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Environmental effects on the biology of the cestode *Schistocephalus solidus* and its interactions with aquatic hosts

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Abstract

The broad aim of the work undertaken in this thesis was to increase understanding of how environmental changes in aquatic environments impact host-parasite interactions in the experimentally amenable copepod - three spined stickleback - *Schistocephalus solidus* model using controlled, laboratory experiments. In natural environments, animals are being exposed to a suite of anthropogenic stressors including temperature and heavy metal pollution. In this thesis the effects of temperature and heavy metals pollution are studied to determine the effect of these stressor on the growth development, survival and life cycle of both hosts and parasites.

This thesis first investigated the effect of the temperature on the survival of infective free-swimming stages (coracidia) of *Schistocephalus solidus*, and their subsequent development as proceroid larvae in copepods (first intermediate hosts). The survival of coracidia was temperature-dependent, with the longest survival times at 10°C, and being reduced at 15°C and 20°C. The growth of proceroids in copepod hosts was faster at 20°C than at 10°C or 15°C. The second part of this thesis investigated the effect of water hardness and the influence of heavy metals on the survival of *S. solidus* coracidia, and on the development of proceroids in copepods. The survival of coracidia was shown to be sensitive to low water hardness (i.e. soft water), but was unaffected by heavy metals at the concentrations used. Proceroids developing in copepod hosts attained a larger size when reared under heavy metal treatments in hard water. The third part of this thesis investigated the effect of elevated zinc concentration (0.2 µg/L, 2 µg/L, 20 µg/L and 200 µg/L) on the viability of developing *S. solidus* eggs, on the survival of coracidia and on the development and growth of proceroids in copepod hosts. Under elevated zinc concentrations, *S. solidus* eggs were able to develop and hatch, and coracidia were shown to survive longer at higher zinc concentrations. After being exposed to zinc, proceroids grew more quickly in copepod hosts. The last part of this thesis investigated how the growth of early larval stages of the parasite in copepod hosts exposed to zinc affected the subsequent performance of the second larval (plerocercoid) stage in the second intermediate host, the three-spined stickleback *Gasterosteus aculeatus*. *Schistocephalus solidus* proceroids, which grew larger in copepods exposed to zinc, developed into larger plerocercoids when transmitted to the three-spined stickleback hosts, suggesting carry-over effects in this complex parasite life cycle.

The overall findings of the thesis are that environmental stressors in aquatic environments, such as temperature and heavy metal pollutants, can have complex and divergent implications for different parasite life cycle stages, with potentially complex implications for the dynamics of parasite life cycles in ecosystems subject to environmental changes.

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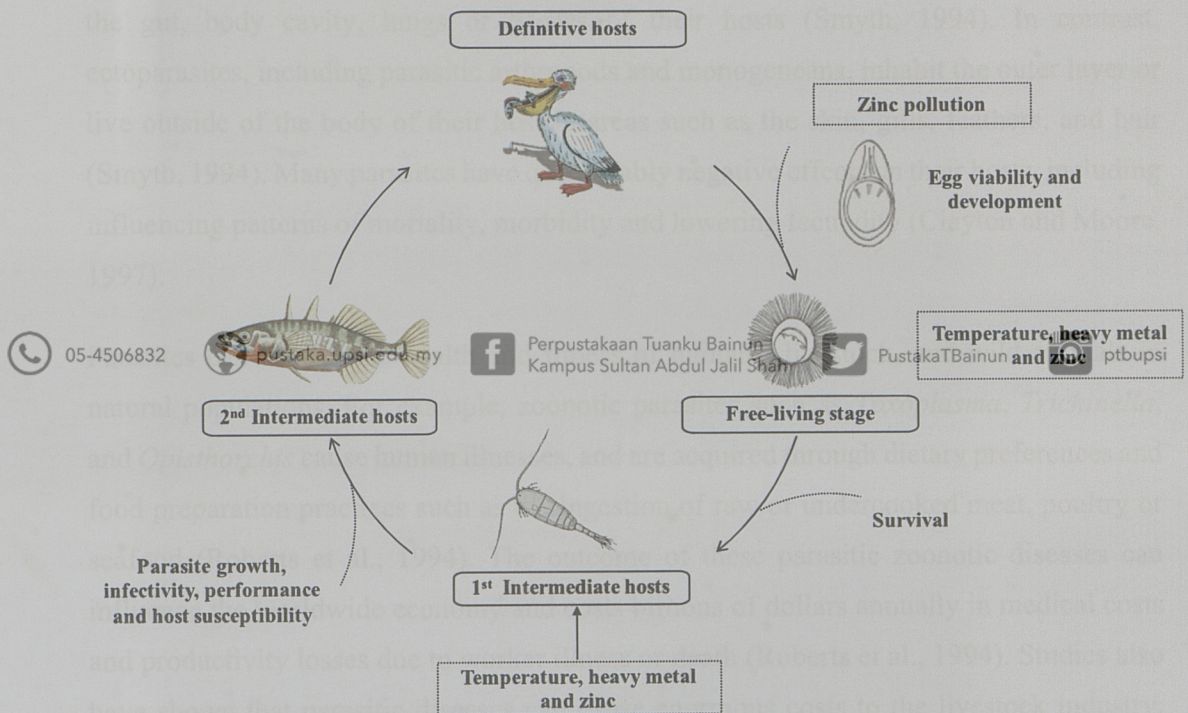
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Chapter 1

General Introduction



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1.1 Parasitism

Parasitism is a relationship between individuals of two different species in which one (the parasite) uses the other (the host) as its environment from which it derives nourishment and can implies harm to the host (Esch and Fernandez, 2013). Parasites are a highly diverse group of organisms that have evolved different strategies for infecting and exploiting their hosts. Parasites have adapted to survive, feed, develop and reproduce on or in their hosts, and in doing so, exploit them (Clayton and Moore, 1997; Poulin, 2011). Parasites can be broadly classified as either endoparasites or ectoparasites. Endoparasites live inside the body of their hosts, and include a wide variety of protozoans, digeneans, cestodes, nematodes, and acanthocephalans (Bush, 2001). Endoparasites can be found in the gut, body cavity, lungs or tissues of their hosts (Smyth, 1994). In contrast, ectoparasites, including parasitic arthropods and monogeneans, inhabit the outer layer or live outside of the body of their host in areas such as the skin, gills, feathers, and hair (Smyth, 1994). Many parasites have quantifiably negative effects on their hosts, including influencing patterns of mortality, morbidity and lowering fecundity (Clayton and Moore, 1997).



Parasites can impact the health and fitness of humans, livestock, and wild animals in natural populations. For example, zoonotic parasites such as *Toxoplasma*, *Trichinella*, and *Opisthorchis* cause human illnesses, and are acquired through dietary preferences and food preparation practices such as the ingestion of raw or undercooked meat, poultry or seafood (Roberts et al., 1994). The outcome of these parasitic zoonotic diseases can influence the worldwide economy and costs billions of dollars annually in medical costs and productivity losses due to worker illness or death (Roberts et al., 1994). Studies also have shown that parasitic diseases can cause enormous costs to the livestock industry. For example, the widespread incidence of parasitic roundworms (*Haemonchus contortus* and *Trichostrongylus* spp.) cost the Australian sheep farming industry millions of dollars annually in production losses (McLeod, 1995).

Changing in the environment condition that inhabited by parasites and their host can influence the disease outbreak as the relationship of the host-parasite also affected by the changes (Poulin, 1992). As a consequences, these changes may affect the evolutionary of parasite and host. For example, environmental changes could increase the transmission rate of the parasite which can cause to the selection for higher virulence (Anderson and May, 1982; Poulin, 1992).





1.2 Parasite life cycles

Life cycles are generally classified as direct or indirect. Direct life cycles do not require an intermediate host (Figure 1.1). For direct life cycles, only a single definitive host species is required – the species in which the parasite reaches sexual maturity and produces progeny (Friend and Franson, 1999). For example, many ectoparasites, including the fleas of mammals and birds (Bitam et al., 2010) have direct life cycles. Other examples include gregarine parasites, such as Apicomplexans which are single-celled eukaryotes that are transmitted by oocysts ingested by a host (David et al., 2012). Hosts ingest oocysts, containing infective sporozoites, which attach to or penetrate hosts' intestinal epithelium cells (Figure 1.1). Also, some nematodes are directly transmitted, for example *Syphacia obvelata*, the roundworm of pigs when the eggs do not hatch, pre-parasitic larvae develop inside their eggs and hatching will take place after these eggs are eaten by another host and the infective larva escapes (Marjorie, 1950).

In contrast, indirect parasite life cycles involve one or more intermediate hosts in addition to the definitive host (Figure 1.2). Intermediate hosts are required by the parasite for completion of its life cycle because of the morphological, developmental and physiological changes that usually take place in the parasite within those hosts (Friend and Franson, 1999). Top level predators, including piscivorous birds, often serve as the definitive hosts for the parasites such as nematodes, cestodes, acanthocephalans, and trematodes (Bråten, 1966; Chappell et al., 1994; Smyth, 1994; Barber and Scharsack, 2010).



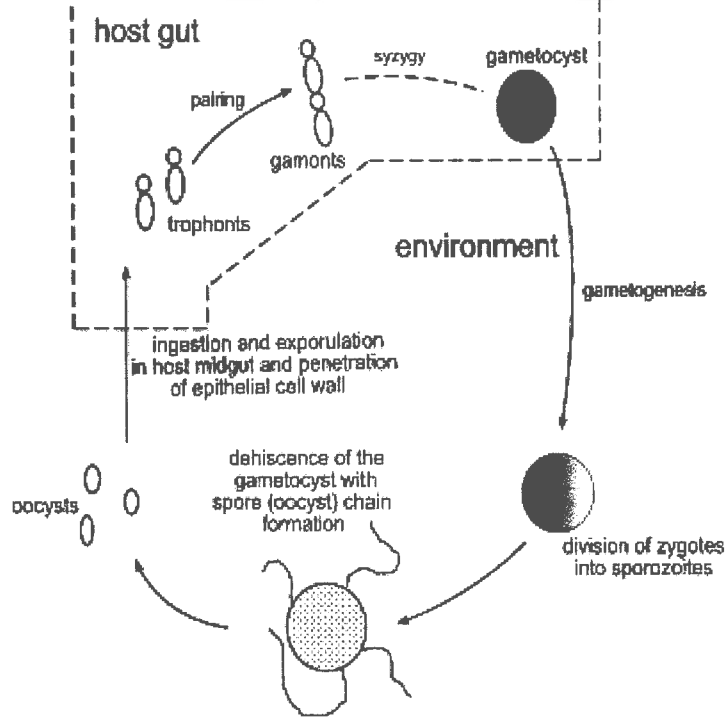


Figure 1.1 The direct life cycle of a gregarine parasite showing the transmission of the infection and the trophont production rate of infective agents (oocysts). Adapted from David et al., (2012).

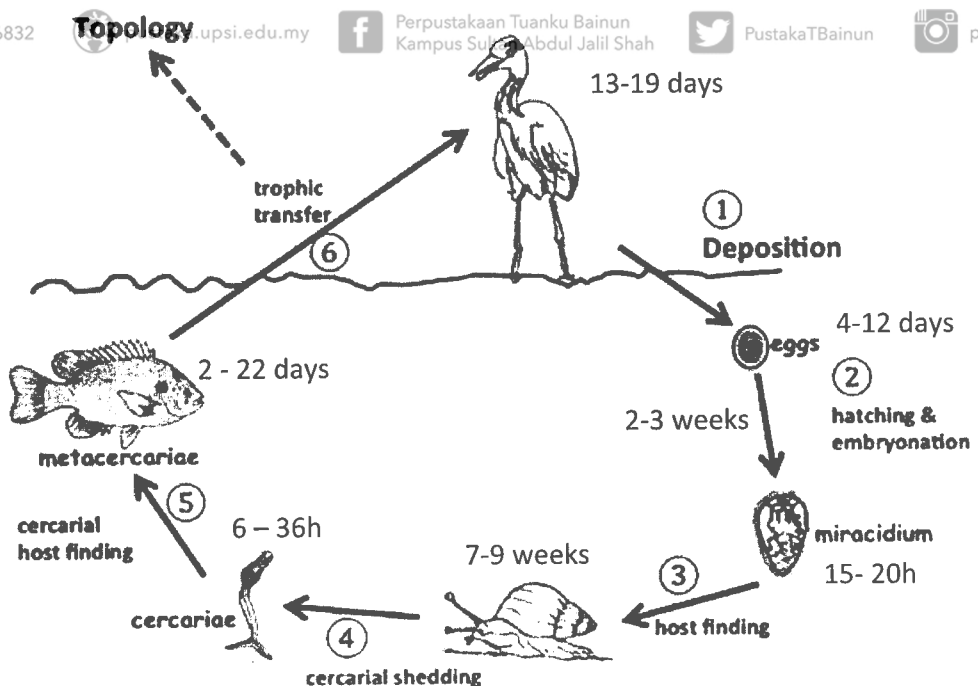


Figure 1.2 Example of an indirect parasite life cycles of a typical, unnamed trematode. Adapted from Sukhdeo (2012).



1.3 Host-parasite interactions in a changing world

Parasite infections and their effects on hosts might vary depending on the environmental context in which hosts and parasites interact. Understanding not only the typical outcome but also how these outcomes differ depending on factors such as environmental conditions (e.g. temperature and pollution) is important because these factors have implications for host populations and the strength of parasite-mediated selection over evolutionary time (Poulin, 2011; Goater et al., 2013). A number of reviews have highlighted the potential impact of climate change and pollution on parasitism such as disease outbreaks, transmission rates of parasites and pathogens and virulence of pathogens and parasites (Marcogliese, 2001; Sures, 2008). However, understanding how environmental changes affect parasites that have complex, multi-stage life cycles is challenging (Figure 1.3) (Morley et al., 2003; Barber et al., 2016).

In indirect life cycles, different life stages of a parasite occupy different hosts that often live in a range of habitats, with each parasite life stage adapted to ensure transmission efficiency and survival (Poulin, 2011). The complex life cycles of parasites, such as those exhibited by trematodes, nematodes and cestodes, often include free-living stages that experience external aquatic conditions directly without being buffered by host physiology, and so are directly vulnerable to environment perturbations such as chemical pollutants or altered temperature regimes. Such life cycle stages might therefore be expected to be highly sensitive to environmental change, with predicted consequences for the subsequent prevalence of infection in susceptible hosts (Evans, 1985; Morley et al., 2001).

Environmental changes may also affect potential host organisms by altering their survival, reproductive success or behaviour, or by otherwise influencing their susceptibility to parasite infection. Following infection, parasites that have established in a host could also be affected by external environmental factors indirectly, through effects on the host organism. Such influences (Figure 1.3) may affect the development growth or infectivity of the parasite, or the growth and survival of the host (Macnab and Barber, 2012).



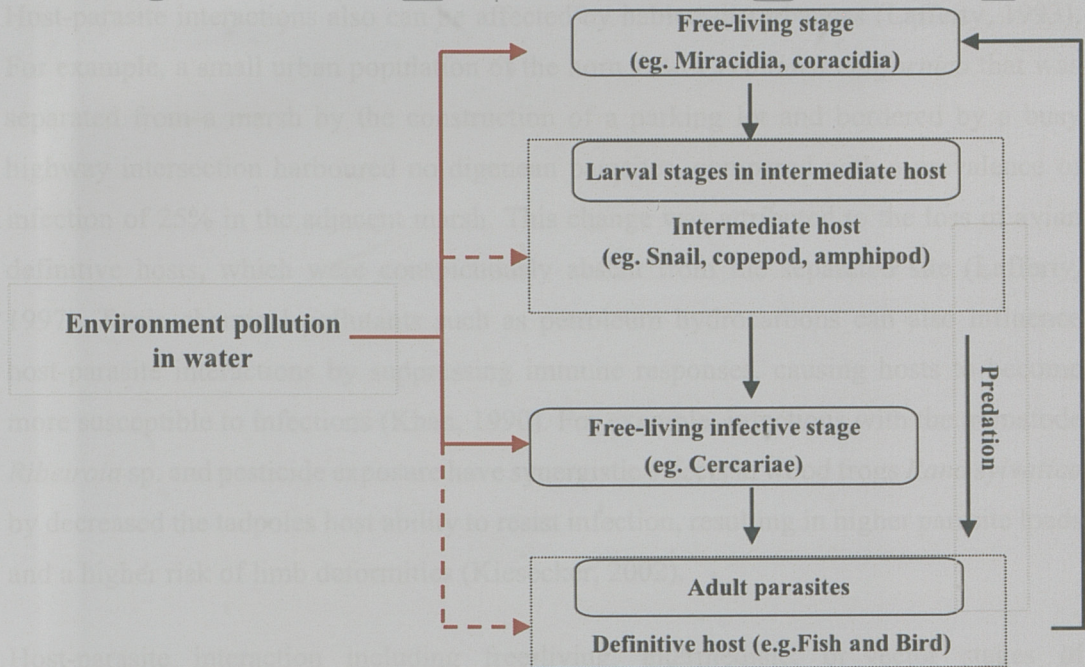


Figure 1.3 Possible effects of a water-borne environmental pollutant on a host-parasite system involving an indirectly-transmitted parasite. Solid red lines leading to a direct environment effect on the free-living stage of the parasite. Broken red lines represent an indirect effect of environmental pollution on the parasite stage via the intermediate or definitive host.

Environmental pollution can affect the host-parasite interaction and identifying the effect would be challenging especially for a parasite that have a complex life cycle. It is important to determine the environmental parameter involved and one way to do it is by generate a data from the controlled laboratory work. Using a *Schistocephalus solidus* experimental host-parasite model, the information on the multi- life cycle and the host-parasite interaction can be develop (Barber and Scharsack, 2010).

1.4 Effects of environmental change on emerging parasitic diseases

Changes in environmental condition can alter the growth and reproduction of parasite and host at once influence the existence of the zoonotic parasitic disease (Graczyk and Fried, 1997; Poulin, 2011). The host-parasite relationship can also be influence by other environmental perturbation such as deforestation and changes in patterns of land use and human settlement (Patz et al., 2000). For examples, to provide area for agricultural and houses (human settlements), more forests area are cleared and this situation can affecting parasitic vector populations (Patz et al., 2000).



Host-parasite interactions also can be affected by habitat disturbances (Lafferty, 1993).

For example, a small urban population of the horn snail *Cerithidea californica* that was separated from a marsh by the construction of a parking lot and bordered by a busy highway intersection harboured no digenean parasites, compared with a prevalence of infection of 25% in the adjacent marsh. This change was attributed to the loss of avian definitive hosts, which were conspicuously absent from the separated site (Lafferty, 1997). Toxic chemical pollutants such as petroleum hydrocarbons can also influence host-parasite interactions by suppressing immune responses, causing hosts to become more susceptible to infections (Khan, 1990). For example, infections with the trematode *Ribeiroia* sp. and pesticide exposure have synergistic effects in wood frogs *Rana sylvatica* by decreased the tadpoles host ability to resist infection, resulting in higher parasite loads and a higher risk of limb deformities (Kiesecker, 2002).

Host-parasite interaction including free-living, intermediate, or vector stages of pathogens infecting terrestrial animal also can be altered by climate change (Harvell et al., 2002). In this thesis, the effects of heavy metal pollutions and temperature change on a host and parasite life cycle have been studied.



1.5 Effects of temperature change on host-parasite interactions

The effect of temperature change on species will depend on their thermal tolerance and how close they are present to their physiological limit (Deutsch et al., 2008). For example, ectotherms are particularly at risk from climate change, as their physiological processes are directly influenced by the environmental temperature that they are experiencing (Harvell et al., 2002). In contrast, endotherms, which possess thermoregulatory mechanisms that allow them to maintain their body at a constant temperature (Deutsch et al., 2008), are expected to be less severely affected. While the majority of parasitic taxa are ectothermic, hosts may be endo- or ectotherms. In ectothermic hosts, parasites experience the same thermal conditions as the host and may be expected to be adapted to historical local environmental temperature regimes. Elevated temperatures therefore may be expected to have implications for both parasites and hosts in ectotherm host-parasite systems.

Altered temperature associated with climate change can potentially influence host-parasite interactions at many different points in the life cycles of multi-host parasites. For example, warmer temperatures may drive faster metabolic rates and speed up





development rates, reducing the length of the embryonation period, meaning that eggs hatch quicker (Sakanari and Moser, 1985; Paull and Johnson, 2011). However, increases in metabolism may also deplete the limited energy reserves of motile free-living – but non-feeding – stages that emerge from eggs (Barber et al., 2016). Consequently, warmer temperatures have the potential to decrease the survival probability and hence persistence of infective stages after hatching (Nollen et al., 1979). As a result, temperature effects on the production of infective stages are predicted to be nonlinear, with intermediate temperatures expected to generate the highest overall hatching success. The temporal consistency of future thermal regimes will also likely have a major influence on egg development times (Barber et al., 2016).

However, most species appear to have the capacity to tolerate temperature ranges within those commonly encountered under natural conditions (Evans, 1985). For example, the activity and infectivity of cercariae *Echinostoma liei* increased at higher temperatures to encounter for their lower survival time (Evans, 1985).

Temperature can also directly influence the multi-host life stage of parasites by controlling the development and growth of parasitic stages. For example, under low temperatures, the development of larvae of *Anguillicola crassus* parasite in eels is reduced (Knopf et al., 1998). In trematode parasites system, the development of metacercariae *Maritrema novaezealandensis* in the amphipod *Paracalliope novizealandiae* also reduced at lower temperatures of 15°C and 20°C - non-infective immature and early cyst stages, compared at 25°C almost a third had completed their development and were infective to the next host (Studer et al., 2010). Therefore, increasing temperatures can enhance parasite development, as long as this temperature can be tolerated by the host (Studer et al., 2010). Macnab and Barber (2012) also showed that plerocercoids growing in fish held at 15 °C grew less quickly, with plerocercoids growing in fish held at 20°C having a larger mass at the end of the study and exceeding the 50 mg threshold for infectivity to the final host, a fish-eating bird. Elevated temperatures, therefore, have the potential to increase the rate at which infective parasite stages are transmitted to definitive hosts. Temperature is likely to have a significant effect on parasites with multi-host life cycles, with free-living stages and ectothermic hosts most likely to be affected (Harvell et al., 2002) but there is also the potential for opposing effects of temperature change on different parts of the life cycle.





1.6 Effects of heavy metal pollutants on host-parasite interactions

1.6.1 General effects of heavy metal pollutants on aquatic organisms

Toxic pollutants are becoming ubiquitous in freshwater ecosystems. Substances such as heavy metals, cyanides, ammonia and pesticides from various chemical industrial and agricultural practices and domestic waste can impact the biology of aquatic organisms (Poulin, 1992; Saeed and Shaker, 2008; Nhi et al., 2013). Pollutants present in the water column may accumulate in the sediment of aquatic biotopes where they can be dissolved again and enter the food chain (Sures, 2006). The uptake and accumulation of pollutants by aquatic organisms may cause harm, depending on the specific substance (Sures, 2006). The concentration at which a compound has lethal effects can depend upon many contributing factors, including species and water quality (Scott and Sloman, 2004), but sub-lethal concentrations can also have ecologically significant effects.

The effects of pollutants may be immediate (i.e. acute toxicity) or may only arise after prolonged exposure (i.e. chronic toxicity) (Sures, 2008). For example, in fish, pollutants are taken up through the gills or the intestine and are accumulated until they reach a steady-state concentration (Sures, 2008). After reaching this steady-state concentration, some pollutants are excreted by the organism (Figure 1.4a). The uptake and accumulation of pollutants are also normally connected with a general physiological response (Figure 1.4b) mainly comprising adverse effects, which can be either specific (e.g. induction of certain proteins, or DNA damage) or more general (e.g. changes in hormone levels or effects on the general metabolism), any of which can cause death (Sures, 2008).

Exposure to heavy metal pollutants can affect animals by changing their physiology or behaviour, including the rate of feeding, digestion, respiration, or excretion, with consequences for their energy budget, growth and reproduction (Luoma and Rainbow, 2008). For example, the hydroid *Campanularia flexuosa* showed a reduced colony growth rate with increasing dissolved metal concentrations (Stebbing, 1976), whereas the freshwater amphipod *Gammarus pulex* had a reduced feeding rate at 101 µg/L copper compared to 8.3 µg/L copper (Taylor et al., 1993).



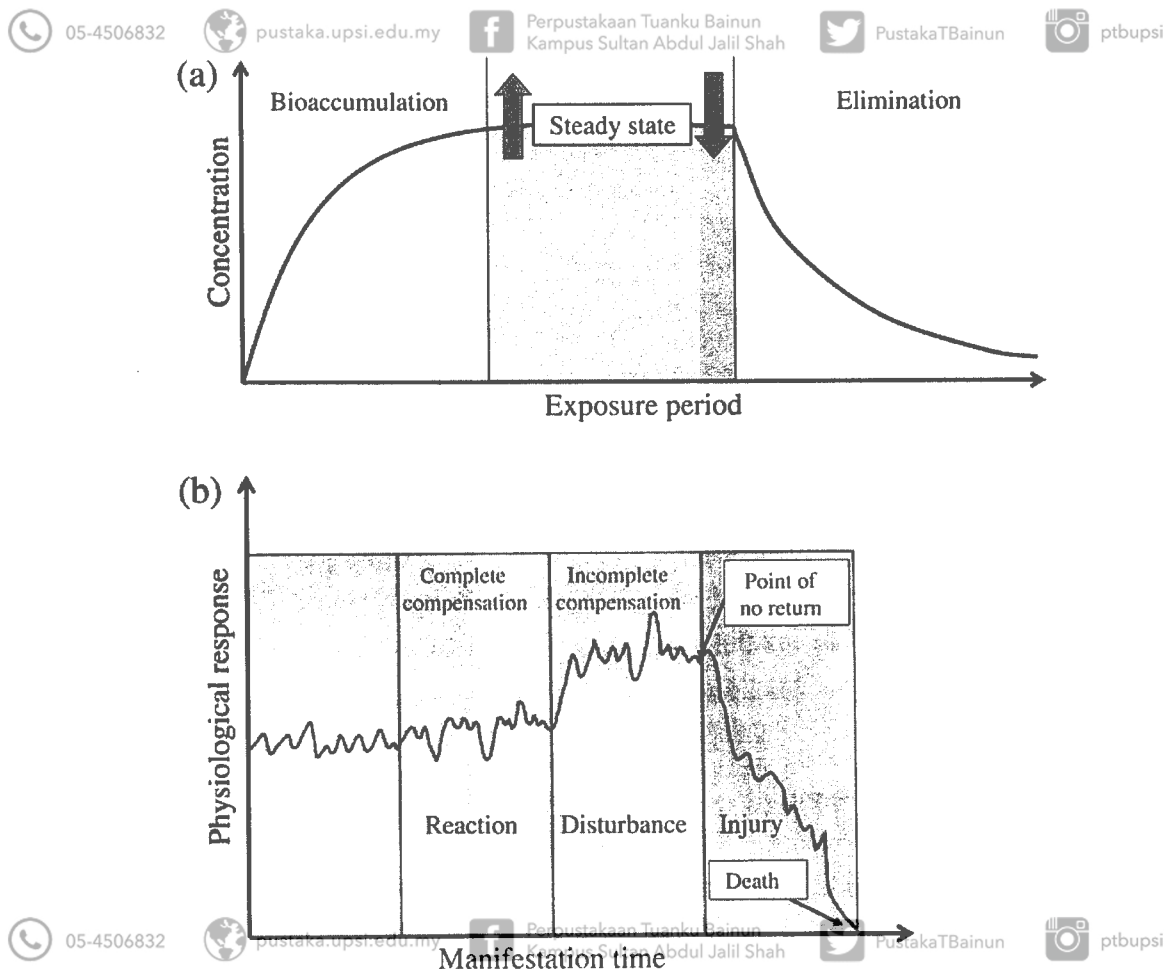


Figure 1.4 Example of (a) Uptake and accumulation of pollutants in organisms and (b) associated intensity of physiological response Sures (2008).

Heavy metal pollution can also affect the behaviour of aquatic animals. For example, mussels *Dreissena polymorpha* exposed to cadmium pollution exhibited altered shell-opening frequency (Sures, 2004) whereas the burrowing behaviour of the marine bivalve, *M. liliانا* was significantly decreased at a sediment concentration of 25 $\mu\text{g/g}$ copper, and at 80 $\mu\text{g/g}$ zinc (Roper et al., 1995).

In this thesis the effects of three different heavy metals are studied; cadmium, copper and zinc as these heavy metals are naturally occurring metals commonly present in surface waters and can reach high concentrations near mining areas (Martins et al., 2017). They have been widely used in industry and are released into the environment as a byproduct of ore smelting, mining activity, domestic waste emission or application of fertilizers and pesticides (Morley et al 2002; Sanchez et al., 2017). In the UK, concentrations chronically polluting the aquatic environment have been recorded as high as $160 \mu\text{g/L}^{-1}$

for cadmium and 8800 $\mu\text{g/L}^{-1}$ for zinc (Morley et al 2002). Meanwhile, copper is a widespread pollutant found in surface waters at concentrations up to 100 g/L^{-1} (Sanchez et al., 2017).

1.6.2 Effects of cadmium in aquatic environments

Cadmium is a biologically non-essential heavy metal that is typically found naturally at low (i.e. parts per billion) concentrations in rivers, lakes, and ponds. Cadmium can enter ecosystems through the application of phosphate fertilizers and other industrial sources (Williams and David, 1973). However, cadmium can become problematic in aquatic environments when its concentration rises above natural background levels. The most toxic form of cadmium is the divalent ion (Cd^{2+}) (Solomon, 2008). Exposure to this form induces the synthesis of low molecular weight metallothionein proteins, which bind with cadmium and decrease its toxicity (Wright and Welbourn, 2002). This normally takes place in the liver of fish and mammals. However, if cadmium concentrations are elevated, the metallothionein detoxification system can become overwhelmed and the excess cadmium will be available to produce toxic effects (Wright and Welbourn, 2002).

Cadmium occurs in low concentrations within the water column of rivers and estuaries and accumulates in the sediments (Bennet-Chambers et al., 1999). Cadmium has been reported to reach concentrations of 0.11–200 $\mu\text{g.kg}^{-1}$ and up to 500 $\mu\text{g.kg}^{-1}$ dry weight in the sediment of a sump adjacent to Lake Leschenaultia, Western Australia (Bennet-Chambers et al., 1999). The concentration of cadmium is generally dependent on whether the sediments were humic (high cadmium) or siliceous (low cadmium).

1.6.3 Effects of copper in aquatic environments

The main process leading to the mobilization of copper into the environment is extraction from its mining, milling and smelting, and also from agriculture and waste disposal (Wright and Welbourn, 2002). In aquatic ecosystems, copper generally enters from agriculture runoff, though the deliberate use of copper sulphate to control algal blooms and also from direct discharges from industrial processes (Wright and Welbourn, 2002). In uncontaminated freshwater, copper concentrations generally range from 0.001 to 0.1 $\mu\text{g/L}^{-1}$ and from 0.03 to 0.6 $\mu\text{g/L}^{-1}$ in uncontaminated ocean water. In contaminated water, copper usually leaves the water column and accumulate in sediment. Copper concentrations ranging from 50 to 100 $\mu\text{g/L}^{-1}$ can occur in aquatic ecosystems that receive mine tailings or where copper sulphate has been added to control algal blooms (Wright



and Welbourn, 2002).

Copper is an essential trace metal, required in small doses by organisms for metabolic functions, but it is potentially highly toxic if the internal available concentration exceeds the capacity of physiological detoxification processes (Sunda and Hanson, 1987). Fish and crustacean generally are sensitive to copper (Wright and Welbourn, 2002). For example, the cyprinid fish *Rutilus kutum* had a lower body weight and survival rate when exposed to 0.23 mg/L⁻¹ of copper sulphate compared to non-exposed fish (Gharedaashi et al., 2013). The effect of copper on osmoregulation in the freshwater amphipod *G. pulex* exposed to 100 µg/L⁻¹, caused a significant reduction in haemolymph sodium concentration and sodium influx within 4h. Copper concentrations as low as 10 µg/L⁻¹ also significantly reduced gill Na⁺/K⁺ ATPase activity (Brooks and Mills, 2003).

1.6.4 Effects of zinc in aquatic environments

Zinc can enter water naturally by the erosion of minerals from rocks and soils, and also as a by-product of steel production, the operation of coal-fired power stations, or the burning of waste materials (Irwin et al., 1997). Zinc is also used in some fertilizers that may leach into groundwater. High natural levels of zinc in water are usually associated with higher concentrations of other heavy metals, such as lead and cadmium (Irwin et al., 1997).

In water, zinc settles onto the sediment and a small amount remains dissolved in the water or is present as suspended particles (Irwin et al., 1997). Zinc is usually more concentrated in the sediments of streams and rivers compared to the water column. In non-polluted areas, zinc concentrations can be as low as 0.1 µg/L. In rivers, the concentration of zinc is higher (20 µg/L) whereas, in streams affected by mine drainage, the zinc concentration can exceed 100 µg/L (Irwin et al., 1997). In aquatic ecosystems, the concentration of zinc is not only influenced by sediment type but also by other abiotic parameters such water hardness (Irwin et al., 1997).

Zinc is an essential metal for most aquatic organisms, due to its involvement in somatic cell functions including protein synthesis and enzymatic regulation (Bury et al., 2003). For example, in teleost fish, zinc is taken up through transporters on the apical membrane of chloride cells in the gill epithelium. However, under pollution, excess of zinc may compete with Ca²⁺ at the apical membrane of chloride cells via nonspecific Ca²⁺ channels,





which may, in turn, disrupt Ca^{2+} homeostasis (Bury et al., 2003). Another example of the acute effect of the zinc was documented in *Branchiura sowerbyi* and *Tubifex tubifex* where this two organism have shown to be more sensitive to zinc compare to the arsenic (Lobo et al., 2016). In nematode, zinc has shown to decreases the growth of the *Caenorhabditis elegans* population in a 500 mM of zinc solution (Dietrich et al., 2016).

1.6.5 Heavy metal pollution and host-parasite interactions

In aquatic environments, organisms are not only confronted with pollutants, but often also with parasites. As environmental pollutants may affect the physiological homeostasis of organisms that act as hosts, it can be expected that host-parasite interactions will also be altered (Sures, 2008). The effects of pollutants on parasitism are variable and may be positive or negative. For example, pollution may increase levels of parasitism by interfering with host defence mechanisms (Sures, 2004). Parasites may also be more resistant than their hosts to pollutants and thus tend to increase in number in polluted condition (Mackenzie, 1999). On the other hand, pollution could also decrease parasitism, if parasitized animals suffer increased mortality under polluted conditions, or if parasites are more susceptible to the pollutant than their hosts.



The complexity of the parasite life cycle may play an important role in determining the patterns of infection that emerge in polluted environments. Levels of infection with endoparasitic helminths with complex, indirect life cycles tend to decrease, while infections with ectoparasites with direct single-host life cycles tend to increase (Mackenzie, 1999). One explanation is that endoparasites with indirect life cycles can be affected directly (as free-living transmission stages or adult forms in the alimentary tract come into direct contact with the pollutant) and indirectly, through adverse effects of the pollutant on other hosts in the parasite life cycle (Mackenzie, 1999). Ectoparasites with direct life cycles, on the other hand, have a constant direct contact with the external environment and in the course of their evolution, they have developed flexibility and resistance to certain natural changes (Mackenzie, 1999). Consequently, many ectoparasites show higher tolerance of certain types of environmental change than their hosts (Mackenzie, 1999). However, there is variation in parasite responses toward pollution depending on the parasite taxa and the pollutants involved (Mackenzie, 1999, Sures, 2008). For example, nematode parasites show an increase in abundance among hosts exposed to crude oil, whereas digenean parasite abundance in crude oil-affected host populations was decreased (Lafferty, 1997).





Pollution also influences parasitism by affecting the swimming behaviour and longevity of free-living infective parasite stages. For example, in *Cryptocotyle lingua*, acute exposure to Iron, zinc, and copper in artificial seawater can affect both the swimming rate and the longevity of cercariae released from snail hosts. The swimming behaviour and longevity of cercariae are relevant because both can influence the probability of transmission to the second intermediate hosts (Rea and Irwin, 1992).

Pollution could influence susceptibility to parasite infections by impairing host immune responses (Khan, 1990). Heavy metal pollutants appear to have a consistently negative effect on parasite numbers, but the magnitude of the effects can be dependent on concentration (Lafferty, 1997). For example, guppies (*Poecilia reticulata*) exposed to intermediate levels of zinc ($15 - 60 \mu\text{g/L}^{-1}$) were less likely to develop ectoparasite *Gyrodactylus turnbulli* infections (Gheorgiu et al., 2006) than when exposed to a concentration of $240 \mu\text{g/L}^{-1}$. This is due to the proliferation of mucus on the fish epidermis as a result of high zinc exposure, which made it difficult for the parasite to attach.

Parasites can also modify the effect that heavy metal pollutants have on their hosts. For example, sticklebacks infected with *S. solidus* were affected more severely when exposed to cadmium than non-infected fish (Pascoe and Cram, 1977). However, some endoparasites, such as acanthocephalans, appear to be capable of accumulating heavy metals (Sures, 2008), and hence reducing their toxic effects on hosts. For example, the acanthocephalan *Pomphorhynchus laevis* accumulates heavy metals (Lead) in much higher concentrations than those found in the organs of its fish host, chub (*Leuciscus cephalus*) (Sures and Siddall, 1999).

At a population level, while the effect of anthropogenic stressors such as heavy metal pollutants may increase the susceptibility of individuals to parasites, if this stress also leads to a reduction in host density (through mortality of parasitized individuals) then disease levels may fall (Lafferty and Holt, 2003).

