FOOD STANDARDS
Australia New Zealand
Te Mana Kounga Kai – Ahitereiria me Aotearoa

2-06
22 March 2006

Attachment 2
A Risk Profile of Dairy Products in Australia

Executive Summary
Parts A and B

DRAFT ASSESSMENT REPORT

PROPOSAL P296

PRIMARY PRODUCTION AND PROCESSING STANDARD FOR DAIRY

EXECUTIVE SUMMARY

The Risk Profile of Dairy Products in Australia brings together information on microbiological and chemical risks that may be associated with dairy products. The risk profile was undertaken within the framework of existing dairy regulations and risk management practices in Australia and comprises two separate parts:

- **Part A: Microbiological Risk Profile**
- **Part B: Chemical Risk Profile**

The purpose of this risk profile is to inform the development of the Primary Production and Processing Standard for dairy products. To this end, the two risk profiles provide an objective analysis of scientific data and information to identify the public health and safety risks arising from microbiological and chemical hazards associated with dairy products.

The scope of the Risk Profile includes the identification and examination of risks along the dairy supply chain from milk production through to consumption of dairy products.

The key findings of the **Microbiological Risk Profile (Part A)** can be summarised as:

- Australian dairy products have an excellent reputation for food safety, and this is supported by the lack of evidence attributing foodborne illness to dairy products;
- A wide range of microbiological hazards may be associated with raw milk and dairy products, but these do not represent a problem under current management practices which:
  - control animal health;
  - ensure adherence to good milking practices;
  - require effective heat treatment e.g. pasteurisation; and
  - have controls to prevent post-pasteurisation contamination in the dairy processing environment.

The key findings of the **Chemical Risk Profile (Part B)** can be summarised as:

- There are extensive regulatory and non-regulatory measures in place along the dairy industry primary production chain resulting in minimal public health and safety concerns regarding the use or presence of chemicals in dairy products.
- Extensive monitoring of chemical residues in milk over many years has demonstrated a high level of compliance with the regulations.
- There are a number of areas where further research or monitoring would assist in providing further reassurance that the public health and safety risk is low.
- Continuation of the current management practices, particularly monitoring programs for chemicals along the primary production chain, will ensure that the dairy industry continues to maintain a high standard of public health and safety.

Part A - The Microbiological Risk Profile

The microbiological risk profile encompasses a detailed discussion of the following elements:

- Microorganisms that may be associated with dairy products including key attributes of each organism and its public health impact;
- Epidemiological data related to the consumption of dairy products and illness;
- An examination of prevalence and concentration data on microbiological hazards found in dairy products and along the entire dairy food chain; and
- A description of the dairy production, dairy processing, distribution and consumption chain and a description of factors that impact on the public health and safety risks associated with microbiological hazards in dairy products.

The safety of dairy products relies on the quality of raw materials, correct formulation, effective processing, the prevention of recontamination of product, and maintenance of temperature control during distribution, retail sale and storage of the product in the home.

There is relatively little data on the presence of pathogens in raw milk in Australia although it is well established that raw milk may be contaminated with pathogenic microorganisms.

Raw milk has a mixed microflora, which is derived from several sources including the interior of the udder, exterior surfaces of the animals, environment, milk-handling equipment, and personnel. In general, there are two means by which pathogens contaminate raw milk. Contamination may occur when microorganisms are shed directly into raw milk from the udder as a result of illness or disease, or through contamination from the external surface of the cow and the milking environment. Primary production factors that impact on these routes of contamination and the microbiological quality of the raw milk include:

- animal-related factors e.g. animal health, herd size, age and production status;
- environment-related factors e.g. housing, faeces, feed, soil, and water; or
- milking and operation of milking equipment factors e.g. cleanliness of equipment and lines.

Some of these primary production factors can be managed to reduce the risk of contamination of raw milk by pathogens, while management of others will have limited impact on the final microbiological status of raw milk.

Following milking, milk is transferred to the dairy processing facility where it subsequently undergoes a series of processes that transform liquid milk into a wide range of dairy products, many of which may be classified as ready-to-eat. The majority of these processes involve a heat-processing stage, typically pasteurisation or an equivalent process. Further steps involve physical processes such as separation, aeration, and homogenisation and product transformation by drying, churning, acidification, etc.

Pasteurisation represents the principal process for rendering dairy products safe for consumption. However, the effectiveness of pasteurisation is dependent upon the microbiological status of the incoming raw milk. Control of risk factors on-farm will minimise the opportunity for microbiological hazards to contaminate raw milk and reduce the likelihood and concentration of these hazards.

A survey of Australian dairy manufacturers determined that all respondents met the minimum time and temperature standards prescribed in the Code for the HTST (high temperature short time) pasteurisation of milk and cream. In many cases, milk was heated to a temperature and/or a time in excess of the prescribed minimums. For the majority of dairy products, pasteurisation also represents an initial treatment before specific processes are used to transform raw milk into various manufactured products.

Dairy products containing elevated levels of fat or solids such as ice-cream mixes, cream and yoghurt, warrant higher time/temperature combinations than those currently specified in the Food Standards Code to compensate for the protective effect of fat and solids on microorganisms.

Post-pasteurisation contamination however, is an ongoing management issue for manufacturers in the provision of safe dairy products. Contamination may result from the environment, including equipment, personnel or contamination of finished product with raw materials. Rigorous control over hygiene, cleaning and sanitation, and product handling is therefore necessary to ensure safety of the final product post-heat treatment.

As many dairy products do not undergo a further pathogen reduction step prior to consumption, prevention of contamination and control over bacterial growth, storage time and temperature is of particular importance in minimising potential exposure to pathogens. Most dairy products have a relatively short shelf-life, especially milk (10-16 days under optimum storage conditions) thus storing dairy products according to manufacturer instructions and following good hygiene and handling practices in the home is also important.

Microbiological survey data for pasteurised dairy products in Australia show a very low incidence of hazards of public health significance in these products. Overseas data demonstrates that pathogens are frequently isolated from raw milk and raw milk products. Pathogens were detected in raw milk in 85% of 126 surveys identified in the literature. In surveys of raw milk cheese pathogens were rarely detected. Pathogens are found infrequently in pasteurised milk and pasteurised milk products.

In Australia, illness from dairy products is rare. Between 1995-2004, there were only eleven reported outbreaks directly attributed to dairy products and eight were associated with consumption of unpasteurised milk. In other Australian outbreaks, dairy products were an ingredient of the responsible food vehicle identified as the source of infection. However dairy products are a component of many foods, therefore it is often difficult to determine whether t they are the ingredients in the food vehicle identified as the cause of an outbreak.

While commercial dairy products have rarely been identified as sources of foodborne illness in Australia, there have been a number of reports of outbreaks associated with consumption of dairy products internationally. Unpasteurised dairy products are the most common cause of these dairy-associated outbreaks of illness.

The microbiological risk profile has identified a range of microbiological hazards potentially associated with the Australian dairy supply chain. The majority of these hazards pose little or no threat to public health because under current risk management conditions they are unlikely to be present in high numbers in raw milk, and the pasteurisation step effectively eliminates all but the spore-forming bacteria. This is supported by the lack of foodborne illness attributed to dairy products in Australia.

While there is a lack of evidence showing foodborne illness attributing illness to pasteurised dairy products, the following organisms can be summarised as the most significant to public health and safety for the dairy industry due to their association with reported incidents of foodborne illness from dairy products and/or their potential to contaminate dairy products post-pasteurisation.

Pathogens	Significance in dairy products
Salmonella	*Salmonella* is destroyed by pasteurisation, however it can be present in the environment and can gain access to product after heat treatment. Initial source is often birds and rodents, although occasionally present in the raw milk. Non-dairy ingredients can be an important source of contamination.
Listeria monocytogenes	*L. monocytogenes* is destroyed by pasteurisation. Its presence in heat-treated products is due to post-pasteurisation contamination. *L. monocytogenes* is a concern to the dairy industry as it can grow at 0ºC (refrigeration temperatures).
Staphylococcus. aureus	*S. aureus* is destroyed by heat-treatment, however its toxins are heat stable, thus control of growth of this organism prior to heat treatment is essential. However, *S. aureus* does not grow well at low temperatures (i.e. refrigeration).
Bacillus cereus	Vegetative cells of *B. cereus* do not survive pasteurisation, however spores will survive heat treatments. *B. cereus* is rapidly outgrown by gram-negative psychrotrophs at refrigeration temperatures, but in their absence, *B. cereus*, if present, may then be able to grow to high levels. This is a concern with extended shelf-life chilled products, such as desserts.
Escherichia coli	*E. coli* is found in cattle and may enter milk through faecal contamination, however *E. coli* is heat-sensitive and does not survive pasteurisation.
***Campylobacter* spp.**	*Campylobacter* spp. is destroyed by pasteurisation and its presence in milk products is due to environmental contamination after heat treatment. *Campylobacter* spp. are fragile organisms unable to grow in foods.
Yersinia enterocolitica	*Y. enterocolitica* is destroyed by pasteurisation and its presence in heat-treated milk products is due to environmental contamination after heat treatment. *Y. enterocolitica* is able to grow in dairy products held at refrigeration temperatures and therefore may be considered as a hazard in prolonged shelf-life products.
Enterobacter sakazakii	*E. sakazakii* will not survive pasteurisation. Recontamination of powdered infant formulae during manufacture is a risk. *E. sakazakii* cannot grow in a dry substrate, but it can survive a long period of time and is a potential hazard when the powder is reconstituted and held for long periods of time at favourable temperatures. Contamination and subsequent growth may occur during reconstitution and preparation.

The factors along the Australian dairy supply chain that have the most significant impact on the safety of processed dairy products are:

- the quality of raw materials;
- correct formulation;
- effective processing (pasteurisation in particular);
- the prevention of recontamination of a product; and
- maintenance of temperature control during distribution, retail sale and storage of the product in the home.

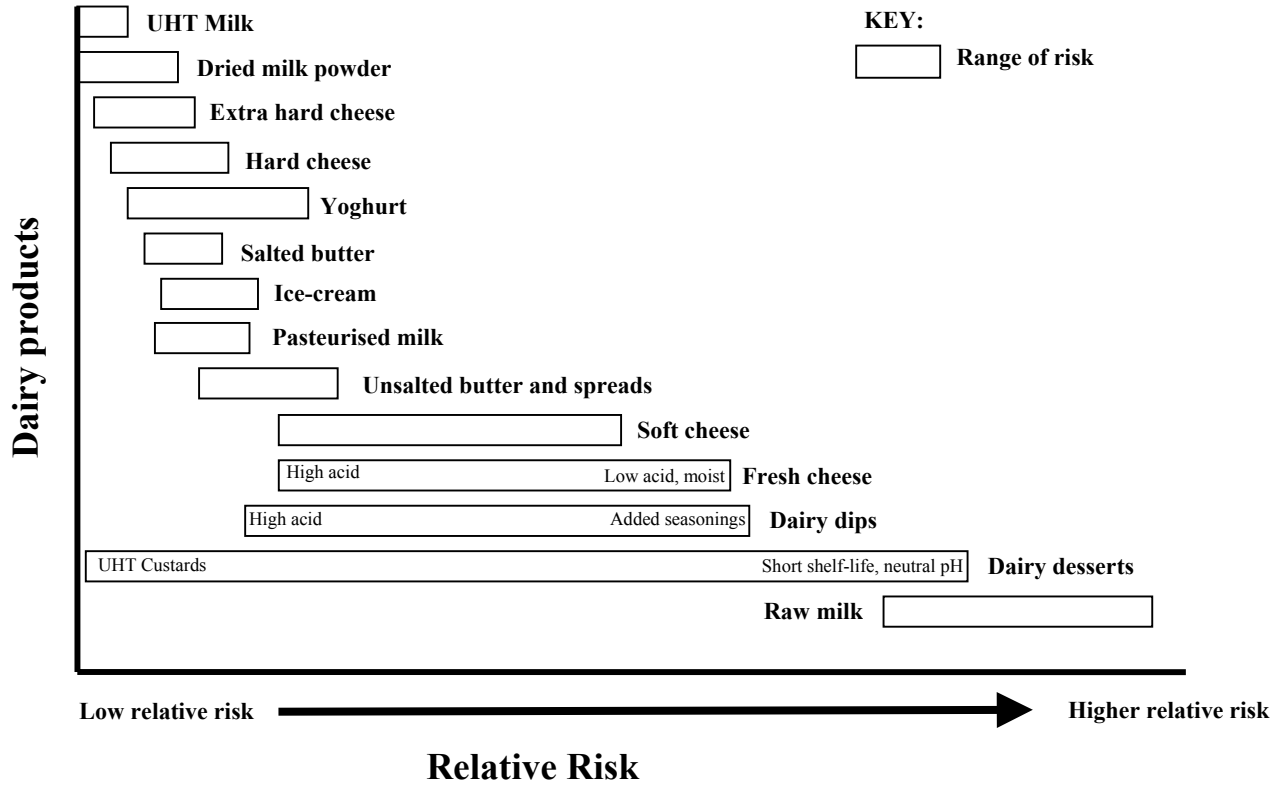
The quality of raw milk is dependent on animal health, exposure to faecal contamination, environmental contamination and temperature control.
The key risk factors that may affect the quality of raw milk on-farm can be summarised as follows:

Risk factor	Effect
Animal health	Disease in, sickness of, and carriers in milking animals can increase shedding of pathogens directly into raw milk, or in animal faeces.
Herd size	Herd size may have some effect on the prevalence of some pathogens.
Age/production status	Calves have an increased susceptibility to infection.
Housing	Intensive housing practices may increase risk of contamination of udders.
Faeces	Faeces contaminate the udder and may introduce pathogens into raw milk.
Effluent	Effluent can contaminate pasture and the animal.
Feed	Contaminated feed can lead to shedding of pathogens into faeces.
Water-stock drinking	Water sources may be contaminated with cud and/or faecal material
Milking	Poor milking practices *i.e.* dirty teats; inadequate maintenance, sanitation and cleaning of equipment; and poor personal hygiene can lead to contamination of raw milk.
Water use during milking	Potential source of contamination during washing of teats and cleaning of milking equipment.
Storage	Poor temperature control of milk after milking can lead to growth of pathogens.
Transport	Poor temperature control of milk during transport can lead to growth of pathogens. Poor maintenance, sanitation and cleaning of tankers can lead to contamination of milk.

The formulation of dairy products, effective processing, and prevention of recontamination of product all contribute to the level of risk a dairy product poses. Those dairy products, which are prone to contamination after final heat treatment and provide a benign environment, may be categorised as being of higher risk to public heath and safety, than products that don't provide a favourable environment for pathogens. The intrinsic properties of the product *i.e.* the impact of water activity, pH, salt concentration, etc., influence pathogen survival and growth as does the storage environment. The following table provides a relative rating for selected dairy products, based on the inherent stability of the product and therefore the degree of risk they may pose to the consumer.

Risk	Dairy product
Higher risk	Soft cheeses Dairy desserts
Intermediate risk	Unsalted butters
Low risk	Yoghurts Salted butters Extra hard cheeses

Qualitative objective methods of describing the relative risk to public health associated with dairy products is extremely difficult. The relative risk from dairy products, based on intrinsic properties of the product, may also be expressed graphically as a continuum:



Part B - The Chemical Risk Profile

The chemical risk profile identifies and examines chemicals introduced along the dairy primary production and processing chain, from the farm environment through to retail dairy produce. Through this whole-chain analysis, an overall picture of the current regulations and controls for chemicals associated with dairy products has been assembled and any inadequacies identified. Issues may however be identified which go beyond the backdoor of retail, and hence are not in the scope of the Dairy Primary Production and Processing Standard.

The profile considers the following:

- Agricultural and veterinary (Agvet) chemicals used in primary production;
- Environmental contaminants, including heavy metals, organic contaminants and micronutrients;
- Naturally-occurring chemicals found in plants or in fungi or bacteria associated with plants;
- Food processing by-products;
- Food additives, processing aids, and those chemicals that may migrate from packaging.

Chemicals used in primary production
Chemicals are used intentionally at the primary production stage for a number of purposes, including pest and weed control, animal health and equipment sanitization. The agricultural chemicals to which cattle are exposed may potentially leave residues. However, the Australian Milk Residue Survey (AMRA) showed that there were no detections of agricultural chemical residues above the maximum residue limit (MRL) in milk in over 33,382 analyses during the seven years of the survey. No residues of agricultural chemicals were found in milk and milk products in the Australian Total Diet Survey (ATDS) either. The low incidence of agricultural chemical residues in cattle is supported by the results of the National Residue Surveys.

Veterinary chemicals administered to dairy cattle are mainly antimicrobials and endo- and ectoparasiticides. Other veterinary chemical uses include reproductive therapy use and use of anti-inflammatory drugs or anaesthetics. During the 1998-2005 period of the AMRA surveys, 89,121 analyses were carried out for antimicrobials with 99.997% compliance with the MRL.

In order to comply with hygienic production and manufacturing practices, cleaning and sanitising agents are utilised throughout the whole production process to ensure that the products remain free from microbial or physical contamination.
The water used on-farm for both agricultural and for cleaning purposes was found to be of high quality and free from chemical contamination.

In addition to the current regulatory and non-regulatory measures in place for chemicals used in primary production, there are areas of uncertainty which have been identified where further data may be necessary to better characterise any potential public health and safety risks (summarised in the Table below):

- Colostrum, which is collected 1-2 days after the birth of a calf for the specific purpose of therapeutics manufacture, may potentially have higher concentrations of agricultural or veterinary chemical residues if critical withholding periods (WHP) before milking are not observed. Monitoring data on Agvet chemical residues would assist in addressing this issue.
- Standard dairy industry practice does not include the first eight milkings (containing colostrum) and Quality Assurance (QA) programs are in place to ensure that colostrum does not enter the milk collection and processing streams.
- There is evidence of substantial off-label usage of veterinary therapeutics for goats, in particular for anthelmintics, which could lead to residues in the milk if the incorrect WHP is used. Monitoring data in goat, sheep and buffalo milk would assist in addressing this issue.
- Sanitizers are an integral part of the dairy production processes, but have the potential to contaminate milk and other dairy products if QA programs fail. Monitoring of valves and manufacturing practices in conjunction with increased training would assist in addressing these concerns.

Chemical Class	Current regulatory and non-regulatory measures chemicals associated with milk	Areas where further measures (regulatory or non-regulatory) may be necessary
Agricultural chemicals (pesticides, herbicides and sanitisers)	• Registration and control of use legislation • Monitoring for residues in milk • Good manufacturing practice/HACCP-based food safety plans	• Monitoring of colostrum (for therapeutics) for Agvet chemicals. • Monitoring of valves and automated processes to ensure removal of sanitizers and cleaning agents
Veterinary chemicals (Ecto- and Endo-parasiticides; other veterinary chemicals)	• Registration and control of use legislation. • Monitoring for residues in milk	• Monitoring of colostrum (for therapeutics) for veterinary chemicals • Monitoring of residues resulting from off-label use for minor dairy species (goat, sheep and buffalo)

Environmental contaminants

Environmental contaminants such as heavy metals and organic chemicals may enter the dairy production chain through stockfeed or though the direct consumption of soil. Stockfeed is an integral factor in dairy production, which may impinge on the quality of milk produced. Stockfeed contamination may also result from the presence of endogenous plant toxicants or mycotoxins, or environmental chemicals.

The available data on arsenic, cadmium, mercury and lead indicate that milk is a very small contributor to the overall dietary intake of these metals and, at the current levels found in milk, there are no public health and safety concerns. Overall, the data suggests that stockfeed and soil do not significantly contribute to heavy metal contamination of milk.

The levels of the micronutrients iodine, selenium and zinc in milk have been examined and do not raise any public health and safety concerns. In the past, iodine content in milk has increased, but the use of iodine has declined with the implementation of alternative, more efficient sanitisers. Selenium and zinc supplementation does not significantly change the micronutrient content of milk. Milk is considered to be an important source of these three micronutrients and has a role in preventing deficiencies for these micronutrients in the community.

Dioxins can occur naturally in the environment although the major source is from industrial practices; the major source of exposure is through the diet. Because of the lipid solubility of dioxins, dairy products can be a significant source of dietary exposure. Although the results of the recent National Dioxin Program indicated that the dietary contribution from dairy products was significant, the overall dietary exposure to dioxins was low and did not raise any public health and safety concerns. PCBs are not naturally occurring but are found at low levels in the environment as a result of industrial activity. PCBs have not been detected in milk in the AMRA survey or in the ATDS.

Plant, fungal or bacterial toxins are potential contaminants in stockfeed. These include aflatoxin, ochratoxin, trichothecene toxins, zearalenone, fumonisin, cyclopiazonic acid, corynetoxins, pyrrolizidine alkaloids, lupin alkaloids, phomopsins and ergot alkaloids. Of these, only alfatoxin M1 is regularly monitored in milk. While earlier data from the Australian Mycotoxin Data Centre survey showed some milk samples with aflatoxin residues, the more recent surveys have not detected any aflatoxin residues in milk.

One area was identified where further data may be necessary to better characterise any potential public health and safety risks (summarised in the Table below):

- In relation to plant, fungal or bacterial toxins, while the information available does not raise any particular public health and safety concerns, additional monitoring of milk would address some of the uncertainty in the current information relating to these toxins.

Chemical class	Current regulatory and non-regulatory measures for chemicals associated with stockfeed	Areas where further measures (regulatory or non-regulatory) may be necessary
Agricultural and veterinary chemicals	• Registration and control of use legislation • Monitoring for residues in stockfeed	
Environmental chemicals Heavy metals, organic chemicals, aflatoxin	• Sound primary production practices • Monitoring for residues in stockfeed	
Plant, fungal and bacterial toxins	• Sound primary production practices	• Monitoring of stockfeed for residues

Chemicals used in processing

At the processing end of the dairy production chain, food additives and processing aids are used in the manufacture of a wide range of dairy products. Food additives may be added to achieve a technological function, such as preservation or colouring, and are present in the final food, whereas processing aids fulfil a technological function during processing, but are not present in the final food.

The use of food additives and processing aids is regulated by the maximum permitted use levels in *the Australia New Zealand Food Standards.* There have been no recorded violations of the Code regarding the use of food additives or processing aids in dairy products. However, there is anecdotal evidence of the use of hydrogen peroxide as a preservative to prolong the shelf-life of cream. Further auditing of adherence to a Food Safety Program would address this potential concern.

Chemical class	Current regulatory and non-regulatory measures	Areas where further measures (regulatory or non-regulatory) may be necessary
Food Additives	• Approval and control of use legislation. • Sound manufacturing practices	• Additional monitoring for unapproved use of hydrogen peroxide as a preservative
Processing Aids	• Approval and control of use legislation. • Sound manufacturing practices	

Chemicals in dairy produce formed during or as a result of processing

Chemicals can be formed within dairy products due to processing or microbiological activity. The levels of biogenic amines and fungal toxins is variable although these toxins would probably only be produced in cheeses under circumstances where the microbial load was imbalanced, and temperature control and storage was not optimal. There is some data from case studies that indicates that there is potential for public health and safety concern for some individuals.

Polycyclic aromatic hydrocarbons (PAH) are by-products of cooking processes and have been found in small quantities in smoked cheeses, although exposure to PAHs through dairy products is considered to be low.

At the end of the production chain, packaging may also lead to the unintentional migration of chemicals from the packaging material into dairy produce. There is a paucity of data on the levels of migration of chemicals from packaging materials into foods in general, although in most cases, the levels are expected to be very low. Because of the high lipid content of dairy products, migration of some plasticizers may be of concern.

Three areas were identified where further data may be necessary to better characterise any potential public health and safety risk (summarised in the Table below):

- Further research is needed in relation to the public health and safety risks associated with biogenic amines. Research is also needed into the factors that influence biogenic amine formation. Further monitoring of levels in food would assist in characterising the potential public health and safety risk.
- Further monitoring of PAHs in smoked cheeses would assist in characterising the potential public health and safety risk. FSANZ is currently carrying out a survey (22nd ATDS) on dietary exposure to PAH in foods.
- Further monitoring of the level of migration of chemicals from packaging would assist in characterising the potential public health and safety risk.

Chemical class	Current regulatory and non-regulatory measures	Areas where further measures (regulatory or non-regulatory) may be necessary
Biogenic amines	• Good manufacturing practice • HACCP-based food safety plans	• Further information in relation to hazard identification and characterisation • Potential for intolerance reaction in certain individuals • Monitoring of levels in food
Fungal by-products	• Good manufacturing practice • HACCP-based food safety plans	• Monitoring of levels in food
Polycyclic aromatic hydrocarbons	• Good manufacturing practice • HACCP-based food safety plans	• Monitoring of levels in food
Chemicals which migrate from packaging	• Good manufacturing practice • HACCP-based food safety plans	• Further information in relation to hazard identification and characterisation • Monitoring of levels in foods

Conclusions from the Risk Profile of Dairy Products in Australia

High quality dairy produce has been produced in Australia for many years. This is due, in the main, to adherence to regulatory measures, good agricultural and manufacturing practices, and the maintenance of hygienic practices along the dairy primary production chain.

Microbiological risk profile

A wide range of microbiological hazards may be introduced into milk during primary production and processing. Raw milk has a mixed microflora, which is derived from including the interior of the udder, exterior surfaces of the animals, the environment (including faeces), milk-handling equipment, and personnel. In addition, the milking procedure, subsequent collection, storage of milk and processing milk into various dairy

products carry the risks of further contamination or growth of intrinsic pathogens. Importantly, the composition of many milk products makes them good media for the growth of many pathogenic microorganisms.

The safety of dairy products is due to the use of heat treatment and a combination of management and control measures along the entire dairy supply chain. Control of animal health, adherence to good milking practices, and control over milking parlour hygiene have been important in reducing the microbial load in raw milk entering Australian dairy processing facilities.

In addition, there have been few reported failures *i.e.* foodborne illness attributed to dairy products in recent years. While dairy products have been the vehicles in some outbreaks, the cause is often multifactorial involving contaminated non-dairy ingredients, post-process (post-pasteurisation) contamination, and poor hygiene practices.

The almost universal use of pasteurisation in milk processing in Australia has resulted in the marketing of dairy products with an excellent reputation for safety and product quality. The dairy industry has introduced significant measures to ensure product safety, including the adoption of codes of practice, adherence to *Listeria* and *Salmonella* control protocols, and the extensive use of HACCP-based Food Safety Programs supported by laboratory verification.

Notwithstanding the above, there is need for ongoing vigilance and further development of safety control measures. Over the past twenty years we have seen the emergence of new pathogens and the re-emergence of traditional pathogens in various foods. These organisms often occupy specific environmental niches and may arise through changing technologies, methods of food handling and preparation, dietary habits and population. Post-processing contamination in-plant and the maintenance of control over contamination and storage conditions during transport, retail display and home use remain major factors impacting on the safety of dairy products.

Chemical risk profile

There are extensive regulatory and non-regulatory measures in place to ensure that chemicals used or present in dairy products present a very low public health and safety risk.

The Chemical Risk Profile has identified two major findings. Firstly, the extensive monitoring of chemical residues in milk over many years has demonstrated a high level of compliance with the regulations. Secondly, the regulations and control measures currently in place along the dairy industry primary production chain have resulted in minimal public health and safety concerns regarding the use or presence of chemicals in dairy products.

The Chemical Risk Profile has also identified a number of areas where further research or monitoring would assist in providing further reassurance that the public health and safety risk is low. These have been summarised above.

Continuation of the current management practices, particularly monitoring programs for chemicals along the primary production chain, will ensure that the dairy industry continues to maintain a high standard of public health and safety.

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I BACKGROUND

Food Standards Australia New Zealand (FSANZ) has responsibility for protecting the health and safety of consumers through the development of food standards. The development of through-chain Primary Production and Processing (PPP) standards requires the thorough assessment of risk[1] to public health and safety.

FSANZ uses a number of tools to assess risks to public health and safety, including risk profiling[2], quantitative and qualitative risk assessments[3] and scientific evaluations. The application of these tools to the assessment of the risks to public health and safety is dependent on the purpose of the assessment and on the availability, quality and quantity of relevant data.

FSANZ follows established international guidelines and incorporates elements of the Codex Alimentarius Commission risk assessment framework when undertaking risk profiles, risk assessments and other scientific evaluations. Guidance for undertaking risk assessments have been drafted internationally by the Food and Agriculture Organization of the United Nations (FAO) and the World Health Organization (WHO).

In assessing risks to public health and safety, available scientific data concerning the safety of the commodity under consideration and the properties of the hazard are evaluated. This requires utilisation of relevant scientific data and includes procedures to address uncertainty and variability in the conclusions drawn from the data, *i.e.* consideration of the relevance and quality of data and the veracity of its source.

The outcome of any assessment of risks to public health and safety may include a statement on the probability and severity of an adverse health effect due to the consumption of a food containing a particular biological, chemical or physical agent. An assessment may also identify where in the production chain controls over hazards will have the greatest impact on minimising risk, *i.e.* informing risk managers where intervention will be most effective. The outcomes of the assessing risks to public health and safety for dairy products are used by FSANZ to inform risk management decisions.

The assessment of risks to public health and safety from microbiological hazards in milk and milk products has been undertaken in the form of a **Microbiological Risk Profile (Part A)**. It provides a broad overview of risks associated with consumption of dairy products in Australia and includes a description of the current status of pasteurisation in Australia and an evaluation of alternative processes to pasteurisation for the production of milk and milk products. The risk profile identifies key food safety hazards and assesses where in the primary production and processing supply chain these hazards might be introduced, increased, reduced or eliminated.

The assessment of risks to public health and safety from chemicals associated with milk and milk products has been undertaken in the form of a **Chemical Risk Profile (Part B)**. This

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1 Codex defines the term risk as 'a function of the probability of an adverse health effect and the severity of that effect, consequential to a hazard(s) in food'

2 Risk profiling is defined by FAO/WHO as 'the process of describing a food safety problem and its context, in order to identify those elements of the hazard or risk relevant to various risk management decisions'.

3 Risk assessment is a scientific process undertaken to characterise the risk to public health and safety posed by foodborne hazards associated with a food commodity.

risk profile identifies and examines where chemicals may enter the dairy supply chain (both intentionally and unintentionally) from the primary produce to processed foods. It also considers the relevant inputs (*e.g.* feed, water, etc) into the dairy primary production and processing chain.

Hazards of a physical nature associated with dairy products have not been considered.

II SCOPE AND PURPOSE

The purpose of the risk profile in dairy products is to provide an objective analysis of relevant scientific data and information to identify the public health and safety risks associated with dairy products. This will enable risk managers to consider the risks associated with dairy products and the reductions in risk that may be achieved with various production and process control options. The risk profile may also identify the need for more detailed microbiological risk assessments for specific dairy commodities[4]; requirements for further monitoring of chemicals or manufacturing practices, or further information in relation to hazard characterisation of chemicals.

The **Microbiological Risk Profile (Part A)** was undertaken to gather the following information:

1. What microbiological hazards are associated with the Australian dairy supply chain, under the current regulatory system, and what is the likelihood that these hazards pose a risk to public health and safety?

2. What are the factors along the Australian dairy supply chain that have the most significant impact on public health and safety risks?

The microbiological risk profile identifies and examines hazards along the dairy supply chain from milk production through to consumption of dairy products and has considered the relevant inputs (*e.g.* feed, water, etc) into the dairy primary production and processing chain. The risk profile encompasses the following elements:

- Identification and description of the microorganisms that may be associated with dairy products including key attributes of each organism and its public health impact (hazard identification/hazard characterisation);
- Examination of epidemiological data (domestic and international) related to the consumption of dairy products;
- Examination of prevalence and concentration data on potential hazards from products along the entire dairy food chain; and
- Description of the dairy production, processing, distribution and consumption chain and what is currently known of the impact of these factors on public health and safety risks.

The **Chemical Risk Profile (Part B)** was undertaken to gather the following information:

1. To identify those chemicals associated with dairy products which may potentially impact on public health and safety in Australia;

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4 There is interest from the Australian dairy industry for FSANZ to consider technological innovations and the production of selected raw milk products. Currently, raw milk and raw milk products are not permitted to be sold in Australia, unless expressly permitted by a State or Territory or a specific exemption has been given as the result of an assessment process *e.g.* the sale of raw goat milk is permitted in some States. In addition, some specific raw milk cheeses are permitted where an assessment process has shown that they can be produced to an equivalent level of safety as cheeses made from heat-treated milk. To date the assessment of raw milk cheeses has been done on a case-by-case basis for selected imported cheeses

2. To assess the potential public health and safety risks associated with these chemicals, in the context of the current regulatory system;

3. To identify any areas in the current regulatory system which require further attention in relation to addressing potential public health and safety risks associated with chemicals in dairy products.

The chemical risk profile identifies and examines where chemicals may enter the dairy supply chain (both intentionally and unintentionally) from the primary produce to processed foods. It also considers the relevant inputs (*e.g.* feed, water, etc) into the dairy primary production and processing chain. The report considers the following:

- Agricultural and veterinary chemicals used in primary production;
- Environmental contaminants, including heavy metals, organic contaminants and micronutrients;
- Natural chemicals found in plants, fungi or bacteria associated with plants;
- Food processing by-products;
- Food additives, processing aids and those chemicals that may migrate from packaging.

The microbiological and chemical risk profiles were undertaken within the framework of existing management and regulations in Australia.

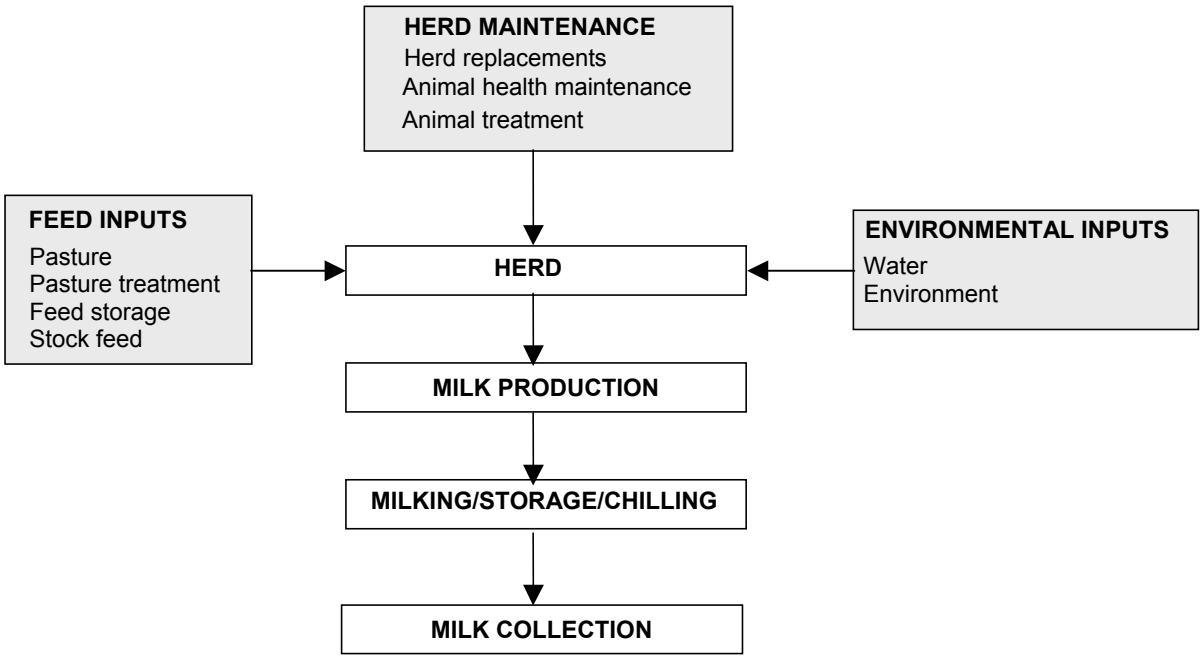
The risk profiles consider microbiological and chemical hazards associated with milk and milk products produced from the main commercial dairy species, including cows, goats, sheep, buffalo and camels.

The Australian dairy industry is predominantly based on bovine species, however other animal species are milked commercially in small operations/enterprises e.g. goats, sheep and buffalo. The bulk of goat milk production is utilised in the cheese making industry, but some is also used for cream, yoghurt and drinking milk. Sheep milk is utilised in cheese and yoghurt making, as is the milk obtained from buffaloes. Whilst there is a camel farm established near Alice Springs, it has not yet commenced commercial milk production.

The focus of this profile is on bovine species. It is assumed that on-farm and processing practices relating to other milking species other than bovine animals are largely the same as those for bovine species. Where practices are different for other species and where there is appropriate available data, specific information is provided. Dairy products from bovine animals represent the great majority of milk and dairy products sold in Australia.

III DAIRY FARMING IN AUSTRALIA

The key stages in the primary production of raw milk are shown diagrammatically as follows:



The dominant dairy cattle breed in Australia is the Holstein-Friesian, accounting for approximately 70% of all dairy cattle. Other important breeds in Australia include the Jersey, the Holstein/Jersey cross, and the Illawarra. The average herd size has been increasing in Australia and was estimated at 195 cows in 2002/03. However there are some very large farm operations where a single property may support up to ten individual herds of 500-1,000 dairy cattle.

The Australian dairying industry is predominantly pasture-based, with approximately 80% of cattle feed requirements coming from grazing. Feedlot-based dairying remains unusual, although the use of feedpads for the provision of supplementary feed, such as hay, silage and grains, is common. Pastures such as white clover, strawberry clover, perennial ryegrasses, paspalum and kikuyu are the major feed sources for cattle. Large quantities of pasture are conserved as hay and silage for feeding during periods of low pasture growth or feed availability. Concentrates, particularly mixed grains, which are commonly based on cereal grains such as barley and may include other ingredients such as lupins are fed as a supplement to pasture.

Grazing systems are either set/continuous or rotational. Animals have continuous access to one paddock all year round in set stocking or continuous grazing, whereas in rotational systems animals move around a series of paddocks in a strictly controlled way.

Australian milk production is strongly seasonal, reflecting the pasture based nature of the industry. Milk production is seasonally more pronounced in southern states such as Victoria and Tasmania where cattle graze on pasture year round. Peak milk production occurs in October/November and lowest production in May/June.

In Australia, approximately 20% of cows from milking herds are lost each year through poor health, calving problems, mastitis, infertility, death or culling for old age or poor milk production. These cows need to be replaced to maintain a constant number of lactating cows each year. Milk quality decreases and mastitis incidence increases with ageing of animals. Farmers therefore need to replace these animals through herd replacement purchases and/or calf rearing. Cattle are mostly bred by artificial insemination, however the adoption of embryo transfer technology is increasing.

Calves receive their first feed of colostrum within the first six hours of birth. Milk or milk replacers are offered to calves until at least 5-6 weeks of age. Weaning usually is based on live weight rather than age, to ensure the calf is sufficiently well developed to deal with bulky roughage feeds such as pasture. It may take 6-12 weeks for calves to reach target weights for weaning. Once calves are trained to drink they can be transferred to a separate calf paddock.

As the young female calf grows and matures she is called a heifer, and it takes about 15 months for her to grow to a size at which she can be mated resulting in a first calving at an age of about 24 months. After parturition, the cow will give milk for about 300 days (termed the lactation period). The average amount of milk produced in a complete lactation period is approximately 5,000 litres. More milk is produced at the start of lactation (average 17 litres per day) than at the end of lactation period (5-10 litres per cow).

Cows are mated about 60 days after calving, so the cow will have a calf to initiate milk production in 9 months from mating or next lactation. After about 300 days, cows are dried off and rested from milking for about 2 months before calving again and repeating the cycle.

In Australia, the milking process is largely mechanised. Animals are milked by suction and the milk transferred to refrigerated farm vats. Types of milking systems include herringbone, rotary, and fully automated. The herringbone-type system is the most popular style in Australia, with cows positioned along each side of a pit. The rotary dairy is the fastest and most efficient way of milking large numbers of cows and consists of a rotating milk platform, which allows a constant flow of cows to enter and leave the platform individually.

Robotic milking is a fully automatic modular milking system capable of milking all herd sizes. The technology is highly advanced, and only one robotic dairy currently operating in Australia. The robotic dairy's automated identification system recognises each cow, directs laser-sensing guides for application of teat cups, and records milk volume and colour. Milk discolouration due to blood or mastitis is recognised and the system will automatically discard this milk. Once milking is completed, a disinfectant is applied, usually as a spray. The cow is then released to the exiting area.

Milk is initially cooled after it leaves the udder most commonly by passage through a heat exchanger (plate cooler), prior to entering the milk vat (bulk milk storage tank). The milk is filtered before it enters the bulk milk storage tank and this provides a safeguard to ensure sediment or other extraneous matter is removed from the milk prior to storage. The milk is further cooled by the refrigeration system in the milk vat.

For a significant part of the year in Australia, milk is collected on alternate days or every 36 hours, depending on the milk vat capacity of individual farmers. The milk is then transported to milk processors in insulated bulk milk tankers.

IV REGULATION OF DAIRY PRODUCTS IN AUSTRALIA

Australia currently has State-based regulations for the dairy sector that cover on-farm activities, milk collection and dairy product manufacture. For most jurisdictions this includes the requirement for HACCP-based food safety programs for on-farm and dairy processing activities. The Authorities responsible for maintaining and implementing these requirements are:

- NSW Food Authority;
- Safe Food Queensland;
- Dairy Authority of South Australian;
- Tasmanian Dairy Industry Authority;
- Dairy Food Safety Victoria;
- Health Department of WA.

There are no dairy farms in the ACT or Northern Territory however, milk processing and packaging is conducted in these jurisdictions. These activities are covered by the requirements of the Food Acts in those jurisdictions. A summary of State legislative requirements is provided in the table below.

Summary of State legislative requirements

State	Legislation	Food Safety Programs(requirement outlined in)
NSW	Food Act 2003 (& Food Standards Code) Food Production (Dairy Food Safety Scheme) Regulation 1999	NSW Dairy Manual
QLD	a) Food Production (Safety) Act 2000 & Food Production (Safety) Regulations 2002 (SFQ) b) Food Act 1981 (QLD Health Dept)	a) FPS Act b) To be implemented under revisions to the Food Act
SA	Primary Produce (Food Safety Schemes) Act 2004 Primary Produce (Food Safety Schemes) (Dairy Industry) Regulations 2005	Dairy Authority of South Australia Code of Practice for Dairy Food Safety
TAS	Dairy Industry Act 1994	Tasmanian Code of Practice for Dairy Food Safety
VIC	Dairy Act 2000	Victorian Code of Practice for Dairy Food Safety
WA	Health Act 1911Health (Food Hygiene) Regulations 1993Food Safety Standards	Code of Practice for Dairy Food Safety (Under development)

A technical group known as the Australia New Zealand Dairy Authorities Committee (ANZDAC), comprising of representatives from the each State jurisdiction, the New Zealand Food Safety Authority and the Australian Quarantine Inspection Service (AQIS), ensures that the statutory responsibilities of each jurisdiction with respect to the legislative requirements for dairy premises and products is applied in a uniform and consistent way across Australia.

On-farm requirements

As outlined in the table above, on-farm food safety programs are required and implemented through licensing arrangements by regulators in New South Wales, Queensland, Victoria, South Australia and Tasmania. They are under development in Western Australia. The elements covered by these programs include:

- animal health;

- environmental hygiene;
- animal feeds, agricultural and veterinary drugs;
- areas and premises for milk and milk production, milk storage and milking equipment;
- pest control;
- hygienic milking, and
- milk storage

Animal health
All regulations require milking animals to be free from diseases and verified through record keeping requirements in relation to the health of the animal to be milked. NSW has an additional requirement for animals to be free of Enzootic Bovine Leucosis (EBL).

Environmental hygiene
On-farm requirements include the management of effluent to minimise contamination from this source, particularly to the use of reclaimed water to irrigate dairy pastures. Additional requirements to those set out in Dairy regulations include:

- Environmental Guidelines for the use of reclaimed Water, (EPA, 2001);
- Reclaimed water on dairy farms – General Information and Requirements for Users, (VDIA, 1999) – Victoria;
- Managing Dairy Farm Effluent in Tasmania – Code of Practice; and
- Guidelines for the Use of Reclaimed Water in Tasmania

Animal feeds, agricultural and veterinary chemicals
An outcome based requirement of State legislation for animal feeds (which includes pasture) is that feeds should not present a risk of introducing hazards into the milk. The use of agricultural and veterinary chemicals is also controlled by State legislation- only registered chemicals should be used and in accordance with instructions for use. Agricultural and veterinary chemicals are assessed as part of a pre-market evaluation and approval process and generally residues in milk are specified in the Australia New Zealand *Food Standards Code*[5]. Record keeping and vendor declarations should be used to verify appropriate controls are in place.

Areas and premises for milk and milk production, milk storage and milking equipment
State legislation covers the requirement for dairy premises for milk production and storage to be designed, constructed and maintained in order to prevent/minimise contamination of the milk. In NSW, additional guidelines are provided in the Code of Practice for Dairy Buildings.

Pest Control
Pests should be controlled on-farm so that they do not contaminate milk through their activities. The pest control method (such as the use of pesticides) should also not result in contamination.

[5] Standard 1.4.2 Maximum Residue Limits (Agricultural and Veterinary Checmials)

Hygienic Milking,
Requirements for hygienic milking include the exclusion of personnel from milking activities if they are ill with, or are carriers of, an infectious disease. Additionally, people undertaking milking activities must employ good sanitary practices to prevent contamination of the milk.
Milk storage
All State requirements specify temperature controls for the storage of milk. In general milk must be cooled within a specified period (3.5 hours) to a temperature of less than 5°C (less than 4°C in NSW).

Milk collection and transport requirements
All States have food hygiene requirements for the collection and transporting of milk from the farm to processing centres. Temperature requirements are specified such that milk must be collected at a temperature of less than 5°C (less than 4°C in NSW). In NSW, there is a Code of Practice for Milk Collection from Dairy farms.

Dairy manufacturing requirements
The requirements of Standard 3.2.2 Food Safety Practices and General Requirements and Standard 3.2.3 Food Premises and Equipment of the *Food Standards Code* apply to manufacturing premises. These are referenced by State and Territory Food Acts. State legislation also requires dairy manufacturers to have food safety HACCP-based food safety programs in place. The elements covered by regulations include:

- chemical, microbiological, physical contamination (from premises, equipment and personnel);
- cleaning and sanitising;
- temperature control; and
- personnel competencies.

State legislation also requires dairy manufacturing establishments to comply with the:

- Australian Manual for the Control of Salmonella in the Dairy Industry published by Australian dairy Authorities Standards Committee (ADASC), and,
- Australian Manual for the Control of Listeria in the Dairy Industry published by ADASC.

Milk for manufacture must be heat-treated in accordance with Standard 1.6.2 – Processing Requirements, of the *Food Standards Code*. Food additives and processing aids used in the manufacture of milk and dairy products undergo pre-market evaluation and approval and are specified in the *Food Standards Code*.[6] Sanitisers are also assessed before use and regulated by the Australian Pesticides and Veterinary Medicines Authority (APVMA).

In addition the Food Standards Code specifies Microbiological Limits[7] and maximum levels for contaminants[8] in various dairy products

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[6] Standard 1.3.1 Food Additives
Standard 1.3.3 Processing Aids
Standard 1.3.4 Identity and Purity
Standard 1.4.3 Articles and Materials in Contact with Food

[7] Standard 1.6.1 Microbiologica Limits for Food

[8] Standard 1.4.1 Contaminants and Natural Toxicants

Distribution of milk and milk products requirements
Dairy distributors or depots are also covered by State dairy legislation. In NSW the requirements emphasise temperature control and record keeping and reference is made to the Code of Practice for Dairy Depots.

In the Queensland Food Production (Safety) Regulation 2002, there are requirements relating to temperature control environmental conditions to avoid contamination.

The requirements of the Code of Practice for Dairy Food Safety currently implemented in Victoria, South Australia and Tasmania, and under development in Western Australia, dairy distributors must have a food safety program based on the Codex HACCP principles

V CONSUMPTION OF DAIRY PRODUCTS IN AUSTRALIA

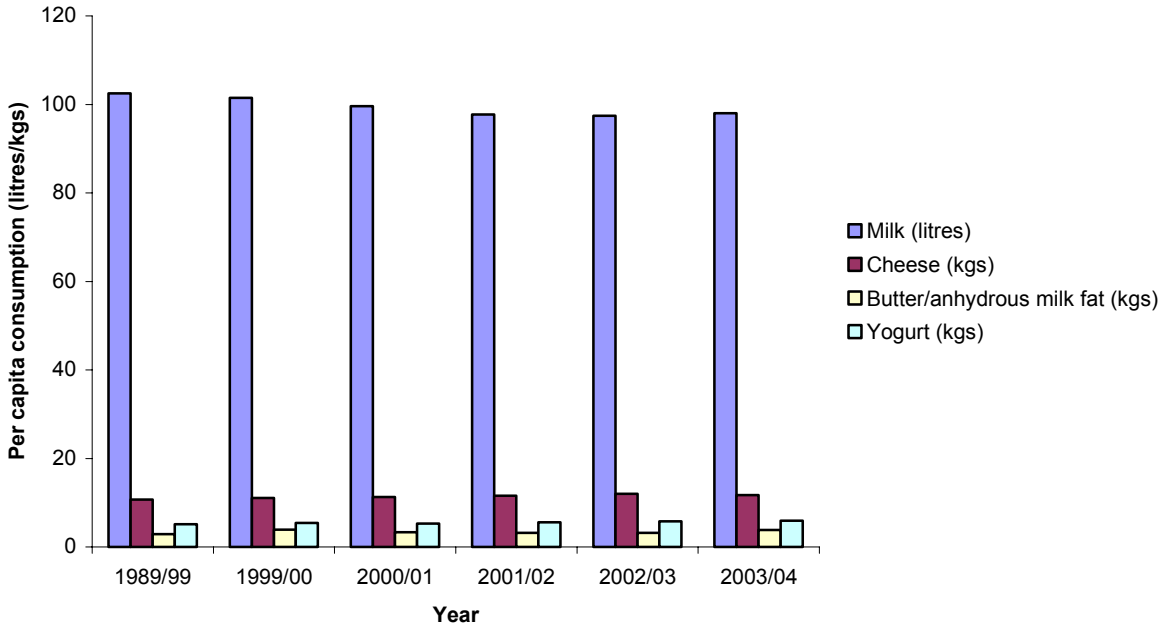
Milk and milk products are a significant component of the diet for the majority of the Australian population. Milk is an extremely valuable food for human nutrition as it contains all the basic components required for human life (Spreer, 1998).

The four major Australian consumer dairy products are drinking milk, cheese, butter and dairy blends, and yoghurt (Dairy Australia, 2004). Consumption trends over the past two decades vary quite significantly by individual product. These reflect changes in consumer tastes and preferences in response to a multitude of variables such as the multicultural influences on the foods we eat; health perceptions of dairy products and manufacturers' responses (such as low fat variants); new product development; flavour and packaging innovations; competitive category offerings; distribution and availability of product.

Consumption data can be calculated from food production statistics or food consumption surveys. Food production statistics provide an estimate of the amount of specific food commodities available to the total population. This type of data may include national statistics on per-capita food production. Consumption surveys (such as national nutrition surveys) provide detailed information regarding the types and amounts of foods consumed by individuals or households and sometimes the frequency with which the foods are consumed.

Consumption data for dairy products from food production statistics

The following Figure illustrates the Australian per capita consumption figures for major dairy products from 1989 - 2004.



Per capita milk consumption has slowly declined since the mid-1990s to an estimated level of 98 litres per head in 2003/04. Milk consumption has been decreasing due to concerns over excess fat in diets and the increased availability of substitute non-dairy products such as soya bean-based drinks. Patterns of milk consumption have also been steadily changing from

regular whole milk to modified milk types, such as reduced and low-fat milks and fortified specialty milks. Flavoured milks are also increasing being consumed in place of regular milk (Dairy Australia, 2004). The annual consumption of cream in 2001 was 4.05 kg per head (Datamonitor, 2002).

Cheese consumption is estimated at nearly 12 kilograms per head per annum. Almost all cheese consumed is cheddar and cheddar types, however, there is an trend to consume increased amounts of non-cheddar cheese types (Dairy Australia, 2004).

Annual consumption of butter in Australia is about 3 kg per head. There has been a decline in butter consumption over the past three decades as consumers have sought to reduce their saturated fat intake. However there has been a slight increase in consumption of these products since 1996 with the introduction of butter and vegetable oil-based dairy blends (Datamonitor, 2002).

Annual consumption of yoghurt has significantly increased over the past decade with 6.46 kg/head being consumed in 2001 (Datamonitor, 2002). Low-fat and diet varieties account for more than 60% of the yogurt market (Dairy Australia, 2004). The consumption of dairy desserts in Australia is 1.27 kg per head/year in 2001 (Datamonitor, 2002). Consumption of dairy desserts is growing with products such as mousses, crème caramels and fromage frais marketed for adults and fromage frais flavoured custards marketed towards children.

Consumption of ice cream has slowly been increasing. In 2001, annual consumption was 16.34 kg per head compared to 14.41 kg per head in 1996 (Datamonitor, 2002).

Annual consumption of dried milk is static and was 0.11 kg per head in 2001 (Datamonitor, 2002).

Consumption of condensed and evaporated milks is estimated at 0.08 and 0.03 kg/head/year respectively in 2001. The consumption of these products appears to be relatively stable since 1996 (Datamonitor, 2002).

Consumption data for dairy products from the 1995 Australian National Nutrition Survey
Data from the Australian National Nutrition Survey (NNS) provides detailed information regarding the types and amounts of dairy foods Australian's are consuming. The most recent national survey was conducted during the period from February 1995 to March 1996. Approximately 13,800 people aged two years or over from urban and rural areas in all States and Territories participated in the survey.

Two approaches were used in the NNS to collect data on food and beverage intake. The daily food consumption (24-hour recall) method was used as the main indicator of food intake. All participants were interviewed by trained nutritionists who sought detailed information on all foods and beverages consumed during the day prior to the interview (from midnight until midnight). A sample of approximately 10% of the NNS participants also provided intake data for a second 24-hour period. A Food Frequency Questionnaire was used to assess usual frequency of intake for those aged 12 years or more.

A summary of consumption of dairy products from the 1995 Australian National Nutrition Survey is outlined in the following Table**(Australian Bureau of Statistics and Department**

of Health and Family Services, 1997). Detailed results from the 1995 Australian National Nutrition Survey for the various dairy products are presented in Appendix 4.

Product	Average no. people surveyed consuming product (%)	Average amount product consumed per day (g)
Milk and other liquid milk products	84	347
All Cheese types	41	34
Very hard cheese	2.3	8
Hard cheese	27.7	32
Semi soft cheese	1.6	23
Soft cheese	5.3	33
Processed cheese	9.4	30
Ice cream	15	112
Butter	14	13
Yoghurt	9	177
Cream	7.7	29
Dairy based dips	1.5	43
Dairy based desserts	4.7	148
Dried milk	1.26	17
Goats milk	1	248
Concentrated milk	<1	57

Milk and other liquid milk products are consumed in significant quantities, with 84% of those people surveyed during the 1995 Australian National Nutrition Survey consuming an average amount of 347 g/day with the quantity ranging from 215–536 g/day. Less than 1% of those surveyed consumed goats milk.

A relatively high proportion of people surveyed consumed cheese, with consumption by 41% of the population. The average daily consumption varied from 25 g/d (65^+ years females) to 50 g/d (19-24 year males). Of the various cheese types available, hard cheese was the cheese most commonly consumed with 27.7% of people surveyed consuming this product. The average amount of hard cheese consumed was 32 g/day. This was followed by processed cheese with consumption by 9.4% of the population surveyed, predominantly by children. The average amount of processed cheese consumed was 30 g/day. Soft cheese was consumed by 5.3% of the population, with consumption greatest among females aged 45-64 years. Only 1.6% of the population consumed semi-soft cheese, with the principal consumers being people aged 45^+ for both genders.

Fifteen percent of people surveyed consumed ice-cream. The greatest consumers of ice cream were females aged from 12-15 years, however at least 10% of all age groups consumed ice-cream. Average amounts eaten varied between 59 g/day for girls aged 2-3 years and 217 g/day for boys aged 16-18 years.

Butter was consumed by 14% of the people surveyed. The greatest consumers of butter were males aged 65^+. The average amount of butter consumed by this age group was 20 g/day.

Yoghurt and cultured dairy products were consumed by 9% of the people surveyed and the greatest consumers were females aged from 45-64 (consuming 137g/day) and the lowest consumers where males aged 12-15 and 19-24.

Dried milk was only consumed by 1.26% of the people surveyed. The greatest consumers were males and females aged from 45-64 and 65^{+}. The average amount of dried milk (after reconstitution) consumed by these age groups was 220 mls. Dried milk probably replaces liquid milk in the diet. In Australia, a significant proportion of manufactured dried milk is exported or used as an ingredient in other foods.

Concentrated milks are consumed by less than 1% of the people surveyed, the greatest consumers were males aged from 45. The average amount of concentrated milks consumed by this age group was 37 g/day.

Dairy based dips and dairy based desserts were consumed by 1.5% and 4.7% of the people surveyed respectively. Of those who consumed dairy based dip products, the greatest consumers were females aged from 16-18 (consuming 46 g/day) and the lowest consumers where females aged 12-15. Of those who consumed dairy based dessert products, the greatest consumers were children aged from 2-7 (consuming between 103-157 g/day).

Although some data is available for whey consumption in Australia, whey products are normally used as functional ingredients in other foods.

VI RISK PROFILES

PART A: MICROBIOLOGICAL RISK PROFILE

1. Introduction

This risk profile identifies the microbiological public health and safety risks associated with dairy products in Australia.

In compiling the risk profile, a wide range of scientific literature, data and information from Australia and overseas was reviewed and evaluated. The risk profile provides a broad overview of microbiological risks associated with consumption of dairy products in Australia and includes a description of the current status of pasteurisation in Australia and an evaluation of alternative processes to pasteurisation for the production of milk and milk products.

Dairy Australia has funded the development of a quantitative microbiological risk assessment model to assess factors influencing the fate of hazards along the dairy supply chain. Dairy Australia permitted FSANZ to access the results and to utilize the model to inform the risk profile. The work was performed by the Australian Food Safety Centre of Excellence (AFSCoE) who modelled the fate of selected microbial pathogens in milk from primary production until after pasteurisation.

FSANZ also engaged external experts to review the status of pasteurisation and other technologies in Australia to:

- Define the effect of pasteurisation on pathogenic microorganisms in milk;
- Determine how current pasteurisation practices compare with regulatory requirements;
- Describe what alternative technologies are currently being investigated for use in the dairy industry worldwide, and what is known about their ability to destroy pathogens;
- Consider processes and challenges in validating these alternative technologies;
- Determine the opportunities for, and limitations on, the use of these technologies; and
- Describe the level of interest in such technologies within the Australian dairy industry.

1.1 Grouping of dairy commodities

For the purpose of this profile, dairy commodities were grouped into broad categories as follows:

- Milk and cream
- Cheese
- Dried milk powders
- Infant formulae
- Concentrated milk products

- Butter and butter products
- Ice-cream
- Cultured and fermented milk products
- Dairy deserts
- Dairy-based dips
- Casein, whey products and other functional milk derivatives
- Colostrum

These categories are based primarily on those foods that are currently included in microbiological standards or guidelines in the *Australia New Zealand Food Standards Code* (the Code). Additional dairy commodities were considered where they are relatively new to the market or little is known of their microbiological status.

1.2 Microbiological hazards associated with dairy products

A wide range of microbiological hazards may be introduced into dairy products during the primary production and processing stage.

Raw milk has a mixed microflora which is derived from several sources including the interior of the udder, exterior surfaces of the animals, the environment, milk-handling equipment, and personnel. Milking animals may carry a wide range of microorganisms, some of which are human pathogens and these may contaminate raw milk.

In addition, the milking procedure, subsequent collection, storage of milk and processing milk into various dairy products carry the risks of further contamination or growth of intrinsic pathogens. Importantly, the composition of many milk products makes them good media for the outgrowth of many pathogenic microorganisms.

A broad range of microorganisms were considered in this assessment. The microorganisms are representative of those that may be present in raw milk, either directly transmitted via the mammary gland or via faecal/environmental contamination. In addition, microorganisms that originate from the milking environment and/or post-pasteurisation contamination were also considered. Table 1 provides a brief summary of the microorganisms, the severity of associated illness and the availability of epidemiological data.

Table 1: Summary of microorganisms considered in the risk profile

Organism	Shed directly in milk[#]	Contaminant of raw milk[##]	Survives pasteurisation	**Severity of illness**[§]	Dairy/dairy products implicated in foodborne illness
Aeromonas spp.	×	✓	×	Serious	+
Bacillus cereus	×	✓	✓	Moderate	++
Brucella spp.	✓	✓	×	Severe	+
Campylobacter jejuni/coli	×	✓	×	Serious	++
Clostridium botulinum	×	✓	✓*	Severe	+
Clostridium perfringens	×	✓	✓	Moderate	+
Corynebacterium spp.	✓	✓	×	Serious	+
Coxiella burnetii	✓	✓	×	Serious	+
Cryptosporidium	×	✓	×	Severe	+
Enterobacter sakazakii	×	✓	×	Severe^	++
Pathogenic *E. coli*	×	✓	×	Severe	++
Listeria monocytogenes	✓	✓	×	Severe^	++
Mycobacterium avium subs. *paratuberculosis*	×	✓	×	–	–
Mycobacterium bovis	✓	✓	×	Severe	+
Salmonella spp	×	✓	×	Serious	++
Shigella spp.	×	✓	×	Serious	+
Staphylococcus aureus	✓	✓	×**	Moderate	++
Streptococcus spp.	✓	✓	×	Serious	+
Yersinia enterocolitica	×	✓	×	Serious	+

[#] transmission through udder; mastitis etc
[##] via faeces, the environment etc
[*] neurotoxin is heat labile
[**] enterotoxin is heat stable
[^] for vulnerable populations
[§] based on ICMSF (2002) severity ranking[9]
– No data/unknown
\+ Reported, but rare
++ More commonly associated with foodborne illness

When examining each dairy commodity category, only those potential pathogens relevant to the commodity being evaluated were assessed *i.e*. only those microorganisms relevant to the particular dairy commodity category were considered. A detailed characterisation of potential hazards is attached as Appendix 5.

[9] The estimate of the severity of adverse health effects caused by a foodborne agent is based on the ranking scheme for foodborne pathogens and toxins described by the International Commission on Microbiological Specifications for Foods {ICMSF, 2002 1843 /id}. The ICMSF ranking scheme categorises hazards by the severity of the threat they pose to human health, taking into consideration the: likely duration of illness; likelihood of death; and potential for ongoing adverse health effects.

The severity of adverse health effects caused by a hazard is ranked as moderate, serious or severe according to the following definitions:

Severity	***Description***
Moderate	Not usually life threatening; no sequelae; normally short duration; symptoms are self-limiting; can be severe discomfort
Serious	Incapacitating but not life threatening; sequelae infrequent; moderate duration
Severe	Life threatening, or substantial sequelae, or long duration

Under the ICMSF ranking, severe hazards are further divided into those applying to the general population and those applying to specific sub-populations, that is, susceptible individuals (for example, the very young and old, the immunocompromised, and pregnant women and their unborn children). This takes into account those situations where a hazard considered to be of moderate or serious severity to the general population may cause a severe illness in certain susceptible sub-populations.

This risk profile does not consider animal health issues associated with microorganisms other than those that specifically impact upon human health via foodborne transmission. The Australian Quarantine and Inspection Service (AQIS) and Biosecurity Australia maintain import requirements that are concerned with animal health and biosecurity issues. A quarantine permit must be obtained in order to import dairy products into Australia. These requirements must be met prior to compliance within the Code.

Bovine Spongiform Encephalopathy (BSE) is an animal and human health issue. FSANZ has undertaken a comprehensive risk assessment of the scientific data and information for BSE and Australia has developed a policy/framework (including a Standard) to manage the risk of BSE in food. Therefore this risk profile does not consider this issue.

There are no viral zoonoses shed in milk that are of concern to human health. Although Foot and Mouth disease[10] is shed in milk, it is of major concern to the dairy industry because it can be the vehicle for animal infection rather than human infection (Desmarchelier, 2001).

1.3 Use of antimicrobials

There are two public health issues arising out of the use of antimicrobial agents in the dairy industry. The first is the emergence of bacteria resistant to antimicrobial agents, which may reduce the ability of health and veterinary professionals to control infections resulting from foodborne transmission of such resistant bacteria. This may lead to an increase in morbidity and mortality and an increase in the costs associated with treatment of specific bacterial diseases. The second issue[11] is the potential for residues of the antimicrobial agent(s) to be present in food products, resulting in toxigenic or allergenic responses in some individuals. These issues are not restricted to the dairy industry. They are common to the use of antimicrobial agents in all food-producing animals. In addition, acquired antimicrobial resistance (AMR) can occur independently of the use of antimicrobial agents. However, antimicrobial agents can exert selective pressure on bacteria, increasing the rate of AMR development. A third issue specifically associated with the dairy industry is the impact on antimicrobials in cultured dairy products, where they can adversely affect starter cultures and their ability to adequately acidify products and hence assist product safety.

1.3.1 Evidence of antimicrobial resistant bacteria in milk and milk products

In general, there is a paucity of published information concerning antibiotic resistance in pathogenic bacteria from food and food-producing animals in Australia. The JETACAR report {Anon, 1999 1844 /id} contains information on diagnostic laboratory results of testing for AMR in food isolates of salmonellae. For the period 1989-1994, 9.7% (26/267)of *Salmonella* isolates from milk and milk products displayed resistance to at least one of ten antibiotics tested.

In Australia, most dairy animals are pasture fed. Antibiotics are predominantly used for therapeutic purposes, under veterinary supervision. Hence long term exposure to antibiotics through prophylactic use or as growth promotants is rare.

[10] Australia is officially free from Foot and Mouth Disease

[11] see Section IV Part B Chemical Risk Profile: subsection 5.7.2

1.4 Existing risk assessments of dairy products

In preparing the Dairy Risk Profile, previous risk assessments conducted by other scientific agencies were reviewed and evaluated (Appendix 6). There have been few assessments undertaken for dairy products, and typically they address specific pathogen:commodity pairs.

This profile considers the entire dairy supply chain, including the wide range of milk and milk products marketed in Australia. Where possible, the results of international risk assessments have been used to inform the development of the Dairy Risk Profile. Table 2 lists the major published risk assessments on dairy products.

Table 2: Existing risk assessments of dairy products

Pathogen	Risk Assessment/Comments
B. cereus	A concise risk assessment on *B. cereus* in the Netherlands predicted that 7% pasteurised milk might have levels of *B. cereus* contamination above 10^5 per ml. (Notermans *et al*., 1997).
Campylobacter	Risk profile explored potential transmission routes of *Campylobacter* in New Zealand, where dairy cows are implicated as a significant source of *Campylobacter jejuni* (Savill *et al*., 2003).
Enterobacter sakazakii	Significant resources have been devoted to assessing the food safety risk of *E. sakazakii* in powdered infant formula. Two comprehensive risk assessments have been completed by WHO/FAO (Food and Agriculture Organization of the United Nations/World Health Organization, 2004) and the European Food Safety Authority (EFSA (European Food Safety Authority) - Scientific Panel on Biological Hazards, 2004). Two risk profiles have been published separately on the topic (CCFH (Codex Committee on Food Hygiene), 2003; Iversen and Forsythe, 2004).
Listeria monocytogenes	Farber et al. (Farber *et al*., 1996) published a *Health risk assessment of L. monocytogenes in Canada*. The USDA, FDA and Centers for Disease Control and Prevention jointly developed a quantitative risk assessment on *L. monocytogenes* in RTE foods in 2003 (FDA/Centre for Food Safety and Applied Nutrition, 2003). The New Zealand Food Safety Authority (NZFSA) prepared a risk profile on *L. monocytogenes* in ice cream (2003) (Lake *et al*., 2003). WHO/FAO developed a risk assessment on *L. monocytogenes* in ready-to-eat foods in 2004 (WHO/FAO, 2004). Specifically related to dairy products, several independent risk assessments have been published on *L. monocytogenes* in cheese made with raw milk including: • A quantitative risk assessment of human listeriosis from consumption of soft cheese made from raw milk (Bemrah *et al*., 1998), • A risk assessment of listeriosis linked to the consumption of two soft cheeses made from raw milk: Camembert of Normandy and Brie of Meaux (Sanna *et al*., 2004), and • A risk assessment of *L. monocytogenes* in Swiss Emmental cheese (Aebi *et al*., 2003). There have been other publications dealing with specific methodologies used in risk assessment for *L. monocytogenes* in foods. One refers to an animal model (Notermans *et al*., 1998) and the other refers to inactivation of *L. monocytogenes* in milk by HTST pasteurisation (Piyasena *et al*., 1998).
M. bovis	NZFSA developed a risk profile on *M. bovis* in milk in 2002 (Lake *et al*., 2002).
Staphylococcus aureus	Lindqvist et al. (Lindqvist *et al*., 2002) prepared a risk assessment on *S. aureus* in raw milk cheese. The European Commission (European Commission, 2003) published an Opinion of the Scientific Committee on veterinary measures relating to public health on Staphylococcal enterotoxins in milk products, particularly cheeses
Others	• (Sumner, 2002) Food safety risk profile for primary industries in South Australia • Agency for Food and Fibre Science, Queensland Department of Primary Industries (2004) Queensland raw goat milk risk assessment, commissioned by Safe Food Qld (unpublished) • Centre for Food Technology (2002) Queensland dairy product risk assessment, commissioned by Safe Food Queensland (unpublished) • AgriQuality New Zealand Ltd. (2002) Risk assessment of sheep and goat milk, for Safefood NSW (unpublished)

2. Occurrence of microbiological hazards associated with Dairy products

Data from a number of sources was used to assess the occurrence of microbiological hazards in dairy products in Australia and overseas. Most data was obtained from the scientific literature, with additional data sourced from Dairy Australia, the National Enteric Pathogen Surveillance Scheme, the Dairy Authority of South Australia, Australia's Imported Food Program and FSANZ's food recall database. The data is summarised in Appendix 3.

2.1 Occurrence of microbiological hazards in dairy products in Australia

Data collated by the National Enteric Pathogen Surveillance Scheme (NEPPS) from 1983 - 2004 showed that *Salmonella* was isolated from 1,156 dairy samples, including raw cow's milk, raw goat's milk, dried milk powders, infant formula, ice cream, concentrated milk, whey powder and casein (Table 1, Appendix 3). The data showed that a large range of *Salmonella* serovars have been isolated from dairy products in Australia, but the total number of samples is not provided in the dataset so prevalence calculations cannot be made.

Analytical data was obtained from the Dairy Authority of South Australia for pasteurised milk, cheese, dip/dessert and yoghurt between 1998 and 2004 (Tables 2-3, Appendix 3). Tests were undertaken for standard plate count, coliforms, yeasts, moulds, *E. coli*, coagulase-positive *S. aureus*, *L. monocytogenes* and antimicrobial substances. The data were recorded as groups of tests, and so an analysis of results against individual tests could not be undertaken. However, generally there was a good level of compliance in the foods tested.

Analytical data was also obtained from Dairy Food Safety Victoria's product testing program for various dairy products between 2002 and 2005 (Tables 4-6, Appendix 3). Tests were undertaken for coliforms, *E. coli, L. monocytogenes,* Coagulase-positive *S. aureus* and *Salmonella*. Detections for *Coliforms, E. coli, L. monocytogenes* and Coagulase-positive *S. aureus* in various dairy products were found (Tables 4-6, Appendix 3).

Microbiological survey data from the scientific literature and Dairy Australia for pasteurised dairy products in Australia showed a very low incidence of hazards of public health significance in these products (Sections 3.1.3 and 3.1.4, Appendix 3). In most surveys for *E. coli*, *Listeria* spp, *Salmonella*, and *S. aureus*, no microorganisms were detected.

There is little data on the presence or absence of pathogens in raw milk and raw milk products in Australia. A small number of surveys however looked at *Aeromonas*, *L. monocytogenes*, and *Y. enterocolitica* in Australian raw milk. *Aeromonas* was detected in 27 – 60% of raw cows milk samples (Table 17, Appendix 3), *L. monocytogenes* was not detected in raw cows milk samples, but was detected in 1.4% of raw goats milk tested (Table 18, Appendix 3). *Y. enterocolitica* was detected in 12.8% of raw goats milk sampled (Table 19, Appendix 3). In addition a number of *Salmonella* isolates have been reported by NEPPS in both raw cows milk and raw goats milk (Table 1, Appendix 3).

A total of 43 dairy products and foods containing a dairy component have been recalled in Australia between 1990 and August 2005 (Section 3.1.2, Appendix 3). The recalls represent 6% of the total number of recalls that have occurred over this time. The products recalled include both domestically produced and imported dairy foods, with most of the recalls attributed to milk, cheese and cream. The majority of recalls were due to the presence of *L. monocytogenes* in product.

2.2 Occurrence of microbiological hazards in dairy products overseas

Surveys conducted overseas showed that pathogens are frequently isolated from raw milk (Section 3.2, Appendix 3). Pathogens were detected in raw milk in 85% of 126 surveys identified in the literature. Pathogens detected in raw cows milk included: *Aeromonas* spp., *B. cereus, Brucella* spp., *Campylobacter* spp., *Coxiella burnetii*, pathogenic *E. coli, L. monocytogenes, Mycobacterium* spp., *Salmonella, S. aureus, Streptococcus* spp., and *Yersinia* spp. Pathogens detected in raw goats milk included: *Brucella* spp., pathogenic *E. coli, Mycobacterium* spp., and *S. aureus*. Pathogens detected in raw sheep's milk included: *Aeromonas, Brucella* spp., pathogenic *E. coli, Mycobacterium* spp., and *S. aureus*.

While pathogens are rarely isolated from pasteurised milk they are more frequently found in pasteurised milk products, with affected products being cheese, infant formula and milk powder (Section 3.2, Appendix 3).

In addition, pathogens have been reportedly been detected in raw milk cheeses. However, in the few surveys of cheese documented, it could not be ascertained whether the cheese was manufactured from raw or pasteurised milk. In these few surveys where raw milk cheeses were specifically identified, pathogens were however rarely detected (Section 3.2, Appendix 3).

In analysis undertaken by AQIS from 2002 to 2004, a low percentage (up to 6.4%) of imported cheese samples failed for *E. coli* and *L. monocytogenes* testing (Table 7, Appendix 3).

3. Foodborne illness associated with dairy products

Foodborne illness causes a range of symptoms associated with gastroenteritis, but may also cause a number of other types of illnesses such as meningitis, septicaemia, neurological conditions and hepatitis. In addition, certain illness may have sequealae including reactive arthritis, irritable bowel syndrome and Guillain-Barré syndrome.

The national Gastroenteritis Survey results have estimated 5.4 million cases of gastroenteritis in Australia each year attributable to food. The most common pathogens associated with these being pathogenic *E. coli*, norovirus, Campylobacter and non-typhoidal Salmonella (Australian Government Department of Health and Ageing, 2005).

It has been reported that food prepared in restaurants and catering establishments, along with the mishandling of food by consumers is responsible for the majority of foodborne illness.

Prior to the introduction of pasteurisation, dairy products such as liquid milk were frequently implicated in various forms of foodborne illness. In the 19th century, milk was a common vehicle for communicable diseases such as scarlet fever, diphtheria and tuberculosis.

In Australia, a major outbreak of typhoid fever in the Melbourne suburb of Moorabbin in 1943 was attributed to raw milk. It lead Dr F.V.G. Scholes, Medical Superintendent , Queen's Memorial Infectious Diseases Hospital at Fairfield to state:

'*But the great lesson of the outbreak is that it is not safe to drink raw milk*'.

The result was that pasteurisation was soon made mandatory, and milk-borne diseases became much more uncommon.

3.1 Foodborne illness associated with dairy products in Australia

Prior to 2000, there was no nationally coordinated system of surveillance of foodborne illness. However since this time, Australia has introduced a national mechanism to enhance surveillance of foodborne illness and to provide a means for facilitating the national investigation of, and determining the causes of, foodborne illness (OzFoodNet).

During 1995-2000, a survey conducted by State and Territory health departments identified six outbreaks of foodborne illness associated with dairy products (Appendix 2,Table 1,) (Dalton *et al*., 2004). In addition a literature review of outbreaks prior to this time, also identified an outbreak of salmonellosis in 1977 associated with infant formula (Appendix 2, Table 1) (Forsyth *et al*., 2003).

Between January 2001 and December 2004 a total of 390 outbreaks of foodborne or suspected foodborne disease have been reported (OzFoodNet, 2005). Of these outbreaks, 3.9% (16/405) were potentially associated with the consumption of dairy products (Table 2, Appendix 2). In four of the sixteen outbreaks, a specific dairy product was identified as the food vehicle that caused infection. The remaining twelve outbreaks involved a food vehicle that contained dairy products as an ingredient.

Of the eleven outbreaks that could be attributed to a specific dairy product (excluding infant formula), unpasteurised milk was responsible for eight outbreaks, cheese, gelati, and cheese

sauce for the remaining three outbreaks (Appendix 2, Table 2). These 11 outbreaks affected 268 people, with a median number of 13 cases per outbreak and a range of 8 to 111 cases.

In 45% of these outbreaks the organism responsible was identified as *Campylobacter* (5 outbreaks). Single outbreaks were caused by *Cryptosporidium*, *Salmonella* Typhimurium 44, *Salmonella* Oranienberg and *Salmonella* Bredeney. In the outbreak associated with consumption of cheese sauce, the organism responsible was identified as C*lostridium perfringens,* and was likely to have been due to poor food handling. A causative organism could not be identified in one outbreak.

Four of the outbreaks occurred on school camps where unpasteurised milk was consumed and two on farms where unpasteurised milk was consumed. Unpasteurised milk was also consumed and led to single outbreaks in the community and in a school. Single outbreaks occurred from gelati serve at a restaurant, cheese sauce prepared by a caterer and from infant formula consumed in the community. The four outbreaks from specific dairy products identified in the OzFoodNet Outbreak Register were investigated using three point source cohort studies and one case control study. Data from before 2001 does not identify how outbreaks were investigated.

In twelve of the outbreaks identified in during 1995-2004, dairy products were an ingredient in the food vehicle identified as the source of infection (Table 2, Appendix 2). Dairy products are a component of many foods; therefore it is often difficult to determine whether they are the ingredients in the food vehicle identified as the cause of an outbreak.

The twelve outbreaks involved: cream filled cakes (4 outbreaks), custard as a food or part of a food (4 outbreaks), ice cream (3 outbreaks), and cheesecake (1 outbreak). Many of these foods also contain raw eggs, hence it is possible that the eggs and not the dairy component of the food was responsible for the infection. The agent responsible for these dairy related outbreaks was *S.* Typhimurium (11 reports). No organism was identified in one outbreak.

It is important to recognise that these outbreak data represent a small proportion of actual cases of foodborne illness, as many outbreaks go unrecognised. It should also be noted that it can be difficult to identify the key ingredient causing foodborne outbreaks, or critical factors contributing to their occurrence.

3.2 Foodborne illness associated with dairy products overseas

While commercial dairy products have rarely been identified as sources of foodborne illness in Australia, there have been a number of reports of outbreaks of illness associated with consumption of dairy products internationally. Information tabulated from a search of the international literature describes 163 outbreaks associated with dairy products from 1973-2003 (Appendix 2, Tables 3-12).

There have been twenty-two outbreaks attributed to pasteurised milk (13.5%) and seventeen outbreaks to cheese made from pasteurised milk or pasteurisation not stated so assumed pasteurised (10.4%). Faults with the pasteurisation process or a post pasteurisation contamination has been identified or suspected as the source of infection in each case.

Unpasteurised dairy products are the most common cause of dairy associated outbreaks of illness. There have been 30 outbreaks attributed to unpasteurised milk (18.4%), 18

unpasteurised milk cheese (11.0%), and 13 unpasteurised milk from non-bovine species (8.0%). The total number of dairy outbreaks associated with unpasteurised products is 61/163 (37.4%).

Ice cream was responsible for 23 outbreaks (14.0%), with raw eggs identified as an ingredient and a possible source of infection in fourteen of these outbreaks. Butter was associated with 6 outbreaks (3.7%) and yoghurt and fermented products associated with 2 outbreaks (1.2%). Dried milk products were associated with 5 outbreaks (3.0%). Eight outbreaks (5.9%) of illness resulted from foods where a dairy product was a component. Infant formula was associated with 19 outbreaks of illness (11.7%).

The extent to which outbreaks of foodborne illness can be attributed to raw or pasteurised milk or milk products does not enable risk assessors to clearly determine the relative risk that raw milk products pose to consumers. The literature is often unclear about the heat treatments given to milk and the term unpasteurised may apply to raw or thermised or improperly pasteurised milk (De Buyser, et al., 2001). Raw milk is as frequently involved as pasteurised milk in outbreaks, yet only a small proportion of milk and milk products are unpasteurised.

3.3 Attribution of foodborne illness to dairy products

While there is enhanced quantitative data on the incidence of illness due to specific pathogens, there is often not the ability or capacity to identify or distinguish specific food vehicles. The causative agent of an illness is usually determined through epidemiological studies, but confirming the identity of a key ingredient or the original source of product contamination, or critical factors contributing to their occurrence is problematic.

This inability to attribute cases of foodborne illness to causal vehicles is a major issue internationally, and is especially difficult where illness is linked to foods with multiple ingredients. Problems arise because of difficulties with:

- Food recall biases when gathering food consumption histories;
- Long exposure windows with specific pathogens;
- Inability to obtain representative food samples for analysis; and
- A lack of precision in, or suitable methods for, sample analysis.

Critical in this process is the capacity to link epidemiological data to animal and food monitoring data.

The development of public health interventions requires accurate data defining the source from which humans are acquiring pathogens and how specific foods contribute to the total burden of foodborne illness. However, outbreak data represents only a small component of actual cases of foodborne illness, as many outbreaks go unrecognised. People do not always seek medical attention for mild forms of gastroenteritis, and not all foodborne illnesses require notification to health authorities.

Nevertheless, the existing epidemiological data supports the proposition that pasteurised dairy products represent a low risk to public health, and that pasteurisation is an effective means of reducing the risk of human illness from dairy products.

4. Primary production factors impacting on milk safety

Raw milk has a mixed microflora which is derived from several sources including the interior of the udder, exterior surfaces of the animals, environment, milk-handling equipment, and personnel. In general, there are two means by which pathogens contaminate raw milk. Contamination may occur when microorganisms are shed directly into raw milk from the udder as a result of illness or disease, or through contamination from the external surface of the cow and the milking environment. Primary production factors that impact on these routes of contamination and the microbiological quality of the raw milk include:

- animal-related factors e.g. animal health, herd size, age and production status;
- environment-related factors e.g. housing, faeces, feed, soil, and water; or
- milking and operation of milking equipment factors.

Some of these primary production factors can be managed to reduce the risk of contamination of raw milk by pathogens, while management of others will have limited impact on the final microbiological status of raw milk.

4.1 Animal factors impacting on milk safety

Animal factors that may impact upon the microbiological quality of raw milk include, animal health, herd size, and age and production status of cattle. These factors impact upon the prevalence of microorganisms in animal herds and subsequent shedding in animal faeces, or directly into the milk itself. Shedding of microorganisms in the faeces can lead to further contamination in the farm environment. Faecal material can also contaminate raw milk directly from the animal's udder, hide, or hair, thereby introducing pathogens into the milk.

4.1.1 Animal health

The health status of animals has a significant impact on the microbiological quality of raw milk. Major diseases of milking animals include mastitis, an inflammatory disease of the mammary tissue caused by organisms colonising the teat duct and the interior of the udder, where high numbers of microorganisms and somatic cells[12] are shed directly into milk.

However sick and diseased animals may also shed other agents into their milk and/or their faeces, including *Mycobacterium bovis*, *Brucella abortus, Br. melitensis, Br. suis, L. monocytogenes*, salmonellae and *Coxiella burnetii*, and viruses such as Foot and Mouth Disease. In addition to clinically infected animals, animals may be asymptomatic carriers of agents also shedding organisms in faecal material and/or directly into milk.

4.1.1.1 Mastitis

There are over 140 different organisms that can cause mastitis in cows, and they may be found on the cow and in the environment (Nickerson, 2002). However, most mammary gland infections are caused by only a few types of bacteria, including streptococci, staphylococci and coliforms (Nickerson, 2002).

The most important contagious (spread from infected to uninfected cows) mastitis-causing microorganisms are the bacteria *Streptococcus agalactiae, S. aureus* and *Corynebacterium*

[12] Somatic cells refers to white blood cells/body cells from the cow. Somatic cells do not increase after the milk leaves the cow.

bovis. The infected mammary glands are the chief reservoirs of these microorganisms and transmission from cow to cow occurs during the milking process. The infection is commonly subclinical and of long duration, with the microorganisms shed into milk from the infected udders in large numbers (Nickerson, 2002). *S. aureus* and *Corynebacterium bovis* can cause illness in humans, whereas there is debate whether *St. agalactiae* is a human pathogen (Appendix 5).

In the absence of good hygiene*, St. agalactiae* can spread rapidly throughout a herd. However it is easily eradicated from dairy herds through strategic antibiotic treatment. Udders infected with *St. agalactiae* typically have a high somatic cell count resulting in elevated levels in the bulk milk, and the milk itself may be slightly discoloured (Nickerson, 2002).

S. aureus are not commonly found on healthy teat skin, but they readily colonize and grow in the teat canal. Teat skin chapping, resulting in lesions or sores, promotes colonisation and infection. Symptoms are most often subclinical with periodic flare-ups into clinical cases of mastitis requiring treatment. Chronic infections are extremely difficult to treat with antibiotics due to development of scar tissue at multiple sites which impede the antibiotic coming into contact with the bacteria (Nickerson, 2002).

Mammary infections with *Corynebacterium bovis* usually result is only a slight elevation in somatic cell counts in the raw milk. Outbreaks are most commonly reported in herds that do not practise post milking teat dipping and dry-cow therapy (Nickerson, 2002).

Current mastitis control programs are based on hygiene, including pre- and post-milking teat disinfection, antibiotic therapy during lactation and at drying-off and culling of chronically infected cows (Oliver and Pighetti, 2002). *St. agalactiae, S. aureus* and *Corynebacterium bovis* can be controlled by: (1) good udder hygiene, (2) correct use of good milking machines, (3) dipping teats after milking; and (4) treatment of all udders at drying-off (Nickerson, 2002).

Environmental pathogens including streptococci, other than *St. agalactiae*, and gram-negative bacteria, also cause mastitis. These microorganisms gain access to the teat canal and enter the interior of the udder between milkings when teats are exposed to mud, manure and dirty bedding materials (Nickerson, 2002). Most environmental pathogens elicit an elevated somatic cell count in the milk. Control of environmental mastitis pathogens is best achieved by maintaining a clean, dry environment for lactating and non-lactating cows and preventing infection (Oliver and Pighetti, 2002).

Listeria and *Salmonella* mastitis are not common; however, shedding of these organisms in raw milk has been documented via intramammary infections (Wiedmann and Evans, 2002; Poppe, 20052). The incidence of *E. coli* mastitis is low and intramammary *E. coli* O157:H7 infections have not been documented (van Kessel *et al*., 2004).

As mastitis is the cause for elevated cell counts in raw milk, the European Economic Commission has regarded milk or milk products made from raw cow's milk with cell counts above 400,000 cells/mL as unsuitable for human consumption (Directive 92/46). Other importing customers are increasingly using this standard.

Mastitis Control in Australia

The Australian Mastitis Advisory Council comprising dairy farmer and processor peak bodies has initiated a program called Countdown Downunder to help farmers and their advisors achieve mastitis control and reduce cell counts. The industry goal is for more than 90% of all farms to supply milk of less than 250,000 cell/mL and 100% of farms to reach a cell count of less than 400,000 cells/mL. Countdown Downunder encourages farmers to adopt best practice for mastitis control by providing clear, consistent management recommendations to milk harvesting in Australia through guidelines, technotes, seminars, and short courses.

4.1.1.2 Other zoonotic diseases/infections

Sick and diseased animals may also shed other disease agents in the milk including those which cause illnesses. In addition to clinically infected animals, animals may be asymptomatic carriers of agents also shedding organisms in faecal material or directly into milk. Zoonotic microorganisms that can cause illness and disease in animals: *Mycobacterium bovis, Brucella* spp., and *Coxiella burnetii.* These disease agents may be shed in milk, and subsequently cause illness in humans (brucellosis, Q Fever and tuberculosis) (Appendix 5). Although these have declined with the control or elimination of infection in milking animals, the risk of other zoonoses is a constant concern. For example, *M. avium* subsp. *paratuberculosis* (Appendix 5) is currently topical as debate continues regarding a possible link between consumption of contaminated milk and Crohn's disease.

Listeria can cause disease both in humans and animals. Encephalitis, abortion, septicaemia and mastitis due to *L. monocytogenes* has been documented in cattle. The clinical manifestation of listeriosis in sheep and goats include encephalitis, septicaemia, and abortion. Sheep are especially susceptible to Listeriosis. In addition to clinically infected animals, a significant number of animals may be asymptomatic carriers of *L. monocytogenes* often shedding the organism in faecal material (Wiedmann and Evans, 2002).

Salmonellosis is a common intestinal illness caused by numerous *Salmonella* serovars and may clinically manifest itself in animals and humans alike as acute or chronic enteritis, an acute septicaemic disease or as subclinical infections. However, ingestion of *Salmonella* by cattle does not necessarily lead to infection or disease (Torrence and Isaacson, 2003). Animals with subclinical infections or animals recovered from clinical salmonellosis may become carriers, shedding the organism in large numbers in the faeces (Poppe, 20052). Infection rates of 10-15% in dairy cattle have been reported (Poppe, 2003). Cattle infected with *Salmonella* may also only shed the organism intermittently (Kabagambe *et al*., 2000) Foot and Mouth disease (FMD) is a virus which is shed in milk. It is of major concern to the dairy industry because contaminated milk can be the vehicle for animal infection and therefore be disseminated countrywide (Desmarchelier 2001). Australia is officially free from (FMD)[13].

Australia is free from bovine brucellosis[14] (*Brucella abortus*), and bovine tuberculosis *(Mycobacterium bovis)*[15] (Australian Quarantine and Inspection Service 1999; Animal Health Australia 2005a; Animal Health Australia 2005b; Animal Health Australia 2005c).

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13 FMD has not occurred in Australia for more than 120 years, the last reported outbreak was in 1872

14 A national eradication program commenced in 1970, and Australia has been free of bovine brucellosis since 1989

15 A national eradication program commenced in 1968, with Australia being recognised as bovine tuberculosis free on 31 December 1997

Ovine brucellosis (*Brucella melitensis*) has never been reported in livestock in Australia (Australian Quarantine and Inspection Service 1999).

The zoonotic microorganism *Cryptosporidium* can infect animals, the predominant species isolated from dairy cattle being *C. parvum* (Becher *et al*., 2004). Infection in milking animals leads to diarrhoea and shedding in the faeces. *Cryptosporidium* can also cause illness in humans (Appendix 5).

Bovine spongiform encephalopathy (BSE) is a non-inflammatory disease of central nervous system in adult cattle, which originated in the United Kingdom in the 1980s. Australia is currently free from BSE. FSANZ has undertaken a comprehensive risk assessment of the available scientific data and information for BSE and has developed a policy/framework (including a Standard) to manage the risk of BSE in food.

4.1.2 Herd size

Herd size in some cases may have an effect on the prevalence of some microorganisms (*e.g. Salmonella, E. coli* and *Campylobacter*) shed by animals. Large herd sizes in the US (>100 cattle) have been associated with salmonellosis and *Salmonella* shedding in cattle herds (Kabagambe *et al.,* 2000; Huston *et al*., 2002; Fossler *et al*., 2004). Herd size however does not appear to have any affect on shiga-toxin producing *E. coli* (STEC) prevalence(Hussein and Sakuma, 2005).

Higher stocking rates in dairies, compared with other grazing cattle and a large herd size (>100 cattle) have been identified as a possible risk factors for *Campylobacter* prevalence (Wesley *et al*., 2000; Bailey *et al*., 2003; Torrence and Isaacson, 2003). Size of herds in Australia have been increasing, with an average size of 195 cows per herd. However, there are some very large farm operations which may support up to ten individual herds of 500-1,000 dairy cattle.

4.1.3 Age/production status

Age and production status (*e.g.* lactating, dry) may influence the prevalence of some microorganisms in cattle. A higher prevalence of *E. coli* O157:H7 has been found in lactating cows compared to dry cows (Fitzgerald *et al*., 2003) and higher prevalence rates have also been reported in culled[16] dairy cattle. It appears stress may be a factor in the increase in prevalence of STEC in lactating and culled cows. Dairy calves are also more susceptible to STEC infection than older cattle due to their lower immunity to infection (Garber *et al*., 1995; Cobbold and Desmarchelier, 2000). Prevalence has been found to increase in calves before weaning (Hussein and Sakuma 2005).

Becher et al., (2004) studied the prevalence of dairy cattle infected by *Cryptosporidium* at two farms in Western Australia. The combined prevalence rate was 48.1%, with a significantly higher isolation rate of *Cryptosporidium* from calves ≤3 weeks of age. Young calves are considered more susceptible to *Cryptosporidium* infection due to their lowered immunity, which often leads to diarrhoea and can result in the death of the animal (Duffy and Moriarty, 2003).

[16] Cows were selected to represent the distribution of cows within a herd, *i.e.* Lactating, dry, or sick. Cull cows were defined as cows expected to be culled within the next 7 days.

4.2 Environmental factors impacting on milk safety

Pathogens may originate from the dairy environment, for example, housing, faeces from cattle, manure handling, feed, soil, and water. These environmental factors may therefore influence the microbiological quality of raw milk. Dairy farming practices in the southern hemisphere are different to those observed in the northern hemisphere, particularly in the way animals are fed and housed and in general animal husbandry practices.

4.2.1 Housing

In the northern hemisphere, dairy farms generally consist of intensive production systems, with cattle often housed inside during winter to protect the animals from temperature extremes. This practice is not common in Australia (Bailey *et al.,* 2003).

Intensive housing of cattle may increase the risk of contamination of cow udders due to the closer proximity of cows to each other, concentration of faeces, contact with bedding etc. Intensive housing systems also mean increased supplementary feeding, which may also impact indirectly on the microbiological quality of raw milk. Studies have found relatively high levels of *L. monocytogenes* in cattle under these conditions which, may be a reflection of these more intensive production systems (Bailey *et al.,* 2003). Communal housing also appears to be a factor in the prevalence of *E. coli* and *B. cereus* in dairy cattle (Rahn *et al*., 1997; Christiansson *et al*., 1999).

Farm buildings can be directly contaminated with microorganisms following outbreaks of disease in the herd or colonisation of animals, or indirectly from other sources such as contaminated water used for cleaning or access by wild animals (Torrence and Isaacson, 2003). *Salmonella* has been isolated from farm buildings (Torrence and Isaacson, 2003).

Similar effects of stocking density on the microbiological quality of sheep's milk have been reported (Sevi *et al*., 1999). Milk from ewes housed in straw-bedded pens with 2 square metres per animal had lower somatic cell counts and lower levels of mesophiles, psychrotrophs and faecal coliforms than milk from animals housed with only 1 or 1.5 m^2/animal. Mastitis was observed in animals at the higher stocking densities, but not at the lower density.

Australian farming is mainly year round pasture based (*i.e.* animals are not housed indoors), therefore Australia does not tend to experience the same problems seen in intensive farming systems. The level of *Bacillus* spores in raw milk in Australia are slightly lower than that reported overseas, due to this fact (Cook and Sandeman, 2000). The majority of isolates in a Victorian survey closely resembled *B. licheniformis*, which on a worldwide scale has been consistently amongst the most frequently isolated mesophile from raw milk. The next most common isolates recovered in this Australian survey included *B. subtilis, B. pumilus* and *B. cereus* (Cook and Sandeman 2000).

4.2.2 Faeces

The faeces of milking animals may contain a variety of enteric pathogens. Pathogens present in the faeces may result from infection of the milking animal, or through ingestion of the organisms from either feed or water.

Faecal soiling of the hide, hooves and the udder is often unavoidable. Faecal material is widely disseminated in the farm environment and potentially contributes to the farm cycle of foodborne pathogens via faecally contaminated water, feed, soil and inadequately treated

faecal waste used to fertilize pastures. Faecal material can also contaminate raw milk directly from the animal's udder, hide, or hair, thereby introducing pathogens into the raw milk. In addition, faecal material can contaminate raw milk during the milking process if the milking suction cups are kicked off by the cow onto the floor, which may be covered with a significant amount of faecal material. Some of this faecal material can then be subsequently be sucked up by the fallen cups directly into the milk line.

4.2.2.1 Prevalence of pathogens in cattle faeces

The reported prevalence of microbiological pathogens in faeces from dairy cattle, both in Australia and overseas, varies significantly. This refects wide variations in geographic and climatic conditions, animal health, and management practices followed by individual dairy farms.

In general, dairy cattle infected with enteric pathogens will excrete the organisms in large numbers in their faeces. In particular *Listeria, Salmonella*, and *E. coli* are shed in the faeces of infected animals (van Kessel *et al.,* 2004). Infection of dairy cattle with STEC (see Appendix 5, Section 5.11 for definitions of pathogenic *E. coli*) can occur from grazing or consuming forages that were fertilised by contaminated manure. Faecal material can also contain very high numbers of *Bacillus* spores (Cook and Sandeman 2000).

Salmonella serotypes have been frequently isolated from cattle showing symptoms of diarrhoea (Torrence and Isaacson, 2003). Calves are generally more susceptible to *Salmonella* infection, with the mortality rate among calves with salmonellosis reported to be between 19 and 24 percent. The prevalence of *Salmonella* in US dairy cattle faeces is also highly variable, ranging from 2.1-27.5% (Losinger *et al*., 1995; Kabagambe *et al.,* 2000). Cattle that recover from *Salmonella* infection may not become permanent carriers, depending upon the *Salmonella* serotype (*e.g.* excretion of *S*. Typhimurium usually limited to a few weeks or months, whereas animals infected with *S*. Dublin may become permanent carriers (Torrence and Isaacson, 2003).

Prevalence of faecal shedding of *Listeria* spp. in European cattle has been found to vary substantially, with reported prevalence from 2%-52% (Husu, 1990). Faecal excretion of *L. monocytogenes* is very common after clinical listeriosis. The number of *Listeria* isolations from faeces has been suggested to be associated with the prevalence of *Listeria* in feeds (Husu 1990).

Table 3: Prevalence of *L. monocytogenes* in faecal samples from dairy cattle

Country	Prevalence (%)	Reference
Finland	6.7	(Husu 1990)
Netherlands	6.0-15.3%	(Kampelmacher and Noorle Jansen, 1969)
Netherlands	2.0%	(Dijkstra, 1965)
Denmark	52%	(Skovgaard and Morgen, 1988)
Germany	33%	(Weber *et al*., 1995)
Canada	14.5%	(Fedio and Jackson, 1992)
Yugoslavia	19%)	(Buncic, 1991)
Scandinavia	3.1% - spring to autumn on pasture to 9.2% - winter indoors	(Husu 1990)

The prevalence of *C. jejuni* and *C. coli* in dairy cattle ranges from 5-53% depending on methods of isolation, age of animal (calf or adult), season, and sample analysed (faeces or intestinal contents) (Stanley *et al*., 1998). In a US survey, (Wesley *et al.,* 2000) found 37.7% of faecal samples from dairy herds to be contaminated with *C. jejuni*, and 1.8% of faecal samples contaminated with *C. coli*.

Studies on the prevalence of pathogenic *E. coli* in overseas herds concentrate on *E. coli* O157:H7 as this has historically been the STEC serovar of most human clinical significance.

Cattle appear to be a major reservoir of STEC. In a review of reported prevalence of STEC in dairy cattle faeces worldwide, Hussein and Sakuma (2005) demonstrated contamination varied between 0.2-48.8% for O157 and 0.4-47.0% for non-O157 STEC. Faecal excretion of *E. coli* O157 by cattle is considered transient, typically lasting 3-4 weeks (Lejeune *et al*., 2001a) and appears to be seasonal with the highest prevalence seen in cattle in late summer to early autumn (Herriott *et al*., 1998).

Table 4: Prevalence of pathogenic *E. coli* in faecal samples from US and UK dairy cattle

Country	Prevalence (%)	E coli type	Reference
US *cattle* *herds*	0.28% (10/3570) 8.3%	O157:H7	(Hancock *et al*., 1994)
UK *lactating cows* *non lactating cows* *calves*	0.9% 6.3% 9.3%	O157:H7	(Mechie *et al*., 1997)
US	1.2%	Verotoxin-producing O157	(Garber *et al*., 1999)
US	0.3-6.1%	O157:H7	(Kudva *et al*., 1998)

In Australia, the prevalence of STEC in dairy cattle faeces have been found to be similar to those derived in surveys from the Northern hemisphere (Cobbold and Desmarchelier 2000). A study of *E. coli* in faecal samples (n=588) found the prevalence of STEC in Australian dairy cattle was 16.7%. *E. coli* O157:H7 represented 11.2% of the total STEC isolates and *E. coli* O26:H11 represented 10.2% (or 1.9% and 1.7% of total samples respectively) (Cobbold and Desmarchelier 2000). The rate of STEC faecal shedding by Australian cattle in South-east Queensland was higher during weaning. The cattle in this study grazed on native pastures in summer and rye grass in winter with various forms of supplemental feed provided. A wide range of environmental samples were analysed in the study, including a variety of soils, tank water, dam water and sediment, river water and sediment, creek water and sediment, trough water and sediment, slurry and irrigation water samples, brewers' grain, molasses, flies (from the surface of the molasses) and feeds for milkers and weanlings. Evidence of STEC presence was demonstrated in a wide range of water samples on each of the farms. The occurrence of environmental contamination was generally low (Cobbold and Desmarchelier 2000).

In a survey of 25 faecal samples taken from six dairy farms in New South Wales and Queensland (total of 150 samples) undertaken by (Bailey *et al.,* 2003) *Campylobacter* was isolated from all farms, with a median prevalence of 6% (range 0-24%) of faecal samples being positive for *Campylobacter*. *C. coli* was isolated in 4% of samples whereas all other *Campylobacter* isolates were *C. jejuni*. *Listeria ivanovii* was isolated from one sample. No *Yersinia enterocolitica* was isolated from any of the dairy farms studied.

4.2.2.2 Survival of pathogens in faecal material

Foodborne pathogens such as *Salmonella* and enteropathogenic *E. coli* can survive for months in faecal material. *L. monocytogenes* have been found to survive in faeces stored at 5°C for several years (Husu 1990). In the environment, C*ampylobacter* has been found to remain viable at 4°C for up to three weeks in faeces and five weeks in urine (Blaser *et al*., 1980).

Wang *et al*. (1996) studied the survival of *E. coli* O157:H7 in bovine faeces and found that it could survive for up to 70 days when stored at 5°C (initial concentration of 10^5 cfu/g). Survival in faeces stored at 37°C was determined to be 49 days. At these higher temperatures, the faecal samples had low moisture contents (about 10%) and water activities of <0.5.

4.2.2.3 Manure handling

Effluents from dairy farming operations include raw manure, untreated slurry (a mixture of manure, urine, spilt feed, and water that is held without aeration), and treated slurry (aerated) that is filtered to separate the solid fraction from the liquid fraction. Dairy effluent contains many bacteria, viruses and parasite eggs and cysts.

Most large farms wash animal faeces, urine, and spilt feed from milking areas creating a slurry mixture. The slurry is held in settling tanks or ponds away from the milking operation and undergoes anaerobic degradation (untreated slurry) for more than 1 month before disposal. Some farms reduce the bulk of untreated slurry by using a mechanical aeration technique that separates the solid and liquid portions of the slurry. Appropriately treated liquids are released into the environment, while the solids, which occupy less space, are degraded by anaerobiosis before being used as fertiliser.

Where cows are housed, the practice of flushing alleys with water to remove manure appears to distribute faecal flora throughout the cow-housing environment, thus exposing large numbers of animals to faecal material. Herds maintained on farms on which alleyways were flushed with water to remove manure were eight times more likely to have samples test positive for *E. coli* O157 than herds maintained on farms cleaned by use of other methods of manure removal (Garber *et al.,* 1999).

The effluent from dairy farms undergoes primary treatment (generally in anaerobic or aerobic lagoons) before application as fertilizer to land used for silage, grazing, or cultivation. Unless appropriately processed, this effluent is a potential hazard capable of transmitting biological agents. Studies have shown that a variety of conditions in the manure can influence the survival of pathogenic bacteria that subsequently infect livestock. These conditions include temperature, solid content, pH, bacterial concentration, aeration, and the length of time that manure or slurry is held before it is applied to pasture land (Kudva *et al.,* 1998). *E. coli* O157:H7 has been shown to survive for more than 1 year in a non-aerated ovine manure pile exposed to environmental conditions by (Kudva *et al.,* 1998). In similar aerated ovine manure and bovine manure piles, the organism can survive for 4 months and 47 days, respectively (Kudva *et al.,* 1998). Proper aeration for appropriate lengths of time (1-3 months) is required before being used as fertilizers to ensure slurry is not a vehicle for environmental spread and propagation of pathogens (Kudva *et al.,* 1998).

In the past, animal waste and bedding were composted for several days, and the compost reached temperatures of 70ºC or more before being used as fertilizer. Composting and drying of manure is known to reduce the number of viable pathogens. While composting is ideal, it

is generally not a practical approach for processing cattle manure as advancements in mechanised farming have led to large numbers of animals per farm, and faster and more efficient methods for disposal of wastes are required (Kudva *et al.,* 1998).

In Australia, dairy effluent is either spayed direct onto pastures or transferred to a pond system for storage and later irrigation. Storing of effluent in ponds helps to kill pathogenic bacteria and viruses.

4.2.3 Feed

Feed plays an important role as a primary vehicle for contamination of cattle at the farm level and as an indirect source of contamination of raw milk. Major types of feeds fed to cattle are grasses (pasture), silage, grains and concentrate. Contamination of feed may originate from storage on farm or from the source of the feed (including contamination during feed manufacture). Animal feed can be contaminated with pathogens of faecal, plant and soil origin (Desmarchelier 2001).

The potential for faecal contamination in feed exists on the farm. For example, equipment used to clean manure from pens is often used for feed handling, and, on many dairies cattle are fed on concrete slabs that receive vehicle and foot traffic (Lynn *et al*., 1998). Regular cleaning of feed troughs may reduce the potential for on-farm contamination.

4.2.3.1 Grasses (pasture)

Irrigation of dairy effluent onto pastures and crops is widely practiced to increase pasture growth. Therefore poorly treated effluent may present a risk of microbial contamination to pastures and crops. Manure fertilizers may also contaminate grasses.

Use of recycled water or reclaimed water (*i.e.* water derived from sewerage systems) must be suitable treated to a standard that is suitable for its intended use.

Listeria contamination in grasses was found to be, to a large extent dependent on their moisture content. Hay, with very low moisture content, was found to be free of Listeria. While in a study by (Fenlon *et al*., 1996), no *L. monocytogenes* was found in pasture at time of harvest, it was detected within 24 hours of cutting (9/10 samples). *C. jejuni* was more frequently recovered in herds where alfalfa or whole cottonseed or hulls were fed (Wesley *et al.,* 2000).

4.2.3.2 Silage

Silage is a moist conserved fodder produced as a foodstuff for cattle. The fodder in silage is naturally preserved by lactic acid fermentation of sugars by bacteria after the fodder has been wilted and stored to exclude air. Silage has a high moisture content and is preserved by the combined effects of a rapid pH reduction to pH 4.2 and storage under anaerobic conditions. Silage is used during seasons when fresh forage is unavailable.

Traditionally, silage was prepared in bunkers, pits, or large concrete or steel silos on farm. The recent trend has been to produce silage in large bales, which are sealed by wrapping in plastic. Silage has higher moisture content than hay and is only microbiologically stable when anaerobic conditions are maintained. Such conditions prevent the growth of aerobic spoilage organisms, while the lactic acid fermentation provides acidity that inhibits the growth of any anaerobic spoilage organisms or pathogens such as *Cl. botulinum*.

Undesirable microorganisms can enter the silage storage by soil and livestock waste during harvest or increase in numbers when the silage storage environment is suitable. Hazards include *Enterobacteria, Listeria, and Clostridia* (Mickan 2002). Both *Bacillus* spp. and *Listeria* spp. have also been isolated from silage (Husu 1990; Sutherland and Murdoch, 1994).

Microorganisms from contaminated silage can spread to the alimentary tract of the animal being fed, resulting in the same organisms being present in the faeces (Fenlon *et al.,* 1996). In addition large numbers of spores in silage may lead to infection of dairy cattle and subsequent excretion of high numbers in faeces (Cook and Sandeman 2000).

E. coli and other *Enterobacteriaceae* and *Bacillus* and *Clostridia* species are a risk when effluent is applied to paddocks closed for silage production. Effluent sprayed onto these paddocks must be well washed in by rain before harvesting (Mickan 2002).

A slow fermentation favours the growth of enterobacteria in the early phase of fermentation as they compete more strongly with the desirable lactic acid bacteria for water-soluble carbohydrates (Mickan 2002).

L. monocytogenes is found in soil, faeces and rotting vegetation and can reproduce at low temperatures as well as in heating silage. *L. monocytogenes* is a ubiquitous organism and is likely to naturally occur in plant materials used for silage preparation and/or in contaminating soil. It is more commonly found in the outer layers of baled silage although it may occur in the layer just below the plastic sheet in chopped stack silage. Damage to the plastic wrapping or ineffective sealing of the silage may result in aerobic spoilage, and this is known to create conditions highly selective for the growth of *L. monocytogenes* and very high numbers of the organism can be present (Fenlon, 1986). Under these circumstances, a combination of rising pH, moisture, and slow air ingress favour the growth of *Listeria*.

Faecal contamination from birds and other animals, especially rodents, which can be asymptomatic carries of *Listeria*, can also contribute to *Listeria* contamination of silage (Wiedmann and Evans, 2002). The occurrence of *L. monocytogenes* in poor quality silage is well documented (Husu 1990). Correctly fermented silage from contaminated grass has been shown to contain little *Listeria* contamination, whereas spoiled silage and residues of silage remaining in feeding troughs had higher levels, up to 1.5×10^5 cfu/g^{-1} (Fenlon *et al.,* 1996).

Silage appears to be the most likely source of infection of listeriosis in cattle and sheep. Goats that are grazed and not exposed to any silage have been known to develop listeriosis, most likely through abrasion to the mouth by rough grazing materials providing an entry port for the organism (Wiedmann and Evans, 2002).

Spores of *Clostridia* can survive the passage through the alimentary tract of the dairy cow, and are subsequently transferred to milk via faeces, mainly though faecal contamination of the udder (Driehuis and Oude Elferink, 2000). The occurrence of clostridial spores in milk however mainly impairs its quality; the species most relevant for the dairy industry is *Cl. tyrobutyricum*. Poor silage quality can also lead to high clostridial spore levels in raw milk (Vaerewijck *et al*., 2001). *Cl. botulinum*, is the cause of botulism and may present a health hazard to animals fed silage. However, *Cl. botulinum* has a limited acid tolerance and does not grow in well fermented silage (Driehuis and Oude Elferink 2000). However risk of *Cl. botulinum* is increased substantially if the crop is contaminated with animal remains. *Cl.*

botulinum has been isolated from silage to which poultry manure has been applied (Mickan 2002).

Proliferation of *Bacillus* spp. (*B. cereus, B. lentus, B. firmus, B. sphaericus, B. licheniformis,* and *B. polymyxa)* usually occurs during the later stages of aerobic spoilage of silage. High numbers of *Bacillus* spores have been detected in the surface layers of grass and maize silage (Driehuis and Oude Elferink 2000).

In Australia, silage is now a major component on many farms as a feed resource and pasture management tool. Considerable areas of Australia are devoted to forage crops and pastures, which are either used for grazing or harvested and conserved as hay or silage. (Kaiser and Piltz 2 A.D.). Australian silage has been found to contain very high numbers of *Bacillus* spores (Cook and Sandeman 2000).

4.2.3.3 Concentrates and grains

Feed concentrate is a dried, pelleted supplementary feed ingredient for cattle. It can be composed of different ingredients such as grains or cereals (corn and barley), maize gluten feed, citrus pulp, soy bean meal and manioc or coconut cake meal (Vaerewijck *et al.,* 2001). Concentrates, particularly grain mixes which are commonly based on cereal grains such as barely, and may include other ingredients such as lupins, canola, cottonseed, maize, oats, sorghum, soybean, sunflower, rice and wheat; carrot and citrus pulps; potato by-product; and whey, are fed as supplements to pasture in Australia (Department of Primary Industries 2005).

In Australia, grain is fed to supplement dairy cattle either as “straight”, “cracked” or “rolled”, or as a mix of cracked or rolled. Grain is more often fed to dairy cattle rather than as a component of pelletised concentrates. The amount of supplementary feed provided to Australian dairy cattle varies throughout the year; 10 kg or more per cow per day of feed concentrates are fed in summer when no green pasture is available, with little or no supplementary feeding in spring when there is sufficient green pasture.

Faecal contamination of concentrates and grain may occur prior to delivery of feeds to the farm and can be due to faecal contamination preharvest (through manure application to crops) or post harvest by contamination from bird or rodent faeces during storage or shipment. Concentrated feed can be contaminated with *Salmonella, E. coli,* and *Campylobacter* (Weis and Seeliger, 1975; Kabagambe *et al.,* 2000; Torrence and Isaacson, 2003).

B. cereus has been isolated from feed concentrates (te Giffel *et al*., 1995; Slaghuis *et al*., 1997; Christiansson *et al.,* 1999; Vaerewijck *et al.,* 2001), including grains and pellets in Australia (Cook and Sandeman 2000). However, feed concentrates have not been shown to be a source of *Listeria* contamination as most are subjected to heat treatment during manufacture, particularly the pelleted variety, and contain moisture levels which are too low to sustain *Listeria* growth (Fenlon *et al.,* 1996). Table 5 lists the prevalence of some pathogens in feeds, and Table 6 lists the incidence and level of *L. monocytogenes* contamination in various habitats.

Table 5: Prevalence of pathogens in feed

Country	Feed type	Organism	Prevalence/levels	Reference
Netherlands	Feed concentrate	*B. cereus spores*	10^1-10^2 spores g^{-1}	(te Giffel *et al.,* 1995)
US	Feed concentrate	*B. cereus spores*	<10-10^3 spores g^{-1}	(Slaghuis *et al.,* 1997)
Sweden	Feed concentrate	*B. cereus spores*	<150-850 spores g^{-1}	(Christiansson *et al.,* 1999)
Belgium	Feed concentrate	aerobic sporeformers	4.0 x 10^3 - 1.1 x 10^6 g^{-1}	(Vaerewijck *et al.,* 2001)
UK	Silage	*B. cereus*	10^5 cfu g^{-1}	(Crielly *et al*., 1994)
Finland	Grass Silage	*L. monocytogenes*	15.6%	(Husu 1990)
Finland	Pasture grass	*L. monocytogenes*	38.2%	(Husu 1990)
US	Feed concentrate	*E. coli* *E. coli* 0157	30.1% 0%	(Lynn *et al.,* 1998)
Australia	Feed concentrate	*B. cereus spores*	1.1 x 10^2-6.3 x 10^2 g^{-1}	(Cook and Sandeman 2000)
Australia	Silage	*B. cereus spores*	7.2 x 10^4-3.4 x 10^5 g^{-1}	(Cook and Sandeman 2000)

Table 6: Incidence and level of *L. monocytogenes* contamination in various habitats (Fenlon *et al.,* 1996)

Date	Sample/site	Positive samples (Total no. samples)	Level (g^{-1})
March 1993	Soil field 1	1 (3)	Present
	Soil field 2	2 (3)	0.9 Present
	Soil field 3	1 (2)	Present
	Silage (field 1) (spoiled)	2 (2)	4.6 x 10^4 >2.3-<20
	Silage (field 2) (spoiled)	1 (2)	1.5 x 10^3
	Cattle faeces (silage field 1)	1 (1)	>2.3-<20
	Cattle faeces (silage field 2)	4 (4)	0.4 3.6 x 10^2 5.0 x 10^2 20
July 1993	Cattle faeces (grazing)	3 (10)	0.4 0.4 Present
	Stored 1year-old used silage bags	1 (6)	Present
February 1994	Following season silage	4 (6)	8.1 x 10^4 1.0 x 10^3 2.0 x 10^3 Present

4.2.4 Soil

Soil represents an important source of pathogens for grazing animals. A wide variety of organisms, including pathogenic bacteria, may be found as typical soil microflora, plus faeces and urine from grazing animals may contaminate soil by the application of fertilisers and effluent.

Bacillus and Clostridium species are commonly found in soil. The levels of *B. cereus* in soil has been found to vary from <50-380,000/g (Christiansson *et al.,* 1999). In Australia, levels of *Bacillus* spores have also been found at levels between 5.6 x 10^2-1.8 x 10^3 cfu/g (Cook and Sandeman 2000).

L. monocytogenes is a ubiquitous organism that is naturally found in the soil and thereby finds its way into grasses and silage made from pastures. Soil contaminated with *L. monocytogenes* (and other microorganisms) can be inadvertently transferred into the milking parlour, from farms to factories via milk tankers, and on the feet of employees.

Foodborne pathogens such as *Salmonella* and enteropathogenic *E. coli* can survive for months in contaminated soil (Desmarchelier 2001).

4.2.5 Water – stock drinking

Water is used extensively on dairy farms for cleaning, cooling, stock drinking and irrigation. Dairy cattle consume large amounts of water daily. Running water and water from wells, bores and large fenced dams are generally less contaminated than stagnant water supplies, particularly those to which cattle have free access (Agriculture Western Australia 2005). Sediment in water can support bacterial growth and may be a reservoir for pathogenic microorganisms. Depending upon the water source, water may contain high levels of bacterial contamination even before it enters water troughs. High spore counts have been observed in Australian dam water, probably due to run off from paddocks containing a high level of suspended soil and organic material (Cook and Sandeman 2000).

E. coli O157:H7 has been reported to survive in pond water at 13ºC and in river water at 18ºC for 20 and 13 days respectively (Wallace, 1999) and can survive in trough sediments for 4 months and in water for 8 days at 5ºC (Rice and Johnson, 2000).

Water may be a significant reservoir of *Campylobacter*, where it has been shown to be able to remain viable for up to 4 weeks at 4ºC (Blaser *et al.,* 1980). Transmission of *C. jejuni* has been observed from groundwater to dairy cattle (Stanley *et al.,* 1998).

Salmonella and *E. coli* O157 were detected in water troughs with a prevalence of 0.8% and 1.3% respectively (Lejeune *et al*., 2001b). However it has been reported that *E. coli* O157 could be repeatedly isolated from environmental sources on farms and be present in as many as 10% of water troughs (Lejeune *et al.,* 2001b). Water troughs can become contaminated with cud and/or faecal material. Extraneous matter including dust, feed, or bedding may also enter the trough.

The position of water troughs on farm may also affect prevalence of *E. coli*. It has been demonstrated that bacterial contamination is higher in troughs that are closest to the feed troughs (Lejeune *et al.,* 2001b). Water troughs close to feed bunks (<7.62 m) have been shown to have higher numbers of *E. coli* O157 than those placed further away (Lejeune *et al.,* 2001b). Metal troughs also have shown lower *E. coli* O157 counts compared with troughs that were manufactured from concrete or plastic (Lejeune *et al.,* 2001b). Water trough sediments contaminated with faeces from cattle excreting *E. coli* O157 therefore may serve as a long-term reservoir of this organism on farms and a source of infection for cattle (Lejeune *et al.,* 2001a).

In addition *E. coli* contamination of cattle has been found to be positively associated with the protection of the water trough from direct sunlight, lower concentrations of protozoa in the trough water, and warmer weather (Lejeune *et al.,* 2001b). Frequent cleaning of water troughs and/or treatment of drinking water with chlorine, UV light, or ozone to kill or inactivate pathogens will also reduce the potential for replication and/or survival of pathogens.

Wet and muddy conditions around drinking points may result in contamination of the legs, udder and tail of cattle with mud and faecal material. This increases the risk of bacterial infections of the teat and udder, leading to environmental mastitis, and high bacterial counts on the udder and teats leading to possible contamination of raw milk during milking. It is important, therefore, to maintain the surroundings of watering points in a dry, solid and stable condition to prevent them becoming boggy and muddy.

4.3 Microbiological monitoring and status of Australian milk

The composition and hygienic status of raw milk is determined by a number of tests on arrival at the dairy processing plant. The outcome of these tests has a direct bearing on the money paid to the farmer under performance payment schemes. Monitoring of cell counts may also occur on farm, and this provides an indirect way of estimating the level of subclinical mastitis in the herd and can trigger action to improve or better manage herd health and to improve milk yields. Early detection and treatment of sick animals reduces the risk of severe and intractable cases developing and reduces the likelihood of infection being passed to other cows.

Milk processors, industry associations and programs in Australia, such as Countdown Downunder, provide guidance, assistance and technical support to dairy farmers to ensure consistent milk quality and safety.

Bulk milk cell counts and herd milk cell counts[17] are monitored by *Countdown Downunder* (a program initialled by Australian Mastitis Advisory Council to help farmers and their advisors achieve mastitis control and reduce cell counts). The industry goal is for more than 90% of all farms to supply milk of less than 250,000 cell/mL and 100% of farms to reach a cell count of less than 400,000 cells/mL (Table 7).

Table 7: Australian milk herd cell counts (Dairy Australia 2005)

Year	Herd Milk Cell Counts below 250,000 cells/mL Goal 90%	Herd Milk Cell Counts below 400,000 cells/mL Goal 100%
2000	64%	91%
2004	71%	95%

[17] Herd milk cell count refers to somatic cell count of individual cows in every herd aggregated into a volume-weighted average for the herd.

During the past three years, the inclusion of cell counts in the buying standards and payment schemes of dairy companies has become universal in Australia. Payment schemes are typically based on milk volume and composition, with bonuses or penalties for conformance with specific milk quality indicators (e.g. Bactoscan, Bulk milk cell count (BMCC)[18], total plate counts, thermoduric count, and sediment). Dairy companies differ in the thresholds they use to define premium milk payments depending on the types of products being manufactured (Tables 8-11). Premium payments are typically about one cent per litre extra. In some states there is a ceiling BMCC above which milk processors will not collect the milk. Milk processors report the results of bulk milk cell counts to farmers.

The majority of Australian milk meets the premium milk quality grade 'Band 1' (Figure 1).

Table 8: Milk quality standards for Bactoscan for Australian processors (Dairy Australia, 2005)

Processor	Band 1 (Premium)		Band 2	Band 3	Band 4
A	<80		81-200	201-600	>600
B	<200		201-500	501-2000	>2000
C	<160		160-218	219-493	>493
D	<80		81-200	>200	
E	<51	51-80	81-200	201-1000	>1000

Table 9: Milk quality standards for Bulk Milk Cell Count for Australian processors (Dairy Australia, 2005)

Processor	Band 1 (Premium)		Band 2	Band 3	Band 4
A	<250		251-400	401-800	>800
B	<250		251-600	601-800	>800
C	<250		251-400	401-800	>800
D	<300		301-600	>600	
E	<250	251-350	351-600	501-700	>700

Table 10: Milk quality standards for thermodurics for Australian processors (Dairy Australia, 2005)

Processor	Band 1 (Premium)	Band 2	Band 3	Band 4
A	<1500	1501-3000	3001-6000	>6000
D	<5000	5001-20000	>20000	
E	<2000	2001-5000	5001-10000	>10000

Table 11: Milk quality standards for sediment for Australian processors (Dairy Australia, 2005)

Processor	Band 1 (Premium)	Band 2	Band 3	Band 4
A	Absent			Present
B	<0.5		0.6-2.0	>2.0
C	Advisory			
D	Disc 1-2		Disc 3-4	

[18] Bulk milk cell count refers to the concentration of somatic cells in the total volume of milk in the milk vat.

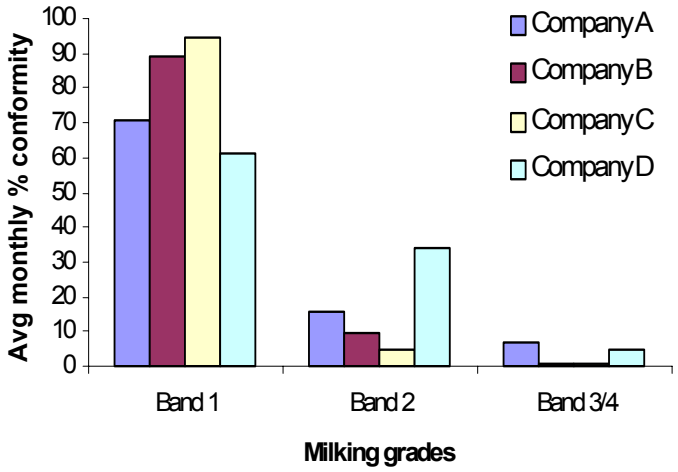

Figure 1: Average monthly percentage of suppliers for each Milk Quality Grade 2004/2005

4.4 Effect of milking on milk safety

4.4.1 Milking practice

4.4.1.1 Teat washing

Poor milking practices may lead to contamination of raw milk. The teat surface is the major avenue of entry of microorganisms into raw milk. It is well recognised that there is significant opportunity for teats to become contaminated by faeces and soil (as dust or mud) (Cook and Sandeman 2000; Vaerewijck *et al.,* 2001). If not removed before milking, this dirt, together with the large number of microorganisms associated with it, may be washed into the milk during milking. Contamination of teats is less when cows are pasture-based rather than intensively housed (Slaghuis *et al.,* 1997).

Significant relationships have been observed between the number of *Bacillus* spores isolated from the surface of the teat, as well as from the surface of cup liners, with the number of spores found in bulk raw milk. These observations were related to the cleanliness of the teat at milking, and may represent accumulative build-up of dirt and bacteria from the teats in milking equipment (Cook and Sandeman 2000). The contribution of *B. cereus* from the exterior of the udder has been shown to decrease following cleaning and disinfecting the animal surface before milking (Christiansson *et al.,* 1999).

In Australia, washing cow teats is only recommended if they are dirty. If teats are washed, it is important that they are dried to minimise the risk of the animal developing mastitis (Brightling *et al*., 2003). If teats are left wet, more bacteria may be found on the teats (Slaghuis *et al.,* 1997). Hence, minimising the use of water on udders and teats is beneficial for teat skin health and also generally leads to better milk quality (reduced coliform counts, sediment, etc) unless very careful drying techniques are used. Research has shown that there is no reduction in milk quality when teat cups are applied to visibly clean, dry teats (Hubble and Mein, 1986).

4.4.1.2 Teat disinfection

Post milking teat disinfection is an effective procedure for reducing the rate of subclinical and clinical mastitis during lactation (Slaghuis *et al.,* 1997). Teat disinfection helps keep teat skin healthy and heal skin lesions, and these actions may be its most important contribution to mastitis control (Hillerton, 1997). The majority of Australian dairy farmers rely on post-milking teat disinfection, applied by spray techniques, as an integral part of their mastitis control programs (Lee, 1994).

One of the biggest variables in successful use of teat disinfection is the quality and consistency of application. Failure to cover the whole teat of every cow at every milking is the most common error in teat disinfection. Disinfectant is applied by dipping each teat separately in a cup or spraying disinfectant on to the teats from below. Dipping has the advantage that complete coverage of the teat barrel is fairy easy to achieve. Spraying disinfectant often coats one side of each teat only. Spraying is the method of teat disinfectant application in most Australian herds as it is quicker and easier to incorporate into milking routines.

The effectiveness of teat sprays is dependent on the quality of water used to make up the disinfectant. Water quality characteristics that alter the bacterial killing power of teat disinfectants include alkalinity, water hardness, organic matter and chlorine concentration. Countdown Downunder recommends using cooled hot water to minimise the bacterial load (Countdown Downunder, 2000).

The Rural Industries Research and Development Corporation recommends that mastitis in sheep be minimised by disinfecting the teats after each milking, which prevents the entry of mastitis-causing bacteria into the teat canal, which is naturally enlarged after milking (Bencini and Dawe 2005).

4.4.1.3 Milk segregation

In Australia, colostrum milk must be withheld from the vat for at least eight milkings after calving. Colostrum has a high number of somatic cells (not due to mastitis) and its changed composition has a significant effect on the processing efficiency of dairy products.

Similarly, milk derived from cows treated with antibiotics must be segregated from the bulk milk collection system. The approval of veterinary medicines is strictly controlled in Australia, and their use must be under the supervision of a veterinarian. Withholding periods for antibiotics must be strictly adhered to, resulting in a minimum period of time that must elapse between the last treatment of an animal with a veterinary medicine and the supply of milk from that animal for food consumption.

4.4.1.4 Cooling and filtration of milk

The composition of milk makes it an excellent growth medium for many microorganisms unless it is frozen or further processed to kill or prevent their growth. As the temperature of raw milk as it leaves the udder is around 37ºC pathogenic bacteria, if present, will grow rapidly. At temperatures between 0 and 5ºC, growth of pathogenic and spoilage mesophilic bacteria are slowed. Therefore, after milking raw milk should be cooled to below 5ºC and stored refrigerated until collection to minimise the potential growth of microorganisms.

As the milk leaves the udder, it is initially cooled by passing through a heat exchanger (plate cooler) prior to it entering the refrigerated milk vat (bulk milk storage tank). It is further cooled in the vat. Any breakdown in the refrigeration system, or failure to properly cool milk prior to collection may impact on the quality and bacterial load in raw milk.

Filtration of milk before it enters the bulk milk tank provides a safeguard to ensure sediment or other extraneous matter is removed from the milk prior to storage. Although filtration removes most of the soil and other particles, it does not remove all the bacteria adhering to these particles, nor does it remove any dissolved matter from the milk. Sediment that has been trapped by the filter continues to be washed by the milk flowing through. This dislodges bacteria adhering to the particles and thus contributes to an increased bacterial count in the bulk milk. If filters are not adequately cleaned, this process may be a source of cross-contamination of the milk.

Forcing milk through an in-line filter by pump is the most common method of filtering milk in modern milking plants. The filters may be made of various elements including paper, fibre or cloth and they fit over a perforated metal support or cage within a cylindrical tube.

4.4.1.5 Cleaning and sanitation

There are various methods for cleaning milking parlours/yards. Walls and floors are typically cleaned using a combination of manual scraping or brushing and then rinsed using hoses with water under medium or high pressure. Some areas are also fitted with automated cleaning systems that clean the floor of the milking parlour and/or holding areas. This may be accomplished by periodic application of water under pressure or by a flush system in which a large volume of water is released to create a wave of water on floor surfaces that carries manure and urine to a collection pit. However care must be taken in automated systems that flush water does not contaminate milking equipment.

4.4.2 Water use in milking

Water is used extensively during the milking process and may be a possible source of contamination if it is of unacceptable quality. Water is used for teat cup washing, washing of cows teats, milking plant flushing and rinsing, milk vat flushing and rinsing, milk pre-cooling, and teat disinfection.

The greatest risk of milk contamination from water is from water used to flush the milking plant following milking.

A qualitative assessment undertaken by Dairy Australia, estimates the risk of microbiological contamination of milk via water in Australian dairy farms to be negligible. However, the use of untreated water to flush the milking plant following milking was found to be of a slightly higher risk (low/medium) (Dairy Australia, 2004).

4.4.3 Milking equipment

Milking equipment and methods have an important effect on both animal health and the microbiological status of raw milk.

Cows with mastitis are responsible for elevated levels of somatic cells and bacterial cells in raw milk. The use of milking equipment is estimated to lead to about 20-25% of mastitis infections in Australia (Countdown Downunder 2003). The main milking-related mechanisms of spread of mastitis infections include:

- spreading of organisms via contaminated liner surfaces, milker's hands and teat lesions;
- impacts and possible reverse pressure gradients may assist the passage of organisms into the teat canal;
- teat damage and loss of keratin lining of the teat canal can lead to a decrease in the natural effectiveness of the teat canal as a barrier; and
- less frequent or less complete emptying of the udder.

Proper sanitation and prevention of faecal contamination of equipment during milking is critically important. Contamination of raw milk can also occur from equipment used for milking, filtering, cooling, storing, and distribution of milk. Milk handling equipment contributes a large proportion of raw milk microflora (ICMSF, 1998). Poor cleaning and hygiene standards in the waiting area and at milking; wet udder preparation using one towel for many cows; failure to apply fore-milking; and poor maintenance of milking equipment can lead to contamination of raw milk with pathogens (Sanaa *et al*., 1993).

Milk residues left on equipment surfaces after inadequate cleaning provide nutrients and high ambient temperatures favour the growth of microbial contaminants. Surfaces often remain wet for long periods, permitting build-up of microorganisms that adhere to equipment surfaces. During subsequent use of equipment, these microorganisms can contaminate milk. The type and number of organisms introduced from milking equipment is largely dependent on the efficiency of cleaning and disinfecting. Bacteria will proliferate in milk residues left on equipment and increase rapidly if milk is cooled slowly, or inadequately (National Milk Harvesting Centre 2000).

Cleaning of milk handling equipment involves a combination of chemical, thermal and physical processes. The key principles of a good cleaning system involves sufficient hot water (temperature and volume), correct wash solutions (detergent), adequate contact time and sufficient turbulence to prevent build up of milk residues and bacteria in the equipment.

Table 12: Mean number of *Bacillus* spores present in environmental and bulk milk samples from Cobden and Stanhope, Victoria (Cook and Sandeman 2000).

Sample	Cobden	Stanhope		
	Mesophilic spores	Mesophilic spores	Thermophilic spores	Anaerobic spores
Bulk milk (cfu/mL)	73	7	2	7
Teat skin (cfu/mL)	7.8×10^3	2.2×10^3	7.0×10^2	1.9×10^3
Cupliners (cfu/mL)	1.3×10^3	82	25	48
Foremilk (cfu/mL)	1.2×10^2	13	4	8
Hot water (cfu/mL)	25	1.0×10^2	1.1×10^2	3.8×10^2
Cold water (cfu/mL)	23.5×10^2	3	1	3
Faeces (cfu/g)		6.8×10^2	1.3×10^3	8.6×10^2

4.4.4 Hygiene of milking personnel

Personnel in direct contact with cows and milking equipment are also a potential source of contamination. Milk handling personnel may contribute various organisms, including pathogens, directly to milk. Micrococci and staphylococci from skin and upper respiratory tissues may gain entrance, especially during hand milking. Workers with illness and/or infections who come into direct contact with dairy equipment can also introduce contamination.

4.5 Raw milk collection and transport to processors

Raw milk must be adequately protected during transport to prevent any further contamination. It is also important to ensure milk is kept cool to prevent the growth of organisms. Cleaning of milk tankers is a critical process to minimise contamination and to maintain a high quality supply of raw milk.

In Australia raw milk is collected from the farm every 12-48 hours in heavily insulated stainless steel tankers. Collection interval depends on herd size, size of the milk vat, and season. In Australia, there is a trend to collect milk less frequently due to financial incentives offered to farmers by milk processors.

Milk is unloaded into insulated silos at the processing facility before results of microbiological testing and somatic cells counts are known. Typically, these silos contain milk from a series of tankers, and this results in significant dilution effects where milk of high bacterial count is mixed with high quality milk. As these silos are often not refrigerated, the temperature of the milk will be reflect the temperatures achieved on farm.

Trucks are cleaned and sanitised at the dairy plant or at an intermediate wash station.

4.6 Summary of major primary production risk factors for milk production in Australia

There are two means by which pathogens contaminate raw milk. Contamination may occur when microorganisms are shed directly into raw milk from the udder through illness or infection of the animal, or through contamination from the external surface of the cow and the milking environment. However there are many factors that impact on these routes of contamination. Table 13 summarises the major risk factors in the production of milk.

Table 13: Major risk factors in production of milk

Risk factor	Effect	Control
Animal health	Disease in, sickness of, and carriers in milking animals can increase shedding of pathogens directly into raw milk, or in animal faeces.	Animal health and mastitis programs
Herd size	Herd size may have some effect on the prevalence of some pathogens (*e.g. Salmonella, E. coli* and *Campylobacter*)	Biosecurity and animal husbandry
Age/ production	Calves have an increased susceptibility to infection, and have been reported to have higher prevalence rates of some pathogens (*e.g. E. coli*)	Calves kept separate from milking herd
Housing	Intensive housing practices may increase risk of contamination of udders due to close proximity of animals, concentration of faeces, bedding etc. This has been shown to be a factor in the prevalence of *Bacillus spp., E. coli,* and *L. monocytogenes*	Australian dairy farming is mainly pasture based
Faeces	Faeces may contain various pathogens – reflecting either illness/infection, or through ingestion of contaminated feed and/or water with faeces. Faeces may contaminate the exterior of the udder and introduce pathogens into raw milk.	Udder hygiene at milking
Effluent	Effluent (containing manure) can also contaminate pasture.	Appropriate treatment and disposal of effluent
Feed	Contamination of feed can lead to shedding of pathogens into faeces. Poorly made silage can be a source of pathogens (*e.g. E. coli, Bacillus spp., Listeria,* and *Clostridia*).	Control over preparation and storage of feed, especially silage
Water – stock drinking	Water is a potential source of contamination. Sediment in water can support bacterial growth and be a reservoir for pathogens. Water sources can become contaminated with cud and/or faecal material, feed, etc.	Ensuring water is of suitable quality
Milking	Poor milking practices, including dirty teats, inadequate cleaning and maintenance of milking equipment, and poor personnel hygiene can lead to contamination of raw milk.	Maintenance, sanitation and cleaning of equipment, appropriate animal and good personal hygiene
Water use - milking	Water is a potential source of contamination during washing of teats and cleaning of milking equipment.	Ensuring water used is of suitable quality
Storage	Inappropriate temperature control of milk after milking can lead to growth of pathogens	Rapid cooling of milk and regular collection.
Transport	Inappropriate temperature control of milk during transportation can lead to out-growth of pathogens. Contamination can occur if tankers do not adequately protect milk, and/or are inadequately cleaned.	Temperature control, tanker maintenance, and cleaning and sanitation

5. Processing and the impact of processing on milk and milk products

5.1 Processing of milk and milk products

After transport from the dairy farms, raw milk is pumped from the bulk milk tanker into insulated silos at the processing plant. After initial screening and testing, the raw milk is processed into a diverse range of dairy products using a range of processes and technologies.

Processing of dairy products is intended to:

- produce microbiologically safe products of acceptable shelf-life;
- develop or maintain desired sensory qualities (appearance, flavour, texture); and
- isolate particular constituents of milk which are used directly or as part of other foods or for non-food purposes.

In order to produce microbiologically safe dairy products, processing of raw milk requires a microbiocidal processing step to eliminate, remove or destroy any vegetative pathogens present. Heat treatment (i.e. pasteurisation) applied by an appropriate time/temperature combination is traditionally used as the key microbiocidal step in the manufacture of dairy products. However this processing step reduces the number of microorganisms only at the point in the manufacturing process where it is applied, and its effectiveness in terms of end product safety depends on the initial microbial load of the raw milk, the effect of any post-treatment contamination and/or growth, and the implementation of further control measures.

Pasteurisation of raw milk is not normally applied as the sole control measure but is used in combination with a number of preventive measures (hurdles[19]). For example pasteurisation is followed by packaging and refrigeration of liquid milk while heat treatment, fermentation, salting and aging are applied in the manufacture of cheese.

Microbial growth is dependent upon many conditions such as nutrients, water activity, pH, temperature, presence of preservatives, competitive microorganisms, and atmospheric conditions. Control of these conditions can therefore be used to limit, retard or prevent microbial growth.

Therefore depending on the type of dairy product being manufactured, the processing methods and hurdles employed may include:

- heat treatment (thermisation, pasteurisation, ultra-high temperature (UHT) sterilisation;
- cold treatment (chilling/refrigeration, freezing);
- mechanical treatment (separation, centrifugation, homogenization, filtration, agitation);
- removal of water (concentration, dehydration, curing/ageing);
- microbiological or biochemical fermentation (acid production, lipolysis, proteolysis); and/or
- combinations of these methods.

Detailed descriptions of the equipment and processes employed to convert liquid milk into processed dairy products may be found in the ***Dairy Processing Handbook*** (published by

[19] The ***hurdle concept*** (Leistner and Rodel, 1976) describes the effect of multiple factors (e.g. temperature, pH, water activity) on microorganisms. Several different hurdles at sub-optimal levels can be used to control the growth of microorganisms in food products, rather than a single, severe hurdle.

Tetra Pak processing Systems, Sweden). The effect of specific processes on microbiological hazards in various dairy commodities is discussed in more detail in Appendix 1. A brief summary of the findings in Appendix 1 is presented in Table 14.

Table 14 Impact of processing on microbiological safety in various dairy commodities

Dairy product	Processing and impact on microbiological safety
Milk and cream	Milk is derived from mammalian animals e.g. cows, sheep, goats, etc. Cream is produced from whole milk by separation. The main steps in milk and cream processing include filtration; homogenisation; and heat treatments e.g. pasteurisation, sterilisation and UHT processing. Pasteurisation is sufficient to destroy the most significant milk-borne vegetative bacteria.
Cheese	Cheese making normally begins with heat treatment of milk, followed by addition of starter culture and rennet, resulting in production of a cheese curd through coagulation and acidification. Mild heating separates the whey, which is drained away. Curds are salted, pressed into moulds and ripened under controlled conditions. A number of processing factors influence the growth and survival of pathogens in cheese, including the severity and duration of heat treatment (including curd cooking); pH; salt concentration; water activity; and maturation/ripening.
Dried milk powders	Liquid milk is initially concentrated and spray-dried to form a powder. Microorganisms associated with dried milk powders will not grow, however, they may survive for long periods of time and resume growth when the powder is reconstituted and stored under favourable conditions. The presence of microorganisms depends on factors such as the bacterial load in the raw milk, preheating temperatures, operating conditions of the evaporator and dryer, and plant hygiene. Post-process contamination is a major factor impacting on the contamination of milk powders. The major factors affecting the survival of pathogenic microorganisms are evaporation and drying.
Infant formulae	Infant formulae are a sub-set of dried milk powders. Formulae may contain milk, soy protein or protein hydrolysates, fat, carbohydrate, vitamins and minerals. These products are either manufactured in powdered form through evaporation and spray-drying, or in liquid form, followed by a high temperature treatment. Microbial pathogens in powdered formulae are the same as those for dried milk powders and as such are not able to grow due to the absence of water.
Concentrated milk products	Evaporated and sweetened condensed milks are both manufactured from milk and sugar that are heated, evaporated and homogenised. Evaporated milk receives a UHT or sterilisation treatment before cooling and packaging, while condensed milk is seeded with lactose, cooled and packaged. Pathogens are generally not associated with these milks due to their low water activity.
Butter	Butter is produced from pasteurised cream. Churning the cream produces butter. Growth of microorganisms in salted butter is unlikely, due to it's moisture distribution and salt content.
Ice-cream	Ice-cream is a frozen aerated emulsion made from cream and/or milk products, and other ingredients. The ice–cream mix is pasteurised, homogenised, aged and whipped to incorporate air while being frozen. The heat treatment applied to ice cream mix destroys pathogenic microorganisms. However, pathogens may be introduced with the addition of ingredients. Pathogens will not grow in ice-cream, but may survive freezing.
Cultured and fermented milk products	Cultured and fermented milk products are prepared by fermentation of milk using specific microorganisms which reduce the pH and coagulate milk proteins. In the production of yoghurt, milk is homogenised and heat treated. After cooling a starter culture is added and allowed to ferment for several hours. Flavours and other ingredients can be added before the product is packaged. The heat treatment of milk is sufficient to destroy vegetative microorganisms and rapid growth of starter cultures inhibits the outgrowth of spore-formers. Pathogenic microorganisms are prevented from growth by the low pH; the presence of lactic acid, and by refrigerated storage.
Dairy desserts	Dairy desserts can be based on fresh milk, milk powder or milk protein concentrates to which flavours, colours and sweeteners may be added. Dairy desserts mixes typically undergo a heat treatment; and further processed (e.g. whipping and freezing). Heat treatment by pasteurisation or UHT results in the destruction of vegetative cells. Contamination may occur after heat treatment with the addition of further ingredients, or through survival of spores of *B. cereus*.
Dairy-based dips	Dairy-based dips range from processed cheese-type products to sour cream-based dips to which herbs/spices, dehydrated vegetables and flavouring agents are added. Where pasteurisation or other heat treatments are employed, vegetative cells will be destroyed. However, spore-formers can survive heat treatments and other hazards can be introduced with the addition of heat labile ingredients after heating. The low pH of these products assists in their microbial stability
Casein, whey and other functional milk derivatives	These products are derived from milk by concentrating components from whey, skim milk, etc. Normally, heat treatment is followed by steps such as ultrafiltration, acid precipitation or proteolysis. These products are derived from milk and cream that have received at least a pasteurisation heat treatment and so will be free from vegetative cells. Most of these products are dried, thus the low water activity ensures that outgrowth of pathogens is very unlikely.

Dairy product	Processing and impact on microbiological safety
Colostrum	Colostrum is the initial mammary secretion after the birth of the calf. Colostrum obtained by milking is pasteurised, concentrated by evaporation, and either spray-dried or freeze-dried. Pathogens may be protected by the elevated fat and total milk solids content compared to standard bovine milk. Contamination after processing is a concern, although the low water activity of colostrum powder will prevent growth and vegetative cells will eventually die off.

Given the efficacy of the pasteurisation process, post-pasteurisation contamination remains a major concern for the safety of dairy products. Rigorous controls over hygiene, cleaning and sanitation, and product handling are necessary to ensure the final product is not contaminated with pathogenic microorganisms and opportunities for growth are limited. Contamination may result from the environment, including equipment, personnel or contamination of finished product with raw materials.

Of particular concern is *L. monocytogenes,* particularly in moist and chilled products. Its psychrotrophic nature enables the organism to colonise and grow in wet and cold environments including condensation on walls and ceilings, equipment surfaces, drains, floor puddles, condensate collected in refrigeration units and condensation in compressed air lines. Likewise, *Salmonella* has presented problems particularly with dried milk products. Dust and powder residues from ledges, filterhoods, wall ceilings, floors and ancillary equipment are common sources of contamination. Powder, dust and water supply the nutrients and the warmth in some processing environments provides ideal growth conditions for *Salmonella*.

5.1.1 Cleaning and sanitation of processing equipment

The safety and quality of dairy products also depends on proper cleaning and disinfection of processing equipment. The soil encountered in dairy processing plants consists mainly of adhering products and product particles such as milkfat, protein and milk minerals. Residues left on equipment surfaces after inadequate cleaning provide nutrients for microbial growth and will permit build-up and adherence of bacteria films on equipment surfaces.

Microorganisms commonly found on food contact surfaces include enterobacteria, lactic acid bacteria, micrococci, streptococci, pseudomonas, and bacilli (Wirtanen et al, 200?). Inadequate cleaning may result in large numbers of lactococci, coliforms, and other Gram-negative organisms such as *Pseudomonas*, *Alcaligenes, Flavobacterium* and *Chromobacterium* (ICMSF, 1998). These organisms are heat sensitive and are readily destroyed by chlorine disinfectants (ICMSF, 1998).

Inadequate cleaning allows microorganisms to adhere and grow on equipment surfaces and form protective extracellular matrices – biofilms. Once formed, elimination of biofilm is very difficult. Equipment design and choice of surface materials are crucial to combating biofilm formation, as is the cleaning regime. Dead ends, corners, cracks, crevices, gaskets, valves and joints are vulnerable points for biofilm accumulation. Biofilms in the dairy industry are characterised by the predominance of a single species of bacteria e.g. *Streptococcus* thermophilus or *Bacillus* spp. (Flint et al, 1997).

Effective cleaning, disinfection and post-rinsing are all important in eliminating microorganisms. Automated cleaning (or CIP – cleaning in place) systems are frequently used in the dairy industry, and can provide high and reproducible standards of cleanliness (ICMSF, 1998). Automated cleaning systems allow rinsing water and detergent solutions to be circuited through tanks, pipes and process lines without being dismantled. The passage of high velocity flow of liquids over equipment surfaces generates a mechanical scouring effect which dislodges dirt deposits.

In addition to automated cleaning systems, it may also be necessary to dismantle and manually clean equipment. Steam, hot water or chemical sanitisers may be used to sanitise the plant and equipment. Water used in cleaning should be of potable quality.

The efficiency of cleaning and sanitation should be subject to regular environmental monitoring and verification. In addition to verifying the effectiveness of cleaning procedures to ensure residual material is removed, regular monitoring of the environment in and around the processing plant can be an effective early warning system for identifying potential sources of contamination of dairy products.

The Australian Dairy Authorities' Standards Committee (ADASC) is responsible for developing and administering legislation and inspection procedures to ensure Australian dairy products are hygienically manufactured and do not present a risk to public health. ADASC has worked with dairy companies to develop manuals to assist the dairy industry to control *Listeria* spp and *Salmonella* spp in the dairy processing environment. State Dairy Authorities have also developed Codes of Practice for Dairy Food Safety. Standards Australia also has standards which set out accepted practices for cleaning and sanitising dairy factory equipment (AS1162 – 1991, *Cleaning and Sanitising Dairy Factory Equipment*).

5.2 Pasteurisation

The *Australia New Zealand Food Standards Code* (the Code) requires that milk and liquid milk products must be pasteurised (or undergo an equivalent heat treatment)[20]. In the case of cheese manufacture, the Code specifies pasteurisation or thermisation (in combination with a minimum storage time). Standard 1.6.2 of the Food Standards Code specifies that, for the pasteurisation of milk in Australia, the minimum heat treatment is no less than 72ºC for no less than 15 seconds, or any other time and temperature combination of equal or greater lethal effect. These processing measures have been in place historically as an important public health measure to manage the microbiological hazards that may be present in raw milk. There is currently no mechanism in the Code, by which non-thermal processes (*e.g.* ultra high pressure treatments) may be considered as valid, alternative processes to pasteurisation.

This means raw milk and raw milk products are not permitted to be sold in Australia, unless expressly permitted by a State or Territory or if a specific exemption has been given as a result of an assessment process. The sale of raw goat milk is permitted in South Australia, Queensland, New South Wales and Western Australia. In addition, some specific raw milk cheeses are permitted in the Food Standards Code where an assessment has shown that they can be produced to an equivalent level of safety as cheeses made from heat-treated milk.

[20] Milk and liquid milk products includes those used in the production of cream and cream products, fermented milks, yoghurt, dried, condensed and evaporated milks, butter and icecream.

5.2.1 History of pasteurisation[21]

In 1911, the National Milk Standards Committee in the United States was the first professional body to recommend a minimum time-temperature combination for the pasteurisation of milk: 62.8ºC (145ºF) for 30 minutes (now known as the batch or holder method). This heat treatment was slightly above what many people at the time considered to be adequate for the destruction of *M. tuberculosis*, one of the main milk-borne pathogens of concern in that era.

However, it was not until further research and investigation of commercial equipment that the 'holding method' of milk pasteurisation was officially and legally recognised as an adequate method of pasteurisation in the United States where, in 1924, the first Pasteurised Milk Ordinance was published. In the Ordinance, pasteurisation was defined as '*a heating process of not less than 142ºF (61.1ºC) for 30 minutes in approved equipment*'. However, it is noteworthy that a temperature 3ºF lower than that which had been recommend earlier, in 1911, was officially adopted.

Following further studies on the thermal destruction of *M. tuberculosis* and other pathogens, a High Temperature Short Time (HTST) pasteurisation standard - 161ºF (71.7ºC) for 15 seconds - was included in the 1933 edition of the U.S. Public Health Service Milk Ordinance and Code.

In the late 1930s, it became apparent that *Coxiella burnetii*, the causal agent of Q Fever, was more heat resistant than *M. tuberculosis*/*M. bovis*. Studies reported in 1956 showed that if *C. burnetii* cells were present in raw milk in large numbers, some might survive 143ºF (61.7ºC) for 30 minutes. These studies resulted in a recommendation by the US Public Health Service to increase the standard for the 'holding method' of pasteurisation to 145ºF (62.8ºC) for 30 minutes. It was also suggested that at least an additional 5ºF (2.8ºC) be added to the holding temperature for products with a fat content higher than whole milk or with added sugar.

Apart from some rounding of numbers to take account of Fahrenheit-Celsius conversions, the above standards for pasteurisation have remained unchanged to the present day. According to the International Dairy Federation, the minimum time-temperature combinations now recognised world-wide are 63ºC for 30 minutes or 72ºC for 15 seconds.

5.3 Impact of pasteurisation on pathogens in raw milk

The impact of pasteurisation on pathogens in raw milk has been discussed widely in recent years. While there is extensive literature on the subject, there has been no definitive study of the impact of pasteurisation on raw milk in Australia. In February 2005, FSANZ commissioned a study titled 'Scientific Evaluation of Pasteurisation and Alternative Processes for Pathogen Reduction in Milk and Milk Products'. In addition, a separate food safety quantitative risk assessment model on the pasteurisation efficacy of the Australian Dairy Industry was developed in collaboration between the University of Tasmania and the Dairy Research and Development Corporation (now Dairy Australia) (Section 10.5).

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[21] Text in this section is from the report to FSANZ **Scientific Evaluation of Pasteurisation and Alternative Processes for Pathogen Reduction in Milk and Milk Products (**Juffs, H and Deeth, H, 2005)

The objectives of the 'Scientific Evaluation of Pasteurisation and Alternative Processes for Pathogen Reduction in Milk and Milk Products' study were to:

- Define the effect of pasteurisation on levels of pathogenic microorganisms in milk;
- Determine how current industry pasteurisation practices compare with regulatory requirements; and
- Identify possible alternative methods and processes for the destruction of pathogenic microorganisms in milk and milk products, including:
  - the current state of knowledge on their effects on microorganisms; and
  - methods for validating their effectiveness, as a basis for any future equivalence comparisons with pasteurisation.

The study involved:

- a desk-top review of the available scientific literature and epidemiological data, from Australian and overseas sources, on the effect of milk pasteurisation and thermisation on the levels of pathogenic microorganisms in milk intended either for human consumption as a liquid milk product or for further processing into other dairy products; and
- a survey of the commercial dairy industry in Australia, with the objective of determining current industry practices for the pasteurisation of milk including the methods employed and time/temperature combinations and their relationship to minimum regulatory requirements.

5.3.1 Methods for determining heat resistance of pathogens and interpretation of the data

Many different techniques and types of equipment have been used to measure heat resistance of milk-borne pathogens, ranging from the very simple to the very sophisticated and from micro- to commercial-scale. However, there is ample evidence to indicate that the method used to determine heat resistance is a major factor in determining:

- the reliability of the heat resistance data generated; and
- its relevance to commercial pasteurisation practice.

Hence, methodology should always be considered when assessing the veracity of any conclusions about the ability of an organism to survive/not survive commercial heat treatments.

From a commercial perspective, it is the overall impact of the integrated heating profile, plus any other relevant system inputs, on the survival/destruction of any pathogens that may be present in the raw milk. Other system inputs during commercial processing include turbulent flow and, in some cases, homogenization. Thus, greatest weight should be given to the results of heat resistance studies carried out using actual HTST pasteurisation equipment, either pilot plant or commercial-scale. Such equipment should, however, comply with recognised design and operational standards.

5.4 Ability of bacterial pathogens to survive pasteurisation[22]

Heat resistance studies conducted using either pilot plant- and/or or commercial-scale HTST pasteurisation equipment, together with additional data from studies using various laboratory techniques, have confirmed that the vegetative forms of 11 of 18 pathogenic species considered in this review are destroyed by both batch (63ºC for 30 minutes) and HTST (72ºC for 15 seconds) pasteurisation, with a reasonable margin of safety. These species are:

- *Brucella abortus*
- *Campylobacter jejuni*
- *Campylobacter coli*
- *Coxiella burnetii*
- Pathogenic *Escherichia coli* (0157:H7)
- *Listeria monocytogenes*
- *Mycobacterium tuberculosis*
- *Mycobacterium bovis*
- *Salmonella enterica serotypes*
- *Streptococcus pyogenes*
- *Yersinia enterocolitica*

The effect of pasteurisation on selected organisms are presented in Table 15.

Table 15: Effect of pasteurisation on selected microorganisms [23]

Mycobacterium avium subsp. paratuberculosis (MAP)	The heat resistance of this organism has been subject to extensive study during the past decade using various laboratory techniques and pilot scale HTST equipment. Obtaining definitive heat resistance data for this organism has proved to be difficult. While there appears to be ample evidence that this organism is destroyed by batch pasteurisation, studies on the ability of MAP to survive heating at 72ºC for 15 seconds, even with pilot scale HTST equipment, have given conflicting results. However, more recent, well-controlled studies have shown that a minimum 4-$\log_{10}$ reduction is obtained during HTST pasteurisation. In view of the numbers MAP likely to be present in raw milk, this level of kill in fact provides a reasonable margin of safety for the consumer. However, population reductions in the order of 6-7- $\log_{10}$ have been reported. The fact that it is necessary for operational reasons to operate HTST equipment at temperatures slightly higher than 72ºC - apart from any decision to use higher temperatures for other reasons - provides an additional margin of safety. A fundamental unanswered question with respect to MAP, is whether it is a human pathogen, or whether its postulated association with Crohn's disease is just serendipitous, rather than causal. If studies eventually establish that there is no causal connection between MAP and Crohn's disease, any concerns that this organism might be able to survive HTST pasteurisation will prove to be unfounded.
Bacillus cereus	Although there is limited data available specifically on the heat resistance of the vegetative form of this organism, and none using commercial HTST equipment, it is generally accepted that the vegetative cells are readily destroyed by both batch and HTST pasteurisation. However, there is more than ample evidence to indicate that the spores of *B. cereus* are very heat resistant and readily survive any heat treatments in the normal pasteurisation range. The pasteurisation heat treatment is sufficient to heat activate the fast-germinating spores of *B. cereus*, but not the slow-germinating spores. Similarly, pasteurisation inactivates diarrhoeagenic toxins produced by *B. cereus*, but not the emetic toxin.
Brucella melitensis	No definitive data on the heat resistance of the organism (which is not present in Australia) were located. However general statements from authoritative sources indicate that the organism is destroyed by pasteurisation.
Enterobacter sakazakii	Although the data is somewhat variable, and data using commercial HTST equipment is lacking, the consensus view is that the heat resistance of this organism falls within the safety margins of commercial pasteurisation. Its presence in pasteurised milk products has been found to be due to re-contamination of the pasteurised product after the pasteurisation step.

[22] Text in this section is from the report to FSANZ **Scientific Evaluation of Pasteurisation and Alternative Processes for Pathogen Reduction in Milk and Milk Products (**Juffs, H and Deeth, H, 2005)

[23] Text in this section is from the report to FSANZ **Scientific Evaluation of Pasteurisation and Alternative Processes for Pathogen Reduction in Milk and Milk Products (**Juffs, H and Deeth, H, 2005)

Staphylococcus aureus	Although this organism has relatively high heat resistance for a mesophilic non-sporing bacterium, and despite the fact that data using commercial HTST equipment is lacking, there is ample evidence from laboratory studies that it is destroyed by both batch and HTST pasteurisation heat treatments with a wide margin of safety. However, the thermal stability of the enterotoxins produced by *S. aureus* greatly exceeds that of its vegetative cells, and they readily survive pasteurisation by a wide margin.
Streptococcus agalactiae	Only one report on the heat resistance of *S. agalactiae* was located. This indicated - under relatively crude experimental conditions - that the organism was inactivated at unspecified population levels in cream by batch pasteurisation.
Streptococcus zooepidemicus	Not a single report on the heat resistance of *S. zooepidemicus* was located. However, human infection with this organism can usually be traced to an animal source, including ingestion of unpasteurised milk and cheese.

5.4.1 Data gaps on heat resistance of pathogens

The main gaps in data and knowledge in respect of pasteurisation identified were:

- definitive evidence on whether or not MAP can be classified as a human pathogen; and
- quantitative heat resistance data for *Brucella melitensis, Streptococcus agalactiae* and *Streptococcus zooepidemicus* in milk.

In addition, it must be noted that:

- heat resistance data obtained using commercial HTST pasteurisation equipment appears to be lacking for the vegetative cells of several of the pathogenic species covered in this review*, e.g. Bacillus cereus, Enterobacter sakazakii* and *Staphylococcus aureus*; and
- the most recent heat resistance data obtained by any method that is available for some of the pathogens is quite dated, *e.g. M. tuberculosis* (1927), *Coxiella burnetii* (1956 and 1961), *M. bovis* (1960), and *St. agalactiae* (1974).

Standardised protocols and methodologies for the determination of heat resistance appear to be lacking. Studies using methodologies known to give unreliable results, *e.g.* open tubes, are still being reported in the literature.

5.5 Quantitative modelling of the effect of pasteurisation on pathogens

The food safety risk assessment model on the pasteurisation efficacy of the Australian Dairy Industry[24] developed in collaboration between the University of Tasmania and Dairy Australia model is a stochastic simulation model and is designed and built in Microsoft Excel with Palisade's @Risk as the simulation engine. The model includes variables on the farm that are used to describe conditions during storage on farm, transport to the processing plant, and following pasteurisation.

The pathogens modelled were selected by a group of dairy industry technical managers and included: *E. coli; Salmonella; L. monocytogenes; Campylobacter* spp.*; Yersinia enterocolitica; B. cereus; and S. aureus.* These pathogens were identified as representing the priority food safety hazards relevant to the Australian dairy industry, recognising that not all hazards could be modelled given the resources available to the project.

[24] Text in this section is from the food safety risk assessment model on the pasteurisation efficacy of the Australian Dairy Industry developed in collaboration between the University of Tasmania and Dairy Australia (Ross *et al*., 2005)

The model uses a variety of biological and physical parameters as inputs, including:

- State (factors such as herd size, production per cow and seasonality effects);
- herd size;
- probability that the herd is contaminated (whether with a pathogen or with a residue);
- for contaminated herds, the number of animals within the herd that is contaminated;
- for each 'contaminated' cow, amount of hazard transferred into raw milk;
- the volume of milk produced per cow;
- time and temperature in the farm milk vat (used to predict growth of pathogens);
- dilution upon mixing into tankers;
- time and temperature of the tanker (used to predict growth of pathogens);
- processing factory silo size (used to estimate effect on hazard concentrations); and
- time and temperature of pasteurisation (used to model thermal inactivation).

The model also includes several steps involving time and temperature combinations to model the progress of pasteurised milk from the factory into storage and distribution, through the retail chain and, finally, to consumer transport, storage and eventual consumption.

The concentration of bacteria remaining in milk after pasteurisation is a function of the initial concentration in raw milk and the combination of the time and temperature conditions of pasteurisation. Under pasteurisation conditions employed in the Australian dairy industry the results for each pathogen are listed in Table 16.

Table 16: Modelled effect of pasteurisation on pathogens (Ross *et al*., 2005)

Pathogen	Effect of pasteurisation
E. coli	*E. coli* is predicted to be effectively eliminated during pasteurisation. The maximum estimated concentration of *E. coli* in raw milk, based on >1 million iterations of the model, was 1.8×10^{-3}/ml. The model estimates the minimum effect of pasteurisation is a 13-log reduction in EHEC numbers. A huge contamination on farm would not overwhelm the effect of pasteurisation against this organism. In 95% of cases pasteurisation achieves a 26-log reduction in concentration, a median value being a 112-log reduction.
Salmonella	*Salmonella* are moderately destroyed by pasteurisation. The highest concentration of *Salmonella* estimated in raw milk is 1.6×10^{6}/ml. To achieve these high levels of the organism in raw milk, high on farm contamination and temperature abuse during transport and storage needs to occur. The model estimates that the minimum thermal inactivation of *Salmonella* during pasteurisation will achieve a 5-log reduction. However, in 95% of cases the pasteurisation process will result in a 7-log reduction in numbers and a mean reduction of 9-log. These estimates were based on the most thermotolerant strains, more 'typical' strains would experience reductions greater than that predicted for EHEC.
L. monocytogenes	*L. monocytogenes* is effectively eliminated by pasteurisation. The highest concentration of this organism was estimated by the model to be 3.9×10^{-1}/ml. With the exception of the large-scale contamination of raw milk on farm, the concentration of *L. monocytogenes* in Australia's raw milk is likely to be low. The model estimates that the minimum reduction of this organism during pasteurisation is 7-log. In 95% of cases an 11-log reduction was estimated. The mean reduction is 59-log.
B. cereus	Spores of *B. cereus* are not inactivated by pasteurisation. The model assumes that any *B. cereus* cells that become contaminants of raw milk enter the factory processing stage of the model as spores. The mean concentration of *B. cereus* spores in milk is estimated at 72 spores L^{-1}. The expected log reduction in the concentration of *B. cereus* spores in milk is 0.02-log, effectively no change in concentration. Growth of *B. cereus*, was estimated using a separate stochastic model during simulated distribution, retail storage and home storage conditions. The results included variability in initial *B. cereus* loads, minimum growth temperatures for *B. cereus*, temperature and storage life (due to temperature variation). The results from modelling suggest that 1 in 100,000 litres of milk could contain in excess of 100,000 cells per 100 milk at the time of consumption.

Pathogen	Effect of pasteurisation
C. jejuni	*C. jejuni* has relatively low thermal tolerance and is effectively eliminated by pasteurisation processes used by the Australian dairy industry. The highest level estimated to be present in raw milk entering the factory is 6.1×10^{-1}/L. Even with high contamination rates and high numbers entering milk at the farm, the effect of pasteurisation will negate risk from this organism. The model estimates that the minimum reduction in numbers of *Campylobacter* spp during the pasteurisation of milk is 61-log. In 95% of cases pasteurisation achieves approximately a 113- log reduction, and the mean reduction is 2000-log.
Y. enterocolitica	*Y. enterocolitica* has a relatively low thermal tolerance and is effectively eliminated by pasteurisation. The highest estimated level of *Y. enterocolitica* in raw milk entering the factory is 7×10^{7}/L. Minimum reduction during pasteurisation is 41-logs. In 95% of cases a 72-log reduction will result from pasteurisation - mean estimate is a 1500-log reduction.
S. aureus	The predicted reduction in *S. aureus* levels is less than that predicted for Gram negative, foodborne, pathogens but a large reduction in hazard levels is still predicted to occur. The organism is a common contaminant of raw milk, with some reports as high as 100% of raw milk samples being positive for the bacterium. The model predicts that the maximum concentration that *S. aureus* will reach just prior to pasteurisation is 7.7×10^{4}/L, considerably less than the levels required to produce toxin levels that would lead to emetic reactions. The minimum predicted effect of pasteurisation on *S. aureus* is a reduction of 7-log. In 95% of cases a 10-log reduction can be expected.

5.6 Overall summary of effectiveness of pasteurisation

The study 'Scientific Evaluation of Pasteurisation and Alternative Processes for Pathogen Reduction in Milk and Milk Products' concluded that consumers of pasteurised milk and dairy products in Australia can be assured that pasteurisation continues to be a very effective public health measure. Three complementary observations allow this conclusion to be drawn:

i) Ample heat resistance data to indicate that the vegetative cells of the most significant milk-borne pathogens are destroyed by pasteurisation, with a reasonable margin of safety [though it is recognised that there are still some gaps in the data for some organisms and that there are other forms (*e.g.* spores) or products (*e.g.* toxins) of some species that can withstand pasteurisation];
ii) With a small number of exceptions (which are related more to process control issues or the interpretation of what constitutes an equivalent treatment, rather than significant deficiencies in the actual times and temperatures used), pasteurisation of milk and cream in Australia meets the minimum time and temperature standards prescribed in the Food Standards Code, or recognised equivalents; in many cases, the product is heated to a temperature and/or a time often in excess of the prescribed minimums; and
iii) Lack of evidence in epidemiological data indicating that pasteurised milk products have been implicated in any outbreaks of foodborne gastrointestinal illness in Australia in recent years whereas, in contrast, such outbreaks continue to be associated with consumption of raw milk, both in Australia and in other countries.

The modelling undertaken by University of Tasmania concluded that the likelihood of survival of pasteurisation by vegetative pathogens is very remote. The modelling estimates range from 1 *Salmonella* survivor among the entire Australian liquid milk production every 2.5 years to 1 *Campylobacter* spp. survivor in approximately 10^{2000} years. With regard to the *Salmonella* data, the estimate is based on the most heat resistant strains known. Data for enterohaemorrhagic *Escherichia coli*, which may be more representative, lead to estimates of 1 survivor in 10^{100} years of Australian production at current levels. A summary of the effect of pasteurisation on bacterial contaminants in milk is presented in Table 17.

Table 17: Summary of the effect of pasteurisation on bacterial contaminants in milk (Ross *et al*., 2005)

Organism	Pasteurisation effect (Log reduction)			Time to encounter a single cell in Australia's milk production (years)
	Min.	95th percentile	Median	Mean
E. coli	13	26	112	10^{100}
Salmonella	5	7	9	2.5
L. monocytogenes	7	11	59	10^{45}
B. cereus	0.02	0.04	0.10	<1
C. jejuni	61	113	2000	10^{2000}
Y. enterocolitica	41	72	1500	10^{1500}
S. aureus	7	10	25	10^{13}

Such estimates highlight the huge margin of safety afforded by pasteurisation of liquid milk products when such equipment is operated reliably. This quantitative risk assessment indicated that the vast majority of liquid milk processors have sufficient controls in place to prevent milk that has not received adequate pasteurisation from reaching the market. Conversely, the results indicate that Australian pasteurisation processes have virtually no effect on spores of *B. cereus*, some strains of which are psychrotrophic. *B. cereus* is expected to be commonly found in raw milk at low levels (~100 spores/litre).

While there are no data to indicate harm to public health, simulations of the potential for germination and outgrowth of cells of *B. cereus* in pasteurised milk during normal distribution and storage suggest that levels of 100,000 cells per 100ml of liquid milk could occur in 1 in 100,000 servings.

While these studies concluded that pasteurisation of raw milk destroys the vegetative cells of the most significant milk-borne pathogens, it should be noted that dairy products containing elevated levels of fat or solids such as ice-cream mixes, cream and yoghurt warrant higher time/temperature combinations than those currently specified in the Food Standards Code to compensate for the protective effect of fat and solids on microorganisms (Appendix 1).

5.7 Times and temperatures used for the pasteurisation of milk in Australia[25]

From the Australia-wide industry survey conducted during this study and additional data from a survey of the Victorian dairy industry by Dairy Food Safety Victoria in 2004, it is clear that batch pasteurisation is used in Australia by small-scale processors.

However the batch method would account for only a very small percentage of all milk pasteurised in Australia. Temperatures and times of heat treatment for batch pasteurisation covered a range, from 62-90ºC and from 15 seconds to 30 minutes. The type of product being manufactured was a major influence on the temperature-time combination used.

Several processors reported using what is essentially a HTST treatment, *e.g.* 72ºC for 15 seconds or similar, under batch conditions.

[25] Text in this section is from the report to FSANZ **Scientific Evaluation of Pasteurisation and Alternative Processes for Pathogen Reduction in Milk and Milk Products (**Juffs, H and Deeth, H, 2005)

All of the temperatures and times for pasteurisation of milk by the HTST method reported during the industry surveys showed that the minimum heat treatment for HTST pasteurisation as specified in the Code, *i.e.* 72ºC for 15 seconds, was being achieved by all respondents. Beyond that, however, temperatures ranged from 72-86ºC and times from 15-50 seconds, with many different combinations within those ranges. As with batch pasteurisation, type of product was again a major influence on the heating regime used, with time and temperatures reported generally being within the expected range for the type of product.

Of particular note is that HTST treatment of milk for liquid milk products, at least by most of the large processors and some of the smaller ones, was mostly in the range 74-78ºC for 15-30 seconds. This reflects a recommendation by the peak Australian dairy industry organisation in 2000 that the times and temperatures for HTST pasteurisation of milk for the liquid milk trade be increased as a precaution against the presence in the raw milk of any MAP organisms that might be resistant to the minimum pasteurisation treatment of 72ºC for 15 seconds. Whether use of this enhanced heat treatment is still warranted in the light of more recent studies on the heat resistance of this organism that have been conducted using commercial HTST equipment, particularly in areas of Australia where Johne's disease in cattle is reported to be not endemic, is a matter for conjecture.

Only one processor reported that they were using the 62ºC for 15 seconds heat treatment (thermisation) option for cheese milk permitted in the Food Standards Code.

Some processors, particularly those in the small and medium size categories, reported that design of their pasteurisers and operational considerations largely dictated the limits on the times and temperatures of heating that they could use in practice.

5.8 Alternative technologies to pasteurisation of milk and milk products[26]

Several alternatives to the traditional thermal processes for the pasteurisation of milk have been under investigation by various research groups around the world. A major driver of this research has been the demand by consumers for 'natural' foods which, they believe, have the colour, flavour and nutritive value of the raw material. Many of the alternative technologies investigated so far have the potential to achieve this aim. To date, however, no single alternative technology has been shown to be capable of replacing heat - applied via the traditional thermal pasteurisation processes - as an effective and reliable means of destroying all of the pathogenic vegetative bacteria that can be found in raw milk.

Overall, the following observations can be made about various alternative technologies:

- There is a lack of information on the effect of most of the alternative technologies on many of the pathogens considered in this review.
- The most researched technologies are high pressure and pulsed electric field. A considerable amount of data is also available for hydrogen peroxide treatment.
- For each technology and each pathogen, a range of bactericidal effects have been reported. While high log reductions and 'complete inactivation' have been reported in many cases, the reported log reductions usually cover a wide range. This may be due to different experimental conditions but the possibility of different resistances amongst

[26] Text in this section is from the report to FSANZ **Scientific Evaluation of Pasteurisation and Alternative Processes for Pathogen Reduction in Milk and Milk Products (**Juffs, H and Deeth, H, 2005)

the strains of bacteria cannot be ignored.

- A wide range of treatment conditions can be used for each technology and therefore it is difficult to compare data from different reports.

In summary, no one alternative technology can be used with confidence at this time as an alternative to thermal pasteurisation. Considerable research still needs to be performed on the technologies with the pathogenic bacteria of interest before properly informed risk assessments can be performed. A summary of the applicability of the major alternative technologies to the dairy industry are outlined in Table 18.

From an Australia-wide industry survey conducted during this study, it was established that, on average, 22% of respondents had 'some knowledge' of the various alternative processes that have potential application for the destruction of pathogenic organisms in milk. There is industry interest in the application of alternative technologies, for a range of reasons. Some interest is purely economic (*e.g.* reduced costs), some is technological (*e.g.* making a better cheese) and some is philosophical (*e.g.* keeping milk in its natural state). Conversely, some respondents also had concerns about the alternative technologies, *e.g.* technical feasibility, effects on manufacturing process and product quality, capital and operating costs, food safety and operator safety.

Table 18: Applicability of major alternative technologies to the dairy industry

Technology	Overall conclusion
High pressure and pulsed electric field	Capable, under certain conditions, of inactivating pathogenic microorganisms that may occur in milk and milk products. However, neither is effective against bacterial spores, although very high pressure at elevated temperature has been shown to be sporicidal. To date, neither technology has been used commercially in the dairy industry. This is partly because the processes have not been adequately validated for food safety and partly because the technologies have not been scaled up to commercial capacities. Both technologies can be expected to be applied in selected areas of the dairy industry in the medium term, provided regulatory issues can be resolved.
Hydrogen peroxide	Capable of reducing the load of bacteria, including pathogens in liquid foods. It is effective against a range of organisms but may not completely destroy some pathogenic organisms.
Microfiltration	Already being used in the processing of market milk and milk for cheese making in some countries. The extent of removal of bacteria is usually only of the order of 3 logs, although there have been reports of higher reductions. The risk of bacteria entering the final product through faulty membranes or equipment without detection is a constant concern. At present, microfiltration is used in conjunction with normal pasteurisation. A major drawback of microfiltration is that whole milk cannot be treated.
Bactofugation	Used commercially, although largely restricted to cheese milk for removal of *Clostridium tyrobutyricum* spores. The maximum level of removal is ~2 logs so this technology cannot be used alone for 'pasteurising' milk.
Ultrasonication	Shows promise but insufficient research on its effect on pathogens has been reported to enable a proper assessment for treating milk at this time.
Irradiation	Effective against most if not all pathogens. However, public attitude together with the risk of off-flavour production will prevent its use for milk and milk products in the foreseeable future.

6. Distribution, retail display, and the consumer-end of the dairy supply chain

After manufacture, dairy products remain vulnerable to contamination (particularly unpackaged products) and susceptible to temperature abuse at all stages up until consumption. As many dairy products do not undergo a further pathogen reduction step prior to consumption (*e.g.* cooking), avoidance of contamination and attention to storage time and temperature are of particular importance in minimising the potential exposure to pathogens.

6.1 Post-processing contamination

Cross-contamination is potentially the most important means by which dairy products are contaminated after processing. The potential for microbiological hazards to be introduced during transport and distribution; retail; food service; and the consumer-end of the supply chain may occur through environmental contamination and via cross contamination with other products.

Microbiological hazards can be introduced into dairy products through environmental contamination including soil and dust, air, birds, rodents and insects. Most dairy products however, are packaged for distribution, thus the integrity of the packaging must be maintained to prevent environmental contamination.

Cross-contamination of dairy products with microbiological hazards can occur through inadequate food handling practices at retail and in the home. Unpackaged cheeses in delicatessens are particularly vulnerable to cross contamination with other foods, food utensils, and from display cabinet surfaces. For example unpackaged cheeses may become contaminated with *L. monocytogenes* from surrounding foods on display, or through contaminated utensils, etc. *L. monocytogenes* is a concern for dairy products in particular as most dairy products require refrigeration, and growth of this organism can occur at refrigeration temperatures.

6.2 Storage time and temperature

Storage time and temperature during retail display, food service and/or consumer household, including transportation, will impact on the number of microorganisms present in dairy products. Improper storage of dairy products may allow growth of pathogenic microorganisms to levels likely to cause illness. Spores, which have survived processing, may grow if storage temperature and times are not controlled. Furthermore, low levels of pathogens, which may have been introduced through environmental contamination during processing, may also grow if storage temperatures and time are not controlled (*e.g. Salmonella, Listeria* and bacteria such as *E. sakazakii*). Correct storage (refrigeration) of dairy products throughout the transportation and retail supply chain and through to the consumer is important to maintain safety, shelf-life, and quality.

Published and unpublished data obtained from surveys in Australia and overseas consistently show the refrigerated retail cabinet as a weak link in the cold chain. Data on retail storage temperatures during a Meat and Livestock Australia study (Figure 2) show that while the majority of temperatures recorded were below 5°C, some temperatures recorded were as high as 15°C (Meat & Livestock Australia, personnel communication).

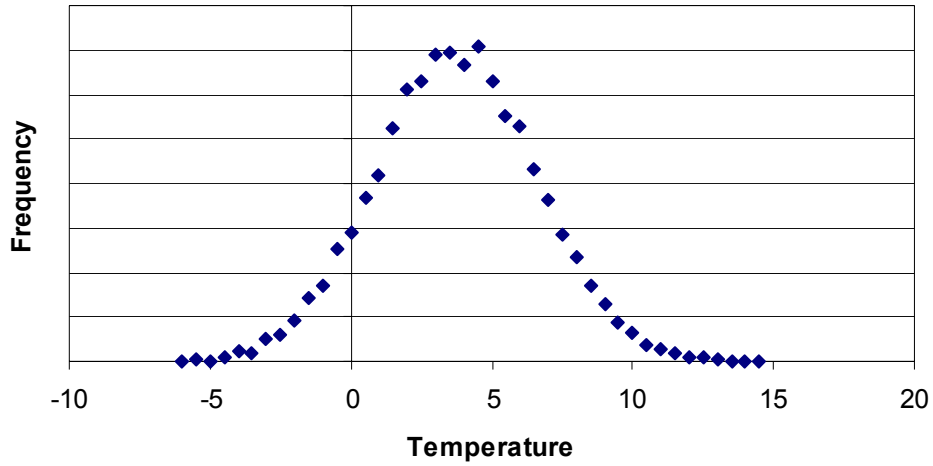


Figure 2: Frequency distribution for retail temperatures in Australia

There is a lack of available data for storage conditions in the food service sector, however temperature control of food stored in domestic refrigerators in Australia is generally poor. In a 1998 survey, 36% of Australian domestic refrigerators (n=171) had their fresh-food compartments above 5°C for greater than 50% of the time (Jay et al 1998). Data on temperatures in domestic refrigerators during the Meat and Livestock Australia study (Figure 3) confirm this finding (Meat & Livestock Australia, unpublished data).

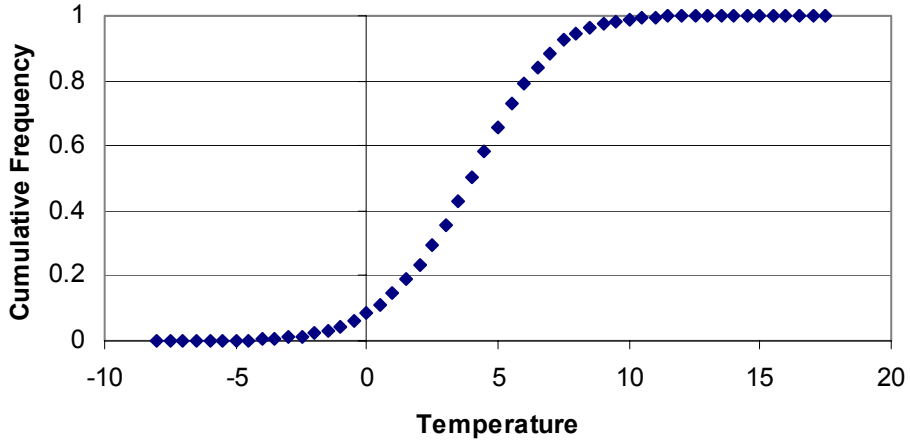


Figure 3: Cumulative frequency distribution for domestic refrigerator temperatures in Australia

As the growth and levels of microorganisms in dairy products are influenced by temperatures during storage, the better the cold chain is maintained the less growth will occur. This is particularly important for *L. monocytogenes* as it can grow at refrigeration temperatures.

6.3 Food handling practices

Poor hygiene and inadequate food handling practices can also lead to contamination of dairy products post manufacture, especially in unpackaged products.

Infected food handlers can also be a source of contamination. The microflora on the hands and outer garments of food handlers generally reflects the environment and habits of the individuals. This flora would normally consist of organisms found on any object handled by the individual as well as those picked up from dust, water, soil etc. Some pathogens are specifically associated with the hands, nasal cavities and mouth of personnel. For example, micrococci and staphylococci from skin (particularly cuts and wounds) and upper respiratory tissues may contaminant dairy products during handling.

Other pathogens that may be transferred to dairy products include intestinal pathogens such as *Salmonella* and *Shigella* which can be deposited onto equipment and surfaces if good sanitary practices are not followed either at retail, during food service operations, or in the home. Viruses such as hepatitis A and nororviruses may also be transmitted to dairy products through infected food handlers. Hepatitis A in particular is excreted in high numbers in the faeces and is spread from person to person by the faecal oral route. In addition, asymptomatic persons may transfer the virus to food during the incubation period for the disease. Viral shedding may begin several days before onset of symptoms and continue after symptoms have ceased. Hepatitis A is also able to survive on environmental surfaces.

Product stored in the home, once opened is also vulnerable to contamination and temperature abuse. Most dairy products have a relatively short shelf-life, especially milk (10-16 days under optimum storage conditions) thus storing dairy products according to manufacturer instructions and following good hygiene and handling practices in the home is also important.

Pathogens such as *B. cereus*, *Salmonella* and *E. sakazakii* can grow in reconstituted milk powders and infant formulae if stored above 5°C for a sufficient time and multiply very readily at room temperatures. Good hygiene practices in the home during reconstitution, storage and feeding are essential to avoid recontamination and/or multiplication of pathogens in these products. The National Health and Medical Research Council guidelines recommend that infant formula be reconstituted with cooled boiled water then stored in the refrigerator for a maximum of 24 hours (NHMRC, 2003). Studies both in Australia and overseas have indicated that some consumers prepare infant formula incorrectly with warm tap water, leave bottles at room temperature for more than 2 hours and store prepared warm infant formula in insulated carriers when travelling (Lilburne *et al*., 1988; Beck-Fein and Falci, 1999).

7. Discussion and summary

The purpose of the microbiological risk profile was to bring together scientific and technical information on microbiological hazards that may be associated with dairy products in order to identify the public health and safety risks associated with microbiological hazards in dairy products. The profile identifies and examines hazards along the dairy supply chain from milk production through to consumption of dairy.

Raw milk has a mixed microflora, which is derived from several sources including the interior of the udder, exterior surfaces of the animals, environment, milk-handling equipment, and personnel. In general, there are two means by which pathogens contaminate raw milk. Contamination may occur when microorganisms are shed directly into raw milk from the udder as a result of illness or disease, or through contamination from the external surface of the cow and the milking environment. Primary production factors that impact on these routes of contamination and the microbiological quality of the raw milk include:

- animal-related factors e.g. animal health, herd size, age and production status;
- environment-related factors e.g. housing, faeces, feed, soil, and water; or
- milking and operation of milking equipment factors.

There is relatively little data on the presence or absence of pathogens in raw milk in Australia although it is well established that raw milk can be contaminated with pathogenic microorganisms, including *Salmonellae, S. aureus, L. monocytogenes. E. coli* (0157:H7), *Campylobacter* spp. and *Yersinia enterocolitica*. Overseas data demonstrate that pathogens are frequently isolated from raw milk. Pathogens were detected in raw milk in 85% of 126 surveys identified in the literature.

The safety of processed dairy products relies on:

- the quality of raw materials;
- correct formulation;
- effective processing;
- the prevention of recontamination of product; and
- maintenance of temperature control during distribution and retail sale of the product.

The most important risk factors affecting raw milk microbiological quality on-farm can be summarised as follows:

Risk factor	Effect	Control
Animal health	Disease in, sickness of, and carriers in milking animals can increase shedding of pathogens directly into raw milk, or in animal faeces.	Animal health and mastitis programs
Herd size	Herd size may have some effect on the prevalence of some pathogens (*e.g. Salmonella, E. coli* and *Campylobacter*)	Biosecurity and animal husbandry
Age/ production	Calves have an increased susceptibility to infection, and have been reported to have higher prevalence rates of some pathogens (*e.g. E. coli*)	Calves kept separate from milking herd
Housing	Intensive housing practices may increase risk of contamination of udders due to close proximity of animals, concentration of faeces, bedding etc. This has been shown to be a factor in the prevalence of *Bacillus spp., E. coli,* and *L. monocytogenes*	Australian dairy farming is mainly pasture based
Faeces	Faeces may contain various pathogens – reflecting either illness/infection, or through ingestion of contaminated feed and/or water with faeces. Faeces may contaminate the exterior of the udder and introduce pathogens into raw milk.	Udder hygiene at milking
Effluent	Effluent (containing manure) can also contaminate pasture.	Appropriate treatment and disposal of effluent
Feed	Contamination of feed can lead to shedding of pathogens into faeces. Poorly made silage can be a source of pathogens (*e.g. E. coli, Bacillus spp., Listeria,* and *Clostridia*).	Control over preparation and storage of feed, especially silage
Water – stock drinking	Water is a potential source of contamination. Sediment in water can support bacterial growth and be a reservoir for pathogens. Water sources can become contaminated with cud and/or faecal material, feed, etc.	Ensuring water is of suitable quality
Milking	Poor milking practices, including dirty teats, inadequate cleaning and maintenance of milking equipment, and poor personnel hygiene can lead to contamination of raw milk.	Maintenance, sanitation and cleaning of equipment, appropriate animal and good personal hygiene
Water use - milking	Water is a potential source of contamination during washing of teats and cleaning of milking equipment.	Ensuring water used is of suitable quality
Storage	Inappropriate temperature control of milk after milking can lead to growth of pathogens	Rapid cooling of milk and regular collection.
Transport	Inappropriate temperature control of milk during transportation can lead to out-growth of pathogens. Contamination can occur if tankers do not adequately protect milk, and/or are inadequately cleaned.	Temperature control, tanker maintenance, and cleaning and sanitation

Pasteurisation represents the principal process for rendering dairy products safe for consumption. Pasteurisation will eliminate most significant milk-borne vegetative microorganisms of concern. This is confirmed by microbiological survey data for pasteurised dairy products in Australia that shows a very low incidence of hazards of public health significance in these products. Pathogens such as *L. monocytogenes*, *Salmonella* and *S. aureus* are rarely isolated.

However, the effectiveness of pasteurisation is dependent upon the microbiological quality of the incoming raw milk. Control of risk factors on-farm will minimise the opportunity for microbiological hazards to contaminate raw milk and reduce the likelihood and concentration of these hazards.

A survey of Australian dairy manufacturers determined that the vast majority met the minimum time and temperature standards prescribed in the Code for the pasteurisation of milk and cream. In many cases, milk was heated to a temperature and/or a time in excess of the prescribed minimums. For the majority of dairy products, pasteurisation also represents

an initial treatment before specific processes are used to transform raw milk into various manufactured products.

Dairy products containing elevated levels of fat or solids such as ice-cream mixes, cream and yoghurt, necessitate higher time/temperature combinations than those currently specified in the Food Standards Code in order to compensate for the protective effect of fat and solids on pathogenic microorganisms.

The effect of pasteurisation on dairy processes on the major microbiological hazards that have been associated with foodborne illness in various dairy commodities can be summarised as follows:

Pathogens	Significance in dairy products
Salmonella	*Salmonella* is destroyed by pasteurisation, however it can be present in the environment and can gain access to product after heat treatment. Initial source is often birds and rodents, although occasionally present in the raw milk. Non-dairy ingredients can be an important source of contamination.
Listeria monocytogenes	*L. monocytogenes* is destroyed by pasteurisation. Its presence in heat-treated products is due to post-pasteurisation contamination. *L. monocytogenes* is a concern to the dairy industry as it can grow down to 0ºC (refrigeration temperatures).
Staphylococcus. aureus	*S. aureus* is destroyed by heat-treatment, however its toxins are heat stable, thus control of growth of this organism prior to heat treatment is essential. However, *S. aureus* does not grow well at low temperatures (i.e. refrigeration).
Bacillus cereus	Vegetative cells of *B. cereus* do not survive pasteurisation, however spores will survive heat treatments. *B. cereus* is rapidly outgrown by gram-negative psychrotrophs at refrigeration temperatures, but in their absence, *B. cereus*, if present may then be able to grow to high levels. This is a concern with extended shelf-life chilled products such as desserts.
Escherichia coli	*E. coli* is found in cattle and may enter milk through faecal contamination, however *E. coli* is heat-sensitive and does not survive pasteurisation.
***Campylobacter* spp.**	*Campylobacter* spp. is destroyed by pasteurisation and its presence in milk products is due to environmental contamination after heat treatment. *Campylobacter* spp. are fragile organisms unable to grow in foods.
Yersinia enterocolitica	*Y. enterocolitica* is destroyed by pasteurisation and its presence in heat-treated milk products is due to environmental contamination after heat treatment. *Y. enterocolitica* is able to grow in dairy products held at refrigeration temperatures and therefore may be considered as a hazard in prolonged shelf-life products.
Enterobacter sakazakii	*E. sakazakii* will not survive pasteurisation. Recontamination of powdered infant formulae during manufacture is a risk. *E. sakazakii* cannot grow in a dry substrate, but it can survive a long period of time and is potential hazard when the powder is reconstituted and held for long periods of time at favourable temperatures. Contamination and subsequent growth may occur during reconstitution and preparation.

Post-pasteurisation contamination is a major risk factor for the safety of dairy products. Contamination may result from the environment, including equipment, personnel or contamination of finished product with raw materials. Rigorous control over hygiene, cleaning and sanitation, and product handling is therefore critical to safety of dairy products.

The major processing factors affecting the safety of specific dairy products are summarised overleaf:

Dairy product	Processing and impact on microbiological safety
Milk and cream	Pasteurisation is sufficient to destroy pathogenic milk-borne vegetative bacteria. Illness resulting from consumption of pasteurised milk is rare. However, where outbreaks have occurred, these were attributed to inadequate pasteurisation, post-pasteurisation contamination and/or temperature abuse.
Cheese	A number of processing factors influence the growth and survival of pathogens in cheese, including the severity and duration of heat treatment (including curd cooking); pH; salt concentration; water activity; and maturation/ripening. A number of outbreaks of foodborne illness have been linked with the consumption of cheese (Appendix 2). These outbreaks have resulted from faulty controls in cheese production; use of contaminated starter cultures or contaminated ingredients; post-pasteurisation contamination; or mishandling during transport and/or distribution.
Dried milk powders	Microorganisms in dried milk powders will not grow due to low water activity, however, they may survive for long periods and resume growth when the powder is reconstituted and stored under favourable conditions. Heat-treatments given prior to spray-drying are severe enough to destroy all vegetative pathogens in raw material. However, there is opportunity for environmental contamination during spray-drying and subsequent storage. Dried milk powders have been implicated in a number of foodborne illness outbreaks (Appendix 2). The outbreaks were caused by preformed staphylococcal enterotoxin; poor plant hygiene; contamination and abuse of reconstituted products; and outgrowth of bacterial spores.
Infant formulae	Microbial pathogens of concern are the same as those for dried milk powders, however control over these hazards is essential because of the vulnerable status of infants. The microbial quality of dry-blended products depends on the quality of ingredients as there is no heat treatment to destroy bacteria in the final product. Several outbreaks have been associated with infant formulae (Appendix 2), many of which have been caused by improper preparation and handling of infant formulae by consumers.
Concentrated milk products	Microbial pathogens are generally not associated with concentrated milks due to the low water activity of these products.
Butter and butter products	Pasteurisation of cream used in butter manufacture results in the destruction of vegetative microorganisms, although preformed toxins and spores may carry over to butter. The preservative properties of butter are based on moisture distribution. In addition salt in moisture droplets also have a preservative effect. Several outbreaks of foodborne illness have been linked to the consumption of butter (Appendix 2).
Ice-cream	The heat treatment applied to ice cream mix destroys pathogenic microorganisms. However, pathogens may be introduced with the addition of ingredients. Pathogens will not grow in ice-cream, but may survive freezing. There have been documented outbreaks of foodborne illness due to consumption of ice-cream (Appendix 2). The outbreaks have been linked to the use of raw ingredients or improper heat treatment during preparation of ice-cream in the home, and contamination during commercial ice-cream manufacture.
Cultured and fermented milk products	The heat treatment of milk is sufficient to destroy vegetative microorganisms and rapid growth of starter cultures inhibits the outgrowth of spore-formers. Pathogenic microorganisms are prevented from growth by the low pH; the presence of lactic acid, and by refrigerated storage. Cultured and fermented milks have been associated with only limited outbreaks of foodborne illness (Appendix 2).
Dairy desserts	Heat treatment by pasteurisation or UHT results in the destruction of vegetative cells. Contamination may occur after heat treatment with the addition of further ingredients, or through survival of spores of *B. cereus*.
Dairy-based dips	Where pasteurisation or other heat treatments are employed, vegetative cells will be destroyed. However, spore-formers can survive heat treatments and other hazards can be introduced with the addition of heat labile ingredients after heating. The low pH of these products assists in their microbial stability.
Casein, whey and other functional milk derivatives	Milk fats, casein and whey protein components are derived from milk and cream that have received at least a pasteurisation heat treatment and so will be free from vegetative pathogens. In some products, vacuum drying leads to the destruction of vegetative cells, and low water activity of many products ensures that the outgrowth of pathogens is very unlikely and would lead to eventual die off.
Colostrum	Bovine colostrum is pasteurised before drying, however, pathogens may be protected by the elevated fat and total milk solids content compared to standard bovine milk. Contamination after processing is a concern, although the low water activity of colostrum powder will prevent growth and vegetative cells will eventually die off.

As dairy products rarely undergo a further pathogen reduction step prior to consumption (*e.g.* cooking), prevention of contamination and control over bacterial growth, storage time and temperature is of particular importance in minimising potential exposure to pathogens. Most dairy products have a relatively short shelf-life, especially milk (10-16 days under optimum storage conditions) thus storing dairy products according to manufacturer instructions and following good hygiene and handling practices in the home is also important.

In addition, good hygiene practices in the home during reconstitution, storage and feeding of reconstituted products such as dried milks and infant formulae are essential to avoid recontamination and/or multiplication of pathogens in these products.

In Australia, illness from dairy products is rare. Between 1994-2004, there were only eleven reported outbreaks directly attributed to dairy products and eight were associated with consumption of unpasteurised milk. The majority of outbreaks associated with unpasteurised milk were a result of consumption of raw milk on a farm or camp setting. In the other outbreaks in Australia where a dairy product was one component of the food vehicle identified, the affected foods typically included cream filled cakes and custards. In these cases it is possible that eggs or other ingredients in these products, and not the dairy component, may have been responsible for the illness.

While commercial dairy products have rarely been identified as sources of foodborne illness by health departments in Australia, there have been a number of reports of outbreaks of illness associated with consumption of dairy products internationally. Of a total of 135 outbreaks associated with dairy products reported during the period 1973-2003, 16.2% were attributed to pasteurised milk and 12.5% were attributed to cheese produced from pasteurised milk. However, in these outbreaks, a fault with the pasteurisation process or post-pasteurisation contamination has been identified or suspected as the source of infection. While ice-cream was responsible for a number of these outbreaks (16.2%) but in the majority raw egg ingredients were identified as the source of infection rather than the dairy component.

Unpasteurised dairy products are the most common cause of internationally reported dairy-associated outbreaks of illness (43.4%). Over 22.8% of outbreaks were attributed to unpasteurised cows milk and 11.8% of outbreaks were attributed to unpasteurised cheese produced from raw cows milk. Clearly both internationally and domestically, unpasteurised dairy products are the most common cause of dairy-associated outbreaks of illness.

The lack of epidemiological data linking pasteurised dairy milk products in Australia to outbreaks of foodborne illness attests to the safety of these products. In contrast, outbreaks continue to be associated with consumption of raw milk, both in Australia and overseas.

Dairy products likely to support the growth of pathogens and prone to contamination after final heat treatment may be categorised as higher risk than other dairy products. While dairy products that are inherently stable with respect to pathogens, if correctly formulated, can be classified as low risk. The degree of risk is based on:

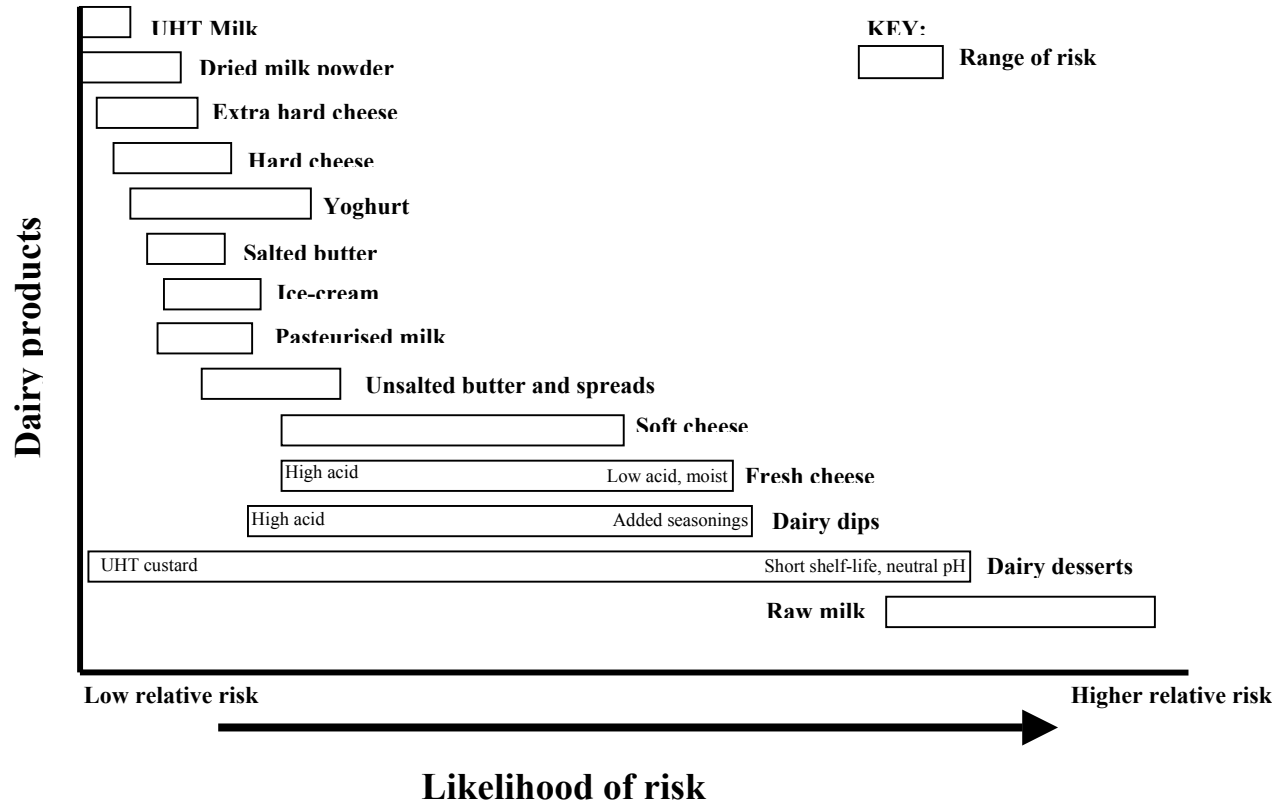
- intrinsic properties of the product (*i.e*. the impact of water activity, pH, salt concentration, etc and their effect on the growth of contaminating microorganisms);
- extent to which food is exposed to factory environment or handling after heat treatment;

- hygiene and control during distribution and retail sale; and
- degree of reheating or cooking before consumption (many dairy products are ready-to-eat, so this is rarely a factor).

Qualitative objective methods of describing the relative risk to public health associated with dairy products is extremely difficult. The following table attempts to provide a relative rating for selected dairy products, and to convey a perception of some level of risk to consumers.

Risk	Dairy product	Context
Higher risk	Soft cheeses Dairy desserts	Mild pH, long shelf-life Mild pH, fermentable carbohydrate, long shelf-life
Intermediate risk	Unsalted butters/low fat spreads Pasteurised milk and cream	Absence of salt, high moisture content Mishandling and poor hygiene, minimal post-pasteurisation hurdles
Low risk	Yoghurts Salted butters Hard cheeses Extra hard cheeses	Low pH High salt concentration Low a_w, low pH Low a_w, low pH

The relative risk from dairy products may also be expressed graphically as a continuum:



The actual ranking of the dairy products is quite variable. Once a shelf-stable UHT product is opened, it may become contaminated and when subjected to temperature abuse it could become a high-risk food. In contrast, the low pH and low water activity of extra hard cheese means its will be very robust and unlikely to support the growth of any pathogen that adventitiously contaminates the surface. Dried milk powders and infant formulae are inherently stable products due to their low water activity, however these products may be prone to contamination, and upon reconstitution become higher risk, especially if improperly reconstituted and stored.

8. Conclusions

Australian dairy products enjoy a reputation for high standards of quality and safety. There have been few reported failures i.e. incidents of foodborne illness attributed to dairy products in the market place in recent years. While dairy products have been the vehicle in some outbreaks, the cause is often multifactorial involving contaminated non-dairy ingredients, post-processing (post-pasteurisation) contamination and/or poor hygiene.

The safety of dairy products is due to the use of heat treatment and a combination of control measures up and down the supply chain. Control of animal health, adherence to good milking practices, cooling of milk, and control over milking parlour hygiene have been important in reducing the microbial load in raw milk entering Australian dairy processing facilities.

The almost universal use of pasteurisation in milk processing in Australia has resulted in the marketing of dairy products with an excellent reputation for safety and product quality. The processing industry has introduced significant measures to ensure product safety, including the adoption of codes of practice, adherence to Listeria control protocols, and the extensive use of HACCP-based food safety programs supported by laboratory verification.

Notwithstanding the above, there is need for ongoing vigilance and further development of safety control measures. Over the past twenty years we have seen the emergence of new pathogens and the re-emergence of traditional pathogens in various foods. These organisms often occupy specific environmental niches and may arise through changing technologies, methods of food handling and preparation, dietary habits and population. Post-processing contamination in-plant and the maintenance of control over contamination and storage conditions during transport, retail display and home use remain major factors impacting on the safety of dairy products.

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PART B: CHEMICAL RISK PROFILE

1. INTRODUCTION

As part of the development of a Primary Production and Processing (PPP) Standard for dairy products, an evaluation has been undertaken of the potential risks that may occur as a result of the use, or presence, of various chemicals at different points through the primary production and processing chain. This information has been used to identify areas where further data or additional controls may be necessary to ensure that any public health and safety concerns are addressed, and also to identify any gaps in the current regulation which should be addressed through a PPP Standard.

The assessment of risk to public health and safety for dairy products from the use, or presence, of chemicals in dairy products has been undertaken in the form of a Chemical Risk Profile, which examines a broad range of chemicals either used in or present in dairy products.

There are many regulations currently in place that control the use or presence of chemicals in dairy products. Agricultural and veterinary chemicals, which are used extensively in primary production, are assessed as part of a pre-market evaluation and approval process and generally have maximum residue levels identified in the Food Standards Code (the Code). Similarly, food additives and processing aids undergo pre-market evaluation and approval and have maximum use levels identified in the Code. Sanitisers are assessed before use and their use controlled through HACCP-based food safety plans. There are a wide variety of regulatory controls for contaminants at both the primary production and food manufacturing levels. Within food regulations, maximum levels (MLs) are established for many heavy metals and also for a variety of organic chemicals found in the environment that may contaminate food. For some metals, there are also so-called 'generally expected levels' (GELs) established, which are non-regulatory measures designed to identify contamination outside the normal range. The general principle used for all contaminants is that the levels found in food should be as low as reasonably achievable.

On-farm QA and legislated food safety programs require farmers to use all agricultural and veterinary chemicals according to label instructions, accurately identify treated animals, keep records of all chemical use and separate milk from treated animals for the duration of the prescribed WHPs. Most dairy companies also carry out screening for antimicrobials in milk at the farm level and from bulk milk tankers.

Sources of potential chemical risks
A paddock-to-plate flowchart identifying potential chemical inputs into dairy products is presented in Figure 1. This perspective helps to define the nature of the chemical inputs at specific stages through the dairy supply chain.

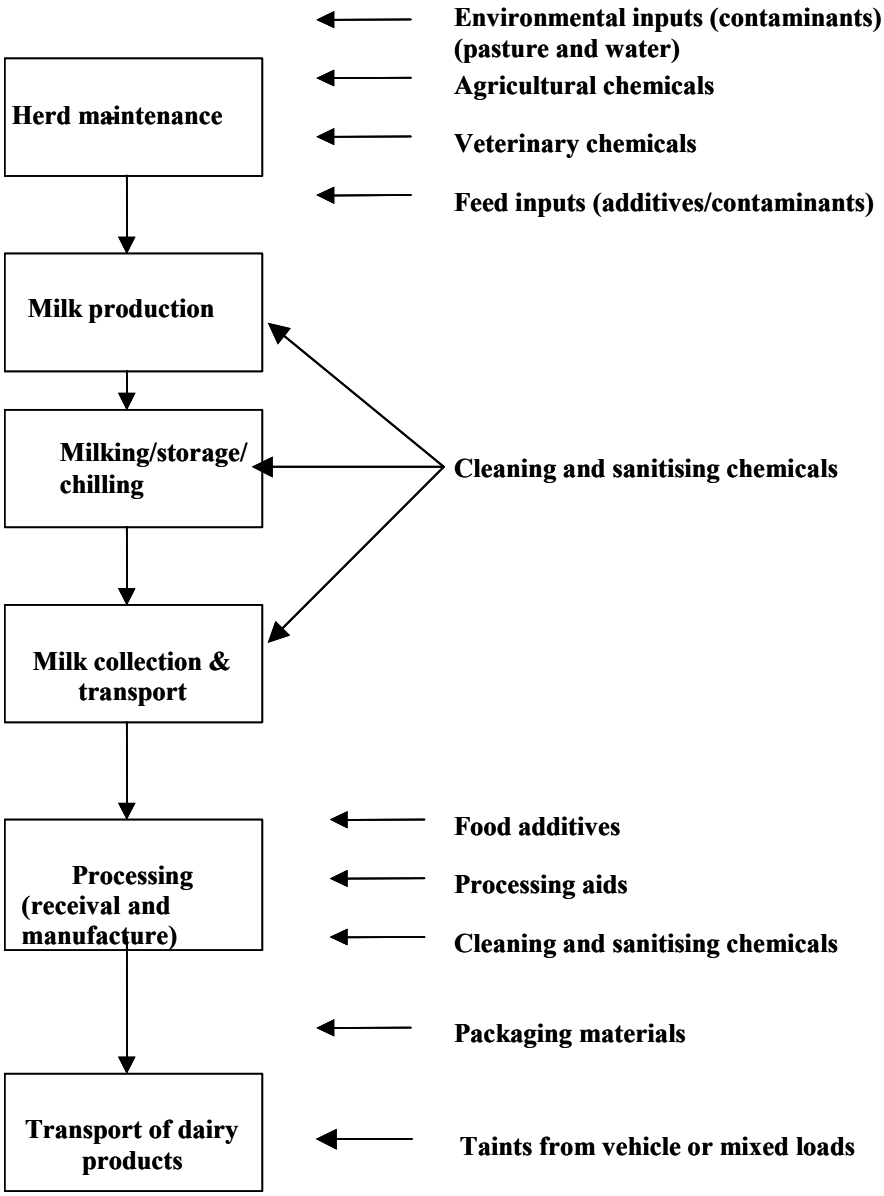

Figure 1 Potential gateways for the introduction of chemicals into the dairy primary production and processing chain

In this assessment the potential sources for chemicals to be introduced into milk and milk products that have been considered include, biological sources, agricultural practices and food processing. Contaminating chemicals, such as heavy metals, endogenous plant toxicants, mycotoxins, or anthropogenically-produced chemicals, such as dioxins, may be ingested by dairy cattle as a result of their presence in the soil or feed. Agricultural chemicals such as herbicides and pesticides are used in association with dairy production. Veterinary applications also include the administration of antimicrobials and anthelmintics, which can be carried-over into the milk. Direct contamination, for example from sanitisers has also been considered. In addition, the potential for undesirable endogenous chemicals to form within dairy products due to processing (*e.g.*, polycyclic aromatic hydrocarbons) or microbiological activity (*e.g.*, biogenic amine or fungal toxin production) has been assessed. Food additives and processing aids may be used during production of dairy products.

Packaging of dairy products may also lead to the unintentional migration of chemicals from the packaging material into dairy produce. Finally, the transport of milk and milk products may lead to potential chemical contamination from containers or from other food commodities.

Physiology & pharmacokinetics of the blood/milk barrier in dairy animals
Milk is synthesised in the alveolar gland. For milk synthesis, milk components and their precursors have to pass the blood milk barrier, a membrane that separates the blood flowing in capillaries from the alveolar epithelial cell of the udder. However, during this process certain environmental chemicals present in the blood can also potentially pass through the membrane and be incorporated into the milk at concentrations comparable to chemical levels in other fatty compartments in the body.

Agricultural and veterinary chemicals, and environmental contaminants, can enter the bloodstream by the two main routes: ingestion and dermal contact (Burnam and Palmiter, 1987; Hale and Ilett, 2002). These chemicals circulate in the bloodstream, either bound to carrier proteins such as albumin and lipoproteins or in their free form, and distribute among tissue compartment throughout the body (Figure 2). The individual carry-over rates of chemicals into milk depend upon the physiology of the animal, the bioavailability of the compound, the general stability, the amount taken up and the time and frequency of exposure in addition to the chemico-physical properties of the chemical molecule (e.g. lipo-or hydrophilicity, molecular weight and preference to carrier proteins). These factors may all influence and limit the transfer of molecules across the gut, into the blood. Furthermore, ingested and absorbed chemicals may be re-distributed, detoxified and excreted by other body organs. The specific detoxification of metal contaminants has been attributed to glutathione metabolism and metallothioneins. These low molecular weight metal-binding proteins actively scavenge sulphydryl-reactive metals (i.e. cadmium, arsenic, mercury and lead) (Burnam and Palmiter 1987).

Indirect contamination from pesticides and herbicides may occur via oral or dermal exposure. In all cases, the chemical may be absorbed, subsequently metabolised and eventually excreted into the milk of the lactating animal.

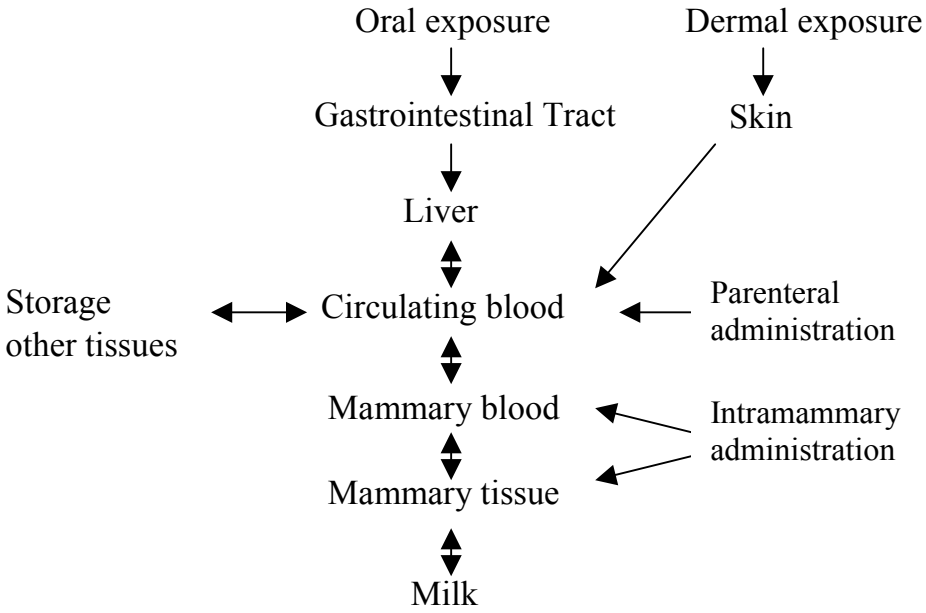

Figure 2: Schematic model of pharmacokinetic pathways in dairy animals

Following administration and absorption, the rate of distribution of chemicals within the body is a function of tissue perfusion, which is the rate of blood flow through the various tissues. The chemicals accumulate first in highly vascular organs. Then, as equilibrium states are reached and metabolic pathways are activated, the chemicals redistribute. Chemicals with high lipid solubility concentrate in tissues with higher fat content, such as adipose tissue, brain, liver, kidney, and may in the case of lactating animals include the milk (Landrigan *et al*., 2002).

There are two pathways by which chemical compounds can cross the blood-milk barrier: 1) paracellular diffusion, which relies on open junctions between cells, and is relevant in colostrums composition and 2) transcellular diffusion, which involves chemicals passing through cells. The latter occurs when the open junctions have 'closed' (about 72 - 96 hours after the mother gives birth). From a physiological perspective, open junctions are important in the transfer of immunoglobulins to colostrum from the mother to the suckling new born. Contaminants and therapeutic compounds can also potentially transfer from blood into milk via paracellular diffusion immediately after birth (Hale and Ilett, 2002). Although this is of limited significance for milk produced in Australia, as routine industry practice excludes the first eight milkings from the supply chain, it may be relevant to the complementary medicine industry producing colostrum-based products.

Transcellular diffusion, occurs mainly via passive transport, which in general allows the passage of lipophilic components of molecular weight <800 Da; thus, lipid solubility of a chemical is a primary factor for its incorporation into milk. Factors that affect the lipophilic character of a chemical include its chemical structure and its degree of ionisation in the body compartments. Low molecular weight water-soluble chemicals (<200 Da) can also cross cell membranes with the bulk transfer of water. Chemicals of high molecular weight (>800 Da) tend not to pass through the membrane and are unlikely to carry-over into milk.

Also, chemicals (e.g. heavy metals) that are highly bound to either plasma proteins or erythrocytes are unlikely to passively diffuse into milk (Clewell and Gearhart, 2002b).

Other factors that affect the presence of chemicals in milk are its degree of biotransformation and its elimination rate. Some weak bases can preferentially enter the milk as a result of the pH gradient that exists between the blood (pH = 7.4) and milk (pH = 6.5-6.8), whereas other ionic compounds are transported into the milk via active uptake mechanisms (Clewell and Gearhart, 2002a).

2. Existing framework for through-chain management of chemicals for dairy products

As mentioned in the General Introduction, the dairy industry (farm and manufacturing sectors) is subject to a number of food safety regulations, both at the Commonwealth and State levels.

In Australia, the Australian Pesticide and Veterinary Medicines Authority (APVMA) is responsible for registering agricultural and veterinary chemical products, granting permits for use of chemical products and regulating the sale of agricultural and veterinary chemical products. The APVMA undertakes its responsibilities under the authority of the Agricultural and Veterinary Chemicals Code Act 1994. Following the sale of these products, the use of the chemicals is then regulated by State and territory 'control of use' legislation.

Before registering such a product, APVMA must be satisfied that the use of the product will not result in residues in food that would present an unacceptable public health and safety risk. When an agricultural or veterinary chemical is registered for use or a permit for use granted, following a risk assessment, the APVMA publishes maximum residue limits (MRLs) in the APVMA MRL Standard. These MRLs are then adopted into control of use legislation in some jurisdictions and assist States and Territories in regulating the use of agricultural and veterinary chemicals (see Appendix 8). When the APVMA registers a new product it also approves the associated product label, which provides the approved directions for use, including withholding periods.

State government agencies have responsibility for administering controls regarding the use of Agvet chemicals, from the point of retail sale. These agencies are mostly contained in departments of agriculture, although in some jurisdictions, some responsibilities are performed by health departments (WA) or the Environmental Protection Agency (NSW). Regulation of Agvet chemicals by States includes:

- promoting best practice and developing codes of practice for chemical use
- licensing pest control operators and aerial spraying operators
- establishing and administering rules and regulations in relation to chemical use, e.g. prohibited uses, allowed on- and off-label uses (includes how veterinarians can use vet chemicals), and control of off-target movement, e.g. spraydrift; and
- audit, compliance and enforcement activities

State/Territory governments and statutory bodies, including SDAs, have legislative responsibility for administering controls regarding the use of agricultural and veterinary chemicals from the point of retail sale.

Australian dairy farms have hazard analysis critical control point (HACCP)-based on-farm food safety programs as required by State government legislation. These food safety programs require record keeping and are subject to audit by State/Territory authorities or company auditors.

The dairy industries' traceability systems are extensive and include QA programs and food safety plan requirements, vendor declarations, record keeping, livestock identification, inventory controls and product recall plans. A summary of the monitoring and auditing of chemical residues in milk and other dairy products are conducted through the Australian Milk Residue Analysis (AMRA) survey) and the Australian Total Diet Survey (ATDS). Although not strictly relevant to the dairy industry, the National Residue Survey (NRS) serves as an additional source of data and reflects the general standards of animal husbandry in Australia, providing verification of control measures for chemical residues. Dairy cattle comprise approximately 12 – 16% of the "cattle" meat commodity monitored in the NRS and therefore chemical residue data from this survey is also included in this report.

2.1 Australian Milk Residue Analysis (AMRA) Survey

The Australian Milk Residue Analysis (AMRA) survey is a national program that audits potential chemical inputs into Australian dairy production, including on-farm administration, prior to harvest, through to the final dairy product. The farm and manufacturing sectors of the dairy industry is subject to a number of food safety regulations at both the Commonwealth and State levels (see Appendix 8 for additional information).

Additional data is collected from targeted testing and from testing conducted by dairy companies.

The Australian Quarantine and Inspection Service (AQIS) uses the AMRA Survey when certifying the residue and contaminant status of milk and milk products for export and is the competent authority that approves the core annual AMRA Survey.

A national dairy auditing program was established in 1998 that is summarised as the AMRA survey. Prior to this, industry and States conducted similar monitoring and auditing programs. The AMRA Survey is conducted by ANZDASC on behalf of the Australian dairy industry, with Dairy Food Safety Victoria (DFSV), a statutory authority, coordinating the Survey. The Survey is funded by the Australian dairy industry through the industry-owned service company, Dairy Australia. Funding for Dairy Australia is derived through an industry milk levy, and for research purposes, contributions from the federal government.

In the risk assessment to determine which chemicals will be included in the AMRA survey, the following matters are considered:

- importing country requirements, including those of the European Union;
- availability of the chemicals for use in Australia;
- approved use patterns of the chemicals for dairy cattle in Australia;
- previous conclusions of AMRA Survey reviews;
- other possible sources of chemical contaminants
- test results from:
  - previous AMRA Surveys;
  - company testing programs conducted either for regulatory or customer requirements;
  - results of the National Organochlorine Residue Management (NORM) Program;

- results of the National Antibacterial Residue Minimisation (NARM) Program;
- results of the NRS Cattle Meat Survey; and
- results of the NRS Grain Products Survey

The core AMRA survey is designed to monitor bulk milk for the presence of chemical residues. The experimental design of the survey is risk-based; samples are collected and analyses carried out on a statistical basis. It provides a mechanism to monitor compliance with required conditions of use, including withholding periods (WHPs[27]) after treatment with antibiotics

For the period 2001-2004, 11,000 analyses have been conducted in the core survey to investigate the potential presence of AgVet chemicals in bovine milk and dairy products. Seven detections above the relevant MRL/ML/ERL for all the agricultural and veterinary chemicals, environmental pollutants, natural toxicants or heavy metals tested, have been found during this period, equating to a compliance of level of 99.94%. There have been a few instances of detection in milk of the anthelmintic, triclabendazole, which is not approved for use in lactating dairy cattle in Australia.

There have also been single breaches of the MRLs for the antibiotics, penicillin G and cloxacillin.

Gentamicin was also detected on one occasion in milk although it is not approved for use in lactating dairy cattle in Australia.

A summary of the formal AMRA Survey test results from 1 January 1998 to 30 June 2004 is shown in Tables 2 to 5. Results are shown as the total number of analyses and the number detected with residues above the maximum residue limit (MRL), ML or extraneous residue limit (ERL) (ADASC, 2003).

2.2 Australian Total Diet Study (ATDS)

FSANZ monitors the food supply to ensure that existing food regulatory measures provide adequate protection to consumer health and safety. The Australian Total Diet Study (ATDS) is part of that monitoring and is conducted approximately every two years (Appendix 7; (FSANZ, 2005d).

The ATDS, formerly known as the Australian Market Basket Survey, is Australia's most comprehensive assessment of consumers' dietary exposure (intake) to a range of food chemicals including pesticide residues, contaminants, nutrients, additives and other substances. Populations are assessed as a whole as well as for different age and gender groups in Australia. Due to the uncertainties in some of the data used for the assessment, certain assumptions needed to be made. These assumptions are likely to lead overall, to a conservative (*i.e.* over-estimate) for dietary exposures (*e.g.* all foods within a food group are assumed to contain the additive, nutrients or contaminant being assessed.)

[27] The withholding period (WHP) s a stipulated time which must elapse between treatment with a particular chemical and slaughter or harvest. It allows time for the chemical to be metabolised or breakdown or dissipate. Withholding periods are given on the label instructions of any agricultural or veterinary chemical sold and must be obeyed by law.

Exposures are estimated by combining usual patterns of food consumption, as derived from the national nutrition survey (NNS) data, with both current and proposed levels of use of the food chemicals in the food. Food consumption data from the 1995 Australian NNS and the 1997 New Zealand NNS are used for dietary modelling, along with concentration data for the food additives, nutrients or contaminants from a variety of sources (including the Code, manufacturers' use data and analytical data from surveys) (Baines J, 1998). Details on consumption data for dairy products from the NNS are given in Section 2.2 and Appendix 5 of the Microbiological Risk Profile of Dairy Products.

The survey estimates the level of dietary exposure of the Australian population through the testing of food representative of the total diet. In order to achieve more accurate dietary exposure, the foods examined in the ATDS are prepared to a 'table ready' state before they are analysed. As a consequence, both raw and, processed and cooked foods are examined. FSANZ coordinate the survey while the States and the Northern Territory purchase and prepare the food samples.

The Australian Market Basket Survey (ANZFA, 1996b) detected levels of permethrin residues in cheese of 0.08 mg/kg (the MRL for permethrin in milk products, in the fat, is 0.05 mg/kg. There have been no subsequent detections of this pesticide.

2.3 National Residue Survey (NRS)

Australia has an active national residue-monitoring program. The National Residue Survey (NRS) was established under the *National Residue Survey Administration Act 1992* for the purposes of monitoring and reporting levels of contaminants in food, inputs to production and or the environment.

Industry participation in the NRS is generally required to meet requirements for market access, export certification or national standards. AQIS uses NRS data when certifying the residue and contaminant status of certain commodities for export. The meat from other minor dairy producers, sheep, goat, buffalo and camel, is also included in the NRS. The NRS also covers some commodities that are used in stockfeed. Each commodity is reviewed in consultation with the relevant industry and where necessary, residue monitoring is modified in accordance with emerging needs and changing circumstances.

Residues are classified as being 'present' if their concentration is greater than the limit of reporting (LOR) established for NRS purposes. The NRS typically sets the LOR at 10-20% of the Australian Standard MRL, ERL or ML.

The 2000 – 2001 cattle meat survey showed that of the 60 222 analyses that were conducted there was a 99.99% compliance to the MRLs, with only 5 samples having residue detections exceeding the MRL. There was a single detection of the antimicrobial neomycin that was above the MRL, but this was in a beef cow. The traceback investigation revealed the residue most likely resulted from failure to observe the WHP and inadequate record keeping of individual animal treatments.

3. Potential carry-over of residues from agricultural production systems

3.1 Introduction

In Australia, the Australian Pesticide and Veterinary Medicines Authority (APVMA) is responsible for registering agricultural and veterinary chemical products, granting permits for use of chemical products and regulating the sale of agricultural and veterinary chemical (AgVet) products. AgVet chemicals are regulated through State authority 'control of use' legislation and the States administer the Food legislation.(see Appendix 8 for further details).

The agricultural and veterinary chemicals used in the dairy production chain undergo rigorous assessment processes by the Office of Chemical Safety, the Environmental Protection Agency, and the National Industrial Chemicals Notification and Assessment Scheme and by State agencies prior to registration by the APVMA. This report provides information on the agricultural chemicals which may have relevance to the dairy industry and the auditing results over the past few years.

In contrast to many of the animal husbandry systems in place internationally, the Australian dairy industry is predominantly pasture-based, *i.e.*, dairy cattle are not confined to dairy sheds for prolonged periods of time. This system, together with the different climatic conditions in Australia, results in different agricultural and veterinary chemicals usage which reflects specific needs. Australia does not experience the same problems as seen in intensive farming systems, which results in a different disease profile and hence different veterinary product usage. APVMA registration requirements in Australia allow for the application of whole herd treatments and a policy of nil milk WHP for anthelmintics.

Agricultural and veterinary residues in milk can arise from a number of potential sources, including indirect exposure through feeds, or direct treatment of cattle.

The same groups of chemicals, for example insecticides, may be used for both agricultural and veterinary purposes, however they are used for different purposes, at different doses and are applied differently.

The AMRA survey is designed such that a particular AgVet chemical can be traced back to its source use. Generally, agricultural pesticides involve indirect exposure, whereas veterinary parasiticides involve direct application, such as drenching or pour-on anthelmintic products.

The MRLs for agricultural and veterinary chemicals for milk, are listed in Appendix 9 (extracted from section 1.4.2 of the Code).

Colostrum

Colostrum is considered within the Dairy Primary Production and Processing Standard and therefore is included in the chemical risk profile. Standard dairy industry practice excludes colostrum from milk entering the production stream, however colostrum is marketed as a complementary medicine, and is regulated by the Therapeutic Goods Agency.

Colostrum is derived from the first four milkings of cows after calving. It is a lactose- and fat-reduced, high-protein product, which is manufactured without the addition of additives or artificial ingredients. However, chemicals may be potentially transferred to colostrum from the cow by paracellular diffusion (see Section 2).

The QA program for colostrum manufacture as a therapeutic is separate from that of the dairy industry. The levels of chemical residues potentially present in colostrum are regulated by MRLs in the Australia New Zealand Food Standards Code. Although individual manufacturers have strict compliance specifications and immunoglobulin concentrations, bacteria counts, somatic cell counts and antimicrobials are measured, there is no monitoring of colostrum by the State Dairy Authorities.

3.2 Pesticides and Herbicides

Organophosphates and synthetic pyrethroids
Insecticides, such as the organophosphates and synthetic pyrethroids are common agricultural chemicals in use in dairy production in Australia, and are used as grain protectants and pest control in pastures and feeds.

Organochlorines
The broad-spectrum insecticide/acaricide endosulphan is an organochlorine which is currently used in agricultural production, for example in the cotton industry. Unlike other organochlorines (see Environmental Contaminants section 6.4.3), it shows low persistence in the soil.

Herbicides
Herbicides are used in plant management and in the dairy industry are used to reduce weeds. Selective spot-spraying is common in pastures and raceways, in addition to the use of herbicides, such as glyphosate, in pre-pasture establishment. As for other AgVet chemicals, MRLs for herbicides in milk are included in Appendix 9. A stock WHP applies to most of these herbicides, varying from anything from 1-2 days, up to several weeks.

Fungicides are included in NRS monitoring, however residue levels have been found to be negligible, if present at all. Due to the very low levels of herbicide residues associated with grain products (NRS Grain Program), they are infrequently included in the NRS. A recent review of herbicide usage by Dairy Food Safety Victoria (DFSV) found that the potential risk of herbicide residues in milk is low and therefore the AMRA survey does not currently include testing for herbicide residues.

3.3 Survey results for agricultural chemicals

3.3.1 Milk

In the past seven years of the AMRA survey there have been 33, 382 analyses of organochlorines, organophosphates and synthetic pyrethroids with no detections of residues at levels greater than the MRL/ERL (Table 1). The decline in the numbers of analyses since 1998 reflects the progressive success of management practices. In the 2005-6 AMRA survey, thirty samples will be collected nationally and analysed for organochlorine residues.

Table 1: Comparison of Annual Pesticide Test Results in bulk milk (ADASC, 2002 – 2005)

Survey Year	Pesticides					
	Organochlorines		Organophosphates		Synthetic Pyrethroids	
	No. analyses	No. >ERL	No. analyses	No. >MRL	No. analyses	No. >MRL
1998/1999	2616	0	3597	0	2289	0
1999/2000	1512	0	2079	0	1323	0
2000/2001	1632	0	2244	0	1428	0
2001/2002	896	0	1568	0	784	0
2002/2003	872	0	1526	0	763	0
2003/2004	600	0	1078	0	539	0
2004/2005	240		3864		1932	

As a result of a locust plague, which occurred in South Australia and New South Wales during spring 2000 and summer 2001, approved insecticides were used as control measures by landholders, the Departments of Agriculture and the Australian Plague Locust Commission. During this period, the APVMA issued permits to allow the use of certain synthetic pyrethroid chemicals to control the Australian plague locust; the permits covered the use (*e.g.* WHPs for domestic and export markets) of the active ingredients: Lambda-Cyhalothrin, Gamma-Cyhalothrin, Betacyfluthrin, Alpha-Cyopermethrin and Cypermethrin.

Additional monitoring of pesticide residues in milk was carried out as a result of this and the results are shown in Table 2. Results from targeted sampling undertaken during locust plague activity in the vicinity of dairy regions in northern Victoria and southern NSW in summer 2004, are shown in Table 3; no residues were detected.

Table 2: Australian Targeted Milk Testing for Locust Plague Chemical Residues (spring 2001 – summer 2001) (ADASC, 2002)

State	Residue Type	No. analyses	No. samples >MRL
SA	Organochlorines	3	0
	Organophosphates	3	0
	Fipronil	3	0
NSW	Organochlorines	41	0
	Organophosphates	41	0
	Fipronil	41	0

Table 3: Australian Targeted Milk Testing for Locust Plague Chemical Residues (summer 2004) (Dairy Australia personal communication)

State	Residue Type	No analyses tested	No. samples >MRL
Vic (northern)	Chlorpyrifos	60	0
	Cypermethrin	60	0
	Diazinon	60	0
	Fenitrothion	60	0
	Fipronil	60	0
	Malathion	60	0
NSW (southern)	Chlorpyrifos	25	0
	Cypermethrin	25	0
	Diazinon	25	0
	Fenitrothion	25	0
	Fipronil	25	0
	Malathion	25	0

3.3.2 Animal tissue

Table 4 shows a summary of the NRS monitoring results for agricultural chemical residues in cattle tissue (urine, fat, kidney, liver, muscle and faeces), in dairy and beef cattle, from 2000 to 2004 (DAFF 2005a). No pesticide residues were found apart from in the 2000 – 2001 season when bioresmethrin residues were detected, this chemical is now de-registered and is no longer permitted for use.

Table 4: NRS Cattle Meat Survey Results for Pesticides 1 July 2000 – 30 June 2004 (DAFF 2005a)

	No. of analyses	Residues Detected > MRL	No. of analyses	Residues Detected > MRL	No. of analyses	Residues Detected > MRL	No. of analyses	Residues Detected > MRL
	2000 - 2001		2001 - 2002		2002 - 2003		2003 - 2004	
Pesticide *	28 227	4 (bioresmethrin)	30 986	0	23 211	0	22 028	0

* organochlorines, organophosphates, synthetic pyrethroids and benzoyl ureas.

The 2002 – 2003 NRS survey was also used as a source of information regarding dairy animals other than cattle; no pesticide residues were detected in sheep, goat, buffalo or camel tissue (see Table 10).

3.4 Stockfeed

Although FSANZ does not regulate stockfeed per se, the paddock-to-plate approach necessitates the consideration of safety aspects of stockfeed for different dairy animals. The Australian dairy industry is mainly pasture-based and approximately 60% of cattle feed comes from grazing, the remaining 40% is provided by supplementary feed. Feedlot-based dairy farming is not common, although feedpads for providing supplementary feed are used.

The types of stockfeed consumed by dairy animals are listed in Table 5 and a comprehensive list of stockfeed assumed by the APVMA in their registration assessments is listed in Appendix 13 (APVMA, 2002). The main stockfeed supply chain includes field peas, lupins, cotton, canola, maize, wheat, barley, sorghum, oats, soybean, grass, grains, hay and silage. These crops are used to produce a wide range of stockfeed products including grain, hay, silage and seed meals. Supplementary feed is used to increase energy conversion and performance. There are three aspects to this feed, nutritional, mineral additions and therapeutic. Dairy cattle are fed nutritionally enhanced supplementary feed on a routine basis. In addition to paddock grasses, the cattle also are fed high protein food such as milled or pelleted products which are administered under strict quality assurance (QA) programs. Depending on environmental conditions, mineral additions may be required, this is supplied as salt licks containing minerals such as magnesium, molybdenum, selenium, copper, boron and cobalt. Grain-fed animals are also fed charcoal to prevent bloat. Supplementary feed may also contain the veterinary therapeutics virginiamycin and monensin for specific short periods of time (see 4.1.1).

In Australia, supplementary feed used in the dairy industry is generally purchased from the stockfeed milling industry or directly from the grain grower and there are established QA systems which manage chemical risks.

A National Commodity Vendor Declaration form accompanies purchased feed or grain stating which chemicals have been used and that they have been used according to label directions, observing the required WHP. In general, for low input production systems, the majority (90%) of the feed (pasture and silage) is grown on farm as compared to purchased feed (10%), whereas for high production systems, up to 55% of the feed is grown on-farm and 45% is bought in (Dairy Australia, personal communication).

The use of agricultural chemicals can potentially result in the presence of chemical residues in stockfeed that may be subsequently fed to livestock. Measurable residues of the chemical may occur in the milk of livestock consuming the previously treated feed commodity as a normal part of the diet. The APVMA establishes guideline MRLs to cover the residues that may arise in milk (as well as animal tissues and eggs) as a result of livestock dietary exposure (APVMA 2002), however the MRLs for stockfeed are not uniformly legislated; stockfeed legislation in States, other than NSW, is under review or development. These commodities are subject to MRLs based on animal transfer studies and livestock dietary exposure. The anticipated chemical residue levels in animal feed commodities are determined from crop residue trials conducted according to good agricultural practice (GAP). Currently, with the exception of NSW, there is no harmonised legislation for stockfeeds in Australia. Products registered by APVMA will include if necessary label restrictions for grazing and cutting for stockfeed (WHPs) (APVMA 2002). Thus, regulation of stockfeed is currently carried out by each State under separate legislation. Regulation of the NSW Stock Foods Act 1940 Act has most recently been updated by NSW (NSW 2005), and adopts a table of MRLs from the APVMA MRL Standard (APVMA 2005a).

Foreign ingredients in stockfeed are prescribed under the NSW legislation, for example prohibited substances, weed seeds and plants and toxic compounds are listed and the allowed proportion (if any) specified.

Stems and leaves of cotton crops are not used in dairy production due to the risk of the uptake of chemical residues (Blackwood *et al.*, 2000). The advent of bovine spongiform encephalitis overseas precipitated the ruminant feed ban in Australia, which was legislated in 1997 by the Department of Primary Industries, including the abolition of feeding materials such as bone meal, feather meal and fishmeal, with the exception of stockfeed containing tallow, gelatine and/or milk products (Bennet 2002). In 2001, the feed bans were broadened to include chicken litter and chicken faeces (QDPI&F 2005).

Care with stockfeed production is required to ensure that there is no cross-contamination of grain between different species' stockfeed and to prevent the inadvertent delivery of Restricted Animal Materials. QA programs that prevent exposure of ruminants to Restricted Animal Materials by the feed millers supplying pre-mixed feed, to ensure that stockfeed produced for chicken feed which may contain antibiotics, is not fed to ruminants.

Table 5: Stockfeed used in dairy production

Animal	Stockfeed
Buffalo	Can survive on poorer pasture areas that are too wet or of marginal quality for cows, can forage in swampy conditions (eg reed beds), can survive on crop stubble and grain/legume by-products
Camel	Trees, grass, shrubs, hay, fresh cut lucerne, pelleted foods, cottonseed meal, barley grass
Cow	Pastures of white clover, perennial ryegrasses, paspalum and kikuyu supplemented with silage, hay, molasses, grain, pelleted foods, green crops
Goat	Pastures, green crops (barley, oats, wheat etc), hay, straw, silage, seeds and grains, protein supplements (coconut, cottonseed, linseed, rapeseed, soybean, peanut meals), cereal bran, sugar cane molasses.
Sheep	Pasture, clover (available in plentiful supply only in high rainfall or irrigated areas), otherwise hay, pelleted feeds

Genetically modified stockfeeds

Australian livestock industries use a wide range of stockfeed components that can potentially be sourced from genetically modified (GM) crops (Lamb and Cunningham, 2003). GM stockfeed may be derived from imported GM grains or feed supplements (and are labelled accordingly) or from Australian-grown GM cotton. Currently cotton is the only GM crop that is included in stockfeed although the Office of the Gene Technology Regulator (OGTR) has also approved GM canola for commercial production, but use is restricted due to State moratoria. Both FSANZ and the OGTR have approved these GM crops as safe for human (and animal) consumption) and that there are no adverse effects resulting from using approved GM crops for stockfeed.

In a recent dairy feeding experiment, GM cottonseed was fed as part of the mixed ration to lactating Holstein cows.

The nutritional value of whole cottonseeds from genetically modified cotton was equivalent to cottonseed from non-transgenic cotton varieties, as indicated by the similar performance of the cows' dry matter intake, milk yield, milk composition, body weight and body condition (Castillo *et al*., 2004).

Many animal feeds are derived from the same GM food crops that are used for human consumption. Concerns are occasionally expressed that this practice may pose an indirect risk to humans, through consumption of the meat, milk and eggs derived from such animals. Scientific evidence published so far (OECD 2003) indicates that feeding GM plant material to livestock and poultry does not affect the nutritional value or safety of the meat, milk and eggs derived from those animals.

Genetically modified stockfeed has been found to have no adverse effects on animal health or commodity production (OECD 2003), and the risk for consumers is considered to be nil or negligible. Novel DNA may not necessarily be present (or detectable) when a GM commodity is used as part of a stockfeed mix (Castillo *et al.,* 2004). Conversely, fragments of plant DNA (both transgenic and non-transgenic) have been detected in animal tissues, including milk, but there is no basis to suppose that novel DNA poses a hazard (OECD 2003). Furthermore, it would be exceptionally unlikely for an expressed protein of any plant gene to be found intact in milk.

Monitoring of chemicals in stockfeed

The Australian dairy industry is largely based on all-year-round pasture feeding, however, as mentioned above, pasture feed is supplemented with additional feed material, predominantly grain. As grains are susceptible to the production of mycotoxins, the AMRA sampling program includes an audit of contaminants in milk originating from cattle feed inputs. In 2005/6 this will include 30 samples to be analysed for residues of Aflatoxin M1, taken from areas that may source hay and straw from crops that are susceptible to aflatoxin e.g. peanuts. A further 120 milk samples will be collected nationally and analysed for organophosphates and synthetic pyrethroids that are contained in registered agricultural products for insect control on harvested and stored grains.

The annual NRS survey is another source of information on chemical residues in agricultural commodities, which may be used in stockfeed. For example, the crops detailed in Table 6, (barley, field peas, lupin, oats, wheat and wheat bran) are all used in stockfeed. The overall compliance levels are high for these crops. Several samples were non-compliant for the organophosphate fenitrothion.

This chemical is registered for use on cereal grains and for disinfestation of grain storage structures and grain handling equipment, but not registered for use on pulses (lupin and field pea) and no Australian Standard (MRL) is set. One barley sample also had dichlorvos and fipronil residues above the Australian Standard (DAFF 2005a). Those samples found with residues greater than the MRL are unlikely to be found at detectable levels in milk. On the whole, the biotransfer of AgVet chemicals from stockfeed to dairy products from stockfeed and via the blood-milk barrier, is considered to be negligible.

Table 6: Agricultural residues measured in crops used for stockfeed, 2003 – 2004 season (collated from NRS survey, 2003-4) (DAFF 2005a)

	Barley		Field pea		Lupin	
	No. analyses	Residues detected> MRL	No. analyses	Residues detected> MRL	No. analyses	Residues detected> MRL
Insect-growth regulators	293	0	42	0	51	0
Fumigants	14	0	5	0	5	0
Fungicides	14293	0	42	0	51	0
Organo-chlorines	2932520	0	378	0	459	0
Organo-phosphates	7819	1 (dichlorvos)	938	1 (fenitrothion)	1211	1(fenitrothion)
Synthetic Pyrethroids	1465	0	214	0	255	0
Physiological Modifiers	293	0	42	0	51	0
Other insecticides	586	1 (fipronil)	84	0	102	0
Environmental contaminants	492	0	24	0	87	0

Table 6 (contd.): Agricultural residues measured in crops used for stockfeed, 2003 – 2004 season (collated from NRS survey, 2003-4) (DAFF 2005a)

	Oat		Wheat		Wheat Bran	
	No. analyses	Residues detected> MRL	No. analyses	Residues detected> MRL	No. analyses	Residues detected> MRL
Insect-growth regulators	32	0	770	0	33	1 (methoprene)
Fumigants	1	0	34	1 (phosphine)	-	-
Fungicides	32	0	770	0	33	0
Organo-chlorines	288	0	6561	0	297	0
Organo-phosphates	707	0	21 329	0	693	2 (fenitrothion)
Synthetic Pyrethroids	96	0	3850	0	165	0
Physiological Modifiers	32	0	770	0	33	0
Other insecticides	64	0	1540	0	66	0
Environmental contaminants	30	0	1755	0	-	-

4. Potential residues from veterinary and animal husbandry systems

4.1 Dairy cattle

4.1.1 Antimicrobials

Antibiotics represent the largest group of veterinary chemicals administered to dairy cattle. These compounds are listed under the S4 schedule and are only prescribed for animals under veterinary control. The different groups of antimicrobials include β-lactams, tetracylines, sulphonamides, macrolides and aminoglycosides. Some of these groups of antimicrobial agents are also used in human medicine. Further information is provided in Appendix 11.

Antibiotics are used therapeutically for a wide range of infectious conditions in cattle. However, with the exception of occasional outbreaks of disease in a herd, the therapeutic use of antibiotics is on an individual animal basis (JETACAR, 1999).

Therapeutic herd treatment in dairy (and beef) cattle with antibiotics typically include:

- Mastitis in dairy herds (e.g. beta-lactams, tetracyclines, lincomycin)
- Respiratory infections in cattle (e.g. tetracyclines, tylosin, tilmicosin, ceftiofur, erythromycin, neomycin, trimethoprim – sulfonamide combinations);
- Dry cow therapy for mastitis and sub-clinical mastitis control in dairy herds (e.g. beta-lactams, neomycin, tetracyclines);
- Control of lactic acidosis and bloat (virginiamycin[28] and polyethers[29])

[28] The approved uses of virginiamycin in sheep and cattle are to reduce the risk of rumen acidosis from grain consumption. Virginiamycin is not used for humans, but is closely related to an antibiotic which is used for humans as an 'antibiotic of last resort' to treat infections which are resistant to other antibiotics. The APVMA published its Review Findings in November 2004 and in February 2005 the APVMA Board made the regulatory decisions to cancel the registration of products whose sole purpose is growth promotion, and to vary the labels of products whose purpose is prevention of lactic acidosis in cattle and sheep so that

- Coccidiosis in young animals (e.g. polyethers);
- Enteric infections (e.g. tetracyclines, neomycin);
- Hoof infections such as footrot (e.g. penicillin, tetracyclines, ceftiofur, trimethoprim-sulfonamide combinations).

Antimicrobials are used in dairy animals as chemotherapeutic agents and are not used as growth promotants. However, some antibiotics may be administered in feed for short periods of time, for example to treat lactic acidosis. If antibiotics are introduced via feed, as large molecules they are not readily absorbed by the gut and are unlikely to be carried-over into the milk. In such cases, a nil WHP is assigned.

Antimicrobial drugs are generally used in dairy animals in one of three ways (Agricultural and Veterinary Chemicals Code Act 1994):

1. Whole herd treatments, where there is "blanket" treatment of the majority of animals *e.g*. intramammary dry cow treatments (in some cows);
2. Partial herd treatments, where a minority of the herd is treated, *e.g.* intramammary dry cow treatments; and
3. Individual cow treatments, where only individual or a few animals are treated at any time, *e.g.* lactating cow intramammary treatments and injections for bacterial disease control.

Problems associated with antimicrobial residues

Antibiotic residues in milk can affect the microbial activity required for cheese and yoghurt production, as starter cultures are sensitive to inhibitors in milk including residues of antimicrobial drugs. Furthermore, starter cultures may be inhibited to varying extents influencing the relative proportion of strains in cheese-milk during manufacture. This can impact on cheese flavour profiles. The adverse impact on dairy starter cultures is avoided by setting milk MRLs less than the minimum concentration of antibiotic which inhibits the most sensitive strain of starter culture (APVMA 2003a). The strict compliance of current dairy regulations ensures that antibiotic levels are kept within their regulatory limits so that there are no potentially costly processing problems downstream. Thus, every tanker of milk is tested through on-site facilities and milk is strictly monitored to check that it is "fit for purpose".

In Australia, dairy producers are required to conform with the WHP on the antibiotic label; this is reinforced by the inclusion of a visual marker (a blue dye) in intramammary preparations. Intramammary antibiotics that do not contain blue dye are not registered in Australia, which restricts their availability for use.

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usage is restrained to a maximum of 28 days in cattle and sheep. An additional restraint is that the product may be used only once in a 12-month period and may not be used for any other animal species. The major registrant of virginiamycin products has sought review of the APVMA's decisions in the Administrative Appeals Tribunal. The review was undertaken to assess concerns relating to human health and toxicity, environmental contamination and efficacy (APVMA, 2005).

[29] Monensin is an example of a commonly used polyether ionophore, which may be used for increasing milk yields or to prevent bloat (JETACAR, 1999). It has a nil WHP based on its residue depletion profile and is therefore not tested for. There is no human usage of this antimicrobial.

Antibiotic residues in food have anecdotally been associated with causing allergic reactions and other toxicities in susceptible people. However, the formative risk assessments carried out on antibiotics determine microbiological end points, which are used to set the microbiological Acceptable Daily Intake (ADI). This takes into account the colonisation barrier in the gut, changes in microflora metabolism, and selection for antibiotic resistant organisms (Munro and Reeves, 2005). The ADIs and MRLs for antimicrobial agents are extremely conservative with large uncertainly factors built-in; antimicrobials exceeding the ADI are not eligible for registration, and no MRL is recommended by the APVMA. It is therefore highly unlikely that antibiotics, if carried over in milk, would induce a toxic response.

The potential development for antimicrobial resistance is discussed in Section 5.7.2.

4.1.2 Ectoparasiticides

Some active insecticidal compounds are the same as those used in agricultural practice, and may be used in the control of external parasites in livestock farming practice in Australia; under veterinary use, these are referred to as ectoparasiticides. Ectoparasiticides include botanicals, synthetic pyrethroids, organophosphates, carbamates, and macrocyclic lactones. Macrocyclic lactones may be used as both internal and external parasiticides (i.e. endectocides).

Organophosphate pesticides, such as chlorfenvinphos, fenthion, malathion and pirimiphos-methyl, were introduced for controlling crop and livestock pests in the 1950s as an alternative to organochlorines. They are generally much less persistent than organochlorines and are degradable, and therefore do not accumulate in animals to any great extent.

4.1.3 Endoparasiticides

Helminths and liver fluke, which are found predominantly in the southern temperate zones of Australia, are commonly controlled with endoparasiticides. Anthelmintic compounds including the benzimidazoles, levamisole and the macrocyclic lactones are widely used in Australian dairy farming practices, with the latter being the most predominantly used of these three chemical groups. In Australia, the flukicide agent triclabendazole is only registered for use in non-lactating cattle.

4.1.4 Other veterinary chemicals used routinely in dairy production

In addition to the use of antibiotics and endo- and ectoparasiticides, other veterinary drugs are used routinely in dairy animal husbandry, these may be either whole-herd veterinary drugs or single animal veterinary medicines, all of which are regulated by the APVMA.

Veterinary medicines include reproductive therapy drugs, such as oestrogen, which is used to synchronise endogenous hormones. These chemicals are listed under Table 5 of the MRL standard (APVMA, 2005). This table includes “situations where residues do not or should not occur in foods or animal feeds; or where the residues are identical to or indistinguishable from natural food components; or are otherwise of no toxicological significance”.

Single animal use medicines include non-steroidal anti-inflammatory drugs or anaesthetics. In addition, there are a range of dermatological preparations which are used routinely following GAP in dairy production (APVMA, 2005).

4.1.5 Bovine somatotropin (BST)

Bovine somatotropin (BST) is a hormone produced naturally by all cows, and is necessary to stimulate milk production. Since the early 1980s, it has been manufactured and this form is known as recombinant bovine somatotropin (rBST).

The Australian Agricultural and Veterinary Chemicals Council (now the APVMA) received an application to register rBST in Australia in 1991. However, although all the data requirements for registration were met, for example, regarding efficacy and safety, the application was not approved due to a number of trade concerns. rBST is not routinely monitored for in imported dairy produce, as it is not detectable above natural levels of BST. Recombinant BST is also not approved for use in New Zealand, Canada or in the European Union. However in the U.S.A., treatment of cows with rBST was approved in February 1994, and has been extensively used over the ensuing period. In addition, 24 other countries have given approval for use of rBST.

The Joint FAO/WHO Expert Committee on Food Additives (JECFA) reviewed the safety data in 1998 and found that the available data on the identity and concentration of residues in animal tissues provide a wide margin of safety for consumption of residues in food when the drug is used according to good practice in the use of veterinary drugs. The Committee concluded that the presence of drug residues in animal products does not present any human health concerns.

4.2 Survey results for veterinary chemical residues

4.2.1 Milk

In the past seven years of the AMRA survey 3, 467 milk samples were tested for antimicrobial residues (equating to 89, 121 analyses) with 99.997% compliance with the MRL. Table 7 details the number of analyses for each antimicrobial tested from 1998 - 2005 In addition to the detection of cloxacillin in this survey, there have previously been two samples with antimicrobial residues that exceeded the MRLs. One of these was for gentamicin during 2000/2001 and the other for penicillin G in 2001/2002. .

Table 7: Comparison of Annual Antimicrobial Test Results in bulk milk (ADASC, 2002 – 2005)

Survey Year	Antimicrobials	
	No. analyses	No. >MRL
1998/1999	15663	0
1999/2000	10143	0
2000/2001	12852	1
2001/2002	14094	1
2002/2003	13689	0
2003/2004	14580	1
2004/2005	8100	0

The AMRA survey also indicated 100% compliance with the MRLs of a large range of endoparasiticides tested over a seven year period; triclabendazole showed 99.88% compliance with its MRL (Table 8). Of the four detections above the MRL, three occurred during 2000/2001 and one in 2002/2003. Macrocyclic lactone testing commenced in 2002/2003 and there have been no residue detections above the MRLs to date.

Levamisole and benzimidazoles were included in the 2003/4 Survey for the first time. These chemicals are widely used, however validated methodologies for testing for these compounds in Australia have only recently been developed. No detections have been reported to date.

Table 8: Comparison of Annual Anthelmintic Test Results (ADASC, 2002 – 2005)

Survey Year	Anthelmintic (triclabendazole)		Anthelmintic (macrocyclic lactones)		Anthelmintic (benzimidazoles)		Anthelmintic (levamisole)	
	No. analyses	No. >MRL	No. analyses	No. >MRL	No. analyses	No. >MRL	No. analyses	No. >MRL
1998/1999	681	0	0	-	0	-	0	-
1999/2000	441	0	0	-	0	-	0	-
2000/2001	476	3	0	-	0	-	0	-
2001/2002	522	0	0	-	0	-	0	-
2002/2003	507	1	436	0	0	-	0	-
2003/2004	540	0	155	0	195	0	38	0
2004/2005	300	0	295		90	0	18	0

4.2.3 Animal tissue

The NRS cattle meat survey results for veterinary chemicals is shown in Table 9; in the 2001 – 2002 season, 63 272 analyses were conducted for the NRS survey and there was a 99.99% compliance with the MRLs/MLs, with only four samples with residues detected above the MRL.

Table 9: NRS Cattle Meat Survey Results for veterinary chemicals 1 July 2000 – 30 June 2002 (DAFF, 2005a) (DAFF, 2005a)

	Number of Analyses	Residues Detected > MRL	Number of Analyses	Residues Detected > MRL
	2000 - 2001		2001 - 2002	
Hormones	5 426	0	3 489	2 (zearanol and boldenone)
NSAID*s	650	0	642	0
Beta- agonists	915	0	921	0
Antimicrobials	20 731	1 (neomycin)	22 813	2 (dihydrostreptomycin and neomycin
Anthelmintics	3 373	0	3 431	0

*non-steroidal anti-inflammatory drugs

Table 9 (contd.): NRS Cattle Meat Survey Results for veterinary chemicals 1 July 2002 – 30 June 2004 (DAFF, 2005a)

	Number of Analyses	Residues Detected > MRL	Number of Analyses	Residues Detected > MRL
	2002 - 2003		2003 - 2004	
Hormones	4 492	1 (zearanol)	5068	0
NSAID*s	996	0	972	0
Beta- agonists	1 625	0	1 715	0
Antimicrobials	18 057	0	18 830	2 (neomycin)
Anthelmintics	1 926	1 (doramectin)	2 033	1 (triclabendazole)

Zearanol was detected in one cattle faeces sample above the MRL, this can occur in beef cattle as a result of treatment with a hormonal growth promotant (HGP) (dairy cattle are not administered HGP), or naturally in animals through the metabolism of ingested zearalenone, a mycotoxin associated with stored grains (see section 7.5.5). Boldenone was detected in one cattle urine sample above the MRL.

Two kidney samples from beef cattle had antibiotic residue detections above the MRL, one for neomycin and one for dihydrostreptomycin. Although no cause for dihydrostreptomycin detection was found, the trace-back investigation for the neomycin detection revealed that the farmer had observed the 30-day withholding period, prior to consigning the cow from which the sample was collected to slaughter.

In the 2002 – 2003 season, 53, 827 analyses were conducted as part of the NRS Cattle Meat Survey and there was 99.94% compliance for Agricultural and Veterinary (AgVet) chemicals. In the 2003-2004 season, 51,627 analyses were carried out with 99.99% compliance. One cattle liver sample contained triclabendazole residues exceeding the MRL and trace-back investigations indicated that the most likely cause of this residue was accidental treatment of an animal destined for slaughter.

4.3 Minor species (goat, sheep, buffalo)

There is little monitoring of chemical residues in milk from the "minor" dairy producers (goat, sheep, camel and buffalo). Furthermore, a recent risk-based assessment of unpasteurised goat milk identified that many of the veterinary products used were not registered for use in goats (Appendix 14; (Pointon *et al*., 2004). There is only a limited range of registered antibiotics for the control of mastitis in goats (Appendix 14; a complete list of registered products can be found on the APVMA website (APVMA, 2005)). Similarly, there is only a limited range of anthelmintics that are registered for use in dairy goats. This may lead to off-label use and potential unregulated residues in goat milk.

The benzimidazoles (e.g. fenbendazole) have a zero WHP in cattle whereas they have a different profile (lower plasma levels) in goats compared with sheep. Therefore, to achieve comparable efficacy it is common practice to double-dose goats. This is also the case for the macrocyclic lactones ivermectin and moxidectin. The MRL for these macrocyclic lactones in bovine milk is 0.08 mg/kg, but no MRL is set for goats and thus the residue level must be zero.

Closantel is an anthelmintic used in sheep against blood feeding worms (such as Barbers Pole and Liver Fluke); there are no MRLs for cow or goat milk. Also, some AgVet chemicals may only be permitted in specified States, for example, there is currently a permit for trichlorfon (neguvon) and morantel in NSW; neither product has been identified as being used in dairy goats in SA (Pointon *et al*., 2004).

A preliminary investigation was recently carried out by the Dairy Authority of South Australia, in conjunction with a South Australian Veterinary Clinic, into potential residue problems with moxidectin and closantel usage in goats. There are no MRLs for these drugs in goat milk and therefore the level in milk should be zero, however, it was found that residues for both drugs were detectable in the milk eight days after treatment (Pointon *et al*., 2004).

The presence of veterinary residues in sheep, goats, camels and buffalo is monitored in the NRS (albeit in non-dairy matrices) (Table 10); more chemicals are monitored in sheep reflecting the relative agricultural importance of the commodity. The only non-compliance in the 2002 – 2003 survey was for moxidectin use in goats.

Table 10: Summary of residues found in sheep, goat, buffalo and camel meat; NRS survey results 1 July – 30 June 2003 (DAFF, 2005a)

Analyte	No. Anal.	Res > MRL	No. Anal.	Res > MRL	No. Anal.	Res > MRL	No. Anal.	Res > MRL
	Sheep		Goat		Buffalo		Camel	
Hormones	2045	0	-	-	-	-	-	-
NSAIDs	879	0	-	-	-	-	-	-
Beta-agonists	1830	0	-	-	-	-	-	-
Pesticides	21 406	0	2522	0	180	0	260	0
Antimicrobials	16 452	0	-	-	-	-	-	-
Anthelmintics	1841	0	515	3*	-	-	-	-

No. Anal: number of analyses Res. >MRL: number of residues exceeding the MRL

* non-compliance of moxidectin use. Moxidectin is not registered for use in goats so there is no established MRL. Moxidectin use under veterinary advice was confirmed in one animal at traceback, with the goat being slaughtered before the WHP recommended by the veterinarian had elapsed. Moxidectin use, without veterinary advice, was confirmed in the other two animals. The WHP for sheep was observed, which is not appropriate for goats (DAFF 2005a).

The overall potential risk with minor dairy producing species is associated with off-label usage of veterinary chemicals. More specifically, there is often no pharmokinetic data to determine WHPs for minor species, so these need to be conservatively applied with advice from a technical expert

5. Potential residues from environmental and anthropogenic chemicals

5.1 Potential contaminants in dairy products

Contaminants in food are regulated, in part, by establishing MLs for various foods in the Code. However MLs are part of a broader risk management framework for food contaminants. Other regulations that encourage practices that in turn reduce contamination of food operate at all levels of government in Australia. These include waste management/disposal programs, water quality programs, industrial zoning regulation and environmental safeguards. The public health risks associated with food contaminants can also be managed through establishing guidelines, codes of practice, or through education of the public on safe food consumption patterns.

In many cases, the potential for contamination of food is limited as a result of these other regulations and specific food regulation may be unnecessary. When a food standard is considered necessary for a particular contaminant as a risk management measure, this is achieved by establishing an ML in particular food commodities.

MLs are the legal limits enforced through the State and Territory Food Acts and are, in general, used only when other mechanisms of control are considered insufficient or inadequate to safeguard the health of consumers.

FSANZ regulates the presence of contaminants in food through Standard 1.4.2 – Contaminants and Natural Toxicants. This Standard sets out the maximum levels (MLs) of specified metal and non-metal contaminants and natural toxicants in nominated foods.

As a general principle, regardless of whether or not a ML exists, the level of contaminants and natural toxicants in all foods should be kept as low as reasonably achievable (the ALARA principle).

Although the Australian dairy industry relies upon all-year-round pasture feeding, additional feed material, predominantly grain, also forms part of the feeding program. Other minor feedstuffs such as hay, straw and such as crop trash may also be used, particularly when feed is less plentiful such as during Australia's recent drought conditions. Some minor feedstuffs such as peanut hay and sorgham are susceptible to fungal infection so monitoring of residues of aflatoxin M1 contaminants in milk, originating from hay and straw and from these crops is included in residue testing programs. During times of drought, more novel feedstuffs may also be utilised, such as oranges and bakery products.

The presence of environmental contaminants in forage is subject to wide variations, mainly depending upon the vicinity of the crops to a source of contamination including mould infestation.

As part of a review of potential chemical hazards for dairy products, eleven environmental contaminants, eight groups of stockfeed contaminants, two forms of natural chemical toxins/residues and one chemical formed during food processing, were reviewed in addition to endogenous plant toxins, which may be present in pasture (Table 11).

Regular monitoring of environmental contaminants occurs to some degree through the AMRA survey and NRS, although not all the potential contaminants covered in this paper are considered in these surveys. Inclusion of particular contaminants in these surveys will depend on the results of previous surveys, the likelihood of significant contamination, and the potential public health risk associated with the contaminant.

Heavy metals are included in the AMRA survey every 2-3 years in order to meet trade criteria and were first included in the AMRA Survey in 2001/2002. No residues were found (in 112 samples) at levels greater than the ML, and for this reason, heavy metals were not included in 2002/2003 or 2003/2004 survey. The NRS Cattle Residue Survey also monitors environmental chemical residues such as PCBs, heavy metals and the mycotoxin, zearanol. Although this survey monitors matrices other than milk, it provides an insight into the levels and types of residues found in all cattle (including ~15% dairy cattle) and residues in other milk-producing animals.

Table 11: Potential chemical contaminants for dairy products and their sources

Contaminant	Source
CHEMICAL CONTAMINANTS	
Arsenic	Environmental contamination.
Cadmium	Environmental contamination
Dioxins and dioxin-like Polychlorinated biphenyls	Environmental contaminant. Contaminated feed (Belgium)
Iodine	Environmental contaminant; milk production and processing
Lead	Environmental contaminant.
Mercury	Environmental contaminant; manometers with mercury used in dairy farms
Polychlorinated biphenyls (PCBs)	Environmental contaminant
Radionuclides	Environmental contaminant
Selenium	Environmental contaminant; AgVet contaminant
Zinc	Environmental contaminant
NATURAL TOXINS	
Biogenic amines	Endogenously formed within dairy products
Fungal toxins	Formed within dairy products
Aflatoxin M1, M2 and M4	*Aspergillus flavus,* and *A. parasiticus* contamination of corn, peanuts and other feed ingredients
Corynetoxin	*Rathayibacter toxicus* contamination of annual rye grass
Cyclopiazonic acid	*Penicillium sp*. and *Aspergillus sp.* contamination of stockfeed and dairy products
Endogenous plant alkaloids	Natural plant defence chemicals
Fumonisin B_1	*Fusarium moniliforme* plus several less common species contamination of corn
Ochratoxin A	*Aspergillus ochraceus* and *Penicillium verrucosum* contamination of barley, wheat and many other commodities
Trichothecenes; T-2 and HT-2 toxin Deoxynivalenol (DON); Vomitoxin	*Fusarium graminearum, F. crookwellense* and *F. culmorum* contamination of wheat, barley and corn
Zearalenone	*Fusarium graminearum, F. crookwellense* and *F. culmorum* contamination of wheat and corn
CHEMICALS FORMED DURING FOOD PROCESSING	
Polycyclic aromatic hydrocarbons (PAHs)	Contaminant formed during processing (e.g. smoking)

5.2 Heavy Metals

5.2.1 Sources of contamination

Metals can potentially contaminate dairy products through their presence in the soil, uptake by crops used in stockfeed or metal contamination during processing. Metals may enter the soil through agricultural practices, for example, as components of fertilisers and /or industrial contamination. In addition to the consumption of crops grown in soils with high metal contents, dairy animals may directly consume soil.

Soil consumption by grazing cattle has been estimated at 0.5 kilograms per day (Van Hooft, 1995), however soil consumption is relative to the grazing pattern and may be more of an issue for beef cattle grazing dryer pastures close to the ground.

Metal contamination through processing may occur through metal pick-up from containers and metal cooking utensils. The bioavailability of trace elements in soil is about 1.5 times lower than in feed (Van Hooft, 1995).
In general, the transfer of heavy metals to milk is limited. Exceptions are the fat-soluble organic mercury and lead compounds. The highest transfer to milk is noted for lead and arsenic, followed by mercury and cadmium (Vito - LUC - RUG, 2003). In order to satisfy export requirements, these four heavy metals have been tested for in the AMRA survey.

Arsenic occurs naturally in both organic and inorganic forms; drinking water contains largely the inorganic form of arsenic, whereas food contains more than 90% of its arsenic in the organic form. It is widely distributed in the environment and has been used in agriculture; therefore arsenic is present in most human foods. The use of phosphate fertilisers on agricultural land may be a significant source of arsenic and, in some circumstances this could lead to elevated levels in crops. The level of arsenic varies in plants and therefore levels in dairy products may be increased when animals consume plants with high levels of arsenic. For instance, sheep and goats may graze plants with higher arsenic contents than cows. Old sheep dips could also be point sources of arsenic.

Cadmium is a widespread contaminant in many agricultural products worldwide. The use of phosphate fertilisers on agricultural land may be a significant source of cadmium and, in some circumstances this could lead to elevated levels in crops. Since cadmium is retained in the topsoil, concentrations can increase if the application of these materials to soils continues over long periods. Exposure of animals to cadmium results from feed intake and is a function of the concentration of cadmium in the feed and the amount of feed consumed. Moreover, uptake of soil during grazing (or soil contaminated feeds) is an additional factor contributing to total exposure of individual animals.

Lead can potentially contaminate dairy products through environmental contamination or through contamination of water supplies. Utensils containing lead, such as tin or pewter, may also cause contamination.
The elimination of lead solder from food cans has reduced the hazard of exposure to lead from canned food, particularly from canned milk and infant formula.

Mercury occurs naturally in the environment with levels in the topsoil varying between 0.02 and 0.15 mg/kg. Therefore, despite barriers to bioavailability, there is potential for ingestion of low levels of mercury by pasture-fed dairy cattle.

<u>Regulation of heavy metals in relation to dairy products</u>
For arsenic, cadmium and mercury there are no MLs in the Food Standards Code (the Code) for milk or other dairy products. Dairy products are considered to be an insignificant dietary source of these heavy metals and therefore do not require a control, such as an ML.

Because of the increased sensitivity of infants for lead toxicity, the Code has an ML for lead in infant formula in standard 1.4.1 – Contaminants and Natural Toxicants. The maximum level (ML) of lead in infant formula is 0.02 mg/kg.

5.2.2 Arsenic

Hazard Identification and Characterisation

A risk assessment on arsenic was last performed by FSANZ[30] in Proposal 157 – Review of the maximum permitted concentrations of metal contaminants in food (ANZFA, 1999e). Arsenic in its inorganic form is toxic to humans. The most relevant toxicological data, other than industrial exposure, are derived from studies of human populations exposed to arsenic in drinking water, with chronic toxicity and cancer the most sensitive indicators of toxicity.

Chronic ingestion of low doses of inorganic arsenic initially produces cutaneous vasodilation, then hyperpigmentation and hyperkeratosis with subsequent atrophy and degeneration of the skin leading over a period of time to the development of skin cancers.

FSANZ established a lowest observed effect level (LOEL) for inorganic arsenic, based on population studies in Taiwan, where drinking water exposures for periods of 12 years to whole-of-life were associated with cancers (skin, liver, bladder, lung). Only skin cancer was detected at the lowest LOEL. There is growing evidence for a threshold in a dose-response relationship between inorganic arsenic and various cancers. The lowest LOEL for human skin cancer was approximately 0.0029 mg/kg bw/day, based on a review of epidemiological data. On the basis of the available data, this level is considered to be close to a 'threshold' value, below which increased incidence of skin cancer was not associated with arsenic exposure.

The provisional tolerable daily intake (PTDI) for inorganic arsenic is 0.003 mg/kg bw/day. While based on exposure to drinking water rather than food, it is considered appropriate for use in assessing the risk from inorganic arsenic in food. It should be noted however, that this PTDI for arsenic does not incorporate any safety factors (ANZFA, 1999d; ANZFA, 1999e).

The Joint FAO/WHO Expert Committee on Food Additives (JECFA) has assigned a provisional tolerable weekly intake (PTWI) of 0.015 mg/kg bw for inorganic arsenic (WHO, 1989a), noting that the margin between the PTWI and intakes reported to have toxic effects in epidemiological studies, was narrow. The provisional status of the maximum weekly intake was continued due to the desire to lower the arsenic intake of those individuals exposed to high levels of inorganic arsenic in drinking water.

The International Agency for Research on Cancer (IARC) has classified inorganic arsenic into group 1 (carcinogenic for humans), for the ability to induce primary skin cancers (IARC, 1987).

Dietary exposure

The arsenic content of tissues and body fluids is markedly influenced by the level of intake, but experiments in cows suggest there is a barrier to excessive mammary uptake, as milk concentrations were not increased by feeding diets containing 25 times the arsenic level in normal rations (Jensen, 1995). Transfer factors from feed to milk between 10^{-4}-10^{-5} kg/L (concentration in milk/concentration in feed, dry matter) have been reported (Van Hooft, 1995; Rosas *et al*., 1999; Perez-Carrera and Fernandez-Cirelli, 2005).

[30] As the Australia New Zealand Food Authority (ANZFA)

A dietary exposure assessment was conducted as part the 19th ATDS (ANZFA, 2001). The Survey reported no detection of total arsenic in feta cheese from cow's milk (limit of reporting 0.01 mg/kg), and mean levels below the limit of reporting (0.002 mg/kg) for feta cheese made from sheep's milk.

The 20th Australian Total Diet Survey (FSANZ, 2003) estimated exposure to total arsenic between 9-48% of the PTDI set for inorganic arsenic. Inorganic arsenic analysis was more expensive than total arsenic analysis. Therefore, only total arsenic was tested, which is an overestimate of inorganic arsenic. The 20th ATDS did not detect total arsenic in full fat milk and, while in cheddar cheese total arsenic was not detectable at 0.02 mg/kg (FSANZ, 2003) These results demonstrate consistent non-detectable or very low levels of total arsenic and as a consequence inorganic arsenic in Australian milk and milk products.

5.2.3 Cadmium

Hazard Identification and Characterisation

A risk assessment on cadmium was last performed by FSANZ[31] in Proposal 144 – Review of the maximum permitted concentration of cadmium in food (ANZFA, 1997). Cadmium has been most recently assessed by JECFA in 2003 (WHO, 2003). Cadmium has an extremely long biological half-life in man and accumulates in the kidneys over time. The kidney has been identified as the critical organ in relation to chronic exposure to relatively low levels of cadmium and in particular the renal cortex.

An early feature of the adverse renal effects in man is the impairment of the reabsorption functions of the tubules with an increase in urinary excretion of low-molecular weight proteins. Renal injury may progress and, in severe cases, involve glomerular damage with proteinuria, aminoaciduria, glucosuria and phosphaturia. It has generally been found that tubular proteinuria, once manifest, persists even when exposure ceases. Intakes of cadmium in the range of 140-255 μg/day have been associated with increased low-molecular weight proteinuria in the elderly.

Low-molecular weight proteinuria is not accompanied by any specific histological changes and the pathological significance of this finding is unclear. However, it can be used to as an indicator of the threshold of a possible toxic effect and it is appropriate to set the provisional tolerable weekly intake on the basis of the dose-response data for this endpoint (WHO, 1989b).

The critical health outcome with regard to cadmium toxicity is renal tubular dysfunction. JECFA established a provisional tolerable weekly intake (PTDI) for cadmium of 7 μg/kg body weight per week (WHO, 2003).

This level was to ensure that cadmium concentration does not exceed 50 μg/g in the renal cortex assuming an absorption rate of 5% and a daily excretion rate of 0.005% of body burden, over a period of 50 years.

The IARC has classified cadmium and cadmium compounds into group 1 (carcinogenic for humans) (IARC, 1993a).

31 As the Australia New Zealand Food Authority (ANZFA)

Cadmium is carcinogenic in experimental animals when given by injection or inhalation, and exposure of workers by inhalation has been shown to result in pulmonary cancer. There was no evidence that cadmium is carcinogenic to humans exposed by the oral route (WHO, 2001).

Dietary exposure

Cadmium can potentially be carried over into milk in lactating cows from intestinal absorption (Van Hooft, 1995). In a 2-week experiment where 3g cadmium (as $CdCl_2$) was administered to cattle, less than 0.022% was found in milk. The transfer factor from feed to milk for cadmium is reported to be between $2.3*10^{-5}$ –$5.8*10^{-5}$ kg/L (concentration milk/concentration dry matter in feed) (Van Hooft, 1995). In a study from Thailand, the levels of cadmium in milk were below the level of reporting and therefore, the transfer factor was reported to be zero (Parkpian *et al*., 2003a).

A dietary exposure assessment was conducted as part of the assessment of cadmium for Proposal P144 – Review of the maximum permitted concentrations of cadmium in food.

A revised dietary exposure assessment for cadmium was conducted on the basis of additional survey information (ANZFA, 2000). Cadmium concentration data used in this assessment were sourced both within FSANZ as well as submissions from external sources. The survey data indicated cadmium was not detected in full cream milk, cheddar cheese. The 19th ATDS reported no detection of cadmium in feta cheese from cow's milk and mean levels below the limit of reporting (0.001mg/kg) for feta from sheep's milk.

The 20th Australian Total Diet Survey did not detect cadmium in full cream milk, the limit of reporting for this analysis was 0.005 mg/kg (FSANZ, 2003) The maximum level of cadmium found in cheddar cheese was 0.007 mg/kg..

Cadmium residues in cow's milk were monitored in 112 samples in the 2001 – 2002 AMRA survey; there were no residue detections. In the 2003-4 NRS survey there were seven cadmium detections in sheep livers above the Australian Standard (ML) of 1.25 mg/kg. The residue levels ranged between 1.3 mg/kg and 2.4 mg/kg. Cadmium residues are a common finding in sheep offal, particularly in older animals across the southern states of Australia. A residue action level of 2.5 mg/kg for trace back purposes has been agreed between NRS and state and territory government regulatory authorities. None of the seven residue detections were above the action level, so trace backs were not done for these detections (DAFF 2005a).

In a study in India it was found that cadmium concentrations in buffalo milk were higher than levels in cow's milk (0.10 μg/L vs. 0.07 μg/L, respectively). The study authors explained the difference, because of the different fat content of buffalo milk (7.5%) in comparison to cow's milk (3.8%) (Tripathi *et al*., 1999a).

Research on Manchego cheese during traditional cheese making and ripening indicated that cadmium concentrations increased during pasteurisation and more noticeably during ferment addition, thereafter falling during coagulation. These differences are related to the moisture content during cheese manufacture, since no differences were found for dry weight.
There were no significant differences in cadmium levels between newly made and mature cheeses (Zurera-Cosano *et al*., 1997).

A French study looked at the levels of cadmium in cows and sheep milk after 10-weeks oral administration with cadmium. Before the oral administration of cadmium, the levels of cadmium in the milk were around 0.4 µg/L in ewes and <0.2 µg/L in cows. They found that almost all the cadmium was transferred from the milk into milk products (in particular into casein fractions) and the mean levels in ewes were 3.3 ± 1.4 µg/L and 2.5 ± 1.4 µg/L in cows (Mehennaoui *et al*., 1999).

5.2.4 Lead

Hazard Identification and Characterisation

More than 40 years ago lead was removed from paint produced in Australia and petroleum products are now lead-free reducing the potential for environmental exposure to lead.

The absorption of lead from grass, hay and soil is around 5% and the gastrointestinal absorption of lead in cows is around 10%, of which 90-95% of lead is excreted in the faeces. The transfer factor of lead from feed into milk is reported between 10^{-4}-10^{-5} kg/L (concentration milk/concentration feed dry matter) (Van Hooft, 1995; Parkpian *et al*., 2003b).

A linear dose related excretion of lead from plasma into milk was found in rats and mice after intravenous injection and the lead concentration in milk was approximately 100 times higher than that in plasma 24 h after administration demonstrating efficient transport of lead into milk (Hallen *et al*., 1995). Oral feeding of lead acetate at the dose rate of 500 mg/day to limited number of lactating cows has been reported to significantly increase the milk lead excretion (Willett *et al*., 1994). However, the level of lead in milk samples from animals seven months after an acute episode of lead toxicosis was undetectable (Galey *et al*., 1990).

The concentration of lead in milk depends on the concentration of unbound lead in blood. The lead concentration in milk was found to be relatively constant up to blood levels between 0.2 and 0.3 µg/ml and increased sharply at higher blood lead levels in an accidental lead exposure over a period of 1 to 2 days through licking of burnt storage batteries by cows (Oskarsson *et al*., 1992). It has also been shown that contamination of lead resulted in blood lead concentrations exceeding 0.20 µg/ml and that the excretion of lead in milk from cows significantly increased. (Swarup *et al*., 2005).

A risk assessment on lead was last performed by ANZFA in Proposal 157 – Review of the maximum permitted concentrations of metal contaminants in food (ANZFA, 1999f).

The most widely used biomarker of exposure to lead is the concentration in blood (measured in µg/dL). The most critical effect of lead at low concentrations is reduced cognitive development and intellectual performance in children.

A number of studies in which various tests of behavioural performance were used have shown an association between blood lead concentration and reduced intelligence quotient in children exposed pre-and postnatally.

At blood concentrations below 10-15 µg/dL, the effect of confounding variables and limits to the precision of analytical and psychometric measurements increase the uncertainty of any estimate of effect. If a threshold does exist, it is unlikely to be detected because of these limitations.

However, there is some evidence of an association between cognitive deficits and exposure to concentrations even below 10 µg/dL. In conclusion, the toxicological review suggests that there is a small safety margin between the PTDI and the LOEL for children (ANZFA, 1999f).

JECFA (1993) established a Provisional Tolerable Weekly Intake of 25 µg/kg bw (equivalent to a PTDI of 3.5 µg/kg bw/day) for all age groups on the basis that lead accumulates in the body and an increase of the body burden should be avoided this level was reinforced in 2000 (WHO, 2000). This upper limit has been adopted by FSANZ as a provisional tolerable daily intake (PTDI) for the purposes of dietary modelling (ANZFA, 1999f). IARC has classified lead into group 2A (probably carcinogenic for humans) (IARC, 2004).

Dietary exposure

A dietary exposure assessment was conducted as part of the assessment of lead for Proposal P157 – Review of the maximum permitted concentrations of metal contaminants in food (ANZFA, 1999f). The modelling estimated that the mean dietary exposure for the Australian population, aged 2 years and older to be 2-6% of the PTDI.

The primary foods that contribute to dietary lead exposure in the Australian population, aged 2 years and older, excluding water, were cattle meat (29.9%), pig meat (11.7%), wine (9.8%), peach (8.7%), pineapple (5.4%) and sugar (5.0%). Dairy products were not a contributor to lead dietary intake.

The modelling estimated that the mean dietary exposure for children, aged 2 years, was 5-17% of the PTDI. The foods that contributed to the exposure of children and infants were milk (16%), pineapple juice (9%), apple juice (8%), sugar (8%), bread (8%), and tea (3%) for schoolchildren, and milk (24%), juice (21%), and bread (5%) for toddlers (WHO, 2000).

The 19th ATDS found a mean level of lead of 0.004 mg/kg in feta cheese from cow's milk and a mean level of 0.028 mg/kg in feta cheese made from sheep's milk. (ANZFA, 2001).

In the 20th Australian Total Diet Survey lead was not detected in full fat milk or cheddar cheese, with a limit of reporting at 0.01 mg/kg. Lead was also not detected in infant formula.

Lead residues in milk were monitored in 112 samples in the 2001 – 2002 AMRA survey; there were no residue detections. In the 2003-4 NRS survey, there was one detection of lead in cattle liver above the Australian Standard (ML) of 0.5 mg/kg for cattle. A residue action level for trace back has been set at 1 mg/kg and therefore no trace back action was required. There were two lead detections in livers above the Australian Standard (ML) of 0.5 mg/kg for sheep. A residue action level for trace back has been set at 1 mg/kg. None of the lead residue detections were higher than the trace back action level (DAFF 2005a).

In two studies in India it was found that lead concentrations in buffalo milk were higher than levels in cows milk (3.35 µg/L vs. 1.70 µg/L, respectively). It could be an indication that buffalo's have a higher tolerance for lead or the different fat content of buffalo milk (7.5%) in comparison to cow's milk (3.8%) (Tripathi *et al*., 1999b; Dwivedi *et al*., 2001).

In an Argentinean study, there was a significant difference ($p<0.05$) for the mean concentration in milk between young (<5 years, 17 µg/L) and old cows (>5 years, 34 µg/L), however there was a poor correlation between age and lead milk concentration ($r=0.038$) (Rubio *et al*., 1998).

In Canada there were no differences in lead content between whole, 2% butterfat and skim milks (Dabeka and McKenzie, 1987). In Kasar cheese from Turkey seasonal variation in lead content was found, with highest levels in winter months. The study authors suggested that this increase could be through differences in feed, in summer the cows eat pasture, while in winter the cows are fed silage (Yuzbasi *et al*., 2003). There were also differences between in lead content of samples from different cheese producers. These findings indicate that exposure to lead contamination in other countries may be different from Australia.

5.2.5 Mercury

Hazard Identification and Characterisation

The risk assessment on mercury was last performed by ANZFA in Proposal 157 – Review of the maximum permitted concentrations of metal contaminants in food (ANZFA, 1999g).

The different chemical forms of mercury can exhibit quite distinct pharmacokinetic and toxicological properties. From the perspective of exposure via food, inorganic mercury appears to represent a lesser hazard than organic forms of mercury. There are essentially two reasons for this. Firstly, the levels of inorganic mercury in food are low and secondly, absorption of inorganic mercury from the gastrointestinal tract is also low, therefore it appears unlikely that many people would be subject to the levels of oral intake that might be expected to have an adverse effect.

In mammals, methylmercury is almost completely absorbed in the gastrointestinal tract. Subsequently, in humans, about 90 percent of methylmercury absorbed is then excreted in the faeces. It is likely that the same patterns occur in dairy animals.

Methylmercury can be excreted at low levels in the breast milk of rats, humans, and guinea pigs (Yoshida *et al*., 1992; Sundberg and Oskarsson, 1992). The transfer factor from feed to milk is reported at 1.7*10-4 kg/L (concentration milk/concentration in dry matter in feed) (Van Hooft, 1995).

Sundberg and coworkers ((Sundberg *et al*., 1998) studied the elimination of radio labeled methylmercury in lactating and non-lactating mice exposed to methylmercuric chloride via a single intravenous injection at 0.5 mg Hg/kg body weight. A three compartment pharmacokinetic model was used to fit the data. The values for the methylmercury kinetic parameters were significantly higher in lactating than non-lactating mice: plasma clearance (93.5 and 47.1 mL/hour/kg, respectively) and volume of distribution (18,500 and 9,400 mL/kg, respectively). The terminal half-life of methylmercury in plasma was 170 hours for lactating and 158 hours for non-lactating mice. The milk to plasma concentration ratios for total mercury after methylmercury administration were lower than those seen with inorganic mercury, and varied between 0.1 and 0.7, with a mean of 0.20.

Mercury concentrations in milk were constant throughout the 9-day follow-up period post exposure. The results indicate that physiological changes during lactation alter the pharmacokinetics for methylmercury in mice (ATSDR, 1999).

In humans, methylmercury can induce toxic effects in several organs such as the nervous system, kidney liver and reproductive systems. Neurotoxicity is considered the most sensitive endpoint.

The majority of toxicological data, on which tolerable limits were previously set; have come from large scale poisonings of human population with methylmercury in Japan and Iraq. Data from these incidences confirmed an association between the consumption of fish contaminated with methylmercury and the development of neurological symptoms in adults and infants exposed *in utero*. The data indicated that the most sensitive section of the population to methylmercury poisoning is the unborn foetus (WHO, 2003).

In June 2003, JEFCA evaluated new information on methylmercury. This information included results of studies performed on laboratory animals and humans, and epidemiological studies investigating possible effects of prenatal methylmercury exposure on child neurodevelopment. A new PTWI of 1.6 μg/kg bw was recommended.

This PTWI is considered sufficient to protect the developing foetus, the most sensitive subgroup of the population (WHO, 2003).

The IARC has classified methylmercury into group 2B (probably carcinogenic for humans – sufficient evidence in animals and inadequate data in humans) and metallic mercury and inorganic mercury compounds into group 3 (not classifiable as carcinogenic to humans (IARC, 1993b).

Dietary exposure

A dietary exposure assessment was conducted as part of the assessment of methylmercury for Proposal P157 – Review of the maximum permitted concentrations of metal contaminants in food. Fish is by far the greatest contributor to dietary mercury exposure. Dairy products did not contribute to methylmercury dietary intake.

The 19th ATDS reported no detection of mercury in feta cheese from cow's milk or from sheep's milk. The 20th Australian Total Diet Survey did not detect mercury in full fat milk, cheddar cheese, and table spread margarine samples (FSANZ, 2003).

Mercury residues in cow's milk were monitored in 112 samples in the 2001 – 2002 AMRA survey; there were no residue detections.

5.2.6 Risk characterisation for heavy metals

An evaluation on arsenic, cadmium, lead and methylmercury was performed to establish whether there are potential public health and safety risks with the consumption of dairy products.

As discussed in the individual sections above, all four metals can result in serious adverse effects when consumed at high concentrations. However, when dairy animals consume these metals, a very small fraction is transferred into the milk. Data from various sources in Australia on the concentrations of these metals were non-detectable or at very low levels in milk and milk products. The data available are mainly from dairy products from cows. However, on the basis of physiology and good agricultural practices, it can be assumed that the levels of metal contaminants in dairy products from sheep, goats, buffalo or camels will be similar to levels found in dairy products from cows, i.e. very low or not detectable.

In conclusion, dietary exposure to arsenic, cadmium, lead and mercury from dairy products does not raise any public health and safety concerns.

5.3 Micronutrients

5.3.1 Essentiality role and sources

Micronutrients, are defined in physiological terms as substances that comprise less than 0.01% of the body mass.

Besides having an essential role, i.e. they are required in the human diet, the potential exits for over consumption of some micronutrients, thereby resulting in a public health and safety risk.

5.3.2 Iodine

The natural content of iodine in milk varies with the amount of iodine ingested by the cow, through drinking water and pasture. In pastures, the iodine content depends upon the concentration and nature of iodine in the soil (Dunsmore and Luckhurst, 1975). There are seasonal variations in iodine concentrations in milk, which are closely related to dietary intake; winter rations containing supplements, such as ethylene dihydroiodide result in considerable increases in milk iodine.

Although a significant amount of iodine present in milk comes from ingestion, iodine can also enter the milk through the use of iodine sanitisers on milking equipment and through the use of iodine teat dips and udder washes used to prevent mastitis.

Iodophors[32] have been used since 1962 as sanitisers by the dairy industry in both Australia and New Zealand (Thomson, 2004). Dairy products had been a major source of iodine in the New Zealand diet, but a move away from the use of iodophors has apparently resulted in lower iodine concentrations in dairy products (Thomson 2004). Iodine is still used in agricultural practice as a teat disinfectant post milking in some parts of Australia (Seal, 2004) Dairy Australia submission to P230, 2005), there are a large number of products registered for use by the APVMA register (APVMA 2005b), and water is naturally high in Queensland and parts of Australia, especially in summer (S. Rice, personal communication).

Hazard identification and characterisation

A risk assessment on iodine was most recently carried out by FSANZ in relation to Application A470 – Formulated Beverages.(FSANZ, 2005a).

A large number of human experimental, clinical, and epidemiological studies on the effects of excess iodine have been reported and reviewed in detail by both the Joint FAO/WHO Expert Committee on Food Additives (WHO, 1989a) and the US Agency for Toxic Substances and Disease Registry (ATSDR, 2004). These studies indicate that the primary effect of excess iodine is on the thyroid gland and regulation of thyroid hormone production and secretion.

Excess iodine can produce an enlargement of the thyroid gland (goitre) and/or affect the production of the thyroid hormones.

[32] Iodophors are iodine-liberating disinfectants which comprise of organic compounds containing iodine in a micellar cage of polyvinyl-pyrrolidone or nonoxyno-complex. When diluted, iodine is released and can exert its bactericidal properties. Commercial preparations contain 0.3 – 1.75% iodine. Of which 80 – 90 % is released upon dilution (Fischer *et al*., 2002).

A diminished production of thyroid hormones is referred to as hypothyroidism (and may be accompanied by goitre), while increased thyroid hormone synthesis and secretion by the thyroid gland is referred to as hyperthyroidism.

The human response to excess iodine can vary. Some individuals can tolerate large intakes (up to 50 μg/kg/day) while others may respond adversely to levels close to recommended intakes (3-7 μg/kg/day). Individuals responding adversely to relatively low intake levels often have an underlying thyroid disorder or have a long history of iodine deficiency.

A tolerable upper intake level of 1100 μg iodine/day for adults has been established by the US Institute of Medicine (US Institute of Medicine, 2001). This is the proposed level to be adopted in Australia by the National Health and Medical Research Council as part of their current review of Nutrient Reference Values (NHMRC, 2004). FSANZ has adopted this level as the upper level of intake (UL)[33] for the purpose of risk assessment for the general healthy population (FSANZ, 2005b).

In summary the ULs for iodine in the various age groups are:

1-3 years	**200 μg/day**
4-8 years	**350 μg/day**
9-13 years	**650 μg/day**
14-18 years	**1000 μg/day**
adults	**1100 μg/day**

It should be noted, however, that individuals with thyroid disorders or a long history of iodine deficiency might respond adversely at levels of intake below the UL. Therefore, the health risk for these individuals needs to be considered separately from the general population.

Dietary exposure

The iodine concentrations in food do not allow a determination of whether the iodine is from natural sources or as a result of contamination. However, an increase of around 30 μg iodine/L in milk was measured when an iodophor post-milking teat dip of 0.5% iodine was applied to Holstein cows (Galton, 2004). In a survey of the nutritional composition of Australian dairy foods co-coordinated by the Australian Dairy Corporation (now Dairy Australia), the iodine content of milk was 50 μg/L. This is the level of iodine thought to be present naturally without contamination from iodophors (Dunsmore and Luckhurst, 1975). The iodine content of key dairy products was also measured: 250ml milk contains 12.5μg of iodine, 200g reduced-fat yoghurt contains 34μg of iodine and 40g Cheddar cheese contains 6μg of iodine (Dairy Australia submission to P230, 2005).

The 22nd ATDS (FSANZ, 2004b) incorporated the iodine content of a broad sampling of dairy produce, for example: yoghurts, custard, cheese (Cheddar, Parmesan, Edam, Feta, cream, Mozzarella, Brie), milk (skimmed, powdered, flavoured, evaporated, condensed), cream (sour) and ice cream. Iodine levels in this survey were full fat milk (0.133 mg/kg), low fat milk (0.159 mg/kg), full fat fruit yoghurt (0.167 mg/kg), full fat ice cream (0.213 mg/kg), butter (0.039 mg/kg), and cheeses (0.153-0.229 mg/kg), (FSANZ, 2004b).

33 The upper level of intake is the highest average daily nutrient intake level likely to pose no adverse health effects to almost all individuals in the general population.

In 1998, the Victorian Dairy Industry Authority carried out a statewide survey to monitor iodine levels in raw tanker milk. Of the 218 samples taken, none exceeded 500 µg/L, which was the Australian standard for iodine in milk until 1998. The majority of samples had iodine concentrations of 100 – 199 µg/L. The combined results for iodine levels for Victoria are shown in Table 17 (VDIA, 1999).

Table 17: Iodine content of raw milk tanker samples from Victoria (1st July 1998 – 30th June 1999) (VDIA, 1999)

Iodine levels (µg/L)	Frequency
< 100	103
100 – 199	150
200 – 299	10
300 – 399	0
400 – 499	0
≥500	0

A dietary exposure assessment was conducted as part of the assessment of iodine for Application A470 – Formulated beverages. The results of this assessment are presented in Table 18.

Table 18: Estimated dietary intakes of iodine in Australia, and percent of upper level of intake (UL) (FSANZ, 2005a)

Age group	Mean intake µG/DAY (%UL)	95th percentile intake µg/day (%UL)
2-3 years	106 (55)	206 (105)
4-8 years	109 (30)	217 (60)
9-13 years	130 (20)	276 (40)
14-18 years	142 (15)	338 (35)
≥19 years	116 (10)	276 (25)

The primary foods that contribute to dietary iodine exposure in the Australian population were dairy (67.8%), fruits (10.6%), cereal foods (5.6%), and seafood (5.3%) (FSANZ, 2005b)

Extent of iodine variability in milk

The main external influences on the level of iodine in milk are geographical variations and seasonal diets, in addition to the use of iodophors as sanitising agents of milking equipment and naturally high levels in water in certain parts of Australia.

In the U.K. seasonal variations can occur when iodine rich stock feed is given to dairy cattle during winter to compensate for reduced access to grazing pastures (FSA 2000). For example, winter milk was found to contain 210 microgram/kg while summer milk contained 90 microgram/kg. This seasonal variation may reflect the greater use of compound feeding stuffs during the winter months (FSA 2000). Iodine may be naturally present in the ingredients used in animal stockfeed or may be added via supplements. Iodine is included in compound stockfeed to protect animal health and incidentally it provides a source of iodine in human diets.

Iodophors have been used widely in the past as sanitising agents for teats and milking equipment, and may contribute significantly to the iodine content of milk. Australia, New Zealand and many other overseas countries have now reduced the use of iodophors, resulting in a lowering of milk iodine content.

However, some nations (e.g. United Kingdom) still maintain the practice of (high) iodophors use, which contributes to the global variability in milk iodine content.

Table 19 demonstrates some of the variability that can exist in milk iodine concentration on a global scale; only a selection of countries are provided due to the lack of information on international milk iodine concentrations. New Zealand data have been obtained from the 2003/4 Total Diet Survey (NZTDS) results, while information on Australia is available from Tasmania, where periodic monitoring is undertaken by two major milk processors, for the Northern Victorian District of the Goulburn Valley, and from the results of the 22nd Australian Total Diet Survey (FSANZ, 2004b).

Table 19: Annual Iodine Concentrations in Milk (μg/L)

	Minimum	Maximum	Mean
Australia (Tasmania) (Personal communications: Seal J, 2004)	110	440	265
Australia (Victoria) (Nestlé submission in response to IAR A528)	31	361	155
Australia (FSANZ, 2004; 22nd ATDS, 2004)	90	210	133
New Zealand (Vannoort, 2004)	41	235	86
United Kingdom (United Kingdom Food Standards Agency 2000)	184	426	315
Germany (Preiss 1997)	<100	150	115
International Mean (FAO/WHO 2002)	34	54	46

5.3.3 Selenium

Selenium in food is predominantly in the form of organo-selenium compounds; selenocysteine is usually the primary form obtained from animal based foods. The selenium content of food varies depending on the selenium content of the soil. Many plants such as *Astragalus* and *Stanleya* accumulate high levels of selenium, although most of these selenium-accumulating plants are unpalatable (Panter and James, 1990).

Due to deficiencies in Australian soils supplementation with selenium in agricultural or veterinary chemicals for example the oral drenches such as Cydectin Se, are registered for use in sheep and cattle (Pointon, 2004). While dairy products are considered to have low concentrations of selenium, the relatively large amount of milk consumed (as compared to solid foods) results in milk being a source of selenium intake.

Hazard Identification and Characterization

The safety of selenium was most recently assessed by FSANZ in Application A470 – Formulated Beverages (FSANZ, 2005a). Furthermore, NHMRC has established upper levels of intake for selenium in specific age groups (NHMRC, 2004).

In food animals, a study found that up to 18% of the selenium in an oral diet might be excreted in milk (Maus, 1980).

There is limited data about toxicity in humans but the most common outcomes are hair and nail brittleness and loss as well as gastrointestinal disturbance, skin rash, fatigue, irritability and nervous system abnormalities. Studies from China give a NOEL for adults of 800μg/day, which was consistent with one US study.

An Uncertainty factor of 2 is applied (US Institute of Medicine, 2000) to protect sensitive individuals because of gaps in data and incomplete knowledge, bearing in mind that the toxic effect of selenium is not severe but may not be reversible. The Upper Level of Intake (UL) is therefore set at 400µg/day for adults including pregnancy and lactation as there is no data to suggest increased susceptibility.

The UL for young infants was based on the studies showing that human milk concentrations of 60µg/L were not associated with adverse effects. This gives a NOEL of 47µg/day (7µg/kg body weight). An Uncertainty Factor of 1 is applied, as there is no evidence that maternal intakes associated with human milk in this range causes infant or maternal toxicity. As there is no evidence of increased toxicity in older children and adolescents, the ULs for these groups was estimated on a body weight basis from the younger infant data using the level of 7µg/kg body weight (NHMRC, 2004).

In summary the ULs for the various age groups are:

1-3 years	**90 µg/day**
4-8 years	**150 µg/day**
9-13 years	**280 µg/day**
14-18 years	**400 µg/day**
adults	**400 µg/day**

Dietary exposure

Where plants are deficient in selenium, milk selenium levels are reported at 5-30 ng/ml, moderate levels in plants are associated with milk levels of 30-66 ng/ml; concentrations of up to 1300 ng/ml were found in milk from cows living in seleniferous areas of South Dakota (Jensen, 1995). Selenium is present in cow's milk in concentrations in direct proportion to selenium intake (Panter and James 1990).

Most of the selenium in cow and goat's milk is found in the skim milk with only 2-10% being in the fat fraction. About 30% of the total selenium in goat milk is found in the whey, compared with over 70% in bovine milk, of which 80% is found with β-lactoglobulin (Jensen, 1995).

A dietary exposure assessment was conducted as part of the assessment of selenium for Application A470 – Formulated Beverages (FSANZ, 2005a). Estimated dietary exposure to selenium, based on the 1995 Australian National Nutrition Survey (whole population aged 2 years and over) resulted in a mean dietary exposure of 15-35% of the UL and dietary exposure at the 95th percentile of 40-80% (ANZFA, 1995; FSANZ, 2005a)

The 20th Australian Total Diet Survey indicated the following primary foods that contribute to dietary selenium exposure in the Australian population, aged 2 years and older: chicken meat (19%), marine fish (11%), pork (10%) eggs (10%), wheat flour (5%) and milk and dairy (5%).

The 20th Australian Total Diet Survey data have indicated selenium levels in full fat milk (0.011 mg/kg), low fat milk (0.013 mg/kg), full fat fruit yoghurt (0.017 mg/kg), various cheeses (0.057-0.107 mg/kg), and full fat vanilla ice cream (0.0013 mg/kg), chocolate milk (0.066mg/kg) (FSANZ, 2003).

5.3.4 Zinc

The zinc content of milk is not constant but influenced by a number of factors such as stage of lactation, nutritional status of the animal, and environmental contamination through drinking water or if galvanised containers were used for storage.

Hazard identification and characterisation

The safety of zinc was most recently assessed by FSANZ in Application A470 – Formulated Beverages (FSANZ, 2005a). Furthermore, NHMRC has established draft upper levels of intake for zinc specific age groups (NHMRC, 2004).

Studies of chronic and sub-chronic toxicity of zinc are well documented. Prolonged intakes of zinc supplements ranging from 50 mg/day up to 300 mg/day have been associated with a range of biochemical and physiological changes.

These changes include hypocupraemia, leucopaenia, neutropaenia, sideroblastic anaemia, decreased concentrations of plasma copper and decreased activity of the copper containing enzymes, superoxide dismutase and caeruloplasmin, altered lipoprotein metabolism and impaired immune function. Many of these biochemical and physiological changes are similar to those observed during copper deficiency. Nevertheless, there are problems with hazard identification in that these changes are not specific to copper deficiency and the clinical relevance of some is unknown.

Systemic evidence of copper deficiency in humans may be observed at doses of 150 mg/day in humans, but doses as low as 50 mg/day may indicate a threshold effects, as observed by changes in biochemical markers of copper deficiency (ANZFA, 1999h).

A LOEL of 60 mg/day is set, based on a study where copper status was evaluated after supplemental intake of 50 mg/day as zinc gluconate in 18 healthy female subjects (aged 25 to 40 years) for 10 weeks. Endothelial superoxide dismutase activity was significantly lower than pre-treatment values. An uncertainty factor of 1.5 was used to account for inter-individual variability in sensitivity and for extrapolation from a LOEL to a NOEL. Because reduced copper status is rare in humans, a higher uncertainty factor was not justified.

For children a study in infants fed 5.8 mg/L of zinc for six months did not reveal effects of zinc on serum copper or cholesterol concentrations or other adverse effects. This would result in an intake of 4.5 mg/day for infants 0 through 6 months of age. This NOEL was divided by an uncertainty factor of 1.0 to obtain an upper limit of 4 mg/day (rounded down) for infants 0 through 6 months. No adverse effects of zinc in children and adolescents could be found. Due to a dearth of information, the UL for young infants was adjusted for older infants, children and adolescents on the basis of relative body weight. Values have been rounded down (FSANZ, 2005a).

In summary the ULs for the various age groups are:

1-3 years	**7 mg/day**
4-8 years	**12 mg/day**
9-13 years	**23 mg/day**
14-18 years	**34 mg/day**
Adults	**40 mg/day**

Dietary intake
A dietary exposure assessment was conducted as part of the assessment of zinc for Application A470 – Formulated beverages (FSANZ, 2005a). The results of this assessment are presented in Table 24.

Estimated intakes were adjusted based on second day intake data from the NNSs. Dietary modelling has been conducted only for food intake. Intake through other sources (i.e. supplements and drinking water) was not included in the modelling.

Table 24: Estimated dietary intakes of zinc, and percent of UL (FSANZ, 2005a)

Age group	Mean intake mg/day (%UL)	95th percentile intake mg/day (%UL)
2-3 years	7.5 (110)	10.4 (150)
4-8 years	8.2 (70)	11.7 (100)
9-13 years	10.9 (45)	16.5 (70)
14-18 years	12.7 (35)	21.3 (65)
≥19 years	11.9 (30)	18.4(45)

The primary foods that contribute to dietary zinc exposure in the Australian population, aged 19 years and older, were meat, poultry, and game products and dishes (35%) cereals and cereal products (14%), milk products (13%), cereal based products and dishes (10%), and vegetable products and dishes (10%) (ABS, 1995).

In Australia the following concentrations of zinc in various dairy products are reported: milk, cow 3 mg/L; milk, goat 4 mg/L; milk powder 30-39 mg/kg; various cheeses (cream, Feta, Mozzarella, Parmesan, blue vein, Edam, Gouda, Neufchatel, Cheddar, Swiss, Ricotta, Cottage) 2-65 mg/kg; yoghurt 4-7 mg/kg; ice cream 5-7 mg/kg; cream 0-4 mg/kg (FSANZ, unpublished data).

Overseas, concentrations of zinc in milk from various dairy animals are reported as cow 4 mg/L, buffalo 0.2-0.3 mg/L, goat 3-6 mg/L and for sheep 1-2 mg/L (Jensen, 1995).

According to the International Dairy Federation (International Dairy Federation, 1992) the zinc concentration in milk will hardly raise if zinc is added to the diet. Zinc concentrations of 4.2, 6.7 and 8.0 mg/l in milk are reported at concentrations of 44, 372 and 692 mg zinc/kg dw in the feed. Further raising of the zinc concentration in the food up to 1279 mg/kg dw did not lead to a further raise in the concentration in milk (Van Hooft, 1995).

5.3.5 Risk characterisation of micronutrients
An evaluation of iodine, selenium and zinc was performed to establish whether there are potential public health and safety risks with high level consumption of these micronutrients present in dairy products.

In addition to having an essential role, there is a potential for over consumption of some micronutrients, thereby resulting in a public health and safety risk.

5.3.5.1 Iodine

Most high consumer population groups, except for the 2-3 year olds (105% UL) are estimated to have intakes of iodine below the UL (FSANZ, 2005a). Due to the use of 24-hour dietary survey data, which tends to over-estimate habitual food consumption amounts for high consumers, it is likely that the 95th percentile dietary intake is an over-estimate. Overall, the potential to exceed the UL, even for 2-3 year olds, is considered to be low.

Comparison of estimated intakes with the UL is not appropriate when considering the health risk for individuals with thyroid disorders or a long history of iodine deficiency, as typically they respond adversely to levels of intake that fall below the UL and, in some cases, at levels that approximate normal dietary intakes. Such individuals may therefore potentially be at risk even from natural fluctuations in the iodine levels in foods.

In conclusion, dairy products contribute significantly to the intake of iodine. The current levels of iodine in dairy products do not raise public health and safety concerns.

5.3.5.2 Selenium

While selenium derived from dairy products makes a contribution to selenium intake, the level of exposure was significantly below the UL. The current levels of selenium in dairy products do not raise public health and safety concerns.

5.3.5.3 Zinc

Dairy products contribute approximately 13% of the overall zinc intake in the population. Recent modelling has indicated that children in Australia, aged 2-8 years, may be exceeding the UL for zinc, both at the mean and at the 95th percentile dietary intake. For these calculations, intake from other sources, i.e. contamination from galvanised containers and intake from supplements have not been included. For adults estimated zinc intakes are below the UL (FSANZ, 2005a).

Recent modelling has indicated that children in Australia, aged 2-8 years, may be exceeding the UL for zinc, both at the mean and at the 95th percentile dietary intake. For adults estimated zinc intakes are below the UL (FSANZ, 2005a).

The UL for children was based on levels in infants that did not reveal effects of zinc on serum copper concentrations or other adverse effects. Due to a dearth of information, the UL for young infants was adjusted for older infants, children and adolescents on the basis of relative body weight.

In conclusion, while there may be a potential risk of exceeding the UL for some sub-population groups, milk is not a major contributor to the zinc intake. Therefore, it is concluded that there are no public health and safety concerns with the current levels of zinc in dairy products.

Overall conclusion

There are no public health and safety concerns with the current levels of iodine, selenium and zinc in dairy products. Milk is a source of iodine, selenium and zinc, and therefore, has a role in preventing deficiencies for these essential micronutrients in the community.

Milk and milk products contribute significantly to the intake of iodine, and in a lesser extent to the intake of selenium and zinc.

5.4 Organic Compounds

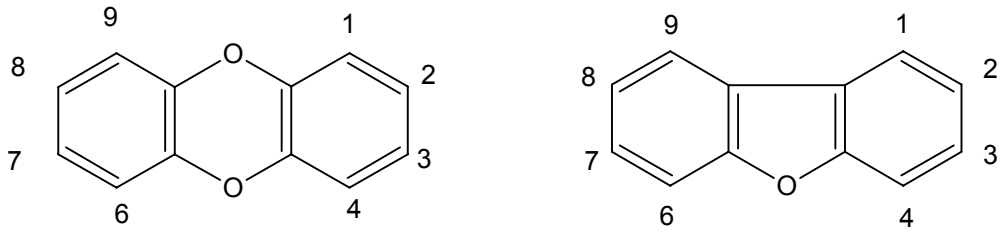
5.4.1 Dioxins and dioxin-like polychlorinated biphenyls

FSANZ carried out a dietary exposure assessment and risk characterisation of dioxins and dioxin-like Polychlorinated biphenyls (PCBs) in food as part of the National Dioxins Program (NDP) in 2004 (FSANZ, 2004a); (NDP, 2004). The Food Standards Code does not contain an ML for dioxins.

The term 'dioxins' is used to describe a group of environmentally persistent halogenated aromatic hydrocarbon chemicals that include polychlorinated dibenzodioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), polybrominated dibenzodioxins (PBDDs), polybrominated dibenzofurans (PBDFs). The chlorinated compounds predominate and are the focus of this review.

PCDDs, PBDDs, PBDFs and PCDFs are not manufactured intentionally but are by-products of combustion. They are formed naturally by volcanoes and forest fires, as well as by industrial processes such as waste incineration and the synthesis of certain chemicals.

The PCDDs and PCDFs are chlorinated tricyclic aromatic hydrocarbons, made up of two benzene rings joined by either two oxygen atoms at adjacent carbons on each of the benzene rings (PCDDs) or by one oxygen atom and one-carbon-carbon bond (PCDFs); their basic structure is given in Figure 3 (NDP, 2004).



Dibenzo-*p*-dioxin Dibenzofuran

Figure 3: Structures of dibenzo-*p*-dioxin and dibenzofuran

Both groups of chemicals may have up to eight chlorine atoms attached at carbon atoms 1 to 4 and 6 to 9. Each individual compound resulting from this is referred to as a congener. The number and position of chlorine atoms around the aromatic nuclei distinguish each specific congener. In total, there are 75 possible PCDD congeners and 135 possible PCDF congeners. The most widely studied of the PCDDs and PCDFs is 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). TCCD is often generically referred to as 'dioxin', and represents the reference compound for this class of chemicals (NDP, 2004).

Certain polychlorinated biphenyls (PCBs), the non-*ortho* and mono-*ortho* congeners, can adopt a coplanar conformation that is structurally similar to the PCDD/PCDFs and appear to elicit dioxin-like responses through similar modes of action.

PCDDs, PCDFs and dioxin-like PCBs are commonly referred to as 'dioxin-like compounds'.

In general, dioxin-like compounds have very low water solubility, high octanol-water partition coefficients, low vapour pressure and absorb strongly to particles and surfaces and are resistant to chemical degradation under normal environmental conditions. Thus, they are persistent in the environment and their high fat solubility results in their bioconcentration into biota and biomagnification up the food chain (NDP, 2004). High fat dairy produce is therefore amenable to higher dioxin levels and detection in milk therefore acts as a signal for potential subsequent problems due to environmental contamination.

Toxic equivalency factors

When found in the environment, biological tissue and industrial sources, dioxins are usually present as complex mixtures; this complicates hazard and risk assessment because different congeners vary significantly in their toxicity. However, the potency of different dioxins can be ranked relative to TCDD, the most toxic member of the dioxin class. These rankings are known as toxic equivalency factors (TEFs). To be included in the TEF scheme, a compound must be structurally related to PCDDs and PCDFs, bind to cellular aryl hydrocarbon (*Ah*) receptor, elicit *Ah* receptor-mediated biochemical and toxic responses, must be persistent, and accumulate in the food chain.

Several schemes for assigning TEFs to PCDD/Fs and PCBs have been used previously. However, the most recent review of TEFs was that of the World Health Organisation (WHO) in 1998 (van den Berg *et al*., 1998). Under the WHO TEF scheme, TCDD is assigned a TEF of 1.0, and other PCDDs, PCDFs and PCBs have TEF values ranging from 1.0 down to 0.00001. To estimate the toxic potency of a given dioxin mixture, the mass concentration of each individual component is multiplied by the respective TEF, and the products are summed to represent the TCDD toxic equivalence (TEQ) of the mixture.

Intake of dioxins for the purpose of this Report will be expressed in units of TEQs applying the 1998 WHO TEFs (NDP, 2004).

Hazard Identification and Characterisation

The most widely studied of all the dioxin-like compounds is TCDD. It has been shown to affect a wide range of organ systems in many animal species and can induce a wide range of adverse biological responses. The binding of TCDD to the so-called aryl hydrocarbon (*Ah*) receptor in cells appears to be the first step in a series of events that manifest themselves in biological responses, including changes at the biochemical, cellular and tissue level.

In humans, the most widely recognised and consistently observed effect following high dose exposure to TCDD is chloracne. The condition can disappear after termination of exposure or can persist for many years. Other effects on the skin include hyperpigmentation and hirsutism. TCDD can cause long-term alteration in glucose metabolism and there is some evidence of a weak correlation between incidence of diabetes and occupational or accidental exposure to dioxins; however, background exposure to dioxins is not a significant risk factor for diabetes. TCDD exposure has been suggested to cause slight changes in thyroid function, but clinical illness associated with immune system disorders does not appear to have been associated with TCDD in any cohort studied. There is suggestive evidence of toxicity to the cardiovascular system. Overall, epidemiology studies on populations exposed occupationally or environmentally to TCDD have not demonstrated any significantly increased all-cause or non-cancer mortality (NDP, 2004).

Experimental studies demonstrate that TCDD is carcinogenic in all species and strains of laboratory animals tested. It has been characterised as a multi-site carcinogen. Epidemiological evidence from the most highly-exposed occupational cohorts studied produces the strongest evidence in humans of an increased cancer risk from exposure to dioxins, when the data is considered for all cancers combined. There is weaker evidence of an increased cancer risk when cancers from particular sites is considered (NDP, 2004). IARC concluded that TCDD is carcinogenic to humans (IARC, 1997).

Australia established a Tolerable Monthly Intake (TMI) for dioxins of 70 pg TEQ/kg bw/month from all sources combined. This tolerable intake is equal to that set by JECFA (JECFA 2002) and includes polychlorinated dioxins, polychlorinated furans and dioxin-like PCBs, as specified under the WHO 1998 TEF scheme.

Exposure Evaluation - National Dioxin Program

The collection of milk samples for the National Dioxin Program (NDP) was co-ordinated by Dairy Australia. In this study 19 composite milk samples were analysed (Table 16). As there were isolated instances of exposure shown in the beef cattle results, it is possible that a particular dairy herd could be similarly exposed. However, it is likely that residues in milk would be lower than those detected in beef fat due to continual excretion via the milk. Furthermore, residues in milk consumed would be further reduced by dilution with milk from other herds.

Table 16: Australian data for dioxin and furan residues compared against the EU standard (NDP, 2004)

Species	EU Standard Maximum pg TEQ/g*	Mean** result from NDP study (%)	Number of samples
Beef	3	0.56 (18.6%)	109
Milk	3	0.43 (14.5%)	19
Sheep	3	0.57 (19.1%)	45

* on a fat basis

** mean results are upperbound concentrations expressed as pg TEQ/g. Values in parentheses are expressed as a percentage of the EU standard for that species or commodity.

The EU standard in EU Regulation (EC) No. 2375/2001 only refers to dioxins/furans and dioxin-like PCBs are not currently included. These results indicate that Australian levels for dioxins/furans are < 20% of the EU standard.

A summary of the mean PCDD/F concentrations for dairy products used in the dietary modelling is shown in Table 17. Individual composite sample PCDD/F and PCB results are summarised in a FSANZ Technical Report (FSANZ, 2004a).

Comparison of dioxin concentrations in food across different monitoring programs is difficult since there are differences in food sampled, analytical methodologies and calculation and reporting of TEQs. Generally Australian foods have levels of PCDD/Fs and PCBs that are similar to those reported in New Zealand and lower than those reported from other areas of the world.

For infants aged 9 months the major contributors to PCDD/F exposure were infant formula (containing non dairy fat *i.e.* plant-derived oils) (82%) and milk and dairy products including cheese, ice cream and infant dessert (5%).

For toddlers (2-4 years), the major contributors to PCDD/F exposure were milk and dairy products including cheese and ice cream (55%). Taking the whole population (2+ years) into account, milk and dairy produce contributed 31% of the PCDD/F dietary exposure.

Table 17: Mean levels of PCDD/F in food (FSANZ, 2004a)

	Number of composite samples	PCDD/F	
		Lower bound pg/g FW	Upper bound pg/g FW
Butter	10	0.011	0.20
Milk chocolate	1	0.0029	0.044
Milk, whole	13	0.0010	0.0065

All samples are composites of three or four purchases.
All results are reported in picograms TEQ per gram of food on a fresh weight basis.
Lower Bound – assumes results reported as below the LOR are zero for each congener. The levels of the individual congeners are then summed.

Upper Bound – assumes results reported as below the LOR are at the LOR for each congener. The levels of the individual congeners are then summed.

Risk Characterisation

Dioxins enter the food chain when animals eat contaminated plants or inhale smoke from burning organic matter. The dioxins are then absorbed in the animal fat, increasing in concentration as they migrate up the food chain. The consumption of animal products with high fat content, including dairy products can therefore theoretically increase human exposure to dioxins.

For the general population, over 95% of exposure to dioxin-like compounds is through the diet, with foods of animal origin such as meat, dairy products and fish being the main sources (NDP, 2004). For infants aged 9 months, the mean estimated exposure to dioxins was in the range of 11.8 and 60.8 pg TEQ/kg bw/month and for all Australians aged 2 years or older, the mean upper bound monthly intake of dioxins is 15.6 pg TEQ/kg bw/month. The relatively high exposure for infants is due to their high food consumption relative to body weight. Overall, these levels are significantly below the TMI of 70 pg TEQ/kg bw/month.

Dairy products are a relatively high contributor to the total dietary exposure of the Australian population to dioxins and dioxin-like compounds, however the levels overall are well within the JECFA PTMI.

Both Australian and New Zealand milk and butter have relatively low PCDD/F concentrations in milk and butter compared to other areas of the world (Table 18); bearing in mind that there are differences in analytical methodologies and calculation of the reporting of TEQs. The overall dietary exposure to dioxins and dioxin-like PCBs in Australia and New Zealand is well below that of values recorded in the U.K., The Netherlands and Europe (Table 19; FSANZ, 2004a).

Table 18: Comparison of mean PCDD/F concentrations in selected foods from different areas of the world (FSANZ, 2004a)

	Mean PCDD/F (pg TEQ/g lipid)						
	Australia	New Zealand[1,2]	UK	Netherlands[3]	Europe[1]	Asia[1,4]	North America[1]
	(NDP, 2004)	(MFE 1998)	(FSA 2003)	(Freijer et al 2001)	(Codex 2003)	(Codex 2003)	(Codex 2003
Milk	0.04-0.23	0.019-0.16	0.46-0.47	0.57	0.3-2.5	0.30-1.8	0.3-0.9
Butter	0.013-0.23	0-0.095	-	0.68	-	-	-

[1] Results reported in I-TEQs, that are 10-20% lower than WHO-TEQs
[2] Results reported in the range of lower to middle bound.
[3] Results reported as lower bound only.
[4].Reported on a fresh weight basis.

Table 19: An international comparison of mean or range of estimated dietary intakes of dioxins

Country/region	Reference	PCDD/Fs (pg WHO-TEQ/kg bw/month)	PCBs (pg WHO-TEQ/kg bw/month)	Total Dioxins (pg WHO-TEQ/kg bw/month)
Australia[1]	FSANZ, 2004a	0.9-10.2	2.8-5.4	3.7-15.6
New Zealand[2]	(Ministry for the Environment, 1998; Ministry for the Environment, 2001)	6.6	4.5	11.1
UK[3,4]	(Food Standards Agency, 2003)	9	9-12	15-21
The Netherlands[4,5]	(Freijer *et al*., 2001)	20.7	18.6	39
Europe[6,7]	(European Commission, 2000)	12-45	24-45	36-90

[1]-Range is lower bound to upper bound for all persons 2+years of age
[2]-Medium bound estimate for adult males
[3]-Range is lower bound to upper bound for the population average
[4]-Sum of PCDD/F and PCB (total dioxins) may not equal sum of separate intakes due to rounding
[5]-Lower bound estimate, mean lifelong-averaged (1-70 years) exposure.
[6]-I-TEQs. WHO-TEQs are 10-20% higher than I-TEQs.
7-Average dietary exposure for an adult person.

Conclusion

There are no public health and safety concerns with the current levels of dioxin in dairy products.

5.4.2 Polychlorinated biphenyls

Unlike dioxins, PCBs do not occur naturally in the environment; they are derived from man-made sources which were manufactured for approximately 50 years for use as components of insulating fluids in transformers and other electrical equipment (NDP, 2004).

The safety of polychlorinated biphenyls (PCBs) was last assessed by FSANZ in Proposal 158 – Review of the maximum permitted concentration of non-metals in food (ANZFA, 1999c).

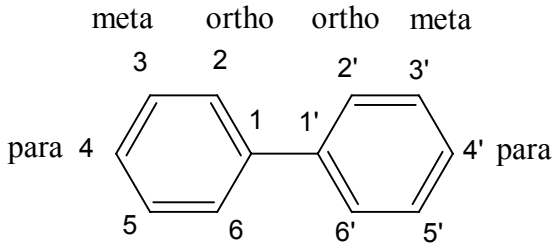


Figure 4: Structure of biphenyl

The PCBs are structurally similar to the PCDDs and PCDFs (dioxins) and include 209 congeners, from the monochloro congener through to the fully chlorinated decachloro congener; the basic aromatic nucleus is shown in Figure 4.

Like the PCDD/PCDFs, the biological effects of PCBs are very dependent on both the degree of chlorination and on the position of the chlorine atom around the aromatic nuclei (i.e. whether they are *ortho*-, *meta*-, or *para*- to the phenyl-phenyl bridge at carbon-1).

Food Regulation

As part of Proposal P158 - Review of the maximum permitted concentrations of non-metals in food – a risk management strategy that included a ML for total polychlorinated biphenyls in milk and milk products was included in Table to clause 3 – Maximum level of non-metal contaminants in food - Standard 1.4.1 – Contaminants and Natural Toxicants (Table 20). Considering the uncertainty surrounding the potential toxicity of PCB's, their persistence within the environment and the necessity to achieve low PCB levels, the ML was set to include total polychlorinated biphenyl concentrations in food i.e. including dioxin-like PCBs.

Table 20: Maximum levels of PCBs in food

Column 1	Column 2 (mg/kg)
Polychlorinated biphenyls, total	
Mammalian fat	0.2
Poultry fat	0.2
Milk and milk products	0.2
Eggs	0.2
Fish	0.5

Hazard Identification and Characterisation

Animal feeding studies, mostly using rodents, have shown that the range and severity of the toxic effects of PCBs is correlated with the PCB congener/mixture used.

The long-term toxic effects of relatively high levels of PCBs include changes in liver enzyme activity and increased liver weights in rats; adverse reproductive effects and neurotoxicity have also been observed in rodents.

The choice of a particular NOEL for human health risk assessment should be identified for the most sensitive effect in the most sensitive species. JECFA (WHO, 2000) has designated non-human primates as the species most sensitive to the toxic effects of PCBs and has assigned a NOEL of 0.04 mg/kg bw/day, based on the general toxicity of Aroclor 1242 in monkeys. However, the limitations of the available data and the toxicological differences in PCB mixtures that were used in animal feeding studies has made it difficult to establish a value for tolerable intake for humans.

Dietary Exposure

For toddlers (2-4 years), the major contributors to PCB exposure were fish (49%) and milk and dairy products (30%). Taking the whole population (2+ years) into account, milk and dairy produce contributed 11% of the PCB dietary exposure (FSANZ, 2004a). No data is available for dietary exposure to PCBs in non-breast-fed infants. Mean levels of PCBs in foods are shown in Table 21.

Table 21: Mean levels of PCBs in food (FSANZ, 2004a)

		PCB	
	Number of composite samples	Lower bound pg/g FW	Upper bound pg/g FW
Butter	10	0.017	0.070
Milk chocolate	1	0.0048	0.012
Milk, whole	13	0.0013	0.0060

All samples are composites of three or four purchases.
All results are reported in picograms TEQ per gram of food on a fresh weight basis.
Lower Bound – assumes results reported as below the LOR are zero for each congener. The levels of the individual congeners are then summed.
Upper Bound – assumes results reported as below the LOR are at the LOR for each congener. The levels of the individual congeners are then summed.

A small number of PCBs were included in the yearly AMRA survey from 1998 – 2003 (Table 22), and no residues were found in milk or finished products. PCBs were not detected in milk products in the Australian Market Basket Survey, or the New Zealand Total Diet Survey.

In the 2003-4 NRS survey, PCBs were analysed in cattle, sheep, goat, buffalo and camel meat and no residues were detected.

Both Australian and New Zealand milk and butter have relatively low PCB concentrations compared to other areas of the world (Table 23); bearing in mind that there are differences in analytical methodologies and calculation of the reporting of TEQs.

Table 22 Comparison of PCB, Test Results (ADASC, 2005)

Survey Year	PCBs	
	No. tested	No. >ML
1998/1999	327	0
1999/2000	189	0
2000/2001	204	0
2001/2002	112	0
2002/2003	109	0
2003/2004	77	0

Risk Characterisation

Toxicological evaluation of PCBs is complicated by many factors, the first of which is the paucity of data concerning human exposure to, and the effects of, PCBs. Much of the animal toxicity data are based on testing mixtures that contain many PCB congeners with varying degrees of chlorination and different stereochemical structures. Differences in toxicity between PCB congeners may also be associated with specific metabolites and/or their specific intermediates.

Oral exposure to PCBs is associated with adverse effects in animals; the most consistent and pronounced is the occurrence of liver tumours in rodents. However, the available human data (mainly from accidental exposures) is equivocal in respect of an association between PCBs and increased cancer mortality.

In summary, a range of surveys has indicated that PCBs are either not detected or detected at very low levels in the Australian and New Zealand food supply. The low level of dietary exposure is well below the reference health standard. It is concluded that there are no public health and safety concerns associated with residues of PCBs in dairy products.

Table 23: Comparison of mean PCB concentrations in selected foods from different areas of the world (FSANZ, 2004a)

	Mean PCBs (pg TEQ/g lipid)						
	Australia	New Zealand[1,2]	UK	Netherlands[3]	Europe[1]	Asia[1,4]	North America[1]
	(NDP, 2004)	(MFE 1998)	(FSA 2003)	(Freijer et al 2001)	(Codex 2003)	(Codex 2003)	(Codex 2003)
Milk	0.04-0.11	0.027-0.15	0.34-0.43	0.69	0.2-1.8	-	0.5
Butter	0.021-0.086	0.15-0.15	-	0.96	-	-	-

[1] Results reported in I-TEQs, that are 10-20% lower than WHO-TEQs
[2] Results reported in the range of lower to middle bound.
[3] Results reported as lower bound only.
[4] Reported on a fresh weight basis.

5.4.3 Organochlorines

Organochlorine pesticides are generally condensed organic compounds in which chlorine averages 60% of the molecular weight. Persistent organochlorines such as DDT, dieldrin, heptachlor and hexachlorobenzene (HCB) have not been available for use in Australia since the 1970s. However, they are still present in soils where they were used for spot and broad acre pest control. Although these compounds have been de-registered for use, full risk assessments have been carried out and extraneous residue limits (ERLs) set by FSANZ for a range of food commodities that have the potential to be exposed to organochlorines during their production, including milk.

Grazing livestock can ingest soil or crops contaminated with environmentally persistent compounds such as DDT and dieldrin and as such, these organochlorines are considered environmental contaminants. These compounds have been de-registered in Australia for many years as agricultural pesticides, but due to their persistent nature in the environment, particularly in soil, low concentrations may be identified from time to time. Extraneous residue limits (ERLs) are established to account for residue due to previous use.

Overall conclusion

Seven years of AMRA survey data have indicated that there are no environmental residues of organochlorines or PCBs in milk (500 – 1050 analyses carried out) *i.e.* there is 100% compliance with respective MRLs. Furthermore, no residues of heavy metals were found in milk over this period (112 analyses carried out).

It can therefore be concluded that dietary exposure to environmental contaminants from dairy products does not raise public health and safety concerns.

5.5 Plant, fungal and bacterial toxins

The susceptibility of stockfeed to contamination by plant, fungal and bacterial toxins will vary according to geographic location. Critical controls to ensure that stockfeed is free from toxins combines on-farm controls for pasture management (GAP), in addition to vendor declarations from suppliers of supplementary feed.

5.5.1 Classification

Mycotoxins and bacterial toxins are secondary metabolites derived from fungi or pathogenic bacteria and may be natural contaminants of food and stockfeed. There are approximately 6000 known mycotoxins, but few of these have complete toxicological profiles. There are dual concerns with natural contaminants: they may cause detrimental effects on animal health and subsequent production losses and also, some toxins can pass the blood-milk barrier and be present in low concentrations in the milk.

Some of the more common naturally occurring toxins, their sources and an indication of whether the toxin is carried over into milk and milk products, are listed in Table 24. This report has focussed mainly on those natural toxins, which are carried over into milk and are potentially of concern to human health.

Table 24: Naturally occurring toxins of plant, fungal and bacterial origin

Type	Toxin	Pathogen	Source/Host	Carry-over	Ref.
Endogenous plant toxin	Pyrrolizidine alkaloids	[a]N/A	Forage plants and weeds (e.g. comfrey, Patterson's curse, heliotrope)	**Yes**	(Cheeke, 1995); (FSANZ, 2001c)
″	Indole alkaloids (and hordenine)	N/A	*Phalaris spp.* (e.g. canarygrass)	No	(Cheeke 1995)
″	Oxalates	N/A	Tropical grasses (e.g. bufflegrass, pangolagrass, settaria, kikuyugrass)	No	(Cheeke 1995)
″	Cyanide	N/A	Tropical forage grasses (e.g. sorghum)	No	(Cheeke 1995)
″	Photosensitising agents e.g. Steroidal saponins, hypericin	N/A	St. Johns Wort; Buckwheat and other pasture grasses	No	(Cheeke 1995)
″	Saponins, oxalates	N/A	Kikuyu grass	No	(Cheeke 1995)
″	Dicoumarol	N/A	Sweet vernal grass, sweet clover	No	(Dwyer *et al*., 2003)
″	Quinolizidine alkaloids	N/A	Lupins	No	(FSANZ, 2001a)
Bacterial toxin	Corynetoxin	*Rathayibacter toxicus*	synergy of bacterium-nematode-grass (e.g. annual ryegrass)	**Yes (limited evidence)**	(Cheeke 1995); (Edgar, 1994)
Mycotoxin	Aflatoxin	*Aspergillus sp.*	Forage and stored grains e.g. corn, sorghum, peanuts, cottonseed and cottonseed meal	**Yes**	(ANZFA, 1999a)
″	Ochratoxin	*Aspergillus sp. and Penicillium sp.*	Forage and stored grains	**Yes**	(JECFA, 2001c)
″	Fumonisin	*Fusarium sp.*	Forage and stored grains, particularly corn and sorghum.	**Yes**	(Cheeke 1995);(JECFA, 2001b)
″	Trichothecenes	*Fusarium sp*	Forage and stored grains, particularly wheat and corn	**Yes**	(Cheeke 1995)
″	Zearalenone	*Fusarium sp.*	Forage and stored grains	**Yes**	(Cheeke 1995); (EFSA 2004b)
″	Cyclopiazonic acid	*Penicillium spp., Aspergillus spp.*	Cereal grains	**Yes**	(EMAN 2005); (Dorner *et al*., 1994); (Finoli *et al*., 1999)
″	Phomopsins	*Phomopsis leptostromiformis*	Lupin	No	(FSANZ, 2001b)
″	Indole-diterpene neurotoxins	*E.g. Acremonium lolii*	Perennial ryegrass pasture	No	(Cheeke 1995)

Type	Toxin	Pathogen	Source/Host	Carry-over	Ref.
″	Sporidesmin	*Pithomyces chartarum*	None - direct infection from fungal spores	No	(Cheeke 1995)
″	Ergot alkaloids	*Claviceps purpurea*	Grain, grass	No	(EMAN 2005)
	Patulin	*Penicillium spp., Aspergillus spp., Byssochlamys spp.*	Fruit, vegetables, cereal grains and silage.	No	(EMAN 2005)
	Citrinin	*Penicillium spp., Aspergillus spp.*	Cereal grains	No	(EMAN 2005)
″	Moniliformin	*Fusarium spp.*	Cereal grains	No	(EMAN 2005)
″	Sterigmatocystin	*Aspergillus sp.*	Cereal grains	No	(EMAN 2005)
″	Paxilline and *N*-formylloline	*Neotyphodium (endophytic)*	*Echinopogon* spp – indigenous Australasian grass	?	(Miles *et al*., 1998)
Other mycotoxins [b] – *Aspergillus clavatus* and *Aspergillus clavatus* toxins, Citreoviridin; lesser-known *Fusarium* toxins e.g. beauvericin, enniatin, fusaproliferin; Griseofulvin, Nitropropionic acid; Kojic acid; Penicillic acid; *Penicillium roquefortii* toxin; Viomellein; Vioxanthin; Xanthomegnin; Waleminols. gliotoxins, mycophenolic acid, PR-toxin, penitrem A, roquefortines A, B and C, sterigmatocystin and cyclopiazonic acid [C]					(EMAN 2005)

[a] not applicable

[b] less studied mycotoxins found in animal feed and known to effect cattle health. Some toxins however can co-occur with other toxins, for example viomellein has been found in conjunction with ochratoxin A, and could possibly also be carried over into milk.

[c] these toxins have all been found to be associated with cheese spoilage. Although potential hazards associated with these toxins have been cited, in many cases lack of data on their occurrence in foods precludes a risk evaluation.

Plant Toxins

A literature review (as summarised by (Colegate *et al*., 1998)) of the transfer of plant-derived toxins to the milk of lactating grazing animals (Panter and James 1990) has identified the following important issues:

- Contaminated milk, sufficient to cause overt toxicoses in suckling young or humans, can be obtained from an asymptomatic animal.
- Physico-chemical properties of the toxins may lead to favoured distribution and concentration in milk.
- The complexity of milk (emulsified fats in an aqueous solution of protein and minerals) makes it a suitable sink for virtually any toxin that is bound to plasma proteins, freely circulating in the plasma or dissolved in blood lipids.
- Chronic, low level, repetitive exposure of animals to toxins may lead to accumulation in the milk, and may result in a chronic, low level, repetitive exposure of humans to the toxins.
- Young animals and young children may be more at risk to milk-borne, plant-associated toxins since they may experience greater exposure and may not be able to detoxify or eliminate the toxins as efficiently as adults.

- Some toxins are preferentially eliminated via the mammary gland and may be bound to milk protein or occur in the aqueous phase or milk fat.
- Modern methods of pooling and processing milk will dilute toxin concentrations but increased risk exists when the milk comes from a few animals such as on a family farm.
- Chronic damage to organs, such as the kidney or liver, as a result of ingestion of toxic plants may affect the ability of the lactating animal to detoxify the xenobiotics and thereby increase transfer via the milk.

Diseases resulting from the consumption of mycotoxins are called mycotoxicoses. In dairy cattle, mycotoxicoses may be expressed through reduced milk production, poor performance among fresh cows and increased incidence of disease. There usually is intermittent diarrhoea and, frequently, reduced or erratic feed intake. Symptoms may be wide-ranging and not specific. They might include: reduced feed intake or feed refusal; an undernourished appearance; rough hair coat; subnormal production; increased abortions or embryonic mortalities; silent heats or irregular oestrus cycles; expression of oestrus in pregnant cows; and decreased conception rates. Some of the general toxicity effects of mycotoxins are summarised in Table 25.

Table 25: Effect classification of mycotoxins found in Australia

Main Effect	Fungal source	Toxins
Hepatotoxicity	*Penicillium* spp.	Rubratoxins
		Luteoskyrin
		Cyclochlorotine
	Diaporte spp.	Phomopsins
	Aspergillus spp.	Aflatoxins
	Pithomyces spp.	Sporidesmin
Nephrotoxicity	*Penicillium* spp	Ochratoxins
		Citrinin
Neurotoxicity	*Penicillium* spp	Penitrems
		Lolitrems
		Patulin
		Citreoviridin
	Acremonium spp.	Ergopeptines
		Lolitrems
	Claviceps spp.	Ergopeptines
Oestrogenic effects	*Fusarium* spp.	Zearalenols
		Zearalenone
Cytotoxicity	*Fusarium* spp.	Trichothecenes
		Nivalenols (eg. DON, Vomitoxin)
		T-2 toxin
		HT2 toxin
Multiple effects	*Penicillium* spp; Aspergillus spp.	Cyclopiazonic acid

5.5.2 Aflatoxins

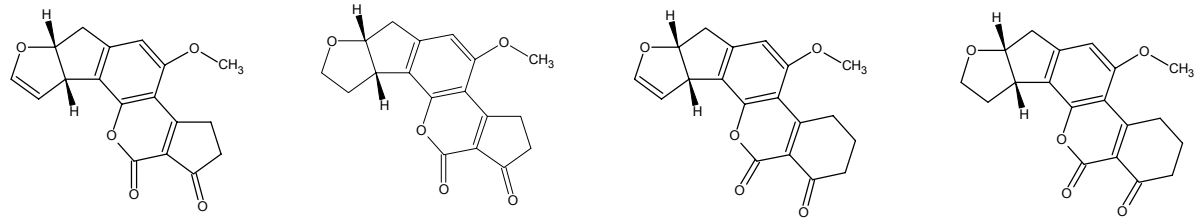
The safety of aflatoxins was last assessed by FSANZ in Proposal 158 – Review of the maximum permitted concentration of non-metals in food (ANZFA, 1999a; ANZFA, 1999b). Aflatoxins are a group of naturally occurring toxic secondary metabolites produced primarily by two species of ubiquitous Aspergillus fungi: *A. parasiticus* and *A. flavus*. These fungi are present in soil and decaying plant material, cause heating and the decay of stored grain, and may invade corn in the field.

Crops and feed ingredients most susceptible to fungi and aflatoxins development include corn, peanuts, peanut meal, cottonseed and cottonseed meal. The use of peanut meal, corn or sorghum in dairy rations are regarded as particularly susceptible to aflatoxin contamination (Dr J.Pitt, personal communication.). Conditions favouring aflatoxin development include drought stressed, insect-damaged feed stored at high temperatures (25 – 32^{o}C) and high relative humidity.

Among the naturally occurring aflatoxins (B_1, B_2, G_1 and G_2), aflatoxin B_1 is the most important compound with respect to both, prevalence and toxicity for humans and animals (EFSA, 2004a; EFSA, 2004b). Aflatoxin dietary intake in humans mainly arises from contamination of maize and groundnuts and their products (JECFA, 1998a). The chemical structures of aflatoxins B_1, B_2, G_1 and G_2 are given in Figure 5.

Aflatoxins M1, M2 and M4 are commonly known as milk aflatoxins, and may be found in cattle, sheep or goat milk after the animal has ingested feed containing aflatoxins B1, B2 or B4 respectively (JECFA, 1998a). The milk aflatoxins are hydroxy-metabolites of aflatoxins B1, B2 and B4 respectively. Both aflatoxins M2 and M4 occur in milk at much lower concentrations compared to aflatoxins M1, and are thus considered as of less public health significance.

There is a linear relationship between the amount of aflatoxin B_1 ingested daily and the level of aflatoxins M_1 in the milk. Milk aflatoxins retain the toxic properties of the parent compound, but do not have the same potency; about 1.5% of aflatoxin is excreted as the metabolite M1 and the concentration of aflatoxins B_1 in milk is approximately 1/300 of the concentration of aflatoxins B1 in the stockfeed (IPCS, 1998).



Aflatoxin B_1 Aflatoxin B_2 Aflatoxin G_1 Aflatoxin G_2

Figure 5: Chemical structures of aflatoxins B_1, B_2, G_1, and G_2.

Hazard identification and characterisation

Aflatoxins are amongst the most toxic of the known mycotoxins and have been implicated in the deaths of humans and animals that have consumed mouldy food. While the liver is the target organ for aflatoxicosis, aflatoxins are also found in other animal tissues and products, such as meat, milk and eggs.

The aflatoxins are among the most potent mutagenic and carcinogenic substances known. Extensive experimental evidence in test species shows that aflatoxins are capable of inducing liver cancer in most species studied (JECFA, 1998a). However, assessment of the risk of liver cancer in humans has proved to be difficult because of confounding factors influencing tumour formation. Sensitivity to aflatoxins varies from one species to another, and, within the same species, the severity of toxicity depends on dose, duration of intake, age and breed of the animals and their dietary protein content.

The liver is the primary target organ in most species, but tumours of other organs also have been observed in animals treated with aflatoxins.

Aflatoxins are metabolised in humans and test species to an epoxide, which usually is considered to be the ultimate reactive intermediate. The effective dose of aflatoxins B_1 for induction of liver tumours varies widely over a wide range of species when the carcinogen was administered by continuous feeding, generally for the lifetime of the animal. Epidemiological studies indicate that individuals who are carriers of persistent viral infection with hepatitis B virus and who are exposed to aflatoxin in their diets are at increased risk for progression to liver cancer (JECFA, 1998a). Some epidemiological evidence indicates the possibility that humans are at substantially lower risk from aflatoxins than other species. While some studies suggest that intake of aflatoxins poses a detectable risk in the absence of other factors, other studies suggest that it poses risks only in the presence of confounding factors such as hepatitis B infection (JECFA, 1998a).

IARC has concluded that aflatoxins are carcinogenic to humans (Group 1) (IARC, 2002a).

JECFA has concluded that aflatoxins should be treated as carcinogenic food contaminants, the intake of which should be reduced to levels as low as reasonably achievable. However, JECFA did not believe that there was a firm foundation for setting absolute limits for aflatoxins intake by humans at this time (JECFA, 1998a; JECFA, 1998b).

Aflatoxin M1 has toxicological properties comparable to those of aflatoxin B_1, but has a carcinogenic potency of one or two orders below that of aflatoxin B_1 (IPCS, 1998). EFSA has set a Maximum Limit (ML) for aflatoxin M1 in milk at 0.05 μg/ kg, and 0.025 μg/ kg for infant formulae, respectively, aiming to reduce human exposure to the lowest achievable level (EFSA 2004a). Codex, however have set an ML of 0.5 μg/ kg for aflatoxin M1 in whole milk. In Australia, there is no ML for aflatoxins in milk and the ALARA principle applies.

Historical data (1987 – 1992) on the presence of aflatoxin M1 in Australian milk samples (ANZFA, 1999a), was collated by The Australian Mycotoxin Data Centre (AMDC). There were ten positive samples found within 227 samples (4.4% positives), these were mainly found in spray dried milk powder (Table 26). In recent AMRA surveys (2000 – 2004) Aflatoxin M1 was not detected in milk samples (vat and tanker) or in finished products.

Dietary exposure

Analysis of Australian and New Zealand commodities have indicated that problems associated with aflatoxins are almost entirely confined to peanuts and nut products (ANZFA, 1999a; ANZFA, 1999b).

The 20th ATDS reports that there were no detections of aflatoxins (B1, B2, G1 and G2) in foods which may potentially contain these substances (i.e. breads, biscuits, rice, oats, processed wheat bran, breakfast cereals, instant coffee, peanut butter, almonds and milk chocolate.)

Although most of the aflatoxin levels recorded in the international literature as residues in single feed, mixed dairy concentrates and tank milk were low (<0.1 – 16 μg/kg), there have been more recent reports of higher aflatoxins levels in animal feeds originating from Europe, with levels ranging from 25 – 40 μg/kg (Vallone and Dragoni, 2005).

Table 26: Aflatoxin M1 (mg/L) in Australian milk product samples (ANZFA, 1999b)

Reporting date	Product	No. samples	No. +ve	% +ve	Range	Min.	Max.	Ave.
Sept. 1990	Milk powder	10	1	10		0.2	0.2	0.2
June 1992	Milk powder	42	5	12		0.2	0.4	0.3
June 1987	Milk	3	3	100		0.26	0.52	0.39
June 1987	Milk powder	1	1	100	0.2	1.5	1.5	1.5
various	Milk	54	0		0.2 – 0.4			
	Dried milk	73	0		0.26 – 0.52			
	Dried skim milk	10	0		1.5			
	UHT milk	25	0					
	Cheese	8	0					
	Goat's milk	1	0					
	Total	227	10	4.4	0.2 – 1.5	0	1.5	-

The carry-over rate for aflatoxins from contaminated feeds into milk of dairy cows is 1 – 2% on average, however this is considered to be considerably higher in high yielding cows. Changes in the plasma-milk barrier and the consumption of significantly higher amounts of concentrated feeds (exposed to European environmental conditions) are thought to contribute to the higher carry-over rates of aflatoxins of 6% (EFSA 2004a). Estimated concentrations of aflatoxin M1 in milk varies for different animal species. Under worst-case conditions, high yielding sheep, goats and camels could potentially have twice the carry-over of aflatoxin M1 in their milk, as compared to dairy cows and buffalo milk could contain up to four times the amount of aflatoxin M1 as cows milk (EFSA 2004a).

In the AMRA survey, aflatoxin M1 testing commenced in 2002/2003, and continued during 2003/2004. During that period, 143 samples were tested with no residue detections identified. The 2003/2004 AMRA survey included 50 samples from targeted areas as well as 39 random milk samples. There is no routine testing for other mycotoxins in the AMRA survey as it is unlikely that they are present in significant levels, due to effective management practices.

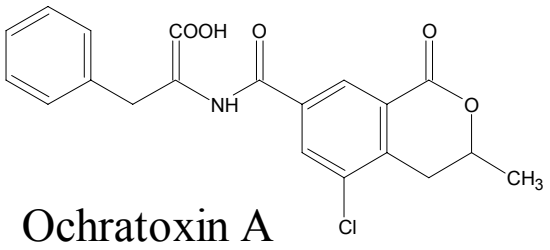
Risk characterisation

Aflatoxins are regarded as human carcinogens, the intake of which should be reduced to levels as low as reasonably achievable. While secondary exposure to aflatoxins through consumption of milk products derived from cattle fed aflatoxin-containing feed can occur, the levels found are very low.

In conclusion, there are no public health and safety concerns with the very low levels of dietary exposure to aflatoxin M1 from dairy products.

5.5.3 Ochratoxin A

Ochratoxins, of which ochratoxin A is the most prevalent, are secondary fungal metabolites of some toxigenic species of *Aspergillus* or *Penicillium*. Ochratoxin A consists of a chlorinated dihydroisocoumarin moiety linked through a 7-carboxyl group by an amide bond to one molecule of L-β-phenylalanine (Bakker and Pieters, 2002). The chemical structure of ochratoxin A is given in Figure 6.



Ochratoxin A

Figure 6: Chemical structure of Ochratoxin A

Hazard Identification and Characterisation

Ochratoxin A is slowly absorbed from the gastrointestinal tract. It is distributed in a number of species via the blood, mainly to the kidneys, with lower concentrations found in liver, muscle and fat. The major metabolite of ochratoxin A in all species examined is ochratoxin α. Ochratoxin α and other minor metabolites that have been identified are all reported to be less toxic than ochratoxin A.

Ochratoxin A have been shown to be nephrotoxic in all mammalian species tested (Bakker and Pieters, 2002). The main target is the renal proximal tubule, where it exerts cytotoxic and carcinogenic effects. Significant sex and species differences in sensitivity to nephrotoxicity were evident, in the order pig>rat>mouse. Carcinogenesis was observed at doses higher than those that caused nephrotoxicity in rodents.

IARC has classified Ochratoxin A into group 2B (possibly carcinogenic to humans – sufficient evidence in animals, and inadequate data in humans) (IARC, 1993c).
JECFA recently reviewed Ochratoxin A and retained the previously established PTWI of 100 ng/kg bw per week pending results of on-going studies on the mechanisms of nephrotoxicity and carcinogenicity. JECFA concluded that the new data raised further questions about the mechanisms by which Ochratoxin A causes nephrotoxicity and renal carcinogenicity and the interdependence of these effects.

In reaching this conclusion, JECFA noted the large safety factor applied to the NOEL for nephrotoxicity in deriving the PTWI, which corresponds to a factor of 1500 applied to the NOEL for carcinogenicity in male rats, the most sensitive species and sex for this end-point (JECFA, 2001c).

Ochratoxin A is also immunotoxic and teratogenic at higher than nephrotoxic doses. Pigs, dogs and poultry are particularly sensitive to the nephrotoxicity and a NOEL has not been established in pigs and dogs. Ruminants are less sensitive due to degradation of ochratoxin A to the less toxic ochratoxin α by the rumen microflora, although sheep have a lower capacity to degrade ochratoxin A than other ruminants (EFSA 1990), (Hohler *et al*., 1999).

Accumulation of ochratoxin A occurs in blood, liver and kidney, and significantly lower residue concentrations have been found in muscle tissue, fat and milk (EFSA 1990).

In the 20th ATDS, ochratoxin A was not detected in any of the following foods: breads, biscuits, rice, oats, processed wheat bran, breakfast cereals, instant coffee, peanut butter, almonds and milk chocolate.

Risk characterisation

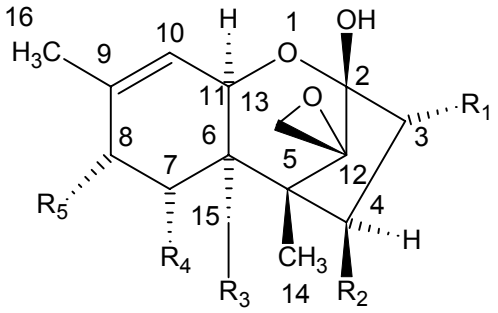
Ochratoxin A has been shown to be nephrotoxic in all mammalian species tested. JECFA established a PTWI of 100 ng/kg bw per week pending results of on-going studies on the mechanisms of nephrotoxicity and carcinogenicity. There is little evidence that humans are exposed to ochratoxin in significant amounts in most diets, however more monitoring and exposure data would be required to enable a definitive risk characterisation to be made.

In conclusion, there are no public health and safety concerns in relation to levels of dietary exposure to ochratoxin A from dairy products.

5.5.4 Trichothecene toxins

Trichothecene mycotoxins are produced by several field fungi, including *Fusarium graminearum* and *Fusarium culmorum*, and are common in cereals and grains, particularly in wheat, barley and maize. Co-occurrence with other *Fusarium* toxins, including zearalenone as well as well as the group of fumonisins, is regularly observed. Most reports describe type A: T-2 and HT-2 toxin; type B: DON and NIV, trichothecenes and will be the focus of this review. The chemical structures of the trichothecene mycotoxins T-2, HT-2, DON and NIV are given in Figure 7.

Among the naturally occurring trichothecenes in foods, T-2 toxin is the most potent, followed by NIV; DON, also known as vomitoxin, was the least toxic in acute toxicity studies. In experimental animals, T-2 toxin produce acute systematic effects, with necrosis of epithelial tissues and suppression of haematopoiesis. In contemporary outbreaks of disease, only gastrointestinal symptoms have been reported (IPCS 1990). Many outbreaks of acute human disease involving nausea, vomiting, gastro-intestinal upset, dizziness, diarrhoea and headache have been attributed to DON in Asia (IPCS 2001).



Trichothecene	R1	R2	R3	R4	R5
T-2 Toxin	-OH	$-OCOCH_3$	$-OCOCH_3$	-H	$-OCOCH_2CH(CH_3)_2$
HT-2 Toxin	-OH	-OH	$-OCOCH_3$	-H	$-OCOCH_2CH(CH_3)_2$
Nivalenol (NIV)	-OH	-OH	-OH	-OH	=O
Deoxynivalenol (DON)	-OH	-H	-OH	-OH	=O

Figure 7: Trichothecene toxins, T-2, HT-2, DON and NIV

Hazard identification and characterisation

Reported cases of human disease associated with trichothecene exposure are limited in number and information. Symptoms of digestive disorders and throat irritation develop rapidly after ingestion of food contaminated with trichothecenes. At present, there is no evidence of human cancer cause by trichothecenes (IPCS 1990).

In an epidemiological study, reporting human food poisoning caused by infected wheat in India in 1989 which affected an estimated 50 000 people, a NOEL of 0.44 μg/kg bw was estimated. The symptoms described include abdominal pain or a feeling of fullness in the abdomen, dizziness, headache, throat irritation, nausea, vomiting, diarrhoea, and blood in the stool. However, samples were collected four months after the outbreak, and the exposure was not limited to DON but included other toxins which leads to gross uncertainties in the estimated NOEL (SCF, 1999).

Although T-2 toxin, HT-2 toxin, DON and NIV appear to cause similar effects at the biochemical and cellular level and there are similarities in toxic effects, there are also substantial differences in the spectrum of toxic effects *in vitro*. Large, non-systematic potency differences between these toxins were seen when different endpoints are considered. There are very few studies addressing the combined effects of these toxins. Moreover, in most of these case studies naturally contaminated feed was used which makes the attribution of a potential effect to a single toxin very difficult (SCF, 1999).

The EU Scientific Committee on Food (SCF) has assigned temporary daily intakes (TDIs) to DON, NIV, T-2 toxin and HT-2 toxin pending among other things, a group evaluation. The TDIs for NIV and T-2 toxin were also made temporary because of gaps in the database. Therefore the Committee established a full TDI for DON (TDI = 1 μg/kg bw/day) only and confirmed the t-TDI for nivalenol (t-TDI = 0.7 μg/kg bw/day) and the combined t-TDI for T-2 toxin and HT-2 toxin (t-TDI = 0.06 μg/kg bw/day) (SCF, 2002e).

There is no data available regarding trichothecene residues in Australian dairy products. Although trichothecene toxins such as DON and T-2 can be carried- over into milk products (IPCS 1990), there is little residue data for residue levels of these toxins found in milk or milk products due to cattle eating mouldy feed.

Risk characterisation

On the basis of the data available there is a possible association between trichothecene exposure and episodes of human disease expressed as gastrointestinal symptoms. Secondary exposure to trichothecene toxins through consumption of dairy products derived from cattle fed trichothecene-containing feed, presents a negligible risk to the consumer. Carry over of DON to food products of animal origin are not thought to be of concern as animals refuse feed when mycotoxins are present in high concentrations, and DON undergoes rapid metabolism and elimination in livestock species.

In conclusion, there are no public health and safety concerns in relation to levels of dietary exposure to trichothecene toxins from dairy products.

5.5.5 Zearalenone

Zearalenone is a non-steroidal estrogenic mycotoxin (SCF, 2000b) that can be produced by several field fungi including *Fusarium graminearum* (*Gibberella zeae*), *F. culmorum*, *F. cerealis*, *F. equiseti* and *F. semitectum*. The main metabolites of zearalenone are α-and β-zearalenol and the glucuronide conjugates of both the parent compound and its metabolites (JECFA, 2000). Zearanol residues can be differentiated by the presence or absence of zearalenone metabolites. If zearanol occurs with other zearalenone metabolites it is more than likely due to the ingestion of *Fusarium* spp. – from infected pasture, or grain, or plant material containing zearalenone by the cattle. The chemical structures of the zearalenone (ZEA) and α- and β- zearalenol (ZOL) are given in Figure 8.

α-zearalenol has been previously assessed by JECFA as a veterinary medicine.

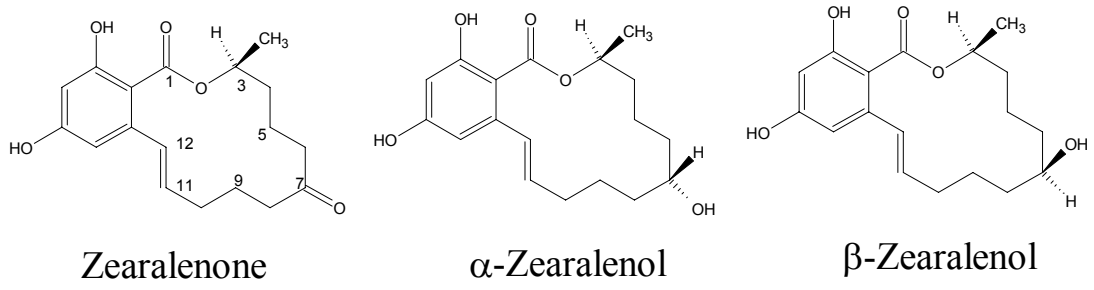


Figure 8: Chemical structures of zearalenone and primary metabolites

Hazard identification and characterisation

Zearalenone causes alterations in the reproductive tract of laboratory animals and domestic animals. Various estrogenic effects like decreased fertility, increased embryolethal resorptions, reduced litter size, changed weight of adrenal, thyroid and pituitary glands and change in serum levels of progesterone and estradiol have been observed but no teratogenic effects were found in mice, rats, guinea pigs and rabbits (Kuiper-Goodman *et al*., 1987; JECFA, 2000).

Pigs and sheep appear to be more sensitive than rodents (SCF, 2000a). Dairy cattle herds with low fertility were found to have higher levels of blood and urinary zearalenone and its metabolites, due to feeding on pastures with levels of about 400ppb of the mycotoxins (Whitlow, 2002).

In humans, zearalenone has been measured in endometrial tissue from 49 women and found at a concentration of 48 ± 6.5 ng/ml tissue from 27 women with endometrial adenocarcinoma, at 170 ± 18 ng/ml in tissue from 11 women with endometrial hyperplasia, and at concentrations below the limit of detection in tissue from 11 women with normal proliferative endometrium. Zearalenone was not detected in eight samples of hyperplastic and five samples of neoplastic endometrial tissue (Tomaszewski *et al*., 1998).

Zearalenone or zearalenol was suspected to be the causative agent in an epidemic of premature thelarche in girls aged six months to eight years, which occurred in Puerto Rico between 1978 and 1981, as these compounds were detected in blood plasma. The authors reported that homogenates of locally produced meat gave strong responses in a cytosol receptor assay with rat uterus, indicating the presence of substances that bind to oestrogen receptors, although the United States Food and Drug Administration later failed to detect any of the oestrogen growth promoters used in food. The involvement of natural sources of estrogenic compounds, such as some plant metabolites and mycotoxins, has not been ruled out (SCF, 2000b). A statistically significant correlation was found between the pubertal changes and the consumption of meat products and soya-based formula, but the association explained only 50% of the investigated cases, and the authors suggested better diagnosis and reporting or some unsuspected factor accounted for the reported increase in precocious pubertal changes (Freni-Titulaer *et al*., 1996).

JECFA concluded that the safety of zearalenone could be evaluated on the basis of the dose that had no hormonal effects in pigs, the most sensitive species. JECFA established a provisional maximum tolerable daily intake (PMTDI) for zearalenone of 0.5 μg/kg bw. This decision was based on the NOEL of 40 μg/kg bw/day obtained in a 15-day study in pigs (JECFA, 2000). The Committee also took into account the lowest observed effect level of 200 μg/kg bw/day in this pig study and the previously established ADI of 0-0.5 μg/kg bw for the metabolite α-zearalenol, evaluate as a veterinary drug (JECFA, 1988). The Committee recommended that the total intake of zearalenone and its metabolite (including α-zearalenol) should not exceed this value (JECFA, 2000).

There is no data available regarding zearalenone residues in Australian dairy products although in the 2003-4 NRS survey, zearanol was monitored in cattle and sheep meat and no residues were detected (DAFF 2005a).

Carry-over of zearalenone, α- zearalenol and β zearalenol into dairy products can occur though and low level of detection has been reported in milk and cheese (Hagler, 1980). In the U.K., for example, zearalenone was detected in 3% of conventional retail milk samples at levels ranging from 1.2 to 5.5 μg/L (EC 2003).

Dietary exposure

Estimated average dietary intakes of zearalenone based on individual diet records have been presented by FAO, indicating an exposure of 0.03 to 0.06 μg/kg bw/day, thus remaining below the PMTDI of 0.5 μg/kg bw/day set by JECFA.

Data from the EU Scientific Cooperation (EU SCOOP) taskforce showed that the mean intake of zearalenone, estimated from various European countries, might range from 1 ng/kg bw to 420 ng/kg bw/day. Bread and other cereal products were the most prominent sources of exposure (EFSA 2004b).

Thus although only few analyses have been performed on residues of zearalenone in animal derived products, the available information indicated that due to rapid metabolism and excretion of zearalenone, the contribution of products from animal origin, including poultry, to dietary exposure of zearalenone is very limited (EFSA 2004b).

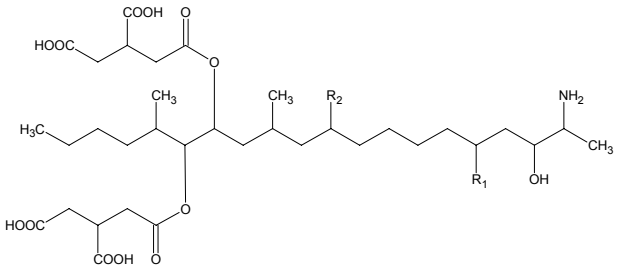
Risk characterisation

Zearalenone is a non-steroidal estrogenic mycotoxin implicated in numerous mycotoxicoses in farm animals, especially pigs. Estimated average dietary exposure internationally is below the PMTDI of 0.5 μg/kg bw/day. Susceptibility varies amongst species and limited experimental studies indicate that, after pigs, sheep are more sensitive to the adverse effects of zearalenone. Secondary exposure to zearalenone through consumption of products derived from dairy animals fed zearalenone-containing feed is very low compared to direct exposure via cereal and grain products.

In conclusion, there are no public health and safety concerns in relation to levels of dietary exposure to zearalenone from dairy products.

5.5.6 Fumonisin

Fumonisins are mycotoxins produced by fungi of the genus *Fusarium* that commonly contaminate maize. Fumonisin B_1 contamination of maize has been reported worldwide at mg/kg levels. Fumonisin B_1 is the diester of propane-1,2,3-tricarboxylic acid and 2*S*-amino-12*S*, 16*R*-diemthyl-3*S*, 5*R*, 10*R*, 14*S*, 15*R*-pentahydroxyeicosane in which the C-14 and C-15 hydroxy groups are esterified with terminal carboxyl group of propane-1,2,3-tricarboxylic acid (JECFA, 2001b). The chemical structures of fumonisin B_1 and closely related chemical substances fumonisin B_2, fumonisin B_3, and fumonisin B_4 are given in Figure 9.



Fumonisin	R_1	R_2
B_1	-OH	-OH
B_2	-H	-OH
B_3	-OH	-H
B_4	-H	-H

Figure 9: Chemical structures of fumonisins

Hazard Identification and Characterisation

In all species studied, fumonisins are poorly absorbed from the digestive tract and are rapidly distributed and eliminated. The liver and kidney retain most of the absorbed material, and fumonisin B_1 persists longer in rat liver and kidney than in plasma. In pregnant rats and rabbits, very low concentrations of fumonisin B_1 were recovered in the uterus and placenta. No fumonisin B_1 was found in the foetuses, indicating an absence of placental transfer.

There was little evidence of significant transfer during lactation, and fumonisins do not appear to be metabolised *in vitro* or *in vivo* (JECFA, 2001a; JECFA, 2001b).

In all animal species studied, the liver was a target for fumonisin B1; the kidney was also a target in many species. In kidney, the early effects are often increases in sphingoid bases, renal tubule-cell apoptosis, and cell regeneration. In liver, apoptotic and oncotic necrosis, oval-cell proliferation, bile-duct hyperplasia, and regeneration are early signs of toxicity (JECFA, 2001b).

A specific role for fumonisins in the development of neural tube defects has been proposed. The hypothesis includes a critical role of fumonisins in disruptions of folate membrane transport, but no specific studies have been designed to confirm this mechanism (JECFA, 2001b).

The IARC has classified fumonisin B_1 into group 2B (possibly carcinogenic to humans – sufficient evidence in animals, and inadequate data in humans) (IARC, 2002b).

Nephrotoxicity, which was observed in several strains of rats, was the most sensitive toxic effect of pure fumonisin B_1. Since the available studies clearly indicate that long-term renal toxicity is a prerequisite for renal carcinogenesis, the potential for the latter is subsumed by the dose-response relationship for renal toxicity. Therefore, the pivotal studies that could serve as the basis for a tolerable intake of fumonisin B_1 were the short-term and long-term studies of toxicity in rodents. On the basis of these studies, the overall NOEL for renal toxicity was 0.2 mg/kg bw/day (JECFA, 2001b).

JECFA allocated a group provisional maximum tolerable daily intake (PMTDI) for fumonisins B_1, B_2, and B_3, alone or in combination, of 2 μg/kg bw/day on the basis of the NOEL of 0.2 mg/kg bw/day in rats and a safety factor of 100 (JECFA, 2001b).

While the acute toxicity if fumonisins is low, it is the known cause of two diseases that occur in domestic animals with rapid onset: equine leukoencephalomalacia and porcine pulmonary oedema syndrome.

Both of these diseases involve disturbed sphingolipid metabolism and cardiovascular dysfunction. Overall, cattle appear to be less susceptible to fumonisins compared to other species.

Only few analyses have been performed on carry-over residues of fumonisins in animal derived products. Although, fumonisin B_1 levels in animal feedstuff can be exceptionally high, and reach maximum values of 330, 70, 38, 9 and 2 mg/kg in North America (USA), Europe (Italy), Latin America (Brazil), Africa (South Africa) and Asia (Thailand), respectively (IPSC, 2000), in milk, fumonisin B1 was found in only very few samples (<0.006%) at levels close to 5ng/ml (Maragos and Richard, 1994) and is not significantly transferred into milk from short-term dietary exposure (Prelusky *et al*., 1996).

Dietary exposure

Maize is the only commodity that contains significant amount of fumonisins (IPCS 2000b). Estimated mean dietary intakes of fumonisin B_1 based on regional diets and published distributions of concentrations of fumonisin B_1 in maize, indicating a mean intake of fumonisin B_1 ranging from 0.2 μg/kg bw/day in European-type diet to 2.4 μg/kg bw/day in the African diet (JECFA, 2001b).

Fumonisin B_1 may be found in low concentrations in dairy products but should not contribute significantly to human dietary exposure. Furthermore, fumonisins are poorly absorbed, rapidly excreted and not metabolised in animal systems.

Risk characterisation

Fumonisin B_1 is carcinogenic in mice and rats and induces fatal diseases in pigs and horses at levels of exposure that humans encounter. Fumonisin B_1 has been associated with sporadic gastrointestinal disorders in humans and, for populations whose diet is based on maize, there are correlative studies linking fumonisins and oesophageal cancer (IPCS 2000b). Secondary exposure to fumonisin B_1 through consumption of dairy products derived from dairy animals fed fumonisin B_1-containing feed is very low and represents a negligible risk to the consumer as there is little carry-over of the toxin into milk.

In conclusion, there are no public health and safety concerns in relation to levels of dietary exposure to fumonisin B1 from dairy products.

5.5.7 Cyclopiazonic acid

Cyclopiazonic acid (CPA) is a toxic indole tetramic acid that is produced by a number of different fungi that infect different foodstuffs, for example, *Penicillium* species (e.g. *P. commune* and *P. camembertii*) and *Aspergillus flavus and A. versicolor*. As it can be formed by *A. flavus*, a species that is a major producer of aflatoxins, it has the potential to co-occur with these mycotoxins in a range of food commodities, including milk, cheese and butter (Dorner *et al.,* 1994).

Hazard Identification and Characterisation

CPA only appears to be toxic when present in high concentrations. It has been found to be a neurotoxin when injected intraperitoneally into rats and the LD50 in male rats was 2.3 mg/kg.

Oral administration produced no convulsions and LD50 values found in rats for administration by this route were 19 - 36 mg/kg and 63 mg/kg for males and females respectively (Morrissey *et al*., 1985). In addition, lesions in the liver, kidney, spleen and other organs were observed. The effects reported include decreased weight gain, diarrhoea, dehydration, depression hyperaesthesia, hypokinesis, convulsion and death. It is reported that some of its effects in the body are due to its interference with the uptake and release of Ca2+ so it could pose a particular risk to humans taking drugs such as calcium antagonists designed to carefully control calcium homeostasis (EMAN 2005).

Studies of the effect of CPA are also reported on broiler chicks. The observed toxicity may be masked or caused by other co-occurring mycotoxins of which aflatoxins and T2-toxin have been cited (EMAN 2005)and also ochratoxin A (Gentles *et al*., 1999). The induction of toxicity seen in exposure to ochratoxin alone and CPA alone indicates that these two mycotoxins express their toxicity by different mechanisms.

CPA toxicity was expressed mainly through increased relative weights of the proventriculus and increased activity of creatine kinase. The combination of ochratoxin and CPA was characterised by increased relative weights of the liver, kidney, pancreas and proventriculus; decreased concentrations of serum albumin, total protein and increased concentrations of triglycerides and uric acid (Gentles *et al.,* 1999).

CPA is mutagenic for *Salmonella typhimurium* TA98 and TA100 in the Ames assay and its ability to co-occur with aflatoxins and may enhance the overall toxic effect when this happens. There is a dearth of human exposure data and this precludes an assessment of possible health effects. However, 'Kodua' poisoning in India resulting from ingestion of contaminated millet seeds has been linked to this toxin.

It has similar pharmacological properties to the anti-psychotic drugs, chlorpromazine and reserpine, in mice and rabbits. Near lethal doses of 11 to 14 mg/kg body weight induce continuous involuntary tremors and convulsions. It may be able to produce similar effects in humans (EMAN 2005).

CPA imine is a related metabolite that occurs in culture but is considered to be much less toxic than the parent compound.

An attempt to estimate an acceptable daily intake has been reported, based on a no observed effect level (NOEL) of 1 mg/kg/day, which takes into account data for several animal species and species variation. This indicates that an appropriate acceptable daily intake (ADI) would be approximately 10 micro g/kg/day or 700 micro g/day. In the context of human exposure, if the uppermost limit of CPA found in cheese is 4 µg/g and the average individual consumes 50 g of cheese daily, this allows an intake of 200 µg, less than one third of a traditionally established ADI (EMAN 2005).

Dietary exposure

CPA has been detected in Europe at levels up to 10 mg/kg or higher in cheese, milk , stockfeed (maize, millet, peanuts, pulses, hay) and mixed feeds. Some cheeses are surface ripened with the species *P. camembertii* that can produce CPA, so there is intensive scrutiny of the strains used to ensure that they are non-toxin producers (EMAN 2005).

When lactating ewes were administered 5mg/kg /bw/day CPA for two days, the effect on the ewes was rapid and milk production and feed intake dropped within 24h.

After 48h, milk production had dropped to 20% and animals had increased respiration rates and body temperatures. CPA was found in the milk (236 ng/g after day 1) and concentrations rose after the second dose. CPA remained detectable in the milk for up to 9 days (Dorner *et al.,* 1994).

In an assessment of moulds isolated from the rind of Taleggio cheese, twenty-seven strain of Penicillium were isolated and all produced CPA (Finoli *et al.,* 1999). Although it the toxin can migrate to the core of the cheese, the highest concentrations were found in the rind.

Risk characterisation

The occurrence of CPA in milk and dairy produce is potentially of concern due to its high toxicity to major organs and due to its interference with the uptake and release of Ca2+; toxic effects have been shown in different animals and in humans ('Kodua' poisoning).

In addition, CPA can be produced by a number of species of *Aspergillus* and *Penicillium*, which increases the potential for natural CPA contamination of stockfeed. However, the incidence of CPA in food is very low, possibly as it occurs in the same products susceptible to aflatoxin contamination, and is therefore indirectly controlled by regulations in place for the aflatoxins More data would be required to complete a full risk characterisation of CPA.

5.5.8 Corynetoxins

Corynetoxins are a group of closely related tunicamycin-like toxins produced by pathogenic plant bacteria, *Rathayibacter toxicus (*also known as *Clavibacter toxicus,* formerly *Corynebacterium spp*.). The disease associated with corynetoxin toxicity is known as annual ryegrass toxicity (ARGT) (Edgar 1994) and is almost unique to Australia due to the specific type of grasses grown for pasture. Large areas of Western Australia and South Australia and, to an unknown extent, areas of Victoria, New South Wales and Queensland are susceptible to infection with *R. toxicus* or *Anguina spp.* or both. Corynetoxins, extracts of toxic seed heads and toxic ryegrass have proven lethal to all animal species exposed naturally, or tested, including sheep, cattle, horses, donkeys, pigs, guinea pigs, rats, mice and chickens.

There is a history of ARGT in Australia since 1955 and cattle and sheep losses have been reported in large numbers since then. Corynetoxicosis of livestock grazing on infected *Agrostis avenacea* caused Flood plain staggers on the flood plains of the Darling river in northern New Sough Wales between spring 1990 and autumn 1991. Over this period 1722 cattle, 2466 sheep and 11 horses died on 31 farms (Davis *et al*., 1995). On average, about 30,000 sheep and several hundred cattle die each year from ARGT in the wheat belt of Western Australia (Allen, 2002). Although the bulk of dairy cattle are reared on the coastal belt of WA, they would be at risk if they were fed toxic hay or toxic grain as supplement (J. Allen, personal communication).

The contamination of hay and straw by corynetoxins is a major concern for Australian hay and straw exports, and the Department of Agriculture Fisheries and Forestry have recently released information on a standard for minimising risk of corynetoxin contamination of hay and straw for export (DAFF 2005b).

Hazard Identification and Characterisation

Corynetoxins cause livestock poisoning through a relatively complex association between a grass, nematode, bacterium and a bacteriophage.

Germinating grass seedlings, including annual ryegrass (*Lolium rigidum*), annual beardgrass (*Polypogon monospeliensis*) and blown grass (*Agrostis avenaceae*) are infected with a nematode (*Anguina sp*.), which carry out their life cycle in the flower heads of the developing grasses (McKay and Ophel, 1993). The bacterium *R toxicus* may be carried by the nematode and produces toxins, during senescence of the pastures, which can then persist in the dry pasture during the spring and summer. Animals grazing such pastures may become intoxicated, characterised by a stagger, although the toxicity of the pasture decreases in autumn with new grass growth. The symptoms of ARGT in livestock closely resemble those of Bovine Spongiform Encephalitis (also known as mad-cow disease) i.e., intermittent episodes of cerebral convulsion superimposed on varying degrees of cerebella dysfunction, and often result in the death of the infected livestock. It is thought though that toxin is only produced if *R. toxicus* is infected with a bacteriophage (Ophel *et al*., 1993).

The corynetoxins are a family of at least eight separate glycolipid molecules (Vogel *et al*., 1981), which are closely related to the tunicamycin group of antibiotics in structure and function (Edgar *et al*., 1982). Structurally, corynetoxins are made up of uracil, N-acetylglucosamine, an 11 carbon sugar (tunicamine) and a fatty acid. The sugar moieties of these toxins are essential for toxicity.

The corynetoxins are potent, irreversible, transition state analogue inhibitors of *N*-acetylglucosamine-1-phosphate transferase (GPT). Since this enzyme catalyses the initial step in the biosynthesis of the dolichol-linked oligosaccharide chains destined for *N*-linking to proteins, the corynetoxins block N-linked glycoprotein synthesis (Jago *et al*., 1983) and consequently have high, general mammalian toxicity**,** and similarly inhibit peptidoglycan synthesis in the cell wall of bacteria. Reduced fibronectin levels are thought to be a cause of the breached blood-brain barrier, impaired cardiovascular function and decreased peripheral circulation and oxygen utilisation seen in corynetoxin poisoned animals (Berry *et al*., 1980). A lethal oral dose of corynetoxins for sheep, cattle or pigs is between 1 and 5 mg/kg. The primary organs of corynetoxin toxicity are the central nervous system and the vascular system.

Much information on the toxicity of the corynetoxins is derived from studies with tunicamycin, which has close structural similarity to corynetoxins and has a similar mechanism of action as the corynetoxins. Tunicamycin produces similar clinical disease in sheep and rats, and has similar toxicity. Depending on the sensitivity of the animals and on the source form of the toxin, the oral lethal dose for tunicamycin or corynetoxins varies between approximately 1 mg/kg and 5.6 mg/kg. For acute toxicity at least, sheep, cattle and pigs are all equally susceptible. The approximate lethal dose for tunicamycin or corynetoxins given by subcutaneous injection to sheep is 20-40 μg/kg. Reported lethal doses by parenteral (subcutaneous and intraperitoneal) administration for nursling rats, adult male rats and adult female rats are 110-160, 350 and 450 μg/kg, respectively (Allen, 2002).

The corynetoxins are cumulative toxins and the total lethal dose is the same whether given as a single dose or as repeated smaller doses up to 2 months apart. This cumulative effect is more obvious in sheep than rats (Jago and Culvenor, 1987).

Although the effects of large doses of corynetoxins have been described (Jago and Culvenor 1987), the effects of long term, low level exposure to these toxins in the diet or environment are unknown.

However, because the corynetoxins are cumulative in their action there is reason to suspect this type of exposure may pose a risk to human and animal health (Colegate *et al*., 1998).

In addition to being fatally toxic to animals, sheep and cattle can apparently ingest up to ¾ of a lethal dose and still appear clinically unaffected. There is a lag time of about 3 or 4 days between ingestion of a lethal dose and the onset of clinical signs. These factors can contribute to apparently normal, but intoxicated animals, being presented for slaughter (MLA, 2003).

Dietary exposure

The major areas of exposure of livestock to pasture-based contaminated feed are in the southern regions of Western Australia and South Australia. All domestic food and food product animals can potentially be exposed to corynetoxins in their feed supply. This includes pasture based feed , grains and fodder based feeds.

Since infected annual ryegrass is a common weed in grain crops, humans can be exposed, in a primary manner, to corynetoxins by inhalation of dust associated with grain harvesting, transportation and processing, or by ingestion of food products from contaminated grain, especially when a local, contaminated crop is sourced by the consumer for home processing. If an animal is primarily exposed to corynetoxins, then humans could be exposed in a secondary manner if the food products derived from the exposed animal are ingested.

Initial data (Stewart *et al*., 2004) indicates that the corynetoxin analogue, tunicamycin, can be translocated to muscle tissue in addition to liver, kidney and heart. However there was no reduction in GPT levels in the livers of rats nursing from tunicamycin-treated dams; this preliminary research indicated that corynetoxins are not carried-over to milk of lactating rats.

Risk characterisation

There have been no instances of human clinical symptoms being ascribed to exposure to corynetoxins. The corynetoxins have an affinity for cellular membranes and thus are cumulative in their action. The clinical and sub-clinical effects of long term, low level exposure to the cumulative corynetoxins are currently unknown. The limited data available indicates that carry-over of corynetoxins into milk is unlikely. Further data is required before a risk characterization can be completed.

5.5.9 Pyrrolizidine alkaloids

Pyrrolizidine alkaloids (PAs) are plant toxins that may find their way into human and animal food in Australia. They are derived mainly from the plants *Heliotropium europaeum* (common heliotrope or potato weed), *Echium plantagineu* (Pattersons' curse), *Senecio spp.* (ragwort), *Symphytum* spp (comfrey). and *Crotalaria retusa* (rattleweed). The *Sympthytum* spp. is deliberately ingested while the remaining species are weeds in various grain crops. There is a long history of toxicity in livestock caused by grazing on PA-containing plants although plants producing PAs are uncommon in improved pastures used in dairy production.

There are more than 50 types of PAs, some of which have been shown to be toxic to animals at very low doses. There have also been a number of outbreaks of human poisoning as a result of ingestion of contaminated grain as well as case reports of poisoning caused by intentional ingestion of herbal medicines containing PAs (FSANZ, 2001c).
No MLs for pyrollizidine alkaloids in food have been established.

Hazard Identification and Characterisation

The PAs of relevance to human health are the hepatotoxic PAs which are esters of 1-hydroxymethyl dehydropyrrolizidine. Such compounds are metabolised in the liver to electrophilic derivatives referred to as pyrroles. These pyrroles cause damage in the hepatocytes in which they are generated, but depending on their persistence in aqueous media, can pass from the hepatocytes into the adjacent sinusoids and damage endothelial lining cells of the sinusoids and smallest hepatic veins. These effects give rise in man to hepatocellular injury, cirrhosis and veno-occlusive disease.

The pyrroles react with macromolecules in the cells in which they are either formed or gain access leading to the formation of S-bound protein adducts and DNA crosslinking.

The pyrones have been shown to have mutagenic activity, mainly in *Drosophila* and many have been shown to be carcinogenic, mainly in the rat. There is no evidence of pyrollizidine alkaloid-induced cancer in humans (FSANZ, 2001c).

In laboratory and domestic animals, marked anti-mitotic activity due to the pyrones has been demonstrated but this is not a prominent feature of their toxicity in humans. The main pathological feature of this effect in animals is in the liver, and less so in other tissues.

In humans, the major toxicological effect of chronic exposure to PAs is veno-occlusive disease. The available data on cases of veno-occlusive disease in humans indicates a tentative no-observed-effect level (NOEL) of 10 µg/kg bw/day can be established. If an uncertainty factor of 10 to account for human variability is applied to this NOEL, the provisional tolerable daily intake (PTDI) for PAs in humans is 1 µg/kg bw/day (FSANZ, 2001c).

Dietary exposure

Apart from the deliberate use of herbal remedies and nutritional supplements containing PAs, humans can become inadvertently exposed through consumption of contaminated food. The foods which have been found to contain PAs include grains, honey, milk, offal and eggs. More specifically, PAs found in goats milk were shown to produce hepatoxic effects in rats (Goeger *et al*., 1982).

In relation to milk from domestic animals, it is likely that no more than about 0.1% of the ingested alkaloid base will be excreted in milk. PAs and PA N-oxides are known to be excreted in cow's milk, but due to milk bulking, it is unlikely that significant exposures would come from this source.

Substantial contamination of grain commodities has been recorded in various countries due to both contaminations by seeds of PA-containing weeds growing in the crop as well as plant dust fragments from the same plants. The levels of PAs found in various grain commodities in Australia have ranged from <50 to >6000 µg/kg, but there has been no systematic analysis of the levels in grains entering the food supply. There is currently no data to indicate whether PAs occur in oilseed crops. On the basis of the very limited data available, the major source of dietary exposure to PAs is grains; eggs, offal, honey and milk are minor dietary contributors.

Risk characterisation

While PAs can cause liver cancer in rats, there is no evidence from the significant human epidemics that have occurred, that PAs cause liver cancer in humans.

Further research on the mechanisms of PA-induced hepatotoxicity may clarify the apparent differences in species specificity. While there is survey data to suggest that significant levels of PAs can be found in some foods, and particularly in grains, there is virtually no data on the levels of PAs in foods as consumed. It is unlikely that significant exposure to PA would come from dairy products as dairy pasture is managed in order to exclude plants with high PA contents, and, if present, the carry-over of PAs into milk is very small. In conclusion, on this basis there are unlikely to be safety concerns in relation to dietary exposure to PAs from dairy products. However further data would assist in further characterising the public health and safety risk.

5.5.10 Lupin alkaloids

The quinolozidine alkaloid, found in the *Lupinus* genus, is of major concern to human and animal health. The levels of alkaloids in seeds or meal can be reduced to approximately 500mg/kg, through a de-bittering process involving soaking or washing with water.

In Australia, lupin varieties with low alkaloid content ("sweet lupins") have been developed through plant-breeding programmes, and levels of alkaloids have been reduced to 130 – 150 mg/kg. Humans consume lupins in the form of seed flour and meal that can be used to prepare pastas, pastries and dairy product substitutes. Lupins are also used in traditional fermented foods such as tempe, miso and soy sauces in Indonesia and Japan (FSANZ, 2001a).

Several species of lupin are poisonous to livestock, producing death in sheep and "crooked calf disease" in cattle (Lopez-Ortiz *et al*., 2004). Pregnant cows have the greatest risk of giving birth to calves with crooked calf disease when the concentration of the teratogen anagyrine is highest and the cows are in the susceptible 40-75 day gestation period when ingesting the plant (Keeler R.F. *et al*., 1976). In addition, milk production is reduced in cows fed lupins, this could be partly due to a reduced true protein supply to the small intestine. Smaller studies have also indicated that *Lupinus formosus* caused clinical toxicoses in cows. In Australia, generally only 2% of dairy rations would include lupin (Dairy Australia, personal communication).

An ML for lupin alkaloids in lupin flour, lupin kernel flour, lupin kernel meal and lupin hulls was included in Table to clause 5 in Standard 1.4.1 of the *Australia New Zealand food Standards Code*. The ML for lupin alkaloids in mixed foods was set at 200 mg/kg

Hazard Identification and Characterisation

Humans appear to be the most sensitive species for alkaloid toxicity. Human poisonings due to lupin alkaloids indicate that the acute lethal dose is approximately 30 mg/kg, where the major alkaloid is sparteine. Traditional consumption of debittered lupins in Europe suggests a dose of 0.35 mg/kg/day is without chronic effect for adults. If a safety factor of 10 is applied to account for the uncertainties in the data and particularly to take into account likely human variation, the provisional tolerable daily intake (PTDI) for humans is 0.035 mg/kg/day (FSANZ, 2001a).

Exposure Assessment

Human exposure to lupin alkaloids is considered to be largely from direct consumption of lupin meal and not from carry-over of the alkaloids in milk, however there is currently no data available on the levels of lupin alkaloid in milk.

Risk Characterisation

There is no data available on potential carry-over of lupin alkaloids into milk and therefore the potential public health and safety risk cannot be characterized.

5.5.11 Phomopsins

The phomopsins are a family of mycotoxins produced by the fungus *Phomopsis leptostromiformis*. Lupins are the main host for the fungus, which is capable of infecting most parts of the plant.

Infection of the vegetative parts of the plant can result in high levels of phomopsin being present on the stubbles, which is the major source of animal exposure to phomopsin. Under certain storage conditions, infected lupin seed can also exhibit significant levels of phomopsin contamination. While the majority of lupin seed is used in animal feed, lupin products are also increasingly being introduced into food for human consumption.

Therefore, whole lupin seed and flour may be a source of human exposure to phomopsins, which have been shown to be stable to processing, including cooking (FSANZ, 2001b).

An ML for phomopsins in lupin seeds and the products of lupin seeds is included in Table to clause 3 of Standard 1.4.1. The ML for phomopsins was set at 0.005 mg/kg.

Hazard Identification and Characterisation

Overall phomopsins are potent cytotoxic agents which predominantly target the liver and which are clearly liver carcinogens in the rat. Phomopsins may be less toxic by the oral route than other routes, although they are still capable of causing severe disease, e.g., lupinosis in sheep Also, some animal species appear more vulnerable than others to the toxic effects of phomopsins. The cytotoxic nature of phomopsins suggests that humans would also be vulnerable to its toxic effects; however, the available animal studies do not allow a determination of a safe level of dietary exposure to phomopsins.

Given these concerns, particularly with regard to the potential carcinogenicity of phomopsins, it would be prudent to ensure that human exposure be kept as low as is reasonably achievable. The paucity of toxicity data available does not make it possible at this time to identify a NOEL in animal studies or assign a tolerable level for human exposure (FSANZ, 2001b).

Dietary exposure

Levels of phomopsins in lupin seed (from Australia) vary from<6µg to 360µg/kg and levels as high as 4522µg/kg in seed have also been detected. However, the overall dietary exposure of dairy cattle to lupins is 2% of their total diet.

There is no data available on the levels of phomopsins carried over to lupin flour. Therefore, it is not clear to what extent the milling process may remove phomopsin contamination. In addition, no data is available for other potential sources of exposure such as other lupin products, offal and milk. Therefore, there is insufficient survey information to enable a dietary exposure assessment to be carried out. However, sub-population groups most likely to have high exposure to phomopsins would be those consuming large amounts of lupin products (FSANZ, 2001b).

Risk Characterisation

Phomopsins have been shown in animal studies to be potent liver toxins and carcinogens in rats. Although no direct evidence of toxicity in humans is available, their mechanism of action is such that humans are likely to be susceptible to their toxic effects. Phomopsins appear to be less toxic by the oral route than by other routes but still capable of causing severe liver disease in sheep following ingestion. If affected, animals show signs of liver disease and may die within a few days. Although there is no data available on whether phomopsins are excreted in milk it is unlikely to be a risk to public health and safety as lupins are not a major feed source for dairy cattle.

5.5.12 Ergot

Ergot alkaloids (ergolines) are produced by the fungus *Claviceps purpurea* that infects the florets of grasses and cereals, forming sclerotia. All the common cereals can be infected with ergot, including rye, wheat, barley, triticale, oats, millet, sorghum and maize.

The ergolines, contained within the scelrotia, are derivatives of lysergic acid and fall into three groups, ergotamine, ergotaminine and clavines (EFSA 1990).

The ML for ergot is set at 500 mg/kg in cereal grains.

Hazard Identification and Characterisation
Ergotism in man is relatively uncommon but it can affect livestock producing the following effects: behavioural effects, convulsions, lack of coordination, lameness, and difficulty in breathing, excessive salivation, diarrhoea and dry gangrene of the extremities. Reproductive effects including abortion, high neonatal mortality, reduced lactation, reduced feed intake and weight gain. These are species-specific effects, which depend upon the ergot source, amount consumed, period of exposure and age and stage of production of the animal (EMAN 2005). Ergot infection of grains, such as sorghum, has been found to reduce milk production; for example, cattle fed infected grain at 1% concentrations reduced milk yield by 30% after 5 weeks. The suggested maximum tolerable sclerote levels in dairy cow feed (whole diet) is 0.3% as these concentrations did not affect milk, if cows were on a full grain ration, the limit has to be reduced to 0.1% sclerotes (DPI 2005).

Exposure assessment
All the common cereals including rye, wheat, barley, triticale, oats, millet, sorghum and maize can be infected with ergot, although rye is the most susceptible. In Europe, rye bread has often been linked to outbreaks of ergotism. Ergot alkaloids are not transferred to the milk of cows consuming ergot (EMAN 2005).

Risk characterisation
Although ergot alkaloids have toxic effects in animals there is no evidence that there is carry-over of ergot into milk and therefore there are no public health and safety concerns associated with ergot residues in milk.

5.6 Water as a source of chemical contaminants

As part of the on-going food safety programme established by the Australian dairy industry, an investigation by the National Milk Harvesting Centre was commissioned to determine if the use of water on dairy farms posed any risk to the food safety of milk (DPI, 2003). This process was based on the water safety plan suggested in the WHO's guidelines for drinking water quality (draft 3rd edition).

All water-milk contact pathways had low risk scores and the pathway with the greatest risk to the food safety of milk was milking plant flush with a risk score of 6.3 from a maximum possible score of 125 (~5%).

Using a complex model that utilises the specific biotransfer factors for each potentially contaminating chemical the maximum limit for chemical contamination from contact with water was calculated as between zero and 1.2%. However, the presence of contaminants at these levels would be undetectable.

Mercury and the organic solvent 1,2-dichlorobenzene (1,2- DCB) were identified as having the greatest potential contribution to contamination of milk through water contamination. Natural release of mercury into drinking water is extremely low, but contamination can result from industrial emission or spills.

Mercury has been estimated to be present at in extremely low amounts below the detectable limit of 0.0001 mg/L (NHMRC 2004) in milk through water contamination. 1,2-DCB is used primarily as a chemical intermediate for dyestuffs and pesticides, however it has not been found in Australian drinking water; when farming is situated adjacent to industrial areas its presence could arise from inadvertent spills, atmospheric deposition or by contact with contaminated soils, as has been found overseas.

Water food safety hazards would be higher resulting from industrial/manufacturing use, however, Dairy primary production is not in vicinity of industrial areas, and if water does become contaminated eg from a chemical spill, this will be highly visible (eg fish kill) and entry of the water into dairy processing would be averted.

Algal blooms in waterways may also be a source of toxins, for example, microcystin, cylindrospermopsin and saxitoxin (Briand and Humbert, 2003). Dramatic intoxification events have occurred in Australia due to toxins released from algal blooms, for example in 1992, 10,000 livestock died along the Darling river from a massive bloom of the Neurotoxic cyanobacteria, *Anabaena circinalis* (Falconer, 1998).

Whilst algal toxins pose a problem for cattle, there is no evidence that the microcystin-LR toxin is carried-over to milk (Feitz A.J. *et al*., 2002) and therefore is of negligible risk to the consumer. There is no data available regarding the carry-over of other algal toxins into milk. Saxitoxin is associated with paralytic shellfish poisoning and is only regulated by FSANZ with regard to bivalve molluscs (under Standard 1.4.1).

In conclusion, no food safety risks were identified with the use of water in dairy farms.

5.7 Miscellaneous

5.7.1 Radionuclides

Australia, in common with all countries in the world, has received fallout from atmospheric nuclear weapons tests conducted by various nations.

Generally fallout has been substantially less in Australia than for countries in the northern hemisphere but there have been small contributions from French atmospheric tests in the Pacific in the 1970s and from British tests in Australia in the 1950s.

The Australian Radiation Protection and Nuclear Safety Agency (ARPANZA) have issued a statement confirming the radiation-safe status of foodstuffs, including milk and milk products, in Australia[34] and AQIS routinely monitors dairy products for the presence of Caesium and Strontium for export certification.

Routine fall-out monitoring has been undertaken in all Australian states since the mid 1950s and detected minimal levels of radioactivity from nuclear testing in the northern hemisphere. During the British testing in Australia extensive monitoring was undertaken. Following cessation of French atmospheric nuclear weapons in the Pacific in 1974, fallout deposition decreased rapidly until the present, where levels are at or below the minimum detectable in air. Monitoring since the Chernobyl accident showed no increase in fallout deposits and all indications are that essentially no fallout from Chernobyl occurred in Australia.

[34] (www. arpanza.gov.au).

Extensive testing since the Chernobyl accident of many agricultural products (inducing foodstuffs) exported from Australia confirms that the radioactivity levels are negligible and significantly less than the 10 Becquerels per kg measured as Caesium 134 and Caesium 137. ARPANSA routinely analyses radioactivity in a range of food products and they are found to be effectively free of any radionuclide contamination

In 1989, Codex established guidelines for radionuclides in foods following accidental nuclear contamination for use in international trade(Codex 1989). In the event of such a nuclear accident occurring, Codex has prescribed a list of agricultural and semi-natural countermeasures. The International Commission on Radiological Protection has also established intervention levels for different foods; this is calculated using the reference level of dose for an accident (5mSv) as a function of the mass of food consumed (kg) and the dose per unit intake factor (Sv/kg). In dairy produce (milk and cream), the intervention levels for adults of isotopes of strontium (^{90}Sr), iodine (^{131}I), alpha emitting isotopes of Plutonium (Pu) and trans Pu-elements, and all other radionuclides of half-life >10 days, notably ^{134}Cs and ^{137}Cs, are 125, 500, 20 and 1000 Bq/kg respectively (Pates 2000). Due to the sensitivity of infants, separate guidelines have been established; the intervention values for ^{90}Sr, ^{131} I, and ^{136} Cs are 160, 1600 and 1800 Bq/L respectively (WHO 1988).

In conclusion, there are no public health and safety concerns associated with dietary exposure to radionuclides from the consumption of milk and milk products. The occurrence of an incident in a country with active radionuclear industries would influence this assessment.

5.7.2 Development of antimicrobial resistance

The development of antimicrobial resistance is a relatively recent problem, which impinges on animal and human health as well as antibiotic usage patterns.

Antibiotics kill most, if not all, of the susceptible bacteria, but may leave behind – or select, in biological terms –bacteria that have developed resistance, which can then multiply and thrive. Pathogenic bacteria that were formerly susceptible to an antibiotic can develop resistance through changes in their genetic material.

These changes can include the transfer of DNA from resistant bacteria, as well as spontaneous changes, or mutations, in a bacterium's own DNA. The DNA coding for antimicrobial resistance (AMR) can be located on either the chromosome or plasmid of a bacterium. Plasmid-based resistance is transferred more readily than chromosomal-based resistance.

Once acquired, genetically determined AMR is passed on to future generation and sometimes to other bacterial species. The dose of antimicrobial and length of time bacteria are exposed to the antimicrobial are factors affecting whether the resistant bacteria population will dominate. Low doses of antimicrobials administered over long periods of time to large groups of animals, such as doses used for growth promotion in animals, favour the emergence of resistant bacteria.

The prophylactic use of antimicrobials (i.e. medications administered for lengthy periods, associated with claims of improved feed efficiency and growth promotion) has been a cause for concern regarding the induction of AMR.

Through the activities of the Joint Expert Advisory Committee on Antibiotic Resistance (JETACAR) and the Expert Advisory Group on Antimicrobial Resistance (EAGAR) (see Appendix 12), the registration of several antibiotics, have been withdrawn, or are under review. The streptogramin, virginiamycin is used therapeutically in feed premix for cattle for the treatment of acidosis.

However, the labelling instructions have recently been revised for dairy cattle usage by the APVMA (APVMA 2003b). No other antimicrobials are registered for in-feed administration for dairy cattle.

AMR and dairy pathogens

In addition to many international reports of AMR as a result of the treatment of mastitis (for example, (Kirk *et al*., 2005), (Makovec and Ruegg, 2003);(Wallmann *et al*., 2003)), the incidence of AMR through antibiotic usage in the Australian dairy industry has been monitored in Queensland since 1999 (Stephens 2003). Table 8 detail results collated by the Toowoomba Veterinary Laboratory between 1999 and 2000. In samples taken from bovine mastitic milk, one third of *S. aureus* isolates were found to be resistant to ampicillin and penicillin, whilst a small number were resistant to novobiocin (Table 27). Over the past three years, there have not been significant increases in antimicrobial resistance and AMR for tetracycline, ampicillin and penicillin has plateaued (C. Stephens, personal communication).

The antibiotics detailed in Table 27 with high and/or multiple AMRs to dairy pathogens, are considered to be of low importance according to the EAGER Importance Rating (EAGAR 2003).

In conclusion, there is no evidence that the use of antimicrobials in the dairy industry contributes significantly to the development of AMR.

Table 27: Antibiotic susceptibility testing of *Staphylococcus aureus* isolated from bovine mastitic milk between 1999 and 2001 **(Stephens 2003)**

Antibiotic	Amp10	CXM30	DA2	N30	NV30	OB5	P10	TE30
Number of strains tested	121	107	121	121	120	121	121	121
Number of strains resistant	40	0	0	0	3	0	40	0
Strains resistant %	33.1	0	0	0	2.5	0	33.1	0

AMP10 = ampicillin (10μg)
CXM30 = cefuroime (30 μg)
DA2 = clindamycin (2 μg)
N30 = neomycin (30 μg); novobiocin (30μg)
OB5 = cloxacillin (5μg)
P10 = penicillin (10 i.u.)
TE30 = tetracycline (30 μg)

5.7.3 Sanitisers and cleaning agents

Milk handling and processing results in milk soils and deposits which comprise mainly of minerals, lipids, carbohydrates and proteins. In addition, other potential contaminants in

milk-handling equipment include dust, microorganisms and lubricants. Thus, cleaning and disinfection are critical aspects of GMP in the dairy industry. Sanitisers and cleaning agents are regulated as Agvet chemicals by the APVMA.

Sanitising reduces microorganisms to acceptably low numbers, unlike sterilisation, which destroys all microbial life. Sanitisers are applied to surfaces that have already been cleaned in order to kill microorganisms that have survived the cleaning and/or equipment storage process. Steam, hot water and chemical sanitisation can be used in dairy plants and chemical sanitisers are commonly circulated through milk-handling equipment (Reinemann, 2003).

Dairy cleaning agents may be acidic or alkali compounds. The primary function of acidic compounds is to dissolve inorganic (mineral) deposits, while alkali compounds are used primarily to dissolve organic deposits (fat and protein). Other constituents are added to amplify the acid/alkali removal processes, for example, chlorine is often added to alkaline detergents as a peptising agent to aid in protein removal and to improve the rinse-ability of the detergent. Cleaning in place chemicals typically are caustic soda (at 0.8% strength) and nitric acid (0.6% strength).

A typical washing cycle of milking equipment and tankers consists of the following steps: immediate pre-rinse with clean cold water, a hot detergent wash using a caustic soda-based product , a second cold wash to remover all traces of detergent and finally a sanitising rinse with peracetic acid or hypochlorite, may be used.

The most commonly used disinfectants are chlorine-containing compounds, such as chlorhexidine and hypochlorite, as well as quaternary ammonium compounds (eg. benzalkonium chloride) and hydrogen peroxide. Iodophors are infrequently used in Australia nowadays (see Section 5.3.2).

Sanitisers can also potentially cause post-milking contamination of milk. However, residues of detergents and disinfectants/sanitisers in milk on the farm and at the dairy plant level are prevented by following HACCP monitoring programmes which ensure cleaning, disinfection, draining and rinsing procedures are carried out optimally. However, sanitizer contamination may potentially occur in milk and milk products at very low concentrations as indirect and incidental food contaminants (FSANZ 2005).

6. Potential risks from processing activities post farm gate

6.1 Biogenic amines

Biogenic amines (BAs) are low molecular weight organic bases, which result from the amino acid decarboxylase activity of microorganisms (Leuschner and Hammes, 1998); (Stratton *et al*., 1991). In cheese, BAs are produced during ripening as the casein is slowly degraded by proteolysis. Biogenic amines are classified as: aromatic biogenic amines (octopamine, dopamine, tyramine, serotonin, histamine, β-phenylethylamine and tryptamine); diamines (putrescine and cadaverine); and polyamines (agmatine, spemidine, and spermine). The characteristic structures of common BAs are shown in Figure 10.

The European Union, the U.S. FDA and several other countries have set regulatory levels in the range of 50 – 200 mg histamine/kg fish (EU, 2005); (Fletcher *et al*., 1998). In Australia, the Food Standards Code regulates histamine levels in fish and fish products, and the level of histamine must not exceed 200mg/kg.

Little is known regarding the toxicological dose of other BAs; for tyramine and phenylethylamine, upper limits of 100-800 mg/kg and 30 mg/kg have been set (Scheurer and Rödel, 1995). In rats, the no-observed adverse effect level for tyramine, putrescine and cadaverine was 180 mg/kg of body weight (Til *et al*., 1995).

Although cheese may contain exceptionally high levels of histamine and other BAs, (>2000 mg/kg), tolerance limits have not been set.

Hazard Identification and Characterisation

BAs occur in a wide variety of foods, such as fish, meat and cheese products, wine and other fermented foods (Izquierdo-Pulido *et al*., 1997). Amine production has been associated with protective mechanisms of microorganisms against acidic environments (Vanderkerckove, 1977) however human health problems may result from the ingestion of foods containing relatively high levels of certain BAs (Ekici *et al*., 2002); (Sancak *et al*., 2005)).

For example, “cheese syndrome” and histamine intoxication are related to increased tyramine and histamine levels respectively.

The adverse effects of these BAs include nausea, respiratory distress, heart palpitations, headache, hyper or hypotension and hypertensive crises due to the interaction with monoaminoxidase inhibitor drugs (MAOI) (Gonzalez de Llano *et al*., 1998), or in individuals with genetic or acquired diaminoxidase deficiency. These reactions can be potentiated by other BAs, such as putrescine, cadaverine, spermine and spermidine (Stratton *et al.,* 1991). Furthermore, in healthy individuals, the diamines, putrescine or cadaverine are not considered to be toxic, although they can potentiate the toxicity of histamine (Bardocz, 1995).

After fish, cheese is the second most commonly implicated food associated with histamine poisoning (Pinho *et al*., 2004). Most of the cases in which large amounts of amines are produced in cheeses have been attributed to lactic acid bacteria and E*nterobacteriaceae* (often used in starter cultures), with decarboxylating activity (Joosten and Northolt, 1987);(Sumner *et al*., 1985). In dairy products, there is a differential distribution and range of concentrations of BAs according to the type and source of that product (Table 28).

Ripened cheeses consistently show the highest levels of BAs, particularly tyramine, cadaverine and putrescine (e.g. maximum level of 611.7 mg/kg putrescine), whereas milk, curd, whey and unripened cheeses had no detectable BAs in many cases, regardless of whether goat or cow milk was tested. BA levels in rennet however were higher (e.g. 69.3 mg/kg tyramine) (Novella-Rodriguez *et al*., 2000); (Novella-Rodriguez *et al*., 2002).

Food poisoning, as a result of biogenic amine production, in particular histamine, is relatively well documented in fish (as summarised by (Lehane and Olley, 1999); OzFoodNet working group, 2002 and 2004; (ESR, 2001)), however there is only sparse mention made of outbreaks of food poisoning specifically due to BAs in dairy products (Sumner *et al.,* 1985); (Sancak *et al.,* 2005).

However, outbreaks of histamine-related food poisoning due to cheese have occurred in New Zealand. The incidences involving cheeses include Swiss, Cheddar, Gruyere and Cheshire cheese. Of the six cases of dairy-related histamine poisoning, the histamine content was 187 mg/100g, and was found to be due to control point failure, i.e. abnormally long storage period with possible incorrect temperature stability ((ESR, 2001)).

Recent research using double-blind placebo testing and open food challenges have shown that biogenic amines present in many cheeses can be a significant trigger of food intolerance reactions amongst patients presenting with symptoms such as urticaria, headaches and gastrointestinal problems (A.Swain and R. Loblay, Allergy Unit, RPA Hospital, Sydney, personal communication).

Dietary exposure

Different cheese types show widely varying BA concentrations, with higher levels for harder, mature cheeses (Table 30; (Novella-Rodriguez *et al.,* 2002); (Fernández-García *et al*., 2005)). This is a reflection of the degree of proteolysis and subsequent free amino acid levels (Sumner *et al.,* 1985), water activity, pH, NaCl concentration and microbiological profile (Novella-Rodriguez *et al.,* 2002); (Pinho *et al.,* 2004); (Galgano *et al*., 2001).

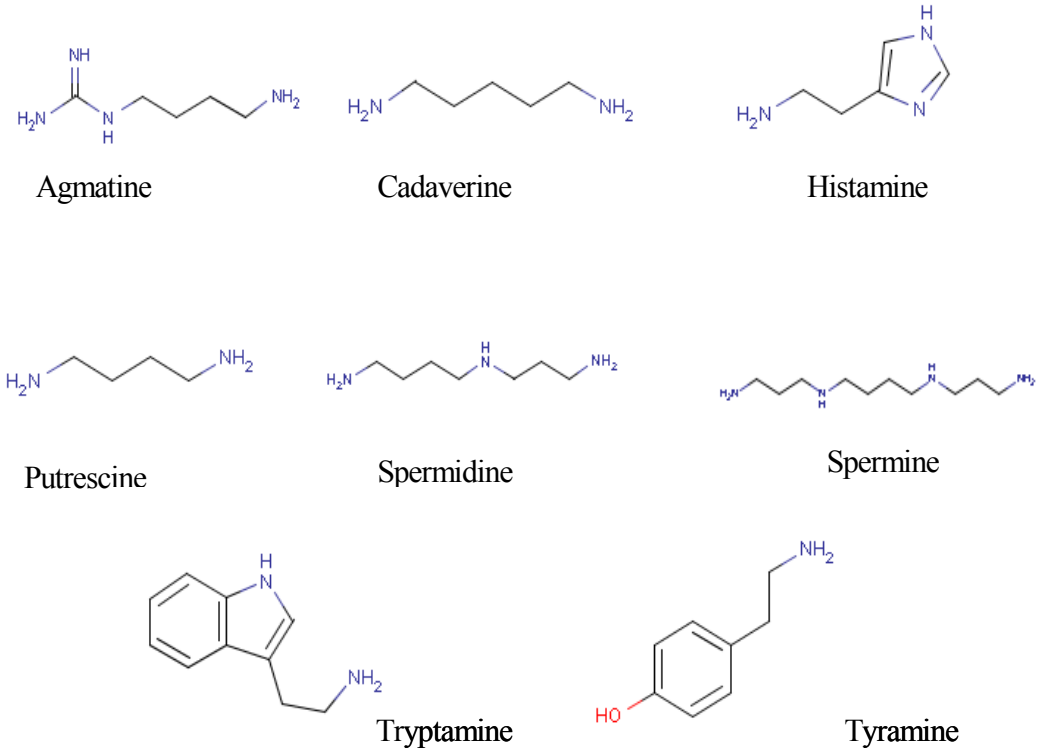


Figure 10: Chemical Structures of some biogenic amines detected in dairy products

Cheeses with comparable microbiological profiles also differ greatly in their BA concentrations (Schneller *et al*., 1997). In addition, the concentration of different BAs varies in different portions of the cheese; for example, at day 18 of ripening, cadaverine concentration is much higher in the rind of Camembert cheese than in the centre of the cheese (Engel *et al*., 2005).

In a comparison of raw-milk vs. pasteurised-milk semi-soft cheeses, the BA profile was also vastly different (Schneller *et al.,* 1997). Semi-soft cheeses produced from pasteurised milk showed much lower total BA concentrations compared to semi-soft cheeses made from raw milk (51 – 1096 mg/kg compared to 1011 – 3133 mg/kg).

The highest histamine concentration was found in a raw milk cheese (573 mg/kg); the highest total BA concentration (4817 mg/kg) was also detected in a raw-milk cheese that had been stored for 36h.

It has been reported that in order to elicit any symptoms of toxicity, according to the health and age of the individual, ingestion of 70 – 1000mg of histamine in a single meal is necessary (Taylor *et al*., 1982). It is further assumed that an intake of 100 mg histamine will usually evoke poisoning symptoms (Joosten, 1988).

Table 28: Biogenic amine content (mg/kg) in different commercial dairy products[1] (adapted from (Novella-Rodriguez *et al.,* 2000)

Biogenic amine	Milk [1]	Yoghurt[1]	Unripened cheese [1]	Ripened cheese[1]	Pasteurised milk[2]	Rennet[2]	Curd[2]	Whey[2]
TY[3]	nd	nd	nd – 0.51	nd – 241.9	nd	69.3	2.0	0.65
PU	nd	nd	nd – 1.43	nd – 611.7	nd	37.07	0.89	0.31
CA	nd	nd – 0.27	nd – 1.49	4.2 – 215.3	0.11	48.98	0.67	0.22
HI	nd	nd	nd	2.21 – 163.6	nd	10.47	0.98	0.28
TR	nd	nd	nd	nd – 45.1	nd	4.6	1.32	nd
PHE	nd	nd	nd	nd – 29.0	nd	5.16	nd	nd
AG	nd – 0.18	nd – 0.39	nd	nd – 22.0	nd	2.43	0.28	0.21
SD	0.16 – 0.18	nd – 0.43	0.39 – 0.82	nd – 43.0	0.35	4.38	1.77	0.24
SM	nd	nd – 0.34	nd – 1.12	nd – 18.7-	0.96	4.83	2.23	nd

[1] samples were taken from Spanish retail stores and represented different commercial brands
2 goats milk-derived products
[3]. TY = tyramine, PU = putrescine, CA = cadaverine, HI = histamine, TR = tryptamine, PHE = *β*-phenylethylamine, AG = agmatine, SD = spermidine, SM = spermine
[3] range (minimum – maximum) nd= not detected

Table 29: Biogenic amine contents of different cheeses

Cheese	Histamine (mg/kg)[3]	Tyramine (mg/kg)	Putrescine (mg/kg)	Cadaverine (mg/kg)	Reference
Traditional Turkish cheeses[1]	0 - 2180	-	-	-	(Ekici *et al*., 2002); (Sancak *et al.,* 2005)
Cheddar (mild)	1 – 108	-	-	-	(Antila *et al*., 1984)
Cheddar and other hard cheeses e.g. Emmenthal[2]	352 - 1720	23 - 520	18 - 254	21 - 254	(Aygün *et al*., 1999)
Swiss	nd – 2500	-	-	-	(Antila *et al.,* 1984)
Hispanico (hard Spanish cheese)	20	103 - 191	-	-	(Fernández-García *et al.,* 2005)
Roquefort and Blue	t - 409	-	-	-	(Antila *et al.,* 1984)
Soft cheeses (e.g. Camembert, Romadur)[2]	4 - 296	4 - 324	5 - 441	5 - 635	(Aygün *et al.,* 1999)
Edam and other semi-hard cheeses e.g. Gouda[2]	4 - 122	7 - 220	7 - 282	7 - 80	(Aygün *et al.,* 1999)
Edam (fresh)	4.0	-	-	-	(Antila *et al.,* 1984)
Edam (ripened).	1.7	-	-	-	(Antila *et al.,* 1984)
Montasio (semi-hard Italian cheese)	5.5 – 378.1	35 – 373.5	2.0 – 286.5	0.3 – 30.2	(Innocente and D'Agostin, 2002)
Cottage	nd	-	-	-	(Antila *et al.,* 1984)1984.

[1] Turkish cheeses included Beyaz, Kasar, Tulum, Civil and Otlu
[2] cheese samples purchased from food shops in SE Germany; hard cheeses (n=31); semi-hard cheeses (n=14) and soft cheese (n=5).
[3] range (minimum – maximum)
t = trace; nd = not detected; - = no data

Exposure to biogenic amines has not been investigated by FSANZ. Exposure to BAs from dairy products is most likely to be through eating hard, mature cheeses (Table 29), which comprise approximately 0.1 – 0.27% of the total diet up to the age of eighteen, and 0.05 – 0.07% of the adult diet (FSANZ, 2003).

Raw milk cheeses are also a source of BAs, but currently there is no information regarding exposure patterns in Australia.

Histamine is only monitored on a random basis in imported fish products. There was a recent incident related to tinned anchovies requiring a food recall in Australia; higher than acceptable levels of histamines were found in the product.

Risk Characterisation

Under certain conditions biogenic amines may be present in mature cheeses in high enough concentrations to induce toxic symptoms . However, the overall importance of dietary BAs as a potential human health risk is still somewhat controversial. For example, the production of BAs may be confused with allergenic symptoms too as the clinical symptoms of such pseudoallergenic reactions are indistinguishable from IgE-mediated allergenic reactions (Melnick *et al*., 1997). This may well lead to the under-reporting of dairy-related food poisoning due to biogenic amine production.

As a general conclusion, the levels of BAs in dairy products are safe for most people but there may be potential problems for high consumers and for some individuals due to intolerances induced by BAs. Finally, a complete risk characterisation cannot be carried out due to poor understanding of hazard characterisation and lack of data regarding exposure levels.

6.2 Fungal by-products

The public health and safety aspects of food-borne bacterial toxins, such as enterotoxins, Shiga toxins and Veroxytotoxins, have been addressed in the Microbiological Risk Profile. Fungal toxins such as gliotoxins, mycophenolic acid, PR-toxin, penitrem A, roquefortines, sterigmatocystin and cyclopiazonic acid (see section 3.2.3.1), which would most likely have originated from infected stockfeed or other environmental sources (i.e. water or air), have also been found in the moulds associated with cheese. A recent study on 122 cheese samples from goat and sheep milks, produced in Southern Italy, revealed high levels (44.3%) of contamination with potentially toxigenic species of *Penicillium*, *Aspergillus* and *Fusarium*, despite there being no superficial (sensory) signs of contamination. The most contaminated cheeses were the medium and long ripened samples (46.3% and 32.2% respectively), and the industrial cheeses (59.1%). The artisan cheeses were the least contaminated (26.8%) (Montagna *et al*., 2004). Although potential hazards associated with these toxins have been cited, in many cases lack of data on their occurrence in foods precludes a risk evaluation.

Toxins produced by *Penicillium roqueforti*

Penicillium roqueforti is a common saprophytic fungus that is widespread in nature and can be isolated from soil, decaying organic substances and plant parts. It is mainly used in the production of blue cheeses, such as Roquefort, Stilton and other blue cheeses. The U.S. Environmental Protection Agency has produced a final risk assessment on one of the key fungal toxins found in cheese, *P. Roqueforti*, which is summarised below (US EPA 2005).

There is considerable evidence to indicate that most strains of *P. roqueforti* are capable of producing harmful secondary metabolites (alkaloids and other mycotoxins) under certain growth conditions (Peberdy, 1985);(Sharpell, Jr., 1985). These mycotoxins include isofumigaclavin C, penicillic acid, PR toxin, patulin, botryodiploidin and roquefortine. The effects noted with ingestion of these mycotoxins are mutagenesis and tumorigenesis as well as extensive liver, kidney and nerve damage. Although there is a lack of documented cases of human toxicity, studies have shown that in the laboratory industrial strains of *P. roqueforti* can produce mycotoxins (Wei *et al*., 1985; Betina, 1989). However, the endpoints that are noted and the doses at which the effects are observed frequently are based on LD50 studies and omit references to No Observable Effect Level (NOEL) dosages (US EPA 2005). Amongst the toxins produced by *P. roqueforti,* roquefortine, PR toxin and mycophenolic acid are most commonly found in cheeses and subsequently more complete toxicological studies have been carried out on them.

Roquefortine

Hazard identification and characterisation

Roquefortine is an indole mycotoxin. It is produced by *P. roqueforti* and some other *Penicillium* species, namely *P. notatum*, *P. oxalicum*, *P. communi*, *P. corymbiferum*, *P. expansium* and *P. urticae* (Scott, 1984); (Arnold *et al*., 1987) reported an LD50 of 169 mg/kg in male and 184 mg/kg in female CR57 mice and 189 mg/kg in male and 184 mg/kg in female Swiss Webster mice.

Schoch (Schoch *et al*., 1984) conducted mutagenicity studies by the Ames test on six strains of *P. roqueforti* used commercially for the production of mould ripened cheese. Neither the fungus or roquefortine showed any mutagenic activity by the Ames test (Frank *et al*., 1977; Schoch *et al.,* 1984) fed a suspension of *P. roqueforti* to rats by gavage over their life span and showed that there was no evidence of a possible carcinogenic effect.

Dietary exposure

Low concentrations of roquefortine C were found in Roquefort type blue cheese by Ohmomo (Ohmomo *et al*., 1977) and Scott and Kennedy (Scott and Kennedy, 1976) found concentrations of roquefortine up to 6.8 mg/kg in samples of market blue cheese. Ware et al. (Ware *et al*., 1980) reported average levels of 0.42 mg/kg of roquefortine in 12 samples of blue cheese and of 0.045 mg/kg in two samples of blue cheese dressing. Roquefortine seems to be produced by most strains of *P. roqueforti* isolated from blue cheese or used as cheese starters (Scott *et al*., 1977).

Risk characterisation

It is unlikely that blue cheese is a potential acute human health hazard given the amounts of roquefortine present.

PR Toxin

Hazard identification and characterisation

PR toxin is one of the most acutely toxic metabolites known to be formed by *P. roqueforti* (Scott, 1981). The oral median lethal dose was 115 mg/kg. Within 10 minutes of an oral dose of about 10 mg (160 mg/kg) animals experienced breathing difficulties which persisted to death (Wei *et al*., 1973). Oral doses above about 130 to 160 mg/kg body weight were fatal to 60g rats in 36 hours or less.

Gross pathology consisted of swollen, gas filled stomach and intestines, while histological changes included congestion and oedema of lung, brains and kidney with degenerative changes in liver and kidney and haemorrhage in the kidney as well.

Chen et al. (Chen *et al*., 1982) studied the toxic effects of PR toxin in a range of animals. Toxic effects in mice and rats included abdominal writhing, decrease of motor activity and respiration rate, weakness of the hind leg and ataxia. It was concluded that PR toxin produced acute toxic effects in animals via an increase of capillary permeability and direct damage to lungs, heart, liver, and kidneys. Despite clear toxicological effects of ip introduced PR toxin, rats administered 0.5 mg PR toxin orally procapite/prodic for two months showed no visible effect. Mutagenicity of PR Toxin was demonstrated by Ueno et al. (Ueno and Ueno, 1978). Polonelli et al. (Polonelli *et al*., 1982) carried out preliminary studies on possible carcinogenic effects of PR toxin in rats, however the results were inconclusive.

The acute toxicities of the PR derivatives were considerably lower than that of the parent compound (Scott and Kanhere, 1979). They conclude that both PR toxin and PR imine are unstable in blue cheese and believe that the agents responsible for destruction of PR toxin formed during ripening of the blue cheese are most likely amino compounds. PR toxin enters into reactions involving its aldehyde function to form crosslinks between DNA and protein (Moulé *et al*., 1980). It also inhibits in vitro transcriptional capacity of nuclei isolated from the liver of male Wistar rats when the compound is administered in vivo. The toxin inhibited both the RNA polymerase systems responsible for ribosomal RNA synthesis and heterogenous nuclear RNA synthesis. PR toxin inhibited the in vitro activities of rat liver DNA polymerases alpha, beta and gamma, as well (Moulé *et al.,* 1980); (Lee and Wei, 1984).

Exposure Assessment

PR toxin has been reported in cheese, mouldy grains and silage (EMAN 2005) although little data is available regarding the levels found.

Risk Characterisation

It is unlikely that PR toxin in cheese is a potential acute human health hazard given that t it is unstable.

Mycophenolic Acid

Mycophenolic acid is a metabolite produced by many strains of *P. roqueforti* and by a few other species of *Penicillium* (La Font et al., 1979). Although Engel et al. (Engel *et al.,* 2005)only found that 25% of all *P. roqueforti* strains produce mycophenolic acid. It has antibiotic activity against bacteria and dermatophytic fungi and also interferes with viral multiplication (Planterose, 1969). Mycophenolic acid is also used in liver transplantation as an immunosuppressive agent.

Hazard identification and characterisation

The toxicity of mycophenolic acid for mammals appears to be low: LD50 in rats is 2,500 mg/kg and 500 mg/kg IV; in mice the LD50 is 700 mg/kg and 450 mg/kg IV (Wilson, 1971). The oral LD50 of 700 mg/kg in mice placed mycophenolic acid in the U.S. Environmental Protection Agency's moderately toxic category. Chronicity tests of daily oral doses of 80 and 320 mg/kg for one year did not cause apparent signs of toxicity in rabbits (Adams *et al*., 1975). However, rats given daily oral doses of 30 mg/kg died within 9 weeks and rhesus monkeys receiving 150 mg/kg daily developed abdominal colic, bloody diarrhoea, weight loss and anaemia after two weeks (Carter *et al*., 1969).

Thirty-five human patients who received high oral doses of mycophenolic acid (2.4 g to 7.2 g daily) had some adverse reactions, including cramps, nausea and diarrhoea, and mutations were induced in a mouse mammary carcinoma cell line with mycophenolic acid (Marinari *et al*., 1977); (Umeda *et al*., 1977).

Exposure Assessment
Mycophenolic acid has been reported in cheese (EMAN 2005), and in particular in blue cheese (Lafont *et al*., 1979) where levels of up to 4mg/g dry culture was determined.

Risk Characterisation
A full risk characterisation cannot be carried out due to lack of data, but there is considered to be low toxicity in mammals.

In summary, the risk associated with *P.roqueforti* lies with its production of a range of mycotoxins, which have been studied to varying degrees. Some of these mycotoxins have been shown to be produced by *P.roqueforti* strains used for cheese production and some have been detected in small amounts in the cheese itself. PR toxin and roquefortines appear to be the most toxic of the mycotoxins produced by *P.roqueforti*. PR toxin, one of the most potent mycotoxins, is unstable and deteriorates rapidly, so apparently under normal production conditions it does not pose a health risk. Roquefortine has been recovered from blue cheese at low levels and there have been no reported adverse effects from consumption of the cheese. The composition of medium used to make cheese and the length of time and conditions of the fermentation lead to highly variable results with respect to the composition and amounts of mycotoxins produced. In general, mycotoxins are produced in media with a high carbon to nitrogen ratio.

6.3 High heat treatment

6.3.1 Polycyclic aromatic hydrocarbons

The term 'polycyclic aromatic hydrocarbons' (PAHs) commonly refers to a large class of organic compounds containing two or more fused aromatic rings made up of carbon and hydrogen atoms. PAHs are soluble in many organic solvents and are highly lipophilic. They are chemically rather inert (IPCS 1998a; IPSC, 1998b).

Raw food does not normally contain high levels of PAHs. Processing procedures, such as smoking and drying, and cooking of food is commonly thought to be the major source of contamination by PAH (SCF, 2002d; SCF, 2002b). Depending on a number of parameters: time, fuel used, distance from the heat source and drainage of fat, type of cooking (grilling, frying, roasting), cooking results in the production in the food of a number of compounds including PAHs.

Hazard Identification and Characterization
The acute toxicity of PAHs is moderate to low. The well characterized PAH, naphthalene, showed oral and intravenous LD_{50} values of 100-500 mg/kg bw in mice and a mean oral LD_{50} of 2700 mg/kg bw in rats. The values of other PAH are similar (IPCS 1998b).

PAHs have been studied extensively in assays for genotoxicity and cell transformation; most PAHs are positive in some genotoxicity assays. The only compounds for which negative results were found in all assays were anthracene, fluorene and naphthalene.

Owing to inconsistent results, phenanthrene and pyrene could not be reliably classified for genotoxicity.

Comprehensive work on the carcinogenicity of PAHs shows that 17 of 33 studied are, or are suspected of being carcinogenic. Only benzo-[a]-pyrene has been adequately tested using dietary administration (SCF, 2002a).

In humans the majority of studies available have examined occupational exposure to PAHs via inhalation, and in a few studies, via dermal exposure. Most of the reports are on exposure to mixtures of PAHs, which also contained other potentially carcinogenic chemicals, in occupational or environmental situation (SCF, 2002c).

Dietary exposure

FSANZ does not have data regarding the exposure of the Australian population to PAHs. The intake of individual PAH from food has been estimated to be 0.10-10 µg/day per person. Cereals and cereal products are the main contributors to the intake of PAH from food because they are a major component of the total diet (IPCS 1998a; IPSC, 1998b). A Swedish study has found that smoked and grilled foods show the highest PAH levels though they make only a modest contribution to total PAH dietary intake, since they are minor components of the usual diet (Larsson, 1986; IPSC, 1998b; IPCS 1998b). However, it should be noted that smoked and grilled food may contribute significantly to the intake of PAH if such foods are part of the usual diet.

PAH in dairy products have been detected in milk (Cavret *et al*., 2005) and in smoked cheese (e.g. (Osborne and Crosby, 1987)). For example, the benzo pyrene content of a smoked Italian Provola cheese was 1.3 µg/kg (Lintas *et al*., 1979), and concentrations of 0.01 – 5.6 µg/kg fresh weight fluranthene, benz anthracene, benzo phenanthrene, benzo pyrene, benzo perylene and indeno pyrene were found in a smoked cheese sample. PAH have also been found in the U.K. in unsmoked cheeses at levels of 0.01 – 0.06 µg/kg (McGill *et al*., 1982) and in British butter and cream samples (Dennis *et al*., 1991). PAHs have also been associated with ash (Yoshida R *et al*., 2003) and therefore may potentially be present in ash cheeses.

Risk Characterisation

Data linking dietary exposure of polycyclic aromatic hydrocarbons to possible human health risks are inconclusive. Some PAH are likely to be genotoxic carcinogens – with no known level of safe exposure. Estimated average dietary exposure for the Australian population is unavailable. Exposure is expected to be highly variable and linked to processing practices however, overall exposure from food is likely to be low. Though there is potential risk due to carcinogenic properties of some PAH's, particularly benzo[a]pyrene and as such exposure should be as low as reasonably achievable, the contribution of PAHs in the diet to the development of human cancer is not considered to be high (IPCS 1998b)

In conclusion, dietary exposure from the consumption of PAH in dairy products represents a negligible risk to the consumer.

7. Chemicals used in further processing

Further processed food products can utilise a range of chemicals such as food additives, processing aids and packaging options to create niche market products. The Standards applicable to the regulation of chemical used in further processed dairy products include;

- Standard 1.3.1 – Food Additives
- Standard 1.3.3 – Processing Aids
- Standard 1.3.4 – Identity and Purity
- Standard 1.4.3 – Articles and Materials in Contact with Food

7.1 Food Additives

Food additives are commonly used in processed dairy products. FSANZ regulates food additives through Standard 1.3.1 – Food Additives. A food additive is any substance not normally consumed as a food in itself and not normally used as an ingredient of food, but which is intentionally added to food to achieve one or more of the technological functions specified in Table 30. A food additive, or its by-products, may remain in the food.

Food additives should always be used in accordance with GMP. As a guide to assist manufacturers in compliance with this provision, the standard cites the Codex Alimentarius Commission Procedural Manual (CAC, 1999), which sets out the following relevant criteria for use in assessing compliance with GMP:

(a) the quantity of additive added to food shall be limited to the lowest possible level necessary to accomplish its desired effect;
(b) the quantity of the additive that becomes a component of food as a result of its use in the manufacture, processing or packaging of a food and which is not intended to accomplish any physical, or other technical effect in the finished food itself, is reduced to the extent reasonably possible; and,
(c) the additive is prepared and handled in the same way as a food ingredient.

Substances added to food in accordance with the Code must also meet appropriate specification for identity and purity. Standard 1.3.4 – Purity and Identity – details the specifications for permitted food additives. A substance must comply with a reference in;

(a) Food and Nutrition Paper 52 Compendium of Food Additive Specifications Volumes 1 and 2, including addenda 1 to 9, published by the Food and Agriculture Organisation of the United Nations in Rome (1992); or
(b) the fourth edition of the Food Chemicals Codex published by the National Academy of Sciences and the National Research Council of the United States of America in Washington, D.C. (1996), including supplements published to take effect on 1 December 1997, 31 March 2000 and 31 December 2001; or
(c) the Schedule to this Standard.

If no relevant specifications exists in one of these documents, a secondary tier of reference documents comprising other recognised national standards or pharmacopoeia.

Table 30: Technological functions which may be performed by food additives

Functional class *sub-classes*	Definition
Acidity regulator acid, alkali, base, buffer, buffering agent, pH adjusting agent	alters or controls the acidity or alkalinity of a food
Anti-caking agent anti-caking agent, anti-stick agent, drying agent, dusting powder	reduces the tendency of individual food particles to adhere or improves flow characteristics
Antioxidant antioxidant, antioxidant synergist	retards or prevents the oxidative deterioration of a food
Bulking agent bulking agent, filler	contributes to the volume of a food without contributing significantly to its available energy
Colouring	adds or restores colour to foods
Colour fixative colour fixative, colour stabiliser	stabilises, retains or intensifies an existing colour of a food
Emulsifier emulsifier, emulsifying salt, plasticiser, dispersing agent, surface active agent, surfactant, wetting agent	facilitates the formation or maintenance of an emulsion between two or more immiscible phases
Firming agent	contributes to firmness of food or interact with gelling agents to produce or strengthen a gel
Flavour enhancer flavour enhancer, flavour modifier, tenderiser	enhances the existing taste and/or odour of a food
Flavouring (excluding herbs and spices and intense sweeteners)	intense preparations which are added to foods to impart taste and/or odour, which are used in small amounts and are not intended to be consumed alone, but do not include herbs, spices and substances which have an exclusively sweet, sour or salt taste.
Foaming agent Whipping agent, aerating agent	facilitates the formation of a homogeneous dispersion of a gaseous phase in a liquid or solid food
Gelling agent	modifies food texture through gel formation
Glazing agent coating, sealing agent, polish	imparts a coating to the external surface of a food
Humectant moisture/water retention agent, wetting agent	retards moisture loss from food or promotes the dissolution of a solid in an aqueous medium
Intense sweetener	replaces the sweetness normally provided by sugars in foods without contributing significantly to their available energy
Preservative anti-microbial preservative, anti-mycotic agent, bacteriophage control agent, chemosterilant, disinfection agent	retards or prevents the deterioration of a food by micro organisms
Propellant	gas, other than air, which expels a food from a container
Raising agent	liberates gas and thereby increase the volume of a food
Sequestrant	forms chemical complexes with metallic ions
Stabiliser binder, firming agent, water binding agent, foam stabiliser	maintains the homogeneous dispersion of two or more immiscible substances in a food
Thickener thickening agent, texturiser, bodying agent	increases the viscosity of a food

A review of the technological functions regulated in Standard 1.3.1 indicates some functional classes, such as propellants, intense sweeteners and raising agents are unlikely to be relevant to dairy products. The Standard, through Schedule 1, have specified permitted uses of food additives by food type for dairy products. The permissions for dairy products relate mainly to preservative and colouring functions.

There is anecdotal evidence that small dairy manufacturers may add homeopathic chemicals to their dairy produce, for example, cider vinegar and plant extracts; in general these are unregulated products.

Benzoic acid

Benzoic acid is one of the oldest chemical preservatives used in the cosmetic, drug and food industries. It occurs naturally at low levels (~0.2 mg/kg) in a range of foods including dairy products (milk, cheese, yoghurt) (IPCS 2000a).

Although benzoic acid is not approved for use as an additive in the manufacture of dairy products, other than dairy (and other fat) based deserts, there is potential for the natural levels of benzoates to concentrate in fermented dairy products. This could potentially be a problem when exporting a dairy product to a country that prohibits the use of benzoic acid as a preservative.

Hazard Identification

Benzoates were evaluated by JECFA in 1996 (WHO, 1997), where the ADI for benzoic acid and its calcium, potassium and sodium salts, expressed as benzoic acid equivalents, of 0-5 mg/kg bw was maintained.

The ADI of 0-0.5 mg/kg bodyweight established by JECFA for benzoic acid and its salts is based on a long-term exposure study in rats. The NOEL was established at the highest dose tested (500 mg/kg bodyweight per day) where no adverse effects were observed. Signs of toxicity were observed in more recent short-term studies at higher dose levels. In establishing the ADI, a safety factor of 100 was applied to the NOEL to take into account species differences and individual human variation.

Dietary exposure

A dietary exposure assessment was conducted as part of the assessment of benzoates for the 21st ATDS (FSANZ, 2005c). The mean estimated dietary exposure to benzoates was less than 50% of the ADI for all population groups assessed. The mean estimated dietary exposure for the population aged two years and over, representing mean lifetime exposure, was approximately 15% of the ADI for males and approximately 10% of the ADI for females. The 95th percentile estimated dietary exposures to benzoates exceeded the ADI for young boys (approximately 140%) and young girls (approximately 120%) aged 2-5 years, and was equivalent to the ADI for schoolboys aged 6-12 years. All other population groups were below the ADI for 95th percentile estimated dietary exposures. The 95th percentile estimated dietary exposure to benzoates for the population aged two years and over, representing lifetime exposure for a high consumer of benzoates, was approximately 60% of the ADI for males and approximately 50% of the ADI for females.

The major foods contributing to dietary exposure to benzoates for young children aged 2-5 years were cordial, non-cola soft drinks and orange juice. For all other age groups assessed, non-cola soft drinks were the greatest contributor to dietary exposure to benzoates. In the 21st Australian Total Diet Survey benzoic acid was detected in various cheeses (3-11 mg/kg), while there was no analysis for milk.

Risk characterisation

The mean estimated dietary exposure to benzoates for all population groups was well below the ADI, indicating that for the majority of the population, there is no public health and safety risk from the consumption of a balanced diet which includes foods containing benzoates.

Dairy products are not a major contributor to the overall exposure of benzoic acid and therefore, benzoic acid dietary exposure from the consumption of dairy products presents a negligible risk to the consumer.

7.2 Processing Aids

Substances can be used in the processing of foods to fulfil a technological purpose relating to a treatment or process, but do not perform a technological function in the final food. For the purposes of the Code these substances are known as processing aids. Examples relevant to dairy products include the use of hydrogen peroxide.

Processing aids are regulated through Standard 1.3.3 – Processing Aids. For the purposes of the Standard a processing aid is a substance used;

(a) in the processing of raw materials, foods or ingredients, to fulfil a technological purpose relating to treatment or processing, but does not perform a technological function in the final food; and
(b) in the course of manufacture of a food at the lowest level necessary to achieve a function in the processing of that food, irrespective of any maximum permitted level specified.

Unless expressly permitted in this Standard, processing aids must not be added to food.

Similarly to food additives, the quality of processing aids is regulated through provisions in Standard 1.3.4 – Purity and Identity. Chemicals used as processing aids listed in the Standard 1.3.3 – Processing Aids - are grouped by functional classes. Permitted usage by functional classes include:

- Generally permitted processing aids;
- Antifoaming agents;
- Catalysts;
- Decolourants, clarifying and filtration agents;
- Desiccating preparations;
- Ion exchange resins;
- Lubricants, release and anti-stick agents;
- Carriers, solvents and diluents;
- Processing aids permitted in packaged water used as an ingredient in other foods;
- Bleaching agents, washing and peeling agents;
- Extraction solvents;
- Processing aids with miscellaneous functions;
- Enzymes of animal origin;
- Enzymes of plant origin;

- Enzymes of microbial origin; and,
- Microbial nutrients and microbial nutrient adjuncts.

The Processing Aid Standard is currently under review (Proposal P276 Review of Enzyme Processing Aids and Proposal P277 – Review of Processing Aids (other than enzymes). The review will address the following:

- safety of currently permitted processing aids;
- removing any obsolete processing aids; and
- correct errors, remove anomalies and improve consistencies within the Code.

It is not anticipated that the structure of Standard 1.3.3 – Processing Aids - will be changed.

The review of Standard 1.3.3 might result in some changes which could be relevant for the Dairy Standard, but is not expected to have a major impact.
The regulation of certain processing aids, for example hydrogen peroxide, benzoyl peroxide and lactoperoxidase system are further clarified below.

Hydrogen peroxide
Hydrogen peroxide is a very effective bactericidal agent (Fox *et al*., 2000). In some European countries and America, hydrogen peroxide is used as an alternative to pasteurisation in certain hard cheeses, though the use is not practiced commercially to any great extent. There is some evidence for the efficacy of using hydrogen peroxide treatments to inactivate penicillin residues in milk (Hanway *et al*., 2005).

The World Food and Agriculture Organisation permit addition of hydrogen peroxide to milk at concentrations of 0.05 – 0.25%, on the condition that all the hydrogen peroxide remaining in the milk after processing is converted by catalase into O_2 and H_2O (Tarhan, 1994).

In Australia, hydrogen peroxide (H_2O_2) is an approved processing aid (Standard 1.3.3 – Processing Aids – Table to clause 12) and may be used as a bleaching, washing and peeling agent in the course of manufacture of all food with a maximum permitted residue level of 5 mg/kg (0.005%). It is not, however, permitted for use as a preservative in Standard 1.3.1. As a processing aid, the hydrogen peroxide may be used to fulfil a technological purpose relating to treatment or processing, but not to perform a technological function in the final food. Thus, there is no permission for the use of hydrogen peroxide as a chemical alternative to pasteurisation in the Australian dairy industry, even though in the past, in a few instances State Dairy Authorities have identified the use of hydrogen peroxide in both cream and brine and appropriate corrective/enforcement actions were taken.

<u>Hazard identification and characterisation</u>
The dissociation of hydrogen peroxide is a violent and exothermic reaction. The systemic effects of hydrogen peroxide result from its interaction with catalase in the tissues with liberation of oxygen and water as it decomposes. Ingestion results in gastrointestinal irritation, the severity of which depends on the concentration. There is also a risk of gas embolism. A number of deaths have been reported in the literature. In most cases the exposure were to concentrated solutions of 30 to 40% (IPCS, 1998; IPSC, 1998a).

Oral ingestion of 3% hydrogen peroxide solutions (household strength) generally does not result in severe toxicity but may result in vomiting, mild irritation to mucosa and burns in the mouth, throat, oesophagus and stomach. Ingestion of higher concentration, e.g. >10%, can result in more dangerous sequelae such as burns to mucus membranes and gut mucosa (ATSDR, 2004).

Most cases of ingestion of hydrogen peroxide result in only mild effects. Persistent exposure to low levels of hydrogen peroxide is unlikely to cause chronic toxicity as hydrogen peroxide is rapidly detoxified in the body.

International Agency for Research on Cancer (IARC) considered that there was inadequate evidence for carcinogenicity in human (Group 3) (IARC, 1999).

The Joint FAO/WHO Expert Committee on Food Additives (JECFA) had evaluated the safety of hydrogen peroxide in 1965, 1973 and 1980 respectively. JECFA considered that ingestion of small amounts of hydrogen peroxide, that may be present in food, would produce no toxicological effects due to rapid decomposition of the chemical by the enzyme catalase of the intestinal cells (FAO, 1967; WHO, 1974; WHO, 1980). No ADI was allocated.

The antimicrobial effects and limitations of hydrogen peroxide use have been summarised in a report commissioned by FSANZ (Juffs, H and Deeth, H, 2005).

Lactoperoxidase system

Lactoperoxidase is a porphyrin-containing peroxidase secreted by the mammary gland. Lactoperoxidase catalyses the oxidation of thiocyanate (SCN^-) to hypothiocyanate by using hydrogen peroxide.

The lactoperoxidase system (LPS) is a major contributor to the antibacterial activity of milk (Kussendrager and Van Hooijdonk, 2000). The antibacterial system functions by the generation of oxyacids that react with protein sulfhydryls in bacterial cell walls, essentially terminating metabolism. LPS has been shown to function effectively as a bacteriocide against several Gram negative bacteria and is bacteriostatic against Gram positive bacteria (Aimutis, 2002).

In bovine milk, lactoperoxidase is the second most abundant enzyme (after xanthine oxidase) and its concentration is approximately 30 mg/l, constituting about 0.5% of the whey proteins. Unlike other anti bacterial proteins, lactoperoxidase levels are relatively low in colostrums, but increase to a maximum level 3-5 days postpartum. In order for this naturally occurring enzyme to exert an antibacterial effect, it requires the presence of both hydrogen peroxide and thiocyanate.

LPS is used in some instances in the preservation of dairy products; it's function is enhanced in the presence of thiocyanate and glucose oxidase (Van Hooijdonk *et al*., 2000).
Bovine lactoperoxidase is thermostable and is not affected by pasteurisation.
The International Dairy Federation has published guidelines for preservation of milk using the lactoperoxidase system.

LPS is not an approved sanitising agent for Australian dairy products, although bovine derived lactoperoxidase is permitted in Standard 1.3.3 as a processing aid for meat surfaces. Codex Alimentarius allows for the use of LPS in countries where pasteurisation and refrigeration facilities are not available for milk processing and storage. The Codex Alimentarius Commission does not consider LPS to be of toxicological concern at the prescribed levels for those countries where it is permitted for use.

Benzoyl peroxide
Benzoyl peroxide (measured as benzoic acid) releases hydrogen peroxide and is permitted in Standard 1.3.3 as a processing aid for bleaching, washing and as a peeling agent to a maximum level of 40 mg/kg. Benzoyl peroxide is not permitted for use as a food additive in Australian dairy products.

Benzoyl peroxide is permitted in the USA as a bleaching agent to a maximum level of 100 mg/kg for whey powder, and as such may be found as a constituent in imported products from the U.S.A, if the level in the final food does not exceed 40 mg/kg; there are no risks associated with its consumption at the permitted level.

7.3 Packaging

The major role of dairy packaging is to retard product deterioration by preventing microbial recontamination and excessive chemical deterioration. Fluid products are especially susceptible to microbial, enzymatic and chemical spoilage. In addition, packaging must also provide containment, facilitate use, identify products and appeal to customers.

Protection of product flavour
A specific issue relating to packaging and dairy products is the ability of dairy products to adsorb taints from primary packaging (in contact with the product) and from contaminating chemicals disseminated in the surrounding air-space of a dairy product.
Packaging can allow the transfer of odours such as stored food (e.g. onions) or distribution odours (e.g. diesel or fresh paint fumes). Packaging made from substandard materials or overheated during formation can transfer odours to products. For example, inks can transfer solvent odours to products and polymer-based materials can transfer plastic-like odours if over heated. In addition, exposure to light can cause the formation of "light-struck" off-flavours (Hotchkiss and Meunier-Goddik, 2003).

Regulation of packaging materials
FSANZ regulates food contact uses of primary packaging materials through Standard 1.4.3 – Articles and Materials in Contact with Food. The Standard regulates food contact materials in general terms. The Standard does not specify individual packaging materials for food contact or how they are produced or used. With respect to plastic packing products, the standard refers to the Australian Standard for Plastic Materials for Food Contact Use, AS 2070-1999. This reference provides a guide to industry about the production of plastic materials for food contact use. AS 2070, in turn, refers to regulations of the United States of America (USA) and European Economic Community (EEC) directives relevant to the manufacture and use of plastics.

Where a public health and safety concern is identified, maximum levels may be established in Standard 1.4.1 – Contaminants and Natural Toxicants. Examples include the maximum levels set for tin (all canned food), acrylonitrile and vinyl chloride (all food) in association with packaging materials.

Currently AS 2070 prohibits the use of recycled plastic materials in plastic materials for food contact use therefore only virgin plastics are used for packaging in Australia.

Migration of chemicals into dairy produce

Although prescription of specific plastic packaging is beyond the scope of the *Australia New Zealand Food Standards Code*, it is pertinent to note that there has been evidence of the migration of chemicals from packaging used to wrap dairy produce.

Cling-film is used both for wrapping foods at the retail level (for example, cheese) as well as for use in the home for a variety of applications. Plasticisers are added to polymers such as PVC to confer different degrees of flexibility in the production of cling-film. Plasticisers are mainly highly lipophillic, organic esters of low molecular weight with a potential to migrate from the packaging material into the packaged food, thereby becoming contaminants. Two such plasticizers are di-(2-ethylhexylexyl)adipate (DEHA) and di-(2-ethylhexyl)phthalate (DEHP). DEHA has replaced the phthalates in thin plasticized PVC food packaging films, due to reports of the induction of testes toxicity and antiandrogenic effects of DEHP (Dalgaard *et al*., 2003). The EU states that DEHA has low acute toxicity, is not genotoxic and does not cause irritation.

Although plasticisers are not an issue in Australia, in other countries, both DEHA and DEHP have been reported as contaminants of dairy produce (Page and Lacroix, 1995); (Castle *et al*., 1987). In a U.K. survey, milk and cream showed very low levels of DEHP (< 0.01 – 3 mg/kg), whereas cheese and butter levels were as high as 114 mg/kg total phthalate in some cases (Sharman *et al*., 1994). The migration of DEHA from food-grade PVC film, containing 28.3% DEHA, into hard and soft cheeses was found to be dependent upon contact time, fat and moisture contents, and consistency of the cheese samples (Goulas *et al*., 2000). The presence of cheese rind also greatly reduced the migration of DEHA in Edam and Kefalotyri (Goulas *et al.,* 2000).

Internationally there are intermittent reports of the migration of other chemicals from food packaging. For example, during the past year there were reports from Europe that the chemical isopropylthioxanthone, commonly used in printing ink on some food packaging, was also present at very low levels in some liquid infant formula products (EFSA, 2005).

7.4 Distribution and Transport

After manufacture, dairy products remain vulnerable to chemical contamination from the environment and containers. Cross-contamination of taints (FSANZ 2005) and traces of chemicals from other foods during transport of mixed loads or from lubricants, refrigerants or paint, for example, may occur during transport. Furthermore, as milk and dairy products are perishable, they must be transported without undue delay to prevent the introduction of contaminants and the growth of toxin-producing pathogens.

Food Safety Programs are in place in order to manage potential contamination during distribution. Thus dairy food transport vehicles, equipment and vessels are designed, constructed and maintained to prevent the introduction of contaminants and temperature increase (that is, maintaining a temperatures $\leq 5^{o}C$). Cleaning and sanitising of dairy food carriers is a key aspect of the Food Safety Program, which relies upon the use of water of suitable quality.

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GLOSSARY

ADASC	Australia New Zealand Dairy Authorities' Standards Committee
ADI	Acceptable Daily Intake
AgVet	Agricultural and Veterinary
AMR	Antimicrobial Resistance
AMRA Survey	Australian Milk Residue Analysis Survey
APVMA	Australian Pesticide and Veterinary Medicines Authority
AQIS	Australian Quarantine and Inspection Service
ARGT	Annual Ryegrass Toxicity
ARPANZA	Australian Radiation Protection and Nuclear Safety Agency
ATDS	Australian Total Diet Survey
BA	Biogenic Amine
BST	Bovine Somatotropin
CCU	Central Coordinating Unit
CIJIG	Commonwealth Interdepartmental JETACAR Implementation Group
Codex	Codex Alimentarius Commission
CPA	Cyclopiazonic acid
1,2-DCB	1,2-dichlorobenzene
DEHA	Di-(2-ethylhexyl)adipate
DEHP	Di-(2-ethylhexyl)phthalate
DFSV	Dairy Food Safety Victoria
DON	Deoxynivalenol (trichothecene mycotoxin)
EAGAR	Expert Advisory Group on Antimicrobial Resistance
EC	European Commission
EEC	European Economic Community
EPA	Environmental Protection Agency (US)
ERL	Extraneous Residue Limit
EU	European Union
EU SCOOP	European Union Scientific Cooperation
FAO	Food and Agriculture Organization
FDA	Food and Drug Administration (US)
FSANZ	Food Standards Australia New Zealand
GAP	Good Agricultural Practise
GM	Genetically Modified
GMP	Good Manufacturing Practice
GPT	*N*-acetylglucosamine-1-phosphate transferase

HACCP	hazard analysis critical control point
HCB	Hexachlorobenzene
HDPE	High Density Poly Ethylene
HGP	Hormonal Growth Promotants
IARC	International Agency for Research on Cancer
IV	intravenous
JECFA	Joint FAO/WHO Expert Committee on Food Additives
JETACAR	Joint Expert Technical Advisory Committee on Antibiotic Resistance
JMPR	Joint Meeting on Pesticide Residues
LD_{50}	Lethal Dose for 50% of the experimental animals tested
LDPE	Low-density Poly Ethylene
LOEL/LOAEL	Lowest Observed Effect Level/Lowest Observed Adverse Effect Level
LOD	Limit of Detection
LOQ	Limit of Quantification
LOR	Limit of Reporting
LPS	Lactoperoxidase System
ML	Maximum Level
MOU	Memorandum of Understanding
MRL	Maximum Residue Limit
NARM Program	National Antibacterial Residue Minimisation Program
NDP	National Dioxins Program
NHMRC	National Health and Medical Research Council
NIV	Nivalenol (trichothecene mycotoxin)
NOEL	No Observed Effect Level
NORM Program	National Organochlorine Residue Management Program
NRA	National Registration Authority
NRS	National Residue Survey
OECD	Organization for Economic Co-operation and Development
OGTR	Office of the Gene Technology Regulator
OIE	World organisation for animal health
PA	Pyrrolizidine Alkaloid
PAH	Polycyclic Aromatic Hydrocarbon
PBDD	Polybrominated dibenzodioxins
PBDF	Polybrominated dibenzofurans
PCB	Polychlorinated biphenyls
PCDD	Polychlorinated dibenzodioxins
PCDF	Polychlorinated dibenzofurans

PET	Poly ethyleneterephthalate
PISC	Primary Industries Standing Committee
PMTDI	Provisional Maximum Tolerable Daily Intake
PPP	Primary Production and Processing
PR	Penicillium Roqueforti
PTDI	Provisional Tolerable Daily Intake
PTWI	Provisional Tolerable Weekly Intake
PVC	Polyvinylchloride
QA	Quality Assurance
rBST	Recombinant Bovine Somatotropin
SCF	Scientific Committee on Food
SDA	State Dairy Authority
TCDD	2,3,7,8-tetrachlorodibenzo-*p*-dioxin
TDI	Tolerable Daily Intake
TEF	Toxicity Equivalency Factor
TEQ	Toxic Equivalence
TI	Tolerable Intake
TMI	Tolerable Monthly Intake
UL	Upper Level of Intake
USA	United States of America
VRE	Vancomycin Resistance Enterococci
WHO	World Health Organization
WHP	Withholding Period
ZEA	Zearalenone
ZOL	Zearalenol