Cystic echinococcosis in sub-Saharan Africa

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Cystic echinococcosis in sub-Saharan Africa

Kerstin Wahlers, Colin N Menezes, Michelle L Wong, Eberhard Zeyhle, Mohammed E Ahmed, Michael Ocaido, Cornelis Stijnis, Thomas Romig, Peter Kern, Martin P Grobusch

Cystic echinococcosis is regarded as endemic in sub-Saharan Africa; however, for most countries only scarce data, if any, exist. For most of the continent, information about burden of disease is not available; neither are data for the animal hosts involved in the lifecycle of the parasite, thus making introduction of preventive measures difficult. Available evidence suggests that several species or strains within the *Echinococcus granulosus* complex are prevalent in sub-Saharan Africa and that these strains might be associated with varying virulence and host preference. Treatment strategies (chemotherapy, percutaneous radiological techniques, but mainly surgery) predominantly target active disease. Prevention strategies encompass anthelmintic treatment of dogs, slaughter hygiene, surveillance, and health-educational measures. Existing data are suggestive of unusual clinical presentations of cystic echinococcosis in some parts of the continent, for which the causes are speculative.

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First Department of Internal Medicine, Hospital of the University of Cologne, Cologne, Germany (K Wahlers MD); **Department of Internal Medicine, University of the Witwatersrand, Johannesburg, South Africa** (C N Menezes MD, M L Wong MD, Prof M P Grobusch MD); **African Medical and Research Foundation, Nairobi, Kenya** (E Zeyhle Dipl Agr Biol); **Faculty of Medicine, Al-Neelain University, Khartoum, Sudan** (Prof M E Ahmed MD); **Faculty of Veterinary Medicine, Makerere University, Kampala, Uganda** (Prof M Ocaido PhD); **Centre of Tropical Medicine and Travel Medicine, Department of Infectious Diseases, Division of Medicine , Amsterdam Medical Centre, University of Amsterdam, Netherlands** (C Stijnis MD, Prof M P Grobusch); **Parasitology Unit, University of Hohenheim, Germany** (T Romig PhD); **Comprehensive Infectious Diseases Centre, University Ulm, Ulm, Germany** (P Kern MD); **and Institute of Tropical Medicine, University of Tübingen, Germany** (Prof M P Grobusch)

Correspondence to: Prof Martin P Grobusch, Centre of Tropical Medicine and Travel Medicine, Department of Infectious Diseases, Division of Medicine, Amsterdam Medical Centre, University of Amsterdam, Amsterdam 1100DD, Netherlands **m.p.grobusch@amc.uva.nl**

Introduction

Cystic echinococcosis is a zoonosis caused by cestodes of the *Echinococcus granulosus* complex. Adult tapeworms inhabit the small intestine of carnivores (the definitive hosts) and produce eggs, which are passed with faeces. The intermediate host (including sheep, cattle, donkeys, and camels) is infected by ingestion of eggs. Subsequently, a larval stage (metacestode) develops as a cyst in internal organs of this host. The metacestode produces many protoscolices, each with the potential to develop into an adult tapeworm when ingested by the definitive host. Cysts can be either viable or non-viable. Viable cysts are usually filled with clear fluid with few calcifications, whereas non-viable cysts are mainly calcified. Viable cysts can be either fertile, containing protoscolices, or sterile, containing only highly antigenic fluid.[1] People can become intermediate hosts after accidental ingestion of eggs; developing cysts cause the morbidity and mortality associated with the disorder. Liver and lungs (figures 1, 2) are the most commonly affected organs.[1–3]

Conventionally, the causative agent of cystic echinococcosis was regarded as one species, *E granulosus*. However, researchers have long known that this species is composed of several different taxa, which differ from each other in adult morphology, host preference, and pathogenicity (including to people).[4] To accommodate this diversity, ten different strains (G1–10) were described, with each being attributed to the intermediate host animal that was thought to be most important for transmission (eg, the sheep strain G1, or the camel strain G6). Eventually, some of these strains were reclassified as separate species, on the basis of substantial molecular differences. This reappraisal continues,[5] but currently, cystic echinococcosis of people or animals, or both, can be caused by *E granulosus* (G1–3), *E felidis* (the so-called lion strain), *E equinus* (G4), *E ortleppi* (G5), and *E canadensis* (G6–10).[6] Surveys done up to now generally do not take this differentiation into account and therefore information about distribution, hosts, and the clinical effect of different species and strains is scarce. *E granulosus* is believed to be the cause of most human cases; *E equinus* is thought to be the only species that cannot infect people. Apart from *E felidis*, all strains use the domestic dog as an intermediate host.[7]

Cystic echinococcosis occurs worldwide, and is endemic in several areas, particularly the Mediterranean, central Asia including the Tibetan plateau, northern and eastern Africa, Australia, and southern South America.[8–10] Generally, the highest prevalences of the disorder are in nomadic populations. Nomadic people keep dogs for various reasons, such as herding and guarding, as food, as bed-warmers, and as sanitation animals. The combination of people and dogs living in close proximity, scarce water resources, and conditions with poor hygiene provide the ideal environment for *Echinococcus* spp.[2] It belongs to the neglected tropical diseases group, which receive little funding for research and treatment relative to the burden of disease.[11,12]

The clinical signs and symptoms, diagnosis and management, and prevention and control of cystic echinococcosis, including in sub-Saharan Africa, have been reviewed.[13–15] With regards to the epidemiology of cystic echinococcosis in Africa, the pattern is patchy. Nowadays, researchers generally accept that this disorder is prevalent across the whole continent, with an area of high prevalence in east Africa (especially the Turkana region).[10] The aim of our Review is to summarise the data for cystic echinococcosis available from sub-Saharan Africa, and to best describe present knowledge of the epidemiology of this disorder in the region and options for treatment and prevention.

Treatment and prevention

In brief, researchers have developed classification systems, based on ultrasound findings, to differentiate early stage disease, active disease with daughter cysts, and end stage cystic echinococcosis.[16,17] Treatment strategies are derived from these classifications, and predominantly target active disease. Treatment options for cystic echinococcosis are chemotherapy, percutaneous radiological techniques, and surgery. Traditionally, surgery was the sole therapeutic option because of its potential to completely remove the parasite. Different surgical approaches have been developed with time,

See Online for appendix

Figure 1: So-called water lily sign in a patient with pulmonary cystic echinococcosis

Figure 2: Pulmonary hydatid cyst

ranging from cystectomy to more aggressive surgery, and ultimately liver transplantation. More invasive surgical techniques are associated with more complications (eg, infection, biliary leakage) but less recurrence of active disease than are conservative approaches.[18]

In the past 20 years, ultrasound-guided percutaneous procedures have partly replaced surgery as the treatment of choice. Puncture, aspiration, injection, and reaspiration (PAIR) and its modified version, percutaneous aspiration of cyst content (PEVAC) are safe and effective alternatives to surgery.[19] Benzoimidazole carbamates (albendazole or mebendazole) are widely established as chemotherapy for cystic echinococcosis. Drug treatment is used alone to suppress early stage disease, and adjuvant to surgical, PAIR, and PEVAC approaches for large, active hydatid cysts.[13]

Despite the possibility of drug treatment, interventional therapy is the mainstay of treatment for cystic echinococcosis in sub-Saharan Africa. This situation arises mainly because the resources needed to provide a reliable supply of expensive medication and laboratory facilities to monitor patients for side-effects are scarce. The nomadic lifestyle of many patients further complicates the matter.[20]

Prevention and control strategies for cystic echinococcosis consist of anthelmintic treatment of dogs, slaughter hygiene, surveillance, and health-focused education about human–dog behaviour. In the future, vaccination of livestock might be possible.[14]

Cystic echinococcosis in sub-Saharan Africa

The table provides an overview of surveys of human hydatid disease reported from sub-Saharan Africa. The appendix provides detailed information about individual case reports and case series of cystic echinococcosis in people in sub-Saharan Africa, and about surveys of echinococcosis in domestic animals from this region.

West Africa

Cystic echinococcosis is generally thought to be uncommon in west Africa and epidemiological studies have only been done in Nigeria (people and livestock) and Burkina Faso (livestock only). However, cases of human cystic echinococcosis are also reported from Senegal, Niger, and Ghana.[33–35]

Researchers have reported a small case series (n=32) from Niger. As in other parts of Africa, a female predominance was noted (n=20), but by contrast with data from other countries, a predominance of extrahepatic disease was recorded. In a livestock survey, 22% of 513 camels were infected with *E granulosus*.[34] One case of a patient from the Central African Republic has been reported, in which molecular typing identified genotype G6 (camel strain). However, the investigators noted that camels are absent from the Central African Republic and that cystic echinococcosis in sheep (another host of the G6 strain) has not been recorded there up to now.[36] Therefore the hosts involved in the lifecycle of *Echinococcus* spp in the Central African Republic remain to be elucidated.

A retrospective survey of cystic echinococcosis in livestock is available from Burkina Faso. Hydatid cysts were found in ten of about a million animals of various species.[37] No data for human disease are available, but this disorder is unlikely to be a major health problem in Burkina Faso. No genetic data exist from this country.

In Nigeria, cystic echinococcosis has been investigated more extensively. Human hydatid disease is not believed to be common, but is likely to be underdiagnosed.[28] A serological survey (complement fixation test) of hospital personnel and patients in the central north and southwest

	Method	Number of individuals	Prevalence
Ethiopia			
Southwest[21]	Physical examination, Casoni skin test	Clinical examination 640 Casoni skin test 175	Clinical examination: Dassanetch 11·3%, Nyangatom 5·8%, Kerre 5%, Hamar 1·6%, Suri 0% Skin test: Dassanetch 39·9%
Countrywide[22]	Physical examination, indirect haemagglutination, hydatid skin test	Clinical examination 3408 Indirect haemagglutination 1428 Hydatid skin test 986	Clinical examination: overall 2%, Dassanetch highest 5·1% Indirect haemagglutination: overall 1·7%, Dassanetch highest 6·4% Hydatid skin test: overall 15·7%, Dassanetch highest 36%
Nyangatom people[9]	Ultrasound survey	1334	2·9%
Boran people[9]	Ultrasound survey	110	1·8%
South (Hamar people)[23]	Ultrasound survey	990	0·7%
Hamar people[9]	Ultrasound survey	369	0·7%
Dassanetch people[9]	Ultrasound survey	267	0%
Kenya			
Turkana[20]	Retrospective, 21 years of surgical records	Total not available	710 procedures for hydatid disease in 663 patients
Turkana[24]	Patients presenting to hospital over 3 years	Total not available	355 patients treated for hydatid disease, male:female ratio 1:2
Turkana[25]	Serological survey, indirect microhaemagglutination test	1190	North Turkana 9·4%, 85/100 000 per year, South Turkana 2·1%, 25/100 000 per year
Turkana[26]	Retrospective review of hospital record	761 operations	4·5% for hydatid disease
Northeast Turkana[27]	Anti-echinococcus antibody ELISA	538	Positive: 16·4%. Strongly positive: 4·1%
Turkana (northwest)[9]	Ultrasound survey	3553	5·6%
Turkana (northeast)[9]	Ultrasound survey	3462	2·7%
Turkana (central)[9]	Ultrasound survey	1508	0·3%
Turkana (south)[9]	Ultrasound survey	1361	0·2%
Turkana (eastern)[9]	Ultrasound survey	607	0%
Pokot people[9]	Ultrasound survey	2389	0·1%
Gabbra people[9]	Ultrasound survey	38	0%
Somali people[9]	Ultrasound survey	1252	0%
Samburu people[9]	Ultrasound survey	368	0%
Rendille people[9]	Ultrasound survey	710	0%
Nigeria			
Sudan zone	Retrospective analysis of hospital records	189 861	0·0005%
Northern Guinea	Retrospective analysis of hospital records	279 827	0%
Bauchi plateau[28]	Retrospective analysis of hospital records	151 007	0%
Nigerstate, Ogunstate[29]	Serological survey, hydatid complement fixation test	176	0·53%
Sudan			
Central region[30]	Ultrasound survey	300	0·33%
Toposa people[9]	Ultrasound survey	278	3·1%
Tanzania			
Maasai people[9]	Ultrasound survey	959	1·1%
Ngorongoro district[31]	Retrospective analysis of hospital records	Unknown	10/100 000 per year; 171 cases
Uganda			
National[32]	Review of all biopsy and autopsy records from 1967–72	Unknown	23 cases

***Table:* Surveys of human cystic echinococcosis in sub-Saharan Africa**

of the country showed the presence of antibodies against echinococcal antigen in 0·53% of 176 individuals,[29] but corresponding clinical data for disease were not available. A review[28] of hospital records from three regions in Nigeria identified only one confirmed case of cystic echinococcosis in more than 500 000 records. However, the investigators questioned the accuracy of this finding because four more cases were identified in one region in northern Nigeria during the study period. They argued that low prevalences were the result of poor diagnostic facilities and inefficient record-keeping, rather than representing true occurrences.[28] The highest prevalences of cystic echinococcosis in livestock were reported from the Niger Delta and the north of the country. In the Niger Delta, pigs were the most commonly affected species (55·9% of 320 pigs infected, 21·5% of cysts were fertile), whereas in the north, cystic echinococcosis was mainly recorded in camels (55·5% of 3598 camels infected, 94·5% fertile cysts).[38,39] By contrast, in central and eastern parts of the country, cystic echinococcosis was rare, with the exception of the northern central parts, where 11% of 1800 sheep were infected with *E granulosus*.[39] In Ibadan only seven cysts were indentified in 164 sheep examined and none in goats.[40] Infection with *E granulosus* was also common among dogs in the Niger Delta (63%) and Ibadan.[38,40] In Ibadan, dogs from native (12 of 17) and residential neighbourhoods (11 of 18) were most commonly affected.[40] By contrast, in the northern, central, and eastern parts of Nigeria prevalences of *Echinococcus* spp in dogs were low.[41]

Surprisingly, most human cases of cystic echinococcosis in Nigeria are reported from the northern parts of the country.[28,42] One potential explanation for this finding is the difference in lifestyle between different regions of Nigeria; raw offal is commonly offered to dogs in the north and south of the country, whereas in the east it is regarded as a delicacy and not offered to dogs at all.[43,44] The differences in infection rates and fertility of cysts suggest that different taxa of *Echinococcus* spp are present in Nigeria, with varying infectivity to people, explaining the relatively low infection rates in people despite very high infection rates in livestock.

East Africa

Cystic echinococcosis has been investigated most extensively in east Africa, particularly in the Turkana and Maasai regions in Kenya (figure 3) and bordering districts in Uganda, Sudan, and Ethiopia.

An ultrasound survey in the extreme southeast of Sudan, near the Turkana border, identified an area of high prevalence of human cystic echinococcosis. The prevalence of this disorder was reported to be 2% among the Bouya people and 3·5% among the Toposa.[45,46] In the rest of the country human cases seemed to be more sporadic.[30,47] Where the disorder does occur in Sudan, pulmonary presentations are common, accounting for 17 of 38 cases in one series from Khartoum.[48] In animals, cystic echinococcosis was first reported in dogs in 1962 when the prevalence of *E granulosus* was 86·4%.[49]

Figure 3: **Typical pastoral landscape in Kenya's Maasai region where cystic echinococcosis is highly endemic**

Data for cystic echinococcosis in livestock are available from central, western, and southern Sudan. From central Sudan, prevalences from 20% (cattle) to 55·6% (camels) were reported.[50] In western Sudan, prevalences were highest among camels (61·4% of 565, 74% of cysts were fertile) and sheep (11·9% of 9272, 19% fertile cysts).[50] Although the prevalence in cattle was lower (5·2% of 4318), the prevalence of fertile cysts was high (75%). 1·9% of 5523 goats in this area were infected with *Echinococcus* spp (33% of cysts were fertile).[50] In southern Sudan, prevalences in cattle (7·1% of 325), sheep (2·7% of 295), and goats (7·1% of 42) were substantially lower than in the west.[50] The camel strain G6 predominates in Sudan. This strain is less infective to people than are others, which might explain the rather sporadic occurrence of human cystic echinococcosis in most parts of Sudan.[50] Although no data are available in the English scientific literature for strains causing disease in the extreme southeast of Sudan, some investigators have suggested that the sheep strain could be prevalent there, causing the increased prevalence of human hydatid disease. Dogs have been examined only in Tambool in central Sudan, where 25 of 49 dogs were deemed to be heavily infected.[51]

In Ethiopia, before the introduction of ultrasonography and modern serological tests as routine diagnostic instruments, Fuller and Fuller[22] showed that the Dassanetch and Nyangatom people from the southwest of the country had a prevalence of cystic echinococcosis of up to 5% on the basis of findings of clinical examination, and more than 5% when the hydatid skin test was taken into consideration.[22] The Dassanetch and Nyangatom peoples live in the same geographic area as the Turkana people of northwest Kenya, and these populations seem to share customs because they all use dogs for cleaning purposes. By contrast, results of an ultrasound survey of the Hamar people of southwestern Ethiopia showed a much lower prevalence (0·7% of 990 people) than for the Dassanetch and Nyangatom peoples.[23] Macpherson and colleagues[9] did ultrasound surveys of various ethnic groups in southern Ethiopia and recorded the highest prevalence in the Nyangatom people (2·9% of 1334).[9] Case series have been reported from central Ethiopia.[52–54] Between 72 and 234 patients were seen over 10–15 years at hospitals in Addis Ababa. By contrast with other countries, researchers did not identify a female predominance in these case series and cases of cystic echinococcosis in lung and liver seemed to be much the same, at about 40%.

Regarding livestock, several researchers have investigated cystic echinococcosis in cattle in several parts of Ethiopia, finding regional differences in prevalence and fertility of cysts. The highest prevalences were recorded in central Ethiopia with up to 52·7% of 632 cattle being infected with *Echinococcus* spp (26·9% of cysts were fertile).[55] The highest prevalence of fertile cysts was recorded in eastern Ethiopia, where 32% of cysts were fertile.[56] The lowest prevalences were recorded in southern parts of central Ethiopia, where 16% of 400 cattle were infected (1·8% fertile cysts).[57]

Kebede and colleagues[58] estimated the financial loss associated with cystic echinococcosis to be US$21 per infected cow. They suggested that the actual loss was even greater because home slaughtering practices were common.[58] In Hawassa, an annual loss of about $138 583 was estimated to be attributable to cystic echinococcosis.[55] Kebede and colleagues[58] argued that in northern Ethiopia, sheep might be the main intermediate host for cystic echinococcosis because they recorded that 10·6% of 380 sheep were infected, with 56·6% of cysts being fertile. By contrast with these findings, Bekele and colleagues[49] did not deem sheep to be the main intermediate host in central Ethiopia, where 16·4% of 560 tested positive but only 18·3% of cysts were fertile.[59] In goats, low prevalence was recorded in central Ethiopia (6·7% of 208),[60] whereas Sissay and colleagues noted that 65% of 632 goats examined in eastern Ethiopia were infected.[61] Kebede and colleagues[58] also investigated dogs for infection with *Echinococcus* spp in northern Ethiopia where 3 of 18 of eight dogs were infected. In this area, few human cases of cystic echinococcosis were indentified. In eastern Ethiopia, Mersie and colleagues[56] showed that two of nine dogs were infected with *Echinococcus* spp.

In central Ethiopia, mainly *E granulosus* G1 has been identified in livestock, whereas in northern Ethiopia (the city of Makale) *E granulosus* G1, *E ortleppi*, and *E canadensis* G6 and G7 were identified in 21 cysts from cattle.[7,62] In a study from central and eastern Ethiopia, *E granulosus* G1 predominated, but *E canadensis* G6 was also identified, mainly in camels.[63]

In Kenya, cystic echinococcosis occurs in most parts of the country but available data are mostly from Turkana communities in the northwest and from Maasai communities in the south. Both communities are nomadic pastoralists rearing huge herds of livestock (sheep and goats, cattle, donkeys, and in the Turkana also camels). In one serological survey,[27] prevalence of cystic echinococcosis was as high as 16·4% in recently settled communities in the Turkana area. Results of another serological survey showed regional difference within the Turkana district, with prevalence being 9·4% in north Turkana and 2·1% in south Turkana, which was much the same as in a control group from other parts of Kenya where hardly any cases of cystic echinococcosis were identified.[25] Ultrasonography is the most commonly used and most reliable diagnostic technique for surveys nowadays. In such surveys, the prevalence of cystic echinococcosis in the Turkana district was 5·6%.[9] Irvin[26] reported that 4·5% of 791 surgical procedures in one hospital were for cystic echinococcosis.[26] In clinical cases, a predominance of women has been noted, with women of child-bearing age having the highest prevalence.[20,24,64,65] This female predominance was not present in serological surveys.[25,27] Hydatid cysts can occur in all parts of the body; however, in all clinical surveys, hydatid cysts of the liver were most common, followed by abdominal cysts, kidney, spleen, lung, and soft tissue. Because most rural hospitals do not have radiograph facilities, lung disease is likely to be underdiagnosed in these populations.[20,26,65]

A domestic lifecycle of *Echinococcus* spp with dogs as the definitive host and small ruminants, cattle, and camels as intermediate hosts was thought to be most important in the Turkana district. Although jackals were also identified as definitive hosts for *Echinococcus* spp, they are unlikely to contribute substantially to the maintenance of the parasites' lifecycle because their access to offal is very limited.[66,67] An independent wildlife cycle has not been described in the Turkana area.

Several studies in livestock (ultrasound surveys and abattoir surveys) have been done in Turkana. Prevalences of cystic echinococcosis varied significantly within Turkana, but generally camels and cattle showed the highest prevalences (cattle 19%, camels 61%) with much variability in fertility rates of cysts.[68] Prevalence of infection in dogs in Turkana was reported to be as high as 60%, but great regional differences were identified, with the highest prevalence in the northwest and the lowest in the south and east of Turkana.[62,66,69] Researchers have identified risk factors for *Echinococcus* spp infestations as: age of dog of less than 5 years, free roaming of dogs, access to raw offal (giving a 12 fold increase in likelihood of infection), frequency of home slaughter, and species of animal slaughtered.[69] Human infections in these communities were associated with the amount of time the dog spent in the home, when they were allowed to clean children and scavenge from bowls and skins.[70] By contrast with these findings from the Turkana district, much lower prevalences of cystic echinococcosis have been identified in the Maasai area of southern Kenya (0·5%, Zeyhle E, unpublished). Despite high infection rates in their livestock and dogs and a favourable climate for the survival of echinococcal eggs in the environment, infection in people was much lower than in Turkana (0·5% *vs* 2·5% in 2010, Zeyhle E, unpublished).[71,72] As in Turkana, sheep and goats seemed to be the most important intermediate hosts, but by contrast with the Turkana area an additional wildlife cycle probably exists.[66,73] Although Maasai lead a lifestyle that is much the same as that of the Turkana, they have more water available to them for daily living and they do not rely on dogs for cleaning purposes, therefore their dog–man contact is less close.[71] Many *Echinococcus* spp isolates from Kenya have been examined genetically, mainly belonging to *E granulosus* G1 (sheep, goats, cattle, camels, pigs, people, and dogs) and *E canadensis* G6 and G7 (camels, cattle, goats, people, and dogs), and only one to *E ortleppi* (pig). Most samples originated from the northwest of the country (Turkana).[74–77] In the Turkana district, the sheep strain is the predominant taxon in people, sheep, cattle, and goats, whereas the camel strain predominates in camels and partly in goats. Only two isolates of 176 hydatid cyst specimens isolated from people were identified as the camel strain (G6) whereas all remaining isolates belonged to the sheep strain (G1).[75,77]

From Uganda, only one study of cystic echinococcosis in people and cattle is available. Via the national pathology service 23 cases were identified retrospectively over a period of 6 years. A female predominance was noted. Most cases were imported from Sudan (n=12) and only ten cases occurred in Ugandan people; these people were exclusively from the northern and northeastern districts of the country bordering southern Sudan and northern Kenya (Turkana). In the district closest to Turkana (Karamoja), where five of the ten Ugandan cases originated, 20% of cattle were infected with *Echinococcus* spp. In the two other districts (Acholi and Lango) where human cases were reported, the prevalence in cattle was 1%. In another district (Teso) south of the districts from which human cases were reported, a prevalence of 10·5% in cattle was noted.[32] About two thirds of dogs in the Moroto district were infected with uncharacterised *E granulosus*.[78] Results of a survey in the Queen Elizabeth National Park in western Uganda showed that a high proportion of the resident lions were infected with *E felidis*. In warthogs from the same location, cysts of *E felidis* and *E granulosus* G1 have been identified.[76]

In the late 1980s in Tanzania, Macpherson and colleagues[71] undertook an epidemiological study of cystic echinococcosis, based on surgical records, in the Maasai people. It showed an annual morbidity of 11 cases per 100 000, with women and children being most commonly operated on. The liver was the most commonly affected organ (55% of 159 cases). With ultrasound examination, the prevalence of cystic echinococcosis was 1·1% in 959 people examined.[71] Another retrospective study[31] from 1990 to 2003 showed much the same incidence of cystic echinococcosis in the Ngorongoro district. Women and young people were most commonly affected by cystic echinococcosis.[31] Epidemiological data for human disease from other parts of the country are not available.

Dogs have also been investigated for echinococcosis in Maasailand. In a small series,[71] five of ten dogs were infected with *Echinococcus* spp. Ernest and colleagues[79] investigated livestock in the Ngorongoro district for the presence of cystic echinococcosis. In a prospective study, they showed that 63·8% of 105 sheep, 34·7% of 619 goats, and 48·7% of 357 cattle slaughtered at abattoirs or at home were infected with cystic echinococcosis. 61% of cysts were of pulmonary origin and 25·4% were fertile.[79] In the Arusha region 4·2% of cattle and 6·0% of sheep and goats (combined) were infected with *Echinococcus* spp in an abattoir survey.[80]

From Somalia only one case report of human cystic echinococcosis is available.[29] Additionally, one isolate from a camel has been genotyped and was allocated to the camel strain G6.[81]

Southern Africa

No epidemiological studies of human cystic echinococcosis have been done in southern Africa. However, 162 cases of cystic echinococcosis in South Africa have been described.[82–107] Most researchers focused on unusual presentations and complications of cystic echinococcosis, such as cysts of the CNS, the spine, heart, or orbital cavity, and these reports are therefore not representative of the epidemiology of this disorder in South Africa. All investigators involved in these reports believed cystic echinococcosis to be common, despite the absence of epidemiological studies. Kayser[105] reported seeing about 20 cases per year at one hospital in the Eastern Cape Province. These case reports provide little information about risk factors associated with human cystic echinococcosis, and therefore which animals are important hosts (definite and intermediate) in the lifecycle of *Echinococcus* spp in South Africa is unclear. Only two cases are reported from Zimbabwe, where cystic echinococcosis is believed to be rare.[108] The investigators noted that while most hydatid cysts of bovine origin were fertile, dogs were not easily infected with *Echinococcus* spp by material of bovine origin, suggesting that people were not at risk of contracting the disease from dogs.[108]

In Zimbabwe, at the examination of lungs of cattle at an abattoir, 0·6% of 2000 sets of lungs were infected with *Echinococcus* spp.[109] By contrast with European findings, most of the cysts were fertile (96·8%).[110] Some data for cystic echinococcosis in animals in South Africa are available. In 1965, Verster and colleagues[111] investigated the prevalence of cystic echinococcosis in livestock at abattoirs nationwide. Prevalences varied greatly between regions and species investigated. For cattle, prevalences ranged between 1·2% and 13·8%, with the highest in the Eastern Cape and the lowest in the Karoo. However, the investigators also noted that prevalences increased with age in cattle, and therefore differences could be attributable to differences in age of animals slaughtered rather than being true variations in prevalence. For sheep, the prevalence ranged from 0·8% in the Karoo to 2·2% in Mpumalanga. For goats, prevalence ranged from 0% in the Western Province to 3·2% in the Eastern Cape but the numbers of slaughtered animals were small. In cattle, infection of lungs predominated, whereas in sheep and goats the liver was the most commonly affected organ. The dog was regarded as the main definitive host, although infected black-backed jackals were identified in the Eastern Cape and Western Transvaal.[111] Only a survey of cystic echinococcosis in cattle is available from Swaziland,[112] where 10·8% of 5886 cattle from different locations had hydatid cysts in their lungs, and 0·3% had cysts in the liver. The highest prevalence was in cattle originating from a farm in the northeast of the country where wild animals and hyenas were abundant, and the investigators suggested that these animals could be hosts for *Echinococcus* spp. From Namibia only the identification of a cyst from a zebra as being *E ortleppi* (G5) is reported.[113]

The available data suggest that cystic echinococcosis is prevalent in southern Africa, but the epidemiology in people and animals remains to be investigated.

Echinococcus in wild animals

In addition to people and domestic animals, echinococcal worms and cysts have been identified in many species of wild mammals in sub-Saharan Africa.[76,114] *E felidis* has been identified in lions in South Africa and Uganda, and in a warthog in Uganda.[67,115] *Echinococcus* spp recovered from various species of wild carnivores and herbivores have so far not been further characterised.

Discussion

Despite cystic echinococcosis being described as endemic in sub-Saharan Africa,[10] studies have shown large regional differences in the prevalence of this disorder (figure 4) and for many countries (particularly in central Africa) no epidemiological data exist. From some countries only data for cystic echinococcosis in livestock are available, from others only a few case reports of human disease.

Comparison of epidemiological data is difficult because of an inevitable selection bias. In livestock surveys, mostly abattoirs have been surveyed, where only animals of a particular age are slaughtered. Such surveys might give a skewed measure of prevalence, because the frequency of cystic echinococcosis is closely correlated with host age.[116] Many studies have examined prevalence in dogs, but these reports must be treated with caution because risk of infection with the relatively short-lived worms might vary with season, depends on the age of the dogs, and can vary on small spatial scales. Thus most data only show active transmission in a particular area.[117] In surveys of human cases, incidence and prevalence will differ depending on whether the survey examines patients presenting to hospitals or individuals who volunteer for the survey at their own initiative. Patients who are symptomatic are more likely to consent than are those without symptoms.

Comparison of results from different regions is difficult because of variations in diagnostic instruments used, with several tests, such as the hydatid skin test, now regarded as obsolete because of poor sensitivity and specificity and because of varying case definitions. Data based on serological surveys might be affected by poor specificity and sensitivity of the test, because these factors vary according to the organ affected, and by cross-reactivity with other helminths that are highly prevalent in Africa. In clinical surveys, cases might have been missed or falsely presumed to have cystic echinococcosis because of limited diagnostic facilities available to the investigators (eg, no ultrasound facilities, no radiograph facilities, diagnosis relying on clinical examination and hydatid skin testing alone) and limitations of the technique used itself (eg, ultrasonography does not detect cases of pulmonary cystic echinococcosis). Therefore the exact pattern of cystic echinococcosis in sub-Saharan Africa remains to be further elucidated. Even in countries where research has been done, investigators believe that cystic echinococcosis is still underdiagnosed because of lack of knowledge, resources, and record-keeping. Additionally many countries are faced with epidemics of far greater magnitude (eg HIV, tuberculosis, and malaria) and they drain the already limited resources. Thus cystic echinococcosis is rightfully considered a neglected tropical disease. But even in countries such as Kenya, Sudan, Ethiopia, and Nigeria where extensive epidemiological research has been done, many questions are unanswered, and new questions arise from the information available at present (panel).

Researchers cannot make the assumption that high numbers of infections in livestock and dogs correspond to high numbers of people affected by cystic echinococcosis and vice versa. As we have discussed, the Maasai and Turkana peoples seem to have much the same exposure to echinococcal eggs, but the Maasai show much lower prevalences of clinical disease. This difference cannot be explained by sociobehavioural reasons alone. Further confusing the matter is evidence that the camel strain (*E canadensis* G6), which is thought to be less virulent to people than other strains, is highly prevalent in livestock in the Turkana area of Kenya, yet human infections are mainly attributable to *E granulosus* G1. Moreover, despite high prevalences of cystic echinococcosis with the G1 strain in Maasailand, the prevalence of human cystic echinococcosis is substantially lower than in the Turkana district. In Nigeria, goats and camels show very high rates of infection and

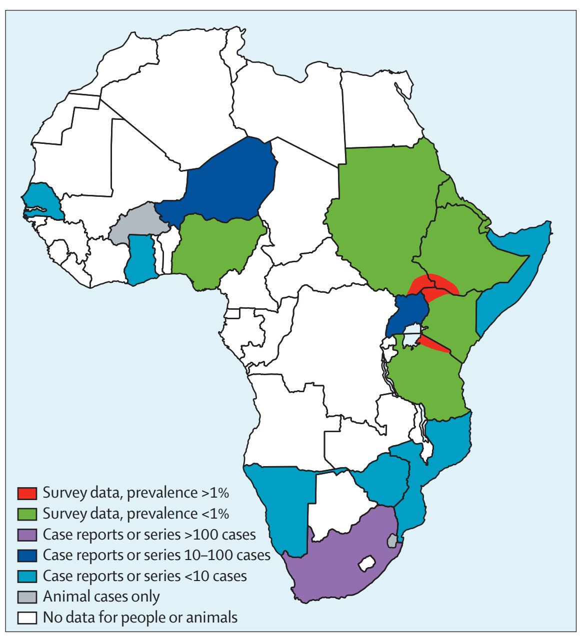

Figure 4: **Overview of the availability and nature of reported epidemiological data**
Prevalences and numbers refer to human cases unless otherwise specified.

Panel: Present knowledge and unanswered questions

Cystic echinococcosis is common in east Africa, especially in the Turkana region, but rare in west Africa
What is different in west Africa to make the disease rare? How important is the strain of *Echinococcosis granulosus* in this context?

Hepatic disease is the most common clinical presentation
Why is pulmonary disease the most common presentation in some areas, in people and livestock?

A female predominance is noted in clinical cases, but not in serological surveys
What effect does immunosuppression have on the clinical course of cystic echinococcosis?

Different strains of *E granulosus* are prevalent in Africa
What effect does the strain of *E granulosus* have on the clinical presentation and course of disease? Why are most people in the Turkana affected by the G1 strain, when most animals in the region are infected with the G6 strain? How is the lifecycle of the G1 strain sustained?

High prevalences in livestock do not correspond to high prevalences in people and vice versa
What factors influence the transmission of *Echinococcus* spp? What role do genetic factors of host and parasite play?

Cases of severe disseminated disease have been reported with co-infection with HIV or tuberculosis
What role do HIV and tuberculosis have in the context of cystic echinococcosis? How does co-infection affect the epidemiology and clinical course of disease?

yet human disease is believed to be uncommon. The explanation for this discrepancy is likely to be multifactorial, including sociobehavioural factors and the respective genotype of *Echinococcus* spp.

In most countries, a female predominance in clinical disease has been noted which is not present in serological surveys, thus giving further rise to the suspicion that immunological host factors and immune-suppression in particular have a role in the pathology of cystic echinococcosis, making development of clinical disease more likely after exposure. However, in Ethiopia no female predominance was identified in clinical surveys and by contrast with other countries, no predominance of hepatic disease was identified, with rates of pulmonary and hepatic disease being much the same. The suspicion that immune-suppression affects the clinical course of cystic echinococcosis might have implications for the future, particularly in view of the ongoing HIV and tuberculosis epidemics in Africa. In view of the long incubation period of this disorder and its unknown epidemiology, increasing prevalence could easily be overlooked in countries overwhelmed by HIV and tuberculosis. So far only very few case reports of co-infection exist, but some of them are concerning,[90] reporting severe disseminated disease. Further research is necessary to identify the different genotypes of *E granulosus* prevalent in Africa and the clinical signs and symptoms associated with them, in addition to further work investigating the host-parasite interaction.

Search strategy and selection criteria

We searched PubMed with the terms "cystic echinococcosis/Africa", "hydatid disease/echinococcosis/Africa", and "hydatid disease/echinococcosis" for each country of sub-Saharan Africa (as defined by the UN), for all available articles without time period restrictions up to May, 2012. We selected case reports, case series, epidemiological studies of human disease and disease in livestock, and studies of prevalence in animal hosts, published in English.

Contributors
MPG, KW, and CNM conceived the paper. KW wrote the first draft. All authors contributed to and approved the final version.

Conflicts of interest
We declare that we have no conflicts of interest.

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