It’s a taxing time for air travelers

Airline passengers are giving an ever-increasing portion of their travel dollars to Uncle Sam, according to data released by MIT’s Global Airline Industry Program and Daniel Webster College.

Airline ticket prices overall have actually dropped over the past several years, the researchers emphasize. However, many of the taxes and fees passengers pay, which fund a significant portion of the costs of U.S. air-traffic control and airport systems, are not linked to the base price of the tickets and have remained about the same.

As a result, the effective tax rate on airline tickets is steadily increasing, and will increase even more under the Bush administration’s recently released federal budget proposal, researchers report.

Which raises the question: Who should pay for the increases? The airlines or U.S. taxpayers?

“The Bush administration’s proposed increase in the security fee added after September 11, 2001, has generated strong reactions from the airline industry,” said MIT Professor Amedeo Odoni, the project’s director. “The increased fees will place further strain on the airlines at a time when several of them are struggling. On the other hand, it is difficult to argue that taxpayers at large should subsidize the security costs of airline customers.”

Odoni believes that this year’s $600 million increase was 16.5 percent of the airline ticket tax. This is up from 15.5 percent in 2002 and 10.5 percent in 1996.

Professor Joakim Karlsson of Daniel Webster College explains the significance of the study’s results: “The airlines have lost the ability to raise fares, even to just keep pace with inflation. The average round-trip ticket has dropped 40 percent in real terms since 1993. Meanwhile, average ticket taxes and fees have stayed relatively unchanged, on top of the ticket price, the researchers found in their update of last year’s study.”

In 2004, passengers paid 16.1 percent in taxes and fees on top of the price of a domestic ticket. This is up from 15.5 percent in 2002 and 10.5 percent in 1996.

Buddhists help rebuild Sri Lanka

Sri Lankan families left homeless by the tsunami could find themselves living in an enclave of MIT-designed housing if the efforts of MIT’s Buddhist chaplain and a researcher in the Department of Urban Studies and Planning are successful.

And there’s every reason to believe they will be.

Immediately after the Dec. 26 tsunami hit the chaplain, Tenzin L. S. Priyadarshini, formed an alliance between MIT’s Buddhist community and the Prajnapa Foundation, which is working with the Sri Bodhiraja Foundation in Sri Lanka to collect funds for rebuilding permanent housing near the Sri Lankan seashores. Priyadarshini and the Prajnapa Foundation, along with the Committee of World Religions for Tsunami Efforts in Taipei, Taiwan, raised more than $300,000 by Jan. 15. On Feb. 27, the alliance dedicated 25 new homes, each built for about $12,500. They hope to build 1,000 homes altogether.

“Many people are still thinking about how to relocate the victims and how to build camps for the victims. But because of the Buddhist monks’ involvement in this project, some new homes have already been built and are housing families,” said Priyadarshini, who is a Buddhist monk from India. He explained that the Buddhist monks in Sri Lanka wield a great deal of influence with the Sri Lankan government, making it possible for them to work quickly.

The houses built by the alliance are being assigned to families on an as-needed basis, with the first homes going to single mothers who lost their husbands in the tsunami.
Researchers identify target for cancer drugs

David Cameron
Whitewater Institute

Researchers at the Whitehead Institute and MIT have discovered a missing piece to the puzzle of how certain cancers work.

For nearly a decade, scientists have been trying to fully understand a particu-
lar molecular communication pathway inside of cells that contributes to many malignant brain and prostate cancers. While scientists have identified elements of this pathway, other key components have remained a mystery.

The new finding may present drug mak-
ers with a significant new cancer target.

“We believe we have identified a component that researchers have been look-
ing for since 1996,” says Whitehead Associate Member David Sabatini, who is also an assistant professor of biology at MIT.

At the heart of this new research is a protein called Akt, an important player in the regulation of cell division and survival.

Abnormally high activation of Akt has long been implicated in a variety of cancers. If Akt travels to the cell membrane, it is switched on and promotes cell division, often contributing to tumor growth. However, as long as it stays within the cell cytoplasm, it remains relatively inactive. That’s because the tumor-suppressor protein PTEN keeps Akt in check by destroying lipids in the cell membrane that normally draw Akt to the face. In essence, PTEN keeps a leash on Akt and thus suppresses cell division.

But when PTEN is mutated and unable to function, Akt breaks free. It makes its way to the cell membrane where other proteins activate it, thereby enabling Akt to contribute to tumor growth. “When a cell loses PTEN through, say, a mutation, Akt goes gangbusters,” says Sabatini.

The exact means by which Akt switches on when it reaches the cell membrane has not recently been understood. As a result, researchers have lacked a clear idea about how to prevent the process. However, in the Feb. 18 issue of Science, researchers from the Sabatini lab report discovering an important missing piece of the activation process.

This missing component, a molecule called mTOR, is a protein that influences a cell’s ability to expand in size. mTOR has been largely studied as the target for the immunosuppressant drug rapamycin (in fact, mTOR is an acronym for “mammalian target of rapamycin”). In July 2004, Dos Sarbaso, a scientist in Sabatini’s lab, dis-
covered a new protein that mTOR interacts with called rictor, but he wasn’t yet sure of what these two proteins do together.

In this latest paper, Sarbaso says that when mTOR and rictor bind and form a complex, they help activate Akt by adding a phos-
phate group to a sequence of its amino acids (a process called “phosphorylation”). According to Sarbaso, if we find a molecule that can block the mTOR/rictor complex, then we may be able to prevent Akt from becoming active and contribut-
ing to tumor formation.

This work was supported by the NIH, the Pew Charitable Trust, the Rita Allen Foundation, the Anna Fuller Fund, the Damon Runyon Cancer Research Foun-
dation, and the Howard Hughes Medical Institute.