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Food Standards Australia New Zealand
WELLINGTON 6143 and CANBERRA BC ACT 2610

Application A1094 – Food derived from Herbicide-tolerant Cotton Line DAS-81910-7 - genetically engineered to provide tolerance to 2,4-D (2,4-dichlorophenoxyacetic acid) and glufosinate ammonium - Dow AgroSciences Australia Limited

Taking into account the following material, the Trustees and Members of PSGR urge Food Standards Australia New Zealand (FSANZ) to reject this application.

1 – Transgenic foods and safety

Pharmaceuticals are distinct and identifiable single agents. A new pharmaceutical is not approved without extensive animal and human trials to demonstrate relative safety and define risks and benefits. Even then it is recognized a high percentage of side effects are not discovered until after the drug is released for general use. It is an acknowledged risk that a new pharmaceutical chemical given o rally is a "prescription poison" and the recognized and unrecognized and the unintended effects of pharmaceuticals are assessed by the medical practitioner and the patient.

A new pharmaceutical requires an individualised prescription from a registered medical doctor and potentially informed consent from patients. Post-marketing surveillance effectively extends indefinitely.

Foods derived using genetic engineering technology contain unpredictable changes in plant chemistry; multiple and complex alterations. They also retain higher residue levels of pesticides; sometimes multiple pesticides where gene stacking is applied. There is no justifiable scientific basis to claim any food plant with novel engineered DNA as "equivalent" to a conventional food plant. By its very definition it is different. It is irresponsible to use such an unsubstantiated statement, especially by food regulators who have a clearly defined duty of care to uphold public safety and health under administrative law.

The inherent difference of transgenic foods from non-genetically engineered counterparts, and the attendant risk any difference creates to human health, dictates that foods containing novel DNA sequences should be regulated as if they were substantially equivalent to pharmaceuticals. This would include significant animal and human testing, and post-marketing surveillance on human health effects. It would also require informed consent from consumers.

We quote the Abstract from 'Compositional differences in soybeans on the market: glyphosate accumulates in Roundup Ready GM soybeans' which describes the nutrient and elemental composition, including residues of herbicides and pesticides, of 31 soybean batches from Iowa, US[v]:

"The soy samples were grouped into three different categories: (i) genetically modified, glyphosate-tolerant soy (GM-soy); (ii) unmodified soy cultivated using a conventional "chemical" cultivation regime; and (iii) unmodified soy cultivated using an organic cultivation regime.

"Organic soybeans showed the healthiest nutritional profile with more sugars, such as glucose, fructose, sucrose and maltose, significantly more total protein, zinc and less fibre than both conventional and GM-soy. Organic soybeans also contained less total saturated fat and total omega-6 fatty acids than both conventional and GM-soy.

"GM-soy contained high residues of glyphosate and AMPA (mean 3.3 and 5.7 mg/kg, respectively). Conventional and organic soybean batches contained none of these agrochemicals.

"Using 35 different nutritional and elemental variables to characterise each soy sample, we were able to discriminate GM, conventional and organic soybeans without exception, demonstrating "substantial *non-equivalence*" in compositional characteristics for 'ready-to-market' soybeans." (PSGR italics.) An extension of this would be that the principle applies to all transgenic food crops.

This application to introduce food derived from herbicide-tolerant cotton will introduce into the New Zealand food supply similar risks of high residues to every consumer, particularly our most vulnerable: pregnant women, their unborn children, infants, the elderly and those with challenged immune systems. As there is no expected health benefit to any transgenic food over a non-transgenic food medical ethics would require that a medical practitioner advise patients to avoid genetically engineered sourced foods.

Prior to their release, the consensus of scientists working at the US FDA was that transgenic foods were inherently dangerous, and might create hard-to-detect allergies, poisons, gene transfer to gut bacteria, new diseases, and nutritional problems. They urged rigorous long-term tests.[vi]

In a recent review of transgenic crops based on cultivation since 1996, the US Department of Agriculture said, "it is not clear that the first generation GE seeds will benefit farmers indefinitely." It is certain they do not benefit consumers.[vii]

Studies to prove the safety claimed have simply not been adequately carried out.[viii] Most studies to claim transgenic food crops to be safe are run for relatively short periods and largely conducted by the developer of the seed; a body that will benefit from sales of the product. This practice is inadequate, unacceptable and irresponsible. We refer you to the guidelines issued by the European Food Safety Authority for two-year whole food rodent feeding studies to assess the risks of long-term toxicity and the establishment of protocols for case-by-case studies.[ix] [x] Following these guidelines would provide much better testing of transgenic foods than is currently required. Although such basic animal tests can not prove the safety of the transgenic food in the human diet, any animal toxicity they reveal may well provide a warning in regards to the effect of the transgenic foods on human consumers.

The 2014 'Hot Debate' at Lincoln University featured six experts, including scientists from Crown Research Institutes. The panel scientists who stated GE foods were safe to eat were asked if they could provide 10 human studies to back up their statements, and to advise where the diagnostic tools are available for health professionals to identify if GE food in the human diet may be contributing to illness. Known genetic engineering proponents, Dr Jon Hickford and Dr Tony Connor, admitted there are no such studies or the diagnostic tools for monitoring the impacts on public health of GE food.[xi]

2 – Transgenic foods, pesticide-resistance and human health

It is largely because of failures in crops resistant to glufosinate ammonium, glyphosate and other pesticides that seed developers want approval for transgenic crops resistant to more toxic chemicals.

2,4-D (2,4-Dichlorophenoxyacetic acid)

In an epidemiological study involving male pesticide applicators in the US, relative telomere length (RTL) of buccal cells was found to decrease significantly in association with increased lifetime days of exposure to 2,4-D, indicating increased cancer risk (Hou et al 2013).[xii] A case-control study in British Columbia found a significant association between prostate cancer risk and exposure to 2,4-D (Band et al 2011).[xiii]

A cross-sectional population study in the US, based on the National Health and Nutrition Examination Survey III, indicated that exposure to 2,4-D was associated with changes in biomarkers that, based on the published literature, have been linked to risk factors for acute myocardial infarction and type-2 diabetes (Schreinemachers 2010).[xiv] A multicenter case-control study comparing lifelong occupational and job task histories to determine associations with parkinsonism, found an elevated risk with use of 2,4-D (Tanner et al 2009).[xv]

2,4-D acid iso-octylester caused the formation of atypical cell foci (ACF) in the pancreata and livers of rats, indicating the herbicide's potential as a cancer initiator (Kalipci et al 2013)[xvi] and induced DNA damage in a comet assay on a fish cell line, epithelioma papillosum cyprini (Bokán et al 2013).[xvii]

In a study of the effects of 2,4-D on brain monoamines and the serum level of hormones involved in milk synthesis and on the milk ejection reflex in rats, the herbicide was found to cause a dose dependent decrease in the amount of milk ejected and circulating prolactin and oxytocin secreted in response to the suckling stimulus. The mechanism was thought to involve stimulation of hypothalamic nitric oxide synthase and dopamine, and the inhibition of hypothalamic serotonin transmission (Stürtz et al 2010).[xviii] Other studies identified significant associations between impaired semen parameters and 2,4-D.[xix xx xxi]

2,4- D was described as a respiratory allergen, following intratracheal exposure of mice, which resulted in immune responses characteristic of immediate-type respiratory reactions, as evidenced by increased total IgE levels in both serum and bronchoalveolar lavage fluid (BALF); an influx of eosinophils, neutrophils, and chemokines (MCP-1, eotaxin, and MIP-1) in BALF; increased surface antigen expression on B-cells IgE and MHC class II production) in both auricular and the lung-associated lymph nodes; and increased Th2 cytokine production (IL-4, IL-5, IL-10, and IL-13) in both auricular and the lung-associated lymph node cells (Fukuyama et al 2009).[xxii]

See also Addendum A.

Glufosinate ammonium

> Glufosinate ammonium inhibits the enzyme glutamine synthetase, necessary for the production of glutamine and for ammonia detoxification. It inhibits the same enzyme in animals.
>
> It is acknowledged as not fully effective in controlling weeds in a transgenic crop to an economic level and reports say 90 percent of US farmers also spray with the herbicide atrazine to improve results. Atrazine is an endocrine-disrupting herbicide taken off the market in Europe more than a decade ago.[xxiii]
>
> One study suggested exposure to even low doses of glufosinate in the infantile period in rats causes changes in the kainic acid receptor in the brain.[xxiv] In another study, mouse embryos exposed to glufosinate in vitro developed apoptosis (fragmentation of the cells leading to cell death) in the neuroepithelium of the brain.[xxv] An earlier study found all embryos in treated groups had specific defects including overall growth retardation, increased death of embryos, hypoplasia (incomplete g/ml, and cleft lips at 20μ development) of the forebrain at 10g/ml.[xxvi]
>
> Despite approving glufosinate in its assessment 'Conclusion regarding the peer review of the pesticide risk assessment of the active substance glufosinate', the European Food Safety Authority states under Critical Areas of Concern, "An acute dietary risk has been identified for toddlers for the representative use (of glufosinate-ammonium) as crop desiccant". It also says "exceedance of the ARfD relevant for children (0.045 mg/kg bw/d), although slight, is a major area of concern due to the narrowness of the margin existing between the ARfD and the level causing mortality in dogs."[xxvii]

A 2010 study found pesticide exposure in general resulted in reduced fertility in males, genetic alterations in sperm, a reduced number of sperm, and damage to germinal epithelium and altered hormone function.[xxviii] In a study published in December 2013, researchers tested the toxicity of nine pesticides involving the active ingredient and the added ingredients. Their results "challenge the relevance of the Acceptable Daily Intake for pesticides because this norm is calculated from the toxicity of the active principle alone. ... Chronic tests on pesticides may not reflect relevant environmental exposures if only one ingredient of these mixtures is tested alone."[xxix xxx xxxi]

Statistics show relevant pesticides use in the US has almost doubled since the introduction of genetically engineered crops grown commercially. Farmers are spraying more simply because they can without harming their herbicide-resistant crops. This process of over spraying leaves standing crops contaminated with increased residual spray and these same plants then grow in ground retaining above-the-norm residues of the chemical spray/s, residues which they can uptake.

Desiccation, spraying close to harvest to suggest uniform maturity and facilitate easy lifting of the yield, also leaves significant residual chemical/s on the crops close to harvesting. MAFF UK states that when used as a desiccant, glufosinate residues are detectable in dried peas, field beans, wheat, barley, oilseed rape, and linseed, all of which can be used as food or feed ingredients. Wheat grain containing residues ground into flour retained 10-100% of the residue; bran residue levels 10-600% of those in grain.[xxxiv] Such residues or a significant portion would be ingested.

Transgenes express in the xylem of plants: leaves, fruit, flowers, pollen, nectar, and guttation fluid of plants. Whatever part of a transgenic plant is used as a food or food ingredient, the consumer will ingest transgenes. The cumulative effects of ingesting growing quantities of multiple and substantially different sequences of novel DNA on a daily basis, potentially for a lifetime has not been pursued officially.

Effectively, populations, especially in the US, have unknowingly acted as guinea pigs for an ongoing experiment that no official body is monitoring or evaluating. Indicative of the effects could be the fact that in the first nine years following commercial introduction of transgenic crops in 1996, the incidence of US citizens with three or more chronic diseases nearly doubled; 7% to 13%.[xxxv] Professional bodies point to the evidence accumulating that consuming genetically engineered foods has adverse effects on human health.[xxxvi xxxvii xxxviii xxxix] Medical professionals and veterinarians in the US are advising patients, pet owners and farmers not to eat transgenic foods or feed them to pets or livestock. The results reported are substantial improvements in health and well-being.

In 2011, 90 percent of the US cotton crop was transgenic,[xl] a statistic which suggests 90 percent of ingested food products containing cottonseed derivatives can potentially contain transgenic DNA. Where it was assumed 50% of the diet came from transgenic foods and transgenes represent an estimated 0.0005% of the total DNA in food, one study put the consumption figure at 0.5–5 μg/day. While DNA is claimed as mostly degraded during the industrial process and in the digestive tract, fragments have been detected in body tissues such as leukocytes, liver, spleen and gut bacteria[xli]. Fragments of orally administered phage M13 and plant DNA were taken up by phagocytes as part of their normal function as immune system cells.[xlii] Fragments could pass into other organs, including a foetus.[xliii]

In food crops developed to resist 2,4-D and glufosinate ammonium consumers will, without knowing, be ingesting the resistant transgene/s, even if as minute fragments, from whatever part of the plant they consume. They will also ingest residues of liberal herbicide applications.[xlviii] Pesticides also include adjuvants and surfactants, ingredients frequently more toxic than the pesticide itself.[xlix]

Official bodies accepting the word of developers, and vested interests continuing to deny the possibility of adverse effects, does not mean none exist.[liii]

There is support for the specificity of the association of transgenic foods and specific disease processes. Multiple animal studies show significant immune dysregulation, including upregulation of cytokines associated with asthma, allergy, and inflammation.[lvii]

The American Academy of Environmental Medicine[lviii] has stated, “GM foods pose a serious health risk in the areas of toxicology, allergy and immune function, reproductive health, and metabolic, physiologic and genetic health and are without benefit. There is more than a casual association between GM foods and adverse health effects. There is causation as defined by Hill's Criteria[lix] in the areas of strength of association, consistency, specificity, biological gradient and biological plausibility. The strength of association and consistency between GM foods and disease is confirmed in several animal studies.” It also says, “Multiple animal studies show significant immune dysregulation," including increase in cytokines, which are "associated with asthma, allergy, and inflammation"—all on the rise in the US. The Academy says animal studies also show altered structure and function of the liver, including altered lipid and carbohydrate metabolism as well as cellular changes that could lead to accelerated aging and possibly lead to the accumulation of reactive oxygen species (ROS).[lx] Changes in the kidney, pancreas and spleen have been documented.[lxi]

Ingested transgenic DNA does transfer to gut bacteria. Studies found intestinal damage in animals fed transgenic foods, including proliferative cell growth[lxii] and disruption of the intestinal immune system.[lxiii] In 2004, it was proven transgenes move from ingested food to bacteria in the human gut.[lxiv] An earlier, four-year study, found the transgene conferring resistance to glufosinate had transferred in bees' guts to microbes.[lxv] Since the pat gene can transfer to gut bacteria in bees, and since genetic material from transgenic soy can transfer to human gut bacteria, the pat gene can potentially transfer from any transgene to human intestinal flora.

There is also an absence of substantive data on the potential interactions of chemicals that a transgenic product has been designed to resist and an absence of data to assess potential human health risks through unique chemical combinations in food accepted as probable or feasible. This is an unmanaged risk. It is crucial to prevent the foregoing risks becoming reality in the interests of public health, and to meet FSANZ's mandated duty of care. The cost to the Health System of ignoring risks could be huge. Equally, without comprehensive labelling, consumers will not know they are ingesting resistant transgene/s, even if as minute fragments. They will also be exposed to residues of greater than average herbicide applications, and be exposed to the spray regime associated with plant desiccation prior to harvest. All this is without monitoring of health effects or independent studies.

PSGR urges FSANZ to curb the risks now.

Uphold public safety by banning transgenic foods from the New Zealand food supply, as there is no scientific proof that they are equivalent to non-transgenic foods or that they are safe.

If transgenic foods continue to be allowed into the New Zealand food supply FSANZ should insist on comprehensive mandatory labelling to identify them, to warn of potential health risks, and to give consumers a choice.

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Addendum A

Extracts from 'Overview of the toxic effects of 2,4-D'
Sierra Club of Canada

"...there is a large body of evidence indicating major health effects, from cancer to immune-suppression, reproductive damage to neurotoxicity."

In mammals, 2,4-D "disrupts energy production (Zychlinkski & Zolnierowicz, 1990), depleting the body of its primary energy molecule, ATP (adenosine triphosphate) (Palmiera et al., 1994). 2,4-D has been shown to cause cellular mutations which can lead to cancer." This mutagen contains dioxins, a group of chemicals known to be hazardous to human health and to the environment (Littorin, 1994).

"Numerous epidemiological studies have linked 2,4-D to non-Hodgkin's lymphoma (NHL) among farmers (Zahm, 1997; Fontana et al, 1998; Zahm & Blair, 1992; Morrison et al. 1992).

"Multi-centre studies in Canada and in Sweden of members of the general public found a 30-50% higher odds of 2,4-D exposure among people with NHL (McDuffie et al. 2001, Hardell & Eriksson, 1999, Sterling & Arundel, 1986).

"The teratogenic, neurotoxic, immunosuppressive, cytotoxic and hepatoxic effects of 2,4-D have been well documented (Blakely et al., 1989; Sulik et al, 1998; Barnekow et al., 2000; Rosso et al., 2000; Venkov et al., 2000; Charles et al., 2001; Madrigal-Bujadar et al., 2001; Osaki et al., 2001; Tuschl & Schwab, 2003).

"Other researchers publishing in the open scientific literature have reported oxidant effects of 2,4-D, indicating the potential for cytotoxicity or genotoxicity. For example, Bukowska (2003) reported that treatment of human erythrocytes in vitro with 2,4-D at 250 and 500 ppm resulted in decreased levels of reduced glutathione, decreased activity of superoxide dismutase, and increased levels of glutathione peroxidase. These significant changes in antioxidant enzyme activities and evidence of oxidative stress indicate that 2,4-D should be taken seriously as a cytotoxic and potentially genotoxic agent."

It continues: "2,4-D causes significant suppression of thyroid hormone levels in ewes dosed with this chemical (Rawlings et al., 1998). Similar findings have been reported in rodents, with suppression of thyroid hormone levels, increases in thyroid gland weight, and decreases in weight of the ovaries and testes (Charles et al., 1996). The increases in thyroid gland weight are consistent with the suppression of thyroid hormones, since the gland generally hypertrophies in an attempt to compensate for insufficient circulating levels of thyroid hormones. Thyroid hormone is known to play a critical role in the development of the brain. Slight thyroid suppression has been shown to adversely affect neurological development in the foetus, resulting in lasting effects on child learning and behaviour (Haddow et al., 1999).

"2,4-D causes slight decreases in testosterone release and significant increases in oestrogen release from testicular cells (Liu et al, 1996). In rodents, this chemical also increases levels of the hormones progesterone and prolactin, and causes abnormalities in the oestrus cycle (Duffard et al, 1995).

"Male farm sprayers exposed to 2,4-D had lower sperm counts and more spermatic abnormalities compared to men who were not exposed to this chemical (Lerda & Rizzi, 1991).

In Minnesota, higher rates of birth defects have been observed in areas of the state with the highest use of 2,4-D and other herbicides of the same class. This increase in birth defects was most pronounced among infants who were conceived in the spring, the time of greatest herbicide use (Garry et al, 1996).

"2,4-D also interferes with the neurotransmitters serotonin and dopamine. In young organisms, exposure to 2,4-D results in delays in brain development and abnormal behaviour patterns, including apathy, decreased social interactions, repetitive movements, tremor, and immobility (Evangelista de Duffard et al, 1995). Females are more severely affected than males. Rodent studies have revealed a region-specific neurotoxic effect on the basal ganglia of the brain, resulting in an array of effects on critical neurotransmitters and adverse effects on behaviour (Bortolozzi et al., 2001).

"A peer-reviewed, developmental neurotoxicity study demonstrated severe neurotoxicity in young rats exposed to 2,4-D from postnatal days 12 to 25 at doses of 70 mg/kg/day. These pups showed decreases in GM1 level, diminution in myelin deposition and alterations in all behavioural tests at all doses (Rosso et al, 2000). This herbicide specifically appears to impair normal deposition of myelin in the developing brain (Duffard et al., 1996). The neurotoxic and anti thyroid effects of 2,4-D make it highly likely that foetuses, infants, and children will be more susceptible to long-term adverse health effects from exposure to this chemical although they may appear normal at birth. Young animals can also be exposed to 2,4-D through maternal milk. Recent research has revealed that 2,4-D is excreted in breast milk, thereby resulting in potentially significant exposures to the nursling. The researchers detected 2,4-D residues in stomach content, blood, brain and kidney of 4-day-old neonates fed by 2,4-D exposed mothers (Sturtz et al., 2000). When maternal exposures stopped, the chemical continued to be excreted in maternal milk for a week. Thus, postnatal exposures to this chemical during the critical period for development of the infant brain are of serious scientific concern."

Sierra Club of Canada
http://www.sierraclub.ca/national/programs/health-environment/pesticides/2-4-D-overview.pdf

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