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

SURVEY OF POLYCYCLIC AROMATIC HYDROCARBONS (PAH) IN AUSTRALIAN FOODS

DIETARY EXPOSURE ASSESSMENT AND RISK CHARACTERISATION

EXECUTIVE SUMMARY

Food Standards Australia New Zealand (FSANZ) commissioned an analytical survey of Polycyclic aromatic hydrocarbons (PAH) in Australian foods and the results have been used in a dietary exposure assessment and health risk appraisal for the Australian population. Based on the available data, and taking into account the inherent uncertainties and limitations, this study indicated that the health risk to the Australian public arising from dietary exposure to PAH is unlikely to be of public health and safety concern.

PAH are naturally occurring compounds found in the environment. They result from natural occurrences such as volcanic activity and bush fires. They are also produced by industrial processes. PAH contamination has been identified in the air, water and food sources. Furthermore, PAH are also produced by some cooking processes, particularly through barbequing, smoking, roasting and frying. PAH contamination in the environment and food has been of world-wide focus due to the potential hazards high levels of these compounds can produce. Many countries have studied the levels of carcinogenic PAH in food samples in an attempt to conduct a risk assessment and determine the level of exposure.

In this survey, a total of 35 foods were examined covering a broad spectrum of foods included in a typical diet. This included foods from the following groups: dairy, meat, vegetables, bread and bakery products, fats and oils, and infant food. The foods were prepared to a 'table ready' state (e.g. steak was cooked, eggs were boiled). Samples were collected from all States and Territories in Australia in July and December 2004, as part of the 22nd Australian Total Diet Study (ATDS). Consequently, not all foods known to contain PAH or known to be major contributors to PAH exposure were analysed. This is a limitation of the survey in estimating exposure to PAH from Australian foods.

The selected foods were analysed for 20 different PAH, giving a maximum of 700 data points, of which 15% were reported as non-detects. Based on the analytical concentration data, total PAH levels were highest in hamburger, chocolate (milk), desiccated coconut and potato crisps. The levels in infant foods were relatively low in comparison.

Dietary exposure assessments were conducted for the following groups: 9 month old infants; 2-5 year old males and females; 6-12 year old males and females; 13-18 year old males and females; 19 years and above males and females. Dietary exposure of the general population was determined for benzo[a]pyrene, a representative PAH and a known carcinogen. This is consistent with the approach taken by Joint FAO/WHO Expert Committee on Food Additives (JECFA). Dietary exposure of the general population to benzo[a]pyrene in food is low with the main contributors being bread, hamburgers and chocolate. For 9 month old infants, the major contributors to dietary exposure were also bread and chocolate.

In the absence of sufficient data to establish a tolerable weekly or monthly intake for benzo[a]pyrene, the margin of exposure (MOE) based on the Bench Mark Dose Lower Confidence Limit (BMDL) was used to determine whether the dietary exposure to benzo[a]pyrene is of concern in the different population groups. The MOEs for all population groups assessed at the mean and 95th percentile were above 10,000.

The data presented in this report represents the most comprehensive analysis of PAH concentrations in Australian foods yet undertaken, and are used to estimate the dietary exposure of the Australian population to PAH. This study is reassuring as the results indicate that dietary exposure to PAH is unlikely to be of public health and safety concern.

ACKNOWLEDGEMENTS

FSANZ would like to thank the following organisations and people:

- The State and Territory food regulatory agencies responsible for collecting the samples.
- The staff of the National Measurement Institute Analytical Unit for sample analyses and technical advice.

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ABBREVIATIONS

BMD	Bench Mark Dose
BMDL	Bench Mark Dose Lower Confidence Limit
DIAMOND	Dietary Modelling of Nutritional Data (FSANZ computer software program), based on food consumption data from the 1995 NNS
FSANZ	Food Standards Australia New Zealand
fw	Fresh weight
GC/MS	Gas Chromatography Mass Spectrometry
HRGC	High Resolution Gas Chromatograph
HRMS	High Resolution Mass Spectrometer
IARC	International Agency for Research on Cancer
JECFA	Joint FAO/WHO Expert Committee on Food Additives
LOD	Limit of Detection, the lowest level at which a chemical can be detected in a sample by the analytical method used.
LOR	Limit of Reporting
LOQ	Limit of Quantification
MOE	Margin of Exposure
ng	Nanogram (10^{-9} g)
ng/g	Nanograms per gram
ng/kg bw/day	Nanograms per kilogram of body weight per day
NMI	National Measurement Institute
NOEL	No Observable Effect Level
PAH	Polycyclic Aromatic Hydrocarbons (used to describe all PAH when not necessarily specifying which hydrocarbon)
PTWI	Provisional Tolerable Weekly Intake
QA/QC	Quality Assurance/Quality Control
WHO	World Health Organisation

Note: A glossary of terms can be found in Appendix 1

1. BACKGROUND

1.1 General introduction

Polycyclic aromatic hydrocarbons (PAH) are a group of organic compounds that are produced during incomplete combustion or pyrolysis of organic matter. They occur naturally in the environment from volcanic activity and bush fires, as well as the burning of fossil fuels and industrial activities (NPIS, 2004; ATSDR 1995). PAH are present in the air, water and soil which can be easily transferred to food sources, and also arise during cooking processes such as smoking, barbequing, drying, roasting, cooking and frying.

The presence of PAH in the environment and the potential for this contaminant to be present in food has highlighted a potential safety concern for human health. This concern is associated with the known or suspected carcinogenic properties of a number of PAH.

To assess any potential risk to human health in Australia, Food Standards Australia New Zealand (FSANZ) conducted an analytical survey to quantify the levels of individual PAH in a number of foods available in Australia. This information was the first step in conducting a dietary exposure assessment. This survey was undertaken as part of the surveillance program in 2004.

1.2 Polycyclic aromatic hydrocarbons (PAH)

Polycyclic aromatic hydrocarbons (PAH) are a group of hydrophobic organic compounds comprising over 100 members. Structurally, PAH consist of two or more fused, unsubstituted aromatic rings or their alkyl-substituted derivatives (IPCS, 1998). PAH compounds have been identified in water, air, soil and food samples. The presence of PAH in the environment is of potential concern as some PAH group compounds have demonstrated carcinogenic properties in animal models.

The presence of PAH in the environment is primarily a result of release following pyrolytic reactions occurring in organic matter. This can be a result of industrial or other processes for coal, petrol, oil, bitumen and paper production; or daily activity such as vehicle exhaust, tobacco smoking, wood-stoves, fireplaces and barbeques. PAH are also found in plastics, dyes and pesticides. For example, naphthalene is a member of the PAH family and is the active constituent in moth balls (NPIS, 2004). Due to the airborne nature of PAH, these compounds readily settle in the environment on particulate matter, food and in water. Although these compounds are hydrophobic in nature, contamination of water sources with PAH has been previously identified. The identification of PAH in air and water together with PAH formation and deposition on food through cooking, raises questions of whether the levels of PAH identified in food are hazardous to human health.

1.3 Presence of PAH in food

PAH in foods can result from the transfer from contaminated air, water and soil, depositing PAH directly on food. More frequently, PAH contamination of food occurs through specific cooking processes, generated at high levels in wood-burning stoves, barbeques and fireplaces. PAH production is high in carbon-containing foods heated at elevated temperatures (e.g. 200°C), particularly where fats are released directly onto heat sources and undergo pyrolysis, producing and

depositing high levels of PAH directly onto the surface of the food. To date, PAH has been identified in meat, fruit, oil, cereals and seafood (Kazerouni *et al*., 2001). PAH levels in cooked meat vary and are dependent on the cooking process. For example, in well-done oven-grilled or pan-fried steak, levels of 0.01 ng/g of PAH, benzo[a]pyrene were detected. In contrast, in well-done grilled/barbequed steak levels of 4.75 ng/g of benzo[a]pyrene were detected (Kazerouni *et al*., 2001). The length of cooking time (e.g. medium to well-done) directly contributes to the level of pyrolysis and therefore PAH formation, as meat is exposed to heat and smoke for longer periods of time (Kazerouni *et al*., 2001). Furthermore, smoked foods such as meat and fish, contain PAH. The level of PAH is dependent on the mode of smoking, where traditional smokehouse preparations contain significantly higher levels in comparison to more recent methods where the smoke is generated externally (European Commission, 2002; Karl and Leinemann, 1996). Drying food techniques also contribute to PAH levels in food.

Additional dietary exposure to PAH is also attributed to seafood. Many fish metabolise PAH effectively, and therefore contain very low levels of PAH. In contrast, mussels and oysters display much higher levels. This difference is considered to reflect the large water filtering capability mussels and oysters have in comparison to fish (Fontcuberta *et al.,* 2006).

1.4 Biological significance of PAH

The toxicological database for PAH is extensive and consists of studies on the various individual PAH, mixtures of PAH (in particular coal tar mixtures) and benzo[a]pyrene, one of the most potent carcinogens and well-studied members within the group. A number of national agencies and international bodies have evaluated the toxicity of PAH including the Joint FAO/WHO Expert Committee on Food Additives (JECFA) (WHO, 2006), the International Agency for Research on Cancer (IARC 1973, 1983, 1989 & 2008), the International Program on Chemical Safety (IPCS) (IPCS, 1998) and the US Agency for Toxic Substances and Disease Registry (ATSDR, 1995). The pivotal adverse effect resulting from exposure to PAH is carcinogenicity. PAH typically occur as mixtures in food (and other media), with variation in the toxicity (or potency) of individual compounds; some compounds are genotoxic and/or carcinogenic or neither.

1.5 Australian action

The Australia New Zealand Food Standards Code (the Code) does not prescribe an upper limit for PAH. However, FSANZ has monitored the situation in relation to PAH and taken a number of actions.

In 2005 a FSANZ staff member participated in the JECFA evaluation of PAH as an invited expert, preparing dietary exposure estimates for the Australian population and contributing to the risk assessment. At the time, there were no Australian data on PAH concentrations in food and therefore Australian dietary exposures to PAH were estimated using PAH concentrations from international concentration data accepted by JECFA. Dietary modelling was carried out using FSANZ's Dietary Modelling of Nutritional data (DIAMOND) program, drawing on individual Australian food consumption records from the 1995 National Nutrition Survey (NNS). The estimated mean dietary exposure for Australian consumers of the biologically relevant benzo[a]pyrene was 0.03-0.15 μg/day (0.0005-0.0025 μg/kg bw/day) using an average bodyweight of 67kg. Intake estimates from 18 countries were assessed by JECFA for 10 of the 13 carcinogenic and genotoxic PAH. Intake estimates for benzo[a]pyrene ranged from <1-2 μg/day (0.0001-0.006 μg/kg bw/day using an average bodyweight of 60kg). Intake estimates for the remaining 9

PAH ranged from <1-12 μg/day (0.0001-0.015 μg/kg bw/day using an average bodyweight of 60kg) (JECFA, 2005).

The 2005 JECFA evaluation estimated that internationally, the representative mean intake of benzo[a]pyrene as a measure of PAH was 4 ng benzo[a]pyrene/kg body weight per day with a high level intake of 10 ng benzo[a]pyrene /kg/body weight per day. To characterise the risk associated with PAH exposure from food, estimated dietary exposures were compared to the Bench Mark Dose Lower Confidence Limit (BMDL) of 100 μg benzo[a]pyrene/kg bw/day to derive a margin of exposure (MOE) (refer to Section 6.1 and Appendix 1 for further information). The MOE is the ratio of the BMDL to the estimated exposure to PAH from food; the larger the MOE the smaller the health risk. The MOEs were 25,000 and 10,000 for mean and high-level intakes, respectively. On this basis, JECFA concluded that the risk of human exposure and subsequent health effects of PAH in the diet was low (JECFA, 2005).

In 2004, FSANZ undertook the current analytical survey to quantify actual levels of various PAH in foods and beverages in Australia. This was necessary to more accurately estimate dietary exposure for the Australian population and assess potential risks of PAH in food.

2. SURVEY OF PAH IN AUSTRALIAN FOOD

The analysis of PAH in food samples was undertaken in accordance with quality assurance procedures and the results forwarded to FSANZ. Using these results, dietary exposure to PAH were estimated.

2.1 PAH sample selection and preparation

The 35 food samples (listed in full in Appendix 2) used for PAH analysis were selected from the range of foods that had already been sampled for the 22nd Australian Total Diet Study (ATDS). These foods included; meat products (e.g. bacon, sausage, pork and lamb chops, steak, hamburger, liver and fish), dairy products (e.g. butter, margarine, cream, cheese, milk, ice cream, yoghurt), infants foods (e.g. infant formula, cereal, dinner and dessert) and other foods including; chocolate, potato, carrots and bread. The selection of composited samples from those available from the 22nd ATDS was based on foods that were likely to contain PAH and that represented the main food groups. Consequently not all foods known to contain PAH were analysed. This is a limitation of the survey in estimating exposure to PAH from Australian foods, however the range of food samples analysed was sufficient to represent overall dietary exposure to PAH.

To best represent the food as consumed, all foods analysed in this study were prepared to a 'table ready' state, in accordance with the usual Total Diet Study methods (see http://www.foodstandards.gov.au/monitoringandsurveillance/australiantotaldiets1914.cfm). For example, chicken breast, beef sausage, potatoes, and lamb and pork chops were cooked whereas cheese required no cooking. The majority of food samples required minimal or no preparation prior to analysis. Appendix 3 indicates how each of the food samples were prepared prior to analysis. It is acknowledged that the cooking method, temperature and length of cooking time can influence the amount of PAH formation and deposition on food (Section 1.3). Since the samples used for PAH analysis were originally prepared for the 22nd ATDS, it was not possible to control for these factors in this study. However, the preparation procedures for the 22nd ATDS reflect the food preparation/cooking procedures used in the home.

The samples analysed for PAH were randomly selected from composite samples collected and prepared for the 22nd ATDS. Each composite sample comprised three primary samples ('purchases') from a particular Australian State or Territory.

2.2 PAH Sample analysis

Given that a mixture of PAH are likely to be encountered in the diet, food samples were analysed for 20 environmentally persistent PAH, representing both genotoxic and non-genotoxic compounds noting that some genotoxic PAH are also carcinogenic (Table 1). This was to ensure that the survey and subsequent risk assessment accounted for the variation in toxicity within the PAH. The genotoxic PAH were selected based on existing evidence of presence in food and potency. The genotoxicity and carcinogenicity classifications were based on the 2005 JECFA evaluation and supplementary classifications by IARC. Details of the PAH analysed in the current survey are summarised in Table 1. The samples were analysed on a fresh weight basis and concentrations reported in ng/g.

Table 1: The genotoxic and carcinogenic classification of individual PAH analysed in the FSANZ survey

PAH	JECFA Classification		IARC Classification[3]
	Genotoxic[1]	Carcinogenic[2]	
Acenaphthene	-/+	-	Group 3
Acenapthylene	-/+	-	Not assessed
Anthrancene	-	-	Group 3
Fluorene	-/+	-	Group 3
Fluoranthene	-/+	-	Group 3
Phenanthrene	-/+	-	Group 3
Pyrene	-	-	Group 3
Benz[a]anthracene	+	+	Group 2B
Cyclopenta[c,d]pyrene	+	-	Group 3
Chrysene	+	+	Group 2B
5-Methyl Chrysene	+	+	Group 2B
Benzo[b+k+j] fluoranthene	+	+	Group 2B
Benzo[a]pyrene	+	+	Group 1
Indeno[1,2,3-c,d]pyrene	+	+	Group 2B
Benzo[g,h,i]perylene	+	-	Group 3
Dibenz[a,h]anthracene	+	+	Group 2A
Dibenzo[a,e]pyrene	+	+	Group 3
Dibenzo[a,h]pyrene	+	+	Group 2B
Dibenzo[a,i]pyrene	+	+	Group 2B
Dibenzo[a,l]pyrene	+	+	Group 2A

1 = based on *in vitro* and *in vivo* data; -/+ = equivocal or insufficient data

2 = in laboratory animals; + = positive; - = negative

3 = Group 1 = carcinogenic to humans
Group 2A = probably carcinogenic to humans
Group 2B = possibly carcinogenic to humans
Group 3 = Not classifiable as to carcinogenicity to humans
Group 4 = probably not carcinogenic to humans

2.3 Analytical method detection limits

The analytical methodology used in this survey is outlined in Appendix 4. The Limit of Quantification (LOQ) is the lowest level at which the PAH can be detected and accurately quantified with an acceptable degree of certainty. In contrast, the Limit of Detection (LOD) is the lowest level of a chemical which can be detected but not accurately quantified using a specified

laboratory method and/or item of laboratory equipment. In this analysis, the LOQ is equal to the LOD, with this limit varying for each PAH in the various food matrices analysed. There is a lower degree of certainty where the results are reported as being less than the LOQ and the relative uncertainty increases further where the LOQ is high. Appendix 4 describes the analytical method used in this study.

2.3.1 Lower-, middle- and upper-bound concentrations

A human dietary exposure assessment needs to take into consideration whether the sampling was representative of all foods and whether the detection assay was sufficiently sensitive. Another important aspect is the procedure used to estimate the contribution of PAH that are detected but cannot be quantified (i.e. values between 0 and LOD). Such values need to be considered because it may mean that either the compound was not present or that the assay method used was not sufficiently sensitive to quantify it. When PAH values were reported as being below the LOD, there are three approaches commonly used to incorporate such estimates into the exposure assessment. These approaches involve assigning a *lower bound (equal to zero), middle bound (equal to ½ LOD) or upper bound (equal to LOD)* concentration value (referred to as LB, MB and UB, respectively). The upper bound estimate is likely to be a gross overestimate of the likely true value since the assumption that all PAH concentrations reported as being < LOD are actually at the LOD is highly conservative. The extent of over-estimation decreases with increasing sensitivity of the analytical method. It should be noted that if the lower bound and upper bound totals are far apart (as is often the case, particularly when assay methodology is not particularly sensitive), the middle bound estimate will not necessarily be any closer to the 'true' exposure than is either of the other two estimates. The lower, middle and upper bound approach was applied to all values reported as less than or equal to the LOD except for Acenapthylene, Acenaphthene and Fluorene where a zero value was assigned in all cases due to difficulties in analysing these compounds. This approach was also applied to 5-methyl chrysene for beef sausage and water as a result of difficulties with the analytical method. Lower, middle and upper bound concentrations are listed in the report.

Lower-, middle- and upper-bound concentrations for total PAH (i.e. sum of all PAH analysed) as well as genotoxic and non-genotoxic PAH have been calculated and can be found in Appendix 5. Total PAH concentrations have been presented in the analytical results for this survey; however the dietary exposure and risk characterisation has used benzo[a]pyrene only. The use of benzo[a]pyrene as a marker of exposure to genotoxic and carcinogenic PAH is consistent with the approach taken by JECFA (JECFA, 2005).

3. DIETARY MODELLING

Dietary modelling is a tool used to estimate exposures to food chemicals from the diet as part of the risk assessment process. Dietary modelling uses analytical results for individual foods in combination with food consumption data to calculate estimates of dietary exposure which can be compared to established reference health standards. Food regulators have used dietary modelling techniques internationally for many years as part of the risk assessment process to determine if dietary exposure to specific food chemicals represents an unacceptable risk to public health and safety. The comparison of dietary exposure estimates to reference health standards is crucial in identifying whether the estimated dietary exposure to food chemicals could potentially result in an unacceptable health risk to any population sub-group.

3.1 Food consumption data

The dietary exposure assessment was conducted using food consumption data from the 1995 National Nutrition Survey (NNS) that surveyed 13,858 Australians aged 2 years and above using a 24-hour food recall methodology. Since infants were not included in this 1995 NNS, food consumption data for infants were from a model diet (see Section 3.4).

The foods reported as being consumed in the 1995 NNS were matched (or mapped) to the 35 foods analysed (refer to Appendix 6). This process assigns the levels of PAH detected in the survey foods to the appropriate food consumption data to estimate dietary exposure to PAH. Given that it is impractical to analyse all foods in the food supply, a single food (for example, carrots) may be assumed to represent a whole group of foods (for example, all vegetables). Recipes are used for mixed foods to assign their ingredients to the appropriate survey food (for example, the proportion of potato in Shepherd's Pie). The mapping process may result in the estimated dietary exposures being overestimated as it is assumed that the analytical level of PAH in an analysed food is representative of all foods in that group.

3.1.2 Population groups assessed

The population groups assessed were aged:

- 9 months
- 2-5 years
- 6-12 years
- 13-18 years
- 19 years and above
- 2 years and above.

These age groups were selected as they represent specific life stages such as infants (9 months), toddlers (2-5 years), school children (6-12 years), teenagers (13-18 years) and adults (19 years and above). The Australian population aged 2 years and above is used as a proxy for lifetime exposure. Males and females were assessed separately for all age groups except for infants aged 9 months.

3.2 Dietary exposure calculations

DIAMOND (Dietary Modelling of Nutritional Data) is a computer program developed by FSANZ to computerise dietary exposure assessment calculations. The dietary exposure to PAH was calculated for each individual in the NNS using his or her individual food records from the dietary survey. The DIAMOND program multiplies the specified concentration of PAH by the amount of food that an individual consumed in order to estimate the exposure to PAH from each food. Once this has been completed for all of the foods specified to contain PAH, the total amount of PAH consumed from all foods is summed for each individual. Population statistics (mean and high percentile exposures) are then derived from the individuals' ranked exposures. This process is repeated based on lower-, middle- and upper-bound PAH concentrations.

Where estimated dietary exposures are expressed per kilogram of body weight, each individual's total dietary exposure is divided by their own body weight, the results ranked, and population statistics derived. A small number of NNS respondents did not provide a body weight. These respondents are not included in calculations of estimated dietary exposures that are expressed per kilogram of body weight. The food consumption patterns of the minor number of respondents who did not provide a body weight are generally consistent with those that did and therefore their non-inclusion in the distribution of estimated dietary exposures on a body weight basis is not considered

to be of significance. A summary of the mean body weights for each age/gender group assessed can be found in Appendix 7.

3.2.1 Assumptions in the dietary exposure assessment

The aim of the dietary exposure assessment was to make as realistic an estimate of dietary exposure to PAH as possible. However, where significant uncertainties in the data existed, conservative assumptions were generally used to ensure that the dietary exposure assessment did not underestimate exposure.

The following broad assumptions were made in the dietary exposure assessment:

- all NNS foods that were mapped to an analysed food contain PAH at the specified concentration for the analysed food;
- where an individual NNS food was not mapped to an analysed food, it contains a zero concentration of PAH (e.g. fruit);
- where a food has a specified PAH concentration, this concentration was carried over to mixed foods where the food has been used as an ingredient e.g. milk in a sauce or custard; and
- consumption of foods as recorded in the NNS represent current food consumption patterns.

The following assumptions were made in the mapping of food consumption data to the survey foods:

- all nuts have the same PAH content (including peanut butter);
- fruit bread and cheese-, bacon- or ham- topped bread and rolls do not contain significantly different PAH concentrations in comparison to plain bread, therefore they have been considered to be equivalent to white bread; and
- all brown and multigrain bread have the same PAH concentrations as white bread.

3.2.2 Food contribution calculations

The percentage contribution each food makes to total estimated dietary exposures was calculated by dividing the sum of all consumers' exposures from one food group by the sum of all consumers' exposures from all foods containing the PAH, and multiplying this by 100. Lower bound results were used to calculate the percentage contribution each food group makes to total estimated exposures. The lower bound results provide the best indication of the food groups most likely to contribute to dietary exposure as it only includes foods containing levels of PAH at or above the LOD.

3.3 Limitations with food consumption data

Conducting dietary modelling based on 1995 NNS food consumption data provides the best estimate of actual consumption of a food and the resulting estimated exposure to a food chemical. However, it should be noted that limitations exist within the NNS data. These limitations relate to the age of the data and the changes in eating patterns that may have occurred since the data were collected. Generally, consumption of staple foods such as fruit, vegetables, meat, dairy products and cereal products, which make up the majority of most people's diet, is unlikely to have changed markedly (Cook *et al.*, 2001a; Cook *et al*., 2001b). However, there is an increasing level of uncertainty associated with the consumption of other foods as consumption patterns of these foods may have changed since 1995, or there may be new foods on the market that were not available in

1995. Trends such as the increasing move towards eating leaner cuts of meat would be reflected in this study through the analytical results as the samples are purchased to best represent foods consumed at the time of the ATDS survey and analysed in their 'table ready' state. In the dietary exposure assessment, the total amount of meat consumed in 1995 is assumed to be the total amount of 'leaner' meat consumed in the present day.

A limitation of estimating dietary exposure over a period of time is that only 24-hour dietary survey data were available, and these tend to overestimate habitual food consumption amounts for high consumers. Therefore, predicted high percentile exposures are likely to be higher than actual high percentile exposures over a lifetime. For commonly consumed foods such as bread, milk and meat, which are generally consumed on a daily basis by the majority of Australians, a 24-hour recall provides a relatively accurate estimate of daily consumption amounts over a longer period of time. For occasionally consumed foods, the predicted daily consumption based on 24-hour dietary survey data is not representative of longer-term daily consumption.

Since the dietary modelling associated with this report was conducted, FSANZ has adopted the convention of using the 90th percentile of exposure to represent chronic, high exposure to a food chemical, when exposure is estimated based on a single 24-hour food recall, as is the case with the 1995 NNS. This is in line with international conventions and was adopted as best practice following a peer review (see http://www.foodstandards.gov.au/educationalmaterial/scienceinfsanz/dietaryexposureassessmentsatfsanz/protectinghighconsum4441.cfm). In this report, the 95th percentile of exposure is reported and is likely to overestimate long term high exposure.

3.4 Infant diet

As there were no food consumption data available from the 1995 NNS for children under two years, a model diet was constructed for infants aged 9 months. The model diet for infants aged 9 months included the consumption of solid foods and infant formula. It was based on recommended energy intakes, mean body weight, the proportion of milk and solid foods in the diet for 9 month old children, and data from the 1995 NNS on foods consumed by 2 year old children.

The energy requirement for a nine-month old boy (FAO, 2004) at the 50th percentile body weight of 8.9 kg (WHO, 2007) was used as the basis for the model diet. Boys' weights were used because boys tend to be heavier than girls at the same age and therefore have higher energy and food requirements. It was assumed that 50% of the energy intake was derived from milk (in the form of infant formula) and 50% from all other foods (Hitchcock *et al.*, 1986). To determine the solid portion of the model diet, the patterns of consumption of a two year old child from the NNS were scaled down. Some foods consumed by 2 year old children were excluded since they are inappropriate for infants (e.g. nuts). Details of the model diet for infants aged 9 months are available in Appendix 8.

4. FOOD SURVEY ANALYTICAL RESULTS

4.1 Total PAH concentrations in foods

Thirty five food samples collected for the 22nd ATDS were analysed for 20 different PAH. This gave 700 data points, of which 15% were non-detects, and a remaining 596 data points (85%) with quantified values or "detections". The following PAH compounds were not detected in any of the foods analysed other than tap water: Acenapthylene, Acenaphthene and Fluorene. It is suggested that these results are due to matrix interference and therefore it is difficult to conclude whether these three PAH are actually present in any of the food samples. As a result of difficulties with the analytical method, the 'nd' results was assigned zero for the purpose of estimating dietary exposure for these PAH. A similar approach has been applied for 5-methyl Chrysene in tap water samples and beef sausage.

A summary of the total PAH concentration at the lower, middle and upper bound for each food analysed is shown in Table 2. Given that a mixture of PAH is likely to be encountered in the diet, the individual PAH compounds and the total sum of genotoxic and non-genotoxic forms at the lower-, middle- and upper-bound mean concentrations are outlined in Appendix 5. Benzo[a]pyrene is also presented for each food analysed as a marker of exposure to genotoxic and carcinogenic PAH (Appendix 5).

Table 2: Upper-, middle- and lower-bound concentrations of total PAH in sampled foods (ng/g)[*]

Foods	Mean Total PAH concentration (fresh weight ng/g)[†, ‡]		
	Upper bound	Middle bound	Lower bound
Meat and Meat Products			
Hamburger	50.3	50.2	50.1
Bacon	15.5	10.2	4.9
Sausage, beef	13.3	7.2	1.1
Tuna, canned in brine	11.0	5.5	0
Liver, sheep	6.7	6.6	6.4
Fish fillets	6.0	6.0	5.9
Beef steak	5.5	3.3	1.1
Lamb chops	4.5	2.8	1.1
Pork chops	4.2	2.1	0
Chicken breast fillet	3.4	3.3	3.3
Dairy Products			
Butter, regular	16.9	10.4	3.8
Margarine	11.1	5.5	0
Cream, pure, not thickened	8.4	5.3	2.3
Cheese, cheddar, full fat	6.8	4.0	1.2
Ice cream	2.0	1.0	0
Yoghurt, fruit, full fat	1.8	1.1	0.3
Milk, full fat	1.1	0.5	0
Milk, modified low fat	1.0	0.5	0
Other Foods			
Coconut, desiccated	43.1	26.4	9.6
Potato crisps	32.9	21.9	10.8
Chocolate, milk type	30.1	29.7	29.3
Peanut butter	7.0	3. 5	0
Pizza	4.7	3.1	1.5
Bread, white	3.7	3.6	3.5
Oil, canola	3.5	1.8	0
Eggs, boiled	3.4	1.7	0
Water tap1[≠]	2.6	1.3	0
Carrots	1.6	1.0	0.4
Water tap 2[≠]	1.4	0.7	0
Salt, table, non-iodised	0.8	0.4	0
Potatoes, cooked	0.5	0.2	0
Infant Foods			
Infant dinner, containing meat	5.0	4.9	4.9
Infant dessert, dairy based	2.0	1.4	0.8
Infant cereal	1.9	1.8	1.7
Infant dessert, fruit based	1.7	1.0	0.4
Infant formula	1.2	0.6	0

Note: Results have been rounded to one decimal place.

[*] Some samples required preparation to a ready-to-eat state. Please refer to Appendix 3 for food sample preparation instructions.

[†] The total PAH concentration in each food category is listed in descending order based on the upper bound, with the highest level in each food category listed first.

[‡] Any contribution Acenapthylene, Acenaphthene and Fluorene may have in the total PAH concentration has been excluded due to suspected matrix interference during analyses. Therefore, non-detect values have been assigned zero in all cases.

[≠] duplicate sample. All values reported for the PAH analysed, other than 5-methyl chrysene were reported as <LOD. Discrepancies in the upper- and middle-bound value for the two tap water samples are due to variation in the LOD for individual PAH.

4.1.1 Meat and meat products

A variety of grilled meat samples such as beef, fish, lamb, chicken and pork were analysed in a ready-to-eat state for the presence of PAH (Table 2, Appendix 3). The highest level of total PAH at the UB was identified in hamburger, with 3 -fold lower levels detected in fried bacon and fried sausage beef, and >3-fold lower levels in canned tuna. Lower levels of total PAH (<10 ng/g UB mean) were detected in liver, fish, beef, lamb pork and chicken. Variation between the lower-, middle- and upper-bounds were noted for bacon, beef sausage and canned tuna, whereas little variation was observed for fish, chicken, sheep liver and hamburger.

4.1.2 Dairy products

A variety of dairy products including butter, margarine, cream, cheese, ice cream, yoghurt and milk were analysed for the presence of PAH (Table 2). The highest level of total PAH was identified in butter (16.9 ng/g UB mean) and margarine (11.1 ng/g, UB mean). Lower levels (<9 ng/g UB mean) were detected in cream, cheese, ice cream, yoghurt and milk. Variation between the lower and upper bound means was identified in butter and margarine.

4.1.3 Other foods

A variety of other foods including eggs, vegetables (carrots and potatoes), bread, coconut, pizza, potato crisps, peanut butter, oil and salt were analysed for the presence of PAH (Table 2 and Appendix 3). The highest level of total PAH was identified in coconut (43.1 ng/g UB mean) followed by potato crisps (32.9 ng/g UB mean) and chocolate (30.1 ng/g UB mean). At the upper bound, lower levels (<7 ng/g) were detected in all other products including peanut butter, pizza, bread, oil and eggs. Large variation between the lower- and upper-bound means was identified in coconut and potato crisps.

4.1.4 Infant food

A variety of infant foods including infant dinners, desserts, cereal and formula were analysed for the presence of PAH (Table 2). Please refer to Appendix 3 for food sample preparation instructions. The highest level of total PAH was identified in infant dinner; however levels in general from foods in this category were very low (<5 ng/g). Slight variation between the lower, middle and upper bound means was found in infant cereal and dinner containing meat. In contrast, infant formula, cereal and dessert showed higher levels of variation between the measured means.

4.2 Predominance of individual PAH in each food category

In this survey, 20 individual PAH (Table 1) were examined in 35 different food samples. The complete analytical data for each individual PAH for foods tested is shown in Appendix 5.

4.2.1 Meat and meat products

The levels of PAH in meat products is influenced by cooking temperature and duration. These factors were not specified in the methods and therefore the range of cooking conditions may be limited (Appendix 3). This limitation should be considered particularly for meat products when analysing the results.

Of the detected values, phenanthrene was the dominant PAH in meat products and to a lesser extent fluoranthene and pyrene in samples. Phenanthrene levels were the highest in hamburger (18 ng/g),

followed by sheep liver and chicken breasts (4 ng/g and 2.1 ng/g, respectively). Fluoranthene and pyrene levels were also detected with the highest levels identified in hamburger (8.3 and 13 ng/g, respectively) followed by bacon (1.7 ng/g for both fluoranthene and pyrene). Beef sausage had also detectable pyrene levels (1.1 ng/g). Benzo[a]pyrene levels were highest in hamburger (1.2 ng/g), with all remaining meat samples having low levels, based on the lower bound values.

Two types of fish were analysed: canned tuna in brine and fish fillets. The predominant PAH in fish samples was phenanthrene (fillets: 3.7 ng/g; canned tuna: <9 ng/g). Fluoranthene and pyrene were also identified in fish fillets (0.73 ng/g, 1.3 ng/g, respectively) but not in canned tuna (<0.7 ng/g and <0.4 ng/g, respectively). Anthracene was detected in fish fillets but not canned tuna (0.11 ng/g and <0.2 ng/g, respectively). Benzo[a]pyrene levels were also not detected in either fish sample, based on lower bound values.

4.2.2 Dairy products

In dairy products, butter contained the highest level of total PAH. When analysed for individual compounds,pyrene was identified as the highest PAH in butter (3.8 ng/g), cream (1.5 ng/g), cheese (1.2 ng/g) and fruit yoghurt (0.34 ng/g). Fluoranthene was also present at detectable levels in cream (0.79 ng/g). Benzo[a]pyrene levels were low in all dairy samples, based on the lower bound values.

4.2.3 Infant food

The most prominent PAH in infant food was phenanthrene. Levels were highest in infant dinner containing meat (2 ng/g) and infant cereal (1.1 ng/g). The next most prominent PAH identified in infant food was pyrene: infant dinner (1.9 ng/g), cereal (0.41 ng/g), and dessert (dairy: 0.54 ng/g; fruit: 0.39 ng/g). Markedly lower levels of fluoranthene and anthracene were also detected. Benzo[a]pyrene levels in infant foods were low based on lower bound values.

4.2.4 Other foods

When analysed for individual compounds, phenanthrene (16 ng/g) was detected as the highest contaminant in milk chocolate followed by both fluoranthene and pyrene (4.9 and 4.7 ng/g, respectively).

Phenanthrene was the predominant PAH in bread (2.3 ng/g), whereas pyrene was the primary PAH in pizza (0.6 ng/g) and potato chips (6 ng/g). Pyrene was also detected in bread (0.59 ng/g).

Fluranthene was also identified in pizza (0.47 ng/g), potato chips (2.9 ng/g) and bread (0.37 ng/g) together with anthracene (1.1 ng/g in potato chips). In contrast, salt contained low PAH, based on lower bound values. The contribution of benzo[a]pyrene to the PAH content in white bread was low (0.075 ng/g).

The dominant PAH for coconut was pyrene at a level of 4.1 ng/g. In contrast, carrots were found to contain 0.35 ng/g, whereas pyrene levels in cooked potatoes were not detected based on lower bound values. Fluoranthene and anthracene were also detected in coconut (3.7 ng/g and 1.6 ng/g, respectively). Benzo[a]pyrene was not detected in potatoes, carrots or coconut, based on lower bound values.

4.3 Comparison of PAH concentrations in foods from other countries

A comparison of PAH levels in food from other countries is difficult due to the variation in foods selected for analysis, the analytical methodologies, limit of quantification or reporting, treatment of non-detect values and the calculation and reporting of individual PAH. For example, data from some studies presents total PAH content whereas others report only benzo[a]pyrene levels as an indicator of PAH content in food. Furthermore, the way in which foods are analysed can vary. For example, some studies have examined individual meat sources such as chicken, beef, pork and lamb, whereas other studies have analysed "meat products" as a whole category. Nevertheless, from the data presented in Table 3, the content of PAH in Australian food measured by the level of benzo[a]pyrene is lower in comparison to some other countries.

Table 3: Multinational comparison of Benzo[a]pyrene as a measure of PAH levels in specific foods, in ng/g fresh weight (fw).

Food	Concentration of Benzo[a]pyrene (ng/gram fresh weight)				
	AUSTRALIA[‡]	USA[ℓ]	UK[≠]	SPAIN[∞]	ITALY[£]
Butter	0	nd	0.45	N/A	0.016
Margarine	0	0.12	0.19-6.0	0.272	N/A
Cheese	0[†]	nd	<0.04	0.078	0.014
Milk, whole	0[†]	0.02	<0.04	0.011	0.336
Milk, formula	0	N/A	<0.01-0.2	N/A	N/A
Ice cream	0	N/A	<0.04	N/A	N/A
Yoghurt	0[†]	0.18	<0.04	0.078	0.336
Eggs	0	0.03	<0.04	0.023	0.015
Fresh fish	0	0.15	<0.08	0.235	0.027
Canned fish	0	0.01	N/A	0.272	N/A
Bacon	0	0.2	0.05	0.098	0.034
Beef steak, grilled	0	4.75	0.01-0.04 unsmoked 0.01-0.14 smoked	0.098	0.613 pan 1.445 bbq
Lamb, grilled	0	N/A	<0.04	0.098	N/A
Pork, grilled	0	0.01	<0.04	0.098	0.035 pan 0.121 bbq
Chicken breast	0	0.39	<0.04	0.098	0.015
Beef sausage	0	0.02	0.03-0.26	0.098	
Hamburger	1.2	1.52		0.098	
Bread	0.075	0.10	0.11	0.262	0.017
Milk chocolate	0.29	0.18[€]	N/A	N/A	0.332

Note: There are limitations when making comparisons of PAH levels in food from other countries due to the variation in foods selected for analysis, the analytical methodologies, limit of quantification or reporting, treatment of non-detect values and the calculation and reporting of individual PAH. N/A: not available; nd: not detectable

[†] Cheese was cheddar; yoghurt was full fat and contained fruit, milk was full fat.

[‡] determined from data presented in this study from composite samples. Values quoted are at the lower bound for all foods.

[ℓ] Kazerouni *et al*., 2001. Yoghurt contained fruit and frozen, fresh fish, bacon and pork were pan fried (well done); beef steak and chicken were grilled (well done); hamburger was grilled or barbequed (very well done); Sausages were pork and bread was white bread. Values represented mean concentrations from composite samples.

[≠] http: www.food.gov.uk/science/surveillance, 2002. Values represent upper bound benzo[a]pyrene levels in ppb fresh weight. NB: Value for butter is for all oils and fats tested. Type of milk not stated; value for cheese, ice cream and yoghurt represents all dairy products. Values for pork and lamb are based on meat product values presented. Milk formula value is assumed as Infant formula (data taken from FSIS09/06, 2006). Values for margarine (range values), bacon, beef steak and sausage were taken from WHO 2006; values for beef steak and sausages are for meat and sausage products in general.

[∞] Falco *et al*., 2003; PAH content is estimated as a mean measure of benzo[a]pyrene in composite food samples. Meat products were analysed together and incorporate beef, hamburger, lamb, pork, pork sausage and chicken. Values for fish represents hake and sardines; milk values include whole and semi skimmed; dairy products include cheese and yoghurt; value for margarine also includes oils, canned fish and meat products such as ham, hot dogs and salami.

[¥] WHO, 1998; PAH content is estimated as a measure of benzo[a]pyrene content; poultry and eggs are given a value together; meat and meat products were analysed together (lamb, pork, sausages).

[£] Lodovici *et al*., 1995; PAH content is estimated as a measure of benzo[a]pyrene content in composite samples, value for bacon represents cured meats.

[€] Result is for chocolate candy not specifically milk chocolate.

5. DIETARY EXPOSURE

5.1 Estimated dietary exposures to benzo[a]pyrene

5.1.1 Estimated dietary exposures for population groups aged 2 years and above

For dietary exposure assessments conducted using the 1995 NNS, 99% or more of the respondents in each age/gender group assessed were consumers of PAH. Approximately 89.5% of respondents were consumers of benzo[a]pyrene. Since all respondents were not necessarily consumers, the results reported below are for consumers only of PAH. Appendix 7 shows the number of respondents and the number of consumers of PAH in each age/gender group assessed (excluding infants).

Table 4 summarises the mean and 95th percentile exposures for each age/gender group assessed and is expressed in nanograms per kilogram bodyweight per day (ng/kg bw/day). Appendix 9 provides a summary of mean food consumption data for consumers only of each survey food derived from the 1995 NNS using DIAMOND. Depending on concentration used (lower-, middle- or upper-bound), the estimated mean dietary exposure to benzo[a]pyrene for 2-5 year old children ranged between 0.7 and 3.7 ng/kg bw/day at the mean and between 1.3 and 8.8 ng/kg bw/day at the 95th percentile. Dietary exposures were slightly lower in older children and adults, on a body weight basis.

For 6-12 year old children, estimated exposures ranged between 0.6 and 2.8 ng/kg bw/day at the mean and 2.1 and 6.4 ng/kg bw/day at the 95th percentile. For 13-18 year old children, estimated exposures ranged between 0.5 and 2.2 ng/kg bw/day at the mean and 2.9 and 5.4 ng/kg bw/day at the 95th percentile. For the 19 years and above age group, estimated exposures ranged between 0.2 and 1.3 ng/kg bw/day at the mean and 0.5 and 3.3 ng/kg bw/day at the 95th percentile. For the Australian population aged 2 years and above (as a proxy for lifetime exposure), the estimated exposures ranged between 0.3 and 1.6 ng/kg bw/day and 95th percentile exposure ranged between 0.9 and 4.4 ng/kg bw/day.

5.1.2 Estimated dietary exposures for infants aged 9 months

Table 4 summarises the mean and 95th percentile dietary exposures for infants, with Appendix 8 providing details on the food consumption amounts for the 9 month old infant model diet. Depending on the concentration used (lower-, middle- or upper-bound), the estimated mean dietary exposure to benzo[a]pyrene for 9 month old infants ranged between 0.2 and 4.3 ng/kg bw/day and 95th percentile exposure ranged between 0.6 and 10.7 ng/kg bw/day.

Table 4: Estimated dietary exposure to benzo[a]pyrene for each population group assessed

Age	Gender	Benzo[a]pyrene concentration Type[†]	Mean dietary exposure (ng/kg bw/day)[‡]	95th Percentile dietary exposure (ng/kg bw/day)
9 month infants	Both Combined	Upper Bound	4.3	10.7
		Lower Bound	0.2	0.6
		Middle Bound	2.2	5.6
2 yrs and above	Male	Upper Bound	1.6	4.4
		Lower Bound	0.4	2.2
		Middle Bound	1.0	3.1
	Female	Upper Bound	1.5	3.7
		Lower Bound	0.3	0.9
		Middle Bound	0.9	2.2
2-5 yrs	Male	Upper Bound	3.7	8.8
		Lower Bound	0.8	2.9
		Middle Bound	2.2	5.7
	Female	Upper Bound	3.3	6.4
		Lower Bound	0.7	1.3
		Middle Bound	2.0	3.7
6-12 yrs	Male	Upper Bound	2.8	6.4
		Lower Bound	0.7	3.9
		Middle Bound	1.8	4.7
	Female	Upper Bound	2.4	4.9
		Lower Bound	0.6	2.1
		Middle Bound	1.5	3.3
13-18 yrs	Male	Upper Bound	2.2	5.4
		Lower Bound	0.6	3.9
		Middle Bound	1.4	4.6
	Female	Upper Bound	1.7	3.9
		Lower Bound	0.5	2.9
		Middle Bound	1.1	3.2
19 yrs and above	Male	Upper Bound	1.3	3.3
		Lower Bound	0.4	1.8
		Middle Bound	0.8	2.1
	Female	Upper Bound	1.2	2.6
		Lower Bound	0.2	0.5
		Middle Bound	0.7	1.4

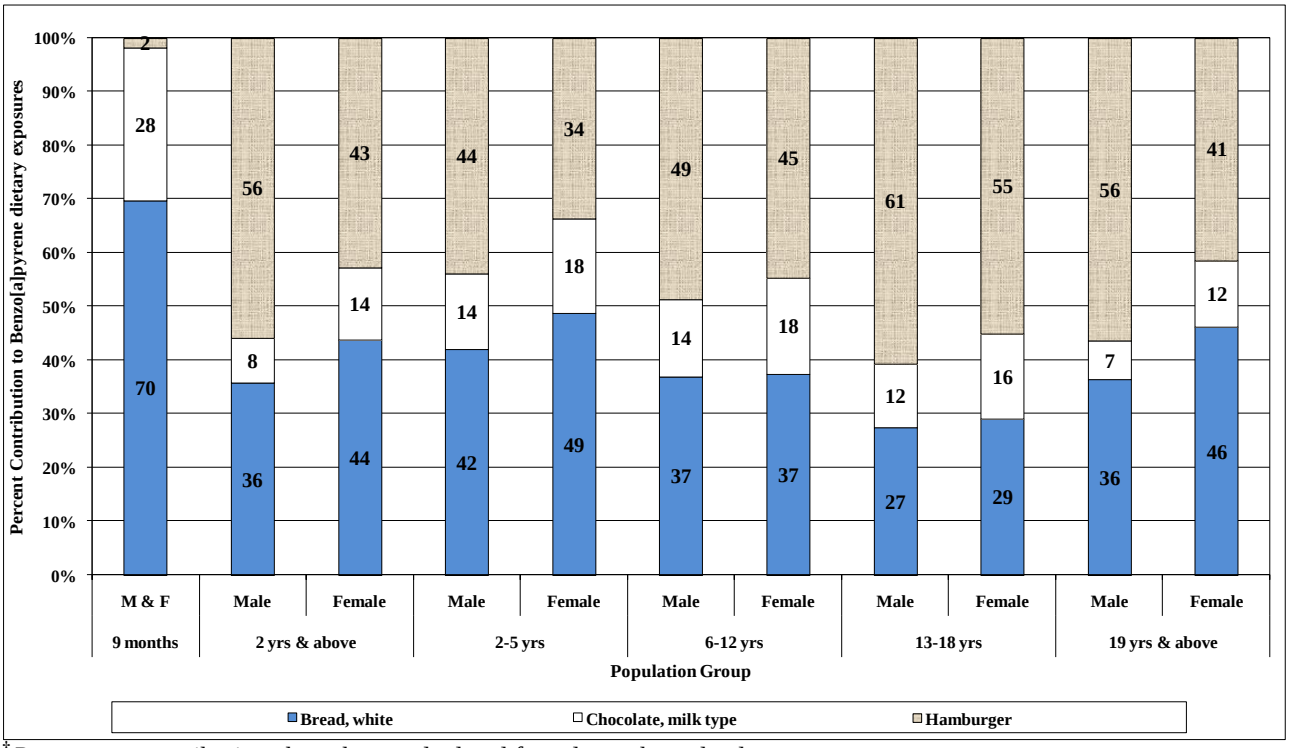
[†] Lower Bound – assumes results reported as being below the LOD are zero, Upper Bound – assumes results reported as being below the LOD are at the LOD, Middle Bound – assumes results reported as being below the LOD are 50% LOD.
[‡] Estimated dietary exposures are based on food consumption data from the 1995 NNS.

5.2 Major contributing foods to estimated dietary exposure to benzo[a]pyrene

The foods which are major contributors (≥5%) to benzo[a]pyrene dietary exposures for one or more of the population groups examined are shown in Figure 1 (Appendix 10 provides detailed information for those foods that contribute to total PAH dietary exposure). Lower bound results were used to calculate the percentage contribution each food group makes to total estimated exposures. This provides the best indication of the food groups most likely to contribute to dietary exposure as it only includes foods containing levels of PAH at or above the LOD. It should be

noted that the percent contribution of each food group is based on benzo[a]pyrene exposure for all consumers of benzo[a]pyrene in the population groups assessed. Therefore benzo[a]pyrene exposures differ for each population group. The major contributors are shown for all population groups assessed.

Figure 1: Percentage contribution of the major contributing foods to benzo[a]pyrene exposure in 9 month old infants and males and females aged 2 years and above†



† Percentage contributions have been calculated from lower bound values.

5.2.1 Infants aged 9 months

The major contributors to benzo[a]pyrene exposure for 9 month old infants were calculated based on the model diet. Figure 1 summarises the foods which were major contributors to benzo[a]pyrene exposure in 9 month old infants who consume infant formula: white bread (70%) and milk chocolate (28%).

5.2.2 All other population groups assessed

For all population sub-groups, the major contributors to benzo[a]pyrene exposure (Figure 1) were white bread (range: 27– 49%), milk chocolate (7%–18%) and hamburgers (34% – 61%).

5.3 Comparison of mean benzo[a]pyrene exposure from food in various countries

The analysis of PAH exposure from food has been of international interest. A comparison between different countries has revealed variations in the level of dietary exposure to PAH. PAH exposure measured as mean benzo[a]pyrene levels, in various countries is shown in Table 5. The mean level of benzo[a]pyrene level in this study was generally lower than those in other countries, however not all foods suspected to contain PAH were analysed in this survey and may account for some of these differences.

When comparing dietary exposure estimates between various countries, the limitations should be highlighted. Variation in the approach taken by the countries in the collection of consumption data, the approach used for modelling and estimating dietary exposure, in addition to analytical approaches outlined in Section 4.2 should be noted.

Table 5: A comparison of the mean benzo[a]pyrene exposure (ng/person/day) in food sampled from various countries

Country	Population Group	Mean Benzo[a]pyrene exposure (ng/person/day)
Australia*	2 years and above	17-102*
Belgium	Whole population	232
Denmark	Whole population	223
Finland	Whole population	185
France	Whole population	245
Germany	Whole population	255
Greece£	Not specified	100
Italy	Whole population	255
Netherlands	Whole population	239
Norway	Whole population	252
Spain∞	Adults (20-65yrs)	97-128
U.K	Whole population	188
New Zealand†	15 years and above	40-160
USA‡	Not specified	160-1600[a]

NB: All data was obtained from EFSA (2008), unless otherwise specified. Values are based on the median of the mean value.
* Data is represented as a range of lower to upper bound exposures and includes males and females aged 2 years and above (consumers only). The data represented in this table is from the current study.
£ European SCOOP Taskforce, 3.2.12, (2004).
∞ Taken from Falcó et al (2003); range is for male and female adults (20-65yrs)
† Exposure estimates taken from WHO, 2006; Values indicate the range of lower to upper bound.
‡ Exposure estimates cited in WHO, 2006.
[a] maximum values

The major contributors to benzo[a]pyrene exposure in Belgium, Denmark, Finland, France, Germany, Italy, Netherlands, Norway and the U.K were cereal and cereal products and seafood and seafood products (EFSA, 2008). Similarly for Spain, the major contributors to mean benzo[a]pyrene exposure for both adult males and females were cereals, fish and shellfish (Falcó et al, 2003).

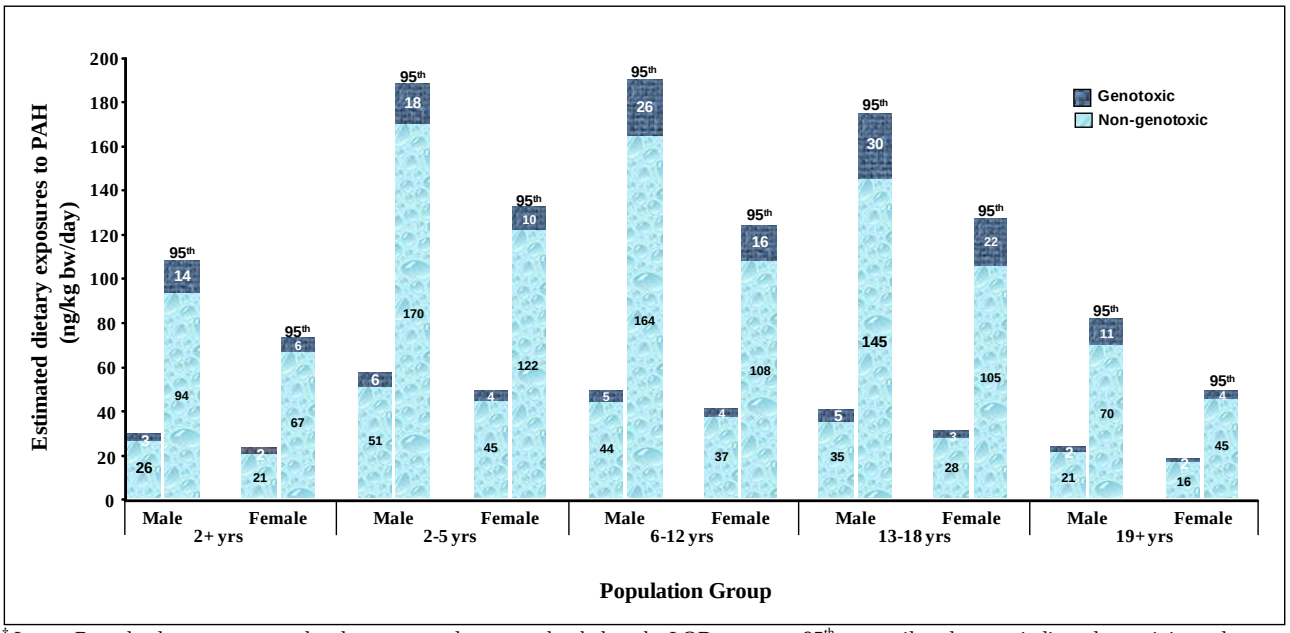
In Greece, the mean exposure to benzo[a]pyrene is predominantly attributed to the consumption of vegetable oils (European SCOOP taskforce 3.2.12, 2004). For New Zealand, the major contributors to benzo[a]pyrene exposure are not specified in the WHO (2006) report, however Phillips (1999) suggests key contributing foods include leafy vegetables, unrefined grains, smoked and barbecued meat and fish. Furthermore, the mean exposure to benzo[a]pyrene in the USA was attributed to grilled/barbecued meats (WHO, 2006).

5.4 The proportion of genotoxic and non-genotoxic PAH in overall dietary exposure at the mean and 95th percentile

The dietary exposure of consumers to total PAH and the relative contribution of genotoxic and non-genotoxic PAH were also estimated in this survey (Appendix 5). The contribution of genotoxic

PAH to the total dietary exposure to PAH was less than or equal to 12% and 17% for population groups at the mean and 95th percentile (high consumers), respectively (Figure 2).

Figure 2: Mean and 95th percentile lower bound estimated dietary exposure to genotoxic and non-genotoxic PAH†#.



† Lower Bound values are presented and assume results reported as below the LOD are zero. 95th percentile values are indicated, remaining values are mean values.

For the purposes of this study, non-genotoxic PAH are considered to be acenaphthene, acenapthylene, anthrancene, fluorene, fluoranthene, phenanthrene and pyrene. The remaining PAH are considered to be genotoxic which is in accordance with JECFA classification (outlined in Table 1).

6. RISK CHARACTERISATION

In characterising the risk associated with PAH exposure through food, it is necessary to consider the nature of the adverse health effects associated with exposure, the timeframe in which these effects are observed, whether there is a threshold dose for these effects, the level of exposure for sensitive subpopulations, and the limitations and uncertainties inherent in the available data.

6.1 Health standard for PAH

In 2005, JECFA reviewed toxicity data on various PAH for the purpose of establishing a health standard for use as a comparator in dietary risk assessments. As 13 of the 33 PAH assessed were considered to be both genotoxic and carcinogenic, a standard threshold approach using a no observed effect level (NOEL) and appropriate safety factors could not be employed. JECFA considered two possible approaches to assessing the risk of mixtures of PAH. The first was to examine the potencies of individual PAH within the mixture and to scale these against a standard compound using toxicity equivalence factors (TEFs). In the absence of suitable TEFs, JECFA was unable to take this approach. The second possible approach was to use the concentration of a single PAH as a surrogate to characterise the toxicity of the mixture. Given that suitable animal carcinogenicity studies conducted with relevant mixtures of PAH were available, benzo[a]pyrene was used by JECFA as a marker of exposure to genotoxic and carcinogenic PAH. Benzo[a]pyrene was also considered appropriate to cover other PAH in a mixture (e.g. possible tumour promoters) because carcinogenicity arising from genotoxicity would occur at lower doses and therefore would provide a conservative margin of safety estimate in the risk assessment of most PAH in a mixture.

Mathematical models were used to fit curves to dose response data from rodent oral carcinogenicity studies to derive a lower confidence interval (BMDL) of the benchmark dose (BMD) for a 10% incidence of tumours. The two studies considered as part of this process were; (i) the study of Culp *et al*., (1998), where mice were dosed orally with either purified benzo[a]pyrene or with two coal tar mixtures containing different PAH, and (ii) the study of Kroese *et al*., (2001), where rats were dosed orally with purified benzo[a]pyrene. For the mouse study, dose-response curves were generated for the incidence of forestomach tumours, lung tumours and total tumour-bearing animals, while for the rat study, dose-response curves were generated for the incidence of liver tumours and total tumour-bearing animals.

Given that people are likely to be exposed to mixtures of PAH in food and that different PAH may act, toxicologically, by different mechanisms (i.e. genotoxic or non-genotoxic), JECFA concluded that the most appropriate basis for the BMDL was the total number of tumour-bearing mice resulting from treatment with coal tar mixtures in the study of Culp *et al*., (1998). A BMDL equivalent to 100 μg benzo[a]pyrene/kg bw/day was established on the basis of carcinogenicity in mice orally dosed with a mixture of PAH representative of genotoxic and carcinogenic PAH present in food.

It is worth noting that Fitzgerald *et al*. (2004) previously established an Australian guideline dose for benzo[a]pyrene of 0.08 μg/kg bw/day for the purpose of deriving a soil guideline value for use in the risk assessments of contaminated soil. While this figure was derived essentially using the same BMD methodology and mouse study Culp *et al*., (1998) there were a several reasons why the number is different than the JECFA standard: (i) the BMDL for a 5% tumour incidence was chosen as the basis of the guideline value rather than a 10% tumour incidence; (ii) data for purified benzo[a]pyrene was used rather than the coal tar mixture; and (iii) the end point of mouse forestomach tumours was used rather than total tumour-bearing mice. For the current survey, the JECFA standard is considered the most appropriate comparator for dietary risk assessment purposes.

It should be noted that the concentration of some PAH in certain foods was higher than benzo[a]pyrene (e.g Phenanthrene) (see Section 4.2). However, given that these PAH are neither genotoxic or carcinogenic they pose a lower risk than benzo[a]pyrene. The use of the BMDL for benzo[a]pyrene therefore provides a conservative margin of safety for these PAH because carcinogenicity arising from genotoxicity would occur at relative low doses.

6.2 Estimated dietary exposure to PAH (benzo[a]pyrene), expressed as a MOE

To characterise the risk associated with PAH exposure from food, the estimated dietary exposures for various population groups were compared to the BMDL of 100 μg benzo[a]pyrene/kg bw/day to derive a MOE. The MOE is the ratio of the BMDL to the estimated exposure to PAH from food; the larger the MOE the smaller the public health risk. The Scientific Committee of the European Food Safety Authority (EFSA) concluded that for compounds that are both genotoxic and carcinogenic, a MOE of >10,000 based on animal cancer bioassay data is likely to be of low public health concern (EFSA 2005).

Table 6 provides the MOEs for various population groups at the lower-, middle- and upper-bounds of dietary exposure to benzo[a]pyrene. The choice of considering the lower-, middle- or upper-bound estimate of dietary exposure has an obvious effect on the magnitude of the MOE. For example, the MOEs for 2-5 year old males are 34,000, 18,000 and 11,000 for 95th percentile exposures at the lower-, middle- and upper-bounds, respectively. The difference in the MOE

between the lower bound, which includes only measured values, and the middle- or upper-bounds, which include default values for foods that were below the LOD, warrants some comment. If there are many food types in which no compounds are detected then the effect on the apparent MOE at the middle- and upper-bound, and therefore the level of concern, will be exaggerated.

Benzo[a]pyrene was detected in less than 10% of the samples analysed. It seems reasonable to conclude that the likely true benzo[a]pyrene concentrations in the remaining foods tested lie closer to the middle bound than the upper bound, which is likely to be an overestimate of actual benzo[a]pyrene concentrations (Hewett & Ganser, 2007). In particular, foods for which no genotoxic PAH (or indeed, no PAH) were detected are considered more likely to have benzo[a]pyrene concentrations closer to the lower bound than the middle bound. Given the number of foods in this survey for which no genotoxic PAH were detected, the MOE is more likely to be closer to the lower bound than the middle bound.

The differences in estimated MOEs for 9-month old infants at both the mean and 95th percentile exposures are broad, with an 18-fold difference between exposures at the lower and upper bounds. This compares with only a 2-5 fold difference between lower and upper bound estimates for all other age groups. The model infant diet assumes that 50% of the infant's energy intake comes from infant formula, thus data for infant formula makes a significant contribution to estimates of exposure. As no PAH were detected in infant formula, it is likely that the lower bound concentration of zero is a more accurate estimate of the concentration of benzo[a]pyrene in infant formula, and the middle- and upper-bound MOEs are likely to be overestimates. Therefore the MOEs at mean and 95th percentile exposures for 9 month infants are >100,000.

On the basis of the above considerations and using the highest estimate of dietary exposure for all population groups (excluding 9-month old infants), the MOEs for all populations groups are >10,000. As such, dietary exposure to PAH is considered to be of low public health concern.

Table 6: MOEs for benzo[a]pyrene in various Australian population groups

Age/	Gender	Concentration Type[1]	Mean dietary exposure[†] (ng/kg bw/day)	MOE[2] (mean exposure)	95th percentile dietary exposure[†] (ng/kg bw/day)	MOE (95th percentile exposure)
9-month infants[*]	Male & female	Upper Bound	4.3	23,000	10.7	9,300
		Lower Bound	0.2	500,000	0.6	167,000
		Middle Bound	2.2	45,000	5.6	18,000
2 years & above	Male	Upper Bound	1.6	62,500	4.4	23,000
		Lower Bound	0.4	250,000	2.2	45,000
		Middle Bound	1.0	100,000	3.1	32,000
	Female	Upper Bound	1.5	67,000	3.7	27,000
		Lower Bound	0.3	330,000	0.9	110,000
		Middle Bound	0.9	110,000	2.2	45,000
2-5 years	Male	Upper Bound	3.7	27,000	8.8	11,000
		Lower Bound	0.8	125,000	2.9	34,000
		Middle Bound	2.2	45,000	5.7	18,000
	Female	Upper Bound	3.3	30,000	6.4	16,000
		Lower Bound	0.7	143,000	1.3	77,000
		Middle Bound	2.0	50,000	3.7	27,000
6-12 years	Male	Upper Bound	2.8	35,000	6.4	16,000
		Lower Bound	0.7	143,000	3.9	26,000
		Middle Bound	1.8	56,000	4.7	21,000
	Female	Upper Bound	2.4	42,000	4.9	20,000
		Lower Bound	0.6	167,000	2.1	48,000
		Middle Bound	1.5	67,000	3.3	30,000
13-18 years	Male	Upper Bound	2.2	45,000	5.4	19,000
		Lower Bound	0.6	167,000	3.9	25,000
		Middle Bound	1.4	71,000	4.6	22,000
	Female	Upper Bound	1.7	58,000	3.9	26,000
		Lower Bound	0.5	200,000	2.9	35,000
		Middle Bound	1.1	90,000	3.2	31,000
19 years& above	Male	Upper Bound	1.3	77,000	3.3	30,000
		Lower Bound	0.4	250,000	1.8	55,000
		Middle Bound	0.8	125,000	2.1	48,000
	Female	Upper Bound	1.2	83,000	2.6	43,000
		Lower Bound	0.2	500,000	0.5	200,000
		Middle Bound	0.7	142,000	1.4	71,000

1 = Lower Bound – zero value assigned to all results below the LOD (non-detections)
Middle Bound – 50% LOD value assigned to all results below the LOD (non-detections)
Upper Bound – the LOD assigned to all results below the LOD (non detections)

2 = BMDL (100 µg benzo[a]pyrene/kg bw/day) ÷ mean dietary exposure (µg/kg bw/day).

[†] Dietary exposure figures have been rounded to the first decimal place. MOE values have been calculated from the rounded dietary exposure figures.

7. CONCLUSIONS

The data presented in this report represent the most comprehensive analysis of PAH concentrations in Australian foods yet undertaken and are used to estimate the dietary exposure of the Australian population to PAH. The dietary exposure assessment has been used in conjunction with the available information on the hazard characterisation of PAH to assess the human health risk associated with exposure to PAH in food.

Benzo[a]pyrene was detected in less than 10% of the samples analysed, and only in hamburger, chocolate and white bread. Furthermore, the following foods, comprising 15% of samples, had no

detectable PAH above the LOD: ice cream, infant formula, milk, margarine, pork chops, tap water, canola oil, peanut butter, eggs, canned tuna, potatoes and table salt. The foods that contained the highest concentrations of total PAH were hamburger, milk chocolate, potato crisps and desiccated coconut. Utilising the analytical data in combination with appropriate food consumption data enabled a dietary exposure assessment to be undertaken for various population groups, including infants. These calculations indicated that dietary exposure to PAH in food is low, with the MOEs for all population groups greater than 10,000 and therefore not a public health and safety concern.

On the basis of the available data and taking into account all the inherent uncertainties and limitations it can be concluded that the risk arising from dietary exposure to PAH for the Australian population, is unlikely to be of public health and safety significance.

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APPENDIX 1: DEFINITIONS AND GLOSSARY OF TERMS

Bench Mark Dose (BMD) Method

Refers to the use of a mathematical model to curve-fit dose-response data for a particular toxicological endpoint in order to estimate the threshold dose corresponding to a level of benchmark response (BMR). The Benchmark Dose (BMD) is the dose that produces a prespecified change in the BMR over the background (US EPA: http://www.epa.gov/riskassessment/glossary.htm).

Bench Mark Dose Lower Confidence Limit (BMDL)

The BMDL is the lower confidence limit of the bench mark dose for a 10% level of the BMR.

Carcinogenic

A carcinogenic compound is one that is capable of causing cancer.

fw (fresh weight)

The amount of a food chemical which is present in a given weight of the food as it is actually eaten. Fresh weight concentrations are used, combined with dietary survey data, to estimate dietary exposure.

Genotoxic

A genotoxic compound is one that is capable of damaging genetic material, which *may or may not* lead to the development of cancer.

Limit of Detection (LOD)

The LOD is the lowest concentration of a chemical that can be qualitatively detected using a specified laboratory method and/or item of laboratory equipment (i.e. its presence can be detected but not quantified).

Limit of Quantification (LOQ)

The LOQ is the lowest concentration of a chemical that can be detected and accurately quantified, with an acceptable degree of certainty, using a specified laboratory method and/or item of laboratory equipment.

Limit of Reporting (LOR)

The LOR is the lowest concentration level that the laboratory reports analytical results. For the purposes of this report, the LOD was chosen as the basis for the LOR (i.e. the LOR is equivalent to the LOD).

Lower bound

An estimate of dietary exposure assuming analytical results reported as being below the LOD are equal to zero.

Mapping

The process that assigns the levels of substances detected in survey foods to the appropriate food consumption data to estimate dietary exposure to the substance. Given that a survey cannot analyse all foods in the food supply, a single survey food may be assumed to represent a whole group of foods with appropriate adjustment factors for concentration.

Margin of Exposure (MOE)

The ratio of the BMDL to the estimated exposure dose.

Middle bound

An estimate of dietary exposure assuming analytical results reported as being below the LOD are equal to 50 % of the reported LOD.

No Observable Adverse Effect Level (NOAEL)

The NOAEL refers to the highest concentration or amount of a substance that can be administered without observing any adverse effects (IUPAC, 2007).

Provisional Tolerable Weekly Intake (PTWI)

The PTWI is an endpoint value that represents the allowable weekly exposure by humans to these contaminants which is associated with the consumption of wholesome and nutritious foods (IPCS, 2009).

Upper bound

An estimate of dietary exposure assuming analytical results reported as being less than the LOD are equal to the LOD.

APPENDIX 2: DERIVATION OF FOODS ANALYSED FOR PAH FROM 22ND ATDS SAMPLING

Table A1: Food samples analysed for PAH from samples collected for the 22nd Australian Total Diet Study

FOOD ANALYSED	22nd ATDS		PAH	
	No. States/Territories sampled in 22nd ATDS	Total no. primary samples ('purchases') for all States/Territories in the 22nd ATDS[ℓ]	No. primary samples ('purchases') analysed for PAH[†]	No. analyses (composites) analysed for PAH[‡]
Bacon, cooked	3	18	3	1
Beef steak, rib/rib eye/sirloin, grilled	5	30	3	1
Bread, white	5	30	3	1
Butter, regular	3	18	3	1
Carrot cooked	5	30	3	1
Cheese, cheddar, full fat	5	30	3	1
Chicken, breast, fillet, cooked	3	18	3	1
Coconut, desiccated	3	18	3	1
Cream, pure (not thickened)	3	18	3	1
Eggs, boiled	5	30	3	1
Fish fillets, cooked	5	30	3	1
Hamburger, cooked	5	30	3	1
Ice Cream, full fat, vanilla	3	18	3	1
Infant Cereal, mixed	3	18	3	1
Infant Dessert, dairy based	3	18	3	1
Infant Dessert, fruit	3	18	3	1
Infant Dinner, meat, chicken or fish	3	18	3	1
Infant Formula, powder, cow's milk based	3	18	3	1
Lamb Chops, loin, grilled	5	30	3	1
Liver, sheep, cooked	5	30	3	1
Margarine/ Spread, Polyunsaturated	3	18	3	1
Chocolate, milk type	3	18	3	1
Milk, full fat	5	30	3	1
Milk, modified, low fat	5	30	3	1
Oil, canola	3	18	3	1
Peanut butter	3	18	3	1
Pizza, meat & vegetable, cooked	3	18	3	1
Pork Chops, grilled	3	18	3	1
Potato crisps	3	18	3	1
Potatoes cooked	5	30	3	1
Salt, table, non-iodised	3	18	3	1
Sausage, beef, cooked	5	30	3	1
Tuna, canned in brine	3	18	3	1
Water, Tap	8	48	3	1
Yoghurt, fruit, full fat	3	18	3	1

[ℓ] In the 22nd ATDS, each time a State or Territory collected samples they were required to collect six individual purchases which were prepared into two composite samples comprising three individual purchases in each composite sample.

[†] A single composite sample was randomly selected from the State/Territory composite sample collected for the 22nd ATDS. Each single composite sample analysed for PAH comprised 3 primary samples ('purchases').

[‡] Of the two composite samples collected by the States and Territories for the 22nd ATDS the sample(s) analysed for PAH were selected at random.

APPENDIX 3: FOOD SAMPLE PREPARATION INSTRUCTIONS

General Instructions

Boiling water
Except where other instructions are provided, 'boiling water' means that the food is to be boiled in 'unsalted' tap water.

Crush
When the preparation instruction states `crush', then the following procedure is to be followed:

- Place food into a plastic bag.
- With a rolling pin crush food so that the food pieces are no larger than half a centimetre in any one direction.

Chop
Except where other instructions are provided, 'chop' means that the food is to be chopped with a stainless steel knife into pieces no larger than 1 cubic centimetre. In most cases, it should be possible to chop into pieces much smaller than this, which is desirable.

Washing
Foods are to be washed in accordance with local practice and the food concerned.

Mix
When the preparation instruction states 'mix' or 'mix thoroughly', then the following procedure is to be followed:
(1) For dry foods (such as coconut) or semi-dry foods (such as cooked chopped meat):

- Form the food into a cone or pile.
- Flatten the cone slightly and separate into four equal segments.
- Pull the segments apart so that four separate piles are formed.
- Combine diagonally opposite piles and mix together thoroughly.
- This process should be repeated until thorough mixing of the group of purchases has been achieved.

(2) For foods containing juice such as tomatoes and oranges:

- If possible, the food being prepared should be chopped in a large glass or stainless steel bowl so that all the juice is collected.
- Mixing of the chopped pieces is then done in the bowl using gloved hands or stainless steel cutlery and should be mixed as thoroughly as possible.
- For the purposes of the ATDS, any juice must be regarded as an integral part of the food being prepared for analysis. A proportional amount of juice and seeds must therefore be included in the sample containers.

(3) For liquids such as milk and soft drink:

- Liquids are to be measured into a large receptacle such as bowl or jug made of stainless steel or Pyrex. Plastic containers are to be avoided.
- The total volume added to the jug or bowl should be thoroughly stirred with a stainless steel utensil before being poured into the sample containers.

Cooking, Frying, Grilling
In the case of samples of meat, it is imperative that typical cooking behaviour be followed.

For example, meat that is fried will exude fat. As the fried food is removed from the fry pan some fat will remain in the fry pan and some will remain on the cooked meat product. The fat remaining in the fry pan is to be discarded and only the fat on the cooked food is to be included for analysis.

Microwaving

The time required for microwaving will depend upon the power of the microwave. Broccoli, beans, peas and spinach are required to be cooked by microwave. The following procedure is proposed:

1. Place 900 grams of broccoli, peas, beans or spinach into a glass/ pyrex cooking dish that has a fitted lid and add one third of a cup of water.
2. Place in 650-Watt microwave on high power setting for 7 minutes. Higher power microwaves should have the setting adjusted to medium or the time of cooking reduced as necessary. It may also be necessary to stir the vegetables during cooking to ensure even heat distribution.
3. Remove from microwave and allow cooling before handling.

Gloves

Gloves are to be worn whenever the food being prepared could come into contact with hands. The gloves to be used are Ansell rubber gloves or latex gloves (subject to allergy concerns) not containing lubricant.

Equipment

- Stainless steel knives
- Wooden cutting board (good quality, smooth, crack free)
- Stainless steel or Teflon coated utensils (i.e. fry pans, spatulas, etc.).
- Glass/Pyrex equipment can also be used.
- For the purposes of mixing liquids, a large stainless steel or Pyrex receptacle such as a jug or bowl is to be used.
- Plastic bags for enclosing sample containers.

Washing of Equipment

The analytical laboratory is to determine the detergent to be used in the washing of food preparation equipment. The detergent chosen should not interfere with the analyses for iodine, selenium, molybdenum, nickel, chromium or nitrites and nitrates.

Handling Purchases in the Food Preparation Facility

Each purchase as provided by the purchasing officer should arrive in separate packaging. Unprocessed, raw foods such as steak and chicken fillets will be in separate packages labelled with the name of the food and the date of purchase. Manufactured, packaged foods will be labelled clearly with the date of purchase. Purchases from each jurisdiction will be in lots of six.

Keeping Samples Separate

Care must be taken to ensure no mixing of any kind between the three groups of samples. This means careful cleaning of utensils between the three preparation procedures. To ensure accuracy and to keep food samples separate, the procedure for preparing one sample in readiness for analysis should be completed and all utensils cleaned thoroughly before the preparation of the next sample is started.

Table A2: Food sample preparation instructions[#]

Food	Preparation
Bacon	Remove bacon rind. Chop and mix together thoroughly. Fry the bacon until tender.
Beef steak, rib/rib eye/sirloin	Grill the three purchases of beef steak. When cooked, trim off excess fat. Discard the fat in the grill tray. Chop the cooked meat as finely as possible.
Bread, white	Include one end crust from each loaf. Chop and mix the bread.
Butter, regular	Mix thoroughly.
Carrots	Top and tail the carrots. If the carrots are unblemished, rinse only, if not, peel and remove blemishes. Slice carrots thinly. Boil carrot slices in unsalted water.
Cheese, cheddar, full fat	Chop into small cubes and mix.
Chicken, breast, fillet, skinless	Grill the three purchases of chicken breast. Discard the fat in the grill tray. Chop the cooked chicken as finely as possible. Mix the cubes together thoroughly.
Coconut, desiccated	Mix together thoroughly.
Cream, pure (not thickened)	Shake and invert containers to ensure thorough mixing of contents. Mix together in large stainless steel or glass bowl.
Eggs, boiled	Hard boil the 18 eggs, remove shells. Chop and mix thoroughly.
Fish fillets, cooked	Grill until cooked through. Remove bones. Chop and mix the pieces of fish.
Hamburger, cooked	Chop and mix hamburgers together thoroughly.
Ice cream, full fat, vanilla	Mix together thoroughly.
Infant cereal, mixed	Mix the contents of three packets of infant cereal. Prepare the cereal in accordance with the instructions in the label.
Infant dessert, dairy based	Combine each purchase into a large glass or stainless steel bowl and mix.
Infant dessert, fruit	Combine 300 grams from each purchase into a large glass or stainless steel bowl. Mix.
Infant dinner, containing meat, chicken or fish	Combine 300 grams from each purchase into a large glass or stainless steel bowl. Mix.
Infant formula, powder, cow's milk based	Make up 360 ml of the formula using tap water according to manufacturer's directions in a stainless steel or Pyrex bowl. Mix together.
Lamb chops, loin, grilled	Grill the three purchases of lamb chops. When cooked, cut all the meat away from the bone and trim off excess fat. Discard the fat in the grill tray. Chop the cooked meat as finely as possible.
Liver sheep, cooked	Weigh 300 grams of sheep liver from each purchase (i.e. 900 grams in total). Trim and slice the liver. Grill slices of liver until cooked. Chop and mix cooked liver.
Margarine or margarine spread, polyunsaturated	Mix thoroughly.
Milk, full fat	Mix together in large stainless steel or glass bowl.
Chocolate, milk type	Chop and mix together thoroughly.
Milk, modified, low fat	Mix together in large stainless steel or glass bowl.
Oil, canola	Mix together in large stainless steel or glass bowl.

Food	Preparation
Peanut butter	Mix thoroughly.
Pizza, meat & vegetable-cooked	Chop and mix thoroughly.
Pork Chops, grilled	Grill the three purchases of pork chops. When cooked, cut all the meat away from the bone and trim off excess fat. Discard the fat in the grill tray. Chop the cooked meat as finely as possible.
Potato crisps	Mix the crushed potato chips/crisps thoroughly in a large bowl.
Potatoes, cooked	Wash thoroughly, peel and halve potatoes. Cook together in unsalted water. When cooked, drain potatoes, chop finely and mix.
Salt, table, non-iodised	Mix together.
Sausage beef, cooked	Separate sausages into individual links. Dry fry each purchase of sausages until cooked through. When cool, chop and mix in a large stainless steel or glass bowl.
Tuna, canned in brine	Chop and mix together.
Water, tap	Mix in a large stainless steel or glass bowl.
Yoghurt, fruit, full fat	Mix together.

[#]Sample preparation indicated in the above table is consistent with the preparation of samples for the 22nd ATDS. Some of the samples prepared for the 22nd ATDS were also used for PAH analysis.

APPENDIX 4: METHODS OF ANALYSIS AND QUALITY ASSURANCE

PAH analysis was conducted on composited food samples by isotope dilution high resolution gas chromatography/high resolution mass spectrometry (HRGC/HRMS).

Homogenous composited samples were prepared following lyophilisation. A representative portion was removed and spiked with a range of isotopically labelled surrogate standards, saponified and extracted with organic solvent.

Sample clean up was conducted by partitioning with formic acid then complexing with a caffeine solution. Further purification using silica gel column chromatography was conducted. Internal standards are added to each extract immediately prior to injection into the GC. PAH are separated by the GC and detected by a high-resolution (>10,000) mass spectrometer. The quality of the analysis is assured through reproducible calibration and testing of the extraction, cleanup, and GC/MS systems. The limits of detection (LOD) for the 20 PAH range from <0.0003 to <30 ng/g, respectively and are shown in Table A3.

All analytical results are corrected for labelled surrogates and reported on a nanogram per gram lipid and per gram fresh weight basis.

Table A3: List of PAH Analysed and Limits of Detection

PAH	**Limit of Detection (LOD)[†] (ng/g)**
Acenaphthene	<0.05 - <0.2
Acenaphthylene	<0.03 - <0.2
Anthracene	<0.003 - <0.6
Benz[a]anthracene	<0.004 - <0.3
Benzo[b,k,j]fluorathene	<0.003 - <0.3
Benzo[g,h,i]perylene	<0.002 - <0.5
Benzo[a]pyrene	<0.003 - <0.4
Chrysene	<0.006 - <0.6
Dibenz[a,h]anthracene	<0.0003 - <0.05
Fluoranthene	<0.03 - <2
Fluorene	<0.05 - <0.3
Indeno[1,2,3-cd]pyrene	<0.001 - <0.3
Phenanthrene	<0.03 - <30
Pyrene	<0.05 - <1
Cyclopenta[c,d]pyrene	<0.002 - <0.08
5-Methyl Chrysene	<0.001 - <0.1
Dibenzo[a,e]pyrene	<0.001 - <0.2
Dibenzo[a,h]pyrene	<0.0008 - <0.2
Dibenzo[a,i]pyrene	<0.002 - <0.1
Dibenzo[a,l]pyrene	<0.004 - <0.4

[†] The Limit of Detection varies and is dependent on the type of food matrix tested. The values presented in the table above are the range for each analyte for all food types.

APPENDIX 5 INDIVIDUAL PAH CONCENTRATIONS IN FOOD ANALYSED INCLUDING A SUMMARY OF TOTAL, GENOTOXIC AND NON-GENOTOXIC PAH CONCENTRATIONS

Table A4: PAH concentrations in specific foods (fresh weight ng/g)

	Food													
	Ice cream	**Infant formula**	**Milk, full fat**	**Milk, modified, low fat**	**Butter, regular**	**Margarine**	**Cheese, cheddar, full fat**	**Cream, pure, not thickened**	**Yoghurt, fruit, full fat**	**Chicken breast fillet**	**Infant dinner, containing meat**	**Pizza**	**Pork chops**	**Fish fillets**
Fresh Weight ng/g														
Acenapthylene	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
Acenaphthene	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
Fluorene	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
Phenanthrene	<0.8	<0.5	<0.7	<0.5	<9	<7	<4	<5	<1	2.1	2	<3	<3	3.7
Anthrancene	<0.5	<0.3	<0.05	<0.3	<0.4	<0.2	<0.2	<0.3	<0.04	0.085	0.1	<0.1	<0.09	0.11
Fluoranthene	<0.1	<0.08	<0.09	<0.05	<2	<0.9	<0.7	0.79	<0.2	0.4	0.7	0.47	<0.4	0.73
Pyrene	<0.3	<0.08	<0.1	<0.1	3.8	<1	1.2	1.5	0.34	0.65	1.9	0.6	<0.4	1.3
Non-genotoxic Lower	0	0	0	0	3.8	0	1.2	2.29	0.34	3.235	4.7	1.07	0	5.84
Non-genotoxic Middle	0.85	0.48	0.47	0.475	9.5	4.55	3.65	4.94	0.96	3.235	4.7	2.62	1.945	5.84
Non-genotoxic Upper	1.7	0.96	0.94	0.95	15.2	9.1	6.1	7.59	1.58	3.235	4.7	4.17	3.89	5.84
Benz[a]anthracene	<0.01	<0.02	<0.007	<0.006	<0.1	<0.2	<0.06	<0.06	<0.01	<0.02	0.017	0.058	<0.03	<0.02
Cyclopenta[c,d]pyrene	<0.007	<0.01	<0.003	<0.002	<0.08	<0.05	<0.03	<0.04	<0.006	<0.004	0.01	0.017	<0.008	<0.005
Chrysene	<0.03	<0.05	<0.02	<0.01	<0.2	<0.4	<0.1	<0.1	<0.04	0.045	0.12	0.13	<0.05	0.073
5-Methyl Chrysene	<0.005	<0.004	<0.004	<0.003	<0.04	<0.05	<0.02	<0.02	<0.004	<0.02	<0.1	0.013	<0.03	<0.003
Benzo[b+k+j]fluorathene	<0.01	<0.01	<0.009	<0.006	<0.1	<0.1	<0.05	<0.05	<0.01	<0.008	<0.01	0.047	<0.03	<0.01
Benzo[a]pyrene	<0.02	<0.02	<0.01	<0.007	<0.2	<0.2	<0.06	<0.09	<0.01	<0.008	<0.005	<0.03	<0.03	<0.01
Indeno[1,2,3-c,d]pyrene	<0.02	<0.01	<0.01	<0.006	<0.1	<0.2	<0.05	<0.07	<0.01	<0.008	<0.005	0.05	<0.01	<0.01
Benzo[g,h,i]perylene	<0.03	<0.03	<0.02	<0.01	<0.3	<0.3	<0.1	<0.1	<0.02	<0.02	0.011	0.093	<0.04	<0.02

	Ice cream	Infant formula	Milk, full fat	Milk, modified, low fat	Butter, regular	Margarine	Cheese, cheddar, full fat	Cream, pure, not thickened	Yoghurt, fruit, full fat	Chicken breast fillet	Infant dinner, containing meat	Pizza	Pork chops	Fish fillets
Fresh Weight ng/g														
Dibenz[a,h]anthracene	<0.004	<0.003	<0.001	<0.002	<0.02	<0.05	<0.01	<0.01	<0.01	<0.002	<0.001	<0.008	<0.004	<0.003
Dibenzo[a,l]pyrene	<0.03	<0.006	<0.02	<0.01	<0.2	<0.2	<0.07	<0.09	<0.02	<0.01	<0.008	<0.03	<0.03	<0.02
Dibenzo[a,e]pyrene	<0.02	<0.01	<0.01	<0.006	<0.1	<0.02	<0.04	<0.06	<0.01	<0.001	<0.003	<0.004	<0.01	<0.01
Dibenzo[a,i]pyrene	<0.03	<0.01	<0.01	<0.01	<0.1	<0.08	<0.06	<0.02	<0.01	<0.004	<0.006	<0.01	<0.01	<0.005
Dibenzo[a,h]pyrene	<0.04	<0.02	<0.02	<0.01	<0.2	<0.1	<0.09	<0.1	<0.02	<0.008	<0.01	<0.01	<0.01	<0.005
Genotoxic Lower	0	0	0	0	0	0	0	0	0	0.045	0.158	0.408	0	0.073
Genotoxic Middle	0.128	0.1015	0.072	0.044	0.87	0.975	0.37	0.405	0.09	0.1015	0.232	0.454	0.146	0.1335
Genotoxic Upper	0.256	0.203	0.144	0.088	1.74	1.95	0.74	0.81	0.18	0.158	0.306	0.5	0.292	0.194
Total Lower bound	0	0	0	0	3.8	0	1.2	2.29	0.34	3.28	4.858	1.478	0	5.913
Middle bound	0.978	0.5815	0.542	0.519	10.37	5.525	4.02	5.345	1.05	3.3365	4.932	3.074	2.091	5.9735
Total Upper bound	1.956	1.163	1.084	1.038	16.94	11.05	6.84	8.4	1.76	3.393	5.006	4.67	4.182	6.034
Benzo[a]pyrene Lower	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Benzo[a]pyrene Middle	0.01	0.01	0.005	0.0035	0.1	0.1	0.03	0.045	0.005	0.004	0.0025	0.015	0.015	0.005
Benzo[a]pyrene Upper	0.02	0.02	0.01	0.007	0.2	0.2	0.06	0.09	0.01	0.008	0.005	0.03	0.03	0.01

	Hamburger	Liver, sheep	Bread, white	Infant cereal	Carrots	Infant dessert, dairy based	Infant dessert, fruit based	Water, tap 1	Water, tap 2	Sausage, beef	Coconut, desiccated	Potato crisps	Bacon	Beef steak
Fresh Weight ng/g														
Acenapthylene	nd	nd	nd	nd	nd	nd	nd	<0.2	<0.1	nd	nd	nd	nd	nd
Acenaphthene	nd	nd	nd	nd	nd	nd	nd	<0.2	<0.1	nd	nd	nd	nd	nd
Fluorene	nd	nd	nd	nd	nd	nd	nd	<0.3	<0.2	nd	nd	nd	nd	nd
Phenanthrene	18	4	2.3	1.1	<0.8	<1	<0.9	<0.7	<0.4	<10	<30	<20	<10	<4
Anthrancene	1.3	0.22	<0.1	<0.1	<0.07	<0.08	<0.06	<0.2	<0.2	<0.6	1.6	1.1	0.98	<0.08
Fluoranthene	8.3	0.75	0.37	0.19	<0.2	0.27	<0.2	<0.3	<0.1	<0.8	3.7	2.9	1.7	0.48
Pyrene	13	1.4	0.59	0.41	0.35	0.54	0.39	<0.2	<0.07	1.1	4.1	6	1.7	0.61
Non-genotoxic Lower	40.6	6.37	3.26	1.7	0.35	0.81	0.39	0	0	1.1	9.4	10	4.38	1.09
Non-genotoxic Middle	40.6	6.37	3.31	1.75	0.885	1.35	0.97	1.05	0.585	6.8	24.4	20	9.38	3.13
Non-genotoxic Upper	40.6	6.37	3.36	1.8	1.42	1.89	1.55	2.1	1.17	12.5	39.4	30	14.38	5.17
Benz[a]anthracene	1.1	<0.05	0.029	<0.006	<0.02	<0.01	<0.01	<0.03	<0.02	<0.1	<0.5	<0.2	0.2	<0.05
Cyclopenta[c,d]pyrene	0.5	<0.01	0.016	<0.004	<0.006	<0.005	<0.005	<0.02	<0.004	<0.04	0.18	0.067	0.09	<0.01
Chrysene	1.5	<0.1	0.065	0.029	<0.04	<0.03	<0.03	<0.02	<0.006	<0.2	<0.6	0.71	0.25	<0.05
5-Methyl Chrysene	0.032	<0.02	<0.02	<0.006	<0.007	<0.01	<0.009	nd	nd	nd	<0.07	<0.1	<0.03	<0.01
Benzo[b+k+j]fluorathene	0.75	<0.02	0.029	<0.003	<0.01	<0.006	<0.007	<0.007	<0.005	<0.08	<0.3	<0.3	<0.08	<0.02
Benzo[a]pyrene	1.2	<0.03	0.075	<0.007	<0.04	<0.01	<0.01	<0.04	<0.04	<0.1	<0.4	<0.3	<0.1	<0.02
Indeno[1,2,3-c,d]pyrene	1.6	<0.03	0.015	<0.001	<0.007	<0.004	<0.005	<0.03	<0.01	<0.06	<0.3	<0.1	<0.1	<0.04
Benzo[g,h,i]perylene	2.4	<0.05	0.028	<0.002	<0.01	<0.009	<0.009	<0.02	<0.03	<0.1	<0.5	<0.4	<0.1	<0.05
Dibenz[a,h]anthracene	0.1	<0.005	<0.002	<0.0003	<0.001	<0.0009	<0.001	<0.01	<0.01	<0.02	<0.05	0.035	<0.02	<0.007
Dibenzo[a,l]pyrene	0.22	<0.05	<0.05	<0.005	<0.03	<0.008	<0.02	<0.03	<0.02	<0.06	<0.4	<0.4	<0.08	<0.05
Dibenzo[a,e]pyrene	0.13	<0.003	0.022	0.0032	0.011	0.0021	0.0029	<0.04	<0.02	<0.02	<0.2	<0.2	<0.008	<0.006
Dibenzo[a,i]pyrene	<0.1	<0.003	<0.004	0.0028	0.003	<0.004	0.0014	<0.1	<0.02	<0.02	<0.03	<0.01	<0.005	<0.004

	Hamburger	Liver, sheep	Bread, white	Infant cereal	Carrots	Infant dessert, dairy based	Infant dessert, fruit based	Water, tap 1	Water, tap 2	Sausage, beef	Coconut, desiccated	Potato crisps	Bacon	Beef steak
Fresh Weight ng/g														
Dibenzo[a,h]pyrene	<0.1	<0.0008	<0.03	0.0081	<0.02	<0.006	<0.005	<0.2	<0.04	<0.04	<0.2	<0.07	<0.008	<0.002
Genotoxic Lower	9.532	0	0.279	0.0431	0.014	0.0021	0.0043	0	0	0	0.18	0.812	0.54	0
Genotoxic Middle	9.632	0.1859	0.332	0.06025	0.1095	0.05355	0.0598	0.2735	0.1125	0.42	1.955	1.852	0.8055	0.1595
Genotoxic Upper	9.732	0.3718	0.385	0.0774	0.205	0.105	0.1153	0.547	0.225	0.84	3.73	2.892	1.071	0.319
Total Lower bound	50.132	6.37	3.539	1.7431	0.364	0.8121	0.3943	0	0	1.1	9.58	10.812	4.92	1.09
Middle bound	50.232	6.5559	3.642	1.81025	0.9945	1.40355	1.0298	1.3235	0.6975	7.22	26.355	21.852	10.1855	3.2895
Total Upper bound	50.332	6.7418	3.745	1.8774	1.625	1.995	1.6653	2.647	1.395	13.34	43.13	32.892	15.451	5.489
Benzo[a]pyrene Lower	1.2	0	0.075	0	0	0	0	0	0	0	0	0	0	0
Benzo[a]pyrene Middle	1.2	0.015	0.075	0.0035	0.02	0.005	0.005	0.02	0.02	0.05	0.2	0.15	0.05	0.01
Benzo[a]pyrene Upper	1.2	0.03	0.075	0.007	0.04	0.01	0.01	0.04	0.04	0.1	0.4	0.3	0.1	0.02

	Lamb chops	Milk chocolate	Oil, canola	Peanut butter	Eggs, boiled	Tuna, canned in brine	Potatoes, cooked	Salt, table, non-iodised
Fresh Weight ng/g								
Acenapthylene	nd	nd	nd	nd	nd	nd	nd	nd
Acenaphthene	nd	nd	nd	nd	nd	nd	nd	nd
Fluorene	nd	nd	nd	nd	nd	nd	nd	nd
Phenanthrene	<3	16	<2	<4	<2	<9	<0.3	<0.03
Anthrancene	<0.07	1.1	<0.2	<0.3	<0.1	<0.2	<0.02	<0.003
Fluoranthene	0.45	4.9	<0.3	<0.6	<0.4	<0.7	<0.06	<0.03
Pyrene	0.62	4.7	<0.3	<0.9	<0.5	<0.4	<0.07	<0.05
Non-genotoxic Lower	1.07	26.7	0	0	0	0	0	0
Non-genotoxic Middle	2.605	26.7	1.4	2.9	1.5	5.15	0.225	0.0565
Non-genotoxic Upper	4.14	26.7	2.8	5.8	3	10.3	0.45	0.113
Benz[a]anthracene	<0.06	0.49	<0.06	<0.1	<0.04	<0.05	<0.004	<0.01
Cyclopenta[c,d]pyrene	<0.01	0.5	<0.01	<0.04	<0.009	<0.02	<0.002	<0.01
Chrysene	<0.07	1	<0.1	<0.2	<0.07	<0.1	<0.01	<0.02
5-Methyl Chrysene	<0.008	<0.04	<0.02	<0.03	<0.07	<0.01	<0.001	<0.001
Benzo[b+k+j]fluorathene	<0.04	0.27	<0.06	<0.08	<0.02	<0.05	<0.003	<0.02
Benzo[a]pyrene	<0.04	0.29	<0.08	<0.1	<0.02	<0.05	<0.003	<0.02
Indeno[1,2,3-c,d]pyrene	<0.04	<0.2	<0.08	<0.08	<0.02	<0.06	<0.004	<0.1
Benzo[g,h,i]perylene	<0.06	<0.3	<0.1	<0.2	<0.04	<0.09	<0.01	<0.5
Dibenz[a,h]anthracene	<0.008	<0.03	<0.01	<0.02	<0.009	<0.02	<0.002	<0.01
Dibenzo[a,l]pyrene	<0.04	<0.1	<0.08	<0.1	<0.009	<0.06	<0.004	<0.01
Dibenzo[a,e]pyrene	<0.006	<0.01	<0.01	<0.07	<0.006	<0.04	<0.001	<0.01
Dibenzo[a,i]pyrene	<0.003	<0.05	<0.06	<0.07	<0.04	<0.05	<0.002	<0.007
Dibenzo[a,h]pyrene	<0.003	<0.07	<0.06	<0.1	<0.06	<0.09	<0.003	<0.009
Genotoxic Lower	0	2.55	0	0	0	0	0	0
Genotoxic Middle	0.194	2.95	0.365	0.595	0.2065	0.345	0.0245	0.3635
Genotoxic Upper	0.388	3.35	0.73	1.19	0.413	0.69	0.049	0.727

	Lamb chops	Milk chocolate	Oil, canola	Peanut butter	Eggs, boiled	Tuna, canned in brine	Potatoes, cooked	Salt, table, non-iodised
Total Lower bound	1.07	29.25	0	0	0	0	0	0
Middle bound	2.799	29.65	1.765	3.495	1.7065	5.495	0.2495	0.42
Total Upper bound	4.528	30.05	3.53	6.99	3.413	10.99	0.499	0.84
Benzo[a]pyrene Lower	0	0.29	0	0	0	0	0	0
Benzo[a]pyrene Middle	0.02	0.29	0.04	0.05	0.01	0.025	0.0015	0.01
Benzo[a]pyrene Upper	0.04	0.29	0.08	0.1	0.02	0.05	0.003	0.02

Note: 'nd' values have been assigned a value of zero in all cases as concentrations could not be determined due to matrix interference. For values reported as less than the limit of detection (<LOD), lower-, middle- and upper-bound values have been assigned.

APPENDIX 6: FOODS ANALYSED AND CORRESPONDING NATIONAL NUTRITION SURVEY FOODS

Table A5: Mapping of foods analysed

Food Category	Foods analysed	NNS foods represented
Dairy products	Milk, full fat	All fluid milks, whole
		Full fat flavoured milks
	Milk, modified, low fat	All fluid milk, reduced or low fat
		Reduced or low fat flavoured milks
	Cheese, cheddar, full fat	Ripened cheeses
		Unripened cheeses
		Processed cheeses
		Whey cheese
	Cream, pure (not thickened)	All cream (whipped, thickened , sour)
	Yoghurt, fruit, full fat	Yoghurt (plain, flavoured, frozen, full fat, skim, fromage frais)
Ice cream	Ice Cream, full fat, vanilla	Ice cream (regular, skim, flavoured, tub or stick)
		Ice confection
		Thick shakes
Edible oils and oil emulsions	Butter, regular	Butter (regular, flavoured, reduced salt, salt free)
	Margarine or margarine spread, polyunsaturated	Margarine, polyunsaturated
		Margarine, monounsaturated
		Margarine ,spreads
		Commercial fats
		Not specified fats
	Oil, canola	All oils (vegetable, seed, nut) including single source or blended
Vegetables	Carrots, cooked	All vegetables (raw, cooked , canned, juices) except potatoes
	Potatoes, cooked	Potato (boiled, baked, canned, mashed)
		Potato (battered, crumbed, patties)
		Salad potato
		Sweet potato
	Coconut, desiccated	All coconut (dry, fresh, milk, cream, canned)
Chocolate	Chocolate (milk)	Chocolate, milk (bars, filled, coated)
		Chocolate, carbohydrate-modified
Breads and Bakery Products	Bread, white	All "regular breads and rolls", including wholemeal or whole grain breads and products
		English style muffins, "crumpets", "flat breads", "sweet breads and buns", 'tortilla and corn bread".
		Doughnuts
Meat and Meat Products	Bacon	All "bacon"
		Ham
		All salamis, cabanossi

Meat and Meat Products	Beef steak, rib/ribeye/sirloin, grilled	All "beef steak", "beef brisket", "beef silverside", "beef patty (meat only)", "minced meats"
		All beef "corned", "smoked", "deli sliced", "cooked"
		All "veal", "kangaroo", "rabbit", "venison"
	Lamb chops, loin, grilled	Lamb or mutton "all chops", "minced", "smoked", "deli sliced"
	Pork chops, grilled	Pork "all chops", "minced", "smoked", "deli sliced"
	Chicken, breast, fillet	All chicken "raw", "cooked", "smoked", "deli sliced"
		All "duck", "quail", "emu" and "turkey"
	Liver, sheep	All liver and internal organs
	Sausage, beef	All "sausages" and sausage patties
		All plain "frankfurts, and saveloys"
Fish and Fish products	Fish fillets	All fish (cooked, uncooked, smoked)
		All crustacea and molluscs (cooked, uncooked, smoked)
	Salmon, canned in brine	Canned salmon pink or red in brine/water
	Tuna, canned in brine	Canned Tuna or sardine in brine/ water/ oil
Eggs and Egg products	Eggs, boiled	All eggs cooked or uncooked, scrambled omelettes
Salt	Salt, table, non-iodised	Salt
Foods for infants	Infant cereal, mixed	Infant Cereal, mixed
	Infant dessert, dairy based	Infant Dessert, dairy based
	Infant dessert, fruit	Infant Dessert, fruit
	Infant dinner, containing meat, chicken or fish	Infant Dinner, containing meat, chicken or fish
	Infant formula, powder, cow's milk based	Infant Formula, powder, cow's milk based
Water	Water, tap	Water, tap and bottled
		Mineral water
		Soda water
		Fruit drinks
		Soft drinks
		Cordials
Mixed Foods and snacks	Pizza, meat & vegetable-containing	All "pizza" and pies (vegetable, seafood, meat)
	Hamburger	All hamburgers and meat patties
	Potato crisps	Potato (plain, flavoured, restructured), corn chips pretzels, bhuja/snack mixes
	Peanut butter	All nuts

Note: Other mixed foods contain foods analysed and listed above (e.g. crumbed fish contains fish fillets). The proportions of the ingredients in these mixed foods, as determined by standard recipes in DIAMOND, were given the concentration of PAH assigned to that food.

APPENDIX 7: SUMMARY OF RESPONDENT AND CONSUMER NUMBERS AND BODY WEIGHTS

Table A6: Mean body weights in kilograms for each age-gender category assessed

Age group	Source	Mean Body Weight (kg)	
		Males	Females
9 month old	WHO, 2007	8.9	
2 years and above	1995 NNS	72	62
2-5 years	1995 NNS	18	17
6-12 years	1995 NNS	33	35
13-18 years	1995 NNS	65	59
19 years and above	1995 NNS	82	68

Note: For populations aged 2 years and above, individual body weight was used in the calculations

Table A7: Number of respondents and consumers of PAH per age gender group assessed from the 1995 NNS

Age group	Number of respondents		Number of consumers of PAH (% of all respondents)		
	Males	Females	Scenario	Males	Females
2 years and above	6,616	7,242	LB	6,615	7,242
			MB	6,615	7,242
			UB	6,596	7,242
2-5 years	380	413	LB	380 (100)	413 (100)
			MB	380 (100)	413 (100)
			UB	380 (100)	413 (100)
6-12 years	664	622	LB	662 (99.7)	619 (99.5)
			MB	664 (100)	622 (100)
			UB	664 (100)	622 (100)
13-18 years	491	437	LB	490 (99.8)	433 (99.1)
			MB	491 (100)	437 (100)
			UB	491 (100)	437 (100)
19 years and above	5,081	5,770	LB	5,064 (99.7)	5,741 (99.5)
			MB	5,080 (99.98)	5,770 (100)
			UB	5,081 (99.98)	5,770 (100)

Note: In some cases there are fewer consumers of PAH for the lower bound scenario. This is because of the way DIAMOND counts consumers. Even if a respondent consumed a food that was analysed for PAH, if the lower bound concentration assigned to that food was a zero, a respondent does not get counted as a consumer of PAH for that food.

APPENDIX 8: CONSTRUCTION OF THE INFANT DIET – FURTHER DETAILS

Table A8: Model diet for Australian infants aged 9 months

Food*	Respondent mean consumption (grams/day)
Bacon	1.9
Beef steak, rib/ribeye/sirloin, grilled	1.1
Bread, white	19.4
Butter, regular	0.4
Carrots, cooked	19.8
Cheese, cheddar, full fat	3.8
Chicken breast	3.3
Coconut, desiccated	0.5
Cream, pure (not thickened)	0.9
Eggs, boiled	2.3
Fish fillets	0.9
Hamburger	0.03
Ice Cream, full fat, vanilla	5.6
Infant cereal, mixed	7.3
Infant dessert, dairy based	1.8
Infant dessert, fruit	2.0
Infant dinner	2.6
Infant formula, powder	544
Lamb chops, loin, grilled	0.6
Liver, sheep	0.02
Margarine/ margarine spread, polyunsaturated	1.4
Chocolate, milk type	2.1
Milk, full fat	0
Milk, modified, low fat	0
Oil, canola	0.3
Peanut butter	0
Pizza	3.6
Pork Chops, grilled	0.4
Potato crisps	2.3
Potato, cooked	12.3
Salt, table, non-iodised	0
Sausage, beef	2.4
Tuna, canned in brine	0.3
Water, tap	544
Yoghurt, fruit, full fat	10.1

* These are the foods as sampled. The food consumption amount for each sampled food represents the consumption of a larger group of foods (see Table A5 for details). Please note: food consumption amounts have been rounded.

Assumptions and limitations for the infant model diet

The model diet constructed for 9 month old infants, assumed that all milk consumption was in the form of infant formula; and that 50 % of the infant's energy intake comes from infant formula (Hitchcock *et al*., 1986).

Certain foods such as nuts, alcohol and tea, were removed from the diet since they are unsuitable for infants to consume (NHMRC, 2003). Consumption of breakfast cereals was assumed to be in the form of either infant cereal or single grain breakfast cereals, excluding bran-based cereals.

As the infant diet estimates a theoretical mean dietary exposure only, the 95th percentile dietary exposure to PAH was also estimated using the internationally accepted formula shown below (WHO, 1985):

95th percentile exposure = mean exposure x 2.5

Assigning PAH concentrations

The food groups and mapping are slightly different for the model infant diet compared to for Australians aged 2 years and above. However, the mapping followed the same principles. For example, the concentration for carrots was assigned to all vegetables other than potatoes and fruit was assigned a zero concentration.

APPENDIX 9: SUMMARY OF FOOD CONSUMPTION DATA

Table A9: Mean consumption (lower bound) for consumers only of each food analysed, derived from the 1995 NNS using DIAMOND

	Consumer mean consumption amount (grams/day)									
	2 years & above		2-5 years		6-12 years		13-18 years		19 years & above	
Food*	Male	Female	Male	Female	Male	Female	Male	Female	Males	Female
Bacon	51	36	36	29	39	33	55	33	53	37
Beef steak, rib/ribeye/sirloin, grilled	143	96	63	56	92	87	145	108	151	98
Bread, white	138	101	86	78	118	101	147	112	144	102
Butter, regular	16	11	6	6	10	8	15	9	18	12
Carrots, cooked	212	189	81	80	122	124	176	151	232	204
Cheese, cheddar, full fat	40	32	27	27	36	28	46	35	41	32
Chicken breast	128	95	66	61	91	84	140	102	134	97
Coconut, desiccated	26	18	7	6	11	7	28	5	31	21
Cream, pure (not thickened)	26	22	9	11	16	14	23	27	30	24
Eggs, boiled	27	21	12	14	20	17	26	20	30	22
Fish fillets	118	92	59	39	82	60	135	84	122	98
Hamburger	197	151	97	95	158	126	194	157	209	159
Ice Cream, full fat, vanilla	132	94	82	71	141	121	207	125	123	85
Infant cereal, mixed	8	100	NC	NC	NC	NC	NC	NC	8	100
Infant dessert, dairy based	60	81	66	100	NC	68	NC	NC	58	58
Infant dessert, fruit	60	74	34	92	20	138	NC	NC	115	13
Infant dinner	110	33	110	NC	NC	NC	NC	NC	NC	33
Infant formula, powder	103	231	103	NC	NC	NC	NC	310	NC	152
Lamb chops, loin, grilled	125	85	59	63	81	84	117	84	133	87
Liver, sheep	61	39	4	6	29	57	102	67	64	39
Margarine/ margarine spread, polyunsaturated	23	16	13	11	19	16	25	17	24	16
Chocolate (milk)	42	38	24	22	32	33	53	44	46	40
Milk, full fat	274	194	402	342	343	261	417	242	238	169
Milk, modified, low fat	292	225	310	300	373	291	422	323	276	216
Oil, canola	9	7	3	3	6	5	9	7	10	7
Peanut butter	16	12	7	7	10	10	12	13	18	13
Pizza	229	159	114	99	180	143	224	152	244	169
Pork chops, grilled	110	81	49	47	65	83	130	73	116	83
Potato crisps	43	37	34	31	33	34	50	38	49	38
Potato, cooked	196	147	122	106	173	168	258	176	196	145
Salt, table, non-iodised	1	1	1	<1	2	<1	2	2	1	1
Sausage, beef	109	82	63	59	86	76	106	79	118	87
Tuna, canned in brine	93	71	55	30	73	81	241	87	91	71
Water, tap**	1,303	1,125	915	800	1,160	1,073	1,744	1,352	1,308	1,137
Yoghurt, fruit, full fat	141	138	111	107	123	138	197	161	144	140

NC = Not consumed

* These are the foods as sampled. These foods have been mapped to other foods (see Table A5 for details).

** Bottled water, mineral water, soda water, fruit drinks, soft drinks and cordials are also represented in the mean consumption for water, tap.

APPENDIX 10: PERCENT CONTRIBUTION OF FOODS TO TOTAL PAH DIETARY EXPOSURE

Table A10: Contribution of each food to PAH dietary exposures (lower bound concentration) for each age/gender group assessed.

	Percent contribution to total PAH dietary exposure (%)									
	2 years & above		2-5 years		6-12 years		13-18 years		19 years & above	
FOOD	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
Bacon	4	4	4	4	3	3	3	2	5	4
Beef steak, rib/rib eye/sirloin, grilled	3	3	2	2	2	2	2	2	3	3
Bread, white	23	26	27	29	24	23	19	19	24	27
Butter, regular	1	1	<1	<1	<1	<1	<1	<1	1	1
Carrots, cooked	4	5	2	3	2	3	2	3	4	6
Cheese, cheddar, full fat	1	1	1	2	1	1	1	1	1	2
Chicken breast	6	7	4	5	5	5	5	5	7	8
Coconut, desiccated	1	1	1	<1	1	<1	1	<1	1	1
Cream, pure (not thickened)	1	1	1	1	1	1	1	1	1	1
Eggs, boiled	0	0	0	0	0	0	0	0	0	0
Fish fillets	4	4	2	2	2	2	2	2	5	5
Hamburger	32	22	25	18	28	25	37	32	33	21
Ice cream, full fat, vanilla	0	0	0	0	0	0	0	0	0	0
Infant cereal, mixed	<1	<1	NC	NC	NC	NC	NC	NC	<1	<1
Infant dessert, dairy based	<1	<1	<1	<1	NC	<1	NC	NC	<1	<1
Infant dessert, fruit	<1	<1	<1	<1	<1	<1	NC	NC	<1	<1
Infant dinner	<1	<1	<1	NC	NC	NC	NC	NC	NC	<1
Infant formula, powder	0	0	0	NC	NC	NC	NC	0	NC	0
Lamb chops, loin, grilled	1	1	<1	<1	<1	1	<1	<1	1	1
Liver, sheep	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Margarine/ margarine spread, polyunsaturated	0	0	0	0	0	0	0	0	0	0
Chocolate (milk)	12	17	19	22	20	24	17	22	10	15
Milk, full fat	0	0	0	0	0	0	0	0	0	0
Milk, modified, low fat	0	0	0	0	0	0	0	0	0	0
Oil, canola	0	0	0	0	0	0	0	0	0	0
Peanut butter	0	0	0	0	0	0	0	0	0	0
Pizza	2	2	1	2	3	2	3	2	2	2
Pork chops, grilled	0	0	0	0	0	0	0	0	0	0
Potato crisps	3	4	9	7	7	9	5	6	2	2
Potato, cooked	0	0	0	0	0	0	0	0	0	0
Salt, table, non-iodised	0	0	0	0	0	0	0	0	0	0
Sausage, beef	1	1	1	2	1	1	1	1	1	1
Tuna, canned in brine	0	0	0	0	0	0	0	0	0	0
Water, tap**	0	0	0	0	0	0	0	0	0	0
Yoghurt, fruit, full fat	<1	1	1	1	<1	<1	<1	<1	<1	1

NC = this food had a PAH concentration however it was not consumed and therefore did not make a contribution to PAH dietary exposure.

0 = this food was consumed however there was a lower bound concentration of zero for this food and therefore did not make a contribution to PAH dietary exposure.

** Bottled water, mineral water, soda water, fruit drinks, soft drinks and cordials are also represented in the percentage contribution to PAH dietary exposure for water, tap.