Ochratoxin A: a new challenge for Australia's grape products industries?

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Ochratoxin A (OA) (Figure 1) was first isolated from *Aspergillus ochraceus* in 1965 in a laboratory study searching for new toxic metabolites from moulds[1]. At the time, there was no connection with any animal or human disease.

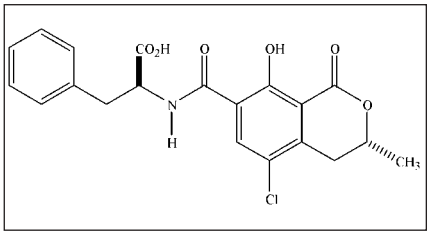
OA was found as a natural contaminant of maize in 1969 in the USA and, about the same time, studies were being conducted in Scandinavia on a kidney disease in pigs which appeared to be related to mouldy feed[2]. These studies showed that OA was the cause of the disease now known as porcine nephropathy.

Since then OA has been found as a contaminant of grains in most European countries and in northern North America. The source of OA in cooler climates is principally from contamination of small grains by P*enicillium verrucosum*[3]. Consequently, OA may be found in cereal products (including bread), pig meat products, where it accumulates in fatty tissue of pigs fed contaminated feed, and in beer if contaminated barley is used for malting.

Human exposure has been demonstrated by detection of OA in blood samples from many European countries[2,4]. OA may also be carcinogenic, teratogenic, immunogenic and genotoxic. The International Agency for Research on Cancer (IARC) in 1993 classified OA as Group 2B, a possible human carcinogen[5].

In 1994, OA production by *Aspergillus niger*, and subsequently by other species of black Aspergilli particularly *Aspergillus carbonarius,* was reported[6]. Studies in our laboratory and also from Spain[7] now recognise that *A. carbonarius* is the primary source of OA in grapes and grape products (Figures 2 & 3).

Figure 1. Ochratoxin A



This is particularly pertinent to the wine and the dried vine fruits industries as black *Aspergillus* species are very common on grapes and, consequently, their metabolites may contaminate grape products. These reports have caught the attention of both food scientists and food regulators, and there have been a number of surveys reporting the occurrence of OA in wines from mainly Mediterranean countries, including Spain, Italy, France and Morocco and also from South Africa.

Australian wines and dried vine fruits have not escaped scrutiny. An extensive survey carried out by MAFF[8] in the United Kingdom in 1998 looked at the incidence of OA in wines, grape juice and dried vine fruits. Positive samples were found in all categories. Of five Australian wines, two tested positive for OA (0.02 and 0.05 µg/L) and three were negative.

The MAFF survey also included a large number of dried fruit samples, comprising 100 currant, 101 raisin and 100 sultana samples imported into the UK from Greece, Turkey, USA and Australia. OA was detected in all but eight of the 100 sultana samples, with positive samples ranging from 0.2 to 24.4 µg/kg OA.

Of the 14 Australian sultana samples, five were negative for OA, with the other nine samples ranging from 0.1 to 1.7 µg/kg OA. Of the 100 currant samples surveyed, three were of Australian origin. Only four samples were free of OA and three of these were the Australian samples. None of the raisin samples originated from Australia. Overall, the Australian fruit compared very favourably with that from the Mediterranean.

We carried out a large survey of Australian wines for occurrence of OA during 1999-2000[9]. A total of 601 wines was assayed, sampling predominantly bottled wines, approximately equal numbers of red and white wines, wines made from a range of grape varieties and wines from all major producing areas and some minor ones.

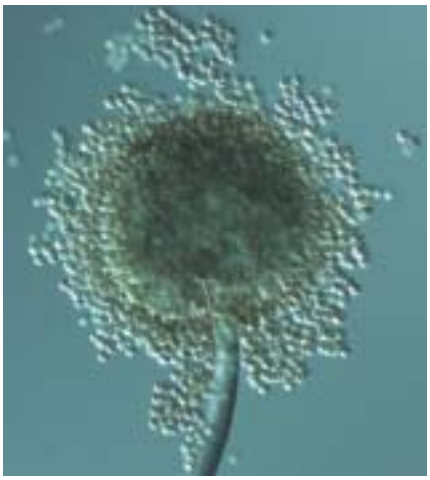
Figure 2. Sporulation of *A. carbonarius* spray-inoculated onto Semillon berries.

OA was detected in 90 (15%) of the 601 samples, but at uniformly low levels, with 85% of positive samples containing less than 0.2 µg/L OA. Only one sample exceeded 0.5 µg/L OA and no sample exceeded 1.0 µg/L OA.

Data from Europe indicate that red wines are more likely to be contaminated with OA than white wines, and that wines produced in hotter, more humid climates (such as those around the Mediterranean region) are more likely to contain OA than wines produced in the cooler regions to the north. However, these trends were not observed in our survey of Australian wines [9].

OA contamination of foods is of greatest concern in northern and eastern Europe, where the direct or indirect source is grain infected with *P. verrucosum.* However, the increasing incidence of reporting of OA from a variety of foods and beverages from both temperate and tropical countries has brought this mycotoxin increasingly into the spotlight.

Figure 3. Typical *A. carbonarius* head.



Exposure assessments for OA have been carried out in Europe where a German study found the total daily OA intake by adults and children was calculated to be 39.9 and 27.9ng respectively [4]. Cereal products were the main contributors, but coffee and beer were also found to be important sources for adults, while red grape juice and sweets were important sources for children.

Consequently, the EU has imposed the following limits for OA: 5 µg/kg in raw cereal grains, 3 µg/kg in all cereal derived products intended for direct human consumption, and 10 µg/kg in dried vine fruits (currants, raisins and sultanas) [10]. A limit for OA in wine of 1.0 µg/L is being considered but has not yet been mandated.

Exposure to OA in the Australian diet is relatively low, but potentially important sources are wine, dried vine fruits and coffee [11]. Our current research efforts are focussed on the ecology of *A. carbonarius* in Australian vineyards, the formation of OA in different grape varieties, and the fate of OA during winemaking. Understanding the origins of the mould at its source may help in the formulation of improved vineyard management and intervention strategies, and modified wine making practices resulting in even lower levels of OA in Australian wines.

References

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