but by others as being more likely to be due to accidental contamination[14]. If contamination is the correct explanation then the 'spontaneous' agent should be identical with one of the small number of strains in use in Pattison's laboratory but material has not been available for agent identification. Pattison also cites the work and interpretations of Parry[15] but fails to quote the critical reanalysis[16] of Parry's data which produced entirely different conclusions.

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Ageing cell cultures

Is there an irreversible loss of mitotic ability in mammalian cell populations during ageing[1]? The results of continuous labelling alone can neither confirm nor refute this hypothesis.

Macieira-Coelho[2] continuously labelled cultures of human embryonic fibroblasts with tritiated thymidine. He found that the proportion of unlabelled cells declined to zero in a convex fashion. His result is an inevitable consequence of the continuous labelling method.

Macieira-Coelho did not distinguish between unlabelled dividing cells and unlabelled non-dividing cells. By definition non-dividing cells will not take up label and will not reproduce. If the initial proportion of unlabelled non-dividing cells is $q/2^0$, then after k population doublings this proportion will be reduced to $q/2^k$. The proportion of unlabelled dividing cells will decline at a faster rate since the number of dividing cells that are unlabelled also will decrease. The proportion of unlabelled cells is the sum of these two proportions. It will decline to zero in a convex fashion (Table 1).

It will decline whether time is measured chronologically, in population doublings, in split ratios or in cell generations[3]. It will decline whether the population is growing or static, for in a homeostatic population, where cell death is balanced by cell birth, the number of unlabelled cells will decline. It will decline whether or not un-

labelled cells continue to enter the division cycle.

The results reported by Macieira-Coelho[2] are artifacts of the continuous labelling method.

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[1] Good, P. I., and Watson, D., *Exp. Geront.*, **8**, 147 (1973).
[2] Macieira-Coelho, A., *Nature*, **248**, 421 (1974).
[3] Good, P. I., *Cell Tissue Kinet.*, **5**, 319 (1972).
[4] Smith, J., and Hayflick, L., *J. Cell Biol.*, **62**, 48 (1974).

DR MACIEIRA-COELHO REPLIES—I think the theoretical speculations made by Dr Good are more likely to be artifacts than the experimental results on which I have based my conclusions.

Dr Good says that the decline in the population of unlabelled cells is an inevitable consequence of the continuous labelling method. As a matter of fact, my data show very clearly that this is not the case, and that the proportion of unlabelled cells depends on the size of the inoculum. With high inocula not all the cells have time to enter the cycle before division stops due to cell crowding. Furthermore, if after a continuous labelling one finds only 6% of unlabelled cells, regardless of how this equilibrium is reached at resting phase (loss of non-dividers, dilution, and so on) it means that there are only 6% of cells which did not enter the S period. The possibility remains that these cells are slowly cycling cells delayed in other periods, a hypothesis most mathematicians do not like to consider.

Eight years ago I published results[1] which led to the conclusion that during the growth decline of human fibroblasts the cell population becomes strongly heterogeneous and the cells show a spectrum between the two extremes, that is, complete inhibition of division and the normal division cycle. What has to be kept in mind is the presence of a spectrum, and the experimental evidence suggests that there is no predominant cell type (that is, cells with fast, intermediate or long generation times, or non-dividers). If there is a fraction of cells which do

not divide at all in the population, this is a small fraction as my data have now shown. There is no reason whatsoever to make speculations about growth kinetics stating that this small fraction would play a bigger role than the rest of the spectrum of cells with a whole range of generation times.

[1] Macieira-Coelho, A., Ponten, J., and Philipsen, L., *Expl Cell Res.*, **42**, 673 (1966).

Intelligence and handedness

GIBSON[1] has stated that his data do not support Levy's[2] contention that left-handed subjects have significantly lower visuospatial IQ's. Cohen's[3] factorial study of the Weschler adult intelligence scale suggests that a more appropriate test of verbal IQ than the full Weschler scales used by both Gibson and Levy would consist of the average of age-weighted scores on subtests weighting on verbal comprehension (information, comprehension, similarities, vocabulary) and a test of visuospatial IQ would consist of subtests weighting on perceptual organisation (block design, object assembly).

Table 1 Comparison of two measures

		Right handed	Left handed
Verbal IQ	mean	123.3	125.5
	s. d.	5.36	7.08
Performance IQ	mean	114.6	112.7
	s. d.	6.82	6.72
Verbal comprehension score	mean	14.23	14.78
	s. d.	1.37	1.27
Perceptual organisation score	mean	12.37	12.4
	s. d.	1.60	3.54

Comparing a small sample of left- and right-handed subjects, ten of each group, no significant differences between the two groups were found on either the full verbal and performance IQ's or on the verbal comprehension and perceptual organisation scores (Table 1). These data support Gibson's findings.

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[1] Gibson, J. B., *Nature*, **243**, 482 (1973).
[2] Levy, J., *Nature*, **224**, 614 (1969).
[3] Cohen, J., *J. Consult. Psychol.*, **21**, 451 (1957).

DR GIBSON REPLIES—A reanalysis of my data along the lines suggested by Roberts has given similar results. There are no significant differences between left-handed and right-handed subjects on either the verbal comprehension or perceptual organisation scores.

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Table 1 Percentage of unlabelled cells at confluence, by type, following continuous exposure to labelled tritiated thymidine

	Observed[2]	Theoretical	
Split Ratio	%	Non-dividers %	Dividers %
No transfer	100	16 (ref. 4)	84 (ref. 4)
1:1	79	16	63
1:2	25	8	17
1:4	3	4	1

No correction was made in this table for cell death or cell loss during transfer[3].