ENFERMEDAD DE CHAGAS

XXX CONGRESO NACIONAL DE LA SOCIEDAD ESPAÑOLA DE MEDICINA INTERNA

VIII CONGRESO DE LA SOCIEDAD DE MEDICINA INTERNA DE LA COMUNIDAD VALENCIANA

VALENCIA NOVIEMBRE DEL 2009

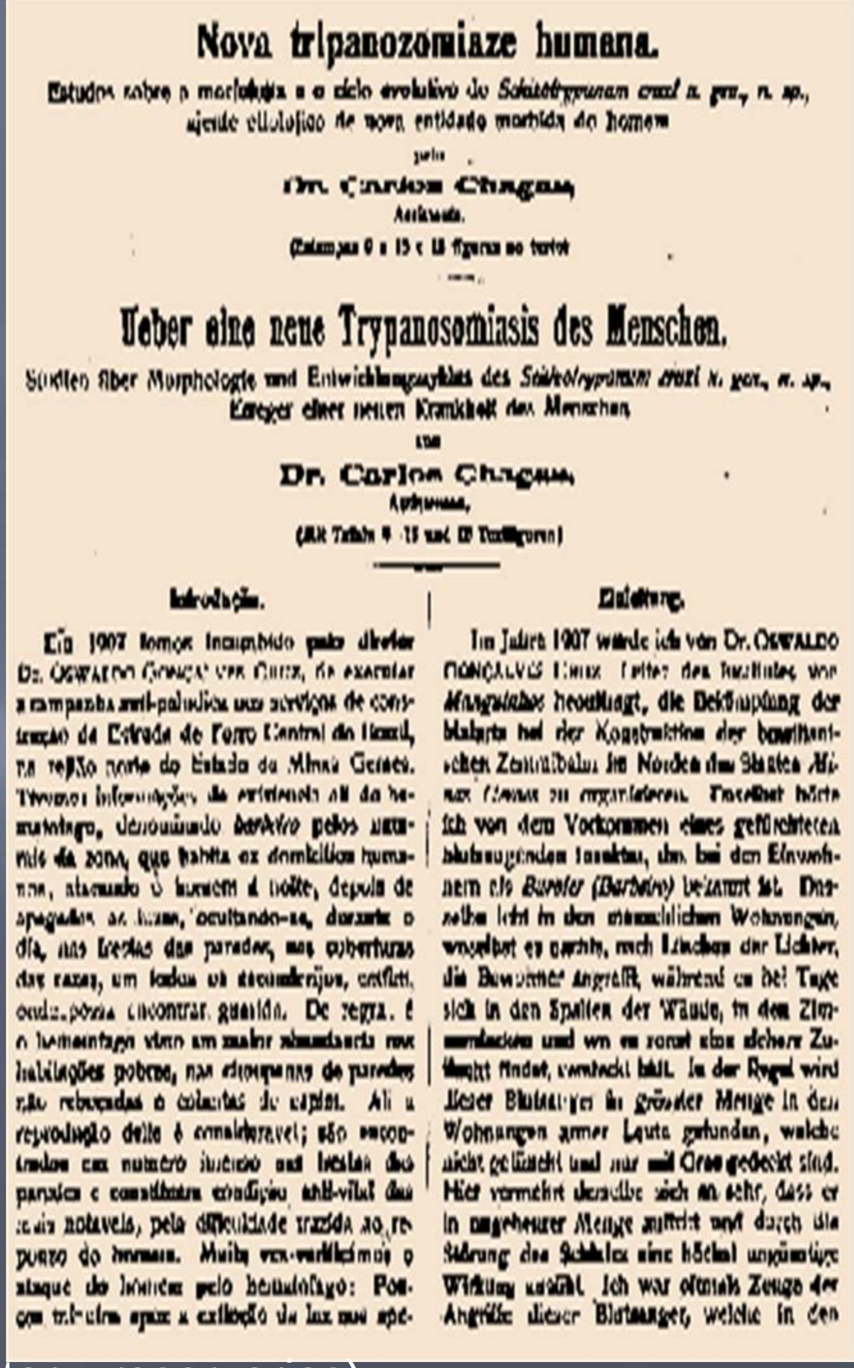
Magdalena García Rodriguez
Unidad de Enfermedades Infecciosas
Sección de Salud Internacional y Consejo al Viajero

CONSORCI
HOSPITAL GENERAL
UNIVERSITARI
VALÈNCIA

Carlos Chagas (1879-1934)

Abril de 1909 nueva enfermedad

Patología infecciosa, agente etiológico, ciclo (vector y reservorios)



Nova tripanozomiaze humana.

Estudos sobre a morfolojia e o ciclo evolutivo do *Schizotrypanum cruzi* n. gen., n. sp., ajente etiolojico de nova entidade morbida do homem

pelo

Dr. Carlos Chagas,
Assistente.

(Estampas 9 a 13 e 13 figuras no texto)

Ueber eine neue Trypanosomiasis des Menschen.

Studien über Morphologie und Entwicklungszyklus des *Schizotrypanum cruzi* n. gen., n. sp., Erreger einer neuen Krankheit des Menschen

von

Dr. Carlos Chagas,
Assistent.

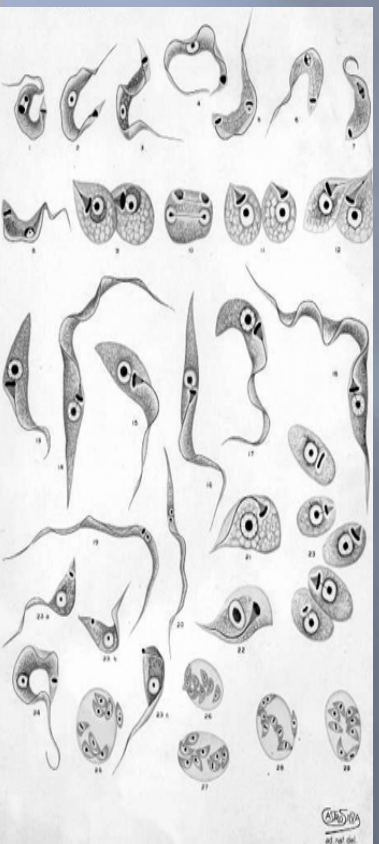
(Mit Tafeln 9–13 und 13 Textfiguren)

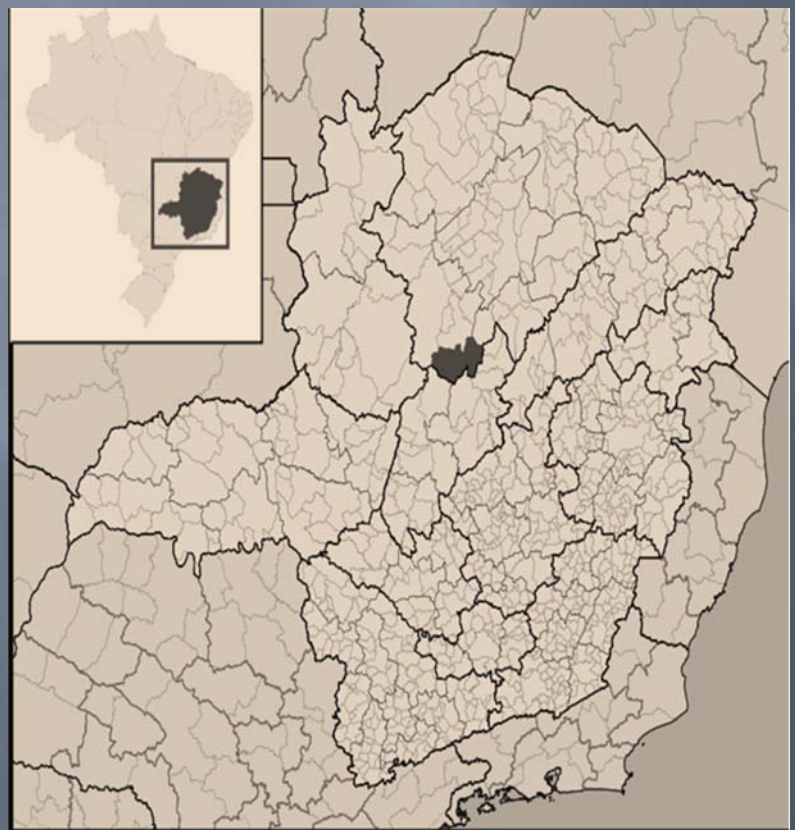
Introdução.

Em 1907 fomos incumbido pelo diretor Dr. OSWALDO GONÇALVES CRUZ, de executar a campanha anti-palustre nos serviços de construção da Estrada de Ferro Central do Brazil, na região norte do Estado de Minas Geraes. Tivemos informações da existencia ali de hematofago, denominado *barbeiro* pelos moradores da zona, que habita os domicilios humanos, atacando o homem á noite, depois de apagadas as luzes, ocultando-se, durante o dia, nas frestas das paredes, nas coberturas das casas, em todos os esconderijos, enfim, onde possa encontrar guarida. De regra, é o hematofago visto em maior abundancia nas habitações pobres, nas choupanas de paredes não rebocadas e cobertas de capim. Ali a reprodução delle é consideravel; são encontrados em numero incrivel nas frestas das paredes e constituem condição anti-vital das mais notaveis, pela dificuldade trazida ao repouso do homem. Muitas vezes verificámos o ataque do homem pelo hematofago: Poucos minutos apoz a extinção da luz nas ape-

Einleitung.

Im Jahre 1907 wurde ich von Dr. OSWALDO GONÇALVES CRUZ, Leiter des Instituts von *Manguinhos* beauftragt, die Bekämpfung der Malaria bei der Konstruktion der brasilianischen Zentralbahn im Norden des Staates *Minas Geraes* zu organisieren. Daselbst hörte ich von dem Vorkommen eines gefürchteten blutsaugenden Insektes, das bei den Einwohnern als *Baratas (Barbeiro)* bekannt ist. Dasselbe lebt in den menschlichen Wohnungen, woselbst es nachts, nach Löschen der Lichter, die Bewohner angreift, während es bei Tage sich in den Spalten der Wände, in den Zimmerdecken und wo es sonst eine sichere Zuflucht findet, versteckt hält. In der Regel wird dieser Blutsauger in grösster Menge in den Wohnungen armer Leute gefunden, welche nicht getüncht und nur mit Gras gedeckt sind. Hier vermehrt derselbe sich so sehr, dass er in ungeheurer Menge auftritt und durch die Störung des Schlafes eine höchst ungünstige Wirkung ausübt. Ich war oftmals Zeuge der Angriffe dieser Blutsauger, welche in den





1907 Lassance (Minas de Ger

Brote de malaria

Insectos hematófogos

Flagelado unicelular

Ministério da Saúde

FIOCRUZ

Fundação Oswaldo Cruz

Anno XXIII — 22 de Abril de 1909 — Num. 16

Brazil-Medico

SUMMARIO

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PATHOLOGIA INTERTROPICAL

Nova especie morbida do homem, produzida por um trypanozoma (trypanozoma Cruzi)

Nota prévia

(Trabalho do Instituto Oswaldo Cruz)

PELO DR. CARLOS CHAGAS

(Assistente do Instituto)

Vimos, desde mezes, estudando o cyclo evolutivo de um hemo-flagellado, o *trypanozoma Cruzi*, que tem para hospedeiro intermediario um hematophago, o *conorrhinus sanguisuga* (?). Fizemos, de nossas pesquisas ainda não concluidas, uma publicação prévia (1), aguardando opportunidade, após esclarecimento de alguns pontos, para publicação definitiva. A infecção que serviu de inicio a nossos estudos fôra obtida experimentalmente pelo Dr. OSWALDO CRUZ, fazendo picar por alguns conorrhinos, levados de Minas, um sagui (*hapalle penicillata*). Por inoculações de sangue e ainda por picada de conorrhinos obtivemos a infecção em diversos animaes, taes como a cobaya, o cão, o coelho, sendo ella sempre mortal para alguns destes vertebrados. Ignoravamos, porém, qual fosse o hospedeiro habitual do trypanozoma e o esclarecimento deste ponto levou-nos a realizar novas pesquisas, na zona onde haviamos colhido o hematophago, pesquisas cujo resultado essencial, pela sua importancia, merecem immediata publicidade.

O *conorrhinus sanguisuga* (?) existe em grande abundancia no norte de Minas, nas zonas percorridas pelo prolongamento da E. de F. Central do Brazil. E' um hematophago, conhecido pelo nome vulgar de *barbeiro*, que habita os domicilios humanos, preferindo sempre o sangue do homem para suas refeições. Nas casas o *conorrhinus* habita as cavidades das paredes, encontrando guarida favoravel nas paredes não rebocadas, e só ataca o homem á noite, depois de apagadas as luzes. Constitue um terrivel flagello, em extremo incommodo ao homem, cujo repouso nocturno elle difficulta. Outros animaes domesticos, aquelles que permanecem no interior dos domicilios, são tambem picados pelo *conorrhinus*. No gato verificamos a infecção natural pelo trypanozoma que aquelle hematophago transmitte.

Dada a preferencia do *conorrhinus* pelo sangue humano, suspeitamos, de accordo com a theoria da evolução phylogenetica dos hemo-flagellados, pudesse ser parasita do homem o trypanozoma encontrado no apparelho digestivo daquelle hematophago. Orientamos dest'arte nossas pesquisas e desde logo chamou nossa attenção um quadro morbido uniforme, apreciavel em quasi todas as crianças da zona onde abunda o invertebrado.

Daquelle quadro, presente ás vezes em adultos, porém mais frequente nas crianças, os elementos mais salientes são os seguintes: grande anemia, decadencia organica accentuada, edema sub-palpebral e frequentemente edemas generalizados, engorgitamento ganglionar consideravel, havendo volumosos ganglios nas paredes perifericas (axilla, regiões inguinal e crural, pescoço, etc.). Em algumas crianças, é notavel a atrophia do desenvolvimento. E' uma condição morbida permanente, com incidentes agudos, que se expressam em reacção febril e outros elementos morbidos. As noções clinicas que temos da molestia são ainda muito incompletas, estando apenas iniciadas, nesse sentido, nossas observações. Nem sabemos muito sobre o prognostico, parecendo, pelas informações colhidas, ser molestia ás vezes mortal, escapando-lhe, porém, alguns doentes, que, segundo nos parece, ficarão immunisados.

Repetidos exames de sangue, em crianças na condição morbida chronica, foram negativos. Num doente febricitante, profundamente anemiado e com edemas, com plêiades ganglionares engorgitados, encontramos trypanozomas, cuja morphologia é identica á do *trypanozoma Cruzi*. Na ausencia de qualquer outra etiologia para os symptomas morbidos observados e ainda de accordo com a experimentação anterior em animaes, julgamos tratar-se de uma trypanozomiase humana, molestia occasionada pelo *trypanozoma Cruzi*, cujo transmissor é o *conorrhinus sanguisuga* (?).

Em nossas pesquisas temos sido vantajosamente acompanhado pelo Dr. BELISARIO PENNA, a quem deixamos aqui os mais sinceros agradecimentos.

Lassance, E. de F. Central, 15 de Abril de 1909.

(1) Neue Trypanosomen.—Tr. Minasense e T. Cruzi, n. 36, in «Archiv. f. Schiff u. Tropenhygiene, 1909, pag. 120.

Ministério da Saúde

FIOCRUZ
Fundação Oswaldo Cruz

23 de Abril de 1908

A 9,000-year record of Chagas' disease

Arthur C. Aufderheide[a,b], Wilmar Salo[a], Michael Madden[a], John Streitz[c], Jane Buikstra[d], Felipe Guhl[e], Bernardo Arriaza[f,g], Colleen Renier[h], Lorentz E. Wittmers, Jr.[i], Gino Fornaciari[j], and Marvin Allison[k]

www.pnas.org/cgi/doi/10.1073/pnas.0307312101
February 17, 2004 | vol. 101 | no. 7

Museo Arqueológico, San Pedro de Atacama

Desierto de Atacama, Chile

Ministério da Saúde
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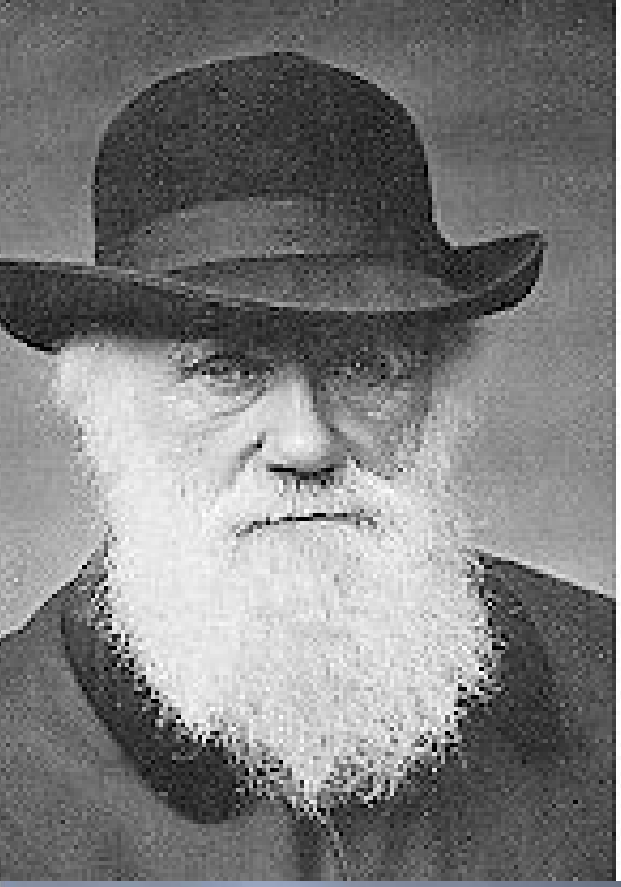
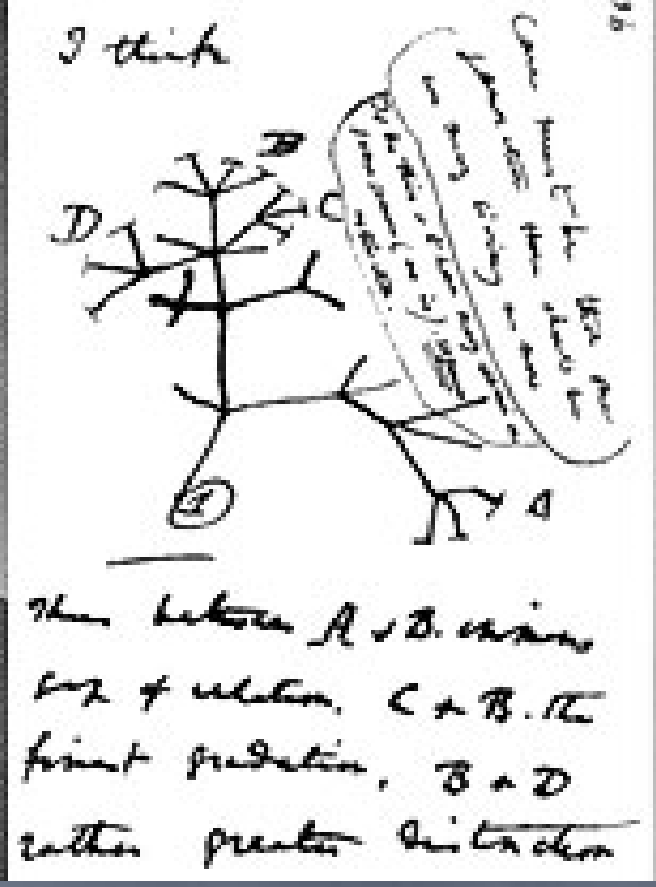




THE ORIGIN OF SPECIES

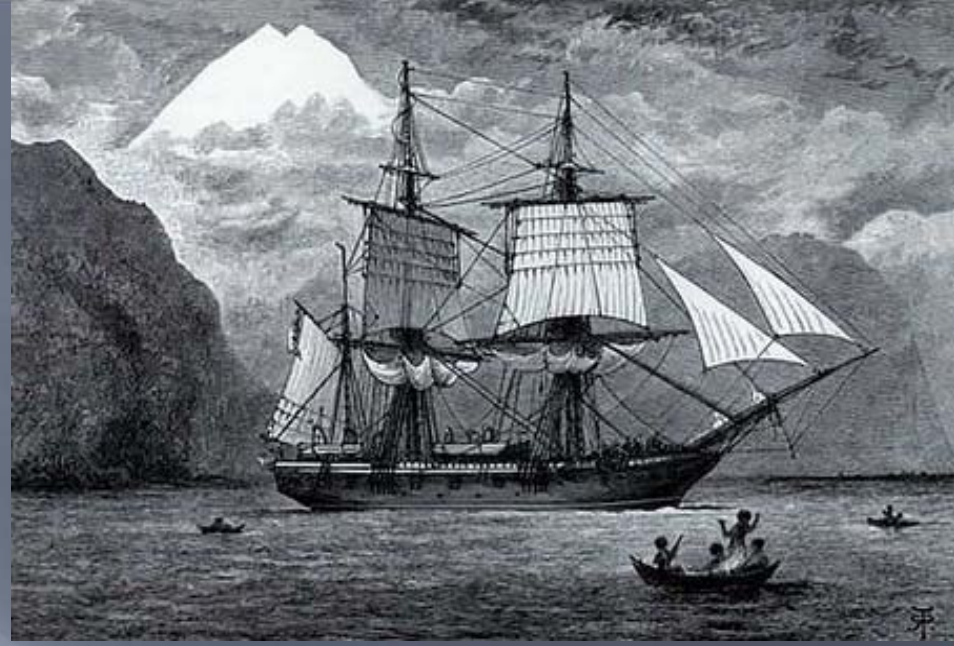
BY MEANS OF NATURAL SELECTION,

OR THE

PRESERVATION OF FAVOURED RACES IN THE STRUGGLE FOR LIFE.

By CHARLES DARWIN, M.A.,

LONDON:
JOHN MURRAY, ALBEMARLE STREET.

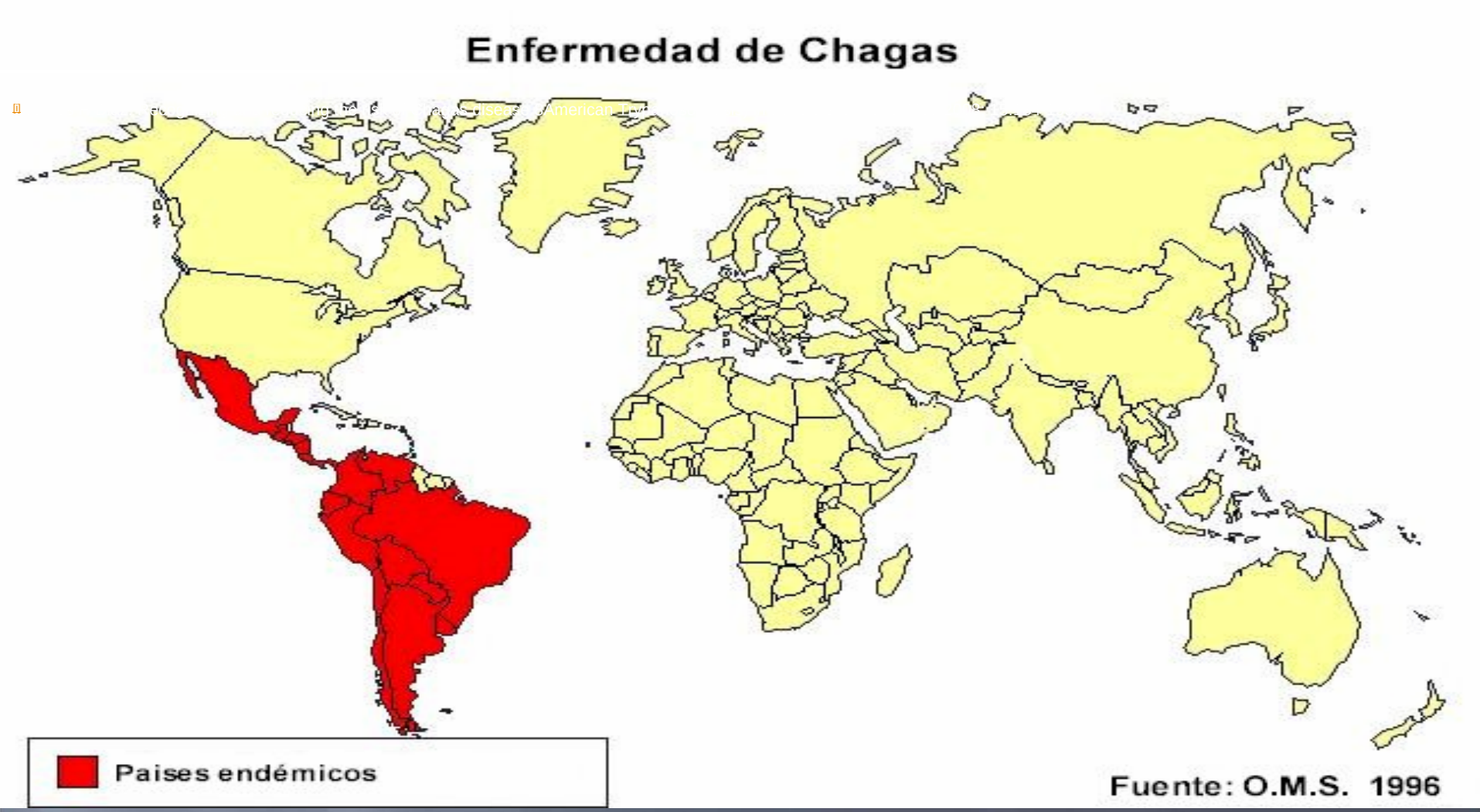


DID DARWIN HAVE SYNCOPE CAUSED BY CHAGAS' DISEASE?

TO THE EDITOR

The year 2009 is the 150th Anniversary of the publication of On the Origin of Species (24 November 1859) and the 200th Anniversary of Darwin's birth. Charles Robert Darwin was born on February 12, 1809 in Shrewsbury, England. From 1831 to 1836, Darwin served as naturalist aboard the H.M.S. Beagle on a British science expedition around the world.[1] In South America, Darwin found fossils of extinct animals that were similar to modern species. On the Galapagos Islands in the Pacific Ocean he noticed many variations among plants and animals of the same general type as those in South America. The expedition visited places around the world, and Darwin studied plants and animals everywhere he went, collecting specimens for further study. Out of this tudy, Darwin formulated several theories. He set forth these theories in his book called, On the Origin of Species by Means of Natural Selection, or the Preservation of Favoured Races in the Struggle for Life (1859) or "The Origin of Species" for short.[1] After publication of Origin of Species, Darwin continued to write on botany, geology, and zoology until his death in 1882. He is buried in Westminster Abbey. In 1832, Darwin arza province, Argentina.[5] There is a general consensus that Benchucas were in fact *Triatoma infestans*, the insect carrier of *Trypanosoma cruzi*, described by the Brazilian scientist Carlos Chagas, in 1909.[5,6] A second supporting factor is the set of symptoms refered by Darwin himself, during his chronic illness, including digestive complaints (vomiting attacks, eructation, flatulence) and particularly the pre-syncopal and syncopal episodes, more frequent at the end of his life, associated with complaints of effort dyspnea and palpitations, in keeping with a cardiomyopathy leading to cardiac syncopal episodes (described as occurring in supine position).[1,7] Nevertheless, the possibility of Chagas' disease acquired by Darwin during his passage in South America is still discussed, as well as even nowadays his genial theory of evolution, in special among some groups of religious theorists.

Acknowledgments – We are grateful to Prof. Francisco Javier Carod-Artal, from The Sarah Network of Rehabilitation Hospitals, Sarah Hospital, Brasilia, DF, Brazil, for his helpful comments.



En Estados Unidos, además de un importante número de casos importados, desde 1955 se han descrito **6 casos de EC autóctonos**.
El último en **Abril del 2007 en una mujer de 74 años en New Orleans, Louisiana**, lo que demuestra la presencia de reservorios y vector en estas latitudes.

James H Diaz. Recognizing and reducing the risk of Chagas disease (American Trypanosomiasis) in travelers. J Tra

DISEASES

A commonly accepted definition of "neglected" is:
1. failure to give proper care or attention to;
2. failure to do something.

Vector control management develops and promotes strategies and guidelines based on the principles of integrated vector management, including sound management of pesticides. Ethiopia, 2008.

Blinding trachoma is a bacterial infection caused by *Chlamydia trachomatis*. It is spread through contact with eye discharge from an infected person and is also transmitted through eye-seeking flies. Untreated, this condition leads to the formation of irreversible corneal opacities and blindness.

Buruli ulcer is a severe skin disorder that is caused by the bacterium *Mycobacterium ulcerans*, which belongs to the same family of organisms as those that cause leprosy and tuberculosis. Left untreated, it causes destruction of the skin and, in some cases, of bone, eyes and other tissue.

Chagas disease (American trypanosomiasis) is mainly a chronic condition caused by a protozoan parasite transmitted by the infected faeces of blood sucking bugs, through transfusion of infected blood, by organ transplantation or congenitally from an infected mother to her fetus.

Dengue is a mosquito-borne viral disease. The more severe forms of the disease are dengue haemorrhagic fever or dengue shock syndrome; these are usually fatal within 12–24 hours.

Dracunculiasis (guinea-worm disease) is transmitted by contaminated drinking water. It is characterized by the emergence of a one-metre-long worm from a skin ulcer usually, but not necessarily, in the leg.

Human African trypanosomiasis (sleeping sickness) is spread by the bite of the tsetse fly in impoverished rural areas of sub-Saharan Africa. A person may be infected for months or years without major signs or symptoms. When symptoms do emerge, the patient is often at a fatal stage of the disease and there is involvement of the central nervous system.

Leishmaniasis is transmitted by the bite of the sandfly. Visceral leishmaniasis, which attacks the internal organs, is the most severe form. Left untreated, it is usually fatal within two years. Cutaneous leishmaniasis commonly causes ulcers of the face, arms and legs and leaves severe and permanently disfiguring scars and disability.

Leprosy is a chronic infection caused by the bacillus *Mycobacterium leprae*. The disease mainly affects the skin, peripheral nerves, mucosa of the upper respiratory tract and the eyes. Once regarded as mutilating, contagious and incurable, the disease can now be easily cured using multidrug therapy.

Lymphatic filariasis (elephantiasis) is a severely debilitating, disfiguring and stigmatizing disease caused by parasitic worms. It usually causes abnormal enlargement of the limbs and the genitals.

Onchocerciasis (river blindness) is caused by a filarial worm that is transmitted to humans through the bites of infected blackflies. The larvae mature to adult worms; these move through the body and, when they die, they cause a variety of conditions, including blindness.

Rabies is a viral zoonotic disease mainly transmitted to humans through the bite of an infected dog. Rabies has a long incubation period and always kills within a few days after onset of symptoms. Human rabies can be prevented by proper wound care and administration of rabies biologicals immediately after a bite.

Schistosomiasis (bilharziasis) is a parasitic disease that leads to chronic ill-health. Infection is acquired from contaminated fresh water that contains the larval forms of blood flukes, known as schistosomes.

Global plan to combat neglected tropical diseases, 2008–2015

For tool-ready diseases, the objective is to expand coverage to its maximum with the help of partners and nongovernmental organizations. The WHO Department of Control of Neglected Tropical Diseases aims to promote broader coverage of rapid-impact, multi-intervention packages and to strengthen vector control, reduce transmission of several diseases and promote high-quality care by:

- assessing the burden of neglected tropical and zoonotic diseases;
- strengthening national health-care systems and building capacity to make primary health care more accessible;
- disseminating evidence for advocacy purposes;
- ensuring free and timely access to high-quality medicines as well as diagnostic and preventive tools;
- improving the development of innovative tools, drugs and control methods;
- strengthening the use of integrated approaches to vector management;
- building partnerships and mobilizing resources;
- promoting intersectoral approaches to enhance the integrated treatment of diseases;
- providing early protection to children;
- implementing surveillance and monitoring.

A young African woman returns from hospital after being successfully treated for a neglected tropical disease.

Decisive action

The prospects for controlling neglected tropical diseases provide unprecedented opportunities to make a real impact on the lives of the world's poorest and most vulnerable people. Effective drugs are available at no cost or very low cost. Delivery strategies have been devised that are compatible with conditions in very low income settings. Interventions can be integrated or bundled, and existing delivery systems can be used, thus increasing operational efficiency.

WHO hopes to control neglected tropical diseases by 2015, and even to eliminate some by that date, in order to alleviate the burden of avoidable mortality and morbidity among the world's poorest people.

"the enjoyment of the highest attainable standard of health is one of the fundamental rights of every human being ..."

WHO Constitution, 1948

"Neglected tropical diseases
Hidden successes,
Emerging opportunities"

NEGLECTED TROPICAL DISEASES

"Neglected tropical diseases
Hidden successes,
Emerging opportunities"

World Health Organization

Epidemiology, control and surveillance of Chagas disease - 100 years after its discovery

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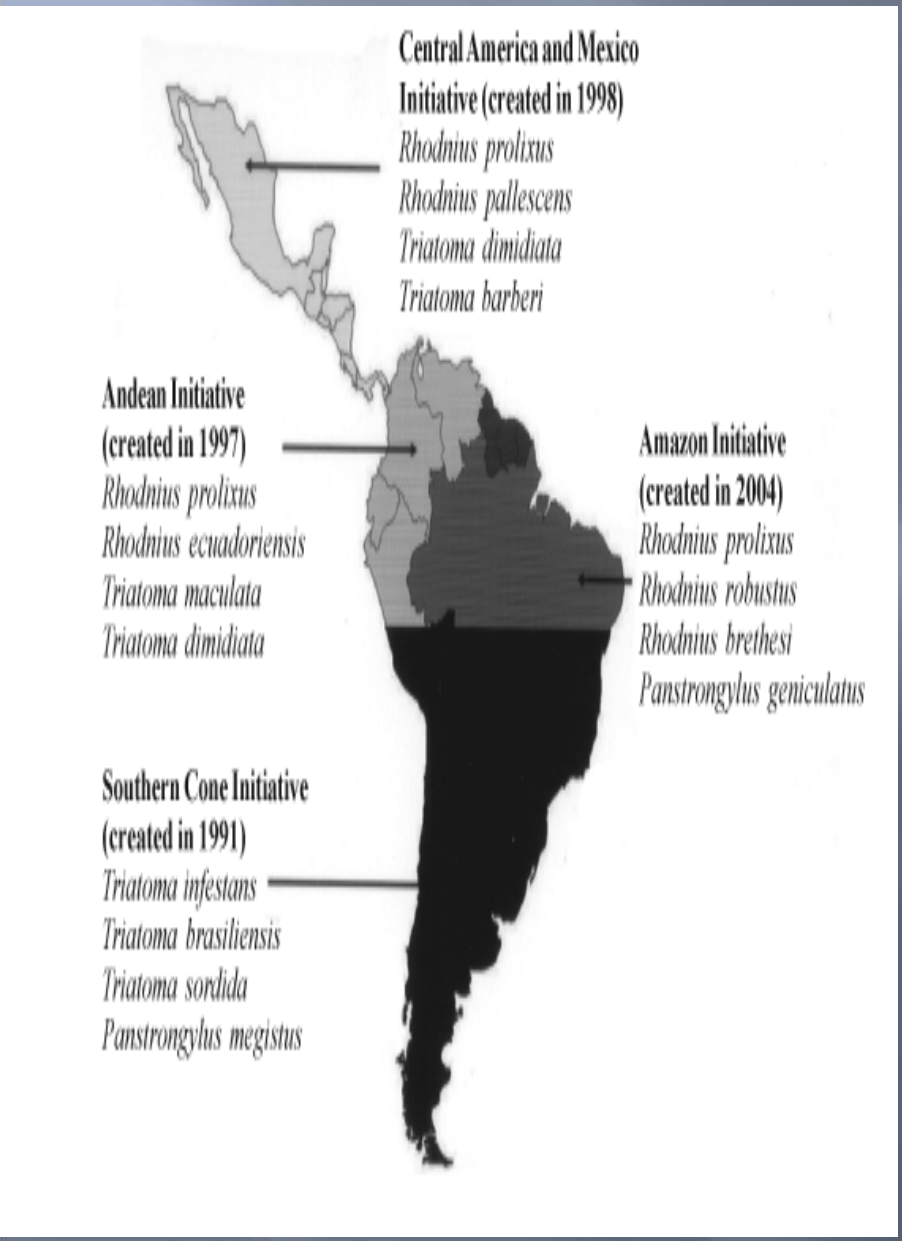
[2]Instituto de Pesquisas René Rachou-Fiocruz, Belo Horizonte, MG, Brasil

Chagas disease originated millions of years ago as an enzootic infection of wild animals and began to be transmitted to humans as an anthropozoonosis when man invaded wild ecotopes. While evidence of human infection has been found in mummies up to 9,000 years old, endemic Chagas disease became established as a zoonosis only in the last 200-300 years, as triatomines adapted to domestic environments. It is estimated that 15-16 million people are infected with Trypanosoma cruzi *in Latin America, and 75-90 million are exposed to infection. Control of Chagas disease must be undertaken by interrupting its transmission by vectors and blood transfusions, improving housing and areas surrounding dwellings, providing sanitation education for exposed populations and treating acute and recently infected chronic cases. These measures should be complemented by surveillance and primary, secondary and tertiary care.*



Organización Mundial de la Salud

62ª ASAMBLEA MUNDIAL DE LA SALUD
Punto 12.12 del orden del día provisional

A62/17
20 de marzo de 2009

Enfermedad de Chagas: control y eliminación

Informe de la Secretaría

6. Las iniciativas intergubernamentales para mejorar el control de la enfermedad de Chagas en Latinoamérica, basadas en el control de los vectores y el tratamiento de los casos, son: la Iniciativa del Cono Sur, iniciada en 1991 (Argentina, Bolivia, Brasil, Chile, Paraguay y Uruguay); la Iniciativa de los Países Andinos, iniciada en 1997 (Colombia, Ecuador, Perú y República Bolivariana de Venezuela); la Iniciativa de los Países de Centroamérica, creada en 1997 (Belice, Costa Rica, El Salvador, Guatemala, Honduras, Nicaragua y Panamá), la Iniciativa de los Países Amazónicos para la vigilancia y el control de la enfermedad de Chagas, iniciada en 2004 (Bolivia, Brasil, Colombia, Ecuador, Guyana, Perú, República Bolivariana de Venezuela y Suriname) y México en 2003.

Current epidemiological trends for Chagas disease in Latin America and future challenges in epidemiology, surveillance and health policy

Álvaro Moncayo[1]/+, Antonio Carlos Silveira[2]

[1]Academia Nacional de Medicina, Bogotá, Colombia [2]Independent consultant on Chagas disease, Brasilia, Brasil

Estimated number of infections by *Trypanosoma cruzi* and annual incidence in Latin America, 1975-2005[a]

Country or region	Total number of infections			New cases		
	1975-1985	1995	2005	1990	1995	2005
Central America and Mexico	1.935,000[b]	ND	1.906,600	209,187	72,677	16,200
Argentina	2.333,000[c]	2.100,000	1.600,000	ND	ND	1,300
Brazil	4.500,000	1.900,000	1.900,000	ND	ND	0
Bolivia	1.134,000	ND	620,000	86,676	ND	10,300
Chile	1.239,000	157,000	160,200	ND	ND	0
Colombia	900,000	ND	436,000	39,162	31,330	5,250
Ecuador	300,000	450,000	230,000	7,488	13,365	2,350
Paraguay	397,000	ND	150,000	14,680	ND	900
Peru	643,000	ND	192,000	24,320	19,072	3,100
Uruguay	37,000	ND	21,700	ND	ND	0
Venezuela	1.200,000	ND	310,000	179,703	22,960	1,400

a: data from Schmunis (2007a); *b*: except Mexico, 1995; *c*: 1990; ND: no data.

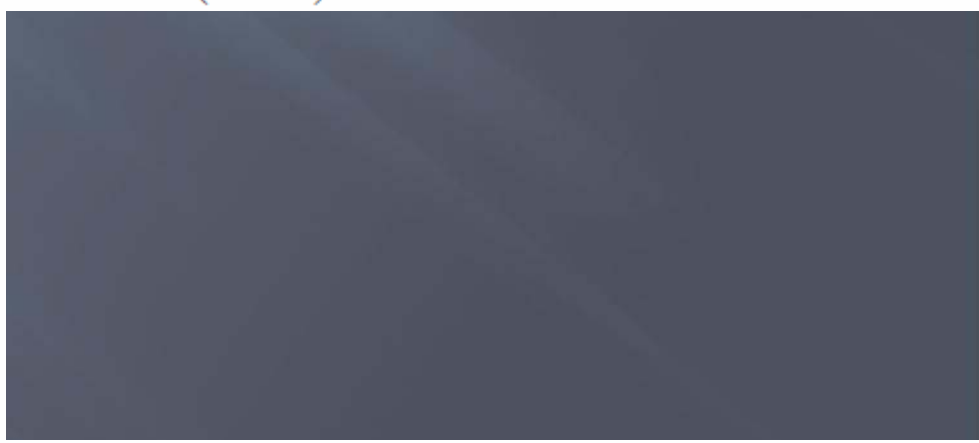
Coverage of screening of blood for transfusion for *Trypanosoma cruzi* (%) and proportion of positive samples in endemic countries, 2005[a]

Country	Coverage of screening for *T.cruzi* (%)	Positive samples (%)
Southern Cone		
Argentina	100	2,47
Bolivia	80	8,0
Brazil	100	0,21
Chile	87	0,6
Paraguay	99	3,2
Uruguay	100	0,47
Andean countries		
Colombia	100	0,44
Ecuador	100	0,15
Peru	99	0,57
Venezuela	100	0,6
Central America and Mexico		
Belize	ND	0,4
Costa Rica	100	0,09
El Salvador	100	2,4
Guatemala	100	0,79
Honduras	100	1,4
Nicaragua	100	0,9
Panama	97,6	1,4
Mexico	100	0,51

a: data from OPS/OMS (2006); ND: no data.

Changes in epidemiological parameters due to interruption of transmission and decrease of incidence, 1990- 2006

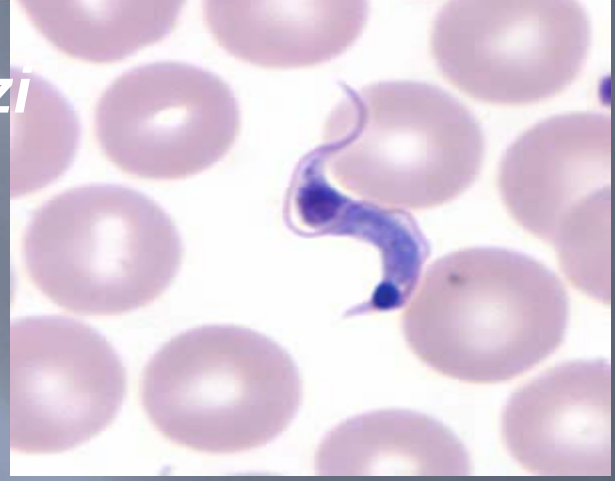
Epidemiological parameters	1990	2000	2006
Annual deaths	> 45,000	21,000	12,500
Annual new cases	700,000	200,000	41,200
Prevalence (million)	30	18	15

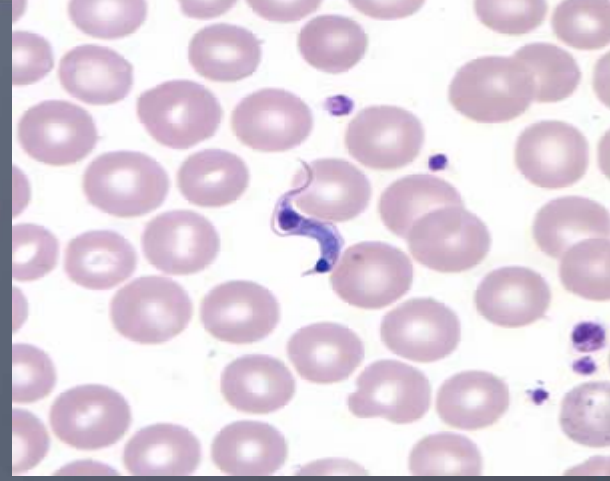


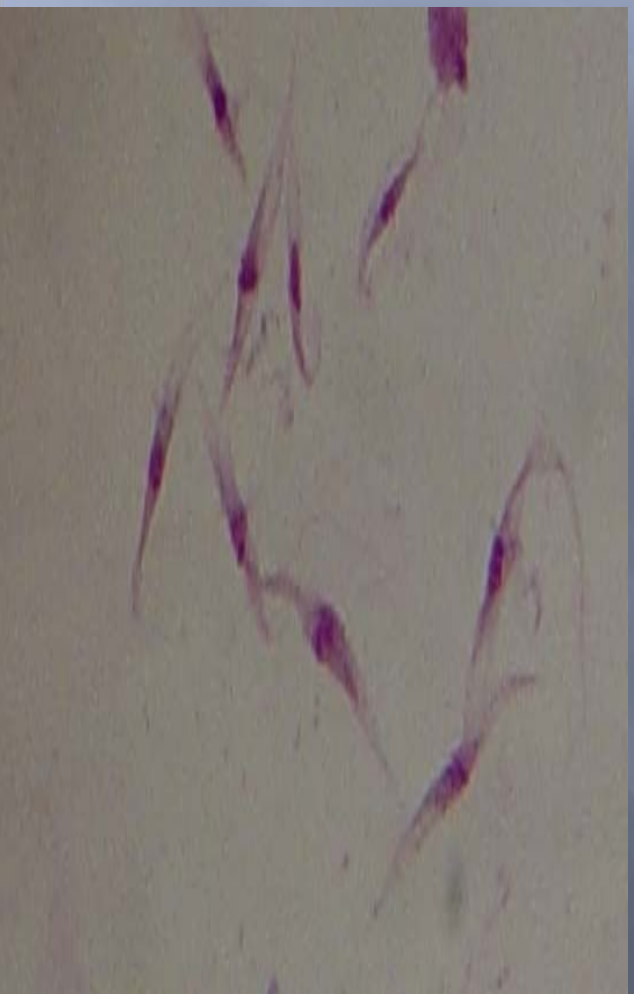
Trypanosoma cruzi

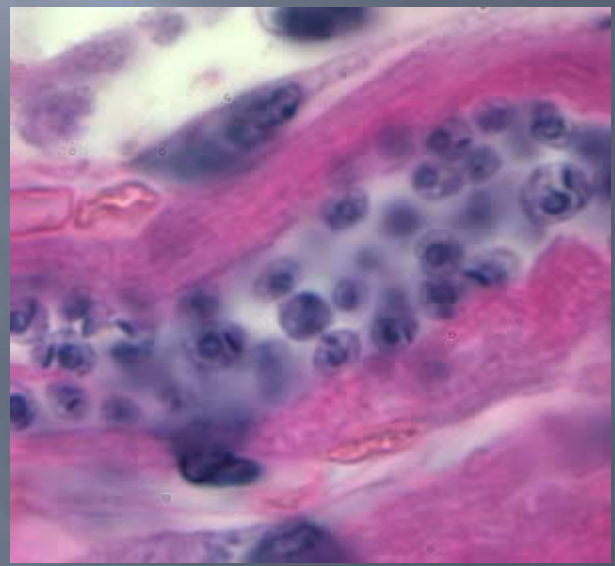
Subreino protozoa

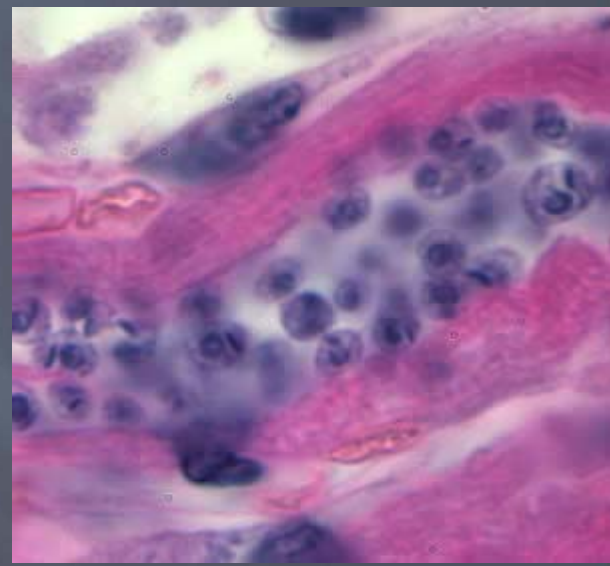
Clase zoomastigophorea











TRANSMISION

Exclusiva de países endémicos

Vectorial …….. ……
. 80-90%

Transfusional /transplante de órganos
…… 5-20%

Vertical ……..
…….. 0.5-8%

Oral Transmission of Chagas Disease by Consumption of Açaí Palm Fruit, Brazil

Aglaêr A. Nóbrega, Marcio H. Garcia, Erica Tatto, Marcos T. Obara, Elenild Costa, Jeremy Sobel, and Wildo N. Araujo

In 2006, a total of 178 cases of acute Chagas disease were reported from the Amazonian state of Pará, Brazil. Eleven occurred in Barcarena and were confirmed by visualization of parasites on blood smears. Using cohort and case–control studies, we implicated oral transmission by consumption of açaí palm fruit.

VINCHUCA

PITO

BARBEIRO

CHIRIMACHAS

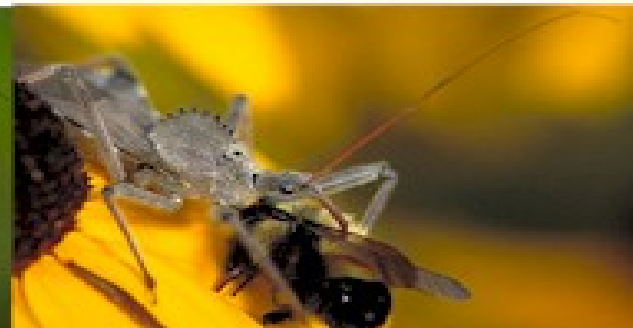
CHINCHE

CHIPO

KISSING BUGS

CHINCHE ASESINA

CHINCHE BESUCONA



Insectos hemípteros hematófagos

Familia Reduviidae

SubfamiliaTriatominae

R. prolixus

T. infestans

P. megistus

T.dimidiata

T. pallidipennis

T. infestans - P. megistus

R. prolixus - T. dimidiata

Las más importantes son las que han colonizado las viviendas humanas

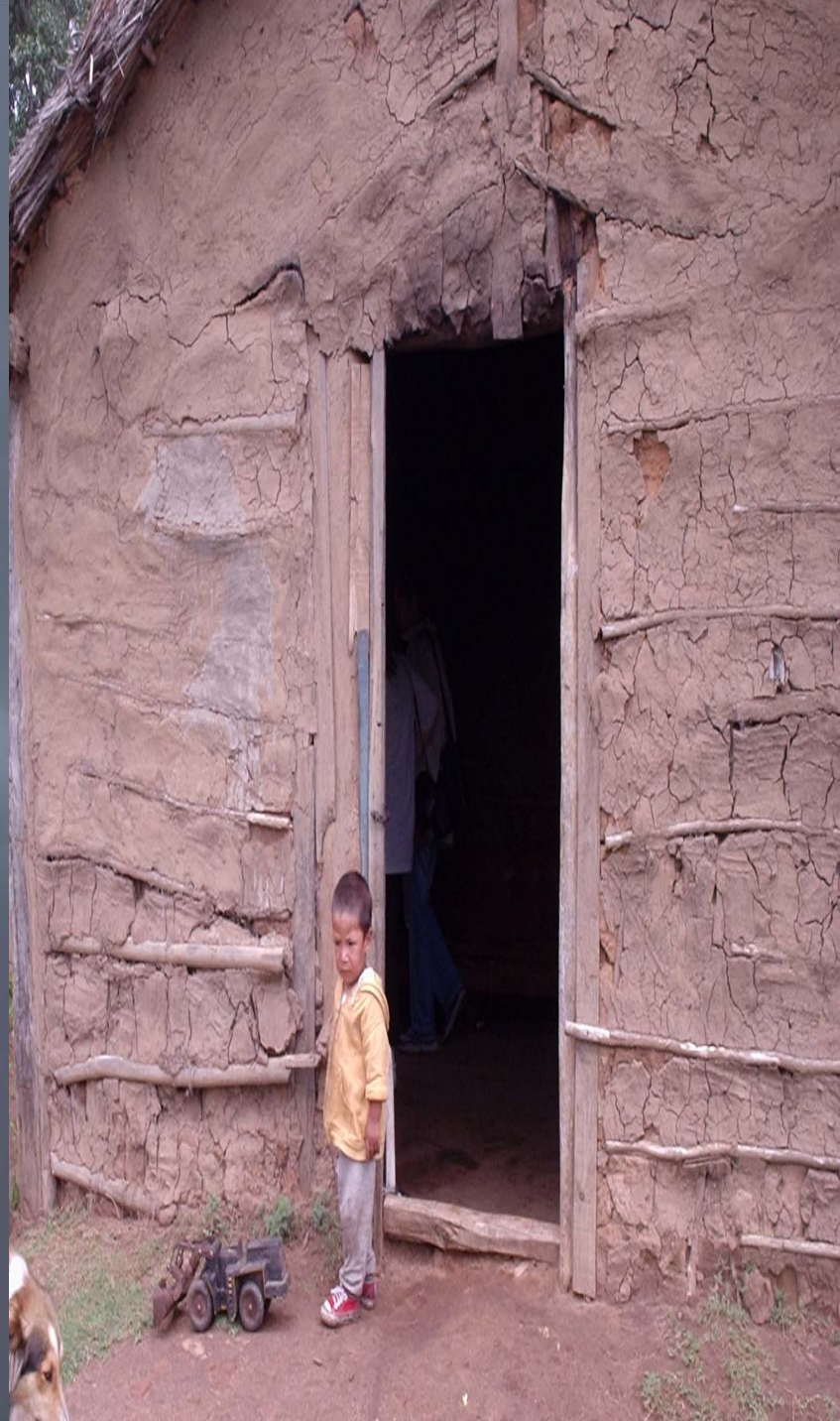
Géneros:

Triatoma

Rhodnius

Panstrongylus

Christian Lombard



NÃO DEIXE O BARBEIRO ENTRAR NA SUA CASA.

Mantenha os arredores da sua casa limpo.

Permita que o agente de saúde borrife sua casa.

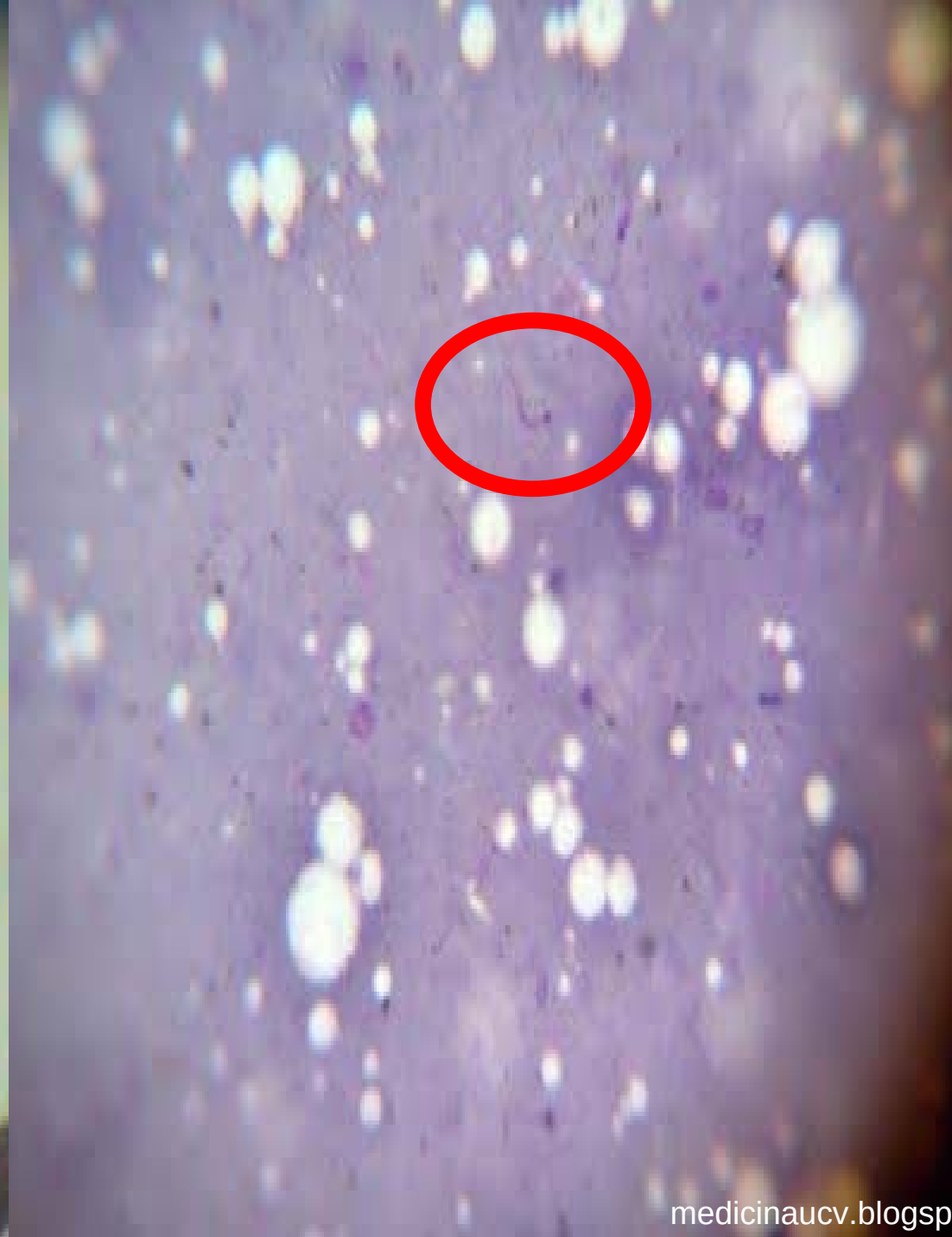
Mantenha currais, galinheiros e cercas o mais distante possível da sua casa.

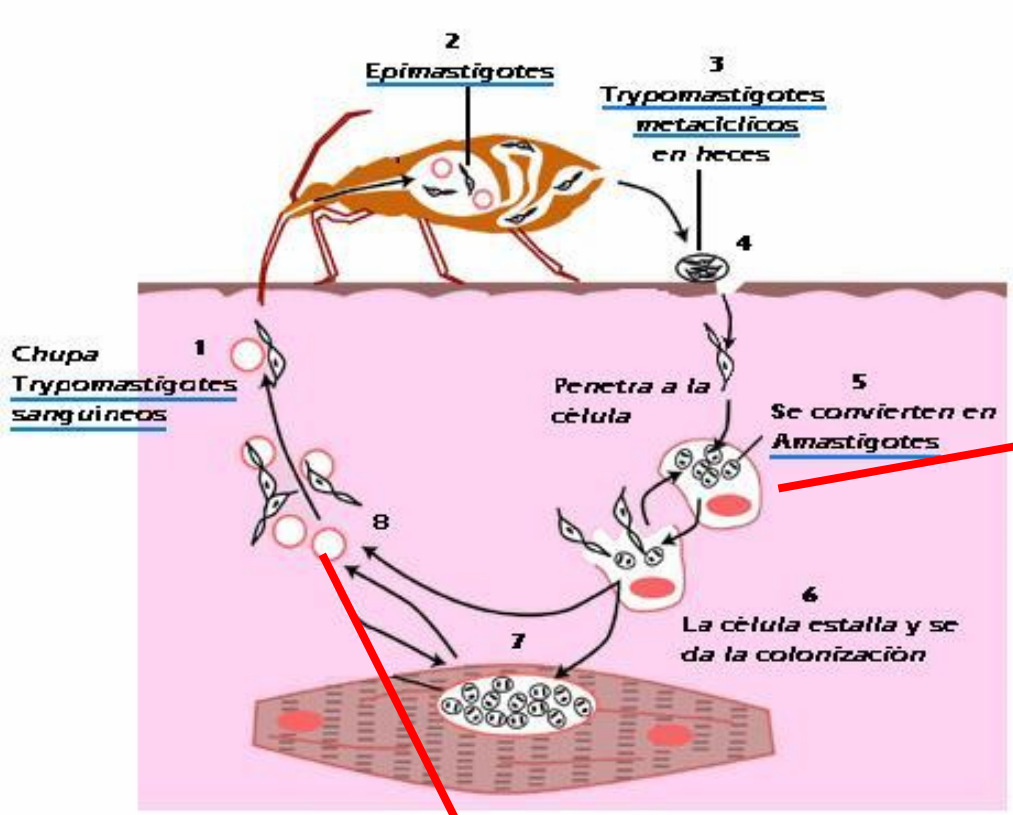
Reboque as paredes da sua casa.

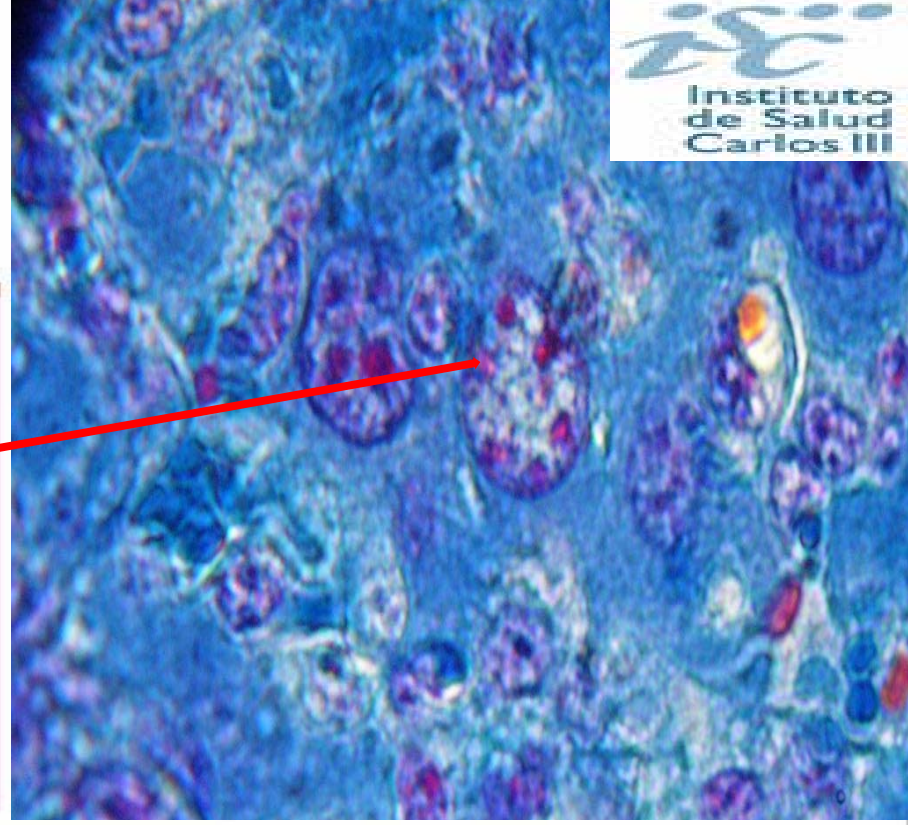
FNS Coordenadoria Regional do Rio Grande do Norte

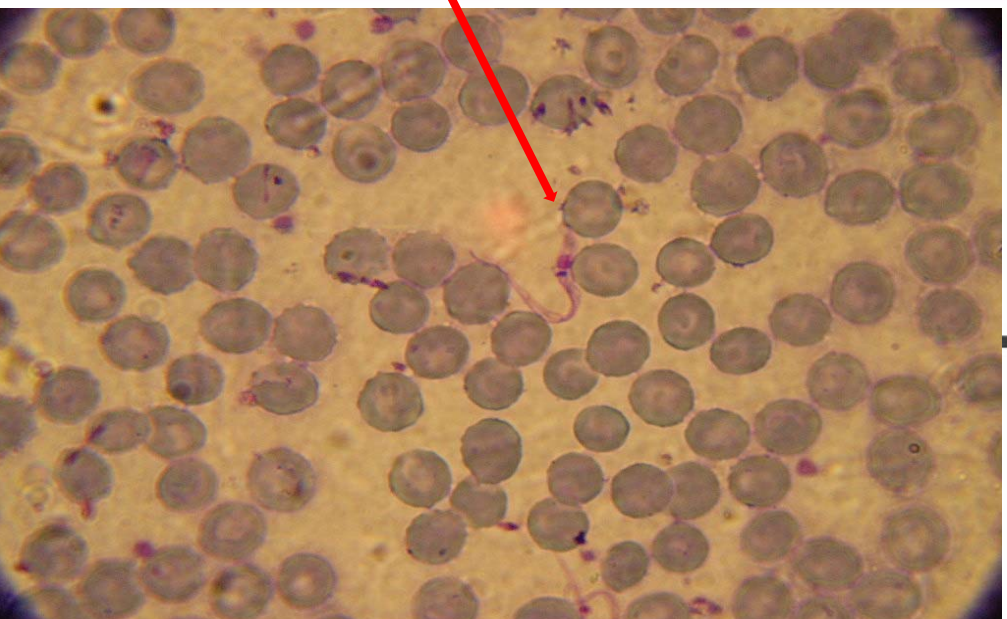
PCDEN PROGRAMA DE CONTROLE DAS DOENÇAS ENDÊMICAS NO NORDESTE

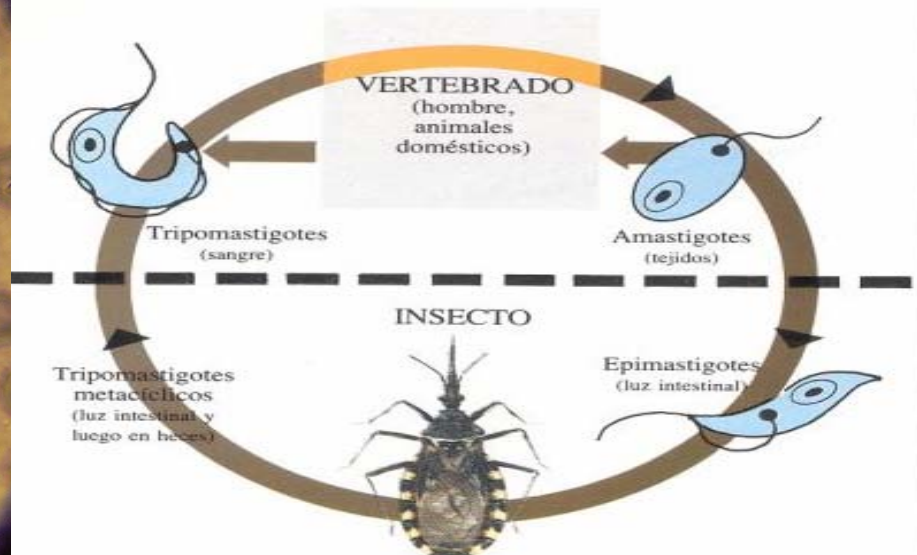
BRASIL UNIÃO DE TODOS




2
Epimastigotes
3
Trypomastigotes
metacíclicos
en heces
4
1
Chupa
Trypomastigotes
sanguíneos
Penetra a la
célula
5
Se convierten en
Amastigotes
8
6
La célula estalla y se
da la colonización
7


Instituto
de Salud
Carlos III

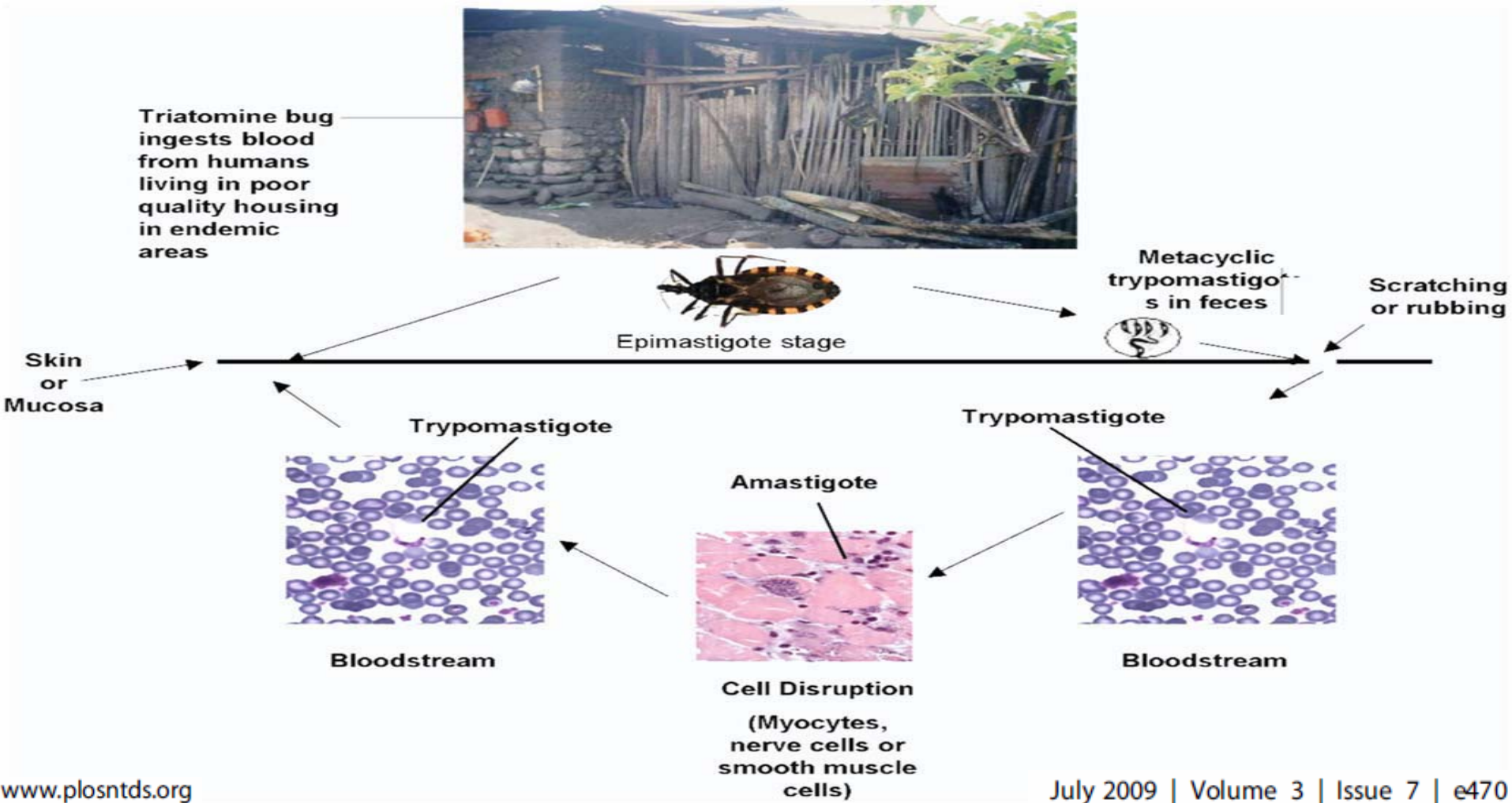


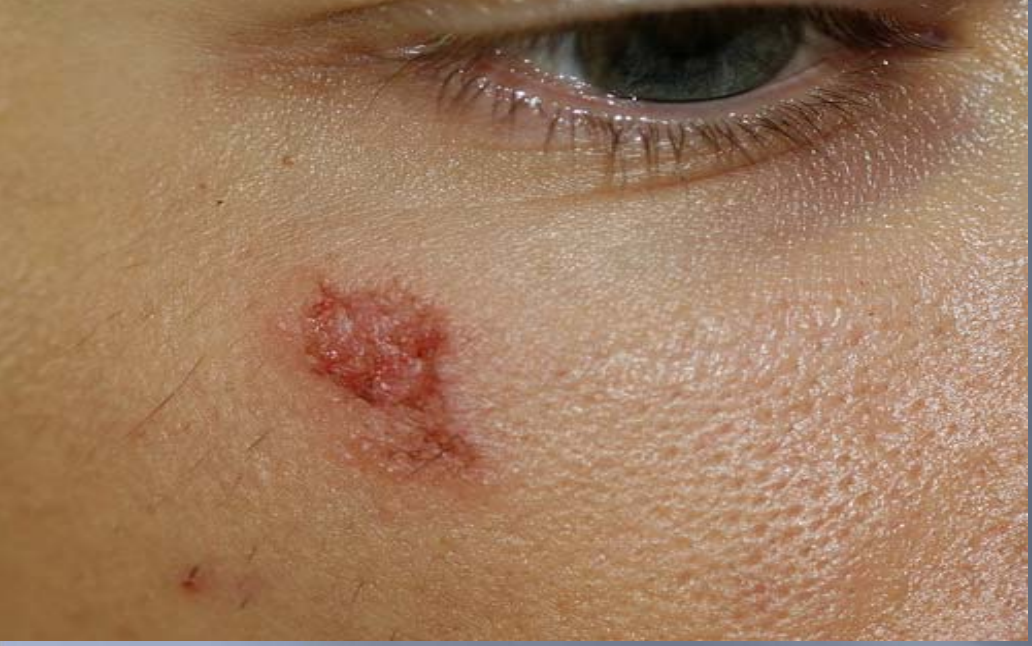

VERTEBRADO
(hombre,
animales
domésticos)
Tripomastigotes
(sangre)
Amastigotes
(tejidos)
INSECTO
Tripomastigotes
metacíclicos
(luz intestinal y
luego en heces)
Epimastigotes
(luz intestinal)

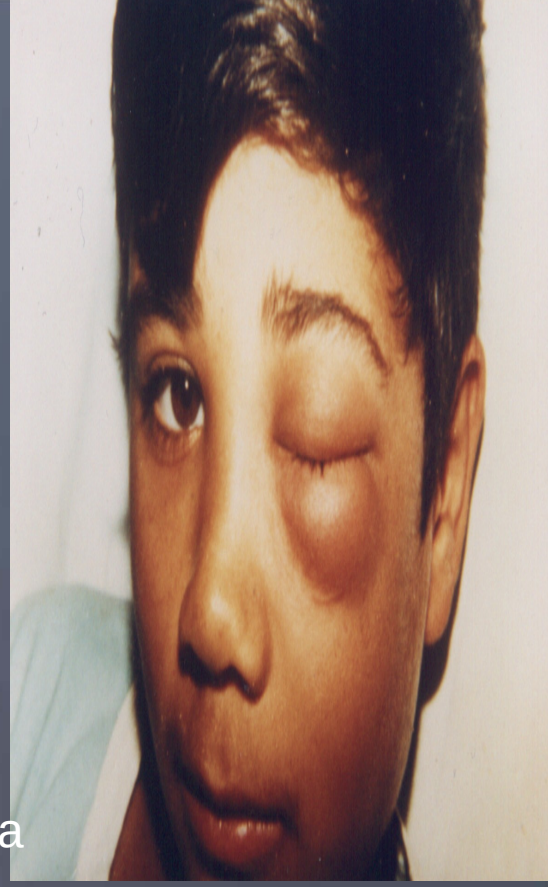
The Unfinished Public Health Agenda of Chagas Disease in the Era of Globalization

Carlos Franco-Paredes[1,2*], Maria Elena Bottazzi[3,4], Peter J. Hotez[3,4]

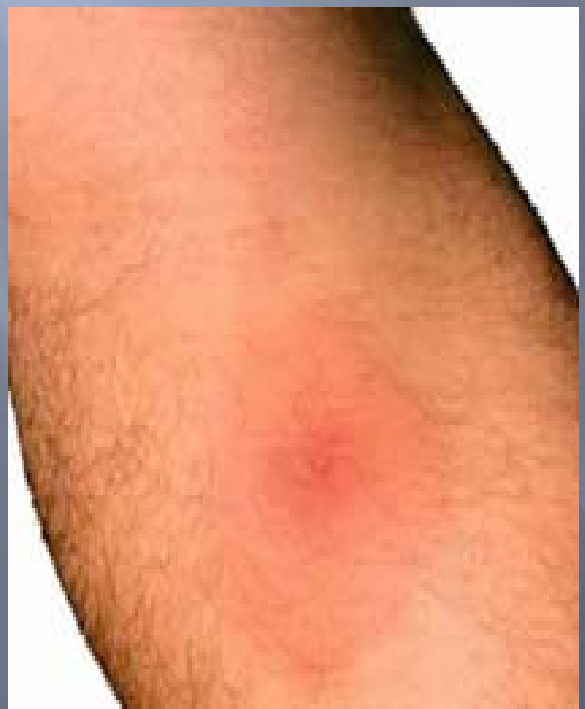
1 Hospital Infantil de México, Federico Gómez, México, D.F., México, **2** Department of Medicine, Emory University, Atlanta, Georgia, United States of America, **3** Department of Microbiology, Immunology, and Tropical Medicine, The George Washington University, Washington, D.C., United States of America, **4** Sabin Vaccine Institute, Washington, D.C., United States of America



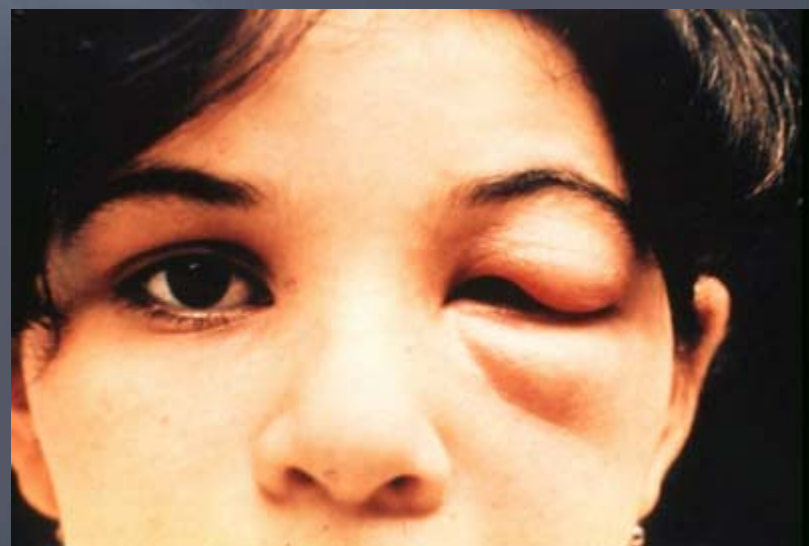




Signo de Romaña



Chagoma



Infección → **Fase aguda** → 90-95% asintomática → Mortalidad 5%
Niños<2 años
miocarditis/meningoenc

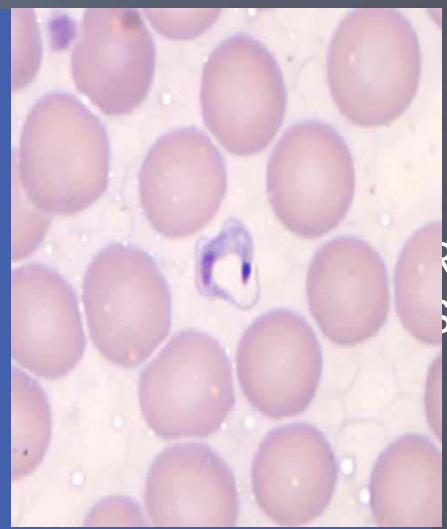
3 meses

Serología positiva (IgG)
Parasitemia baja e intermitente
PCR +/-

Fo **nic**

ebre
nfadenopatías
epatesplenomegalia
orexia
Cefalea
Mialgias
Astenia
Chagomas

al



Reactivación
SNC/Corazó

Parasitemia elevada
Serología positiva Ig M
(semanas)

Curación espontánea

Cardiaca 20-30%
10%
Digestiva 5%

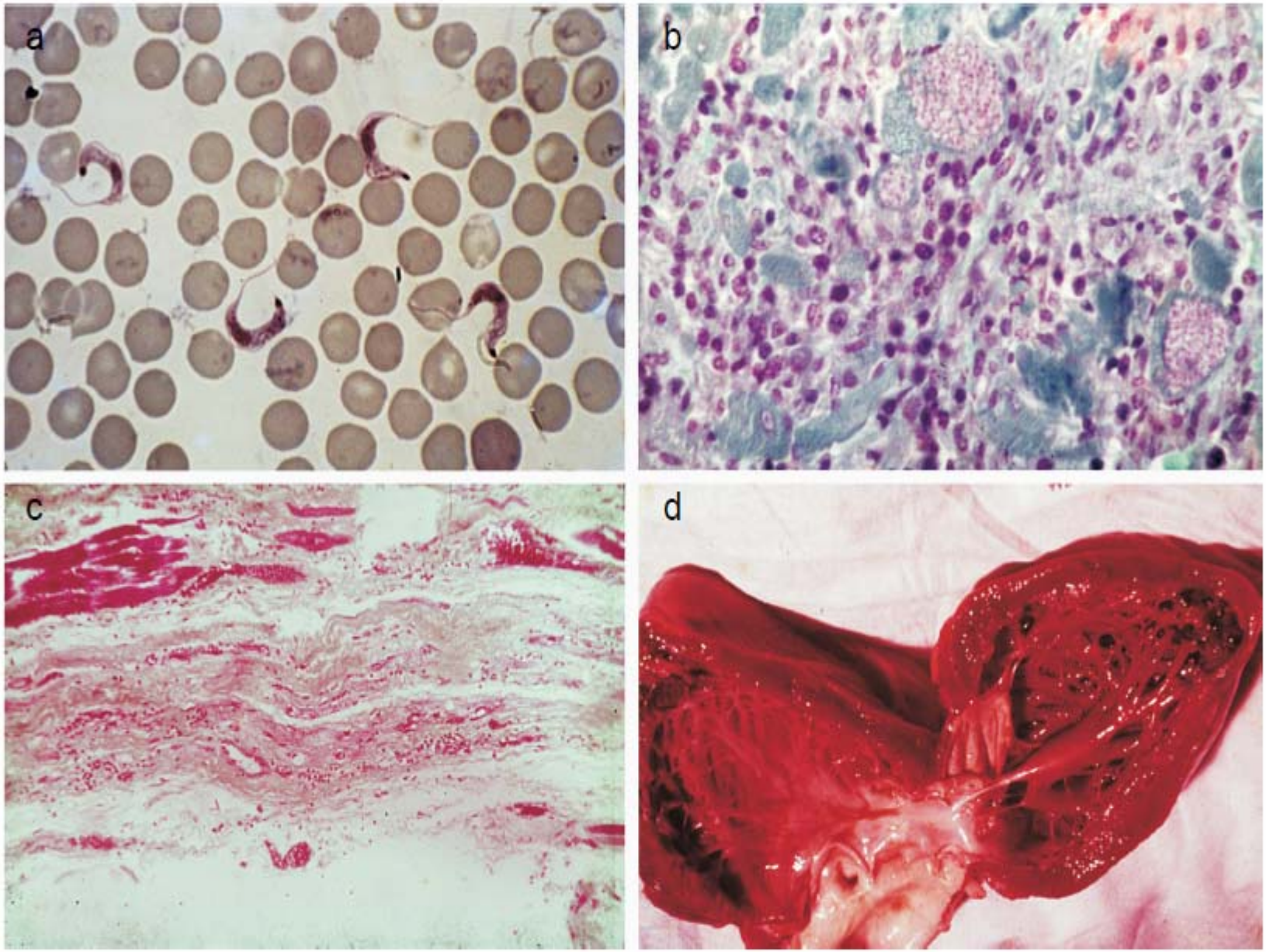


Fig. 2a: trypomastigotes circulating in blood during acute phase; b: pseudocysts of amastigotes in myocardial fibers in the acute phase of Chagas disease; c: fibrosis of the myocardial conducting system in chronic phase of Chagas disease; d: hipertrophy of myocardium and dilatation of the heart cavities with the presence of thrombi in chronic Chagas heart disease (Coura et al. 2007).

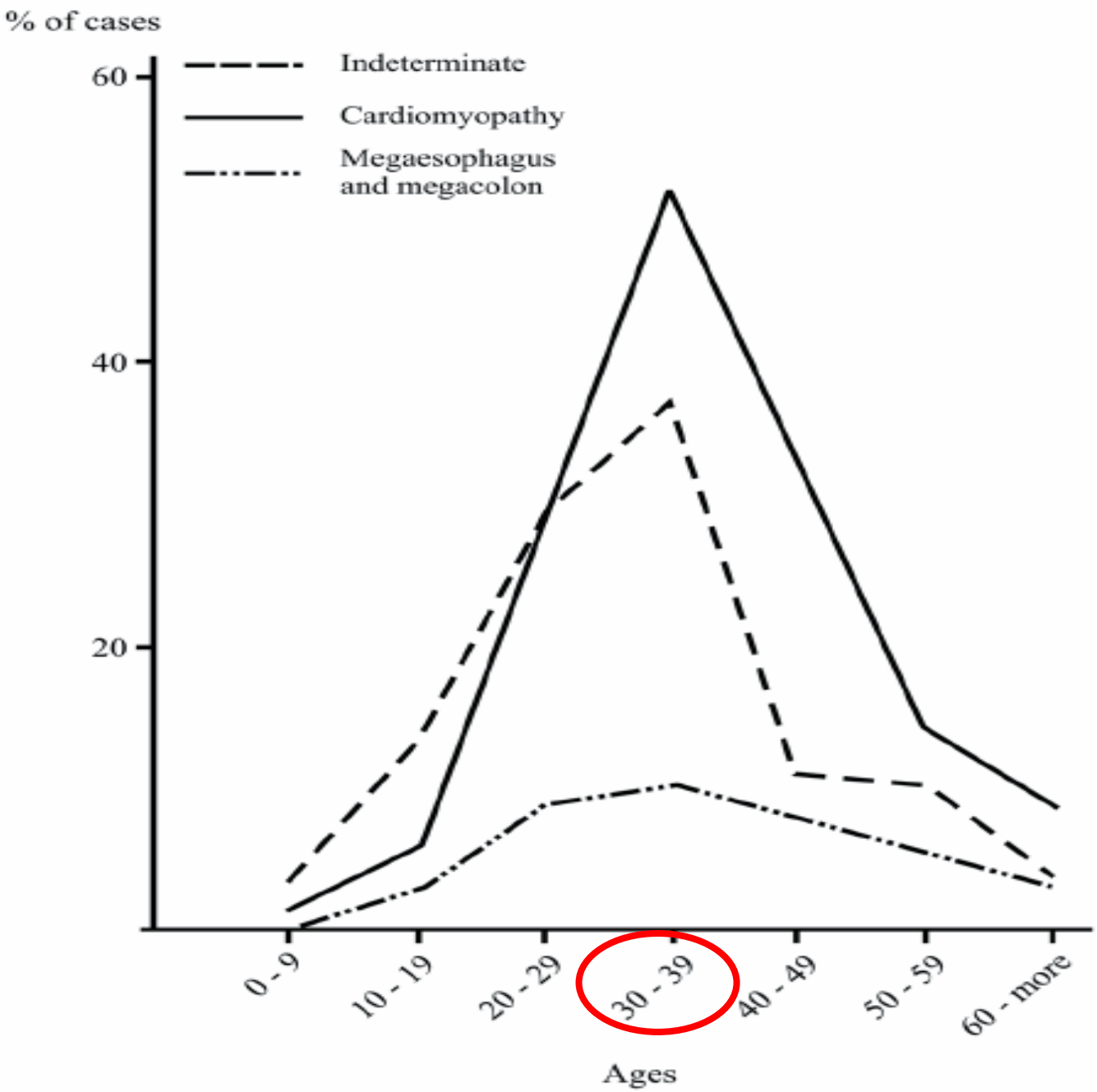


Fig. 5: distribution by clinical forms and ages of 510 cases of chronic Chagas disease (Coura et al. 1983).

FASE CRONICA FORMA INDETERMINADA

Factores predictores de la evolución

Ausencia de afectación orgánica
Serología positiva Ig G *T cruzi* +
Parasitemia transitoria y de bajo grado (PCR+/-)

Salar de Uyuni, **Bolivia**

FASE CRONICA FORMA DETERMINADA

Lago Rojo. Reserva Nacional de Fauna Andina Eduardo Avaroa, Bolivia

CARDIOPATIA CHAGASICA

Forma más grave y frecuente de presentación de la fase crónica en su forma determinada

Causa de muerte cardiovascular más frecuente entre los 30-50 años en zonas endémicas

Es una **forma inflamatoria de miocardiopatía dilatada** que conduce a una amplia fibrosis cardiaca y un deterioro progresivo de la función contráctil ventricular

Tres síndromes principales que pueden coexistir en el mismo paciente:

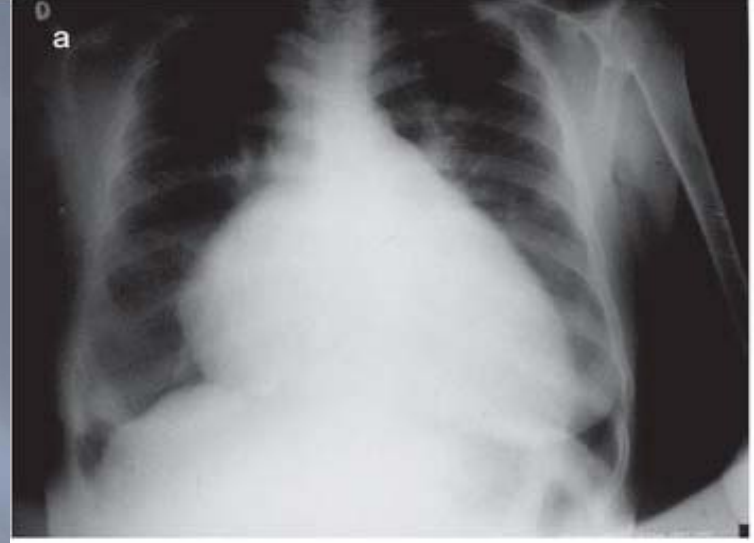
Insuficiencia cardiaca

Arritmias cardiacas

Fenómenos tromboembólicos (sistémico y pulmonar)

Marín-Neto Jose A., Rassi Anis Jr. Actualización sobre la cardiopatía de la enfermedad de Chagas en el primer centenario de su descubrimiento. Rev Esp Cardiol. 2009; 62(11)

Disnea de esfuerzo
Palpitaciones
Síncope
Dolor torácico
Edema
Muerte súbita
Ictus



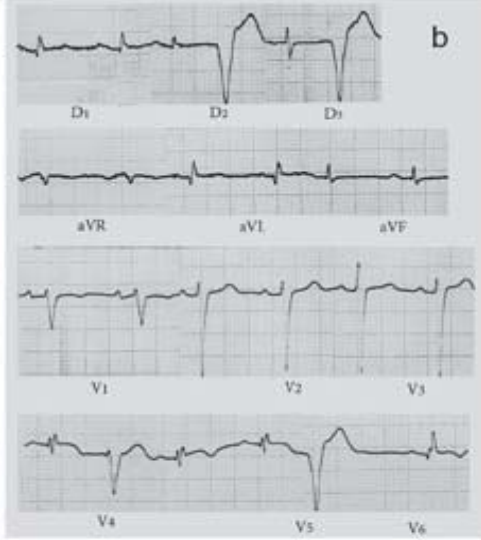


Fig. 6a: r-x showing enlargement of heart in chronic Chagas cardiomiopathy; b: ECG showing ventricle extrasystoles, A-V block, schemia, and myocardial fibrosis in chronic Chagas heart disease (Junqueira et al. 2005).

Los factores de riesgo de ictus en la miocardiopatía chagásica son:

•Presencia de miocarditis crónica inflamatoria (insuficiencia cardiaca)

•Alta prevalencia de trastornos de la conducción y de arritmias

•Alta prevalencia de lesiones segmentarias, incluyendo aneurisma apical

Más de 80% de los pacientes con EC que tiene un ictus presentan algún tipo de arrritmia, clínica o silente en el ECG

Carod Artal FJ, Vargas AP, Melo M, Horan T. American Trypanosomiasis (Chagas`disease): an unrecognized cause of stroke. J Neuro

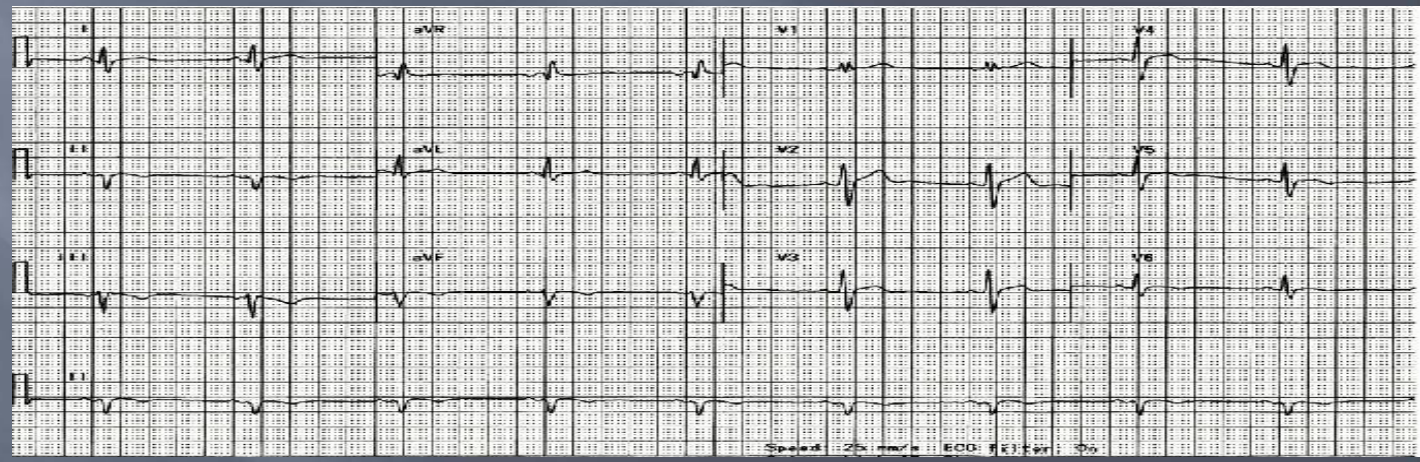

Jujuy
Paraguay
Salta
Formosa
Misiones
Catamarca
Chaco
Sgo. del Estero
Brasil
La Rioja
Corrientes
San Juan
Santa Fe
Córdoba
Entre Ríos
Uruguay
San Luis
Mendoza
Chile
Río de La Plata
La Pampa
Buenos Aires
Capital Federal
Neuquén
Río Negro
Chubut
Oceano Atlántico
Santa Cruz
Is. Malvinas

ELECTROCARDIOGRAMA

Los hallazgos más frecuentes en el paciente con EC son:

- Bloqueo de rama derecha con/sin hemibloqueo anterior de rama izquierda
- Extrasístoles ventriculares, aisladas o repetitivas
- Alteración primaria de la repolarización ventricular
- Zonas eléctricamente inactivas (ondas q)
- Bloqueos auriculoventriculares
- Bloqueo de rama izquierda
- Bradicardia sinusal
- Taquiarritmia supraventricular

El bloqueo incompleto o completo de rama derecha solo o asociado a hemibloqueo anterior izquierdo es el principal hallazgo electrocardiográfico en los pacientes con EC seguido del hemibloqueo anterior izquierdo aislado

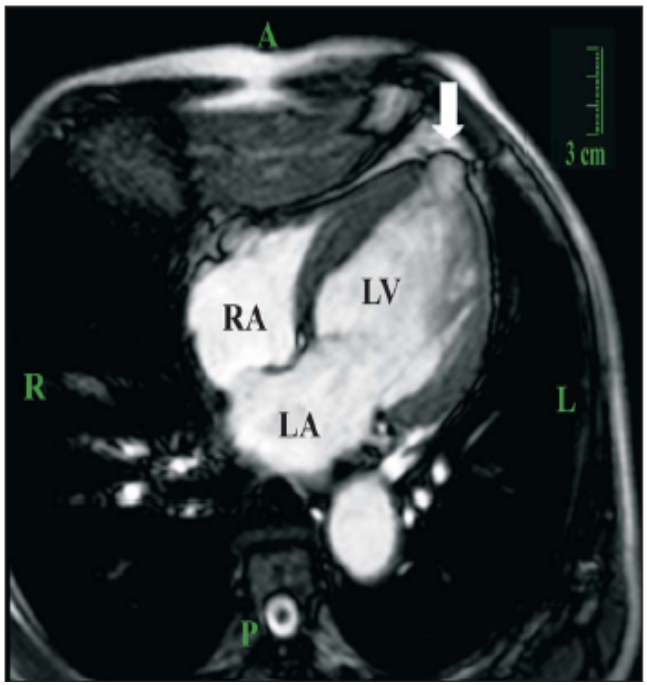


Storino R, Jörg M, Auger S: Manual práctico de la atención médica del paciente chagásico. Ed. Masson. Doyma, B

Ventricular aneurysm in a chronic Chagas disease patien
from the Brazilian Amazon region

Aneurisma ventricular em paciente chagásico crônico
da Amazônia brasileira

João Marcos Bemfica Barbosa Ferreira[1], Jorge Augusto de Oliveira Guerra[2,3]
and Maria das Graças Vale Barbosa[2,3]



Revista da Sociedade Brasileira de Medicina Tropical 42(4):474-475, jul-ago, 2009

estudio del paciente con serología positiva para determinar la fase de la enfermedad en la que se encuentra (indeterminada/crónica).

Los hallazgos más frecuentes en la cardiopatía chagásica son

•Alteraciones segmentarias de la contractilidad miocárdica (75% de los casos). Las regiones afectadas con más frecuencia son la pared posteroinferior y el ápex del ventrículo izquierdo.

•Aneurismas en el ápex del ventrículo izquierdo (generalmente de cuello estrecho con trombos murales)

•Hipocinesia o acinesia de la pared posteroinferior

•Afectación de la porción basal del septo anterior , con acinesia o incluso formación de aneurismas subaórticos

•Miocardiopatía dilatada

•Dilatación y disfunción del ventrículo derecho

Gascón J, Albajar P, Cañas E, Flores M, Gómez i Prat J, Herrera RN, Lafuente CA, Luciardi HL, Moncayo A, Molina L, Muñoz J, Puente S, Sanz G, Treviño B, Sergio-Salles X; Working Group of the second workshop on "Imported Chagas' Disease, a New Challenge in Public Health"; Spanish Society of Tropical Medicine and International Health. Diagnóstico, manejo y tratamiento de la cardiopatía chagásica en áreas donde la infección por *trypanosoma cruzi* no es endémica. Enferm Infecc Microbiol Clin 2008

AFECTACION DIGESTIVA

La **denervación de los plexos del tubo digestivo ocasionan trastornos de la motilidad con incoordinación motora y dilatación.**

El megaesófago y el megacolon caracterizan la fase crónica gastrointestinal de la EC, aunque pueden afectarse otros tramos del tracto digestivo. Es más frecuente en personas procedentes de países del cono sur a diferencia de los andinos donde predominan las alteraciones cardíacas.

Esofago:

La disfagia es el síntoma más frecuente, en fases avanzadas podemos encontrar cuadros de regurgitación, dolor retroesternal, pirosis, hipertrofia de las glándulas salivares y desnutrición.

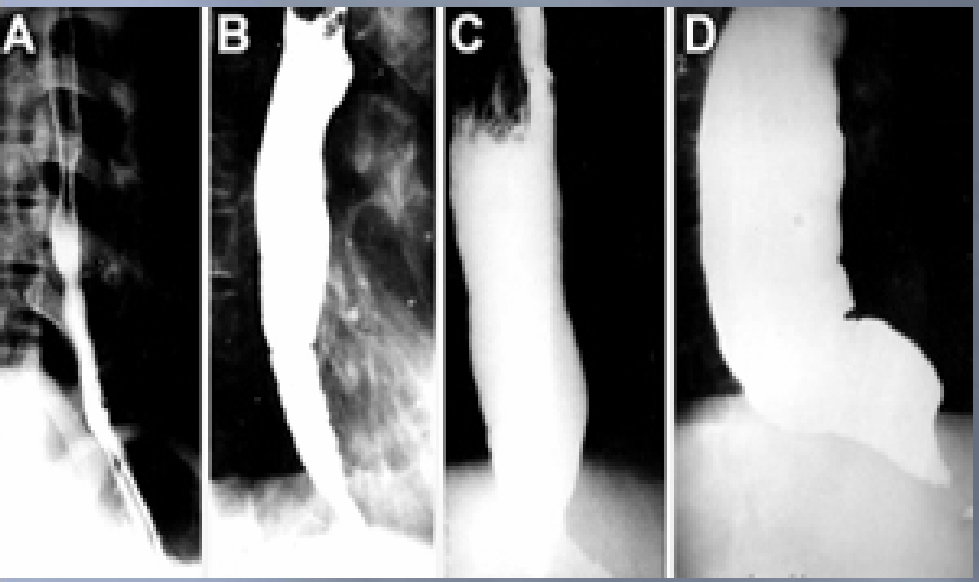
Colon:

La existencia de un megacolon en un adulto procedente de un área endémica debe hacernos sospechar la etiología chagásica del mismo. El síntoma más frecuente es el estreñimiento. Pueden aparecer también distensión, meteorito, vólvulos y en fases muy avanzadas puede requerir tratamiento quirúrgico. **Los segmentos más afectados son el recto y el colon sigmoide.**

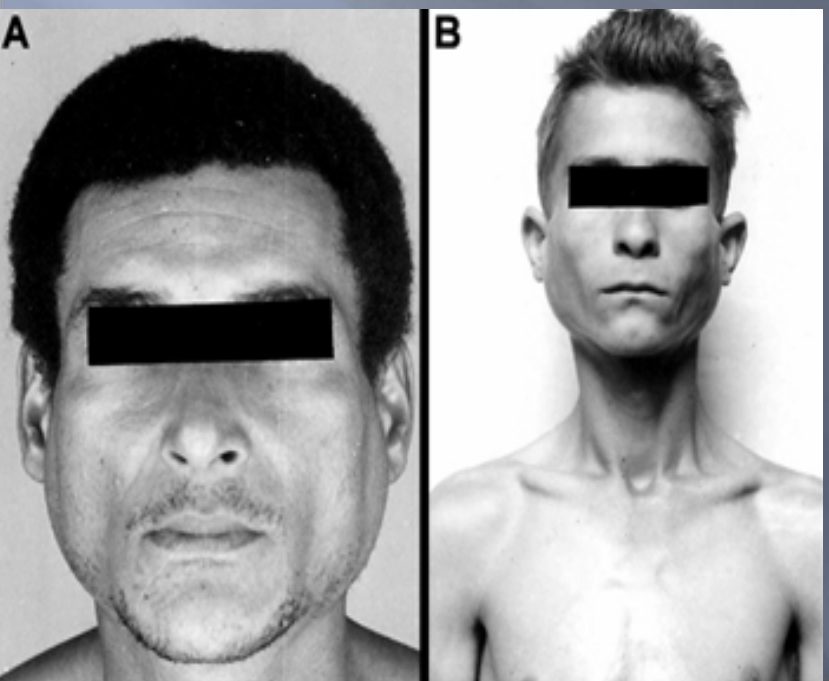
Diagnosis, management and treatment of chronic Chagas' gastrointestinal disease in areas where Trypanosoma cruzi infection is not endemic.

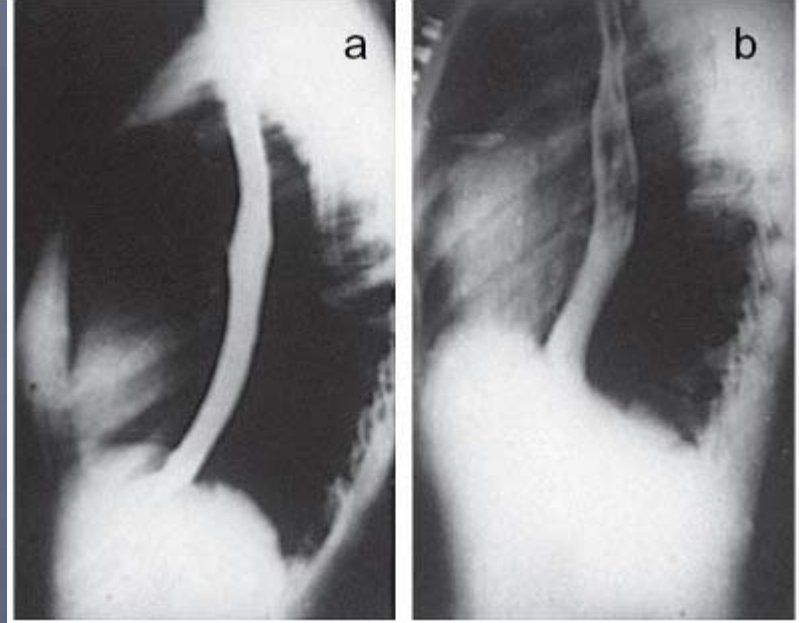
Pinazo MJ, Cañas E, Elizalde JI, Gascón J, Gimeno F, Gomez J, Guhl F, Ortiz V, Posada ED, Puente S, Rezende J, Salas J, Saravia J, Faustino, Torrus D, Treviño B

Gastroenterol Hepatol. 2009 Oct 17. [Epub ahead of print



Rezende





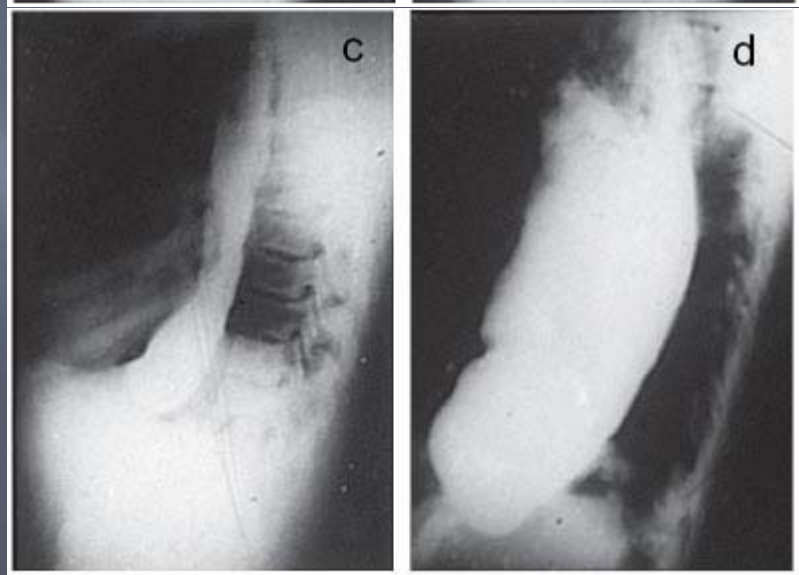
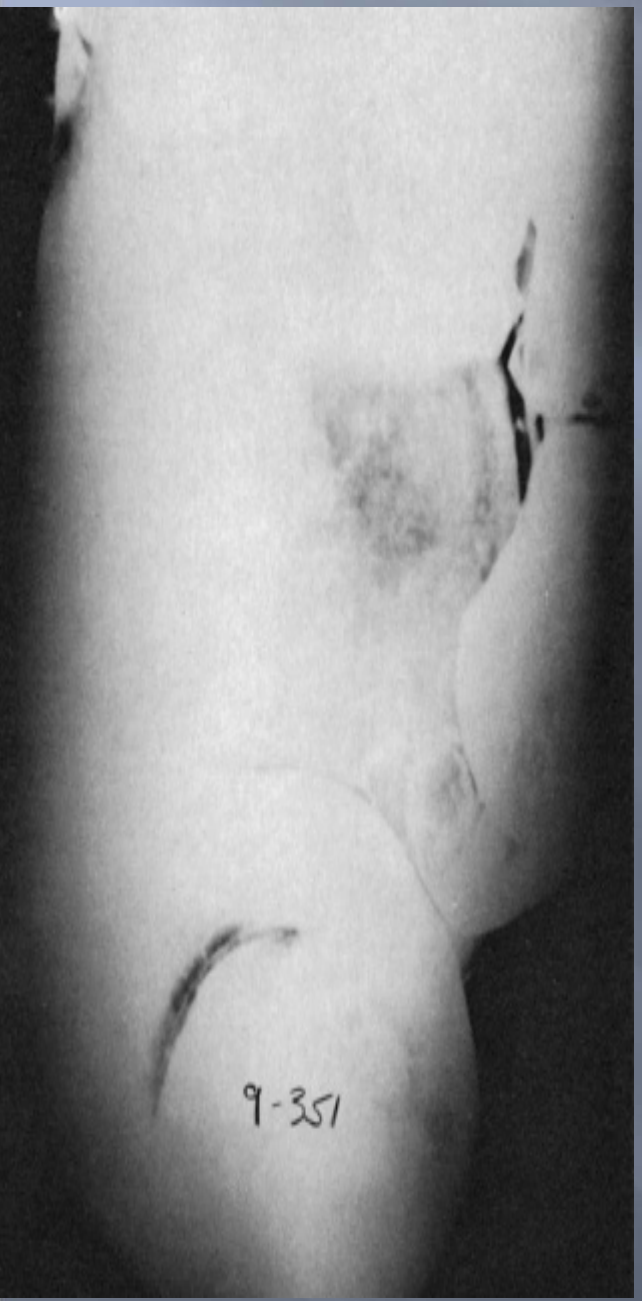
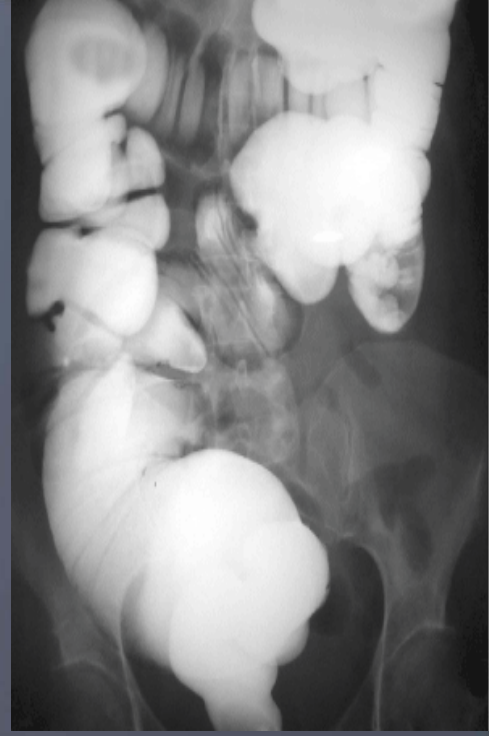
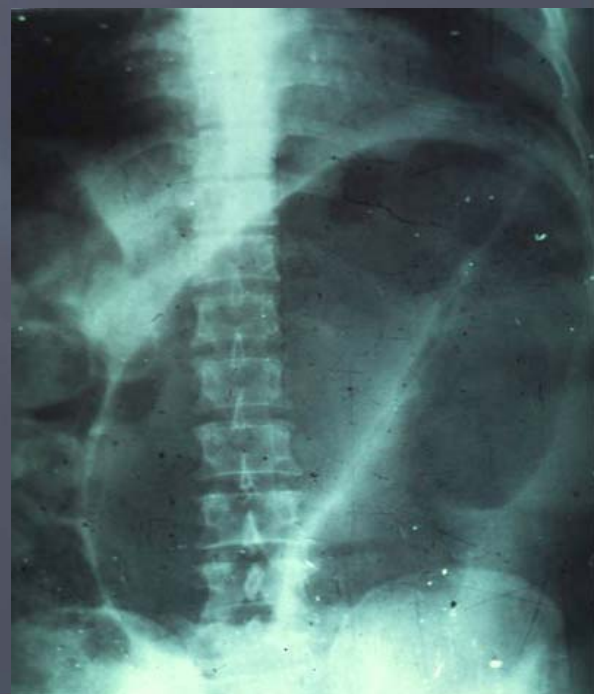


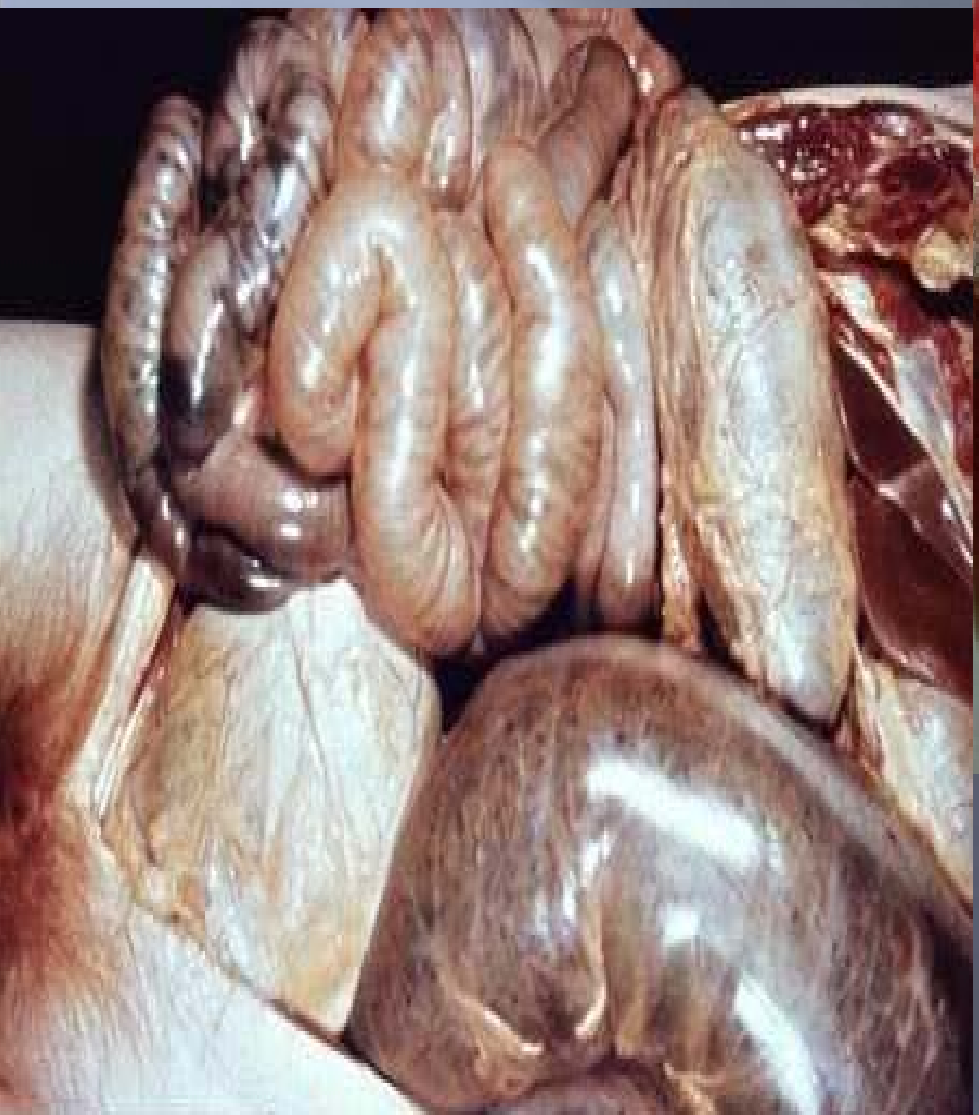
Fig. 7: esophagus dysperistalsis grade I (a), and megaesophagus grade II (b), grade III (c) and grade IV (d) (Coura et al. 2007).

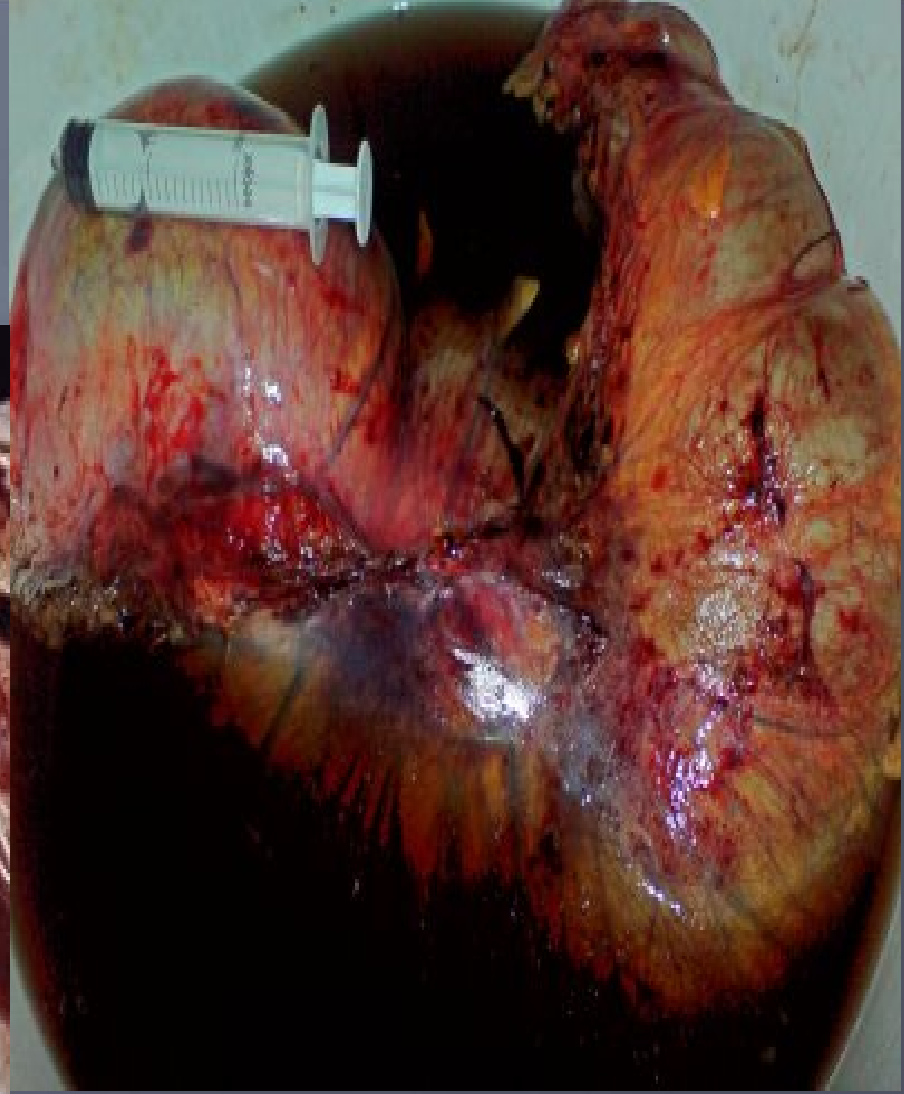

9-351


9-351









Mem Inst Oswaldo Cruz, Rio de Janeiro, Vol. *102*(Suppl. I): 113-122, 2007

Chagas disease: what is known and what is needed – A background article

José Rodrigues Coura

Laboratório de Doenças Parasitárias, Instituto Oswaldo Cruz-Fiocruz, Av. Brasil 4365, 21045-900 Rio de Janeiro, Brasil

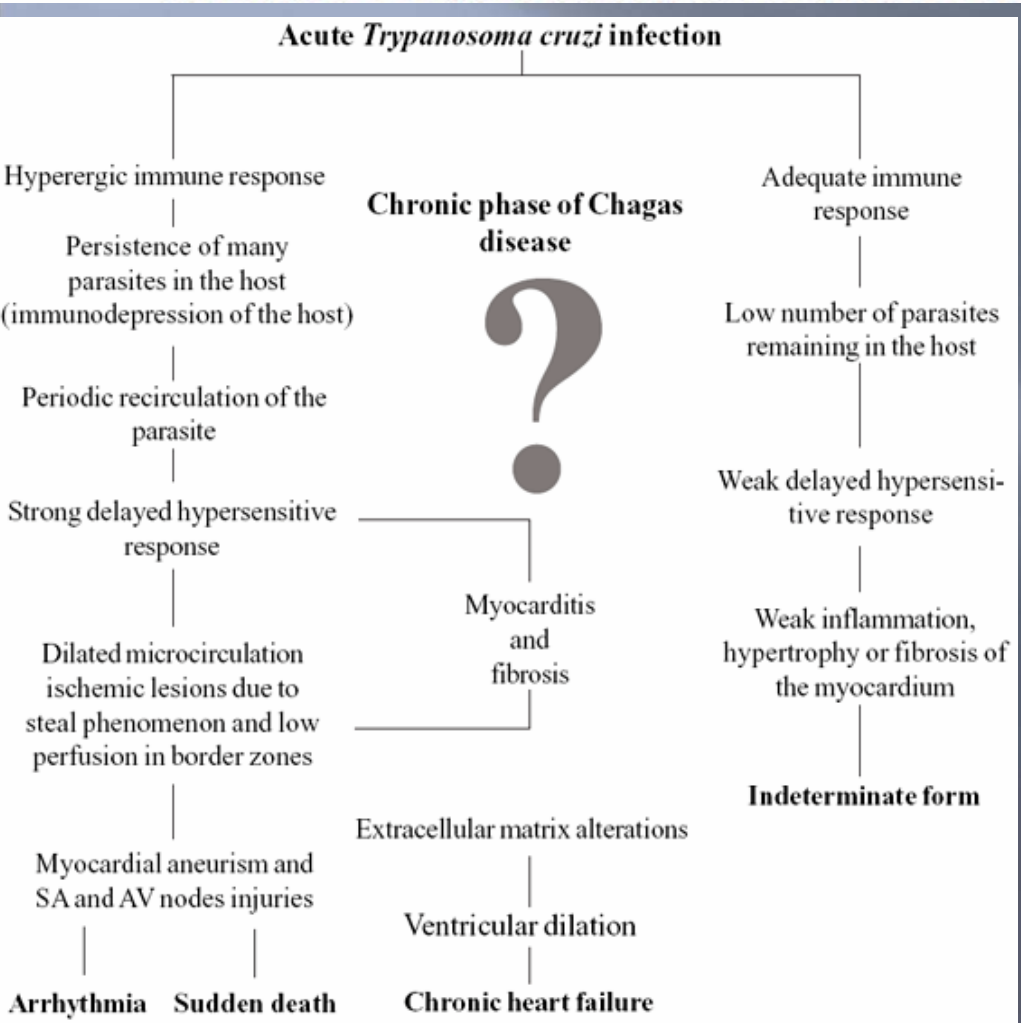


Fig 3: evolutive schedule of pathogenesis of Chagas heart disease (adapted from Higushi 1999).

La patogenia de la enfermedad en la actualidad no es del todo conocida , aunque la tendencia es a pensar en una etiología mixta:

Daño tisular directo por parte del *trypanosoma cruzi*

Mecanismo mediado por la **respuesta inmunológica** del huésped frente al microorganismo

Alteraciones **microvasculares**

Denervación autonómica.

Todos estos factores podrían justificar los síntomas y los signos de la fase crónica de la enfermedad.

La razón por la cual solo un porcentaje de pacientes llegan a esta fase mientras otros permanecen asintomáticos toda la

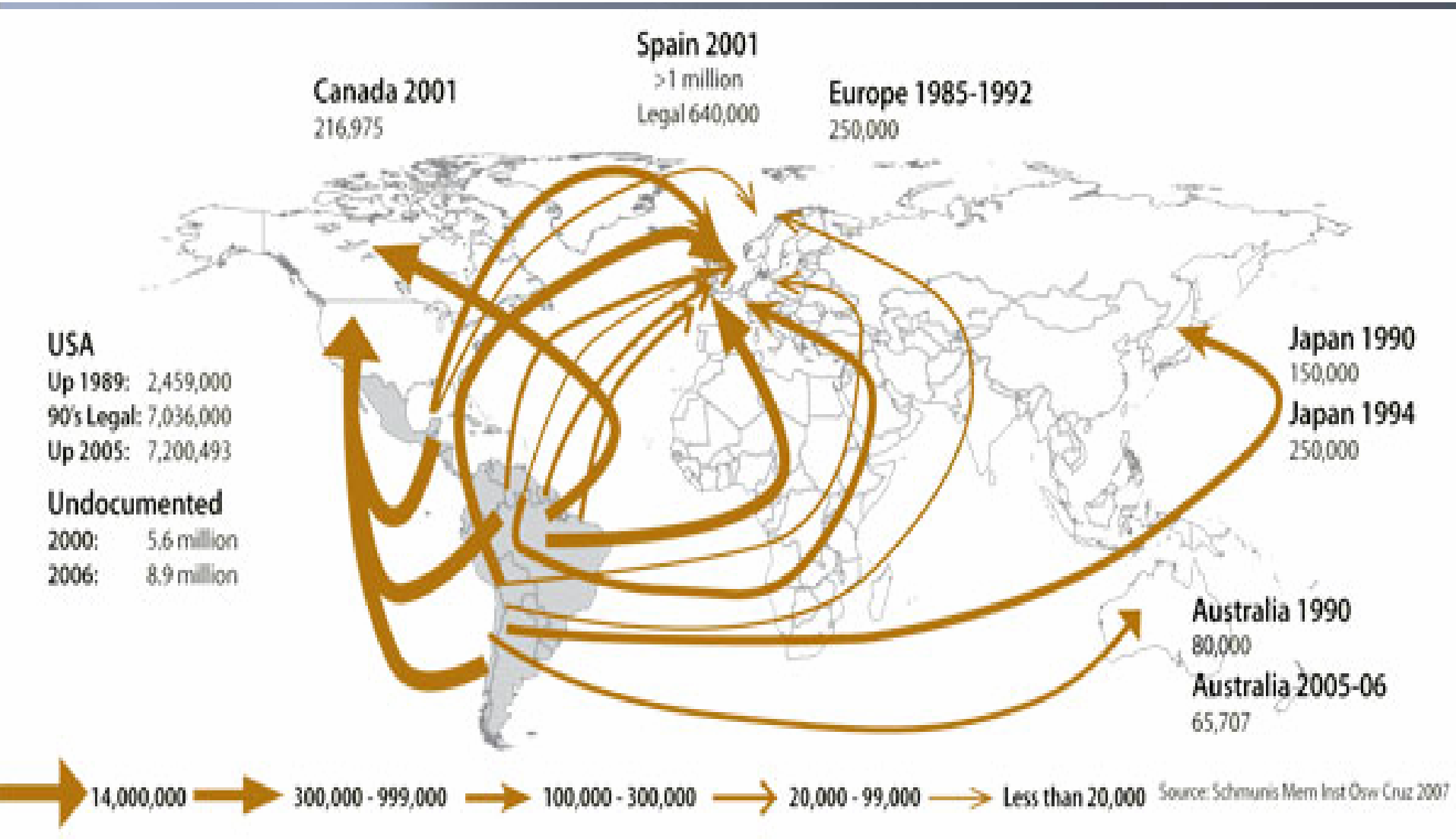
ENFERMEDAD DE CHAGAS IMPORTADA

Mem Inst Oswaldo Cruz, Rio de Janeiro, Vol. *102*(Suppl. I): 75-85, 2007

Epidemiology of Chagas disease in non-endemic countries: the role of international migration

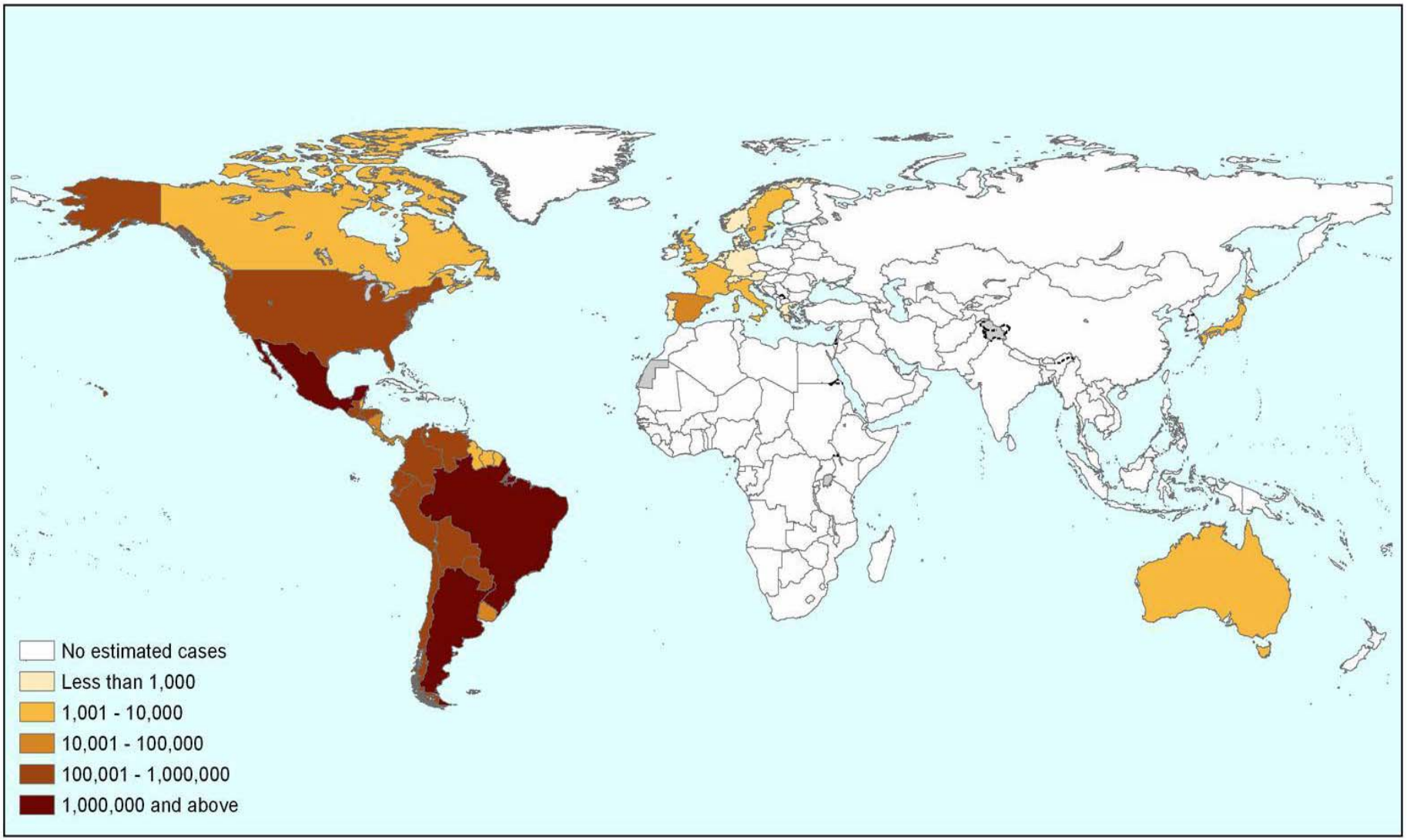
Gabriel A Schmunis

Pan American Health Organization/World Health Organization, 525 23rd Street, NW Washington, DC 20037, USA



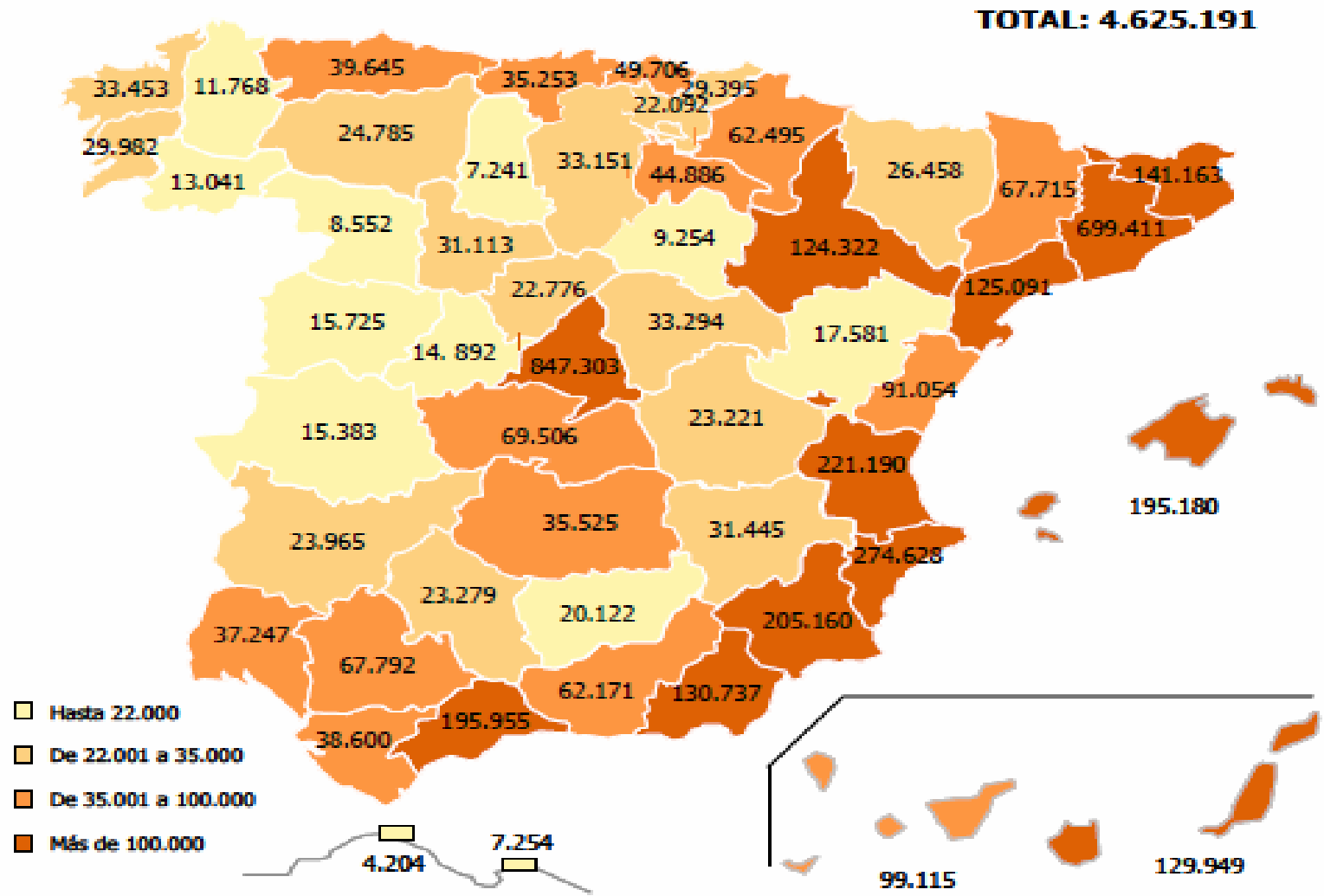
We will update this map regurlarly (version: June 2009)

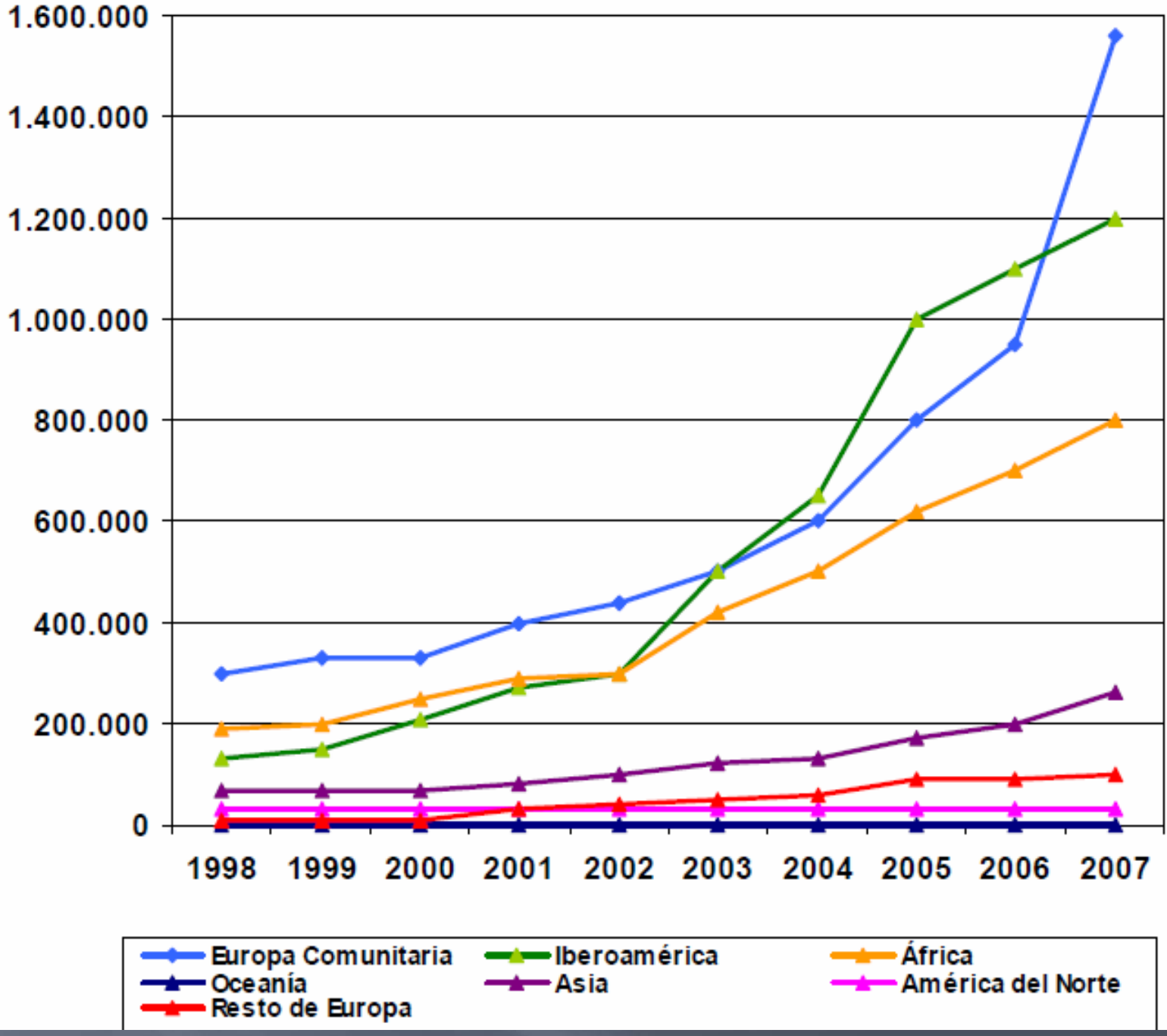
Estimated global population infected by *Trypanosoma cruzi*, 2009



Sources:
1. OPS/HDM/CD/425-06 Estimación cuantitativa de la enfermedad de Chagas en las Américas.
2. Guerri-Guttenberg RA, Grana D.R., Giuseppe Ambrosio, Milei.J. Chagasic cardiomyopathy: Europe is not spared! European Heart Journal (2008); 29: 2587-2591.
3. Schmunis. G. A. Epidemiology of Chagas Disease in non-endemic countries: the role of international migration. Mem Inst Oswaldo Cruz, Rio de Janeiro, Vol. 102(Suppl. I): 75-85, 2007.
4. De Ayala A.P, Pérez-Molina J.A, Norman F., and López-Vélez R.Chagasic cardiomyopathy in inmigrants from Latin America to Spain. Emerging Infectious Disease Volume 15, Number 4–April 2009.
5. According to the numbers of inmigrants registered for 2007in the website of the Japanese Ministry of Justice and estimated seroprevalence for non endemic countries according to Paricio-Talayero J.M. Vigilancia epidemiológica de la transmisión vertical de la enfermedad de Chagas en tres maternidades de la Comunidad Valenciana. Enferm Infecc Microbiol Clin 2008;26(10):609-13.

Gráfico 4. Extranjeros con certificado de registro o tarjeta de residencia según provincia. 30-06-2009




1.600.000
1.400.000
1.200.000
1.000.000
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600.000
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1998
1999
2000
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2002
2003
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2006
2007
Europa Comunitaria
Iberoamérica
África
Oceanía
Asia
América del Norte
Resto de Europa

PLAN DIRECTOR DE INMIGRACIÓN Y CONVIVENCIA 2008-2011

	COMUNITAT VALENCIANA			ALICANTE			CASTELLÓN			VALENCIA		
	TOTAL	HOMBRES	MUJERES	TOTAL	HOMBRES	MUJERES	TOTAL	HOMBRES	MUJERES	TOTAL	HOMBRES	MUJERES
Reino unido	134.874	67.688	67.186	121.711	60.789	60.922	1.836	946	890	11.327	5.953	5.374
Rumania	127.750	67.941	59.809	30.072	16.010	14.062	51.947	27.027	24.920	45.731	24.904	20.827
Marruecos	65.534	41.956	23.578	29.419	18.843	10.576	16.666	10.512	6.154	19.449	12.601	6.848
Ecuador	51.402	26.294	25.108	22.983	12.122	10.861	2.285	1.128	1.157	26.134	13.044	13.090
Colombia	43.142	19.900	23.242	19.923	9.327	10.596	4.499	2.018	2.481	18.720	8.555	10.165
Alemania	41.613	21.126	20.487	35.367	17.840	17.527	1.834	962	872	4.412	2.324	2.088
Bulgaria	34.272	19.260	15.012	11.946	6.724	5.222	1.046	563	483	21.280	11.973	9.307
Bolivia	30.378	13.529	16.849	6.491	3.083	3.408	189	74	115	23.698	10.372	13.326

ESTIMACION DEL IMPACTO DE LA CARDIOPATIA CHAGASICA EN POBLACION INMIGRANTE LATINOAMERICANA EN EUROPA

M. Navarro. et al
Hospital Ramón y Cajal de Madrid

320 muestras de suero se realizaron serología de EC

Positivas 92 (28.7%)

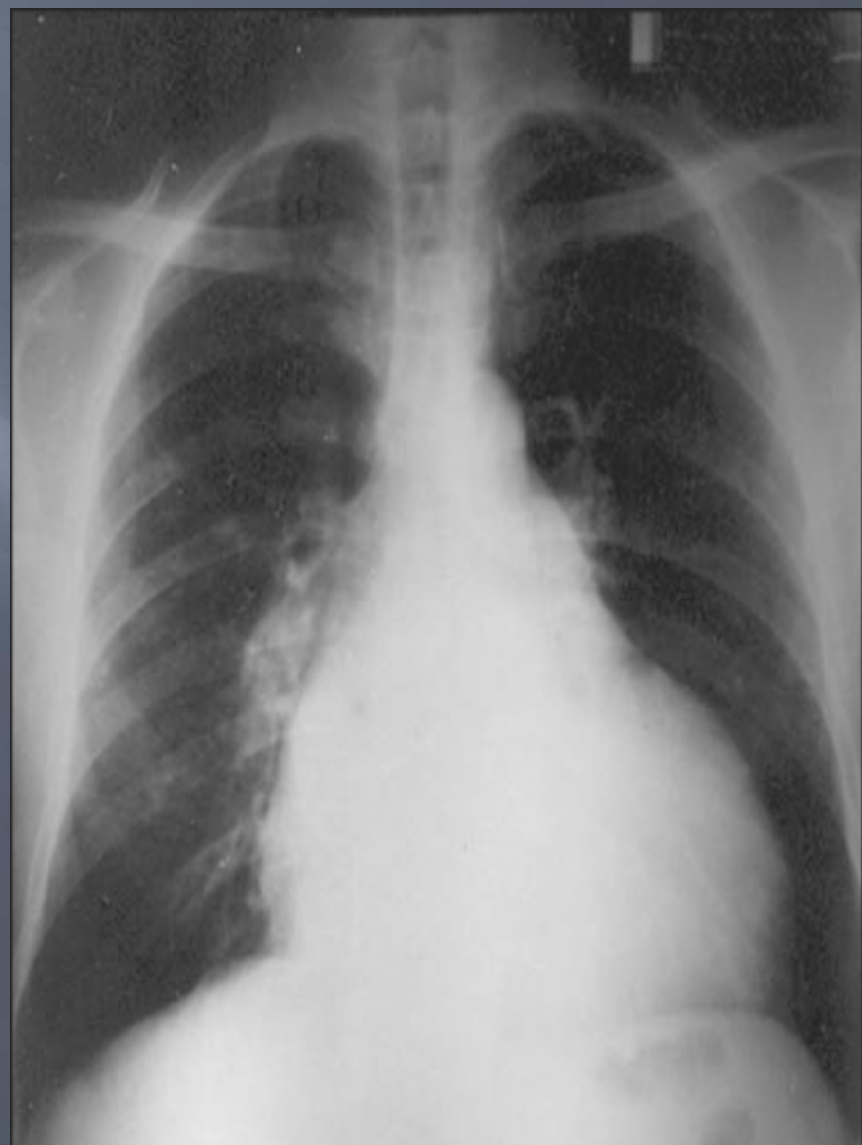
57% síntomas cardiacos

22.5% alteraciones en el ECG

1.307.444 inmigrantes de aréas endémicas

225000 infecciones

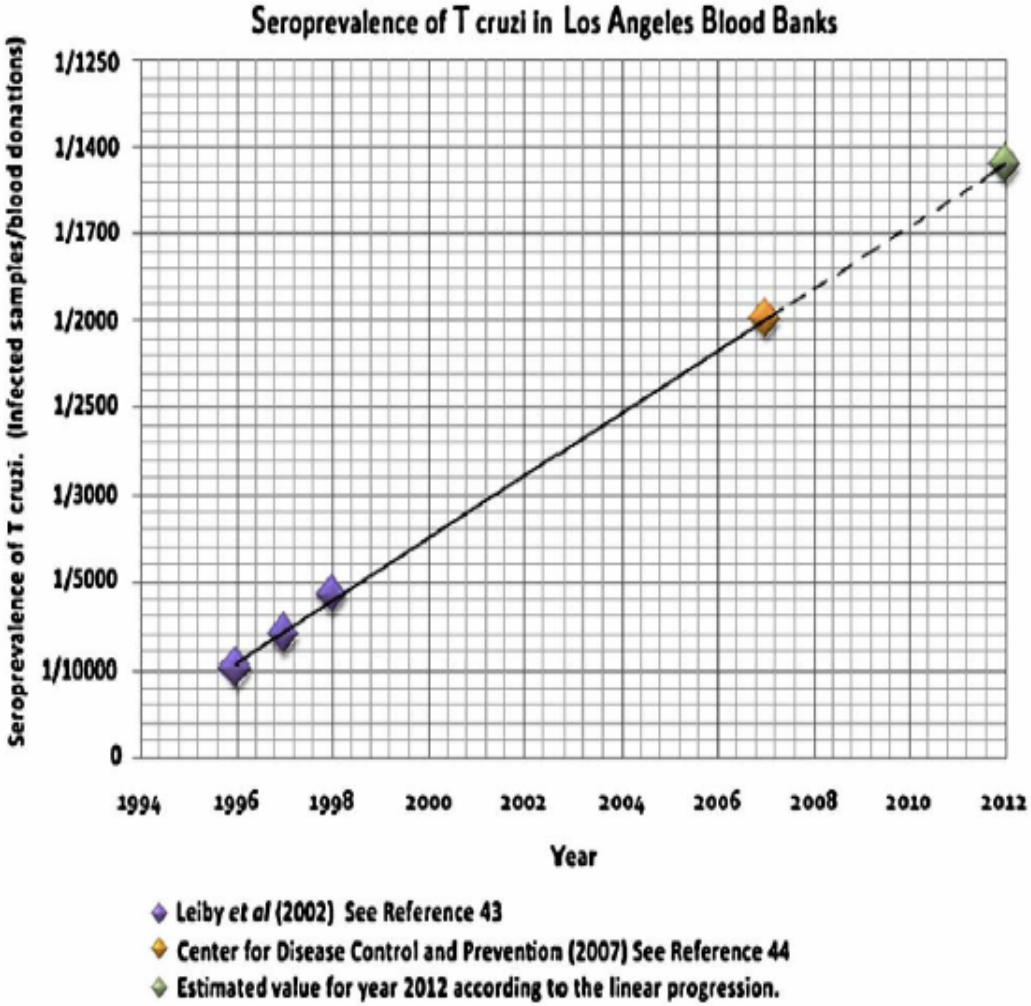
67500 cardiopatías



XIII CONGRESO SEIMC MADRID 11-14 MAYO 2008

Prognostic impact of Chagas disease in the United States

José Milei, MD, PhD, Roberto Andrés Guerri-Guttenberg, MD, Daniel Rodolfo Grana, VMD, and Rubén Storino, MD, PhD *Buenos Aires, Argentina*



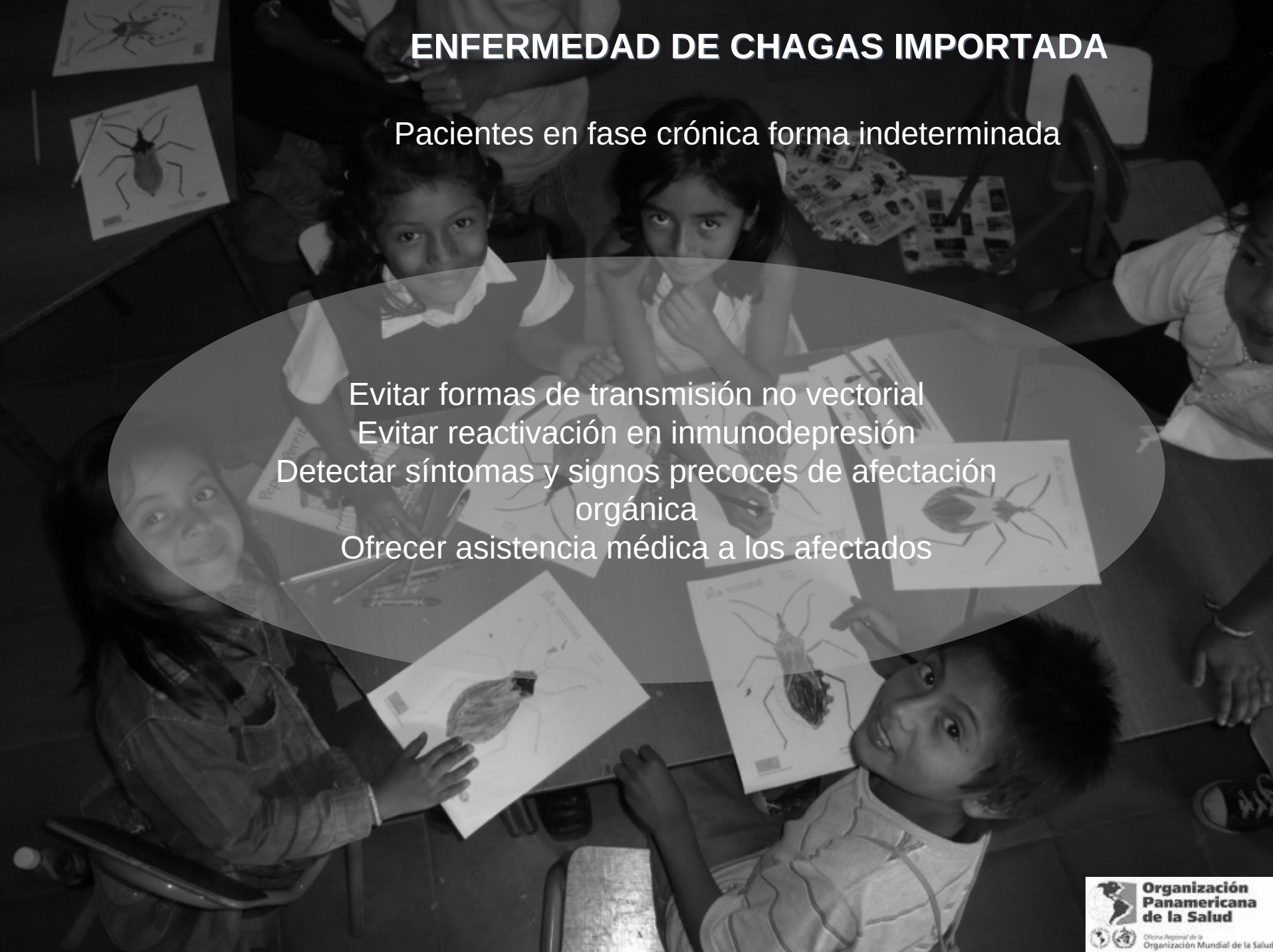
Am Heart J 2009;157:22-9.

According to an estimation made in 1992 by Milei et al, 370,000 people had *T cruzi* infection in the United States and 75,000 of those had ChrChC.[3] According to the Pan American Health Organization and World Health Organization,[52] around 89,221 to 693,302 infected Latin Americans migrated to the United States in the period 1981 to 2005. Mexican-infected immigrants range

ENFERMEDAD DE CHAGAS IMPORTADA

Pacientes en fase crónica forma indeterminada

Evitar formas de transmisión no vectorial
Evitar reactivación en inmunodepresión
Detectar síntomas y signos precoces de afectación orgánica
Ofrecer asistencia médica a los afectados



Organización Panamericana de la Salud
Oficina Regional de la Organización Mundial de la Salud

Todos los componentes sanguíneos y hemoderivados tienen capacidad de transmisión, siendo los concentrados de **plaquetas** los de mayor riesgo.

Se estima que la tasa de infección a través de productos sanguíneos procedentes de personas infectadas es del 20%.

El parásito resiste la refrigeración a 4ºC durante más de 18 días, la criopreservación y la descongelación posterior.

Desde 1980 se han descrito 7 casos de transmisión de la enfermedad por transfusión de productos hematológicos en Estados Unidos y Canadá, también se han producido en nuestro país de forma previa a la ley del 2005.

Es posible, sin embargo, que se hayan producido más casos de transmisión que por ser el receptor inmunocompetente produjeran una clínica leve o incluso inexistente, pasando desapercibidos.

El primer caso conocido de transmisión a través de la transfusión fue en el año 1984, tras un trasplante de médula y fue publicado en 1992 (Villalba et al. Clin Infect Dis 1992)

Schmunis G, Cruz JR. Safety of the blood suplía in Latin América. Clin microbial Rev 2005;18
Young C, Losikoff P, Chawla A, Glasser L., Forman E. Trasfusion-acquired *Trypanosoma Cruzi* infecton. Transfusion 2007 ;47(3)
Florez-Chavés M, Fernández B, Puente S, Torre P, Rodriguez M, Monedero C, Cruz I, Gárate T, Cañavate C. Transfusional Chagas disease: Parasitological and serological monitoring of an infected recipiente and blood donor. CID 2008; 46:

COMUNIDADES AUTÓNOMAS	2004			2005			2006			2007		
	Unidades Testadas	Unidades positivas confirmadas	%	Unidades Testadas	Unidades positivas confirmadas	%	Unidades Testadas	Unidades positivas confirmadas	%	Unidades Testadas	Unidades positivas confirmadas	%
ANDALUCÍA							72	1	1,39	1.439	19	1,32
ARAGÓN										85	0	0,00
ASTURIAS	304	0					184	0	0,00	201	2	1,00
BALEARES							195		0,00	318	0	0,00
CANARIAS							1.227	16	1,30	4.878	18	0,37
CANTABRIA							81	1	1,23	148	0	0,00
C.MANCHA							20	1	5,00	6	0	0,00
C.LEON							442	0	0,00	380	1	0,26
CATALUÑA				697	6	0,86	4.653	16	0,34	8.194	24	0,29
EXTREMADURA							0	0		5	0	0,00
GALICIA				130			1.812	1	0,06	1.087	0	0,00
MADRID	509	6	1,18	1.539	16	1,04	3.819	37	0,97	3.822	35	0,92
MURCIA							488	3	0,61	483	3	0,62
NAVARRA							87		0,00	104	0	0,00
PAIS VASCO				74	1	1,35	336	5	1,49	403	2	0,50
LA RIOJA							31	0	0,00	32	1	3,13
VALENCIA	465	5	1,08	1.386	13	0,93	2.369	1	0,04	4.599	16	0,35
TOTAL	1.278	11	0,86	3.826	36	0,94	15.816	82	0,52	26.184	121	**0,46**

Fuente: Estadística Estatal de Centros y Servicios de Transfusión

Seroprevalence of *Trypanosoma cruzi* infection in at-risk blood donors in Catalonia (Spain)

Maria Piron, Mireia Vergés, José Muñoz, Natàlia Casamitjana, Sergi Sanz, Rosa María Maymó, José Manuel Hernández, Lluís Puig, Montserrat Portús, Joaquim Gascón, and Sílvia Sauleda

TRANSFUSION Volume 48, September 2008

RESULTS: Overall seroprevalence was 0.62 percent, with 11 donors confirmed positive among the 1770 at-risk donors studied; the highest rate (10.2%) was in Bolivian donors. Interestingly, 1 of the 11 positive donors was a Spaniard who had resided various years in a Chagas disease endemic area. Furthermore, 1 of the positive donors presented detectable parasitemia.

TABLE 3. Distribution of donors born in an endemic region and of positive donors by country of origin

Country	Tested for anti-*T. cruzi**	Percentage of official immigrant population in Catalonia	Anti-*T. cruzi*–positive donors	
			Number	Rate by country (%)
Colombia	340 (22.3)	13.8		
Argentina	298 (19.5)	11.7	2	2/298 (0.67)
Ecuador	223 (14.6)	29.2	1	1/223 (0.45)
Uruguay	127 (8.3)	4.4		
Peru	123 (8.1)	8.9		
Brazil	113 (7.4)	3.9		
Venezuela	86 (5.6)	2.4		
Chile	77 (5.0)	4.2		
Bolivia	59 (3.9)	8	6	6/59 (10.2)
Mexico	40 (2.6)	2.6		
Paraguay	15 (1.0)	1.1	1	1/15 (6.7)
Honduras	10 (0.7)	1.3		
El Salvador	6 (0.4)	0.4		
Nicaragua	3 (0.2)	0.1		
Costa Rica	2 (0.1)	0.1		
Guatemala	1 (<0.1)	0.1		
Panama	1 (<0.1)	0.1		
Total	1524		10	

* Data are reported as number (%).

Transfusional Chagas Disease: Parasitological and Serological Monitoring of an Infected Recipient and Blood Donor

María Flores-Chávez,[1] Begoña Fernández,[2] Sabino Puente,[3] Pilar Torres,[2] Mercedes Rodríguez,[1] Carolina Monedero,[1] Israel Cruz,[1] Teresa Gárate,[1] and Carmen Cañavate[1]

[1]Servicio de Parasitología, Centro Nacional de Microbiología, Instituto de Salud Carlos III, and [2]Centro de Transfusión de la Comunidad de Madrid, and [3]Unidad de Enfermedades Tropicales, Hospital Carlos III, Madrid, Spain

Chagas disease is endemic to Latin America, but human migration is extending its distribution. This report describes the parasitological and serological course of disease in a Spanish patient fatally infected via a blood product transfusion, as well as the monitoring of the donor. Before undergoing immunosuppression, multitransfused patients should be screened for anti–*Trypanosoma cruzi* antibodies.

Results. The Spanish patient was a 25–year-old man who had a history of leukemia [9] that eventually required a cord blood transplant; he received blood derivatives from at least 176 persons who donated blood at different transfusion centers. In January 2005, 45 days after infection onset, the patient was examined for fever of unknown origin. None of the infectious

Clinical Infectious Diseases 2008; 46:e44–7

MINISTERIO DE SANIDAD Y CONSUMO

15514 *REAL DECRETO 1088/2005, de 16 de septiembre, por el que se establecen los requisitos técnicos y condiciones mínimas de la hemodonación y de los centros y servicios de transfusión.*

Martes 20 septiembre 2005

B. *Criterios de exclusión de donantes*

Si la donación se destina exclusivamente al fraccionamiento del plasma, no se requieren las pruebas y los periodos de exclusión señalados con un asterisco (*).

1. Criterios de exclusión permanente para donantes homólogos. Se excluirá de forma definitiva a los candidatos a donantes con:

1.1 Enfermedad cardiovascular: padecer o haber padecido enfermedad cardiovascular grave, excepto anomalías congénitas curadas.

1.2 Enfermedad del sistema nervioso central (SNC): historia de enfermedad del SNC grave.

1.3 Diátesis hemorrágica: historia de coagulopatía hemorrágica.

1.4 Episodios repetidos de síncope, o antecedentes de convulsiones:

a) Exclusión definitiva si existe historia de epilepsia bajo tratamiento continuado. Se aceptarán los que en los últimos tres años no presentaron crisis ni requirieron tratamiento anticonvulsivante.

b) Se descartarán las personas con episodios convulsivos no etiquetados, estén o no sometidas a tratamiento. Los antecedentes de síncopes o convulsiones en la infancia o adolescencia no son motivo de exclusión.

1.5 Enfermedad gastrointestinal, genitourinaria, hematológica, inmunológica, metabólica, renal o respiratoria grave, activa, crónica o recidivante.

1.6 Diabetes que precisa tratamiento con insulina.

1.7 Hipertensión arterial grave.

1.8 Enfermedades infecciosas. Padecer o haber padecido:

a) Hepatitis B: excepto las personas negativas al antígeno de superficie de la hepatitis B (AgHBs), cuya inmunidad haya sido demostrada.

b) Hepatitis C.

c) Síndrome de Inmunodeficiencia Adquirida o ser portador del VIH I/II.

d) Infección por Virus Linfotrópico Humano de células T (HTLV I/II) o ser portador de anticuerpos anti-HTLV I/II.

e) Babesiosis*.

f) Kala Azar (Leismaniosis visceral)*.

g) Tripanosomiasis americana por Tripanosoma Cruzi (enfermedad de Chagas)*: los donantes nacidos, o hijos de madres nacidas, o que han sido transfundidos en países donde la enfermedad es endémica, podrán ser aceptados si una prueba validada, dirigida a la detección de portadores de la enfermedad, resulta negativa.

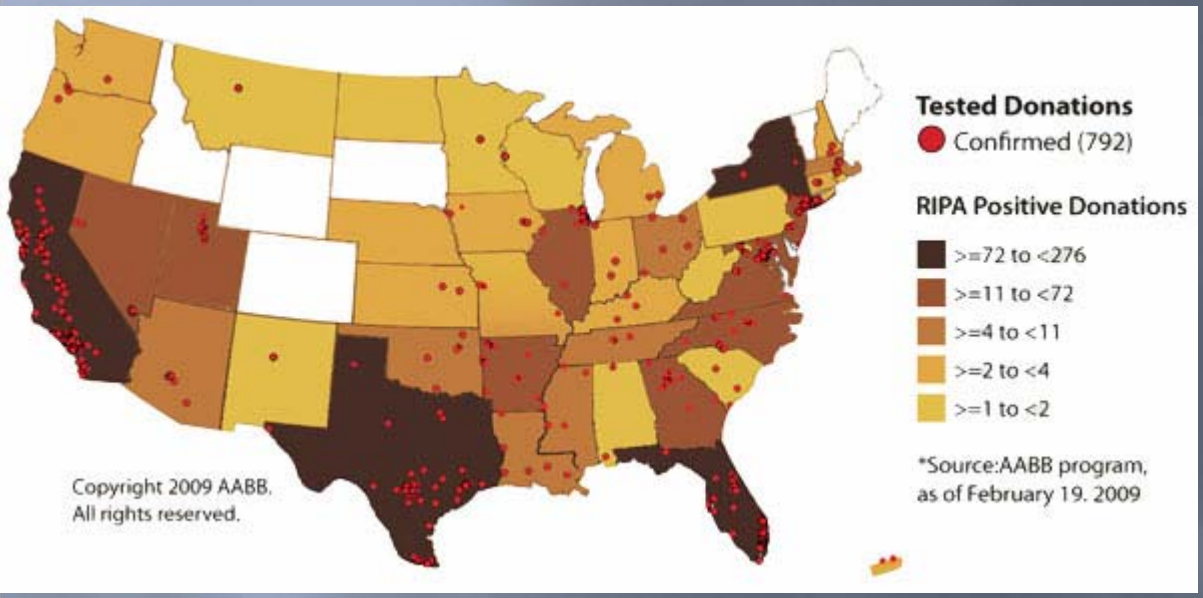
2 pruebas serológicas negativas

1.9 Cáncer: excepto tumor localizado con completa recuperación.

1.10 Encefalopatías espongiformes transmisibles; enfermedad de Creutzfeldt-Jacob y variante de la enfermedad de Creutzfeldt-Jacob: personas con antecedentes familiares, o personas que hayan sido sometidas a trasplante de córnea o duramadre o que en el pasado hubieran recibido tratamiento con medicamentos derivados de glándula pituitaria humana. Quedan asimismo excluidas las personas con estancia superior a 12 meses en el Reino Unido durante el periodo 1980-1996.

1.11 Consumo de drogas: antecedente de consumo de drogas por vía intravenosa o intramuscular no prescritas, incluido tratamiento esteroideo u hormonal para

Blood donors confirmed positive for Chagas Disease, 2007-2009*, n=792



CDC
CENTERS FOR DISEASE CONTROL AND PREVENTION

España (desde Sept. 2005)
Francia (desde Enero 2007)
En Estados Unidos, el cribado para T.cruzi es universal y obligatorio desde Enero de 2007

La madre india Santa Cruz

GENERALITAT VALENCIANA
CONSELLERIA DE SANITAT

AGÈNCIA VALENCIANA DE SALUT

Direcció General de Salut Pública

CIRCULAR 3/2007/8/1

Se incluye el **screening de enfermedad de Chagas a toda gestante de riesgo para es**

Protocolo de actuación ante el recién nacido de madre con serología positiva

0.7 al 10% de sus hijos presentarán Chagas congénito

Esta transmisión depende del nivel de parasitemia y de la inmunidad materna

De estos **recién nacidos el 80% serán asintomáticos y en un 20% presentarán sintomatología** (prematuridad, bajo peso, hepatoesplenomegalia, ictericia, fiebre, meningoencefaliis, miocarditis …). La mortalidad puede ser del 5%

Herman E, Truyens C, Alonso–Vega C, Rodriguez P, Berthe R, Torrico F, Carlier Y. Congenital Trasmision of *trypanosoma cruzi* is associated with maternal enhanced parasitemia and drecreased productins of interferon gamma in response to parasite antigens. J infect Dis 2004; 189(7)

Vigilancia epidemiológica de la transmisión vertical de la enfermedad de Chagas en tres maternidades de la Comunidad Valenciana

José María Paricio-Talayero[a], María José Benlloch-Muncharaz[a], José Ignacio Collar-del-Castillo[b], Amparo Rubio-Soriano[c], Concepción Serrat-Pérez[d], Josefa Magraner-Egea[d], Leonardo Landa-Rivera[a], Marta Sánchez-Palomares[a], Beatriz Beseler-Soto[a], Luis Santos-Serrano[a], Manuel Ferriol-Camacho[a], José Mut-Buigues[a], Miguel Tomás-Vila[b], María del Carmen Alonso-Jiménez[e], Victoria Domínguez-Márquez[e] y Rafael Igual-Adell[e]

Servicio de Pediatría. [a]Hospital Marina Alta. Denia. Alicante. [b]Hospital Francesc de Borja. Gandía. Valencia. [c]Hospital Virgen de los Lirios. Alcoi. Alicante. Servicio de Microbiología. [d]Hospital Marina Alta. Denia. Alicante. [e]Hospital Francesc de Borja. Gandía. Valencia. España.

Enferm Infecc Microbiol Clin 2008;26(10):609-13

País de origen	Número de madres analizadas	Madres con test de Chagas positivo	
		n	% (IC 95 %)
Ecuador	195	3	1,5 (0-3,3)
Colombia	131	2	1,5 (0-3,6)
Bolivia	137	24	17,5 (11,2-23,9)
Argentina	60	0	—
Uruguay	26	0	—
Venezuela	18	0	—
Brasil	17	0	—
Perú	9	0	—
Otros*	31	0	—
Total	*624*	*29*	*4,7 (3,0-6,3)*

*Cuba 7, Chile 6, Paraguay 6, El Salvador 5, Honduras 3, México 2, Panamá 1, República Dominicana 1.
IC 95 %: intervalo de confianza del 95 %.

PREVALENCIA DE TRIPANOSOMIASIS AMERICANA EN MUJERES GESTANTES DE UN ÁREA DE SALUD. VALENCIA, 2005-2007 (*)

Rafael Manuel Ortí Lucas y María Cristina Parada Barba

Servicio de Medicina Preventiva. Hospital Clínico Universitario de Valencia.

Rev Esp Salud Pública 2009; 83: 543-555

País origen	Mujeres estudiadas	Serología positiva		Prevalencia
		N	%	
Argentina	50	5	13,5%	10,0%
Bolivia	77	20	54,1%	26,0%
Brasil	8	2	5,4%	25,0%
Colombia	63	3	8,1%	4,8%
Chile	8	1	2,7%	12,5%
Ecuador	118	2	5,4%	1,7%
El Salvador	2	0	0%	0%
Guatemala	2	0	0%	0%
Honduras	7	1	2,7%	14,3%
México	5	0	0%	0%
Nicaragua	5	1	2,7%	20,0%
Panamá	1	0	0%	0%
Paraguay	6	1	2,7%	16,7%
Perú	10	1	2,7%	10,0%
Uruguay	12	0	0%	0%
Venezuela	8	0	0%	0%
Total	383	37	100%	9,7%

Prevalence and Vertical Transmission of *Trypanosoma cruzi* Infection among Pregnant Latin American Women Attending 2 Maternity Clinics in Barcelona, Spain

José Muñoz,[1] Oriol Coll,[2] Teresa Juncosa,[4] Mireia Vergés,[5] Marta del Pino,[2] Victoria Fumado,[4] Jordi Bosch,[3] Elizabeth J. Posada,[1] Sara Hernandez,[2] Roser Fisa,[5] Josep Maria Boguña,[4] Montserrat Gállego,[5] Sergi Sanz,[1] Montserrat Portús,[5] and Joaquim Gascón[1]

Clinical Infectious Diseases 2009; 48:1736–40

We performed a prospective screening for *Trypanosoma cruzi* infection in 1350 Latin American pregnant women and their offspring in Barcelona, Spain. The rate of seroprevalence was 3.4%, and 7.3% of the newborns were infected. Routine screening and management programs in maternity wards may be warranted.

Fatal congenital Chagas' disease in a non-endemic area: a case report

María Flores-Chávez[1], Yamile Faez[2], José M Olalla[2], Israel Cruz[1], Teresa Gárate[1], Mercedes Rodríguez[1], Pilar Blanc[2] and Carmen Cañavate*[1]

Address: [1]Servicio de Parasitología, Centro Nacional de Microbiología, Instituto de Salud Carlos III, Ctra. Pozuelo-Majadahonda Km 2, 28220 Majadahonda, Madrid, Spain and [2]Hospital Carlos Haya, Av. Carlos Haya s/n, 29010 Málaga, Spain

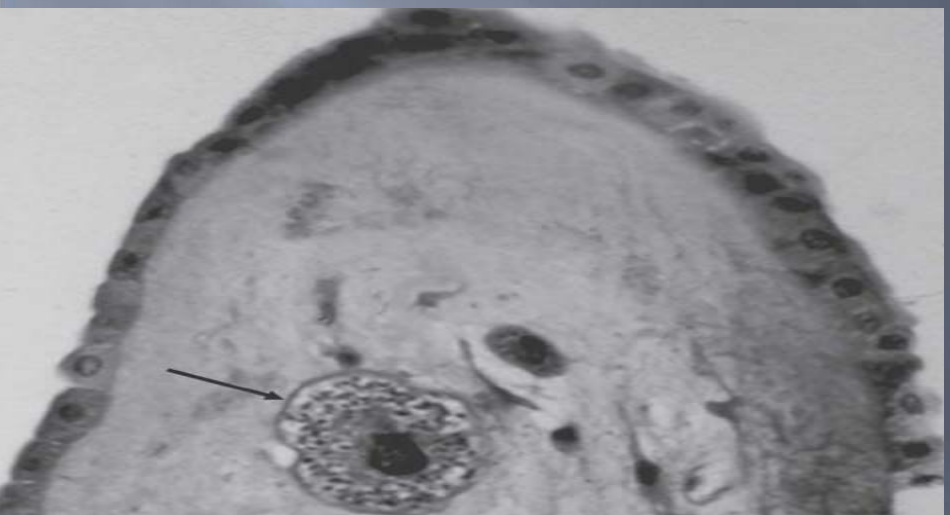
Cases Journal 2008, 1:302

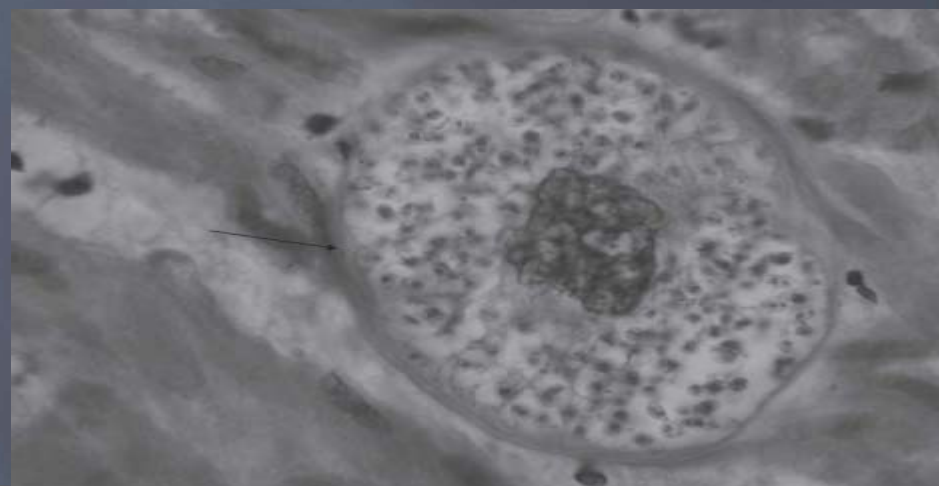
CONGENITAL TRANSMISSION OF *TRYPANOSOMA CRUZI* IN EUROPE (SPAIN): A CASE REPORT

CRISTINA RIERA,* ANNA GUARRO, HOUSSEIN EL KASSAB, JOSÉ MARIA JORBA, MONTSERRAT CASTRO, ROSER ANGRILL, MONTSERRAT GÁLLEGO, ROSER FISA, CARMEN MARTIN, ALEJANDRO LOBATO, AND MONTSERRAT PORTÚS

Laboratori de Parasitologia, Facultat de Farmàcia, Universitat de Barcelona, Barcelona, Spain; Servei de Pediatria, Hospital Residència Sant Camil, Sant Pere de Ribes, Barcelona, Spain; Servei d'Anatomia Patològica, Hospital Residència Sant Camil, Sant Pere de Ribes, Barcelona, Spain; Servei de Microbiologia, Hospital Residència Sant Camil, Sant Pere de Ribes, Barcelona, Spain

Am. J. Trop. Med. Hyg., 75(6), 2006,





La Laguna Verde Reserva Nacional de Fauna Andina Eduardo Abaroa, Bolivia

El compromiso de la inmunidad celular, como es el caso de los pacientes con VIH, predispone de forma especial a los cuadros de reactivación de la EC

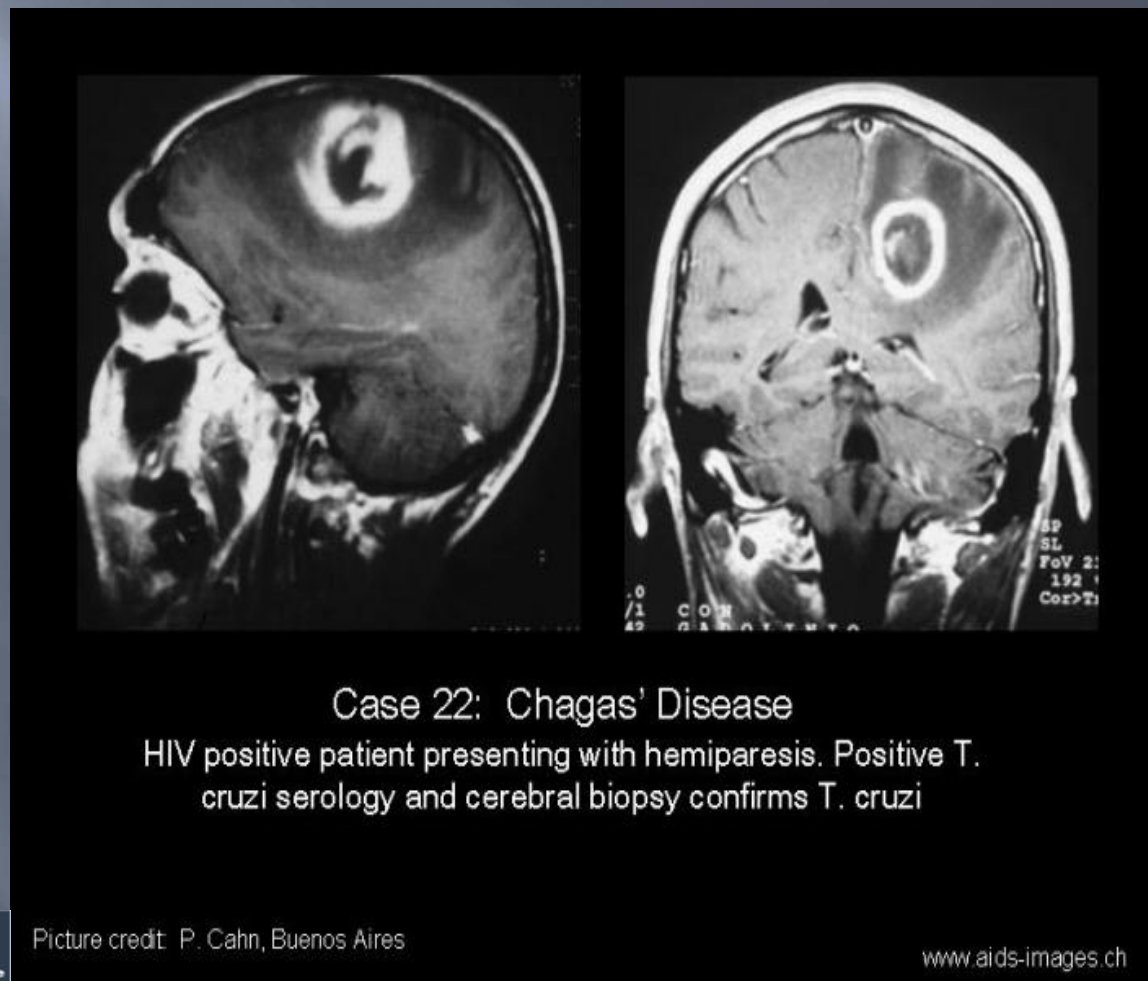
El mayor riesgo se produce cuando la cifra de linfocitos **T CD4 se encuentra por debajo de 200 cel/µl**.

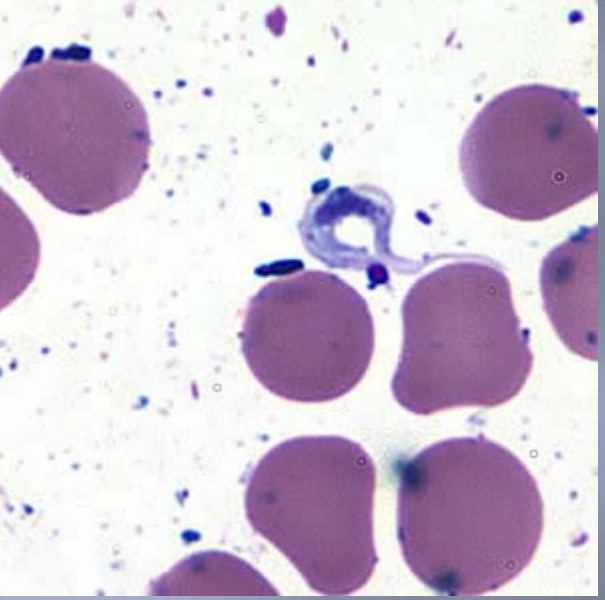
Estas reactivaciones se suelen acompañar de **elevadas parasitemias** y se caracterizan por el compromiso del sistema **nervioso central (SNC) y el miocardio.**

Corti M. AIDS and Chagas`disease. AIDS patient care and STDs 2000
[i]Sartori AMC, Neto JE, Nunes EV, et al. *Trypanosoma cruzi* parasitemia in chronic disease: comparision between human inmunodefiviency virus (HIV)-positive and HIV-negative patients. J infect dis 2002,
Cordova E, Boschi A, Ambrosioni J, Cudos C, Corti M. Reactivation of Chagas disease with central nervous system involvement in HIV-infected in Argentina 1992-2007. Int J Infect Dis 2008 Nov; 12(6):

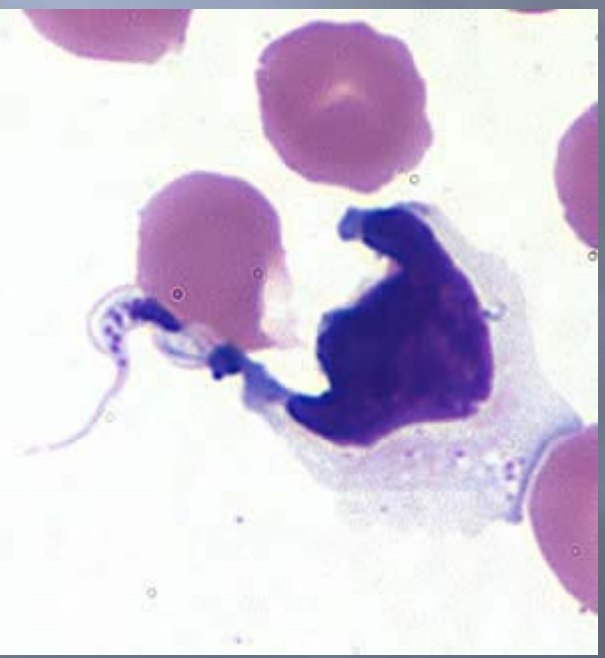
La afectación del SNC es la más frecuente y suele ser en forma de **lesiones ocupantes de espacio (chagomas**) o meningoencefalitis aguda.

Los chagomas suelen ser múltiples, con necrosis, edema perilesional, efecto masa y en las técnicas radiológicas captan contraste en anillo de forma similar a las lesiones de la toxoplasmosis, aunque la localización puede ser diferente afectando los chagomas a la sustancia blanca.





Trypomastigotes en LCR



La meningoencefalitis y la miocarditis son menos frecuente como forma de presentación. **La meningoencefalitis presenta características de meningitis parasitaria con abundantes tripomastigotes en el líquido cefalorraquideo.**

La mortalidad en estos casos puede llegar a ser muy elevada, según algunos estudios hasta del 79%.

Laguna Suárez . Beni. Bolivia.

Clinical Infectious Diseases

MAJOR ARTICLE

Transmission of *Trypanosoma cruzi* by Heart Transplantation

Heather Kun,[1] Anne Moore,[2] Laurene Mascola,[4] Frank Steurer,[2] Gena Lawrence,[2] Bernard Kubak,[5] Suman Radhakrishna,[6] David Leiby,[10] Ross Herron,[7] Tom Mone,[8] Robert Hunter,[9] Matthew Kuehnert,[3] and the Chagas Disease in Transplant Recipients Investigation Team[a]

[1]Epidemic Intelligence Service, Career Development Division, Office of Workforce and Career Development, [2]Division of Parasitic Diseases, and [3]Division of Healthcare Quality Promotion, Centers for Disease Control and Prevention, Atlanta, Georgia; [4]Los Angeles Department of Health Services, Acute Communicable Disease Control Program, [5]Department of Medicine, University of California, [6]Department of Medicine, University of Southern California, [7]American Red Cross, Southern California Region, [8]OneLegacy, and [9]California Department of Health, Laboratory Field Services, Biologics, Los Angeles, California; and [10]American Red Cross Holland Laboratory, Rockville, Maryland

Background. *Trypanosoma cruzi* infection (i.e., Chagas disease) is an unusual complication that can occur after solid-organ transplantation and that can result in severe illness or death. In 2006, there were 2 heart transplant recipients in Los Angeles, California, reported to have acute trypanosomiasis during the same month. We conducted an investigation to determine the source of these infections.

1 June 2009 Volume 48, Number 11

En los casos de **transplante cardiaco** en el paciente con cardiomiopatía chagásica la tasa de **reactivación** de la enfermedad se estima entre un **20-30%** aunque hay autores que describen cifras más altas

Suele aparecer con mayor frecuencia en el **primer año tras el transplante**

La **afectación cardiaca** con aparición de miocarditis es la forma clínica más frecuente

El uso de profilaxis de forma previa al transplante estaría indicada en estos pacientes para intentar minimizar el riesgo de reactivación.

En el hipotético caso de que un receptor recibiera un órgano de un donante infectado se debería realizar un seguimiento exhaustivo con técnicas de PCR para detectar de forma precoz la parasitemia que se produciría en caso de transmisión (infección aguda en inmunodepresión, no reactivación) y administrar tratamiento

Benvenuti L, RoggerioA, Sambiase N, Fiorelli A, Higuchi M. Polymerase chain reaction in endomyocardial biopsies for monitoring reactivation of Chagas`disease in heart transplantation: a case reort and review of literature. Cardivasc. Pathol 2005

TRATAMIE

El desconocimiento actual de la eficacia real del tratamiento es uno de los factores que aportan mayor confusión en la decisión final de tratar o no tratar

En la **fase aguda** de la enfermedad las tasas de curación se producen en **cifras cercanas al 100%**

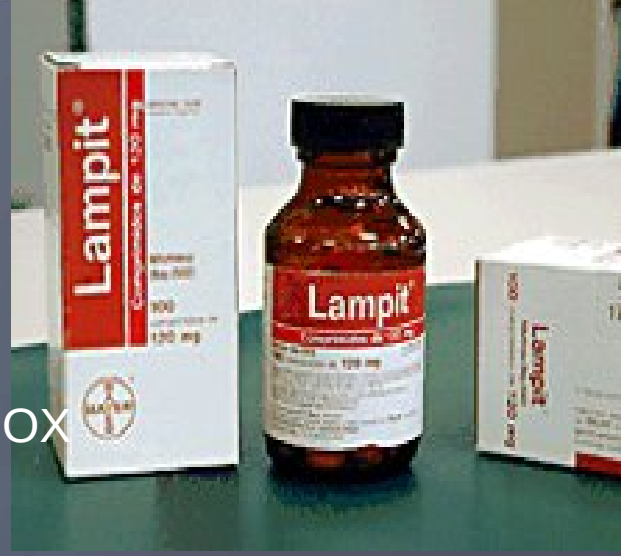
En **la fase crónica**, aunque se han descrito casos de negativización serológica las tasas de respuestas son muy variables, de un **8 a un 60%** según diferentes estudios, aunque siempre más bajas que en la fase aguda

Esta variabilidad puede estar en relación con la sensibilidad de diferentes cepas de *trypansoma*, reinfecciones o falta de consenso en los marcadores de curación.

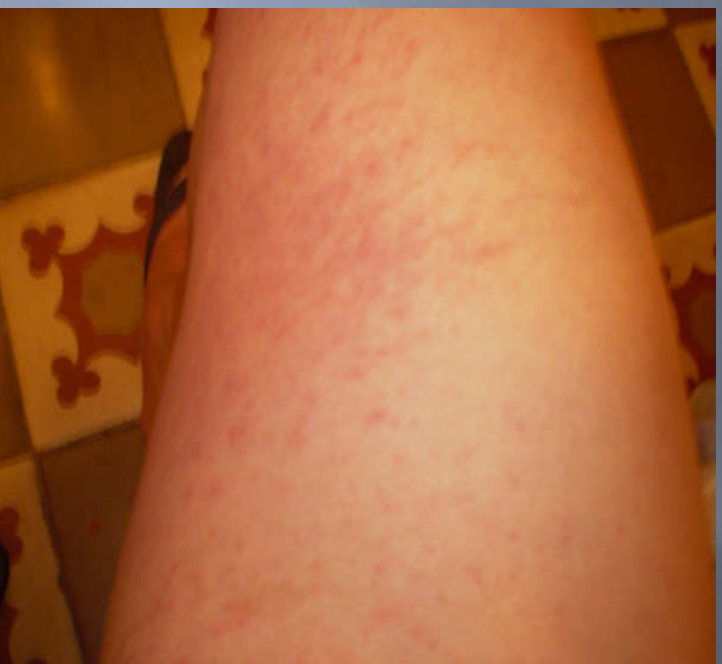
En publicaciones recientes si parece que la utilización de tratamiento en la fase crónica o indeterminada reduce el riesgo de progresión de la enfermedad sin efectos adversos graves frecuentes

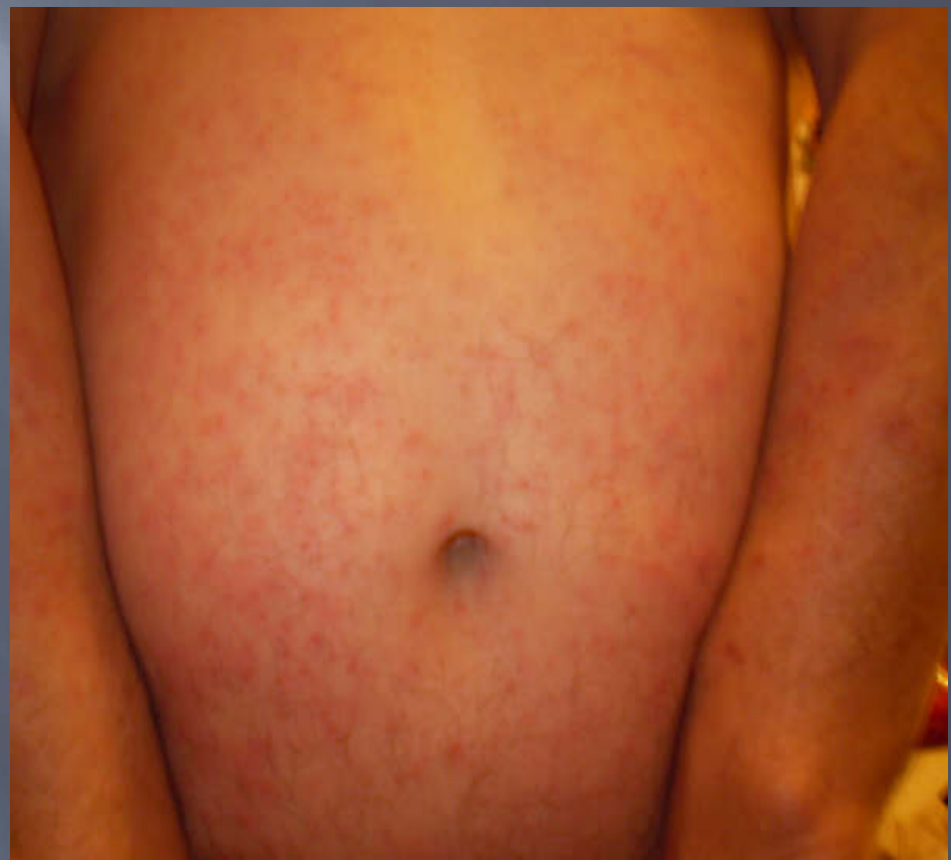
Gascón et al. Diagnóstico y tratamiento de la Enfermedd de Chagas importada. Med Clin (Barc) 2005;125(6)
Cancado JR. Long term evaluation of etiological treatment of Chagas disease with benznidazol. Rev Inst med Trop Sao Paulo.2002
Rodolfo Viotti, MD; Carlos Vigliano, MD; Bruno Lococo, MD; Graciela Bertocchi, MD; Marcos Petti, MD; MarGabriela Alvarez, MD;Miriam Postan, MD, PhD; and Alejandro Armenti, MD. Long-Term Cardiac Outcomes of Treating Chronic Chagas Disease with Benznidazole versus No Treatment. Ann Intern Med 2006

BENZNIDAZOL



NIFURTIMOX





ALGORITMO TERAPEUTICO DE LOS PACIENTES CON ENFERMEDAD DE CHAGAS

AGUDO

(cualquier mecanismo de transmisión)

CONGENITO

REACTIVACION

INMUNOSUPRESION

PREVISION DE TRATAMIENTO INUNOSUPRESOR

NIÑOS

ADULTOS JOVENES

(INFECCION RECIENTE)

ADULTOS EN FASE CRONICA FORMA INDETERMINADA

ADULTOS EN FASE CRONICA FORMA DETERMINADA

(LESIONES INICIALES PREFERENTEMENTE)

-

ADULTOS EN FASE CRONICA

LESIONES AVANZADAS

INESTABILIDAD CLÍNICA

CONTRAINDICACION DE TRATAMIENTO

TRATAMIENTO
NO TRATAMIENTO

OFRECER TRATAMIENTO

Marcadores de curación ????

Disminución progresiva de AC (años)

PCR- ????

Use of benznidazole to treat chronic Chagas' disease: a systematic review with a meta-analysis

José A. Pérez-Molina[1]*, Ana Pérez-Ayala[1], Santiago Moreno[2], M. Carmen Fernández-González[2], Javier Zamora[3] and Rogelio López-Velez[1]

Journal of Antimicrobial Chemotherapy Advance Access published October 9, 2009

Reference	Study design	Age, years	Follow-up	Clinical form	Sample size	Groups	Primary endpoint (secondary endpoints)	Adverse effects
Viotti *et al.*, 2006[42]	prospective cohort study	30–50 (mean 39)	mean 9.8 years	chronic phase (indeterminate and symptomatic)	566	283: BNZ 5 mg/kg/day×30 days	development of heart disease 12/283; mortality 3/283 (negative serology values 32/218)	37 patients discontinued treatment due to adverse effects [allergic dermatitis (33) and GI intolerance (4)]; a further 55 patients had adverse effects that did not lead to discontinuation (dermatitis, headache, fever, GI intolerance and pruritus)
						283: not treated	development of heart disease 40/283; mortality 12/283 (negative serology values 12/212)	not reported
de Castro *et al.*, 2006[37]	observational prospective study	23–88 (mean 49)	24 months	chronic phase	40	27: BNZ 5 mg/kg/day×60 days	negative blood culture values 24/27	three patients discontinued BNZ due to adverse reactions
						13: not treated	negative blood culture values 6/13	
Fabbro *et al.*, 2007[38]	prospective–retrospective observational study	17–46	BNZ mean 20.6 years; not treated mean 21.7 years	chronic phase (indeterminate and symptomatic)	84	27: BNZ 5 mg/kg/day×45–60 days	negative serology values 9/27 (negative XD 27/27) (clinical progression 2/27)[a]	adverse effects in 9/33 patients (6 discontinuations): maculopapular erythema, oedema, nausea, headache, pruritus and liver profile abnormalities
						57: not treated	negative serology values 0/57 (negative XD 1/57) (clinical progression 9/57)	

Annals of Internal Medicine | ARTICLE

Long-Term Cardiac Outcomes of Treating Chronic Chagas Disease with Benznidazole versus No Treatment

A Nonrandomized Trial

Rodolfo Viotti, MD; Carlos Vigliano, MD; Bruno Lococo, MD; Graciela Bertocchi, MD; Marcos Petti, MD; María Gabriela Alvarez, MD; Miriam Postan, MD, PhD; and Alejandro Armenti, MD

Background: Benznidazole is effective for treating acute-stage Chagas disease, but its effectiveness for treating indeterminate and chronic stages remains uncertain.

Objective: To compare long-term outcomes of patients with nonacute Chagas disease treated with benznidazole versus outcomes of those who did not receive treatment.

Design: Clinical trial with unblinded, nonrandom assignment of patients to intervention or control groups.

Setting: Chagas disease center in Buenos Aires, Argentina.

Patients: 566 patients 30 to 50 years of age with 3 positive results on serologic tests and without heart failure.

Measurements: The primary outcome was disease progression, defined as a change to a more advanced Kuschnir group or death. Secondary outcomes included new abnormalities on electrocardiography and serologic reactivity.

Intervention: Oral benznidazole, 5 mg/kg of body weight per day for 30 days (283 patients), or no treatment (283 patients).

Results: Fewer treated patients had progression of disease (12 of 283 [4%] vs. 40 of 283 [14%]; adjusted hazard ratio, 0.24 [95% CI, 0.10 to 0.59]; $P = 0.002$) or developed abnormalities on electrocardiography (15 of 283 [5%] vs. 45 of 283 [16%]; adjusted hazard ratio, 0.27 [CI, 0.13 to 0.57]; $P = 0.001$) compared with untreated patients. Left ventricular ejection fraction (hazard ratio, 0.97 [CI, 0.94 to 0.99]; $P < 0.002$) and left ventricular diastolic diameter (hazard ratio, 2.45 [CI, 1.53 to 3.95]; $P < 0.001$) were also associated with disease progression. Conversion to negative results on serologic testing was more frequent in treated patients than in untreated patients (32 of 218 [15%] vs. 12 of 212 [6%]; adjusted hazard ratio, 2.1 [CI, 1.06 to 4.06]; $P = 0.034$).

Limitations: Nonrandom, unblinded treatment assignment was used, and follow-up data were missing for 20% of patients. Loss to follow-up was more common among patients who were less sick. Two uncontrolled interim analyses were conducted.

Conclusions: Compared with no treatment, benznidazole treatment was associated with reduced progression of Chagas disease and increased negative seroconversion for patients presenting with nonacute disease and no heart failure. These observations indicate that a randomized, controlled trial should now be conducted.

Ann Intern Med. 2006;144:724-734. www.annals.org

For author affiliations, see end of text.

The BENEFIT trial: testing the hypothesis that trypanocidal therapy is beneficial for patients with chronic Chagas heart disease

J Antonio Marin-Neto[1]/+, Anis Rassi Jr[2], Alvaro Avezum Jr[3], Antonio C Mattos[3], Anis Rassi[2]

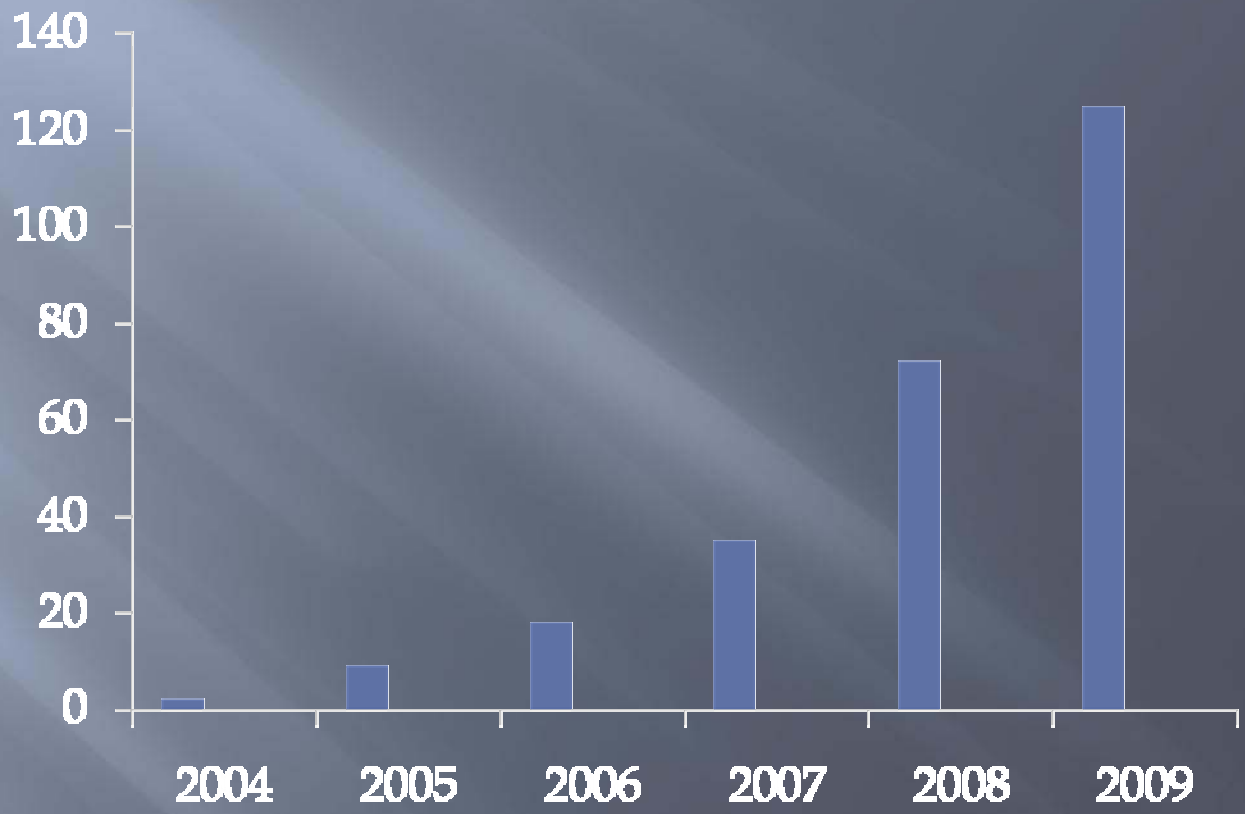
Mem Inst Oswaldo Cruz, Rio de Janeiro, Vol. *104*(Suppl. I): 319-324, 2009

Benznidazole Evaluation For Interrupting Trypanosomiasis (BENEFIT)Trial

ed double-blind placebo-controlled trial of benznidazole in patients with chronic Chagas' he

Lago Titicaca, Bolivia

PACIENTES CON ENFERMEDAD DE CHAGAS ATENDIDOS EN L



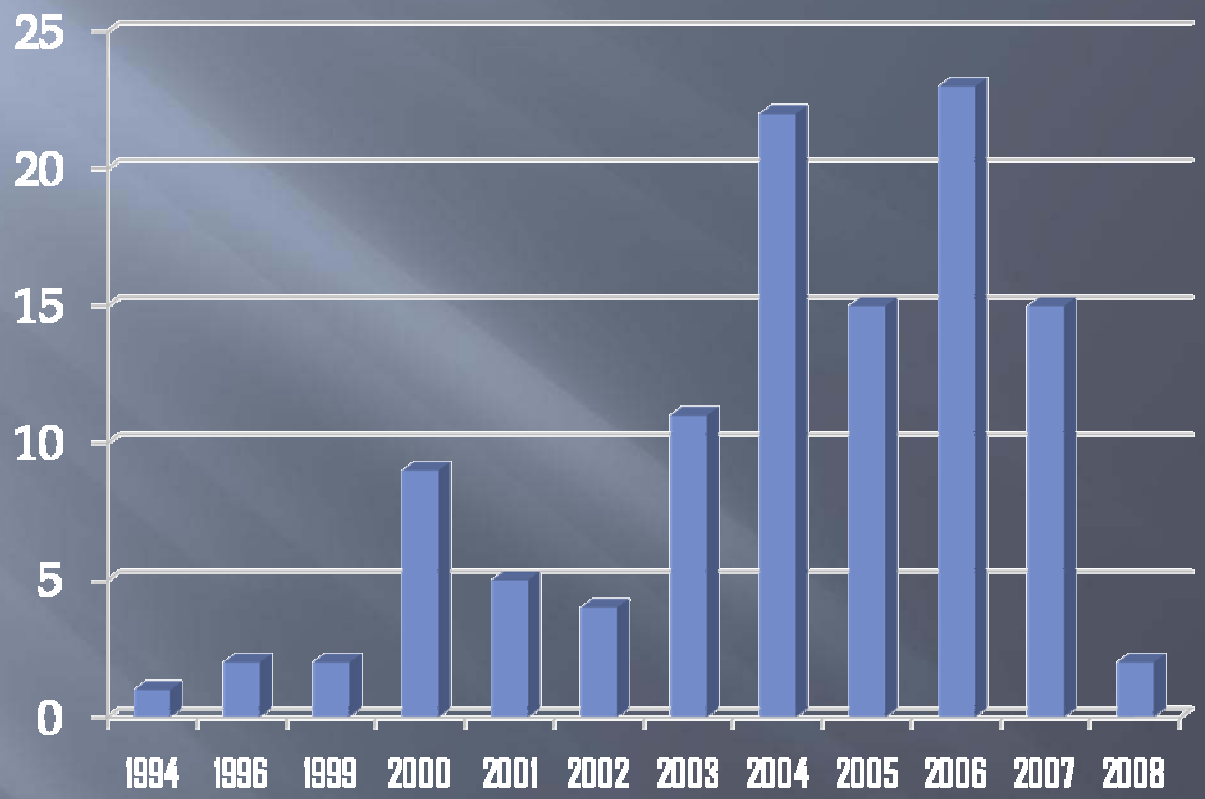
111 pacientes

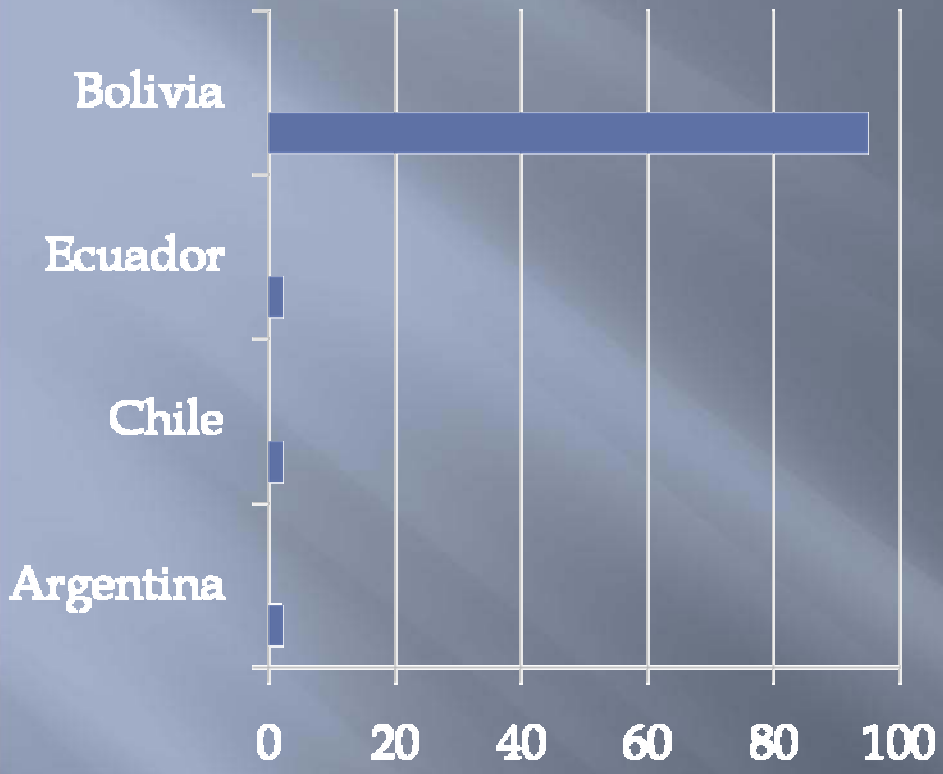
Edad media 38.21 años

66.6% mujeres

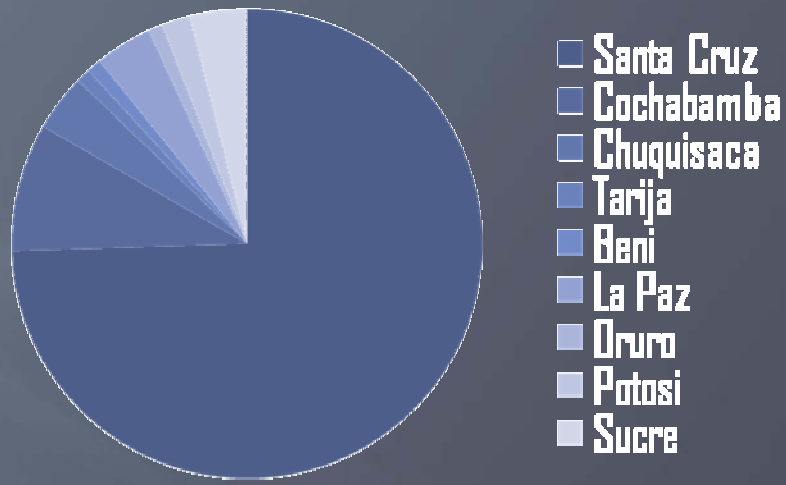
33.3% hombres

Año de llegada a España





Países de procedencia



Departamentos en Bolivia

Procedencia:

-Centro de transfusiones
-Centros de atención primaria
-Cribado en gestantes
-Diagnóstico previo
-Petición propia

Asintomáticos 78 (70.2%)

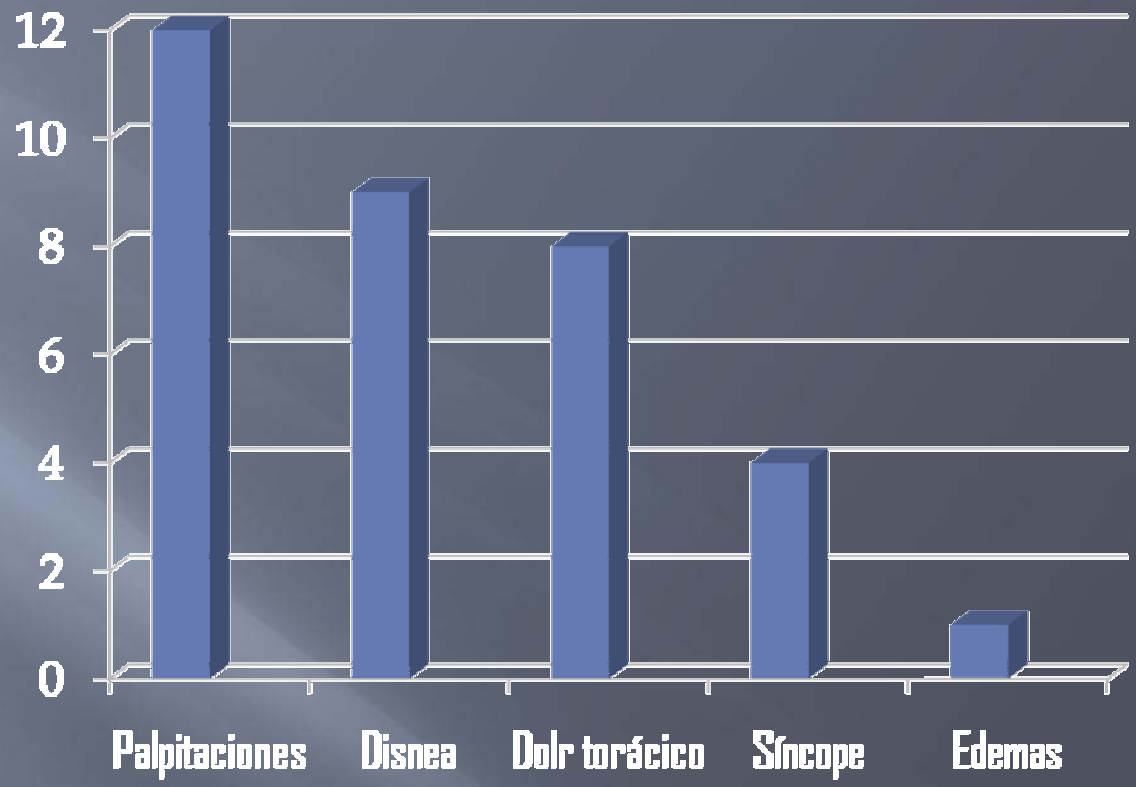
Sintomáticos 33 (29.7%)

Síntomas/signos cardiacos 18 (54.5%)

Síntomas digestivos 13 (39.3%):

-Estreñimiento 100%
-Disfagia 38.4%

Todas las personas con dolicosigma referían estreñimiento



-Palpitaciones
-Disnea
-Dolor torácico
-Pre-Síncope
-Edemas

>1 síntoma (38.8%)

Palpitaciones-disnea

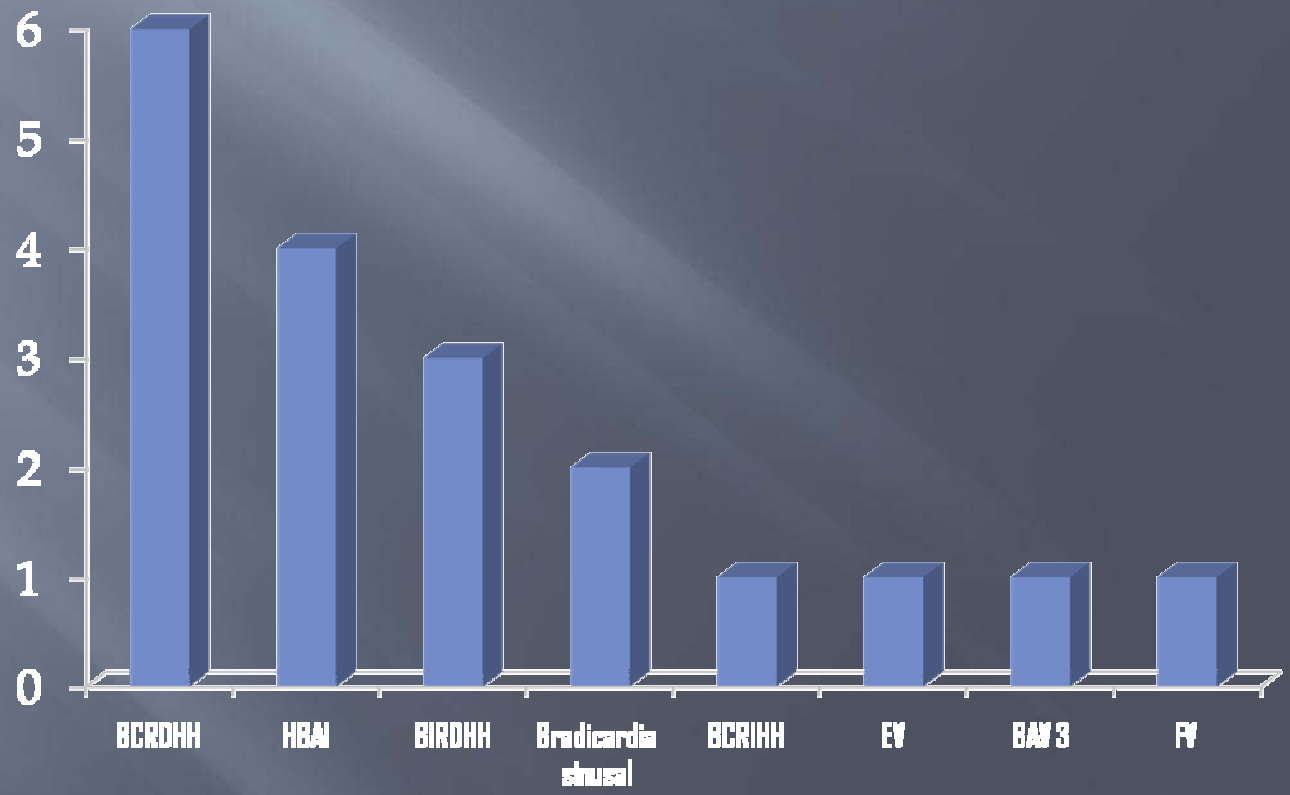
RX DE TORAX (90)

Normales 86 (96%)

Cardiomegalia
Tracto fibroso LSD
Enfisema

3 marcapasos

ECG (84) alterados16 (19.7%)



ENEMA OPACO (48)

Dolicosigma 5 (10.41%)
Malrotación de colon
Diverticulosis

TRÁNSITO ESOFAGOGÁSTRICO (59)

Esofagopatía grado I 4 (6.7%)

MANOMETRÍAS (2)

Normales

TRATAMIENTO

54 pacientes tratados

Benznidazol 5 mg/kg/día durante 60 días (inicio con dosis crecientes)

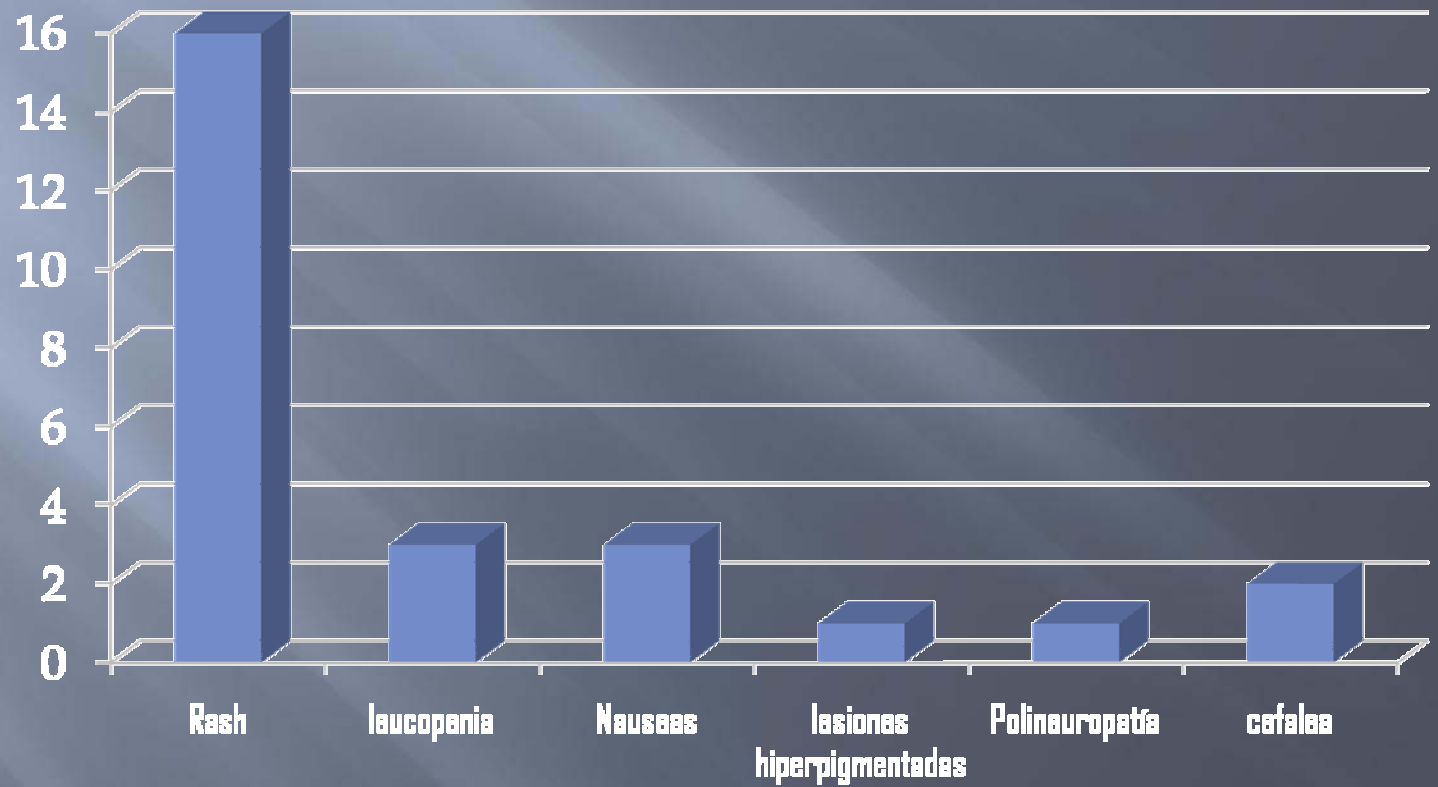
Inmunosupresión

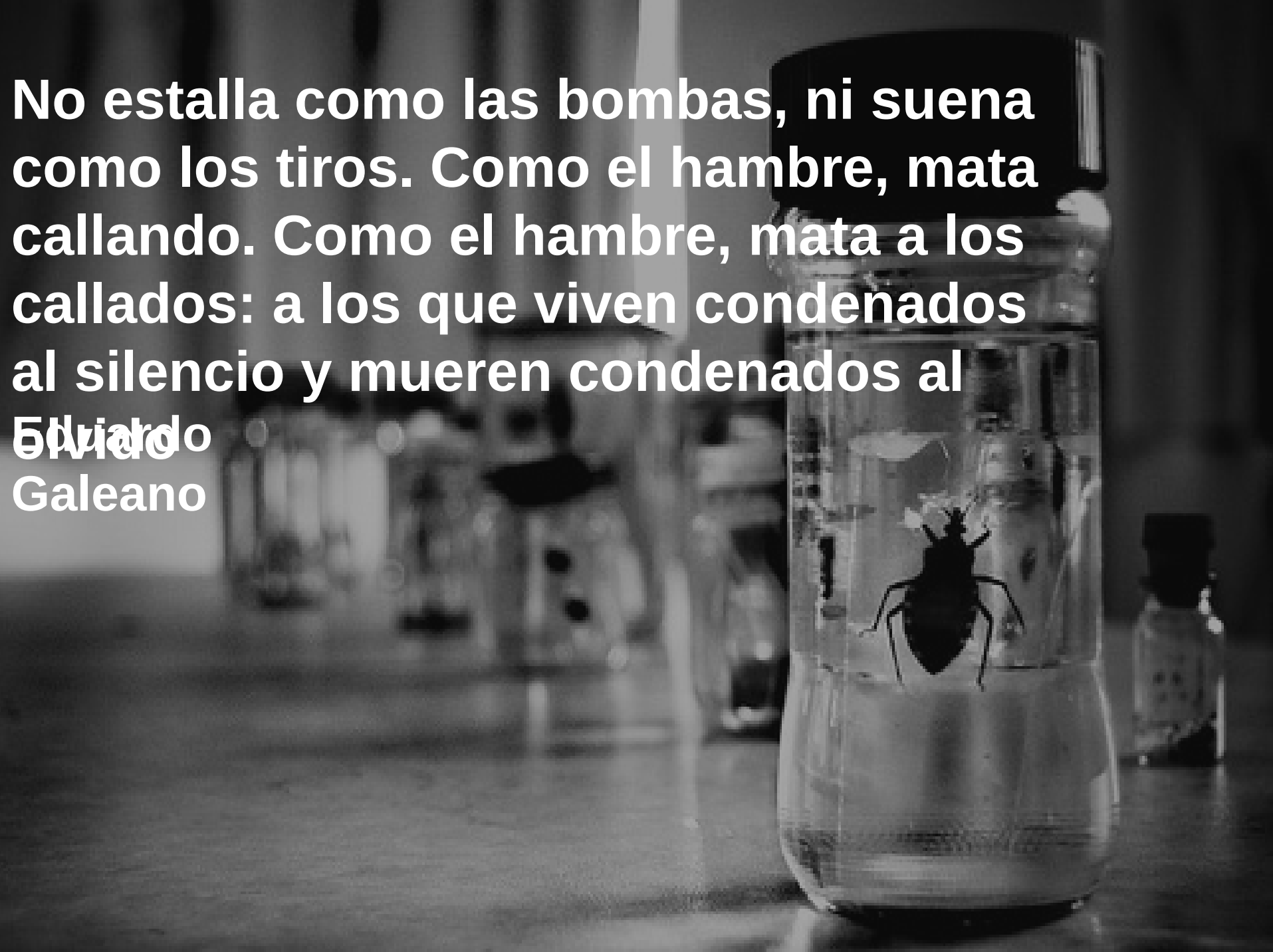
2 pacientes con VIH
1 patología respiratoria (corticoides)
1 patología autoinmune

PCR postratamiento –

1 Negativizacción de tasa de Ac

Efectos secundarios más frecuentes





No estalla como las bombas, ni suena como los tiros. Como el hambre, mata callando. Como el hambre, mata a los callados: a los que viven condenados al silencio y mueren condenados al olvido

Eduardo Galeano