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), Torger 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.

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 stud-
ies 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 prob-
ability 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 sho-
wed that all of them were infected with
*C. parvum* (13). *C. parvum* has been repor-
ted from dogs in some individual cases
(14), but most infections are caused by
*Giardia*, which is considered to be a dog-
specific type (e.g. 15). Unpublished data
from Norway (Parasitology Laboratory,
NMBU, Oslo) suggests that the same situa-
tion is also true of Norwegian dogs. The
risk of infection of healthy people with
Cryptosporidium spp. from dogs is there-
fore considered to be low (16). Thus, the
available data indicate that dogs do not usu-
ally act as reservoirs for infection with
Cryptosporidium or *Giardia* that are consid-
ered 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 com-
plaints 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 genera-
lised immunological responses.

In *Giardia* infections, the symptoms are
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 condi-
tion (21), and several mechanisms have
been suggested as contributors to the de-
velopment 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 pro-
longed 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 chro-
nic symptoms in some patients after the out-
break in 2004. When the outbreak occurred,
the drinking water was treated by chlorina-
tion, which excludes most bacterial infec-
tions, and there is little to suggest that viru-
ses with zoonotic potential occur in dog
faeces.

Based on Landvik’s previous interest in
this field, we suspect that he could be think-
ing that the nematode *Toxocara canis* (the
dog roundworm) or tapeworms may be an
undiagnosed cause of these patients’ ail-
ments. Some of these are indeed potentially
zoonic and can cause a wide variety of pro-
trated symptoms in humans (25), although
with clinical signs and symptoms that are
very different from those of *Giardia* 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 relati-
vely heavy that they do not get through the
treatment process. They are therefore usu-
ally 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 (Støke’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 tape-
worm is even more unusual. *T. canis* is con-
sidered endemic in the Norwegian dog popu-
lation, 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 infect-
ious; 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.

*Toxocara* 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 multi-
cellular parasites, including *Toxocara* infec-
tion. 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 consid-
erable, and discussion around the contamina-
tion of water sources is important. Know-
ledge that can contribute towards reducing
the risk of new outbreaks of disease in the
future is desirable. It is important to put for-
ward hypotheses that are based on empiri-
cal data; speculation without substantial
evidence should be avoided.

Based on scientific studies and facts as
described above, we believe that it is unli-
kel y 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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comments.

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

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