Chlamydophila pneumoniae (CPN)

The most dangerous germ?

How do you calculate how dangerous a germ is? You have to consider (a) how awful its effects are and (b) how likely you are to become infected.

For example, the Ebola virus is pretty awful. It kills most of its victims, and it kills them horribly. It is one of the most lethal germs we know of, but your chances of getting Ebola are pretty slim.

CPN, on the other hand, seems so innocuous that your doctor is not likely to know or care very much about it. Almost everyone who lives to the age of sixty will be infected by CPN, and it will kill many of the people it infects.

CPN takes decades to kill its victims, however, so it doesn't generate much excitement in the press. It doesn't kill beautiful young people, it kills people in their middle and later years. It's not glamorous; we are not likely to ever see a movie about courageous young researchers braving the hazards of an old folk's home to save the natives from a CPN outbreak.

As boring as CPN is, if you are middle aged or elderly, and you live in a prosperous Western county, CPN is more dangerous to you than the Ebola virus. A lot more dangerous.

Chlamydophila-related disorders

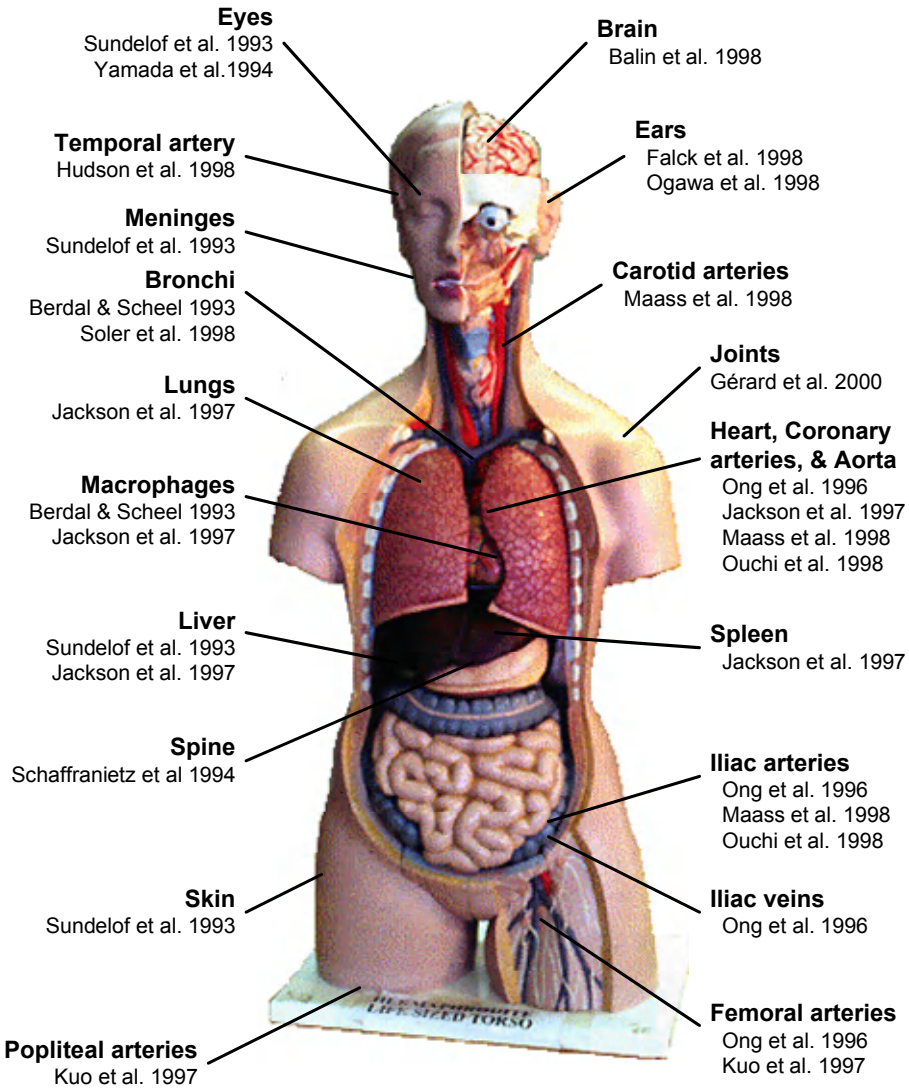
Here's a short list of diseases that have been linked to CPN:

- Alzheimer's
- Arthritis
- Asthma
- **Atherosclerosis**
- Atrial fibrillations
- Benign prostatic hyperplasia
- Bronchitis
- CFIDS
- COPD
- **Type 2 Diabetes**
- Earache
- Encephalitis
- Endocarditis
- Erythema nodulosum
- Eye problems
- Giant-cell arteritis
- Guillain-Barre syndrome
- Hypertension
- Immune suppression
- Interstitial cystitis
- Kidney failure
- Lung cancer
- Meningitis
- Morgellons
- Multiple sclerosis
- Myocarditis
- **Obesity**
- Pericarditis
- Pharyngitis
- Pneumonia
- Porphyria
- Prostate cancer
- Prostatitis
- Pyoderma gangrenosum
- Sinusitis
- SUDS--Sudden unexpected death syndrome
- Syndrome X
- Vasculitis

Further research may remove some of these disorders from the list, but others may be added. You can look most of these up on PubMed at http://www.pubmed.gov. You can find a longer list at: http://www.cpnhelp.org/?q=Cpndiseases.

Body Parts

CPN can be found in many different parts of the body, as shown below.



Organs and tissues infected by *Chlamydia pneumoniae*.

Picture of cutaway torso courtesy of Dr. Murray Thompson, University of Western Sydney

Monocyte Stickiness and heart disease

Monocytes are large immune cells that eventually turn into macrophages, the garbage collectors of the immune system. Before they turn into macrophages, monocytes drift along in the bloodstream, eating a germ here, a dead cell there, and generally behaving themselves. In the course of their travels they brush against the endothelial cells in our arteries, bounce off, and continue to drift. Occasionally, however, a monocyte will stick to an endothelial cell, then creep into the wall of the artery where it turns into a macrophage and where, sometimes, it becomes a foam cell. When millions of macrophages have become foam cells in one area, we have an atherosclerotic plaque.

Researchers from the University of Wisconsin designed a study to find out why some monocytes, but not others, stick to endothelial cells. They found that monocytes infected with CPN were stickier than uninfected monocytes.[1] Furthermore, the more heavily infected the monocytes were, and the longer they had been infected, the stickier they became. The CPN didn't have to be alive--monocytes that ate dead CPN organisms also became sticky.

Treating *Chlamydophila* infections

CPN is extremely difficult to eradicate because its life-cycle includes three forms (described below), and it is hard to kill all of them at one time.

Chapter 3 of *The Potbelly Syndrome* tells how to obtain "protocols" for eradicating CPN infections. The protocols were originally developed by Charles Stratton, M.D., and William Mitchell, Ph.D., at Vanderbilt University. Dr. David Wheldon, a British medical microbiologist, has developed an anti-CPN protocol that is tailored to people with multiple sclerosis. The latest versions of these protocols can be found at: http://www.cpnhelp.org/?q=cpnbook.

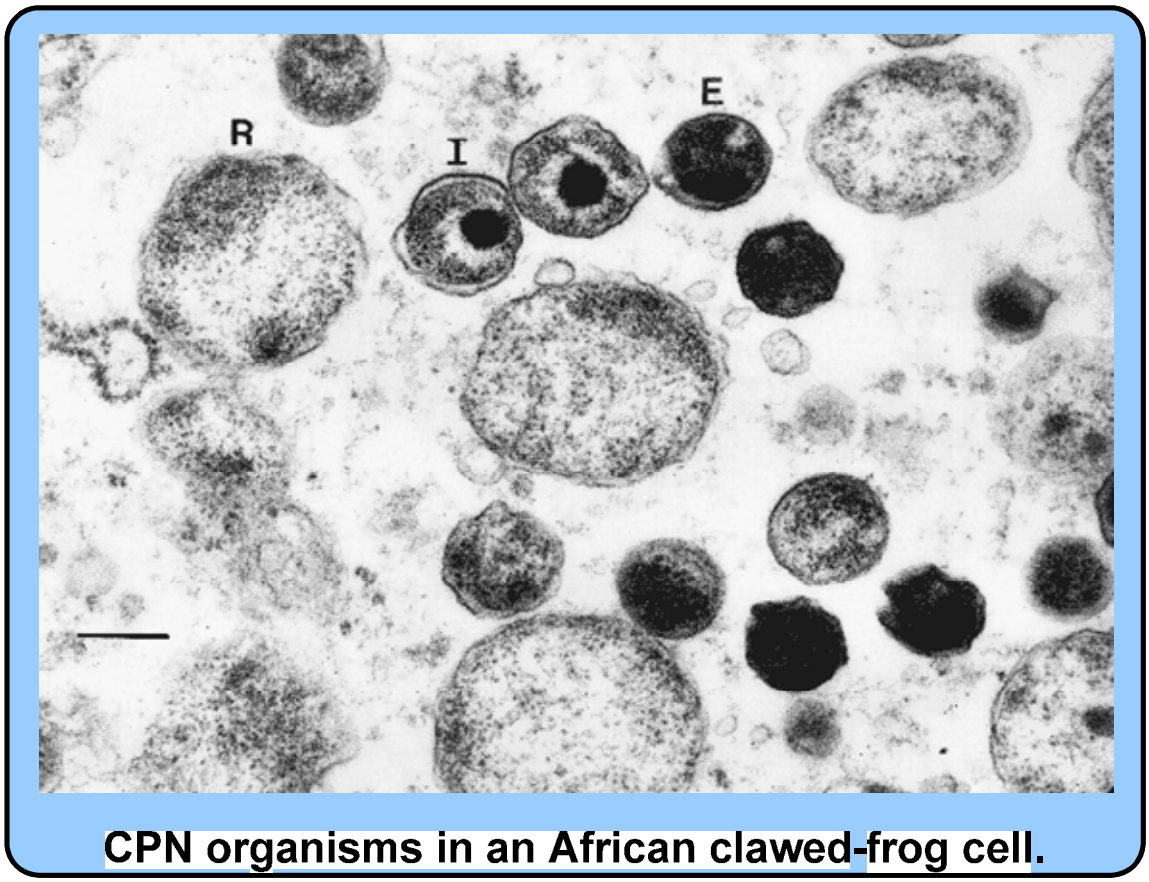
Note: ***Chlamydophila pneumoniae*** **(CPN) used to be called** ***Chlamydia pneumoniae*****, and it is a distant cousin of the sexually-transmitted** ***Chlamydia trachomatis*** **(CTR). CPN and CTR are very different germs, but doctors still get them mixed up with each other because of the similar names.**

Chlamydophila's three forms

One reason that CPN is so hard to kill is that it exists in three forms. The elementary body (EB) is a very small, spore-like form. It is

biologically inactive, and it does not generate much activity from our immune systems. When EBs come in contact with certain cells, the cells draw the EBs inside. If conditions are right, the EBs change into reticulate bodies (RBs) and begin to reproduce. When the host cell is full of RBs, the RBs change into intermediate bodies (IBs). The IBs quickly change into new Ebs. The host cell ruptures, and the Ebs spread out to repeat the cycle.

The photo below shows all three forms together in a single cell from a frog. The Ebs, RBs, and IBs in our cells look just like the ones in the frog cell. This just happens to be a particularly good picture.



CPN organisms in an African clawed-frog cell.

Note and legend:
Photo courtesy of Reed et al. from an article in *Emerging Infectious diseases*.
E = Elementary body (EB), the spore like form of CPN. It is about the same size as a smallpox virion. It is biologically inactive until it becomes a reticulate body. EBs are impervious to most antibiotics.
I = Intermediate body (IB), a temporary form of CPN that exists briefly during the transition from an RB to an EB.
R = Reticulate body, the growing and reproductive form of CPN. It can be killed by many antibiotics.

Aneurysms and *Chlamydophila*

Aneurysms are balloon-like swellings in arteries that often rupture and cause death from internal bleeding. Many strokes are caused by ruptured aneurysms in the brain.

CPN causes aneurysms as well as arterial blockages. In 1996, Ong et al. reported finding CPN in 11 of the 25 abdominal aortic aneurysms (AAAs) they examined.[2] Since then more than a dozen researchers have found CPN in aortas. Here are some of their findings:

- IgA levels do not give a very accurate indication of the severity of a person's infection with CPN, but they are not completely useless, either. Lindholt et al. found that, over a period of a few years, the AAAs of men with high IgA levels expanded 75% more than did the AAAs of uninfected men.[3] [4]
- Researchers from Gavle, Sweden were able to detect CPN in 20 of 26 sections of aneurysms removed from patients. They were able to grow CPN from 10 of those sections in vitro.[5]
- Patients with AAAs are six times as likely as controls to be infected with CPN.[6]
- Mice infected with cytomegalovirus (CMV) develop sores in their aortas, and these sores are much worse if the mice are also infected with CPN.[7]
- Researchers from the Netherlands found CPN in 67% of the human aortas they examined *post mortem*. They also found CPN in other arteries.[8]

Additional information on CPN

There is more information on CPN in *The Potbelly Syndrome* and on other pages of **http://www.potbellysyndrome.com**. There are almost 3000 articles on *Chlamydophila pneumoniae* at the PubMed website: **http://www.pubmed.gov**.

Disclaimer

http://www.potbellysyndrome.com, including this page, is maintained by Russell Farris, and the information contained here is based upon the research and personal and professional experiences of Russell Farris. It is not intended as a substitute for consulting with your physician or other healthcare provider. Any attempt to diagnose and treat an illness should be done under the direction of a healthcare professional.

Russell Farris does not advocate the use of any particular healthcare protocol but believes the information in this website should be available to the public. Russell Farris is not responsible for any adverse effects or consequences resulting from the use of the suggestions, preparations, or procedures discussed in this website. Should the reader have any questions concerning the appropriateness of any procedures or preparation mentioned, the web owner strongly suggests consulting a professional healthcare advisor.

References

[1] Kalayoglu MV, Perkins BN, Byrne GI. Chlamydia pneumoniae-infected monocytes exhibit increased adherence to human aortic endothelial cells. Microbes Infect 2001 Oct;3(12):963-9

[2] Ong G, Thomas BJ, Mansfield AO, Davidson BR, Taylor-Robinson D Detection and widespread distribution of Chlamydia pneumoniae in the vascular system and its possible implications. J Clin Pathol 1996 Feb;49(2):102-6

[3] Lindholt JS, Juul S, Vammen S, Lind I, Fasting H, Henneberg EW. Immunoglobulin A antibodies against Chlamydia pneumoniae are associated with expansion of abdominal aortic aneurysm. Br J Surg 1999 May;86(5):634-8

[4] Lindholt JS, Ashton HA, Scott RA. Indicators of infection with Chlamydia pneumoniae are associated with expansion of abdominal aortic aneurysms. J Vasc Surg 2001 Aug;34(2):212-215

[5] Karlsson L, Gnarpe J, Naas J, Olsson G, Lindholm J, Steen B, Gnarpe H. Detection of Viable Chlamydia pneumoniae in Abdominal Aortic Aneurysms. Eur J Vasc Endovasc Surg 2000 Jun;19(6):630-635

[6] Blanchard JF, Armenian HK, Peeling R, Friesen PP, Shen C, Brunham RC. The relation between Chlamydia pneumoniae infection and abdominal aortic aneurysm: case-control study. Clin Infect Dis 2000 Jun;30(6):946-7

[7] Burian K, Berencsi K, Endresz V, Gyulai Z, Valyi-Nagy T, Valyi-Nagy I, Bakay M, Geng Y, Virok D, Kari L, Hajnal-Papp R, Trinchieri G, Gonczol E. Chlamydia pneumoniae Exacerbates Aortic Inflammatory Foci Caused by Murine Cytomegalovirus Infection in Normocholesterolemic Mice. Clin Diagn Lab Immunol 2001 Nov;8(6):1263-6

[8] Vink A, Poppen M, Schoneveld AH, Roholl PJ, de Kleijn DP, Borst C, Pasterkamp G. Distribution of Chlamydia pneumoniae in the Human Arterial System and Its Relation to the Local Amount of Atherosclerosis Within the Individual. Circulation 2001 Mar 27;103(12):1613-1617