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Diseases and Parasites of Scallops

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INTRODUCTION

Knowledge of the diseases and parasites of scallops has continued to change since the second edition of this book (McGladdery et al., 2006). Advancements in the field of scallop health research have followed the broader improvements in the ability to identify disease agents in other aquatic animals. Significant progress has been made in diagnostic capability, particularly the ability to distinguish between infectious pathogens and cryptic histopathological lesions in scallops using molecular techniques. Evidence of this is the progress made in combating opportunistic infections of larvae and juveniles of several scallop species reared under hatchery conditions. There are many investigators pursuing the unexplained mortalities in the bivalve hosts using a new generation of diagnostic tools. The diversification of scallop species being brought into stock enhancement and aquaculture programs on a global scale has led to increased contributions from our Asian colleagues. Parallel development of remote underwater photography technology for surveillance and stock assessment purposes as well as suspension culture techniques has greatly improved our capability to detect health challenges in many scallop species, challenges which previously evaded direct observation in the scallop's benthic domain.

The present review follows that of Getchell (1991), dividing sections by taxonomic group of pathogen. The update of these taxonomic divisions attempted to keep up with changes made over the last decade. The listing of pathogens by scallop species also has been continued in Table 10.1, to facilitate scallop-specific cross-referencing. The infectious agents described in detail in the prior two editions remain to ensure as comprehensive a reference as possible. For clarity, a more encompassing definition of the term pathogen has been embraced as a microorganism capable of causing host damage (Paillard et al., 2004). The general approach is to first discuss the aetiology of the scallop disease, then cover the pathogenesis or host syndromes associated with the disease and finally, describe the mode of action of the pathogen (pathogenicity) if known (Paillard et al., 2004). The components of pathogen virulence emphasised are the genetic, biochemical, and structural features that enable it to damage the host.

MICROBIAL DISEASES

Viruses

Prior to 2001, only two viral observations from scallops had been reported. The first was a salmonid pathogen, infectious pancreatic necrosis virus (IPNV). This unenveloped double-strand RNA Birnavirus was found in the king scallop (*Coquille St. Jacques*) *Pecten maximus* (Mortensen et al., 1992, 1998; Mortensen, 1993a) from Norway. The IPNV particles were isolated from hepatopancreas, gonad, kidney, mantle, gill, rectum, and haemolymph preparations of scallops within 24 h of exposure to bath challenges (Mortensen et al., 1992). The virus could still be detected in the hepatopancreas 50 days later, suggesting possible sequestering of the viral particles in this organ. Titres dropped below detectable levels after day 8 in the haemolymph and day 30 from the rectum. No evidence of

TABLE 10.1 Diseases and Parasites of Scallops

Scallop species	Parasite or disease	Effect on scallop	References
<i>Adamussium colbecki</i>	<i>Cibicides refulgens</i> (Foraminifera)	Normally superficial fouling, but penetration of shell can lead to feeding on mantle fluids and scallop weakening	Alexander and DeLaca (1987)
<i>Aequipecten (Chlamys) opercularis</i>	Rickettsial-like organism (Bacteria)	None reported	LeGall et al. (1992)
	Unidentified microsporidian (Protista)	No adverse effects observed, under natural conditions	Lohrmann et al. (1999, 2000b)
	<i>Licnophora auerbachii</i> (Protista)	Histological damage to the eyes	Harry (1980)
	Unidentified apicomplexan	None reported	Kristmundsson et al. (2011b)
	<i>Paranthessius pectinis</i> (Copepoda, Crustacea)	None reported	Reddiah and Williamson (1958)
	<i>Modiolicola inermis</i> (Copepoda, Crustacea)	None reported – common in mantle cavity	Reddiah and Williamson (1958)
	<i>Cibicides lobatulus</i> (Foraminifera)	None reported	Howard and Haynes (1976)
<i>Aequipecten tehuelchus</i>	<i>Suberites ficus</i> ssp. <i>rubrus</i> (Porifera)	None reported – associated with reduced fouling by other colonies	Armstrong et al. (1999)
	<i>Nematopsis</i> sp.	None reported	Cremonte et al. (2005)
	<i>Trichodina</i> sp. (Oligohymenophora, Protista)	None reported	Cremonte et al. (2005)
	<i>Polydora rickettsi</i> (Polychaete)	Mud blisters associated with shell weakening	Diez et al. (2013)
	<i>Tumidotheres maculatus</i> (Pinnotheridae)	Associated with reduced condition index	Narvarte and Saiz (2004)
<i>Amusium balloti</i>	<i>Sulcascaris sulcata</i> (Nematoda)	Associated with brown lesions in adductor muscle	Cannon (1978); Lester et al. (1980)
<i>Amusium pleuronectes</i>	<i>Pinnotheres</i> sp. (Crustacea)	None	Llana (1979)
<i>Argopecten gibbus</i>	<i>Marteilia</i> sp. (Ascetospora, Protista)	Associated with extreme mortalities in SE USA	Moyer et al. (1993, 1995)
	<i>Echeneibothrium</i> sp. (Cestoda)	Associated with gonad atrophy	Singhas et al. (1993)
	<i>Sulcascaris sulcata</i> (Nematoda)	None reported – associated primarily with the gonad	Lichtenfels et al. (1978, 1980), Blake et al. (1984)
	<i>Porrocaecum pectenisi</i> (Nematoda)	Brown discolouration of the adductor muscle (associated with haplosporidian hyperparasite of the encysted nematode)	Cheng (1967, 1973, 1978), McLean (1983)
	<i>Ceratonereis tridentata</i> (Polychaeta)	Pest	Wells and Wells (1962)
	<i>Tumidotheres (Pinnotheres) maculatus</i> (Crustacea)	Mechanical compression of soft tissues (gills, mantle, gonad)	Getchell (1991)
	<i>Odostomium seminuda</i> (Pyramidellidae: Gastropoda)	Mantle retraction	Wells and Wells (1961)

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TABLE 10.1 (Continued)

Scallop species	Parasite or disease	Effect on scallop	References
<i>Argopecten irradians</i>	<i>Vibrio natriegens</i> (Vibrionaceae, Bacteria)	Associated with mass mortalities in adult scallops at a hatchery in China	Zhang et al. (1998)
	Chlamydia-like organisms (Rickettsiales, Bacteria)	Mass mortalities of larvae associated with infections in US hatchery. Mass mortalities of adult scallop in China also associated with digestive gland infections. Infections elsewhere show no clinical effects	Morrison and Shum (1982), Leibovitz (1989), Wang et al. (1998)
	Rickettsial-like organisms (Rickettsiales, Bacteria)	None reported except hypertrophy of the infected cell	Morrison and Shum (1983), Leibovitz et al. (1984), McGladdery et al. (1993a)
	Unidentified haplosporidian (Haplosporidia, Protista)	Associated with high post-spawning mortalities in China, but infection levels were relatively low	Chu et al. (1996)
	<i>Nematopsis duorari</i> and <i>N. ostrearium</i> (Coccidia, Protista)	None reported	Léger and Duboscq (1925) (cited in Sprague, 1970), Kruse (1966), Sprague (1970)
	<i>Pseudoklossia</i> -like sp. (Coccidia)	Associated with adult mortalities under unnatural holding conditions	Leibovitz et al. (1984), McGladdery (1990, 1993a,b), Karlsson (1991), Getchell (1991), Cawthorn et al. (1992), Whyte et al. (1994)
	<i>Perkinsus karlsoni</i> (taxonomic affinity under re-investigation)	Associated with adult mortalities under hatchery holding conditions	McGladdery et al. (1991), Whyte et al. (1993a), Goggin et al. (1996)
	<i>Stichotricha marina</i> (Ciliophora, Protista)	None reported	Hu and Song (2001)
	<i>Proctoeces maculatus</i> (Digenea, Platyhelminthes)	None reported	Karlsson (1991)
	Unidentified metacercaria (Digenea)	None reported	Karlsson (1991)
	Unidentified Echinostome (Digenea)	Focal haemocyte infiltration only	McGladdery et al. (1993a)
	<i>Himasthla quissetensis</i> (Digenea)	None reported	Stunkard (1938)
	Sanguilicolid sporocyst (Digenea)	Castration	Linton (1915)
	<i>Parachristianella</i> sp. (Cestoda, Platyhelminthes)	None reported	Cake (1976, 1977)
	<i>Rhinebothrium</i> sp. (Cestoda)	None reported	Cake (1976, 1977)
	<i>Acanthobothrium</i> sp. (Cestoda)	None reported	Cake (1976, 1977)
	<i>Anthobothrium</i> sp. (Cestoda)	None reported	Cake (1976, 1977)
	<i>Eutetrahyynchus</i> sp. (Cestoda)	None reported	Cake (1976, 1977)
	<i>Tylocephalum</i> sp. (Cestoda)	None reported	Cake (1976, 1977)
	<i>Polypocephalus</i> sp. (Cestoda)	None reported	Cake (1976, 1977, 1979)
	Unidentified turbellarian (Turbellaria)	None reported	Leibovitz et al. (1984)
	<i>Sulcascaris sulcatus</i> (Nematoda)	None reported	Lichtenfels et al. (1978)

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TABLE 10.1 (Continued)

Scallop species	Parasite or disease	Effect on scallop	References
<i>Argopecten (Chlamys) purpuratus</i>	<i>Porrocaecum pectenisi</i> (Nematoda)	Brown discolouration of the adductor muscle (associated with haplosporidian hyperparasite of the encysted nematode)	Cheng (1967, 1973, 1978), McLean (1983)
	<i>Polydora ciliata</i> , <i>Polydora websteri</i> , <i>Polydora</i> spp. (Polychaeta)	Extensive mud blisters associated with shell weakening	Turner and Hanks (1959), Russell (1973), Leibovitz et al. (1984), Karlsson (1991)
	<i>Tumidotheres (Pinnotheres) maculatus</i> , <i>Pinnotheres</i> spp. (Brachyura, Crustacea)	Mechanical compression of soft tissues (gills, mantle, gonad), associated with weakening and weight loss	Cheng (1967), Kryczynski (1972), Karlsson (1991)
	<i>Odostomium seminuda</i> (Pyramidellidae: Gastropoda)	Mantle retraction	Wells and Wells (1961), Leibovitz et al. (1984)
	<i>Prorocentrum</i> sp.	Dinoflagellate toxicity and mechanical damage under hatchery conditions	Leibovitz et al. (1984)
	<i>Zoothorella</i> sp. (Algae)	Green proliferative granulomas in the mantle and tentacles	Leibovitz et al. (1984)
	<i>Scypha</i> sp. (Porifera)	Induced shell deformities	Leibovitz et al. (1984)
	<i>Siroplodium zoophthorum</i> (Oomycete, Eumycota)	Pathogenic to larval scallops	Martin et al. (1997)
	<i>Vibrio</i> spp. (Vibrionaceae, Bacteria)	Hatchery-related mortalities	DiSalvo (1994), Chavez and Riquelme (1994), Riquelme et al. (1995, 1996, 1997, 2000)
	Rickettsial-like organisms (Rickettsiales, Bacteria)	None reported	Lohrmann et al. (2000a, 2002)
	<i>Trichodina</i> sp. (Oligohymenophora, Protista)	None reported	Lohrmann et al. (2000a, 2002, 2009)
	Kidney coccidia (Coccidia, Protista)	Associated with poor-quality gamete production	DiSalvo (1994)
	Hemiurid (Platyhelminthes)	Associated with castration	Mateo et al. (1975)
	Unidentified Digenean metacercariae	Encysted in the palps – no pathology observed	Lohrmann et al. (1991)
	<i>Derogenes varicus</i> (Trematoda, Platyhelminthes)	None reported	Oliva and Sanchez (2005)
	Unidentified Phyllobothriidae (Cestoda, Platyhelminthes)	None reported	Oliva et al. (1986)
	Unidentified Oncobothriidae (Cestoda, Platyhelminthes)	None reported	Oliva et al. (1986)
	Unidentified larval cestode	None reported	Lohrmann and Smith (1993)
	<i>Acanthobothrium</i> sp. and <i>Rhinebothrium</i> sp. (Cestoda, Platyhelminthes)	None reported	Oliva and Sanchez (2005)
	<i>Alexandrium catenella</i> (Dinophyceae)	Melanisation of epithelia	Hegaret et al. (2012)
	<i>Ciona intestinalis</i> (Chordata, Ascidiaceae)	Smothering related mortalities by fouling coverage	Uribe and Etchepare (1999)

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TABLE 10.1 (Continued)

Scallop species	Parasite or disease	Effect on scallop	References
<i>Argopecten ventricosus</i>	<i>Vibrio alginolyticus</i> (Bacteria)	Signs of the disease similar to larval bacillary necrosis	Luna-Gonzalez et al. (2002)
	<i>Vibrio</i> spp. (Bacteria)	Comparatively high TCBS counts in female gonads	Sainz-Hernández and Maeda-Martínez (2005)
	<i>Pseudomyicola spinosus</i> (Copepoda)	None reported	Caceres-Martinez et al. (2005)
<i>Argopecten ventricosus</i> (<i>circularis</i> ssp. <i>aequisulcatus</i>)	<i>Stephanostomum</i> sp. (Digenea, Platyhelminthes)	Black spot lesions – no adverse clinical affects but impacts marketability	Pérez-Urbiola and Martínez-Diaz (2001)
	<i>Echinocephalus pseudoduncinatus</i> (Nematoda)	None reported	McLean (1983)
<i>Chlamys (Mimachlamys) asperrina</i>	<i>Bucephalus</i> sp. (Digenea, Platyhelminthes)	Reduced reproductive potential in heavily infected scallops	Heasman et al. (1996), Hutson et al. (2004)
<i>Chlamys farreri</i> (<i>nipponensis</i>)	<i>Trichodina jadranica</i> (Oligohymenophora, Protista)	None reported	Xu et al. (1995)
	<i>Trichodina</i> sp. (Oligohymenophora, Protista)	None reported	Xiao et al. (2005)
	<i>Stichotricha marina</i> (Ciliophora, Protista)	None reported	Hu and Song (2001)
	<i>Polydora onagawaensis</i> (Polychaete)	Infestation usually light	Sato-Okoshi and Okoshi (2013)
	<i>Pectenophilus ornatus</i> (Copepoda, Crustacea)	Gill hypertrophy at the attachment site of the copepod along with blood-feeding is associated with chronic weakening	Nagasawa et al. (1988, 1991), Nagasawa and Nagata (1992)
<i>Chlamys islandica</i>	<i>Pinnotheres</i> sp. (Brachyura, Crustacea)	None reported	Cheng (1967)
	<i>Gymnophallus</i> sp. (Digenea, Platyhelminthes)	None reported	Chubrick (1966) (cited in Lauckner, 1983)
	<i>Margolisella islandica</i> (Apicomplexa: Eimeridae)	None reported	Kristmundsson et al. (2011a)
<i>Chlamys nobilis</i>	<i>Tylocephalum</i> sp. (Cestoda, Platyhelminthes)	None reported	Sakaguchi (1973) (cited in Laucknerm, 1983)
<i>Chlamys (Pecten) varia</i>	<i>Nematopsis pectinis</i> (Porosporidae, Protista)	None reported	Léger and Duboscq (1925) (cited in Sprague, 1970)
<i>Euvola (Pecten) ziczac</i>	<i>Flavobacterium</i> sp. (Gracilicutes, Bacteria)	Associated with larval mortalities	Lodeiros et al. (1989)
	<i>Pseudomonas</i> sp. (Bacteria)	Associated with larval mortalities	Lodeiros et al. (1992)
	<i>Vibrio</i> spp. (Bacteria)	Associated with larval mortalities	Freites et al. (1993)
<i>Mizuhopecten (Patinopecten) yessoensis</i>	<i>Vibrio splendidus</i> (Bacteria)	Associated with abscesses and lesions on the adductor muscle of adult scallops	Liu et al. (2013)
	Intracellular bacterium (possible Mycoplasma) (Tenericutes, Bacteria)	Associated with weakening and pinkish orange pustules in adult scallops	Bower and Meyer (1991), Bower et al. (1992), Bower and Meyer (1994)
	Rickettsial-like organisms (Rickettsiales, Bacteria)	None reported	Elston (1986), Friedman (1994)

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TABLE 10.1 (Continued)

Scallop species	Parasite or disease	Effect on scallop	References
	<i>Trichodina pectenisi</i> , T. sp. (Oligohymenophora, Protista)	None reported	Lauckner (1983), Kurochkin et al. (1986)
	<i>Nematopsis</i> sp. (Porosporidae, Protista)	None observed	Bower and Meyer (personal communication)
	<i>Perkinsus</i> sp. (Apicomplexa, Protista)	None reported	Kurochkin et al. (1986)
	<i>Perkinsus qugwadi</i> (Scallop Protistan Unknown, SPX) (Perkinsea, Protista)	Associated with mass mortalities and creamy white pustules (especially in gonad, digestive gland, and mantle)	Bower et al. (1990, 1992, 1995, 1997, 1998, 1999), Bower and Meyer (1994), Blackbourn et al. (1998), Itoh et al. (2013)
	<i>Haplosporidium nelsoni</i>	Associated with a mass mortality event	Wang et al. (2012)
	Scallop Protistan 'Ghost' (SPG) (Protista)	Focal lesions with epithelial ulceration in gut, gonoduct, gill, and mantle	Bower et al. (1992, 1994)
	<i>Pseudodistylochus ostreophagus</i> (Turbellaria)	Predation-related mortalities	Bower and Meyer (1994)
	<i>Polydora ciliata</i> (Polychaeta)	None reported	Mori et al. (1985)
	<i>Polydora concharum</i> (Polychaete)	Effects to shell microstructure only reported	Mori et al. (1985), Sato-Okoshi & Okoshi (1993)
	<i>Polydora convexa</i> (Polychaete)	Effects to shell microstructure only reported	Sato-Okoshi and Okoshi (1993)
	<i>Polydora variegata</i> (Polychaete)	Effects to shell microstructure only reported	Mori et al. (1985), Sato-Okoshi and Okoshi (1993)
	<i>Polydora websteri</i> (Polychaete)	Heavy shell infestation can produce clinical soft-tissue effects, but infestation usually light	Kurochkin et al. (1986), Sato-Okoshi and Okoshi (1993), Bower and Meyer (1994)
	<i>Polydora onagawaensis</i> (Polychaete)	Infestation usually light	Sato-Okoshi and Okoshi (2013)
	<i>Polydora brevipalpa</i> (Polychaete)	Heavy shell infestation can produce clinical soft-tissue effects	Sato-Okoshi and Okoshi (2013)
	<i>Pectenophilus ornatus</i> (Copepoda, Crustacea)	Gill hypertrophy at copepod attachment, plus blood-feeding associated with chronic weakening	Nagasawa et al. (1988, 1991), Nagasawa and Nagata (1992)
	<i>Cliona</i> sp. (Porifera)	Heavy shell infestation can produce clinical soft-tissue effects, but infestation usually light	Kurochkin et al. (1986)
	<i>Eutima japonica</i>	Heavy polyp load associated with juvenile mortalities	Baba et al. (2007)
	Pollutant effects	Weakening and muscle atrophy, necrosis. Gametogenesis abnormalities	Syasina et al. (1996), Usheva (1999)
	Copper and Zinc toxicity	Embryo toxicity reported	Karaseva and Medvedeva (1994)
	Mussel fouling	Smothering suspended scallops	Kurata et al. (1996)
	<i>Solidobalanus hesperius</i> (Cirripeda, Crustacea) fouling	Smothering	Ovsyannikova and Levin (1982)
<i>Pecten alba</i>	<i>Bucephalus</i> -like trematode (Digenea, Platyhelminthes)	Castration by gonad displacement	Sanders and Lester (1981)
<i>Pecten</i> (<i>Patinopecten</i>) <i>caurinus</i>	<i>Nematopsis</i> sp. (Porosporidae, Protista)	None observed	Bower and Meyer (personal communication)

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TABLE 10.1 (Continued)

Scallop species	Parasite or disease	Effect on scallop	References
<i>Pecten fumatus</i>	<i>Bucephalus</i> -like trematode (Digenea, Platyhelminthes)	Castration by gonad displacement	Heasman et al. (1996), Hutson et al. (2004)
	<i>Sabella spallanzanii</i> (Polychaeta)	Fouling-related mortalities	Clapin and Evans (1995), O'Connor et al. (1999)
<i>Pecten maximus</i>	Infectious Pancreatic Necrosis Virus (Finfish virus)	None reported	Mortensen (1993a), Mortensen et al. (1992, 1998)
	Ostreid Herpesvirus-1var	Larval mortalities	Arzul et al. (2001)
	<i>Vibrio pectenicida</i> (Vibrionaceae, Bacteria)	Larval mortalities	Lambert and Nicolas (1998), Nicolas et al. (1996), Lambert et al. (1999a), Sandlund et al. (2006)
	<i>Vibrio splendidus</i> (Bacteria)	Brown inner shell deposits in broodstock	Lambert et al. (1999b)
	<i>Vibrio splendidus</i> (Bacteria)	Larval mortalities	Torkildsen et al. (2005), Sandlund et al. (2006)
<i>Pecten novaezealandiae</i>	Rickettsial-like organisms (Rickettsiales, Bacteria)	Associated with mass mortalities in France	Comps (1983), LeGall et al. (1988, 1991, 1992), LeGall and Mialhe (1992), Kellner-Cousin et al. (1993)
	<i>Pseudoklossia pectinis</i> (Coccidia, Protista)	None reported	Leger and Duboscq (1917)
	Unidentified apicomplexan	None reported	Kristmundsson et al. (2011b)
	<i>Polydora</i> sp. (Polychaete)	Shell damage and associated spat mortalities	Mortensen et al. (1999)
	<i>Pomatoceros triquetus</i> (Polychaete)	None reported	Burnell et al. (1995)
	<i>Modiolicola maxima</i> (Copepoda, Crustacea)	None reported	Reddiah and Williamson (1958)
	<i>Modiolicola</i> sp. (Copepoda, Crustacea)	None reported	Mortensen (1993b)
	Mussel fouling	Smothering related mortalities	Minchin and Duggan (1989)
	Unknown environmental disturbances	Abnormal melanisation and microstructural distortions of the shell	Larvor et al. (1996)
	Virus Like Particles (VPL) (Virus)	Necrotic digestive gland epithelial cells with associated mortalities	Hine and Wesney (1997)
<i>Placopecten magellanicus</i>	<i>Paravortex</i> sp. (Turbellaria)	Associated with weak and dying scallop but direct clinical effect unclear	Woods and Hayden (1998)
	Bacterial Abscess Disease or brown spot disease (various Bacteria)	Abscess lesions in adductor muscle	Sherburne and Bean (1986), McGladdery (1990), Getchell (1991)
	Rickettsial-like organism (RLO) (Rickettsiales, Bacteria)	Associated with adult mortalities in the eastern United States, but none reported in other cases	Ballou (1984), Gulka et al. (1983), Gulka and Chang (1984), Leibovitz et al. (1984)
	Chlamydial-like organism (Rickettsiales)	None reported apart from slight cell hypertrophy	Morrison and Shum (1982)
	<i>Trichodina</i> sp. (Oligohymenophora, Protista)	None reported	Beninger et al. (1988), McGladdery et al. (1993a), McGladdery and Stephenson (unpublished data)
	Unidentified digenetic (Platyhelminthes)	None reported	McGladdery et al. (1993a)

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TABLE 10.1 (Continued)

Scallop species	Parasite or disease	Effect on scallop	References
	<i>Urastoma</i> -like turbellarians (Turbellaria)	None reported	McGladdery et al. (1993a)
	<i>Polydora concharum</i> (Polychaete)	Shell damage with severe infestations	Evans (1969), Blake (1969)
	<i>Polydora socialis</i> (Polychaete)	None reported	Blake (1969)
	<i>Polydora websteri</i> (Polychaete)	Shell erosion and weakening in extreme infestations may render scallop more susceptible to predation	Evans (1969), Bergman et al. (1982)
	<i>Dodecaceria concharum</i> (Polychaete)	Enlarges <i>Polydora</i> tunnels, increasing shell damage	Evans (1969), Leibovitz et al. (1984)
	<i>Pseudopotamilla reniformis</i>	Secretion of large ridges of shell material to contain worms up to 100 mm in length	Blake (1969)
	Pea Crabs (Crustacea)	Compression of gonad possibly affecting gonadal development	Cheng (1967), Karlsson (1991), Getchell (1991)
	<i>Coccomyxa parasitica</i>	Green discolouration of the mantle and associated granulomas in scallops over 3 years old	Naidu (1971), Stevenson and South (1975)
	<i>Cliona celata</i> ; <i>Cliona vastifica</i> (Porifera)	None reported	Medcof (1949), Evans (1969), Sindermann (1971), McGladdery et al. (1993a)
	<i>Hydractinia echinata</i> (Cnidaria)	If attached to internal edge of shell may induce shell deformities	Merrill (1967)
	Mussels	Fouling-related mortalities	Claereboudt et al. (1994), Gryska et al. (1996)
	Cadmium and Copper	Larval toxicity	Yevich and Yevich (1985)
<i>Placopecten meridionalis</i>	<i>Polydora websteri</i>	Formed mud blisters between the shell and mantle	Skeel (1979)

viral replication or pathological effects was found in the scallop tissue; however, it is believed that these scallops may play a significant role as reservoirs of viable IPNV in the natural environment (Mortensen et al., 1992). Other challenges using an orthomyxovirus-like (enveloped single-strand RNA) virus, responsible for infectious salmon anaemia showed no evidence of uptake or carriage by the same scallop species (Bjoersholt et al., 1999).

The other observation of 'virus-like particles' was reported from the New Zealand scallop, *Pecten novaezelandiae*, and was associated with mortalities of up to 39% (Hine and Wesney, 1997). The toheroa clam, *Paphies ventricosum*, from independent mortalities, was found to carry similar viral-like particles. Light microscopy of diseased scallops revealed necrotic digestive gland epithelial cells, associated with sloughed or pyknotic cells in the tubule lumens. Some sloughed cells contained abnormal granular cytoplasmic inclusions, which were DNA-negative. Transmission electron microscopy (TEM) revealed electron dense, unenveloped virus-like particles (22–30 nm in diameter) in the cytoplasm adjacent to the outer nuclear membrane, or in orderly arrays along the cisternae of highly modified endoplasmic reticulum (ER) of digestive cells. Some cisternae were dilated to form vacuolar inclusions. Secretory cells showed similar viral-like particle arrays with dilated ER but rarely vacuolar inclusions. The ribonucleic acid component, along with the close association with the rough ER of infected cells, suggests an enterovirus-like (Picornaviridae) or calicivirus affiliation; however, these characteristics are indicative of a new group of viruses from molluscs and require more examination in order to determine viral family affinities and their precise role in the mortalities observed.

Ostreid Herpesvirus-1

Farley et al. (1972) made the initial finding of herpes-like viral infections in the eastern oyster, *Crassostrea virginica*, by TEM. After several other bivalve species were documented with herpesviruses during the 1990s, the king

scallop, *Pecten maximus*, became the first scallop species exhibiting high mortalities in association with cellular lesions and viral particles in the connective tissue of moribund larvae collected at 7 or 10 days old (Arzul et al., 2001). PCR amplification and DNA sequencing of the products from moribund scallop larvae showed high similarity to Ostreid Herpesvirus-1 (OsHV-1) and near identity with a variant of OsHV-1 (OsHV-1var), which had been shown to infect *Crassostrea gigas* and Manila clam larvae, *Ruditapes philippinarum*. Intraspecies and interspecies transmission of OsHV-1var also was demonstrated by exposure of *P. maximus* and *C. gigas* larvae to extracts from moribund *P. maximus* larvae (Arzul et al., 2001). Five days after exposure, 100% of the *P. maximus* larvae were dead. Inspection using TEM revealed intracellular particles in these larvae (Arzul et al., 2001).

Amplification of nucleic acid by PCR has made the rapid diagnosis of viral infections in shellfish a reality (Renault and Arzul, 2001; Batista et al., 2007). There are caveats to this statement, and caution must always be exercised because the detection of viral DNA could be due to contaminants from the digestive tract as well as carrier, latent, low-grade persistent, or active infections. The presence of inhibitors that interfere with DNA extraction also can create false-negative PCR results. Since it is still not possible to culture molluscan viruses due to the lack of continuous cell lines, confirmation with other techniques such as immunochemistry and TEM is necessary. For example, Arzul et al. (2001) documented all the microscopic features of productive viral replication in scallop larvae in addition to conducting PCR analysis and *in situ* hybridisation. Hybridisation signals were seen in positive adult *P. maximus* in the connective tissues of gills, gonad, mantle, and muscle.

Acute Viral Necrosis Virus

During the mid-1990s, repeated mortality events occurred in the summer among farms culturing *Chlamys farreri* along the northern coast of China. Losses approached 90% in 1- to 2-year-old scallops within 5–8 days once an outbreak started with necrotic lesions in the gills, mantle, kidney, and digestive gland (Guo et al., 1999; Wang et al., 2007). Song et al. (2001) identified acute viral necrosis virus (AVNV) as the causative agent of these mortalities. Tang et al. (2010) suggested a correlation between elevated seawater temperature and the AVNV-infection associated *C. farreri* mortalities and demonstrated that the viral infection provoked multiple physiological and immune responses in the host scallops. Typical gross signs in affected scallops included a loss of response to stimuli, excessive mucus on the viscera, and shrunken mantle (Wang et al., 2007).

Other Viruses

Other viruses or viral-like particles have been documented from a number of other bivalve species, with increasing frequency, since the first edition of this volume (Hine et al., 1992; Comps and Cochennec, 1993; Norton et al., 1993; Elston, 1997; Hine et al., 1998; Comps et al., 1999; Miyazaki et al., 1999; Meyers et al., 2009). Many of these more recent investigations have even been successful in determining biochemical characteristics that aid in classification of the viruses under investigation, for example, the *Herpes*-like viruses of the Pacific oyster, *Crassostrea gigas* (Le Deuff and Renault, 1999). Viral detection is likely to increase with better access to TEM; however, the persistent lack of self-replicating cell lines for isolation of marine bivalve (and other invertebrate) viruses continues to hamper investigation of diseases with possible viral aetiology. Though Getchell (1991) previously stated ‘With the imminent development of marine invertebrate cell cultures, the coming decade should be one of rapid expansion of knowledge about viruses of marine animals (Sindermann, 1984), including scallops’, research into such techniques has been limited, compared to that for finfish and terrestrial cell-lines. The need for a continuous molluscan cell-line still exists. Until such techniques are developed, scallop (and other aquatic invertebrate) viruses will remain difficult to detect and many infectious diseases of ‘unknown aetiology’ will continue to defy accurate diagnosis. Use of finfish cell lines to isolate viruses from molluscs must also be treated with due caution, since these have been shown to detect pathogens of finfish (and other contaminants) sequestered by the molluscs, rather than obligate viruses requiring molluscan cells for replication (Hill et al., 1986; Mortensen et al., 1992).

Prokaryota

The most commonly recorded bacterial infections and diseases of scallops are attributed to members of the Gram-negative Vibrionaceae and Rickettsiales (intracellular Rickettsia and Chlamydia). As noted by Getchell (1991), these organisms are not usually harmful to adult scallops, except where bacterial concentrations exceed levels found under natural conditions. This is consistent with the effect of the same and related bacteria on other bivalve species, especially under culture conditions (Elston, 1984).

Vibrionaceae

Leibovitz et al. (1984) list three bacterial diseases of cultured *Argopecten irradians*: (i) bacterial swarming, (ii) bacillary necrosis, and (iii) vibriosis. The overwhelming majority of bacteria associated with marine bivalves are Gram negative. Due to their efficient filtering mechanism, bivalves are able to accumulate large numbers of microorganisms from the surrounding seawater and thus harbour a rich bacterial flora. The role of bacteria as organisms causing disease in bivalves is not always clear. Species of *Pseudomonas* (*Photobacterium*) and *Vibrio* are natural constituents of the molluscan digestive tract, and yet species of these genera have been shown to cause most bacterial diseases of affected pelecypods. The pathogenicity of bacteria for bivalves appears to be negatively correlated to age of the mollusc, decreasing, as they grow older (Lauckner, 1983). Studies by Lambert and Nicolas (1998) have clearly demonstrated a direct inhibition of haemocyte chemiluminescent (CL) activity (associated with intracellular degradation of phagocytosed particles) in both *Pecten maximus* and *Crassostrea gigas*, when challenged with several strains of *Vibrio* spp. and *Alteromonas* spp. Bacterial strains associated with mortalities of oyster larvae produced less CL inhibition in scallop haemocytes than in oysters and vice versa. This is consistent with other observations where bacterial strain and species can be clearly identified and host specificity examined (Nicolas et al., 1996). Although still recognised as principally a larval problem, exceptions are beginning to emerge for adult bivalves, including scallops (Zhang et al., 1998).

Under hatchery conditions, *Vibrio* most commonly attacks scallops at the velar stage of their development. Susceptibility of cultured scallop larvae to *Vibrio* has been demonstrated in several scallop species such as *Argopecten ventricosus* (Luna-Gonzalez et al., 2002), *Euvola* (*Pecten*) ziczac (Freites et al., 1993), *A. purpuratus* (Riquelme et al., 1995), *A. irradians* (Tubiash et al., 1965), and *P. maximus* (Nicolas et al., 1992, 1996). The role of similar infections in wild population dynamics is poorly understood; however, hatchery holding of high concentrations of larvae in flow-through systems, often fed by waters at higher than ambient temperature to accelerate growth and feeding, appear conducive to bacterial 'blooms' and subsequent mortality events (Elston 1984). Any physiological stress due to crowding, feed detritus accumulation and rapid water quality changes (pH, oxygen, turbidity, etc.) should, therefore, be regarded as a potential trigger for bacterial proliferation and bacillary necrosis.

Tubiash et al. (1965) determined the pathogenicity of various bacteria by inoculating 24 h suspensions of the test organism into 400-mL cultures of 2- to 7-day-old larvae. Host susceptibility was determined in a similar fashion, with *Argopecten irradians* larvae as well as other bivalves being challenged with an isolated bacterial strain of high virulence. The Gram-negative bacilli were obtained from dead and moribund bivalve larvae. The disease noted was termed 'bacillary necrosis'.

The course of the disease was swift and dramatic. Early signs, 4–5 h after inoculation with the bacterial pathogen, included a reduction of motility and the presence of many larvae lying on the bottom with either their rudimentary foot or velum extended. With this quiescent behaviour came the spreading of 'swarms' of bacteria from separate foci within the culture. After 8 h, death began to appear, with widespread granular necrosis of the tissues. Examination by light microscopy revealed massive invasion by bacteria throughout the larval tissues. In a heavily infected larval culture, mortality often reached 100% within 18 h. Ciliated protistans appeared as secondary invaders at the height of the epizootic, possibly feeding on the bacteria. Larval *A. irradians* experienced 100% mortality after 12 h when challenged with Tubiash's pathogen M 17, presumably a species of *Vibrio*.

Guillard (1959) also described the course of bacterial infection of bivalve larval cultures. The mechanism by which the bivalve larvae were destroyed was suggested to be by invasion or at least contact, rather than by a bacterial exotoxin. Tubiash et al. (1965, 1970) concluded that the entry of the pathogen was via the alimentary tract because malformed non-feeding larvae were the last to show signs of the infection. Finally, Brown and Losee (1978) have suggested that bacterial pathogens attach to and penetrate the velum and presumably spread from this site.

Numerous studies have described hatchery based vibriosis problems in the Chilean scallop, *Argopecten* (*Chlamys*) *purpuratus*, from northern Chile (Chavez and Riquelme, 1994; DiSalvo, 1994; Riquelme et al., 1995, 1996, 1997, 2000). These authors found the predominant bacteria to be *Vibrio anguillarum*-like strains. Challenges with cell-free supernatant prepared from bacterial cultures reduced larval scallop survival indicating a possible endotoxin effect, rather than bacterial penetration of the tissues. Mortalities were most rapid and significant at water temperatures of 25 °C. Similar results were found with *Pseudomonas* sp. and *V. anguillarum* infections of larval scallops *Euvola* (*Pecten*) ziczac from Venezuela (Lodeiros et al., 1989, 1992).

Larval *Pecten maximus* in scallop hatcheries in France also have suffered mass mortalities due to *Vibrio*. Although vibriosis has been a common feature of *P. maximus* production for over 15 years in Brittany hatcheries, only recently has differentiation and identification of a new species of *Vibrio* (related to the *Vibrio splendidus*

group) been possible via immunoassays and nucleic acid sequencing (Nicolas et al., 1996; Lambert and Nicolas, 1998; Lambert et al., 1999a). Torkildsen et al. (2005) also isolated bacterial strains associated with *P. maximus* mortalities at a Norwegian hatchery. Among 100 isolates, they found a cluster of *V. splendidus* strains and a strain of *Alteromonas/Pseudoalteromonas* that produced losses similar to a known pathogen (*Vibrio pectenicida*) during bath challenge tests with 10- to 16-day-old larvae (Sandlund et al., 2006). Phenotypic characterisation of six of these strains gave the same groupings as that obtained by 16S rDNA sequencing (Torkildsen et al., 2005). The *Pseudoalteromonas* sp. isolate LT-13 was also detected by PCR during *P. maximus* larval mortalities, and the investigators concluded the bacterium was not an obligate pathogen, but an opportunist (Sandaa et al., 2008).

Interestingly, another *V. splendidus*-related strain was isolated from brown inner shell deposits in 3-year-old *P. maximus* broodstock, showing signs of debilitating disease. Although challenges with isolates from these shell deposits reproduced the disease, identical isolates from control scallops as well as inlet water precluded determination of the exact cause–effect relationship (Lambert et al., 1999b). Infection by another vibrio, *Vibrio tapetis*, was also associated with brown shell deposits and high levels of mortality in the clam species *Tapes decussatus* and *T. philippinarum* in Atlantic Europe (Nicolas et al., 1992; Borrego et al., 1996).

Another documented case of mortalities of adult scallops attributed to *Vibrio* infection was a report from China, where adult *Argopecten irradians* suffered mass mortalities in a hatchery in Shandong Province in 1996. Bacteria isolated from the kidney of one of the affected scallops was characterised serologically as *Vibrio natriegens* and challenge experiments using this isolate reproduced the clinical signs observed during the initial outbreak, namely digestive tract and kidney haemocyte infiltration, along with gonad and mantle atrophy (Zhang et al., 1998). Most recently, a strain of *Vibrio splendidus* that was virulent at low temperature and shown to cause a fatal disease with adductor muscle lesions in the yesso scallop *Patinopecten yessoensis* has been reported in northeastern Chinese culture facilities (Liu et al., 2013). Moribund scallops from one of these commercial farms were sampled and 96 bacterial isolates were characterised, including 88 strains of *Vibrio*. The predominant strain (43/88) was *Vibrio splendidus* and the pathogenicity of the JZ6 strain was established with both injection and immersion challenges causing 80% and 43% mortalities, respectively. The lesions in the challenged scallops were identical to the original moribund specimens as confirmed by histopathology (Liu et al., 2013). As mentioned above, *Vibrio* spp. have commonly been associated with disease outbreaks during the warmer months of the year when water temperatures are more than 20 °C (Lacoste et al., 2001); however, the JZ6 strain described above was a cold-adapted *Vibrio* with high haemolytic capabilities, caused massive mortalities of yesso scallops at 10 °C and fulfilled all four of Koch's postulates.

Intracellular Prokaryotes (*Rickettsiales*, *Chlamydiales*, and *Mycoplasma*)

Obligate intracellular prokaryotes, notably the Rickettsiales and Chlamydiales, are commonly found in the epithelial cells of the gills and digestive diverticula of a wide range of bivalves, including scallops (Chang et al., 1980). Most infections appear benign, despite relatively dense colonisation; however, Leibovitz (1989) reported chlamydiosis as a serious disease of larval and post-metamorphic bay scallops, *Argopecten irradians*, in a hatchery, and Morrison and Shum (1982) described a chlamydia-like organism from the digestive diverticula of older juveniles and adults of the same species. The presence of reticulate and elementary bodies within the inclusions in infected epithelial cells indicated that the pathogen was more related to the Chlamydiales than Rickettsiales, since the latter possess a single developmental stage compared with at least two forms that comprise the chlamydial life cycle. About 40% of the bay scallops examined were infected, as well as many sea scallops, *Placopecten magellanicus* (Morrison and Shum, 1982). Basophilic staining (haematoxylin and eosin) spherical, or sub-spherical, inclusion bodies were seen within intracellular vacuoles in the epithelial cells of the blind-ending tubules of the digestive diverticula. The inclusions varied in size and in number. Heavy infections were associated with similar inclusion bodies in the kidney epithelia. Morrison and Shum (1982) suggested that the initial invasion could have occurred by endocytosis of the elementary bodies by the tubule epithelial cells (which are responsible for nutrient absorption and waste disposal via endocytosis and pinocytosis, respectively). Since the infections were sometimes heavy and some degeneration of host cells was present, these authors believed that the pathogen should have some deleterious effects on the scallop host, but none was evident. Other bivalves present had a much lower prevalence of infection than either of the two scallop species. A recent investigation of mass mortalities of bay scallops, introduced to Japan for culture purposes, revealed high levels of a similar chlamydial-like infection of the digestive diverticula. This was directly attributed to the mass mortalities by the investigating authors (Wang et al., 1998); however, the precise linkage between infection and pathogenicity remains unclear.

Observations by [Morrison and Shum \(1983\)](#) showed a similar infection in the kidney epithelia of F1 and F3 generations cultured from wild US bay scallops, *Argopecten irradians*, and kept in quarantine in Prince Edward Island, Canada. They identified the agent as a rickettsial-like organism, due to the lack of different development stages characteristic of chlamydiales. The kidney rickettsial-like colonies were characterised by separate membrane-bound vacuoles within the infected cell cytoplasm. The prokaryotes multiplied by binary fission and could expand colony size to a scale that induced hypertrophy of the infected cell.

[Gulka et al. \(1983\)](#) reported a mass mortality of sea scallops, *Placopecten magellanicus*, in Narragansett Bay, Rhode Island. Gross clinical signs of infection included retracted mantles and greyish, flaccid adductor muscles, similar to the pathology described by [Medcalf \(1949\)](#), which he had associated with ageing scallops and heavy shell infestation by clionid sponges and polychaete worms (*Polydora*). Affected scallops were of varying ages and demonstrated little shell infestation. Histologically, there were myodegenerative changes in the adductor muscle and intracellular basophilic inclusion bodies within the epithelial cells of the gill, plicate membrane, and other body surfaces. Heavily infected tissues, especially the gills, often lost normal tissue architecture. Ultrastructural investigation showed that the infectious prokaryote cells possessed a thin cell wall, and measured 1.9–2.9 µm × 0.5 µm. Uniformity of structure supported tentative assignation to the rickettsial-like intracellular organisms. There was strong correlation between heavy infections and adductor muscle degeneration, described as 'grey muscle disease' ([Ballou, 1984](#)), which the authors postulated was due to gill dysfunction precipitating metabolic stress and pathological changes in the muscle tissue.

In their second examination of this prokaryotic infection, [Gulka and Chang \(1984\)](#) transplanted uninfected *Placopecten magellanicus* to cages in Narragansett Bay. Within 2.5–3.5 months, up to 53% of the transplants became infected. Scallops held in aquaria using the same water were 100% infected after 5 months. Mortality in the caged population was substantially higher than in scallops maintained in the laboratory. Inoculation challenges using homogenates of infected gill tissue resulted in heavy infections of gill tissue within 20–25 days. Attempts to infect the blue mussel, *Mytilus edulis*, and soft-shell clam, *Mya arenaria*, were unsuccessful. Attempts to culture the pathogen also failed, except in gill tissue preparations or via direct inoculation of homogenates into *P. magellanicus*; however, both field and laboratory challenges during this study failed to reveal the adductor muscle degenerative changes associated with the earlier investigation. This weakens the suggested linkage between the rickettsial infections and mortalities of *P. magellanicus* in Narragansett Bay. [McGladdery et al. \(1993a\)](#) also reported intracellular basophilic inclusion bodies in other sea scallops, with no apparent clinical effect.

Light-to-moderate rickettsial-like infections of the gills ([Figure 10.1](#)) also were found in wild, captive, and cultured adult bay, *Argopecten irradians*, and sea scallops, *Placopecten magellanicus*, by [Leibovitz et al. \(1984\)](#). In these cases, no significant mortality was detected and larval and juvenile scallops remained free of infection. The organisms were morphologically similar to the prokaryotes in the gills of *P. magellanicus* in Rhode Island.

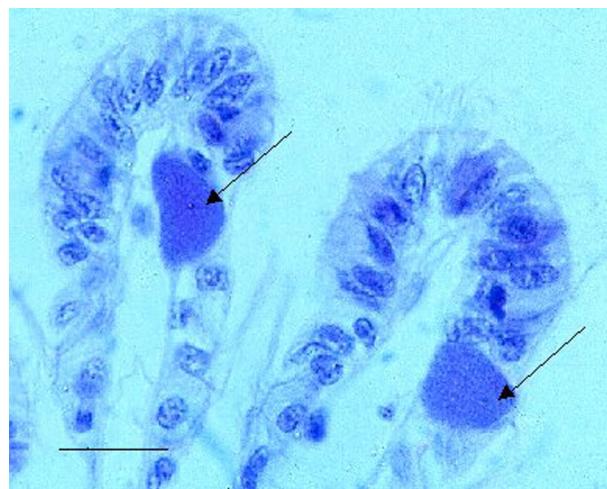


FIGURE 10.1 Rickettsial-like microcolonies (arrows) in the gill epithelia of *Placopecten magellanicus* (sea scallop). Haematoxylin and eosin stain; scale bar=30 µm.

(Gulka et al., 1983) and those found in other molluscs by Comps (1983), but differed from the rickettsial-like organisms described from the kidney of bay scallops by Morrison and Shum (1983).

The apparent lack of disease in some cases of rickettsial-like infections in sea scallops, compared with extreme pathogenicity in other cases is a conundrum, which occurs in other pectinids; such as *Aequipecten* (*Chlamys*) *opercularis* (LeGall et al., 1992), *Argopecten purpuratus* (Lohrmann et al., 2000a, 2002), *Mizuhopecten* (*Patinopecten*) *yessoensis* (Elston, 1986; Friedman, 1994), and *Pecten maximus* (Comps, 1983; LeGall et al., 1988, 1991, 1992; LeGall and Mialhe, 1992; Kellner-Cousin et al., 1993). However, McGladdery (1998) noted that similar organisms have been clearly linked to pathogenic infections in other molluscan species. An exacerbatory effect under sub-optimal growing conditions, as suggested by some larval mortality under hatchery conditions, is possible. In addition, distinguishing between species, with no marine invertebrate cell line to assist in isolation and purification, complicates differentiation between pathogenic and non-pathogenic strains. Advances with immuno- and nucleic acid labelling, however, show great promise for overcoming this diagnostic challenge (LeGall et al., 1992).

The occurrence of an interesting third group of intracellular prokaryotes – the Mycoplasma (formerly known as the Mollicutes) has been speculated in association with a pustule disease affecting Japanese scallops, *Mizuhopecten* (*Patinopecten*) *yessoensis*, on the Pacific coast of Canada (Bower and Meyer, 1991, 1994; Bower et al., 1992; Bower, 1998). Complaints of pinkish orange pustules up to 10 mm in diameter in the soft tissues (including the adductor muscle) and conchiolin-lined shell erosions along the edge of the shell were investigated and revealed the presence of intracellular prokaryotes in a few haemocytes within abscess lesions or other foci of haemocyte infiltration (Figure 10.2).

Pustule lesions may become encapsulated and necrotic. Under ultrastructural examination, a few infected haemocytes and autophagy of infected haemocytes were detected. Diagnosis is speculative because intracellular prokaryotes could result from the phagocytosis of bacteria from secondary infections and such bacteria could resemble Mycoplasma especially because few infected haemocytes were available for examination; however, the lesions appeared to be aseptic apart from the few haemocytes containing prokaryotic organisms. The lesions occurred in Japanese scallops from six grow-out localities experiencing poor growth and mortalities in 1989. The condition appeared to be related to sub-optimal culture practices and has not reappeared in subsequent years.

Other Bacterial Pathogens of Scallops

Another adductor muscle abscess condition, called bacterial abscess disease or brown spot disease, has been reported from sea scallop, *Placopecten magellanicus*, from several beds along the Maine and Atlantic Canadian coasts. First observed from Harpswell Sound in 1977, this disease was noted in Muscongus Bay in 1979, Damariscotta River in 1980, and St. Croix River in 1981 (Sherburne and Bean, 1986). Scallops examined from the Damariscotta River during 1985–1987 had similar mucoid abscess lesions 1–2 mm in diameter scattered throughout the muscle. Five to ten per cent of the larger specimens (mean shell height=124 mm) were infected at two sampling stations in the Damariscotta River. Gram-positive pleomorphic bacteria were consistently observed from the necrotic foci and morphologically similar bacteria were seen in histological sections prepared from infected adductor muscle, gonad, and kidney tissue (Getchell, unpublished data). Attempts to culture this probable pathogen failed. Similar observations in scallops collected off southwest Nova Scotia and Cape Breton yielded

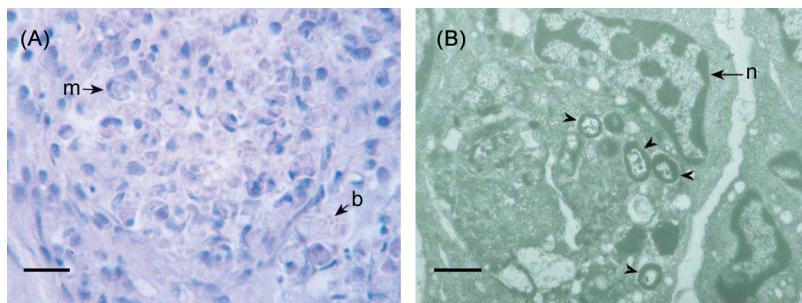


FIGURE 10.2 (A) Histological section through a pustule in *Mizuhopecten* (*Patinopecten*) *yessoensis* consisting of haemocytes at various stages of necrosis including a haemocyte (m) that appears to be engulfing necrotic tissue and another associated with a colony of intracellular prokaryotes (b). Haematoxylin and eosin stain; scale bar = 15 µm. (B) Electron micrograph of a pustule showing a healthy haemocyte (n indicates its nucleus) which appears to have engulfed a necrotic haemocyte that is infected with prokaryotes (arrows). Uranyl acetate and lead citrate stain; scale bar=1 µm.

bacterial cultures containing both Gram-positive, as well as Gram-negative bacterial species (McGladdery et al., 1993a; McGladdery, 1998). Secondary infections probably complicated isolation and identification of the primary cause. Similar to the Japanese scallop pustule disease case, there is an apparent correlation with sub-optimal growing conditions that may trigger development of the 'brown spot disease' condition in sea scallop. Many sea scallops showed extensive shell damage by polychaetes *Polydora* spp. (mainly *P. concharum*) and clionid sponges, including perforation of the adductor muscle attachment site. In Maine, many affected scallops came from estuarine beds subject to wide salinity fluctuations that could compromise scallop condition and their ability to remove entrained sediment resulting in irritation of the soft tissues. Obviously, this condition requires more investigation.

In 2012, during dredging design experiments, orange/pink bacterial abscesses were noted in a low numbers of adult *Placopecten magellanicus* fished from Georges Bank and adjacent areas off the east coast of the United States (Figure 10.3). Irregularly shaped abscesses of 2 cm diameter were primarily noted in the adductor muscle, but smaller variable numbers of abscesses (1 cm and less) could be identified in the gonad and soft tissues surround the digestive gland. Microscopic evaluation and acid-fast staining showed abundant rod-shaped acid-fast positive bacteria consistent with *Mycobacteria* sp. in the abscesses. Subsequent molecular identification, using three different primer sets, confirmed the genus designation but suggested a new un-named mycobacterial species (Smolowitz, unpublished data).

Bacterial Management Under Hatchery Conditions

Antibiotic preparations may offer a practical short-term means of control for bacillary necrosis in cultured scallop larvae, however, such practices are not recommended for flow-through systems or chronic/prophylactic bacterial management. In the United States, the use of drugs as a disease preventative to treat larvae is not approved by the FDA. Additionally, scallop larvae may use many bacteria as a nutritional supplement (Samain et al., 1989; Moal et al., 1996), and rapid development of resistance to many antibiotics is now well documented for a number of aquatic bacteria, especially *Vibrio* spp. (Alderman and Barker, 1997). Revisions to government approved antibiotic use in seawater facilities have further restricted their application (Robert et al., 1996). Chloramphenicol was one of the first therapeutic agents banned in the production of organisms intended for human consumption and alternatives such as florfenicol, oxytetracycline, oxolinic acid, and neomycin were studied (Torkildsen et al., 2002). Chilean scallop hatcheries are currently using florfenicol in their larval cultures of *Argopecten purpuratus*, adding it to the rearing water at 2 mg per litre every 48 h after the fifth day of culture (Miranda et al., 2014). In addition, seawater appears to have properties which are antagonistic to the efficacy of some antibiotics (Torkildsen et al., 2000).

Another way to avoid difficulties with bacillary necrosis is to optimise water quality and maintain vigilant monitoring of influent pipes, algal food sources, and tank surfaces. Every hatchery and laboratory has its own unique set of challenges and must establish its own controls, standards, and preventative measures. Brown and Russo (1979) found that a combination of filtration and ultraviolet irradiation of seawater reduced the occurrence of bacterial diseases. Brown (1981) noted, however, that many shellfish hatcheries do not use any disinfection methods and continue to be plagued by intermittent occurrences of bacteria-related diseases that often destroy larval

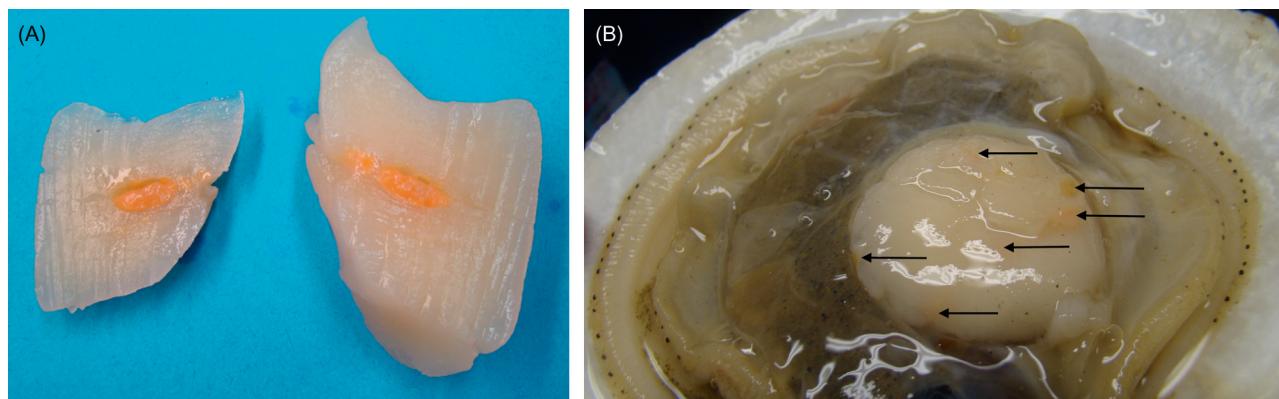


FIGURE 10.3 (A) Localised mycobacterial nodule in the adductor muscle of the sea scallop *Placopecten magellanicus*. (B) Multiple mycobacterial nodules in the adductor muscle of the sea scallop *Placopecten magellanicus*.

cultures around the sixth day of development. It is essential to determine how the pathogens enter the facility so that effective prophylactic measures can be adopted. An excellent 1-year study by Sainz-Hernández and Maeda-Martínez (2005) examined the sources of *Vibrio* at a bivalve hatchery on the shore of Bahía de la Paz lagoon in Mexico. Contaminated sources included surface seawater, tap water, mass-produced microalgae, and scallop broodstock *Argopecten ventricosus*, suggesting a perpetual risk to the hatchery. At spawning, *Vibrio* from infected gonads was released into the tanks and then multiplied to moderate titres within a day. Riquelme et al. (1994) has shown the transfer of *Vibrio* from parent to larvae in *Argopecten purpuratus*. Low numbers of bacteria are sufficient to produce larval vibriosis, depending on the species of scallop and environmental conditions (Luna-Gonzalez et al., 2002). Another deficiency documented by Sainz-Hernández and Maeda-Martínez (2005) was the inadequate UV filtration system, which lowered bacterial numbers, but did not remove them completely. Rinsing disinfected surfaces such as tanks and floors with tap water containing *Vibrio* and using the same water source for algal culture was a significant issue that needed to be addressed as well. On the other hand, the steam generator/gun was a useful tool for the final treatment of larval rearing tanks, hoses, and sorting equipment. It would be ideal if every mollusc hatchery could afford the time and effort needed to conduct these types of bacterial studies.

Any evidence of reduced feeding should be treated as an alarm for potential bacterial build up in the system. Feeding of non-feeding larvae rapidly provides an ideal nutrient medium for bacterial proliferation, especially under warm water conditions. Sanitation and quality of food cultures may be more important than sanitation of the larval cultures themselves (Elston 1984); however, as discussed for vibriosis in *Pecten maximus* larvae, work on probiotic bacteria (Ruiz-Ponte et al., 1999; Riquelme et al., 2000; Karim et al., 2013) also shows promise for controlling bacterial proliferation. Future use of probiotics or prebiotics in commercial scallop culture may reduce the need for chemotherapeutic agents, which currently represent an important cost factor in larval production, as well as a potential environmental hazard (Jorquera et al., 2001).

MYCOTA

Among recent emerging infectious diseases of aquatic organisms, fungal, and fungal-like microbes have become important pathogens of fish and amphibians (Fisher et al., 2009; Gozlan et al., 2009). Gozlan et al. (2014) have reviewed these true fungal pathogens and fungal-like parasites from the Oomycota and Mesomycetozoea. Few cases of fungal infection of scallops have been reported despite the ubiquitous distribution of marine fungi. Lauckner (1983) mentions a shell disease where fungi biodegrade bivalve shells and describes mycotic infections in the soft parts of larval and adult pelecypods, but de-emphasises the dangers fungi pose to bivalve health. The Phycomycete, *Sirolopidium zoophthorum*, has been reported to attack cultured larvae of several species of bivalves and was implicated in severe epizootic mortalities in cultivated oyster larvae (Davis et al., 1954). Adult bay scallops, *Argopecten irradians*, at the same hatchery, however, rarely developed epizootic infections. Larvae of all ages can be affected, with branched mycelia evident inside the shell (Figure 10.4). Infection occurs by the release of

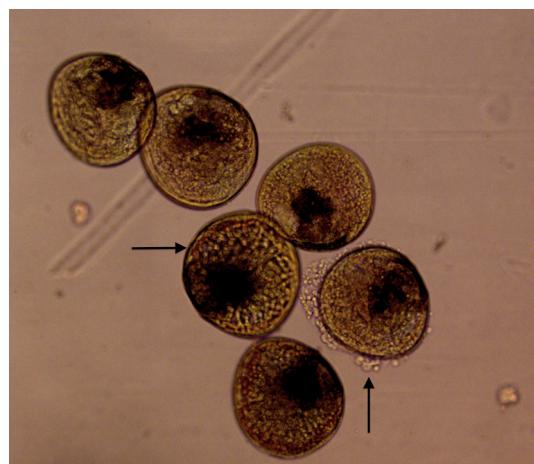


FIGURE 10.4 Fungal infection in larval bay scallops (*Argopecten irradians*). The fungus extends out of the shell edges (vertical arrow). In another, the granularity is due to fungus growing in/destroying the tissues, but still confined to the shell (horizontal arrow).

zoospores from sporangia that protrude from the shell. In a heavy infection, most of the larvae die within 2–4 days, but a small percentage may survive. The *S. zoophthorum* zoospores can be cultured on nutrient agar incubated between 20 and 30 °C. Septate thalli show branched development (Loosanoff and Davis, 1963). In an abstract, Martin (2001) outlined the continued occurrence of *S. zoophthorum* infections in the soft tissues of cultured bay scallop larvae (Mercaldo-Allen et al., 2001). This phycomycetous fungus was first observed in bivalve mollusc larvae at the Milford Laboratory in Connecticut more than 50 years ago and in 2001 remained endemic in cultures of bay scallop larvae at that laboratory. Biflagellate zoospores released from mature thalli were infective. Clonal cultures were obtained from scallop larvae and maintained successfully on enriched sea-water agar. In limited experiments, the fungus appeared to cause heavy mortality under simulated hatchery conditions.

Getchell (1991) reported a single case of a fungal infection in the adductor muscle of *Placopecten magellanicus* from the Sheepscot River, Maine. Examination by light microscopy revealed typical fungi with both mycelial growth and clusters of spores within the lesion (Figure 10.5) (Sherburne, 1982). Hyphae with septate walls and apparent budding were purplish red in colour by PAS staining. Initial work placed the fungus in the genus *Hormodendrum*. While studying another fungus (*Hormoconis resiniae* Deuteromycetes) from lesions in American plaice (*Hippoglossoides platessoides*) collected from the Sable-Western Bank complex off the Scotian Shelf of Nova Scotia, Strongman et al. (1997) investigated complaints of greenish black adductor muscle nodules in sea scallop meats from the same area. In addition to culturing a *Cladosporium* sp., the authors also isolated a *Penicillium* sp. from the lesions. No further reports of this condition have been recorded. As with 'brown spot' and other infections of this nature, collection of samples from fisheries is often complicated by offshore discard of grossly affected adductor muscle 'meats'. No similar conditions have been reported from cultured scallops. The fungi associated with the yesso scallop, *Mizuhopecten yessoensis*, as epibionts included six genera, *Aspergillus*, *Aphanocladium*, *Cladosporium*, *Penicillium*, *Phialophorophoma*, and *Eurotium* (Borzykh and Zvereva, 2012). No pathology or shell invasions were observed in this limited sample size that focused on the taxonomic composition.

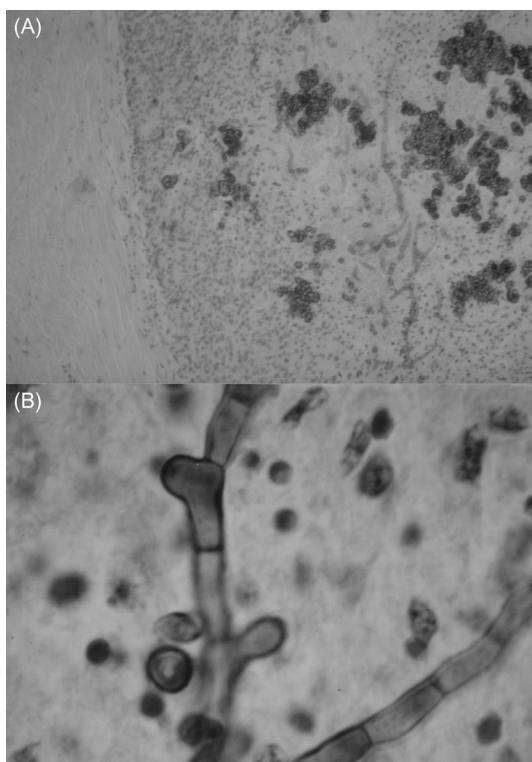


FIGURE 10.5 (A) *Placopecten magellanicus* (sea scallop) histological section of adductor muscle. Mycelial growth and clusters of spores are present within the lesion. Normal tissue is on the left side. (B) Same specimen with fungus showing septate walls and apparent budding from several hyphae. Two rounded spores are evident to the left of the hyphae (haematoxylin and eosin stain).

PROTISTA

Sarcomastigophorea (Amoebae and Flagellates)

Several flagellate and amoeboid infections have been reported from a variety of marine bivalve and crustacean species; however, none have been reported to date from scallops. An interesting possible exception is an unidentified protistan, described as 'scallop protistan with a ghostly appearance' (SPG, [Figure 10.6](#)), which infects the Japanese scallop, *Mizuhopecten (Patinopecten) yessoensis*, along the Pacific coast of Canada ([Bower et al., 1992, 1994](#)). Although not conclusively identified to phylum, the parasite does demonstrate characteristics typical of the Amoebida, namely translucent cytoplasmic inclusions, pleomorphic body shape, no surface ornamentation, or evidence of flagellae/cilia. The mononuclear cells measure 10–15 µm in diameter and are found within focal haemocyte aggregations located close to the epithelia of the gut, gonad, gill, and mantle. Histological lesions are frequently associated with grossly visible ulcerative lesions in the mantle surface, but infections are normally light. The significance of these infections is not clearly understood, and further study is complicated by the sporadic occurrence of the infection. A similar looking organism was also observed in *M. yessoensis* broodstock imported into Ireland from Japan (Bower, unpublished data).

Labyrinthomorpha (Thraustochytrids and Labythinuloids)

No labyrinthuloids have been reported from scallops, to date; however, the infectious agent of bay scallops, *Argopecten irradians*, initially described by [McGladdery et al. \(1991\)](#) as *Perkinsus karlssoni*, but subsequently recognised as not being a perkinsiid ([Goggin et al., 1996](#)), shows ultrastructural features that may place it closer to the thraustochytrid/labyrinthuloid complex, than the apicomplexa. Although no longer considered to be a valid species, the identity of the parasite encountered in bay scallops by [Karlsson \(1991\)](#), [McGladdery et al. \(1991, 1993\)](#), and [Whyte et al. \(1993a, 1994\)](#) remains unknown ([Bower, 2010](#)).

Apicomplexa

The phylum Apicomplexa no longer contains the perkinsiids (Perkinsorida). They have been given their own phylum, the Perkinsozoa, which will be covered in section '*Perkinsozoa*' below. The Apicomplexa still contain two groups of molluscan pathogens that have been identified in scallops, the coccidiains (Eucoccidiia), and gregarines (Eugregarinida).

Eucoccidiia

The first report of a coccidian infection in scallops was that of [Léger and Duboscq \(1917\)](#) who described a new species, *Pseudoklossia pectinis*, from the kidney of European king scallop, *Pecten maximus*, from Roscoff, France. Oocysts of this species measured 32–35 µm in diameter and were found solely in the kidney epithelial cells, where gamonts were also detected. A review by [Desser and Bower \(1997\)](#), however, questions the assignation of

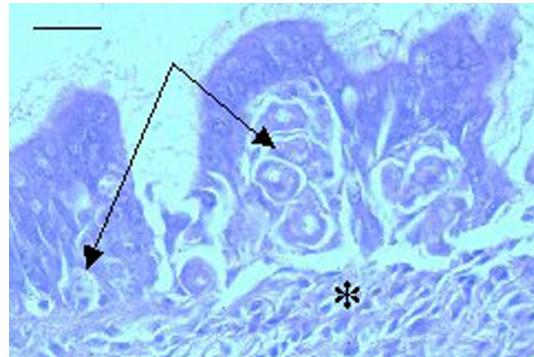


FIGURE 10.6 'SPG' lesions in *Mizuhopecten (Patinopecten) yessoensis* (Japanese or yesso scallop) are adjacent to the epithelium of the intestinal tract containing abnormally vacuolated cells (arrows) surrounded by intense focal haemocyte infiltration (*). (haematoxylin and eosin stain, $\times 630$; scale bar = 10 µm).

this species to the genus *Pseudoklossia* due to the description by Léger and Duboscq (1917) of syzygous division in the gamont stage. Desser and Bower (1997) note that this is a taxonomic feature of the family Adeloridae, rather than a feature in Aggregatidae within which *Pseudoklossia* currently occurs. No meronts (schizonts) were found which is consistent with the belief that most eimeriid coccidians have a heteroxenous life cycle. Schizogony (merogony) takes place in one host and gametogony and sporogony take place in another host. No further descriptions, reports, or investigation of the significance of this parasite to *P. maximus* have been reported.

Leibovitz et al. (1984) were the first to describe heavy renal coccidial infections in captive and cultured adult bay scallops *Argopecten irradians* (Figure 10.7). The disease consisted of coccidia at different life stages infecting the epithelium of the kidney, causing tissue destruction within the kidney and renal tubule impaction by coccidia and epithelial debris. In cultured scallops, mortalities often exceeded 80%, and coccidia also occurred in the digestive diverticula, gills, and gonad. Coccidian infections were later found in both wild and cultured bay scallops but less frequently and with limited pathology. Similar observations were subsequently reported from bay scallops held in captivity in Atlantic Canada as part of a host specificity study for an unrelated infection (Whyte et al., 1994). Although more than one type of oocyst was described by Leibovitz et al. (1984), suggesting the possible presence of more than one species, only one coccidian species is suspected in the Canadian studies (McGladdery, 1990, 1993b; Cawthron et al., 1992; Whyte et al., 1994), as well as independent eastern US observations (Getchell, 1991; Karlsson, 1991).

DiSalvo (1994) reported another coccidian in the kidneys of broodstock Chilean scallops, *Argopecten purpuratus*. This was related to production of poor-quality gametes, but no further descriptions of the infection have been reported. Other studies of this scallop species have not found this parasite (Lohrmann et al., 1991, 2000a; Lohrmann and Smith, 1993), and it is speculated that the infection detected by DiSalvo (1994) may have been opportunistic.

Most recently, Kristmundsson et al. (2011a) described a new apicomplexan, *Margolisiella islandica* sp. nov., heavily infecting wild Iceland scallops, *Chlamys islandica* (Figures 10.8 and 10.9). The wild population was

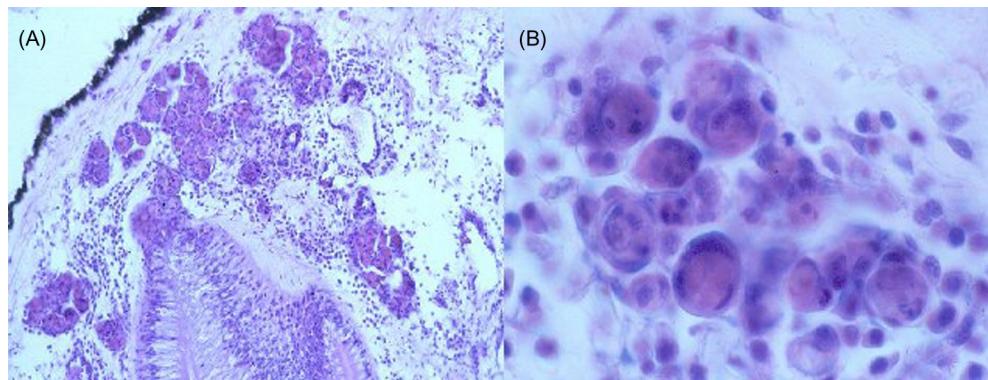


FIGURE 10.7 (A) Extensive proliferation of an unidentified coccidian throughout the renal tubules of *Argopecten irradians* (bay scallop) (haematoxylin and eosin stain, $\times 160$). (B) Meront development and release from epithelial cells of the renal tubule (haematoxylin and eosin, $\times 630$).

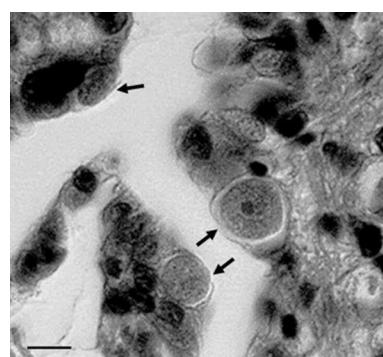


FIGURE 10.8 Histological section showing *Margolisiella islandica* trophozoites inside auricular endothelial cells (arrows) (scale bar = 10 μm). Source: Reprinted from Kristmundsson et al. (2011a). Copyright 2011, with permission from Elsevier.

suffering extensive natural mortality with adductor muscle sizes small relative to shell height. The prevalence of these intracellular infections was 95–100% from both Icelandic locations and was only detected in the auricle of the heart. Gamont-infected endothelial cells were hypertrophied and appeared to be rupturing to release the parasites. Oocytes were only seen free in the haemolymph. DNA sequencing was performed to confirm the classification of this new species. Kristmundsson et al. (2011b) also found an unknown species of apicomplexan from the same specimens of *C. islandica* as well as from queen scallops, *Aequipecten opercularis*, from Scottish and Faroese waters, and king scallops, *Pecten maximus*, from Scotland. Sexual and asexual stages of this parasite were found in the muscle tissue. Infections in Icelandic and Faroese scallops were common, but those from Scotland were very light in the cases examined. Histological observations showed numerous cysts in the adductor muscles associated with areas of necrosis and various developmental stages present. Ultrastructural examination of the sporozoite sections showed all the characteristics of apicomplexans, though different than those previously identified. The gamonts and oocysts of this parasite are elongated and much larger than those from other bivalves.

Eugregarinida

Most gregarines complete their development within an arthropod host. Members of the family Porosporidae, however, undergo intermediate developmental stages in marine bivalves. Lauckner (1983) summarises the life cycle of these gregarines. At the mid-point in the cycle gymnospores are drawn into the mantle cavity of a compatible bivalve host where they are engulfed by phagocytes on the surface of the gill or mantle epithelium. It is within these cells that the sporozoites form. The genus *Nematopsis*, which includes most species infecting bivalves, is characterised by the sporozoites being encapsulated by a resistant spore (actually the oocyst stage, according to Lauckner (1983)).

The gregarine, *Nematopsis pectinis*, was identified in *Chlamys (Pecten) varia* from Europe (Léger and Duboscq, 1925). Although originally assigned to the genus *Porospora*, Sprague (1970) reviewed the taxonomic description and based on the encapsulation of the sporozoites, transferred them to the genus *Nematopsis*. Their final decapod host was unknown. Similar gregarines have been observed in the Japanese scallop, *Mizuhopecten (Patinopecten) yessoensis* and *Pecten (Patinopecten) carinus* (Bower and Meyer, personal communication). The bay scallop, *Argopecten irradians*, has been found to be susceptible to *N. ostrearum*, which has an oocyst size of approximately 14 µm × 10 µm (Sprague, 1970). *Nematopsis duorari*, with spores 19 µm × 10 µm, has also been found in *A. irradians*. The final host is the shrimp, *Penaeus duorarum*. Kruse (1966) found that *A. (Aequipecten) irradians* shed *Nematopsis* spores in their mucous strings. The shrimp then ingest these spores with other bottom debris. Based on results of infection studies with oysters, Sprague and Orr (1955) decided that the parasites were detrimental to their intermediate hosts but did not believe that *Nematopsis* was a significant mortality factor in the wild. No pathogenicity has been described for *Nematopsis* infections of scallops. Prevalences of *Nematopsis* in the Tehuelche scallop, *Aequipecten tehuelchus*, approached 100% during surveillance in Argentina (Cremonte et al., 2005).

Perkinszoa

As stated above, the Perkinszoa is a new phylum inside the infra-kingdom Alveolata, branching close to the lineage leading to dinoflagellates and apicomplexans (Bachvaroff et al., 2011). The first perkinsiid reported from scallops was a *Perkinsus* sp. from *Mizuhopecten (Patinopecten) yessoensis* imported to the Popov Islands on east coast of Russia from Japan (Kurochkin et al., 1986). The pathological effects of the lesions described were not elucidated, and no further reports on this infection have been published.

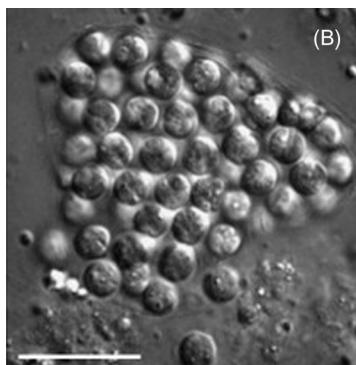
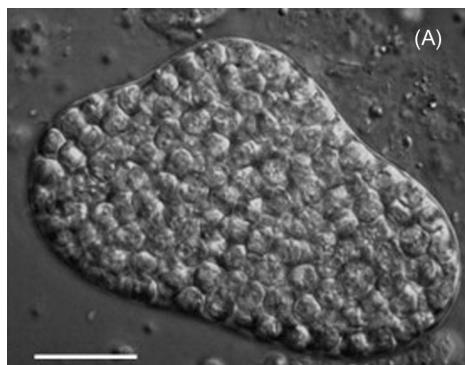


FIGURE 10.9 (A) Live mature *Margolisella islandica* oocyst with numerous sporocysts. (B) Ruptured oocyst, sporocysts spreading into the haemolymph. Scale bar = 20 µm. Source: Reprinted from Kristmundsson et al. (2011a). Copyright 2011, with permission from Elsevier.

Another perkinsiid was encountered in the same scallop species cultured in British Columbia, Canada. Initially, the infectious agent was reported as Scallop Protistan Unknown (SPX) (Bower et al., 1990, 1992, 1995, 1997, 1998; Bower and Meyer, 1994); however, detailed taxonomic investigation has confirmed SPX as a new species of perkinsiid, *Perkinsus qugwadi* (Blackbourn et al., 1998) (Figure 10.10). This infection is believed to be native to BC and is characterised by massive proliferations throughout the connective tissues of all organs, leading to weakening and death. Between 1988 and 1995, *P. qugwadi* produced mortality rates approaching 98% in juveniles and 60% in 2-year-old scallops (Bower et al., 1998). Infected scallops may demonstrate creamy white pustules up to 5 mm in diameter in many tissues, especially the gonad, but these appear to bear no direct correlation to level of infection. The disease is not easily transmitted from scallop to scallop, with most transmission being confined to a few heavily infected juvenile scallops (<40 mm shell height) within which zoospores develop (a characteristic, which differs from all other described *Perkinsus* species where sporocyst development is external (Perkins, 1996)). Progeny of *M. yessoensis* that survived an epizootic outbreak of *P. qugwadi* had a significant increase in resistance to infection and resulting mortalities. Hybrid scallops, resulting from a cross between *M. yessoensis* females (from the same group of scallops that survived an epizootic outbreak of *P. qugwadi*) and *Patinopecten caurinus* males (native to British Columbia), had similar resistance to *P. qugwadi*. The identification of scallop stocks that are resistant to *P. qugwadi* has facilitated the development of a scallop culture industry in British Columbia. No other native sympatric scallop species (*Chlamys rubida* or *C. hastata*) appear to be susceptible (Bower et al., 1999).

Prior to 2011, *P. qugwadi* was last reported in 1997 in *M. yessoensis* stocks. But recent studies by Itoh et al. (2013) revealed that infections were again occurring in British Columbia. One hundred juvenile scallops were examined, and *P. qugwadi* was detected in 44% of them by histology. Two new PCR assays were developed that detected parasite DNA in 52% of histology tissue samples equating to a 34% greater detection than by histology alone.

As discussed under section 'Labyrinthomorpha (Thraustochytrids and Labythinuloids)', the taxonomic status of a bay scallop parasite identified as *Perkinsus karlsonni* (Figure 10.11) by McGladdery et al. (1991) is now

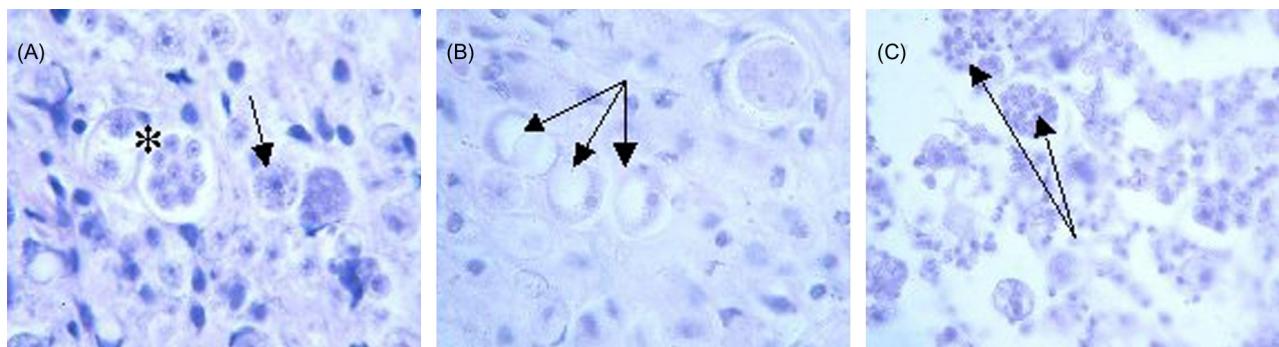


FIGURE 10.10 (A) *Perkinsus qugwadi* trophozoites (arrow) and tomites (*) in connective tissue of *Mizuhopecten* (*Patinopecten*) *yessoensis* (Japanese or yesso scallop); (B) vacuolated trophozoites ('signet-ring' form) of *Perkinsus qugwadi*; and (C) zoospore stage of *Perkinsus qugwadi* (arrows) (haematoxylin and eosin stain; $\times 1000$ (oil)).

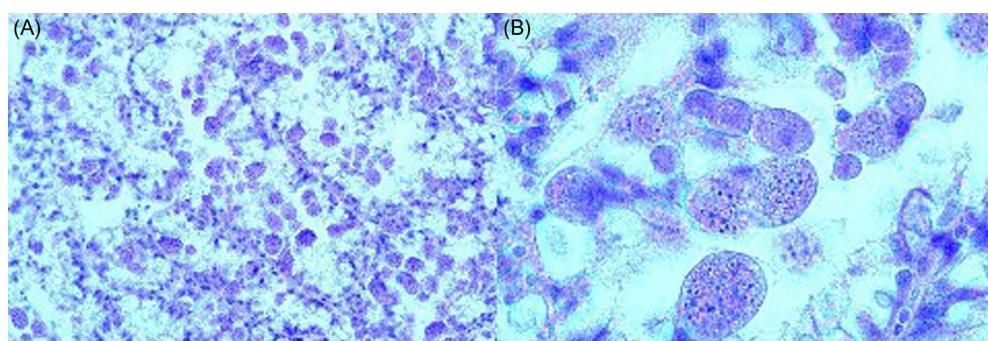


FIGURE 10.11 (A) '*Perkinsus*' *karlsonni* lesions in the connective tissue and stomach epithelium of a pre-spawning *Argopecten irradians* (bay scallop) (haematoxylin and eosin, $\times 160$). (B) High power ($\times 630$) of protistan-like inclusions within lesions and surrounding hyalinocyte response (haematoxylin and eosin).

recognised to be invalid, based on the morphology of the biflagellate stage isolated from infected scallops by culture in fluid thioglycollate media (Whyte et al., 1993a) and by DNA analysis of cultured isolates (Goggin et al., 1996). Nevertheless, the possibility that the biflagellate isolates analysed to date were not free-living contaminants from scallop tissues but are representatives of the pathogen observed histologically remains to be confirmed. Karlsson (1991) originally described the infection from bay scallops in Rhode Island. At about the same time, McGladdery and co-workers were investigating bay scallops introduced for culture purposes into Atlantic Canada (McGladdery et al., 1993b; Whyte et al., 1993b). Since bay scallops are an exotic species to Atlantic Canadian waters, there was concern over the potential to introduce new infections into Canadian bivalve population; thus, these investigations concentrated on host specificity and transmission potential. Results indicated that the infection was specific to bay scallops (Bower, 1996). The infection was characterised by intense swirl-like haemocyte aggregations, within which the parasite was frequently engulfed and masked (McGladdery et al., 1991). Infections were boosted under warm water (20 °C) recirculating holding conditions where the parasites became more evident in proliferative masses which distended the basal membranes of digestive tubule and duct epithelia (Whyte et al., 1993b). During these investigations, another parasite infection was also stimulated – a kidney coccidian, described further under section '*Eucoccidiia*'. Interestingly, the coccidian induced a similar haemocyte encapsulation response to that caused by the *Perkinsus*-like parasite, making it difficult to distinguish the two species in advanced cases (Whyte et al., 1994).

Micromspora

Although microsporidians are obligate intracellular parasites of a wide variety of vertebrate and invertebrate hosts, few bivalves have been found to be infected, and only one case in scallops has been reported (Lohrmann et al., 1999, 2000b). The infection was found in the queen scallop *Aequipecten (Chlamys) opercularis* collected from several sites around the English and Welsh coasts of the United Kingdom between 1997 and 1998 (Figure 10.12). Prevalences ranged 4.5–20%. Two stages of spore development were observed within the blood sinuses and

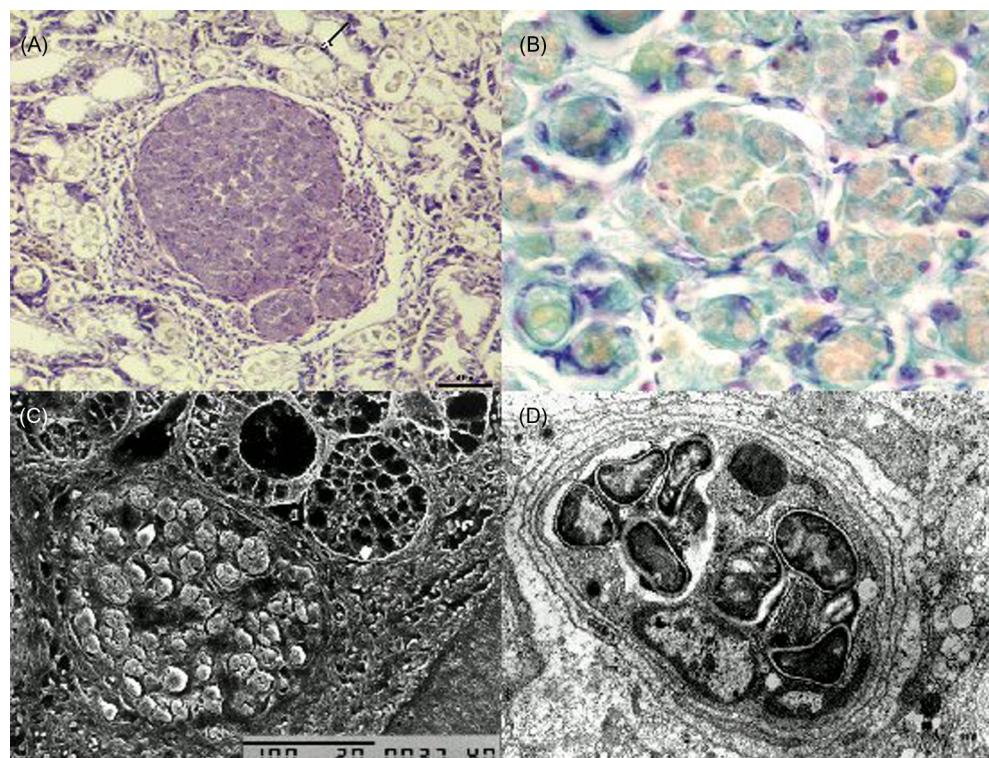


FIGURE 10.12 (A) Microsporidian colony in the digestive diverticula of *Aequipecten (Chlamys) opercularis* (queen scallop) (haematoxylin and eosin, scale bar = 50 µm). (B) Individual sporocysts within colony, Feulgen stain. (C) Microsporidian colony within digestive gland diverticula, SEM. (D) Individual sporocyst ultrastructure, TEM. Source: Images kindly provided by Dr K. Lohrmann, Universidad Católica del Norte, Coquimbo, Chile.

tissues of the digestive gland, but no other development stage was detected. The authors speculated that other developmental stages may be present in other tissues of the same host or an alternate host may be involved in the life cycle. Mature spores measured $2.3\text{ }\mu\text{m} \times 1.2\text{ }\mu\text{m}$, contained polar bodies with 7–9 coils, and were found within the vascular sinuses. Immature spores measuring $2.3\text{ }\mu\text{m} \times 1.2\text{ }\mu\text{m}$ were found within hypertrophied host cells (up to $300\text{ }\mu\text{m}$ in diameter) in the digestive gland. The polar tube of some spores had 10–12 coils, while others showed only 7–8 coils. There was no overt pathology associated with these infections. Cells infected by immature spores showed only minor focal infiltration and fibroblast-like encapsulation layers.

Ascetospora

Marteiliida

Several species of *Marteilia* have been described from European populations of flat oysters, *Ostrea edulis*, mussels, *Mytilus edulis* and *M. galloprovincialis* and a pelecypod, *Scrobicularia piperata*. In addition, the Sydney rock oyster, *Saccostrea commercialis*, and possibly the black-lip oyster, *Crassostrea echinata*, are also host of the marteiliad, *Marteilia sydneyi*. Only one marteiliad has been reported from scallops, and its impact was so severe and rapid that no material was available post-epizootic to permit detailed taxonomic investigation. The *Marteilia* sp. was found in mass mortalities of the calico scallop, *Argopecten gibbus*, off the Atlantic coast of Florida in late 1989–1990 (Moyer et al., 1993, 1995). The marteiliad overwhelmed the digestive tubules of the scallops, which showed rapid weakening and death. The host appeared to utilise its own tissues by catabolism, leading to shrunken adductor muscles and gaping shells. No further infections of calico scallop have been detected since the original reports.

Balanosporida

The Balanosporida contain some of the most devastating parasites affecting bivalve molluscs: *Haplosporidium nelsoni* and *Haplosporidium costale*, the causative agents of MSX and SSO disease, respectively, of eastern oysters *Crassostrea virginica*; and *Bonamia ostreae* and *Bonamia exitiosus* of European and New Zealand flat oysters, *Ostrea edulis* and *Ostrea lutaria*, respectively (OIE, 2003). To date, however, only two Balanosporida isolates have been reported from scallops (Chu et al., 1996; Wang et al., 2012). More than 83% of bay scallops, *Argopecten irradians*, from two stocks being cultured in the Yellow Sea area of China were found infected with an unidentified species of Balanosporida (Chu et al., 1996). Most infections found were light (Figure 10.13), but heavy post-spawning

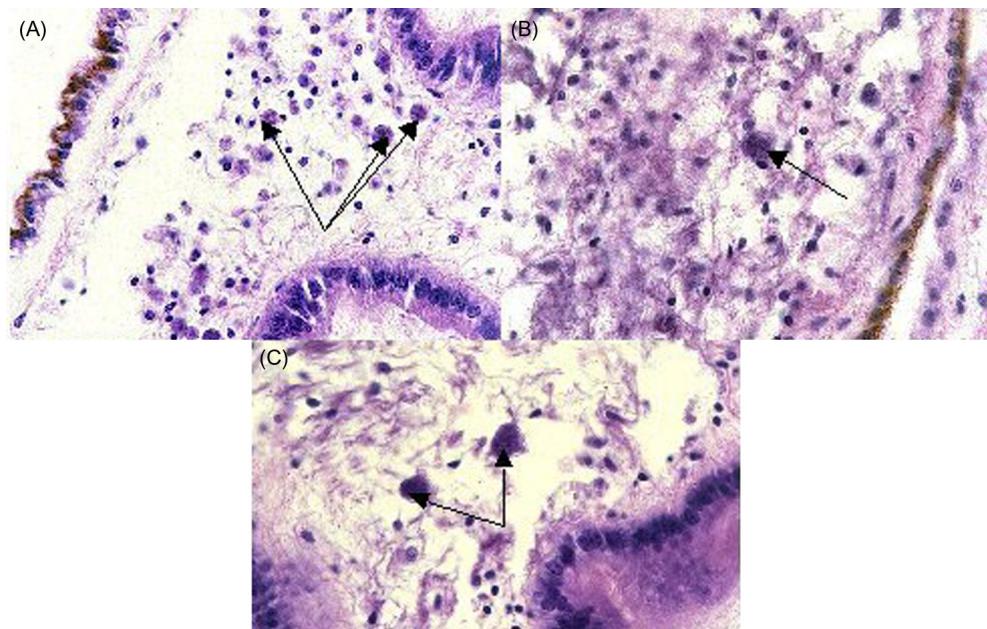


FIGURE 10.13 (A) Multinucleated schizont stage of an unidentified Haplosporidian parasite infecting the connective tissues of the bay scallop, *Argopecten irradians*, collected from China (haematoxylin and eosin, $\times 100$). (B and C) Denser plasmodial stages of the same parasite in the mantle and digestive diverticula, respectively (haematoxylin and eosin, $\times 250$). Source: Images kindly provided by Dr E.M. Burreson, Virginia Institute of Marine Science, VA, USA.

mortalities in the same area led the authors to suggest that this haplosporidian may be a potential causative factor. The second isolate was identified as *H. nelsoni* and confirmed during a survey of yesso scallops suffering mortalities by histopathology, PCR amplification, and *in situ* hybridisation (Wang et al., 2012). Plasmodia-like structures <5 µm in size were observed in the digestive glands without detecting the characteristic spores. Infiltration of host haemocytes also was documented. The monthly infection rate varied between 10% and 40% in the summer, with a peak in August. Parasites were only found from scallops raised by suspension culture, not bottom-culture methods. The morphology of *H. nelsoni* was similar to that reported by Wang et al. (2010) in *Crassostrea gigas* collected in China. The authors suggest the mortalities may have been related to increasing stocking densities (Wang et al., 2012).

Ciliates

The majority of ciliates associated with bivalves are, like the flagellates described under section 'Sarcomastigophorea (Amoebae and Flagellates)', probably harmless commensals. More than 150 species of ciliates have been found in the mantle cavity, on the gills, or in the digestive diverticula of marine bivalves. Most are primarily commensal but can become pathogenic if their numbers become unusually high, the physiological state of the host is compromised or an environmental stress factor shifts the equilibrium (Lauckner, 1983).

Trichodinids are peritrichous ciliates that are common symbionts of amphibians, fishes, and bivalves (Figure 10.14). Most are commensals feeding on bacteria and occurring at low prevalence and intensity of infestation within the mantle cavity. Some trichodinid infections in bivalves, however, have been linked to tissue damage and mortalities (Lauckner, 1983). Trichodinids are easily recognised by their dome shape, rows of cilia, conspicuous circle of hooklets, and horseshoe-shaped macronucleus.

In scallops, *Trichodina pecten* has been reported in the mantle cavity of *Mizuhopecten (Patinopecten) yessoensis* and *Trichodina polandiae* from *Chlamys* sp. collected from the Gulf of Peter the Great (Sea of Japan) (Stein, 1974 cited in Lauckner, 1983). The former species is described as having a ring of 22–31 denticles, each denticle having 7–9 radial rods. The species described from *Chlamys* sp. has 20–26 denticles with 7–10 radial rods on each (see table 13.9 in Lauckner, 1983). Another, possibly identical, trichodinid was reported on *M. yessoensis* by Kurochkin et al. (1986). Xu et al. (1995) reported another species, *Trichodina jadranica*, from the gills of *Chlamys farreri*. This ciliate averaged 37.8 µm in diameter with 21–24 denticles and an adoral ciliated membrane spiral of approximately 400 degrees. Lohrmann et al. (2000a, 2002, 2009) reported a *Trichodina* sp. from the gills of the Chilean scallop, *Argopecten purpuratus*, while prevalence of *Trichodina* sp. in the Tehuelche scallop, *Aequipecten tehuelchus*, approached 100% during surveillance in Argentina (Cremonte et al., 2005). Trichodinids are also commonly found in the mantle cavity of giant sea scallops *Placopecten magellanicus* from Atlantic Canada (Beninger et al.,

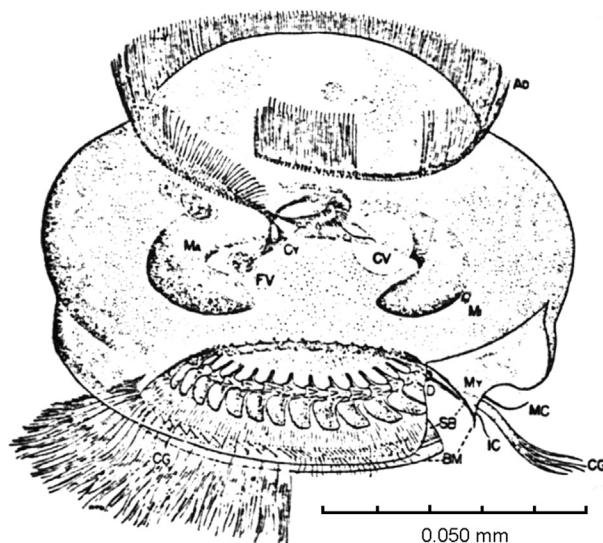


FIGURE 10.14 *Trichodina myicola* from the clam, *Mya arenaria*, showing rows of cilia and typical circle of hooklets. Source: Adapted from Uzmann and Stickney (1954). *J. Protozool.* 1:152.

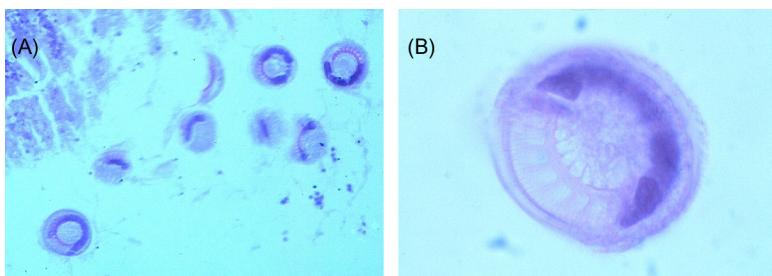


FIGURE 10.15 (A) Unidentified trichodinid ciliates on the surface of the sea scallop (*Placopecten magellanicus*) from Atlantic Canada (haematoxylin and eosin, $\times 160$). (B) High magnification ($\times 630$) of denticle arrangement of one of the sea scallop trichodinids (haematoxylin and eosin stain).

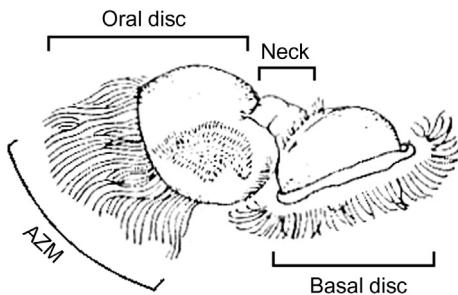


FIGURE 10.16 *Licnophora auerbachi* from the eye of *Chlamys opercularis* showing (i) oral disc with cilia making up the adoral zone of membrane (AZM); (ii) neck region, and (iii) basal disc. Source: Adapted from Harry (1980).

1988; McGladdery et al., 1993a) (Figure 10.15). In some *P. magellanicus*, numbers may exceed more than 100 per 5- μm -thick tissue section of gill but no tissue damage has been found to date. Xiao et al. (2005) observed an increase in *Trichodina* sp. numbers during the mass summer mortalities of cultured zhikong scallops, *Chlamys farreri*, in China. *Trichodina* were seen in the mantle cavity of up to 67% of the scallops examined with as many as 116 per section counted. The authors suggested that the large numbers were more a measure of a degraded environment and stressed scallops than of a direct cause of mortality.

Whilst most trichodinids usually occupy many areas within the mantle cavity of a bivalve, the heterotrich, *Licnophora auerbachi*, resides in a more unusual niche, the eyes of the scallop. These ectoparasites are highly motile and very difficult to detach from the eye surface. Harry (1977) found that 85 out of 88 queen scallops, *Aequipecten* (*Chlamys*) *opercularis*, collected from County Down, Ireland, harboured *L. auerbachi*. Only a few *Pecten maximus* and *Chlamys varia* were found infested, indicating a degree of host specificity that Harry (1977) was able to confirm with *in vitro* investigations. One specimen of *A. opercularis* was parasitised by ciliates on 94 out of 103 eyes. Loss of pigment from the iris and signs of disintegration were two of the prominent pathological features of this heavily infected individual. Harry (1980) suggested that the action of the basal disk as it attaches caused damage to the epidermis of the eye. Like *Trichodina*, *Licnophora auerbachi* is normally a filter feeder probably thriving on bacteria (Figure 10.16).

The eyes of scallops are not able to form focused images, so it is unlikely that the presence of these ciliates affect their visual response to stimuli. However, *L. auerbachi* might have an effect on young developing scallops by interfering with their light–dark shadow detection escape response (Harry, 1977, 1980).

During studies of cultured *Chlamys farreri* and *Argopecten irradians* from the coast of Qingdao, China, Hu and Song (2001) re-described a little known marine ciliate, *Stichotricha marina*. Although the infestation frequency in the mantles was nearly 100%, infection density was light, so the parasites were considered to be ectocommensals.

PLATYHELMINTHES

Trematodes

Bucephalid and fellodistomid digenetic trematodes parasitise many species of marine bivalves, and some are known to cause significant damage to the reproductive tissue of the host. Sanders and Lester (1981) documented a bucephalid trematode infection of *Pecten alba* from Bass Strait, Australia, which turned the scallop gonad bright



FIGURE 10.17 Unidentified digenetic trematode embedded in the connective tissue of the digestive gland of *Placoplecten magellanicus* (haematoxylin and eosin stain; $\times 250$, scale bar = 0.1 mm).

red or orange in colour. Histological observations showed the gonads contained sporocysts with cercariae throughout most of the year. The sporocysts occupied the bulk of the gonad as the scallop reached a state of sexual maturation. Over the 6-month period during which sporocysts produced cercariae, the average weight of the adductor muscle fell below that of unparasitised scallops. The loss of reproductive potential and energy reserves stored in the adductor muscle reflect the strain this trematode has on its scallop host. [Heasman et al. \(1996\)](#) reported other *Bucephalus* sp. sporocyst infections of Australian scallops. These authors followed infections in two species of scallops (*Chlamys asperrima* and *Pecten fumatus*) from New South Wales, Australia, and documented significant increases in prevalence of infection. Over a 2-year period prevalence increased to 66% in *P. fumatus* over 80 mm in length and 40% in *C. asperrima* over 75 mm in length, from 5.1% and 4.3% in 1991 and 1992, respectively. [Hutson et al. \(2004\)](#) took a molecular genetic approach to identify bucephalid parasites where morphological features were of limited value. They studied the ecological relationships of bucephalids between different South Australian scallops including *C. asperrima*, *C. bifrons*, and *P. fumatus* and possible adult hosts. Sequencing the mitochondrial cytochrome c oxidase subunit 1 gene permitted the identification of at least two different types of bucephalid parasites infecting these scallops but failed to identify the hosts of the adult trematodes.

[Mateo et al. \(1975\)](#) reported castration of *Argopecten purpuratus* from Peru in association with sporocysts showing hemiuroid characteristics. Fellodistomid digenetic trematodes belonging to the genus *Proctoeces* are also well documented as causing varying degrees of tissue damage in bivalves ([Bray, 1983](#)). The most commonly reported species is *Proctoeces maculatus*; however, the broad geographic and host ranges reported for this species have led some authors to speculate that this name contains more than one species and requires re-investigation ([Lauckner, 1983](#); [Bower, 1995](#)). The metacercarial stage of *P. maculatus* is reported from various Mollusca, Polychaeta, and Echinoidea. Adults occur in mollusc-eating fishes such as labrids and sparids; however, progenetic development is also documented in mussels, which normally host the sporocyst developmental stage ([Bray, 1983](#)). [Karlsson \(1991\)](#) described adult worms, tentatively identified as *Proctoeces* sp., from the kidney and opening of the gonoduct into the kidney of bay scallops from Rhode Island. Some trematodes showed attachment to the kidney epithelia via the oral sucker. Although most tissue sections revealed only single worm infections, one scallop kidney section harboured over 50 *Proctoeces*. [Bray \(1983\)](#) also reported *P. maculatus* in bay scallops collected from Rhode Island.

Unidentified metacercariae belonging to the Echinostomatidae, Fellodistomidae, or Gymnophallidae have also been reported from bay scallops from Rhode Island. [Karlsson \(1991\)](#) noted varying numbers of metacercariae encysted in the labial palps, digestive gland, and gill tissues. Despite numbers exceeding 50 metacercariae per tissue section in some specimens, there was little evidence of a strong host response. Similar metacercariae have been reported from the labial palps of *Argopecten purpuratus* in Chile ([Lohrmann et al., 1991](#)), *Chlamys islandica* from the Barents and White Seas ([Chubrick, 1966](#), cited in [Lauckner, 1983](#)) and in *Placoplecten magellanicus* from Atlantic Canada ([McGladdery et al., 1993a](#)). The echinostomatid *Himasthla quissetensis* uses *Argopecten irradians* as a second intermediate host, before final maturation in the intestine of the herring gull, *Larus argentatus*. The metacercariae, 140–190 μm in diameter, occur in the mantle, gills, and foot but do not undergo any further development in the cyst ([Stunkard, 1938](#)). A similar lack of growth occurs when metacercariae of *Renicola thoidis* parasitise *Argopecten irradians*. The adult trematode infects the kidney of shore birds ([Getchell, 1991](#)). Adult trematodes are rare in scallops and possibly only occur as accidental infections (Figure 10.17). [Oliva and Sanchez](#)

(2005) identified the digean, *Derogenes varicus* (Derogenidae), in *A. purpuratus* while conducting research to test whether parasites may serve as biological tags for stock discrimination.

Pérez-Urbiola and Martínez-Díaz (2001) found a condition known as 'pimentilla' disease in catarina scallop, *Argopecten ventricosus (circularis)*, from Baja California Sur, México, to be caused by *Stephanostomum* sp., a species of acanthocolpid digenaeans. This is the first description of this group of digeneans using molluscs as second intermediate hosts. Most other species of *Stephanostomum* encyst in the body cavity of benthic fish and mature in the guts of piscivorous fish. The metacercariae encyst throughout the soft tissues of the scallops, eliciting a strong melanisation response, which gives the scallop a peppery ('pimentilla') appearance and renders it unmarketable. Except for signs typical for metacercarial infections (i.e. a focal haemocyte infiltration and encapsulation), the effect on the health of the scallop was not reported despite extremely high levels of infection (Pérez-Urbiola and Martínez-Díaz, 2001).

Unidentified larval sanguinicolid have been recorded from *Argopecten irradians* from Woods Hole, Massachusetts (Linton, 1915). Interestingly, this group of digeneans develop into adults directly from cercariae, omitting the usual metacercarial encystment stage. Within the bay scallop, the only report of pathology was castration caused by sporocyst proliferation throughout the gonadal tissues. There have been no further reports of sanguinicolid sporocyst infections of bay scallops (or any other scallop) since Linton's (1915) note; however, Lauckner (1983) provides a thorough review of infections of other molluscan groups.

Cestodes

Among the helminth parasites of shellfish, cestodes, particularly larval forms (metacestodes), are common inhabitants within the digestive tract of Atlantic bay scallops (*Argopecten irradians*). Seven species of larval cestode were identified in the eastern Gulf of Mexico by Cake (1977, 1979). These include the four most prevalent, *Parachristianella* sp. that encysts singly along the walls of the intestine, *Tylocephalum* sp. in the digestive tract walls and digestive gland, *Rhinebothrium* sp. in the stomach and digestive diverticula, and *Polycephalus* sp. in the visceral masses and digestive gland (Figure 10.18). The other single isolations were *Acanthobothrium* sp., *Anthobothrium* sp., and *Eutetrahynchus* sp. Metacestodes, *Tylocephalum* sp., were also reported from 5 of 25 *Chlamys nobilis* from Japan (Sakaguchi, 1973 cited in Lauckner, 1983). *C. nobilis* are initially infected by ingesting eggs, gravid proglottids, or free-swimming coracidia. Most larval forms can be identified only to genus because the plerocercoids lack taxonomic characteristics.

None of these genera have been associated with overt pathology, most infections being light and eliciting a focal haemocyte response similar to that described for metacercarial digeneans. High levels of infection, however, may cause physiological stress that can affect growth, reproduction, and edibility. In calico scallops *Argopecten gibbus* from North Carolina, high levels of infection by metacestodes belonging to the genus *Echeneibothrium* have been associated with significant gonad atrophy (Singhas et al., 1993). Infections by cestodes of the same genus have also been associated with aberrant behaviour in littleneck clams *Protothaca staminea* and *P. laciniata* from California (Sparks and Chew, 1966; Warner and Katkansky, 1969).

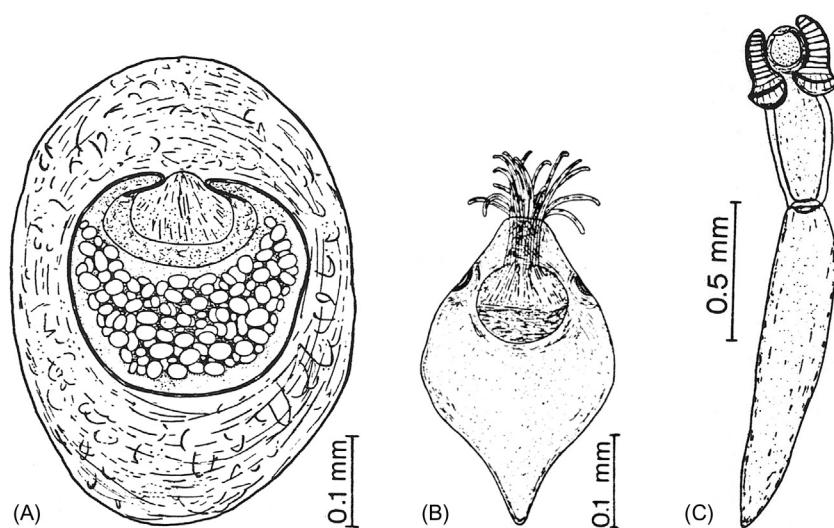


FIGURE 10.18 Larval cestodes from the scallop, *Argopecten irradians concentricus*. (A) Encapsulated, acaudate glando-procercoform of *Tylocephalum* sp. (B) Tentaculo-plerocercoid of *Polycephalus* sp. (C) Bothridio-plerocercoid of *Rhinebothrium* sp. Source: Adapted from Cake (1976).

The Chilean scallop, *Argopecten purpuratus*, was also infected by larval cestodes. Oliva et al. (1986) found metacestodes, which they tentatively assigned to the Phyllobothriidae and Oncobothriidae, in the gonadal tissues of samples collected from Antofagasta, Chile; and Lohrmann and Smith (1993) found another unidentified larva in the intestine of the same species collected from Coquimbo, Chile. None of the infections was associated with overt pathology. While testing the value of parasites and commensals of *A. purpuratus* as biological tags for stock discrimination, Oliva and Sanchez (2005) identified the larval cestodes *Acanthobothrium* sp. and *Rhinebothrium* sp. with infection prevalence ranging 0–44% and 1–97%, respectively, at four locations in northern Chile.

Because the larval forms infect the viscera of the scallop, which is discarded during processing, there is little chance of humans consuming cestodes with the edible adductor muscle. Any cestodes not discarded during processing would be destroyed by the cooking, and Cake (1977) mentions experimental evidence that suggests that *Tylocephalum* larvae are destroyed by human digestive acids and enzymes. The final hosts for all these cestode genera are elasmobranch fish (sharks, skates, and rays).

Turbellaria

Turbellarian flatworms are commonly found on the gills or within the guts of a wide range of bivalves (Lauckner, 1983); however, few have been reported in scallops. An unidentified turbellarian was reported on the digestive diverticula of bay scallop, *Argopecten irradians* (Leibovitz et al., 1984), with no apparent pathological effect.

A turbellarian resembling *Urastoma cyprinae*, a common inhabitant of the gills of eastern oysters, *Crassostrea virginica*, in Atlantic Canada has been reported from the gills and body surfaces of sea scallop from the same area (McGladdery et al., 1993a) (Figure 10.19). Compared with oyster infestation levels, those found on the scallops occur in much lower numbers (1–2 per tissue section and maximum prevalence of 10%) than occur on oysters (up to 1000s of *U. cyprinae* and 100% prevalence). In both scallops and oysters from Atlantic Canada, there was no obvious histopathological effect on the gills; however, hypertrophy of gill epithelial cells in mussels from Spain has been attributed to the same turbellarian (Robledo et al., 1994), and there is some evidence for biochemical alteration in the mucoid chemistry of heavily infected oysters (Brun et al., 2000).

A report by Woods and Hayden (1998) associated the presence of a *Paravortex* sp. of turbellarian in the digestive diverticula with weakening and mortalities in the New Zealand scallop, *Pecten novaezelandiae*. Clinically, the scallops showed mantle retraction, gaping, and impaired swimming behaviour prior to death. Prevalences of the *Paravortex* were greater in moribund scallops, but intensity of infections was low (<3 turbellarians per scallop) and not all affected scallops were infected. Other diseases, such as Malpeque disease of eastern oysters, *Crassostrea virginica*, have also been found to show a positive correlation with turbellarian proliferation (Drinnan, R.E., unpublished data); however, this is believed to be a secondary opportunistic proliferation, rather than a primary cause due to inconsistencies between manifestation of disease signs and presence of the turbellarian. The ongoing work of Brun et al. (2000) may be able to shed some light on bivalve–turbellarian interactions at a physiological level. Documentation of effects at the predation level are more clear, for example, *Pseudostylochus ostreophagus* predation occurs on small juveniles of Japanese scallops, *Mizuhopecten* (*Patinopecten*) *yessoensis* (Bower and Meyer, 1994).



FIGURE 10.19 *Urastoma cyprinae* on the gills of the sea scallop, *Placopecten magellanicus*, from Newfoundland (haematoxylin and eosin stain, scale bar = 1 mm).

Nematodes

There is limited published information on parasitic larval nematodes of marine molluscs because they are difficult to identify and require special procedures, such as enzyme digestion, to isolate them from their embedded locations within bivalve tissues (Cheng, 1978).

Abundant literature exists on the ascarid nematode, *Sulcascaris sulcata*, which infests larval scallops and, as an adult, inhabits the stomach of sea turtles, *Chelonia mydas* and *Caretta caretta* (Sprent, 1977). Two scallops that serve as intermediate hosts are *Amusium balloti* and *Chlamys* sp. in Australian waters (Cannon, 1978; Lester et al., 1980). Lichtenfels et al. (1978, 1980) and Blake et al. (1984) identified larval *S. sulcata* from the Atlantic scallops, *Argopecten gibbus* and *A. irradians*. Parasitised green and loggerhead turtles occur in the same general area as the infected scallops.

The life cycle of *S. sulcata* starts with the release of negatively buoyant eggs from adult worms living in the intestine of the sea turtles. The eggs are shed with the turtle faeces, sink and adhere to the sea floor. After about 1 week, third stage larvae hatch and infect their molluscan host. Fourth stage larvae within the molluscs are ingested by the sea turtles and attach to the oesophageal gastric junction where they moult into adults within 7–21 days. Subsequent time to maturity takes at least 5 more months (Berry and Cannon, 1981).

In Australia, scallop adductor muscle was infected, while all *S. sulcata* occurred in the gonad of *Argopecten gibbus* off the Atlantic coast of the United States. The worms are white and inconspicuous when small and yellow to pale orange or brown when larger. There is cellular infiltration around the larvae, which usually lie loosely coiled near the surface of the adductor muscle. The presence of many larvae can damage the muscle enough to cause a loss of tonicity (Cannon, 1978, cited in Berry and Cannon, 1981).

The presence of nematodes and the brownish capsule in which they were found, make some of the processed adductor muscles unsuitable for export. Up to 64% of commercially harvested saucer scallops, *Amusium balloti* were infected by *S. sulcata* (Lester et al., 1980). Depending on whether an infected scallop is consumed cooked or raw, *S. sulcata* may be regarded as an aesthetic problem or as a threat to public health. However, infectivity trials have failed to infect teleost or elasmobranch fish, or chickens or cats; thus, there is no evidence that they are a threat to human health (Berry and Cannon, 1981) and no human cases of ascariasis have been reported from consumption of infected scallops.

A small percentage of *Amusium balloti* were also infested with a larval gnathostome, *Echinocephalus* sp., and two other scallop species, *Anachlamys leopardus* and *Chlamys asperrimus*, were found to contain *S. sulcata* (Lester et al., 1980). McLean (1983) identified second stage juveniles of *Echinocephalus pseudouncinatus* from the adductor muscles of *Argopecten aequisulcatus* (*A. ventricosus*) from Baja California, Mexico. The worms measured 14–17 mm in length and were located within yellow or brown spots on the surface of the adductor muscle. The same species of nematode infects pink abalone, *Halichoeres corrugata*, from California and causes blistering of the foot. Heavy infections are associated with weakening of the foot, holdfast attachment, and grazing capability (Sindermann, 1990). The final hosts of *E. pseudouncinatus* are elasmobranch fish.

Another nematode, *Porrocaecum pectenisi*, reported in *Argopecten irradians* and *A. gibbus* is usually invisible macroscopically. However, brown discolouration of the worm occurs when it becomes infected by an unidentified haplosporidian hyperparasite (Cheng, 1967, 1973, 1978; McLean, 1983). This renders the worm visible and reduces the quality of the scallop meat.

POLYCHAETES

Even though polychaete worms do not generally penetrate the soft tissues of the bivalves that host their refugia, the excavation of their tunnels in the matrix of the bivalve shell can have a significant indirect impact on physiology and overall survival of the bivalve. In addition, the mud substrate used to line the tunnels created by the most common family of shell dwellers, the polydoriids, can exacerbate the erosion effect. Subsequent enlargement of the tunnels creates opportunities for other polychaetes, which, unlike the relatively innocuous *Polydora* spp., are easily visible by consumers, for example, *Ceratonereis tridentata* (Wells and Wells, 1962) and *Nereis diversicolor* (McGladdery et al., 1993a). Shell damage and lively polychaete occupants can significantly impact live mollusc markets. Although this is not usually a factor for scallop meat markets, many species are marketed in shell. Another effect of shell excavation is tunnel perforation of the inner shell surface – especially at the adductor muscle attachment site – which can create access for soft-tissue irritants, abrasion, and secondary opportunistic microbial infection (see ‘Other bacterial pathogens of scallops’). Weakened adductor muscle attachment also impairs shell closure, swimming, feeding, and escape behaviour in scallops. Bergman et al. (1982) reported that

scallop shells weakened by *P. websteri* boring are more vulnerable to crushing by predatory decapods. The patterns of breakage among scallop (*Placopecten magellanicus*) shells damaged by rock crabs and lobsters suggest that successful predation is often accomplished by fracture of the upper valve near the umbo where the level of polydorid infestation was generally high. It was unclear what effect shell infestation had on scallop natural mortality (Elner and Jamieson, 1979). Turner and Hanks (1959) considered *P. websteri* infestation a contributing factor to unusually high mortalities of bay scallops in Fairhaven, MA. Polychaete burrows close to the scallop hinge may have interfered with the function of the adductor muscle, which in many cases was in poor condition. Similar damage to the same scallop species has been linked to *Polydora ciliata* and *Polydora* spp. by Russell (1973), Leibovitz et al. (1984) and Karlsson (1991).

The harmful effects of polychaete shell damage vary with the intensity of infestation and the type of burrow formed. The most obvious chronic effect is development of mud blisters. These form as sand or mud breach the nacre layer of the shell. Continuous erosion of non-peripheral surfaces is progressively walled off by conchiolin and nacre secretion from the epidermal tissues in contact with the irritant. Such effects from *Polydora concharum*, *P. socialis*, and *P. websteri* are well documented for the sea scallop, *Placopecten magellanicus* (Blake, 1969; Evans, 1969; Bergman et al., 1982; McGladdery et al., 1993b). Wells and Wells (1962) and Blake and Evans (1973) describe effects in bay scallops, *A. irradians*, from the eastern United States, and Skeel (1979) described *Polydora* infections in four species of cultivated bivalves in Australia. The Tasmanian scallop, *Notovola (Placopecten) meridionalis*, was infected with *P. websteri*, which formed mud blisters between the shell and mantle (Skeel, 1979). An alternative route for inducing such damage is for polychaete larvae to swim into the mantle cavity and burrow between the shell and mantle; however, most evidence supports externally driven penetration to the inner surface. Shell damage at the peripheral edges can also induce mantle retraction and growth deformities (indentations, overgrown, or double lipped edges, etc.). Regardless of route and site of irritation, this challenge can assume extreme proportions if it persists and the resultant mud blisters take over a significant percentage of the inner shell cavity (Figure 10.20).

Mori et al. (1985) investigated the polydorid infestation of scallops, in this case *Mizuhopecten (Patinopecten) yessoensis*, in Japan. The boring polychaetes were identified as *Polydora variegata*, *P. ciliata*, and *P. concharum* (Figure 10.21). The worms settled almost exclusively on the left valve of these cultured scallops. Two years after seeding, the shell region near the attachment of the adductor muscle was heavily penetrated by polydorid tunnels, which frequently contacted the muscle attachment surface. These authors suggested that these polydorids could have a significant influence on the growth of scallops in that area. Similar observations have also been reported for this scallop species in Japan (Sato-Okoshi and Okoshi, 1993), Russia (Kurochkin et al., 1986), and Pacific Canada (Bower and Meyer, 1994). Sato-Okoshi and Okoshi (2013) also have described the polydorid parasites associated with *M. yessoensis* and *Chlamys farreri* in northeast China, including detailed morphological, ecological and molecular biological characteristics. *P. onagawaensis* infested both scallop species but created little damage. On the other hand, *P. brevipalpa* caused heavy infestation of cultured yesso scallops.

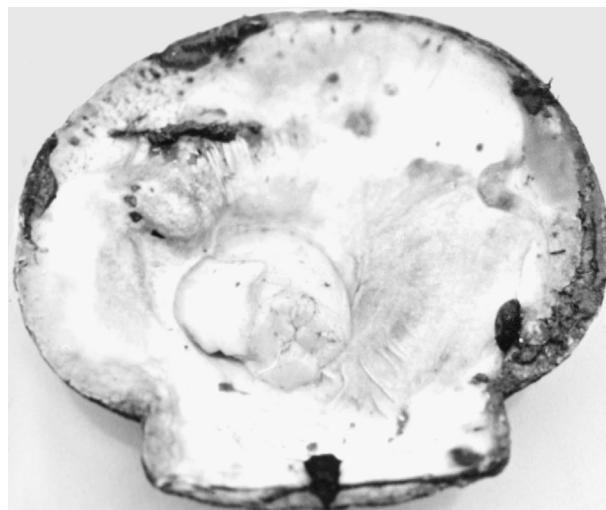


FIGURE 10.20 *Placopecten magellanicus* with extensive mud blisters occupying one-third of the inner surface of the valve. Note poor condition of adductor muscle attachment site.

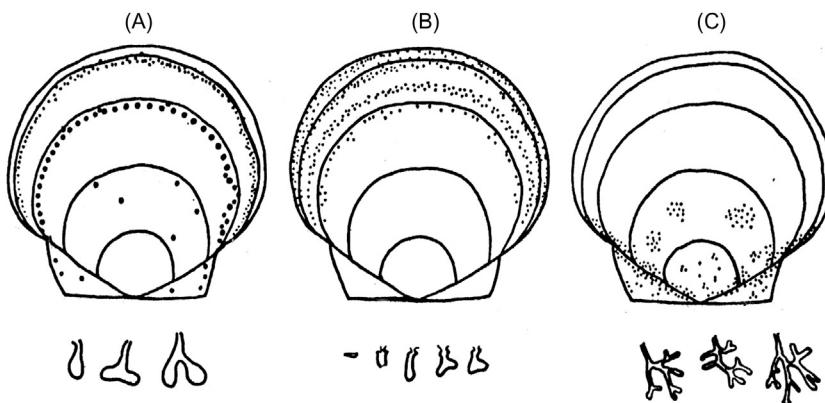


FIGURE 10.21 Comparison of burrowing regions and morphology of burrows in three species of *Polydora*. (A) *P. variegata*; (B) *P. ciliata*, and (C) *P. concharum*. Source: Adapted from: Mori et al. (1985).

(approximately 30–80 worms per individual) with damaging impact on the scallop aquaculture industry ([Sato-Okoshi and Okoshi, 2013](#)). [Gabaev \(2013\)](#) also examined the effects of fouling by *P. brevipalpa* on yesso scallops cultivated on bottom sediments in the Russian Far East. On the west coast of Canada, shell damage (thickening, growth stunting, impairment of adductor muscle function, and associated mortalities) from a related species *P. websteri* is so severe that it renders culture of *M. yessoensis* impractical in some locations ([Bower et al., 1994](#)). Similar observations have been reported for polydoriid infestation of great scallop, (*Coquille St. Jacques, Pecten maximus*), spat from scallop nurseries in Norway ([Mortensen et al., 1999](#)). The spat measuring only 5–7 mm were found to have shells heavily infested by a *Polydora* sp. This infestation was associated with losses representing a third of Norway's cultured scallop production in 1997. Another polychaete, the tubeworm *Pomatoceros triquetus*, has been associated with fouling-related mortalities of up to 65% in the same scallop species in Ireland ([Burnell et al., 1995](#)). In Argentina, 82% of *Aequipecten tehuelchus* in the shelf waters off of Buenos Aires were infested with polychaetes, including *Serpula* sp. (75%), *Sirorbinae* (28%), *Phyllochaetopterus socialis* (21%), *Idanthyrsus armatus* (11%), and *Chaetopterus antarcticus* (0.4%) ([Souto et al., 2012](#)). [Diez et al. \(2013\)](#) also characterised the infestation patterns of *A. tehuelchus* by *Polydora rickettsi*.

A fouling spionid, *Sabella spallanzanii*, has been reported on *Pecten fumatus* shells from Cockburn Sound, Western Australia, by [Clapin and Evans \(1995\)](#). Resultant smothering losses have necessitated re-evaluation of suspension culture methods ([O'Connor et al., 1999](#)) and development of shell attachment methods (gluing) rather than cage-culture.

Cirratulids, such as *Dodecaceria concharum*, may occupy the empty burrows of *Polydora* spp. in the shells of *Placopecten magellanicus* ([Evans, 1969; Leibovitz et al., 1984](#)). And sabellids, like *Pseudopotamilla reniformis*, have also been found in the shells of *P. magellanicus* from Maine. The large size of the worms (up to 100 mm) and the secretion of large ridges of shell material to contain them may be a potential problem for scallops with heavy infestations. [Blake \(1969\)](#) reported up to 30 worm tubes from a single scallop shell infested with *P. reniformis*.

CRUSTACEA

Pinnotheriidae

Many species of pinnotheriid crabs, also known as 'pea crabs', live parasitically in the mantle cavity of marine bivalves. The scallops, *Argopecten irradians concentricus*, *A. gibbus*, and *Placopecten magellanicus*, are hosts for the species *Tumidotheres (Pinnotheres) maculatus* along the eastern United States ([Getchell, 1991](#)). Disease conditions associated with pinnotheriid crabs include emaciation, reduced filtering capacity, damage to the gills, palps, and mantle, and compression of the gonad, which may affect gonadal development. There are no observations of crabs feeding directly on host tissues, but their location within the mantle cavity inflicts a variety of host conditions ranging from slight irritation to severe structural alterations and pathology ([Lauckner, 1983](#)).

Infestation with *T. maculatus* was found to cause stunting in *Argopecten irradians concentricus* from the eastern United States. Bay scallops containing adult female pea crabs were slightly smaller than uninfested scallops. Growth of three size groups of scallops was measured over a 3-month period. Smaller scallops hosting pea crabs grew significantly less than uninfested scallops, although the larger scallops showed no significant difference between infested and uninfested groups ([Kruczynski, 1972](#)).

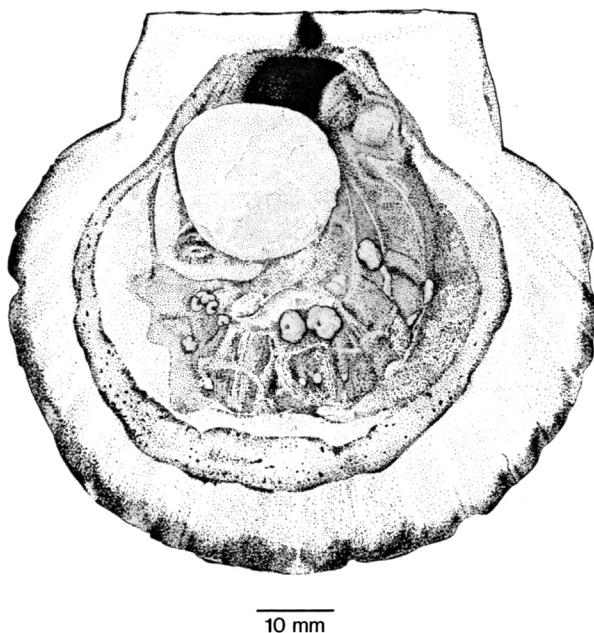


FIGURE 10.22 *Patinopecten yessoensis* left valve with 15 small and large specimens of *Pectenophilus ornatus* attached to the left demibranchs. Source: Adapted from: Nagasawa et al. (1988).

Other scallops documented as hosting pinnoetheriid crabs include *Amusium pleuronectis* (Llana, 1979) and *Chlamys farreri* (Cheng, 1967). Narvarte and Saiz (2004) described the effects of *T. maculatus* on *Aequipecten tehuelchus* in the San Matias Gulf, Argentina. Larger crabs were found in the mantle cavity near the adductor muscle and beneath the gonad, mostly by themselves. Differences were found between the condition indices of infested and non-infested scallops because of the reduced weights of the muscle and gonad. The prevalence of infested scallops was 56%. The presence of the crabs may increase the processing time due to the extra effort to remove each one prior to canning.

Copepodidae

Several copepod parasites have been reported from scallops. Reddiah and Williamson (1958) recorded *Paranthessius pectinis* and *Modiolicola inermis* from *Aequipecten opercularis* collected from around the Isle of Man, off the United Kingdom, as well as *Modiolocola maxima* from *Pecten maximus* in the same area. The effects of these copepod infestations were not reported as being significant. A similar *Modiolicola* sp. infestation of *Pecten maximus* from Norway was, likewise, not associated with pathological effect (Mortensen, 1993b). And finally, a *Pseudomyicola spinosus* infestation of the catarina scallop, *Argopecten ventricosus*, was studied for 1 year, and it was determined that there was no evidence of pathogenicity or abnormal scallop mortality (Caceres-Martinez et al., 2005). The copepod was found in the pallial cavity, gills, labial palps, digestive gland, and in the lumen of the stomach, where epithelial erosion was observed.

In contrast, *Pectenophilus ornatus*, a copepod parasite on the gills of the scallops, *Mizuhopecten (Patinopecten) yessoensis*, *Chlamys farreri nippensis*, and *C. f. farreri* from Japan (Nagasawa et al., 1988, 1991; Nagasawa and Nagata, 1992) is associated with evident hypertrophy of the host tissue at the attachment site of the highly modified copepod (Figure 10.22). The female assumes a modified sac-like morphology reaching 8 mm in diameter, resembling a rhizocephalan barnacle (Elston et al., 1985). The adult copepods are brilliant orange except for the area immediately surrounding the male vesicle, which is reddish (Nagasawa et al., 1988). The parasite is devoid of appendages of any sort and is completely unsegmented. Its body is attached to the host by means of a tapering stalk (Figures 10.22 and 10.23). The eggs are incubated within a spacious cavity communicating with the exterior by an unpaired birth pore through which the larvae are emitted. The nauplii closely resemble other copepod nauplii (Figure 10.24). *Pectenophilus ornatus* feeds on the haemolymph of the scallop and may infest up to 100% of the cultured populations. It causes reduced marketability and decreased physiological condition. Combatting the parasite or controlling its distribution will depend on improved knowledge of its life cycle, reproductive dynamics, and longevity.

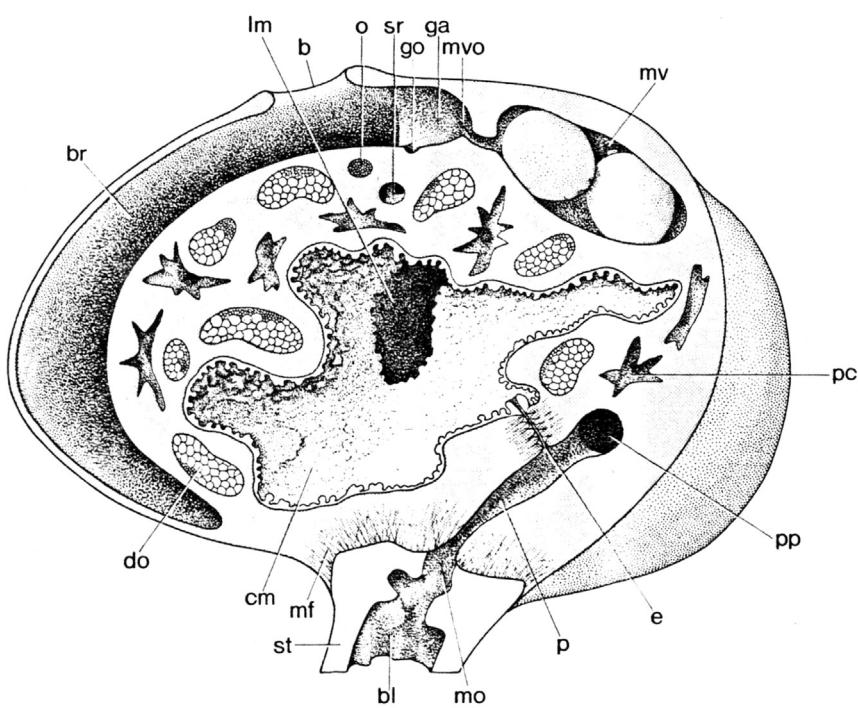


FIGURE 10.23 *Pectenophilus ornatus* sectioned in median plane. Microanatomy of female labelled as follows: b, birth pore; bl, blood lacuna; br, brood pouch; cm, midgut, central part; do, diverticulum of ovary; e, oesophagus; ga, genital atrium; go, genital orifice; Im, lateral diverticulum; mg midgut; mf, muscle fibres; mo, mouth; mv, male vesicle with two males; mvo, opening for male vesicle; o, unpaired part of ovary; p, pharynx; pc, pharyngeal channel; pp, pharyngeal pouch; sr, seminal receptacle; st, stalk. Source: Adapted from Nagasawa et al. (1988).

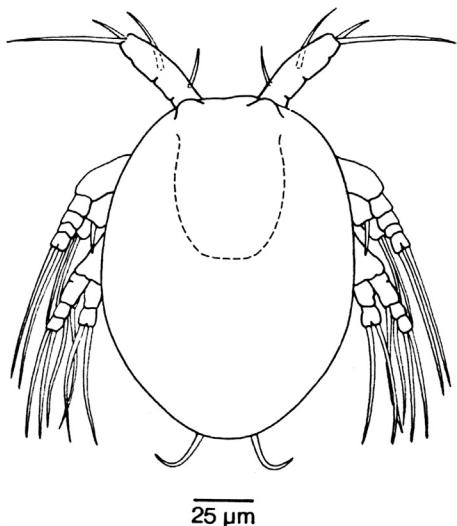


FIGURE 10.24 *Pectenophilus ornatus* nauplius in dorsal view. Source: Adapted from Nagasawa et al. (1988).

GASTROPODS

The Family Pyramidellidae comprises a few small (3–5 mm in length) odostomid gastropods which parasitise marine bivalves by feeding on haemolymph drawn from near the edge of the mantle or, in the case of clams, the siphon (Lauckner, 1983; McGladdery et al., 1993a). Leibovitz et al. (1984) observed large numbers of the gastropod, *Odostomia* spp., on the sides of holding tanks, and on the valves, mantle, and pallial cavity of captive bay scallops, *Argopecten irradians*. Developing embryos of the gastropod were also found within the soft tissues. These authors associated these gastropods with high mortalities among their broodstock and experimental bay scallops; however, *Odostomia* spp. were considered to be relatively benign haemolymph predators in other species.

The snail, *Odostomium seminuda*, was common on the upper valve of *Argopecten irradians* and on the 'ears' of *A. gibbus* from North Carolina (Wells and Wells, 1961). Constant irritation of the mantle margin of the calico scallop by penetration of the pyramidellid proboscis during feeding reportedly resulted in retraction of the mantle in affected areas.

Another pyramidellid, *Turbonilla interrupta*, is known to attack scallops along the eastern North American coast (Morton, 1967); however, no further details of pathology or distribution have been reported.

The common oyster drill, *Urosalpinx cinerea*, has demonstrated predatory behaviour in a laboratory study of attacks on bay scallop, *Argopecten irradians*. Predation resulted in 72% mortality. The snails also demonstrated a positive chemotactic response to scallop effluent in a choice chamber (Ordzie and Garofalo, 1980).

ALGAE

Many algae have been labelled as symbionts of marine bivalves, for example, *Zoothorella* spp., which colonise the mantle, eyes, and tentacles of wild, captive, and cultured adult bay (*Argopecten irradians*) and sea scallops (*Placopecten magellanicus*) south of Cape Cod. The infections produced green proliferative granulomatous lesions of the mantle and tentacles and haemocyte infiltration into the eyes. The disease was not seen in young scallops (Leibovitz et al., 1984).

Early work by Naidu (1971) showed another alga, *Coccomyxa parasitica*, to be parasitic on *Placopecten magellanicus* from Newfoundland. The peripheral mantle tissue, as well as portions of the gonad and adductor muscle was affected when exposed to ambient light. When infestation levels were high, there was a decline in the condition and weight of the viscera, including the adductor muscle. The mantle had a dark green colour, was slippery due to a layer of mucus and had a musty odour. Histological work demonstrated the encapsulation of the algal colonies by a network of connective tissue fibres. Because the normal mantle morphology is disrupted, shell deformities consisting of extra shell margins and grossly warped valves may occur. Naidu (1971) sampled almost 3000 scallops and found that 17.6% were infested. Scallops 8–10 years of age had the highest prevalence, while no algal cells were found in scallops younger than 3 years of age. Levels of infestation were positively correlated with light intensity.

Stevenson and South (1975) have described the causative agent in detail. They also showed that endocytosis occurs throughout the infestation and contributes to the spread of the algal cells. The alga also enters through normal feeding by the scallop, but seems highly resistant to digestion. The algal cells do not appear to be highly pathogenic or cause a rapid death of the host. Infestations of *Coccomyxa parasitica* are restricted to shallow water populations of *Placopecten magellanicus* inhabiting the more northern latitudes of its range (Stevenson and South, 1975).

Leibovitz et al. (1984) described a die-off of post-metamorphic juvenile cultured bay scallops, *Argopecten irradians*, in association with a bloom of the dinoflagellate, *Prorocentrum* sp. in the water supply of a Long Island Sound hatchery. The juvenile scallops showed signs of distress including gaping shells, injuries to the soft tissues by the dinoflagellates' minute spines, and impaction of the pallial cavity. Melanisation of the resulting lesions in the mantle followed, with secondary invasion of bacteria and ciliates. Wikfors and Smolowitz (1993) demonstrated that *Prorocentrum minimum* (a dinoflagellate considered nontoxic to mammals) produced toxicity in post-metamorphic bay scallops *Argopecten irradians* resulting in significant necrosis of digestive gland absorptive cells and thrombi in the vascular system. In the same study, older juveniles of *A. irradians* were not affected suggesting that as the larvae age new enzymes may occur that can digest the organisms.

Around the same time, many coastal embayments of Long Island, New York, experienced algal blooms during the summers of 1985 and 1986 that were unprecedented in their persistence and cell density ($>10^9$ /litre). Small microalgae decreased light penetration to less than 1 m, severely affecting eelgrass (*Zostera*) beds. The 1985 bloom coincided with the spawning period of the bay scallop, *Argopecten irradians*, and was blamed for the massive recruitment failure of that year-class (Cosper et al., 1987). TEM indicated that the responsible microalgae was a previously undescribed species, similar to the chrysophyte species tentatively designated *Aureococcus anophagefferens*, which also bloomed in Narragansett Bay, Rhode Island in 1985 (Sieburth et al., 1986). Grazing experiments revealed that bay scallops were inefficient at retaining the 'brown tide' cells. Possible mechanisms investigated were toxicity effects, reduced ingestion rates, reduced absorption efficiencies at high densities and poor nutritional qualities of the brown tide alga (Cosper et al., 1987). Tettelbach et al. (1999) showed evidence supporting the hypothesis that bay scallops, *Argopecten irradians*, spawning during brown tides results in poor recruitment and reduced commercial harvest in subsequent years.

Following a picoplankton bloom in Long Island Sound, NY, 1985, [Bricelj et al. \(1987\)](#) measured a 76% reduction in adductor muscle weight of adult bay scallops compared with 1984 data; however, survivors rebounded in September to triple their mean muscle weight. Mortality rates were not documented during either 1985 or 1986 so the impact on survival was unknown.

[Hegaret et al. \(2012\)](#) investigated the impact of the dinoflagellate *Alexandrium catenella* on the histopathological responses of juvenile scallops, *Argopecten purpuratus*. After 6 days of exposure, increased melanisation of mantle, labial palps, gills, and gonad epithelia was documented (68% prevalence compared to 29% for the control treatment). Muscle alterations were also seen with narrow holes in the muscle fibres filled in by haemocytes. Finally, increased prevalence of inclusions with rickettsiales-like organisms in the digestive gland tubule cells was observed in the scallops exposed to *A. catenella*. Similar investigations exposing the Pacific calico scallop, *Argopecten ventricosus*, to the dinoflagellate *Gymnodinium catenatum* by [Escobedo-Lozano et al. \(2012\)](#) produced almost the same histopathological results – epithelial melanisation of mantle and gill tissue, as well as haemocyte aggregations in muscle, gills, and mantle.

FORAMINIFERANS

The calcareous foraminiferan, *Cibicides refulgens*, is a conspicuous and abundant component of the epifaunal community, living on the valves of the Antarctic scallop, *Adamussium colbecki*. One of its modes of nutrient acquisition is parasitism by eroding through the scallop shell and using dissolved free amino acids from the highly concentrated pool in the extrapallial cavity ([Alexander and DeLaca, 1987](#)). These authors showed that 50% of attached *C. refulgens* significantly erode the surface of the scallop shell to excavate channels into the extrapallial cavity. Whether the cumulative effects of shell erosions have a detrimental influence on the bivalve in the marginal Antarctic environment, remains unknown, but seems likely. [Hayward and Haynes \(1976\)](#) reported the association of other foraminiferans including *Cibicides lobatulus* on the queen scallop, *Aequipecten (Chlamys) opercularis*.

PORIFERA

As with polydorid polychaetes, clionid sponges are also known as ubiquitous shell-colonising organisms in both live and dead marine bivalves ([Lauckner, 1983](#)). Shell destruction follows excavation through etching and discarding of shell fragments to permit sponge proliferation and development of inhalant and exhalant passages throughout the shell.

[Evans \(1969\)](#) detailed the extent of shell destruction by *Cliona vastifica* in *Placopecten magellanicus* from Newfoundland, Canada. The radiographs of infested shells showed how little shell material was left between the inner and outer surfaces of heavily colonised individuals. Externally, clionid invasion of the shell can be identified by small holes, which open into the inhalant and exhalant tunnels, usually starting at the hinge end of the shell with predominant damage to the ventral valve ([Warburton, 1958](#)).

Clionid invasion of the shell may also affect the soft parts of scallops and cause a condition [Medcof \(1949\)](#) termed 'dark-meat'. The adductor muscles of affected individuals have a greyish brown tinge, are flaccid and stringy, and reduced in size. The meat yield of lightly infested scallops was about 59%, while that of heavily infested scallops was only 46% of the yield of healthy individuals. Off Digby, Nova Scotia, Canada, only those scallops 8–9 years or older were affected by clionid sponges. Invasion by the pest starts near the hinge and spreads until the entire shell is honeycombed with burrows. The shell can reach over three times its normal thickness because of repeated nacre deposited on the inner shell surface to attempt shell repair. Several other reports of *Cliona celata* and *C. vastifica* shell damage have also been reported from *P. magellanicus*, but most are not associated with adductor muscle discolouration ([Evans, 1969; Sindermann, 1971; McGladdery et al., 1993a](#)). [Kurochkin et al. \(1986\)](#) report clionid damage to the shells of *Patinopecten (Mizuhopecten) yessoensis* in the Sea of Japan/Peter the Great Sea. These infestations were also associated with soft-tissue retraction and reduction of physiological condition. [Duckworth and Peterson \(2013\)](#) suggested that lower seawater pH due to global warming may increase boring rates of *C. celata* in scallops, with implications for both wild and cultured populations.

[Leibovitz et al. \(1984\)](#) described clionid-induced shell deformities in juvenile and adult bay scallops, *Argopecten irradians*, probably caused by a *Scypha* sp. These are tubular sponges that attach to the internal surface of the valve, as well as the ventral margins and external surface. No significant mortalities were seen, so the fate

of these deformed scallops is unknown. More recently, a reciprocal shell colonisation relationship was reported on *Aequipecten (Chlamys) opercularis*, where the sponge *Siberites ficus* ssp. *rubrus* was associated with reduced shell colonisation by other fouling agents (Armstrong et al., 1999). Within the geographic range of this species, this provides a natural alternative to chemical/salinity/shock treatments for problem colonisation and may provide potential for aquaculture use to control shell matrix colonisation.

Several species of scallop have evolved a mutualism with epizoic sponges on their shells. The sponge protects the scallop from predatory starfish, while being protected from nudibranchs and/or deriving additional nutrients generated by the inhalant current of the host (Bloom, 1975; Forester, 1979; Chernoff, 1987; Pitcher and Butler, 1987; Gappa and Landoni, 2007).

CNIDARIA

The colonial hydroid, *Hydractinia echinata*, a common inhabitant on the external surface of the shell of the sea scallop, *Placopecten magellanicus*, sometimes attaches to the internal shell surface. This intrusion interferes with the normal activities of the scallop mantle, often causing shell deformities. The scallop reacts by secreting a new shell edge within the existing perimeter and bypassing the hydroid colony. Batteries of nematocysts are abundant at the leading edge of the colony and the mantle withdraws presumably because of the discharge of the nematocysts into it. Successful control of this intrusion is accomplished when the scallop overgrows the impinging hydroid colony. No evidence has been found that *H. echinata* may ultimately cause the death of the scallop (Merrill, 1967).

A more recent investigation of age zero Japanese scallop (*Mizuhopecten yessoensis*) mortalities associated with simultaneous heavy loads of the hydroid, *Eutima japonica*, in Funka Bay, Hokkaido, showed that *Mytilus galloprovincialis* was the likely source of the infectious planulae produced by medusa released by polyps on the mussels (Baba et al., 2007). Their study found some scallop-rearing sites with high incidence of *E. japonica* polyps but low mortality. The researchers suggested the transfer of these juvenile scallops to different cages 1-month prior probably produced additional stress precipitating the mortality event. Polyps of these hydroids inhabit soft body parts of host bivalves. Monthly monitoring of these juveniles showed the presence of polyps probably impeded feeding, which reduced shell height growth by 43% and triglyceride accumulation in the digestive gland by 24–47%.

NON-INFECTIOUS DISEASES

In addition to the organisms that colonise scallops or cause infectious diseases, scallops also suffer from diseases that are non-infectious. The most familiar cases are associated with aquaculture when animals are kept in sub-optimal conditions, particularly in a hatchery setting, where the presence of opportunistic bacteria (often *Vibrio* spp. as described in section 'Vibrioaceae') can take advantage of stressful conditions. Scallops cultured in open-water systems, however, have also encountered problems such as smothering by various organisms, including mussels (Minchin and Duggan, 1989; Kurata et al., 1996) and barnacles (Ovsyannikova and Levin, 1982). In wild stocks, abiotic factors, such as unknown environmental disturbances, were associated with abnormal melanisation and microstructural distortions of the shell of *Pecten maximus* in Brittany, France (Larvor et al., 1996). Anthropogenic pollutants have also been attributed to diseases affecting both cultured and wild stocks. For example, Syasina et al. (1996) and Usheva (1999) have described weakening muscle atrophy associated with necrosis and gametogenesis abnormalities in *Mizuhopecten yessoensis* from polluted areas of Peter the Great Bay, Sea of Japan. Also, Karaseva and Medvedeva (1994) reported embryo toxicity in the same species of scallop after parental exposure to copper and zinc. Increases in atmospheric carbon dioxide and global temperatures will lead to changes in ocean chemistry that may impact calcifying marine taxa like scallops. Talmadge and Gobler (2011) tested the effects of elevated temperature and carbon dioxide on bay scallops, *Argopecten irradians*, hard clams, *Mercenaria mercenaria*, and the eastern oysters, *Crassostrea virginica*, and found negative impacts on both larval and juvenile life stages. These impacts were described and compared between species and life stages. One example will be included here. Increases in temperature and carbon dioxide each significantly depresses survival, development, growth, and lipid accumulation of *M. mercenaria* and *A. irradians* larvae. The authors rightfully warn that 'the sum of the environmental stressors that affect marine organisms in the coming decades, particularly in coastal ecosystems, is substantial...these effects may have serious implications for the future of these bivalves and marine calcifying organisms faced with global climate change.'

SUMMARY

With growing interest in scallop enhancement, stock regeneration, and molluscan aquaculture species diversification comes a growing opportunity for improved health and disease understanding for these pectinids. Suspension culture, hatchery manipulated reproduction, and increased proprietary observation are bound to reinforce the many gaps evident in this parasite, pest, and disease summary. Many parasites, which have been reported in wild populations and subsequently forgotten, may become more readily available for study, or more problematic under culture conditions. In addition, the new tools being developed for other mollusc groups (primarily oysters) will be more accessible in the near future for distinguishing between significant scallop pathogens and innocent commensals – especially at the microbial level. With this in mind, it is clearly anticipated that this chapter will also be superseded in the next 10 years – these authors look forward to reading it.

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