

Review

A Scoping Review of Naturally Occurring Xenomas in Fish: Clinical Features, Diagnostic Approaches, and Knowledge Gaps

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Abstract

Xenomas are distinctive hypertrophic host-cell lesions caused by intracellular parasites and represent a recurrent pathological finding in wild and farmed fish. Their presence has implications for fish health, diagnostic workflows, aquaculture productivity, and in some cases product quality and consumer acceptability. Despite this relevance, information on xenoma diversity, associated pathogens, and diagnostic practices remains fragmented across decades of literature. This scoping review synthesised available evidence on naturally occurring xenomas in fish, following PRISMA-ScR guidelines. Eligible sources included studies reporting clinical, pathological, or diagnostic information on xenomas in fish. Data were charted on host species and families, taxonomic identification of the aetiological agents, xenoma morphology, and diagnostic approaches. Across 114 publications published between 1968 and 2024, xenomas were reported in a wide range of teleost families and were attributed mostly to microsporidian infections, particularly species of *Glugea*, *Loma*, *Spraguea*, *Pleistophora*, and *Microgemma*, although myxosporean-associated cases (*Kudoa*, *Myxidium*, *Nephrocystidium*) were also documented. Light and electron microscopy were the most frequently applied diagnostic methods, whereas molecular techniques were used less consistently, with increasing use in the most recent decade. Macroscopic xenomas were typically described as whitish, rounded to oval, and well delimited, yet substantial morphological variation occurred across hosts and tissues. Overall, the review highlights major heterogeneity in pathogen identification and diagnostic pathways, underscoring the need for more standardised and integrative approaches.

Keywords: electron microscopy; light microscopy; histopathology; microsporidia; myxozoa; teleosts; xenoparasitic complex



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1. Introduction

Aquaculture has become one of the fastest growing food-producing sectors worldwide, yet infectious diseases remain a major constraint to its sustainable expansion [1,2]. While economic losses associated with ectoparasitic infestations have long been recognised,

since the late twentieth century the most severe impacts on wild and farmed fish populations have been attributed to infections caused by intracellular parasites, particularly microsporidia and myxozoa [3]. Microsporidia are currently recognised as organisms closely related to Fungi, whereas myxozoans are metazoan parasites within the phylum Cnidaria; nevertheless, both groups include taxa that develop intracellular stages in fish hosts and are associated with major disease outbreaks. These parasites are responsible for important conditions, such as whirling disease and proliferative kidney disease in salmonids [4,5], leading to considerable mortality and economic losses in both freshwater and marine species.

A distinctive feature of many microsporidian and some myxosporean infections is the formation of xenomas, hypertrophic host cells in which the pathogen proliferates [6,7]. The host cell and the infectious organism are morphologically and physiologically integrated within the xenoma, and the current understanding is that this conformation (also known as a 'xenoparasitic complex') shields the pathogen from the host's immune response. Infected host-cells in fish may reach several millimetres in size, with their cytoplasm containing one or more developmental stages of the pathogen and its spores [8].

Xenomas are characteristic lesions in several fish diseases, and they can severely impair tissue function, contributing to morbidity and mortality in affected stocks [9]. Their occurrence has been documented in a variety of farmed species, including commercial valuable ones, where they represent an additional challenge for fish health and welfare management. Beyond aquaculture, xenoma-forming infections also affect laboratory model species such as zebrafish (*Danio rerio*) [10]. In this context, microsporidian infections can alter the host's behaviour and neurophysiology, raising concerns about the reliability of zebrafish as a biomedical model when infections go undetected [11].

The impact of xenoma development on fish health varies depending on the affected organs and tissues, as well as on the severity of infection (e.g., the number and size of xenomas) [12,13]. Xenomas developing in gills can compromise the respiratory efficiency, whereas those occurring in the central nervous system may induce neurological dysfunctions or behavioural alterations. In the muscular tissue, severe infections may lead to necrosis or liquefaction of fibres, resulting in impaired swimming performance, scoliosis or lordosis. Indirect mortality can increase due to impaired swimming and diminished predator avoidance [14]. In the intestine, xenomas can interfere with nutrient absorption, contributing to reduced growth and body condition [15], and in the gonads they may impair reproductive capacity through ovarian degeneration [16]. The combined negative metabolic effects of the infection and the loss of functional tissues ultimately lead to reduced growth rates and performance in both wild and farmed fish populations [17]. Visible xenomas may also have commercial implications: nodules or deformities in marketable species can render fish unsuitable for sale or consumption [18].

The diagnosis of xenoma-forming infections is inherently challenging. Gross lesions may appear as whitish nodules, granulomas or areas of tissue discolouration, but these are not pathognomonic and can easily be confused with other infectious or non-infectious lesions. Light microscopy of wet mounts or histological sections can reveal spores or altered host cells; moreover, ultrastructural characterisation by transmission electron microscopy has historically provided insights into xenoma-forming organisms structure and biology [19,20]. Over the past two decades, molecular methods have gained increasing importance for species-level identification, introducing the possibility of non-lethal sampling [21].

Historically, in the absence of molecular tools, aetiological identification relied on a combination of morphological, ultrastructural and ecological criteria, such as the size and shape of spores, ultrastructural features including the number and arrangement of polar filament coils, the dimensions and anatomical location of the xenoma, and the known host

specificity of the parasite [15]. However, subsequent molecular reassessments have revealed that phenotypic traits do not always correlate with genetic classification, highlighting the limitations of morphology-based taxonomy. Despite their conspicuous size and accessibility, macroscopic xenomas have often been overlooked in molecular investigations, likely due to the absence of suitable in vitro systems [8]. As a result, diagnostic approaches remain heterogeneous across studies and are rarely standardised, making comparisons difficult and hindering the development of surveillance and control programmes. Such gaps also limit early detection efforts in both aquaculture and laboratory facilities, where a timely diagnosis is critical to reducing transmission and losses.

In this review, we adopt the definition of xenoma proposed by Lom and Dyková [7], namely “a host cell with a completely changed structure, with the parasites proliferating inside it. Both components, host and parasite, are morphologically and physiologically integrated to form a separate entity with its own development in the host, at the expense of which it grows”, while applying it in a broader sense to all intracellular developmental stages of infectious agents capable of inducing such lesions in fish. Retrospective application of this definition is not always straightforward, as many earlier publications employed heterogeneous terminology, such as “xenoparasitic complexes”, “xenoma-like formations”, “cysts”, or “tumour-like structures”. Furthermore, lesions caused by members of the family Xcellidae have frequently been described in the literature as ‘xenomas’ [22], although they consist of proliferating parasite cells rather than hypertrophic host cells, and therefore do not conform to the adopted definition. These terminological and conceptual inconsistencies emphasise the importance of systematically re-evaluating the existing literature using clearly defined inclusion criteria.

The present scoping review collates reports of naturally occurring xenomas in fish, mapping the affected species and the corresponding aetiological agents when available. It also examines the diagnostic methodologies employed, including clinical and gross examination, light and electron microscopy, histopathology, and molecular techniques, and some epidemiological aspects, such as water salinity, temperature, and prevalence, highlighting methodological gaps and inconsistencies that limit the detection and study of xenoma-forming infections in fish.

2. Materials and Methods

2.1. Search Strategy

The aim of this scoping review was to examine how the clinical or pathological finding of xenomas in fish has been investigated through different approaches (e.g., histology, electron microscopy, molecular biology), and to identify existing knowledge gaps. No protocol was registered for this review. A systematic literature search was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for scoping reviews (PRISMA-ScR) [23].

The databases PubMed, Web of Science (‘all databases’ option), and Scopus (‘all fields’ option) were queried using the search string <<fish xenoma*>>. Eligible records were limited to full-text articles published in English from database inception to 15 May 2025. Preliminary tests with broader search strings, incorporating fish-specific terms and synonyms of xenomas (e.g., combining ‘teleosts’, ‘osteichthyes’, or ‘cartilaginous fish’ with ‘xenoma’, or including terms such as ‘xenoparasitic complex’ or ‘cyst’), either retrieved very few records, often not pertinent to the research question, or generated a large number of irrelevant results (e.g., ‘cyst’ yielding thousands of non-specific records). The final minimal string, ‘fish xenoma’, proved to be the most effective compromise, ensuring both sensitivity and specificity, while avoiding unnecessary noise. No geographical or taxonomic restrictions were applied to ensure a comprehensive search.

Application of this string yielded 120, 201, and 485 publications, on PubMed, Web of Science, and Scopus, respectively. In addition, six further records were identified through manual screening of reference lists. After removal of duplicates ($n = 301$), non-English publications ($n = 20$), and unavailable full-texts ($n = 15$), a total of 476 records were retained for title and abstract screening.

2.2. Study Selection

Three reviewers (A.M., C.C., and M.C.) independently screened titles and abstracts, while a fourth reviewer (G.F.) performed an independent calibration screening of 10% of the excluded papers, achieving high level of agreement (>90%). Initials refer to the authors listed in the title page. When uncertainties arose during the screening process, decisions were reached by consensus among the reviewers; in cases of persistent disagreement, G.F. acted as an arbitrator and provided the final decision.

The 319 records remaining after title and abstract screening were assessed in full text by three reviewers (A.M., C.C., M.C.), applying the selected exclusion criteria (see Section 3 for details). An independent calibration screening of 10% of the excluded papers was again performed by G.F., achieving >90% agreement.

2.3. Data Extraction and Synthesis

From each included study, three reviewers (A.M., S.M., M.C.) independently extracted relevant data, which were organised into predefined categories (Table 1) and compiled them into a structured database (Supplementary Table S1). Each reviewer extracted data from a subset of the studies. Discrepancies were resolved through discussion, with the senior reviewer (G.F.) providing the final decision when necessary. Supplementary Materials and appendices were also reviewed when available. For each host species, the aquatic environment was classified as freshwater, saltwater, or brackish-water, based on habitat information from FishBase (www.fishbase.org, last accessed 26 November 2025) [24]. Likewise, common names of fish species were standardised according to the Food and Agriculture Organization (FAO) English nomenclature reported in FishBase, to ensure consistency across taxa; in cases where an FAO-English common name was not available, the common or vernacular (local) name provided by the original authors was retained (indicated as ‘auth’). Taxonomic nomenclature for microsporidia and myxozoans was retained as reported in the original studies; current taxonomic status was cross-checked against the World Register of Marine Species (WoRMS, last accessed 2 January 2026) [25]. Data synthesis was descriptive, with results summarised in tables and narratively integrated to identify trends and knowledge gaps.

Table 1. Data extracted from the selected publications.

Main Topic	Category	Definition
Biblio-graphic Metadata	Authors	List of authors
	Title	Title of the publication
	Reference	Journal, Volume, Issue, Pages
	Year	Year of publication
Host and Biology	Family	Host taxonomy Family
	Species	Host genus and species
	Common name	FAO English common name for the fish species
	Habitat	FW = fresh-water species, SW = salt-water species, BW = brackish-water species
	Origin	C = captive-bred specimens, W = wild caught specimens, Local fish market
	Captivity condition	Aquaculture Farm (F), Aquarium (A) or Laboratory setting (L)
	Temperature	Water temperature in °C

Table 1. Cont.

Main Topic	Category	Definition
Host and Biology	Clinical signs	Signs observed at clinical examination
	Sample size	Number of examined fish specimens
	N. positive	Number of fish positive for presence of xenoma
	Prevalence (%)	Prevalence (N° positive for xenoma/N° examined) % If multiple categories assessed, prevalence % in different categories
	Xenoma described from Single (S) or Multiple (M) organs Affected organ(s)	Single (S) or Multiple (M) organs affected by xenoma formation List of all xenoma-affected organs
Diagnostic Protocol	Xenoma visible at naked eye	Possibility of detecting xenoma at naked eye, through clinical or pathologic examination (yes Y/no N)
	Gross description of visible xenomas	Morphological features of visible xenomas
	Size	Size of xenomas (millimetres)
	Fresh smears	Applied when direct light microscopy of wet mounts was performed (yes Y/no N)
	Semi-thin sections	Applied when direct light microscopy of thin sections was performed (yes Y/no N)
	Histology	Applied when histology of xenomas or affected organs was applied on fixed, embedded, sectioned, and stained tissue samples (yes Y/no N)
	Electron microscopy	Applied when xenoma ultrastructure was studied by electron microscopy (yes Y/no N)
	Molecular diagnosis	Applied when molecular techniques were used for aetiological identification (yes Y/no N)
	Public sequences	Sequences deposited in public repositories (yes Y/no N)
	Molecular diagnostic approach	Molecular diagnostic approach (PCR, cloning, sequencing)
	Genes	Target genes
	Accession number	GenBank (GB)/European Nucleotide Archive (ENA) accession number
	Aetiological agent detected	An aetiological agent responsible for xenoma-formation was identified in the study (yes Y/no N)
	Agent Phylum	Phylum of the aetiological agent identified
	Agent Species	Genus and species of the aetiological agent identified

3. Results

3.1. Literature Search and Study Selection

The literature search yielded a total of 476 records. After screening of titles and abstracts, 157 records were excluded. The remaining studies underwent full-text assessment, after which further exclusions were made for the following reasons: (i) not involving fish species ($n = 9$); (ii) experimental or in vitro studies ($n = 74$); (iii) no clinical or pathological description of affected fish, including studies addressing xenomas only at the tissue, cellular, or ultrastructural level without reference to individual infection cases ($n = 22$); (iv) papers not referring to xenomas ($n = 40$); (v) lesions initially resembling xenomas but histologically diagnosed as different conditions ($n = 17$); (vi) book chapters or review articles not reporting new xenoma cases ($n = 38$); and (vii) duplicate or preliminary reports superseded by more comprehensive publications referring to the same infection event or dataset ($n = 5$).

A total of 114 studies met the inclusion criteria and were incorporated into the scoping review. The complete study selection process is depicted in Figure 1 (PRISMA-ScR flow diagram). Full details of all charted variables for each included study are available in Supplementary Table S1, which provides the complete data extraction sheet.

PRISMA-ScR Flow Diagram

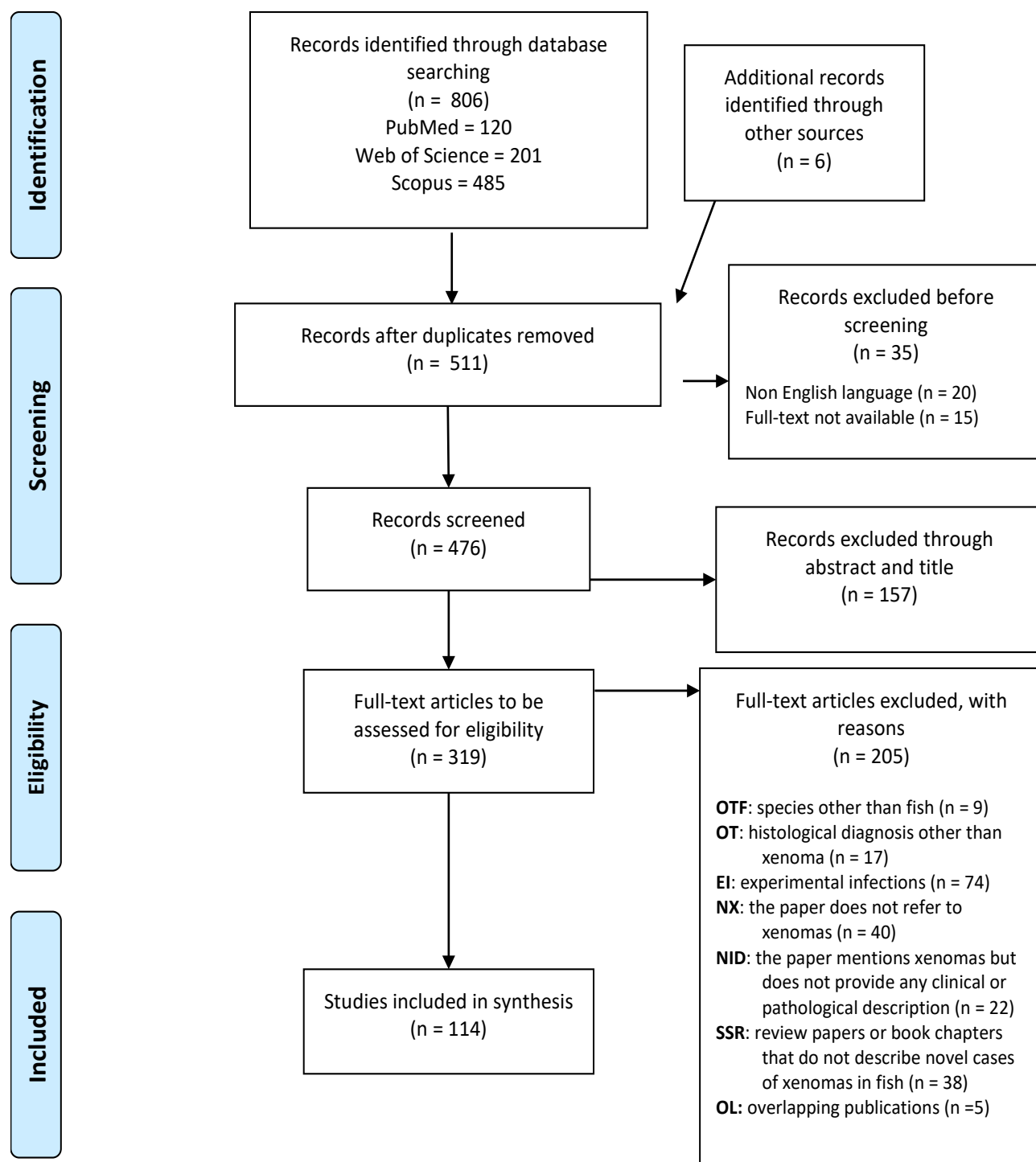


Figure 1. PRISMA-ScR Flow Diagram.

3.2. Characteristics of Included Studies and Host Fish

3.2.1. Study Characteristics

The earliest included publication dates to 1968 [26]. Most studies were published between 2010 and 2019 (40/114; 35.1%). The proportion observed for the most recent period (2020–May 2025: 14/114; 12.3%) is not directly comparable with previous decades due to the shorter time span (Table 2).

Table 2. Number of included studies published over time.

Decade	Number of Studies	%
1968–1989	20	17.6%
1990–1999	16	14%
2000–2009	24	21%
2010–2019	40	35.1%
2020–2025	14	12.3%
Total	114	

Most publications focused on a single fish species, while a subset of studies (17/114; 15%) reported clinical–pathological findings and diagnostic procedures for xenomas occurring in multiple species or families. For this reason, data extraction was performed per fish species rather than solely per study, resulting in a total of 150 species-specific xenoma records.

Most fish examined across these records originated from wild populations ($n = 126$). Captive fish were less frequently represented, including aquaculture stocks ($n = 17$), aquarium specimens ($n = 3$), and laboratory fish ($n = 2$). Additionally, seven records involved fish sampled from local markets. The total exceeds 150 because several studies included fish originating from multiple sources.

3.2.2. Host Species and Families

Across the 150 species-specific records, 113 fish species belonging to 52 families were documented as hosts of xenoma-forming pathogens (Table 3). The most frequently represented families were Sparidae (11 species), Carangidae, Lophiidae, and Salmonidae (10 species each), followed by Gadidae (nine species).

Table 3. List of the fish families and species examined in the studies included in the review. N° indicates the number of studies per fish family for which xenomas were reported. Detailed sample sizes at the study and species level are provided in Supplementary Table S1. The asterisk in the Aetiological agents column (*) indicates species belonging to the myxozoa SubPhylum (Phylum Cnidaria).

Fish Family	Fish Species	N° of Studies per Fish Family	Aetiological Agents Identified (Genus and Species)	Phylum of Aetiological Agents Identified	References
Ammodytidae	<i>Hyperoplus lanceolatus</i>	1	<i>Microgemma caulleryi</i>	Microsporidia	[27]
Anoplopomatidae	<i>Anoplopoma fimbria</i>	1	<i>Loma richardi</i>	Microsporidia	[28]
Apogonidae	<i>Vincentia conspersa</i>	1	<i>Glugea vincentiae</i>	Microsporidia	[29]
Atherinidae	<i>Atherina boyeri</i>	1	<i>Loma acerinae</i>	Microsporidia	[30]
Belonidae	<i>Potamorhaphis guianensis</i>	1	<i>Potasporea morhaphis</i>	Microsporidia	[31]
Caesionidae	<i>Caesio striata</i>	1	<i>Glugea eda</i>	Microsporidia	[32]
Callionymidae	<i>Callionymus filamentosus</i>	1	<i>Obruspora papernae</i>	Microsporidia	[33]
Carangidae	<i>Flavocaranx bajad</i> , <i>Trachinotus carolinus</i> , <i>Trachurus trachurus</i> , <i>Selene dorsalis</i> , <i>Caranx</i> <i>senegallus</i> , <i>Caranx crysos</i> , <i>Carangoides malabaricus</i> , <i>Atule mate</i>	7	<i>Glugea malabaricii</i> , <i>Glugea</i> <i>sp.</i> , <i>Kudoa azevedoi</i> *, <i>Microgemma carolinus</i> , unidentified Microsporidia	Microsporidia, Myxozoa	[34–40]

Table 3. Cont.

Fish Family	Fish Species	N° of Studies per Fish Family	Aetiological Agents Identified (Genus and Species)	Phylum of Aetiological Agents Identified	References
Cepolidae	<i>Cepola macrophthalmia</i>	2	<i>Microgemma ovoidea</i>	Microsporidia	[41,42]
Cichlidae	<i>Oreochromis niloticus</i> , <i>Aequidens plagiozonatus</i>	2	<i>Loma camerounensis</i> , <i>Potasporea aequidensis</i>	Microsporidia	[43,44]
Cottidae	<i>Taurulus bubalis</i> , <i>Cottus bairdii</i> , <i>Cottus cognatus</i>	3	<i>Glugea</i> sp., <i>Microgemma vivaresi</i>	Microsporidia	[45–47]
Cyclopteridae	<i>Cyclopterus lumpus</i>	1	<i>Tetramicra brevifilum</i>	Microsporidia	[48]
Cyprinidae	<i>Pimephales promelas</i> , <i>Culter alburnus</i> , <i>Danio rerio</i> , <i>Cultrichthys erythropterus</i>	4	<i>Glugea pimephales</i> , <i>Pseudokabatana alburnus</i> , <i>Pseudoloma neurophilia</i>	Microsporidia	[49–52]
Cyprinodontidae	<i>Nothobranchius eggersi</i> , <i>Nothobranchius korthausae</i>	1	<i>Glugea</i> sp.	Microsporidia	[53]
Doradidae	<i>Hassar orestis</i>	1	<i>Amazonspora hassar</i>	Microsporidia	[54]
Dorosomatidae	<i>Sardinella aurita</i>	4	<i>Glugea plecoglossi</i> , <i>Glugea sardinellensis</i> , <i>Glugea</i> sp.	Microsporidia	[17,55–57]
Embiotocidae	<i>Cymatogaster aggregata</i>	1	<i>Loma embiotocia</i>	Microsporidia	[58]
Epinephelidae	<i>Epinephelus chlorostigma</i> , <i>Epinephelus polyphekadion</i>	3	<i>Glugea arabica</i> , <i>Pleistophora</i> sp.	Microsporidia	[59–61]
Esocidae	<i>Esox lucius</i>	2	<i>Myxidium lieberkuehni</i> *, <i>Nephrocystidium pickii</i> *	Myxozoa	[62,63]
Gadidae	<i>Gadus microcephalus</i> , <i>Gadus chalcogrammus</i> , <i>Microgadus proximus</i> , <i>Gadus morhua</i> , <i>Melanogrammus aeglefinus</i>	7	<i>Loma branchialis</i> , <i>Loma kenti</i> , <i>Loma morhua</i> , <i>Loma pacificodae</i> , <i>Loma wallae</i> , <i>Loma</i> sp.	Microsporidia	[28,64–69]
Gasterosteidae	<i>Gasterosteus aculeatus</i> , <i>Apeltes quadracus</i>	4	<i>Glugea anomala</i> , <i>Glugea weissenbergi</i>	Microsporidia	[20,26,70,71]
Gobiidae	<i>Trypauchen vagina</i> , <i>Taenioides nigrimarginatus</i> , <i>Neogobius melanostomus</i> , <i>Neogobius fluviatilis</i> , <i>Proterorhinus semilunaris</i> , <i>Clevelandia ios</i>	4	<i>Glugea</i> sp., <i>Ichthyosporidium weissii</i> , <i>Loma acerinae</i> , <i>Microgemma tilanpasiri</i> , unidentified Microsporidia		[30,72–74]
Hexagrammidae	<i>Ophiodon elongatus</i>	1	<i>Loma lingcodae</i>	Microsporidia	[28]
Hypopomidae	<i>Brachyhypopomus beebei</i> , <i>Brachyhypopomus brevirostris</i>	2	<i>Microsporidium brevirostris</i> , <i>Pleistophora beebei</i>	Microsporidia	[75,76]
Labridae	<i>Symphodus tinca</i> , <i>Cheilinus chlorourus</i>	2	<i>Glugea</i> sp., <i>Microgemma tincae</i>	Microsporidia	[39,77]
Lophiidae	<i>Lophius litulon</i> , <i>Lophius piscatorius</i> <i>Lophius gastrophysus</i> , <i>Lophius budegassa</i>	8	<i>Spraguea americana</i> , <i>Spraguea gastrophysus</i> , <i>Spraguea lophii</i> , <i>Spraguea</i> sp.	Microsporidia	[18,78–84]
Lotidae	<i>Lota lota</i>	1	<i>Myosporidium spraguei</i>	Microsporidia	[85]
Lutjanidae	<i>Lutjanus bohar</i> , <i>Lutjanus fulgens</i>	2	<i>Glugea jazanensis</i> , <i>Microfilum lutjani</i>	Microsporidia	[86,87]

Table 3. Cont.

Fish Family	Fish Species	N° of Studies per Fish Family	Aetiological Agents Identified (Genus and Species)	Phylum of Aetiological Agents Identified	References
Macruridae	<i>Coryphaenoides nasutus</i>	1	<i>Pleistophora duodecimae</i>	Microsporidia	[88]
Moronidae	<i>Dicentrarchus labrax</i>	1	<i>Loma psittaca</i>	Microsporidia	[89]
Mugilidae	<i>Chelon labrosus</i>	1	<i>Microgemma hepaticus</i>	Microsporidia	[90]
Myctophidae	<i>Myctophum punctatum</i>	1	<i>Glugea capverdensis</i>	Microsporidia	[88]
Nannopercidae	<i>Nannoperca vittata</i>	1	Unidentified Microsporidia	Microsporidia	[91]
Ophichthidae	<i>Myrophis platyrhynchus</i>	2	<i>Loma myrophis</i>	Microsporidia	[92,93]
Osmeridae	<i>Osmerus eperlanus</i> , <i>Osmerus mordax</i>	2	<i>Glugea hertwigi</i>	Microsporidia	[94,95]
Oxudercidae	<i>Boleophthalmus dussumieri</i>	1	<i>Microgemma</i> sp.	Microsporidia	[96]
Percidae	<i>Sander lucioperca</i>	1	<i>Myosporidium spraguei</i>	Microsporidia	[85]
Plecoglossidae	<i>Plecoglossus altivelis</i>	1	<i>Glugea plecoglossi</i>	Microsporidia	[97]
Pleuronectidae	<i>Platichthys flesus</i> , <i>Parophrys vetulus</i> , <i>Platichthys stellatus</i> , <i>Glyptocephalus cynoglossus</i> , <i>Pseudopleuronectes americanus</i>	6	<i>Glugea stephani</i> , <i>Glugea</i> sp.	Microsporidia	[15,98–102]
Poeciliidae	<i>Gambusia affinis</i>	1	<i>Glugea</i> sp.	Microsporidia	[103]
Pomacentridae	<i>Abudefduf saxatilis</i> , <i>Abudefduf bengalensis</i> , <i>Pomacentrus brachialis</i>	2	<i>Glugea</i> sp.	Microsporidia	[39,104]
Retropinnidae	<i>Retropinna retropinna</i>	1	<i>Glugea</i> sp.		[105]
Salmonidae	<i>Salvelinus fontinalis</i> , <i>Salmo trutta</i> , <i>Oncorhynchus mykiss</i> , <i>Salmo salar</i> , <i>Oncorhynchus tshawytscha</i> , <i>Oncorhynchus kisutch</i>	8	<i>Loma salmonae</i> , <i>Loma</i> sp., unidentified Microsporidia, Unidentified Myxosporean (CKX) *	Microsporidia, Myxozoa	[106–113]
Sciaenidae	<i>Leiostomus xanthurus</i>	1	<i>Ichthyosporidium giganteum</i>	Microsporidia	[114]
Scombridae	<i>Thunnus thynnus</i>	2	<i>Glugea thunni</i> , <i>Microsporidium milevae</i>	Microsporidia	[115,116]
Scophthalmidae	<i>Scophthalmus maximus</i>	2	<i>Tetramicra brevifilum</i>	Microsporidia	[117,118]
Serranidae	<i>Epinephelus tauvina</i> , <i>Cephalopholis hemistiktos</i> , <i>Serranus atricauda</i> , <i>Epinephelus lanceolatus</i> ♂ × <i>E. fuscoguttatus</i> ♀ (hybrid)	4	<i>Glugea nagelia</i> , <i>Glugea serranus</i> , <i>Glugea</i> sp., <i>Loma psittaca</i>	Microsporidia	[119–122]
Soleidae	<i>Dicologlossa cuneate</i> , <i>Synaptura cadenati</i> , <i>Dagetichthys lusitanicus</i> , <i>Vanstraelenia chirophthalma</i>	1	<i>Microsporidium dicologlossae</i> , <i>Microsporidium synapturae</i> , <i>Microsporidium vanstraeleniae</i>	Microsporidia	[123]

Table 3. Cont.

Fish Family	Fish Species	N° of Studies per Fish Family	Aetiological Agents Identified (Genus and Species)	Phylum of Aetiological Agents Identified	References
Sparidae	<i>Pagrus pagrus</i> , <i>Sparus caeruleostictus</i> , <i>Dentex maroccanus</i> , <i>Dentex canariensis</i> , <i>Boops boops</i> , <i>Sparus aurata</i> , <i>Pagrus major</i>	8	<i>Glugea pagri</i> , <i>Kudoa pagrusi</i> *, <i>Loma hoopsi</i> , <i>Microsporidium aurata</i> , <i>Microsporidium caeruleosticti</i> , <i>Microsporidium canariensis</i> , <i>Microsporidium maroccani</i> , <i>Microsporidium pagri</i> , <i>Pleistophora pagri</i> , <i>Pleistophora senegalensis</i> , unidentified Microsporidia	Microsporidia, Myxozoa	[59,124–130]
Synodontidae	<i>Saurida undosquamis</i> , <i>Saurida tumbil</i> , <i>Saurida lessepsianus</i>	5	<i>Heterosporis lessepsianus</i> , <i>Heterosporis</i> sp., <i>Pleistophora aegyptiaca</i> , <i>Pleistophora dammami</i> , unidentified Microsporidia	Microsporidia	[59,131–134]
Tetraodontidae	<i>Colomesus psittacus</i>	1	<i>Loma psittaca</i>	Microsporidia	[135]
Xenocyprididae	<i>Squaliobarbus curriculus</i> , <i>Hemiculter leucisculus</i> , <i>Pseudolaubuca engraulis</i> , <i>Toxabramis swinhonis</i> , <i>Elopichthys bambusa</i>	1	<i>Pseudokabatana alburnus</i>	Microsporidia	[52]

Xenoma occurrences were reported from freshwater (12.4%), saltwater (31.0%), and mixed aquatic environments. Mixed-environment records were frequent, particularly saltwater–brackish (23.0%). Water temperature at the site of origin was rarely reported, with 120/150 records (80%) classified as ‘not available’. In the remaining cases, authors provided a temperature range that varied according to sampling month and locality. Only one study reported a fixed temperature range for aquarium-held fish.

3.3. Quantitative Aspects (Sample Size and Prevalence)

Sample size could be evaluated for 118 out of 150 species-specific records (78.7%), whereas it was not reported in 32 cases (21.3%). Sample sizes ranged from single individuals to over 11,000 fish, with the largest values generally corresponding to multi-year surveys.

Prevalence was not reported in 27 studies (18%). Among the 123 studies that did provide prevalence estimates (82%), values ranged from 0.7% to 100%, with substantial heterogeneity across host species, infectious agents, and study designs. Detailed study-level prevalence data are provided in Supplementary Table S2.

3.4. Clinical Signs in Xenoma-Affected Fish

Most studies focused on pathological lesions and structural features of xenomas and their intracellular agents, while behavioural or clinical alterations were often not reported. Clinical signs were classified as ‘not available’ in 96 out of 150 records (64%). In 10.7% of cases, authors explicitly stated that no clinical abnormalities were observed, or that xenomas did not appear to affect the examined fish. Approximately one quarter of records (38/150; 25.3%) reported variable clinical presentations (Table 4), most commonly abdomi-

nal swelling, lethargy, and impaired swimming. Relatively frequent increases in mortality were also observed, ranging from age-specific excess mortality and high overall mortality to stranding events and the presence of moribund fish within the examined groups.

Table 4. Clinical signs reported in xenoma-affected fish. The table summarises all clinical signs described across the 38 studies that reported clinical observations. A total of 65 distinct observations were extracted, as individual studies frequently documented more than one clinical sign. Percentages refer to the proportion of each sign out of these 65 recorded observations.

Clinical Signs in Xenoma-Affected Fish	Number of Observations	%
Mortality	11	16.9%
Abdominal swelling	10	15.4%
Erratic swimming	7	10.8%
Lethargy	7	10.8%
Emaciation	5	7.7%
Pale or haemorrhagic gills	5	7.7%
Xenoma visible through body wall	5	7.7%
Stunted growth	4	6.1%
Signs attributable to secondary bacterial infection	2	3.1%
Other (exophthalmos, corneal lesions, skin lesions, morphological anomalies, atypical colourations)	9	13.8%

3.5. Anatomical Distribution and Lesion Characteristics

Xenomas were grossly visible in 75.3% of records (113/150), whereas this information was not available for 6% of records (9/150). In the remaining cases, lesions were detected only during dissection under a stereomicroscope or by light microscopy of selected tissues.

Xenomas occurred across a wide range of organs and tissues. Because not all studies examined the full organ set in each fish, and several focused on one or two target organs, the number and diversity of reported xenoma sites are likely underestimated. With this limitation in mind, single-organ involvement occurred in 92/150 records (61.3%), while multiple organs were affected in 55/150 records (36.7%).

Descriptions of xenoma localisation were highly heterogeneous among studies. Some authors reported broad categories such as “gastrointestinal tract”, “coelomic cavity”, or “viscera”, while others specified precise anatomical locations (e.g., “intestinal submucosa”). For this reason, full details of xenoma locations are provided in Supplementary Table S1. Overall, the most frequently affected organs were the liver and intestine, followed by the gills, ovaries, kidney, and skeletal muscle. Additional sites included the heart, spleen, and central or peripheral nervous system.

Xenoma size showed substantial variability, ranging from very small lesions (<0.1 mm) to large macroscopic structures exceeding 20 mm in diameter. Most xenomas fell within the 1–5 mm range, with small lesions (<1 mm) and large lesions (>10 mm) reported less frequently. Very large xenomas (>20 and up to 40 mm) were rare. Overall, the typical size observed across studies was approximately 2–3 mm.

Macroscopic descriptions of xenomas were available for 88% of records. Across studies, xenomas were most commonly reported as whitish lesions, typically spherical, ovoid, or oval in shape. Whitish or opaque-white colouring represented by far the predominant presentation, while yellowish, brownish, or melanised (black) nodules were reported less frequently. In terms of shape, spherical or ovoid xenomas accounted for the majority of descriptions, followed by elongated or cylindrical forms, and more rarely by irregular, fibrous, dendritic, or lobulated structures. Xenomas could be solitary, multifocal, or organised in clusters, including characteristic “grape-like” aggregates in some species. Consistency varied from soft and creamy to firm or occasionally calcified. Many xenomas contained a creamy or opaque white material, while others were described as granular or crumbly.

Marked morphological variability was noted both between and within studies, with several authors reporting xenomas of different sizes or shapes within the same host, or variations related to tissue location.

3.6. Diagnostic Methods

Following detection of xenoma-like lesions upon clinical or pathological examination, the diagnostic workflow across studies relied on light microscopy, electron microscopy, and molecular techniques. Within light microscopy, we considered three distinct approaches: fresh smears (wet mounts, often prepared by squash technique for xenomas to release spores), semi-thin sections (typically stained with toluidine blue), and routine histology on fixed, embedded, sectioned, and stained tissues.

Light microscopy was used in 143/150 records (95.3%): fresh smears in 79.3%, semi-thin sections in 52.7%, and histology in 51.3%. Only 22/150 records (14.7%) applied all three techniques. Seven records (4.7%) did not use light microscopy, relying instead on TEM only ($n = 2$), molecular techniques only ($n = 2$), or clinical–pathological/epidemiological evidence alone ($n = 3$). Electron microscopy (mainly TEM, occasionally SEM) was applied in 73.3% of records, and molecular techniques in 46%. A fully integrated workflow combining all three methods was reported in 13 records (8.7%).

Temporal trends were observed across decades (Figure 2). Molecular methods became increasingly common from the 2000s onwards, whereas the use of TEM and semi-thin sections has gradually declined. Fresh smears and histology remained consistently employed across all decades.

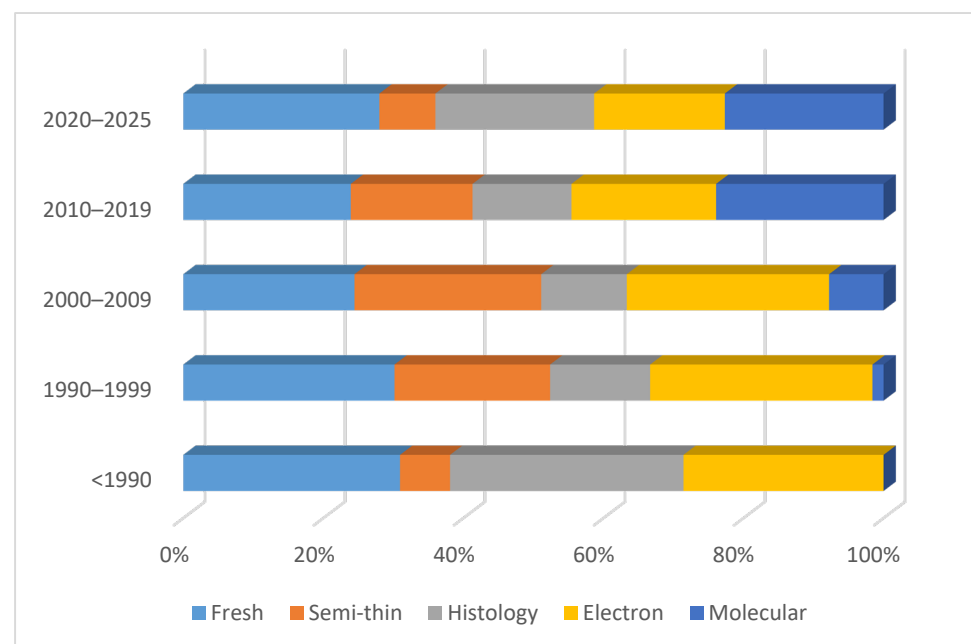


Figure 2. Relative use of diagnostic techniques across decades of publication. Each bar represents the proportion of studies published in a given decade that employed: fresh smears (light blue), semi-thin sections (orange), histology (grey), electron microscopy (yellow), and molecular methods (blue). Values denote the percentage of records per decade in which each technique was reported.

3.7. Aetiological Agents

An intracellular parasitic agent was detected in all 150 records. Microsporidia accounted for 145/150 records (96.7%), while myxozoans were reported in 2.6% of cases, including *Myxidium lieberkuehni*, *Kudoa azevedoi*, *Nephrocystidium pickii*, and one unidentified

myxosporean (labelled CKX). A single study reported a mixed infection in four specimens of *Pagrus pagrus*, involving an unidentified microsporidian together with *Kudoa pagrusi* [59].

Among 145 microsporidian records, the agent was identified to species level in 109 records (75.2%), to genus level in 28 (19.3%), and remained unassigned in 8 (5.5%). Several species-level identifications corresponded to newly proposed taxa. Sixteen distinct microsporidia genera were reported (full list in Supplementary Table S1). *Glugea* was the most frequently represented genus, followed by *Loma*, *Microsporidium*, and *Spraguea* (Table 5). Among the overall 75 xenoma-associated agents reported at species level in the reviewed studies, 44 are currently listed as accepted species in the World Register of Marine Species (WoRMS), three are listed under superseded nomenclature, and 28 are not presently included in the database.

Table 5. Microsporidian genera reported in the 137 records in which the aetiological agent was identified to genus or species level.

Genus of Microsporidia	N	%
<i>Glugea</i>	45	32.8%
<i>Loma</i>	31	22.6%
<i>Microsporidium</i>	11	8.0%
<i>Spraguea</i>	10	7.3%
<i>Microgemma</i>	9	6.6%
<i>Pleistophora</i>	9	6.6%
Others (<i>Amazonspora</i> , <i>Heterosporis</i> , <i>Ichthyosporidium</i> , <i>Microfilum</i> , <i>Myosporidium</i> , <i>Obruspora</i> , <i>Potasporea</i> , <i>Pseudokabatana</i> , <i>Pseudoloma</i> , and <i>Tetramicra</i>)	22	16.1%

Within *Glugea*, 17 species were recorded; *Glugea stephani* (6/45; 13.3%) and *Glugea anomala* (3/45; 6.5%) were the most frequently reported and showed high host specificity, occurring exclusively in Pleuronectidae and Gasterosteidae, respectively. Among the 14 species of *Loma*, *Loma salmoneae* (6/31; 19.4%) and *Loma acerinae* (4/31; 12.9%) were most frequently reported, affecting Salmonidae and Gobiidae/Atherinidae respectively. A complete overview of aetiological agents by host family is provided in Table 3.

Across the included records, *Glugea* was represented by 17 species infecting 22 host fish families. Among these, *G. stephani* showed a strong association with Pleuronectidae, while *G. anomala* was restricted to Gasterosteidae. The genus *Loma* comprised 14 species infecting 13 host families. *L. salmoneae* displayed a clear host association with Salmonidae, whereas *L. acerinae* was restricted to Gobiidae and Atherinidae. Ten *Microsporidium* species were reported across four families; half of these cases occurred in Sparidae, which were affected by five distinct *Microsporidium* species. *Microgemma* included seven species infecting eight host families, and *Pleistophora* included six species across five families. The strongest host specificity observed in the dataset concerned the genus *Spraguea*: its three reported species (*S. americana*, *S. gastrophysus*, *S. lophii*) were exclusively associated with Lophiidae.

Patterns of tissue involvement varied across microsporidian genera. *Glugea* and *Loma* displayed broad organ tropism but differed in their most common sites of infection: intestinal xenomas predominated in *Glugea* (36.7% of cases), while gill involvement was most frequent in *Loma* (30.3%). A marked organ specificity was observed for several other genera. *Microgemma* and *Microsporidium* primarily infected the liver (90% and 80% of cases, respectively). *Spraguea* almost exclusively targeted the nervous tissue (81.8%), consistent with the known neurotropism of this genus. *Pseudokabatana alburnus* represented the clearest example of combined host–tissue specificity, being confined to the ovaries of cyprinid hosts.

3.8. Methodological and Reporting Gaps

Several reporting inconsistencies were noted. In 3 of the 150 records (2%), it was not possible to determine single versus multiple organ involvement, and the affected organs were not specified. Water temperature at the collection site was not reported in 120 of 150 records (80%). Sample size information was unavailable in 32 studies; additionally, two multi-species studies provided only cumulative values, without species-specific sample sizes or prevalence estimates. In 18% of records, prevalence data were absent.

Information on the macroscopic visibility of xenomas was missing in 9 of 150 records (6%). Among studies confirming grossly visible xenomas, 12% did not include a description of the lesions. Clinical signs were most frequently unreported: 96/150 records (64%) contained no observations or explicit statement regarding their presence or absence.

Diagnostic methods were generally reported, but three records (two studies) did not apply any formal diagnostic technique, relying solely on epidemiological, clinical, or pathological evidence.

4. Discussion

This scoping review provides a comprehensive synthesis of xenomas in fish, integrating data from 114 studies and 150 species-specific records. The findings highlight patterns in host range, tissue tropism, lesion morphology, clinical presentation, and diagnostic practices, while revealing substantial methodological and reporting gaps.

The review process faced several limitations inherent to the historical literature. First, in earlier publications pre-dating the adoption of Lom and Dyková's definition [7], the term xenoma was inconsistently applied ("tumors", "pseudo-tumors", "cell-hypertrophy-tumor-type", "syncytial xenomas", "cyst-like bodies", "xenoma-like formations", "cysts", etc). In this review, eligibility was determined by verifying whether the lesions described fulfilled the key criteria of xenoma formation (intracellular localisation, hypertrophy of the host cell, and the establishment of a host-infectious agent functional unit). This approach was necessary to avoid including extracellular cysts or unrelated pathological structures.

Xenoma formation represents a specific host–pathogen interaction, characterised by abnormal hypertrophy of the host cell. This response is most commonly associated with infection by microsporidians and, in some cases, by intracellular developmental stages of myxozoans, as also observed in the present review. It should be noted, however, that many microsporidians and myxozoans (e.g., *Encephalitozoon*, *Ceratomyxa*, *Myxobolus*) do not induce classical xenomas, despite causing intracellular infections and host–cell or tissue-level hypertrophic lesions that may appear morphologically or clinically similar. Such lesions were not considered xenomas under the operational definition adopted in this review and were therefore excluded.

A related point of contention concerns lesions caused by parasites of the family Xcellidae. These infections, reported in several marine fish species, have historically been described as "xenomas" or "xenoma-like pseudo-tumours". However, ultrastructural and molecular investigations have since shown that X-cell masses consist of proliferating parasite cells rather than hypertrophic host cells harbouring intracellular organisms [22]. This organisation does not conform to the definition of xenoma adopted in the present review, which requires an altered host cell to be the structural unit of the lesion [7]. For this reason, Xcellidae-related lesions were not included in our synthesis. Nonetheless, their frequent misclassification as xenomas in earlier reports illustrates the terminological inconsistency that complicates retrospective analyses and further underscores the importance of establishing and applying clear diagnostic criteria.

A historical overview of microsporidian biology was provided by Franzen [136], while Lom and Dyková [7] focused specifically on the categorisation of microsporidian xenoma

types in fish. Earlier parasitological catalogues, such as the comprehensive compilation by Lom [137], offered valuable inventories of microsporidian diversity, host associations, and infection sites. More recently, Bojko et al. [138] presented an updated taxonomic framework for microsporidian species and synthesised physiological, pathological, and ecological information across a broad range of hosts. However, these publications did not follow formal consensus guidelines for evidence synthesis (e.g., PRISMA or PRISMA-ScR), nor did they evaluate clinical, diagnostic, or pathological features specifically associated with xenoma formation, and several were not centred on xenomas in fish.

In recent years, several guideline-based systematic reviews have focused on microsporidian infections in mammals, including cats, dogs, pigs, rodents, and rabbits [139–144], as well as in humans [145], water and food items [144,146], and birds [144,147]. This growing body of work reflects the increasing interest in microsporidian infections. Nevertheless, to the best of our knowledge, no review to date has applied a PRISMA-ScR framework to investigate microsporidia or xenoma lesions in fish.

The present review complements and extends these contributions by offering the first PRISMA-guided synthesis focused on xenomas of fish as a distinct clinical–pathological entity. By standardising definitions, applying transparent eligibility criteria, and compiling species-specific information into a unified dataset (Supplementary Table S1), this review provides an updated reference for researchers and practitioners. Owing to its broad scope and systematic methodology, it supports improved diagnosis, surveillance, and future research on xenoma-forming infections in both wild and farmed fish.

Xenomas were documented across 113 fish species belonging to 52 families. Microsporidia were the main aetiological agents (96.7% of records), with *Glugea* and *Loma* being the most frequently reported genera. Host specificity varied among genera: some, such as *Spraguea*, were strictly associated with a single fish family (Lophiidae), whereas others, including *Glugea* and *Loma*, infected multiple families. Known preferential associations, such as *G. stephani* in Pleuronectidae, *G. anomala* in Gasterosteidae, or *L. salmonae* in Salmonidae [12,15,148,149], were reflected in our dataset. Although myxozoans are metazoans rather than unicellular parasites, several myxosporean taxa included in this review also exhibit intracellular developmental stages that induce hypertrophic host–cell responses consistent with the operational definition of xenomas adopted here.

Tissue tropism differed among genera. Intestinal xenomas predominated in *Glugea*, gill involvement was most common in *Loma*, and nervous tissue was preferably affected by *Spraguea*. Other genera, such as *Microgemma* and *Microsporidium*, displayed strong organ specificity, typically targeting the liver, while *P. alburnus* was confined to the ovaries of cyprinid hosts. These patterns may guide targeted sampling strategies and contribute to improved diagnostic efficiency.

Reporting of sample sizes and prevalence was inconsistent across studies. Sample sizes were missing in 21% of species-specific records, and prevalence estimates in 18%. When reported, prevalence estimates spanned significantly, ranging from 0.7 to 100%. Such variation reflected multiple factors, including sampling criteria (e.g., “only fish with visible lesions”), capture area, host sex (e.g., xenomas restricted to ovaries), year and season of sampling, and fish age. Prevalence was also influenced by the diagnostic methods used, although only a few studies explicitly compared detection rates across techniques [51]. In some marine fish studies, prevalence was reported to follow seasonal patterns, with higher infection rates during colder months, illustrating the influence of environmental factors on xenoma occurrence [59]. These gaps highlight the need for systematic, standardised reporting to facilitate ecological and epidemiological analyses.

Xenomas occurred in diverse organs, often with multi-organ involvement. Although some studies reported single organs as preferential sites, occasional or erratic localisations

were also documented. Morphological descriptions were highly variable, with lesions ranging from microscopic to macroscopic. The most commonly reported lesions were whitish, spherical, or ovoid, though colour, shape, consistency, and organisation varied widely. Lesions resembling xenomas may, upon further microscopic examination, prove to be of non-parasitic origin, or caused by extracellular parasites, emphasising that clinical examination of fish or gross appearance alone is insufficient for accurate diagnosis.

Histopathological and ultrastructural features also contributed to the morphological variability. Xenomas may be surrounded by host responses, including collagen deposition, fibroblast layers, or inflammatory infiltrates, particularly when spores are released into adjacent tissues. Reported observations include local myositis, necrosis, and macrophage infiltration. The maturation state of xenomas also influences morphology, with mature cysts losing resistance to host immune reactions, which may explain the broad range of reported lesion sizes [12].

Clinical signs were frequently absent or unreported (64% of records). When described, common manifestations included abdominal swelling, lethargy, impaired swimming, and increased mortality. The frequent absence of clinical observations suggests that xenomas may often be subclinical or that historical studies prioritised pathological and ultrastructural findings over behavioural or physiological outcomes. As also noted by Dyková [150], clinical signs and gross lesions typically occur in heavily infected fish.

Diagnostic practices varied widely. Light microscopy, particularly fresh smears, remained the most common method owing to its low cost and rapid turnaround. Semi-thin sections, routine histology, electron microscopy, and molecular techniques were used less consistently. Fully integrated multi-modal diagnostics, combining light microscopy, electron microscopy, and molecular methods, were rare (only 8.7% of records). This finding aligns with longstanding concerns about the limitations of morphology-based microsporidian identification, due to the wide structural variation in spores and developmental stages. Lom and Nilsen [151] highlighted the restricted diagnostic value of morphology for species-level differentiation, and subsequent molecular work has exposed considerable taxonomic instability. Pomport-Castillon et al. [152], for example, reported limited genetic divergence among *G. anomala*, *G. stephani*, and *G. atherinae* based on the molecular markers available at the time, while suggesting a closer affinity of *G. americanus* with *Spraguea lophii*. Subsequent phylogenetic analyses using expanded datasets and additional loci have since clarified species boundaries among these taxa, confirming their distinctiveness [153–155]. Taken together, these discrepancies highlight that a proportion of species descriptions based solely on morphological traits should be interpreted as working hypotheses, which may require reassessment using contemporary integrated approaches.

Molecular diagnostic approaches were applied in a subset of the records included in this review (69/150; 46%). These approaches most often relied on PCR-based amplification, cloning and sequencing or on PCR-based amplification and sequencing, and just one work used whole-genome sequencing [79]. The most amplified regions were conserved ribosomal gene regions, particularly the small subunit ribosomal RNA (SSU rRNA), and in some cases with the Internal Transcribed Spacer (ITS) and the large ribosomal RNA (LSU rRNA), to support genus- or species-level identification of xenoma-associated pathogens. However, the adoption of molecular tools was heterogeneous across studies, and sequence data were not always made publicly available, limiting retrospective comparability and taxonomic re-evaluation.

To improve comparability and reliability, future studies should adopt a minimal integrated diagnostic set, including light microscopy, histology, electron microscopy, and molecular analyses. Standardised reporting of host species, tissue involvement, lesion

morphology, clinical signs, sample size, prevalence, and environmental parameters would facilitate cross-study comparisons and meta-analyses.

Further research should investigate the ecological determinants of xenoma occurrence, host–pathogen specificity, and the influence of environmental variables such as temperature and seasonality. Molecular and cross-infectivity studies are required to resolve taxonomic uncertainties in microsporidian infections and to evaluate potential range expansions or introductions. Sequencing of additional molecular markers, as recommended for *Glugea* and *Spraguea* species [152], could help resolve persistent ambiguities. Longitudinal and multi-organ surveys would provide more accurate assessments of prevalence, tissue distribution, and clinical impact, ultimately supporting fish health management in both wild populations and aquaculture systems.

5. Conclusions

Xenomias represent a widespread but under-characterised phenomenon in fish, with considerable variability in host specificity, tissue tropism, and lesion morphology. While Microsporidia dominate as aetiological agents, other groups, such as myxozoans, are occasionally involved. Future research should aim to standardise methodological approaches, improve reporting of clinical and ecological parameters, and address existing knowledge gaps to advance understanding of xenoma biology and its impact on fish health and management.

Supplementary Materials: The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/parasitologia6010010/s1>, Table S1: Complete data extraction sheet according to PRISMA-ScR guidelines, Table S2: Study-level prevalence and sample size data for xenoma-associated pathogens reported in fish, Supplementary File S3: PRISMA-ScR (Preferred Reporting Items for Systematic reviews and Meta-Analyses extension for Scoping Reviews) checklist for the present scoping review.

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Abbreviations

The following abbreviations are used in this manuscript:

<i>auth</i>	Common or vernacular fish names as provided by the original authors
FW	Fresh-water species
SW	Salt-water species
BW	Brackish-water species
C	Captive-bred specimens
W	Wild-caught specimens

F	Aquaculture farm
A	Aquarium
L	Laboratory setting
S	Single organ affected
M	Multiple organs affected
OTF	Species other than fish
EI	Experimental infections
NID	No clinical or pathological description provided
NX	Not referring to xenoma
OT	Histological diagnosis other than xenoma
SSR	Review papers or book chapters (not describing novel cases)
OL	Overlapping publications
TEM	Transmission electron microscopy
SEM	Scanning electron microscopy
FAO	Food and Agriculture Organization
WoRMS	World Register of Marine Species

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