



PERSONAL VIEW

Long-term clinical remission of chronic lymphocytic leukaemia by dietary means

W M Politzer

In November 1988 my full blood count indicated a normal picture except for an increased lymphocyte level ($11.3 \times 10^9/l$). A peripheral blood film showed that the predominant cell was a small, well-differentiated lymphocyte; no blasts were seen. All blood chemistry was within normal limits. Quantitative analysis showed immunoparesis involving immunoglobulin G (IgG), IgA and IgM. Immunophenotyping showed a B-cell expansion with co-expression of CD5 and low expression of kappa and lambda surface markers (CD5 99%, CD19 89%, and CD19 and CD5 89%). Over the next 5 years my lymphocyte count reached a peak of $85.5 \times 10^9/l$. During the following 5 years a daily intake of bovine milk (700 ml) resulted in a progressive and continuous fall in my lymphocyte count from

$85.5 \times 10^9/l$ to $12.5 \times 10^9/l$, a reduction of 85%. These findings were recorded in the *SAMJ*¹ and discussed in an editorial in the same *Journal* by Professor Peter Jacobs, emeritus professor of haematology at the University of Cape Town.²

Between February 1998 and May 1999 my lymphocyte count decreased to $6.9 \times 10^9/l$ on milk only, a further reduction of 45%. An average lymphocyte count of $6.44 \times 10^9/l$ was maintained on the same treatment until March 2004.

WIL-2 immortal human lymphocyte cell cultures were exposed to casein-free filtrates of various bovine milk brands. The presence of active fractions caused enhanced metabolic activity, followed by cell death (apoptosis).³

[Metadata, citation and similar papers at core.ac](#)

degree from Oxford University. He trained at the West London Hospital, was head of chemical pathology at the South African Institute for Medical Research for 25 years, and since 1976 has worked in haematology at MEDUNSA.

on certain milk brands but not others. This is illustrated by the following examples:

- While on holiday in Europe in the spring of 1995, intake of local milk led to an increase in my lymphocyte count from 26.2×10^9 to $33.9 \times 10^9/l$, an increase of 29% over a 6-week period.



- One brand of milk used came from cattle farmed using the 'zero grazing method'.⁴ This milk led to an increase in my lymphocyte count from $8.6 \times 10^9/l$ to $9.95 \times 10^9/l$ over a 1-month period.
- Of 6 milk powders, only 1 was effective in reducing my lymphocyte count. It was subsequently established that this product was the only powder to have been fortified with vitamin D.
- It must be mentioned that the milk I used came from cows grazing between latitudes 25°S and 26°S where sufficient ultraviolet light occurs throughout the year, while there is inadequate UV light at latitudes 46° - 54°N in Europe in spring.⁵

Vitamin D occurs in variable quantities in bovine milk. The amount depends on seasonal light conditions relating especially to latitude, pigmentation of the cattle and stabling conditions. In South Africa fresh milk is not usually fortified with vitamin D, but the feed may contain added vitamin D.

In March 2004 I commenced treatment with Rocaltrol (an active form of vitamin D) at a dose of 0.25 µg (10 IU) daily and at the same time milk intake was discontinued. My lymphocyte count decreased from $6.72 \times 10^9/l$ to $5.5 \times 10^9/l$ over a period of 1 month. In April 2004 another experiment was commenced whereby 15 ml of cod liver oil was taken daily in place of the Rocaltrol until June 2004, when my lymphocyte count was $5.42 \times 10^9/l$. Although the absolute lymphocyte count was only slightly elevated, flow cytometry revealed that 73.9% of these cells co-expressed CD5 and CD19 indicating that the malignant lymphocyte clone was still present. My serum calcium remained within normal range all the time, but serum protein electrophoresis still showed immunoparesis.

Throughout the entire 16-year period of the disease there was no evidence of my leukaemia deteriorating. At no time have I taken tumour-suppressive or cytotoxic drugs and I have not received any radiation therapy.

Discussion

Tissue culture results indicated a potent constituent in bovine milk which initially stimulated the 'immortal' lymphocytes and then caused cell death.

Programmed cell death (apoptosis) is a normal physiological process vital to the maintenance of tissue homeostasis. In B-cell chronic lymphocytic leukaemia there is no aggressive proliferation of lymphocytes but a defect in apoptosis that causes a prolonged lifespan and accumulation of these cells in the blood, bone marrow, lymph nodes, spleen and liver.⁶ In most B-chronic lymphocytic leukaemia cells there is an increase in the anti-apoptotic protein Bcl-2 and a decrease in the pro-apoptotic protein Bax resulting in a high Bcl-2/Bax ratio. This

high ratio is the cause of the prolonged lifespan and the rapid development of resistance of the malignant lymphocytes to cytotoxic drugs. On consumption of bovine milk these lymphocytes are released into the circulation from storage sites, which may lead to a temporary increase in the peripheral lymphocyte count.

My observations of the influence of sunlight on dairy cattle in different locations indicate that it may have a direct influence on the absolute lymphocyte count in malignant lymphocytosis. The synthesis of vitamin D₃ from sunlight depends on seasonal light conditions which are particularly favourable at latitudes 25° - 26°S. To support this hypothesis I took vitamin D₃ for a month and my absolute lymphocyte count decreased to a near-normal level. All the abovementioned investigations indicate that the so-far-unidentified constituent in milk is activated vitamin D₃. Bovine milk contains cholecalciferol, 25-hydroxycholecalciferol, 24,25-dihydroxycholecalciferol and 1,25-dihydroxycholecalciferol (calcitriol). These compounds bound to vitamin D-binding protein are transported in the bloodstream. Most tissues and cells in the body, including the heart, stomach, pancreas, brain, activated T- and B-lymphocytes and activated macrophages have nuclear receptors for calcitriol (vitamin D receptors) which have numerous biological effects and do not seem to be related directly to bone and mineral metabolism. It appears that apoptosis of malignant lymphocytes is induced as a response to activation of the surface receptor for calcitriol.

Oral calcitriol inhibits proliferation and induces maturation and apoptosis of human and animal tumours.^{7,8}

Conclusion

Calcitriol appears to be the constituent maintaining my lymphocyte count at a near-normal level.

This research was supported in part by a grant from the MEDUNSA Research, Ethics and Publications Committee.

I wish to thank Dr Roger Pool for medical advice and interest in my research and Bernice Pretorius for her excellent librarian assistance.

1. Politzer WM. Chronic lymphocytic leukaemia – a personal story. *S Afr Med J* 1999; **89**: 45-46.
2. Jacobs P. Non-chemotherapy of malignant lymphocytosis (Editorial). *S Afr Med J* 1999; **89**: 53-54.
3. Politzer WM, Whitcutt JM. Chronic lymphocytic leukemia: an experimental approach. *South African Journal of Science* 2001; **97**: 449.
4. Brown V. Zero grazing — an option for difficult conditions.2002; <http://www.dardni.gov.uk/pa2002/pa020111.htm>
5. Norman AW. Sunlight, season, skin pigmentation, vitamin D, and 25-hydroxyvitamin D: integral components of the vitamin endocrine system (Editorial). *Am J Clin Nutr* 1998; **67**: 1108-1110.
6. Pepper C, Hoy T, Bentley P. Bcl-2/Bax ratios in chronic lymphocytic leukemia and their correlation with *in vitro* apoptosis and clinical resistance. *Br J Cancer* 1997; **76**: 935-938.
7. Reeve LE, Jorgensen NA, De Luca HF. Vitamin D compounds in cow's milk. *J Nutr* 1982; **112**: 667-672.
8. Smith DC, Johnson CS, Freeman CC, Miundi J, Wilson JW, Trump DL. A phase I trial of calcitriol (1,25-dihydroxy-cholecalciferol) in patients with advanced malignancy. *Clin Cancer Res* 1995; **5**: 1339-1345.