

LETTER TO THE EDITOR

Gene-Eden, a broad range, natural antiviral supplement, may shrink tumors and strengthen the immune system

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To the Editor

Gene-Eden (by polyDNA) is a broad range, natural antiviral supplement that targets viruses during their latent or chronic phase. The development of Gene-Eden was inspired by Hanan Polansky's discovery [1] of the Starved Gene phenomenon. According to Polansky, the promoter/enhancer of certain common viruses, such as Epstein Barr Virus (EBV), Cytomegalovirus (CMV), and Herpes Simplex Virus (HSV), competes during latency with the promoter/enhancer of certain cellular genes for the limiting transcription complex GABP•p300/cbp. Retinoblastoma (Rb) and BRCA1 are two cellular genes that bind this complex. Since GABP•p300/cbp transactivates these genes, and since both genes encode a cell cycle suppressor, the presence of a high copy number of a sequestering latent virus in the nucleus diminishes the expression of the Rb and BRCA1 genes, which leads to excessive cell replication and a tumor.

The patient is a 61-year-old female with an inoperable pancreatic carcinoma in the head of the pancreas with vascular involvement. The patient was receiving chemotherapy treatment of gemcitabine (Gemzar) + oxaliplatin (Eloxatin) + erlotinib (Tarceva) for a period of two months. During that time, test results and medical observations were showing a general worsening in the patient condition. The pancreatic tumor increased by 64% and was progressing towards mesenteric root (the size of the tumor was measured by the use of unidimensional measurements and the sum of the longest diameters).

Hepatic mass increased by 22% and new hypodense masses were observed. Lymph nodes were enlarged and

increased in number. Concentration of red blood cells, hemoglobin, hematocrit, lymphocytes, eosinophils and basophils decreased by 21%, 23%, 10%, 78% and 100%, respectively. As a result, the original treatment was discontinued and a new chemotherapy treatment of capecitabine (Xeloda) was initiated. A week later, the patient began a treatment with Gene-Eden in addition to the chemotherapy. After five weeks, a CT revealed a decrease of 35% in the tumor size, and a decrease of 18% in the hepatic mass. Blood test showed an increase in the concentration of lymphocytes (1.72×10^3 cells/ μ l compared with 0.70×10^3 cells/ μ l), eosinophils (0.11×10^3 cells/ μ l compared with 0 cells/ μ l) and basophils (0.02×10^3 cells/ μ l compared with 0 cells/ μ l), the new numbers were in the normal range and at levels similar to those observed prior to the initiation of the first chemotherapy treatment. In addition, the patient gained weight, felt a relief in pain, was able to get up from bed alone and to walk around and was feeling better in general.

The question is whether these remarkable results should be attributed to the new chemotherapy treatment with capecitabine alone, or whether the addition of Gene-Eden had a major role in producing the observed remission. According to latest studies, treatments with capecitabine alone or in combination with other chemotherapy agents achieved results that range between stable disease and mild or partial responses. There are only a few studies to demonstrate physical tumor shrinkage. One such study [2], which examined the effect of capecitabine on tumor size, reports that only three of the 42 patients (7%) treated with capecitabine for advanced or metastatic pancreatic cancer

showed any reduction in tumor size after median treatment time of 85 days. In our case, the decrease in tumor size was visible in the single patient that was treated, and after only five weeks of treatment. In light of that study, it is unlikely that the capecitabine treatment alone was responsible for the observed remission in our case. The possibility that Gene-Eden treatment contributed to the effect should be considered.

In addition to the decrease in tumor size, blood tests showed that the addition of Gene-Eden had a positive effect on immune markers.

Studies report that chemotherapy can have different immuno-suppressive effects, and specifically, can decrease the number of white blood cells, including lymphocytes and leukocytes. For instance, one study [3] examined the influence of chemotherapy on lymphocytes depletion in 258 breast cancer patients. In all patients, grade 3 or grade 4 lymphopenia was observed after five cycles of treatment. Another study [4] revealed a loss of antibodies against measles and rubella in children treated with intensive chemotherapy. A third study [5] compared the immunity conditions of 34 patients with breast cancer after chemotherapy and radiotherapy treatment with those of patients with primary breast cancer. T cells proportions were significantly lower in patients who received the treatment. (Note that in this study the treatment included radiotherapy, which is known to have an immuno-suppressive effect.)

Capecitabine has also been reported to cause immuno-suppression in cancer patients. A study [6] examined the efficiency of combined therapy of bevacizumab, capecitabine and gemcitabine on 50 patients with advanced pancreatic cancer. Toxicity results revealed a grade 3 neutropaenia in 22% of the patients. Another study [7] showed a 30.5% lymphocytopenia following treatment with capecitabine in 60 patients with advanced/metastatic colorectal cancer. A third study [8] reported grade 3 and 4 leucopenia in 66% of the patients with metastatic breast cancer following treatment with capecitabine-docetaxel. A fourth study [9], which examined the effect of an oral vinorelbine and capecitabine combination on 54 patients with metastatic breast cancer, reported grade 3 and grade 4 neutropaenia in 49% of the patients. These and others studies [10–13] suggest that capecitabine can be immuno-suppressive. The results of the treatment with capecitabine (Xeloda) + Gene-Eden reveal a positive effect on immune markers. We propose that the addition of Gene-Eden facilitated this effect. Considering the observed lymphopenia and leucopenia in the above cited studies, it is very unlikely that capecitabine alone caused the positive effect. Since capecitabine therapy is known to cause lymphopenia, we would like to suggest that the improvement in lymphocytes concentration in our case is due to the addition of Gene-Eden.

Conclusion

According to the Starved Gene discovery, latent viruses can cause pancreatic cancer, and an antiviral supplement that targets latent viruses can be effective in treating the disease. Gene-Eden is a promising broad range, natural antiviral supplement that targets latent viruses. The results of this study show that the addition of Gene-Eden to chemotherapy may have participated in shrinking the tumor and in strengthening the immune system in a case with poor prognosis. Such participation is consistent with the expected effects of Gene-Eden on pancreatic cancer according to the Starved Gene discovery.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

References

- [1] Polansky H. Cancer, Microcompetition with foreign DNA and the origin of chronic disease. Rochester, New York: CBCD Publishing; 2003. p. 303–32.
- [2] Cartwright TH, Cohn A, Varkey JA, Chen YM, Szatrowski TP, Cox JV, et al. Phase II study of oral capecitabine in patients with advanced or metastatic pancreatic cancer. *J Clin Oncol* 2002;20:160–4.
- [3] Tolaney SM, Najita J, Winer EP, Burstein HJ. Lymphopenia associated with adjuvant anthracycline/taxane regimens. *Clin Breast Cancer* 2008;8:352–6.
- [4] Nilsson A, De Milito A, Engström P, Nordin M, Narita M, Grillner L, et al. Current chemotherapy protocols for childhood acute lymphoblastic leukemia induce loss of humoral immunity to viral vaccination antigens. *Pediatrics* 2002;109:633–6.
- [5] Solomayer EF, Feuerer M, Bai L, Umansky V, Beckhove P, Meyberg GC, et al. Influence of adjuvant hormone therapy and chemotherapy on the immune system analysed in the bone marrow of patients with breast cancer. *Clin Cancer Res* 2003;9:174–80.
- [6] Javle M, Yu J, Garrett C, Pande A, Kuvshinoff B, Litwin A et al. Bevacizumab combined with gemcitabine and capecitabine for advanced pancreatic cancer: A phase II study. *Br J Cancer* 2009;100:1842–5. Epub 2009 Jun 2.
- [7] Sakamoto J, Kondo Y, Takemiya S, Sakamoto N, Nishisho I; Clinical Study Group of Capecitabine. A phase II Japanese study of a modified capecitabine regimen for advanced or metastatic colorectal cancer. *Anticancer Drugs* 2004;15:137–43.
- [8] Chan S, Romieu G, Huober J, Delozier T, Tubiana-Hulin M, Schneeweiss A, et al. Phase III study of gemcitabine plus docetaxel compared with capecitabine plus docetaxel for anthracycline-pretreated patients with metastatic breast cancer. *J Clin Oncol* 2009;27:1753–60.
- [9] Tubiana-Mathieu N, Bougnoux P, Becquart D, Chan A, Conte PF, Majois F, et al. All-oral combination of oral vinorelbine and capecitabine as first-line chemotherapy in HER2-negative metastatic breast cancer: An International Phase II Trial. *Br J Cancer* 2009;101:232–7. Epub 2009 Jul 7.
- [10] Bogenrieder T, Weitzel C, Schölmerich J, Landthaler M, Stolz W. Eruptive multiple lentigo-maligna-like lesions in a

- patient undergoing chemotherapy with an oral 5-fluorouracil prodrug for metastasizing colorectal carcinoma: A lesson for the pathogenesis of malignant melanoma? *Dermatology* 2002;205:174–5.
- [11] Nishida M. Pharmacological and clinical properties of Xeloda (Capecitabine), a new oral active derivative of fluoropyrimidine. *Nippon Yakurigaku Zasshi* 2003;122:549–53.
- [12] Dong N, Jiang W, Li H, Liu Z, Xu X, Wang M. Triweekly oxaliplatin plus oral capecitabine as first-line chemotherapy in elderly patients with advanced gastric cancer. *Am J Clin Oncol* 2009 Jul 2. [Epub ahead of print]
- [13] Wagstaff AJ, Ibbotson T, Goa KL. Capecitabine: A review of its pharmacology and therapeutic efficacy in the management of advanced breast cancer. *Drugs* 2003;63:217–36.