# Report on animals exposed to GM ingredients in animal feed.

prepared for the Commerce Commission of New Zealand
by Professor Jack A. Heinemann, PhD
24 July 2009

Table of Contents

## Abbreviations

| | |
|---|---|
| ALT | Alanine aminotransferase |
| CAT | Catalase |
| DNA | Deoxyribonucleic acid |
| EFSA | European Food Safety Authority |
| ELISA | Enzyme-linked immunosorbent assay |
| EU | European Union |
| FC | Fibrillar centres |
| GE | Genetically engineered/engineering |
| GGT | Gamma glutamyltransferase |
| GI | Gastrointestinal |
| GIT | Gastrointestinal tract |
| GM | Genetically modified |
| GMO | Genetically modified organism |
| HFA | Human flora associated |
| IgA | Immunoglobulin A |
| IgG | Immunoglobulin G |
| LDH | Lactate dehydrogenase |
| mRNA | Messenger RNA |
| NZ | New Zealand |
| PCR | Polymerase chain reaction |
| RNA | Ribonucleic acid |
| SER | Smooth endoplasmic reticulum |
| SOD | Cu/Zn-superoxide dismutase |
| TAG | Triacyl glycerol |
| UK | United Kingdom |
| USA | United States of America |

Gendora, Ltd.

## Opening

This is my expert opinion based on experience and research (full CV attached in Appendix One) in relation to the questions posed by the Commission and outlined in the Summary below. All assertions I make and conclusions that I draw are my opinion.

In brief, I am a professor of genetics and molecular biology primarily employed by the University of Canterbury, Christchurch, but I consult with permission under the name Gendora, Ltd. (http://gendora.net/). Previously, I was a staff fellow at the National Institutes of Health, Institute of Allergy and Infectious Diseases in the USA. My PhD in Molecular Biology was conferred by the University of Oregon, Eugene, USA and my dual undergraduate degrees in biochemistry and molecular biology by the University of Wisconsin, Madison, USA. I represented the University of Canterbury at the Royal Commission on Genetic Modification. I served a Parliamentary Select Committee as an expert witness on "Corngate". I am listed as a United Nations Expert in Biosafety, serve on the Ad Hoc Technical Experts Group for the Protocol on Biosafety (United Nations), and have authored nearly one hundred peer-reviewed or scholarly publications in books and journals such as *Science, Nature, Nature Biotechnology, Trends in Biotechnology* and others. I have provided expert advice to agencies of the USA, New Zealand and Norwegian Governments.

I have no financial conflicts of interest in this matter. As far as I am aware, I hold no investments in Inghams Enterprises or its competitors and I have never received research funding from Inghams Enterprises or its competitors.

## Summary of opinion

The Commerce Commission requested that I research and report to the Commission on whether animals exposed to feed containing genetically modified material ("GM feed") do in fact contain "no GM [genetically modified] ingredients". The provision of expert opinion to the Commission was sought in relation to 'Inghams Enterprises (NZ) Pty Limited chicken product/s as advertised as containing "no added hormones, GM [genetically modified] ingredients" and sold in New Zealand. I was to comment on (including comment on the likelihood of the event occurring) with regard to GM plants used in food or feed:

- could DNA from GM plants be transferred to the animal;
- could GM plants be incorporated into other products sold as chicken products, including breading or stuffing;
- could proteins from GM plants be transferred to the product or could the GM feed alter metabolites in the animal;
- could the GM feed cause physiological or immunological responses in the animal?

I was not asked to consider the validity of safety claims made in the name of GM-free or GM-containing products, biological significance of any reported effects in animals exposed to this material, or to evaluate animal welfare issues.

The issue in essence is herein framed as not whether GM feed makes a chicken a product of gene (or more commonly called, modern) biotechnology (i.e., a GM chicken), but whether the use of GM feed itself might be a GM ingredient.

There is substantial and credible literature that reports the detection of DNA and protein unique to GM plants within animals and animal products. In the absence of competent and dedicated testing to the contrary, it is not possible to conclude that animals and derived products are free of GM material when they have been exposed to GM plants through i) feeding, ii) proximity to other animals on GM feed, or iii) subsequent processing. The most consistent finding in the literature is that animals not exposed to GM feed were unlikely to be contaminated with GM material.

There is compelling evidence that animals provided with feed containing GM ingredients can react in a way that is unique to an exposure to GM plants. This is revealed through metabolic, physiological or immunological responses in exposed animals. In the absence of appropriate testing, it is not possible to conclude that an effect of growing an animal on GM feed will not persist to the final product even in the absence of residue from the GM material.

The cumulative strength of the positive detections reviewed below leave me no reasonable uncertainty that GM plant material can transfer to animals exposed to GM feed in their diets or environment, and that there can be a residual difference in animals or animal-products as a result of exposure to GM feed.

**Explanation of opinion**

*Background*
Genetic engineering/modification (GE/GM) is one of a family of techniques that are internationally recognised under the heading "modern biotechnologies" and the products of these techniques are regulated separately from other biotechnologies for assuring their safety to human health and the environment (Biosafety Assessment Tool, 2009, Heinemann, 2009). Genetic modification involves removing genetic material (nucleic acids such as DNA) from the normal physiological context of a cell or virus and introducing it into another organism. The technique can introduce new, or delete existing, genetic material. Either outcome creates a genetically modified organism (GMO). A GMO is made through the use of genetic material from any source whether or not of the same species. Even if DNA were isolated from and then introduced back into one-in-the same individual, the organism would become a GMO.

Most, perhaps all, commercial GM plants available now for use in making animal feed are created by the insertion of DNA. Most of these plants are designed to produce one or more proteins according to the code of the inserted DNA, and that then impart an

agronomic trait such as herbicide or pest tolerance (IAASTD, 2009). That DNA and any associated gene product in the GM plant can be consumed by and may persist in animals.

Animals exposed to GM plants through inhalation or feed may react to their unique composition. This reaction may be seen as changes in physiology, metabolites or an immune response.

In considering the statement "no GM ingredients", I was to comment on (including comment on the likelihood of the event occurring) with regard to GM plants used in food or feed:

- could DNA from GM plants be transferred to the animal;
- could GM plants be incorporated into other products sold as chicken products, including breading or stuffing;
- could proteins from GM plants be transferred to the product or could the GM feed alter metabolites in the animal;
- could the GM feed cause physiological or immunological responses in the animal?

To advertise that something has no GM ingredients is to make a claim that is understood in some way by consumers. There is at least evidence from overseas that such labels appeal to some consumers. A survey conducted in the USA found that nearly a third of respondents to the question "would you be 'willing to consume meat products from cows or chickens fed on GM corn or soybeans?'" responded in the negative (Onyango et al., 2004). A second USA-based survey found that a large majority of Americans wanted chickens fed GM plants to be labelled as such, a simple majority associated some health risk with chickens raised on GM feed (Bernard et al., 2005).

European Union regulations presumably also preserve the consumer's choice to avoid GM ingredients when the GMO may be present (above a threshold limit) and in addition to the animal that may have eaten it (p. 4 Asensio et al., 2008):

> Additionally, according to Regulation (EC) 1830/2003 of the European Parliament and of the Council, traceability requirements for food and feed produced from genetically modified organisms (GMOs) should be established to facilitate accurate labeling of such products, in accordance with the requirements of Regulation (EC) 1829/2003 on genetically modified food and feed. Therefore, foods and food ingredients that are to be delivered to the final consumer in which either protein or DNA resulting from genetic modification is present, are subjected to additional specific labeling requirements.

However, the EU does not require labelling simply because GM feed was used (Kain, 2007, Novoselova et al., 2007).

Retailers are linking the use of GM feed with the GM status of their animal products (EU Commission). For the United Kingdom and Ireland:

> "All of Marks & Spencer's fresh meat and poultry, salmon, shell eggs and fresh milk comes from animals fed on a non-GM diet. The Kepak Group, which controls 60% of Irish beef exports, requires some farmers who produce meat for its flagship KK Club brand to exclude the use of GM animal feed.
> "All Kepak's chicken meat comes from birds reared on a vegetarian, non-GMO diet. The Silver Pail Dairy in Co Cork has signed multi-million euro foreign direct investment deals with Baskin Robbins (the world's largest ice-cream retailer) and with Ben & Gerry's, to produce GM-free ice cream (made from milk from cows fed a certified non-GMO diet) for the European market.
> "Tesco, Sainsburys, M&S and Budgen Stores all have quality labels for meat and dairy produce from livestock fed on certified GM-free animal feed. All of Marks & Spencer's fresh meat and poultry, salmon, shell eggs and fresh milk comes from animals fed on non-GM diet. Moreover, standard poultry sold in most UK supermarkets now carries a label certifying GM-free feed" (GMO Free Regions).

Similar practices are reported for Italy, France and Switzerland. TraceConsult™, which describes itself as a consultancy, reported on 20 July 2009 that the Swedish Dairy Association "were suddenly unable to continue their claim of supplying GMO-free milk" due to inadvertent distribution of GM feed to member farmers (TraceConsult). According to a translation of the Swedish agricultural business newspaper ATL, the Swedish milk giant "Arla was informed [of the feed mix-up] earlier in the week. The company has promised consumers that their milk is GM-free in every step. 'Now we cannot keep that promise, which is a concern'" (TraceConsult).

Consumers may have different and complex reasons for wishing to avoid GM ingredients (Frewer, 2003, Novoselova et al., 2007). As the UK Food Standards Agency says: "some people will want to choose not to buy or eat genetically modified (GM) foods, however carefully they have been assessed for safety" (UK FSA). It is not within the brief of this report to list or evaluate what those reasons may be. However, I also do not assume that all consumers of this type wish to avoid GM ingredients solely because they are reacting to the DNA that may have been used to produce GM plants, or the unique protein(s) that those plants make. There are other associated social issues, agricultural technologies and processes that are inseparable from the use of GM plants. For example, most GM soybeans are modified to be tolerant of a commercial herbicide which, because of the modification, may be applied directly to the GM soybeans, more frequently or at higher doses than it could be on conventional soybeans. A consumer may be wishing to avoid any food chain effect of the herbicide. The market-dominating herbicides and their corresponding tolerant GM maize, cotton, oilseed rape, and soybean varieties are owned by large multinational corporations. A consumer may wish to avoid contributing to this kind of business (Novoselova et al., 2007).

I was not asked to consider the validity of safety claims, e.g., whether eating GM plants poses an overall health risk to the animal or transfers a health risk to humans through the animal. Likewise, whether significant differences between animals fed GM-derived substances were of 'biological significance', or within the range of physiological diversity seen in those species, was not considered.

Much research in this field is meant to contribute to the formation of a judgment about the overall similarity between GM and conventional organisms, or to detect an adverse effect of genetic engineering or from consuming a GMO. Papers that may report significant differences may not herald these facts in the abstract, summary or conclusion, because the presence of significant differences was not the focus of the research exercise. The focus of many of these papers is on endpoints not pertinent to the matter at hand. Conclusions of overall nutritional equivalence or efficacy, animal performance and health do not establish or disprove the possibility that animals provided with GM feed, or in the proximity of other animals provided this feed, are changed in a measurable way. My purpose was to consider whether there was evidence that animals eating GM plants could be demonstrated to be different from those that have not, in the ways outlined below, regardless of whether any individual difference would be sufficient to cause the authors of the research to be concerned about overall adverse effects or performance.

Does the current evidence support the contention that a consumer would be, with a high likelihood, able to avoid ingestion of DNA, protein or other substances that might be unique to a GM plant or its method of cultivation and processing, or able to avoid animal physiological or immunological responses to substances unique to GM plants, through consumption of animals raised on GM feed (Figure 1)? The answer is no.

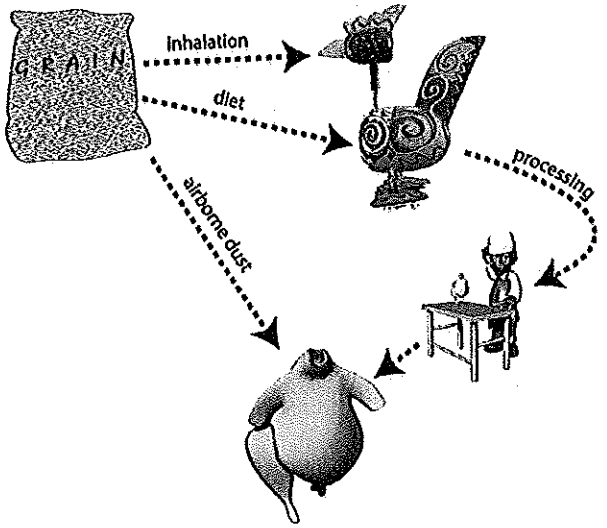



Figure 1. Pathways of exposure to GM plant material.

The research is clear on the following. If a consumer were avoiding the ingestion of DNA unique to a GM plant by avoiding animals fed GM plants, then this consumer would have a high likelihood of success purchasing meat products from animals raised on GM-free feed. For products that are breaded or stuffed, that consumer could probably avoid

exposure to the DNA unique to a GM plant if the ingredients in the breading and stuffing were certified organic or GM-free. If a consumer were avoiding the ingestion of proteins or metabolites unique to GM plants, then this consumer would have a high likelihood of success purchasing meat products from animals raised on GM-free feed. If a consumer were avoiding the ingestion of metabolites or proteins in animals that were only present, or present at different concentrations, when the animal was fed a GM plant, then this consumer would have a high likelihood of success purchasing meat products from animals raised on GM-free feed.

*A priori*
Commerce Commission investigators provided me with copies of Inghams' advertisements. Claims in these advertisements and others I sourced independently are represented by the following selected quotes:

> "Ingham is committed to sourcing non-GM ingredients for its poultry feeds and uses its best endeavours to source non-GM ingredients. Because these ingredients must meet specific quality standards and be available in quantities that are economically sustainable, Ingham chickens may sometimes consume poultry feed which could contain GM ingredients. This does not however compromise the absolute GM-free status of Ingham chicken products.
>
> "Research confirms that animals that consume feed with a component of GM are no different compared to animals that have been fed a low GM or GM free diet.
>
> "Inghams meets or exceeds all regulatory guidelines, script of practice and standards in New Zealand and Australia...As is the case with all Inghams products, our chickens contain no GM content and are not genetically modified."
> And
> "The use of GM Soya in feed does not compromise the absolute GM-free status of the poultry products the company produces. Animals that eat feed with a component of GM Soya are no different to other animals that may have been fed a low GM or GM-free diet. This position is verified by numerous feeding studies:
>
> (i) 'NZ Royal Commission Report & Recommendations (2001)'
> (ii) 'Federation of Animal Science Societies (2000) FASS Facts, On Biotech Crops - Impact on Meat, Milk and Eggs. Savoy IL'
> (iii) 'The Royal Society (2002) Genetically modified plants for food use and human health - an update. Policy document 4/02 (February)'" (http://www.inghams.co.nz/consumernz/aboutus.aspx?docId=285).

Of the documents that Inghams uses as references for its position, all are at least seven years old, which is remarkably old in such an active area of science and intense public interest. Importantly, one of the three references used, The UK Royal Society's 2002 *Update*, does not address the issue of what constitutes "GM free". It mentions a few older animal studies looking for detection of DNA in animals fed GM feed, and concludes that "DNA present in food can find its way into mammalian cells at some low frequency" (p. 9). The document called *FASS Facts* which I sourced from the internet is not a scholarly publication with references, but appears to be a brochure. I reproduce this document in Appendix Two. The NZ Royal Commission reported in Chapter 8 (paragraphs 121-126)

that they had heard from a variety of sources, including the predecessor of Food Standards Australia New Zealand and a submitter from Iowa State University that there were as of 2000-1 no detectable human health issues proven to be related to the use of GM plants as animal feed, and that under present labelling laws animals that consumed GM plants were not considered "genetically modified". While the Royal Commission deliberated on the evidence of safety to humans, I could find no deliberation on the specific issue of whether chickens or other food animals fed GM plants would constitute the use of GM ingredients. Their concluding paragraph on this issue was:

> "Products from animals or birds fed on genetically modified pasture or stock feed do not require assessment under Division 1 of Standard A18 because they are not considered to be genetically modified, nor will they require labeling under the labelling provisions to be implemented later this year. *It is important that consumers are able to choose to avoid consuming the products of animals and birds fed on genetically modified feed. Where a claim that animals and birds have not been fed genetically modified food can be sustained, labelling that identifies the product as being free of genetic modification will be appropriate.* We discuss genetic modification-free labelling later in this chapter. Without such a label, consumers must assume that a genetically modified food may have been used" (paragraph 126, emphasis added).

The above and the Royal Commission's recommendation 8.2:

> "that Government facilitate the development of a voluntary label indicating a food has not been genetically modified, contains no genetically modified ingredients and has not been manufactured using a process involving genetic modifification [sic]"

in my opinion indicate that the Royal Commission saw that it was important to clearly differentiate between that which was GM or raised on GM feed, from those things that were not GM or exposed to GM feed.

In sum, the references that Inghams Enterprises uses to support its claims are both out of date and of questionable support for its policy position.

**Is there evidence of DNA unique to GM plants in animals given GM feed?**

Yes, albeit that DNA is inconsistently detected. Inconsistent detection is not unusual. Especially when the proportion of input material containing the DNA can vary from time to time or between consignments, it would be expected that target DNA sequences in the food chain may fall below limits of detection of present methodologies (Heinemann et al., 2004). Inconsistency in detection is not evidence against the possibility that this material can be found in animals, only that the absolute amounts in animals varies above and below the detection limit (Alexander et al., 2007, Einspanier et al., 2004, Mazza et al., 2005).

There are convincing demonstrations that within animals fed commercial GM plants there can be DNA unique to those plants. Here I summarise examples of positive detections. This is not a comprehensive survey of the literature and not balanced for reports of no detection. For that, see Alexander et al. (2007). The focus here is on positive

detections because the purpose of this report is to establish if the science indicates that the DNA of GM plants can be in animal products.

*Pigs*
Pigs were fed on controlled diets with some groups receiving 60% GM and some conventional maize (Chowdhury et al., 2003). DNA unique to the transgene used in GM maize event Bt11 was detected in pig stomachs, small intestine (duodenal, ileum), rectal and cecal contents but not in peripheral blood. Others have reported detection of DNA unique to GM plants in the blood of pigs fed GM- but not conventional-maize (Mazza et al., 2005). The first set of authors concluded that "maize DNA and GM DNA were considered not totally degraded but rather present in a form detectable by PCR in the gastrointestinal tract" (p. 2549 Chowdhury et al., 2003). PCR is a reaction that is used to amplify DNA, to increase the ability to detect it.

*Cows*
On an estimated consumption of 24kg of dry matter per day, a dairy cow can conceivable consume 54 µg/day of DNA unique to a GM plant (Agodi et al., 2006) and 7.4 mg of protein unique to a GM maize plant such as MON810 (Alexander et al., 2007). Neither proteins nor DNA sequences uniquely from GM plants have been detected by some researchers in the milk of cows fed for short times on GM plants (Guertler et al., 2009, Phipps et al., 2002, Phipps et al., 2003). However, in a survey of milk products sold in stores in Italy, researchers found evidence of target DNA unique to GM plants in 38% of samples, including those labelled "organic" (Agodi et al., 2006). This indicates that longer term animal feeding studies may be necessary in testing done with animals. Another possible explanation for the Agodi et al. (2006) results is bacterial contamination after milking, or contamination of the milk with feed dust after it leaves the animal. While the DNA found in commercial milk products may or may not be the full length of DNA fragments unique to the GM plant, their presence in commercial milk suggests that GM ingredients could persist in animals and cross tissue boundaries or enter the food chain in a form that the consumer could directly experience.

*Fish*
GM plant-specific target DNA was detected in the gastrointestinal (GI) tract of rainbow trout fed on a defatted GM soybean variety. The target DNA was detected for up to three days post transfer to a non-GM diet (Chainark et al., 2008). This DNA was subsequently detected in leukocytes, head kidney and muscle. The target DNA was confirmed to be identical to the DNA in the GM soybeans.

Using Atlantic salmon force fed with purified (naked) DNA added exogenously to food, Nielsen et al. (2005) showed that dietary DNA could transfer to organs. DNA was detected in all three parts of the intestinal contents, blood, kidney and liver (Nielsen et al., 2005). In later studies, the DNA detected in the mid-intestine was shown to be intracellular. "The present findings demonstrate that Atlantic salmon intestinal cells are capable of taking up foreign DNA, both dietary and naked" (p. 541 Sanden et al., 2007).

*Chickens*
Using quantitative PCR the fate of DNA unique to the GM corn Bt176 was followed in broilers. This study found that the DNA was not completely digested and could be detected for various lengths of time post-consumption in the crop, proventriculus, gizzard, small intestine (duodenum, jejnum, ileum) and finally the caeca and rectum (Tony et al., 2003). This same group of researchers reported evidence of plant-specific DNA in the blood, pectoral and thigh muscles, liver, spleen and kidney up to four hours after feeding, but did not detect the DNA unique to Bt176. No further detection was possible after 24 hours from feeding. This finding establishes that DNA can persist, circulate and transfer to deeper tissues although any particular fragment may fall below the detection limit.

Researchers have found plant-specific DNA on chicken meat in supermarkets (Klotz et al., 2002). While the target was not DNA unique to a GM plant *per se*, "it can be considered that an incomplete degradation of ingested DNA fragments may take place in the GI tract of birds, enabling the detection of residual plant gene fragments. Due to a fast passage of feed through the GI tract of avians the appearance of DNA fragments might be more likely than for mammals" (p. 274 Klotz et al., 2002). DNA unique to a GM plant would be as likely to persist in animals fed GM-feed as any plant-specific DNA. These researchers could not distinguish between several causes of DNA on the chickens, including residual undigested DNA from feed or contamination with feed dust which was not removed through the slaughter, preparation and packaging process (Figure 1). They confirmed that the DNA was from an external source and not because the chickens were genetically modified, because the target DNA was not detected in chicken embryos. For the purposes of this report, the cause is irrelevant because whether the GM-specific DNA is present as a partial digestion product on the meat or whether the meat is contaminated as a result of airborne material from GM-feed, it ultimately is on the chicken because of the use of GM feed.

> "In summary, all results coincide with former propositions about a possible transfer of small DNA fragments from feed into distinct farm animals. First data are now available for pigs, and a recent report first observing foreign DNA within various chicken organs is supported" (p. 274 Klotz et al., 2002).

> "All studies on DNA degradation in the GI tract suggest that foreign DNA ingested by animals is not completely degraded in their GI tracts" (p. 380-381 Chainark et al., 2008).

*Rats*
Gnotobiotic (free of intestinal microbial flora) and HFA (rats with a human intestinal microbial flora) rats were fed on maize flour. Using a quantitative PCR technique, a maize-specific single gene (as a surrogate for a GM-specific gene) was detected in the upper GI, from stomach to duodenum, and a gene maintained at multiple copies was detected throughout the GI down to the jejunum, ileum, caecum, colon and in the faeces (Wilcks et al., 2004).

Gendora, Ltd.

*Sheep*
The *cry1ab* toxin gene unique to GM-maize was detected by PCR of rumen juice up to 5 hours after feeding. Targeting a smaller fragment to increase the efficiency of PCR allowed detection up to 24 hours after feeding (Duggan et al., 2003). No DNA was amplified from faeces.

*Comment*
A report from the European Food Safety Authority (EFSA) emphasised negative detections of DNA (EFSA, 2007). A strength of their consideration on the issue of GM feed was to consider the entire supply chain including the effects of ensilaging and processing on the stability of DNA and proteins. They draw on a review by Flachowsky et al. (2007). That review cites a 2003 abstract published in German describing the effects of processing on oilseed rape DNA. This abstract apparently reported a decline in the ability to amplify DNA specific to a variety of GM oilseed rape as it was toasted for longer times. Nonetheless, plant-specific fragments of DNA of at least 248 nucleotide pairs were still detected after three toasting treatments. The most rigorous regime was a series of four toasting treatments from which a GM-specific DNA fragment of at least 194 nucleotide pairs could still be amplified. Similarly, Flachowsky et al. cite a description of one of their own studies also published as an abstract in 2004 which indicates that mechanical treatments had no effect on the stability of DNA from GM maize but ensiling did (reference in Flachowsky et al., 2007). Nevertheless, a DNA fragment of at least 194 nucleotide pairs that was diagnostic of the GM plant was still amplified from ensiled maize after 200 days.

In one study reviewed here, GM plant-specific DNA could not be detected by PCR in the rumen fluid of sheep whereas that DNA could be detected in grain-fed sheep (Duggan et al., 2003). It is clearly possible that processing steps may influence the quantity of full length DNA sequences and full size proteins available to animals.

For the purposes of this report it is not assumed, however, that the entire DNA sequence that was modified using the techniques of modern biotechnology must be recovered to be relevant. If the recombinant DNA material in the GM plant were 5000 nucleotide pairs in length and an unambiguous identification of it could be made from a partially digested or degraded fragment now of a few hundred nucleotide pairs in length, the material is not GM-free any more than would be a plant made into a product of modern biotechnology by the insertion of DNA that was only a few hundred nucleotide pairs in size.

Flachowsky et al. proclaim in the abstract of their review that: "[t]o date, no fragments of recombinant DNA have been found in any organ or tissue sample from animals fed" GM plants (p. 3 Flachowsky et al., 2007). This strong statement seems to have heavily influenced EFSA, but is perhaps misleading. As EFSA admit, the: "DNA introduced into crops through recombinant DNA technology is not different from other sources of DNA in the diet" (p. 2 EFSA, 2007) and this kind of DNA has unambiguously been found in organs and muscle. The proportion of DNA that is being targeted in studies is tiny compared to the total dietary DNA intake by the animal. Based on estimates of dietary DNA a cow might consume in a day (on feed with a 60% GM content), this target is only

0.000094% (or about one 1 millionth) of dietary DNA spread over the volume of the animal (Beever and Phipps, 2001). Thus, any detection of a specific fragment of DNA, which is already at small concentrations in the animal, is actually dramatic evidence that DNA is not thoroughly degraded or digested. These positive detections serve to assure us that DNA survives degradation and digestion because single copy DNA markers can be recovered from animals. Despite the strong statement in the abstract, the authors more cautiously conclude their review by saying:

> "However, in the case that plant DNA-fragments should be absorbed, it might be that transgenic DNA-fragments are also absorbed" (p. 27 Flachowsky et al., 2007).

In fact, Flachowsky et al. (their Table 27) cite four studies in which a plant-specific DNA marker was found in animal muscle, organs, or tissues out of only seven total studies they cite for positive detections of plant-specific DNA in animals. Even in this far from exhaustive survey of the literature, more than 50% of the studies indicated that dietary DNA can pass beyond the GIT of animals and it is only a matter of chance whether the detected DNA is natural to the plant or it is recombinant (a product of modern biotechnology). Furthermore, unlike this report their survey of the literature included papers published only up to 2005.

In most studies in which animals were fed whole foods derived from a GM and conventional plant, control animals and diets were used. In general, no GM-specific DNA was detected on animals not fed material derived from GM plants. Unless there was a breach in handling of material, there appears to be little or no likelihood that a product derived from animals raised on conventional plants will ever have DNA from GM plants. Thus, a consumer choosing chicken and chicken products from a supplier that does not use GM feed could reasonably expect to avoid exposure to GM plant material.

**Is there evidence of DNA unique to GM plants in the stuffing, breading or other products sold as chicken products?**

It is increasingly difficult to source maize and soya flours that are GM-free. However, Inghams Enterprises claims that it tests these ingredients before use.

> "Inghams abides by all regulations in Australia and New Zealand, regarding food safety, labelling and packaging. It has food safety procedures in place to ensure the integrity of all its non-GM ingredients and monitors suppliers to ensure that this high level of integrity is maintained"
> (http://www.inghams.co.nz/consumernz/aboutus.aspx?docId=285).

Provided that this is the case, and that suppliers meet their testing obligation, then the level of GM in these products should be below the labelling threshold if not GM-free.

**Is there evidence of proteins unique to GM plants in animals fed GM plants, or metabolic differences in these animals?**

Yes, but not in every study. This may be expected because of variations in exposure to GM material and accumulations of protein near the limit of detection.

*Pigs*
Returning to the study of pigs fed on either a diet of conventional or GM maize, using both an enzyme-linked immunosorbent assay (ELISA) and immunochromatography researchers found in pigs peptides derived from the protein uniquely produced by the GM maize and only in pigs fed this maize (Chowdhury et al., 2003). Fragments of the target protein were detected in the stomach, duodenum, ileum, cecum and rectum. The concentration of the protein in the rectal contents was only reduced 50% from the concentration in the feed. While detected protein fragments were smaller than the target protein, these fragments were large enough to retain the epitopes used to identify the protein, and were on the order of half the size of the original protein (Chowdhury et al., 2003). Epitopes are structural features of the protein to which an animal raises protein-specific antibodies.

*Cows*
Studies using cows fed conventional or GM (Bt176) maize reported fragments of the protein Cry1Ab, which is unique to the GM maize, in the rumen and intestinal juice and the fragments remained detectable even in the faeces, but not in washed intestinal epithelia tissue. This finding was based on ELISA which can overestimate the amount of full size protein because even fragments large enough to retain a recognition epitope will be detected. In a follow-up study using immunoblotting instead of an ELISA, the majority and perhaps all of the positive results from ELISA were attributed to partially digested but still large (34 of 60 kDa) protein fragments (Lutz et al., 2005).

*Fish*
Atlantic salmon fed on (MON810) GM maize-derived fish meal differed significantly in several metabolites from control animals fed on the conventional equivalent meal (Sagstad et al., 2007).

In another study, Atlantic salmon fed on GM-derived full-fat soybean meal (FFSBM) fish food differed significantly in several metabolites from control animals fed on the conventional equivalent meal. The GM soybeans were modified to be tolerant of the commercial herbicide Roundup and not to alter physiological parameters in animals fed the soybeans. Nevertheless,

> "[m]uscle protein content increased significantly with increased GM FFSBM in diet. Also, there were some small differences in the muscle fatty acid profile between fish fed GM compared to fish fed [non-GM] FFSBM. Fatty acid 22:6n-3 and the ratio n-3/n-6 in muscle increased significantly, and the sum of n-6 fatty acids decreased significantly, with increasing GM FFSBM" (p. 563 Sagstad et al., 2008).

The authors associated lower levels of plasma glucose and triacyl glycerol (TAG) in fish fed on GM with higher levels of 'anti-nutritional factors' in GM compared to non-GM soybeans (Sagstad et al., 2008). In a subsequent study, which may have used different varieties of GM and non-GM soybeans but from this same research group, the plasma TAG levels were significantly higher in fish on GM meal (Sissener et al., 2009). While the actual differences in TAG levels were not reproducible, it is clear that in each case fish on the GM meal had a statistically significant difference in metabolites when compared to fish on the non-GM meal. The authors draw a different conclusion, saying that "[t]he contradictory nature of our results [in the two studies] suggests that this is not a "GM-effect", but rather related to natural variations in levels of anti-nutritional factors, antigens, metabolites or other unknown factors in the plants" such as possible herbicide residues (p. 115 Sissener et al., 2009).

Over the course of three publications (Sagstad et al., 2007, Sagstad et al., 2008, Sissener et al., 2009), this research group consistently saw significant effects of GM-supplemented meal on metabolite levels and physiological parameters. The metabolite and physiological changes were not identical in magnitude and direction, but that is not necessarily a contradiction to be explained. The biochemical path between exposure and biological response has not been identified and thus there is no reason to expect that the biological response will always be in the same direction or of the same magnitude, especially when these studies used different species (soybean and maize), and potentially different varieties[1], of GM plants.

Interestingly, these three studies were based on material supplied by the Monsanto Company, which makes the GM plants used in these experiments. While most other research studies reviewed tested their control diets for contamination by GM plants, there is no mention of independent testing by this research group. It is possible that the results are tainted by contamination, since in other studies where materials are directly sourced from Monsanto the control diets were contaminated with GM material (for example, see Scheideler et al., 2008, Taylor et al., 2003). Contamination of the control diet would most likely cause an underestimation of the number and magnitude of significant differences between diets.

Regardless of whether the consistent observation of differences in nutritionally matched meals is due to changes in the plant's DNA or associated agronomic or processing technologies may not matter to the consumer who may wish to avoid any effects associated with the use of GM plants as animal feed.

*Chickens*

A 2002 study funded by the Agriculture Livestock Industry Corporation found no evidence that the protein unique to the GM maize variety called Starlink could be detected in broiler chicks' blood, liver or muscles (Yonemochi et al., 2002). Again, inconsistencies in detections are not unexpected and the inconsistency of detection does

[1] In Sagstad et al. (2008) the variety of soybean is not reported. In Sissener et al. (2009) the variety of GM soybean is reported as event GTS 40-3-2.

not reduce the certainty that such products are found in animals, only that the absolute amount of the substance varies for complex reasons.

A study conducted by the Monsanto Company found that their test strips for the GM plant-specific protein Cry3Bb1 (MON863) reacted to eggs from test chickens fed both GM-derived feed and conventional feed, as well as eggs purchased from a local store (Scheideler et al., 2008). Monsanto researchers interpreted this result as indicating that the test strip was triggered non-specifically by some other substance in eggs. There is another possibility. The same researchers admitted that the conventional feed used in the study was contaminated with GM maize producing the unique target protein Cry3Bb1 and two of these hens also produced Cry3Bb1 positive faeces (Scheideler et al., 2008). Since GM maize is so common in the USA feed supply, the supermarket eggs could also have been derived from chickens fed GM maize. Thus, the ability of proteins unique to GM feed to pass into eggs is not disproved by this study.

Chickens fed the GM diet had detectable fragments of the Cry3Bb1 protein in their faeces, large intestines, cecums, small intestines and crops (Scheideler et al., 2008). Based on their quantifications, Monsanto estimated that 98-99% of the dietary Cry3Bb1 was digested. However, this is not to completion but to the relatively large fragments of proteins that are still detected by antibody or polyclonal serum binding.

*Comment*
Importantly, in the studies mentioned above, control animals and diets were used. These control animals were fed non-GM equivalent material (for an exception, see the flawed study by Scheideler et al., 2008). In general, no GM-specific DNA or protein was detected from animals not fed material derived from GM plants.

**Is there evidence of physiological or immunological responses specific to GM plants in the animal?**

Most evidence of physiological or immunological response comes from oral ingestion. However, animals often breathe in feed dust which can expose the lungs to proteins unique to the GM plant. Both exposure routes were considered.

*Fish*
Atlantic salmon fed on (MON810) GM maize-derived fish meal differed significantly in the activity of catalase (CAT) and Cu/Zn-superoxide dismutase (SOD) enzymes extracted from livers as compared to fish fed conventional maize meals. CAT and SOD are part of a biochemical pathway that reduces free radicals in cells by converting superoxide anions into hydrogen peroxide and ultimately oxygen and water. There was significantly less CAT and more SOD activity as measured by enzyme extracted from the liver. There was significantly more SOD activity as measured by enzyme extracted from the distal intestine. None of these differences was due to changes in mRNA levels for these enzymes and thus was attributed to enzyme function (Sagstad et al., 2007).

In addition, fish fed GM maize had a significantly higher proportion of granulocytes and a lower proportion of lymphocytes compared to fish on conventional maize diets.

> "Differential leucocyte counts showed altered proportions of white blood cell populations, suggestive of an immune response taking place in the blood as a response to the GM maize in the diet" (p. 210-211 Sagstad et al., 2007).

*Rats and mice*
Rats fed GM rice uniquely producing the Cry1Ab protein or PHA-E lectin were monitored for allergic responses (Kroghsbo et al., 2008). Some of the most significant changes were observed in rats on the GM diet for 90 days, where the PHA-E lectin caused a dose-dependent increase in IgA (immunoglobulin A) levels, and the absolute and relative weight of mesenteric lymph nodes were increased in these animals (references within Kroghsbo et al., 2008). Rats fed GM rice uniquely producing Cry1Ab had significantly higher white blood cell counts and male rats had reduced adrenals.

Most striking, this study found an antigen (i.e., Cry1Ab or PHA-E)-specific IgG response even in control animals (those not fed the GM rice).

> "As the nasal and bronchial mucosal sites are potent sites for induction of an immune response, the results may be explained by inhalation of particles from the powder-like non-pelleted diet containing PHA-E lectin or [Cry1Ab] toxin, thereby inducing an anti-PHA-E or anti-[Cry1Ab] response...These results support our assumption that the induction of the [Cry1Ab]-specific antibody response in the control groups occurred after inhalation" (p. 31 Kroghsbo et al., 2008).

Thus, exposure to GM plant material could cause immunological changes in animals even if the material is kept out of their food but is used in animals contained within range of the feed dust.

In another study in which rats were fed meal using GM or non-GM soya, there were reported differences in plasma amylase levels between the two groups of animals. Animals fed the GM soya had a transient depletion in zymogen granules and an increase in pancreas acinar cell disorganisation, similar to what is observed in pancreatitis. Zymogens are inactive enzymes that are secreted from the pancreas and activated when needed. Their transient depletion may indicate that the cells recuperated in time. "The results appear to indicate that rats fed on a GM diet had a pancreatic supraphysiological stimuli or synergism with cholecystokinin (CCK); although not severe, it was sufficiently strong to induce a mild pancreatic injury with an adaptive response" (p. 224 Magaña-Gómez et al., 2008).

Pancreatic acinar cells were also the focus of studies involving the feeding of a GM soya diet to mice, compared to a non-GM control soya diet (Malatesta et al., 2003). The soybean component of both diets was 14% and the mice presumably began this diet at weaning and were sacrificed for analysis at 1, 2, 5 or 8 months of age. Their pregnant mothers were also fed the same diet before they were born. In this study, more fibrillar centres (FCs) were observed in GM fed mice, and they were on average much smaller in

GM fed mice compared to those observed in mice on the control diet. FCs are found in the primary nuclear organelle called the nucleolus, the site of ribosome biogenesis (Raska, 2003). The authors interpreted this as indications that in GM soya fed mice, nucleolar activity is depressed and there could be more general effects on RNA processing, ultimately affecting the production of some enzymes in animals on GM feed.

Hepatocytes from the liver of mice were examined after they were maintained on a 14% GM or conventional soya diet (Malatesta et al., 2002).

> "Hepatocytes are involved in numerous metabolic pathways: they metabolise and transform most of the products of digestion, degrade and detoxify substances and excrete them in the bile, synthesise many protein components of blood plasma and are able to store glycogen and to release glucose, thus playing a primary role in the maintenance of carbohydrate homeostasis" (p. 179 Malatesta et al., 2002).

Their mothers had been introduced to the same diet (either GM or conventional) during pregnancy. The younger mice began the diet after weaning and were sacrificed for analysis at 1, 2, 5 or 8 months old. While gross features of the mice and liver were the same between the groups, there were noticeable differences at the sub-cellular level. For example, hepatocyte nuclei in GM-fed animals had irregular shapes compared to mice on GM for less than one month and the control group throughout the study. The nucleoli of GM fed mice were also irregular and less compact, which the authors associated with a higher metabolic rate (Malatesta et al., 2002). As above, differences in FCs were observed. "[I]n our animals the modifications of FC size...are related to food only" (p. 178 Malatesta et al., 2002).

In an innovative follow-up study, the mice raised from weaning to three months old on the GM diet were given conventional soya in their food and vice versa for the conventional control group for one additional month (Malatesta et al., 2005). Mice that swapped a conventional for a GM soya diet had more FCs with an associated increase in the dense fibrillar component, whereas the other group had more compact nucleoli and fewer FCs with a pronounced granular component. The diet swapping experiment caused the differences between the mice to reduce, indicating that the some or all effects of GM feed may be reversible, and that the GM feed is able to induce rapid changes even in adults (Malatesta et al., 2005).

Male mice born of mothers fed either a 14% GM soya or conventional soya diet, and then maintained on the parental diet following weaning until 2, 5 or 8 months old had observable differences in Sertoli cells of the seminiferous tubule, spermatogonia and spermatocytes (Vecchio et al., 2004). Sertoli cells had enlarged vesicles of the smooth endoplasmic reticulum (SER) in GM-fed mice. There was a transient (between 2 and 8 months) increase in the size of nucleoli in GM-fed mice. Perichromatin granules were increased, and the number of nuclear pores decreased, in both Sertoli cells and spermatocytes of mice on a GM diet (Vecchio et al., 2004). The authors associated these changes with a transient decrease in transcriptional activity in these cells. Transcription is the central biochemical pathway by which RNA is made. RNA is a key co-factor in

protein synthesis and is a catalytic component of ribosomes. Various RNA molecules perform roles in regulating gene expression and RNA-processing reactions.

The physiological effects of GM feed observed in this study reversed by eight months of age, except for SER dilation (Vecchio et al., 2004). The authors attributed this effect to either persistence of herbicide residues uniquely on herbicide-tolerant GM soybean varieties or an unanticipated effect of the genetic engineering itself.

Rats fed on a diet with GM-, expressing a lectin for the purpose of pest tolerance, or conventional-potato content had significant histopathological differences. Mucosal linings from the stomach were thicker for rats on the GM feed (or on conventional supplemented with purified lectin). Crypt lengths of the jejunum were greater in rats on GM potato (and not on conventional or conventional supplemented with lectin) diets (Ewen and Pusztai, 1999).

A study originally conducted under contract to the Monsanto Company in which rats were fed GM maize (MON863) or a control diet of conventional maize was reanalysed by independent researchers (Seralini et al., 2007). This reanalysis found evidence for multiple GM-feed-specific physiological changes in the liver, kidney, pancreas and bone marrow of rats, some of which were sex-specific. Liver alkaline phosphatase and alanine or aspartate aminotransferase activities differed by 8-23% in GM and non-GM fed rats.

The Seralini et al. (2007) study was affirmed by an Environmental Science and Research (ESR) Ltd. analysis (Gallagher, 2007) and later by a second review of the data again published under the same lead author but including the ESR, Ltd. author (Seralini et al., 2009).

*Sheep*
Sheep were fed on hay supplemented with GM (Bt176) or non-GM maize over a three year period. Using a staining technique, the researchers found evidence of significantly different levels of proliferative activation of ruminal epithelium basal cells in ewes fed GM maize (Trabalza-Marinucci et al., 2008). "Moreover, preliminary [electron microscopy] analyses of hepatocytes and pancreatic acinar cells revealed smaller, irregularly shaped cell nuclei containing increased amounts of heterochromatin and perichromatin granules (ribonucleoprotein structural components involved in transport and/or storage of already spliced pre-mRNA)" in lambs fed GM maize (p. 186 Trabalza-Marinucci et al., 2008).

*Rabbits*
New Zealand rabbits were fed either a diet supplemented with GM-soya (Roundup Ready brand) or conventional soya (Tudisco et al., 2006). How the soya was sourced and confirmed (as GM or GE free) was not reported. Animals on the GM soya diet had significantly higher levels of lactate dehydrogenase (LDH), alanine aminotransferase (ALT) and gamma glutamyltransferase (GGT) in kidneys than animals on a conventional soya diet. LDH was also significantly elevated in heart muscle (Tudisco et al., 2006).

Gendora, Ltd.

**Summary**

Inghams Enterprises (Pty) Ltd. does use GM feed at some frequency or proportion of total feed. It writes that this practice is consistent with its claims of using no GM ingredients because "[r]esearch confirms that animals that consume feed with a component of GM are no different compared to animals that have been fed a low GM or GM free diet." However, whether the animals are the same or different in terms of their performance or safety as a result of using a particular ingredient in their preparation is not what is at issue. The issue is whether the use of GM feed is introducing an ingredient of GM into their product.

The references Inghams Enterprises uses to support its position that chickens exposed to GM feed are the same as chickens raised on conventional feed are uniformly very old and either do not address this issue or in my view do not explicitly support Inghams' claim. The age and suitability of the reference list used to support its GM policy is not consistent with its further claim that:

> "Inghams understands that there is considerable community interest in the uses of genetic modification and we believe it is important to keep customers informed or our policies and relevant facts"
> (http://www.inghams.co.nz/consumernz/aboutus.aspx?docId=285).

Table 1: Animal evidence of significant positive detections.

| Animal<br>Parameter detected | Pig | Cow | Fish | Chickens | Rabbits | Rats and mice | Sheep |
|---|---|---|---|---|---|---|---|
| GM DNA | | | | | | | |
| GM protein | | | | | | | |
| GM-induced metabolites | | | | | | | |
| GM-induced physiological changes | | | | | | | |
| GM-induced immunological responses | | | | | | | |

This report is enriched for positive detections of the parameters I was asked to investigate. There is a moderately larger pool of published studies that report no effect of GM feed on animals (e.g. Alexander et al., 2007, Flachowsky et al., 2007, Pryme and Lembcke, 2003). It should be emphasised, however, that the number of research studies that report no detection of physiological, immunological or metabolic effects, or absence of DNA or protein, is about the same as the number that report detection (e.g. Table 27 Flachowsky et al., 2007). In the relatively small literature which measures these particular parameters, there is a large proportion that reports significantly different effects of GM and conventional feed on animals or the presence in animals of DNA and protein unique to GM plants.

For the purposes of this report it is not assumed that the DNA sequence that was used to modify the GM plant must be identical in size to the DNA subsequently found in animals, or that any reduction in size of that DNA or its gene product(s) in the animal will make that animal "GM-free". If the recombinant DNA material in the original GM plant were 5000 nucleotide pairs in length and an unambiguous identification of it could be made from a partially digested or degraded fragment now of a few hundred nucleotide pairs in length, the material in which this detection is made is not GM-free any more than would be a plant made into a product of modern biotechnology by the insertion of DNA that was only a few hundred nucleotide pairs in size.

The majority of papers measuring the effects of GM feed measure endpoints, such as animal weight, mortality, performance, egg size and weight and animal rate of growth (Flachowsky et al., 2007) that are not relevant for reasons mentioned earlier. Furthermore, animals fed conventional or GM feed may achieve the same endpoints and still have individual and significant differences between them. In addition, many of these studies do not use whole food in their testing, but instead the protein unique to the GM plant expressed from a surrogate, usually the bacterium *Escherichia coli* (Pryme and Lembcke, 2003). Tests using surrogate sources of protein may not be appropriate because commercial animal feed is supplied as a whole food.

To attempt to argue whether animals exposed to GM plants through feed products are different from animals only exposed to conventional feed, using a simple tally of the number of researchers who detect or do not detect differences would be a mistake. The inconsistency of detection as catalogued in literature reports is an indication that there is uncertainty in what parameters to measure, what feeding regimes are most informative (Pryme and Lembcke, 2003) and what techniques are best suited. The small number of researchers in this field is spread over many different animals, varieties and species of GM plants and parameters to measure, and thus differences in practitioners' technical expertise or knowledge of the biology, molecular biology, biochemistry and physiology involved will be an important contributor to negative results.

The cumulative strength of the positive detections reviewed above leave me no reasonable uncertainty that GM plant material can transfer to animals exposed to GM feed in their diets or environment, and that there can be a residual difference in animals or animal-products as a result of exposure to GM feed (Table 1).

Gendora, Ltd.

**References**


Agodi, A., Barchitta, M., Grillo, A. and Sciacca, S. (2006). Detection of genetically modified DNA sequences in milk from the Italian market. Int. J. Hyg. Environ.-Health *209*, 81-88.

Alexander, T. W., Reuter, T., Aulrich, K., Sharma, R., Okine, E. K., Dixon, W. T. and McAllister, T. A. (2007). A review of the detection and fate of novel plant molecules derived from biotechnology in livestock production. Animal Feed Sci. Technol. *133*, 31-62.

Asensio, L., González, I., García, T. and Martín, R. (2008). Determination of food authenticity by enzyme-linked immunosorbent assay (ELISA). Food Control *19*, 1-8.

Beever, D. E. and Phipps, R. H. (2001). The fate of plant DNA and novel proteins in feeds for farm livestock: A United Kingdom perspective. J. Anim Sci. *79*, E290-295.

Bernard, J. C., Pan, X. and Sirolli, R. (2005). Consumer attitudes toward genetic modification and other possible production attributes for chicken. J. Food Distribution Res. *36*, 1-11.

Biosafety Assessment Tool. https://bat.genok.org/bat/. Date of Access: 15 July 2009.

Chainark, P., Satoh, S., Hirono, I., Aoki, T. and Endo, M. (2008). Availability of genetically modified feed ingredient: investigations of ingested foreign DNA in rainbow trout Oncorhynchus mykiss. *74*, 380-390.

Chowdhury, E. H., Kuribara, H., Hino, A., Sultana, P., Mikami, O., Shimada, N., Guruge, K. S., Saito, M. and Nakajima, Y. (2003). Detection of corn intrinsic and recombinant DNA fragments and Cry1Ab protein in the gastrointestinal contents of pigs fed genetically modified corn Bt11. J. Anim. Sci. *81*, 2546-2551.

Duggan, P. S., Chambers, P. A., Heritage, J. and Forbes, J. M. (2003). Fate of genetically modified maize DNA in the oral cavity and rumen of sheep. Br. J. Nutr. *89*, 159-166.

EFSA (2007). EFSA statement on the fate of recombinant DNA or proteins in meat, milk and eggs from animals fed with GM feed. European Food Safety Authority. http://www.efsa.europa.eu.

Einspanier, R., Lutz, B., Rief, S., Berezina, O., Zverlov, V., Schwartz, W. and Mayer, J. (2004). Tracing residual recombinant feed molecules during digestion and rumen bacterial diversity in cattle fed transgene maize. Eur Food Res Technol *218*, 269-273.

EU Commission. http://ec.europa.eu/agriculture/publi/gmo/fullrep/ch4.htm. Date of Access: 21 July 2009.

Gendora, Ltd.

Ewen, S. W. B. and Pusztai, A. (1999). Effect of diets containing genetically modified potatoes expressing Galanthus nivalis lectin on rat small intestine. Lancet *354*, 1353-1354.

Flachowsky, G., Aulrich, K., Böhme, H. and Halle, I. (2007). Studies on feeds from genetically modified plants (GMP) - Contributions to nutritional and safety assessment. Anim. Feed Sci. Technol. *133*, 2-30.

Frewer, L. (2003). 10. Societal issues and public attitudes towards genetically modified foods. Trends Food Sci. Technol. *14*, 319-332.

Gallagher, L. (2007). Statistical and toxicological evaluation of two analyses on 90-day rat feeding study for MON863 transgenic corn. Environmental Science and Research, Ltd. www.nzfsa.govt.nz/consumers/gm-ge/r-gm-maize1.pdf . .

GMO Free Regions. http://www.gmo-free-regions.org/gmo-free-regions/gmo-free-retailers.html. Date of Access: 21 July 2009.

Guertler, P., Paul, V., Albrecht, C. and Meyer, H. (2009). Sensitive and highly specific quantitative real-time PCR and ELISA for recording a potential transfer of novel DNA and Cry1Ab protein from feed into bovine milk. Anal. Bioanal. Chem. *393*, 1629-1638.

Heinemann, J. A. (2009). Hope not Hype. The future of agriculture guided by the International Assessment of Agricultural Knowledge, Science and Technology for Development (Penang, Third World Network).

Heinemann, J. A., Sparrow, A. D. and Traavik, T. (2004). Is confidence in monitoring of GE foods justified? Trends Biotechnol. *22*, 331-336.

IAASTD, ed. (2009). International Assessment of Agricultural Knowledge, Science and Technology for Development (Washington, D.C., Island Press).

Kain, M. R. (2007). Throw another cloned steak on the barbie: examining the FDA's lack of authority to impose mandatory labeling requirements for cloned beef. N.C.J.L. Tech. *8*, 303-348.

Klotz, A., Mayer, J. and Einspanier, R. (2002). Degradation and possible carry over of feed DNA monitored in pigs and poultry. Eur Food Res Technol *214*, 271-275.

Kroghsbo, S., Madsen, C., Poulsen, M., Schroder, M., Kvist, P. H., Taylor, M., Gatehouse, A., Shu, Q. and Knudsen, I. (2008). Immunotoxicological studies of genetically modified rice expressing PHA-E lectin or Bt toxin in Wistar rats. Toxicol. *245*, 24-34.

Gendora, Ltd.

Lutz, B., Wiedemann, S., Einspanier, R., Mayer, J. and Albrecht, C. (2005). Degradation of Cry1Ab protein from genetically modified maize in the bovine gastrointestinal tract. J. Agric. Food Chem. *53*, 1453-1456.

Magaña-Gómez, J. A., López Cervantes, G., Yepiz-Plascencia, G. and Calderón de la Barca, A. M. (2008). Pancreatic response of rats fed genetically modified soybean. J Appl Toxicol *28*, 217-226.

Malatesta, M., Biggiogera, M., Manuali, E., Rocchi, M. B. L., Baldelli, B. and Gazzanelli, G. (2003). Fine structure analyses of pancreatic acinar cell nuclei from mice fed on genetically modified soybean. Eur J Histochem. *47*, 385-388.

Malatesta, M., Caporaloni, C., Gavaudan, S., Rocchi, M. B. L., Serafini, S., Tiberi, C. and Gazzanelli, G. (2002). Ultrastructural morphometrical and immunocytochemical analyses of hepatocyte nuclei from mice fed on genetically modified soybean. Cell Struct. Funct. *27*, 173-180.

Malatesta, M., Tiberi, C., Baldelli, B., Battistelli, S., Manuali, E. and Biggiogera, M. (2005). Reversibility of hepatocyte nuclear modifications in mice fed on genetically modified soybean. Eur J Histochem. *49*, 237-242.

Mazza, R., Soave, M., Morlacchini, M., Piva, G. and Marocco, A. (2005). Assessing the transfer of genetically modified DNA from feed to animal tissues. Transgenic Res. *14*, 775-784.

Nielsen, C. R., Berdal, K. G., Bakke-McKellep, A. M. and Holst-Jensen, A. (2005). Dietary DNA in blood and organs of Atlantic salmon (Salmo salar L.). Eur Food Res Technol *221*, 1-8.

Novoselova, T. A., Meuwissen, M. P. M. and Huirne, R. B. M. (2007). Adoption of GM technology in livestock production chains: an integrating framework. Trends Food Sci Technol *18*, 175-188.

Onyango, B., Nayga Jr, R. M. and Schilling, B. (2004). Role of product benefits and potential risks in consumer acceptance of genetically modified foods. AgBioForum *7*, 202-211.

Phipps, R. H., Beever, D. E. and Humphries, D. J. (2002). Detection of transgenic DNA in milk from cows receiving herbicide tolerant (CP4EPSPS) soyabean meal. Livestock Prod. Sci. *74*, 269-273.

Phipps, R. H., Deaville, E. R. and Maddison, B. C. (2003). Detection of Transgenic and Endogenous Plant DNA in Rumen Fluid, Duodenal Digesta, Milk, Blood, and Feces of Lactating Dairy Cows. J. Dairy Sci. *86*, 4070-4078.

Gendora, Ltd.

Pryme, I. F. and Lembcke, R. (2003). In vivo studies on possible health consequences of genetically modified food and feed - with paticular regard to ingredients consisting of genetically modified plant materials. Nut. Health *17*, 1-8.

Raska, I. (2003). Oldies but goldies: searching for Christmas trees within the nucleolar architecture. Trends Cell Biol. *13*, 517-525.

Sagstad, A., Sanden, M., Haugland, Ø., Hansen, A.-C., Olsvik, P. A. and Hemre, G.-I. (2007). Evaluation of stress- and immune-response biomarkers in Atlantic salmon, *Salmo salar* L., fed different levels of genetically modified maize (Bt maize), compared with its near-isogenic parental line and a commercial suprex maize. J. Fish Dis. *30*, 201-212.

Sagstad, A., Sanden, M., Krogdahl, Å., Bakke-McKellep, A. M., Froystad, M. and Hemre, G.-I. (2008). Organs development, gene expression and health of Atlantic salmon (*Salmo salar* L.) fed genetically modified soybeans compared to the near-isogenic non-modified parental line. Aquaculture Nutr. *14*, 556-572.

Sanden, M., Berntssen, M. H. G. and Hemre, G. I. (2007). Intracellular localization of dietary and naked DNA in intestinal tissue of Atlantic salmon, Salmo salar L. using in situ hybridization. Eur Food Res Technol *225*, 533-543.

Scheideler, S. E., Hileman, R. E., Weber, T., Robeson, L. and Hartnell, G. F. (2008). The in vivo digestive fate of the Cry3Bb1 protein in laying hens fed diets containing MON 863 corn. Poult Sci *87*, 1089-1097.

Seralini, G.-E., Cellier, D. and Spiroux de Vendomois, J. (2007). New analysis of a rat feeding study with a genetically modified maize reveals signs of hepatorenal toxicity. Arch. Environ. Contam. Toxicol. *52*, 596-602.

Seralini, G.-E., Spiroux de Vendomois, J., Cellier, D., Sultan, C., Buiatti, M., Gallagher, L., Antoniou, M. and Dronamraju, K. R. (2009). How subchronic and chronic health effects can be neglected for GMOs, pesticides or chemicals. Internat. J. Biol. Sci. *5*, 438-443.

Sissener, N. H., Sanden, M., Bakke, A. M., Krogdahl, Å. and Hemre, G. I. (2009). A long term trial with Atlantic salmon (Salmo salar L.) fed genetically modified soy; focusing general health and performance before, during and after the parr-smolt transformation. *294*, 108-117.

Taylor, M., Hyun, Y., Hartnell, G. F., Nemeth, M. A., Karunanandaa, K., George, B., Glenn, K. C. and Heydens, W. F. (2003). MSL- 18883 Sponsor summary report for the study 02-01-72-16 (comparison of broiler performance when fed diets containing LY038, LY038 x MON 810, negative segregant control, or commercial maize). Monsanto Company. http://dx.doi.org/10.1111/j.1444-2906.2008.01535.x.

Gendora, Ltd.

Tony, M. A., Butschke, A., Broll, H., Grohmann, L., Zagon, J., Halle, I., DÃ¤nicke, S., Schauzu, M., Hafez, H. M. and Flachowsky, G. (2003). Safety assessment of Bt 176 maize in broiler nutrition: degradation of maize-DNA and its metabolic fate. Arch. Animal Nutr. *57*, 235 - 252.

Trabalza-Marinucci, M., Brandi, G., Rondini, C., Avellini, L., Giammarini, C., Costarelli, S., Acuti, G., Orlandi, C., Filippini, G., Chiaradia, E., *et al.* (2008). A three-year longitudinal study on the effects of a diet containing genetically modified Bt176 maize on the health status and performance of sheep. Livestock Sci. *113*, 178-190.

TraceConsult. http://www.traceconsult.com/index.php?option=com_content&view=article&id=125:swedens-lantmaennen-embarrassed-by-39--gmo-content-in-maize-for-dairy-cows&catid=47:newsticker&Itemid=50&lang=en. Date of Access: 21 July 2009.

Tudisco, R., Lombardi, P., Bovera, F., d'Angelo, D., Cutrignelli, M. I., Mastellone, V., Terzi, V., Avallone, L. and Infascelli, F. (2006). Genetically modified soya bean in rabbit feeding: detection of DNA fragments and evaluation of metabolic effects by enzymatic analysis. Anim. Sci. *82*, 193-199.

UK FSA. http://www.food.gov.uk/gmfoods/gm/gm_labelling. Date of Access: 21 July 2009.

Vecchio, L., Cisterna, B., Malatesta, M., Martin, T. E. and Biggiogera, M. (2004). Ultrastructural analysis of testes from mice fed on genetically modified soybean. Eur J Histochem. *48*, 449-454.

Wilcks, A., van Hoek, A. H. A. M., Joosten, R. G., Jacobsen, B. B. L. and Aarts, H. J. M. (2004). Persistence of DNA studied in different ex vivo and in vivo rat models simulating the human gut situation. Food Chem Toxicol. *42*, 493-502.

Yonemochi, C., Fujisaki, H., Harada, C., Kusama, T. and Hanazumi, M. (2002). Evaluation of transgenic event CBH 351 (StarLink) corn in broiler chicks. Animal Sci. J. *73*, 221-228.

Appendix One: Complete CV

**CURRICULUM VITAE**

**JACK A. HEINEMANN**

| | |
|---|---|
| CURRENT POSITIONS: | Professor<br>Adjunct Professor (GENØK – Centre for Biosafety) |
| ADDRESS: | School of Biological Sciences (formerly PAMS)<br>University of Canterbury, Christchurch, New Zealand |
| EMAIL ADDRESS: | jack.heinemann@canterbury.ac.nz |
| TELEPHONE/FAX: | 64 03 364-2926/2500 |
| CITIZENSHIP: | U.S.A. and New Zealand |

EDUCATION:

| | |
|---|---|
| 1985-1989 | Ph.D. in Biology/Molecular Biology<br>University of Oregon, Eugene, OR, USA |
| 1980-1985 | B.Sc(Honours) in Biochemistry<br>B.Sc(Honours) in Molecular Biology<br>University of Wisconsin, Madison, WI, USA |

PROFESSIONAL EXPERIENCE:

| | |
|---|---|
| 2007-present | Professor, School of Biological Sciences, University of Canterbury |
| 2003-2007 | Associate Professor |
| 1994-2002 | Senior Lecturer |
| 2001-present | Director, Centre for Integrated Research in Biosafety, University of Canterbury<br>Adjunct Professor, Norwegian Institute of Gene Ecology (GENØK), Tromsø, Norway<br>Member, Biomathematics Research Centre (2001) University of Canterbury |
| 1997-2000 | Biochemistry Programme Coordinator<br>(managed 5 undergraduate courses, ~ 20 postgraduate (PhD and MSc) students and 10 academic and technical staff) |
| 1992-1994 | Staff Fellow, National Institutes of Health, NIAID,<br>Laboratory of Microbial Structure and Function |

Gendora, Ltd.

| | |
|---|---|
| 1989-1992 | Intramural Research Training Award Fellow<br>NIAID, NIH, Laboratory of Microbial Structure and Function |
| 1985-1989 | Graduate student, University of Oregon, Institute of Molecular Biology |
| 1983-1984 | Undergraduate Research Assistant, University of Wisconsin-Madison, Department of Biochemistry |

INTERESTS AND EXPERTISE:

Genetics and molecular biology of prokaryotic and eukaryotic microorganisms; horizontal gene transfer, particularly conjugation; effects of stress, particularly induced by antibiotics; evolution and biosafety risk assessment; eugenics (historical); influence of language on science.

HONORS AND SPECIAL RECOGNITION:

| | |
|---|---|
| 2009 | Chosen by the (United Nations) Convention on Biological Diversity Secretariat to serve on the Ad Hoc Technical Expert Group (AHTEG) on Risk Assessment and Risk Management |
| 2008 | Chosen by the (World Bank and UN agencies) IAASTD Secretariat as author representative to the intergovernmental meeting on the IAASTD Report |
| 2007 | Selected by the IAASTD Advisory Bureau to serve as an author on the Biotechnology theme of the Synthesis Report |
| 2006 | Appointed Lead Author in the IAASTD Global Assessment Report (nominated by Norway) |
| 2005 | UN Roster of Experts (Biosafety Protocol) |
| | Distinguished Lecture in Microbiology, University of Wisconsin-Madison |
| 2004 | Speaker in the New Zealand Royal Society's Science for Parliament Series |
| 2002 | Recipient, New Zealand Association of Scientists Research Medal (The Association's Research Medal is awarded each year to a single scientist aged under 40 for outstanding research work, principally undertaken in New Zealand during the three preceding years.) |

Gendora, Ltd.

| | |
|---|---|
| 2002-2004 | Editorial Board of Targets (Elsevier "Trends" series journal) |
| 2001 | Visiting Professor, Norwegian Institute for Gene Ecology and the University of Tromsø (with Prof. T. Traavik), Tromsø, Norway |
| | Visiting Scholar, The Rockefeller University (with Prof. J. Lederberg), New York, USA |
| 1999-2004 | Editorial Board of Drug Discovery Today |
| 1993 | Young Investigator Award from the American Society for Microbiology Interscience Conference on Antimicrobial Agents and Chemotherapy (ICAAC) [one of four awarded in an international competition] |
| 1989-1992 | Intramural Research Training Award (National Institutes of Health) |

1990-2003 Various recognition: *National Business Review* Achiever of the Week (14 Feb. 2003); featured in Saunders, J. 2003. Multiple Drug Resistant Bacteria. Microbiology Today (http://www.socgenmicrobiol.org.uk/pubs/micro_today/book_reviews/MTNOV03/MTN03_24.cfm) ; featured in: Delwiche, C.F. 2000. Griffins and Chimeras: Evolution and Horizontal Gene Transfer. BioScience 50, 85-87; featured in: Ankenbauer, R.G. 1997. Reassessing Forty Years of Genetic Doctrine: Retrotransfer and Conjugation. Genetics 145, 543-549; keynote addresses, The Norwegian Biotechnology Advisory Board Meeting (Oslo, Norway, 1997) and International Conference on Gene Transfer Mediated by Bacterial Plasmids (Banff, Alberta, Canada, 1990); invited speaker, "Microbial Stress Response" Gordon Conference, 1994.
1980-1989 Undergraduate and graduate school awards include: 1984, **Outstanding Senior** (final year) **Student Award** (University of Wisconsin-Madison Alumni Association); 1983, **Mary Shine Peterson Award** (Department of Biochemistry, University of Wisconsin); University of Wisconsin Forensics Team Scholarship; 1981, Phi Eta Sigma, the Freshman's Honor Society, MACE, the Chancellor's Men's Honor Society; 1986-1986 NIH Molecular Biology Predoctoral Traineeship (University of Oregon).

GRANTS: Total value since 1995 ~NZ $3.1 million

| | |
|---|---|
| 2009-2013 | GE Biosafety Forecast Service (NZ $492,000) |
| 2008 | GE Biosafety Forecast Service (NZ $123,000) |
| 2006-07 | Constructive Conversations (subcontract FRST) (NZ$35,000) |
| 2005-07 | GE Biosafety Forecast Service (NZ $767,000)<br>University of Canterbury (NZ $30,000) |
| | United Nations Food and Agriculture Organisation (FAO) report on Gene Flow (NZ $50,000) |

Gendora, Ltd.

| | |
|---|---|
| | Erskine Fund Teaching Fellowship (NZ $20,000) |
| 2004 | GE Biosafety Forecast Service (NZ $324,000) |
| 2003 | GE Biosafety Forecast Service (NZ $31,000) |
| 2002 | FRST: Postdoctoral fellowship (to RJ Weld to work in my laboratory for 3 years) |
| | OECD Fellowship (~NZ $40,000 for RJ Weld to work in Norway for 6 months) |
| | Brian Mason Trust: NZ $15,000 for research on GMOs |
| 2001 | Miscellaneous: GENØK (US $10,000); Rockefeller University (US $6,000); University of Canterbury (US $3,000); US-New Zealand ISAT Bi-lateral Relations Grant ($3,200) |
| 2000 | Marsden Fund (Associate Investigator) (NZ $447,000)<br>Ministry of Health (NZ $3,000) |
| 1999 | Marsden Fund (Primary Investigator) (NZ $528,000)<br>Joint U. Canterbury/Crop & Food Res. (NZ $171,000)<br>Ministry of Health (NZ $8,000) |

1995-1998 (1998) Lotteries Health Research Grant (NZ $71,350), University of Canterbury Research Award (NZ $45,000); (1997) Christchurch School of Medicine Summer Studentship Award (to sponsor an undergraduate researcher), Don Beaven Trust Travelling Fellowship (NZ $3,000), University of Canterbury Research Award ($20,000); (1996) Lotteries Science Research Grant (NZ $35,000), (1995) University of Canterbury Research Award (NZ $25,000), University of Canterbury Equipment Award (NZ $90,000)

CONSULTATIONS, SYMPOSIA and PROFESSIONAL ACTIVITIES:

Spoken at **about 25 international conferences** (~80% at invitation), presented **4 keynote addresses** and **chaired 6 sessions**. Served on the organising committees of 5 international meetings. *Referee* on occasion for **Applied and Environmental Microbiology, Bioessays, Biology Letters Review, Drug Discovery Today, FEMS Microbiology, FEMS Microbiology Ecology, Food and Chemical Toxicology, Environmental Biosafety Research, Environmental Science and Technology, Journal of Applied Microbiology, Journal of Bacteriology, Microbiology, Molecular Biology and Evolution, Molecular Ecology, Molecular Microbiology, Nature Biotechnology, Nature Genetics, New Zealand Journal of Zoology, Pharmacological Research, Plasmid, Science** and **World Journal of Microbiology and Biotechnology,** and eight granting agencies (NSF, USA; FRST, Marsden, HRC and Lotteries

Grants Board, New Zealand; MacQuarie, Australia; NERC and Wellcome Trust, UK, Alzheimer's Foundation, Danish National Research Foundation, Denmark, Slovak Research and Development Agency, Slovak Republic). Chief organiser of the 1999 International Osmoregulation Conference, Christchurch, New Zealand. ***Organiser and Instructor*** of two prominent international courses: School of Bioinformatics and Genomics Summer Course in Phylogenomics (2003, Sweden) and International Biosafety Course (2003-continuing, Norway).

Since 1989 I have been an invited speaker at over 50 academic, governmental or industrial institutions in 10 different countries. Recent/upcoming talks:

CPIT Institute of Polytechnic, Christchurch
Dartmouth University, USA
Iberamerican University, Dominican Republic
Göteborg University, Sweden
University of Wisconsin-Madison, USA

2008 — Expert witness to Tasmanian Joint Select Committee on Gene Technology in Primary Industries (nominated by Hon David Llewellyn, Chair)

Invited Keynote to Feed the World Conference, London

2006 — Invited speaker, International Biosafety Symposium Meeting of the Parties (MOP3) of the Cartegena Protocol on Biosafety, Curitiba, Brazil

Expert reviewer, Denmark Centre of Excellence Programme.

2005 — Expert reviewer on New Zealand Environmental Risk Management Authority's policy paper: Horizontal Gene Transfer
Keynote Speaker, UNEP/GEF National Biosafety Framework Initiative, Dominican Republic

2004 — Invited speaker, International Biosafety Symposium Meeting of the Parties (MOP1) of the Cartegena Protocol on Biosafety, Kuala Lumpur, Malaysia
Invited speaker, School of Bioinformatics and Genomics Summer Course in Phylogenomics, Göteborg University, Sweden

2004-2005 — Executive Committee, United Nations Environment Programme and GENØK Biosafety Capacity Building Partnership

Gendora, Ltd.

| | |
|---|---|
| 2003 | Scientific consultant to the New Zealand Parliamentary Local Government and Environment Select Committee on "Corngate".<br>Invited Speaker, American Society for Microbiology ICAAC conference. |
| 2002 | Speaker: ERMANZ conference on Horizontal Gene Transfer<br>Microbial Genetics Conference, Bergen, Norway<br>New Zealand Microbiology Society Meeting |
| 2001 | Advisor to New Zealand Minister of Science in the "Horizontal Gene Transfer Round Table Meeting" |
| 2000 | Expert panel New Zealand Ministry of Health<br>New Zealand PGSF Biotechnology Tender Panel<br><br>University of Canterbury Representative to the NZ Royal Commission on Genetic Engineering |
| 1999 | Expert Panel on Antibiotic Residues for the New Zealand Ministry of Health |
| 1997 | Keynote speaker, The Norwegian Biotechnology Advisory Board Meeting, Oslo, Norway |
| 1993 | Advisor to the United States Department of Energy, under the auspices of the American Academy of Microbiology, for genetic modification of bacteria |

POSTGRADUATE TEACHING (1995-present)

*Experience:* Primary supervisor of 13 completed MSc theses, 12 BSc (Hons) theses and 7 PhD theses, and associate or co-supervisor for more than 20 BSc (Hons), MSc and PhD students since joining the University of Canterbury (1994). My research laboratory presently has 2 PhD students and 1 postdoctoral scholar.

*Achievements:* My research students received 5 of the 6 poster awards in the 1996 Queenstown International Molecular Biology Meeting attended by researchers from all over the world and uniformly represented by New Zealand and Australian universities. Joanne Kingsbury and Tim Cooper, while PhD students in my laboratory, won the first and second prizes, respectively, for best research talks at the 1998 national meeting of the Microbiology and Biochemical Societies of New Zealand. Tim was a postdoctoral scholar at Michigan State

University and is now at Auckland University. Joanne is a postdoctoral scholar at Duke University. Tim was subsequently nominated for the American Society of Microbiology Sternberg Thesis Award. Gayle Ferguson, another of my PhD students, won first prize for her talk at the Microbiology Society national meeting in 2001 and was a postdoctoral scholar at Columbia University, New York.

EXTERNAL TEACHING ACTIVITIES:

| | |
|---|---|
| 2009 | Faculty and Coordinator for the Gateways Partners Symposia Course and Conference on (trans)gene Flow, Tromsø, Norway |
| 2005 | Faculty and organiser of the Solomon Islands Biosafety Course |
| 2003-2005 | Faculty and instructor International Biosafety Course |
| 2003-4 | Principal Organiser and Instructor (2003), Göteborg University's Bioinformatics summer graduate course, Sweden |
| 2000-present | PhD examiner: 3 x University of Otago; 1x Massey; 2 x Lincoln; 1 x Macquarie University; 1 x Dartmouth University<br>MSc. examiner: 1 x Massey University, 3 x Otago University; 1 x Macquarie University<br>Assessor (MSc proposals): 3 x Auckland University |

Teaching experience during NIH (1990-1994), under- and post-graduate years (1980-1989): 1990-1994 Supervisor, NIH Summer Student Program, Rocky Mountain Laboratories, USA (resulting in a research paper in the journal **Genetics** by an undergraduate student in 1996); 1992-2000, University of Montana USA affiliate faculty; Guest lecturer, University of Montana, 1992-1994 "Advanced Topics in Microbiology", (course 595) University of Montana, Department of Biology; Teaching Assistant for Core Biology Lecture and Laboratory, Department of Biology, University of Oregon, Eugene, OR, USA; Presenter, Special Project Course in Bioethics, Department of Botany, University of Wisconsin, Madison, WI, USA.

STAFF LEADERSHIP ROLES:

Serving the University of Canterbury on 12 *ad hoc* committees in addition to standing committees (listed below): chair of the College of Science Biosecurity Programme Committee (2004); Science Faculty Working Committee evaluating proposals for establishing a Department of Biochemistry (1995-6); the AUS Workloads Committee (1996); lead workshops at the Canterbury-hosted Education Forum (1999); and served on the AAC Subcommittee on Appeals Procedures (2000). Since 1995, I have served on 3 and chaired 4 Search

Gendora, Ltd.

Committees (total of 7) for new academics. Participating in the staff mentorship and buddy programme.

| | |
|---|---|
| 2009 | UC Academic Audit Working Group on the role of critic and conscience of society |
| 2007-2008 | President, Association of University Staff (AUS) Canterbury Branch |
| 2006-2007 | Canterbury representative AUS National Council |
| 2006 | AUS National Bargaining Team |
| 2005-2006 | Academic Representative (elected) on the Canterbury Branch AUS |
| 2005-2006 | School of Biological Sciences Research Committee |
| 2002-continuing | Chair, University Institutional Biosafety Committee |
| 2001-2003 | Departmental Supervisor of Postgraduate Studies |
| 2002 | University Teaching and Learning Committee |
| 2000-2001 | Department HSNO-Biology Officer and University representative to the HSNO Consultative Group |
| 2002-2004 | Department Safety Committee |
| 1996-2005 | Chair (2000), University Joint Academic Student Grievance Committee |
| 1998-2001 | Plant and Microbial Sciences Workload Committee |
| 1996-1998 | Branch Committee of the Association of University Staff (AUS) |
| 1994-1998 | Plant and Microbial Sciences Curriculum Committee |
| 1994-1998 | Academic Supervisor of the Graduate Seminar Series |

PROFESSIONAL ORGANIZATIONS:

| | |
|---|---|
| 1989-continuing | American Society for Microbiology |
| 1994-continuing | New Zealand Microbiology Society |
| 1995-2002 | New Zealand Molecular Biology Society |
| 1998-2002 | New Zealand Society for Biochemistry and Molecular Biology |

Gendora, Ltd.

2002-2004 New Zealand Association of Scientists

SCIENCE and COMMUNITY:
2008: Call for Government to invest more in agricultural research, Radio New Zealand, 16.4.08. Arts to get the chop, The Dominion Post, 30.4.08. Executions and amputations as staff protest job cuts, Westport News, 29.4.08. Restructuring goes ahead, Westport News, 30.4.08. Plans for restructuring go ahead, Gisborne Herald, 30.4.08. Claims that GM foods are needed to avert a food crisis are rubbished, Radio New Zealand, 9.6.08; Claims that GM crops are needed to prevent food shortages are disputed by experts, Radio New Zealand, 9.6.08.

2007: GM Corn, 30 minute interview on RNZ Nine to Noon programme 19.7.07; Discussion as to whether new type of genetically modified corn safe for human consumption, RNZ (Morning Report), 7.2.07; Food safety minister asked to reject new type of genetically modified corn, RNZ (6.00am news), 7.2.07; Minister asked to reject GM animal feed, New Zealand Herald, 7.2.07; Lobby tries to halt feed imports, Marlborough Express, 7.2.07; GM maize fears raised, Bay of Plenty Times, 7.2.07; Food lobbyists: Govt must act fast to stop GE corn, Northern Advocate, 8.2.07; Academic research under pressure, Gulf News, 15.2.07; Review of approval of genetically modified corn for animal feed, RNZ (Checkpoint), 21.2.07

2006: The Press (Christchurch) "Gene claims a rationale for abuse" (15 August, p. A8); ABC Science Online "Food Regulator Criticised over new GM corn" (4 August); Interview National Radio's Morning Report (6 June on High Lysine Corn); Interview National Radio's Checkpoint (5 June on Corn Food Safety); The Press (Christchurch) Heinemann, J.A. 5 May 2006 Perspectives article "Alarm bells over GM food approval: part 2. Featured in New Zealand Herald 24.03.06 Company wants stockfeed GE corn approved for people; TVNZ and TV3 interview on Frank Sin's "gay gene", 6 and 10 pm news 13.03.06; Christchurch Press interview on Frank Sin's "gay gene".

2005: Heinemann, J.A., Bungard, R. and Goven, J. Confidence in biotechnology requires greater commitment. 2005.3.3. Otago Daily Times p. 11.

2004: Featured on Checkpoint (National RadioNZ, 25.05.04); Speaking engagements: March Presentation to the WEA; April Palmerston North branch of the Royal Society; Royal Society Parliament Series; July lecturer in National Science Teachers Conference; September Skeptics Society Annual Conference; Presenter in Natural History New Zealand pilot for Discovery "Dr. Know" series.

2003: Heinemann, J.A. 9 May 2003. Economics of GE models fail to convince. **National Business Review** p. 21. Presentation to University of the Third Age. Heinemann, J.A. 25 August 2003. Food chain in NZ must be protected. **New Zealand Herald** p. A15.

2000-2: Heinemann, J.A. 2002. GE or not to be. **NZ Listener** *185*, 8. Interview (April 2002), Morning Programme National Radio "Canterbury research wins international

accolades"; and CTV (same topic). Invited speaker for the New Zealand Association for Impact Assessment (May 2002). Instructor "Marvels and Menaces of Microscopic Life" University of Canterbury Continuing Education Course; "Radioactive" Wellington Student Radio interview on antibiotic resistance; Talk on horizontal gene transfer to Canterbury Botanical Society; Featured in news article by Pockley, P. 2000. New law threatens to undermine genetics in New Zealand. **Nature** *406*, 8; Letter to the Editor of the Christchurch **Press**: "Genetic Engineering"; Interviewed by Paul Holmes (Auckland radio) for NewstalkZB (27 June); Radio New Zealand News interviews (30 June and 20 July); Featured in 4 news articles by the Christchurch **Press** on genetic engineering regulations; Heinemann, J.A. June 2000. Open letter to Helen Clark. **The Best Underground Press – Critical Review** (6), *9*, 2; University of Canterbury student newspaper **CANTA** articles: "Why do students but not academics have to be world-class?" (10 May 2000) and "Teaching is as teachers do" (17 May 2000); Heinemann, J.A. 2000. Research hazards. **New Zealand Education Review** (Sept. 8, 2000, p. 9); Heinemann, J.A. 2000. National security risk. NZ Listener (Jul 7), 7-8; interview on horizontal gene transfer by CHTV (1 Nov.); interview National Programme **Eureka!** (Nov. 26-27, 2000); Heinemann, J.A. 2001. The fate of students within our hands. **New Zealand Education Review** (Jan. 12, 2001, p. 7).

Presentations to Lions, Rotary (x2), WEA, University of the Third Age.

1999: Talk on Genetically Modified Food to the Canterbury WEA; Talk on Genetically Modified Food to the Probus Club; Article to University of Canterbury public relations magazine, **Canterbury Research**, entitled: Are all Genes made of DNA?

1998: Talk on Genetically Modified Food to the WEA Bishopdale Community Centre; Article to community magazine, **City Habitat**, entitled "What is a University?"; Article to community magazine, **City Habitat**, entitled "Why You Don't Want to be my Client".

1997: Interview National Programme, New Zealand Public Radio: "Superbugs"; Article to University of Canterbury public relations magazine, **Canterbury Research**, entitled: "The Life and Times of the Undead"; Debate Plains FM, Christchurch, New Zealand: "Risk and Ethics of Genetic Engineering".

1995: Interview National Programme, New Zealand Public Radio: "Antibiotic Resistance"; Advisor for a nationally ranked high school student science project competition.

TOTAL PROFESSIONAL PUBLICATIONS: 81

Peer-Reviewed Publications (*invited): Total: 44

Journals (32)

Heinemann, J.A. and Kurenbach, B. 2008. Special threats to the agroecosystem from the combination of genetically modified crops and glyphosate. Third World Network Biosafety Briefing, August 2008.

Filutowicz, M., Burgess, R., Gameli, R.L., Heinemann, J.A., Kurenbach, B., Rakowski, S.A. and Shankar, R. 2008. Bacterial conjugation-based antimicrobial agents. **Plasmid** 60, 38-41.

Tsuei, A.C., Carey-Smith, G.V., Hudson, J.A., Billington, C. and Heinemann, J.A. 2007. Prevalence and numbers of coliphages and Campylobacter jejuni bacteriophages in New Zealand foods. **International Journal of Food Microbiology** 116, 121-125.

Silby, M.W., Ferguson, G.C., Billington, C. and Heinemann, J.A. 2007. Localization of the plasmid-encoded proteins TraI and MobA in eukaryotic cells. **Plasmid** 57, 118-130.

Willms, A.R., Roughan, P.D. and Heinemann, J.A. 2006. Static recipient cells as reservoirs of antibiotic resistance during antibiotic therapy. **Theoretical Population Biology** 70, 436-451.

Heinemann, J.A., Rosén, H., Savill, M., Burgos-Caraballo, S. and Toranzos, G.A. 2006. Environment Arrays: A possible approach for predicting changes in water-borne bacterial disease potential. **Environmental Science and Technology** 40, 7150-7156.

Carey-Smith, G., Billington, C., Cornelius, A.J., Hudson, A. and Heinemann, J.A. 2006. Isolation and characterization of bacteriophages infecting Salmonella spp. **FEMS Microbiology Letters** 258, 182-186.

Roy Chowdhury, P. and Heinemann, J.A. 2006. The General Secretory Pathway of *Burkholderia gladioli* pv. *agaricicola*, BG164R, is necessary for 'Cavity Disease' in white button mushrooms. **Applied and Environmental Microbiology** 72, 3558-3565.

Cooper, T.F. and Heinemann, J.A. 2005. Selection for plasmid postsegregational killing depends on multiple infection: Evidence for the selection of more virulent parasites through parasite-level competition. **Proceedings of the Royal Society London Biological Science Series B** *272*, 403-410.

Heinemann, J.A. and Traavik, T. 2004. Problems in monitoring horizontal gene transfer in field trials of transgenic plants. **Nature Biotechnology** *22*, 1105-1109.

*Heinemann, J.A., Sparrow, A.D. and Traavik, T. 2004. Is confidence in the monitoring of GE foods justified? **Trends in Biotechnology** *22*, 331-336. (Featured on AgBiotechNet www.agbiotechnet.com)

Bland, M. Ismail, S., Heinemann, J.A. and Keenan, J. 2004. The action of bismuth against *Helicobacter pylori* mimics but is not caused by intracellular iron deprivation. **Antimicrobial Agents and Chemotherapy** *48*, 1983–1988.

Weld, R.J., Butts, C. and Heinemann, J.A. 2004. Models of phage growth and their applicability to phage therapy. **Journal Theoretical Biology** *227*, 1-11.

Ferguson, G.C., Heinemann, J.A. and Kennedy, M.A. 2002. Gene transfer between *Salmonella enterica* serovar Typhimurium inside epithelial cells. **Journal of Bacteriology** *184*, 2235-2242. (This paper was selected by ASM as the best published in all ASM journals in April, 2002.)

Weld, R.J., Bicknell, R., Heinemann, J.A. and Eady, C. 2002. Ds transposition mediated by transient transposase expression in *Heiracium aurantiacum*. **Plant, Cell, Tissue & Organ Culture** *69*, 45-54.

Heinemann, J.A. Alternative medicines: a clash of culture or science? 2001. **NZ College Midwives Journal** *24*, 23-25.

Weld, R.J., Heinemann, J. and Eady, C. 2001. Transient GFP expression in *Nicotiana plumbaginifolia* suspension cells following co-cultivation with *Agrobacterium tumefaciens*: the role of gene silencing, cell death and T-DNA loss. **Plant Molecular Biology** *45*, 377-385.

Cooper, T.F. and Heinemann, J.A. 2000. Postsegregational killing does not increase plasmid stability but acts to mediate the exclusion of competing plasmids. **Proceedings National Academy Sciences USA** *97*, 12643-12648.

Heinemann, J.A. Ankenbauer, R.G. and Amábile-Cuevas, C.F. 2000. Do antibiotics maintain antibiotic resistance? **Drug Discovery Today** *5*, 195-204. (Featured on Biomednet.com)

Cooper, T.F. and Heinemann, J.A. 2000. Transfer of conjugative plasmids and bacteriophage λ occurs in the presence of antibiotics that prevent *de novo* gene expression. **Plasmid** *43*, 171-175.

Heinemann, J.A. 2000. The complex effects of gyrase inhibitors on bacterial conjugation. **Journal of Biochemistry Molecular Biology & Biophysics** *4*, 165-177.

Heinemann, J.A. 1999. Genetic evidence of protein transfer during bacterial conjugation. **Plasmid** *41*, 240-247.

*Heinemann, J.A. 1999. How antibiotics cause antibiotic resistance. **Drug Discovery Today** *4*, 72-79. (Featured on Biomednet.com)

Heinemann, J.A., Scott, H.E. and Williams, M. 1996. Doing the conjugative two-step: evidence for recipient autonomy in retrotransfer. **Genetics** *143*, 1425-1435.

Heinemann, J.A., Ankenbauer, R.G. and Horecka, J. 1994. Isolation of a conditional suppressor of leucine auxotrophy in *Saccharomyces cerevisiae*. **Microbiology** *140*, 145-152.

*Heinemann, J.A. Summer, 1993. Transfer of antibiotic resistances: a novel target for intervention. **Alliance for the Prudent Use of Antibiotics (APUA) Newsletter** *11*, 1, 6-7.

Heinemann, J.A. and Ankenbauer, R.G. 1993. Retrotransfer of IncP plasmid R751 from *Escherichia coli* maxicells: evidence for the genetic sufficiency of self-transferable plasmids for bacterial conjugation. **Molecular Microbiology** *10*, 57-62.

Heinemann, J.A. 1993. Bateson and peacocks' tails. **Nature** *363*, 308.

Heinemann, J.A. and Ankenbauer, R.G. 1993. Retrotransfer in *Escherichia coli* conjugation: bi-directional exchange or *de novo* mating? **Journal of Bacteriology** *175*, 583-588.

*Heinemann, J.A. 1991. Genetics of gene transfer between species. **Trends in Genetics** *7*, 181-185.

Heinemann, J.A. and Sprague, G.F., Jr. 1990. Transmission of plasmid DNA to yeast by conjugation with bacteria. **Methods in Enzymology** *194*, 187-195.

Heinemann, J.A. and Sprague, G.F., Jr. 1989. Bacterial conjugative plasmids mobilize DNA transfer between bacteria and yeast. **Nature** *340*, 205-209.

Reports (4)

IAASTD. 2009. Agriculture at a Crossroads: The Synthesis Report of the International Assessment of Agricultural Knowledge, Science and Technology for Development. Edited by B.D. McIntyre, H.R. Herren, J. Wakhungu, R.T. Watson. Island Press, Washington DC. (http://www.agassessment.org/index.cfm?Page=Plenary&ItemID=2713)

IAASTD. 2009. International Assessment of Agricultural Knowledge, Science and Technology for Development. Edited by B.D. McIntyre, H.R. Herren, J. Wakhungu, R.T. Watson. Island Press, Washington DC.

Heinemann, J.A. 2008. Human lactoferrin biopharming in New Zealand scientific risk assessement. Constructive Conversations/Kōrero Whakaaetanga (Phase 2). Report no. 13.

*Heinemann, J.A. 2007. A typology of the effects of (trans)gene flow on the conservation and sustainable use of genetic resources. UN FAO Background Study Paper 35 (ftp://ftp.fao.org/ag/cgrfa/bsp/bsp35r1e.pdf).

Gendora, Ltd.

Book Chapters (9)

*Heinemann, J.A. and Kurenbach, B. (2009) Horizontal transfer of genes between microorganisms. *In* Encyclopedia of Microbiology (M. Schaechter, editor-in-chief, third edition Academic Press).

*Heinemann, J.A. and Bungard, R.A. 2005. Horizontal Gene Transfer. *In* Encyclopedia of Molecular Cell Biology and Molecular Medicine (Meyers R.A. ed, second edition Wiley-VCH) p. 223-243.

Heinemann, J.A. 2004. Challenges to regulating the industrial gene: Views inspired by the New Zealand experience. *In* Challenging Science: Science and Society Issues in New Zealand (Dew, K. and Fitzgerald, R. ed, Dunmore Press) p. 240-257.

*Ferguson, G.C. and Heinemann, J.A. 2002. A brief history of trans-kingdom conjugation. *In* 2nd Ed. Horizontal Gene Transfer (M. Syvanen and C. Kado, eds, second edition Academic Press) p. 3-17.

*Weld, R.J. and Heinemann, J.A. 2002. The horizontal transfer of proteins between species: part of the big picture or just a genetic vignette? *In* 2nd Ed. Horizontal Gene Transfer (M. Syvanen and C. Kado, eds, second edition Academic Press) p. 51-62.

*Heinemann, J.A. 2000. Horizontal transfer of genes between microorganisms. *In* Encyclopedia of Microbiology (Joshua Lederberg, editor-in-chief, second edition Academic Press), 698-707.

*Heinemann, J.A. 1999. Looking sideways at the evolution of replicons. *In* Horizontal Gene Transfer (M. Syvanen and C. Kado, eds, first edition London: International Thomson Publishing), pp. 11-24..

*Singh, K. and Heinemann, J.A. 1997. Yeast plasmids. **Methods in Molecular Biology** *62*, 113-130.

*Heinemann, J.A. 1992. Conjugation, genetics. *In* Encyclopedia of Microbiology (Joshua Lederberg, editor-in-chief, first edition Academic Press), 547-558.

Scholarly Publications (*invited) Total: 37

Books

Heinemann, J.A. 2009. Hope Not Hype. The future of agriculture guided by the International Assessment on Agricultural Knowledge, Science and Technology for Development. **Third World Network Press (Penang).**

Journals

Heinemann, J.A. 2008. Desert Grain. **The Ecologist** 38, 22-24.

Kiers, E.T., Leakey, R.R.B., Izacs, A.-M., Heinemann, J.A., Rosenthal, E., Nathan, D. and Jiggins, J. 2008. Agriculture at a crossroads. **Science** *320*, 320-321.

Heinemann, J.A. Off the rails or on the mark? **Nature Biotechnology** *26*, 499-500.

Heinemann, J.A. and Traavik, T. 2007. GM soybeans-revisiting a controversial format. **Nature Biotechnology** *25*, 1355-1356.

Heinemann, J.A. Letter to the Editor. **Environmental Planning and Law Journal** *24*, 157-160.

Moore, B., Goven, J. and Heinemann, J. 2005. Terminator Vista. **New Scientist** *185*, 30.

*Heinemann, J.A. and Traavik, T. 2004. Reply to Monitoring horizontal gene transfer from transgenic plants to bacteria. **Nature Biotechnology** *22*, 1349-1350.

Anker, P., Zajack, V., Lyautey, J., Lederrey, C., Dunand, C., Lefort, F., Mulcahy, H., Heinemann, J. and Stroun, M. 2004. Transcession of DNA from bacteria to human cells in culture. A possible role for oncogenesis. **Annals NY Academy Science** *1022*, 195-201.

*Heinemann, J.A. and Billington, C. 2004. How do genomes emerge from genes? **ASM News** *70*, 464-471. (This paper was selected by ASM for a special author feature.)

Amábile-Cuevas, C.F. and Heinemann, J.A. 2004. Shooting the messenger of antibiotic resistance: Plasmid elimination as a potential counter-evolutionary tactic. **Drug Discovery Today** *9*, 465-467.

*Heinemann, J.A. 2003. Is horizontal gene transfer the Cinderella of genetics? **New Zealand Bioscience** *12*, 51-54.

*Heinemann, J.A. 2002. Bacterial Resistance to Antimicrobials (Review). **Drug Discovery Today** *7*, 758.

*Heinemann, J.A. 2002. Are DNA sequences too simple as Intellectual Property? Reply to Williamson—Gene patents: are they socially acceptable monopolies, essential for drug discovery? (Commentary) **Drug Discovery Today** *7*, 23-24.

Heinemann, J.A. 2001. Genetic scientists under siege: What next? **NZ Microbiology** *6*, 15-17.

Heinemann, J.A. 2001. A 'bias' gene? (Commentary) **BioEssays** *23*, 1081-1082.

Gendora, Ltd.

Heinemann, J.A. 2001. Can smart bullets penetrate magic bullet-proof vests? **Drug Discovery Today** *6*, 875-878.

*Heinemann, J.A. 2001. The art of courtship. (Commentary) **Drug Discovery Today** *6*, 234.

Heinemann, J.A. 2001. The fate of students within our hands. (Editorial) **New Zealand Education Review** (Jan. 12, p. 7).

Heinemann, J.A. 2000. How can we build a 'knowledge economy' if research is handcuffed? (Editorial) **Nature** *406*, 13.

Heinemann, J.A. 2000. Research hazards. **New Zealand Education Review** (Sept. 8, p. 9).

Heinemann, J.A. 2000. Funding for knowledge-sake (Letter) **Drug Discovery Today** *5*, 222-223.

*Heinemann, J.A. and Roughan, P.D. 2000. New hypotheses on the material nature of horizontally mobile genes. **Annals NY Academy Science** *906,* 169-187.

Adams, B. and Heinemann, J.A. 2000. Antibacterial Viruses and antibacterial agents: a one-two punch? **New Zealand Medical Journal** *113*, 107.

Gunn, A. and Heinemann, J.A. 2000. Stealth antibiotic resistance. **New Zealand Medical Journal** *113*, 107.

*Heinemann, J.A. 1998. Superbugs: by killing them we have made them stronger. **New Zealand Science Monthly** *9*, 6-8.

Reports

*Heinemann, J.A. 1997. Assessing the risk of interkingdom DNA transfer. In Nordic Seminar on Antibiotic Resistance Marker Genes and Transgenic Plants. pp. 17-28. Oslo: Norwegian Biotechnology Board.

Book Chapters

*Heinemann, J.A. and Goven, J. 2006. The social context of drug discovery and safety testing. In Antimicrobial Resistance in Bacteria (C.F. Amábile-Cuevas, ed., second edition). Horizon Bioscience, 179-196.

*Heinemann, J.A. 2004. Horizontal transfer of genes between microorganisms. In Desk Encyclopedia of Microbiology (specially selected modified version of original 2000 article appearing in the second edition of the Encyclopedia of Microbiology Academic Press), Elsevier, Ltd. 580-588.

Gendora, Ltd.

*Heinemann, J.A. and Silby, M.W. 2003. Horizontal gene transfer and the selection of antibiotic resistance. In Multiple Drug Resistant Bacteria (C.F. Amábile-Cuevas, ed). Horizon Scientific Press, p. 161-178.

*Heinemann, J.A. 1996. Virile sensitive males resist drugs. **Microbiology Australia** 17, 17.

Other

Heinemann, J.A. 1996. M.D.s and Ph.D.s: Differences in Pay (Editorial) **ASM News** *62*, 234-235.

*Heinemann, J.A. 1993. Review of "Materials for the Study of Variation Treated with Especial Regard to Discontinuity in the Origin of Species" by William Bateson. **Quarterly Review of Biology** *66*, 429-430.

Heinemann, J.A. 1993. Differential Salary Scales (Editorial) **Nature** *363*, 202.

Heinemann, J.A. 1993. "Doctor Old-Boy Network?" (Editorial) **ASM News** *59*, 588-589.

Pincus, S.H., Rosa, P.A., Spangrude, G.J. and Heinemann, J.A. 1992. The Interplay of Microbes and Their Hosts. **Immunology Today** *13*, 471-473.

Heinemann, J.A. 1992. Obtaining Information on Candidates for ASM Offices (Editorial). **ASM News** *58*, 588.

*Heinemann, J.A. and Walsh, T.J. 1991. Cover illustration. **Trends in Genetics** *7*.

Significant Public Submissions (*for the University of Canterbury) Total: 10

2006 Submission to Codex Alimentarius Commission on Recombinant DNA Plants Modified for Nutritional or Health Benefits

2006 Submission to Food Standards Australia/New Zealand on A580 Food Derived From Amylase-Modified Corn Line 3272 Initial Assessment Recommendation

2006 Submission to Food Standards Australia/New Zealand on A549 High Lysine Corn Draft Assessment Recommendation

2005 Submission to Food Standards Australia/New Zealand on A549 High Lysine Corn Initial Assessment Recommendation

*2004 Submission to the Ministry of Foreign Affairs and Trade on the question of ratifying the Cartagena Protocol on Biosafety

2004 Submission to Food Standards Australia New Zealand on application A524 Food Derived from Herbicide-Tolerant Wheat MON 71800.

*2003 To the Education and Science Committee call for submissions on the New Organisms and Other Matters Bill.

2002 To the Ministry of Science Research and Technology on the Public Discussion Paper "New Zealand Biotechnology Strategy".

Gendora, Ltd.

*2002 To the Finance Select Committee on the Hazardous Substances and New Organisms (Genetically Modified Organisms) Amendment Bill/Inquiry.

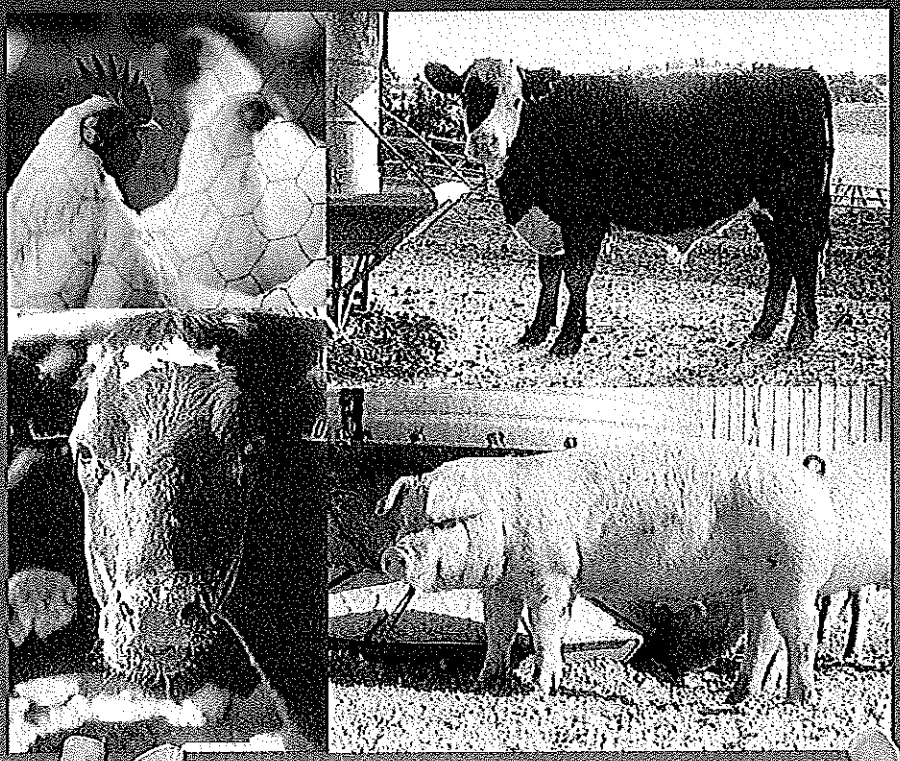
2002 Submission to the New Zealand Environmental Risk Management Authority on AgResearch Application GMD01194.

# FASS Facts

*On Biotech Crops – Impact on Meat, Milk and Eggs*

Are the meat, milk and eggs
from livestock fed biotech feeds

## safe to eat? Yes!

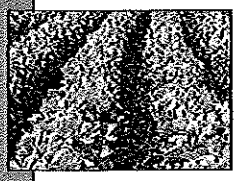


FEDERATION OF ANIMAL SCIENCE SOCIETIES

## Background

The term "biotechnology" has sparked controversy in recent years. Much of the controversy is fueled by activist groups who perceive genetic enhancement as somehow "unnatural." There are also concerns about introduction of genes that may produce allergenic responses or have adverse effects on the environment. However, biotechnology is a remarkable technology that has produced many benefits to consumers. Unfortunately, Americans don't have the information they need to sort facts from fear about this technology and its benefits.

Today's biotechnology is simply a more precise means of doing what has been done for centuries through conventional breeding – striving to develop crops and foods that have desirable characteristics. These characteristics might include protection against insect pests, which minimizes the need for pesticides; higher crop yields; or improved nutritional properties.



Conventional plant breeding was done through trial and error. Scientists could spend 10 to 15 years crossing plants and growing them to bring out certain characteristics from the tens of thousands of genes that each plant possesses. Oil seed rape (the progenitor of canola) was one of the successes of this type of crossbreeding. In fact, rapeseed oil was an industrial lubricant unfit for human consumption until canola was genetically modified to become low erucic acid rapeseed oil, which eliminated some of its anti-nutritional properties. Today, it is one of the healthiest oils on the market. Most foods consumed today – like corn, wheat and tomatoes – are long-term, conventional breeding success stories. And now, through genetic modification, desirable traits can be selected and more quickly incorporated rather than waiting a decade for results.

Genes from different species are often highly related. The same genetic material may be found in multiple species. New genetic material adds selected, special characteristics to the new plant. These special characteristics or traits benefit everyone: both the consumer as well as the farmer.

## Do Livestock Consume Biotech Feeds?

Yes, livestock have been fed biotech feeds since biotech crops were first introduced in 1996. Recently, livestock feeds have been improved using modern methods of agricultural biotechnology, such as recombinant DNA technology. The application of recombinant DNA technology frequently has been referred to as genetic modification. Crops developed using modern methods of agricultural biotechnology are referred to as biotech crops as opposed to crops developed using conventional plant breeding. Two important types of commercially available biotech crops include crops tolerant to herbicides and crops protected against insect pests.

Both conventional and biotechnology techniques have benefited agriculture immensely because they make feed more plentiful and affordable. When inputs are less costly, so are the outputs purchased by consumers: meat, milk and eggs. In fact, we spend significantly less of our disposable income in the United States on food than any other nation in the world thanks to the successes of our agricultural system, of which agricultural biotechnology is a key part.

## Why Do Farmers Raise Biotech Crops?

Farmers raise biotech crops because they are more reliable and profitable than conventional crops.

First, the amount of insecticide applied to insect-protected crops is reduced. Yields of corn, cotton and soybeans are increased in many instances. The majority of these cost savings are enjoyed by the grower. Overall, the cost of producing an acre of the crop is reduced and some of these cost savings ultimately can be passed on to the consumer.

Since seeds for biotech corn and soybeans were first sold in the United States in 1996, farmers have continued to plant increasing acreage. More than one-half of the soybeans and more than one-third of the corn planted in 2000 were biotech crops.

## Farmers and Consumers Enjoy the Benefits of Biotech Crops

Consumers have reaped the benefits of biotech crops in the form of higher quality products. In the future, consumers will see expanding benefits of biotech crops as the use and sophistication of biotechnologies grow.

For example, a corn called *Bt* corn has been bred to be protected against a common pest called the European corn borer. This results in less damage to the corn plant which, in turn, reduces the infection by a fungus that produces a mycotoxin called fumonisin. *Bt* corn varieties therefore contain less fumonisin. Fumonisin has been shown to be a carcinogen in humans, so risk of human exposure to fumonisin from corn-based products is being reduced thanks to biotechnology.

There will be many biotech crops with enhanced levels of nutrients or other beneficial substances in the plant. For example, "golden rice" is being developed with increased levels of vitamin A and iron. Golden rice could be a significant addition to the diet and health of many persons throughout the world who are currently deficient in vitamin A. Other plants will produce nutritionally enhanced oils, or will improve the shelf life of the food.

## Are Nutrients or Anti-Nutrients in Biotech Crops Different?

No, both the levels of nutrients and anti-nutrients in the current biotech crops are the same as in conventional crops. As stated above, some crops are being developed which will have increased levels of nutrients, including feeds, like the lysine and methionine content in corn grain. Likewise, anti-nutrients, or undesirable proteins, such as trypsin inhibitor in soybeans or gossypol in cotton, are unchanged in biotech crops compared to conventional crops.

Livestock feeds such as corn grain, whole-plant chopped corn, corn stover and soybeans from the current biotech crops have been compared with conventional feeds to measure any changes in feed composition. The research clearly shows that the levels of nutrients – such as protein, carbohydrates, fat, energy, amino acids, fatty acids, minerals, vitamins and other components of biotech and conventional feeds – are substantially equivalent and are well within the normal range of values reported in the scientific literature.

## Are Biotech Feeds Safe for Livestock?

Yes, biotech feeds are safe for livestock. Livestock digest and absorb nutrients from biotech feeds in the same way they do conventional feeds. The digestive process in all livestock breaks down the nutritional components in feeds and uses these nutrients for the growth and development of the animal.

In addition, livestock growth, milk production, milk composition and health are not different, whether fed conventional or biotech feeds. Over 30 different animal feed performance studies have been conducted. All of these studies have shown that corn grain or soybean meal from biotech plants performs similarly to the grain or meal from conventional plant varieties.

## Are Nutrients in Meat, Milk and Eggs Different?

Nutrients in meat, milk and eggs from livestock fed biotech feeds are the same as those from livestock fed conventional feeds. Because most components of feeds are broken into smaller components during digestion by the animal, plant proteins have not been detected in milk, meat or eggs.

The introduced DNA and newly expressed protein(s) from biotech crops have not been found in the meat, milk or eggs from animals fed biotech crops.

## Are Meat, Milk and Eggs Safe to Eat?

Yes, meat, milk and eggs from livestock and poultry consuming biotech feeds are safe for human consumption. By 2020, global protein consumption from meat, milk and eggs is predicted to increase dramatically, a "Livestock Revolution." Therefore, with biotech crops and animal food products, we will benefit the nutrition and well-being of the world's population, especially children in developing countries.

## U.S. Government Agencies Heavily Regulate Biotech Crops by Requiring Extensive Field and Safety Tests

FOOD AND DRUG ADMINISTRATION (FDA)

The FDA ensures that any human food or animal feed derived from new plant varieties are safe to eat. After completion of the voluntary FDA consultation process, more than 40 crops have been developed for market. The FDA has recently proposed to change the process

from voluntary to mandatory. Foods derived from biotechnology must be labeled only if they differ significantly from their conventional counterparts. For example, if the nutritional value or the potential to cause an allergic reaction is altered.

UNITED STATES DEPARTMENT OF AGRICULTURE (USDA)

The USDA is the U.S. government's lead agency regulating the safe field-testing of new biotech plant varieties. Impact on the environment, on endangered or threatened species and on "non-target" species are all considered.

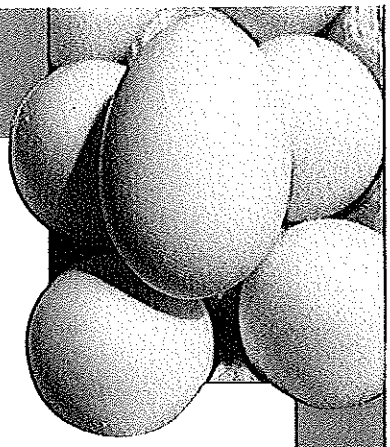
ENVIRONMENTAL PROTECTION AGENCY (EPA)

The EPA has authority over all new pesticides, including biotech plants, which produce their own protection against pests. In deciding whether to register a new biotech product, the EPA considers human safety, impact on the environment, effectiveness on the targeted pest and any effects on other endangered and threatened species.

Recently StarLink corn, which was approved only for animal consumption, was found in human foods. The EPA now has a policy of not approving biotech crops intended for animal feeding without simultaneously approving the crops for human use. This action is taking precautions against a recurrence of a StarLink situation.

## Should We Label the Meat, Milk and Eggs?

FASS recognizes the significant logistical problems that labeling incurs for meat, poultry, egg and milk processors. FASS does not support labeling of food derived from animals fed biotech crop materials because the scientific evidence consistently indicates that meat, milk and eggs derived from animals fed biotech feeds are equivalent to products from animals fed conventional feeds. FASS supports food labeling that is meaningful to the consumer and serves a specific purpose. FASS supports food labeling if a food product is substantially changed in nutritional composition or safety.



## Conclusions

The Federation of Animal Science Societies has reviewed the scientific information concerning the consumption of biotech feeds by livestock. We conclude that:

- Acceptance of biotech feeds for livestock must be based on sound science;
- The use of biotechnology techniques will be essential to improving agricultural plants and animal products;
- Agricultural biotechnology is capable of improving supplies of livestock feeds and healthful animal and plant food products;
- The safety of meat, milk and eggs is adequately assured by the science-based risk assessment procedures used by government agencies and developers;
- The DNA introduced in biotech plants and the proteins encoded by this DNA have not been detected in the meat, milk or eggs from animals fed these products; and
- **Meat, milk and eggs from animals fed biotech feeds are safe for human consumption.**

For more information, contact the Federation of Animal Science Societies.


The Federation of Animal Science Societies (FASS) is a professional organization made up of approximately 10,000 scientists in academia, government and industry which exists to serve society through the improvement of all aspects of food animal production. FASS represents the combined memberships of the American Dairy Science Association, the American Society of Animal Science and the Poultry Science Association.

FASS Federation of Animal Science Societies

**Federation of Animal Science Societies**
1111 North Dunlap Avenue, Savoy, IL 61874
Phone: (217) 356-3182, Fax: (217) 398-4119, E-mail: fass@assochq.org, Web site: www.fass.org