News
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******Top fever researchers gather in Dallas April 11 and 12.

DALLAS--As every mother knows, fever is a sure sign of disease. But despite its familiarity, the fever mechanism itself remains a medical mystery.

Researchers still do not understand the details of how fevers begin and end. And the scientists are unable to fully explain how aspirin and other "antipyretics" act to lower high body temperatures.

At the International Symposium on Fever--which is to be held Wednesday and Thursday, April 11 and 12, at the Dallas Fairmont Hotel--the world's top fever experts will gather to discuss recent developments in this important field of research.

At a 2 p.m. Wednesday session in the Parisian Room, Dr. James Lipton, associate professor of physiology and neurology at The University of Texas Health Science Center at Dallas, will present new findings that may help to explain both the fever mechanism and the effects of aspirin. The new data might even prove to be useful in development of a better antipyretic--one without the side effects produced by those now in use.

According to the current generally accepted model, fever results when an infection or some other reaction causes the body to produce a mysterious substance called "pyrogen," explains Dr. Lipton. The blood then carries this substance--which thus far hasn't been chemically identified--to the brain where it stimulates the body's "thermostat," a group of neurons responsible for temperature control. Thus, the level of fever depends on the amount of pyrogen circulating in the bloodstream.

"Nobody really knows how aspirin and the other antipyretics work," Dr. Lipton continues. One theory is that the aspirin molecules grab on to the receptor sites on the thermostat's nerve cells that are normally reserved for pyrogen molecules, thus blocking the pyrogen's effects.

But this theory does not explain what happens to pyrogen molecules that do find unoccupied receptors, which is normally the case. If there wasn't a mechanism for stopping the action of pyrogen in the brain, once a fever began it would persist indefinitely, Dr. Lipton points out. Since this does not occur, the brain must somehow be inactivating the pyrogen.

Dr. Lipton's findings indicate that a "facilitated transport" process plays a key role in stopping fever and that antipyretics reduce fever by enhancing this process. In other words, aspirin inactivates pyrogen by aiding in its removal from receptor sites on thermostat cells.

"The idea that transport of pyrogen is essential for its inactivation is new," Dr. Lipton says. "This data should be useful in determining how untreated fever stops, how high levels of fever develop when the transport process fails, and how antipyretics bring down fever."

The International Symposium on Fever is sponsored by the American Physiological Society and co-sponsored by The University of Texas Health Science Center at Dallas and McNeil Consumer Products.

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