But Maybe Counting Is the Easiest Part

DID I MISS OUT ON SOMETHING? DID WE all finally reach agreement on what a "species" is (News Focus, "Up for the count?" by A. Lawler, 26 Oct., p. 769)? I distinctly remember, the last time I was awake, rather nasty squabbles going on among the different schools of taxonomy and various fields of science. Just about everybody had a different idea about what really counted in defining what a species is: "genetics," "morphology," "history," "range and niche," "fertility and interfertility," and others. I didn't think all the factions would ever agree.

Did all the squabbling end? Are we finally able to go out there and take a look at every living thing, put them in their proper categories, and name them and make a list? Wow!

RUSSELL HUSTED
13150 Pomerado Road, Poway, CA 92064, USA. E-mail: rrhusted4@home.com

Letters to the Editor

Letters (~300 words) discuss material published in *Science* in the previous 6 months or issues of general interest. They can be submitted by e-mail (science_letters@aaas.org), the Web (www.letter2science.org), or regular mail (1200 New York Ave., NW, Washington, DC 20005, USA). Letters are not acknowledged upon receipt, nor are authors generally consulted before publication. Whether published in full or in part, letters are subject to editing for clarity and space.

Research on Resistance to Cancer Drug Gleevec

IN A STUDY OF 11 PATIENTS WITH ADVANCED-stage chronic myeloid leukemia (or Philadelphia chromosome positive acute lymphoid leukemia) who relapsed during treatment with the kinase inhibitor STI-571 (Gleevec), our group reported *BCR-ABL* gene amplification and kinase domain mutation as potential mechanisms of resistance (*1*). Specifically, three patients had gene amplification and six patients had a point mutation resulting in a threonine-to-isoleucine change at amino acid position 315 (T315I) of the ABL kinase domain. Subsequently, Technical Comments were published from two groups (Barthe *et al.* and Hochhaus *et al.*) who reported that they were unable to detect the T315I mutation in such patients (*2*). Both groups did, however, detect kinase domain mutations in a different residue in two patients (E255K and E255V).

As described in abstracts for the 2001 Annual Meeting of the American Society of Hematology, four groups, including Hochhaus and colleagues (*3*), have now detected patients with the T315I mutation (*3*, *4*). All the groups, including ours, have detected additional kinase domain mutations. The overall contribution of these mutations versus other potential resistance mechanisms remains to be defined.

CHARLES L. SAWYERS
Department of Medicine and Molecular Biology Institute, University of California, Los Angeles, CA 90095, USA. E-mail: csawyers@mednet.ucla.edu

References and Notes

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3. S. Kreil *et al.*, Abstracts for the 43rd Annual Meeting of the American Society of Hematology, Orlando, FL, 7 to 11 December 2001, abstr. 1823. Available at http://www.abstracts-on-line.com/abstracts/hem/
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