Alzheimer Australia)* The American peak body counterpart, declares that the 2012 direct costs to the US economy of caring for AD patients “will total \$200 billion including \$140 billion to Medicare and Medicaid” ([www.alz.org](http://www.alz.org)). Furthermore, the Alzheimer’s Association predicts that, unless an answer is found by 2050, the costs of Alzheimer’s to the US economy are estimated to increase to \$1.1 trillion (in today’s dollars) ([www.alz.org/facts](http://www.alz.org/facts)).

Further complications and costs from treating Alzheimer’s arise when these patients have other medical conditions such as, for example, cancer (extra cost of 53%) or diabetes (extra cost of 81%) ([www.alz.org/facts](http://www.alz.org/facts)).

## **Possible causes of AD**

Alzheimer's dementia is usually attributed to damage to brain cells, resulting in brain cells no longer being able to communicate with each other, thereby compromising the integrity of the neural network within the brain. The inability of certain brain cells to

interact efficiently with each other has dire consequences for many of the components which are responsible for normal function. The brain's different regions — those that deal with memory, thinking, emotional and psychological equilibrium and physical movement and body coordination — need to operate in unison-. As each successive region of the brain is affected, the associated behaviour becomes aberrant. The deterioration of the brain is revealed by autopsy of deceased individuals, in combination with MRI scans and other nuclear imaging equipment.

The cause or causes of Alzheimer's are still regarded as unknown. Indeed, the mortality numbers continue to increase so that between:

*"2000 and 2008 death increased 66%, while deaths from the number one cause of death, that is, heart disease, decreased" (Alzheimer's Association, [www.alz.org](http://www.alz.org))*

Aside from some 'trial and error' drugs that may offer slight relief, the general consensus is that there is no clear idea of the cause and thus no known rational defence strategy to prevent its occurrence or progression. The drugs currently used have limited capability and/or are largely unsuccessful.

A number of suggestions and theories have been proposed: age, genetics, excessive use of alcohol, smoking, lifestyle, the side effects of certain medications, psychological and manifest behavioural depression, hypo- or hyper- thyroid issues, chronic stress, chronic sleep deprivation, high blood pressure, continuous low-level inflammatory illness, obesity, diabetes and cardiovascular disease. Head trauma of some kind, whether internal from a stroke or external from a sports-related injury to the head, or participating in head and brain-targeting sports, such as from boxing or mixed martial arts often resulting in dementia

puglistiva. Despite all of these strongly regarded possibilities, our focus here will be on food intake and a food product that is consumed by large populations in regular calculable quantities.

In recent decades (a high cholesterol diet has been proposed as a possible cause of AD. To this end a statin drug study was performed in about 2004 in an effort to slow down the onset of AD. However, analysis of the results in 2008-9 revealed that the most popular statin cholesterol –reducing drugs were unsuccessful in reducing both mental and physical decline in Alzheimer's patients, or, in particular, will not prevent the disease. The placebo group developed Alzheimer's at the same rate as the statin takers, indicating no difference. Mental decline may not be as readily detected or assessed as easily as physical changes.

There are frequent reports involving some of the common and known side-effects of statins such as muscle-weakening and strange psychological behaviours, including mental and psychological disturbances and aberrant psycho-social activity (Gravelines D, 2012), paralleling many symptoms of various types of dementias.

The promotion of cholesterol reduction as a solution persists. A boost in that direction came from a study from France in 2004 presenting a finding that the use of statin cholesterol –lowering drugs slowed cognitive decline (Masse I et al. 2004). It was claimed that there are a large number of these studies. The problem with these types of studies and the extraordinary claims that come with them, (such as “70% reduction in AD incidence in people who use statins” (Masse 2004)) is, that, they are funded with an obvious agenda driven by corporations that already own the global rights, for the use of statin drugs with a ready profit motive, which might even be acceptable, if the drugs actually worked to the



percentages that they claim). In looking for new applications for the highly profitable statin drugs industry, is the obvious incentive, rather than genuine health concerns, to push the possibility, that statins may be useful, in combating AD, despite an earlier review by Scott and Laake (2001) indicating that there was no real evidence for statins to have any appreciable positive effect on AD. Zamrini et al. (2004), considered a nested case-controlled study and concluded that it would be useful to look at it from a different direction. They considered more AD trials after observing an inverse association with AD and statins. One of the largest studies over an 18 month trial (2008) involving Alzheimers and Lipitor, with a cohort of 640 persons indicated that there was no significant difference in cognitive decline between statin and placebo groups. Nevertheless, at the end of the decade of research (and more recently for the present discussion) with a later analysis by McGuinness et al. (2010) that despite subjecting three more studies to statin and dementia scrutiny, the McGuinness group of different authors also concluded, that there again, was not sufficient evidence that statins may be of assistance, or recommended for the prevention of dementia. Their research, essentially indicating that the 'jury has to stay out' on statins, and that it would be at the present time, an unjustifiable recommendation regarding the use of statins, for dementia (McGuinness et al. 2010). However, research seems to be ongoing, with those who hope to use a statin or something akin to cholesterol reduction processes, in total contrast to others who are very sceptical of the supposed benefits of statin drug cholesterol reduction therapy. To add fuel to the issue a reduced risk of AD has been associated with an increased level of HDL cholesterol, (Reitz C et al. 2010) which strengthens links with concepts, that would consider that basic biology indicates the possibility of neuronal destruction which could result from unnecessary excessive artificial cholesterol reduction.



A large percentage of the brain is composed of cholesterol and many different types of fats, some of which are saturated and others which are highly unsaturated (DHA and EPA). Indeed, despite recent studies proclaiming the possible benefits of reducing cholesterol for AD the use of cholesterol-reducing drugs, have many unacceptable neurological side-effects, including cognitive decline, memory loss, psychological disturbances and muscle wasting and other physical effects generated in normally healthy patients who merely have slightly raised cholesterol levels (Gravelines 2012). It is therefore irresponsible at this stage until more definitive and independent research has been completed, to allow for the push to prescribe more cholesterol-reducing drugs to patients who already have some form of motor/muscular imbalance/neurological disruption in their lives. In the light of such reports and even personal accounts and experiences ( which is where we often get the clues to start with research), it does not make sense to inflict any more unnecessary neurological damage with the continued reduction of cholesterol paradigm (MaGuinness B, et al. 2010) (Graveline D, 2006-2012).

Perhaps such research projects would be better off assessing whether statins should be removed from the prescription list of Alzheimer dementia patients. Dietschy and Turley (2001) clarify the numbers:

“the CNS accounts for only 2% of the whole body mass, yet it contains 25% of the total cholesterol in the whole individual ... “cholesterol is required everywhere in the brain as an antioxidant, an electrical insulator ... is a structural scaffold for the interconnecting ‘wiring architecture’ and ‘communications organization’ , and to continue the limited analogy a little further, cholesterol acts like the plastic coating which stops the “wires “ from shorting each other out. Unfortunately the analogy may have to stop just there, as in actual fact cholesterol is also actually part of the internal





composition of each neuron, besides being used throughout the whole interconnecting network of the brain's neural network. Indeed the neurotransmitters even utilize cholesterol in the synaptic delivery process"

Cholesterol thus plays a crucial role in the integrity of the myelin membrane (Pfrieger, 2003; Bjorkhem & Meaney, 2004). The whole process may start a whole lot sooner than previously thought in that, myelinogenesis is also very dependent on the health state of the thyroid. If it has sufficient iodine myelination proceeds as per normal. However, if the thyroid is denied sufficient iodine due to the action of phytate restriction, (a commonly recognised aspect of soy antinutrient behaviour), the myelinogenesis process slows down on one hand. However, if there is an excess of iodine the process starts sooner and in consequence burns out rapidly and stops too quickly. Neither extreme is good for healthy brain myelination.

The low cholesterol paradigm should be put to rest at the earliest opportunity. The Framingham Massachusetts decades long-study has indicated that cholesterol is not the issue for cardiovascular and heart disease (Castelli 1992). (It could be argued that many of the Alzheimer symptoms could also be coming from the side-effects of statin drug usage) The low cholesterol theory, widely promulgated and accepted from the 1950s, and widely pushed from the 1960s onwards by impatient commercial interests disregarded the scientific shortcomings seen and expressed by a few dissenting voices, ( who realized that the machinery of science needs to sometimes turn slowly needing extra time to uncover correct information and give us confidence in a better understanding of new knowledge), has now become a firmly entrenched global nutritional ideology (as is evident in the frequent push for more statin trials to reduce cholesterol). Other work (McKully 1999) has

clarified the issue in that a low cholesterol regime suitable for a minor percentage of the population identified as hypercholesteroleemics, is not necessarily a healthy option to include everybody in the population, let alone for children. A simpler approach is to examine conditions to which the whole population is subject; that is food, and food consumption (which may also involve cholesterol reduction). We have previously described the heavy reliance on processed soy foods and products (Roccisano & Henneberg 2011). Soy has been used extensively in nearly all low-fat and low-cholesterol processed food products (Roccisano and Henneberg 2011). The acceptance of cholesterol reduction in any way possible as a 'good health' strategy led to the promotion of polyunsaturated fats in soy oil as a means of lowering cholesterol. Not only was the use of the poly-unsaturated oil perceived to be beneficial, but many of the spin-off byproducts (eg. margarines via hydrogenation) of the oil including any byproducts of the soybean itself were seen as a better option as a source of protein in food preparations than foods from other sources, especially those of animal origin. We would argue that the use of soy as a means of lowering cholesterol for AD is not necessarily a good practice. The realisation as far back as the 1950s that soybean oil could reduce cholesterol lent great impetus to the low cholesterol, polyunsaturated money-spinning margarine craze that took off in the 1960s. The commercial interests were very opportunistic in attempting to take advantage of the very financially 'useful' and simplistic idea that saturated fat and cholesterol were the basis of the problem of heart disease, thereby opening an apparently simple solution to CVD with very profitable fringe benefits that did not remain for very long on the fringe. Unfortunately things are not always simple or as straight forward as was presented in the admonition to replacing saturated fats with polyunsaturated fats, and advocating foods low in cholesterol. It took long term studies like

the Frammingham heart study to demonstrate the falsity and inadequacy of such a paradigm. Eventually, it was realized that the adoption of the polyunsaturated fat and blanket recommendation of reduction of cholesterol paradigm has been a global health policy disaster still driven by vested interests and costing ill-informed governments unspeakable billions of wasted financial economic and medical resources, from mounting continual social and medical costs to misdirected medical research funds. Soy may indeed be able to reduce cholesterol to some extent. However, studies such as the long term Framingham cardiovascular study have indicated that cholesterol is not the culprit for cardiovascular disease (Castelli 1992). Therefore, soy's main promoted advantage for human health and controlling CVD, via cholesterol reduction, is now largely irrelevant; non oxidised cholesterol has been unjustly maligned and is not the culprit. The cholesterol reducing properties of soy are no advantage whatsoever, when you consider the disadvantages from the many anti-nutrients readily found in soy. The capacity of soy to reduce cholesterol in a way similar to statins will also be of no real benefit to Alzheimer dementia patients, and may turn out to be actually be deleterious. However, processed soy food products have been widely proliferated and are almost unavoidable in the processed food supply, so much so, that the whole population inadvertently is forced into consumption of soy and its industrialised food products throughout the food supply of many populations makes it a serious issue or factor for consideration regarding the maintenance of good health in each population. Soy products are found everywhere you turn. From your morning breakfast packet cereals — usually some extruded soy and grain paste with sugar content off the scale, with colour and excitotoxic flavour enhancers to make the products palatable — to the morning tea cake or bun with unfermented soy flour additive content, ( soy flour

has been demonstrated to initiate pancreatic cancer in rats (McGuinness et al. 1980)), and to a cafe or restaurant dinner which often uses a cheap brand of unlabelled vegetable oil, most of which, comprises a mixed percentage of vegetable oils in combination with up to exclusively 100% soybean oil. If there is a label, it will describe that it is a vegetable oil in prominent form somewhere on the container, while the oil content profile will be difficult to find if it actually exists.

With the acceptance and consequent promotion of the polyunsaturated fatty acid paradigm, sixty or more years ago, soy was marketed for its purported ability to reduce cholesterol. At the time, one of the major promoted household food products was polyunsaturated margarine, which was predominately manufactured from hydrogenated soybean oil and claimed to have a beneficial impact on CVD. Eventually, it was realised that the very stable trans fats, (much preferred by the baking industry as it gave their products a longer shelf-life) which were artificially formed saturated fats made from subjecting the polyunsaturated soybean oil to the hydrogenation process, were more likely implicated in cardiovascular degredation(Willet WC, 2006). For many years industry representatives claimed that trans fats had no real differences in metabolic behaviour for mammals than natural saturated fats. The trans fats escaped scrutiny for decades as possible contributors to heart disease because of their polyunsaturated origins according the low saturated fat, low cholesterol paradigm. The focus of research during the heart conscious early decades of the anti-saturated fat and lowering cholesterol paradigm, involved the use of hydrogenated and artificially hardened vegetable fats, since it was believed that they presented no appreciable difference to any other type of saturated fat. Later research revealed sufficient information

that transfats were indeed more destructive to the cardiovascular system than natural fats, that eventually, with that realization, governments finally got convinced and got involved, such that, eventually it was acknowledged as serious enough to legislate( as late as in 2006 for the USA, with many EU countries having close to zero transfat tolerance in place well before the US) the enforced labelling of foods that contain the percentage of transfats as distinct from other saturated fats

In addition to polyunsaturated soybean oil, another supposedly healthy soybean product investigated in the mid-1960s for preventing CVD was tofu, a non-fermented soy product. A long-term study in Hawaii observed and assessed an 8,000 cohort of Japanese-Americans for tofu consumption over a 30 year period. Despite any shortcomings, the long-term nature of the study was both useful and quite revealing. Most studies that are quoted in support of soy products are often of short duration and do not allow for a fuller possible outworking of the damage done by the antinutrient factors in soy (White L et al. 2000). Besides the pursuing of the supposed cardiovascular benefits, subjects were also assessed for brain function using MRI imaging techniques, psychological testing procedures, and autopsy of those who passed away during that time.

To the dismay of the soy industry, the results revealed significant associations with cognitive decline, accelerated brain aging and actual brain size atrophy, so much so, that they would have readily met the criteria for diagnosis of AD (White L et al 2000). The data and analysis strongly indicated that the more tofu a person consumed, the more likely there would be brain atrophy and associated mental disruption through cognitive decline, uncannily resembling the symptoms of AD.

Many have tried to explain away these results, but there is some substantial research from diverse fields that lends strong explanatory backup to the problems generated by soy consumption.

## **Hypothesis**

Soy is a strong goitrogen (Kimura et al 1976) containing many antinutrients and shows phytoestrogens, genistein, and trypsin inhibitors of phytates in larger quantities than other vegetables, which cooking modes do not neutralise.

Consumption of soy food products may contribute to the increasing incidence of Alzheimer's and other dementias.

## **Material and methods**

Databases of mortality from Alzheimer's per country (WHO), soy consumption (FAO) and GDP per capita by country (UN) were used. Soy consumption information was converted to kg/yr/person using national population sizes. Only WHO data, for countries that provided "reliable" information, on mortality from AD were used. This provided a sample size of 90 countries. Information on mortality from AD was analysed both separately and together for the age groups 15–59 years and 60+ years.

Because of skewed distributions, for the purposes of calculations, product correlations data were logarithmed.

Linear correlation was calculated between soy consumption and total mortality from AD and compared averages of soy consumption for countries with low and high Alzheimer's mortality. For the age group 15–59 years, mortality was considered low if it was reported to be less than one person per 100,000, whereas for the 60+ group low mortality was that below 3.5 persons per 100,000. T-tests were used to assess the significance of correlation and differences between averages.

## Results

The log–log correlation of soy consumption and Alzheimer's mortality was significant ( $r=0.24$ ,  $p<0.05$ )-(Fig.1). For both age groups, average soy consumption per capita in countries with low Alzheimer's mortality was lower than in countries with high mortality (Fig. 2). The difference was especially large in the 15–59 age group, where the countries with the highest Alzheimer's mortality were China and the USA.



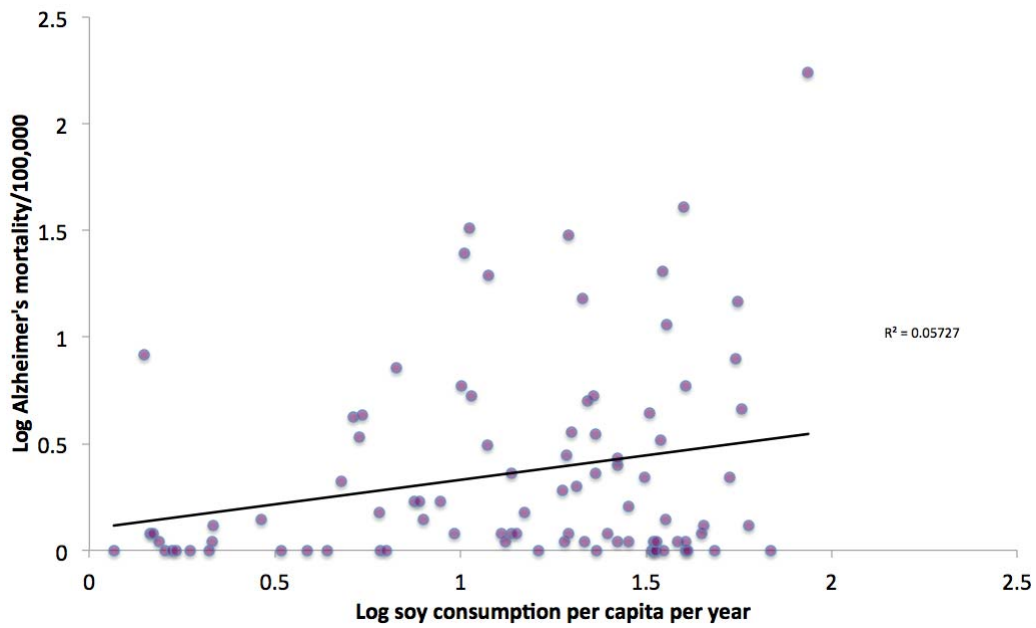


Figure 1a. Log–log correlation of soy consumption and Alzheimer's mortality

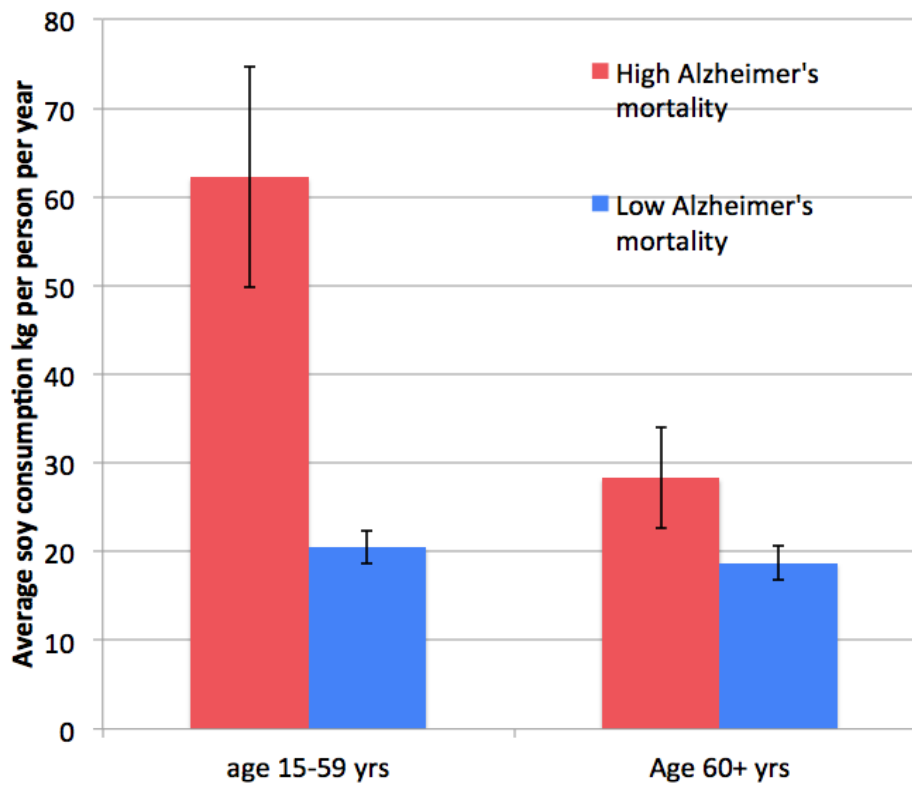


Figure 1b.

Average soy consumption in countries with high and low mortality due to Alzheimer's disease in younger and older age groups.



## Discussion

Soy has been promoted in many countries as a cheap, healthy food for both humans and animals. However, what has not been made clear to both the public and the medical community, is that soy also has many detrimental chemicals including topoisomerase type II poisons, as well as the bioflavonoids genistein and diadzin (Yamashita Y et al. 1990; Markovits J et al 1989). It has been shown that even low maternal exposure to doses of dietary soy topoisomerase type II poisons and the like (Hengstler et al. 2002) may cumulatively contribute to the development of many diseases both at birth, with child leukemia (Hengstler et al. 2002), and over the course of a lifetime, resulting from accelerated brain atrophy and shrinkage (White et al. 2000).

The traditional form of soy used in Asian cuisines differs markedly from that used in the current Western diet. Traditionally soy was fermented before its incorporation into the Asian cuisine. Soy was never considered the mainstay of meals. Prior to western influence with processed soy products, the inbetween foods were often dried foods made from real or natural fruits meats and vegetables. Some of these exotic foods are still available through different culturally oriented supermarket outlets. Unfortunately, the ready and easy convenience of the Western style of the pervasive snack food industry found in Western diets will eventually erode such cultural differences. Furthermore, in the many packaged Western food products soy is industrially processed without fermentation. By the time, most processed soy products reach the market place they are anything, but, natural, even though the often promoted health qualities of soybean are questionable in the first place.

The use of Western-style industrially processed food products, such as most processed or packaged foods and margarines (which were predominately made from hydrogenated soybean oil) as well as the majority of bakery products (utilising unfermented soy flour additive), have now been heavily marketed in Asian and other non-Western countries. The subsequent and ready adoption and consumption of Western-style processed foods has increased rapidly in these countries as Western forms of soy processing and usage are exported back to Eastern cultures.

As we have seen, the antinutrients in soy can affect DNA replication, leading to aberrant biochemical changes in the unborn child with an undeveloped immune system (Hengstler et al. 2002). Because of the process of rapid cell growth in the unborn, the genistein in soy is toxic to topoisomerase type II nuclear enzymes, interfering in their job of maintaining the normal process of DNA replication, thereby leading to a cascade of chromosomal aberrations resulting in a higher likelihood of unusual and inappropriate biochemical processes in the unborn (seen in the high prevalence of child leukemia) (Roccisano & Henneberg 2011, Hengstler et al. 2002), as well as other leukaemias and immune dysfunctions (Conner B, et al. 1997) in the aging and deteriorating immune systems of the elderly, as can be seen by the SEERS data with the increasing prevalence of leukaemia from the age of about 45 years and increasing up until the 70s and 80's. The antinutrients in soy can also, through the diet, increase these risks to DNA replication (Morris SM, et al. 1998), thereby inducing biochemical changes that generate accelerated cerebral atrophy and shrinkage in the aging brain with mid-life tofu consumption, as seen in the long-term Hawaii Honolulu study (White et al. 1996). In a later animal model experiment Lund TD, et

al.(2001) found that the phytoestrogens in soy can disturb both the proteins involved in neural stability and inflammatory response, as well as parts of the cerebral cortex that assist with memory and the learning process. Furthermore damage to the brain can also be initiated by disrupting neurotrophic factors that are normally generated in the in the brain itself. Not only are such neurotophic elements affected, there is also an effect on mRNA expression in crucial areas of the brain especially the hippocampus. (File SE, 2003). And following again in another later independent study also using an animal model, Choi and Lee (2004) confirmed that genistein from soy interrupts normal DNA replication in the cerebral cortex so that brain cells are not replaced, forcing the cell die-off and consequent brain atrophy as observed in the Hawaii study (White L, et al. 1996).

The versatility of the soybean and soybean products has seen soy consumption take many forms. A whole meal table could easily be filled with the different processed products generated from the soybean, including soy imitation cheeses, soy imitation milk, soy textured vegetable protein, soy burgers and other soy imitation meat products, soy imitation fish items and crabsticks, tofu, and unfermented soy flour as an additive to wheat and other grain flours in the baking industry. Indeed ,when examining the content different snack items Soy is made use of extensively in nearly all baking goods with little or no exception. Indeed ,when examining the content of some different snack items except for the shape, colour and taste, the ingredients in some instances are virtually the same, and giving people the impression they are eating different things. Again, in most western countries the biscuit and bakery industry uses soy flour as an additive and predominately

soybean oil under the guise of vegetable oil, with the main competitor being canola oil. These same countries have also learnt to produce a multitude of snack foods and soy oil in their own style even for export.

Not only are the unfermented solids of the soybean (e.g. soy flour, soy protein, hydrolysed soy (veg) protein), used extensively in baked goods, packet soup preparations and other packeted or boxed foods including most processed breakfast cereals, but the oil produced from the soybean (which enabled the US to become the dominant exporter of soybean fats and oils for many decades after the Second World War). The US still plays a highly influential role in global soybean oil production despite strong competition from Latin America and other countries that have been heavily influenced and enticed by the profits generated by the growing worldwide demand for cheap fats and oils.

There is worldwide uncritical consumption of soybean oil. Aggressive marketing and exporting of the abundant surplus, of soybean oil throughout the world, has led to soybean oils and fats becoming dominant by its cheapness, to influence the industrial processing of many different types of foods throughout the food supply, in great diversity. The intense and prolonged worldwide marketing of polyunsaturated fatty acids (PUFAs from vegetable oils predominantly from soybean), over and above those of the much maligned saturated fatty acids (SFAs), has further entrenched the low fat, low cholesterol paradigm initiated from the mid 1950s. Commercial interests disregarding any dissenting voices, spearheaded the push which has led to soy's uncritical embrace in nearly all communities, including medical, educational and the general populations in nearly every country. The advertising literature of the pharmaceutical and oil-producing companies extols the virtues of

polyunsaturated oils that promote a saturated fat-free and low cholesterol regime, with facile but convenient supposedly scientific support. The proliferation of polyunsaturated vegetable oils and spread throughout the food supply is essentially a twentieth century phenomenon, as the technologies to process, refine, treat and store most seed oils that would readily go rancid under normal conditions, have only been developed over the last 100 years or so. Unfortunately the benefits of many vegetable oils are quite limited and, from the literature, negated when the oils are subject to normal cooking processes (Csallany et al. 2005) or have not been kept in proper conditions, such as dark containers or under refrigeration, and away from light or heat. The oils are readily oxidised when internalised by ingestion and metabolized by normal processes which create reactive molecular fragments commonly called free radicals. The cascading free radical process is heavily associated with polyunsaturated fatty acids when exposed to light, heat or the body's own metabolic processes. Lipid peroxidation in tissues is an automatic reaction that occurs because of the unstable double-bond chemical structure of polyunsaturated fatty acids. In the brain, arachidonic acid and the DHA and EPA omega 3 highly unsaturated oils (HUFA's) are particularly vulnerable to ready degradation by the free radical process, which creates neuronal apoptosis (Kruman et al. 1997).

Such bio-degradation processes occurring in the brain make it no easy task for the brain to keep itself intact and maintain its normal functional integrity (Lovell & Markesbury, 2001). Furthermore, aside from its free radical bio-degenerative properties, when soybean oil is heated to cooking temperatures, along with a rampant increase in free radicals, there is the proliferation of other toxic compounds being formed.

The omega-6 and omega-3 fatty acids can readily undergo oxidation and fragmentation, creating a swirling deluge of a free radical “soup” reforming and creating very toxic aldehydes. Notorious and best-known among these toxins is 4-Hydroxy-2-Nonenal (HNE), which is a very toxic aldehyde (Csallany et al. 2005). However, another hydroxy alkenal, 4-Hydroxy-2-Hexanal (HHE), is just as toxic, and is also highly reactive with biomolecules, whether they are phospholipids, proteins or DNA (Catala 2009).

The continued ingestion of polyunsaturated soybean oil, (or for the same principle most poly-unsaturated vegetable oils) especially when they are utilised for cooking in the home, restaurant, on the road cafes, hotels, other fast food industry outlets or even the school or hospital cafeteria, as well as food prepared and supplied to hospital patients, does not auger well for the health of the general population in the long term. Indeed, the above information would also apply to consumption of most polyunsaturated vegetable oils, wherein the higher the unsaturation the more volatile and harmful they can become, simply because of the stereochemistry involved with fragile unsaturated double bonds. From the growing literature it is becoming increasingly apparent that such aldehydes (Williams et al. 2006) formed from heated soybean oil or the free radicals generated by simple cooking processes, become major contributors to a whole host of degenerative physiological body processes (Catala 2009) and which not unexpectedly engender inflammatory responses which may open the doors to other illnesses, besides toxifying and contributing to the acceleration of brain deterioration in particular as well as possibly causing problems in other parts of the body.



Esterbauer (1991) summarised the toxic properties of the 4-hydroxyalkenals that are formed when vegetable oils in general and soybean oil in particular (Seppanen & Csallany 2002) are heated to cooking or frying temperatures. Since that time such compounds have received and deserve the attention they are getting because of their direct linkage as catalysts for neuro-degenerative diseases and frequent neuro-inflammatory reactions. The important public health work of Csallany and colleagues(2005), has identified the toxicity of polyunsaturated soybean and vegetable oils, especially under frying and baking conditions, as in the home or the fast food industry such as hotels cafés restaurants, hospital cafeterias and the heavily used fast food outlets (McGrath LT et al. 2001). Even the use of imitation cheeses in the pizza industry baking process produces the toxic compound 4-hydroxy-2-trans nonenal (HNE), as often the fat source in soy cheese is partially hydrogenated vegetable (not unexpectedly from soybean) oil. Such oils that have high levels of linoleic acid, when subject to heat, transform readily into the toxic compound HNE as part of the cooking process. As HNE is a relatively stable molecule, with continued cooking, quantities continue to accumulate and the continually cooked oil becomes even more toxic with repeated cooking. HNE is directly incorporated into foods that are cooked with polyunsaturated soybean oils. McGrath et al.(2001). conclude that if, there are insufficient antioxidant mediators (e.g. ascorbate) *“high levels of 4-HNE have a positive relationship with impaired cognitive function”*.

The use of Western-style industrially processed food products such as the majority of vegetable oils and margarines (which were predominately made from hydrogenated soybean oil) as well as the majority of bakery products (utilising soy flour additive), has been

heavily marketed in Asian and other non-Western countries. The adoption and consumption of Western-style processed foods has increased rapidly as Western forms of soy usage are exported back to Eastern cultures.

In parallel with the anti nutrient phytate and protease inhibitor properties that soy manifests in both obesity and child leukaemia. The claim that soy is a good source of the important vitamin B<sub>12</sub>, may also need some clarification. The B<sub>12</sub> vitamin in soy is not bioavailable to the human body. The soy B<sub>12</sub> vitamin analogue, may therefore create more problems than is readily apparent. The phytic acid binding properties in soy can ensnare not only calcium, iodine and zinc but also cobalt which in particular, is part of the molecular matrix essential to thyroxine synthesis, necessary for the proper functioning of the thyroid (Liener IE, 1994). Furthermore, consumption of soy will then increase the body's needs for the correct form of vitamin B<sub>12</sub>. The implications of restricting the body's access to the proper form of B<sub>12</sub>, can have far reaching consequences. Firstly B<sub>12</sub> is a necessary precursor to the blood formation process with obvious implications as to aberrant cell processing that can lead to the development of leukaemia as indicated in the previous section. Secondly, the correct form of vitamin B<sub>12</sub>, along with folate (Smach MA, et al. 2011) is required to keep homocysteine (Morris MS. 2003) at safe levels (Smith AD, et al. 2010) thereby avoiding unnecessary cardio-vascular damage, both in the body and cardiovascular system, as well as the arterial vascular system network of the brain. Thirdly, as a consequence of the previous discussion, if homocysteine is out of control, earlier work by (McCaddon et al. 2001) has shown that with high levels of homocysteine there is age related mental decline. This was verified by a European study (Ravaglia G et al 2003) which indicated that

high homocysteine levels can lead to elderly cognitive impairment. Later on Vogiatzoglou et al (2008) have also made the connection between low vitamin B<sub>12</sub>, and high homocysteine levels as well as resulting in reduced brain volume(Vogiatzoglou et al. 2008) as was also reflected in the observed brain atrophy described in the earlier long term Hawaii study (White et al. 2000). Furthermore the indication and association of high homocysteine levels increases the co-occurrence of other afflictions such as stroke (Morris MS, 2003). Alzheimer disease has often shown up on autopsy in patients who have also revealed low levels of B-group vitamins and folate. This may indicate that any possible preventative measure may require the inclusion of adequate levels of the B-vitamins as well as folate(Smith AD et al.2010), (Smach MA,et al. 2011) The double benefit here is that such a strategy will also have benefits for prevention of CVD in general and the vascular network of the brain in particular.Other work by Walker et al. 2010 has indicated that “long term B12, folate and niacin deficiencies may contribute to the development of age related dementia and that by instigating active supplementation even after two years may assist in improving cognitive and memory function”(Walker JG, et al 2010). Furthermore, lack of B12 allows inflammation in the vascular system indicated by high homocysteine levels to get out of hand. Such an inflammatory response is also deleterious for the neuronal network of the brain.

Not only does the soybean, through its antinutrient profile engender goitrogenicity, and topoisomerase poisons such as genistein, found in its unfermented solids become a strong contributor to the ill health of many individuals, but the polyunsaturated fatty acids in soybean oil, by virtue of their chemical instability, contribute to ill health in both humans and animals, as unsaturated fatty acids by their very nature create much free radical (Sayre

LM, et al. 1997, 2002) and oxidative damage throughout the human body. This happens in two ways, by producing free radical damage throughout the body because of their inherent unstable polyunsaturated stereochemistry and by accumulating highly bio-reactive toxic compounds (e.g. HNE) and in the reactive molecular fragments that are produced during the normal metabolic process or introduced to the body by consuming foods prepared with soybean oil and/or made with soybean meal, since many solid processed soy foods are also cooked or fried in polyunsaturated soybean oil.

It can be considered that the Abeta amyloid peptide forms and strands of precursor protein in the body and in the brain in particular, are broken into reactive molecular fragments by the toxic effects of rancid molecules of polyunsaturated soybean oil and any other fatty acids that they contaminate by the free radical process which incidentally is also evident in Huntington's disease(Borlongan CV, et al 1996). At the same time, the formation of amyloid plaques could also be considered as the body's defence mechanism when under attack by damaging free radicals from ingested soybean oil. Because of its polyunsaturated chemistry, when used for food processing, and usually under cooking conditions, soybean oil provides the basis for free radical proliferation and the generation dangerous chemicals like HNE (Csallany & Seppanen 2005). The destructive bioreactive process produced by soybean oil's volatility can disturb the integrity of the strands of precursor proteins and lead to the formation of amyloid plaques. As the precursor protein strands get broken up into smaller molecules they can reform and convert into peptides, which form the basis for Abeta plaque proliferation. The process can continue by the interaction of the Abeta

peptides “sticking” together, generating solidified groups or more often described as clumps. Perhaps a partly plausible explanation of the process may follow the idea that when these molecules form in the body they are dispersed and eliminated. However, it is conceivable that the blood–brain barrier traps them in the brain. As they accumulate, initially they may be stored in certain areas that are not immediately essential for normal life functioning; this may show up in the early stages by interfering with certain parts of the “brain machinery”, as more frequent memory lapses and the beginnings of the loss of the higher order brain activities. Most of the time, the blood–brain barrier works to keep inappropriate blood-borne toxins and molecules away from the sensitive processes of the brain. It could be argued that in AD, when inappropriate molecules and or debris from inside the brain need to be eliminated, the blood–brain barrier may work against the brain’s best interests by preventing the peptides from being removed efficiently. So if the free radicals produced by the volatile soybean oil have managed to bypass the blood–brain barrier into the brain, that once there, they can create great damage inside the brain. As the brain has a number of very vulnerable, very highly polyunsaturated fatty acids (HUFA’s) which include eicosapentanoic, docohexanoic and arachidonic acids, the scene is set for much cellular damage; the highly polyunsaturated fatty acids are very vulnerable to the radicalising process, thereby creating even further molecular and cellular damage. The brain’s sensitive dependency on essential fatty acids, which are themselves highly unsaturated, may enable toxins such as HNE to create further protein damage. Perhaps in the process of attempting to protect itself from these accumulating peptides, the brain clumps them together in certain regions as temporary storage sites out of the way with minor brain activities. Unfortunately, storage place in such an environment would be extremely limited, so that

eventually the brain is overwhelmed with amyloid plaques beyond the normal plaque collection sites. Following this model, the plaque accumulation intrudes into parts of the brain that deal with memory and reasoning, before the lower order functions that are necessary to maintain life processes. Further discussion here may require the actual clinical autopsy examination of the different regions of the brain of deceased Alzheimer' patients at different stages of Alzheimer dementia development. It would be educative to examine Alzheimer patients when they are not at an advanced stage or yet affected by the hardened amyloid plaques that accumulate in the damaged brain. That type of research may require tests that help to identify persons who may be at risk.

By penetrating the blood–brain barrier and flooding the brain with excess free radicals, the polyunsaturated fatty acids from soybean oil can potentially damage the brain in three ways. First, contrary to 60 years of health advice, vascular damage in the arteries of the body and brain, most arterial plaque is composed not of saturated fats, but of unsaturated fats (Felton et al. 1994). Second, the damage to the protection cholesterol provides in the myelin sheath formation of the nerve fibres, as well as the cholesterol content of the neuronal structures and nerve cells, may be caused by free radical formation. Under normal conditions the cholesterol provides sufficient protection to the nerve cells, but under continual free radical attack it will eventually give way, destabilising the integrity of the myelin sheath. Third, by-products of the cooking process using soybean oil (Csallany 2005) create very toxic compounds such as HNE and acrylamides, which, as well as being toxic to the brain, are also carcinogenic in general. A properly functioning brain depends on

a proper balance of different fats and cholesterol that the body uses to maintain, protect and continually replace the neuronal network fibres.

The currently promoted low-fat, low-cholesterol paradigm of the last 70 years has produced neither the claimed health solutions in general nor the proper balance of appropriate fats and cholesterol that is necessary for the brain to maintain its integrity in the face of a continual free radical onslaught. Redefining which fats are appropriate will of necessity require more open clinical and experimental work that is not subject to dogma and preconceived agendas driven by commercial interests..

Abeta amyloid peptide forms and, in particular, strands of precursor protein in the body and brain, are broken into reactive molecular fragments by the toxic effects of rancid molecules of polyunsaturated soybean oil. Because of its polyunsaturated chemistry, when used for food processing (Csallany & Seppanen 2005), usually under cooking conditions, soybean oil provides the basis for free radical proliferation and the formation of highly toxic compounds such as HNE. The destructive bio reactive process caused by soybean oil's volatility can disturb the integrity of the strands of precursor proteins and lead to the formation amyloid plaques. As the precursor protein strands get broken up into smaller molecules they can reform and convert into peptides, which may form the basis for Abeta plaque proliferation. The process can continue by the interaction of the Abeta peptides "sticking" together, generating solidified groups. A partly plausible explanation of the process may follow the idea that when these molecules form in the body they may be dispersed and eliminated. However, it is possible that the blood-brain barrier mistakenly traps them in the brain. As they accumulate, initially they may be stored in certain areas

that are not immediately essential for normal life functioning, showing up in the early stages as more frequent memory lapses and the beginnings of the loss of the higher order brain activities by interfering with certain parts of the “brain machinery”. Most of the time, the blood–brain barrier, works to keep inappropriate blood-borne toxins and molecules away, from the sensitive processes of the brain. It could conceivably be argued that in AD, when inappropriate molecules and or debris from inside the brain need to be eliminated, the blood–brain barrier works against the best interests of the brain itself by preventing the peptides and their precursors from being removed efficiently from the brain. So if the free radicals generated by the volatile nature of soybean oil and associated products have somehow managed to bypass the blood–brain barrier into the brain, once there, they can create great damage inside the brain. The scene is set for much cellular damage. The brain’s very fragile and highly polyunsaturated fatty acids (e.g. EPA, DHA and arachidonic acid)s are highly vulnerable to the radicalising process generated by the readily oxidisable double bonds available in the fatty acid profile evident in soybean oil , which creates even further molecular and cellular damage depending to what it would be exposed. Not only is damage caused by the brain’s sensitive dependency on essential fatty acids, which are themselves highly unsaturated, but toxins such as HNE can create further protein damage and cellular havoc. Perhaps by a simple process of resolving internal brain injuries from the accumulating peptides, the brain clumps them together in certain regions as temporary storage sites, out of the way of the major life maintenance activities. Unfortunately, storage places in such an environment would be extremely limited and eventually the brain becomes overwhelmed with amyloid plaques accumulating beyond the normal plaque collection sites. Following this model, the plaque accumulation intrudes into parts of the



brain that deal with memory and reasoning before the low order autonomic functions that are necessary to maintain life processes. Confirmation of this model may require regular examination by clinical autopsy examination of the different regions of the brain of deceased Alzheimer patients at different stages of Alzheimer dementia development. It would be further instructive to examine Alzheimer patients with a view to developing tests for either early detection of AD as well as the different stages of the of the disease, especially where patient functioning is only partially affected by accumulated hardened amyloid plaques in the damaged brain.

Most of the possible answers to Alzheimers disease presently held are generally considered to be unsatisfactory. With the continued high prevalence of global obesity and the rapidly increasing incidence of cancer worldwide, we must also add the alarming statistics of the increasing incidence of Alzheimer dementia throughout the world. For the most part, it is not seriously considered that dementia of most types is an affliction that develops quickly. Therefore short term studies may not necessarily be a viable option in helping us to both understand such a disease and come to terms with attempting a cure. It will thus require research that may have to focus on long term planning and research of establishing prevention strategies as there do not appear to be any 'Alzheimer magic bullets' soon to be available. The research should perhaps focus on prevention in the first instance , and if a definite cure solution soon, appears, so much the better. The data results point us in the direction of any preventionstrategy that will reduce the amount of soy that is consumed by each community. Unfortunately it may require an evolutionary model for humanity to adapt while many thousands of people will have to be victims in the

polyunsaturated soybean oil consumption experiment of the 20th and 21<sup>st</sup> centuries. The depressing thought, is that the giant agribusiness corporations, may get away with both inflicting misery on thousands of people and collecting trillions of dollars at the same time. These problems are not restricted to political or geographical borders, affluence or poverty. Governments need to take deeper responsibility for the health and medical consequences of their decisions and actions. Short-sighted economic gains arrived at or obtained from supporting politically influential profiteering agribusiness companies and supporting industries that (of which soy has been a major beneficiary) compromise the health of their people and communities need to be regularly reviewed, seriously reconsidered, and difficult economic decisions made, as there are now, too many lives involved, for the problem to continue to be ignored.

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17 Jun 2009 ... Death rates among children have fallen dramatically, and most ...  
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Cancer and Vitamin D Posted by admin on November 27, 2008 under Cancer, Vitamin D

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Cancer survival for children aged 0–14 years was high, with an all-cancer ... Of the three most common childhood cancers, only leukaemia showed a significant ...  
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chronic diseases.fm In children, leukaemia (a cancer of white blood cells) is the most  
common cancer, accounting for approximately one-third of all childhood cancers.

The majority ...

[www.aihw.gov.au/WorkArea/DownloadAsset.aspx?id=6442459364](http://www.aihw.gov.au/WorkArea/DownloadAsset.aspx?id=6442459364)

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DOI: 10.1017/S0029665100000781 (About DOI)

National Cancer Institute Fact Sheet 1.21, Care for Children and Adolescents With Cancer  
(<http://www.cancer.gov/cancertopics/factsheet/NCI/children-adolescents>)

National Cancer Institute Fact Sheet 2.11, Cancer Clinical Trials

(<http://www.cancer.gov/cancertopics/factsheet/Information/clinical-trials>)