



## RISK PROFILES OF FOOD-BORNE PATHOGENS ANISAKID NEMATODES

*Anisakid nematodes are parasitic roundworms found in fish and seafood, increasingly recognized as a cause of foodborne illness. Human exposure occurs through the consumption of raw or insufficiently processed fish, fish products, and cephalopods (mainly squid), sharing or crossing the habitat of the end hosts (marine mammals). Ingestion of viable larvae can lead to acute, non-specific gastrointestinal symptoms and exposure to parasite proteins has been linked to allergic reactions and chronic conditions. Routine food processing methods do not consistently control anisakid nematodes, and specific control steps such as freezing or heat treatment are required to ensure consumer safety.*



### Hazard Identification

**Family and species:** Within the family of Anisakidae, human cases have been most frequently reported in association with *Anisakis* spp., and less frequently with *Pseudoterranova* spp. and *Contracaecum* spp. However, genotyping is rarely performed, and reliable identification of L3-stage larvae within the Anisakidae family is difficult. Mis- or underreporting is likely to occur [1]. Human infections are mainly associated with the ingestion of third-stage larvae (L3) through the consumption of raw or lightly processed fish and cephalopods (mainly squid), which share or cross the habitat of the end hosts (marine mammals). Consumed fish from aquaculture that are not protected against first-stage larvae (L1), or that are fed L1-contaminated feed, may also pose a risk to humans. In addition to the larval stage, anisakid nematodes contain several inherent allergenic proteins [2]. The larvae are typically around 2 cm in length and are easily detected in the visceral cavity but are more difficult to detect in the musculature and belly flaps of white fish [3]. Key properties of anisakid nematodes relevant to food safety are summarized in Table 1.

Table 1: Characteristics of Anisakidae

<b>Infective stage of relevance</b>	Third-stage larvae (L3)
<b>Environmental persistence</b>	<i>Anisakis</i> larvae are highly resilient marine parasites that persist in fish, squid, and crustaceans, surviving cold temperatures, mild curing, and acidic environments for extended periods.
<b>Morphology</b>	Macroscopic nematode larvae, typically around 2cm in length.
<b>Occurrence in ordinary host</b>	Primarily located in the visceral cavity, including the peritoneum, mesenteries, and on the surface and wall of the gastrointestinal tract of fish and seafood [3].
<b>Replication</b>	Replication in intermediate and end hosts. No replication in food or in humans.
<b>Allergenic properties</b>	More than 28 <i>Anisakis</i> allergens have been identified so far; allergens can persist after larval death; freezing and heat treatment do not eliminate allergenic potential [2].

General characteristics of the parasite	
<b>Family/Genus</b>	Anisakidae / <i>Anisakis</i> spp., <i>Pseudoterranova</i> spp., <i>Contracaecum</i> spp.
<b>End host</b>	Marine mammals
<b>Intermediate hosts</b>	Zooplankton (krill), fish, and cephalopods
<b>Role of humans</b>	Humans are accidental hosts



Image 1: *Anisakis* larva.

Source: [https://commons.wikimedia.org/wiki/File:Anisakis\\_\(YPM\\_IZ\\_095329\).jpeg](https://commons.wikimedia.org/wiki/File:Anisakis_(YPM_IZ_095329).jpeg) ; Photo by Daniel J. Drew

**Lifecycle:** Anisakid nematodes (Figure 1) complete their life cycle in marine mammals, which act as definitive hosts and shed parasite eggs into the marine environment via faeces [4]. After hatching, larvae are taken up by zooplankton, particularly krill, which serve as key intermediate hosts. When infected krill are consumed by fish or squid, the parasites develop into infective third-stage larvae (L3) that encyst in the viscera and occasionally the musculature of these paratenic hosts. The life cycle is completed when infected fish or squid are eaten by marine mammals, where larvae mature to adults. Humans are accidental hosts and acquire infection through the consumption of raw or insufficiently cooked fish or cephalopods containing viable L3 larvae.

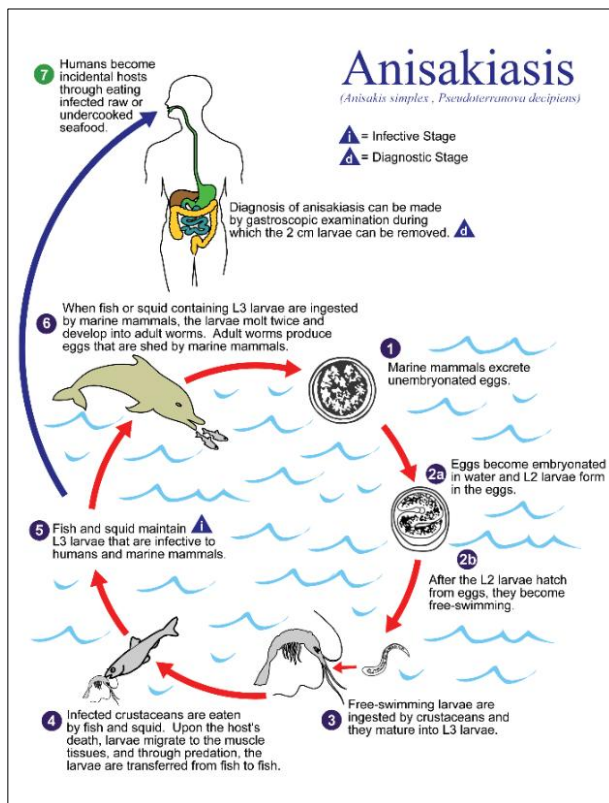


Figure 1: Lifecycle of Anisakidae. Source : CDC/ Alexander J. da Silva, PhD; Melanie Moser

**Inactivation:** Reliable inactivation of *Anisakis* larvae requires defined physical treatments (Table 2). Freezing at  $-20\text{ }^{\circ}\text{C}$  for at least 24h, heating to  $\geq 60\text{ }^{\circ}\text{C}$  for at least 1 minute, as well as high-pressure processing, irradiation, and hot smoking are effective when properly applied. In contrast, salting, marinating, or cold smoking may reduce larval viability but do not ensure complete inactivation.

Table 2: Inactivation of Anisakidae

Inactivation of larvae	
Parameter	Conditions
Freezing	$-20\text{ }^{\circ}\text{C}$ for at least 24h or $-35\text{ }^{\circ}\text{C}$ for 15h [5], [6].
Heating	$60\text{ }^{\circ}\text{C}$ for at least 1 minute [5], [6].
Salt & acid	Vinegar and salt can reduce the hazard associated with <i>A. simplex</i> , but they do not eliminate the parasite nor reduce the risk to an acceptable level [3].
Irradiation	Fish and shellfish at doses up to 3 kGy [3].
High pressure	A pressure of 200 MPa for 10 minutes at $0-15\text{ }^{\circ}\text{C}$ kills <i>A. simplex</i> larvae, as well as pressures down to 140 MPa when the treatment time is increased up to 60 minutes [3].
Smoking	Only hot smoking ( $>60\text{ }^{\circ}\text{C}$ ) is effective. Cold smoking does not inactivate anisakid nematodes [3].
Low voltage current	Not systematically evaluated, but Pulsed Electric Fields have proven to be effective in inactivating <i>Anisakis</i> in fish fillets [3], [7].



## Hazard Characterisation

**Disease characteristics:** Anisakid nematodes can cause clinical anisakiasis in humans, although not every exposure necessarily causes disease. Symptoms occur within hours to 7 days after ingestion and are often non-specific [8]. They range from mild, self-limiting gastrointestinal symptoms to severe forms mimicking acute abdomen with intestinal obstruction or gastro-allergic reactions, following the migration of larvae through the gastrointestinal wall.

Four principal clinical syndromes associated with anisakiasis have been described: gastric, intestinal, ectopic (or extra-gastrointestinal), and allergic. Gastric manifestations account for more than 95% of anisakiasis cases and are primarily driven by the proteolytic activity of the larvae shortly after ingestion. The resulting acute phase is

characterized by severe epigastric pain, often accompanied by vomiting, diarrhoea, and mild fever, with symptoms typically resolving within several days. Intestinal anisakiasis typically presents with persistent or episodic abdominal pain, abdominal tenderness, and diarrhoea. In rare cases, severe complications such as intestinal perforation, peritonitis, intussusception, or pneumoperitoneum have been reported. Allergic reactions are triggered by thermostable *Anisakis* proteins, such as Ani s 1 and Ani s 7, which retain their biological activity despite standard freezing or cooking. These allergens can elicit IgE-mediated hypersensitivity reactions, ranging from urticaria and oedema to severe anaphylaxis. In addition, sensitization has been documented in individuals without clinical symptoms, adding complexity to the characterization of the associated health risk [2]. The ingestion of a single viable larva may be sufficient to cause disease [8]. A summary of disease characteristics is presented in Table 3.

Andreu-Ballester et al. (2024) reported an association between *Anisakis* exposure and colon cancer based on the presence of anti-*Anisakis* antibodies [9]. Similarly, a Norwegian study observed a potential association between *Anisakis* exposure and altered immune responses in patients with inflammatory bowel disease compared to healthy individuals [10]. However, both studies were observational in nature and therefore demonstrate correlation rather than causation. Establishing a causal relationship would require experimental studies to determine whether *Anisakis* infection can directly contribute to colon carcinogenesis or immune dysregulation.

Table 3: Disease characteristics of anisakiasis

Disease Characteristics	
<b>Incubation period</b>	Typically, within 24 hours after ingestion, but it can take up to 7 days [8].
<b>Pathogenesis</b>	Clinical manifestations follow the migration of larvae through the gastrointestinal wall, while the allergic reaction is associated with the exposure to several proteins of the larvae.
<b>Symptoms</b>	Gastric pain, vomiting, nausea, fever, diarrhoea, allergic reactions [11].
<b>Progression</b>	Typically, self-limiting within a few days. Can progress into severe conditions in rare cases.
<b>Infectious period (shedding)</b>	No replication in humans.
<b>Vulnerable populations</b>	Pre-sensitised individuals to allergic proteins of anisakid nematodes [12].
<b>Minimal infective dose</b>	One larva can cause clinical symptoms in humans [8].

**Occurrence:** Anisakiasis is considered an emerging foodborne disease concern [2]. Globally, anisakiasis was estimated at approximately 20,000 reported cases prior to 2010, with over 90% occurring in Japan, reflecting traditional consumption of raw fish [3]. In Western countries, cases are on the rise and linked to increased consumption of raw/minimally processed seafood (e.g., sushi) and improved clinical recognition [10].

Epidemiological data on anisakiasis in Europe are inconsistent and often incomplete, as the reported number of new cases per year varies widely depending on whether figures are based on surveillance data, modelling approaches or histologically confirmed diagnoses. Across Europe, the estimated average burden corresponds to approximately 0.32 new cases per 100,000 people per year, which is substantially lower than in Japan, where around 20,000 new cases are reported annually. However, marked national differences exist: in Spain, estimates suggest between 3.87 and 19.3 new cases per 100,000 inhabitants per year, while a quantitative risk assessment focusing on anchovy consumption indicated that approximately 7,770–8,320 cases per year in Spain may require medical attention [8], [13]. No data on the epidemiological situation in Switzerland exist (Table 4).

Table 4: Epidemiological situation in Switzerland

Epidemiological Situation in Switzerland	
<b>Annual reported cases</b>	No documented cases of illness in Switzerland.
<b>Mandatory reportable disease</b>	Not listed as a notifiable disease.



## Exposure Assessment

**Sources and reservoirs:** Anisakiasis cases worldwide have increased over the past few years [8]. The main sources of infection are marine fish (e.g. salmon, hake, anchovy, herring, cod, pollock, mackerel) and cephalopods (e.g. squid and octopodes) [3]. Among the 15 most frequently consumed fish species in France, anisakid parasites have been detected in 66% of samples. This high prevalence provides important context for assessing the likelihood of exposure [14]. A further meta-analysis investigating fish on the Belgian market found mean prevalences of 33% and 5% for Cod and Atlantic salmon, respectively. Fish caught in the Northeast Atlantic had the highest rate of infection (68%). Furthermore, higher prevalences were found when looking at the viscera (mean prevalence 59%) compared to the muscle (29%) and with superior techniques such as enzymatic digestion or UV press (46%) compared to candling, the routine method (23%). Farmed fish were found to be the least infected (2%) [15]. Globally, prevalences ranging from 0–

100% were found in a systematic review [16]. An overview of the main sources and transmission routes is provided in Table 5.

Notifications concerning *Anisakis* spp. in the Rapid Alert System for Food and Feed (RASFF) between 2001–2023 show a total of 46 notifications recorded between 2021 and 2023 (21 in 2021, 7 in 2022, and 18 in 2023) [17].

**Outbreaks:** Documented outbreaks of anisakiasis appear to be rare in Switzerland. No outbreaks were reported between 2007–2021 [18]. As the disease is not notifiable and self-limiting in most cases, anisakiasis is likely to be underreported. In 2025, however, one clinical case of imported anisakiasis was published in Switzerland [19].

and may fail to detect larvae embedded in the musculature. Consequently, routine inspection alone cannot reliably prevent consumer exposure, and preventive measures such as freezing or adequate heat treatment are essential for risk management [3].

Table 2: Main sources and transmission routes in Switzerland.

<b>Main Sources and Transmission Routes in Switzerland</b>	
<b>Main reservoir</b>	Marine fish (e.g. salmon, hake, anchovy, herring, cod, pollock, mackerel) and cephalopods (e.g. squid and octopodes).
<b>Prevalence</b>	Fish on the Belgian market: 5% - 68% [15]. Globally, prevalences from 0-100% were found.
<b>Transmission</b>	Foodborne, through consumption of raw or undercooked fish and cephalopods.
<b>High risk food items</b>	Raw or mildly heated marine fish and seafood.
<b>Populations at elevated risk for infection</b>	Individuals frequently consuming raw or undercooked fishery products.
<b>Documented outbreaks</b>	The disease is not notifiable. In 2025, one clinical case of imported Anisakiasis was published in Switzerland [19].



## Regulatory Aspects

**Regulation:** In Switzerland and the European Union, it is legally required that fishery products intended for consumption in raw, marinated, or salted form undergo a freezing treatment to inactivate viable parasites. The freezing treatment must be carried out at a temperature of at least –20 °C for a minimum of 24 hours or at –35 °C for at least 15 hours [5], [6].

**Detection:** Detection of *Anisakis* in fishery products during routine food inspection relies mainly on visual examination of viscera and fillets, sometimes supported by candling. However, these methods have limited sensitivity



## RESOURCES

Website of the Federal Food Safety and Veterinary Office:  
<https://www.blv.admin.ch/blv/en/home/lebensmittel-und-ernaehrung.html>

## CONTACTS

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