This Week's Citation Classic®
CC/NUMBER 13
MARCH 28, 1988

Larkin P J & Scowcroft W R. Somaclonal variation—a novel source of variability from cell cultures for plant improvement. *Theor. Appl. Genet.* 60:197-214, 1981.
[Division of Plant Industry, CSIRO, Canberra, ACT, Australia]

The hypothesis was proposed that plant cell culture results in high-frequency genetic change in regenerated plants. This phenomenon was called somaclonal variation, and a review of the literature indicated its observation in many species. Various mechanisms of origin and practical applications to plant improvement were discussed. [The *SCI*® indicates that this paper has been cited in over 260 publications, making it the most-cited paper from this journal.]

Philip J. Larkin
Division of Plant Industry
CSIRO
P.O. Box 1600
Canberra 2601, Australia
and
William R. Scowcroft
Biotechnica Canada
170,6815 8 Street NE
Calgary, Alberta T2E 7H7
Canada

December 17, 1987

The circumstances leading to this work were initiated by the appointment of P.J. Larkin to CSIRO to investigate whether sugarcane cell cultures could be used to find resistance to eyespot disease. The pathogen, *Helminthosporium sacchari*, was known to produce toxin(s) that were major determinants of pathogenicity. We both had the commonly held conception that cultured plant cells could be considered like microbes and that mutations might arise at similar frequencies (10^5-10^6). These rare events might be recoverable by using the toxin as an *in vitro* selection pressure. Mitosis was overwhelmingly considered by us and other plant biologists as a conservative process.

The cell culture system was readily established with an astonishing capacity for plant regeneration. Toxins were isolated and a bioassay was developed. One of us (Larkin) remembers well an embarrassing number of weeks in 1980. During the development of the bioassay, suitable susceptible parental sugarcane plants became unavailable. In their place a small group of regenerants from cultures of that genotype were used. These had not been exposed to toxin in culture. To our extreme annoyance a couple of those plants gave "resistant" reactions to the bioassay. The expectation of their being susceptible was so strong that the compelling initial conclusion was that the bioassay was unreliable. For some weeks these results were not conveyed to anyone. As true control plants and a fresh isolation of pathotoxin became available, the assays were repeated many times. The assay method was indeed vindicated and the presumptive resistant regenerants were also confirmed.

Extreme is the embarrassment of goals achieved too readily. Now at least the data were solid enough to be scrutinized by colleagues. These results[1] were not published in full until after the cited paper. However, this was the trigger to the mental shift required for the *Classic* paper. Cell culture itself results in genetic changes and sometimes at exceptionally high frequencies.

Embarrassment gave way to an excitement in something new, albeit not understood. Despite popular conceptions of science as a strictly objective enterprise, the truth is that by and large it is driven by expectations. There does come a time when conflicts among data become sufficiently acute that expectations and presuppositions are cast aside and truly innovative thinking becomes possible. We suspect that the pressures of modern science in terms of publications and careers are such that researchers by and large are very reactionary.

At W.R. Scowcroft's insistence, a number of weeks were spent searching and analysing the literature for evidence of tissue culture induced variation. Most, but not all, authors had discarded such variation as epigenetic in nature and not worthy of further study. Nevertheless, the precedence for the required mental shift may justifiably be ascribed to others.[2-4] The cited paper is the fruit of that review of the literature and an endeavour to assess some of the mechanisms that might be responsible. These discussions were specifically designed to point to promising topics of experimentation for ourselves and others. Pleasingly, much research has ensued in most of these areas. We signaled six phenomena that might be involved in somaclonal variation. Five of these have been vindicated together with a few others. Summaries of recent research have been published.[5,6]

The frequent citation of this paper may be ascribed to the timeliness in calling attention to this phenomenon, to the fact that we had the temerity to give it a name, and to our colleagues at CSIRO and scientists elsewhere who subsequently provided definitive data to substantiate the concept of somaclonal variation.

1. **Larkin P J & Scowcroft W R.** Somaclonal variation and eyespot toxin tolerance in sugarcane. *Plant Cell Tissue Organ Cult.* 2:111-21, 1983. (Cited 10 times.)
2. **Heinz D J, Krishnamurthi M, Nickell L G & Maretzki A.** Cell, tissue and organ culture in sugarcane improvement. (Reinert J & Bajaj Y P S, eds.) *Applied and fundamental aspects of plant cell, tissue and organ culture.* Berlin: Springer-Verlag, 1977. p. 3-17. (Cited 55 times.)
3. **Skirvin R M.** Natural and induced variation in tissue culture. *Euphytica* 27:241-66, 1978. (Cited 95 times.)
4. **Shepard J F, Bidney D & Shahin E.** Potato protoplasts in crop improvement. *Science* 208:17-24, 1980. (Cited 160 times.)
5. **Scowcroft W R, Davies P A, Ryan S A, Brettell R I S, Pallotta M A & Larkin P J.** The analysis of somaclonal mutants. (Freeling M, ed.) *Plant genetics.* New York: Liss, 1985. p. 799-815.
6. **Larkin P J.** Somaclonal variation: history, method and meaning. *Iowa State J. Res.* 61:393-434, 1987.