Dogs as the source of *Giardia* in Bergen in 2004 – barking up the wrong tree?

In an article published in this journal on the outbreak of giardiasis in Bergen, dog faeces were suggested as a more probable source of infection than sewage leakage. In our opinion this is unlikely and this hypothesis could create unnecessary worry. Based on available knowledge, *Giardia* infection from humans remains the most probable cause of both the outbreak of infection and the long-term health problems.

In a recently published article on the outbreak of giardiasis in Bergen in 2004 (1), Torgeir Landvik puts forward arguments for two different hypotheses. The first is that faeces from dogs were the most likely source of the *Giardia* that was found in the drinking water and was the cause of the outbreak, and not sewage leakage from houses in the neighbourhood as had been concluded in a previously published report (2). The second hypothesis was that the long-term health problems in some of the patients were due to zoonoses from dogs other than giardiasis. We welcome discussion around the management of drinking water sources, but in our opinion the author in this instance omits several important factors in his arguments. These factors are discussed here.

Is infection from dogs probable?

In his article (1), Landvik cites Sherlock Holmes «When you have eliminated the impossible, whatever remains, however improbable, must be the truth». Landvik believes that the evaluation committee, having excluded animals as a likely source of infection, therefore landed upon sewage leakage as the only possible cause, solely because they had eliminated everything else. This also implies that the sewage leakage theory is unlikely. However, many outbreaks of waterborne parasitic infections, where the source of infection has actually been identified, demonstrate contamination from sewage (3). In other words, an association between sewage leakage and waterborne disease outbreaks is not uncommon, and there appears to be no clear explanation of why Landvik believes it to be unlikely in this particular case.

Based on publications about previous waterborne disease outbreaks, faeces from dogs have never before been identified as a source of infection. Landvik himself emphasises how unusual this is by calling it «the first drinking water epidemic due to faeces from dogs».

Although we agree that direct investigations of sewage from the relevant houses ought to have been conducted after the outbreak was detected, something that was also recommended, we were nevertheless obliged to respect the regulations intended to protect the privacy of individuals. In the wake of the large waterborne cryptosporidiosis outbreak in Östersund, Sweden in 2010, sewage leakage from a building containing several apartments was considered the most likely source of infection, but closer investigations to identify the specific apartments were not conducted (4). Additionally, because contamination of the water occurred several months before the outbreak was discovered, the results of such an investigation in Bergen would probably have been misleading.

However, one thing is clear – the sewer outlet from the buildings in the neighbourhood was located only a short distance (200–300 m) from the raw water intake, while run-off of dog faeces from the area along the footpath around the lake would depend on where they are deposited and the terrain down to the lake. After the outbreak was identified, between 5–10 kg of dog faeces were collected from this footpath. Ten sub-samples were analysed at the Veterinary School, NMBU for both *Cryptosporidium* and *Giardia*; all were negative (2). These results do not exclude *Giardia* infection in dogs in the area, but neither do they provide any evidence for greater suspicion.

«It is important to put forward hypotheses that are based on empirical data; speculation without substantial evidence should be avoided»

Rather than presenting these findings, Landvik has concentrated on a survey of young dogs (5). Although the cumulative prevalences found in this study were, as quoted, relatively high (44 % for *Cryptosporidium* and 21 % for *Giardia*), the individual prevalences were only half as high in puppies (23 % for *Cryptosporidium* and 12 % for *Giardia*) and far lower in adult bitches (< 3 % for *Giardia* and < 4 % for *Cryptosporidium*). It is known that these parasites are most common in younger animals, and therefore the age distribution amongst the dogs that were being walked around Svartediket should be taken into consideration. Furthermore, the intensity of the infection should be taken into account – of the puppies that were positive in the study by Hamnes and colleagues (5), approximately 50 % had high numbers of *Giardia* cysts in their faeces, while only 17 % had a high number of *Cryptosporidium* oocysts. The infection pressure is not only determined by the quantity of dog faeces on the footpath that contain *Giardia* cysts or *Cryptosporidium* oocysts, but also the number of cysts and oocysts per gram. Although the estimated amount of dog faeces (130–521 kg) in the area around Svartediket can be perceived as alarming, interpretation of its significance regarding the amount of *Giardia* or *Cryptosporidium* is far more complex. Furthermore, studies at Norwegian sewage works have shown that *Giardia* and *Cryptosporidium* occur frequently in high concentrations (from 4,000 to over 20,000 cysts/oocysts per litre of sewage) and the results from Bergen (in samples taken before the outbreak) were particularly high (6).

Giardia and *Cryptosporidium* in dogs

There has been a considerable increase in our knowledge about parasitic species and genotypes during the last 10 years, and currently it is accepted that dogs can be infected by four different genotypes (A-D) of *Giardia duodenalis*. Of these, only genotypes A and B are considered to be infectious for humans (potentially zoonotic genotypes). Giardiasis in dogs can thus be caused by the zoonotic genotypes, but the dog-specific variants, genotypes C and D, are much more common, and most scientists believe that *Giardia* in dogs has little significance as a potential zoonosis.

Genotyping of *Giardia* from 27 dogs in Norway showed that most (about 48 %) were genotype D, approximately 22 % were genotype C, 20 % were genotype A, and 4 % were genotype B (7). This is not an unusual result; in a review article of studies investigating *Giardia* genotypes in dogs, it emerges that they often are infected with

genotypes C and D. A small number of studies showed a higher incidence of genotype A (which can also infect humans), but genotype B in dogs is considered very rare (8). Similar data are reported by Ballweber et al. (9). Our analyses showed that the *Giardia* outbreak in Bergen was due to genotype B3 (10–12), and thus the probability that this infection originated from dog faeces containing this genotype on the footpath is extremely low.

Several species of *Cryptosporidium* have been described, of which *Cryptosporidium hominis* and *Cryptosporidium parvum* cause most human infections. Analyses from 12 different patients in Bergen showed that all of them were infected with *C. parvum* (13). *C. parvum* has been reported from dogs in some individual cases (14), but most infections are caused by *C. canis*, which is considered to be a dog-specific type (e.g. 15). Unpublished data from Norway (Parasitology Laboratory, NMBU, Oslo) suggests that the same situation is also true of Norwegian dogs. The risk of infection of healthy people with *Cryptosporidium* spp. from dogs is therefore considered to be low (16). Thus, the available data indicate that dogs do not usually act as reservoirs of infection for *Cryptosporidium* or *Giardia* that are considered to be infectious for healthy humans (17).

Can *Giardia* cause long-term health problems?

Research conducted at the University of Bergen and Haukeland University Hospital has so far been unable to provide definitive answers to explain the relationship between *Giardia* infection and the chronic disease progression that was observed. However, in addition to our observations, an increasing number of publications have shown that it is not infrequent for intestinal infections to trigger long-lasting gastrointestinal complaints in infected patients (18). Infections can also be followed by fatigue (19). It is also well known that infections that are confined to the intestines can elicit generalised immunological responses.

Giardia is known to be associated with a wide spectrum of symptoms (20), and the outbreak in Bergen is not the first parasitic infection to be associated with prolonged irritable bowel, so-called post-infectious irritable bowel syndrome (PI-IBS); as far back as 1962, bacterial and/or amoebic dysentery was associated with this condition (21), and several mechanisms have been suggested as contributors to the development of long-lasting illness following *Giardia* infection (22). In a study of people infected with *Giardia* in the USA (it should be noted that these infections were not in association with outbreaks), extra-intestinal symptoms were found to be relatively common (23). It is also worth noting that the waterborne cryptosporidiosis outbreak in Östersund was also associated with prolonged gastrointestinal symptoms and joint pains (24).

Landvik contends strongly that infection with zoonotic pathogens from dogs (without specifying in greater detail which agents he has in mind) could be the cause of the chronic symptoms in some patients after the outbreak in 2004. When the outbreak occurred, the drinking water was treated by chlorination, which excludes most bacterial infections, and there is little to suggest that viruses with zoonotic potential occur in dog faeces.

Based on Landsvik's previous interest in this field, we suspect that he could be thinking that the nematode *Toxocara canis* (the dog roundworm) or tapeworms may be an undiscovered cause of these patients' ailments. Some of these are indeed potentially zoonotic and can cause a wide variety of protracted symptoms in humans (25), although with clinical signs and symptoms that are very different from those of giardiasis that the patients had during the outbreak.

Helminths (such as roundworms and tapeworms) are rarely found in the water supply as the eggs are so large and relatively heavy that they do not get through the treatment process. They are therefore usually only considered a problem in untreated irrigation water. Although the treatment of drinking water in Bergen at around the time of the outbreak did not include filtration, it is reasonable to assume (Stoke's law) that sedimentation of helminth eggs in water is much faster than for protozoan cysts and oocysts.

Roundworms are diagnosed relatively infrequently in Norwegian dogs, and tapeworm is even more unusual. *T. canis* is considered endemic in the Norwegian dog population, but because adult dogs largely harbour hypobiotic larval stages, it is mainly young puppies with acute *Toxocara* infection that will have sexually mature adult worms in their intestines and that will excrete worm eggs in their faeces. *Toxocara* eggs excreted with faeces will not be immediately infectious; it takes several weeks under optimal conditions for the eggs to develop into embryos and thus be infectious to humans. During this period, it would be reasonable to assume that the eggs would have sunk below the lowest point of entry to the water supply.

T. canis infection (larval migrans) in humans results in systemic infections with typical symptoms such as fever and enlarged liver and spleen, or visual impairment. An increase in eosinophil granulocytes is a common finding in infections with multicellular parasites, including *Toxocara* infection. A survey from Sweden in 1989 found serology consistent with previous exposure to infection in 7 % of healthy people (26), but visceral larval migrans has not been diagnosed in patients at Haukeland hospital during the past 20 years. Patients examined for clinical signs after the outbreak in Bergen did not have eosinophilia. Likewise, MRI of the brain was performed in patients with chronic fatigue syndrome and this would have revealed any *Toxocara* focal lesions, should these have been present.

Concluding remarks

The economic and health consequences of the outbreak in Bergen were considerable, and discussion around the contamination of water sources is important. Knowledge that can contribute towards reducing the risk of new outbreaks of disease in the future is desirable. It is important to put forward hypotheses that are based on empirical data; speculation without substantial evidence should be avoided.

Based on scientific studies and facts as described above, we believe that it is unlikely that dog faeces were the source of the *Giardia* cysts that were in the water from Svartediket in 2004.

Moreover, we believe that there is no reason to suspect that other zoonoses from dogs were the cause of the long-term health problems that occurred after the outbreak.

As the physiologist Claude Bernard once said «*La meilleure théorie est celle qui a été vérifiée par le plus grand nombre de faits*» (27).

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The Parasitology Laboratory at the Norwegian University of Life Sciences is accredited for analysis of water for Giardia *and* Cryptosporidium. *During the giardiasis outbreak in 2004 the institute where LJ Robertson and K. Relling Tysnes are employed was paid by Bergen municipality for some analyses of water for contamination with Giardia.*

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