



# Nematodiasis poco frecuentes

*en Colombia*

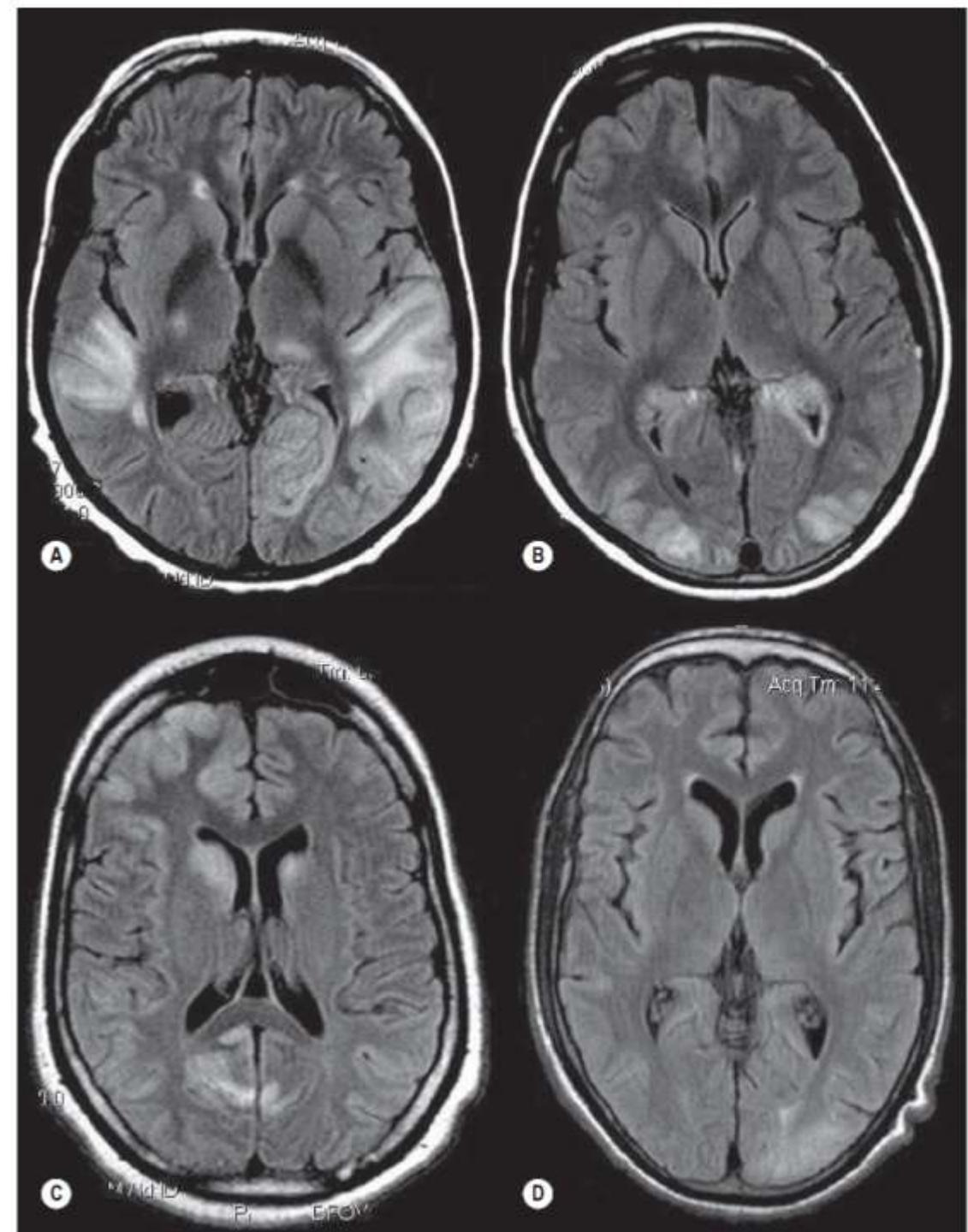
**Prof. Alfonso J. Rodríguez-Morales**

Parasitología Grupos 4 y 5

Semestre I-2015

# Nemátodos poco frecuentes (en Colombia)

- Angiostrongiliasis
- Anisakiasis
- Baylisascariasis
- Capilariasis
- Dracunculiasis
- Gnathostomiasis



**Figure 71.1** Axial T2 FLAIR images demonstrating the characteristic polioencephalopathy (grey matter) in subacute measles encephalitis. (A) Bilateral temporal-parietal cortical hyperintensities; (B) parieto-occipital cortical hyperintensities; (C) superficial cortical (left frontal and bilateral occipital) and deep grey matter (bilateral head of caudate) hyperintense signal abnormalities and (D) hyperintense signal changes in the right occipital cortex. (From Albertyn C, van der Plas H, Hardie D, et al. Silent casualties from the measles outbreak in South Africa. *S Afr Med J* 2011;101:313-4, 6-7<sup>2</sup>)

Table 1. Clinical Events and Laboratory Results in a 22-Year-Old Male with *Angiostrongylus* Eosinophilic Meningitis.

dpi <sup>a</sup>	dpos <sup>b</sup>	Event/Status	Treatment	Blood: wbc × 10 <sup>3</sup> /µl / eosinophils	Blood: absolute eo- sinophil count (cells/µl)	CSF: wbc/µl / eosinophils	PCR for <i>A.</i> <i>cantonesis</i>
0		Approximate time of snail ingestion					
9	0	Myalgias begin					
13	4	Seen in outpatient clinic	Analgesics	17.5/12%	2100		
15	6	First hospital admission	Prednisolone 60mg/d	11.7/14%	1670	338/15%	Negative
17	8	Lumbar puncture; MRI		13.2			
18	9	First discharge	Prednisone 20mg/d	16.6/12%			
20	11	Treated in ED		21.9/9%			
23	14	Second hospital admission	Prednisone 80mg/d; begin albendazole	18.0/5%	860	1248/27%	
24	15	Abducens palsy					Positive
26 <sup>c</sup>	17	Mental status worse	Prednisone 80mg/d + albendazole continue; therapeutic lumbar puncture				
28	19	Staff learns of snail ingestion					
29	20	MR angiogram					
30	21	Ataxia, paresthesias					
32	23	First signs of clinical improvement					
34	25	Second discharge					
37	28	First outpatient follow up visit	Finish albendazole; begin prednisone taper				
44	35	Headache worse	Prednisone 80mg/d				
57	48	PCR data received	Tapering prednisone more slowly				
85	76	Diplopia resolved					
99	90	Mild abducens and limb weakness persists	Off prednisone				

<sup>a</sup>days post ingestion of snail. <sup>b</sup>days post onset of symptoms. <sup>c</sup>no further laboratory data available*Achatina fulica*

Hawaii J Med Public Health. 2013 Jun;72(6 Suppl 2):41-5.  
 A severe case of *Angiostrongylus* eosinophilic meningitis with encephalitis and neurologic sequelae in Hawai'i.  
 Kwon E, Ferguson TM, Park SY, Manuzak A, Qvarnstrom Y, Morgan S, Ciminera P, Murphy GS.

# Angioestrongiliasis

- El nematode *Angiostrongylus cantonensis*, gusano del pulmón de la rata, es la causa más común de **meningitis eosinofílica**.
- *Angiostrongylus (Parastongylus) costaricensis* es el agente causal de la angioestrongiliasis abdominal o intestinal.

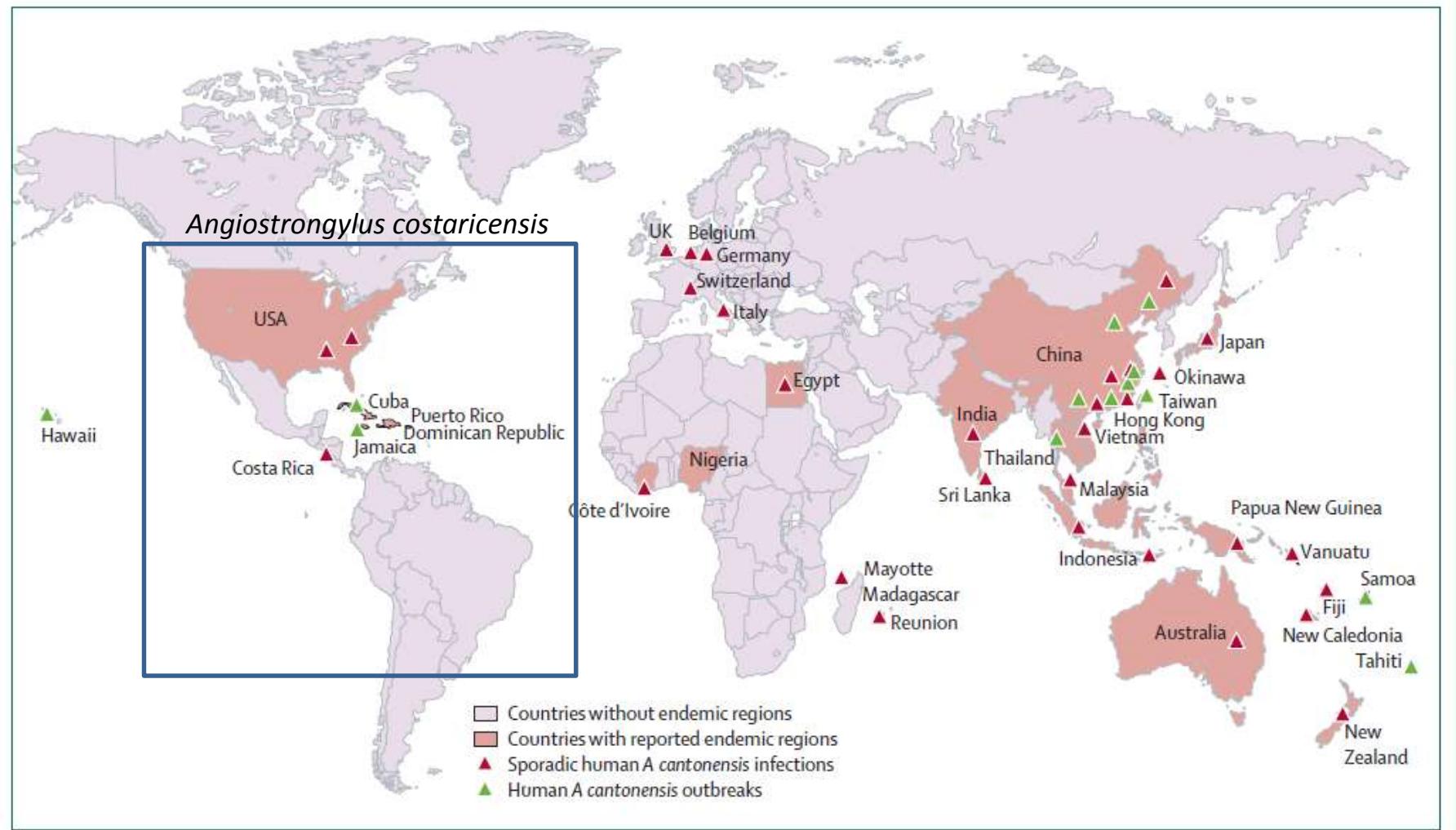


Figure 3: Distribution of *A cantonensis* and human *A cantonensis* infections or outbreaks worldwide

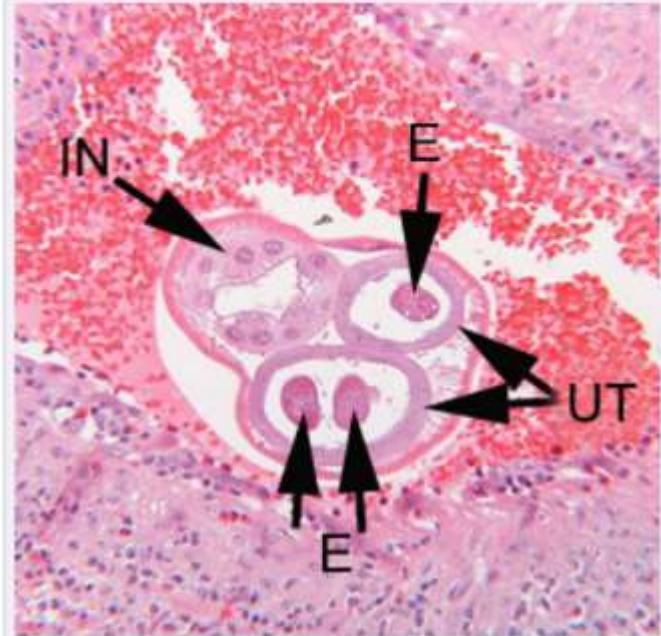
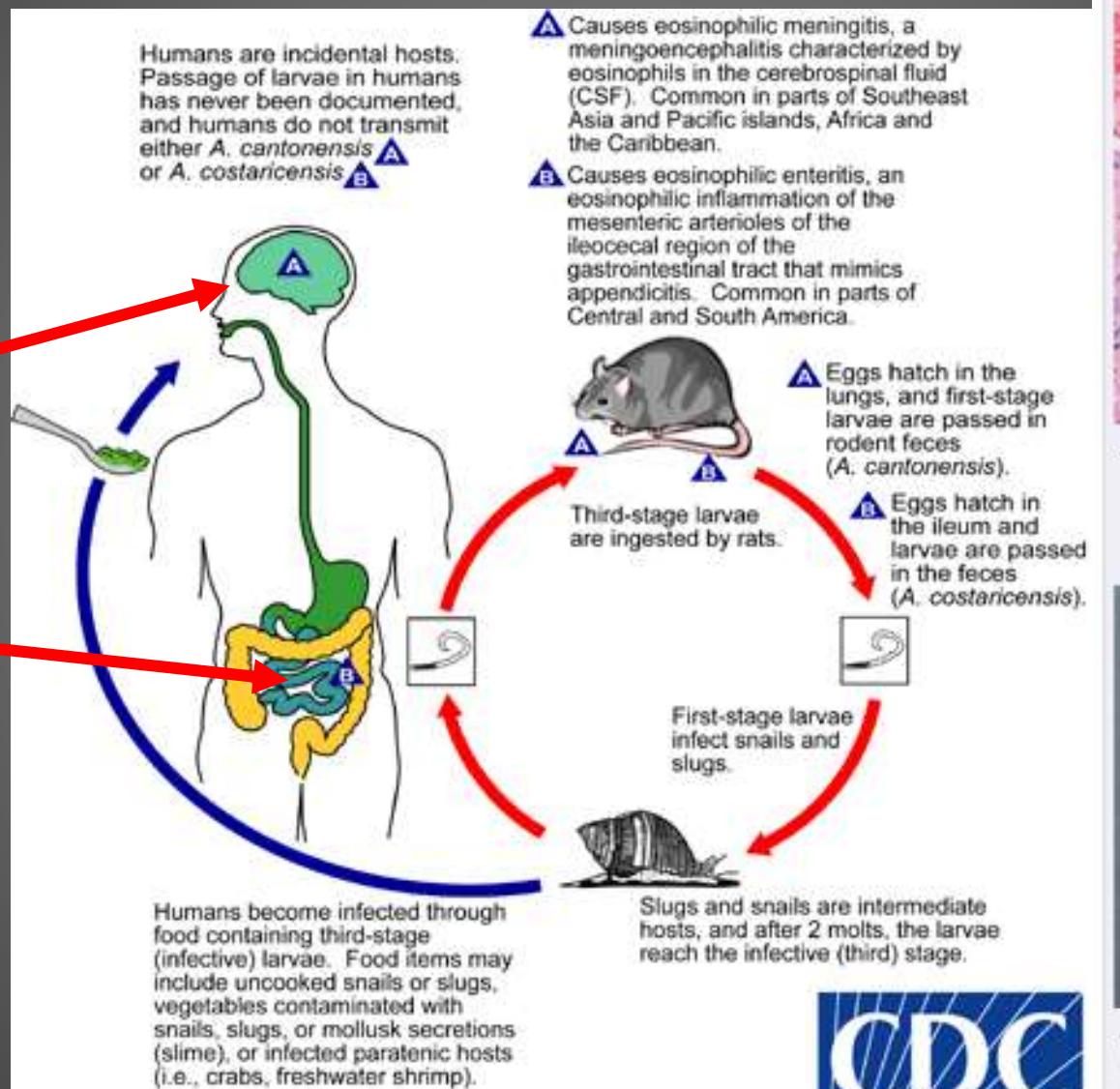
Endemic regions are those countries in which human cases of angiostrongyliasis or animal reservoirs of *A cantonensis* have been identified. This is not intended to imply that *A cantonensis* is endemic throughout these entire countries. Conversely, those areas marked as non-endemic regions may well be countries where no investigation has taken place and so cannot be considered free from *A cantonensis* infection.

	Cases (%)
Thailand <sup>17,24,25,26,27-28</sup>	1337 (47.33)
China (including Taiwan and Hong Kong) <sup>19,26-28,30,31,32,33,34,35,36,37,38,39</sup>	769 (27.22)
Tahiti, French Polynesia <sup>1,9</sup>	256 (9.06)
USA <sup>1,6,8,11,34,109-121</sup>	116 (4.11)
Cuba <sup>42,103-104</sup>	114 (4.04)
New Caledonia <sup>100,105</sup>	72 (2.55)
Japan <sup>5,7,26-27,31</sup>	63 (2.23)
Australia <sup>30,113-116</sup>	24 (0.85)
Vanuatu <sup>13</sup>	19 (0.67)
India <sup>37,101,102,112,113</sup>	10 (0.35)
Vietnam <sup>39,100,109</sup>	8 (0.28)
Malaysia <sup>100</sup>	6 (0.21)
Mayotte <sup>100,120</sup>	6 (0.21)
Réunion island, France <sup>100</sup>	4 (0.14)
Egypt <sup>121</sup>	3 (0.11)
Sri Lanka <sup>123-124</sup>	3 (0.11)
Cambodia <sup>100</sup>	2 (0.07)
Samoa <sup>100</sup>	2 (0.07)
Fiji <sup>125</sup>	2 (0.07)
Belgium <sup>3</sup>	1 (0.04)
Costa Rica <sup>126</sup>	1 (0.04)
Germany <sup>127</sup>	1 (0.04)
Indonesia <sup>12</sup>	1 (0.04)
Jamaica <sup>128</sup>	1 (0.04)
Italy <sup>5</sup>	1 (0.04)
Côte d'Ivoire <sup>100</sup>	1 (0.04)
New Zealand <sup>129</sup>	1 (0.04)
Papua New Guinea <sup>100</sup>	1 (0.04)
Switzerland <sup>1</sup>	1 (0.04)
UK <sup>18</sup>	1 (0.04)
Total	2827

Table 2: Cases of human angiostrongyliasis reported in countries or regions

# Angiostrongiliasis

*Angiostrongylus cantonensis*  
*Angiostrongylus costaricensis*



*Angiostrongylus costaricensis* female worm in appendix tissue sections stained with hematoxylin and eosin. Notice the thick, multinucleate intestine (IN) and eggs (EG) within the uterus (UT).

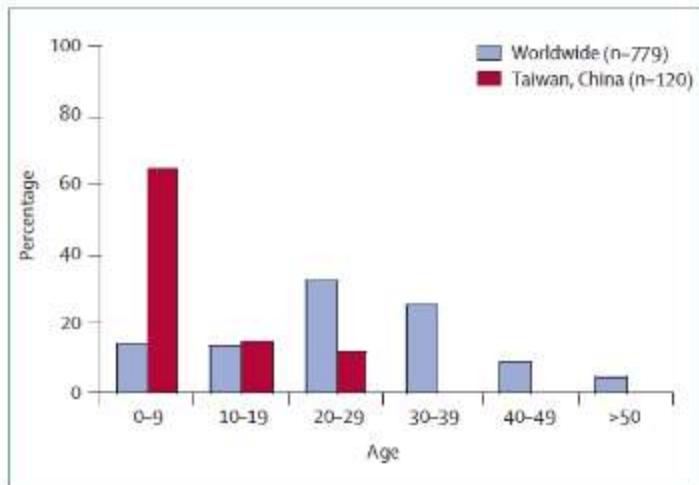


*Angiostrongylus cantonensis* third stage (L3), infective larva recovered from a slug. Image captured under differential interference contrast (DIC) microscopy.

**TABLE  
71.3**

## Causes of Seizures/Epilepsy

### Infections



**Figure 4:** Ages of patients who have acquired angiostrongyliasis  
Data are summarised from references 3, 11, 19, 28, 29, 36, and 111. Although 125 cases of *A cantonensis* were reported in Taiwan, the exact ages of five patients were not recorded, and are therefore not shown in this figure.

### Focal brain lesions

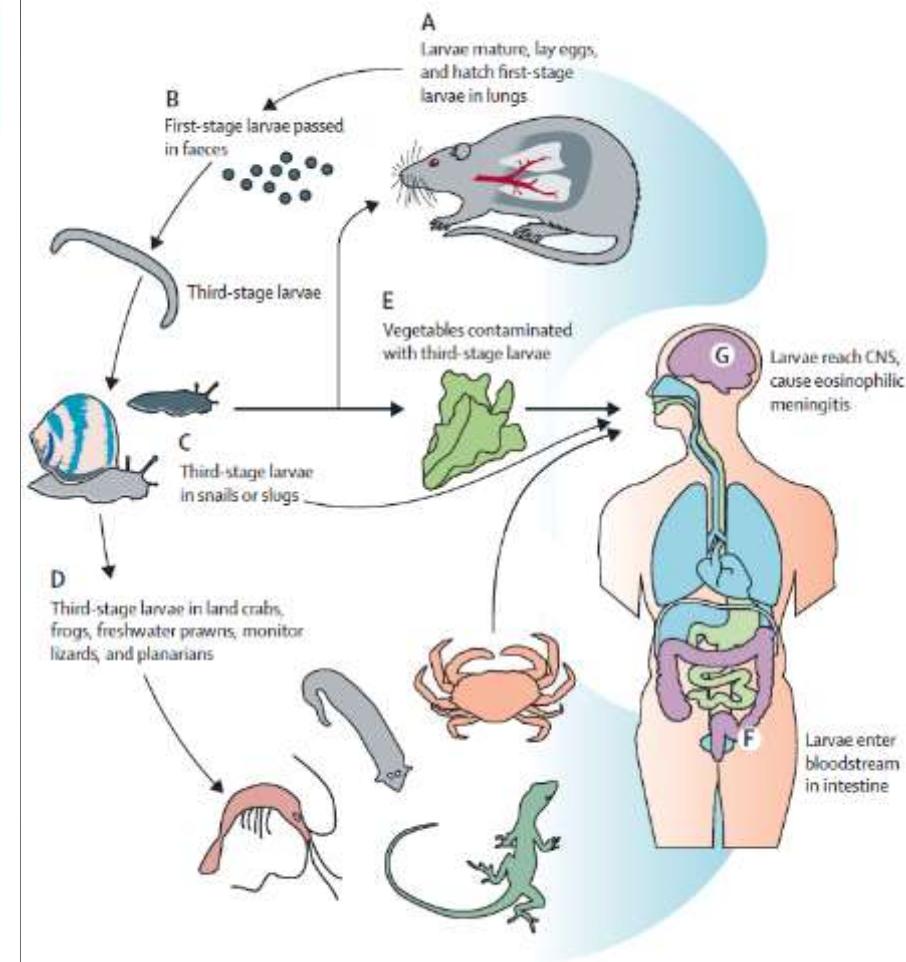
### Toxins/drugs/metabolic

### Metabolic Stroke

Bacterial meningitis  
Cerebral malaria  
TB meningitis  
Cryptococcal meningitis  
Neurocysticercosis  
Schistosomiasis  
Cerebral hydatid disease  
Paragonimiasis  
Cerebral toxoplasmosis  
Cerebral amoebiasis

**Neuroangiostrongyliasis  
Gnathostomiasis  
Baylisascariasis**

Tetanus (pseudoepilepsy)  
Tumours, cysts, granulomas,  
tuberculoma  
Alcohol, opiates, altered glucose  
levels  
Hypoglycaemia  
Haemorrhage, intracerebral/  
subarachnoid



Lancet Infect Dis 2008; 8: 621–30

**TABLE  
71.4** **Helminths that May Infect the Central Nervous System**

Organism	Main Areas of Distribution	Presentation	Management
<b>CESTODES</b>			
Neurocysticercosis	Latin America, sub-Saharan Africa, India	Seizures, mass lesion, hydrocephalus	Albendazole 15 mg/kg/day in 2 divided doses for 8 days. Cover with corticosteroids (see text)
<i>E granulosus</i> (Hydatid disease)	Worldwide	Mass lesion, seizures	Surgical resection. Pretreat: albendazole 15 mg/kg/day in 2 divided doses (40 days) to shrink cyst. If unresectable: albendazole 10–15 mg/kg/day in two divided doses twice a day ≥ 3 months
<i>Taenia multiceps</i> (coenurosis)	Worldwide	Hydrocephalus	Surgical removal
<i>Spirometra</i> sp. (Sparganosis)	Far East and S-e Asia, East Africa	Seizures, infarcts	Surgical removal
<b>NEMATODES</b>			
<i>Angiostrongylus cantonensis</i>	S-e Asia, Caribbean, Southern USA	Meningoencephalitis	Repeated LPs to reduce ICP. Corticosteroids + Albendazole 10–15 mg/kg/day in two divided doses – 2 weeks
<i>Gnathostoma spinigerum</i>	S-e Asia, Mexico, Ecuador, Japan	Meningoencephalitis, seizures, myeloradiculopathy	Albendazole 10–15 mg/kg/day in 2 divided doses – 21 days + corticosteroids. Surgical removal if accessible
<i>Onchocerca volvulus</i>	West Africa, Yemen, Latin America	Chorioretinitis, keratitis, seizures	Ivermectin single oral doses of 0.15 mg/kg single oral dose
<i>Baylisascaris procyonis</i>	Worldwide	Meningoencephalitis	Corticosteroids, albendazole 10–15 mg/kg/day in 2 divided doses
<i>Trichinella</i> sp.	Worldwide	Myopathy, strokes, meningoencephalitis	Corticosteroids, repeated LPs
<b>TREMATODES</b>			
Schistosomiasis	Africa, Asia, Brazil	cauda equina/conus syndrome/ cerebral granuloma	<i>S. mansoni</i> , <i>S. haematobium</i> and <i>S. intercalatum</i> – Praziquantel 40 mg/kg/day – 3 days, corticosteroids (see text) <i>S. japonicum</i> and <i>S. mekongi</i> – Praziquantel 60 mg/kg/day – 3 days, corticosteroids (see text)
<i>Paragonimus</i> sp.	Latin America, Asia, West Africa	Encephalitis, mass lesion, infarcts, seizures, myelopathy	Praziquantel 25 mg/kg three times a day – 3 days, corticosteroid
<i>Fasciola hepatica</i>	Worldwide	Meningitis, mass lesion, infarct	Triclabendazole 10 mg/kg single dose or 20 mg/kg in two divided doses

S-e, South-east; LP, lumbar puncture; ICP, intracranial pressure; meningoencephalitis in this table refers to an eosinophilic meningoencephalitis.

**Panel: Differential diagnosis of eosinophilic meningitis<sup>41</sup>**

**Infectious causes**

- *Angiostrongylus cantonensis*
- *Gnathostoma spinigerum*
- *Paragonimus* spp
- *Strongyloides stercoralis*
- *Toxocara canis*
- *Loa Loa*
- *Toxoplasma gondii*
- *Taenia solium*
- *Coccidioides immitis*
- *Schistosoma japonicum*
- *Fasciola hepatica*
- *Trichinella spiralis*

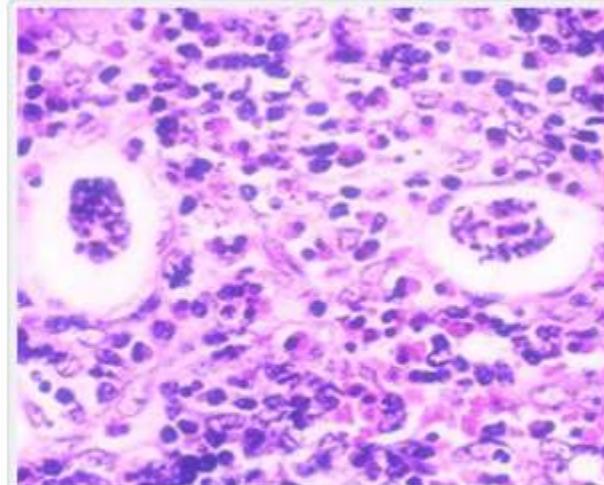
**Non-infectious causes**

- Malignant tumours
  - Glioblastoma
  - Hodgkin's disease
  - Non-Hodgkin lymphoma
  - Acute leukaemia
  - Meningeal carcinomatosis
- Drugs
  - Post myelography
  - Ibuprofen
  - Ciprofloxacin
  - Vancomycin
  - Gentamicin
- Foreign bodies
  - Ventriculoperitoneal shunts
- Primary eosinophilic meningitis

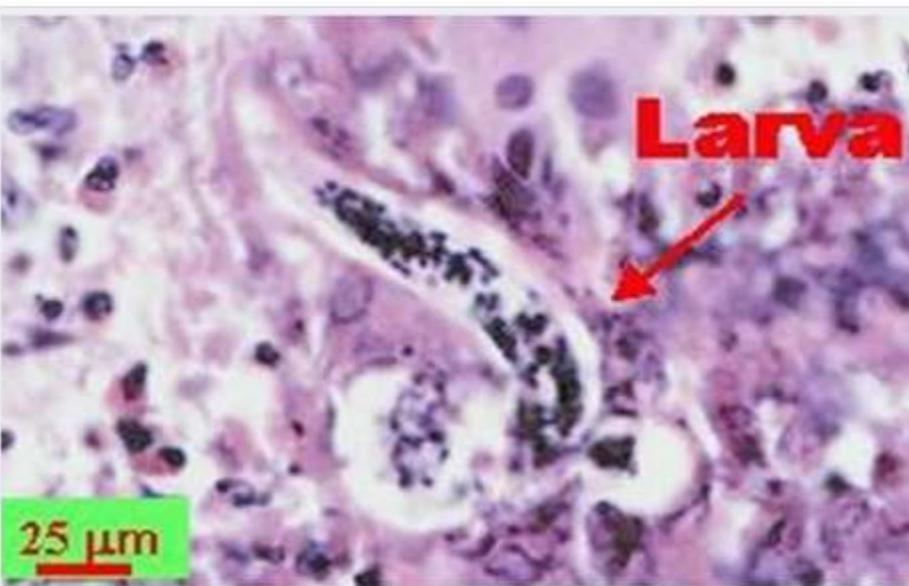
▼ *Angiostrongylus costaricensis* eggs and larvae in tissue stained with hematoxylin and eosin (H&E).



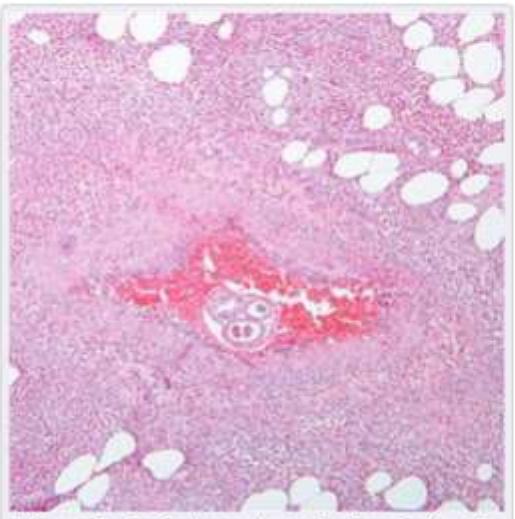
**Figure A:** *Angiostrongylus costaricensis* eggs in intestinal tissue stained with hematoxylin and eosin (H&E).



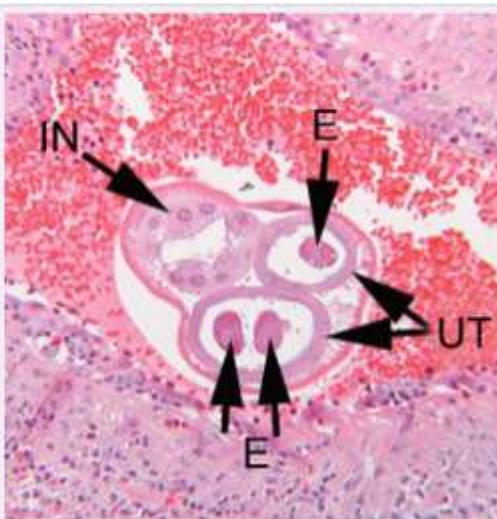
**Figure B:** Thin-shelled *A. costaricensis* eggs in intestinal tissue stained with H&E, a feature consistent with the presence of mature female worms.



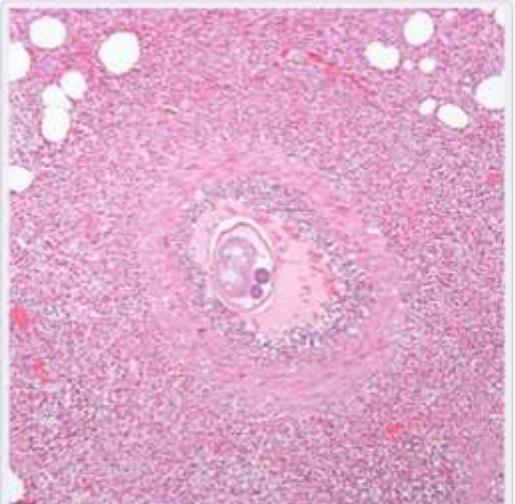
**Figure C:** *A. costaricensis* first stage (L1) larva in intestinal tissue stained with H&E

\* *Angiostrongylus costaricensis* adult female in tissue sections stained with H&E.

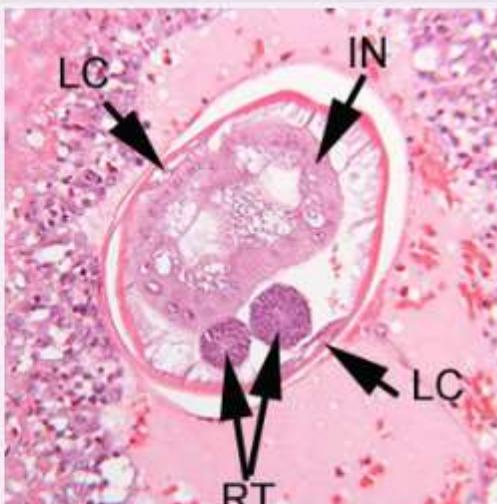
**Figure A:** *Angiostrongylus costaricensis* female worm in appendix tissue sections stained with hematoxylin and eosin (H&E). Image courtesy of Regions Hospital, St. Paul, MN.



**Figure B:** Higher magnification of the specimen in Figure A. Notice the thick, multinucleate intestine (IN) and eggs (EG) within the uterus (UT).



**Figure C:** Another image from the specimen seen in Figure A.



**Figure D:** Higher magnification of the specimen in Figure C. Shown here are the thick, multinucleate intestine (IN), reproductive tubes (RT), and lateral chords (LC).

Diagnóstico  
Clínico-epidemiológico  
Inmunológico  
Molecular

▼ ***A. cantonensis* larvae recovered from slugs.**

Third stage (L3), infective larvae would not be found in human tissue; the images below are not clinically diagnostic. L3 larvae are infective to humans, who serve as incidental hosts.



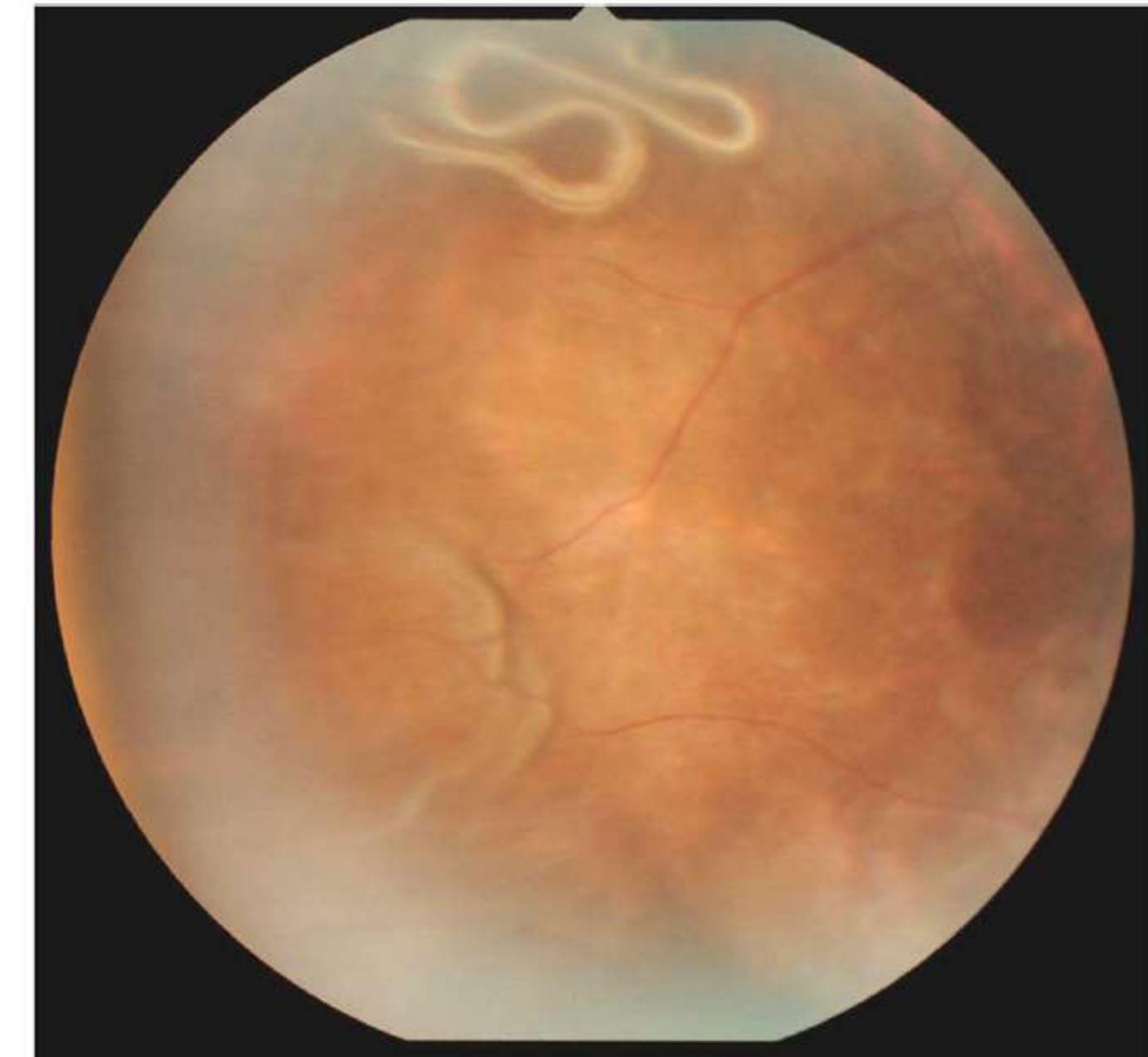
**Figure A:** *Angiostrongylus cantonensis* third stage (L3), infective larva recovered from a slug.  
Image captured under differential interference contrast (DIC) microscopy.



**Figure B:** *A. cantonensis* (L3), infective larvae recovered from a slug.  
Image captured under DIC microscopy.



**Figure C:** Higher magnification of Image B. Note the terminal projection on the tip of the tail which is characteristic of *A. cantonensis*.



**Figure 1** The subretinal parasite and subretinal tract at the superotemporal retina.

Clin Ophthalmol. 2013;7:977-9. doi:  
10.2147/OPTH.S43565. Epub 2013  
May 27.  
Subretinal angiostrongyliasis-induced  
optic neuritis.  
Sinawat S, Yospaiboon Y, Sinawat S.

Table 1. Comprehensive review of the clinical features of ocular angiostrongyliasis cases in the world.

		Age	Sex	Affected side	VA Rt	VA Lt	duration	Worm in	Size of worm	Ocular disease	Meningo- eosinophilia	treatment	outcome	Year	Reference	
1	Thai	34	M	R	LP	20/20	6 weeks	AC	13 mm		N	N	survival removal	count finger; slightly improved	1962	4
2	Thai	22	M	L	6/6	LP	2 months	AC	18.86 mm		N	N	survival removal	not improved	1966	5
3	Thai	21	M	L	6/6	2/60	1 day	AC	8.55 mm	chronic retinitis and vitreous opacity	N	N	paracentesis	Improved, 6/24	1985	6
4	Thai	34	M	L	6/6	CF	10 days	VF	11.7 mm	panophthalmitis	Y	Y	survival removal	not improved	1971	7
5	Thai	36	M	L	20/20	CF 2 feet	9 days	retina	12.5 mm	retinal and macular edema	Y	NA	survival removal	not improved	1974	8
6	Thai	72	F	R	CF		1 month	VF	9.22 mm		N	N	survival removal	not improved	1986	9
7	Thai	28	M	R	CF 1 foot	20/20	14 days	VF	< 10 mm		N	N	aspirator and vitrectomy	Improved, 20/20	2003	10
8	Thai	44	M	L	NA	NA	NA	VF	12 mm + 1 dead worm		N	N	aspirator and vitrectomy	Improved, 6/200		10
9	Thai	21	M	L	6/9	2/60	14 days	VF	NA	optic neuritis	Y	Y	laser, oral steroid	not improved	2007	11
10	Thai	36	F	L	6/6	CF	NA	VF	9.8 mm	papilledema	Y	NA	survival removal	slightly improved		11
11	Thai	22	F	R	1/60	6/6	2 months	VF	10.9 mm		N	N	survival removal	NA		11
12	Thai	39	M	R	6/6	6/6	10 days	VF	NA		N	NA	laser	normal VA		11
13	Thai	33	F	L	6/6	1/60	7 days	subretinal	NA		N	N	laser, steroid	slightly improved, 5/60		11
14	Thai	28	M	R	6/24	6/6	4 days	subretinal	NA		N	NA	laser, oral steroid	not improved		11
15	Thai	48	M	R	CF	6/6	21 days	VF	11.4 mm		N	N	laser, surgical removal	not improved		11
16	Thai	47	M	L	6/6	CF 2 feet	21 days	AC	ND	optic neuritis	N	NA	laser; aspirator IV methyl-prednisolone	Improved, 2/60	2008	12
17	Thai	27	M	L	6/6	1/60	21 days	VF	ND	optic neuritis??	Y	NA	laser, surgical removal	slightly improved, 6/60		12
18	Thai	36	M	R	2/60	6/6	7 days	subretinal	ND	optic neuritis??	N	NA	laser	slightly improved, 6/60		12
19	Thai	27	M	R	CF 1 foot		1 month	subretinal	15 mm	optic neuritis	N	N	albendazole, steroid laser	slightly improved 2/60	2013	13
20	Sri Lanka	30	M	R	No LP	NA	1 month	AC	11.6 mm		N	NA	forceps extraction	NA	1993	14
21	Sri Lanka	30	M	R	6/60	6/6	2 days	subretina to VF	6.3 mm	retinal edema	Y	Y	forceps extraction	not improved	1998	15
22	Sri Lanka	20	M	L	6/6	6/9	3 days	AC	6.5 mm		N	N	needle aspiration	improved	2001	16
23	Sri Lanka	M	R	LP	6/6	days	AC	11.4 mm			N	NA	survival removal	Improved, 6/60	2004	17
24	Sri Lanka	25	F	R	6/60	6/6	14 days	retina	8.5 mm		N	NA	survival removal	NA	2007	18
25	India	12	M	L	6/6	6/60	14 days	AC	28 mm	optic neuritis??	N	Y	survival removal	improved, 6/6	2006	19
26	India	40	F	L	NA	NA	NA	VF	13 mm		Y	Y	survival removal	NA	2008	20
27	Mainland China	34	M	L				retina	12 mm		N				1999	21
28	Mainland China	35	M	L				AC	12.3 mm	optic neuritis	N				2000	22
29	Mainland China							AC							2001	23
30	Mainland China	47	M	R	blurred	NA	NA	optic nerve		optic nerve compression	Y	NA	survival removal	not improved	2009	24
31	Taiwan	52	F	R/L	CF	LP	7 days	(-)	15 mm	bilateral necrotizing retinitis	Y	NA	IV methylprednisolone	not improved	2006	25
32	Taiwan	38	M	L	20/20	20/50	2 days	not identified	(-)	optic neuritis	Y	Y	mebendazole, IV methylprednisolone	slightly improved, 20/25	2006	26
33	Japan	62	F	L	20/25	2/200	1 day	VF	12 mm	optic neuritis??	N	Y	survival removal	not improved	2002	27
34	Japan	24	M	L	6/6	6/9 10/6/100	1 day	VF	5 mm		Y	Y	oral steroid, LP	Improved; normal VA 6/6	1988	28
35	Vietnam								12 mm		N				1974	29
36	Vietnam	3	F	R			3 days	AC	15 mm		Y		survival removal	VA 0.6	2002	30
37	Indonesia	23	F	L	5/5	3/60	14 days	AC	11.1 mm		N	N	paracentesis	not improved	1977	31
38	Papua New Guinea	45	F	R	6/36	6/6	3 months	VF	< 1 cm	acute ciliary injection with blepharospasm	N	Y	topical steroid, topical anti-biotics	not improved	1982	32
39	Malaysia	57	M	L	6/6	6/36	3 days	retina	ND		N	Y	survival removal	Improved, 6/24	2003	33
40	South Africa (UK)	33	M	R	6/9	6/6	2 days	AC	22 mm	anterior uveitis	N	NA	needle aspiration	Improved, normal VA 6/6	2005	34
41	Nepal		M	R	20/80	20/20		VF	15 mm	uveitis	N				2008	35
42	Jamaica	30	F	L	6/5	CF	1 month	AC	19.9 mm		N	N	survival removal	Improved, 6/36	2009	36

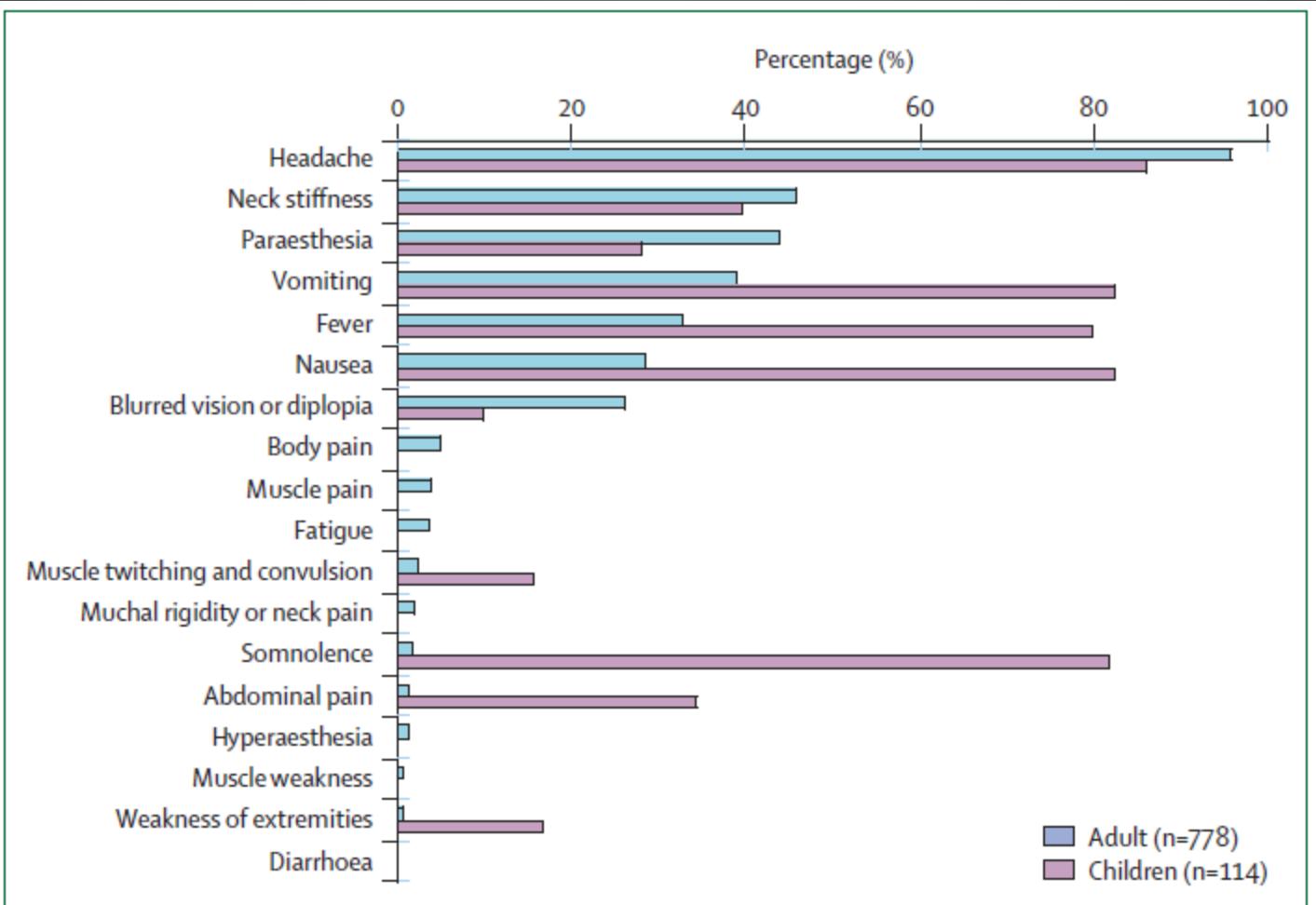
M: male, F: female, R: right, L: left, CF: count finger LP: light perception, AC: anterior chamber, VF: vitreous fluid, Y: Yes, N: No, ND: not determined, NA: not available

# Compromiso ocular

Table 1. Ocular involvement in parasitic infections<sup>28</sup>

Disease	Etiologic agent	Ocular findings	Main Diagnostic Methods
<i>Round worms (nematodes)</i>			
Toxocariasis	<i>Toxocara canis</i> and <i>T. cati</i>	Peripheral white mass is often visible in affected eyes	Serology
Angiostrongyliasis	<i>Angiostrongylus cantonensis</i>	Blurred vision and poor visual acuity	Identification of <i>Angiostrongylus cantonensis</i> in the eye. Direct and indirect immunofluorescent
Onchocerciasis	<i>Onchocerca volvulus</i>	Chorioretinitis, keratitis, uveitis, corneal opacification, neovascularisation, blindness	Slit lamp, sclerocorneal punch biopsy, Xenodiagnosis
Loiasis	<i>Loa loa</i>	Conjunctival congestion and pain with movement of the eye. May affect vision transiently. Retinal hemorrhages and perivascular inflammation	Extraction of adult worm or microfilaria
Trichinosis	<i>Trichinella spiralis</i>	Edema around the eye, conjunctivitis and exophthalmoses	Muscle biopsy
Bancroftian and Brugian filariasis	<i>Wuchereria bancrofti</i> , <i>B. malayi</i>	Retinal vasculitis, decreased vision and panuveitis with secondary glaucoma	An aqueous tap and a peripheral blood smear isolate microfilariae or adult worm
Thelaziasis	<i>Thelazia callipaeda</i>	Epiphora, conjunctivitis, keratitis, corneal opacity and ulcers	Eggs or larvae can be seen when tears or other eye secretions are examined under light microscope
Dirofilariasis	<i>Dirofilaria repens</i>	Pain, edema, and congestion of the conjunctiva, diplopia, foreign body sensation in the eye	Excision biopsy
Baylisascariasis	<i>Baylisascaris procyonis</i>	Vision loss, transient visual obscuration, and diffuse unilateral subacute neuroretinitis	Exclusion of other known causes of <i>ocular larva migrans</i>
<i>Flat worms (cestodes)</i>			
Cysticercosis	<i>Cysticercus cellulosae</i>	Subconjunctival and eyelid masses, papilloedema, cranial nerve palsies, vitritis and optic neuritis	Imaging with ultrasound, MRI and CT. Serology can be useful
Schistosomiasis	<i>Schistosoma mansoni</i> , <i>S. haematobium</i> , <i>S. japonicum</i>	Uveitis and subretinal granuloma	Eggs in the feces, urine or eggs/cercariae in the eye
Hydatid cyst	<i>Echinococcus granulosus</i>	Orbital swelling, exophthalmus and proptosis	Imaging
Fascioliasis	<i>Fasciola hepatica</i>	Painful red eye, and there may be visual defect	Adult worm in the eye
Protozoa			
Acanthamoebic keratitis	<i>Acanthamoeba spp</i>	Conjunctival edema, sever pain, ring infiltrate around the cornea, hypopyon, hyphema, uveitis, loss of vision	Corneal scrapings, culture
Chagas disease	<i>Trypanosoma cruzi</i>	Palpebral and periorbital oedema	Blood smear, Buffy coat, culture, Xenodiagnosis
Giardiasis	<i>Giardia intestinalis</i>	Salt and pepper retinal changes, chorioretinitis, retinal haemorrhage and uveitis	Diagnosing intestinal disease and exclusion
Leishmaniasis	<i>Leishmania spp</i>	Visceral: conjunctivitis, uveitis and retinal haemorrhage Cutaneous: lesions on eyelid, blepharoconjunctivitis Mucocutaneous: severe ulceration, loss of the eye	Tissue smears or biopsy, culture in NNN medium
Malaria	<i>Plasmodium falciparum</i>	Retinal haemorrhage, papilloedema, cotton wool spots	BFMP, Buffy coat, PCR, serological
Microsporidiosis	<i>Microsporidia spp</i>	Conjunctival hyperemia, punctate epithelial keratitis, hyphema, necrotizing keratitis, corneal ulcer	Corneal scrapings, biopsy, serological
Rhinosporidiosis	<i>Rhinosporidium seeberi</i>	Conjunctival granuloma	Histopathologic demonstration
Toxoplasmosis	<i>Toxoplasma gondii</i>	Congenital: Strabismus, nystagmus and blindness Acute acquired: Primarily; necrotizing chorioretinitis, vitritis is common Secondary findings include scotoma, photophobia, blindness, Glaucoma, ↑ IOP, necrotizing inflammation, loss of central vision	Serology (IgM, IgG), PCR

Buffy coat: The thin layer of concentrated white blood cells that forms when a tube of blood is spun in a centrifuge. BFMP: Blood Film for Malaria Parasite. PCR: polymerase chain reaction. NNN: Novy-MacNeal-Nicolle medium.

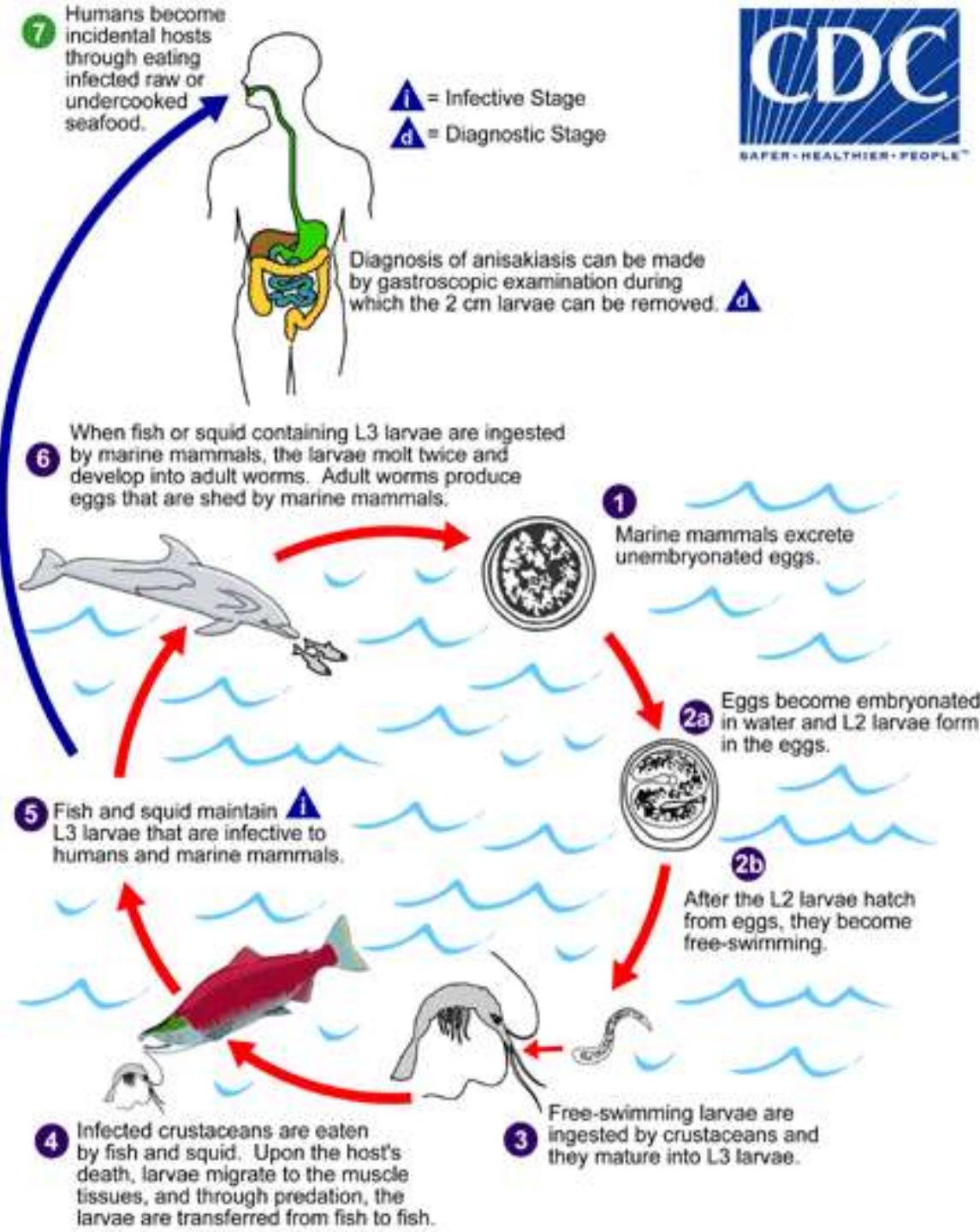


**Figure 2: Clinical symptoms and signs of human angiostrongyliasis with eosinophilic meningitis**  
Data are summarised from references reported in Thailand, Taiwan, mainland China, and the USA.<sup>11,19,24,26,27,31-38</sup>

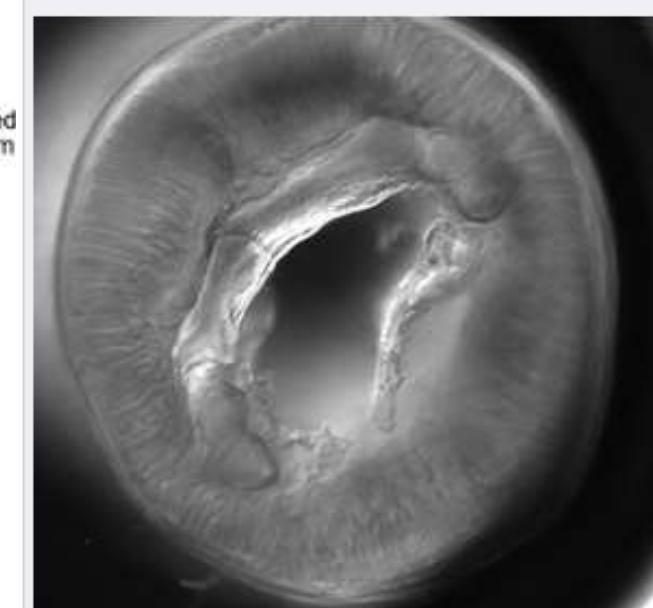
# Angiostrongiliasis

- Etiologic Agents: *Angiostrongylus cantonensis* (rat lungworm).
- Source: Ingestion of neurotropic immature larvae in intermediate hosts: snails, slugs, shrimp, frog (paratenic hosts), or greens contaminated by minute slugs.
- Clinical Manifestations: Nausea, vomiting, constant headache, meningismus, usually insidious onset. Parasthesias, cranial nerve palsies may occur.
- Pathology: Leptomeningitis, lymphocytic & eosinophilic reaction around larvae on surface of brain & in parenchyma.
- Laboratory Diagnosis: Serology available only in SE Asia. CSF eosinophilia, inconstant peripheral eosinophilia. MRI with generalized meningeal thickening.
- Epidemiology: Mainly Thailand, Taiwan, South Pacific. Possibly emerging in the Caribbean (2000 Jamaica). Associated with imported giant African snails.
- Prevention and Control: Cooked food.
- Treatment: Repeated CSF taps, analgesia. Anthelmintics contraindicated. Steroids may be of benefit.

# Anisakiasis



L3 larva of *Pseudoterranova* sp. Ten units = one centimeter.



Cross-section of *Pseudoterranova* sp. viewed under differential interference contrast (DIC) microscopy.

するめいかー Anisakis – a foo... Anisakis – A foo... Haematologica... CDC - DPDx - D... CDC - DPDx - A... CDC - DPDx - G... CDC - DPDx - B... CDC - DPDx - In... FITO PAEZ - Gir...

https://www.youtube.com/watch?v=9kNH65njNRM

anisakis video

YouTube Subir un video

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森く(うごめく)アニサキス (Wriggling Anisakis)  
de ryouchanpapa1 361,690 vistas 0:26

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de takamini905 170,661 vistas 5:24

胡瓜の変わった切り方(way of cutting of the cucumber which is special)  
de itasan16 206,296 vistas 10:20

スルメイカのさばき方.flv  
de anshinada5345 98,790 vistas 2:12

ヤリイカのおろし方と刺身  
de Kinniku ryourin 肉内料理人 134,034 vistas 8:29

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平目のサク取りと皮引き  
de kyuuzan kurozumoushi 924,505 vistas 7:40

さばのさばき方 (Mackerel)  
de itasan16 455,606 vistas

wasyoku kazuya presents

するめいかのさばき方(塩辛の作り方付)Japanese Flying Squid

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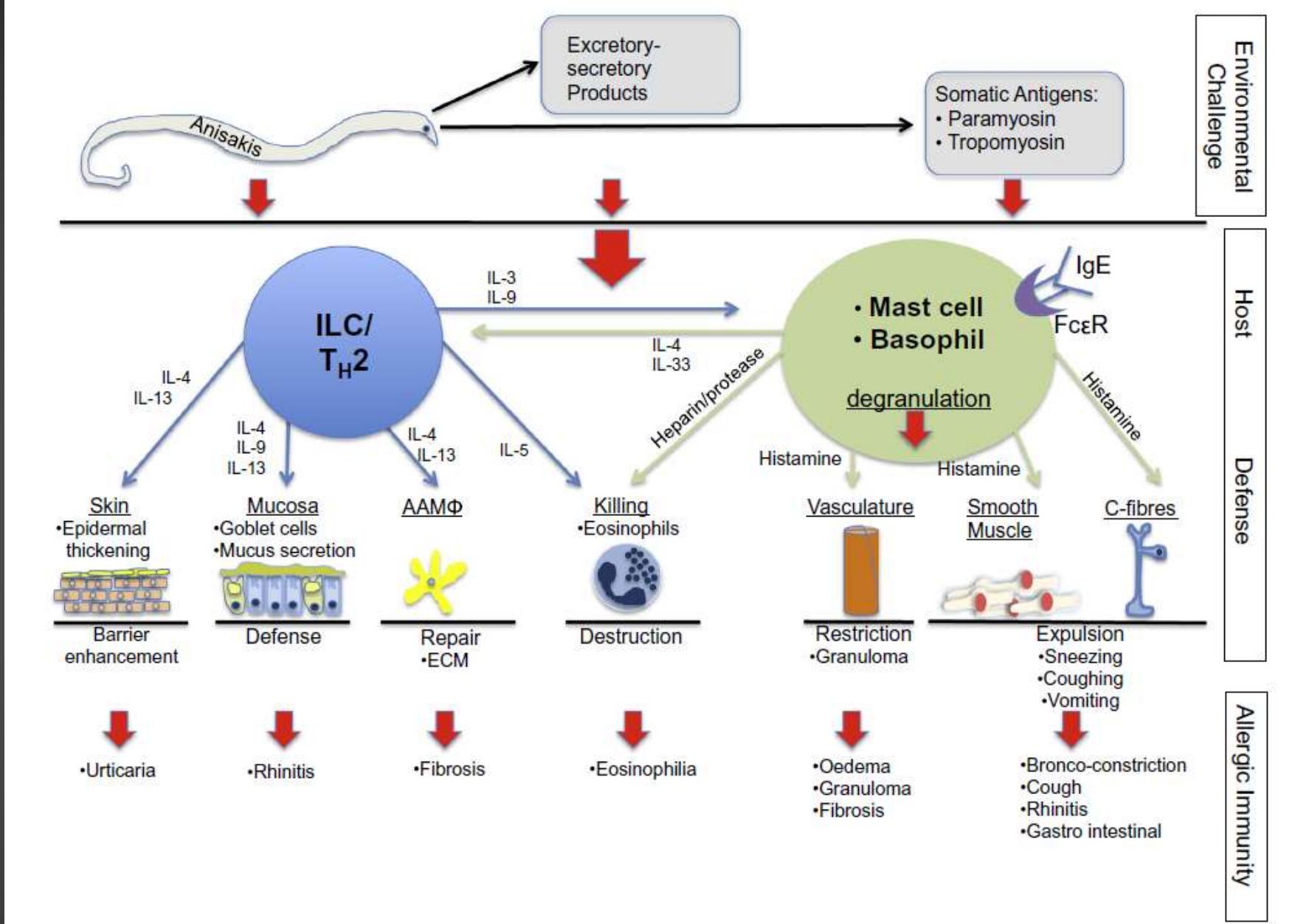
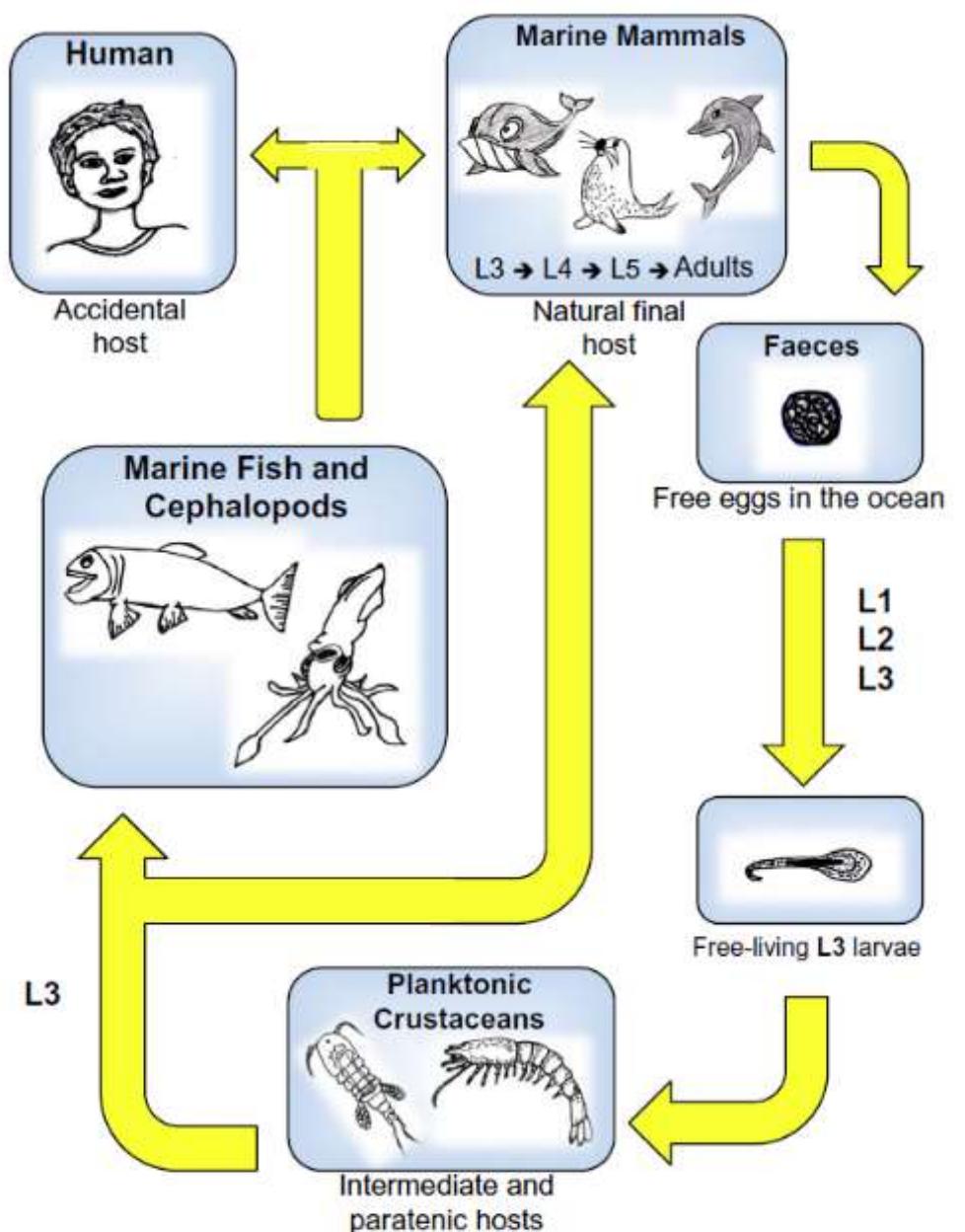
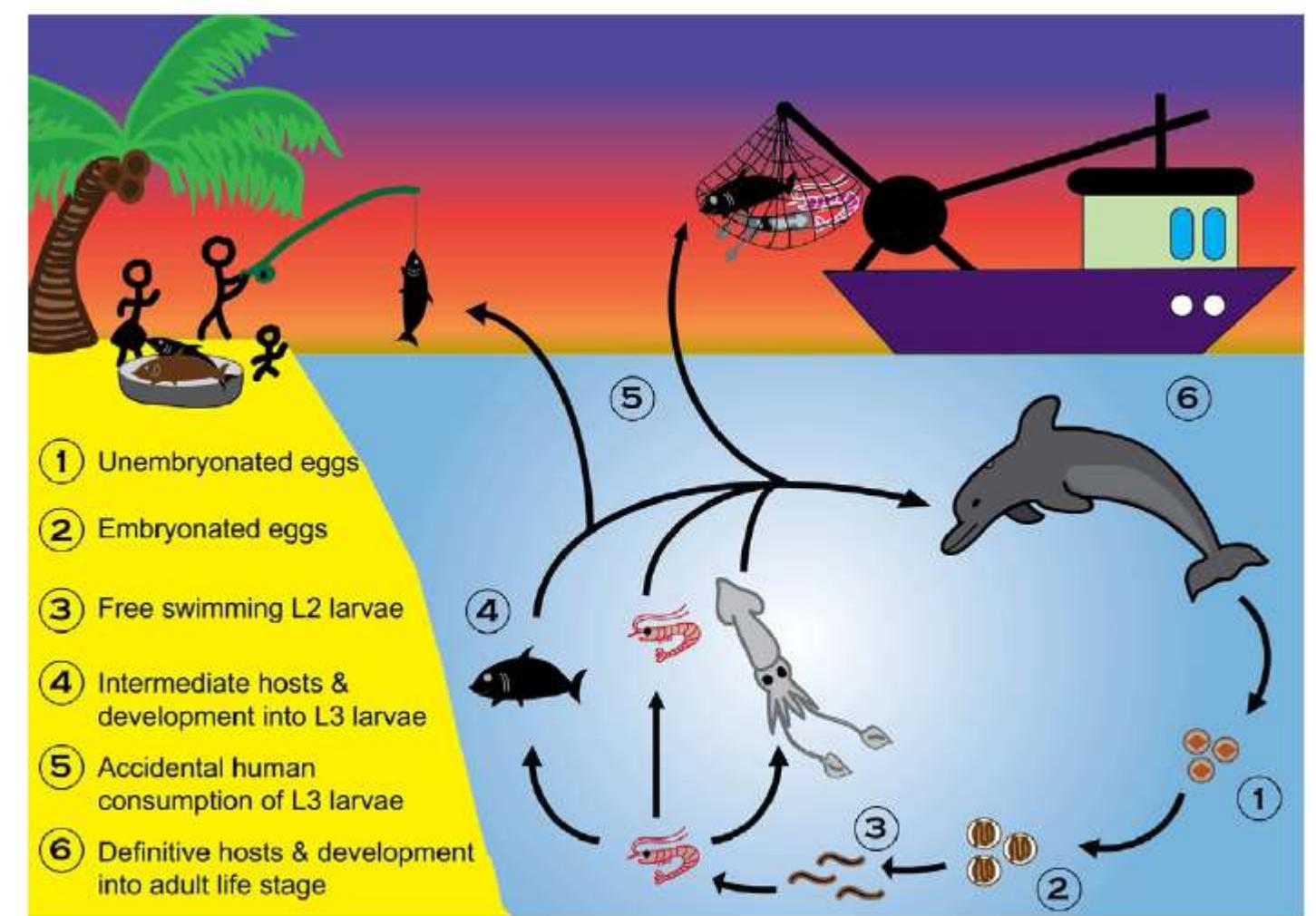


Fig. 4. Functional module of allergic host defence response. Various stimuli activate the allergic host defence response to *Anisakis*: the live helminth itself, a range of



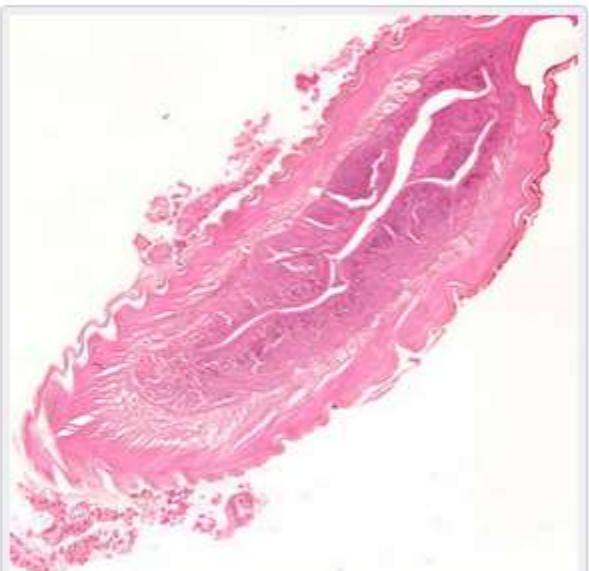
**Fig. 1.** Life cycle of *Anisakis simplex* including accidental human hosts. Adult parasites live in the stomach of marine mammals and unembryonated eggs are expelled with the faeces. These eggs develop and hatch, releasing free-living *A. simplex* L3s. These L3s are ingested by krill (euphausiid) and copepods, which form the intermediate hosts. Marine fish and cephalopods, which are paratenic hosts, contribute to the dissemination of this parasite by ingesting crustaceans, fish and cephalopods infected with L3s. The infective L3s are mostly embedded in the viscera and muscle and transferred to the final hosts (marine mammals) by ingestion of infected fish, cephalopods or krill. The L3 develops to the adult in the final host, closing the life cycle of this parasite by producing and releasing eggs. Ingestion of raw fish or cephalopods infected with L3s by humans, who are accidental hosts since the larvae do not develop further, can generate adverse reactions through activation of various host-defence responses.

Nieuwenhuizen NE, Lopata AL.  
*Anisakis--a food-borne parasite that triggers allergic host defences.*  
 Int J Parasitol. 2013 Nov;43(12-13):1047-57. doi: 10.1016/j.ijpara.2013.08.001.  
 Epub 2013 Aug 27.

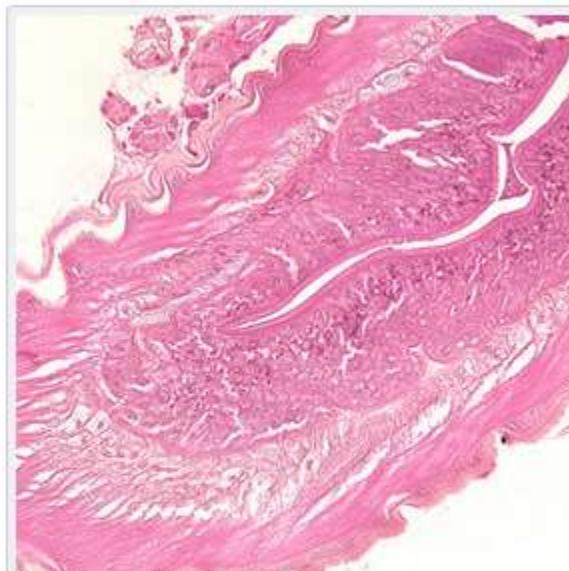


**Fig. 1.** Life cycle of the species of *Anisakis*, including humans as accidental paratenic hosts.

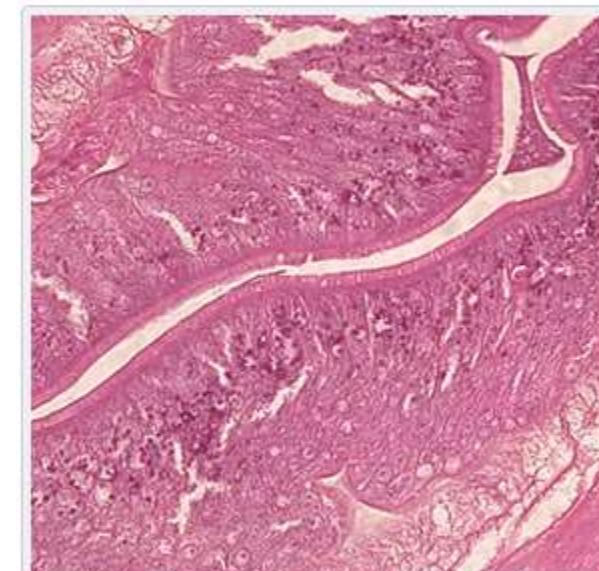
▼ Anisakid worms in tissue specimens, stained with hematoxylin and eosin (H&E).



**Figure A:** Larva of an anisakid worm from a gastric biopsy specimen, stained with H&E. Image taken at 100x magnification.



**Figure B:** Higher magnification (200x) of the specimen in **Figure A**, showing a close-up of the thick, multi-layered cuticle and tall, prominent muscle cells.



**Figure C:** Higher magnification (400x) of the specimen in **Figures A** and **B**, showing a close-up of the folded intestine with a brush border.

Diagnóstico  
Clínico-epidemiológico  
Inmunológico  
Molecular

# Anisakiasis

- **Etiologic Agents:** *Anisakis simplex*, *Anisakis physeteris*, *Pseudoterranova decipiens*
- **Source:** Ingestion of intermediate stage larvae in undercooked infected fish or shellfish. Definitive development in stomach of dolphins & other mammals.
- **Clinical Manifestations:** Variable abdominal pain may be accompanied by nausea, diarrhea, or vomiting. Mild to moderate circulating eosinophilia. Intestinal disease may not present for 1 or 2 weeks. May cough up dead *Pseudoterranova*.
- **Pathology:** Eosinophilic granuloma surrounding attachment site(s) in gastric or intestinal mucosa; edema more pronounced around nematode.
- **Laboratory Diagnosis:** Confirmed diagnosis from recovered anisakid nematode. May be presumptively diagnosed by food history, eosinophil count.
- **Epidemiology:** Worldwide, especially where raw seafoods are commonly consumed.
- **Treatment:** Removal of worm by endoscope or surgical resection of lesion and nematode.
- **Prevention and Control:** Abstinence from raw and undercooked seafoods. Pre-freeze fish.

## CONTRIBUCIÓN ESPECIAL:

### Anisakidosis: ¿Una zoonosis parasitaria marina desconocida o emergente en el Perú?

TABLA 1 . Características de los casos de anisakidosis humana reportados en el Perú (1993-2002)

Fecha de Diagnóstico	Edad/ Sexo	Localización de la larva	Procedencia (departamento)	Agente etiológico	Autor
1993?	?	Boca	Lima	P. decipiens	12
1993?	?	Boca	Lima	P. decipiens	12
12/01/98	38 M	Estómago	Lima	Anisakis sp.	14
12/1997	22 H	Estómago?	Ica	A. physeteris*	15
01/1998	36 H	Estómago?	Ica	A. physeteris*	15
11/1997	42 M	Boca	Lima	A. simplex	C P
19/07/2001	42 M	Boca	Lima	P. decipiens ?*	N R
				Como: Toxocara sp.	16, 17
24/07/2002	17 M	eliminó	Ica	P. decipiens	13

(\*) Casos probables, Cp: Comunicación personal, Nr: Nuevo registro.

## CONTRIBUCIÓN ESPECIAL:

### Anisakidosis: ¿Una zoonosis parasitaria marina desconocida o emergente en el Perú?

**TABLA 2.-** Peces portadores de larvas de *Anisakis phystesis* y *Pseudoterranova decipiens* en la costa peruana: dinámica de infección

Espezie de larva	Pez hospedador	Muestra	Fecha	Procedencia (costa)	Prevalencia %	Intensidad Media/Rango	Autor
<b>A. phystesis</b>							
	S. japonicus "caballa"	41	1995	Islas Ballestas, Lagunillas, Laguna Grande Pisco (Ica)	36,5	? (1-16)	29
		100	2000?	Callao	20,0	?	30
	T. murphyi "jurel"	45	Julio-Agosto 1994	Lagunillas, Pisco (Ica)	6,66	1	N R
		30	Diciembre 1994	Laguna Grande, Pisco (Ica)	10,00	1	N R
		39	Enero-Febrero 1995	Laguna Grande, Pisco (Ica)	12,83	1,4 (1-2)	N R
		25	Febrero 1995	Lagunillas, Pisco (Ica)	16,00	1	N R
		70	Abril-Junio 1997	Callao	1,4	?	27
	S. sarda chilensis "bonito"	70	Abril-Junio 1997	Callao	24,3	?	27
	C. hippurus "perico", "dorado"	12	Diciembre 1997	Pisco (Ica), Ilo (Moquegua)	58,33	34,28 (14-63)	15
		57	Mayo 2002	Paita (Piura)	33,33	1,37 (?)	31
P. decipiens	T. murphyi "jurel"	?	1993?	Lima y Callao*	?	1	12
	S. japonicus "caballa"	?	?	Lambayeque	?	?	32

NR= Nuevos registros

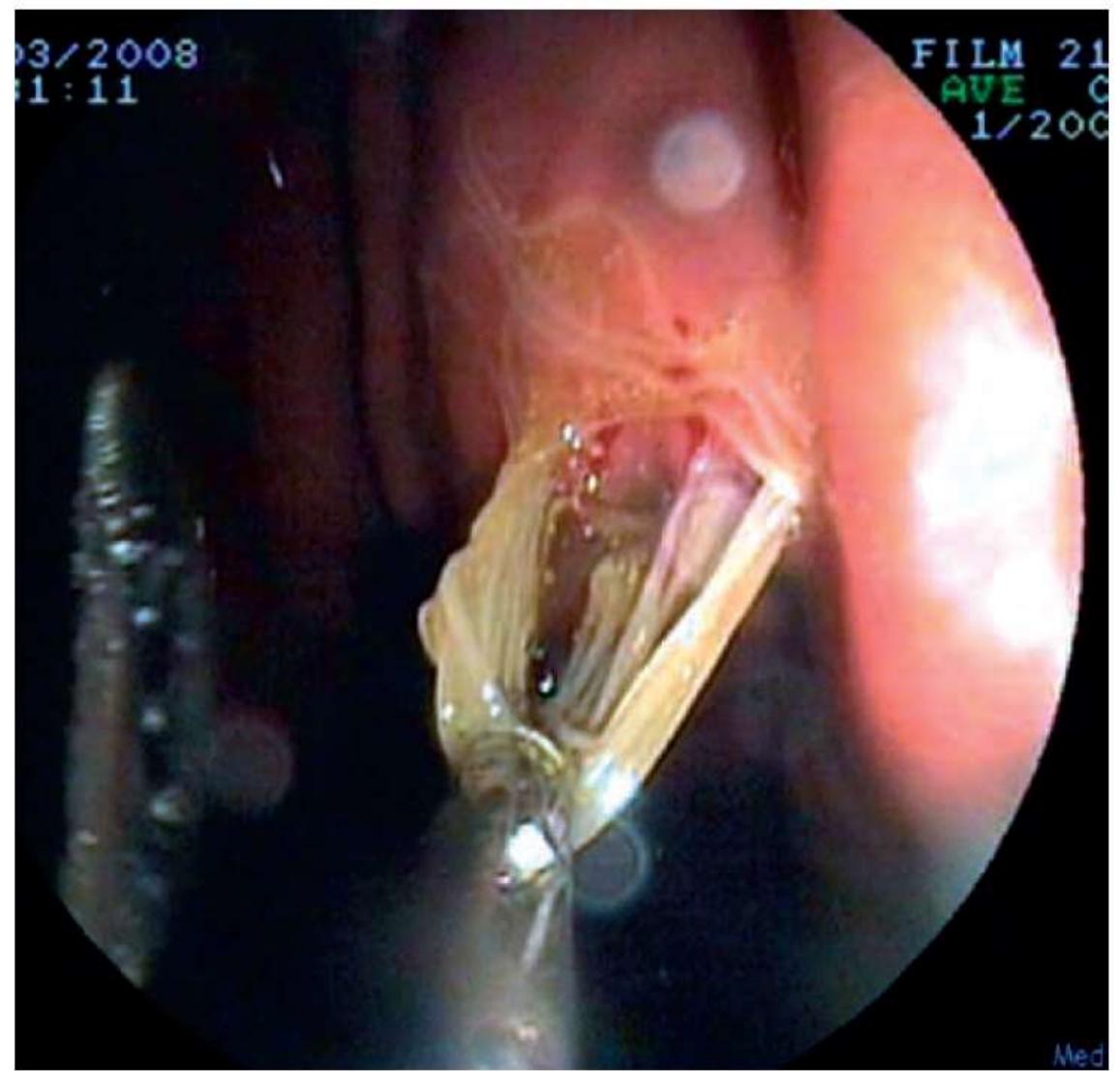


Fig. 1. Nido de AS en antro.

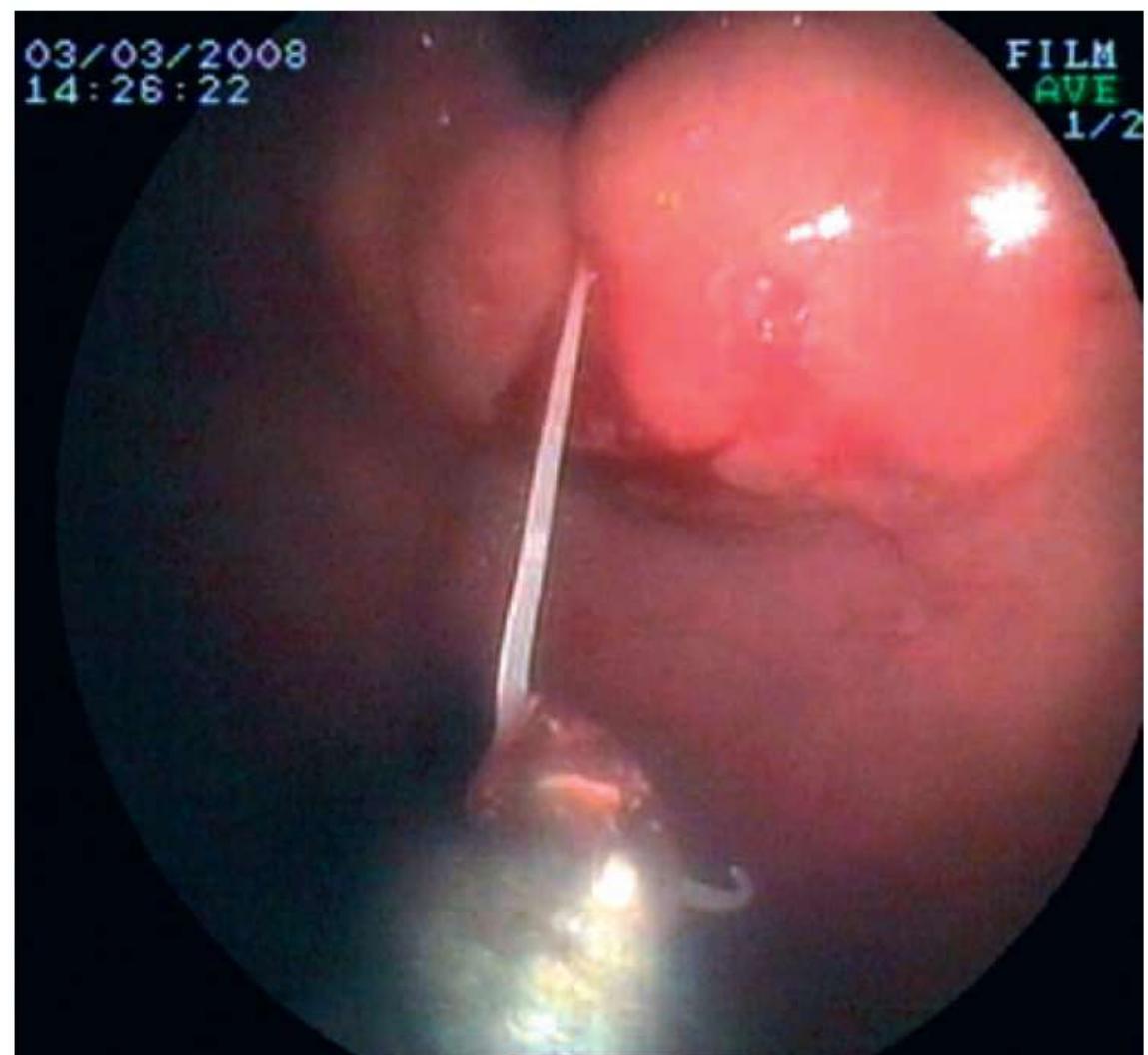
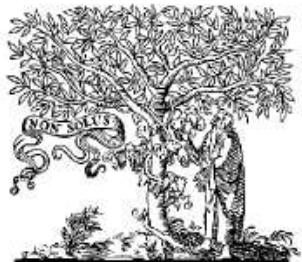


Fig. 2. AS en región subcardial.



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# *Contracaecum* sp. infection in *Hoplias malabaricus* (moncholo) from rivers and marshes of Colombia

Jesús Olivero-Verbel <sup>\*</sup>, Rosa Baldiris-Ávila, Jorge Güette-Fernández,  
Amparo Benavides-Alvarez, Jairo Mercado-Camargo, Barbara Arroyo-Salgado

*Environmental and Computational Chemistry Group, University of Cartagena, Cartagena, Colombia*

Received 25 May 2005; received in revised form 2 March 2006; accepted 8 March 2006

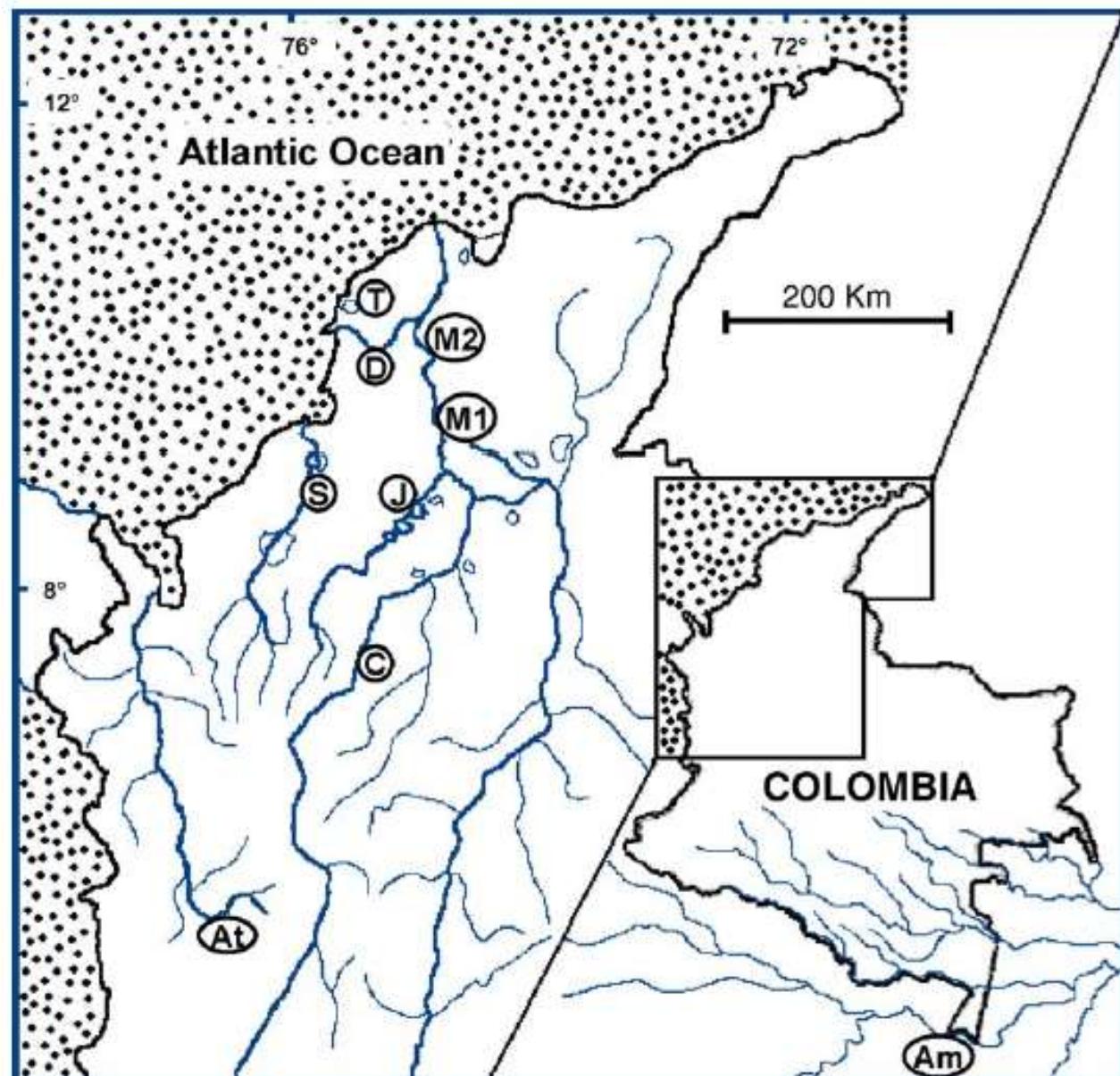


Fig. 1. Map of sampling sites in Colombia. M1, Magdalena River (Magangué); M2, Magdalena River (Zambrano); S, Sinú River; C, Cauca River; J, San Jorge River; D, Dique Channel; T, Totumo marsh; At, Atrato River; Am, Amazonas River.

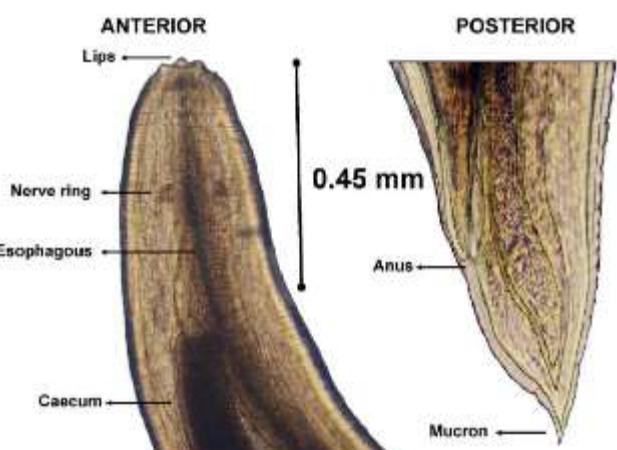


Fig. 2. Morphological features of parasites found in *Hoplaxius malabaricus*.

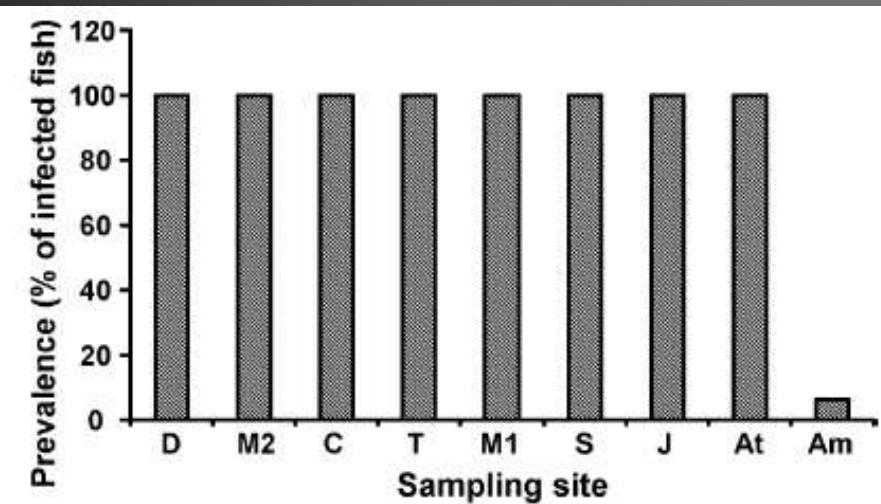


Fig. 3. Prevalence of parasite infection in *Hoplias malabaricus* from different freshwater waterbodies in Colombia.

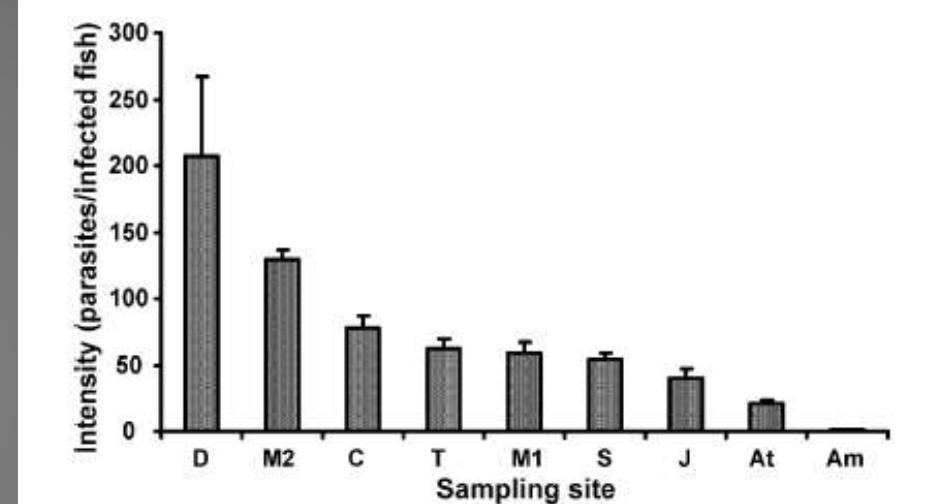
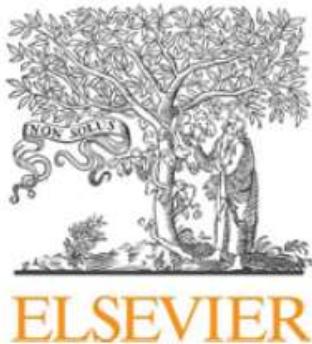


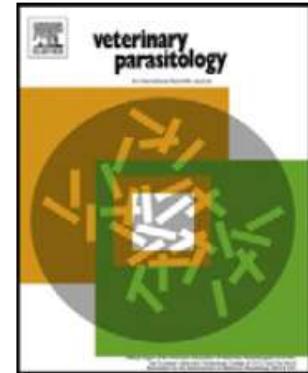
Fig. 4. Intensity of parasite infection in *Hoplias malabaricus* from different freshwater waterbodies in Colombia.



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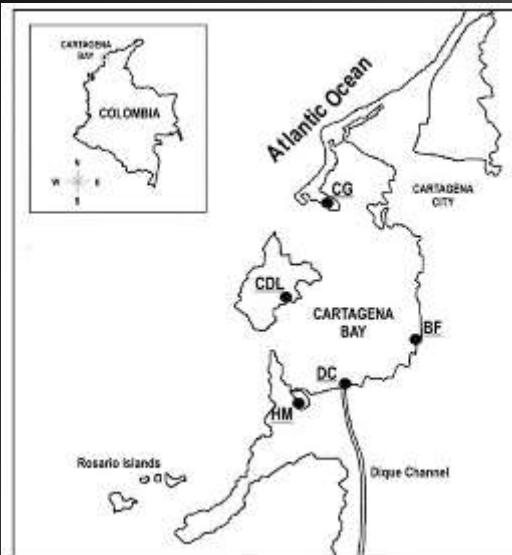
journal homepage: [www.elsevier.com/locate/vetpar](http://www.elsevier.com/locate/vetpar)



## Nematode infection in fish from Cartagena Bay, North of Colombia

Jesús Olivero Verbel\*, Karina Caballero-Gallardo, Bárbara Arroyo-Salgado

*Environmental and Computational Chemistry Group, Faculty of Pharmaceutical Sciences, University of Cartagena, Campus of Zaragocilla, Cartagena, Colombia*



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# Baylisascariasis

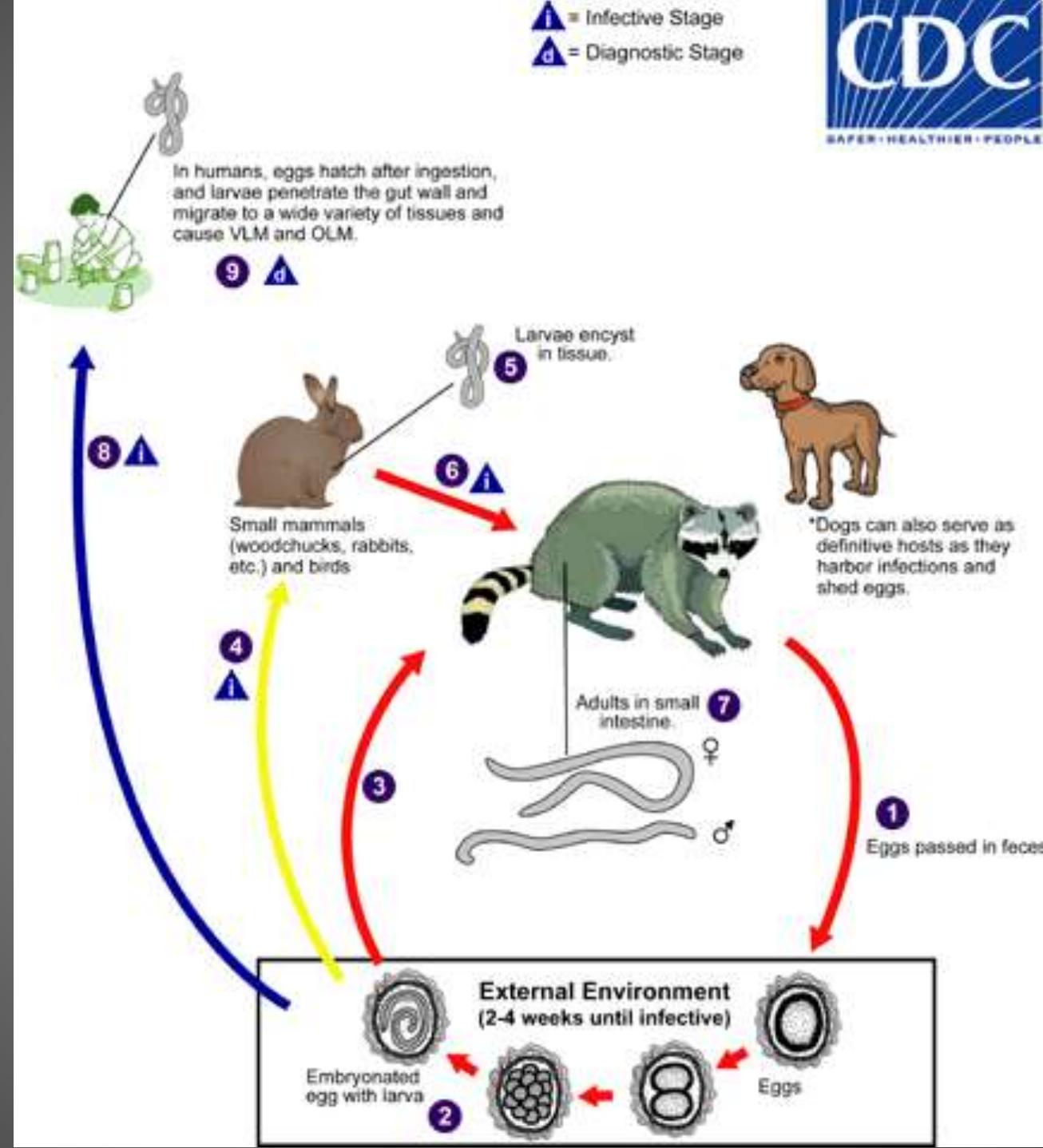
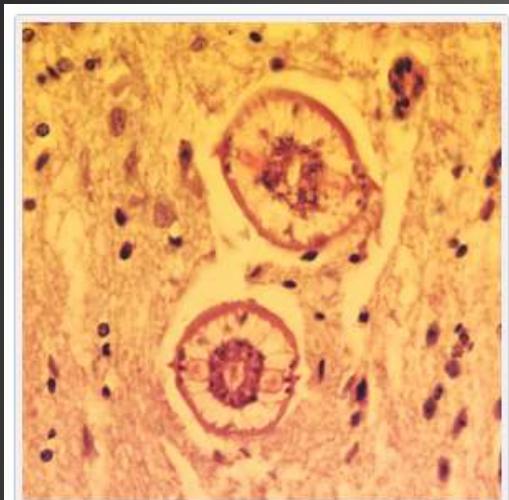


TABLE 1. Summary of published cases of human *Baylisascaris procyonis* neural larva migrans

Yr <sup>a</sup>	Age <sup>b</sup>	Location	Risk factor(s)	Treatment	Outcome(s)	Reference
1980	10 mo	Pennsylvania	Pica	None	Died	25
1984	18 mo	Illinois	Down syndrome and pica	Thiabendazole	Died	16
1986	21 yr	Oregon	Developmental delay, pica/geophagia	Not recorded	Persistent residual deficits	Cited in reference 10
1990	13 mo	New York	Pica	Thiabendazole, ivermectin, and prednisone	Severe residual deficits and cortical blindness	10
1993	9 mo	Michigan	Pica	Not recorded	Severe residual deficits and cortical blindness	Cited in reference 44
1993	13 mo	California	Pica/geophagia	Solumedrol and prednisolone	Severe residual deficits, visual impairment, and epilepsy	51
1996	6 yr	Illinois	Developmental delay, pica/geophagia	Albendazole and prednisone	Severe residual deficits and epilepsy	18
1996	13 mo	Minnesota	Unknown	Methylprednisolone, vincristine, and thioguanine	Died	41
1997	19 mo	Minnesota	Klinefelter syndrome	Prednisone, vincristine, and thioguanine	Died	41
1998	11 mo	California	Pica	Albendazole and methylprednisolone	Severe residual deficits, visual impairment and epilepsy	47
2000	17 yr	California	Developmental delay and geophagia	Albendazole and anti- inflammatories	Died	8
2000	2.5 yr	Illinois	Pica/geophagia	Albendazole and solumedrol	Severe residual deficits and visual impairment	8, 18
2002	11 mo	California	Pica/geophagia	Albendazole and antiinflammatories	Severe residual deficits, cortical blindness, and epilepsy	Cited in references 44 and 53

<sup>a</sup> Year patient first presented.<sup>b</sup> All patients were male.

**TABLE  
71.4** **Helminths that May Infect the Central Nervous System**

Organism	Main Areas of Distribution	Presentation	Management
<b>CESTODES</b>			
Neurocysticercosis	Latin America, sub-Saharan Africa, India	Seizures, mass lesion, hydrocephalus	Albendazole 15 mg/kg/day in 2 divided doses for 8 days. Cover with corticosteroids (see text)
<i>E granulosus</i> (Hydatid disease)	Worldwide	Mass lesion, seizures	Surgical resection. Pretreat: albendazole 15 mg/kg/day in 2 divided doses (40 days) to shrink cyst. If unresectable: albendazole 10–15 mg/kg/day in two divided doses twice a day ≥ 3 months
<i>Taenia multiceps</i> (coenurosis)	Worldwide	Hydrocephalus	Surgical removal
<i>Spirometra</i> sp. (Sparganosis)	Far East and S-e Asia, East Africa	Seizures, infarcts	Surgical removal
<b>NEMATODES</b>			
<i>Angiostrongylus cantonensis</i>	S-e Asia, Caribbean, Southern USA	Meningoencephalitis	Repeated LPs to reduce ICP. Corticosteroids + Albendazole 10–15 mg/kg/day in two divided doses – 2 weeks
<i>Gnathostoma spinigerum</i>	S-e Asia, Mexico, Ecuador, Japan	Meningoencephalitis, seizures, myeloradiculopathy	Albendazole 10–15 mg/kg/day in 2 divided doses – 21 days + corticosteroids. Surgical removal if accessible
<i>Onchocerca volvulus</i>	West Africa, Yemen, Latin America	Chorioretinitis, keratitis, seizures	Ivermectin single oral doses of 0.15 mg/kg single oral dose
<i>Baylisascaris procyonis</i>	Worldwide	Meningoencephalitis	Corticosteroids, albendazole 10–15 mg/kg/day in 2 divided doses
<i>Trichinella</i> sp.	Worldwide	Myopathy, strokes, meningoencephalitis	Corticosteroids, repeated LPs
<b>TREMATODES</b>			
Schistosomiasis	Africa, Asia, Brazil	cauda equina/conus syndrome/ cerebral granuloma	<i>S. mansoni</i> , <i>S. haematobium</i> and <i>S. intercalatum</i> – Praziquantel 40 mg/kg/day – 3 days, corticosteroids (see text) <i>S. japonicum</i> and <i>S. mekongi</i> – Praziquantel 60 mg/kg/day – 3 days, corticosteroids (see text)
<i>Paragonimus</i> sp.	Latin America, Asia, West Africa	Encephalitis, mass lesion, infarcts, seizures, myelopathy	Praziquantel 25 mg/kg three times a day – 3 days, corticosteroid
<i>Fasciola hepatica</i>	Worldwide	Meningitis, mass lesion, infarct	Triclabendazole 10 mg/kg single dose or 20 mg/kg in two divided doses

S-e, South-east; LP, lumbar puncture; ICP, intracranial pressure; meningoencephalitis in this table refers to an eosinophilic meningoencephalitis.

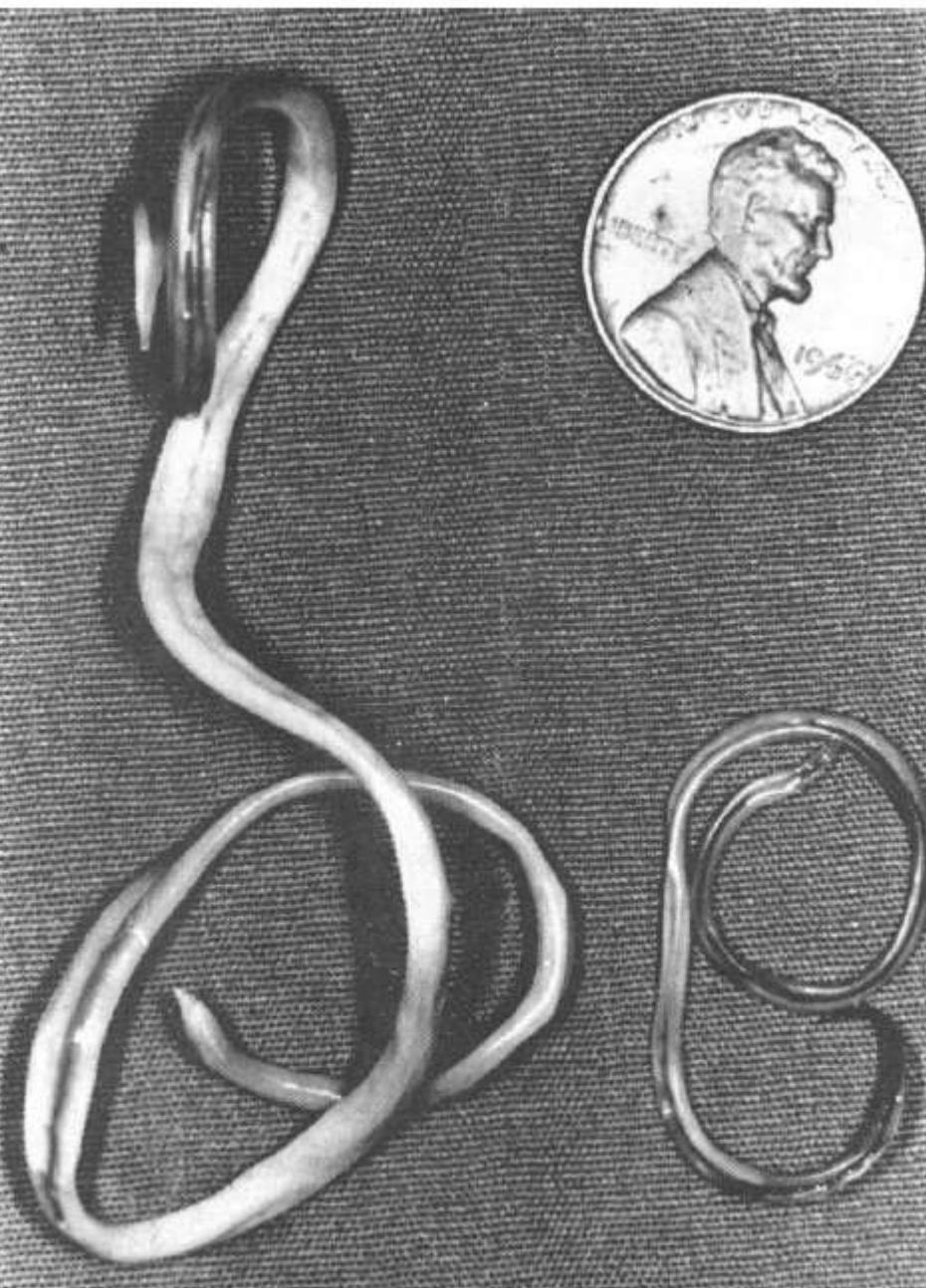


FIG. 2. Adult *B. procyonis* nematodes. The female, on the left, is 24 cm long; the male, on the right, is 12 cm long. (Reprinted from reference 43 with permission from Elsevier.)

Gavin PJ, Kazacos KR, Shulman ST. Baylisascariasis. Clin Microbiol Rev. 2005 Oct;18(4):703-18.



FIG. 3. Infective *B. procyonis* egg (diameter, 70  $\mu\text{m}$ ) containing a coiled second-stage larva. Recovered from soil and debris at a raccoon latrine. Magnification,  $\times 40$ .



FIG. 4. Large group of raccoons living in a suburban Chicago cemetery and feeding on bread left by well meaning but misguided wildlife enthusiasts. (Photograph courtesy of Jon Randolph, Chicago, Ill., reproduced with permission.)

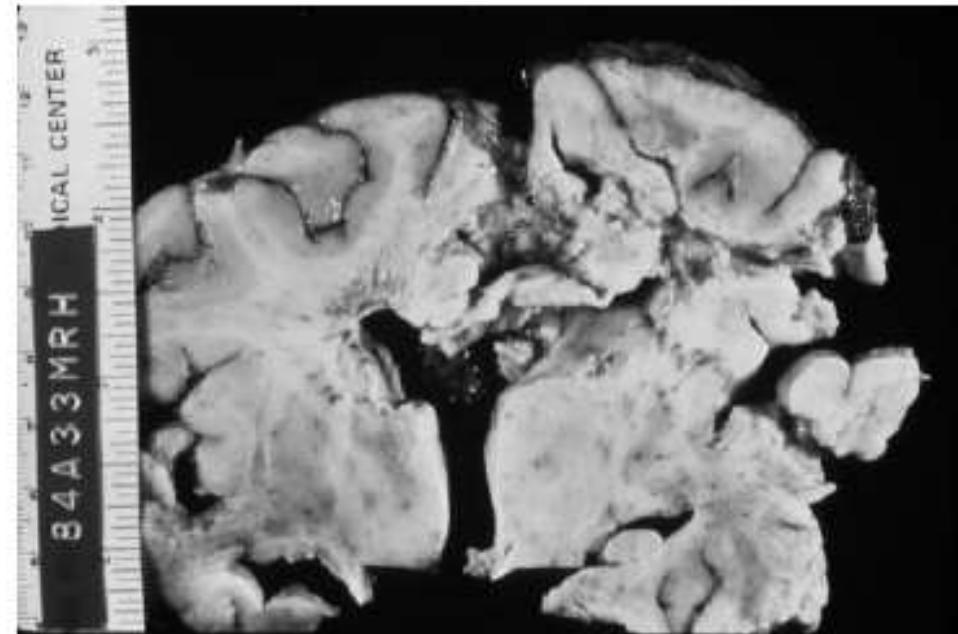


FIG. 6. Coronal section of the brain of an 18-month-old boy who died of acute *B. procyonis* NLM. The leptomeninges are congested. There is marked swelling and softening of the brain parenchyma. Necrosis is evident in the deep periventricular white matter, and there are numerous track-like spaces visible to the naked eye.

**TABLE  
71.3**

## Causes of Seizures/Epilepsy

Infections	Bacterial meningitis Cerebral malaria TB meningitis Cryptococcal meningitis Neurocysticercosis Schistosomiasis Cerebral hydatid disease Paragonimiasis Cerebral toxoplasmosis Cerebral amoebiasis  Neuroangiostrongyliasis Gnathostomiasis Baylisascariasis
Focal brain lesions	Tetanus (pseudoepilepsy) Tumours, cysts, granulomas, tuberculoma
Toxins/drugs/metabolic	Alcohol, opiates, altered glucose levels
Metabolic Stroke	Hypoglycaemia Haemorrhage, intracerebral/ subarachnoid



FIG. 5. Typical raccoon latrines, found at the base of a group of trees (A), on logs (B), and on the roof of a home in suburban California (C). (Reprinted from reference 43 with permission from Elsevier.)

Gavin PJ, Kazacos KR, Shulman ST. Baylisascariasis. Clin Microbiol Rev. 2005 Oct;18(4):703-18.

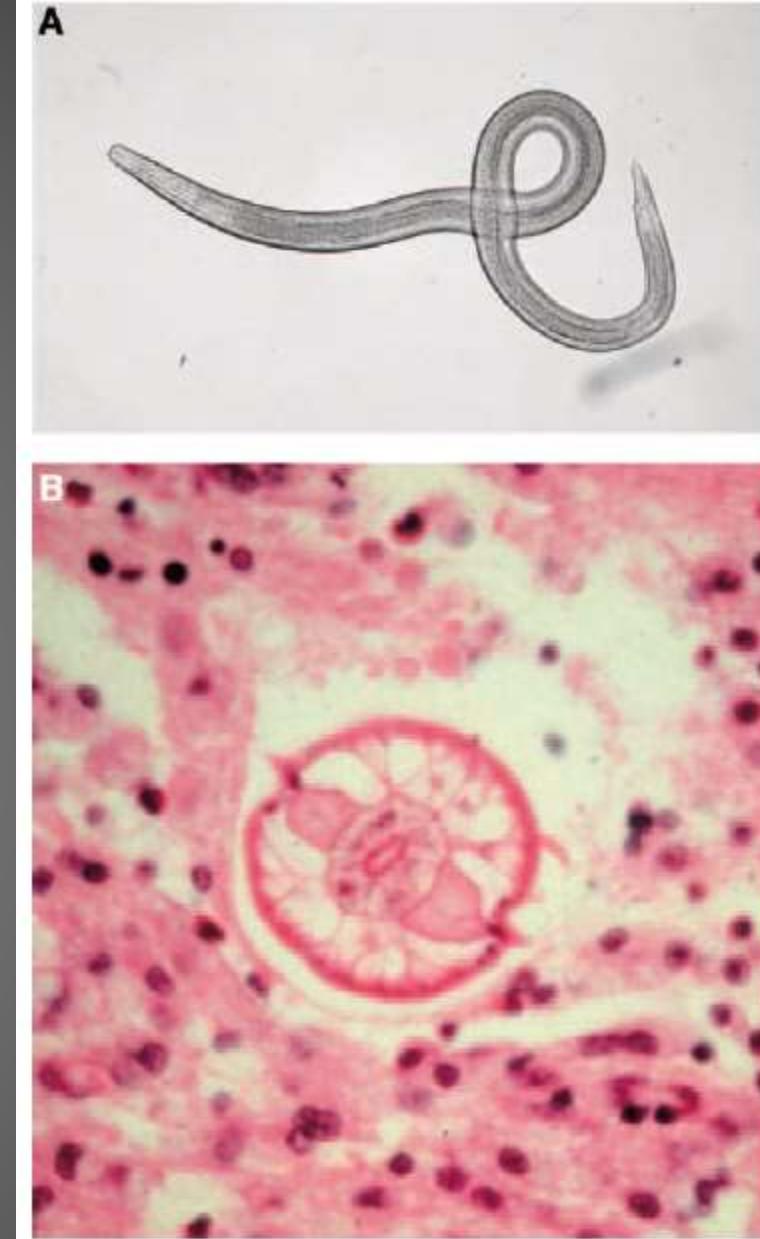


FIG. 7. (A) *Baylisascaris procyonis* larva recovered from the brain of a pet New World parrot with fatal NLM; (B) cross-section (at midbody level) (diameter, 60  $\mu$ m) recovered from the cerebrum of a rabbit with NLM. Characteristic features of the larva include a centrally located (slightly compressed) intestine, flanked on either side by large triangular excretory columns. Prominent lateral cuticular alae are visible on opposite sides of the body. Hematoxylin and eosin stain.

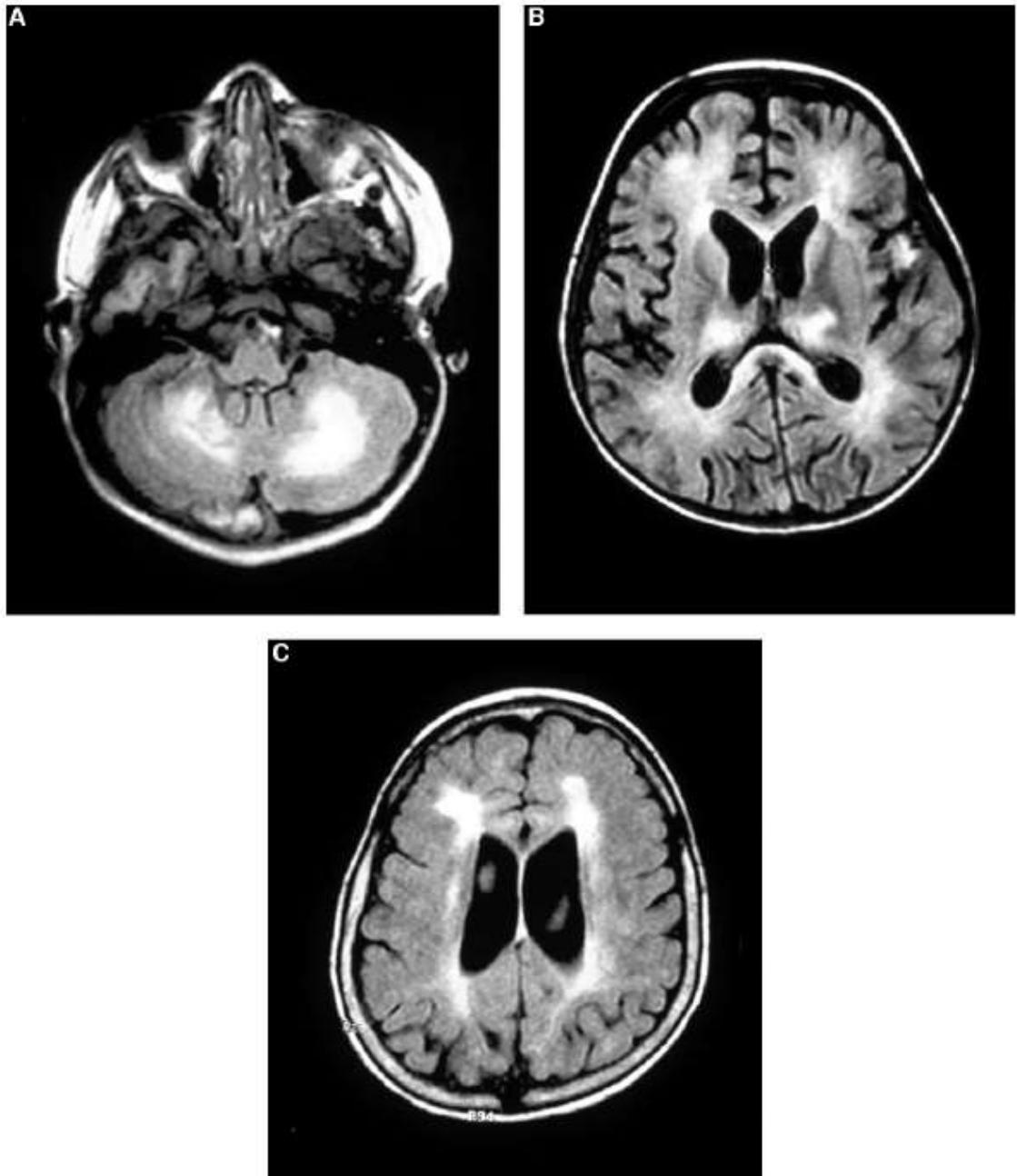


FIG. 8. Neuroimaging of human brain with *B. procyonis* NLM. (A) In acute NLM, an axial-flair magnetic resonance (MR) image (at the level of the posterior fossa) demonstrates an abnormal hyperintense signal of cerebellar white matter; (B) an axial T2-weighted MR image (at the level of the lateral ventricles) demonstrates an abnormal patchy hyperintense signal of periventricular white matter and basal ganglia; (C) an axial T2-weighted MR image (at the level of the lateral ventricles) of a patient with subacute/chronic NLM demonstrates residual abnormal hyperintense signal of the periventricular white matter, loss of white matter volume, and dilation of ventricles and sulci, consistent with generalized cerebral atrophy.

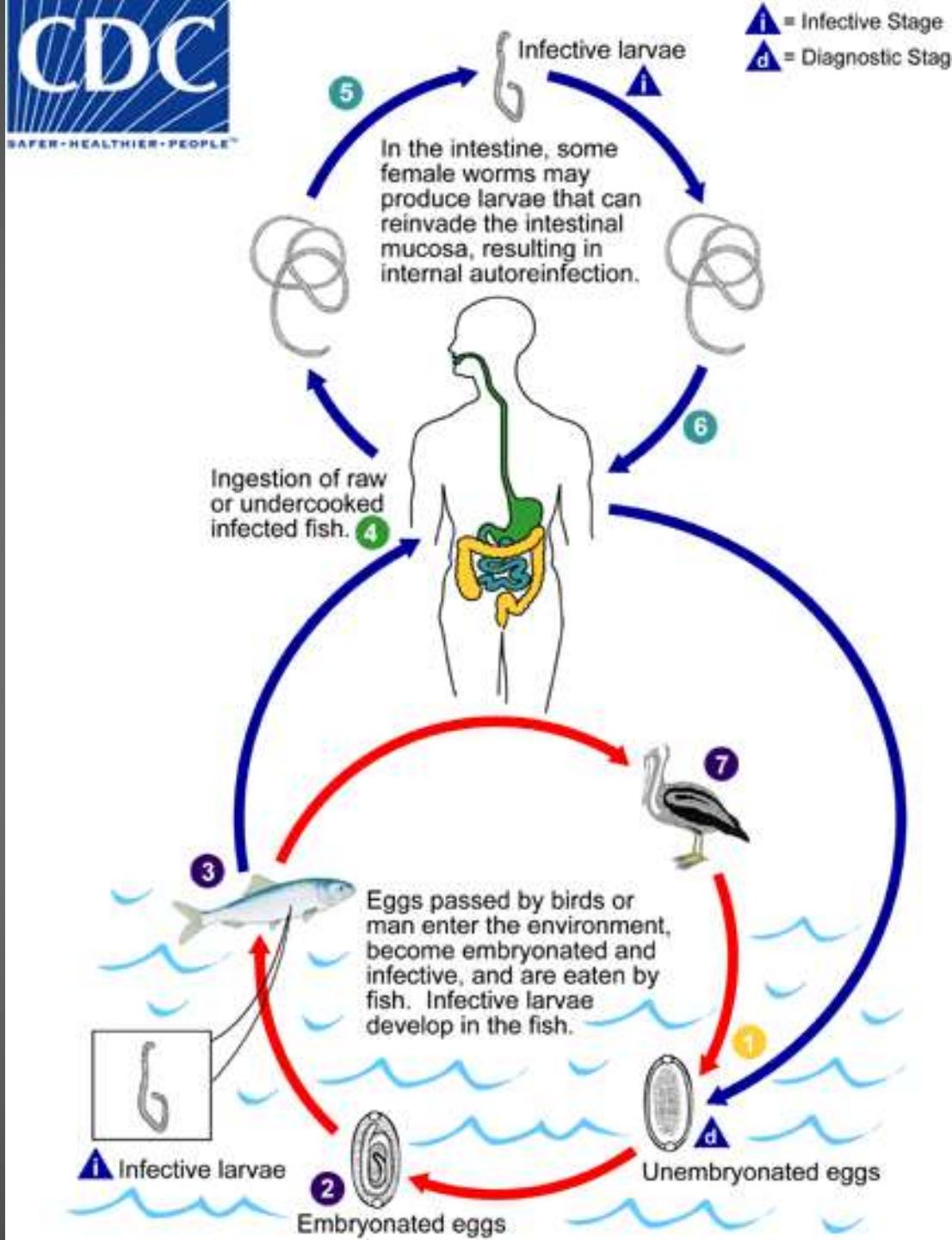
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Molecular

Gavin PJ, Kazacos KR, Shulman ST. Baylisascariasis.  
Clin Microbiol Rev. 2005 Oct;18(4):703-18.

# Capillariasis



Egg of *C. philippinensis* in an unstained wet mount of stool.



Adultos: Intestino delgado del ser humano

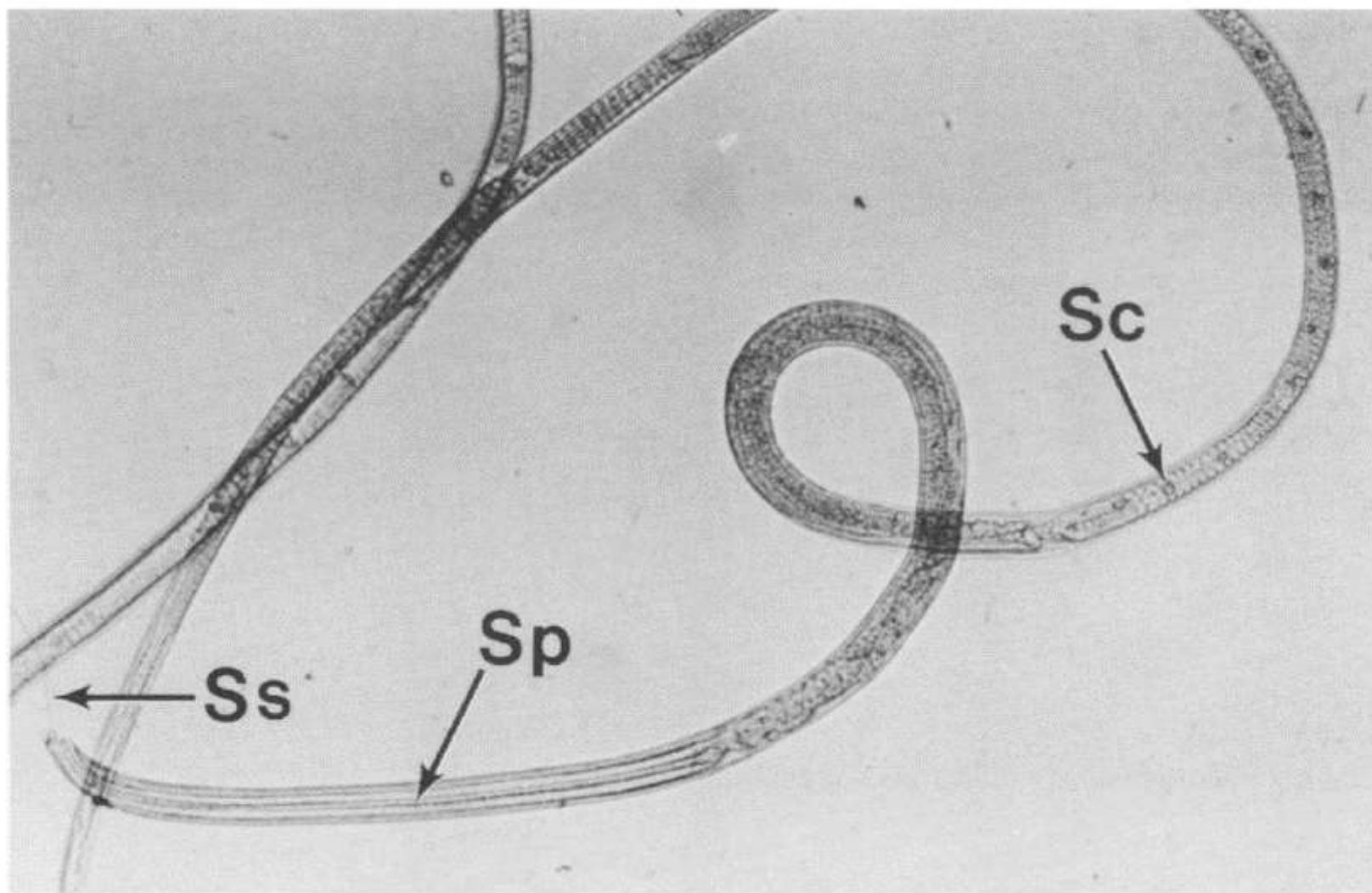


FIG. 1. Adult male *C. philippinensis* indicating spicule (Sp), extended spicular sheath (Ss), and stichocyte (Sc). Magnification,  $\times 32$ .



FIG. 5. Larva of *C. philippinensis* from the intestines of a fish. Note the formation of stichocytes in the anterior end. Magnification,  $\times 100$ .

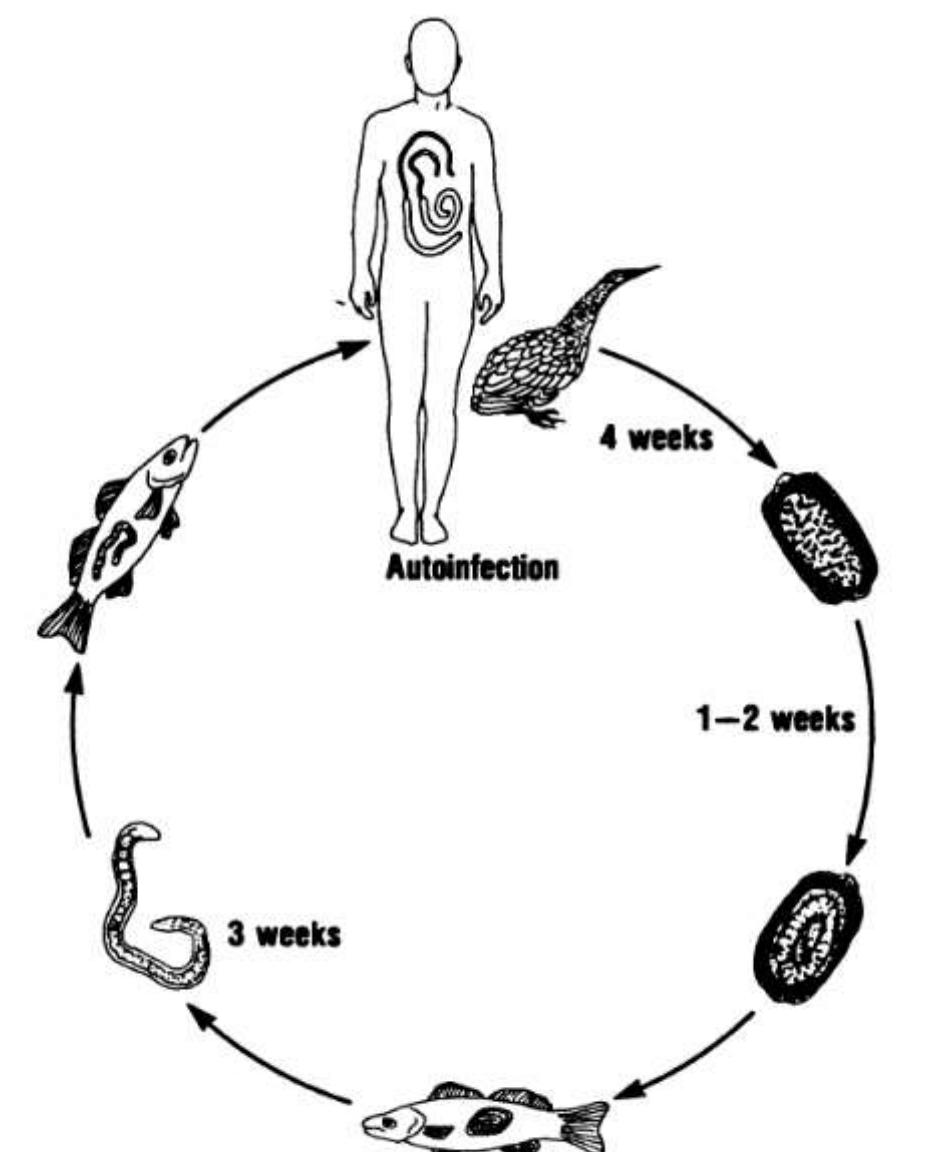


FIG. 6. Proposed life cycle of *C. philippinensis* based on experimental infection in Mongolian gerbils. Reprinted from reference 14 with permission of the publisher.

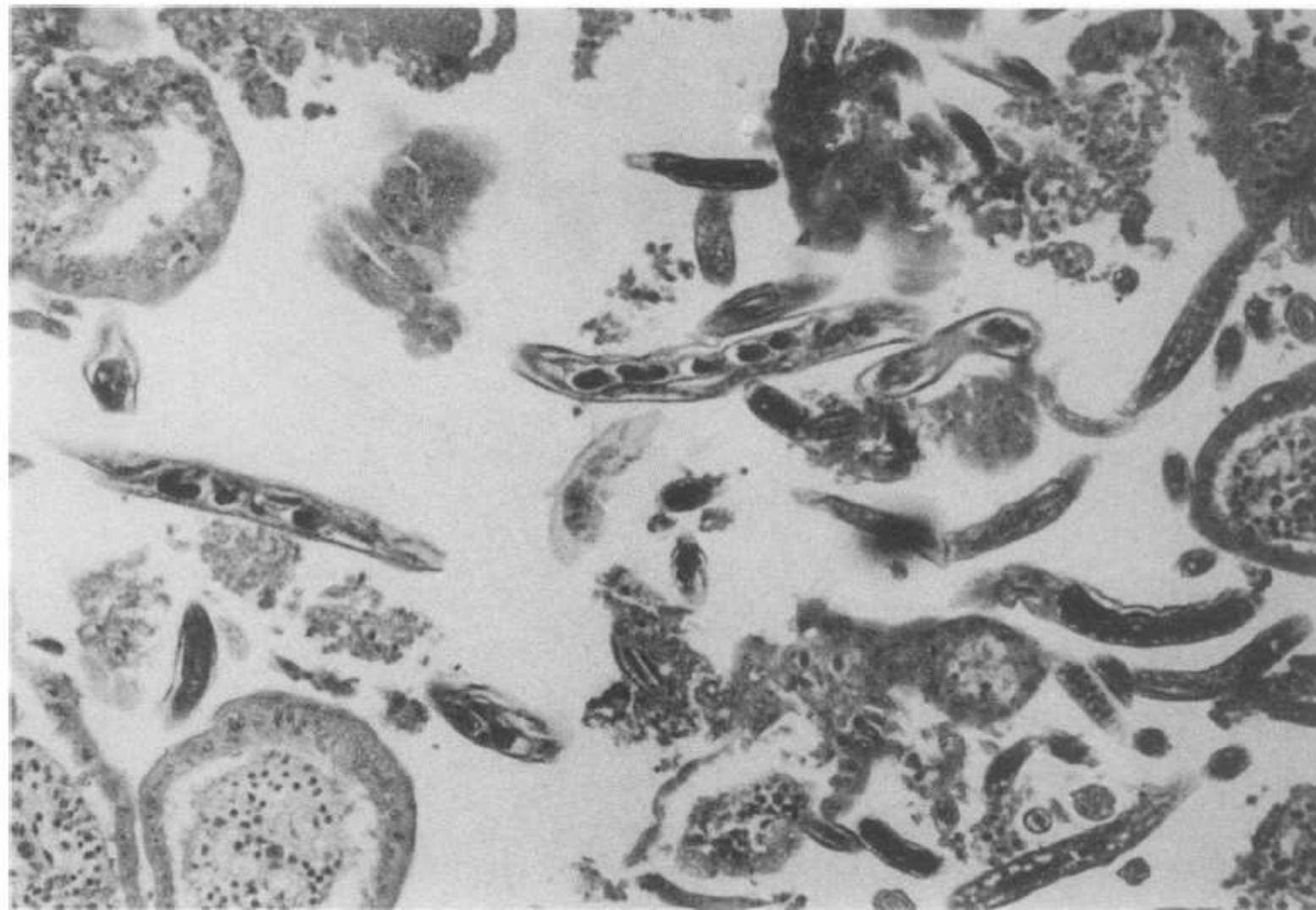


FIG. 7. Human intestinal tissue at autopsy showing multiple histologic sections of *C. philippinensis*. Female worms with eggs in the uterus are visible. Magnification,  $\times 63$ .



FIG. 10. Female *C. philippinensis* (Cp) in crypts (Cr) of human intestinal tissue at autopsy. Note the larva (Lv) in the uterus. Magnification,  $\times 63$ .

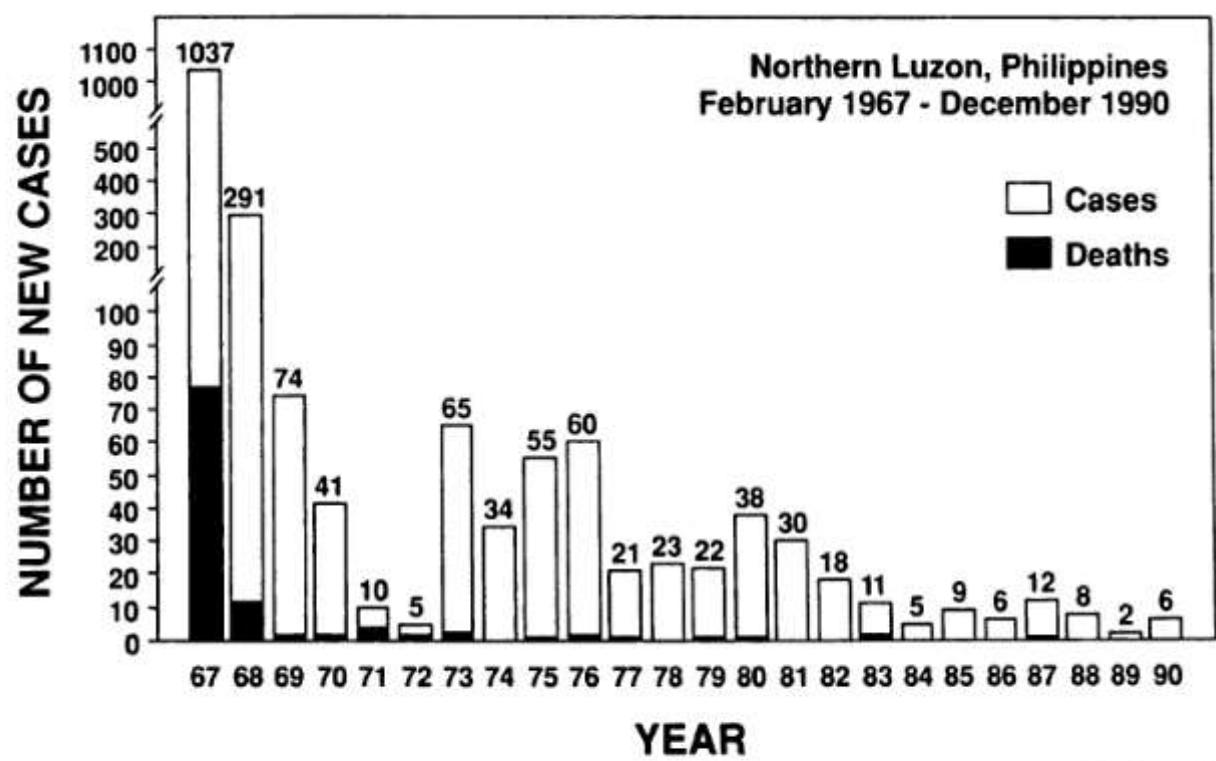
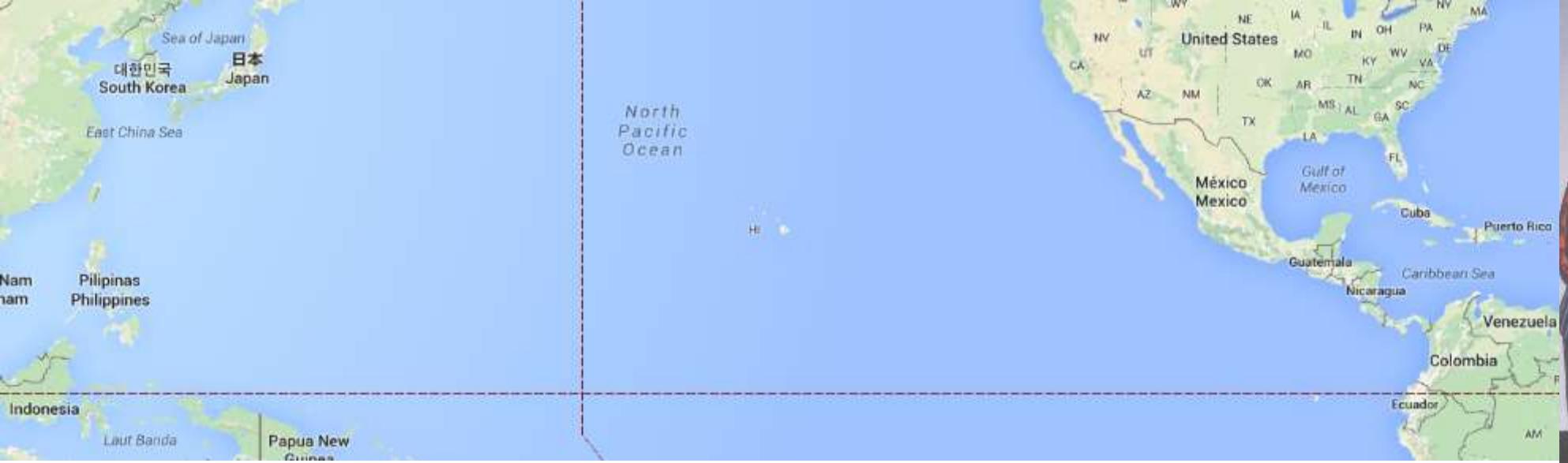


FIG. 15. Intestinal capillariasis cases (total, 1,884) and deaths (total, 110) recorded in Northern Luzon in the Philippines from 1967 through 1990.



IASC Travel Award,  
Manila, Filipinas, 2005

# Diagnóstico coproparasitológico

Huevos, larvas, adultos en heces

Biopsia intestinal



## ▼ *Capillaria philippinensis* eggs.

*Capillaria philippinensis* eggs are 35 to 45 µm in length by 20-25 µm in width. They have two inconspicuous polar prominences and a striated shell. Eggs are unembryonated when passed in feces.



Figure A: Egg of *C. philippinensis* in an unstained wet mount of stool.



Figure B: Egg of *C. philippinensis* in an unstained wet mount of stool.



Figure C: Egg of *C. philippinensis* in an unstained wet mount of stool.

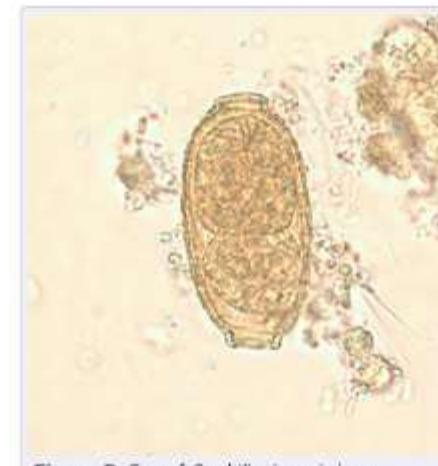


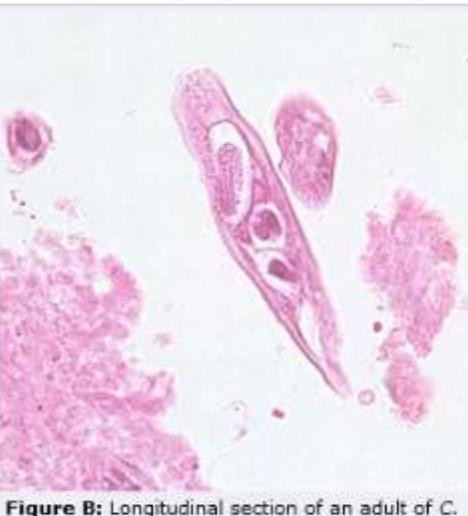
Figure D: Egg of *C. philippinensis* in an unstained wet mount of stool.

▼ *Capillaria philippinensis* adults.

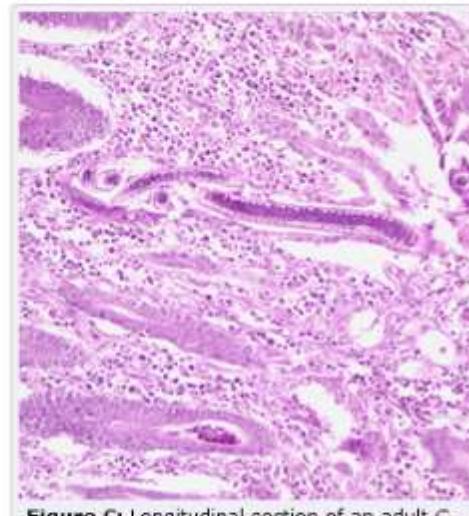
*Capillaria philippinensis* adult males are 2.0-3.5 mm in length and females are 2.5-4.5 mm in length. Females may contain embryonated or unembryonated eggs in utero. Fish-eating birds are the usual definitive host, but humans may harbor adults after consuming larvae in undercooked or raw fish.



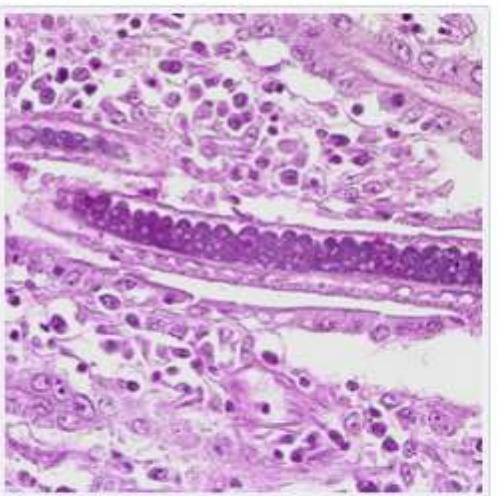
**Figure A:** Longitudinal section of an adult of *C. philippinensis* from an intestinal biopsy specimen stained with hematoxylin and eosin (H&E).



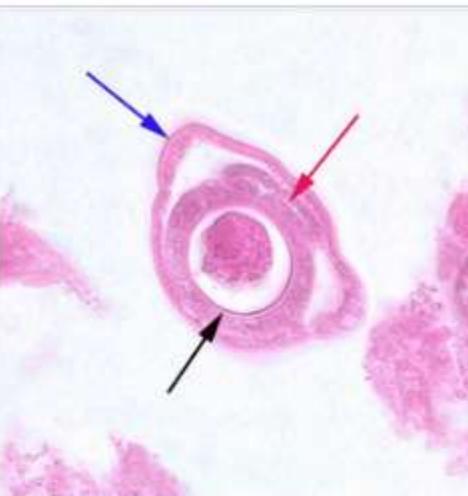
**Figure B:** Longitudinal section of an adult of *C. philippinensis* from an intestinal biopsy specimen stained with hematoxylin and eosin (H&E).



**Figure C:** Longitudinal section of an adult *C. philippinensis* from an intestinal biopsy specimen, stained with H&E.



**Figure D:** Higher magnification of **Figure C**, showing stichocytes within the adult worm.



**Figure E:** Cross-section of a gravid adult female *C. philippinensis* from an intestinal biopsy specimen, stained with H&E. Shown in this figure are a bacillary band (**blue arrow**), the intestine (**red arrow**) and uterus containing an egg in cross-section (**black arrow**).

# Capillariasis

- Etiologic Agents: *C. philippinensis*
- Source: Raw fish, bird reservoir
- Clinical Manifestations: Profuse watery diarrhea with massive malabsorption, emaciation, abdominal pain, eosinophilia.
- Pathology: Invasion and inflammation of intestinal mucosa. Local maturation and autoinfection.
- Laboratory Diagnosis: Eggs in stool, sometimes adult worms.
- Epidemiology: Mainly Philippines & Thailand. Sporadic: Egypt, Korea, Taiwan, India, Egypt, Japan.
- Treatment: High dose albendazole for prolonged periods.
- Prevention and Control: Avoid raw fish.

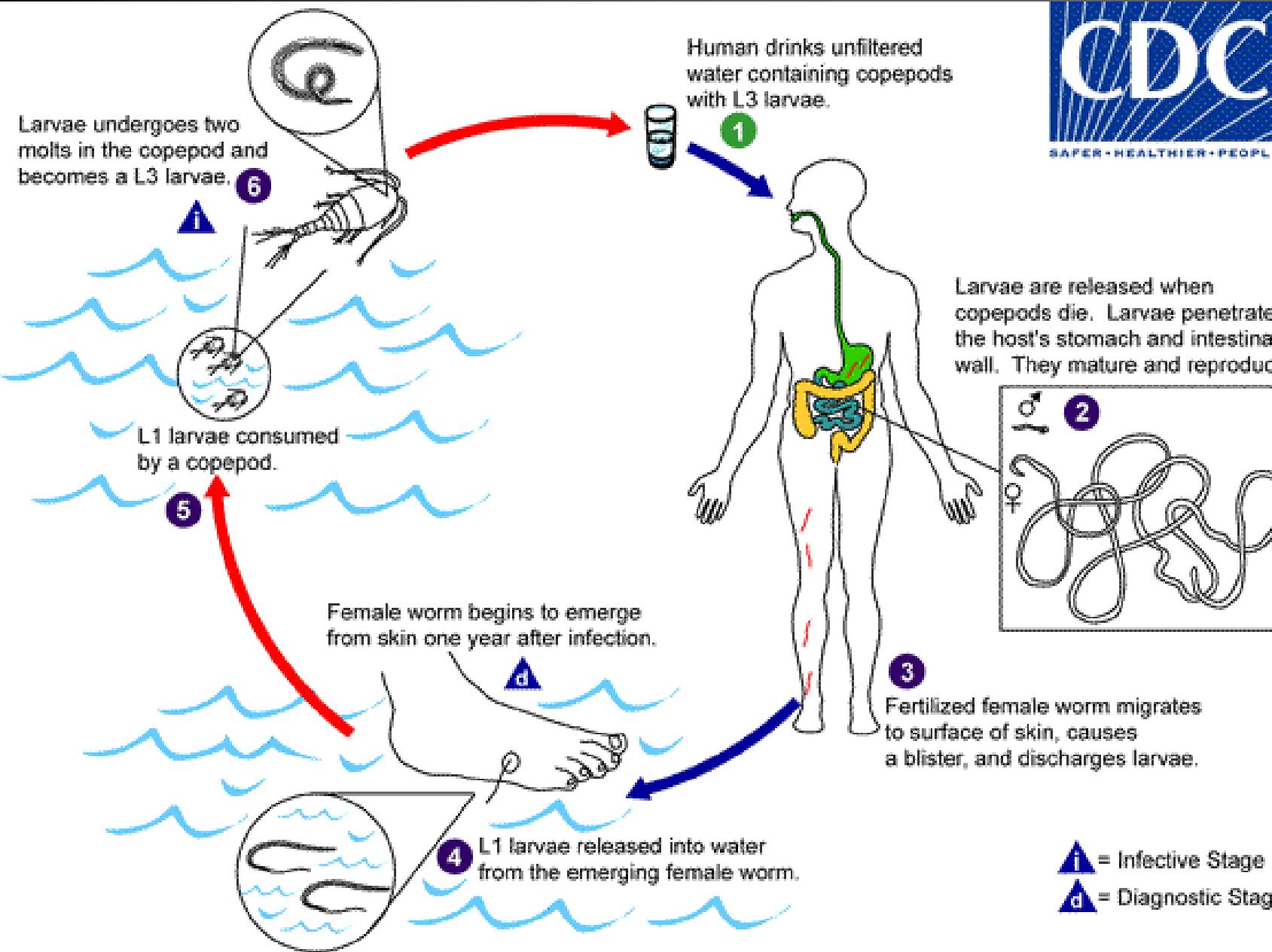
# Dracunculiasis

Goal 1

Eradicate  
extreme  
poverty and  
hunger



- Helmintiasis como NTDs (Enfermedades Desatendidas u Olvidadas)
- Enfermedades Olvidadas, Desatendidas, de la Pobreza (*neglected tropical diseases*)
  - 13 infecciones bacterianas y parasitarias:
    1. Ascariasis
    2. Anquilostomiasis
    3. Trichuriasis
    4. Filariasis linfática
    5. Oncocercosis
    6. Dracunculiasis
    7. Esquistosomiasis
    8. Enfermedad de Chagas
    9. Tripanosomiasis Africana Humana
    10. Leishmaniasis
    11. Ulcera de Buruli
    12. Lepra (Enf. de Hansen)
    13. Tracoma



The female Guinea worm induces a painful blister.



After rupture of the blister, the worm emerges as a whitish filament in the center of a painful ulcer which is often secondarily infected. (Images contributed by Global 2000/The Carter Center, Atlanta, Georgia).

TABLE  
54.1**Characteristics of Filarial Parasites and Guinea Worm and Common Clinical Manifestations in Humans**

Species	Distribution	Vectors	Main Location of Adult Worms	Main Location of Microfilariae	Common Disease Symptoms
<i>Wuchereria bancrofti</i>	Tropics	Mosquito spp.	Lymphatic vessels	Blood	Lymphangitis, elephantiasis hydrocele
<i>Brugia malayi</i>	South and South-east Asia	Mosquito spp.	Lymphatic vessels	Blood	Lymphangitis, elephantiasis
<i>Brugia timori</i>	Eastern Indonesia, Timor Leste	Mosquito spp.	Lymphatic vessels	Blood	Lymphangitis, elephantiasis
<i>Loa loa</i>	Central and West Africa	Chrysops spp.	Connective tissue	Blood	Angioedema, "eye worm"
<i>Mansonella perstans</i>	Africa, Central and South America	Culicoides spp.	Serous membranes of body cavities	Blood	Usually symptomless
<i>Mansonella streptocerca</i>	Central and West Africa	Culicoides spp.	Skin	Skin	Usually symptomless
<i>Mansonella ozzardi</i>	Central and South America	Culicoides spp. <i>Simulium</i> spp.	Serous membranes of body cavities	Blood and skin	Usually symptomless
<i>Onchocerca volvulus</i>	Africa, Yemen, Central and South America	<i>Simulium</i> spp.	Skin	Skin	Rash, pruritus, papules, skin atrophy, nodules, visual impairment and blindness
<i>Dracunculus medinensis</i>	Africa	Copepods	Connective tissue, including skin	Not applicable	Pain, ulceration, emerging worm

2. *Cyclops* die and release  
the larvae into stomach.  
Larvae develop, mature  
and reproduce. after  
10–14 months, female  
worms emerge

case containment



1. Individual drinks  
unfiltered water  
containing *Cyclops* with  
ingested larvae

surveillance



health education



3. Infected man enters water  
ponds. Larvae are released into  
the water



vector control



2. *Cyclops* swallow the  
larvae and undergo two  
moult to become infective



5. Individual collects  
water containing infected  
*Cyclops*

access to improved  
water source

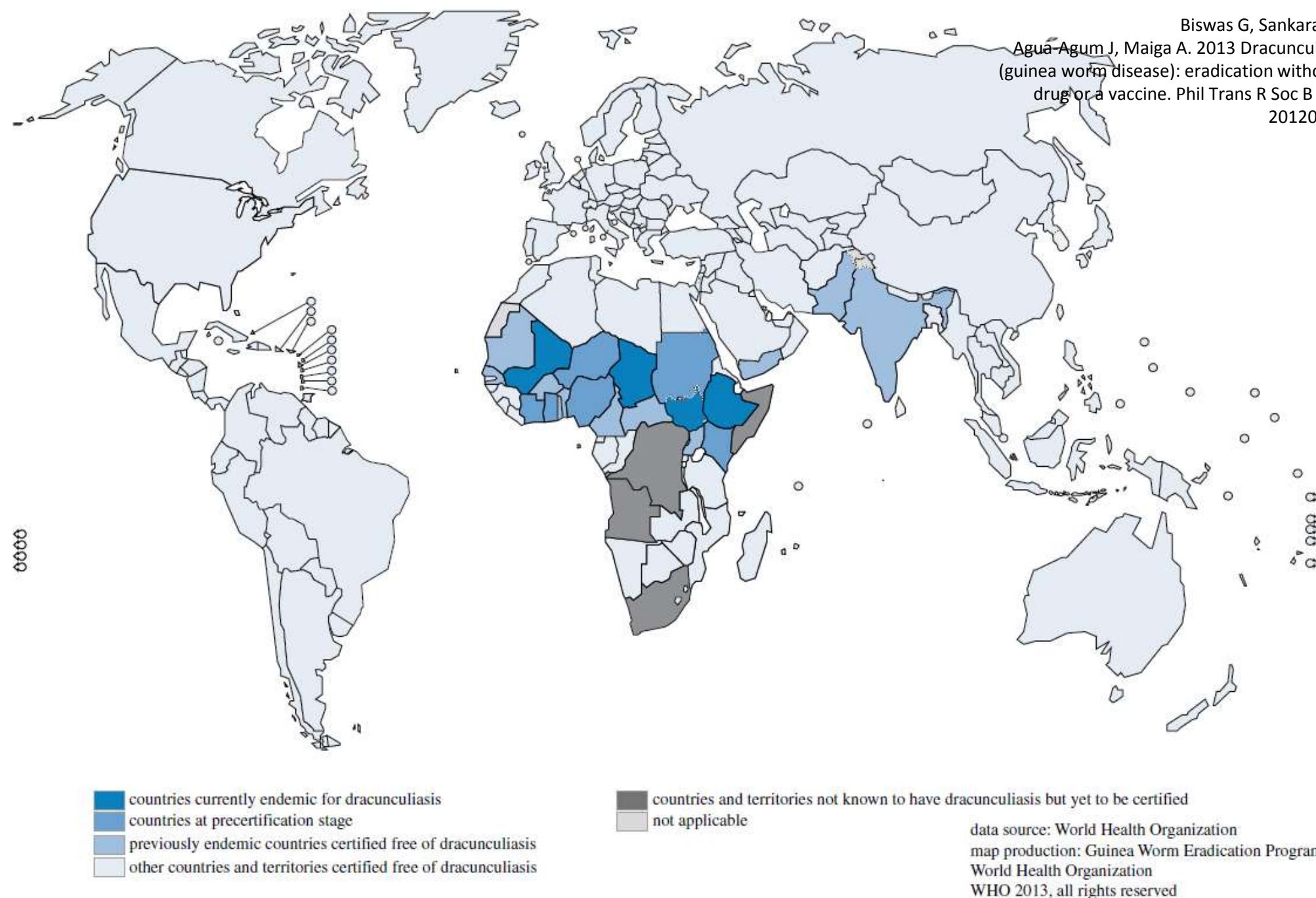
Figure 1. Guinea worm life cycle and interventions to interrupt transmission [9].



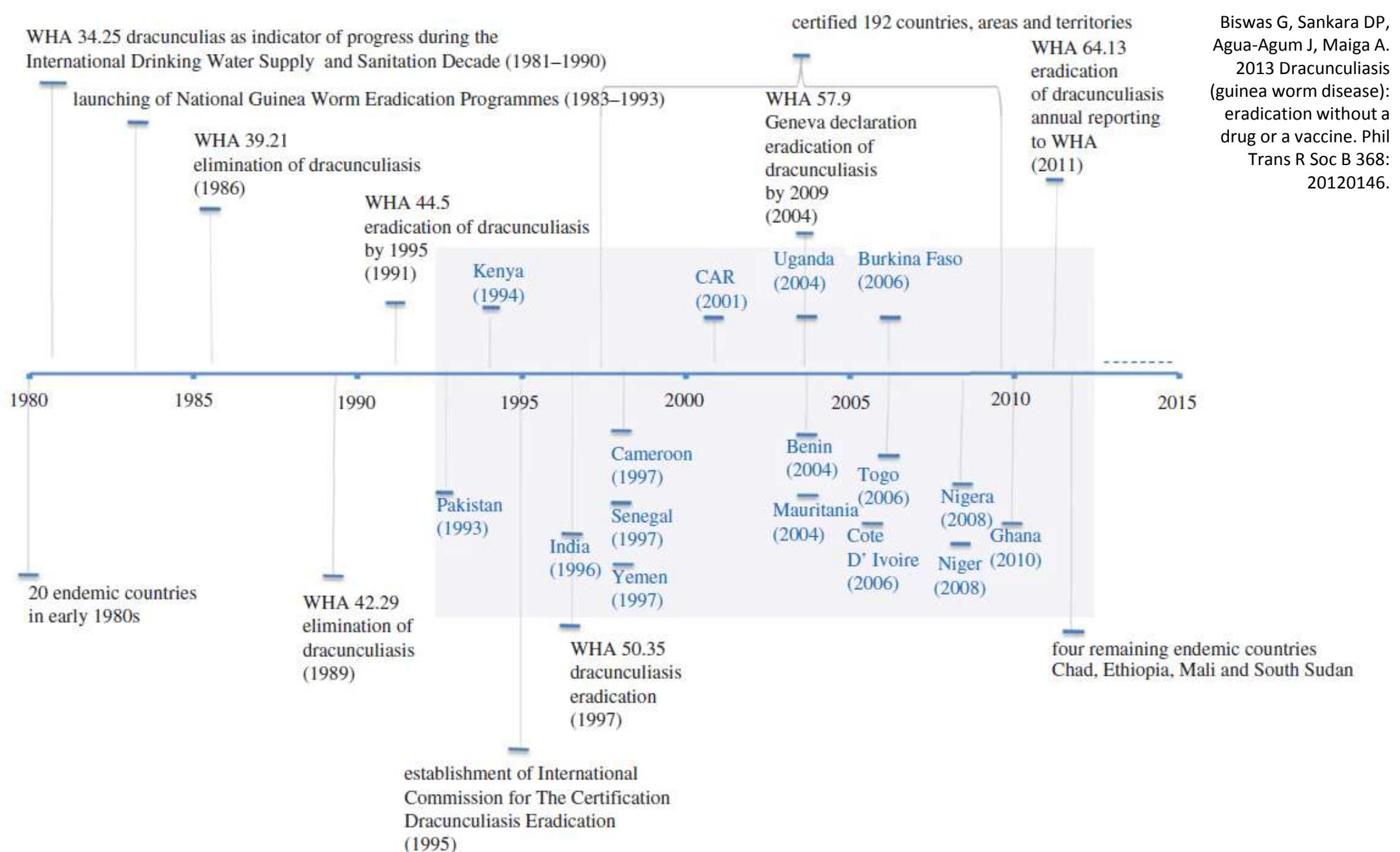
**Figure 54.38** Guinea worm intervention: drinking using a handheld water filter. (Courtesy of The Carter Center.)

**Table 1.** World Health Assembly resolutions on dracunculiasis.

<b>World Health Assembly</b>	<b>year</b>	<b>main focus</b>
WHA 34.25	1981	International Drinking Water Supply and Sanitation Decade: dracunculiasis as indicator of progress [32]
WHA 39.21	1986	endorsed combined strategy: safe water provision, active surveillance, health education, community mobilization, vector control personal prophylaxis [10]
WHA 42.29	1989	declared goal for eliminating dracunculiasis in the 1990s, invited other development agencies, organizations, foundation to support the country and ensured funding [33]
WHA 44.5	1991	country by country certification of elimination of dracunculiasis, certification by WHO. National goals to interrupt transmission by 1995 [11]
WHA 50.35	1997	political support and availability of resources for completion of dracunculiasis and support the work of International Commission for the Certification of Dracunculiasis Eradication (ICCDE) [34]
WHA 57.9	2004	Geneva Declaration for the Eradication of Dracunculiasis by 2009 [35]
WHA 64.16	2011	provision of adequate resources for interrupting transmission and certification of eradication of disease, supporting surveillance in dracunculiasis-free areas, annual reporting of the progress to WHA [36]



**Figure 4.** Endemicity status of certification. (Online version in colour.)



**Figure 3.** Dracunculiasis eradication timeline. The shaded box indicates the respective years (in parenthesis) in which countries interrupted transmission. (Online version in colour.)

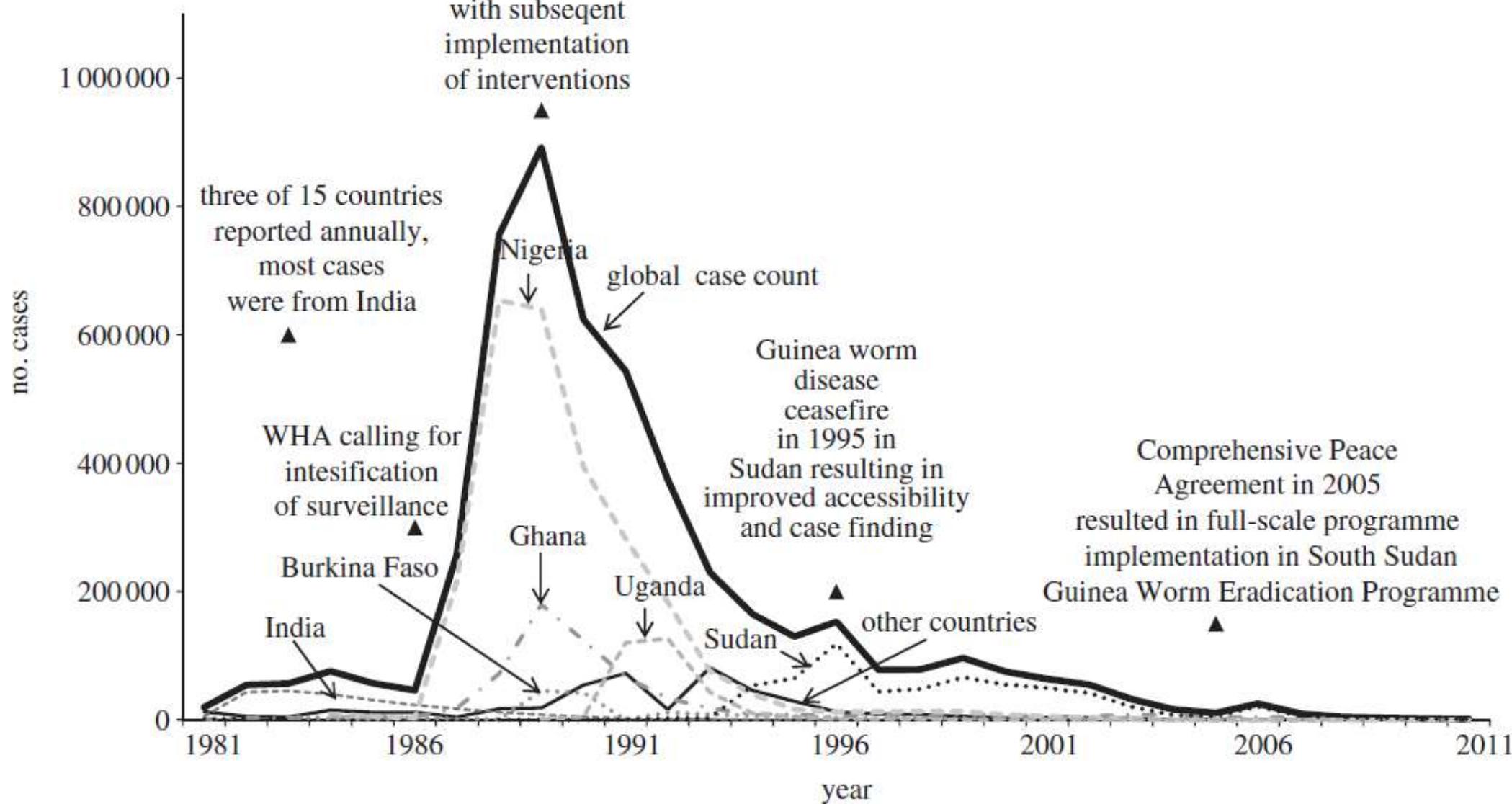


Figure 2. Dracunculiasis cases reported worldwide, 1981–2011 [80].

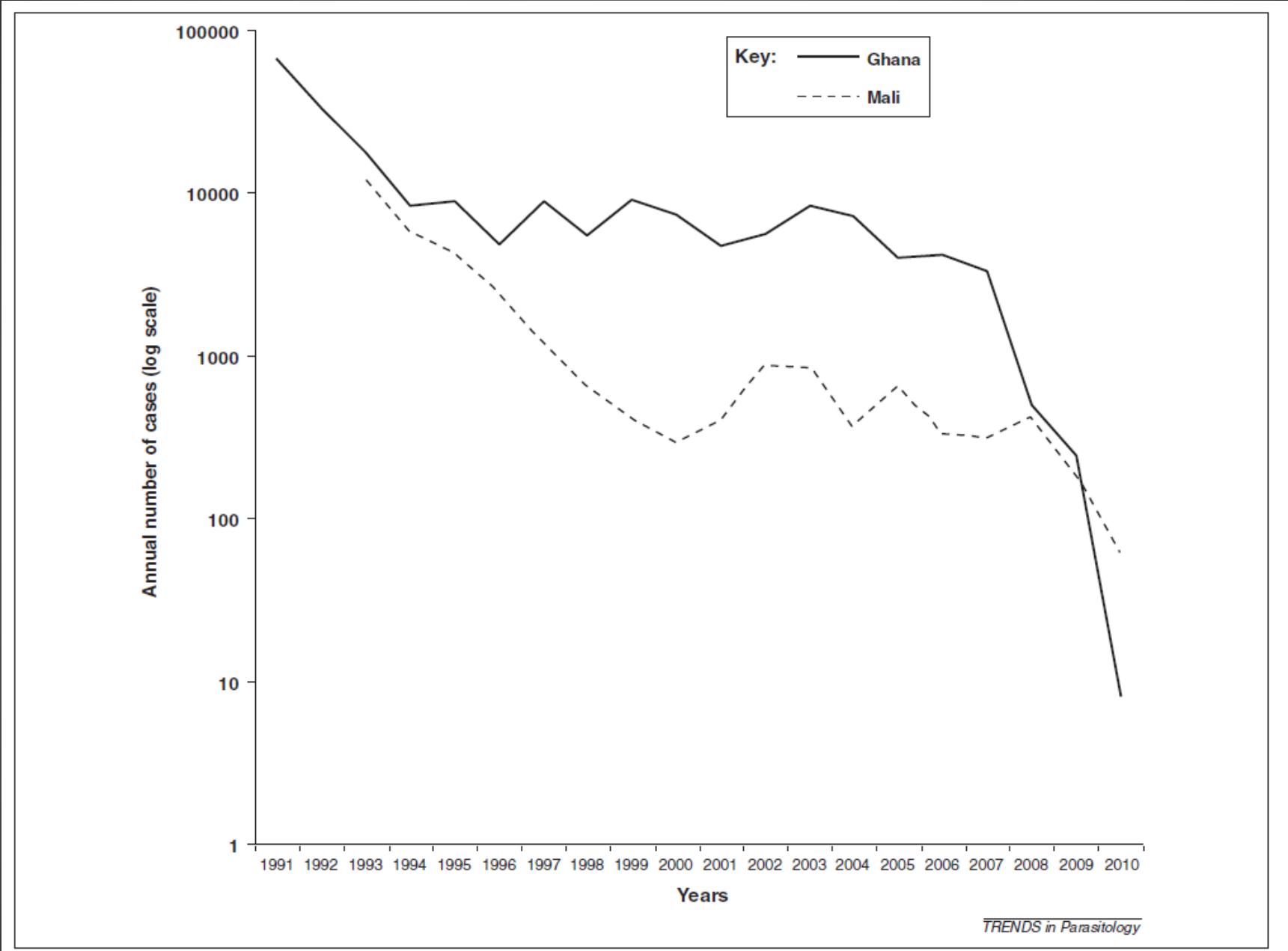


Figure 1. Examples of stagnation: the number of dracunculiasis cases reported annually in Ghana and Mali, 1991–2010. Once they had set up national eradication programmes, most other endemic countries did better than this, achieving annual reductions of the order of 50% in most years [2].



**Figure 54.37** Traditional method for removing a guinea worm. (Courtesy of The Carter Center.)

Diagnóstico clínico.  
No se emplea tratamiento antiparasitario específico.



**Figure 54.36** Blister on the skin related to an emerging guinea worm. (Courtesy of P. Bloch.)

# Dracunculiasis

- Etiologic Agents: *Dracunculus medinensis*
- Source: Ingestion of water containing infected copepods.  
1-Year maturation.
- Clinical Manifestations: Painful blister on leg with systemic symptoms. Release of larvae with freshwater contact.  
Bacterial superinfection
- Pathology: Acute inflammation due to dying worms.
- Laboratory Diagnosis: none
- Epidemiology: Sudan and West Central Africa.
- Treatment: Supportive only. Keep wound clean.
- Prevention and Control: Larvicide, clean water, keep actively infected people out of the water.

## DERMATOSIS DE ORIGEN ALIMENTARIO AL REGRESO DE UN VIAJE: GNATHOSTOMIASIS

TOMÁS A. ORDUNA, SUSANA C. LLOVERAS, SOFÍA E. ECHAZARRETA, SANTIAGO L. GARRO,  
GUSTAVO D. GONZÁLEZ, CLAUDIA C. FALCONE

*Servicio de Patologías Regionales y Medicina Tropical (CEMPRA-MT),  
Hospital de Infecciosas F. J. Muñiz, Buenos Aires*

**Resumen** Se describe el caso de un paciente de 32 años de edad, residente en Buenos Aires, con manifestaciones dermatológicas compatibles con gnathostomiasis. Había realizado un viaje a Colombia en el mes previo a la aparición de la sintomatología. Allí consumió cebiche (pescado crudo marinado en jugo de limón) en reiteradas oportunidades. El cuadro clínico se presentó como paniculitis eritematosa y migratoria acompañada de eosinofilia sanguínea. Se le realizó biopsia cutánea de una lesión y el diagnóstico anatomo-patológico fue “paniculitis eosinofílica”. La tríada de paniculitis migratoria, eosinofilia sanguínea y el consumo de pescado crudo durante el viaje a Colombia fue sugestiva de gnathostomiasis por lo que se indicó tratamiento con ivermectina con buena evolución inicial y recaída posterior. Se realizó un nuevo tratamiento con la misma droga con buena evolución y sin recaídas durante tres años de seguimiento. La afección dermatológica es un motivo frecuente de consulta al regreso de un viaje, y representa la tercera causa de morbilidad en viajeros. Es muy importante el reconocimiento de las enfermedades que pueden tener manifestación cutánea, ya que muchas de ellas son potencialmente graves y pueden poner en riesgo la vida del paciente si no son oportunamente diagnosticadas y tratadas.

del mismo por la infrecuencia de presentación de pacientes con esta afección.

*Tomás A. Orduna, Susana C. Lloveras, Sofía E. Echazarreta, Santiago L. Garro, Gustavo D. González, Claudia C. Falcone*

Servicio de Patologías Regionales y Medicina Tropical (CEMPRA-MT), Hospital de Infectosas F. J. Muñiz, Buenos Aires, Argentina  
e-mail: torduna@intramed.net

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#### ¿Hay gnathostomiasis en Colombia? Reflexiones a partir de un posible caso importado a la Argentina

Hemos leído con interés el artículo de Orduna y col.<sup>1</sup>, sobre un caso importado de gnathostomiasis diagnosticado en Buenos Aires, Argentina, que tenía como antecedente haber realizado 20 días atrás un viaje a "varias ciudades de la costa caribeña de Colombia". Al respecto quisieramos hacer algunas consideraciones y complementar, especialmente desde la perspectiva epidemiológica, de salud pública y de medicina del viajero las implicaciones de dicho reporte.

Concordamos en que el diagnóstico de esta zoonosis parasitaria transmitida por alimentos incluye la triada de la eosinofilia, lesiones migratorias y una clara exposición en zonas de riesgo. Dicho riesgo de exposición incluye el vivir o viajar a una zona endémica y consumo de alimentos que potencialmente contengan las formas larvarias del parásito (incluido pescado crudo)<sup>2</sup>. Sin embargo, es de hacer notar que clínicamente los principales diagnósticos diferenciales incluyen a la angiostrongiliasis, la trichinosis y el síndrome de larva migrans cutánea<sup>3</sup>. En ese sentido lo que más nos llama la atención del caso reportado en Buenos Aires es que el paciente haya probablemente adquirido la infección en Colombia.

La gnathostomiasis no se considera endémica en Colombia, formalmente hablando. En América Latina se consideran endémicos México, Ecuador y Perú<sup>2</sup>. Aun cuando, ciertamente, en otros países de la región como Brasil y Argentina se han reportado algunos casos previamente<sup>1,4</sup>. En Colombia solo se ha informado previamente un caso en el cual se diagnosticó la enfermedad en un agricultor de Antioquia (departamento del norte) con costas en el mar Caribe) que había hecho un paseo en río en el municipio de Unguia (departamento de Chocó), que tiene costas en el golfo de Urabá (mar Caribe) y es

fronterizo con la Comarca Emberá-Wounaan y la Provincia de Darién de Panamá<sup>5</sup>. Aparte de ello no hay otros reportes en la literatura sobre estudios o casos adicionales de gnathostomiasis en Colombia. Tanto en el caso diagnosticado en Colombia<sup>2</sup>, como en el recientemente diagnosticado en la Argentina, procedente de nuestro país<sup>1</sup>, el diagnóstico es solo presuntivo o probable, pero no confirmado. En ninguno de los dos se aisló e identificó la larva a partir de muestras de las lesiones (lo cual es difícil, impráctico y no se suele emplear), ni se realizaron técnicas serológicas como la ELISA o el inmunoíblot para detectar la banda específica de 24-kDa<sup>1,2</sup>. Debe mencionarse además, que ya se está trabajando en el desarrollo de nuevos antígenos recombinantes con el uso de técnicas de biología molecular<sup>6</sup>, que ayudarán a mejorar su diagnóstico etiológico.

Por todo ello nos surge la inquietud sobre la posible transmisión de *Gnathostoma* spp. en Colombia. ¿Podrían decir que existe, o más aún, que es endémico en Colombia? Con la excepción de los estados de Tamaulipas (fronterizo con EE.UU.), Veracruz y Tabasco, de México, y los casos informados de Colombia<sup>1,4</sup>, no existen otras áreas en el mar Caribe donde se hayan reportado casos de gnathostomiasis. Sin embargo, por su importancia epidemiológica, e incluso por la posibilidad de hacer estudios serológicos, hubiese sido de gran importancia precisar las zonas del Caribe colombiano donde estuvo el paciente diagnosticado en la Argentina. Los departamentos de Colombia con costa o en el mar Caribe incluyen: Antioquia, Atlántico, Bolívar, Cesar, Córdoba, La Guajira, Magdalena, San Andrés y Providencia, y Sucre. En esta región se incluyen ciudades de gran atractivo turístico no solo nacional, sino también internacional, como Barranquilla, Cartagena de Indias y Santa Marta, entre otras. Por lo cual la exposición en turistas y viajeros es otro aspecto a tomar en cuenta como consecuencia de este caso y la presente discusión.

Este artículo<sup>1</sup> sirve en todo caso para reflexionar acerca de la posibilidad de ocurrencia de esta enfermedad en el Caribe colombiano y la necesidad de investigar más al respecto para conocer su situación real, así como en la importancia del desarrollo y disponibilidad de pruebas diagnósticas específicas, para esta y otras parasitosis que pueden ser de importancia para la salud pública y la medicina del viajero.

Alfonso J. Rodríguez-Morales, Erika Vanessa Cárdenas-Giraldo,  
Santiago Manrique-Castaño, Dayron Fernando Martínez-Pulgarín

Grupo y Semillero de Investigación  
Salud Pública e Infección  
Facultad de Ciencias de la Salud, Universidad  
Tecnológica de Pereira,  
Pereira, Risaralda, Colombia

e-mail: arodriguezm@utp.edu.co

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#### En respuesta: Sobre un posible caso de gnathostomiasis

Agradecemos la carta enviada por los Dres. Alfonso Rodríguez-Morales y col.<sup>1</sup>, que nos permite conocer un trabajo al que no habíamos accedido para nuestra publicación y que, como se menciona, representa el primer caso descripto en Colombia de posible gnathostomiasis<sup>2</sup>.

Estamos de acuerdo en que la falta de más casos reportados puede obedecer a una baja exposición al riesgo de la población colombiana en general y sobre todo de la zona litoral (tanto de región Caribe como de la región Pacífico) sumado a un potencial subdiagnóstico de casos interpretados bajo otros diagnósticos etiológicos.

En referencia a las ciudades visitadas por nuestro paciente, él mismo relató haber estado en Cartagena, Santa Marta y en la isla de San Andrés. Así mismo manifestó haber consumido cebiche en varias oportunidades y en diferentes locales.

Coincidimos en que se trata de un caso altamente probable, tal como se mencionara, por la epidemiología, cuadro clínico e histopatología de paniculitis eosinofílica migratoria, pero sin la confirmación diagnóstica por carecer de métodos serológicos específicos en nuestro país y la falta de hallazgo del parásito en la muestra de la biopsia realizada.

Por último, consideramos que la triquinosis o trichinelosis no debería incluirse en el diagnóstico diferencial de la gnathostomiasis, ya que si bien puede producir edemas localizados y migrañas, entre otros signos y síntomas, no hay expresión clínica de larva migrante cutánea o de paniculitis migratoria en esta zoonosis. En nuestro país, con brotes periódicos anuales de triquinosis, no hemos visto esta forma de presentación clínica<sup>3,4</sup>, lo que coincide con la guía de referencia de triquinelosis de la Organización Mundial de la Salud<sup>5</sup>.

*Tomás A. Orduna, Susana C. Lloveras, Sofía E. Echazarreta, Santiago L. Garro, Gustavo D. González, Claudia C. Falcone*

Servicio de Patologías Regionales y Medicina Tropical (CEMPRA-MT), Hospital de Infectosas F. J. Muñiz, Buenos Aires, Argentina  
e-mail: torduna@intramed.net

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#### El sistema peptídico natriurético y sus biomarcadores

En *Medicina (B Aires)* 2013, los Dres. Ogawa y de Bold publicaron un excelente Artículo Especial: "El corazón endocrino y el proceso inflamatorio"<sup>1</sup>. En él, los autores aceptan que la disfunción ventricular no es la única causa de activación del sistema peptídico natriurético. Otras (inflamatorias endocrinas, renales, hepáticas<sup>2</sup>, sepsis<sup>3</sup>) pueden alterar los niveles de estos péptidos. Se sugiere que la activación del sistema peptídico con elevación de sus biomarcadores (ANF, BNP, proBNP) es un indicador de mal pronóstico aun en enfermedades extracardiacas. En mi tesis doctoral expliqué la relación entre los niveles plasmáticos de BNP al ingreso con la mortalidad hospitalaria en 357 internaciones clínicas de pacientes con cáncer de pulmón, de mama y gastrointestinal. Fueron excluidos aquellos pacientes con sepsis, insuficiencia cardiaca, ictericia y falla renal. La mediana de BNP plasmático en los datos de alta fue 64 pg/ml (3-1669) y la de los fallecidos en la internación fue de 81 pg/ml (15-4000) ( $p = 0.0854$ ). Entre los 119 casos de cáncer de pulmón, la mediana de BNP fue de 63 pg/ml (3-1669) entre los datos de alta, y 87 pg/ml (25-675) en los fallecidos ( $p = 0.0635$ ). Por lo

La gnathostomiasis no se considera endémica en Colombia, formalmente hablando.

En América Latina se consideran endémicos México, Ecuador y Perú

Aun cuando, ciertamente, en otros países de la región como Brasil y Argentina se han reportado algunos casos previamente

**En Colombia solo se ha informado previamente un caso en el cual se diagnosticó la enfermedad en un agricultor de Antioquia (departamento del noroeste con costas en el mar Caribe) que había hecho un paseo en río en el municipio de Unguía (departamento de Chocó), que tiene costas en el golfo de Urabá (mar Caribe) y es fronterizo con la Comarca Emberá-Wounaan y la Provincia de Darién de Panamá.**

## golfo de Urabá



En Colombia solo se ha informado previamente un caso en el cual se diagnosticó la enfermedad en un agricultor de Antioquía (departamento del noroeste con costas en el mar Caribe) que había hecho un paseo en río en el municipio de Unguía (departamento de Chocó), que tiene costas en el golfo de Urabá (mar Caribe) y es fronterizo con la Comarca Emberá-Wounaan y la Provincia de Darién de Panamá.

## Comarca Emberá-Wounaan de Panamá

## Provincia de Darién de Panamá





**Figura 1.** En la parte inferior del abdomen se observa la primera lesión que sanó con fibrosis; en la parte superior la lesión más reciente que se extendía hacia atrás y de donde se tomó la biopsia.



**Figura 2.** Observación del corte histológico que muestra la reacción inflamatoria en la hipodermis.



**Figura 3.** Aproximación de la lesión tisular que muestra infiltrado por polimorfonucleares neutrófilos, linfocitos plasmocitos y eosinófilos en el tejido adiposo.

1. Orduna TA, Lloveras SC, Echazarreta SE, Garro SL, González GD, Falcone CC. Dermatosis de origen alimentario al regreso de un viaje: gnathostomiasis. *Medicina (B Aires)* 2013; 73: 558-61.
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3. Vargas TJ, Kahler S, Dib C, Cavalieri MB, Jeunon-Sousa MA. Autochthonous gnathostomiasis, Brazil. *Emerg Infect Dis* 2012; 18: 2087-9.
4. Kaminsky CA, De Kaminsky AR, Costantini SE, Abulafia J. Paniculitis nodular migratoria eosinofílica (gnathostomiasis humana). *Med Cutan Ibero Lat Am* 1989; 17: 158-62.
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6. Janwan P, Intapan PM, Yamasaki H, et al. Application of recombinant *Gnathostomaspinigerum* matrix metalloproteinase-like protein for serodiagnosis of human gnathostomiasis by immunoblotting. *Am J Trop Med Hyg* 2013; 89: 63-7.

### **En respuesta: Sobre un posible caso de gnathostomiasis**

Agradecemos la carta enviada por los Dres. Alfonso Rodríguez-Morales y col.<sup>1</sup>, que nos permite conocer un trabajo al que no habíamos accedido para nuestra publicación y que, como se menciona, representa el primer caso descripto en Colombia de posible gnathostomiasis<sup>2</sup>.

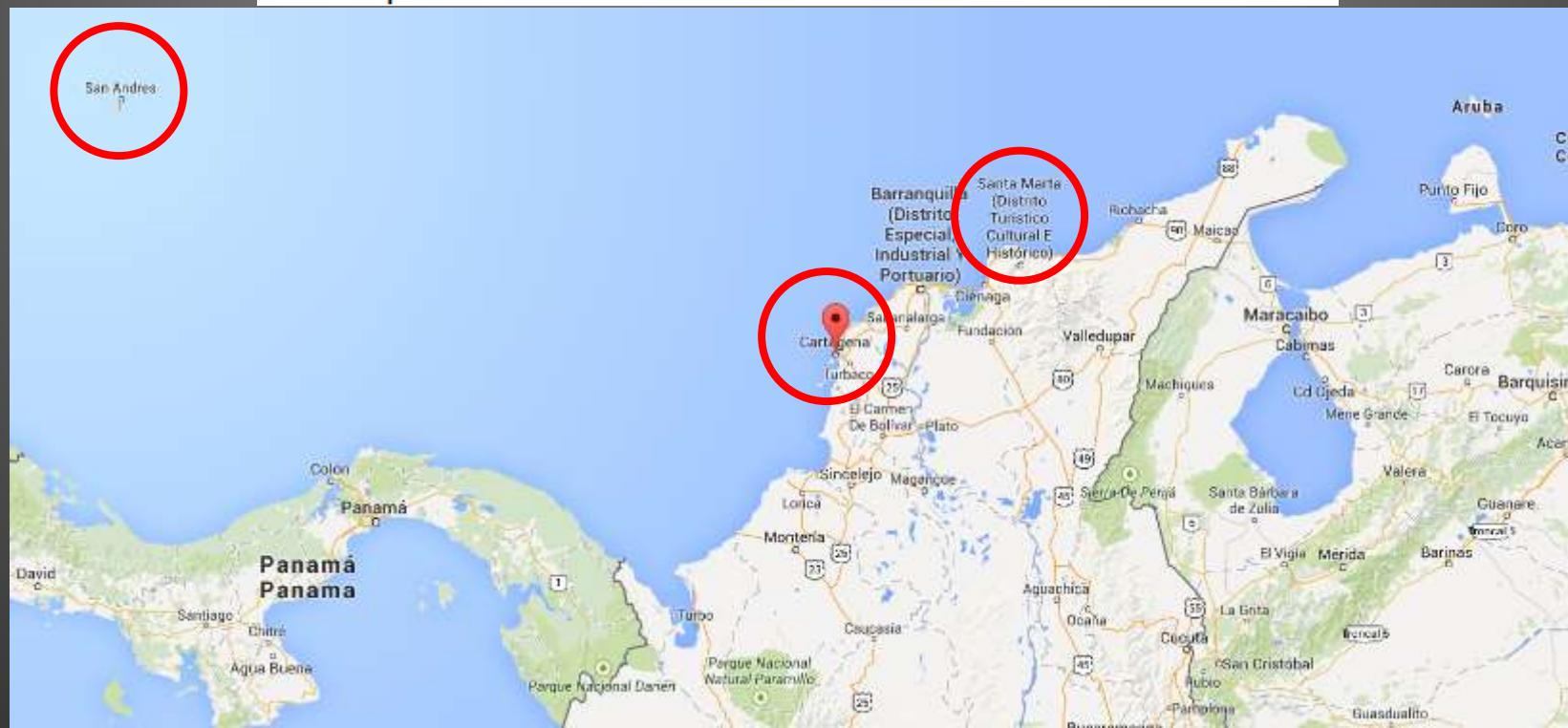
Estamos de acuerdo en que la falta de más casos reportados puede obedecer a una baja exposición al riesgo de la población colombiana en general y sobre todo de la zona litoraleña (tanto de región Caribe como de la región Pacífico) sumado a un potencial subdiagnóstico de casos interpretados bajo otros diagnósticos etiológicos.

*Tomás A. Orduna, Susana C. Lloveras, Sofía E. Echazarreta, Santiago L. Garro, Gustavo D. González, Claudia C. Falcone*  
Servicio de Patologías Regionales y Medicina Tropical (CEMPRA-MT), Hospital de Infectuosas F. J. Muñiz, Buenos Aires, Argentina  
e-mail: torduna@intramed.net

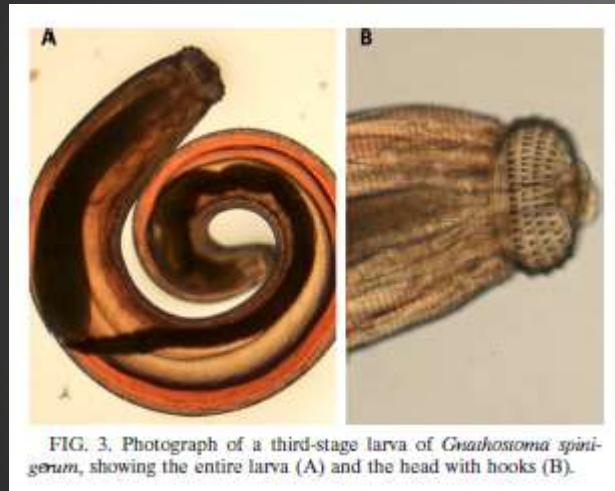
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En referencia a las ciudades visitadas por nuestro paciente, el mismo relató haber estado en Cartagena, Santa Marta y en la isla de San Andrés. Así mismo manifestó haber consumido cebiche en varias oportunidades y en diferentes locales.

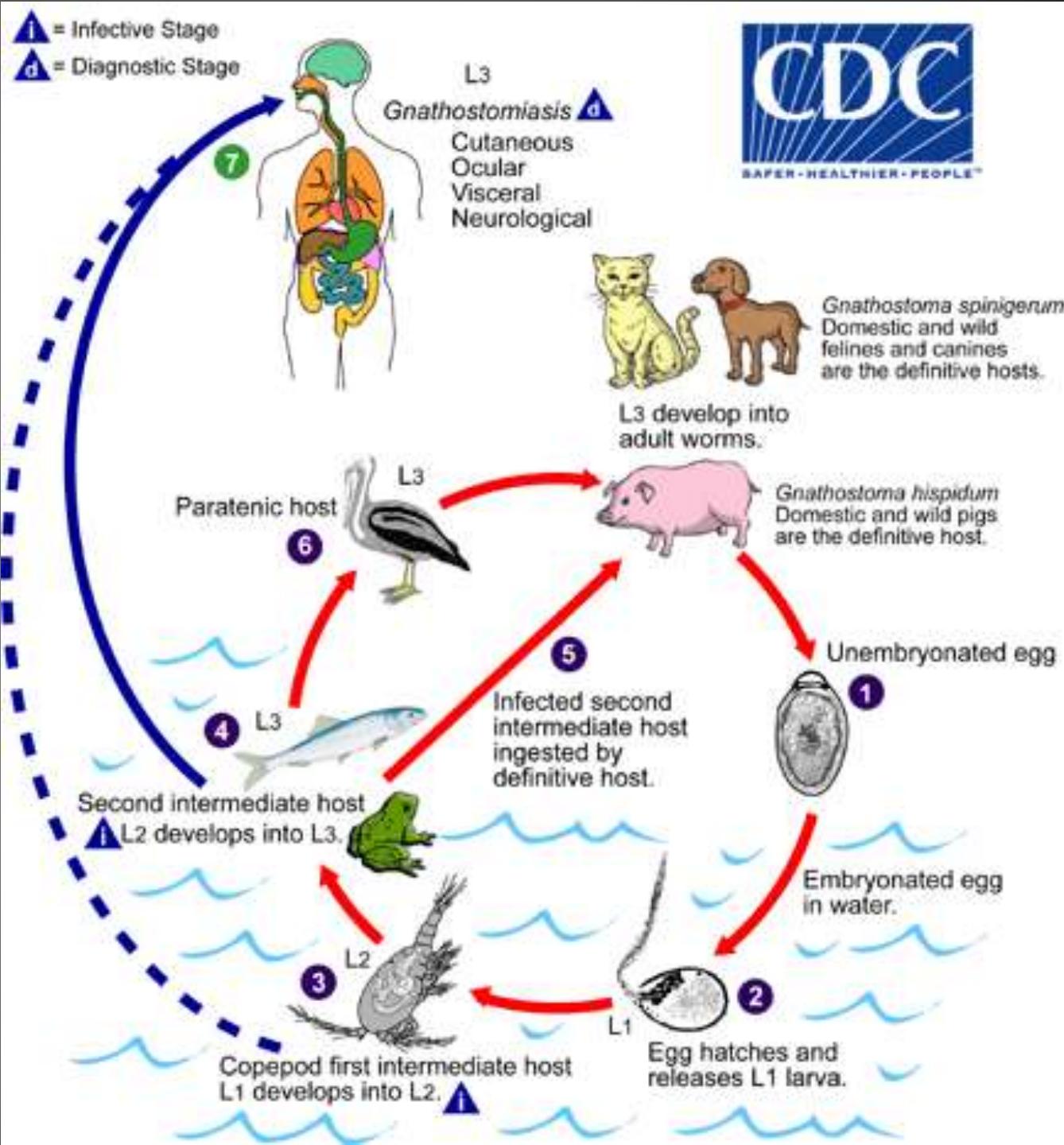
Coincidimos en que se trata de un caso altamente probable, tal como se mencionara, por la epidemiología, cuadro clínico e histopatología de paniculitis eosinofílica migratoria, pero sin la confirmación diagnóstica por carecer de métodos serológicos específicos en nuestro país y la falta de hallazgo del parásito en la muestra de la biopsia realizada.



# Gnathostomiasis



Herman JS, Chiodini PL.  
Gnathostomiasis, another emerging imported disease.  
Clin Microbiol Rev. 2009 Jul;22(3):484-92



## Gnathostomiasis, Another Emerging Imported Disease

Joanna S. Herman<sup>1\*</sup> and Peter L. Chiodini<sup>1,2</sup>

Department of Clinical Parasitology, Hospital for Tropical Diseases, 3rd Floor, Mortimer Market, Capper Street, London WC1E 6JB, United Kingdom,<sup>1</sup> and London School of Hygiene and Tropical Medicine, London, United Kingdom<sup>2</sup>

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### INTRODUCTION

International travel to the tropics has dramatically increased over the past few decades, with a subsequent and significant increase in the number of patients presenting with tropical diseases in countries where such infections are not endemic. It is estimated that 50 million residents of industrialized countries travel annually to such areas (42, 44), which brings exposure to a broad range of pathogens rarely, if ever, encountered at home. These may vary from short-lived, easily detected and treatable infections (e.g., gastrointestinal infections) to more exotic infections such as filarial and helminthic infections (e.g., loiasis, strongyloidiasis, and schistosomiasis). Such infections may be seen rarely by physicians in temperate climates, and therefore diagnosis can prove elusive if these infections are not considered. Travelers are becoming ever more adventurous in choice of country, pursuit of remoteness and immersion in local culture, which will frequently include eating all local delicacies (e.g., ceviche or “drunken crab”) without consideration of what organisms they might be harboring. Migration has also increased substantially over the past few decades, with people from the tropics and subtropics settling in the West, and many come harboring parasites of which they are unaware.

Gnathostomiasis is a parasitic infection caused by the third-stage larvae of the helminths *Gnathostoma* spp., which are seen mostly in tropical and subtropical regions. It is a food-borne zoonosis and is endemic in areas where people eat raw freshwater fish or shellfish, especially Thailand and other parts of

Southeast Asia, Japan, and increasingly Latin America, particularly Mexico. Previously, the disease was rarely seen outside areas of endemicity; however, over the past decade, the number of cases seen in countries where it is not endemic has increased. Few clinicians outside areas of endemicity are familiar with the disease, and therefore diagnosis is often missed or prolonged, with potentially serious consequences. The classic triad of intermittent migratory swellings, eosinophilia, and a history of travel to Southeast Asia or other areas of endemicity should alert physicians to the possible diagnosis. Visceral disease is more serious than the cutaneous manifestations and, in the case of central nervous system (CNS) disease, may be fatal.

This article describes the epidemiology, life cycle, clinical features, diagnostic tools, treatment, and prevention of this disease.

### EPIDEMIOLOGY

*Gnathostoma* was first discovered in the stomach wall of a tiger that died at London Zoo in 1836 (35) and was first described in humans in 1889 in Thailand by G. M. R. Levinson (cited in references 5 and 24). The next case was not described until 1934, and shortly after, its life cycle was elucidated (37).

The foci of endemicity have been predominantly in Japan and Southeast Asia, particularly Thailand, but the disease is also endemic in Cambodia, Laos, Myanmar, Indonesia, Philippines, and Malaysia (Fig. 1). Cases have also been reported in China, Sri Lanka, and India (41). In more recent years it has become an increasing problem in Central and South America, particularly in Mexico (due to the consumption of ceviche [raw fish marinated in lime]) (12, 39), and also in Guatemala, Peru, and Ecuador (14, 23). There have also reports of cases in Myanmar, Zambia, and, most recently, Botswana (6, 16, 17).

Glasgow, Escocia, Reino Unido, 2010



\* Corresponding author. Mailing address: Department of Clinical Parasitology, Hospital for Tropical Diseases, 3rd Floor, Mortimer Market, Capper Street, London WC1E 6JB, United Kingdom. Phone: 020 7387 4411, ext. 5418. Fax: 020 7383 0041. E-mail: joherman@doctors.org.uk.

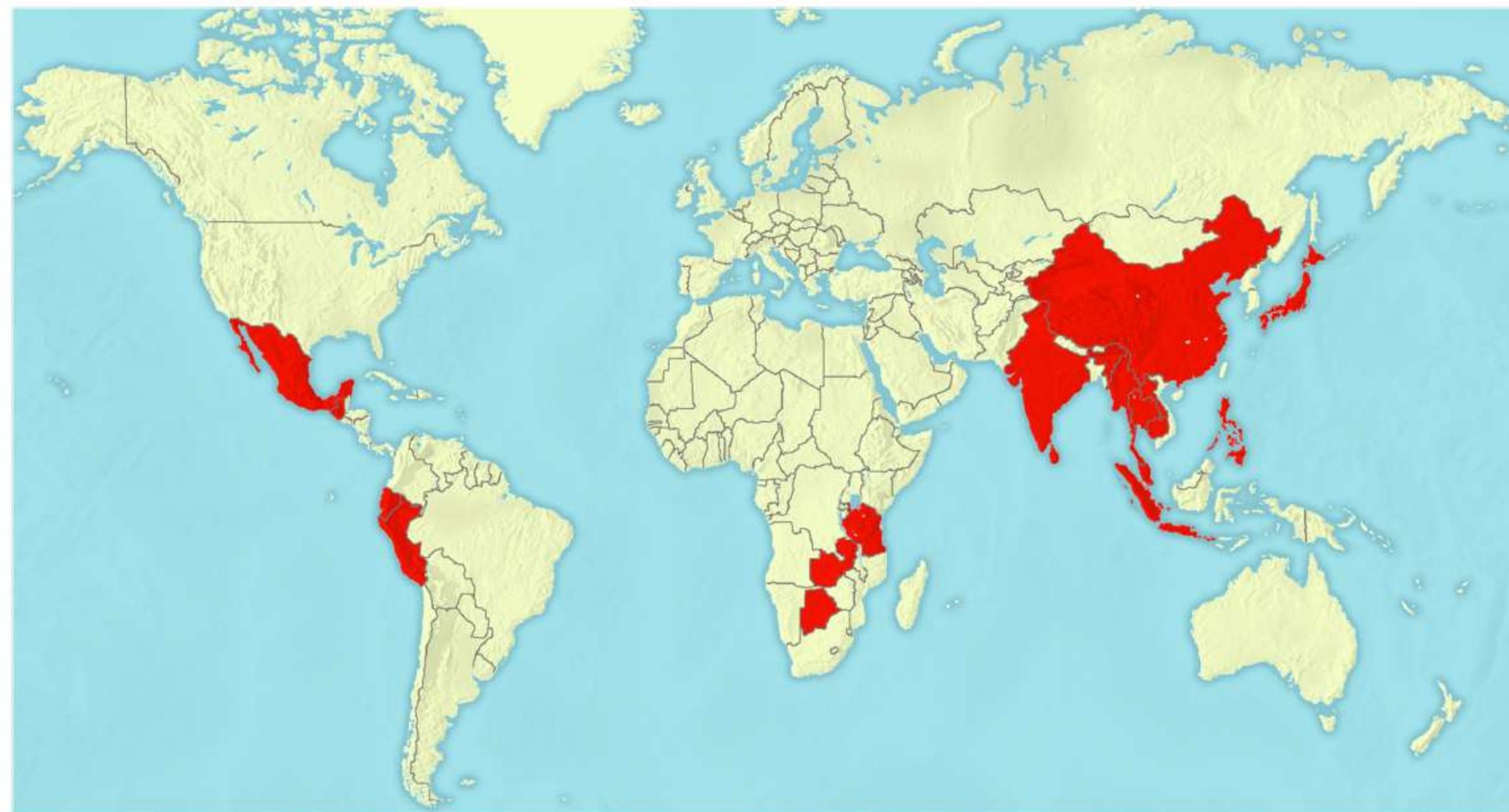


FIG. 1. Map of countries with reported acquisition of gnathostomiasis.

# Gnathostomiasis



**Etiologic Agents:** *Gnathostoma spinigerum*

**Source:** Ingestion of immature larvae in intermediate hosts: fish, amphibians, reptiles, birds, and mammals (low specificity of larvae for 2nd intermediate hosts). Adults in dogs/cats.

**Clinical Manifestations:** Subcut. migratory swellings, creeping eruption, myeloencephelitis, cerebral bleeding, conjunctival edema.

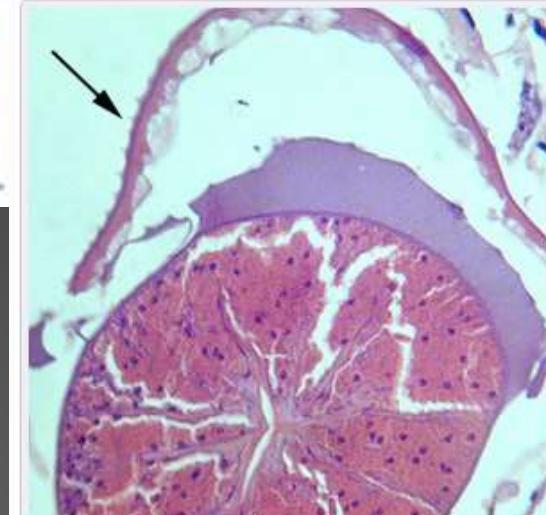
**Pathology:** Track like necrosis with hemorrhage along path of the nematode. Eosinophilic infiltration in subcutaneous lesions.

**Laboratory Diagnosis:** Serology available only in SE Asia.

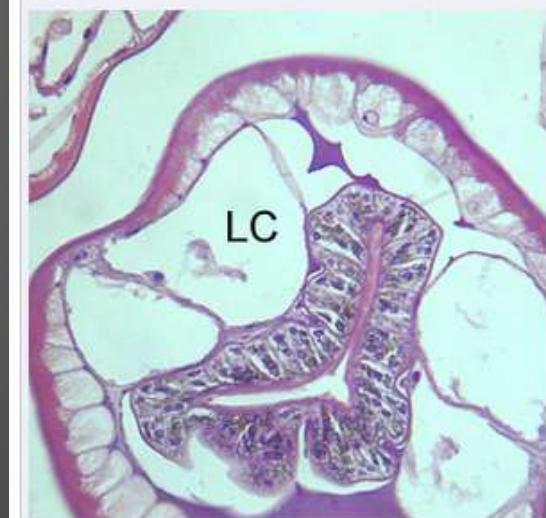
**Epidemiology:** Mainly Thailand and Japan but reported from other Asian countries and Australia. Emerging in Mexico, Peru, Ecuador

**Prevention and Control:** Cooked food.

**Treatment:** Mainly surgical removal; increasing experience with Albendazole (X21d) and ivermectin



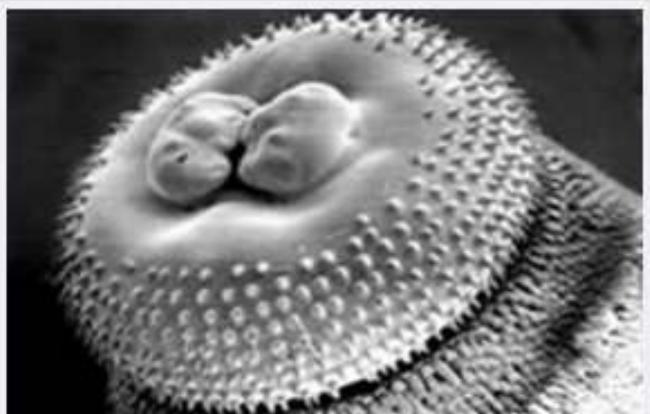
Hematoxylin and eosin (H&E) stained cross-section of *Gnathostoma* sp., taken from a subcutaneous nodule above the right breast of a patient, showing the esophagus. Note the presence of cuticular spines (arrow). Image courtesy of Diagnostix Pathology Laboratories LTD, Canossa Hospital, Hong Kong, China.



Another H&E-stained cross-section of *Gnathostoma* sp., taken of the same specimen in above figure showing the intestinal cells and characteristic large lateral chords (LC). Note the multinucleate intestinal cells and the presence of pigmented granular material in the intestinal cells.

▼ **Head bulb and cuticular spines.**

Scanning electron micrographs of a *Gnathostoma spinigerum* female worm depicting the cuticular armature of the body surface. The cuticular armature is important for identification of *Gnathostoma* spp.



**Figure A:** Head bulb.

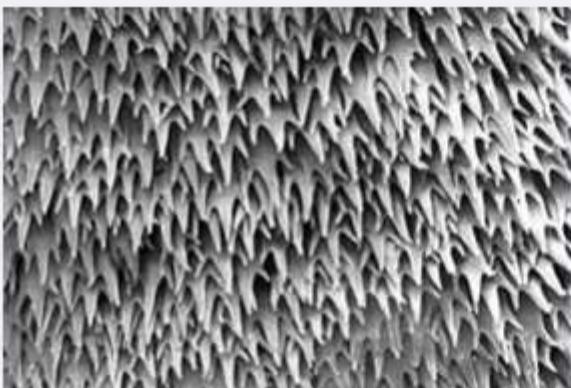


**Figure B:** Cuticular spines of the posterior body part.

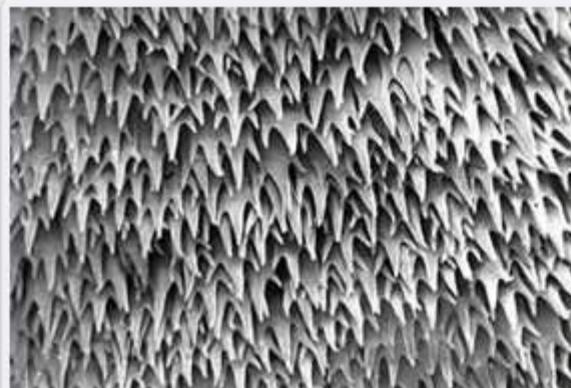
**Reference**

Scholtz T, Ditrich O. Scanning electron microscopy of the cuticular armature of the nematode *Gnathostoma spinigerum* Owen, 1836 from Cats in Laos. J Helminthol 1990 Sep;64 (3):255-262.

▼ Detail of cuticular spines of the anterior body part.



**Figure A:** Detail of cuticular spines of the anterior body part.

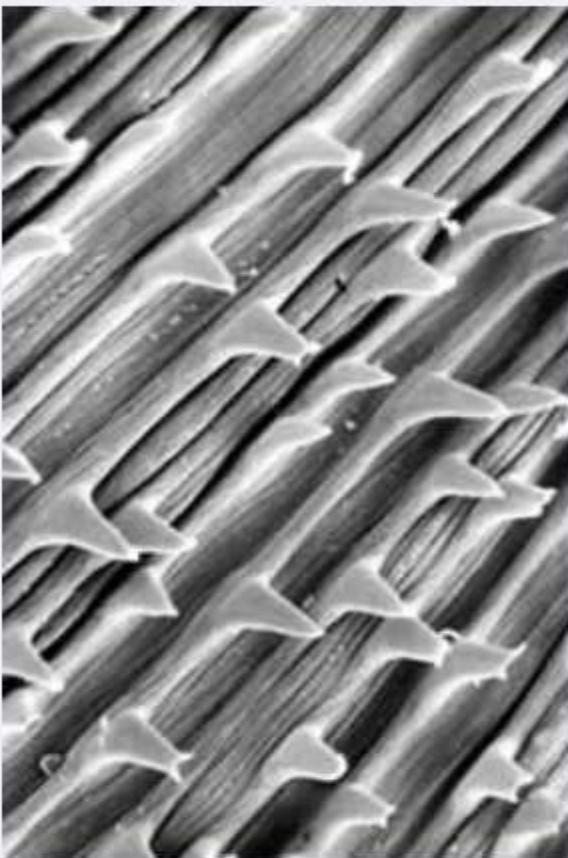


**Figure B:** Detail of cuticular spines of the anterior body.

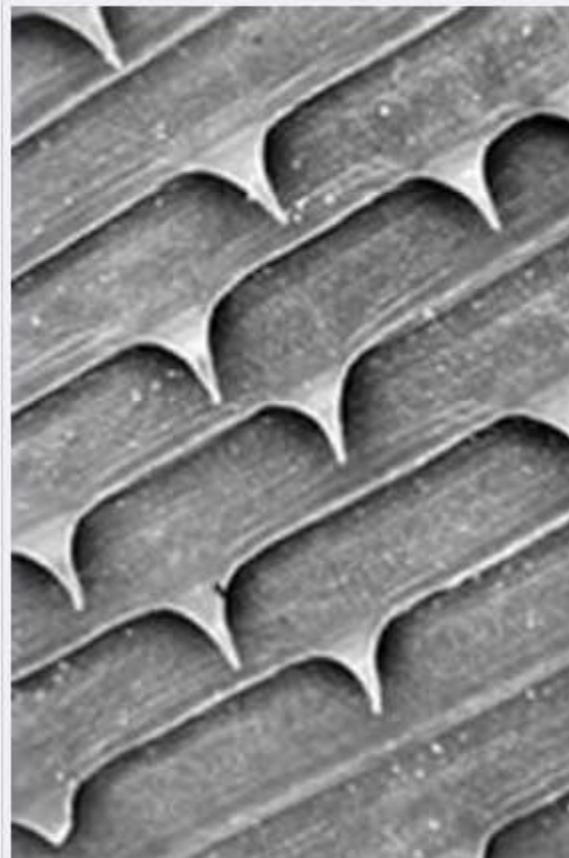
**Reference**

Scholtz T, Ditrich O. Scanning electron microscopy of the cuticular armature of the nematode *Gnathastoma spingerum* Owen, 1836 from Cats in Laos. J Helminthol 1990 Sep;64 (3):255-262.

▼ Detail of nondendiculated cuticular spines.



**Figure A:** Detail of nondendiculated cuticular spines.



**Figure B:** Detail of nondendiculated cuticular spines.

**Reference**

Scholtz T, Ditrich O. Scanning electron microscopy of the cuticular armature of the nematode *Gnathostoma spinigerum* Owen, 1836 from Cats in Laos. J Helminthol 1990 Sep;64 (3):255-262.

**TABLE  
71.3**

## Causes of Seizures/Epilepsy

Infections	Bacterial meningitis Cerebral malaria TB meningitis Cryptococcal meningitis Neurocysticercosis Schistosomiasis Cerebral hydatid disease Paragonimiasis Cerebral toxoplasmosis Cerebral amoebiasis  Neuroangiostrongyliasis Gnathostomiasis Baylisascariasis
Focal brain lesions	Tetanus (pseudoepilepsy) Tumours, cysts, granulomas, tuberculoma
Toxins/drugs/metabolic	Alcohol, opiates, altered glucose levels
Metabolic Stroke	Hypoglycaemia Haemorrhage, intracerebral/ subarachnoid

**TABLE  
71.4** **Helminths that May Infect the Central Nervous System**

Organism	Main Areas of Distribution	Presentation	Management
<b>CESTODES</b>			
Neurocysticercosis	Latin America, sub-Saharan Africa, India	Seizures, mass lesion, hydrocephalus	Albendazole 15 mg/kg/day in 2 divided doses for 8 days. Cover with corticosteroids (see text)
<i>E granulosus</i> (Hydatid disease)	Worldwide	Mass lesion, seizures	Surgical resection. Pretreat: albendazole 15 mg/kg/day in 2 divided doses (40 days) to shrink cyst. If unresectable: albendazole 10–15 mg/kg/day in two divided doses twice a day ≥ 3 months
<i>Taenia multiceps</i> (coenurosis)	Worldwide	Hydrocephalus	Surgical removal
<i>Spirometra</i> sp. (Sparganosis)	Far East and S-e Asia, East Africa	Seizures, infarcts	Surgical removal
<b>NEMATODES</b>			
<i>Angiostrongylus cantonensis</i>	S-e Asia, Caribbean, Southern USA	Meningoencephalitis	Repeated LPs to reduce ICP. Corticosteroids + Albendazole 10–15 mg/kg/day in two divided doses – 2 weeks
<i>Gnathostoma spinigerum</i>	S-e Asia, Mexico, Ecuador, Japan	Meningoencephalitis, seizures, myeloradiculopathy	Albendazole 10–15 mg/kg/day in 2 divided doses – 21 days + corticosteroids. Surgical removal if accessible
<i>Onchocerca volvulus</i>	West Africa, Yemen, Latin America	Chorioretinitis, keratitis, seizures	Ivermectin single oral doses of 0.15 mg/kg single oral dose
<i>Baylisascaris procyonis</i>	Worldwide	Meningoencephalitis	Corticosteroids, albendazole 10–15 mg/kg/day in 2 divided doses
<i>Trichinella</i> sp.	Worldwide	Myopathy, strokes, meningoencephalitis	Corticosteroids, repeated LPs
<b>TREMATODES</b>			
Schistosomiasis	Africa, Asia, Brazil	cauda equina/conus syndrome/ cerebral granuloma	<i>S. mansoni</i> , <i>S. haematobium</i> and <i>S. intercalatum</i> – Praziquantel 40 mg/kg/day – 3 days, corticosteroids (see text) <i>S. japonicum</i> and <i>S. mekongi</i> – Praziquantel 60 mg/kg/day – 3 days, corticosteroids (see text)
<i>Paragonimus</i> sp.	Latin America, Asia, West Africa	Encephalitis, mass lesion, infarcts, seizures, myelopathy	Praziquantel 25 mg/kg three times a day – 3 days, corticosteroid
<i>Fasciola hepatica</i>	Worldwide	Meningitis, mass lesion, infarct	Triclabendazole 10 mg/kg single dose or 20 mg/kg in two divided doses

S-e, South-east; LP, lumbar puncture; ICP, intracranial pressure; meningoencephalitis in this table refers to an eosinophilic meningoencephalitis.



**Figure 71.5** (A) Gnathostomiasis; larval tracks evident on brain and spinal cord MRI. (B) Gnathostomiasis; larvae can be surgically removed if in an accessible site (see arrow). (Courtesy of R Shakir & N Poungvarin.)

## Case Report: Intraocular Gnathostomiasis: Report of a Case and Review of Literature

Gopal S. Pillai, Anil Kumar,\* Natasha Radhakrishnan, Jayasree Maniyelil, Tufela Shafi,  
Kavitha R. Dinesh, and Shamsul Karim

*Departments of Ophthalmology, Microbiology, and Pathology, Amrita Institute of Medical Sciences, Ponekara, Kochi, Kerala, India*

### INTRAOCULAR GNATHOSTOMIASIS

621



FIGURE 1. Third-stage larvae of *G. spinigerum* showing transparent white globular heads are easily distinguishable from the rest of the body, which are black in color with a rounded posterior end.



FIGURE 3. Longitudinal section (hematoxylin/eosin) of the anterior end showing four rows of hooklets and lateral spines. Magnification: 400 $\times$ .



FIG. 4. Photograph showing cutaneous larva migrans due to *Gnathostoma spinigerum* on the forehead (A) and shoulder (B). (Reprinted from reference 17.)

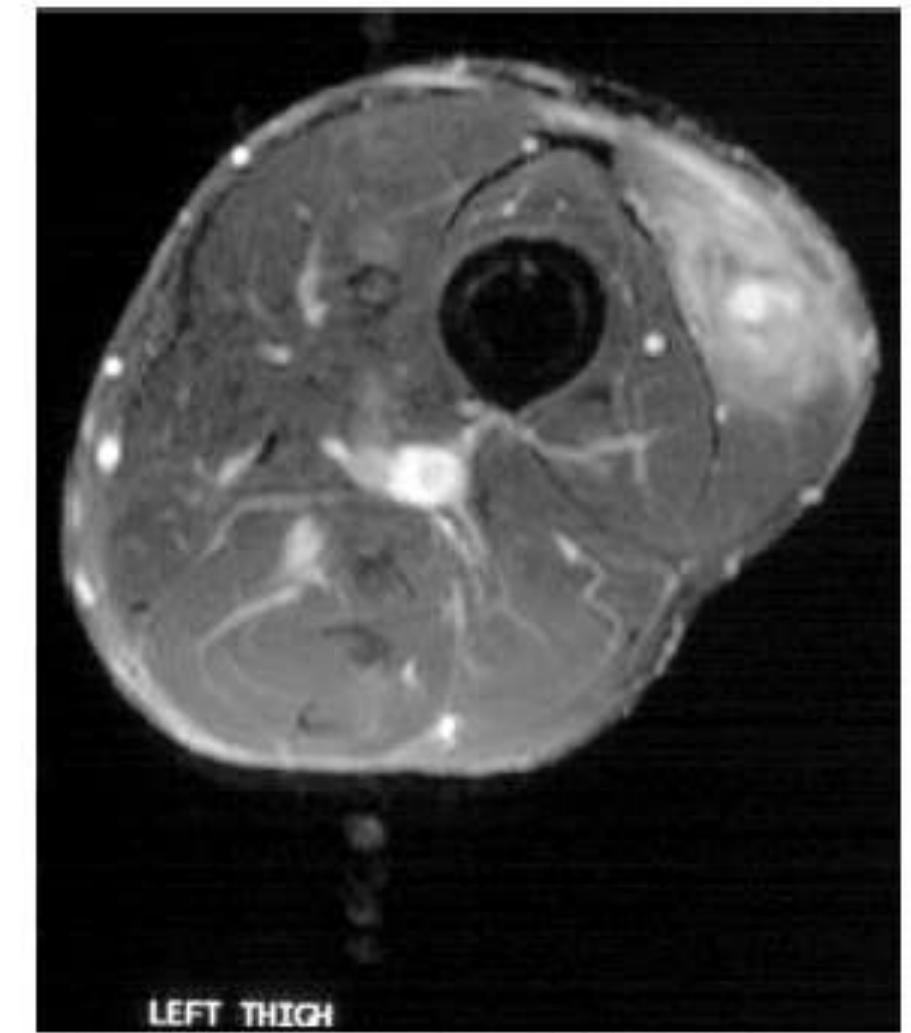


FIG. 5. Magnetic resonance image of thigh with *Gnathostoma* sp. larva. (Reprinted from reference 25.)

Herman JS, Chiodini PL.

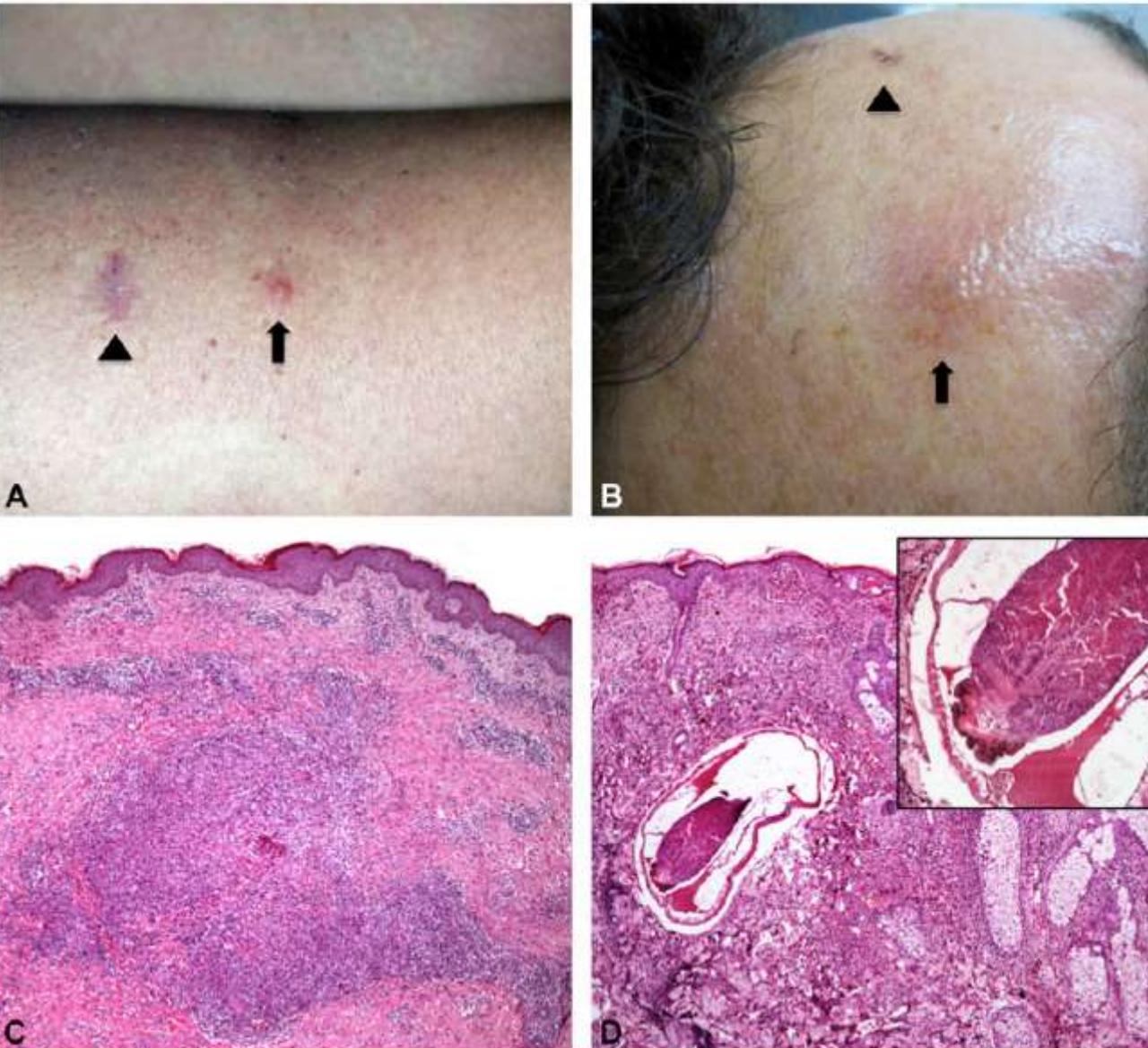
Gnathostomiasis, another emerging imported disease.

Clin Microbiol Rev. 2009 Jul;22(3):484-92

# Paniculitis migratoria

## CAPSULE SUMMARY

- Gnathostomiasis is a parasitic infestation difficult to confirm on random skin biopsy specimen because of the migratory nature of the larva.
- Prescribing oral treatment may stimulate development of a papule or pseudofuruncle containing the larva.
- Biopsy of a papule or pseudo furuncle subsequent to oral treatment increases the likelihood of demonstrating the larva on skin biopsy specimen, which allows definitive diagnosis and may have therapeutic benefit.



**Fig 1.** Migratory panniculitis of gnathostomiasis. **A** and **B**, Initial biopsy sites (arrowheads) and subsequent papules developing after oral treatment (arrows). **C**, Dense dermal and subcutaneous inflammatory infiltrate with numerous eosinophils noted on initial biopsy specimens before treatment. **D**, Section of late-stage larva of *Gnathostoma* species identified when obtaining biopsy specimen of papule or pseudofuruncle developed after oral treatment. (**C** and **D**, Hematoxylin-eosin stains; original magnifications: **C** and **D**,  $\times 40$ ; **D** inset,  $\times 400$ .)



**Fig 2.** *Gnathostoma* species. Intact late-stage larva was recovered by patient from pustule that developed subsequent to oral treatment.

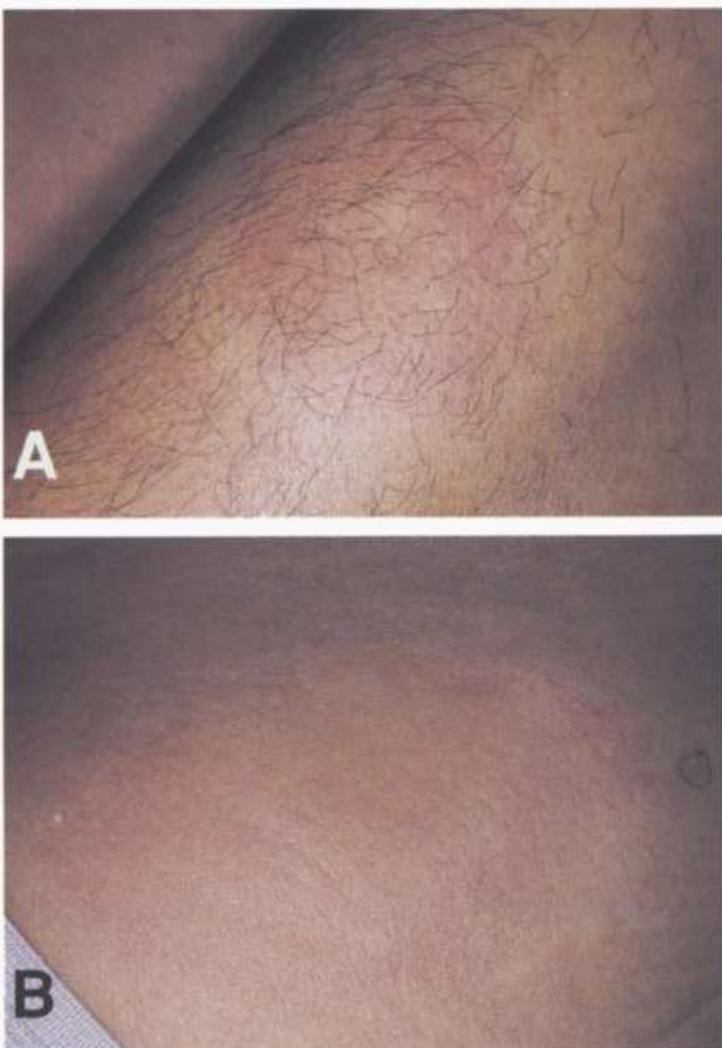
**Table I.** Demographic and clinical characteristics of 6 patients with cutaneous gnathostomiasis

Patient no.	Age, y	Gender	Clinical course*	Exposure	Lesion site	Treatment	Time to positive biopsy, d <sup>†</sup>
1	39	M	1 mo	Raw fish	Face	Ivermectin	1
2	43	M	2 mo	Raw fish	Right side of abdomen	Ivermectin	73
3	44	F	1 mo	Raw fish	Right thigh	Albendazole	7
4	45	F	6 mo	Raw fish	Right thigh	Albendazole	30
5	42	F	10 d	Raw fish	Left flank	Albendazole	1
6	12	F	1 mo	Raw fish	Left lateral aspect of thorax	Albendazole	14

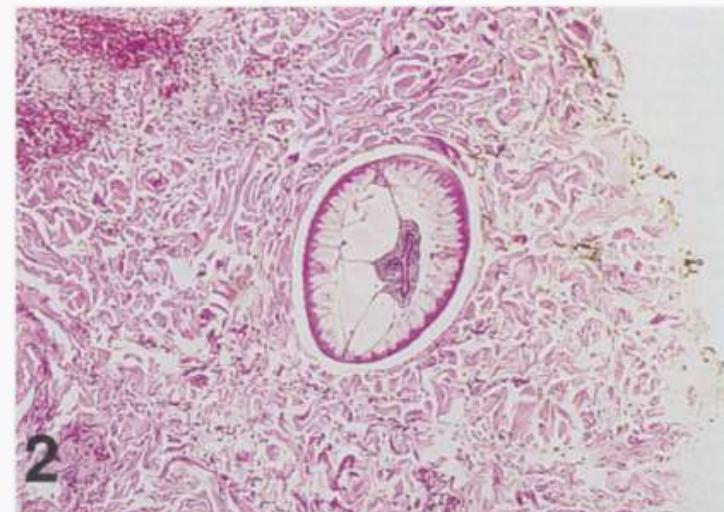
*F*, Female; *M*, male.

\*Symptomatic period before medical treatment.

†Time elapsed between initiation of treatment (coincident with initial biopsy specimen) and second (positive) biopsy specimen.



**Fig. 1. A,** Erythematous indurated plaque on thigh of patient. **B,** Erythematous indurated plaque on trunk of patient with "pseudopod" extending beyond the plaque (*circled in photograph*).



**Fig. 2.** Cross-section of nematode. (Hematoxylin-eosin stain;  $\times 100$ .)

#### Cutaneous gnathostomiasis

Jeffrey J. Crowley, MD, and Youn H. Kim, MD *Palo Alto, California*

From the Department of Dermatology, Stanford University School of Medicine.

Reprint requests: Youn H. Kim, MD, Stanford University Medical Center, Department of Dermatology, 900 Blake Wilbur Dr. W0064, Palo Alto, CA 94304.

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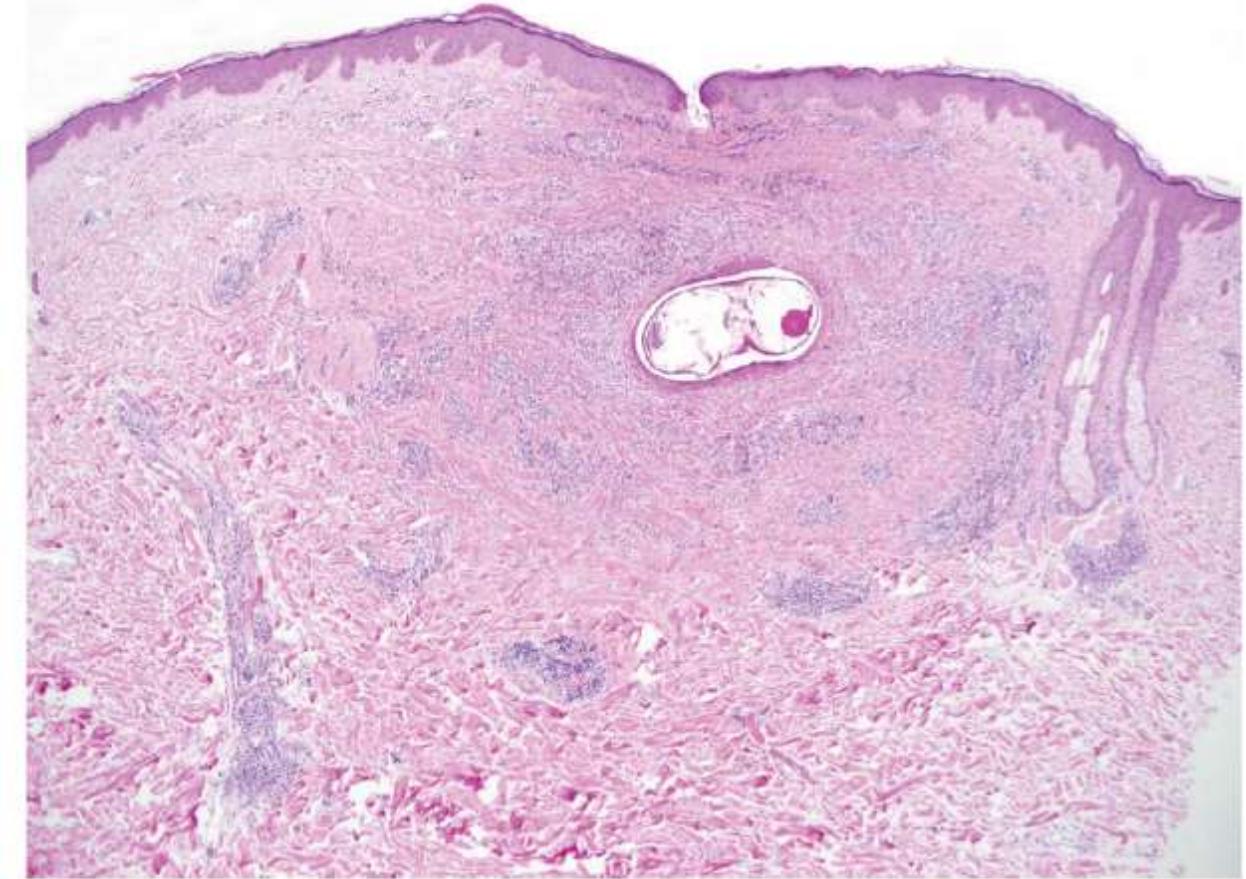


EXTRAORDINARY CASE REPORT

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## Gnathostomiasis in a Patient Who Frequently Consumes Sushi

*Abel D. Jarell, MD,\*† Michael J. Dans, MD, PhD,\* Dirk M. Elston, MD,‡ Blaine A. Mathison, BS,§ and Beth S. Ruben, MD\*†*



**FIGURE 1.** Histological sections show skin with a dense mixed inflammatory infiltrate and a necrotic zone surrounding a large intradermal organism (hematoxylin and eosin, original magnification,  $\times 40$ ).



**FIGURE 3.** The parasite demonstrated prominent lateral chords, a well-developed muscular esophagus, and an intestine with a brush border and multinucleate cells (hematoxylin and eosin, original magnification,  $\times 200$ ).



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Parasitology International 52 (2003) 133–140



[www.elsevier.com/locate/parint](http://www.elsevier.com/locate/parint)

## Acute outbreak of gnathostomiasis in a fishing community in Sinaloa, Mexico

Sylvia Paz Díaz Camacho<sup>a</sup>, Kaethe Willms<sup>b,\*</sup>, Ma. del Carmen de la Cruz Otero<sup>a</sup>, Magda Luz Zazueta Ramos<sup>a</sup>, Sergio Bayliss Gaxiola<sup>a</sup>, Rafael Castro Velázquez<sup>a</sup>, Ignacio Osuna Ramírez<sup>a</sup>, Angel Bojórquez Contreras<sup>a</sup>, Edith Hilario Torres Montoya<sup>a</sup>, Sergio Sánchez González<sup>a</sup>

<sup>a</sup>Facultad de Ciencias Químico Biológicas, Universidad Autónoma de Sinaloa, Culiacán, Sinaloa 80000, Mexico

<sup>b</sup>Departamento de Microbiología y Parasitología, Facultad de Medicina, Universidad Nacional Autónoma de México, Mexico DF 04510, Mexico

Received 25 July 2002; accepted 8 January 2003

## Serum antibodies to *Gnathostoma*

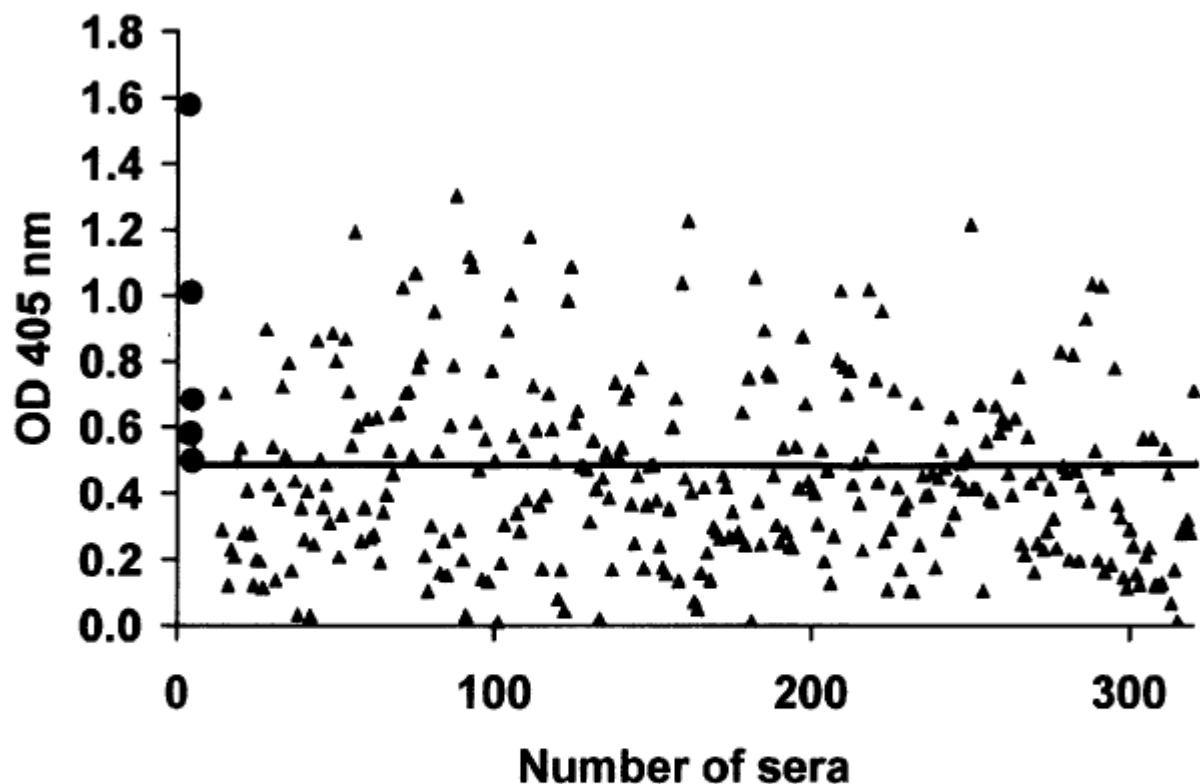


Fig. 1. Results of ELISA to *Gnathostoma doloresi* antigen in 314 individuals from Tecualilla, Escuinapa, Sinaloa. Black triangles correspond to 309 sera from random sample subjects. Black circles are readings obtained in five patients with acute symptoms.

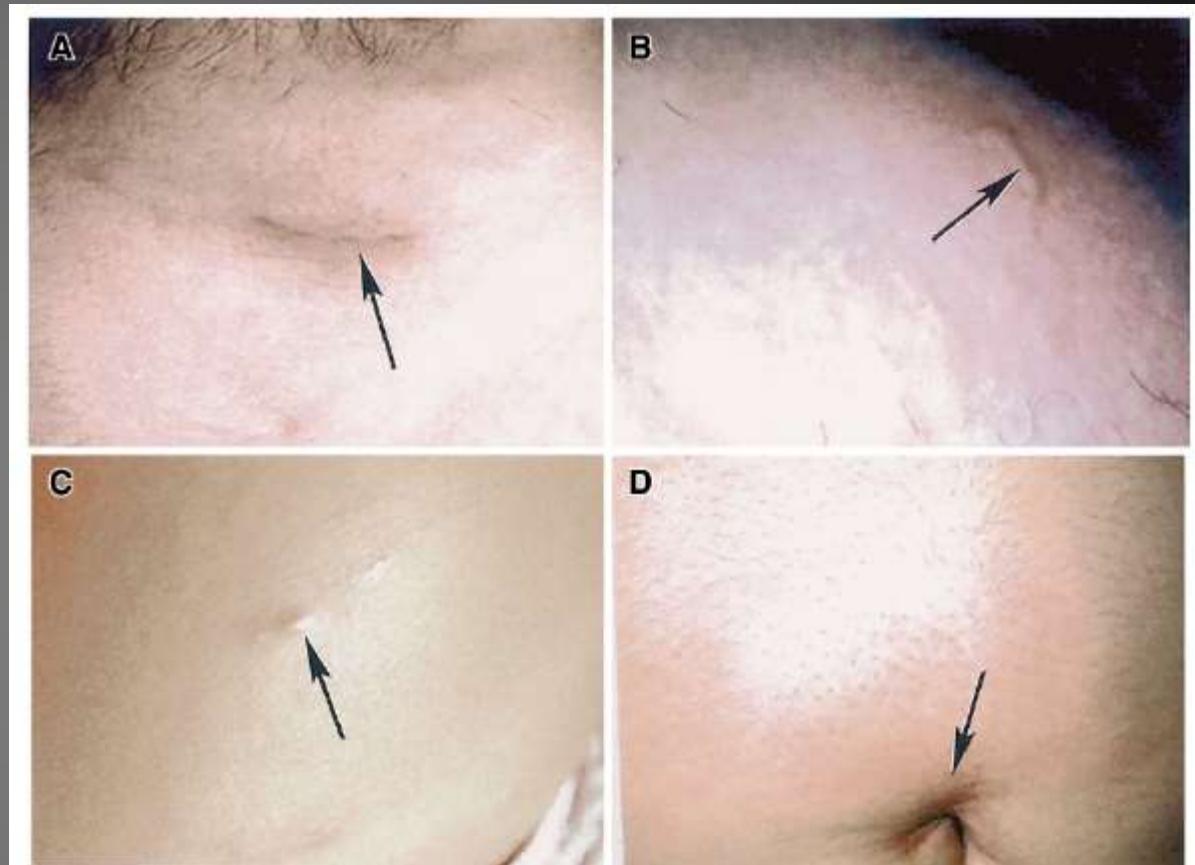


Fig. 3. Images of skin lesions in four patients with acute symptomatology that appeared 8 and 9 days after eating raw ceviche. (a) Creeping lesion on the chest of a 30-year-old male (arrow). (b) Creeping edematous lesion on shoulder of a 55-year-old male patient (arrow). (c) Nodular lesions on the abdomen of a 32-year-old female (arrow). (d) Edematous swelling on abdominal wall of a 28-year-old female patient (arrow).

Table 1  
Clinical and laboratory results in five patients with acute gnathostomiasis in Escuinapa, Sinaloa

Patients		ELISA OD ≥ 0.500	Eosinophils <sup>a</sup> (cells/μl)		Total IgE <sup>b</sup> (mg/dl)	Number of skin lesions <sup>c</sup>
Sex	Age (years)			(%)		
M	30	1.014	506	6	180	5
M	33	0.574	71	1	140	3
M	55	1.578	3772	46	130	12
F	28	0.693	800	9	160	4
F	32	0.524	156	2	170	4

<sup>a</sup> Normal range < 700 cells/μl (7%).

<sup>b</sup> Normal range < 100 mg/dl.

<sup>c</sup> Lesions in all patients appeared on days 8 and 9 after acute onset.

# Conclusiones

- Puede haber la aparición de enfermedades emergentes, globalización, viajes, importación de alimentos
- El desconocimiento de una patología no excluye la posibilidad de que se presente en el país o en la ciudad
- Algunas de diagnóstico fundamentalmente clínico-epidemiológico
- Algunas ameritan más estudio en el país para determinar su real endemidad (ej. gnathostomiasis, anisakiasis)



Manila, Filipinas, 2005

