Table 1 Methylation of Pb^{2+} by microorganisms

Pb^{2+} Concentration* ($\mu g\ ml^{-1}$)	Seed type†	Incubation time (d)	Pb(μg)‡ analysed	MePb(μg)§ produced
10	I	7	6	8
1	I	7	12	15
0.1	I	7	~10	~13
1	II	14	82	106
1	II	7	80	103
		(+7)‖	(+10)	(+13)
1	II	7	56	72
		(+7)‖	(+20)	(+26)

*Starting concentration of $Pb(OAc)_2$ in a seeded 100-ml water sample (nutrient: glucose; seed type I or II) in a 250-ml gas wash bottle.
†Seeds were prepared from 10 ml A-water (= water from an aerated aquarium), 200 mg glucose and 10 mg urea, made up with water to 100 ml, and then incubated under N_2. Seed type I: 10 ml seed (incubated for 2 weeks)+10 ml seed (incubated for 6 weeks) + 10 ml A-water II: 10 ml seed (incubated for 5 d)+10 ml A-water.
‡'Volatile' Pb found in scrubber solution (see text).
§Amount of Me_4Pb equivalent to analysed amount of Pb.
‖Same solution, additional incubation time in N_2, after exchange of gas atmosphere, giving additional quantities of reaction product.

another source for Me_4Pb in the experiments with Me_3PbOAc (refs 1, 2). (The possibility of direct methylation of Me_3PbOAc still has to be investigated.) Furthermore one can expect that the portion of Me_4Pb chemically formed by redistribution is higher in sulphide systems[2], as the redistribution rate greatly increases with increasing concentration of added salt MX and with increasing polarisability of X[3,4]. Also, since Pb^{2+} formed according to reactions (2) or (3) is precipitated as PbS, not enough Pb^{2+} is in solution to allow appreciable microbial alkylation to Me_4Pb.

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1 Wong, P. T. S., Chau, Y. K., and Luxon, P. L., *Nature*, **253**, 263–264 (1975).
2 Jarvie, A. W. P., Markall, R. N., and Potter, H. R., *Nature*, **255**, 217–218 (1975).
3 Haupt, H.-J., Huber, F., and Gmehling, J., *Z. anorg. allg. Chem.*, **390**, 31–40 (1972).
4 Gmehling, J., thesis, Univ. Dortmund (1973).
5 *Deutsche Einheitsverfahren zur Wasseruntersuchung, H5, 12* (edit. by GDCh, Fachgruppe Wasserchemie, Verlag Chemie, Weinheim, 1972).
6 *Standard Methods for the Examination of Water and Wastewater, 13th ed.*, 489 (edit. by APHA, AWWA, WPCF, Washington, 1971).
7 Snyder, L. J., and Henderson, S. R., *Analyt. Chem.*, **33**, 1175–1180 (1961).
8 Dagnall, R. M., West, T. S., and Young, P., *Talanta*, **12**, 583–588 (1965).

Muscle regeneration in dystrophic mice

IN their letter[1], Hamburgh *et al.* set out to show that other workers in their field[2] had drawn incorrect conclusions from scanty experimental evidence. There are, however, certain inaccuracies in their text which do not do anything towards their claim of clarification.

In one paragraph we read that, "foetal cord from 15-d-old mouse embryos . . . were explanted". A little later we read, "spinal cords were dissected from foetal mice aged 13–14 d *in utero*". This may seem a trivial point, but the foetal age is critical in these experiments, and such factual mistakes could lead other workers seriously astray.

Their most serious error is in the confusion of the two allelic mutant genes dy^2J and dy. Their experiments were carried out with the dy^2J mutant exclusively. They state, however, that, "normal muscle coupled with either normal or dy foetal spinal cord regenerated in culture". One can only assume that these authors were using 'dy' as an abbreviation for dystrophic, or else they are guilty of negligence. Whichever applies, it seriously detracts from the value of the work.

They have also misinterpreted my letter[3] in which I did not confirm their work because my experimental system was entirely different. I did not say that "dystrophic" muscle would regenerate normally, since of the two mutants I described, only one (dy^2J) showed regeneration which was normal. It is obvious that little attention was paid to my concluding paragraph, which emphasised the need for caution and accuracy when working with murine muscular dystrophy.

With an air of finality the authors, to whom these criticisms are directed, concede that tissue culture may no longer be a fruitful research tool in this field.

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1 Hamburgh, M., Peterson, E., Bornstein, M. B., and Kirk, C., *Nature*, **256**, 219–220 (1975).
2 Gallup, B., and Dubowitz, V., *Nature*, **243**, 287–289 (1973).
3 Parsons, R., *Nature*, **251**, 621–622 (1974).

HAMBURGH ET AL. REPLY—We admit that the reference to foetal age may have been misleading! Timing of foetal age depends on the method of counting: some investigators call fertilisation, as shown by the appearance of the vaginal plug, day 0 whereas others call it day 1. It is always implied that any designation of foetal age is ± 12 h.

The designation 'dy' is an acceptable abbreviation for dystrophic and as the text clearly states that "phenotypically dystrophic mice were obtained from matings between tested homozygotes for the dystrophic gene dy^2J", this should have been sufficient to clarify the point.

As for Parsons' letter[2] confirming our earlier work, he himself states[2] that "my results are essentially similar to those of Paul and of Hamburgh *et al.*[3]". Although there may be a difference between "results that are essentially similar" and results that confirm, it is, however, so fine, that we admit, it escaped us. Parsons states[4] that little attention was paid to his concluding paragraph[2] which emphasised the need for caution and accuracy when working with murine muscular dystrophy. We are fully familiar with the differential regenerative capacity exhibited by minced muscle obtained from the 129/ReJdy strains and the C57BL/6J dy^2J strains in his culture conditions.

Our comment that the tissue culture set up may not be as well suited as originally anticipated merely expresses an experience shared by other investigators that many genetic defects do not express themselves *in vitro*. Parsons, interprets the sense of this comment to mean that "tissue culture may no longer be a fruitful research tool in this field".

We should like to take this opportunity to mention, however, that whatever the reasons for the different result, between the experiments by Gallup and Dubowitz[5] and our own[1], they may well be related to differential expressivity of the dy^2J gene. Slightly different tissue culture environments, differences in foetal age sex and other, yet to be identified factors, come to mind. Genes differ both in expressivity and penetrance in different environments, and gene mutations can often be revealed only by proper challenges. We consider Gallup and Dubowitz's series to be valid and most stimulating experiments.

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1 Hamburgh, M., Peterson, E., Bornstein, M. K., and Kirk, C., *Nature*, **256**, 219–220 (1975).
2 Parsons, R., *Nature*, **251**, 621–622 (1974).
3 Hamburgh, M., Bornstein, M. B., Peterson, E., Masurovsky, E. B., and Kirk, C., *Progr. Brain Res.*, **40**, 497–508 (1973).
4 Parsons, R., *Nature*, **259**, 158 (1976).
5 Gallup, B., and Dubowitz, V., *Nature*, **243**, 287–289 (1973).