Evolution épidémiologique et écologique des pneumonies de l'enfant

8éme Journées d'Urgences Pédiatrique

Dr CANTAIS Aymeric

Pneumonie de l'enfant

2 August 2019

Key facts

- **Pneumonia accounts for 15% of all deaths of children under 5 years old, killing 808 694 children in 2017.**
- **Pneumonia can be caused by viruses, bacteria, or fungi.**
- **Pneumonia can be prevented by immunization, adequate nutrition, and by addressing environmental factors.**
- **Pneumonia caused by bacteria can be treated with antibiotics, but only one third of children with pneumonia receive the antibiotics they need.**

- Un monde dichotomisé -

Pneumonie de l'enfant

Pays en voie de développement

- Mortalité élevée
- Comorbidités
- Pas (peu) de vaccins
- Pneumopathies bactériennes

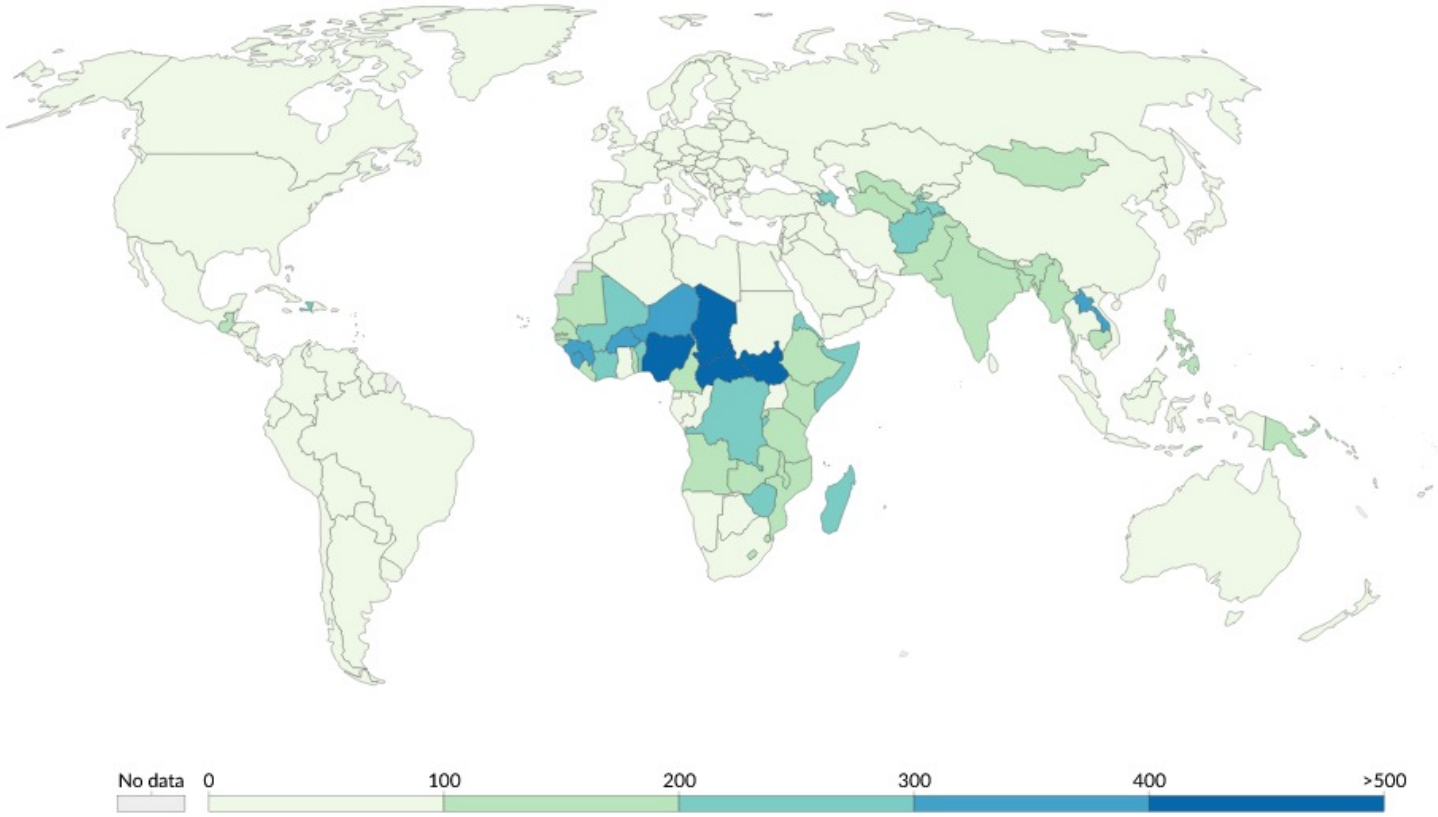
Pays développés

- Mortalité faible
- Vaccins
- Pneumopathies virales
- *Impact sur le système de santé (coûts ++)*

- Un monde dichotomisé -

Death rates from pneumonia in children under 5, 2017

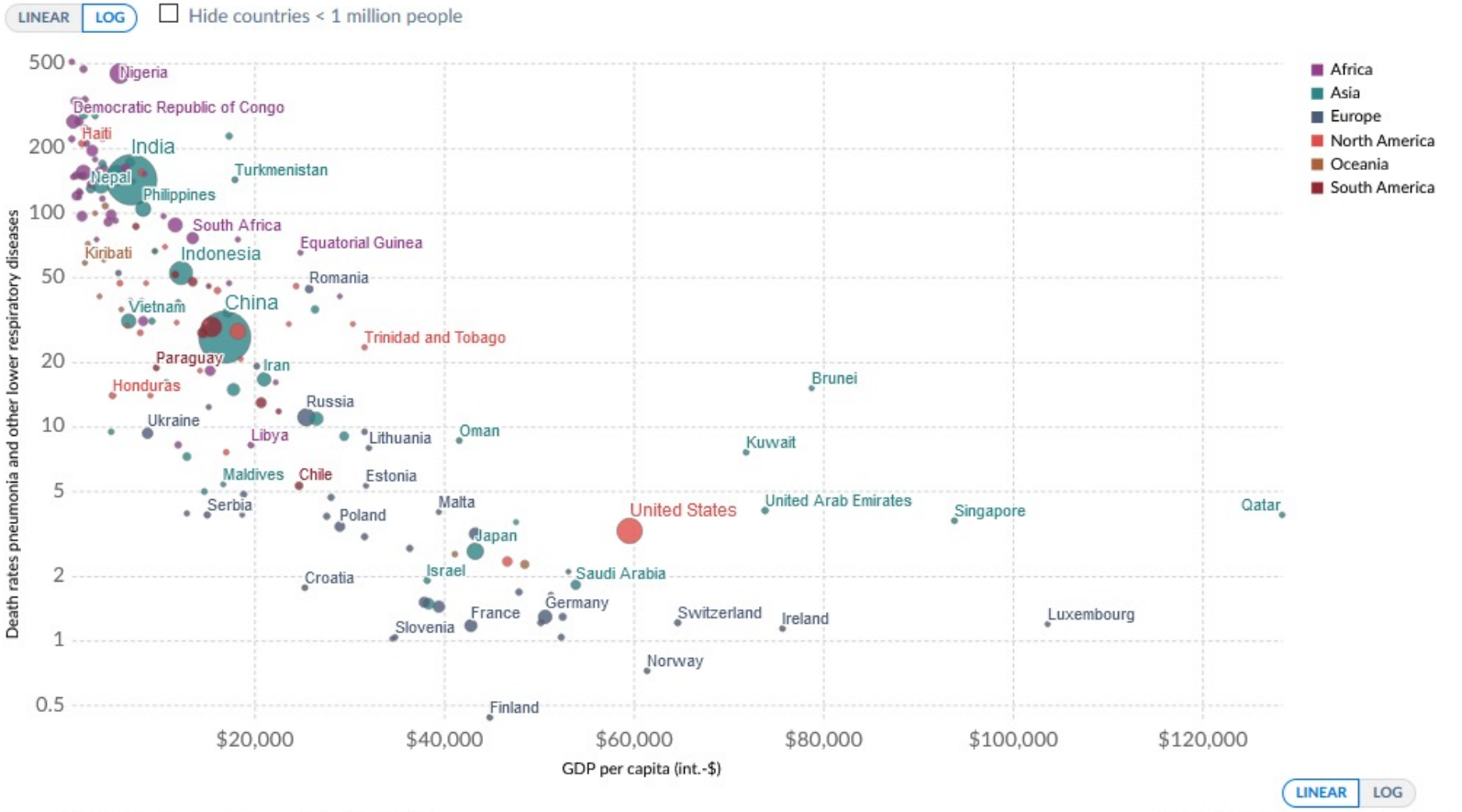
The annual number of deaths per 100,000 children under 5.



Source: IHME, Global Burden of Disease Study (2018)
OurWorldInData.org/pneumonia • CC BY
Note: Deaths from 'clinical pneumonia', which refers to a diagnosis based on disease symptoms such as coughing and difficulty breathing and may include other lower respiratory diseases.

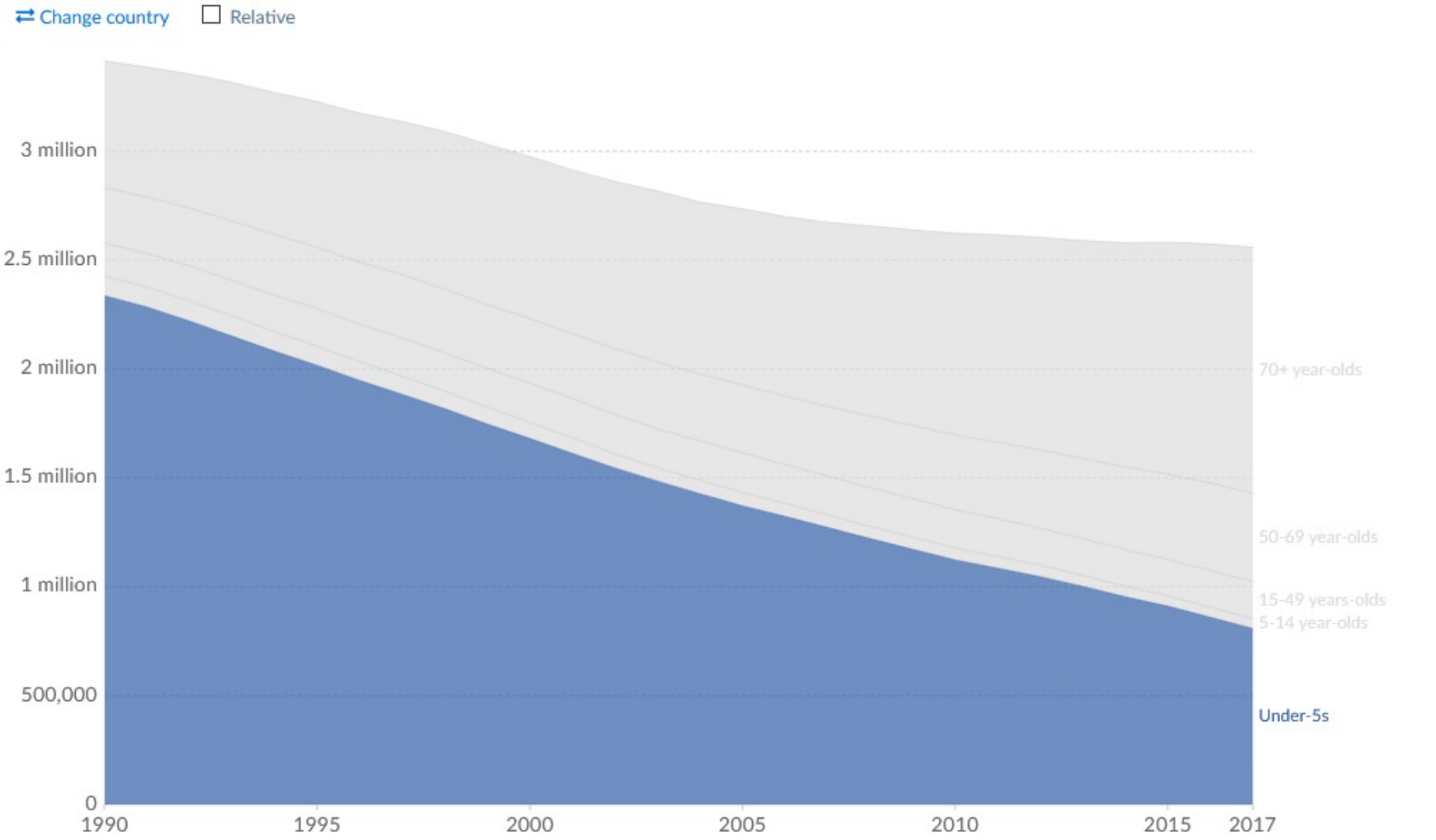
Death rate from pneumonia for children vs. GDP per capita, 2017

Death rate from pneumonia and other lower respiratory diseases in children under 5 per 100,000 children.
Gross domestic product (GDP) per capita is measured in 2011 international-$.



Source: IHME, Global Burden of Disease Study, World Bank

Deaths from pneumonia, by age, World, 1990 to 2017

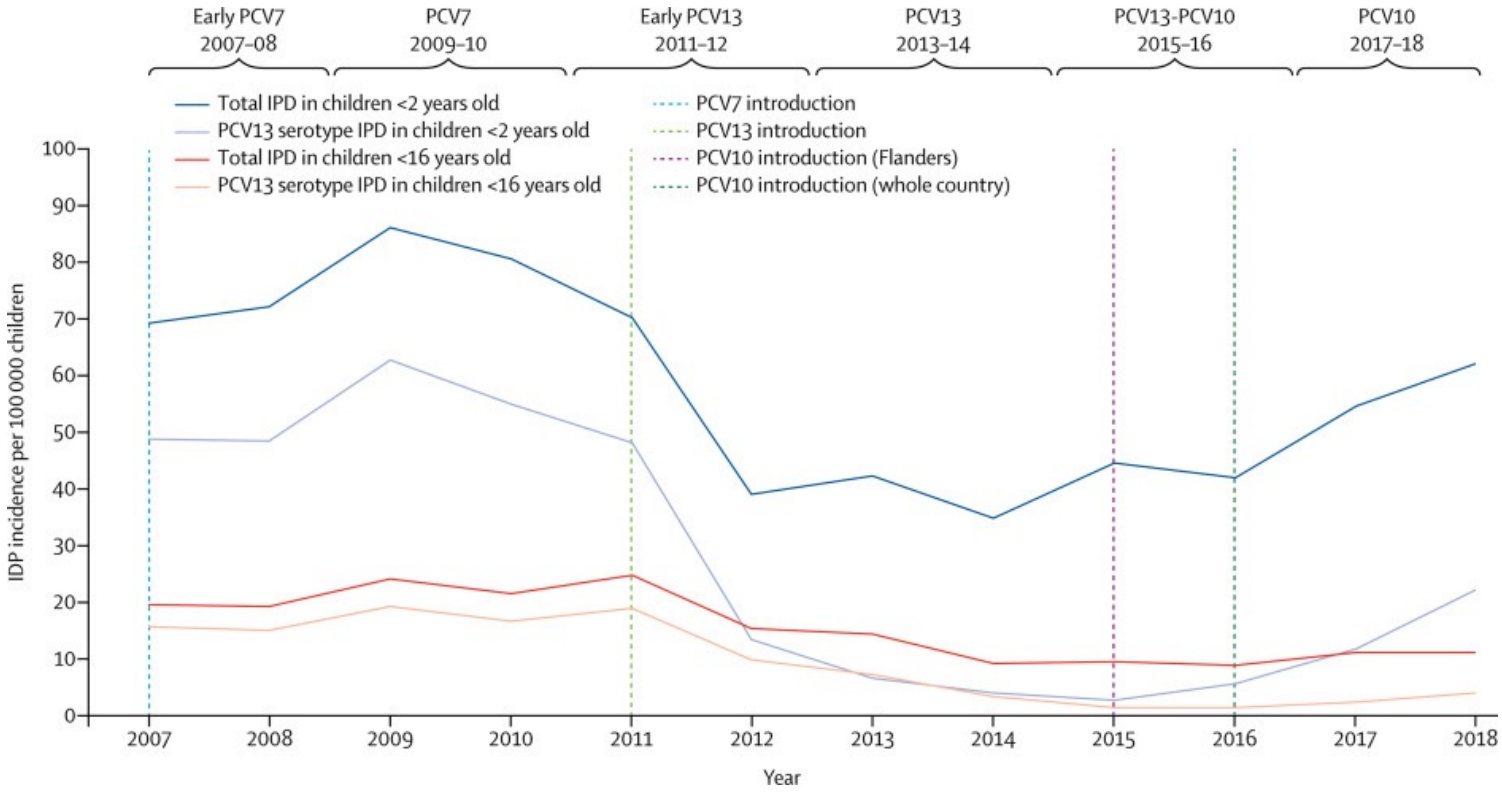


Source: IHME, Global Burden of Disease Study (GBD)
OurWorldInData.org/pneumonia • CC BY
Note: Deaths from 'clinical pneumonia', which refers to a diagnosis based on disease symptoms such as coughing and difficulty breathing and may include other lower respiratory diseases.


Pneumococcal
Vaccine
Polyvalent
10 ml
309485-495867

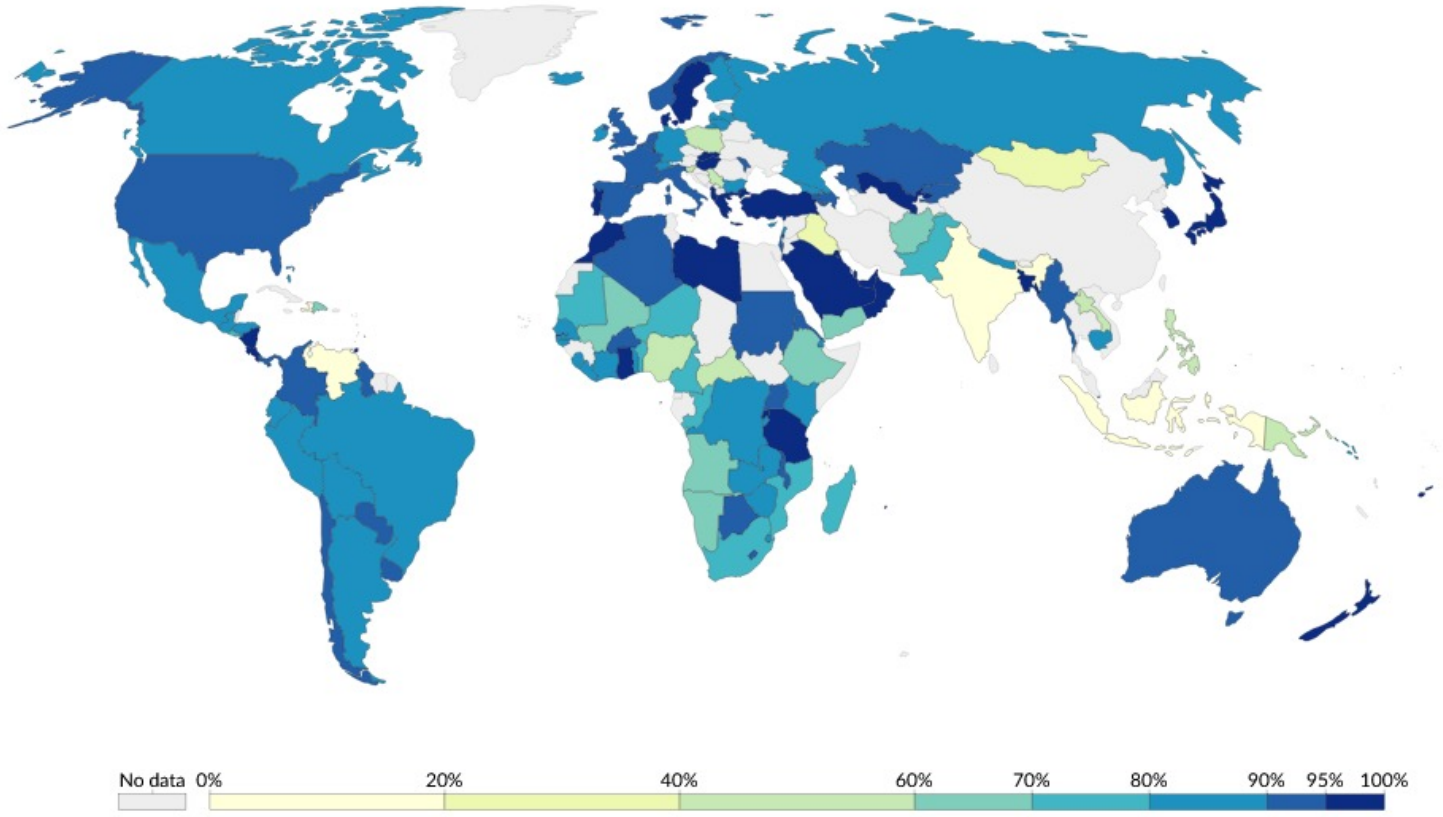
Dynamic changes in paediatric invasive pneumococcal disease after sequential switches of conjugate vaccine in Belgium: a national retrospective observational study

Stefanie Desmet, Katrien Lagrou, Chloé Wyndham-Thomas, Toon Braeye, Jan Verhaegen, Piet Maes, Steffen Fieuws, Willy E Peetermans, Sophie Blumental



Share of infants vaccinated with the Pneumococcal Conjugate Vaccine, 2018

Share of one-year-olds vaccinated with the 3rd dose of the Pneumococcal Conjugate Vaccine (PCV3), which protects against pneumonia and other life-threatening diseases.



Source: WHO

OurWorldInData.org/pneumonia • CC BY

2008 2018

RESEARCH ARTICLE

Impact of Pneumococcal Conjugate Vaccination of Infants on Pneumonia and Influenza Hospitalization and Mortality in All Age Groups in the United States

Lone Simonsen,[a,b] Robert J. Taylor,[a] Yinong Young-Xu,[c] Michael Haber,[d] Larissa May,[a,e] and Keith P. Klugman[f]

Sage Analytica, Bethesda, Maryland, USA[a]; Department of Global Health, School of Public Health and Health Services, George Washington University, Washington, DC, USA[b]; EpiPatterns, North Haverhill, New Hampshire, USA[c]; Department of Biostatistics and Bioinformatics, Rollins School of Public Health, Emory University, Atlanta, Georgia, USA[d]; School of Medicine, George Washington University, Washington, DC, USA[e]; and Hubert Department of Global Health, Rollins School of Public Health, Emory University, Atlanta, Georgia, USA[f]

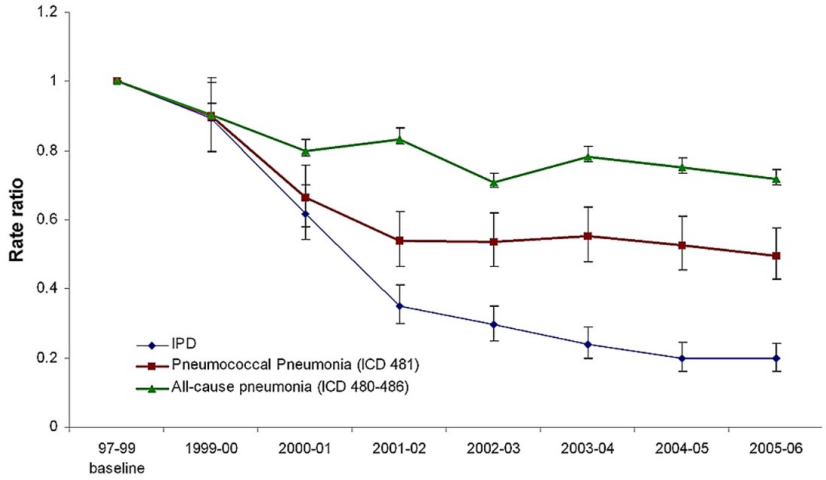
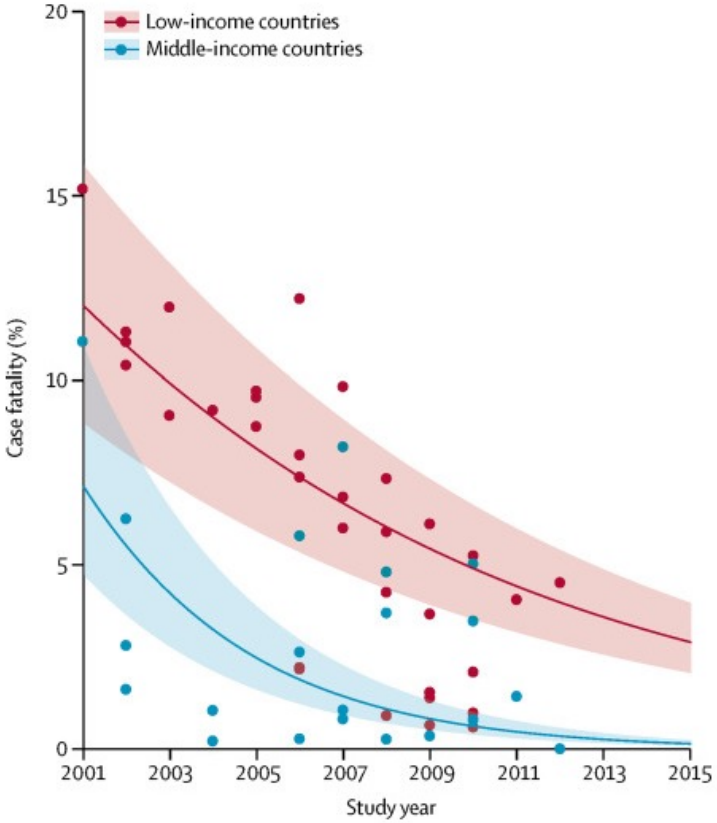
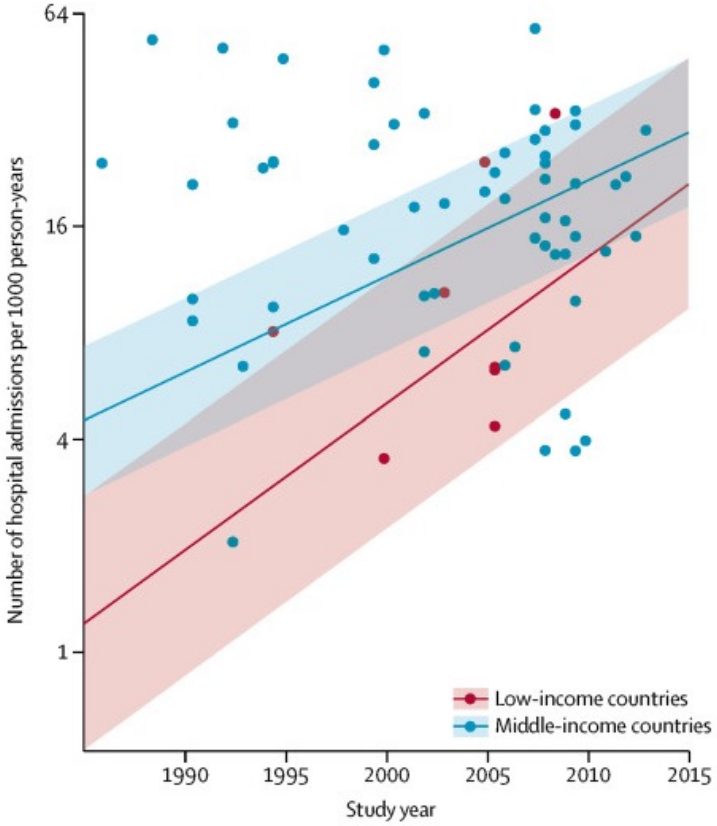


TABLE 1 Annual hospitalization rates per 100,000 in the pre-PCV7 period in the 10 study states at baseline[a] and in the 2005–2006 season

	Rate of IPD cases			Rate of pneumococcal (lobar) pneumonia cases (ICD9 481)			Rate of nonbacteremic pneumococcal pneumonia cases (ICD9 481 with no IPD code			Rate of all-cause pneumonia cases		
Age (yr)	Baseline	2005–2006	RR (95% CI)	Baseline	2005–2006	RR (95% CI)	Baseline	2005–2006	RR (95% CI)	Baseline	2005–2006	RR (95% CI)
0-1	27.8	5.5	0.20 (0.16–0.24)	25.9	13.4	0.49 (0.43–0.58)	22.1	12.4	0.53 (0.46–0.62)	1,026.5	753.7	0.72 (0.70–0.73)
2–4	5.4	2.0	0.35 (0.27–0.47)	11.8	9.1	0.83 (0.72–0.96)	10.5	8.5	0.88 (0.76–1.02)	307.5	303.4	0.99 (0.96–1.01)
5–17	1.4	0.7	0.47 (0.36–0.60)	4.9	2.6	0.52 (0.46–0.59)	4.4	2.4	0.54 (0.47–0.62)	76.1	67.0	0.87 (0.85–0.90)
18–39	3.5	1.4	0.38 (0.34–0.43)	11.6	4.3	0.37 (0.35–0.40)	9.4	3.5	0.39 (0.36–0.42)	88.5	62.6	0.68 (0.67–0.70)
40–64	8.9	6.0	0.64 (0.60–0.68)	30.2	16.6	0.56 (0.54–0.58)	24.8	13.6	0.56 (0.54–0.58)	250.9	234.6	0.92 (0.91–0.93)
≥65	30.6	17.1	0.53 (0.50–0.56)	144.9	64.7	0.46 (0.44–0.47)	126.1	55.9	0.46 (0.44–0.47)	1,875. 2	1672. 6	0.88 (0.87–0.89)

[a] 1996–1997 through 1998–1999 seasons.





McAllister DA, Liu L, Shi T, Chu Y, Reed C, Burrows J, Adeloye D, Rudan I, Black RE, Campbell H, Nair H. Global, regional, and national estimates of pneumonia morbidity and mortality in children younger than 5 years between 2000 and 2015: a systematic analysis. Lancet Glob Health. 2019 Jan;7(1):e47-e57. doi: 10.1016/S2214-109X(18)30408-X. Epub 2018 Nov 26. PMID: 30497986; PMCID: PMC6293057.

SUPPLEMENT ARTICLE

The Pneumonia Etiology Research for Child Health Project: A 21st Century Childhood Pneumonia Etiology Study

Orin S. Levine,[1] Katherine L. O'Brien,[1] Maria Deloria-Knoll,[1] David R. Murdoch,[2,3] Daniel R. Feikin,[1,4] Andrea N. DeLuca,[1] Amanda J. Driscoll,[1] Henry C. Baggett,[5] W. Abdullah Brooks,[1,6] Stephen R. C. Howie,[7] Karen L. Kotloff,[8,9] Shabir A. Madhi,[10,11] Susan A. Maloney,[5] Samba Sow,[8,9,12] Donald M. Thea,[13] and J. Anthony Scott[14,15]

Figure 1. Map of study sites of the Pneumonia Etiology Research for Child Health project.

Clinical Infectious Diseases

SUPPLEMENT ARTICLE

Introduction to the Epidemiologic Considerations, Analytic Methods, and Foundational Results From the Pneumonia Etiology Research for Child Health Study

Katherine L. O'Brien,[1] Henry C. Baggett,[2,3] W. Abdullah Brooks,[1,4] Daniel R. Feikin,[1,5] Laura L. Hammitt,[1,6] Stephen R. C. Howie,[7,8,9] Maria Deloria Knoll,[1] Karen L. Kotloff,[10] Orin S. Levine,[1,11] Shabir A. Madhi,[12,13] David R. Murdoch,[14,15] J. Anthony G. Scott,[7,16] Donald M. Thea,[17] and Scott L. Zeger[18]

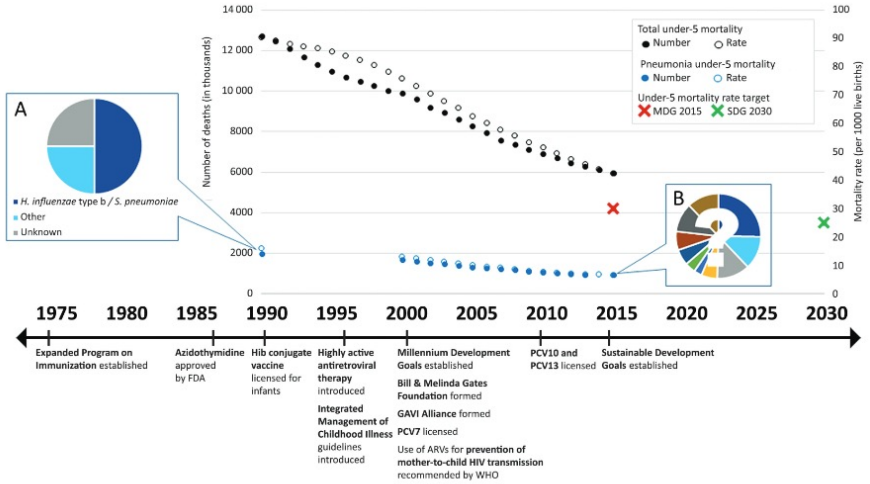
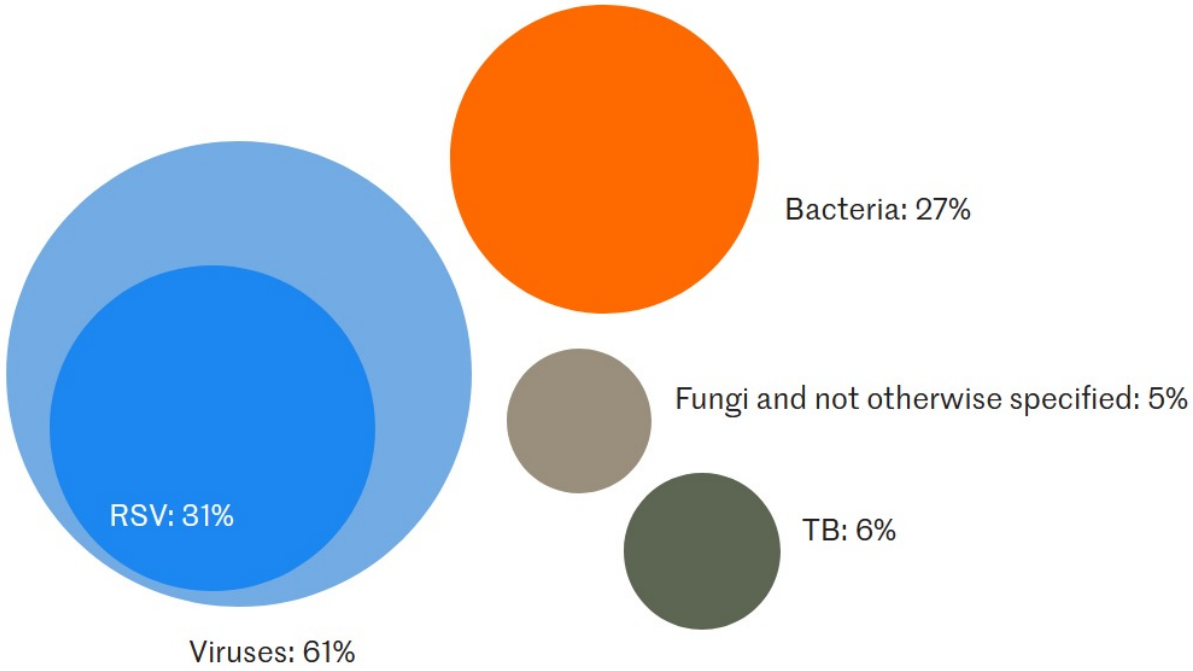
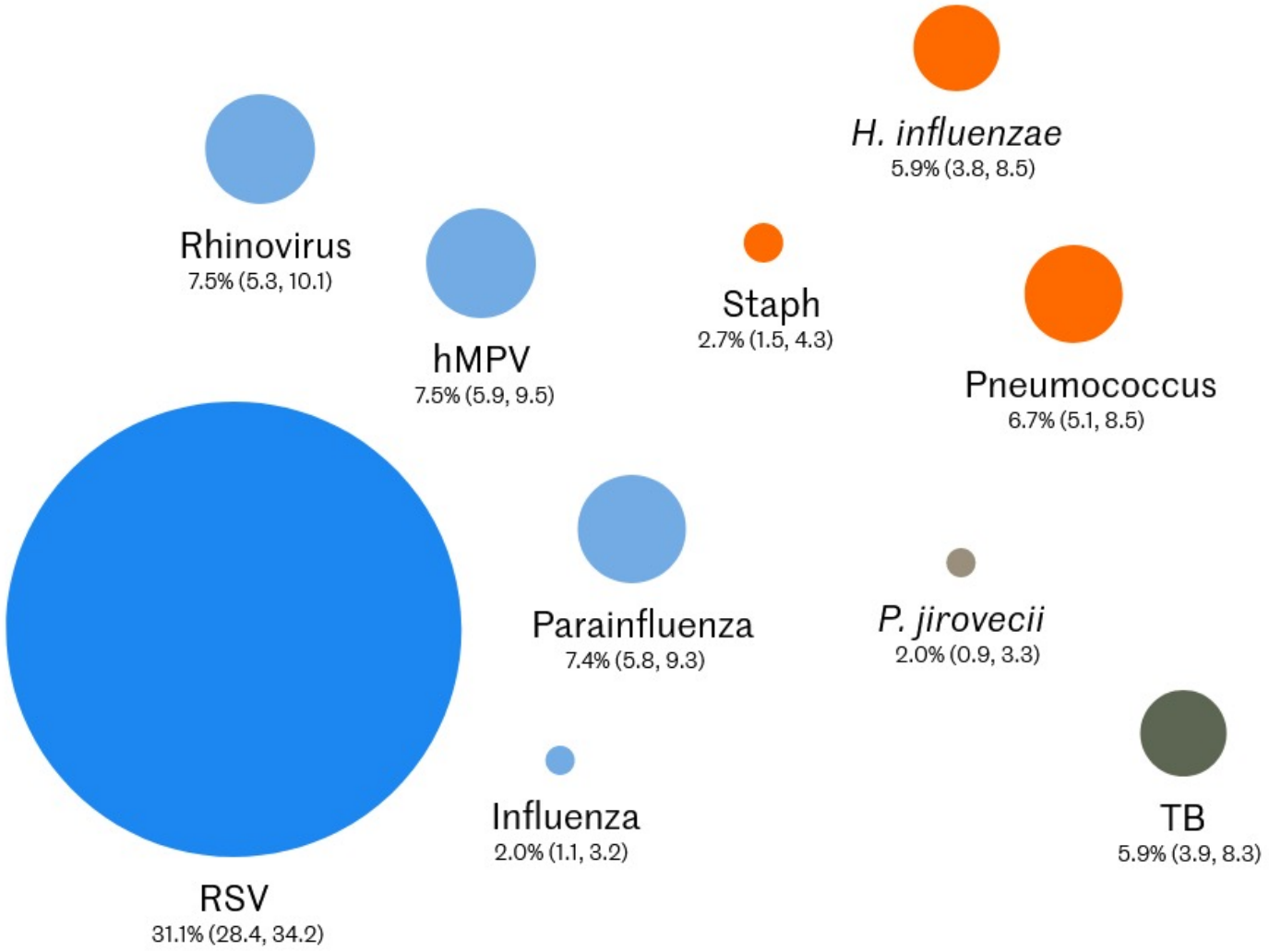


Figure 1. Annual under-5 all-cause and pneumonia mortality rates with global targets and pivotal health interventions, by time. *A*, The conceptual pneumonia mortality etiology distribution from the preconjugate vaccine era and *B*, the unknown etiologic distribution of the present and future. Abbreviations: ARVs, antiretrovirals; FDA, US Food and Drug Administration; Hib, *Haemophilus influenzae* type b; MDG, Millenium Development Goal; PCV, pneumococcal conjugate vaccine; SDG, Sustainable Development Goal; *S. pneumoniae*, *Streptococcus pneumoniae*; WHO, World Health Organization.

Over the course of two years in 7 countries, the PERCH study team enrolled and tested children hospitalized with pneumonia for the presence of over 30 viruses, bacteria and fungi.

Viruses caused most of the severe pneumonia cases (61%), and respiratory syncytial virus (RSV) was the leading pathogen (31%) at all sites.



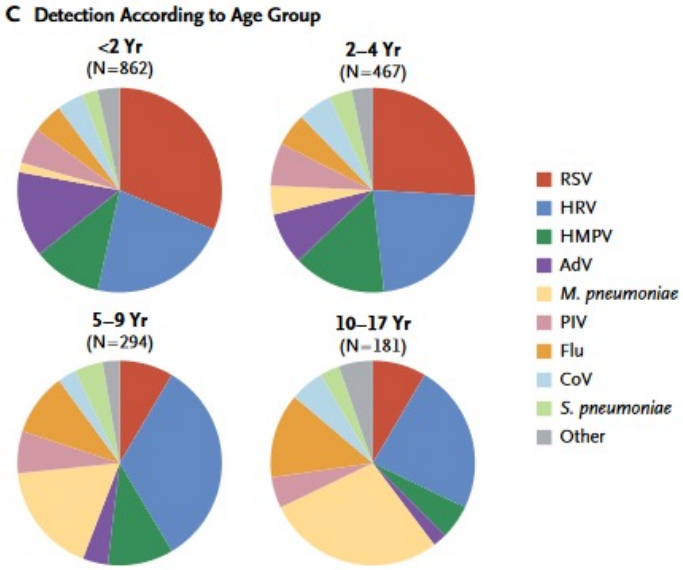

H. influenzae
5.9% (3.8, 8.5)
Rhinovirus
7.5% (5.3, 10.1)
hMPV
7.5% (5.9, 9.5)
Staph
2.7% (1.5, 4.3)
Pneumococcus
6.7% (5.1, 8.5)
Parainfluenza
7.4% (5.8, 9.3)
P. jirovecii
2.0% (0.9, 3.3)
Influenza
2.0% (1.1, 3.2)
TB
5.9% (3.9, 8.3)
RSV
31.1% (28.4, 34.2)

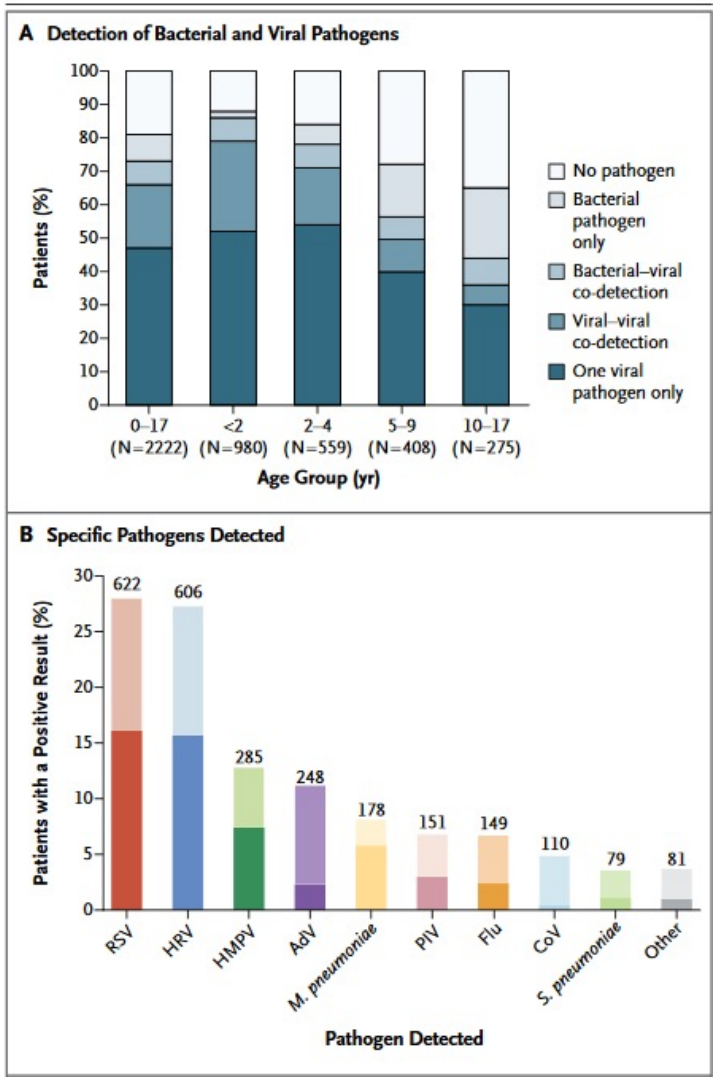
The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Community-Acquired Pneumonia Requiring Hospitalization among U.S. Children

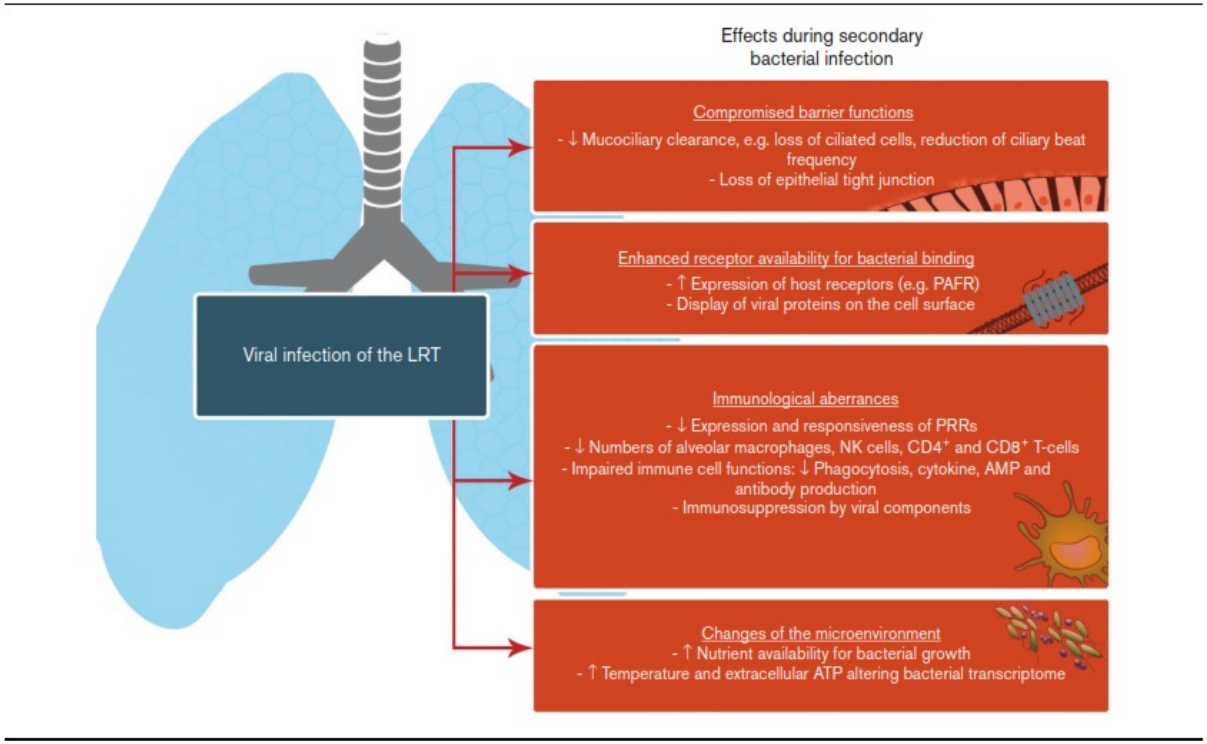
Seema Jain, M.D., Derek J. Williams, M.D., M.P.H., Sandra R. Arnold, M.D., Krow Ampofo, M.D., Anna M. Bramley, M.P.H., Carrie Reed, Ph.D.,

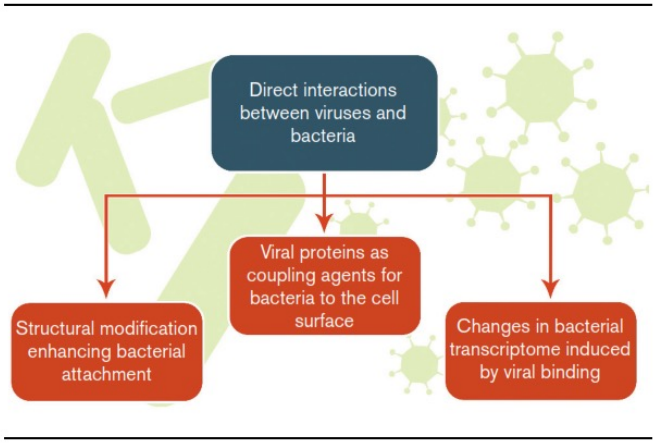




La compréhension de la **pathophysiologie** progresse :

- Interactions bactéries-virus dans la survenue d'une infection
- Infections secondaires ou primitives ?
- Infections séquentielles ?





Bellinghausen C, Rohde GG, Savelkoul PH, Wouters EF, Stassen FR. Viral-bacterial interactions in the respiratory tract. J Gen Virol. 2016 Dec;97(12):3089-3102. doi: 10.1099/jgv.0.000627. Epub 2016 Oct 18. Review.

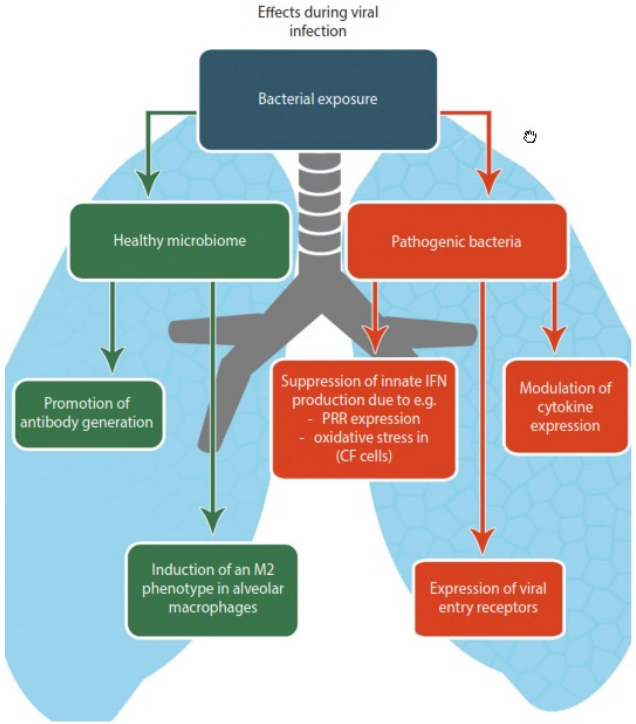
Microbiome / Virome

=> Effet protecteur ? Fragilisant ?

=> Impact des antibiothérapies intempestives ?

« La réponse virale est déterminée aussi par la présence de bactéries. »

« La réponse bactérienne est déterminée aussi par la présence de virus »



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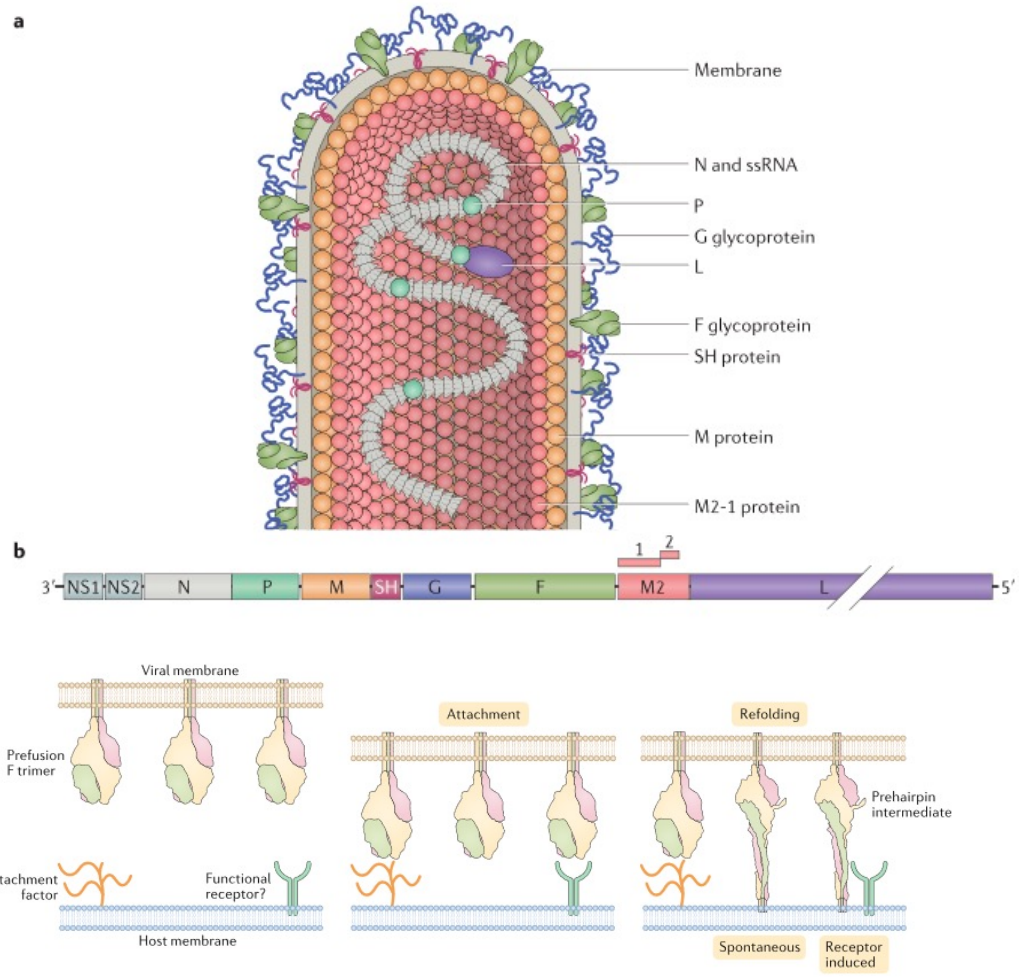


Fig. 4 | **Attachment and fusion.** The fusion (F) protein can interact with one or more attachment factors to bind the virus to the host cell, a process that is greatly enhanced by the viral attachment protein (not shown). The F protein may also interact with a functional receptor that induces refolding of the prefusion conformation to the prehairpin intermediate. Alternatively, or in addition, the F protein spontaneously refolds at a basal rate.

Respiratory syncytial virus entry and how to block it

*Michael B. Battles[1] and Jason S. McLellan[2]**

Abstract | Respiratory syncytial virus (RSV) is a leading cause of lower respiratory tract disease in young children and elderly people. Although the virus was isolated in 1955, an effective RSV vaccine has not been developed, and the only licensed intervention is passive immunoprophylaxis of high-risk infants with a humanized monoclonal antibody. During the past 5 years, however, there has been substantial progress in our understanding of the structure and function of the RSV glycoproteins and their interactions with host cell factors that mediate entry. This period has coincided with renewed interest in developing effective interventions, including the isolation of potent monoclonal antibodies and small molecules and the design of novel vaccine candidates. In this Review, we summarize the recent findings that have begun to elucidate RSV entry mechanisms, describe progress on the development of new interventions and conclude with a perspective on gaps in our knowledge that require further investigation.

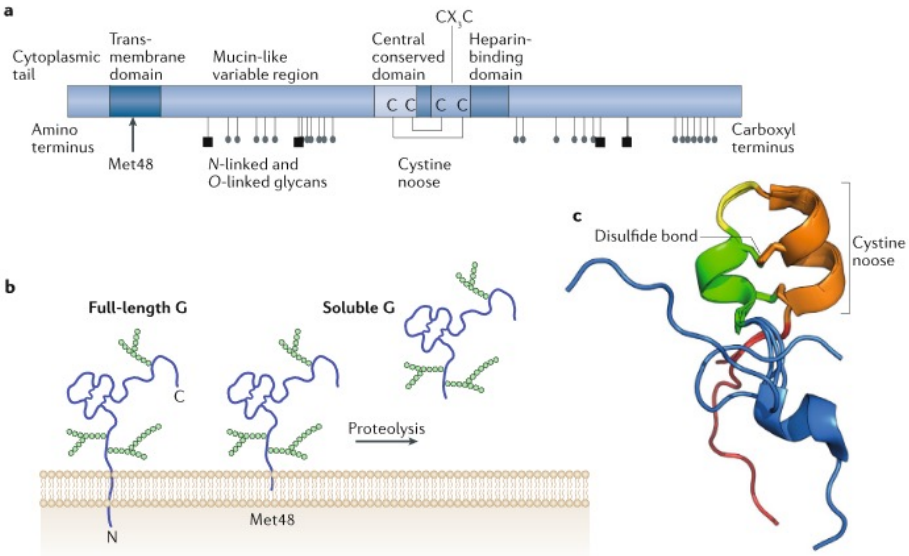


Fig. 2 | **Attachment protein structure. a** | Full-length respiratory syncytial virus (RSV) attachment (G) protein domains and post-translational modifications. **b** | Schematic of the two RSV G isoforms. Several *O*-linked glycans are represented by chains of green hexagons. **c** | Superposition of the cystine noose and flanking regions derived from four crystal structures of RSV G-derived peptides in complex with different antigen-binding fragments (Fabs). The peptide is coloured following the spectrum from blue (amino terminus) to red (carboxyl terminus). The two disulfide bonds in the cystine noose are shown as sticks linking the helices.

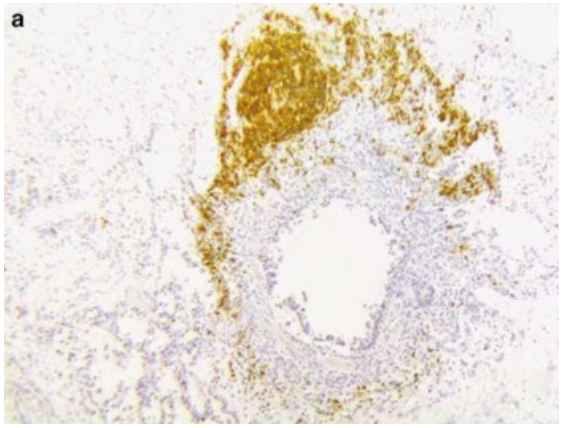
Modern Pathology (2007) 20, 108–119

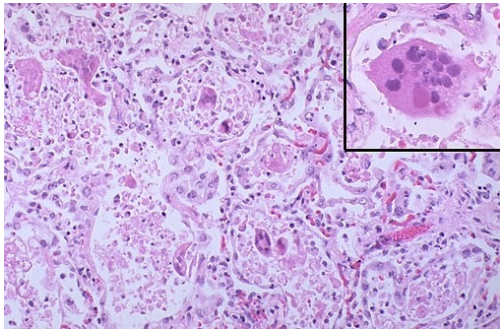
www.modernpathology.org

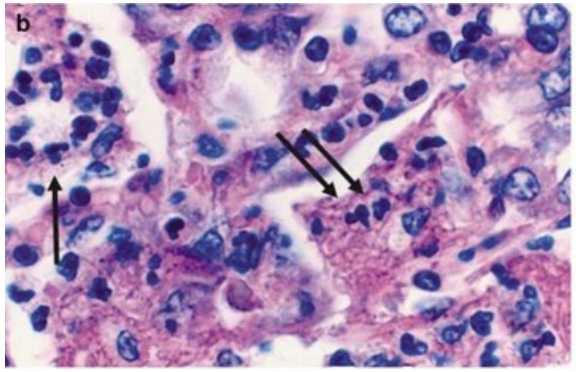
The histopathology of fatal untreated human respiratory syncytial virus infection

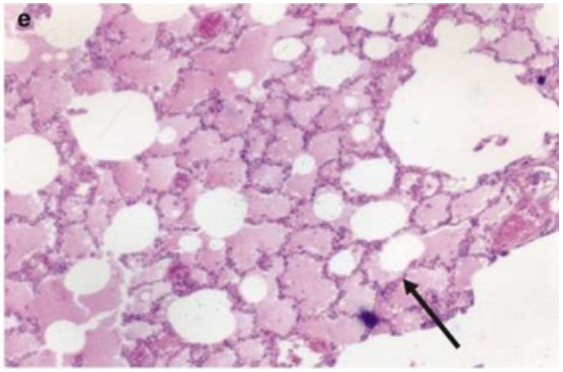
Joyce E Johnson[1], Ricardo A Gonzales[2], Sandy J Olson[1], Peter F Wright[1,2,3] and Barney S Graham[4]

[1]*Department of Pathology, Vanderbilt University School of Medicine, Nashville, TN, USA;* [2]*Department of Pediatrics, Vanderbilt University School of Medicine, Nashville, TN, USA;* [3]*Department of Microbiology & Immunology, Vanderbilt University School of Medicine, Nashville, TN, USA and* [4]*Vaccine Research Center, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD, USA*









De fait, la majorité des infections à VRS de l'enfant sont en fait l'atteinte pulmonaire globale et polymorphe de zones d'inflammation bronchiolaires pures avec des vastes plages d'atteintes alvéolaires ou interstitielles aggravant la difficulté des échanges gazeux.

viruses

MDPI

Review

Rhinoviruses and Respiratory Enteroviruses: Not as Simple as *ABC*

Léna Royston [1,2,*] and Caroline Tapparel [1,2]

Received: 27 October 2015; Accepted: 28 December 2015; Published: 11 January 2016
Academic Editor: George Belov

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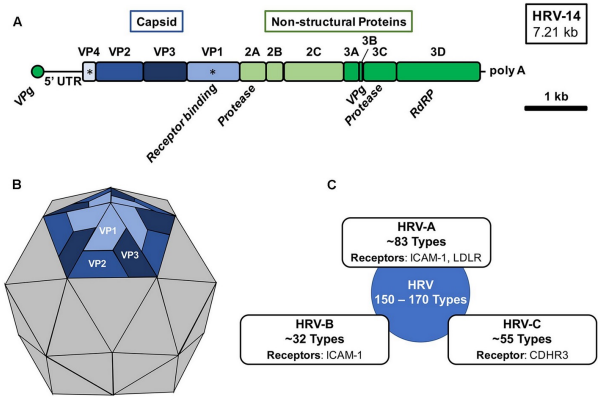
frontiers in Microbiology

MINI REVIEW
published: 05 December 2017
doi: 10.3389/fmicb.2017.02412

Rhinovirus Biology, Antigenic Diversity, and Advancements in the Design of a Human Rhinovirus Vaccine

Christopher C. Stobart[1], Jenna M. Nosek[1] and Martin L. Moore[2,3*]*

[1] Department of Biological Sciences, Butler University, Indianapolis, IN, United States, [2] Department of Pediatrics, Emory University, Atlanta, GA, United States, [3] Children's Healthcare of Atlanta, Atlanta, GA, United States



frontiers in Microbiology

REVIEW
published: 13 November 2018
doi: 10.3389/fmicb.2018.02677

Enterovirus D68 – The New Polio?

*Hayley Cassidy, Randy Poelman, Marjolein Knoester, Coretta C. Van Leer-Buter and Hubert G. M. Niesters**

Department of Medical Microbiology and Infection Prevention, Division of Clinical Virology, University of Groningen, University Medical Center Groningen, Groningen, Netherlands

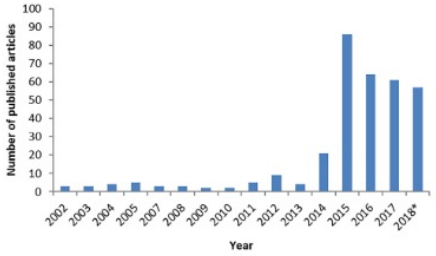
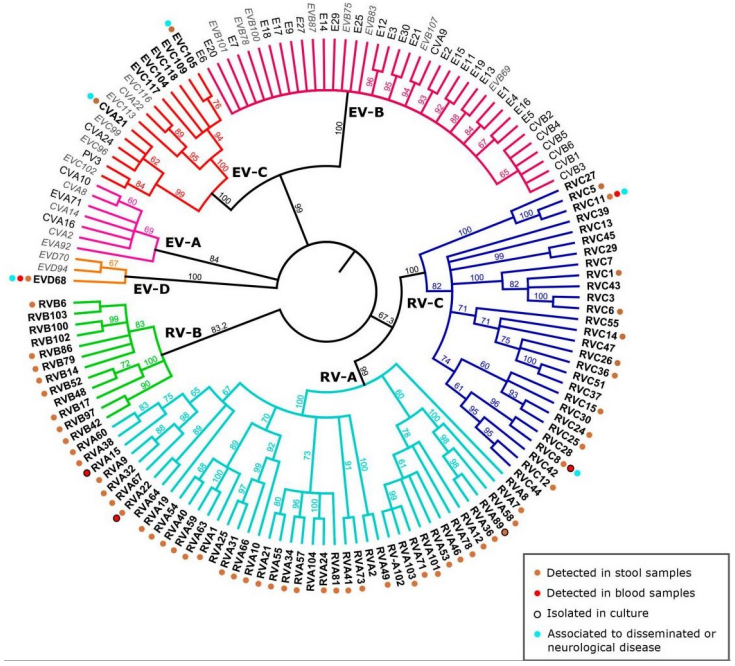


FIGURE 1 | The number of published articles on PubMed describing Enterovirus-D68 from January 2002 to October2018. The number of published articles for each year is in accordance with EV-D68 interest from the previous year. *Until October.



International Journal of Infectious Diseases 25 (2014) 45–52

Contents lists available at ScienceDirect

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journal homepage: www.elsevier.com/locate/ijid

Human metapneumovirus: review of an important respiratory pathogen

Swagatika Panda [a], Nirmal Kumar Mohakud [b], Lindomar Pena [c], Subrat Kumar [a,*]

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[b] Department of Paediatrics, Kalinga Institute of Medical Sciences, KIIT University, Bhubaneswar, Orissa, India
[c] Department of Cell and Molecular Biology, Centre for Biotechnology, Federal University of Paraiba, Joao Pessoa, Paraiba, Brazil

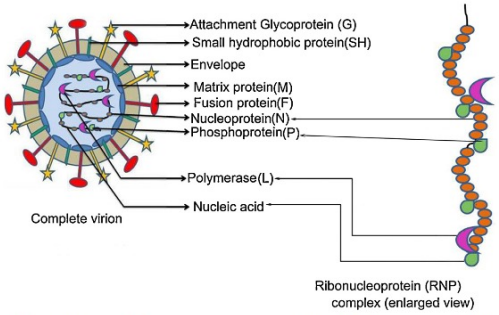

Figure 1. Schematic diagram of the human metapneumovirus particle and the ribonucleoprotein (RNP) complex.

- Atteinte cytopathogénique VRS-like (y compris syncitia)
- Co-infection VRS fréquentes
- Enfants plus âgés
- Immunité sous-jacente

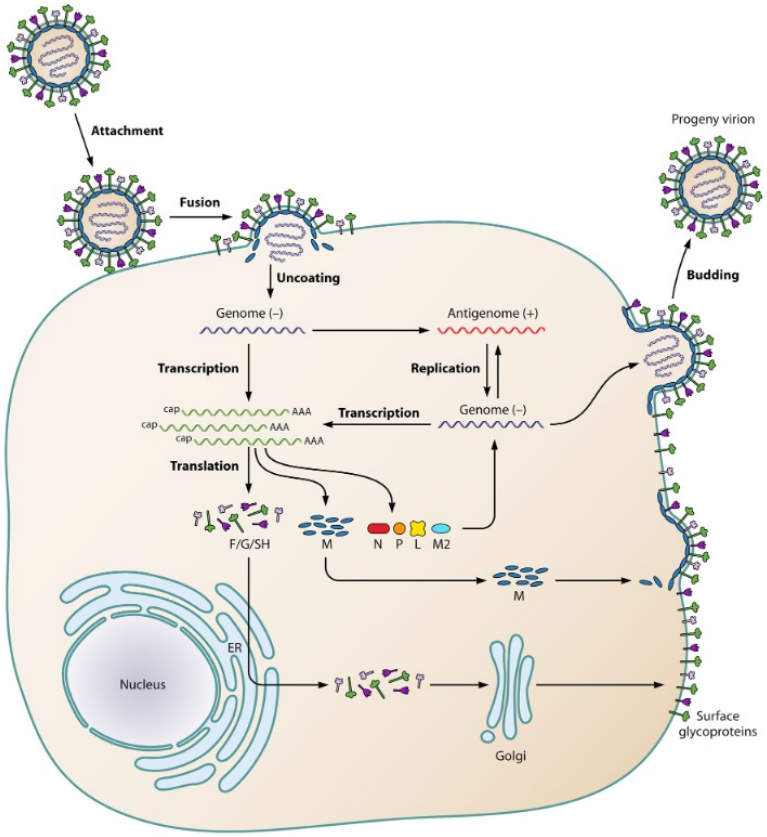

FIG. 3. Schematic representation of the HMPV life cycle. After attachment of the virion to the plasma membrane, the viral and plasma membranes fuse, resulting in uncoating of the virion and release of the RNP (containing the negative-sense viral RNA) into the cytoplasm. After primary transcription, the genome is replicated to produce the antigenome. The antigenome is used to synthesize genomic RNA, which is used to produce additional antigenomes for incorporation into progeny virions or as a template for secondary transcription. After translation, M proteins and RNPs are transported intracellularly to the plasma membrane and the viral glycoproteins F (fusion), G (glycoprotein), and SH (small hydrophobic) are transported from the endoplasmic reticulum (ER) to the Golgi apparatus and then the plasma membrane. Finally, new virions are assembled and are subsequently released from the plasma membrane by a budding process.

frontiers in Microbiology

REVIEW
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Infection with and Carriage of *Mycoplasma pneumoniae* in Children

Patrick M. Meyer Sauteur[1,2,3], Wendy W. J. Unger[2], David Nadal[3], Christoph Berger[3], Cornelis Vink[4] and Annemarie M. C. van Rossum[1]*

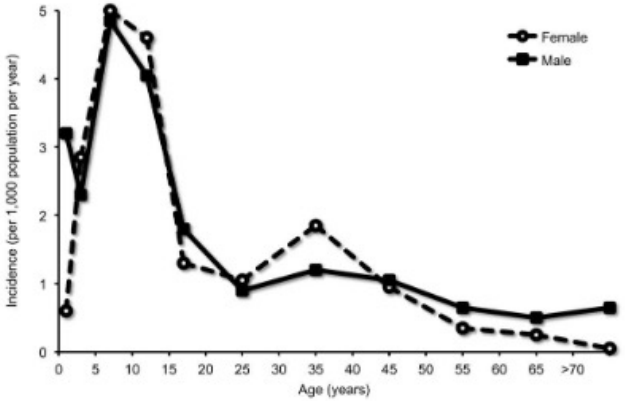

FIGURE 2 | Detection of *M. pneumoniae* in community-acquired pneumonia (CAP) according to age group. Infection was diagnosed by culture of respiratory specimens and/or a fourfold titer rise in complement fixation test (CFT). Adapted with permission from Foy et al. (1979).

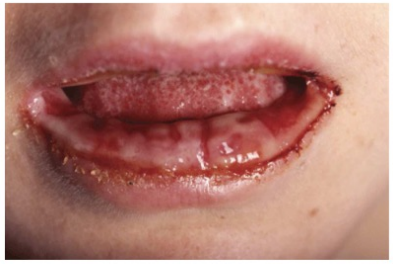

FIGURE 3 | *M. pneumoniae*-associated mucositis (MPAM). Erosive oral lesions limited to the mucosa in this form of MPAM in a 24-year-old woman. Reprinted with permission from Meyer Sauteur et al. (2012).

REVIEW ARTICLE
Front. Microbiol., 31 March 2016 | https://doi.org/10.3389/fmicb.2016.00414

Inflammation-inducing Factors of *Mycoplasma pneumoniae*

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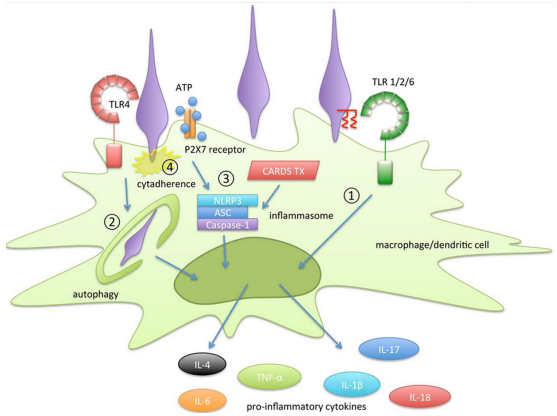
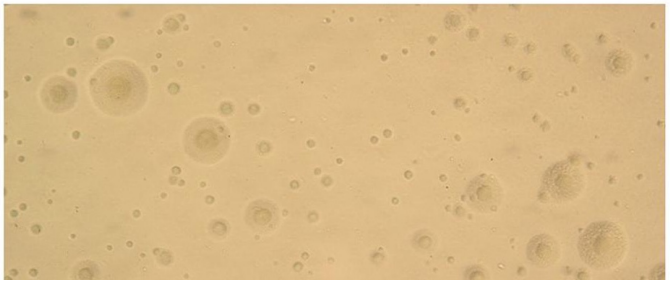


Appearance of colonies of *Mycoplasma pneumoniae*



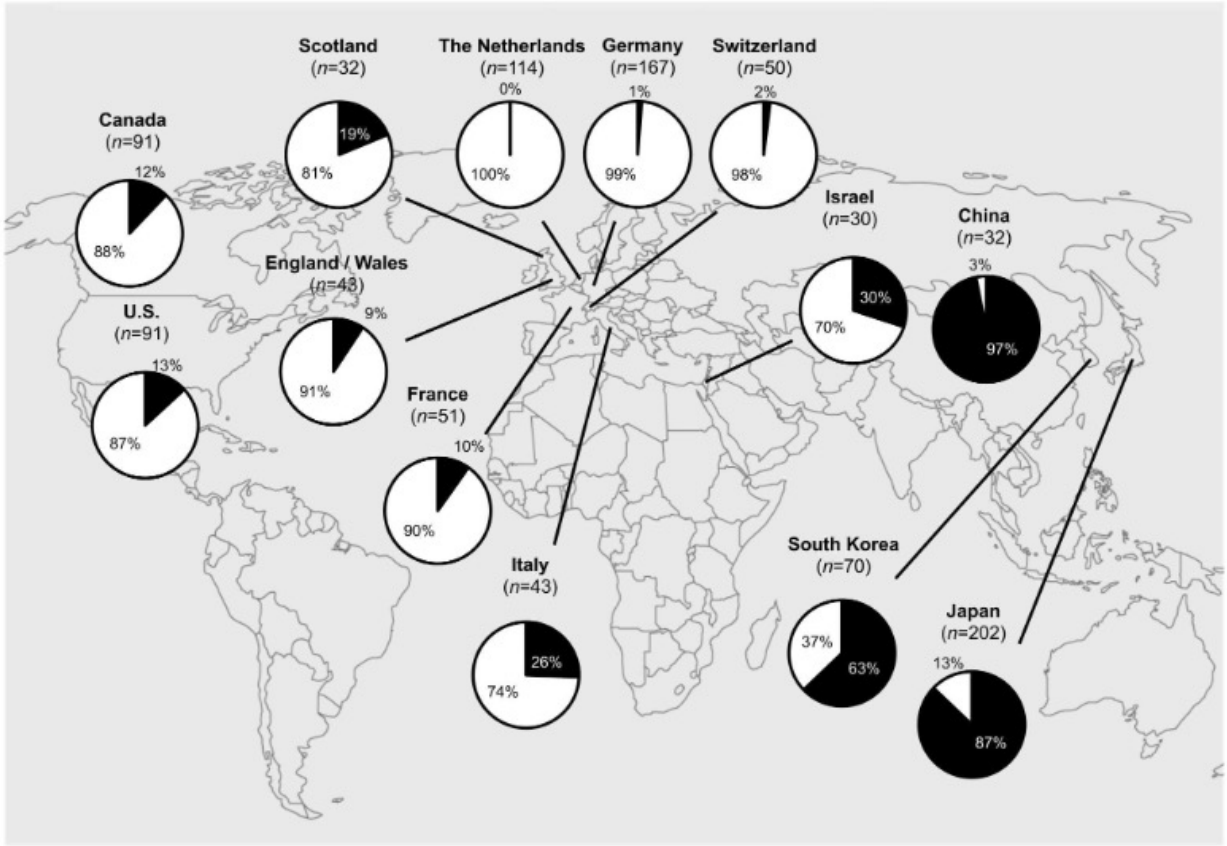


FIGURE 6 | Worldwide macrolide-resistant *M. pneumoniae* (MRMP) rates. Actual MRMP rates are punctually depicted in pie charts (in black) over the world map. **Asia:** Japan (2011): 87% (176/202) (Okada et al., 2012), South Korea (2011): 63% (44/70) (Hong et al., 2013), China (2012): 97% (31/32) (Zhao et al., 2013), Israel (2010): 30% (9/30) (Averbuch et al., 2011); **North America:** U.S. (2012–2014): 13% (12/91) (Zheng et al., 2015), Canada (2010–2012): 12% (11/91) (Eshaghi et al., 2013); **Europe:** The Netherlands (1997–2008): 0% (0/114) (Spuesens et al., 2012), Germany (2003–2008): 1% (2/167) (Dumke et al., 2010), France (2005–2007): 10% (5/51) (Peuchant et al., 2009), Italy (2010): 26% (11/43) (Chironna et al., 2011), Scotland (2010–2011): 19% (6/32) (Ferguson et al., 2013), Switzerland (2011–2013): 2% (1/50) (Meyer Sauteur et al., 2014a), England and Wales (2014–2015): 9% (4/43) (Brown et al., 2015).

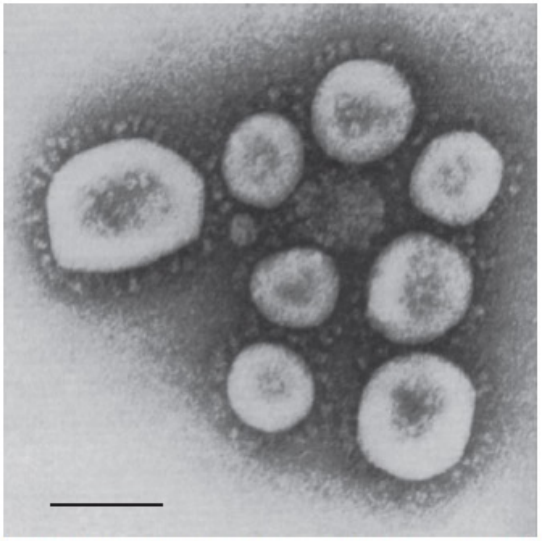


FIG. 188.1 Human coronavirus. Electron micrograph of one of the earliest coronaviruses found from an adult with a cold (OC16), negatively stained with phosphotungstic acid. The bar is 100 nm.

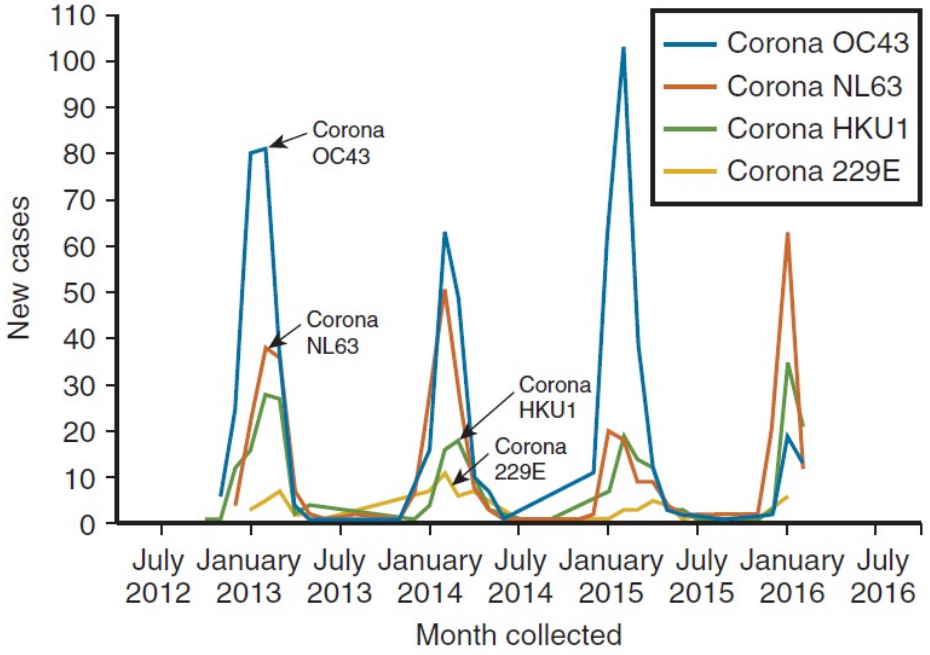


FIG. 188.2 Distribution of human coronavirus types in respiratory specimens from symptomatic inpatient and outpatient pediatric patients over 4 years at Seattle Children's Hospital using a commercial rapid multiplex polymerase chain reaction (FilmArray, Biofire Diagnostics). This demonstrates concurrent circulation of all four types of human coronavirus (HCoV) during winter months, with predominance of HCoV-OC43 in three seasons and HCoV-229E in another. (Unpublished data courtesy J. Stapp, Seattle Children's Hospital Microbiology Laboratory.)

Pediatric RESEARCH

www.nature.com/pr

SYSTEMATIC REVIEW

Coronavirus disease 2019 (COVID-19) in children and/or adolescents: a meta-analysis

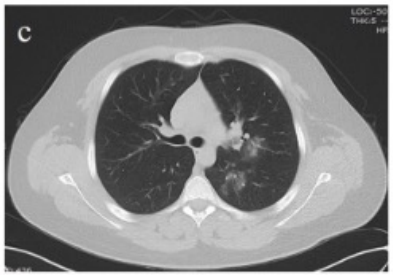
Alessandro Mantovani[1], Elisabetta Rinaldi[1], Chiara Zusi[1,2], Giorgia Beatrice[1], Marco Deganello Saccomani[3] and Andrea Dalbeni[4]

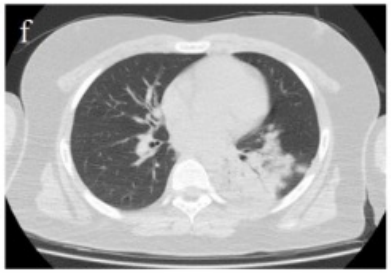
BACKGROUND: To assess the overall prevalence of clinical signs, symptoms, and radiological findings in children and/or adolescents with COVID-19.
METHODS: We systematically researched in PubMed, Scopus and Web of Science databases observational studies describing COVID-19 in children and/or adolescents until April 11, 2020. Data regarding clinical and radiological features were extracted from eligible studies and meta-analysis was performed using random-effects modeling.
RESULTS: We examined 19 eligible studies for a total of 2855 children and/or adolescents with COVID-19. Approximately 47% of subjects had fever (95% confidence interval [CI] 22–72%; $I^2 = 98.6\%$), 37% cough (95%CI 15–63%; $I^2 = 98.6\%$), 4% diarrhea (95%CI 0–12%; $I^2 = 92.2\%$), 2% nasal congestion (95%CI 0–7%; $I^2 = 87.7\%$), 1% dyspnea (95%CI 0–7%; $I^2 = 91.5\%$) and 0% abdominal pain (95%CI 0–1%; $I^2 = 76.3\%$). Subjects presented mild symptoms in 79% (95%CI 65–91%; $I^2 = 93.5\%$) of cases, whereas only 4% (95%CI 1–9%; $I^2 = 76.4\%$) were critical. Among those with pneumonia on computed tomography, 26.4% (95%CI 13–41%; $I^2 = 80.8\%$) presented a unilateral involvement, 16% (95%CI 5–29%, $I^2 = 81.2\%$) had bilateral involvement and 9% (95%CI 0–24%; $I^2 = 88.7\%$) had interstitial pneumonia.
CONCLUSIONS: Children and/or adolescents tend to have a mild COVID-19 course with a good prognosis.

Pediatric Research _####################_; https://doi.org/10.1038/s41390-020-1015-2

IMPACT:

- Compared to adults, children and/or adolescents tend to have a mild COVID-19 course with a good prognosis.
- This study provides new and consistence information on the clinical and radiological characteristics of COVID-19 in pediatrics.
- This study may help to fight COVID-19 in pediatric population.





Research

JAMA Pediatrics | Original Investigation

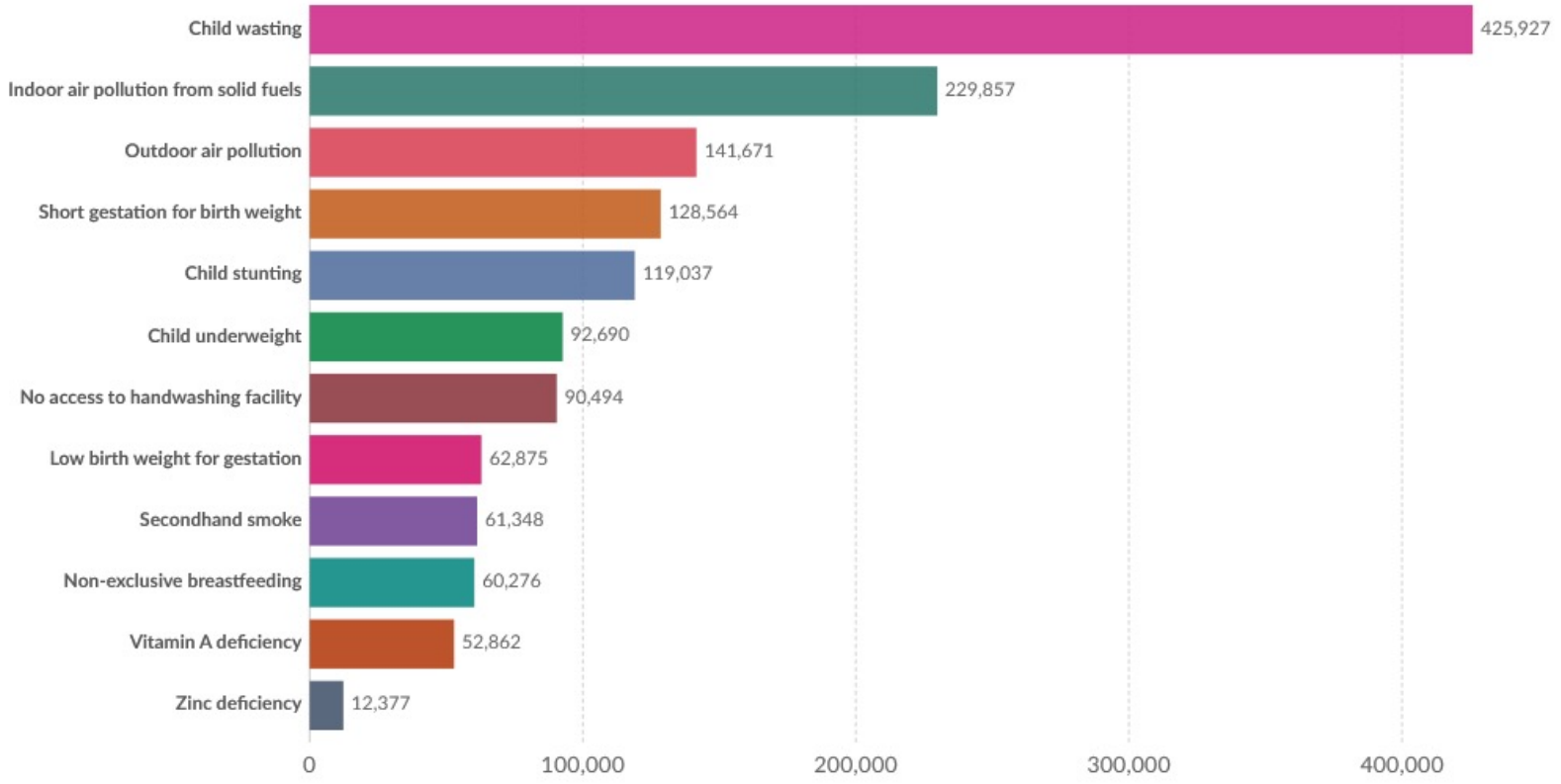
Susceptibility to SARS-CoV-2 Infection Among Children and Adolescents Compared With Adults
A Systematic Review and Meta-analysis

Russell M. Viner, PhD; Oliver T. Mytton, PhD; Chris Bonell, PhD; G. J. Melendez-Torres, PhD; Joseph Ward, MBBS; Lee Hudson, PhD; Claire Waddington, DPhil; James Thomas, PhD; Simon Russell, PhD; Fiona van der Klis, PhD; Archana Koirala, MBChB; Shamez Ladhani, MD; Jasmina Panovska-Griffiths, PhD; Nicholas G. Davies, DPhil; Robert Booy, MD; Rosalind M. Eggo, PhD

Child deaths from pneumonia by risk factor, World, 2017

Shown is the number of deaths of children younger than five from pneumonia and other lower respiratory diseases by risk factor that was estimated to be responsible for these deaths.

⇄ Change country



Source: IHME, Global Burden of Disease Study (2017)

Note: Risk factors can interact and overlap for a given cause of death; therefore the sum of deaths attributed to risk factors will exceed the total number of deaths from pneumonia.

American Journal of Epidemiology
Vol. 180, No. 10
DOI: 10.1093/aje/kwu234
Advance Access publication:
October 16, 2014

Original Contribution

Air Pollution and Acute Respiratory Infections Among Children 0–4 Years of Age: An 18-Year Time-Series Study

Lyndsey A. Darrow*, Mitchel Klein, W. Dana Flanders, James A. Mulholland, Paige E. Tolbert, and Matthew J. Strickland

Environmental Science and Pollution Research (2019) 26:3208–3225
https://doi.org/10.1007/s11356-018-3769-1

REVIEW ARTICLE

Understanding the effect of indoor air pollution on pneumonia in children under 5 in low- and middle-income countries: a systematic review of evidence

Enemona Emmanuel Adaji[1] · Winifred Ekezie[1] · Michael Clifford[2] · Revati Phalkey[1,3]

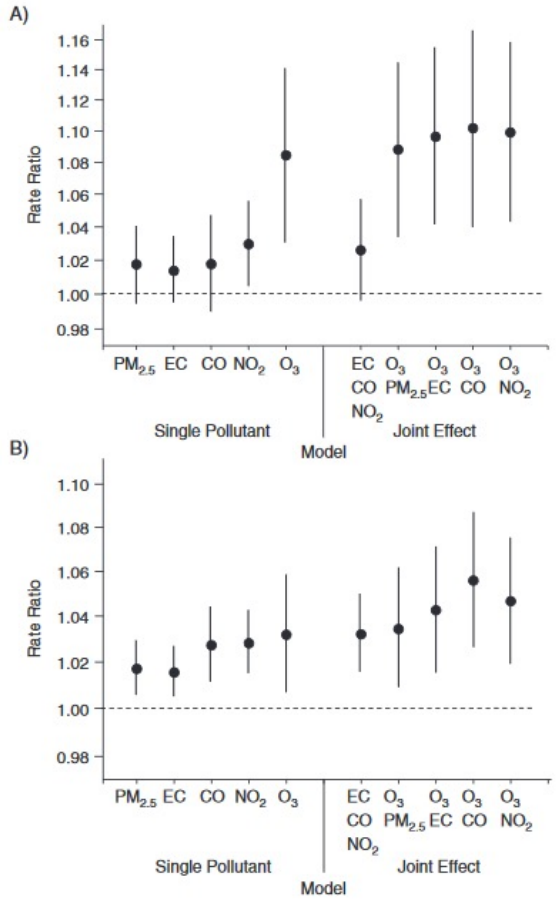


Figure 2. Rate ratios per 1–interquartile range increase in ozone (O_3), nitrogen dioxide (NO_2), carbon monoxide (CO), particulate matter less than 2.5 μm in diameter ($PM_{2.5}$), and the elemental carbon fraction of $PM_{2.5}$ (EC) from single-pollutant models and joint-effects models (for a combined increase in the interquartile range of all pollutants listed) for A) pneumonia and B) upper respiratory infection, Atlanta, Georgia, 1998–2010. All estimates are based on 4,186 observation days with measurements for all 5 pollutants. Bars, 95% confidence intervals.

Jones et al. Respiratory Research 2011, 12:5
http://respiratory-research.com/content/12/1/5

RESPIRATORY RESEARCH

RESEARCH Open Access

Parental and household smoking and the increased risk of bronchitis, bronchiolitis and other lower respiratory infections in infancy: systematic review and meta-analysis

Laura L Jones[1*], Ahmed Hashim[1], Tricia McKeever[1], Derek G Cook[2], John Britton[1], Jo Leonardi-Bee[1]

8.1.5 Pneumonia

Hassan 2001	2.16 [1.42, 3.28]
Suzuki 2009	1.55 [1.25, 1.92]
Victora 1994	0.94 [0.72, 1.22]
Subtotal (95% CI)	**1.43 [0.93, 2.21]**

Heterogeneity: Tau² = 0.12; Chi² = 13.87, df = 2 (P = 0.0010); I² = 86%

Test for overall effect: Z = 1.63 (P = 0.10)

Total (95% CI) **1.54 [1.40, 1.69]**

Heterogeneity: Tau² = 0.04; Chi² = 94.60, df = 36 (P < 0.00001); I² = 62%

Test for overall effect: Z = 8.85 (P < 0.00001)

Test for subgroup differences: Chi² = 29.50, df = 4 (P < 0.00001), I² = 86.4%