GREENPEACE

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Submission to the NSW Review of the Gene Technology (GM Crop Moratorium) Act 2003
30 August 2007

Introduction

Thank you for the opportunity to register a submission concerning the state moratorium on genetically modified canola. The New South Wales moratorium was introduced in 2003 because of concerns within industry, the farming sector and regional comunities about the impact of GE crops on markets. The concerns that led to the introduction of the moratorium include liability and insurance issues, problems with segregation and cross contamination and export market sensitivities.

In the four years since the moratorium was introduced these concerns have proven highly justified. GE contamination scandals have plagued countries, such as the US, that have adopted GE crops. These have resulted in hundreds of millions of dollars of lost export revenue and and costly litigation. Segregation of non-GE canola in Canada has failed, leading to the collapse of its non-GE and organic canola industries. Farm incomes in Canada have plumetted since the introduction of GE canola and Canada has entirely lost its canola seed exports to Europe. Furthermore, consumers in Australia, and major export markets such as Europe and Japan, remain resolutely opposed to GE food. Recent studies questioning the science behind GE and the safety of GE food have only served to heighten consumer concern on the issue.

In 2005 Ian MacDonald, the NSW Agriculture Minister stated that “at this point the lack of segregation trials means that there has been no practical demonstration of the capacity to segregate GM and non-GM product across the supply chain to differing market standards.”[1] He argued that “it is important that independent, small-scale agronomy trials of GM canola occur prior to larger-scale segregation trials being conducted to address marketing issues.”[2] This work has still not been completed.

Greenpeace believes that the reasons for a moratorium on commercial GE food crop production are stronger than ever, and that the current moratorium should be extended by a further 5 years.

1. Economic benefits of the moratorium

1.1. Benefits for consumers

Polling by Swinburne University and Biotechnology Australia last year shows that the majority of Australians are uncomfortable with eating GE food and are unlikely to eat it.[3] Similar attitudes exist in our key export markets, such as Europe and Japan. A survey by the Pew Global Attitudes Project shows that Western Europeans and Japanese consumers are overwhelmingly opposed to scientifically altered fruits and vegetables because of health and environmental concerns.[4] A 2006 poll, by the Japanese Ministry of Agriculture, Fishery and Forestry (MAFF), found that 78% of Japanese consumers were uncertain about the impacts of eating GE food.[5] The current moratorium has allowed the market to supply what consumers want – non-GE canola with a low risk of GE contamination, and without the need for costly segregation and identity preservation (IP) systems. Segregation and IP costs, along with the costs of recalls when the inevitable contamination occurs will invariably be passed on to food companies and, ultimately, to consumers.

1.2. Benefits for user industries

The moratorium has benefited industry in several ways since it’s implementation. Benefits include:

- price premiums and preferential market access for Australian canola;
- lower production costs, since costly segregation and identity preservation processes are not required;

- an absence of costly recalls due to unwanted GE contamination – such as recently happened with rice products in the US;
- a reputation among domestic and export markets for high quality non-GE products.

The issues surrounding market access, price premiums, and segregation costs will be discussed in more detail in the following sections.

1.3. Benefits for farmers

"Over the past decade, corporate and government managers have spent millions trying to convince farmers and other citizens of the benefits of genetically-modified (GM) crops. But this huge public relations effort has failed to obscure the truth: GM crops do not deliver the promised benefits; they create numerous problems, costs, and risks; and Canadian consumers and foreign customers alike do not want these crops.

It would be too generous even to call GM crops a solution in search of a problem: These crops have failed to provide significant solutions, and their use is creating problems— agronomic, environmental, economic, social, and (potentially) human health problems." Canadian National Farmers Union (2005)[6]

As Table 1 demonstrates, the vast majority of the world's canola is non-GE. Claims that Australia is being 'left behind' by not adopting GE canola are nonsense. Canada is the only major canola producing country to have adopted GE canola and its farmers are heavily subsidised. If Australia adopted GE canola without subsidising its farmers, it would be the first major canola producer in the world to do so. Europe decided not to adopt GE canola because of widespread community opposition to GE crops and concerns about the biodiversity impacts of GE canola.[7]

Table 1: Key characteristics of the world canola seed market

Producer	***Percentage of world production[8]***	***GE canola adopted?***
European Union	32	No
China	27	No
Canada	18	Yes
India	15	No
Australia	3	No

1.3.1. Price Premiums

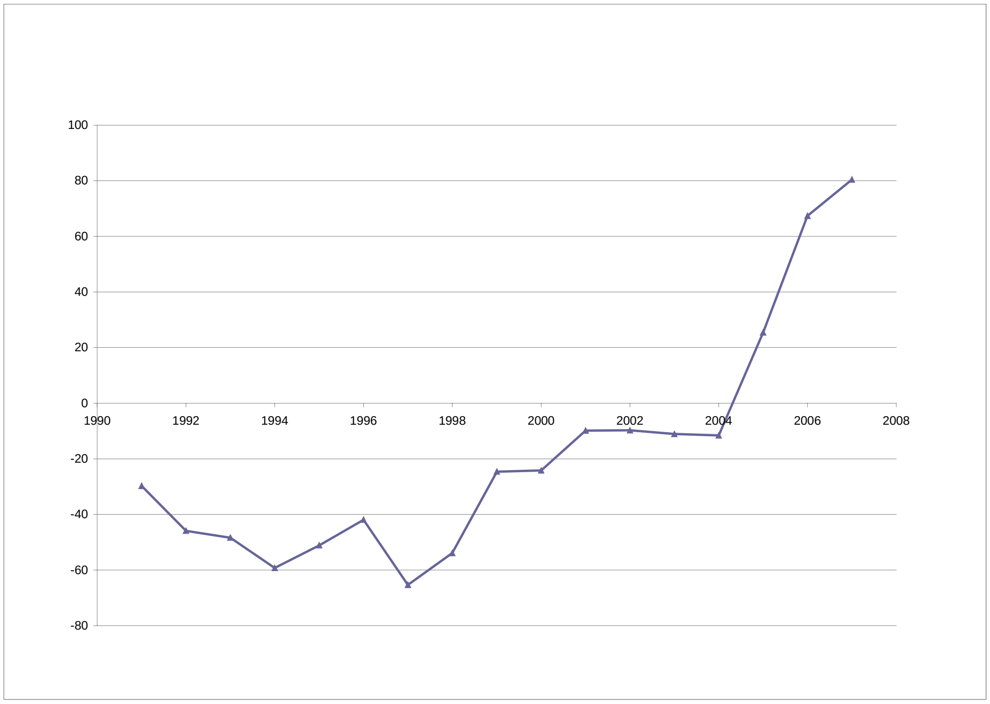


Figure 1: Annual average price difference between Australian and Canadian canola

As illustrated in Figure 1, in 1998, the difference between Australian and Canadian canola prices was about $70 a tonne in favour of Canada. However, by May 2006 Australian prices had exceeded Canadian prices by some $50 a tonne.[9] It is difficult to determine how much of this price premium is due to Australian canola's non-GE status, since the information is commercially confidential. However a WA Department of Agriculture report observes that premiums for non-GE canola exist in both Japan and Europe. [10] Premiums of $12 to $14 per tonne have been reported in Europe.[11] However, Portmann and Tucek noted that it was unlikely that the real price differential would be seen until there was a shortage of non-GE product.[12] This is exactly what happened in 2006 when Australian canola producers were badly hit by the drought. This saw Australian canola prices soar to $115 a tonne more than Canadian canola.[13]

1.3.2. Supply and production costs at all stages

The moratorium currently protects farmers from costs associated with segregation, identity preservation and contamination. Currently, non-GE farmers are expected to bear many, if not most, of the costs of introducing genetically engineered (GE) crops. The Australian Bureau of Resource Economics (ABARE) has estimated that the introduction of GE canola will cost non-GE farmers 5-15% of the farmgate value of their crop.[14] That figure is based on a 1% contamination acceptance threshold – a threshold not currently accepted by farmers or markets. If a 'zero tolerance' segregation system is implemented, costs are likely to be significantly higher.

Even maintaining a 1% threshold is likely to be extremely difficult. A UK study demonstrated that a single GE canola crop contaminated subsequent grain crops in the same field at levels above 1% for up to 16 years unless the most stringent of measures were implemented.[15] Currently, there are no on-farm conditions imposed on the commercial release of GE canola.[16]

Additionally, non-GE farmers will be exposed to increased financial risk, including potential liability and lost market access, when the inevitable contamination occurs.[17] Initial attempts to segregate non-GE canola in Canada have categorically failed and this issue is discussed in greater detail in section 3. Contamination costs must be considered an inevitable cost of doing business if Australia embraces GE food crops.

1.3.3. Productivity and profitability

"Those who assert that GM seeds increase farmers' net income need to produce some data. And, as we stand ten years after the introduction of these seeds, and as we stand mired in the worst farm income crisis in Canadian history, it is probable that such data will be hard to produce. The claim that GM seeds make our farms more profitable is false." Canadian National Farmers Union (2005)[18]

In a recent report, Nuffield scholar Andrew Broad estimated that genetically engineered (GE) Roundup Ready canola would cost $27.82 per hectare more to grow than conventional canola. He claimed that this would be compensated for if there was a yield gain of 4%, not taking into account the additional costs of segregation and identity preservation.[19] However, there is no evidence that GE canola actually increases yields.

No independent trials have been conducted to assess any potential yield gains associated with the GE canola varieties approved for commercial production in Australia. Bayer and Monsanto have failed to enter their GE canola varieties into national seed listing trials in order that they can be independently assessed. The data from Monsanto and Bayer's own studies suggests that there may actually be a substantial yield penalty associated with GE canola. Despite Monsanto adding the Roundup Ready gene to 'elite varieties', the best Australian trials of Roundup Ready canola yielded only 1.055t/ha – at least 16% below the national average of 1.26t/ha. Monsanto has since removed the details of the trials from its website.

In 2005 Ian MacDonald, the New South Wales Agriculture Minister, argued that before the moratorium is lifted "it is important that independent, small-scale agronomy trials of GM canola occur prior to larger-scale segregation trials being conducted to address marketing issues."[20] This work has still not been completed. In 2003, the New South Wales Government approved field trials of Monsanto and Bayer's GE canola in order that their agronomic performance could be evaluated. However, both companies pulled out of the trials with Bayer citing 'poor seasonal conditions' as the reason.[21]

As Figure 2 shows, Canadian canola yields from 1965 to 1994 increased by an average of 0.2 bushels per acre per year. This was the result of selective breeding and improvements to farming techniques. GE canola was introduced to Canada in 1996, however from 1995 to 2004, yield only increased by an average of

0.1 bushel per year. Those who claim that GE crop technologies positively contribute to yield - either directly or indirectly - have no data to prove that assertion.[22]

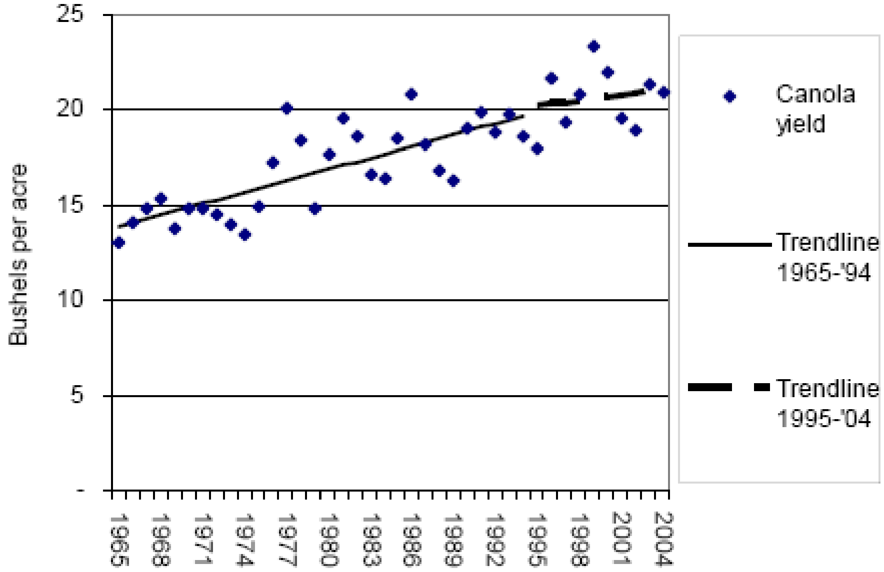


Figure 2: Canadian canola yields, 1965-2004[23]

"Farmers' profits haven't just disappeared; they've been taken. The farm crisis didn't just happen; it was caused. The family farm isn't dying; it's being killed. And the perpetrations of this destruction are the agribusiness corporations who are using their market power to extract profits that would otherwise end up on our farms. Farmers can't make a living because agribusiness giants insist on making a killing."
Canadian National Farmers Union (NFU) 2005[24]

Even if GE canola did have the potential to increase yields, as the Canadian National Farmers Union point out "any initial economic benefits will be quickly outweighed as farmers are drawn further under corporate control."[25] As Figure 3 shows, net farm incomes in Canada have plummeted since the introduction of GE canola. The last five years in Canada have been the worst five years of realised net farm incomes in the history of the country.[26] At the same time, we have seen corporate agribusiness "earn" record profits. In Canada, corporations have captured more than 100% of the profits associated with GE (the shortfall has been made up by Government subsidies) and farmers have become increasingly dependant on high-tech seeds and chemicals. The knowledge and power has shifted from the farmers to giant agri-business corporations such as Bayer and Monsanto. And as the power has shifted, so have the profits.[27]

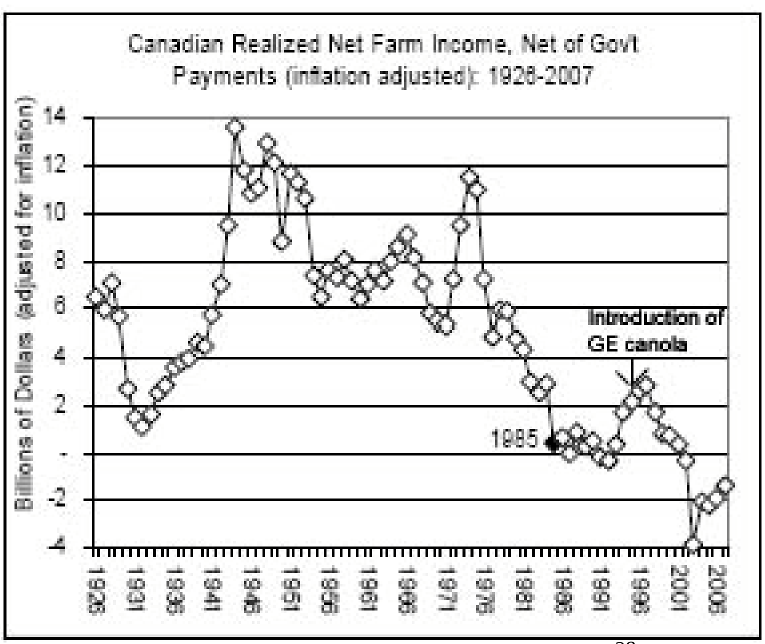


Figure 3: Canadian Realised Net Farm Incomes[28]

Companies such as Monsanto have gone to great lengths to ensure that they capture the full benefits of the introduction of GE crops. Monsanto spends over US$10 million annually investigating, intimidating, pressuring, and suing farmers.[29] The company has a staff of 75 employees devoted to these pursuits and Monsanto also contracts dozens of lawyers from outside firms. It has sued for, and won, judgements as high as US$3 million and several more over $1 million.[30]

While the net income numbers in Figure 3 represent farmers in general (not just canola farmers), the net income trend is representative of the experience of canola farmers. According to the NFU, if anything, the trend is optimistic, because - if it were available - data on net income from canola production would produce a graph line that would fall much more precipitously than the line in Figure 3. The profitability of canola production, like that of crop production in general, has crashed over the past decade.[31]

Data from Statistics Canada shows that small and medium-size Canadian farms now rely on off-farm income for approximately 90% of their total income; large farms rely on off-farm income for over half (52.1%) of their total income; and even Canada's largest farms, depend on off-farm income of between 25.9% and 33.5% of their total income.[32]

The NFU states that a combination of government subsidies, increased debt loads (now exceeding C$52 billion), and off-farm income are the main factors that allow farming to continue despite sub-Depression level net incomes.[33] Whilst Australian farmers remain unsubsidised, according to the NFU, Canadian subsidies work out to about A$150 per ha of cropland.[34] This year ABARE estimates that over 1million ha in Australia will be sown to canola this year.[35] If the Australian Federal Government were to subsidise Australian canola farmers to the same extent it would cost $150 million a year. The NFU also estimates that in order for most Canadian farmers to take a "wage" from their farms, current subsidies would have to nearly double. In other words, if the Australian Government wants GE canola farmers to stay in business it needs to be prepared to subsidise them to the tune of over $300 million a year.

The adoption of GE canola has certainly not improved grower profitability in Canada, and there is no reason to suggest that it will in Australia. Since there are no demonstrated agro-economic benefits associated with the GE canola varieties approved for commercial use in Australia, the uptake of the technology by farmers is likely to be low without heavy discounting. However the increased costs associated with segregation and contamination will be born by all canola farmers, regardless of whether they choose to adopt the technology.

Extending the moratorium will retain preferential market access for New South Wales canola and ensure the continued profitability of canola production for farmers.

1.3.4. Export market access and reputation

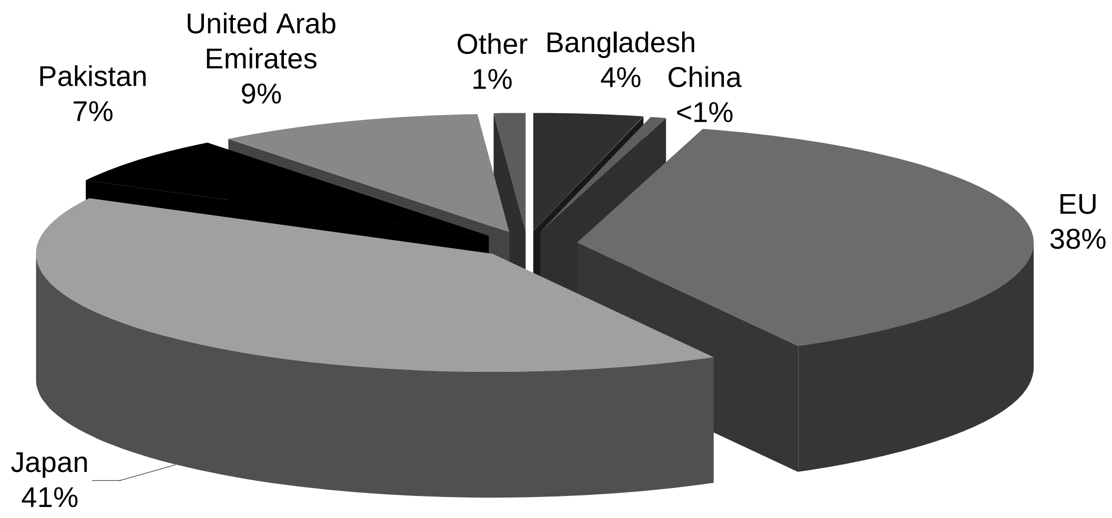
An ABARE research report concluded that "a range of market access restrictions related to GM products means that it is easier to trade non-GM grains in the current market environment than it is to trade GM grains."[36]

In the early and mid-1990s, before the widespread introduction of GE canola, Canada sold much of its canola crop to the EU. The EU took 16% of total Canadian exports in 1993, 32% in 1994 and 25% in 1995. However, as a result of the introduction of GE canola Canada entirely lost its canola seed exports to Europe. Over the past decade, China has become a major buyer of Canadian canola. However, China is a low-price market, whereas the EU was a premium-price market. Today, Canadian canola prices, adjusted for inflation, are at a record low.[37] Because of the introduction of GE varieties and attendant market loss, canola prices have fallen.[38]

USDA figures indicate that high levels of carry-in stock (stock not sold immediately) are afflicting Canadian GE canola producers, indicating an inability to immediately sell their GE canola into the world's markets. While Department of Agriculture figures for Australian carry-in stock of canola are not available, there is no evidence that Australia is having similar problems.

As can been seen from Figure 4, Australia's most important export markets for canola are Japan and Europe, accounting for 41% and 38% of exports in 2006.[39] The EU has instituted strict rules regarding the import and labelling of GE products, reflecting the strong and continuing resistance to GE in Europe.[40] Other countries that are planning to introduce GE labelling laws include Quatar and Kuwait. Consumer campaigns are currently underway in Europe, Japan, the US and Canada for tighter labelling laws regarding GE food.

Figure 4: Destinations for Australian canola exports 2006



Japan

Japan, in particular, is an extremely important market - receiving 50% of Australian canola seed exports between 2001 and 2004. Consumer resistance to GE is extremely strong in Japan, and the recent scandal regarding GE canola contamination around Japanese ports has further heightened fears. If GE canola is commercialised in Australia the negative consequences for Australian grain markets could be significant. The WA inquiry observed that "Australia was able to secure greater market access because it was producing non-GM canola"[41] According to Perry Gunner from ABB Grain, Japan's interest in buying Australian canola is growing. He states that there are further opportunities to sell canola there because of Australia's GE free status.[42] This month it was reported that Canadian canola exports to Japan will be down this year due to competition from Australia.[43]

Several Japanese companies specifically market Australian GE-free canola oil and attract premium prices for their products. Several Japanese consumer co-ops have expressed concerns about GE crops. Uni Co-op reportedly switched from Canadian canola to supply from Australia in response to consumer demand for GE-free product. Similarly, Shutoken Co-op has indicated it wants GE-free canola.[44] Organic export companies have noted that export opportunities for organic canola oil are not being captured due to major immediate supply shortages.[45]

Europe

GE canola was introduced to Canada in 1996, and by 1998 Canada had entirely lost its sales of canola seed to Europe. The EU is a growing market for Australian canola, accounting for 38% of Australia's exports in 2006. The increase in demand is driven largely by the biodiesel sector, and the EU is sourcing canola from Australia precisely because of its non-GE status. According to the Canola Council of Canada, if it weren't for the GE restrictions, between 300-400,000 tonnes of Canadian canola could have been exported to the EU in 2005-2006. Furthermore, the Council has estimated that the level of demand could easily double in 2006-2007, making the opening of EU borders to Canadian canola an important priority for Canada.[46] Clearly Australia has a important marketing advantage over Canada when it comes to serving EU markets. There is no evidence to support the claim that the EU is likely to lift its ban on Canadian canola seed imports. According to the Canadian Canola Council "despite the recent World Trade Organization Panel ruling on the approval and marketing of biotech products in the EU, it could be some time before genetically modified (GM) canola is welcome in the EU".[47]

A recent ABARE report has noted that the unintended presence of GE canola in Australian shipments could be a problem if the EU becomes a larger and more regular importer of Australian canola, because of the EU labelling threshold of 0.9%.[48] Based on the current trend towards increased demand for biodiesel, there is an extremely high probability of this happening.

Domestic markets
The domestic market accounts for approximately 20-30% of total canola seed production (depending on the year), making it the third largest market for Australian canola. The two largest buyers are Goodman Fielder and Unilever, both of which have a policy to avoid the use of GE derived canola oil.

1.3.5. Effects on other industries

The moratorium protects not only canola farmers from the negative impacts of GE contamination but also prevents the contamination of other crops such as barley and wheat. Both AWB Limited and the Australian Barley Board (now ABB Grains), key grain marketers in Australia, have expressed concerns over the commercialisation of GE canola in Australia because of the possibility that unintended presence of GE canola seed in wheat and barley shipments would jeopardise some of their markets. Similarly, numerous marketers of Australian livestock products have claimed that there is a market advantage to not feeding GE material to livestock.[49] Canola meal is commonly used as stock feed in the dairy industry which is extremely sensitive to GE contamination. Consumer concern regarding the use of GE stock feed, both in Australia and in important export markets such as Japan and Europe, has resulted in the majority of Australian dairy suppliers having non-GE policies. These companies include:

- Attiki
- Bega Cheese
- Dairy Farmers
- Jalna Dairy Foods
- Lactos
- Murray Goulburn Co-operative
- National Foods
- Norco Co-operative
- Parmalat Australia
- Snowy Mountains Organic Dairy
- Tatura Milk Industries
- Warrnambool Cheese & Butter Factory

A full list of these companies' policies regarding GE ingredients can be found in Appendix A.

1.3.6. Research and development and access to new varieties

In Australia, although private companies are exerting increasing control over seed research and development, there is at least some publicly funded seed development. In Canada the rise of global seed corporations has resulted in the decimation of public seed development. Corporations, such as Monsanto and Bayer, are using patents, contracts, and ever-tighter Plant Breeders' Rights legislation to ensure that farmers pay for seed. When farmers don't pay, corporations sue. Such lawsuits over seeds are fast proliferating, with the companies seeking and receiving farm-destroying amounts of money. Not satisfied with these legal tools, companies are working to overcome resistance to Terminator Technology (seed genetically engineered to be sterile after one generation) in order to force farmers to buy new seed each year. Companies have also successfully pressured governments to do less publicly funded plant breeding, meaning that farmers have fewer and fewer alternatives to corporate seeds.[50]

1.3.7. Sustainability on farm

The negative environmental impacts of GE crops are well documented. A summary of these can be found in our attached briefing *The Environmental Impacts of GE crops*. [51] GE crops threaten to undermine the economic basis of agriculture by unbalancing the biosphere, increasing weed resistance problems, endangering beneficial insects, and eroding bio-diversity.[52] Biodiversity is a vital source of raw materials for agriculture and an essential component of environmental well-being.[53] The moratorium has protected farmers from these threats to the sustainability of farming.

2. Two very good reasons why the moratorium should be extended

2.1. Consumer resistance

Consumer resistance is the strongest economic argument for remaining GE free. As the Australian Wheat Board has noted, "Legislation might allow GMs but that doesn't mean our customers want them."[54] The Western Australian inquiry noted "the unpredictable nature of world commodity markets" and acknowledged "the shortcomings of any attempt to predict future market conditions and consumer behaviour"[55] It also concluded that "there exists no certainty in the market acceptability of GM foods, with consumer attitudes being both varied and unstable on the issue."[56] Since there is no market in the world with a preference for GE canola over non-GE canola; and Australian canola is currently receiving favourable market access and premiums because of its non-GE status; it would be reckless for the New South Wales Government to lift the moratorium in the current market environment.

As the Canadian National Farmers Union puts it:

"While the benefits are questionable, risks and costs are real. Consumers are rejecting GM foods. Markets in Europe, Japan, and elsewhere are closing and domestic markets are likewise threatened. This is driving prices down. Closing markets and falling prices threaten to overwhelm any small, short-term economic benefits that GM crops or livestock may offer. Further, the proliferation of some GM crops has effectively deprived many organic farmers of the option to grow those crops."[57]

There is no evidence of waning consumer opposition to GE crops in either Australia, or our major export markets such as the EU and Japan. As more studies come to light that question the scientific basis of GE and the safety of GE foods, this is likely to have a further detrimental effect on consumer attitudes to GE foods. This will obviously have an effect on the marketability of GE crops. Another major health or contamination scandal involving GE food crops, such as the Starlink scandal or the recent illegal GE rice scandal in the US could have catastrophic impacts on the marketability of GE crops.[58]

In June this year, new research published in the leading scientific journal *Nature* revealed serious flaws in the science behind genetic engineering. The research calls into question the assumption that each DNA sequence can be isolated and has its own function. Instead, genes operate in a complex network where they react, interact and overlap with each other in ways that are still far from being understood. This new research shows that genes cannot be considered isolated units - nor can they be controlled. The research raises serious questions about the safety of GE crops.[59]

This incomplete understanding of genetics explains why so many unexpected effects have occurred in GE feeding studies. For example, the attached peer reviewed study, published this year, found evidence of liver and kidney toxicity when rats were fed an approved GE maize variety (MON863).[60] Similar effects were observed when Monsanto fed its GT73 Roundup Ready canola variety to rats. The rats showed a 12-16% increase in liver weight, yet Food Standards Australia New Zealand (FSANZ) still rubber stamped the canola as safe for human consumption.[61]

In 2005 CSIRO abandoned a decade-long project to develop GE peas after tests showed they caused allergic lung damage in mice. [62] The allergic reaction is believed to have been caused by unexpected changes to the protein when it was expressed in the pea. FSANZ typically uses proteins expressed by bacteria in its toxicity studies, rather than proteins isolated from the plants in which they are expressed.[63] This allergenic pea would therefore have been approved for human consumption had it gone through FSANZ's normal testing regime.

Greenpeace does not believe that any potential benefits promised by the technology could ever outweigh the potential risks posed by the technology to human health, the environment and the economy. Furthermore, most of the purported benefits of GE crops such as drought and salt tolerance can be achieved by other techniques which don't pose the same risks to human health and the environment. For example, last year John Brumby, the then Victorian Minister for Innovation, announced that Victorian scientists had developed non-GE drought tolerant canola using marker assisted selection.[64]

2.2. Segregation is impossible

"GM crop agriculture is incompatible with other forms of farming—non-GM and organic, for instance—because GM crops contaminate and because segregation is impossible." Canadian National Farmers Union (2005)

The introduction of GE canola would require the implementation of segregation and identity preservation (IP) in order to serve market demand. In 2005 Ian MacDonald, the NSW Agriculture Minister, argued that "at this point the lack of segregation trials means that there has been no practical demonstration of the capacity to segregate GM and non-GM product across the supply chain to differing market standards." [65]. This is still the case. Greenpeace believes that the moratorium should be extended, since no satisfactory measures have been suggested that would protect non-GE farmers and consumers from unwanted GE contamination.

A Western Australian Parliamentary inquiry into genetic engineering formed the view that “contamination of non-GM crops by GM crops is inevitable, segregation is not practical and that identity preservation (IP) can be achieved, but at a significant cost.”[66] The WA inquiry found that “extra costs will arise with an identity preservation system due to the additional work involved throughout the supply chain, including in growing, handling, storage, transport, processing, cleaning and administration. Certification and/or testing of the GM status of bulk commodities in the marketing chain and labelling will also contribute to the additional costs.”[67]

Europe currently has a 0.9% threshold for GE contamination. However this is for “adventitious or technically unavoidable presence”, not a legislated tolerance threshold. A 'zero tolerance' segregation system would therefore be required to serve EU markets. The Australian Bureau of Agricultural and Resource Economics (ABARE) has noted that, “zero tolerance in an importing country for contamination with GM canola would make it very difficult, if not impossible, for a country producing a mix of GM and non-GM canola to address that market.”[68]

Segregation advocates point to organic growers who successfully segregate their crops from the rest of the food supply. However such comparisons fail to appreciate how segregation systems work. Keeping the general pool of product from contaminating a small subset is a very different task to trying to keep grains separate within the commercial system, with its huge bulk-handling facilities, intermixing, port blending, sketchy paperwork, and numerous delivery points – to say nothing of pollen drift and seed contamination.[69]

The experience in countries that have adopted GE food crops has shown that the contamination of non-GE and organic crops would be inevitable. The introduction of GE canola would result in greatly increased on-farm costs for both GE and non-GE farmers; lost market access; as well as greatly increased costs for user industries; which would ultimately be passed onto consumers.

Initial attempts to segregate non-GE canola in Canada failed and it is now nearly impossible to grow non-GE canola in most of Canada. The proliferation of GE canola, uncertainty over seed supply purity, and the risk of contamination from windblown pollen mean that non-GE farmers have little certainty that their canola will be free of GE seeds. If these farmers try to grow non-GE canola, they face huge risks that their products may be rejected by buyers, possibly when those products reach overseas ports.[70]

Based on the North American experience, it is virtually guaranteed that a GE/non-GE segregation system will fail. Canadian researchers tested 33 samples of certified non-GM canola seed and found that 32 samples were contaminated with GE varieties—and three of those samples had contamination had levels above 2%.[71] Another study in the US found that virtually all samples of non-GE corn, soybeans, and canola seed were contaminated by GE varieties.[72] Widespread contamination is not surprising. A recent UK study found that GE canola cross-pollinated with non-GE canola more than 26 km away.[73]

Peter Portman from CBH (the largest grain handler for export in Australia) has stated that segregation is only likely to take place for a couple of years for 'political reasons'. The lifting of the moratoria would remove choice for both food producers and consumers with a preference for non-GE ingredients.

3. Additional Measures

In addition to the extension of the moratorium, Greenpeace believes that strict liability legislation and tighter labelling laws are needed to protect markets and consumers from unwanted GE contamination.

3.1. Strict liability legislation

It is inequitable to allow biotech companies to privately reap profits and not require that they also assume all costs. The State Government must hold biotech companies accountable for the costs their products create for farmers, industry and the consumers. Introducing strict liability legislation so that companies are held responsible for the damage caused by their products would be an equitable way to achieve this.

3.2. Labelling

Consumers should have the right to choose whether or not they want to eat GE food. Polls consistently show that the overwhelming majority of consumers want GE food to be labelled as such. Food derived from GE crops, including products from animals fed GE feed should be clearly labelled as such.

Conclusion

Greenpeace believes that the reasons for a moratorium on commercial GE food crop production are stronger than ever, and that the current moratorium should be extended by a further 5 years.

Appendix A: Dairy company policies on GE ingredients

Company	GE-Policy
Attiki	"We do not use any GE ingredients in the manufacture of Attiki yoghurt or cheese. All of our milk purchases are made through the Dairy Farmers Co-Op, which has assured us that feed used contains no GE products. All other ingredients used in the manufacturing process have been certified GE free". November 2002
Bega Cheese	"It is our policy not to use any ingredient containing GMO's in our manufacturing process. The process is managed through our Ingredient Supplier Assessment Program which requires the supplier to declare products to be supplied are from non GM sources. To manage the possible impact of GMO's in the milk supply chain it is our policy not to accept milk from transgenic or cloned cows from our suppliers and to limit the possible use of GM feed to the lowest practicable level. This is facilitated by a declaration from the milk suppliers that includes a commitment to ensure that the cow's nutritional diet will not contain more than 1% GM sourced or possibly contaminated feed." Elvis Amair, Technical Services Manager, July 2003."
Dairy Farmers	"Dairy Farmers policy is to exclude all genetically modified ingredients. This includes both those ingredients that contain modified protein or novel DNA and those ingredients that are derived from genetically modified plants, but do not contain modified protein or novel DNA. Some Dairy Farmers products are manufactured using milk sourced entirely from cattle in South Australia and Victoria where, to the best of our knowledge, no cottonseed is used for supplementary feeding. In these regions, when supplementary feeding is required, Canola Meal is used instead. At this stage, this product is GM free (Dairy Vale, Coon, Shape Cheese, Take Care). For these brands, we can be confident that the milk supply is not from cattle fed on genetically modified crops. Dairy Farmers is not able to guarantee that material from genetically modified crops is not currently in use as a feed in Queensland and New South Wales. Dairy Farmers will work with its suppliers and with the suppliers of stockfeed to identify any sources of supplementary feed that may contain material from genetically modified crops and to phase out the use of supplementary feeds containing material from genetically modified crops." July 2003"
Jalna Dairy Foods	"We declare that all our products are made using only non-GE ingredients; non GE derived ingredients; and non-GE feed." Jeff Carlin, Promotions & Business Development Manager 17th June 2003"
Lactos	"..use no GE ingredients or GE-derived ingredients in the manufacture process." Heidi Behrens, July 2003. "GM livestock feed will not be permitted in Tasmania outside of research and physical containment facilities." Tasmanian Government Gene Technology Policy 2001. "Lactos' commitment to the use of GE-free stockfeed has been communicated to all of our milk suppliers." Michel Duleu-Burre, Managing Director July 2003.
Murray Goulburn Co-operative	"Our "MG Milkcare" program specifically excludes the use of GM feed materials for the production of milk to Murray Goulburn. Our farmers are required to provide a declaration showing that feeds used does not contain GM products. Our policy is also very clear against the use of GM modified animals or cloning of animals for milk production. We do not use GM ingredients or GM additives in the manufacture of our dairy products."
National Foods	"National Foods Ltd has a contractual requirement with supplier farmers that raw milk supplied to the company comes from cows which themselves are not genetically modified and have not been fed rations from crops using recombinant DNA technology." Ian Greenshields - Group General Manager 09/04/02"

Norco Co-operative	Norco does eliminate ingredients derived from GE crops which constitute an important aspect of the food... Norco Rural Stores has a policy (where) stockfeed products either purchased-in or manufactured in-house are derived from grains which do not contain genetically modified technology. All bought stockfeed be sourced from non GE crops and not to contain GM products.
Parmalat Australia	"In relation to genetically engineered or genetically modified (GE/GM) materials, Parmalat has a clear policy to avoid their use whenever possible. Accordingly, we require from all our suppliers, warranties in regard to the GE/GM status of ingredients and additives. For our dairy products, fruit juices and carbonated drinks, none of the ingredients or additives used are GE/GM. For our soy products, the soy beans used to make the soy protein isolate are from non-GE/GM soy beans under a strict "Identity Preservation" system... In the case of milk, our preference is that our farmers avoid GE/GM feed for their cows and seek warranties from their suppliers of compounded feeds when in doubt." Dr Roger MacBean, Technical Manager Australia Asia, February 2003
Snowy Mountains Organic Dairy	"It is our policy that products containing, or suspected of containing, derived or produced using GE methods are not used. The feed that is given to the organic dairy herds is produced on [the] farms and is grown organically, without the use of GE ingredients. No ingredient used in the processing facility [milk] can be traced back to a GE ingredient." Rod McCormack, Sales & Marketing Manager, June 2003."
Tatura Milk Industries	"Tatura Milk Industries is committed to ensuring that all ingredients through the food chain are GE free. The Tatura Quality Milk Program (TQMP) requires our farmers to exclude the use of GE feed materials for the production of milk to Tatura Milk Industries. Tatura Milk Industries actively seeks the purchase of ingredients for manufacturing purposes, which are not derived from genetically engineered crops." Andrea Farago, Quality Assurance Manager, July 2003."
Warrnambool Cheese & Butter Factory	"Our policy is (a)none of our products contain GE ingredients (b)no GE derived ingredients used (c) procedures in place, as part of the on-farm QA program, to exclude GE feedstuffs being used by supplier farmers." Steve Billington 23/7/03

Attachments

Greenpeace (2006) Spotlight on GE – the environmental impacts of GE crops.

Séralini, G-E, Cellier, D. & Spiroux de Vendomois, J. (2007) New analysis of a rat feeding study with a genetically modified maize reveals signs of hepatorenal toxicity. *Archives of Environmental Contamination and Toxicology* DOI: 10.1007/s00244-006-0149-5.

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New Analysis of a Rat Feeding Study with a Genetically Modified Maize Reveals Signs of Hepatorenal Toxicity

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Abstract. Health risk assessment of genetically modified organisms (GMOs) cultivated for food or feed is under debate throughout the world, and very little data have been published on mid- or long-term toxicological studies with mammals. One of these studies performed under the responsibility of Monsanto Company with a transgenic corn MON863 has been subjected to questions from regulatory reviewers in Europe, where it was finally approved in 2005. This necessitated a new assessment of kidney pathological findings, and the results remained controversial. An Appeal Court action in Germany (Münster) allowed public access in June 2005 to all the crude data from this 90-day rat-feeding study. We independently re-analyzed these data. Appropriate statistics were added, such as a multivariate analysis of the growth curves, and for biochemical parameters comparisons between GMO-treated rats and the controls fed with an equivalent normal diet, and separately with six reference diets with different compositions. We observed that after the consumption of MON863, rats showed slight but dose-related significant variations in growth for both sexes, resulting in 3.3% decrease in weight for males and 3.7% increase for females. Chemistry measurements reveal signs of hepatorenal toxicity, marked also by differential sensitivities in males and females. Triglycerides increased by 24–40% in females (either at week 14, dose 11% or at week 5, dose 33%, respectively); urine phosphorus and sodium excretions diminished in males by 31–35% (week 14, dose 33%) for the most important results significantly linked to the treatment in comparison to seven diets tested. Longer experiments are essential in order to indicate the real nature and extent of the possible pathology; with the present data it cannot be concluded that GM corn MON863 is a safe product.

Very little data have been published on mid- or long-term feeding studies with genetically modified plants, approved and commercialized, in equilibrated diets, given to mammals, with numerous blood and organs parameters measured (Domingo 2000; Meningaud *et al.* 2001) and only one study with the MON 863 maize in such conditions. It has been performed under the responsibility of Monsanto Company and was recently published after the authorities' assessment (Hammond *et al.* 2006). The crude data at first kept confidential were subjected to questions from regulatory reviewers in Europe, where it was finally approved in 2005. This necessitated, in particular, a new assessment of kidney pathological findings, and because the study was claimed afterwards to provide an assurance of safety (Hammond *et al.* 2006), we independently re-analyzed these data here obtained after a Court action. The whole approval was based on the statement that all the significant differences were not biologically meaningful. To assess this hypothesis, we wanted to link the statistical differences per organ and to apply new methods of analysis. This transgenic maize was modified to produce in its cells a new artificial insecticidal and modified toxin Cry3Bb1 (49-97 μg/g) that was exempted from subchronic toxicity *in vivo* studies (Hammond *et al.* 2006), and its mechanism of action is not known in mammals, because it was not tested, and the target receptor has not been characterized precisely in insects.

Most, if not all, of the commercialized genetically modified organisms (GMOs) in open fields contain pesticide residues that they tolerate and/or produce (Clive 2006). Regulatory rules do not require 3-month tests with three mammalian species, then with a mammal for 1 year and yet another for 2 years, such as those employed for the testing of pesticides or drugs. This is why it appears crucial to analyze carefully the longest toxicity tests available only in one mammalian species, where numerous parameters have been measured for 400 rats, according to Organisation for Economic Co-operation and Development (OECD) standards during only 90 days. Other independent studies over 8 months with mice fed a GM Roundup tolerant soy were very detailed but only at an ultrastructural level, and showed nuclear transcription abnormalities in hepatocytes during the feeding (Malatesta *et al.* 2002), in pancreas (Malatesta *et al.* 2003), and testes (Vecchio *et al.* 2004), and hypothesized that these changes might be due to Roundup herbicide (Monsanto) toxic effects, similar to those observed on mammalian cells (Richard *et al.* 2005), but

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the parameters measured in these longest toxicity tests published on GMOs did not concern almost all organs and blood and urine chemistry, as in the present experiment.

Materials and Methods

Biological Context: The In Vivo Protocol of Monsanto

All OECD standards were claimed to be followed by the Monsanto Company: individual cages, animal randomly distributed in each group after a 1-week stabilization period, standard and validated measurement methods, and so on. This feeding study served to authorize the MON863 maize by the European and American authorities. It included young adult Sprague-Dawley-derived rats (Crl:CD®(SD)IGS BR, Charles River laboratories, NY), approximately 6 weeks old separated in 10 groups of 20 males and 10 of 20 females analyzed in details (organ weights and histology), but the biochemical parameters were measured only for half of these at weeks 5 and 14. For each sex, two groups were fed with GMOs, one with 11% and the second with 33% of MON 863 in the equilibrated diet, and two with the closest control line and regimen, grown in the same location (Hawaii), called control herein, indicated to be substantially equivalent (Hammond *et al.* 2006), in similar proportions. The closest control plant possible will then be the equivalent isogenic or parental nontransformed line, grown in similar conditions. In this article, the control is called the LH82 × A634 line. The six other groups were given diets without GMOs but that did not have the same final chemical composition, even if these diets also met PMI specifications for Certified 5002 Rodent Diet. They contained 33% of conventional different maize lines (MON 847 Repl, Asgrow RX-770, LH235 × LH185, LH200 × LH172, B73Ht × LH82, Burrus BX-86). These were not grown in the same locations (Illinois or other places in Hawaii), and were not demonstrated to be substantially equivalent to the GMO and control diet, but were supposed to mimic the variability of regular reference regimens, called reference herein, and other details have been described (Hammond *et al.* 2006).

The genetic modification in the maize tested here was inserted by chance by particle bombardment in the plant genome of immature cells. This may cause insertional mutagenesis effects, which may not be directly visible by compositional analysis; the latter can then be only partially compared for a nonexhaustive list of substances to conventionally bred lines, for instance, to test "substantial equivalence." The genetic construction itself comprises a transgene with an ubiquitous adapted 35S promoter encoding a modified toxin directed against the coleopteran insect Diabrotica. This dangerous parasite was probably introduced several times by airplane in Europe from the late 1990s (Miller *et al.* 2005). The problem apparently has been anticipated by the first trials of MON 863 or similar GMOs in Europe. This maize also contains a neomycin phosphotransferase II marker gene, coding for antibiotic resistance, to facilitate the selection of the transformed plants.

Statistical Methods

The present feeding experiment was designed and statistically assessed by Monsanto Company (St. Louis, MO), but animals were analyzed by Covance Laboratory (Vienna, VA). We first repeated the same statistical analysis as that of Monsanto to verify descriptive statistics (sample size, means, standard deviations) and one-way analysis of variance (ANOVA) by sex and by variable. For that, the normality of the residues was tested using the Shapiro test and the homoscedasticity (homogeneity of the variances) using the Bartlett test. In the case where the Shapiro and Bartlett tests were nonsignificant ($*p > 0.05$ and $**p > 0.01$, respectively) we performed an ANOVA, and in the case of heteroscedasticity the approximate Welch method was used. In the case where the Shapiro test was significant, we performed the Kruskal-Wallis rank sum test.

In addition, we undertook a multivariate analysis of the growth curves and the consumption of the rats. For the weight growth curve of the rats, after linear regression, the weekly relative increase rate can be considered proportional to the logarithm of the weight, and thus we used a Gompertz model (Ratkowsky 1990; Huet *et al.* 2004), $Y = a \cdot \exp(-\exp(-b(X-c)))$. The parameter a represents the top of the curve, b is related to the growth rate, and c is a position parameter with the X axis. These parameters were estimated by nonlinear regression. In order to see whether the growth curves are significantly different, we compared the models by testing the null hypothesis (which would give the same curves with identical parameters for both groups) against the alternative (different curves). For that, we used the F test to compare the sum of square errors under the two hypotheses. The Akaike's Information Criteria (AIC, Akaike 1974) was also used to evaluate the probability of differences.

We then analyzed the GMO effects for each sex and each diet by pairwise comparisons of the parameters of GMO-fed rats to the control groups and after to the reference groups. In order to select the appropriate two-tailed comparison test (Crawley 2005), we again studied first normality (Shapiro test) and variance equality (F test). According to the results, we performed the adapted test, i.e., an unpaired t test, a Welch corrected t test or a Mann-Whitney test (which is generally more appropriate with a sample size of 10).

We used the R language (Crawley 1995) version 2.2.1 for statistical computations (Comprehensive R Archive Network, CRAN - http://cran.r-project.org), except for the weight growth curves statistical study, for which nonlinear regressions were performed using GraphPad Prism (version 4.02 for Windows, GraphPad Software, San Diego, CA, www.graphpad.com).

Results

We first checked all the crude data, and we noticed a concordance for descriptive statistics (sample size, means, standard deviations) and one-way ANOVA by sex and by variable between our calculated values and those published by Hammond *et al.* (2006) from Monsanto Company.

Body Weights

Our study consisted of a multivariate analysis of the growth curve and the consumption of the rats for the four groups receiving GMOs or equivalent diets. If the animal consumption was not noticeably changed, it appeared for the growth curves that the variations for the two controls for each sex are superimposed, whereas the GMO feeding trials provoked different growths (Fig. 1). The 11% GMO groups were always under the 33% groups for both sexes. All the males are growing less than the controls from week 2, and all the females more. This sex- and dose-related effect resulted in the fact that the growth variations of the 11% GMO males are highly statistically lower than their controls, and 33%-GM fed females higher (Table 1). All p values of different groups versus controls are <0.01. This results in 3.3% decrease in weight for males and 3.7% increase for females.

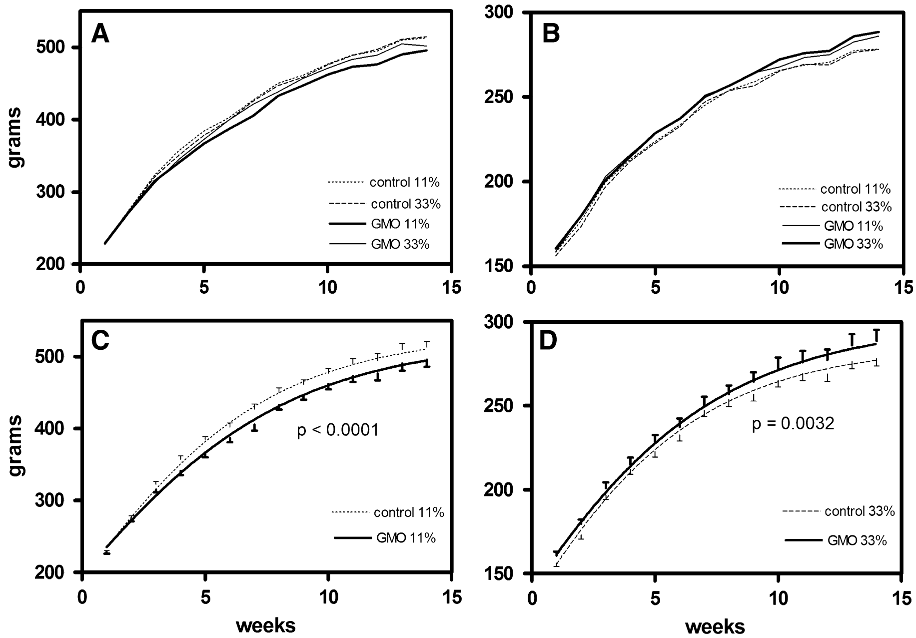

Fig. 1. Body weight growth for males **(A, C)** and females **(B, D)** over a period of 14 weeks. The experimental (A, B) and corresponding theoretical curves according to Gompertz models (**C, D**) are presented. The most important effects in each sex are in bold lines and statistically different from controls (see Materials and Methods)

Table 1. Statistical differences between weight curves

a

	Gompertz models for males		
Par.	Control 11%	GMO 11%	One model
a	533.6	524.6	528.8
b	0.2240	0.2011	0.2126
c	0.1251	−0.0939	0.0185
	Gompertz models for females		
	Control 33%	GMO 33%	One model
a	286.1	300.1	292.9
b	0.2272	0.2016	0.2142
c	−1.185	−1.376	−1.282

b

Sex	*F* test	AIC
Males	$p < 0.0001$	Prob. > 99.99%
	$F = 11.73$	Diff. 28.34
Females	$P = 0.0032$	Prob. = 98.04%
	$F = 4.66$	Diff. 7.83

The parameter estimates for Gompertz models have been calculated (a) for parameters (Par.) a, b and c and tested for statistical differences (b, F test column 2) with p values and the F ratio. The Akaike's Information Criteria (AIC) and the probabilities (Prob.) for differences (Diff.) in curves are precised (b, column 3).

Other Parameters

We then studied first the GMO effects in comparison to the isogenic, nontransgenic, equivalent maize (control) in Table 2, then the effects of different nonequivalent maize compositions on rat physiology (six different reference groups versus controls. Finally, we studied the GMO effects versus all different diets (double frame, Table 2). In total, 58 biochemical parameters reflecting most physiological functions were measured two times (week 5 and 14), in particular through serum and urine chemistry, and hematology. Organ weights and relative ratios were added. We thus performed 494 comparisons: 40 differences (8%) were statistically significant (*$p < 0.05$); 25 would have been expected under the global null hypothesis of no differences between GMO and control diet effects. Among the 40 significant differences, we retained only the 33 with a relatively ≥±5% difference to the mean; this most probably also excluded potential incidental differences, if any. Table 2 summarizes only the list of significantly disturbed parameters at least for one sex or one treatment, and also shows the percentage of variations of the means. The same Table 2 is obtained if we systematically use the

Table 2. Differences between GMO−fed rats and controls

	Week	m 11%	m 3 %	f 11%	f 33%
Liver parameters					
Albumin/globulin ratio	5	11*	-3	-9	4
Albumin/globulin ratio	14	6	-2	-18**	7
Albumin	5	-3	-2	-2	5*
Albumin	14	-2	3	-6*	5
Globulin	5	-12*	2	9*	1
Globulin	14	-8	7	15*	-2
Alanine aminotransferase	14	-30*	-8	37	4
Total protein	14	-5*	5*	1	3
Triglycerides	5	22	-2	-11	40**
Triglycerides	14	15	-1	24*	6
Liver weight	14	-1	-2	7**	6
Liver/brain ratio	14	-1	-3	6*	4
Kidney parameters					
Creatinin	14	-7	13*	13*	-2
Urine sodium	14	-23	-25*	11	-26
Urine sodium excretion	14	3	-35*	35	-24
Urine chloride excretion	5	35	3	50*	67*
Urine potassium	5	35*	-20	-3	-13
Urine phosphorus	5	3	-35*	24	-15
Urine phosphorus	14	-34	-31*	12	-8
Urea nitrogen	14	-8	4	17*	-1
Kidney weight	14	-3	-7*	3	2
Kidney/brain ratio	14	-3	-7*	1	1
Kidney % body weight	14	-1	-5*	-1	-1
Pancreas					
Glucose	14	-4	9	9*	10**
Bone marrow					
Neutrophils	5	5	22*	-14	3
Eosniophils	14	32	54*	20	0
Reticulocytes	14	15	-17	-35	-52*
Reticulocytes % RBC	14	16	-16	-36	-55*

Study of the GMO effects indicated by mean differences (%) for each parameter with the corresponding control group per sex and per dose. The significant differences *versus* controls (*p < 0.05, **p < 0.01), for all the parameters measured in the subchronic feeding tests, are presented. The parameters were grouped by organs according to the sites of synthesis or classical indicators of dysfunction. They were indicated for all groups only if they showed at least for one sex or one diet a significant and relatively ≥ ± 5% difference to the mean. The animals were male (m) or female (f) young adult rats fed during 5 or 14 weeks with GMO (MON 863, 11 or 33% in the diet) and compared with controls fed with a "substantially equivalent" isogenic maize line (LH82 × A634) grown in the same location (Hawaï). The parameters were measured for 10 rats, except for the organ weights (20 rats), obtained only at the end of the experiment. In single−boxed numbers, we indicate the statistical differences between GMO−fed rats and controls, which are not found between the mean of the six reference groups and controls. A difference between reference and control groups could indicate an effect of the diet per se. In double−boxed numbers, among the effects due to the GMO, are indicated the statistical differences between the GMO groups and the mean of the six reference groups (which have not even eaten the same composition as the control and the GMO treated groups).

Mann-Whitney test for all the biological parameters, except for albumin–14–f11%, urine phosphorus–5-m33%, and urea nitrogen–14-f11%; the *p* values in this case are comprised between 6.3% and 10.6%; these were not considered below. Table 3 corresponds to physiological values of the significantly disturbed parameters in GMO-fed rats in comparison to their corresponding controls. It emphasizes the impressive quantity of abnormalities.

Table 2 indicates that GMO-linked variations in comparison to controls were concentrated mostly on five male and nine female liver parameters, and nine and four kidney parameters for males and females, respectively, on all organs studied. We then measured the significant variations between the six reference groups and controls (isogenic to GMO), which allowed us to study the potential effects of the diet composition alone. The parameters that were also disturbed in this case were deducted from the first ones, and still three and five liver parameters and seven and one kidney parameters at least appeared to be specifically linked to the GMO diet. We consecutively compared the parameters of GMO-fed rats to the six reference groups given other diets, focusing on the GMO effects as being more important than any other diet effects, and

Table 3. Effects of GMO treatments classified by organs

Parameters	Week	Sex	Dose	Control mean ± sem	GMO mean ± sem	Units
Liver parameters						
Albumin / Globulin Ratio	5	m	11%	1.782 ± 0.053	1.974 ± 0.043	Ratio
Albumin / Globulin Ratio	14	f	11%	2.334 ± 0.085	1.914 ± 0.083	Ratio
Albumin	5	f	33%	4.600 ± 0.054	4.850 ± 0.056	g/dl
Albumin	14	f	11%	5.130 ± 0.104	4.830 ± 0.091	g/dl
Globulin	5	m	11%	2.450 ± 0.090	2.150 ± 0.072	g/dl
Globulin	5	f	11%	2.110 ± 0.041	2.300 ± 0.080	g/dl
Globulin	14	f	11%	2.220 ± 0.080	2.560 ± 0.097	g/dl
Alanine aminotransferase	14	m	11%	67.100 ± 11.078	47.300 ± 1.422	u/l
Total protein	14	m	11%	7.140 ± 0.092	6.810 ± 0.099	g/dl
Total protein	14	m	33%	6.860 ± 0.090	7.1778 ± 0.112	g/dl
Triglycerides	5	f	33%	39.300 ± 1.578	54.900 ± 3.743	mg/dl
Triglycerides	14	f	11%	40.900 ± 3.889	50.900 ± 2.479	mg/dl
Liver weight	14	f	11%	7.250 ± 0.116	7.789 ± 0.163	g
Liver / brain ratio	14	f	11%	3.664 ± 0.059	3.890 ± 0.085	Ratio
Kidney parameters						
Creatinin	14	m	33%	0.520 ± 0.013	0.589 ± 0.031	mg/dl
Creatinin	14	f	11%	0.560 ± 0.016	0.630 ± 0.021	mg/dl
Urine sodium	14	m	33%	26.980 ± 3.487	20.122 ± 5.699	meq/l
Urine sodium excretion	14	m	33%	0.290 ± 0.028	0.189 ± 0.020	meq/time
Urine chloride excretion	5	f	11%	0.220 ± 0.025	0.330 ± 0.042	meq/time
Urine chloride excretion	5	f	33%	0.150 ± 0.022	0.250 ± 0.037	meq/time
Urine potassium	5	m	11%	112.210 ± 13.860	151.000 ± 10.039	meq/l
Urine phosphorus	5	m	33%	166.970 ± 24.719	108.310 ± 7.922	mg/dl
Urine phosphorus	14	m	33%	119.120 ± 13.479	81.822 ± 10.468	mg/dl
Urea nitrogen	14	f	11%	13.200 ± 0.742	15.500 ± 0.792	mg/dl
Kidney weight	14	m	33%	3.446 ± 0.070	3.201 ± 0.078	g
Kidney / brain ratio	14	m	33%	1.600 ± 0.030	1.483 ± 0.034	Ratio
Kidney % body weight	14	m	33%	0.705 ± 0.015	0.667 ± 0.009	%
Pancreas						
Glucose	14	f	11%	103.300 ± 2.495	112.600 ± 3.497	mg/dl
Glucose	14	f	33%	105.300 ± 2.432	115.800 ± 2.476	mg/dl
Bone marrow						
Neutrophils	5	m	33%	0.860 ± 0.058	1.050 ± 0.054	$\times 10^3/\mu l$
Eosinophils	14	m	33%	0.130 ± 0.015	0.200 ± 0.024	$\times 10^3/\mu l$
Reticulocytes	14	f	33%	0.085 ± 0.015	0.041 ± 0.008	$\times 10^6/\mu l$
Reticulocytes % RBC	14	f	33%	1.040 ± 0.201	0.470 ± 0.092	%

Based on Table 2, all the parameters significantly different between GMO-fed rats and corresponding controls are represented by their crude means ± SEM in exactly corresponding units. The differences were always $p < 0.05$ or <0.01 to controls according to one or two asterisks in Table 2. The controls are submitted to a substantially equivalent isogenic maize with the same diet, with all other conditions (genetic, temperature, light, space of caging, and so on) are identical. The time of exposure (weeks 5 and 14 corresponding, respectively, to 4 and 13 weeks of GMO diet), the sexes (males: m, females: f), and the dose (11 or 33% of GM Bt maize MON 863 in the equilibrated diet) are indicated.

always for males and females, respectively, four and zero kidney parameters and one and two liver parameters remained significantly different in all cases.

The significant liver changes in the 11% GMO-fed male rats that had the lowest growth rate was a total serum protein decrease (5%), possibly linked to a globulin decrease (12%). In females, the triglycerides were specifically enhanced in the animals that had liver and body weight increases above normal. In fact, triglycerides increased by 24–40% in females (either at week 14, dose 11% or at week 5, dose 33%, respectively).

At the kidney level, phenomena corresponding to urine phosphorus and sodium excretions diminished in males by 31–35% (week 14, dose 33%) for the most important results significantly linked to the treatment in comparison to seven diets tested, whereas other diets enhanced sodium excretion in some instances (data not shown).

Moreover, for males, none of these significantly changed parameters were similar to the variations due to the composition of the diet. The effect of the GMO diet was concomitant with a kidney weight decrease.

Other sporadic effects on serum glucose, urine chloride excretion, or reticulocytes, depending on the sex or the dose, are apparent.

Discussion

The statistical analysis used in the conclusion of Hammond *et al.* (2006) was only carried out for this experiment by the Monsanto statistics center. The goal of this experiment is to study the possible toxicological effects of introducing the genetic construction producing an insecticide into the maize; thus, it should be guaranteed that the only variability sources in the results are related to the presence, or not, of this transgene apart from purely random effects. In a sense, the presence of the 6 reference groups fed with other commercial varieties of corn, which are not substantially equivalent (with more or less salts or sugars), introduces the simultaneous study of other parameters. Moreover, the reference groups representing 60 rats per sex, measured for their biological parameters, have been compared to 10 rats fed with 33% GMO, by Monsanto. We think that this difference in size favors the uncertainties. We thus preferred to separate the analysis first between the GMO groups and the control ones, and then between GMO groups and the reference groups, in contrast to Monsanto analysis.

Moreover, a study with 20 animals per group already has a limited power of discrimination. Consequently, we could consider possible toxic effects if several parameters are disturbed for the same organ in a non-negligible manner. Unfortunately, besides controls and references, only 40 rats per sex in a total of 400 animals have been given GMOs in this study, and only half of those have been analyzed for biochemical parameters, *i.e.*, 10 per dose and per sex after 5 and 14 weeks, as indicated.

The body weight growth variations, usually hardly modified by a normal diet with very little quantities of toxin, represent an important factor to follow. This study was absent from the statistical report of Monsanto. The significant variations were not tested by Hammond *et al.* (2006), although the 11% GMO males form the lowest curve after week 2. However, we clearly proved very significant differences in weight growths for both males and females, with a lower effect with the 11% diet in comparison to 33% and controls. This increase was over controls in females with the 33% diet, and under controls for the 11% diet given to males. This may be not only an indication of the dysfunction of several organs as shown in Table 3, but also a sex-dependent effect related to endocrine disruption and/or hormonal metabolism differences. Surprisingly, sexual hormones were not measured in these regulatory tests. This could have explained some of these observations. In fact, the results of Table 2 concur with signs of possible hepatorenal toxicity with a greater kidney sensitivity in males and liver sensitivity in females. A differential sensitivity for toxicants among sexes is usual, the hepatic detoxification being hormone-dependent, for instance.

The differences were significant even if the reference diets had specific effects between them, such as 8–23% differences in liver alkaline phosphatase, alanine or aspartate aminotransferase activities, or small different sodium chloride exchanges and urine volume, probably due to different lipid or salt contents in the diets (data not shown).

The GMO-linked differences are illustrated at an hepatic level by a protein or triglyceride metabolism disruption. It is known that some hepatotoxics, such as the drug metabolite hydrazine, may cause liver necrosis and steatosis with hypertriglyceridemia in the blood (Sarich *et al.* 1996). These changes may have differential thresholds according to the sex or hormonal status, as with classical reactions to hepatocarcinogens (Castelli *et al.* 1986; Pitot *et al.* 1989). Moreover, nothing in the protocol allowed the conclusion that the 11% or 33% GMO proportions chosen in the diets were in the linear portion of a dose–response curve, after intoxication by the Bt protein, for instance. Some Bt toxins may cause human hepatotoxicity by a nonapoptotic mechanism (Ito *et al.* 2004), or hepatic lipid peroxidation in rats (Shaban *et al.* 2003). However, it should be emphasized that a pleiotropic metabolic effect due to insertional mutagenesis and independent of the new insecticide produced in the GMO cannot be excluded.

To interpret the kidney data, although we did not have access to the kidney slices after the Appeal Court, Hammond *et al.* (2006) from Monsanto published that there were small increases of focal inflammation, and tubular regenerative changes in this group, in comparison to controls. They commented on a small decrease of serum chloride. After questions from the regulators in Europe, two board-certified pathology experts, proposed by Monsanto and who re-examined the slides, concluded that a classic chronic progressive nephropathy, for which male rats are sensitive (Hard and Khan 2004), had an incidence of 18/20 in the MON863 male group, higher than in controls (14/20), even if this was not considered as relevant by Hammond *et al.* (2006). If all the data are taken together, and overall in regard to the specifically disturbed urine chemistry parameters at weeks 5 and 14 (Table 2), which were not indicated by Hammond *et al.* (2006), it could be concluded that a GM-linked male renal toxicity is observed in this work.

To explain the sporadic results observed in the blood, we have little data. However, it is known in some instances that Bt toxins may also perforate blood cells (Rani and Balaraman 1996).

In conclusion, the two main organs of detoxification, liver and kidney, have been disturbed in this study. It appears that the statistical methods used by Monsanto were not detailed enough to see disruptions in biochemical parameters, in order to evidence possible signs of pathology within only 14 weeks. Moreover, the experimental design could have been performed more efficiently to study subchronic toxicity, in particular with more rats given GMOs in comparison to other groups. Considering that the human and animal populations could be exposed at comparable levels to this kind of food or feed that has been authorized in several countries, and that these are the best mammalian toxicity tests available, we strongly recommend a new assessment and longer exposure of mammals to these diets, with cautious clinical observations, before concluding that MON863 is safe to eat.

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The environmental impacts of genetically engineered crops

A new form of pollution

GE crops are an entirely new form of environmental contamination. As living organisms, they can reproduce and once released, are very difficult to control or recall. It is nearly impossible for scientists to predict the myriad of possible changes that may occur as a result of the genetic engineering process. These changes may result in environmental impacts that simply cannot be predicted.

Despite having been approved by regulators in many countries, serious concerns remain regarding the environmental impact of GE crops. These concerns include biodiversity impacts, genetic pollution, increased pesticide use and the emergence of pesticide resistant weeds and insect pests.

There has been no assessment in Australia of the impacts of GE crops on native wildlife, beneficial insects, native plants or the ecology of Australia's agricultural areas. Likewise there is very little data available on the environmental impacts of GE crops overseas.[1]

As Keith Hayes of The Commonwealth Scientific and Industrial Research Organisation (CSIRO) has pointed out, "there are important gaps in the following areas: food-web and trophic interactions, the transfer of viral particles to other viruses, increases in the host range of viruses, fungi and other pathogens, altered farm practice and physical habitat changes. National regulatory authorities should encourage data collection and research in these areas. High consequence, high uncertainty impacts (such as the creation of new viruses) are unlikely to be satisfactorily addressed by quantitative techniques in the near future."[2]

Biodiversity Impacts

The last trial in a four-year UK study, published in the Royal Society's journal Proceedings B in March 2005, compared herbicide resistant GE canola, to its conventional non-GE equivalent. It found there were fewer seeds, bees and butterflies in GE fields. There were also fewer broad-leaved weeds, which are important because they feed insects and birds. The effects were attributed to the herbicide regime associated with the crops.[3]

While the Australian Office of the Gene Technology Regulator (OGTR) dismissed the study as 'irrelevant' to Australia because of differences in cropping areas, CSIRO pointed out that the need to conduct similar studies in Australia.[4]

Concerns have also been raised about the impacts of insect resistant Bt crops on biodiversity. Most of these crops are created by inserting a synthetic version of a gene from the soil bacterium *Bacillus thuringiensis* (Bt), so that the plants produce their own Bt toxins to destroy pests.

In its natural form, Bt has been used by organic farmers since the 1950s as a spray to kill pests without damaging non-targeted wildlife. However the toxin produced by Bt crops, such as MON810 maize, are significantly different and have been shown to be harmful to non-target insects.

A recent study, conducted over a period of 2 years in which Monarch butterfly larvae were exposed to naturally deposited pollen from MON810, showed that over 20% fewer larvae reached the adult butterfly stage than in the control group. Before this research, MON810 was regarded as containing levels of toxin in its pollen too low to cause adverse effects on non-target insects.[5] Many species of butterfly and other insects are already under threat from factors such as climate change and loss of habitat.[6] Increased stress from exposure to toxic pollen could further threaten certain species.

Other studies have shown that the use of Bt crops can exacerbate populations of secondary pests, including aphids, lygus bug, whitefly, carmine spider mite and thrips.[7] Research suggests that transgenic Bt plants could also be harmful to organisms that feed upon pests exposed to the toxins. Swiss laboratory studies, for example, have demonstrated that the mortality of Green Lacewing (*Chrysoperla carnea*) larvae almost doubled after ingesting European corn borers fed on GM maize.[8] *Chrysoperla* is a beneficial insect for pest control in organic agriculture.

Additionally, Bt crops have been shown to secrete toxins from the root into the soil where they can persist for over 200 days.[9] Bt crops may be problematic for long-term soil health since they are toxic to certain insects and are suspected of being toxic to other organisms, such as earthworms, as well.[10]

There is also a danger of pests becoming resistant to Bt toxins due to widespread exposure to the compounds in GE crops. This would deprive organic farmers of a powerful pest control mechanism and other users may switch to more environmentally damaging pesticides.

Based on these concerns, in March 2005, the Austrian Government called for the postponement of commercial growing of MON810 "until the open questions concerning a comprehensive environmental risk assessment and a suitable monitoring programme have been resolved and thresholds for the adventitious presence of GMOs in conventional seed varieties have been established".[11]

Increased Herbicide Use

Scientists have warned for years that increased reliance on a single herbicide for weed management will lead to increase in herbicide resistant weeds, and hence increased herbicide use. This conclusion has been confirmed by data from the US Department of Agriculture that shows an overall increase in pesticide use of 56 million kilograms, about 4%, since 1996 as a result of the introduction of GE crops.[12]

Dr Charles Benbrook's[13] two studies into herbicide use on Roundup Ready soy represent the most thorough examination of chemical use on GE plants anywhere in the world. Based on nine years of United States Department of Agriculture figures, he calculated changing levels of chemical use on GE plants since GE was planted in the United States. He found that after an initial 3 year decline in herbicide use, application rates of glyphosate in herbicide resistant (HR) weed management systems have jumped sharply and that herbicide use on GE varieties of soy now exceeds the amounts used on conventional varieties.[14] Even the biotech companies have recognised emerging resistance by increasing recommended doses of herbicide for certain weeds. In the US, the recommended application rate for glyphosate use on certain weeds has increased by 50% to 200% since 1990.[15]

Genetic Pollution and 'Superweeds'

"Resistance to glyphosate has emerged as a serious concern across most of the intensively farmed regions of the US. The number of resistant weeds and their rate of spread is not surprising given the degree of selection pressure imposed on weed populations by farmers applying glyphosate herbicides multiple times per year, and sometimes year in and year out on the same field."
Charles Benbrook, 2004[16]

Experiences with GE canola in Canada are showing that 'super-weeds' are emerging. Volunteer canola weeds that are tolerant to as many as three herbicides (Liberty, Roundup and Clearfiled) were first identified in Canada in 1998, only three years after HR canola was first grown.[17] This resistance to more than one herbicide is termed 'gene stacking' and arises through pollination of one herbicide tolerant variety by another. An Agriculture Canada project found evidence of stacking at all 11 sites it sampled in 1999.[18]

As a result, toxic chemicals such as 2,4-D are being used to control the new weeds. The use of GE crops is also leading to the genetic contamination of seed production by GE varieties. As a result, seed production is being driven out of the prairies to other parts of North America and overseas to GE free producer nations such as New Zealand. Ultimately, contamination of crops with multiple HR seeds is likely to compromise its marketability.[19]

Herbicide resistance among other weed species is also spreading. Scientists have identified 15 weed species that are resistant to glyphosate and are warning farmers that they may become a serious problem, unless a strategy for dealing with them is developed.[20]

Herbicide resistance (HR) in weeds can occur not only as a result of prolonged exposure to the same herbicide, but also as a result of gene transfer between herbicide resistant crops and their wild relatives. Most commercialised HR crops have wild or weedy relatives in all or parts of their range, suggesting the potential for geneflow. Corn has wild and weedy relatives in Mexico and Guatemala; soybean has wild relatives in Asia; and canola has many wild and weedy relatives throughout its distribution range.[21] GE crops can also become weeds themselves when they contaminate other crops and may swamp out existing wild populations.[22] Traits such as insect or disease resistance may have even more serious effects than HR in terms of invasiveness and ecological impact.[23]

In the UK, government researchers recently discovered the first potential GE herbicide resistant superweed there - the result of GE canola (*Brassica napus*) breeding with a common weed, charlock (*Sinapis arvensis*) and passing on its herbicide resistance genes. What is most alarming in this instance is that scientists had dismissed this problem as virtually impossible. A review of the evidence by the European Environment Agency in 2000, concluded that "there appears to be general agreement that natural gene flow is not likely to occur between *B. napus* and *S. arvensis*."[24]

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