overcome deformities caused by leprosy. Priest, PANACEA’s president, welcomes both financial help and the professional support of physicians interested in short-term relief work in India. Inquiries should be sent to PANACEA, 201–60 Richmond St., New Westminster, BC V3L 5R7; 604 540-8464 (phone and fax).

### Research Update • Mise à jour de la recherche

#### Catching a culprit in the act

Two rabbits with atherosclerosis have provided the first direct evidence that a common bacterial infection is a major culprit in heart disease. In a study published in the *Journal of Clinical Microbiology* (1997;35[1]:48-52), researchers at St. Michael’s Hospital in Toronto, the University of Toronto and McMaster University in Hamilton, Ont., showed that a human isolate of *Chlamydia pneumoniae* caused atherosclerotic plaques to form in the aortas of some infected rabbits. Of 11 rabbits infected, one had a fatty streak and evidence of *C. pneumoniae* in the aorta and another had a grade III atherosclerotic lesion, from which *C. pneumoniae* was cultured. There was no atherosclerosis in a group of control rabbits.

Finding 2 rabbits with heart disease may not sound like a groundbreaking result, but it provides a key piece of the puzzle surrounding the pathogenic basis for atherosclerosis.

“This was just a pilot study,” explains Dr. Bill Fong, but he and his associates, Dr. Brian Chiu, Esther Vira, Michael Fong, Dan Jang and Dr. James Mahony, have since confirmed these results in a larger rabbit study, as yet unpublished. “The difference [between infected and control groups] was highly significant, so we’re very confident about our results.”

Using a human isolate of a microorganism to reproduce a disease in an animal and subsequently recovering the organism from the experimental disease are crucial steps in establishing that an organism causes a particular disease, according to the Henle–Koch postulates.

*Chlamydia pneumoniae*, first described 12 years ago as a cause of community-acquired pneumonia, is a very common infection from childhood onward. In addition to pneumonia, it may cause sinusitis, bronchitis and cold-like symptoms. Fong estimates that 60% to 80% of adults have antibodies to the bacterium. Seroprevalence studies have linked previous infection with a risk of atherosclerosis. In previous studies, the organism has been isolated from plaques in an average of 45% to 50% of patients with atherosclerosis. Fong says his group has found the organism in the plaques of up to 70% of such patients.

*Chlamydia pneumoniae* probably plays an important role in atherosclerosis. We think that the disease is multifactorial. Infection and lifestyle factors may act together consecutively or sequentially. The infection may cause damage to the blood vessels, and then lifestyle factors may contribute to more mature atherosclerosis.

The researchers are planning studies in which infected and uninfected rabbits will be fed foods with cholesterol levels comparable to the human diet.

Fong says he and his colleagues are “very close” to a clinical application for their research. There are plans to try to reverse the disease with antibiotic therapy in animals and then in humans. Fong is concerned that antibiotics may not do much good once the infection has damaged blood vessels. More promising is a possible vaccine. Animal studies have indicated that natural infection is not completely protective, but multiple-dose vaccination may provide continuing protection in humans. — C.J. Brown

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The smoking gun: grade III atherosclerotic lesion in the aorta of a rabbit. Avidin-Biotin immunohistochemical stain with Chlamydia Cel Pn. Hematoxylin counterstained; arrow shows positive result in nodule of vascular smooth-muscle cells. Original magnification 100×.