

## Indication of infectious cause

Of all the forms of arthritis, rheumatoid arthritis is the most crippling and puzzling in terms of its causes. One theory which is gaining in popularity is that the disease is caused by an infectious agent. The fact that rheumatoid arthritis is accompanied by inflammation, fever, lack of appetite, weight loss and a high white blood cell count, symptoms similar to those of infection, supports this theory.

Some scientists feel that mycoplasma, a type of microorganism somewhere between bacteria and viruses in size, may be the agent responsible for the disease. Mycoplasma, unlike bacteria, do not have cell walls and can exist in a variety of forms; unlike viruses, they do not rely on living cells for reproduction.

Although evidence linking the mycoplasma to rheumatoid disease is fragmentary, mycoplasma have been isolated from many domestic animals and are known to cause arthritic diseases in animals. However, no human strain of mycoplasma has yet been specifically related to the disease.

**One reason** for this, according to Dr. Thomas M. Brown of George Washington University, is that no animal has ever come down with human rheumatoid arthritis, so no animal model has existed for study of the mycoplasma-arthritis link in relation to human disease. And in humans, these organisms are difficult to culture by present methods; occasionally some infectious agent will trigger the disease, but will disappear from affected joints.

Now an animal—a gorilla—has been found to have a disease which appears to be human rheumatoid arthritis, and a specific link to mycoplasma seems to have been established, though not everyone is convinced.

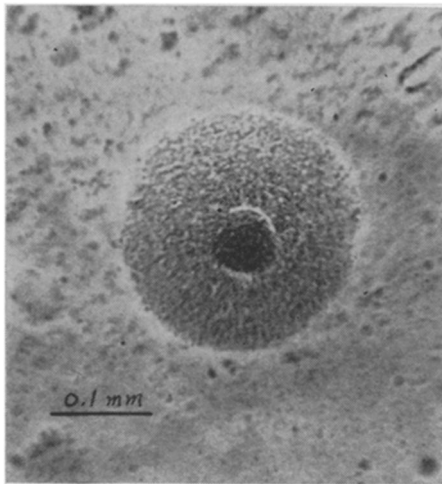
The controversy is being generated by a suspected new strain of mycoplasma isolated from a throat culture taken from an eight-year-old gorilla named Tomoka, an apparent victim of rheumatoid arthritis, living at the National Zoo in Washington, D.C.

Dr. Brown told the Interscience Conference on Antimicrobial Agents and Chemotherapy in Washington this week that the new strain, called TK, had cross-reactivity to the human strain, *M. salavarium*.

**The gorilla** is a better animal model than any yet available. It is serologically one of man's closest relatives. In addition, Tomoka had a two-and-a-half year history of illness similar to rheumatoid arthritis. The findings confirming the diagnosis were stunted growth, elevated serum gamma globulin



Photos: George Washington Univ.  
*Tomoka: Focus of arthritis research.*



*Mycoplasma from Tomoka's throat.*

(one of the body's chief antibodies), positive rheumatoid test and a non-specific inflammatory reaction in the joint tissue similar to that of arthritis.

Specific antibodies to the gorilla mycoplasma were detected during the course of antibiotic treatment, with which Dr. Brown says he achieved remission. "The fact that antibodies emerged indicates the disease is infectious and that mycoplasma may be the causative agent," he declares.

Primarily because animal strains of mycoplasma respond to antibiotics, arthritis conditions in domestic animals have been controlled, Dr. Brown says.

Some scientists are more skeptical and feel that the mere isolation of a mycoplasma, regardless of the presence of antibodies, does not indicate it is the causative agent.

Nevertheless, Dr. Brown plans to inject the mycoplasma in other apes to see if the disease can be reproduced.

Dr. John T. Sharp of Baylor University School of Medicine says Dr. Brown's findings add more evidence to the theory that the disease is caused by a mycoplasma. "From a long-range

view, it can be predicted that some day manifestation of some form of arthritis will be shown to be due to mycoplasma but that it is as yet too early to be confirmed. Even if the disease was caused by the mycoplasma, 14 or 15 diseases in animals are due to mycoplasma, and this may just be another animal disease. Arthritis is too ill-defined, and too many cases cannot be diagnosed," he says. □

## ECONOMICS

### Models bring Nobel Prize

The Swedish Academy of Sciences this week awarded the first Nobel Prize in Economics to Prof. Jan Tinbergen of the Netherlands and Prof. Ragner Frisch of Norway. The award, which was instituted last year in connection with the 300th anniversary celebration of the Swedish National Bank, cited Drs. Tinbergen and Frisch "for having developed and applied dynamic models for the analysis of economic processes."

Drs. Tinbergen and Frisch were pioneers in the attempt to quantify economic processes so that they could be analyzed by mathematical techniques; their econometric techniques were then applied to the problems of central planning.

Once a sufficiently complete mathematical model of an economy has been developed, its future performance can be predicted, under various conditions, by changing the right variables within the equations and analyzing the results.

**Prof. Tinbergen** constructed a working mathematical model of the economy of the Netherlands which is given credit for much of the postwar economic success of that country.

The Government of the Netherlands still makes an annual forecast of the country's growth rate based on Prof. Tinbergen's model. The success of Prof. Tinbergen's econometric model was also an important indication of the possibility of combining detailed central planning with democratic processes.

Prof. Frisch was the editor of *ECONOMETRICA*, for years the principal journal devoted to the theories of mathematical economics. Until his retirement in 1965, Dr. Frisch was the director of the Institute for Social Economy at the University of Oslo, and an influential teacher, having trained the majority of the postwar economists working for the Norwegian Government.

He is currently working on an econometric model to be used in wage negotiations between the Norwegian Trade Union Organization and the Employers Association. The model would answer such questions as the probable effect of Government subsidies to agriculture on industrial wages. □