ARE ORGANIC FOODS SAFE?

W.F.T. HARTILL

DSIR Plant Protection, Private Bag, Auckland

SUMMARY

People are concerned about the acute and chronic toxicity hazards associated with pesticides and their presence as residues on food. However, few worry about the toxicity hazards of untreated crops, particularly those associated with the antifungal compounds often constitutively present in plants (inhibitins) and the metabolites formed following infection, synthesised either by the plant (phytoalexins), or by the fungus (mycotoxins). Some of the naturally formed antifungal compounds and mycotoxins that have been characterised are described. The concentration of these metabolites may be low in plants protected by fungicides. It is suggested that comparisons of the toxicological hazards of consuming food from treated and untreated crops would lead to a more rational approach in ensuring food is 'safe' than that now in use which considers only the toxicological properties of the pesticides themselves.

INTRODUCTION

Pesticides may have negative side effects on the environment, the applicator, and the consumer. In some cases these effects may be obvious, particularly when a pesticide is grossly misused. In most cases, however, the effect is small and potential hazards can be detected and quantified only through carefully controlled laboratory experimentation on test organisms. Because the hazards exist it is essential that in any benefit/risk analysis carried out to determine whether a particular use of a pesticide is justified, the advantages derived from use of the pesticide should clearly outweigh the inherent risks. The most obvious benefits are those that can be measured, such as increased yield, longer storage life, or improved appearance of the end product. There is, however, one form of benefit that pesticides provide that is almost, if not entirely, overlooked by regulatory authorities and consumers. The use of pesticides can significantly reduce the toxicological hazard constitutively or potentially present in the food we eat.

This benefit is most evident in the control of fungal diseases. There are three major sources of toxicological hazard which may arise, directly or indirectly, through the chemical expression of the interactions of fungal pathogens with their hosts. These are the mycotoxins, the inhibitins, and the phytoalexins. This paper is not intended to provide an exhaustive review of these metabolites but a few examples are described in order to provide a background to issues raised regarding the principles now used in legislating for the use of pesticides in food. For a more exhaustive review of the toxic compounds which occur naturally in food see Ames (1983).

MYCOTOXINS

Mycotoxins are secondary metabolites produced by mould and other fungi when they grow upon their hosts and stored food products. Mycotoxins are toxic to vertebrates. Historically the best known is ergotamine present in grain contaminated by ergot (Floss and Anderson 1980). In the past there were many examples of people dying unpleasantly of 'St Anthony's Fire' after consuming the contaminated grain. Disorders have also been reported in animals fed grain contaminated with bunts and smuts (Brook and White 1966). More recently the dangers of the aflatoxins, formed when *Aspergillus* spp. attack peanuts and other grains, have been of greater concern. Aflatoxin-contaminated grain is rapidly lethal to some animals, e.g. turkeys (Blount 1961) but of

Proc. 43rd N.Z. Weed and Pest Control Conf. 1990: 292-295

greater concern is its probable implication in liver cancer in Africans (Palmgren and Ciegler 1983). Aflatoxin can also be present in the milk of grain-fed cattle. In New Zealand, the most destructive mycotoxin is sporodesmin, formed when *Pithomyces chartarum* (Berkely and Curtis) M.B. Ellis invades grass litter (Brook and White 1966). When cattle and sheep ingest severely contaminated litter their livers may be damaged and, more obviously, they suffer facial eczema.

The invasion of all these fungi can be checked by the use of fungicides. The incidence of ergot can be reduced by spraying the flowering ears of grain with benomyl. The same fungicide, used as a seed dressing, can reduce the incidence of *Aspergillus* in peanuts and, used as a spray, can control *Pithomyces*.

INHIBITINS

Inhibitins are constitutive anti-fungal metabolites present in some plants for part or all of their lives (Ingham 1973). Their concentration varies between different cultivars and may provide the deliberate or accidental basis of selection for disease resistance in some crop species. They are often present in an inactive bound form and are liberated slowly or only after the plant cells are damaged by invading pathogens.

Brassicas, young pawpaws, horseradishes and a number of other plants contain isothiocyanates. Allyl isothiocyanate, and other derivatives, present in expanding cabbage and cauliflower leaves effectively prevent infection by a number of leaf spot pathogens (Hartill 1979). Their release, from inactive forms, is enhanced during the production of coleslaw. The amount of isothiocyanates normally consumed is probably harmless, but consumption of certain cabbage varieties is known to have caused thyroid damage in Dalmatia (Michajlovski *et al* 1969) and may once have been an important cause of goitre in New Zealand (Connor 1981). Allyl isothiocyanate is also carcinogenic (Dunnick *et al* 1982).

Potatoes contain a range of phenolics and alkaloids which are probably harmless when present at low concentrations. Under stress, including disease stress, the concentration of these compounds rises and they become effective in checking fungal invasion (Renwick 1972). Plant breeders have successfully produced disease- and pest-resistant cultivars by selecting for high levels of these compounds but the cultivars have been found unfit for human consumption because, at these high levels, the inhibitins are toxic (Zitnak and Johnson 1970).

These two examples, of brassicas and potatoes, indicate that the use of natural resistant cultivars is not necessarily a safer option than the use of fungicides in disease control.

PHYTOALEXINS

Phytoalexins are a more dynamic defence mechanism than inhibitins. Phytoalexins are secondary metabolites that appear in plants after the plant has been invaded, stressed, or injured. A highly selective matching system between fungus and host determines the disease reaction. In an incompatible (resistant) reaction, the rapid production of a phytoalexin halts the spread of the pathogen. In the compatible (susceptible) reaction, production of the phytoalexin is delayed and the pathogen grows ahead of the region where the defence reaction is initiated. Because the pathogen continues to stimulate but is little affected by the defence reaction, the concentration of phytoalexins can be high in susceptible diseased plants. Some of the research into biological disease control systems is aimed at stimulating the phytoalexin-mediated incompatible reaction.

There is little knowledge concerning the mammalian toxicity of phytoalexins and that which we do have is far from reassuring. Some, e.g. phaseollin from beans, and glyceollin from soyabeans, can lyse red blood cells (Kuc and Currier 1976). A group of furocoumarins, the psoralens, produced in the Umbelliferae and other plants, are strong photosensitising agents. People handling *Sclerotinia*-infected celery develop large skin blisters (Connor 1981). Parsnips contain three analogues of this group (Ivie *et al* 1981). One of the foot and mouth disease scares in New Zealand arose when pigs, fed on diseased parsnips, developed blisters in their mouths, almost certainly because of the high psoralen content of these parsnips.

Cattle have been killed after being fed diseased sweet potatoes. *Ceratocytis fimbriata* (Ellis & Halsted), the pathogen responsible for the disease, induces the formation of several phytoalexins including two hepatotoxins, ipomeamarone and ipomeamaranol, and a group of respiratory toxins including 4-ipomeanol (Wilson and Burka 1983).

Potatoes produce a range of phytoalexins in addition to the glycoalkaloids described earlier. Renwick (1972) found a correlation between the consumption of potatoes and the incidence of spina bifida and suggested that enhanced concentrations induced by late blight were a major factor in this association. Although later evidence did not support the theory of a close correlation with spina bifida there remained evidence of a more general relation with birth defects (Renwick *et al* 1974). There are many examples of direct, i.e. acute, toxicity where potatoes with high levels of antifungal metabolites have been eaten by humans and animals (Kuc and Currier 1976).

DISCUSSION

The concentration of inhibitins and phytoalexins in the food we eat can be controlled by selecting cultivars with inherently low levels of the metabolites and by protecting them with fungicides. If we had complete information on the toxicology and dynamics of the components of disease management schemes, whether based on the use of fungicides, or on their avoidance, we could choose the one that offers the least threat to human health. The authorities that regulate the use of pesticides ensure that the toxicological hazards of each new chemical is carefully studied before it is released. Some of the compounds released prior to 1970 were subjected to less rigorous screening than occurs now but most, if not all, of these are now in the final stages of re-evaluation. This position contrasts markedly with our knowledge of the toxicology of metabolites associated with fungal attack. I believe our state of knowledge of these metabolites is, at best, similar to our pre-1950s ideas on the side effects of fungicide use, i.e., with few exceptions, studies have been confined to the metabolites having obvious debilitating effects, such as human or stock animal poisonings. The data that have raised doubts about the use of fungicides have been obtained from controlled experiments mainly on carefully bred laboratory animals. Similar studies have been carried out for some of the mycotoxins but not, apparently, for the metabolites associated with anti-fungal defence. Although in many host resistant reactions it is known or assumed that the basis of the reaction is probably due to a chemically-determined antagonism, the chemistry involved often has not been characterised. Even in the cases where the basis is reasonably clear, mammalian toxicological studies on the metabolites involved are rare. There is also considerable doubt as to how well studies on animals relate to the human condition, e.g. thalidomide is clearly more teratogenic to humans than to rabbits, aspirin is equally clearly less so (Maddy 1989). There is another difficulty in that some people are much more sensitive than others to specific toxicants, a fact recognised for at least 2000 years ('Ut quod ali cibus est allis fiat acre venenum'¹ — Lucretius c. 58 BC).

It is therefore impossible in our present state of knowledge to answer the question in the title "Is organic food safe?" and in this context neither can we answer the question "Is fungicide-treated food less or more safe?" Each question reflects upon the other with no "safe-food" baseline to guide us. Only when feeding studies are based on a comparison of treated versus untreated crop plants will it be possible to choose the least hazardous option. The present procedure of merely adulterating a standard food with the potential toxicant cannot provide an answer because it takes no account of the dynamic processes that occur during fungal invasion of living plants. The regulatory authorities' inability to consider the balance between the natural and synthetic toxicants arrived at in different disease management systems is a major weakness in their attempts to protect consumers.

This line of argument is not an appropriate one to take for herbicides because weeds are not known to elicit the same defence responses in crop plants (though the stresses they cause through competition for space and nutrients may have similar

¹Colloquially = one man's meat is another man's poison.

effects). There is some justification to consider it in relation to insecticides since plants have metabolites, e.g. the protease inhibitors, myristicin in carrots, that defend them against insects and which are potentially toxic to man. But with fungicides the paradox that the use of a potentially toxic material can result in the production of food that is safer to eat than that produced by "organic" methods is, I believe, an important one which makes nonsense of such statutes as the Delaney amendment (Anon 1987).

REFERENCES

- Ames, B.N., 1983. Dietary carcinogens and anticarcinogens. *Science* 221: 1256-1263.
- Anon., 1987. Regulating pesticides in food: the Delaney Paradox. Committee on Scientific and Regulatory Issues underlying pesticide use patterns and agriculture innovation. Board of Agriculture, National Research Council. Washington D.C.: National Academy Press. 272 pp.
- Blount, W.P., 1961. Turkey 'X' disease. *J. Brit. Turkey Federation* 9: 52, 55-88.
- Brook, P.J. and White, E.P., 1966. Fungus toxins affecting mammals. *Ann. Rev. Phytopathol.* 4: 171-194.
- Connor, H.E., 1981. The Banks Lecture, 1981 Poisonous plants and horticulture in N.Z. *A. Jl. R. N.Z. Inst. Hort.* 9: 5-23.
- Dunnick, J.K., Prejean, J.D., Haseman, J., Thompson, R.J., Giles, H.D. and McConnell, E.E., 1982. Carcinogenesis bioassay of allyl isothiocyanate. *Fundam. Appl. Toxicol.* 2: 114-120.
- Floss, H.G. and Anderson, J.A., 1980. Biosynthesis of ergot toxins. In: The Biosynthesis of Mycotoxins — A Study in Secondary Metabolism. P.S. Steyn, (Ed.) Academic Press, London pp 17-67.
- Hartill, W.F.T., 1973. Effect of volatile constituents of brassicas in relation to disease resistance. *49th ANZAAS Congr. Abs.* 1: 357.
- Ingham, J.L., 1973. Disease resistance in higher plants. *Phytopathol. Z.* 78: 314-335.
- Ivie, G.W., Holt, D.L. and Ivey, M.C., 1981. Natural toxicants in human food — psoralens in raw and cooked parsnip root. *Science* 213: 909-910.
- Kuc, J. and Currier, J., 1976. Phytoalexins, plants, and human health. *Adv. Chem.* 149: 356-368.
- Lucretius (c.58 BC). *De Rerum Natura IV*: 637.
- Maddy, K.T., 1989. The fate of insecticides, miticides and other pesticides as a result of the reregistration process in the United States and the State of California and the increasing level of public concern about exposure to any pesticide. *Proc. 1st Asia-Pacific Conf. of Entomology*: In press.
- Michajlovski, N., Sedlak, J., Jusic, M. and Buzina, R., 1969. Goitrogenic substances of kale and their possible relation to the endemic goitre on the Island of Krk (Yugoslavia). *Endocrinol. Exp.* 3: 65-72.
- Palmgren, M.S. and Ciegler, A., 1983. Aflatoxins. In: Handbook of Natural Toxins 1. Plant and Fungal Toxins. R.F. Keeler and A.T. Tu (Eds.) Dekker, New York. pp 299-323.
- Renwick, J.H., 1972. Hypothesis: Anencephaly and spina bifida are usually preventable by avoidance of a specific but undefined substance present in certain potato tubers. *Br. J. Prev. Soc. Med.* 26: 67-83.
- Renwick, J.H., Possamai, A.M. and Munday, M.R., 1974. Potatoes and spina bifida. *Proc. Roy. Soc. Med.* 67: 360-364.
- Wilson, J.W. and Burka, L.T., 1983. Sweet potato toxins and related toxic furans. In: Handbook of Natural Toxins 1. Plant and Fungal Toxins. R.F. Keeler and A.T. Tu (Eds.) Dekker, New York. pp 299-323.
- Zitnak, A. and Johnson, G.R., 1970. Glycoalkaloid content of B5i41/6 potatoes. *Am. Potato J.* 47: 256.