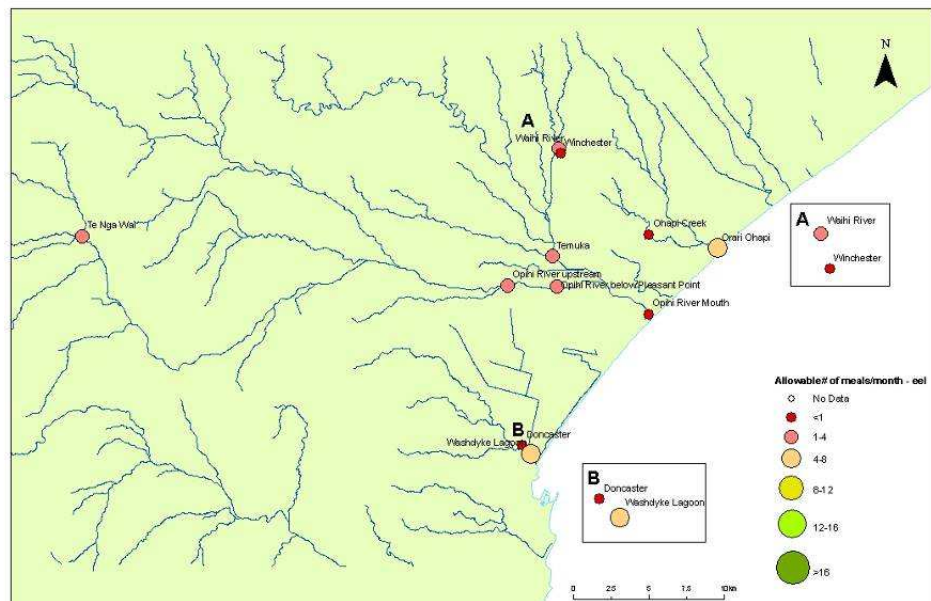


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## Contaminants in kai – *Arowhenua rohe* Part 2: Risk Assessment

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NIWA Client Report: HAM2010-116  
September 2010

NIWA Project: HRC08201

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# **Contaminants in Kai – Arowhenua rohe Part 2: Risk Assessment**

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# Contents

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Executive Summary	iv
1. Introduction	1
1.1 Background	1
1.2 Synopsis of first report	1
1.3 Aim of this study	2
2. Methods	3
2.1 Sampling	3
2.2 Analysis of contaminants in kai and sediment	3
2.3 Risk Assessment	4
3. Discussion on contaminants in risk assessment	8
3.1 Organochlorine pesticides	8
3.1.1 DDT	8
3.1.2 Aldrin/dieldrin	9
3.1.3 Lindane ( $\gamma$ -hexachlorocyclohexane; $\gamma$ -HCH)	9
3.1.4 Chlordane	10
3.1.5 Hexachlorobenzene (HCB)	10
3.2 PCBs	10
3.3 Heavy metals	11
3.3.1 Cadmium	11
3.3.2 Mercury	11
3.3.3 Arsenic	12
3.3.4 Lead	12
4. Risk Assessment	13
4.1 Arowhenua contaminant data	13
4.2 Arowhenua consumption data	13
4.3 Arowhenua catchment risk assessments	16
4.4 Individual site risk assessments	18
4.4.1 Temuka region	20
4.4.2 Washdyke Region	33
4.4.3 Winchester Region	38
4.4.4 Reference Site: Te Nga Wai	43
4.5 Mercury contamination as a component of risk	45
5. Implications of risk assessment to Arowhenua iwi	47
6. Conclusions	56
7. References	59
8. Abbreviations	61
9. Glossary	63
10. Appendices	66

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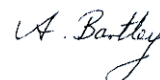
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## Executive Summary

Gathering of wild kai (food) has always been of importance to Māori society. With kai increasingly susceptible to accumulation of anthropogenic contaminants, the potential impact of environmental contamination on the resident wild kai and, in turn, on Māori consuming them, is also likely to increase. However, this issue has not been investigated to date.

Many toxic contaminants are stored in the lipids of biota and can biomagnify up through the food-chain, increasing the risk of consuming higher predatory animals, such as eel and trout. Bioaccumulative contaminants that are of potential concern include organochlorine pesticides (DDTs, dieldrin and lindane), polychlorinated biphenyls (PCBs), pentachlorophenol, dioxins, polyaromatic hydrocarbons (PAHs), and selected heavy metals such as mercury, arsenic, cadmium, lead, copper and zinc.

The aim of this project was to quantify the risk to local Māori of consuming wild kai gathered from the rohe (territory of iwi or hapū) of Arowhenua, New Zealand (NZ). A companion report (Stewart et al., 2010) presented data assessing the concentrations of selected heavy metal and organic contaminants in the aquatic environment and how these contaminant levels related to tissue concentrations in resident kai. This report describes the assessment process that was undertaken to quantify the potential risk to local Māori from the consumption of wild kai gathered from the rohe of Arowhenua.

Data on local consumption rates was derived using a questionnaire on kai consumption rates and portion sizes. Local average consumption rates of wild kai were calculated as 6.1, 4.0 and 4.7 g/day for eels, trout and flounder, respectively. Watercress consumption was calculated at 6.0 g/day. Total fish consumption was in the NZ 'high' consumption category, but only 13% of this was from wild caught fish. Meal sizes were calculated at 213 g/meal for all fish species and 175 g/meal for watercress, which equated to actual consumption rates of 0.9, 0.6, 0.7 and 1.0 meals/month for eel, trout, flounder and watercress, respectively.

A risk assessment was carried out on the contaminant data, using established US EPA formulae. The risk assessment calculated *allowable monthly meals* for the whole catchment, using median and 95<sup>th</sup> percentile contaminant concentration data to approximate harvesting of kai with random contamination concentrations that might be expected from harvesting randomly across all sites (median) or predominantly from the most contaminated kai (95<sup>th</sup> percentile), that might be expected from harvesting predominantly at the most contaminated sites. Furthermore, a risk assessment was performed for each species harvested from each site to gain an understanding of potential "hotspots" in the catchment.

The results of the risk assessment were clear. In terms of the whole catchment, if harvesting was carried out randomly across all sites *and* consumption rates were as calculated from the questionnaire data, then there is *no significant risk* to members of Arowhenua iwi. However, if harvesting were to occur predominantly at the most contaminated sites *and* consumption rates were as calculated from the questionnaire data, then a *significant risk* exists for consumption of eel. Trout are also a probable cause for concern with safe consumption limit being virtually the same as the consumption rate. The risk of eating eels in Arowhenua was greater than other species, with 8 out of 10 eels sampled allowing for less than 4 meals per month, which is less than 1 meal per week. The area of Washdyke consistently showed the highest risk of consumption of kai, however significant risk of consuming kai was not limited to this area.

Te Nga Wai proved to be a satisfactory reference site (i.e., as far removed from human influence as practical), with generally lower contamination of sediments and kai than the other sites. The exception was mercury concentrations in eels, for which Te Nga Wai was roughly the same as other sites, suggesting that mercury uptake by eels in the rohe of Arowhenua is largely from background (i.e., non-elevated) levels of mercury.

Limitations of this study were the small number of Māori who were able to complete the questionnaire (which therefore limits the reliability of the consumption rate estimates) and the low number of specimens collected of each species - typically only a single specimen per site. Because of the inherent assumptions and associated error involved with any risk assessment process, it would have been beneficial to collect multiple specimens at each site, including other important species which were out of season, such as whitebait. This would have enabled a more robust assessment of the risk, both spatially and seasonally, associated with consuming kai gathered from the Arowhenua area. However, notwithstanding these limitations, this study has provided a valuable screen of potential risks associated with kai consumption in the Arowhenua rohe.

The major recommendations that can be made from this study involve:

- obtaining more species (e.g., both long fin and short fin eels), samples from more sites and with larger sample sizes of kai to provide a more representative spatial assessment of kai contamination in the region
- obtaining more robust datasets of contaminants - PCBs in all fish species, arsenic and mercury speciation
- obtaining more robust consumption data and meal size portions through participation of larger numbers of Māori and non-Māori consumers of wild kai in the questionnaire, and
- conducting a risk assessment for total fish diet which incorporates both wild and commercial dietary consumption.

## 1. Introduction

### 1.1 Background

Wild kai (food), gathered from the sea, rivers, and lakes, has always been of significant cultural, recreational and economic importance in both traditional and contemporary Māori society. Today, such resources are increasingly susceptible to contamination, as a consequence of urban expansion or land use changes in agricultural catchments. The impact of environmental contamination on the resident wild kai and, in turn, on Māori consuming them, however, has not been investigated to date.

Many toxic contaminants are stored in the lipids of biota and can biomagnify up through the food-chain increasing the risk of consuming higher predatory animals, such as eel and trout. Bioaccumulative contaminants that are of potential concern include organochlorine pesticides (DDTs, dieldrin and lindane), polychlorinated biphenyls (PCBs), pentachlorophenol, dioxins, polyaromatic hydrocarbons (PAHs), and selected heavy metals such as mercury, arsenic, cadmium, lead, copper and zinc.

### 1.2 Synopsis of first report

This report is the second of two reports on contaminants in kai from the rohe of Arowhenua. The first is a data report (Stewart et al., 2010), with key findings summarised below.

A survey of past and present kai consumption patterns was undertaken by questionnaire to establish historic and contemporary consumption rates of key species. The levels of bioaccumulative contaminants were characterised in a number of commonly gathered fish (shortfin and longfin eel, brown trout, black flounder) and plant species (watercress) from 12 sites throughout the rohe of Arowhenua, as well as in associated aquatic sediments.

Local average consumption rates of wild kai were calculated as 6.1, 4.0, 4.7 and 6.0 g/day for eels, trout, flounder and watercress, respectively. The consumption rates of wild caught fish were considerably lower than the average NZ consumption rate of 32 g/day. In contrast, the average total fish consumption rate from the survey was 43 g/day, putting these rates into the NZ 'high' consumption category and highlighting that wild caught kai is only a small proportion of the main source of aquatic food for local Māori.

The following broad conclusions could be made about the contaminant concentrations from the first report:

- 1) sediment contaminant concentrations were generally below the Australian and New Zealand Environment Conservation Council (ANZECC) Interim Sediment Quality Guideline (ISQG) (ANZECC 2000) guidelines, with a few exceptions:
  - a) Zinc exceeded ANZECC ISQG-low guideline concentrations (200 mg/kg) at Doncaster (220 mg/kg).
  - b) Total Organic Carbon (TOC)-normalised total DDT ( $\Sigma$ DDT) concentrations reached or exceeded ANZECC ISQG-low guideline concentrations (1.6  $\mu$ g/kg) at Washdyke Lagoon (8.3  $\mu$ g/kg), Washdyke Creek (3.8  $\mu$ g/kg), Doncaster (3.3  $\mu$ g/kg) and Winchester (1.6  $\mu$ g/kg).
- 2) contaminant uptake was species-specific, with the following trends in concentrations typically being observed:
  - a) for  $\Sigma$ DDT: eel >> trout = flounder
  - b) for mercury: eel > trout > flounder > watercress
  - c) for arsenic: trout > flounder > watercress > eel
  - d) for cadmium, lead and chromium: watercress >> all fish.
- 3) the sites which consistently had the highest contamination in collected kai were the Washdyke region, followed by Winchester and Ohapi Creek
- 4) Te Nga Wai was a valid reference site with consistently lower contaminant concentrations in both sediments and kai.

### 1.3 Aim of this study

The overall aim of this study was to determine the risk to Māori and non-Māori of consuming key kai species which are harvested from sites around the rohe of Arowhenua, in South Canterbury. The contaminant data from a companion report (Stewart et al., 2010) forms the basis for a cumulative risk assessment, of which the implications to human health are presented in this report.

## 2. Methods

### 2.1 Sampling

The focus of this study was South Canterbury, New Zealand, an area including the towns of Timaru (population 36,500), Temuka (pop. 4000) and Geraldine (pop. 2200) south of the Rangitata River. In the wider Timaru district of 42,867 people, those who identify as Māori make up 6.1% of the population (Statistics New Zealand 2006).

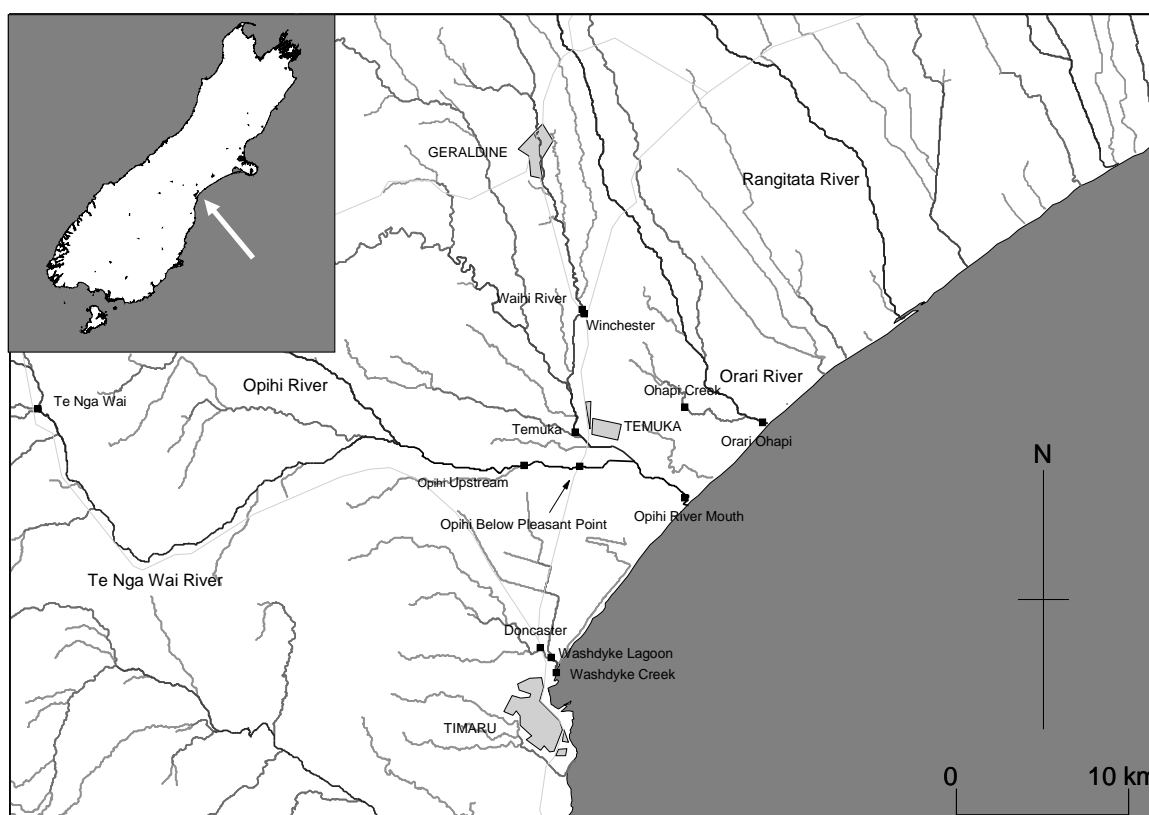
The major River networks include the Temuka, Orari and Opihi Rivers and Te Nga Wai River located further inland. The full information pertaining to collection of biota is contained in Appendix 2, however briefly, we surveyed 12 sites and collected a total of 9 short fin eels (*Anguilla australis*) 1 long fin eel (*Anguilla dieffenbachii*), 5 brown trout (*Salmo trutta*) and 4 black flounder (*Rhombosolea retiaria*). Washdyke Creek did not contain any kai of suitable size for consumption, so no kai were collected from this site. All samples were kept on ice and then frozen prior to processing. Collections of fish were undertaken in 2009 either between 12<sup>th</sup> and 14<sup>th</sup> of May, or on the 3<sup>rd</sup> of June. Watercress (*Nasturtium officinale*) was collected from eight sites between 12<sup>th</sup> and 14<sup>th</sup> of May. Composite sediment samples were collected from all sites at the time of biota collection, with the top 0-20 mm being sampled from areas where the fine fraction of deposited sediments was likely to be high.

### 2.2 Analysis of contaminants in kai and sediment

Fish and sediment samples were analysed for a range of organochlorine compounds, including DDT and DDT metabolites (p,p'-DDT, p,p'-DDE, p,p'-DDD and o,p isomers), chlordanes (cis/trans nonachlor, cis/trans chlordane) and chlordane metabolites (heptachlor, cis/trans heptachlor epoxide), hexachlorobenzene (HCB), lindane ( $\gamma$ -hexachlorocyclohexane;  $\gamma$ -HCH) and dieldrin. The samples were analysed for eight selected heavy metals; arsenic (As), cadmium (Cd), chromium (Cr), copper (Cu), lead (Pb), mercury (Hg), nickel (Ni) and zinc (Zn). Eel tissue was also analysed for selected PCBs (32 congeners ranging from PCB8 to PCB209). Watercress was analysed for the eight heavy metals only.



**Figure 1:** Collection sites in this study with map of South Island of New Zealand (inset) showing location of South Canterbury region.



### 2.3 Risk Assessment

For the risk assessments, contaminant concentrations were converted from dry weight to wet weight concentrations using water content values measured for each of the various kai. Accordingly, unless otherwise specified, all concentrations and kai consumption rates in this report are calculated on a wet weight basis.

Human health risk assessment is defined by the US EPA as a four step process:

1. *Hazard identification.* This assesses the likelihood that exposure to specific chemicals under defined exposure conditions will pose a threat to human health.
2. *Dose-response assessment.* Results in the derivation of toxicity values such as cancer potencies and non-cancer reference doses by evaluating the results of human and animal studies with controlled and quantified exposures.

3. *Exposure assessment.* This covers a range of assessments including chemical occurrences in fish, geographic distribution of contaminated fish, individual or population exposure assessment, multiple species exposure and multiple chemical exposure.
4. *Risk characterization.* In general, the risk characterization step of the risk assessment process combines the information for hazard identification, dose-response assessment, and exposure assessment in a comprehensive way that allows the evaluation of the nature and extent of risk.

Points 1 and 2 above are continually being modified as further information is incorporated and this is carried out by the US EPA and other environmental agencies.

Exposure assessment (point 3) in this study was limited, due to the small sample size and in many cases only a single specimen collected per site.

Risk characterisation (point 4) was performed by following established US EPA procedures, calculating risk for both cancer and non-cancer health endpoints. Cancer oral slope factor (CSF) and reference doses (RfD) for chronic non-cancer oral exposure were obtained from US EPA Integrated Risk Information System (IRIS) (US EPA 2010), with the exception of CSF and RfD for PCBs and RfD for mercury which were based on US EPA guidelines (US EPA 2000). As no information for the heavy metal lead could be obtained and lindane was not detected in any sample these two contaminants were removed from the risk assessment calculations.

For carcinogenic effects we calculated both *individual* contaminant consumption limits (see Appendix 1 for values) and *additive* consumption limits for each species. An additive risk consumption limit is possible for carcinogenic chemicals as the effects (i.e., the development of cancer) is the same. *Individual* contaminant consumption limits were calculated using equation 2.3.1, based on US EPA equation 3-1, while *additive* consumption limits were calculated using equation 2.3.2, based on US EPA equation 3-14 (US EPA 2000).

$$(2.3.1) \quad CR_{lim} \text{ (individual)} = \frac{ARL \bullet BW}{Cm \bullet CSF}$$

$$(2.3.2) \quad CR_{lim} \text{ (additive)} = \frac{ARL \bullet BW}{\sum_{m=1}^x Cm \bullet CSF}$$

where

$CR_{lim}$  = maximum allowable fish consumption rate (kg/day)

ARL = maximum acceptable lifetime risk level (unitless)

BW = consumer body weight (kg)

$C_m$  = concentration of chemical contaminant  $m$  in species (mg/kg)

CSF = cancer slope factor,  $([mg/kg\text{-day}]^{-1})$ .

Body weight (BW) was set at 80 kg based on a previous study (Kim & Smith 2006). An “acceptable” lifetime cancer risk (ARL) level of  $10^{-6}$  (1 in 1,000,000) is considered by some countries or institutions as negligible (World Health Organization 2009) and a level of  $10^{-5}$  (1 in 100,000) is set by US EPA in their “Guidance for assessing chemical contaminant data for use in fish advisories” (US EPA 2000). As such, we set the ARL at  $10^{-5}$  for Arowhenua risk calculations.

For assessment of non-carcinogenic risks an additive approach is only possible if effects are the same. Organochlorines such as DDT, lindane and dieldrin cause liver lesions, whereas the heavy metal mercury causes hand tremors and/or memory problems while arsenic causes hyper-pigmentation (US EPA 2010). As these effects are notably different, non-carcinogenic risk assessment was calculated on a single contaminant class basis only, using equation 2.3.3, based on US EPA equation 3-3 (US EPA 2000);

$$(2.3.3) \quad CR_{lim} = \frac{RfD \bullet BW}{Cm}$$

where

$CR_{lim}$  = maximum allowable fish consumption rate (kg/day)

RfD = reference dose (mg/kg-day)

BW = consumer body weight (kg)

$C_m$  = measured concentration of chemical contaminant  $m$  in a given species of fish (mg/kg).

The maximum allowable consumption rate  $CR_{lim}$  (kg/day) was converted into a more useful measure of meals/month using equation 2.3.4;

$$(2.3.4) \quad CR_{lim} \text{ (meals/month)} = \frac{CR_{lim} \text{ (kg/day)}}{MS \bullet \text{ days/month}}$$

where

MS = meal size (kg)

days/month = 30

### 3. Discussion on contaminants in risk assessment

This report is concerned with contaminants that are a long term risk to human health. As such, the contaminants selected are environmentally persistent (slow to break down), have a tendency to bioaccumulate in biota and are known (or suspected) to be toxic to humans.

Bioaccumulative contaminants that are covered in this report are the organochlorine pesticides (DDTs, dieldrin, lindane and chlordane), polychlorinated biphenyls (PCBs) and the heavy metals mercury, arsenic, cadmium and lead.

The Agency for Toxic Substances & Disease Registry (ATSDR) is a federal public health agency of the U.S. Department of Health and Human Services. The ATSDR has a toxic substances portal for useful information about toxic substances and how they affect human health (ATSDR 2010). All contaminants that are covered in this risk assessment are included in this portal and a brief summary of each is supplied below, supported, where available, with locally relevant information (e.g., use and potential sources).

#### 3.1 Organochlorine pesticides

The organochlorine pesticides and PCBs listed above are all listed under the Stockholm Convention on Persistent Organic Pollutants (POPs), a global treaty (which New Zealand ratified in 2004) to protect human health and the environment from chemicals that remain intact in the environment for long periods, become widely distributed geographically, accumulate in the fatty tissue of humans and wildlife, and have adverse effects on human health and/or the environment. Exposure to POPs can lead to serious health effects including certain cancers, birth defects, dysfunctional immune and reproductive systems, greater susceptibility to disease and diminished intelligence (Stockholm Convention 2010).

##### 3.1.1 DDT

Dichlorodiphenyltrichloroethane (DDT) is a pesticide that was used extensively throughout the world to control insects that affect agriculture and horticulture. It is still used in some countries as a control measure for insects, such as mosquitoes, that carry malaria. DDT was used largely as an insecticide to control grass grubs and porina caterpillars in NZ, with its use restricted in 1970 and finally banned in 1989 (Taylor et al., 1997). DDT breaks down in the environment to dichlorodiphenyldichloroethylene (DDE) and dichlorodiphenyldichloroethane (DDD), all of which persist for years.

Exposure to DDT, DDE, and DDD occurs mostly from eating foods containing low concentrations of these compounds, particularly meat, fish and poultry. High levels of DDT can affect the nervous system causing excitability, tremors and seizures. In women, DDE can cause a reduction in the duration of lactation and an increased chance of having a premature baby (ATSDR 2010). DDT is classified by US EPA as a probable human carcinogen (US EPA 2010).

### 3.1.2 Aldrin/dieldrin

Aldrin and dieldrin are insecticides with similar chemical structures. Aldrin rapidly breaks down to dieldrin in the body and in the environment. Exposure to aldrin and dieldrin occurs mostly through eating contaminated foods, such as root crops, fish, or seafood. Aldrin and dieldrin accumulate in the body after years of exposure and can affect the nervous system (ATSDR 2010). The US EPA has classified dieldrin as a probable human carcinogen (US EPA 2010).

In NZ, aldrin and dieldrin were introduced in 1954 for use as stock remedies in sheep sprays or dips for controlling sheep ectoparasites. Aldrin was used to control horticultural pests such as wireworm, soldier fly and blackvine weevil, and in limited quantities, to control household spiders. Dieldrin was used for controlling carrot rust fly, crickets and armyworm and was also used for timber preservation (mostly in plywood glues) and to mothproof carpets (Buckland et al., 1998).

### 3.1.3 Lindane ( $\gamma$ -hexachlorocyclohexane; $\gamma$ -HCH)

Lindane ( $\gamma$ -HCH) is one of eight isomers formed during the manufacture of technical grade (crude) hexachlorocyclohexane (HCH). Technical grade HCH typically contained about 10–15% of  $\gamma$ -HCH. It is used as an insecticide on fruit, vegetables, and forest crops (ATSDR 2010).

In NZ, lindane was used as an insecticide in agriculture for the control of lice on cattle, ectoparasites (lice, keds and blowflies) in sheep and grass grub in pasture. Lindane was also used for insect control on vegetable and fruit crops, and as an active component of fly sprays, flea control and carpet moth products for household use. Technical grade HCH was not officially used in New Zealand, although many dip sites show evidence of the use of crude HCH (Buckland et al., 1998).

Exposure to hexachlorocyclohexane (HCH) happens mostly from eating contaminated food or by breathing contaminated air in the workplace. Exposure to high levels of HCH can cause blood disorders, dizziness, headaches, seizures, and changes in the

levels of sex hormones. The US EPA has determined there is not enough evidence to determine whether lindane is a human carcinogen (US EPA 2010).

### **3.1.4 Chlordane**

Technical chlordane is a mixture of chlordane and many related chemicals, of which the composition varies. Exposure to chlordane occurs mostly from eating contaminated foods, such as root crops, meats, fish, and shellfish, or from touching contaminated soil. High levels of chlordane can cause damage to the nervous system or liver (ATSDR 2010). The US EPA classes technical chlordane as a probable human carcinogen (US EPA 2010).

In NZ, chlordane was used as a broad spectrum agricultural insecticide, in the timber industry as a treatment against termites and borer, and as an insecticide in glues used for the manufacture of plywood, finger jointed and laminated timber (Buckland et al., 1998).

### **3.1.5 Hexachlorobenzene (HCB)**

HCB was widely used as a pesticide to protect the seeds of onions, sorghum, wheat and other grains against fungus. It was also used to make fireworks, ammunition, and synthetic rubber (ATSDR 2010). In NZ, HCB was used experimentally between 1970 and 1972 as a seed dressing fungicide for cereal grain (Buckland et al., 1998). Exposure to HCB occurs primarily from eating contaminated food. Much lower exposures can occur from drinking water and breathing air contaminated with HCB (ATSDR 2010).

The main health effect from eating food contaminated with HCB is a liver disease called porphyria cutanea tarda. The USEPA has classified HCB as a probable human carcinogen (US EPA 2010).

## **3.2 PCBs**

Polychlorinated biphenyls (PCBs) are mixtures of up to 209 individual chlorinated compounds, referred to as congeners. PCBs have been used as coolants and lubricants in transformers, capacitors, and other electrical equipment because they have low flammability and are good electrical insulators (ATSDR 2010).

Exposure to PCBs can be via multiple pathways. Skin exposure can occur via old electrical devices (>30 years old) that leak small amounts of PCBs and in the workplace where contact may be made with equipment or devices containing PCBs.

Ingestion of PCBs is largely via contaminated food (fish, meat and dairy) and drinking contaminated well water, while inhalation exposure can occur by breathing air near hazardous waste sites (ATSDR 2010).

Health effects that have been associated with exposure to PCBs include acne-like skin conditions in adults and neurobehavioral and immunological changes in children (ATSDR 2010). The US EPA classifies PCBs as a probable human carcinogen (US EPA 2010).

### **3.3 Heavy metals**

#### **3.3.1 Cadmium**

Cadmium is a natural element in the Earth's crust. It is usually found as a mineral combined with other elements such as oxygen (cadmium oxide), chlorine (cadmium chloride), or sulfur (cadmium sulfate, cadmium sulfide). All soils and rocks, including coal and mineral fertilizers, contain some cadmium. Most cadmium used in the United States is extracted during the production of other metals like zinc, lead, and copper. Cadmium does not corrode easily and has many uses, including batteries, pigments, metal coatings, and plastics (ATSDR 2010).

Exposure to cadmium happens mostly in the workplace where cadmium products are made. The general population is exposed from breathing cigarette smoke, eating cadmium contaminated foods or drinking cadmium contaminated water (ATSDR 2010).

Long-term exposure to lower levels of cadmium in air, food, or water leads to accumulation of cadmium in the kidneys and possible kidney disease. Other long-term effects are lung damage and fragile bones (ATSDR 2010). The US EPA classifies cadmium as a probable human carcinogen (US EPA 2010).

#### **3.3.2 Mercury**

Mercury is a naturally occurring metal which has several forms. Mercury combines with other elements, such as chlorine, sulfur, or oxygen, to form inorganic mercury compounds or "salts". Mercury also forms organic mercury compounds of which methylmercury is the most common. Metallic mercury is used to produce chlorine gas and caustic soda, and is also used in thermometers, dental fillings and batteries. Mercury salts are sometimes used in skin lightening creams, antiseptic creams and ointments (ATSDR 2010).



Exposure to high levels of metallic, inorganic, or organic mercury can permanently damage the brain, kidneys, and developing foetus. The detrimental effects on normal brain function include irritability, shyness, tremors, changes in vision or hearing, and memory problems (ATSDR 2010). The US EPA does not classify metallic mercury as a human carcinogen, but classes methylmercury and mercuric chloride as possible human carcinogens (US EPA 2010).

### **3.3.3 Arsenic**

Arsenic is a naturally occurring element widely distributed in the Earth's crust. In the environment, arsenic is combined with oxygen, chlorine, and sulfur to form inorganic arsenic compounds. Arsenic also forms organic arsenic compounds. Inorganic arsenic compounds are mainly used to preserve wood, with copper chromium arsenic (CCA) used to make "pressure-treated" timber. Organic arsenic compounds are used as pesticides, primarily on cotton fields and orchards (ATSDR 2010).

Exposure to higher than average levels of arsenic occur mostly in the workplace, near hazardous waste sites, or in areas with high natural levels (e.g., geothermal areas). When exposed to high concentrations, inorganic arsenic can cause death. Exposure to lower levels for a long time (i.e., chronic exposure) can cause discoloration of the skin and the appearance of small corns or warts (ATSDR 2010). Inorganic arsenic is classified by the US EPA as a carcinogen (US EPA 2010).

### **3.3.4 Lead**

Lead is a naturally occurring bluish-gray metal found in small amounts in the Earth's crust. Lead can be found in all parts of our environment. Much of it comes from human activities including burning fossil fuels (particularly petrol containing tetraethyl lead additives), mining and manufacturing. Lead has many different uses. It is used in the production of batteries, ammunition, metal products (solder and pipes), and devices to shield X-rays. Because of health concerns, lead from paints and ceramic products, caulking, and pipe solder has been dramatically reduced in recent years. New Zealand has used lead free petrol since 1996 (Ministry of Economic Development).

Exposure to lead can be via breathing workplace air or dust, eating contaminated foods, or drinking contaminated water. Children can be exposed from eating lead-based paint chips or playing in contaminated soil. Lead can damage the nervous system, kidneys, and reproductive system (ATSDR 2010). The US EPA has classified lead as a probable human carcinogen (US EPA 2010).

## 4. Risk Assessment

### 4.1 Arowhenua contaminant data

For the purposes of the risk assessments, wet weight corrections were made on all dry weight contaminant data. Median and 95th percentile values were calculated for each contaminant for each species of fish and for watercress across all sites (Table 1). The median value was chosen over an arithmetic mean to remove the large influence of contaminant outliers in a relatively small sample size and is used to determine what likely contaminant loads would be expected from harvesting randomly across all sites. The 95<sup>th</sup> percentile data is a worse case scenario in which harvesting was only the most contaminated kai, that might be expected from harvesting at only the most contaminated sites.

### 4.2 Arowhenua consumption data

Local average consumption rates of harvested kai were calculated as 6.1, 4.0, 4.7 and 6.0 g/day for eels, trout, flounder and watercress, respectively (Stewart et al., 2010). Meal sizes were calculated at 213 g/meal for all fish species and 175 g/meal for watercress.

**Table 1:** Median and 95<sup>th</sup> percentile contaminant data ( $\mu\text{g}/\text{kg}$ ; wet weight) for kai from Arowhenua and input data assumptions used in risk assessment calculations.

Species	Compound	Contaminant Concentration ( $\mu\text{g}/\text{kg}$ wet weight)		Risk Values <sup>a</sup>		
		Median	95th percentile	CSF ( $\text{mg}/\text{kg}\text{-day}$ ) <sup>-1</sup>	BW (kg)	RfD ( $\text{mg}/\text{kg}\text{-day}$ )
Eel	p,p-DDT	2.39	21.71	0.34	80	5.0E-04
	p,p-DDD	1.56	35.99	0.24	80	NA
	p,p-DDE	29.5	261.0	0.34	80	NA
	Dieldrin	0.43	10.71	16.00	80	5.0E-05
	$\Sigma$ Chlordanes	0.20	6.48	0.35	80	5.0E-04
	HCB	0.09	0.35	1.60	80	8.0E-04
	$\Sigma$ PCBs	2.89	48.43	2.00	80	2.0E-05
	Cadmium	1.22	10.57	NA	80	1.0E-03
	Mercury	343.8	549.1	NA	80	1.0E-04
	Arsenic <sup>b</sup>	0.00	0.00	1.50	80	3.0E-04
	Zinc	10013	13578	NA	80	3.0E-01
	Nickel	0.00	0.00	NA	80	2.0E-02
Chromium	0.00	47.03	NA	80	3.0E-03	
Trout	p,p-DDT	0.19	0.35	0.34	80	5.0E-04
	p,p-DDD	0.07	0.33	0.24	80	NA
	p,p-DDE	16.53	18.34	0.34	80	NA
	Dieldrin	0.16	0.55	16.00	80	5.0E-05
	$\Sigma$ Chlordanes	0.02	0.06	0.35	80	5.0E-04
	HCB	0.02	0.02	1.60	80	8.0E-04
	$\Sigma$ PCBs	ND	ND	2.00	80	2.0E-05
	Cadmium	0.00	0.45	NA	80	1.0E-03
	Mercury	113.7	427.1	NA	80	1.0E-04
	Arsenic <sup>b</sup>	265.7	811.1	1.50	80	3.0E-04
	Zinc	4228	4783	NA	80	3.0E-01
	Nickel	0.0	29.3	NA	80	2.0E-02
Chromium	0.00	0.00	NA	80	3.0E-03	

Species	Compound	Contaminant Concentration (µg/kg wet weight)		Risk Values <sup>a</sup>		
		Median	95th percentile	CSF (mg/kg-day) <sup>-1</sup>	BW (kg)	RfD (mg/kg-day)
Flounder	p,p-DDT	1.08	4.13	0.34	80	5.0E-04
	p,p-DDD	0.84	2.42	0.24	80	NA
	p,p-DDE	9.74	25.27	0.34	80	NA
	Dieldrin	0.32	0.57	16.00	80	5.0E-05
	ΣChlordanes	0.20	0.39	0.35	80	5.0E-04
	HCB	0.02	0.03	1.60	80	8.0E-04
	ΣPCBs	ND	ND	2.00	80	2.0E-05
	Cadmium	0.00	0.00	NA	80	1.0E-03
	Mercury	42.5	89.3	NA	80	1.0E-04
	Arsenic <sup>b</sup>	120.2	495.9	1.50	80	3.0E-04
	Zinc	6799	7248	NA	80	3.0E-01
	Nickel	0.00	71.93	NA	80	2.0E-02
Chromium	0.00	0.00	NA	80	3.0E-03	
Watercress	Cadmium	8.1	17.7	NA	80	1.0E-03
	Mercury	0.0	0.7	NA	80	1.0E-04
	Arsenic	12.4	31.8	1.5	80	3.0E-04
	Zinc	2657	4815	NA	80	3.0E-01
	Nickel	76.6	189.8	NA	80	2.0E-02
	Chromium	43.0	54.0	NA	80	3.0E-03

<sup>a</sup> CSF = cancer slope factor; BW = body weight, RfD = reference dose, NA = not applicable, ND = not determined.

<sup>b</sup> Arsenic risk calculation subsequently reduced by a factor of 10 for risk assessment of fish to reflect an estimated inorganic fraction of total arsenic of 10% and provide a protective estimate of health risk (US EPA 2003).

### 4.3 Arowhenua catchment risk assessments

Median and 95<sup>th</sup> percentile contamination data (Table 1) were used to create risk assessments for lifetime cancer risk and chronic non-cancer risk. Monthly allowable fish consumption limits for each kai species sampled in the Arowhenua catchment (Figure 1) were calculated using equations 2.3.2, 2.3.3 and 2.3.4 in the methods section.

Median contamination risk data are shown in Table 2. These data approximate the risk associated with harvesting at all sites in the area randomly. As presented in Table 2, consumption limits are generally low for all the species. Lifetime cancer risk is the dominant risk factor in Table 2, accounting for lowest consumption limits of 2.3, 5.1 and 7.4 meals/month for trout, flounder and watercress, respectively (Table 2). For eel, the greatest risk is from a chronic non-cancer endpoint, with a consumption limit of 3.3 meals/month (Table 2).

**Table 2:** Risk assessments for the **median** contamination profile for each kai species from Arowhenua rohe.

Species	Risk Based Consumption Limit <sup>a</sup> (meals/month)	
	Cancer Health Endpoint <sup>b</sup>	Non-cancer Health Endpoint <sup>c</sup>
Eel	4.7	3.3
Trout	2.3	9.9
Flounder	5.1	32.4
Watercress	7.4	333.0

<sup>a</sup> The assumed meal size is 213 g for fish and 175 g for watercress.

<sup>b</sup> Refers to lifetime cancer risk (based on 1 in 100,000 risk level).

<sup>c</sup> Chronic systemic effects.

The risk assessment using the 95<sup>th</sup> percentile contamination data is a worse case scenario that approximates the risk associated with harvesting the most contaminated kai only. Risk assessment data for this “worse case scenario” are presented in Table 3. For all 4 species, the dominant factor to risk is a cancer health endpoint (i.e., lifetime cancer risk), with consumption limits of 0.3, 0.8, 1.5 and 2.9 meals/month for eel, trout, flounder and watercress, respectively (Table 3).

**Table 3:** Risk assessments for the **95<sup>th</sup> percentile** contamination profile for each kai species from Arowhenua catchment.

Species	Risk Based Consumption Limit <sup>a</sup> (meals/month)	
	Cancer Health Endpoint <sup>b</sup>	Non-cancer Health Endpoint <sup>c</sup>
Eel	0.3	2.1
Trout	0.8	2.6
Flounder	1.5	15.4
Watercress	2.9	129.9

<sup>a</sup> The assumed meal size is 213 g for fish and 175 g for watercress.

<sup>b</sup> Refers to lifetime cancer risk (based on 1 in 100,000 risk level).

<sup>c</sup> Chronic systemic effects.

To ascertain which sites and which species are affording the highest risk, site by site risk assessments were undertaken.

#### **4.4 Individual site risk assessments**

The eleven sites sampled were divided into geographical regions, around Temuka, Washdyke, Winchester and the reference site Te Nga Wai. Risk assessments for lifetime cancer and non-cancer risks were carried out as described earlier.

The consumption limits for each species at each site are summarised in Table 4.

**Table 4:** Summary of individual consumption limits for each species at each site.

Site	Site # <sup>d</sup>	Species	Risk Based Consumption Limit <sup>a</sup> (meals/month)	
			Cancer Health Endpoint <sup>b</sup>	Cancer Health Endpoint <sup>c</sup>
Temuka	1	Eel	5.3	3.0
		Trout	1.4	2.3
		Watercress	4.9	220
Opihi River upstream	2	Eel	6.7	2.1
		Watercress	6.6	298
Opihi River below Pleasant Point	3	Eel	20	2.5
		Watercress	2.7	123
Opihi Rivermouth	4	Trout	0.8	10
		Flounder	6.4	55
		Watercress	3.2	145
Ohapi Creek	5	Eel	0.7	4.9
		Watercress	8.4	377
Orari Ohapi	6	Eel	8.3	5.3
		Trout	2.6	22.3
		Flounder	9.2	35.3
Doncaster	7	Eel	0.2	3.4
		Flounder	3.0	12
Washdyke Lagoon	8	Eel	6.6	48
		Flounder	1.1	21
Waihi River	9	Eel	3.0	3.5
		Trout	15	8.4
		Watercress	9.7	435
Winchester	10	Eel	0.7	2.0
		Trout	4.2	14
		Watercress	ND <sup>d</sup>	1798
Te Nga Wai	11	Eel	11	3.1
		Watercress	ND <sup>d</sup>	1323

<sup>a</sup> The assumed meal size is 213 g for fish and 175 g for watercress.

<sup>b</sup> Refers to lifetime cancer risk (based on 1 in 100,000 risk level).

<sup>c</sup> Chronic systemic effects.

<sup>d</sup> No contaminants detected in watercress.



#### 4.4.1 Temuka region

Six sites were sampled from around Temuka, stretching as far east as Orari Ohapi. This region is predominantly rural and includes the Opihi, Temuka and Orari Rivers as well as Ohapi Creek, which feeds into the Orari River. The sites sampled were Temuka, Opihi River upstream, Opihi River below Pleasant Point, Opihi River mouth, Ohapi Creek and Orari Ohapi (Figure 2).

**Figure 2:** Sites sampled in the Temuka region of rohe of Arowhenua<sup>a</sup>



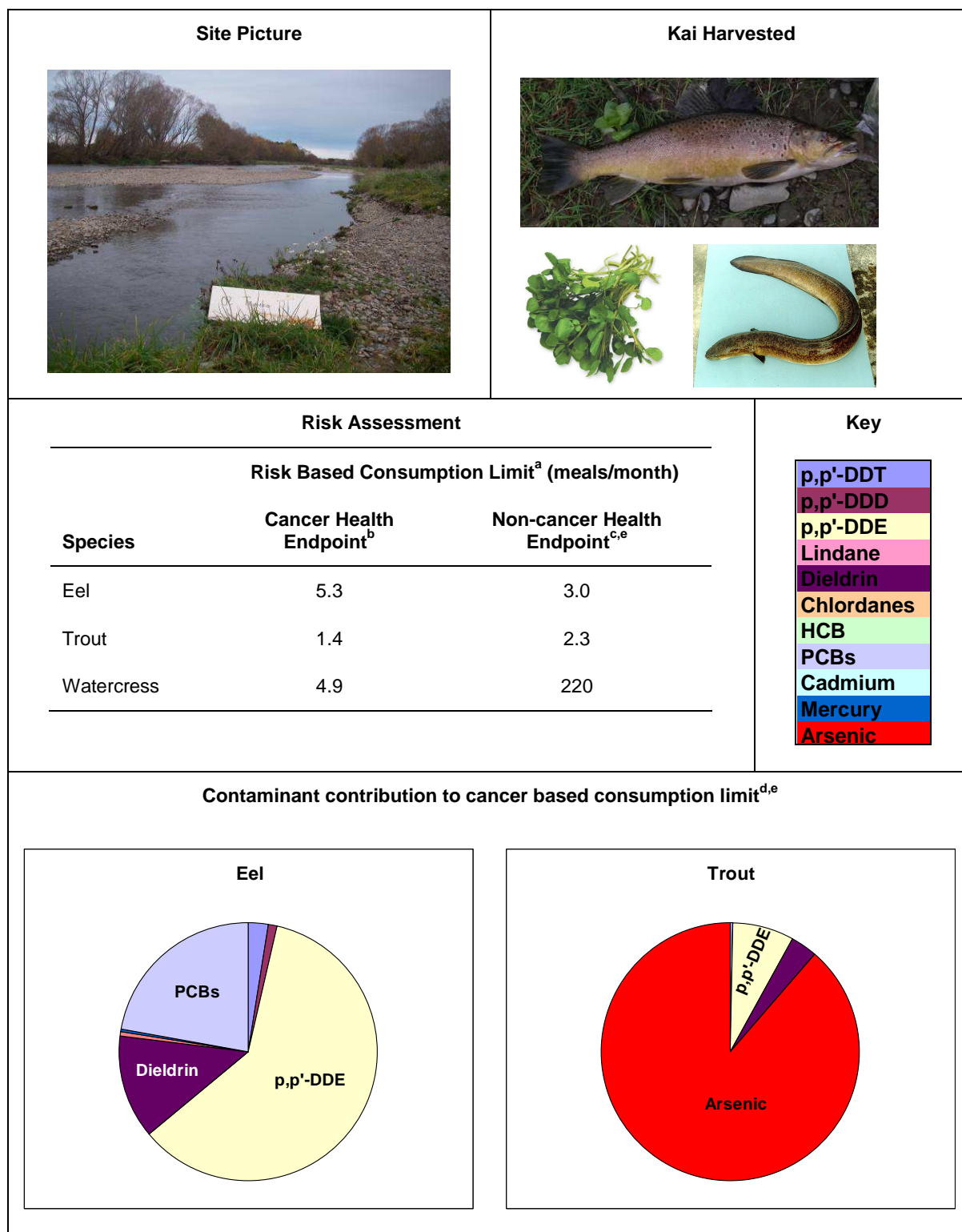


<sup>a</sup> 1 = Temuka River; 2 = Opihi River upstream; 3 = Opihi River below Pleasant Point; 4 = Opihi River mouth; 5 = Ohapi Creek; 6 = Orari Ohapi.

### Temuka River

Eel, trout and watercress were collected from the Temuka River (Figure 2, site 1). All relevant data for the Temuka River site are summarised in Figure 3. Trout was the greatest concern from this site with consumption limits of 1.4 and 2.3 meals/month for cancer and non-cancer risks respectively. The main contaminants contributing to these consumption limits were arsenic and mercury respectively (Appendix 1a). No arsenic was detected in eel tissue, however, mercury afforded the lowest non-cancer consumption rate of 3.0 meals/month (Figure 3). Arsenic was the sole cancer risk determinand for watercress, affording a consumption limit of 4.9 meals/month.

**Figure 3:** Temuka River summary of site, biota sampled and risk assessment information.



<sup>a</sup> The assumed meal size is 213 g for fish and 175 g for watercress.

<sup>b</sup> Refers to lifetime cancer risk (based on 1 in 100,000 risk level).

<sup>c</sup> Chronic systemic effects.

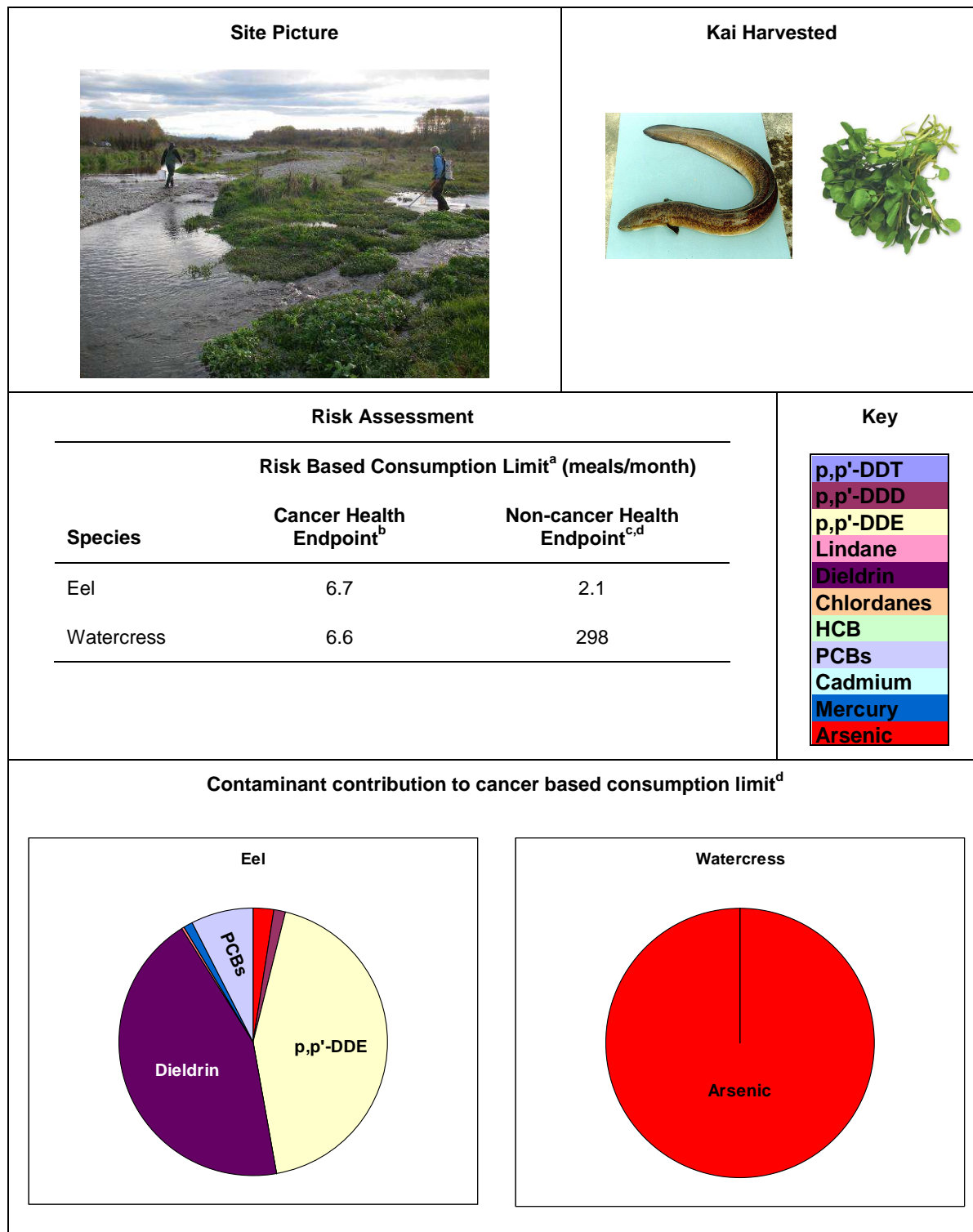
<sup>d</sup> The only contaminant detected with a contribution to cancer risk from watercress was arsenic.

<sup>e</sup> For non-cancer based consumption limits mercury was the limiting contaminant with 96% and 97% contribution for eel and trout respectively.

### **Opihi River upstream**

Eel and watercress were collected from upstream in the Opihi River (Figure 2, site 2). All relevant data for Opihi River upstream are summarised in Figure 4. The most concerning risk was a chronic non-cancer risk from mercury contamination (Appendix 1b) of eel, leading to a consumption limit of 2.1 meals/month. Arsenic was the sole cancer risk determinand for watercress, affording a consumption limit of 6.6 meals/month.

**Figure 4:** Opihi River ‘upstream’ summary of site, biota sampled and risk assessment information.



<sup>a</sup> The assumed meal size is 213 g for fish and 175 g for watercress.

<sup>b</sup> Refers to lifetime cancer risk (based on 1 in 100,000 risk level).

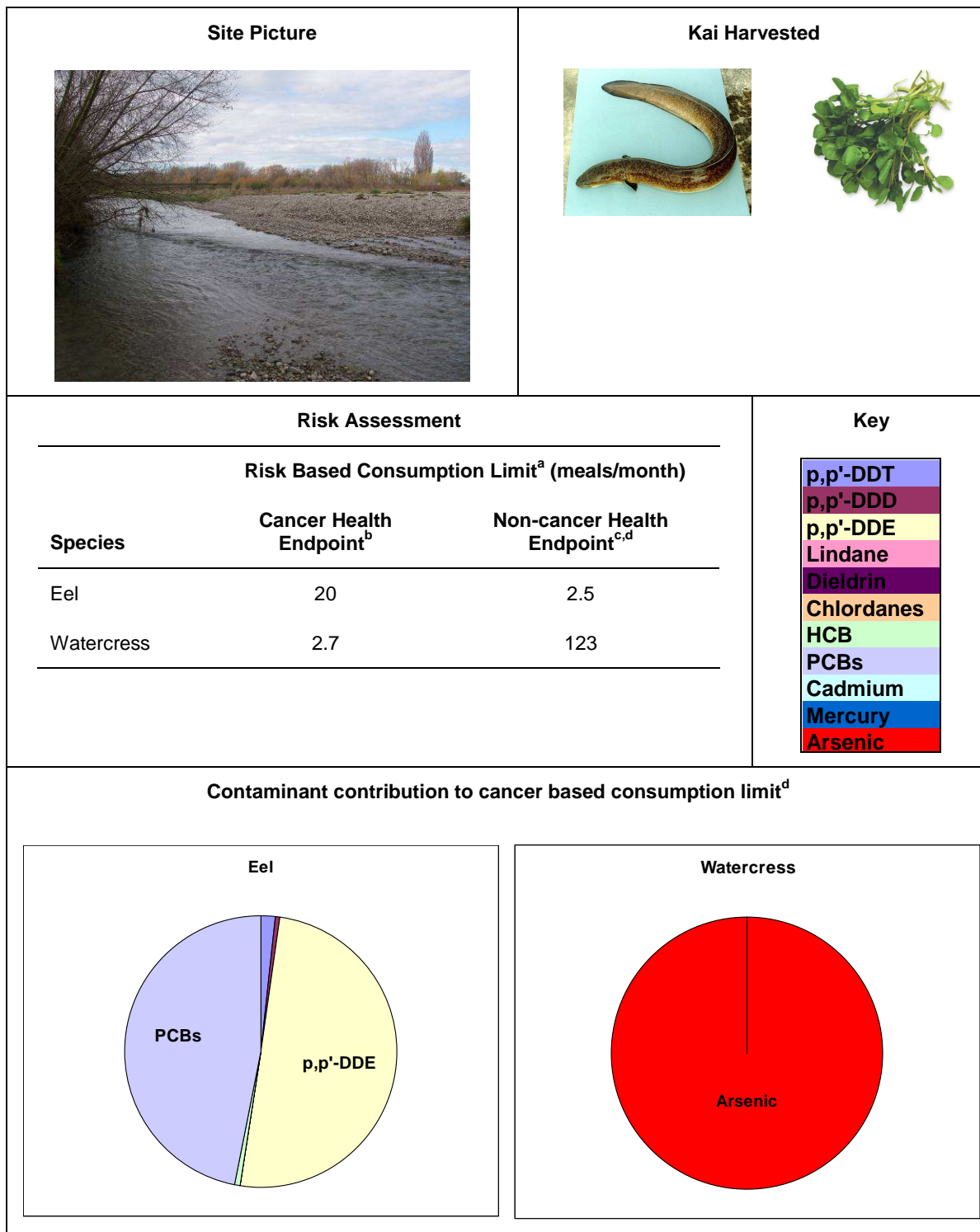
<sup>c</sup> Chronic systemic effects.

<sup>d</sup> For non-cancer based consumption limits mercury was the limiting contaminant with 98% contribution for eel.

### **Opihi River below Pleasant Point**

Eel and watercress were collected below Pleasant Point on the Opihi River (Figure 2, site 3). All relevant data for this are summarised in Figure 5. As for Opihi River upstream, the most concerning risk was a chronic non-cancer risk from mercury contamination of eel (Appendix 1c), leading to a consumption limit of 2.5 meals/month (Figure 5). Arsenic was the sole cancer risk determinand for watercress, affording a consumption limit of 2.7 meals/month, somewhat lower than that calculated for Opihi River upstream of 6.6 meals/month (refer to Figure 4).

**Figure 5:** Opihi River ‘below Pleasant Point’ summary of site, biota sampled and risk assessment information.



<sup>a</sup> The assumed meal size is 213 g for fish and 175 g for watercress.

<sup>b</sup> Refers to lifetime cancer risk (based on 1 in 100,000 risk level).

<sup>c</sup> Chronic systemic effects.

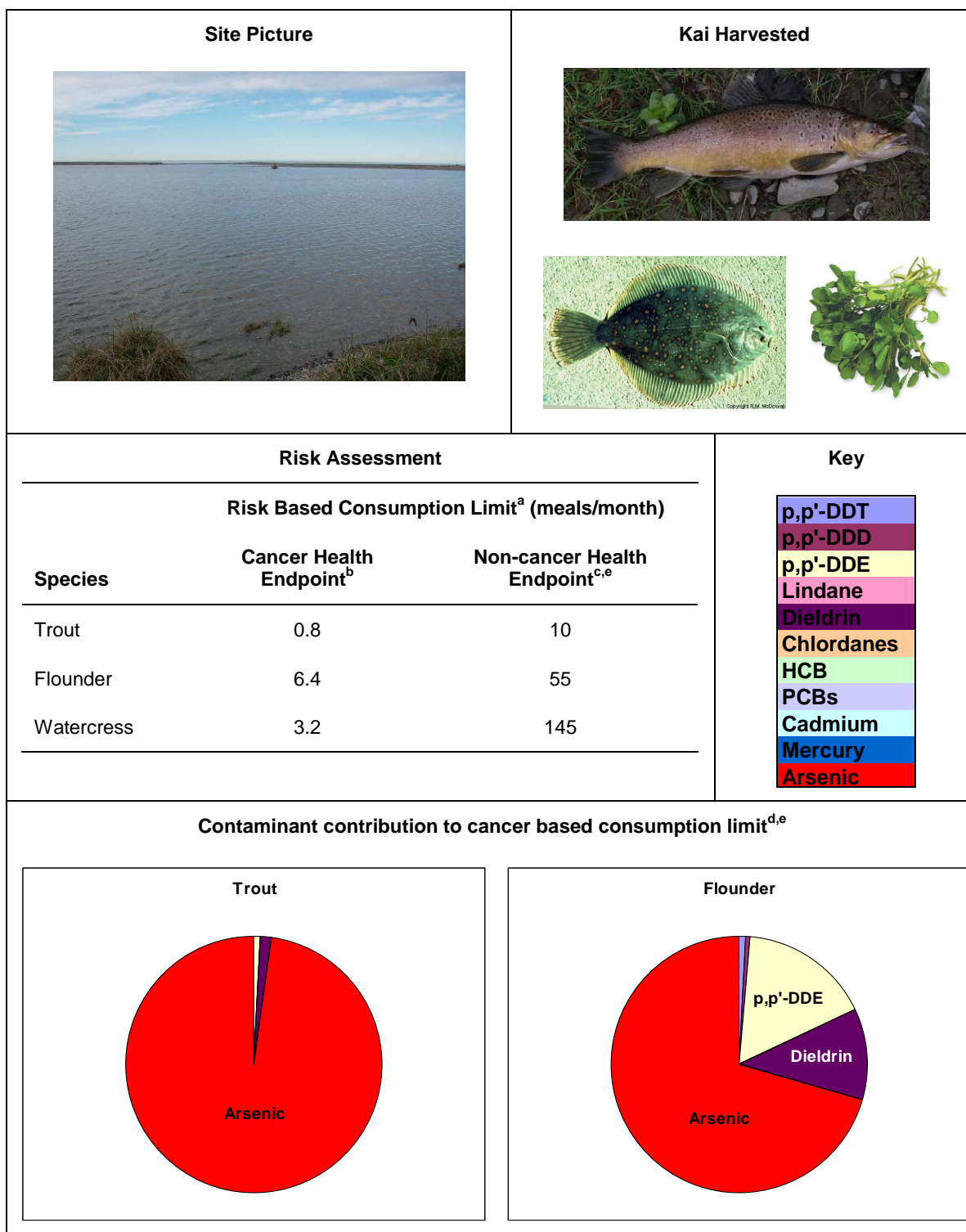
<sup>d</sup> For non-cancer based consumption limits mercury was the limiting contaminant with 98% contribution for eel.

## **Opihi River Mouth**

Trout, flounder and watercress were obtained from Opihi River mouth (Figure 2, site 4). All relevant data for this site are summarised in Figure 6. Trout was the highest risk concern from this area, with arsenic being the main contributing contaminant for the lifetime cancer risk (Appendix 1d). Consumption limits for trout were 0.8 meals/month (Figure 6). Arsenic was the sole contributor to lifetime cancer risk, with a consumption limit of 3.2 meals/month (Figure 6), similar to Opihi River below Pleasant Point (refer to Figure 5). Flounder provided the lowest risk from Opihi River mouth, with a lifetime cancer consumption limit of 6.4 meals/month, with major contributing contaminants being arsenic, p,p'-DDE and dieldrin.



**Figure 6:** Opihi River ‘mouth’ summary of site, biota sampled and risk assessment information.



<sup>a</sup> The assumed meal size is 213 g for fish and 175 g for watercress.

<sup>b</sup> Refers to lifetime cancer risk (based on 1 in 100,000 risk level).

<sup>c</sup> Chronic systemic effects.

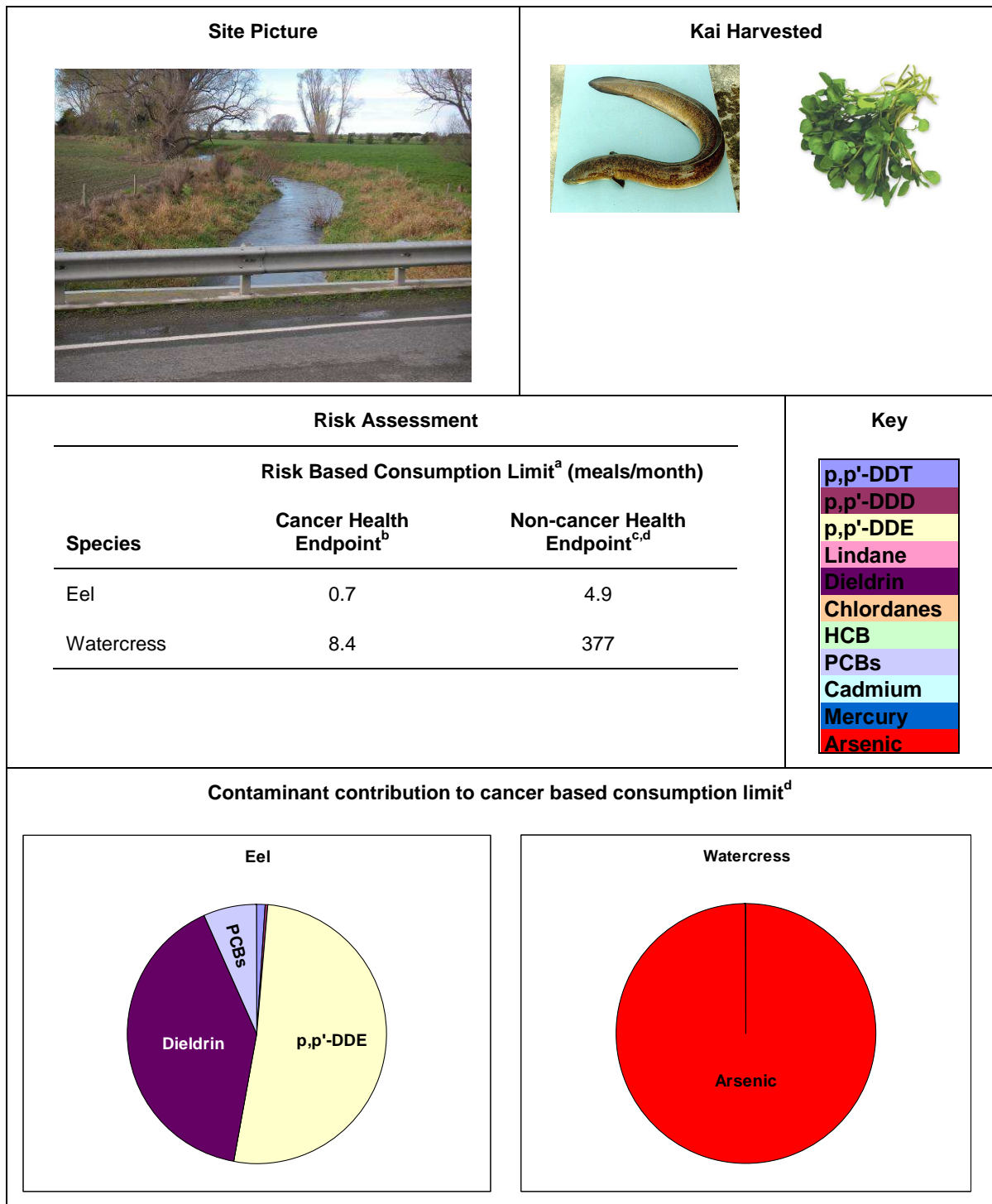
<sup>d</sup> The only contaminant detected with a contribution to carcinogenic risk from watercress was arsenic.

<sup>e</sup> For non-cancer based consumption limits mercury was the limiting contaminant with 78% and 80% contribution for trout and flounder, respectively. Arsenic was the second major contributor to non-cancer risk with 21% and 11% for trout and flounder, respectively.

## Ohapi Creek

Eel and watercress were collected from Ohapi Creek (Figure 2, site 5). All relevant data for the Ohapi Creek site are summarised in Figure 7. Eel was of significant concern, especially for lifetime cancer risk, with a calculated consumption limit of 0.7 meals/month. Contaminants contributing most to this risk were p,p'-DDE, dieldrin and arsenic with individual consumption limits of 10.3 and 13.1 g/day respectively (Appendix 1e). Watercress was much less of a concern than eel from this site, with a consumption limit of 8.4 meals/month for lifetime cancer risk.

**Figure 7:** Ohapi Creek summary of site, biota sampled and risk assessment information.



<sup>a</sup> The assumed meal size is 213 g for fish and 175 g for watercress.

<sup>b</sup> Refers to lifetime cancer risk (based on 1 in 100,000 risk level).

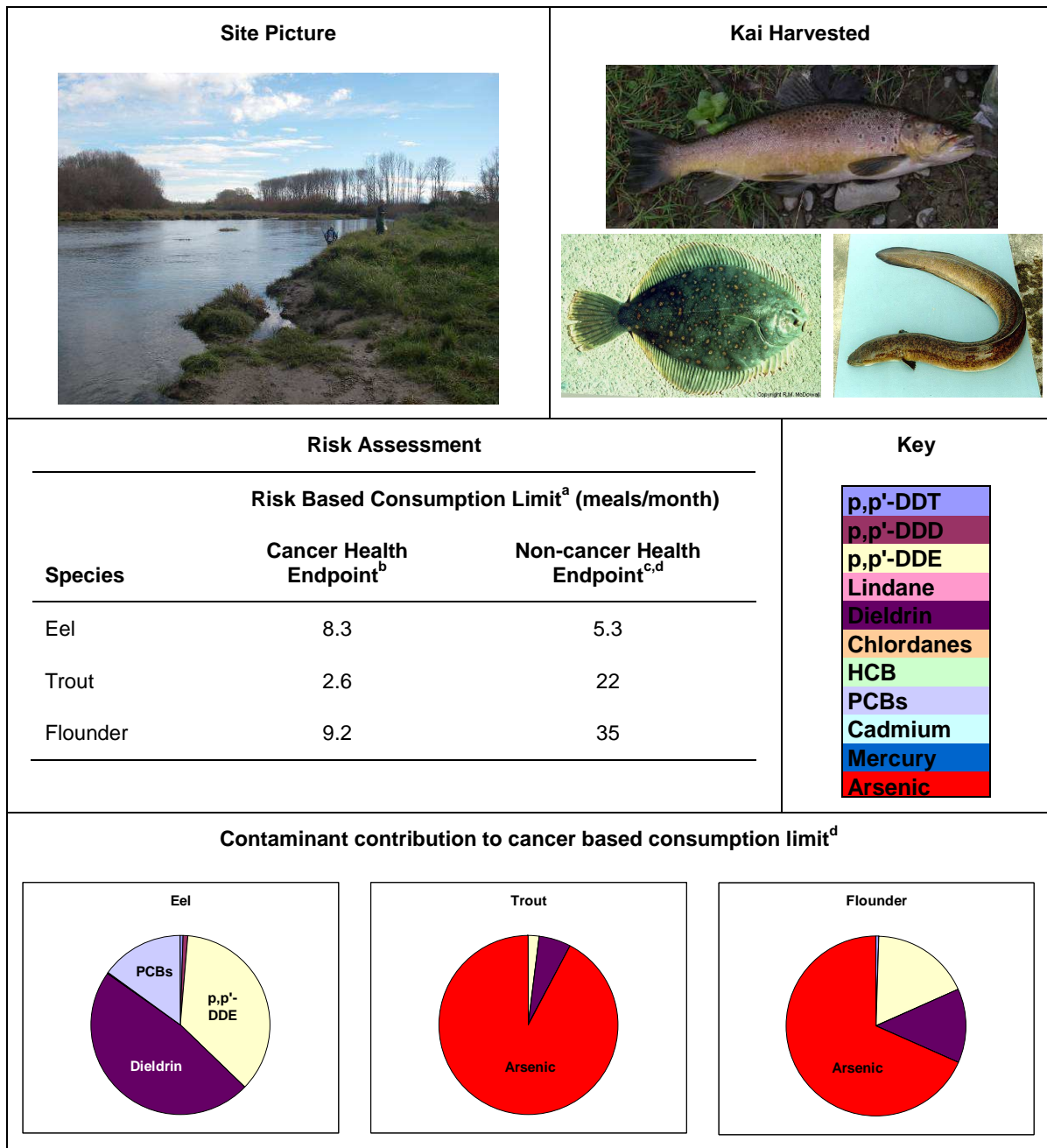
<sup>c</sup> Chronic systemic effects.

<sup>d</sup> For non-cancer based consumption limits mercury was the limiting contaminant with 87% for eel. PCBs were the second major contributor to non-cancer risk with 9%.

## Orari Ohapi

The Orari Ohapi collection site was situated at the mouth of the Ohapi River (Figure 2, site 6). All relevant data for the Orari Ohapi site are summarised in Figure 8. Eel, trout and flounder were harvested from this site. For eel the highest risk was a chronic non-cancer risk with the main contributing contaminants being mercury and PCBs. The consumption limit for eel was calculated at 5.3 meals/month. Trout had the lowest consumption limit of the three species studied, with a consumption limit of 2.3 meals/month based on lifetime excess cancer risk. The main contributing contaminant was arsenic, with minor contributions from dieldrin and p,p'-DDE. Flounder had a similar contamination profile from this site to trout but with a higher calculated consumption limit of 9.2 meals/month (carcinogenic endpoint).

**Figure 8:** Orari Ohapi summary of site, biota sampled and risk assessment information.



<sup>a</sup> The assumed meal size is 213 g for fish and 175 g for watercress.

<sup>b</sup> Refers to lifetime cancer risk (based on 1 in 100,000 risk level).

<sup>c</sup> Chronic systemic effects.

<sup>d</sup> For non-cancer based consumption limits mercury was the limiting contaminant with 96%, 82% and 96% contribution for eel, trout and flounder, respectively. In trout and flounder, arsenic was the second major contributor to non-cancer risk with 14% and 13%, respectively.

#### 4.4.2 Washdyke Region

Two sites were sampled from the Washdyke region. This area is just north of Timaru and is predominantly industrial (Figure 9).

**Figure 9:** Sites sampled from Washdyke region of the rohe of Arowhenua<sup>a</sup>

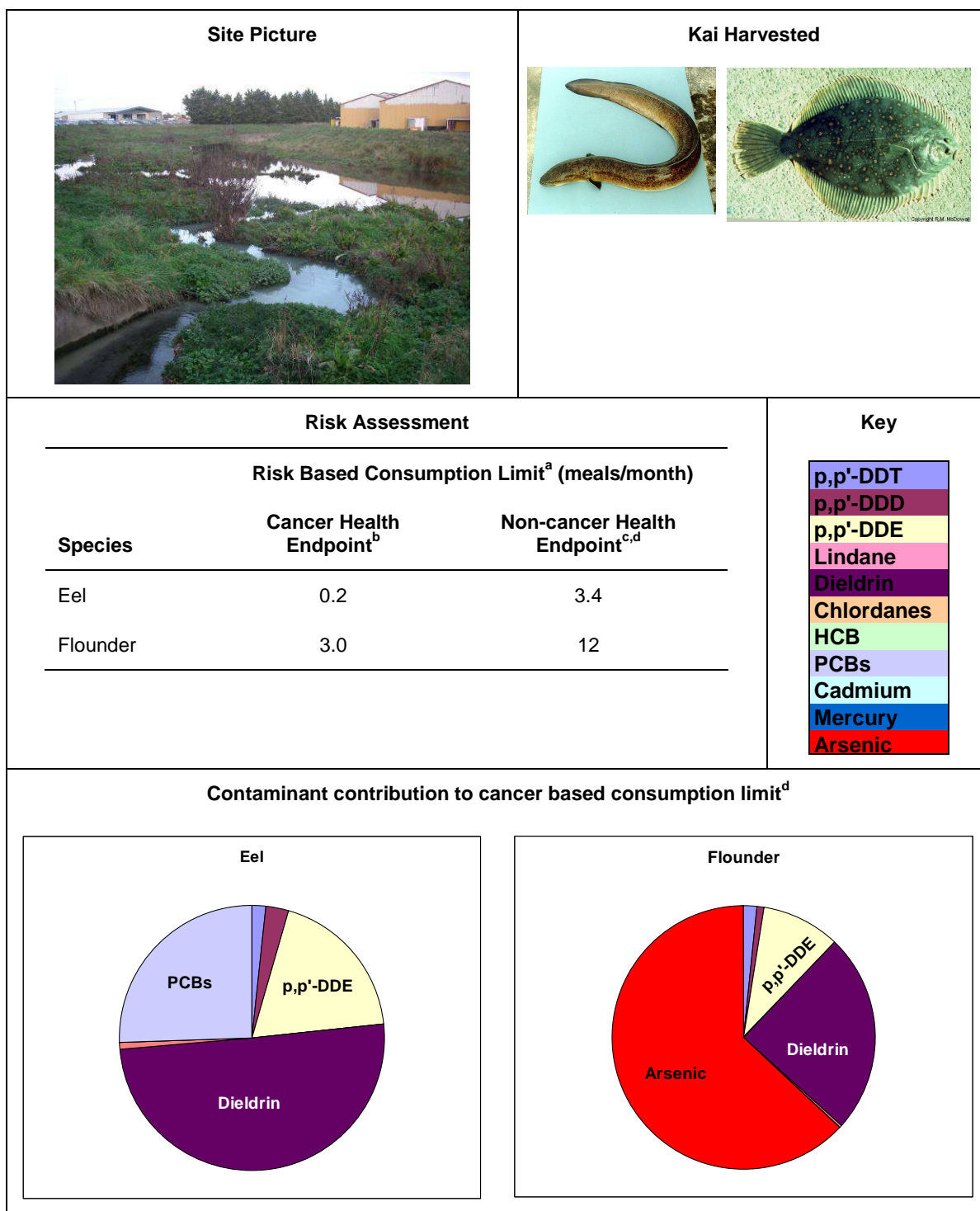


<sup>a</sup>7 = Doncaster; 8 = Washdyke Lagoon.

## **Doncaster**

The site of Doncaster was just off SH1 with industrial stormwater inputs as potentially major sources of contamination (Figure 9, site 7). All relevant data for the Doncaster site are summarised in Figure 10. Eel and flounder were collected from Doncaster. Eel from Doncaster had the highest risk to human health of any of the species studied, from any site, with a cancer risk consumption limit of 0.2 meals/month, or less than 3 meals/year. The major contribution to this risk was dieldrin, with significant contributions by PCBs and p,p'-DDE and minor contributions by p,p'-DDT and p,p'-DDD. Flounder had a similar contaminant profile to eel, where the risk for PCBs in eel was substituted with arsenic in flounder. The consumption limit for flounder from Doncaster was 3.0 meals/month.

**Figure 10:** Doncaster summary of site, biota sampled and risk assessment information.



<sup>a</sup> The assumed meal size is 213 g for fish and 175 g for watercress.

<sup>b</sup> Refers to lifetime cancer risk (based on 1 in 100,000 risk level).

<sup>c</sup> Chronic systemic effects.

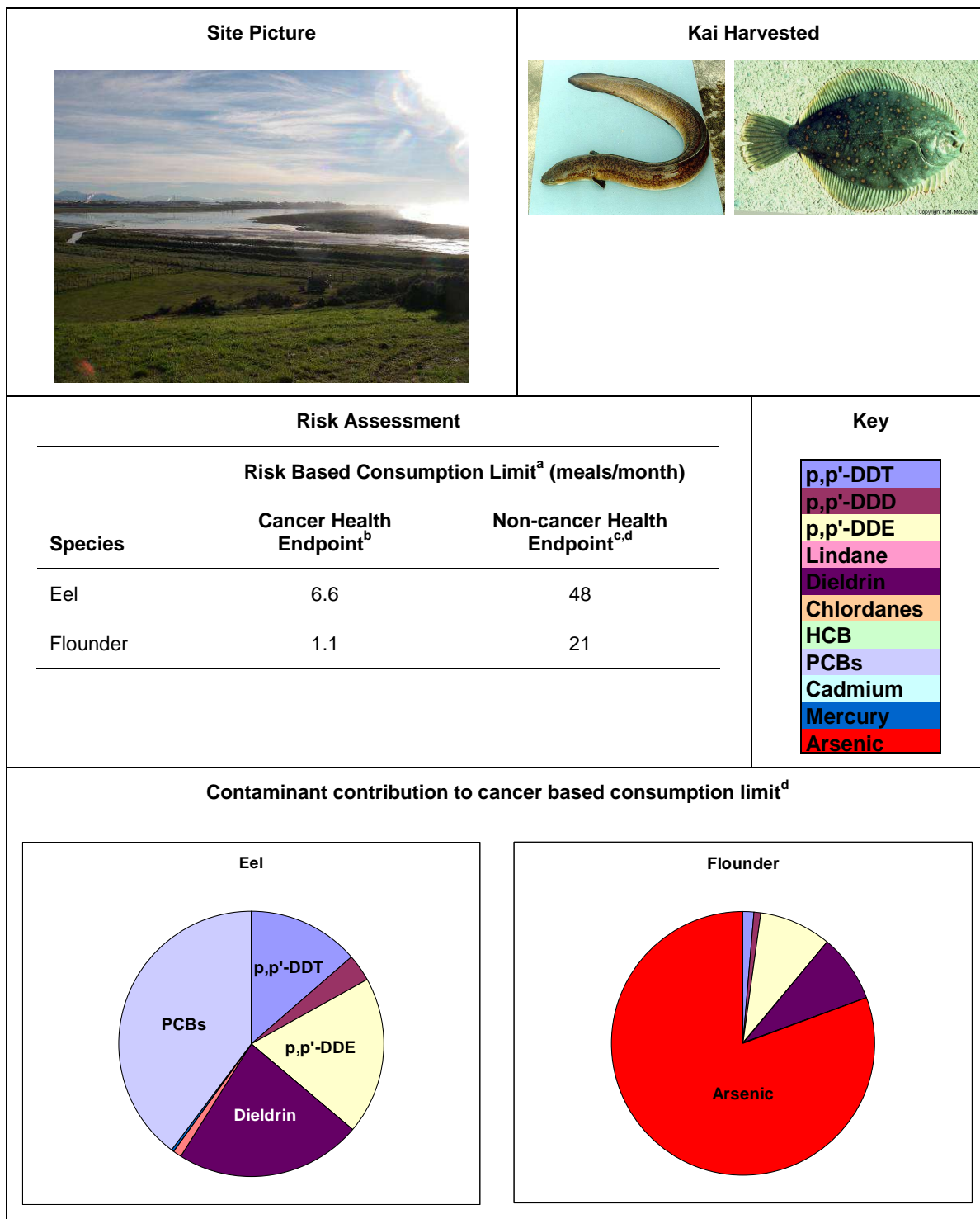
<sup>d</sup> For eel non-cancer based consumption limits ΣPCBs was the limiting contaminant with 63%, followed by mercury with 29%. For flounder the major non-cancer risk contribution was mercury, with 77%, with a contribution of 13% due to arsenic.



### **Washdyke Lagoon**

Washdyke Lagoon is further removed from the industrial area than Doncaster and doesn't have direct stormwater inputs into it (Figure 9, site 8). All relevant data for the Washdyke Lagoon site are summarised in Figure 11. Eel and flounder were collected from Washdyke Lagoon. Unexpectedly, flounder had a higher lifetime cancer risk than eel from this site with a consumption limit of 1.1 meals/month. Arsenic was the dominant cause of risk in flounder from this site, followed by smaller contributions from dieldrin and DDE. Eel had a consumption limit of 6.6 meals/month, based on a lifetime cancer risk. Unlike flounder, there was no dominant contaminant causing this risk, with PCBs, dieldrin, DDE and DDT all contributing significant amounts to the overall risk.

**Figure 11:** Washdyke Lagoon summary of site, biota sampled and risk assessment information.



<sup>a</sup> The assumed meal size is 213 g for fish and 175 g for watercress.

<sup>b</sup> Refers to lifetime cancer risk (based on 1 in 100,000 risk level).

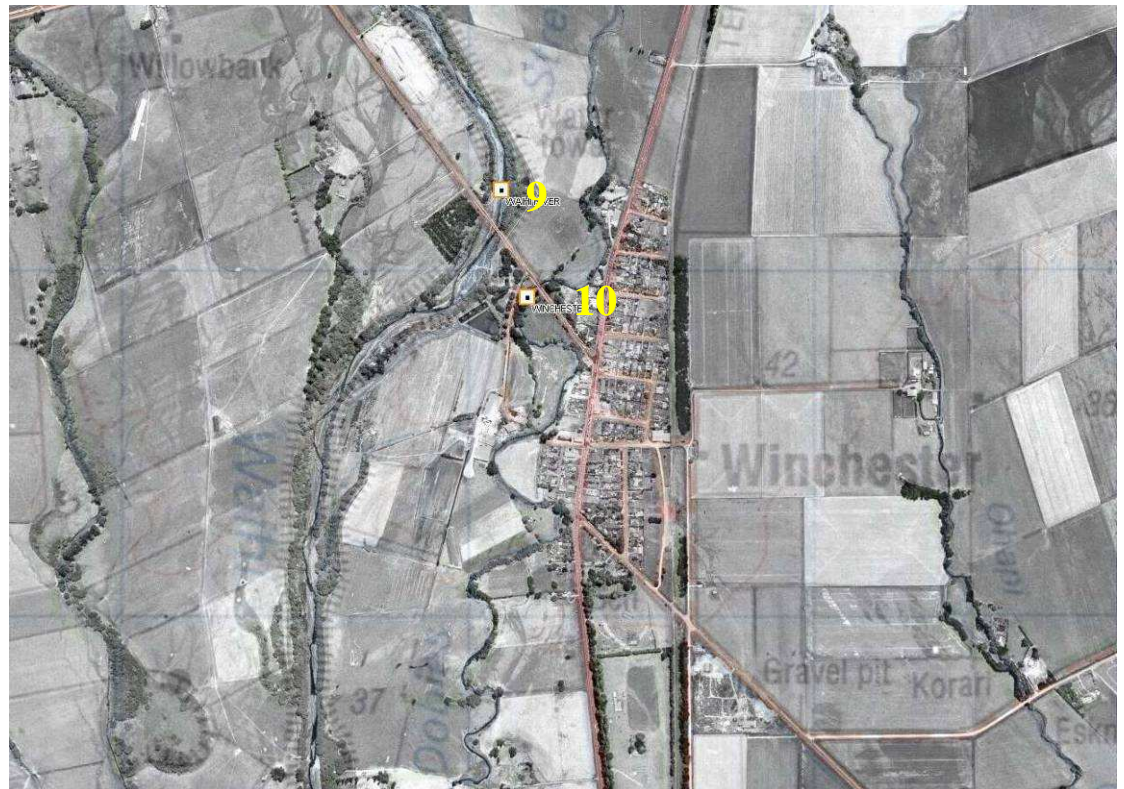
<sup>c</sup> Chronic systemic effects.

<sup>d</sup> For eel non-cancer based consumption limits mercury was the limiting contaminant with 50%, followed by ΣPCBs with 36%. For flounder the major contribution to non-cancer risk was mercury, with 70%, followed by arsenic with 24%.

#### 4.4.3 Winchester Region

Two sites were sampled around the town of Winchester, which is a small rural town situated on state highway 1. Both sites were in close proximity to each other, with Waihi River site on the Waihi River and Winchester site on Dobies Stream, a small tributary of the Waihi River (Figure 12). The Waihi River site is upstream of a historic wool scour - where detergents were used to clean or “scour” the wool - while the Winchester site is downstream.

**Figure 12:** Sites sampled in the Winchester region of the Arowhenua rohe<sup>a</sup>.

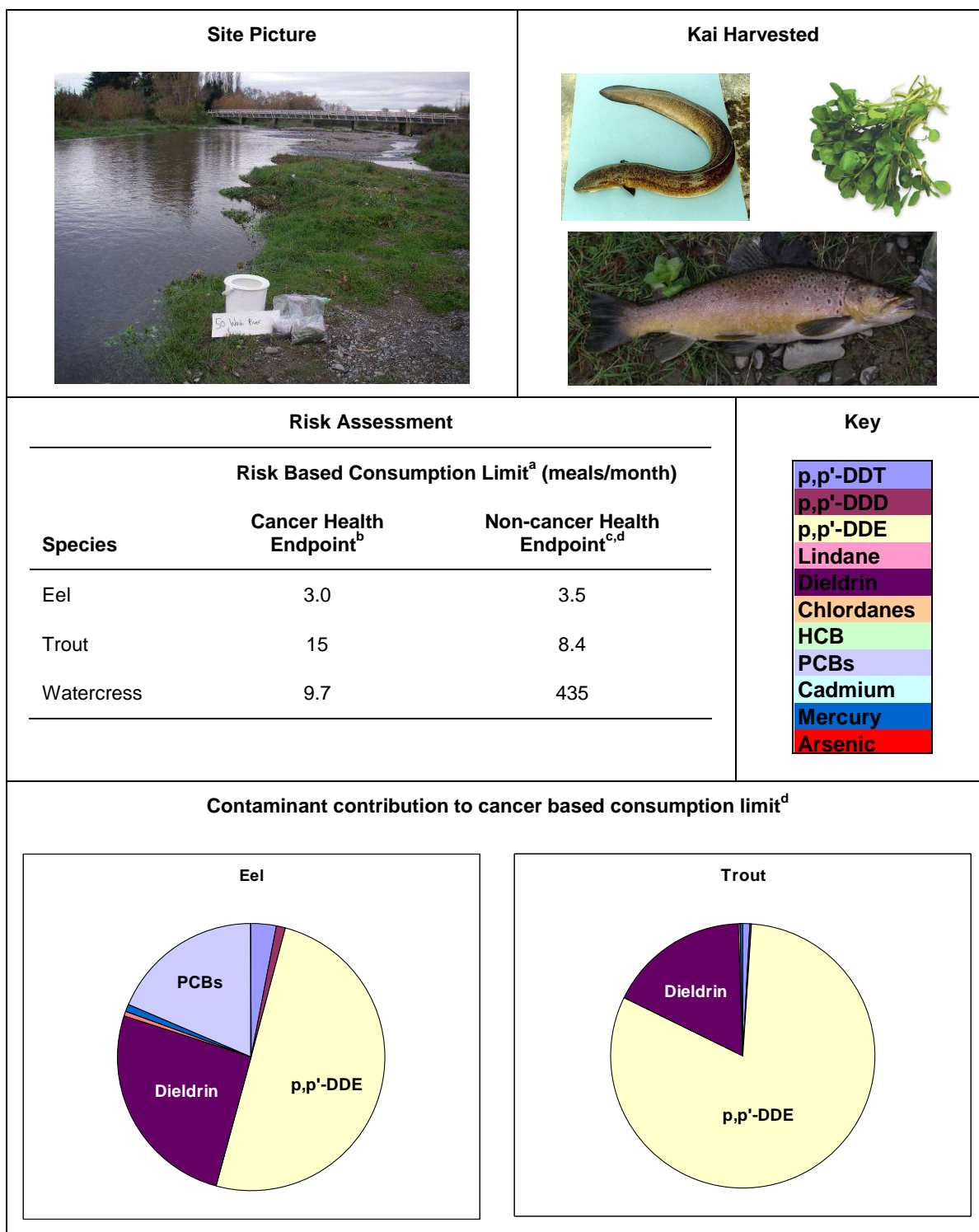


<sup>a</sup> 9 = Waihi River; 10 = Winchester.

## **Waihi River**

Eel, trout and watercress were harvested from the Waihi River site (Figure 12, site 9). All relevant data for the Waihi River site are summarised in Figure 13. For eel, the lifetime cancer risk and chronic non-cancer risk consumption limits were virtually equal, with 3.0 and 3.5 meals/month respectively. For the cancer risk, DDE, dieldrin and PCBs were the main contributors to this risk, while mercury was the most significant non-cancer risk (see appendix 1i). Trout had a consumption limit of 8.4 meals/month, based on chronic non-cancer risk, with mercury being the dominant contaminant component of this risk. Watercress had the highest consumption limit of the three kai of 9.7 meals/month, based on lifetime cancer risk.

**Figure 13:** Waihi River summary of site, biota sampled and risk assessment information.



<sup>a</sup> The assumed meal size is 213 g for fish and 175 g for watercress.

<sup>b</sup> Refers to lifetime cancer risk (based on 1 in 100,000 risk level).

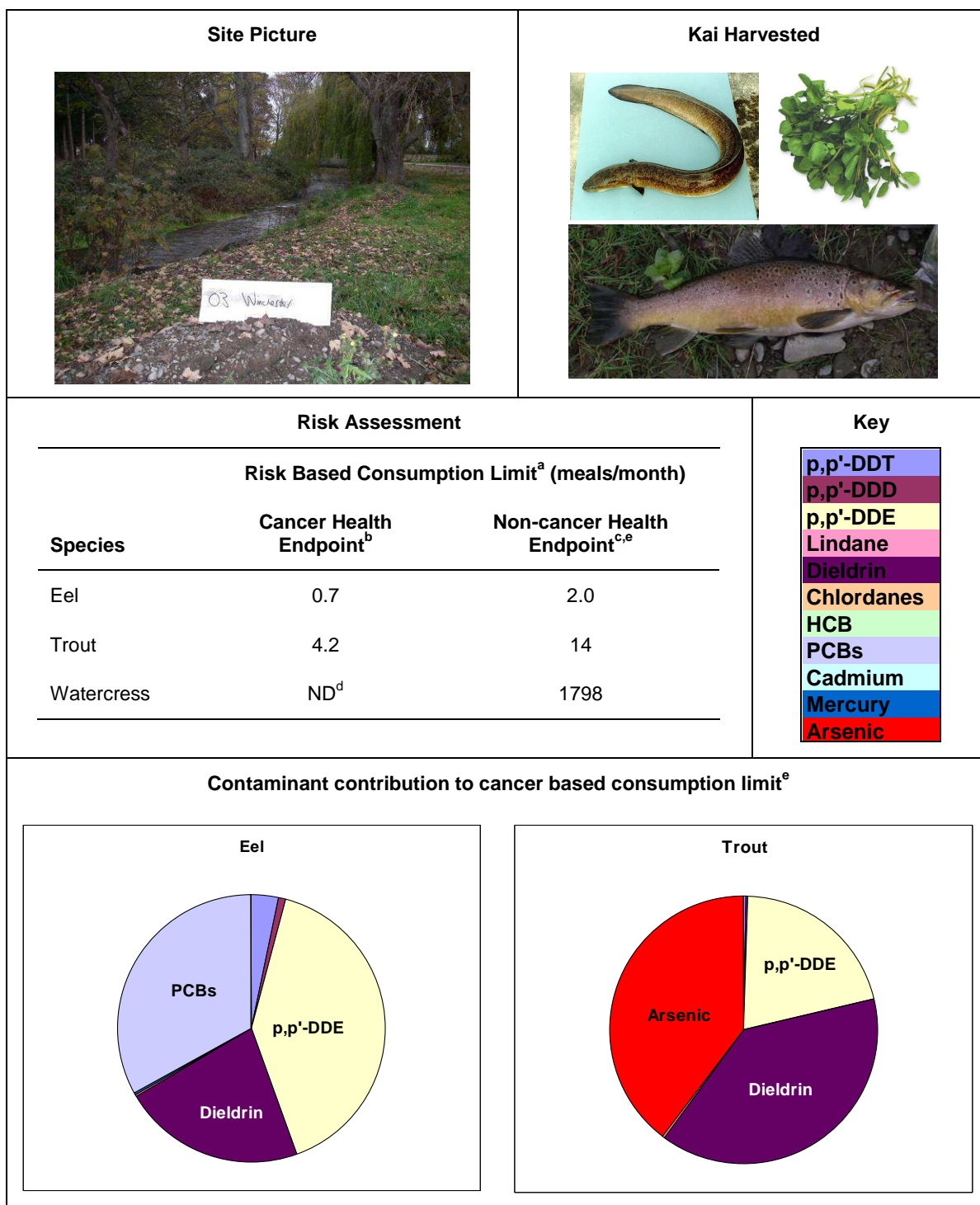
<sup>c</sup> Chronic systemic effects.

<sup>d</sup> For non-cancer based consumption limits mercury was the limiting contaminant with 94% and 99% contribution for eel and trout, respectively.

## **Winchester**

The Winchester site was on a tributary of the Waihi River, downstream of an historic wool scour (Figure 12 site 10). All relevant data for the Winchester site are summarised in Figure 14. The risk from consuming eel from this site was greater than for the Waihi River site, with consumption limits of 0.7 and 2.0 meals/month based on carcinogenic and non-carcinogenic endpoints, respectively. As for the Waihi River site, DDE, dieldrin and PCBs were the main contributors to lifetime cancer risk. This risk pattern was consistent for trout from Winchester, but with a much higher consumption limit of 4.2 meals/month. Watercress had virtually limitless consumption limits from the Winchester site.

**Figure 14:** Winchester summary of site, biota sampled and risk assessment information.



<sup>a</sup> The assumed meal size is 213 g for fish and 175 g for watercress.

<sup>b</sup> Refers to lifetime cancer risk (based on 1 in 100,000 risk level).

<sup>c</sup> Chronic systemic effects.

<sup>d</sup> No contaminants detected in watercress.

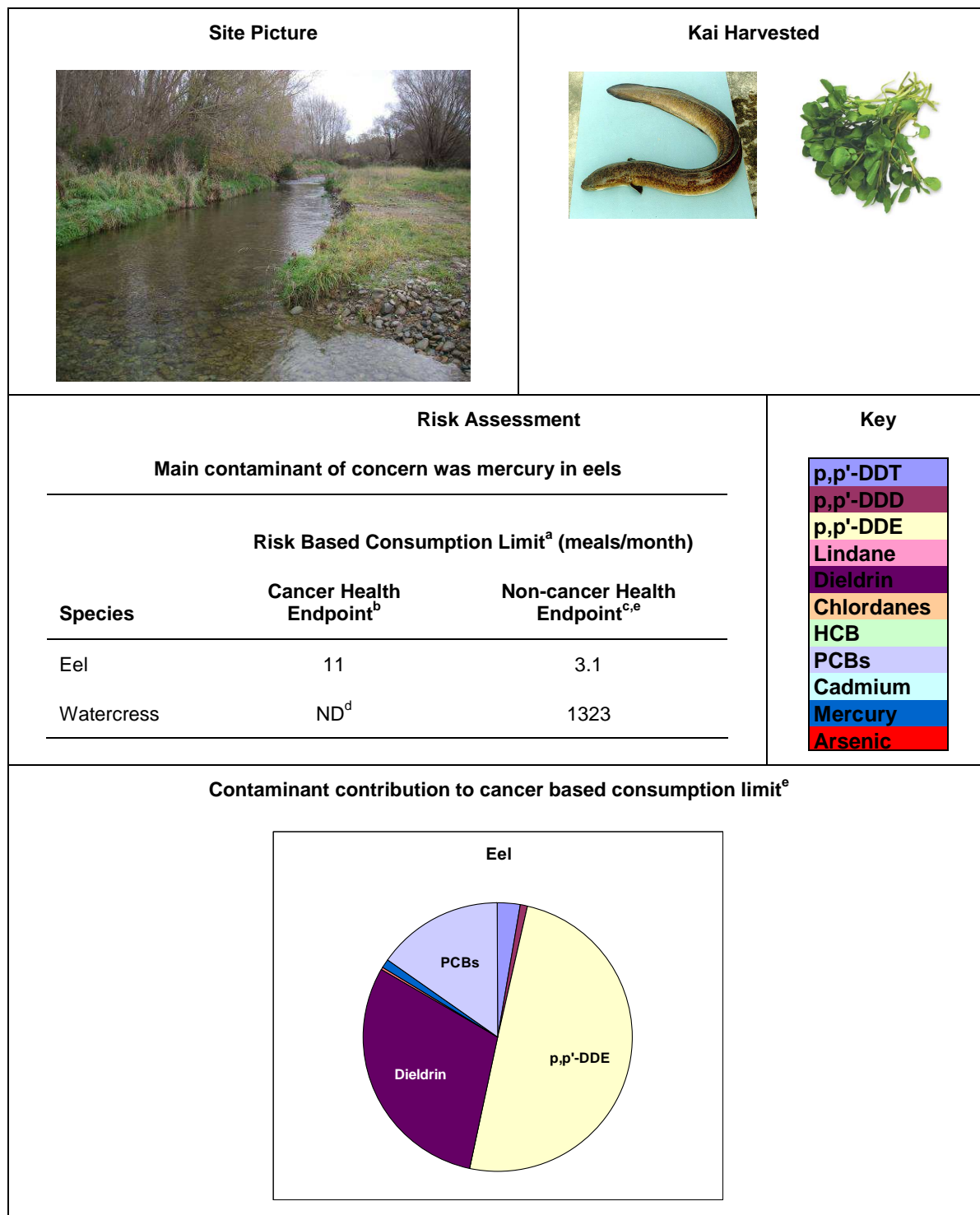
<sup>e</sup> For eel non-cancer based consumption limits mercury was the limiting contaminant with 79%, followed by ΣPCBs with 19%. For trout the major contribution to non-cancer risk was mercury, with 93%.

#### **4.4.4 Reference Site: Te Nga Wai**

A freshwater river/stream reference site was chosen as far as practical from human habitation. Eel and watercress were harvested from Te Nga Wai (refer to Figure 1 for site location within the Arowhenua rohe). All relevant data for the reference site are summarised in Figure 15. Eel had a consumption limit of 3.1 meals/month, based on a chronic non-cancer risk. The main contributor to this risk was mercury. Watercress harvested from Te Nga Wai posed virtually no health risk.



**Figure 15:** Te Nga Wai summary of site, biota sampled and risk assessment information.



<sup>a</sup> The assumed meal size is 213 g for fish and 175 g for watercress.

<sup>b</sup> Refers to lifetime cancer risk (based on 1 in 100,000 risk level).

<sup>c</sup> Chronic systemic effects.

<sup>d</sup> No contaminants detected in watercress.

<sup>e</sup> For non-cancer based consumption limits mercury was the limiting contaminant with 98% for eel.

#### 4.5 Mercury contamination as a component of risk

As stated in section 4.3 of this report, the most significant risk to consumers in the rohe of Arowhenua from consumption of wild caught kai was an excess risk of developing cancer during a lifetime. The non-cancer risk only exceeds the cancer risk when eels are consumed with median contamination concentrations.

Non-cancer risk in this study is dominated by mercury contamination (refer to sections 4.4.1 to 4.4.4). The percentage contribution of mercury to non-cancer risk is presented in Table 5.

**Table 5:** Comparison of percentage mercury contribution to non-cancer risk with the dominant risk for fish species at all sites in the rohe of Arowhenua.

Site	Eel %Hg <sup>a</sup>	Risk <sup>b</sup>	Trout %Hg <sup>a</sup>	Risk <sup>b</sup>	Flounder %Hg <sup>a</sup>	Risk <sup>b</sup>
Waihi	94	C	99	NC		
Winchester	79	C	93	C		
Temuka	96	NC	97	C		
Te Nga Wai	98	NC				
Opihi below PP	98	NC				
Opihi river mouth			78	C	80	C
Ohapi Creek	87	C				
Orari Ohapi	96	NC	82	C	96	C
Opihi upstream	98	NC				
Doncaster	29	C			77	C
Washdyke Lagoon	50	C			70	C
<b>max</b>	<b>98</b>		<b>99</b>		<b>96</b>	
<b>min</b>	<b>29</b>		<b>78</b>		<b>70</b>	
<b>median</b>	<b>95</b>		<b>93</b>		<b>78.5</b>	
<b>% non-cancer risk</b>		<b>50%</b>		<b>20%</b>		<b>0%</b>

<sup>a</sup> Percentage mercury contribution to non-cancer risk.

<sup>b</sup> Highest risk for specified species and site; C = cancer risk, NC = non-cancer risk.

The data in Table 5 further illustrate the previous conclusion that the defining risk of consuming fish from the rohe of Arowhenua (i.e., cancer vs non-cancer risk) is largely determined by mercury contamination. For eels, if the mercury proportion of the total risk is > 97% then non-cancer risk prevails. If mercury contamination is < 95% then cancer risk prevails. A similar relationship exists for trout, where non-cancer risk dominates only at 99% mercury contribution. For flounder, cancer risk dominated all risk.

The finding that mercury is such a dominant toxicant is unexpected, especially as there are no geothermal inputs in this region. Mercury is ubiquitous in the environment and bioaccumulates in fish tissue as methylmercury. The mercury concentrations observed in the fish could be due to background level mercury. This point is illustrated by the reference site, Te Nga Wai, which had a consumption limit of 3.1 meals/month (refer to Figure 15), that was dominated by mercury contamination (Table 5). Te Nga Wai is far removed from urbanisation, so little anthropogenic input of mercury would be expected. This was supported by the ratio of sediment concentrations of contaminants for each site relative to Te Nga Wai (Stewart et al., 2010). Te Nga Wai contaminant sediment concentrations were generally below other sites and for mercury was equal to one site (Opihi River upstream) and below all other sites. Therefore elevated mercury contamination of fish is presumably not driven by sediment mercury concentrations.

Eel tissue mercury concentrations were compared with eel age and % lipid which allowed us to determine whether age or condition were factors contributing to our observations of elevated mercury. There was no apparent relationship between mercury eel tissue concentrations and the age of the eel ( $r = 0.14$ ) or between mercury eel tissue concentrations and % lipid of the eel tissue ( $r = 0.42$ ). A multivariate regression for age and % lipid was also not significant ( $r = 0.28$ ), suggesting the sample size was too small ( $n=9$  for eel age and  $n=10$  for % lipid)

## 5. Implications of risk assessment to Arowhenua iwi

Local consumption rates and meal sizes were calculated from the interview data, with results presented in Table 6. Total fish consumption (43 g/day) for people who contributed to the questionnaire is comparable with the New Zealand ‘high’ consumption rate of 43 g/day (Kim & Smith 2006), showing that fish is a major dietary component for these people. However, traditional fish (i.e., harvested kai species) comprised only a small proportion (13%) of the total amount of fish consumed. With respect to the three species of wild fish, eels had the highest consumption rate (0.9 meals/month), followed by flounder (0.7 meals/month), and then trout (0.6 meals/month). Overall, watercress had the highest consumption (1.0 meals/month), possibly because of its ease of collection and abundance compared to local fish.

**Table 6:** Kai consumption rates and meal sizes of Maori from the rohe of Arowhenua.

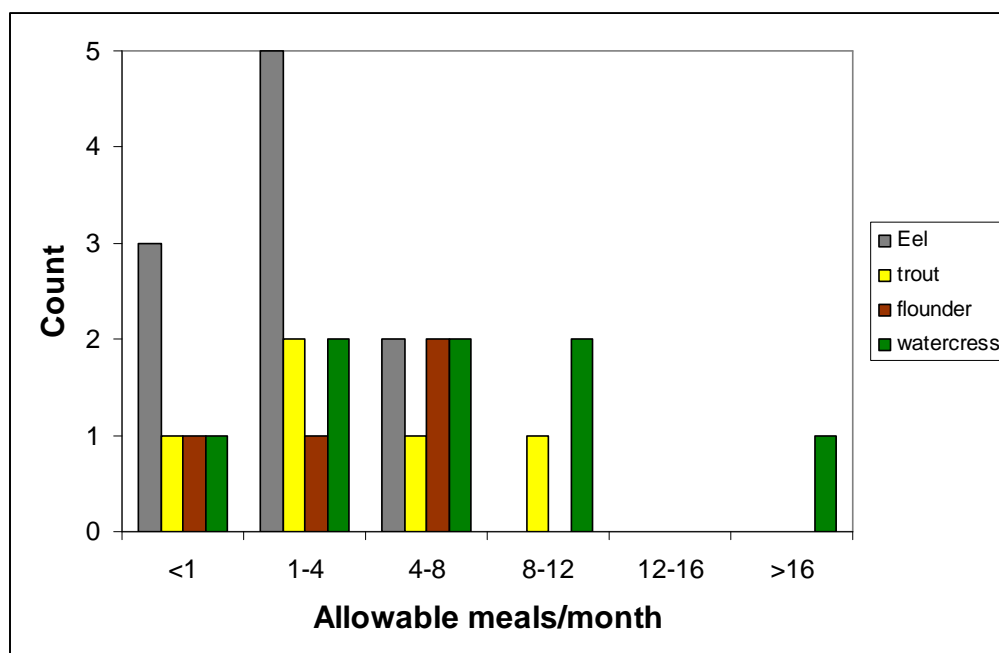
Kai species	Average meal size per sitting (g)	Consumption		
		g/month	g/day	meals/month
Total fish <sup>a</sup>	213	1300	43	6.1
Traditional fish <sup>b</sup>	220	174	5.7	0.8
Eel	213	184	6.1	0.9
Trout	213	121	4.0	0.6
Flounder	213	142	4.7	0.7
Watercress	175	183	6.0	1.0

<sup>a</sup> Includes fish from all sources.

<sup>b</sup> Fish harvested from the wild.

The distribution of each species across the allowable meals/month categories was correlated and is shown in Figure 16. These data clearly show that eel represent the highest risk to consumers, with 8 out of the 10 eels caught having an ‘allowable meal per month’ consumption rate of less than 4. The allowable consumption rate data for trout and flounder were more widely spread, with watercress even more so.

**Figure 16:** Distribution of local kai by allowable meals/month.



Given that local consumption rates are 0.9, 0.6, 0.7 and 1.0 meals/month for eel, trout, flounder and watercress, respectively, some allowable consumption limits are within the range of actual consumption rates (Figure 16).

There are two risk scenarios that need to be addressed. The first is where consumption of kai occurs from sites randomly throughout the rohe of Arowhenua, and the second is what would be the risk of consumption of the most contaminated kai only. The first scenario can be approximated by a risk assessment of the median concentrations of contaminant in biota. Comparisons of the allowable consumption rates of the highest risk (i.e., either cancer or non-cancer risk), using the median contaminant concentration data, with actual consumption rates enables assessment of the first scenario (Table 7). This shows that if harvesting was carried out randomly across all sites *and* consumption rates were as calculated from the questionnaire data, then there is *no significant risk* to members of Arowhenua iwi.

**Table 7:** Comparison of allowable consumption limits for *median* contamination data and actual consumption rates for questionnaire participants.

Kai species	Monthly fish consumption limits (meals/month)	Actual consumption rate (meals/month)
eel	3.3	0.9
trout	2.3	0.6
flounder	5.1	0.7
watercress	7.4	1.0

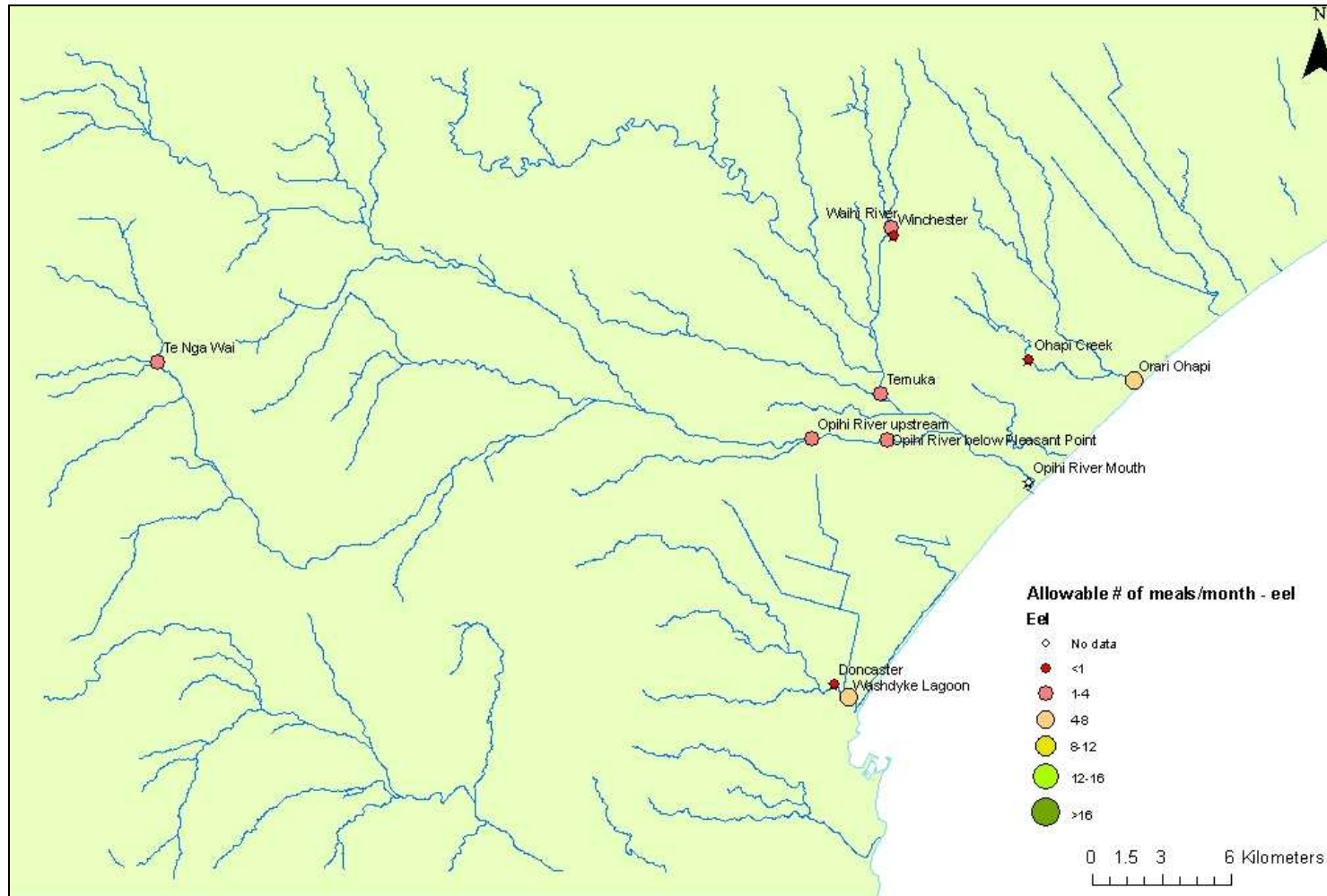
The second scenario, where harvesting is carried out primarily of the most contaminated biota, is defined by the 95<sup>th</sup> percentile contaminant concentrations (Table 8). If this was to occur *and* consumption rates were as calculated from the questionnaire data, then a *significant risk* is apparent for the consumption of eel. Trout are also a probable cause for concern with the consumption limit of 0.8 meals/month being very close to the actual consumption rate of 0.6 meals/month.

**Table 8:** Comparison of allowable consumption limits for 95<sup>th</sup> percentile contamination data and actual consumption rates for questionnaire participants. Bold white indicates exceedance of allowable consumption limit.

Kai species	Monthly fish consumption limits (meals/month)	Actual consumption rate (meals/month)
eel	<b>0.3</b>	0.9
trout	0.8	0.6
flounder	1.5	0.7
watercress	2.9	1.0

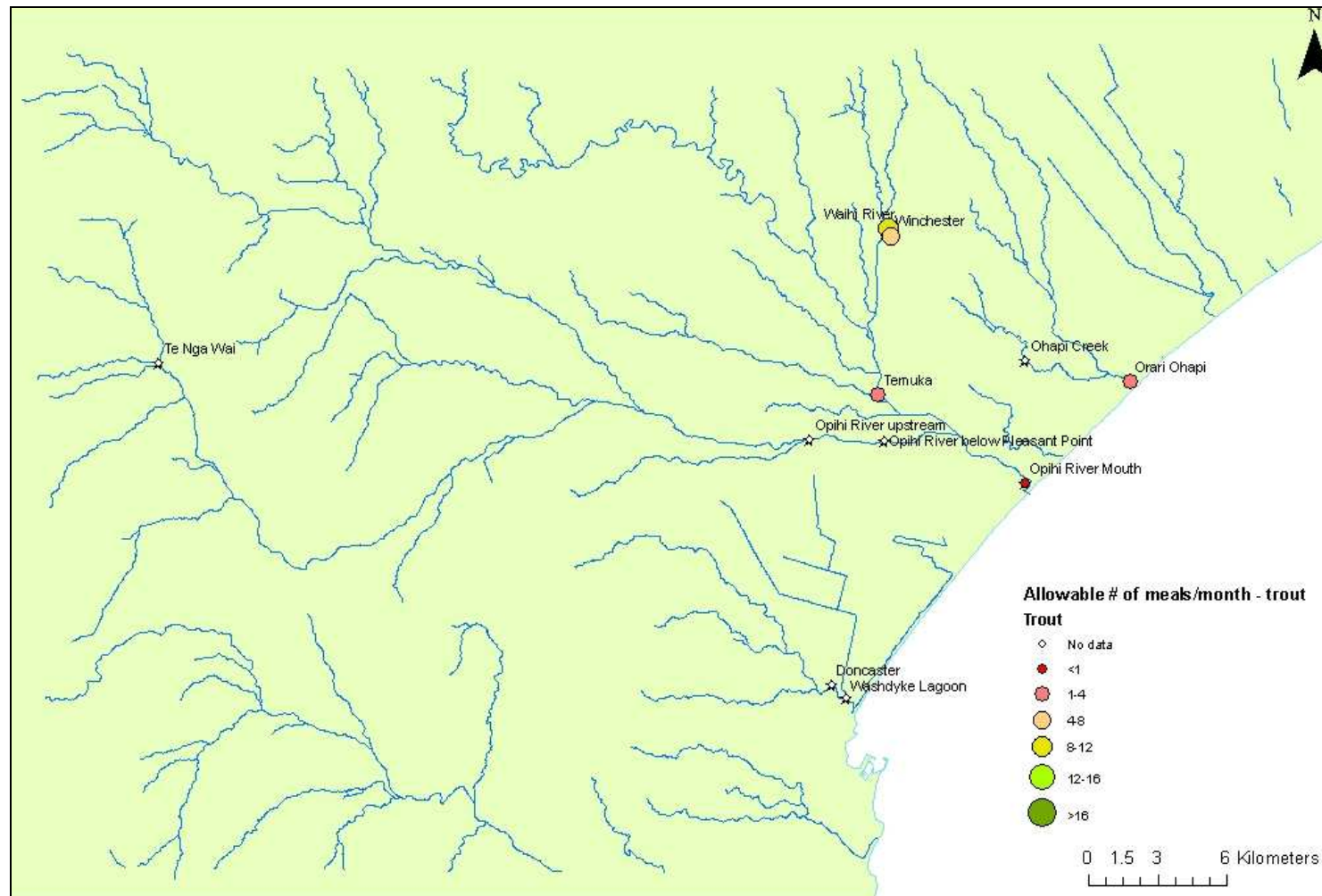
To assess which sites are those of concern across the whole region, a summary of allowable meals/month for each of the 4 species in this study is shown in Figures 17 to 20. Each figure gives a pictorial account, binned into categories of allowable meals, for ease of interpretation.

**Figure 19:** Allowable number of meals per month for eels from the rohe of Arowhenua<sup>a</sup>.



<sup>a</sup> Based on highest risk factor, which may vary between sites.

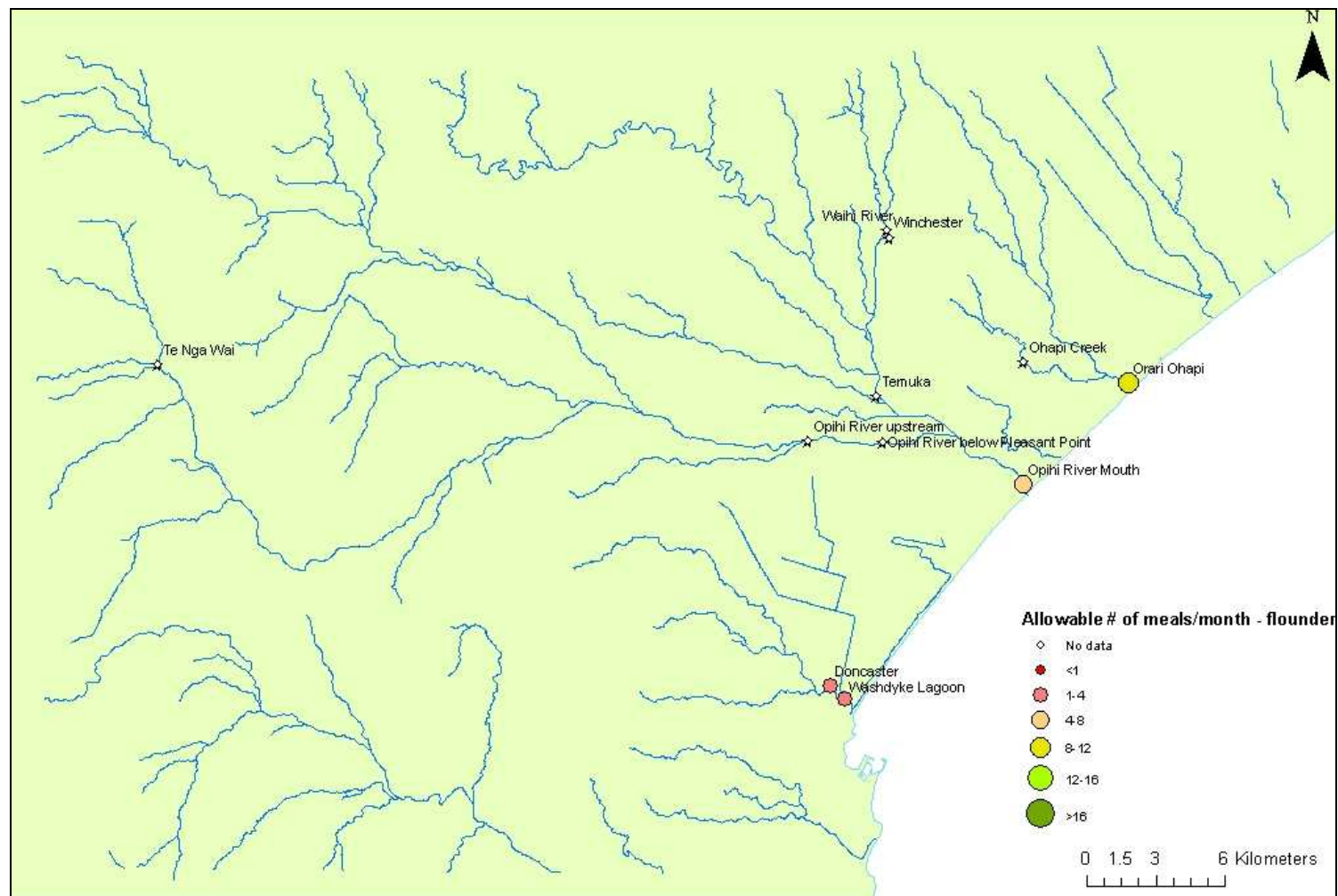
**Figure 20:** Allowable number of meals per month for trout from the rohe of Arowhenua<sup>a</sup>.



<sup>a</sup> Based on highest risk factor, which may vary between sites.

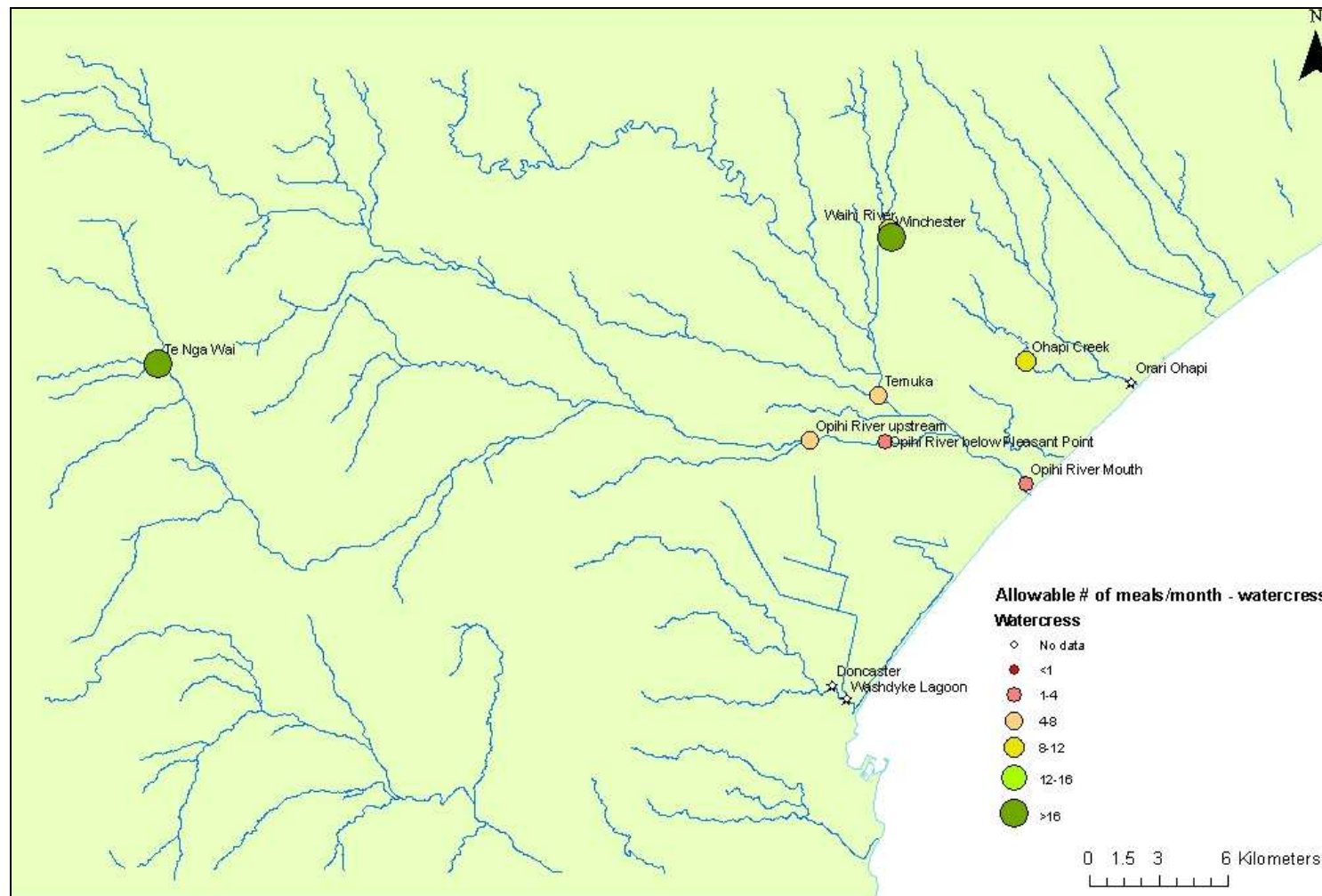


**Figure 21:** Allowable number of meals per month for flounder from the rohe of Arowhenua<sup>a</sup>.



<sup>a</sup> Based on highest risk factor, which may vary between sites.

**Figure 22:** Allowable number of meals per month for watercress from the rohe of Arowhenua<sup>a</sup>.



<sup>a</sup> Based on highest risk factor, which may vary between sites.

Based on the risk assessment process in this report, consumption of eel from Doncaster, Ohapi Creek or Winchester should be less than once per month (Figure 19). Furthermore, consumption should also be limited for eel harvested from Waihi River, Temuka, Opihi River upstream and below Pleasant Point to 1- 4 meals/month. Sampling was limited, however, it is of interest to note that the most contaminated eels (Doncaster site) should not be consumed at a rate exceeding 0.2 meals/month (Figure 10), which corresponds to around one meal every six months.

Another interesting observation was that no eel tissue had detectable concentrations of arsenic. Arsenic is highly carcinogenic (in the inorganic form) so would contribute significantly to the cancer risk. This was noted for the cancer risk assessment of trout and flounder from this study. Arsenic detection limits in this study were sample-dependant but around 0.1 mg/kg (dry weight). Arsenic was detected in trout from this study at concentrations ranging from below detection limits (0.1 mg/kg) to 3.7 mg/kg and a median concentration of 1.55 mg/kg (Stewart et al., 2010). Flounder from this study had median arsenic concentrations of 0.59 mg/kg, ranging from 0.28 mg/kg to 2.2 mg/kg (Stewart et al., 2010).

With respect to trout, one specimen harvested from Opihi River mouth had contaminant concentrations resulting in a strict consumption limit of less than one meal per month (Figure 20). The risk from this trout from Opihi River mouth was dominated by arsenic (Figure 6). These trout are possibly sea-run trout, i.e., they have recently returned from the ocean. If this was the case, then the contamination profile is not representative of that site, but of a marine fish. The US EPA concluded that an assumption that 10% of total arsenic ( $As_{tot}$ ) is the toxic inorganic arsenic ( $As_i$ ) in resident freshwater fish provided a protective estimate of health risk (US EPA 2003). This was supported by a more recent survey of the literature, which concluded that for freshwater fish,  $As_i$  was 10% of total arsenic ( $As_{tot}$ ) at the 75<sup>th</sup> percentile (Schoof & Yager 2007). For marine and estuarine fish, the  $As_i$  proportion is reportedly much lower at only 2-3% (Schoof & Yager 2007), lower than the 10% approximation used in this risk assessment. Accordingly, this would reduce the risk for marine or estuarine fish by a factor of 3-5 where arsenic is the predominant contaminant. Schoof and Yager (2007) stated that there was “little correlation between  $As_{tot}$  concentrations and  $As_i$  concentrations, however, when only  $As_{tot}$  data are available to assess health risks from arsenic in seafood, these data could support conservative, upper end estimates of the percent of  $As_{tot}$  likely to be  $As_i$ ”. However, without arsenic speciation studies to determine accurate  $As_i$  concentrations, a conservative approach is always more prudent and has been used in this case, i.e., a 10% approximation of  $As_{tot}$  that is present as  $As_i$ .

Based on the tissue concentrations from trout collected at Temuka and Orari Ohapi, a recommended consumption limit of 1-4 meals/month was derived. As such, a degree of caution should be exercised when consuming trout from these sites. Again, arsenic was the dominant contaminant in these fish and for the reason outlined above (i.e., proximity to ocean), the risk calculated for trout from the Orari Ohapi site may be overly conservative.

Trout are highly mobile fish and in this study only one specimen was analysed per site. Therefore, high flesh contaminant concentrations at a specific site does not necessarily mean that all trout caught at that site will have high contaminant concentrations. Conversely, a trout that has low flesh contaminant concentrations does not necessarily infer all trout from that site will also have low flesh contaminant concentrations. To more accurately assess spatial contamination of trout (and other highly mobile fish), it would be necessary to do a more extensive study, analysing multiple trout from multiple sites. However, the risk assessment data does suggest *the possibility* that some trout that are harvested from the rohe of Arowhenua will have high levels of contamination.

Flounder from Washdyke Lagoon and Orari Ohapi are the greatest risk, with allowable consumption limits of 1-4 meals/month (Figure 21). For the flounder from Washdyke Lagoon, the consumption limit is 1.1 meals/month. The dominant contaminant is arsenic (81%), with significant inputs of p,p'-DDE (9%) and dieldrin (8%). The flounder from Orari Ohapi had a consumption limit of 4.1 meals/month, with the dominant contaminants being arsenic (86%), p,p'-DDE (8%) and dieldrin (6%). There is good agreement between the contaminant proportions for the flounder from Washdyke Lagoon and Orari Ohapi. In contrast, flounder from Doncaster had contaminant proportions of 41% for dieldrin, 38% for arsenic and 16% for p,p'-DDE.

Watercress consumption risk was highest when harvested from the Opihi River (Figure 22), with consumption limits of 2.7 meals/month (below Pleasant Point site), 3.2 meals/month (river mouth site) and 6.6 meals/month (upstream site) (Figures 4-6). These were all lifetime cancer risks and the only contaminant contributing to the risk associated with consumption was arsenic (Figures 4 to 6).

## 6. Conclusions

Any conclusions made from this study need to bear in mind certain limitations, specifically:

- there was a small sample size of people who completed the kai consumption questionnaire (n=12), so consumption rates and meal sizes are not based on statistically robust data
- it was not possible to collect all species at all sites, with some relevant species (whitebait, marine mussels) unable to be collected at all, so the species studied may not be a good representation of the catchment as a whole for dietary consumption
- there was usually only a single specimen collected per site (with the exception of all watercress and flounder at one site, which were composite samples), so caution must be made when applying consumption limits on a site by site basis
- not all contaminants were analysed in all kai, e.g., PCBs were only analysed in eels (since these represented the highest bioaccumulation risk based on their high lipid levels).

On the basis of the contaminant concentrations found in kai species from 12 sites around the rohe of Arowhenua (Stewart et al., 2010) and the risk assessment described herein, the following key findings are summarised as follows:

- actual local average consumption rates of wild kai were calculated as 6.1, 4.0, 4.7 and 6.0 g/day for eels, trout, flounder and watercress, respectively. Total fish consumption was in the NZ 'high' consumption category, but only 13% of this was from wild caught fish
- meal sizes were calculated as 213 g/meal for all fish species, and 175 g/meal for watercress
- sediment contaminant concentrations were generally below the ANZECC Interim Sediment Quality Guidelines (ISQGs), suggesting these ecological health guidelines are not appropriate for assessing human health
- contaminant uptake was species-specific, with concentrations:

- for ΣDDT: eel >> trout = flounder
- for mercury: eel > trout > flounder > watercress
- for arsenic: trout > flounder > watercress > eel
- for cadmium, lead and chromium: watercress >> all fish.
- the results of the risk assessment were clear. In terms of the whole catchment:
  - if harvesting was carried out randomly across all sites *and* consumption rates were as calculated from the questionnaire data, then there is *no significant risk* for consumption of any kai species analysed
  - if harvesting were to occur predominantly of the most contaminated kai *and* consumption rates were as calculated from the questionnaire data, then a *significant risk* exists for consumption of eel and trout.
- the risk of eating eels in Arowhenua was greater than other species, with 8 out of 10 eels sampled allowing for less than 1 meal per week with:
  - mercury the dominant toxicant for non-cancer risk in 5 out of 10 eels
  - p,p'-DDE, dieldrin and PCBs the dominant toxicants for cancer risk.
- the area of Washdyke consistently showed the highest risk of consumption of kai, however significant risk of consumption was not limited to this area
- Te Nga Wai proved to be satisfactory as a reference site (i.e., as far removed from human influence as practical), with generally lower contamination of sediments and kai than the other sites. The exception was mercury in eels, for which Te Nga Wai was approximately the same as other sites, suggesting that mercury uptake by eels in the rohe of Arowhenua is largely from background (i.e., non-elevated) levels of mercury.

The results from this study clearly illustrate the need to more accurately assess the risk of consuming wild kai in the rohe of Arowhenua by:

- collecting samples from more sites, species (e.g., both long fin and short fin eels) and with multiple specimens at each site, so a more statistically robust spatial assessment can be made of risk
  
- expanding the contaminant dataset to include:
  - PCB analyses in all fish
  
  - metal speciation studies on arsenic and mercury - for at least a subset of each kai species at representative locations, i.e., estuarine, river, marine - to more accurately gauge risk
  
  - obtaining a more robust dataset of kai consumption in the region, by including more local Māori and non-Māori consumers of wild kai, in the questionnaire process, and
  
  - conducting a risk assessment for total fish diet which incorporates both wild and commercial dietary consumption.

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## 8. Abbreviations

<b>ANZECC</b>	Australian and New Zealand Environmental Conservation Council.
<b>DDD</b>	Dichlorodiphenyldichloroethane.
<b>DDE</b>	Dichlorodiphenyldichloroethylene.
<b>DDT</b>	Dichlorodiphenyltrichloroethane.
<b>γ-HCH</b>	Gamma-hexachlorocyclohexane = lindane.
<b>HCB</b>	Hexachlorobenzene.
<b>ISQG</b>	Interim sediment quality guidelines.
<b>kg</b>	kilogram(s).
<b>MfE</b>	Ministry for the Environment.
<b>mg</b>	milligram
<b>MoH</b>	Ministry of Health.
<b>mm</b>	millimetre(s).
<b>NZ</b>	New Zealand.
<b>OCP</b>	Organochlorine pesticide.
<b>PAH</b>	Polycyclicaromatic hydrocarbons.
<b>PCB</b>	Polychlorinated biphenyl.
<b>PCP</b>	Pentachlorophenol
<b>ppb</b>	1 part per billion = 1 µg/kg.

<b>ppm</b>	1 part per million = 1 mg/kg.
<b>TOC</b>	Total Organic Carbon.
<b>µg</b>	microgram.
<b>US EPA</b>	United States Environmental Protection Association.

## 9. Glossary

<b>Anthropogenic</b>	Effects, processes, or materials that are derived from human activities.
<b>Aquatic</b>	Dwelling in water.
<b>Bioaccumulation</b>	Accumulation of a chemical by an aquatic organism.
<b>Biomagnification</b>	The increase in concentration of a substance up the food chain.
<b>Catchment</b>	An area of land from which water from rainfall drains toward a common watercourse, stream, river, lake, or estuary.
<b>Chronic toxicity</b>	Long-term effect on an organism, usually caused by toxic substances.
<b>Concentration</b>	The measure of how much of a given substance there is mixed with another substance.
<b>Congener</b>	In chemistry, congeners are related chemicals, e.g., There are 209 congeners of polychlorinated biphenyls (see PCB).
<b>Contaminant</b>	Any substance (including gases, odorous compounds, liquids, solids, and micro-organisms) or energy (excluding noise), or heat, that results in an undesirable change to the physical, chemical, or biological environment. Also called pollutant.
<b>Detection limit</b>	A value below which the laboratory analyst is not confident that any apparent concentration is real.
<b>Dioxins</b>	The by-products of various industrial processes (such as bleaching paper pulp, and chemical and pesticide manufacture) and combustion activities (such as burning rubbish, forest fires, and waste incineration).

<b>Guideline</b>	Numerical limit for a chemical, or a narrative statement, recommended to support and maintain a designated water use.
<b>Hazardous</b>	Having the capacity to adversely affect either health or the environment.
<b>Indigenous</b>	Native, or belonging naturally to a given region or ecosystem, as opposed to exotic or introduced (can be used for people, animal, or plant species or even mineral resources).
<b>Iwi</b>	A Maori tribal group.
<b>Kai</b>	Traditional Māori food.
<b>Median</b>	In statistics, the middle score in a range of samples or measurements (that is, half the scores will be higher than the median and half will be lower).
<b>Organochlorine</b>	A chemical that contains carbon and chlorine atoms joined together. Some organochlorines are persistent (remain chemically stable) and present a risk to the environment and human health, such as dioxin, DDT and PCBs.
<b>ppb</b>	1 part per billion = $1 \text{ mg m}^{-3} = 1 \text{ } \mu\text{g L}^{-1}$ .
<b>ppm</b>	1 part per million = $1 \text{ g m}^{-3} = 1 \text{ mg L}^{-1}$ .
<b>Risk Assessment</b>	The determination of a quantitative or qualitative value of risk related to a concrete situation and a recognised threat.
<b>Rohe</b>	The geographical territory of an iwi or a hapu.
<b>Runanga</b>	The governing council or administrative group of a Māori hapu or Iwi.
<b>Screen</b>	A low-cost monitoring method used to make an initial assessment.

<b>Sediment</b>	Particles or clumps of particles of sand, clay, silt, or plant or animal matter carried in water.
<b>Soluble</b>	Fraction of material that passes through a filter (international convention uses a 0.45 $\mu$ m membrane filter).
<b>Species</b>	One of the basic units of biological classification. A species comprises individual organisms that are very similar in appearance, anatomy, physiology, and genetics, due to having relatively recent common ancestors; and can interbreed.
<b>Stormwater</b>	Flow of water from urban surface areas after rainfall.
<b>Total metal</b>	The concentration of a metal in an unfiltered sample that is digested in strong acid.
<b>Toxic substance</b>	A material able to cause adverse effects in living organisms.
<b>Toxicity</b>	Is the inherent potential or capacity of a material to cause adverse effects on living organisms.
<b>Vascular</b>	Containing vessels which conduct fluid.

## 10. Appendices

**Appendix 1a: Temuka consumption limit calculations<sup>a</sup>**

Species	Compound	Contaminant Concentration (mg/kg wet weight)	Input Data/Assumptions			Daily Consumption Limits (g/day)		Monthly Fish Consumption Limits (meals/month)	
			Slope Factor (mg/kg/day) <sup>-1</sup>	Body weight (kg)	Reference Dose (mg/kg/day)	ARL	Cancer Risk	Non Cancer Risk	Cancer Risk
Eel	p,p-DDT	1.52E-03	0.34	80	5.0E-04	1.0E-05	1548	26322	3716.0
	p,p-DDD	1.15E-03	0.24	80	NA	1.0E-05	2909		
	p,p-DDE	3.75E-02	0.34	80	NA	1.0E-05	63		
	Lindane	0.00E+00	1.30	80	3.0E-04	1.0E-05			
	Dieldrin	1.73E-04	16.00	80	5.0E-05	1.0E-05	289	23146	3267.6
	Chlordanes (total)	2.18E-04	0.35	80	5.0E-04	1.0E-05	10496	183686	25932.2
	HCB	5.64E-05	1.60	80	8.0E-04	1.0E-05	8870	1135300	160277.7
	PCBs (total)	2.35E-03	2.00	80	2.0E-05	1.0E-05	170	681	96.1
	Cadmium	0.00E+00	NA	80	1.0E-03	1.0E-05			
	Mercury	3.83E-01	NA	80	1.0E-04	1.0E-05		21	<b>3.0</b>
	Arsenic 10%	0.00E+00	1.50	80	3.0E-04	1.0E-05			
	Zinc	8.75E+00	NA	80	3.0E-01	1.0E-05		2743	387.2
	Nickel	0.00E+00	NA	80	2.0E-02	1.0E-05			
	Chromium	0.00E+00	NA	80	3.0E-03	1.0E-05			
					<b>TOTAL</b>	38		<b>5.3</b>	
Trout	p,p-DDT	3.65E-04	0.34	80	5.0E-04	1.0E-05	6448	109624	15476.3
	p,p-DDD	3.75E-04	0.24	80	NA	1.0E-05	8897		
	p,p-DDE	1.85E-02	0.34	80	NA	1.0E-05	127		
	Lindane	0.00E+00	1.30	80	3.0E-04	1.0E-05			
	Dieldrin	1.66E-04	16.00	80	5.0E-05	1.0E-05	302	24158	3410.6
	Chlordanes (total)	4.82E-05	0.35	80	5.0E-04	1.0E-05	47387	829277	117074.4
	HCB	2.31E-05	1.60	80	8.0E-04	1.0E-05	21600	2764777	390321.5
	Cadmium	0.00E+00	NA	80	1.0E-03	1.0E-05			
	Mercury	5.00E-01	NA	80	1.0E-04	1.0E-05		16	<b>2.3</b>
	Arsenic 10%	4.76E-02	1.50	80	3.0E-04	1.0E-05	11.2	504	71.1
	Zinc	3.57E+00	NA	80	3.0E-01	1.0E-05		6718	948.5
	Nickel	0.00E+00	NA	80	2.0E-02	1.0E-05			
	Chromium	0.00E+00	NA	80	3.0E-03	1.0E-05			
						<b>TOTAL</b>	10		<b>1.4</b>
Watercress	Cadmium	7.39E-03	NA	80	1.0E-03	1.0E-05		10827	1861.3
	Mercury	5.91E-04	NA	80	1.0E-04	1.0E-05		13533	2326.6
	Arsenic	1.87E-02	1.50	80	3.0E-04	1.0E-05	28.5	1282	<b>4.9</b> <b>220.4</b>
	Zinc	2.17E+00	NA	80	3.0E-01	1.0E-05		11073	1903.6
	Nickel	6.40E-02	NA	80	2.0E-02	1.0E-05		24985	4295.3
	Chromium	5.42E-02	NA	80	3.0E-03	1.0E-05		4429	761.4

<sup>a</sup> Additive cancer risk consumption limits and greatest non-cancer risk consumption limits indicated by black box.



**Appendix 1b: Opihi River upstream consumption limit calculations<sup>a</sup>.**

Species	Compound	Input Data/Assumptions					Daily Consumption Limits (g/day)		Monthly Fish Consumption Limits	
		Contaminant Concentration (mg/kg wet weight)	Slope Factor (mg/kg/day) <sup>-1</sup>	Body weight (kg)	Reference Dose (mg/kg/day)	ARL	Cancer Risk	Non Cancer Risk	Cancer Risk	Non Cancer Risk
Eel	p,p-DDT	1.30E-03	0.34	80	5.0E-04	1.0E-05	1806	30707		4335.1
	p,p-DDD	7.80E-04	0.24	80	NA	1.0E-05	4272			
	p,p-DDE	2.15E-02	0.34	80	NA	1.0E-05	109			
	Lindane	0.00E+00	1.30	80	3.0E-04	1.0E-05				
	Dieldrin	4.61E-04	16.00	80	5.0E-05	1.0E-05	108	8680		1225.4
	Chlordanes (total)	9.22E-05	0.35	80	5.0E-04	1.0E-05	24800	433995		61269.9
	HCB	1.31E-04	1.60	80	8.0E-04	1.0E-05	3826	489683		69131.8
	PCBs (total)	6.22E-04	2.00	80	2.0E-05	1.0E-05	643	2574		363.4
	Cadmium	1.71E-02	NA	80	1.0E-03	1.0E-05		4692		662.3
	Mercury	5.41E-01	NA	80	1.0E-04	1.0E-05		15		<b>2.1</b>
	Arsenic 10%	0.00E+00	1.50	80	3.0E-04	1.0E-05				
	Zinc	9.98E+00	NA	80	3.0E-01	1.0E-05		2404		339.5
	Nickel	0.00E+00	NA	80	2.0E-02	1.0E-05				
	Chromium	0.00E+00	NA	80	3.0E-03	1.0E-05				
						<b>TOTAL</b>	48		<b>6.7</b>	
Watercress	Cadmium	1.79E-02	NA	80	1.0E-03	1.0E-05		4464		767.4
	Mercury	0.00E+00	NA	80	1.0E-04	1.0E-05				
	Arsenic	1.38E-02	1.50	80	3.0E-04	1.0E-05	39	1733	6.6	<b>297.9</b>
	Zinc	2.85E+00	NA	80	3.0E-01	1.0E-05		8418		1447.2
	Nickel	2.36E-01	NA	80	2.0E-02	1.0E-05		6773		1164.4
	Chromium	1.79E-02	NA	80	3.0E-03	1.0E-05		13392		2302.3

<sup>a</sup> Additive cancer risk consumption limits and greatest non-cancer risk consumption limits indicated by black box.

**Appendix 1c: Opihi River below Pleasant Point consumption limit calculations<sup>a</sup>.**

Species	Compound	Input Data/Assumptions				Monthly Fish Consumption Limits (meals/month)				
		Contaminant Concentration (mg/kg wet weight)	Slope Factor (mg/kg/day)-1	Body weight (kg)	Reference Dose (mg/kg/day)	Daily Consumption Limits (g/day)		Non Cancer		
						ARL	Cancer Risk	Risk	Cancer Risk	Non Cancer Risk
<b>Eel</b>	p,p-DDT	2.77E-04	0.34	80	5.0E-04	1.0E-05	8496	144437		20391.1
	p,p-DDD	1.17E-04	0.24	80	NA	1.0E-05	28394			
	p,p-DDE	8.55E-03	0.34	80	NA	1.0E-05	275			
	Lindane	0.00E+00	1.30	80	3.0E-04	1.0E-05				
	Dieldrin	0.00E+00	16.00	80	5.0E-05	1.0E-05				
	Chlordanes (total)	2.86E-05	0.35	80	5.0E-04	1.0E-05	79929	1398756		197471.5
	HCB	1.75E-05	1.60	80	8.0E-04	1.0E-05	28549	3654289		515899.6
	PCBs (total)	1.36E-03	2.00	80	2.0E-05	1.0E-05	295	1181		166.7
	Cadmium	2.64E-03	NA	80	1.0E-03	1.0E-05		30299		4277.5
	Mercury	4.56E-01	NA	80	1.0E-04	1.0E-05		18		<b>2.5</b>
	Arsenic 10%	0.00E+00	1.50	80	3.0E-04	1.0E-05				
	Zinc	7.68E+00	NA	80	3.0E-01	1.0E-05		3125		441.1
	Nickel	0.00E+00	NA	80	2.0E-02	1.0E-05				
	Chromium	0.00E+00	NA	80	3.0E-03	1.0E-05				
					<b>TOTAL</b>	138		<b>19.5</b>		
<b>Watercress</b>	Cadmium	7.98E-03	NA	80	1.0E-03	1.0E-05		10030		1724.3
	Mercury	7.44E-04	NA	80	1.0E-04	1.0E-05		10746		1847.5
	Arsenic	3.35E-02	1.50	80	3.0E-04	1.0E-05	15.9	716	2.7	<b>123.2</b>
	Zinc	2.02E+00	NA	80	3.0E-01	1.0E-05		11877		2042.0
	Nickel	9.57E-02	NA	80	2.0E-02	1.0E-05		16716		2873.9
	Chromium	4.47E-02	NA	80	3.0E-03	1.0E-05		5373		923.7

<sup>a</sup> Additive cancer risk consumption limits and greatest non-cancer risk consumption limits indicated by black box.

**Appendix 1d: Opihi River mouth consumption limit calculations<sup>a</sup>.**

Species	Compound	Input Data/Assumptions					Daily Consumption Limits (g/day)		Monthly Fish Consumption Limits	
		Contaminant Concentration (mg/kg wet weight)	Slope Factor (mg/kg/day) <sup>-1</sup>	Body weight (kg)	Reference Dose (mg/kg/day)	ARL	Cancer Risk	Non	Cancer Risk	Non
								Cancer Risk		Cancer Risk
Trout	p,p-DDT	1.34E-04	0.34	80	5.0E-04	1.0E-05	17553	298409		42128.3
	p,p-DDD	6.78E-05	0.24	80	NA	1.0E-05	49163			
	p,p-DDE	3.19E-03	0.34	80	NA	1.0E-05	738			
	Lindane	0.00E+00	1.30	80	3.0E-04	1.0E-05				
	Dieldrin	1.08E-04	16.00	80	5.0E-05	1.0E-05	461	36878		5206.3
	Chlordanes (total)	9.90E-06	0.35	80	5.0E-04	1.0E-05	230829	4039506		570283.3
	HCB	1.48E-05	1.60	80	8.0E-04	1.0E-05	33774	4323122		610323.2
	Cadmium	0.00E+00	NA	80	1.0E-03	1.0E-05				
	Mercury	1.14E-01	NA	80	1.0E-04	1.0E-05		70		9.9
	Arsenic 10%	8.95E-02	1.5000	80	3.0E-04	1.0E-05	6.0	268		37.9
	Zinc	4.59E+00	NA	80	3.0E-01	1.0E-05		5223		737.4
	Nickel	0.00E+00	NA	80	2.0E-02	1.0E-05				
	Chromium	0.00E+00	NA	80	3.0E-03	1.0E-05				
							<b>TOTAL</b>	5.8		<b>0.8</b>
Flounder	p,p-DDT	0.0004	0.34	80.00	5.0E-04	1.0E-05	6031	102532		14475.1
	p,p-DDD	3.75E-04	0.24	80	NA	1.0E-05	8885			
	p,p-DDE	8.75E-03	0.34	80	NA	1.0E-05	269			
	Lindane	0.00E+00	1.30	80	3.0E-04	1.0E-05				
	Dieldrin	1.25E-04	16.00	80	5.0E-05	1.0E-05	401	32055		4525.4
	Chlordanes (total)	3.05E-05	0.35	80	5.0E-04	1.0E-05	75048	1313347		185413.7
	HCB	0.00E+00	1.60	80	8.0E-04	1.0E-05				
	Cadmium	0.00E+00	NA	80	1.0E-03	1.0E-05				
	Mercury	2.07E-02	NA	80	1.0E-04	1.0E-05		387		54.6
	Arsenic 10%	8.27E-03	1.5000	80	3.0E-04	1.0E-05	64.5	2901		409.5
	Zinc	4.70E+00	NA	80	3.0E-01	1.0E-05		5105		720.7
	Nickel	8.46E-02	NA	80	2.0E-02	1.0E-05		18908		2669.4
	Chromium	0.00E+00	NA	80	3.0E-03	1.0E-05				
							<b>TOTAL</b>	45.4		<b>6.4</b>
Watercress	Cadmium	0.0173	NA	80	1.0E-03	1.0E-05		4630		796.0
	Mercury	0.00E+00	NA	80	1.0E-04	1.0E-05				
	Arsenic	2.85E-02	1.5000	80	3.0E-04	1.0E-05	18.7	842	3.2	144.7
	Zinc	3.63E+00	NA	80	3.0E-01	1.0E-05		6615		1137.2
	Nickel	1.04E-01	NA	80	2.0E-02	1.0E-05		15434		2653.4
	Chromium	5.36E-02	NA	80	3.0E-03	1.0E-05		4481		770.3

<sup>a</sup> Additive cancer risk consumption limits and greatest non-cancer risk consumption limits indicated by black box.

**Appendix 1e:** Ohapi Creek consumption limit calculations<sup>a</sup>.

Species	Compound	Input Data/Assumptions					Daily Consumption Limits (g/day)		Monthly Fish Consumption Limits (meals/month)	
		Contaminant Concentration (mg/kg wet weight)	Slope Factor (mg/kg/day) <sup>-1</sup>	Body weight (kg)	Reference Dose (mg/kg/day)	ARL	Cancer Risk	Non Cancer Risk	Cancer Risk	Non Cancer Risk
<b>Eel</b>	p,p-DDT	4.73E-03	0.34	80	5.0E-04	1.0E-05	497.3	8454		1193.5
	p,p-DDD	2.60E-03	0.24	80	NA	1.0E-05	1284.3			
	p,p-DDE	2.29E-01	0.34	80	NA	1.0E-05	10.3			
	Lindane	0.00E+00	1.30	80	3.0E-04	1.0E-05				
	Dieldrin	3.82E-03	16.00	80	5.0E-05	1.0E-05	13.1	1046		147.7
	Chlordanes (total)	1.78E-04	0.35	80	5.0E-04	1.0E-05	12865.1	225139		31784.3
	HCB	1.06E-04	1.60	80	8.0E-04	1.0E-05	4726.9	605045		85418.2
	PCBs (total)	5.03E-03	2.00	80	2.0E-05	1.0E-05	79.5	318		44.9
	Cadmium	7.47E-04	NA	80	1.0E-03	1.0E-05		107113		15121.9
	Mercury	2.32E-01	NA	80	1.0E-04	1.0E-05		35		<b>4.9</b>
	Arsenic 10%	0.00E+00	1.50	80	3.0E-04	1.0E-05				
	Zinc	1.03E+01	NA	80	3.0E-01	1.0E-05		2330		328.9
	Nickel	0.00E+00	NA	80	2.0E-02	1.0E-05				
	Chromium	0.00E+00	NA	80	3.0E-03	1.0E-05				
					<b>TOTAL</b>	5.3		<b>0.7</b>		
<b>Watercress</b>	Cadmium	2.31E-03	NA	80	1.0E-03	1.0E-05		34702		5966.0
	Mercury	0.00E+00	NA	80	1.0E-04	1.0E-05				
	Arsenic	1.09E-02	1.50	80	3.0E-04	1.0E-05	48.8	2195	8.4	<b>377.3</b>
	Zinc	2.10E+00	NA	80	3.0E-01	1.0E-05		11437		1966.3
	Nickel	2.51E-02	NA	80	2.0E-02	1.0E-05		63689		10949.3
Chromium	4.14E-02	NA	80	3.0E-03	1.0E-05		5800		997.2	

<sup>a</sup> Additive cancer risk consumption limits and greatest non-cancer risk consumption limits indicated by black box.

### Appendix 1f: Orari Ohapi consumption limit calculations<sup>a</sup>.

Species	Compound	Input Data/Assumptions				Daily Consumption Limits (g/day)		Monthly Fish Consumption Limits (meals/month)		
		Contaminant Concentration (mg/kg wet weight)	Slope Factor (mg/kg/day) <sup>-1</sup>	Body weight (kg)	Reference Dose (mg/kg/day)	ARL	Cancer Risk	Non Cancer Risk	Cancer Risk	Non Cancer Risk
Eel	p,p-DDT	2.23E-04	0.34	80	5.0E-04	1.0E-05	10546	179279	25310.0	
	p,p-DDD	4.69E-04	0.24	80	NA	1.0E-05	7114			
	p,p-DDE	1.43E-02	0.34	80	NA	1.0E-05	164			
	Lindane	0.00E+00	1.30	80	3.0E-04	1.0E-05				
	Dieldrin	4.01E-04	16.00	80	5.0E-05	1.0E-05	125	9973	1408.0	
	Chlordanes (total)	4.86E-05	0.35	80	5.0E-04	1.0E-05	47020	822843	116166.1	
	HCB	2.49E-05	1.60	80	8.0E-04	1.0E-05	20043	2565560	362196.8	
	PCBs (total)	1.01E-03	2.00	80	2.0E-05	1.0E-05	396	1582	223.3	
	Cadmium	1.79E-03	NA	80	1.0E-03	1.0E-05		44780	6321.8	
	Mercury	2.14E-01	NA	80	1.0E-04	1.0E-05		37	<b>5.3</b>	
	Arsenic 10%	0.00E+00	1.50	80	3.0E-04	1.0E-05				
	Zinc	1.10E+01	NA	80	3.0E-01	1.0E-05		2183	308.2	
	Nickel	4.12E-02	NA	80	2.0E-02	1.0E-05		38809	5478.9	
	Chromium	0.00E+00	NA	80	3.0E-03	1.0E-05				
						<b>TOTAL</b>	59.0		<b>8.3</b>	
Trout	p,p-DDT	6.97E-05	0.34	80	5.0E-04	1.0E-05	33739	573561	80973.3	
	p,p-DDD	4.36E-05	0.24	80	NA	1.0E-05	76431			
	p,p-DDE	2.21E-03	0.34	80	NA	1.0E-05	1063			
	Lindane	0.00E+00	1.30	80	3.0E-04	1.0E-05				
	Dieldrin	1.58E-04	16.00	80	5.0E-05	1.0E-05	317	25387	3584.1	
	Chlordanes (total)	6.42E-06	0.35	80	5.0E-04	1.0E-05	356146	6232548	879889.2	
	HCB	1.38E-05	1.60	80	8.0E-04	1.0E-05	36162	4628770	653473.5	
	Cadmium	0.00E+00	NA	80	1.0E-03	1.0E-05				
	Mercury	5.07E-02	NA	80	1.0E-04	1.0E-05		158	<b>22.3</b>	
	Arsenic 10%	2.66E-02	1.50	80	3.0E-04	1.0E-05	20.1	903	127.5	
	Zinc	4.83E+00	NA	80	3.0E-01	1.0E-05		4968	701.4	
	Nickel	0.00E+00	NA	80	2.0E-02	1.0E-05				
	Chromium	0.00E+00	NA	80	3.0E-03	1.0E-05				
						<b>TOTAL</b>	18.5		<b>2.6</b>	
	Flounder	p,p-DDT	1.70E-04	0.3400	80	5.0E-04	1.0E-05	13876	235893	33302.5
p,p-DDD		8.82E-05	0.2400	80	NA	1.0E-05	37795			
p,p-DDE		6.39E-03	0.34	80	NA	1.0E-05	368			
Lindane		0.00E+00	1.3	80	0.0003	0.00001				
Dieldrin		1.02E-04	16	80	0.00005	0.00001	491	39277	5545.0	
Chlordanes (total)		1.31E-05	0.35	80	0.0005	0.00001	174885	3060492	432069.4	
HCB		1.20E-05	1.6	80	0.0008	0.00001	41583	5322641	751431.6	
Cadmium		0.00E+00	NA	80	0.001	0.00001				
Mercury		9.57E-02	NA	80	0.0001	0.00001		84	<b>11.8</b>	
Arsenic 10%		5.58E-03	1.5	80	0.0003	0.00001	95.5	4299	606.8	
Zinc		2.10E+00	NA	80	3.0E-01	1.0E-05		11437	1614.6	
Nickel		2.51E-02	NA	80	2.0E-02	1.0E-05		63689	8991.3	
Chromium		4.14E-02	NA	80	3.0E-03	1.0E-05		5800	818.9	
						<b>TOTAL</b>	65.2		<b>9.2</b>	

<sup>a</sup> Additive cancer risk consumption limits and greatest non-cancer risk consumption limits indicated by black box.

**Appendix 1g : Doncaster consumption limit calculations<sup>a</sup>.**

Species	Compound	Contaminant Concentration (mg/kg wet weight)	Slope Factor (mg/kg/day) <sup>-1</sup>	Input Data/Assumptions			Daily Consumption Limits (g/day)		Monthly Fish Consumption Limits (meals/month)		
				Body weight (kg)	Reference Dose (mg/kg/day)	ARL	Cancer Risk	Non Cancer Risk	Cancer Risk	Non Cancer Risk	
Eel	p,p-DDT	2.70E-02	0.34	80	5.0E-04	1.0E-05	87.1	1481		209.1	
	p,p-DDD	6.02E-02	0.24	80	NA	1.0E-05	55.4				
	p,p-DDE	2.87E-01	0.34	80	NA	1.0E-05	8.2				
	Lindane	0.00E+00	1.30	80	3.0E-04	1.0E-05					
	Dieldrin	1.63E-02	16.00	80	5.0E-05	1.0E-05	3.1	245		34.6	
	Chlordanes (total)	1.06E-02	0.35	80	5.0E-04	1.0E-05	216.4	3786		534.6	
	HCB	4.35E-04	1.60	80	8.0E-04	1.0E-05	1148.1	146958		20747.0	
	PCBs (total)	6.62E-02	2.00	80	2.0E-05	1.0E-05	6.0	24		<b>3.4</b>	
	Cadmium	1.36E-03	NA	80	1.0E-03	1.0E-05		58846		8307.6	
	Mercury	1.52E-01	NA	80	1.0E-04	1.0E-05		52		7.4	
	Arsenic 10%	0.00E+00	1.50	80	3.0E-04	1.0E-05					
	Zinc	1.36E+01	NA	80	3.0E-01	1.0E-05		1765		249.2	
	Nickel	0.00E+00	NA	80	2.0E-02	1.0E-05					
	Chromium	0.00E+00	NA	80	3.0E-03	1.0E-05					
							<b>TOTAL</b>	1.5		<b>0.2</b>	
Flounder	p,p-DDT	1.77E-03	0.34	80	5.0E-04	1.0E-05	1330.1	22611		3192.2	
	p,p-DDD	1.31E-03	0.24	80	NA	1.0E-05	2541.2				
	p,p-DDE	1.07E-02	0.34	80	NA	1.0E-05	219.3				
	Lindane	0.00E+00	1.30	80	3.0E-04	1.0E-05					
	Dieldrin	5.73E-04	16.00	80	5.0E-05	1.0E-05	87.3	6980		985.5	
	Chlordanes (total)	3.69E-04	0.35	80	5.0E-04	1.0E-05	6190.8	108339		15294.9	
	HCB	2.61E-05	1.60	80	8.0E-04	1.0E-05	19131.1	2448785		345710.9	
	Cadmium	0.00E+00	NA	80	1.0E-03	1.0E-05					
	Mercury	9.57E-02	NA	80	1.0E-04	1.0E-05		84		<b>11.8</b>	
	Arsenic 10%	1.58E-02	1.50	80	3.0E-04	1.0E-05	33.8	1522		214.9	
	Zinc	6.82E+00	NA	80	3.0E-01	1.0E-05		3519		496.9	
	Nickel	0.00E+00	NA	80	2.0E-02	1.0E-05					
	Chromium	0.00E+00	NA	80	3.0E-03	1.0E-05					
							<b>TOTAL</b>	21.3		<b>3.0</b>	

<sup>a</sup> Additive cancer risk consumption limits and greatest non-cancer risk consumption limits indicated by black box.

**Appendix 1h: Washdyke Lagoon consumption limit calculations<sup>a</sup>.**

Species	Compound	Contaminant Concentration (mg/kg wet weight)	Input Data/Assumptions				Daily Consumption Limits (g/day)		Monthly Fish Consumption Limits (meals/month)	
			Slope Factor (mg/kg/day)-1	Body weight (kg)	Reference Dose (mg/kg/day)	ARL	Cancer Risk	Non Cancer Risk	Cancer Risk	Non Cancer Risk
<b>Eel</b>	p,p-DDT	6.95E-03	0.34	80	5.0E-04	1.0E-05	339	5755	812.4	
	p,p-DDD	2.25E-03	0.24	80	NA	1.0E-05	1481			
	p,p-DDE	9.68E-03	0.34	80	NA	1.0E-05	243			
	Lindane	0.00E+00	1.30	80	3.0E-04	1.0E-05				
	Dieldrin	2.46E-04	16.00	80	5.0E-05	1.0E-05	203	16229	2291.2	
	Chlordanes (total)	5.68E-04	0.35	80	5.0E-04	1.0E-05	4023	70401	9939.0	
	HCB	1.17E-05	1.60	80	8.0E-04	1.0E-05	42829	5482108	773944.7	
	PCBs (total)	3.42E-03	2.00	80	2.0E-05	1.0E-05	117	468	66.0	
	Cadmium	0.00E+00	NA	80	1.0E-03	1.0E-05				
	Mercury	2.33E-02	NA	80	1.0E-04	1.0E-05		343	<b>48.4</b>	
	Arsenic 10%	0.00E+00	1.50	80	3.0E-04	1.0E-05				
	Zinc	8.94E+00	NA	80	3.0E-01	1.0E-05		2685	379.0	
	Nickel	3.30E-02	NA	80	2.0E-02	1.0E-05		48426	6836.7	
	Chromium	5.83E-02	NA	80	3.0E-03	1.0E-05		4116	581.1	
					<b>TOTAL</b>	47		<b>6.6</b>		
<b>Flounder</b>	p,p-DDT	4.55E-03	0.34	80	5.0E-04	1.0E-05	517	8792	1241.2	
	p,p-DDD	2.61E-03	0.24	80	NA	1.0E-05	1276			
	p,p-DDE	2.78E-02	0.34	80	NA	1.0E-05	85			
	Lindane	0.00E+00	1.30	80	3.0E-04	1.0E-05				
	Dieldrin	5.23E-04	16.00	80	5.0E-05	1.0E-05	96	7646	1079.4	
	Chlordanes (total)	3.88E-04	0.35	80	5.0E-04	1.0E-05	5891	103099	14555.2	
	HCB	2.63E-05	1.60	80	8.0E-04	1.0E-05	19017	2434158	343645.9	
	Cadmium	0.00E+00	NA	80	1.0E-03	1.0E-05				
	Mercury	5.30E-02	NA	80	1.0E-04	1.0E-05		151	<b>21.3</b>	
	Arsenic 10%	5.56E-02	1.50	80	3.0E-04	1.0E-05	9.6	432	61.0	
	Zinc	7.32E+00	NA	80	3.0E-01	1.0E-05		3277	462.7	
	Nickel	0.00E+00	NA	80	2.0E-02	1.0E-05				
	Chromium	0.00E+00	NA	80	3.0E-03	1.0E-05				
						<b>TOTAL</b>	7.7		<b>1.1</b>	

<sup>a</sup> Additive cancer risk consumption limits and greatest non-cancer risk consumption limits indicated by black box.

**Appendix 1i: Waihi River consumption limit calculations<sup>a</sup>**

Species	Compound	Input Data/Assumptions				Daily Consumption Limits (g/day)		Monthly Fish Consumption Limits (meals/month)		
		Contaminant Concentration (mg/kg wet weight)	Slope Factor (mg/kg/day) <sup>-1</sup>	Body weight (kg)	Reference Dose (mg/kg/day)	ARL	Cancer Risk	Non Cancer Risk	Cancer Risk	Non Cancer Risk
Eel	p,p-DDT	3.27E-03	0.34	80	5.0E-04	1.0E-05	720	12236		1727.4
	p,p-DDD	1.97E-03	0.24	80	NA	1.0E-05	1689			
	p,p-DDE	5.44E-02	0.34	80	NA	1.0E-05	43			
	Lindane	0.00E+00	1.30	80	3.0E-04	1.0E-05				
	Dieldrin	6.05E-04	16.00	80	5.0E-05	1.0E-05	83	6612		933.4
	Chlordanes (total)	4.54E-04	0.35	80	5.0E-04	1.0E-05	5039	88181		12449.1
	HCB	2.18E-04	1.60	80	8.0E-04	1.0E-05	2296	293867		41487.1
	PCBs (total)	3.44E-03	2.00	80	2.0E-05	1.0E-05	116	464		65.6
	Cadmium	1.09E-03	NA	80	1.0E-03	1.0E-05		73672		10400.7
	Mercury	3.26E-01	NA	80	1.0E-04	1.0E-05		25		<b>3.5</b>
	Arsenic 10%	0.00E+00	1.50	80	3.0E-04	1.0E-05				
	Zinc	8.24E+00	NA	80	3.0E-01	1.0E-05		2913		411.3
	Nickel	0.00E+00	NA	80	2.0E-02	1.0E-05				
	Chromium	0.00E+00	NA	80	3.0E-03	1.0E-05				
						<b>TOTAL</b>	22			<b>3.0</b>
Trout	p,p-DDT	1.85E-04	0.34	80	5.0E-04	1.0E-05	12686	215661		30446.2
	p,p-DDD	7.43E-05	0.24	80	NA	1.0E-05	44839			
	p,p-DDE	1.76E-02	0.34	80	NA	1.0E-05	133			
	Lindane	0.00E+00	1.30	80	3.0E-04	1.0E-05				
	Dieldrin	7.97E-05	16.00	80	5.0E-05	1.0E-05	628	50219		7089.8
	Chlordanes (total)	1.89E-05	0.35	80	5.0E-04	1.0E-05	120901	2115775		298697.7
	HCB	1.73E-05	1.60	80	8.0E-04	1.0E-05	28966	3707684		523437.7
	Cadmium	5.63E-04	NA	80	1.0E-03	1.0E-05		142222		20078.4
	Mercury	1.35E-01	NA	80	1.0E-04	1.0E-05		59		<b>8.4</b>
	Arsenic 10%	0.00E+00	1.50	80	3.0E-04	1.0E-05				
	Zinc	3.94E+00	NA	80	3.0E-01	1.0E-05		6095		860.5
	Nickel	3.66E-02	NA	80	2.0E-02	1.0E-05		43761		6178.0
Chromium	0.00E+00	NA	80	3.0E-03	1.0E-05					
					<b>TOTAL</b>	108			<b>15.3</b>	
Watercress	Cadmium	8.21E-03	NA	80	1.0E-03	1.0E-05		9741		1674.7
	Mercury	0.00E+00	NA	80	1.0E-04	1.0E-05				
	Arsenic	9.48E-03	1.50	80	3.0E-04	1.0E-05	56	2533	9.7	<b>435.4</b>
	Zinc	2.46E+00	NA	80	3.0E-01	1.0E-05		9741		1674.7
	Nickel	3.98E-02	NA	80	2.0E-02	1.0E-05		40202		6911.5
	Chromium	4.49E-02	NA	80	3.0E-03	1.0E-05		5351		919.9

<sup>a</sup> Additive cancer risk consumption limits and greatest non-cancer risk consumption limits indicated by black box.



**Appendix 1j: Winchester consumption limit calculations<sup>a</sup>**

Species	Compound	Input Data/Assumptions				Daily Consumption Limits (g/day)		Monthly Fish Consumption Limits (meals/month)		
		Contaminant Concentration (mg/kg wet weight)	Slope Factor (mg/kg/day) <sup>-1</sup>	Body weight (kg)	Reference Dose (mg/kg/day)	ARL	Cancer Risk	Non Cancer Risk	Cancer Risk	Non Cancer Risk
Eel	p,p-DDT	1.52E-02	0.34	80	5.0E-04	1.0E-05	154	2624		370.5
	p,p-DDD	6.40E-03	0.24	80	NA	1.0E-05	521			
	p,p-DDE	1.91E-01	0.34	80	NA	1.0E-05	12			
	Lindane	0.00E+00	1.30	80	3.0E-04	1.0E-05				
	Dieldrin	2.21E-03	16.00	80	5.0E-05	1.0E-05	23	1813		256.0
	Chlordanes (total)	1.48E-03	0.35	80	5.0E-04	1.0E-05	1540	26943		3803.7
	HCB	2.37E-04	1.60	80	8.0E-04	1.0E-05	2111	270150		38138.9
	PCBs (total)	2.67E-02	2.00	80	2.0E-05	1.0E-05	15	60		8.5
	Cadmium	2.58E-03	NA	80	1.0E-03	1.0E-05		30988		4374.8
	Mercury	5.56E-01	NA	80	1.0E-04	1.0E-05		14		<b>2.0</b>
	Arsenic 10%	0.00E+00	1.50	80	3.0E-04	1.0E-05				
	Zinc	1.39E+01	NA	80	3.0E-01	1.0E-05		1726		243.7
	Nickel	0.00E+00	NA	80	2.0E-02	1.0E-05				
	Chromium	0.00E+00	NA	80	3.0E-03	1.0E-05				
					<b>TOTAL</b>	5			<b>0.7</b>	
Trout	p,p-DDT	2.97E-04	0.34	80	5.0E-04	1.0E-05	7919	134626		19006.1
	p,p-DDD	1.49E-04	0.24	80	NA	1.0E-05	22385			
	p,p-DDE	1.65E-02	0.34	80	NA	1.0E-05	142			
	Lindane	0.00E+00	1.30	80	3.0E-04	1.0E-05				
	Dieldrin	6.52E-04	16.00	80	5.0E-05	1.0E-05	77	6133		865.8
	Chlordanes (total)	5.87E-05	0.35	80	5.0E-04	1.0E-05	38962	681838		96259.5
	HCB	1.74E-05	1.60	80	8.0E-04	1.0E-05	28656	3667967		517830.7
	Cadmium	0.00E+00	NA	80	1.0E-03	1.0E-05				
	Mercury	7.93E-02	NA	80	1.0E-04	1.0E-05		101		<b>14.2</b>
	Arsenic 10%	7.13E-03	1.50	80	3.0E-04	1.0E-05	74.8	3364		474.9
	Zinc	4.23E+00	NA	80	3.0E-01	1.0E-05		5677		801.4
Nickel	0.00E+00	NA	80	2.0E-02	1.0E-05					
Chromium	0.00E+00	NA	80	3.0E-03	1.0E-05					
					<b>TOTAL</b>	30			<b>4.2</b>	
Watercress	Cadmium	7.65E-03	NA	80	1.0E-03	1.0E-05		10457		1797.8
	Mercury	0.00E+00	NA	80	1.0E-04	1.0E-05				
	Arsenic	0.00E+00	1.50	80	3.0E-04	1.0E-05				
	Zinc	4.10E+00	NA	80	3.0E-01	1.0E-05		5849		<b>1005.5</b>
	Nickel	1.53E-02	NA	80	2.0E-02	1.0E-05		104570		17977.7
	Chromium	2.78E-02	NA	80	3.0E-03	1.0E-05		8627		1483.2

<sup>a</sup> Additive cancer risk consumption limits and greatest non-cancer risk consumption limits indicated by black box.

Appendix 1k: Te Nga Wai consumption limit calculations<sup>a</sup>.

Species	Compound	Input Data/Assumptions					Daily Consumption Limits (g/day)		Monthly Fish Consumption Limits (meals/month)	
		Contaminant Concentration (mg/kg wet weight)	Slope Factor (mg/kg/day)-1	Body weight (kg)	Reference Dose (mg/kg/day)	ARL	Cancer Risk	Non Cancer Risk	Cancer Risk	Non Cancer Risk
Eel	p,p-DDT	8.23E-04	0.34	80	5.0E-04	1.0E-05	2857.4	48575	6857.7	
	p,p-DDD	3.43E-04	0.24	80	NA	1.0E-05	9732.2			
	p,p-DDE	1.46E-02	0.34	80	NA	1.0E-05	161.6			
	Lindane	0.00E+00	1.30	80	3.0E-04	1.0E-05				
	Dieldrin	1.88E-04	16.00	80	5.0E-05	1.0E-05	265.6	21248		2999.7
	Chlordanes (total)	7.43E-05	0.35	80	5.0E-04	1.0E-05	30745.5	538046		75959.4
	HCB	6.96E-05	1.60	80	8.0E-04	1.0E-05	7182.9	919417		129800.0
	PCBs (total)	7.56E-04	2.00	80	2.0E-05	1.0E-05	529.1	2117		298.8
	Cadmium	0.00E+00	NA	80	1.0E-03	1.0E-05				
	Mercury	3.62E-01	NA	80	1.0E-04	1.0E-05		22		<b>3.1</b>
	Arsenic 10%	0.00E+00	1.50	80	3.0E-04	1.0E-05				
	Zinc	1.21E+01	NA	80	3.0E-01	1.0E-05		1990		280.9
	Nickel	3.62E-02	NA	80	2.0E-02	1.0E-05		44219		6242.7
	Chromium	0.00E+00	NA	80	3.0E-03	1.0E-05				
						<b>TOTAL</b>	80.2		<b>11.3</b>	
Watercress	Cadmium	1.04E-02	NA	80	1.0E-03	1.0E-05		7694		1322.7
	Mercury	0.00E+00	NA	80	1.0E-04	1.0E-05				
	Arsenic	0.00E+00	1.50	80	3.0E-04	1.0E-05				
	Zinc	5.20E+00	NA	80	3.0E-01	1.0E-05		4616		<b>793.6</b>
	Nickel	8.91E-02	NA	80	2.0E-02	1.0E-05		17952		3086.4
	Chromium	1.26E-02	NA	80	3.0E-03	1.0E-05		19009		3267.9

<sup>a</sup> Additive cancer risk consumption limits and greatest non-cancer risk consumption limits indicated by black box.