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Foodborne anisakiasis and allergy

Fiona J. Baird [a,b,d], Robin B. Gasser [c], Abdul Jabbar [c], Andreas L. Lopata [a,b,d,*]

[a] School of Pharmacy and Molecular Sciences, James Cook University, Townsville, Queensland 4811, Australia
[b] Centre of Biosecurity and Tropical Infectious Diseases, James Cook University, Townsville, Queensland 4811, Australia
[c] Department of Veterinary Science, The University of Melbourne, Victoria 3010, Australia
[d] Centre for Biodiscovery and Molecular Development of Therapeutics, James Cook University, Townsville, Queensland 4811, Australia

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ABSTRACT

Human anisakiasis, a disease caused by *Anisakis* spp. (Nematoda), is often associated with clinical signs that are similar to those associated with bacterial or viral gastroenteritis. With the globalisation of the seafood industry, the risk of humans acquiring anisakiasis in developed countries appears to be underestimated. The importance of this disease is not only in its initial manifestation, which can often become chronic if the immune response does not eliminate the worm, but, importantly, in its subsequent sensitisation of the human patient. This sensitisation to *Anisakis*-derived allergens can put the patient at risk of an allergic exacerbation upon secondary exposure. This article reviews some aspects of this food-borne disease and explains its link to chronic, allergic conditions in humans.

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1. Introduction

Gastrointestinal worms represent a huge global burden of disease, with more than 2.5 billion people infected [1]. These worms are usually contracted through the ingestion of infective eggs or larvae, or through larval penetration of the skin [2]. Traditionally, these parasites are much more prevalent in developing than developed countries [3]. Some nematodes, such as *Necator americanus* (hookworm) and *Trichuris trichiura* (whipworm), are actually thought to be beneficial in preventing the development of allergy and other chronic inflammatory conditions, such as colitis, through their anti-inflammatory or immuno-modulatory excretory/secretory (ES) molecules [4–6]. In contrast, other nematodes, such as *Anisakis simplex sensu stricto* (*s.s.*), are the causes of such allergic diseases [7,8].

Anisakid nematodes (Nematoda: Anisakidae) affect a growing number of people in developed countries. Commonly reported species are *Anisakis simplex* (*s.s.*) and *Anisakis pegreffii,* and are known as herring, seal or cod worms. They were first implicated as a cause of gastrointestinal illness in 1876 [9], with immunological confirmation in the early 1980s [10,11]. The causative agent was later described by van Thiel et al. [12] in a Dutch patient, who had consumed raw herring. Since this discovery, more than 20,000 cases have been reported worldwide, with the majority of cases being in Japan, where, today, 2000–3000 cases are reported annually [13,14]. Anisakidosis refers to the disease caused by any member of the family Anisakidae, whereas anisakiasis is caused by members of the genus *Anisakis.* This brief review focuses on anisakiasis and its significance in relation to chronic, allergic conditions.

2. Anisakid biology

Different anisakids can be found in different cetacean hosts, depending on local environments [2,15,16]. Genetically confirmed *A. simplex (s.s.)* has been recorded in nine species of cetacean hosts, whereas *A. pegreffii* has been found as an adult in three species of dolphin (family Delphinidae) [17]. Infected cetacean definitive hosts excrete *Anisakis* eggs in their faeces into the aquatic environment. Individual first-(L1) and then second-stage (L2) larvae develop inside these eggs. Larvated eggs then hatch to release motile, free-living L2s, which are ingested by crustacean

* Corresponding author. Centre for Biodiscovery and Molecular Development of Therapeutics, James Cook University, Townsville, Queensland 4811, Australia. Tel.: +61 7 4781 14563; fax: +61 7 4781 6078.
E-mail address: andreas.lopata@jcu.edu.au (A.L. Lopata).

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intermediate hosts, in which L2s develop to third-stage (L3) larvae [2]. L3-infected crustaceans are then consumed by fish or squid (paratenic hosts), in which L3s penetrate the intestine and encapsulate in tissues, particularly of the mesentery and liver. Other paratenic hosts, such as lamprey [18], fish and cephalopods [2,19], can also be involved and accumulate L3s in a similar way. Cetaceans then consume L3-infected fish and squid, after which the L3s are released and then develop to fourth-stage larvae (L4s) and subsequently to adult stages (males and females) in the stomach (Fig. 1).

Humans are accidental hosts in the anisakid life cycle. People infected by ingesting raw or undercooked fish or squid can be affected by anisakiasis, of which there are gastric, intestinal, ectopic and allergic forms [20–23]. Gastric anisakiasis often has a rapid onset, with the patient experiencing epigastric pain, nausea, vomiting and low-grade fever [23]. Intestinal anisakiasis is usually characterised by intermittent or constant abdominal pain, five to seven days following ingestion of infected fish or squid, with possible complications, such as peritonitis and/or ascites [21]. Ectopic anisakiasis, also known as extra-gastrointestinal or intraperitoneal anisakiasis, results when ingested larvae penetrate the stomach or intestinal wall, and then migrate into and through the viscera [22,24]. In contrast, an allergic reaction can also result from anisakid infections, reflected in urticaria, gastrointestinal signs, angioedema and/or anaphylaxis [7]. Clinical signs can subside following endoscopic-guided removal of larvae from the gut, and a diagnosis can be made by identifying such larvae using microscopic or molecular techniques [25].

3. Molecular identification and classification of anisakids

There are many species of *Anisakis* which can vary considerably in their host and geographical distributions as well as biology (reviewed in Ref. [17]). For example, it has been reported that *A. simplex (s.s.)* penetrates the musculature of *Scomber japonicas* (mackerel) better than *A. pegreffii*, leading to increased establishment and a higher intensity of infection [26]. *Anisakis* represents a complex of species [16], and is related to species of *Pseudoterranova* and *Contracaecum* based on the presence of three bilobed lips, a ventral boring tooth and large excretory glands [20,25,27]. Based on larval morphology, there are two main clades (I and II). Clade I includes *A. simplex (s.s.)*, *A. pegreffii*, *A. simplex* C, *Anisakis* sp., *Anisakis typica*, *Anisakis ziphidarum* and *Anisakis nascettii*, with the former three sibling species representing the *A. simplex* complex.

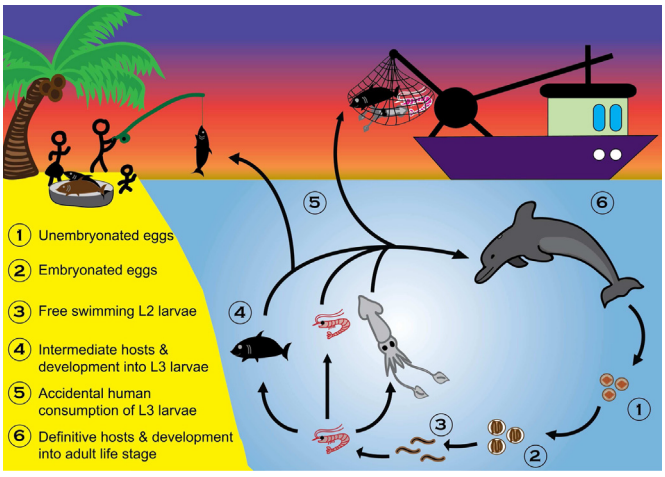

Fig. 1. Life cycle of the species of *Anisakis*, including humans as accidental paratenic hosts.

Clade II comprises of *Anisakis physeteris*, *Anisakis brevispiculata* and *Anisakis paggiae* [28], characterised morphologically by their long, sigmoidal ventriculus and thin, long and uneven male spicules [28].

However, members of the *Anisakis* complex cannot be readily identified to species using traditional, morphological methods [29], such that molecular approaches are often used [27]. Biochemical methods have been employed [30], but PCR-coupled approaches have been more suitable, because they allow the specific amplification of DNA from minute amounts of fresh, frozen or ethanol-fixed material from individual nematodes (larvae). Critical for the identification of worms to the species level is the use of reliable genetic markers. The first and second internal transcribed spacers (ITS-1 and ITS-2, respectively) of nuclear ribosomal DNA have proven useful for the specific or genotypic identification of many anisakids [31,32]. The sequence differences between or among species are usually much higher than variation within species [33].

Mutation scanning-coupled sequencing of ITS-1 and/or ITS-2, followed by phylogenetic analysis of sequence data, is presently the best approach to identify and classify operational taxonomic units of *Anisakis* (see Fig. 2) and related nematodes, such as *Hysterothylacium* and *Contracaecum* species [32,34–38]. This approach has been widely used for the high resolution analysis of genetic variation in ITS-1 and/or ITS-2 amplicons of up to 500 bp [39]. The phylogenetic analyses of ITS-1 and/or ITS-2 sequence data mostly support the original taxonomic division of *Anisakis* species into two clades [32,34,38,40]. This division is further supported by mitochondrial cytochrome c oxidase 2 (*cox*2) sequence data sets as well as allozyme electrophoretic data, which confirm that Clade I comprises the *A. simplex* complex (i.e. *A. simplex (s.s.)*, *A. pegreffii* and *A. simplex* C), *A. typica*, *A. ziphidarum*, and *Anisakis* sp. and that Clade II includes *A. physeteris*, *A. brevispiculata*, and *A. paggiae* [17,28]. Importantly, members of Clade I, such as *A. simplex* s. s., *A. pegreffii*, *A. simplex* C, are recognised as the principal causative agents of anisakiasis (reviewed in Refs. [41,42]).

4. Geographical and host distributions

The Anisakis species within Clade I are distributed mainly in the Atlantic Ocean, and the East and West Pacific, where their definitive hosts (family Delphinidae) are common. The distribution of *A. simplex (s.s.)* and *A. pegreffii* extends throughout the Mediterranean sea, with both species reaching as far north as the Arctic Circle and as far south as the Antarctic waters [43]. Multiple anisakid species can be isolated from one parasitised fish. For example, *Scomber japonicus* (chub mackerel) caught in Japan was found to harbour both *A. simplex (s.s.)* and *A. pegreffii* larvae, and other species [41]. Interestingly, it has been hypothesised that anisakiasis in Japan is mainly caused by *A. simplex (s.s.)*, because it seems to penetrate the muscle tissue at a higher rate than *A. pegreffii*, leading to a higher exposure rate when mackerel is eaten [26]. One study reported that the diversity of multiple anisakid species in parasitised fish decreased with latitude, as more species were isolated from fish in northern compared with southern regions [37].

Geographical regions in which human anisakiasis are common include Japan, Europe and countries in Africa and South America – here anisakids can be found consistently in fish caught locally. In northern Morocco, for instance, *A. pegreffii* has been found in *Trachurus trachurus* (horse mackerel) caught in both Atlantic or Mediterranean waters, yet no *A. simplex (s.s.)* has been isolated from this species of fish [44]. In Italy, *Anisakis* species have been found in numerous species of fish from the Ligurian Sea, but *A. pegreffii* was found at a high prevalence in *T. trachurus* [45]. *A. pegreffii* can also be found in different squid species, such as *Todaropsis eblanae* (lesser flying squid), *Todarodes sagittatus* (European flying squid) and *Todarodes angolensis* (Angola flying squid); and, *A. simplex (s.s.)*

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