CXCR3 expression in T cells for treatment of autoimmune and inflammatory diseases.

Joseph Barbi, Steve Oghumu, Claudio M. Lezama-Davila, and Abhay R. Satoskar

Conflict-of-interest disclosure: The authors declare no competing financial interests.

Correspondence: Dr. Abhay R. Satoskar, Department of Microbiology, The Ohio State University, 484 West 12th Ave, Columbus, OH 43221; e-mail: satoskar.2@osu.edu.

This work was supported by the National Institutes of Health (NIH) grant RO1 51328 to A.R.S.

We thank Drs Virginia Sanders, Michael Caliguiri, and Joan Durbin for critical review of the paper and for suggestions.

Contribution: J.B. and S.O. performed experiments and wrote the paper. C.L.D. analyzed data and wrote the paper. A.R.S. designed experiment, analyzed data, and wrote the paper.

References

1. Haddeland U, Sletten GB, Brandtzaeg P, Nakstad B. Impaired interleukin (IL)-4-associated generation of CCR4-expressing T cells in neonates with hereditary allergy risk. Clin Exp Immunol. 2005;139:314-322.
2. Nakajima C, Mukai T, Yamaguchi N, et al. Induction of the chemokine receptor CXCR3 on TCR-stimulated T cells: dependence on the release from persistent TCR-triggering and requirement for IFN-gamma stimulation. Eur J Immunol. 2002;32:1792-1801.
3. Kim CH, Nagata K, Butcher EC. Dendritic cells support sequential reprogramming of chemoattractant receptor profiles during naive to effector T cell differentiation. J Immunol. 2003;171:152-158.
4. Pearce EL, Mullen AC, Martins GA, et al. Control of effector CD8+ T cell function by the transcription factor Eomesodermin. Science. 2003;302:1041-1043.
5. Glimcher LH, Townsend MJ, Sullivan BM, Lord GM. Recent developments in the transcriptional regulation of cytolytic effector cells. Nat Rev Immunol. 2004; 4:900-911.
6. Suto A, Wurster AL, Reiner SL, Grusby MJ. IL-21 inhibits IFN-gamma production in developing Th1 cells through the repression of Eomesodermin expression. J Immunol. 2006;177:3721-3727.

To the editor:

Epigallocathechin-3-gallate in AL amyloidosis: a new therapeutic option?

This unusual letter comes from Heidelberg, where the first description of amyloidosis was published in 1859.[1] The first autologous transplantation of blood-derived hematopoietic stem cells was conducted here in 1985 in my university hospital department and reported in *Blood* in 1986.[2]

At the present time, I am—as an emeritus professor of internal medicine and hematology—a patient, suffering from lambda light-chain amyloidosis since 2001, when I was 72, although it was not diagnosed until 2004. The illness has peculiarities that make it worth reporting. A short-term therapy with the "Boston scheme" (4 mg melphalan daily for 21 days) showed no response. Therapy according to the "Palladini scheme" (0.22 mg/kg melphalan and 40 mg dexamethasone for 4 days every 28 days) stabilized the disease, but at high cost because of the side effects. After 14 cycles (a total of 872 mg melphalan and 2400 mg dexamethasone), "drug holidays" were recommended by the German, Italian, and American physicians in charge of my treatment.

These "holidays" have been extended since September 2006. The drug treatment was not resumed because of the apparent effectiveness of an entirely different approach: since September 2006, I have ingested daily 1.5 to 2.0 L of green tea, effective presumably because of its contents of EGCG (epigallocatechin gallate) and other phenols.

Recommendation of this "therapy" came from former members of my staff on the basis of a lecture by Prof E. E. Wanker alerting them to in vitro experiments reporting the effects of EGCG on light chains and amyloid fibrils (Ehrnhoefer DE, Bieschke J, Boeddrich A, et al; submitted manuscript). These results impressed me, a hematologist rooted deeply in the natural sciences, so I decided to follow the advice to drink green tea on a daily basis.

The results are incredible. For 20 months, the interventricular septum had remained constant at 16.5 mm, which we considered the successful effect of chemotherapy. Since September 2006, it has decreased month by month to what is at present a constant thickness of 13.2 mm. The longitudinal myocardial deformation showed a clear improvement. The light chains do not exceed 50 mg/L; the kappa/lambda ratio remains steady between 0.74 and 0.50. The renal insufficiency remains unchanged with values of creatinine at 229.84 μmol/L (2.6 mg/dL) and urea at 24.99 μmol/L (70 mg/dL). And, most surprisingly, my quality of life has improved dramatically despite my now 78 years of age.

Since I am experiencing the objective and subjective improvements of my health, I am encouraged to present my case for your consideration. Because of the rarity of the disease, international clinical studies in amyloidosis should take note of this singular case, as with patients with lymphoma[3] after taking notice of "furtive green tea drinkers" among them. EGCG, a commercially available white powder, should be made a therapeutic option soon.

Werner Hunstein

Correspondence: University of Heidelberg, Schlosswolfsbrunnenweg 41, Heidelberg, D 69118, Germany; e-mail: werner.hunstein@urz.uni-heidelberg.de.

Conflict-of-interest disclosure: The author declares no competing financial interests.

References

1. Friedreich N, Kekulé A. Zur Amyloidfrage. Virchows Arch Path Anat Klin Med. 1859;16:50-65.
2. Körbling M, Dörken B, Ho AD, Pezzutto A, Hunstein W, Fliedner TM. Autologous transplantation of blood-derived hemopoietic stem cells after myeloablative therapy in a patient with Burkitt's lymphoma. Blood. 1986;67:529-532.
3. Shanafelt TD, Lee YK, Call TG, et al. Clinical effects of oral green tea extracts in four patients with low grade B-cell malignancies. Leuk Res. 2006;30:707-712.

2007 110: 2216
doi:10.1182/blood-2007-05-089243

Epigallocathechin-3-gallate in AL amyloidosis: a new therapeutic option?

Werner Hunstein

Blood (print ISSN 0006-4971, online ISSN 1528-0020), is published weekly by the American Society of Hematology, 2021 L St, NW, Suite 900, Washington DC 20036.