References


To the editor

The role of interleukin-7 and interleukin-15 in cutaneous T-cell lymphoma

Elucidation of any mechanism underlying neoplastic proliferation is welcome news. The article by Qin et al was no exception. The authors have clearly demonstrated, as well as confirmed, that interleukin-7 (IL-7) and IL-15 regulate the expression of bcl-2 and c-myb genes in cutaneous T-cell lymphoma (CTCL). It was disappointing to note, however, that Qin et al appear to be completely unaware of the underlying stimulus, which initiates the proliferation machinery in the first place. It has been recognized for some time that practically all patients with CTCL carry human T-cell lymphotropic virus-1 (HTLV-I) Tax in their circulating and skin-infiltrating lymphocytes. Such patients also have Tax mRNA and antibodies to p40 Tax, the gene product of this sequence. In addition, it has been shown that IL-15 overexpression is attributable, specifically, to Tax transactivation of its promoter, as well as other NFkappaB transcription factors (reviewed in Gitlin et al). Therefore, while the data presented by Qin et al are not disputed, the report would have been more meaningful if the underlying cause for the upregulation of IL-7 and IL-15 had not been ignored. Such information not only is of cursory interest but also may have therapeutic implications since it has already been shown that in vitro proliferation of CTCL cells can be inhibited with antisense to Tax.

Dorothea Zucker-Franklin

Correspondence: New York University School of Medicine, 550 First Ave, New York, NY 10016

Response:

Why human T-cell lymphotropic virus-1 Tax is not the cause of cutaneous T-cell lymphoma

Dr Zucker-Franklin claims that the human T-cell lymphotropic virus-1 (HTLV-I) Tax protein is the main stimulus for mycosis fungoides (MF) and Sézary syndrome (SS), which are the main forms of cutaneous T-cell lymphoma (CTCL). But this view is not shared by the vast majority of the scientific community. Initially, in the early 1990s HTLV-I Tax DNA was reported to be present in 0% to 20% of CTCL cases, and only the Zucker-Franklin group reported numbers up to 95%. To settle the issue several groups checked their methods, and in 1996 and 1997 a number of papers appeared showing that the number of HTLV-I Tax–positive cases decreased in parallel to increased stringency of the polymerase chain reaction (PCR) conditions and the numbers of controls for the reagents used. All these studies indicated that HTLV-I Tax DNA is not present in MF and SS. Even in Japan, where HTLV-I is endemic, no HTLV-I tax DNA could be detected in CTCL cells of MF and SS patients. Thus, to put it bluntly, HTLV-I Tax cannot be the main stimulus of MF or SS, as it is not present in the malignant or surrounding cells. But one cannot exclude that another virus (for example, an endogenous retrovirus, or ERV) is activated in CTCL cells and that these cells contain amplified DNA sequences that are similar to HTLV-I Tax sequences and proteins that crossreact with HTLV-I Tax.

Reasons for finding HTLV-I Tax DNA in CTCL are probably inadequate PCR conditions and misdiagnosis of CTCL. Patients with adult T-cell leukemia (ATL) often show skin lesions that resemble hypopigmented MF. Such a misdiagnosis occurred when the HUT78 and HUT102 cell lines were established. Both cell lines were initially described as SS and MF cell lines, respectively. But later it turned out that the patient from whom the HUT102 cell line had been derived died from ATL. Further examinations showed that HUT78 is a true SS cell line, whereas HUT102 is an HTLV-I–producing ATL cell line.

Udo Döbeling

Correspondence: Department of Dermatology, University Hospital Zurich, Gloriastrasse 31, Zurich, Switzerland
References
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To the editor:
The “academic cartel”: another pernicious weed in the field of academic medicine

In his timely editorial, Dr Kaushansky articulates his concerns about the various forms of research misconduct that, as he pointed out, are certainly on the increase. He considers certain types of misconduct pernicious. I would like to bring attention to another form of misconduct that has not received much attention in the scientific arena but that is, nevertheless, a pernicious development: the “academic cartel.” This is analogous to the industrial, financial, and drug cartels that use devious techniques to promote their products and to sabotage or destroy their opponents. A handful of academics and institutions decide that a particular subject or area of research is “their domain” and make conscious and conceited efforts to propagate and promote their points of view while dismissing or suppressing any differing views. They assume a self-appointed “expert” role in the area of their interest to disseminate their views. Unfortunately, these alleged experts are very often asked to referee scientific work generated by others working in their domain. Consequently, the articles supporting their point of view are accepted and published often with fantastic proclamations (“breakthrough,” “revolutionary,” “cutting edge,” etc) while suppressing works that differ from or challenge their views. In my opinion, this is pernicious and deleterious to scientific and medical research in general, especially if the subject matter has an important public health (for example, blood transfusion medicine) or clinical implication. I think it is the responsibility of the editors of various high-impact journals, which I think includes Blood, to be aware of this practice and to choose appropriate referees to review articles submitted. I also think that editors should be made accountable if they fail to curb this practice of prejudiced publication.

Muttuswamy Sivakumaran
Correspondence: Department of Haematology, Peterborough District Hospital, Thorpe Road, Peterborough, PE3 6DA, United Kingdom; e-mail: muttuswamy.sivakumaran@pbh-tr.anglo.nhs.uk

Reference
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Dorothea Zucker-Franklin

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