Histological patterns of the intestinal attachment of *Corynosoma australe* (Acanthocephala: Polymorphidae) in *Arctocephalus australis* (Mammalia: Pinnipedia)

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Received: 6 June 2012 / Accepted: 18 January 2013 / Published online: 27 February 2013
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Abstract The mucosal attachment pattern of *Corynosoma australe* in the intestines of *Arctocephalus australis* is described. Normal and abnormal tissue were sampled from 32 hosts to be submitted to histological routine protocol to embedding in paraffin and permanent mounting in balsam. *Corynosoma australe* shows three different degrees of body depth intestinal attachment (BDINA-1–3). BDINA-1: it is exclusive of the small intestine and the parasite attaches on the villi; BDINA-2: parasite affects the Lieberkühn crypts in several depth levels and, BDINA-3: the parasite reaches the submucosa. These attachment patterns alter the mucosa by degeneration and dysfunction due to necrosis of mucosal structure, great quantities of cellular debris and significant reduction of the mucosal thickness. Other aspects are crater-like concave holes (CLCHs) as sites where *C. australe* could be attached–detached several times according to adult migratory processes within luminal intestine space. The submucosa shows edema probably due to the local mucosal alterations resulting in homeostatic break. There is no severe inflammatory response by host but BDINA-1 to BDINA-3 and CLCH could represent foci to secondary opportunistic infections and significant areas of malabsorption in severely infected hosts contributing to increase clinical signs of preexistent pathologies.

Keywords Intestinal histopathology · Parasite attachment pattern · Corynosomiasis · Mammalian host · Arctocephalinae

Introduction

The host–parasite relationship is complex, occurring at different trophic levels and may cause tissue changes as observed in *Pontoporia blainvillei* (Silva and Cousin 2004). Otariidae mammals, as South American fur seal *Arctocephalus australis* (Zimmermann 1783), comprise definitive hosts to Acanthocephala to finish their life cycles adequately (Bush et al. 2001; Raga et al. 2002). Despite some studies on the biology of *A. australis* as well as other aquatic mammals in South America, there is a lack or little information to helminthiasis and to helminthiasis causing histopathologies (Silva and Cousin 2004). There are a greater number of histopathological and parasitic pathogenicity studies to Pinnipedia species in the North Hemisphere (Ridgway 1972; Lauckner 1985). Maybe this can be the result from an alternative data utilization of marine mammals stocks for researches during historical hunting or commercial fishery bycatch (Crespo and Hall 2001; Jennings et al. 2001).

Parasitosis is recognized as a significant factor in massive strandings and mortality of Pinnipedia (Ponce de León 2000; McKenzie et al. 2005) but Acanthocephala is considered a group of low negative impact in Pinnipedia (Ridgway 1972; Vlasman and Campbell 2003). However attachment sites of Acanthocephala can represent locals for secondary opportunistic infections and the effect of parasites in their hosts should be considered, at least, as a despoiling or plundering (Bush et al. 2001). *Corynosoma australe* Johnston 1937 (Acanthocephala, Polymorphidae)

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is commonly found in *A. australis* but histopathological aspects caused by this parasite on mammalian host is absent or scarcely contemplated in the literature.

The mucosal attachment pattern of *C. australe* in the intestines of *A. australis* is described.

Materials and methods

For this study the carcasses (15 males:15 females) of *A. australis* were collected from Cassino Beach (ca. 32°11'14.23"S; 52°09'21.70"W) to Chui (ca. 33°44'35.96"S; 53°22'12.70"W)—Rio Grande do Sul State—Brazil. The total length (TL) of the hosts was linearly measured in meters (m) (*sensu* Dierauf 1994) and the sex was determined by external examination (Pinedo et al. 1992). Only carcasses respecting the conservation status code 2 and 3 from Dierauf (1994) were utilized for tissue and parasite's samplings.

Normal and abnormal (attached *C. australe*) tissues samplings from small and large intestines were extracted for the histological routine protocol for embedding in paraffin and permanent mounting in balsam. Tissues samples were fixed (Bouin's fluid). Paraffin blocks were sliced (microtomy) under 7 µm in thickness and the histological slides were stained with Hematoxylin–Eosin and Alcian-Blue *sensu* Silva and Cousin (2006a, b). Under stereomicroscopy, portions of the intestine were analyzed to detect and characterize attachment patterns of *C. australe*. The collection and preparation of parasites for permanent mounting in balsam and staining (Semichon's Carmim and Eosin) followed and adapted from Amato et al. (1991) and Silva and Cousin (2006a, b). Parasites were identified *sensu* Petrochenko (1971), Zdzitowiecki (1984), Pereira and Neves (1993) and Sardella et al. (2005) holdfast criteria: proboscis oncotaxia, body shape, trunk (presoma and metasoma) spine (structure and distribution pattern) and organology.

Histometric statistical comparisons (Variance Analysis; Tukey's test; $p < 0.05$), according to the thickness, was done only between normal and abnormal layer affected (=mucosa) by *C. australe* per each intestine anatomical division (small and large intestine). The histometries were performed with a metric eyepiece lens calibrated with a micrometric slice (Nikken–Tokio; 0.01 mm) according to the different magnitudes (4×10 , 10×10 , 20×10), following Silva and Cousin (2004, 2006a, b). The gross and microscopic aspects of the lesions were utilized to typify the severity of the injury (*sensu* Thomson 1983).

Results

In the sampling the hosts' TL range were: $0.84 \geq TL \leq 1.76$ m to males and $0.8 \geq TL \leq 1.34$ m to females. Male

and female hosts infected by *C. australe* comprised $0.95 \geq TL \leq 1.76$ m and $0.87 \geq TL \leq 1.34$ m, respectively.

Macroscopically, the attachment of *C. australe* in the intestines showed three different degrees of body depth intestinal attachment (BDINA): 1, outer or superficial (proboscis attached in the intestines intervilli spaces); 2, medial (presoma intestines-embedding) and 3, inner or deepest (metasoma partially intestines-embedding). Mainly in cases two and three delicate borderline elevations or demarcations in the attachment site with a deep central hole, housing the proboscis, were noted. There were no color intestine changes around the *C. australe*-attachment site. Other lesional features associated with *C. australe* are CLCH as sites where *C. australe* could be detached.

Microscopic aspects of corynosomiasis are presented below.

BDINA-1

Proboscis attachment occurs on intervilli spaces not invading the Lieberkühn crypts. There are villi folding, broking and erosion. The villi can be absent. The Lieberkühn crypts show necrotic features and architectural disorganization. The submucosa is edematous and shows dilated lymphatic vessels added with the non-modeled dense connective tissue with collapsed collagen fibers. The large intestine has no villi, thus BDINA-1 pattern is absent (Fig. 1a).

BDINA-2

Parasite can reach the Lieberkühn crypts causing mucosal damage in several depths proceeding toward, probably, BDINA-3 or CLCH patterns. Beyond the hooks of the proboscis, the mucosal damage is exacerbated by the action of the spines of the presoma. There is total erosive destruction of villi and different alterations of the Lieberkühn crypts according to the depth of the parasite on the mucosa. Thus, great quantities of cells debris (mainly pyknosis from cylindrical epithelium and loose connective tissue) occur near the attachment site and around the parasite. The muscularis mucosa is not eroded. Villi on periphery of lesion are strongly pushed laterally and this confers to the lesion a parasite's body mold-like aspect. The submucosa is edematous as in BDINA-1, shows low lymphocyte infiltration and foreign-body-like giant cells were not present. There are hypertrophy of the Lieberkühn crypts due to folding and/or erosion of the villi. The Lieberkühn crypts are filled with remnants of mucous and enterocytes remains (Figs. 1b, 2a, b).

Fig. 1 Microphotographs of body depth intestinal attachment (BDINA) pattern of *Corynosoma australe* (CORAUS) within small intestine of *Arctocephalus australis*. MU mucosa, SB submucosa, ML muscular layer; SE serosa. **a** BDINA-1. Note the villi (VIL) attachment of the armed (arrows) proboscis (PRO) and the edema of the submucosa. Stain H–E. Scale bar 270 μ m. **b** BDINA-2 in initial phase. Observe the absence of villi in the attachment site and the cellular remains. Parasite is reaching the Lieberkühn crypts (arrows). Stain H–E. Scale bar 220 μ m. **c** BDINA-3. Parasite destroys completely the mucosa and reaches submucosa. Stain H–E. Scale bar 140 μ m

BDINA-3

It is a deeper lesion feature caused by parasite's holdfast. There is total mucosal destruction beyond the muscularis mucosae. *C. australe* reaches the submucosa, sometimes causing a slight concave depression on it. Cell and tissue alterations observed are similar that found in BDINA-1 and BDINA-2 (Figs. 1c, 2c).

CLCH

Similar to BDINA-3 but without the parasite, resembling sites where *C. australe* detached. The submucosa is exposed directly to intestine lumen. In some cases, occurs

incomplete re-epithelization of the affected area of the mucosa (Fig. 2d).

In BDINA-2 and BDINA-3, the spines of trunk (presoma and metasoma) of *C. australe* are strongly attached on the intestine tissues as a parasitic proboscis auxiliary mechanism to mucosal embedding (Fig. 2e). The parasite can folds the area between proboscis and presoma utilizing neck retractors muscle bundles what increases the body attachment (surface contact) area. The attachment pattern observed leads to a significant reduction of the thickness of the mucosa of the small as well as of the large intestine, where the parasite was attached (Table 1). There were no *C. australe*-related inflammatory processes (Figs. 1a, c, 2a, e).

Fig. 2 Microphotographs of body depth intestinal attachment (BDINA) and crater-like concave holes (CLCHs) pattern of *Corynosoma australe* (CORAUS) within large intestine of *Arctocephalus australis*. MU mucosa; SB submucosa; ML muscular layer; SE serosa. **a–b**: BDINA-2. In **a** note the direct contact of the parasite with the intestine wall, the cellular debris between parasite and intestine and in **b** the proboscis (PRO) attachment in the Lieberkühn crypts and the edema of the submucosa. Stain H–E. Scale bar 520 μ m. **c** BDINA-3. Mucosa is completely destroyed and parasite reaches the submucosa. Stain H–E. Scale bar 260 μ m. **d** CLCH. Observe the absence of parasite within mucosal lesion, the mucosal removal and the re-epithelization process. Stain H–E. Scale bar 260 μ m. **e** Parasite trunk as a secondary holdfast. Note the trunk spines (arrows) anchoring the intestine mucosal tissue. Stain H–E. Scale bar 130 μ m

Discussion

Corynosomiasis, in this study, did not show inflammation as in *Pontoporia blainvillei* (Cetacea, Pontoporiidae) stomachs by *C. (=Polymorphus) cetaceum* infection (Silva and Cousin 2006b) but differed from intestinal *Bolbosoma*-associated colonic granuloma in this host (Silva and Cousin 2006a). Nevertheless Acanthocephalosis are recorded in mammalian

hosts causing *Profilicolis*-associated (Profilicolidae) septic peritonitis by migration in *Enhydra lutris* (Kreuder et al. 2003) and *Macracanthorhynchus*-associated (Macracanthorhynchidae) neoplasia, chronic enteritis, intestinal perforation and intestinal eosinophilic granuloma in *Hemiechinus auritus* (Taman 2009), thus contributing to mortalities. In *Halichoerus grypus* Fabricius 1791 (Pinnipedia, Phocidae) *Corynosoma*-associated intestinal “ulcerations” are recorded (Bergman et al. 2003).

Several ecologic pressures influenced many aspects of the functional morphology of the Acanthocephala species and selected these parasites to an effective attachment mechanism (Petrochenko 1971), but there are scarce studies investigating the relationship between Acanthocephala morphology and attachment performance (Petrochenko 1971; Taraschewski et al. 1989; Taraschewski 1990; Aznar et al. 1999). The histopathological pattern from BDINA-1 to CLCH may represents a parasite attachment progressive mechanism (APM). APM could begins with BDINA-1 (as an initial attachment phase to newly-recruited, newly-adulthood and adult parasites) towards CLCH (to senescent-died or newly-detached parasites), according to *C. australe* biological needs. APM is supported by Acanthocephala’s migration capacity within intestinal lumen due to reproductive processes (maturation, copulation and oviposition) while they move mainly jejunum-to-ileum-ward (George-Nascimento and Marin 1992; Aznar et al. 2004). Parasite’s migratory processes can also be influenced by circadian and seasonal cycles such as availability of the food, host’s food intake rigor, food bulk intestinal flow, nutritional composition of the food as well as intra-inter-specific relationships as crowding effect (Mettrick and Podesta 1974; Nickol 1985). Males of Acanthocephala are more active in copulation than females; they move seeking for females to mate with as many as possible (Parshad and Crompton 1981). On the other hand, within taxa such as Acanthocephala the infrapopulation sex ratios are commonly female-biased (Poulin 1997; Aznar et al. 2001, 2004) and females *Corynosoma* spp. have longer life-span than males (Aznar et al. 2001) and they migrate intestine posteriad, according to ova maturation (Aznar et al. 2004). Thus, sexual selection can influence Acanthocephala’s spatial distribution (Sini-salo et al. 2004) and attach-detach rates. Male-to-female attachment’s ratio was not studied here, but there are evidences that *C. australe* could attach-detach several times lifelong at different intestinal length levels (Parshad and Crompton 1981; George-Nascimento and Marin 1992; Aznar et al. 2004), conducting to several CLCH by each specimen within host’s intestine.

In Corynosomiasis, the parasite’s gradual and chronic mucosal embedding can be explained by the use of the trunk as a secondary holdfast due to the anatomy of the powerful muscles that allow precise movements forward and

Table 1 Variance Analysis (Tukey's test; $p < 0.05$) for histometries (thickness) of the normal and abnormal mucosa (μm) to small and large intestines of *Arctocephalus australis* parasitized by *Corynosoma australe*

Intestine layer	Intestine portion			
	Small intestine		Large intestine	
	Normal dimension (μm)	Abnormal dimension (μm)	Normal dimension (μm)	Abnormal dimension (μm)
Mucosa	1116.75 $\pm$ 380.82 ^a	313.27 $\pm$ 239.69 ^b	880.81 $\pm$ 40.62 ^a	280.04 $\pm$ 257.72 ^b

Different letters mean significant differences between normal and abnormal mucosa according to each intestine. Variance Analysis (ab) $p < 0.05$

backward (Hayunga 1991; Aznar et al. 1999). The morphology of some Acanthocephala groups permits deep penetration within the digestive tube wall, what causes extensive tissue damage (Dezfuli et al. 2002). An example of this mechanism can be observed concerning to *Bolbosoma* spp. (Polymorphidae, Acanthocephala) that embed its trunk disc deeply within intestinal mucosa of mammalian definitive hosts (Petrochenko 1971; Measures 1992; Silva and Cousin 2006a). The depth penetration of this parasite group and the density of helminths comprise the two principle factors responsible by Acanthocephala pathogenicity as records Taraschewski (2000). *Pomphorhynchus laevis* (Pomphorhynchidae) infecting the European chub *Leuciscus cephalus* (= *Squalius cephalus*) (Cyprinidae), shows four gradual attachment–embedding patterns involving different parasite's body parts (proboscis, neck, trunk) in different depths within the intestine gut wall (mucosa, submucosa, muscular layer and serosa) (Dezfuli et al. 2002). The involvement of different parasite's body parts for attach in a surface of the digestive tube is similar to that recorded in this survey. *Pomphorhynchus laevis* shows a rivet-like mode of attachment that trespass the intestine wall and sometimes reaches the liver and pancreas (Dezfuli et al. 2002), whereas *C. australe* shows a body mold-like aspect does not trespassing the totally the intestine wall (*i.e.*, the attachment and damage is restrict to the mucosa).

Although severe inflammatory responses were not observed, the new areas of attachment (reattached specimens) and CLCH of *C. australe* can also represent foci of secondary infection and dysfunctional areas (Thomson 1983; Carlton and McGavin 1998). Concerning to the dysfunctionality, the abnormal mean area of attachment of 1.5 mm² by each *C. australe* (Aznar et al. 2004), addicted with edema of the submucosa and luminal exposition, could be considered as an important factor to nutrients malabsorption in hard infected hosts. It is important to maintain the isotonicity of the luminal contents because hypertonic solutions within the lumen cause structural and functional damage on the mucosa (Mettrick and Podesta 1974; Pácha 2000). Intestinal epithelium promotes strong exchange of water and electrolytes with body's extracellular fluids and disturbances in this orderly intestinal secretion–reabsorption function can quickly promote severe body fluid depletion

(Mettrick and Podesta 1974). The alterations in enterocytes can promotes disturbances in the boundary between apical and basolateral membrane (=tight junctions) that prevents free movements of water and solutes through the lateral intercellular spaces, affecting water channels of the aquaporin family recently identified in enterocytes and salts channels absorption (sodium chloride, potassium, calcium and phosphate) (Pácha 2000) what explain the observed edema in the submucosa that could to lead to malabsorptions. Malabsorption syndrome can be caused by gastrointestinal parasites (Mettrick and Podesta 1974; Horn and Fine 1977). Moreover, in severally infected hosts, the malabsorption by the host (as results of mucosal reduction as observed in Table 1) added with the host-parasite nutritional competition [due to a complex syncytium in the body wall with several pores and canals to absorption in Acanthocephala (Hayunga 1991; Bush et al. 2001)] can increase clinical signs of preexistent pathologies or contributes to break of the homeostasis in the host (Mettrick and Podesta 1974; Thomson 1983; Carlton and McGavin 1998). Thus, the parasite ecological pattern associated with its high number, as recorded by George-Nascimento and Marin (1992) and Aznar et al. (2004), could result in considerable intestinal damage affecting the physiology and histology or as windows for secondary infections. Dezfuli et al. (2002), record *Pomphorhynchus*-related catarrhal enteritis and strong infiltration of granulocytic cell series. For *A. australis*, the catarrhal enteritis was Cestoda-related [Diphyllbothriasis and Anoplocephalosis (RZ Silva et al. unpublished data)] but granulocyte infiltration was not present.

Conclusion

Corynosoma australe does not cause inflammatory reactions in the intestines of *A. australis*, but its attachment-detachment sites during adult life stage show a chronicle and continuous pattern of mucosal embedding until reach the intestinal submucosa.

Acknowledgments The authors thank to the Instituto Brasileiro do Meio Ambiente e dos Recursos Naturais Renováveis (IBAMA) by the permission of biological sampling licence (SISBIO licence 17529-1), to M.Sc. Alessandra Alves da Rocha and M.Sc. Paulo Henrique

Mattos by contributions of the host specimens and the Ph.D. Eduardo Resende Secchi and the Ph.D. Luis Alberto Romano for the general review of this work.

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