



Mana Kai Rangahau

Scombrototoxin in seafood

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1 *Scombrototoxin*

Histamine (scombroid) seafood poisoning can result from eating spoiled fish, usually, although not exclusively, of the families Scombridae and Scomberesocidae, which include mackerel and tuna. In New Zealand, this type of food poisoning is most commonly reported from kahawai, which is in the Arripidae family. These fish species are more frequently associated with scombroid fish poisoning than others because their flesh contains higher levels of the naturally occurring amino acid, histidine. The conversion of histidine to histamine by bacteria and the subsequent consumption of fish containing high histamine levels causes the illness. 'Temperature abuse' of fish is generally required before significant histamine levels develop in fish. Once produced, histamine is heat stable so processing such as hot-smoking, cooking or even canning will not remove it from the flesh.

2 *The disease - scombroid poisoning (histamine poisoning)*

Scombroid poisoning (histamine poisoning) is caused by the ingestion of foods that contain high levels of histamine and possibly other vasoactive amines and compounds. Symptoms mimic an acute IgE-mediated food allergy. Early symptoms may include a tingling or burning sensation in the mouth, a rash on the upper body and a drop in blood pressure. Headaches and itchy skin are common. The symptoms may progress to nausea, vomiting, and diarrhea. Sufferers may require hospitalisation, particularly the elderly or those with existing health conditions.

Symptoms occur very rapidly, from immediately to 30 minutes after eating contaminated fish. They usually subside between 3 and 24 hours after consumption, but may last several days.

While rarely fatal, the condition can be very distressing, with individuals often requiring medical consultations and occasionally hospitalisation.

Symptoms vary between individuals exposed to the same dose of histamine in contaminated fish. Some individuals react to relatively low levels of histamine, while in others high doses fail to provoke symptoms. Given the magnitude of under-reporting of gastrointestinal illness experienced in the community, and the fact that many individuals do not consult a doctor due to the self-limiting nature of the illness, many cases are probably never reported to the public health authorities.

3 *Incidence in New Zealand*

In New Zealand most occurrences of scombroid poisoning result from eating contaminated smoked fish. Hot smoked kahawai (*Arripis trutta*) is frequently implicated (Table 1). This species has also been implicated in outbreaks of scombroid fish poisoning in Australia where it is known as Australian salmon.

Table 1. Auckland scombroid outbreaks linked to hot-smoked kahawai 2001-02.*

Date of outbreak	Number of cases	Outbreak status	Number of fish linked samples	Histamine level in kahawai (mg/kg)
31/8/01	2	Probable	None	-
03/01/02	5	Probable	None	-
03/01/02	5	Confirmed	2	190 - 985
13/05/02	2	Confirmed	2	1700 - 2100
6/12/02 to 8/12/02	4	Probable	None	-
5/12/02 to 8/12/02	16	Confirmed	9	200 - 3940

* for the greater Auckland region and reported to Auckland Regional Public Health

4 *Associated foods*

Fishery products that have been implicated in scombroid poisoning in New Zealand include tuna, kahawai, kingfish, mackerel, mullet and trevally. Other foods, such as cheese and wine, have also had toxic effects. The primary cheese involved has been Swiss cheese.

5 *Conditions for histamine production*

Histamine and other amines are formed during the growth of certain bacteria that produce decarboxylase enzymes. Histidine decarboxylating bacteria can be present in the normal microflora of live fish. However, most of the bacteria that cause histamine spoilage result from contamination on fishing vessels or in processing plants.

Because most of the common histamine producing bacteria are mesophiles it is unlikely that high levels of histamine will occur unless the fish are 'temperature abused'. In experiments carried out by Crop & Food Research scientists, a New Zealand species known to cause histamine poisoning (kahawai) was held at different temperatures to determine the level of 'temperature abuse' necessary to cause high histamine concentrations.

Although elevated levels of histamine (>200 mg/kg) were induced in kahawai stored at temperatures between 20 and 35°C, they only occurred after more than 15 hours at these temperatures. At 15°C it took more than 24 hours for histamine levels to exceed 100 mg/kg. Further, elevated histamine levels only occurred after the bacteria had entered the stationary growth phase with total bacterial counts at 35°C exceeding 10⁶ cfu/g. From these trials it appeared that fresh kahawai only become a hazard when subjected to very poor handling procedures.

In general, while histamine producing bacteria are capable of growing over a wide temperature range, their rate of growth and subsequent histamine production is faster at temperatures greater than 20°C than at temperatures around 7°C. One scientific paper reports that the optimum temperature for the development of histamine by toxigenic bacteria, particularly *Morganella morganii*, is around 25°C, with histamine production being halved at 15°C and ceasing at 4°C. Therefore, while there is the possibility of histamine being produced under conditions of moderate temperature abuse, elevated histamine concentrations in fish are more usually the result of high temperatures rather than exposure to relatively low temperatures over sustained periods.

The enzyme histidine decarboxylase can be active at refrigeration temperatures, so even if the bacteria are not active the enzyme can continue to convert histidine to histamine. The enzyme is likely to remain stable in the frozen state and to be reactivated very rapidly after thawing. Both the enzyme and the bacteria that produce it can be inactivated by cooking. After cooking, recontamination of the fish with histamine-forming bacteria must occur before more histamine is formed. However, it is temperature abuse of raw, unfrozen fish that has been implicated in most reports of scombroid poisoning.

6 *Histamine in fish*

Most of the bacteria that subsequently cause histamine spoilage problems seem to be due to post-catch contamination of the fish, from fishing vessels or processing plants. The types of bacteria associated with histamine development are also common in the marine environment and can naturally occur in the gills, skin and within the gut of marine fish. On death, the natural defence mechanisms of the fish can no longer inhibit bacterial growth and this, coupled with an increase in temperature, can lead to a rapid increase in bacteria numbers. During filleting, the bacteria can spread to the flesh of the fish.

Some harvesting practices, such as long-lining or set netting, can result in the death of the fish before it is removed from the water. Therefore, histamine formation may be underway before the fish is harvested and the warmer the water, the faster this process happens. Contamination can be a problem when set-netting in warm tidal areas where seawater temperatures increase as the tide goes out. In extreme cases the fish may be exposed to ambient air temperatures.

Once formed, histamine concentrations in fish muscles can vary, depending on a combination of factors including time, elevated temperature and the type and number of bacteria that are present. Distribution of the toxin within an individual fish fillet or between cans in a case lot can be uneven, with some sections of a product causing illness and others not. Neither cooking, canning, nor freezing will eliminate the toxin.

Although sensory or 'organoleptic' evaluations (sight and smell) can provide a reasonable indication of possible temperature abuse, they can be poor indicators of the presence of histamine in fish as under some conditions high concentrations of histamine may occur in fish that appear to be unspoiled.

Chemical testing for histamine is the only reliable test for the hazard in a product, although bacterial counts can also give a good indication that temperature abuse is likely to have occurred.

7 *Detection*

While sensory evaluation is generally used to screen fish for spoilage, fish exposed to high temperature spoilage do not always produce the expected spoilage profile; histamine may form before indicators of spoilage such as odour and appearance changes are evident. Therefore, sensory examination alone is an ineffective indicator of the presence of the scombrotoxin histamine.

While chemical testing is required and is an effective means of detecting the presence of histamine in fish, the distribution of histamine within a fish and between fish within a catch can be quite variable. The amount of sampling required to accommodate such variability is large. For this reason chemical testing alone will not normally provide adequate assurance that the hazard has been controlled. Rather, the hazard is better managed through the adoption of a HACCP plan which restricts the time the product is at temperatures that allow bacterial growth.

Rapid test kits are available for histamine and a list of suppliers of commercial test products can be found at <http://seafood.ucdavis.edu/haccp/compendium/Chapt27.htm>

8 *Food safety regulations*

New Zealand: The Australian New Zealand Food Standards Code – Volume 2, Standard 2.2.3 (Page 22301, September 2002) states “the level of histamine in fish or fish products must not exceed 200 mg/kg”.

International: The USFDA considers that as histamine levels within a fish can vary dramatically, two action levels are required. A concentration of 50 mg/kg is considered to be an indicator of decomposition, which suggests

that histamine concentrations may be higher in other fish or in different parts of the tested product. A concentration of 200 mg/kg is considered a hazardous level.

The Codex Alimentarius Standards for fish provide histamine levels as indicators for:

1. decomposition
2. hygiene and handling

For decomposition, the relevant standards state”:

‘The products shall not contain more than 10 mg/100 g of histamine based on the average of the sample unit tested. This applies only to species of *Clupeidae*, *Scrombridae*, *Scrombresocidae*, *Pomatomidae* and *Coryphaenidae* families.’

For hygiene and handling, the relevant standards state:

‘No sample unit shall contain histamine that exceeds 20 mg per 100 g. This applies only to species of the families Scrombridae, Clupeidae, Coryphaenidae, Scrombresocidae and Pomatomidae .’

These Codex Standards cover:

- (a) quick frozen fish fillets,
- (b) quick frozen blocks of fish fillet, minced fish flesh and mixtures of fillets and minced fish flesh,
- (c) eviscerated and uneviscerated quick frozen finfish,
- (d) quick frozen fish sticks (fish fingers), fish portions and fish fillets - breaded or battered,
- (e) canned sardines and sardine type products, and
- (f) canned tuna and bonito.

9 Control procedures

Control of scombrototoxin formation is dependent on the development of an effective HACCP plan that incorporates the following elements.

Rapid chilling: Rapid chilling of fish immediately after death is the most important step in the prevention of scombrototoxin, especially for fish that are exposed to warmer water or air, or harvested some time after capture.

Rapid chilling to 4°C or less prevents bacterial growth and the formation of the histidine decarboxylase enzyme. Holding fish on ice (0°C) is required to prevent the longer term, low temperature development of histamine.

Internationally recognised controls require that for susceptible species:

- the fish internal temperature be brought to 10°C or less within 6 hours of death,

- the core temperature of the fish be brought to 4°C within 24 hours of death,
- chilled fish should not be exposed to temperatures above 4°C for more than 4 hours cumulatively after the initial chilling,
- chilled fish should not be stored for more than 14 days at 0°C or more than 7 days at 4°C before smoking,
- frozen fish should not be exposed to temperatures above 4°C for more than 12 hours cumulatively or 6 hours uninterrupted.

The New Zealand Fishing Industry Agreed Implementation Standards (IAIS 3.6 section 2.2) require that chilled fish are stored and transported at -1 to +1°C.

The time/temperature regime to which the fish are exposed from capture until the internal temperature of the fish reaches 4°C or less is dependent on a number of factors, including the harvest method. In species that are recognised as being especially at risk of causing scombroid poisoning, techniques such as long-lining or set netting that can result in the fish remaining in the water for a long time after death should be avoided, especially in areas or seasons when the water temperature is high. Care should also be taken to ensure that the quantity of fish taken, either by long-line, purse seine or trawl, does not exceed the vessel's ability to rapidly chill the product.

Critical Control Points: Within a HACCP plan the following steps are likely to be identified as critical control points.

1. Receiving:
 - a. All lots accompanied by documentation that show the fish has been chilled to 4°C or less with 12 hours of death
 - b. Sensory examination of incoming fish for signs of decomposition
 - c. Fish arrive at factory at 4°C or less
 - d. Histamine concentration of representative fish periodically determined
2. Processing:
 - a. Controlling temperature of plant
3. Distribution
 - a. Setting time/temperature
 - b. Setting a shelf-life
 - c. Having adequate traceability and recall procedures

For companies that produce hot smoked or cooked seafood, additional critical control points will include:

1. Brining
 - a. Time/temperature control

2. Hot smoking
 - a. Time/temperature to eliminate the causative bacteria
3. Packaging
 - a. Avoiding contamination of the cooked product

10 *Acknowledgements*

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A web link for additional information on Hazards and Controls Guidance for Scombrototoxin is <http://www.cfsan.fda.gov/~comm/haccp4g.html>.

Guidelines for the safe preparation of hot smoked seafood in New Zealand are available through this link <http://www.crop.cri.nz/psp/seafood-pathogens/index.htm>.

11 *Web links for additional information on scombrototoxin*

US/FDA Bad Bug Book <http://www.cfsan.fda.gov/~mow/chap38.html>

12 *References for additional information on scombrototoxin*

Bartholomew, B.A.; Berry, P.R.; Rodhouse, J.C.; Gilbert, R.J. 1987: Scombrototoxic fish poisoning in Britain: features of over 250 suspected incidents from 1976 to 1986. *Epidemiology and Infection* 99: 775-782.

Becker, K.; Southwick, K.; Reardon, J.; Berg, R.; MacCormack, J. 2001: Histamine poisoning associated with eating tuna burgers. *Journal of American Medical Association* 285: 1327-1330.

Bryan, M. 1988: Risks of practices, procedures and processes that lead to outbreaks of foodborne diseases. *Journal of Food Protection* 51: 663-678.

Chen, K.; Malison, M. 1987: Outbreak of scombroid fish poisoning. *American Journal of Public Health* 77: 1335-1336.

Etkind, P.; Wilson, M.; Gallagher, K.; Courmoyer, J. 1987: Bluefish-associated scombroid poisoning and example of the expanding spectrum of

food poisoning from seafood. *Journal of the American Medical Association* 258: 3409-3410.

Fletcher, G.; Summers, G.; van Veghel, P.W.C. 1998: Levels of histamine and histamine-producing bacteria in smoked fish from New Zealand markets. *Journal of Food Protection* 61: 1064-1070.

Fletcher, G.C.; Summers, G.; Winchester, R.V.; Wong, R. 1995: Histamine and histidine in New Zealand marine fish and shellfish species, particularly kahawai (*Arripis trutta*). *Journal of Aquatic Food Product Technology* 4: 54-74.

Foo, L. 1975: Scombroid-type poisoning induced by ingestion of smoked kahawai. *New Zealand Medical Journal*: 476-477.

Frank, H.A.; Yoshinaga, D.H.; Nip, W.K. 1981: Histamine formation and honey combing during decomposition of skipjack tuna, *Katsuwonus pelamis*, at elevated temperature. *Marine Fish Reviews* 14: 9-14.

Gilbert, R.; Hobbs, G.; Murray, C.; Cruikshank, J.; Young, S. 1980: Scombrototoxic fish poisoning: features of the first 50 incidents to be reported in Britain (1976-9). *British Medical Journal* 281: 71-72.

Huss, H. 1991: Assurance of seafood quality. 334. Rome, *Fisheries technical paper*.

Inenistea, C. 1971: Bacterial production and destruction of histamine in foods, and food poisoning caused by histamine. *Die Nahrung* 15: 109-113.

Jorgensen, L.V.; Huss, H.H.; Dalgaard, P. 2000: The effect of biogenic amine production by single bacterial cultures and metabiosis on cold-smoked salmon. *Journal of Applied Microbiology* 89: 920-934.

Kim, S.H.; Field, K.; Chang, D.S.; Wei, C.I.; An, H. 2001: 2001. Identification of bacteria crucial to histamine accumulation in Pacific Mackerel during storage. *Journal of Food Protection* 64: 1556-1564.

Klausen, N.K.; Huss, H.H. 1987: Growth and histamine production by *Morganella morganii* under various temperature conditions. *International Journal of Food Microbiology* 5: 147-156.

Lehane, L.; Olley, J. 2000: Histamine fish poisoning revisited. *International Journal of Food Microbiology* 58: 1-37.

Leрке, P.; Werner, S.; Taylor, S.; Guthertz, L. 1978: Scombroid poisoning - report of an outbreak. *Western Journal of Medicine* 129: 381-386.

Lieber, E.; Taylor, S. 1978: Thin layer chromatographic screening methods for histamine in tuna fish. *Journal of Chromatography* 153: 143-152.

Lopez-Sabater, E.I.; Rodriguez-Jerez, J.J.; Hernandez-Herrero, M.; Roig-Sagues, A.X.; Mora-Ventura, M.T. 1996: Sensory quality and histamine formation during controlled decomposition of tuna (*Thunnus thynnus*). *Journal of Food Protection* 59.

Mitchell, J. 1984: Scombroid food poisoning from fish. *New Zealand Medical Journal* 97: 127.

- Morrow, J.D.; Margolies, G.R.; Rowland, J.; Roberts, L.J. 1991: Evidence that histamine is the causative agent of scombroid-fish poisoning. *The New England Journal of Medicine* 324: 716-720.
- Muller, G.; Lamprecht, J.; Barnes, J.; de Villiers, R.; Honeth, B.; Hoffman, B. 1992: Scombroid poisoning. Case series of 10 incidents involving 22 patients. *South African Medical Journal* 81: 427-430.
- Osborne, C.M.; Bremer, P.J. 2000: Application of the bigelow (z-value) model and histamine detection to determine the time and temperature required to eliminate *Morganella morganii* from seafood. *Journal of Food Protection* 63: 277-280.
- Rawles, D.; Flick, G. 1996: Biogenic amines in fish and shellfish. *Advances in Food and Nutrition Research* 39: 329-365.
- Rogers, P.; Staruszkiewicz, W. 2000: Histamine test kit comparison. *Journal of Aquatic Food Product Technology* 9: 5-17.
- Simmons, G.; Pitches, S.; Hicking, J. 2003: An outbreak of scombroid poisoning associated with consumption of smoked kahawai, December, 2002. Auckland, Public Health Protection, Auckland District Health Board. 30 p.
- Smart, D. 1992: Scombroid poisoning. A report of seven cases involving the Western Australian, *Arripis truttaceus*. *Medical Journal of Australia* 157.
- Summers, G. 2002: Investigation into aspects of hot smoked Kahawai processing. Auckland, New Zealand Institute for Crop & Food Research Ltd.
- Taylor, S.; Stratton, J.; Nordlee, J. 1989: Histamine poisoning (scombroid fish poisoning): an allergic-like intoxication. *Journal of Toxicology - Clinical Toxicology* 27: 225-240.
- Taylor, S.L. 1986: Histamine food poisoning: toxicology and clinical aspects. *CRC Critical Reviews in Toxicology* 17.
- Thornton, R.; Calder, L.; Baker, M. 1993: Scombroid fish poisoning in Auckland. *Communicable Diseases New Zealand* 93: 14-16.
- Wheeler, J.; Sethi, D.; Cowden, J.; Wall, P.; Rodrigues, L.; Tompkins, D.; Hudson, M.; Roderick, P. 1999: Study of infectious intestinal disease in England: rates in the community, presenting to general practice, and reported to national surveillance. *British Medical Journal* 318: 1046-1050.