adult and larval *Ostertagia circumcincta* was postulated to be due to the parasites releasing chemical/s with possible detrimental effects on parietal cell function and survival[54]. A sequential study in sheep transplanted with 20,000 adult *O. circumcincta* described features in parietal cells suggestive of necrosis[55], the normal process of death in parietal cells[27]. A nematode-mediated parietal cell dysfunction was reported in sheep infected with *O. leptospicularis* infective larvae[19] and was purported by a similar claim in an observation of *O. ostertagi* infection in calves that resulted in the loss of acid-secreting parietal cells[15]. An indirect suppression of parietal cells by inhibiting secretory activity of enterochromaffine-like (ECL) cells was induced by *Haemonchus contortus* ES product[18]. Both excretory-secretory products of *O. circumcincta* and *H. contortus* have been implicated in the inhibition of gastric acid secretion and vacuolation, and the loss of parietal cells associated with abomasal parasitism[41,47]. Simpson[57] suggested that parietal cell dysfunction was the key event that leads to loss of mature zymogenic cells and mucous cell hyperplasia in abomasal nematodosis. Nomura et al.,[43] established that parietal cells secrete a number of growth factors that influence the differentiation of other gastric lineages. The accompanying loss of zymogenic cells was likely a consequence of the interruption of the normal development pathway in the gastric mucosa that followed after destruction of parietal cells[39]. Parietal cells were observed to play a central role in the regulation of mucosal proliferation during gastric inflammations[3].

Apparent loss of parietal cells even before mucosal hyperplasia developed in SCID mice at 2 weeks post infection might be related with the reports in genetically engineered ablation of parietal cells that resulted in hyperplastic gastropathy[10,33]. Parietal cells are suggested to be able to influence proliferation of stem cells and modulate the terminal differentiation programs of mucous and zymogenic cells[10]. Blocking in the maturation of parietal cells was reported also to inhibit the terminal differentiation of zymogenic cell[33]. It was suggested that members of the parietal cell lineage are required for instructive interaction that affects differentiation of the zymogenic cell lineage at later stages of morphogenesis[23]. A form of compensatory enhancement of progenitor production involved due to the depletion of two major components of the gastric unit[33]. Pluripotential stem cells responded through increase mitotic activity continually accumulating cells that never differentiate into parietal and zymogenic cells.

Gastroenteropathy and growth factors

Transforming growth factor-alpha (TGF-α) is an important regulator of the proliferation, differentiation and physiological activity of gastric epithelial cells[32]. It has physiological functions similar to epidermal growth factor (EGF) and shares a receptor with it. Transgenic mice overexpressing TGF-α displayed mucous cell hyperplasia and diminution of parietal and zymogenic cells[56] similar to the *T. taeniaeformis* induced gastric hyperplasia. How parasites could alter production of growth hormone is still unknown. We have presumed in a previous report that ES products might contain cytotoxic molecules probably causing injury to the gastric mucosa[30]. This supposition was made in the light of the pathologic responses similar to other forms of gastric injury. Mucosal proliferation was observed following aspirin injury in rat stomach[44] and acute injury from hypertonic saline-induced denudation of gastric mucosa elicited a rapid upregulation of pathways leading to production of pit mucous cells[37]. Taken into consideration that excretory-secretory products can cause parietal cell loss and mucosal damage, both these events upregulate TGF-αexpression and promote foveolar hyperplasia and may further inhibit parietal cells[57]. In acute gastric injury, expression of TGF-αmRNA and protein in mucosa was reportedly increased[46]. In creased gastrodoudenal concentration of TGF-$\alpha$was also observed in aspirin induced gastric mucosal damage[51]. TGF-αtransgenic mice demonstrated severe mucous cell hyperplasia, glandular cystic dilatation, increased gastric neutral mucin staining and achlorhydia, the failure to secrete detect-

able gastric acid[13,61], which were characteristic of the *T. taeniaeformis* induced gastropathy. Furthermore, chronic TGF-α overexpression was described to disrupt the normal program of gastric epithelial cell differentiation[66]. Histologically, pit cells and isthmal precursor cells were greatly expanded while parietal and zymogenic cells were depleted. Moreover, mucous neck cells, precursors of zymogenic cells were found to be increased in transgenic mice and occupied the position taken by zymogenic cells in base region. These features were supportive for a case of TGF-α being involved with the anomaly described in this study.

Extracellular factors capable of receptor-mediated signal transduction were believed to play a prominent role in the complex regulation of stomach function, including cellular growth and differentiation[34]. These include epidermal growth factor (EGF) family of which TGF-α is a member[52]. *H. pylori* infection was reported to upregulate expression of amphiregulin and heparin-binding EGF-like growth factor at the mRNA and protein levels which was suggested to stimulate epithelial cell proliferation[64]. TGF-α and EGF immunoreactivity, however, was less observed in *T. taeniaeformis* infected rats showing gastric mucosal hyperplasia suggesting a pathogenesis independent from these growth factors[24]. Konno[24] assessed the involvement of this growth factor by EGF/TGF-α immunostaining the submandibular glands largely secreting EGF. Results showed that gastric hyperplasia still developed while there were no EGF/TGF-α positive cells stained within the hyperplastic mucosa. Development of mucosal hyperplasia even with the lack or absence of these growth factors supposedly involved in such lesions could evoked a presumption that TtLES product might contain molecules/proteins which can act similarly as EGF/TGF-α do. Plerocercoids of Spirometrid tapeworms synthesize and release plerocercoid growth factor that is transported by the blood, interacts with growth hormone receptors and mimics many of the biological actions of growth hormone[20].

Enteropathy, goblet and mucosal mast cell number increase

Enteropathy was also observed in *T. taeniaeformis* infected rats, although no such lesion was observed in SCID mice models. Majority of investigations using rodent-nematode model strongly suggested that loss of worm burden from the host could be partly achieved by an increase in mast and goblet cells[35]. Goblet cell secretes mucus considered to serve as a protective function by excluding or trapping worms in immune animals or by hindering intimate contact with the mucosa, thus preventing their establishment. Goblet cell hyperplasia and alteration of the terminal sugars of goblet cell mucins were observed in the expulsion of *Nippostrongylus brasiliensis* from host rats[22]. Mast cells on the other hand, were associated with the expulsion of *Stongyloides ratti*[42]. Highly sulfated glycoconjugates derived from mast cells and goblet cells, act as mucosal surface barrier against Strongyloides worms[22].

The reason why this hepatic parasite would induce such lesions in the small intestine and colon is incomprehensible. Although, TtLES contains glycosaminoglycans[16] that might be a type of glycoconjugate most intestinal helminths secreted as adhesion substances. Goblet and mast cell hyperplasia is the host's response against parasite-derived glyconconjugates to avoid attachment[22].

Conclusions

TtLES products have been proved to induce the digestive tract lesions in this unique phenomenon between hepatic larvae and gut mucosa. Development of hyperplasia is dependent on the volume of product partly influenced by the immune status of the host. It is speculated that larvae-derived factors may have similarities with that of gastrointestinal nematodes and their actions revealed identical host responses.

Host response in gastric mucosa is related with responses to gastric nematode infection. Another speculative idea however regards components of TtLES to have EGF or TGF-α like

actions that induce the hyperplatic lesions. Intestinal response showed some comparable lesions with intestinal nematodes such as *N. brasiliense* and others.

Proliferative studies clarified that in gastric mucosa, initiation of hyperproliferation was in the stem cells by direct initiation of TtLES or indirectly through parietal cell ablation. Massive accumulation of precursor cells occurred due to inhibition of differentiation and maturation into parietal and zymogenic cells. Enteric mucosa did not show increase in proliferating cells but delayed cell cycle seemed to cause villi length increase and mucosal cell hyperplasia.

The bulk of information is still insufficient to fully comprehend the mechanism of gastroenteropathy. Until recently, the focus of interest was on the pathophysiological effects of larvae infection on gut mucosa. Based on the above findings it is suggested that direction of researches should lean towards molecular characterisation of TtLES product components and their interactions with host defence mechanisms. That could provide deep insights on host-parasite relationships and may provide hints about immunomodulatory molecules or the role of helminths in carcinogenesis. Both are significant in greater understanding of the prevention and control of related parasitic diseases.

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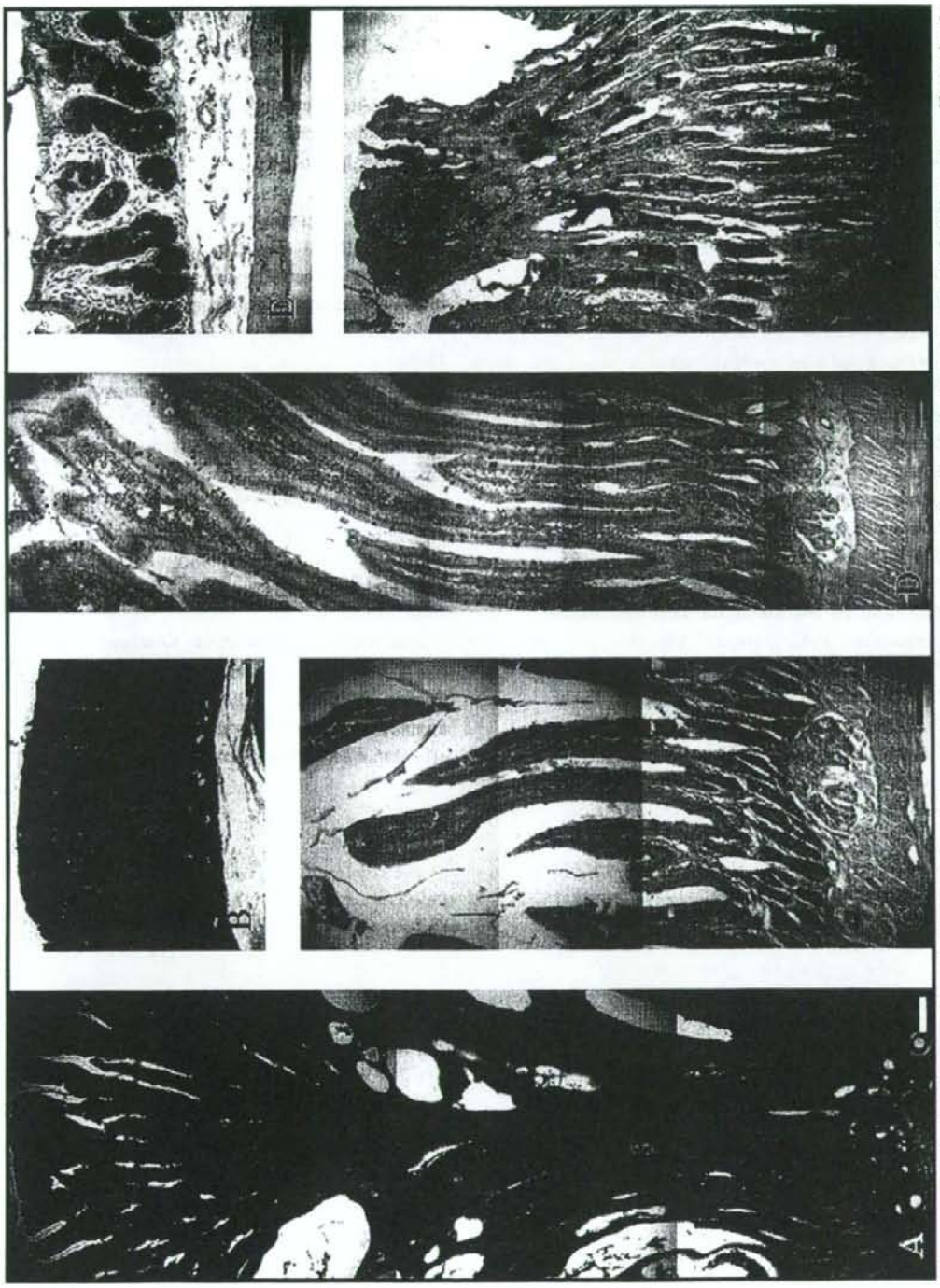

Figure 1 Histological features of the *Taenia taeniaeformis* larvae induced gastroenteropathy stained by alcian blue-periodic acid-Schiff reaction. Hyperplastic mucosa of the gastric gland (A) of an infected rat showing 3 to 4 times thickened when compared to a control gastric mucosa (B). Duodenal epithelia of an uninfected rat (C) is twice shorter compared to the hyperplastic duodenum of an infected rat (D). Colonic mucosa of a control rat (E) is lesser in villi length compared to the colonic mucosa of an infected rat (F). Bars = 100 μm.

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Water and food under pressure by pathogens such as *Cryptosporidium* and *Echinococcus*

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Abstract

Environmental contamination with domestic and wildlife fecal materials is increasingly recognized as a potential pathway for transmission of zoonotic parasites such as *Echinococcus* and *Cryptosporidium*. *Cryptosporidium* species is becoming a threat to public health due to contamination of drinking and recreational water. *Echinococcus multilocularis*, a helminth parasite causing a fatal zoonotic alveolar echinococcosis, is also considered one of the major threats to public health in Japan. To counter devastating outbreaks of cryptosporidiosis we advocate for sustainable control options against environmental contamination and endogenous development. Overall, we promote to counter these emerging environment-borne zoonoses at the source of contamination to create a healthy environment for a healthy people.

Introduction

Environment-borne diseases from air pollution, chemical and other environmental hazards are well known. Nevertheless, environmental contamination with domestic and wildlife fecal materials is increasingly rec-

ognized as a potential pathway for transmission of zoonotic parasites such as *Echinococcus* and *Cryptosporidium*[1,2]. Contamination of terrestrial and aquatic environment by these parasites pose enormous threat to public health thus requires further understanding and development of sustainable control options.

Cryptosporidium is a genus of pathogenic protozoa that infect a wide range of vertebrate hosts which recently is considered as an emerging water-borne zoonosis in Japan. It is one of the major causes of diarrheal disease both in immunologically and immunocompromised humans and animals, worldwide[3]. Although infection in human host is self-limiting, potentially life threatening disease may develop in young, elderly or immunocompromised hosts[4]. Dehydration and cardiovascular failure secondary to the intractable diarrhea have caused death in the above patients[5]. Moreover, an effective therapy for treating the infection is yet to be discovered.

Cryptosporidium species is becoming a threat to public health due to contamination of drinking and recreational water[6]. Outbreaks documented caused by the contamination of community water systems did involve a large number of populace and created economic consequences beyond the health care of the victims[7]. Aside from a known waterborne disease, the life cycle of this protozoan is also suited to foodborne transmission[8]. The fecal-oral transmission of the protozoan can cause contamination of raw or undercooked food handled by a person infected with Cryptosporidium.

On the other hand, *Echinococcus multilocularis*, a helminth parasite causing a fatal zoonotic alveolar echinococcosis, is considered one of the major threats to public health in Japan. Reportedly, around 500 patients in this country were confirmed by surgical method[9]. Most often the status of the patients was considered severe and difficult to treat. Although the exact means of transmission is not clear, wild fox population is high-

ly recognized as the primary source of infective eggs that contaminates the environment through their faeces. Humans and intermediate host animals could be infected by ingesting *E. multilocularis* eggs through food or water from the contaminated environment.

Human echinococcosis, although relatively rare and generally considered an accidental spill-over from the wildlife, is one of the most difficult invasive helminthic infections to diagnose, to treat effectively and also to undertake post-treatment evaluation[10]. The disease in humans is characterized by alveolar, hepatic, and cerebral disorders caused by the larval form (metacestode) of the tapeworm *E. multilocularis*. In humans, who may serve as accidental intermediate hosts, the metacestode cells of *E. multilocularis* proliferate like those of tumor cells. When clinical signs are manifested, it becomes very difficult to treat and without therapy the disease is fatal. Complete cure could only be achieved if confirmatory diagnosis is done during the early stage of the disease followed by complete resection of all lesions.

Water under pressure by Cryptosporidiosis

Cryptosporidium is an ubiquitous, obligate intracellular protozoan parasite with oocyst as the only stage found outside the host and is the infectious form. The life cycle is direct and infective oocysts are released with the feces of hosts. These oocysts contaminate the environment and can survive for several months in suitable conditions such as cool and damp areas, and can remain infective for considerable period of time in water[11]. Susceptible hosts get the infection by ingesting the oocysts through contaminated drinking or recreational water and sometimes through contaminated food[8]. Single oocyst is sufficient to produce infection and disease in the animal model[12].

A major zoonotic species, *C. parvum* is recognized as a common

pathogen of domesticated livestock and poultry, companion animals, and wildlife[13]. It is considered a major contributor to environmental contamination and their marked resistance to environmental and water treatment stresses made them more potential for transmission to humans[8]. *C. parvum* has been recognized as a common pathogenic species of the gastrointestinal tract of humans and animals[14]. Human infection is through fecal-oral route usually by ingestion of contaminated food and water (drinking and recreational). This zoonotic species was detected in high prevalence among dairy cattle farms in Japan[15]. Zoonotic genotype 2 of *C. parvum* was identified both from cattle and human isolates in Japan[16] that proved cattle as an important source of zoonotic human cryptosporidiosis.

Information on *Cryptosporidium* oocysts in the drinking water supply system in Japan is very limited. It was previously believed that environmental waters in Japan were cleaner and safer compared to other countries. Recently, however, the waterworks system here appears to have entered a critical phase. This was proven due to the emerging waterborne outbreaks of cryptosporidiosis both from contaminated drinking and recreational water[3]. A study confirmed the contamination of river waters with oocysts of *C. parvum* in northern Japan island of Hokkaido that were linked to agricultural activities like dairy farming[17]. The population (year 1991) of cattle in Tokachi district of Hokkaido, one of the largest dairy farming zones in Japan, was almost 300 thousand and the excrement of the cattle were inadequately treated resulting a serious environmental contamination in nearby bodies of water[18] (Sakai et al., 2002).

Table 1 shows the summary of reported Cryptosporidiosis outbreaks in Japan. The first documented outbreak was in the year 1994 in Kanagawa prefecture where about 500 patients were confirmed. In this outbreak, the cause was contamination of drinking water where the

Table 1. Documented outbreaks of cryptosporidiosis in Japan.

Year	Place	Number of Infected persons	Causes
1994	Kanagawa	~ 500	Contaminated drinking water (Kuroki et al,1996)
1996	Saitama	8,705	Contaminated drinking water (Yamamoto et al, 2000)
2002	Hokkaido	---	(Tsushima et al, 2003)
2004	Nagano Chiba	222	Swimming pool (Takagi et al, 2008; Yokoi et al, 2005; Ichinohe et al, 2005)
2006	Osaka	4	raw meat dish (Yoshida et al, 2007)

oocysts were detected in tap water and other water samples taken from several water tanks and pits of the building. There was post treatment contamination of municipal drinking water with sanitary sewage through the connecting pipes. Accidental malfunction of the drainage system was mentioned as the suspected cause. Moreover, the waste-water pump was found broken at the time of the outbreak[7,19].

The second and the largest outbreak in terms of infected patients occurred in Saitama prefecture in the year 1996 that affected more than 8,000 people. It was also associated with contaminated drinking water since oocysts were detected in untreated and treated water[7,20].

Two outbreaks have been claimed to occur also in Hokkaido[3]. Although there are no published data on number of patients involved, the two cases were associated with dairy cattle farming which is a common agricultural venture in the island.

An outbreak in 2004 involving 222 among 273 school children in a sports center was reported in Nagano Prefecture. A secondary transmission outbreak of cryptosporidiosis was documented in Chiba Prefecture

after recovered patients from the Nagano outbreak who are still excreting oocysts used pools and infected several people. Water from both swimming pools tested positive to *Cryptosporidium* oocysts[21, 22].

Recently, in 2006 a rare food-borne cryptosporidiosis outbreak was recorded in Osaka Prefecture. The case involved 4 members of a company who ate raw meat dish called "Yukke: Korean-style beef tartar" and a raw liver at a rotisserie[23]. The patients showed signs typical to cryptosporidiosis and laboratory examination detected *Cryptosporidium* oocysts from their fecal samples. PCR analysis identified the isolates as the zoonotic *C. parvum* genotype II[23].

Food under pressure by Echinococcus

Echinococcus multilocularis, is a highly pathogenic tapeworm of the Family Taeniidae. Humans are infected by ingestion of the parasite eggs, suspected to be through water but food such as agricultural products contaminated with infected eggs from wild fox feces is also thought as an important transmission route.

The parasite is maintained naturally in the wild by predator/prey life cycle. Foxes, dogs, coyotes, raccoon dogs and other canids can serve as definitive hosts. Cats are susceptible to infection but appear to have only a minor role in the maintenance of *E. multilocularis* in endemic areas, and infections in cats may be of minimal public health significance[24]. However, recently a cat was diagnosed to have excreted *E. mutilocularis* eggs in Hokkaido[25].

The definitive hosts of *E. multilocularis* in Hokkaido are known to be the red foxes, *Vulpes vulpes schrenki*, as well as the dogs and cats[26]. Based on the increasing population of red foxes in Hokkaido and their high prevalence rate of echinococcosis Fig.1 they are considered the major definitive host in the wildlife[9]. Prevalence rates in cats and dogs

are very low, nevertheless their role in transmission of the parasite to humans cannot be overlooked due to proximity and frequency of contacts with humans[27]. Wild rodents such as voles are known intermediate hosts while pigs, horses, primates and humans can be infected as accidental intermediate hosts. It has long been known that the most important intermediate host of *E. multilocularis* in Hokkaido is the Bedford vole, *Clethrionomys rufocanus bedfordiae*.

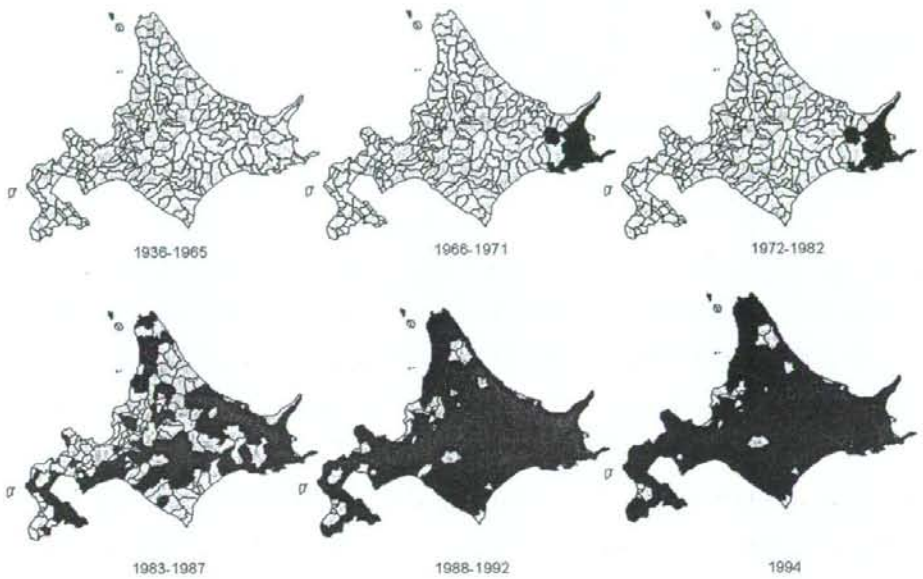

Figure 1. Expansion of endemic areas of *Echinococcus multilocularis* infection in foxes in Hokkaido from 1936 to 1994.

Alveolar echinococcosis in humans is endemic in Japan; however, the causal agent, *E. multilocularis*, has been restricted to the northernmost insular prefecture of Hokkaido, where the Tsugaru Strait acts as a natural physical barrier against migration to the mainland. The early human cases of alveolar echinococcosis were reported from Sendai on northern Honshu in 1926 and on Rebun Island in 1937. Since then, more

than 500 human cases have been diagnosed in Hokkaido and around 20 new patients have been reported every year since 1982. One study calculated that the rate of occurrence during the endemic period was 48 cases per 100,000 residents every year[28].

In September 2005, a stray dog in Saitama prefecture in mainland Honshu was found to be positive for *E. multilocularis* infection by PCR (mitochondria 12S RNA gene)[29]. The sequence was identical to the Hokkaido isolate (GenBank accession no. AB244598). This raised an alarm because the area in which the infection was found is adjacent to the Tokyo metropolis, the most populous zone in Japan. Reports also claimed that 2 of 69 dogs moved from Hokkaido to Honshu were positive for *E. multilocularis* by coproantigen examination[30]. Moreover, a non-Hokkaido resident dog became infected with *E. multilocularis* despite being permitted to roam freely for just a few hours during its 5-day stay in Hokkaido[31]. This finding suggested a high infection pressure of *E. multilocularis* to domestic dogs within the area. Furthermore, in April 2004 to August 2005, from a total of 1460 domestic dogs examined, 4 (0.27%) were confirmed positive to echinococcosis by PCR, all from Hokkaido[32].

While the threat of echinococcosis spreading into Honshu had raised fears, an emergent concern is the possible role of domestic dogs in dispersing the disease from disease-endemic areas during relocation of residences by owners or when accompanying owners during domestic travel. Nearly 10,000 pet dogs were estimated to have been transported in 1 year to and from Honshu and Hokkaido by planes and ferries; this presumably included up to 30 *E. multilocularis*-infected pet dogs per year[33].

Confirmed cases of infection in dogs further showed the potential threat of domestic dogs transmitting *E. multilocularis* to humans in this region, as well as the potential for dispersal to other islands of Japan if proper preventive measures are not implemented.

Despite that only sporadic reports of human cases are reported in

Japan, it is predicted that the incidence of alveolar echinococcosis will increase in the near future if no effective preventive measures are put in place[34].

Perspectives on control options

Cryptosporidiosis

Practical interventions that could reduce environment-borne cryptosporidiosis include good animal husbandry and control of mechanical vectors, and proper manure/feces disposal[35]. Reducing oocyst excretion in livestock may be important using agents such as paromycin and decoquinate[36]. In spite of extensive screening of a large number of chemotherapeutic agents, to date there is no reliable curative treatment of cryptosporidiosis[37].

Monitoring of *Cryptosporidium* oocysts is one of the most important steps in dealing with cryptosporidiosis[3]. It is also especially important to determine the genotype and infective potential of the oocysts[38,39]. Assessing infective potential of the *Cryptosporidium* oocysts will help provide accurate data for estimating risks in different environments[38].

Surveys on the prevalence of cryptosporidiosis in cattle farms and water sources should continually be conducted. More significant are the cattle farms located near or in watersheds where manure or feces may contain potentially zoonotic genotypes. An important step in the quantification of risk is to understand the dynamics of infection with, and the perpetuation of, the organism in these farms[4].

Investigation of environmental waters such as rivers and lakes that are used as sources for drinking and recreational waters is equally important. Sampling from rivers in Hokkaido, Japan in areas heavily populated with dairy farms revealed significant presence of viable *Cryptosporidium* oocysts[3,17]. These merit further surveillance in other

parts of the country as well.

Frequent and routine monitoring of filtration performance offer the best options for control of cryptosporidiosis in water treatment systems[40]. They also suggested that the reduction of between 1.25 and 2.45 log10 oocysts in conventional water treatment systems is achievable. The use of ultraviolet irradiation and ozone is proven useful in inactivating oocysts[41].

The best option for control of cryptosporidiosis therefore is vaccination. Vaccines have been shown to reduce clinical signs, but in most cases have not eliminated or reduced oocyst shedding[41]. However, considering that *C. parvum* is common among neonates, passive immune protection by vaccination of dams is a promising approach[42]. It was reported that calves receiving colostrums containing high concentration of immunoglobulin G antibodies to *Cryptosporidium* showed reduced diarrhea and oocyst shedding[43].

Echinococcosis

It has since been asserted that there is no reliable, cost-effective method for the sustainable control or eradication of *E. multilocularis*[44]. In spite of this assertion, a deworming trial of red foxes was explored in Koshimizu (200 km^2), Hokkaido, Japan since 1997. Anthelmintic-fortified bait, which consisted of commercial fish sausages (1.5 cm long) embedded with one half of a 25 mg praziquantel tablet (Droncit; Bayer, Germany), was distributed manually in approximately half of the total area around the fox dens on a monthly basis on foot. The baiting trial showed that there was a decrease in the taeniid egg infection rate in foxes in the baited area after one month, and that this suppressive effect persisted in the following years, despite a decrease in the number of times the bait was distributed. In a follow-up trial conducted in April 2001, praziquantel- fortified bait was distributed throughout the entire

area of Koshimizu alongside roads, at intersections and at wind-shield forests by local residents using cars to allow for faster mobility. The bait was made from fish-waste products, using the same procedure that is used for manufacturing "kamaboko" (Fig. 2) fortified with praziquantel (50 mg/ piece of bait). Post baiting trial results revealed that taeniid egg and coproantigen prevalence rates were significantly decreased in foxes inside the treated area[45].

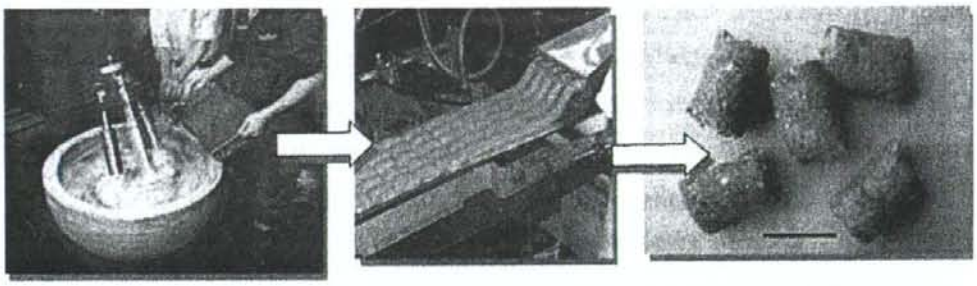

Figure 2. Manufacture of baits using local resources such as the fish-waste products. Baits are fortified with praziquantel.

In Kutchan, another echinococcosis endemic area in Hokkaido, Japan, a monthly distribution of approximately 1,500 pieces of bait was conducted between May and November of 2006. The bait was distributed with the use of GIS- based maps to identify the foraging habitat of wild foxes. Remarkably, the prevalence rates of taeniid egg and coproantigen positive feces dropped significantly after less than a year of baiting.

Strengthening public health policy

Improvement of reporting system is an important tool in monitoring and developing early warning systems of outbreaks. In alveolar echinococcosis, a compulsory reporting system has strengthened a monitoring system to trace infections and prevent further contamination of

susceptible individuals. During amendment of the Infectious Disease Law in Japan, certain specific zoonotic diseases were included in the 4th category of diseases, i.e. diseases which must be reported. Echinococcosis in dogs was incorporated into this category along with bacterial dysentery in primates and West Nile fever in birds. In its 20th session, the Infectious Disease Evaluation Committee of the Ministry of Health, Labour, and Science, passed a resolution that made it mandatory for veterinarians who diagnose echinococcosis in dogs to report the case to the health authorities. Thus, a national reporting system for dogs infected with *E. multilocularis* has been implemented by veterinarians since October 2004 to monitor and control the occurrence of infection in dogs throughout the country[46]. This policy might be applicable should Cryptosporidiosis be taken into serious consideration.

Research laboratory initiatives

It is believed that research institutions have an important role to play in extending practical assistance to the public as the knowledge and understanding of the disease advances, especially with regards to its control and prevention. Thus, according to Zinsstag[47] "Although there is no doubt that progress in animal health research must continue, it must also respond to societal needs and lead to solutions that can be delivered quickly." Therefore, in 1999, Forum on Environment and Animals (FEA, Fig. 3) was established to link important organizations including government offices, academic institutions, international agencies (e.g. the OIE), veterinary associations and non-governmental organizations, such as NPOs comprised of local residents, that all have the primary goal of controlling echinococcosis in Hokkaido. A spearheading institution is significant in the countermeasure of environment-borne zoonotic diseases.

Figure 3. Composition and aims of the Forum on Environment and Animals(FEA).

Endogenous development as a revolutionary tool

"Endogenous development" involves building on local resources, enhancing in situ development, maximizing local control of the development process, and recognizing the needs and the values of local residents[48]. As the Dag Hammarskjöld Report[49] puts it, such development relies on what a human group has: its natural environment, its cultural heritage, the creativity of the men and women who constitute it, becoming richer through exchange between them and with other groups and entails the autonomous definition of development styles and of life styles.

In all of the baiting campaigns for Echinococcosis control mentioned above, the endogenous initiative of local residents, which was facilitated by NonProfit Organization (NPO), was highly instrumental. Zoonotic diseases are of concern not only to public health personnel but also to individual residents who are at risk of infection. Moreover, the use of local