

From •>>August 2006

*Kuan-Teh Jeang answers a few questions about this month's emerging research front in the field of **Biology & Biochemistry**.*

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## **Biology & Biochemistry**

**Article: Life, death, and tax: Role of HTLV-I oncoprotein in genetic instability and cellular transformation**

Authors: **Jeang, KT**; Giam, CZ; Majone, F; Aboud, M

Journal: J BIOL CHEM | 279 (31): 31991-31994, JUL 30 2004

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**Biology & Biochemistry**

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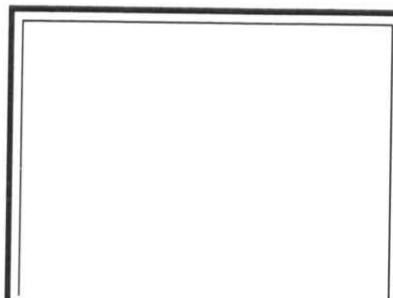
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**ST: Why do you think your paper is highly cited?**

The paper was published during a timely period. 2005 marked the 25th anniversary since the discovery of human T-cell leukemia virus type 1, HTLV-1, and this milestone probably contributed to heightened interest about the virus.

Additionally, over the past quarter century, we have come to appreciate the intimate involvement of the HTLV-1 Tax oncoprotein in many functional aspects of cellular metabolism, including NF- $\kappa$ B activation, creation of genomic instability, and dysregulation of cell cycle checkpoints. These are highly investigated areas of research, and papers on these topics



frequently cite findings from HTLV-1 Tax.

**ST: Does it describe a new discovery, methodology, or synthesis of knowledge?**

The paper synthesizes extant and recent findings on the functions of the viral Tax oncoprotein.

**ST: Could you summarize the significance of your paper in layman's terms?**

There are only five or six viruses that cause human cancers. HTLV-1 is one of these viruses. What we described in this paper are the different ways that scientists currently think as to how the virus makes a normal white blood cell into a leukemic (i.e., cancerous) blood cell.

**ST: How did you become involved in this research, and were any problems encountered along the way?**

I have been studying HTLV-1 for 20 years. I first began studying herpes viruses in graduate school.

In the 1980s, when human retroviruses were first discovered and HTLV-1 was established by Japanese and American scientists as the causative agent for adult T-cell leukemia, this virus caught my interest because I wanted to understand how viruses cause cancer.

One confounding problem with HTLV-1 is that its oncoprotein is frequently present only in early leukemic cells, and the oncoprotein then is absent from the same cancer cells at a later time. This suggests that HTLV-1 is different from other transforming viruses in that its Tax oncoprotein is required to initiate transformation but not to maintain transformation. Later, we came up with an explanation for this conundrum by demonstrating Tax's extreme facility for creating genomic instability in HTLV-1 infected cells.

**ST: Are there any social or political implications for your research?**

I believe that any time you work on a human pathogen that causes cancer, you elicit general public interest. To the extent that your research potentially hastens a cure for cancer and can save human lives, your work has some level of social and political significance.▮

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*“What we described in this paper are the different ways that scientists currently think as to how the virus makes a normal white blood cell into a leukemic (i.e., cancerous) blood cell.”*

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