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How parasitism and pollution affect the physiological homeostasis of aquatic hosts

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Abstract

Parasitism poses a serious threat to hosts under certain circumstances, while the well-being of organisms is also negatively affected by environmental pollution. Little information is available on the simultaneous effects of parasites and pollutants on the physiological homeostasis of organisms. The present paper demonstrates that parasites: (i) may influence the metabolism of pollutants in infected hosts, (ii) interact with pollution in synergistic or antagonistic ways, and (iii) may induce physiological reactions in hosts which were thought to be pollutant-induced. Experimental studies on the uptake and accumulation of metals by fish reveal that fish infected with acanthocephalans have lower metal levels than uninfected hosts; e.g. *Pomphorhynchus laevis* reduces lead levels in fish bile, thereby diminishing or impeding the hepatic intestinal cycling of lead, which may reduce the quantity of metals available for fish. Alterations in pollutant uptake and accumulation in different intermediate and final hosts due to parasites are thus very important in the field of ecotoxicology. In addition to such alterations, there is a close interaction between the effects of pollutants and parasites which seems to be mediated at least partly by the endocrine system, which itself is closely related to the immune system in fish. Laboratory studies on eels experimentally infected with the swimbladder nematode *Anguillicola crassus* reveal that toxic chemicals such as polychlorinated biphenyls produce immunosuppressive effects which facilitate parasite infection. Similarly, an increase in serum cortisol concentration in eels due to chemical exposure and infection is correlated with decreasing levels of anti-*A. crassus* antibodies. Furthermore, parasites are able to elicit physiological changes which are attributed to chemicals with endocrine disrupting activity, e.g. the cestode *Ligula intestinalis* is known to suppress gonad development in roach. The most thoroughly documented examples of endocrine disruption in wild fish are in roach, and it is conceivable that this disruption is not only due to chemical activity but also to parasites such as *L. intestinalis* or species of the phylum Microspora.

Introduction

Pollution of aquatic environments is an important aspect of globalization. In addition to their presence in the water column, many substances accumulate in the sediment of aquatic biotopes where they can be dissolved again and enter the food chain. The uptake and accumulation of pollutants by organisms may cause a certain degree of harm, depending on the specific substance. The effects of pollution on parasitism are variable and may be positive or negative. On the one hand, pollution may increase parasitism, while on the other it may be fatal for certain parasite species leading to

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a decrease in parasitism (Lafferty, 1997; Overstreet, 1997; Pietrock & Marcogliese, 2003; Sures, 2004, 2005). An increase in parasitism is likely if the pollutant mainly affects the host rather than the parasite. For example, immunotoxic chemicals may suppress the immune response of a host, thereby enabling parasites to establish. In contrast, pollution can also negatively affect parasitism, e.g. if parasites are more susceptible to the particular pollutant than their hosts. Additionally, pollution may also drive necessary intermediate and final hosts to extinction, or infected hosts may suffer more from environmental exposure than uninfected conspecifics.

Recent studies have revealed that the interaction between pollution and parasitism is much more complex than only the direct effects of pollution on parasites. For example, aquatic parasites, mainly acanthocephalans, are known to reduce pollutant levels within their hosts. Therefore, parasites might even have a positive influence on their hosts if the latter face environmental pollution. Additionally, in some cases parasites and pollution act together, resulting in synergistic effects that might be more harmful to the host than single effects of parasites or pollution. The third example deals with effect of parasites on the gonad development of their hosts. Feminization as well as suppression of gonad development and maturation are phenomena which are currently discussed mainly from the ecotoxicological point of view (Kime, 1998; Vos *et al.*, 2000). Parasitism is largely ignored within this discussion but might have a significant impact (Jobling & Tyler, 2003; Hecker & Karbe, 2005; Schabuss *et al.*, 2005).

Parasites reduce the metal burden of their hosts

There are several reports on the uptake and accumulation of metals by parasites of fish, especially acanthocephalans (Sures, 2003, 2004, 2005). The most conspicuous metal accumulation described so far was found in *Pomphorhynchus laevis* parasitizing chub (Sures *et al.*, 1994; Sures & Taraschewski, 1995). Due to the unexpectedly high metal uptake by this parasite, experimental studies were initiated to investigate the metal uptake kinetics of *P. laevis* in more detail (Sures & Siddall, 1999, 2003; Sures *et al.*, 2003). In addition to the evaluation of the uptake kinetics by fish tissues and the parasite, a comparison between metal concentrations in tissues of infected versus uninfected fish showed lower metal levels in infected fish (fig. 1). Accordingly, the presence of *P. laevis* in the intestine reduces the metal level in the fish host. Sures & Siddall (1999) developed a model to explain metal uptake by *P. laevis* and the associated reduction of lead in host tissues. A central element of this hypothesis is that the acanthocephalans take up bile-bound lead in the small intestine. The liver expels lead ions by binding them to steroids in the bile. These bile–lead complexes then pass down the bile duct into the intestine. In uninfected fish, bile-bound lead in the intestine can be reabsorbed by the intestinal wall and runs through the hepatic intestinal

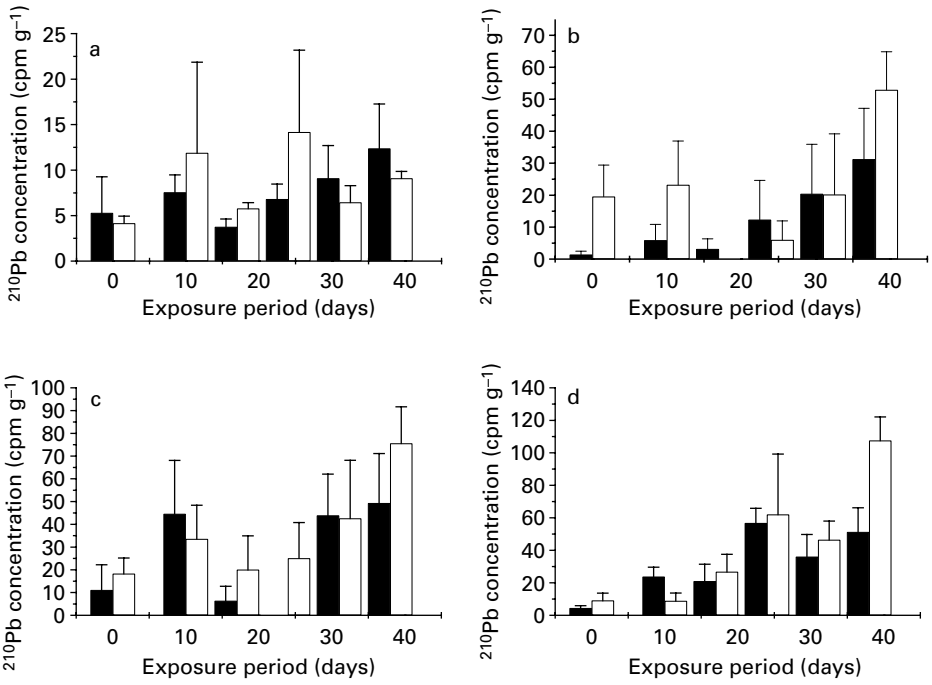

Fig. 1. Mean (+S.E.) concentrations of ^{210}Pb in different tissues of infected (■) and uninfected (□) *Leuciscus cephalus* following experimental infection of fish with larvae of *Pomphorhynchus laevis* and subsequent exposure to ^{210}Pb in the water. While no difference exists for muscle (a), infected chub show, especially at the end of the exposure period, reduced metal levels compared with uninfected conspecifics in liver (b), bile (c) and intestine (d) (for details see Sures *et al.*, 2003).

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cycle. This cycle is interrupted by the parasites, which take up bile bound lead and thus reduce the available amount of lead that could be reabsorbed by the fish host. As a consequence, infection not only reduces the amount of lead which is reabsorbed by the intestinal wall and runs through the hepatic-intestinal cycle, but also the amount which is available for accumulation in the fish organs. This could explain why metal levels in the various organs of infected fish ranged below the respective levels in uninfected chub (fig. 1). This enormous uptake is, however, not only described for lead but also for a number of other elements, including cations of physiological importance (e.g. Sures *et al.*, 1999; Sures & Reimann, 2003). Statistical analysis has revealed that parasites also affect mineral concentrations in the inner organs of fish, as the levels of several essential elements in the liver of perch *Perca fluviatilis* were negatively correlated with the size of the infrapopulation of *Acanthocephalus lucii* (Sures, 2002; see fig. 2). In addition to the physiological importance of minerals, it appears likely that acanthocephalans take up such elements as a by-product when taking up bile acids in the intestine of their hosts. It is known that acanthocephalans are extremely efficient in taking up bile salts (Kennedy *et al.*, 1978; Nickol, 1985; Starling, 1985) as most intestinal parasites are unable to synthesize their own steroids and fatty acids (Barrett *et al.*, 1970). Therefore, helminths rely on the uptake of substances which have a basic sterol structure from the host's intestinal lumen. With respect to toxic metals, however, the uptake of metals by acanthocephalans and the related reduction of metal levels in host tissues might be advantageous for the infected host. Due to the enormous uptake capacity of parasites for metals, it is conceivable that pollutant levels in the host are reduced below toxic threshold levels.

Interaction between parasites and pollution

A common strategy against infection is the production of specific antibodies as part of an acquired immune response. However, a number of chemicals are released into the environment, which are known to have immunomodulatory effects, for example polychlorinated biphenyls (PCBs) (Thuvander, 1989; Arkoosh *et al.*, 1998a,b; Zelikoff, 1998). The immunotoxicity of PCBs correlates with the binding affinity to the cytosolic aryl hydrocarbon receptor (Ahr) (Kafafi *et al.*, 1993), which is a well-described transcription factor for a variety of gene products, including cytochrome P 450 1A (Hahn & Stegemann, 1994). PCBs were manufactured for industrial purposes, such as dielectric fluids in transformers and as paint additives, and are still present in the environment due to their high persistence (Safe, 1994). In a recent study, Sures & Knopf (2004a) demonstrated that European eels experimentally infected with third stage larvae (L3) of the swimbladder nematode *Anguillicola crassus* are able to produce specific antibodies. This ability was prevented if eels were simultaneously exposed to PCB 126 (see fig. 3). Similarly, Regala *et al.* (2001) also found suppression of the antibody response to *Vibrio anguillarum* in channel catfish following intraperitoneal injection of *Ictalurus punctatus* with PCB-126. Immunosuppression by environmental pollutants might, therefore, be an important factor in the infection success of parasites in general. In the case of *A. crassus* infecting European eels, PCBs are unlikely to be involved in the infection success as the protective role of the *Anguillicola*-specific antibodies is doubtful (Knopf *et al.*, 2000). However, as studies on the immune response to helminths are largely lacking, the paper by Sures & Knopf (2004a) stands out as an example of the immunological response to infection with helminths of a host facing environmental pollution with immunosuppressive chemicals.

Because immune and endocrine systems are closely related in fish (Weyts *et al.*, 1999), stress and immune responses should be taken into account simultaneously. Accordingly, the stress response of eels experimentally infected with *A. crassus* was also analysed (Sures *et al.*, 2001, 2006). In these studies, the infection alone causes a significant increase in serum cortisol levels in eels (Sures *et al.*, 2001; fig. 4). Even if infection is combined with different forms of pollution (PCB 126, Cd), the presence of *A. crassus* appears to be the overlying stress factor. As it is known that increased plasma cortisol levels in fish are often associated with immunosuppression (e.g. Wendelaar Bonga, 1997; Weyts *et al.*, 1999), a correlation analysis between levels of cortisol and specific antibodies was performed. This revealed that high

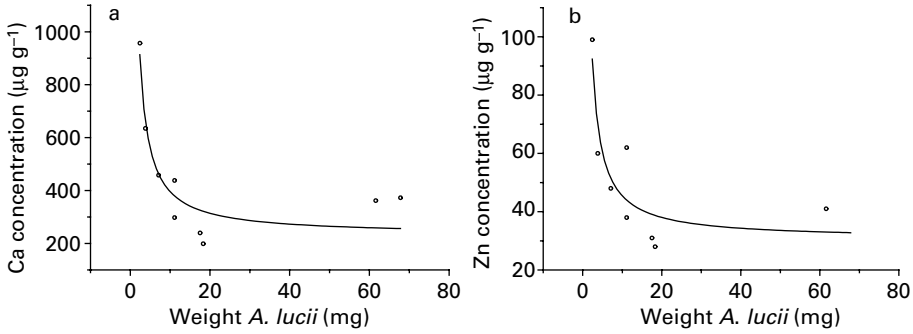

Fig. 2. Regression analysis showing negative hyperbolic associations between element concentrations in the liver of perch and the weight of the infrapopulation of *Acanthocephalus lucii*, sampled from naturally infected perch (details in Sures, 2002).

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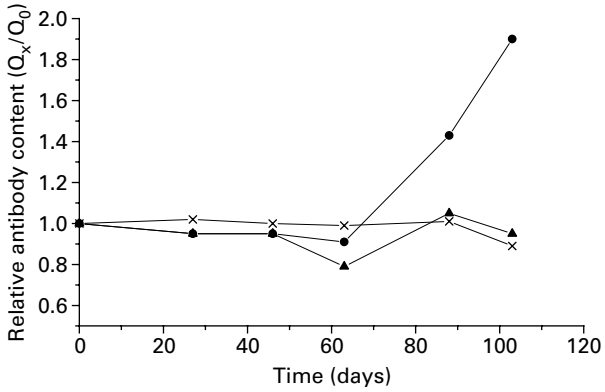

Fig. 3. Time course of the antibody response of eels inoculated with ten third stage larvae of *Anguillicola crassus* on day 27 as measured by ELISA using a crude antigen from the body wall of adult *A. crassus*. Uninfected controls (x) show no increase in antibody concentration whereas a distinct increase in antibody level occurs in infected eels (●). Simultaneous exposure of infected eels to PCB 126 (▲) completely suppresses the immune response of eels (details in Sures & Knopf, 2004a).

plasma cortisol levels in eels were significantly associated (Spearman correlation analysis $P \leq 0.05$) with low antibody levels using a data set obtained from 70 experimentally infected and chemical exposed eels. It appears likely that parasites evoke a clear stress response, i.e. an increase in cortisol concentrations, which then suppress the host's humoral immune response. This could form part of an extremely effective colonizing strategy by *A. crassus* (Sures & Knopf, 2004b) to induce stress in its newly adopted final host (the European eel), suggesting that successful infection could then be aided by the resulting immunosuppression. However, this hypothesis deserves further experimental studies.

Parasites induce endocrine disruption in fish

Feminization, particularly in aquatic vertebrates, raises a number of interesting questions and related research

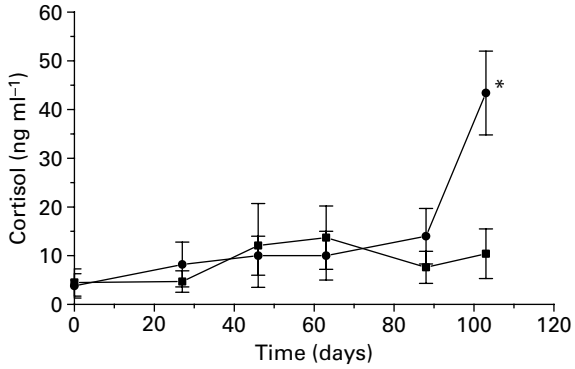

Fig. 4. Time course of mean serum cortisol concentrations in uninfected control eels (■) as compared with eels which were inoculated with ten third stage larvae of *Anguillicola crassus* (●) on day 27 (details in Sures *et al.*, 2006). *Indicates a significant difference (Mann-Whitney *U*-test, $P \leq 0.05$)

(e.g. Wiklund *et al.*, 1996; Jobling & Taylor, 2003; Rodgers-Gray *et al.*, 2004; Hecker & Karbe, 2005; Schabuss *et al.*, 2005). From an ecotoxicological point of view, the main causative agent for endocrine disruption, e.g. in fish, is the presence of oestrogen-like chemicals in the environment (Kime, 1998; Vos *et al.*, 2000; Jobling & Tyler, 2003). Accordingly, there is a fast growing body of literature on the effects of oestrogen-like chemicals on gonad development in amphibians and fish from laboratory investigations. However, several field studies show that endocrine disruption also occurs in fish which are not exposed to any chemicals (Wiklund *et al.*, 1996; Schabuss *et al.*, 2005). Thus, environmental pollution might not be the only reason for feminization or other gonad impairment. There is strong indication from different groups of animals that endocrine disruption is also a result of infection with parasites. For example, species of the phylum Microspora, such as *Nosema granulosis*, are known to cause feminization in crustaceans (Rodgers-Gray *et al.*, 2004). Infection of *Gammarus duebeni* with *N. granulosis* causes either phenotypic females or intersexes with fully developed ovaries and an undifferentiated androgenic gland. Similarly, fish such as roach *Rutilus rutilus* are also commonly infected with microsporidians of the genus *Pleistophora*. In a field study along the Finnish coast, an increasing prevalence of infection in female and male roach with *P. mirandellae* was positively associated with the occurrence of hermaphroditism (Wiklund *et al.*, 1996). These authors described an infection of oocytes with this microsporidian, therefore feminization of the host could favour the establishment of *P. mirandellae*. Not only protozoans but also metazoans, and among them plerocercoids of the cestode *Ligula intestinalis*, might be capable of endocrine disruption in their fish hosts (Schabuss *et al.*, 2005). *Ligula intestinalis* has a significant negative effect on the gonadosomatic index, plasma vitellogenin level, CYP19 aromatase activity, and plasma concentrations of the sex steroid hormones 17β-estradiol, 11-keto-testosterone and testosterone in *Abramis brama* (Hecker & Karbe, 2005). The underlying molecular and biochemical processes causing gonad impairment remain unclear, although Arme (1997) has shown that the secretion of gonadotrophins or gonadotrophin-releasing hormones is suppressed by plerocercoids of *L. intestinalis* which will result in reduced levels of sex steroids produced by the gonads. It is, however, conceivable that *L. intestinalis* takes up the sex steroids and uses them for its own growth. Only experimental studies will help to understand the physiological and molecular interaction of this cestode and its intermediate host.

Is there a common strategy among the examples presented?

At a first glance, the three examples presented here are isolated results without a common feature. They deal with systematically different groups of parasites (acanthocephalans, nematodes, cestodes), different forms of pollution (metals, PCBs, oestrogen-like chemicals) and different effects on the hosts (uptake of bile-bound metals, induction of cortisol, reduction of sex

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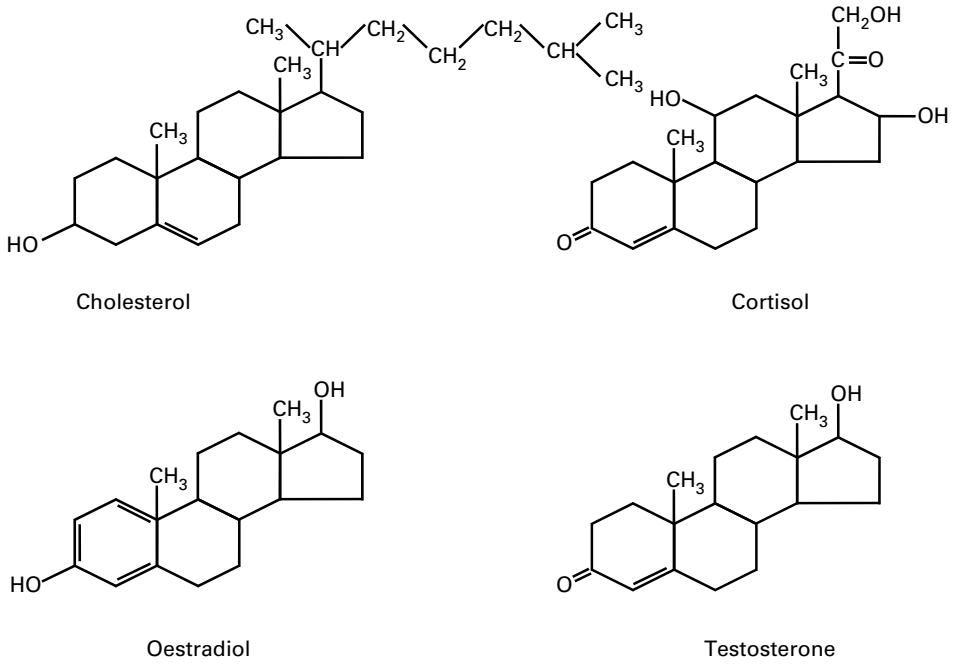

Fig. 5. Chemical structure of different steroids.

steroids). Nevertheless, the effects on the hosts, in particular, show a common strategy for all of these parasite groups. If we look at the general chemical structure of bile acids (= cholesterol), cortisol and sex steroids (see fig. 5), it is clear that these molecules are all steroids. In the case of acanthocephalans, it is apparent that worms take up these substances whereas in *L. intestinalis*, the plerocercoids are likely to take up sex steroids. It is, however, uncertain why *A. crassus* induces a cortisol response in infected hosts. This stress induction is probably an important part of the infection process, but it is also conceivable that *A. crassus* takes up cortisol. Due to its location in the swimbladder, a microhabitat where the parasite has only limited access to nutrients, absorption of cortisol from the blood could be an effective way of taking up steroids. As previously mentioned, helminth parasites lack basic pathways for sterol synthesis, and hence rely on the uptake of these chemicals from the host. In all these cases, it appears unlikely that helminths utilize the original effect the substance has on its vertebrate host, rather than just using the basic steroid structure, which can then be altered to fulfil the function induced by the helminth. Sterols such as cholesterol have multiple functions. On the one hand they are membrane constituents that regulate membrane fluidity and permeability by condensing the membrane bilayer (Demel & De Kruyff, 1976; Bloch, 1983) and are thus essential for cell growth and maintenance. On the other hand, sterols are also involved in a variety of developmental processes as well as in signalling pathways (Benveniste, 2002). The dilemma that helminths (as well as other invertebrates) face is a lack of steroid biosynthesis pathways although they need sterols for their own growth and physiological homeostasis. This sterol deficit might be balanced by an increased absorption of any kind of steroid produced by the host. This hypothesis can explain how parasites such *A. crassus* and *L. intestinalis* can grow so quickly without producing their own sterols. Due to their location outside of the intestine, both *A. crassus* and *L. intestinalis* have developed alternative ways of taking up steroids wherever they occur in the body of the host. Even if this hypothesis appears likely, it should be elucidated in more detail in future experimental studies.

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