UnaSalud
Con enfoque a Inocuidad Alimentaria

Andrés Cartín-Rojas
DMV, MS

Concepto OneHealth

La mejora de la salud y el bienestar a través de: (i) la prevención de riesgos y la mitigación de sus efectos originados en la interfaz entre humanos, animales y ecosistemas, y (ii) promover un enfoque intersectorial de colaboración en materia de salud, como un cambio sistémico en la perspectiva en torno a la gestión de riesgos emergentes y re-emergentes.

Okello et al., 2011

El esfuerzo integrador de múltiples disciplinas trabajando localmente. a nivel nacional y mundial para lograr una salud óptima para las personas, los animales y el medio ambiente.

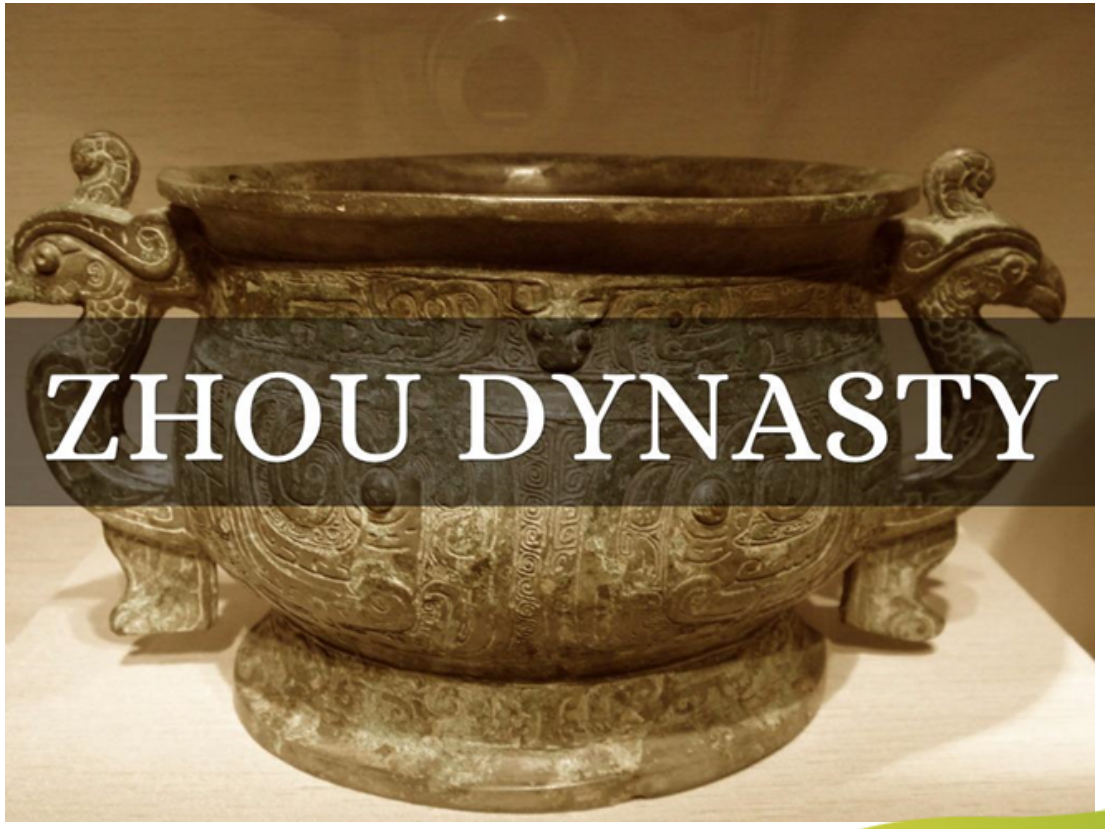
AVMA, 2008

Antecedentes del término

´´Los fundamentos de la medicina veterinaria son tan completos y sutiles como los de la medicina humana y no es posible colocar uno encima del otro´´

(Dinastía Zhou).
China, Asia

Siglos XI-XIII, Integración de un sistema único de salud



Antecedentes del término

´´Entre la medicina humana y animal no existen líneas divisorias, ni debería haberlo. El objeto de estudio es distinto, pero la experiencia obtenida constituye la base de toda ciencia médica´´ (Robert Virchow, 1821-1902).

Alemania, Europa

Siglo XIX, creciente interés en patología comparada

Acuña el término ´´Una Patología´´

Define zoonosis

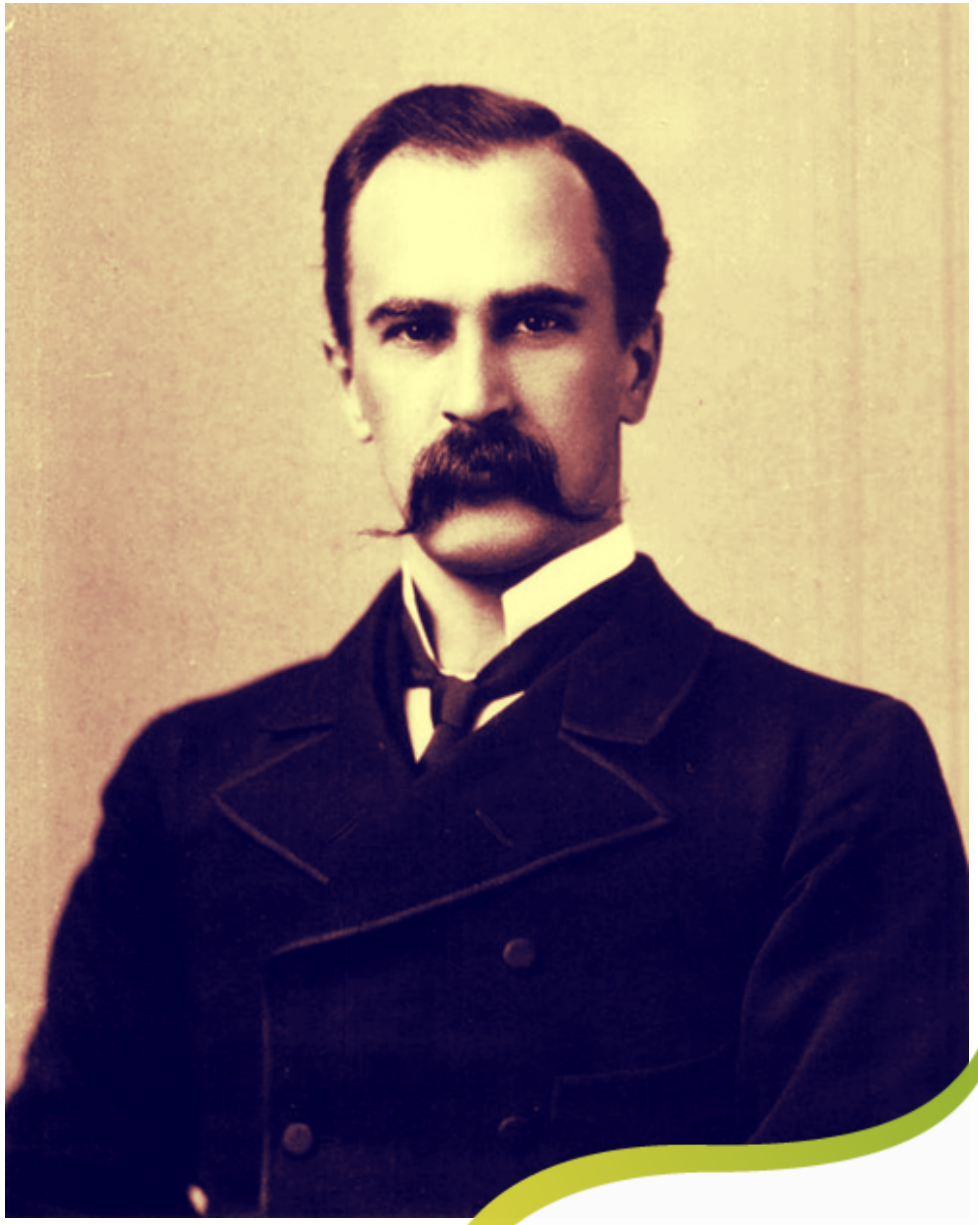
Antecedentes del término

´´La medicina veterinaria y humana se complementan una a la otra y deben ser consideradas como una sola medicina´´

(William Osler, 1849-1919).

USA, América

Padre de la patología norteamericana

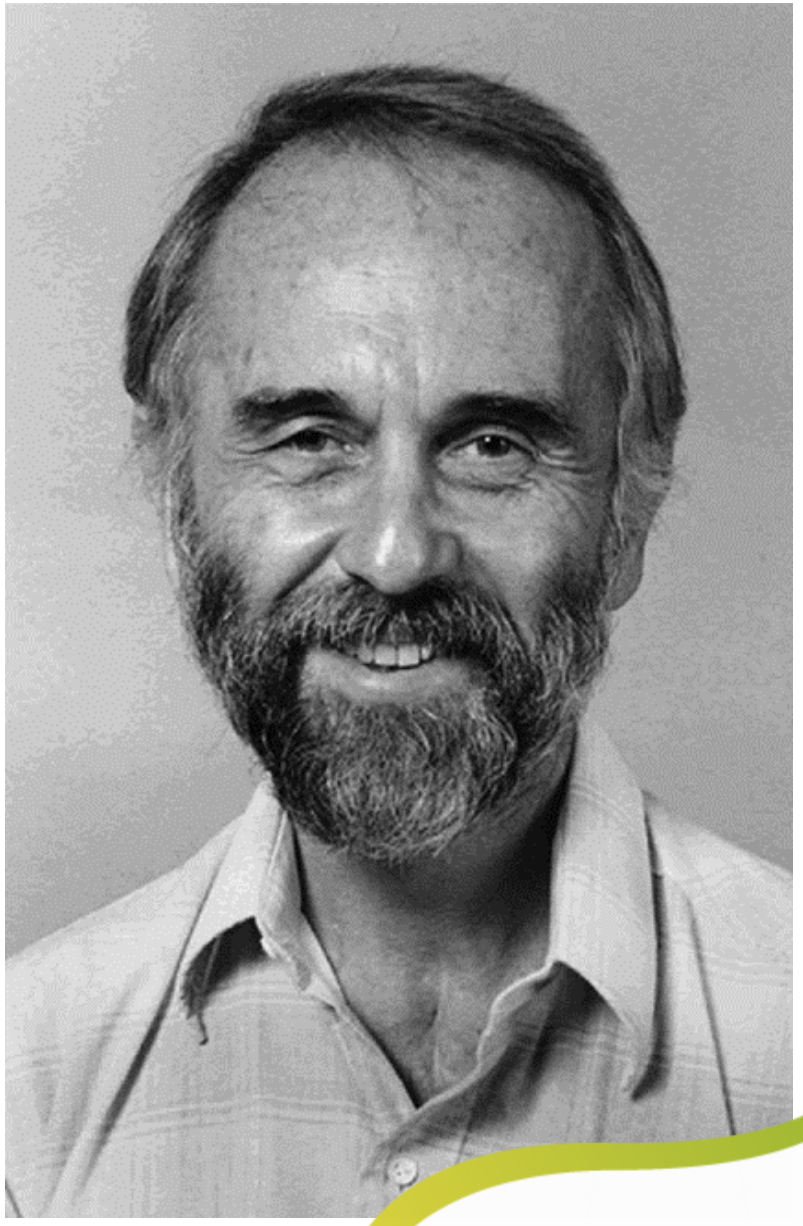


Antecedentes del término

´´Las necesidades críticas del hombre incluyen el combate de las enfermedades, asegurando suficiente alimentación, adecuada calidad ambiental y una sociedad en la que prevalezcan los valores humanos´´

(Calvin Schwabe, 1927-2006).
USA, América

Siglo XX, el término lo acuña este autor (inicialmente como OneMedicine) en su libro Medicina Veterinaria y Salud Humana



Fuente: Hristovski, M., Cvetkovik, A., Cvetkovik, I., & Dukoska, V. (2010). Concept of one Health-a new professional imperative. Macedonian Journal of Medical Sciences, 3(3), 229-232.

UnaSalud

- El concepto de UnaSalud indica que tanto animales como humanos y los ecosistemas que habitan, subsisten en una marcada y dinámica interconexión.
- En la era moderna el concepto fue ampliamente empleado por el Dr. Calvin Schwabe (1927-2006).

Fuente: Hristovski, M., Cvetkovik, A., Cvetkovik, I., & Dukoska, V. (2010). Concept of one Health-a new professional imperative. Macedonian Journal of Medical Sciences, 3(3), 229-232.

UnaSalud

Interface:

- Animal,
- Ecosistema, y
- Ser humano.

UnaSalud

SALUD:
Estado en que el ser orgánico ejerce normalmente todas sus funciones (RAE, 2018).

SALUD PÚBLICA:
Conjunto de condiciones mínimas de salubridad de una población determinada, que los poderes públicos tienen la obligación de garantizar y proteger (RAE, 2018).

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ENFERMEDAD:
Designa la manifestación clínica o patológica de una infección* o infestación*.

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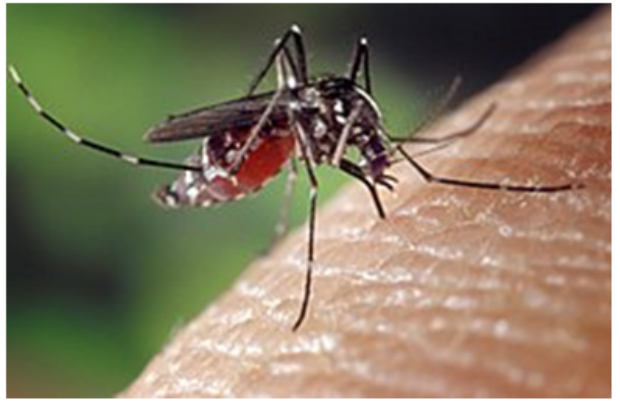
Algunas enfermedades son emergentes y además zoonosis

UnaSalud

De acuerdo al tipo de animal que actúa como fuente de infección, se distinguen dos tipos de zoonosis:

Ciclozoonosis

Metazoonosis



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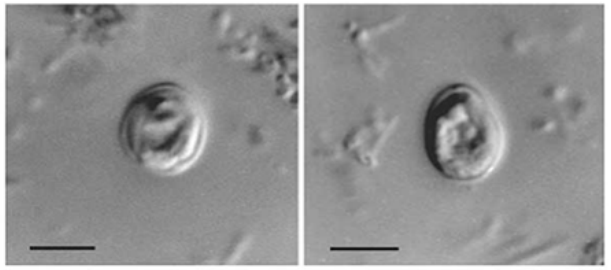
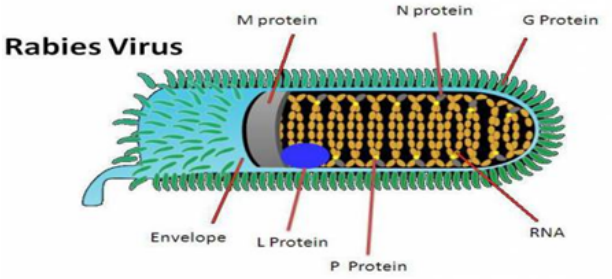
De acuerdo al ciclo biológico del agente patógeno, las zoonosis se dividen en:

Sinantrópicas



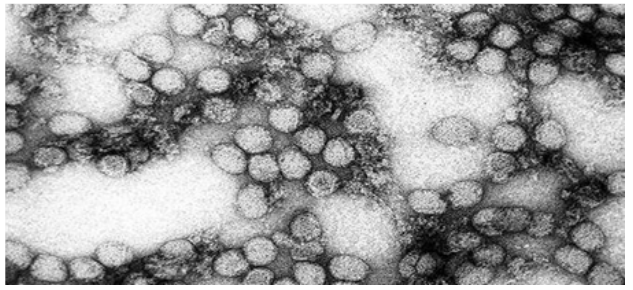
C. parvum

Exoantrópicas



Lyssavirus

Isotrópicas



Flavivirus

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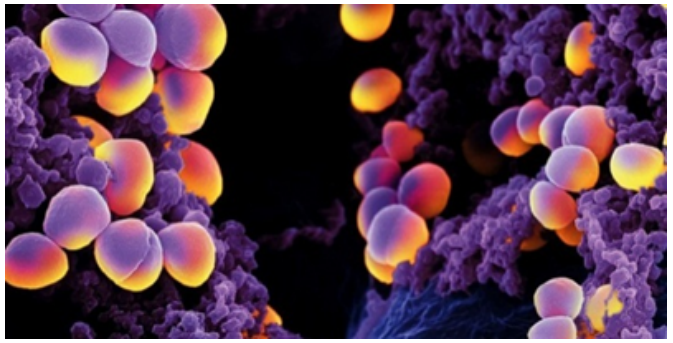
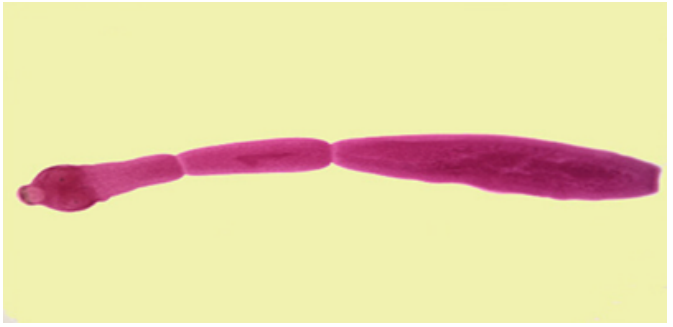
Antropozoonosis



S. aureus

Anfixenosis



E. granulosus

Huésped-Ecosistema-Microorganismo

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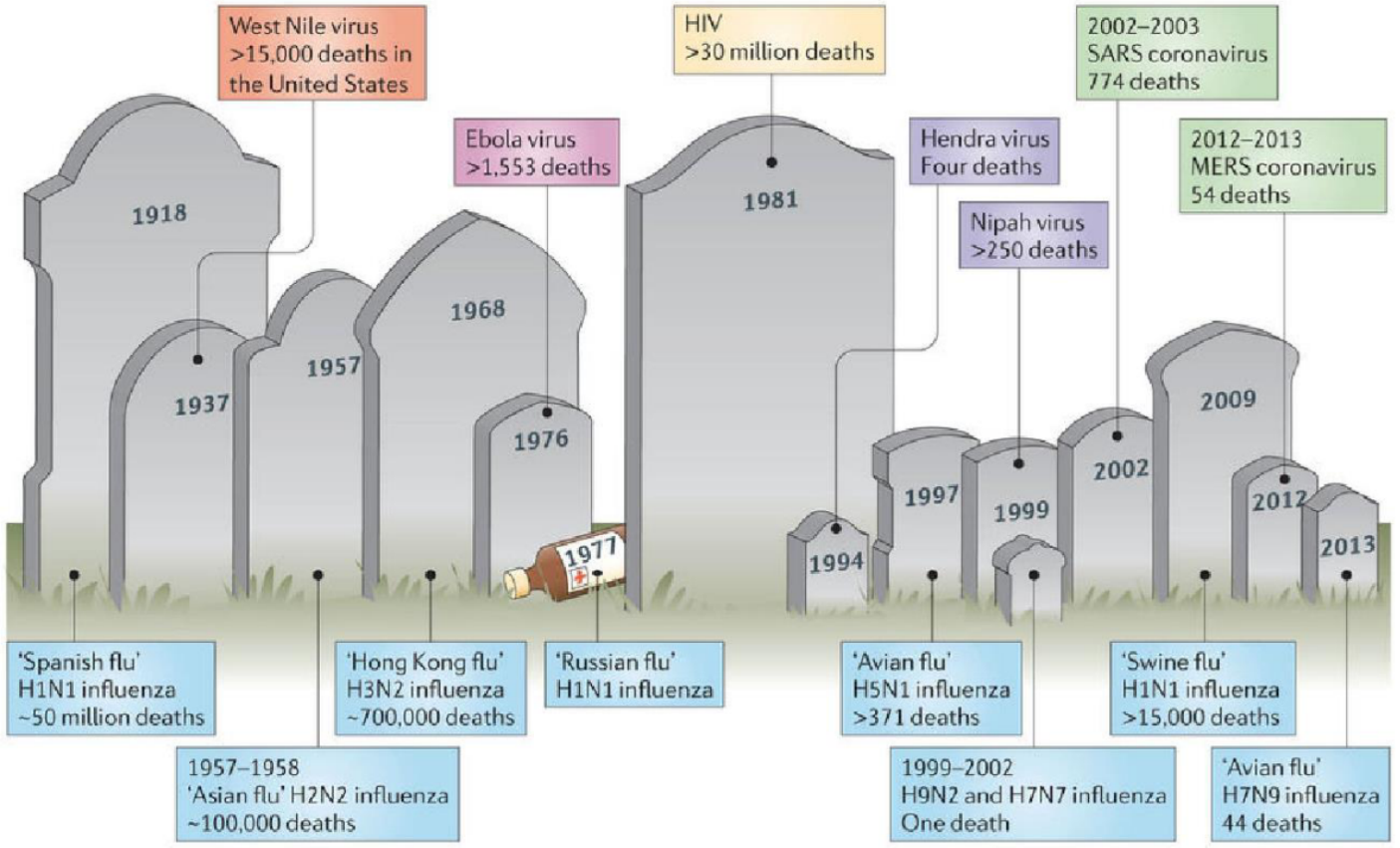


Nature Reviews | Immunology

Studying immunity to zoonotic diseases in the natural host — keeping it real. Andrew G. D. Bean, Michelle L. Baker, Cameron R. Stewart, Christopher Cowled, Celine Deffrasnes, Lin-Fa Wang & John W. Lowenthal. Nature Reviews Immunology. 2013, 13: 851–861. doi:10.1038/nri3551

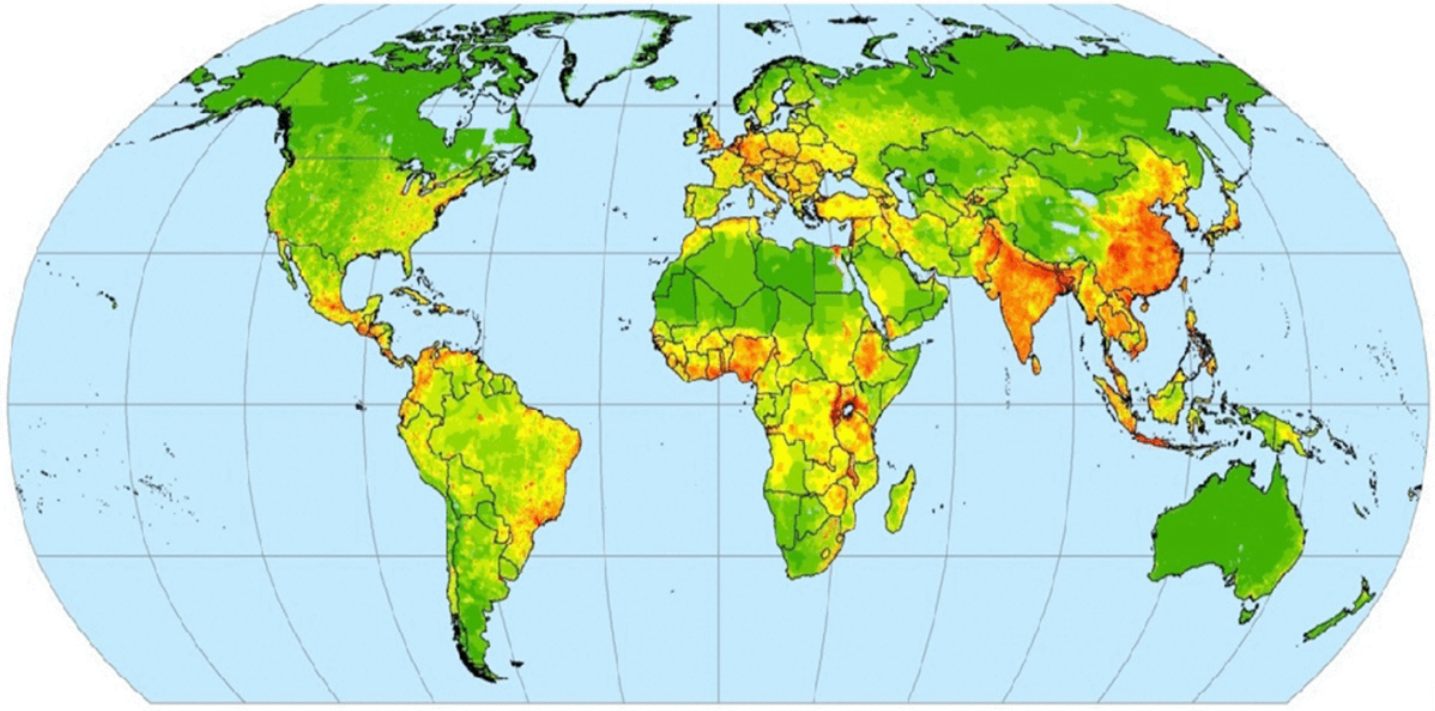
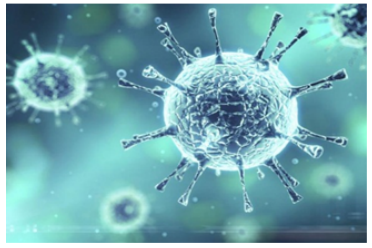


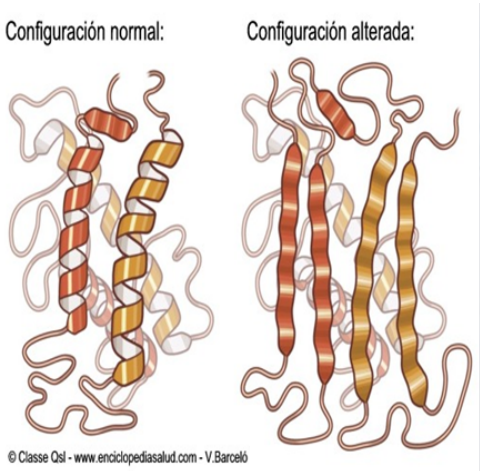
FIGURE WO-27 Global emerging disease "hot spots."
NOTE: Update of model found in Jones et al., 2008, using driver datasets as of 2009 and events as of 2010.
SOURCE: Daszak presentation, 2013 (adapted and updated from Jones et al., 2008).

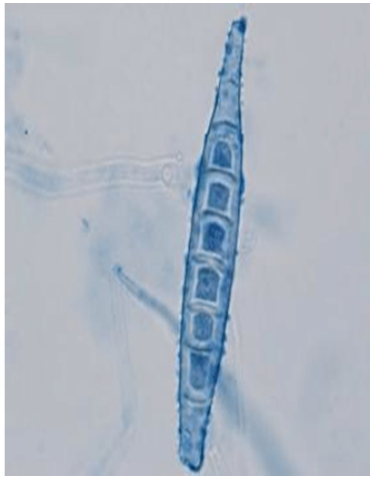
Jones, K. E., Patel, N. G., Levy, M. A., Storeygard, A., Balk, D., Gittleman, J. L., & Daszak, P. (2008). Global trends in emerging infectious diseases. Nature, 451(7181), 990.

Vías de transmisión

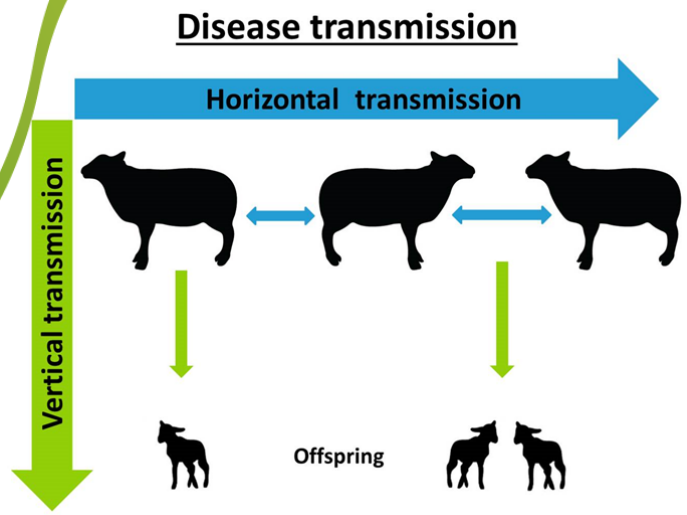
El término mecanismo de transmisión se refiere a la vía o ruta mediante la cual un determinado microorganismo ingresa al huésped, logra burlar los sistemas de defensa endógenos del hospedador y, finalmente, desencadena la enfermedad.

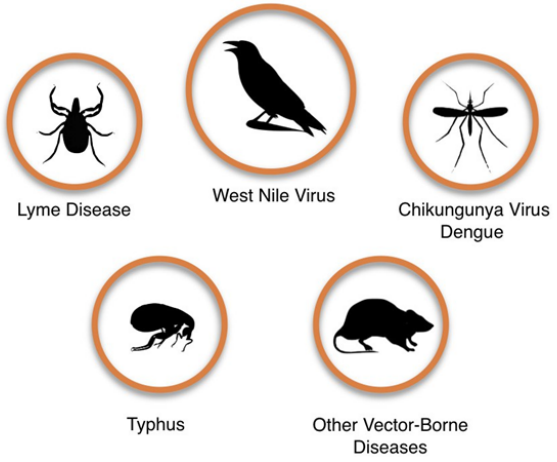






Vías de transmisión





Transmisión directa:

- Horizontal,
- Vertical

Fuente: OMS (2017)

Transmisión indirecta:

- Vectores,
- Latrogénica,
- Alimentos, y
- Fómites.

REVIEW

doi:10.1038/nature09575

Impacts of biodiversity on the emergence and transmission of infectious diseases

Felicia Keesing[1], Lisa K. Belden[2], Peter Daszak[3], Andrew Dobson[4], C. Drew Harvell[5], Robert D. Holt[6], Peter Hudson[7], Anna Jolles[8], Kate E. Jones[9], Charles E. Mitchell[10], Samuel S. Myers[11], Tiffany Bogich[3] & Richard S. Ostfeld[12]

Current unprecedented declines in biodiversity reduce the ability of ecological communities to provide many fundamental ecosystem services. Here we evaluate evidence that reduced biodiversity affects the transmission of infectious diseases of humans, other animals and plants. In principle, loss of biodiversity could either increase or decrease disease transmission. However, mounting evidence indicates that biodiversity loss frequently increases disease transmission. In contrast, areas of naturally high biodiversity may serve as a source pool for new pathogens. Overall, despite many remaining questions, current evidence indicates that preserving intact ecosystems and their endemic biodiversity should generally reduce the prevalence of infectious diseases.

In June 2010, a new organization, the Intergovernmental Science-Policy Platform on Biodiversity and Ecosystem Services (IPBES)—patterned after the Intergovernmental Panel on Climate Change (IPCC)—was established to assess changes to the diversity of life on the Earth and how these changes will affect human well-being[1]. Human well-being would be adversely affected by biodiversity losses if ecosystems with reduced biodiversity are less able to provide the ecosystem services—such as carbon sequestration, nutrient cycling and resistance to drought—on which humans rely. In recent years, a consensus has emerged that ecosystem functions decline as biodiversity is lost[2]. Here we examine how biodiversity affects the transmission and emergence of infectious diseases and evaluate the evidence that reduced disease transmission is an important ecosystem service provided by high biodiversity.

Biodiversity encompasses the diversity of genes, species and ecosystems. Increases in human populations have resulted in an unprecedented and precipitous loss of biodiversity[3]. Current extinction rates are estimated to be at least 100–1,000 times background extinction rates and future extinction rates (over the next 50 years) are estimated to be 10 to 100 times present extinction rates[3]. A large proportion of species in all assessed taxa are currently threatened with extinction (12% of birds, 23% of mammals, 32% of amphibians; 31% of gymnosperms; 33% of corals[4]) and the best estimate of population trends of birds, mammals, amphibians, reptiles and fish indicates that since 1970 global population sizes have declined by almost 30% (ref. 5). Global and local extinction rates of some taxa, particularly microbes, have not been well characterized. For the many organisms that are symbionts of other organisms, extinction of their hosts can cause their extinction too[6]. Collectively, these declines and extinctions are caused by changing the Earth's ecosystems to meet growing demands for food, fresh water, fibre, timber and fuel, and by climate change.

Changes in biodiversity have the potential to affect the risk of infectious disease exposure in plants and animals—including humans—because infectious diseases by definition involve interactions among species. At a minimum, these species include a host and a pathogen; often many more species are involved, including additional hosts, vectors and other organisms with which these species interact. Intriguingly, biodiversity may play a dual role in the emergence and transmission of infectious diseases. On the one hand, high biodiversity may provide a larger potential source of novel pathogens, but on the other hand, biodiversity can reduce further pathogen transmission for both long-established and newly emerging diseases. We first review the effects of biodiversity on the transmission of established diseases and then turn to disease emergence.

Biodiversity and pathogen transmission

Transmission of pathogens between species

Biodiversity loss might affect disease transmission through several mechanisms (Box 1). If the effect of each species on pathogen transmission were entirely idiosyncratic, one would expect that diversity declines would be equally likely to cause a decrease or an increase in disease transmission in the remaining species. However, in recent years, a consistent picture has emerged—biodiversity loss tends to increase pathogen transmission and disease incidence. This pattern occurs across ecological systems that vary in type of pathogen, host, ecosystem and transmission mode (Table 1). As an example, West Nile virus is a mosquito-transmitted virus for which several species of passerine birds act as hosts. Three recent studies detected strong correlations between low bird diversity and increased human risk or incidence of West Nile encephalitis in the United States[7–9]. Communities with low avian diversity tend to be dominated by species that amplify the virus, inducing high infection prevalence in mosquitoes and people, while communities with high avian diversity contain many species that are less competent hosts. For hantavirus pulmonary syndrome, a directly transmitted zoonotic disease, correlational and experimental studies have shown that a lower diversity of small mammals increases the prevalence of hantaviruses in their hosts, thereby increasing risk to humans (Box 2). Diversity has a similar effect for plant diseases, with species losses increasing the transmission of two fungal rust pathogens that infect perennial rye grass and other plant species[10].

[1]Department of Biology, Bard College, Annandale, New York 12504, USA. [2]Department of Biological Sciences, Virginia Tech, Blacksburg, Virginia 24061, USA. [3]EcoHealth Alliance, New York, New York 10001, USA. [4]EEB, Eno Hall, Princeton University, Princeton, New Jersey 08544-3417, USA. [5]Department of Ecology & Evolutionary Biology, Cornell University, Ithaca, New York 14853, USA. [6]Department of Biology, University of Florida, Gainesville, Florida 32611, USA. [7]Center for Infectious Disease Dynamics, Pennsylvania State University, College Station, Pennsylvania 16802, USA. [8]College of Veterinary Medicine, Oregon State University, Corvallis, Oregon 97331-4801, USA. [9]Institute of Zoology, Zoological Society of London, London, NW1 4RY, UK. [10]Department of Biology, The University of North Carolina at Chapel Hill, Chapel Hill, North Carolina 27599, USA. [11]Harvard Medical School, Harvard University, Cambridge, Massachusetts 02138, USA. [12]Cary Institute of Ecosystem Studies, Millbrook, New York 12545, USA.

Nidalidad

Keesing, F., Belden, L. K., Daszak, P., Dobson, A., Harvell, C. D., Holt, R. D., ... & Myers, S. S. (2010). Impacts of biodiversity on the emergence and transmission of infectious diseases. Nature, 468 (7324), 647.

Variación antropogénica

Stephens, P. R., Altizer, S., Smith, K. F., Alonso Aguirre, A., Brown, J. H., Budischak, S. A., ... & Ezenwa, V. O. (2016). The macroecology of infectious diseases: a new perspective on global-scale drivers of pathogen distributions and impacts. Ecology letters, 19(9), 1159-1171.

ECOLOGY LETTERS

Ecology Letters, (2016) doi: 10.1111/ele.12644

REVIEW AND SYNTHESIS

The macroecology of infectious diseases: a new perspective on global-scale drivers of pathogen distributions and impacts

Patrick R. Stephens,[1]* Sonia Altizer,[1] Katherine F. Smith,[2] A. Alonso Aguirre,[3] James H. Brown,[4] Sarah A. Budischak,[1] James E. Byers,[1] Tad A. Dallas,[1] T. Jonathan Davies,[5] John M. Drake,[1] Vanessa O. Ezenwa,[1] Maxwell J. Farrell,[5] John L. Gittleman,[1] Barbara A. Han,[6] Shan Huang,[7] Rebecca A. Hutchinson,[8] Pieter Johnson,[9] Charles L. Nunn,[10] David Onstad,[11] Andrew Park,[1] Gonzalo M. Vazquez-Prokopec,[12] John P. Schmidt,[1] and Robert Poulin[13]

Abstract

Identifying drivers of infectious disease patterns and impacts at the broadest scales of organisation is one of the most crucial challenges for modern science, yet answers to many fundamental questions remain elusive. These include what factors commonly facilitate transmission of pathogens to novel host species, what drives variation in immune investment among host species, and more generally what drives global patterns of parasite diversity and distribution? Here we consider how the perspectives and tools of macroecology, a field that investigates patterns and processes at broad spatial, temporal and taxonomic scales, are expanding scientific understanding of global infectious disease ecology. In particular, emerging approaches are providing new insights about scaling properties across all living taxa, and new strategies for mapping pathogen biodiversity and infection risk. Ultimately, macroecology is establishing a framework to more accurately predict global patterns of infectious disease distribution and emergence.

Keywords

Biodiversity, conservation, disease ecology, infectious diseases, macroecology, pathogens.

Ecology Letters (2016)

INTRODUCTION

Each year infectious diseases cause 9.6 million human deaths globally (Lozano *et al.* 2013) and cost about $120 billion in the U.S. alone (US Centers for Disease Control and Prevention 2008). Most of these diseases have a long history of infecting humans, but growing population size, global connectivity and habitat disruptions collectively boost the chances that a novel infectious disease will emerge in humans (Morse *et al.* 2012). At the same time, infectious diseases have caused die-offs among terrestrial and marine biota ranging from bats and birds to frogs and sea stars (Pedersen *et al.* 2007; Frick *et al.* 2010; Heard *et al.* 2013). The problem of identifying high-risk pathogens ranks among the greatest challenges facing modern science; critical to this effort is the need to predict geographic locations where disease outbreaks are likely to occur, identify the reservoir hosts from which pathogens will emerge, and predict host species at greatest risk of pathogen-mediated declines. A new perspective is needed to develop integrative, broad-scale models that examine determinants and constraints on pathogen distributions and predict their responses to environmental change. Macroecology can provide this perspective.

Macroecologists search for statistical relationships explaining species, abundance, and trait distributions at broad scales of organisation and from both historical and geographical perspectives (Brown 1995). In contrast to traditional experimental and mechanistic approaches in ecological disciplines such as population and community ecology (Johnson *et al.* 2016), macroecological studies generally use existing data to investigate and generate hypotheses. The emergence of macroecology roughly 25 years ago coincided with the new age of informatics that has fostered studies at broad spatial and temporal scales, where localised ecological phenomena transition into the global processes of biogeography, paleobiology and evolutionary diversification (Brown 1995; Burnside

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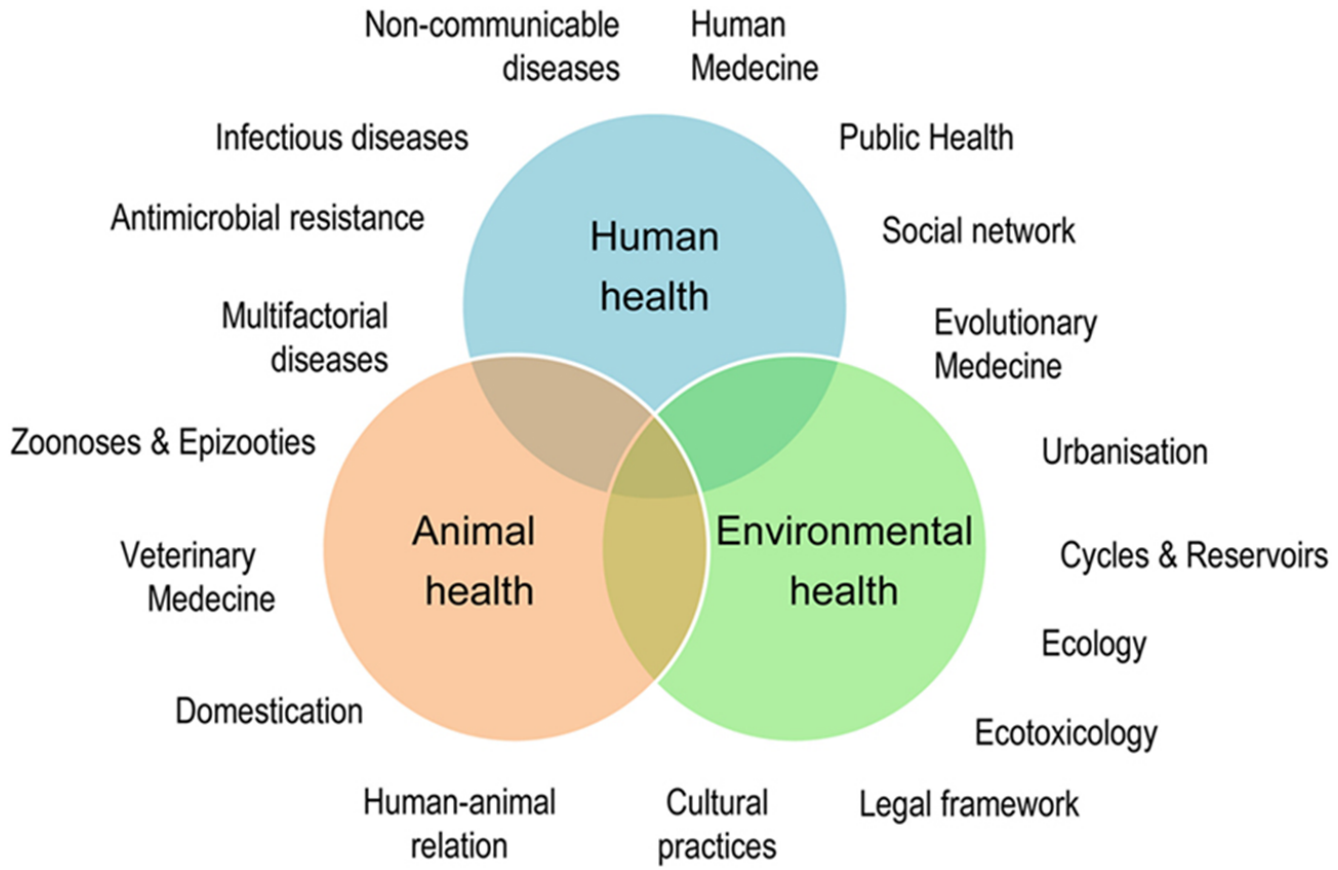
[4]*Department of Biology, University of New Mexico, Albuquerque, NM 87131, USA*
[5]*Department of Biology, McGill University, Montreal, Quebec H3A 0G4, Canada*
[6]*Cary Institute of Ecosystem Studies, Millbrook, New York 12545, USA*
[7]*Senckenberg Biodiversity and Climate Research Centre (BiK-F), Senckenberg Gesellschaft für Naturforschung, Senckenberganlage 25, 60325 Frankfurt, Germany*
[8]*School of Electrical Engineering and Computer Science, Oregon State University, Corvallis, OR 97331, USA*
[9]*Department of Ecology and Evolutionary Biology, University of Colorado, Boulder, CO 80309, USA*
[10]*Biological Sciences, Duke University, Durham, NC 27708, USA*
[11]*ITD Data Analysis and Modelling, DuPont Agricultural Biotechnology, Experimental Station E353/317, Wilmington, DE 19803, USA*
[12]*Department of Environmental Sciences, Emory University, Atlanta, GA 30322, USA*
[13]*Department of Zoology, University of Otago, Dunedin 9054, New Zealand*
**Correspondence: E-mail: prsteph@uga.edu*

Factores promotores



Fuente: Destoumieux-Garzón, D., Mavingui, P., Boetsch, G., Boissier, J., Darriet, F., Duboz, P., ... & Paillard, C. (2018). The one health concept: 10 years old and a long road ahead. Frontiers in Veterinary Science, 5, 14. https://doi.org/10.3389/fvets.2018.00014

Comercio y viajes internacionales

- El aumento de la frecuencia y la velocidad de los viajes locales e internacionales, favorecido por el proceso de globalización promueve la diseminación de microorganismos a una escala global;

Fuente: Destoumieux-Garzón, D., Mavingui, P., Boetsch, G., Boissier, J., Darriet, F., Duboz, P., ... & Paillard, C. (2018). The one health concept: 10 years old and a long road ahead. Frontiers in Veterinary Science, 5, 14. https://doi.org/10.3389/fvets.2018.00014

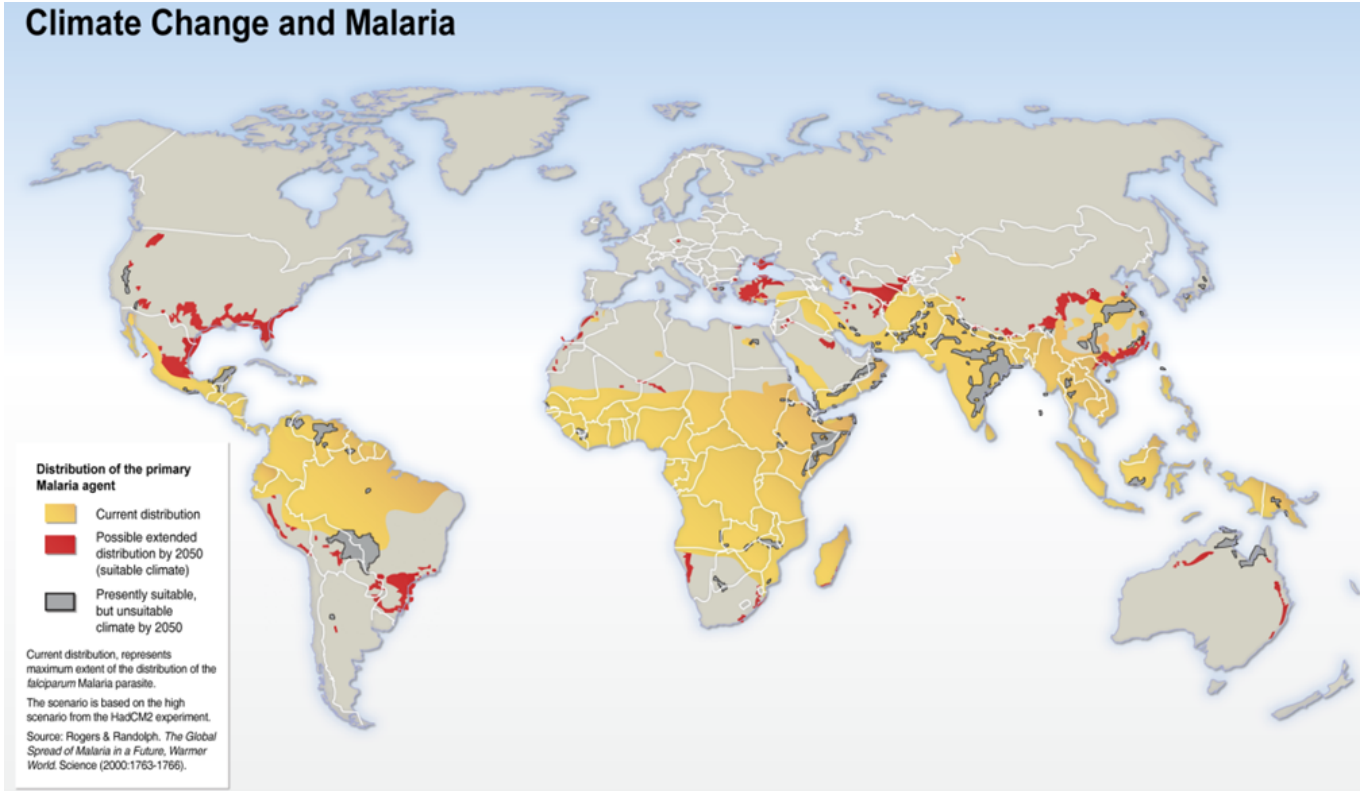
Cambios demográficos

- Migración, aumento de la densidad poblacional, cambios socioculturales;

Hassell, J. M., Begon, M., Ward, M. J., & Fèvre, E. M. (2017). Urbanization and disease emergence: Dynamics at the wildlife–livestock–human interface. Trends in ecology & evolution, 32(1), 55-67.

Cambio climático

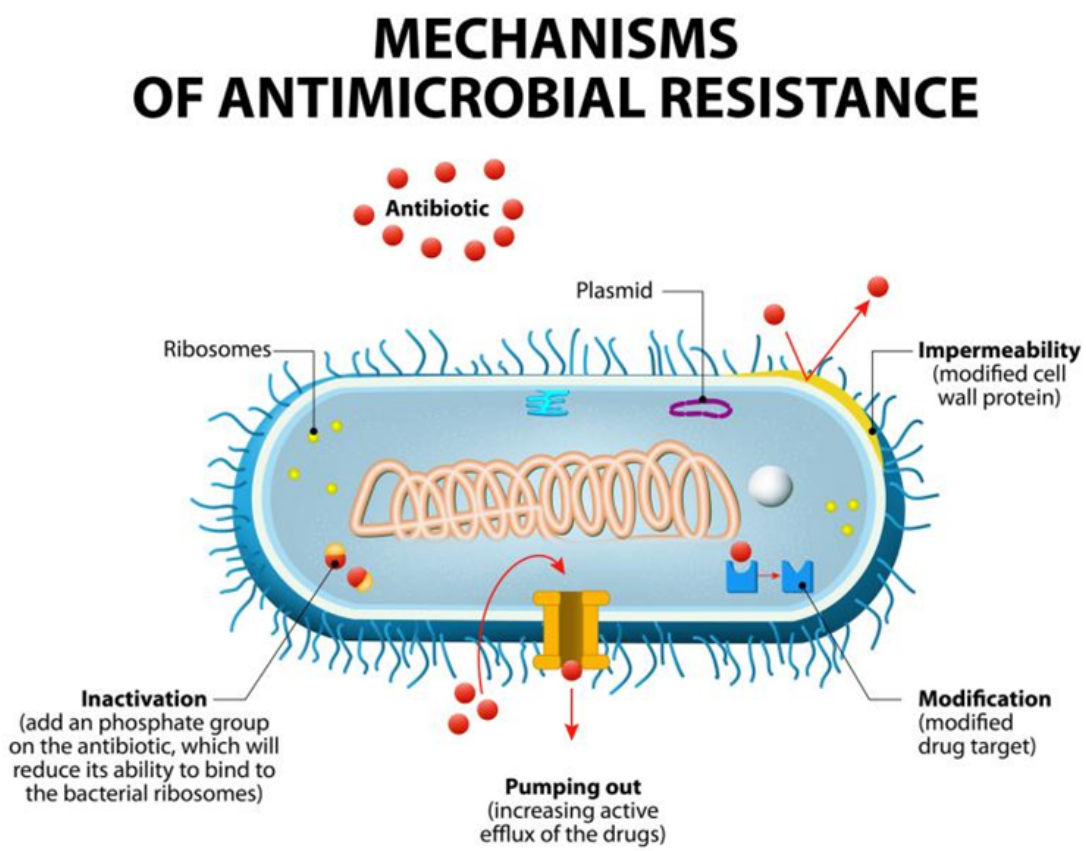
- Lo cual produce cambios en la distribución ecogeográfica de los vectores



De Souza, D. K., Owusu, P. N., & Wilson, M. D. (2012). Impact of climate change on the geographic scope of diseases. In Human and social dimensions of climate change. InTech, Croatia.

Adaptación microbiana

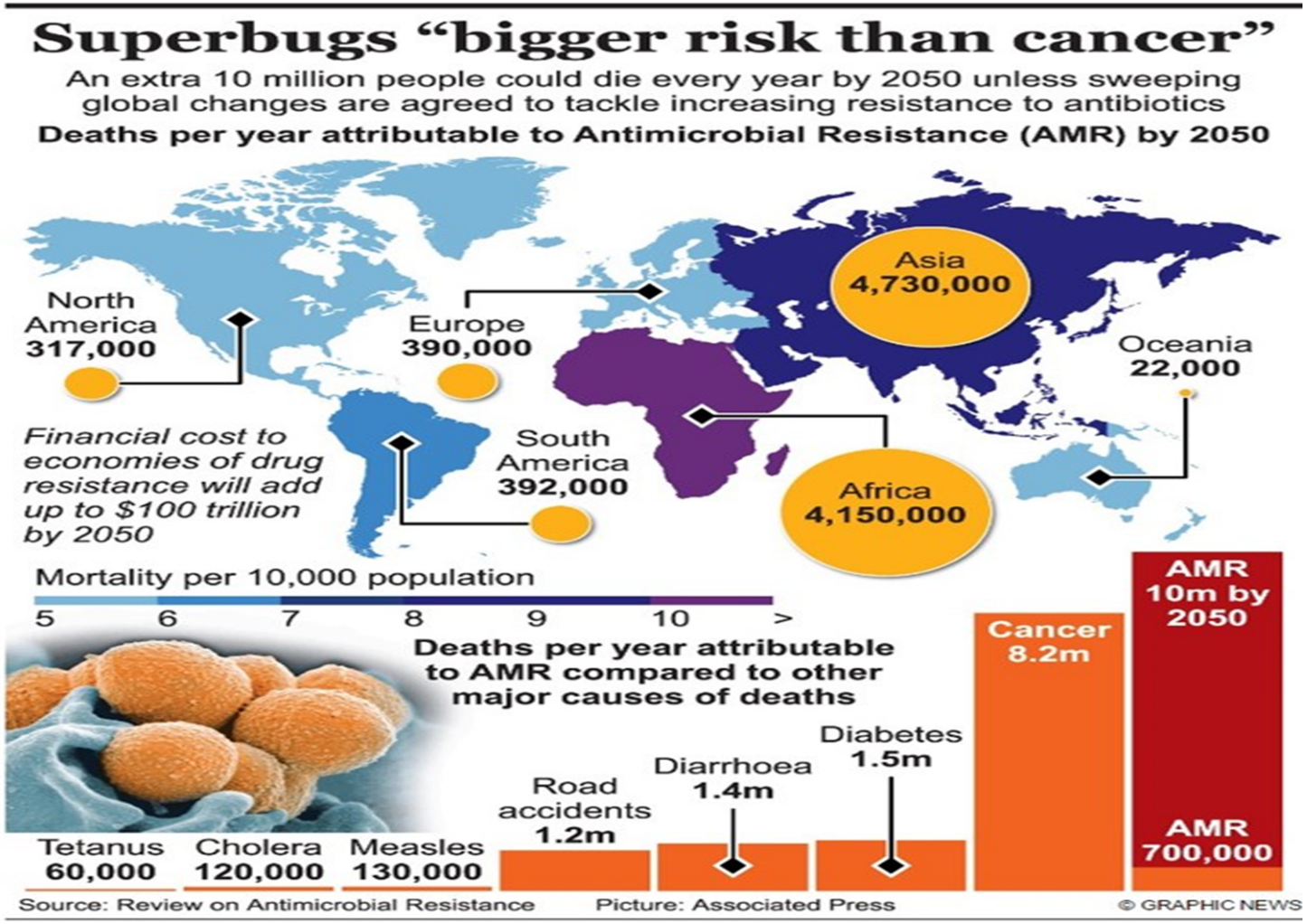
- Resistencia a fármacos antimicrobianos y colonización de nuevos huéspedes.



Adaptación microbiana

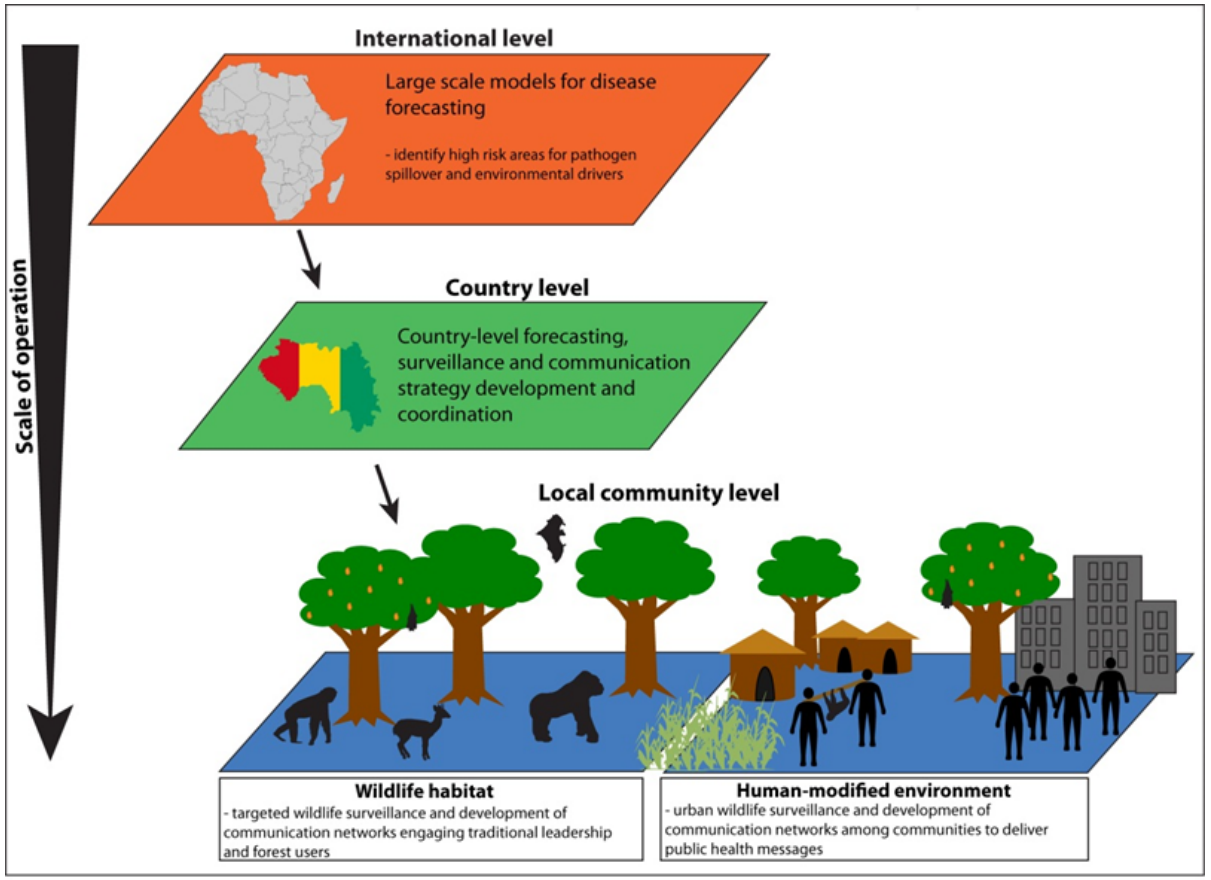
- La transferencia de los genes que codifican la resistencia en una población bacteriana son dispersados a través de tres mecanismos:

1. **Conjugación:** consiste en la transferencia de material genético entre una célula bacteriana donante y una receptora, a través de una estructura conocida como Pili.
2. **Transducción:** es el paso de material genético mediada por un bacteriófago.
3. **Transformación:** ocurre cuando una bacteria logra adquirir e incorporar (recombinar) dentro de su información genética el ADN contenido en un plásmido.


Superbugs "bigger risk than cancer"
An extra 10 million people could die every year by 2050 unless sweeping global changes are agreed to tackle increasing resistance to antibiotics
Deaths per year attributable to Antimicrobial Resistance (AMR) by 2050
North America 317,000
Europe 390,000
Asia 4,730,000
Oceania 22,000
South America 392,000
Africa 4,150,000
Financial cost to economies of drug resistance will add up to $100 trillion by 2050
Mortality per 10,000 population
5 6 7 8 9 10 >
Deaths per year attributable to AMR compared to other major causes of deaths
Tetanus 60,000
Cholera 120,000
Measles 130,000
Road accidents 1.2m
Diarrhoea 1.4m
Diabetes 1.5m
Cancer 8.2m
AMR 10m by 2050
AMR 700,000
Source: Review on Antimicrobial Resistance
Picture: Associated Press
© GRAPHIC NEWS

Degradación de hábitats

- Reducción del hábitat y aumento del contacto con vectores/reservorios silvestres.



Alexander, K. A., Sanderson, C. E., Marathe, M., Lewis, B. L., Rivers, C. M., Shaman, J., ... & Eubank, S. (2015). What factors might have led to the emergence of Ebola in West Africa?. PLoS neglected tropical diseases, 9(6), e0003652.

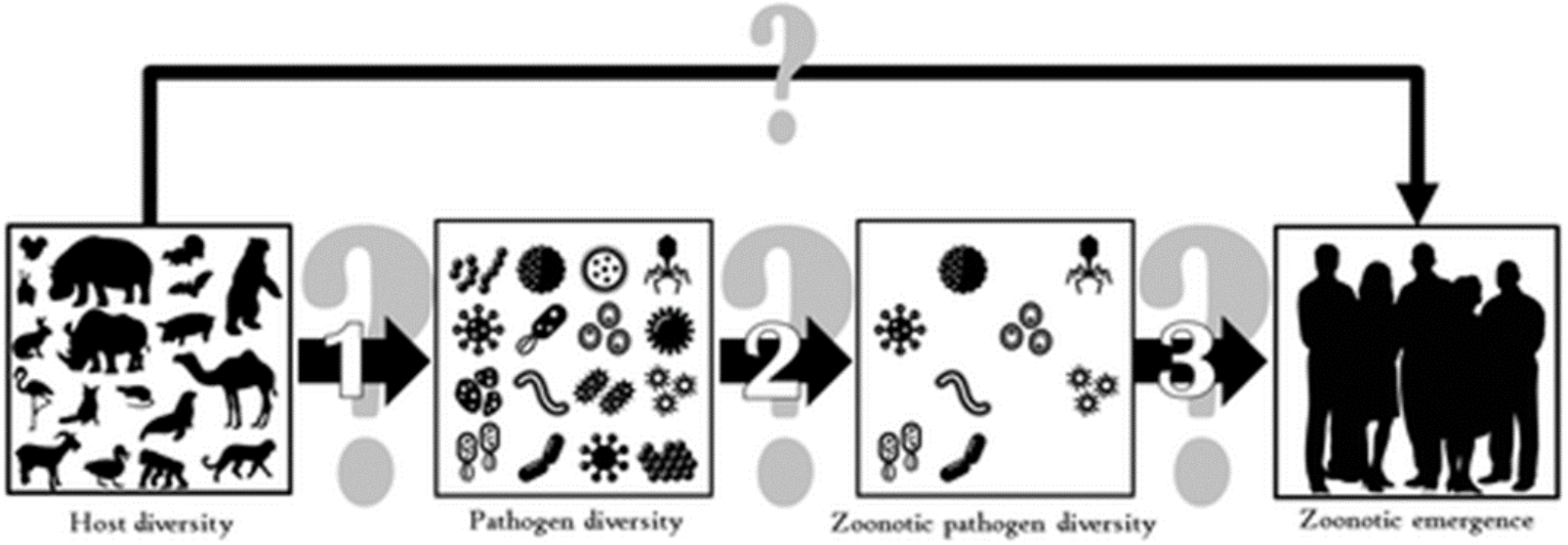
Comercio de fauna

- Como mascotas o fuente de alimento: el tercero más grande del mundo después del tráfico ilegal de drogas y armas.

Karesh, W. B., & Noble, E. (2009). The bushmeat trade: increased opportunities for transmission of zoonotic disease. Mount Sinai Journal of Medicine: A Journal of Translational and Personalized Medicine, 76(5), 429-434.

Especies invasoras

- Introducción de animales silvestres y domésticos a nuevas áreas geográficas donde no son endémicos: aumenta el pool zoonótico y favorece la contaminación microbiana.



Ostfeld, R. S., and F. Keesing. (2017). Is biodiversity bad for your health? Ecosphere 8(3):e01676. 10.1002/ecs2.1676 (OA)

Cambios agropecuarios

- La domesticación animal fue uno de los principales promotores de la evolución microbiana al facilitar las disponibilidad de nuevos huéspedes susceptibles en altas densidades poblacionales, producto de la intensificación y complejidad de los sistemas pecuarios actuales.

Morand, S., McIntyre, K. M., & Baylis, M. (2014). Domesticated animals and human infectious diseases of zoonotic origins: domestication time matters. Infection, Genetics and Evolution, 24, 76-81.

UnaSalud

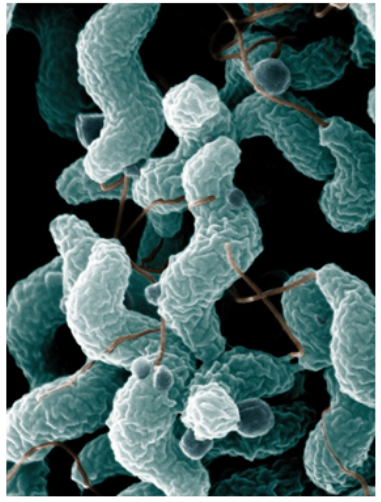
La complejidad de las cadenas productivas permite la amplia distribución de agentes zoonóticos transmitidos por alimentos.

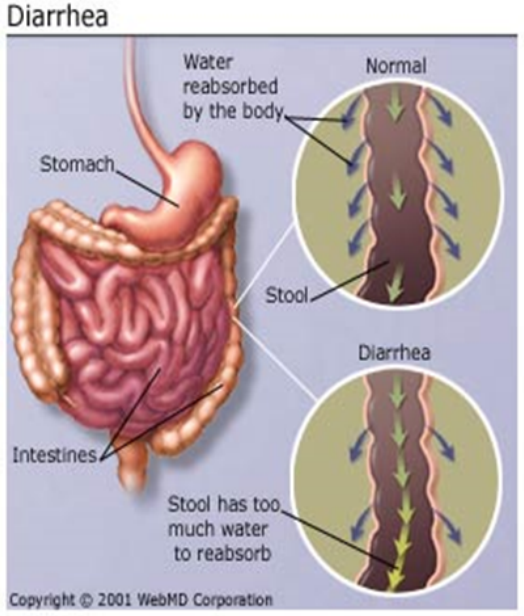
Cerca del 69% de ellos son organismos bacterianos, 9,7% por virus y el 1,8% por parásitos.

Varela, Z., Lavalle, L., & Alvarado, D. (2016). Bacterias causantes de enfermedades transmitidas por alimentos: una mirada en Colombia. Revista Salud Uninorte, 32(1), 105-122.

UnaSalud

Principales alimentos relacionados con ETAs son de origen animal, por tanto un gran número de ETAs son zoonosis de transmisión alimentaria.





Campylobacter spp

Table x: Pathogen X Foodborne Exposure

Percent of Foodborne Cases in a Typical Year

Food Consumed**	lower credible value (5th percentile)	central value (50th percentile)	upper credible value (95th percentile)
Beef	10%	15%	25%
Goat, lamb and other small ruminants' meat	3%	5%	18%
Dairy (milk and milk products)	0%	5%	8%
Pork	25%	35%	50%
Poultry Meat	9%	10%	12%
Vegetables (excluding dried legumes)	4%	15%	20%
Fruits	1%	5%	10%
Nuts	0%	1%	2%
Other foods	1%	5%	10%
		96%	

**Attribute cases of illness to the foods that were already contaminated when they entered the home kitchen or other place of final food preparation. Do not consider cross-contamination in the home kitchen or other place of final food preparation.

3. Start by thinking about how all foodborne cases in a typical year are distributed across foods.

Then think about your 90% cconfidence bounds. Think about factors affecting their width.

4. Mathematically, 50th percentile estimates (medians) may not necessarily add to 100%.

But logically, since they represent the relative contribution of all foods, they will probably add to something close to 100%.

2. Footnotes are to remind you how WHO wants you to define the point of exposure.

5. Remember, the three requested quantiles are unique and should have ascending values.

ALWAYS: 5^{th} %ile < 50^{th} %ile < 95 %ile

Hoffmann, S., Devleesschauwer, B., Aspinall, W., Cooke, R., Corrigan, T., Havelaar, A., Angulo, F., Gibb, H., Kirk, M., Lake, R., Speybroeck, N., Torgerson, N., Hald, T. (2017) Attribution of global foodborne disease to specific foods: Findings from a World Health Organization structured expert elicitation. PLoS one 12, e0183641

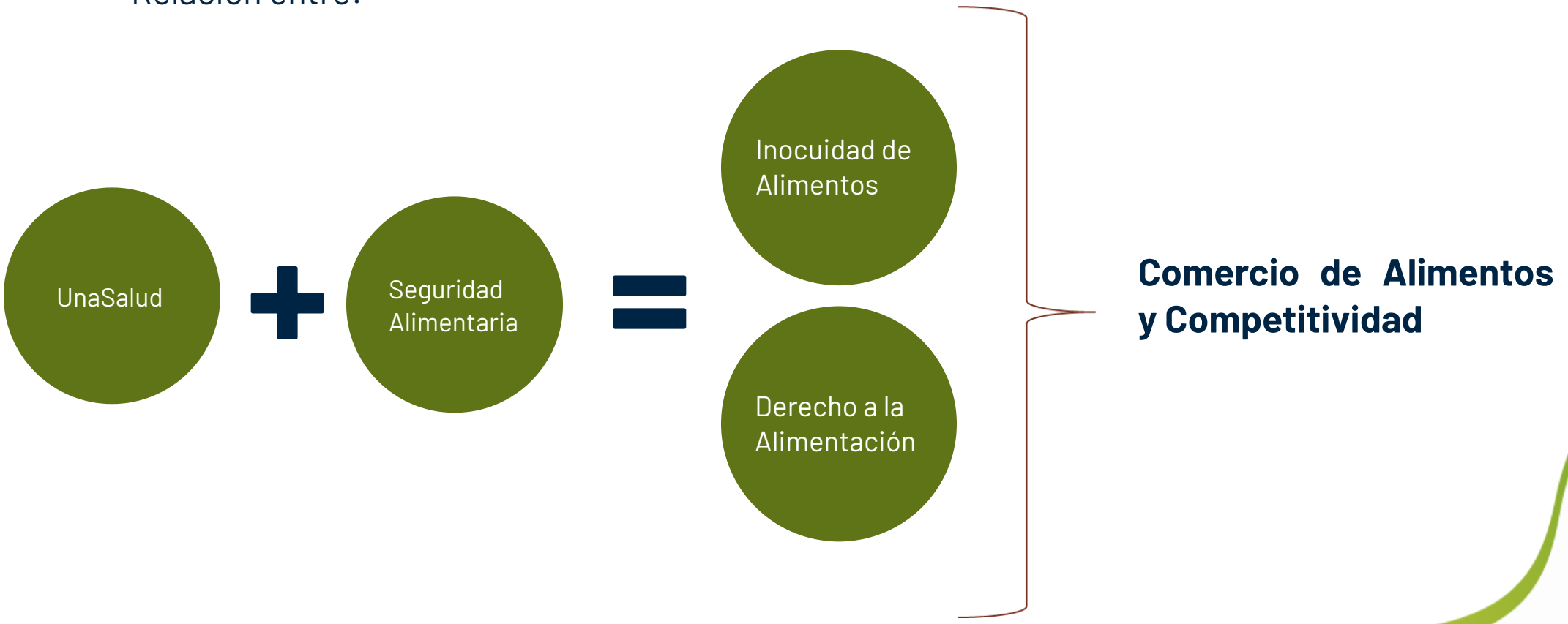
ETAS, datos de OMS

Más de 200 agentes infecciosos	600 millones de personas/año se enferman (1/10)	420 mil personas/año mueren
33 años ajustados perdidos	40% afectados son niños 0-5 años	125.000 muertes/niños de 0-5 años
ETAs generan malnutrición	Importante impacto económico a países	Inocuidad = barrera no arancelaria

Fuente: https://www.who.int/es/news-room/fact-sheets/detail/food-safety

UnaSalud y Seguridad Alimentaria

Relación entre:



Bacterias


PELIGROS DE INOCUIDAD ALIMENTARIA
1. IDENTIFICACIÓN DEL PELIGRO
Biológicos
• Patógenos Alimenticios
• E. coli O157:H7
• Listeria monocytogenes
• Salmonella spp.
• Organismos toxigénicos
• Hongos
• Parásitos
• Virus
• Priones
Químicos
• Toxinas naturales
• Aditivos
• Residuos de Plaguicidas
• Residuos de drogas
• Contaminantes ambientales
• Metales pesados
• Químicos del Envasado
• Alérgenos
Físicos
• Vidrio/ Cristales
• Escorio/ Limo
• Metal/ Joyas
• Plástico
• Piedras
• Conchas/ Semillas
• Madera/ Papel
• Huesos

Lista OMS de patógenos prioritarios para la I+D de nuevos antibióticos

Prioridad 1: CRÍTICA

1. *Acinetobacter baumannii*, resistente a los carbapenémicos
2. *Pseudomonas aeruginosa*, resistente a los carbapenémicos
3. Enterobacteriaceae, resistentes a los carbapenémicos, productoras de ESBL

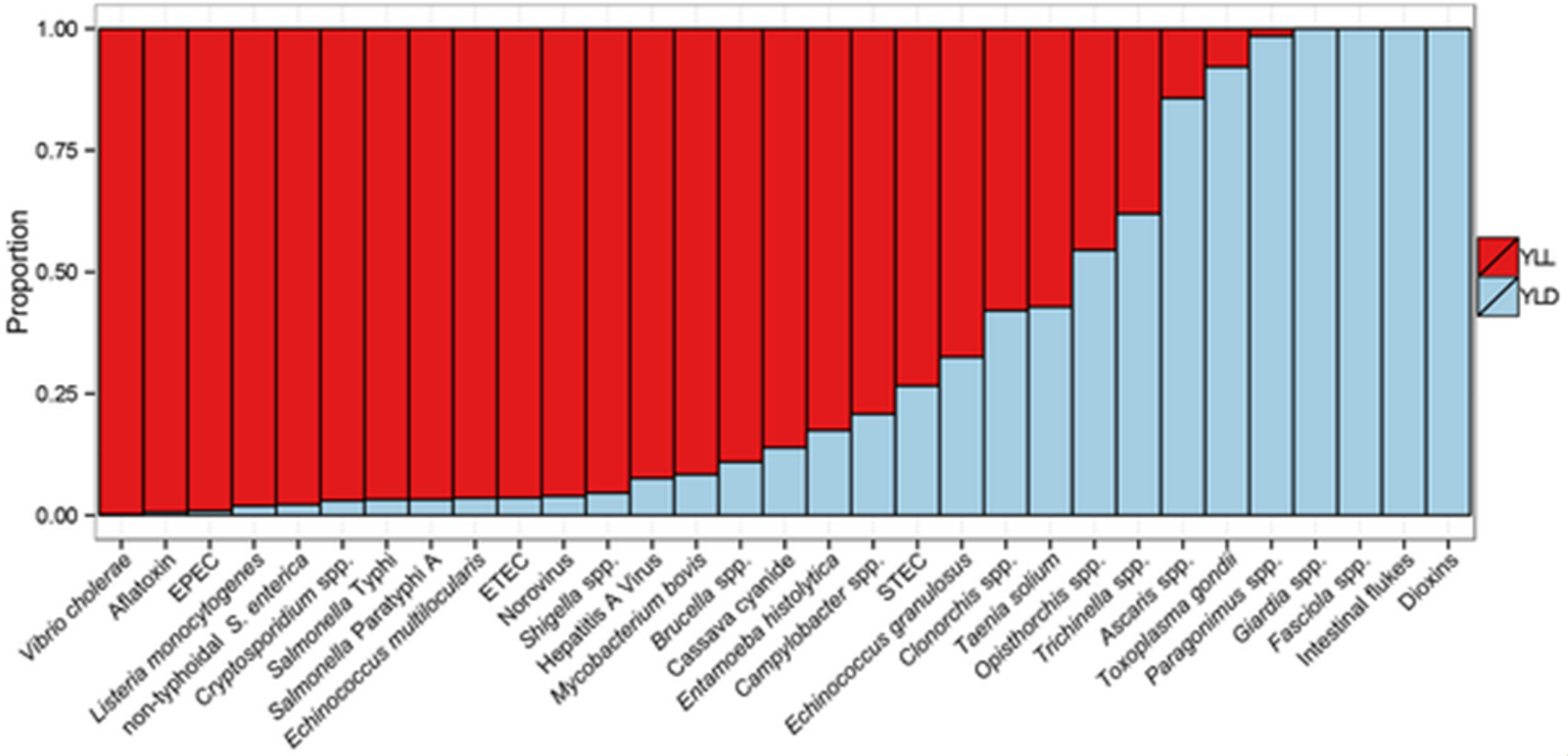
Prioridad 2: ELEVADA

1. *Enterococcus faecium*, resistente a la vancomicina
2. *Staphylococcus aureus*, resistente a la meticilina, con sensibilidad intermedia y resistencia a la vancomicina
3. *Helicobacter pylori*, resistente a la claritromicina
4. *Campylobacter* spp., resistente a las fluoroquinolonas
5. *Salmonellae*, resistentes a las fluoroquinolonas
6. *Neisseria gonorrhoeae*, resistente a la cefalosporina, resistente a las fluoroquinolonas

Prioridad 3: MEDIA

1. *Streptococcus pneumoniae*, sin sensibilidad a la penicilina
2. *Haemophilus influenzae*, resistente a la ampicilina
3. *Shigella* spp., resistente a las fluoroquinolonas

Fuente: http://www.who.int/mediacentre/news/releases/2017/bacteria-antibiotics-needed/es/



Havelaar AH, Kirk MD, Torgerson PR, Gibb HJ, Hald T, et al. (2015) World Health Organization Global Estimates and Regional Comparisons of the Burden of Foodborne Disease in 2010. PLOS Medicine 12(12): e1001923. https://doi.org/10.1371/journal.pmed.1001923

UnaSalud en las políticas públicas

1. Fortalecimiento de convenios público-privados
2. Fortalecimiento de programas de inocuidad a nivel universitario
3. Robustecimiento de los Servicios Zoosanitarios
4. Colaboración académica para el monitoreo de ETAs (observatorios de SAN)
5. Actualización y homologación de legislación sanitaria
6. Monitoreo de Riesgos Emergentes en inocuidad
7. Programas de apoyo a pymes agroalimentarios
8. Establecimiento de programas de rastreabilidad y recall

Edwardsiella spp

***Edwardsiella* spp.**

- Inhibidor lisosímico (ivyEt) → Bloqueo de acción de neutrófilos
- Adhesinas → Fimbrias I y proteínas (eta1 y AIDA), flagelinas (tipos flhDC/fliA/fliC).
- Invasinas → Inv1, ITGB1 o CD29 → 1. Inmunoestimulación (TNF-α, IL-1α, IL-1β, IL-8 y el factor estimulante de las colonias de macrófagos (GM-CFS), y 2. Estimulación T3SS

Liberación de efectores:

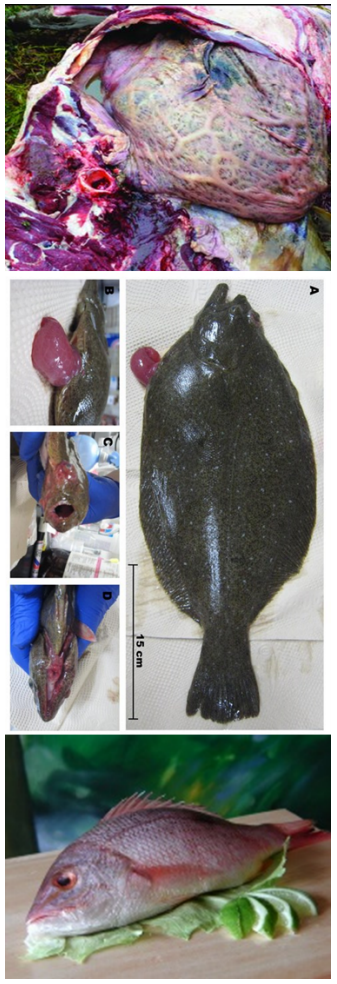
1. Porina (Ompk37),
2. Sialasa (NanA),
3. Hemoaglutinina (HA),
4. Peptidoglicanidasa (mltA),
5. Condroitinasa (glucosaminoglicanidasa),
6. Citolisinas A/B (ClyA/B o EthA/B),
7. EnvZ/OmpR (inductor de porinas)
8. Lipoproteínas (tipo vacJ), importante en la formación de biofilms,
9. Superóxido-dismutasa (sodB),
10. Catalasas (katB/katG), y
11. Regulador de la recaptación de hierro (Fur)

E. tarda
Patógeno emergente

Patogénesis:

Cuadro intestinal: leve (caracterizado por gastroenteritis leve) o grave (enterocolitis o colitis ulcerativa).

Cuadro exoentérico: Sistema Circulatorio (endocarditis), Sistema Osteomuscular (artritis séptica y osteomielitis), sistema Tegumentario (empiema y celulitis), Sistema Reproductivo (abcesos ováricos y endometritis), Sistema Hepatobiliar (abcesos hepáticos, colangitis y colangiocistitis) y Sistema Nervioso (meningitis)



Francisella spp

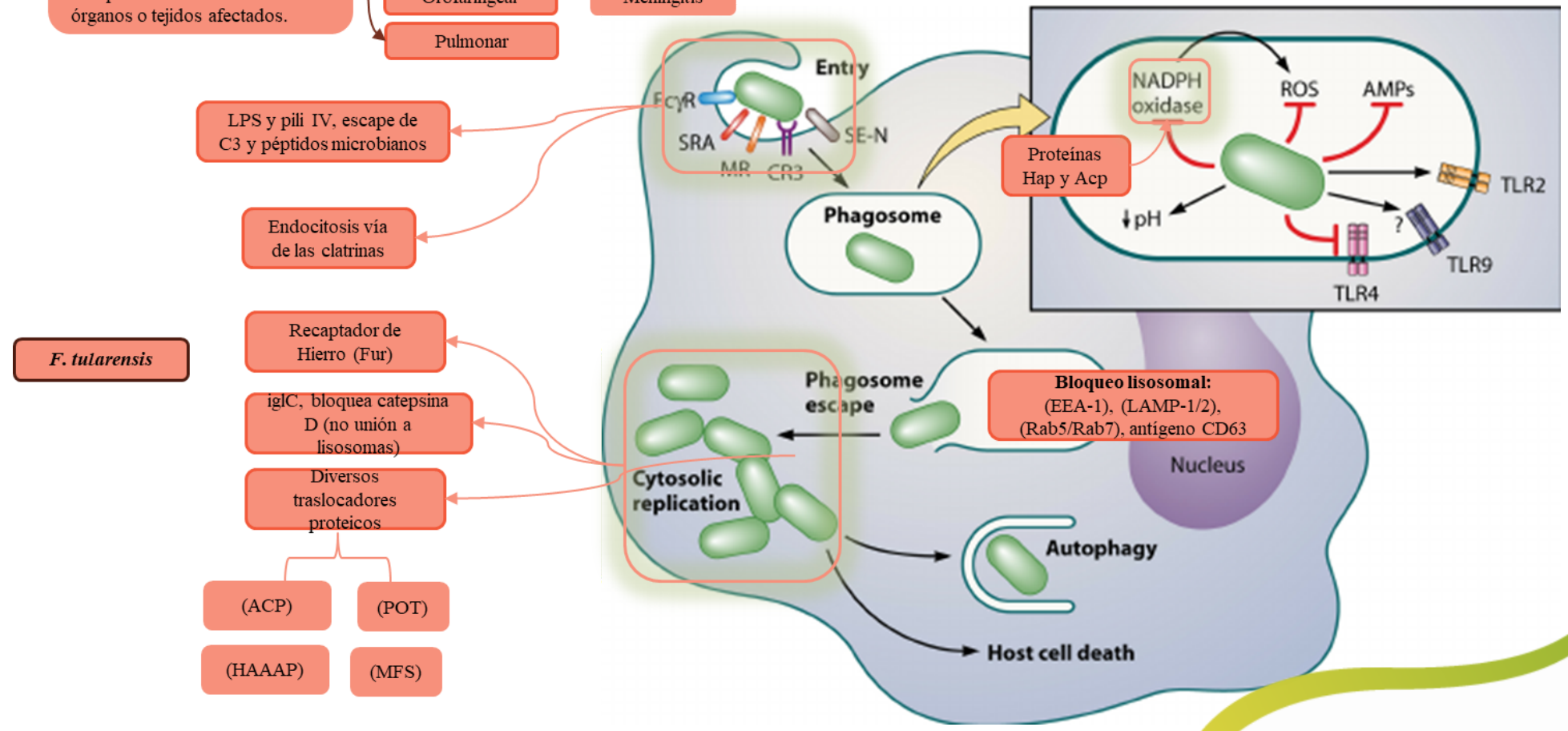
El proceso inflamatorio genera infiltración mononuclear (neutrófilos) y, en consecuencia, se produce necrosis de los órganos o tejidos afectados.

- Oculoglandular
- Glandular
- Tifoidea
- Orofaringeal
- Pulmonar

Tifoidea:
- Diarreas sanguinolentas por ulceración de los intestinos
- Órganos linforeticulares
- Meningitis

F. tularensis

- LPS y pili IV, escape de C3 y péptidos microbianos
- Endocitosis vía de las clatrinas
- Recaptador de Hierro (Fur)
- iglC, bloquea catepsina D (no unión a lisosomas)
- Diversos traslocadores proteicos
  - (ACP)
  - (POT)
  - (HAAAP)
  - (MFS)



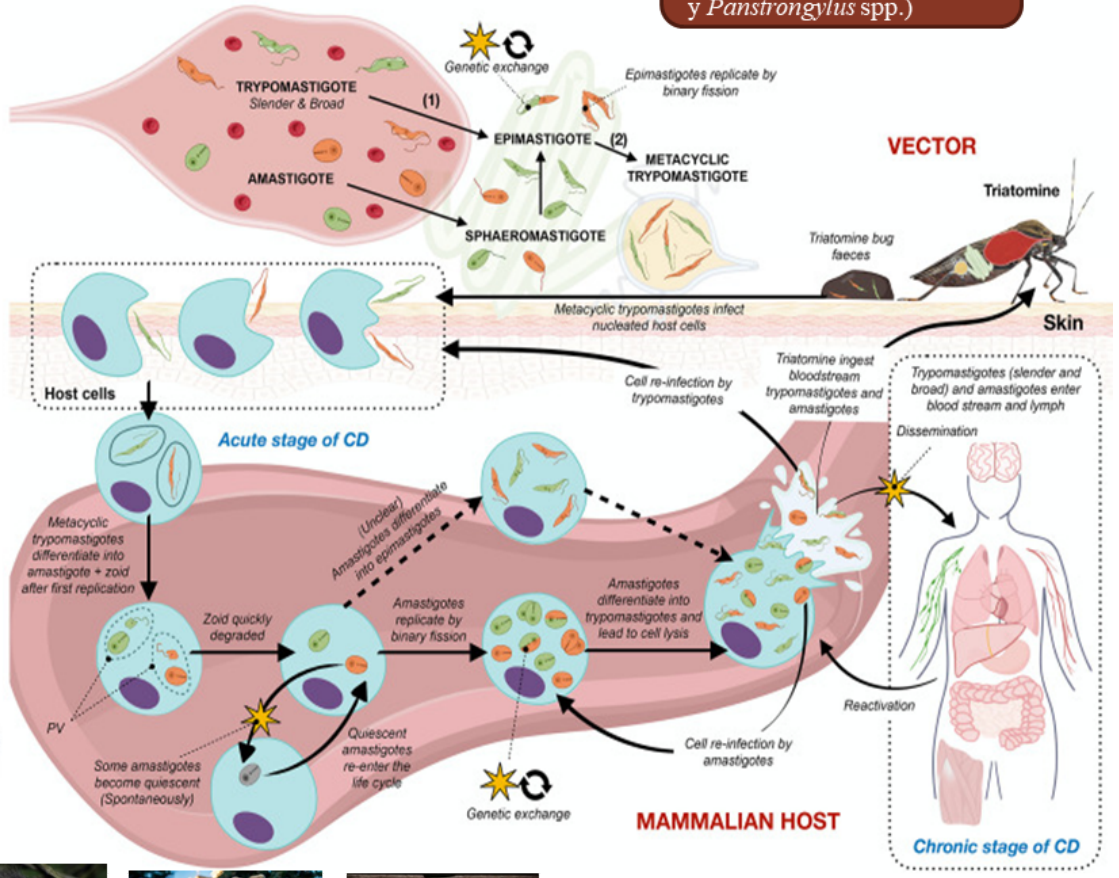
Trypanosoma spp

Orden Hemiptera, Subfamilia Triatominae (géneros *Triatoma* spp., *Rhodnius* spp., y *Panstrongylus* spp.)

T. cruzi

1. Adhesión a receptores con residuos ácido siálico y muscinas/trans-sialasa,
2. Invasión de diversos grupos celulares,
3. Sobrecarga en liberación Ca del ERL.
4. Producción importante EROs
5. Vacuolización y citólisis,
6. Inflamación e infiltración leucocitaria

Órganos de choque: corazón y TGI



DOI: 10.7589/2013-01-005

Journal of Wildlife Diseases, 49(4), 2013, pp. 1014–1018

White-Nosed Coatis (*Nasua narica*) Are a Potential Reservoir of *Trypanosoma cruzi* and Other Potentially Zoonotic Pathogens in Monteverde, Costa Rica

Parasitol Latinoam 62: 148 - 153, 2007 FLAP

EXPERIENCIA CLÍNICA

Miocarditis y miocardiopatía dilatada por Trypanosoma cruzi: Reporte de un caso

EVA MORENO-MEDINA*, IDALIA VALERIO-CAMPOS**, y PABLO GOYENAGA-CASTRO***

Rev. Ciencias Veterinarias, Vol. 36, N° 2, [1-14], E-ISSN: 2215-4507, julio-diciembre, 2018
DOI: http://dx.doi.org/10.15359/rcv.36-2.1
URL: http://www.revistas.una.ac.cr/index.php/veterinaria/index

Revista de Ciencias Veterinarias

Detección de anticuerpos contra *Trypanosoma cruzi* en caninos de Costa Rica

Detection of antibodies against *Trypanosoma cruzi* in dogs from Costa Rica

Marta C. Bonilla[1]✉, Marco Vinicio Herrero-Acosta[1], Andrea Urbina-Villalobos[2], Gaby Dolz[1]

Parasitol Latinoam 57: 66 - 68, 2002
FLAP

COMUNICACIÓN

Presencia de anticuerpos contra Trypanosoma cruzi en perros de Costa Rica

LILIANA REYES, ERICKA SILESKY, CARLOS CERDAS, MISAEL CHINCHILLA y OLGA GUERRERO

PREVALENCIA SEROLOGICA DE INFECCION POR *Trypanosoma cruzi* EN DONADORES DE SANGRE EN ZONAS ENDEMICAS PARA ENFERMEDAD DE CHAGAS EN COSTA RICA*

*Andrea Urbina, Luis Vargas, Miguel Rojas, Fernando Retana y Rodrigo Zeledón***

Paragonimus spp

Tipos:

1. Paragonimiasis Pulmonar,
2. Paragonimiasis Cardiaca,
3. Paragonimiasis Cutánea, y
4. Paragonimiasis Nerviosa.

Morphological and molecular characterization of the metacercaria of *Paragonimus caliensis*, as a separate species from *P. mexicanus* in Costa Rica

Roderico Hernández-Chea [a,*], Ana Eugenia Jiménez-Rocha [b], Ruth Castro [a], David Blair [c], Gaby Dolz [a,b]

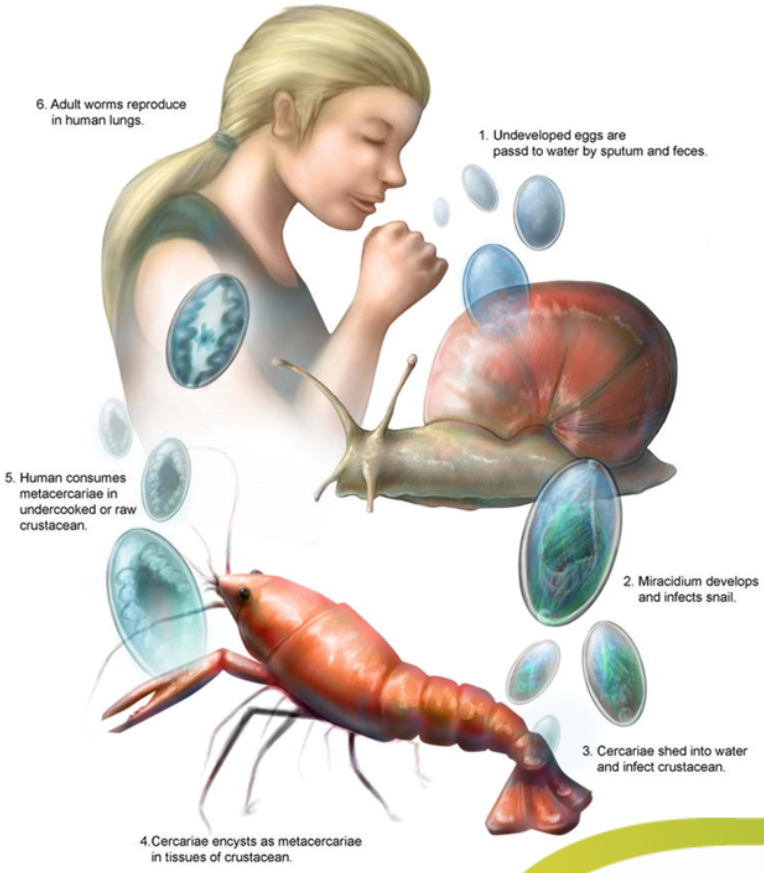
[a] Maestría en Enfermedades Tropicales, Posgrado Regional en Ciencias Veterinarias Tropicales, Universidad Nacional, Campus Benjamín Nuñez, Barreal de Heredia, P.O. Box 86, 3000 Heredia, Costa Rica
[b] Escuela de Medicina Veterinaria, Universidad Nacional, Campus Benjamín Núñez, Barreal de Heredia, Costa Rica
[c] College of Marine and Environmental Sciences, James Cook University, Townsville, QLD, 4811, Australia

Rev. Inst. Med. trop. São Paulo
27(1):23-26, *janeiro-fevereiro*, 1985

CDU 616.122.22

INFECCION NATURAL DE PTYCHOPHALLUS TRISTANI **(CRUSTACEA: DECAPODA) CON METACERCARIAS DE** PARAGONIMUS MEXICANUS **(TREMATODA) EN TABARCIA DE MORA, COSTA RICA (1)**

Life Cycle of *Paragonimus westermani*



Raillietina spp

Especies:

1. *R. celebensis*,
2. *R. demarariensis*, y
3. *R. siriraji*.

1. Expulsión de proglótidos en heces,
2. Distención abdominal,
3. Irritación gastroentérica,
4. Dolor intestinal,
5. Heces diarreicas de color amarillo-café y fiebre

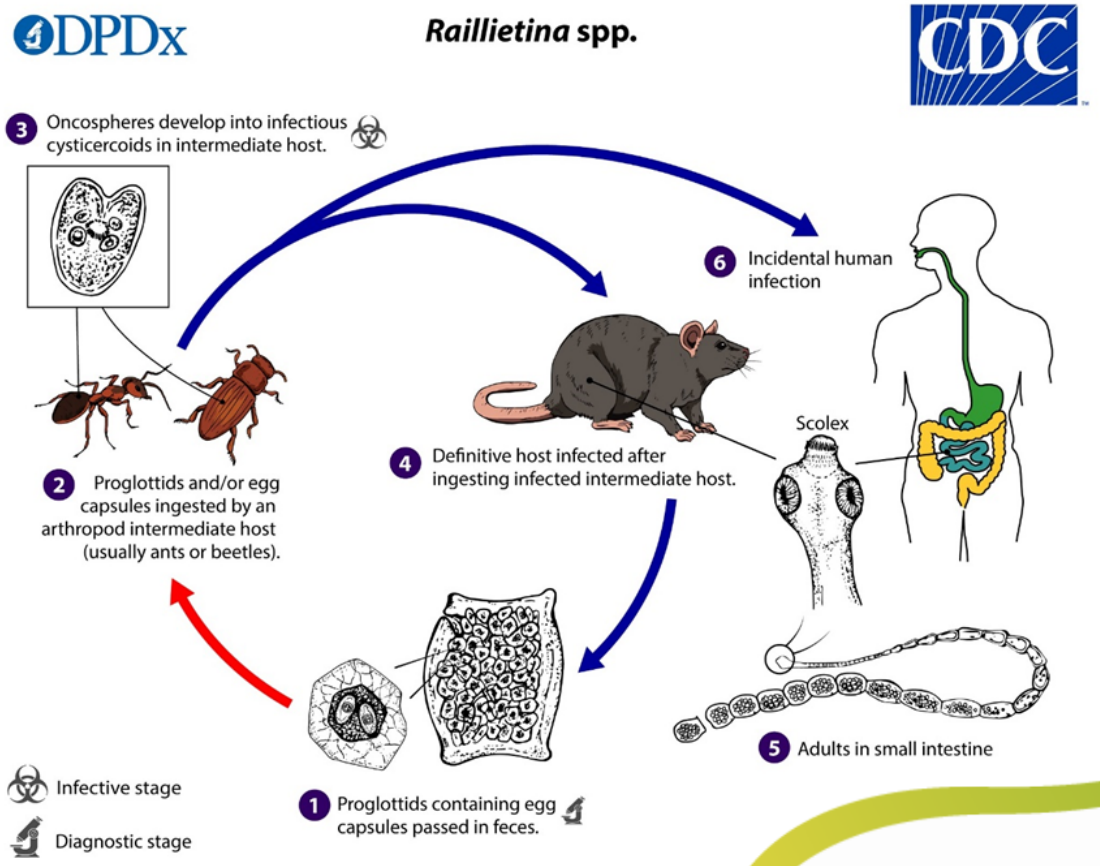
Rev. Colegio de Microb. Quím. Clín. de Costa Rica, Vol 25, N.° 1, enero – abril 2019• ISSN: 2215-3713

Parasitosis intestinal inusual.
Reporte de un caso de *Raillietina* spp.

Unusual intestinal parasitosis. Report of a case of *Raillietina* spp.

Luis Diego Ramírez-Fallas [1], Álvaro Vargas-Campos [1]

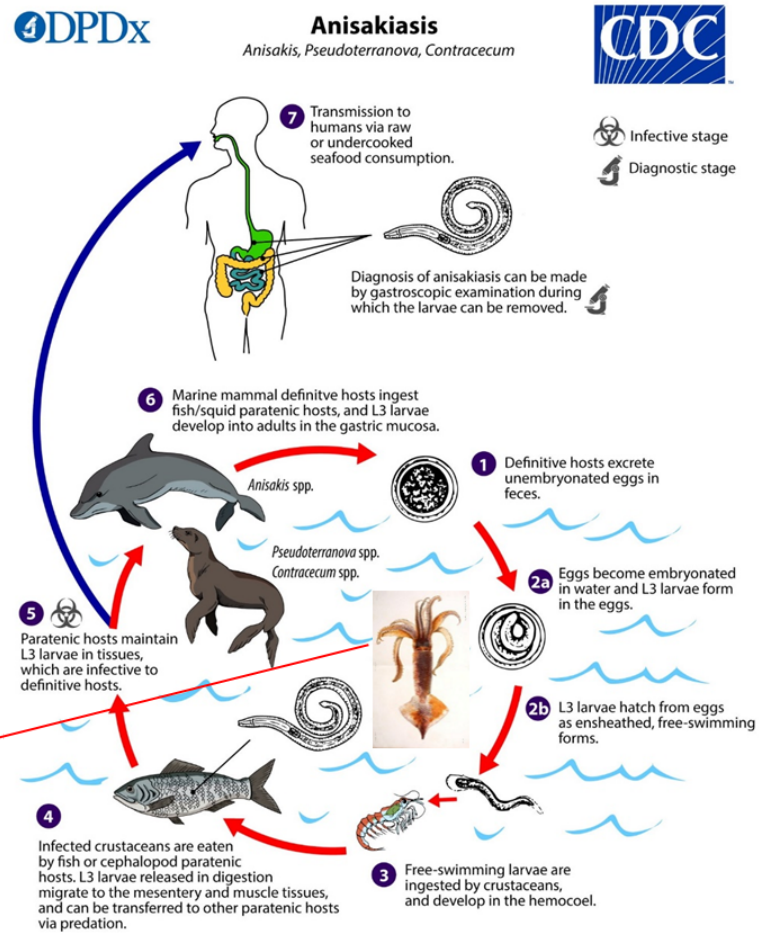
[1]Microbiólogo Químico Clínico, Caja Costarricense de Seguro Social



Familia Anisakidae

1. L3 rompe mucosa intestinal,
2. Formación de granuloma,
3. Vómito, diarrea mucosanguinolenta,
4. Shock anafiláctico (alérgenos termoestables y antiproteolíticos)

H. Paraténico



¿Preguntas?

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acartin@outlook.com