



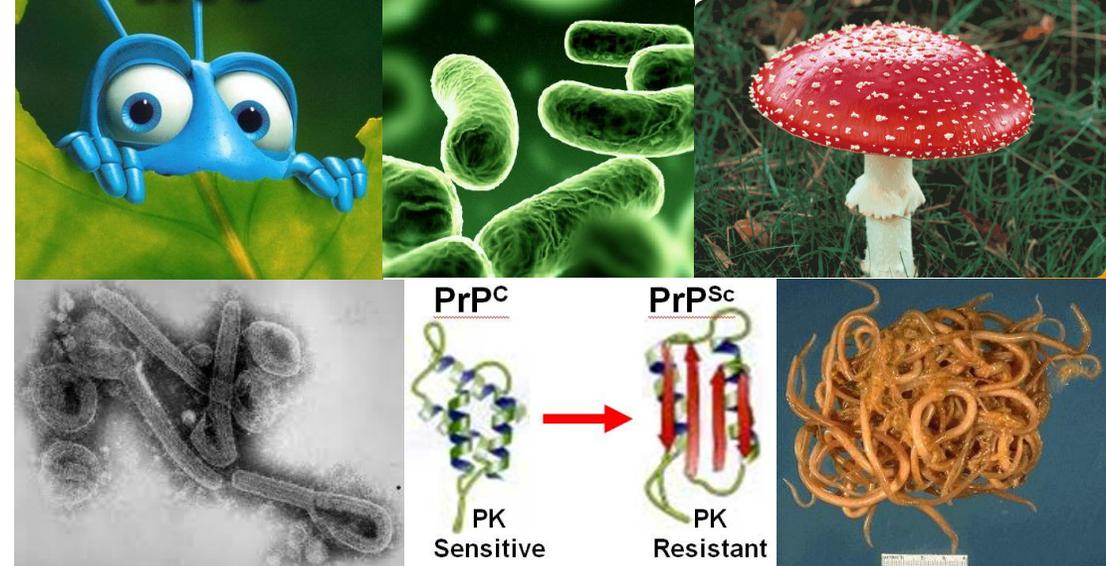
Facultad de Ciencias de la Salud
Programa de Medicina

Factores de Riesgo en Enfermedades Infecciosas

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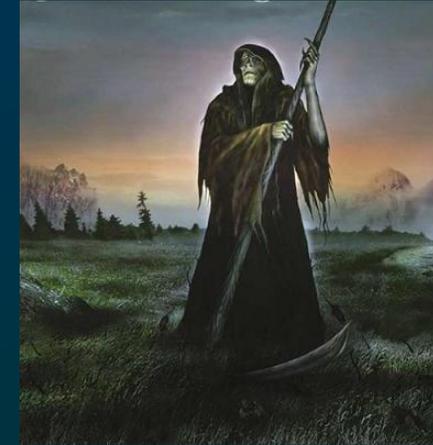
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Miembro del American College of Epidemiology (ACE).
Miembro de la Asociación Internacional de Epidemiología (IEA).*



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Historia de la Epidemiología

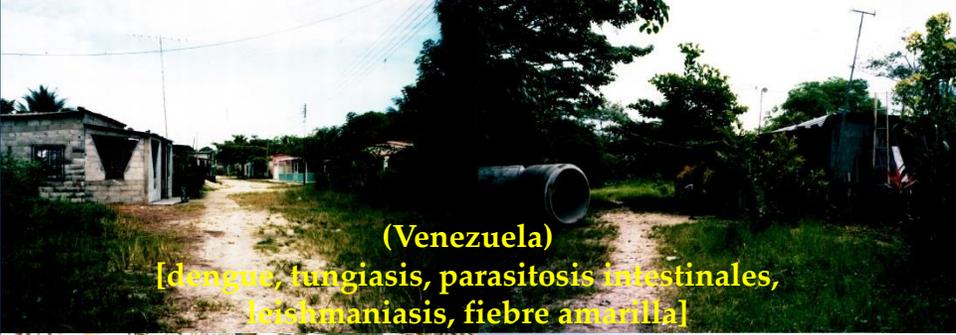
- Griegos y Romanos “*Pestis*”
- Quarante Giorni, “*Isolamento*”
(Ragusa, Venecia, 1374)
- Legado importante, pero que termina siendo el origen del vínculo “casi” indisoluble entre la epidemiología y las enfermedades infecciosas.



Contribuciones de la Epidemiología

- Escorbuto (James Lind) – ensayo de intervención, deficiencia nutricional
- Cáncer escrotal (Percival Pott) – salud ocupacional, carcinógenos
- Sarampión (Peter Panum) – período de incubación, período infeccioso
- Cólera (John Snow) – transmisión hídrica, experimento natural
- Fiebre Puerperal (Ignatius Semmelweis) – prevención por higiene
- Rubéola y defectos congénitos del nacimiento (Gregg) – exposición prenatal
- Cáncer de pulmón y el hábito de fumar – maduración de la epidemiología de las enfermedades crónicas
- Flúor y caries dentales – epidemiología comunitaria, prevención ambiental
- Ensayo de inmunización contra la poliomielitis - un gran experimento que demostró la efectividad de la vacuna contra este virus que era muy temido
- Enfermedad cardiovascular – estudios comunitarios longitudinales, ensayos de intervención comunitarios





(Venezuela)
[dengue, tungiasis, parasitosis intestinales,
leishmaniasis, fiebre amarilla]



(Venezuela)
[Chagas, parasitosis intestinales]



(Venezuela)
[dengue]



(Brasil)
[enfermedades diarreicas]



(Perú)
[parasitosis intestinales, leptospirosis, fiebre tifoidea,
gastroenteritis bacterianas]



(Ecuador)
[leishmaniasis, Chagas]



(Venezuela)
[malaria]



(Bolivia)
[histoplasmosis]



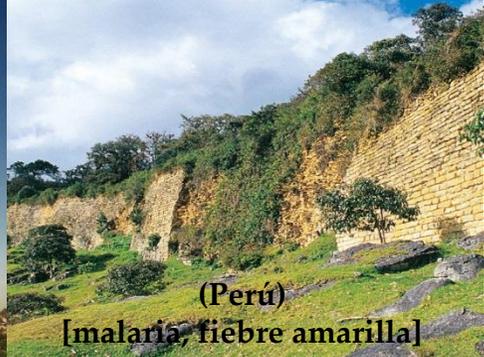
(Colombia)
[fiebre amarilla]



(Venezuela)
[esquistosomiasis]



(Paraguay-Brasil)
[enfermedades importadas]



(Perú)
[malaria, fiebre amarilla]

Factores de Riesgo para Enfermedades Infecciosas

- Supongamos que deseamos ver la asociación entre la presencia de piso de tierra en las casas de niños familias de Caimalito y la ocurrencia de ascariasis
 - Se encontró que:
 - De 107 niños con ascariasis: **89 vivían en casas con piso de tierra** y 18 no.
 - De 113 niños sin ascariasis: 15 vivían en casas con piso de tierra y **98 no**.
- ¿Cuál es la razón de chances (OR) de encontrar un niño con ascariasis que viva en una casa con piso de tierra en comparación con aquellos que viven en casas con otros materiales de piso?



Análisis ep

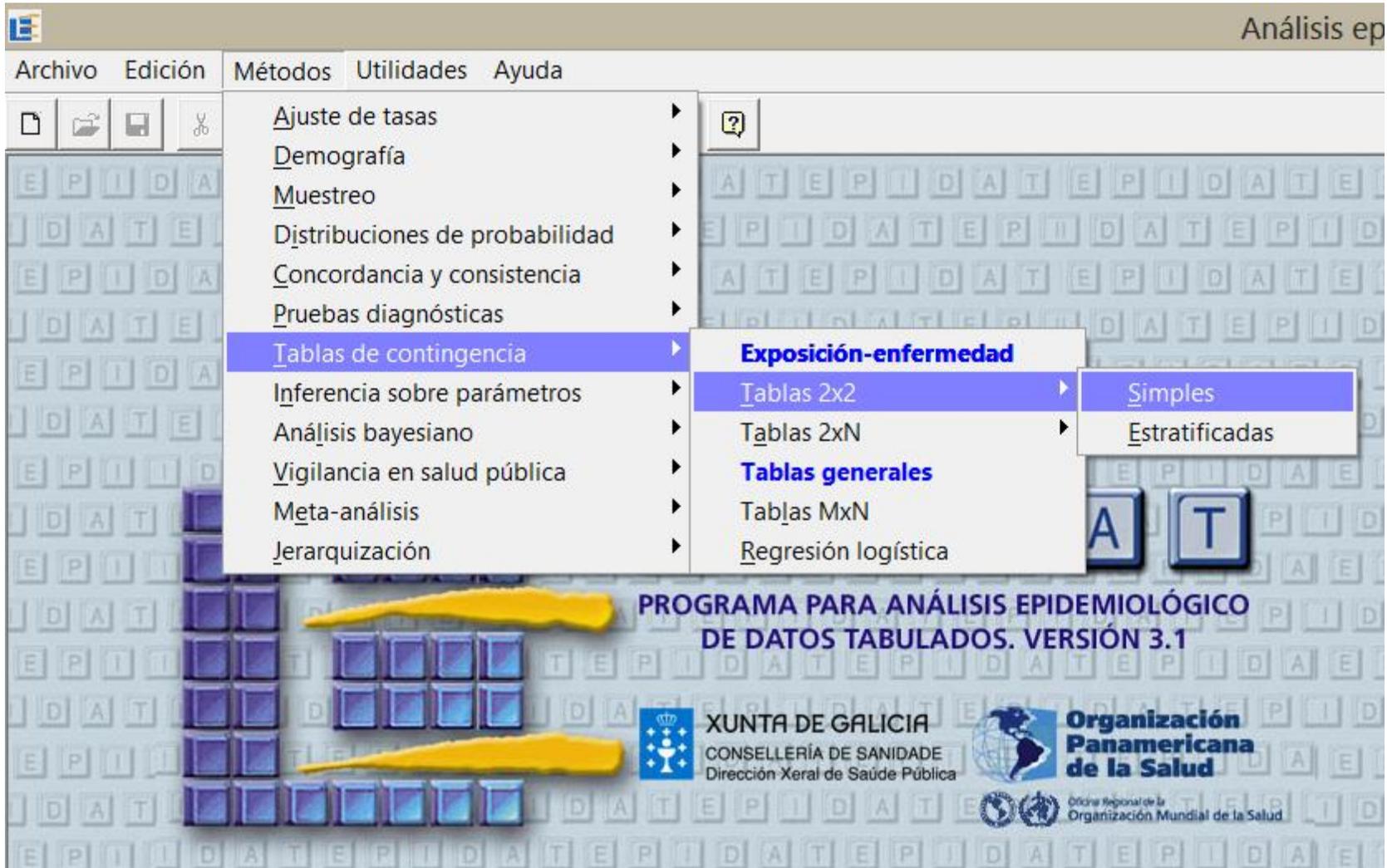
Archivo Edición Métodos Utilidades Ayuda

- Ajuste de tasas
- Demografía
- Muestreo
- Distribuciones de probabilidad
- Concordancia y consistencia
- Pruebas diagnósticas
- Tablas de contingencia**
 - Exposición-enfermedad**
 - Tablas 2x2**
 - Simple
 - Estratificadas
 - Tablas 2xN
 - Tablas generales**
 - Tablas MxN
 - Regresión logística
- Inferencia sobre parámetros
- Análisis bayesiano
- Vigilancia en salud pública
- Meta-análisis
- Jerarquización

PROGRAMA PARA ANÁLISIS EPIDEMIOLÓGICO DE DATOS TABULADOS. VERSIÓN 3.1

 **XUNTA DE GALICIA**
CONSELLERÍA DE SANIDADE
Dirección Xeral de Saúde Pública

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



Tablas de contingencia: Tablas 2x2 simples

Origen de datos | Resultados

Tipo de estudio

- Transversal
- Cohortes
- Caso-control
- Caso-control emparejado

Nivel de confianza (%)

95,0

Sumar 0,5 a todas las frecuencias si hay ceros

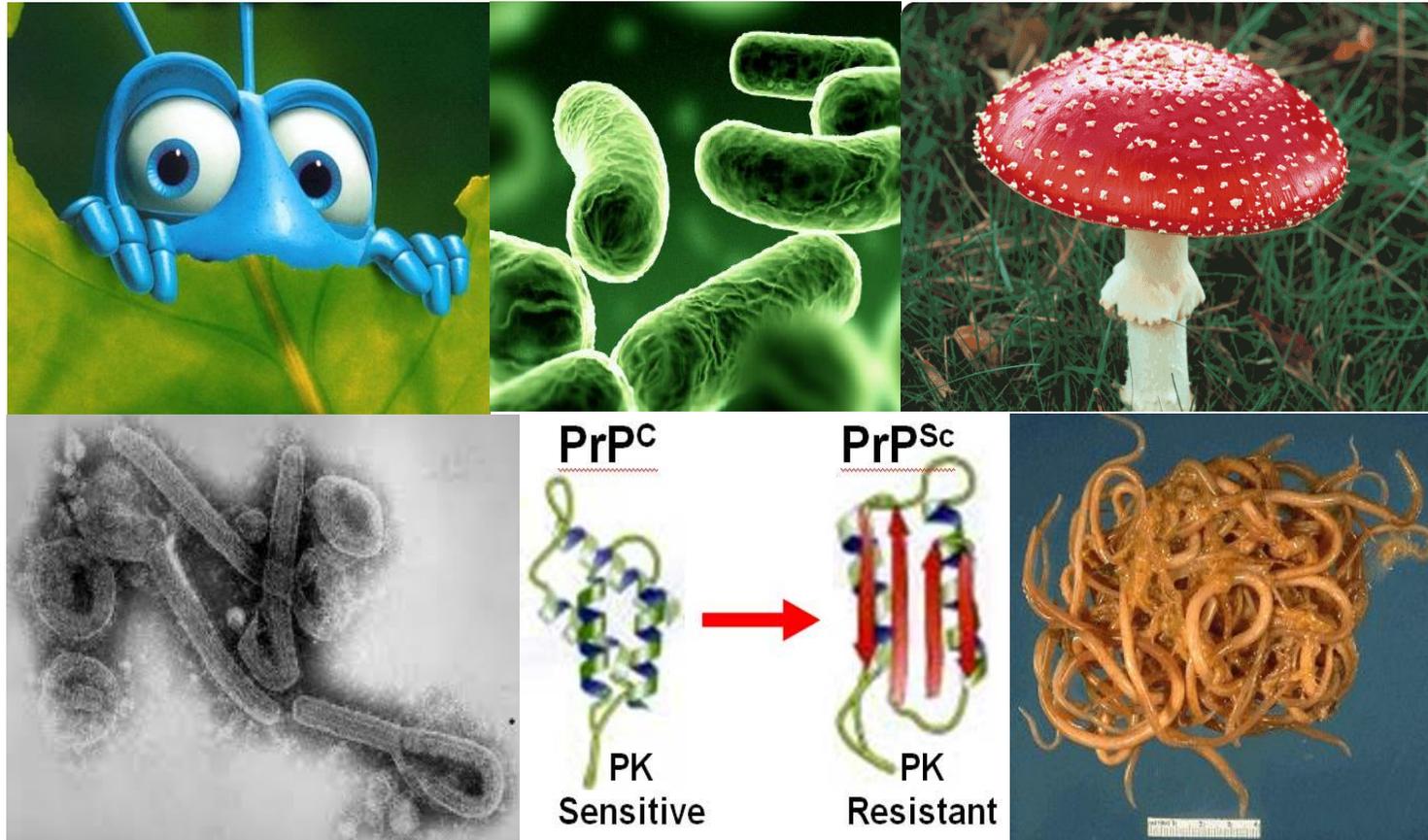
		Enfermedad		Total
		Enfermos	Sanos	
Factor de riesgo	Expuestos	89	15	104
	No expuestos	18	98	116
Total		107	113	220

Prevalencia de exposición		Estimación	
En enfermos			0,831776
En no enfermos			0,132743
	OR	IC (95,0%)	
	32,303704	15,368202 - 67,901846	(Woolf)

107 sujetos con ascariasis, 89 tuvieron piso de tierra
 $89/89+18=0,8318$ (**83,18%**)
 113 sujetos sin ascariasis, 15 tuvieron piso de tierra
 $15/15+98=0,1327$ (**13,27%**)

OR = 32,3 (IC95% 15,37-67,9)

¿Como se clasifican los agentes infecciosos?



¿Como se clasifican los agentes infecciosos?

- Bacterias
- Hongos
- Virus
- Priones
- Parásitos
 - *Helmintos*
 - *Protozoarios*



Factores de Riesgo para Parasitosis



Table 4

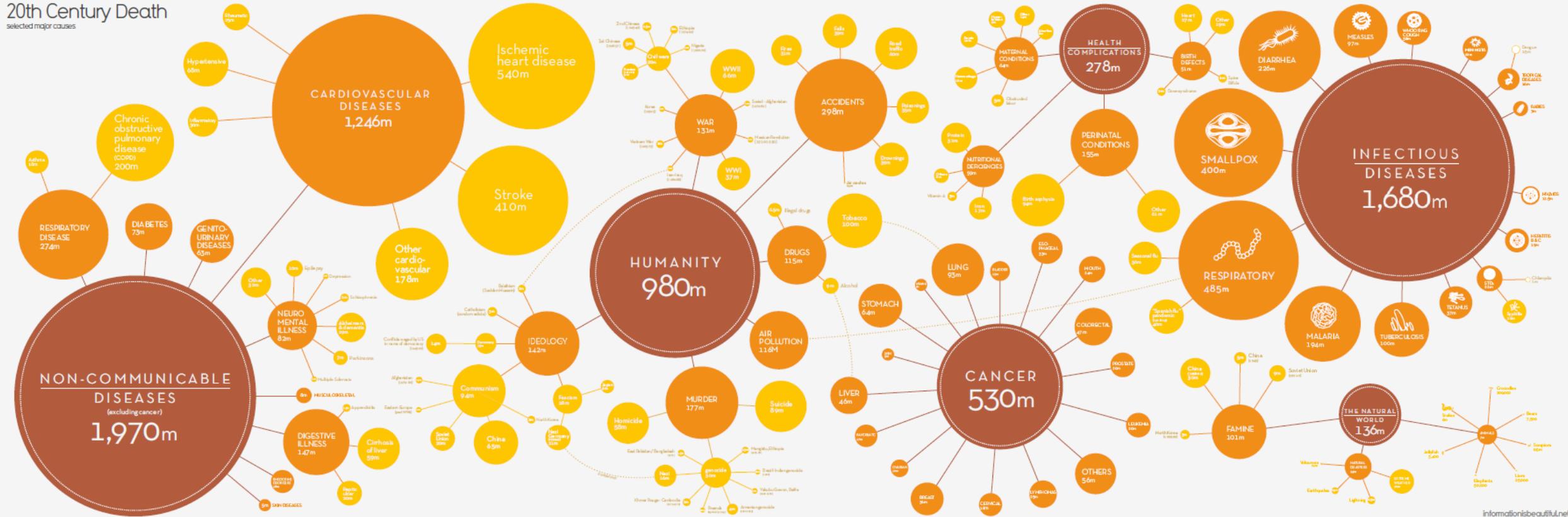
Univariate and multivariate analysis of household risk factors for ascariasis and trichuriasis in individuals from North Central Venezuela (May 2007 to December 2008)

Variable	Ascariasis		Trichuriasis	
	Crude OR (univariate) (95% CI)	Adjusted OR (multivariate) (95% CI)	Crude OR (univariate) (95% CI)	Adjusted OR (multivariate) (95% CI)
Vulnerable house				
Yes	4.242 (4.198–4.287)	1.479 (1.428–1.532)	2.598 (2.547–2.650)	10.519 (9.971–11.097)
No	1.000	1.000	1.000	1.000
In a rural area				
Yes	5.597 (5.543–5.652)	2.067 (2.035–2.101)	2.610 (2.564–2.657)	1.918 (1.868–1.970)
No	1.000	1.000	1.000	1.000
Near to small rivers or wetlands				
Yes	4.928 (4.838–5.020)	NS	NS	NS
No	1.000			
Rudimentary wall materials				
Yes	4.097 (4.055–4.139)	NS	1.598 (1.564–1.634)	NS
No	1.000		1.000	
Soil floor				
Yes	13.283 (13.127–13.440)	5.027 (4.895–5.162)	3.726 (3.630–3.825)	5.190 (4.944–5.448)
No	1.000	1.000	1.000	1.000
Tap water access				
No	8.719 (8.626–8.809)	2.512 (2.465–2.560)	3.014 (2.950–3.080)	NS
Yes	1.000	1.000	1.000	
Collection of water in inappropriate receptacles				
Yes	1.734 (1.708–1.759)	NS	1.453 (1.417–1.490)	1.118 (1.089–1.149)
No	1.000		1.000	1.000
Appropriate disposal of sewage waters				
No	6.728 (6.597–6.862)	2.315 (2.254–2.378)	1.091 (1.023–1.163)	NS
Yes	1.000	1.000	1.000	
Appropriate waste disposal				
No	3.061 (3.031–3.091)	1.798 (1.775–1.820)	1.700 (1.671–1.729)	NS
Yes	1.000	1.000	1.000	

NS: not significant.

¿De qué se murió la gente en el siglo XX?

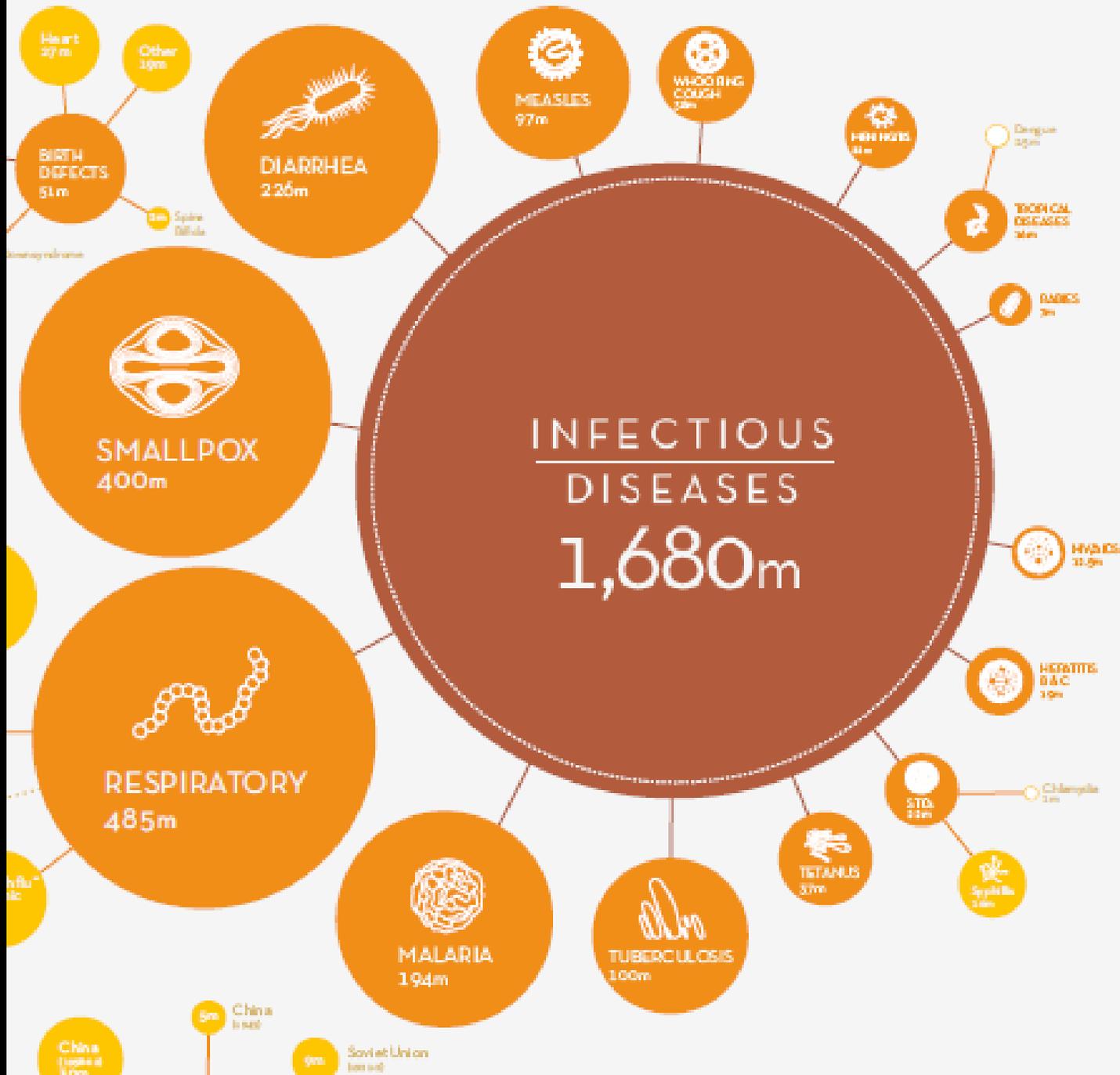
20th Century Death
selected major causes



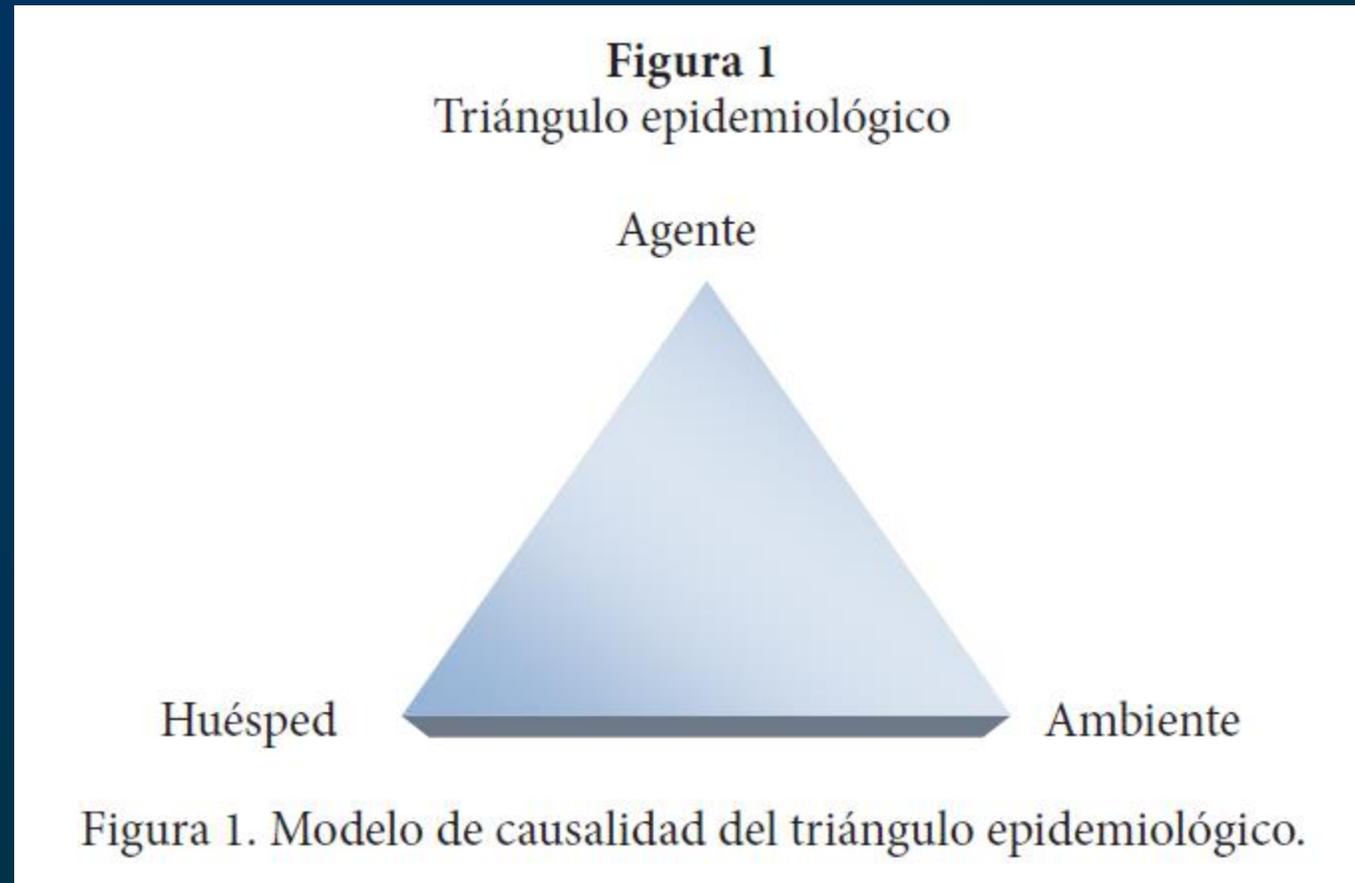
informationisbeautiful.net

<http://www.informationisbeautiful.net/visualizations/20th-century-death/>
WHO Mortality report (PDF) & app, WHO Global Burden of Disease (PDF), OECD Mortality Stats

¿De qué se murió la gente en el siglo XX?



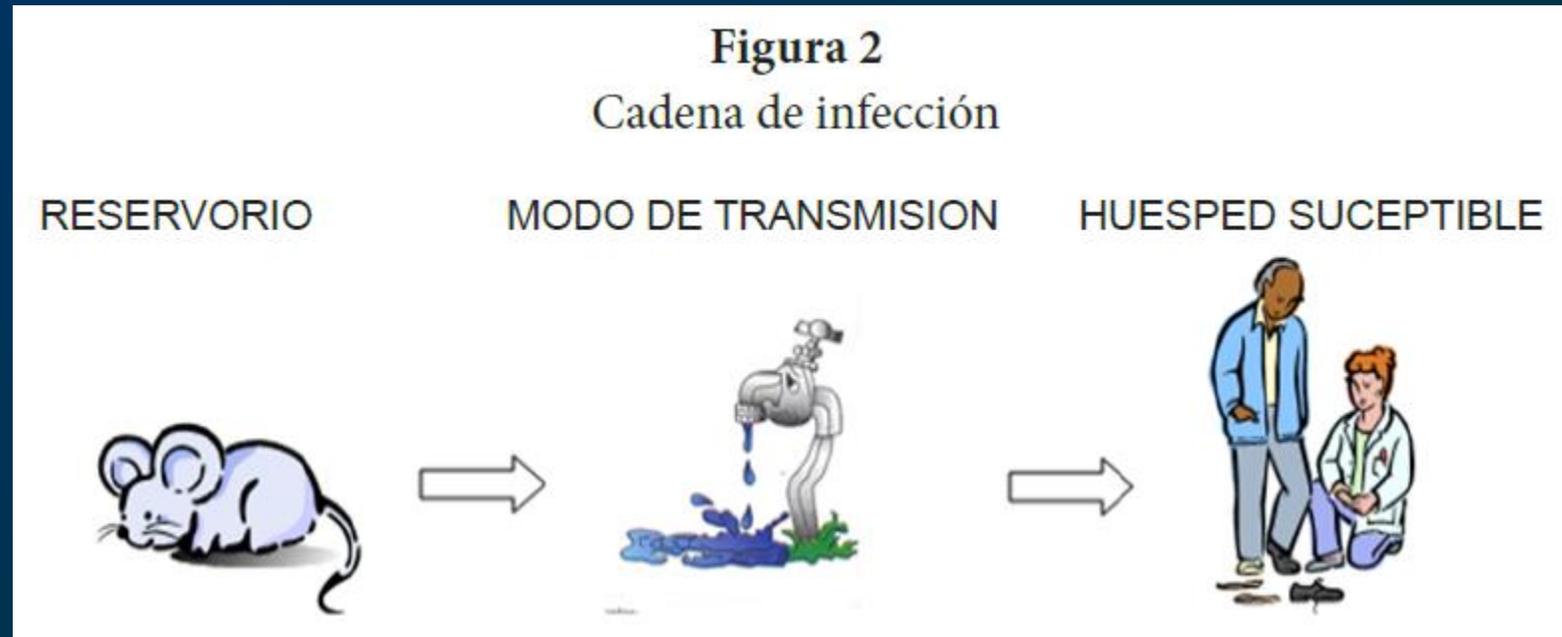
Epidemiología de las Enfermedades Transmisibles



Echezuria, Riskey, Fernández, Rodríguez-Morales.
Temas de Epidemiología y Salud Pública. Tomo I, 2013.

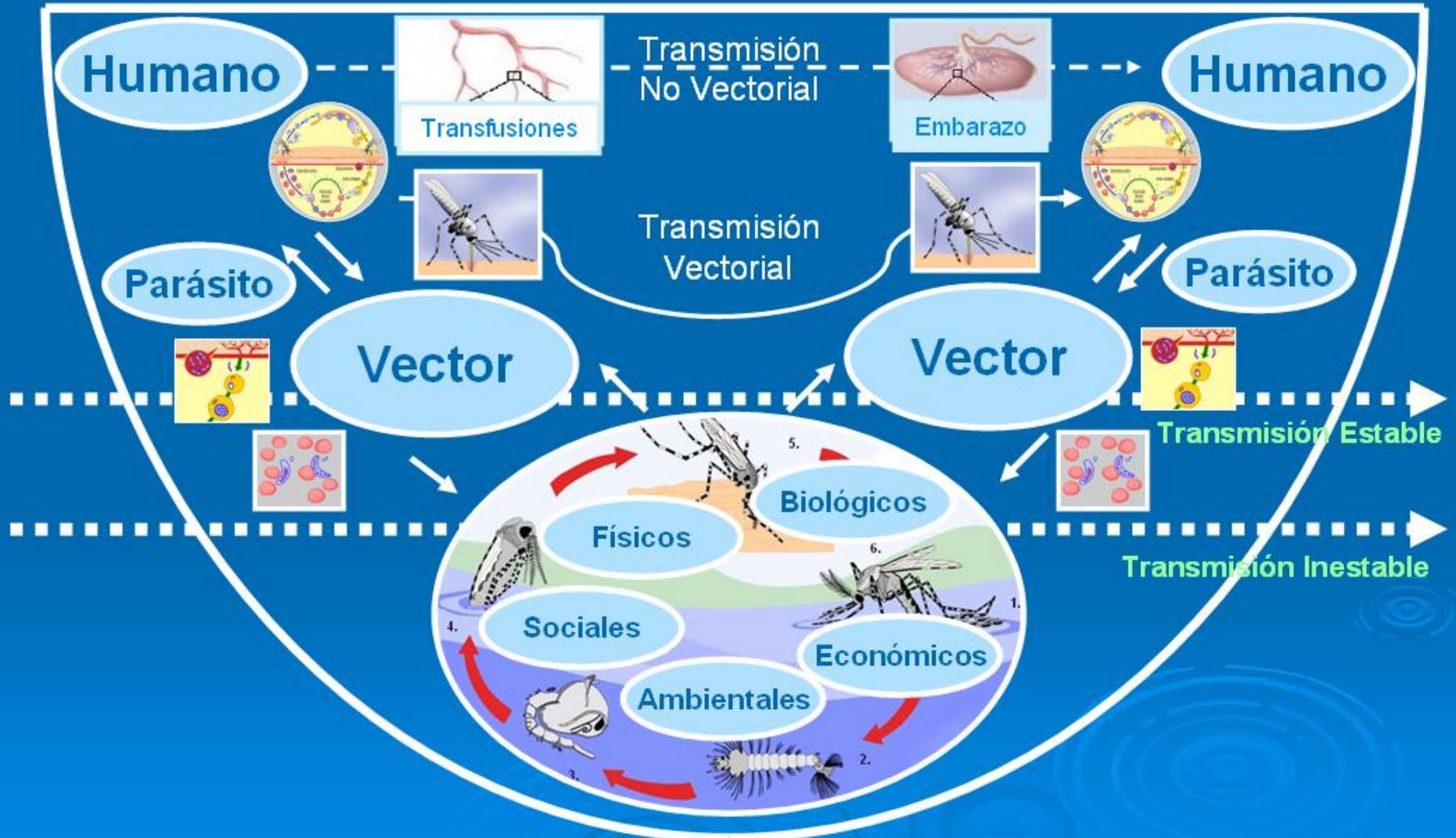


Epidemiología de las Enfermedades Transmisibles



Echezuria, Riskey, Fernández, Rodríguez-Morales.
Temas de Epidemiología y Salud Pública. Tomo I, 2013.

Ecoepidemiología de la Malaria



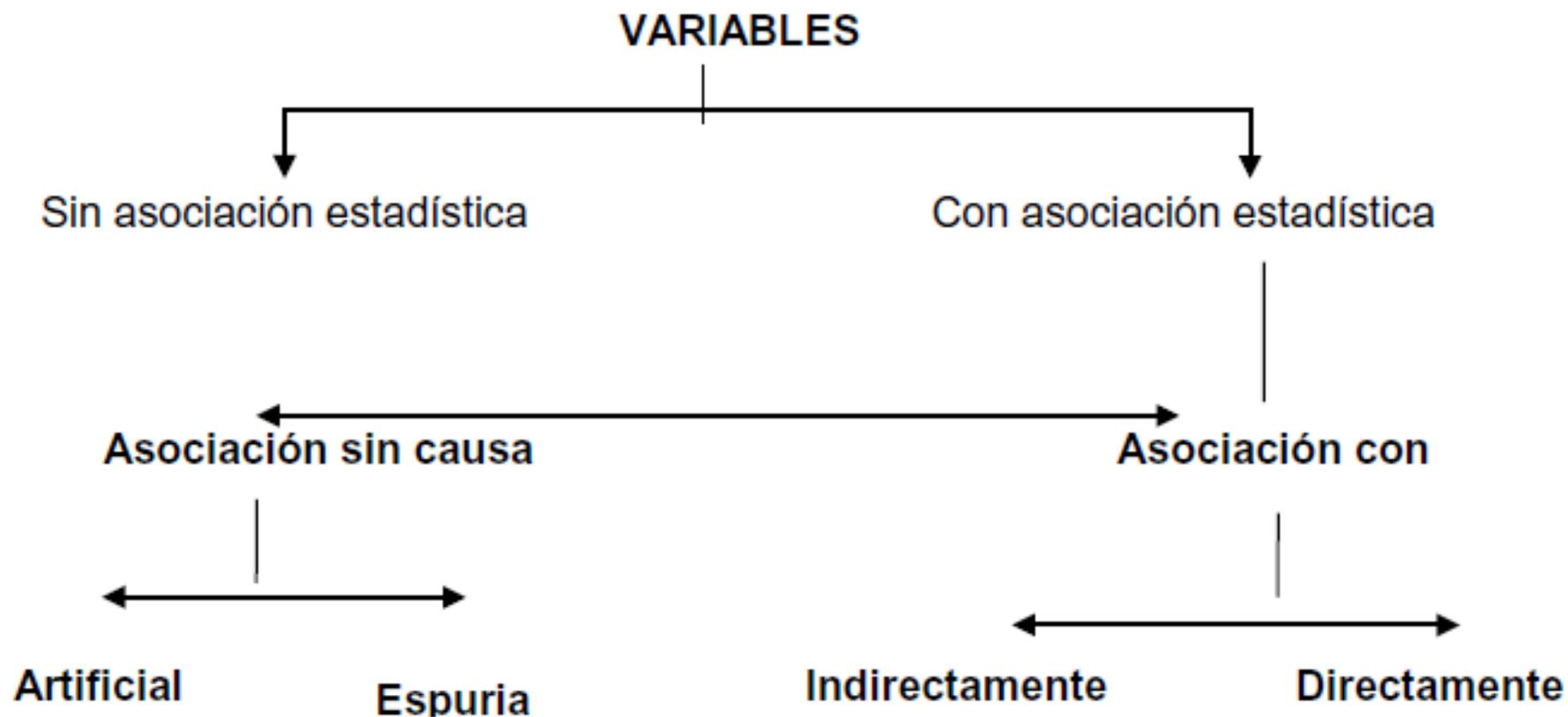
Causalidad y Factores de Riesgo

- El concepto clásico de causa viene así a ser substituido por el de ***factor de riesgo***,
 - el atributo o variable que consideramos que está relacionado al incremento de la probabilidad de que un individuo desarrolle la enfermedad.
- Una definición más completa de factor de riesgo nos dice que
 - “es la característica o circunstancia detectable en una persona o en un grupo de ellas, asociada con un aumento en la probabilidad de padecer, desarrollar o estar especialmente expuesta a un daño de salud”.

Echezuria, Riskey, Fernández, Rodríguez-Morales.
Temas de Epidemiología y Salud Pública. Tomo I, 2013.



Figura 4
Tipos de asociación entre variables



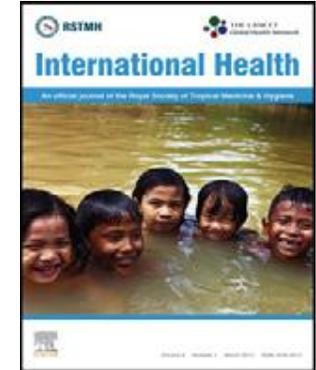
Estudios con análisis multivariado

Table 4

Univariate and multivariate analysis of household risk factors for ascariasis and trichuriasis in individuals from North Central Venezuela (May 2007 to December 2008)

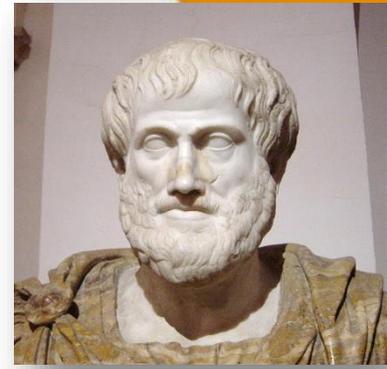
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NS: not significant.



Quintero K, Durán C, Duri D, Medina F, Garcia J, Hidalgo G, Nakal S, Echeverria-Ortega M, Albano C, Nino Incani R, Cortez J, Jiménez S, Díaz M, Maldonado C, Matute F, Rodríguez-Morales AJ. Household social determinants of ascariasis and trichuriasis in North Central Venezuela. *International Health* 2012 Jun; 4(2): 103-110

EVOLUCIÓN DEL PENSAMIENTO CAUSAL



En la Grecia antigua, Aristóteles preconizaba la importancia de conocer las causas de los fenómenos. Pero el “estagirita” (así también se le conoce a Aristóteles, por haber nacido en la ciudad de Estagira) reconocía que una sola causa no bastaba para explicar la producción de un efecto entendiendo que para ello se requería la presencia de cuatro factores, a saber. (5)

- 1- La causa **material** que constituiría el receptáculo pasivo sobre el que actuarían las demás causas.
- 2- La causa **formal** que vendría siendo la esencia o ideal de lo tratado.
- 3- La causa **eficiente** sería la fuerza motriz o compulsión externa a la cual obedecen los cuerpos y, por último.
- 4- La causa **final**, a la cual todo tiende o sirve.

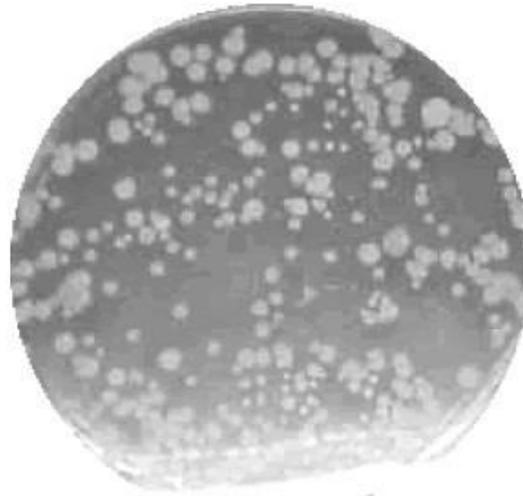
R Koch and Causality

The microorganism must be found in all cases of the disease

It must be isolated from the host and grown in pure culture

It must reproduce the original disease when injected into a susceptible host

It must be found in the experimental host so infected.



Postulados de Koch

1. El agente patógeno debe estar presente en cada caso de la enfermedad en las condiciones apropiadas y ausente en las personas sanas.
2. El agente no debe aparecer en otra enfermedad de manera fortuita o saprófita.
3. El agente debe ser aislado del cuerpo en un cultivo puro a partir de las lesiones de la enfermedad.
4. El agente debe provocar la enfermedad en un animal susceptible al ser inoculado.
5. El agente debe ser aislado de nuevo de las lesiones producidas en los animales de experimentación.

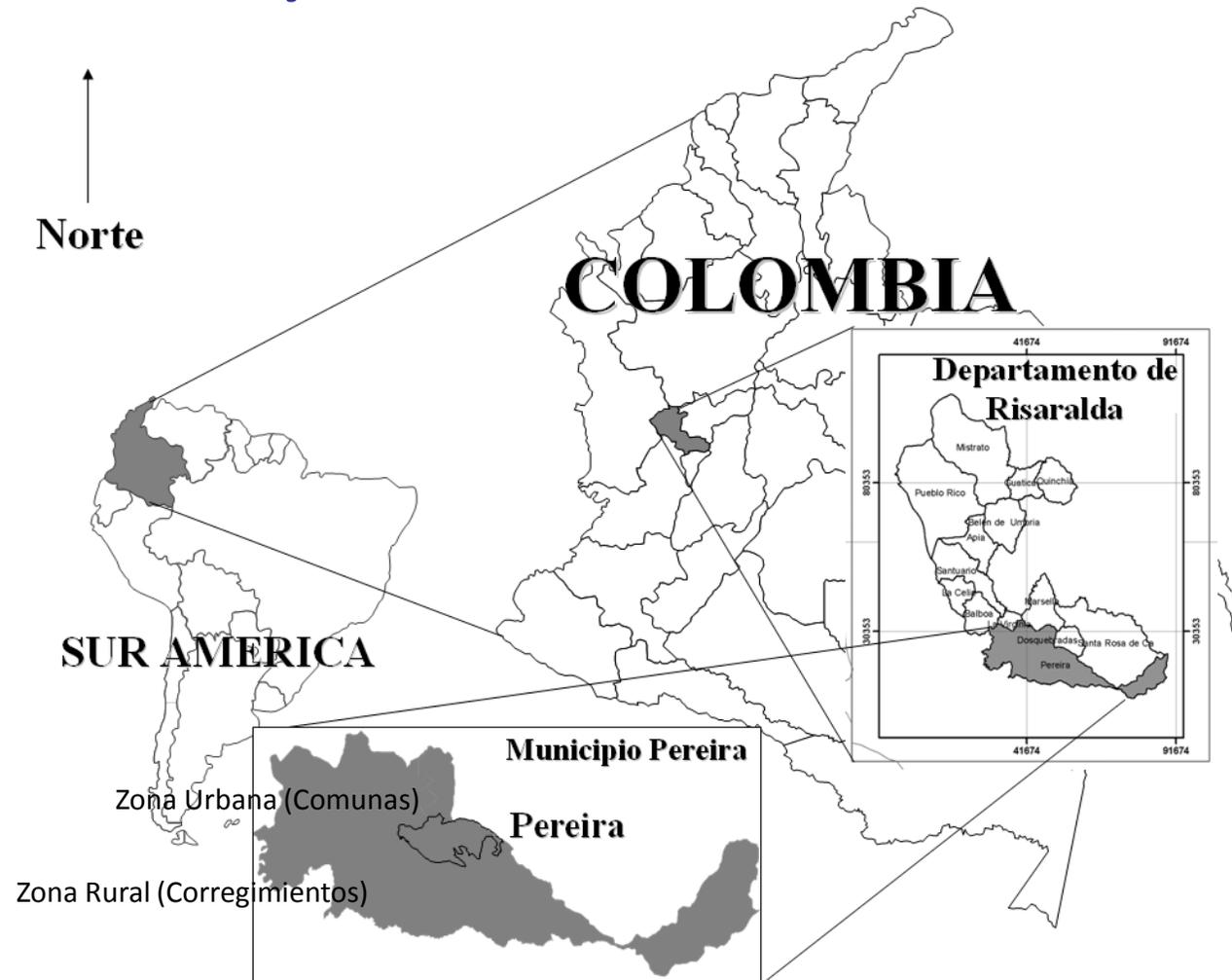
¿Qué es una causa?

“Un evento o estado de la naturaleza que inicia o permite el comienzo, ya sea en forma solidaria o en conjunción con otras causas, de una secuencia de eventos que dan como resultado un efecto”.

Clasificación de Factores de Riesgo

1. Biológicos (sexo, ciertos grupos de edad, la herencia).
2. Ambientales (contaminación atmosférica, disposición de excretas inadecuada, carencia de agua potable).
3. De comportamiento (ingerir bebidas alcohólicas en exceso, fumar cigarrillos, dietas inadecuadas en cantidad y calidad).
4. Infraestructura sanitaria (relacionada con la atención a la salud, como baja calidad de la misma o ausencia de la misma, cobertura insuficiente).
5. Socioeconómicos (nivel educativo, ingreso, ocupación, vivienda, trabajo, recreación).

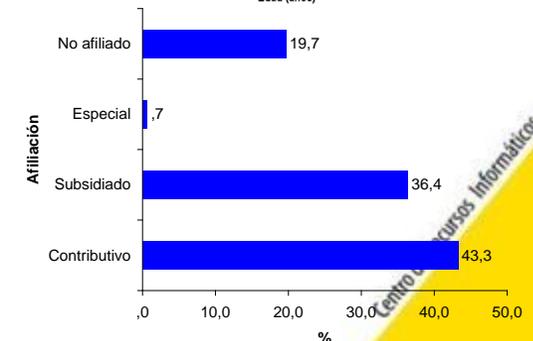
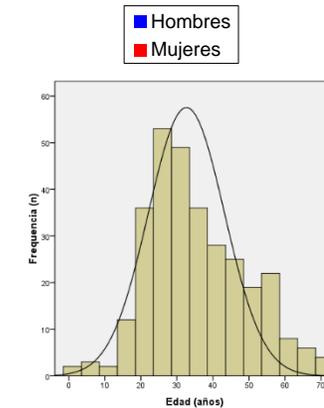
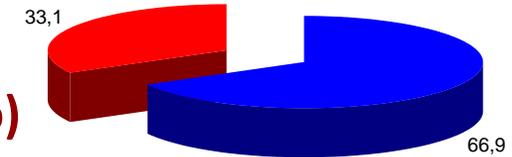
Materiales y métodos



Ubicación relativa del Municipio Pereira, Departamento de Risaralda, Colombia

Resultados

- Se evaluaron 305 casos de pacientes con VIH/SIDA:
 - 2010: 146 casos; incidencia de 31,94 casos/100.000 hab.
 - 2011: 159 casos; incidencia de 34,59 casos/100.000 hab.
- Distribución por género:
 - 66,9% hombres (♂) y 33,1% mujeres (♀) (sd por año)
- Edad:
 - Promedio: 35,5 años ($\pm 13,8$) (sd por año)
 - ♂: 37,25 años; ♀: 32,08 años ($p=0,002$)
 - 2010: ♂: 36,92 años; ♀: 30,50 años ($p=0,01$)
 - 2011: ♂: 37,59 años; ♀: 33,30 años ($p=0,06$)
- Procedencia:
 - 96,7% de zonas urbanas y 3,3% de zonas rurales
- Afiliación al SGSSS:
 - 80,3% afiliado a seguridad social, 19,7% no.

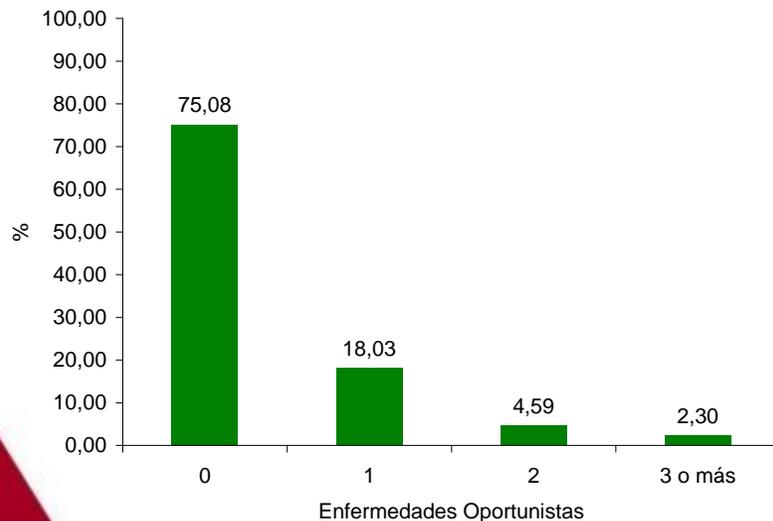




Resultados

- **Del total (n=305)**
 - **Ocurrencia de oportunistas:**
 - 24,9% (IC95% 19,9-29,9) presentaron ≥ 1 EO
 - 75,08% (IC95% 70,1-80,1) no
 - **Hospitalización:**
 - 37% (IC95% 31,5-42,6) se hospitalizaron

Número de Oportunistas Encontradas



Hospitalización según ocurrencia de EO

Ocurrencia de EO	Hospitalización		Total	
	Sí	No		
Sí	n	49	27	76
	%	64,5%	35,5%	100,0%
No	n	64	165	229
	%	27,9%	72,1%	100,0%
Total	n	113	192	305
	%	37,0%	63,0%	100,0%

$\chi^2=32,642$; $p<0,001$; OR=4,679 (IC95% 2,696-8,120)



Resultados

- **VARIABLES ASOCIADAS A LA OCURRENCIA DE EO**
 - **Ocurrencia de oportunistas fue mayor en sujetos ≥ 35 años**
 - 30,5% (IC95% 22,5-38,5) (OR=1,742; IC95% 1,032-2,941).
 - **Ocurrencia de oportunistas fue mayor en aquellos no afiliados al SGSSS**
 - 36,7% (IC95% 23,6-49,7) (OR=2,048; IC95% 1,117-3,753).

Ocurrencia de EO según grupos de Edad

Edad (años)	Ocurrencia de EO		Total
	Sí	No	
≥ 35	n	43	98
	%	30,5%	69,5%
<35	n	33	131
	%	20,1%	79,9%
Total	n	76	229
	%	24,9%	75,1%

$\chi^2=4,362$; $p=0,037$; OR=1,742 (IC95% 1,032-2,941)

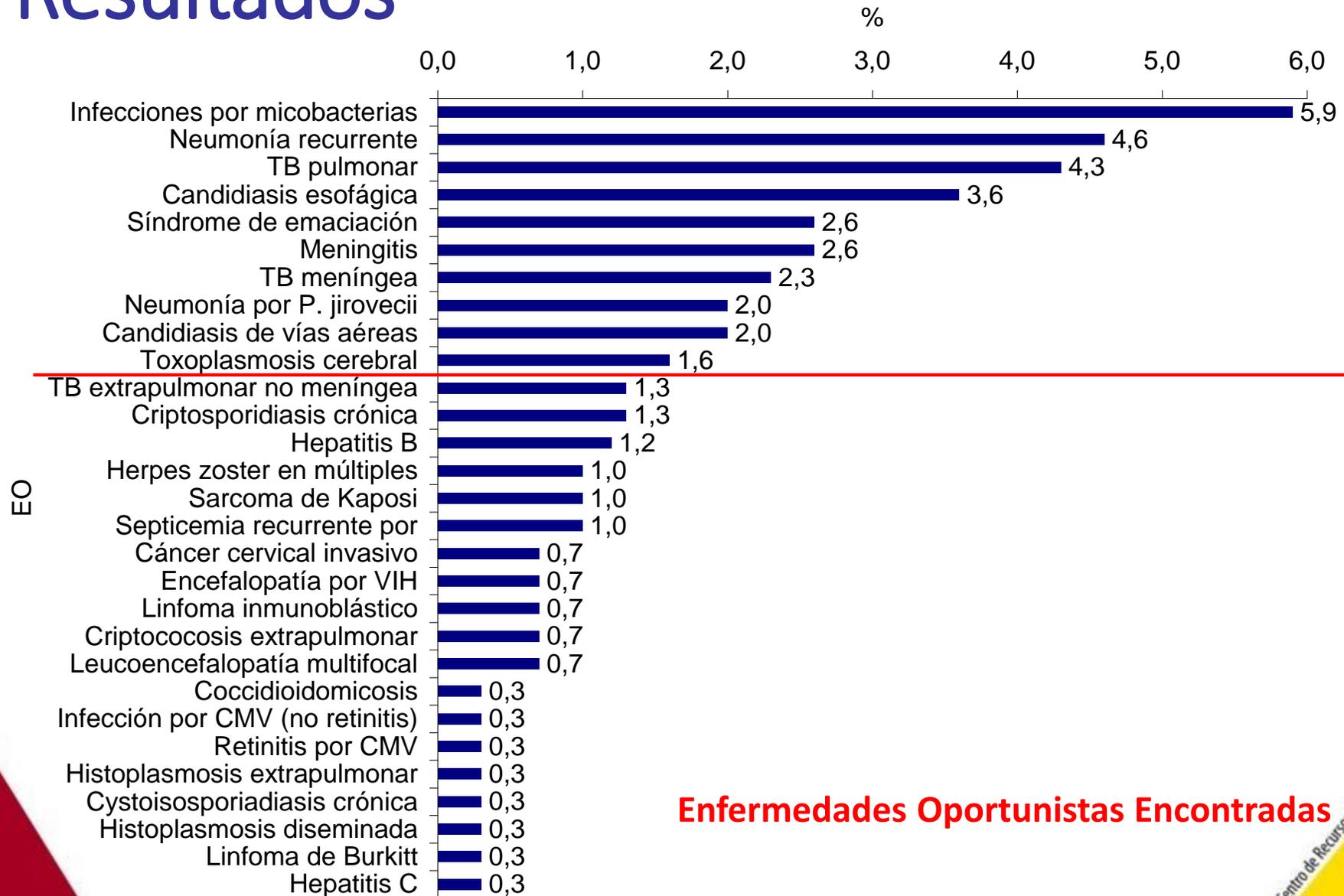
Ocurrencia de EO según afiliación a SGSSS

Afiliación	Ocurrencia de EO		Total
	Sí	No	
No	n	22	38
	%	36,7%	63,3%
Sí	n	54	191
	%	22,0%	78,0%
Total	n	76	229
	%	24,9%	75,1%

$\chi^2=5,511$; $p=0,019$; OR=2,048 (IC95% 1,117-3,753)



Resultados



Enfermedades Oportunistas Encontradas

Resultados

- **Letalidad**

- **7,2%, mayor en aquellos con EO (OR=6,3; IC95% 2,5-15,8)**

Muerte según ocurrencia de EO

Ocurrencia de EO	Muerte		Total
	Sí	No	
Sí	n	14	75
	%	18,7%	81,3%
No	n	8	229
	%	3,5%	96,5%
Total	n	22	305
	%	7,2%	92,8%

$\chi^2=19,376$; $p<0,001$; OR=6,34 (IC95% 2,543-15,810)



Resultados

- **Algunas oportunistas específicas fueron significativamente más frecuentes en aquellos ≥ 35 años**

Enfermedad Oportunista (%)	Edad (años)		OR	IC95%
	≥ 35	< 35		
Candidiasis esofágica	6,4	1,2	5,556	1,182-2,632
Síndrome de emaciación	5,0	0,6	8,547	1,035-71,429
Candidiasis de vías aéreas	4,3	0,0	1,045	1,009-1,082
TB extrapulmonar	2,8	0,0	1,029	1,001-1,059



Resultados

- Algunas oportunistas específicas conllevaron significativamente más a la muerte de los pacientes.

		Muerte (%)	OR	IC95%
Septicemia recurrente por <i>Salmonella</i>	Sí	66,7	28,100	2,442-323,34
	No	6,6		
Neumonía por <i>P. jirovecii</i>	Sí	50,0	14,684	2,774-77,725
	No	6,4		
Candidiasis esofágica	Sí	36,4	8,698	2,329-32,490
	No	6,2		
Meningitis	Sí	37,5	8,747	1,942-39,397
	No	6,4		
Candidiasis de la vía aérea	Sí	33,3	6,925	1,195-40,130
	No	6,7		
Leucoencefalopatía multifocal	Sí	50,0	13,381	0,808-221,601
	No	7,0		

- Solo la meningitis fue significativa en el análisis multivariado, para la muerte: $OR_{ajustado} = 7,738$ (IC95% 1,368-43,777).



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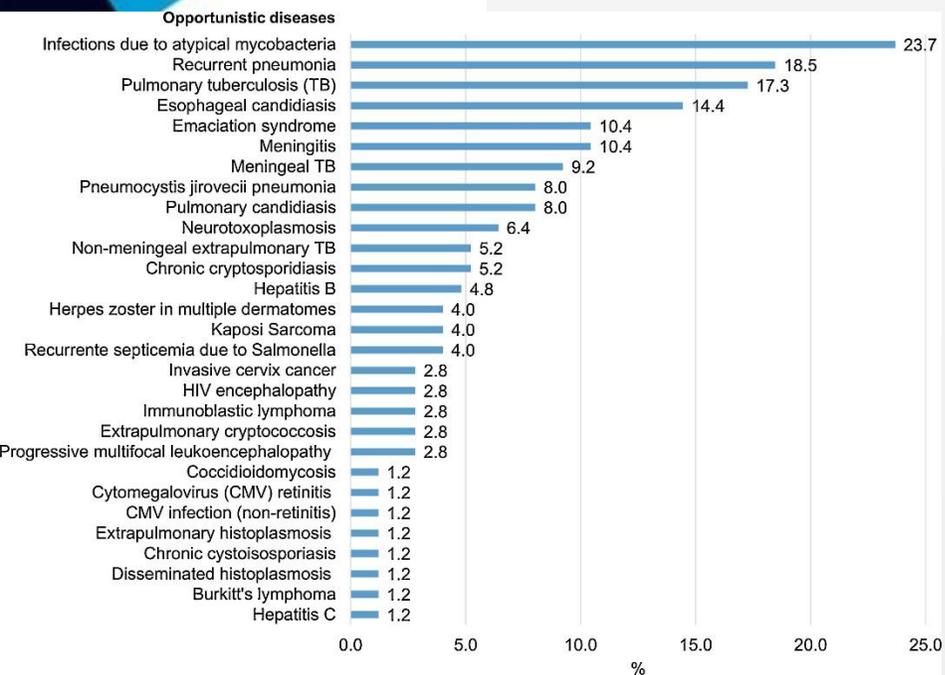
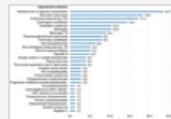


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Article outline

Funding
Conflict of interest
Ethical approval
Acknowledgment
References

Figures and tables



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Volume 6, Issue 6, December 2013, Pages 496–498



Letter to the Editor

Epidemiology of opportunistic diseases in AIDS patients from Pereira municipality, Colombia, 2010–2011

Paola A. Saldarriaga-Arenas^{a, b}

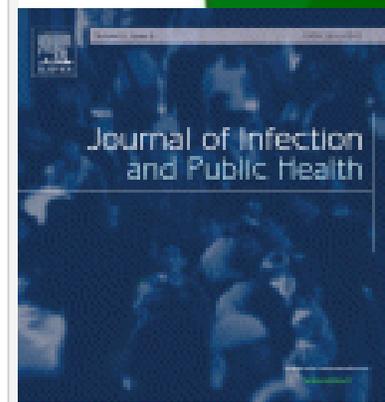
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Morbidity and mortality related to acquired immunodeficiency syndrome (AIDS)-defining opportunistic diseases (ODs) have been significantly reduced since the introduction of highly active anti-retroviral therapy (HAART). However, they still represented a significant epidemiological burden among patients with AIDS in some developing countries [1] and [2]. Even more, there is few recent data, particularly population-based, about the prevalence and factors associated to ODs in AIDS patients of some countries of South America, with limited access to HAART, such as Colombia [3] and [4]. Surveillance studies on it should be frequently done. According to the World Health Organization, this country is in the list of nations with 40–59% of eligible people receiving HAART at the end of 2011 [4].

For these reasons we assessed the prevalence of ODs in the population of AIDS patients living and attended in the municipality of Pereira, the capital area of Risaralda department, in western Colombia, during 2010–2011. This population is included in the HIV control program of Pereira municipality. Pereira (459.667 pop. for 2011) is one of the municipalities with highest incidence of HIV/AIDS in the country, 34.6 cases/100,000 pop. for 2011, with a significant increase in the last 6 years (2006–2011) [5].

Patients were diagnosed based on epidemiological, clinical and serological confirmation (ELISA HIV-1 and HIV-2 tests and Western-blot, with voluntary counseling and testing). Data was collected through the Epidemiological Surveillance System (SIVIGILA), HIV/AIDS trimester program reports and through HIV/AIDS treatment cohort reports. Opportunistic diseases were clinically, microbiologically and pathologically diagnosed. Collected data was compiled in Excel and then analyzed with SPSS v.17.0[®].



En las enfermedades infecciones es importante tomar en consideración los siguientes factores, para el análisis epidemiológico:

1. Descripción de los patrones de infección y su aparición en la población.
2. Determinar si las tasas del proceso infeccioso representa un aumento inusual o no endémico.
3. Apoyarse de toda la tecnología diagnóstica posible a fin de determinar etiológicamente el agente infeccioso involucrado.
4. Determinar la población de portadores asintomáticos o enfermedad subclínica.
5. Describir el comportamiento clínico de las enfermedades infecciosas que ocurren en la población, es decir, definir los casos.

Echezuria, Riskey, Fernández, Rodríguez-Morales.
Temas de Epidemiología y Salud Pública. Tomo I, 2013.



En las enfermedades infecciosas es importante tomar en consideración los siguientes factores, para el análisis epidemiológico:

6. Ayudar a la comprensión de la patogenia de la enfermedad.
7. Identificar la cadena de infección que contribuye a la transmisión del agente.
8. Evaluar y dirigir los protocolos diagnósticos y de tratamiento.
9. Desarrollar programas de prevención y medidas de control para los individuos.
10. Ampliar dicha vigilancia a un rango poblacional de manera permanente.

Echezuria, Risquez, Fernández, Rodríguez-Morales.
Temas de Epidemiología y Salud Pública. Tomo I, 2013.



Figura 1

Triángulo epidemiológico

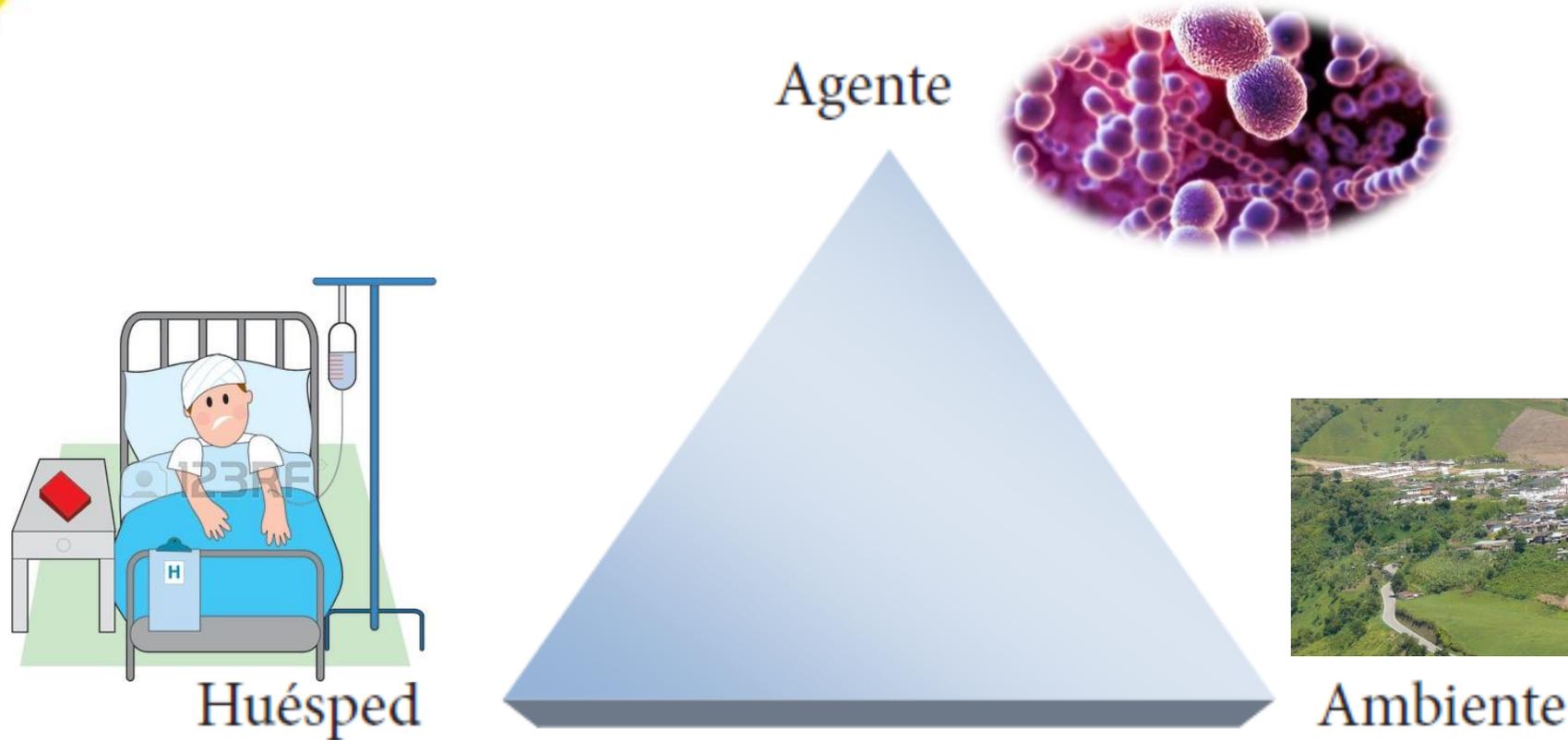
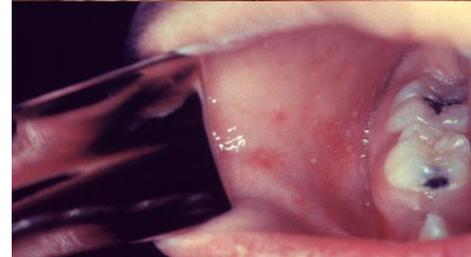
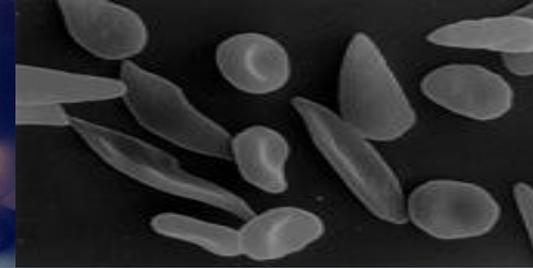


Figura 1. Modelo de causalidad del triángulo epidemiológico.

Factores del Huésped

- Edad
- Genética
- Sexo
- Estado Socioeconómico
- Inmunidad
- Estado nutricional
- Hábitos, estilos y calidad de vida
- Grado de instrucción o escolaridad
- Grupo étnico, religioso
- Estados mórbidos previos



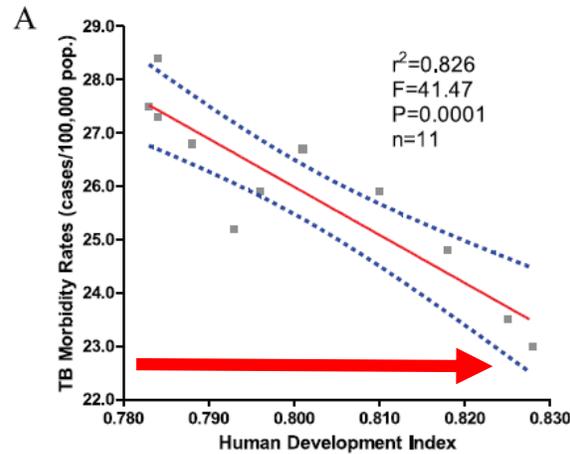


Más educación, menos TB

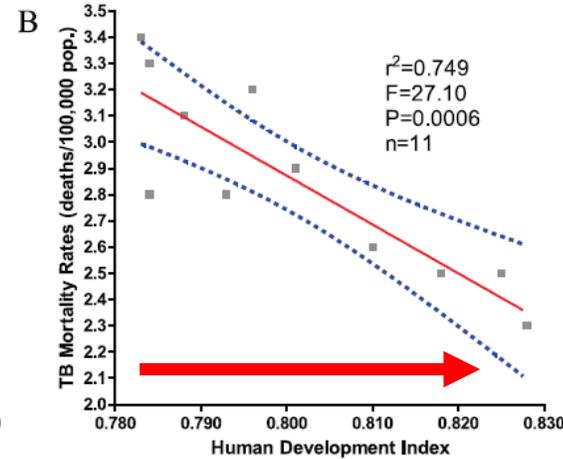
Rodríguez-Morales AJ, Castañeda-Hernández DM. Relationships between morbidity and mortality from tuberculosis and the human development index (HDI) in Venezuela, 1998-2008. *Int J Infect Dis.* 2012 Sep;16(9):e704-5.



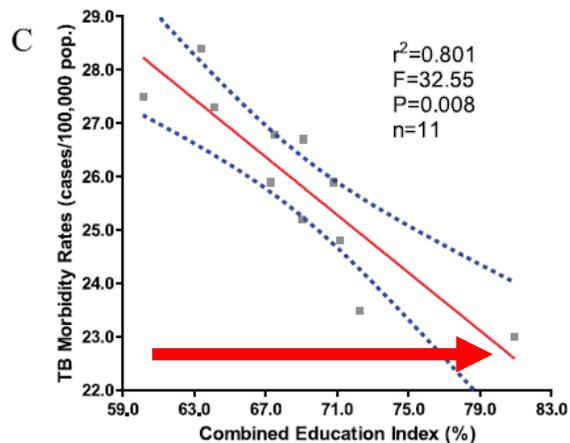
**Mejor Índice de
Desarrollo Humano
Menos
Morbilidad por TB**



**Mejor Índice de
Desarrollo Humano
Menos
Mortalidad por TB**



**Más Educación
Menos
Morbilidad por TB**



**Más Educación
Menos
Mortalidad por TB**

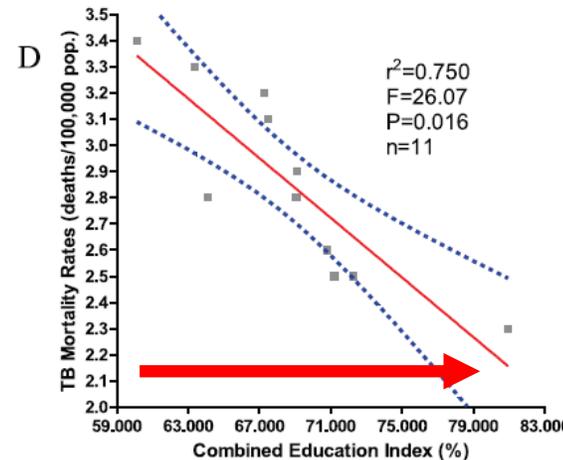


Figure 1. Relationships between the human development index (HDI) and morbidity and mortality rates due tuberculosis in Venezuela, 1998-2008. (A) Linear regression between HDI and TB morbidity rates. (B) Linear regression between HDI and TB mortality rates. (C) Linear regression between education and TB morbidity rates. (D) Linear regression between education and TB mortality rates.

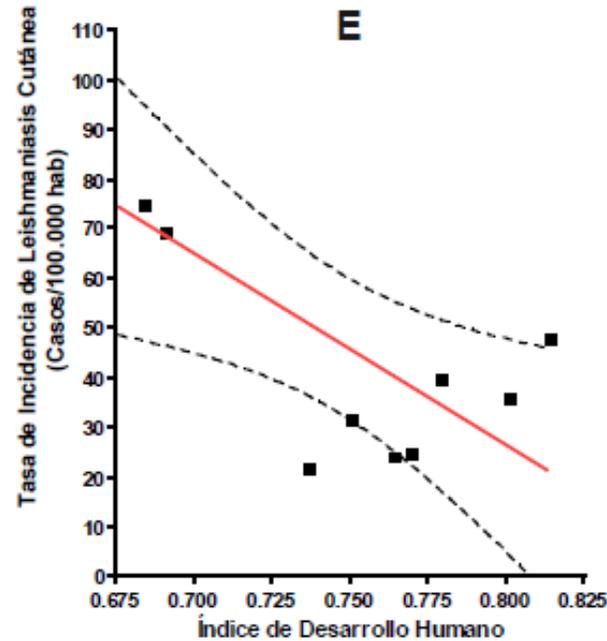
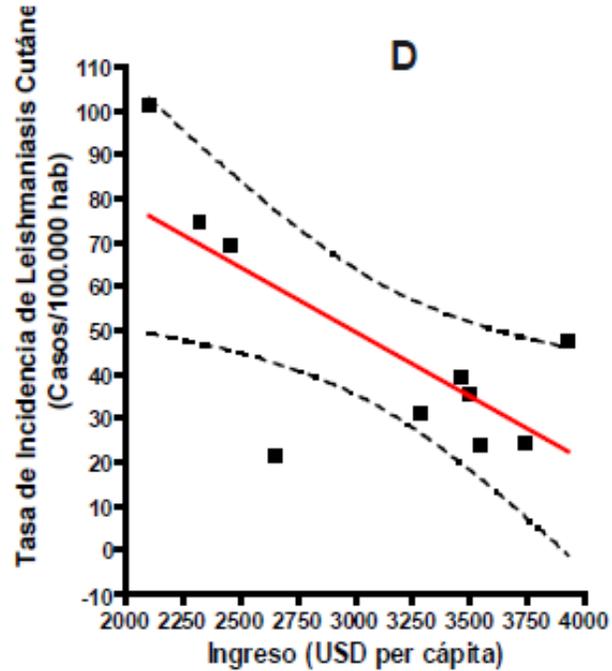
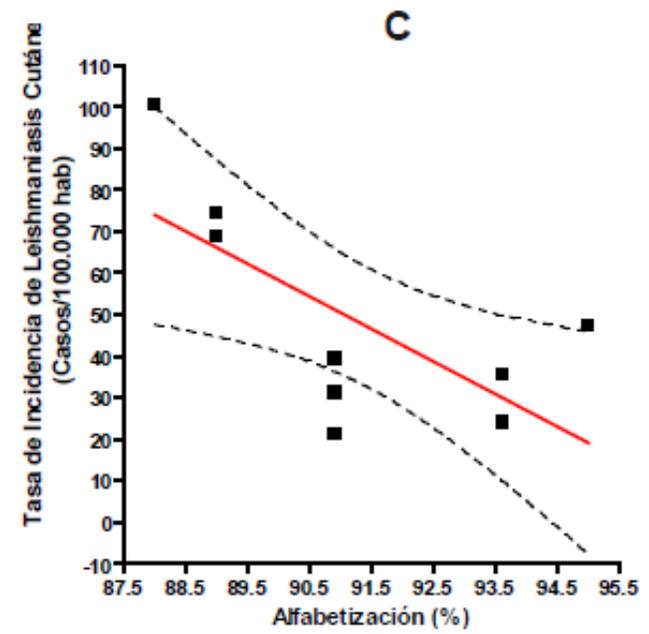
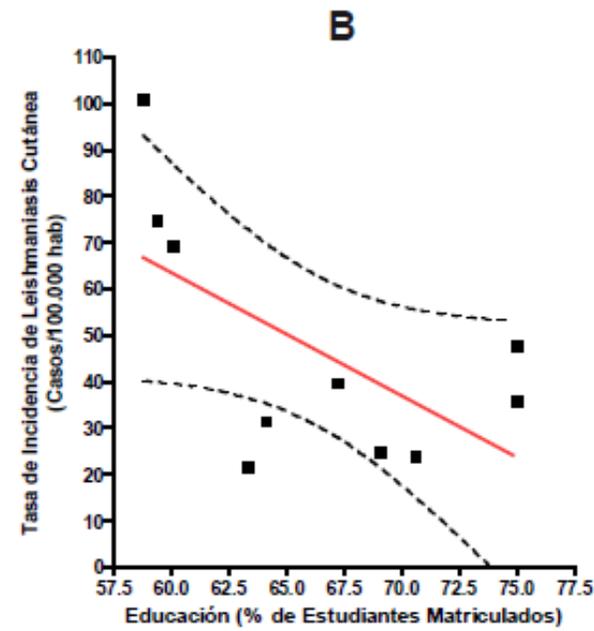
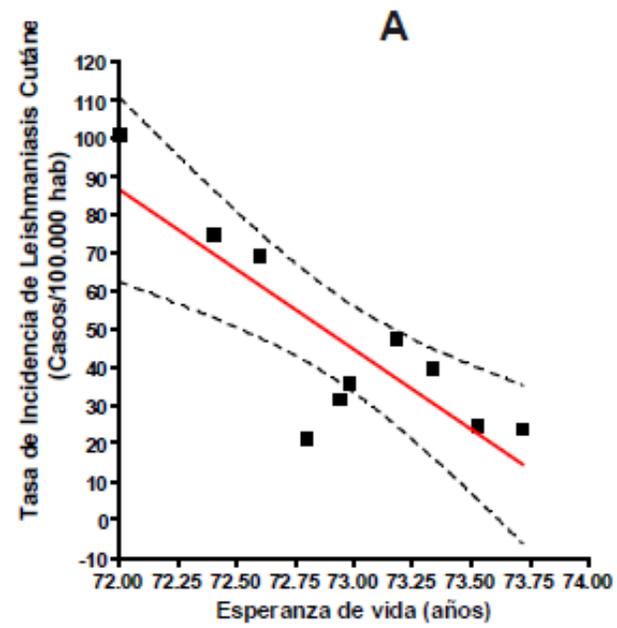
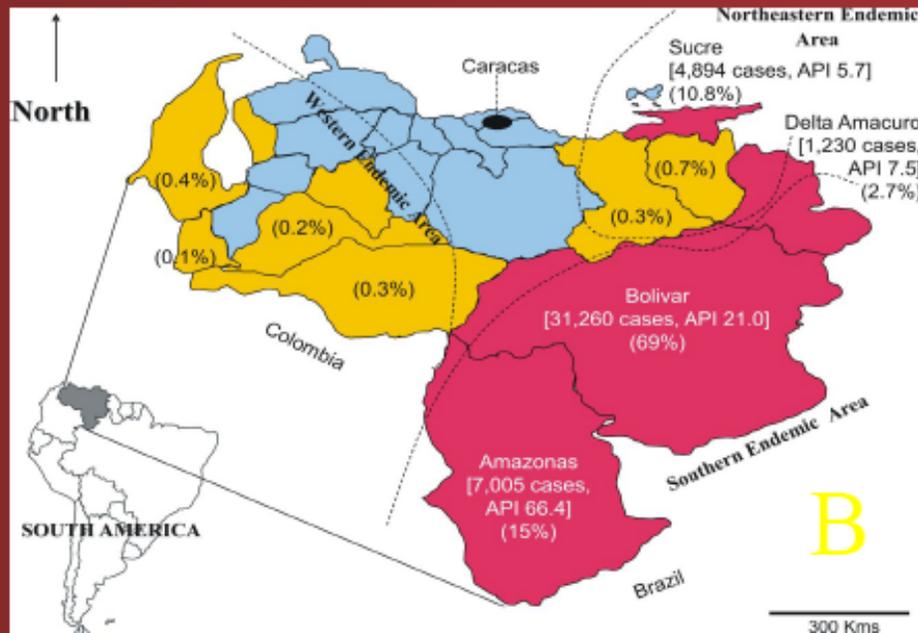
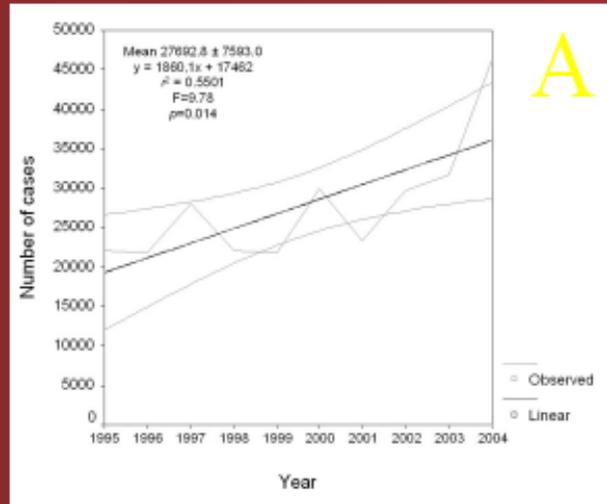


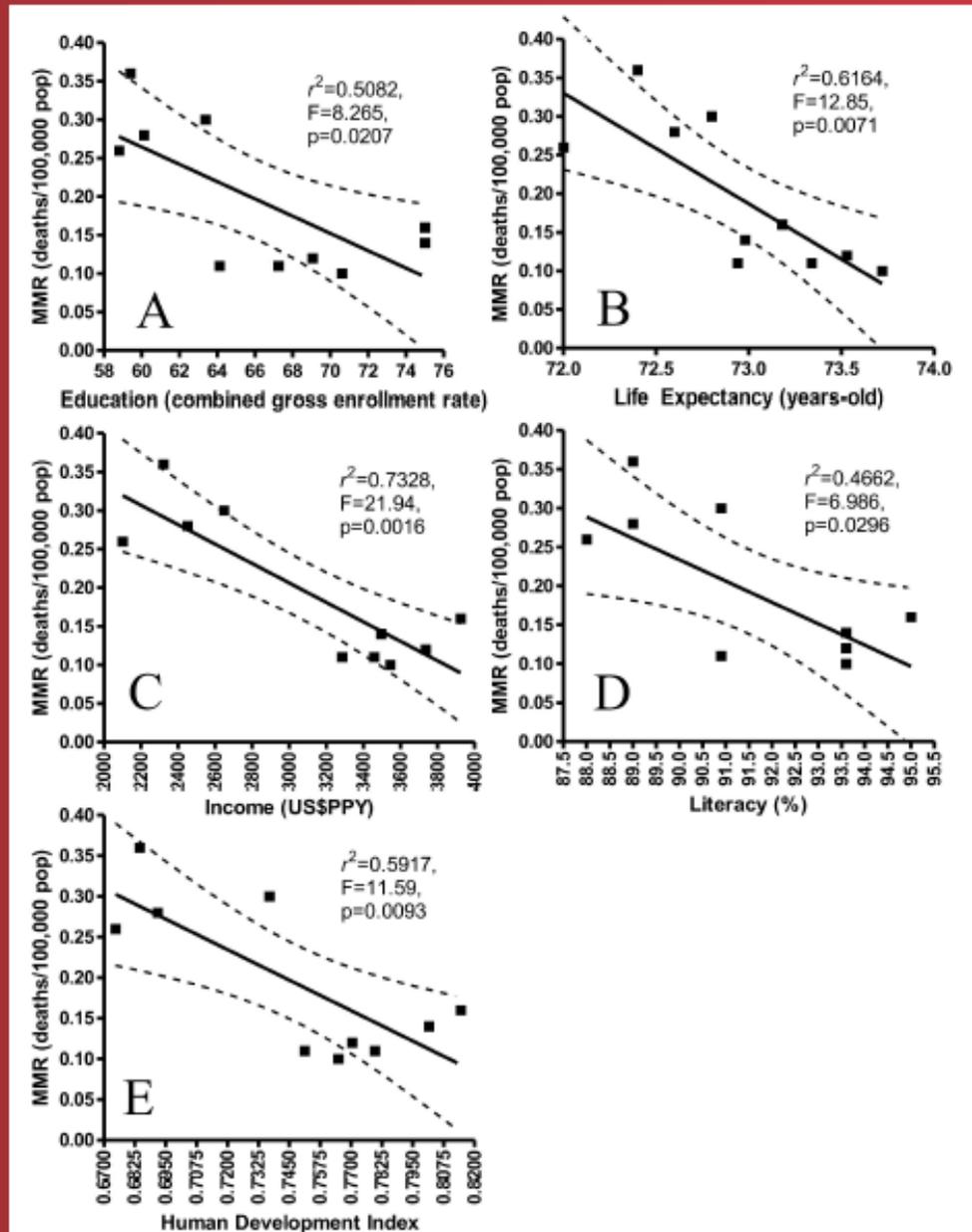
Figura 4. Regresiones lineales entre la tasa de incidencia de la Leishmaniasis cutánea y las variables socioeconómicas: (A) educación, (B) esperanza de vida, (C) ingreso, (D) alfabetización e (E) IDH en el Estado Trujillo (Venezuela), 1994-2003.

Figure 1. Malaria morbidity in Venezuela, 1995-2004 (A) and spatial distribution of cases for year 2004 (B) [13, 14].



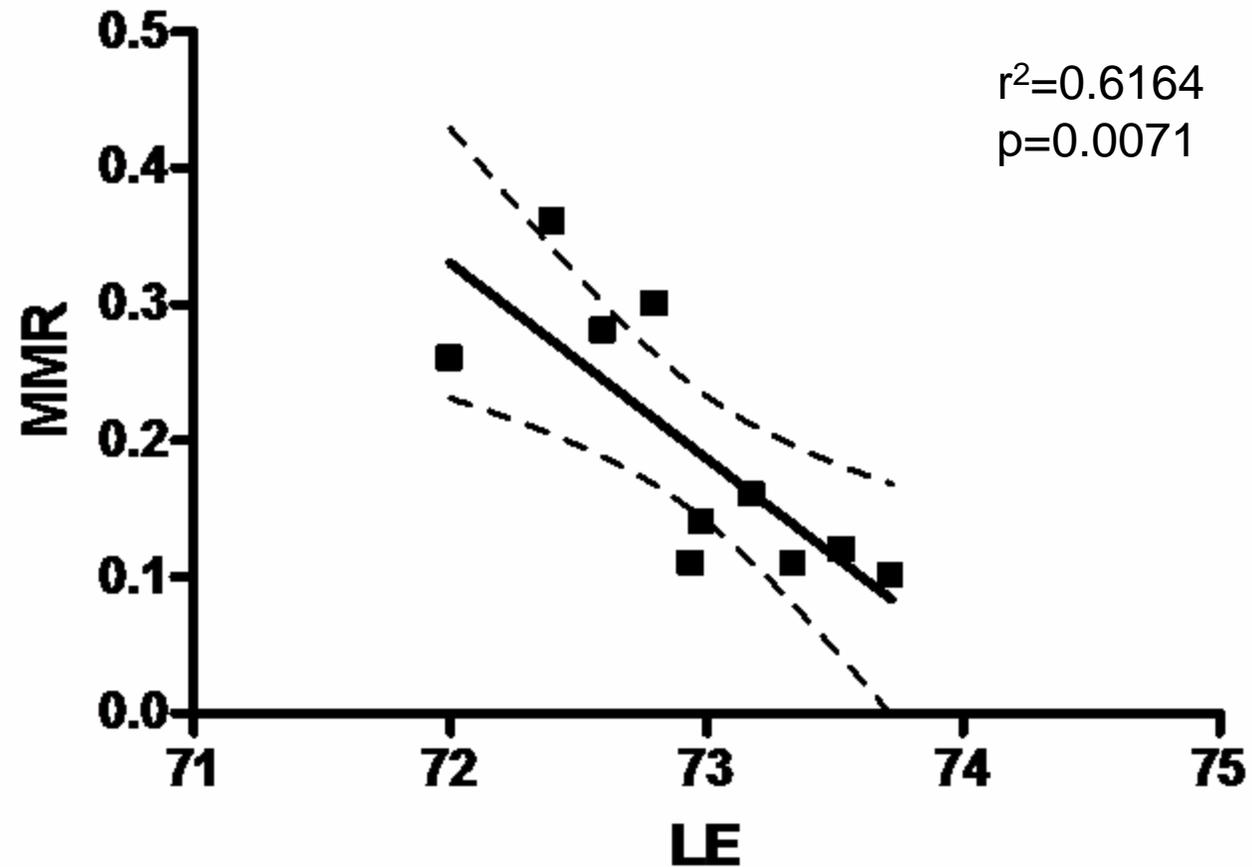
Note: States in red are those with high levels of transmission, contributing with more than 10% of country cases and/or an Annual Parasitic Index (API) (malaria cases/1,000 pop.) higher than 5.0; states in yellow are those with low-to-moderated levels of transmission, contributing with 0.1-10% of country cases and/or an API of 0.5-5.0; and states in blue are those non-endemic.

Figure 2. Linear regressions between malaria mortality rates (MMR) and socioeconomic variables (A, education, B, life expectancy, C, income, D, literacy and E, HDI), Venezuela, 1995-2004.



Venezuela, 1995-2004

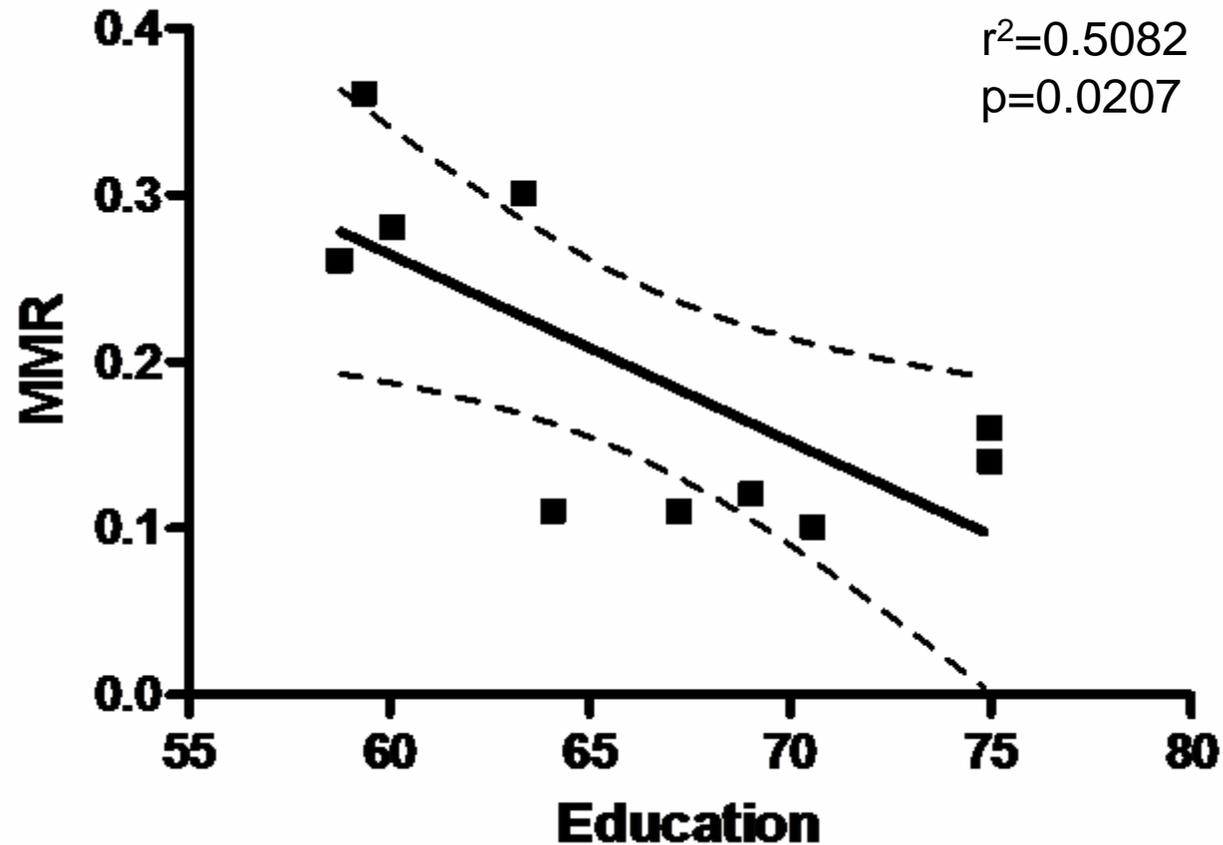
Malaria Mortality



MMR, malaria mortality rate (deaths/100,000pop)
LE, Life Expectancy (years)

Venezuela, 1995-2004

Malaria Mortality



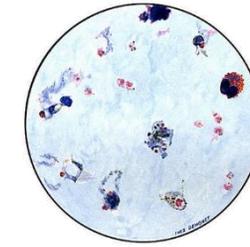
MMR, malaria mortality rate (deaths/100,000pop)
Education (combined gross enrollment ratio)

Determinantes Sociales de la Malaria y las Parasitosis Intestinales en Latinoamérica

Educación y Parasitosis

- Se encontró que aquellos sujetos con madres que tienen primaria incompleta tienen **5,237 (IC 95%: 5,164 – 5,310)** veces más riesgo de sufrir ascariasis, que aquellos cuyas madres tienen primaria completa hasta universitaria.
- Mientras que aquellos sujetos cuyas madres son analfabetas tienen casi **2 veces más riesgo de padecer esta parasitosis (OR 1,846 IC 95%: 1,782-1,912)**.

Malaria y Diabetes Mellitus



- Estudio de casos y controles
 - n=1466 (675 con DM)

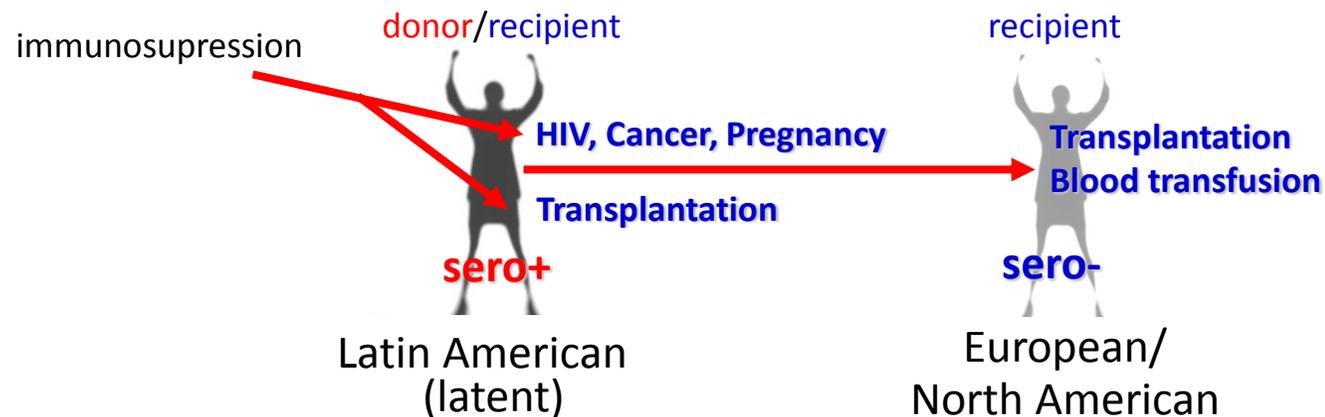
Table 2. Univariate and multivariate associations with *Plasmodium falciparum* infection, Kumasi, Ghana, 2007–2008*

Parameter	Total no. patients	<i>P. falciparum</i> infection, no. (%)	Univariate analysis		Multivariate analysis	
			OR (95% CI)	p value	aOR (95% CI)	p value
Diabetes mellitus type 2						
No	791	81 (10.3)	1			
Yes	675	108 (16.0)	1.67 (1.22–2.27)	0.001	1.46 (1.06–2.03)	0.021
Gender						
F	1,113	124 (11.2)	1			
M	353	65 (18.5)	1.80 (1.29–2.50)	<0.0001	2.13 (1.50–3.03)	<0.0001
Wealth score						
≥25th percentile	923	94 (10.2)				
<25th percentile †	536	94 (17.6)	1.88 (1.38–2.56)	<0.0001	1.76 (1.27–2.42)	0.001
Literacy						
Able to read	947	103 (10.9)	1			
Unable to read	514	85 (16.6)	1.63 (1.20–2.23)	0.002	1.59 (1.11–2.28)	0.011

Risks for Chagas Disease



- *Trypanosoma cruzi* and immunosuppression
 - Reactivation of latent infection in immunosuppressed individuals, such as the HIV-infected and transplant recipients
 - Reactivates from the donated organ or in a previously infected host due to immunosuppression in transplant recipients
 - Transmitted through blood transfusion.

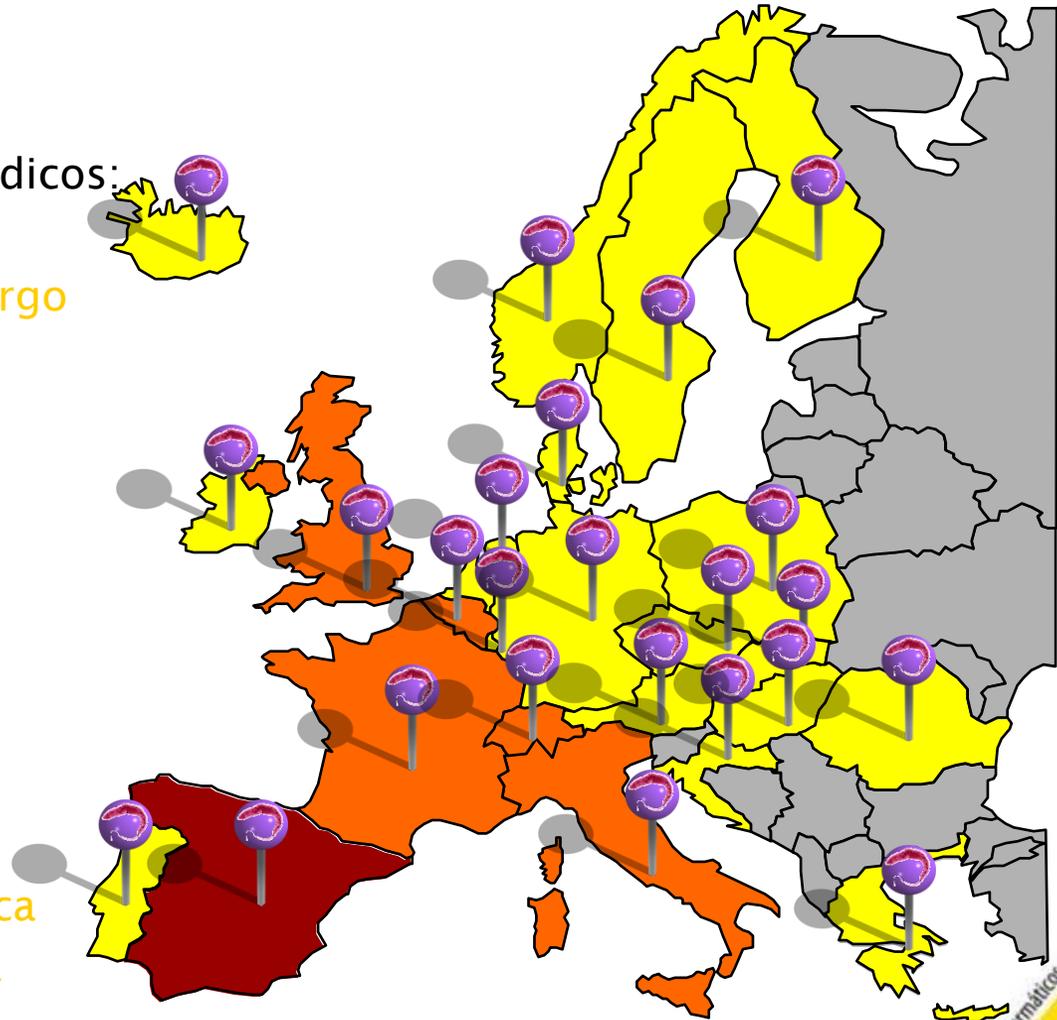


Enfermedad de Chagas en el Viejo Continente



• Países Afectados

- Norte América:
 - EUA
 - Canada
- Europa:
 - España
 - RU
 - Italia
 - Francia
 - Suiza
 - Bélgica
- Casos esporádicos:
 - Austria
 - Croacia
 - Dinamarca
 - Alemania
- Casos esporádicos:
 - Grecia
 - Luxemburgo
 - Holanda
 - Noruega
 - Portugal
 - Rumania
 - Suecia
 - Irlanda
 - Polonia
 - Islandia
 - Finlandia
 - Rep. Checa
 - Eslovaquia
 - Hungría



Chagas y Diabetes Mellitus



- Estudio de casos y controles
 - n=647 (362 con Chagas)

Tabela 2 - Características gerais e metabólicas do grupo controle e das formas cardíaca, megas e assintomática, registradas em 647 mulheres com idade > 40 anos, atendidas no HE-FMTM.

Parâmetros	Controle (n = 285)	Cardíaca (n = 178)	Megas (n = 58)	Assintomática (n = 125)
Idade (anos) ^(a)	57,0 ± 11,3	57,8 ± 10,5	60,0 ± 10,1	55,4 ± 10,5
Cor (branca, em %)	75,8	75,4	75,9	80,0
IMC (kg/m ²)	26,2 ± 5,6	27,7 ± 5,9	23,1 ± 4,3	27,0 ± 5,8
Glicemia 1 (mg/dl) ^(b)	95 (60-363)	100 (62-445)	94,5 (69-309)	95 (52-100)
Glicemia 2 (mg/dl)	98,5 (70-476)	101 (60-346)	98 (61-338)	94 (71-352)
Hiperglicemia (%) ^(b)	26,7	37,4	25,9	27,2
DM (%) ^(b)	7,4	15,1	7,4	5,6

IMC = índice de massa corporal. DM = diabetes *mellitus*. ^(a) Grupo com a forma megas difere dos demais. ^(b) Grupo com a forma cardíaca difere dos demais.

10,5%

Leishmaniasis y Diabetes Mellitus



- Reportes de Casos – Formas diseminadas



Case Report

Open Access

HIV, visceral leishmaniasis and Parkinsonism combined with diabetes mellitus and hyperuricaemia: A case report

Krishna Pandey*, Prabhat Kumar Sinha, Vidya Rabidas, Nawin Kumar, Sanjiva Bimal, Neena Verma, Chandrasekhar Lal and Pradeep Das

Address: Rajendra Memorial Research Institute of Medical Sciences (Indian Council of Medical Research), Agamkuan, Patna, – 800 007, India

Email: Krishna Pandey* - drkrishnapandey@yahoo.com; Prabhat Kumar Sinha - pksinha18@yahoo.com;
Vidya Rabidas - drvnrdas@yahoo.com; Nawin Kumar - drnawinkumar@gmail.com; Sanjiva Bimal - drsbimal@yahoo.com;
Neena Verma - verma_neena@yahoo.com; Chandrasekhar Lal - drclslal@sify.com; Pradeep Das - drpradeep.das@gmail.com

* Corresponding author

Published: 25 September 2008

Received: 11 September 2008

Cases Journal 2008, 1:183 doi:10.1186/1757-1626-1-183

Accepted: 25 September 2008

Short communication

Is there an association between positive *Strongyloides stercoralis* serology and diabetes mellitus?

Suzan C.L. Mendonça, Maria do Rosário F. Gonçalves-Pires,
Rosângela M. Rodrigues, Álvaro Ferreira Jr., Julia M. Costa-Cruz*

- The frequency of positive *S. stercoralis* serology in diabetics was **23%** versus **7.1%** in the control group ($P < 0.05$).
- The odds ratio for diabetics was **3.9** (CI, 1.6–15.9, $P < 0.05$).
- Diabetic patients with $HbA1c \leq 7$ had a greater chance of testing negatively for *S. stercoralis* infection (OR: 1.5, $P > 0.05$).

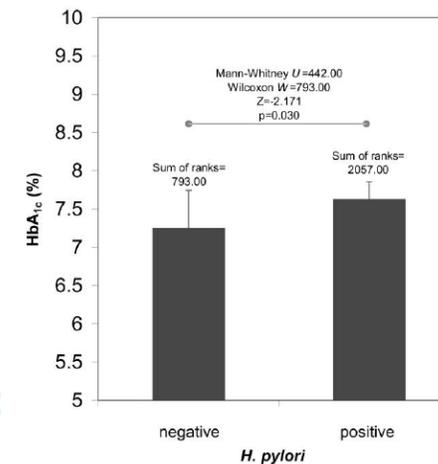
Diabetes Mellitus, Insulin, and Melioidosis in Thailand

Andrew J. H. Simpson,^{1,3,a} Paul N. Newton,^{1,3} Wirongrong Chierakul,¹ Wipada Chaowagul,² and Nicholas J. White^{1,3}

CID 2008:47 (1 July) • 144-6

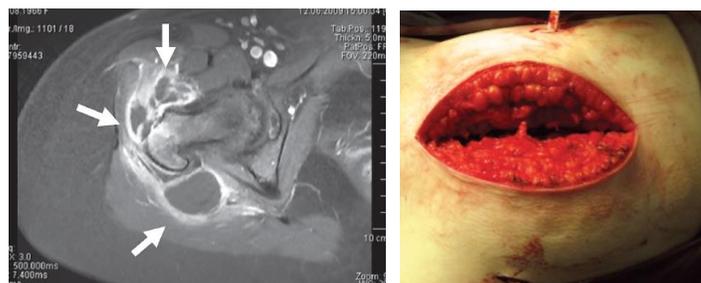
In Patients with Type 2 Diabetes Mellitus, Are Glycosylated Hemoglobin Levels Higher for Those with *Helicobacter pylori* Infection Than Those without Infection?

Gino G. Fernandini-Paredes,¹
Edward Mezones-Holguin,^{1,2}
Rolando Vargas-Gonzales,¹
Eugenio Pozo-Briceño,¹
and Alfonso J. Rodriguez-Morales^{3,4}



Pyomyositis associated with diabetes mellitus and liver cirrhosis

Daniela Vichiato Polizelli¹, Geise Geraldino Cristina¹, Eduardo Narvaes², Elisete Funes⁴,
Acayaba Roberto de Toledo⁵, Rita Menin⁴
Bras J Rheumatol 2010;50(4):472-7



TB y DM: Convergencia de 2 epidemias

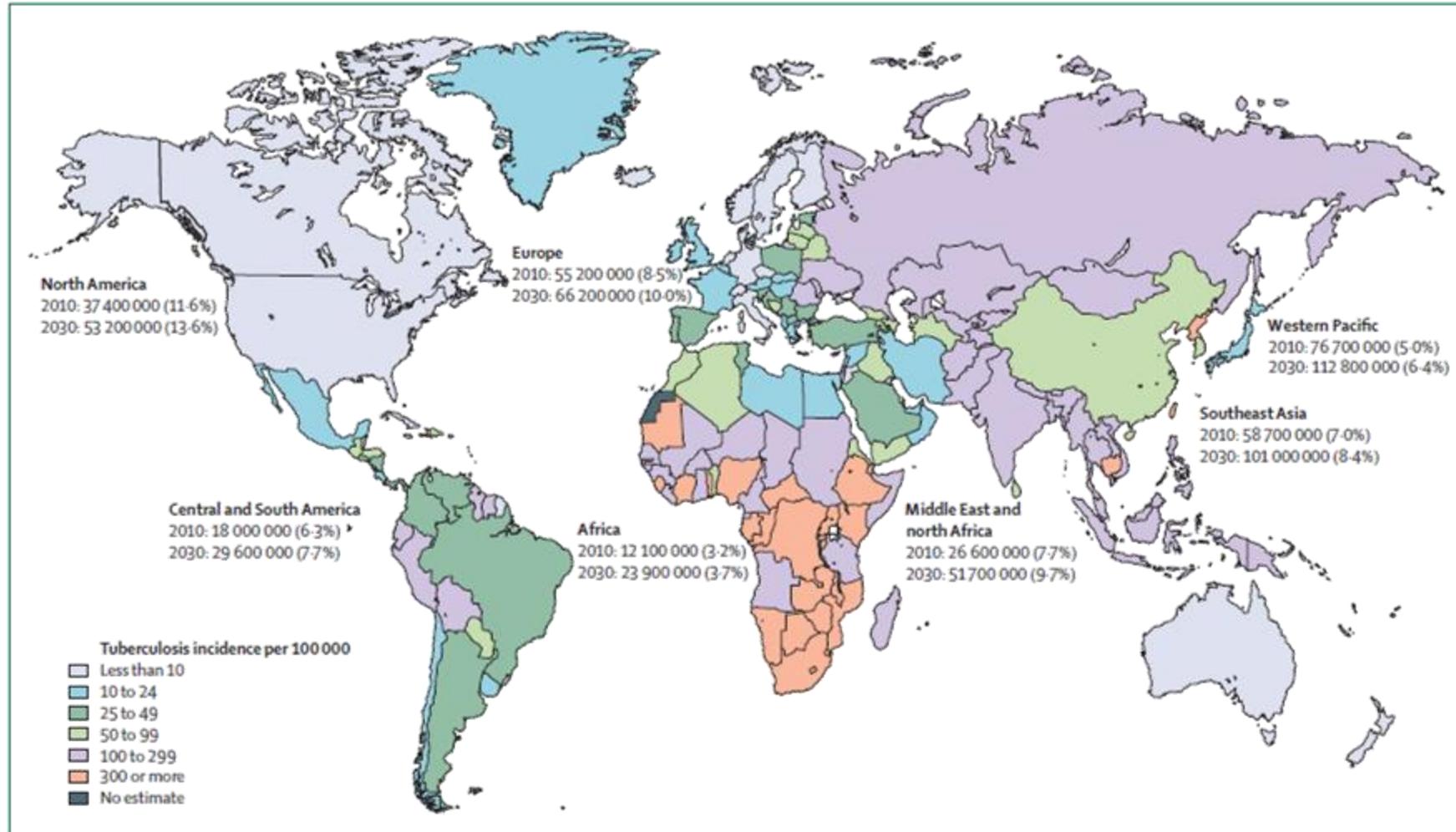


Figure: Projected prevalent diabetes cases and current worldwide tuberculosis incidence

Estimated number and percent of individuals with diabetes mellitus in 2010 compared with 2030 projections are shown. Tuberculosis incidence per 100 000 population data for 2007 are shown. Data from International Diabetes Foundation and WHO.^{10,11}

DM como factor de riesgo para TB

	Year	Location	Setting	Type of study	Participants (n)	Outcome variable and findings
Boucot et al ²⁵	1952	Philadelphia, USA	..	Chest radiograph survey comparing DM patients with healthy industrial workers	73 873	Prevalent TB by chest radiograph: 8.4% in DM (2 times that of controls)
Olmos et al ²⁶	1989	Chile	Teaching hospital	Retrospective cohort of DM patients followed 10 years	1529	10-year risk of TB in IDDM or NIDDM vs general population: 24% IDDM, 4% NIDDM, 0.8% general population
Swai et al ²⁷	1990	Tanzania	Inpatient and outpatient clinics	Prospective cohort of DM patients followed 1-7 years	1250	Risk of pulmonary TB: 9.0% in IDDM, 2.7% in NIDDM
Bermejo et al ²⁸	1995	Spain	General medicine clinics	Cross-sectional study of TST results in DM patients	163	TST positivity at 3 days: 42.2%
Kim et al ²⁹	1995	Korea	Civil servants	Longitudinal cohort study using insurance claims	8015 cases, 806 698 controls	RR of developing pulmonary TB: 3.47 (DM vs non-DM); 95% CI 1.19-1.45
Pablos-Mendez et al ³⁰	1997	California, USA	Inpatient hospitals	Case-control study using discharge diagnoses	5290 cases, 37 366 controls	OR of DM comparing tuberculosis patients with patients with deep venous thrombosis, pulmonary embolus, or appendicitis: 2.95 for Hispanic people (95% CI 2.61-3.33), 1.31 for white people (1.19-1.45)
Mboussa et al ³¹	2003	Congo	University hospital	Case-control study using chart review	32 cases, 100 controls	OR for TB: 8.33 (DM vs non-DM)
Shah and Hux ²⁵	2003	Ontario, Canada	Inpatient and outpatient	Retrospective cohort study using province-wide administrative database	513 749 in each group	OR for TB: 1.12 (DM vs non-DM); 95% CI 1.03-1.23
Ponce-De-León et al ³²	2004	Mexico	Inpatient and outpatient clinics	Population-based cohort linked to statewide cluster household survey	1915	IRR of TB: 6.8 (DM vs non-DM); 95% CI 5.7-8.2
Coker et al ²⁹	2006	Russia	TB clinics in urban setting	Case-control study with controls sampled from general population	334 cases, 334 controls	AOR for TB: 7.83 (DM vs non-DM), controlling for assets, overcrowding, employment, and financial security; 95% CI 2.37-25.9
Jabbar et al ³⁰	2006	Pakistan	Teaching hospital	Case-control study using discharge diagnoses	1458 cases, 40 900 controls	OR for TB: 7.83 (DM vs non-DM); 95% CI 6.55-9.37
Jick et al ³³	2006	UK	General practices	Case-control study using large countrywide database	497 cases, 1966 controls	AOR for TB: 3.8 (DM vs non-DM), adjusting for steroid use, smoking, body-mass index, pulmonary diseases, immunosuppressive use; 95% CI 2.3-6.1
Perez et al ³⁴	2006	Texas, USA	Inpatient clinic	Case-control study using hospital discharge database	4915 cases, 70 808 controls	AOR for TB (DM vs non-DM), adjusting for sex, age, and race/ethnicity: 1.51 in non-border Texas (95% CI 1.36-1.67), 1.82 in counties bordering Mexico (95% CI 1.57-2.12)
Shetty et al ³⁵	2006	India	Outpatient clinic	Matched case-control study using chart review; controls were relatives of cases.	189 cases, 189 controls	OR for TB: 2.44 (patients with diabetes, hypertension, or heart disease vs those without), matched for age and sex; 95% CI 1.17-5.09
Dyck et al ³⁶	2007	Saskatchewan, Canada	Inpatient and outpatient clinics	Retrospective cohort study using large health database	2122 cases*	IRR for TB: 1.53 (DM vs non-DM); 95% CI 1.25-1.87
Leung et al ³⁷	2008	Hong Kong	Elderly health service	Prospective population-based cohort	42 116	AHR for TB: 1.77 (95% CI 1.41-2.24), DM vs non-DM; 3.11 (95% CI 1.63-5.92) in diabetics with HbA1C > 7% vs HbA1C < 7%

DM como factor de riesgo para TB

	Year	Study location	Participants (n)		Lower lung more commonly involved?	More cavitary lesions?	More diffuse involvement?
			With diabetes	Without diabetes			
Weaver ⁴⁹	1974	USA	20	182	Yes	--	--
Marais ⁵⁰	1980	South Africa	9	427	Yes	--	--
Ikezoe et al ⁵⁴	1992	Japan	31†	71	No	Yes	Yes
Morris et al ⁵⁵	1992	Texas, USA	20	20	No	No	No
Umut et al ⁵⁶	1994	Turkey	37	37	No	Yes	Yes
Kuaban et al ⁵⁷	1996	Cameroon	--	273‡	Yes	--	--
al-Wabel et al ⁵⁸	1997	Saudi Arabia	28	38	No	--	--
Bacakoglu et al ⁵⁹	2001	Turkey	92	92	No§	No§	No
Perez-Guzman et al ^{60,61}	2000-01	Mexico	192	130	Yes	Yes	Yes
Shaikh et al ⁶²	2003	Saudi Arabia	187	505	Yes	--	--
Wang et al ⁶³	2005	Taiwan	99	362	No	Yes	--
Wang et al ⁶⁴	2008	Taiwan	74	143	Yes	Yes	--
Al-Tawfiq et al ⁵¹	2009	Saudi Arabia	57	78	--	No	--

*Apart from the study by Ikezoe et al⁵⁴ in which computed tomography was used. †Patients with diabetes mellitus or who were immunocompromised. ‡Patients with tuberculosis, of whom 28 had lower-lung disease. §Insulin-dependent diabetes mellitus had more cavitary disease than non-insulin-dependent diabetes mellitus; in subgroup analysis, diabetes mellitus was a risk factor for lower-lung disease in patients aged >40 years. --=not reported.

Table 2: Studies assessing chest radiographic findings* in patients with tuberculosis, comparing diabetic to non-diabetic patients

DM como factor de riesgo para TB

	Year	Location	Setting	Type of study	Participants (n)	Outcome variables and findings (diabetes vs non-diabetes)*
Treatment failure						
Morsy et al ⁷⁷	2003	Egypt	TB treatment centres	Case-control study assessing risk factors for treatment failure, matched for sex and centre	119 cases, 119 controls	Crude OR 3.91 (1.65-9.5) for sputum smear positivity after 5 months of treatment; AOR 9.32 (2.7-31.7) adjusted for factors including age, sex, distance to tuberculosis centre, health education, and disease knowledge
Alisjahbana et al ⁷⁸	2007	Indonesia	Outpatient clinics	Prospective cohort study of new pulmonary TB patients	634	Proportion with positive sputum culture at 6 months: 22.2% vs 6.9%. AOR 7.65 (1.89-30.95), adjusted for age, sex, BMI, radiographic findings, 2-month sputum results, non-compliance, and drug resistance
Mortality						
Oursler et al ⁷³	2002	Maryland, USA	Outpatient clinic	Retrospective cohort study of culture-confirmed TB patients	139	HR 4.8 (2.0-11.6), AHR 6.7 (1.6-29.3), adjusted for renal disease, COPD, HIV infection, and age
Mboussa et al ⁷⁹	2003	Congo	University hospital	Case-control study using chart review	32 cases, 100 controls	25.1% vs 8%
Lindoso et al ⁸¹	2008	Sao Paulo, Brazil	Urban	Retrospective study of all TB-related deaths using death certificates, surveillance data, hospital records	416	Proportion of patients with TB-related death who had diabetes mellitus: 16%
Dooley et al ²⁰	2009	Maryland, USA	TB patients in three counties	Retrospective cohort study of culture-positive TB patients	297	OR 2.0 (0.74-5.2), AOR 6.5 (1.1-38.0), adjusted for HIV status, age, weight, and foreign birth
Wang et al ⁸⁴	2009	Taiwan	Teaching hospital	Retrospective study of culture-positive pulmonary TB patients	217	OR 2.56 (1.08-6.03), AOR 5.5 (2.27-13.5), adjusting for age and sex

* Unless otherwise indicated. AHR=adjusted hazards ratio (HR). AOR=adjusted odds ratio (OR). BMI=body mass index. COPD=chronic obstructive pulmonary disease.

Table 4: Studies assessing the effect of diabetes mellitus on treatment failure and death in patients treated for tuberculosis (TB)

Factores del Agente

Bouزيد M, Hunter PR, Chalmers RM, Tyler KM.
Cryptosporidium pathogenicity and virulence.
Clin Microbiol Rev. 2013 Jan;26(1):115-34

- Virulencia
- Resistencia
- Infectividad
- Patogenicidad

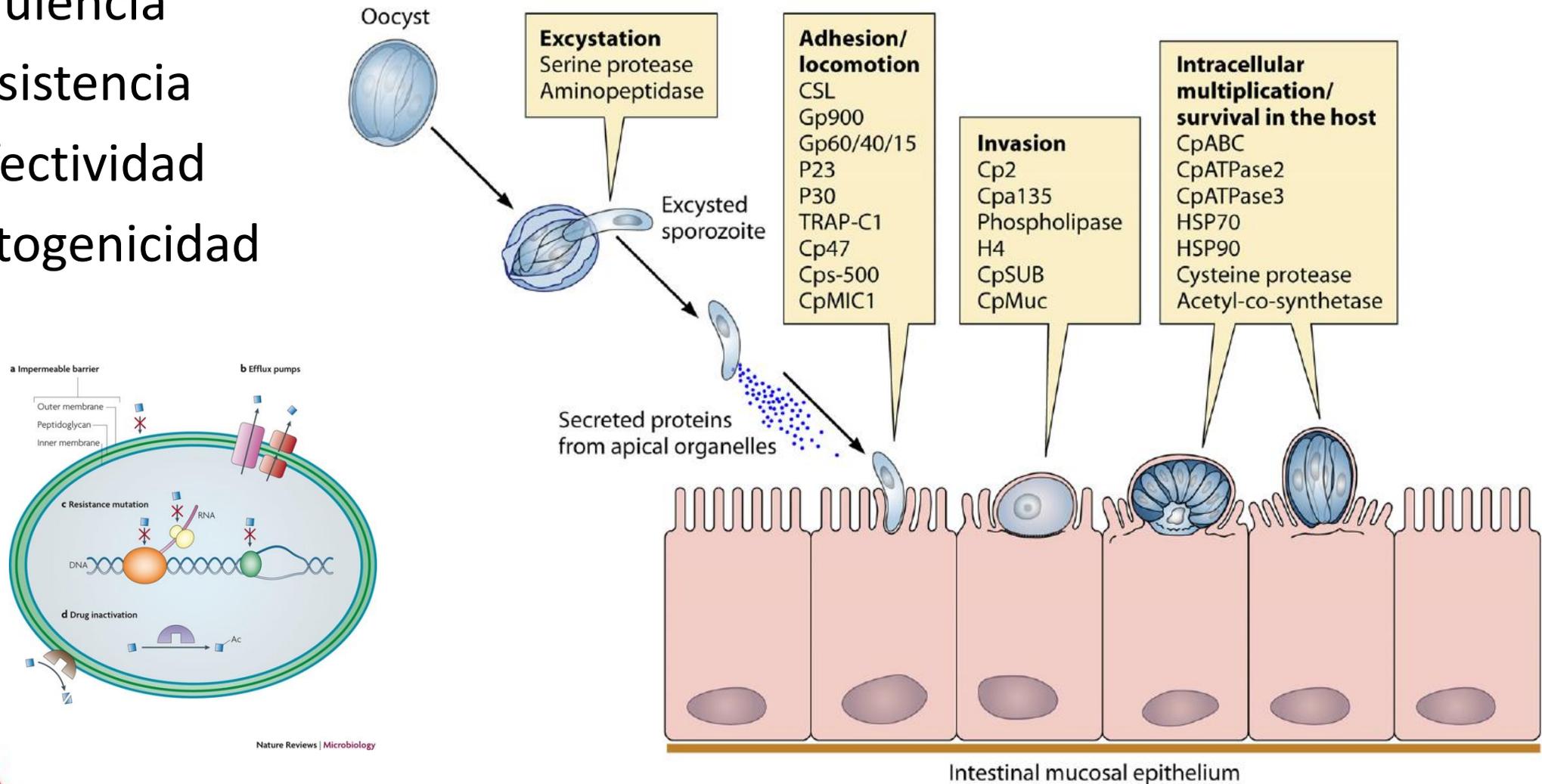
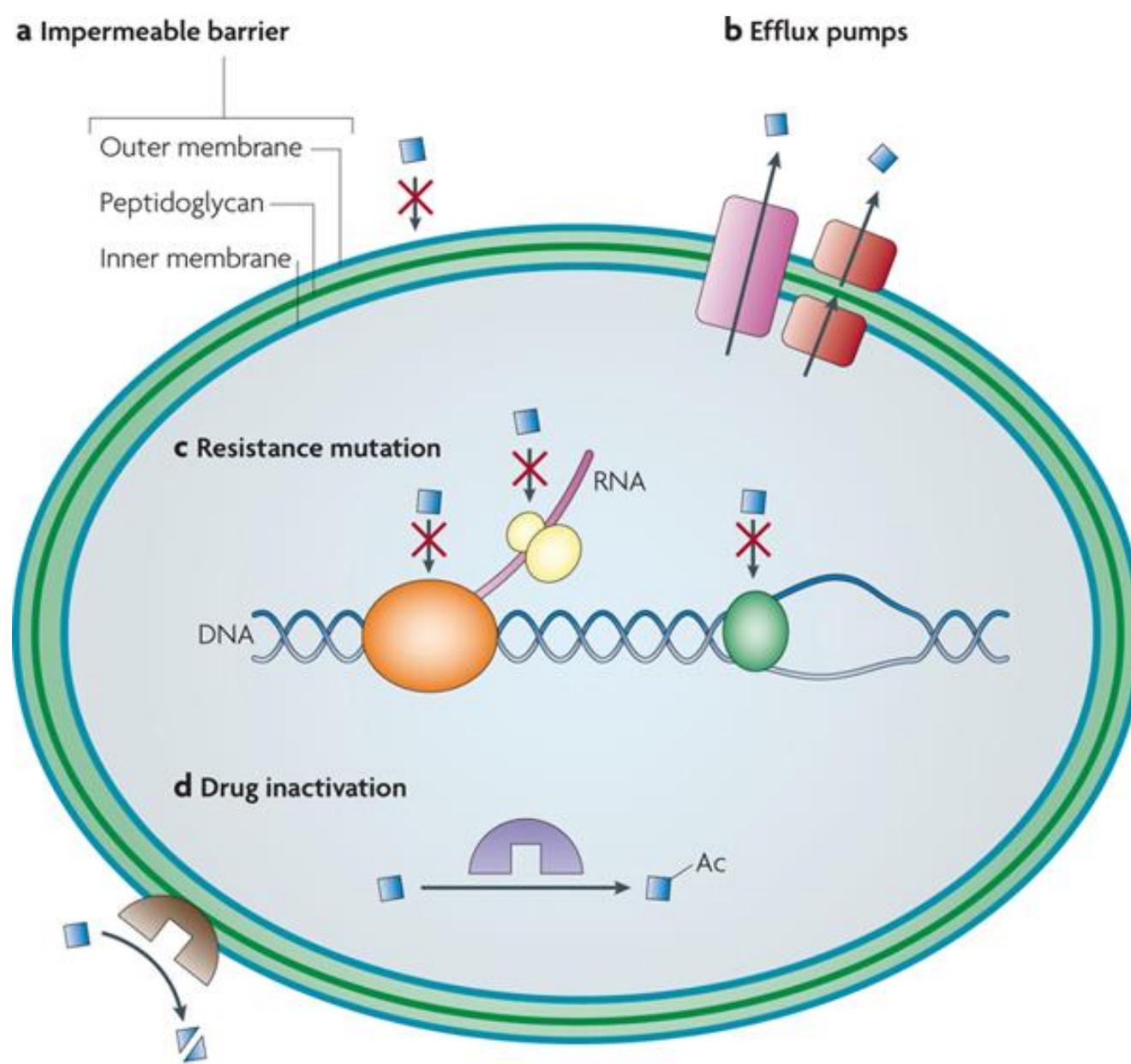
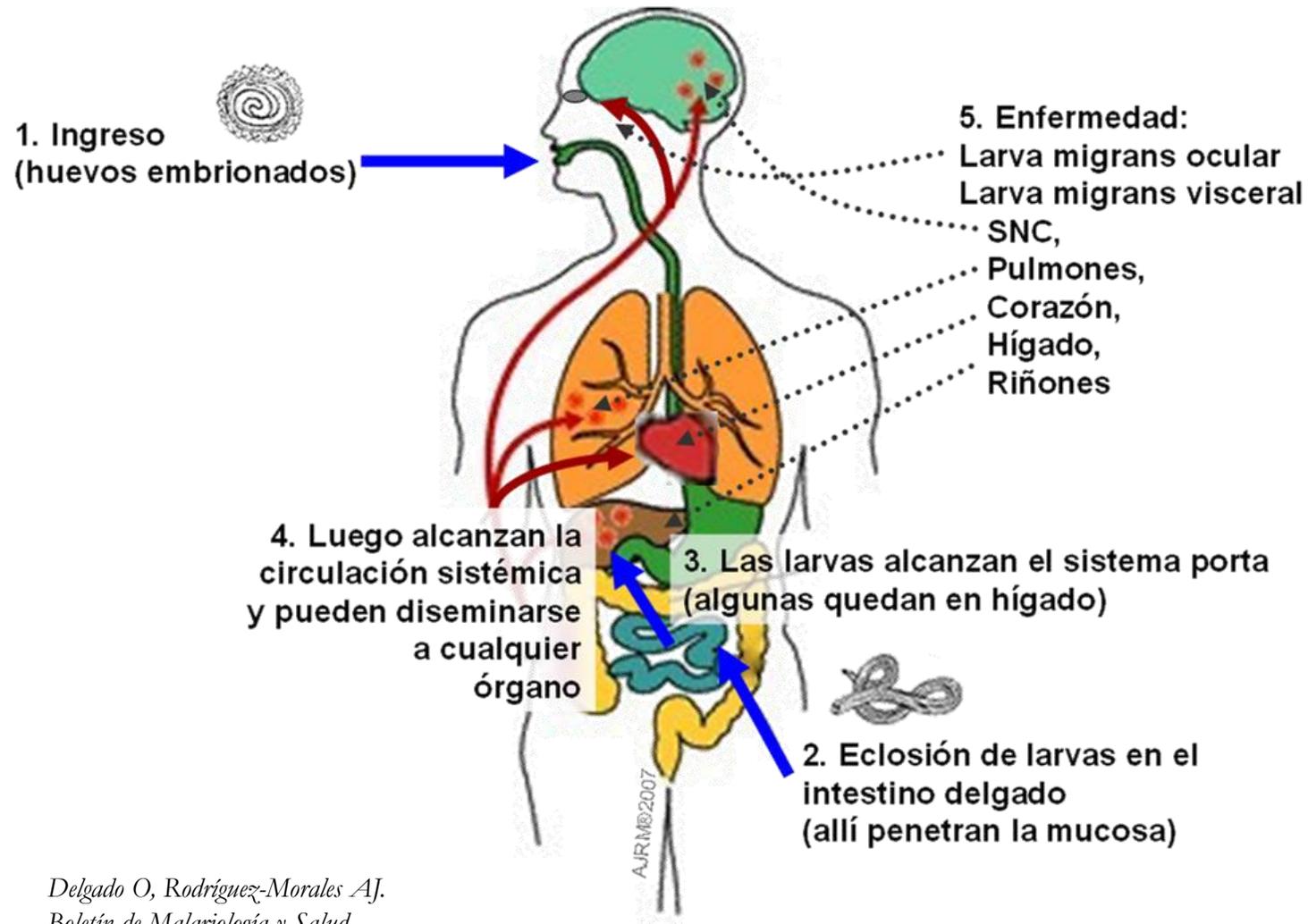


FIG 2 *Cryptosporidium* virulence factors described to date and their contribution to the parasite life cycle.



Ciclo de *Toxocara spp.* en el Hombre



Delgado O, Rodríguez-Morales AJ.
Boletín de Malariología y Salud
Ambiental 2009 Ene/Jul; 49(1):1-
33.

(Delgado & Rodríguez-Morales, 2008)

Factores del Ambiente

- Biológicas (vectores y reservorios)
- Físicas (Humedad, Temperatura)
- Sociales (organización, costumbres, cultura)

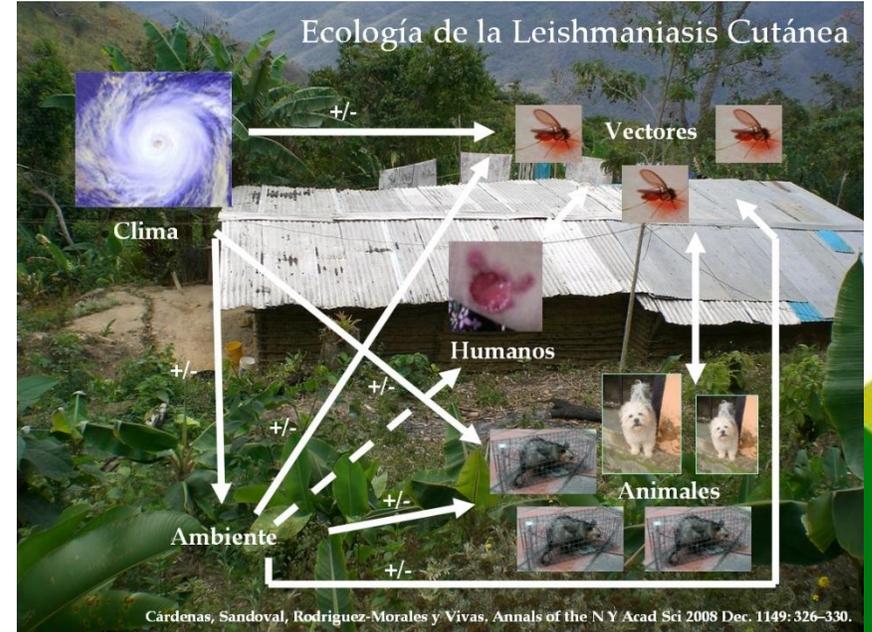
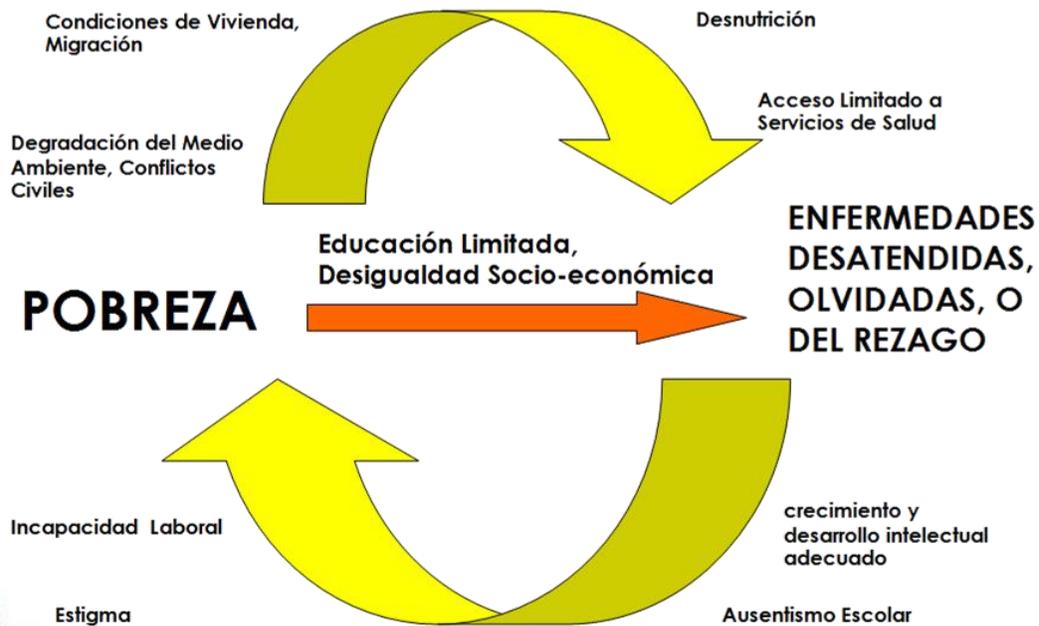


Figura 1. Ciclo de Destitución entre Pobreza y Salud. Franco Paredes C, Woodworth M, Rodríguez Morales AJ. Las Enfermedades Desatendidas en Latino América: Un Circulo Vicioso entre Pobreza y Salud. Acta Científica Estudiantil 2007 Oct-Dic;5(4):173-177. (Special Issue **Global Theme Issue on Poverty and Human Development**)

Determinantes Sociais de las Enfermedades Infecciosas en Latinoamérica

Figura 1 - Determinantes sociais: modelo de Dahlgren e Whitehead



Determinantes Sociales de las Enfermedades Infecciosas en Latinoamérica

Figure 1.1 A Model of the Determinants of Health

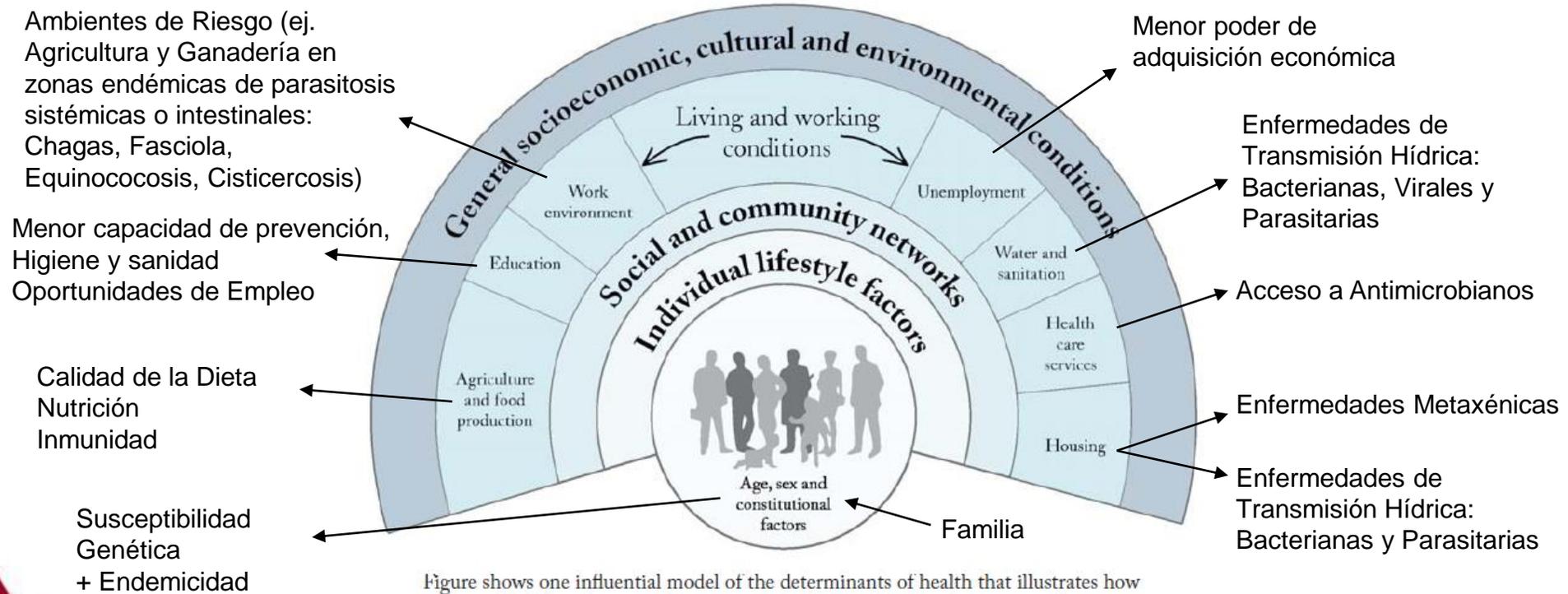
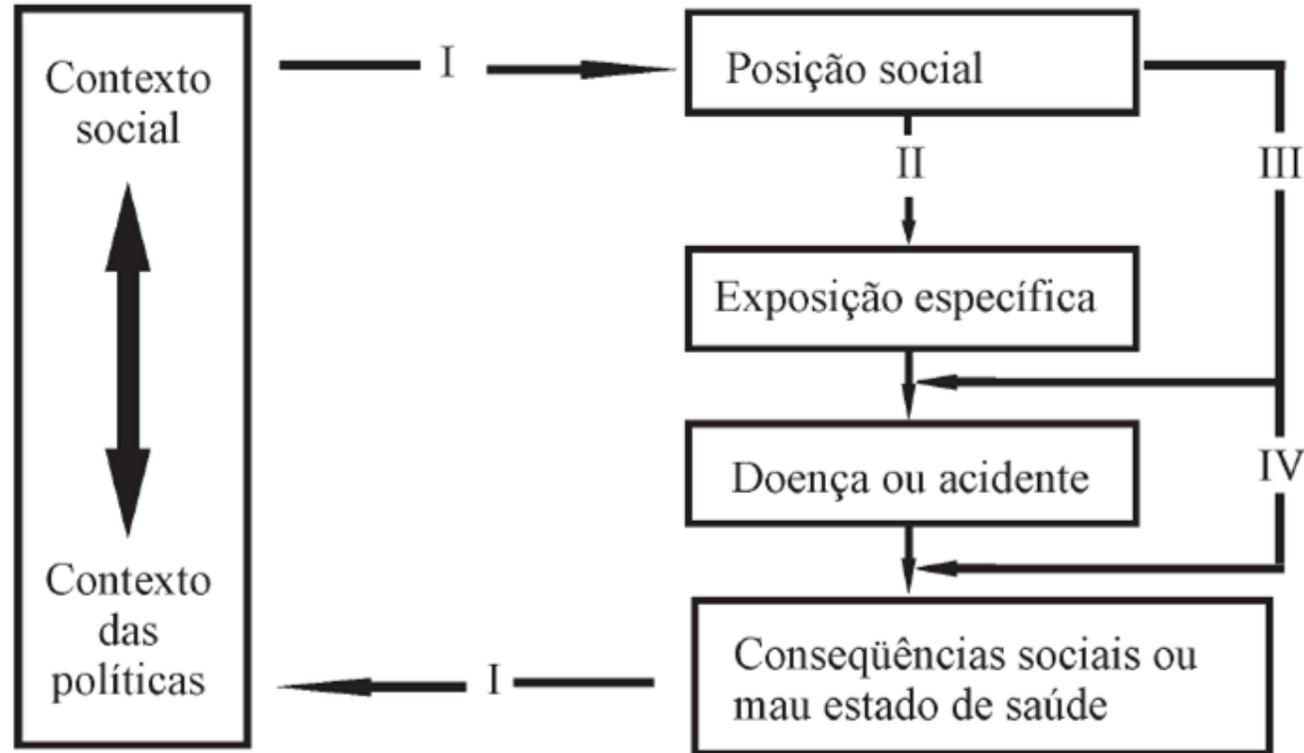


Figure shows one influential model of the determinants of health that illustrates how various health-influencing factors are embedded within broader aspects of society.

Source: Dahlgren, G. and Whitehead, M. (1991). Policies and Strategies to Promote Social Equity in Health. Stockholm: Institute for Futures Studies.

Determinantes Sociais de las Enfermedades Infecciosas en Latinoamérica

Figura 2 - Determinantes sociais: modelo de Diderichsen e Hallqvist



Determinantes Sociales de las Enfermedades Infecciosas en Latinoamérica

Figure 2.1 Social Determinants of Health and the Pathways to Health and Illness

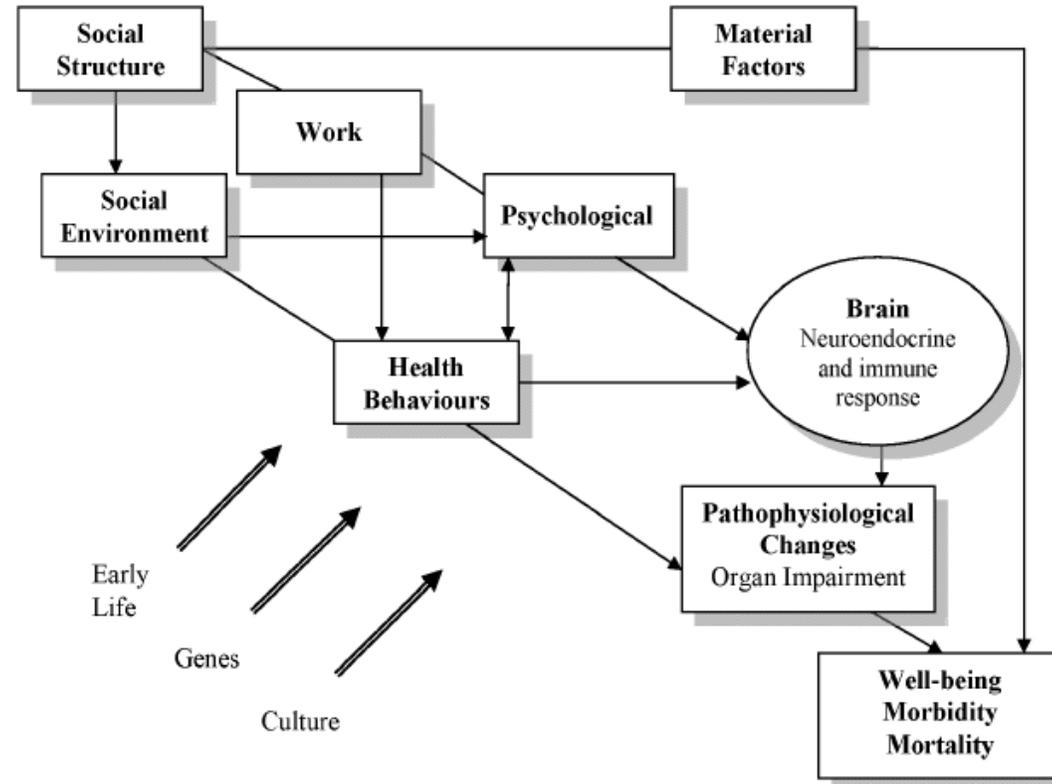
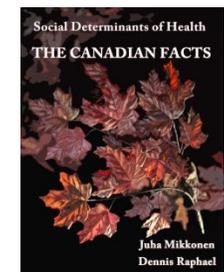


Figure shows how the organization of society influences the living and working conditions we experience that then go on to shape health. These processes operate through material, psychosocial, and behavioural pathways. At all stages of life, genetics, early life, and cultural factors are also strong influences upon health.

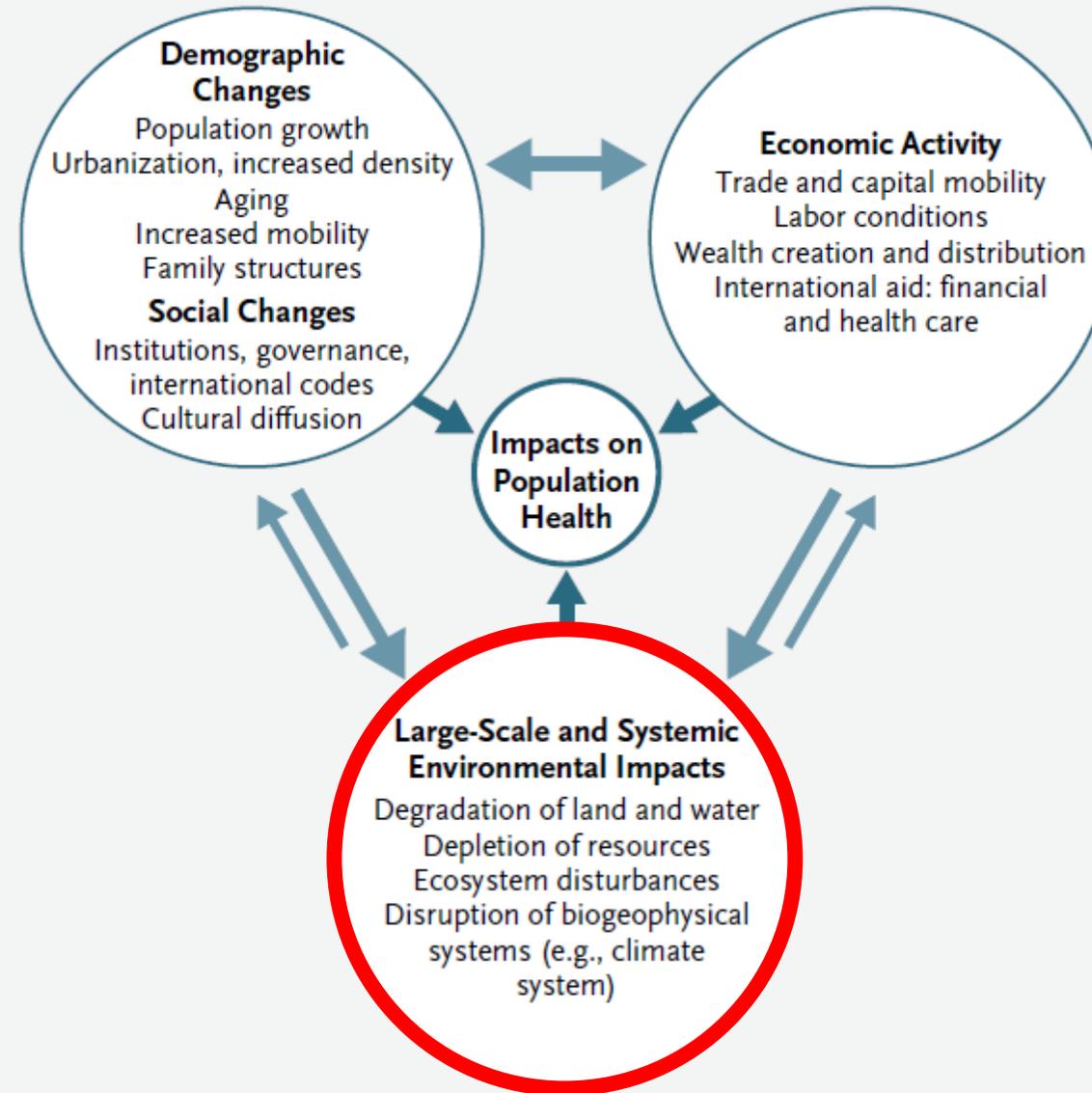
Source: Brunner, E., & Marmot, M. G. (2006). 'Social Organization, Stress, and Health.' In M. G. Marmot & R. G. Wilkinson (Eds.), *Social Determinants of Health*. Oxford: Oxford University Press, Figure 2.2, p. 9.



by Educativos - CRIF

Globalization and Global Changes

Increases in interpopulation connectivity and increases in scale and intensity of action and impact



World Health Organization



World Meteorological Organization



Figure 1. Influence on Human Health of Changes Related to Globalization.

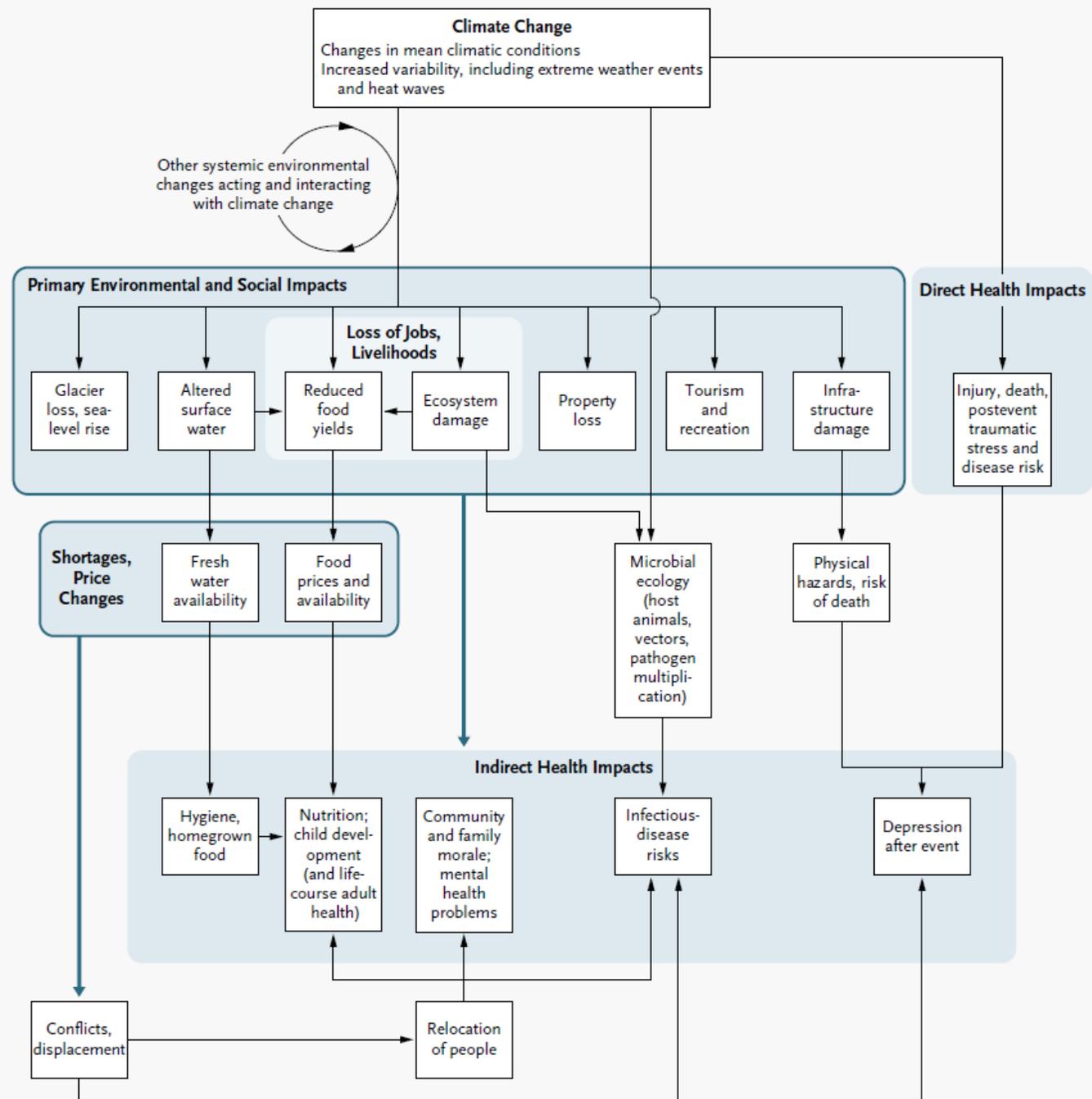


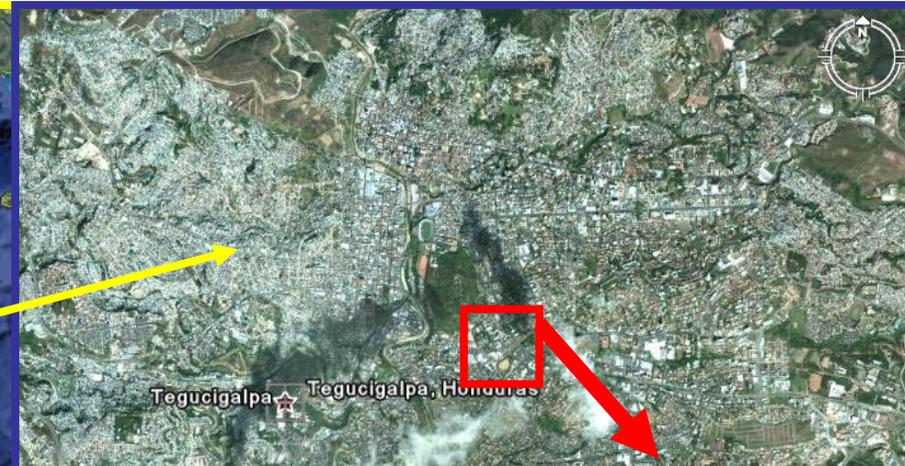
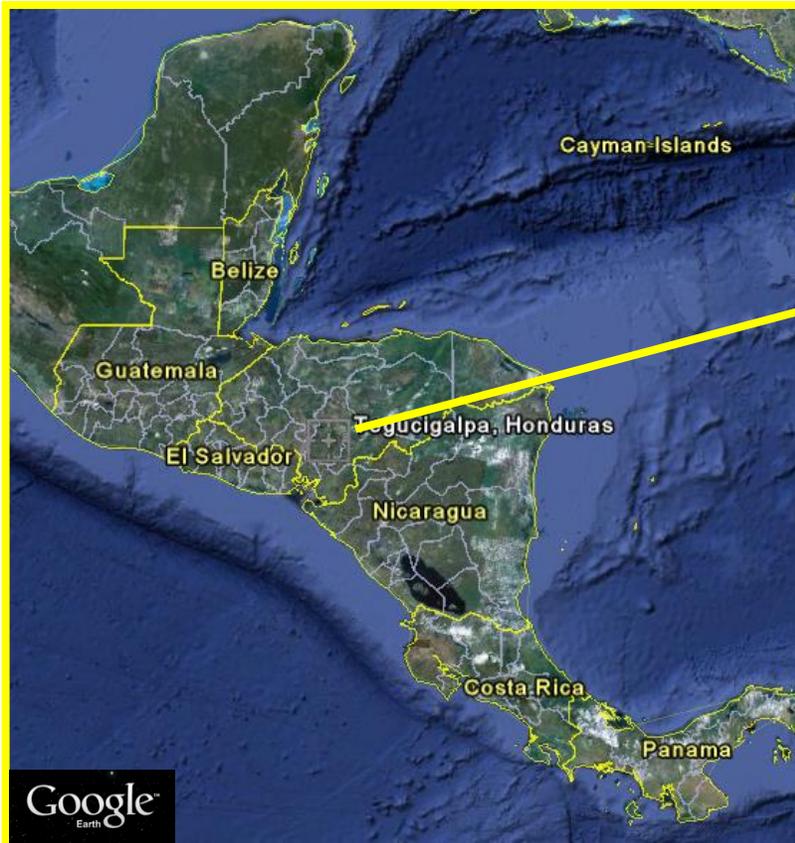
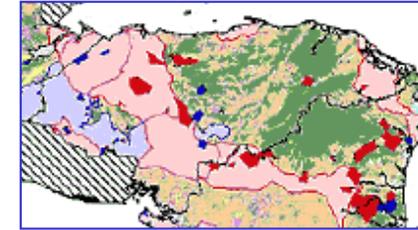
Figure 2. Processes and Pathways through Which Climate Change Influences Human Health.





Materiales y Métodos

- Área de estudio y recopilación de datos:
Hospital Escuela, Tegucigalpa, Honduras.

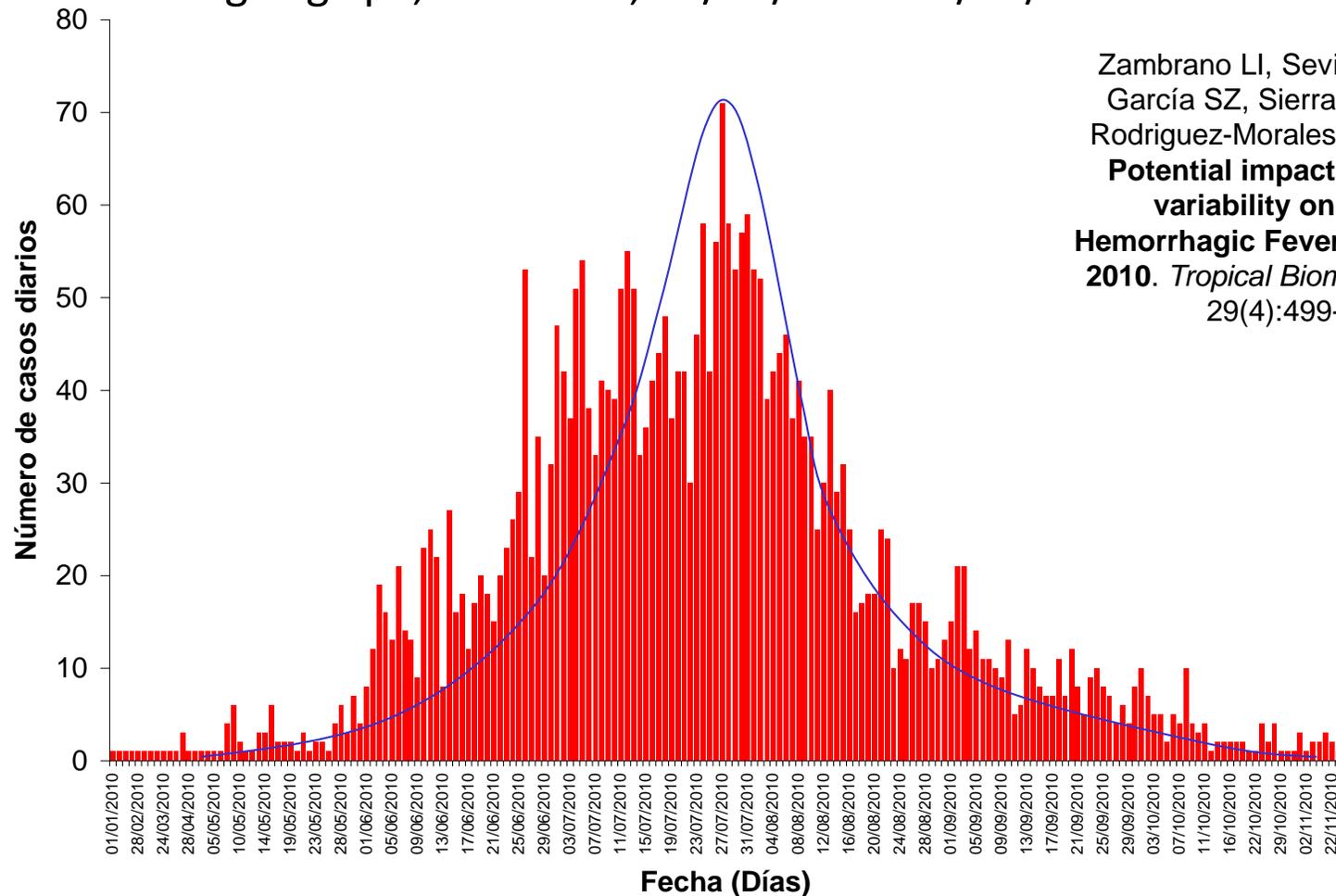




Resultados

Estadística descriptiva

Figura 1. Reporte diario de casos de Dengue Hemorrágico en el Hospital Escuela, Tegucigalpa, Honduras, 01/01/2010-31/12/2010.



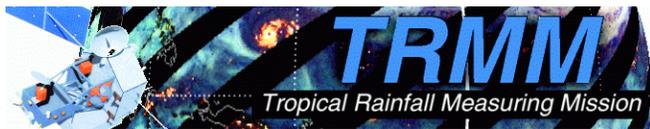
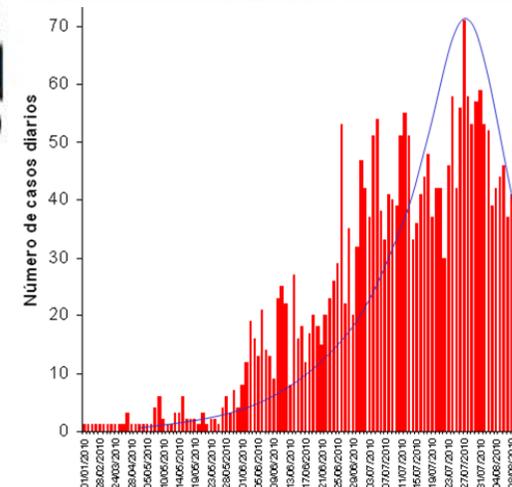
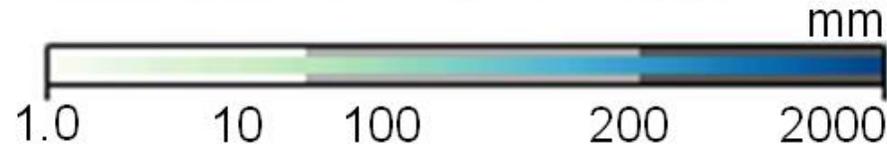
Zambrano LI, Sevilla C, Reyes-García SZ, Sierra M, Kafati R, Rodríguez-Morales AJ, Mattar S.
Potential impacts of climate variability on Dengue Hemorrhagic Fever in Honduras, 2010. *Tropical Biomedicine* 2012; 29(4):499-507.



Resultados

Estadística descriptiva

Figura 2. Mapas de patrones de lluvia obtenidos del satélite TRMM para Honduras, durante Enero a Agosto 2010 (NEO/NASA).



Zambrano LI, Sevilla C, Reyes-García SZ, Sierra M, Kafati R, Rodríguez-Morales AJ, Mattar S. Potential impacts of climate variability on Dengue Hemorrhagic Fever in Honduras, 2010.

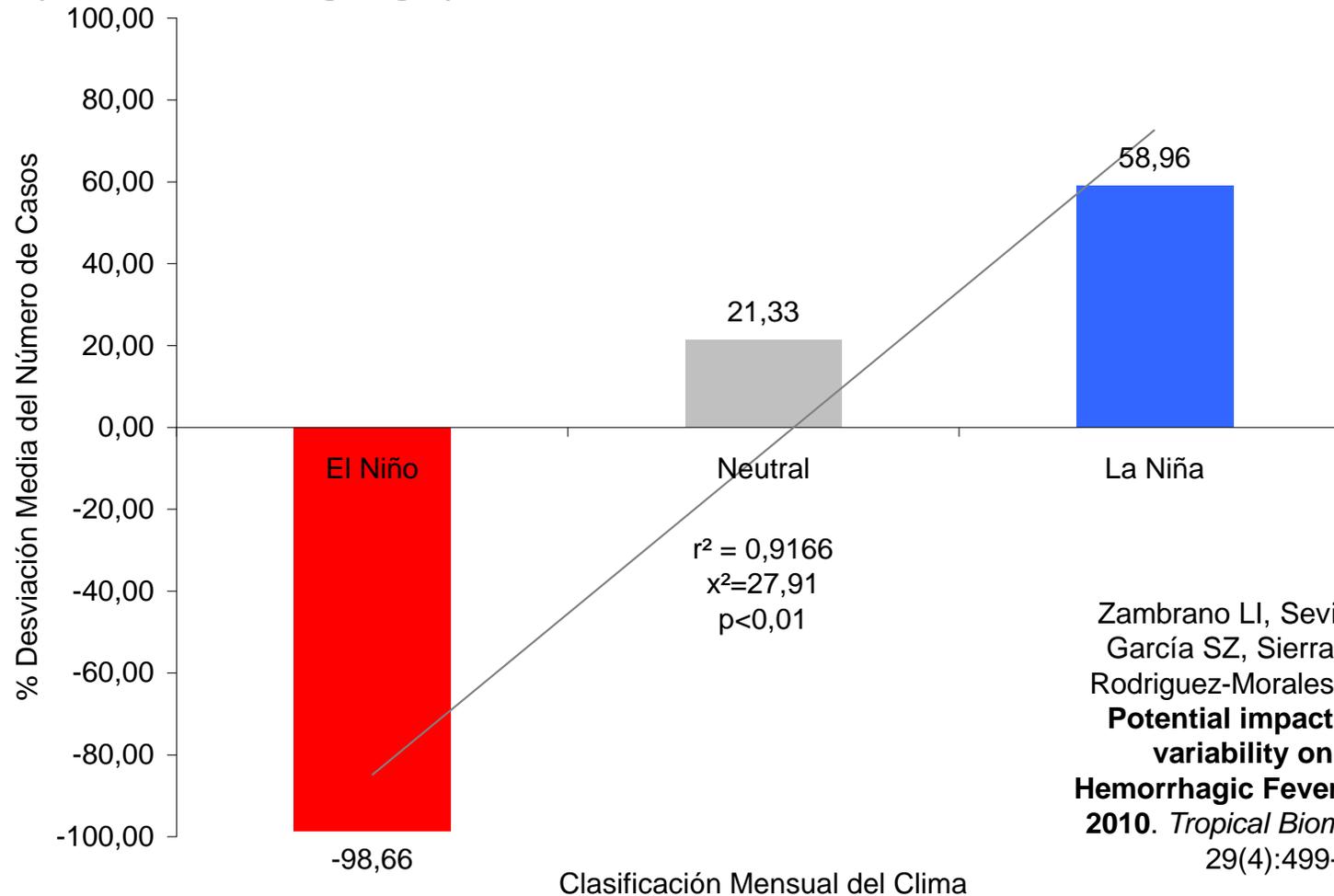
Tropical Biomedicine 2012; 29(4):499-507.



Resultados

Análisis bivariado

Figura 3. Diferencia en incidencia media de los casos por Dengue, durante los meses de El Niño y La Niña, Hospital Escuela, Tegucigalpa, Honduras, Enero-Diciembre 2010.



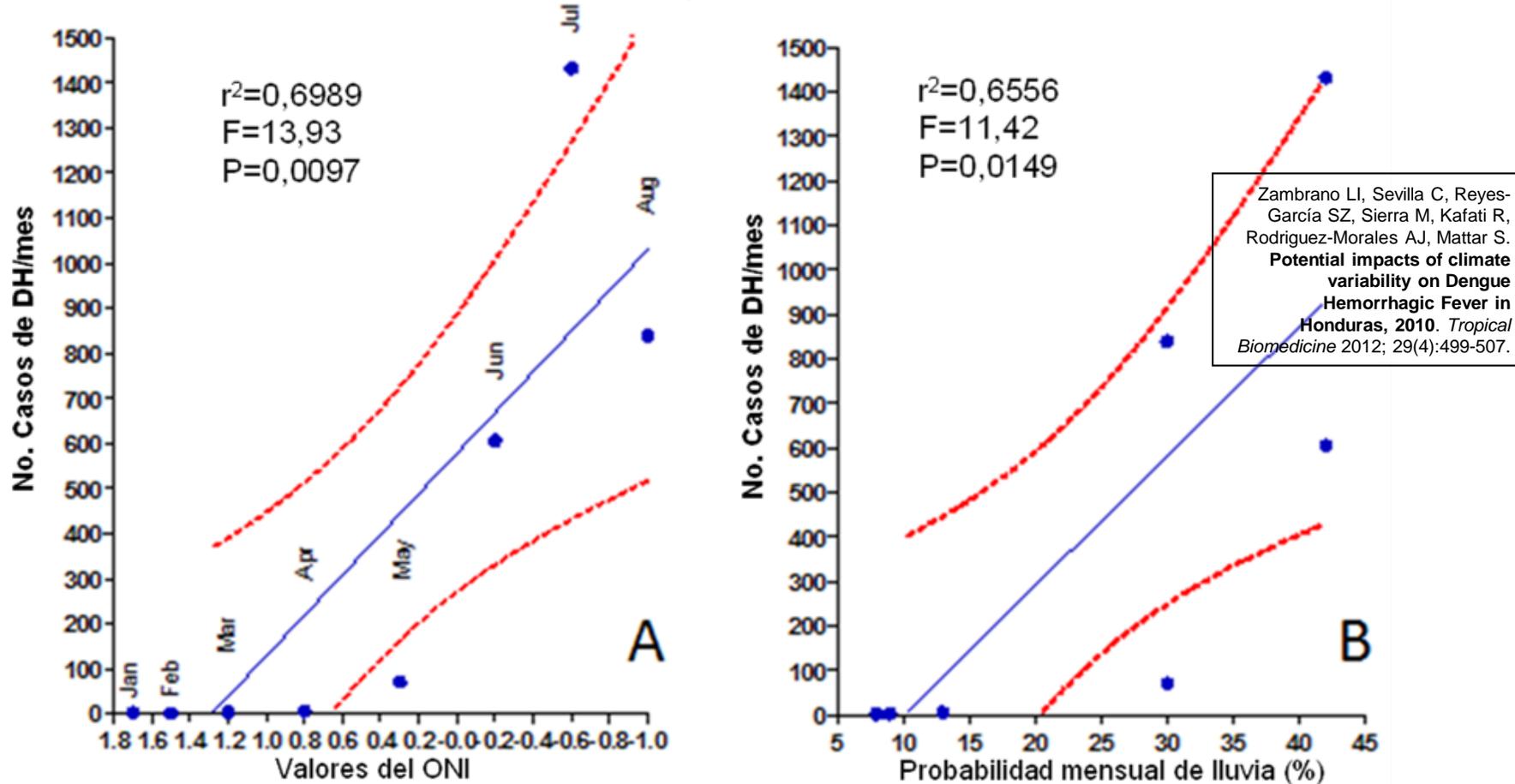
Zambrano LI, Sevilla C, Reyes-García SZ, Sierra M, Kafati R, Rodríguez-Morales AJ, Mattar S.
Potential impacts of climate variability on Dengue Hemorrhagic Fever in Honduras, 2010. *Tropical Biomedicine* 2012; 29(4):499-507.



Resultados

Análisis bivariado

Figura 4. Relación entre las variables macro y microclimáticas y el registro mensual de casos de Dengue Hemorrágico en el Hospital Escuela, Tegucigalpa, Honduras, Enero-Agosto 2010.
A. Indicador macroclimático ONI. **B.** Indicador probabilidad mensual de lluvia (%).



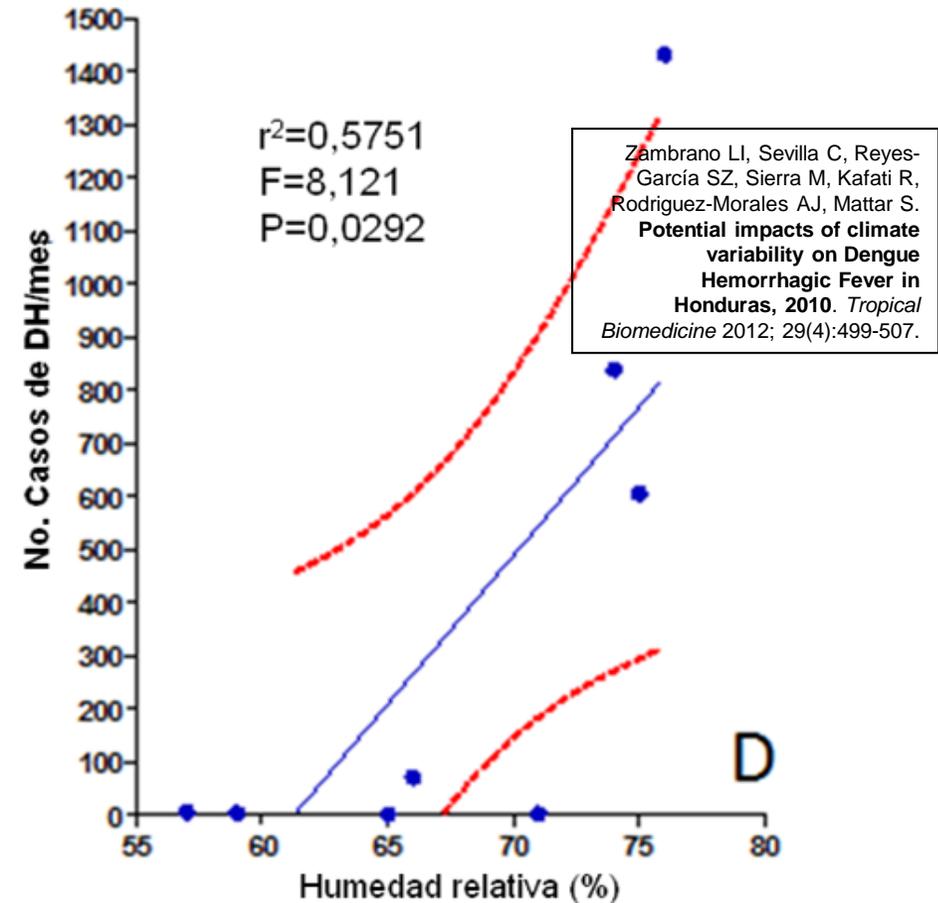
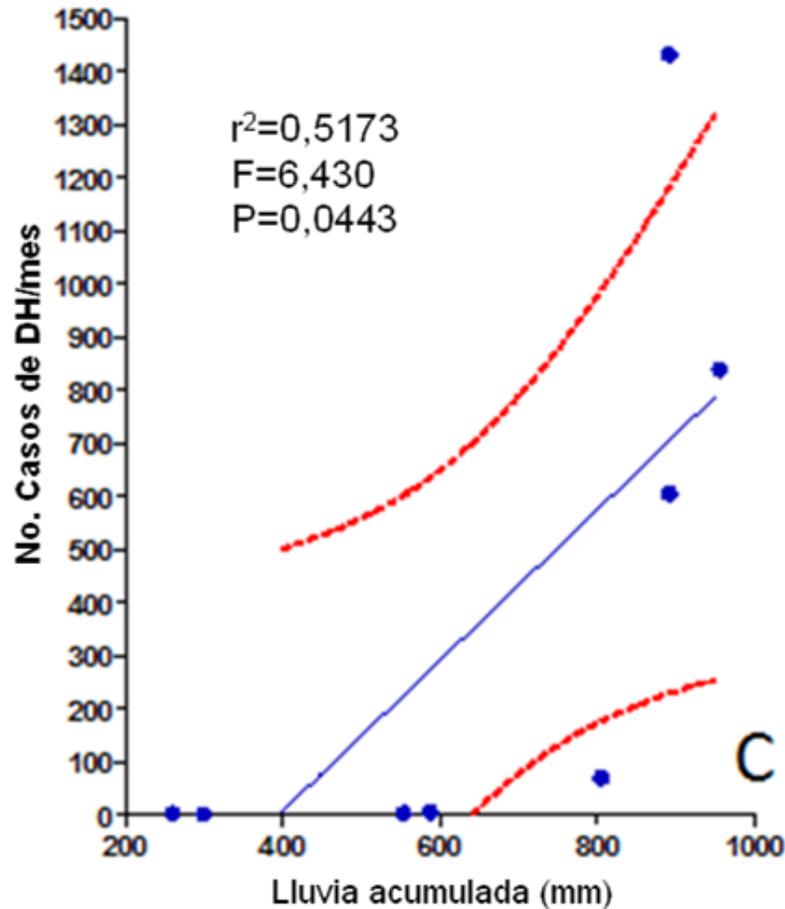


Resultados

Análisis bivariado

Figura 4. Relación entre las variables macro y microclimáticas y el registro mensual de casos de Dengue Hemorrágico en el Hospital Escuela, Tegucigalpa, Honduras, Enero-Agosto 2010.

C. Lluvia acumulada (mm). **D.** Humedad relativa (%).



Zambrano LI, Sevilla C, Reyes-García SZ, Sierra M, Kafati R, Rodríguez-Morales AJ, Mattar S. Potential impacts of climate variability on Dengue Hemorrhagic Fever in Honduras, 2010. *Tropical Biomedicine* 2012; 29(4):499-507.

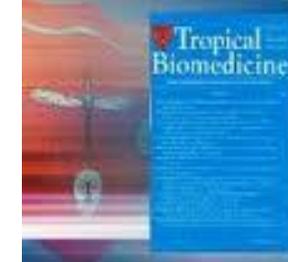


Resultados

Análisis multivariado

Cuadro 1. Resumen del modelo de regresión lineal múltiple para predicción de casos mensuales de DH, usando humedad relativa, lluvia acumulada, probabilidad de lluvia y valores de ONI.

		Estadísticos de cambio						
R	r ²	r ² ajustado	Error estándar	Cambio del r ²	Cambio F	gl1	gl2	p
0,836	0,699	0,649	319,11	0,699	13,929	1	6	0,01
Valores del ONI	Coeficiente no estandarizado		Coeficiente estandarizado		t	p	IC 95% para β	
	β	Error estándar	β				Límite inferior	Límite superior
	-450,193	120,625	-0,836		-3,732	0,01	-745,351	-155,035



Potential impacts of climate variability on Dengue Hemorrhagic Fever in Honduras, 2010

Zambrano, L.I.¹, Sevilla, C.¹, Reyes-García, S.Z.¹, Sierra, M.², Kafati, R.³, Rodríguez-Morales, A.J.^{4,5,6,7,*} and Mattar, S.^{6,8}

¹Hospital Escuela, Faculty of Medical Sciences, Universidad Nacional Autónoma de Honduras (UNAH), Tegucigalpa, Honduras

²Scientific Research Unit, Faculty of Medical Sciences, Universidad Nacional Autónoma de Honduras (UNAH), Tegucigalpa, Honduras

³Department of Epidemiology, Hospital Escuela, Secretary of Public Health, Tegucigalpa, Honduras

⁴Research Group Infection and Immunity, Faculty of Health Sciences, Universidad Tecnológica de Pereira (UTP), Pereira, Risaralda, Colombia

⁵Office of Scientific Research, Cooperativa de Entidades de Salud de Risaralda (COODESURIS), Pereira, Risaralda, Colombia

⁶Committee on Zoonoses and Hemorrhagic Fevers of the Colombian Association of Infectious Diseases (Asociación Colombiana de Infectología, ACIN), Bogotá, Colombia

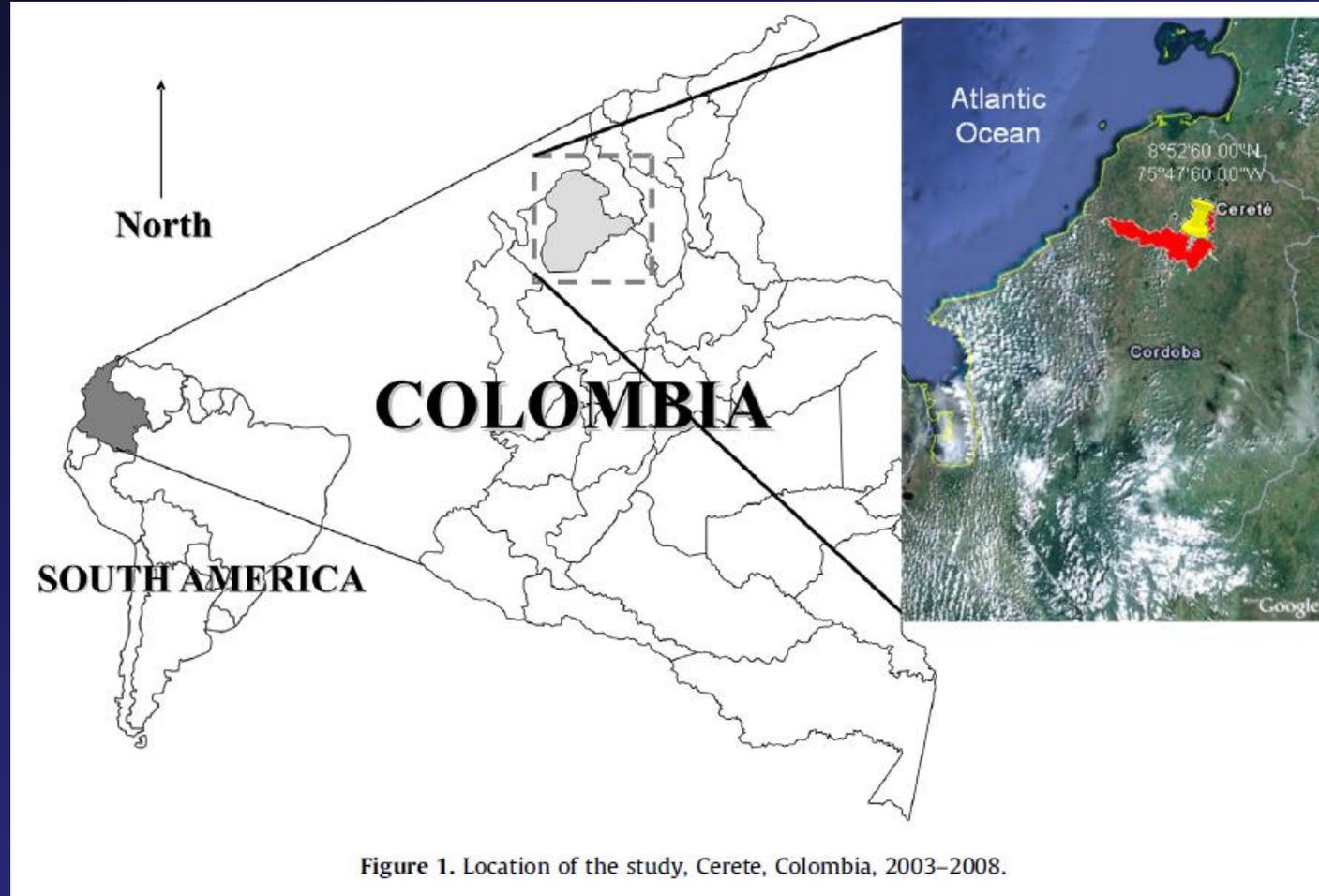
⁷Instituto Experimental José Witremundo Torrealba, Núcleo Universitario Rafael Rangel, Universidad de Los Andes, Trujillo, Venezuela

⁸Instituto de Investigaciones Biológicas del Trópico, Universidad de Córdoba, Montería, Córdoba, Colombia

*Corresponding author email: arodriguezm@utp.edu.co

Received 21 March 2012; received in revised form 30 July 2012; accepted 31 July 2012

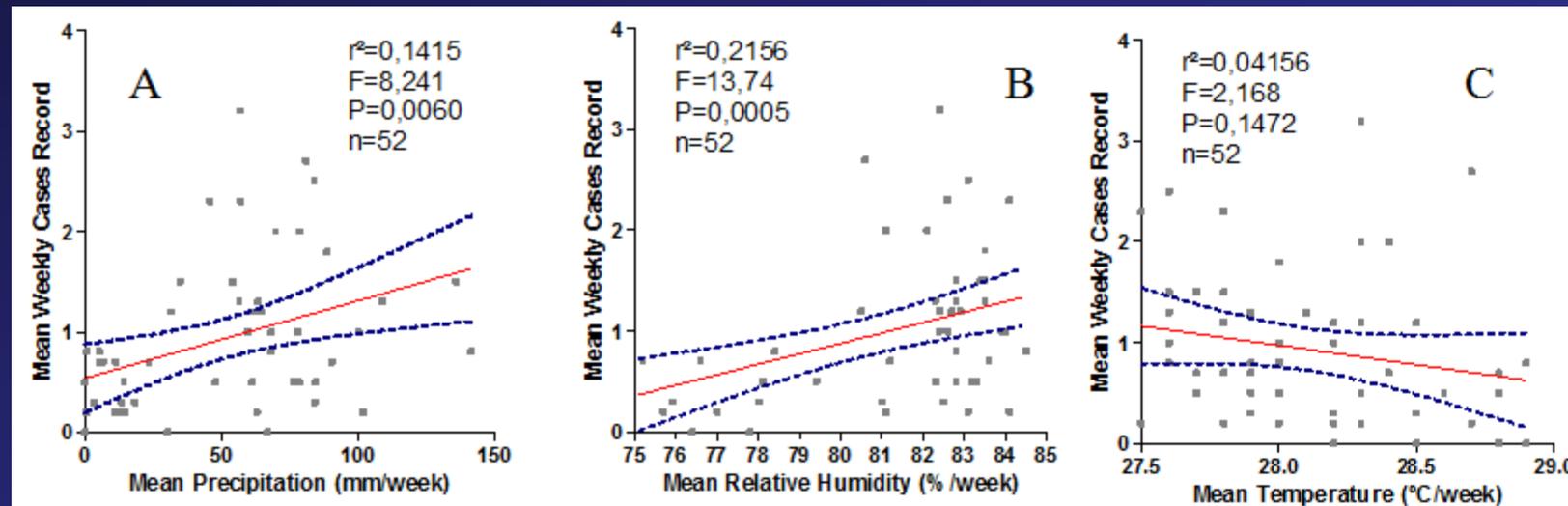
Dengue en Cerete



Salim Mattar, Victor Morales, Alexander Cassab, Alfonso J. Rodríguez-Morales. Effect of Climate Variables on Dengue Incidence in a Tropical Caribbean Municipality of Colombia, Cereté, 2003-2008. IJID 2013 May;17(5):e358-9.

Dengue en Cerete

Figure 1. Relations between climate variables and the morbidity due dengue in Cerete, Colombia, 2003-2008. A. Linear regression between precipitations and mean dengue weekly cases. B. Linear regression between relative humidity and mean dengue weekly cases. C. Linear regression between temperature and mean dengue weekly cases.

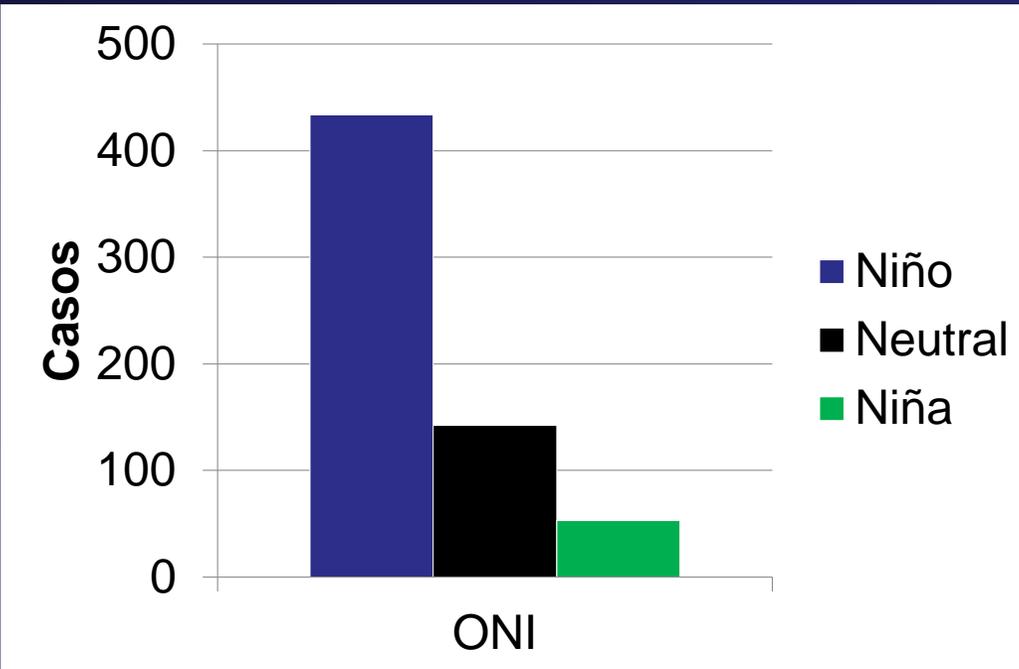




3	P242	Potential impacts of climate change and variability on dengue epidemiology in Risaralda, Colombia, 2010-2011	Alfonso	J.	Rodriguez-Morales
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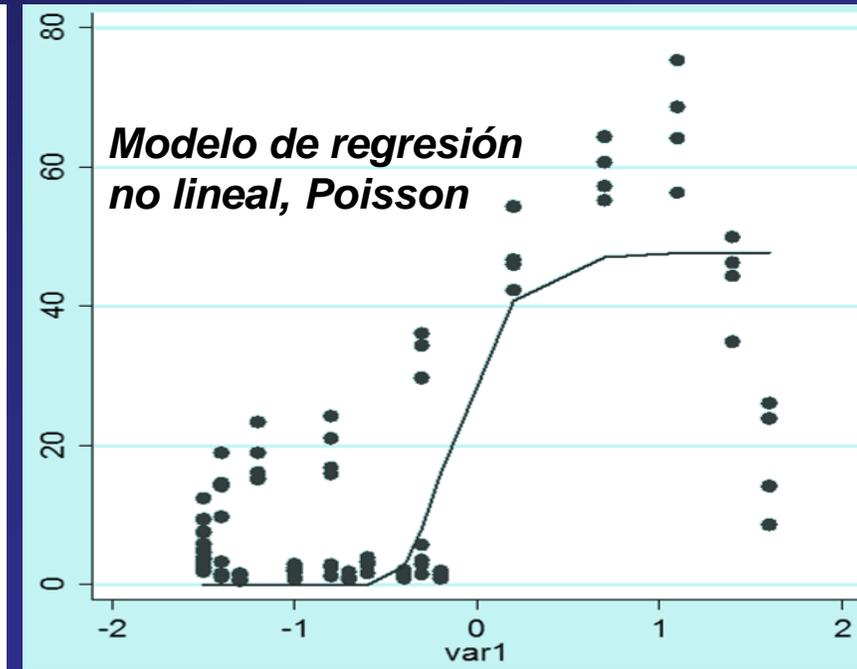
A. J. Rodríguez-Morales^{1,2,*}, J. W. Martínez¹, A. C. Herrera-Giraldo^{1,3}, S. Botero³, S. Bernal-Gutiérrez¹, E. V. Cárdenas-Giraldo¹, E. A. Guerrero-Matituy¹, A. H. Molina-Delgado¹, C. P. Montoya-Arias¹, L. L. Quintero-Herrera¹, V. Ramírez-Jaramillo¹, J. A. Rico-Gallego¹

¹Faculty of Health Sciences, Universidad Tecnológica de Pereira, ²Office of Scientific Research, Cooperativa de Entidades de Salud de Risaralda (COODESURIS), ³Secretary of Health of Risaralda, Government of Risaralda, Pereira, Colombia



ANOVA F=66,59, P<0,001

Bonferroni en todas las comparaciones p<0,01



ONI (coef 0,329; 95%CL 0,209-0,450)

pluviometría (coef 0,003; 95CI 0,002-0,004)

ajustado por año y semana (p<0,001, pseudo r²=0,6913).

Zoonoses and Climate Variability

The Example of Leishmaniasis in Southern Departments of Colombia

**Rocio Cardenas,^{a,b} Claudia M. Sandoval,^{b,c}
Alfonso J. Rodriguez-Morales,^b and Paul Vivas^d**

^aInstituto Departamental de Salud de Norte de Santander, Cucuta, Colombia

*^bInstituto Experimental Jose Witremundo Torrealba, Universidad de Los Andes,
Trujillo, Venezuela*

*^cGrupo de Investigación en Enfermedades Parasitarias, Tropicales e Infecciosas, Instituto
de Investigación en Ciencias Biomédicas, Universidad de Pamplona, Pamplona,
Norte de Santander, Colombia*

*^dHospital Regional de Especialidades No. 1 Licenciado Ignacio Garcia Tellez, Instituto
Mexicano del Seguro Social, Merida, México*

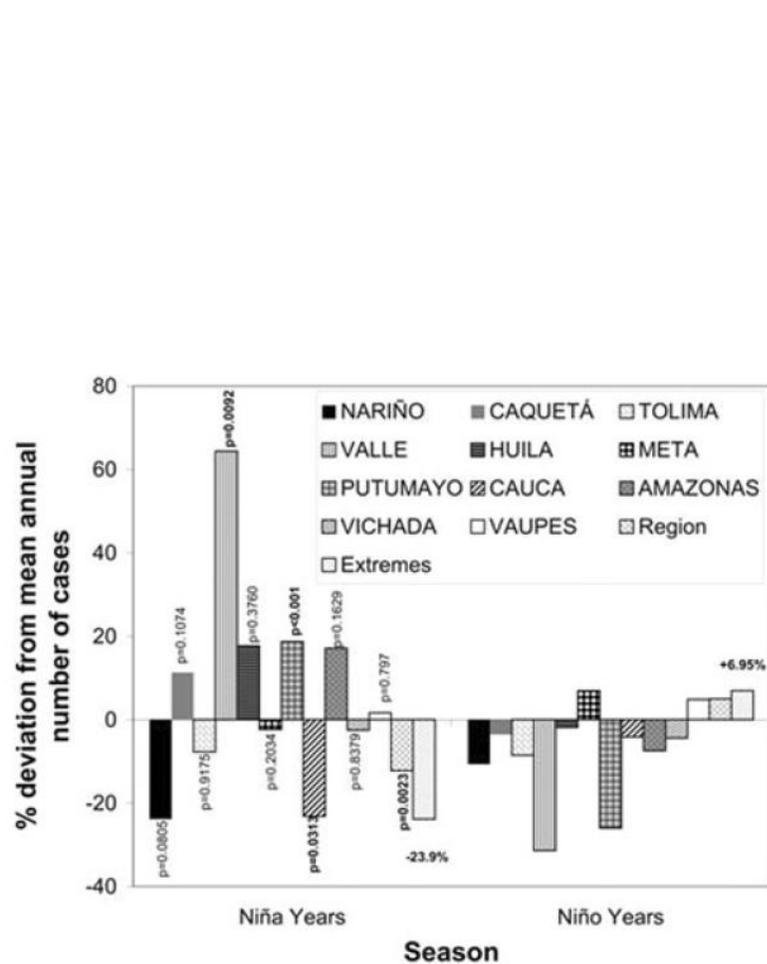
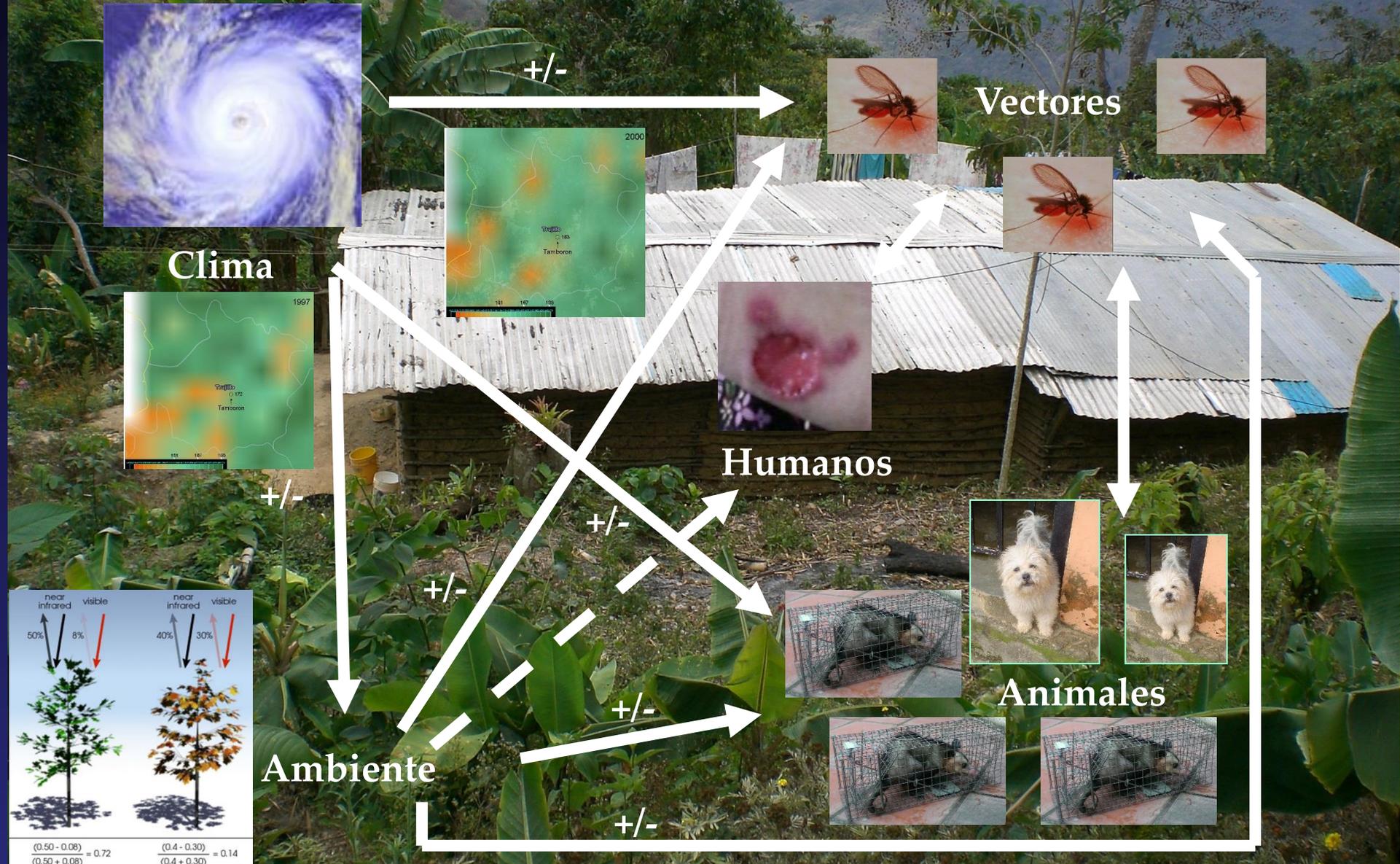


Figure 3. Comparisons of the effects of El Niño Southern Oscillation seasons on the incidence of leishmaniasis in southern departments of Colombia, 1985–2002.



Figure 1. Map of Colombia with the relative position and the departments included in this study.

Ecología de la Leishmaniasis Cutánea



CL Vectors and Climate

- Main findings of an entomological survey in Norte de Santander, Colombia
 - During the study period (2006-07) **5,079** sandflies were collected, *Lu. spinicrassa* represented 95.2% of them.
 - The climatic period corresponded to a dry season of El Niño (highest Oscillation Niño Index in the last 2006 trimester, 1.2 SST).
 - In general, the MCA evidenced a significant inverse relation between *L. spinicrassa* abundance and the
 - **relative humidity ($p < 0.05$)**, as well also with
 - **the rainfall ($p < 0.05$)**,
 - but not for the average temperature ($p > 0.05$).



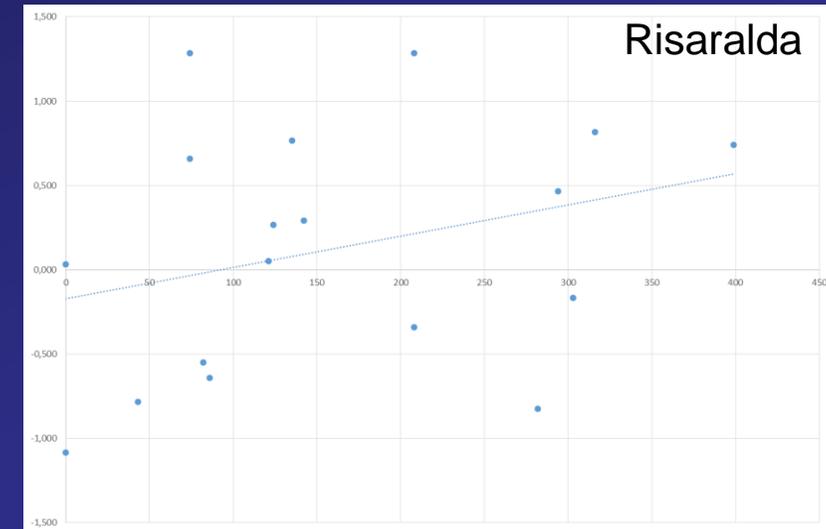
Potential impacts of climate change and variability on cutaneous leishmaniasis epidemiology in Risaralda and Magdalena, Colombia, 1985-2002

A. J. Rodriguez-Morales^{1, 2, 3*}, A. C. Herrera-Giraldo^{1,4}, S. Botero⁴, J. C. Dib⁵

¹Faculty of Health Sciences, Universidad Tecnológica de Pereira, ²Office of Scientific Research, Cooperativa de Entidades de Salud de Risaralda

(COODESURIS), ³Candidato a Doctor en Parasitología, Postgrado Nacional de Parasitología, Universidad Central de Venezuela, Caracas, Venezuela,

⁴Secretary of Health of Risaralda, Government of Risaralda, Pereira, Colombia, ⁵Center of Research in Tropical Diseases, Universidad Cooperativa de Colombia, Santa Marta, Colombia.



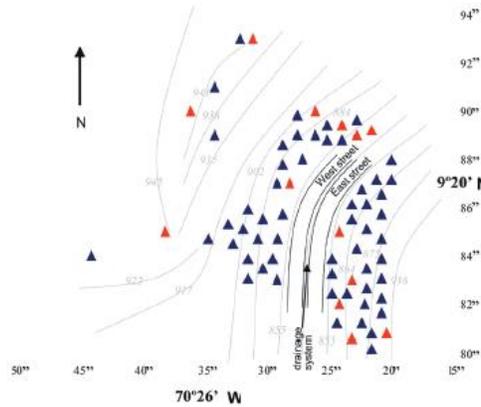
Regression model showed that ONI and SOI were significantly associated with cutaneous leishmaniasis incidence rates in Risaralda ($p=0.0002$, pseudo $r^2=0.0609$) but not in Magdalena ($p=0.2478$, pseudo $r^2=0.0283$).

Modelo de Elevación Digital de Terreno del Municipio Trujillo destacando la ubicación de Tamborón, Parroquia Monseñor Carrillo.

Figure 2.

Geographical distribution of houses, with negative MST dogs (represented in blue) and with positive MST dogs (represented in red) (numbers represent altitude levels).

Répartition géographique des maisons avec les chiens négatifs au MST (bleu) et les chiens positifs au MST (rouge) (les nombres correspondent à l'altitude).



Cardenas R et al.
Bull Soc Pathol Exot, 2006, 99, 5, 355-358



Image © 2007 DigitalGlobe
Image © 2007 TerraMetrics

© 2007 Europa Technologies

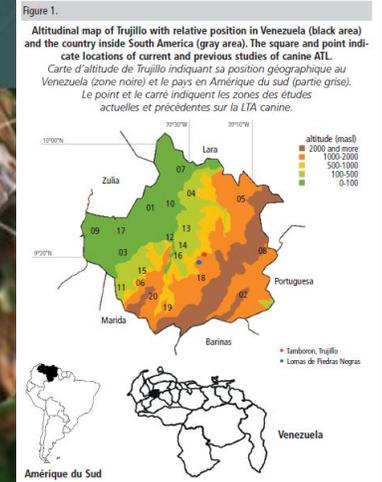


Photo 2a.
Female boxer of 1 year-old with suspected lesions of ATL in genitals.
Femelle boxer d'un an avec des lésions suspectes de LTA sur les organes génitaux.



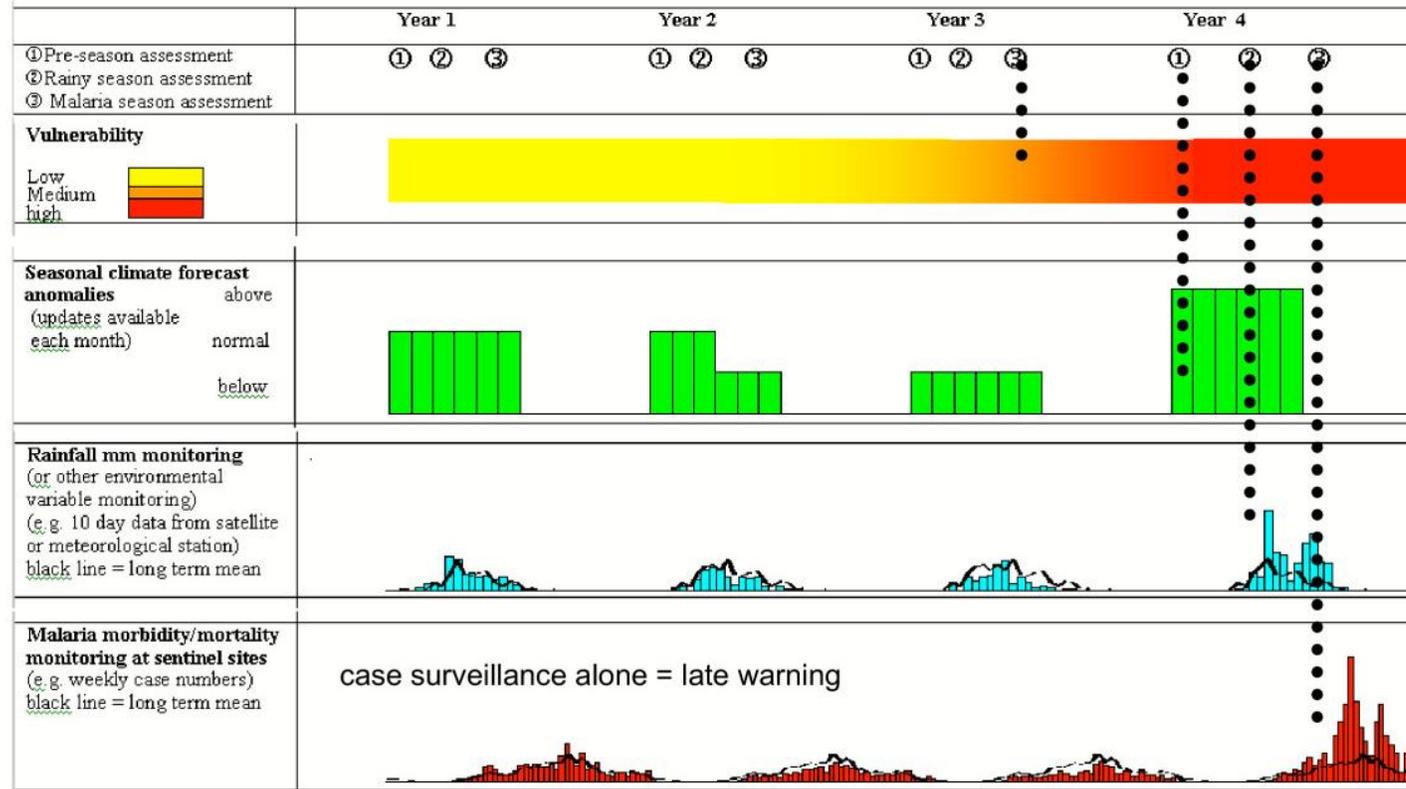
Photo 2b.
Female boxer of 1 year-old with suspected lesions of ATL in legs.
Femelle boxer d'un an avec des lésions suspectes de LTA sur les pattes.



Fuente: Modificado y analizado por A. J. Rodriguez Morales a partir de imágenes satelitales obtenidas con el software Google Earth Plus, con la función de Modelo de Elevación Digital de Terreno (MDT).

Malaria Early Warning Systems: the rationale

Gathering cumulative evidence for early and focused response



Flag 1 – Flag 2 – Flag 3

Year 3 Pre-season assessment – vulnerability increasing due to period of drought.

Year 4 Pre-season assessment – vulnerability still increasing due to period of drought and seasonal forecast above normal – Flag 1.

Year 4 Rainy season assessment – vulnerability remains high, weather monitoring indicates higher than normal rainfall – Flag 2.

Year 4 Malaria season assessment – vulnerability remains high, rainfall higher than normal through much of season, malaria cases pass epidemic threshold – Flag 3.

Figure 1: The use of early warning indicators in the malaria control planning cycle (WHO, 2001a)

Epidemiología de la Enfermedad de Chagas



- **Reservorios**

- Prácticamente cualquier mamífero (incluso doméstico)





*Rural Barinas,
Venezuela, April 2007*

The socio-ecology of zoonotic infections

A. Cascio^{1,2}, M. Bosilkovski^{2,3}, A. J. Rodriguez-Morales^{2,4} and G. Pappas^{2,5}

1) Tropical and Parasitological Diseases Unit, Department of Human Pathology, University of Messina, Messina, Italy, 2) Working Group on Zoonoses, International Society of Chemotherapy, 3) University Clinic for Infectious Diseases and Febrile Conditions, Skopje, Former Yugoslav Republic of Macedonia, 4) Public Health Division, Department of Preventive and Social Medicine, Razetti Medical School; Faculty of Medicine, Central University of Venezuela (UCV), Caracas, Venezuela and 5) Institute of Continuing Medical Education of Ioannina, Ioannina, Greece

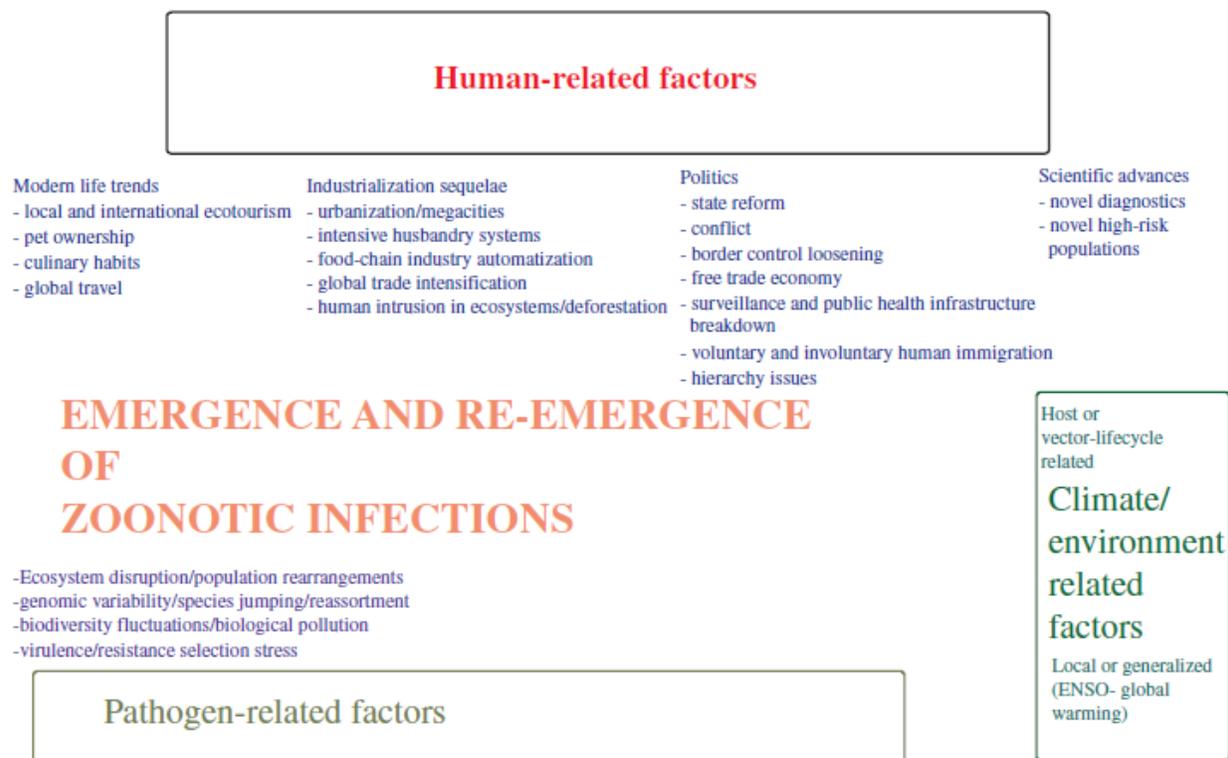


FIG. 1. Factors influencing the resurgence of zoonotic infections and their interplay. ENSO, El Niño southern oscillation.

Transmisión Vectorial

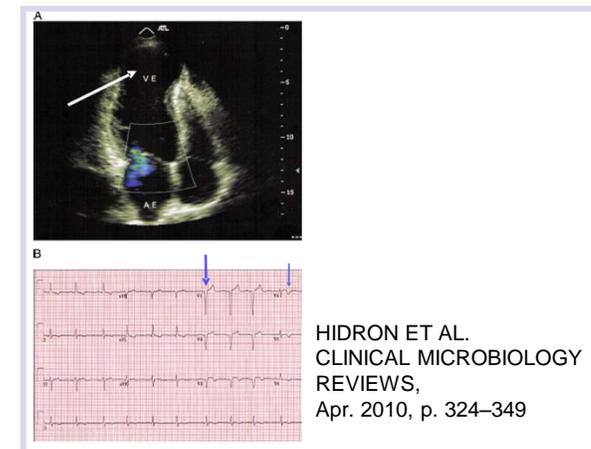
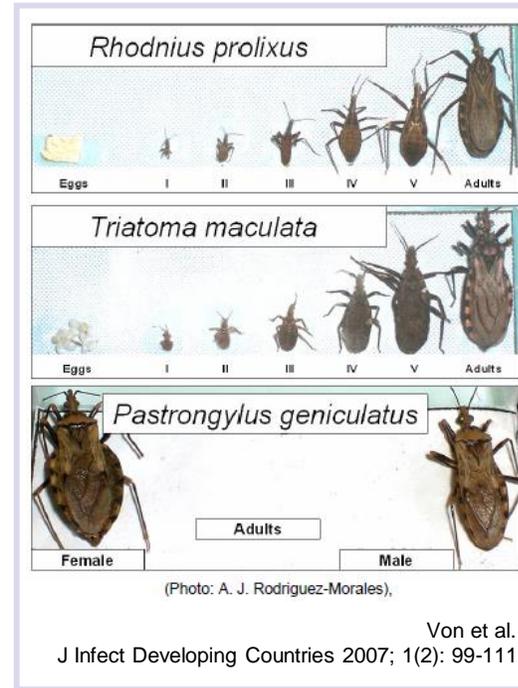
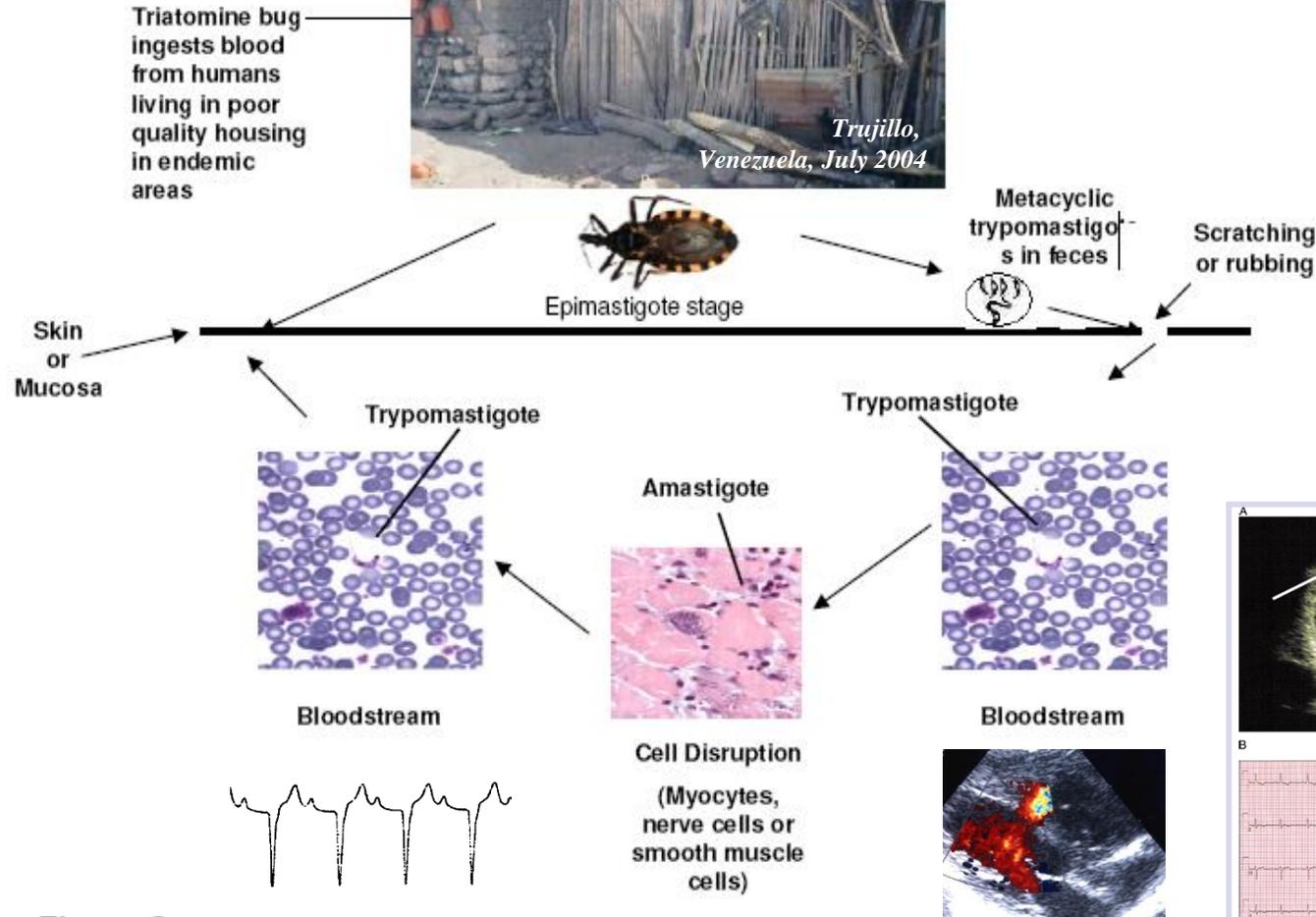


Figure 2

Title Cycle of transmission of *Trypanosoma cruzi* and its vectors to humans. Triatomine bugs live in the crevices of poorly constructed houses in impoverished areas in Latin America. Metacyclic trypomastigote is the infecting form to humans, while the amastigote is the intracellular form responsible for the immunopathogenesis in target human organs.

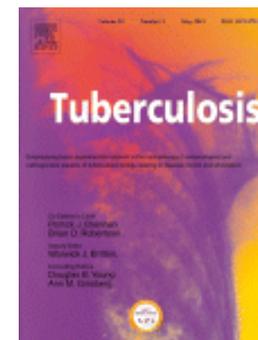
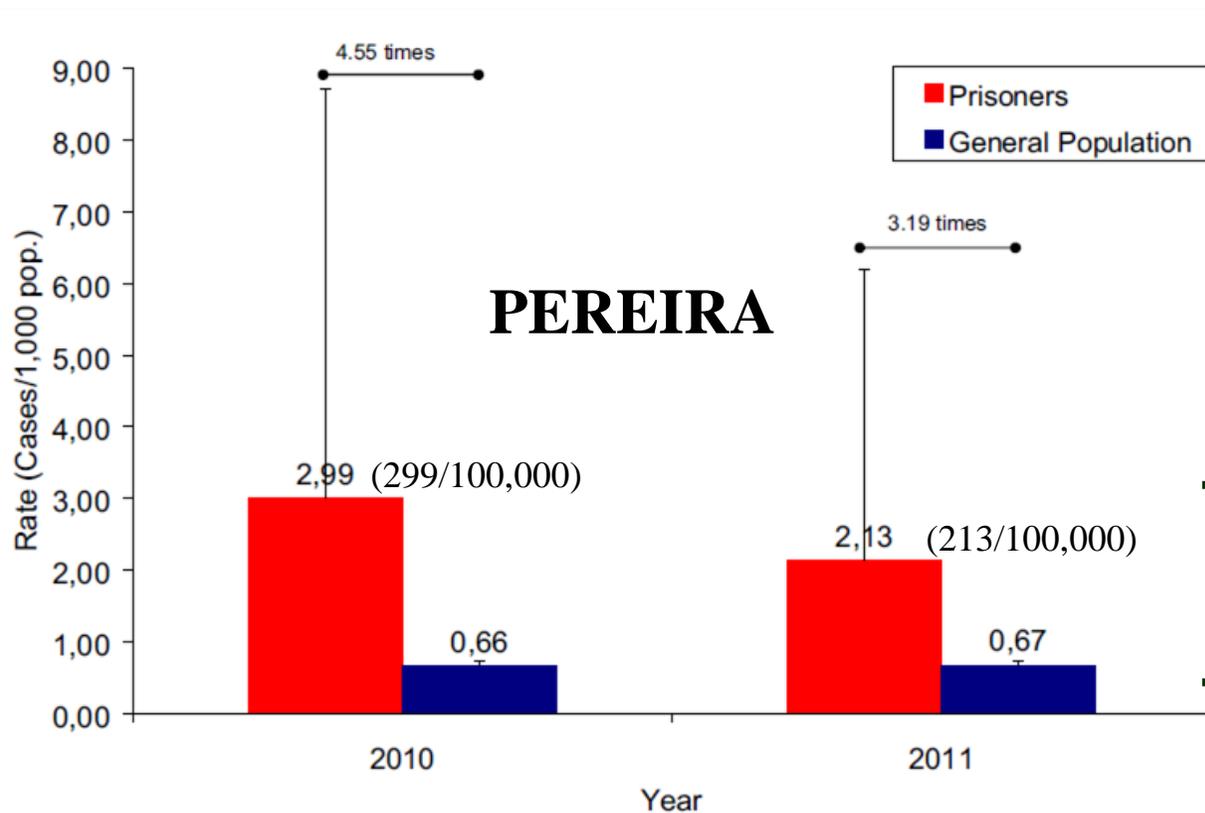
Para que la cadena de infección genere algún impacto en la salud del huésped es importante que estén presentes tres factores:

1. La presencia del agente infeccioso en cantidades suficientes para afectar al individuo o individuos.
2. Un modo de transmisión adecuado de este patógeno al huésped susceptible
3. Un huésped o grupo de huéspedes susceptibles que se expongan al agente patógeno.

En la cadena de infección pueden suceder cinco circunstancias que conllevan a la aparición de brotes o epidemias. Estas circunstancias son las siguientes:

1. Cuando un nuevo grupo de individuos susceptibles se introduce o incorpora en un medio en el que una enfermedad es endémica.
2. Cuando una nueva fuente de infección se introduce en una zona donde el agente infeccioso ha estado ausente y muchos o todos los individuos son susceptibles, como por el ejemplo la presencia de visitantes en un país extranjero, desplazamientos demográficos o contaminación de los alimentos por un agente poco común.
3. Cuando la cadena de infección se hace más eficaz como resultado de cambios en la vida social, de comportamiento, de hábitos, prácticas sexuales, o culturales. El hacinamiento en una prisión o la exposición a una nueva puerta de entrada son ejemplos.

Epidemiología de la TB en las Cárceles



las tasas de incidencia de TB en población carcelaria pueden ser de 3 a 50 veces mayores que en población general

Figure 1. Comparison of smear positive TB morbidity rate among prisoners and general population of Pereira, Risaralda, Colombia (2010–2011).

En la cadena de infección pueden suceder cinco circunstancias que conllevan a la aparición de brotes o epidemias. Estas circunstancias son las siguientes:

5. Un aumento de la susceptibilidad a la infección o la enfermedad o de ambos a través de la inmunosupresión u otros factores que influyen en la respuesta del huésped como los trastornos nutricionales, el tratamiento con fármacos inmunosupresores, o la presencia de una enfermedad crónica. El efecto devastador del VIH sobre el sistema inmunológico resultó en una epidemia mundial de enormes proporciones.
6. Un aumento en la virulencia, el incremento del inóculo de un agente microbiano.

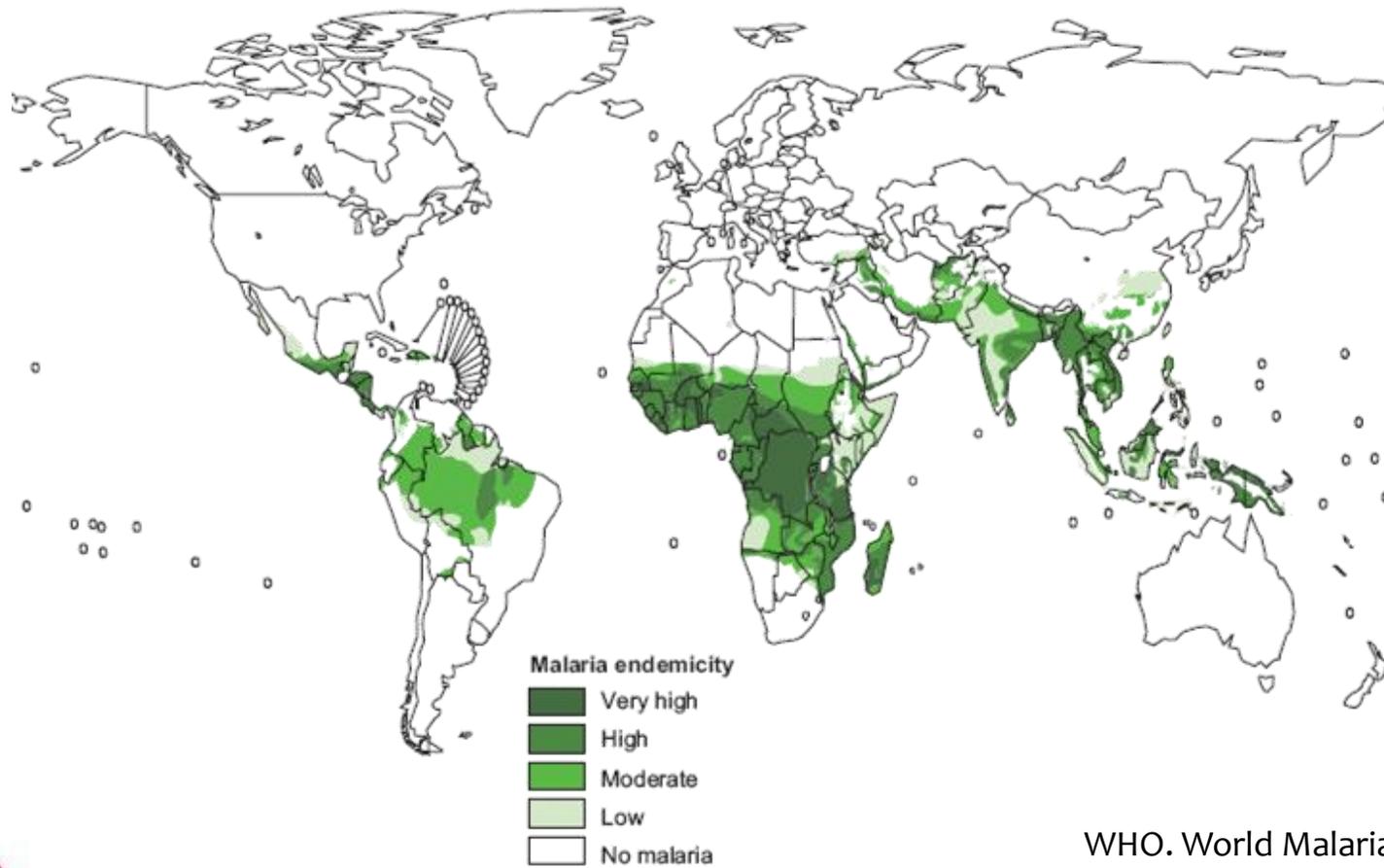
VIH/SIDA, Tuberculosis y Malaria



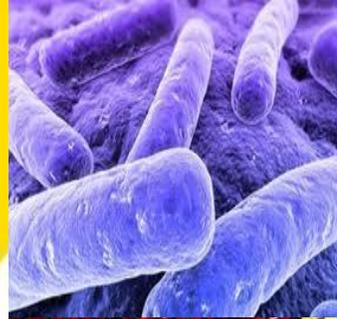
U.S. GLOBAL HEALTH POLICY

An online gateway for the latest data and information on the U.S. role in global health.

Malaria



WHO. World Malaria Report, 2005.

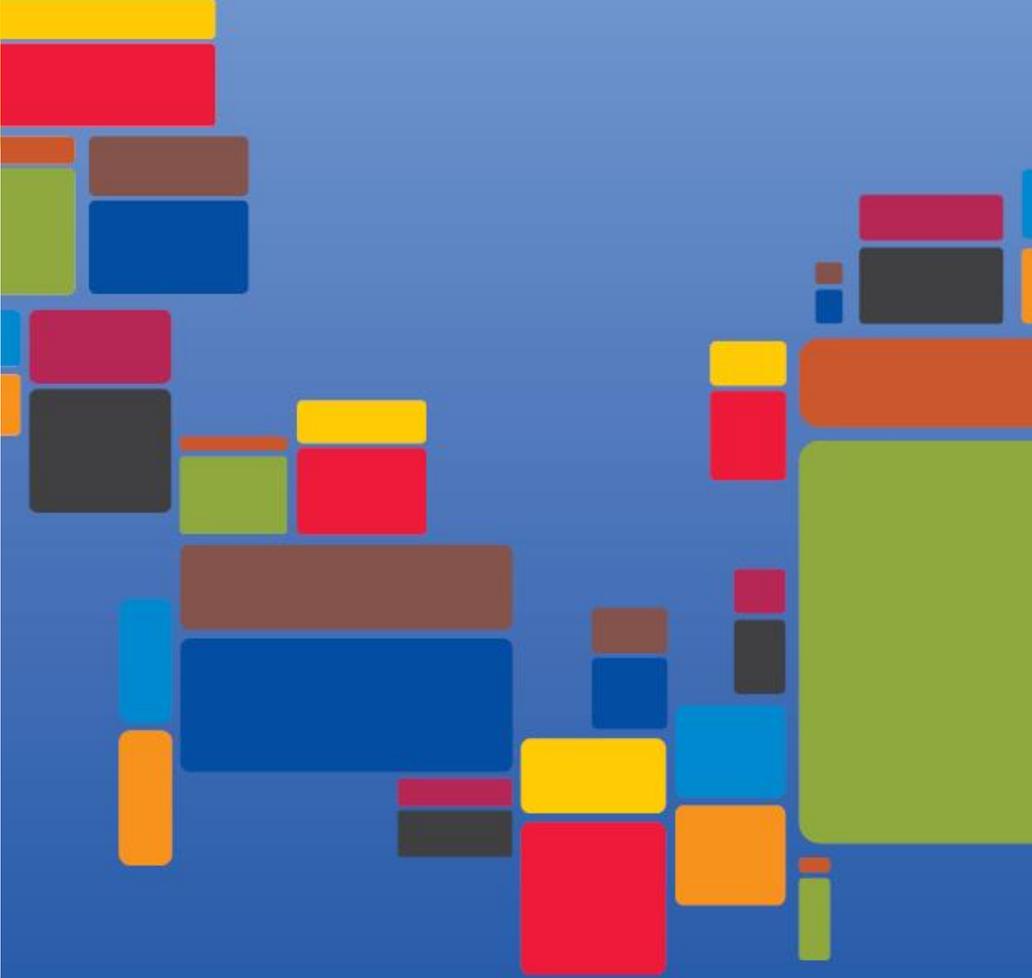


Mortalidad por Tuberculosis Por Regiones del Mundo, OMS 2010



Region	Population	Mortality (deaths)			Mortality rates (deaths/ 100,000pop)		
		Best	Low	High	Best	Low	High
Global burden	6,869,573,000	1,100,000	920,000	1,200,000	16.0	13.4	17.5
WHO African Region	836,970,000	250,000	220,000	280,000	29.9	26.3	33.5
WHO South-East Asia Region	1,807,594,000	500,000	370,000	640,000	27.7	20.5	35.4
WHO Eastern Mediterranean Region	596,747,000	95,000	74,000	120,000	15.9	12.4	20.1
WHO Western Pacific Region	1,798,335,000	130,000	120,000	150,000	7.2	6.7	8.3
WHO European Region	896,480,000	61,000	48,000	75,000	6.8	5.4	8.4
WHO Region of the Americas	933,447,000	20,000	17,000	23,000	2.1	1.8	2.5

Table 3. Estimated epidemiological burden of mortality due to TB, according regions by the WHO in 2010 (World Health Organization 2011) (excluding HIV positive deaths). Mortality rates were calculated for this chapter.

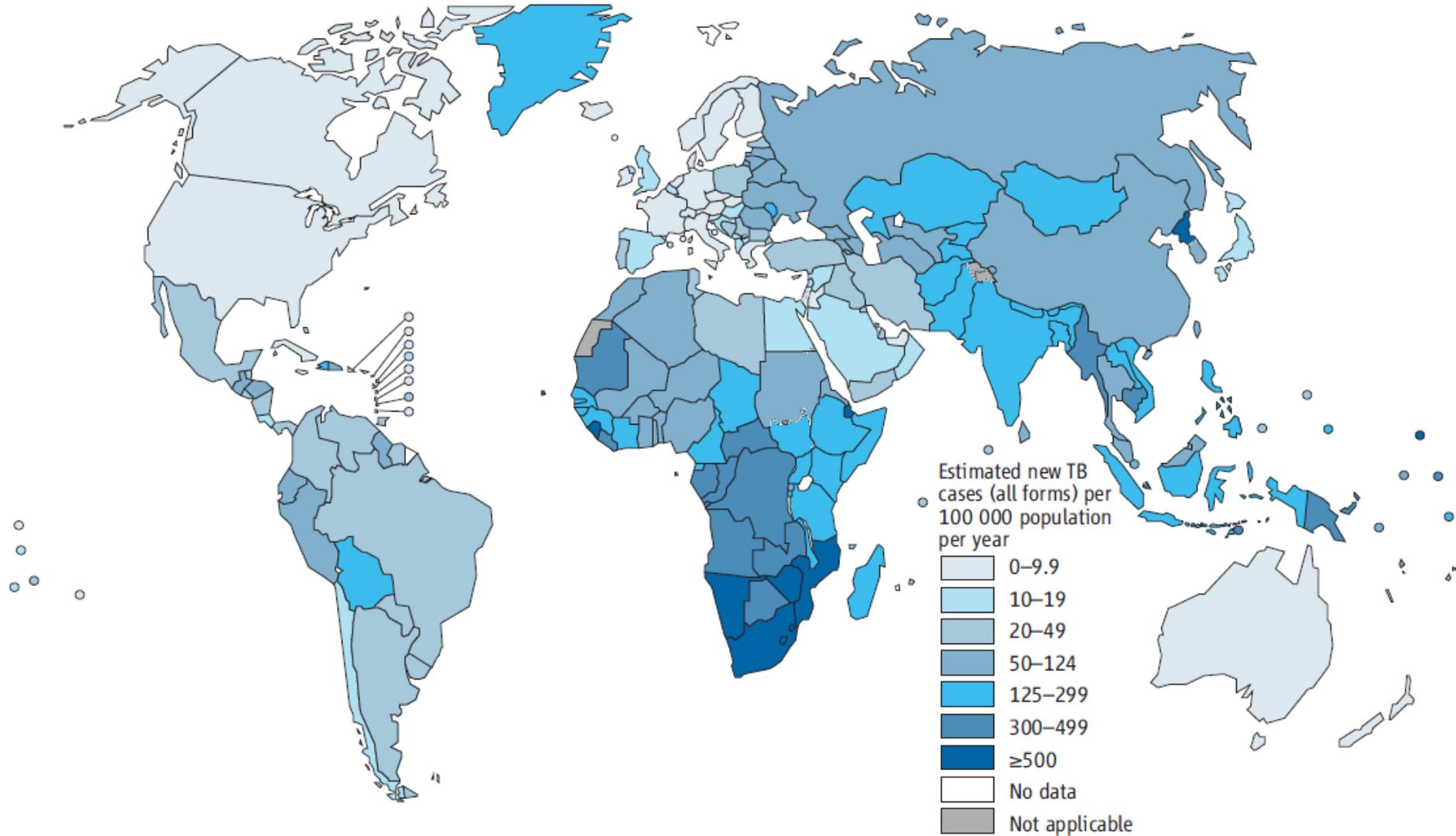


Global Tuberculosis Report 2013

Global tuberculosis report 2013

FIGURE 2.5

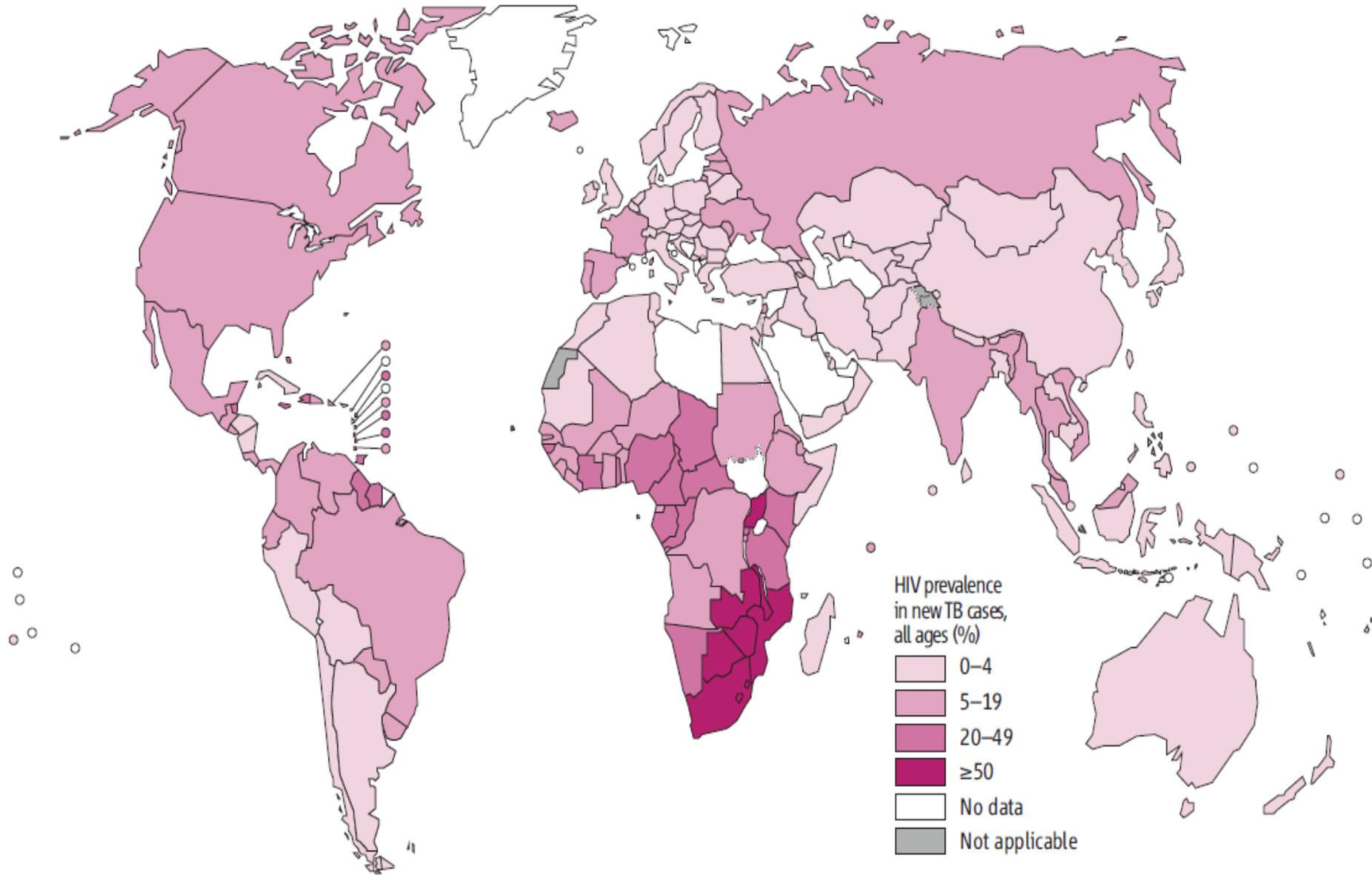
Estimated TB incidence rates, 2012



→ y Educativos - CRIE

FIGURE 2.4

Estimated HIV prevalence in new TB cases, 2012



32 años después...



**34 MILLONES DE
PERSONAS VIVÍAN
CON EL VIH
A FINALES DE 2010**

MMWRTM

MORBIDITY AND MORTALITY WEEKLY REPORT

-1-

1981 June 5;30:250-2

Pneumocystis Pneumonia — Los Angeles

In the period October 1980-May 1981, 5 young men, all active homosexuals, were treated for biopsy-confirmed *Pneumocystis carinii* pneumonia at 3 different hospitals in Los Angeles, California. Two of the patients died. All 5 patients had laboratory-confirmed previous or current cytomegalovirus (CMV) infection and candidal mucosal infection. Case reports of these patients follow.

Patient 1: A previously healthy 33-year-old man developed *P. carinii* pneumonia and oral mucosal candidiasis in March 1981 after a 2-month history of fever associated with elevated liver enzymes, leukopenia, and CMV viruria. The serum complement-fixation CMV titer in October 1980 was 256; in May 1981 it was 32.* The patient's condition deteriorated despite courses of treatment with trimethoprim-sulfamethoxazole (TMP/SMX), pentamidine, and acyclovir. He died May 3, and postmortem examination showed residual *P. carinii* and CMV pneumonia, but no evidence of neoplasia.

MMWRTM

MORBIDITY AND MORTALITY WEEKLY REPORT

The diagnosis of *Pneumocystis* pneumonia was confirmed for all 5 patients ante-mortem by closed or open lung biopsy. The patients did not know each other and had no known common contacts or knowledge of sexual partners who had had similar illnesses. The 5 did not have comparable histories of sexually transmitted disease. Four had serologic evidence of past hepatitis B infection but had no evidence of current hepatitis B surface antigen. Two of the 5 reported having frequent homosexual contacts with various partners. All 5 reported using inhalant drugs, and 1 reported parenteral drug abuse. Three patients had profoundly depressed numbers of thymus-dependent lymphocyte cells and profoundly depressed *in vitro* proliferative responses to mitogens and antigens. Lymphocyte studies were not performed on the other 2 patients.

Reported by MS Gottlieb, MD, HM Schanker, MD, PT Fan, MD, A Saxon, MD, JD Weisman, DO, Div of Clinical Immunology-Allergy, Dept of Medicine, UCLA School of Medicine; I Pozalski, MD, Cedars-Mt. Sinai Hospital, Los Angeles; Field Services Div, Epidemiology Program Office, CDC.

Editorial Note: *Pneumocystis* pneumonia in the United States is almost exclusively limited to severely immunosuppressed patients (1). The occurrence of pneumocystosis in these 5 previously healthy individuals without a clinically apparent underlying immunodeficiency is unusual. The fact that these patients were all homosexuals suggests an association between some aspect of a homosexual lifestyle or disease acquired through sexual contact and *Pneumocystis* pneumonia in this population. All 5 patients described in this report had laboratory-confirmed CMV disease or virus shedding within 5 months



PERSPECTIVE

RETROSPECTIVE

The Discovery of HIV as the Cause of AIDS

Robert C. Gallo, M.D., and Luc Montagnier, M.D.

Progress in scientific research rarely follows a straight path. Generally, it entails many unexpected meanderings, with a mix of good and bad ideas, good and bad luck. The discovery of the human immunodeficiency virus (HIV) as the cause of AIDS did not avoid this pattern.

The story began in an unfavorable environment: during the late 1970s, many people thought that epidemic diseases caused by microbes, including vi-

sion of putative latent retroviruses. This effort was helped greatly by the isolation of specific factors — in particular, the T-cell growth factor (now called interleukin-2) in Bethesda, Maryland. The role of interferon in repressing the production of retroviruses in mouse cells was demonstrated in Paris, and this discovery led to the use of anti-interferon serum in the search for human retroviruses. Thus, at the beginning of the 1980s, we had the essential tools re-



WHO's election throws agency into bitter turmoil

This week the executive board of the World Health Organisation will decide who will be the organisation's director general for the next five years. Usually a routine event, this election is throwing the UN's largest agency into turmoil. Battle lines have been drawn behind the two main candidates—the incumbent, Dr Hiroshi Nakajima, and his former deputy, Dr Mohamed Abdelmoumène. The build up to the vote, scheduled for 20 January, has left WHO's staff committee split, the secretariat demoralised, and the Japanese government accused of foul play and abuse of power. In the words of the chairman of WHO's staff committee, Dr Jan Stjernswärd, "never before has health been so adversely politicised."

There is nothing unusual in the incumbent director general standing for re-election: the last director general, Dr Halldén Mahler, remained in office for three terms. What is unprecedented is the strength of feeling against Dr Nakajima (Dr Mahler's re-election was in each case unopposed) and the ferocity of the lobbying. Last September the United States and the 12 countries of the European Community took the unheard of step of declaring their opposition to Dr Nakajima's re-election, saying that it would not be "in the best interests of the organisation." The Scandinavian countries have now joined the coalition in favour of Dr Abdelmoumène. Japan is the only major industrial nation still supporting Dr Nakajima.

Japan's spirited defence of its man is not easily explained. Within the UN Japan is already well represented by the much respected Japanese high commissioner for refugees, Dr Sadako Ogata. Yet reports in the press, confirmed by diplomats at European and American missions in Geneva, suggest that the Japanese government has been pressuring the poorer members of the executive board, threatening to withdraw trade agreements unless they vote for Dr Nakajima. The Japanese foreign ministry argues that Dr Nakajima has not been in office long enough for his efforts to bear fruit.

The European and American camps in Geneva are anxious that their stance should not appear anti-Japanese. But behind the diplomatic expressions of support for Dr Abdelmoumène there is anger at what they believe is Japan's abuse of power. In the words of one Scandinavian diplomat, "the organisation is too valuable to be allowed to run as a private Japanese concern."

Staff at WHO, members of the secretariat, and even UN diplomats talk openly, though



Dr Nakajima: his bid for re-election has split WHO

most will not be quoted, about their fears for the future. Dr Nakajima's term has, they say, been disastrous. Observers at all levels talk of his lack of vision, his poor communication skills, his high handed managerial style. Under his headship the organisation has, they say, lost its reputation for excellence.

The Japanese government denies that it will withdraw its funding for WHO—currently \$45m a year—if Dr Nakajima is defeated. But diplomats at the American and European missions in Geneva have fears for the organisation's financial security if he is re-elected. Half of the two yearly budget of \$1.5bn comes from voluntary contributions to WHO's international programmes, including those on AIDS, immunisation, and diarrhoeal diseases. These, say observers, have continued to perform well despite rather than because of Dr Nakajima, and 90% of their funding comes from countries now supporting Dr Abdelmoumène. Members of WHO's secretariat predict that Dr Nakajima's re-election would leave some donors unwilling to continue financing these programmes.

Few people are pretending that Dr Abdelmoumène is the perfect candidate. In his favour, say observers, is his stated desire to reform the structure of WHO. An Algerian doctor, he has been on special leave since being dismissed by Dr Nakajima after declaring his candidacy in September.

Only 31 of WHO's 182 member states have a vote on the executive board. Each of these has nominated a person—in Britain's case the

chief medical officer, Dr Ken Calman—to cast its vote in a secret ballot. The final nominations will not be known until the opening of the executive board meeting on 18 January. But there seems little doubt that a third candidate, Dr Otkoye Ransome-Kuti of Nigeria, will be included. Nigeria has no vote on the board, and Dr Ransome-Kuti is unlikely to attract much support from the seven African countries with votes, most of whom are believed to be heavily committed to Dr Nakajima. But a third horse in the race may divide the vote sufficiently to force a second round. A close vote is also likely to mean continued lobbying in the three months to May, when the World Health Assembly meets to ratify the executive board's decision.—HONNA GODLEE, BMJ

Gallo guilty of AIDS misconduct

Robert Gallo, once credited as the discoverer of HIV, has been found guilty of scientific misconduct by an official government inquiry in the US. Gallo, head of a laboratory at the US National Cancer Institute near Washington, deliberately concealed experiments that would have made it clear that French scientists had discovered HIV a year earlier than he claimed.

The Office of Research Integrity—an investigative arm of the US Department of

Headlines

Euro 999 service: A pan-European emergency number came into operation in the European Community on 1 January. Anyone dialling 112 will be linked to the ambulance, police, and fire services in a scheme which runs parallel to the UK's 999 number. Spain and the Netherlands are expected to join the scheme by 1995.

RHA chairman resigns: Sir James Ackers has resigned as chairman of West Midlands Regional Health Authority and will be succeeded by Sir Donald Wilson, chairman of Mersey RHA. The authority was criticised by the National Audit Office for wasting £4m on computer contracts. Sir James has also resigned from his position on the NHS Policy Board.

Clinton faces soaring health costs: Figures from the US Commerce Department show that health care cost more than 14% of the nation's economic output last year. Costs have nearly doubled since 1985 and are predicted to reach \$940 bn this year. Bill Clinton has promised a health plan in his first 100 days as president.

Russia's infant death rate rises: The infant death rate in Russia has increased from 16.8 per 1000 births in 1991 to 17.1 in 1992. The number of births has fallen by 30% over the past four years. The government said that couples are putting off having children until the recession improves.

Intoxication main reason for police surgeons to visit: Doctors see one in four police prisoners in inner London, mostly for intoxication, says a study carried out for the Royal Commission on Criminal Justice. The commission says that there is a need for standardisation in the role of doctors in assessing a suspect's fitness for interview.

Beds alert in London: Many hospitals in north and east London have been put on an official alert because of a shortage of beds for emergency admissions. The hospitals affected have been asked to reduce substantially their admissions from waiting lists.

Compulsory AIDS tests: The Singapore government has introduced compulsory AIDS tests for its 300 000 foreign labourers. Foreign executives have been excluded. Anyone found to be HIV positive will have to leave the country.



Gallo announcing his research findings in 1984

Health and Human Services—said that in a research paper published in *Science* in 1984 Gallo wanted to hide a critical fact that would have given credit for the discovery of the cause of AIDS to a team led by Luc Montagnier at the Pasteur Institute in Paris. Gallo was given a sample of the French virus in 1983 but said in his paper that it failed to grow in tissue culture. The misconduct inquiry found that this was "knowingly false when written" and accuses Dr Gallo of concealing information that would have been helpful to other scientists.

"Furthermore, the misrepresentation had the potential to impede the rapid advancement of research efforts with LAV [the name for the French virus]," the inquiry concludes. "Because of Dr Gallo's senior status and his highly visible role in the research community, the finding of scientific misconduct... carries a high degree of opprobrium."

The inquiry also found that Gallo exhibited actions reflecting his "propensity to misrepresent and mislead in favour of his own research findings or hypotheses." Dr Gallo is also guilty of "irresponsible laboratory management that has permanently impaired the ability to trace the important steps taken" in research into AIDS and of imposing "restrictive conditions" on access to scientific reagents needed by other medical researchers. "Overall, [the Office of Research Integrity] finds that the events surrounding the discovery of the AIDS virus... are... a tragedy for science."

Gallo said that he intends to appeal against the inquiry report, which "could only take issue with a few trivial mistakes and a single sentence written by me." He added, "The mindless pursuit of fantasised misconduct can have devastating consequences for scientific research."

The conclusions of the Office of Research Integrity overturn the verdict of an earlier investigation into Gallo by the Office of Scientific Integrity set up by the National Institutes of Health (NIH), which found that

he was not guilty of scientific misconduct. The misconduct finding has therefore put Bernadine Healy, the director of NIH, who supported Gallo, in an awkward position. She said: "I concurred with the conclusions of the NIH Office of Scientific Integrity based on the facts as they were presented to me after a lengthy investigation."

Gallo is facing a wider investigation by a subcommittee of Congress, which will review evidence that he misappropriated the French virus. It will also investigate a possible cover up of the Gallo affair, a Congressional source said. Dr Gallo also faces another investigation into allegations that he gave false information in an application for a patent—a possible criminal offence.

Lawyers for the Pasteur Institute, meanwhile, intend to press for a greater share in royalties on the blood test for AIDS. "The US Government's position—built on a faulty two-virus theory—has been sitting at the [Department of Health] like a ticking time bomb," said Robert Odle, an attorney for the Pasteur Institute. "It has now exploded."—STEVE COOPER, science correspondent, *Independent*

Canadian medical research expands

Having completed what it calls the most thorough self appraisal in its history, the Medical Research Council of Canada will publish later this month a five year strategic plan that will greatly expand its role. Broadening its remit to cover the widest definition of health sciences research, the council wants to probe the socioeconomic factors in ill health, ask fundamental questions about the outcomes and cost effectiveness of medical interventions, undertake research into bioethical issues, and form creative partnerships with industry.

The council says that the pharmaceutical industry is a \$60bn business in Canada and wants to support more research. Other industries, such as those making medical devices, computer software, electronics, and chemicals, might also enter partnerships with the Medical Research Council of Canada.

"The range of specialties contributing expertise to health issues now includes lawyers and philosophers, engineers and physicists, social scientists, even historians, says the agency's report. Where once only researchers in traditional biomedical subjects were funded by the council, under the new plan "we may find ourselves supporting projects proposed by an economist, a bio-ethicist, a statistician, a public administration specialist and others."

Over the years the council's role has been largely reactive: rather than initiate research it has relied to a large extent on the scientific community to submit proposals. Each year more than 3000 people do so, while some 2000 Canadian and international experts give their time to evaluate them. In 1992 the council distributed \$230m to some 2200 researchers and 1500 research trainees.



The Nobel Prize in Physiology or Medicine 2008

Harald zur Hausen, Françoise Barré-Sinoussi, Luc Montagnier

The Nobel Prize in Physiology or Medicine 2008

Nobel Prize Award Ceremony

Harald zur Hausen

Françoise Barré-Sinoussi

Luc Montagnier



Photo: U. Montan

Harald zur Hausen



Photo: U. Montan

Françoise Barré-Sinoussi



Photo: U. Montan

Luc Montagnier

The Nobel Prize in Physiology or Medicine 2008 was divided, one half awarded to Harald zur Hausen *"for his discovery of human papilloma viruses causing cervical cancer"*, the other half jointly to Françoise Barré-Sinoussi and Luc Montagnier *"for their discovery of human immunodeficiency virus"*.

NUMBER OF PEOPLE NEWLY INFECTED WITH HIV, GLOBAL, 1990–2011



Nuevos infectados

NUMBER OF PEOPLE LIVING WITH HIV, GLOBAL, 1990–2011

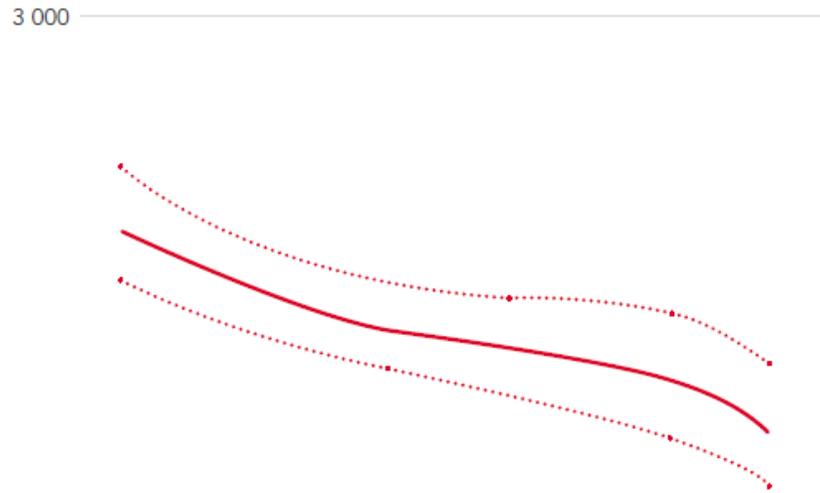


ADULT AND CHILD DEATHS DUE TO AIDS, GLOBAL, 1990–2011

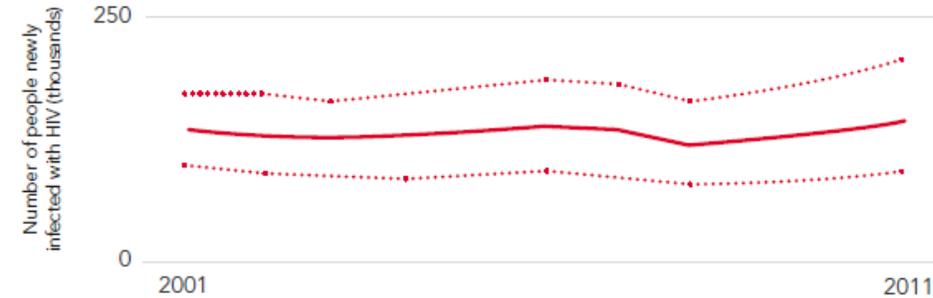


Number of people newly infected with HIV, 2001–2011, by region

SUB-SAHARAN AFRICA



EASTERN EUROPE AND CENTRAL ASIA



LATIN AMERICA



Adults and children living with HIV

Adults and children newly infected with HIV

LATIN AMERICA

2011

1.4 million
[1 100 000–1 700 000]

83 000
[51 000–140 000]

2001

1.2 million
[970 000–1 500 000]

93 000
[67 000–120 000]

Figure 2.2

Changes in the incidence of HIV infection, 2001 to 2009

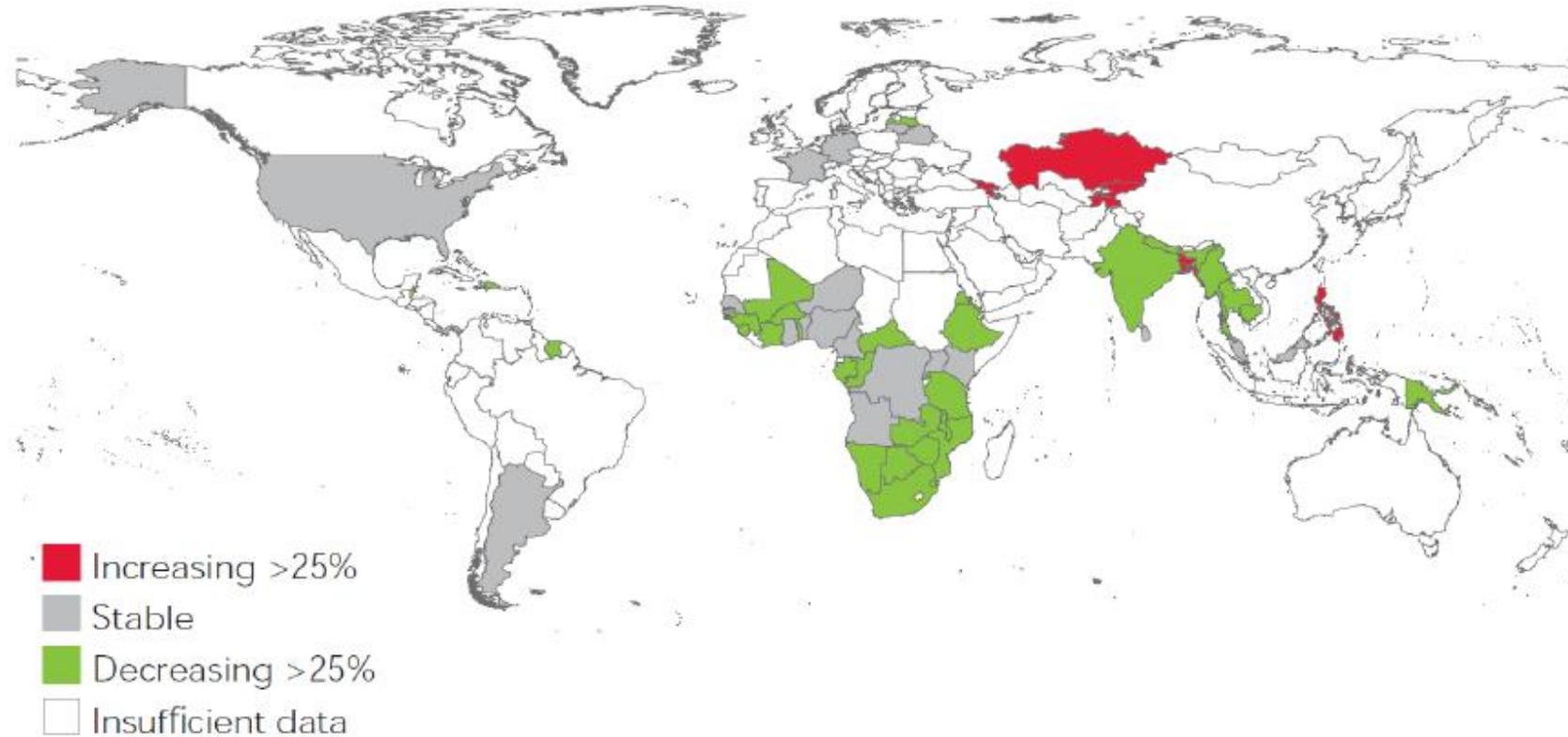
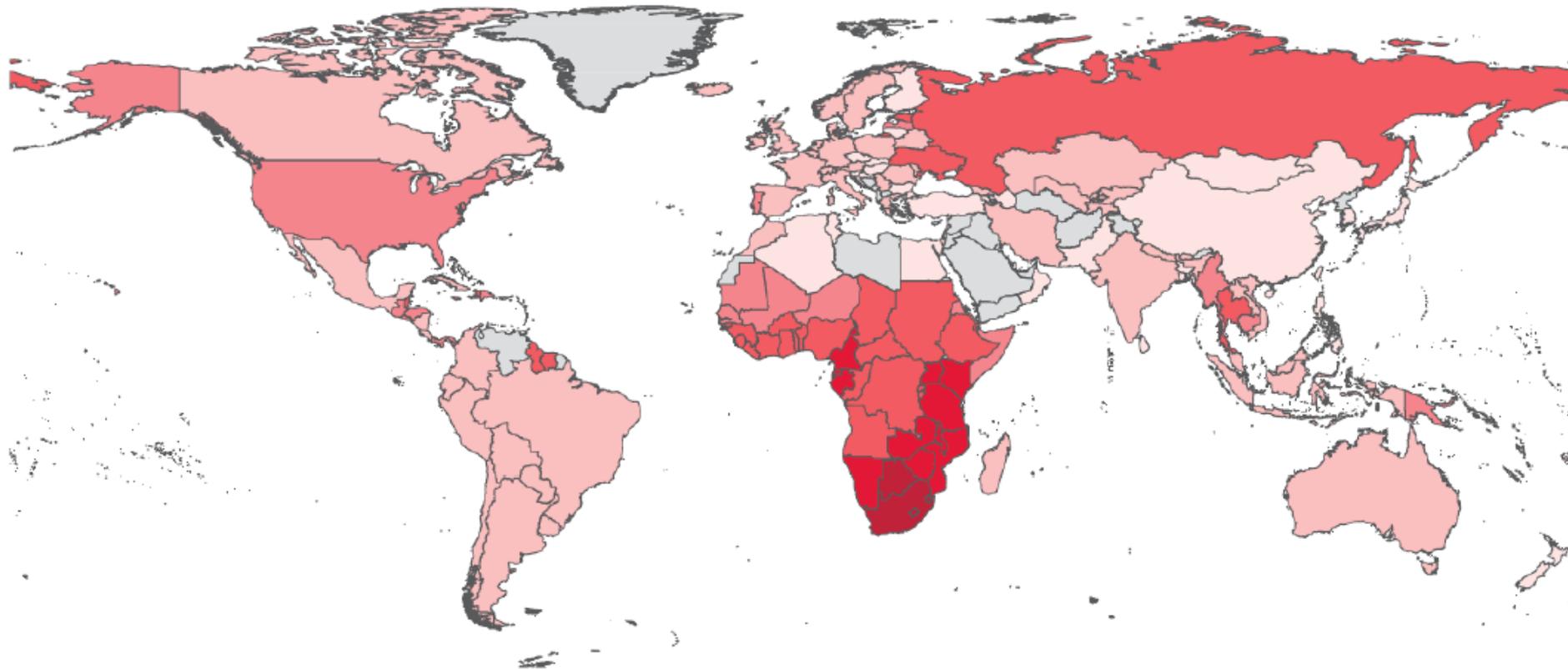


Figure 2.4

Global prevalence of HIV, 2009



Legend for HIV prevalence (2009):

- No data
- <.1%
- .1% – <.5%
- .5% – <1%
- 1% – <5%
- 5% – <15%
- >15% – 28%

Table 2.2 (3/6)

Regional HIV and AIDS statistics, 2001 and 2009

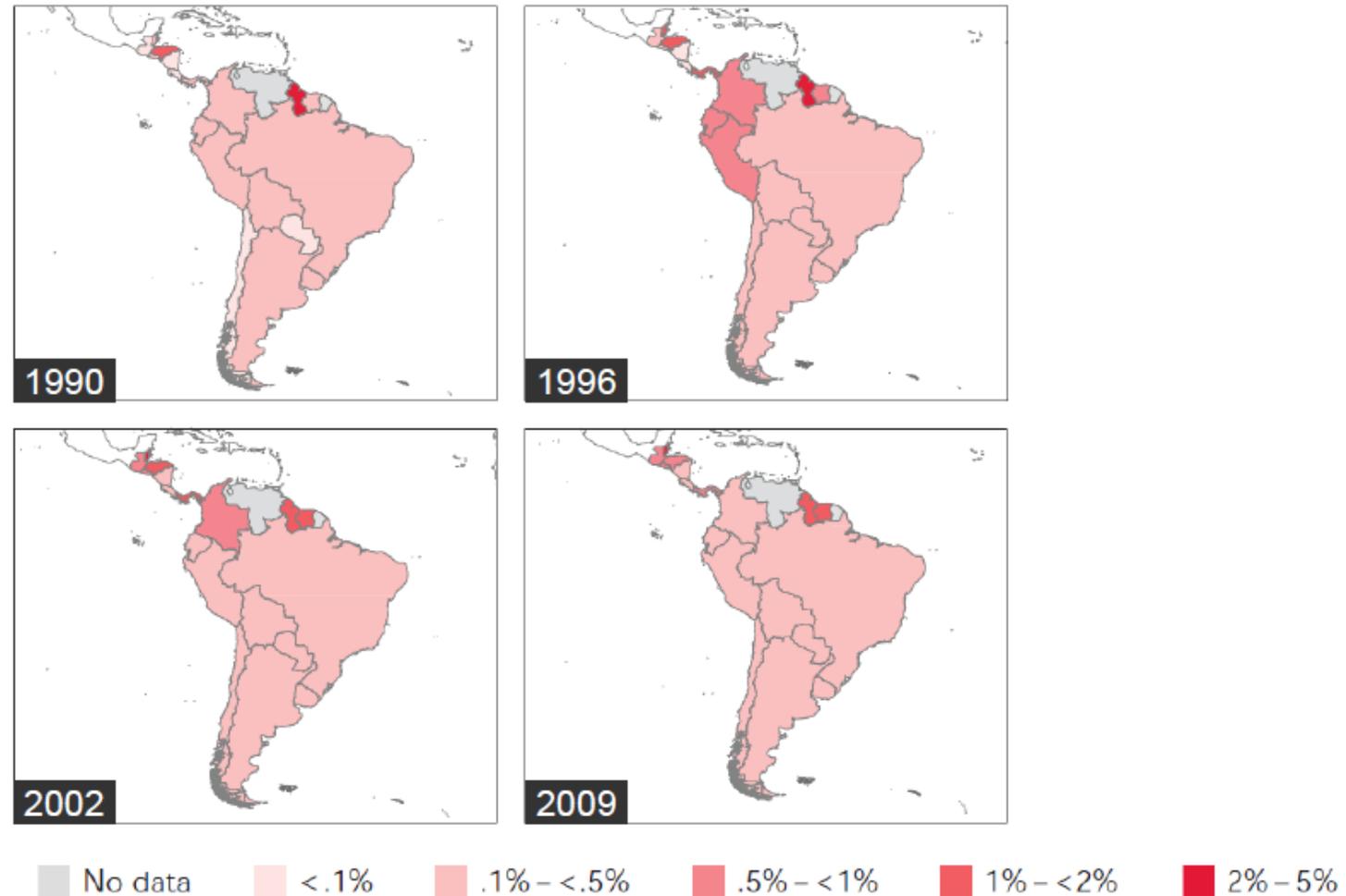
Regional figures on adults and children newly infected and living with HIV and AIDS-related deaths

		Adults and children living with HIV	Adults and children newly infected	% Adult prevalence (15–49 years)	AIDS-related deaths among adults and
OCEANIA	2009	57 000 [50 000–64 000]	4500 [3400–6000]	0.3 [0.2–0.3]	1400 [<1000–2400]
	2001	29 000 [23 000–35 000]	4700 [3800–5600]	0.2 [0.1–0.2]	<1000 [<500–1100]
CENTRAL AND SOUTH AMERICA	2009	1.4 million [1.2–1.6 million]	92 000 [70 000–120 000]	0.5 [0.4–0.6]	58 000 [43 000–70 000]
	2001	1.1 million [1.0–1.3 million]	99 000 [85 000–120 000]	0.5 [0.4–0.5]	53 000 [44 000–65 000]

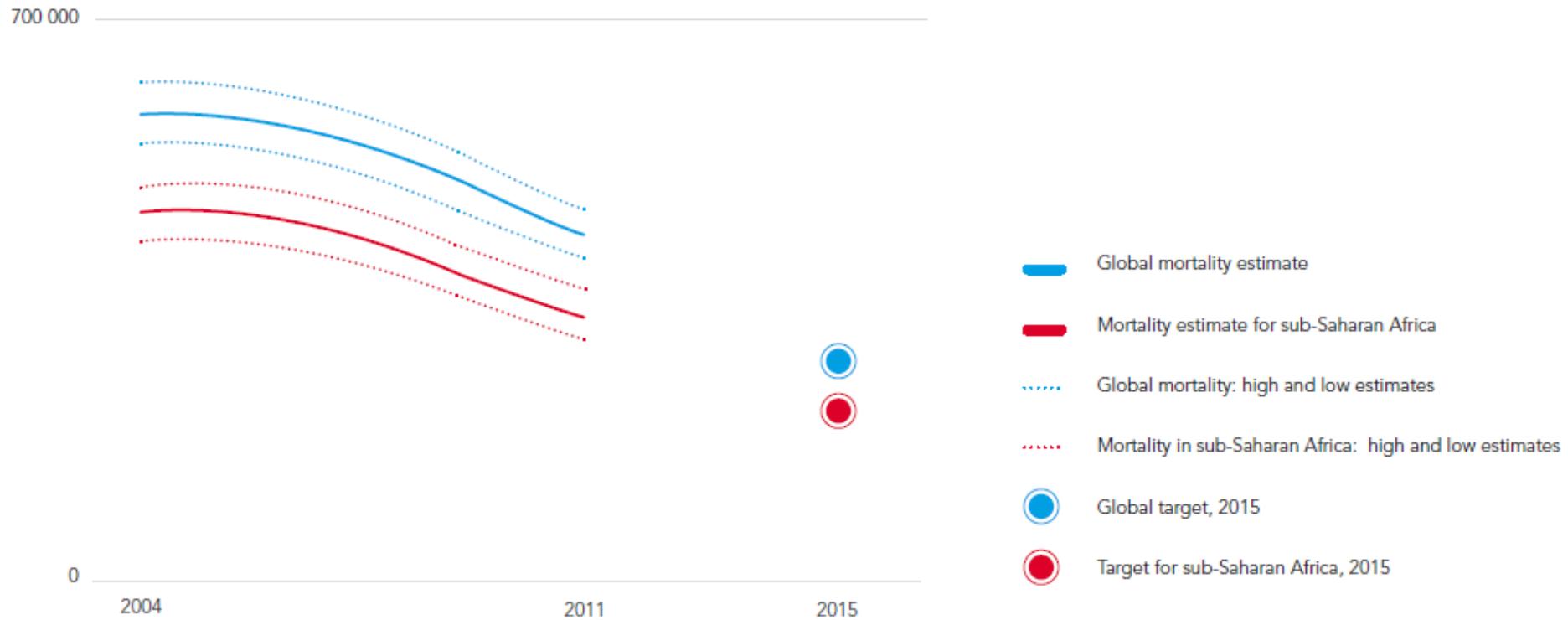
Figure 2.16

HIV prevalence in Central and South America

HIV prevalence among adults aged 15–49 years old in Central and South America, 1990 to 2009.



Estimated number of TB-related deaths among people living with HIV, 2004–2011



EPIDEMIOLOGICAL FACTSHEET

for more country data visit <http://aidsinfo.unaids.org>

COLOMBIA



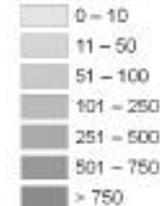
HIV prevalence among men having sex with men, 2004–2008

HIV prevalence (%)



○ Cities and towns

**Population density (2005)
(pers./sq.km)**



The boundaries and names shown and the designations used on this map do not imply the expression of any opinion whatsoever on the part of the World Health Organization concerning the legal status of any country, territory, city or area or of its authorities, or concerning the delimitation of its frontiers or boundaries. Dotted lines on maps represent approximate border lines for which there may not yet be full agreement.
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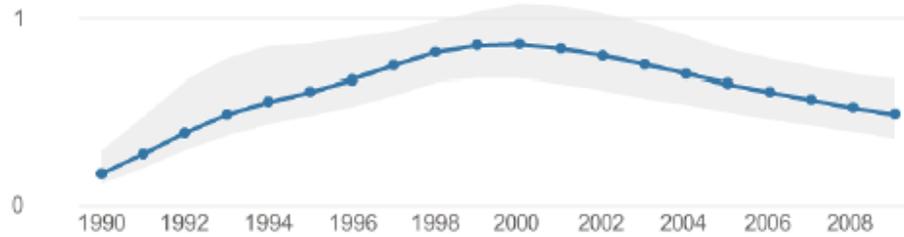
Data source: UNAIDS, WHO, CIESIN/FAO/CIAT, DCW, GeoNames, USCB.
Map production: Public Health Information and Geographic Information Systems (GIS), WHO

EPIDEMIOLOGICAL FACTSHEET

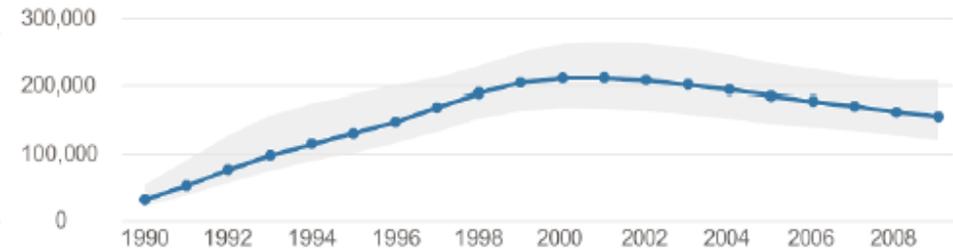
for more country data visit <http://aidsinfo.unaids.org>

COLOMBIA

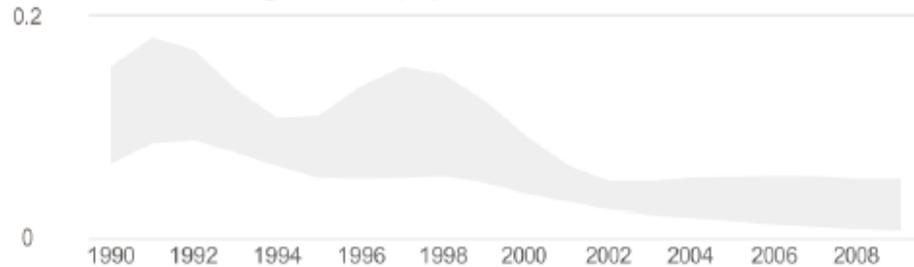
HIV prevalence-Ages 15-49(%)



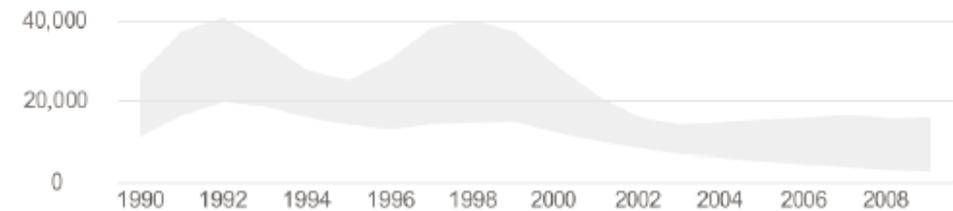
Number of people living with HIV



HIV incidence rate-Ages 15-49(%)



Number of new infections-All ages

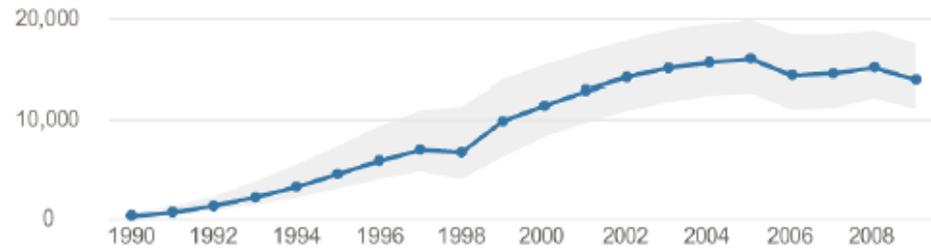


EPIDEMIOLOGICAL FACTSHEET

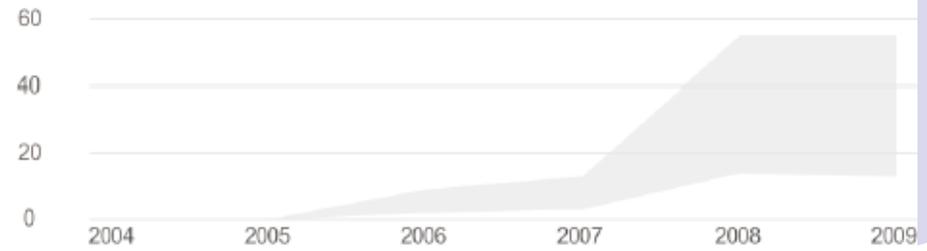
for more country data visit <http://aidsinfo.unaids.org>

COLOMBIA

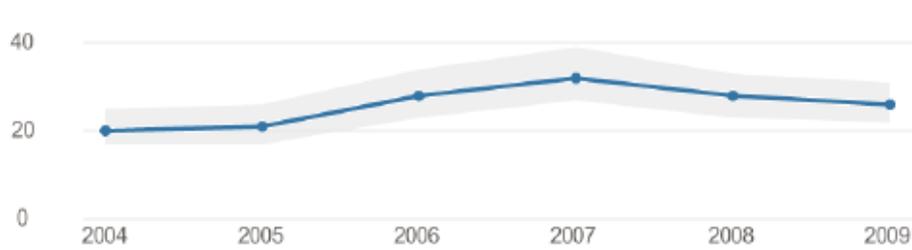
Annual number of AIDS deaths



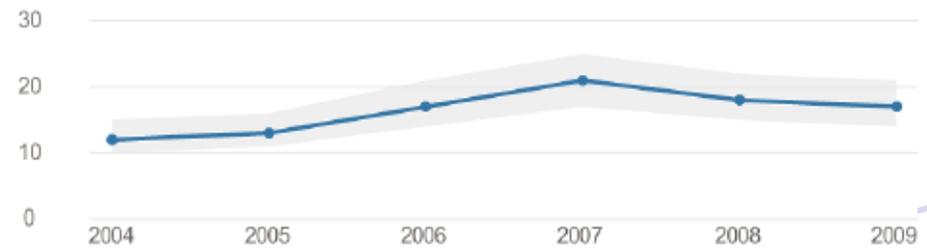
PMTCT coverage (%)



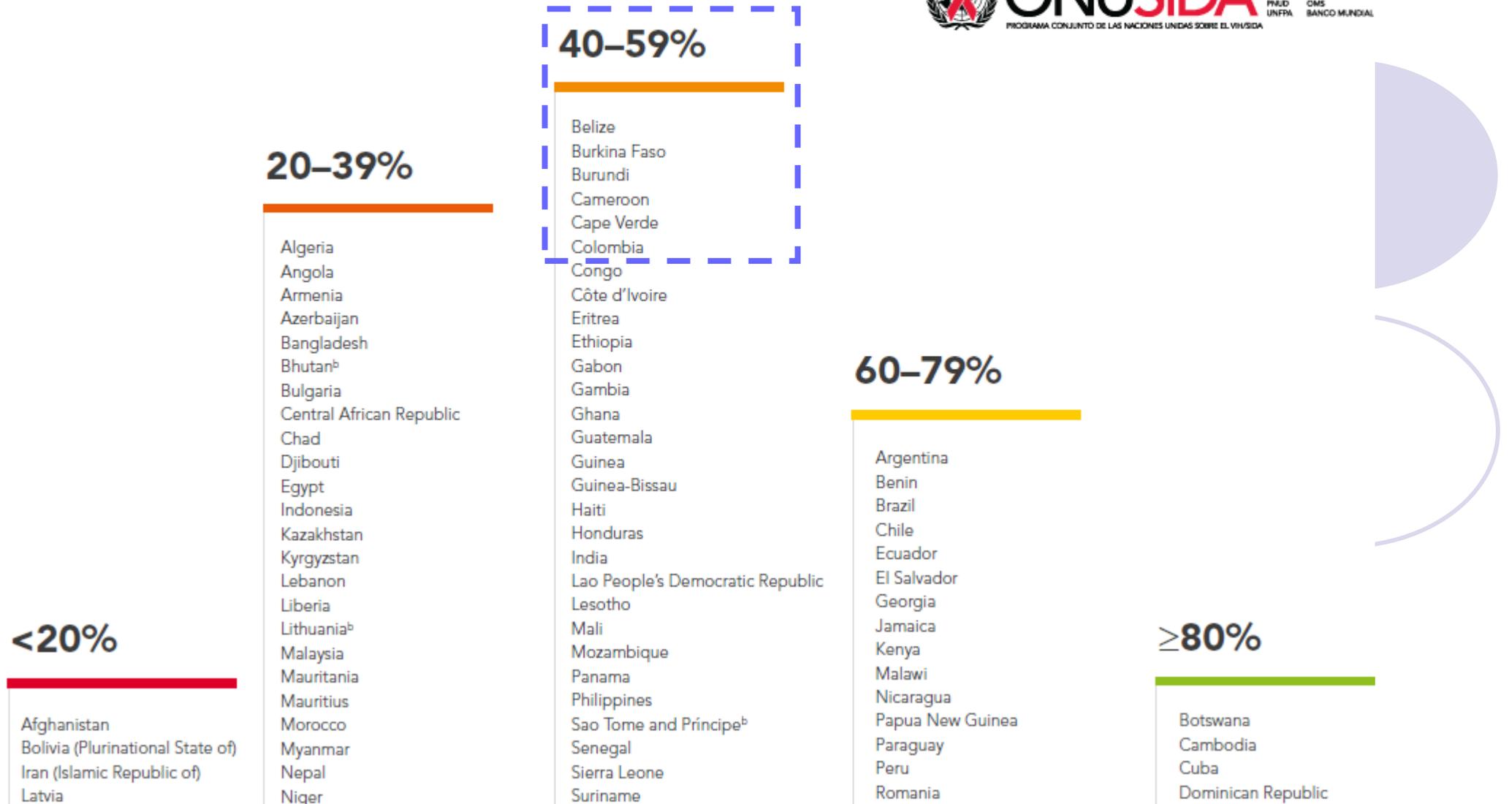
ART coverage (%) CD4<200



ART coverage (%) CD4<350



Proportion of eligible people receiving antiretroviral therapy in selected low- and middle-income countries at the end of 2011^a



Informe UNGASS - 2010

Seguimiento de la Declaración de compromiso
sobre el VIH/sida

INFORME NACIONAL

República de Colombia.



EXISTENCIA



COMUNICACIÓN



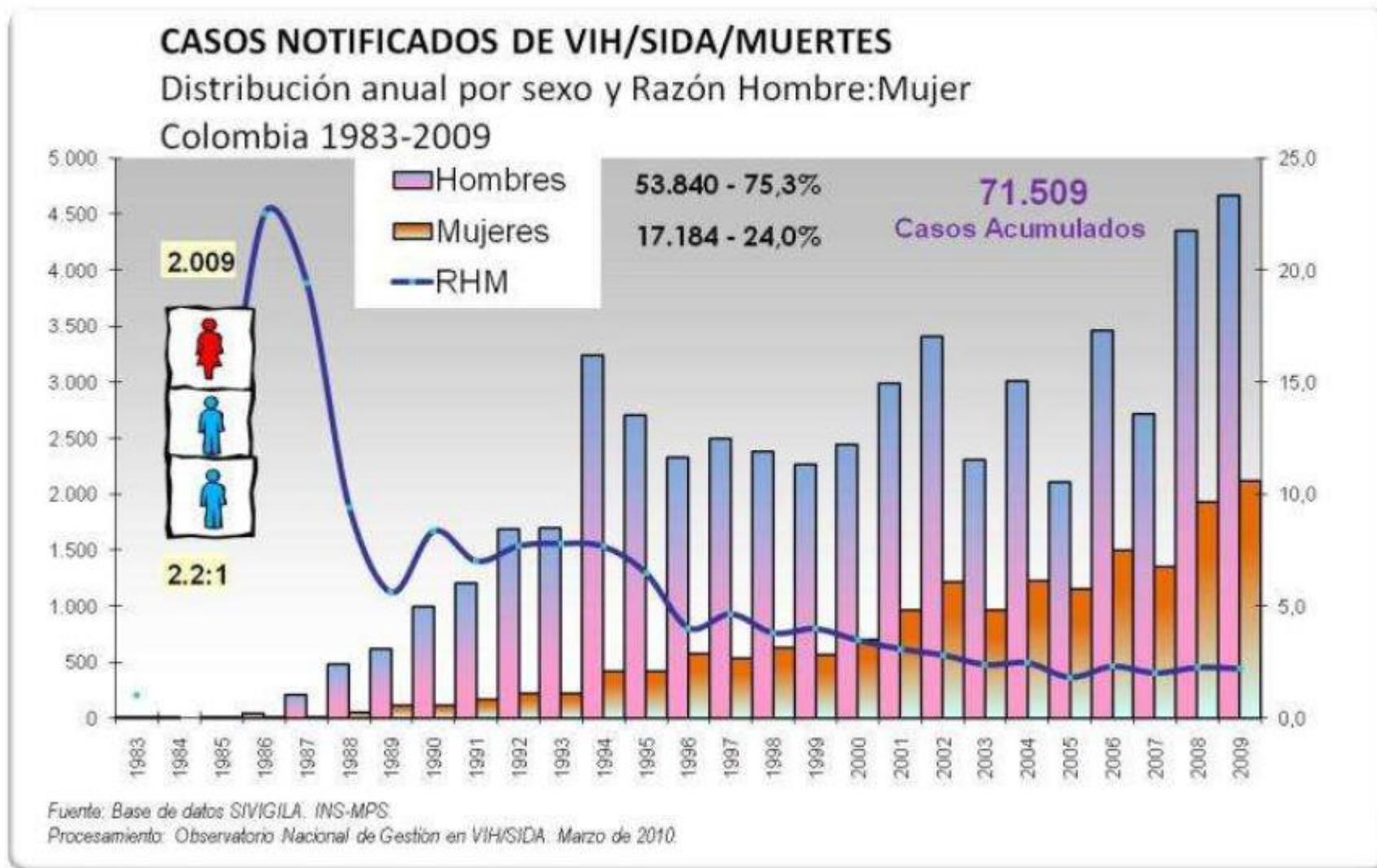
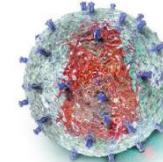
RESPUESTA



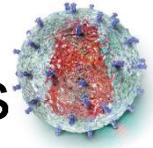
Libertad y Orden

Ministerio de la Protección Social
República de Colombia

GRÁFICA 1. CASOS NOTIFICADOS DE VIH/SIDA/MUERTES.

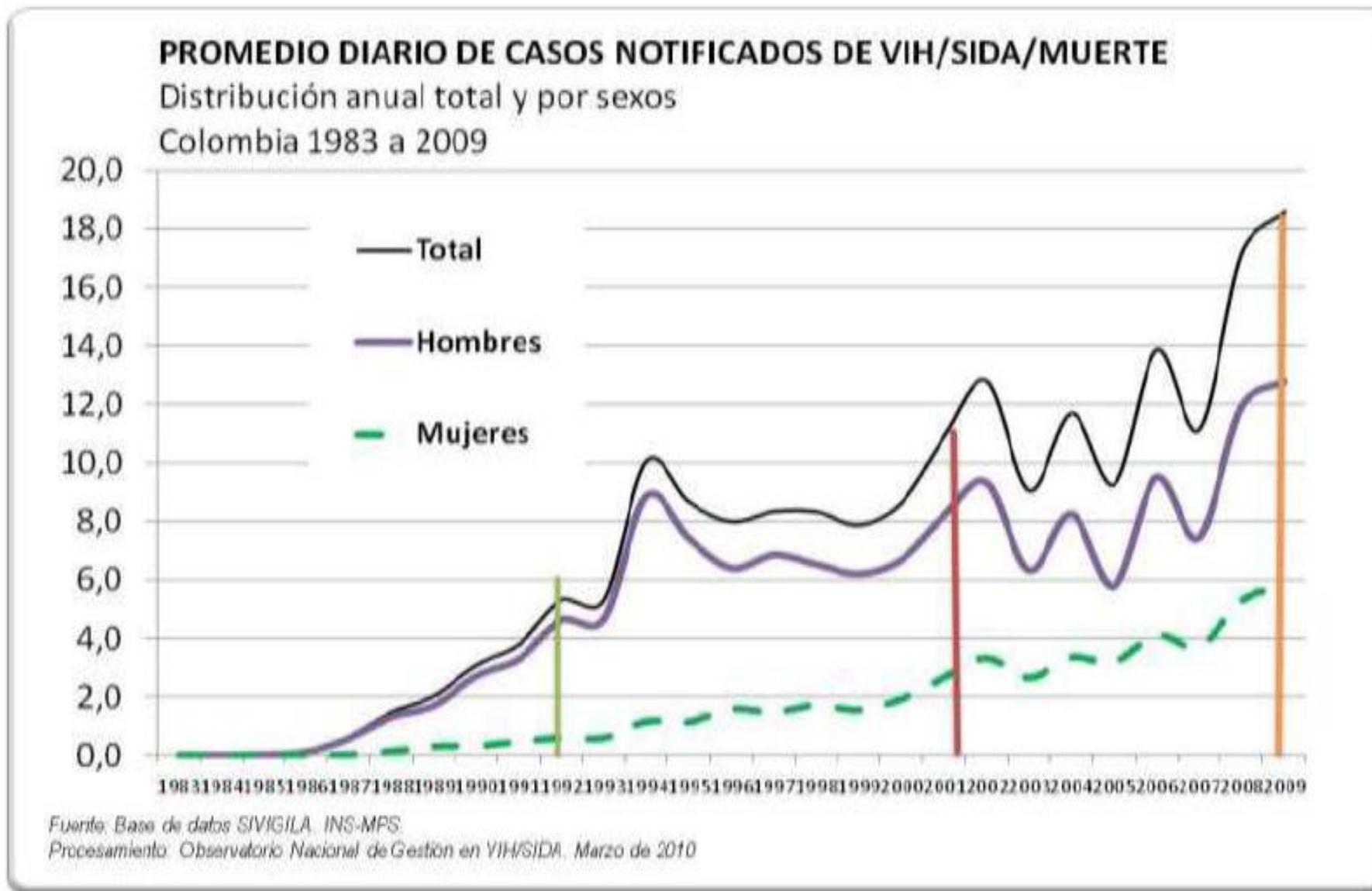
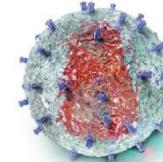


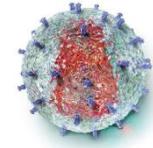
La tendencia general es de incremento en la frecuencia de casos notificados que puede obedecer a varios factores:



- El crecimiento de casos de infecciones recientemente adquiridas.
- Mayor acceso a la asesoría y pruebas voluntarias.
- Oferta masiva al grupo de gestantes.
- Mayor demanda de servicios diagnósticos.
- Disminución de barreras administrativas para autorización de servicios.

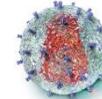
GRÁFICA 2. PROMEDIO DIARIO DE CASOS NOTIFICADOS DE VIH/SIDA/MUERTE.



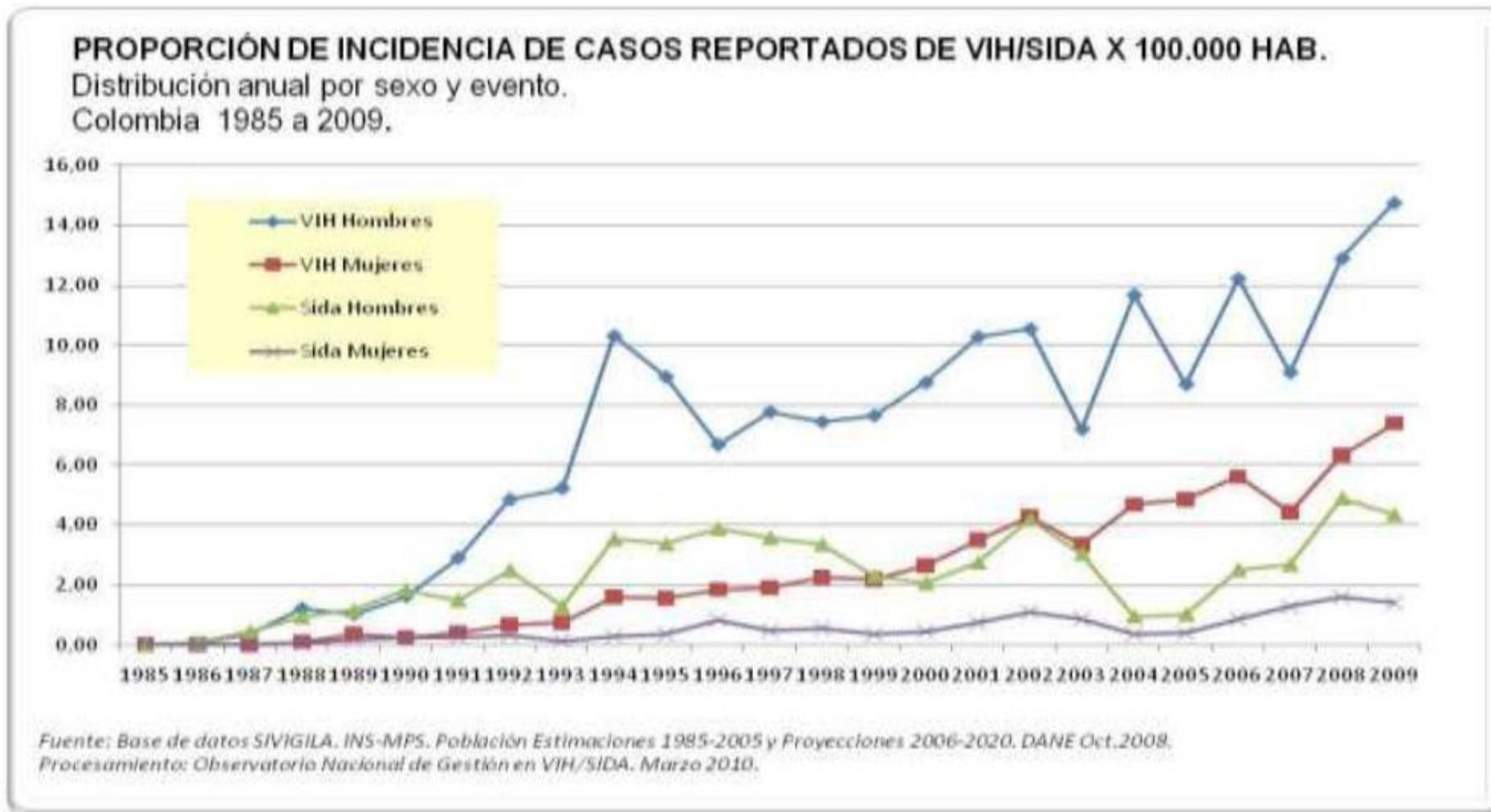


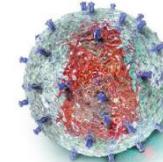
GRÁFICA 4. PROPORCIÓN DE INCIDENCIA DE VIH Y SIDA POR 100.000 HAB.





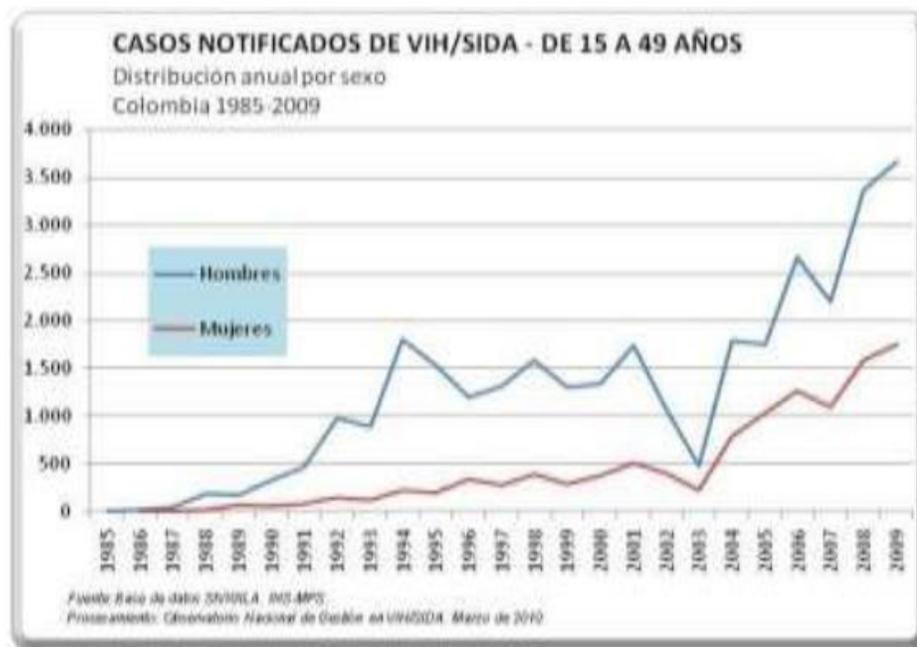
GRÁFICA 5. PROPORCIÓN DE INCIDENCIA DE CASOS REPORTADOS DE VIH/SIDA. SEXO Y EVE





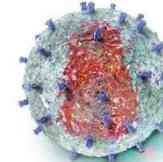
GRÁFICA 13. CASOS NOTIFICADOS DE VIH/SIDA – 15 A 49 AÑOS. PROYECCIÓN.

Gráfica 12.



Gráfica 13.





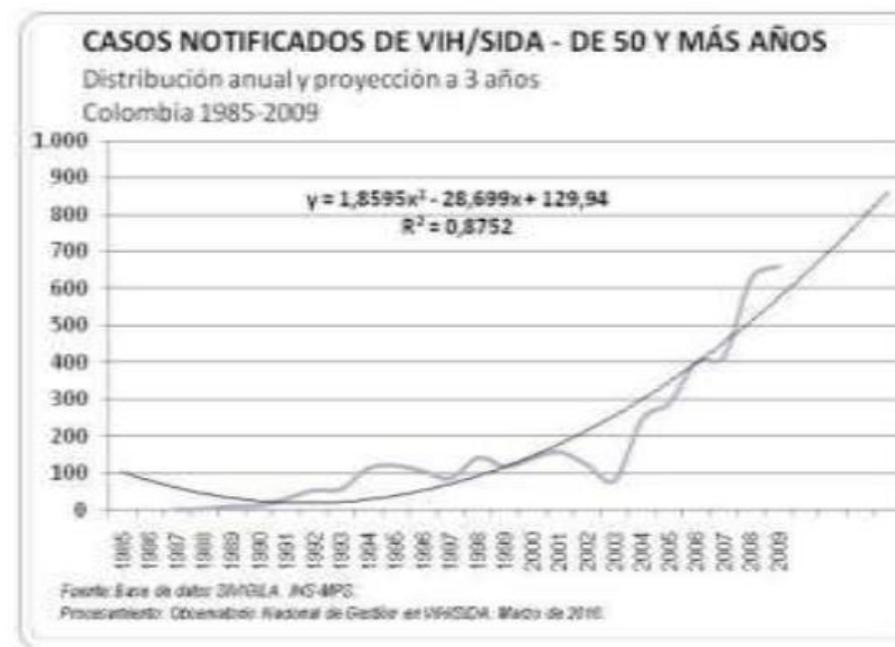
GRÁFICA 14. CASOS NOTIFICADOS DE VIH/SIDA – 50 Y MÁS AÑOS. SEXO.

GRÁFICA 15. CASOS NOTIFICADOS DE VIH/SIDA – 50 Y MÁS AÑOS. PROYECCIÓN.

Gráfica 14.



Gráfica 15.



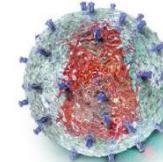


TABLA 1. PREVALENCIA GLOBAL DE VIH/SIDA.

Estudios Centinela varias subpoblaciones. Colombia 1988 a 2009.

Estudio	Año	Ciudades	Muestra	Prevalencia
I	1.988	1	4.085	0.02%
II	1.991	6	7.025	0.10%
III	1.994	6	12.044	0.30%
IV	1.996	5	7.037	0.40%
V	1.999	12	22.069	0.49%
VI	2.003	11	3.239	0.65%
VII	2.009	13	18.934	0.22%

Fuente: Observatorio Nacional de VIH. Ministerio de la Protección Social. Febrero 2010.

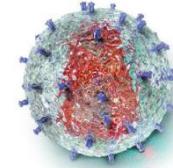


TABLA 2. PREVALENCIA DE VIH EN GESTANTES. ESTUDIOS CENTINELA.

Distribución por año.

Colombia, varias ciudades 1988 a 2009.

Estudio	Año	Muestra	Confirmados	Prevalencia
I	1988	4.085	1	0,02%
II	1991	10.825	4	0,04%
III	1994	11.265	23	0,20%
IV	1996	8.739	19	0,22%
V	1999	8.690	21	0,24%
VI	2003	1.548	10	0,65%
VII	2009	18.934	42	0,22%

Fuentes: Infección por VIH y sida en Colombia. Ministerio de Salud y ONUSIDA. 1999. pp.95.

V Estudio Centinela Nacional de Vigilancia de Infección por VIH-1. Colombia 1999. Instituto Nacional de Salud y Ministerio de Salud. Publicado en 2000. pp.10.

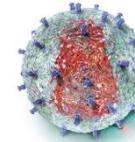
VI Estudio Nacional Centinela de VIH 2003-2004. Informe Final. Instituto Nacional de Salud. Agosto de 2004. pp.7.

Infección por VIH y sida en Colombia. 2000-2005. Ministerio de la Protección Social y ONUSIDA. 2006. pp.67-69.

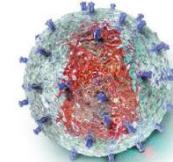
VII Estudio Nacional Centinela de VIH en Gestantes. Informe de resultados. Instituto Nacional de Salud y Ministerio de Salud. Diciembre 2009. pp. 14.

TABLA 4. CASOS DE MORTALIDAD ASOCIADA AL VIH/SIDA

Distribución anual de frecuencias, tasa de mortalidad por 100.000 Habitantes
Colombia 1991 a 2006

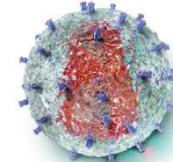


Año.	Mortalidad por el VIH (SIDA).1	Población. 2	Tasa por 100.000 Hab.
1991	262	34.833.548	0,75
1992	637	35.530.177	1,79
1993	849	36.208.244	2,34
1994	1.117	36.862.626	3,03
1995	1.326	37.489.666	3,54
1996	1.479	38.076.638	3,88
1997	1.467	38.646.043	3,80
1998	1.444	39.201.321	3,68
1999	1.713	39.745.714	4,31
2000	1.907	40.282.217	4,73
2001	2.112	40.806.313	5,18
2002	2.115	41.327.459	5,12
2003	2.205	41.847.421	5,27
2004	2.430	42.367.528	5,74
2005	2.305	42.888.592	5,37
2006	2.343	43.405.387	5,40
2007	2.489	44.450.260	5.60



PROPORCIÓN DE INCIDENCIA DE CASOS NOTIFICADOS DE VIH/SIDA/MUERTE
DISTRIBUCIÓN POR DEPARTAMENTO DE RESIDENCIA Y EVENTO
Colombia 2009 x 100.000 Hab.

DEPARTAMENTO	POBLACION 2009	1. VIH	2. Sida	3. Muerte	Total
ATLANTICO	2.284.840	20,7	5,7	0,4	26,9
QUINDIO	546.566	20,1	4,2	1,5	25,8
CESAR	953.827	18,5	1,8	1,6	21,8
VALLE	4.337.909	15,2	4,4	2,1	21,7
CASANARE	319.502	16,9	2,8	0,3	20,0
SANTANDER	2.000.045	13,5	2,6	1,8	17,9
GUAVIARE	78449	12,7	5,1	0,0	17,8
BOGOTÁ	7.259.597	13,5	2,6	1,6	17,7
MAGDALENA	1.190.585	12,3	3,2	1,7	17,1
RISARALDA	919.653	12,1	3,0	2,0	17,1



CASOS REGISTRADOS DE VIH/SIDA/MUERTE

Distribución por DTS y evento

Colombia 2009

DTS	1. VIH	2. Sida	3. Muerte	Total
BOGOTÁ	978	191	115	1.284
ANTIOQUIA	698	257	50	1.005
VALLE	660	190	92	942
BARRANQUILLA DT.	317	105	6	428
SANTANDER	270	53	36	359
CORDOBA	185	54	20	259
CESAR	176	17	15	208
ATLANTICO	157	25	4	186
CARTAGENA. DT.	151	23	11	185
RISARALDA	111	28	18	157

Situación del VIH-Sida en Colombia

- Epidemia de bajo nivel
- Epidemia Concentrada: Cuando la prevalencia en grupos más expuestos supera el 5% y cuando en mujeres gestantes esta es menor del 1%**
- Epidemia Generalizada

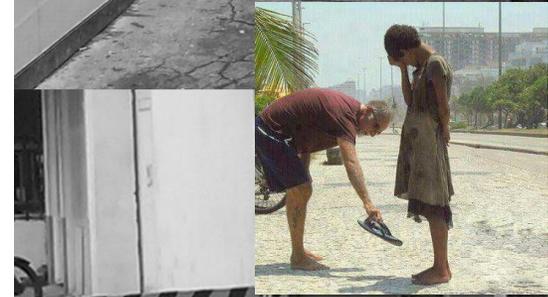
Grupos más expuestos:

- HSH
- Mujeres trabajadoras sexuales
- Jóvenes
- Población en situación de desplazamiento: 200 desde 2004**



Condiciones de Riesgo

- **HSH y Mujeres Trans**
- **Mujeres trabajadoras sexuales**
- **Jóvenes**
- **Población en situación de desplazamiento**
- **Habitantes de calle**
- **Personas privadas de la libertad**
- **Poblaciones móviles y fuerzas militares**





PREVALENCIA DEL VIH EN HSH EN COLOMBIA

Ciudad	Prevalencia
Barranquilla	13,6%
Cartagena	10%
Medellín	9,1%
Pereira	5,6%
Bogotá	15%
Cali	24,1%
Cúcuta	10,9%

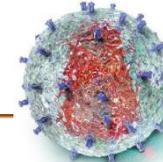
Fuente: MSPS, UNFPA, OPS, SDS Btá, 2010



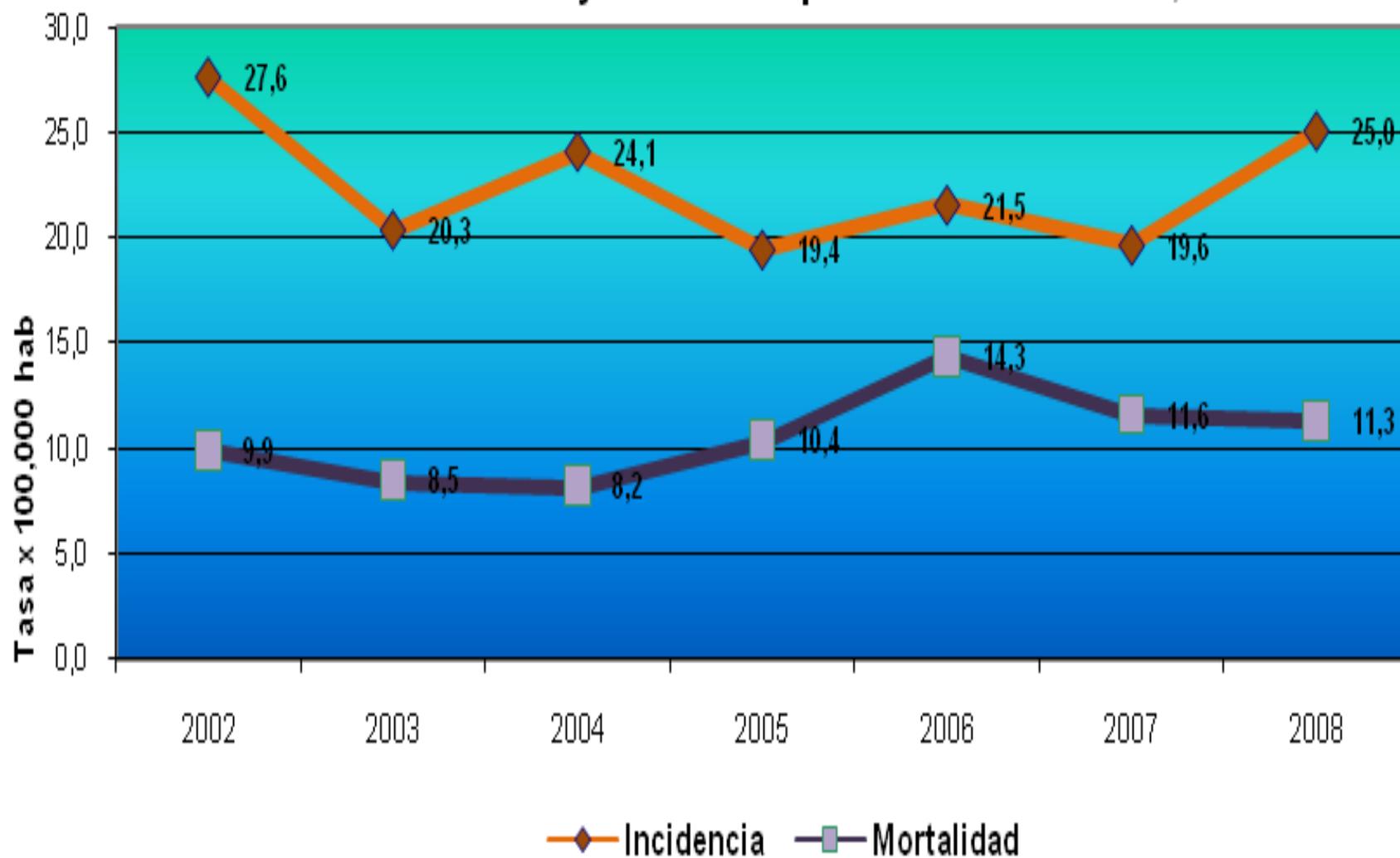
PREVALENCIA DEL VIH EN **MTS** EN COLOMBIA

Ciudad	Prevalencia
Barranquilla	4,54%
Medellín	1,19%
Cali	1,67%
Bucaramanga	3,82%

Fuente: MSPS, UNFPA, OPS, SDS Btá, 2010



Grafica 11. Incidencia y mortalidad por VIH-SIDA. Pereira, 2002 - 2008



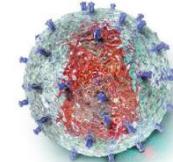


Tabla 25. Primeras causas de mortalidad. Pereira, 2008

ORDEN	CAUSA	2008		
		No	TASA*	%
1	Tumores malignos	521	115,1	19,2
2	Homicidios	379	83,7	14,0
3	Infarto agudo del miocardio	347	76,6	12,8
4	Enfermedad pulmonar obstructiva crónica	198	43,7	7,3
5	Hemorragia Intraencefálica no especificada	118	26,1	4,4
6	Diabetes mellitus	97	21,4	3,6
7	Accidente de Transito	61	13,5	2,2
8	Sida	51	11,3	1,9
9	Otras enfer. cerebrovasculares no especificadas	38	8,4	1,4
10	Insuficiencia renal crónica no especificada	34	7,5	1,3
	Resto de Causas	1348		49,7
	Total	3193		117,7
	Población		451645	
	* Tasa x 100000 hab			

Jimenez et al. **Características epidemiológicas de pacientes VIH – SIDA.** *Revista Médica de Risaralda* 2001; 7(1):19-23.

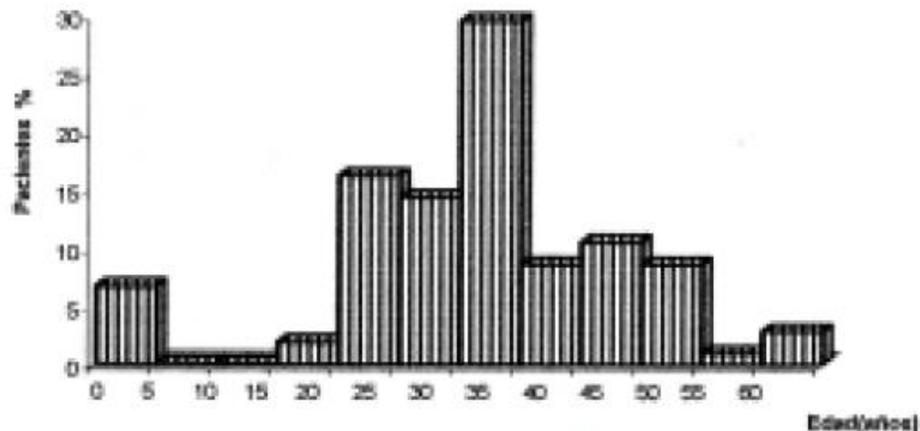


Figura 1. Porcentaje pacientes VIH/SIDA según edad. Empresa Social del Estado. Hospital Universitario San Jorge. Pereira 1991-1999.

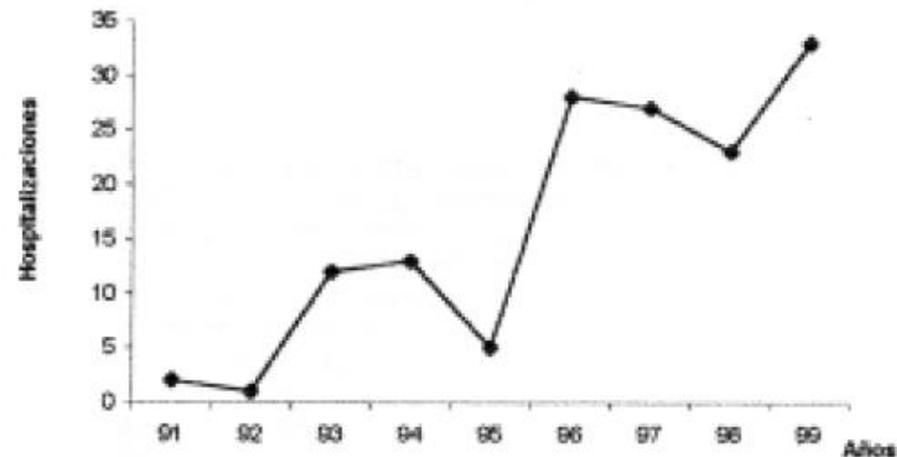


Figura 2. Hospitalizaciones por año pacientes VIH/SIDA. Empresa Social del Estado. Hospital Universitario San Jorge. Pereira 1991-1999.

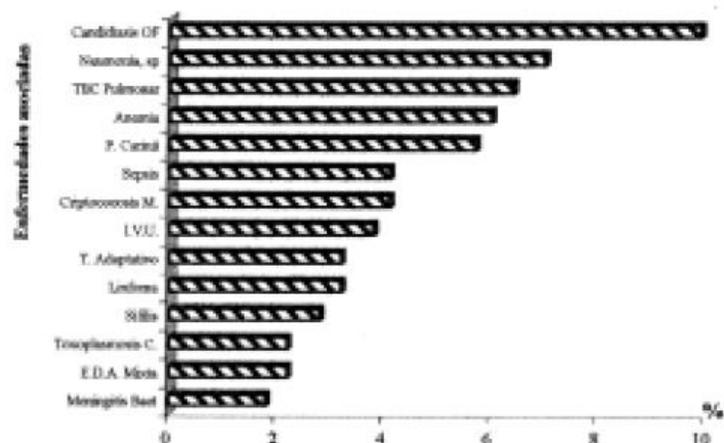


Figura 4. Porcentaje enfermedades asociadas a pacientes VIH/SIDA según año. Empresa Social del Estado. Hospital Universitario San Jorge. Pereira 1991-1999.

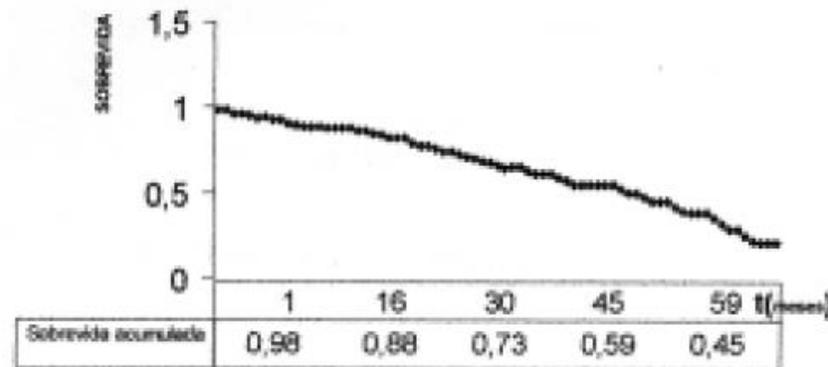


Figura 5. Sobrevida en los pacientes VIH/SIDA según año. Empresa Social del Estado. Hospital Universitario San Jorge. Pereira 1991-1999.



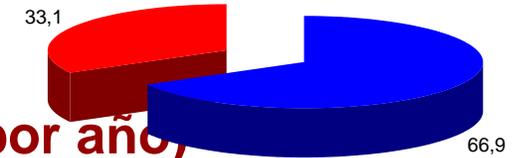
Resultados

- **Se evaluaron 305 casos de pacientes con VIH/SIDA:**

- 2010: 146 casos; incidencia de 31,94 casos/100.000 hab.
- 2011: 159 casos; incidencia de 34,59 casos/100.000 hab.

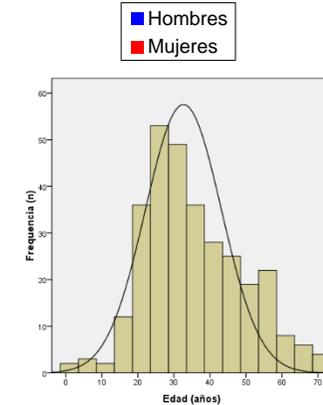
- **Distribución por género:**

- 66,9% hombres (♂) y 33,1% mujeres (♀) (sd por año)



- **Edad:**

- Promedio: 35,5 años ($\pm 13,8$) (sd por año)
- ♂: 37,25 años; ♀: 32,08 años ($p=0,002$)
 - 2010: ♂: 36,92 años; ♀: 30,50 años ($p=0,01$)
 - 2011: ♂: 37,59 años; ♀: 33,30 años ($p=0,06$)

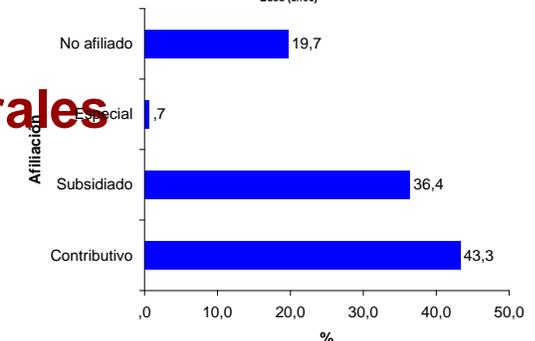


- **Procedencia:**

- 96,7% de zonas urbanas y 3,3% de zonas rurales

- **Afiliación al SGSSS:**

- 80,3% afiliado a seguridad social, 19,7% no.





Resultados

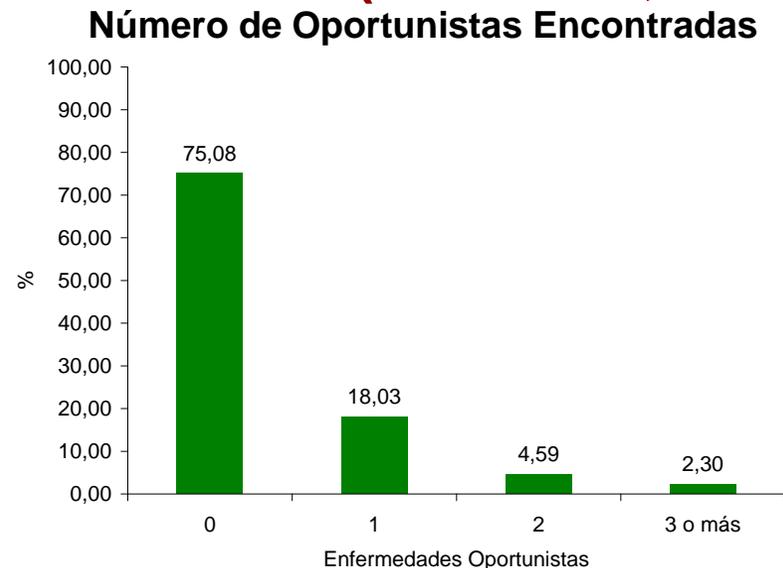
- **Del total (n=305)**

- **Ocurrencia de oportunistas:**

- **24,9% (IC95% 19,9-29,9) presentaron ≥ 1 EO**
- **75,08% (IC95% 70,1-80,1) no**

- **Hospitalización:**

- **37% (IC95% 31,5-42,6) se hospitalizaron**



Hospitalización según ocurrencia de EO

Ocurrencia de EO	Hospitalización		Total
	Sí	No	
Sí	n	49	76
	%	64,5%	100,0%
No	n	64	229
	%	27,9%	100,0%
Total	n	113	305
	%	37,0%	100,0%

$\chi^2=32,642$; $p<0,001$; OR=4,679 (IC95% 2,696-8,120)



Resultados

● Variables asociadas a la ocurrencia de EO

○ Ocurrencia de oportunistas fue mayor en sujetos ≥ 35 años

● 30,5% (IC95% 22,5-38,5) (OR=1,742; IC95% 1,032-2,941).

○ Ocurrencia de oportunistas fue mayor en aquellos no afiliados al SGSSS

● 36,7% (IC95% 23,6-49,7) (OR=2,048; IC95% 1,117-3,753).

Ocurrencia de EO según grupos de Edad

Edad (años)	Ocurrencia de EO		Total	
	Sí	No		
≥ 35	n	43	98	141
	%	30,5%	69,5%	100,0%
<35	n	33	131	164
	%	20,1%	79,9%	100,0%
Total	n	76	229	305
	%	24,9%	75,1%	100,0%

$\chi^2=4,362$; $p=0,037$; OR=1,742 (IC95% 1,032-2,941)

Ocurrencia de EO según afiliación a SGSSS

Afiliación	Ocurrencia de EO		Total	
	Sí	No		
No	n	22	38	60
	%	36,7%	63,3%	100,0%
Sí	n	54	191	245
	%	22,0%	78,0%	100,0%
Total	n	76	229	305
	%	24,9%	75,1%	100,0%

$\chi^2=5,511$; $p=0,019$; OR=2,048 (IC95% 1,117-3,753)



Resultados



Enfermedades Oportunistas Encontradas



Resultados

● Letalidad

- 7,2%, mayor en aquellos con EO (OR=6,3; IC95% 2,5-15,8)

Muerte según ocurrencia de EO

Ocurrencia de EO	Muerte		Total
	Sí	No	
Sí	n	14	75
	%	18,7%	100,0%
No	n	8	229
	%	3,5%	100,0%
Total	n	22	305
	%	7,2%	100,0%

$\chi^2=19,376$; $p<0,001$; OR=6,34 (IC95% 2,543-15,810)



Resultados

- **Algunas oportunistas específicas fueron significativamente más frecuentes en aquellos ≥ 35 años**

Enfermedad Oportunista (%)	Edad (años)		OR	IC95%
	≥ 35	< 35		
Candidiasis esofágica	6,4	1,2	5,556	1,182-2,632
Síndrome de emaciación	5,0	0,6	8,547	1,035-71,429
Candidiasis de vías aéreas	4,3	0,0	1,045	1,009-1,082
TB extrapulmonar	2,8	0,0	1,029	1,001-1,059



Resultados

- **Algunas oportunistas específicas conllevaron significativamente más a la muerte de los pacientes.**

		Muerte (%)	OR	IC95%
Septicemia recurrente por <i>Salmonella</i>	Sí	66,7	28,100	2,442-323,34
	No	6,6		
Neumonía por <i>P. jirovecii</i>	Sí	50,0	14,684	2,774-77,725
	No	6,4		
Candidiasis esofágica	Sí	36,4	8,698	2,329-32,490
	No	6,2		
Meningitis	Sí	37,5	8,747	1,942-39,397
	No	6,4		
Candidiasis de la vía aérea	Sí	33,3	6,925	1,195-40,130
	No	6,7		
Leucoencefalopatía multifocal	Sí	50,0	13,381	0,808-221,601
	No	7,0		

- **Solo la meningitis fue significativa en el análisis multivariado, para la muerte: $OR_{ajustado} = 7,738$ (IC95% 1,368-43,777).**



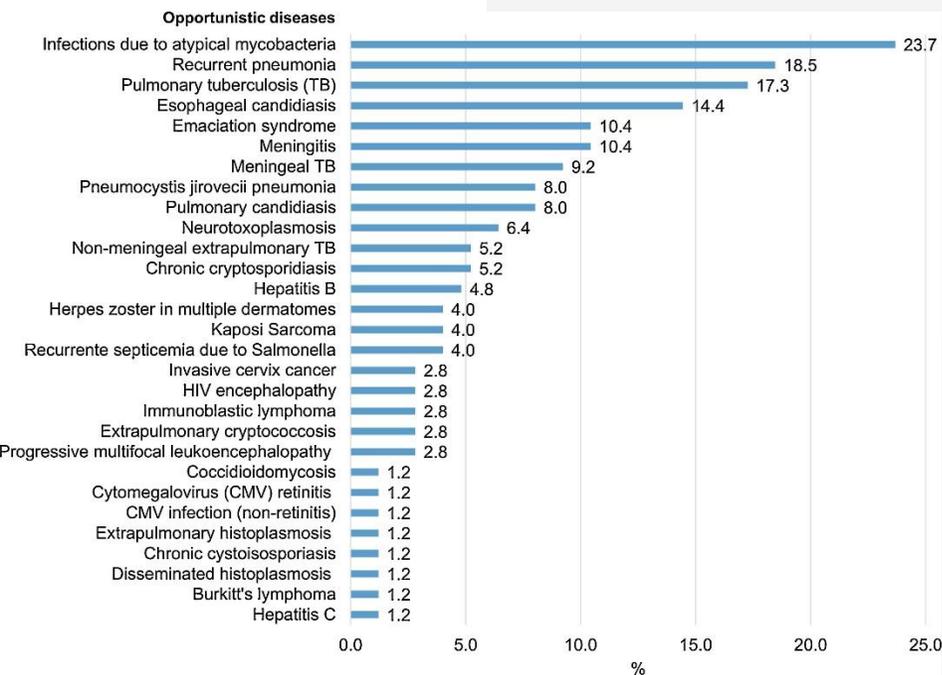
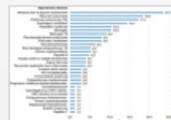
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Ethical approval
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Letter to the Editor

Epidemiology of opportunistic diseases in AIDS patients from Pereira municipality, Colombia, 2010–2011

Paola A. Saldarriaga-Arenas^{a, b}

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Morbidity and mortality related to acquired immunodeficiency syndrome (AIDS)-defining opportunistic diseases (ODs) have been significantly reduced since the introduction of highly active anti-retroviral therapy (HAART). However, they still represented a significant epidemiological burden among patients with AIDS in some developing countries [1] and [2]. Even more, there is few recent data, particularly population-based, about the prevalence and factors associated to ODs in AIDS patients of some countries of South America, with limited access to HAART, such as Colombia [3] and [4]. Surveillance studies on it should be frequently done. According to the World Health Organization, this country is in the list of nations with 40–59% of eligible people receiving HAART at the end of 2011 [4].

For these reasons we assessed the prevalence of ODs in the population of AIDS patients living and attended in the municipality of Pereira, the capital area of Risaralda department, in western Colombia, during 2010–2011. This population is included in the HIV control program of Pereira municipality. Pereira (459.667 pop. for 2011) is one of the municipalities with highest incidence of HIV/AIDS in the country, 34.6 cases/100,000 pop. for 2011, with a significant increase in the last 6 years (2006–2011) [5].

Patients were diagnosed based on epidemiological, clinical and serological confirmation (ELISA HIV-1 and HIV-2 tests and Western-blot, with voluntary counseling and testing). Data was collected through the Epidemiological Surveillance System (SIVIGILA), HIV/AIDS trimester program reports and through HIV/AIDS treatment cohort reports. Opportunistic diseases were clinically, microbiologically and pathologically diagnosed. Collected data was compiled in Excel and then analyzed with SPSS v.17.0[®].

